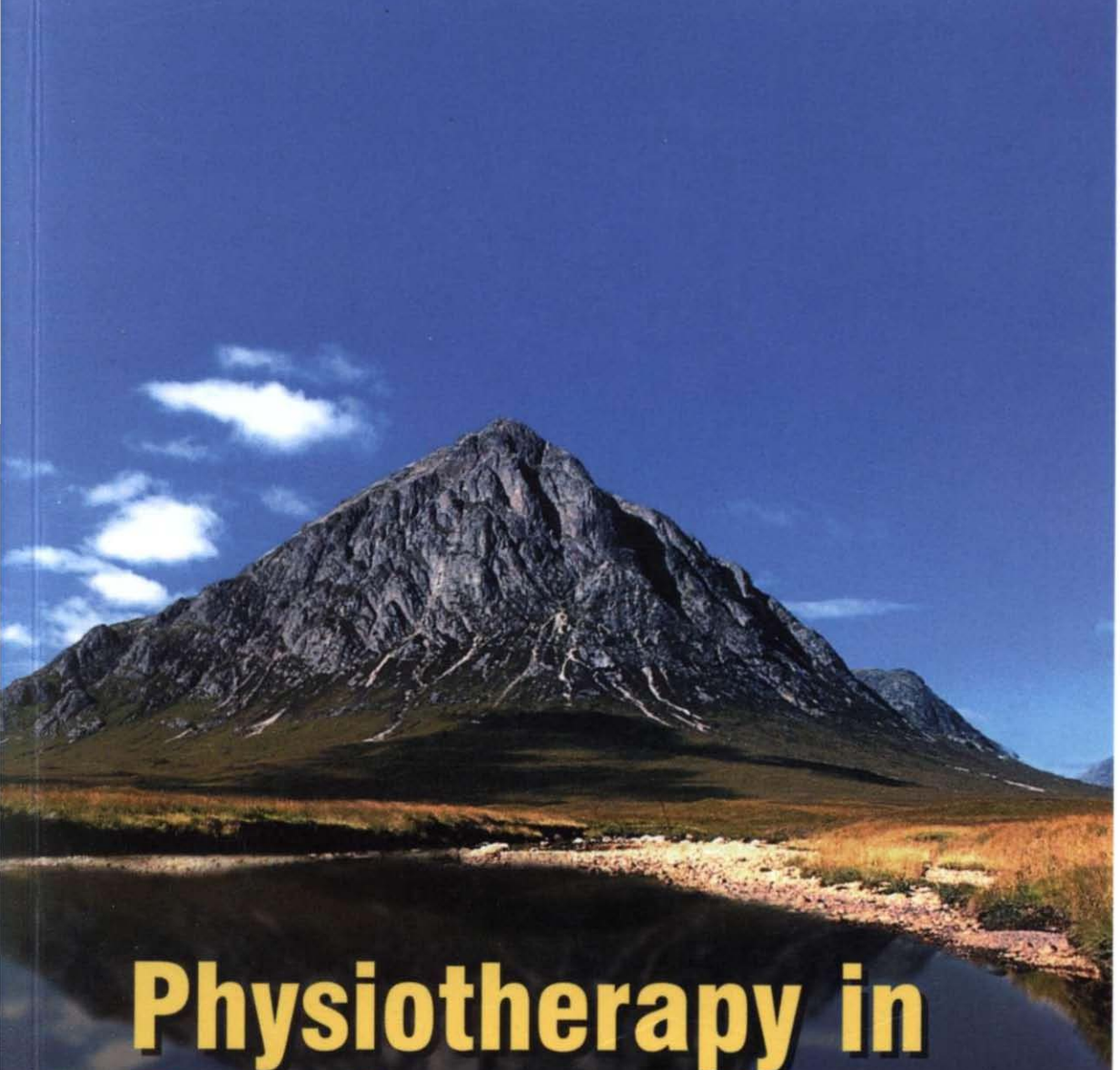


Physiotherapy in Respiratory Care

An evidence-based approach to respiratory and cardiac management

Third Edition

Alexandra Hough

nelson thornes

Physiotherapy in Respiratory Care

Third edition

By Alexandra Hough

Physiotherapy in Respiratory Care, in its third edition, continues to use a practical, problem-solving approach, based on research, to provide clarity of thought and to challenge traditional assumptions.

Chapters 1 and 2 lay the foundation for problem solving by integrating physiology and practice. The majority of the book is then devoted to physiotherapy management and its rationale. It specifically addresses the problems of patients with breathlessness, those undergoing surgery or in intensive care, the elderly and children. Pulmonary rehabilitation and home management reflect changing policies in health care. Advice on emergencies and on-calls is given with flow charts to assist decision-making. Physiological reasoning, outcome measures and critical review are themes throughout, and the book concludes with an evaluation of respiratory physiotherapy.

The new edition has been widely updated and expanded. It includes the latest research and an extensive reference list. Readers are encouraged to think through a range of situations by answering questions on case studies, and recommended reading is provided at the end of each chapter.

This is essential reading for physiotherapy students, clinicians, educators and nurse specialists in respiratory and intensive care.

Alexandra Hough is a clinical specialist at Eastbourne District General Hospital and a freelance lecturer.

nelson thornes

www.nelsonthornes.com

ISBN 0-7487-4037-6

9 780748 740376

Physiotherapy in Respiratory Care

An evidence-based approach to respiratory and cardiac management

THIRD EDITION

Alexandra Hough
Physiotherapy Respiratory Specialist
Eastbourne District General Hospital
Sussex, UK

First published in 1991 by:
Chapman & Hall
Second edition 1996

Third edition published in 2001 by:
Nelson Thornes Ltd
Delta Place
27 Bath Road
Cheltenham
Glos.
GL53 7TH
United Kingdom

03 04 05 / 10 9 8 7 6 5 4

A catalogue record for this book is available from the British Library

ISBN 0-7487-4037-6

Page make up by Acorn Bookwork, Salisbury, Wiltshire

Printed and bound in Italy by Canale

Contents

Preface

Respiratory care is an immensely satisfying branch of physiotherapy. It challenges our intellect, exploits our handling skills and employs our humanity to the full.

Respiratory physiotherapy is both art and science. It is not an exact science. Effectiveness depends on problem-solving. This requires practice in defining problems, evidence-based knowledge to address problems, and a clear perspective of patients' needs. Clinicians, students and educationists expect integration of theory and practice, explanations that are referenced and physiologically sound, and exact detail of technique. This book is written for such readers and for those who question fundamental assumptions and traditional rituals.

The third edition is updated for a health-care system that is discharging patients from hospital 'quicker and sicker'. This edition also takes account of patients who have become more knowledgeable through the media and the Internet. There is extra coverage of practical and safety tips because pressures on students allow less time for practice.

There are patient handouts, tables of exercises, flow charts and many illustrations in the third edition. Outcomes are identified for each problem, and evaluation of practice continues to be developed in response to the needs of patients, clinicians and employers. The glossary serves as a quick reference guide or can be read in its own right.

An abbreviated case study with each chapter reinforces problem-solving, goal-setting and X-ray interpretation. Snippets of literature appraisal are interspersed to hone critical thinking. Patient experiences and research findings are incorporated throughout.

The book is suitable for physiotherapists from student level to accomplished clinician because problem-solving requires thinking rather than experience. It is also suitable for specialist respiratory nurses. The clinician will find here the opportunity to achieve clarity of thought and develop mastery in respiratory care. Enjoy it.

Acknowledgements

Profound thanks to the patients who have taught me much over the years. I am also indebted to Veronica Bastow for her wisdom, perception and meticulous criticism of the manuscript, to Sarah Davies for her invaluable advice, and to Clive Liles for his detailed manuscript review. Specialist advice has been gratefully received from Alison Aldridge, Jon Anderson, Helen Davies, Diana Davis, Suzanne Roberts, Liz Ruffle, Arti Shah, Ruth Vardy, Nick Watson, Fran Woodard and Christine Young. Many thanks to Mel Calman for his cartoons and Nicholas Taylor for his photographs. And to the students with whom it has been my privilege to work and learn, thank you.

Royalties donated to Amnesty International.

Dedicated to Carol.

1 Physiological basis of clinical practice

Summary

Introduction

Breathing is the basic rhythm of life.

Hippocrates

Breathing is unique. Most of us give it little thought, yet it can be automatic or voluntary and is preserved in unconsciousness. It is associated with a respiratory system of remarkable ingenuity. An understanding of how this system works creates a foundation for logical practice.

The respiratory system is involved in the pumping of gas into the lungs, gas exchange from lungs to tissue cells, acid–base balance, metabolism, speech, and defence of the body against the elements. This chapter will place less emphasis on the textbook lungs of hefty young males than on those of patients who may smoke and may be overweight, stressed and past middle-age. Keep a finger in the Glossary throughout.

Defence

Every day, 300 million alveoli in the adult lung expose a surface area of 80 square metres, or nearly the size of a tennis court, to a volume of air and pollutants that could fill an average swimming pool (Hanley and Tyler, 1987). It is

only by means of a sophisticated biological barrier that the body does not succumb to this onslaught. Indeed, so effective is the pulmonary defence system that the lung is normally sterile below the larynx (Ferdinand, 1998). However, the lung is vulnerable to systemic events such as septicaemia (Murch, 1995).

Lung defence is based on a network of filters, secretions, reflexes and specialized cells. Physiotherapists treat patients whose defences are breached when the nose is bypassed by mouth-breathing or artificial airways, cilia damaged by smoking or disease, and cough inhibited by pain or weakness.

Nose

The nose is the gatekeeper of the respiratory tract, providing the first line of defence by means of:

- sensing suspicious smells
- sneezing in response to irritating substances
- filtering large particles
- protecting against cold dry air and insulating against swings in atmospheric temperature and humidity.

During inspiration, the nasopharynx exposes inspired gas to a large area of highly vascular, moist mucus membrane. The respiratory tract loses an average 250 mL of water a day (Branson, 1999), but nasal mucosa can supply nearly a litre of fluid to inspired air a day if required (Eubanks and Bone, 1994, p. 50). During exhalation, the upper airways reclaim a majority of the heat and moisture added during inspiration. Nose-breathing is three times as efficient at humidification as mouth-breathing.

Pharynx

The entrance to the oropharynx is guarded by tonsils and adenoids, the removal of which renders children extra vulnerable to passive smoking (Chen *et al.*, 1998). The lower pharynx houses the epiglottis, a leaf-like lid that snaps shut over the larynx during swallowing to prevent aspiration into the trachea.

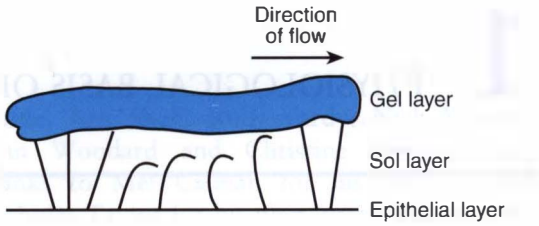


Figure 1.1 The mucociliary escalator. The sol layer is a permanent source of water in the airways and enables the cilia to beat efficiently. Claws on the tips of the cilia grip the gel layer (mucus) and a whip-like movement propels it mouthwards.

Bronchoconstriction

If irritant particles are inhaled, normal bronchoconstrictor tone is increased reflexly to protect the airway. In diseases such as asthma, this mechanism is exaggerated and is then termed bronchospasm, which increases the work of breathing and interferes with gas exchange.

Mucociliary escalator

Particles that escape filtration in the nose are trapped on a sticky mucus blanket lining the airways, then carried by cilia from the terminal bronchioles to the throat over a period of several hours (Pavia, 1991). This moving staircase (Figure 1.1) propels the mucoid secretions to the pharynx and larynx, from where they are swallowed or, if excessive, expectorated. Secretions are propelled by cilia beating synchronously at approximately 20 strokes a second. They move at speeds of between 0.5 mm/min in the small airways and 20 mm/min in the trachea (Rankin, 1998).

The mucociliary blanket normally clears 10–100 mL secretions a day, or up to 300 mL when necessary (Hodgkin *et al.*, 1993, p. 469). Other protective functions of the mucus are humidification, waterproofing, antibacterial activity and insulation.

This finely co-ordinated mechanism is compromised by dehydration, smoking, hypoxia, inflammation or pathological conditions that affect the viscosity of mucus or function of cilia. Impaired mucociliary clearance predisposes to infection (Jansen, 1995).

Cough

Clearance of secretions depends primarily on mucociliary transport and secondarily on cough. The cough is the body's strongest physiological reflex whose function is to clear blockages in the upper airway and, as a reserve mechanism, to expel secretions and debris when mucociliary clearance is damaged or overwhelmed.

A cough occurs by voluntary effort, or reflexly from irritants inside or outside the lung that stimulate inflammatory, chemical, mechanical or thermal receptors. These are located in the pleura, the airways between the larynx and segmental bronchi, and, unexpectedly, in the external auditory canal. They are most sensitive at the glottis and carina and least sensitive beyond the fourth-generation bronchi. Stimulation of the pharynx causes a gag rather than a cough.

A cough comprises:

- an inspiratory gasp to 90% of total lung capacity
- closure of the glottis and trapping of air in the lungs to create intrathoracic pressures of up to 300 mmHg (Irwin *et al.*, 1998), narrowing the trachea and main bronchi by 60% (Rees, 1987)
- sudden opening of the glottis, causing air to explode outwards at up to 500 mph or 85% of the speed of sound (Irwin *et al.*, 1998), shearing secretions off the airway walls.

Coughing is accompanied by violent swings in intrapleural pressure which cause dynamic compression of airways and speeding of gas flow. Dynamic compression is initiated in the trachea at high lung volumes and extends peripherally as lung volume decreases, ensuring that the full length of the tracheobronchial tree is affected (Irwin *et al.*, 1998). In most people, the airways reopen with a subsequent deep breath but in those unable to take a deep breath they stay closed for lengthy periods (Nunn *et al.*, 1965). Beyond about the 10th generation (Pavia, 1991), airflow cannot attain sufficient speed to expel inhaled irritants by coughing.

The cough mechanism is less efficient in people with obstructive airways disease because of poor expiratory flow rates and airways that tend to collapse on expiration. Coughing may fail in the presence of coma, neuromuscular disease or postoperative pain. It is weakened if the glottis is bypassed by intubation or tracheostomy.

Bronchospasm and exhaustion may follow sustained bouts of coughing. The abdominal pressure associated with coughing predisposes people with a chronic cough to stress incontinence. Despite high pressures, overdistension of alveoli and barotrauma (p. 344) are avoided by the presence of the rib cage and contraction of intercostal and abdominal muscles to buttress the chest wall.

Other lung defences

Further mechanisms await pollutants that evade the above defences. They include an immunoglobulin in respiratory secretions called IgA, 600 million scavenger macrophages (multiplied fourfold in smokers) and alpha$_1$-antitrypsin, a plasma protein that combats proteolytic enzyme activity, which would otherwise destroy alveoli. Asbestos particles circumvent these and other defences because of their peculiar shape. Gases can pass through the alveolar–capillary membrane, a process that forms the basis of chemical warfare, carbon monoxide poisoning and inhalational anaesthesia (Denison, 1996).

The entire blood volume passes through the lungs, which help to detoxify circulating foreign substances, perform a range of metabolic functions and act as a filter to protect the arterial system, particularly the coronary and cerebral circulations, from blood clots, fat cells, detached cancer cells, gas bubbles and other debris. Extracorporeal support systems such as cardiopulmonary bypass include a filter to perform some of these functions.

CONTROL

Breathing is normally controlled with such exquisite sensitivity that O_2 and CO_2 in the blood are maintained within exact limits despite

unpredictable metabolic changes. Clusters of neurones in the pons and medulla receive and integrate a multitude of stimuli from the rib cage, lungs, chemoreceptors, metabolic and other systems. They then discharge impulses to the respiratory muscles, which, unlike cardiac muscle, do not contract spontaneously.

The respiratory centres perceive and respond to altered posture, exercise and other variables. Respiratory control occurs at a subconscious level but can be overridden by voluntary action such as breathing exercises or reflexes such as speech, laughter, emotion, pain, sudden cold and some pathological states.

MECHANICS

The respiratory muscles

Respiratory muscles are the only skeletal muscles vital to life. They provide the power for the 'respiratory pump'. Other components of this pump are the chest wall, nerves and respiratory centres. The chest wall comprises the rib cage and abdominal-contents-plus-diaphragm, which act as a mechanical couple. Respiratory muscles extend from the mastoid process to the pubic symphysis.

Inspiration

The diaphragm separates two compartments of markedly different densities, the thorax and abdomen, and generates two-thirds of the vital capacity (Denison, 1996). This muscle was thought to be the seat of the soul by the ancient Greeks but, despite this distinction, its exact mechanism is still a source of some mystery. It is a dome-shaped sheet of muscle upon which the lungs sit, and is attached to the bottom of the rib cage. At rest it extends upwards almost to nipple level. Contraction flattens it, displacing the abdominal viscera downwards by 5–7 cm and creating negative intrathoracic pressure, which sucks air into the lungs.

The contracting diaphragm presses down against the fulcrum of the abdominal contents and, when the limit of abdominal wall compliance is reached, outwards against the lower rib cage, causing expansion of the lower chest. The abdomen protrudes out on inspiration unless prevented voluntarily or by tight clothing.

The external intercostal muscles stabilize the chest wall so that diaphragmatic contraction can create these pressure changes. Other necessary respiratory muscles are the scalenes, which stabilize the upper rib cage to prevent it being pulled downwards (Tobin, 1990), and pharyngeal muscles, which prevent collapse of the upper airway. Accessory muscles become major inspiratory muscles when there is increased work of breathing, e.g. by airflow obstruction or exercise, leading to sequential recruitment of chest wall, mandibular and facial muscles (Breslin, 1996). During arm activity, intercostal and accessory respiratory muscles are obliged to stabilize the torso, leaving the diaphragm to take a greater load.

Expiration

Normal expiration is largely passive, lung elastic recoil providing the driving pressure. Elastic recoil is caused firstly by surface tension acting throughout the vast gas–liquid interface lining the alveoli, and secondly by elasticity of lung tissue that has been stretched during inspiration. If not counterbalanced by outward recoil of the chest wall, elastic recoil would pull the lung inward to a litre below its natural resting position (Sykes and Young, 1999, p. 22). Elastic recoil pressure decreases at low lung volume.

The transition between inspiration and expiration is smoothed by a brake on expiratory flow caused by airway resistance, especially at the larynx, and continued low-grade inspiratory muscle activity. Airways are narrower during expiration than inspiration so that it is more difficult to empty the lungs than to fill them. This becomes significant in obstructive airways disease, when abdominal and internal intercostal muscles may be recruited to augment passive recoil. These expiratory muscles are thought to tire more easily than inspiratory muscles (Fuller *et al.*, 1996). Active expiration

also occurs with exercise, speech, coughing, and sneezing.

Pressure

Alveolar pressure: pressure inside the lung
Pleural (intrapleural/intrathoracic) pressure: pressure in the pleural space
Transpulmonary (transmural) pressure: pressure difference inside and outside lung, i.e. the difference between the above two pressures, representing the driving pressure responsible for inflating the lungs.

Alveolar pressure is negative on inspiration and slightly positive on expiration. Pleural pressure is normally negative because of inward pull from lung recoil and outward pull from chest wall recoil. This creates an average negative pleural pressure of $-2\,cmH_2O$ at end-expiration and $-6\,cmH_2O$ at end-inspiration. The inward and outward recoil forces are in equilibrium at the end of a quiet exhalation (functional residual capacity). Recoil of the chest wall assists inspiration, especially from low lung volumes. A change in alveolar pressure of only $1\,cmH_2O$ is usually enough for airflow but diseases that obstruct airflow or restrict lung expansion cause an increase in this requirement.

These pressures are disturbed by:

- pneumothorax, which neutralizes pleural pressure so that the lung's inward pull is unopposed and it shrivels inwards
- emphysema, which reduces lung elastic recoil, so that the outward pull of the chest wall is unopposed and the lung hyperinflates.

Resistance

Resistance is present whenever there is airflow through a tube because gas slides against the walls and over itself. Airflow resistance depends on the calibre of the airway. Peripheral airflow resistance is low because the large number of small airways creates a wide total cross-sectional area (Figure 1.2). The upper respiratory tract, whose total cross-section is narrow and airflow turbulent, causes higher resistance.

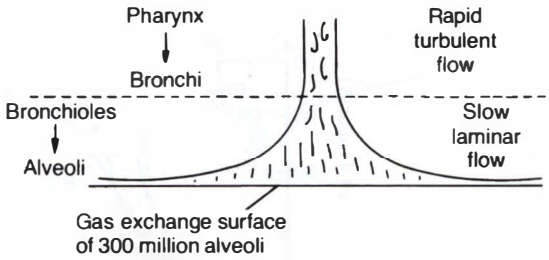


Figure 1.2 Increase in total cross-section of airways as they subdivide.

The nasal passages contribute 50% of normal airway resistance (Turner, 1997). The rest is shared out (Eriksson, 1996):

- larynx: 25%
- trachea to 8th generation: 20%
- peripheral airways: 5%.

These differences are most relevant when turbulence and resistance are increased by obstructive airways disease. The nasal route resists airflow more than the oral route, which is why we breathe through the mouth when breathless or exercising.

Airflow resistance is responsible for about 80% of the work of breathing. Lung parenchyma contributes the remaining 20% (Levitzky, 1995, p. 34).

Compliance

$$\text{Compliance} = \frac{\text{change in volume}}{\text{change in pressure}}$$

Compliance is the ease with which the lungs inflate. It reflects their ability to extend and recoil. It is represented by the relationship between volume and pressure, which is curved rather than linear (Figure 1.3). The lung is least compliant, i.e. stiffest, at either extreme of lung volume, so that it is difficult to inflate alveoli that are closed or hyperinflate those that are fully inflated, in the same way that blowing up a balloon is most difficult at either extreme.

The contribution of lung parenchyma to compliance is related partly to tissue elasticity

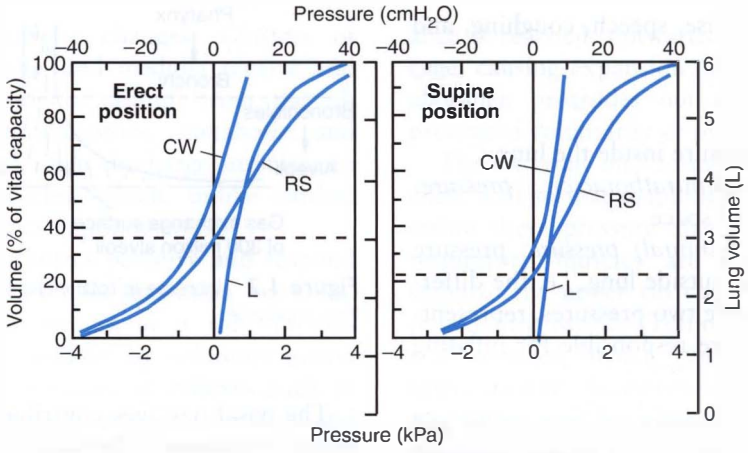


Figure 1.3 Pressure–volume curve describing compliance of lung (L), chest wall (CW) and total respiratory system (RS). Compliance is greatest on the steep part of the curve, and more pressure (effort) is needed to increase lung volume at either extreme of inflation. Examples for a low-volume state are atelectasis or fibrosis, and for a hyperinflation state, emphysema or acute asthma. The dotted line shows the lower functional residual capacity in supine. Residual volume excluded. (From Sykes, K. (1999) *Respiratory Support*, BMJ publishing, London)

but mostly to surfactant in alveolar fluid. This acts like detergent to decrease surface tension and prevent the wet alveolar walls sticking together, a force that can be likened to trying to peel open a plastic bag that is wet inside. Surfactant stabilizes the lungs by preventing small alveoli collapsing and emptying their contents into large alveoli. It also has antioxidant and anti-inflammatory properties (Nicholas, 1997).

The contribution of airways to compliance relates to their calibre, resistance being increased and compliance decreased by bronchospasm, oedema, the floppy airways of emphysema and, to some extent, secretions in the large airways where there is greater overall resistance.

Low compliance occurs with obstructed airways, fibrotic lungs, a stiff chest wall, low lung volumes and disorders of surfactant production such as the respiratory distress syndromes.

Static compliance is measured during a breath-hold such that equilibrium is achieved between alveolar pressure and mouth pressure, alveoli being filled to a volume determined by their regional compliance. Dynamic compliance is measured during breathing. It normally approximates static compliance but may be less in diseased lungs if regional variations in compliance and resistance mean that alveolar filling is not completed during inspiration.

Work of breathing

Work is done during inspiration to overcome the resistive and elastic forces of airways, lungs and chest wall. Work of breathing (WOB) can be defined in two ways:

- the pressure required to move a volume of gas, i.e. transpulmonary pressure × tidal volume
- oxygen consumed by the respiratory muscles, i.e. the oxygen cost of breathing (Tobin and Yang, 1990).

The maximum pressures achievable are +120 cmH_2O for a forced expiratory effort with open glottis and −80 cmH_2O for forced inspiration (Levitzky, 1995, p. 40). Normally, breathing is surprisingly efficient, helped by slippery fluid coating the moving surfaces of alveoli and pleura. The pleura, however, does not appear to be essential, and serves mainly as a 'drip pan' for pulmonary oedema fluid. The pleura is also handy for thoracic surgeons, who would find it difficult to operate if humans had

evolved in the same way as elephants, which have no pleura (Hamm and Light, 1997).

In healthy people, WOB uses 2–5% of total oxygen consumption at rest. This can be increased to 30% during exercise and 40% in patients with chronic obstructive pulmonary disease (COPD) at rest (Pilbeam, 1998, p. 111). When it reaches over 50%, as in shock, oxygen is stolen from white blood cells, which may be battling infection, and the kidneys and liver, which are trying to detoxify byproducts of the shocked state (Pilbeam, 1998, p. 141).

Deep breathing increases the work performed against elastic resistance, while rapid breathing increases the work against airways resistance (Lumb, 2000, p. 128). Most patients find the right balance, but some need assistance to find the optimal breathing pattern to minimize their WOB (p. 171).

Inspiratory muscle fatigue

Fatigue is loss of the capacity to develop force in response to a load, and is reversible by rest. It is usually associated with a more abrupt decrease in respiratory muscle strength than weakness. It can be due to failure of any of the links in the chain of command from brain to muscle. Failure within the central nervous system is called central fatigue and failure at the neuromuscular junction or within the muscle is called peripheral fatigue. Both types of fatigue are thought to affect the diaphragm (Roussos, 1996) and respiratory muscle fatigue has been identified in 10% of patients hospitalized with an exacerbation of COPD (Ramonatxo *et al.*, 1995).

Inspiratory muscle fatigue is less common than systemic muscle fatigue because the diaphragm has a large reserve capacity. It differs from other skeletal muscles in its requirement for a lifetime of sustained action against elastic and resistive loads rather than irregular action against inertial loads. It is equipped for this by having a high proportion of fatigue-resistant fibres and by the unusual way in which perfusion increases instead of decreases during contraction (Anzueto, 1992). It is thought that fatigue can occur if energy demand exceeds supply, as when WOB is increased by severe airflow obstruction (Figure 1.4). However, fatigue is often prevented by control mechanisms that reduce respiratory drive and protect the muscles from damage (Shneerson 1996b).

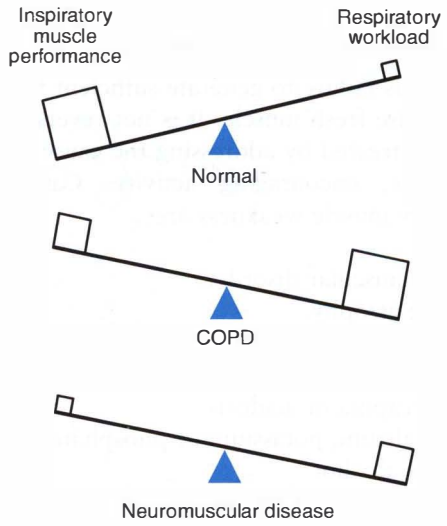


Figure 1.4 Balance between inspiratory muscle performance and respiratory workload. Normally the balance favours the inspiratory muscles, but severe COPD or neuromuscular disease increases the load and impairs endurance. (From Tobin, M. J. (1994) *Principles and Practice of Mechanical Ventilation*, McGraw Hill, New York, with permission.)

Subjectively, fatigue of respiratory muscles creates or increases breathlessness, which can be modified by release of endogenous opioids during loaded breathing (Roussos, 1996). Management of fatigue is by rest, energy conservation including use of efficient breathing and activity patterns, and sometimes non-invasive ventilation.

Fatigue serves a protective function to avoid depletion of enzymes; if the diaphragm is allowed to fatigue, recovery may take at least 24 hours (Bruton *et al.*, 1999). Procedures that force patients to overuse fatigued muscles can cause damage (Goldstone and Moxham, 1991).

Inspiratory muscle weakness

Weakness is failure to generate sufficient force in an otherwise fresh muscle. It is not reversible by rest but is treated by addressing the cause and, if appropriate, encouraging activity. Causes of respiratory muscle weakness are:

- neuromuscular disorder
- disuse atrophy
- malnutrition
- hypoxaemia
- hypercapnia or acidosis
- low calcium, potassium or phosphate
- excess alcohol
- steroids
- sepsis and multisystem failure.

Weakness predisposes a muscle to fatigue. Fatigue differs from weakness in that even a normal muscle can become fatigued with sufficient effort. Fatigue and weakness often coexist, especially in respiratory failure or during weaning from mechanical ventilation. The clinical features of fatigue and weakness are similar (p. 37). Both are expressed by breathlessness, which is covered in Chapters 7 and 9.

VENTILATION

Breathing: the process by which the ventilatory pump creates ventilation
Ventilation: gas movement between the outside of the body and the alveoli, i.e. inspiration and expiration
Respiration: (a) exchange of gases between environment and tissue cells (by external respiration at alveolar–capillary level and internal respiration at capillary-tissue level); (b) regulation of the acid–base, metabolic and defence functions of the respiratory system.
Minute ventilation or *minute volume*: ventilation per minute, i.e. tidal volume × respiratory rate.

Gas that moves in and out of the lungs is made up of:

- *alveolar ventilation*, which is the fresh air that gets into alveoli and participates in gas exchange
- *dead space ventilation* (V_D), which does not contribute to gas exchange.

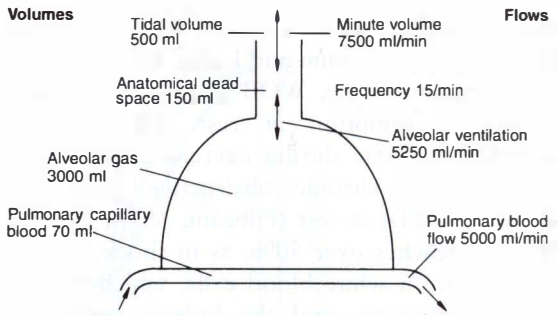


Figure 1.5 Lung unit with average volumes and flows of gas and blood for both lungs. (From West, J. B. (1995) *Ventilation/Blood Flow and Gas Exchange*, 5th edn, Blackwell, Oxford, with permission.)

Most dead space is made up of *anatomical dead space* (Figure 1.5), which is air in the conducting passages that does not reach the alveoli, i.e. that which is last in and first out.

It comprises one-third of tidal volume (V_T) in an average human, more in a giraffe. *Alveolar dead space*, representing air that reaches the alveoli but does not get into the blood, is minimal in normal lungs.

The sum of anatomical and alveolar dead space is called physiological dead space. The presence of dead space is one reason why it is more economical to increase ventilation by breathing deeper rather than faster. Dead space is most usefully expressed in relation to tidal volume (V_D/V_T).

Ventilation is not distributed evenly within the lungs (Figure 1.6). In most spontaneously breathing adults, dependent regions are better ventilated, for two reasons:

- Alveoli in upper regions are more inflated, but mostly with dead space gas. Gas travels more easily at first to the open spaces of these non-dependent regions, but the nearly inflated alveoli are rapidly filled and gas then preferentially travels to dependent regions. Alveoli in dependent regions are compressed

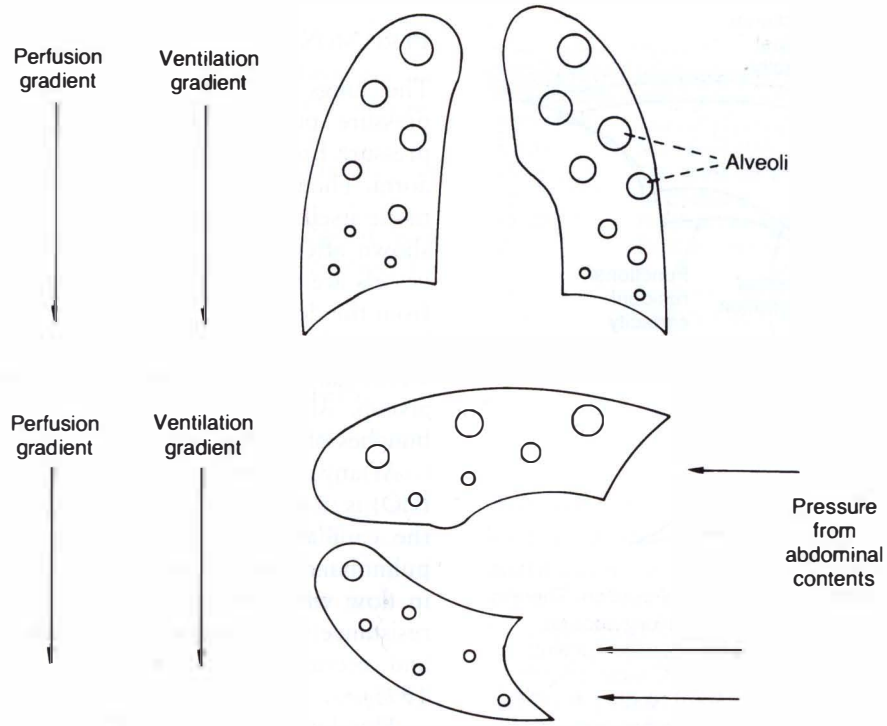


Figure 1.6 Effect of gravity on the distribution of ventilation and perfusion in the lung in the upright and lateral positions.

by the weight of the lungs, heavy with blood, above and around them. They therefore have more potential to expand, allowing greater ventilation with fresh gas to dependent regions.

- In the horizontal position, the excursion of the dependent portion of the diaphragm is greater than that of the upper portion because the lower fibres are more stretched by abdominal pressure and therefore contract from a position of mechanical advantage.

This distribution of ventilation therefore causes a gradient with greater ventilation in dependent areas. This is augmented in the side-lying position (Figure 1.7), partly because of the greater vertical distance and partly because the mediastinum is lifted on inspiration by the cushion of air that preferentially enters the lower lung.

This provides the lower lung with twice the ventilation of the upper lung (Lumb, 2000, p. 122). Although fresh gas in the lower lung provides a greater contribution to gas exchange, the upper lung is more expanded and therefore responds most to deep breathing exercises to increase lung volume. For most clinical problems, patients are usually placed with the affected lung upwards (p. 151)

The ventilation gradient is slight and therefore responsive to minor upsets. It is obliterated in the prone position because of pressure from the abdominal contents. It is reversed in grossly obese people (p. 19), in children (p. 426) and those on some modes of mechanical ventilation (p. 345).

Quiet breathing creates a tidal volume of one-tenth the vital capacity, but oscillations in V_T and involuntary sighs every 5–10 minutes help prevent alveolar collapse. Patients who are drowsy or sedated lose this mechanism.

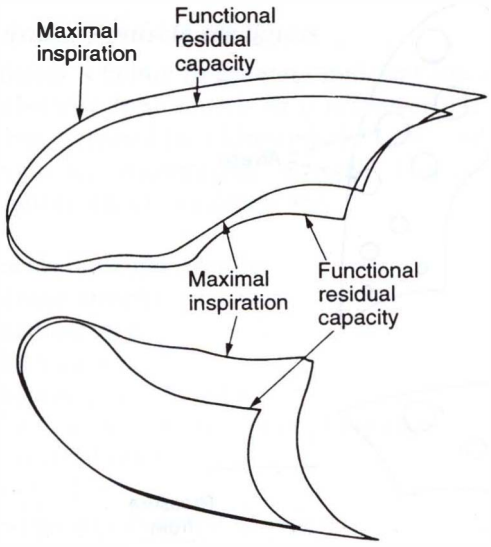


Figure 1.7 Lung volumes in the lateral position. There is greater volume change in the dependent lung because gravity causes greater pressure from abdominal contents against the lower side of the diaphragm. Greater volume change means greater ventilation. (From Nunn, J. F. (1993) *Applied Respiratory Physiology*, 2nd edn, Butterworth-Heinemann, London, p. 122, with permission.)

DIFFUSION

The wide total cross-section of the peripheral airways means that airflow essentially ceases and gas movement from the respiratory bronchioles to alveoli continues by gaseous diffusion. In the alveoli, diffusion of gases across the alveolar–capillary membrane occurs in both gaseous and liquid states, leading to equilibration of gas between air and blood.

The alveolar–capillary membrane is just 0.2–0.5 μm thick, the blood flowing between two sheets of endothelium held together by occasional connective tissue supports. Only 0.01 second is needed for oxygen to combine with haemoglobin. Diffusion is so efficient that oxygen tension is equalized in one-third of the time that the blood takes to pass each alveolus. Defects in diffusion do not play a major role in gas exchange abnormalities. Diffusion is measured by TLCO (p. 60).

PERFUSION

The lungs have a dual circulation: the low-pressure pulmonary circulation and the high-pressure bronchial circulation supplied from the aorta. The bronchial circulation services the lung tissue itself but is not essential to survival, as is shown after lung transplant when the bronchial vessels are tied. The lungs are awash with blood from the dominant pulmonary circulation, which is equivalent to 7000 km of capillaries (Denison, 1996) but acts more like a sheet enwrapping the alveoli. Alveoli are more like pock marks than bunches of grapes.

At any one time, 10% of the cardiac output (CO) is in the pulmonary circulation and 20% of the capillary beds are normally perfused. The pulmonary vasculature can respond to changes in flow with little change in pressure, reducing resistance by widening the calibre of capillaries and recruiting others that are closed (West, 1995).

This low-pressure system responds to gravity to create a perfusion gradient from top to bottom of the lung (Figure 1.6). This is steeper than the ventilation gradient because of the density of blood. The perfusion gradient is represented by the following zones (West, 1995):

- Zone I (non-dependent lung), where alveolar pressure exceeds pulmonary arterial pressure: capillaries are flattened and no blood flows
- Zone II (middle), where pulmonary arterial pressure exceeds alveolar pressure, which exceeds venous pressure
- Zone III (dependent lung), where venous pressure exceeds alveolar pressure.

There is no blood flow in zone I, which in health is small or non-existent, but in the apex of the upright lung, the vessels collapse easily if, for example, hypovolaemic shock reduces arterial pressure or mechanical ventilation increases alveolar pressure. In the base of the upright lung, where zone III predominates, the pressure of blood may lead to airway closure.

Distribution of perfusion is also affected by:

- lung volume: vessels are stretched in the hyperinflated state and compressed in low volume states
- position, e.g. perfusion is more uniform in prone than supine (Nyren, 1999)
- pathological change, e.g. alveolar destruction in
  perfusion than to ventilation.

Table 1.1 Shunt fractions with typical implications

Shunt (%)	Implications
5	Normal 'physiological shunt'
< 10	Shunt compatible with normal gas exchange
15	Typical first-day postoperative shunt
15	Lung collapse
15–20	Elderly person with lung disease
20	Persistent hypoxaemia despite F_IO_2 of 1.0
> 30	Significant cardiopulmonary support required
> 50	Critically ill patient

F_IO_2 = fraction of inspired oxygen

VENTILATION/PERFUSION RELATIONSHIPS

It is no good having a well-ventilated alveolus if it is not supplied with blood, or a well-perfused alveolus that is not ventilated. Fresh air and blood need to be in the same place at the same time for gas exchange to occur. The matching of these two essentials is expressed as the ratio of alveolar ventilation to perfusion ($\dot{V}_A/\dot{Q}$).

$\dot{V}_A/\dot{Q}$ matching varies within the normal lung. In the upright lung, the base receives 18 times more blood and 3.5 times more gas than the non-dependent apices (Thomas, 1997). A degree of $\dot{V}_A/\dot{Q}$ mismatch can be due to either a high or low $\dot{V}_A/\dot{Q}$ ratio. A low ratio means that lung is perfused but not adequately ventilated. This creates a shunt, defined as the fraction of cardiac output that is not exposed to gas exchange in the pulmonary capillary bed. Shunt is measured by comparing arterial and mixed venous blood (p. 329), expressed as % of cardiac output. A small shunt is normal because part of the bronchial circulation mingles with pulmonary venous drainage (Table 1.1).

The mixing of shunted venous blood with oxygenated blood is known as venous admixture, normally 5% of cardiac output.

Systemic hypoxia stimulates selective vasodilation to assist perfusion of vital tissues. Pulmonary hypoxia stimulates the opposite response. If a fall in alveolar PO_2 is detected in the pulmonary circulation, an ingenious mechanism called hypoxic vasoconstriction helps maintain gas exchange. Pulmonary hypoxia causes increased tone in the muscles of adjacent arteriolar walls, constricting the arterioles, limiting wasted perfusion and improving $\dot{V}_A/\dot{Q}$ distribution. When the lung bases are affected, e.g. in the early stages of COPD or pulmonary oedema, local shutdown of vessels forces blood to the better ventilated upper regions, shown on X-ray as upper lobe diversion (p. 49). Hypoxic vasoconstriction becomes counterproductive when alveolar hypoxia occurs throughout the lung, as in advanced COPD, when generalized vasoconstriction causes pulmonary hypertension.

> ***We breathe to ventilate and ventilate to respire.***
>
> Tobin 1991

ARTERIAL BLOOD GASES

PO_2

- partial pressure or tension of oxygen.

P_aO_2

- partial pressure of oxygen in arterial blood, i.e. oxygen dissolved in plasma
- normal: 11–14 kPa (80–100 mmHg).

S_aO_2

- extent to which haemoglobin in arterial blood is saturated with oxygen, i.e. capacity of blood to carry oxygen
- normal: 95–98%.

Oxygen content

- total amount of oxygen in blood, i.e. oxygen in both plasma and haemoglobin.

P_aCO_2
- partial pressure of CO_2 in arterial blood
- the basis of respiratory acid–base balance
- normal: 4.7–6.0 kPa (35–45 mmHg).

Hypoxaemia
- reduced oxygen in arterial blood
- P_aO_2 < 8 kPa (60 mmHg) or S_aO_2 < 90%.

Hypoxia
- reduced oxygen at tissue level
- final common pathway of the cardiorespiratory system, more relevant to body function than hypoxaemia but more difficult to measure.

Hypocapnia/hypocarbia
- reduced CO_2 in arterial blood.

Hypercapnia/hypercarbia
- increased CO_2 in arterial blood.

F_IO_2
- fraction of inspired oxygen, e.g. F_IO_2 of 0.6 = 60% inspired oxygen.

Arterial blood gas measurements give an indication of ventilation, gas exchange and acid–base status. Readings should be related to previous values, the clinical state of the patient and the level of inspired oxygen (F_IO_2). Arterial blood samples are taken either by intermittent puncture of the radial artery using local anaesthesia, from an indwelling arterial catheter, or by using arterialized capillary blood from the earlobe (Dar, 1995).

Neither oxygen *tension* nor *saturation* tell exactly how much oxygen is being carried in blood. P_aO_2 describes only the 3% of oxygen dissolved in plasma. It determines the extent to which haemoglobin (Hb) can be saturated with oxygen and reflects the pressure needed to push oxygen from blood into tissue cells. S_aO_2 describes the 97% of oxygen that is bound to Hb. An anaemic person may have a normal S_aO_2 but deliver a subnormal load of oxygen. Only oxygen *content* describes the total amount of oxygen carried in the blood, and incorporates P_aO_2, S_aO_2 and Hb. In practice, oxygen content is assumed from P_aO_2 or S_aO_2. None of these terms give a measure of oxygenation at tissue level, and resting P_aO_2 does not reflect P_aO_2 during exercise, nor predict accurately the nocturnal P_aO_2 (Hodgkin *et al.*, 1993, p. 66).

Oxygen dissociation curve

The relationship between S_aO_2 and P_aO_2 is expressed by the oxygen dissociation curve, which represents the normal variation in the amount of oxygen that combines with Hb. Its peculiar shape illustrates the protective mechanisms that function in both health and disease (Figure 1.8). An understanding of the curve helps to complete the physiological picture and can be used, with oximetry, to assist respiratory assessment when blood gases are not available (Goodfellow, 1997).

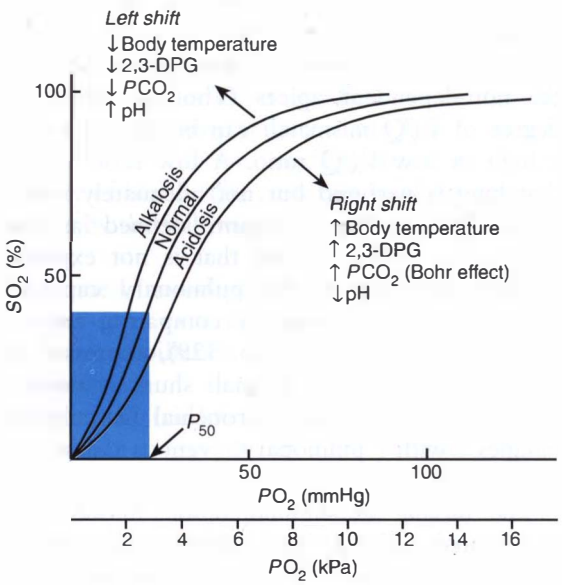


Figure 1.8 Oxygen dissociation curve relating oxygen saturation to oxygen tension. 2,3-DPG is an enzyme in red blood cells, increased in chronic hypoxaemia, which allows easier unloading of O_2 to hypoxic tissues. P_{50} is the P_aO_2 at which Hb is 50% saturated with oxygen. It is the most sensitive indicator of a shift in the curve because the middle portion of the curve displaces to a greater degree than either end. High value suggests poor affinity of Hb for oxygen. Shaded area represents critical tissue hypoxia.

Upper flat portion of the curve

At the plateau of the curve, the combination of oxygen with Hb is favoured by a high PO_2, and its stability is not unduly disturbed by changes in P_aO_2. In health, this encourages loading of oxygen in the high PO_2 environment of the lung, and discourages unloading of oxygen before blood reaches the capillary bed. In disease, a drop of P_aO_2 to 10.7 kPa (80 mmHg) hardly affects the amount of oxygen in the blood.

Haemoglobin cannot be more than fully saturated, and hyperventilation cannot supersaturate arterial blood supplied by functioning alveoli to compensate for hypoxaemia resulting from poorly functioning alveoli.

Steep portion of the curve

The dissociation of Hb becomes proportionately greater as PO_2 falls, so that small changes in P_aO_2 greatly affect S_aO_2. In health, this means that Hb can offload quantities of oxygen at cellular level with maintenance of oxygen tension in the blood. In disease, large amounts of oxygen can be unloaded when tissues are hypoxic. A P_aO_2 of 7.3 kPa (55 mmHg) marks the point where a significant reduction in oxygen delivery to the tissues begins, and further small drops in P_aO_2 result in tissue hypoxia.

Shift of the curve

Another singular way in which the body responds to need is to adjust the affinity of Hb for oxygen, as reflected by a shift of the curve. A right shift means that Hb unloads oxygen more easily at a given PO_2. In health, this occurs during exercise, when active muscle generates heat and makes blood hypercapnic and acidic. In disease, this occurs with fever and when tissues need extra oxygen. A left shift occurs when Hb holds tightly on to its oxygen, as occurs in hyperventilation, hypometabolism or a cold environment. Pink ears and noses on frosty mornings are due to the reluctance of Hb to unload oxygen.

Hypoxia and hypoxaemia

Causes

Causes of hypoxia are:

- hypoxaemia
- reduced CO, e.g. myocardial infarct
- reduced oxygen carrying capacity of the blood, e.g. anaemia, sickle cell disease
- reduced blood flow, e.g. haemorrhage, peripheral vascular disease
- disrupted blood flow, e.g. multisystem failure
- reduced ability of tissues to extract oxygen, e.g. septic shock.

Causes of hypoxaemia are:

- low $\dot{V}_A/\dot{Q}$ ratio due to wasted perfusion (↑ shunt)
- high $\dot{V}_A/\dot{Q}$ ratio due to wasted ventilation (↑ dead space)
- hypoventilation
- diffusion abnormality
- F_IO_2, e.g. fire entrapment, high altitude, inadequate oxygen therapy.

Wasted perfusion occurs when blood is shunted through consolidated, collapsed or damaged lung without picking up oxygen, leading to $\dot{V}_A/\dot{Q}$ mismatch (Figure 1.9), somewhat attenuated by hypoxic vasoconstriction. Hypoxaemia associated with shunt shows limited response to oxygen therapy because added oxygen cannot reach the shunted blood.

Wasted ventilation occurs when a perfusion defect such as pulmonary embolism prevents fresh gas from reaching arterial blood. This increases alveolar dead space and causes $\dot{V}_A/\dot{Q}$ mismatch at the other end of the spectrum (Figure 1.9).

Diffusion abnormalities occur in disorders such as pulmonary oedema or fibrosing alveolitis.

Hypoventilation leads to a fall in P_aO_2 that is roughly equivalent to the increase in P_aCO_2. It can be distinguished from other causes of hypoxaemia by the $P_{A-a}O_2$ (see Glossary).

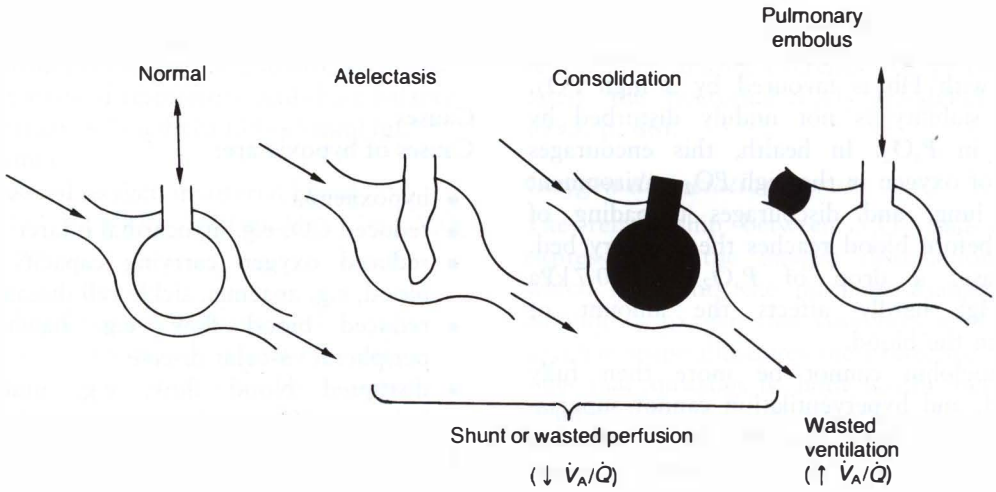


Figure 1.9 Alveoli and surrounding capillary network, showing how impaired ventilation or perfusion can upset $\dot{V}_A/\dot{Q}$ balance.

Effects

Prolonged or repetitive hypoxaemia is thought to be worse than a single episode (Hanning, 1995). The brain is exquisitely sensitive to ischaemia and typically responds to hypoxia as follows:

P_aO_2 < 7.3 kPa (55 mmHg): memory defect, impaired judgement
< 5.3 kPa (40 mmHg): tissue damage
< 4 kPa (30 mmHg): unconsciousness
< 2.7 kPa (20 mmHg): death.

The gut lining and kidney are also sensitive to hypoxia, which can be identified by monitoring their responses. Gut monitoring (p. 330) is in its infancy. Kidney failure is identified by reduced urine output and increased potassium, creatinine or urea. Table 1.2 shows how the body responds to hypoxaemia and hypercapnia.

The circulatory response to acute hypoxia is to increase CO and improve blood flow to the brain, respiratory muscles and liver, at the expense of reduced flow to gut, skin and bone (Kuwahira, 1993). Significant cardiac arrhythmias can occur when S_aO_2 drops below 80% (RCP, 1999).

Hypercapnia reflects hypoventilation due to respiratory depression, severe weakness, fatigue

Table 1.2 Clinical features of hypoxaemia and hypercapnia

Hypoxaemia	Hypercapnia
Cyanosis	Flapping tremor of hands
Tachypnoea	Tachypnoea
Tachycardia → arrhythmias/bradycardia	Tachycardia → bradycardia
Peripheral vasoconstriction	Peripheral vasodilation leading to warm hands and headache
Respiratory muscle weakness	Respiratory muscle weakness
Restlessness → confusion → coma	Drowsiness → hallucinations → coma
	Sweating

Tachycardia = rapid heart rate; tachypnoea = rapid respiratory rate

or an attempt to avoid fatigue by reducing ventilation and inspiratory muscle overload (Green and Moxham, 1993). Both hypoxaemia and hypercapnia impair endurance of the diaphragm (Tobin, 1988).

Interpretation

P_aO_2 is affected by one or a combination of the causes of hypoxaemia. P_aCO_2 is affected only by ventilation because CO_2 is freely diffusible and not affected by $\dot{V}_A/\dot{Q}$ mismatch. P_aCO_2 is therefore used to assess ventilatory adequacy.

Examples of blood gas abnormalities are:

- $\downarrow P_aO_2$ with $\uparrow P_aCO_2$, i.e. hypoxaemia with hypercapnia: exacerbation of lung disease in a patient who is unable to ventilate adequately
- $\downarrow P_aO_2$ with $\downarrow P_aCO_2$, i.e. hypoxaemia with hypocapnia: exacerbation of disease in a patient who is breathing rapidly, e.g. pneumonia, fibrosing alveolitis, pulmonary oedema, pulmonary embolus
- normal P_aO_2 with $\downarrow P_aCO_2$: emotion, hyperventilation syndrome, painful arterial puncture or any cause of hyperventilation.

The above examples could, in reverse order, represent a developing asthma attack.

If we reduce our minute ventilation, P_aCO_2 rises and P_aO_2 falls, but the reverse is not true. Increased ventilation will blow off P_aCO_2 but P_aO_2 is maintained because Hb cannot be supersaturated.

Acid–base balance

The pH reflects acid–base balance. It responds to metabolic and respiratory change but cannot differentiate between them. Body functions occur optimally at a pH of 7.35–7.45.

Bicarbonate ion concentration (HCO_3^-) measures metabolic acid–base balance.

- normal: 22–26 mmol/L
- metabolic acidosis: < 22 mmol/L
- metabolic alkalosis: > 26 mmol/L.

Base excess (BE) is the quantity of strong acid or base required to restore pH to normal. It measures metabolic acid–base balance but takes buffering of red blood cells into account. It therefore provides more complete analysis of metabolic buffering than HCO_3^-, which accounts for only half the buffering capacity of blood. BE is calculated from pH, P_aCO_2 and haematocrit.

- normal: minus 2 to plus 2 mmol/L
- metabolic acidosis: < –2 mmol/L
- metabolic alkalosis: > 2 mmol/L.

Regulation

Acid–base balance is disturbed if removal of CO_2 from the lungs is abnormal (respiratory acidosis or alkalosis) or production of acid from the tissues or elimination elsewhere is abnormal (metabolic acidosis or alkalosis).

Body cells and chemical reactions are acutely sensitive to the pH of their environment, and any deviation from the normal slight alkalinity of body fluids is fiercely resisted, at whatever cost, by three homeostatic mechanisms. These work to dispose of the acids that are continually produced by the body's metabolic processes, mostly by the interaction of CO_2 and water to create carbonic acid.

1. The buffer system acts as a chemical sponge, which neutralizes acids or bases by means of reactions that give up or absorb hydrogen ions, all within seconds. The bicarbonate base–buffer equation depends on the dissociation of carbonic acid in solution, acting as a sink for hydrogen ions:

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-.$$

An increase in P_aCO_2 shifts this equilibrium to the right, increasing H^+ and causing respiratory acidosis. A decrease in P_aCO_2 shifts the equation to the left, decreasing H^+ and causing respiratory alkalosis.

Standard bicarbonate may be used in order to eliminate the influence of acute changes in P_aCO_2. The measurement is adjusted as if P_aCO_2 were valued at a standard 5.3 kPa, and allows evaluation of the purely metabolic component.

Standard bicarbonate is similar to bicarbonate in a person with normal acid–base status.

2. If buffering is not adequate, the lungs then present an avenue for regulating CO_2. Hyper- or hypoventilation can stabilize the acid–base balance within 1–15 minutes.

3. If this is still not adequate, the kidneys then begin to eliminate acid, but take up to 3 days to normalize pH. Bicarbonate or base excess indicates the extent of renal compensation and quantify the metabolic component of an acid–base disturbance.

Interpretation

Step 1: look at pH:

- ↓ pH means acidosis
- ↑ pH means alkalosis.

Step 2: look at P_aCO_2: does it account for the abnormal pH?

- ↑ P_aCO_2 means respiratory acidosis
- ↓ P_aCO_2 means respiratory alkalosis.

Step 3: look at HCO_3^-: does it account for the abnormal pH?

- ↑ HCO_3^- or BE means metabolic alkalosis
- ↓ HCO_3^- or BE means metabolic acidosis.

A change in pH due to respiratory or metabolic disturbance is usually offset by a compensatory change in the other system so that pH normalizes. Respiratory compensation is quicker than metabolic compensation.

When pH is restored to normal, full compensation has occurred. The stages can be identified as follows:

- Abnormal pH + change in P_aCO_2 or bicarbonate/BE = non-compensation, i.e. a recent process
- Abnormal pH + change in P_aCO_2 and bicarbonate/BE = partial compensation
- Normal pH + change in P_aCO_2 and bicarbonate/BE = full compensation.

Respiratory and metabolic factors are often combined, and complex interactions can occur. If pH is below 7.2, assessment for mechanical assistance is mandatory.

Table 1.3 clarifies the causes, effects and recognition of arterial blood gas imbalances. Table 1.4 gives examples. Table 1.5 shows how two respiratory disorders can affect arterial blood gas readings.

Table 1.3 Interpretation of arterial blood gas trends

Condition	Causes	Effects	Recognition
Acute respiratory acidosis	Hypoventilation, e.g. exhaustion, weakness	↑ PCO_2, ↓ pH, normal HCO_3^- (no time for renal compensation)	Shallow breathing, slow breathing, drowsiness
Chronic (compensated) respiratory acidosis	Chronic hypoventilation	↑ PCO_2, normal pH, ↑ HCO_3^-, BE > 2 (conservation of HCO_3 to restore pH)	Chronic severe respiratory disease, e.g. COPD
Respiratory alkalosis	Acute hyperventilation, e.g. excess mechanical ventilation, anxiety, pain, acute asthma	↓ PCO_2, ↑ pH, ↓ HCO_3^-, BE> 2 (renal excretion of HCO_3)	Breathlessness, hyperventilation, distressed breathing pattern
Metabolic acidosis	Ketoacidosis, e.g. diabetes; loss of alkali, e.g. diarrhoea; renal failure	↓ PCO_2, ↓ pH, ↓ HCO_3^-, BE < –2 (respiratory compensation to blow off PCO_2)	Hyperventilation
Metabolic alkalosis	Volume depletion; diuretics; removal of acid, e.g. vomiting	↑ PCO_2, ↑ pH, ↑ HCO_3^-, BE > 2 (renal excretion of HCO_3^-)	Delirium

Note that if the primary problem is metabolic, pH and bicarbonate/BE change in the same direction, while if the primary problem is respiratory, pH and P_aCO_2 change in opposite directions.

Table 1.4 Examples of acid–base interpretation

1. pH 7.3 P_aCO_2 6.5 kPa (49 mmHg) HCO_3^- 30 mmol/L	Partially compensated respiratory acidosis, since both P_aCO_2 and HCO_3^- are increased but pH is low
2. pH 7.5 P_aCO_2 4 kPa (30 mmHg) HCO_3^- 19 mmol/L	Partially compensated respiratory alkalosis, since both P_aCO_2 and HCO_3^- are decreased but pH is high
3. pH 7.48 P_aCO_2 6.0 kPa (45 mmHg) HCO_3^- 30 mmol/L	Uncompensated metabolic alkalosis, since both HCO_3^- and pH are high but P_aCO_2 has barely moved
4. pH 7.45 P_aCO_2 6.5 kPa (34 mmHg) HCO_3^- 20 mmol/L	Fully compensated respiratory alkalosis

Table 1.5 Arterial blood gas responses to two disorders (numbers in brackets indicate mmHg)

	Normal	Acute asthma	COPD
P_aO_2	12.7 (95)	9.3 (70)	7.3 (55)
P_aCO_2	5.3 (40)	3.3 (25)	8 (60)
pH	7.4	7.5	7.4
HCO_3^-	24	24	29

Both disorders show hypoxaemia. P_aCO_2 values reflect breathlessness in acute asthma and hypoventilation in COPD. pH and HCO_3^- values reflect an acute non-compensated condition in acute asthma and full compensation in COPD.

THE OXYGEN CASCADE (Figure 1.10)

The *raison d'être* of the cardiorespiratory system is to get oxygen to the tissues. Even if ventilation, diffusion and perfusion are in order, oxygen still has to reach and enter the tissues. Oxygen **transport** is the passage of oxygen to the tissues. This term is often used synonymously with, and is virtually the same as, oxygen **delivery**, which is the oxygen presented to the tissues. Tissue oxygenation depends on the oxygen content of blood, CO, haemoglobin levels and local perfusion. Oxygen **consumption** (uptake) by the tissues is roughly equivalent to oxygen demand, determined by the metabolic need of the tissues for oxygen.

Tissue oxygenation is determined by a balance between supply (oxygen delivery or DO_2) and demand (oxygen consumption or $\dot{V}O_2$). The respiratory system, like other systems, has reserve capacity, and DO_2 is normally three or four times greater than $\dot{V}O_2$ (Epstein and Henning, 1993).

Oxygen availability to the tissues depends on:

- oxygen content
- cardiac output
- distribution of CO
- oxygen dissociation curve.

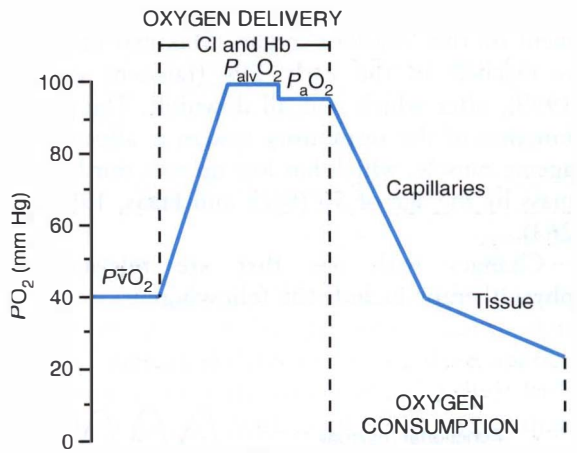


Figure 1.10 The oxygen cascade, representing the journey of oxygen through the body. $P\bar{v}O_2$, $P_{alv}O_2$ and P_aO_2 are the partial pressures of oxygen in the pulmonary artery, alveoli and arteries respectively. PO_2 is reduced in the capillaries as it is extracted by the tissues and further reduced in the tissues as it is consumed. Cl = oxygen content. See Appendix F for conversion of mmHg to kPa. (From Tobin, M. J. (1994) *Principles and Practice of Mechanical Ventilation*, McGraw-Hill, New York, with permission.)

$\dot{V}O_2$ varies with metabolic rate. An increase in $\dot{V}O_2$ is usually met without difficulty by increased DO_2 (mostly through a rise in CO, partly through increased minute ventilation) and increased oxygen extraction by the tissues. Once maximum oxygen extraction is reached, further increases in demand, or falls in supply, lead to hypoxia.

Critically ill patients with sepsis can demand 50–60% extra oxygen, while patients with multiple trauma, septic shock or burns may need 100% extra oxygen (Epstein and Henning, 1993). If the body is not able to transport, deliver, extract and utilize this oxygen, sustained lactic acidosis occurs.

Compared to gas exchange in the lung, which is easily monitored in arterial blood, tissue oxygenation has to be measured from the pulmonary artery, which contains the only reserves of oxygen in the body (Ahrens, 1999a).

EFFECT OF AGEING

The gas exchange function of the ageing lung is affected by the cumulative effect of the environment on this 'outdoor' organ. Maximal function is reached in the early 20s (Janssens *et al.*, 1999), after which it is all downhill. The pump function of the respiratory system is affected by ageing muscle, which has lost up to a third of its mass by the age of 50 (Bach and Haas, 1996, p. 263).

Changes with age that are relevant to physiotherapy include the following:

- ↓ elastic recoil, dilation of alveoli, ↑ lung volume ('senile emphysema'), leading to reduced surface area for gas exchange (Janssens *et al.*, 1999)
- narrowing of small airways, leading to raised closing volume (Figure 1.11), premature closure of small airways, alveolar collapse and $\dot{V}_A/\dot{Q}$ mismatch
- ↑ residual volume because closure of small airways prevents full exhalation (this appears as hyperinflation on X-ray, which can be misinterpreted as emphysema)
- greater dependence on collateral ventilation because of airway closure
- ↓ diffusion, leading to ↑ $P_{A-a}O_2$
- ↓ respiratory muscle strength, strongly correlated with nutritional status (Janssens *et al.*, 1999) and sedentary lifestyle
- ↓ vital capacity by 30 mL per year (Bach and Haas, 1996)
- ↓ FEV_1 by 30 mL/year (45 mL/year in smokers) (Fehrenbach, 1998), and ↓ response to β_2-agonist drugs such as salbutamol (Connolly, 1995)
- ↓ exercise capacity by an average 10% per decade (Hellman, 1994)
- ↓ chest wall compliance
- ↓ ventilatory response to both hypoxaemia and hypercapnia (Janssens *et al.*, 1999)
- ↓ total blood volume, which impairs circulatory function (Davy and Seals, 1994)
- postural hypotension
- prolonged reaction times, ↓ coordination (Laporte *et al.*, 1999)

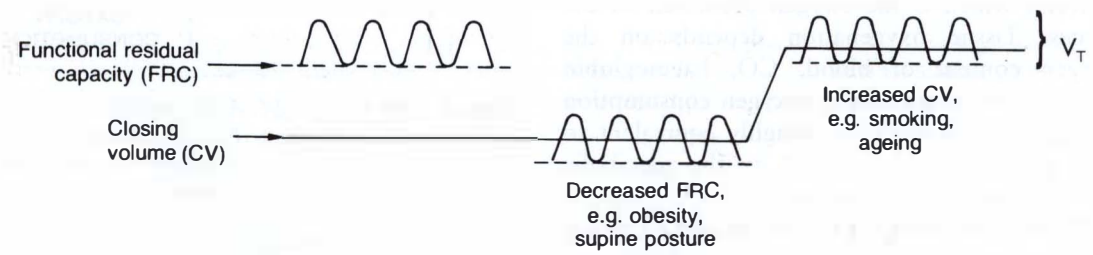


Figure 1.11 Factors that shift tidal breathing into the closing volume range, leading to airway closure in the lung bases during quiet breathing.

- ↑ blood pressure (BP), especially systolic (Hellman, 1994), which helps maintain tissue perfusion because a greater pressure is needed to overcome the resistance of hardening arteries.

Clinical implications

It is necessary to take time when assisting elderly patients out of bed, in case of postural hypotension. During deep breathing, collateral ventilation can be exploited with an end-inspiratory hold (p. 153). During exercise training, an ageing cardiovascular system is less able to adapt to the stress of exercise. During weaning from mechanical ventilation, extra help is needed because lung volume is especially compromised by the supine posture and low tidal volumes.

EFFECT OF OBESITY

Obesity: weight 20% greater than ideal body weight
Morbid obesity: weight 100% greater than ideal body weight
Malignant obesity: weight 200% greater than ideal body weight.

Obesity is the commonest chronic disease in the USA (Guernelli *et al.*, 1999), and Britain is catching up. The obese and the elderly share a tendency towards poor basal ventilation. Obesity reduces lung volumes (Carella, 1999 and Figures 1.11 and 1.12) and lung compliance (Jenkins and Moxham, 1991). The normal downward ventilation gradient is obliterated or reversed because of compression from the abdomen (Hurewitz, 1985), leading to reduced ventilation in the well-perfused bases, $\dot{V}_A/\dot{Q}$ mismatch and some hypoxaemia. Hypercapnia is also a risk (Bégin, 1991). Exercise demands high oxygen consumption. Breathing patterns tend to be rapid, shallow and apical. Morbidity and mortality are increased by cardiovascular, pulmonary, metabolic and sleep abnormalities (Carella, 1999).

Morbid obesity threatens body functions, leading to increased risk of respiratory disease,

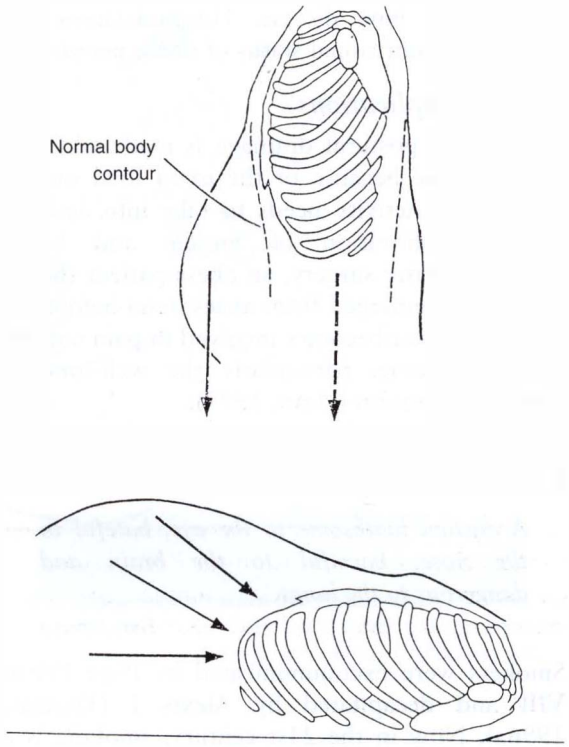


Figure 1.12 Effect of obesity on the mechanics of breathing. When upright, the weight of the viscera (dotted arrow) is normally borne by the pelvis but in obese people it pulls down on the ribs (solid arrow) and increases the work of inspiration . When supine, the pressure of the viscera on the diaphragm hinders inspiration. (From Wilkins, R. L., Sheldon, R. L. and Krider, S. J. (1995) *Clinical Assessment in Respiratory Care*, Mosby, Toronto, p. 350.)

cardiovascular disease, diabetes, digestive disease (Chen *et al.*, 1993), gallstones, gout, skin disease, musculoskeletal problems, sleep apnoea and some cancers (Guernelli *et al.*, 1999). Risk of sudden cardiac death is 40 times greater than normal (Guernelli *et al.*, 1999). Functioning lung volume may be reduced by half during surgery, compared to a 20% reduction in non-obese people (Wahba, 1991). During surgery, position-related complications are above average and are not reduced by increased tidal volume or PEEP (p. 351) (Buckley, 1997).

Obesity does not ensure good nutrition because inactivity and steroid medication are

common in lung disease. Hospitalization can worsen the nutritional status of obese people.

Clinical implications

Head-down postural drainage is inadvisable for obese people because of the extra load on the diaphragm. Activity needs to take into account the fat infiltration of muscle and heavy workload. After surgery, an obese patient should barely have emerged from anaesthesia before the physiotherapist becomes involved in pain control and positioning, particularly the well-forward side-lying position (Dean, 1997).

EFFECT OF SMOKING

> ***A custom loathsome to the eye, hateful to the nose, harmful to the brain and dangerous to the lungs.***
>
> King James I

Smokers were excommunicated by Pope Urban VIII and decapitated by Alexis I (Thomas, 1996a). Now in the 21st century, smoking is a form of legal drug addiction and the main preventable cause of premature death (Balfour, 1993). It is escalating most in the developing world, where cigarettes tend to have a higher tar and nicotine content (Panos, 1994). Smoking kills half of all persistent smokers worldwide, including one person every 5 minutes in the UK (Venables, 1994). This comes as no surprise considering the 6000 chemicals in tobacco smoke (Hoozen, 1997), including cyanide, butane, ammonia, carbon monoxide and 50 known carcinogens (Kritz, 1995).

Carnage to the respiratory and cardiovascular systems is well-known (Figure 1.13) but virtually every organ system is affected (British Medical Journal, 1997). The cumulative effect is shown in Figure 1.14 and the litany of destruction is outlined below.

- Smoking worsens outcome in rheumatoid arthritis (Saag, 1997) and ankylosing spondylitis (Averns, 1996); is associated with low back pain and widespread musculoskeletal pain (Andersson, 1998); accelerates ageing

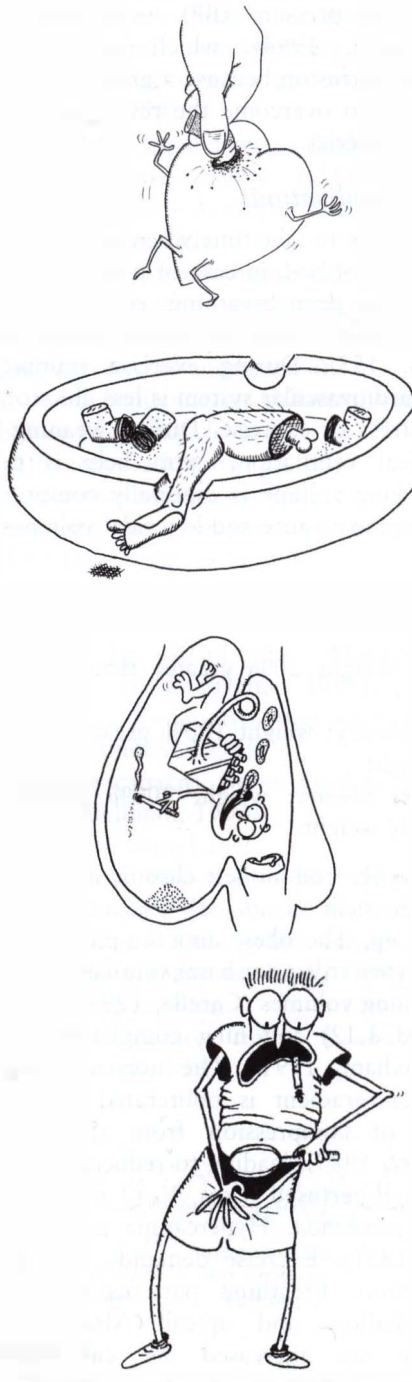


Figure 1.13 Effect of smoking on the heart, vascular system, foetus and potency. (From Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford, with permission. Artist: James Northfield)

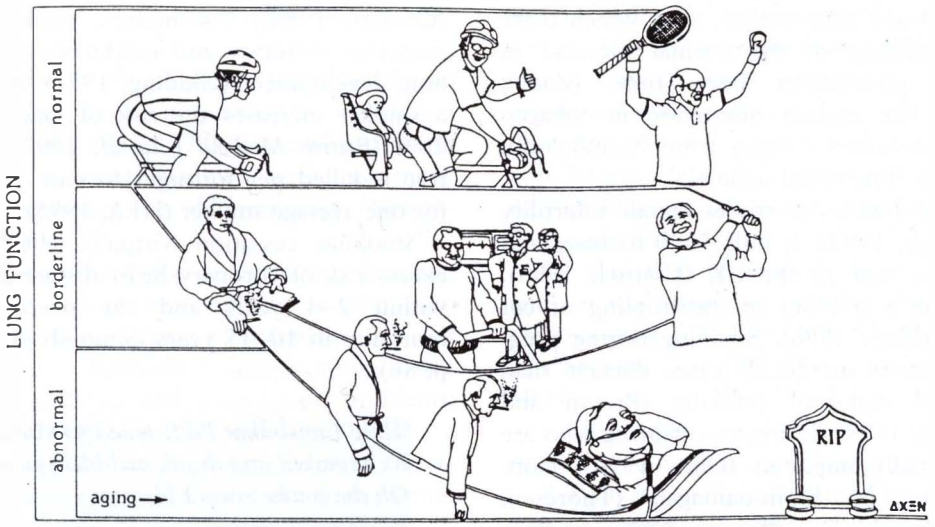


Figure 1.14 Long-term effects of smoking. Top: Lifelong non-smoker continuing with active life. Middle: Smoker recovering some lung function with smoking cessation and rehabilitation. Bottom: Continuous smoker faces loss of function and premature death. (From Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester, with permission.)

(Kauffmann, 1993); doubles the risk of dementia (Ott *et al.*, 1998); depletes vitamin C by 30% (Strachan, 1991); ulcerates the gut (Thomas, 1996a); dislodges teeth (Jette, 1993); causes cataract (Christen, 1992), glue ear (Couriel, 1994) and squint in children (Medical Monitor, 1992); demineralizes bone (Prescott, 1998); depletes antioxidants (Li, 1996); causes more bronchial hyperreactivity than cocaine (Tashkin *et al.*, 1993); increases the risk of diabetes (Rimm, 1995), head and neck cancer (Koufman and Burke, 1997) and breast cancer (Bennicke, 1995); causes 87% of deaths from lung cancer (Dresler, 1996); and increases the risk of postoperative complications two to six times (Bluman *et al.*, 1998), macular degeneration two to three times (Christen, 1996), subarachnoid haemorrhage sixfold (Partridge, 1992) and pneumothorax 13-fold (Light, 1993). Smoking weakens the immune system; damages cilia (Verra, 1995) and surfactant (Pearce, 1984); and leads to hypertension, reduced exercise tolerance (Gidding, 1994), anxiety and depression (Jorm *et al.*, 1999).

- Nicotine is the ingredient that imprisons smokers in the habit. It is more addictive than heroin, seven times as addictive as alcohol (Haas and Haas, 1990, p. 67) and is delivered to the central nervous system within seven seconds (Fisher *et al.*, 1990). It initially stimulates the brain, then acts as a sedative. The one redeeming feature of nicotine is that it is reported to ameliorate ulcerative colitis in the active phase (Thomas, 1996a), and nicotine patches have been advised.
- Smoking increases bronchial secretions while reducing mucociliary clearance (Bluman *et al.*, 1998) and causes high closing volumes and mismatched $\dot{V}_A/\dot{Q}$ (Figure 1.11). Smoking increases the risk of pneumonia (Almirall *et al.*, 1999). Premature closure of small airways occurs before the onset of symptoms or lung

function test abnormality, after which there is a doubling of the normal increase in airflow obstruction over time (Zadai, 1991). The carbon monoxide in tobacco smoke dislodges oxygen from haemoglobin to create 'functional anaemia'.

- Smoking doubles or triples female infertility (Partridge, 1992). It kills 5000 foetuses and infants a year in the UK (Couriel, 1994), including a trebling or quadrupling of cot deaths (Blair, 1996). Smoking during pregnancy causes marginally more damage than postnatal maternal smoking (Brown and Halonen, 1999). It creates offspring who are intellectually impaired (Olds, 1994), more likely to be brain-damaged (Thoresen, 1999), hypertensive (Beratis, 1996), smaller, slower growing and with increased respiratory and allergic disease throughout life (Partridge, 1992). The low birthweight is associated with greater mortality up to the teenage years (Power and Li, 2000). Even grandchildren do not escape, mothers born to women who smoked during pregnancy being more likely to have a miscarriage (Golding, 1994). One cigarette a week can cause menstrual problems (Charlton and White, 1996) and, because smoking lowers oestrogen levels, it creates early menopause and brings postmenopausal women's risk of cardiovascular disorder closer to that of men (Prescott, 1998).
- Smoking is neither virile nor sexy. Smoking damages sperm, and 15% of all childhood cancers have been attributed to paternal smoking (Sorahan, 1997). Most smokers also have breath that smells like an ashtray.
- Smoking exacerbates the poverty of those on the lowest incomes (Smeeth, 1998).

Passive smoking creates lung carcinogens in the recipient within hours (Hecht, 1993), retards foetal growth, increases age-related hearing loss (Cruickshanks, 1998) and increases risk of coronary heart disease by 70% (Brannon *et al.*, 1998, p. 388) and risk of asthma by 50% (Coultas, 1998). For infants, passive smoking increases mortality and morbidity, and impairs lung development (Gidding, 1994). Marriage to a smoker increases the risk of lung cancer by 26% (*British Medical Journal*, 1997). And one tree is killed per fortnight to cure the tobacco for one average smoker (HEA, 1995).

Smoking cessation virtually eliminates the excess risk of coronary heart disease and stroke within 2–4 years and the overall risk of mortality in 10–15 years (Simonds *et al.*, 1996, p. 86).

> ***'How I wish that I'd listened to Mum***
> ***As I smoked and drank and blew gum.***
> ***Oh the smoke rings I blew***
> ***But if only I knew***
> ***That the moment of reckoning would come.'***
>
> Barton, 2000 (just before dying while awaiting a lung transplant)

Clinical implications

Motivate, educate and cajole.

EFFECT OF PREGNANCY

Pregnancy requires a 20% increase in oxygen consumption to service the extra metabolism. Demand is met by a 40–50% increase in minute ventilation (MV), which lowers P_aCO_2 and causes mild respiratory alkalosis. The swelling uterus restricts resting lung volume, but vital capacity is maintained at the cost of increased work of breathing. Three-quarters of pregnant women experience breathlessness (Nelson-Piercy, 1996).

Clinical implications

Patients on bedrest are at risk of loss of lung volume and will need monitoring of their chest and attention to positioning. Those beyond 20 weeks gestation should not be nursed supine in case of aortocaval occlusion which could compromise mother and baby (Bird, 1997). For patients whose respiratory system is already

compromised, the late stages of pregnancy may require other measures; for example, kyphoscoliotic patients with nocturnal hypoventilation may benefit from non-invasive ventilation (Restrick *et al.*, 1997). The course of asthma in pregnancy is unpredictable, with as many patients improving as deteriorating (Nelson-Piercy, 1996).

The commonest cause of obstetric admission to intensive care is pre-eclampsia or eclampsia, which is the gravest form of pregnancy-induced hypertension. Relevant complications are pulmonary oedema and coagulation problems, but physiotherapy is not indicated unless a seizure causes aspiration. Most obstetric admissions to the intensive care unit are post-partum but, for pregnant patients, a caesarean section pack must be available.

EFFECT OF EXERCISE

> ***Those who think they have not time for bodily exercise will sooner or later have to find time for illness.***
>
> Edward Stanley, Earl of Derby, 1826–93

During exercise, oxygen delivery, consumption and extraction increase. Extra oxygen is delivered to the heart and skeletal muscles by several mechanisms.

1. Ventilation can increase from 6 L/min to 200 L/min (Salazar, 1991). During low-intensity exercise, deeper breathing makes the largest contribution to MV, while at high intensity, rapid breathing is the main contributor.

2. CO can increase fourfold in an unconditioned young adult and up to sixfold in a fit male (Epstein and Henning, 1993), mostly as a result of increased heart rate. Systolic BP increases in proportion to oxygen consumption and may reach over 200 mmHg in a healthy man. Diastolic pressure increases slightly during isotonic exercise and significantly during isometric exercise.

3. Increased CO means a shorter transit time as blood rushes past the alveoli, but increased diffusing capacity ensures equilibrium (Dantzker, 1983). This might explain the excessive hypoxaemia seen in some exercising patients with interstitial lung disease, whose diffusion is impaired.

4. Metabolic acidosis may develop if buffering mechanisms are unable to cope with the extra CO_2 and lactic acid.

5. Vascular resistance drops precipitately and, in the lungs, previously closed capillaries are recruited and distended. Muscle blood flow can increase 25-fold (Epstein and Henning, 1993).

6. Dead space can drop from a third to a fifth of tidal volume (Bach and Haas, 1996, p. 248).

7. P_aO_2 is usually maintained because distribution of perfusion and $\dot{V}_A/\dot{Q}$ become more uniform and diffusion increases. Oxygen extraction by the tissues can increase 20-fold (Epstein and Henning, 1993).

8. pH is usually maintained because extra hydrogen ions stimulate the arterial chemoreceptors to increase ventilation.

9. Bronchodilation occurs so long as asthma is not present.

10. Mucus transport increases (Houtmeyers, 1999).

11. Work of breathing increases because high flow rates increase turbulence and active expiration causes dynamic compression of airways. MV above 40 L/min is usually accompanied by mouth breathing.

12. Mouth breathing and raised MV increase the inhalation of pollutants. A marathon runner can inhale in 3 hours the same air and pollutants as a sedentary person in 2 days (Atkinson, 1997). This may be one factor precipitating exercise-induced asthma.

Cardiovascular delivery of oxygen to the peripheral muscles imposes the primary limit to exercise in normal subjects (Hsia, 1993). When blood flow becomes inadequate to maintain aerobic metabolism, the anaerobic threshold is reached, demand exceeds supply and lactic acidosis develops, with a disproportionate increase in MV relative to oxygen consumption.

Obstructive lung disease may cause a lower P_aO_2 with exercise. Restrictive disease can cause a precipitate drop in P_aO_2 (Wagner, 1992).

Inspiratory muscle fatigue may develop in people with reduced respiratory compliance. Subjectively, exercise can be experienced as incapacitating, joyful or somewhere in between.

The effects of exercise training are more related to cardiovascular and muscle metabolism than to the respiratory system.

The following have been described after regular physical activity:

- ↓ morbidity and mortality (Kerr, 1999)
- ↑ respiratory muscle strength (Ioli *et al.*, 1991)
- ↓ blood lactate levels for a given amount of exercise
- ↓ requirements for oxygen uptake, CO_2 output and ventilation for a similar degree of exercise
- ↑ maximum oxygen uptake, mainly due to ↑ maximum cardiac output
- ↓ resting heart rate
- ↓ hypertension, heart disease, diabetes, osteoporosis, some cancers, anxiety and depression (Powell and Pratt, 1996)
- ↑ glucose tolerance
- ↓ cigarette smoking (Todd, 1996)
- for a trained athlete, enlargement of the heart by up to 50% (Wilkins *et al.*, 1995)
- with swimming training, ↑ lung volumes (Gaultier and Crapo, 1997).

Exercise that is vigorous, regular and current reduces the risk of myocardial infarction by 50% (Todd, 1996).

Clinical implications

When supervising exercise, judgement is aimed at achieving optimum activity without losing the patient's co-operation or causing complications. Much encouragement is required to assist a patient towards a lifestyle of regular exercise.

EFFECT OF IMMOBILITY

Look at the patient lying long in bed.
What a pathetic picture he makes.
The blood clotting in his veins,
The lime draining from his bones,
The scybola stacking up in his colon,
The flesh rotting from his seat,
The urine leaking from his distended bladder,
And the spirit evaporating from his soul.

Asher, cited by Morris 1999

Times have changed since bed rest was considered 'the greatest advance of which practical medicine can boast in the last quarter century' (Playfair, 1881). Immobility is now known to increase the risk of pneumonia, deep vein thrombosis, osteoporosis and bedsores (Allen *et al.*, 1999), to reduce lung volume, cognition, co-ordination (Bach and Haas, 1996, p. 201) and lead to constipation, urine retention, deconditioning and depression (Mulley, 1993).

The acute stage of contractures begins immediately, especially in extension (Trudel *et al.*, 1999), although this is not significant for the average respiratory patient who is immobile for a few days. Disuse muscle atrophy is most marked in the first week, but subsequent loss is more than 10% per week (Dobson, 1993). Twenty days' bed rest can reduce work capacity by 30%, returning to normal only after 3 weeks of intensive exercise (Saltin *et al.*, 1968). Muscles lose 20% of their strength per week (Sciaky, 1994). Tendons and ligaments may take months to recover, and cartilage shows irreversible changes within a fortnight (Morris, 1999).

Loss of gravitational stimulus to the cardiovascular system causes a negative fluid balance within 24 hours and augments deconditioning. Reduced circulating blood volume and impaired vasoconstrictive ability cause postural hypotension, increased work of the heart and increased work of breathing (Dean and Ross, 1992). And far from being a treatment for chronic fatigue syndrome, bed rest creates its symptoms (Sharpe, 1998).

Deterioration occurs more rapidly in the respiratory and cardiovascular systems than the musculoskeletal systems, and recovery is slower than deterioration (Dean and Ross, 1992). The more immobile the patient, the higher the risk of developing respiratory complications and pressure sores.

Clinical implications

If immobility is caused by pain, fatigue or depression, these should be addressed, e.g. by analgesia, rest or a listening ear, so that they do not prevent mobilization. If immobility is unavoidable for medical reasons, regular position change reduces some of the complications of bed rest. Passive and/or active exercise are necessary, and encouragement of upright positions minimizes orthostatic intolerance.

EFFECT OF SLEEP

Sleep is restorative but, for some respiratory patients, risky. Changes during sleep include:

- ↓ mucociliary clearance (Houtmeyers, 1999)
- ↓ cough
- ↓ muscle tone, including muscles that preserve patency of the airway in the throat (McNicholas, 1997)
- dissociation of diaphragmatic from intercostal activity during rapid-eye-movement (REM) sleep (Mohsenin, 1994)
- for people whose respiratory system is already compromised, possible diaphragmatic fatigue
- ↓ ventilatory response to hypoxia and hypercapnia (McNicholas, 1997)
- ↓ MV by 10–15%, with consequent rise in P_aCO_2 of 0.4–1.1 kPa (Laursen, 1988)
- ↓ lung volumes (McNicholas, 1997)
- during REM sleep, 25% drop in tidal volume (Lumb, 2000, p. 346).
- $\dot{V}_A/\dot{Q}$ mismatch due to ↓ lung volumes and hypoventilation (Schenkel, 1996)
- for people with COPD, oxygen desaturation, which can be twice that experienced during exercise (McNicholas, 1997)
- bronchoconstriction, which is of little consequence except in people with asthma (Douglas, 1993)
- arrhythmias, variable heart rate and BP (Wilkins *et al.*, 1995, p. 356).

REM sleep occupies about 20% of total sleep time and is the restorative, dreaming and physiologically eventful phase when oxygen consumption is highest. It is also the time when changes are greatest and when respiratory patients are at their most vulnerable.

Sleep and COPD have a particular relationship. Nocturnal oxygen desaturation speeds pulmonary hypertension and hypercapnia (McNicholas, 1997), and sleep itself is disturbed by breathlessness and coughing. Sleep-disordered breathing is a risk for people with COPD, the elderly and the obese (Fletcher, 1992). Sleep also has a particular effect on asthma (Chapter 3).

Clinical implications

> *Sleep ...*
> *Balm of hurt minds, great Nature's second course,*
> *Chief nourisher in life's feast.*
>
> William Shakespeare, *Macbeth* II, 1

People on home oxygen should use it continuously during the night because the normal nocturnal dips in S_aO_2 can be damaging for people who are already chronically hypoxaemic. Hospitalized patients on oxygen should maintain this at night, sometimes with a higher flow rate. Certain postoperative patients may need nocturnal oxygen for a longer period than daytime oxygen (p. 250). Death from lung disease usually occurs at night.

EFFECT OF STRESS

All ill people suffer some degree of stress, usually as a result and sometimes as a predisposing factor of illness. Stress has adverse effects on the cardiovascular, gastrointestinal and central

nervous systems, and other organs and physiological processes (Basmajian, 1998). The physiological effects of stress relevant to physiotherapy are:

- ↑ secretion of catecholamines, glucocorticoids and insulin
- ↑ catabolism and protein breakdown, which consumes energy that could otherwise be used for healing (O'Leary and Coakley, 1996)
- ↓ gastric emptying and ↑ risk of aspiration (Beards and Nightingale, 1994)
- ↑ respiratory rate, heart rate and BP
- release of thyroid hormones (which further increase oxygen consumption), and antidiuretic hormone (ADH, which retains fluid)
- perceptual distortion and impaired judgement and memory, which limit response to advice and education
- sleep disruption, which further augments stress
- tendency to infection, gastric ulceration, muscle tension and blood clotting abnormalities
- depression
- exhaustion (Brannon *et al.*, 1998).

Clinical implications

Stress depends less on the extent of illness and more on the circumstances, including how the patient is handled. Helplessness is a common accompaniment to illness, especially in hospitalized patients, and the physiotherapist can do much by giving patients choices, listening to their needs, involving them in decisions and writing down advice to compensate for forgetfulness.

MINI CASE STUDY: MS LL

Identify this 62-year-old patient's problems from the selected details of her case study, and answer the questions. She has an exacerbation of COPD.

Background

RMH: heart failure, hypertension
HPC: ↑ SOB two weeks
ABGs on air: P_aO_2 10.2, P_aCO_2 6.4, pH 7.4, HCO_3^- 28.

Subjective assessment

Can't stop coughing.
Occasionally brings up phlegm.
Can't sleep.
Daren't lie down.
Exhausted.

Objective assessment

Apyrexial.
Oxygen via nasal cannulae at 2 L/min.
Rapid shallow breathing with prolonged expiration.
Fluid chart and clinical assessment indicate dehydration.
Speaking sets off paroxysms of coughing.
Wheezy cough, usually non-productive.
Clutches between legs when coughs.
Sits in chair day and night.
Can mobilize slowly.

Questions

1. Analysis?
2. Problems?
3. Goals?
4. Plan?

ABGs = arterial blood gases; HPC = history of present complaint; RMH = relevant medical history; SOB = shortness of breath.

RESPONSE TO MINI CASE STUDY

1. Analysis

Breathing pattern suggests ↑ WOB.
Blood gases indicate hypoxaemia, hypercapnia and compensated respiratory acidosis.
Uncontrolled coughing is largely ineffective and contributes to fatigue.
Coughing, stress incontinence, immobility and fluid restriction are inter-related.

2. Problems

SOB.
Fatigue.
Sputum retention.
Stress incontinence.
↓ mobility.

3. Goals

Short term: control cough, clear chest, balance rest and exercise.
Long term: educate patient and carers for home management.

4. Plan

- Identify cause of poor sleep, e.g. SOB/cough/noise/anxiety, then remedy as able
- Educate on cough suppression for use when cough is uncontrolled and non-productive
- Educate on mucociliary clearance, including fluid intake
- Educate on effective cough for when secretions are accessible
- Show breathlessness management strategies
- Teach pelvic floor exercises, including during coughing
- Mobilize to toilet
- Provide written daily programme for self-chest-management and self-mobility
- Liaise with team re oxygen therapy, getting dressed, mobility
- Refer to specialist colleague for assessment of continence
- Rehabilitate to independence, including family.

LITERATURE APPRAISAL

Comment on the logic of the following conclusion from a research study.

> *Our data suggest that the use of postural drainage and chest percussion in patients without sputum production is not indicated.*

Chest 1980; 78: 559–64

RESPONSE TO LITERATURE APPRAISAL

There is no logic to comment on!

RECOMMENDED READING

Anderson, S. (1990) ABG's – six easy steps to interpreting blood gases. *Am. J. Nurs.*, 90(8), 42–45.

Cornock, M. A. (1996) Making sense of arterial blood gases and their interpretation. *Nurs. Times*, 92(6), 30–31.

Gunning, M. P. and Hayes, M. A. (1999) Oxygen transport. *Curr. Anaesth. Crit. Care*, 10, 319–324.

Halliwell, B. (1994) Free radicals, antioxidants, and human disease: curiosity, cause or consequence? *Lancet*, 344, 721–724.

Higgins, T. L. and Yared, J.-P. (1993) Clinical effects of hypoxemia and tissue hypoxia. *Respir. Care*, 38, 603–616.

Leach, R. M. and Treacher, D. F. (1998) Oxygen transport: tissue hypoxia. *Br. Med. J.*, 317, 1370–1373.

Mitchell, P. (1999) Smoking is a major cause of blindness. *Med. J. Austr.*, 171, 173–174.

Shapiro, C. M. and Flanigan, M. J. (1993) Function of sleep. *Br. Med. J.*, 306, 383–385.

Stringfield, Y. N. (1993) Acidosis, alkalosis, and ABGs. *Am. J. Nurs.*, 93, 43–44.

Tobin, M. J. (1988) Respiratory muscles in disease. *Clin. Chest Med.*, 9, 263–286.

Treacher, D. F. and Leach, R. M. (1998) Oxygen transport: basic principles. *Br. Med. J.*, 317, 1302–1306.

Wagner, P. D. (1991) Clinical advances in pulmonary gas exchange. *Am. Rev. Respir. Dis.*, 143, 883–888.

Westbrook, J. L. and Sykes, M. K. (1992) Peroperative arterial hypoxaemia. The interaction between intrapulmonary shunt and cardiac output. *Anaesthesia*, 47, 307–310.

2 Assessment

Summary

Introduction

Accurate assessment is the linchpin of physiotherapy and forms the basis of rational practice. A problem-based assessment leads to reasoning such as: 'This patient cannot cough up his sputum by himself. Why? Because it is thick. Why? Because he is dehydrated. Why? Because he feels too ill to drink.' Illogical assessment leads to reasoning such as: 'This is COPD, therefore I will turn the patient side-to-side and shake her chest.'

A thoughtful assessment will lead to both effectiveness and efficiency because time will be saved by avoiding unnecessary treatment. Relevant parts of the assessment should be repeated after treatment to assess outcome.

Specific aspects of assessment for rehabilitation, intensive care and children are in Chapters 9, 14 and 16.

Background information

Ward reports and meetings

It is the physiotherapist's job to clarify the indications for physiotherapy to other staff and to explain which changes in a patient's condition should be reported. No patient is 'too ill' or 'too

well' for physiotherapy. The ward report or handover also provides the opportunity to check essentials such as whether the patient is drinking and eating.

Apart from a daily report from the nurse in charge, any other opportunity to communicate should be taken, such as ward rounds and meetings. This not only improves patient care and job satisfaction, it also boosts efficiency (Gosbee, 1998). If physiotherapy notes are kept separately from the medical notes, verbal communication can be reinforced by writing physiotherapy information in the medical notes, e.g. a resumé of treatment or request for a minitracheostomy.

Medical notes

Necessary details from the doctor's notes include:

- Past and present relevant history
- Social history, accommodation
- Other disorders requiring physiotherapy
- Conditions requiring precautions in relation to certain treatments, e.g. light-headedness, bleeding disorder, history of falls, swallowing difficulty/tendency to aspirate
- Relevant investigations
- Response to medical treatment
- Recent cardiopulmonary resuscitation (requiring close X-ray examination in case of gastric aspiration or fracture)
- Possibility of bony metastases
- Long-standing steroid therapy, leading to a risk of osteoporosis
- History of radiotherapy over the chest.

The last three findings contraindicate percussion or vibrations over the ribs.

Haematology

A full blood count assesses blood cells and coagulation. Haematocrit (packed cell volume) is the ratio of red blood cells to whole blood. Haemoglobin is the protein that carries oxygen to the tissues and acts as a buffer for acid–base balance. Reduced haemoglobin indicates anaemia, which causes fatigue and is poorly tolerated in people with heart disease. White blood cells, including neutrophils and eosinophils, are part of the immune system and are increased with infection. Clotting studies indicating that a patient might bleed easily include low platelet count, prolonged prothrombin time and raised INR (see Glossary for details).

Chemistry

The following are common electrolytes:

- Sodium (Na^+) affects the osmotic pressure of extracellular fluid.
  - ↓ serum Na^+ (hyponatraemia) is due to excess water administration or inappropriate ADH secretion
  - ↑ serum Na^+ (hypernatraemia) indicates dehydration
- Potassium (K^+) can impair diaphragmatic contraction if its value strays either way from normal
  - ↓ serum K^+ (hypokalaemia) predisposes to cardiac arrhythmias and can be caused by nebulized sympathomimetic drugs (Hung *et al.*, 1999) or respiratory alkalosis
  - ↑ serum K^+ (hyperkalaemia) suggests kidney failure.
- Chloride (Cl^-) is the chief anion in extracellular fluid
  - ↓ serum Cl^- accompanies acidosis, some kidney problems and prolonged vomiting
  - ↑ chloride in the sweat can be diagnostic of cystic fibrosis.

Urea is formed from protein breakdown and is excreted by the kidneys. High levels are caused by kidney failure, resulting from either disease or impaired perfusion due to heart failure or shock. Creatinine is formed from muscle breakdown and is also renally excreted. Levels rise with kidney failure and drop with malnutrition.

Albumin is secreted by the liver and forms over 60% of serum protein. Reduced levels, due to malnutrition, liver disease, nephrotic syndrome, chronic inflammation or severe acute disease, reduce osmotic pull from the vascular space so that fluid escapes and causes oedema, including pulmonary oedema.

Microbiology/bacteriology

Microorganisms are identified by culturing specimens of sputum, pleural fluid or blood on various media which promote their growth. Most bacteria grow in 24–48 hours but the tubercle bacillus may require 6 weeks. Sensitivity tests identify appropriate antibiotics capable of killing the bacteria.

Patient observation charts

Charts record the vital signs of body temperature, blood pressure (BP), heart rate (HR) and respiratory rate (RR).

Core temperature is one of the most tightly guarded of physiological parameters and is maintained within 0.2°C of normal in humans (Lenhardt, 1997). It should be checked at every visit because fever is the main harbinger of infection. It also helps to clarify diagnosis because patients may be incorrectly referred with 'a chest infection' when they have a different problem such as sputum retention or pulmonary oedema. Fever may be accompanied by increased RR and HR because excess heat raises metabolic rate and oxygen consumption, causing 10% elevation for every 1°C rise in temperature. Clinical examination may distinguish respiratory from other infection. Pyrexia can have a non-infectious origin, e.g. atelectasis, pulmonary embolism, lung fibrosis, blood transfusion and drug reaction or overdose (Meduri, 1990). A slight pyrexia following surgery is a normal reaction to tissue trauma, but fever beyond 48 hours raises suspicions of infection. The mechanism of fever is thought to be phagocytosis.

Normal BP is 120/80. BP persistently above 140/90 is hypertension. BP below 90/60 in adults is hypotension. Patients with a diastolic pressure above 95 mmHg should not normally be tipped head down. Those with a systolic pressure below 90 mmHg should be mobilized only with close observation for light-headedness. The relevance of BP to exercise training, heart surgery and manual hyperinflation is discussed in Chapters 9, 10 and 14.

HR is normally 60–100 bpm in adults. A heart rate of over 100 (tachycardia) may reflect increased sympathetic activity, hypoxaemia, hypotension, dehydration, anxiety, pain, fever or drugs such as the sympathomimetics, caffeine and nicotine. The effect is to increase myocardial oxygen demand. HR below 60 (bradycardia) may indicate profound hypoxaemia, arrhythmia, heart block, effect of drugs such as beta-blockers or vagal stimulation due to suctioning. Bradycardia may be normal during sleep and in the physically fit.

Drugs and oxygen are documented on the prescription chart. Their effects are monitored by, for example, peak flow and oxygen saturation. Details are given in Chapter 5. The fluid chart should show a positive daily balance of about a litre, because of insensible loss from the skin and respiratory tract (Luce *et al.*, 1993, p. 41). There are many reasons for a wide variation in this, including major fluid shifts after surgery. However, a trend towards fluid overload might be associated with pulmonary oedema, while a trend towards a negative balance increases the risks of dehydration. Fluid loss to the interstitial space is caused by altered hydrostatic or oncotic pressures, or increased capillary membrane permeability, leading to effective hypovolaemia.

SUBJECTIVE ASSESSMENT

> ***Osler supposedly said, 'Listen to the patient. He is telling you the diagnosis', to which I would add 'And she just might be telling you the best management too'.***
>
> Pitkin, 1998

The subjective assessment is what matters to the patient. Problems such as breathlessness are more closely related to quality of life than to physiological measurements (Mahler, 1995).

A well-lit area is needed that is quiet, warm and private. We can minimize the inequality of the relationship by:

- positioning ourselves at eye level if possible
- addressing adults by their surname, even if they are comatose (Wilkins *et al.*, 1995)

- asking permission before assessment and treatment.

Permission not only encourages patients' self-respect, it is a legal necessity in some countries. It is also good practice to ask before moving personal items.

Before asking questions, introductions and explanations are required because the public perception of physiotherapy is often limited to football and backache. Patients then need to define their problems and how these influence their lifestyle. It is worth building up rapport at this stage to encourage accuracy and set the foundations for a co-operative relationship. Respect for a patient's opinion is a potent motivating factor.

Respiratory symptoms

How long have symptoms been troublesome? What is their frequency and duration, their quality and severity? Are they getting better or worse? What are aggravating and relieving factors? The four cardinal symptoms of chest disease are wheeze, pain, breathlessness and cough with or without sputum.

A wheeze is caused by narrow airways and increases the work of breathing. The feeling should be explained to patients as tightness of the chest on breathing out, not just noisy, laboured or rattly breathing. Is the wheeze aggravated by exertion or allergic factors, suggesting asthma? Is it confirmed objectively by auscultation?

Is there pain? Chest pain may be musculoskeletal, cardiac, alimentary or respiratory in origin. Many patients associate chest pain with heart attacks, and anxiety may modify their perception and description of it. Lung parenchyma contains no pain fibres but chest pains relevant to the physiotherapist are the following:

- Pleuritic pain: this denotes the nature of the pain rather than the pathology. It is sharp, stabbing and worse on deep breathing, coughing, hiccuping, talking and being handled. Causes include pleurisy, some pneumonias, pneumothorax, fractured ribs or pulmonary embolism.
- Angina pectoris: paroxysmal suffocating pain, greater with exertion or stress, due to myocardial ischaemia. It is substernal or left anterior, sometimes radiating to the left arm or jaw.
- Musculoskeletal pain: e.g. costovertebral tenderness due to hyperinflation, abdominal muscle strain due to chronic coughing.
- Raw central chest pain: worse on coughing, caused by tracheitis and associated with upper respiratory tract infection or excessive coughing.

Breathlessness may be cardiovascular, metabolic, neurogenic, neuromuscular or respiratory. Respiratory breathlessness reflects excess work of breathing and is abnormal if inappropriate to the level of physical activity. Patients may deny breathlessness if it has developed gradually. Significant breathlessness is indicated by a need to pause during undressing or talking, or an inability to walk and talk at the same time. A key question at each visit can be a comparative measurement for that individual, e.g. how much can you do at your best/worst, what are you unable to do now because of your breathing?

If breathlessness increases in supine it is called orthopnoea. In lung disease this is caused by pressure on the diaphragm from the abdominal viscera. In heart disease a poorly functioning left ventricle is unable to tolerate the increased volume of blood returning to the heart in supine. Paroxysmal nocturnal dyspnoea is breathlessness at night caused by orthopnoeic patients sliding off their pillows during sleep, leading them to seek relief by sitting up over the edge of the bed.

Breathlessness caused by lung or heart disorders can be distinguished by peak flow readings (McNamara, 1992), auscultation, X-ray signs or exercise testing. Detailed measurement of breathlessness is described in Chapter 9.

Cough is abnormal if it is persistent, painful or productive of sputum. It is caused by inflam-

Table 2.1 Characteristics of cough

Type of cough	Possible causes
Dry	Asthma, interstitial lung disease, recent viral infection, pollutants, hyperventilation syndrome, ACE inhibitor drugs, mucosal irritation
Productive	COPD, bronchiectasis, cystic fibrosis, chest infection
With position change or lying down	Asthma, GOR, heart failure, bronchiectasis
Early morning	COPD, postnasal drip
Chronic persistent	Postnasal drip or GOR
With eating or drinking	Aspiration of stomach contents, e.g. neurological disease, elderly people
With exertion	Asthma, COPD, interstitial disease
Inadequate	Weakness, pain, poor understanding
Paroxysmal	Asthma, aspiration, upper airways obstruction

mation, irritation, habit or excess secretions, but may be underestimated by smokers and people who swallow their sputum. Suggested questions are:

- What started off the cough?
- Is there sputum?
- If so, what is the sputum like?
- Has it changed in quality or quantity?
- Is there sometimes blood?
- Does the cough occur at night (suggesting gastro-oesophageal reflux (GOR) and/or asthma)?
- Does it cause pain?

Table 2.1 identifies the causes of different coughs.

A cough caused by asthma or GOR should disappear once the condition is controlled. ACE inhibitor drugs cause a cough in 10% of patients (Mathewson, 1997), which disappears about 4 months after starting the drug. Other non-productive and 'habit' coughs, such as those following viral infection, usually disappear over time, but dry coughs can perpetuate themselves by irritating the airways. Factors that exacerbate coughing include irritants such as perfumes and cigarette smoke, or a change in air temperature, especially when breathing through the mouth. A postnasal drip is identified by the feeling of secretions sliding down the back of the throat, followed by throat-clearing.

Listening to the cough will help the clinician to check for weakness and pick up sounds that may be missed on auscultation but stimulated by a cough. It is best to ask patients to show how they would cough to clear secretions, rather than to ask them to 'show me a cough'.

Other symptoms

Fatigue, weakness or both may be present, exacerbated by chronic disease, anaemia, depression or anxiety. Fatigue is closely associated with breathlessness (Kellner *et al.*, 1992) and depression (Small and Graydon, 1992), which can reduce motivation and the ability to co-operate. Depression and anxiety may be expressed as pain (Duckworth, 1999).

Dizziness needs to be clarified (Lakhani, 1996). Does the patient mean true vertigo, i.e. a spinning feeling suggesting a lesion of the 8th cranial nerve or brain stem? Does s/he have postural hypotension or hyperventilation syndrome? Does dizziness precede a fall?

A history of falls needs to be related to the history. Are falls related to blackouts, weakness, breathlessness, footwear, eyesight, balance, lack of confidence or one of the causes of dizziness? Fainting or near-fainting may be caused by cardiovascular disorder, hyperventilation syndrome or 'cough syncope' following paroxysms of coughing.

Reasons for poor mobility need to be identified. Reduced mobility can lead to constipation, exacerbated by dehydration, and urinary incontinence, exacerbated by excess coughing. It is useful to adopt the practice of asking patients

the cause of their symptoms. Their perceptions are often surprisingly accurate.

Functional limitations

Problems with activities of daily living, finance, employment and housing loom large for people with respiratory disease. How much daily exercise do they take? Are they employed? How many stairs are there at work or home? Is the environment well-heated, smoky, dusty? Do they live alone, eat well, smoke? Is it difficult to bathe, dress or shop? What support is available? Limitation of activity is not in itself an accurate indicator of respiratory disease because of the many variables, but a change in activity level is noteworthy.

How does the patient feel about the disease? This question provides the opportunity for patients to describe their feelings but does not pressurize them. Anxiety is common if symptoms are unpredictable. Other distressing factors are frustration, embarrassment, restricted social function and a feeling of loss of control. If the patient spends the day flopped in front of the TV, is this because of preference, exercise limitation or depression?

A questionnaire is an efficient way of assessing symptom-related problems, functional activity and the patient's emotional reaction to the disease (e.g. Box 9.2). If the patient is unable to give a history, relatives can be questioned, bearing in mind that they may identify fewer problems and see them from a different perspective. Details of previous experience with physiotherapy give an indication of which interventions have been beneficial.

Quality of life scales are sensitive to mild disease (Ferrer, 1997), more related to clinical decisions than pulmonary function tests (Osman, 1997) and a useful predictor of survival (Squier *et al.*, 1995). These are discussed in Chapter 9.

OBSERVATION

Preliminary observation of the breathing rate and breathing pattern should be made before the patient is aware of the physiotherapist's presence. Detailed observation can then be undertaken.

General appearance

Does the posture suggest fatigue, pain, altered consciousness or respiratory distress? Breathless people characteristically brace their arms so that their shoulder girdle muscles can work as accessory muscles of respiration. For mobile patients, the gait gives an indication of mood, co-ordination, breathlessness or lack of arm swinging, which suggests muscle tension.

Is the patient obese, thus compromising diaphragmatic function, or cachectic, indicating poor nutrition and weakness? If the patient is unkempt, does this reflect difficulty with self-care or a measure of how the disease has affected self-esteem? Is the patient restless or incoherent, possibly because of hypoxia?

Colour

Pallor is associated with anaemia, reduced cardiac output or hypovolaemic shock. A plethoric appearance shows as a florid face indicating the excess red blood cells of polycythaemia. Cyanosis is blue coloration due to unsaturated haemoglobin in the blood, caused by respiratory or circulatory disorders.

Peripheral cyanosis shows at the fingers, toes and ear lobes, and signifies a problem with circulation. Stagnant blood gives up its oxygen and the peripheries appear blue. Causes are a cold environment or pathology such as peripheral vascular disease.

Central cyanosis shows at the mouth, lips and tip of the tongue, and indicates a gas exchange problem. It is an unreliable guide to hypoxaemia and is identified at S_aO_2 levels that vary between 72% and 95% (Martin, 1990b). Its detection depends not just on haemoglobin in the blood but also on skin pigmentation, patency of vessels, ambient lighting and keenness of the observer's eye. It can be masked by anaemia or exaggerated by polycythaemia. Cyanosis is a warning rather than a measurement and its absence should not lead to a false sense of security.

Hands

The hands are a rich source of information. A poor cardiac output causes cold hands. CO_2 retention is indicated by warm hands caused by peripheral vasodilation, and a flapping tremor of the outstretched hands (asterixis) that disappears when the hands drop to the patient's side. A fine tremor may be a side effect of bronchodilator drugs, particularly in the elderly. Generalized muscle wasting may be seen most clearly in the hands. For patients who are unable to give a smoking history, nicotine stains provide irrefutable evidence of the deadly habit.

Clubbing is recognized by loss of the angle between nail and nail bed, and in later stages by bulbous ends to the fingers. Causes are:

- pulmonary: 75%
- cardiac: 10%
- liver or gut: 10%
- other: 5% (Jefferies and Turley, 1999, p. 117).

Pulmonary causes include fibrosing alveolitis and infective disorders such as cystic fibrosis and abscess. Recent-onset clubbing may be the first sign of bronchial carcinoma (Sridhar *et al.*, 1998). The exact mechanism of clubbing is unknown but it is associated with increased local perfusion due to fluid accumulation (Currie and Gallagher, 1988). Physiotherapist find clubbing supremely uninteresting because it is not affected by physiotherapy. In relation to lung pathology, it is only known to be reversed by lung resection or transplantation.

Oedema

Oedema is excess fluid in interstitial spaces. Peripheral oedema accumulates at the ankles or sacral area, depending on posture, and is usually caused by kidney, liver, cardiac or respiratory disease. In the respiratory patient, it is associated with poorly perfused kidneys due to chronic hypoxaemia and heart failure. More details are given on page 72.

Jugular venous pressure

In advanced lung disease associated with pulmonary hypertension, pulmonary vascular resistance impedes emptying of the right ventricle and engorges the jugular vein. With the patient lying at 45°, the head symmetrical and supported to prevent accessory muscle activity, elevated venous pressure is indicated by jugular venous distension. A flickering impulse represents jugular venous pressure (JVP). If the JVP is more than 3–4 cm above the sternal angle at end-exhalation, the patient usually has right heart failure secondary to left heart failure or chronic hypoxaemia. In the absence of cardiovascular disease, JVP represents the volume status of the patient. JVP increases with abdominal contraction, decreases in dehydrated patients and may not be visible in obese patients.

Chest shape

The chest and abdomen should be as visible as the patient feels comfortable with. A normal chest shape is shown in Figure 2.1. Chronic lung disease can lead to a rigid, barrel-shaped, hyperinflated chest, with horizontal ribs and increased anteroposterior diameter relative to transverse diameter (p. 68).

Abnormalities of the chest wall may increase the work of breathing (WOB); for example, a restrictive defect can be caused by kyphoscoliosis. A kyphotic curvature exceeding 70° increases the risk of respiratory failure and a curvature exceeding 100° is associated with hypoxaemia and cor pulmonale (Ras *et al.*, 1994). Rarer conditions are pigeon chest (pectus carinatum), which protrudes the sternum, and funnel chest (pectus excavatum), which depresses the sternum; these do not usually restrict lung function but may require cosmetic surgery.

Respiratory rate

A full minute is required to count the respiratory rate acurately. Measurement obtained by counting for 15 seconds and multiplying by 4 is now considered worthless (Barnes, 1994, p. 17),

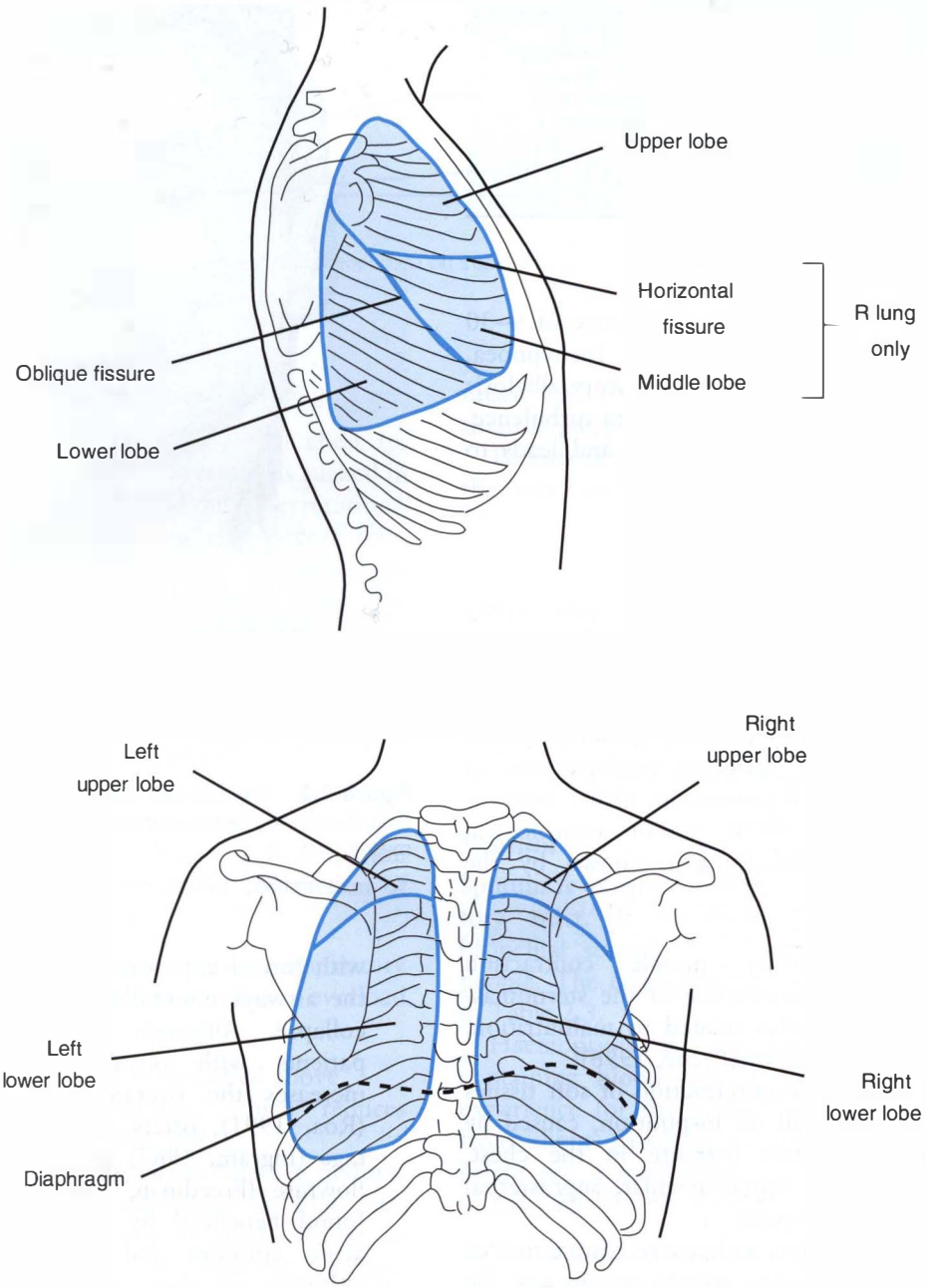


Figure 2.1 Lateral and posterior views of the lobes and fissures of the lung.

Table 2.2 Causes of abnormal respiratory rate

↑ RR	↓ RR
Lung or heart disease	Drug overdose
Pain or anxiety	Brain damage
Anaemia	Diabetic coma
Inspiratory muscle fatigue or weakness	Exhaustion
Pulmonary embolus	
Spontaneous pneumothorax	
Fever	

partly because of the wide adult range of 9–30 breaths/min (average 10–20/min). Tachypnoea, or RR over 40/min, leads to respiratory alkalosis and increases WOB because of extra turbulence. RR below 8/min increases P_aCO_2 and leads to respiratory acidosis (Table 2.2).

Breathing pattern

Normal breathing is rhythmic, with active inspiration, passive expiration and an inspiratory to expiratory (I:E) ratio of about 1:2. Many individual variations are normal, the same ventilation being achieved by different combinations of rate and depth or different combinations of chest and abdominal movement. Other patterns suggest increased WOB and/or neurological defect. Laboured breathing is shown by the following:

- Obvious accessory muscle contraction (Figure 2.2); prominence of the sternomastoid muscle is also related to malnutrition, not just hypertrophy (Peche, 1996)
- Indrawing/recession/retraction of soft tissues of the chest wall on inspiration, caused by excessive negative pressure in the chest, which sucks in supraclavicular, suprasternal and intercostal spaces
- Forced exhalation with active contraction of abdominal muscles, which compresses the airways and increases WOB yet further (Ninane *et al.*, 1992) without speeding expiratory flow (Tobin, 1988)
- Pursed lip breathing (PLB), often associated with forced expiration, adopted to stabilize the airways internally and delay expiratory collapse. Although necessary for some patients with obstructed airways, PLB increases the overall work of breathing (Roa, 1991), offers no mechanical advantage (Ingram, 1967) and does not increase flowrate (Freedman, 1987). However it is found beneficial by some patients during acute episodes and may improve S_aO_2, although it does not improve oxygen uptake (Breslin, 1992), probably because of impaired cardiac output (Cameron and Bateman, 1990). More details are in Chapter 9.

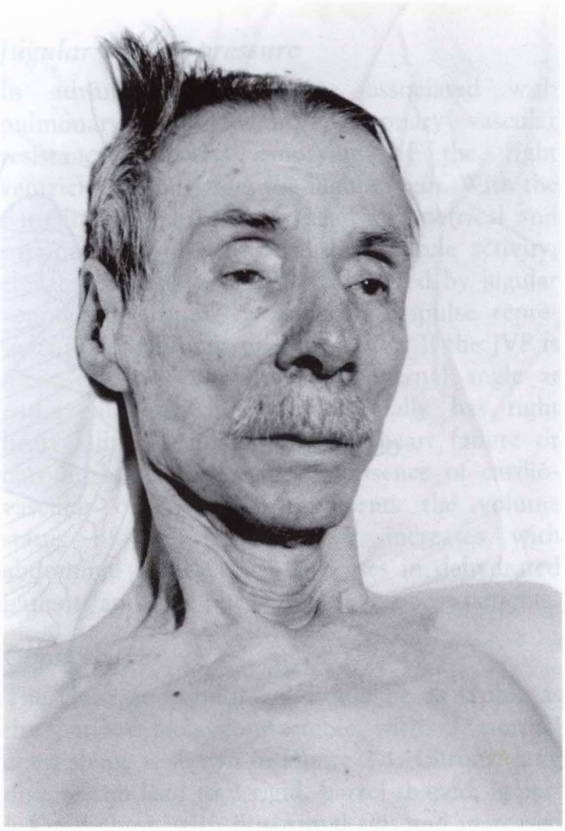


Figure 2.2 Malnourished patient with soft tissues draped over the bones and prominent sternomastoid muscle.

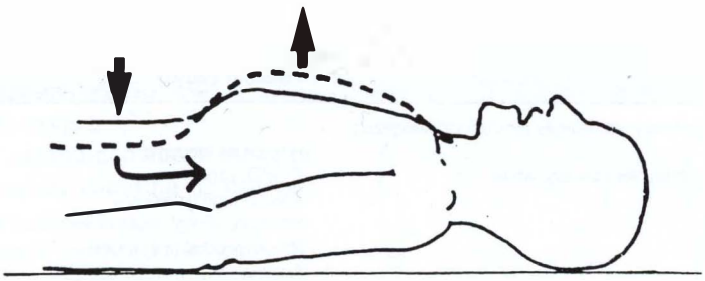


Figure 2.3 Paradoxical inward movement of the abdomen on inspiration, due to weakness or fatigue of the diaphragm.

Paradoxical breathing increases WOB, e.g.:

- Rib fractures may lead to a flail chest (p. 407), when part of the chest wall is sucked in on inspiration and pushed out on expiration.
- The flattened diaphragm that occurs with hyperinflation can become in effect an expiratory muscle, pulling in the lower ribs on inspiration (Hoover's sign, p. 69)
- If there is increased inspiratory load, or severe diaphragmatic weakness or paralysis, abdominal paradox may be observed, in which the ineffective diaphragm is sucked up into the chest by negative pressure generated in the chest during inspiration so that the abdomen is sucked in (Figure 2.3). Palpation distinguishes this from active contraction of the abdominal muscles.

The following three signs indicate inspiratory muscle fatigue, weakness and/or overload (Mador, 1991):

- abdominal paradox, as described above
- rapid shallow breathing, which reduces elastic loading (Mador, 1991)
- less commonly, alternation between abdominal and rib cage movement so that each muscle group can rest in turn, similar to shifting a heavy suitcase between alternate hands.

Tests for severe weakness or paralysis are described on page 61.

Exhaustion is presaged by lowered RR with raised P_aCO_2. This is a danger sign indicating that the patient may need mechanical assistance.

Periods of apnoea with waxing and waning of the rate and depth of breathing are called Cheyne–Stokes breathing when regular and Biot's breathing when irregular. These indicate neurological damage, but Cheyne–Stokes breathing is also associated with end-stage heart failure due to impaired blood supply to the respiratory centres, or may be normal in some elderly people. Irregular breathing often occurs in normal REM sleep. Sighing respiration may indicate hyperventilation syndrome.

Sputum

Sputum is expectorated mucus from the respiratory tract. It is always abnormal because bronchial secretions are swallowed in healthy people. The characteristics of sputum are listed in Table 2.3.

Haemoptysis is expectoration of sputum containing blood, which can be an alarming experience for the patient. It varies in severity from slight streaking to frank bleeding. It is bright red if fresh, pink if mixed with sputum, or rusty brown if it is old blood. Causes are:

- bronchiectasis (intermittent, bright red)
- lung cancer (persistent)
- pulmonary tuberculosis (intermittent)
- lung abscess (copious)
- pneumococcal pneumonia (rusty red)
- pulmonary oedema (pink, frothy)

Table 2.3 Characteristics of sputum

Appearance	Possible cause
Serous, i.e. frothy (mixed with air), sometimes pink (blood squeezed into alveoli)	Pulmonary oedema
Mucoid, i.e. clear, grey or white, like raw egg white	COPD, cancer
Thick	Infection, dehydration
Purulent, yellow, green	Infection, allergy, stasis of secretions e.g. bronchiectasis
Purulent, rusty red	Pneumococcal pneumonia
Thick plugs	Asthma
Stringy	Asthma, poor oral hygiene
Thick, green, musty-smelling	*Pseudomonas* infection
Blood-stained	See haemoptysis, p. 37

- pulmonary embolus (bright red)
- blood clotting abnormality (fresh)
- trauma such as intubation, tracheostomy, lung contusion or frequent tracheal suction (fresh).

Haematemesis occurs when blood is vomited, and may be confused with haemoptysis. It is more likely to contain blood mixed with food than with mucus and is distinguished by acidity and a dark brown colour that resembles coffee grounds. It may be accompanied by melaena (digested blood passed per rectum) or nausea. Close questioning is needed to identify whether expectorated blood has been swallowed and vomited or if vomited blood has been aspirated and expectorated.

Sputum specimen and sputum induction

Sputum cultures help to identify the pathogen responsible for a chest infection so that the appropriate antibiotic can be given. They can also identify whether the presence of eosinophils or neutrophils indicate an allergic or inflammatory component respectively. However, often only upper respiratory organisms are identified (Thistlethwaite, 1998) and most specimens are contaminated by these bacteria, especially in intubated patients (Meduri, 1990). Patients are advised to blow their nose, rinse their mouth and spit out saliva before expectorating (Gershman, 1996). Bronchoscopic brushings provide cleaner specimens (p. 143).

For patients who require suction, a sterile mucus trap is incorporated into the circuit. This should be kept upright during suction to prevent the specimen bypassing the trap.

Sputum induction is used when secretions cannot be produced by mucociliary clearance techniques and coughing, or when specimens are required from the lower respiratory tract. It can provide a greater yield than bronchoscopy (Anderson, 1995) but tends to produce specimens contaminated with oral pathogens, especially with hospitalized or immunocompromised patients.

Hypertonic saline is used to irritate the airway walls and draw water into the airways. Side effects include bronchospasm, breathlessness, oxygen desaturation and nausea. If TB or HIV are suspected, a negative-pressure room is required to minimize cross-infection. The following sequence is advised:

- Explain procedure to patient including possible side effects, obtain consent
- Ask patient to avoid food for two hours to reduce risk of nausea
- Ask patient to remove any dentures, then to brush teeth, tongue, cheeks and gums with water, not toothpaste, and a new toothbrush
- Pretreat with a bronchodilator (Magnussen and Holz, 1999)
- Attach oximeter to patient, prepare oxygen equipment in case of desaturation
- Deliver 20–30 mL hypertonic (2.7% = 3 ×

N) saline by ultrasonic nebulizer over 10–20 minutes, the patient sitting up and taking occasional deep breaths
- If S_aO_2 drops, give oxygen
- If S_aO_2 cannot be maintained above 90%, or if there is haemoptysis, vomiting or distress, stop
- Ask patient to gargle with sterile water to reduce oral pathogens, then spit out saliva
- Ask patient to cough and expectorate into a sterile container
- If mor
discard the first
- Deliver promptly to the laboratory, as it must be processed within 2 hours (Magnussen and Holz, 1999)
- If secretions are not forthcoming, teach ACB/AD (p. 194) or other mucociliary clearance technique
- Continue monitoring S_aO_2 for 30 minutes
- Record procedure and outcome in medical notes
- Ensure patient is fed.

Successful specimens are usually watery and look like saliva to the naked eye.

Equipment

Is oxygen being used as prescribed? Is it comfortable? Is the humidifier working? Are drips, drains, chest drains and machinery in order? Details of equipment are covered in the relevant chapters. Oximetry is described on page 323.

PALPATION

Abdomen

The abdomen enjoys a close relationship with the diaphragm and should be gently palpated at every assessment. A distended abdomen inhibits diaphragmatic movement, restricts lung volume and increases WOB. Causes include pain and guarding spasm, obesity, flatulence, paralytic ileus, constipation, enlarged liver, ascites and acute pancreatitis.

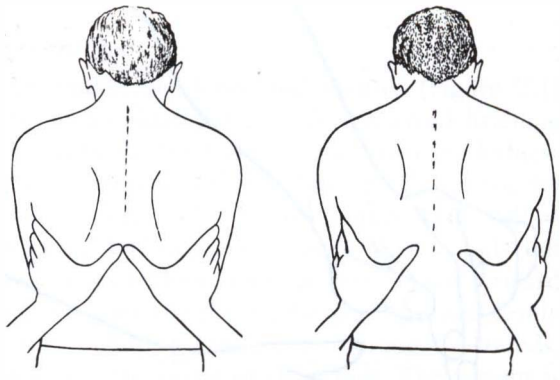


Figure 2.4 Palpation for expansion. The fingers hold the sides of the chest and the thumbs rest lightly an each side of the spine. On inspiration, symmetrical separation of the thumb tips indicates equal chest expansion. (From Wilkins, R. L., Sheldon, R. L. and Krider, S. J. (1995), *Clinical Assessment in Respiratory Care*, Mosby, Toronto)

Chest expansion

Chest movement gives an indication of lung expansion. It can be evaluated by inspection or palpation. Apical expansion is best assessed by standing at the foot of the bed and observing the supine patient. For the rest of the chest, the patient sits over the edge of the bed if possible, and the clinician palpates from behind (Figure 2.4).

While palpating for expansion, other signs may be identified such as the crackling of sputum or, around the neck and upper chest, the popping of surgical emphysema (air in subcutaneous tissue), which feels like crackling cellophane.

Percussion note

A percussion note (PN) is elicited by tapping the chest wall (Figure 2.5). This is similar to tapping a wine barrel to check how full it is, or tapping a wall to see if it is hollow. The PN evaluates the density of underlying tissue to a depth of 5 cm (Wilkins *et al.*, 1995, p. 61). It is useful for confirming breath sounds (e.g. bronchial breathing or diminished breath sounds) and is

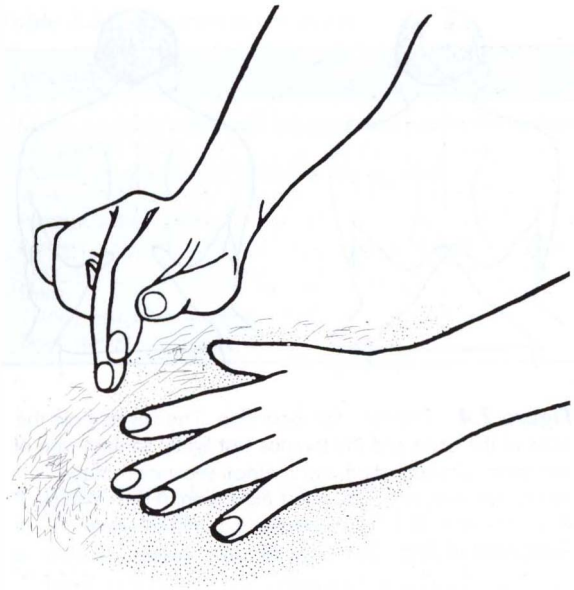


Figure 2.5 Eliciting a percussion note over the chest. One finger is placed firmly along an intercostal space and struck by a finger of the opposite hand. To avoid damping the vibrations, the percussing finger should recoil sharply like a woodpecker striking a tree. (From Wilkins, R. L., Sheldon, R. L. and Krider, S. J. (1995), *Clinical Assessment in Respiratory Care*, Mosby, Toronto)

especially helpful if breath sounds are obscure, e.g. in patients unable to take a deep breath, those on noisy CPAP (p. 156) or those with loud crackles or wheezes. Each side of the chest should be percussed alternately for comparison, remembering that the upper lobe predominates anteriorly and lower lobe posteriorly.

The PN is resonant over normal lung tissue. Hyperresonance indicates excess air, as in hyperinflation or a large pneumothorax. A stony dull note is an unmistakable sound heard over a pleural effusion larger than 500 mL. The PN is an inexact guide to these conditions, which are more easily detected by X-ray. Most useful to the physiotherapist is the dull note of atelectasis, when air has been absorbed and alveoli have collapsed, or consolidation, which increases lung density by filling alveoli and creating a semi-solid area of lung.

Systemic hydration

Dehydration predisposes to:

- sputum retention
- pressure sores
- constipation
- confusion
- hypernatraemia (Palevsky, 1996)
- fatigue and ↓ exercise tolerance (Barr, 1999).

A minimum 1500 mL of fluid is required per day, which may not be achieved by people who are ill or in an unfamiliar environment. Patients at particular risk are:

- people who feel too sick to drink
- patients not on intravenous fluids
- the elderly, who often have reduced total body water, altered perception of thirst, impaired renal function and reduced mobility, which inhibits self-regulation of fluids and increases fear of urinary incontinence.

A patient who has cor pulmonale and does not have swollen ankles should be closely examined for dehydration.

Dehydration causes inelastic skin – but so does ageing; it produces a dry tongue and lips – but so do mouth breathing, oxygen therapy and a blocked nose. Clinical assessment for dehydration is imperfect but the following are guidelines:

- The skin over the sternum shows little loss of elasticity in the elderly. When pinched gently, it should bounce back rather than 'tent', which indicates reduced turgor and dehydration
- The axilla has a dry, velvety feel in most dehydrated people (Eaton *et al.*, 1994).

Dehydration is also suspected in a patient with dark urine, postural hypotension with a racing pulse, or increased urea, creatinine, sodium and potassium levels. Weakness, malaise, headache, nausea, vomiting, cramps and low-grade fever are indicative of, but not specific to, dehydration.

Trachea

Tracheal deviation is detected by palpating with one finger on each side of the trachea. In the absence of thyroid enlargement, deviation is due to shift of the mediastinum away from a large pleural effusion or tension pneumothorax, or a shift towards upper lobe atelectasis or fibrosis, as confirmed by X-ray. A hyperinflated chest forces down the diaphragm and causes a tracheal tug in which the thyroid cartilage is pulled down on inspiration.

Capillary refill

With good circulation, pressing briefly on the fingernail is followed by rapid return of blood flow. If capillary refill is slower than 3 seconds, reduced cardiac output or impaired digital perfusion is suspected.

Tactile vocal fremitus

Palpation for the vibration of the voice gives similar information to vocal resonance (p. 43). Vibrations are reduced in people who are obese or very muscular.

AUSCULTATION

Auscultation is used to verify observed and palpated findings before and after treatment. Prior to reaching for the stethoscope, it is worth listening for sounds at the mouth, which are barely audible in a person with normal lungs. Noisy breathing indicates increased airflow turbulence due to obstructed upper airways, manifest as crackles or wheezes or both. Crackles heard at the mouth should be cleared by coughing in order to prevent them masking other sounds during auscultation. A monophonic (single note) wheeze in the upper airways creates a faint strangled sound at the mouth, greater on inspiration, called stridor. This is a serious sign denoting laryngeal or tracheal narrowing to a diameter as small as 5 mm (Thomas and Manara, 1998). Stridor is a warning that nasopharyngeal suction should be avoided and the patient's head kept elevated to minimize oedema.

Technique

The underlying lobes and fissures (Figure 2.1) should be visualized in order to avoid listening optimistically for breath sounds over the kidney. The diaphragm of the stethoscope is used for the high frequencies of breath sounds. The bell is used for the low frequencies of heart sounds and for small children. The ear pieces face forward into the ears and the diaphragm is pressed firmly on the chest to minimize extraneous sounds, including the rustle of chest hair. The patient is asked to breathe through the mouth, slightly deeper than normal but not rapidly because this causes light-headedness. Each area of lung is compared on alternate sides, asymmetry usually indicating pathology.

The patient is best positioned sitting upright over the edge of the bed with arms forward to protract the scapulae. Leaning forward in bed from long-sitting can be used as a compromise, but this position squashes the lung bases, and breath sounds over this important area may be indecipherable. In patients who cannot sit up, side-lying can be used, with allowance for a louder sound in the dependent lung (Jones *et al.*, 1999) because of greater turbulence through more compressed airways and stronger sound transmission through denser lung. However, there may be quieter sounds from the dependent lung if it is so compressed that airflow is reduced. The diaphragm of the stethoscope should be cleaned with alcohol wipes between patients (Smith *et al.*, 1996).

Breath sounds

Breath sound intensity indicates either regional ventilation or factors that affect their transmission. Breath sounds are generated by turbulent airflow in the large airways, then transmitted through air, liquid and solid to the chest wall, each substance attenuating the sound to a different degree. Sounds at the surface are filtered versions of those at the trachea. Sounds are not generated beyond lobar or segmental bronchi because the total cross-sectional area is too wide to create turbulence (Jones, 1995a).

The term 'breath sounds' is more accurate than 'air entry', because air may enter the lung but transmission of the sound can be blocked. Breath sounds may be normal, abnormal or diminished.

Normal breath sounds are muffled because air in the alveoli filters the sound. Expiration is shorter and softer than inspiration. Normal breath sounds are quieter in the base than the apex because the greater volume of the lung bases filters the sound further. If breath sounds are difficult to hear and the patient is unable to help by voluntary deep breathing, it is possible to utilize the natural deep breathing following exertion by listening immediately after the patient has talked or turned or been suctioned.

Bronchial breathing is an *abnormal* sound that is distinguished by:

- a hollow blowing quality on expiration
- long expiration
- a pause between inspiration and expiration.

It is heard over consolidation, which acts acoustically like a lump of meat in the lung, the solid medium transmitting sounds more clearly than air-filled lung (Figure 2.6). Bronchial breathing is also heard over small areas of collapse provided there is a patent bronchus.

Bronchial breathing can also be heard over the upper level of a pleural effusion. The displaced and compressed lung transmits the sound as if consolidated (Sapira, 1995). Low-pitched bronchial breathing may be heard over fibrotic lung tissue. Bronchial breath sounds indicate loss of functioning lung volume.

Diminished breath sounds are heard if:

- the patient is obese, in a poor position or not breathing deeply
- there is no air entry to generate the sound, e.g. atelectasis with occluded airway
- there is air entry but transmission of sound is deflected by an acoustic barrier such as the air–solid or air–liquid interface of a pneumothorax or pleural effusion (Figure 2.6)

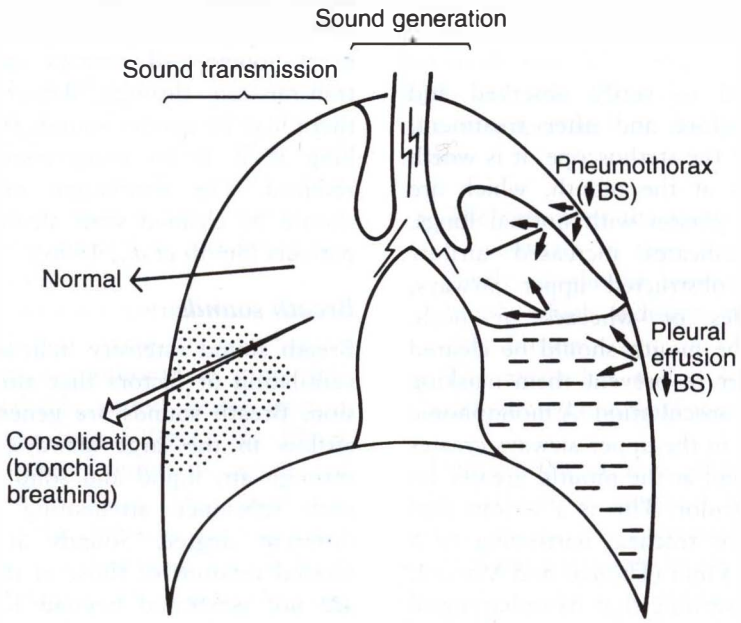


Figure 2.6 Normal, abnormal and diminished breath sounds heard at the chest wall. *BS: breath sounds.*

- there is air entry but insufficient airflow to generate sound, or excess air in the lung that filters sound, e.g. hyperinflation as in emphysema or acute asthma (Pasterkamp, 1997).

Hyperinflated chests can sometimes be manually deflated to reduce FRC so that breath sounds are clearer.

Inaudible breath sounds over the chest of a person with acute asthma are a danger sign (p. 77).

Added sounds

Added sounds are superimposed on breath sounds. They are sometimes more obvious and can mask breath sounds. If added sounds are louder on one side of the chest than the other, this may be caused by increased added sounds on the same side or reduced breath sounds on the opposite side. Non-respiratory sounds occur independently of the breathing cycle and may be transmitted from the abdomen, voice or water in humidifier tubing.

Crackles

Crackles indicate secretions or parenchymal disorder (Piirilä *et al.*, 1991) and are created when air is forced through airways that have been narrowed by oedema, inflammation or secretions, or when airless alveoli or peripheral airways snap open. They are principally heard on inspiration and their timing depends on the source. Early-inspiratory crackles arise in the large airways, may be heard at the mouth, are independent of gravity and are often heard in COPD. Early and mid-inspiratory crackles are characteristic of bronchiectasis or other hypersecretory disease. Absence of crackles does not always indicate absence of secretions (Jones and Jones, 2000). Late-inspiratory crackles originate in alveoli and peripheral airways as they open at the end of inspiration and are associated with pneumonia, fibrosis or pulmonary oedema.

The weight of the lung itself causes a degree of airway closure so that late-inspiratory crackles may be heard in dependent regions, especially in elderly obese people who have been recumbent for some time. Late-inspiratory crackles are sometimes called fine crackles, dry crackles, Velcro crackles or crepitations. Crackles are heard predominantly on inspiration but both inspiratory and expiratory crackles are heard in bronchiectasis (coarse) and fibrosing alveolitis (fine).

Wheezes

Wheezes are generated by vibration of the walls of a narrowed airway as air rushes through. Expiratory wheeze, combined with prolonged expiration, is usually caused by bronchospasm. Wheeze on inspiration and expiration can be caused by other forms of airways obstruction such as mucosal oedema, pulmonary oedema, sputum, tumours and foreign bodies. A monophonic wheeze can mean local airway obstruction from a foreign body or tumour. A wheeze increases the work of breathing.

Pleural rub

Inflammation of the pleural surface occurs in pleurisy, producing the sound of roughened surfaces rubbing on each other. This pleural rub sounds like boots crunching on snow and is localized but best heard over the lower lobes because excursion of the pleura is greater basally.

Voice sounds

The vibrations of the spoken word can be felt by the hands (tactile vocal fremitus) or heard through the stethoscope (vocal resonance). The patient is asked to say '99' or engage in conversation.

Voice sounds are normally an unintelligible mumble because vowels are filtered through air-filled lung. Increased voice sounds, known as bronchophony, are usually associated with bronchial breathing and are heard when the voice is transmitted through a denser medium, e.g. consolidation or atelectasis with a patent airway. Reduced voice sounds are heard when there is atelectasis with a blocked airway, or with pneumothorax or pleural effusion. Voice

Table 2.4 The relation of respiratory conditions to clinical signs

	Observation	Percussion note	Breath sounds (BS)	Added sounds	Vocal resonance/tactile vocal fremitus
Consolidation	Normal	Dull	Bronchial breathing	–	Increased
Atelectasis with patent airway	Expansion sometimes decreased over affected area	Dull	Bronchial breathing	–	Increased
Atelectasis with occluded airway	As above	Dull	BS decreased	–	Decreased
Pneumothorax	Expansion normal or decreased	Hyperresonant	Normal or decreased or absent	–	Normal or decreased or absent
Pleural effusion	Expansion normal or decreased	Stony dull	BS decreased Aegophony at upper level of effusion	–	Decreased Increased at upper level of effusion
Acute asthma	Hyperinflated chest	Hyperresonant	BS decreased or absent	Expiratory wheeze	Normal
Emphysema	Prolonged expiration Pursed lip breathing Barrel chest	Hyperresonant	BS decreased	–	Normal or decreased
Chronic bronchitis	Normal	Resonant, i.e. normal	Normal	Early inspiratory crackles ± wheeze	Normal
Bronchiectasis	Normal	Resonant	Normal	Inspiratory and expiratory crackles	Normal
Pulmonary oedema	Normal	Resonant	Normal	Crackles at bases ± wheeze	Normal
Interstitial lung disease	Expansion symmetrically decreased	Resonant	Normal or decreased	End-inspiratory crackles	Normal

sounds transmitted above the liquid–air interface at the top of a pleural effusion have a characteristic nasal bleating quality, a slightly different form of increased vocal resonance called aegophony.

Another confirmatory test is to ask the patient to whisper '99'. Over normal lung tissue, whispered words are barely audible, but through a solid medium such as consolidation, individual syllables are recognizable. This is known as whispering pectoriloquy.

Table 2.4 relates physical signs to different disorders and Table 2.5 differentiates the signs of excess sputum and pulmonary oedema.

EXERCISE TOLERANCE

Exercise testing is used to assess patients for exercise training and to provide outcome measures. Other applications include assessing the effects of lung surgery, for which the dynamic values of exercise testing are more accurate than the static values of spirometry (Tsubota *et al.*, 1994).

Exercise capacity is best assessed functionally because:

- lung function tests are not a good predictor of excercise capacity (Bradley *et al.*, 1999)

Table 2.5 Comparative signs of excess secretions and pulmonary oedema

	Excess secretions	Pulmonary oedema
History	Lung disease	Heart disease (may be secondary to lung disease)
Temperature	↑ if chest infection	Normal
Fluid balance chart	Normal	Fluid retention usually
Crackles	Patchy	Bilateral, late-inspiratory, dependent
Secretions	Mucoid or purulent	Frothy, white or pink
Clearance of secretions	By cough or suction	By diuretics
Chest X-ray	Normal, or related to lung disease	Bilateral hilar flare, often enlarged heart, sometimes pleural effusion
Albumin	Normal	May be reduced

- laboratory tests are for physiological measurement rather than monitoring of progress
- the patients own estimate of exercise tolerance is not objective and accommodates to a slowly deteriorating capacity

Details are on page 219.

IMAGING THE CHEST

The chest X-ray provides a unique insight into the state of the lungs and chest wall. It does have certain limitations, and physiotherapists should not fall into the trap of 'treating the X-ray'.

- X-ray findings tend to lag behind other measurements; for example, they are a later indication of chest infection than pyrexia, and pneumonia may have been resolved for days or even weeks while X-ray signs still linger.
- A normal radiograph does not rule out disease because its contribution is structural only. For example, the physical damage of emphysema is more apparent than the hypersecretion of chronic bronchitis because secretions do not show on X-ray, and postoperative patients with impaired oxygenation may have a normal film (Wiener, 1992).
- The two-dimensional representation of a three-dimensional object can obscure the relationship between certain structures and hinder the accurate location of lesions.

If possible, a posteroanterior (PA) view is taken, in which the beam is directed from the back (Figure 2.7).

This makes for an optimum view of the lungs, the patient taking a deep breath in the standing position with shoulders abducted so that the medial borders of the scapulae do not obscure the lungs. The erect position ensures that gas passes upwards, so that a pneumothorax is easier to detect, and fluid passes downwards, so that a pleural effusion is easier to see.

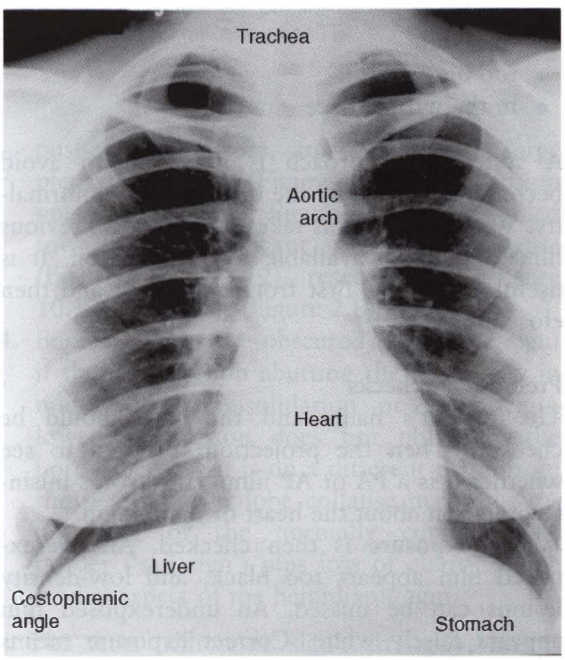


Figure 2.7 Normal PA film.

For less mobile patients, a portable film is taken, with the rays passing anteroposteriorly (AP), and the patient sometimes unable to take a deep breath. The heart is magnified by 15–20% (Wiener *et al.*, 1991), the anterior ribs are less clear and the lung fields are partly obscured by the scapulae and a raised diaphragm. Pleural effusions appear as non-specific homogenous densities that are difficult to identify, although they differ from parenchymal densities in that vascular markings are visible through the density. Whether patients are slumped ('erect portable' film) or supine, results are similar.

Dense structures absorb rays and are opaque, while air has a low density and appears black. Allowance should be made for normal variations between individuals such as different-shaped diaphragms. Chest films show bilateral symmetry for many structures, enabling opposite sides to be compared.

Systematic analysis

Abnormalities can be identified as:

- too black
- too white
- too big
- in the wrong place.

A systematic approach is necessary to avoid becoming diverted by the first obvious abnormality. With practice this takes 30 seconds. Previous films should be available for comparison. It is useful to observe first from a distance and then close up.

Preliminary checks

The patient's name and the date should be checked. Then the projection is noted to see whether it is a PA or AP film. This avoids misinterpretation about the heart or diaphragm.

The exposure is then checked. An overexposed film appears too black, and low-density lesions can be missed. An underexposed film appears falsely white. Correct exposure means that vertebral bodies are visible through the upper but not the lower heart shadow.

Symmetry is correct if the spinous processes, which appear as teardrop shapes down the spine, are midway between the medial ends of the clavicles. This check avoids misinterpretation about displacement of the heart, which is at the front of the chest. If the patient is rotated to either side, the heart shadow appears shifted towards that side.

Trachea

The dark column of air overlying the upper vertebrae represents the trachea, which is in the midline down to the clavicles and is then displaced slightly to the right by the aortic arch before branching into the main bronchi. It may move with the mediastinum if the heart is displaced, or it can be locally displaced (Figure 2.8).

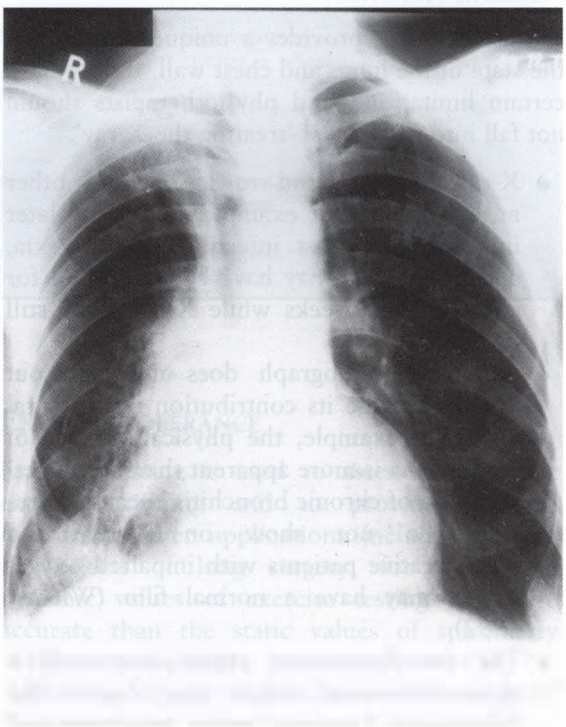


Figure 2.8 Fibrosis in the right upper lobe pulling the trachea to the right. Fibrosis and an abscess are visible in the right mid and lower zones. The patient has TB.

Heart

The heart, sandwiched between the lungs, is the main occupant of the mediastinum. Points to note are:

1. *Size*: The transverse diameter is normally less than half the internal diameter of the chest in the PA film. An apparently big heart could be the result of ventricular enlargement, pulmonary hypertension or poor inspiratory effort. A narrow heart is caused by hyperinflation, when the diaphragm pulls down the mediastinum (Figure 2.9), or it may be normal in tall thin people.
2. *Shape*: In right ventricular hypertrophy, the heart is boot-shaped, i.e. enlarged with the apex lifted off the diaphragm. A rounded heart might indicate pericardial effusion.
3. *Position*: The heart is normally extended slightly left of midline. If displaced, it is pushed away from, for example, a large pleural effusion (Figure 2.10) or tension pneumothorax (see Figure 14.10) and pulled towards a significant unilateral collapse (Figures 2.11, 2.12), resection (see Figure 10.12) or fibrosis (Figure 2.8).
4. *Borders*: These are obscured (silhouette sign) if there is a lesion abutting the heart, e.g. in middle lobe consolidation or collapse. A lower lobe lesion does not obliterate the border because it is on a different plane from the heart. Lower lobe collapse may show as flattening of the normally curved heart border ('sail sign') plus loss of clarity of the medial aspect of the hemidiaphragm.

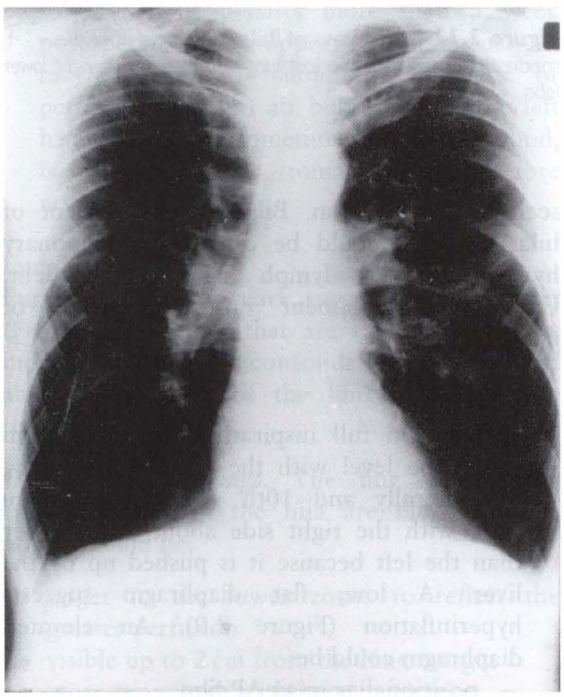


Figure 2.9 Hyperinflation. The dark lung fields, low flat diaphragm and narrow heart suggest that this patient has emphysema.

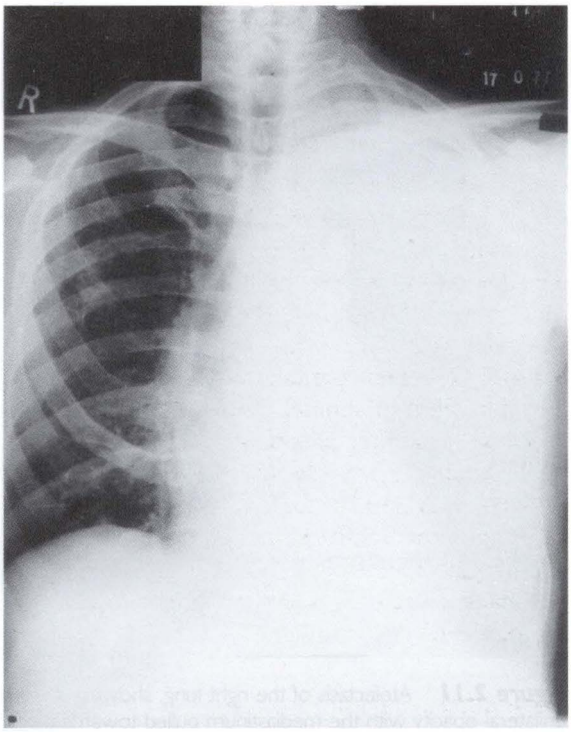


Figure 2.10 A large pleural effusion fills the left chest, obliterating the left lung and pushing the mediastinum away from the effusion. The patient has TB.

The term 'silhouette sign', strangely, means blurring of a border when air-filled lung on the same plane as the border is replaced by an

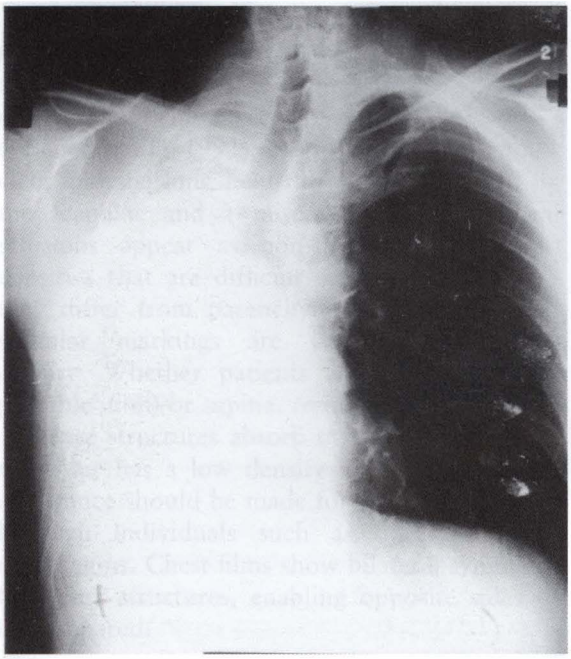


Figure 2.11 Atelectasis of the right lung, showing a unilateral opacity with the mediastinum pulled towards the collapse. The lung has collapsed because the right main bronchus is blocked by a tumour.

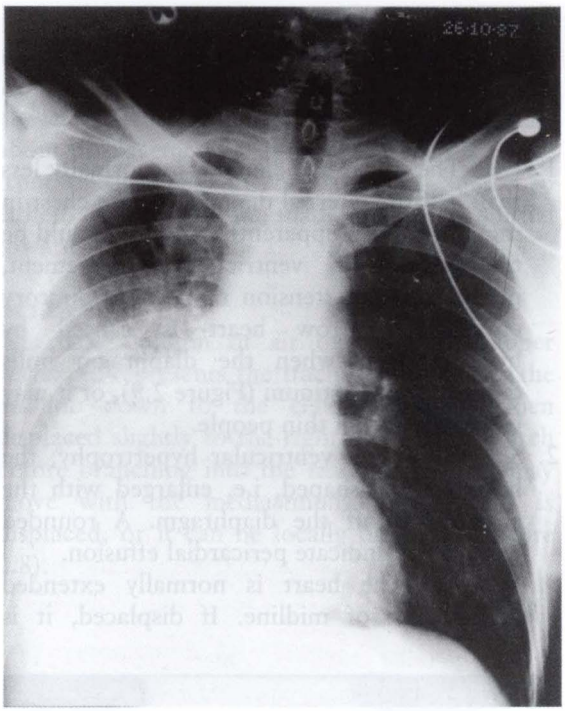


Figure 2.12 Atelectasis of RLL, showing shift of the mediastinum towards the lost lung volume. *RLL = right lower lobe.*

opacity. Specific lobes are collapsed or consolidated if the following borders are obscured:

- LLL: left hemidiaphragm
- RLL: right hemidiaphragm
- LUL: aortic arch
- RUL: right upper mediastinum
- lingula: left heart border
- middle lobe: right heart border.

(LLL = left lower lobe; RUL = right upper lobe, etc.)

Hila

Blood and lymph vessels make up the hilar shadows, the left hilum being slightly higher due to the left main pulmonary artery passing above the left main bronchus. Hila are elevated by upper lobe fibrosis, atelectasis or lobectomy and depressed by lower lobe atelectasis. Ring shadows near the hilum are normal large airways seen in cross-section. Bilateral enlargement of hilar shadows could be caused by pulmonary hypertension or lymph node enlargement. Unilateral enlargement raises suspicions of malignancy.

Diaphragm

1. *Height*: On full inspiration, the diaphragm should be level with the 6th rib anteriorly, 8th laterally and 10th posteriorly (Figure 2.1), with the right side about 2 cm higher than the left because it is pushed up by the liver. A low, flat diaphragm suggests hyperinflation (Figure 2.9). An elevated diaphragm could be:
   - positional as in an AP film
   - physiological due to lack of a full inspiration
   - pathological due to pressure from below,

e.g. abdominal distension, or a shrinking lung above, e.g. generalized lung fibrosis.

If one side of the diaphragm is raised, this could be due to lower lobe atelectasis, paralysed hemidiaphragm or, on the left, excess gas in the stomach.

2. *Shape*: The diaphragm should be dome-shaped and smooth. Flattening is caused by hyperinflation. Tenting is caused by fibrotic lungs pulling upwards. Loss of clarity of the smooth surface may be caused by lower lobe or pleural abnormality.
3. *Costophrenic angles*: These may provide the first clue to problems that lurk behind the dome of the diaphragm. The normal acute angle may be obliterated by the patchy shadow of consolidation or the meniscus of a small pleural effusion. 200 mL of fluid needs to accumulate in the pleura before blunting the costophrenic angle.
4. *Subphrenic*: Air under the right hemidiaphragm is expected after abdominal surgery. If it persists more than a week postoperatively, or appears spontaneously, it may indicate a subphrenic abscess or perforated gut. An air bubble under the left hemidiaphragm, sometimes containing fluid, is usually in the stomach and therefore normal.

Lung fields

Lungs that are too dark suggest hyperinflation (Figure 2.9). Lungs that are too white usually indicate infiltrates or consolidation. Normal and abnormal features of the lung fields are the following:

1. *Vascular markings*. The fine white lines fanning out from the hila are blood vessels, which should be:

- larger in the lower zones to reflect the greater perfusion
- visible up to 2 cm from the lung margin
- more prominent with poor inspiration.

A black non-vascular area demarcated medially by the white line of the visceral pleura

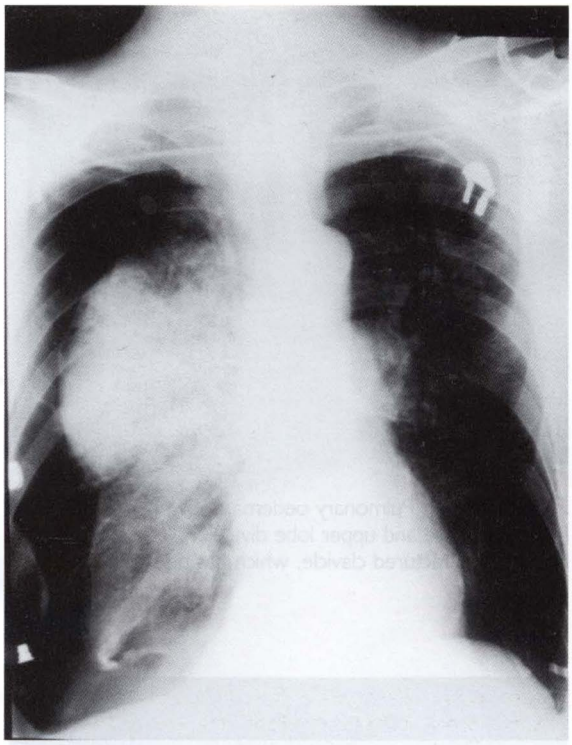


Figure 2.13 Right pneumothorax.

indicates a pneumothorax (Figure 2.13), sometimes seen more clearly when the film is turned horizontal. In conditions which reduce ventilation to the bases, such as COPD or pulmonary oedema, hypoxic vasoconstriction causes upper lobe diversion by squeezing blood from the bases to match the better ventilated upper lobes (Figure 2.14).

2. *Horizontal fissure*. If this is visible, it is opposite the right hilum and meets the 6th rib in the axilla. More than a 10° incline is considered abnormal.

3. *Diffuse shadowing*, e.g.:

- ground glass appearance, a hazy density like a thin veil over the lung, suggesting alveolar pathology
- reticular or a coarser honeycomb pattern, representing progressive damage in interstitial disease (Figure 2.15)

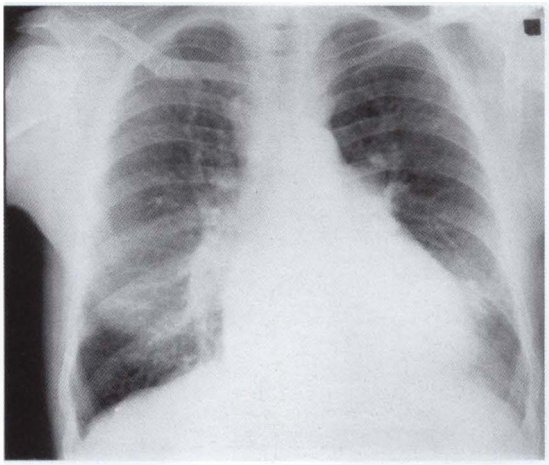


Figure 2.14 Pulmonary oedema, showing enlarged heart, hilar flare and upper lobe diversion. The patient has a coincidental fractured clavicle, which has been wired.

- perihilar pattern fanning out from the hila, suggesting pulmonary oedema (Figure 2.14)
- the snowstorm appearance of acute respiratory distress syndrome (see Figure 15.10).

4. *Localized opacities.* Consolidation is represented by a patchy opacity, often seen with pneumonia, and usually occupying a lobe or segment (see Figure 4.5). Bronchial tumours are usually located proximally, while metastases may be scattered. Streaky shadowing with some traction on moveable structures suggests fibrosis (Figure 2.8).

5. *Unilateral white-out.* Dense opacities can be caused by lung collapse (Figure 2.11) or pneumonectomy (see Figure 10.12), which pull the mediastinum towards the lesion, or a large pleural effusion, which pushes the mediastinum away (Figure 2.10).

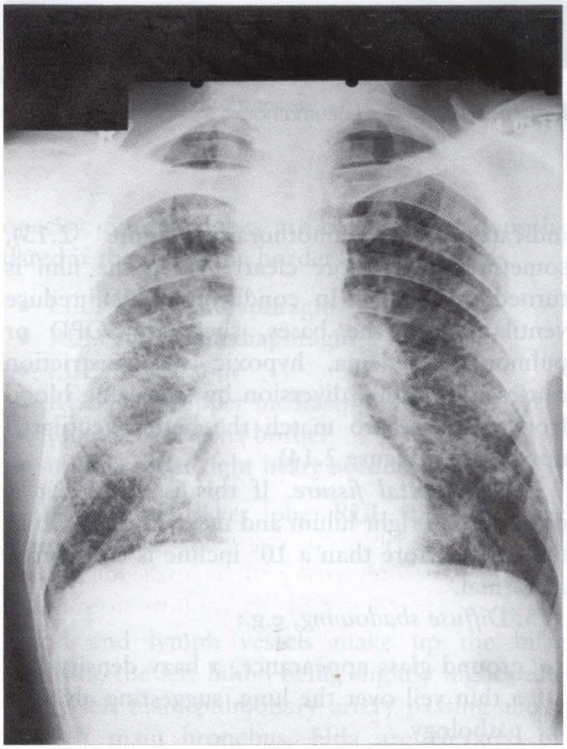


Figure 2.15 Interstitial lung disease, showing reticular pattern of lung fields and blurred heart borders.

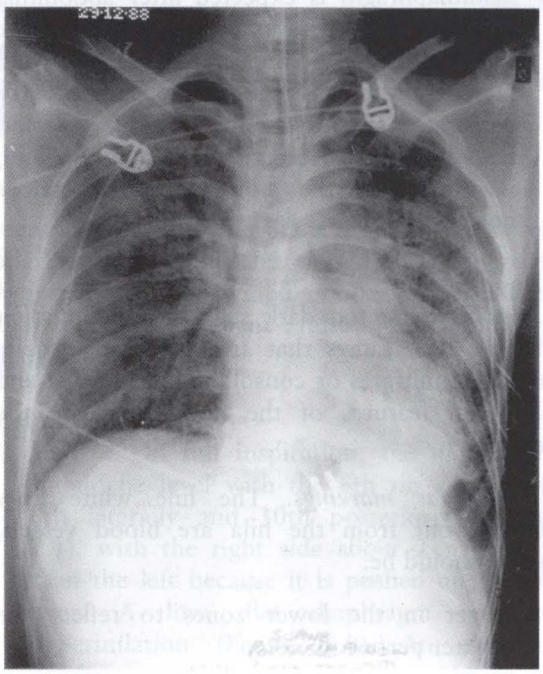


Figure 2.16 Diffuse shadowing of lung fields indicating generalised pneumonia. Ring shadow at left costophrenic angle is a bulla. Endotracheal tube and ECG leads are present.

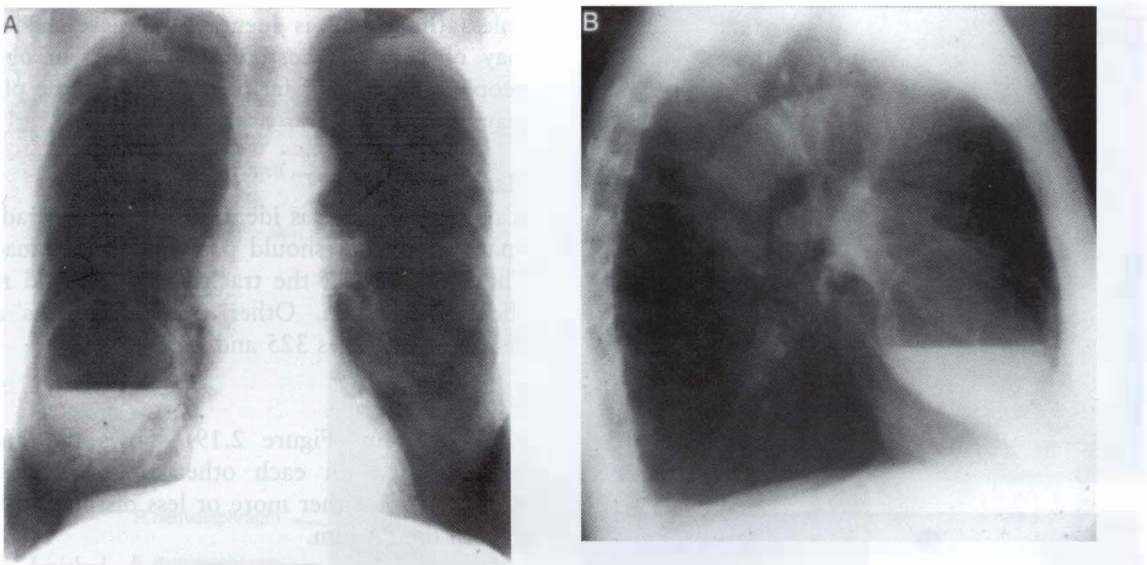


Figure 2.17 PA and lateral films showing a lung abscess in the posterior basal segment of the middle lobe.

6. *Ring shadows.* These represent:

- a bulla (Figure 2.16), which has a hair-line border, is air-filled and associated with emphysema or barotrauma
- a cyst, with a wall thickness over 1 mm, often associated with bronchiectasis
- an abscess, sometimes containing a fluid level (Figures 2.8 and 2.17).

7. *Air bronchogram.* Airways are visible if they are contrasted against an opacity (Figure 2.16). If an area of collapse has no air bronchogram, the airway is obstructed.

8. *Fluid line.* This is a horizontal line, sometimes with a meniscus at the edge, atop a dense opacity. If it spans the width of the lung, it suggests a pleural effusion (Figure 2.18).

Shadows caused by collapsed lung tissue may not be obvious, but atelectasis is indicated by shift of an adjacent structure (diaphragm, mediastinum, fissure, hilum) towards the collapsed area. There may also be crowding of vascular markings, with compensatory hyperaeration of adjacent lung which appears darker.

Bones

The bones are examined with care following cardiopulmonary resuscitation or other trauma, or if the patient is suspected of having osteoporosis or malignant secondary deposits. A fresh rib fracture is seen as a discontinuation of the border of the rib, to be distinguished from overlapping structures that can be misleading. Old fractures are identified by callous formation. Bony secondaries may appear as densities.

If a patient has fractured ribs and the film has not yet been reported, it is advisable to ask a radiologist to check the film before contemplating any positive pressure treatment because a pneumothorax may be hiding behind the cluster of rib shadows at the apex.

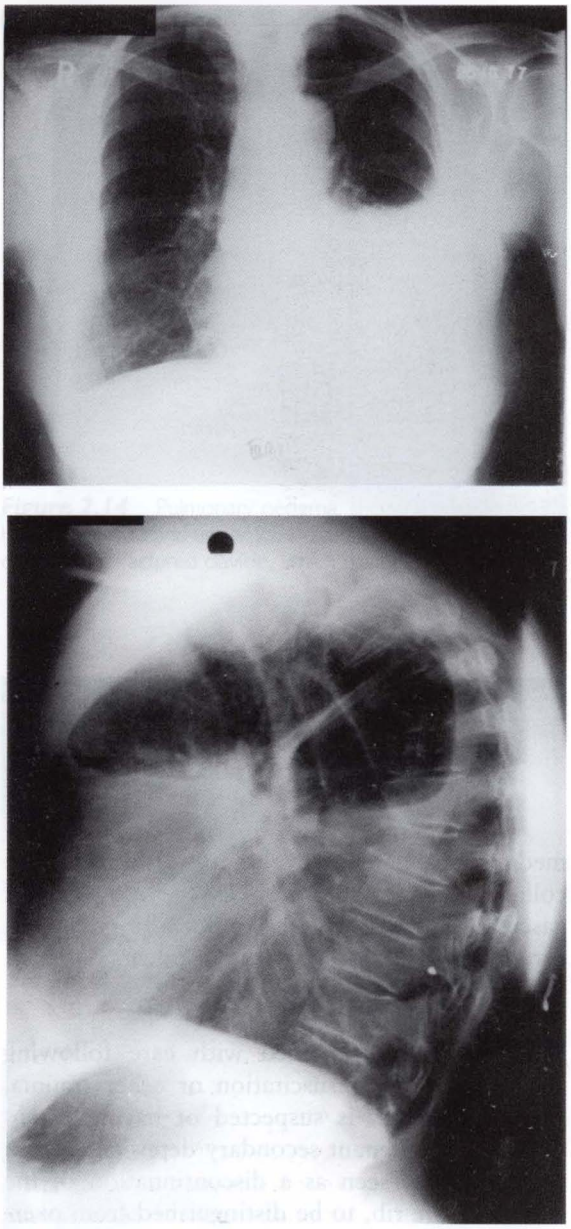


Figure 2.18 Lateral and PA films showing the fluid line of a pleural effusion. Lateral film shows fluid seeping up into the oblique fissure.

Soft tissues

Extrathoracic tissues cause shadows that project onto the lung fields and can cause confusion unless the origin is identified. Breast shadows may obscure the costophrenic angles in obese people, and rolls of fat pressed against the plate may be visible.

Hardware

A nasogastric tube is identified by its thin radio-opaque line and should pass into the stomach. The distal end of the tracheal tube should rest above the carina. Other tubes and lines are discussed on pages 325 and 329.

Lateral film

A lateral film (Figure 2.19) shows the lungs superimposed on each other so that various structures are either more or less distinguishable than in the PA film.

Lesions that were concealed behind the diaphragm or heart are now apparent, e.g.:

- lower lobe collapse may appear as a white triangle at the costophrenic angle
- a pleural effusion of just 50 mL can now blunt the costophrenic angle
- if the oblique fissure is visible, any lesion behind it is in the lower lobe.

Figure 2.20 shows middle lobe collapse, seen through the heart shadow as a shrunken opacity with clear margins indicating the fissures. The horizontal fissure is no longer horizontal because it has been pulled downwards by the collapsing middle lobe. Lateral films are also useful if accurate postural drainage is required, e.g. for an abscess.

Other tests

Fluoroscopy

Fluoroscopy projects moving images onto a monitor. Diaphragmatic paralysis can be identified.

Radionuclide imaging

A $\dot{V}/\dot{Q}$ scan maps the distribution of ventilation and perfusion in the lung. Radioactive gas is inhaled and then radioactive material is injected into the blood stream. The distribution of each

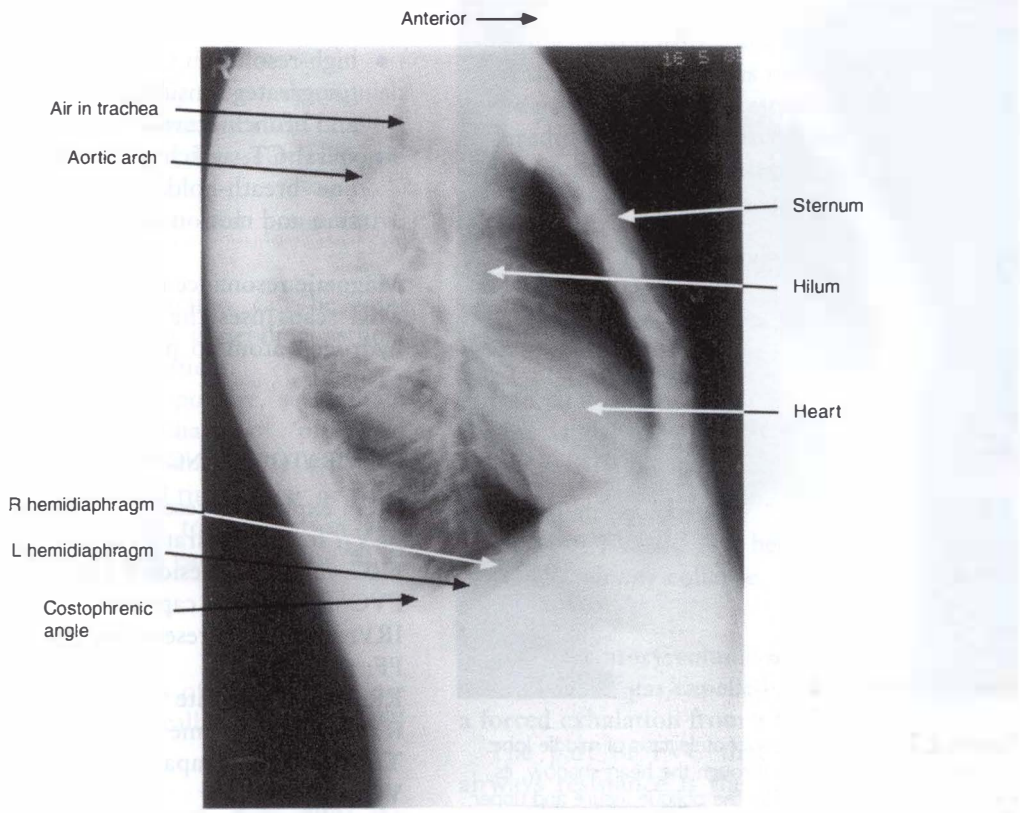


Figure 2.19 Lateral film of a normal lung. The aorta is seen arching above and behind the heart. Dark spaces in front and behind the heart are where the two lungs touch each other. The vertical white borders of the scapulae and the dark outline of the trachea can be seen. The patient has a tracheostomy bib.

is traced by gamma camera, and the two images are projected and compared. Areas of poor perfusion but good ventilation suggest pulmonary embolism or thrombosis (Figure 2.21).

Arteriography and bronchography

A pulmonary arteriogram is obtained by injecting contrast medium through a peripheral vein, via the right heart and into the pulmonary artery. This opacifies the pulmonary vascular tree and identifies pulmonary emboli. A bronchogram involves injecting a contrast medium into the airways to identify the dilated airways of bronchiectasis (see Figure 3.14). Angiography and bronchography have been largely superseded by the less invasive $\dot{V}/\dot{Q}$ scan.

Computed tomography (CT)

CT scans provide computed digital imaging from cross-sectional X-rays, viewed as if from the patient's feet. Computer manipulation of the data produces images in any plane, creating greater sensitivity to soft tissues than conventional X-rays without interference from overlying structures, at the cost of 100 times the radiation dose of a plain chest film. CT scans identify consolidation, atelectasis, abscesses, cavities, pleural effusions, bullae, the thick-walled dilated airways of bronchiectasis and the progressive destruction of emphysema (Morgan, 1992). They are particularly useful with pneumothorax (Engdahl, 1993) and hyperinflation conditions (Newman *et al.*, 1994).

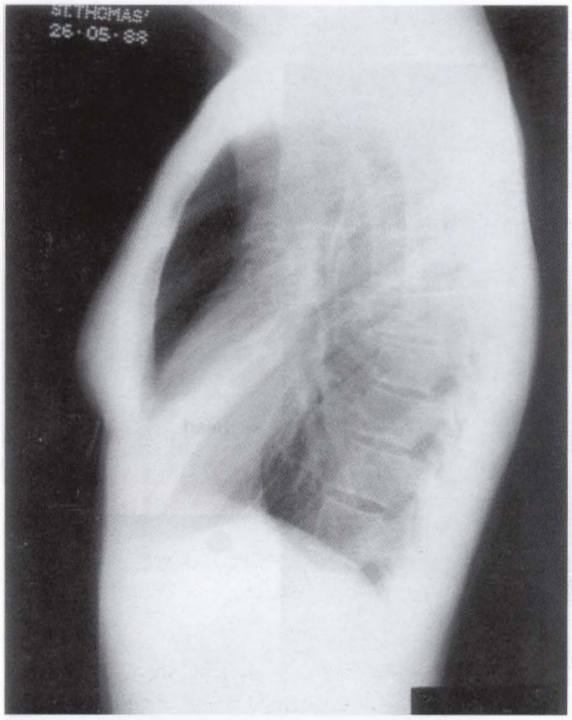


Figure 2.20 Lateral view of atelectasis of middle lobe. The shrunken lobe is seen through the heart shadow, its lower boundary bordered by the oblique fissure and upper boundary bordered by the horizontal fissure. The horizontal fissure is no longer horizontal because it has shifted downwards to take up the lost volume.

Variations are:

- high-resolution CT, which uses thinner slices for greater sensitivity to diffuse lung disease and bronchiectasis
- spiral CT, which scans the whole chest with one breath-hold, reducing radiation exposure and motion artefact due to breathing.

Magnetic resonance imaging (MRI)

MRI scans uses the magnetic properties of the hydrogen atom to produce clear images of soft tissues.

RESPIRATORY FUNCTION TESTS

ERV: expiratory reserve volume
FEV_1: forced expiratory volume in one second
FRC: functional residual capacity
FVC: forced vital capacity
IRV: inspiratory reserve volume
PF: peak flow
RR: respiratory rate
RV: residual volume
TLC: total lung capacity
VC: vital capacity
V_T: tidal volume
WOB: work of breathing

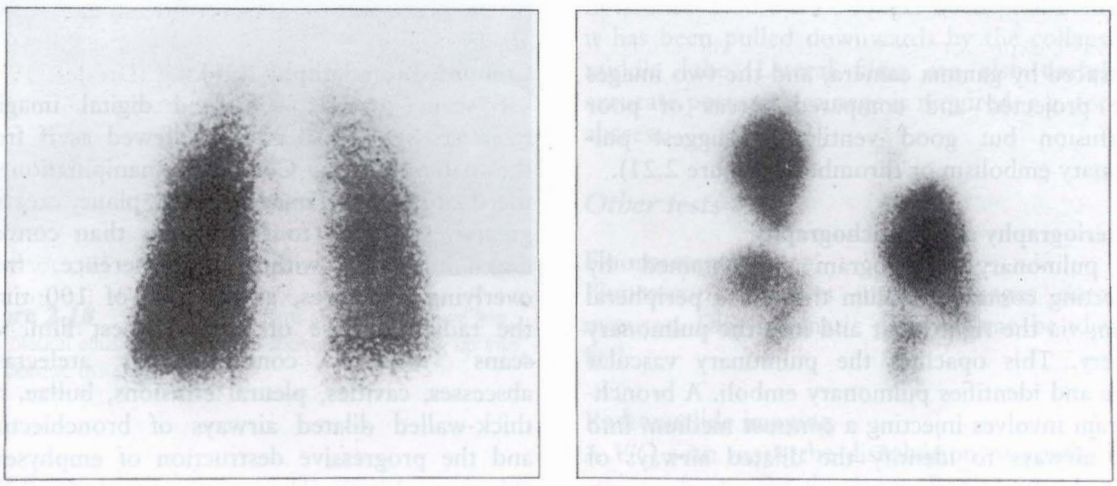


Figure 2.21 Ventilation–perfusion scan showing normal ventilation (left) and patchy abnormal perfusion (right), suggesting multiple pulmonary emboli.

Respiratory function tests (RFTs) quantify lung function in order to:

- define an abnormality, e.g. distinguish restrictive from obstructive disorders
- indicate the progress of a disease or response to treatment
- provide risk assessment and preoperative assessment.

Tests for airflow obstruction can be vital when used for detecting an impending asthma attack in an asymptomatic patient.

Measurements vary with posture, sex, ethnic origin, stature and age. Charts of 'predicted values' take these into account. Some measurements depend on fitness and time of day or year. Respiratory function tends to be best in late afternoon and worst in the early morning (Buff *et al.*, 1995).

Working definitions

If two or more subdivisions of lung volume are taken together, the sum is called a capacity. All values are approximate.

Peak expiratory flow or peak flow (PF): the highest flow that can be achieved during a forced expiration from full inspiration.

PF measures the ease with which the lungs are ventilated and reflects resistance in the large airways, expiratory muscle strength and effort. It is more useful in assessing the effectiveness of drug therapy for airflow obstruction than as a tool for clinical assessment (Holleman, 1995). It is one of the parameters measured on the flow–volume loop (p. 321).

- Normal value: 300–600 L/min
- Severe airways obstruction: 75–100 L/min.

Vital capacity (VC): the volume of gas that can be exhaled after a full inspiration (Figure 2.22).

VC represents the three volumes under volitional control (IRV, V_T, ERV, see definitions following) and is useful for measuring ventilatory reserve in a co-operative patient. It indicates ability to breathe deeply and cough, reflecting inspiratory and expiratory muscle strength. VC is sometimes reduced in obstructive disorders and always in restrictive disorders. It is also reduced by malnourishment (Lewis *et al.*, 1986) and obesity (Buckley, 1997). It is subject to day-to-day fluctuations.

- Normal: 3–6 L, or approximately 80% of TLC
- For adequate cough: > 1 L.

Forced vital capacity

Forced vital capacity (FVC): as above but with forced exhalation.

- Normal: equal to VC
- COPD: FVC < VC because the manoeuvre causes airway collapse.

Forced expiratory volume in one second (FEV_1): the volume of gas expelled in the first second by a forced exhalation from a full inhalation.

The part of FVC that is most sensitive to airways resistance is the first second of expiration. This is a similar measurement to peak flow but more accurate (Morice, 1998), although the accuracy of both measurements is limited in people with hyperinflated chests because of the effect of the deep breath on FRC (Pellegrino *et al.*, 1998).

Low FEV_1 relates to smoking (Dresler, 1996) and progression of obstructive lung disease. It is an important indicator of disease severity, but there is only weak correlation with breathlessness and quality of life.

Decline in FEV_1 averages 70 mL/yr in COPD and 5 ml/yr in asthma (de Guia, 1995). As with any forced manoeuvre, it is difficult for breathless patients to perform, may bring on bronchospasm in susceptible patients and is impaired when muscles are weakened by poor nutrition. It is subject to day-to-day fluctuations.

- Normal: 70–80% of VC, or 2–4 L
- Severe airways obstruction: < 60% predicted

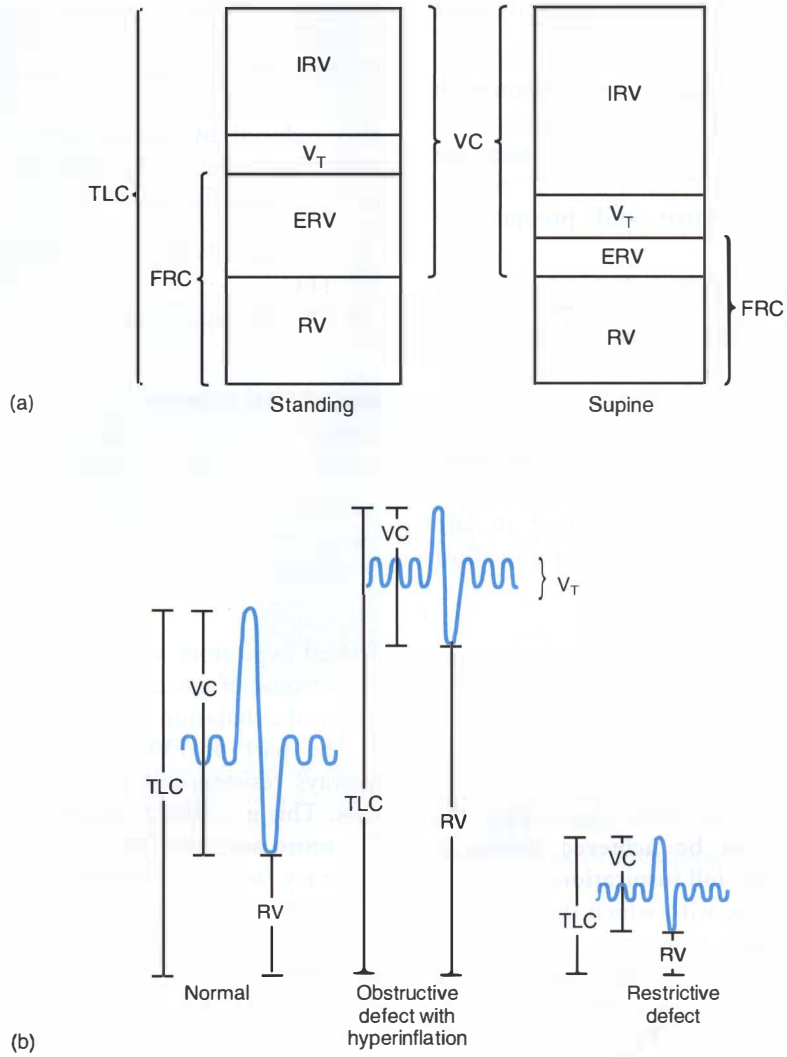


Figure 2.22 (a) Volumes and capacities. From Levitzky, M.G. (1999) *Pulmonary Physiology*, 5th edn, McGraw Hill, New York) (b) Variations for different disorders. Hyperinflated lungs show increased TLC, RV and FRC. Restrictive disorders show a decrease in all volumes.

FEV_1/FVC

This expresses FEV_1 in relation to vital capacity and is more accurate than FEV_1 alone.

- Normal: 70–80, i.e. FEV_1 = 70–80% of FVC
- Moderate airflow obstruction: 50–60
- Severe airflow obstruction: 30 (both values are reduced but there is a greater drop in FEV_1)
- Restrictive disease: up to 100 (both values reduced but a greater drop in FVC).

Total lung capacity (TLC) (Figures 2.22, 2.23): total volume of gas in the lungs after maximum inspiration.

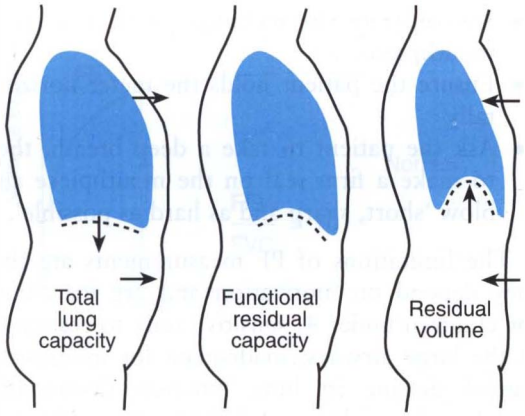


Figure 2.23 Volumes and capacities and the effect of a deep inspiration and deep expiration. Arrows represent the direction of chest wall movement. (From Luce, J. M. and Carver, B. H. (1982) Respiratory muscle function in health and disease. *Chest*, 81(1), 82–90, with permission.)

This is the sum of the four primary lung volumes (V_T, IRV, ERV, RV). Respiratory muscles need to generate transpulmonary pressures of 30–40 cmH_2O (MacIntyre, 1996).

- Normal: 3–8 L.

Functional residual capacity (FRC) (Figures 2.22, 2.23): the volume of gas remaining at the end of a tidal exhalation.

This is a useful indicator of lung volume because it does not depend on effort. It reflects the resting position when inner and outer elastic recoils are balanced. FRC decreases with restrictive disorders. It increases with air trapping, and the ratio of FRC to TLC is an index of hyperinflation.

One purpose of the large volume of FRC is to dilute extreme changes in alveolar oxygen tension with each breath.

- Normal in standing: 40% of TLC, approximately 2.4 L
- Normal in supine: up to 2.2 L
- COPD: up to 80% of TLC.

Tidal volume (V_T): the volume of air inhaled and exhaled during one respiratory cycle (Figure 2.22).

This reflects the functions of the respiratory centres, respiratory muscles and the mechanics of the lung and chest wall. Tidal volume mixes fresh gas with residual gas but never empties the lungs, as when the sea refreshes a tidal pool.

- Normal: 10% of VC, approximately 300–800 mL, average 7 mL/kg
- Increased to up to 50% of VC on exercise (Luce *et al.*, 1993, p. 21).

Inspiratory reserve volume (IRV): the extra volume of gas that can be inhaled voluntarily from end-inspiratory tidal volume (Figure 2.22).

This is usually kept in reserve, but is used during deep breathing. It is determined by inspiratory muscle strength, inward elastic recoil of the lungs and the size of the starting point (FRC + V_T).

- Normal: 3.1 L.

Expiratory reserve volume (ERV): the extra volume of gas that can be exhaled forcefully from end-expiratory tidal volume (Figure 2.22).

This is decreased with obesity, ascites or after upper abdominal surgery.

- Normal: 1.2 L.

Residual volume (RV): the volume of gas remaining in the lungs after maximum exhalation (Figure 2.22).

RV is inhaled with the first breath at birth and not exhaled until death, because the chest wall prevents the lungs emptying completely.

At low lung volumes, RV prevents the lungs collapsing, which would otherwise require a mighty inspiratory effort for reinflation.

RV is measured by gas dilution or body plethysmography. It is reduced with restrictive disease and increased with air trapping or age. The ratio of RV to TLC is an index of hyperinflation.

- Normal: 20–30% of TLC, average 1.2 L
- Hyperinflation: approximately 75% of TLC.

Minute volume/ventilation: The volume of gas breathed in or out per minute, i.e. $V_T \times RR$

- Normal: 5–7 L/min
- COPD: approximately 9 L/min
- Acute respiratory failure: approximately 10 L/min, but the patient may not be able to sustain the WOB required to maintain a stable P_aCO_2
- On brief hard exercise: up to 150 L/min.

Maximum voluntary ventilation (MVV): volume of air inhaled and exhaled with maximum effort over 15 seconds.

Correlates with FEV_1 but particularly relates to maximum ventilation on exercise. Susceptible to motivation. Reduced with smoking (Dresler, 1996).

- Normal: 50–200 L/min.

Measurement of airflow obstruction

Serial measurements should be taken on well-rested patients in the same posture at the same time each day. This minimizes the normal daily variation, which can be greater than the improvement with bronchodilator drugs (Burge, 1992). In asthma, peak flow and FEV_1 are lowest in the early morning.

Peak flow

A peak flow meter provides a quick and simple indication of airways obstruction. Three tests are performed, with a rest in between, and the best is recorded. Suggested guidelines are:

- Ask the patient to avoid tight clothes, a full bladder, vigorous exercise within the last 30 minutes, a heavy meal within 2 hours and, if possible, smoking within 24 hours (BTS, 1994)
- Explain the purpose and technique of the test, because the meter is effort-dependent and reliability depends on the patient understanding and not feeling hurried
- Have the patient seated upright if possible, avoid occluding the exhaust holes, check the pointer is at zero
- Demonstrate the technique with a separate mouthpiece
- Ensure the patient holds the meter horizontally
- Ask the patient to take a deep breath, then to make a firm seal on the mouthpiece and blow 'short, sharp and as hard as possible'.

The limitations of PF measurements are that they depend on motivation and are inaccurate for children under 4, sensitive only to resistance in the large airways, inadequate for monitoring annual decline in lung function (Tirimanna, 1996) and variable in reliability at middle and high flows (Miller and Ouanjer, 1994). PF meters should be tested regularly, the portable models replaced annually, and the same device used for the same patient. They are available on prescription in the UK.

Spirometry

A spirometer such as the Vitalograph is used to measure FEV_1 and FVC (Figure 2.24). It is more tiring than measuring peak flow, and values vary with posture and effort.

Instructions are similar but, instead of a short sharp blow, patients are exhorted to 'blow the living daylights out of the machine and keep blowing until your lungs are empty'. Much uninhibited encouragement is required, repeated on subsequent measurements. Nose clips are not necessary (Pina *et al.*, 1997).

If a relaxed, not forced, vital capacity is required, the patient blows out from maximal inspiration but at a comfortable and sustained speed until no more can be exhaled (BTS, 1994).

Further measurement of large airways resistance

Airflow resistance in the large airways depends on flow at the mouth and the pressure difference between mouth and alveoli. Flow at the mouth is measured by a pneumotachograph, which detects the pressure drop across a slight resistance placed in the airstream. Pressure difference is measured in an airtight body box called the plethysmograph.

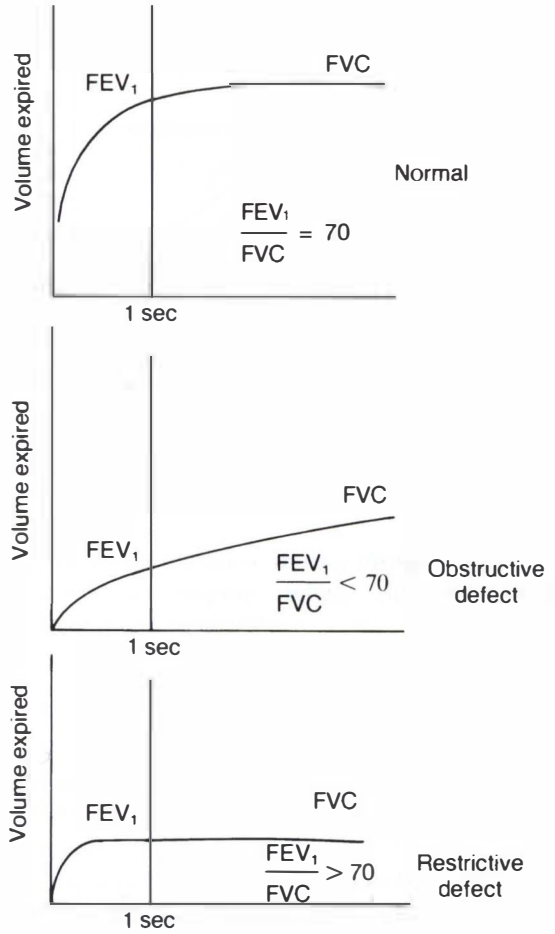


Figure 2.24 Spirograms. Normal trace shows most of the FVC expelled within 1 second. The decreasing slope of the curve is caused by progressive airway compression and lower elastic recoil as the subject exhales. **Obstructive pattern** shows prolonged expiration. **Restrictive pattern** shows reduced FVC, all of which is expelled within 1 second due to augmented recoil.

Further measurement of small airways resistance

Detection of resistance in the 'silent zone' of the small airways, where changes occur in the early stages of COPD, can improve the success of treatment before changes are irreversible.

The flow-volume loop records flow and volume during forced inspiration and expiration (Figure 2.25).

During inspiration, flow is dependent on effort throughout. During expiration, the highest flow occurs initially, where it is effort-dependent and represents large airway function. After a small proportion of VC has been expired, flow is independent of effort because of greater dynamic compression with more positive intrapleural pressures (Levitzky, 1995, p. 47). Flow then depends solely on elastic recoil and small airways resistance. In obstructive disease, expiratory flow shows a concave appearance representing sudden attenuation of expiration as floppy airways collapse or narrowed airways obstruct. Interstitial restrictive disease shows rapid flow during exhalation due to stiff lungs.

Maximum mid-expiratory flow (MMEF, MEF_{50} or FEF_{25-75} or FEF_{50}) is the mean forced expiratory flow during the middle half of FVC and is used to measure small airways resistance. It is independent of effort because only the inspiratory phase and first 25% of the expiratory phase is effort-dependent.

Measurement of lung volumes

The lung cannot be completely emptied voluntarily and always retains a residual volume of gas, so lung volumes are measured indirectly. FRC is estimated by one of the following:

- plethysmography: air in the chest is compressed and lung volume calculated from the change in pressure
- gas dilution: air in the lungs is mixed with an inert gas such as helium, the dilution of which gives an indication of lung volume
- nitrogen washout: the nitrogen content of air is known to be about 80%, and lung volume can be calculated by having the patient breathe nitrogen-free gas and measuring the expired nitrogen.

TLC and RV can be measured by using one of these measurements plus spirometry.

Table 2.6 compares the RFTs for obstructive and restrictive lung disease.

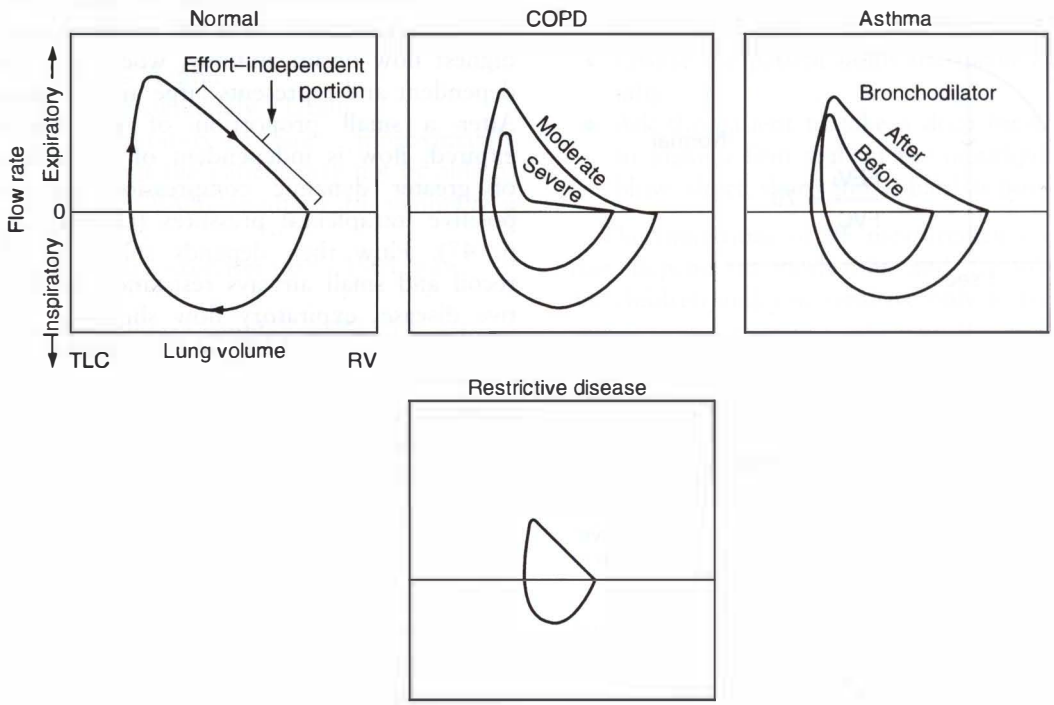


Figure 2.25 Flow–volume loops. The inspiratory loop is below the line and the expiratory loop above the line. Increasing severity of obstructive lung disease (asthma and COPD) is reflected by increasing concavity of the effort-independent portion of the expiratory curve. Restrictive pattern is represented by a small loop and rapid expiration.

Gas transfer

Gas transfer (transfer factor) indicates the transfer of gas by measuring the surface area of the alveolar–capillary membrane. The total lung transfer capacity for carbon monoxide (TLCO) is measured by the patient taking a single deep breath of a gas that includes carbon monoxide, breath-holding for 10 seconds, then exhaling. The amount of expired carbon monoxide reflects its diffusion across the alveolar–capillary membrane.

Reduced TLCO indicates $\dot{V}_A/\dot{Q}$ abnormality, low haemoglobin, advanced age or impaired diffusion due to an abnormal alveolar–capillary membrane, especially in relation to smoking (Dresler, 1996). TLCO is closely correlated with exercise limitation (Wijkstra, 1994) and breathlessness due to emphysema or lung fibrosis (O'Donnell and Webb, 1992). It is reduced in bronchiectasis and emphysema. It is low in heavy smokers (who have excess carboxyhaemoglobin), malnutrition and anaemic states, and high in polycythaemia.

Table 2.6 Effect of obstructive and restrictive disease on volume and flow measurements

	Obstructive	Restrictive
Tidal volume	N	N or ↓
VC	N or ↓	↓
Peak flow	↓	N or ↓
FEV_1	↓	N
FVC	N or ↓	↓
FEV_1/FVC	↓	N or ↑
RV	↑	N or ↓
FRC	↑	↓
TLC	↑	↓

Gas transfer is affected by diffusion properties, alveolar volume and capillary blood. The old term 'diffusing capacity' is less accurate because it encompasses only the passage of gas from alveoli to blood.

Respiratory muscle function

Inspiratory muscle strength is proportional to exercise capacity (Wijkstra, 1994). Bilateral paralysis or severe weakness of the diaphragm shows the following signs:

- orthopnoea unexplained by heart or lung disease
- accessory muscle activity unexplained by lung disease
- abdominal paradox during inspiration, especially in supine when the weakened diaphragm is unable to counteract pressure from the abdominal contents
- postural fall in vital capacity of 50% in supine compared to upright (Tobin and Yang, 1990)
- symptoms of nocturnal hypoventilation such as morning headache and daytime somnolence
- non-specific symptoms such as breathlessness or recurrent chest infections.

Bilateral diaphragmatic paralysis effectively removes a portion of the chest wall. When upright, patients exhale by contracting the abdominal muscles to push up the diaphragm, then allow passive inspiration by relaxing them. Unilateral diaphragmatic paralysis shows nocturnal hypoxaemia due to $\dot{V}_A/\dot{Q}$ mismatch in supine, unilateral abdominal paradox on sniffing and a raised hemidiaphragm on X-ray.

The following tests for inspiratory and expiratory muscles are suitable for patients who are able to co-operate:

- Vital capacity (VC) is simple but relatively insensitive and non-specific. Small pressures are required to inflate the lung and a fall in VC only occurs with severe muscle weakness. Results are influenced by effort, fitness and compliance of the lung and chest wall.
- Maximum static mouth pressures (Chatham *et al.*, 1994) can measure inspiratory or expiratory pressures:
  - maximum inspiratory pressure (MIP), indicating strength of the inspiratory muscles is measured from either RV or FRC and maintained for one second
  - maximum expiratory pressure (MEP), indicating strength of the expiratory muscles, is measured from TLC.

  A pressure gauge or transducer is connected to a mouthpiece, the patient inhales or exhales sharply, keeping a firm lip seal and taut cheeks, and the best of three efforts is recorded. A small leak in the system prevents inspiratory mouth suction, which would give artificially high readings for MIP. Technique must be meticulous and patient position standardized because normal values vary 10-fold (McKenzie, 1994) as a result of variations in:
  - initial lung volume
  - learning effect of the test
  - patient effort
  - ventilatory drive
  - nutritional status.

  MIP and MEP above 80 cmH_2O indicate adequate inspiratory muscle strength and ability to cough respectively. High values exclude muscle weakness but the reverse is not necessarily true because low values may be due to insufficient patient co-operation (Siafakas *et al.*, 1999).

Other tests include the following:

- For non-paralysed ventilated patients, inspiratory strength can be measured with a one-way valve, after explaining to the patient that the airway will be briefly occluded (Wilkins *et al.*, 1995, p. 257).
- The following non-volitional tests measure the strength of the diaphragm only (Harris and Moxham, 1998):
  - transdiaphragmatic pressure is obtained

by comparing oesophageal (pleural) and gastric (abdominal) pressures, using swallowed balloons
  - phrenic nerve stimulation or relaxation rate of muscle are relatively accurate measurements
  - magnetic nerve stimulation monitors the progression of muscle weakness and assists diagnosis.
- Nocturnal hypercapnia suggests that inspiratory muscle strength is below 30% of normal (Hahn *et al.*, 1997).

Respiratory muscle endurance is difficult to measure and cannot necessarily be inferred from strength. It is assessed by MVV (p. 58), which is influenced by co-ordination, pulmonary mechanics and effort. It is not valid in people with COPD who are unable to generate high flow rates (Hopp *et al.*, 1996).

Other tests

Oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$)

$\dot{V}O_2$ and $\dot{V}CO_2$ are measured by analysis of inhaled and exhaled gas volumes. When compared to predicted values for age and sex, aerobic and anaerobic contributions to metabolic activity can be assessed.

Maximum oxygen consumption ($\dot{V}O_{2\ max}$)

$\dot{V}O_{2\ max}$ quantifies maximum exercise tolerance by indicating the point at which the anaerobic threshold is reached. Normal $\dot{V}O_{2\ max}$ is > 25 mL/kg/min, or 25 times the resting level, representing the point at which oxygen demand exceeds availability and lactic acid is produced. It is an exhausting test that entails measuring expired air while workload is increased. Less

Box 2.1 Checklist of the major points of pulmonary assessment

Patient's notes
- History (past, present, social, occupational)
- Investigations
- Risk factors

Charts
- Temp, BP, HR, RR
- Medication
- Oxygen prescription
- Oxygen saturation
- Arterial blood gases
- Peak flow
- Fluid balance

Subjective assessment
- Symptoms
- Functional limitations

Observation
- Appearance
- Colour
- Hands
- Oedema
- Chest shape
- Respiratory rate
- Breathing pattern
- Sputum

Equipment
- Oxygen, oxygen analyser, oximeter
- Humidification
- Drips, drains, chest drains
- Monitors, ventilator

Palpation
- Abdomen
- Chest expansion
- Percussion note
- Hydration

Auscultation
- Breath sounds
- Added sounds
- Voice sounds

Exercise tolerance

Chest X-ray

distressingly, submaximal $\dot{V}O_{2\ max}$ can be used to estimate $\dot{V}O_{2\ max}$. $\dot{V}O_{2\ max}$ is not a reliable guide to aerobic capacity in patients with respiratory disease if peak exercise is limited by breathlessness. It is expensive and lengthy, and is normally used for research or clinical purposes such as assessing suitability for lung resection (Bolliger, 1998).

Oxygen cost of breathing

This is assessed by determining the total $\dot{V}O_2$ at rest and the increased level of ventilation produced by hyperventilation. The added oxygen uptake is attributed to metabolism of the respiratory muscles.

MINI CASE STUDY: MR TA

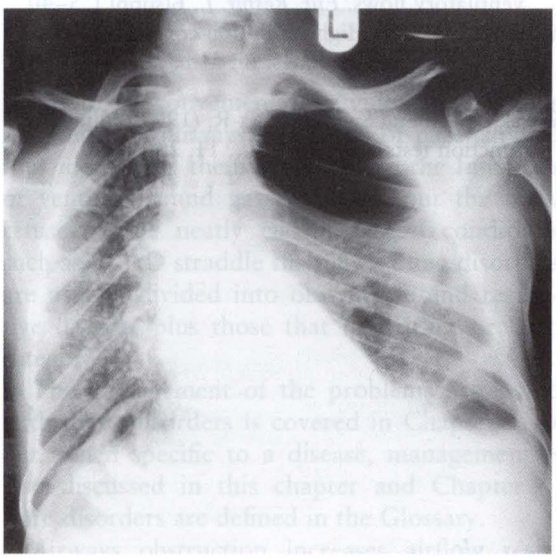


Figure 2.26 Mr TA.

Identify this young man's problems after he has returned to the ward following lengthy mechanical ventilation and difficult weaning. Then answer the questions.

Background

Impaired swallowing due to cerebral palsy.
Lives at home with his mother who is his carer.
Fully dependent.

Subjective

Unable to speak because of cerebral palsy.
Patient's mother says he feels tired and would like to go home. He has not eaten for a week since returning to the ward.

Objective

Cachectic.
Kyphoscoliotic.
Curled up in bed.
Breathing pattern shallow but not distressed.
Chest clear.
Observations normal.

Questions

1. Analysis?
2. Patient's problems?
3. Goals?
4. Plan?

RESPONSE TO MINI CASE STUDY

1. Analysis

Figure 2.26 shows a pneumothorax in the left upper zone and bulla in the left lower zone, probably due to protracted mechanical ventilation and malnutrition. The radiograph also shows diffuse opacities, probably due to multiple aspirations of food into the lungs because of difficulty swallowing. Kyphoscoliosis is due to cerebral palsy. Lung problems would not be directly responsive to physiotherapy.

2. Problems

Swallowing difficulties and malnutrition.
Immobility.

3. Goals

Improve nutrition through multidisciplinary teamwork.
Optimize mobility.
Rehabilitate to home circumstances.

4. Plan

Liaise with speech–language therapist, doctor, head nurse, dietician

Daily written programme of bed mobility exercises, using diary to document progress
Liaise with patient's mother over manual handling
Liaise with social worker and occupational therapist for home support.

LITERATURE APPRAISAL

Comment on the logic of the following statement in a study evaluating exercise performance:

> *[We] examined ... leg muscle training, inspiratory muscle training and postural drainage ... the largest effect occurring with leg muscle training.*
>
> *Austr. J. Physiother.* 1992; 38: 189–193

RESPONSE TO LITERATURE APPRAISAL

Not surprising. Training is specific. Exercise performance would not be expected to improve with postural drainage. Exercise performance may or may not improve with inspiratory muscle training, depending on the limiting factors.

RECOMMENDED READING

Hodgkinson, D. W., O'Driscoll, B. R. and Driscoll, P. A. (1993) Chest radiographs. *Br. Med. J.*, **307**, 1202–1206, plus correction: *Br. Med. J.*, **307**, 1417.

McCord, M. and Cronin, D. (1992) Operationalizing dyspnea: focus on measurement. *Heart Lung*, **21**, 167–179.

Muldoon, M. F. (1998) What are quality of life measurements measuring? *Br. Med. J.*, **316**, 542–545.

Pasterkamp, H. (1997) Respiratory sounds. *Am. J. Respir. Crit. Care Med.*, **156**, 974–987.

Quanjer, P. H. (1993) Lung volumes and forced ventilatory flows. *Eur. Respir. J.*, **6**(suppl.), 5–40.

Worthy, S. (1995) High resolution computed tomography of the lungs. *Br. Med. J.*, **310**, 615–616.

Zurek, A. M. and Swinburn, C. R. (1995) Pulmonary function tests. *Care Crit. Ill.*, **11**, 230–234.

3 Obstructive disorders

Summary

Introduction

It would be convenient to divide lung diseases into those of airways and those of parenchyma, thus identifying them according to the functions of ventilation and gas exchange, but the body refuses to be neatly classified, and conditions such as COPD straddle the fence. Lung disorders are usually divided into obstructive and restrictive disease, plus those that fit neither or both categories.

The management of the problems associated with these disorders is covered in Chapters 5–9 but, when specific to a disease, management is also discussed in this chapter and Chapter 4. Rare disorders are defined in the Glossary.

Airways obstruction increases airflow resistance and the work of breathing, as indicated by decreased peak flow rates. Causes are:

- reversible factors, e.g. inflammation, bronchospasm or mucus plugging
- irreversible factors, e.g. fibrotic airway walls or floppy airways as a result of loss of the elastic recoil that normally supports them (Figure 3.1)
- localized lesions, e.g. upper airway tumour or foreign body.

Chronic obstructive pulmonary disease

> *The insidious onset, lacking the jolt of a first heart attack, may take away its ability to provide a sharp motivational shock.*
>
> Jarvis, 1995

The common disease entity of chronic bronchitis and emphysema is known as COPD (chronic obstructive pulmonary disease), COAD (chronic obstructive airways disease), CAO (chronic airflow obstruction) or CAL (chronic airflow limitation). Asthma can overlap with COPD (Figure 3.2) but is usually classified separately, even though it is a chronic obstructive disease of the airways. COPD is a slowly progressive disease and most airways obstruction is fixed, although some reversibility may be demonstrated with medication (O'Driscoll, 1997).

COPD is laden with gloomy statistics:

- It is the third most common cause of certified illness in the UK (Gravil *et al.*, 1998)
- It is the fifth greatest cause of disability worldwide (WHO, 1996)
- It is the only major cause of death increasing in prevalence (Oh, 1997, p. 228)
- It is common in elderly people but often

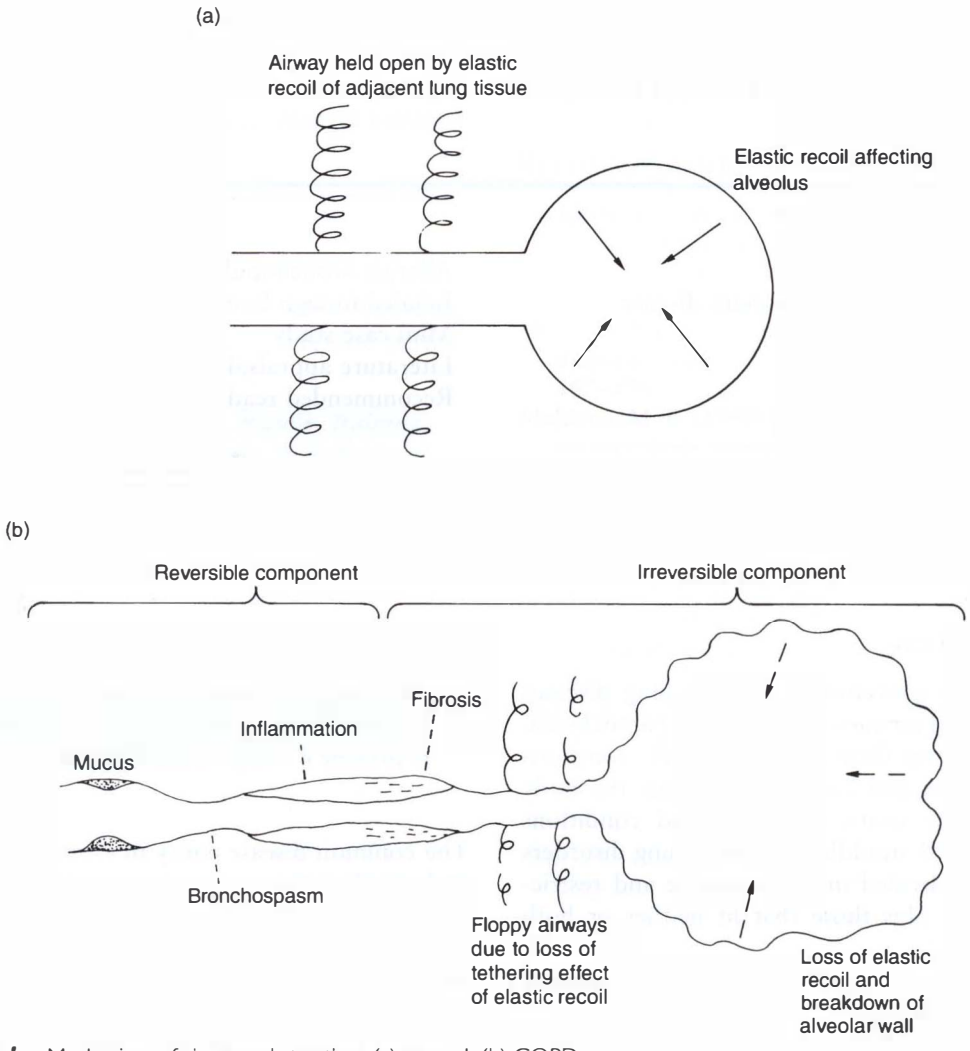


Figure 3.1 Mechanism of airways obstruction: (a) normal; (b) COPD.

underdiagnosed and undertreated despite its 89 symptoms (Kinsman *et al.*, 1983)
- It is largely preventable (Huib, 1999).

Causes

Smoking is the major contributor to COPD. Risk factors are poverty (Prescott, 1999) and being male, which are both associated with smoking. Other factors are occupation, housing, climate, childhood respiratory illness (Clarke, 1991) and *in utero* exposure to smoking or malnourishment (Barnes, 1995).

Pathophysiology

Chronic bronchitis

Chronic bronchitis is a disease of the airways. It is characterized by excess mucus secretion and productive cough. The cough is called a smokers' cough in the early stages but once mucus production has been excessive for 3 months a year for over 2 years, this becomes the inadequate but traditional definition of chronic bronchitis.

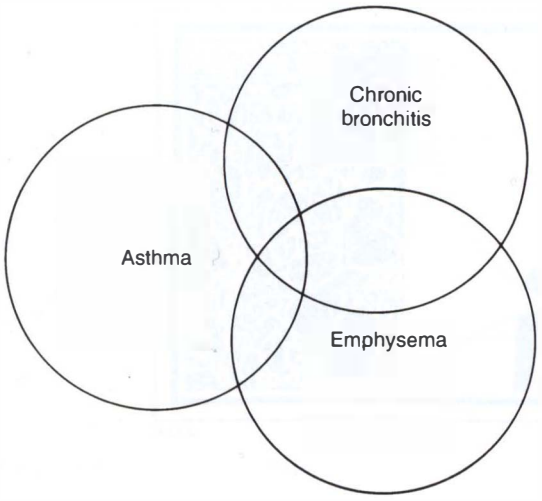


Figure 3.2 Relationship between the commonest obstructive lung diseases.

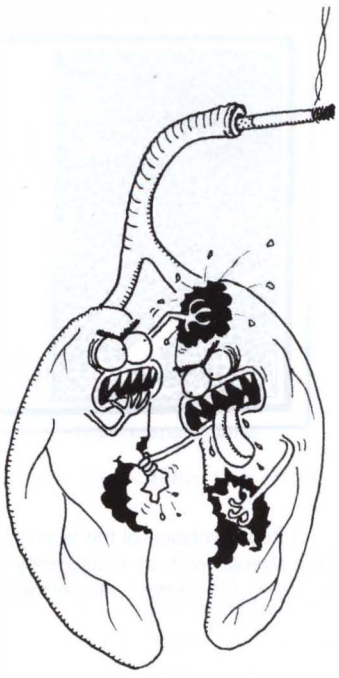


Figure 3.3 Destruction of the alveolar walls caused by smoking. (From Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford, with permission. Artist: James Northfield)

Repeated inhalation of tobacco smoke irritates the sensitive lining of the airways, leading to inflammation, mucus hypersecretion and sometimes bronchospasm. Inflammation is the key process. It causes narrowing first in the distal small airways and then in the proximal large airways. Acute inflammation resolves but chronic inflammation leads to fibrotic changes and scarring. Mucus hypersecretion due to mucosal damage is associated with a rampant increase in the size and number of mucus-secreting goblet cells. Excess mucus has traditionally engaged the attention of physiotherapists but causes little overall airways obstruction and does not correlate with physiological disturbances (Faling, 1986) nor mortality (Wiles and Hnizdo, 1991). Breathlessness is more significant to the patient and more related to inflammatory damage and airway narrowing (Bach and Haas, 1996). Bronchospasm, when present, is thought to be caused by acetylcholine release due to inflammatory stimulation of the parasympathetic nervous system.

Emphysema

Emphysema usually occurs with chronic bronchitis and shares a similar aetiology, but is primarily a disease of alveoli and smallest airways, with secondary effects on other airways. It is usually caused by damage to the alveoli from smoking. Occasionally a congenital lack of alpha$_1$-antitrypsin causes primary emphysema in earlier life (Wencker *et al.*, 1998).

Protein breakdown is the villain of emphysema (Figure 3.3), leading to erosion of alveolar septa, dilation of distal airspaces (Figure 3.4) and destruction of elastic fibres.

The walls of the terminal bronchi are normally supported by radial traction exerted by alveolar septa, but loss of elastic tissue means that, during expiration, compressive forces are not opposed by radial traction and the floppy airways collapse (Figure 3.1).

Two types of emphysema are described, although they may coexist. Centrilobular emphysema affects mainly the respiratory bronchioles. Panlobular/panacinar emphysema affects the alveoli.

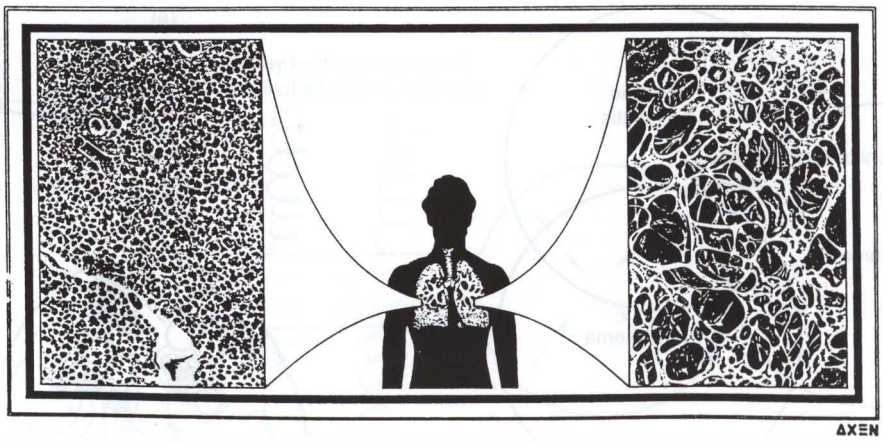


Figure 3.4 (a) Representation of the tight sponge-like appearance of a healthy lung. (b) The large air spaces resulting from destruction of the alveolar walls by emphysema. (From Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester, with permission.)

The obstructed airways of emphysema lead to hyperinflation by two mechanisms:

- *Passive hyperinflation* is caused by reduced elastic recoil, which allows the airways to collapse on expiration, causing gas trapping.
- *Dynamic hyperinflation* is caused by the patient having to actively sustain inspiratory muscle contraction in order to hold open the airways (McCarren, 1992). This unfortunate but necessary process is achieved at the cost of excess work of breathing (WOB), a barrel chest, reduced diaphragmatic contribution to breathing and a lung volume that can exceed the predicted TLC (Decramer, 1997).

Airways obstruction reduces expiratory flow, which prevents expired air from being fully expelled before the next inspiration starts, causing air trapping distal to the obstructed airways and positive pressure in the chest known as intrinsic PEEP (Figure 3.5), especially during exacerbations or with rapid breathing. The lungs are prevented from emptying to their usual relaxed volume between inflations by an average positive pressure of 2 cmH_2O (Ninane *et al.*, 1993). This imposes an extra threshold load at the start of inspiration because the inspiratory muscles have to offset this positive pressure before inspiration can begin (Ninane, 1997). It also hinders cardiac output and impairs perfusion to the labouring inspiratory muscles (Kawagoe, 1994). Stabilization occurs at volumes and pressures that are higher than normal, which reduces lung compliance (see Figure 1.3). The distended alveoli require a greater than normal pressure for inflation, thus overturning the old concept that emphysematous lungs are overcompliant (Macklem and Eidelman, 1990).

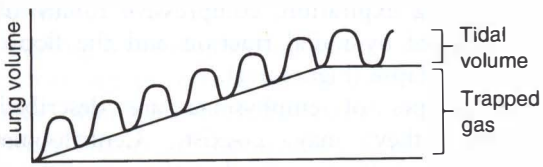


Figure 3.5 Development of intrinsic PEEP. The sloping line indicates FRC.

Excess WOB is required to:

- overcome the resistance of obstructed airways
- assist expiration, which becomes active

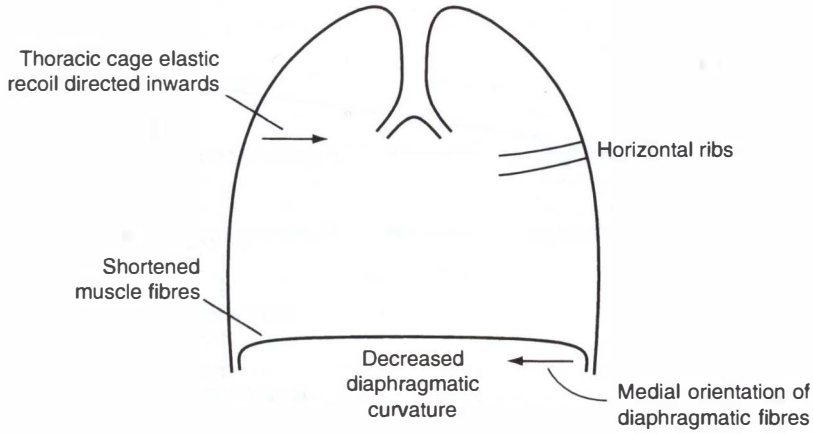


Figure 3.6 The detrimental effects of hyperinflation on the mechanics of breathing. (From Tobin, M. J. (1988) Respiratory muscles in disease. *Clinics in Chest Medicine*, 9, 263–286, with permission.)

rather than passive when air has to be forced out through narrow airways

- sustain inspiratory muscle action throughout the respiratory cycle so that high lung volumes are maintained, alveoli being opened at a high point on the compliance curve (see Figure 1.3)
- compensate for the altered geometry and interaction of the respiratory muscles, the flat diaphragm having to work paradoxically by pulling in the lower ribs on inspiration, thus becoming expiratory in action (Hoover's sign)
- compensate for loss of the bucket handle action of the ribs (Figure 3.6)
- compensate for reversed action of rib cage recoil, which in the hyperinflated chest is directed inwards rather than outwards, thus resisting instead of assisting inspiration (Figure 3.6)
- overcome threshold resistance at the start of inspiration, caused by intrinsic PEEP.

Some patients can only inhale by lifting up their entire rigid rib cage with their accessory muscles. These accessory muscles have a dual role when unsupported arm activities are required. Excess use of accessory muscles increases the sensation of breathlessness because these muscles are richly supplied with muscle spindles and tendon organs to increase afferent feedback (Chatham, 1995). Normal muscle is able to respond to increased load by hypertrophy, but an emphysematous diaphragm often labours under further handicaps such as malnutrition, and diaphragmatic weakness is common (Duranti, 1995). Although the patient often feels exhausted, the diaphragm itself may avoid fatigue (Mador *et al.*, 2000).

Malnourishment is common, caused by excess energy demand and impaired energy supply (p. 131). This leads to cannibalism of the respiratory muscles for their protein. Malnutrition accelerates the process of emphysema (Schlichtig and Sargent, 1990) and is an independent risk factor for mortality (Landbo, 1999). Muscles are weakened further by physical inactivity, chronic heart failure, electrolyte imbalance and prolonged steroid use (Heijden *et al.*, 1996). People with emphysematous disease show more than twice as much oxygen cost of breathing as those with chronic bronchitis (Jounieaux, 1995).

Chronic bronchitis and emphysema

The gradual patchy airway narrowing of chronic bronchitis, augmented by the floppy airways of emphysema, leads to uneven distribution of ventilation. Damaged alveoli further hinder gas

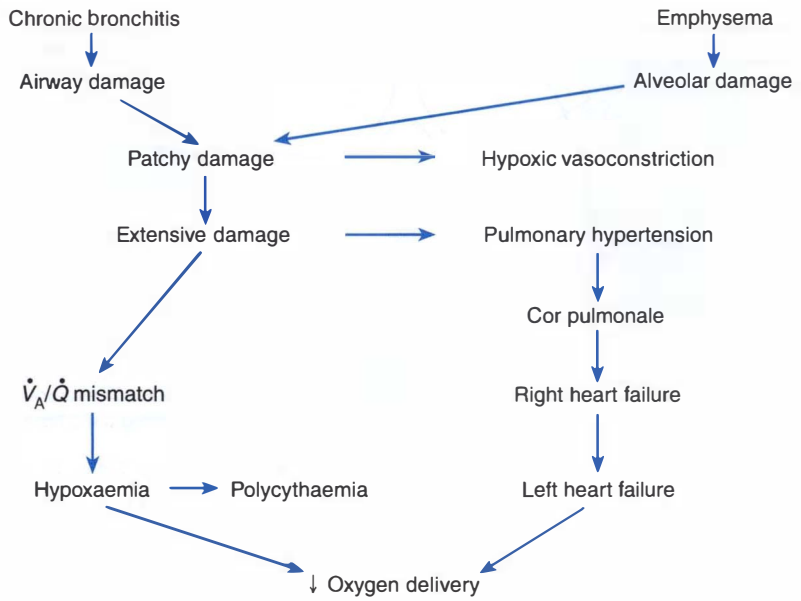


Figure 3.7 Progression of COPD. *Polycythaemia: excess red blood cells.*

exchange, and anaerobic metabolism develops (Mathur, 1999). The inexorable downhill path of advanced COPD is illustrated in Figure 3.7.

Chronic hypoxia leads to compensatory proliferation of red cells, known as polycythaemia. This increases the oxygen-carrying capacity of blood at first but, once packed cell volume reaches 55%, the thickened blood impairs oxygen delivery, burdens the heart, augments pulmonary hypertension and causes headaches. If the disadvantages of polycythaemia outweigh the advantages, haematocrit can be reduced by multiple venesections (blood-letting), exchange transfusion or haemodilution (Wedzicha, 1986). Well-managed long-term oxygen therapy can stabilize or reverse polycythaemia.

Capillary destruction and widespread hypoxic pulmonary vasoconstriction further augment pulmonary hypertension. This increases the load against which the right ventricle must pump, leading to hypertrophy and dilation of the right ventricular wall, a condition known as cor pulmonale. Once nocturnal oxygen saturation drops below 90%, right heart failure develops (Vos *et al.*, 1995). Systemic BP rises in order to overcome the increased right atrial pressure and maintain cardiac output. This process eventually strains the left ventricle and leads to left heart failure. Meanwhile lung damage is continuing, and death is ultimately due to inadequate gas exchange rather than cardiac involvement (Harris, 1989).

Clinical features

The natural history of COPD spans 20–50 years, but the disease is asymptomatic at first because changes in small airways barely affect total airways resistance. Patients may not seek medical advice until symptoms become troublesome and FEV_1 has declined to 70% of normal (Quanjer, 1993) because a morning cough is tolerable and considered normal for smokers.

Once hyperinflation develops, this becomes a major cause of symptoms (Brusasco and Fitting, 1998). The extra energy expenditure of breathing is accompanied by reduced physical activity (Hugli, 1996). Fatigue is widespread and sleep of poor quality (Girault *et al.*, 1996). Significant depression is present in over half of people with COPD (Bach and Haas, 1996,

p. 342) and 82% of people with severe COPD (Lacasse *et al.*, 1998).

Objectively, there is a rich tapestry of signs such as laboured breathing, a plethoric or cyanotic appearance, weight loss, barrel chest, forced expiration with pursed lip breathing, and prolonged expiration with I:E ratio of 1:3 or 1:4. Soft tissue recession and other signs of laboured breathing are evident (see Figure 2.2) as a result of inspiratory effort and malnutrition (Tobin, 1988). Patients may lean forward on their elbows to force the diaphragm into a more efficient dome shape and stabilize the shoulder girdle for optimum accessory muscle action. Auscultation demonstrates the crackles of chronic bronchitis (Piirilä *et al.*, 1991) or the quiet breath sounds of emphysema.

Gas exchange is preserved in the early stages, $\dot{V}_A/\dot{Q}$ match being maintained by collateral ventilation (Morrell, 1994). However, hypoxaemia gradually takes over, with nocturnal oxygen desaturation playing a particularly damaging role (Mulloy, 1996). Levels of hypoxaemia out of proportion to FEV_1 raise suspicions of sleep apnoea (RCP, 1999).

Hypercapnia is a sign of advanced disease and is related to hyperinflation (Gibson, 1996), although patients do not have to retain CO_2 to have severe airflow obstruction (Gorini, 1996). The variation in blood gas response to COPD is represented by the spectrum of the 'pink puffer' (PP) patient, who maintains near-normal blood gases at the expense of breathlessness and weight loss, and the 'blue bloater' (BB) patient (Figure 3.8).

The BB patient is less breathless, abandons the fight for normal blood gases, suffers more nocturnal hypoxaemia (Sliwinski, 1994) and pays for symptomatic relief with oedema, poor gas exchange and double the mortality of the PP patient (Clague and Calverley, 1990). Some BB patients can tolerate a P_aCO_2 of over 12 kPa (90 mmHg) for years (Hodgkin *et al.*, 1993, p. 436). This is manageable because the chief disadvantage of hypercapnia, acidosis, is normalized by kidney retention of bicarbonate.

It was originally thought that repeated hypoventilation in BB patients desensitized their chemoreceptors to hypercapnia so that they became dependent on low oxygen tension as a stimulus to breathe. However, the respiratory centres continue to be fully active, and the purpose of hypoventilation may be to preserve the respiratory muscles by 'choosing' the wise option in order to rest the muscles (Bégin, 1991), thus preventing breathlessness and fatigue (Gorini, 1996).

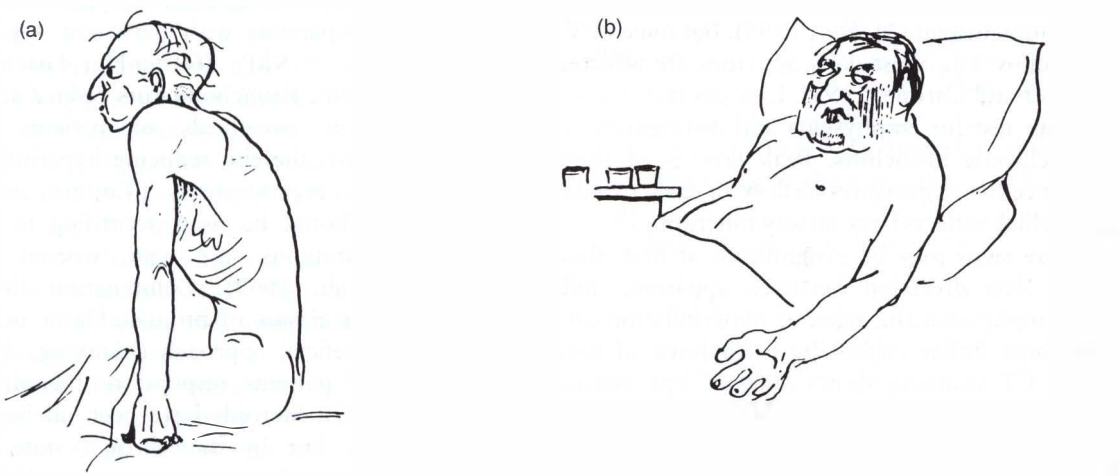


Figure 3.8 Patients with (a) pink puffer and (b) blue bloater characteristics of COPD. (From Brewis, R. A. L. (1977) *Lecture Notes in Respiratory Disease*, Blackwell, Oxford, with permission.)

The relevance to physiotherapists of the PP/BB spectrum is that PP patients in particular tend to show the following characteristics:

- anxiety and physical tension
- a counterproductive tendency to rush at activities
- more daytime hypoxaemic episodes (Sliwinski, 1994)
- tendency to desaturate on exercise (Mulloy, 1996).

Half of all COPD patients aged over 50 have cardiovascular disorders (Hodgkin *et al.*, 1993, p. 66) because of the common aetiology of smoking.

The appearance of peripheral oedema is a turning point in the progression of COPD, indicating P_aO_2 below 7.3 kPa (55 mmHg; Stewart and Howard, 1992) and 5-year survival of less than 50% (Baudouin, 1997). Although associated with heart failure, oedema in COPD is caused by impaired renal perfusion (Baudouin, 1997), especially in patients with no renal reserve (Sharkey, 1997). The gut lining is also sensitive to hypoxia, leading to the association of COPD with peptic ulceration.

Respiratory function tests are useful indicators of obstruction but relate weakly to breathlessness (Lareau, 1999), hypercapnia (Gorini, 1996), nocturnal desaturation (Miyahara, 1995) or functional impairment (Mahler, 1995). But once FEV_1 falls below 1 L, most daily activities are affected (Donner and Carone, 1998). Low gas transfer is a sensitive test for emphysema and distinguishes it from chronic bronchitis. Peak flow is of some relevance but it measures airflow in early exhalation, which reflects large airway function.

X-ray signs may be insignificant at first, then upper lobe diversion becomes apparent, and, with emphysema the signs of hyperinflation and sometimes bullae caused by breakdown of lung tissue. CT scanning shows areas of low density and blood vessel attenuation (Morgan, 1992).

Exacerbation

Survivors of exacerbations are usually left with a reduced quailty of life (Arunabh, 2000). Exacerbations occur on average one to four times a year (Postma, 1998). The airways of 40% of people with stable COPD are chronically colonized with bacteria (Zalacain *et al.*, 1999), which may double when infection is added. Exacerbation is commonly due to infection, but bacteria may be absent (Smith *et al.*, 1999). Other causes are panic attacks (Tiep, 1991), cold temperature (Donaldson, 1999), inflammation due to air pollution (Anderson *et al.*, 1997) and the effects of hypoxia on the central nervous system or cardiovascular system (Curtis, 1994).

Exacerbation causes increased airflow obstruction, hyperinflation, breathlessness and sputum production, but not necessarily fever (Tiep, 1991). Mucus clearance is hampered by cilia rendered inefficient by damaged epithelium, airway collapse and abnormal hydration (Smalldone, 1993). Hospital mortality is 10%, or 25% if hypercapnic respiratory failure develops (Baldwin, 1997), and 40% die within a year (Postma, 1998).

Medical management

The pathological process is irreversible. Smoking cessation can slow the damage and, without this, treatment is akin to running a bath without the plug. Oxygen therapy can reduce hypoxaemia and some of its effects, such as oedema (Howes *et al.*, 1995). Long-term oxygen reduces mortality for patients with persistent hypoxaemia at $P_aO_2 < 8$ kPa (60 mmHg; Leach and Treacher, 1998). Bronchodilators reduce airflow obstruction in two-thirds of patients with chronic disease, thereby reducing hyperinflation and possibly breathlessness (Tantucci *et al.*, 1998), but should be used according to need because continuous use can worsen lung function (Huib, 1999). Combination therapy with different classes of bronchodilator may be the most beneficial approach (Manning, 2000). A quarter of patients respond to theophylline (Mahon, 1999). Steroids have been advised for exacerbations but in the chronic state they reduce airways obstruction in only 10% of patients, and continued use is associated with myopathy (Davies *et al.*, 1999). However, indivi-

duals vary and should be individually assessed (Yildiz, 2000).

Drug assessment should include quality of life scores, peak flow monitoring and sequential testing of different bronchodilators, steroids, combinations and various delivery systems (p. 138). Short-term reversibility studies should not be substituted for long-term assessments. Inhalers are indicated for acute and chronic disease unless nebulizers are objectively found to be more effective (BTS, 1997). Some patients respond to drugs for breathlessness (p. 136). Many COPD patients have disturbed sleep, for which the hypnotic drug zolpidem has been found to be beneficial without affecting oxygenation, ventilation or physical performance (Girault *et al.*, 1996).

Severe exacerbations may indicate the need for non-invasive (Poponick, 1999) or invasive ventilation. Severe chronic emphysema may indicate the need for surgery, varying from laser ablation of giant bullae to lung volume reduction, discussed in Chapter 10.

Physiotherapy

For exacerbations, physiotherapy is often required to help clear secretions and reduce WOB, including non-invasive ventilation to prevent intubation. Most patients survive hospitalization but the ensuing 6 months see high levels of morbidity, mortality, readmission and relocation to long-term care (Connors, 1996). Physiotherapy must therefore include educating the patient and family about restoration and maintenance of exercise tolerance and basic self-management. A trip to the gym before discharge is motivating. If patients just sit beside their bed in their nightclothes and take the odd potter to the toilet, they may find that when they get home they are so deconditioned that they cannot get up their front steps.

In stable disease, quality of life is primarily affected by breathlessness, exercise limitation (Engström *et al.*, 1996) and the interaction of depression and fatigue (Breslin *et al.*, 1998). Mortality is directly related to ability to cope (Ashutosh, 1997). Physiotherapy is therefore best provided in the form of pulmonary rehabilitation (Chapter 9).

End stage

There is a striking difference between the management of people with end-stage COPD and that of those dying from cancer. COPD patients tend to be subjected to invasive treatments and experience poor symptom control (Connors, 1996). The physiotherapist can be instrumental in ensuring recognition of the patient's needs and a teamwork approach to palliation. Some patients would like the option of non-invasive ventilation at home if it has been carefully explained to them.

ASTHMA

Asthma is more common, more serious and more manageable than is generally thought. It is the only treatable life-threatening condition in the Western world with a rising death rate. It shows the following trends:

- Prevalence has doubled over the last two to three decades and asthma now affects 5% of adults and 10–15% of children in the UK (Flood and Partridge, 1996), killing nearly 2000 people a year (Cruickshank and Lumley, 1999), many of them young
- Over 10 years, prescriptions for asthma have increased by three-quarters and the GP consulting rate has more than doubled (Hospital Update, 1995)
- Despite this, some GPs treat less than half the asthmatics in their practice, while up to half the patients treated will only take half the dose prescribed (Pearson, 1996)
- Up to 86% of asthma deaths are considered preventable (GRASSIC, 1994)
- Asthma is more frequent in advanced age than in young adults but is frequently overlooked because of the medical perception of its association with younger people (Plaza *et al.*, 2000).

People die because they, their relatives or doctors do not see asthma as a potentially fatal

Table 3.1 Distinguishing features of asthma and COPD

	Asthma	COPD
Smoking history	Not necessarily	Yes
May start in childhood	Yes	No
Onset	Variable	Slow
Atopy	Sometimes	No
Timing of symptoms	Episodic, diurnal, seasonal	Minor variations only
Provocation of symptoms	Weak stimulus, e.g. cold air	Strong stimulus, e.g. infection
Cough at night	Patient wakes coughing	Wakes then coughs
Sputum	Contains eosinophils	Contains neutrophils
Bronchodilator response	Yes	Sometimes
Steroid response	Yes	Sometimes

disease, nor grasp the importance of prevention, nor recognize deterioration.

Asthma is a chronic inflammatory condition of the airways, characterized by undue responsiveness to stimuli that are normally innocuous, a mechanism known as hyperreactivity. Airway narrowing usually reverses spontaneously or with treatment. It is distinguished by the variability and reversibility of its presentation, which makes evaluation of severity difficult, especially as the symptoms of wheeze, breathlessness and cough are general respiratory complaints. Asthma shares with COPD the pathology of small airways obstruction, but the differences are shown in Table 3.1.

Diagnosis is made from a history of recurrent attacks, then confirmed by respiratory function tests. If the peak flow (PF) varies by more than 15%, either diurnally, after exercise or after bronchodilator treatment, the patient is considered to have asthma. This is confirmed by a 15% increase in FEV_1 after a 14-day trial with prednisolone (Fehrenbach, 1988) or by induced sputum (Pin, 1992).

Causes and pathophysiology

Predisposing factors include poverty (Smy, 1995), smoking parents, anxious parents, *in utero* allergen sensitization, history of a stressful birth, lack of breast feeding or a gene that causes atopy (Brown and Halonen, 1999). An atopic person is one who is prone to allergy and who may develop asthma if exposed to allergens, e.g. certain foods, or the faeces of house-dust mite, a tiny creature whose purpose in life is to multiply in bedding and clear up dead skin cells shed by humans. Viral infection can contribute to the pathogenesis of the disease or trigger an attack (Watson, 1997). Viruses share with passive smoking a tendency to damage epithelium so that it becomes more sensitive to allergens. Junk food may play a part because of reduced antioxidant intake (Soutar, 1997). Contributing factors include sleep, thunderstorms (Anto and Sunyer, 1997), premenstruation (O'Connor, 1997) and pollution (Cogswell, 1994).

Two phases of response occur (Figure 3.9):

1. The *sensitization stage*, which occurs in atopic people: exposure to allergens, especially in foetal or early life, stimulates production of excess immunoglobulin-E antibodies (IgE) in the serum. IgE becomes fixed to mast cells, which then react to antigens by releasing bronchoconstrictor mediators such as histamine. Serum IgE levels are five-times higher in asthma patients than in controls (Silkoff and Martin, 1998).
2. The *hyperreactive stage*: continued exposure to allergens, or response to other stimuli, leads to mast cell degranulation and release of inflammatory cytokines such as interleukins and eosinophils (Allen, 1996). Chronic low-grade inflammation damages the surface epithelial layer, causing bronchial hyperreactivity.

Once asthma is established, hyperreactive airways develop bronchospasm intermittently in

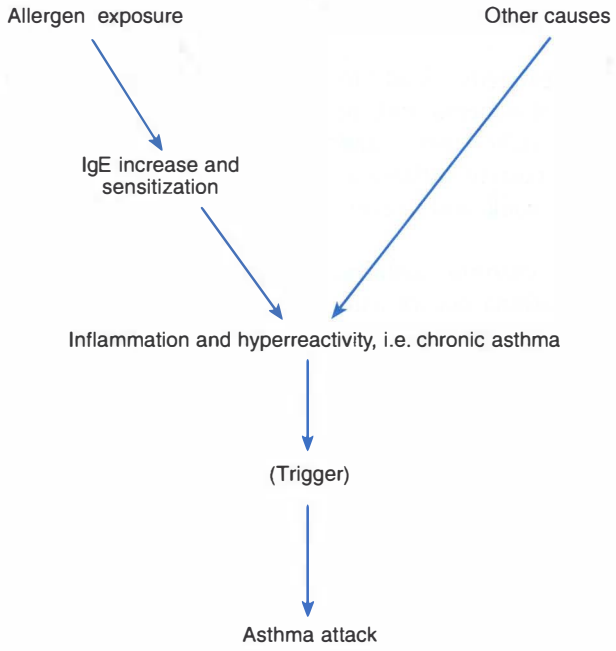


Figure 3.9 Development of asthma.

response to a variety of stimuli, which increases the work of breathing. Over time, structural changes stiffen the airway walls and parenchyma, contributing further to airway narrowing (Sterk, 1994).

The mechanism for the persistence of asthma is thought to be localized because asthmatic recipients of transplanted lungs lose their asthma while non-asthmatic patients who receive asthmatic lungs develop the disease (Corris and Dark, 1993). Once allergic asthma has developed, removal from the allergen, if delayed, does not always prevent continuing asthma.

When hyperreactivity is established, other factors, which may or may not be related to the original cause, can trigger an asthma attack, e.g.:

- allergenic foods, e.g. dairy products, eggs, wheat, nuts, fish, additives, cola or other acidic drinks
- exercise without warm-up
- weather, especially change in temperature
- drugs such as timolol (used for glaucoma), non-steroidal anti-inflammatory drugs, beta-blockers or aspirin (Empey, 1992)
- stress, through multiple central nervous system interactions (Busse, 1995)
- chest infection, especially in infants
- warm-blooded pets
- pollen
- car exhaust
- hyperventilation (Groen, 1979)
- frustrated expression of emotion (Groen, 1979)
- active or passive smoking.

Some factors may be cause, effect or both. Anxiety, depression and social isolation are associated with asthma (Ramsay, 1994). Symptoms of gastro-oesophageal reflux (GOR) are found in 82% of patients (Harding *et al.*, 1999), especially when symptoms worsen at night, after meals, on lying down or after exercise. GOR may be due to microaspiration of acid into the upper airway, or lower oesophageal relaxation caused by bronchodilators.

Treatment of GOR reduces asthma symptoms (Field, 1998).

Inflammation and hyperreactivity lead to airways obstruction by mucosal oedema and, in the acute phase, by bronchospasm and sometimes mucus plugging. Persistent inflammation leads to fibrosis of airway walls and irreversibility.

Allergic asthma, known as *extrinsic* asthma, occurs in early life. *Intrinsic* asthma occurs with normal IgE levels, develops in adulthood, is more fulminant and less responsive to treatment.

Classification and clinical features

Mild chronic asthma

This manifests as an intermittent dry cough, often at night, or a morning wheeze once or twice a week. PF varies by less than 25%. Even when asymptomatic, peripheral airflow resistance can be five times normal (Wagner, 1992), and severe attacks are possible. Deconditioning is common because of the dislike of breathlessness.

Severe chronic asthma

This means frequent exacerbations and symptoms that affect quality of life. Psychosocial factors play a more prominent role than other factors (Miller and Barbers, 1999). PF varies by more than 25%, and daily anti-inflammatory drugs are required.

Brittle asthma

The most severe form of chronic asthma is unstable or brittle asthma, which shows greatly fluctuating peak flows, persistent symptoms despite multiple medication, and unpredictable drops in lung function (Balfour-Lynn, 1999).

Acute asthma

This reflects failure of preventive management and/or exposure to a relevant stimulus. The large airways are obstructed by bronchospasm and the small airways by oedema and sometimes mucus plugging. Work of breathing is increased by airflow resistance up to 15 times normal (Mador, 1991) and hyperinflation (Wheatley, 1990). $\dot{V}_A/\dot{Q}$ mismatch reduces P_aO_2 and rapid breathing reduces P_aCO_2. Monitoring by oximetry is usually adequate but blood gases are required if S_aO_2 falls below 92% (Juniper and Davies, 1998) or PF drops below 30% predicted (Levy *et al.*, 1998). Patients feel as if they are breathing through a narrow straw, and many are extremely frightened.

> ***'The attack is like being in the sea when you can't swim.'***
>
> Castledine, 1993

Severe acute asthma

This usually develops slowly, sometimes after several weeks of wheezing. Deterioration can be deceptive and even paradoxical: subjectively, there may be denial or a blunted perception of dyspnoea (Weiner *et al.*, 2000) and objectively, the patient may appear less distressed (but more drowsy). Medical help should be sought if the patient shows the signs in Table 3.2 or the following:

- pallor or sweating
- peak flow < 50% predicted or < 200 L/min
- ↓ response to bronchodilator
- as fatigue progresses, decreased respiratory effort, retention of P_aCO_2 (Figure 3.10),

Table 3.2 Some features of acute asthma

	Severe	Life-threatening
P_aO_2	↓	↓↓
RR	> 25	↓
P_aCO_2	↓	↑
Pulse	> 110	↓
BP	↑	↓
PF	< 50% predicted	Unrecordable
Speech	Difficult	Impossible
Auscultation	Wheeze	Silent
Colour		Any change
Consciousness		Any change

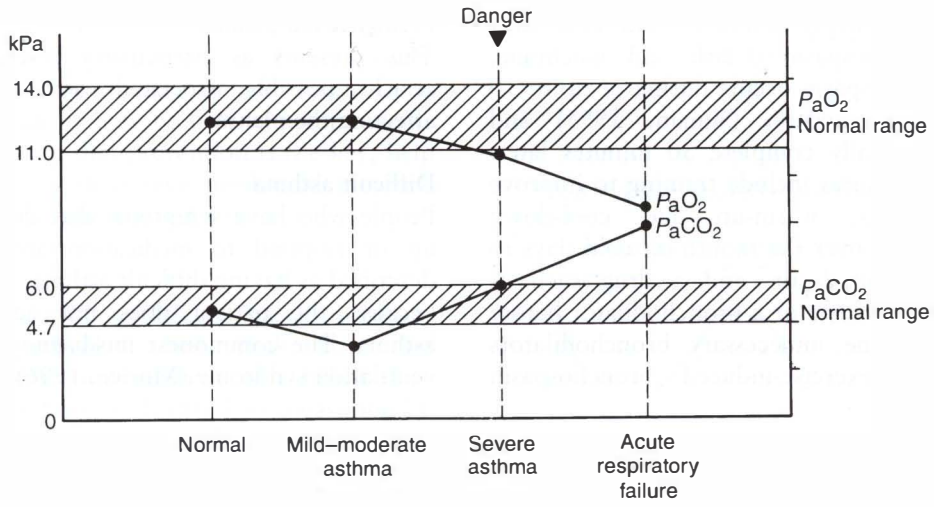


Figure 3.10 Progressive changes in arterial blood gases during acute severe asthma. (From Smith, M. (1982) In case of emergency. *Nursing Mirror*, **154**(suppl.), 11, with permission.)

which is associated with FEV_1 < 20% predicted (McFadden and Warren, 1997)
- loss of wheeze, and silent chest on auscultation if airflow is too slow to oscillate the airways
- hypotension as pulmonary capillaries are compressed by the hyperinflated chest
- cyanosis or altered consciousness, which only occur in 1% of cases but indicate grave illness (McFadden, 1995).

If P_aCO_2 rises over 6.7 kPa (50 mmHg), intensive care is required (Rossi *et al.*, 1993).

Very breathless patients cannot produce reliable PF or spirometry readings and, in those too breathless to speak, the manoeuvre can exacerbate bronchospasm (Fanta, 1992).

Some attacks may be accompanied by only mild inflammation and little mucus plugging (Gibson, 1995). Sudden deaths have been reported without exacerbation of airflow obstruction, in which case impaired respiratory drive has been implicated, related to depressed mood (Allen *et al.*, 1994).

Near-fatal attacks should be closely investigated because 10% of patients will die of their illness within a year (McFadden and Warren, 1997). The commonest predisposing factor is failure to recognize the seriousness of the final episode (McFadden and Warren, 1997).

> ***All one's strength, that one feels becoming weaker and weaker, is concentrated into one last effort to take one slight breath that will allow the respiration to continue.***
>
> Ruiz, 1993

Status asthmaticus

This term is sometimes used interchangeably with severe acute asthma, but specifically describes an asthma attack prolonged over 24 hours, leading to dehydration and exhaustion.

Asphyxic asthma

Otherwise known as 'catastrophic asthma', this acute attack leads to respiratory arrest within hours, or occasionally within minutes (Levy *et al.*, 1998).

Exercise-induced asthma

This is present in 80% of asthma sufferers and in some is the only manifestation of the disease. Hyperventilation during exercise, especially in cold weather, leads to evaporation of airway

surface liquid, hyperosmolality and heat loss, causing bronchospasm (Clark and Cochrane, 1999). Bronchospasm occurs during or up to 10 minutes after exertion (Suman, 1995) and recovery is usually complete 30 minutes later. Preventive measures include training to improve physical fitness, warm-up and cool-down periods, a scarf over the mouth on cold days to warm the inspired air, and a drug such as sodium cromoglycate or a bronchodilator before exercise. Routine unnecessary bronchodilators can worsen exercise-induced bronchospasm (Inman, 1996).

Nocturnal asthma

This occurs in 80% of people with asthma (Douglas, 1993), mostly during REM sleep. It is diagnosed from a morning dip in PF of over 20% compared to the previous evening. The term is used loosely, but accurately applies only to those who suffer at night and are symptom-free in the day. It causes fatigue and interferes with sexual activity, but there is sometimes lack of awareness of the treacherous diurnal variation.

Possible trigger factors are an exaggerated bronchial response to cold bedrooms, reduced lung volume in supine, allergens in bedding, GOR due to reduced lower oesophageal sphincter tone, or hormonal circadian oscillations in airway patency. Airways are narrowest at about 4 am (Bellia, 1993).

Once avoidable factors are removed, treatment consists of a slow-release bronchodilator and, if nocturnal attacks are recurrent, anti-inflammatory drugs, preferably not steroids in the first instance (Bellia, 1993). If the asthma is triggered by snoring, a sleep study may identify sleep apnoea (Douglas, 1993).

Occupational asthma

This may take weeks or years to develop. Symptoms usually worsen during the week and ease at weekends but several work-free days may be needed before improvement is apparent. It is usually diagnosed by a fall in FEV_1 of more than 20% over the working day or working week (Bright, 1996).

Premenstrual asthma

This presents as particularly severe monthly attacks, possibly associated with endometriosis affecting the pleura.

Difficult asthma

People who have symptoms that do not match up or respond to medication are sometimes described as having difficult asthma. This may be because the patient does not actually have asthma. The commonest misdiagnosis is hyperventilation syndrome (Morice, 1996).

Education and prevention

People with asthma benefit from pulmonary rehabilitation (Chapter 9), including education, exercise training, breathing techniques and relaxation (Emtner *et al.*, 1998). Prevention by education is central because the characteristics of asthma discourage patients from adhering to treatment. It is a chronic condition with long periods of remission, and drug regimes may show no immediate benefit. Patients tend to underestimate their symptoms (Crockett, 1997), and most do not monitor PF during an exacerbation nor call an ambulance during a life-threatening attack, probably because of anxiety (Milgrom and Bender, 1997). Following life-threatening acute asthma, a 40% incidence of denial and fear has been identified (Yellowlees, 1989).

Comprehensive preventive measures have shown:

- a 73% reduction in acute admissions (Lahdensuo, 1996)
- freedom from symptoms for most people with stable disease (Crockett, 1997)
- ↓ medication use and ↑ quality of life (Make, 1994)
- the potential to reduce asthma deaths to zero (Cochrane, 1995).

Structure

Information by booklets alone does not change behaviour (Thorax, 1997). Personal instruction is the most effective method (Make, 1994).

Education during hospitalization has the advantage that motivation is high but the disadvantage that information is not taken in if anxiety is high. It may be best to use the acute phase to explain that prevention is the key, help patients identify their own needs and motivate them to attend follow-up education.

Content

1. Self-management by drugs is based on identifying the individual best peak flow and adjusting medication when it falls below this. Optimal peak flow is identified by measuring PF within 30 minutes of waking and in the evening, and adjusting bronchodilators until best values are achieved. If this is less than 80% predicted for sex, age and height, a 2-week course of anti-inflammatory drugs, and sometimes bronchodilators, is needed to find the maximum PF. If a nebulizer is used, an initial period of saline reduces the placebo effect. Thereafter, PF readings should be taken twice daily for people with chronic asthma and four times a day for those with severe chronic asthma (D'Alonzo, 1995), using charts and advice from asthma organizations or drug manufacturers (Figure 3.11). The following action is then advised:

- PF > 80% of optimal: continue routine treatment
- PF 50–80% of optimal: start preplanned drug regime, e.g. extra bronchodilator and steroid inhalers, and/or oral steroids
- PF < 50% of optimal: start self-treatment and seek urgent medical attention as arranged in advance (Partridge, 1994).

2. Zero tolerance of symptoms.

3. Identification of precipitating factors, e.g. Figure 3.12.

4. Preventive measures based on this information, e.g. keeping pets out of bedrooms, using a DIY mask for dusty jobs, regularly washing soft toys and bedding or putting them in the freezer to kill house dust mite, removing curtains and carpets, avoiding spray polishes, bottom bunk beds, certain foods and the not-uncommon scenario of an inhaler in one hand and a cigarette in the other.

5. Identification of individual warning signs of an exacerbation, e.g. reduced exercise tolerance, waking at night, reduced effectiveness of bronchodilator.

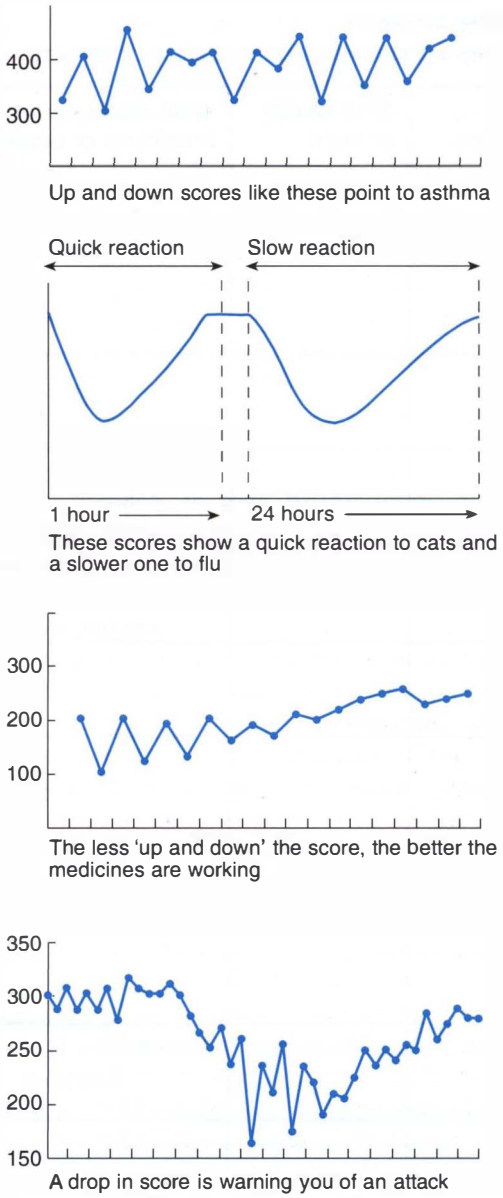


Figure 3.11 Asthma education booklet on peak flow readings. (From National Asthma Campaign booklet (see Glossary))

ASTHMA DIARY
Times when I felt extra breathless or wheezy

Date	Time of day or night	What made me breathless or wheezy?	What did I do to help myself?	How much did it help?

Figure 3.12 Example of a diary for the self-management of asthma.

6. Explanation on the action and administration of drugs, with emphasis on the importance of taking preventive drugs even when feeling well. Inhaler technique should be regularly checked because poor technique contributes to 80% of inadequate asthma control (Jones and Barrett, 1995).

7. For women with a family history of asthma, low consumption of allergenic foods during pregnancy and breast feeding, with similar care of the baby's diet in the first year of life.

8. For people who have taken long-term steroids, advice on bone mineral density checks (Laatikainen, 1999).

9. Smoking cessation if relevant and, until this is successful, augmented vitamin C intake (Butland *et al.*, 1999)

10. Reduction of nocturnal asthma by trying different drug timings, sleeping positions, room temperatures and a stress-free period before bed.

11. Identification and treatment of GOR (p. 115).

12. Avoidance of room humidifiers, which nurture house dust mite.

13. For high-risk patients, advice to keep with them at all times their inhalers, subcutaneous salbutamol and, if necessary, an auto-injector for adrenaline (Barrow, 1998), an information bracelet (BTS, 1990) and a note from their GP for ambulance personnel to administer unrestricted oxygen. Spare inhalers should be kept in the car and at work.

14. Information on organizations such as the National Asthma Campaign, which provides diary cards and educational material (Appendix C).

Drug management

Underuse, overuse and inappropriate use of drugs is common. Medication should hinge on drugs to prevent and suppress inflammation (p. 118) because prolonged inflammation can double hyperreactivity, thus increasing morbidity and mortality (Cockcroft *et al.*, 1993). Surveys on asthma deaths invariably implicate underuse of steroids prior to the fatal attack (Neville *et al.*, 1991).

Patients find bronchodilators attractive but these do not prevent inflammatory damage to the airways. They can be used regularly for acute asthma or severe chronic asthma (Niederman, 1998) but otherwise regular use is unhelpful because:

- smothering of symptoms means that a wheeze no longer acts as a warning to avoid the offending stimulus
- in the acute state, over-reliance may cause delay in seeking medical assistance
- used inappropriately, bronchodilators can actually worsen asthma (Harrison, 1999) and lengthen hospital stay (Bradding *et al.*, 1999).

The paradox is that β_2-stimulants can be beneficial immediately but detrimental in the long run.

Chronic asthma

The frequency of the need for bronchodilators provides a useful marker for adjusting prophylactic treatment. Accurate monitoring and a stepwise protocol are advocated (Box 3.1). Charts on the recognition and drug management of chronic and acute asthma are published in the *British Medical Journal* (BTS, 1993).

For patients not controlled on steroids, the

Box 3.1 Stepwise use of drugs in chronic asthma with increasing severity of disease

Step 1 Short-acting bronchodilator as required
Step 2 Add inhaled anti-inflammatory drug
Step 3 Add long-acting bronchodilator
Step 4 Increase dose of anti-inflammatory drug
Step 5 Sequential trials of different bronchodilators and oral steroids.

If 'as required' bronchodilators (step 1) are needed more than once a day, taken appropriately, patients move to step 2. Patients who are still symptomatic move through the steps until symptoms are controlled.

antileukotrienes may reduce inflammation with one oral daily dose. They work specifically on the inflammatory leukotrienes that cause long-lasting smooth muscle contraction (Weisberg, 2000). They may cause oedema if combined with prednisone (Geller, 2000).

Acute asthma

High concentrations of oxygen and high-dose nebulized bronchodilators may be required, either in small frequent doses (Bennett, 1991), continuously (Weber, 1999) or intravenously (IV) (Nelson, 1995). Oral or IV steroids are usually given, although their role is less clear in acute episodes than in chronic asthma (Allen, 1996). Inhaled heliox may prevent the need for intubation in acidotic patients (p. 131). Antibiotics are not recommended (Cruickshank and Lumley, 1999).

Breathing techniques

Certain breathing strategies can be used to aid relaxation, give patients a feeling of control and improve the efficiency of breathing. If a patient finds a technique helpful, this should be practised regularly, then used if an attack is anticipated, but not in place of appropriate medication. The emphasis is on gentle improvements in the efficiency of breathing, not deep breathing, which can exacerbate bronchospasm (Lim *et al.*, 1989).

Girodo *et al.* (1992) showed how simple relaxed abdominal breathing (p. 154) can reduce symptoms. Peper (1992) used biofeedback to facilitate abdominal breathing, resulting in reduced drug use and asthma attacks. Innocenti (1974) described how patients could gain control by learning to change back and forth between abdominal and upper chest breathing and to alter, breath by breath, the rate and depth of breathing. Weissleder (1976) claimed that asthmatic attacks could be aborted by teaching the 'complete breath technique', which consists of the following instructions, with the patient in supported sitting:

- Listen to and feel the quality of your breathing, including any wheeze
- Inhale through your nose, slowly enough to eliminate the wheeze, while increasing the depth of your breathing temporarily to compensate for the slow breath
- Adjust the rate as breathing becomes comfortable and there is less hunger for air, a hunger that will be modified by feeling in control
- Observe and modify any muscle tension
- Smoothly inhale in three different segments, abdominal, lateral costal and then upper chest expansion, then exhale in reverse order
- Re-check muscle tension
- Progress to unsupported sitting and standing positions.

The physiotherapist can start by breathing alongside the patient, but the aim is for patients to recognize their ability to manage their own condition.

The Buteyko technique is based on reducing minute volume by slowing the respiratory rate with breath-counting, using distraction by rocking and walking, and at night lying on the left side and taping the mouth closed. The rationale is that hyperventilation causes bronchospasm, which is true but simplistic in that there are many other causes of bronchospasm. Bowler *et al.* (1998) attempted to provide evidence but their study was flawed by:

- unequal groups in that the Buteyko group initially required 1½ times the steroids of the control group
- the fact that the Buteyko group received seven times as many follow-up phone calls as the control group, plus extra breathing classes
- no significant difference being found in end-tidal CO_2 or quality of life scores at the end of the trial
- most patients proving either not to have asthma or to have unstable asthma.

However, there is strong anecdotal evidence of excellent results in some patients, indicating that the overlap between hyperventilation syndrome and asthma is often missed. The physiotherapist's

role is to give any patients diagnosed with asthma the simple Nijmegen questionnaire (p. 298) to identify coexisting hyperventilation syndrome, then treat appropriately.

Exercise

Asthma and exercise have a strange relationship. Exercise can trigger an acute episode, but aerobic training with appropriate precautions has shown the following outcomes (Emtner *et al.*, 1996):

- ↓ exercise-induced asthma
- ↑ conditioning
- ↑ confidence and independence
- ↓ asthma attacks.

Adherence rates tend to be good (Emtner, 1998), even at high intensity training, and Emtner *et al.* (1996) successfully motivated patients to exercise at 80–90% maximum heart rate. Outdoor exercise is best taken in the least polluted areas and times of day. Face masks protect against some pollutants but the filter must be changed regularly, and they may increase the work of breathing (Atkinson, 1997).

Other physical measures for chronic asthma

Relaxation is well-established as a therapy for asthma, and is highly valued by patients (Emtner *et al.*, 1998). Freedberg *et al.* (1987) showed objective benefit by increased PF readings. Techniques that achieve profound relaxation have shown positive outcomes, especially meditation, yoga (Fried, 1993, p. 234) and hypnotherapy (Morrison, 1988). Acupuncture can increase PF and decrease medication, and homeopathy is sometimes useful (Lewith, 1996). Naturopathic assessment techniques may indicate a need for supplementary vitamin C and antioxidants, or use food exclusion diets to identify certain additives and allergens (Lewith, 1996). The majority of patients have tried complementary therapy, and breathing techniques have been found the most popular (Ernst, 1998). This indicates the scope for physiotherapy. Complementary therapy may reduce the incidence of acute asthma but there is no evidence that it is helpful during an attack.

Muscle tension and a habitually hyperinflated chest are occasionally evident, for which postural advice and emphasis on exhalation are indicated. Exhalation is not to be encouraged during acute episodes when hyperinflation is necessary to hold open obstructed airways.

It has been claimed that inspiratory muscles can be damaged during an attack and that the risk is reduced by strengthening these muscles. Weiner *et al.* (1992) found that 6 months of inspiratory muscle training, using a pressure-threshold device for 30 minutes five days a week, reduced symptoms, medication use and hospitalization.

Coughing can relieve or exacerbate asthma (Young *et al.*, 1991) and the physiotherapist can advise on effective coughing or cough suppression (p. 204) or both.

Outcome measures have traditionally comprised respiratory function tests, hospitalization and death rates, but functional measures most usefully relate to physiotherapy outcomes. Quality of life questionnaires are described by Rowe (1993) and Juniper (1999a).

Physical assistance for acute asthma

> ***'Most conversations in A&E are directed over and about me, and rarely involve me in any meaningful way, which is a pity as I believe that I know quite a bit about myself as an asthmatic.'***
>
> Carter, 1995

Some patients do not want to be touched during an attack. Some do not want to be talked to. Most do not want to be left alone. All want to be consulted. Noise, light and crowding should be minimal.

Some of the following strategies may be helpful for some patients.

- Sit upright, or lean slightly forward resting the arms on a table, or sit astride a chair backwards with the arms resting on the chair's back.

- Keep warm.
- Sit near fresh but not cold air.
- Take sips of warm water (some patients prefer cold), although this should not be attempted in the throes of a bad attack.
- Breathe through the nose unless breathlessness makes this impossible.
- If there is dizziness with tingling hands and feet, breathing is faster than it needs to be. This can be reassuring and indirectly help slow the breathing.
- Practise previously learned techniques of relaxation, abdominal breathing and control over breathing. These should be started at the first intimation of an acute episode.
- The Innocenti technique to raise resting lung volume (p. 173) may help to open the narrowed airways. Extra elastic work imposed by hyperinflation is offset by less airflow resistance so that total work is reduced (Wheatley, 1990). Some patients will have already achieved optimal inflation spontaneously.

The patient can be asked if s/he would like relatives to be involved, which may reduce family anxiety so that they are less likely to transmit their own fear. Patients know best what helps them but relatives can be shown how to apply acupressure by pressing or massaging the bronchospasm or breathless points (Ellis, 1994):

- CV17: anterior midline between nipples, level of 4th intercostal space
- Lu1: just below each coracoid process
- Bl13: 1½ thumb-widths lateral to the lower border of each T3 spinous process.

General stress points are sometimes helpful, e.g. Co4 on each dorsal thumb web (to be avoided in pregnant women as it may bring on premature labour), or Li3 on each dorsal space between first and second metatarsals.

Some patients find it helpful to cuddle a warm hot water bottle or vibrating pillow. Some benefit from rhythmic slow chest percussion to help promote relaxation. Other techniques to reduce the work of breathing are described in Chapter 7.

Mucus may be present with a slow-onset attack (Picardo, 1996), in which case slow percussion can both promote relaxation and help clear secretions. If secretions are too thick to shift, warm humidification with normal saline may be indicated (Phillips and Millard, 1994) but this can increase airflow resistance in children or those with severe airways obstruction (Wissing, 1988) and close observation for desaturation is required.

Patients who continue to tire may need rehydration and mechanical assistance. Low to medium levels of CPAP will relieve inspiratory muscles from their exhausting work of holding open the obstructed airways (Shivaram *et al.*, 1987). If CPAP is not available, IPPB (p. 159) or non-invasive ventilation (Mak *et al.*, 1995) can be used to ease the work of breathing. If positive pressure aids are needed, the X-ray should be checked beforehand in case of pneumothorax.

Mechanical ventilation for asthma is discussed in Chapter 15, and children's asthma in Chapter 16.

BRONCHIECTASIS

Bronchiectasis is characterized by chronic irreversible distortion and dilation of the bronchi. It has been called the orphan disease because its incidence is unknown, its diagnosis is often missed and its prognosis is poorer than that of asthma (Keistinen, 1997).

Bronchiectasis is not a final diagnosis so much as a common pathway of several conditions predisposing to persistent lung infection. It is associated with severe respiratory infection, foreign body inhalation, cystic fibrosis, purulent rhinosinusitis, tuberculosis, smoke inhalation, inflammatory bowel disease, primary ciliary dyskinesia and a reactive form of rheumatoid arthritis in which joint pain responds when antibiotics are given for the bronchiectasis (Steinfort *et al.*, 1987). Causes of bronchiectasis may be multiple or unknown. The disease is diminishing in countries where living standards are rising and

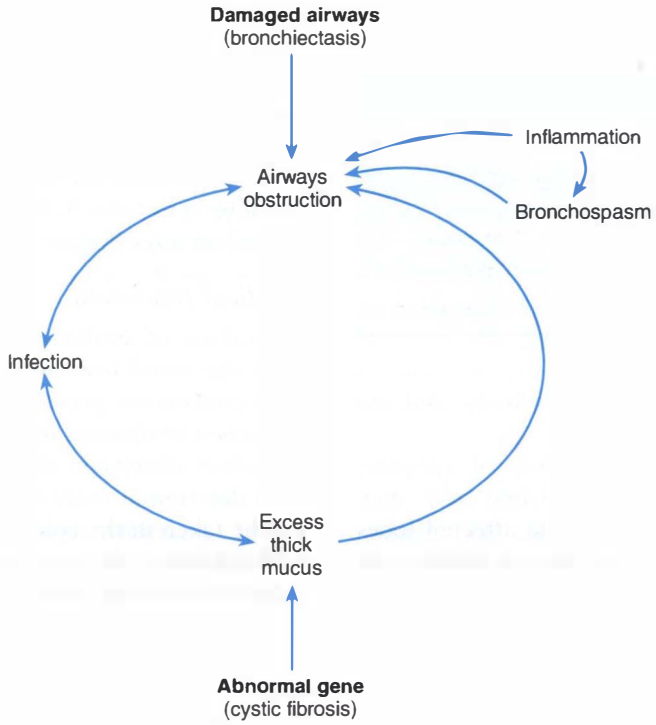


Figure 3.13 Vicious cycle that augments the processes of cystic fibrosis and bronchiectasis. CF is progressive, whereas the course of bronchiectasis varies according to cause and management.

where children are vaccinated against diseases such as whooping cough and measles.

Pathophysiology

Chronic inflammation damages the elastic and muscular components of subsegmental airways and sometimes the parenchyma. The warm moist environment of the lung combines with excess mucus to set up a vicious cycle of infection, persistent inflammation and further obstruction (Figure 3.13).

Thick mucus sits heavily on the tender cilia and causes further damage. An over-exuberant immune response to the colonizing microbes releases toxic inflammatory chemicals, which impair lung defences. Continuous inflammation leads to fibrosis and sometimes sets off bronchospasm, which augments the cycle. Abscesses may occur.

Anatomical disorganization is greatest if it starts in early childhood before the lungs are fully developed. Progressive destruction occurs in anything between 3% and 48% of patients (Munro, 1992), depending partly on medical and physiotherapy intervention to protect the airways. Advanced disease brings pulmonary hypertension and cor pulmonale.

Clinical features

Voluminous quantities of sputum are produced despite the inefficient clearance mechanisms caused by corrugated airways and damaged cilia. Mucosal ulceration can cause haemoptysis, indicating that the airways are particularly vulnerable to infection, and some physicians recommend prophylactic antibiotics at this time. Secretions and collapsing airways on expiration cause coarse wheezes and crackles (Piirilä *et al.*, 1991). A variant called 'dry bronchiectasis' appears to be a contradiction in terms but occa-

Table 3.3 Distinguishing features of bronchiectasis and chronic obstructive pulmonary disease

	Bronchiectasis	COPD
Age	Varied	Older
Smoking history	Not necessarily	Usually
Auscultation	Noisy, may be localized	Diffuse crackles
Sputum	Excessive, often thick and green	Moderate
Haemoptysis	Sometimes	No
Finger clubbing	Sometimes	No
X-ray	Specific	Variable

sionally patients have few secretions and no chronic infection.

Other features are fatigue, loss of appetite, finger clubbing and dyspnoea. Chest ache may occur, usually associated with the affected lobes (Munro *et al.*, 1998). Bronchiectasis tends to be misdiagnosed as COPD. Table 3.3 clarifies the distinction.

X-rays show focal or diffuse signs. In severe disease, parallel tramlines represent thickened airway walls and 1 cm 'bunch of grapes' ring shadows represent dilated airways seen end-on, usually clustered in groups. Neglected disease shows 'glove finger shadows', which are dilated bronchi full of thick secretions, and the ring shadows may have fluid levels. A normal X-ray does not exclude the diagnosis, and CT scanning is more sensitive. A bronchogram outlines the dilated airways (Figure 3.14).

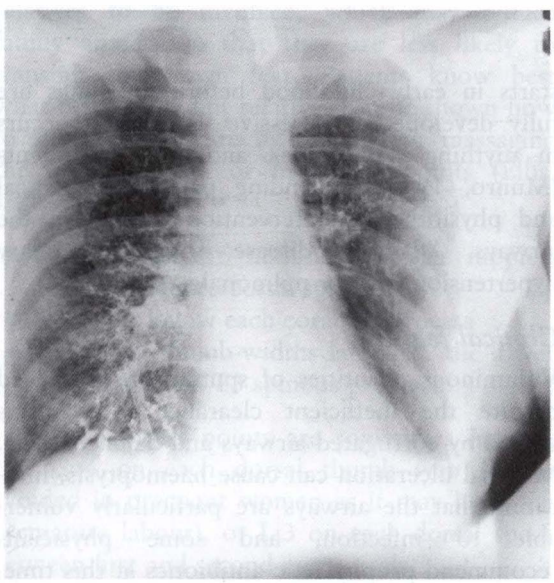


Figure 3.14 Bronchogram illustrating the dilated airways of bronchiectasis in the right lower lobe. The straight left heart border (sail sign) indicates previous left lower lobectomy

Medical treatment

Liberal use of antibiotics helps control infection, with the trend towards infection-specific rather than continuous prescription. Patients are given a store of antibiotics to be taken at the first sign of colour change in their sputum. For patients who deteriorate every winter, regular antibiotics can be taken in the cold months.

Antibiotics do not control the persistent inflammation that may be progressively destroying the airways (Shum *et al.*, 1993) but inhaled steroids can assist this and reduce the volume of sputum (Elborn *et al.*, 1992). Other drugs that may decrease sputum volume are dry powder mannitol (Daviskas, 1999) and erythromycin (Tsang *et al.*, 1999). Bronchodilators are used if there is demonstrable hyperreactivity.

Surgical resection of non-perfused lung may be indicated for localized and disabling disease (Ashour, 1996). Occasionally, transplantation is possible in late-stage disease. Sometimes the cause of the disorder might be treatable, e.g. topical steroids for rhinosinusitis to prevent mucus sliding from the back of the nose into the lung.

Physiotherapy

Bronchiectasis reduces mucociliary clearance to an average 15% of normal (Houtmeyers *et al.*, 1999) and patients need education in sputum clearance to compensate for this. A daily programme is required that is sufficient to eliminate coughing in between clearance sessions. Hydration, an exercise programme and ACB/AD (p. 94) are often adequate but other measures (Chapter 8) may be required, sometimes including postural drainage. Patients should be discouraged from coughing until they

are ready to expectorate in order to minimize fatigue and cough-related stress incontinence. Much encouragement is needed to help patients set up a life-long programme that is effective and suited to their lifestyle. Thereafter, occasional reviews are needed.

Non-invasive ventilation is not well-established in progressive end-stage disease, but it can benefit those with diffuse disease (Benhamou *et al.*, 1997).

CYSTIC FIBROSIS

Cystic fibrosis (CF) is a chronic progressive obstructive disorder affecting the exocrine glands. It is the commonest lethal inherited disease among white people (Ramsey, 1996), acquired as an autosomal recessive disorder. The gene is carried by 1 in 25 Caucasians and comes to life when inherited from both parents. Two carriers have a 1-in-4 chance of having an affected baby and a 1-in-2 chance that their baby will be a carrier.

The diagnosis is suspected if infants show failure to thrive, meconium ileus or repeated chest infections. Confirmation is by a test for abnormally salty sweat at age 6 weeks.

Recent developments include identification of the rogue gene, prenatal diagnosis and organ transplantation. But improved survival (Figure 3.15) is mainly due to attention to detail in conventional treatments, i.e. antibiotics, physiotherapy and nutrition. However, the disease is still eventually fatal and treatment is aimed primarily at improving quality of life.

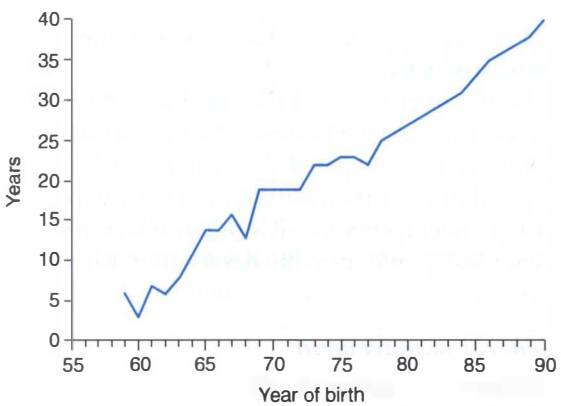


Figure 3.15 Projected median survival of patients with CF by year of birth. (From Elborn, J. S, Shale, D. J. and Britton, J. R. (1991) Cystic fibrosis: current survival and population estimates to the year 2000. *Thorax*, **46**, 881–885.)

Pathophysiology

In most cells the gene encoding CF is dormant, but in epithelial cells it is switched on. This impairs ion and water transport across epithelial surfaces of the body, causing dehydration of secretions and obstruction of various body lumens. In the gut, this causes malabsorption and pancreatic insufficiency. In the lungs, sodium and chloride ions cannot escape from the epithelial cells into the airways in order to maintain hydration of mucus, which becomes thick and sticky. Viscid mucus encourages bacterial adherence (Figure 3.13), augmented by inflammatory mediators such as neutrophils (Costello, 1996). Dying neutrophils release DNA, whose strands bind together and thicken secretions further.

The respiratory component determines the quality of life and is the usual cause of death. The lungs are structurally normal at birth, but inflammatory changes are evident as early as 4 weeks old (Jaffé *et al.*, 1999) and intractable infection soon becomes established, even when the patient is clinically well, leading to progressive damage by a smouldering course of bacterial colonization punctuated by exacerbations. Viruses and fungi play a role, and long-term antibiotics predispose the lungs to *Aspergillus* colonization (Bargon *et al.*, 1999).

The range of bacteria is curiously restricted. *Staphylococcus aureus* causes significant harm and the acquisition of *Burkholderia cepacia*, the organism responsible for onion rot, poses a particular threat; some strains are untreatable and reduce lifespan by 10 years, 20% of patients developing fatal fulminant pneumonia (Ledson, 1998). Preventive measures against *Burkholderia cepacia* include segregation of patients who do and do not have the organism, at great personal

cost to those who have previously socialized freely. Even sibling separation is tolerated by some families. Respiratory equipment and treatment locations are segregated, and physiotherapists must wash their hands in an antiseptic such as Hibiscrub.

Malnutrition may contribute to impaired respiratory defence. Pneumothorax occurs in up to 10% of children and 20% of adults, as a result of rupture of a subpleural bleb or bulla (Noppen *et al.*, 1994). Resting energy expenditure is 20% higher than normal in adults, half of it caused by the inefficiency of breathing with hyperinflated lungs (Elborn, 1996).

Survival to adulthood is now the norm (Elborn, 1996), which has given rise to new difficulties. The liver and gall bladder can cause problems, pancreatic fibrosis can lead to diabetes and dehydration, vasculitis can affect joints, skin and brain, bronchial artery hypertrophy may lead to pulmonary haemorrhage, and excessive coughing predisposes to stress incontinence (White *et al.*, 1999). A sixfold increase in the incidence of gut cancer (Webb and Govan, 1998) may be related to survival of an older population and/or gastro-oesophageal reflux. Most patients die of respiratory and cardiac failure.

Clinical features

> ***'Coughing and spluttering like an old man does not endear one to the general public, and neither does the popular misconception that one is scattering infections round like confetti. ... My fingers are like spoons and I can't wear nail polish. ... It doesn't do too much for one's confidence to know that one has probably got halitosis – so I tend to talk to people sideways on....'***
>
> Hall, 1984

The antisocial nature of the disease is caused by features such as incessant coughing, small stature, delayed puberty, flatus, increasing breathlessness and unrelenting weariness. If chest pain occurs, it may be due to pleural inflammation, muscle strain from excessive coughing or pneumothorax. Most males cannot conceive naturally, because the sperm tail is structurally similar to cilia, but fatherhood is possible (McCallum, 2000). Women can have children, in which case optimum nutrition and respiratory care are required prior to pregnancy.

By the age of 3 months, 50% of babies have respiratory symptoms in some form (Dinwiddie, 2000). Objectively, auscultation gradually shows wheezes as a bronchiolitis-like process develops in the small airways, then widespread crackles develop. Other signs are similar to bronchiectasis. If there is hepatomegaly, diaphragmatic function is impaired. The radiograph is normal at first, then shows patchy opacities in the apical regions, then signs of widespread bronchiectasis, emphysema and finally cor pulmonale.

Growth may be stunted because of energy imbalance, energy supply being reduced by malabsorption and anorexia, and energy demand increased by up to 25% (Shepherd, 1988) because of excess WOB. However, good nutrition means that the patient should not appear malnourished except in the terminal phase or if the liver is involved. The fact that patients usually look well leads to misunderstandings about fatigue and other invisible problems (Eigen *et al.*, 1987).

Exacerbation is indicated by weight loss or worsening respiratory symptoms. If the cause is respiratory, secretions are thicker than normal and the patient may become less rather than more productive.

In the later stages, FEV_1 declines, P_aO_2 falls and eventually P_aCO_2 rises. The inexorable deterioration is anticipated by patients, who each respond in their individual way. They often form strong attachments to each other, which provide comradeship but can be devastating when one dies.

General management

Children and adults should always be under the care of a cystic fibrosis centre (Mahadeva *et al.*, 1998).

Prevention

Screening is possible at three stages. Carrier

screening helps when making decisions about reproduction; if two carriers want to have a child they can be offered *in vitro* fertilization, genetic screening and implantation of a healthy embryo. Prenatal diagnosis provides information on which to base a decision about continuing a pregnancy, but this only occurs after the birth of the first unexpected cystic child or if there is a family history of CF. Neonatal screening leads to early diagnosis but is not yet routine.

Research into gene therapy is proceeding at a dizzy pace. The accessibility of the airway means that patients could inhale a normal copy of the gene on an adenovirus, so long as the body does not build immunity to the virus. Treatment would be required monthly because of cell turnover, and the damage that had already occurred would not be reversible.

Education

When a baby with CF is born, education for the parents begins as soon as they have accommodated sufficiently to the diagnosis. General points to note are:

- No-one should smoke in the home.
- CF children are of normal intelligence and should go to normal schools.
- Within their limitations they should take part in normal physical activities.
- They are not infectious.
- They should share with healthy siblings the disciplines and standards of the family.

Lifelong treatment is time-consuming and the nature of the disease places stresses on the family that can double the divorce rate (Jennings, 1992). Self-help groups and professional support limit family isolation and allow attention for siblings.

Medication

At present the backbone of medical management is an unremitting onslaught against bacteria. High doses of antibiotics compensate for difficulty in reaching the lungs through obstructed airways and poor perfusion. Access is by peripheral line or an implantable venous device (Yung, 1996), either electively every 3 months, or symptomatically (Elbom *et al.*, 2000). This can be managed at home, which reduces nosocomial infection, is cheaper and is usually preferred by the family, but disadvantages include lack of respite for the family and increased fatigue for the patient (Wolter, 1997). Close supervision is essential for it to be effective (Bosworth and Nielson, 1997). Nebulized antibiotics are suitable for some patients but are time consuming, polluting and variable in effect.

The drug rhDNase (recombinant human deoxyribonuclease) contains a clone of the gene responsible for breaking down DNA, and when given as an aerosol can decrease sputum viscosity, improve FEV_1, reduce exacerbations by 30% (Conway, 1997) and lessen intractable atelectasis (Shah *et al.*, 1994). It benefits 50% of patients and nearly doubles the cost of their care (Conway, 1997), with some of the cost being offset by reduced infectious episodes (Böllert *et al.*, 1999). If the drug causes deterioration, it does so by overliquefying secretions so that mucociliary clearance becomes as difficult as eating soup with a fork.

A specialist centre is needed to evaluate rhDNase. It is tested at different times of the day to take account of when the chest is clearer, and with accurate monitoring by spirometry and subjective scores. A positive response may not be demonstrated for weeks or even months (Conway and Littlewood, 1997). The drug should be taken after bronchodilators and physiotherapy to maximize distribution and at least 30 minutes before nebulized antibiotics to prevent them denaturing the rhDNase protein. A mains-powered specialized jet nebulizer is required and should not be used for other drugs.

Aerosolized $alpha_1$-antitrypsin helps replenish the patient's defences (Briars and Warner, 1993). Aerosolized amiloride, a sodium channel blocker, helps restore normal hydration to secretions but requires up to four treatments a day and has been challenged as no better than inhaled saline (Middleton *et al.*, 1993). Anti-inflammatory therapy is either by steroids or non-steroidal anti-inflammatory drugs (Konstan

et al., 1995). Bronchodilators show objective benefit in a minority of patients but can paradoxically increase airways obstruction in as many (Dinwiddie, 2000). Mucolytic drugs have been considered ineffective in CF (Fiel, 1993), but some patients love them.

Nutrition

Malnutrition is associated with poor survival (Elborn, 1996), and is closely tied to respiratory status (Davis *et al.*, 1996). Up to 200% of normal calorie intake is required, as well as enzyme supplements to make up for pancreatic insufficiency. These supplements can cause constipation, especially if a full meal is not taken after the enzymes. Supplementary feeds in advanced disease slow the decline in lung function (Elborn, 1996).

Surgery

A pneumothorax is managed by chest tube drainage if minor, but recurrent pneumothoraces require thoracoscopic pleurodesis (Noppen *et al.*, 1994).

Evaluation for transplant of heart, lung and/or liver depends on the rate of decline in FEV_1 (Milla, 1998). Transplantation can transform a chair-ridden invalid into an active individual within weeks of the operation. Most of the pulmonary problems of CF can be eliminated because donor lungs do not have the genetic abnormality. But the obstacles are formidable, including the stress of waiting for donor organs, lifelong immunosuppressive drugs for the successful, and devastated families if the wait is too long or the transplanted organs are rejected (Whitehead and Leval, 1994).

Selection criteria include short life-expectancy, oxygen dependence, cor pulmonale and high motivation. Exclusion criteria may include multiresistant organisms and questionable psychosocial support. Children have their own criteria (Gaynor, 1998). The shortage of donors has raised moral issues. Gentle palliative management may now be supplanted by vigorous gastrostomy feeding, mechanical ventilation and other heroics to keep an increasingly desperate patient alive. Living-related transplantation has given rise to further ethical dilemmas but, if acceptable, a lower lobe may be donated by each parent, or by two siblings (Dark, 1997).

Patients must be free to make their own choices, especially in the later stages. Options include gentle autogenic drainage (p. 196), nocturnal oxygen therapy (Coates, 1992), non-invasive ventilation (Regnis, 1994) and/or palliation.

Physiotherapy

Once CF has been diagnosed, physiotherapy is started immediately, even if no secretions are produced, with the intention of minimizing the cycle of excess secretions and airway damage. Physiotherapy is the most gruelling and least tolerated aspect of treatment and shows adherence rates below 50% (Abbott *et al.*, 1994). Daily treatment regimes produce no immediate improvement in well-being, and sputum quantity is the only reinforcement to encourage this repetitive task. Parents of CF children generally adhere to treatment, but Fong (1994) found that less than half of CF adults believe physiotherapy to be effective. Some physiotherapists consider that it is not necessary for all patients (Samuels *et al.*, 1995) but most consider it central to CF management. Treatment to clear secretions is best individualized according to patient preference.

If convenient for the patient and family, physiotherapy should be co-ordinated with nebulizer treatments, i.e. before antibiotics so that absorption of the drug is not hampered by mucus-filled airways, and after bronchodilators or nebulized saline. Hypertonic saline clears more secretions than isotonic saline (Riedler, 1996) and has been considered superior to rhDNase in one study (King *et al.*, 1997). Details of sputum clearance techniques are given in Chapter 8, with aspects specific to CF outlined below.

The active cycle of breathing, autogenic drainage and devices such as the PEP mask and flutter are popular because they encourage independence. Interesting data has emerged suggesting that abdominal breathing with biofeedback

can reduce airways obstruction (Delk *et al.*, 1993).

Exercise has the advantage that most patients will actually do it (Abbott *et al.*, 1994). Outcomes of exercise training include reduced breathlessness, improved lung function (Dodd, 1991) and increased exercise tolerance, mucociliary clearance and well-being (Bye *et al.*, 1997). An exercise programme has enabled some motivated patients to recover from partial disablement to a near-normal life (Heijerman, 1992). Exercise usually complements other techniques but in less severe cases can be the primary treatment (Andréasson *et al.*, 1987). Swimming is especially beneficial, but patients should choose their favourite activity.

For training, patients exercise to a pulse rate of 50–75% of their maximum exercise capacity, aiming at a minimum 30 minutes four times a week. A little-and-often approach may be more suitable for some patients, because fatigue is common. Successful exercise training requires regular reviews with a physiotherapist and commitment from the patient and family. Assessment can take the form of a 2-minute walk test, which is more suited to children than the somewhat tedious 6-minute distance, a 3-minute step test with metronome and stopwatch (Balfour-Lynn *et al.*, 1998) or modified shuttle test (Bradley *et al.*, 1999). Patients with advanced disease are less likely to benefit from exercise, partly because of fatigue and partly because little extra tidal volume can be superimposed on hyperinflated lungs.

If postural drainage is the chosen treatment, drainage time is about 15 minutes in younger children, more in older people or if there are excessive secretions. Length of time depends on fatigue, patient preference, quantity of secretions and effectiveness of other measures. Percussion and vibrations are often included if they produce more sputum or if the patient finds them effective. Some authorities consider them unnecessary (Sutton *et al.*, 1985), and they can be a burden for the family, but the combination of all three techniques has shown positive outcomes (Reisman, 1988).

Inspiratory muscle endurance is normally already elevated because of the extra work required to breathe through obstructed airways, but it has been claimed that inspiratory muscle training can improve exercise tolerance (Sawyer and Clanton, 1993).

The optimal frequency for physiotherapy is not known (Eigen *et al.*, 1987) but is usually performed twice daily, with variations depending on secretions and remission or exacerbation. Treatment is best continued until sputum is no longer expectorated or a rest is needed.

Parents should be given advice and support until children are able to manage themselves. Early independence should be encouraged, with young children actively participating and 10-year-olds able to do their own treatment when staying with friends overnight. Older children are advised against cough suppression, by which they attempt to conceal the illness from their peers.

The 'optimum' recommended treatment programme is not always the most effective in the teenage years. Management is best negotiated, with the physiotherapist 'complying' with the patient's wish rather than the other way round. People with CF are particularly worth listening to because they are medically streetwise and understand much about their treatment. When patients are hospitalized, motivation is enhanced by simple measures such as offering a choice of treatment times and techniques.

Patients require 3-monthly physiotherapy reviews (RCP, 1996). Outcomes are based on quality of life measures such as exercise tolerance, questionnaires and well-being scales (Orenstein and Kaplan, 1991).

Precautions

History and symptoms should be checked for gastro-oesophageal reflux, which has been found in 81% of young children with CF, 25% of older children (Malfroot and Dab, 1991) and frequently in adults (Tattersall *et al.*, 1997). These patients should avoid head-down postural drainage when possible (p. 115).

Patients should not be asked to cough unnecessarily because excessive coughing causes collapse of central airways with impairment of sputum clearance (Zapletal *et al.*, 1983), and can cause fatigue, haemoptysis and stress incontinence.

When using CPAP or non-invasive ventilation (Chapter 7), high pressures are not necessary for respite from fatigue and should be avoided because of the risk of pneumothorax.

Liver cirrhosis occurs in 10% of patients, which may lead to oesophageal varices and haematemesis, in which case all physiotherapy except abdominal breathing is contraindicated until bleeding is controlled.

Blood streaking of sputum is common in CF and should be disregarded, but frank haemoptysis should be reported and physiotherapy temporarily halted.

Measures to prevent cross infection include scrupulous hand-washing, the covering of sputum pots and single-patient use of PEP and flutter devices.

Osteoporosis is universal in adults with late-stage disease (Aris, 1998), manifest as increased kyphosis and fracture risk, but it begins during skeletal growth and deficits in total bone mineral average 20% in children and young adults (Henderson, 1999). Causes include abnormal bone growth during childhood, steroid use and immunosuppressive drugs after transplantation. Prevention is by optimum nutrition in childhood, minimizing long-term steroids and teaching impact exercise.

It is not known if transient oxygen desaturation during exercise is harmful, but those with an FEV_1 below 50% predicted are likely to show desaturation. When exercising, they should avoid desaturating by more than 5% or an absolute level below 80% (Dodd, 1991), using interval training, reduced workload or added oxygen. In advanced disease, added oxygen allows longer periods of exercise and may postpone the development of pulmonary hypertension (Marcus, 1992). Fluids and free access to the salt shaker are needed in hot weather because of the high salt concentration in sweat and a tendency to underestimate fluid needs. Haemoptysis contraindicates exercise training.

PRIMARY CILIARY DYSKINESIA

Primary ciliary dyskinesia (PCD) is an inherited condition whose prevalence is thought to be underestimated and diagnosis made either late (Bush *et al.*, 1998), or mistakenly as bronchiectasis or CF. It is characterized by an uncoordinated ciliary beat, leading to excess secretions and an effect similar to an escalator malfunctioning in rush hour. The outcome is recurrent infection of ears, sinuses and lungs.

PCD is suspected in children with a perpetually runny nose, glue ear and frequent chest infections. Males are subfertile. Half of patients have mirror-image organ arrangement so that the X-ray shows dextrocardia and the middle lobe is on the left (Bush *et al.*, 1998).

PCD is not a progressive disorder but the twin pillars of selective antibiotics and regular physiotherapy are needed to delay the onset of bronchiectasis. Reflexology or homeopathy may help boost the immune system (Pollack, 1999). Some patients benefit from rhDNase drugs (Berge, 1999) but β_2-agonists can cause deterioration, so both should be monitored objectively. Ear grommets are contraindicated because of ensuing ear discharge (Hadfield, 1997).

ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS

Aspergillosis is an inflammatory disease manifesting mainly in the lungs as allergic bronchopulmonary aspergillosis. It is a reaction to the *Aspergillus* fungus, which is responsible for more than half of all fungal infections (Calvo *et al.*, 1999). It rarely invades immunologically competent people but occurs in 10% of people with CF (Sharma, 1998) and sometimes in cavitating lung diseases such as TB.

Patients present with malaise, weight loss, breathlessness, fever, haemoptysis and a cough productive of brown rubbery mucus casts,

sometimes in the shape of the bronchial tree. X-ray signs are cavitating lesions containing white fungus balls. The disorder may continue for years, with episodes of pulmonary infiltration and wheezing, sometimes leading to fibrosis and cor pulmonale.

Diagnosis is by bronchoscopy or CT scan. Treatment is by inhaled steroids (Slavin, 1996) to help prevent the development of bronchiectasis, which tends to affect the upper lobes. Antifungal agents can be delivered bronchoscopically or percutaneously. Surgical resection may be required (Jackson *et al.*, 1993).

INHALED FOREIGN BODY

Children are prone to inhale objects which they put in their mouths, and 70% of patients who aspirate a foreign body are under the age of 3 (Denholm and Goodwin, 1995). There is usually a history of choking and paroxysmal coughing, followed by a relatively asymptomatic interval (Tariq, 1999). Clinical signs may then arise, such as localized wheeze, stridor, persistent cough refractory to treatment, occasional haemoptysis and, if there is complete obstruction, gradual atelectasis over 18–24 hours as a result of absorption of trapped air (Figure 3.16).

Foreign bodies tend to lodge preferentially in the right bronchial tree in adults and centrally in children (Baharloo, 1999). Small objects can be retained for months or even years, causing no symptoms or a chronic cough. Many foreign bodies are made of vegetable matter and do not show on X-ray. Most are capricious and, in young children, difficult to diagnose.

Physiotherapy is contraindicated because of the risk of shifting the object to a more dangerous location. The foreign body is best removed by bronchoscopy, following which there can be inflammatory secretions or localized collapse that may require physiotherapy.

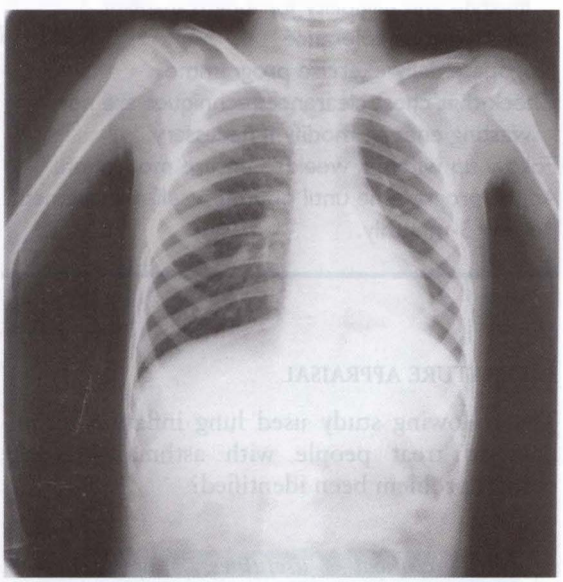


Figure 3.16 Following aspiration of a foreign body by this young child, the left lower lobe has collapsed, shifting the mediastinum to the left.

MINI CASE STUDY: MR MB

This 25-year-old man has primary emphysema due to alpha$_1$-antitrypsin deficiency.

Background

HPC: recurrent childhood infections.
SH: Unemployed, lives alone, 'finished with girlfriend because I'm too busy with hospital appointments'. Non-smoker.

Subjective

Yellow sputum, cleared independently.
SOB worse since admission last April.
Watch TV much of the time.
Hoping for lung transplant.

Objective

Hyperinflation.
Breathing pattern normal.
↑ RR on slight exertion.
Stooped posture.
Scattered crackles on auscultation.

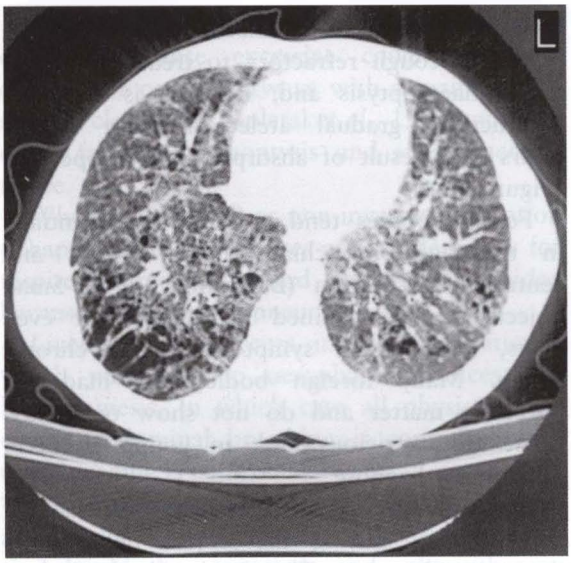


Figure 3.17a CT scan of Mr MB.

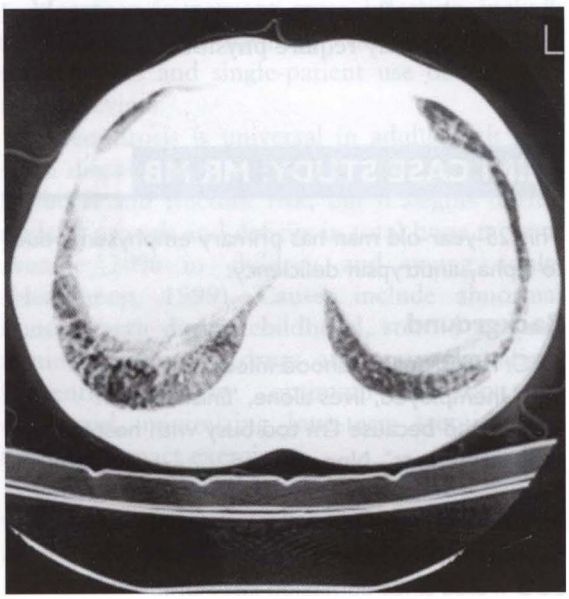


Figure 3.17b CT scan of Mr MB at a lower level

Questions

1. CT scan: evidence of emphysema (Figure 3.17)?
2. Analysis?
3. Problems and goals?
4. Plan?

RESPONSE TO MINI CASE STUDY

1. Scan

(a) Black airspaces in lung fields indicate emphysema.
(b) 'double border' of diaphragm, indicating breathlessness.

2. Analysis

Little ventilatory reserve.
Previous fitness not regained since hospital admission.
Inactive lifestyle.
Poor posture contributing to inefficiency of breathing.

3. Problems and goals

SOB.
Exercise tolerance.

4. Plan

Educate on breathlessness management.
Educate on posture correction.
Educate on relevance of exercise tolerance to lifestyle and eligibility for lung transplant.
Assess exercise tolerance.
Negotiate daily exercise programme.
Check that chest clearance techniques are not wasting energy; modify if necessary.
Follow up within a week to ensure motivation.
Adjust programme until optimum self-management.
Review 3-monthly.

LITERATURE APPRAISAL

The following study used lung inflation techniques to treat people with asthma. Has the correct problem been identified?

> *The clinical usefulness of chest physiotherapy techniques in bronchial asthma is still being discussed. Lung inflation techniques, such as incentive*

spirometry, voluntary deep breathing, intermittent positive pressure breathing and continuous positive airways pressure, are used to increase lung volumes during acute attacks and to reduce atelectasis, but published studies have failed to document their usefulness in the treatment of asthma.

Eur. Respir. J. 1993; 3: 353–355

RESPONSE TO LITERATURE APPRAISAL

Is the patient's problem:

- loss of lung volume? – no, acute asthma causes hyperinflation and 'lung inflation techniques' would be counterproductive
- excess work of breathing? – yes, but the methods described would increase the work of breathing
- sputum retention? – maybe, but these methods are not designed for sputum clearance.

This is the type of article that is interpreted as meaning that 'physiotherapy' is unhelpful for people with asthma.

RECOMMENDED READING

Barnes, P. J. (1995) Chronic obstructive pulmonary disease. *Update*, **51**, 91–97.

British Thoracic Society (1993) Guidelines for management of asthma: a summary. *Br. Med. J.*, **306**, 776–782.

Davis, P. B., Drumm, M. and Konstan, M. W. (1996) Cystic fibrosis. *Am. J. Respir. Crit. Care Med.*, 154, 1229–1256.

Demeter, S. L. (1986) Hyperventilation syndrome and asthma. *Am. J. Med.*, **81**,89–94.

Fehrenbach, C. (1998) Chronic obstructive pulmonary disease. *Prof. Nurse*, **13**, 771–777.

Gibson, G. J. (1996) Pulmonary hyperinflation. *Eur. Respir. J.*, **9**, 2640–2649.

Henderson, A. (1994) Chronic respiratory failure. *Practitioner*, **238**, 345–350.

Luce, P. J. (1996) Asthma in the elderly. *Br. J. Hosp. Med.*, 55, 118–124.

Parker, A. E. and Young, C. S. (1991) The physiotherapy management of cystic fibrosis in children. *Physiotherapy*, 77, 584–586.

Partridge, M. R. (1996) Self-management plans (asthma). *Br. J. Ther. Rehab.*, **3**, 271–275.

4 Restrictive and other disorders

Summary

Introduction

Restrictive disorders are characterized by reduced lung volume, poor compliance and increased work of breathing (WOB). Restriction is caused by:

- shrunken lung tissue, e.g. interstitial disease
- lung compressed from within the chest wall, e.g. pleural effusion or pneumothorax
- lung compressed by the chest wall, e.g. skeletal disorders
- reduced ability to expand the lung, e.g. neuromuscular disorders.

Other respiratory disorders covered in this chapter do not necessarily restrict the lung nor obstruct the airways but the respiratory system can be affected by infections, cancers and systemic disease.

Interstitial lung disease

Diseases that affect the supporting structures of the lung rather than the airspaces are covered by the umbrella term 'interstitial lung disease'. Over 200 disorders have been identified, usually related to immune disturbance or exposure to toxic agents. Inflammatory changes lead to alveolitis, which may resolve or progress to patchy fibrosis, thickened alveolar septa, remodelling of parenchyma and shrunken, stiff lungs. Smoking augments the damage. Stiff lungs mean fewer functioning alveoli and often excess elastic recoil. Involvement of alveoli means involvement of capillaries, and the term 'collagen vascular disease' can overlap with interstitial lung disease.

The two main effects are:

- ↑ lung stiffness, which increases the work of breathing
- ↓ surface area of the alveolar–capillary membrane, which impairs gas exchange.

Examples are described below.

Fibrosing alveolitis is the commonest interstitial lung disease. Causes may be obscure, e.g. a side effect of the drug amiodarone (Mathewson, 1997), or obvious, e.g. occupational pollutants such as metal and wood dust. If there is no obvious cause it is known as cryptogenic or idiopathic fibrosing alveolitis. Progression is variable, with average survival less than 3 years from diagnosis (Hubbard *et al.*, 1998). The term fibrosing alveolitis may incorporate the end result of other disorders, such as those described below, which may or may not be classified separately.

Bird fancier's and *farmer's lung* cause

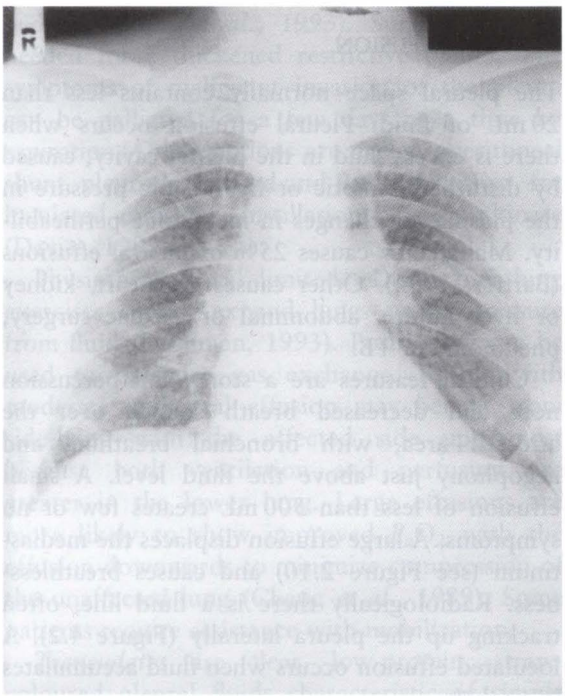


Figure 4.1 Ground-glass appearance of pneumoconiosis in a miner after working in the pit from age 13 to age 24.

extrinsic allergic alveolitis, leading to fever and malaise 4–8 hours after exposure to the relevant organic dust. Lung fibrosis may develop if patients are reluctant to separate from their birds or farming, but some protection can be provided by masks and antigen avoidance (Bourke and Boyd, 1997).

The *pneumoconioses* are slowly developing inhalation diseases. The body reacts to each inhaled particle by creating an inflammatory wall of cells around it. *Miners' lung* (Figure 4.1) and *silicosis* are examples related to occupational exposure. *Asbestosis* is an example characterized by a 'shaggy heart' appearance on X-ray and a delay of up to 20 years between inhaling asbestos dust and developing the disease. Asbestosis is less common than the pleural manifestations of asbestos exposure (p. 109).

Systemic lupus erythematosus (SLE) is characterized by exacerbations and remissions, including joint pain. Lung involvement is by pleurisy, pleural effusion and fibrosis (Leach, 1998). Patients are at risk of pneumonia because treatment is with immunosuppressive agents.

Scleroderma is a connective tissue disorder confined to the skin at first but often progressing to internal organs, including the lung.

Rheumatoid disease is a systemic disease best known for its inflamed joints, but in 10–15% of patients also manifesting as 'rheumatoid lung' (Jefferies and Turley, 1999, p. 201), which incorporates pleural, vascular, airway and fibrotic components (Hayakawa *et al.*, 1996).

Sarcoidosis is a multisystem granulomatous disorder of unknown cause with widespread variation in severity, commonly presenting at ages 20–40. A third of patients are symptom-free, being identified by routine X-ray showing bilateral lymphadenopathy and sometimes infiltrates. Skin, eyes and joints may be affected. Lung involvement is common; it stabilizes or clears in 80% of patients, but the remainder suffer irreversible fibrosis, and most deaths from sarcoidosis are due to lung damage (Judson 1998).

Clinical features and diagnosis

The lungs have a large reserve capacity and the following only emerge after considerable injury:

- shallow breathing to ease the elastic load, and rapid breathing to sustain ventilation
- dry cough (Lalloo, 1998)
- on auscultation, fine end-inspiratory crackles caused by popping open of peripheral airways, unchanged by deep breathing, coughing or position change
- progressive X-ray signs of 'ground glass' (Figure 4.1), reticular patterning and honeycombing, as alveoli are pulled apart to form cystic spaces (see Figure 2.14), then an overdomed diaphragm as the lung shrinks
- ↓ P_aO_2 because of $\dot{V}_a/\dot{Q}$ mismatch and ↓ P_aCO_2 because of rapid breathing
- hypoxaemia on exercise, not predicted by resting S_aO_2
- dyspnoea that becomes progressively incapacitating

- fatigue
- digital clubbing in over half of patients (Johnston *et al.*, 1997).

Respiratory function tests show impaired gas transfer and reduced lung volumes. Diagnosis is by CT scan (Johnston *et al.*, 1997), but suspicions are raised by an exercise test that shows an abnormal response of rapid shallow breathing, increased minute ventilation and high $P_{A-a}O_2$ (de Lucas, 1996).

Treatment

Only 15% of patients respond to steroids (MacNee, 1995), because fibrosis is often established and irreversible, but in combination with interferon, substantial improvement is possible (Britton, 2000). Collagen-inhibitors also show promise (Nagler, 1996). Symptoms are sometimes alleviated by immunosuppressive drugs and breathlessness temporarily relieved by nebulized local anaesthetic. Oxygen is needed in the later stages, especially on exercise. Lung transplantation offers hope for some patients.

Physiotherapy to change the breathing pattern is often unhelpful because the rapid shallow breathing adopted by patients reduces the effect of excessive lung recoil and is probably the most efficient for them. Patients who find relief by deep breathing might also have hyperventilation syndrome caused by the rapid breathing associated with interstitial disease.

Patients may respond to some measures to reduce WOB (Chapter 7), including judicious use of non-invasive ventilation for those who find it brings relief. Advice and encouragement help to maintain functional activities within the limits of dyspnoea, desaturation and fatigue. Help with positioning is appreciated in the late stages, following the patient's need, usually avoiding the forward-lean positions which might restrict the lung further.

In the unlikely event of a patient being mechanically ventilated, manual hyperinflation should be used minimally because the non-compliant lungs are at risk of pneumothorax.

PLEURAL EFFUSION

The pleural space normally contains less than 20 mL of fluid. Pleural effusion occurs when there is excess fluid in the pleural cavity, caused by disturbed osmotic or hydrostatic pressure in the plasma, or changes in membrane permeability. Malignancy causes 25% of pleural effusions (Bartter, 1994). Other causes are heart, kidney or liver failure, abdominal or cardiac surgery, pneumonia or TB.

Clinical features are a stony dull percussion note and decreased breath sounds over the affected area, with bronchial breathing and aegophony just above the fluid level. A small effusion of less than 500 mL creates few or no symptoms. A large effusion displaces the mediastinum (see Figure 2.10) and causes breathlessness. Radiologically there is a fluid line, often tracking up the pleura laterally (Figure 4.2). A loculated effusion occurs when fluid accumulates in pockets.

Medical treatment is directed at the cause, plus symptomatic relief of breathlessness by needle aspiration (thoracocentesis), performed slowly to avoid 're-expansion pulmonary

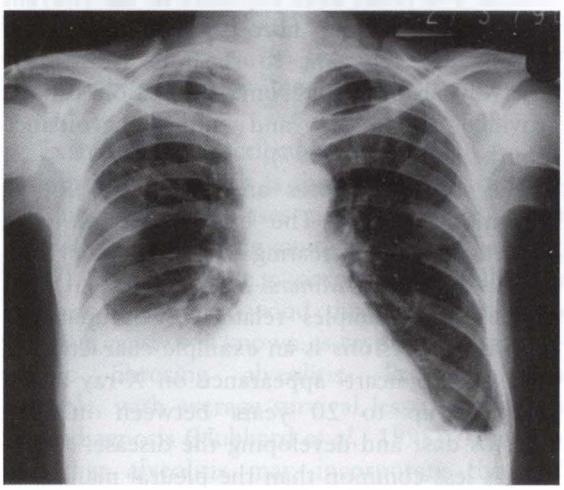


Figure 4.2 Bilateral pleural effusions. The right side shows a dense opacity with a smooth horizontal border and meniscal edge. The left shows a small effusion obliterating the costophrenic angle.

oedema' (Kam *et al.*, 1993). Surgery may be needed for a thickened restrictive pleura. The symptoms of malignant invasion of the pleura can be palliated for a few days at a time by aspiration. Other options are a pleuroperitoneal shunt, pleurodesis (Reid and Rudd, 1993) or, for loculated effusions, instillation of streptokinase (Davies, 1999).

Physiotherapy is limited. Deep breathing exercises cannot expand lungs under pressure from fluid (Dechman, 1993). Positioning can be used to optimize gas exchange. People with moderate unilateral effusion may benefit from side-lying with the affected side uppermost because both ventilation and perfusion are greater in the lower lung. Large effusions are more likely to show improved P_aO_2 with the effusion downwards to minimize compression of the unaffected lung (Chang *et al.*, 1989). Some patients require assistance with mobilization.

Transudates are clear, low-protein, straw-coloured pleural fluids characteristic of simple effusions caused by liver or kidney problems, heart failure, malnutrition or fluid imbalance. *Exudates* are cloudy, high-protein effusions containing cells that pass through a damaged pleura, and are associated with malignancy, trauma and infection. *Haemothorax* is blood in the pleura as a result of malignancy or trauma, and is managed by treating the cause, plus tube drainage if necessary.

Empyema is pus in the pleural cavity following localized infection. It can complicate pneumonia, bronchiectasis, chronic aspiration, abscess or chest surgery, especially oesophageal surgery. The patient may be asymptomatic or toxic, depending on the organism and volume of pus. Treatment is by local and systemic antibiotics. Other options are needle aspiration, tube drainage either into a bag or with strong suction using an underwater seal system, lavage, debridement via thoracoscopy, open drainage with rib resection, or thoracotomy with decortication. Surgery is required if pus fills more than 40% of the hemithorax (Ferguson *et al.*, 1996). Surgical patients are debilitated and need attention to mobility.

PNEUMOTHORAX

> ***'When the pneumothorax happened I was totally conscious of the puncturing of the thorax, the unbearable pain, the cold sweat, being afraid that the final moment would come quicker than the help that you could give me.'***
>
> Ruiz, 1993

If either pleural layer is ruptured, air rushes into the pleural space, causing a pneumothorax. The lung shrinks towards the hilum, not always symmetrically, in proportion to the amount of pleural air. Air continues to escape into the pleura until pressure is equalized or the collapsing lung seals the hole.

Clinical features are diminished breath sounds, breathlessness due to a reflex arc with afferents carried in the vagi, and pain in 75% of patients (Light, 1993). Radiographic signs are shown in Figures 2.13 and 4.3.

For a large pneumothorax, the collapsed lung is seen shrivelled around the hilum, sometimes with the mediastinum shifted away from the affected side, especially if the pneumothorax is under tension (see below). A small pneu-

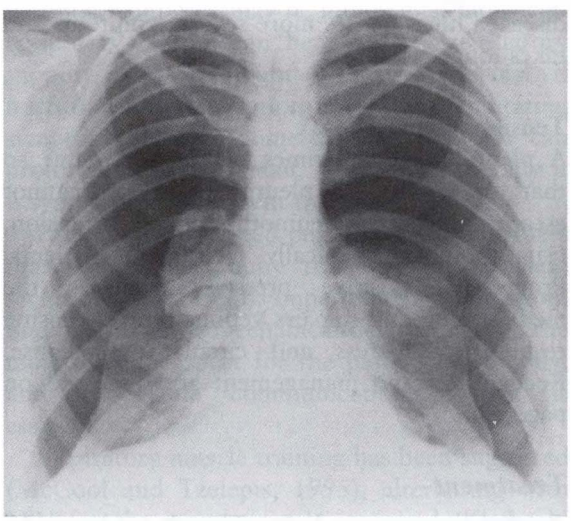


Figure 4.3 Large bilateral pneumothoraces in a patient with emphysema.

mothorax can be identified more easily when the film is taken on expiration and the lung is smaller. CT signs are shown in Figure 15.11.

Types of pneumothorax

Spontaneous pneumothorax
The beehive shape of the lungs means that the apex of the upright lung is subject to greater mechanical stress than the base because the weight of the lung pulls down on it. A spontaneous pneumothorax usually occurs in this region, especially in tall, thin young men who are thought to grow faster than their pleura is able to keep up with. Although 'spontaneous', many patients are smokers and have blebs on X-ray (Light, 1993). The recurrence rate is 23–50% (Tschopp, 2000).

Secondary pneumothorax
A pneumothorax may occur secondary to puncture from a fractured rib, inaccurate insertion of a cannula, high-volume positive pressure ventilation, rupture of an emphysematous bulla or drug abuse leading to prolonged Valsalva breath-holds or attempted central venous injection. A pneumothorax secondary to diseased lungs causes more severe symptoms and takes longer to heal.

Tension pneumothorax
A pleural tear sometimes works as a valve so that air enters the pleural space but cannot escape, causing a pneumothorax under tension, usually in mechanically ventilated patients. Progressive positive pressure displaces the mediastinum and impairs venous return, causing respiratory distress and circulatory collapse. Recognition and management are discussed on page 383.

Treatment

To drain or not to drain? This depends on the size of the pneumothorax and medical opinion. If small and asymptomatic, it can be left to heal itself. A moderate first pneumothorax can be managed by needle aspiration without admission to hospital. For hospitalized patients, high concentrations of inspired oxygen speed resolution by increasing the absorption of pleural air fourfold, the inert nitrogen being displaced by absorbable oxygen (Light, 1993). A larger pneumothorax can be treated with a Heimlich valve, which enables air to escape but not to re-enter.

A chest drain (p. 269) is used if simpler methods are not adequate or the patient is on a ventilator. Once the air leak has ceased, i.e. when there is no more bubbling in the drainage bottle, the drain is clamped for some hours and then removed if the X-ray shows no recurrence. Sclerosing agents may be instilled through the chest drain to encourage adherence of the pleura to the chest wall. Surgical intervention (p. 268) is necessary if these measures fail or if the condition is recurrent or bilateral.

Physiotherapy is based on education in chest drain management and positioning if necessary. Lying on the good side is often the most comfortable and is usually best for $\dot{V}_A/\dot{Q}$ matching, but lying on the affected side may speed absorption of air (Zidulka *et al.*, 1982). Patients may require assistance with mobilization, vigorously if the cause is traumatic, e.g. stab wound (Senekal, 1994), but gently if recurrence is likely, e.g. immediately after surgery, in case the pleura becomes unstuck. Shortwave diathermy to increase the absorption of air has been suggested (Ma *et al.*, 1997).

Precautions include avoidance of positive pressure techniques (CPAP, IPPB and other non-invasive ventilation strategies, or manual hyperinflation) if there is no functioning chest drain. Patients should be advised to avoid paroxysms of coughing.

NEUROMUSCULAR DISORDERS

Weak inspiratory muscles restrict expansion. Sputum clearance may also be impaired because weak expiratory muscles impair cough and

reduced mechanical movement of the lung slows mucociliary clearance (Mier *et al.*, 1990).

Pathophysiology and clinical features

If a patient has generalized muscle weakness, the respiratory muscles are usually included. Respiratory muscle weakness may go undetected if limb weakness reduces mobility, and ventilatory failure may arrive unexpectedly. Fatigue is common and sleep apnoea may further impair quality of life. Assessment of the respiratory muscles is described on page 61. Some aspects of specific disorders are discussed below.

Respiratory complications are the major cause of morbidity and mortality in *multiple sclerosis*, as a result of aspiration and pneumonia secondary to bulbar weakness and immobility. Coughing is a particular problem because expiratory muscles lack the usual stimulus of strenuous activity and are disproportionately weak compared to the inspiratory muscles. There is evidence of some ability to strengthen the expiratory muscles but whether this ameliorates the symptoms is unknown (Gosselink *et al.*, 1999).

A weak cough is also the main respiratory problem for people with *motor neurone disease*, 50% of whom die from complications such as aspiration and pneumonia within 3 years of diagnosis (Sykes and Young, 1999, p. 95). If there is bilateral diaphragmatic paralysis out of proportion to weakness of other muscles, non-invasive ventilation (NIV) is indicated. If there is bulbar weakness, swallowing assistance (p. 102) or PEG feeding (p. 265) is required. If there is intermittent adduction of the vocal cords (signalled by episodic stridor in the day and snoring at night), some patients are willing to accept tracheostomy (Shneerson, 1996a). The main fears of patients in late-stage disease are breathlessness and choking. Breathlessness may be controlled by some of the methods in Chapter 7 and can be relieved with an injection of combined diamorphine/chlorpromazine/hyoscine, or rectal diazepam. Excess salivation can be reduced by an anticholinergic such as atropine or hyoscine. Death by choking is rare.

Post-polio respiratory insufficiency may occur decades after the acute illness. Chronic overuse of weak muscles, and ageing, lead to fatigue, chronic pain and respiratory distress. Patients may need advice on energy conservation, balanced functional activities and NIV.

Physiotherapy

Patients may need advice on balancing rest and exercise, including avoiding overuse of compensatory muscles. Some require treatment for excess work of breathing or sputum retention.

Upright positioning to facilitate breathing is advised for patients with muscle weakness. Manual support can assist coughing (p. 202). Regular position change and incentive spirometry help to prevent atelectasis. If abdominal muscles show hypertonicity, full inspiration may be hampered, which further reduces lung volume. Postures that encourage inhibitory control over spasticity can modify this.

Monitoring is required once vital capacity falls below 50% predicted, because ventilatory failure is inevitable when VC is less than 30% and P_aCO_2 starts to rise (Anzueto, 1999). The patient needs to make a decision on NIV before an acute episode precipitates action. If deterioration is progressive, the patient needs to understand that weaning from NIV may be impossible. However, temporary relapse can be eased by ventilatory support, and overnight NIV is beneficial if nocturnal hypoventilation is causing debilitating symptoms. There is some evidence that NIV can prolong life (Aboussouan, 1997) but generally it is symptom management that is the aim.

Patients with assisted peak cough flows of less than 160 L/min require tracheostomy to clear secretions (Bio, 1998). Impaired communication, due to disease or mechanical devices, can be the most difficult aspect for the patient and family, and a reliable communications system is essential.

Inspiratory muscle training has been suggested (McCool and Tzelepis, 1995), alternating with NIV for the more severely impaired (Klefbeck, 1999). Oxygen therapy is not advisable for people in ventilatory failure and can exacerbate

nocturnal hypoventilation (Bach and Haas, 1996, p. 423), especially in post-polio patients whose respiratory centres may have been damaged by the primary viral infection (Bach and Haas, 1996, p. 371).

Management of chronic aspiration

Chronic aspiration is common in neuromuscular disease because of its association with dysphagia and poor gag reflex. A certain amount can be tolerated if clearance mechanisms are normal but, if symptoms are present, teamwork with the speech–language therapist, nursing staff and carers is required.

Dysphagia is suspected if there is excess salivation, deterioration after meals or lack of elevation of the larynx on swallowing. A tracheostomy with inflated cuff exacerbates dysphagia but may reduce the risk of aspiration. Swallowing problems often develop insidiously but dysphagia usually parallels or shortly follows the development of speech problems. Patients are fearful of choking and suffocation (Jacobsson, 2000).

Aspiration is suspected in patients with recurrent right lower lobe pneumonia, spiking temperatures, excess oral secretions, reluctance to eat or drink, weight loss, dehydration, gastro-oesophageal reflux or feeding that is associated with coughing or crackles on auscultation over the right main bronchus. Silent aspiration occurs without coughing and is present in 40% of people with dysphagia (Gauwitz, 1995) but is also common during sleep in elderly people (Kikuchi *et al.*, 1994). A quarter of patients with stroke aspirate (Odderson, 1995) and half of long-term tracheostomized patients aspirate during feeding (Nava 1998a). Aspiration can cause bronchospasm, bronchitis, atelectasis, pneumonia and abscess (Bach and Haas, 1996, p. 392).

Radiological signs are regional or disseminated nodular shadows (see Figure 2.26), which may progress to interstitial scarring and honeycomb lung. Videofluoroscopy confirms delayed swallowing and reduced peristalsis. This procedure is carried out by a radiologist and speech-language therapist, but the physiotherapist may be required to stand by with suction equipment. Cervical auscultation is used by speech–language therapists to assess swallowing (Cichero and Murdoch, 1998).

Prevention is by:

- head and chest elevation
- periodic turning from side to side
- avoidance of neck extension
- when eating, upright sitting with chin tucked in, neck slightly flexed and the patient able to see the food
- avoidance of eating when tired
- little-and-often feeding
- use of finger foods to avoid utensils
- avoidance of straws for drinking
- keeping the drinking glass full to prevent the patient tilting his/her head back
- if there is too little saliva, extra stimulation with the tongue
- if there is too much saliva, advice to swallow, and/or reduce dairy intake (p. 132)
- avoidance of distractions while eating
- maintenance of the upright position for 30–60 minutes after eating
- keeping suction equipment to hand.

Further advice on management is given by Odderson (1995) and patients' organizations (Appendix C).

Gauwitz (1995) advises chopped food rather then purees, placing the food in the unaffected side of the mouth, leisurely meals and avoidance of food that is too hot or cold, bland, dry, sticky or mucus-forming. Other tips are small mouthfuls, restriction of clear liquids, encouragement of regular gentle coughs and swallowing several times for each bolus. The speech–language therapist provides further advice on dietary texture and administration. If assisted feeding is not adequate, a nasogastric tube itself can cause feelings of choking, and PEG feeding is preferable (Norton, 1996).

Swallowing involves virtually all levels of the central nervous system and about 50 paired muscles of the mouth, pharynx and oesophagus

(Bach and Haas, 1996, p. 392). Logemann (1986) claims that the risk of aspiration can be reduced by muscle strengthening. Pharyngeal function may be helped by isometric neck exercises to encourage laryngeal elevation.

Dysphagia may lead to dehydration, which limits secretion clearance, and weight loss, which limits mobility. Nutrition is often neglected and the physiotherapist may need to initiate multidisciplinary management.

The management of acute aspiration is discussed on page 105. The management of neurological patients in intensive care is discussed on page 393.

SKELETAL DISORDERS

Kyphoscoliosis

A distorted spine increases the work of breathing because of reduced chest wall compliance, microatelectasis and altered alveolar surface tension (Elliott, 1995). The configuration of the chest wall forces the diaphragm to work inefficiently. Surgery is sometimes undertaken to prevent progression of scoliosis or improve body image, but is unlikely to improve pulmonary function (Wong *et al.*, 1996).

Ankylosing spondylitis

This is a systemic disease that affects breathing because of the rigid thoracic cage and kyphotic spine. Chest wall compliance is impaired but lung compliance and diaphragmatic movement are preserved. Occasionally, fibrosis and bullous disease affect the lung. The chest X-ray shows apparent hyperinflation because the chest wall becomes fixed in an inspiratory position. Attention to posture, thoracic mobility and exercise training are advocated (Leite, 1995).

Severe cases of neuromuscular and skeletal disorders may lead to type II respiratory failure (p. 117), with hypercapnia and sometimes cor pulmonale.

PNEUMONIA

Pneumonia is the commonest cause of death from infection in the West because of its predilection for the elderly and immunosuppressed. Infective or chemical agents breach lung defences, inflame lung parenchyma and smallest bronchioles, then fill and consolidate alveoli with fibrous exudate. Risk factors are acute stroke, poor nutrition, smoking, alcoholism, winter and infancy. The changing pattern is shown in Figure 4.4.

Clinical features include fever, breathlessness, tachycardia, myalgia and often dehydration. If localized, the affected area demonstrates a dull percussion note, bronchial breath sounds or fine crackles, sometimes decreased expansion and a pleural rub. The X-ray often lags behind clinical presentation (Ferdinand, 1998) but tends to

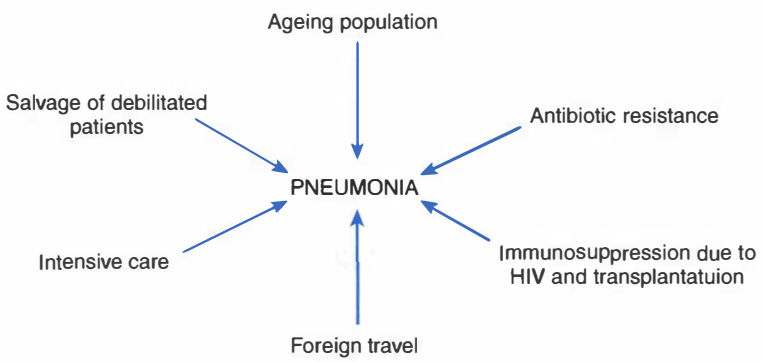


Figure 4.4 How the causes of pneumonia are changing.

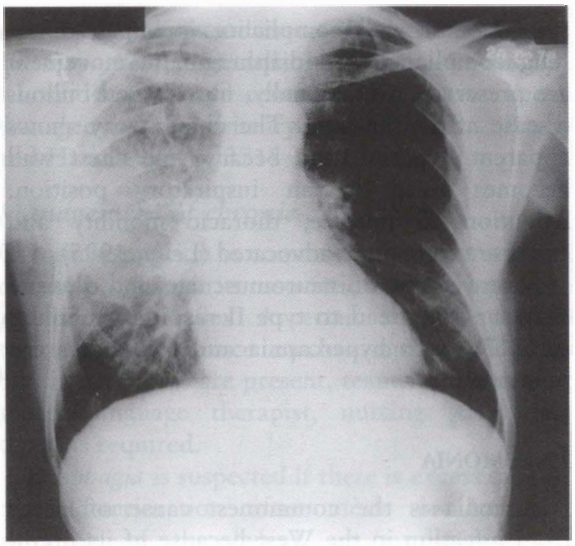


Figure 4.5 Consolidation of right upper lobe.

show a patchy opacity with ill-defined margins (Figure 4.5).

There may be a dry cough at first, which can become productive of purulent and sometimes rusty blood-stained sputum as the consolidation resolves. The structure of the lung is preserved and complete resolution is possible, although sensitized nerve endings may leave a dry irritating cough for some time.

Treatment is by oral or intravenous fluids, antiviral or antibacterial drugs, and oxygen if indicated. In the acute stage, physiotherapy is limited to positioning for $\dot{V}_A/\dot{Q}$ matching and CPAP if hypoxaemia persists despite 40% oxygen (Brett and Sinclair, 1993). Other patients may need assistance with mobility or simply advice to get dressed and walk around when ready.

There is overlap between different types of pneumonia but the common classifications, with details of how physiotherapy is modified when appropriate.

Bronchopneumonia

Bronchopneumonia is patchy and diffuse, often favouring the lower lobes. It is common in the immobile and elderly. Early signs are fine crackles that persist despite deep breathing. Physiotherapy is based on hydration and early mobility. Other measures may be needed to increase lung volume (Chapter 6). If excess secretions develop and cannot be cleared independently, assistance is required (Chapter 8).

Lobar pneumonia

When pneumonia is confined to a lobe, localized pleuritic pain is often a prominent feature. Organisms include *Streptococcus*, for which smoking is the main risk factor (Nuorti, 2000) and the less common but more aggressive *Klebsiella*, which may show cavitating consolidation on X-ray. Pain limits breathing and mobility, and, if not controlled by analgesia, may respond to transcutaneous electrical nerve stimulation (TENS).

Pneumocystis carinii pneumonia

People whose defence mechanisms are weakened by HIV, post-transplant medication or severe debility are vulnerable to opportunist infection by organisms that are not normally pathogenic. *Pneumocystis carinii* pneumonia (PCP) is a manifestation of this and is the common first AIDS-defining illness in people with HIV who are not taking prophylactic medication, especially smokers.

Invading fungal organisms damage the alveolar lining and a foamy exudate interferes with gas exchange. Clinical features include dry cough, breathlessness, chills, sweats and hypoxaemia. Some patients are wasted from diarrhoea, malabsorption, dysphagia or mouth ulcers which restrict food intake. They may or may not be pyrexial because immunocompromised people cannot always mount a fever in response to infection. Patients may be reluctant to take a full inspiration because of pleuritic pain or coughing fits. Auscultation may be normal or show fine scattered crackles. The X-ray may be normal at first if immune deficiency delays the appearance of an inflammatory response, but later signs are a perihilar haze, progressing to diffuse symmetrical shadowing and air bronchograms (see Figure 2.16). Sudden deterioration raises suspicions of a pneumothorax.

Non-invasive diagnosis is by X-ray, a low TLCO (p. 60), or exercise-induced oxygen desaturation below 90% (Vilar, 1998). Arterial blood gases show hypoxaemia and a low P_aCO_2 due to breathlessness. Lung biopsy results are variable because of the patchy nature of the disease (Vilar, 1998) but induced sputum, bronchoscopy or bronchoalveolar lavage can assist diagnosis.

Drug prophylaxis is by monthly nebulized pentamidine for patients at risk. Treatment is by early use of a combination of antiviral and other drugs (Vilar, 1998).

Physiotherapists are involved in the following ways:

- Diagnostic assistance by sputum induction (p. 36) or oxygen desaturation during exercise (Chouaid *et al.*, 1993).
- Administration of nebulized pentamidine to the lung parenchyma, using a filter and special nebulizer that produces particles which reach lung tissue without creating side effects in the upper airways. To limit environmental contamination, a separate room is required, preferably with an air extraction system. Prior bronchodilator inhalation reduces the side effects of coughing and bronchospasm, and patients are advised to change position regularly to ensure delivery to all parts of the lung.
- Patients need support when in the grip of acute breathlessness because they are intensely frightened and often think that they are dying. Physical assistance is described on page 169. Positioning depends on the patient's choice, but when breathlessness is severe, minimal handling is preferred. Reassurance can be given because although people with AIDS know that they have an eventually fatal disease, death rarely occurs during episodes of acute breathlessness.
- CPAP (p. 156) at pressures of 5–10 cmH_2O improves gas exchange in patients with severe disease (Miller and Semple, 1991) and buys time for discussion with the patient about further treatment. If required, some patients may choose mechanical ventilation while antimicrobial drugs have time to work. Ventilated patients with PCP rarely have a problem with bronchial secretions and physiotherapy is based on positioning for $\dot{V}_A/\dot{Q}$ matching and maintenance of the musculoskeletal system.

Other measures for people with AIDS and immunocompromise are discussed on page 107.

Nosocomial pneumonia

Pneumonia that develops after hospitalization for more than 48 hours is considered to be hospital-acquired or nosocomial pneumonia. It occurs in 1% of hospitalized patients, including 10% of intensive care patients (Juniper, 1999b), and is the leading cause of hospital-related mortality (Niederman, 1998). The disease may be caused by cross-infection between patients, usually carried by staff, or acquired from other colonized sites such as a nasogastric tube (Guérin, 1997), tracheostomy or the gut. A major cause is misuse of broad-spectrum antibiotics (Fiorentini, 1992). Nosocomial pneumonia involves different pathogens from community-acquired pneumonia.

Legionella pneumonia

Legionnaires' disease is one of the 'atypical' pneumonias. It occurs in local outbreaks, especially in connection with cooling systems or inadequately cleaned small-volume nebulizers (Mastro *et al.*, 1991). Mortality is 5–10%.

Aspiration pneumonia

Pulmonary aspiration is the spilling of gastric contents or foreign matter below the vocal cords. People who have inhaled unfriendly substances such as vomit or gastric acid can develop aspiration pneumonia within 2 hours, although post-anaesthetic patients do not develop the signs and symptoms for some hours (Beards and Nightingale, 1994). A vicious pneumonitis corrodes the alveolar–capillary membrane and leaves a legacy of pulmonary oedema, haemorrhage and necrosis. Aspiration pneumonia should be suspected in

anyone who has experienced a period of unexpected unconsciousness.

Signs of acute aspiration include crackles and wheezes on auscultation, tachypnoea, tachycardia, fever and sometimes cyanosis. Unless aspiration is silent, coughing or choking may occur. X-ray signs of consolidation are evident within a few hours, increase over 24–48 hours, then clear up within 1–2 weeks in uncomplicated cases. The location of these infiltrates helps to identify which lobe is affected,

Material aspirated in the supine position tends to collect in the posterior segments of the upper lobes or apical segments of the lower lobes. Other targets are shown in Figure 4.6.

Risk factors (Beards and Nightingale, 1994) are:

- altered conscious level, e.g. head injury, alcohol intoxication, seizure, stroke
- pregnancy, because of increased abdominal pressure and a high incidence of gastro-oesophageal reflux
- obesity
- stress, anxiety and pain
- children, because of their low gastric pH
- endotracheal tube, tracheostomy or nasogastric tube.

Patients require immediate physiotherapy by postural drainage, percussion, vibrations, shaking and cough or suction. If the patient is able, other techniques to clear secretions can be used (Chapter 8). Associated trauma should be checked because any person found on the floor must first have got there. Once consolidation has set in, as indicated by bronchial breathing, these manual techniques may be less effective, but CPAP may be beneficial, or PEEP for ventilated patients (Behera *et al.*, 1995).

Medical intervention by intubation and suction may be indicated, with fluid replacement to compensate for fluid sequestration in the lungs and systemic tissues. Routine antibiotics are not indicated and tend to encourage secondary infection by resistant organisms (Beards and Nightingale, 1994).

Outcome depends on the volume and type of

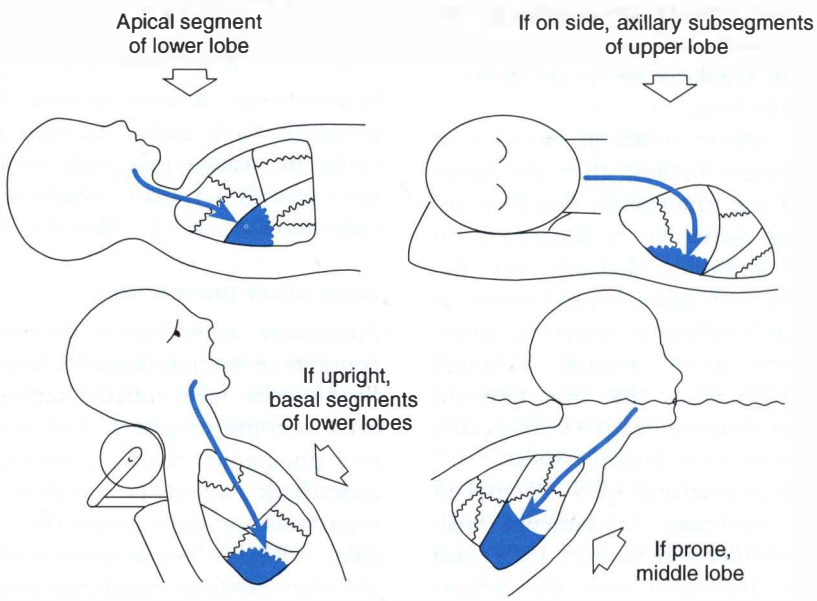


Figure 4.6 Aspiration in varying positions causing pneumonia in different lobes.

aspirate and the immune status of the patient. Severe aspiration contributes to 25% of cases of acute respiratory distress syndrome (Oh, 1997, p. 319). Chronic aspiration is discussed on page 102.

PLEURISY

Pleurisy is inflammation of the pleural membranes, sometimes associated with lobar pneumonia. It causes a pleural friction rub and a wicked localized pain because of the sensitivity of the parietal pleura. This results in rapid shallow breathing. The pain may be eased by heat or TENS. Dry pleurisy sometimes develops into a pleural effusion. This brings relief from symptoms as the raw pleural membranes are separated by fluid.

HIV, AIDS AND IMMUNOSUPPRESSION

Respiratory complications affect the majority of people with the acquired immunodeficiency syndrome (AIDS). These complications manifest as PCP (p. 104), Kaposi's sarcoma (p. 109), lung abscess, tuberculosis or pleural effusion. Necrotic lung tissue may rupture and cause a pneumothorax, which is notoriously difficult to treat in this group of patients (Light, 1993). Oral herpes or a sore mouth can make eating difficult and oxygen therapy intolerable.

In the West, where powerful drugs are available, AIDS is, like COPD, a chronic and treatable disease which is ultimately fatal. The physiotherapist's role is to provide assistance with respiratory problems, mobilization, relaxation, exercise to improve immune function (Lang, 1991), and massage to relieve neuropathic pain.

Other immunocompromised patients are those who are very young, very old, malnourished, addicted to drugs or alcohol, taking steroids or suffering malignancy.

When working with immunocompromised people, specific attention should be given to:

- hand washing, sterilization of equipment and use of a mask by any health worker with a cold, because of the patient's susceptibility to infection
- for HIV patients, autonomy because the syndrome is long-standing and prone to misconceptions
- the patient's many individual requirements, because of the effects of undernourishment, dehydration and, in the later stages of AIDS, the discomforts of terminal disease and its treatment.

Any patient may be HIV-positive and precautions should be universal. Body fluids known to contain the HIV are blood, semen, vaginal secretions and urine. The virus has not been found in normal sputum, but any sputum may contain blood. Precautions against transmission include covering cuts or abrasions with waterproof plasters, wearing gloves during contact with body fluids and using visors to cover eyes, nose and mouth during suction. Masks give some protection.

PULMONARY TUBERCULOSIS

Pulmonary tuberculosis (TB) is not a disease of the past. The incidence is increasing in Europe and the USA, and new drug-resistant strains are emerging. One-third of the world's population is infected by the TB bacillus, which may become active if the host's defence mechanisms are compromised by poor living conditions, drug dependency or HIV infection. TB in an HIV-infected person is an AIDS-defining illness, creating a cruel duet as the two infections exacerbate each other. TB is the only disease likely to be transmitted from AIDS patients to the community.

TB of the lung is the commonest form of the disease, causing 3 million deaths a year, more than any other infection (Empey, 1993). Coughing disseminates infected aerosol, which can remain suspended in the air for hours. Symptoms are fever, night sweats, cough, chest wall pain, weight loss, haemoptysis and breath-

lessness. The X-ray shows cavitating lesions in the most stretched and poorly perfused areas of lung, which are the apices in humans and the bases in bats. Miliary TB shows evenly scattered nodules.

The tubercle bacillus is slow growing and tough, responding only to 6 months of treatment with a combination of powerful antibacterial drugs. The patient is no longer infectious after 2 weeks' treatment, providing sputum is clear of the bacillus. The physiotherapist's role is usually confined to eliciting sputum specimens in a negative pressure room and devising ways to encourage exercise in an isolation cubicle. A high-efficiency particulate air-filtering mask must be used throughout. Patients in isolation need a window, a telephone and reassurance that they are not stigmatized.

ABSCESS

Lung abscess is a focal collection of pus within the lung parenchyma, caused by a virulent event such as inhalation of septic material. It leads to cavitation and necrosis. Patients may have a swinging pyrexia, and the X-ray shows an opaque lesion until communication with the airways is established, when drainage of the necrotic debris shows a ring shadow with fluid line (see Figures 2.8 and 2.17). Medical treatment is by antibiotics. Physiotherapy is effective if the abscess is open, postural drainage being safe so long as the correct antibiotic is given and positioning is accurate and thorough to avoid dissemination of infection.

LUNG CANCER

Carcinoma of the lung is the commonest cancer in the UK and the commonest cause of death from cancer (Simmonds, 1999). It is increasing alarmingly in women as they catch up with men in smoking habits. Mean survival is less than 6 months (Falk, 1997), depending on the type of tumour (Figure 4.7). Most tumours arise in the large bronchi, whose bifurcations are first to be bombarded with tobacco smoke (Figure 4.8). Tobacco causes at least 87% of deaths (Dresler, 1996).

Clinical features are a diffuse or aching chest pain, haemoptysis, clubbing, cough, unresolving pneumonia and breathlessness. Breathlessness is caused by the tumour, the treatment, e.g. radiotherapy, or the effect of having cancer, e.g. cachexia. Systemic symptoms include loss of energy, appetite and weight. Recurrent pneumonia in a smoker is a suspicious sign. A large tumour in a main bronchus may produce

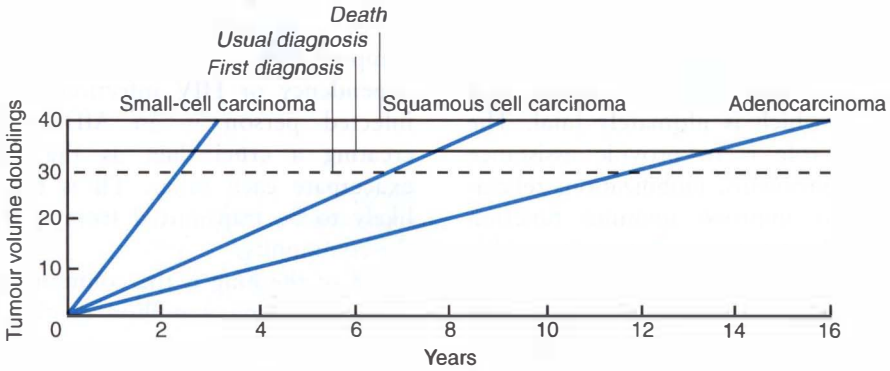


Figure 4.7 Lung cancer growth rates. A tumour usually becomes evident on X-ray when 1 cm in diameter, followed by symptoms, then diagnosis (Redrawn from Bourke, S. J. and Brewis, R. A. L. (1998) *Lecture Notes on Respiratory Medicine*, Blackwell, Oxford.)

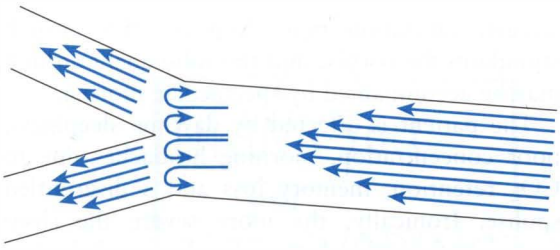


Figure 4.8 Turbulence of airflow at branching of airways.

no radiological change until obstruction causes lung collapse, but stridor or a monophonic wheeze may be heard. Hoarseness indicates involvement of the recurrent laryngeal nerve, which may impair speech and cough.

Lung and other cancers can cause a pleural effusion without tumour growth in the pleura, and this is a poor prognostic indicator (Martinez-Moragon, 1998). An early sign that sometimes appears before diagnosis is hypertrophic pulmonary osteoarthropathy, manifest as pain and swelling of the wrists and ankles (Penson and Rudd, 1997).

The common cancers

A quarter of people with lung cancer suffer from small-cell lung cancer, the most vicious and rapidly spreading of the cancers. Other lung cancers are squamous-cell, large-cell and adenocarcinoma, which are more likely to be localized. Medical treatment is aimed at inflicting the greatest damage to the cancer with the least damage to the patient, but the disease is usually disseminated at presentation. Radiotherapy and/or chemotherapy may ease symptoms or reverse obstructive atelectasis. Multiple medication may cause 'chemotherapy lung', leading to diffuse infiltrates on X-ray and breathlessness. Surgery (p. 265) is occasionally curative. Most people with lung cancer have COPD and need attention to this.

Other cancers

Kaposi's sarcoma

This is a proliferative process that affects the skin, gut and connective tissue of immunocompromised people. Pulmonary Kaposi's sarcoma (KS) is the commonest malignancy associated with AIDS (Miller, 1996) and affects the parenchyma, lymph nodes or pleura, manifesting as progressive dyspnoea and cough, hypoxaemia and sometimes respiratory failure. CT findings include nodules, masses and pleural effusions. KS represents late-stage disease and a poor prognosis. Treatment is by radiotherapy or chemotherapy (Antman and Chang, 2000).

Mesothelioma

This arises in the mesothelial cells of the peritoneum, pericardium or, most commonly, the pleura, and is caused by asbestos exposure. In the pleura it is associated with malignant pleural effusion, chest pain and clubbing. Mesothelioma may not develop until 50 years after exposure (Jefferies and Turley 1999, p. 214), and is usually fatal within 2 years. Palliation is by chest drainage. Asbestos-related deaths are expected to peak in about 2015 (Boylan, 2000).

Bronchoalveolar carcinoma

This represents 3% of primary lung malignancies (Jefferies and Turley 1999, p. 208), developing in peripheral lung, manifesting as local or diffuse infiltrates on X-ray and causing breathlessness, cough and sometimes extreme quantities of watery sputum. It is not caused by smoking.

Metastases and spreading tumours

Metastatic disease carries a poor prognosis but occasionally responds to surgery if the primary tumour is controlled (Kandioler, 1998).

Spreading tumours can cause various complications:

- *Upper airway obstruction* leads to breathlessness and sometimes lung collapse. It can be palliated and sometimes a lung temporarily re-expanded by cryotherapy, laser resection,

localized radiotherapy or stenting to splint open the airway (Mehta, 1999). Stenting may cause an irritating cough.

- *Superior vena caval obstruction* causes oedema, headache, difficulty in breathing, stridor and faintness on bending down. It may be eased by radiotherapy and raising the head of the bed.
- *Pancoast's syndrome* is invasion of anatomical structures such as the chest wall, lymphatics and sympathetic chain (Muscolino, 1997). Consequences include:
  - loss of sympathetic tone
  - if the upper rib is involved, shoulder pain
  - if the inferior brachial plexus is involved, ulnar nerve pain and small muscle wasting in the hand.

Physiotherapy

Physiotherapists may be involved at any stage, including exercise programmes to aid prevention (Francis, 1996), sputum induction (Khajotia, 1991), relaxation (Sims, 1987), postoperative management and terminal care. Weakness and fatigue are frequent accompaniments to the disease and its various treatments. Depression is common and relates to prognosis (Buccheri, 1998), which may be why the encouragement of independence and self-esteem appears to improve survival (Zimmerman *et al.*, 1997).

SLEEP APNOEA

Falling asleep at the wheel is the cause of a quarter of fatal car accidents (Cassel *et al.*, 1996). Sleeping at the wheel occurs 70 times more often in people with sleep apnoea than in normal subjects (Haraldsson, 1990), and one study found that 100% of people with sleep apnoea have experienced near-miss car accidents (Yamamoto, 2000). Sleep apnoea has been blamed for oil-tanker spills, nuclear contaminations and the space shuttle explosion (Smith and Mayer, 1998).

Sleep apnoea exists when breathing stops for more than 10 seconds during sleep. Nocturnal oxygen saturation may drop to 75%, which stimulates the cortex, and the subsequent arousal may be accompanied by spectacular snoring.

The patient is affected by daytime sleepiness, poor concentration, morning headaches due to CO_2 retention, memory loss and a disgruntled spouse. Ironically, the more severe the sleep fragmentation, the deeper the sleep and the less likely the patient is to report sleep problems (Wilkins *et al.*, 1995, p. 358).

Risk of sleep apnoea is increased by smoking (Wetter *et al.*, 1994) and high alcohol intake because of reduced muscle tone (Jalleh *et al.*, 1993). It is doubled by being male, possibly because progesterone is a respiratory stimulant.

Sleep apnoea often goes unrecognized because patients make adjustments to their lifestyle, may not realize the severity of the problem and misinterpret sleepiness as fatigue. Nearly half of people referred for chronic fatigue syndrome have been found to have sleep disorders (Strollo, 1998). Physiotherapists may be the first to suspect the condition.

Types of sleep apnoea

Obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is caused by nocturnal upper airway obstruction despite respiratory effort, and worsens as the night progresses (Charbonneau, 1994). It affects 1–4% of the population (Kiely, 1999) and 10% of patients also have COPD, usually with the blue bloater pattern (Noureddine, 1996).

OSA may be associated with obesity because the thick neck virtually chokes patients in their own fat when muscle tone wanes at night. Children are not immune (p. 430). OSA should be suspected in COPD patients whose nocturnal desaturation is disproportionate to their spirometry.

It takes on average 10 years of snoring before OSA is detected, during which the ongoing vibration appears to disturb the complex musculature of the upper airway, which has to juggle breathing, swallowing, talking, laughing, crying and singing (Strollo, 1998). Diagnosis may not

be forthcoming until unexpected right heart failure develops because of recurrent hypoxaemia. Pulmonary and systemic hypertension, arrhythmias and death from cerebral and cardiac events cause significant mortality. Some 50% of people with OSA are hypertensive in the daytime (Wilkins *et al.*, 1995, p. 358).

Central sleep apnoea

This is associated with neuromuscular disorders, heart failure and abnormal control of breathing, possibly as a result of depressed response to CO_2 during sleep. Snoring is not a characteristic and, in contrast to OSA, there is a lack of respiratory effort. Pure central sleep apnoea is rare and most people with sleep apnoea have both obstructive and central components (Wilkins *et al.*, 1995, p. 359).

Restrictive sleep apnoea

For those who have little respiratory reserve because of disorders such as scoliosis, ankylosing spondylitis or diaphragmatic paralysis, apnoea can occur because of inhibition of accessory muscle action during sleep.

Conditions associated with sleep apnoea

The *Pickwickian* or *obesity–hypoventilation syndrome* is a severe form of OSA seen in markedly obese people. They may require non-invasive ventilation to unload the respiratory muscles (Pankow, 1997). The *Prader–Willi syndrome* is a congenital disorder of obesity, hypotonia and impaired cognition (Smith *et al.*, 1998). *Nocturnal hypoventilation* is an exaggeration of the normal reduction in respiratory drive at night.

Management

Diagnosis is made from symptoms, history and a sleep study to evaluate airflow, effort, S_aO_2, ECG, positioning and limb movements. Sleep apnoea can worsen in hospital because of sedative drugs, the supine position and sleep deprivation.

Management of OSA is firstly by weight loss and smoking cessation if relevant, avoidance of evening alcohol or sedatives and strategies to avoid sleeping supine (Strollo, 1998), e.g. a strategic pillow or backpack at night.

CPAP (p. 156) can be used to pneumatically splint open the upper airway at night. Pressures of 5–15 cmH_2O are advised, and a reduction is often possible after a fortnight (Jokic, 1998), but smart machines are able to titrate the pressure for each individual (Strollo, 1998). CPAP relieves symptoms, lowers mortality and can reverse problems such as road accidents (Janson, 2000), heartburn, bedwetting (Kiely, 1999), impotence and Cheyne–Stokes breathing, as well as acting as a catalyst to weight loss, which itself may allow discontinuation of CPAP (Bradley, 1993). Figure 4.9 shows how CPAP reduces sleep-related fatigue and road accidents. Problems with domiciliary CPAP are:

- noise, in which case the machine can be put

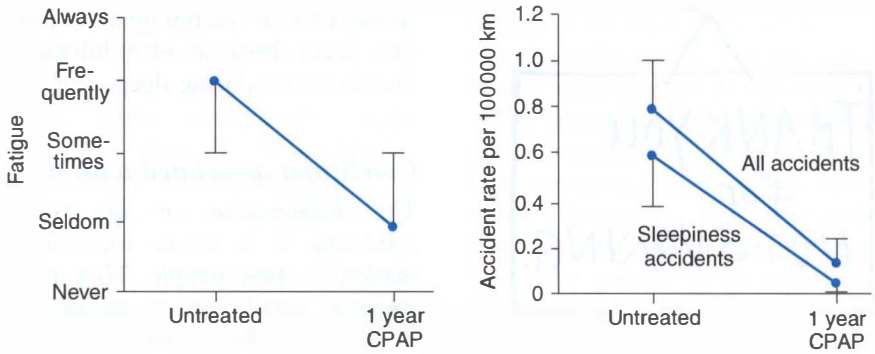


Figure 4.9 Reduction in fatigue and number of accidents in people with sleep-disordered breathing after a year of using CPAP. (From Cassel, W. (1996) Risk of traffic accidents in patients with sleep-disordered breathing. *Eur. Resp.*, 9, 2606–2611.)

in another room and the tubing extended, so long as pressures are re-checked
- dryness, in which case a humidifier can be added, which also requires subsequent rechecking of pressures
- coldness, which can be lessened by keeping the tubing under the bedclothes.

BiPAP (p. 179) is useful for people who cannot tolerate CPAP or for those with hypoventilation (Strollo, 1998).

Other strategies are various contraptions such as a nasopharyngeal airway, tongue retainer or mandibular device (Stradling *et al.*, 1998). Severe disease may require surgery such as tongue reduction, uvulopalatopharyngoplasty, which is as complicated as it sounds, or reconstruction to advance the mandible.

Central sleep apnoea may be helped by drugs to reduce REM sleep or stimulate respiration, CO_2 therapy (Badr *et al.*, 1994) or nocturnal nasal ventilation (Bott *et al.*, 1992). Nocturnal oxygen therapy may be helpful (Wilkins *et al.*, 1995, p. 361), but if there is COPD overlap it may suppress the respiratory drive (Bach and Haas, 1996).

PULMONARY MANIFESTATIONS OF SYSTEMIC DISEASE

The respiratory system is influenced by most systemic disturbances; for example, fever increases minute volume, anaemia hinders oxygen delivery, acid–base imbalance affects breathing and malnutrition predisposes to chest infection. Breathing itself affects heart rate and BP (Fried, 1993). Specific disorders are discussed below.

Cardiovascular disease

Neighbourly relations between heart and lung are reflected in their integrated response to each other's disorders, especially when intravascular pressures are involved.

Pulmonary oedema

Pulmonary oedema is extravascular water in the lungs, usually caused by back pressure from a failing left heart. The main symptom is breathlessness, leading occasionally to a misdiagnosis of asthma, from which the confusing term 'cardiac asthma' arises. Breathlessness caused by pulmonary oedema is distinguished by orthopnoea and paroxysmal nocturnal dyspnoea (p. 31). Fatigue is caused by reduced cardiac output. Radiographic signs are enlarged heart, upper lobe diversion and bilateral fleecy opacities spreading from the hila, which are known as batswing or butterfly-wing shadows (see Figure 2.14). Crackles on auscultation, more evident in dependent lung, are due to the popping open of dependent alveoli compressed by peribronchial oedema.

Non-cardiogenic pulmonary oedema, distin-

guished by a normal-sized heart on X-ray, can be caused by fluid overload, systemic vasoconstriction, oncotic pressure changes (e.g. cirrhosis, malnutrition, nephrotic syndrome) or increased capillary permeability due to toxins or inflammatory damage.

Interstitial pulmonary oedema barely affects lung function but, if the lymphatics become overloaded, fluid squeezes into alveoli, causing alveolar oedema, a widened $P_{A-a}O_2$ gradient and hypoxaemia. If alveolar fluid moves into the airway, it mixes with air and is coughed up as frothy sputum.

Heart failure

If the heart is unable to pump all the blood returned to it, it is said to have failed. Heart failure may be acute or chronic and is a response to heart or lung disease or heart surgery. Compensation for reduced oxygen delivery is by increased sympathetic drive. Decreased oxygen delivery to the kidney leads to salt and water retention.

Heart failure is suspected if a patient with predisposing factors develops oedema, fatigue and breathlessness due to pulmonary oedema. Survival rates are lower than for many cancers (Dargie, 1994), but people can live with chronic heart failure for some years. Indeed, 1% of the population is considered to have heart failure (Cleland, 1996). It is helpful to explain the term to patients because of its misinterpretation in the media as a heart attack or cardiac arrest.

Left ventricular failure (LVF) is the commonest reason for heart failure and is usually caused by coronary heart disease. The failing left ventricle forces up pressure in the left atrium and the pulmonary vascular system behind it, leading to pulmonary oedema.

Treatment of LVF is by dealing with the cause where possible, plus oxygen, venodilator drugs to reduce filling pressures, and diuretics (p. 138). The role of the physiotherapist is limited to giving symptomatic relief by positioning the patient upright, with support of the feet to prevent the inexorable slide down the bed. The supine posture, that anathema for physiotherapists, is particularly unhelpful for patients with an enlarged heart because of compression of the left mid and lower lung zones (Wiener *et al.*, 1990). CPAP can improve gas exchange (Takeda, 1997). If mechanical ventilation is necessary, patients rarely require physiotherapy other than monitoring, positioning and musculoskeletal care.

Right ventricular failure is caused by LVF, valvular disease of the left heart or chronic pulmonary hypertension caused by hypoxic conditions such as COPD. The term *congestive cardiac failure* (CCF) means both right and left heart failure with congestion in the pulmonary and systemic circulations, often associated with COPD.

Pulmonary embolism

Pulmonary embolism (PE) is blockage of the pulmonary vasculature, usually by a blood clot. A small embolus enables secondary blood supply from the bronchial circulation to keep lung tissue viable, but this blood exudes into alveoli, causing haemoptysis and an inflammatory reaction that manifests as sharp localized pleuritic pain, breathlessness, pallor and sometimes pleural rub. Massive embolism causes circulatory collapse, and occlusion of more than 50% of the pulmonary vascular bed is fatal (Jefferies and Turley, 1999, p. 176). PE has been found at autopsy to contribute to death in up to 70% of patients (Wood and Spiro, 2000).

PE is difficult to diagnose clinically, but 70% of patients have evidence of deep vein thrombosis (Edmondson, 1994), 20% of patients show the classic triad of chest pain, dyspnoea and haemoptysis (Reed, 1996), and 10% are asymptomatic (Wood and Spiro, 2000). The X-ray may show a small pleural effusion or a peripheral wedge-shaped shadow indicating infarcted lung. A $\dot{V}/\dot{Q}$ scan is 50% conclusive (see Figure 2.21) and a spiral CT with intravenous contrast medium is 90% conclusive (Hansell, 1998). Recognition of PE in ventilated patients is discussed on page 384.

Prevention of PE is by avoidance of DVT (p. 262). Immediate management of PE is by giving

oxygen and placing the patient supine, thus boosting venous return to the left heart, which is deprived of pulmonary artery flow (Gray, 1992). Treatment is by thrombolytic therapy to dissolve the clot, followed by heparin infusion and sometimes insertion of caval filters. Embolectomy is rarely performed.

It is possible that manual chest techniques or mobilization could dislodge the clot. Theoretically this might be beneficial if the clot moves into a smaller blood vessel, but any active physiotherapy is considered risky until anticoagulation therapy is established.

Kidney disease

Late-stage hypoxaemic respiratory disease impairs perfusion to the kidneys (Howes *et al.*, 1995). Kidney disease and its treatment affect most body systems, the respiratory system being influenced by:

- fluid overload due to kidney dysfunction, leading to pulmonary oedema and sometimes pleural effusion
- breathlessness due to either pulmonary oedema or metabolic acidosis if the kidney is unable to maintain acid–base balance
- muscle wasting due to steroid treatment or uraemia
- following transplantation, opportunistic chest infection due to immunosuppressive drugs
- sleep apnoea associated with end stage renal disease (Kimmel *et al.*, 1989).

Renal support systems are discussed on page 408.

Liver disease

The liver boasts over 500 functions and is served by two blood supplies. For the physiotherapist, precautions when treating people with liver disorders include the following:

- Tracheal suction is performed with caution if clotting factors are abnormal.
- Before nasopharyngeal suction, it is advisable to check for oesophageal varices associated with portal hypertension, in case the catheter enters the oesophagus.
- Impaired manufacture of albumin may disturb fluid balance, especially in advanced liver disease when the kidneys are also involved.
- Lung expansion is restricted if the diaphragm is splinted by hepatomegaly or ascites due to portal hypertension and reduced albumin, and some patients with grossly enlarged livers are immobile and may not be able to roll.
- Encephalopathy is caused by circulating toxins and reduces the patient's ability to cooperate.
- Cerebral oedema causes hyperventilation, which may be severe enough to require mechanical ventilation (Cowley, 1993).
- Asterixis (p. 34) may be associated with liver disease rather than hypercapnia.
- Bilirubin in the plasma of jaundiced patients limits the accuracy of oximetry.

For management of patients in liver failure, see page 408.

Sickle cell disease and thalassaemia

Sickle cell disease is a common genetic disorder characterized by the sickling phenomenon, in which red blood cells crystallize into a sickle shape. The sickled cells become rigid, suffer accelerated haemolysis and are unable to squeeze through small vessels. Morbidity and mortality result from haemolytic anaemia, vaso-occlusion of the microvasculature in multiple organ systems and infections due to a disturbed immune system. Intermittent vaso-occlusive crises are precipitated by:

- exercise
- fatigue
- dehydration
- infection
- cold
- extreme temperature change
- damp housing
- poor diet
- smoking.

When sickling occurs in the pulmonary vasculature, an acute chest syndrome of chest pain, breathlessness, atelectasis and infiltrates develops. The symptoms may be reduced by 2-hourly incentive spirometry (Bellet, 1995). Patients may need assistance with gentle mobilization because of anaemia and fatigue. Surgery is often needed for gallstones or avascular femoral necrosis, and physiotherapy is needed postoperatively to reinforce pain relief and encourage activity. Diaphragmatic splinting due to pain can lead to atelectasis and cause acute chest syndrome (Area, 1994). Patients need advice on joint protection. Ice treatment is contraindicated.

Sickle-call disease is commonest in black people, but Mediterranean and occasionally white people can be affected. The excruciating ischaemic pain of vaso-occlusion mandates that these patients are under the care of a specialist unit, where epidural analgesia (Yaster *et al.*, 1994) or other potent pain relief is available. Non-specialist staff sometimes suspect narcotic abuse in this group of patients, although the risk is less than 1% (Lancet, 1995a). Some patients carry a note from their specialist defining the required analgesia in case of admission to an unfamiliar hospital.

Thalassaemia is an inherited disorder of haemoglobin production leading to anaemia. Patients require lifelong blood transfusions. Iron overload is common and causes an obstructive and/or restrictive lung defect (Dimopoulou, 1999).

Gastro-oesophageal reflux

Gastro-oesophageal reflux (GOR) is the involuntary passage of gastric contents, with its pH of only 1.0, into the oesophagus. It is often associated with cystic fibrosis, asthma or obstructive sleep apnoea (Ing *et al.*, 2000). In adults it is related to chest disease as cause or effect, possibly because the oesophagus and bronchial tree share vagal innervation. In children it is also associated with cough, recurrent croup (Yellon *et al.*, 2000) or spastic cerebral palsy. In infants it is common and usually asymptomatic (Dodge, 1999).

GOR often occurs at night when sleep-related reduction in oesophageal motility slows clearance, allowing refluxed material more time to harm the mucosa. Other risk factors are smoking, alcohol, the extremes of age, chronic aspiration, obesity (Locke *et al.*, 1999) and raised abdominal pressure as occurs in coughing and wheezing. GOR increases the risk of oesophageal cancer (Lagergren *et al.*, 1999).

Symptoms include heartburn, discomfort on swallowing, nocturnal cough, morning hoarseness and regurgitation with a bitter taste in the mouth after recumbence, stooping or large meals. There may be recurrent pulmonary infiltrates on X-ray. Symptomatic children feed poorly and vomit. Vomiting may be the only indication of GOR in people with CF. Confirmation of the diagnosis is by endoscopy, barium swallow or oesophageal pH monitoring.

Management is by encouragement of side-lying (Dean, 1997), raising the head of the bed at night, weight reduction if appropriate, and drug review. Patients should avoid late evening meals, large meals, stooping, smoking, alcohol, caffeine and aminophylline, which relaxes the cardiac sphincter. Anti-reflux H_2-antagonist drugs, or occasionally surgery (Hogan, 2000), may be indicated.

The head-down postural drainage position is to be avoided (Button *et al.*, 1994) but the effect of different positions is variable, and slumped sitting may be worse because of increased abdominal pressure. No physiotherapy should be given immediately after meals. GOR in children is covered on page 427.

Diabetes

Over 15% of the world's population has diabetes (Roizen, 1997), 2% in the UK (Marshall, 1996). Some develop pulmonary complications due to collagen and elastin changes (Ljubic *et al.*, 1998). Surgical patients are at risk because lack of insulin leads to unrestrained catabolism and raised circulating glucose, so that insulin is required to avoid dehydration and acid–base disturbance.

Complications of diabetes include fluid upset,

hypotension and pressure sores. In relation to exercise, people with stable diabetes benefit so long as they maintain hydration, look after their feet and when necessary increase their insulin and carbohydrates to avoid hypoglycaemic events. Warning signs of hypoglycaemia include light-headedness, weakness and fatigue, and a sugar source must be available during exercise sessions. Haemodynamic responses to exercise may be attenuated by autonomic neuropathy.

Drug-induced lung disease

Immunological or cytotoxic lung damage can be caused by medication. Examples are interstitial pneumonitis caused by amiodarone and asthma caused by aspirin. Reactions to illicit drug use depend on the substance, contaminants, route of administration and use of shared equipment.

Narcotics and other drugs that depress consciousness can promote basal atelectasis and aspiration, increasing the likelihood of pneumonia and lung abscess. Narcotics also increase membrane permeability, and heroin can cause non-cardiogenic pulmonary oedema (Sykes and Young, 1999, p. 106), either immediately or up to 24 hours later. The antidote to narcotic poisoning is naloxone, which can itself induce pulmonary oedema (Heffner, 1990).

Cocaine can damage any major body organ, but two-thirds of deaths from cocaine use are due to homicide, suicide, traffic accidents and falls (Boghdadi, 1997). Crack cocaine causes respiratory symptoms in 25% of users, including necrosis of the nasal septum and alveolar haemorrhage (Heffner, 1990).

Reactions to Ecstasy are unpredictable, depending on contaminants and the degree of associated exercise. Frequent use diminishes the positive effects and increases the negative effects. Dangerous effects include arrhythmias, hyperthermia (Dobbs and Coad, 1999), ARDS (p. 411), renal failure, psychosis, convulsions and barotrauma (Rezvani, 1996).

Cannabis smoking appears to be relatively benign (Lancet, 1995b) in the short term because of its brief duration of action, but acute central nervous system effects include reduced reaction times and forgetfulness (Sharpe, 2000). Long-term use can cause memory loss (Campbell, 1999a), bullae (Johnson, 2000) and problems related to the tobacco with which it is usually mixed. Non-psychoactive preparations have been used beneficially for bronchospasm, pain, nausea, poor appetite (Campbell, 1999a) and multiple sclerosis (Consroe *et al.*, 1997). Patients report a beneficial effect for glaucoma and other visual problems, fatigue, tremor, imbalance, sexual dysfunction and bowel and bladder problems (Campbell, 1999a).

Damage caused by the administration of illicit drugs include the following:

- Intravenous drug users risk septic thrombophlebitis and pulmonary emboli.
- As sites for peripheral venous access diminish, venepuncture of neck veins creates a risk of pneumothorax, and bullae may occur in those who also smoke tobacco (Heffner, 1990).
- Injecting crushed tablets can damage lung tissue.
- Paint inhalation provokes airway obstruction and alveolitis.
- Glue sniffing increases pulmonary artery pressure and decreases gas transfer (Heffner, 1990).

CHEST INFECTION

Infection from viruses, bacteria or fungi can occur anywhere from the upper respiratory tract to the lung parenchyma. Chest infection includes anything from acute bronchitis, a common and usually self-limiting viral infection of the upper bronchial tree, to life-threatening pneumonia. Influenza is particularly unpredictable, occurring in periodic pandemics including the 1918 Spanish flu, which caused more deaths than the First World War (Wiselka, 1994). Viruses and bacteria damage cilia and slow mucus clearance (Wills and Cole, 1996).

Predisposing factors are being young, old, immunocompromised, stressed (Cobb, 1996) and having chronic lung disease. Symptoms

include fever, malaise and cough. Chest infections are a common cause of exacerbation of lung disease (Wilson, 1988). Most patients do not benefit from antibiotics (Kuyvenhoven, 2000) but these drugs are still widely used (Liu and Douglas, 1998). Physiotherapy is required if patients are unable to clear secretions, and to ensure full rehabilitation.

RESPIRATORY FAILURE

Failure of the respiratory system to provide adequate gas exchange for metabolic requirements is known as respiratory failure (RF). This is divided into the following:

- *Type I (hypoxaemic) RF* is failed oxygenation, represented by P_aO_2 below 8 kPa (60 mmHg). It is caused by failure of the gas-exchanging function of the respiratory system and can be acute (e.g. pneumonia) or chronic (e.g. pink puffer type of COPD).
- *Type II (hypoxaemic and hypercapnic) RF* is failed ventilation, represented by P_aCO_2 over 6.7 kPa (50 mmHg) as well as hypoxaemia. It is caused by failure of the respiratory pump and can be acute (e.g. severe acute asthma) or chronic (blue bloater type of COPD, severe restrictive disease, Pickwickian syndrome). Type II RF is also known as ventilatory failure and is a clinical manifestation of impaired central respiratory drive, muscle weakness or fatigue, reflected by respiratory muscle strength falling below 30% of normal (Simonds, 1996, p. 9). It is accompanied by a fall in pH until renal compensation takes effect, which allows assessment of the relative degrees of acute and chronic hypoventilation (Curtis, 1994).

The process of respiration includes more than gas exchange in the lung, but the term respiratory failure is reserved for disorders that result in a disturbance of arterial blood gases only.

Respiratory insufficiency occurs when adequate gas exchange is maintained but at great cost to the breathing mechanism. It is sometimes a sign of impending respiratory failure.

MINI CASE STUDY: MS TP

Identify the problems of this 24-year-old woman who has been admitted with pneumonia, then answer the questions.

Background

SH: unemployed, mobile, independent.
HPC: heroin user.

Subjective

Well.

Objective

Patient in nightie, tucked up in bed.
Apyrexial.
Fluid balance normal.
S_aO_2 normal.
Auscultation – bronchial breathing left lower lobe, no crackles.
Respiratory rate normal.
Breathing pattern normal.

Questions

1. Identify the consolidation in Figure 4.10

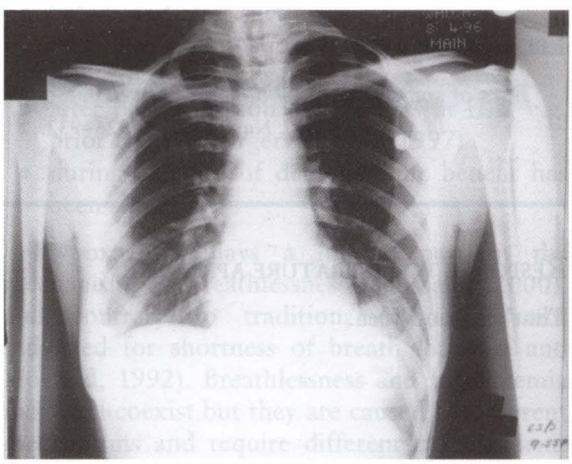


Figure 4.10 Ms TP.

2. Analysis?
3. Problems?
4. Goals?
5. Plan?

RESPONSE TO MINI CASE STUDY

1. Location of consolidation

Area of consolidation seen behind left pierced nipple.

2. Analysis

No accessible secretions. No atelectasis. No desaturation.
Bronchial breathing may linger but is not causing problems.
It is not necessary to 'treat the X-ray'.

3. Problems

No physiotherapy problems at present.

4. Goals

Self-rehabilitation.

5. Plan

Advise patient to get dressed and mobilize.
Discharge.

LITERATURE APPRAISAL

Bars of soap, if used, should be kept dry.

Br. J. Nurs. 1995; 4: 926–932.

RESPONSE TO LITERATURE APPRAISAL

That's a good idea.

RECOMMENDED READING

Antman, K. and Chang, Y. (2000) Kaposi's sarcoma. *N. Engl. J. Med.*, 342, 1027–1038.

Cleland, J. G. F. (1996) Heart failure. *Update*, 52, 658–663.

Daniels, S. K. (1998) Aspiration in patients with acute stroke. *Arch. Phys. Med. Rehab.*, 79, 14–19.

Douglas, N. (1995) The sleep apnoea/hypopnoea syndrome. *Practitioner*, 239, 194–197.

Edmondson, R. (1994) The causes and management of pulmonary embolism. *Care Crit. Ill*, 10, 26–30.

Henderson, A. (1994) Chronic respiratory failure. *Practitioner*, 238, 345–350.

Horner, J. (1988) Aspiration following stroke. *Neurology*, 38, 1359–1362.

Johns, C. J. and Michele, T. M. (1999) The clinical management of sarcoidosis. *Medicine*, 78, 65–111.

Johnston, I. D. A., Prescott, R. J. and Chalmers, J. C. (1997) British Thoracic Society study of cryptogenic fibrosing alveolitis. *Thorax*, 52, 38–44.

MacFarlane, J. (1995) Acute pneumonia in the hospital patient. *Hosp. Update*, 21, 19–24.

McClure, J. (1993) The role of physiotherapy in HIV and AIDS. *Physiotherapy*, 79, 388–393.

Michie, J. (1994) An introduction to lung cancer. *Physiotherapy*, 80, 844–847.

Miller, R. F. (1995) *Pneumocystis carinii* pneumonia. *Thorax*, 50, 191–200.

Mills, N. (2000) Does Tai Chi/Qi Gong help patients with multiple sclerosis? *J. Bodywork Movement Ther.*, 4, 39–48.

Nollet, F., Reelen, R. and Prins, M. (1999) Disability and functional assessment in former polio patients. *Arch. Phys. Med. Rehab.*, 80, 136–143.

Thistlethwaite, J. E. (1998) Respiratory tract infections. *Update*, 56, 108–112.

5 General management

Summary

Introduction

Respiratory medicine has been transformed in the past 20 years by technical advances and an understanding that, for these advances to be effective, patients must become central players in their own care. This chapter looks at current knowledge in the medical management of respiratory disorders.

Oxygen therapy

For over 200 years, oxygen has been much used and sometimes abused. It is an odourless, colourless drug that has side effects and specific risks, but, with rational prescription, precision of administration and objective monitoring, it is a potent therapy for the respiratory patient.

Indications

Supplementary oxygen is rarely indicated for anything other than hypoxaemia. It should be prescribed if resting P_aO_2 is below 8 kPa (60 mmHg) or S_aO_2 below 90%. Potential or temporary hypoxaemia is included, e.g.:

- before and after suction
- first-time administration of bronchodilator drugs in case of adverse effects
- after premedication for patients at risk, e.g. prior to heart surgery (Royse, 1997)
- during exercise, if demonstrable benefit has been shown.

Hypoxaemia plays a limited part in the mechanism of breathlessness (Janssens, 2000), and contrary to tradition, oxygen is not indicated for shortness of breath (Stewart and Howard, 1992). Breathlessness and hypoxaemia may well coexist but they are caused by different mechanisms and require different management. Oxygen is considered an expensive placebo if used as a routine tonic for breathlesness (Leach

and Bateman, 1994). The following may be exceptions:

- Terminally ill patients who are breathless may find some relief with oxygen therapy (Bruera *et al.*, 1993).
- Breathlessness on exercise may or may not be relieved by supplementary oxygen (Dean *et al.*, 1992; McDonald, 1995) and patients should be assessed individually. Before or after exercise, oxygen appears unhelpful (Williamson, 1993). For people with interstitial lung disease, oxygen is likely to improve exercise performance (Harris-Eze, 1996).

Prescription 'as required' is never appropriate (Hodgkin *et al.*, 2000, p. 142). It makes no physiological sense because chemoreceptor signals are not consciously appreciated and people do not feel a physical need for oxygen. Education is more beneficial than the psychological crutch of an expensive drug.

Assessment for acute and long-term oxygen therapy includes nocturnal monitoring by oximetry, because day and night requirements are poorly related (Schenkel, 1996). This is particularly important for patients who have a poor hypercapnic ventilatory response and run a greater risk of nocturnal hypoxaemia (Vos *et al.*, 1995).

Limitations

- Directing oxygen into the throat does not guarantee its arrival at the mitochondria. Tissue oxygenation also depends on lung perfusion, haemoglobin levels, cardiac output, vascular sufficiency and tissue perfusion.
- Oxygen does not improve ventilation directly, although ventilation may be enhanced by oxygen delivery to components of ventilation such as the respiratory muscles.
- If hypoxaemia is due to a large shunt (p. 13), benefit is limited because shunted blood does not 'see' the added oxygen.

Complications

- High concentrations of inspired oxygen may impair the respiratory drive in people with hypercapnic COPD (p. 125).
- Excess oxygen depletes protective antioxidants. This causes oxygen toxicity, an inflammatory response of lung tissue following exposure to 100% oxygen for between 40 hours and 7 days (Heulitt, 1995). Any organ can be harmed but the lung is exposed to the highest PO_2. Oxygen toxicity impairs the actions of cilia, macrophages and surfactant, and the patient experiences substernal pain, cough and dyspnoea. The risk of oxygen toxicity is increased by high-volume ventilation and malnutrition, but pre-existing lung disease affords some protection (Durbin, 1993). If oxygen toxicity is suspected, monitoring by P_aO_2 is advisable because the shape of the oxygen dissociation curve means that measurements of S_aO_2 at high levels of oxygenation are less sensitive.
- Blindness may be caused if neonates are given high concentrations of oxygen (p. 436).
- 'Absorption atelectasis' can occur if absorption of oxygen from alveoli exceeds replenishment of alveolar gas, so that the alveoli are no longer held open by a cushion of inert nitrogen. This can occur in mechanically ventilated patients receiving more than 70% oxygen at low tidal volumes (Pilbeam, 1998, p. 160). Absorption atelectasis is exploited when oxygen is used to increase absorption of air from a pneumothorax (p. 100).
- Discomfort can be caused by dry mucous membranes, eye irritation, a sense of being smothered or excess work of breathing caused by inadequate flow. A patient 'oxygenating' his/her forehead is a familiar sign of this problem.
- Oxygen is not addictive, but dependency occurs when patients rely on it unnecessarily.
- Oxygen creates a fire hazard by supporting combustion. Smoking is banned.

Delivery devices

> *'The nasal specs caused so many problems; they kept falling out or were too tight. They were one of the most uncomfortable appliances, yet better than the mask which seemed to suffocate you.'*
>
> Ludwig, 1984

Low-flow (variable performance) mask

These simple masks (Figure 5.1) provide only a proportion of the patient's inspired gas. They deliver a flow rate that is less than the patient's peak inspiratory flow (PIF) so that room air is sucked in through holes in the mask to dilute the oxygen. The fractional inspired oxygen concentration (F_IO_2) therefore varies with the patient's own flow. The more rapid the patient's ventilation, the more room air is entrained and the lower the F_IO_2. This provides inaccurate and uncontrolled oxygen but is adequate for certain patients.

Flows of 6–8 L/min provide approximately 40–50% oxygen, i.e. an F_IO_2 of 0.4–0.5 (Gribbin, 1993). The flow should be maintained above 5 L/min to avoid rebreathing CO_2. (Leach and Bateman, 1993).

Simple masks are suitable when accurate concentrations are not necessary, e.g. after routine surgery. F_IO_2 is altered by changing the oxygen flow rate at the flow meter.

High-flow (fixed performance) mask

These masks, also known as Venturi masks, deliver a prescribed gas mixture at flows intended to be greater than demand, i.e. higher than the patient's PIF (Figure 5.2). This minimizes rebreathing expired air from the mask, even if it is loosely fitted, and delivers an accurate F_IO_2 as specified on the device. Oxygen rushes from a nozzle and entrains a proportion of room air through a fixed-size entrainment port, so that a specific concentration is delivered. This concentration depends on the size of the entrainment ports, not the patient's PIF, and is reasonably accurate.

The flow indicated on the colour-coded Venturi valve is the minimum, and can be increased as high as the patient requires for comfort.

If humidification is required, large-volume nebulizing humidifiers are available that can deliver a specific F_IO_2 (p. 188). Venturi masks cannot be humidified effectively because moisture is condensed on reaching the Venturi mechanism and can upset the delicate balance of entraining the correct proportion of air. Details of humidifiers are on page 185.

High-flow systems are used for:

- patients needing an accurate F_IO_2, e.g. hypercapnic COPD patients who are depen-

Figure 5.1 Oxygen delivery systems. From left: simple mask, nasal cannula, transtracheal catheter (From Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester, with permission.)

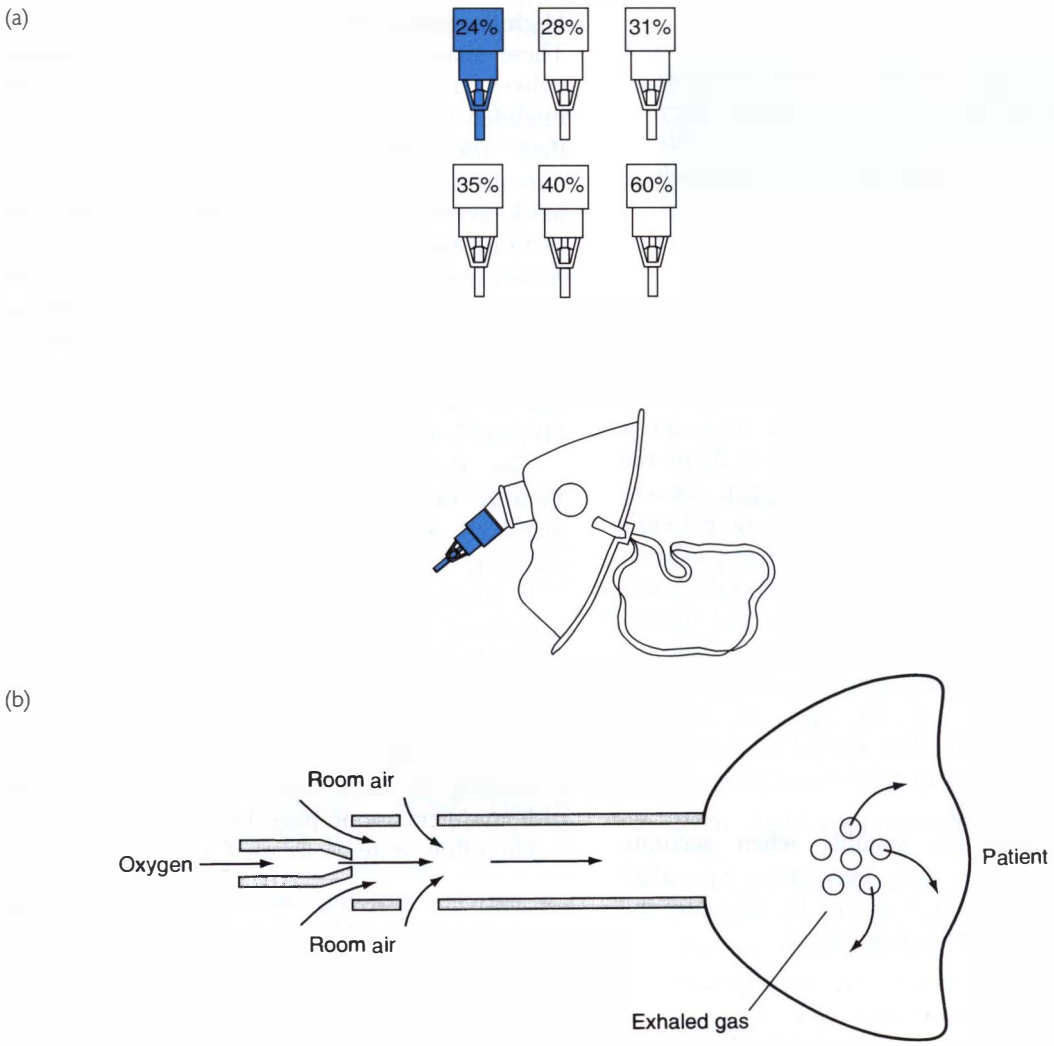


Figure 5.2 (a) High-flow fixed performance 'venturi' mask with colour-coded valves to entrain room air and produce different oxygen percentages (Intersurgical with permission). (b) Interaction of oxygen flow and entrained air.

dent on their hypoxic drive and need controlled oxygen therapy (Figure 5.4)
- breathless patients whose PIF is too high to tolerate a low-flow system.

High- and low-flow masks relate to high and low accuracy, not to high and low F_IO_2.

High-concentration reservoir mask

Reservoir systems (Figure 5.3) incorporate a 1 litre reservoir bag and deliver high-percentage oxygen. During exhalation, oxygen fills the bag instead of being wasted, then during inhalation this oxygen enriches the inspired gas (Branson, 1993).

A non-rebreathing system has valves at the reservoir bag and side vents to prevent expired CO_2 mixing on exhalation and room air mixing on inhalation. It delivers 55–90% oxygen at 6–15 L/min. A *partial-rebreathing* system has no

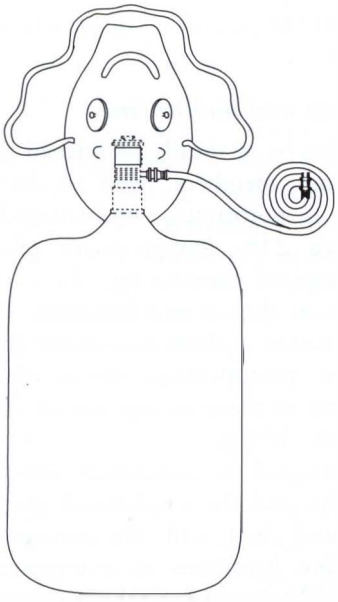


Figure 5.3 High-concentration reservoir mask (Intersurgical, with permission).

valve and about one-third of the expired CO_2 enters the bag, delivering 35–60% oxygen at 6–15 L/min (Bolgiano, 1990).

The system provides a fixed F_IO_2 if flow is sufficient to keep the bag inflated during inspiration. For breathless patients this may require up to 12 L/min to prevent extra WOB being imposed. The bag must be allowed to fill with oxygen before fitting to the patient, and must be kept inflated throughout the respiratory cycle, otherwise the patient is at risk from the combination of a sealed system and inadequate flow. Reservoir masks cannot be humidified.

Nasal cannula

Cannulae, or prongs, deliver oxygen directly into the nostrils so that the patient can talk, cough, eat and drink unhindered (Figure 5.1). 80% of the oxygen is wasted (Tiep, 1991). They are low-flow systems but Branson (1993) gives rough guidelines on the percentage delivered to stable awake patients at rest:

- 1 L/min delivers 24% oxygen
- 2 L/min delivers 28% oxygen
- 3 L/min delivers 32% oxygen
- 4 L/min delivers 36% oxygen.

Flows above 4 L/min may cause irritation and damage the nasal septum. In theory, 8 L/min can deliver 50% oxygen, but high flows provide little additional oxygen because the anatomical reservoir of the nasopharynx is already filled with oxygen (Wissing, 1988). Mouth-breathers are partly accommodated by entrainment of oxygen into this reservoir during exhalation, but F_IO_2 is higher with the mouth closed (Dunlevy and Tyl, 1992). Drying of mucus membranes is not helped by humidification (Campbell *et al.*, 1988), but lanolin gives some relief (not petroleum jelly or Vaseline, which are petrol-based and react with oxygen). The flow rate can be marked on the flow meter with tape to remind patients and others of the correct setting.

Indications for nasal cannulae are:

- long-term oxygen therapy
- patients who find masks uncomfortable
- confused patients, especially if a high concentration is needed, when cannulae can be used with a mask in case the mask is pulled off
- hypoxaemic patients using an incentive spirometer, inspiratory muscle trainer, ultrasonic nebulizer and for certain patients using a jet nebulizer (p. 141).

Nasal catheter

These devices are inserted, after lubrication, into one nostril to reach just behind the uvula and are then taped to the face. They have several holes near the tip so that the force of the oxygen flow is diffused, but patients often complain of a sore throat. Catheters are impractical in infants because they occlude most of the nasal airway, and are often not tolerated by children. They are sometimes used for short periods when a device is needed that must not become dislodged. A flow of 3–4 L/min usually delivers inspired concentrations of 30–40% (Sykes and Young, 1999, p. 100).

Transtracheal catheter

Small plastic transtracheal catheters can be surgically introduced into the trachea, percutaneously or through a tunnelled route, for long-term oxygen therapy (Figure 5.1). They are suited to patients who are sufficiently motivated to follow a protocol of self-care including irrigation with saline. Advantages are:

- halving the flow rate required, leading to extended portable use (Simonds *et al.*, 1996, p. 131)
- high patient adherence because of its unobtrusive presence and a comfortable nose (Kampelmacher *et al.*, 1997).

A major disadvantage is the need for surgical placement, with accompanying risks of infection, surgical emphysema, haemoptysis, displacement and dermatitis.

Tent

Humidified oxygen is still occasionally delivered to children via tents, but these are isolating, wet and deliver fluctuating levels of oxygen. Oxygen escapes each time the edges are untucked. If undisturbed, levels of 50% oxygen can be achieved but CO_2 retention then becomes a problem.

Head box

Clear plastic boxes over the heads of babies control the delivery of humidified oxygen. High flows are required and care should be taken to direct the gas flow away from the baby's face and to ensure that the edges of the box do not rub the skin. Alternatives are oxygen chairs, which incorporate a plastic canopy or hood to deliver oxygen to an upright baby.

T-piece circuit

A T-piece delivers oxygen to an intubated spontaneously breathing patient. It is a large-bore non-rebreathing circuit that attaches to the tracheal tube. Humidified oxygen is delivered through one end and exhaled gases leave through the other. So long as the flow rate is greater than the patient's PIF, it acts as a high-flow device.

Prescription and monitoring

Oxygen must be medically prescribed in writing, but all team members need to be involved because it is notoriously mismanaged. Studies have shown 21% inappropriate prescription, 86% inadequate monitoring, 56% inaccurate administration (Leach and Bateman, 1993) and 88% termination without assessment (Fitzgerald, 1988). One postoperative survey showed the mask staying in place in one out of 20 patients (Baxter *et al.*, 1993).

PIF is affected by respiratory drive, inspiratory muscles and the mechanical properties of the lungs and chest wall. On average it is 40–50 L/min, but breathless or exercising patients may reach 120 or even 200 L/min. Extra energy is then required to entrain room air, so that reversal of hypoxaemia is at the expense of increased WOB, inspiratory muscle fatigue and sometimes hypercapnia (Dodd *et al.*, 1998).

Prescription should take account of patient comfort, including a high enough flow rate for breathless patients with a high PIF, in the same way as with CPAP (p. 158). Venturi masks can be accurately titrated, as shown in Table 5.1.

Table 5.1 Oxygen concentrations delivered to the patient from high-flow systems; flow from the oxygen source can be altered to provide different total flows to the patient according to need, while maintaining control of the concentration (adapted from Dodd *et al.*, 1998)

Concentration	Oxygen flow	Total flow
24%	2 L/min	50 L/min
	3 L/min	75 L/min
	4 L/min	100 L/min
28%	4 L/min	44 L/min
	6 L/min	66 L/min
	8 L/min	88 L/min
35%	8 L/min	45 L/min
	12 L/min	67 L/min
	16 L/min	90 L/min
40%	10 L/min	41 L/min
	15 L/min	62 L/min
	20 L/min	82 L/min
60%	15 L/min	30 L/min

For patients requiring a flow rate above 100 L/min, a high-flow generator can be used, i.e. a CPAP (p. 156) system without a pressure valve.

Oximetry is required to monitor response, and astute budget holders find it cheaper to supply all their relevant beds with oximeters rather than waste unnecessary oxygen. For breathless patients, monitoring respiratory rate indicates if fatigue is severe, but the most sensitive monitor is the patient, who may demonstrate excess WOB subjectively or objectively (p. 36), or, if asked, say that they are not receiving enough flow.

PIF is reduced at night. A simple mask delivers a higher F_IO_2 for the lower PIP, which could tip some patients into respiratory failure (Dodd *et al.*, 1998). For those using a Venturi mask, a lower flow rate may need to be prescribed.

Medical prescription should therefore specify:

- method of delivery
- flow rate and/or F_IO_2
- nocturnal modifications.

Acute oxygen therapy

In the acute setting, oxygen should be administered continuously unless hypoxaemia has been demonstrated only in specific situations such as sleep, eating or exercise (AARC, 1992).

People with hypercapnic COPD vary in their response to oxygen. Sustained hypercapnia has left a small proportion (Singer and Webb, 1997, p. 2) dependent on hypoxia as a ventilatory stimulus (p. 71). Uncontrolled oxygen may deliver excess F_IO_2, leading to gradual hypoventilation, drowsiness and respiratory acidosis, which can be lethal. Normocapnic COPD

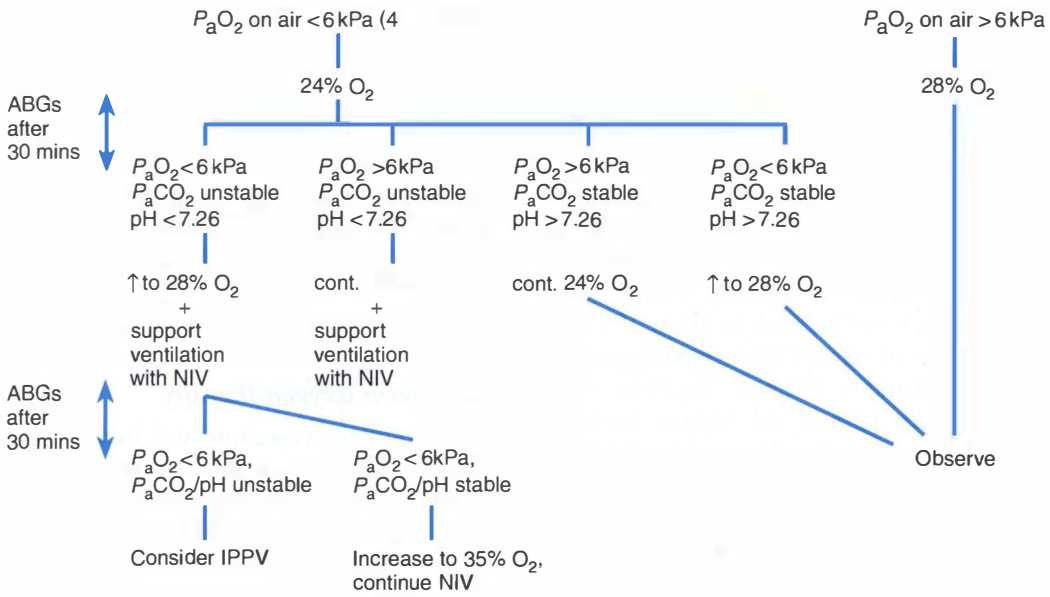


Figure 5.4 Flow chart of controlled oxygen therapy for exacerbations of hypercapnic COPD, showing the levels of P_aO_2 and P_aCO_2 that indicate the need for supplementary oxygen and ventilatory support respectively. Arterial blood gases are taken 30 min after each change in treatment, and treatment is adjusted accordingly. A more detailed flow chart is illustrated in Gribbin (1993). *ABGs = arterial blood gases; IPPV = intermittent positive pressure ventilation; NIV = non-invasive ventilation.*

patients are not at risk (Fulmer and Snider, 1984), nor are people with other disorders, except occasionally those with acute severe asthma (Wissing, 1988) or restrictive disorder (Bach and Haas, 1996, p. 228).

Patents with a limited response to high P_aCO_2 require controlled oxygen therapy titrated to their individual response in order to preserve their respiratory drive. Simple low-flow systems are inadequate. Nasal cannulae are unsatisfactory in acute disease because exhausted patients may hypoventilate and entrain little room air, thus receiving dangerously high F_IO_2 levels (Davies and Hopkin, 1989). If nasal cannulae are necessary for patient comfort, monitoring of blood gases is required.

Controlled oxygen is best delivered by a high-flow Venturi mask. Arterial blood gases are first taken on air, and patients with the lowest P_aO_2, who are most at risk, are given a low F_IO_2 to avoid the possibility of upsetting the respiratory drive. This must only be temporary while the patient is monitored. After 30 minutes, if the P_aCO_2 is stable or rises by no more than 1.3 kPa (10 mmHg), the F_IO_2 can be increased if indicated. If the P_aCO_2 rises excessively, ventilatory support is needed, preferably by non-invasive means, in order to reduce the WOB. At the same time, F_IO_2 is increased if indicated (Figure 5.4).

Several arterial stabs may be required, for which local anaesthesia is specified in both UK and American guidelines (Lightowler, 1996). An alternative is arterialized capillary blood taken almost painlessly from the earlobe (Dar, 1995).

Small amounts of oxygen can relieve hypoxaemia in these patients because reactions take place on the steep part of the oxygen dissociation curve. In practice, the danger of giving too much oxygen is commonly overestimated and patients can be deprived of much-needed oxygen. Most COPD patients do not develop CO_2 retention (Oh, 1997, p. 232), and acute hypercapnia should not be interpreted as a response to high F_IO_2 as it may be due to a deteriorating condition. Hypoxaemia is more dangerous than hypercapnia, and if the guidelines in Figure 5.4 are not followed, oxygen must be titrated to keep S_aO_2 at 90–92% (Oh, 1997, p. 232). Some physicians are happy for patients with COPD to be given high levels of oxygen so long as they are kept under observation in a high-dependency area.

Intermittent oxygen therapy given in the acute phase of COPD (Figure 5.5), especially for hypercapnic patients, is like intermittent drowning (Hanning, 1995). Oxygen stores in the body are $<1.5\%$ of CO_2 stores (Chin *et al.*, 1997) because CO_2 is needed for acid–base balance, so if F_IO_2 is allowed to fall, CO_2 crowds out oxygen (Collins, 1976).

Patients with acute problems other than hypercapnic COPD, e.g. those with pneumonia or acute asthma, may need a generous F_IO_2 of 0.4–0.6 or more, delivered at high flow rates if they are breathless.

Postoperatively, oxygen prescription depends on the patient and type of surgery. Hypoxaemia may be transient, and low-risk patients usually require only a few hours' oxygen after surgery, but for people with lung disease or those undergoing heart or lung surgery, several days and nights of supplementary oxygen may be required (p. 250).

Patients on acute oxygen therapy should only have their mask removed for expectoration or other brief reasons. Oximetry is required for prescription, monitoring and withdrawal of oxygen. All patients should have an oxygen saturation chart, which also encourages reluctant patients to accept the need for initiation and withdrawal of their oxygen.

Long-term oxygen therapy

Only smoking cessation and long-term oxygen therapy (LTOT) can increase survival in COPD patients with severe hypoxaemia (Scalvini *et al.*, 1999). Accurately prescribed domiciliary oxygen has also shown the following benefits:

- ↓ cor pulmonale
- ↑ quality of life
- ↑ sleep
- ↓ exacerbations and hospital admissions (Leach and Bateman, 1994)

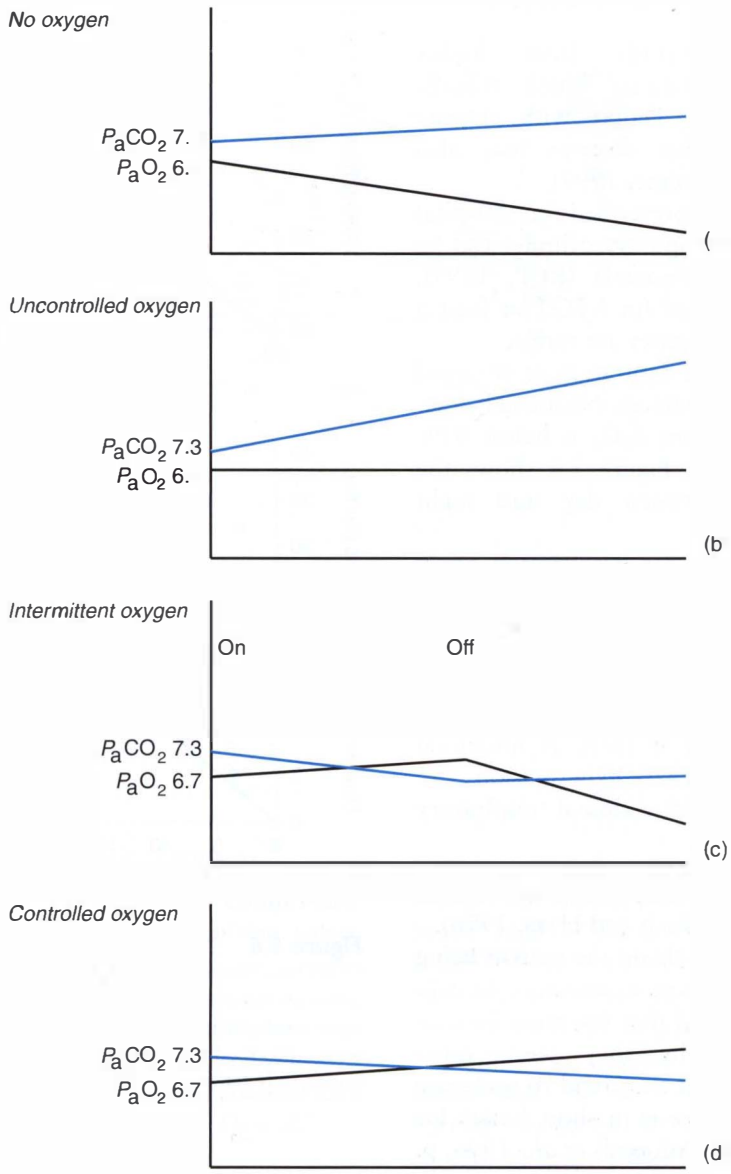


Figure 5.5 Potential effects of different methods of oxygen administration on arterial blood gases (in kPa) for COPD patients in acute hypercapnic respiratory failure: (a) continued deterioration; (b) uncontrolled oxygen – in this case delivering excessive oxygen, leading to reduced respiratory drive, hypoventilation and further P_aCO_2 retention; (c) gradual hypercapnia and rapid hypoxaemia; (d) normalization of blood gases.

- improvement or stabilization of disease progression (Simonds *et al.*, 1996, p. 117).

Improvement stems mainly from higher nocturnal oxygen saturation, which relieves pulmonary hypertension. People with chronic hypoxaemia due to other diseases may also benefit (Petty and O'Donohue, 1994).

LTOT must not be prescribed on hospital discharge, although a temporary cylinder can be supplied for severe hypoxaemia (RCP, 1999). Patients should be assessed for LTOT at least a month later, when blood gases are stable.

Monitoring of oxygen saturation is required during sleep, rest and exercise. Nocturnal desaturation is likely if daytime S_aO_2 is below 93% (Little *et al.*, 1999), but Figure 5.6 shows the limited relationship between day and night oxygen requirements.

The following criteria for prescription apply in the UK (RCP, 1999):

- chronic stable hypoxaemia with P_aO_2 < 7.3 kPa (55 mmHg) breathing air, on two samples taken at least 3 weeks apart, or up to 8 kPa (60 mmHg) if there is nocturnal hypoxaemia or pulmonary hypertension
- disabling dyspnoea in terminal respiratory disease.

Ongoing need for LTOT should be verified annually by a physician (Bach and Haas, 1996).

Prescription also depends on the patient being willing to use it. Education is essential. Rudkin and White (1995) showed that less than 50% of patients understood why they were using oxygen, which means that they tend to underuse it (Make, 1994) or misuse it in short bursts for 'relief of breathlessness' (Simonds *et al.*, 1996, p. 128). Many unnecessarily restrict their activity.

Cylinders and concentrators must be kept away from heaters, and tubing must be positioned to avoid falls. Prescribers, too, must educate themselves, as shown by studies indicating that only seven out of 60 eligible patients were prescribed LTOT and, conversely, only 33% of patients receiving LTOT fulfilled the criteria (Simonds *et al.*, 1996, p. 127).

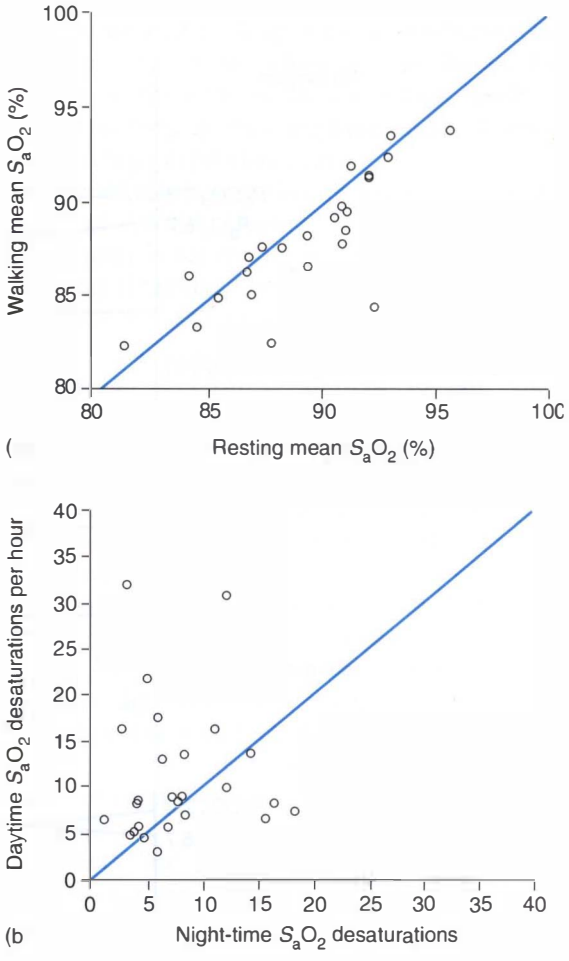


Figure 5.6 Comparison of oxygen needs of people with COPD during exercise and sleep. (a) Linear relationship between requirements during rest and exercise. (b) Non-linear relationship between requirements during rest and sleep. (Modified from Schenkel, S. (1996) Oxygen saturation during daily activities in COPD. *Eur. Resp. J.*, **9**, 2584–2589)

Patients are advised that oxygen should be used for as long as they can manage without unnecessary disruption to their lifestyle (Luce *et al.*, 1993). 'The more the merrier' is their maxim. The minimum effective prescription is for nocturnal oxygen therapy, but over 15 hours per 24 hours is preferable, and near-continuous oxygen is ideal (Hodgkin *et al.*, 2000, p. 135).

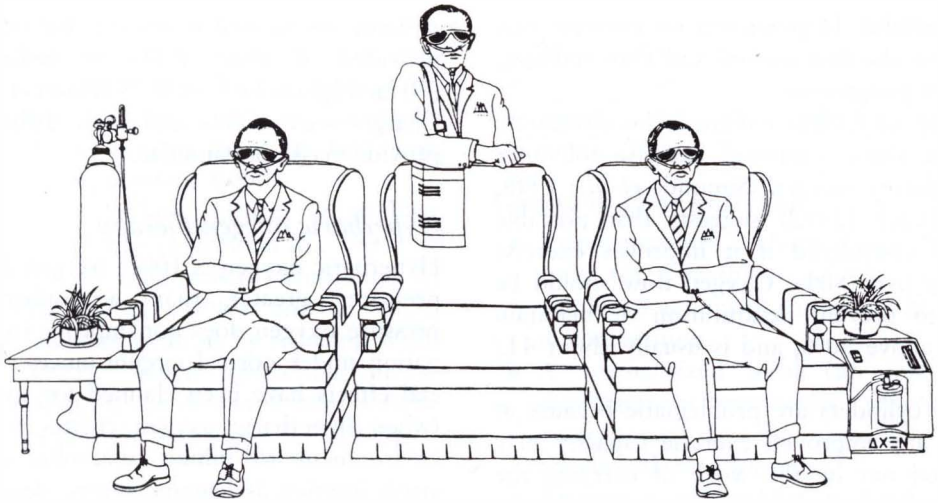


Figure 5.7 Home oxygen systems. From left: cylinder, portable liquid oxygen, concentrator. (From Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester, with permission.)

The goal is to achieve a P_aO_2 at least 8.7 kPa (65 mmHg) without a rise in P_aCO_2 by more than 1.3 kPa (10 mmHg). The flow rate for this is generally 1.5–2.5 L/min, which can be increased by 1–2 L/min during sleep or exercise if indicated. Ongoing patient support is mandatory.

Three systems are available (Figure 5.7):

- Oxygen cylinders contain compressed oxygen delivered through a regulator valve. They are cumbersome, require repeat prescriptions and regulator changes, are dangerous if not secured carefully, run out of oxygen rapidly, provide a limited pressure that is inadequate for driving a nebulizer and may not cope with long tubing. They deliver cold, dry oxygen.
- Oxygen concentrators separate ambient oxygen from nitrogen and are cheaper if more than four cylinders a week are needed (Dodd *et al.*, 1998). They are noisy, cannot be modified for portable use and do not have enough pressure to power a nebulizer unless two are used in parallel. The oxygen is at room temperature and humidity.
- Liquid oxygen is stored at nearly absolute zero in thermos containers. Advantages are that electricity is not required, and easy portability means that re-employment is more viable (Lock, 1992). Disadvantages are that it evaporates over time, is twice as expensive as a concentrator and is rarely available on the British NHS.

Nasal cannulae are useful for convenience, except for mouth-breathers and those with a heavy cold. They cannot be humidified and systemic hydration should be optimized, with a recommended fluid intake of about 2.5–3 litres over 24 hours (Heslop and Shannon, 1995).

Domiciliary oxygen means that the disease is visible and can no longer be denied. Veteran patients on long-term oxygen are often willing to talk to new patients and their carers, who may feel dismayed at the prospect of a life spent tethered to bulky equipment by the nose.

Portable oxygen

If oxygen is required at rest, it is needed on exercise, and sometimes during eating or lengthy talking (Sliwinski, 1994). Transient hypoxaemia sustained during routine activities is unlikely to be damaging, but prolonged or profound hypox-

aemia is harmful. Hypoxaemia on exercise may occur within the first minute and then stabilize, or it may be progressive.

A quarter of COPD patients who desaturate on exercise show improved exercise tolerance with ambulatory oxygen (Simonds *et al.*, 1996, p. 127). Leach (1992) suggests that portable oxygen be considered if it improves exercise capacity by over 50%. Oxygen flow should be sufficient to prevent desaturation or maintain saturation above 90%, and is usually about 4 L/min.

Portable cylinders are problematic because at low flows any improved exercise capacity may be cancelled out by the work of carrying the 2.3 kg cylinder, and they are best wheeled on a shopping trolley or custom-built walker. At high flows, the cylinder can empty before the patient has even settled into the car, especially as it has often not been completely filled because this needs a full F-size cylinder. Patients describe the anxiety of using portable cylinders as like driving with the fuel gauge on red, and adherence is therefore poor.

Duration of oxygen supply is increased by oxygen-conserving devices such as light-weight demand-valve cylinders and pulsed dose oxygen (Garrod *et al.*, 1999). Some facilities offer a loan or refilling service. Patients have to pay for portable cylinders in the UK, but semi-portable 300 L cylinders can be prescribed on the NHS (Table 5.2).

For air travel, commercial airline cabins contain the equivalent of 15% oxygen, so patients are advised to arrange for oxygen to be provided if their P_aO_2 is below 9.4 kPa (70 mmHg) on F_IO_2 of 0.15 (Dodd *et al.*, 1998). Charges vary widely and some airlines do not provide masks or cannulae.

Table 5.2 Characteristics of domiciliary and portable oxygen systems (adapted from Dodd *et al.*, 1998 and RCP, 1999)

Source	Capacity (L)	Flow (L/min)	Duration at 2L/min
'F' cylinder	1360	2 or 4	11 h
Concentrator	(continuous)	0.5–5	(continuous)
Liquid O_2:			
tank	25 800	0.25–10	8 days
portable	1032	0.25–10	8 h
PD 300	300	2 or 4	2.5 h
Portable	230	2 or 4	2 h

Hyperbaric oxygen therapy

Hyperbaric oxygen is 100% oxygen delivered at pressures greater than atmospheric. High-pressure oxygen does not improve tissue oxygenation under normal circumstances, but beneficial effects have been claimed for gas gangrene (when hyperbaric oxygen creates a high PO_2 environment to inhibit anaerobic organisms), crush injuries, ischaemia, burns, decompression illness, post-radiation damage, compromised skin grafts (Slotman, 1998) and severe cerebral air emboli after heart surgery (Dexter and Hindman, 1997).

Hyperbaric oxygen is no longer used for people suffering from carbon monoxide poisoning because it has not been proved to increase the transport of dissolved oxygen, and may be detrimental (Scheinkestel *et al.*, 1999).

Hyperbaric chambers can accommodate either one patient or a patient and attendants. Precautions in this high pressure environment are:

- awareness that pulse oximetry is inaccurate
- avoidance of glass vials and bottles
- for patients with an underwater chest drain, checking that fluid does not get sucked back into the pleural cavity
- for intubated patients, filling the tracheal tube cuff with liquid rather than air
- for patients on a ventilator, modification of pressures to protect against barotrauma.

Hyperbaric oxygen is contraindicated if there is an undrained pneumothorax (Pitkin, 1997).

Heliox therapy

Helium is an inert gas with one-eighth the density of nitrogen. When blended with oxygen it is called heliox, and a mixture with 21% oxygen is one-third as dense as air. This can more easily bypass obstructed airways.

Heliox is used for people with acute asthma, in whom it can reduce airflow resistance by 40% and increase peak flow by 35% (Manthous *et al.*, 1995). This buys time while awaiting the effects of medication, or it can be used for those refusing intubation (Austan, 1996).

Heliox can also relieve stridor, or swelling from tumours or burns (Marino *et al.*, 1995). It can be used with non-invasive ventilation in severe COPD (Jolliet, 1999) and a 70:30 mixture can facilitate weaning by reducing WOB (Harrison, 1995). When delivering heliox through a ventilator, the delivered tidal volume may be greater than that set (Lee *et al.*, 1999).

NUTRITION

Breathing and eating are basic life processes that are intimately related in their mechanics, emotive associations and physiology. Air and food share common pathways during ingestion, separate briefly for processing and then blend in the blood for distribution and the production of energy.

Despite this interdependence, nutrition is still a neglected area of respiratory medicine and the poor relation in medical and nursing undergraduate curricula. This 'skeleton in the hospital closet' was identified in 1974 (Edington *et al.*, 1997) but still only a third of UK hospitals have nutrition teams (Hindle *et al.*, 1996), and most cases of malnutrition are not recognized (Powell-Tuck, 1997). Malnutrition often reaches 'marasmic proportions' in COPD (Donner and Howard, 1992); for example, 47% of stable patients have shown nutritional abnormalities (Sahebjami, 1993) and this can exceed 50% in hospitalized patients (Fitting, 1992).

Physiotherapists treating malnourished patients are working uphill. The following facts are directly related to physiotherapy and will assist liaison with the health care team:

- malnourished people are unable to improve muscle function and exercise tolerance (Fitting, 1992)
- malnourishment impedes mobility (Powell-Tuck, 1997) and drains motivation (Powell-Tuck, 2000)
- well-nourished patients with stable COPD show no evidence of chronic fatigue (Similowski, 1991).

Causes of poor nutrition

- Eating becomes a chore rather than a pleasure for breathless people because the combined actions of eating and breathing are in competition.
- A normal-sized meal can interfere with diaphragmatic mechanics, especially when accompanied by the air-swallowing associated with breathlessness.
- Appetite is reduced by smoking, depression, the taste of sputum and some drugs.
- Exercise limitation and fatigue discourage the preparation of healthy food.
- Desaturation during meals can be caused by the breath-holding required for swallowing and the metabolic activity required for digestion and assimilation.
- Increased WOB raises calorie requirements. People with emphysema have shown a 25% increase in energy expenditure (Mowatt-Larssen, 1993).
- Salbutamol increases energy demand (Burdet, 1997).
- Oxygen therapy or mouth breathing can dry the mouth.
- Nutrition is impaired by hypoxaemia (Donahoe *et al.*, 1992), alcoholism (Bridges *et al.*, 1999) and being ill (Lennie, 1999).
- 'Hospital malnutrition' is exacerbated by unappetizing food, missed meals because of tests or procedures, the low priority given to nutritional support and, for patients on steroids, exacerbation of muscle wasting (Saudny, 1997). The nutrition of most patients deteriorates over an average 2-week admission (Powell-Tuck, 1997).

Effects of poor nutrition

Nutritional depletion impairs ciliary motility, aggravates the emphysematous process, erodes muscle (Bach and Haas, 1996), depletes surfac-

tant, increases WOB, impairs tissue elasticity (DeMeo, 1992), hinders fluid balance, can precipitate hypercapnic respiratory failure (Bach and Moldover, 1996), decreases exercise capacity (Palange *et al.*, 1998), causes depression and apathy (Powell-Tuck, 1997), increases infection risk and mortality (Powell-Tuck, 2000) and reinforces the whole unhappy process by blunting hunger.

Surprisingly, the diaphragm fails to enjoy preferential status, and both inspiratory and expiratory muscle strength are disproportionately reduced in malnutrition (DeMeo, 1992), as shown by decreased spirometry and cough pressures.

Management

Attention to nutrition should be a routine preventive measure for all people with COPD, and indeed for many others. This should not be left until debilitated patients have cannibalized the protein from their own respiratory muscles.

Education includes the following suggestions:

- Eat multiple small meals, preferably six times a day.
- If breakfast is difficult, try liquidizing it.
- Ensure adequate intake of vitamin E (Dow, 1996) and vitamin C, which help prevent infection, reduce inflammation and mop up oxidants in tobacco smoke (Sridhar, 1995).
- Make use of high energy drinks such as home-made milk shakes and fresh fruit juice.
- Take liquids separately from meals.
- Avoid hard or dry food, or add sauces such as gravy or custard.
- Avoid gas-forming foods.
- Clean teeth or use a mouthwash before meals if inhaled drugs or sputum have left a bad taste.
- Meals should be leisurely, enjoyable and taken sitting up with elbows on the table to stabilize the accessory muscles.

Patients can experiment with reducing or abstaining from:

- dairy foods, if secretions are a problem, because these can increase the viscosity of mucus (Enderby, 1995)
- caffeine, which potentiates peptic ulceration, to which people with COPD are susceptible (Bach and Haas, 1996, p. 253)
- additives and spicy food (Hodgkin *et al.*, 1993)
- alcohol, which increases pulmonary hypertension and hypercapnia in COPD (Jalleh *et al.*, 1993), encourages snoring in normal people and sleep apnoea in snorers (Chan, 1990), and, for everyone else, impairs ciliary action and immune function (Hodgkin *et al.*, 1993, p. 113).

Supplementary feeds provide concentrated nutrition orally or nasogastrically but can cause bloating, and pulmonary aspiration if a nasogastric tube is used. They have proved laborious in practice, especially in the home. They are sometimes successful for people with exacerbation of disease, and occasionally for those with advanced chronic disease in (Figure 5.8).

Oral feeds should be taken with a glass rather than through a straw to avoid excess WOB. Enteral feeds are best given at night to encourage daytime eating, and nasojejunal tubes improve tolerance (Whittaker, 1990). Slow

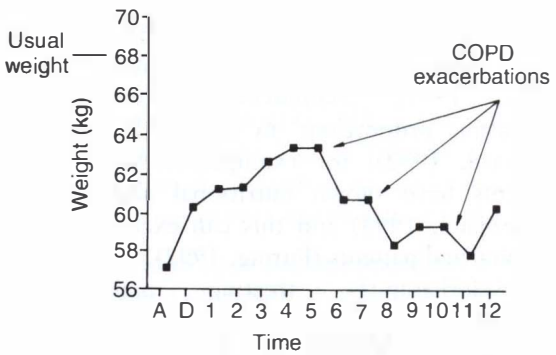


Figure 5.8 Weight of a patient with severe COPD, showing beneficial effects of nutritional support and detrimental effects of exacerbations. *A = admission; D = discharge.* (From Donahoe, M. and Rogers, R. M. (1990) Nutritional assessment and support in COPD. *Clinics in Chest Medicine*, 11, 487–504., with permission)

continuous infusion prevents excess metabolic activity which can lead to desaturation (Ryan *et al.*, 1993).

High-fat, low-carbohydrate formulae should be used for patients with a tendency for hypercapnia because the carbohydrate loading of normal supplementary feeds can increase CO_2 production, oxygen consumption and breathlessness for up to 1½ hours (Kuo *et al.*, 1993). It is not known if normal eating with high-carbohydrate food has a similar effect, and patients should not be discouraged from eating any food that they enjoy. However, it is possible that excess consumption of the 'empty calories' of high-sugar, high-additive drinks such as Lucozade may affect a respiratory patient who already retains CO_2.

Both obesity (p. 19) and malnutrition impair lung function (Chen *et al.*, 1993). Physiotherapists may be the first to identify the need for nutritional guidance and give basic advice, but a dietician is required for accurate assessment and treatment. Respiratory patients can show a complicated pattern of weight loss, fluid retention, obesity and masked malnourishment. Body water can be altered by oxygen therapy (Donahoe *et al.*, 1992), and body weight is not an adequate measuring tool.

For budget holders and bed managers, the fact that nutritional support can reduce the length of hospital stay by 5–16 days (Lennard-Jones, 1992) might give this aspect of patient care a higher priority.

> ***A hospital is a meaningless edifice if even one patient we are caring for has pain which is not eased, has a sleepless night, is given an unwholesome meal, is in unaesthetic surroundings or is treated without basic human dignity.***
>
> Khadra, 1998

DRUG THERAPY

Medication is normally prescribed by doctors and administered by nurses, but physiotherapists are involved in requesting and sometimes administering respiratory drugs. People with diseases such as COPD tend to be subjected to blind polypharmacy, even though effectiveness is limited in irreversible conditions. Physiotherapists need to discriminate and understand the indications, side effects and delivery systems of different drugs. Adherence rates are classically around 50%, especially with multiple dosing, but patients are responsive to education about drugs and devices (Wright, 1993), and it must be clarified in each workplace whether this is the role of the pharmacist, nurse or physiotherapist.

This chapter will use the generic name (e.g. salbutamol) or trade name (e.g. Ventolin) according to which is commonly used. Some British generic names will shortly become redundant as international terminology is to become universal. Table 5.3 clarifies the names used in the UK at present.

Definitions

- *Agonists* activate a receptor response.
- *Antagonists* block a receptor response.
- *'Half-life'* measures the rate of elimination of a drug by indicating the time for plasma levels to drop to 50%. A drug is said to be completely eliminated after about five half-lives (Baterman and McLay, 1999).
- *Drug metabolism* is determined by patient age, size and the drug's route of excretion. The very young and very old are slower to metabolize drugs. Drugs excreted by the kidney or metabolized by the liver are affected by kidney or liver failure.

Drugs to prevent inflammation

The mechanism of allergic asthma can be influenced by the chromones (Intal or Tilade), which inhibit the release of inflammatory mediators such as neutrophils and eosinophils from mast cells. They protect against allergic and exercise-induced asthma but do not reverse an established attack. They can only be inhaled, and are particularly effective in children, for whom they should be the first-line preventive medication (Korhonen *et al.*, 1999). They can be used prophylactically before exercise or allergen exposure (Spooner, 2000).

Table 5.3 Medication for airways obstruction (trade names in parentheses)

Drug	Delivery	Side effects
Chromones	Inhalation	
Sodium cromoglycate (Intal)		
Nedocromil sodium (Tilade)		
Corticosteroids	Inhalation	Hoarse voice (inhalation)
Beclomethasone (Becotide, Becloforte)		Oropharyngeal candidiasis (inhalation)
Budesonide (Pulmicort)		Osteoporosis
Fluticasone (Flixotide)		Fluid retention
Prednisone		Weight gain
Prednisolone	Intravenous/oral	Muscle atrophy
Hydrocortisone		Infection risk
		Peptic ulceration
		Fragile skin
		Bruising
		Hyperglycaemia
		Diabetes
		Hypertension
		Cataract
		Mood change
		Adrenal suppression
		Delayed healing
		Retarded growth
Bronchodilators	Inhalation	Tremor
β_2-agonists	Oral	Tachycardia
Salbutamol (Ventolin)	Intravenous	Agitation
Terbutaline (Bricanyl)	Subcutaneous	Atrial fibrillation
Slow-release β_2-agonists	Oral or inhalation	
Bambuterol		
Salmeterol (Serevent)		
Eformoterol (Foradil)		
Anticholinergics	Inhalation	Dry mouth
Ipratropium (Atrovent)		Constipation
Oxitropium (Oxivent)		Urine retention
		Glaucoma
Xanthines	Oral or intravenous	Headache
Theophylline		Gastric ulcer
Aminophylline		Insomnia
		Nausea and vomiting
		Arrhythmias
		Nasty taste

The only discernible side effect is a taste of burning tyres, which can be reduced by a mint-flavoured preparation. Because of their low toxicity, chromones should be tried before steroids but, although they are effective in 70% of asthmatics (Holgate, 1996), they take 4 weeks to show an effect in 33% of patients, and 12 weeks in 84% of patients (Corsico, 1993).

Drugs to treat inflammation

Corticosteroids are hormone-based agents that reduce the inflammatory reactions that set off bronchospasm, oedema and mucus hypersecretion. For accurate prescription, a home trial with serial peak flow measurements (Figure 3.10) is necessary because of spontaneous fluctuations in

airway calibre (Wiggins, 1991). Maximum benefit may not be felt by the patient for 2 months (Irwin *et al.*, 1998). Side effects are listed in Table 5.3 and details are explained below.

- Systemic side effects are reduced by using the inhaled route. Local side effects from inhalation can be minimized by using a spacer (p. 140), inhaling slowly and afterwards rinsing the mouth, gargling or cleaning the teeth. Toothbrushes should be renewed frequently. If using a mask, the face should be wiped afterwards.
- In children, high-dose inhaled steroids retard growth rate but not ultimate stature (McCowan *et al.*, 1998).
- Inhaled steroids can reduce bone density (Boulet *et al.*, 1999) and oral steroids can double the risk of hip fracture (Cooper, 1995). Patients at risk should have preventive treatment and should have their bone mineral density regularly monitored (Cowan, 1998). Doses of inhaled steroids below 1000 μm/day in adults and 400 μm/day in children show no significant effect on bones or growth (Efthimiou and Barnes, 1998).
- High-dose steroids upset sleep and mood, with 20–50% of patients suffering depression (Mitchell, 1998).
- Even at low doses, oral steroids can weaken systemic and respiratory muscles, which reduces exercise tolerance and can manifest as increased breathlessness. This may be ascribed mistakenly to deterioration of the disease and lead to increased steroid dosage (Decramer, 1994).

Concern about side effects tempts prescribers to nibble at the problem with low-dose therapy. Both undertreatment and side effects can be minimized by high dosage and early weaning (acute disease) or alternate-day dosing (chronic disease). To minimize adrenal suppression, a course of steroids lasting over 3 weeks should be phased out gradually. Patients and health workers are prone to 'steroid phobia', especially as patients may confuse corticosteroids with the anabolic steroids abused by some athletes.

Drugs to treat bronchospasm

Response to bronchodilators is usual in asthma, less frequent in chronic bronchitis and rare in emphysema. All acute patients on bronchodilators should use a peak flow chart until response is confirmed, and patients with chronic disease need a drug trial (Spence, 1991) with peak flow and symptom monitoring. These will identify reversible bronchospasm, defined as improvement in peak flow or FEV_1 by at least 15% or 150 mL (Dekker *et al.*, 1992). Measurements are taken 20 minutes after Ventolin and 30 minutes after Atrovent. A drug trial also pinpoints which drug, combination of drugs, dosage and route of administration are optimal.

Both sympathetic (adrenergic) and parasympathetic (cholinergic) receptors have been identified in bronchial smooth muscle. *Sympathomimetics* are versatile drugs that mimic the action of the sympathetic nervous system. Those which stimulate β_2-receptors in bronchial smooth muscle are known as β_2-stimulants, β_2-adrenergics or β_2-agonists. Examples are:

- Ventolin or Bricanyl: onset of action 7 min, peak effect 20 min, duration of action 3–5 hours
- Serevent: onset 15 min, peak effect 1 hour, duration 12 hours.

Short-acting drugs such as Ventolin should be taken symptomatically rather than regularly. Regular use should be confined to those with acute asthma, severe chronic asthma, or as prophylaxis before exercise-induced bronchospasm or allergen exposure. Most COPD patients do not show objective improvement with β_2-stimulants but some find symptomatic relief, either as placebo or by reducing hyperinflation (Gibson, 1996).

Long-acting β_2-stimulants such as Serevent are not for use as symptomatic or rescue medication. These slow-release bronchodilators are prescribed regularly for controlling nocturnal asthma and some of the effects of brittle asthma.

Disadvantages of regular β_2-stimulants are that they can reduce sensitivity to Ventolin itself (Giannini and Bacci, 1999), may counterbalance the benefits of steroids (Taylor, 2000) and, for people with arrhythmias and hypoxaemia, they can have an adverse effect on the myocardium (Cazzola, 1998). In severe COPD, the side effect of trembling hands can impair ADL, and extra energy expenditure due to a thermogenic effect can be significant in malnourished people (Burdet, 1997). β_2-stimulants tend to be prescribed freely, and patients may overuse them if not educated adequately. Unnecessary and frequent use can worsen the course of COPD (Postma, 1991) and asthma (Barrett, 1995).

Anticholinergic (antimuscarinic) bronchodilators, such as Atrovent, block the effect of acetylcholine on autonomic nerve endings. They primarily affect the larger airways, have a slow onset of 30–45 minutes and are most effective in infants and older people. They take second place as bronchodilators in asthma unless the side effects of β_2-stimulants are troublesome, but they may be more effective in COPD (Allen, 1996). They can be used sequentially or in combination with β_2-stimulants because of their additive effect (Dorinsky *et al.*, 1999), e.g. Combivent. Individuals show different response patterns and may react better to β_2-stimulants, anticholinergics or both. There is an association between glaucoma and nebulized Atrovent, so a mouthpiece should be used and eyes should be kept shut. In contrast to β_2-stimulants, the recommended dose of two puffs is often suboptimal and some doctors are happy for patients to double or triple their intake.

Theophylline and its derivatives, such as aminophylline, are part of the xanthine group of drugs, which have an interesting variety of effects. They appear to bronchodilate, promote gas exchange, reduce breathlessness and improve exercise capacity (Cahalin and Sadowsky, 1995), reduce cough and sleep disturbance (Luce, 1996), promote mucociliary clearance and reduce pulmonary hypertension (Banner, 1994). Their anti-asthma properties are also related to reducing inflammation and boosting the immune system, and these effects can be achieved at lower dosage than required for bronchodilation, thereby reducing the side effects that have hitherto limited their use (Barnes and Pauweis, 1994). If given over months, steroid dosage can sometimes be reduced.

The theophyllines are too insoluble to be given by inhalation, and precise dosage is required because of their narrow therapeutic window. Slow-release preparations are used to control nocturnal asthma, and continuous intravenous therapy can be used for brittle asthma. Clearance rates are increased in smokers and children, thus lowering blood levels, and decreased in elderly people and those with viral infection or heart failure.

If both β_2-stimulants and anticholinergics are prescribed, the anticholinergic is taken first to open up the large airways and provide better access for the β_2-stimulant (Mathewson, 1993). If bronchodilator and preventive drug (chromones or steroids) are prescribed, the bronchodilator should be taken first to ensure maximum penetration of the preventive drug. However, complicated instructions can demotivate patients and it is more important that the drugs are taken than the exact sequence is adhered to.

Drugs to treat breathlessness

Breathlessness in chronic lung disease becomes significant to the patient when roughly half the ventilatory capacity is lost. Little of this function is recoverable, which often leads to an attitude of therapeutic defeatism.

Sometimes the cause of breathlessness can be treated, e.g. by diuretics, bronchodilators or steroids. For a direct effect on breathlessness, the options are limited. *Bronchodilators* may reduce breathlessness in some patients independent of their bronchodilating effect, possibly by improving the efficiency of the diaphragm (Hatipoglu *et al.*, 1999). *Morphine* increases the exercise tolerance that is limited by breathlessness (Young, 1989), reduces respiratory drive and oxygen consumption, lessens anxiety and, in a proportion of patients, reduces breathlessness

directly. The risk of morphine-induced respiratory depression is reduced by a slow-release preparation and titration to the individual's need (Light, 1989). *Dihydrocodeine* has shown a 20% reduction in exercise breathlessness (Burdon, 1994) but constipation is a problem.

Buspirone is particularly useful because it is an anxiolytic which reduces breathlessness and increases exercise tolerance without sedative effect (Argyropoulou, 1993). Inhaled *lignocaine* ameliorates breathlessness associated with bronchoconstriction (Manning, 1995), but patients should not eat or drink for an hour afterwards.

Drugs to treat infection

An antibiotic is indicated if a patient's condition is caused by bacterial infection and if the organism responsible is sensitive to the antibiotic prescribed. Antibiotics often have to be given blind at first because 24 hours are needed for microbiological results. Antibiotics show only a small benefit in exacerbations of COPD, but those most likely to benefit show all three of the following symptoms:

- ↑ dyspnoea
- ↑ sputum volume
- ↑ sputum purulence (Smith *et al.*, 1999).

Antibiotics should be specific and time-limited. Prophylactic use is reserved for people with chronic sepsis such as cystic fibrosis. Unnecessary antibiotics simply select for resistant bacteria (Man *et al.*, 2000), leading to superinfection. Despite 65% of respiratory tract infections being viral, 75% of patients are prescribed antibiotics (Carroll, 1989).

Antibiotics are administered orally, intravenously or by nebulizers with powerful compressors designed for viscous solutions and which have an exhalation filter. Side effects of antibiotics include skin reactions and the emergence of resistant organisms, partly because of interference with the friendly flora of the respiratory tract.

Bacteria are becoming resistant to antibiotics faster than new drugs are invented because of overuse globally, mis-selling to the developing world, their use as growth promoters in agriculture and the virtual shrouding of hospitals in an antibiotic blanket. Those who anticipate a post-antibiotic era are no longer considered doom-mongers, and Amyes (1995) has predicted that we will run out of effective antibiotics by 2020.

There is increasing interest in Chinese herbal medicines, which show benefit in some lower respiratory tract infections (Liu and Douglas, 1998), and serious consideration is now given to maggot therapy for antibiotic-resistant wound infections (Thomas *et al.*, 1999).

Drugs to help clear secretions

Primary agents to clear secretions are systemic hydration, effective humidification or drinking a steamy cup of tea. If drugs are needed, they should be aimed at improving mucus transport rather than irritating the airways so that more secretions are created.

Mucus transport may be enhanced by *vitamin C* (Silver and Foster, 1990), dry-powder *mannitol* (Daviskas, 1997) and certain bronchodilators such as *bamiphylline* (Todisco, 1995) and *Serevent* (Rusznak, 1991). The volume of sputum expectorated can be reduced by inhaled *oxitropium* (Tamaoki, 1994), inhaled *indomethacin* (Tamaoki, 1992) or inhaled *steroids* (Elborn *et al.*, 1992).

Cilia are made to transport a viscoelastic gel. They have difficulty propelling either liquids or thick mucus. Acetylcysteine is a mucolytic that can reduce the viscosity of thick mucus but at the expense of risking bronchospasm (Eng, 1998), so it needs to be accompanied by a bronchodilator. There is no evidence that it offers any advantage over humidification or saline instillation, and it is not available on the NHS except for palliation and sometimes for people with cystic fibrosis. If used, it can be nebulized or 1–2 mL can be instilled down a tracheal tube (AHFS, 1999).

Surfactant has mucokinetic properties and is showing some promise in aiding mucociliary clearance (Wills and Cole, 1996). The surface tension of mucus can also be reduced by

iodinated glycerol (Petty, 1990). Drugs such as rhDNase for CF are described on page 89.

Drugs to inhibit coughing

To suppress a non-productive and irritating cough, medication is available if physical means (p. 204) are to no avail. Antitussive agents include *baclofen* (Dicpinigaitis, 1998), *pholcodine* and *dextromethorphan* (Parvez *et al.*, 1996). Nebulized *local anaesthetics* block sensory nerve traffic and provide temporary relief at night (Trochtenberg, 1994). A cough caused by asthma or ACE-inhibitors can be reduced by *Tilade* (Hargreaves, 1995), and a post-infection cough by *Atrovent* (Holmes *et al.*, 1992).

Non-specific cough suppressants are best avoided (Irwin *et al.*, 1998). 'Cough mixtures' may contain both expectorant and suppressant but are strong placebos and the sugar content is thought to briefly soothe irritated airways.

Drugs to improve ventilation

Respiratory stimulants should be used with caution if the respiratory muscles are already working maximally, because further stimulation will override the protective function of fatigue (Barnes, 1995). Respiratory stimulants do not reverse the underlying cause of ventilatory failure.

Doxapram drives ventilation via chemoreceptors and the respiratory centre but at the cost of central nervous system stimulation, agitation (Simonds 1996, p. 170), hallucinations, increased WOB, breathlessness and sometimes panic attacks (Abelson, 1996). An infusion is sometimes tolerated by drowsy patients with post-anaesthesia hypoventilation or to avoid the need for mechanical ventilation (Hirschberg, 1994). Doxapram is widely used for ventilatory failure, especially with acute oxygen therapy for COPD patients who cannot reach satisfactory oxygen saturations without excessive hypercapnia. This practice is not evidence-based and both Gribbin (1993) and Angus (1996) advise against it except as a temporary holding measure. If non-invasive ventilation is not available to rest the inspiratory muscles, the *theophyllines* may improve diaphragm contractility (Roussos, 1996). Other pharmacological possibilities are reviewed by Heijden *et al.* (1996).

Drugs to relieve oedema

Salt and water excretion can be promoted by *diuretics*, either loop diuretics such as frusemide or osmotic diuretics such as mannitol. They are potent and non-selective, and over-enthusiastic use can lead to volume depletion, dizziness on standing, loss of calcium and potassium, anorexia and vomiting (Weill *et al.*, 1998). Diuretics are the commonest cause of adverse drug reactions in old age (Rhodes, 1993).

Diuretics are useful to reduce cardiac workload in pulmonary oedema associated with chronic heart failure but they do not prevent progression of the disease (Cleland, 1996) nor affect survival (Krämer *et al.*, 1999) and the associated activation of the sympathetic nervous system can exacerbate tachycardia and vasoconstriction while hindering stroke volume and tissue perfusion (Northridge, 1996). Diuretics are not advised for acute heart failure (Northridge, 1996) or oedema associated with immobility, venous insufficiency or premenstruation (Rhodes, 1993). Some patients taking diuretics restrict their fluid intake, especially if they have stress incontinence or poor mobility. However, diuretics can usually be taken at a time of day to minimize disruption from the obligatory trips to the toilet (Gordon & Child, 2000).

Drugs specifically for asthma are discussed on page 81, for CF on page 89, for primary heart disorders on page 333 and for palliative care on page 311.

Delivery devices

Are respiratory drugs best ingested or inhaled? Inhaled therapy delivers an aerosolized drug, suspended as fine liquid or solid particles in air, directly to the respiratory tract. It brings the following advantages:

- rapid onset of action
- local delivery to maximize the positive effects and minimize adverse effects

- delivery of drugs that are not active by other routes (Manthous, 1994).

Disadvantages are:

- less effective lung deposition with increased airflow obstruction, leading to patchy and less peripheral distribution in people with advanced COPD or acute severe asthma (Lipworth, 1995)
- reduced effectiveness in breathless people whose rapid airflow favours deposition to the central airways
- loss of much of the drug to the atmosphere, stomach and pharynx, although high doses compensate for this.

Large particles more than 5 μm diameter are lost in the upper airways. Small particles less than 2 μm are deposited in the alveoli and are used for antibiotic delivery. Tiny particles less than 0.5 μm are inhaled and exhaled untouched (Figure 5.9).

Particles from 2–5 μm target the bronchi and bronchioles and are used for bronchodilators and steroids (Manthous, 1994). The task of assessment for delivery systems may fall to the physiotherapist.

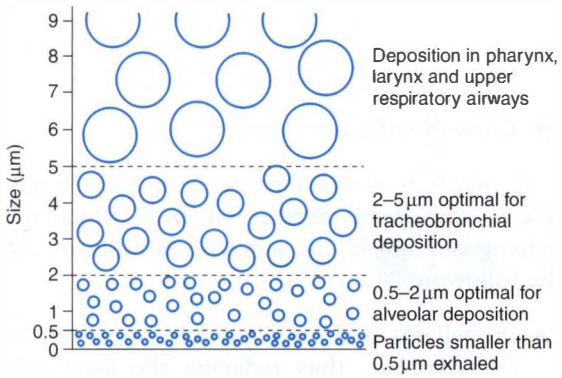


Figure 5.9 Aerosol deposition at varying particle sizes (Intersurgical, with permission).

Inhalers

Pressurized inhalers deliver an aerosol by suspending an active drug in a propellant. The traditional metered dose inhaler is portable and cheap but the device will shortly be outlawed because of its ozone-unfriendly propellant. Poor inhaler technique is common; 60% of COPD patients use them incorrectly (Reina-Rosenbaum *et al.*, 1997). Other inhalers such as the breath-actuated Autohaler or Easi-Breathe (Figure 5.10) co-ordinate drug release with inhalation.

Slow inhalation and end-inspiratory pause are advised with pressurized inhalers (Pedersen, 1996). Details of technique are available from pharmacists and supplied with the inhaler. Instructions for the Easi-Breathe are as follows:

- Shake inhaler.
- Fold down cap from over mouthpiece.
- Hold inhaler upright.
- Breathe out.
- Place mouthpiece in mouth, close lips firmly.
- Ensure fingers are not blocking airholes.
- Inhale slowly and deeply through mouthpiece, continue as inhaler puffs dose into mouth, continue until end of deep breath.
- Take inhaler out of mouth, hold breath for 10 seconds or as long as comfortable.
- Breathe out slowly.
- Close cap.
- If taking a second puff, wait one minute to re-prime inhaler.

Dry powder inhalers draw air through dry powder to create an aerosol, which is released on inspiration. They require minimal co-ordination and no breath-hold. Disadvantages are that children under 6 years, breathless people and those with bronchospasm may not be able to release the drug if they are unable to generate the required flow, i.e. 30–120 L/min (Dhand and Fink, 1999). The powder is sensitive to moisture, so it is either stored in foil blisters or patients advised not to exhale into the device. Devices have their own characteristics; for example, the Turbohaler is efficient but requires a forceful inspiration and does not indicate when

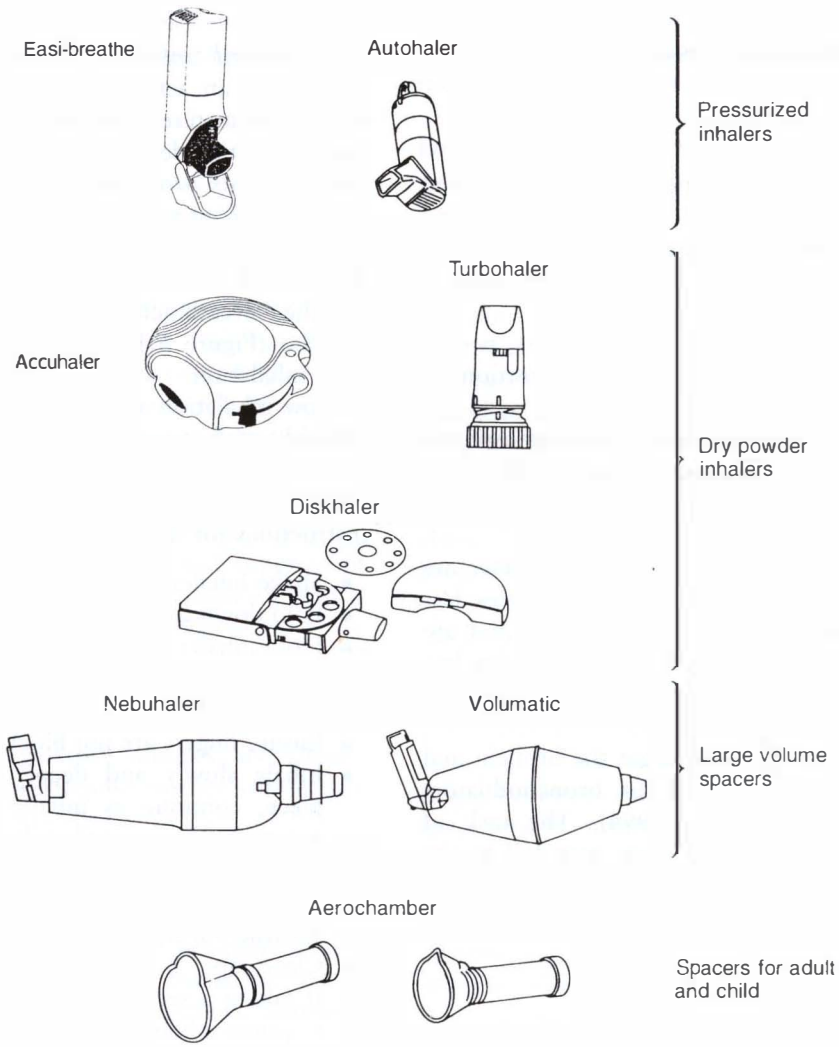


Figure 5.10 Inhaler devices.

the dose has been delivered. Instructions for the Clickhaler are as follows:

- Shake inhaler.
- Remove mouthpiece cover.
- Hold upright.
- Press button until click heard.
- Breathe out.
- Close lips around mouthpiece.
- Inhale deeply.
- Remove device from lips, close lips.
- Hold breath for 10 seconds.
- Close mouthpiece cover.

A *spacer* is a chamber between patient and inhaler that forms a reservoir from which the patient can inhale the aerosol. Advantages are the following:

- Propellants and large particles drop out in the chamber, thus reducing the local side effects of steroids (Everard *et al.*, 1992).
- Aerosol momentum is slowed so that less is lost by impaction on the back of the throat.

- Less co-ordination is required because the drug remains suspended in the spacer until the patient breaths in, although early inhalation is advised because the half-life of drug aerosol within the spacer is often less than 10 seconds (Thorax, 1997).
- High doses can be delivered during acute episodes.

Spacers should always be used for children taking steroids (Barry, 1994). Infants can use a soft face mask attached to the spacer (Everard *et al.*, 1992). The large pear-shaped spacers such as the Nebuhaler or Volumatic are cumbersome but most efficient (Barry, 1996), simulating the aerosol cloud from an inhaler. Slow quiet tidal breathing is advised (Pedersen, 1996). One dose at a time should be fired into the spacer because multiple dosing is inefficient (Pedersen, 1996). Large spacers should be washed with detergent once a week, without rinsing, and then air-dried, in order to reduce static charge, which attracts the drug to the walls of the spacer rather than the lungs (Piérart *et al.*, 1999).

Problems for patients with inhalers are:

- the need for co-ordination and/or manual dexterity
- confusion about when to use which inhaler, e.g. mistaken use of steroids on a symptom-related basis
- difficulty in understanding instructions, e.g. mistaken spraying of the drug up the nose, on to the chest or into the armpits!

Small-volume nebulizers

A jet nebulizer uses the Venturi principle to transform a drug in solution into a mist of droplets. A high-pressure system such as oxygen from a wall supply or air from a compressor forces the gas at high velocity through a narrow hole known as a Venturi. This creates an area of low pressure, which draws the drug solution into the fast-moving gas stream and produces a flow of droplets. Large droplets are impacted on a baffle and fall back into the reservoir (Figure 5.11).

Technique

- If the patient does not need oxygen, an air compressor is advisable as the driving force because the relative humidity of ambient air stabilizes particle size (Hodgkin *et al.*, 2000, p. 123).
- Select a mouthpiece if possible, unless patient preference or excessive breathlessness

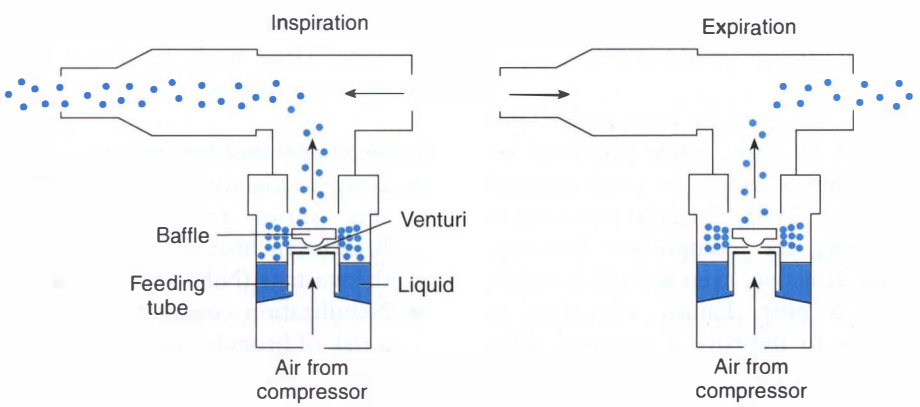


Figure 5.11 Small-volume jet nebulizer for delivery of saline or aerosolized drugs in droplet form. (From O'Callaghan, C. and Barry, P. W. (1997) The science of nebulised drug delivery. *Thorax*, suppl. 2, S31–S44, with permission.)

precludes this. Nose breathing filters the drug and reduces lung deposition by a quarter (Salmon *et al.*, 1990) and aerosol escaping from a mask can affect the eyes.

- If possible, have the patient sitting upright in a chair or side-lying to maximize basal deposition. Some nebulizers do not function when angled. Ensure breathless patients have support for their elbows.
- Fill to between 2.5 and 6 mL, depending on the nebulizer. Dilute with normal saline if required. Set the flow rate to 8 L/min, unless a compressor is used, which has a preset flow rate.
- Advise the patient:
  - to mouth-breathe if possible
  - to intersperse tidal breaths with some deep breaths and some end-inspiratory holds to improve deposition (Hess, 1994)
  - if using a mouthpiece, not to obstruct the excess air port
  - to allow 10 minutes for completion
  - after each use to empty and dry the nebulizer with a paper towel rather than a tissue, which can clog the system (or when in hospital, or follow infection control protocol)
  - once a day to wash in hot soapy water, rinse and dry with air from the compressor or oxygen supply, or follow hospital protocol.

    Drying is the most important aspect of the cleaning process (Dodd (1996).

For domiciliary use, patients must understand the importance of cleaning, since one-third of nebulizers have been found to be contaminated with bacteria (Jones, 1985). Regular servicing is required, otherwise the compressor becomes progressively less effective, even though continuing to produce a mist. Family education is advisable in order to improve a reported 44% adherence rate (Cochrane, 1997) and to ensure that patients do not interpret '4-hourly nebs' as requiring them to interrupt an already disrupted night.

Some nebulizer solutions should not be mixed, and the hospital pharmacy can be consulted for up-to-date information. Tapping the nebulizer when the liquid is beginning to fizz increases the delivered dose by 38% (Everard *et al.*, 1994); this is unnecessary with the high doses used for bronchodilation and simply extends treatment time, but is useful for exact drugs such as antibiotics. Demand nebulizers increase efficiency by delivering the drug on inspiration only. Ultrasonic nebulizers (p. 188) are popular with patients because the density of the mist facilitates more rapid completion of the process, but they do not suit all drug suspensions, e.g. budesonide or antibiotics.

For patients who remain symptomatic despite inhaler use, a 3-week home nebulizer trial is more accurate than laboratory measurements (Hosker, 1995). A typical home trial comprises nebulized saline, nebulized Ventolin and then a nebulized Ventolin/Atrovent mixture 6-hourly, each for 1 week. Symptoms and twice-daily peak flows are monitored and a positive result is defined as producing a 15% increase in peak flow during a week on active treatment compared to the week on saline (Goldman *et al.*, 1992). The first dose is best administered in hospital in case of side effects such as cardiac arrhythmias. Trials can also compare bronchodilators with steroids and nebulizers with inhalers. If patients use nebulizers at home, they must be given adequate assessment, advice (BTS, 1997) and back-up servicing. Drug trials for home use are invalid during acute illness.

Disadvantages and precautions.

- Drug availability between different nebulizers varies fourfold (MacNeish, 1997). Breath-synchronized devices reduce some of the wastage (Nikander, 2000).
- Nebulization cools the inspired gas, causing a risk of bronchospasm in some patients.
- Angina or hypoxaemia occasionally occurs with nebulized Ventolin (Simpson, 1993).
- 'Horrifying tales' of bacterial contamination and inadequate servicing have been reported

with domiciliary nebulizers (Lane, 1991). For hospital nebulizers, Botman (1987) found one-third to be contaminated.
- Patient adherence may be hindered by the long time required to complete nebulization.
- People with severe acute asthma may be over-reliant on repeated use when their airways are dangerously obstructed and nebulization is ineffective, with possibly lethal results (Lane, 1991).
- The inspired gas must be suited to the patient. Acutely hypoxaemic asthmatic patients require high F_IO_2 levels and acute hypercapnic COPD patients may require air, sometimes with oxygen via a nasal cannula.

Indications

The following may be suited to nebulizers:

- people who are too breathless to use an inhaler, such as during an asthma attack, so long as over-reliance does not prevent medical assistance being sought when indicated
- people who need large doses rapidly
- antibiotic, antifungal and local anaesthetic drug delivery
- patients in whom assessment has shown improved outcome compared to inhalers.

Inhaler or nebulizer?

Patients love nebulizers. They prefer them to inhalers even when they contain placebo saline (Jenkins *et al.*, 1987), perhaps because patients are automatically changed to nebulized drug delivery when admitted to hospital so nebulizers 'must be best', or perhaps because nebulizers create an impressive mist and do not demand respiratory gymnastics for co-ordination. However, less drug reaches the lung by nebulizer and ten times the dose needs to be prescribed. According to Hess (1994), nebulizers are less efficient, less convenient and twice as expensive as inhalers. According to Sahn and Heffner (1994, p. 162), their inefficiency can reach 'staggering proportions', with only 3–9% reaching β_2-receptors. Even so, individual assessment is required.

BRONCHOSCOPY AND LAVAGE

Access to the bronchial tree for diagnostic or therapeutic purposes is achieved with a fibreoptic bronchoscope, a thin flexible instrument passed through the nose and into the subsegmental bronchi, using local, nebulized or general anaesthesia.

Diagnostically, bronchoscopy can be used for:

- observation
- biopsy, e.g. to identify malignant lesions
- brushings, e.g. to obtain lower airway microbiology samples in patients with pneumonia or exacerbations of COPD, or to identify parenchymal lung disease, using a protected specimen brush to prevent contamination by upper airway flora (Wilson, 1999)
- washings or lavage.

Bronchoalveolar lavage involves wedging the bronchoscope into a bronchus, washing 120–200 mL of saline through it, then aspirating this along with fluid and cells from the lower respiratory tract and alveoli for diagnosing parenchymal lung disease. Complications include hypoxaemia and inflammation, which are lessened by using less saline and a telescopic or standard catheter rather than a bronchoscope (Fabregas, 1996).

Therapeutically, bronchoscopy can be used for removing foreign bodies, placing stents (Cowling, 2000) and laser-resecting tumours (Todisco, 1996). Bronchoscopy is rarely justified as a substitute for physiotherapy in clearing secretions (Brooks-Brunn, 1995) and can be complicated by bronchospasm, haemorrhage, pneumothorax, discomfort, subcutaneous emphysema, arrhythmias or the transport of microorganisms from the upper airway to the sterile lower airways. However, bronchoscopy may be appropriate if there is intractable sputum retention with no air bronchogram on X-ray, i.e. with blocked central airways. To re-

expand atelectatic areas, it can be combined with selective insufflation of air (Brooks-Brunn, 1995) or followed by physiotherapy.

Rigid bronchoscopy can be used for removal of large foreign bodies. This requires a general anaesthetic and does not have the flexibility of fibreoptic bronchoscopy, which allows rotation in every direction. Virtual bronchoscopy can be used for patients at high risk of complications (Haponik *et al.*, 1999).

Patients are usually told the 'why' of the procedure, but not always the 'how'. Physiotherapists can check that patients understand to procedure to avoid disproportionate fear (Poi *et al.*, 1998).

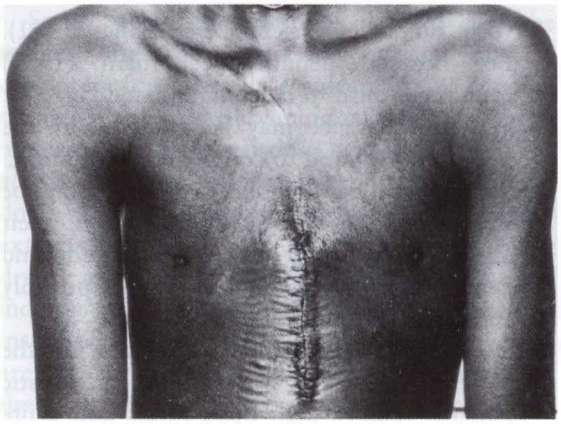


Figure 5.12a Mr FJ.

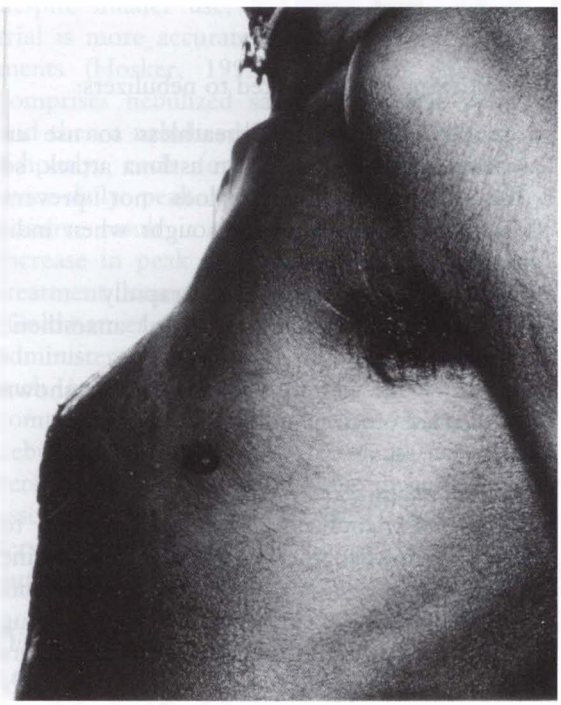


Figure 5.12b Mr FJ.

MINI CASE STUDY: MR FJ

How would you treat this 32-year-old father who has been referred for twice-weekly percussion and postural drainage? He has polychondritis (chronic inflammation of the cartilage), which has led to collapse of his tracheal and bronchial cartilages.

Background

HPC: surgery on deformed chest and formation of tracheostomy 15 years ago, discharged with instructions to change and clean tracheostomy tube twice-weekly.
Drugs: prednisolone.
SH: lives with wife and three children, started office job 2 months ago, non-smoker.

Subjective

Occasional chest infections, last one 6 weeks ago which never quite resolved.
Always have a bit of phlegm, usually no problem clearing it but slightly more difficult over the last 6 weeks.
Change trachy tube 9-monthly.
Slight SOBOE but not bothersome.
Trachy sometimes causes a dry cough.

Objective

Abnormal chest shape (Figure 5.12).
RR normal.

Breathing pattern slightly laboured.
Clinically well hydrated.
Posture: round shoulders.
Auscultation: scattered crackles.

Questions

1. Why might Mr FJ have chest infections?
2. What could have prevented the last chest infection from being fully resolved?
3. What could be causing the slight difficulty in clearing his chest?
4. Is the SOBOE a problem?
5. Is the dry cough a problem?
6. Analysis?
7. Problems?
8. Goals?
9. Plan?

RR = respiratory rate; SH = social history; SOBOE = shortness of breath on exertion.

RESPONSE TO MINI CASE STUDY

1. Tracheostomy bypassing upper airway defences, infrequent tube changes.
2. and 3.
   Change in lifestyle 2 months ago: inactivity, dry office atmosphere, possibly smokers at work.
4. It is not bothersome at present but may become so as Mr FJ ages and loses respiratory reserve.
5. It depends on whether it worries the patient.
6. No major disruption to lifestyle at present but potential for deterioration over time. Percussion unsuitable because of long-term steroids. Postural drainage unlikely to suit patient's lifestyle.
7. Risk of chest infections.
   Some sputum retention.
   Mild increase in work of breathing, probably because of chest shape. Potential for reduced exercise tolerance.
8. Reduce risk of infection.
   Develop exercise tolerance.
9.

- Negotiate more frequent tube changes. Twice-weekly changes are unlikely to be adhered to and are probably not necessary at home (which is safer than the infection-prone hospital atmosphere). Advise Mr JF that a clean tube might reduce his dry cough.
- Negotiate lifetime programme to prevent chest infections and maintain exercise tolerance, preferably based on enjoyable exercise, e.g. football with children, but offer variety of techniques for mucus clearance (Chapter 8).
- Discuss possible effects of office atmosphere.
- Assess whether postural correction is possible.
- Advise patient to ask GP's advice on bone mineral density scan to check for steroid-induced osteoporosis.
- Review 6-monthly by phone and yearly face to face.

LITERATURE APPRAISAL

The following study investigated chest physiotherapy (CPT) for people with pneumonia, as identified by infiltrates on X-ray. Does the physiotherapy fit the pathophysiology? And what do you make of the complications of CPT?

> ***Inclusion criteria for group 1 comprised the presence of unilateral pneumonic infiltrates on chest X-ray ... we found no statistically significant short-term therapeutic benefit from percussion or vibration.***
> ***CPT can cause ... barotrauma, bended endotracheal or ventilation tubing.***

Acta Anaesthesiol. Belg. 1991; 42: 165–170.

RESPONSE TO LITERATURE APPRAISAL

A passing acquaintance with the pathophysiology of pneumonia would have saved the researchers the trouble of beginning the study. Physical treatment cannot influence a lung that is solid with inflammatory consolidation.

Imagination boggles at the image of CPT causing barotrauma and bending an endotracheal tube....

RECOMMENDED READING

Baterman, D. and McLay, J. S. (1999) Clinical pharmacology: the basics. *Medicine*, 27(3), 1–4.

British Thoracic Society (1997) Guidelines for nebulizers. *Thorax*, 52(suppl. 2), S4–S16.

Cahalin, L. P. and Sadowsky, H. S. (1995) Pulmonary medications. *Phys. Ther.*, 75, 397–414.

Dodd, M. E., Kellet, F., and Davis, A. (2000) Audit of oxygen prescribing before and after the introduction of a prescription chart. *Br. Med. J.*, 321, 864–865.

Emad, A. (1997) Bronchoalveolar lavage. *Respir. Care*, 42, 765–780.

Heslop, K. and Harkawat, R. (2000) Nebulizer therapy from a practical perspective. *Eur. Respir. J.*, 10, 213–215.

Hess, D. R. (2000) Nebulizers: principles and performance. *Respir. Care*, 45, 609–622.

Hobbs, J. (1995) On patients and inhalers. *Respir. Dis. Pract.*, 12(1), 8–12.

Kacmarek, R. M. (2000) Delivery systems for long-term oxygen therapy. *Eur. Respir. Care*, 45, 84–92.

Knoch, M. and Sommer, E. (2000) Jet mobilizer design and function. *Eur. Respir. Care*, 45, 95–103.

Niederman, M. S. (1998) Update in pulmonary medicine. *Ann. Intern. Med.*, 128, 208–215.

Paton, J. (1994) The safety of inhaled steroids in childhood asthma. *Practitioner*, 238, 322–324.

Pedersen, S. (1996) Inhalers and nebulizers: which to choose and why. *Respir. Med.*, 90, 69–77.

Powell-Tuck, J. (2000) Nutrition. *Best Pract. Res. Clin. Anaesth.*, 13, 411–425.

Schols, A. M. W. J. (2000) Nutrition in chronic obstructure pulmonary disease. *Curr. Opin. Pulm. Med.*, 6, 110–115.

Spring, C. and McCrirrick, A. (1999) The fibreoptic bronchoscope in the ICU. *Br. J. Intens. Care*, 9(1), 14–22.

6 Physiotherapy to increase lung volume

Summary

Introduction to repiratory physiotherapy
What is loss of lung volume, and does it matter?
Controlled mobilization
Positioning
Breathing exercises
- **Deep breathing**
- **End-inspiratory hold**
- **Abdominal breathing**
- **Sniff**
- **Neurophysiological facilitation**
- **Rib springing**

Mechanical aids to increase lung volume
- **Incentive spirometry**
- **Continuous positive airway pressure**
- **Intermittent positive pressure breathing**

Outcomes
Mini case study
Literature appraisal
Recommended reading

Introduction to respiratory physiotherapy

What is respiratory physiotherapy? And does it work?

Respiratory physiotherapy, to be effective, includes education, pain relief, accurately targeted mobilization, manual and mechanical techniques, and response to patients in distress. It is ineffective to intervene with a process as personal as breathing without attention to the person as a whole.

Other aids to effectiveness are to avoid the routine and to ensure that any improvement achieved is maintained. Ongoing management includes a negotiated plan of self-care and liaison with nursing staff or relatives. Brief follow-up checks during the day may be appropriate, rather than ticking off the patient's name in a notebook. One of a physiotherapist's most useful skills is in motivating patients, especially by providing positive feedback and encouraging patients' own ideas (Kerr, 1999).

A suggested approach is to:

- assess the patient
- identify problems
- clarify the patient's expectations
- negotiate goals
- agree on a management plan and time frame
- treat the patient
- re-assess
- discuss and modify the plan according to ongoing assessment
- check if goals are met.

Goals should be specific, meaningful to the patient and challenging but achievable.

Patients who have difficulty communicating or who are on a ventilator can still be involved with decisions on treatment. For helpless patients, a degree of autonomy is particularly important. Physiotherapy usually includes advice, which should be explicit, short, clear, written down and copied for the physiotherapy notes.

The next three chapters will relate techniques to the available evidence, using the three main respiratory problems of reduced lung volume, increased work of breathing and sputum retention.

What is loss of lung volume, and does it matter?

Loss of lung volume takes a variety of forms.

- *Atelectasis* is collapse of anything from a few

alveoli to the whole lung. Segmental, lobar and lung collapse are visible radiographically, but microatelectasis is not obviously detectable. Causes include shallow breathing, bronchial obstruction, absorption of trapped gas, surfactant depletion and compression from abdominal distension or pleural disorder. Atelectasis has been reported in 74% of patients with acute spinal cord injury, 85% with neuromuscular disease, up to 90% of patients after cardiac surgery and 25% of patients after upper abdominal surgery (Raoof *et al.*, 1999). Physiotherapy is indicated to treat or prevent atelectasis if it is caused or anticipated by immobility, poor positioning, mucous plug, shallow breathing and/or postoperative pain, especially in non-alert patients.

- *Consolidation* causes loss of functioning lung volume. It is not directly responsive to physiotherapy but in a dehydrated patient it is responsive to hydration, and further complications may be prevented by positioning or mobilization.
- *Pleural effusion, pneumothorax* and *abdominal distension* compress the lung but are inaccessible directly to physiotherapy. Positioning may assist comfort and gas exchange, and sometimes re-expansion of the lung may need assistance, e.g. after a pleural effusion has been drained.
- *Restrictive disorders* of the lung or chest wall reduce lung volume but are less responsive to physical treatment.

Even when the condition is not directly responsive to physiotherapy, the patient may still need attention.

When increasing lung volume, the distribution of the extra air should be directed to poorly ventilated lung regions. In postoperative or immobile patients this is usually the lower lobes.

Loss of lung volume is a problem when it causes a significant degree of:

- ↓ surface area for gas exchange
- ↓ lung compliance (Figure.1.3)
- ↑ work of breathing.

CONTROLLED MOBILIZATION

The most fruitful technique for increasing lung volume is exercise (Dean, 1994). When accurately targeted, this combines upright posture, which reduces pressure on the diaphragm and encourages basal distribution of air, with natural deep breathing. It is the first-line treatment for patients who can get out of bed.

To ensure accuracy, the level of activity is controlled so that the patient becomes just slightly breathless but avoids muscle tension, then s/he is asked to lean back against a wall to get his/her breath back, while being discouraged from talking, which would upset the breathing rhythm. Relaxing against a wall minimizes postural activity of the abdominal muscles, allowing the diaphragm to descend more freely. The controlled 'slight breathlessness' then becomes therapeutic as deep breathing, rather than wasted as shallow apical breathing. For patients who have not just had surgery, some find that holding their hands behind their backs while leaning against the wall further frees their breathing.

Patients who are not able to walk can use controlled activity by simply transferring from bed to chair, then they 'get their breath back' by relaxing against the back of the chair. Even less ambitiously, when bed-bound patients have simply rolled into side-lying, they can be encouraged to relax in the appropriate position while returning to normal tidal breathing.

Once patients understand these principles and can identify the feeling of 'slight breathlessness' and 'getting their breath back', they can practise on their own, using walking and their normal functional activities as a medium for improving lung volume. Regular graded exercise can then be encouraged and monitored by the physiotherapist.

Principles of safety when mobilizing patients are the following:

- Check brakes on beds, chairs and wheelchairs.
- Place chairs strategically in advance, supported against a wall.

- Place chairs on stair landings if there is space.
- Watch intravenous lines.
- Ensure that patients dangle their legs over the edge of the bed for a period before standing.
- Avoid holding a patient's arm if it is being used for a walking aid.
- Ensure that patients keep their hands out of their pockets.
- For the first 24 hours after surgery, watch the patient's face for colour change that might indicate postural hypotension caused by preoperative fluid restriction and perioperative fluid shifts.
- Discourage breath-holding; encourage steady relaxed breathing.
- When sitting a patient in a chair or wheelchair, add extra stability by tucking a foot behind a chair leg or wheel.
- Stand below the patient when going up or down stairs.
- If a patient falls, hospital manual handling protocol should be followed, but it is the patient's head that is vulnerable and it can sometimes be held against the physiotherapist for protection during the fall.

POSITIONING

Changing a patient's position may not seem a dramatic procedure but this simple action often prevents recourse to more time-consuming techniques that can be tiring for the patient. Positioning should be an integral part of all respiratory care, especially when prophylaxis is the aim. It is used in its own right or in conjunction with other techniques. No physiotherapy treatment should be carried out without consideration of the position in which it is performed.

Positioning affects several aspects of lung function:

- Lung volume is related to displacement of the diaphragm and abdominal contents. Functional residual capacity (FRC) decreases from standing to slumped sitting (Figure 6.1). Macnaughton (1995) found that FRC can drop by up to 1 litre from the standing to the supine position.
- Lung compliance decreases and work of breathing (WOB) increases progressively from standing, to sitting, to supine. In supine, lung volume is restricted by (1) the load of the viscera, (2) increased thoracic blood volume and (3) small airway closure. Wahba (1991) found that WOB was 40% higher in supine than in sitting.

(a)

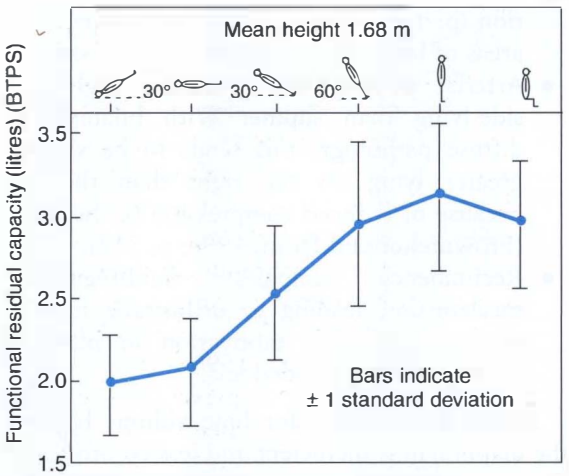


Figure 6.1 (a) Functional residual capacity (FRC) in different positions. (From Lumb, A. B. (2000) *Nunn's Applied Respiratory Physiology*, 5th edn, p. 52, with permission.)

(b)

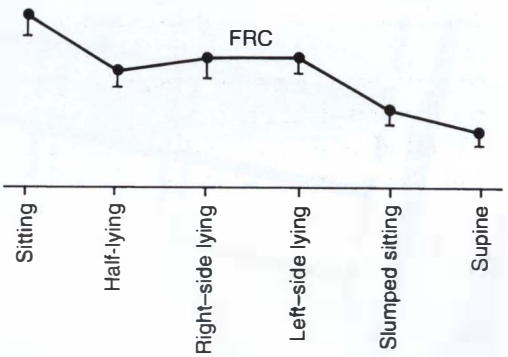


Figure 6.1 (b) FRC as a percentage of the sitting value. 'Sitting' means sitting upright with legs dependent. (From Jenkins *et al.*, (1988) The effect of posture on lung volumes. *Physiother.*, **74**, 492–496.)

- Despite compensatory hypoxic vasoconstriction (p. 13), a degree of perfusion persists in areas of low volume, which increases shunt.
- Arterial oxygenation is usually higher in side-lying than supine. With bilateral or diffuse pathology, this tends to be slightly greater lying on the right than the left because of reduced compression of the heart (Frownfelter and Dean, 1996, p. 312).
- Recumbency impairs fluid-regulating mechanisms, leading to orthostatic intolerance and reduced motivation to mobilize because of light-headedness.

Supine is unhelpful for lung volume because the diaphragm is inefficient and less co-ordinated with chest wall mechanics (Wahba, 1991). The slumped position is unhelpful because of pressure against the diaphragm from the abdominal contents.

The following principles apply to immobile or relatively immobile patients with atelectasis or potential atelectasis:

- Time should be spent in side-lying, well forward so that the diaphragm is free from abdominal pressure (Figure 6.2). Side-lying can also be encouraged for sleeping. A 2-hourly position change has been recommended (Brooks-Brunn, 1995).
- Half-lying in bed rapidly becomes the slumped position for most patients as they slide down the bed (Figure 6.3). Time in half-lying should be limited for patients with loss of lung volume, unless necessary for a specific medical reason or to minimize pain.

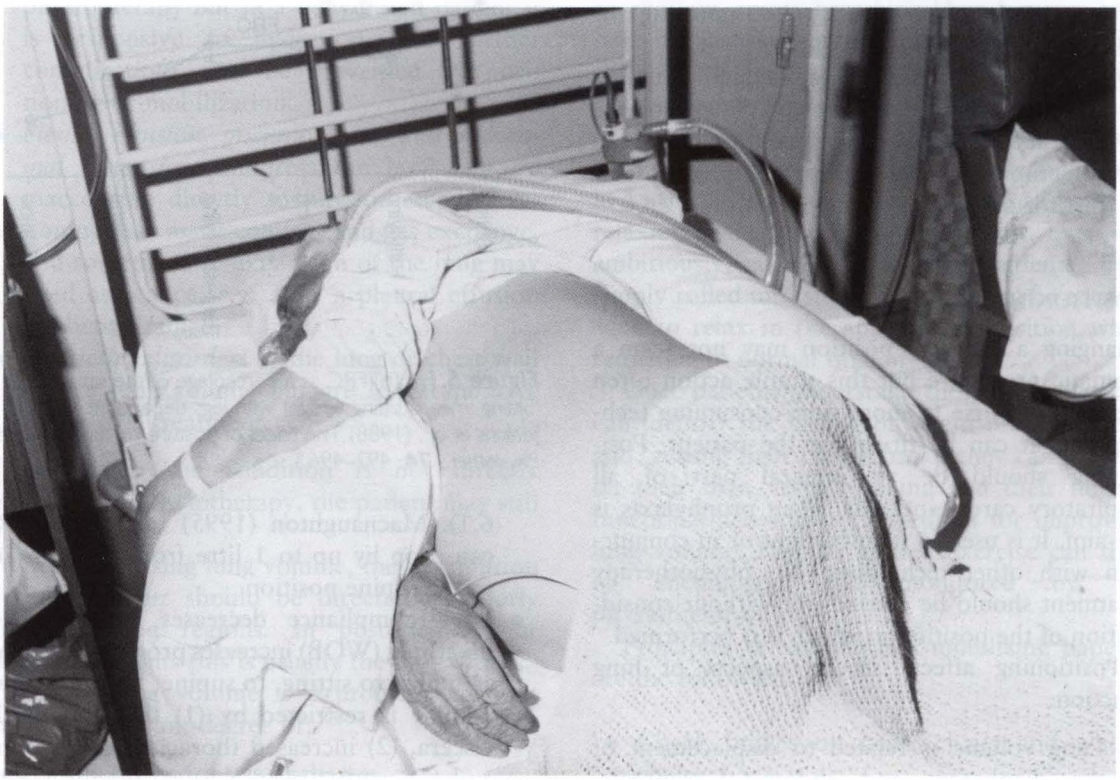


Figure 6.2 Side-lying position. The patient has an acutely distended abdomen, but the diaphragm is relieved of pressure by the patient being rolled well forward.

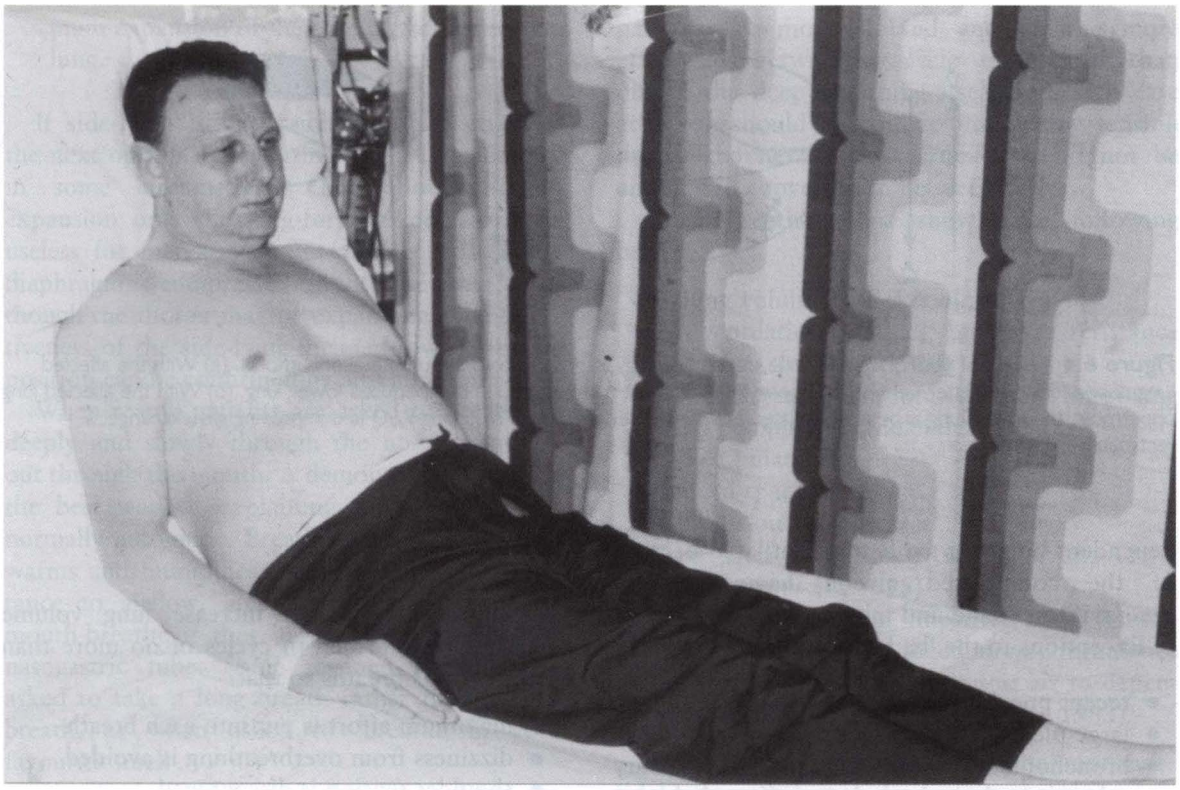


Figure 6.3 The slumped position.

Manoeuvres to increase volume, such as deep breathing, are relatively ineffective in half-lying because of pressure from abdominal viscera.

- When sitting out a patient after treatment, a footstool may be inadvisable unless the patient has ankle oedema or a recent vein graft, or finds this position more comfortable.
- Lengthy positioning in supine is best avoided for those who have a high closing volume, e.g. people who are elderly, obese or smoke heavily.

Positioning also affects the $\dot{V}_A/\dot{Q}$ ratio. Ventilation and perfusion are usually matched because the better-ventilated dependent lung is also better perfused. For people with one-sided pneumonia, reduced ventilation on the affected side overrides the physiological ventilation gradient. Lying with the affected lung uppermost means that the better ventilation of the dependent normal lung is matched with better perfusion (Figure 6.4). Perfusion is always greater in dependent areas, and $\dot{V}_A/\dot{Q}$ match is therefore enhanced in the 'bad lung up' position, sometimes resulting in a dramatic improvement in gas exchange. $\dot{V}_A/\dot{Q}$ is usually mismatched if the affected lung is dependent (Gillespie and Rehder, 1987).

As well as optimizing gas exchange, the 'bad lung up' rule suits other situations. It promotes comfort following thoracotomy or chest drain placement, facilitates postural drainage, and helps improve lung volume when atelectatic lung is positioned uppermost to encourage expansion. With atelectasis, the uppermost areas are stretched and better expanded, even though the

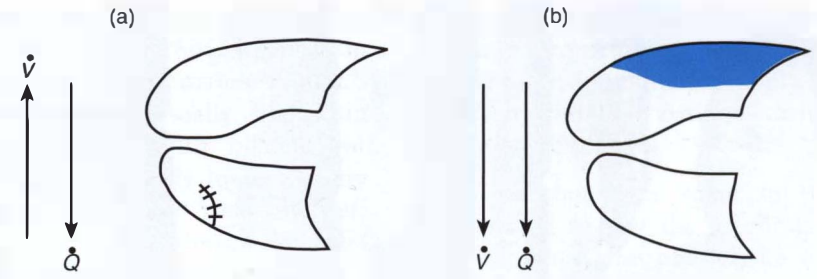


Figure 6.4 Effect of positioning with one-sided pathology, e.g. thoracotomy, unilateral pneumonia. (a) With the affected lung dependent, the better-ventilated uppermost lung is not matching the better-perfused lower lung. (b) With the affected lung uppermost, the lower lung is better-ventilated and better-perfused, thus matching $\dot{V}_A/\dot{Q}$ and improving gas exchange. $\dot{V}$ = *ventilation;* $\dot{Q}$ = *perfusion.*

dependent lung may be better ventilated because of the compressed alveoli having greater potential to expand and take in fresh gas.

Exceptions to the 'bad lung up' rule are:

- recent pneumonectomy (p. 268)
- large pleural effusion (p. 99)
- bronchopleural fistula, in case any unsavoury substances drain into the unaffected dependent lung
- occasionally if there is a large tumour in a main stem bronchus, when positioning the patient with the affected side uppermost may obstruct the bronchus and cause breathlessness
- any situation in which the oximeter or patient comfort indicates otherwise.

The above are guidelines only and patients need individual assessment. After treatment, the physiotherapist should explain to nursing staff why the patient has been left in a specific position, and that this should be maintained until the patient wants to move or it is time to turn. Night staff should be included in training on positioning. Oximetry is useful to demonstrate the effectiveness of positioning to both patient and staff. Accurate positioning and regular position change should be incorporated into a patient's management plan 24 hours a day.

BREATHING EXERCISES

Breathing exercises to increase lung volume should be performed in cycles of no more than three or four breaths so that:

- maximum effort is put into each breath
- dizziness from overbreathing is avoided
- shoulder tension is discouraged.

Once inflated, alveoli stay open for about an hour at normal tidal breaths, so it is advisable to do at least 10 deep breaths every waking hour to maintain lung volume (Bartlett *et al.*, 1973). This is a tall order for those who are distracted by the events and uncertainties of hospital life, and patients can use reminders such as food and drink trolleys or nurses' hourly observations.

Deep breathing

Optimum conditions are needed to ensure that deep breaths reach peripheral regions. The following will facilitate this:

- relief of pain, nausea, dry mouth, discomfort, fatigue, anxiety or tension
- avoidance of distractions
- minimum breathlessness, e.g. patients need time to get their breath back after turning
- accurate positioning, usually side-lying-inclined-towards-prone, to facilitate maxi-

mum expansion of the base of the uppermost lung.

If side-lying is impossible, upright sitting is the next option. Long-sitting might be necessary in some circumstances but allows limited expansion only. Leaning-forward-long-sitting is useless for increasing lung volume because the diaphragm is compressed up into the chest, even though the thorax may be expanding. The effectiveness of the side-lying-inclined-towards-prone position can be confirmed by auscultation.

When ready, patients are asked to breathe in deeply and slowly through the nose, then sigh out through the mouth. A demonstration is often the best way of explaining an action that is normally automatic. Breathing through the nose warms and humidifies the air but doubles resistance to airflow, and patients may prefer to mouth-breathe if they are breathless or have a nasogastric tube. Some respond better when asked to take a long breath rather than a deep breath, or when asked to 'breathe in your favourite smell'.

Distribution of ventilation is related to position, flow and pathology (Menkes and Britt, 1980). The physiotherapist's hands may be placed over the basal area for monitoring purposes and for patient reassurance, but not with any assumption that this magically redistributes ventilation to the underlying lung. 'Localized' breathing exercises do not make physiological sense because humans are unable to deform individual portions of the chest wall (Martin *et al.*, 1976). But patients can still be found obediently performing 'unilateral breathing' and 'basal costal breathing'. Even if localized breathing were physically possible, as in some yoga masters, the way in which the two layers of pleura slide on each other means that the lung responds generally rather than locally to a deep breath.

After every few breaths, the patient should relax and regain his/her rhythm. Breathing rate and pattern should be observed at this time, and the patient may need praise or a change in instruction before proceeding. Sometimes patients are more relaxed and breathe more effectively between a cycle of breaths than during the deep breathing itself, in which case attention should be paid to minimizing tension during the next cycle. Patients should not be engaged in conversation between cycles.

Deep breathing has shown the following benefits:

- ↑ lung volume (Jones *et al.*, 1997)
- ↑ ventilation and ↓ airways resistance (Menkes and Britt, 1980)
- ↑ surfactant secretion, thereby improving lung compliance (Melendez, 1992)
- ↑ $\dot{V}_A/\dot{Q}$ matching
- ↓ dead space ratio
- ↑ diffusion (Prabhu *et al.*, 1990)
- ↑ oxygen saturation (Ruggier *et al.*, 1994; Dallimore *et al.*, 1998)
- with slow breathing, improved basal ventilation as a result of reduced airway turbulence and preferential distribution of air to dependent regions (Reid and Loveridge, 1983).

Shallow breathing is inefficient because more tidal volume is lost to dead space as the same air is inhaled and exhaled more often. Breathless people require a special approach and should not be asked to breathe slowly (p. 174).

The term 'thoracic expansion exercises' is synonymous with deep breathing. Thoracic expansion can be readily observed but does not guarantee a deep breath. Literal interpretation of the terminology, or the patient's position, may inhibit the more subtle abdominal excursion.

End-inspiratory hold

Air can be coaxed into poorly ventilated regions by interspersing every few deep breaths with breath-holds for a few seconds at full inspiration. This distributes air more evenly between lung segments and boosts collateral ventilation (Cormier *et al.*, 1991). Ideally, inflating pressures of 30–50 cmH_2O should be held for 5 seconds at 2–6 times tidal volume (Brooks-Brunn, 1995) but many postoperative patients cannot achieve this. Observation will identify if the end-inspiratory hold is effective, comforta-

ble or, conversely, disturbs the breathing pattern. Accurate instruction is needed to prevent shoulder girdle tension. The end-inspiratory hold is unsuitable for breathless people, who should not be asked to hold their breath. It should be used with caution in patients with a tendency to bronchospasm (Mutatkar, 1999).

Abdominal breathing

Emphasis on abdominal movement during inspiration leads to slower, deeper breathing, less turbulence, reduced dead space and shoulder girdle relaxation. The term 'diaphragmatic breathing' is sometimes understood by patients, although abdominal movement does not ensure greater diaphragmatic contribution to breathing (Gosselink, 1995). The term 'breathing control' is synonymous with abdominal breathing at tidal volume.

The patient is asked to get comfortable in a symmetrical position such as upright sitting. The manoeuvre is first explained and demonstrated unhurriedly, avoiding words like 'push', 'pull', 'try' and 'harder'. If relaxed abdominal breathing has not started naturally, then the patient is taken through the following steps:

- Rest the dominant hand on your abdomen, with elbows supported, and, keeping your shoulders relaxed, allow your hand to rise gently while visualizing air filling your abdomen like a balloon.
- Sigh the air out.
- Check that shoulders remain relaxed and heavy.
- Gradually increase the depth of breathing while maintaining relaxation. (If the aim is increased lung volume.)
- If appropriate, progress to side-lying and relaxed standing.

Many patients respond to the physiotherapist's hands placed on the lower abdomen to encourage breathing 'in and down'.

Variations include:

- putting the other hand on the quiet upper chest to compare it with movement of the abdomen
- reminding patients that, when filling a kettle, they don't fill the top half first
- imagining a piece of elastic round the waist stretching during inhalation
- if supine, placing a box of tissues on the abdomen to visually reinforce the movement
- incorporating incentive spirometry (p. 156) to encourage a slow flow rate and give feedback on the larger volume inhaled (Peper, 1992)
- some non-surgical patients find that other positions facilitate abdominal movement, e.g. prone lying, four-point kneeling or standing with hands on the back of the hips and elbows pushed backwards.

Abdominal breathing usually increases the lung volume but is not thought to alter the distribution of ventilation (Martin *et al.*, 1976). However, the slow deep breathing that it incorporates favours peripheral distribution (Fixley, 1978).

Sniff

Even after a full inspiration, it is often possible to squeeze in a wee bit more air and further augment collateral ventilation by taking a sharp sniff at end-inspiration. Sceptical patients can be won over by a reminder that however packed a rush-hour underground train is, an extra person can always be crammed in.

Neurophysiological facilitation

Although positioning is the most cost-effective way of maintaining lung volume, neurophysiological facilitation (NPF) is useful for some non-alert patients such as those who are drowsy postoperatively, those with neurological conditions or those partially breathing on a ventilator, especially if they are unable to turn. It is thought that cutaneous and proprioceptive stimulation reflexly increases the depth of breathing (Jones, 1998), albeit in the short term. The perioral technique (Figure 6.5) is thought to relate to the suckling reflex, and may facili-

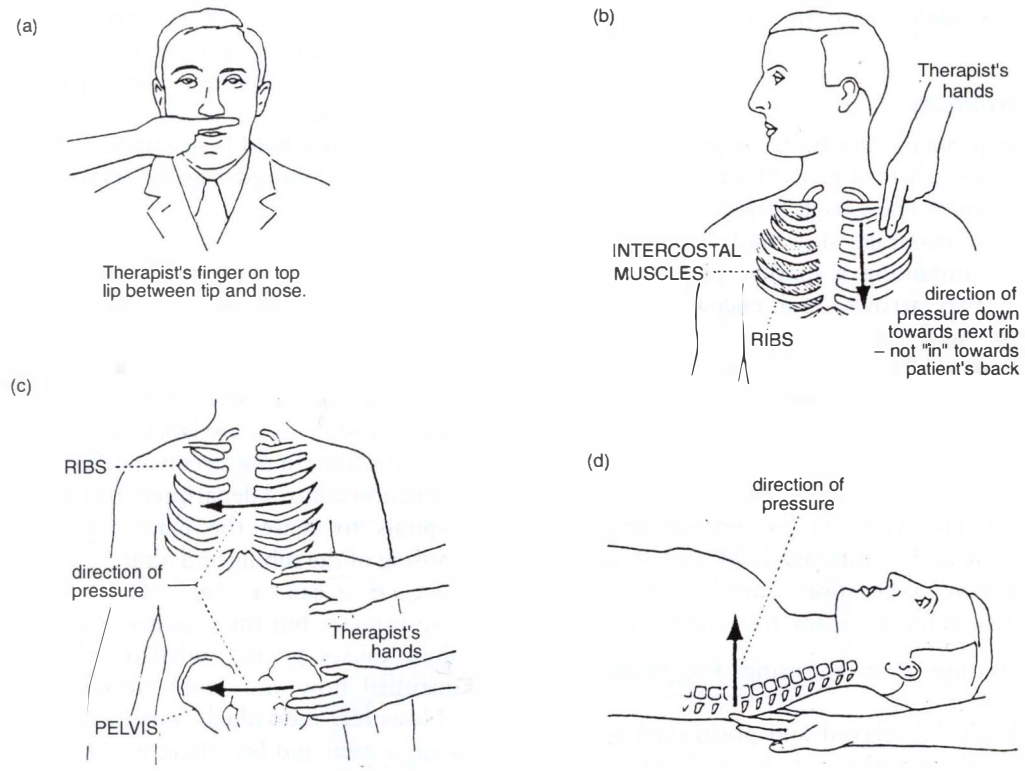


Figure 6.5 (a) Perioral stimulation: moderate finger pressure is maintained inwards and downwards, just above the lip, as long as the patient is required to deep-breathe. The effect may continue for some minutes afterwards. (b) Intercostal stretch: pressure is downwards towards toes, on the upper border of the rib at end-expiration. Unilateral or bilateral. Not for floating ribs. (c) Co-contraction of abdominal muscles: pressure laterally over lower ribs and pelvis, at right angles to patient, alternating right and left sides and maintaining pressure for up to 2 minutes or until desired effect. (d) Vertebral pressure: finger pressure against thoracic vertebrae between T2 and T10 (D. D. Bethune, 1975, *Physiother. Can.* **27**, 242–245).

tate slow as well as deep breathing. Other effects seen with NPF are yawning, coughing, swallowing, abdominal contraction and occasionally change in consciousness (Jones, 1998). Some patients vary in their response from breath to breath and day to day. It is worth trying slightly different finger positions and pressures, and sometimes finger vibrations. Effects may be cumulative.

Rib springing

Rib springing is chest compression on expiration, with overpressure downwards and inwards in the bucket-handle direction of rib movement, then a quick release at end-expiration. This may cause a deeper subsequent inspiration, especially when performed slowly and smoothly. It is sometimes used with paralysed patients but is less effective, less comfortable and less safe than NPF.

MECHANICAL AIDS TO INCREASE LUNG VOLUME

If previous measures are ineffective, a variety of mechanical aids are available to increase lung volume. Physiotherapists are ideally suited to match people to machines because of their

patient-handling skills and understanding of physiology.

Incentive spirometry

A sustained deep breath can be facilitated by an incentive spirometer, which gives visual feedback on flow and volume. The Coach (Figure 6.6) and Voldyne devices encourage slow and controlled inhalation by maintaining a marker (indicating flow) between two arrows, and encourage an end-inspiratory hold while a disc (indicating volume) descends. In the Triflo device, two out of three plastic balls should be raised and the breath sustained for some seconds while they are suspended. The third ball is a control and should not be raised because this indicates high flow and turbulence. The Triflo is less encouraging for sustaining an end-inspiratory-hold and it is possible to cheat by taking short sharp breaths.

The suggested technique is the following:

1. A demonstration is given using a separate device.
2. Patients should be relaxed and positioned as for deep breathing, either side-lying or sitting upright, preferably in a chair.
3. With lips sealed around the mouthpiece, the patient inhales slowly and deeply. Throughout the procedure the patient watches the incentive spirometer while the physiotherapist monitors the patient's breathing pattern.
4. An end-inspiratory hold is sustained.
5. After exhalation, shoulder girdle relaxation is rechecked.

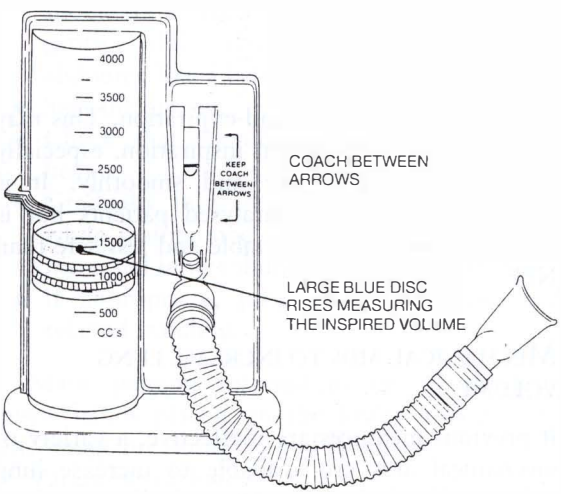


Figure 6.6 Incentive spirometer

Those on oxygen can use nasal cannulae or an incentive spirometer which entrains oxygen. People with tracheostomies can use a connecting tube. Once technique is faultless, patients are asked to take 10 incentive spirometry breaths per waking hour. Most devices are labelled for single-patient use but a filter in the circuit and discussion with the infection control department has enabled some hospitals to reuse the main component while disposing of the tubing and filter.

The same effect can be obtained without the incentive spirometer but the incentive of using a device often causes greater inhaled volume, a more controlled flow and more enthusiasm to practise. However, individuals vary, and observation of expansion and breathing pattern shows whether the patient breathes more effectively with or without the device. Incentive spirometry is also suited to children and those with learning difficulties because it can be learnt by demonstration. It is not suitable for breathless patients.

Mini literature appraisal

Literature:

'There is little evidence to support the use of incentive spirometry in airway clearance....'

Eur. Respir. J. 1999; 14: 1418–1424

Appraisal: Incentive spirometry is not intended for airway clearance.

Continuous positive airway pressure

For spontaneously breathing patients who cannot muster the breath for incentive spirometry, resting lung volume and gas exchange can be

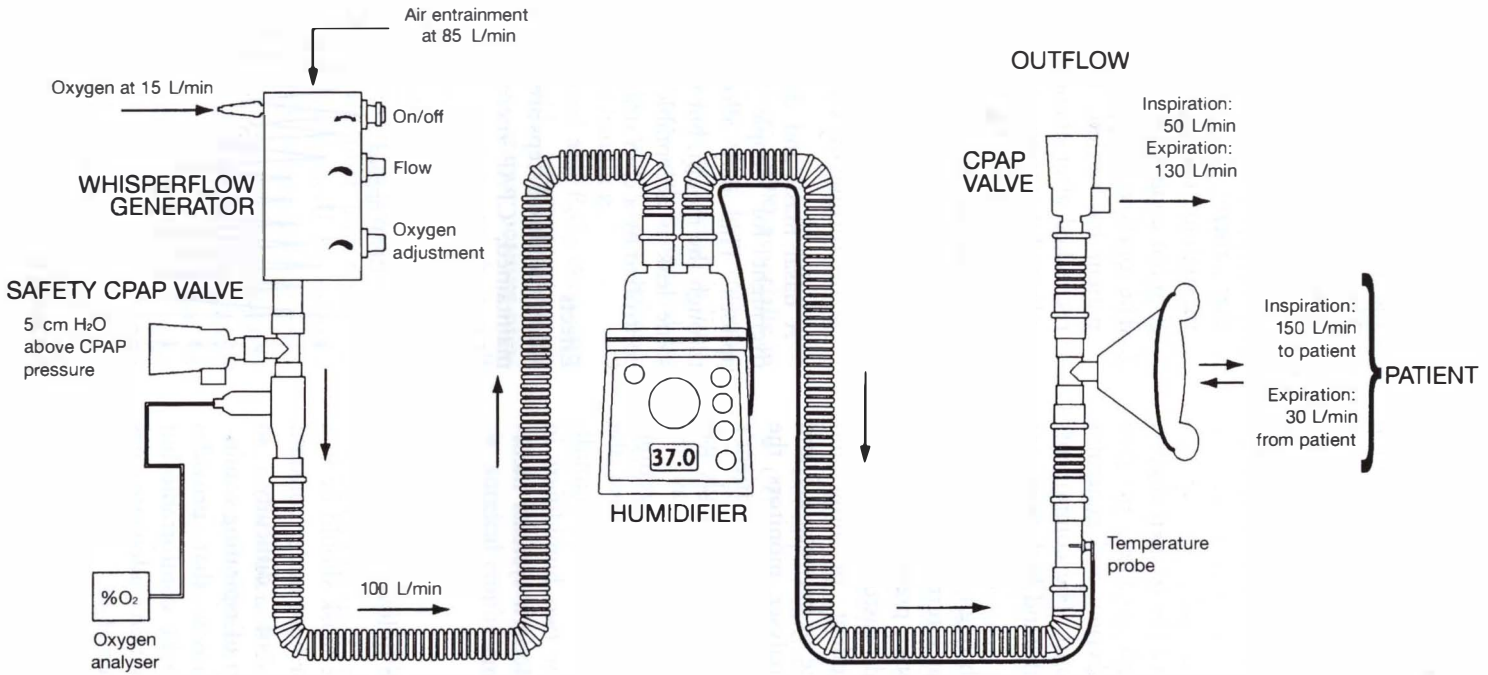


Figure 6.7 CPAP circuit. The main CPAP valve is positioned on the opposite side of the rest of the circuit to prevent CO_2 rebreathing, and a spare valve at 5 cmH_2O above the threshold pressure acts as a pop-off safety valve (Medicaid, with permission).

improved by pneumatically splinting open the airways and alveoli with continuous positive airway pressure (CPAP). A CPAP device delivers a constant flow of gas throughout inspiration and expiration. This exceeds the flow rate of patients even when they are breathless. It is like a person putting their head out of the window of a rapidly moving car.

The system

A flow generator connects to the oxygen supply, entrains room air through a filter to give between 30% and 100% oxygen and generates gas flows of up to 150 L/min. The components needed to generate and withstand high flows are illustrated in Figure 6.7.

The patient breathes through a face mask, nasal mask, mouthpiece (for intermittent use) or T-piece (if intubated). Positive pressure is maintained by a preset threshold resistor valve (CPAP valve), which is independent of flow and provides a constant pressure of between 2.5 and 20 cmH_2O. An oxygen analyser monitors the F_IO_2 and an oximeter monitors the patient's response. To reduce the dryness of high gas flows, a heat-moisture exchanger may be adequate but, for patients with thick secretions or who cannot acclimatize to the dryness, the high flows of CPAP require an efficient humidifier (Wiest *et al.*, 1999), or two humidifiers in series (Harrison, 1993). Modern systems incorporate a high-pressure alarm.

Technique

Suggested guidelines are the following:

1. Patients using a full face mask should be in a high dependency area or kept under constant observation because of difficulty in expectoration and danger of aspirating vomit.
2. A CPAP valve is chosen that provides pressure low enough to be comfortable but high enough to maintain adequate gas exchange, usually 5–10 cmH_2O.
3. The patient is introduced to the mask.
4. Oxygen is adjusted to the required F_IO_2.
5. The flow is turned on.
6. The patient assists with putting on the mask if possible in order to reduce anxiety. It is best that the mask is not strapped on until the patient has felt the flow and is ready. Flow should be at a level sufficient to maintain an open CPAP valve, even during a deep breath. Fine tuning trims it to just above the patient's peak inspiratory flow so that there is outflow from the valve throughout the cycle, while the mask has a firm but comfortable seal.
7. The outflow should be rechecked after the patient has settled. The oximeter should be rechecked after changing the flow, and F_IO_2 adjusted if appropriate.
8. Regular checks are required on the comfort and seal of the mask, the fluid level and temperature of the humidifier, and the oximeter.
9. After use, the mask should be removed before turning off the flow.

A nasal mask and domestic device are used overnight for people with obstructive sleep apnoea. This may allow some air to escape through the mouth, but the high flow means that some leak is acceptable. Excessive leak can be controlled by a chin strap or soft collar.

Effects

When the above steps are followed and comfort is maintained, CPAP increases FRC (Figure 6.8),

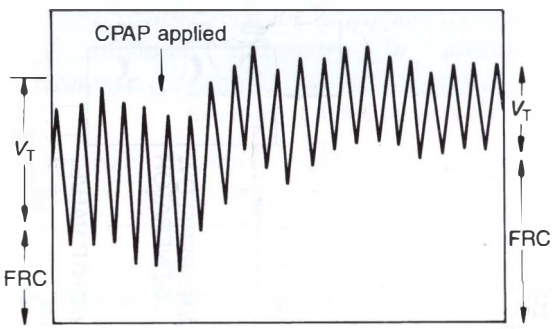


Figure 6.8 Effect of CPAP on lung volumes. V_T = *tidal volume; FRC = functional residual capacity.*

improves gas exchange and may avoid the need for intubation and mechanical ventilation (Keilty and Bott, 1992). Atelectasis may be prevented, but re-expansion of collapsed lung tissue requires sustained pressures of $>15\ cmH_2O$ (Andersen *et al.*, 1980), which are usually intolerable for an alert patient and bring significant complications.

CPAP can also be used for patients with pneumonia (p. 104) or increased WOB due to obstructed airways (p. 84). It can assist gas exchange for people with pulmonary oedema as an interim measure until medication takes effect (Wysocki, 1999).

Complications

- Discomfort is common, and uncomfortable patients restrict their depth of breathing. Individual adjustment of the mask, or a change of mask, may be needed to prevent chafed skin, sore ears or dry eyes. The bridge of the nose should be protected before rather than after a pressure sore develops, using a dressing such as Granuflex (Callaghan, 1998) especially in patients who are hypotensive, hypovolaemic or with thin skin because of ageing or long-term steroids. The mask seal is assisted by having the dentures in. Claustrophobic patients need sensitive handling.
- WOB may be increased and P_aO_2 decreased (Romand and Donald, 1995) because of difficulty in exhalation against positive pressure. If there is loss of lung or chest wall elasticity, patients might be forced to use even more active exhalation.
- At high pressures, gas can be forced into the stomach, causing discomfort and restricted breathing. The risk is reduced by using a nasogastric tube, which is advisable at pressures over $10\ cmH_2O$. If girth is measured to assess for abdominal distension, this should be explained, as it has been interpreted as measuring for a coffin (Waldmann and Gaine, 1996).
- Aspiration is a risk for patients unable to remove the mask rapidly by themselves. The mask must be removed for eating and drinking.
- Coughing without removing the mask can create high pressures, which may damage the ears and, with emphysema or late-stage CF, risk causing a pneumothorax. Some protocols advise that positive pressure techniques should only be used on wards with access to chest drain equipment.
- The system is noisy, which may be detrimental to the patient and neighbours.
- The haemodynamic effects of CPAP vary. Positive pressure may compress alveolar vessels, redistribute blood from chest to abdomen and, at pressures above $10\ cmH_2O$, increase right ventricular afterload. Although cardiac output normally depends on preload, poor ventricular function renders it dependent on afterload (Romand and Donald, 1995), in which case cardiac output may be impaired by CPAP, especially in hypovolaemic patients. For people with normal heart function, pressures above $15\ cmH_2O$ can impair cardiac output (Mayor, 1997).
- CO_2 retention can occur if a hypercapnic patient breathes with a small tidal volume against a high pressure valve.

Precautions

CPAP should not normally be used in the presence of:

- an undrained pneumothorax
- surgical emphysema
- bullae
- facial trauma
- excessive secretions.

It should be used with caution in the presence of:

- bronchopleural fistula
- a large tumour in the proximal airways, because inspired gas under pressure may be able to enter but not exit past the obstruction.

Two conditions for which CPAP may be useful, with precautions, are the following:

- following oesophageal surgery, CPAP assists

gas exchange, but a nasogastric tube is required to prevent positive pressure jeopardising the anastomosis

- a flail chest can be stabilised with CPAP, so long as there is no undrained pneumothorax.

Intermittent positive pressure breathing

The slings and arrows of fashion have not been kind to intermittent positive pressure breathing (IPPB) and attitudes have swung from hero-worship to ostracism. This technique has been scrutinized mercilessly in the literature and found wanting, usually because it has been used in the wrong way for the wrong patients. IPPB is simply pressure-supported inspiration using a non-invasive ventilator such as the Bird (Figure 6.9) Inspiration is triggered by the patient, sustained by positive pressure, and followed by passive expiration.

Indications

Patients with atelectasis who are drowsy, weak

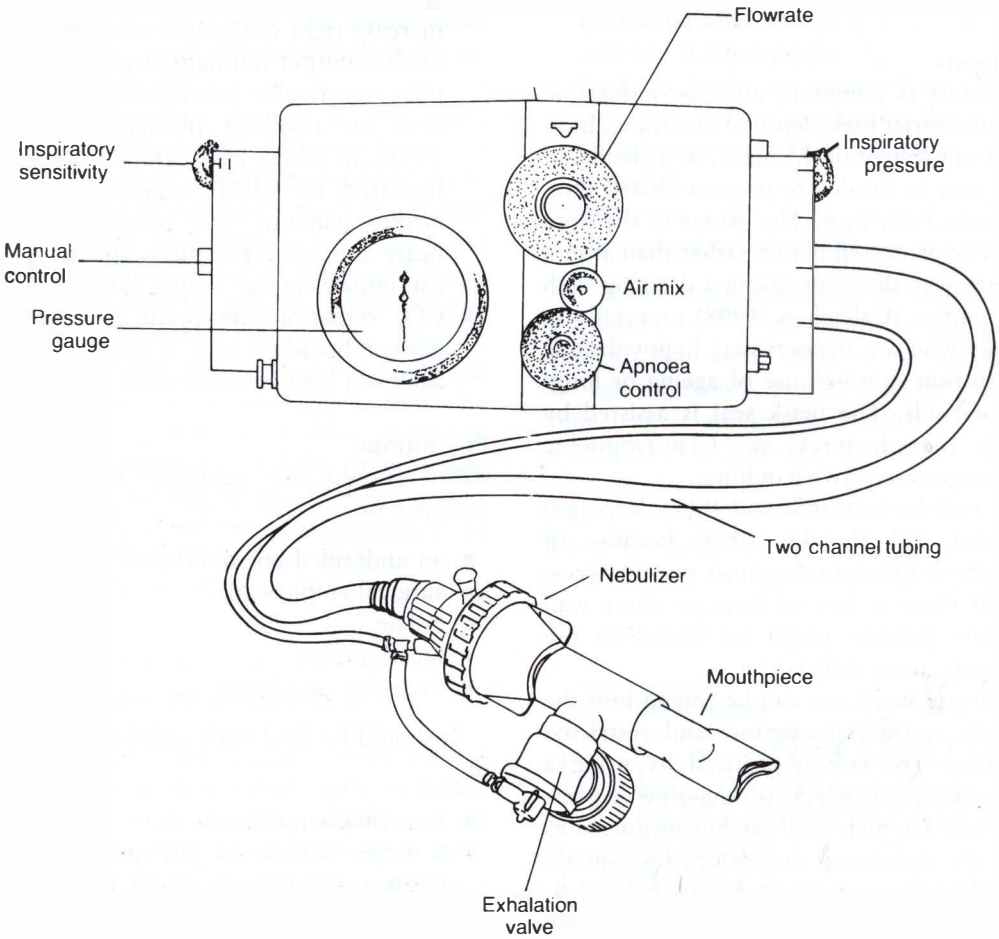


Figure 6.9 Bird ventilator. **Inspiratory sensitivity** regulates the ease with which the machine triggers into inspiration. **Manual control** can override the patient-trigger and machine-cycling mechanisms. The **pressure gauge** indicates the airway pressure. The **flow rate** controls the rate at which gas is delivered to the patient. The **inspiratory pressure** is the pressure that should be reached before cycling into expiration. The **air-mix knob** allows entrainment of room air. The **apnoea knob** controls automatic function and should be off throughout.

or fatigued may benefit from IPPB. Patients who are unwilling, restless or in pain do not. Pain is not a contraindication in itself but, if atelectasis is caused by pain, it is best to deal first with the pain because muscle splinting will prevent the patient from accepting the positive pressure. Sputum retention may be an indication for drowsy, weak or exhausted patients, e.g. those with neurological problems. Excess WOB can also be eased by IPPB (p. 179). IPPB is not indicated for administration of drugs because it does not offer any advantage over a simple jet nebulizer and the positive pressure results in 30% less aerosol delivery to the lungs than a nebulizer alone (Fink and Dhand, 1999).

Technique for the Bird ventilator

The nebulizer is filled with saline and tested by activating inspiration with the red manual button.

If the aim is to increase volume to the lung bases, patients are positioned comfortably in side-lying with the affected lung uppermost. After turning into this position, they are allowed to return to normal tidal breathing, then asked to hold the mouthpiece firmly with their lips. They are advised that extra air will flow into their lungs to help their breathing and reassured that the procedure can be stopped at any time, either by request or, if using a mouthpiece, by inhaling through the nose so that the machine is not triggered.

The inspiratory *sensitivity* determines how much negative pressure the patient must generate in order to trigger a breath, a low number indicating that little effort is required. For the Bird Mark 7 or 8, it is set usually at about 7, or so that the patient can trigger inspiration with ease ('Is it easy to breathe in?').

The *flow rate* determines how fast the gas is delivered, a low number for a long breath and a high number for a short breath. It is set as low as comfortable, starting at about 7–10, to ensure minimal turbulence and optimum distribution of ventilation. Breathless patients need a high flow for comfort ('Is that enough air?').

The inspiratory *pressure* should be set according to patient comfort ('Is that blowing too hard?'). Starting pressure may be about 10. Collapsed lung is difficult to re-expand because of low compliance, and positive pressures of 20 cmH_2O are considered necessary when using bronchoscopy (Spring *et al.*, 1999). The pressure dial should show a smooth rise to the preset pressure at each breath to indicate patient co-ordination.

The patient takes a small breath and the machine does the rest, without the patient prematurely stopping inspiration by active exhalation. When the patient's breathing pattern has settled, the pressure can be gradually increased until maximum expansion is obtained without disturbance to the breathing pattern. The physiotherapist's job is to:

- adjust the pressure, and occasionally adjust the flow rate to compensate, because flow governs the speed with which the preset pressure is reached
- reassure and advise the patient to allow the air to fill the lungs and not to blow out
- observe the abdomen for unwanted active expiration
- observe the face for discomfort
- observe rib cage excursion to ensure that expansion is improving
- afterwards, wash and dry the nebulizer
- liaise with nursing staff and leave written instructions to avoid the patient or family altering the knobs.

The air-mix knob is maintained in the 'out' position by a clip, which ensures that air is entrained and 40–45% oxygen is delivered. For patients who require high levels of oxygen, 100% is delivered by pushing the air-mix knob in. For 24% oxygen, the machine is run on air with oxygen entrained at 2 L/min via a needle (through the red bung if the reusable circuit is used). Finer adjustments are achieved with an oxygen blender attachment. For Entonox, the air-mix knob is pushed in to ensure the patient receives all of the gas. The flow rate may need to be increased with these modifications.

The apnoea switch should be turned fully

clockwise to the off position to prevent operation as a conventional ventilator. The manual button is not used routinely.

A retard cap can be used to create a slight positive end-expiratory pressure, which the manufacturers claim may prevent recollapse of alveoli. A mouth flange can be used to assist the mouthpiece seal. A mask can be held on the face of semi-conscious people, but is frightening, and patients need explanations and the freedom to say no.

IPPB can be used via tracheostomy or endotracheal tube with inflated cuff but barotrauma is a risk because of the sealed system.

For acute patients, the physiotherapist should be present throughout in order to make the fine adjustments needed. Occasionally, well-practised and alert patients can use it independently, e.g. those with asthma or fibrosis, who are sometimes reassured by having it available by their beds at night. IPPB is best used for short periods of time, after which other techniques such as positioning or, if necessary, CPAP can be used to maintain the lung volume achieved.

If secretion clearance is to be included, measures that decrease lung volume, such as the head-down tip or manual techniques, are usually inappropriate during IPPB, although percussion may be suitable during expiration if it does not upset the breathing pattern.

Troubleshooting

- If there is prolonged inspiration and the preset pressure is not reached, check for leaks in the circuit, at the mouth or through the nose. If these are not the cause, try reducing pressure and/or increasing flow.
- If the preset pressure is reached too quickly, check that the patient is not actively breathing out, blocking the mouthpiece with the tongue or letting pressure generate in the mouth only. If a semi-conscious patient blocks their airway, the head should be slightly extended and the jaw protracted.
- If the machine triggers into inspiration too early, turn up the sensitivity; check apnoea knob is off.
- If the machine repeatedly triggers during inspiration, the patient may need a greater delivery of gas by increasing flow or pressure. Check that servicing is up to date.
- If a reusable circuit does not nebulize, check that the red bung is uppermost.

Effects and complications

If the patient is relaxed, comfortable and well positioned, with controls accurately adjusted, IPPB should increase lung volume, this increase lasting for about an hour (AARC 1993a), or longer with correct positioning. Compared to CPAP, positive pressure is intermittent and typically reaches higher pressures, thus increasing tidal volume, whereas CPAP increases FRC. In practical terms, IPPB is best for opening up collapsed alveoli and CPAP is best for maintaining the increased lung volume.

IPPB can also improve ventilation and gas exchange and, if the patient does not attempt to assist the machine, reduce WOB (Bott *et al.*, 1992). IPPB has certain disadvantages compared to other mechanical aids:

- It is less effective than spontaneous deep breathing (Bynum *et al.*, 1976), which is why IPPB is unnecessary for patients who can deep-breathe independently.
- The extra volume is distributed preferentially to areas already well ventilated, because of the passive nature of inspiration and absence of normal diaphragmatic activity (Celli *et al.*, 1984), hence the importance of positioning the collapsed area uppermost.
- It is possible that hypercapnic COPD patients may lose their hypoxic respiratory drive because of the 40% oxygen delivered. Starke *et al.* (1979) claim that this is not a problem if adequate tidal volumes are delivered, but it would be advisable for patients at risk to be kept under observation after treatment. Alternatively, air can be used as the driving gas instead of oxygen, with modest amounts of supplemental oxygen added, either via a nasal cannula (if a mouth-

piece is used) or entraining oxygen as described above.
- Air swallowing may occur, especially if there is cheek distension or the patient burps afterwards. This may be relieved by left-side-lying.

A side effect that can sometimes be used to advantage is that techniques such as IPPB, incentive spirometry and deep breathing can make patients slightly breathless, even though this is not the aim. These patients can be positioned for optimum distribution of ventilation, then allowed to return to normal tidal breathing. If undisturbed, this encourages comfortable deep breathing using the same 'slight breathlessness' principle as with controlled mobilization.

Box 6.1 Characteristics of mechanical aids to increase lung volume (*FRC = functional residual capacity*)

Incentive spirometry
Full patient participation
End-inspiratory hold
Physiological distribution of ventilation
Minimal supervision
Minimal infection risk
Quiet
Cheap

CPAP
Positive pressure continuous
Face or nasal mask
Can accommodate breathless patient
Can accommodate tired patient
Used for raising FRC

IPPB
Positive pressure on inspiration only
Mouthpiece or face mask
Used periodically
Can accommodate breathless patient
Can accommodate tired patient
Can accommodate semiconscious patient
Used for raising tidal volume

Precautions
These are similar to those for CPAP, although the risk is greater if higher pressures are used.

Box 6.1 compares the different devices.

Mini literature appraisal

Title: Efficacy of chest physiotherapy and intermittent positive pressure breathing in the resolution of pneumonia. *N. Engl. J. Med.* 1978; 299: 624–627.
Question: Do we need to read further than this?
Comment 1: Neither 'chest physiotherapy' nor IPPB could logically influence the pathology of pneumonia.
Comment 2: Two modalities were used together. If one of the two variables had been effective, it could not have been identified.

OUTCOMES

Success in the treatment of patients with reduced lung volume can be measured by the following:

- improved breath sounds
- more resonant percussion note
- clearer X-ray
- greater chest expansion
- improved S_aO_2, so long as other variables which affect this are excluded, e.g. ↑ F_IO_2.

MINI CASE STUDY: MS MB

Identify the problems of this 72-year-old postoperative patient, then answer the questions.

Background

SH: sheltered accommodation, walks with frame.
HPC: OA knee.
14/10/98 right total knee replacement.
16/10/98 transferred to ICU due to respiratory distress, disorientation and sputum retention, intubated and ventilated.
17/10/98 extubated and returned to ward.

Subjective

Sleepy, wakeful night.
Little pain.

Objective

Apyrexial.
Good fluid balance.
Obese.
Slumped in bed.
Restless.
Rapid asymmetrical breathing pattern.
Feeble non-productive coughs.
Frequently falls asleep → mask slips → S_aO_2 drops.
Percussion note: dull LLL.
Auscultation: bronchial breathing RLL and middle lobe, ↓ breath sounds LLL, scattered coarse crackles.
S_aO_2: 52% on air, 60% on F_IO_2 of 0.6.

When ABGs were taken, P_aCO_2 was found to be raised and a diagnosis of COPD was made. F_IO_2 was reduced to 0.28.

Questions

1. Analysis?
2. Problems?
3. Goals?
4. Plan?

ABGs = arterial blood gases; ICU = intensive care unit; LLL = left lower lobe, OA = osteoarthritis; RLL = right lower lobe.

RESPONSE TO MINI CASE STUDY

1. Analysis

CXR indicates loss of lung volume bibasally (Figure 6.10).
Possible causes of disorientation: hypoxia, hypercapnia, experience of ICU environment, lack of sleep.
Immobility, poor position and shallow breathing conducive to ↓ lung volume.
Disorientation and immobility conducive to sputum retention.

2. Problems

Inability to fully co-operate.
Atelectasis.
Sputum retention.
Poor gas exchange.
Knee potentially weak and immobile.

3. Goals

Short term: orientate, improve ventilation and optimize gas exchange, mobilize.
Long term: rehabilitate for sheltered accommodation.

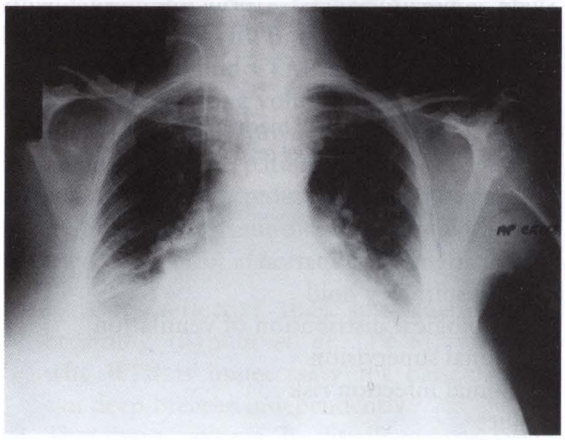


Figure 6.10 Ms MB.

4. Plan

- Liaise with ICU physiotherapist about previous management.
- Request ABGs after change in F_IO_2
- Communicate with patient, family and health team to assist orientation.
- Optimize environment for autonomy, familiarity, rest and sleep.
- Position for gas exchange, mobilization of secretions, knee comfort and function.
- IPPB with controlled oxygen, progressing to incentive spirometry and deep breathing exercises as patient becomes more alert.

- Percussion and vibrations, progressing to ACB/AD.
- Daily programme of knee exercises.
- Sit out, mobilize with walking frame, progress.

ACB/AD = active cycle of breathing/autogenic drainage.

LITERATURE APPRAISAL

Comment on the logic and physiology of the following:

> ***[I]n patients with suspected pulmonary emboli there is no evidence that IPPB would increase alveolar ventilation more than deep breathing....***
>
> ***From the term 'deep breathing', it is understood that by voluntarily moving regions of the wall of the thoracic cage, underlying lung tissue is appropriately aerated.***
>
> *S. Afr. J. Physiother.* 1991; 41: 63–67

RESPONSE TO LITERATURE APPRAISAL

The premise defies logic. To increase alveolar ventilation would not assist a patient with a pulmonary embolus.

The physiology defies logic. Deep breathing can increase tidal volume 10-fold (p. 56). A glance at a patient on IPPB shows only a minor increase in tidal volume.

The logic defies logic. The only way of 'moving regions of the wall of the thoracic cage' is to fracture the ribs.

RECOMMENDED READING

Barnitt, R. and Fulton, C. (1994) Patient agreement to treatment: a framework for therapists. *Br. J. Ther. Rehabil.*, 1, 121–127.

Bott, J., Keilty, S. E. J. and Noone, L. (1992) Intermittent positive pressure breathing – a dying art? *Physiotherapy*, 78, 656–660.

Macnaughton, P. D. (1995) Posture and lung function in health and disease. *Br. J. Intens. Care*, 1(4), 133–137.

Ntoumenopoulos, G. (1995) Topical issues in cardiopulmonary physiotherapy. *Physiotherapy*, 81, 92–94.

Sully, P. (1996) The impact of power in therapeutic relationships. *Nurs. Times*, 92, 40–41.

7 Physiotherapy to reduce the work of breathing

Summary

Introduction

Increased work of breathing (WOB) in spontaneously breathing patients is manifest subjectively by breathlessness and objectively by a distressed breathing pattern. Breathless patients are caught in a pincer of decreased ventilatory capacity and increased ventilatory requirements. The basic principle of reducing WOB is therefore to balance supply and demand, as summarized in Table 7.1.

Table 7.1 Measures to optimize the balance between energy supply and demand

Measures to increase energy supply	Measures to decrease energy demand
Nutrition	Stress reduction
Oxygen therapy	Sleep and rest
Fluid and electrolyte balance	Positioning
O_2 delivery to inspiratory muscles (e.g. haemoglobin, cardiac output)	Relaxation
	Breathing re-education
	Mechanical support

Breathlessness

> ***'It's very difficult not to panic when you're fighting for breath ... you feel as if a vacuum is sucking the air out of you ... it threatens your very existence ... you're quite literally fighting for your life.'***
>
> Patient quoted by Williams, 1993

Breathlessness is the major link between lung disease and disability (Wilson and Jones, 1989). It is the commonest respiratory symptom and considered one of the most frightening and distressing symptoms that a patient can experience (Molen, 1995). Like pain, it is subjective and shows wide variation between individuals because it includes reactions to the symptom as well as the symptom itself. Unlike pain, it usually goes untreated, and the experience is difficult for others to fully understand because 'normal' breathlessness such as running for a bus is of known duration and under control.

Breathing normally occurs subconsciously. *Breathlessness* is defined as awareness of the

Figure 7.1 Breathlessness. (From Leboeuf, C. (2000) *A Practical Approach to the Late Effects of Polio*, British Polio Fellowship, Middlesex)

intensity of breathing (Figure 7.1). *Dyspnoea* is difficult breathing occurring at a level of activity where it would not normally be expected. In practice the words breathlessness and dyspnoea tend to be used interchangeably. They should be distinguished from the objective terms:

- **tachypnoea**: rapid breathing
- **hyperpnoea**: increased ventilation in response to increased metabolism
- **hyperventilation**: ventilation in excess of metabolic requirements.

Mechanism

> ***A respiratory physiologist offering a unitary explanation for breathlessness should arouse the same suspicion as a tattooed archbishop offering a free ticket to heaven.***
>
> Campbell and Howell, 1963

Breathlessness is a private phenomenon, inaccessible through the traditional techniques of physiology. It incorporates both sensory physiology and the psychology of perception (Mahler, 1990). The mechanics and the emotional experience are inseparable.

Unlike pain, the precise stimulus that causes breathlessness has not been defined. Specialized receptors have not been found, and the region of the cerebral cortex that processes the information has not been mapped. If a person touches a hot iron, pain arises from a quantifiable thermal stimulus, whereas breathlessness arises from a variety of interlinked processes.

Three components contribute to the mechanism of breathlessness.

First, breathlessness relates to the work of breathing. Without lung disease, respiratory drive and respiratory load are balanced, e.g. when exercise increases the respiratory drive in order to increase ventilation. The breathlessness of disease occurs when either drive or load is increased and the balance is upset by mechanical abnormalities.

Examples of this mechanical component of breathlessness are:

- ↑ resistive load, e.g. ↑ airflow resistance caused by obstructive airways disease
- ↑ elastic load e.g. ↓ compliance caused by rigid chest, distended abdomen, fibrotic lungs
- ↓ energy supply, e.g. malnutrition, shock states in which perfusion to the diaphragm is impaired
- ↑ drive to breathe, e.g. parenchymal lung disorders such as pulmonary oedema, interstitial disease or pneumonia (which stimulate nerve impulses from interstitial receptors), acidosis, anaemia, thyrotoxicosis
- ↑ alveolar surface tension, e.g. pulmonary oedema, ARDS (p. 411)
- ↓ power, which reduces the ability to cope with excess drive or load, e.g. neuromuscular deficiency, inoperative diaphragm due to hyperinflated lungs, weakness, fatigue.

Respiratory muscle fatigue increases the central motor command to the respiratory muscles and magnifies the perception of effort in the same way that a suitcase feels heavier the longer it is carried.

There is often overlap in the mechanical components of breathlessness e.g. increased airflow resistance causes a stronger drive to breathe (Duranti, 1995).

Second, there are cortical and subcortical inputs, as with any subjective sensation. Exacerbating factors are uncertainty, distress, anxiety (Muers, 1993), life experiences, frustration and lack of social support (Reardon, 1994). Hence the variation between breathlessness and the

experience for each individual. Anxiety is particularly detrimental because it creates a vicious cycle of muscle tension and excess WOB.

Third, central chemoreceptors make some contribution to breathlessness by sensing a rise in P_aCO_2, mediated through pH. Reduced P_aO_2 makes little contribution to breathlessness, which explains the limited effectiveness of oxygen therapy on breathlessness (p. 119). A patient can be severely hypoxaemic without feeling short of breath, and vice versa.

These mechanisms lead to a deluge of impulses from the respiratory centres. The degree of breathlessness correlates with motor output as reflected in the pressure generated by the respiratory muscles, consciously perceived as a sense of effort (O'Donnell, 1994). Effort is central to the concept of breathlessness, as shown by a direct relationship with peak airway pressures and only an indirect relationship with elastic or resistive loads (Burdon, 1994).

The result is an effort to breathe that is not satisfied by the breath achieved, causing an inappropriate relationship between ventilatory work and total body work.

Acute asthma shows how a combination of these factors causes breathlessness:

- Bronchial irritant receptors stimulate an abnormal drive to breathe.
- Airway obstruction and inefficient breathing at high lung volumes increase workload.
- Anxiety intensifies and perpetuates the breathlessness.

Cardiorespiratory disease accounts for two-thirds of cases of breathlessness (Pratter, 1989). Other causes are neuromuscular or skeletal disorders, hyperthyroidism, anaemia, psychogenic problems, obesity and pregnancy. As with pain, the quality of breathlessness may help to identify its cause (Table 7.2).

Effects on the patient

> ***'It's the worst feeling in the world, the worst way to die, it's like smothering to death . . . to lose control of your breathing.'***
>
> DeVito, 1990

The experience of breathlessness can vary from feeling that breathing is no longer automatic, to total preoccupation and unremitting fear. Fear itself makes breathing more difficult. It is not easy for patients to communicate these feelings.

Lung disease is not blessed with high social standing. Other conditions may elicit more empathetic responses; for example, paraplegics are seen as brave, heart attacks are assumed to afflict high achievers, and a white cane elicits instant sympathy. People labelled as 'only bronchitic', however, are often elderly and depressed, they spit and wheeze, and smokers are considered to have brought it on themselves. This attitude is shared by some health workers. A degree of imaginative skill is needed when working with people who are breathless in order to identify with the experience of, for example, spending night after night in a chair unable to

Table 7.2 Some characteristics of breathlessness with different disorders; these are guidelines only, and patients vary

Disorder	Breathlessness	Other symptoms
COPD	Slow onset	Productive cough
Asthma	Episodic, on exhalation	Chest tightness, wheeze
Interstitial lung disease	Progressive, exertional	Rapid shallow breathing, dry cough
Pneumonia	Exertional	Pleuritic pain, cough
Pneumothorax	Moderate/severe	Sudden pleuritic pain
Hyperventilation	Air hunger, not relieved by rest	Symptoms of ↓ P_aCO_2
Pulmonary oedema	Positional, on inhalation	Bilateral fine crackles on auscultation
Neuromuscular	Exertional, on inhalation	Rapid breathing
Deconditioning	Heavy	
Obesity/pregnancy	Exertional	

sleep, or dreading the effort of going to the toilet, or anticipating the cruel slowness of death.

HANDLING BREATHLESS PEOPLE

Clare is a physiotherapist whose description of the breathlessness that she experienced during pneumonia indicates why some patients are not always pleased to see us:

> *'At every breath I felt: was it going to be enough? I thought life was over, even though I knew that was irrational. I didn't want to have to be polite, I didn't want the effort of please and thank you. I didn't mind how much phlegm was there, it could just stay there. The thought of a physio coming near me made me feel even more ill.'*

Breathless people have lost control of their most basic physiological requirement. They therefore need some control of their treatment. They need to take their time and not be expected to talk unless they want to. They need acknowledgement of the reality of their experience, not empty phrases like 'Don't be frightened' or 'Try to get control'.

Communication should be clear because anxiety increases oxygen consumption. With severely breathless people, questions should require only a yes or no answer. It is best not to pretend to understand if we do not, and patients should not have their sentences finished for them.

With long-term breathless patients, as with anyone who is chronically disabled, it is important to respect their knowledge. They know more than we do about the experience of their disease and we learn much by listening to how they prefer to be handled.

The physical handling of acutely breathless patients requires maximum support, minimum speed and a rest between each manoeuvre. When patients are getting their breath back after moving, they should not be asked questions.

SLEEP AND REST

> *'There's no peace, no let up with this thing, you can't even escape when you go to bed, it's with you 24 hours a day.'*
>
> Patient quoted by Williams, 1993

The only treatment for fatigue is rest. This can be achieved most satisfactorily by sleep. One of the cruel ironies of breathlessness is its effect on sleep. Fragmentation of sleep impairs respiratory performance, blunts response to hypercapnia and hypoxaemia, reduces inspiratory muscle endurance (Neilly, 1992) and is wretched. Many patients say that sleep is what they need most in order to recover.

Sleep deprivation in respiratory patients is due to breathlessness, coughing and anxiety, aggravated in hospital by noise, an unfamiliar environment and being woken for nebulizers. Physiotherapists should avoid waking patients unnecessarily, ensure that their treatment does not cause excess fatigue, and contribute to the teamwork required to allow adequate sleep. It is a continuing puzzle that there is so little emphasis in the health care system on facilitating the healing effects of sleep.

POSITIONING

Many breathless people automatically assume a posture that eases their breathing, but others need advice to find the position that best facilitates their inspiratory muscles. Patients with a flat diaphragm may benefit from positions that use pressure from the abdominal contents to dome the muscle and provide some stretch to its fibres so that it can work with greater efficiency. The arms are best supported, to optimize accessory muscle function, but without tension or active fixation. Positions to facilitate efficient breathing in breathless people include:

- high side-lying (Figure 7.2).
- sitting upright in a chair with supported arms; for many patients, it is easier to breathe in this position than in bed. Some like to lean back for support, others prefer

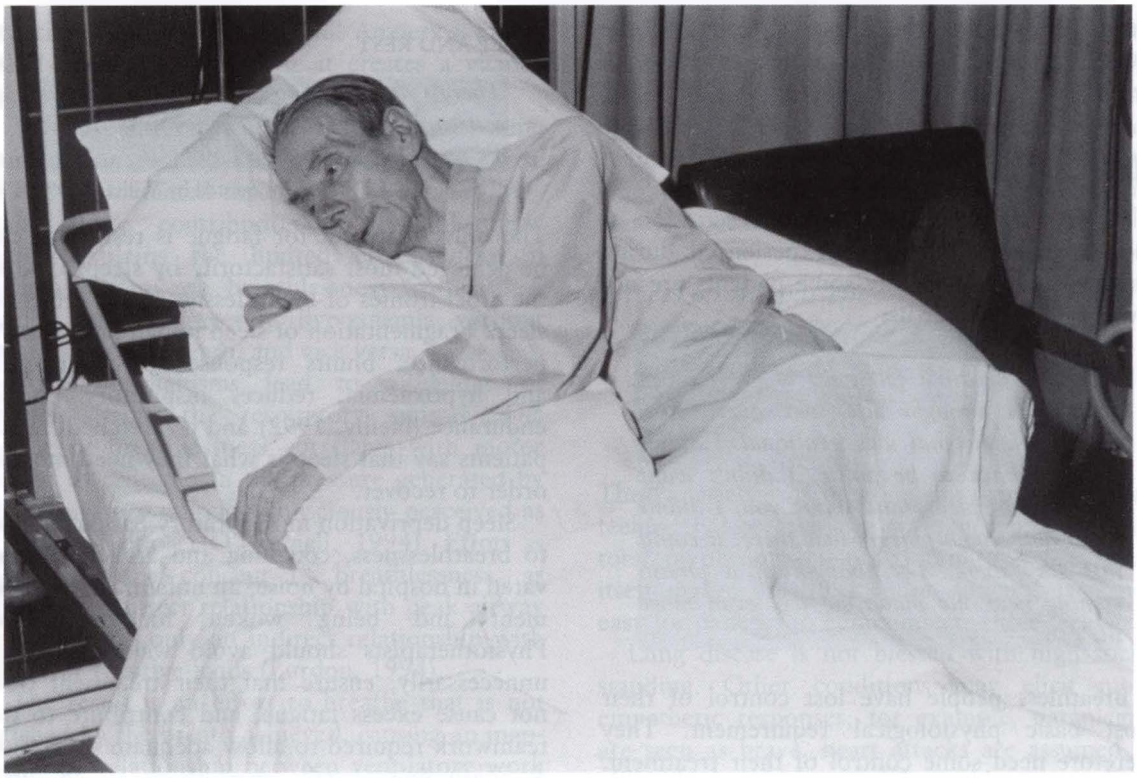


Figure 7.2 High side-lying to minimize the work of breathing in a breathless patient. The head rest is relatively low to prevent the patient slipping down the bed and to avoid kinking the spine.

to lean slightly forward to put some stretch on the diaphragm

- sitting leaning forward from the waist, arms resting on pillows on a table, feet on the floor
- standing relaxed, leaning forwards with arms resting on a support such as a window sill.
- standing relaxed, leaning back against a wall with legs slightly apart.
- standing relaxed leaning sideways against a wall, arms in pockets if support is needed for the accessory muscles.
- occasionally, lying flat is beneficial because of pressure from the abdominal contents against the diaphragm. A few patients even find a slight head-down tip helpful.

Individuals should experiment with different positions. Some find the forward-lean positions claustrophobic, others unpredictably desaturate in different positions. Oximetry is useful as biofeedback. Some severely distressed people are relieved by being held and rocked. The combination of support and rhythmic movement soothes and relaxes them. Neck massage may help.

If breathlessness is due to pulmonary oedema, the upright supported sitting position is preferred because hydrostatic pressure is more relevant than diaphragmatic mechanics.

RELAXATION

Relaxation is facilitated by positioning, sensitive handling and the provision of information to reduce anxiety. Deeper relaxation may be achieved by learning a relaxation technique. Patients should be warm, comfortable and have

Box 7.1 Patient handout on relaxation

Silence the phone, check that room temperature and ventilation are right for you.
Clear your chest if necessary to prevent disturbance by coughing.
Take up your preferred position.
Close your eyes. Notice any sounds, then release them from your attention.
Become aware of any thoughts, notice them, then let them go.
Imagine that you are in a place that you find peaceful, such as a beach or sunny field.
Breathe abdominally if comfortable. Let the stomach muscles soften, as if taking off a tight belt.
Feel the soft sensation of your abdomen rising and falling. Allow the breath to flow peacefully throughout your body.
Focus your mind down your arms to your hands, allow the fingers to soften and arms to feel heavy.
Focus your attention down your legs, let them feel heavy. (If sitting) feel your feet flat on the floor.
Feel your head becoming heavier, let the muscles of your face soften, let your jaw loosen and slacken, teeth parted.
Feel your neck a little longer and your shoulders heavier.
Feel the heaviness of your body on the bed or chair. Feel your body melt as if you are meat without bone, feel warm energy spreading through your body.
Feel the rhythm and flow of your breathing, allow your body to relax more with each breath out.
Enjoy the sensation as long as you like.
When you are ready: slowly, in your own time, become aware of the sounds in the room once more, begin to move gently, open your eyes, stretch.
When you are ready: get up very slowly.
Try to maintain the calmness for a while.

adequate fresh air. The physiotherapist's bleep should be re-routed. A technique should be chosen, such as the physiological method (Payne 1995, p. 77), which does not entail breath-holding or strong muscle contraction. Other methods can be used that incorporate breathing itself, while maintaining rhythmic breathing and an adequate breathing rate. Some suggestions are given in Box 7.1.

Patients should be reassured that relaxation is not difficult to learn, that there is no right or wrong way of doing it and that they can follow what feels right for them. Although it does not matter if they fall asleep, the aim is to stay awake to enjoy the experience of alert tranquillity so that they can re-create it as desired and integrate aspects of it into their daily activities.

The effects of this hypometabolic conscious state are:

- ↓ breathlessness, anxiety, airway obstruction (Gift, 1992)
- ↓ respiratory rate, oxygen consumption, heart rate and BP (Hodgkin and Petty, 1987).

These benefits are maintained for varying lengths of time, but Renfroe (1988) found that the reduced respiratory rate was sustained the longest. Many patients find that relaxation improves their breathing pattern without direct breathing re-education.

BREATHING RE-EDUCATION

If patients have not adopted an efficient breathing pattern with the above measures, they may benefit from smoothing their breathing into

a rhythmic pattern or they may find more structured breathing re-education helpful. The aims are to reduce WOB and give patients confidence in their ability to control breathless attacks.

When intervening in a person's breathing pattern, a minimalist approach is advised. Compensatory mechanisms such as dynamic hyperinflation should not be interfered with mindlessly. Even if a patient's breathing appears unnatural, this may be the optimum for an individual's pathology; for example, Hoover's sign (p. 69) may be unavoidable and reliance on shoulder girdle muscles may be making the best of a bad job. If, however, breathing is irregular, paradoxical or unnecessarily tense, it is likely to respond to re-education to improve efficiency.

Overview

The following steps are best taken one at a time. Close observation will then determine whether this step has been helpful and/or if the next step should be initiated.

1. The position is chosen by the patient, but the physiotherapist might suggest sitting upright in a chair, forward-lean-sitting or other resting position (p. 169).
2. Awareness of breathing is encouraged by bringing the patient's attention to their breathing pattern. Are they breathing apically, abdominally, using pursed lips and prolonged expiration, breathing through nose or mouth?
3. Relaxation can be a full body technique or simply raising awareness of tense areas, e.g. jaw or hands, and advising on localized relaxation. Patients will not be able to relax the shoulder girdle if they depend on accessory muscles to breathe. The physiotherapist's own relaxed posture, calm voice and steady breathing pattern will help reduce the patient's tension.
4. Relaxed breathing can be facilitated by a modified yoga technique: patients sit with their feet flat on the floor and visualize that they are breathing air 'in through your head and out through your feet into the floor'. This is not exactly anatomical but almost invariably facilitates relaxation.
5. If patients are not yet breathing rhythmically, they can be encouraged directly, by demonstration and by suggestions to 'breathe smoothly with a nice steady rhythm, in your own time'. A mirror may help.
6. Patients may then be able to develop gently an abdominal pattern of breathing, and/or raise the resting lung volume, as described on the next page.
7. Relaxation is rechecked.
8. Praise is given liberally!

During this sequence, breathing usually becomes slower and deeper naturally. Shallow breathing wastes energy because of ventilating dead space, and rapid breathing wastes energy because of turbulence. However, encouraging slow deep breathing beyond that developed naturally tends to be counterproductive. Breaths that are too deep are working against elastic recoil and can increase the WOB, a twice-normal tidal volume quadrupling elastic workload (Haas and Axen, 1991, p. 17). The following points clarify this concept for different conditions:

- Rapid shallow breathing adopted by those with restrictive lung disease is logical because of high elastic recoil and low lung compliance (Mador, 1991), i.e. the breathing pattern is usually optimal and should rarely be changed.
- Rapid shallow breathing in 'pink puffer' (p. 71) patients should not be disturbed if it is steady, but will need correcting if it is irregular.
- Hypercapnic 'blue bloater' patients are conserving energy wisely and their breathing pattern may be best left undisturbed (Roussos 1996).

Slow, deep breathing may benefit people with moderate obstructive lung disease but this is best encouraged indirectly by the methods described above; if imposed directly it can disrupt the breathing pattern (Faling, 1986) and tire the inspiratory muscles (Bégin, 1991).

Pursed lip breathing is often adopted voluntarily by breathless people because it can relieve breathlessness by acting as a form of PEP (p. 189) to prevent airway closure. It may be inefficient (Spahija, 1996), but can help some patients subjectively in time of need.

Abdominal breathing

Relaxation may be facilitated by abdominal breathing, as described on page 154, but without progression to side-lying or increased depth of breathing. Abdominal breathing may visibly break through a patient's wall of tension, but for others it can be counterproductive, especially if they have severe disease with a finely balanced breathing pattern that is readily upset.

Positive outcomes include:

- ↓ BP (Fried, 1993, p. 177)
- ↓ breathlessness (Breslin *et al.*, 1990)
- ↑ inspiratory muscle strength (McConnochie and Chatham, 1991).

Negative outcomes include:

- disruption of breathing pattern (Gosselink, 1995)
- ↑ WOB (Vitacca, 1998).

Patients vary greatly in how they respond.

Innocenti technique

Forced expiration consumes excess energy and does not improve expiratory airflow (Tobin, 1988). If patients continue to use forced expiration despite the previous manoeuvres, they can be helped by a simple technique that raises resting lung volume above the level at which abdominal recruitment occurs (Innocenti, 1966). This technique acts like CPAP to hold airways open and occurs naturally with exercise training (Pellegrino *et al.*, 1999). It helps to prevent airways shutdown, consuming less energy than pursed lip breathing. Reported outcomes are improved P_aO_2, exercise tolerance and quality of life (Innocenti, 1997).

Patients should not change their rate or depth of breathing. They simply start inhalation just before the point at which visible abdominal muscle contraction begins, i.e. when active expiration takes over from passive expiration. The following steps are suggested:

- positioning, relaxation and rhythmic breathing as described on the previous page
- observation of the patient's breathing pattern
- at each breath, instruction to the patient to inhale just before abdominal muscle recruitment, then allow a smooth transition from inspiration to expiration
- practice in this, at first with the physiotherapist's voice, then without.

Patients should avoid holding their breath or making other changes in their breathing pattern. Any rise in the JVP, or a flicker of the abdominal muscles, indicates active expiration, in which case the technique must be modified. Ongoing reinforcement is needed, but it is a pleasure to see the relief that it can then bring.

Much encouragement is needed to alter a familiar breathing pattern, but the earlier in the disease process these techniques are learnt, the more easily patients can incorporate them into their lifestyle. There is no clear evidence that a voluntary act can become automatic but, if repeated regularly, learning can occur by a change in the process underpinning its control (Gallego and Perruchet, 1991). If not, practice can enable it to be used when required.

TIPS ON REDUCING BREATHLESSNESS

Patients can be advised that breathlessness is affected by talking, eating, posture and muscle tension. Many know this but it is difficult for some to adapt to, and reinforcement by regular reminders helps raise awareness.

Avoidance of breath-holding

A habit that is common in tense patients is breath-holding, which increases tension and breathlessness. Breath-holding can be observed when patients are concentrating, making an effort or listening to advice. If this is pointed out to them at each opportunity, with advice to

'keep the rhythm going', they are often able to bring it under control. Patients find this habit easier to change than altering a lifetime of rapid talking or body tension. Physiotherapist, patient and family members can compete to be the first to notice each instance of breath-holding.

Desensitization to breathlessness

To reduce the fear that inhibits activity, patients can learn to desensitize themselves to breathlessness. First and foremost, they are told that breathlessness itself is not harmful. This is a revelation to some patients, who feel that it is causing damage (Bellamy, 1997) and that every breathless attack further progresses their disease. They are reminded that smoking, lack of oxygen and the disease process are harmful, but breathlessness is a symptom and not damaging in itself.

Once this is understood, patients are free to attempt activities that increase breathlessness in a way that they control, and then gently regain their own breath. Patient and physiotherapist start by walking together, the patient being reminded to maintain relaxed rhythmic movement, relaxed rhythmic breathing, a good posture and to stop to get their breath back whenever they want. Patients who are deconditioned and fearful might simply walk round the bed and sit down. They are then praised for their success in increasing and controlling their breathlessness and encouraged to switch their attitude from fear of breathlessness to confidence in their own ability to control it.

Desensitization is progressed by the patient being exposed to graduated increases in breathlessness, then integrating this with other activities, using the same rhythmic breathing and steady movement. For those who rush at activities, a slower pace is advised.

Other tips

A fan reduces breathlessness by influencing receptors in the trigeminal nerve distribution that provides information to the sensory cortex (Manning, 1995). Other tips are mechanical vibration over the chest wall (Sibuya *et al.*, 1994) and acupressure or self-acupressure to any of the breathless points (p. 84).

PACING

Once breathing is controlled, through either direct breathing re-education or other techniques, it can be incorporated progressively into standing, eating, talking, walking, stair-climbing and ADL. Pacing assists this process of integration by allowing patients to maintain steadiness and control during activities. Walking alongside patients, steadily and sometimes more slowly than they are used to, teaches patients to achieve control and understand the relevance of energy conservation. Recreating and managing situations that typically increase breathlessness for each individual will improve confidence.

OTHER RESPIRATORY PROBLEMS

If a breathless person has a problem of reduced lung volume (Chapter 6), e.g. postoperative atelectasis, positioning is the first-line treatment because it is least disruptive to the breathing pattern. So long as patients are relaxed and pain-free, lung expansion will be facilitated as they get their breath back after turning to the appropriate position.

If further measures such as deep breathing are necessary, the breathing rate should be maintained throughout. When asked to take a deep breath, breathless patients sometimes respond by holding their breath instead. This can be avoided by advising them to 'keep breathing in and out', or telling them when to breathe in and out, until they find their own rhythm. No more than two deep breaths should be taken at a time, then they are advised to breathe comfortably. Breathing rate and pattern are observed while they return to normal tidal volume.

If a breathless person has a problem of sputum retention (Chapter 8), vibrations may sometimes disturb the breathing pattern. Percussion may be better tolerated and can even be relaxing if a slow, rhythmic technique is used. The head-down postural drainage position is

usually contraindicated for breathless people. Very occasionally it is beneficial for emphysematous patients because their flat diaphragm is pushed into a more functional dome shape. However, this must be done slowly and only if comfortable. Oximetry can be reassuring for both patient and clinician.

MECHANICAL AIDS

> ***'From our very first night she made a quite startling difference to my life. Just one night converted me to the joys and thrills of home ventilation.'***
>
> Brooks, 1990

Non-invasive ventilation (NIV) provides inspiratory muscle rest for people who are burdened with ventilatory failure due to excessive WOB. This can be due to obstructive or restrictive disorder in either the acute or chronic state.

NIV delivers a predetermined volume or pressure either automatically or, more usually, in response to patient effort. Positive pressure is delivered via mask or mouthpiece, or less commonly via the natural airway using negative pressure. Compared to invasive mechanical ventilation, NIV carries less risk of infection (Guérin, 1997), is more comfortable, easier for speech and swallowing, safer and more convenient (Bach, 1994). Intensive care is not required and patients can participate in their own management. NIV does not protect the airway and provides no direct access to the trachea for suction.

Nasal mask positive pressure ventilation is the commonest system (Figure 7.3), but other ventilators are discussed at the end of this section.

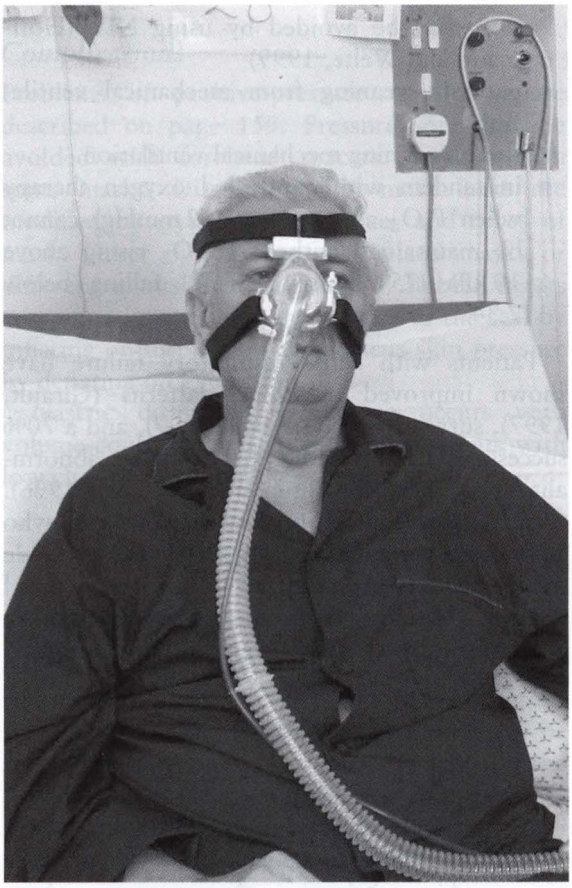


Figure 7.3 Non-invasive ventilation by nasal mask (Medicaid).

Effects for people with acute disorders

For acutely ill patients, NIV unloads the inspiratory muscles, reduces breathlessness and corrects respiratory acidosis. In COPD, this has resulted in reduced need for intubation, ICU length of stay and deaths (Wysocki, 1999).

For patients unable to tolerate oxygen therapy without loss of respiratory drive, better outcomes are shown with NIV than with the respiratory stimulant doxapram (Angus, 1996).

Patient handling skills are required to talk frightened patients through the process and encourage them to allow the machine to do its job.

Patients who most benefit include those with:

- exacerbation of COPD or CF with rising P_aCO_2 and falling pH
- acute asthma, to reduce the effort of maintaining dynamic hyperinflation
- acute restrictive disorders such as pneumonia, postoperative atelectasis or ARDS
- severe pulmonary oedema, for which intuba-

tion may be avoided by using NIV (Hoffmann and Welte, 1999)
- patients weaning from mechanical ventilation
- those declining mechanical ventilation.
- in tandem with controlled oxygen therapy when P_aO_2 above 7 kPa (52 mmHg) cannot be maintained without P_aCO_2 rising above 10 kPa (75 mmHg) and pH falling below 7.34

Patients with acute respiratory failure have shown improved breathing patterns (Girault, 1997), survival rates (Keenan, 1997), and a 70% success rate in correcting gas exchange abnormalities and avoiding intubation (Meduri, 1996). Outcomes are most positive in patients who have some pump failure, e.g. inspiratory muscle fatigue, rather than solely airway or parenchymal disease. High concentrations of oxygen can be entrained if necessary, even with hypercapnic COPD patients if there is a safety backup of mandatory breaths irrespective of respiratory drive.

NIV is contraindicated in patients with some unstable medical conditions such as shock, arrhythmias or upper GI bleeding. Before initiating NIV, a decision must be reached with the medical team and patient about whether intubation or palliation is appropriate if NIV fails. Success is likely if pH and P_aCO_2 respond within an hour (Figure 7.4).

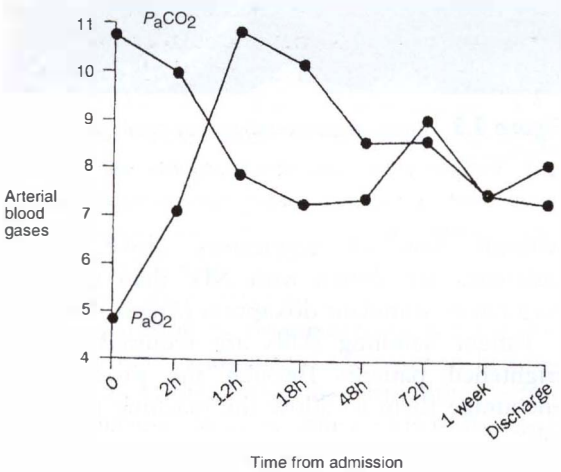


Figure 7.4 Arterial blood gases of a 60-year-old woman with acute COPD, ventilatory failure and respiratory acidosis. On admission, S_aO_2 was 57% and pH 7.21. CPAP raised S_aO_2 to 82% but hypercapnia and acidosis persisted. NIV corrected acidosis to pH 7.39 and improved hypercapnia within an hour. (From Keilty, S. E. J. and Moxham, J. (1995) Noninvasive ventilation in acute-on-chronic airways disease. *Hospital Update*, **21**, 165–171).

Effects for people with chronic disorders

> ***'For the first time in months I felt reasonably clear-headed, my thinking felt keener, I no longer fell asleep in mid-sentence, my headaches disappeared. ... Over the following months, as my strength slowly returned, my posture and balance noticeably improved.'***
>
> Brooks 1990

There is often a reluctance to take full advantage of home ventilation because:

- ventilator use has traditionally been associated with institutionalization
- doctors tend to assume that ventilator users have a poor quality of life and the patient's view is not always sought.

NIV should be considered if there is daytime hypercapnia and symptoms of nocturnal hypoventilation (morning headache, daytime sleepiness, breathlessness and often anorexia), confirmed by a sleep study. Patient and family need to be motivated to manage a ventilator at home, after acclimatization in hospital.

Home NIV has shown an 87% positive response (Goldstein, 1995), with sustained improvement in gas exchange and reduced hospitalization (Leger, 1994). The mechanisms for improvement are thought to relate to respiratory control, i.e. increased response to hypercapnia by lowering bicarbonate in the cerebrospinal fluid in response to reduced P_aCO_2, and reversal of hypoxic depression of the respiratory centres (Shneerson, 1996b). Relief from symptoms of

hypoventilation is rapid, especially improved quality of sleep (Shneerson, 1996b), and patients often fall asleep as soon as the machine is set up.

For people with chronic disorders, NIV has shown:

- early hospital discharge with cost savings of 200% a year (Bach, 1994)
- improved arterial blood gases (ABGs), reversed pulmonary hypertension and cor pulmonale (Simonds, 1998)
- tolerance of oxygen therapy when used at night and during daytime naps (Sivasothy *et al.*, 1998)
- increased vital capacity (Pehrsson, 1994)
- prolonged life (Muir, 1993).

Patients use the ventilator until ABGs are optimal, then regularly according to symptoms. Patients who benefit include:

- those with advanced hypercapnic COPD, scoliosis, neuromuscular disease, especially those with daytime P_aCO_2 above 6 kPa (45 mmHg)
- those awaiting transplantation
- those wanting to come off invasive ventilation.

Patients have to fulfil the following criteria:

- $P_aO_2 > 8.0$ kPa (60 mmHg)
- glottic control adequate to prevent recurrent aspiration
- maintenance of unassisted or assisted peak cough flows greater than 3 L/sec (Bach and Haas, 1996, p. 373).

Most patients require at least 6 hours of ventilatory support per night, but improvements have been found with periods of rest from 8 hours a week to 10 hours a night (Axen, 1991). Nocturnal ventilation is used when possible because this compensates for loss of accessory muscle activity during sleep, but daytime NIV can be effective for patients who prefer this (Schönhofer *et al.*, 1997). Patients may well be up and about in the daytime, but live in a precarious balance that leaves them unable to respond to increased energy demand such as a chest infection.

Complications

Problems of positive-pressure ventilation are described on page 159. Pressure sores can be avoided with forehead spacers supplied with some masks. Discomfort and mask leaks are modified by trying different sizes and types of mask. Other options are customized masks or different masks used in rotation. Bubble masks contain an inner lining that improves the seal by inflating on inspiration and lessens skin pressure by deflating on expiration.

Gastric distension sometimes occurs with volume-controlled machines or in patients with low chest wall compliance. Options are:

- adopting the left-side-lying position
- using the lowest effective pressures
- waiting to see if it eases with time
- trying a different ventilator.

Nasal dryness may be helped by nasal drops. Mouth dryness usually responds to reducing air leaks through the mouth. Skin irritation may be solved by daily washing of mask, spacer and the patient's skin, and using minimal strap tension. If the straps feel too tight, a smaller mask may allow them to be loosened, or a skull cap can be used.

Patients may pull off the mask if they are suffering hypoxic confusion, anxiety or administration of doxapram. They need explanations and observation.

Equipment

Advantages of pressure-controlled (p. 343) machines are the comfort of a limited peak pressure, reduced risk of pneumothorax in advanced emphysema and compensation for leaks. Volume-controlled machines are more suited to people with high or fluctuating airway resistance or lung compliance. However, there are differences in the 'feel' of individual machines, and patients have their own preferences.

Machines should have a sensitive trigger, short response time, variable flow, be capable of delivering an adequate tidal volume (V_T) or

pressure, and be quiet and portable. Some machines have a PEEP (p. 351) option which reduces the risk of atelectasis in neuromuscular disease and gas trapping in patients with hyperinflated chests. Occasionally PEEP may reduce cardiac output and oxygen delivery in patients with obstructive disease (Ambrosino *et al.*, 1993).

Nasal masks should be comfortable and small enough to fit from half-way down the bridge of the nose to just below the nares. Full face masks double deadspace and increase the likelihood of complications, but they are suited to people who mouth-breathe. For patients with glasses or those who find nasal masks claustrophobic, nasal 'pillows' avoid contact with the bridge of the nose or cheeks. Mouthpieces are best if pressures above 25 cmH_2O are needed, or for mobility when using a wheelchair. Some ventilator-dependent people prefer a mouthpiece in the daytime and lipseal device or customized mask at night.

A bacterial filter or heat-moisture exchanger can be added to the circuit, after checking the handbook to ensure that it does not upset the pressures. Patients requiring humidification will need a high-efficiency humidifier.

Technique

For acute patients, NIV should be set up at an early stage when the pH falls below 7.35 and respiratory rate (RR) rises above 30/min (Baldwin, 1997). Patients with chronic disease must be fully rehabilitated, and a maintenance service operational. For all patients, medical management must be optimal.

One of the following can be chosen:

- a spontaneous option, which superimposes inspiratory and, usually, expiratory pressures on the patient's own breathing
- a spontaneous/timed option, which adds mandatory breaths if the patient does not breathe after a set time interval
- a timed option, usually used by the physician, which is fully controlled ventilation for patients who are unable to breathe at all.

For the spontaneous/timed option, RR is set at 2–5 less than the patient's spontaneous rate.

Parameters are set according to ABGs and comfort. Alternatives to ABGs are capillary blood gases or transcutaneous monitoring (Janssens, 1998). The machine should match the patient rather than the patient being obliged to conform to the machine. If inspiratory and expiratory times are used, they are set to synchronize with chest wall movement. For volume-controlled machines, CO_2 can be blown off by increasing V_T or I:E ratio in order to raise minute volume. For pressure-controlled machines, a typical starting pressure is 10 cmH_2O, building up to 15–20 cmH_2O.

The machine is turned on before applying the mask. Anxiety is minimized by allowing patients to feel the air blowing against their hand. When strapping the mask on, the top straps are tightened first, and straps need to be equally tight on both sides. If oxygen is added, an oxygen analyser and oximeter are used as for CPAP. For machines with pressure alarms, the low pressure alarm is set 8–10 cmH_2O below the lowest working pressure, and the upper pressure limit is set at about 40 cmH_2O. Exhalation ports are designed to exhaust CO_2 and must not be blocked, except initially to test that flow is present. They should be directed away from the patient's face.

Patients are asked to keep their mouths shut. Some may voluntarily keep it closed once they feel relief but others are committed mouth-breathers. If they do not want a chin strap or collar, side-lying with a pillow supporting the chin may help prevent mouth leaks. Small leaks may be acceptable so long as they do not interfere with triggering into inspiration or cycling into expiration, and so long as V_T and ABGs are adequate. Patients may find that slight neck extension helps to optimize airflow. The mask should be removed before turning the machine off. If a full face mask is used, it is best for the patient to avoid eating or drinking 2 hours beforehand.

Improvement in RR and pH in the first hour is a predictor of success. P_aCO_2 should not be

forced down too quickly if there is high bicarbonate, otherwise metabolic alkalosis may supervene. Acutely ill patients may be given continuous NIV for 24 hours, removing the mask only to talk, drink and eat, or they may use it for a couple of hours morning and afternoon, plus overnight. Intermittent support may then be appropriate, with longer periods of spontaneous breathing as weaning progresses. The mask is removed for administration of nebulizers, which should be delivered through normal nebulizer devices.

Modes

Continuous positive airways pressure

CPAP (p.156) does not support ventilation directly, and respiratory muscle unloading is limited (Wysocki, 1999). However, in hyperinflation conditions, CPAP takes over the work of sustained muscle activity during inspiration and keeps the airways open to allow greater gas emptying during expiration (Greenwald, 1993). Pressures of 4–5 cmH_2O may be adequate to counteract the inspiratory threshold load caused by gas trapping (O'Donnell, 1994). The instructions on page 158 are followed but, instead of the goal being to increase S_aO_2, pressures are titrated to the individual's comfort to ensure that hyperinflation is not increased.

Many people with an exacerbation of COPD find CPAP frightening and claustrophobic, and outcomes are patchy:

- even when ameliorating the WOB induced by hyperinflation, CPAP does not necessarily ease breathlessness (Fessler, 1995)
- desaturation can occur (Elliott *et al.*, 1994)
- CPAP is less successful than inspiratory pressure support (Gibson, 1996) or bilevel positive pressure.
- it is contraindicated in TypeII respiratory failure)p. 117) because of the risk of CO_2 retention

Inspiratory pressure support

Positive pressure on inspiration is provided by IPPB (p. 159) or a multimode machine. This can reduce WOB so long as the patient is relaxed and does not attempt to assist or resist. For the Bird, the instructions on page 161 are followed, with the following modifications:

- the most comfortable resting position is used (p. 169)
- the flow is turned up to match breathlessness
- the pressure need not be progressively increased because the aim is not to increase lung volume but to ease WOB.

Treatment by the Bird may be needed two-hourly in the acute phase, unless the patient is asleep. Very occasionally an exhausted patient is unable to initiate a pressure-triggered breath, in which case a flow-triggered machine is required (see below).

Bilevel positive airway pressure

'BiPAP' is the trade name for a specific machine but, like Biro and Hoover, the word has become synonymous with the generic and the term BiPAP is commonly used to describe the BiPAP mode on any machine. This mode delivers continuous positive pressure with independent control of inspiratory and expiratory pressures. Bilevel pressures are more comfortable than CPAP and more flexible than IPPB. BiPAP is the non-invasive equivalent of pressure support with PEEP (Chapter 13).

Inspiratory positive airway pressure is usually started at about 10–14 cmH_2O and increased in increments of 2 cmH_2O until there is least effort during inspiration and optimum ABGs. Expiratory positive airway pressure is started at 2–4 cmH_2O and increased to achieve optimum comfort and S_aO_2, but levels over 6 cmH_2O tend to impose an unacceptable expiratory load and interfere with sleep. A minimum 2 cmH_2O is required to prevent CO_2 building up in the circuit. A plateau valve extubation port may be available to prevent CO_2 retention. If inspiratory and expiratory pressures are equal, CPAP is delivered.

BiPAP can improve sleep, reduce breathlessness and increase exercise capacity (Renston, 1994). For acute patients, the need for intubation was reduced to zero in one study of severe

exacerbations of COPD, using an average inspiratory pressure of 14 and expiratory pressure of 4 (Confalonieri, 1994).

BiPAP is usually flow-triggered, pressure-controlled and flow-cycled (p. 203). It adjusts to compensate for minor leaks, which do not have to be eliminated by ruthless tightening of headgear.

To summarize:

- CPAP may be useful for hyperinflation or type I respiratory failure.
- IPPB is useful when the problem is exhaustion, hypercapnia or type II respiratory failure.
- BiPAP is ideal and can be individually titrated to each of these problems.

Non-invasive ventilation is now well-established as an evidence-based practice that can reduce morbidity and mortality, but it is still the exception rather than the rule in many UK hospitals. Physiotherapists are well placed to initiate and co-ordinate the teamwork required to set up a service. Whether it is doctor-led or physiotherapist-led, all relevant staff need to be trained, including the medical team to provide timely ABGs, and on-call physiotherapists to understand the teamwork, indications and practicalities.

Negative pressure ventilation

Negative pressure is applied externally via a machine that encloses part of the patient's body, sucking air into the lungs through the patient's natural airway. Negative pressure ventilators are more time-consuming and difficult to adjust than positive pressure ventilators, but they suit some individuals, especially those with:

- nocturnal hypoventilation and restrictive disorders such as post-polio syndrome, who require long-term support
- severe right heart overload or haemodynamic instability
- hypercapnic coma (Corrado, 1996).

Advantages are:

- avoidance of the complications of positive pressure and mask
- normal physiological distribution of ventilation
- for patients unable to bring their hands to their face to manoeuvre a nasal mask, easier communication.

Disadvantages are:

- awkward machinery
- risk of sleep apnoea due to upper airway collapse (Thomson, 1997), especially in ventilator-triggered modes.

The *tank* ventilator encloses all the patient except the head in an airtight iron lung. Disadvantages are size, noise, discomfort from the neck seal, immobility, fear of suffocation and inaccessibility of the patient. *Jackets* and the rigid *cuirass*, which apply negative pressure over the chest and abdomen, are less efficient but more convenient (Bach, 1994), but may restrict sleep or have air leaks.

Patients in tanks may require physiotherapy because of immobility and ineffective cough. If secretions are a problem, vibrations and percussion through the portholes may be helpful. Coughing is assisted manually (p. 203). Treatment in prone requires a person to sit by the patient's head to watch his or her colour. If the patient vomits, pressure must be equalized immediately by opening a porthole because of the danger of aspiration.

Motivated patients can be taught to master the art of glossopharyngeal breathing (Bach, 1994). Using the lips, soft palate and tongue, 6–9 mouthfuls of 60–100 mL of air are collected in the mouth and throat, then gulped into the lungs using the tongue, pharynx and larynx, creating a respectable V_T. Success provides the following advantages for those with no measurable vital capacity:

- the independence of a few hours' ventilator-free time
- the ability to call for help
- a safety margin in case of ventilator failure.

Using a mirror and an upright or other

symmetrical position, training requires much concentration, with short daily sessions to avoid fatigue. A video is available (Appendix C).

Other ventilators

High-frequency *oscillators* deliver high-flow bursts of gas either through a mouthpiece or externally by generating an oscillating pressure through a cuirass device such as the Hayek, which conforms to the surface of the body from neck to hips (Hardinge, 1995). It is expensive, comfortable and reduces WOB by overriding spontaneous ventilation. It may assist clearance of secretions.

The *rocking bed* uses gravity-assisted displacement of abdominal contents to augment diaphragm excursion, usually for people with isolated bilateral diaphragm weakness. It is most effective and comfortable with some degree of head elevation throughout the rocking cycle. For immobile patients, the variation in pressure reduces the risk of skin breakdown.

The *pneumobelt* is used in sitting and standing only. For expiration, it inflates a bladder at 50 cmH_2O around the abdomen to push up the diaphragm. For passive inspiration, it deflates to allow diaphragmatic descent.

Tracheostomy ventilation

> ***The kind of life lived by a patient under conditions of vigorous response to a challenge is infinitely preferable to a crunching, desperate winding down.***
>
> Cousins, 1981

Long-term invasive ventilation may be needed for some people with bulbar weakness or ventilator-dependency for over 16 hours/day, such as those with high spinal cord lesions, or occasionally for those with COPD who have been unable to wean from mechanical ventilation (Muir *et al.*, 1994). A cuffed tracheostomy tube is necessary if airtight ventilation is required. Uncuffed or deflated cuffs can be used for the following:

- to allow spontaneous breathing in case of ventilator failure
- with full mechanical ventilation if the machine is volume cycled and can deliver three times the volume of air that would be required using a cuffed tube
- to permit speech, if there is a speaking valve attachment.

Invasive ventilation can be managed at home if the patient or carer is educated to deflate and inflate the cuff, change and clean the tracheostomy tube and suction aseptically.

OUTCOMES

Reduced work of breathing can be judged by the following:

- ↓ breathlessness (see p. 216 for assessment)
- ↓ fatigue
- ↓ RR
- more synchronous breathing pattern
- ↑ exercise tolerance and ADL, if the limiting factor for these is breathlessness.

MINI CASE STUDY: MS IU

You are called in to see a 69-year-old woman with an exacerbation of COPD. Identify her problems from the selected details and answer the questions.

Background

SH: lives alone, manages stairs, independent, supportive son.
Medical notes: not for IPPV.

Subjective

Can't breathe.
Dry mouth.

Objective

P_aO_2 9.5, P_aCO_2 11.3, pH 7.21, HCO_3^- 32, BE 1.4.
S_aO_2 85% but varying.
On 24% dry oxygen.
Temperature 36°C.
On infusion of salbutamol and doxapram.

Propped up in bed.
Rapid shallow breathing.
Body shaking continuously.
Difficulty speaking.
Appears fearful ++.

Questions

1. Is the goal of medical treatment palliative or curative?
2. Analysis?
3. Problems?
4. Goals?
5. Plan?

IPPV = intermittent positive pressure ventilation.

RESPONSE TO MINI CASE STUDY

1. Medical treatment

The fact that Ms IU is not for invasive ventilation suggests that palliation is the goal. However, the doxapram infusion is not palliative.

2. Analysis

Teamwork is required before decisions can be made on goals of physiotherapy.
Patient is severely acidotic, but is normally independent. However, decisions on quality of life are best made by the patient rather than the health team.
Auscultation and X-ray are irrelevant at present because they would not supply any further information that would direct treatment.

3. Problems

Type II ventilatory failure and respiratory acidosis.
Distress.

4. Goals

Support ventilation non-invasively. This could fulfil palliative or curative criteria.
Liaise with medical team about goals of treatment and whether patient has given previous opinion on DNR status.

5. Plan

Explanations to patient, including information on the fact that doxapram causes shaking, which might reduce her fear.
BiPAP.
Humidify oxygen.
Liaise with nursing staff or relatives on mouth care and assistance with oral fluids.
Liaise with medical staff over common goals.
Fan, positioning, other techniques to reduce WOB.

DNR = do not resuscitate.

LITERATURE APPRAISAL

Comment on the connection between the following two statements. Does the conclusion fit?

> *[P]erceived quality of life appears to be linked with peripheral muscle force in COPD patients....*
>
> *Consequently, peripheral muscle training may be an important tool in improving quality of life in COPD patients.*
>
> *Eur. Respir. J.* 1996; 9(23): 144S

RESPONSE TO LITERATURE APPRAISAL

Chickens and eggs. The second statement may be a correct conclusion, but the first statement does not prove that weak peripheral muscles cause impaired quality of life. Peripheral muscles may weaken as a result of exercise limitation or malnutrition. Both are common in COPD, as is impaired quality of life.

RECOMMENDED READING

Adam, K. and Oswald, I. (1984) Sleep helps healing. *Br. Med. J.*, **289**, 1400–1401.
Barberger-Gateau, P. (1997) Dyspnoea and disability. *Crit. Rev. Phys. Rehabil. Med.*, **9**, 265–299.
Carrieri-Kohlman, V. (1993) Desensitization and

guided mastery. *Heart Lung*, **22**, 226–234.
Jobst, K., McPherson, K. and Brown, V. (1986) Controlled trial of acupuncture for disabling breathlessness. *Lancet*, **ii**, 1416–1418.
Nisell, O. (1992) Causes and mechanisms of breathlessness. *Clin. Physiol.*, **12**, 1–17.
Olivier, F. I. (1998) Suggested guidelines for the use of exercise with adults in acute care settings. *Phys. Canada*, **50**, 127–135.
Roland, M. and Peper, E. (1987) Inhalation volume changes with inspirometer feedback and diaphragmatic breathing coaching. *Clin. Biofeedback Health*, **10**, 89–97.
Wolkove, N. and Kreisman, H. (1984) Effect of transcendental meditation on breathing and respiratory control. *J. Appl. Physiol.*, **56**, 607–612.

8 Physiotherapy to clear secretions

Summary

Sputum in perspective

> *It may be gob to you but it's my bread and butter.*
>
> Cole, physician, 1999

Question 1

Do bronchial secretions matter? In the short term, can they obstruct breathing? In the medium term, do they correlate with lung function or quality of life? In the long term, are they implicated in the natural history of disease?

In the short term, if superficial secretions are seen or heard to obstruct breathing, causing distress and airflow limitation, they do matter and need to be cleared, with or without assistance.

In the medium term, secretions in people with COPD do not correlate with airflow obstruction (Baldwin, 1994; Bateman *et al.*, 1979; Peto *et al.*, 1983) or mortality (Wiles and Hnizdo, 1991). People with non-acute COPD complain little about sputum or its effect on their quality of life. The evidence that sputum clearance improves lung function in stable COPD is underwhelming.

But in patients with excess secretions and chronic infection, the following must be considered:

- uncleared secretions augment infection

- chronic infection damages airways by precipitating the inflammatory cascade.

People with diseases such as cystic fibrosis (CF) or bronchiectasis still show little relationship between expectorated sputum and pulmonary function (Rubin, 1997), but Williams (1995) suggests that secretion clearance slows the deterioration in lung function over time. At present we must assume that secretions do matter for these patients, especially as few researchers would care to investigate the effects of depriving, say, a sample of children with CF of their physiotherapy for a lifetime.

Therefore, on balance:

- for patients with sputum retention: secretions do matter
- for patients with excess secretions: secretions do matter if their presence augments progression of the disease or compromises oxygenation, but appear not to matter for people with stable COPD if they do not complain of this and are capable of clearing their own secretions.

Question 2

When secretions do matter, how do we evaluate our clearance techniques? The literature is a minefield when trying to assess secretion clearance. Studies *in vitro* or in people with normal lungs bear limited relation to clinical practice. Studies that do not correct for cough alone are suspect because most physiotherapy techniques to clear secretions include coughing. Studies that do not follow up secretion clearance for several hours after treatment are of limited usefulness (Mortensen *et al.*, 1991). Studies that measure sputum volume or sputum weight do not compensate for saliva or swallowed secretions (Hasani *et al.*, 1994). Sputum volume is adequate for outcome measurement in clinical practice but is not valid for research.

Research is most useful when secretion clearance is measured by labelling inhaled radioactive aerosol, whose particles become entrapped in airway mucus, then scanning mucus clearance by gamma camera (Mortensen *et al.*, 1991). For secretion clearance from the large airways, where total cross-section is narrower, FEV_1 measurements may be helpful in long-term studies if airway obstruction has been affected by progressive damage caused by excess secretions. However, the procedure itself alters the bronchial status quo by shearing secretions off the airway wall.

A more sensitive test is specific airways conductance, which measures airflow obstruction without a forced expiration. For this we have one aged study to rely on, which suggests that conventional chest physiotherapy reduces airflow obstruction in the short term (Cochrane *et al.*, 1977).

Question 3

How do we identify the problem?

- Is mucociliary clearance the problem, impaired by hypoxia, infection, damaged airways, dehydration, cigarette smoke, immobility, anaesthetic agent or pollution (Houtmeyers, 1999)?
- Is coughing the problem, impaired by weakness or pain?
- Is expectoration the problem, impaired by a dry mouth or embarrassment?

This section assumes that patients need physiotherapy if they have sputum retention, or if excess secretions are detrimental.

HYDRATION AND HUMIDIFICATION

Humidity: water content of a gas, expressed as absolute or relative.

Absolute humidity: water in a given volume of gas (mg/L), which increases or decreases with temperature.

Relative humidity: water expressed as a percentage of that which would fully saturate the volume of gas at a given temperature (%).

The major determinants of mucociliary clearance are the quality and quantity of mucus and the health of the cilia (King, 1998), all of which depend on systemic hydration. The mucociliary

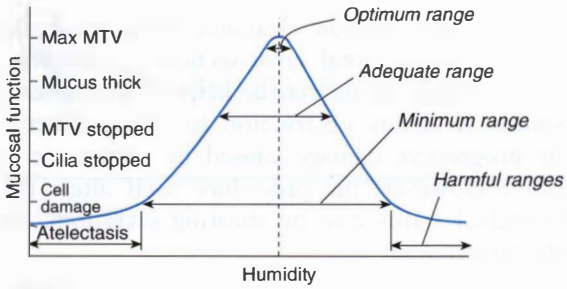


Figure 8.1 Variation of inspired humidity from core temperature and 100% relative humidity, and how this might affect mucosal function. *MTV = mucociliary transport velocity*. (From Williams, R., Rankin, N., Smith, T. *et al.* (1996) Relationship between the humidity and temperature of inspired gas and the function of the airway mucosa. *Critical Care Medicine*, **24**, 11)

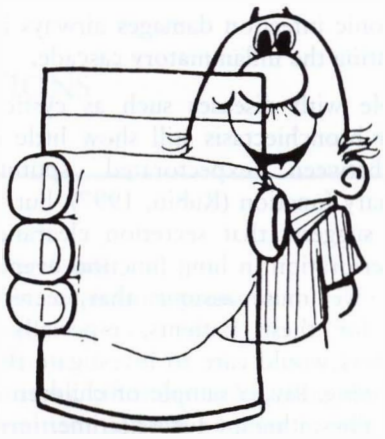


Figure 8.2 Systemic hydration. (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

escalator provides a frontier against the onslaught of 10 billion inhaled particles a day, but this depends on the sol layer of watery fluid in which the cilia must be able to move freely. Dehydration immobilizes cilia more than death (Clarke, 1989) and causes mucosal drying, inflammation and ulceration (Figure 8.1).

At body temperature, a small increase in temperature causes a relatively large increase in water content (Joynt and Lipman, 1994), and the bronchial tree is normally fully saturated with water vapour from just below the carina onwards. From here to the alveoli, with the temperature at 37°C, water content at 43.9 mg/L maintains relative humidity at 100% (Branson, 1999).

To maintain this and prevent secretions thickening, the following can be administered:

- systemic hydration by oral or intravenous fluids
- humidification by inhalation of a vapour
- nebulization by inhalation of an aerosol
- heat-moisture exchange using a filter.

Classification

Hydration

There is no evidence that overhydration speeds mucociliary clearance, but dehydration is common (Blower, 1997) and reduces mucus transport by 25% (Luce *et al.*, 1993, p. 136). Systemic fluid intake is cheap, safe and not baffled out in the upper airways as is much inhaled moisture. Systemic hydration should be the priority (Figure 8.2).

Some patients restrict their fluid intake. They may have stress incontinence due to chronic cough. They may have frequency due to diuretics. Hospitalized patients may not be near the toilet. They may reduce their drinking because of the change in environment and daily routine. Blower (1997) found that over a third of hospitalized patients were unable to drink as often as needed, and over half were thirsty. Causes were inability to reach their drink and unwillingness to bother busy staff.

For hospitalized patients it is recommended that daily fluid intake is 1.5 litres (DoH, 1995). Hot weather, fever and exacerbation of disease markedly increase requirements. For patients at home, a trial of increased fluid intake would indicate whether or not this helps expectoration, in which case it can be incorporated into their routine, a jug of water being accessible as a reminder. Liquid diets and blenderized meals do not hydrate because of their osmotic load. Juices and canned drinks are adequate but not as efficient as water. Caffeine drinks such as tea,

Table 8.1 Comparison of systems

	Hot water humidifier	Nebulizer	HME
Moisture output (g/m^3)	35–50	20–1000	25–35
Infection risk	Reservoir and circuit	Reservoir, circuit and aerosol	Low
Advantages	Bacteria not transmitted with vapour	For tenacious secretions	Simple, cheap
Disadvantages	Labour-intensive Bulky	Labour-intensive	May block with mucus May be inadequate

coffee and cola have some diuretic action and are less helpful but are preferred by some patients.

Some disorders may be complicated by acid–base or electrolyte disturbance, kidney dysfunction, pulmonary oedema or diuretic therapy, and liaison with medical staff is then advisable.

Hot water humidification

A *hot water humidifier* or *hot water bath* (Table 8.1) creates a vapour by passing gas over or through sterile liquid, which is maintained at 45–60°C. The vapour is allowed to cool along a specific length of tubing to reach the patient with a relative humidity of 100% at 37°C.

When used with non-intubated patients, the nose and larynx cause the vapour to condense into drops that are too large to navigate the airways. Hot water humidifiers are therefore best used for patients with endotracheal or tracheostomy tubes, which allow passage of the vapour. They are also used for small children to keep their narrow upper airways clear, under supervision. For adults with an intact upper airway, hot water humidifiers may assist in moistening a dry throat, but a steamy cup of tea is more pleasurable.

To ensure safety, the humidifier should:

- incorporate an over-heating alarm
- be kept heated continuously to minimize colonization with bacteria
- be kept below the patient to prevent condensed water tipping into the airway.

Condensed water should be viewed as infectious waste. Tubing requires regular emptying, either manually (away from the patient) or by water traps in the circuit (Figure 8.3). Heated wire circuits prevent condensation, but if these permit cooler temperatures in the hot water chamber, there is increased risk of infection (Branson, 1996).

A *steam inhalation* uses the same principle by delivering vapour from near-boiling water to the patient via a mouthpiece. Some patients find it beneficial but the temperature of the water is not controlled and the container is easily knocked

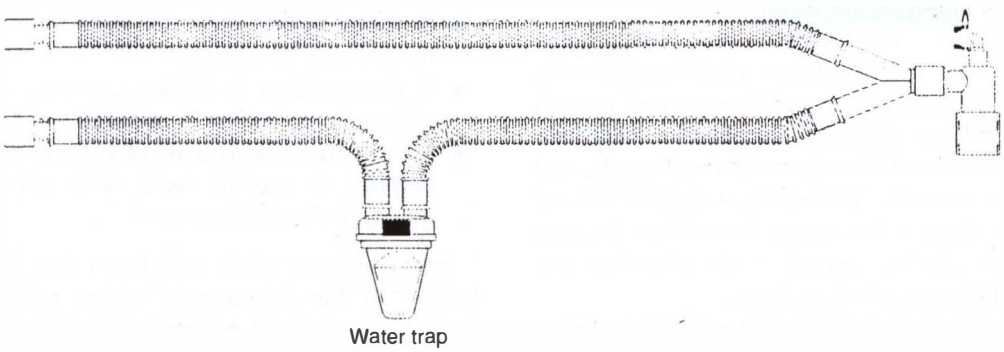


Figure 8.3 A water trap to collect condensed water in the tubing between humidifier and patient.

over. It is contraindicated for hospitalized patients, and if patients at home want to use it they need safety advice. Menthol or other additives are found useful by some patients.

Cold water humidification

A cold water humidifier bubbles cold gas through cold water, preferably via a diffuser which breaks up air into small bubbles to increase the gas-water interface. Gas passes too quickly to pick up much moisture and the higher the flow rate, the poorer the humidification (Mason, 1993, p. 231). The system is thought to be incapable of achieving a positive water balance in the airways (Darin, 1982), is insufficient to prevent insensible water loss (Hodgkin *et al.*, 1993, p. 207), cools the system below room temperature (Campbell *et al.*, 1988) and was condemned as 'dangerously inadequate' over three decades ago (Graff and Benson, 1969).

These 'bubble-through' humidifiers may be indicated for some patients on dry oxygen if they feel that this makes their mouths more comfortable. Otherwise they simply increase airflow resistance (Mason, 1993, p. 231) and grow bacteria, although this is rarely a problem because so little infected humidity reaches the patient. Sceptical ward staff can be won over by being invited to observe the time it takes for the water to be used up (if ever). The untiring enthusiasm of sales representatives has kept most hospitals stocked up with these humidifiers, and we await research on any new devices.

Nebulized humidification (Figure 8.4)

Large-volume nebulizers use the same mechanism as the nebulizers used for delivering drugs (p. 141). Sterile liquid is converted into an aerosol, whose droplets are small enough to navigate the nasal passages and vocal cords, and reach the airways. They pose a greater risk of infection than a hot-water humidifier because organisms can be carried in droplets but not vapour (Pilbeam, 1998, p. 161).

Large-volume jet nebulizers commonly use a cold liquid because heat is not necessary for this mechanism, but heated nebulizers are available

Figure 8.4 Humidification. (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

which combine the advantages of both vapour and aerosol. Large-volume nebulizers incorporate a Venturi system which ensures delivery of a fixed percentage of oxygen (Figure 8.5). Some nebulizers collect the larger droplets and recycle them to be re-nebulized. Large-volume nebulizers are used continuously.

Small-volume jet nebulizers, used periodically before physiotherapy, are the same as those used for drug delivery and carry the same risk of infection (p. 142). These 'saline nebs' deliver typically just 2 mL of fluid (Hodgkin *et al.*, 1993, p. 206). They have been shown to increase sputum 'yield' but create no significant increase in radioaerosol clearance (Sutton *et al.*, 1988). This may be because they wet the throat and assist expectoration but are not adequate for loosening secretions. It is therefore suggested that:

- if the problem is a dry mouth or throat, mouth care is required
- if the problem is thick secretions, a large-volume jet nebulizer is required
- if the patient finds a small-volume nebulizer helpful, it can be used with appropriate infection control measures.

Small volume saline nebulizers may be more helpful in the community, where bacteria are less vicious than in hospital, but patients must not leave them wet and untended after use.

The *ultrasonic nebulizer* is a self-contained

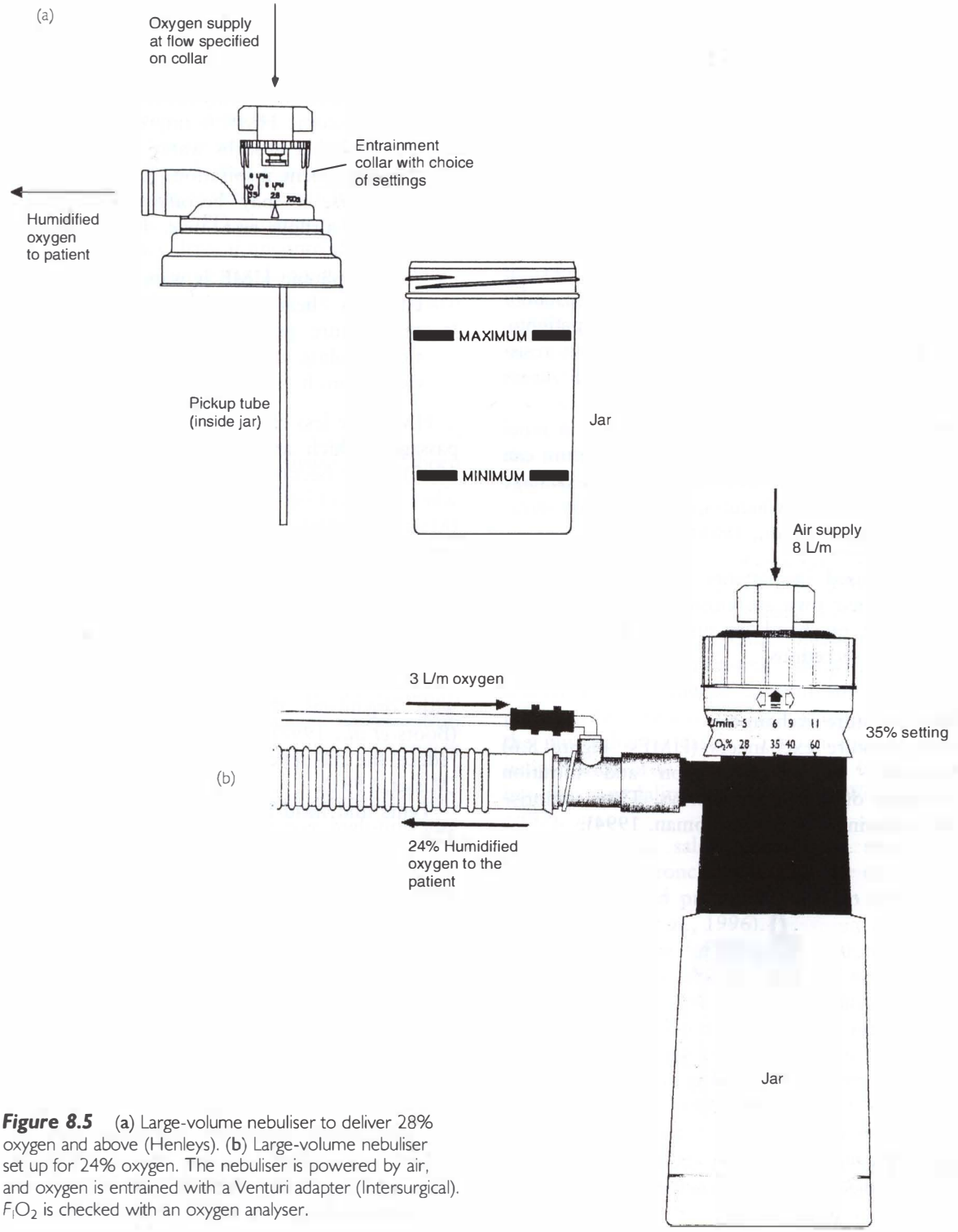


Figure 8.5 (**a**) Large-volume nebuliser to deliver 28% oxygen and above (Henleys). (**b**) Large-volume nebuliser set up for 24% oxygen. The nebuliser is powered by air, and oxygen is entrained with a Venturi adapter (Intersurgical). F_IO_2 is checked with an oxygen analyser.

electrical device that transmits vibrations through a liquid to atomize the particles, producing a 2–10-fold greater output than a jet nebulizer (Phillips and Millard, 1994). Oxygen can be added with a nasal cannula. Advantages are its silence and efficiency. Disadvantages are expense and, sometimes, the less beneficial effects of efficiency:

- Deeper penetration into the airways creates a greater risk of infection (Suda, 1995). If the device is not well maintained, a 'pseudomonas soup' can be poured into the patient.
- The dense aerosol increases airflow resistance, which may be a problem with severe acute asthma.
- Fluid overload is a risk for people in renal failure or for babies. Water intoxication can inactivate surfactant, block airways, damage cilia and overwhelm the mucociliary escalator (Shelly *et al.*, 1988).

When used in patients who have difficulty clearing their own secretions, a physiotherapist should be on hand because of the increased volume of secretions.

Heat-moisture exchange

Heat-moisture exchangers (HMEs; Figure 8.6) reproduce the humidification and filtration functions of the upper airways. They comprise the following (Joynt and Lipman, 1994):

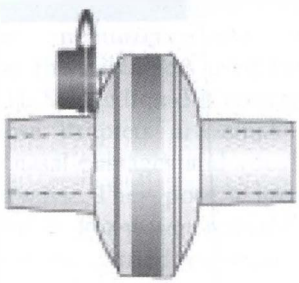


Figure 8.6 Heat–moisture exchanger used to return heat and moisture in exhaled gas to the patient's own airways.

- A condenser HME, sometimes known as a Swedish nose, traps expired water vapour and some body heat. It is 50% efficient and fits over a tracheostomy tube.
- A hygroscopic HME is impregnated with a chemical that absorbs water and humidifies the subsequent inspiration. If used for over 24 hours, it may become saturated and increase airflow resistance. It is 70% efficient.
- A hydrophobic HME is water-repellent and conducts heat poorly, thus causing a temperature gradient, leading to evaporation, cooling and conservation of water on expiration. It is 60–70% efficient.

HMEs are less effictive than the natural nasal passages, which are 80–85% efficient (Lloyd, 1987). They become less efficient over time or when a patient breathes with high tidal volumes (Martin, 1990a). HMEs are not adequate for patients with thick secretions (Branson, 1996) but are convenient for mobile patients, for limited periods of mechanical ventilation or when using devices such as CPAP or a manual inflation bag (p. 371). Some HMEs incorporate a bacterial filter. HMEs show less colonization with organisms than hot water humidifiers (Boots *et al.*, 1997) but have not been found to reduce the incidence of nosocomial pneumonia (Misset, 1991).

Tents and head boxes are discussed on page 124.

Effects of humidification

The superficial gel layer of the mucus blanket acts as a protective barrier between the body and atmosphere, and is mostly waterproof, but it can absorb some inhaled moisture (Conway, 1992b).

Complications of humidification

- Ubiquitous hospital bacteria enjoy nothing more than stagnant humidifier water, especially if it is lukewarm. Hot-water baths are less risky, partly because they are hot and partly because vapour cannot convey bacteria (Branson, 1996). Infection is also a

risk in home humidifiers (Patterson *et al.*, 1998).
- Bronchospasm can be caused in susceptible patients by:
  - an unheated nebulizer
  - dense ultrasonic mist
  - use of a liquid other than isotonic saline in nebulizing systems (Church, 1991).
- Hypercapnic COPD patients may suffer loss of respiratory drive if uncontrolled oxygen is used as the driving gas. Large-volume nebulizers can be set up to run on 24% oxygen (Figure 8.5b).

Indications

Humidification is necessary for:

- people whose upper airway has been bypassed with a tracheal tube
- people with thick secretions
- babies at risk of airway blockage with secretions
- people on oxygen therapy who have hyperreactive airways (heated system required) or if using a mask for prolonged periods, with high flow rates (Fulmer and Snider, 1984), or who are mouth-breathing, nil-by-mouth or have a dry mouth and find expectoration difficult, especially if they are on oxygen therapy
- patients using non-invasive mechanical aids such as CPAP, which deliver high-flow dry gases (Conway, 1992b).

Humidification is not indicated for the following:

- people using nasal cannulae, because at low flow rates the patient's nose provides adequate humidification, and any added moisture is lost by condensation in the narrow tubing (Campbell *et al.*, 1988)
- people with permanent tracheostomies, because adaptation occurs (Shelly *et al.*, 1988)
- people using venturi masks, because the entrained ambient air is not as dry as piped oxygen, water may condense in the entrainment ports and alter F_IO_2, and attempts to humidify the oxygen and/or entrained air with a humidity adapter are rarely effective (Gribbin, 1993); it is better to set up a venturi nebulizer.

Technique

A mask or mouthpiece can be used, depending on patient comfort. Lung deposition is enhanced by the upright-sitting or side-lying position. Condensation is minimized by wide-bore tubing, avoidance of lengthy convolutions of tubing, or use of a heated wire circuit. When using a heated system, the manufacturer's safety instructions should be followed; for example, use the correct length of tubing and ensure that, when the heater is on, gas flow is maintained and the reservoir is not dry.

When small nebulizers are used for drug delivery, they are diluted with isotonic saline. Large, non-disposable nebulizers use sterile water because repeated filling leads to encrustation. Water should not be used for people with hyperreactive airways. Other fluids for nebulization include hypertonic saline, which is usually used to induce sputum for diagnostic purposes (p. 38) but has been shown to double the mucociliary clearance rate in sputum from people with bronchiectasis and CF by increasing the osmolality of secretions (Wills *et al.*, 1997).

If hypertonic saline is used, it should be preceded by a bronchodilator in case of bronchospasm and used preferably with an ultrasonic nebulizer (Eng *et al.*, 1996).

Regular checks for a visible mist may show that there is no blockage in the system, but production of a mist does not indicate correct droplet size because the therapeutic range of 2–5 μm diameter is not visible.

Room humidifiers have no influence on the respiratory tract and can create a source of infection (Wissing, 1988), but a well-maintained system may reduce the incidence of 'sick building syndrome' (Nordstrom *et al.*, 1994).

EXERCISE

Exercise is an efficient way to assist clearance of secretions. It creates a shearing force along the airway walls (Andersen *et al.*, 1979) and leads to sympathetic stimulation of cilia by catecholamine release and parasympathetic stimulation of airway mucus glands (Bye *et al.*, 1997). The benefits of exercise have been mostly documented with CF patients and measured by FEV_1 (Thomas *et al.*, 1995). In children with PCD (p. 92), exercise has been found more beneficial than drugs in aiding bronchodilation prior to physiotherapy (Phillips *et al.*, 1998).

POSTURAL DRAINAGE

Postural drainage (PD) is thought to use gravity to assist drainage of secretions, but the exact mechanism is unclear.

Technique

Timing depends on the individual, but PD in the early morning helps clear the night's accumulated secretions, and PD an hour before sleep reduces night coughing. PD should be avoided after meals. If bronchodilators are prescribed, these are best taken 15 minutes beforehand.

Patients are positioned with the area to be drained uppermost (Appendix B), bearing in mind that these positions may need modification for patient comfort or if lung architecture has been distorted by surgery, fibrosis, a large abscess or bullae. The most affected area is drained first to prevent infected secretions spilling into healthy lung. Patients on monitors should be checked for arrhythmias or desaturation before, during and after PD.

Drainage times vary, but ideally each position requires 10 minutes (Gumery *et al.*, 2001). If the disease affects the whole lung, each lobe requires drainage, but a maximum of three positions at a session keeps it tolerable. People with localized bronchiectasis or an abscess should be positioned with the affected area upwards. The procedure should be discontinued if the patient complains of headache, discomfort, dizziness, palpitations, breathlessness or fatigue.

Modifications include:

- alternate-side-lying, the most commonly used position for patients with generalized secretions
- sleeping in a modified postural drainage position (Verboon *et al.*, 1986), using telephone directories to prop up the foot of the bed, so long as this does not cause coughing during the night
- bending forward over the toilet, or leaning over to clean the bath, which are positions reported by some patients to spontaneously clear secretions.

Effects

PD is effective if it provides symptomatic relief or long-term benefit. It is unhelpful if patients find it uncomfortable or unacceptably inconvenient. Long-term benefit is difficult to evaluate because most studies do not isolate it from other techniques.

Wong *et al.* (1977) suggest that thin mucus is more responsive to gravity than thick mucus, and that, while mucus in the large airways is responsive to gravity in CF patients, surface forces may limit drainage in the smaller airways. Mortensen *et al.* (1991) have shown that total clearance is unchanged several hours after treatment. This suggests that it might be less useful for the long-term effect of a disease process, e.g. the vicious cycle of CF, but may be useful for symptomatic management so that time at school is more amenable or time at the dentist more comfortable.

The direct effect of gravity may not be the only mechanism. Tannenbaum and Davids (1995) cast doubt on the ability of gravity to influence thick secretions, and Lannefors (1992) found, surprisingly, that dependent lung showed greater clearance than non-dependent lung. The rationale may be that the greater ventilation in dependent regions (p. 9) encourages movement of secretions by mechanical squeezing. It may also be that the motion of changing into a postural drainage position has as much effect as the position itself, as shown by patients who

expectorate immediately after changing position.

Unhelpful effects are mainly related to the head-down position, which increases the work of breathing (Marini *et al.*, 1984), tends to reduce tidal volume (Olséni, 1994) and decreases FRC in people with normal lungs (Jenkins *et al.*, 1988).

Indications

PD is used for people who find it preferable or more effective than other means, especially if they have difficulty in using more active methods. PD assists people with lung abscess usually, bronchiectasis often and CF sometimes. If used for patients with acute problems, modified positions are often required. For people with chronic conditions, poor compliance with this antisocial procedure is renowned (Currie *et al.*, 1986). A week's trial should include motivating patients to fit an individual programme into their daily routine so that results can be assessed accurately and decisions can be made on whether this and/or other measures are most suitable.

Precautions

It is unwise to tip a patient head down in the presence of:

- cerebral oedema, e.g. acute head injury or recent stroke
- hypertension
- surgical emphysema
- trauma, burns or recent surgery to the head or neck
- recent pneumonectomy or surgery to the aorta, oesophagus or cardiac sphincter of the stomach
- headache
- breathlessness
- symptomatic hiatus hernia
- history of seizures
- epistaxis or recent haemoptysis
- abdominal distension, pregnancy, obesity
- acute spinal cord lesion
- pulmonary oedema, arrhythmias or cardiovascular instability
- undrained pneumothorax
- bronchopleural fistula
- risk of aspiration
- gastro-oesophageal reflux (Button *et al.*, 1994).

MANUAL TECHNIQUES

Percussion or vibrations are performed in a postural drainage position. They aim to jar loose secretions from the airway walls.

Technique

Percussion consists of rhythmic clapping on the chest with loose wrist and cupped hand, creating an energy wave that is transmitted to the airways. A sheet or pyjama top should cover the patient but thick covering dampens transmission through the chest wall (Frownfelter and Dean, 1996, p. 345), and correct cupping of the hand ensures that the procedure is completely comfortable. Indeed, performed correctly, it can soothe children and sometimes give relief to people who are acutely breathless. Patients may prefer a slow single-handed technique (Bastow, personal communication, 1996) or rapid double-handed technique, but the latter can cause breath-holding and occasionally bronchospasm (Wollmer *et al.*, 1985).

Vibrations consist of a fine oscillation of the hands directed inwards against the chest, performed on exhalation after a deep inhalation. Shaking is a coarser movement in which the chest wall is rhythmically compressed. Both are less effective on a squashy mattress. Vibrations, shaking and percussion should be interspersed with relaxed deep breathing to prevent airway closure, desaturation or bronchospasm.

Effects

Many studies on these traditional techniques are old, unreliable or contradictory, but some claims are the following:

- When combined with postural drainage, manual techniques accelerate clearance from peripheral lung regions (Bateman *et al.*,

1981) and produce more sputum than exercise alone (Bilton *et al.*, 1992) but it is unclear which modality is the effective one
- Percussion speeds mucociliary transport (Radford, 1982).

Self-percussion is objectively ineffective and can cause oxygen desaturation in vulnerable patients because of the physical effort (Carr *et al.*, 1995). Patients sometimes claim subjective benefit, in which case those at risk of desaturation are advised to include regular rests with relaxed breathing.

Indications

Manual techniques reinforce patient dependency but are suited to some people who find these methods helpful. Patients with chronic problems usually prefer to choose an independent technique. Manual techniques may benefit people who are exhausted, e.g. with an exacerbation of disease, or weakened by neurological disease, or young children, the very elderly or those with learning difficulties.

Precautions

Percussion and vibrations are to be avoided or modified in the presence of:

- rib fracture, or potential rib fracture, e.g. metastatic carcinoma or osteoporosis
- loss of skin integrity, e.g. surgery, burns or chest drains
- pain, e.g. the above, pleurisy or post-herpetic neuralgia
- recent or excessive haemoptysis, e.g. due to abscess or lung contusion
- severe clotting disorder, e.g. platelet count below 50 000 (see Glossary)
- surgical emphysema
- unstable angina or arrhythmias.

Mini literature appraisal

Literature: Connors *et al.* (1980) stated that 'postural drainage and chest percussion in patients without sputum production is not indicated'.

Appraisal: Postural drainage and chest percussion in patients with ingrowing toenails is not indicated either.

BREATHING TECHNIQUES

Both of the following breathing techniques are flexible, efficient and effective when taught correctly. They foster independence because once taught they can be used without assistance. They are particularly suited to people with chronic lung problems but are adaptable to those with acute disease, autogenic drainage being preferable for fatigued patients. They are described separately but are based on the same principles and physiotherapists are advised to develop their own technique which incorporates both.

Active cycle of breathing

The active cycle of breathing (ACB) consists of a cycle of huffs from mid to low lung volume interspersed with deep breathing and relaxed abdominal breathing (Pryor and Webber, 1998, p. 140).

Mechanism

During huffing or forced expiration, the pleural pressure becomes positive and equals the alveolar pressure at a point along the airway called the equal pressure point, usually in the segmental bronchi. Towards the mouth from this point, the transmural pressure gradient is reversed so that pressure outside the airway is higher than inside, thus squeezing the airway by a process known as dynamic compression (p. 3). This limits airflow, but the squeezing of airways mouthwards of this point mobilizes secretions. At high lung volumes, the equal pressure point is more proximal because pleural pressure decreases and alveolar elastic recoil pressure increases (Figure 8.7). It is thought that huffing at low lung volume mobilizes secretions from the more distal airways.

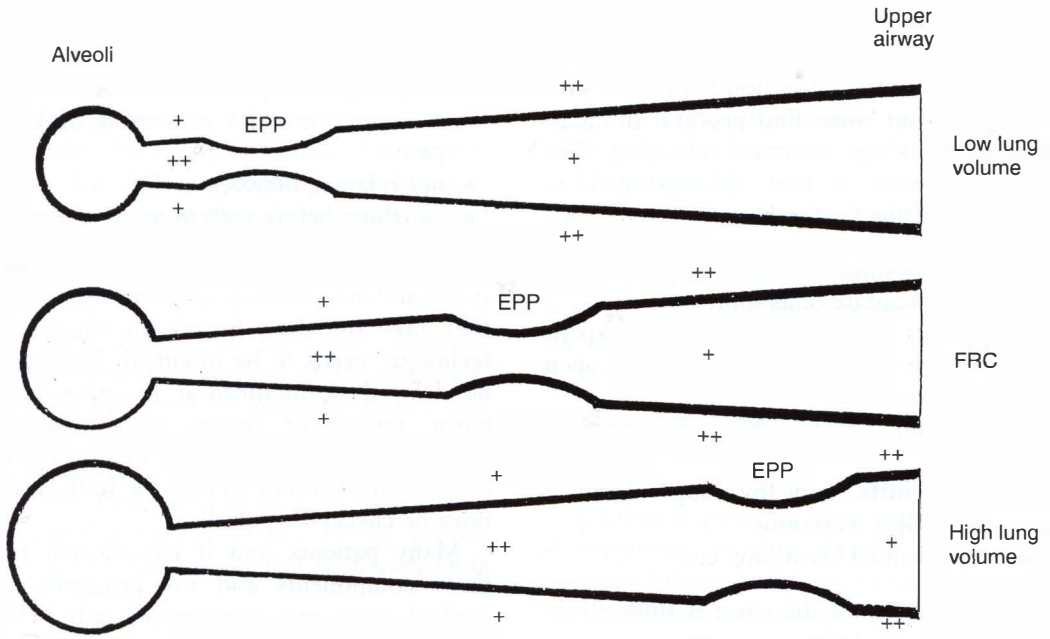


Figure 8.7 Greater pressures outside the airways (pleural pressure) than inside, caused by huffing. The equal pressure point (EPP) at different lung volumes moves towards the mouth as lung volume increases. *FRC = functional residual capacity.*

To counteract airway closure, the huffing phase of the cycle is interspersed with deep breathing. Relaxed abdominal breathing is also interspersed to reduce risks of bronchospasm, paroxysms of coughing or desaturation (Steven *et al.*, 1992).

Effects

The depth from which mucus is cleared has not yet been validated (AARC, 1993b) and the effect is still thought to be in the larger airways (Conway, 1992b), but ACB may be working quietly in the small airways by 'milking' the mucus mouthwards.

Technique

Correct teaching is essential because huffing alone can cause more airways obstruction than coughing (Figure 8.8), worsen pulmonary function (Boyd, 1994) and impair mucus clearance (David, 1991). Patients at risk of bronchospasm need to control the airflow and

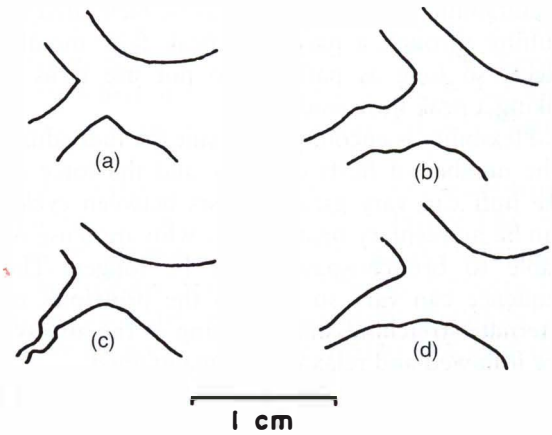


Figure 8.8 A section of the bronchial tree (A) at FRC, (B) at full inspiration, (C) at full expiration and (D) during coughing. (From Marshall, R. and Holden, W. S. (1963) Changes in calibre of the airways in man. *Thorax*, **18**, 54–58)

give particular attention to relaxed breathing and rests.

Patients take up their position of choice. This is often sitting but some find postural drainage positions helpful, e.g. alternate side-lying. The following sequence is best demonstrated to patients while sitting facing them:

- relaxed abdominal breathing (breathing control) to facilitate relaxation
- three or four deep breaths (thoracic expansion) to reverse airway closure and open collateral channels
- relaxed abdominal breathing, to maintain relaxation
- one or two huffs, from low lung volume at first, to mobilize secretions
- relaxed abdominal breathing, etc.

Cycles continue until the chest is subjectively or objectively clear, or the patient tires.

Avoidance of high lung volumes is easier if patients are told to inhale only a 'half-breath' before the huff, or to huff at the end of expiration only. Higher lung volumes can be used once secretions are mobilized from distal airways. Many patients will be able to identify when secretions are shifting, or can be shown by the technique used in autogenic drainage, below. Maintaining an open glottis may be facilitated by huffing through a paediatric peak flow mouthpiece, so long as patients do not use it as if taking a peak flow reading.

Flexibility is encouraged to suit the individual. The number of huffs can vary and the force of the huff can vary greatly. Rests between cycles can be momentary or, for those who are tense or liable to bronchospasm, may be longer. The sequence can vary so long as the principles of alternate stretching and squeezing of the airways are followed and relaxation is maintained.

The patient will make spontaneous adaptations and the physiotherapist can check whether these are helpful. Unhelpful examples include:

- huffing at too high a lung volume at first, by taking a deep breath before the huff
- not alternating the stretching components (deep breathing or abdominal breathing) with the squeezing component (the huff)
- taking too sharp a deep breath, thus forcing the secretions back or stirring up bronchospasm
- not relaxing between cycles
- coughing before secretions are accessible.

Patients with undiagnosed hyperventilation syndrome may develop symptoms (p. 295) when they take the deep breath, in which case the technique needs to be modified. Huffing should be delayed or modified if it causes bronchospasm, fatigue or spasms of coughing. Some patients prefer to do several cycles of deep and abdominal breathing before the huff. Technique must be checked regularly.

Many patients find it complicated to learn three components and the principles can be applied using two components only. The deep breath can be combined with the abdominal breath by taking 'a nice big comfortable relaxed sigh'. This stretches the airways but maintains relaxation, and can be interspersed with the huff.

Autogenic drainage

Autogenic drainage (AD) shares a similar rationale to ACB, with special emphasis on creating high airflow in different generations of bronchi without allowing airway collapse. Controlled breathing clears secretions from small to large airways by gradually increasing FRC. For people with CF or bronchiectasis, the full sequence can take up to 30–45 minutes to complete, but it is less burdensome when combined with activities such as nebulizing drugs or watching TV. For other patients, length of treatment is shorter and flexible. Control of the speed of inhalation and exhalation is the key.

Indications

AD is particularly suited to people with chronic hypersecretory disease, but selected components can be used for the acute hospital patient, e.g. postoperative patients who are anxious about

pain and stitches, people with haemoptysis or asthma or for those at risk of panic attacks. For breathless people, short sessions are required, with modifications as necessary to avoid upsetting the breathing pattern. Adolescents appreciate that AD can reduce their hyperinflated chests so long as they do not start inhalation before fully breathing out.

Effects

AD improves airflow in the small airways, clearing secretions that are not easily accessible, and is often preferred by patients (Butler and Sutherland, 1998). It is less likely to cause oxygen desaturation than PD and percussion (Giles, 1995). Compared to ACB, it shows faster mucus clearance (Miller *et al.*, 1995), greater increase in S_aO_2 and reduction in hypercapnia (Savci *et al.*, 2000).

Technique

Patients choose their position. Most sit upright, although some prefer supine. For facilitation of abdominal movement, some patients find prone helpful. During teaching, the physiotherapist's hands can be used to assess secretions and facilitate exhalation.

Face muscles, shoulders and arms remain relaxed throughout. The mouth, throat and glottis are kept open and the neck is maintained in slight extension because any obstruction prevents free laminar flow of air (Figure 8.9). Upper airway closure and air swallowing are less likely if there is little movement of the larynx.

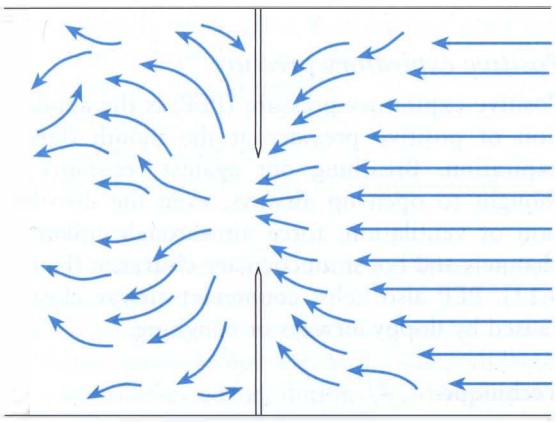


Figure 8.9 Disruption of airflow by upper airway constriction.

The nose is blown if necessary, and the throat cleared of secretions to reduce resistance to airflow. The location of secretions is identified by the patient exhaling until the rattle of secretions is heard. The later the rattle on exhalation, the more peripheral are secretions. The AD cycle is then followed:

1. Inhalation through the nose at 1.5–2 times tidal volume, slow enough for the breath not to be heard, using an abdominal breathing pattern if possible. Slow inspiration prevents secretions moving distally and encourages equal filling of all areas of lung.
2. End-inspiratory pause for 1–3 seconds to encourage air to get behind secretions by collateral ventilation.
3. Exhalation at a steady rate, with the highest flow that does not cause airway collapse, breathing out to a low enough volume to locate and 'unstick' the mucus. Some patients exhale through the nose or through pursed lips to utilize the extra resistance and create a form of PEP (p. 198).

If the mucus has been heard to rattle early on exhalation, it is advisable to clear the upper airways first at higher lung volumes. If not, the breaths are started from residual volume (Figure 8.9).

When the patient feels the secretions moving upwards, breaths are taken at a higher FRC to 'collect' the mucus. These two phases may need to be repeated several times before the rattle of secretions is felt more proximally. Breathing at higher volumes can then be used to 'evacuate' the mucus to the upper airways, from where they can be expectorated by a huff or gentle cough.

The aim is a mucus rattle and not a wheeze. Patients at risk of bronchospasm may need to exhale 'as gently as a receding wave leaving foam on the beach'. The upper airway should remain open throughout. Exhaling against a

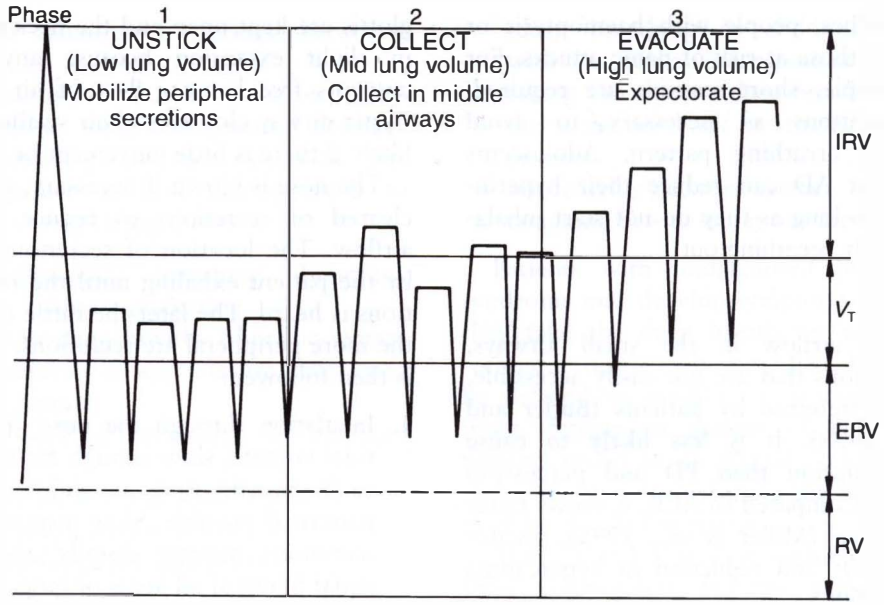


Figure 8.10 The three phases of autogenic drainage. (From Prasad, S. A. and Hussey, J. (1995) *Paediatric Respiratory Care*, Nelson Thornes)

tissue held at arm's length, or exhaling to mist up glasses or a mirror, encourages maximum airflow and discourages noise in the throat, which indicates upper airway closure. Cupping a hand over one ear accentuates the sound of airflow and enables the patient to minimize it.

Some patients with bronchiectasis can skip the second phase if the unstick phase brings up secretions quickly. Patients do not need to follow three rigid sequences so long as they use the principle of gradually increasing lung volume so that distal airways are cleared first. Patients who find it difficult to breathe at low lung volumes can vary the volumes (Prasad, 1993).

Coughing is re-educated to ensure that it is effective and not just noisy, and it may be avoided altogether if the huff is successful. Unnecessary huffing or coughing closes airways and stimulates the bronchospasm that has been avoided with such care.

AD is best interspersed with relaxation. Regular exercise is advised, and the PEP or flutter device, described in the next section, may be incorporated into the chest clearance sequence. A written handout is needed for reinforcement. Box 8.1 gives an example that includes the principles of ACB and AD. Box 8.2 is a useful accompaniment.

MECHANICAL AIDS

Positive expiratory pressure

Positive expiratory pressure (PEP) is the application of positive pressure at the mouth during expiration. Breathing out against resistance is thought to open up airways, even the distribution of ventilation, force air through collateral channels and boost mucociliary clearance (Figure 8.11). PEP also helps counteract airway closure caused by floppy airways or coughing.

Technique

If using a mask (Figure 8.12), patients adjust the air seal for the correct fit. Children need to acclimatize to the mask, or take it home to try out,

Box 8.1 Patient handout: breathing technique for clearing secretions

This technique squeezes and stretches your airways so that secretions can be brought up from deep in your lungs and expectorated with the minimum of effort.

1. Sit comfortably, or use any position that makes it easiest to clear your lungs.
2. Take one or two or three deep slow comfortable relaxed breaths, like sighs, preferably through your nose.
3. Relax for a few seconds to get your breathing steady again.
4. Take a very small breath in, then huff hard enough to move the secretions but not hard enough to make you wheeze or cough. Keep your throat open as you huff, head slightly raised.
5. Relax for a few seconds.
6. Repeat no. 2.
7. Continue the cycle, and as you feel the secretions shift, allow yourself slightly deeper breaths before the huff. This squeezes the airways higher up in your chest as the secretions move mouthwards.
8. Take sips of water between cycles. Keep relaxed.
9. When the secretions are ready, huff them out, or if necessary cough them out. Do not cough unnecessarily. When huffing or coughing, do your pelvic floor exercise before and during the cough.
10. Continue the cycle until your lungs are clear. If you are not sure, breathe out until you feel a rattle. The longer it takes to reach the rattle, the deeper are the secretions. If there is no rattle, your lungs are clear.

Box 8.2 Patient handout: pelvic floor exercises (Jane Goudge, Eastbourne DGH, with modifications)

Your pelvic floor can be strained by excessive coughing. Each cough is like a bounce on the pelvic floor muscles, which form a sling between the base of the spine and the pubic bone. Strengthening your pelvic floor muscles helps to prevent leaking.

Exercise

Feet slightly apart, close back passage (anal sphincter) and tighten front passages (urethra and vagina), draw them up inside.
Hold for a count of up to 4.
Let go slowly.
Do not hold your breath.
Avoid tightening buttock or tummy muscles.

How, when, where

Practise in different positions, e.g. lying with knees up, sitting, standing.
Practise little and often!
Set a daily programme that you know you can achieve.
Practise on the telephone, at the sink, while waiting for the kettle to boil, at the bus stop, in the supermarket queue, during TV advertisements.
Tighten pelvic floor muscles before any action causing a downward thrust, e.g. coughing, sneezing, laughing, pushing.

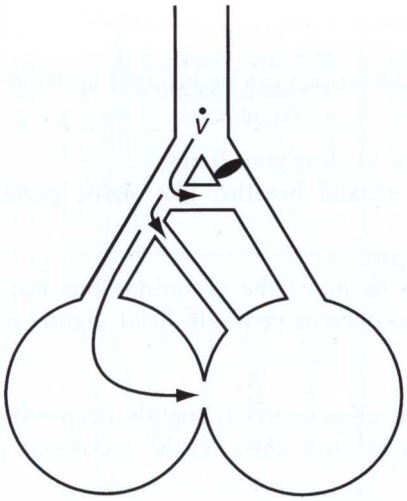


Figure 8.11 Collateral ventilation. Ventilation ($\dot{V}$) finds its way behind the mucus plug through collateral channels.

before adding the resistance. If using a mouthpiece (Figure 8.13), a nose clip is used and the same principles applied.

Patients then take up their position of choice but, for those with advanced disease, sitting with elbows resting on a table may protect the lungs from over-distension. The smallest diameter resistor is chosen which the patient can use comfortably for 2 minutes to achieve a pressure of 12–15 cmH_2O during mid-exhalation. As a guideline, a child might start with a 1.5 mm

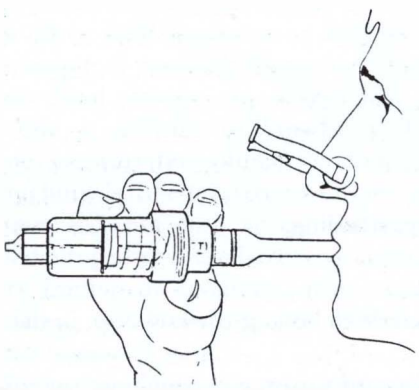


Figure 8.13 Mouthpiece PEP (Medicaid).

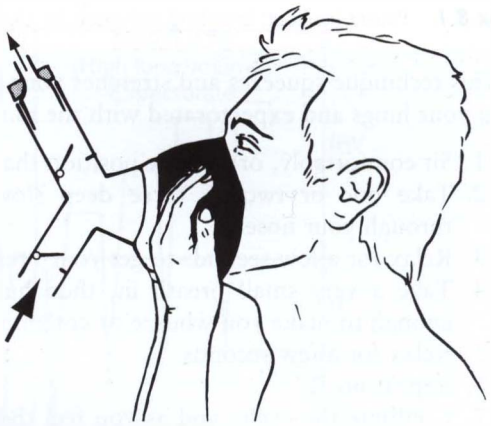


Figure 8.12 Mask PEP.

resistance and an adult a 2.5–3.5 mm resistance.

With the PEP mask held firmly over nose and mouth, patients inhale slowly at normal tidal volume, hold their breath briefly at end-inspiration, then exhale actively but not fully or forcefully. They should experience a comfortable effort, as if giving way to the resistance. As the secretions move centrally, breaths can be taken at higher tidal volumes. A manometer between the valve and resistor monitors the pressure. This is observed by the physiotherapist and not the patient, who might otherwise try to reach the target pressure by altering their breathing pattern.

Exhalation should last no more than 4 seconds. About 10 PEP breaths are alternated with several relaxed breaths. When secretions have been mobilized, they can be cleared by ACB/AD. This continues until the lungs are clear. During stable disease, most patients find that two 15-minute or three 10-minute sessions a day are adequate. Modifications of technique for mouthpiece PEP are enclosed with the device. The resistance should be checked approximately fortnightly for 6 weeks, then every month for 3 months.

'High pressure PEP' creates pressures of 60 cmH_2O (Hardy, 1994), using a manometer and flow volume curves to gain maximal homo-

geneity of expiration. It is not used regularly because of the equipment required.

Indications

PEP is mostly used by people with CF, especially adolescents and those seeking freedom from PD, but people with bronchiectasis, or those with COPD who have difficulty clearing secretions, also find it helpful (Christensen *et al.*, 1990). It is suited to patients with moderate amounts of sputum and can be used by children as young as 4. Those with large amounts of sputum need extra emphasis on accompanying techniques such as ACB/AD.

PEP can reduce the incidence of chest infection and improve lung function (Plebani, 1997). There are claims that it is more effective than PD (Mortensen *et al.*, 1991), PD and percussion (McIlwaine *et al.*, 1996) or the flutter (McIlwaine *et al.*, 1997).

Flutter

The combined effects of PEP and oscillation are exploited by the flutter (Figure 8.14), a device resembling a short fat pipe and suited to anyone who can blow bubbles. By exhaling into the flutter, the patient creates a positive oscillatory pressure of 10–20 cmH_2O in the airways. Oscillations are caused by the vibrations of a steel ball within a cone. The device encourages slow breathing in order to keep up the oscillations and gives more feedback than PEP by its vibrations.

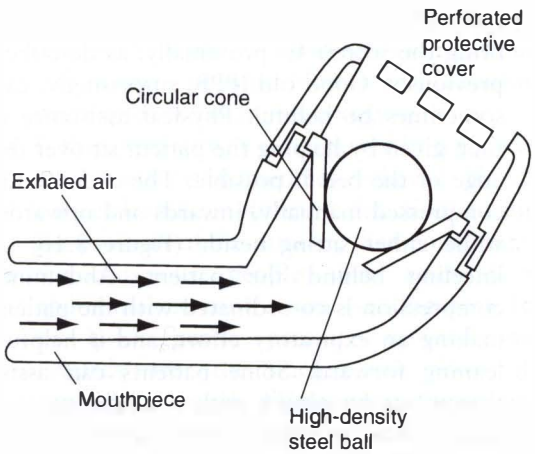


Figure 8.14 Flutter device (VarioRaw).

The flutter can be used for patients who find it helpful subjectively. Rigorous research is lacking, although claims have been made that it assists people with COPD (Callegari, 1994), CF (Konstan, 1994) and productive asthma (Girard and Terki, 1994). The oscillations may reduce sputum viscosity (App *et al.*, 1998) by rearranging crosslinks and reducing molecular size (King, 1997). Some patients find the flutter helpful for a final clear-out after other techniques.

Technique

Patients sit as if using the PEP mask, hold the mouthpiece in the lips, inhale through the nose, hold for 2–3 seconds, then exhale at twice-normal speed through the mouth. Patients must keep their cheeks taut and avoid blocking the holes on the device.

The aim is for maximum oscillation, which is assessed subjectively by the patient and objectively by the physiotherapist palpating for vibrations over the chest. The angle of the device should be varied until maximum frequency of oscillation is experienced. Mobilization of distal secretions is emphasized by tilting the flutter slightly upwards, and for more proximal secretions it is held more horizontal. The location from which secretions are mobilized, for both flutter and PEP, can also be influenced by breathing from different lung volumes, as with ACB/AD. The flutter is used for between 5 minutes (e.g. in COPD) and 20 minutes (e.g. in CF). Some devices break if dropped, and the steel ball should be kept away from toddlers, who might put it in their mouths.

The flutter is less evidence-based than PEP, but patients vary in their preference and should use whichever they find most helpful. PEP can be taught in two sessions and the flutter in one. Videos for teaching PEP and the flutter are available (Appendix C).

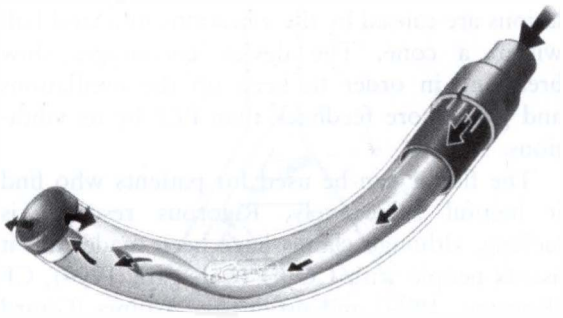


Figure 8.15 Cornet (RC cornet, with permission).

Cornet

Positive pressure and oscillations can be created by actively breathing out through a curved plastic tube called a cornet (Figure 8.15). This contains a flexible hose, which acts as a valve. Feng *et al.* (1998) claim that this decreases sputum viscosity.

Percussors, vibrators and oscillators

A variety of expensive devices are available to shake up the airways:

- mechanical percussors and vibrators (Bauer *et al.*, 1994)
- inflatable vests known as high-frequency chest wall compressors (Arens *et al.*, 1994)
- oral high frequency oscillators or intrapulmonary percussors, described on page 181 (Kluft *et al.*, 1996)
- small mechanical vibrators, which are cheaper and may help clearance from the lung periphery (Gross and King, 1984).

Intermittent positive pressure breathing

Some weak or drowsy patients with sputum retention may respond to IPPB. If other interventions have been ineffective, mechanical assistance can promote deep breaths in order to mobilize secretions or to maintain ventilation while other techniques are applied.

COUGH

Cough facilitation

Poor technique may be camouflaged by making loud but ineffectual noises in the throat. Tips to overcome problems are described below:

- Pain following surgery inhibits coughing. Pain relief and manual support are covered in Chapter 10.
- Thick secretions reduce the effectiveness of coughing (King, 1985). Assistance is by hydration if necessary, or a large volume nebulizer or, for rapid effect, an ultrasonic nebulizer.
- A dry mouth inhibits expectoration. This can be helped by a hot steamy drink, semi-frozen juice or gin and tonic (Reynard, 1997) mouthwash, sips of water or sucking ice.
- Inhibition may be caused by embarrassment, disgust or anxiety. Patients may be anxious about 'stitches splitting', in which case they can usually be reassured because wound dehiscence is rare (see p. 251). Anxiety about stress incontinence can inhibit coughing, especially in elderly people. There may also be anxiety about nausea or paroxysms of coughing.
- Weakness due to neuromuscular or terminal disease demands a resourceful physiotherapist. All measures should first be taken to bring the secretions proximally, as described previously. Good old IPPB, surprisingly, can sometimes be helpful. Physical assistance is then given by helping the patient sit over the edge of the bed if possible. The abdomen is compressed manually, inwards and upwards, while either sitting beside (Figure 8.16) or kneeling behind the patient. Abdominal compression is co-ordinated with the patient making an expiratory effort, and if helpful, leaning forward. Some patients can assist themselves by sitting with a pillow pressed against the abdomen, then, after a deep breath, bending forward while exhaling sharply. Some patients can learn breath-

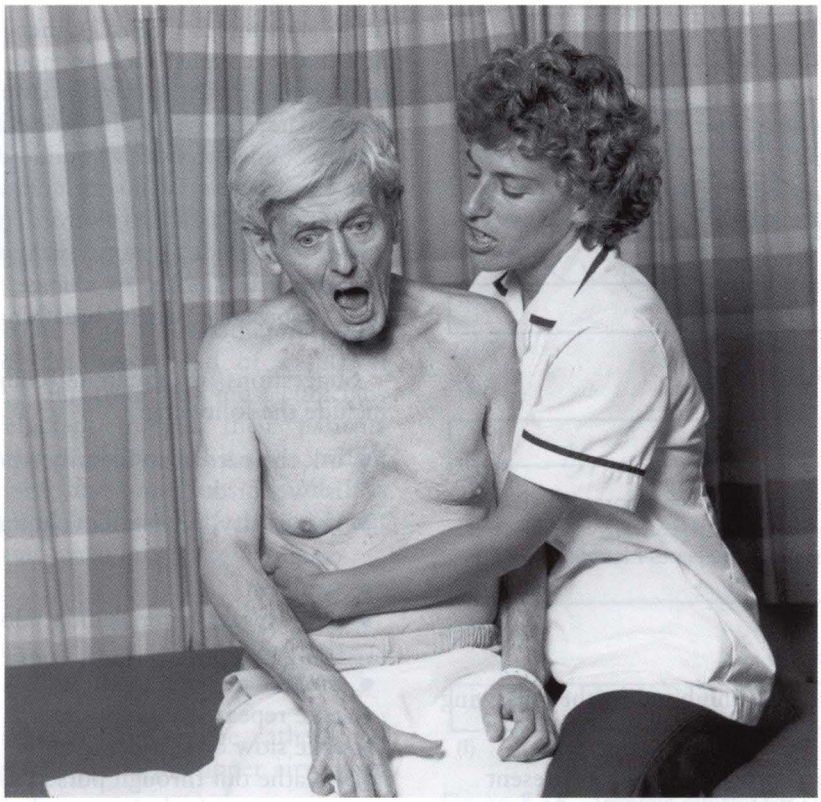


Figure 8.16 Supported cough.

stacking or glossopharyngeal breathing (p. 180) to increase the inhaled volume prior to the cough (Irwin *et al.*, 1998). For the well-resourced, Bach (1993) describes a mechanical positive pressure blower with expulsive decompression that aids coughing.

- If the upper airway is narrowed by a tumour, obstruction is sometimes relieved by the patient leaning in different directions to shift the position of the tumour, then coughing.
- Semiconscious people may respond to abdominal co-contraction (see Figure 6.5c), or quick gentle pressure upwards and inwards over the trachea just above the suprasternal notch. Moderate or strong pressure should be avoided, especially in elderly people because of potentially brittle calcified tracheal cartilages.
- Other tips are:
  - acupressure to CV17 (p. 84)
  - blowing out through a straw into a glass of water
  - gentle stimulation in one or other ear canal (Irwin *et al.*, 1998) to stimulate Arnold's nerve response but without using any instrument, which is risky.

Precautions

Coughing should be avoided immediately after eye or cranial surgery, or in the presence of an aneurysm. It is also best discouraged, when possible, if there is raised intracranial pressure, surgical emphysema, recent pneumonectomy or (depending on the cause) haemoptysis. Huffing can sometimes be substituted. Manually assisted coughing should be avoided after eating.

Figure 8.17 The need for cough suppression. (From Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford, with permission. Artist: James Northfield.)

Cough suppression

Coughing needs to be inhibited in the following situations:

- if one of the above precautions is present
- during ACB/AD, before secretions are accessible
- if the cough is dry and irritates the airways
- if coughing brings on bronchospasm, paroxysms of coughing or distress (Figure 8.17).

Multiple or paroxysmal coughs can lead to impaired mucus transport, fatigue, airway closure (Menkes and Britt, 1980) and occasionally cough syncope from excessive intrathoracic pressure.

Some patients inhibit their cough for social reasons or because of anxiety about stress incontinence. These are understandable but not healthy reasons to abstain, and patients will need advice and, if appropriate, referral.

The first step when dealing with an unproductive cough is to identify the cause (p. 31). A cough caused by asthma, post-nasal drip or gastro-oesophageal reflux should disappear once the inflammation is controlled. A quarter of patients taking ACE inhibitor drugs develop a cough, which disappears on average 4 months after starting the drug. Other non-productive and 'habit' coughs, such as those following viral infection, usually disappear over time but can perpetuate themselves by irritating the airways. Coughs and throat-clearing due to hyperventilation syndrome will melt away once the syndrome is treated. Factors that exacerbate coughing include irritants such as strong smells and cigarette smoke, or change in air temperature, especially when breathing through the mouth.

Suggestions to facilitate cough suppression include the following:

- ask the patient to identify whether the cough is 'wet or dry'
- if it is dry, advise to voluntarily inhibit the cough
- swallow
- take sips of water, lemon juice or cold green grape juice
- breathe through the nose
- take repeated short sniffs
- take slow or shallow breaths
- breathe out through pursed lips
- use autogenic drainage techniques to control airflow
- suck lozenges, ice pops or frozen seedless green grapes
- paradoxically, one manual percussion given once on the chest
- inhaling the steam from hot water poured over root ginger, then drinking the warm solution
- 'imagine warm honey sliding down your throat'
- for patients with floppy airways, e.g. late-stage CF, using a PEP mask to stabilize airways and reduce expiratory flow
- drugs as described on page 138
- for nocturnal cough, avoidance of the supine position, or, if intractable, use of CPAP (Bonnet, 1995).

Occasionally a dry cough may be helpful and need not be suppressed, e.g. if a patient finds that one brief cough will settle it as if 'scratching an itch'.

PHARYNGEAL SUCTION

> *'The worst part is the initial introduction of the catheter into the nostrils. Once past the turn at the back of the nose, it is not too unpleasant, until a cough is stimulated; then it feels like hours as the catheter is brought back up. It felt as if I was choking.'*
>
> Ludwig, 1984

These remarks come from a physiotherapist who found herself at the wrong end of a suction catheter, and they illustrate why most clinicians are, rightly, reluctant to put their patients through the ordeal of pharyngeal suction, which is usually distressing and sometimes painful. It is also dirty, risky and limited in effectiveness, but there are occasions when it is necessary.

Indications

Suction is performed if all the following criteria are met:

- secretions are accessible to the catheter, as indicated by crackles in the upper airway on auscultation
- secretions are detrimental to the patient
- the patient is unable to clear secretions by other means.

Patients who are semiconscious, weak or neurologically impaired may require suction, but those who are fatigued rarely do, because unless fatigue is extreme enough for the patient to need mechanical ventilation, coughing is usually still possible. Risks are increased in a combative patient, and those who need physical restraint for suction rarely need to undergo the procedure because they are usually strong enough to cough effectively, even though they choose not to. Forcible suction is unethical, illegal in the UK and acceptable only in life-threatening situations.

Catheters

Catheters have an end-hole through which the mucus is suctioned and side-eyes to relieve vacuum if the end-hole becomes blocked. Both end-hole and side-eyes are best slightly depressed

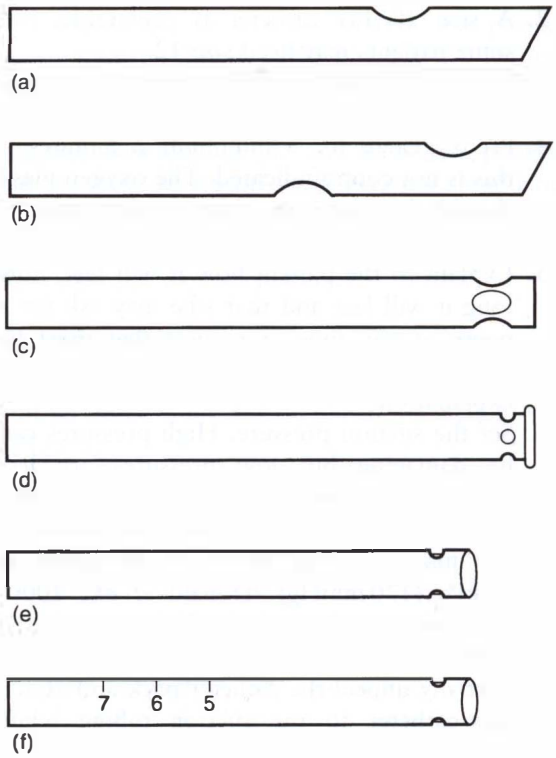


Figure 8.18 Different catheter tips.

to limit damage to the mucosa (Lomholt, 1982a). The side-eyes should not be too large (Figure 8.18c) or they reduce suction efficiency, and their total size should be less than that of the end-hole so that they do not become suction channels. Catheters with multiple side-eyes cause less invagination than those with one or two eyes (Link *et al.*, 1976).

A beaded tip is designed to prevent the side-eyes grabbing the mucosa, but the bead makes it unsuited to nasal suction because of the enlarged tip (Figure 8.18d). The ideal catheter is flexible and has a smooth rounded tip and small, multiple, countersunk side eyes.

Technique

The following steps are suggested:

1. Ensure that the resuscitation status of the patient is known.

2. A size 10 FG catheter is preferable, but some patients may need size 12.
3. Ensure the patient is upright or side-lying in case of vomiting.
4. Preoxygenate for a minimum 2 minutes if this is not contraindicated. The oxygen mask should then be kept close to the patient's face throughout.
5. Explain to the patient how it will feel, how long it will last and that s/he may ask for a pause at any time, a request that must be responded to. Unconscious patients need an explanation.
6. Set the suction pressure. High pressures can be damaging but low pressures are less effective and may prolong suction time (Lomholt, 1982b). Consensus suggests that the maximum safe pressure for adults is 23 kPa (170 mmHg) (Donald *et al.*, 2000) but original research is lacking and the lowest effective pressure should be used.
7. Partially unpeel the catheter pack and attach the catheter to the suction tubing while keeping the rest of the catheter in the pack. Put gloves on both hands. The dominant hand needs a sterile glove and both gloves should be non-powdered in case the powder finds its way into the lungs. Remove the catheter from the pack and lubricate the tip with water-soluble jelly. Maintain sterility of catheter and sterile glove.
8. With the suction port open, slide the catheter gently into the nostril, directing it parallel to the floor of the nose. If resistance is felt at the back of the pharynx, rotate the catheter slowly between the fingers and ease very gently forwards.
9. To reduce the risk of entering the oesophagus, ask the patient to tilt the head back, stick the tongue out and cough. If coughing is not possible, slide the catheter down during inspiration, when the glottis is more open. If the patient swallows, the catheter has slipped into the oesophagus, in which case it should be slightly withdrawn, the head repositioned and the procedure continued. The catheter is usually in the trachea if the patient coughs.
10. When resistance is felt, this is probably the carina and the catheter should be withdrawn slightly before applying vacuum pressure in order to limit trauma. Note roughly the length of catheter that has been inserted so that the carina can be avoided if further suction is necessary.
11. Apply suction and bring up the catheter slowly and smoothly, avoiding catheter rotation or sudden intermittent suction. Slow withdrawal reduces the need for a second attempt, but if the patient appears distressed, the catheter should be partially withdrawn until distress stops, then the vacuum should be removed and oxygen applied with the catheter still *in situ*, until the patient is ready to continue.

Rotation is unnecessary with multiple-eyed catheters and ineffective with other catheters (Emergency Care Research Institute, 1977) because torsional stiffness prevents transmission of rotation to the catheter tip *in vivo*.

Intermittent suction involving the sudden on/off application of vacuum pressure has two disadvantages:

- it reduces effectiveness by decreasing flow from an average 19 to an average 8.5 L/min (Brown, 1983), making further suction more likely
- sudden release of pressure may damage mucosa (Frownfelter and Dean, 1996, p. 768).

Czarnik *et al.* (1991) found that intermittent suction had no advantages over continuous suction.

Protection of mucosa is best maintained by continuous withdrawal, without stopping to change the position of the dominant hand on the catheter. If suction pressure rises unacceptably, the rocking thumb technique should be used, which is the smooth and partial removal of the thumb from the control port of the catheter to reduce pressure gently.

Afterwards, remove the gloves inside out over the catheter and discard, rinse out suction tubing, give the patient oxygen and comfort, check monitors.

If the nasal route is uncomfortable, the other nostril can be tried or the oral route used.

For oral suction, the catheter is inserted into an oral Guedel airway, a plastic tube shaped to conform to the palate with a flange to prevent it slipping into the throat (Figure 8.19b). A size 6 is average, but the airway is best sized by holding it against the ear lobe and measuring it to the corner of the mouth. With the catheter tip protruding just beyond the end of the airway, both airway and catheter are passed into the mouth, curve upwards, the patient is advised to 'breathe it in', then it is rotated and passed gently into the throat, curve downwards. During insertion it should be held downwards so that it does not press on the soft palate and cause gagging. Introducing the airway is not painful but is often distressing, and patients should be reassured that it will not stop them breathing. Passage of the catheter then proceeds through the airway, as described above.

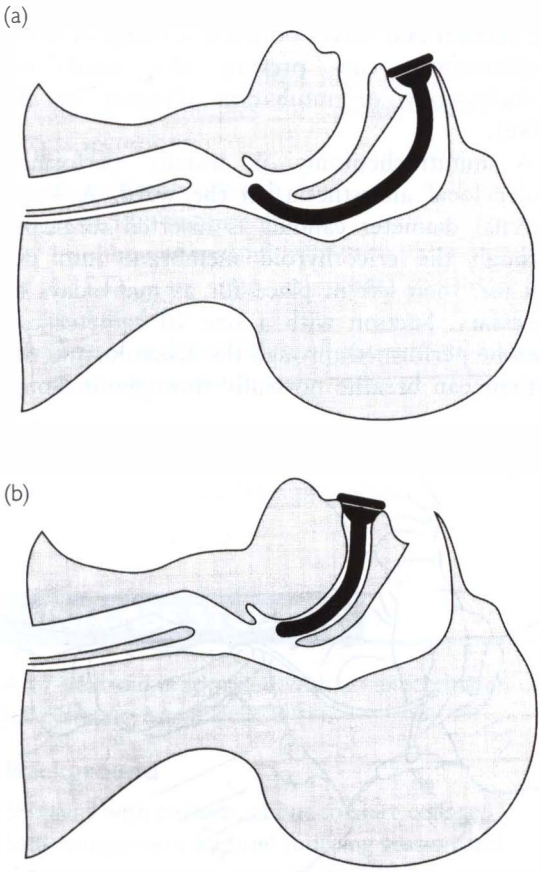


Figure 8.19 (a) Nasopharyngeal airway and (b) oral airway.

Complications

Untoward effects of suction may be subclinical and go unrecognized. Complications include the following.

- Airway mucosa is exquisitely sensitive and is damaged by both passage of the catheter (Swartz, 1996) and pull from the vacuum (Kleiber *et al.*, 1988), exacerbated by poor technique. Damage can be tantamount to a crude biopsy, leading to bleeding and up to 50% reduction in mucociliary transport (Landa *et al.*, 1980), with short- and long-term effects.
- Infective organisms find an easy target once the protective mucosa is damaged.
- The vacuum can cause atelectasis.
- Sustained hypoxia can result from atelectasis, removal of oxygen, enforced apnoea (Petersen *et al.*, 1979) and increased oxygen demand.
- Hypoxaemia or irritation of the vagus nerve can cause arrhythmias, bradycardia or unstable BP. Stress can cause tachycardia.
- Laryngospasm is a rare but dangerous complication. If the patient stops breathing and the catheter feels stuck, the crash team should be called. Laryngospasm may be relieved by gentle positive pressure via a mask and oxygen from the crash trolley, or intubation may be necessary (Leisure *et al.*, 1995).

Precautions

- Pharyngeal suction is contraindicated if stridor is present because of the risk of total airway obstruction.

- Relative contraindications are an unstable cardiovascular system, undrained pneumothorax, haemoptysis of unknown origin and acute face, neck or head injury.
- If there is cerebrospinal fluid leak after basal skull fracture, an oral airway should be used because there is a risk of infection if organisms are dislodged and come into contact with cerebrospinal fluid.
- Bleeding may occur in patients who have clotting disorders or are receiving heparin or thrombolytic drugs.
- If the patient has pulmonary oedema, suction does not help the condition and will remove surfactant if performed repeatedly.
- Suction aggravates bronchospasm but so too does excess mucus.
- Following recent pneumonectomy or lung transplant, the catheter should not be taken beyond the pharynx in case it impinges on the bronchial stump or anastomosis.
- After recent oesophagectomy with a high anastomosis, or with a tracheo-oesophageal fistula, the catheter may miss the trachea and enter the oesophagus. Insertion should therefore not be beyond the pharynx, or a minitracheostomy can be requested.

Clinicians should wear a visor (or mask and goggles) to prevent cross-infection because the patient may be infected with *Pseudomonas*, MRSA (p. 320), TB, HIV or other invisible bug.

NASOPHARYNGEAL AIRWAY

A nasopharyngeal airway (Figure 8.19a) can be used for patients who need frequent suction, but insertion is painful and sinus infection is a risk. Size 6 mm is usually suited to women and 7 mm to men. The size is correct if the airway can be slightly rotated inside the nose. It is lubricated with aqueous or lignocaine gel before insertion, passed gently into the largest nostril, directed along the floor of the nose parallel to the hard palate, then left in place for a maximum 24 hours, after which if necessary it can be cleaned and re-inserted. The tip rests behind the tongue just above the epiglottis. A safety pin across the top prevents it disappearing into the patient. It should not be used in patients who have polyps, congenital deformities or old fractures of the nose, CSF leak or bleeding from the nose or ear (Jenkin, 1996).

MINITRACHEOSTOMY

Minitracheostomy: small opening into the trachea to facilitate suction.

Minitracheotomy: procedure to create a minitracheostomy.

A relief for both physiotherapist and patient has been the advent of the minitracheostomy (Figure 8.20), which allows access for safe and comfortable suction and leaves minimal scarring. A minitracheostomy can prevent the need for bronchoscopy or intubation (Preston *et al.*, 1986).

A minitracheotomy is usually performed under local anaesthesia on the ward. A 4 mm internal diameter cannula is inserted surgically through the cricothyroid membrane into the trachea, then left in place for as many days as necessary. Suction with a size 10 catheter can then be performed through the aperture, and the patient can breathe normally throughout. Some

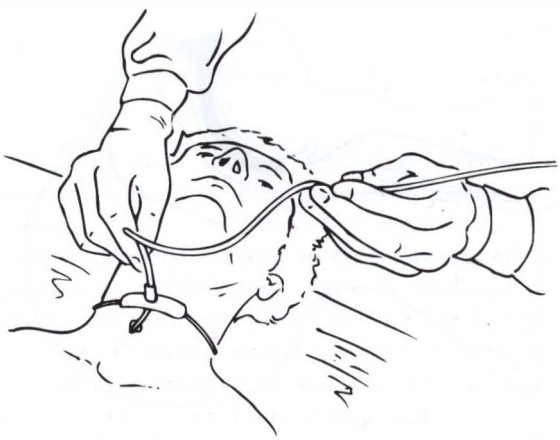


Figure 8.20 Minitracheostomy.

secretions are too thick for a minitracheostomy, although saline instillation may be helpful. A spigot protects the airway when the tube is not in use.

A minitracheostomy tube is uncuffed and preserves the function of the glottis so that natural humidification is maintained and the patient can cough, speak, eat and breathe spontaneously.

Minitracheotomy is often performed later than optimal. The physiotherapist can act as instigator to ensure that it is used early enough to be most effective. Prophylactic placement during surgery is useful for patients at high risk of postoperative sputum retention (Kirk, 1996).

OUTCOMES

The following can be used to evaluate the effectiveness of techniques for sputum retention or excess secretions:

- the patient's opinion
- ↓ crackles on auscultation
- ↑ volume of sputum
- ↑ S_aO_2, so long as other variables are excluded
- greater independence of patients to manage their own secretions.

Physiotherapists can evaluate their manual techniques through a bronchoscope, if their patient is to undergo this procedure.

MINI CASE STUDY: SP

A 17-year-old is admitted with an exacerbation of his cystic fibrosis (Figure 8.21).

Background

SH: lives with parents, about to start college.
Self-management by brief morning session and longer evening session: ACB, PEP, flutter, postural drainage.
Regular reviews with domiciliary physiotherapist.
Exercise mainly by biking to school, little exercise in holidays.
Frequent admissions.

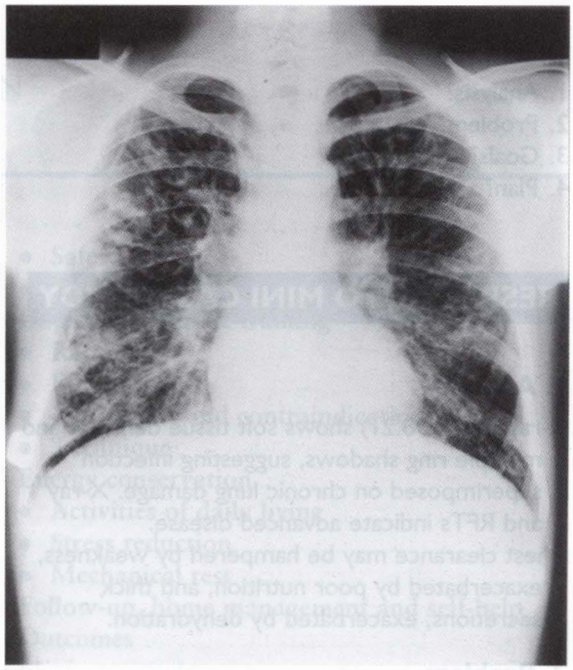


Figure 8.21 SP.

Subjective

Bored.
Not clearing phlegm.
Not hungry or thirsty.

Objective

Hyperinflated chest.
Thin.
Top-up feeding by gastrostomy at night.
IV antibiotics.
Clinically dehydrated.
FEV_1/FVC 67, FEV_1 1.2 L, FVC 1.8 L – not reversed with bronchodilators.
S_aO_2 95%
Spiking temperature.
Auscultation: widespread crackles.
Frequent small, non-productive coughs.

Questions

1. Analysis?
2. Problem?
3. Goals?
4. Plan?

RESPONSE TO MINI CASE STUDY

1. Analysis

X-ray (Figure 8.21) shows soft tissue densities and multiple ring shadows, suggesting infection superimposed on chronic lung damage. X-ray and RFTs indicate advanced disease.

Chest clearance may be hampered by weakness, exacerbated by poor nutrition, and thick secretions, exacerbated by dehydration.

2. Problem

Sputum retention.

3. Goals

Clear chest.

Motivate re. future self-management.

4. Plan

Liaise with team re increasing fluid intake through IV.

Check ACB technique.

If patient is tired or weak, discuss with him which techniques might suit, e.g. postural drainage, percussion, vibrations, IPPB.

Re-educate cough: suppress ineffective coughs, cough only when secretions accessible.

As patient improves, start daily gym sessions.

Check effect of car fumes when biking. Consider mask.

Liaise with domiciliary physiotherapist.

LITERATURE APPRAISAL

Are the following statements problem-based? Are they evidence-based?

1. 'Deep breathing exercises have been proposed to assist the tachypnoeic patient.'
2. 'IPPB is claimed to be useful in delivering aerosolized bronchodilators.'
3. 'Available evidence suggests that postural drainage and controlled coughing or FET may be the most effective components'.

Eur. Respir. J. 1993; 3: 353–355

FET = forced expiration technique, predecessor to ACB.

RESPONSE TO LITERATURE APPRAISAL

1. A tachypnoeic patient's problem is increased WOB, not loss of lung volume. Deep breathing is counterproductive for breathless patients.
2. The words 'claimed to be' are unreferenced. IPPB is an expensive and inefficient way of delivering medication.
3. Where is the 'available evidence'?

RECOMMENDED READING

Anstey, K. H. and Roskell, C. (2000) Hydrotherapy: detrimental or beneficial to the respiratory system? *Physiotherapy*, **86**, 5–13.

Atwood, C. W. (1998) Positive pressure therapy. *Respir. Care*, **43**, 307–317.

Bremner, H., Carriere, B. (1999) Evaluation of breathing mechanisms in patients with pelvic floor dysfunction. *WCPT Proc.*, p. 578.

Bye, P. T. P., Alison, J. A. and Regnis, J. A. (1997) Exercise performance and rehabilitation in CF. *Crit. Rev. Phys. Rehabil. Med.*, 9, 1–33.

Houtmeyers, E. and Gosselink, R. (1999) Regulation of mucociliary clearance in health and disease. *Eur. Respir. J.*, **13**, 1177–1188.

Irwin, R. S., Boulet, L.-P. and Cloutier, M. M. (1998) Managing cough as a defense mechanism and as a symptom. *Chest*, **114**, 133S–179S.

Pryor, J. A. (1999) Physiotherapy for airway clearance in adults. *Eur. Respir. J.*, **14**, 1418–1424.

Schöni, M. H. (1989) Autogenic drainage. *J. Roy. Soc. Med.*, **16**(suppl.), 32–37.

Williams, M. T. (1995) Chest physiotherapy and cystic fibrosis. *Chest*, **106**, 1872–1882.

9 PULMONARY REHABILITATION

SUMMARY

INTRODUCTION

> *Pulmonary rehabilitation is the only approach to chronic lung disease short of lung transplantation that improves the long-term outlook for these patients.*
>
> Tiep, 1991

Rehabilitation for people disabled by breathlessness is a neglected area of health care but one of the most rewarding aspects of physiotherapy. It is neglected because of a widespread attitude that patients have reached a dead end. It is rewarding because it can improve independence for people who have become entangled in a web of inactivity and helplessness. Physiotherapists themselves may feel helpless when faced with someone who has uncontrolled breathlessness, lungs like tissue paper, a pessimistic outlook, an unglamorous disease and no nice straightforward problem, such as excess secretions, which can be dealt with by time-honoured techniques.

Pulmonary rehabilitation should be integral to the management of people with chronic respiratory disability (Griffiths *et al.*, 2000), not an optional extra, but its provision in the UK lags behind the rest of Europe and the US (Steiner *et al.*, 2000). The need is greater now that patients are being discharged from hospital 'quicker and sicker'.

Rehabilitation does not reverse lung damage but it modifies the disability that derives from it and normally shows greater benefit than medication (Lacasse, 1996). Participants report a sense of well-being due to gaining control over symptoms, especially the fear of breathlessness.

Evidence of the benefits of pulmonary rehabilitation are legion (Appendix E), with some outcomes summarized below:

- ↓ breathlessness by 65% (Votto, 1996)
- ↑ exercise capacity and quality of life, according to 14 trials (Lacasse, 1996), even for severely impaired patients (Griffiths *et al.*, 1996)
- ↓ health care costs (Figure 9.1), with at least

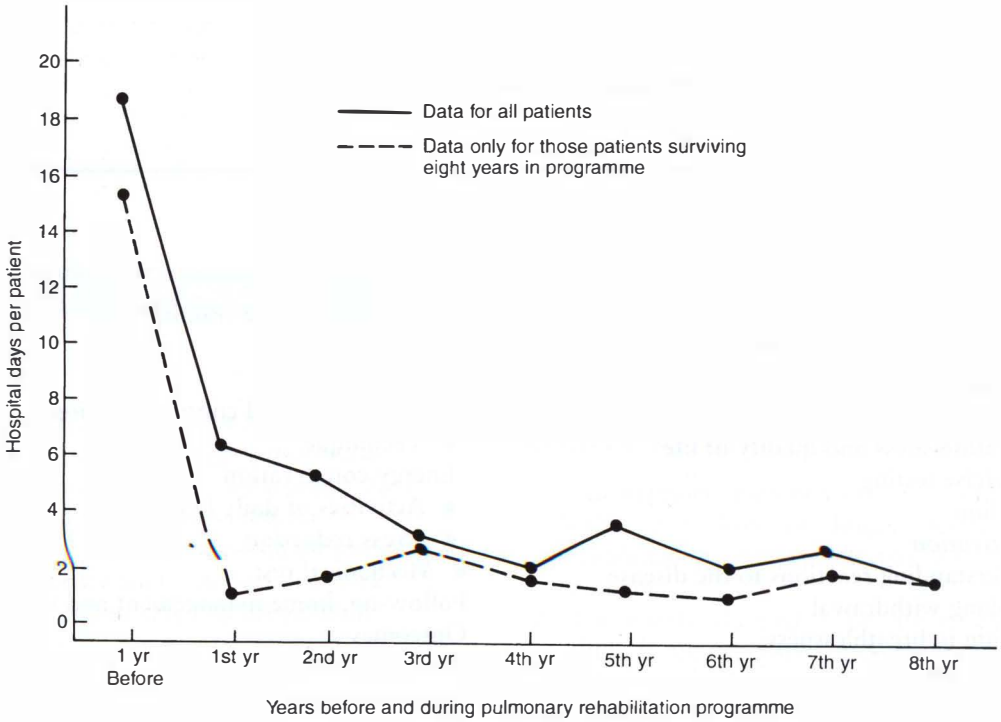


Figure 9.1 Hospital admissions after initiation of pulmonary rehabilitation. (From Hodgkin, J. E., Connors, G. L. and Bell, C. W. (1993) *Pulmonary Rehabilitation: Guidelines to Success*, J. B. Lippincott, Philadelphia, PA)

12 studies demonstrating reduced hospitalization (Reina-Rosenbaum *et al.*, 1997)
- in modified form, ↑ ability to wean from mechanical ventilation (Kozu, 1999).

Quality of life shows the most sustained improvement (Foglio *et al.*, 1999). Positive outcomes depend on realistic expectations, teamwork and follow-up.

Chronic lung disease links physical and psychological factors by a potent blend of breathlessness and chronic disability, and the attitude and encouragement of the rehabilitation team play a major role. Potential team members should be involved at the planning stage so that they have a vested interest in its success. The team typically comprises physiotherapist, occupational therapist, respiratory nurse, physician, dietician and clinical psychologist or social worker, one of whom is the co-ordinator. The contribution of the participant and family is central.

Participants

> ***No patient is 'too sick' or 'too well' to benefit from a pulmonary rehabilitation programme.***
>
> Menier, 1994

A programme includes about eight to 10 participants, with stratification for mild, moderate and severe disease. Suggested selection criteria are the following:

- breathlessness limiting activity
- motivation towards self-help and lifestyle change
- stable condition
- optimal medical management
- adequate ability to hear or communicate
- ability to attend the full programme.

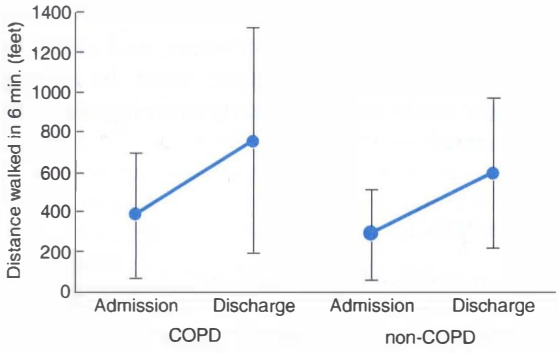


Figure 9.2 Distance walked in 6 minutes before and after exercise training, showing comparable benefits for people with and without COPD. (From Foster, S. and Thomas, H. M. (1990) Pulmonary rehabilitation in lung disease other than COPD. *American Review of Respiratory Diseases*, **141**, 601–604)

COPD is the most common cause of disability in the community (Allison, 1995), but people also benefit if they have asthma (Didour, 1997), cystic fibrosis, interstitial or other restrictive disease, neuromuscular disorders (ACCP/AACVPR, 1997), or following surgery (Chumillas *et al.*, 1998) or long-term institutionalization (Schleifer *et al.*, 1994). Figure 9.2 shows the comparable benefits.

People with restrictive lung disease show benefit in the early stages (Novitch, 1995), although the following precautions are advisable:

- for interstitial disease, monitoring for hypoxaemia during exercise
- for neuromuscular disease, preventing overuse of compensating muscles.

Outcomes are unrelated to lung function, and patients can benefit regardless of severity of disease, including those with chronic hypercapnia (Celli, 1994) or old age (Clark, 1996).

Rehabilitation is especially needed after an acute episode when patients are at their most teachable, and particularly to prevent the stepwise loss of function that follows hospitalization (Peach and Pathy, 1981). Smokers should not be excluded, because helping them to quit can be part of the programme. Patients who suffer depression should not be excluded because depression does not reduce attendance (Garrod *et al.*, 1996). Exclusion criteria are discussed under the sections 'Background information' and 'Safety'.

The set up

The options are:

- an outpatient programme
- an inpatient programme in a dedicated rehabilitation ward
- a discharge programme after exacerbation, either in a pre-discharge ward or at home
- a home-based programme, useful for severely disabled people or as a cost-effective alternative to hospitalization for mild exacerbations
- a community-based programme in a day centre, physiotherapy practice (Cambach, 1997) or other facility that has single-story access and a more upbeat atmosphere than hospital.

Resuscitation and safety training are necessary for programmes held outside hospital.

Pre-discharge wards do not prolong hospital stay (James *et al.*, 1998) and are ideal for frail elderly patients who would otherwise remain in an acute medical ward becoming deconditioned and prey to hospital bacteria.

Home-based programmes are becoming popular because, although they lack peer support, exercise and energy conservation can be adapted to an individual's environment (Garrod, 1998), involve the family, reduce hospitalization (Allison and Yohannes, 1999) and have shown a tripling of the time that benefits are sustained compared to a hospital-based programme (Strijbos, 1996).

Resources

The following are needed for a programme based in a hospital or day centre:

- large, warm room with easily opened

windows, cheerful atmosphere, wall space and non-slip floor, free from dust-collecting furniture, and with acoustics that can cope with choruses of coughing
- comfortable upright chairs
- treadmill, exercise bike, trampoline, quoits, weights, stretchy bands, springs and other gym equipment
- steps
- rollators and high walking frame
- full length mirror
- fan
- demonstration inhalers
- oxygen
- oximeter
- nebulizer system and drugs
- sputum pots and tissues
- audiovisual teaching aids
- handouts, exercise booklets, diaries, writing materials
- name labels to encourage group interaction
- refreshments
- crash trolley and team members trained in life support
- individual transport arrangements such as taxis or cars to avoid the stress and delays of public or ambulance transport.

Financial planning needs to take account of staffing, venue, equipment, stationary, photocopying, telephone and administration time.

Structure and timing

Sessions are arranged typically twice weekly for 6–12 weeks. Once a week tends to be less successful in maintaining motivation between sessions. The initial physiotherapy session, either on a one-to-one basis or with half the group at a time, includes assessment, identification of participant needs, goal setting, breathing re-education and a suggested home programme.

Thereafter, each session would typically be based around an hour's education on a specified topic, a break for socializing, an exercise session and relaxation. A half-way review helps participants to take stock and plan for the end. A final session might include a question-and-answer discussion with all team members, and plans for the future. The programme must be during daylight hours and avoid early mornings or rush-hour travel.

ASSESSMENT

Assessment should take account of:

- respiratory impairment: ↓ lung function, e.g. FEV_1
- respiratory disability: the effect of this impairment, e.g. anxiety or ↓ exercise capacity
- respiratory handicap: social and other disadvantages.

There is much variation between degree of impairment and an individual's disability or handicap.

Rehabilitation is aimed at symptoms rather than the disease process.

Participants are assessed as described in Chapter 2, with factors relating specifically to rehabilitation described below.

Background information

The case notes should be scrutinized to check that exercise training is safe. Contraindications include acute disease, symptomatic angina, recent embolism or myocardial infarct, second- or third-degree heart block, deep vein thrombosis and resting systolic BP above 240 mmHg or diastolic above 120 mmHg. Relative contraindications include disabling stroke or arthritis, haemoptysis (depending on the cause), metastatic cancer, unstable asthma, resting heart rate (HR) below 100 and resting systolic pressure above 180 mmHg or diastolic above 95 mmHg (Bach and Haas, 1996). Liaison with the physician is suggested if P_aCO_2 is above 8 kPa (60 mmHg).

People with insulin-dependent diabetes benefit from exercise training, which can also improve glucose tolerance, but extra vigilance is required to identify hypoglycaemia (p. 116). Steroid-

induced osteoporosis is not a contraindication and indeed is an indication for sensible weight-bearing exercise (Inman *et al.*, 1999). People with heart failure usually benefit from exercise training: those with mild disease may take longer to recover from activity and those with more advanced disease require a low-intensity programme (Piña, 1996). People with intermittent claudication can improve their walking distance (Brandsma *et al.*, 1998).

The following drug history is relevant:

- drugs such as beta-blockers render the BP and pulse unreliable for monitoring purposes (p. 334)
- if prescribed and indicated, bronchodilators and anti-angina drugs should be taken before exercise
- steroids should be at the lowest effective dose to minimize muscle weakness.

Respiratory function tests

RFTs are generally unhelpful for outcome measurements. FEV_1 does not relate to disability (Bestall *et al.*, 1999), but it helps distinguish COPD from asthma, and may assist stratification, e.g.:

- FEV_1 > 60% predicted indicates mild COPD: aerobic programme suitable
- FEV_1 40–60% predicted indicates moderate COPD: peripheral muscle strengthening and use of multigym suitable
- FEV_1 < 40% predicted indicates severe COPD: isolated muscle group work and range of movement suitable.

If oximetry is not available, RFTs may provide information on gas transfer, which, if above 55% predicted, indicates that oxygen desaturation is unlikely during exercise (Mak *et al.*, 1993). This suggests that diffusion characteristics play a role in exercise-induced desaturation.

Breathlessness and quality of life

It is helpful to ask participants how breathlessness affects their lives and why they think they are breathless. Participants are often relieved to be asked if their breathlessness is frightening, because this may not have been acknowledged before. Breathlessness can only be interpreted

Box 9.1 Measurements of breathlessness. The activity scale reflects the amount of effort required to induce breathlessness. The Borg scale (Box 9.7a) can also be used.

Visual analogue scale

Greatest breathlessness

No breathlessness

Activity scale

1. Breathlessness with strenuous activity
2. Breathlessness on stairs
3. Breathlessness forcing patient to give up at least one activity
4. Breathlessness forcing patient to abandon most activities or give up work
5. Breathlessness on dressing, or preventing patient leaving home.

and reported by the person experiencing it (Molen, 1995), and Box 9.1 helps them to quantify the symptom. Visual analogue scales are easier for participants when they are vertical rather then horizontal (Molen, 1995). Breathlessness must be explained to participants so that they distinguish it from sensations such as fatigue or chest tightness.

Participants often describe breathlessness in terms of the effect on their lifestyle, and this can be measured functionally. Health-related quality of life (QoL) scales characterize well-being and include the effect of deconditioning caused by a lifestyle restricted to minimize breathlessness (Figure 9.3). Also known as health status or functional status scales, QoL

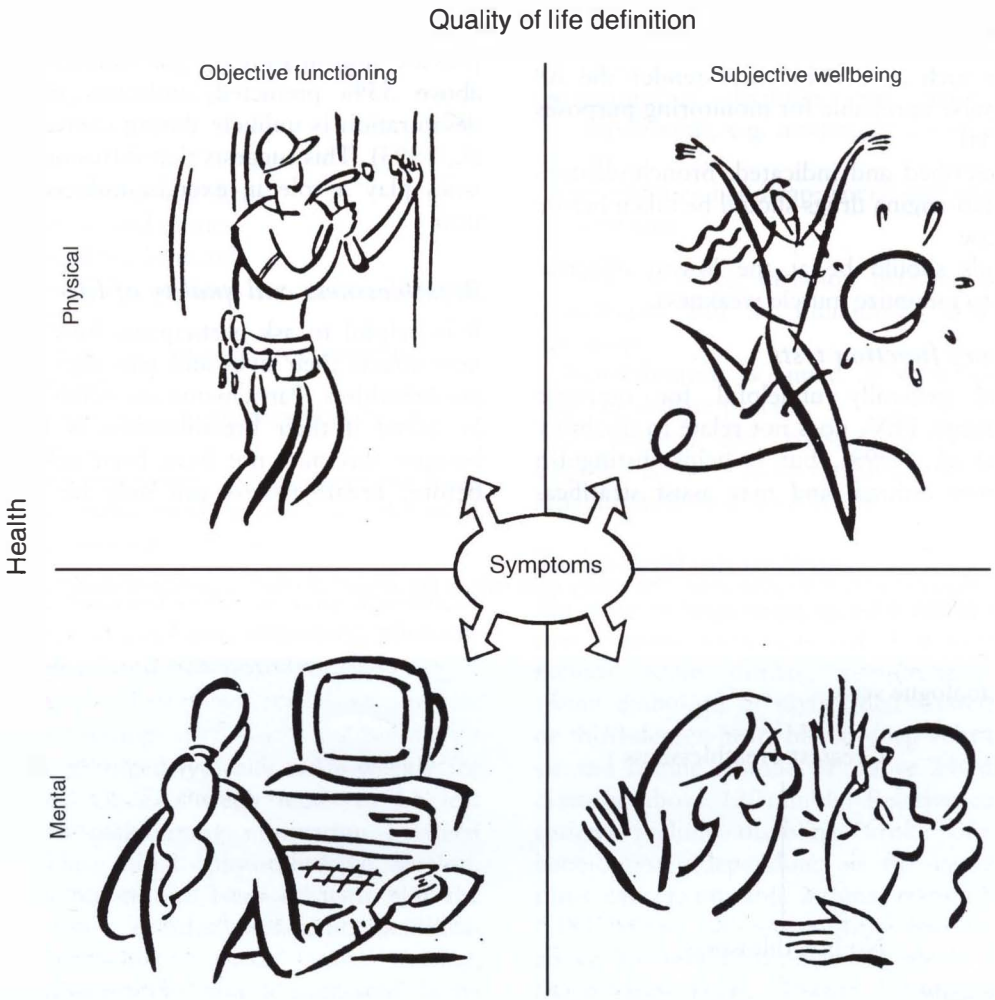


Figure 9.3 A classification scheme for quality of life measures. Physical well-being (top right) relates to the effect of symptoms on vitality and physical health. Mental well-being (bottom right) involves subjective appraisal of factors such as anxiety, depression and social support. Physical functioning (top left) reflects ability to perform specific tasks and includes employment. Mental functioning (bottom left) indicates ability to rise to cognitive and social challenges. (From Muldoon, M. F., Barger, S. D., Flory, J. D. and Manuck, S. B. (1998) What are quality of life measurements measuring? *British Medical Journal*, **316**, 542–545)

scales can be generic or disease-specific, and several examples are available (Harper *et al.*, 1997; Muldoon, 1998; Eakin *et al.*, 1998; Bestall *et al.*, 1999). Figure 9.4 illustrates a QoL scale that can be filled out by patients without assistance.

Quality of life is described by deLateur (1997):

> ***Though difficult to study, it's not impossible;***
> ***Though qualitative, it can be quantified;***
> ***Though subjective, it can be observed;***
> ***Though theoretical, it's certainly not impractical.***

QoL scales are useful if they take account of participants stopping 'wanting' to do what they cannot do, and if they distinguish breathlesness and distress (Wilson and Jones, 1989). Causes of distress include difficulty with conversation (Lee *et al.*, 1998), frustration, embarrassment, dependency, reduced capacity for spontaneity, and the sensation of breathlessness itself. Individual stressors and coping strategies can be identified.

Exercise testing

Exercise testing can be measured objectively by walking or stair-climbing. This gives an accurate indication of progress so long as the patient is not suffering an acute illness, but is not for comparison between patients. Observation of the participant during activity gives information on tension and fatigue.

Tests by the physiotherapist

Oximetry (p. 324) on exercise testing is advisable because resting S_aO_2 is not a predictor of exercise desaturation (Mak *et al.*, 1993). If oxygen is required at rest, an increment of 1–2 L/min is often needed on exercise. The oximeter should be validated under exercise conditions and is not considered reliable when recording values below 90% on exercise (Carone, 1997). Small oximeters can be attached to a belt, the wrist or a finger (Figure 9.5). Ear oximeters may not be valid during heavy exercise under hypoxic conditions (Webb *et al.*, 1991).

Oxygen prescription is based on the flow required to maintain S_aO_2 at over 91% during the last 30 seconds of a 5-minute corridor walk (Hagarty, 1996). Participants are discouraged from becoming obsessed with their oximeter and to maintain awareness of their subjective response to exercise. Transient desaturation is acceptable.

For severely impaired patients, a brief test such as sit-to-stand or stand-up-and-go can be used. For patients who are able, the following are available.

Six-minute distance: For endurance testing, participants are asked to walk for 6 minutes as fast as reasonably possible along a measured flat corridor, following standardized instructions. Stopping to rest is allowed but included within the 6 minutes. Participants should feel at the end that they have performed to their maximum capacity. The physiotherapist can inform the participant when each minute is completed but should not walk alongside because this might influence his/her speed.

The data to record are the 6-minute distance, symptoms, HR and S_aO_2. Modifications are required if there are orthopaedic or neurological problems. Three or four practice walks are needed, with 20 minute rests in between. Repeat tests should be performed at the same time in relation to any bronchodilator drugs. A 15% change in distance is said to be clinically meaningful (Savci *et al.*, 2000).

Stair climbing: The stair climbing test is done under the same conditions and involves counting the number of steps that can be climbed up and down in 2 minutes.

Shuttle: The shuttle test (Revill *et al.*, 1999) is incremental, externally paced, needs only one practice walk and is more reproducible and less dependent on motivation. Participants are asked to walk around a 10-metre oval circuit, with two cones at each end to prevent an abrupt turn (Figure 9.6).

The speed of walking is dictated by a taped bleep which increases in line with the participant raising their speed gradually from 1 to 5 miles

BREATHING PROBLEMS QUESTIONNAIRE

The purpose of this questionnaire is to find out how your breathing problems affect your life.

For each sentence please choose the ending which best describes yourself. Please tick the circle O to show your choice.

Do not spend too long over any one sentence, just tick the ending which is most like you.

Please make sure that you have ticked one box for every sentence

1. Because of my breathing problems, I walk on the flat

Please tick one only ✓

- ○ as fast as normal
- ○ just below normal
- ○ slowly
- ○ very slowly

2. Because of my breathing problems, I can walk on the flat without stopping for

Please tick one only ✓

- ○ less than 20 paces (less than 10 yards)
- ○ about 40 paces (about 20 yards)
- ○ about 80 paces (about 40 yards)
- ○ I never need to stop because of my breathing

3. When I wash myself down I usually

Please tick one only ✓

- ○ dry myself without any problems
- ○ dry myself slowly
- ○ sit and dry off
- ○ need assistance to dry myself

4. If I wanted to, I could do light gardening or DIY

Please tick one only ✓

- ○ as much as I want so long as I take it slowly
- ○ for a short time as long as I can take it slowly
- ○ I could not do these jobs
- ○ Don't know/not interested

Figure 9.4 Breathing problems questionnaire (Michael Hyland and Julia Bott, with permission).

5. I usually feel that I have

Please tick one only ✓

- ◯ more energy than other people of my age
- ◯ as much energy as other people of my age
- ◯ slightly less energy than other people of my age
- ◯ much less energy than other people of my age
- ◯ no energy at all

6. When I am with family or friends I am

Please tick one only ✓

- ◯ not embarrassed by my breathing
- ◯ occasionally embarrassed by my breathing
- ◯ often embarrassed by my breathing
- ◯ nearly always embarrassed by my breathing

7. When I am at social gatherings my breathing problems mean that

Please tick one only ✓

- ◯ I go right in and enjoy myself
- ◯ I go in but keep an eye on where the door or window is
- ◯ I stay on the edge or near a window or door
- ◯ I never go to social gatherings

8. On average, my breathing problems usually keep me awake at night

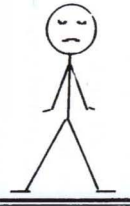
Please tick one only ✓

- ◯ hardly ever at all
- ◯ up to half an hour
- ◯ about one to two hours
- ◯ most of the night

9. My breathing problems

Please tick one only ✓

- ◯ never make me depressed
- ◯ sometimes make me depressed
- ◯ often make me depressed
- ◯ nearly always make me depressed



10. My breathing problems

Please tick one only ✓

- ◯ never make me worried or anxious
- ◯ sometimes make me worried or anxious
- ◯ often make me worried or anxious
- ◯ nearly always make me worried or anxious

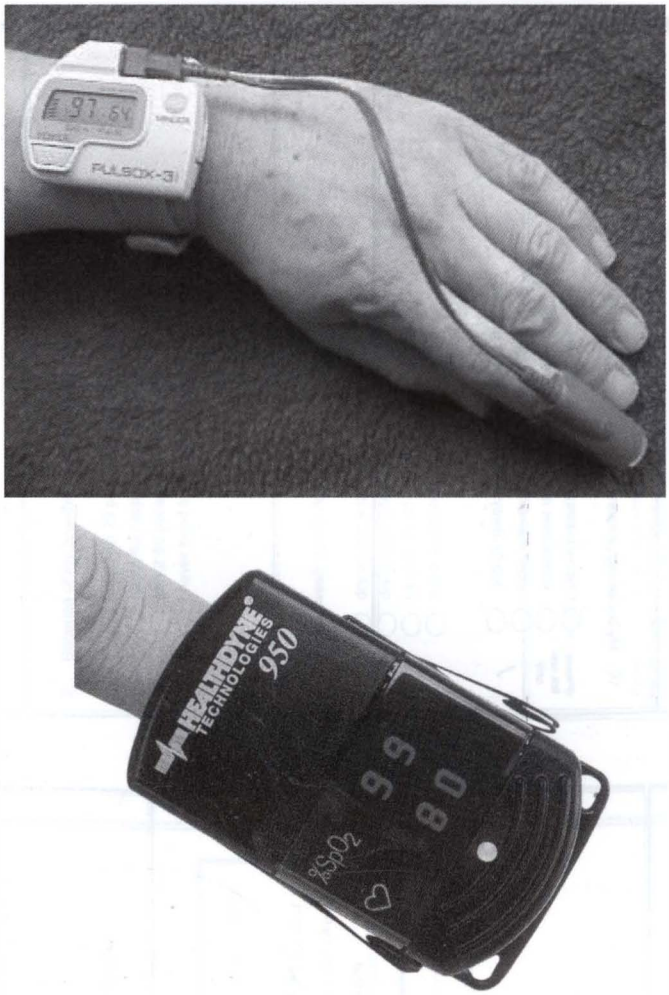


Figure 9.5 Portable oximeters (see Appendix C for manufacturers).

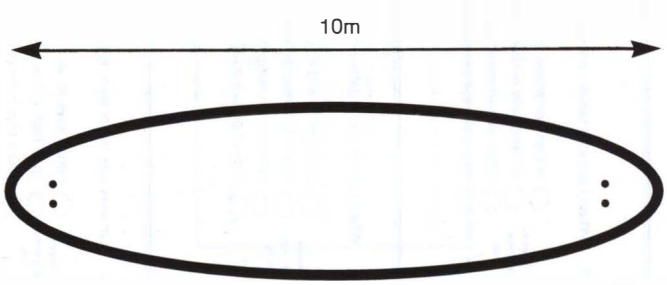


Figure 9.6 Shuttle walking test.

per hour. The tape (Appendix C) gives standardized instructions, and no verbal encouragement is given. The physiotherapist walks alongside for the first minute to discourage the participant from exceeding the initial speed. Thereafter, if the cone is reached early the participant waits for the beep before continuing. The end point is when symptoms prevent the participant completing a circuit in the time allowed. Ideally the maximum should be reached within 10–15 minutes. Half-an-hour's rest is needed after the practice test. The shuttle is closely related to maximum oxygen consumption ($\dot{V}O_{2\ max}$) (Singh *et al.*, 1994). Some participants have trouble co-ordinating the cones with the beep.

Tests in the laboratory

Exercise testing based on treadmill-walking or cycle ergometry is unfamiliar to participants, unreliable in relation to everyday activity (Mak *et al.*, 1993) and less related to exercise capacity than breathlessness (Wijkstra, 1994). However, measurable workloads can be imposed in the laboratory while monitoring minute ventilation, CO_2 output, HR, BP, S_aO_2, blood gases and oxygen consumption ($\dot{V}O_2$). This helps to highlight the interaction between various systems involved in oxygen delivery to the tissues. An ECG stress test detects myocardial ischaemia by identifying ST segment changes.

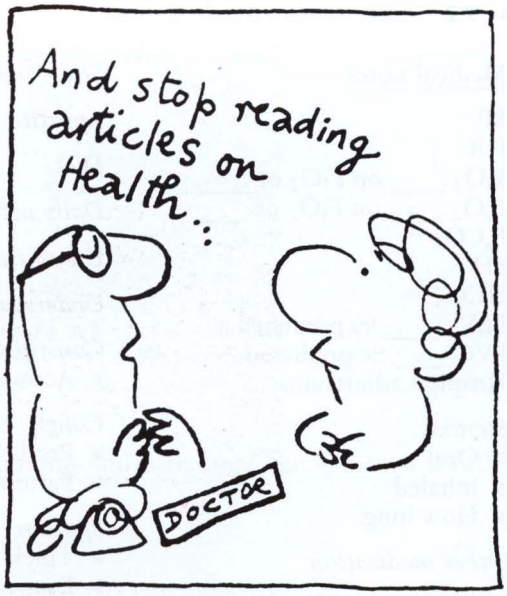
Exercise testing can help determine the cause of exercise limitation. Respiratory disease is likely if breathlessness is the limiting factor. If a person reaches the anaerobic threshold early, i.e. at less than 40% predicted $\dot{V}O_{2max}$, or if maximum predicted HR is reached early, limitation is probably due to cardiovascular disease.

A checklist can be made up from any of the suggestions in Box 9.2 and selected parts used for initial assessment, interim assessment and final outcome.

EDUCATION

Real education must entail emancipation: liberating people to make their own decisions on their own terms.

Fahrenfort, 1987



Education is the most cost-effective aspect of rehabilitation (Tougaard *et al.*, 1992), and underpins all other components. It increases participants' confidence and reduces uncertainty and fear (Small and Graydon, 1992), whereas ignorance can breed a catastrophizing of life events. Most participants want to know as much as possible about their condition but are often reluctant to ask questions in a clinical setting. A rehabilitation programme provides time and a non-threatening atmosphere for discussion.

Age does not itself hinder intellectual ability, but elderly participants may need time for processing information, and hypoxaemia may impair memory. Retention of information is optimal if:

- the room is free of distractions
- the teaching plan is set out clearly
- the most important points are made first
- teaching sessions are brief
- language is simple and jargon-free
- advice is specific rather than general
- information is reinforced regularly throughout the programme
- booklets and handouts are included

Box 9.2 Assessment for rehabilitation

Medical notes

BP
HR
S_aO_2 ____ on F_IO_2 of ____
P_aO_2 ____ on F_IO_2 of ____
P_aCO_2
pH
HCO_3^-
FEV_1 ____ % predicted
FVC ____ % predicted
Hospital admissions

Steroids
- Oral
- Inhaled
- How long

Other medication

Relevant medical history
- Hypertension
- Angina
- MI
- Heart failure
- Peripheral vascular disease
- Musculoskeletal disease
- Neurological disease
- Diabetes
- Osteoporosis
- Other

Social history
- Stairs
- Family support
- Social services
- Employment, hobbies

Subjective

Fatigue

Sleep

Depression

Anxiety

Panic

Frustration

Ability to relax

Stress incontinence

Appetite

Diet

Daily activity level

Use of transport

Understanding of the disease

Constipation Y/N
- Action taken

Cough Y/N
- Productive/dry
- Pattern, e.g. am/pm

Sputum Y/N
- Thick/thin
- Easy to clear Y/N

Smoker Y/N
- How many
- Understanding of effects

Ex-smoker Y/N
- How many
- When stopped

Self-management
- Symptoms
- When to call GP

Home oxygen
- Hours per day
- Cylinder/concentrator
- Flow rate
- On exercise Y/N
- When eating Y/N
- When breathless Y/N
- At night Y/N

Factors limiting mobility
- Breathlessness
- Fatigue
- Weakness
- Chest pain
- Other pain
- Imbalance
- Dizziness
- Poor eyesight
- Use of oxygen
- Loss of confidence
- Footwear
- Other

Does breathlessness limit:
- bending
- reaching over head
- climbing stairs
- shopping
- housework
- sleeping
- dressing
- bath/shower
- toilet
- conversation
- sexual relations
- preparing meals
- eating
- feeling angry
- being angry
- playing with children
- going for a walk
- walking around at home
- walking with others on the level

Which of the above are most important for the patient?

What would the patient most like to do but cannot because of breathlessness?

Expectations

Goals

Objective

S_aO_2
- Rest
- Talking
- After walk or shuttle

F_IO_2 to maintain S_aO_2 > 91% during last 30 s of 5 min walk.

Breathing pattern

Box 9.3 Topics for education and discussion

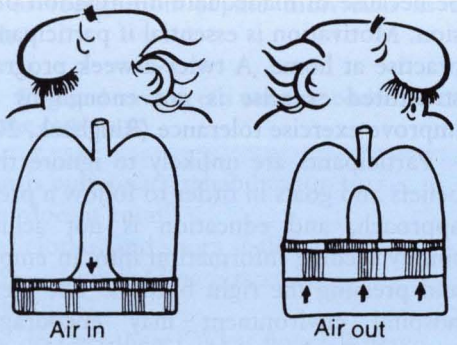


Figure 9.7 How we breathe. (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

How we breathe (Figure 9.7), the relation between symptoms and pathology, the nature of breathlessness

Medical tests, procedures, interpretation of results

Oxygen therapy: effects, side effects, equipment, consequences of non-adherence

Drug therapy: effects, side effects, inhalers and nebulizers, consequences of non-adherence, records and charts to aid memory

Fluids and nutrition

Smoking cessation

Relation between symptoms and interventions such as relaxation, breathing re-education, chest clearance, exercise training and energy conservation

Self-assessment, symptom management, recognition and management of exacerbations, recognition of the need for medical attention, e.g. change in symptoms or new symptoms

Prevention of infection, e.g. avoidance of people with respiratory infections, influenza vaccination

Management of the environment, e.g. indoor exercise if outside air is polluted, covering nose and mouth when exercising in cold weather, bowls of water by radiators, prevention of dust

Community resources, benefits and entitlements (with corresponding advice to welfare agencies on the needs of the 'invisible' respiratory patient)

Vocational guidance to improve self-esteem and social participation while avoiding jobs or hobbies with respiratory irritants or excess energy expenditure

Tips such as a walking stick to advise motorists of a slow walking pace, advice to carers on simple massage techniques

Advice for participants before visiting the doctor, e.g. write down questions to ask in advance, clarify points that are not understood

Management of panic attacks (p. 304)

Home equipment

Travel tips, including use of nebulizers and oxygen abroad and while travelling (RCP, 1999)

- participants are reminded to check body tension, take medication and practise their breathing: suggestions include memory aids such as stickers on kettles, reminders on toothbrushes, use of dead time such as queues or TV advertisements.

The respiratory nurse teaches the understanding and practical management of oxygen and medication. The dietician identifies individual nutrition problems, suggests six-meal-a-day menus, advises on healthy eating and explains which foods are mucus-forming, gas-forming, constipating or hard to digest. The physician answers medical questions and discusses advance directives in relation to life-prolonging treatment (Heffner, 1996). Participants are reminded that rehabilitation is not a course of treatment to make them better but more of a lifeplan. Educational topics are suggested in Box 9.3.

Motivation

> ***The therapist-patient relationship can succeed or fail, depending on the care that the therapist takes in understanding the needs and circumstances of her patients.***
>
> Walker, 1995

Motivation is the best predictor of the success of rehabilitation (Brannon *et al.*, 1998, p. 346). Over 70% of patients with COPD do not adhere to treatment (Mellins *et al.*, 1992), which may be because of inadequate information or depression. Motivation is essential if participants are to practise at home. A twice-a-week programme of structured exercise is not enough by itself to improve exercise tolerance (Ringbaek, 2000).

Participants are unlikely to ignore their own beliefs and goals in order to follow a prescriptive approach, and education is not achieved by simply feeding information into an empty vessel and pressing the right buttons. The hierarchical hospital environment may encourage some patients to take up the sick role and assume that the experts know best. This apparent compliance is counterproductive in the rehabilitation process. Motivation is enhanced by participants taking responsibility for their own management.

Factors that increase motivation are:

- clear advance information (Box 9.4) in large print
- realistic expectations
- active participation, e.g. self-monitoring, invitations to question, comment, design programmes, contribute ideas
- verbal commitment from participants
- praise, warmth, humour, honesty and responsiveness from the rehabilitation team
- family involvement
- focus on health rather than disease
- short simple regimes (Mellins *et al.*, 1992)
- understanding the rationale of each component
- early success, reinforced by progress charts
- access to notes (McLaren, 1991)
- continuity of personnel
- certificate of completion.

Factors that decrease motivation are:

- fatigue
- fear of failure
- anxiety or depression
- advice that is inconvenient or difficult to follow
- embarrassment
- boredom, e.g. repetitive exercise, 12-minute walking test, waiting for transport

- coercion
- lack of recognition of the individual as a whole.

Box 9.4 Suggested contents of a welcome booklet sent to prospective participants

- What is pulmonary rehabilitation for?
- What does it entail?
- What clothes and shoes shall I wear?
- Do I need my reading glasses or medication?
- How do I get there? What floor? Where do I report?
- Who will be there?
- Can I bring someone with me?

Most participants are enthusiastic learners, and liberal use of teaching aids, using pictures rather than graphs, can explain the disease process in a way that is enjoyable. A large-print diary is useful to log daily exercise, symptoms, feelings, diet, drugs and side effects, action taken and the results. The diary can include a negotiated written contract stating achievable and functional goals, the time to achieve them, and obligations of the participant and the rehabilitation team. Achievement of the first goal gives participants a motivating boost.

The relationship between participants can facilitate motivation. They may share transport to the sessions, swap ideas and exercise together outside classes. Participant beliefs and individual attitudes are central to motivation. One participant, for example, did not use her oxygen until she was told of the difficulty for her heart in 'pumping thick blood'.

Understanding reactions to the disease

> ***Psychological dimensions carry a remarkable weight in the way patients ... face rehabilitation.***
>
> Lera, 1997

Depression is often accepted as a manifestation of the disease and not addressed, even though the symptoms of sleeplessness, poor appetite, social isolation and low energy can sabotage rehabilitation. Anxiety is another frequent accompaniment to breathlessness and uncertainty. When depression and anxiety coexist, patient and physician often fail to recognize the depression (Dudley *et al.*, 1980??).

Participants may not have considered the relevance of psychosocial factors to their disability, despite their myriad of feelings. Care should be taken with language because the word 'psychological' may be interpreted as a psychiatric disorder and the word 'disabled' is difficult for people who have not thought of themselves in this context. To use emotionally charged words without preparation is like using the word 'stump' to a new amputee.

It should be explained that feelings are closely connected with breathing for all people, including those with normal lungs, that it is natural for breathless people to feel depressed and anxious, and that this is an expression of humanity, not weakness.

Anxiety is exacerbated by fears, e.g. that death will be by suffocation, a common misconception that can contribute to panic attacks. Most respiratory patients will die after lapsing into a coma.

Topics to discuss include:

- identification of stressors
- recognition and management of depression (without taking comfort in smoking!)
- living with limitations (Figure 9.8)

Figure 9.8 Lifestyle adaptation (From Leboeuf, C. (2000) *A Practical Approach to the Late Effects of Polio*, British Polio Fellowship, Middlesex, with permission.)

- the importance of activity and maintaining a social life
- relationships with partner and family, including concepts of guilt, dependence and resentment
- relationships with others: coping with embarrassment or perceived stigma, how to explain about using oxygen or walking slowly, how to cope when the legitimacy of an invisible condition is doubted, how to ensure that adequate time is allowed for speaking.

Relationships may be affected by lack of spontaneity because breathless people often cannot waste breath in expressing anxiety, anger, love or happiness. This emotional straightjacket can isolate partners from each other. Any family member can be welcomed to educational sessions.

It is natural for chronically disabled people to harbour resentment at the loss of their dreams. This may be projected on to their family or any of the rehabilitation team. Allowing patients to talk gives them an opportunity to understand this process. If there is no appropriate outlet, the resentment turns inward and augments depression. People who are depressed usually respond to a receptive ear. Time is always needed when working with troubled people, but this is time well invested by a member of the team with whom the participant feels comfortable, because little progress can otherwise be made.

Self-esteem and sexuality are closely linked, and loss of sexual expression reinforces low confidence. Education can help discriminate between the effects of myth, illness and drugs on sexual activity. Myths perpetuated by society include the expectation that elderly people cannot have, do not want or should not want sexual relations, and that disabled people are sexually neutered. Illness causes deconditioning, poor self-image, fatigue or breathlessness. Alternative positions may be helpful, although some men find the change to a passive position unsettling at first. Kissing may be difficult for breathless people. Drugs, such as certain antihypertensives, antiulcer and cardiac drugs, can affect sexual function, in which case drug review is indicated.

Many physiotherapists are comfortable to listen to patients talking about feelings, but referral to a an appropriate agency may be required because this can be a complicated area. Denial, for instance, has been shown to reduce adherence (Borak *et al.*, 1991), but some level of denial may be necessary as a coping strategy (Bosley, 1996). The relevance of participants' feelings is shown by evidence that attitudes and beliefs bear more relation to exercise tolerance than ventilatory capacity (Morgan *et al.*, 1983).

Smoking withdrawal

> ***Tobacco ... disturbeth the humors and spirits, corrupteth the breath ... exsiccateth the windpipe, lungs and liver.***
>
> Tobias Venner, 17th century physician (Bach and Haas, 1996)

Smoking cessation is the first priority in the management of people with COPD. Some 70% of smokers want to give up (Venables, 1994) but their endeavours are hampered by the tenaciously addictive properties of nicotine (Figure 9.9), as well as less specific obstacles such as

Figure 9.9 The killer weed. (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

comradeship amongst smokers, stress or boredom.

Multiple reinforcements are more successful than a single intervention, and every strategy for encouragement should be employed because failure drains the will. A quit date should be decided, preferably when something unusual is happening and with a reward for success at the end. Goals can be set, low priority cigarettes stopped, a diary (Figure 9.10) initiated, habits

A smoking diary

Keep a smoking diary for two days - one at work and one at home. When it is complete, start asking yourself some questions:

What sort of activities provoke me into having a cigarette? (eg having a cup of coffee, or answering the telephone).

Who provokes me into having a cigarette?

Which cigarettes did I find most enjoyable, and why?

Which cigarettes could I easily have not smoked, and why?

Which cigarettes did I feel I could not have done without, and why?

Time	What were you doing?	Who were you with?	How were you feeling?	How much did you enjoy it?	How much did you need it?

Figure 9.10 Smoking diary. (From Quit, with permission; see Appendix C.)

linked with smoking can be changed, e.g. orange juice instead of coffee and avoidance of passing the cigarette shop.

Alternative strategies can be devised for difficult situations or in case of relapse. Although total cessation is best (Flaxman, 1978), reduced levels of smoking can be an alternative (Klech, 1998) so long as compensatory deep inhalation is avoided.

Plenty of water is advised, and distraction from cravings might include:

- sucking mints, chewing gum or dried fruit, eating an apple
- brushing teeth, taking a shower
- phoning a friend or helpline (e.g. Quitline, Appendix C).

A pre-planned phone call to the participant is helpful a few days after the quit date, when motivation, determination and support from others might be waning.

Advice should not appear patronizing but specific information can be given such as the effects of tobacco on the smoker's family (p. 22), suggestions on alternative uses of the £1000 per year spent on the average British habit, and the fact that, while smoking appears to help clear the chest, it only does so by irritating the airways and creating extra secretions.

Participants need to understand the physical and psychological difficulties of withdrawal but also the pleasures of sweeter-smelling breath and clothes, improved appetite and bank balance, reduced cough and even some recovery of lung function (Hodgkin *et al.*, 2000, p. 356).

Weight gain can be a problem for up to a year, and it is usually wiser to emphasize healthy eating rather than trying to lose weight at the same time. Temporary irritability can strain relationships but should be understood as the body recovering rather than a reason to return to smoking.

Nicotine replacement can double success rates (Tonnesen, 1999) but is not at present available on the NHS, although it would be cost-effective (Stapleton *et al.*, 1999). Up to two patches can be used over 24 hours, or just in the daytime for less sleep disturbance. Side effects include skin irritation, eased by changing the site daily, and mild effects of quitting, which disappear after about 10 days of patch-wearing. For rapid boluses in time of need, nicotine gum, inhalers or nasal sprays can be used in tandem with patches. Gum should not be chewed but compressed with the teeth and left in the mouth for 1 minute. Contraindications to nicotine replacement are:

- pregnancy and breast feeding
- acute MI, unstable angina, severe arrhythmias
- recent stroke
- for patches: some skin problems
- for gum: peptic ulcers.

A variety of drugs are available to assist withdrawal (Covey, 2000), one or other of which may be helpful. Changing to 'light' cigarettes is normally unhelpful because of altered smoking patterns (Klech, 1998). Herbal cigarettes contain no nicotine but produce tar and carbon monoxide.

Other tips include posters (Figure 9.11), acupuncture (Aiping, 1994), hypnotherapy, biofeedback by carbon monoxide monitoring (Jarvis, 1986), group counselling and role play, e.g. asking friends not to smoke around them.

Participants themselves provide ideas for each other and can set up a 'buddy system' by swapping phone numbers. Positive support helps counteract patients' previous experience of being treated as if they should be punished for smoking (maybe some of this punitive energy could be directed at the tobacco companies!).

But – would it not be preferable for a person who is disabled and housebound to continue with one of life's few remaining pleasures? That is the patient's decision. The job of the rehabilitation team is to educate, to explain that it is never too late to stop and then to support patients after they have made their decision.

There's nothing to giving up smoking. I've done it hundreds of times.

Mark Twain

Changes your body goes through when you quit

20 minutes
Blood pressure and pulse rate return to normal. Circulation improves in hands and feet, making them warmer.

8 hours
Oxygen levels in the blood return to normal. Chances of a heart attack start to fall.

24 hours
Carbon monoxide is eliminated from the body. The lungs start to clear out mucus and other debris.

48 hours
Nicotine is no longer detectable in the body. The ability to taste and smell is improved.

72 hours
Breathing becomes easier as the bronchial tubes relax. Energy levels increase.

2-12 weeks
Circulation improves throughout the body, making walking easier.

3-9 months
Breathing problems such as cough, shortness of breath, and wheezing improve. Overall, lung function is increased by 5-10%.

5 years
Risk of a heart attack falls to about half that of a smoker.

10 years
Risk of lung cancer falls to about half of that found in a smoker. Risk of a heart attack falls to about the same as someone who has never smoked.

Based on information from the 1990 U.S. Surgeon General's report.

Figure 9.11 Encouragement to quit. (From Quit, with permission; see Appendix C.)

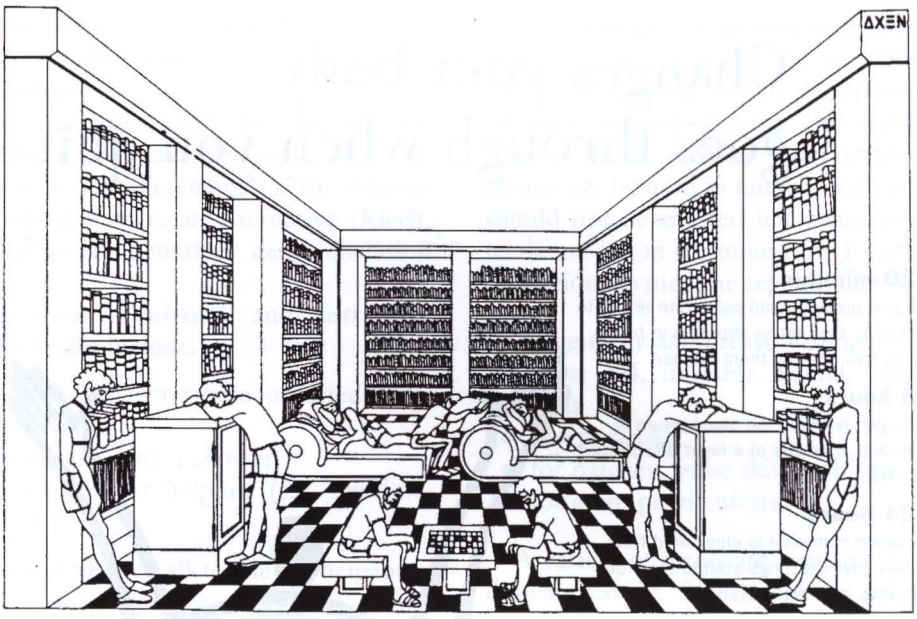


Figure 9.12 Positioning for breathless people (From Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester, with permission.)

REDUCTION IN BREATHLESSNESS

Any of the techniques discussed on page 173 can be taught to participants, including a variety of resting positions (Figure 9.12). An addition for people with chronic breathlessness is mobilization of the thorax. Some participants may need individual attention at first, and thereafter they can choose from various self-management strategies.

Thoracic mobility

Thoracic mobility may be impaired by chronic muscle tension, shortening of anterior chest and shoulder muscles, abnormal mechanics of breathing and a forward head posture. This can add a restrictive element to an obstructive condition and cause pain, sometimes misdiagnosed as pleurisy.

Carr (1993) claims beneficial effects from the following:

- in forward-lean-sitting: Maitland mobilizations to vertebral and scapular joints
- sitting astride a chair to fix the pelvis: passive thoracic rotation
- half-lying with a roll under the thorax: thoracic extension assisted by passive arm elevation
- for some participants who have developed a stiff hyperinflated chest: manual compression on exhalation in a bucket-handle direction
- stretches to muscles around the shoulder.

Kolaczkowski (1989) has developed a series of 40 techniques, some of which have been shown to increase S_aO_2. A video is available (Appendix C).

The normal precautions apply, especially steroid-induced osteoporosis. Handling and positioning must not exacerbate breathlessness. Participants are encouraged to do their own stretching exercises, including side-flexion, rotation and hand-over-head exercises. They are motivated by understanding that expansion of their lungs is hindered by a stiff rib cage.

Box 9.5 shows how participants can mobilize their own thorax. Exercises such as these have

Box 9.5 Flexibility exercises, mostly in sitting

1. Head movements
- While breathing in, look up to ceiling. While breathing out, slowly bring chin down to chest.
- Keeping shoulders still, move your head sideways to bring your ear towards your shoulder. Repeat the other side.
- Turn your head to look over your shoulder. Repeat the other side. Maintain steady breathing throughout.

2. Shoulder girdle circling
Circle shoulders slowly forward, upward, backward, downward. Relax. Repeat in opposite direction.

3. Chest stretch
Hands behind back, breathe in, push chest forward and shoulders back. Breathe out and relax.

4. Back stretch
Lock hands, stretch hands forward at shoulder level, feel stretch between shoulder blades, relax.

5. Arm circling
Hold arm sideways at shoulder height, circle arm in progressively increasing circles for count of 4, then decrease for count of 4. Repeat with other arm. Maintain relaxed breathing throughout.

6. Trunk rotation
With arms folded across chest, keeping pelvis still and knees forward, breathe in. While breathing out, rotate trunk to look over shoulder. Repeat other side. Maintain steady breathing.

7. Trunk rotation
As above, with hands behind head.

8. Trunk extension
With hands behind head, lean over back of (low-backed) chair while breathing in. Return while breathing out.

9. Trunk side-flexion
With hands across chest or behind head, and keeping buttocks flat on the chair, bend from side to side.

10. Trunk flexion
Breathe in gently. While breathing out, bend forward towards toes. Sit up slowly while breathing in.

11. Pelvic circling
Standing with hands on hips: rotate pelvis in slow circle.

12. Calf stretch
Standing with one foot in front of the other: lean forward and bend front leg, keeping back heel down. Repeat with other leg.

13. Pectoral stretch
Standing holding the inside of a door frame: while breathing in, step through frame with one foot and feel stretch across front of chest. While breathing out, step back. Repeat with other foot.

14. Arms up
Standing with elbows straight: while breathing in, lift rolled-up newspaper above head in one hand, pass to other hand. While breathing out, bring arms down.

shown benefits such as reduced breathlessness and increased vital capacity (Kakizaki *et al.*, 1999).

Participants should be reminded of the following:

- avoid straining, pain or discomfort ('stretch is good, pain is bad')
- keep movements relaxed and fluid, allow arms and legs to move independently of the body
- maintain an efficient and relaxed breathing pattern.

EXERCISE TRAINING

> ***Mr Smith becomes short of breath when he exerts himself. Mr Smith has lung disease and I know that lung disease causes shortness of breath. Therefore Mr Smith's exercise limitation is due to his lung disease.***
>
> Quoted by Schwartzstein, 1992

This myth that breathless people cannot benefit from exercise training is at last eroding under the onslaught of evidence to the contrary. But exercise training is still not provided as an integral part of respiratory care for many patients, perhaps because of an assumption that patients cannot reach a training threshold if exercise is limited by breathlessness. But:

- Rampulla (1992) has shown that more COPD patients stop exercising because of fatigue than breathlessness.
- Donner and Howard (1992) have shown that for people with moderate disease, cardiovascular or peripheral muscles are the main limiting factors, especially when chronic hypoxia impairs muscle function (Wuyam *et al.*, 1992).
- A significant limiting factor is the patient's fear of breathlessness rather than breathlessness itself. Success relies on desensitization to breathlessness so that patients can break out of their vicious cycle of breathlessness and deconditioning (Figure 9.13).
- Exercise itself acts as a form of desensitization to breathlessness (Belman *et al.*, 1991), which might explain evidence that exercise training causes a greater reduction in the distress associated with dyspnoea than in the intensity of dyspnoea itself (Carrieri, 1992).
- Pulmonary rehabilitation aims at endurance, flexibility and some strength, which emphasize factors such as nutrition and physical fitness, rather than maximal capacity, which depends on ventilation, gas exchange and oxygen delivery to the respiratory muscles.

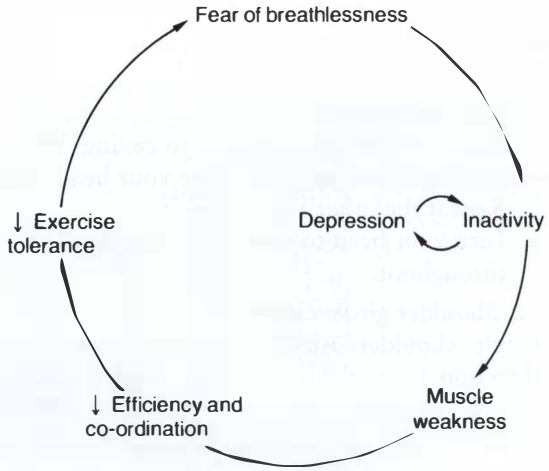


Figure 9.13 Vicious cycle that augments breathlessness in patients with chronic lung disease.

The programme must be individually planned, acceptable to the participant, accessible, safe, show tangible benefits and be designed so that it can be maintained unsupervised at home. Long-term commitment is needed because de-training occurs faster than training.

Effects

The benefits of exercise for people with normal lungs are well-known (p. 23). Extra benefits are found in people who have respiratory disease:

- Respiratory patients who train have shown improved exercise tolerance (Figure 9.2), cardiovascular fitness and raised anaerobic

threshold (Schwartzstein, 1992). Maximal oxygen consumption ($\dot{V}O_{2max}$) can be increased in people with less severe disease, and improved muscle strength can be comparable to that in healthy young people (Simpson *et al.*, 1992). Prior deconditioning means that modest exercise is likely to induce a physiological training effect, even in elderly people (Casaburi, 1992).

- Breathlessness is relieved by improved exercise tolerance, activity-related sensory input and reduced gas trapping because of freer airflow, represented by a minor 7% improvement in FEV_1 (O'Donnell, 1995). The distress component of breathlessness decreases more than the intensity component, showing how desensitization and reduced anxiety have a direct effect on the perception of breathlessness (Bach and Haas, 1996, p. 348).
- A sense of well-being and confidence, along with reduced anxiety and depression, is consistently reported and is greater than objective change (Hodgkin *et al.*, 1993, p. 286).
- Exercise reduces smoking (Russell *et al.*, 1988), BP (Brannon *et al.*, 1998, p. 76) and risk of chest infection (Karper and Boschen, 1993). It promotes relaxation and sleep, stabilizes blood sugar and reduces gut problems (Hodgkin *et al.*, 1993, p. 109).

Mechanism of training

The severity of disease dictates how training improves exercise tolerance. People with moderate COPD ($FEV_1 > 1.2$ L) can reach their anaerobic threshold and develop lactic acidosis, which occurs at a higher percentage of $\dot{V}O_{2\ max}$ (e.g. 80–90% versus 60–70% in those with normal lungs).

In severe COPD ($FEV_1 < 1.2$ L), exercise is commonly limited by ventilatory function and gas exchange abnormalities, and improved exercise tolerance is thought to be due to greater mechanical skill, which reduces the oxygen cost of exercise, a more efficient ventilatory pattern, and desensitization to breathlessness (Ries, 1994). This has been confirmed by Clark *et al.* (1996), who found major improvements in endurance with unchanged cardiorespiratory fitness, and suggested a further mechanism of improved neuromuscular coupling.

People with COPD generate lactic acidosis at low exercise levels. Hypercapnic patients may find it particularly difficult to work above the anaerobic threshold because CO_2 is generated by lactic acid buffering, and fatigue may be caused by the excess ventilation required to compensate for metabolic acidosis (Casaburi, 1992).

Safety

Breathlessness is not an adequate indicator to limit exercise, because patients can drop their oxygen saturation to 70% without increased dyspnoea. Oximetry during assessment is advisable, and can be used as biofeedback and reassurance.

For hypercapnic patients, P_aCO_2 will show an increase during exercise because of extra metabolism, and if it does not fall back to normal afterwards, training intensity must be reduced.

Some 50% of COPD patients aged over 50 have cardiovascular disease (Haas and Haas, 1990, p. 133). Breathlessness may prevent exercise from stressing the cardiovascular system, but the boundaries of safety should be defined. Guidelines for cardiovascular patients are the following.

- Exercise should be terminated if there is failure to increase heart rate (HR) or failure to raise systolic BP at least 10 mmHg above the resting level
- For participants on beta-blockers such as propranolol, resting BP cannot be used as a predictor of BP during exercise (Potempa *et al.*, 1991)

See also cardiac rehabilitation, p. 289.

Practical safeguards for all participants are:

- scrutiny of the notes following comprehensive medical screening
- detailed explanations and education on self-monitoring, including identifying the appro-

priate balance of feeling 'breathless but not speechless'
- optimum nutrition to prevent depletion of muscle proteins and minimize fatigue (Rampulla, 1992), a factor underscored by the response to exercise training being dependent on nutrition (Palange *et al.*, 1998)
- optimum fluid and drug therapy
- treatment of any anaemia
- isotonic rather than isometric exercise to reduce the risk of hypertension, impaired blood flow and fatigue
- for participants who put their hands in their pockets to support the shoulder girdle, advice to maintain the support with hands outside their pockets
- discouragement of competition
- steady exercise with no rushing at the start or finish
- adequate rest, with placement of chairs at intervals
- termination of exercise if there is angina, cyanosis, pallor, cold clammy skin, fatigue, confusion, headache, dizziness, nausea, desaturation below 80% or BP rise to more than 250 mmHg systolic or more than 120 diastolic (AARC, 1992).

Further details are given by Olivier (1998) and comprehensive risk assessment by Pollock and Wilmore (1990).

Technique

Even when a conventional training response is not anticipated, the three principles of training are followed:

- *overload*, i.e. intensity must be greater than the muscle's normal load
- *reversibility*, i.e. cessation of training loses the benefit gained
- *specificity*, i.e. only the specific activities practised will show improvement.

Endurance training, comprising low-resistance, high-repetition exercise, is more suitable for respiratory patients than strength training. Endurance training forestalls the onset of inefficient anaerobic metabolism and enhances the use of oxygen.

Preliminaries

Participants are reminded that exercise is not synonymous with pumping iron. They set their own goals, such as being able to walk to the pub. Inpatients should be dressed in their day clothes and all participants should have cleared their chests of secretions before exercising.

Warming up in a group allows participants to enjoy movement for its own sake, distracting them from preoccupation with breathlessness and reducing the seriousness associated with a therapeutic environment. Five minutes may be sufficient for respiratory patients. Stretching exercises can be chosen from those in Box 9.5.

Music may be used for pleasure but not as a metronome, and participants can be invited to bring their own favourites. Participants should feel free to move at their own pace or not join in if they wish. For severely breathless people, the warm up period should be brief and may simply mean starting their modified exercise training slowly.

During activity, participants are discouraged from rushing or breath-holding, which can disturb the breathing pattern and increase BP (Linsenbardt *et al.*, 1992). They are encouraged to take comfortable strides and maintain a rhythmic quality of movement.

Exercise prescription

Four components make up the exercise prescription: mode, intensity, duration and frequency.

The *mode* of exercise relates to the participants' lifestyles. Many choose walking, stair-climbing or occupation-based exercise. Some prefer the stationary bike or treadmill because they feel in control, can use oxygen easily and have support for their shoulder girdle. About 85% of body weight is supported by a bike, and large muscle groups can be exercised with less

Box 9.6 Circuit exercises

Each exercise is continued for 1 minute. Repetitions are recorded by participants on individual clipboards. Participants rest between each exercise at 'breathing control stations' until breathlessness returns to baseline, usually in about 1 minute. Instructions are best pinned to the wall.

1. **Knee tensing.** Long-sitting on plinth, bed or sofa with knees on a pillow or coffee jar: tighten thigh muscles, hold for count of 4, relax. Repeat with other leg. Maintain steady breathing throughout.
2. **Biceps curl.** Sitting with elbows on table: lift weight or bag of sugar, lower slowly. Repeat with other arm. Maintain steady breathing throughout.
3. **Heel–toe.** In sitting, raise alternate heels and toes, in time with breathing.
4. **Ball throwing and catching**
5. **Quadriceps exercises.** In sitting, straighten one knee, hold for count of 4, lower leg slowly. Repeat with other leg.
6. **Towel wringing.** In sitting, wring towel tightly, hold for count of 4. Slowly untwist towel. Keep breathing steadily throughout.
7. **Lift ups.** In sitting, inhale gently. While breathing out, push down with both hands and lift pelvis off seat. While breathing in, let yourself down slowly.
8. **Step ups.** Breathe out, step up with one foot. Breathe in, bring up other foot. Step down with one foot, then the other.
9. **Walking sideways.**
10. **Wall press-ups.** Stand with feet a comfortable distance from the wall, put hands on wall, bend at elbow (keeping heels on floor), push arms straight again.
11. **Abdominal contractions.** In sitting, pull in abdominal muscles, relax and breathe.
12. **Static bike, hula-hoops, trampet.**
13. **Bounce ball off wall.**
14. **Calf exercise.** Holding back of chair, go up on toes, return heels to floor.
15. **High knee marching.** Holding back of chair with one hand, march on the spot, lifting knees high.
16. **Arm raise.** Sitting or standing, raise arm, with or without weight, above head. Lower slowly. Repeat with other arm.
17. **Bend down, stretch up.** In time with breathing.
18. **Sit to stand.** Using dining room chair, sit–stand–sit. Repeat, holding a ball.

strain than walking (Bach and Haas, 1996, p. 309). Treadmill-walking enables participants to learn the feel of different speeds, so that they can structure their home programme. Other participants enjoy simple activities that can be continued at home, such as chair exercises. Low-intensity individual limb exercises are well-tolerated and translate into improved whole-body exercise capacity (Clark *et al.*, 1996). Upper limb exercise needs to be included (ACCP/AACVPR, 1997), and hobbies such as bowls are both enjoyable and useful for chest mobility.

Circuit training can involve six to ten exercise stations, choosing from examples in Box 9.6. Exercises are best alternated between upper/lower limb exercises, and easy/difficult exercises.

Gentle progressive arm exercises reduce the breathlessness associated with upper limb activities, with a carry-over effect on the respiratory muscles that can be equivalent to inspiratory muscle training (Hodgkin *et al.*, 2000, p. 158). Unsupported arm exercise should be included unless this causes abdominal paradox (p. 37). Loss of shoulder girdle support forces the intercostal and accessory muscles to stabilize the arms and torso, which shifts the breathing load to the diaphragm, creating a challenge for people with COPD (Figure 9.14).

Figure 9.14 The effort of unsupported upper limb activity (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

The *intensity* of exercise can vary from the use of precise physiological monitoring to a laissez-faire approach. Some methods are described below.

1. The predicted maximum HR can either be estimated as 220 minus age or measured during an incremental stress test. Exercise is traditionally maintained at 70% of maximum HR, but respiratory patients can achieve a training effect at 30–40% of maximum (Hellman, 1994). HR is linearly related to $\dot{V}O_{2max}$, so the pulse can be taken at, say, 50% of $\dot{V}O_{2max}$ and used as a target. Multiples of $\dot{V}O_{2max}$ are expressed as METs (see Glossary). These complicated methods of monitoring exercise intensity are widely described but may not be suitable because:

- many respiratory patients are too breathless to reach true maximal HR or $\dot{V}O_{2max}$ (Mejia, 1999)
- HR is affected by cardiorespiratory drugs such as beta-blockers, digoxin and salbutamol
- even people with normal lungs show a wide variation in HR (Belman *et al.*, 1991) and $\dot{V}O_{2\,max}$ (Bach and Haas, 1996).

2. Breathlessness scales can be kept on clipboards for participants to assess their perception of breathlessness (Box 9.7). Exercise can be increased gradually while maintaining breathlessness at a constant tolerable level. Breathlessness scales are reproducible, correlate with physiological measures of exercise intensity and even in people with normal lungs have been shown to result in greater improvement in endurance than when using HR (Koltyn and Morgan, 1992). It is thought safer for the participant to choose the level of breathlessness rather than the physiotherapist (Myles and Maclean, 1986).

3. Participants achieve a moderate training response if they are able simply to exercise enough to achieve an increase in breathlessness at a constant tolerable level while avoiding distress or desaturation. This allows for the variation in intensity that suits different individuals. Some exercise gently while others can

Box 9.7 Breathlessness scales

a) Borg shortness of breath scale, which assigns numerical values to degrees of breathlessness. The level chosen by the participant is maintained while the amount of exercise gradually increases.

0 – Nothing at all
0.5 – Very very slight
1 – Very slight
2 – Slight
3 – Moderate
4 – Somewhat severe
5 – Severe
6
7 – Very severe
8
9 – Very very severe
10 – Maximal

b) Breathlessness rating scale. The participant chooses to work at rating 2,3 or 4.

Rating 1	Comfortable breathing throughout
Rating 2	*During*: deeper breathing *After*: recovery 2–5 min *Day after*: comfortable
Rating 3	*During*: harder breathing *After*: recovery 4–7 min *Day after*: comfortable
Rating 4	*During*: breathless but not speechless *After*: recovery 5–10 min *Day after*: not tired
Rating 5	*During*: breathless and speechless *After*: recovery > 10 min *Day after*: tired

exercise safely at over 80% of their maximum (Ries, 1994).

The balance of duration and frequency depends on individual preference because the result is similar if total work is the same. Low-intensity, unstructured programmes appear to be as beneficial as high-intensity, highly structured programmes (Bach and Haas, 1996). Supervised training sessions usually last for 30–60 minutes but, for home practice, respiratory patients find it more acceptable to exercise for one or more short sessions a day with brief warm up and cool down periods. Severely breathless people may prefer interval training, which alternates 15- to 30-second episodes of exercise with rest.

People who tend to rush at their exercise in an attempt to get it over quickly may find that counting breaths with their steps helps them to pace themselves in the early stages, e.g.: in/one, out/one, or in/one, out/one/two. For others this disturbs their rhythm and distracts them from focusing on awareness of their breathing and level of effort. Stair-climbing may be more efficient if performed by inhaling as one leg is raised, exhaling as the body is raised and interspersing every few steps with a rest.

Cool down

Participants are asked to slow down their activity for a few minutes, e.g. by slow walking, to prevent sudden pooling of blood in the lower extremities.

Progression

Participants progress by increasing duration or frequency, usually in weekly increments. Intensity usually stays the same but some participants are happy to increase this. Improvement usually continues for 4–6 months and, when a plateau is reached, moderate exercise should be maintained at a minimum 15 minutes a day. An indoor programme is substituted when there is air pollution, wind or rain. Urban patients are advised to choose the least polluted times and places for training. A fixed routine at a regular time of day helps adherence to the programme. Progression includes adaptation to uneven terrain and any anticipated problems identified by the patient. Figures 9.15 and 9.16 are suggested documentation to record results and monitor progress.

Once a week, participants should put themselves back on the same programme as on the final day of their training. If this is difficult, they

Stage 1: Level 1–2 on shuttle or **recovering from exacerbation** **Name:**				
Date **Activity**				
Warm up				
Walk in parallel bars				
Biceps curls				
Bike without resistance				
Quadriceps				
Sit-to-stand				
Cool down				
Rest				
S_aO_2				
Borg				
Stage 2: Level 3–6 on shuttle or **able to bike for 15 min** **Name:**				
Date **Activity**				
Warm up				
Treadmill 2 min				
Quadriceps + 1 kg				
Wall press-ups				
Bike resistance 25				
Small step				
Arms 50 reps				
Cool down				
Rest				
S_aO_2				
Borg				
Stage 3: Level 7 on shuttle or **bike 15 min + arm exercises 50 reps** **Name:**				
Date **Activity**				
Warm up				
Bike resistance 50				
Balls, sandbags, etc				
Multigym				
Stairs				
Cool down				
Rest				
S_aO_2				
Borg				

Figure 9.15 Circuit record sheet.

Name

Date	Resting	Distance or no. shuttles	Completion of exercise	2 min after exercise
	S_aO_2 Borg		S_aO_2 Borg	S_aO_2 Borg
	S_aO_2 Borg		S_aO_2 Borg	S_aO_2 Borg
	S_aO_2 Borg		S_aO_2 Borg	S_aO_2 Borg
	S_aO_2 Borg		S_aO_2 Borg	S_aO_2 Borg

Figure 9.16 Progress sheet, used as baseline and at 3 months, 6 months and 12 months

have lost fitness and will need to increase their maintenance exercise. If training is interrupted by illness or holiday, the programme is restarted at a lower level. The difficult task of maintaining fitness needs follow-up encouragement from the rehabilitation team.

> ***The condition of exercise is not a mere variant of the condition of rest, it is the essence of the machine.***
>
> Sir John Bancroft, quoted by Clark 1996

INSPIRATORY MUSCLE TRAINING

The concept that strengthening the respiratory muscles would allow a patient to sustain a higher level of ventilation during exercise is an attractive one if this increased exercise tolerance, but does it work?

Rationale

Question 1

Respiratory disease can make inspiratory muscles either weaker or stronger than normal (Heijdra, 1994). How?

Strong muscles develop by working against the resistance of obstructed airways or stiff lungs (Newell, 1989). Weak muscles are due to:

- poor nutrition, in which case dietary management is indicated, which can improve inspiratory muscle strength by 40% (Donahoe and Rogers, 1990)
- inadequate oxygen delivery to the muscles because of heart failure or blood gas abnormalities, which may respond to fluid, drug and oxygen therapy
- steroid-induced weakness, which should respond to drug review
- mechanical disadvantage, which might respond to breathing re-education (Martinez *et al.*, 1991) or exercise training (O'Donnell, 1995).

Therefore, for strong inspiratory muscles, it is presumed inappropriate to add a further load. For weak muscles, the cause should be addressed.

Question 2

Training can make the diaphragm either more or less susceptible to fatigue (Braun *et al.*, 1983). How?

A diaphragm that becomes more susceptible to fatigue after training is thought to have reached maximum adaptability and can improve performance no further. It is already chronically fatigued and is more likely to benefit from nutrition than training. Jederlinic *et al.* (1984) claim that inspiratory muscle training (IMT) can override the protective mechanism of fatigue and lead to exhaustion and desaturation. Fatigue responds to rest.

A diaphragm that becomes less susceptible to fatigue after training is in a fit state to adapt to

the training stimulus. This is when IMT may be appropriate, especially as people with COPD show parallel decrements in strength of limb and respiratory muscles, each contributing independently to reduced exercise capacity (Simonds *et al.*, 1996, p. 27).

Effects

Increased strength: ability to generate greater force
Increased endurance: ability to generate the same work for a longer time.

When used appropriately, IMT can improve inspiratory muscle strength and endurance (O'Kroy, 1993) but there is limited evidence that this benefits the patient (ACCP/AACVPR, 1997). Results have been mixed but, in rested and nourished patients, the following outcomes have been reported:

- ↓ breathlessness and ↑ exercise tolerance (Lisboa *et al.*, 1997)
- ↑ nocturnal saturation (Heijdra, 1996)
- ↑ motivation to exercise (McConnell *et al.*, 1998).

One study found there was no effect unless nutrition was providing 1.5 times the basal metabolic needs (Rochester, 1992).

Some patients have shown reduced breathlessness by training the expiratory muscles (Suzuki and Sato, 1995). Expiratory muscle training may benefit people with multiple sclerosis (Smeltzer, 1996) whose poor cough pressures are caused by weak expiratory muscles, partly due to deconditioning because of inactivity.

Indications and contraindications

How can we select those patients with weak muscles for whom training might provide protection against chronic fatigue, and avoid overburdening those with fatigued muscles? The clinical symptoms of fatigue and weakness are similar but the two states are distinguishable (p. 7–8). Fatigued muscles are unsuited to training, and excessive exercise may split fibres, create 'overuse atrophy' (Braun *et al.*, 1983) and cause muscle damage (Anzueto, 1992). Patients with weak muscles may benefit from training, regardless of how breathless they are, and are most likely to respond if:

- they are fearful of activity, because IMT can be used to desensitize them to breathlessness prior to venturing into exercise training
- they are unable to do exercise training for other reasons, in which case IMT can be a substitute
- they find breathing re-education difficult, in which case using the device might familiarize them to an altered breathing pattern, before progressing to self-regulation of breathing
- they enjoy it!

Most studies have investigated people with COPD, but benefits have been reported for people with CF (Sawyer and Clanton, 1993), asthma (McConnell *et al.*, 1998), restrictive disease (Chatham, 2000) and those awaiting heart transplantation (Cahalin, 1997). Others include patients with steroid-induced respiratory muscle weakness (Weiner, 1995) and those in respiratory failure who fail to wean from mechanical ventilation because of respiratory muscle atrophy (Aldrich, 1985).

For people with neurological disease, damaged nerves must be respected and weak muscles not overworked. However, exercise-induced injury has not been reported, possibly because patients would not tolerate fatiguing loads. Disorders that leave the intact muscles unaffected, such as quadriplegia, have shown improvement with both inspiratory and expiratory muscle training (p. 396). Progressive disorders such as muscular dystrophy have shown some benefit before the stage of advanced disease when CO_2 is retained (McCool and Tzelepis, 1995).

Some benefit has been shown for those without disease, e.g. the elderly (Copestake and McConnell, 1994) and sportsmen who want to maintain their fitness when injury prevents training (Chatham, 2000).

Technique

Devices are cheap and simple. The principles of training are followed:

- alternate exercise with rest
- avoid distressing levels of fatigue
- progress by time and/or resistance.

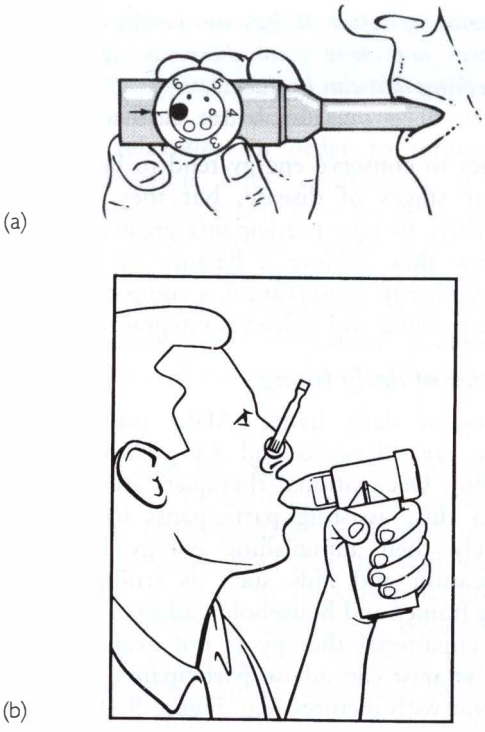
For strength training, the target is generally 80% of MIP (p. 61) and for endurance training it is 60%, but benefits have been found at 30% of maximum (Nield, 1999). If the aim is desensitization to breathlessness, resistance should be at a level that leaves the patient more breathless than normal but not speechless or distressed. More simply, a resistance can be set that the patient can tolerate for 10 minutes (Brannon *et al.*, 1998, p. 431). Patients should be relaxed but inhale with sufficient force to overcome the resistance. They should work at different ranges to prevent muscle fatigue, while avoiding excess hyperinflation. If oxygen is needed, nasal cannulae can be used.

If progressing by time, this increases from about 5 minutes twice a day to about 15 minutes three times a day. If the patient prefers, the timing remains stable, e.g. five 2-minute periods three times a day, with resistance increased fortnightly for the first 6 weeks and then monthly.

When patients have mastered the art, training can be combined with watching TV or reading. Adherence is reasonable when IMT fits into the patient's schedule and the resistance is not uncomfortably high. Training diaries and further details of technique are provided by manufacturers (Appendix C).

A **flow-dependent device** (Figure 9.17a) sets resistance by the size of various inspiratory orifices, but this load can be lessened by the patient taking slow breaths to reduce turbulence.

These devices are less likely to produce a training effect and are best used for desensitization to breathlessness. PEP masks can be used as flow-dependent inspiratory muscle trainers by attaching the resistance to the inspiratory port.



(a)

(b)

Figure 9.17 (a) Flow-dependent inspiratory muscle trainer. (b) Pressure-threshold inspiratory muscle trainer.

A **pressure-threshold device** (Figure 9.17b) incorporates a spring-loaded one-way valve, which opens to permit airflow only when a preset inspiratory pressure has been reached (Gosselink, 1996). The load is independent of airflow and can be set at a percentage of MIP. This obliges the patient to generate a set inspiratory force with every breath and is able to create a training effect.

Incentive spirometry has been shown to provide sufficient resistance to create a training response in some elderly people (Marinho *et al.*, 1999).

ENERGY CONSERVATION

One of my ambitions is to dissuade health professionals from saying 'there's nothing more that can be done'. Apart from the

> ***devastating effect it has on people, it is simply not true ... there is always something that can be done.***
>
> Clay, cited by Ahmedzai, 1997

Strategies to conserve energy tend to be used in the later stages of disease, but they are best taught early to give participants greater control over how they achieve a balance of rest and exercise. Energy conservation is compatible with exercise training and indeed is integral to it.

Activities of daily living

Activities of daily living (ADL) training can improve breathlessness and S_aO_2 (Aronsson *et al.*, 1996). Occupational therapists are valuable allies in this, assisting participants to allocate selectively their diminishing energy by work simplification and aids such as trolleys, high walking frames and household gadgets.

If occupational therapy is not available, the physiotherapist can advise participants, or make a handout with pictures (e.g. Figure 9.18), based on the following:

- Prioritize activities, eliminate non-essentials.
- Plan in advance, allow time, alternate hard and easy chores, spread energy-demanding tasks over the week, pace activities and work in stages.

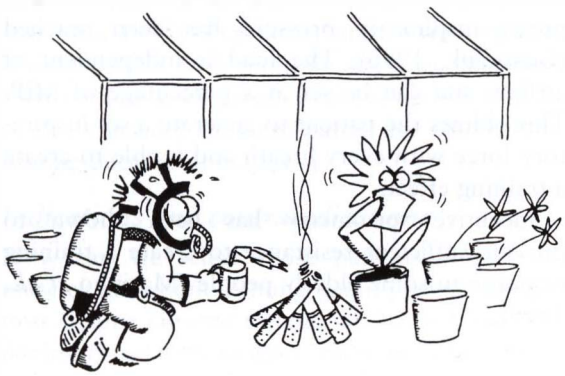


Figure 9.18 Activities of daily living. (From Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford, with permission. Artist: James Northfield.)

- Organize chores by location to avoid multiple trips.
- Co-ordinate breathing, e.g. inhale with pulling and exhale with pushing, bending or the strenuous part of an activity ('blow as you go').
- Move smoothly, avoid extraneous movements, use a rollator rather than a Zimmer frame, which destabilizes the shoulder girdle and requires twice the oxygen consumption (Foley *et al.*, 1996).
- Lean on shopping trolleys.
- Organize work space to reduce clutter and minimize reaching and bending.
- Ensure that work surfaces are the correct height.
- Keep heavy items on top of the work surface.
- Rest elbows on worktop for arm activities.
- Develop economic lifting methods using leg power rather than back and shoulders.
- Slide pots and pans along the worktop rather than lifting them.
- Prepare large one-dish meals such as casseroles, serve in baking dish, freeze leftovers.
- Soak washing up.
- Use a stool for kitchen work and ironing, as this can save 24% of energy (Bach and Haas, 1996, p. 336).
- Sit to dress, put on two items at once e.g. underwear with trousers or skirt.
- Reduce bending by crossing one leg over the other to put on socks, trousers and shoes.
- Avoid aerosols or strongly scented perfumes.
- For bed-making, have a raised bed on casters away from walls, unfold sheets on the bed, make only one trip round the bed.
- Use non-iron clothes, electric toothbrush, long-handled sponge, soap-on-a-rope, towelling bathrobes, slip-on shoes or Velcro closures.
- Plan ahead for socializing because the energy expenditure can equal that of walking (Jette *et al.*, 1997).

Participants can share their own strategies

Figure 9.19 Energy conservation in the shower (From Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: A Guide to Better Living and Breathing*, C. V. Mosby, St Louis, MO)

such as finding inconspicuous 'puffing stations' during shopping trips, e.g. window shopping.

Advice needs to be individualized. Some people might find it more important to use their energy to get to the shops than to be independent with dressing. Some prefer to sleep downstairs rather than suffer the 'stigma' of a stairlift. Some find sitting in a shower easier than using a bath (Figure 9.19), while others find that water on their face upsets their breathing. Some are not happy to have their spouse bathe them.

Stress reduction

> ***Voluntary control of respiration is perhaps the oldest stress-reduction technique known. It has been used for thousands of years to reduce anxiety and promote a generalized state of relaxation.***
>
> Everly, 1989

Stress is physiologically detrimental (p. 25). Putting a tense person through a physical training programme without advice on stress management is silly. People with chronic lung disease suffer muscle tension from breathlessness, stress and the body positions needed to ease their breathing. A rhythmically active muscle such as the diaphragm is in particular need of relaxation in order to return to its resting position after contraction (Coirault, 1999), especially when it is being overused to maintain hyperinflation. Some have become accustomed to muscle tension and forget how it feels to be relaxed. Relaxation helps breathing and breathing helps relaxation. It should be taught early and reinforced throughout.

Relaxation

Relaxation can be taught by several methods, e.g. page 170, or self-taught from books, tapes or classes. Daily practice is needed until the sensation is appreciated and the skill mastered, whereupon a degree of relaxation is integrated into everyday life by identifying stressful situations and practising in different positions. Relaxed walking can be consciously maintained. Spot checks during the day can identify body tension.

Relaxation can be achieved in other ways. Participants often have their own ideas, e.g. sewing, jigsaws or, for insomniacs, watching a lighted aquarium at night. Activities such as circle dancing or Tai Chi provide rhythmic exercise with a meditative effect, which emphasizes trunk rotation (Wolf *et al.*, 1997), and improve balance, posture, immune function and conditioning (Lan, 1996).

Complementary therapies

> ***The best way to still the mind is to move the body.***
>
> Roth, 1990

Complementary therapies may help ease breathlessness and stress, depending on the practitioner. It is useful to have some knowledge of complementary therapies and local resources for participants who request this information.

Yoga incorporates breathing techniques, meditation and postures that consume minimal energy and induce physiological effects charac-

teristic of deep relaxation. One study demonstrated improved lung function (Singh, 1990) and another a reduced ventilatory response to CO_2 (Stanescu, 1981). Yogic breathing promotes breathing awareness, nose-breathing and 'complete breathing', which begins with abdominal breathing, then expands the lower chest and finally the upper chest (Fried, 1993, p. 239). Meditation clears interfering thoughts from the mind, reducing respiratory rate, HR and BP (Fried, 1993, p. 235).

The *Alexander technique* inhibits muscle tension, which reduces WOB and can improve objective measures such as peak flow and respiratory muscle strength (Austin and Ausubel, 1992). The *Feldenkrais method* uses the principle of least effort and is useful for energy conservation (Hannon, 2000). *Massage* can reduce BP (Hernandez-Reif, 2000). *Biofeedback* to reduce muscle tension allows the sensation to be recognized and control gained over it (Hodgkin *et al.*, 2000, p. 12). *Imagery* uses visualization of peaceful scenes, which can achieve a relaxed alpha brain-wave state (Haas and Axen, 1991, p. 285). *Hypnotherapy* reduces the metabolic rate through deep relaxation (Sato *et al.*, 1986). *Acupuncture* works directly on reducing the perception of breathlessness and has shown increased exercise tolerance (Jobst *et al.*, 1986). Patients contemplating *aromatherapy* need to be aware that different oils can affect breathing positively or negatively.

Mechanical rest

For chronically fatigued patients, non-invasive ventilation at home may be part of rehabilitation.

FOLLOW-UP, HOME MANAGEMENT AND SELF-HELP

> ***The increased exercise capacity, improved skills and sense of empowerment achieved in rehabilitation do little good if the patient's sole exercise thereafter is pushing remote control buttons to change TV channels.***
>
> Thomas, 1996b

Follow-up plans should be set at the start of the programme. It takes a minimum of 6 weeks, and often longer, for participants to see an improvement. If expectations are not met, they may lose heart. Some supervised training is best continued for a period after the initial programme, to prevent detraining and demotivation (Swerts, 1990). Thereafter, follow-up in patients' homes or by telephone (Pal, 1998), newsletter or further training sessions (Ojanen, 1993) may be needed, 3- and then 6-monthly.

The hospital may be able to provide a regular venue, or a leisure centre may be appropriate. Transport to follow-up meetings and social get-togethers may be available through voluntary organizations.

Home visits are especially useful for people who are elderly, anxious, forgetful, using new equipment or at the end-stage of disease. The home environment is where people feel most in control and are most responsive to advice. Patients are now cared for at home when they have relatively acute disease or when using intravenous therapy, tracheostomies or home ventilators.

Home visits provide the opportunity to check for adequate heating, and health or safety hazards. They are also supportive for the family. Spouses may be stressed, neglect their own health, feel guilty or be fearful of sleeping lest their partner die in the night. Children may lack attention and be caught up in conflicting emotions. Between visits, patients and families need a contact telephone number.

Respiratory patients can withdraw into social isolation because of the nature of their symptoms, and the mutual support that develops between participants during the rehabilitation programme may become one of its most enduring assets. This support can be built into self-help groups using the Internet, or Breathe Easy, co-ordinated by the British Lung Foundation (Appendix C), or peer outreach programmes in which patients are visited by volunteers with lung disease who have been selected for their ability to cope with their disabilities. Social outings, monthly lunches and annual celebra-

tions may develop, which are particularly supportive for people who do not like to be seen in public with their oxygen.

> ***If I go and visit a Breathe Easy supporter who hasn't been to a group meeting for a while, I know what I'll find. Someone sitting in an armchair, the TV in front, a nebulizer on one side and the remote control and telephone on the other. We all know it – it's the siege situation.***
>
> *Breathe Easy Newsletter*, 1997

OUTCOMES

Evaluation of rehabilitation can be by the following:

- number of participants completing the programme
- scales on breathlessness and QoL
- diary review
- hobbies, job (if wanted)
- GP visits, admissions to hospital, time in hospital
- anxiety and depression scores
- smoking
- medication e.g. ↓ beta$_2$-agonists
- independence in ADL
- video evidence of improved flexibility, posture and gait
- weight ↑ or ↓ as appropriate
- specifically in relation to exercise training: ↑ walking distance, ↑ shuttle test, ↑ $\dot{V}O_{2\ max}$, ↓ exercise HR, ↓ blood lactate levels (Mohsenifar *et al.*, 1983)
- improved well-being of carers.

Changes in lung function are not anticipated. Boxes 9.1 or 9.2 can be used to record the relevant outcome measures.

It is hoped that an abiding legacy of the programme is the friendship and courage that participants give each other. For those labouring under the double burden of disease and ageing, the outcome should be a more optimistic attitude towards a life that can be active and fulfilling.

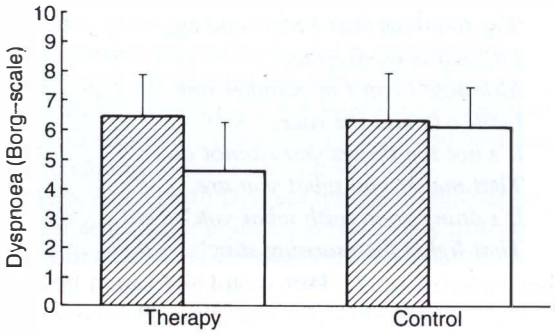


Figure 9.20 Pre- and post-rehabilitation scores for dyspnoea, showing the effectiveness of breathing re-education, relaxation and exercise training. (From Strijbos, J. H. *et al.* (1989) Objective and subjective performance indicators in COPD. *European Respiratory Journal*, **2**, 666)

Western medicine has yet to make full adjustment to the increasing prevalence of chronic over acute disease, and tends to focus on illness rather than prevention and rehabilitation. Physiotherapists can play a part in educating others on the effectiveness of pulmonary rehabilitation by lecturing to medical and other students, initiating meetings with physicians and providing proof of benefit to patients (Figure 9.20) and cost-effectiveness to budget-holders (Figures 9.1, 9.20, 9.21, Appendix E).

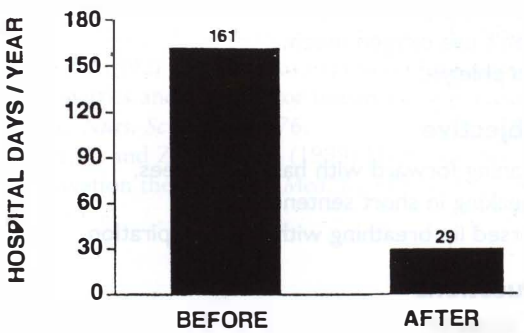


Figure 9.21 Pre- and postrehabilitation hospital days. (From Make (1994) Collaborative self-management strategies for patients with respiratory disease. *Respiratory Care*, **39**, 566–577)

'I'm thankful that I have one leg,
To limp is no disgrace.
Although I can't be number one
I still can run the race.
It's not the things you cannot do
That makes you what you are,
It's doing good with what you've got
That lights the morning star.'

Hart, quoted by deLateur, 1997

MINI CASE STUDY: MR EH

Identify the problems of this 66-year-old man with emphysema who was referred as an outpatient after disappointment following rejection for transplantation.

Background

SH: lives with wife, first floor flat with lift.
Drugs: bronchodilators, steroids, diuretics.
HPC: Recent admission for exacerbation, discharged with home oxygen, making slow recovery.

Subjective

Can't do much since leaving hospital.
Able to look after self.
Able to drive.
Unable to walk any useful distance.
Unable to help in house or with shopping.
Poor sleep since hospital.
Not hungry.
Don't use oxygen much.
No phlegm.

Objective

Leaning forward with hands on knees.
Speaking in short sentences.
Pursed lip breathing with active expiration.

Questions

1. Analysis?
2. Problems?
3. Goals?
4. Plan?

RESPONSE TO MINI CASE STUDY

1. Analysis

Loss of confidence and exercise tolerance since discharge.

2. Problems

Breathlessness.
Inefficient breathing pattern.
↓ exercise tolerance.
Depression.
Misuse of oxygen.

3. Goals

Short term: daily walk to bandstand.
Medium term: return to pre-admission function including steps up to flat.
Long-term: lifelong programme.

4. Plan

- Obtain further information from patient on fluids, nutrition, limitations to exercise tolerance (e.g. anxiety, SOB, deconditioning).
- Identify cause of poor sleep, liaise with multidisciplinary team where appropriate.
- Educate, or refer to respiratory nurse for education, on oxygen therapy. Refer to dietician for nutritional advice. Check knowledge of medication.
- Following preliminaries (e.g. education for anxiety and desensitization for breathlessness) retrain efficient breathing pattern and initiate written daily exercise programme.
- Follow up in a week to maintain motivation, then monthly, then 6-monthly reviews.

LITERATURE APPRAISAL

Comment on the logic of the following statements.

Pursed lip breathing should be employed during and following exercise and during any activity.
[T]he authors concluded that the inability

of pursed lip breathing to decrease oxygen consumption meant that this breathing method failed to decrease the work of breathing.

... using a tilt board such as an ironing board....

Clin. Chest Med. 1986; 7: 599–618

RESPONSE TO LITERATURE APPRAISAL

Pursed lip breathing is recommended even though it is unable to decrease oxygen consumption or decrease WOB.

Discrepancy between findings and conclusion is not unusual, possibly because authors find it difficult to accept what they had not anticipated. It might have been more useful if the discussion had looked at possible reasons why some patients find pursed lip breathing helpful even if it does not reduce their WOB.

And an ironing board is for ironing....

RECOMMENDED READING

(See also appendix E)

AARC Clinical Practice Guidelines (1999) Suctioning of the patient in the home. *Respir. Care*, 44, 99–104.

ACCP/AACVPR (1997) Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. *Chest*, **112**, 1363–1396.

Ambrosino, N. and Clini, E. (1996) Evaluation in pulmonary rehabilitation. *Respir. Med.*, **90**, 395–400.

Ambrosino, N. and Foglio, K. (1996) Selection criteria for pulmonary rehabilitation. *Respir. Med.*, **90**, 317–322.

Carrieri, V., Douglas, M. K. and Gormley, J. M. (1993) Desensitisation and guided mastery: treatment approaches for the management of dyspnea. *Heart Lung*, **22**, 226–234.

Celli, B. R. (1994) Physical reconditioning of patients with respiratory diseases: legs, arms and breathing retraining. *Respir. Care*, **39**, 481–499.

DeVito, A. J. (1990) Dyspnea during hospitalization for acute phase of illness as recalled by patients with COPD. *Heart Lung*, **19**, 186–191.

Gaskin, L. and Thomas, J. (1995) Pulse oximetry and exercise. *Physiotherapy*, **81**, 254–261.

Janssens, J. P. (2000) Management of dyspnea in severe COPD. *J. Pain Symptom Man.*, **19**, 378–392.

Kakizaki, F., Shibuya, M. and Yamazaki, T. (1999) Preliminary report on the effects of respiratory muscle stretch gymnastics on chest wall mobility in patients with COPD. *Respir. Care*, 44, 409–414.

Lan, C., Lai, J. S. and Chen, S. (2000) Tai Chi Chuan to improve muscular strength and endurance in elderly individuals. *Arch. Phys. Med. Rehab.*, **81**, 604–607.

Lareau, S. C. (1996) Functional status instruments: Outcome measures in patients with COPD. *Heart Lung*, **25**, 211–224.

Molen, B. van der (1995) Dyspnoea: a study of measurement instruments for the assessment of dyspnoea. *J. Adv. Nurs.*, **22**, 948–956.

Nicholls, D. (2000) Breathlessness. A qualitative model of meaning. *Physiotherapy*, **86**, 23–27.

O'Donnell, D. E. (1994) Breathlessness in patients with chronic airflow limitation. *Chest*, **106**, 904–912.

Peel, C. and Mossberg, K. A. (1995) Effects of cardiovascular medications on exercise responses. *Phys. Ther.*, **75**, 387–396.

Steele, B. (1992) The dyspnea experience: nociceptive properties and a model for research and practice. *Adv. Nurs. Sci.*, **15**, 64–76.

Vickers, A. and Zollman, C. (1999) Hypnosis and relaxation therapies. *Br. Med. J.*, **319**, 1346–1349.

10 Physiotherapy for people undergoing surgery

Summary

Introduction

Surgery is now available for those previously denied it because of disease or debility. Many operations can now be performed via laparoscopy (keyhole incision through the abdominal wall) or thoracoscopy (keyhole incision through the chest wall), including aortic aneurysm repair (Cerveira *et al.*, 1999) and heart surgery (Burke, 1998).

Minimal access techniques use fibreoptic endoscopes, which provide illuminated fields of vision and allow keyhole surgery by instruments inserted through several small stab incisions. Video-assisted techniques enable the operative field to be viewed by the team on a monitor displaying a magnified view from within the patient's body. Major surgery is also possible at the bedside or in the intensive care unit (ICU), often under local anaesthesia (Dennison *et al.*, 1996). Virtual surgery may become a reality (Cregan, 1999).

These changes have altered requirements for physiotherapy. Sicker patients who are now able to have surgery require extra input, and policies of early discharge demand rapid rehabilitation. However, complications related to pain and bedrest have decreased.

Physiotherapists working on a surgical ward need acumen in order to identify patients who need treatment, and empathy for the individual because what is routine for physiotherapists is a unique event for patients. Patients anticipate surgery with their own mixture of hope and dread.

Respiratory complications are the leading cause of postoperative morbidity and death (Brooks-Brunn, 1995). Most patients gain from physiotherapy input to the team management of pain, positioning and handling. All could doubtless benefit from direct physiotherapy, but most physiotherapists select for assessment only those at risk due to (Bluman *et al.*, 1998):

- smoking history, especially current smokers who have a six-fold greater incidence of postoperative complications
- surgery to the upper abdomen or chest
- prolonged preoperative stay
- prolonged anaesthesia
- the presence of lung or heart disease
- obesity or malnourishment

- advanced age
- excessive anxiety
- emergency surgery

RESPIRATORY COMPLICATIONS OF SURGERY

Lung complications occur in 25–50% of patients undergoing major surgery (Ferguson, 1999). After abdominal or chest surgery these reach a maximum within 48 hours. The following have been demonstrated after full-incision surgery.

Atelectasis

Atelectasis (collapsed alveoli) occurs in typically 10–15% of the lung and lasts an average 2 days postoperatively (Hedenstierna, 1999). The left lower lobe is the commonest site, possibly because of compression from the heart (Raoof *et al.*, 1999). Causes are described below.

- Pain is the major culprit (Simpson *et al.*, 1992), dull at rest and sharp on movement. This leads to immobility and, after chest or abdominal surgery, guarding spasm of the trunk muscles and inhibition of breathing (Figure 10.1) so that tidal breathing falls into the closing volume range (Sutcliffe, 1993).
- Prolonged recumbency affects the amount of ventilation causing intrathoracic pooling of blood which further displaces air from the lung.
- Drowsiness and immobility obliterate the regular oscillations in tidal volume that normally punctuate breathing and stimulate surfactant production.
- Absorption atelectasis (p. 120) is due to supplemental oxygen during anaesthesia. This begins after about 40 min of 40% O_2 and 5 min of 100% O_2 (Clarke *et al.*, 1998). It is augmented by the closed gas pockets created by reduced lung volume (Hedenstierna, 1999).
- Muscle tone is reduced.
- Diaphragmatic function may be impaired by abdominal distension (Frost, 1996).
- Sympathetic pleural effusion is a common, though usually minor, reaction to fluid overload.

Atelectasis creates a restrictive lung defect, reducing lung compliance, increasing airway resistance (Wahba, 1991) and depleting surfactant (Brooks-Brunn, 1995). Persistent atelectasis may be associated with chest infection but there is little evidence of causality (Brooks-Brunn, 1995).

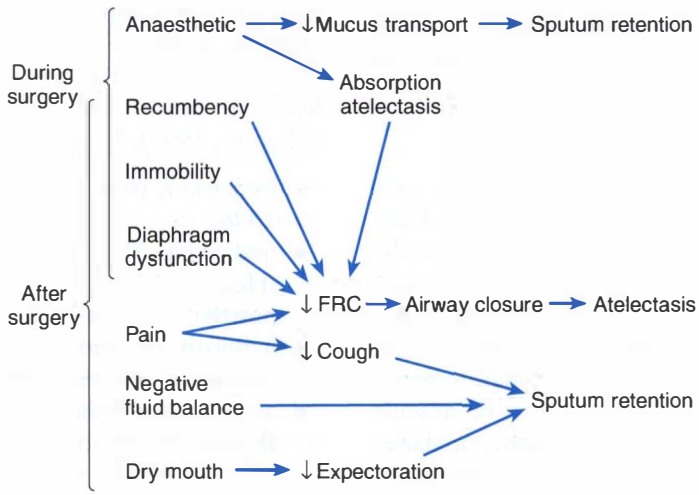


Figure 10.1 Interrelation of factors affecting postoperative lung function.

Atelectasis can be reduced by measures to increase lung volume (Chapter 6). Greater efforts are needed to inflate collapsed alveoli than to inflate those that are partially open. Prevention is therefore better than cure.

Hypoxaemia

There is a close correlation between atelectasis and shunt. Hypoxaemia is caused by shunting of blood through airless lung and inhibition of hypoxic vasoconstriction (p. 11) by volatile anaesthetic agents. Hypoxaemia may not be significant and many patients leave hospital happily ignorant of its existence. For others it can impair healing, promote infection and contribute to postoperative confusion (Hanning, 1992). When present for a few hours it is related to the anaesthetic. When present for several days it is related to the surgery and postoperative factors (Hudes, 1989).

Patients at risk may suffer nocturnal hypoxaemia for up to five nights after surgery (Roberts *et al.*, 1993). They have missed out on their rapid-eye-movement sleep due to disruption and medication, and as they catch up on this part of their sleep cycle, their oxygen requirements increase (p. 25). Patients who have had major surgery, or those with respiratory or cardiovascular disease, should be monitored for nocturnal oxygen desaturation to prevent premature cessation of oxygen therapy. During surgery, the provision of 80% oxygen has been shown to halve the incidence of wound infection (Grief, 2000).

Chest infection

Intubation overrides defence mechanisms and anaesthetic agents impair ciliary action. After halothane–nitrous oxide anaesthesia, mucociliary transport slows after 30 minutes and stops after 90 minutes (Houtmeyers, 1999). This predisposes to chest infection, especially in smokers (Konrad, 1993). Signs may emerge some days after surgery, e.g. crackles on auscultation, purulent bronchial secretions, malaise, fever and sometimes tachypnoea. If pneumonia develops, mortality can reach 30–40% (Brooks-Brunn, 1995).

Other chest problems

Intubation can cause bronchospasm in susceptible patients (Wong and Shier, 1997). Toxic levels of opioids can cause respiratory depression. Vital capacity can drop by 40–50% (Wahba, 1991) because of pain, leading to impaired cough.

OTHER COMPLICATIONS OF SURGERY

Fatigue is related to the degree of trauma during surgery and lack of nutrition after surgery (Christensen and Kehlet, 1984). It is more severe and prolonged than expected by most patients, persisting for a month in two-thirds of people and directly affecting exercise capacity (Delaunay, 1995). Frequent short walks should be negotiated rather than infrequent long ones.

Some drugs contribute to the 'big little problem' of postoperative *nausea.* This is experienced by 20–30% of patients (Diflorio, 1999), some of whom find it a more wretched experience than pain (Watcha, 1996). It is commonest after lengthy surgery or in patients who are hypovolaemic, in pain, anxious, obese or female (Broomhead, 1995). Nausea inhibits deep breathing, and vomiting can lead to complications such as fatigue, bleeding, dehiscence (separation of the incision or rupture of the wound), aspiration of gastric contents, delayed hospital discharge and increased readmissions (Watcha, 1996). Rapid-onset analgesics such as fentanyl cause less nausea than morphine (Claxton, 1997). Nausea may be relieved by:

- hydration, pain relief, drug review (Watcha, 1996)
- midazolam (Diflorio, 1999), dexamethasone (Henzi, 2000), cannabis (Russo, 1998) powdered ginger (Phillips, 1993)
- hypnosis (Faymonville, 1997), preoperative relaxation (Enqvist, 1997)
- acupressure (Fan *et al.*, 1997) or TENS (Frazer, 1999) applied to P6 located two thumbs' widths proximal to the distal wrist crease on the inner arm in line with the middle finger (Ellis, 1994, p. 157)

- supplemental oxygen, which can halve the incidence of nausea and vomiting (Greif *et al.*, 1999).

Anxiety increases diaphragmatic splinting and stimulates metabolic and hormonal stress responses which can delay healing and promote infection (Salmon, 1992). Anxiety is reduced by giving preoperative information and granting postoperative autonomy.

Depression may occur, especially if surgery affects body image, e.g. colostomy, head and neck surgery or mastectomy. An understanding ear or referral to a self-help group (Appendix C) may prevent a sense of loss degenerating into long-term depression.

Fluid imbalance causes electrolyte disturbance and can lead to desaturation even if gas exchange is adequate (Westbrook and Sykes, 1992). Hypovolaemia is due to pre- and post-operative fluid restriction, the drying effect of premedication, unhumidified anaesthetic gases and tissue trauma (Rosenthal, 1999). *Postural hypotension* may be a sign of unrecognized hypovolaemia and is a reminder to avoid sudden motion or position change. Fluid overload may be caused by over-enthusiastic fluid replacement.

Urine retention, flatulence or constipation impairs excursion of the diaphragm. Urine retention may be helped by acupressure to CV2 on the midline of the upper border of the symphysis pubis (Ellis, 1994, p. 104). Flatulence may be relieved by pelvic tilting and knee rolling in crook-lying.

Hypoxaemia increases the risk of *wound infection* (Whitney, 1989), which is suspected if there is fever, swelling, erythema or increased localized pain. Wound infection increases the risk of *dehiscence*, especially if the patient is malnourished, obese, immunocompromised or has malignant disease or a history of radiotherapy or longterm steroid use. Wound infections are not detected for several days postoperatively, but perioperative oxygen reduces their incidence (Greif *et al.*, 2000).

Hypothermia occurs during surgery because of exposure of internal organs, fluid administration, vasodilating anaesthetic drugs and prevention of shivering by paralysing drugs. Complications include wound infection, impaired coagulation and delayed hospital discharge (Leslie and Sessler, 1999).

Incessant *hiccups*, caused by irritation of the diaphragm, cause sharp pain at the wound site. They may be inhibited by baclofen (Walker, 1998a), sugar, acupressure to CV17 (p. 84), an array of techniques to raise P_aCO_2 (breath-holding, rebreathing, drinking a glass of water from the wrong side of the glass), dropping a piece of ice down the back to hyperextend the neck, or prayers to St Jude, the patron saint of lost causes.

Hypertension is most likely after abdominal aneurysm repair, carotid endarterectomy or intracranial surgery (Frost, 1996).

Neurological problems such as dementia or stroke may follow apparently uncomplicated surgery (Sharpe and Hanning, 1999).

Postoperative haemorrhage, due to surgical complications or deficient clotting mechanisms, is suspected if there is:

- obvious bleeding
- rapid filling of drainage bottles
- signs of hypovolaemic shock (p. 362).

Persistent bleeding that does not respond to correction of haemostatic abnormalities may require electrocoagulation or exploratory surgery.

Deep vein thrombosis (DVT) is a blood clot that develops surreptitiously, usually during surgery. Causes are calf compression, immobility, fluid loss, manipulation of blood vessels, the surgical stress response which upsets clotting (Edmondson, 1994) and depression because of the serotonin effect on platelet aggregation (Seiner, 1999). DVT complicates one-fifth of major operations (Ashby, 1995) but is under-diagnosed because it is clinically silent in 50% of patients (Bright, 1995). Signs may include tenderness, swelling and warmth of the calf, or sometimes pain on dorsiflexion (Homan's sign), any of which must be reported. Diagnosis can be confirmed by ultrasound or Doppler imaging. A

DVT becomes serious if it breaks free and causes *pulmonary embolism* (p. 113) by lodging in the pulmonary vascular bed. Patients most at risk are those who:

- are elderly, obese, or have malignant, vascular or blood disorders
- are undergoing lengthy surgery, especially of the knee, hip or pelvis, which involve distortion and traction of blood vessels
- have had a previous history of DVT.

Awareness during anaesthesia is a feared complication that occurs in one out of 500 operations. If patients speak of this, it should be reported because it can lead to post-traumatic stress disorder (Ghoneim, 2000).

Postoperative recovery and shortened hospital stay is facilitated by adequate pain relief, intensive nutrition, early mobilization (Moniche *et al.*, 1995), a leafy view through the window (Ulrich, 1984), positive suggestions under anaesthesia (Williams *et al.*, 1994) and the measures described in the next section.

PREOPERATIVE MANAGEMENT

Is physiotherapy necessary preoperatively? Preoperative physiotherapy consisting of information and advice on positioning, mobilization and chest clearance has shown a reduction in postoperative complications, increased S_aO_2 and improved mobilization (Olsen *et al.*, 1997). Preoperative exercise and education have shown more speedy postoperative recovery (Athur *et al.*, 2000). Information alone has been shown to reduce complications (Cupples, 1991), increase ability to deep breathe and cough (Lindeman, 1971), reduce analgesic requirements by half and lead to discharge nearly 3 days earlier (Egbert *et al.*, 1964). Some of these studies would not be current with today's surgery but illustrate the power of information. This is especially important for children and those expecting to wake up in the ICU, where they will feel relieved at the sight of a familiar face.

Information relieves anxiety, and anxiety is related to postoperative complications (Gilbert, 1996). Stress lengthens hospital stay (Liu, 1994) and can contribute to muscle breakdown, delayed healing and immunosuppression (Salmon, 1992). Anxious patients should be seen early if possible because anxiety at impending surgery inhibits receptivity (Cupples, 1991). If musculoskeletal problems are anticipated, e.g. after IMA grafting (p. 270), liaising with surgeons is advisable (El-Ansary *et al.*, 2000).

Some patients find it beneficial to have relatives present for the preoperative visit. The following can be included:

- Assessment
- Explanations, e.g.:
  - surgery leads to inadequate lung expansion, so that activity and sometimes deep breathing are needed after the operation
  - if there is extra sputum, breathing exercises and coughing may be necessary
  - prevention is paramount
- Advice to ask for adequate relief of pain or nausea.
- Information specific to the operation. Most patients like to know about the wound, drips, drains and what it will feel like. People undergoing complex procedures may benefit from visits by patients who have had similar surgery.
- Advice to keep active before surgery.
- For high-risk or anxious patients, information on how to roll, deep breathe, use the incentive spirometer, sit up and cough with minimum pain.
- Any questions?

People with hypersecretory lung disease may need assistance with sputum clearance. Those in hospital for longer than a day preoperatively need advice on a mobility regime. Those expecting to wake up on a ventilator need information on the endotracheal tube, the experience of positive pressure ventilation and suction (Jablonski, 1994), advice that they may hear before being able to respond, suggestions on methods of communication, and reassurance that there will be a nurse watching over them. Visits

to the ICU by the patient and family are often helpful, after careful explanations. Patients are advised to stop smoking, although this is best started months previously. However, last-minute smoking cessation reduces carbon monoxide levels and improves cardiovascular status (Munday *et al.*, 1993). Some of this information may be covered by other members of the team.

Anxious people benefit from relaxation (Mogan *et al.*, 1985). Mindless and inaccurate reassurance does not engender trust and can impair the 'work of worry', which is a natural and necessary part of adjusting to the operation and its outcome (Salmon, 1992). Reassurance is helpful if anxiety is unrealistic (Teasdale, 1995). Postoperative distress is related to lack of knowledge (Salmon, 1992), and information can be reinforced by written advice.

The tradition of prolonged preoperative fluid and food restriction is now considered unjustified. Fluid restriction may lower stomach pH because of reduced dilution of gastric secretions (Greenfield, 1997), and hypovolaemia causes more perioperative deaths than water in the stomach (Thomas, 1987). Clear oral fluids 2 hours before surgery improves comfort, reduces dehydration and makes it easier to expectorate postoperatively without compromising safety (Phillips *et al.*, 1993). For patients at risk of nutritional compromise, preoperative carbohydrate-rich drinks can be beneficial and can pass safely through the stomach within 90 minutes (Nygren *et al.*, 1996).

Drug dosage to prevent pain is significantly less than that required to abolish pain after it has occurred (Katz *et al.*, 1994). Before surgery, pre-emptive analgesia reduces postoperative pain by preventing noxious impulses gaining entry into the central nervous system, where they 'wind up' the response to subsequent afferent inputs. Neural blockade of these stimuli is assisted by adding anti-inflammatory drugs or morphine to the premedication (Lascelles, 1997) or using preoperative nerve blocks (Lindgren, 1997). Postoperative phantom pain is related to the degree of pre-amputation pain (Nikolajsen, 1997) and can be eliminated by preoperative epidural analgesia (Cousins, 1989), or local anaesthetic infused into the sciatic nerve (Pavy and Doyle, 1996).

PAIN MANAGEMENT

> ***A visit to most postoperative wards will show you the time-honoured ritual of inadequate pain management. Like most unpleasant things in life we have done our best to ignore the situation in the hope that it will disappear.***
>
> Harmer, 1991

Unnecessary postoperative pain still occurs despite advances in medication, improved surgical technique and the advent of acute pain teams. These teams have reduced postoperative morbidity (Hall and Bowden, 1996) and surgical patients now receive better pain management than medical patients (Gray, 1999). However, unnecessary pain still occurs because of:

- a wide variation in patients' perception of pain
- a wide and unpredictable variation in response to analgesics
- rudimentary pain assessment
- inexperience, tradition and staff overwork (Justins and Richardson, 1991)
- ignorance of the difference between abuse and therapeutic use of opioids (Hanks, 1996)
- ignorance of the fact that addiction occurs in fewer than 1 in 3000 people who take analgesic drugs (Lavies, 1992)
- ignorance of the fact that post-opioid euphoria is not the same as respiratory depression (Lindley, 1990)
- an attitude that pain is unimportant, inevitable and to be borne with fortitude, especially in cultures such as Britain's that see stoicism as a virtue and distress as a weakness
- patients' low expectations of pain relief and anxiety about side effects (Sutcliffe, 1993).

The concept of pain includes both the sensation and the individual's reaction to that

sensation. Pain is a subjective experience, but one study found that 50% of nurses doubt patients' reports (McCaffery and Ferrell, 1992). It is not unusual to hear patients dismissed as having a 'low pain threshold' or even 'making a fuss'.

> ***Whose pain should the physician control: The patient's? That of the relatives? Or his own, generated by his inability to help the patient?***
>
> Szasz, 1968

Effects of pain

The relationship between pain and atelectasis (Figure 10.2) can be understood readily by anyone trying to take a deep breath when in the dentist's chair. Pain not only inhibits breathing, it increases oxygen consumption and the risk of infection, delays healing and hospital discharge, and can increase morbidity and mortality (Cheever, 1999). It is associated with anxiety (McGrath and Frager, 1996), causes hypertension and hyperglycaemia, upsets electrolytes, further increases pain by causing muscle spasm and can mask signs of hypovolaemia, which in severe cases predisposes to multiple organ failure (Sutcliffe, 1993). Unrelieved pain is thought to be the main factor limiting the expansion of day surgery (Rawal, 1997).

Prolonged pain can become chronic pain by central sensitization (Anand, 1998). Crombie (1998) found that at one pain clinic, long-term postoperative pain was shown to be the cause of referral in 22.5% of patients.

Assessment

Problems arise when staff make decisions for patients, which can lead to one-quarter the prescribed analgesia being given (Rosenberg, 1992). Postoperative pain should be assessed and graphed like any other vital sign (Sjöström *et al.*, 1997), e.g. Figure 10.3, 10.4 or for computer buffs, interactive computer animation (Swanston, 1993). Pain on movement should be included in the assessment (Hall and Bowden, 1996).

Pain assessment is a right for people who are cognitively impaired or do not speak English, not just those who can complain in a way that is easy to understand. For patients with confusional states or intellectual impairment, pain assessment needs to be modified in order to avoid problems that can themselves increase medical complications (LaChapelle *et al.*, 1999). Elderly people are at particular risk of undertreatment and the majority of those with dementia have been found to suffer severe post-

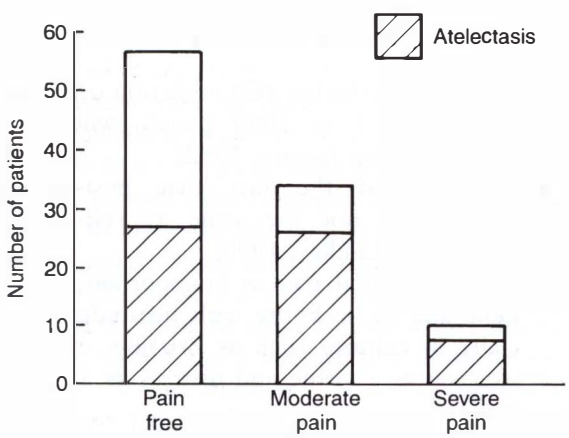


Figure 10.2 The effect of pain on atelectasis. (From Embling, S. A. (1985) *Incidence, Aetiology and Implications of Atelectasis following Cardiopulmonary Bypass Surgery*. MSc dissertation, University of Southampton, with permission.)

Verbal rating score for pain	
Patients' feeling	Score
No pain on movement	0
Mild pain on movement	1
Moderate pain on movement	2
Severe pain on movement	3

Figure 10.3 Pain scale that incorporates movement and is particularly helpful before physiotherapy. (From Hall, P. A. and Bowden, M. I. (1996) Introducing an acute pain service. *British Journal of Hospital Medicine*, 55, 15–17)

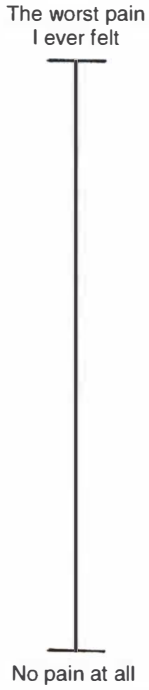


Figure 10.4 Visual analogue scale.

operative pain (Morrison and Siu, 2000). The opinion of relatives may be helpful. Pain assessment for children and infants is on page 432.

If pain cannot be assessed by the patient, objective signs are pallor, sweating, shallow breathing, breath-holding and increased pulse, BP and respiratory rate. Severe pain causes nausea, vomiting and reduced pulse and BP.

Reduction in the perception of pain

> ***There are many things that make pain worse, such as the spirit in which it is inflicted. You are indeed acutely vulnerable to the attitude of people surrounding you.***
>
> Donald, 1977

Perception of pain varies with some factors that physiotherapists cannot modify such as the operative technique and previous experience. However, physiotherapists can modify other factors:

- anxiety or fear
- physical discomfort
- physical tension
- lack of autonomy or privacy
- depression
- sleep fragmentation (McIntosh, 1989).

This is fertile ground for the physiotherapist. Physical tension can be eased by posture change or relaxation (Miller and Perry, 1990). Anxiety can be reduced by keeping patients informed. Autonomy can be enhanced by including them in decisions. During activity, patients need reassurance in words and actions that they will be heard and responded to. 'Tell me if it hurts and I'll stop' is music to their ears.

Handling patients in pain

> ***'... pain works subversively, undermining one's self-confidence and self-control, worn dismayingly fragile. The sense of anticipation is honed, to hysteria almost, and one quickly learns to be thoroughly suspicious of the well-meant: "this won't hurt".'***
>
> Brooks, 1990

Physiotherapists should be seen as experts in the relief of pain rather than its perpetrators. The essence of physiotherapy is skilful handling, and there are few rewards greater than relief on the face of a patient whose pain we have eased. Handling and positioning have been found to be as important in relieving acute pain as drugs (Sutcliffe, 1993).

Guidelines are the following:

- Patients must be assured that they are in control.
- Analgesia should be given automatically before physiotherapy, instead of first checking to see if treatment causes pain, a strategy known as shutting the stable door after the horse has bolted.
- Unnecessary handling should be avoided.
- The patient should be informed of how and when each movement will take place. Words

to avoid are 'just relax', which signals to any seasoned patient that they are about to be hurt, or 'sorry' after an unexpected movement instead of clear explanations before the movement. The 'wince–sorry' scenario is familiar to those who have witnessed patients being routinely hurt and then routinely apologized to.

Manual handling for patients in pain incorporates the principles of offering advice and support but allowing patients to move themselves as much as possible.

For rolling into side lying, patients can be asked to bend their knees, shift away from the direction in which they are to roll, hold onto the bed rail, push with their knees and roll in one piece. If a bed rail is not available, physiotherapists can ensure that the bed is the right height, then press their own fist into the bed, to protect their back, and the patient holds on to their straight supported arm. Patients are encouraged to emphasize pushing with their legs rather than pulling with their arms, in order to inhibit abdominal muscle work.

For any manoeuvre that entails abdominal muscle work, e.g. eccentric contraction when lying back against the pillows, laparotomy pain can be reduced by facilitating active back extension and thus reciprocal abdominal relaxation.

Medication and routes of administration.

> ***Freedom from pain should be a basic human right.***
>
> Liebeskind and Melzack, 1987

If an acute pain team is not available, physiotherapists need to be involved in the team management of pain. Analgesics are based on the medication being titrated to effect rather than on a per-kilogram basis (Tobias, 1994).

Morphine remains the favourite opioid analgesic, with a half-life of several hours. Side effects include nausea, constipation, abdominal distension, elimination of spontaneous sighs and, in hypovolaemic patients, hypotension. Large doses of morphine depress respiration, but sedation should not be interpreted as respiratory depression (Pasero, 1994), and clinically significant respiratory depression is accompanied by signs such as somnolence, mental clouding (Cherny and Foley, 1997), rapid shallow breathing and greater rib-cage contribution to tidal volume (Leino *et al.*, 1999). Depression of respiration is reversible by the opiate antagonist naloxone (Narcan). Opioid addiction is rare unless administration is continuous in a patient who has no pain (Aitkenhead, 1989). Well-managed narcotic drugs improve ventilation and gas exchange because breathing is made easier by relief of pain (Harcus, 1977).

'Balanced' analgesia combines drugs to influence different physiological processes, and can provide almost complete analgesia at rest and during mobilization (d'Amours, 1996). Non-steroidal anti-inflammatory drugs (NSAIDs) engage the peripheral nervous system and help reduce opioid-induced side effects (Ganea and Bogue, 1999). The synergistic effect of epidural opioids and local anaesthetic is particularly useful for pain on movement (Cook, 1997). Balanced analgesia is necessary for opioid addicts, who have a high tolerance to the drug and may need a combination of epidural, PCA and infusion (Connor and Muir, 1998).

Analgesia can be delivered systemically (oral, intramuscular, intravenous), or regionally. Regional analgesia blocks transmission within the peripheral nervous system, does not befuddle the entire central nervous system and causes little nausea.

Intramuscular route

The time-honoured 'p.r.n.' injection is unjokingly referred to as 'pain relief never'. It is delivered as required, has no rational basis, produces wide fluctuations in serum levels, leaves pain unrelieved in half the recipients (Jacox *et al.*, 1992) and augments a vicious cycle of anxiety and pain. Patients in pain also require more staff time. P.r.n. analgesia is berated in the literature but still used in some hospitals.

Regular intermittent dosage is more effective,

because it takes less drug to prevent pain than to subdue it, but dosage may still be inadequate because of wide variations in uptake, distribution and elimination, especially in patients who are cold, dehydrated (d'Amours, 1996) or elderly. Blood concentration varies by at least a factor of 5 and the concentration at which each individual becomes pain-free varies by a factor of 3 or 4 (Justins and Richardson, 1991).

Intravenous route

The IV route works immediately and can be delivered continuously or in boluses. Morphine is commonly used. Fentanyl is a synthetic opioid that is 100 times as potent as morphine and useful prior to physiotherapy because of its rapid onset, short duration of action and lack of disturbance to hypovolaemic or haemodynamically compromised patients (Oh, 1997, p. 680).

Patient-controlled analgesia (PCA) delivers a preset dose of drug, usually intravenously, by syringe pump when the patient presses a button. This accommodates to individual need, reduces anxiety, encourages mobility, reduces sleep disturbance, is preferred by patients for the autonomy it allows, requires less drug to achieve the same pain control and can lead to earlier discharge (Thomas, 1995). A programmed lock-out interval of, say, 5–10 minutes, ensures that each dose achieves peak effect before the next dose is released. Patients must be reassured that they can use the device freely, although their relatives should not press the button. Respiratory depression is rare, although oximetry is advisable if the patient has limited understanding and staffing levels are low. PCA is more effective for dull pain than the sharp pain of coughing. It does not reduce the incidence of nausea, and antiemetics must be given separately if required.

Intercostal route

Blocks to intercostal nerve transmission are used for rib fractures. They are administered by repeated injections into multiple nerves, or more comfortably by continuous and/or extrapleural infusion (Majid, 1992). Respiration is not depressed, but pneumothorax is a risk and the X-ray should be scrutinized if positive pressure techniques are anticipated.

Epidural

The epidural route alters spinal processing by delivering drugs to the epidural space, the catheter being inserted in the operating theatre and left *in situ*. Morphine works directly on the opiate receptors along the spinal cord, and can control pain originating anywhere below the cranial nerves. Analgesics are usually combined with local anaesthetic drugs such as bupivacaine (Berti *et al.*, 2000). Patients should be told that postoperative epidurals are not the same as the well-known anaesthetic epidurals given during childbirth and they will not be completely numb below the catheter. In increasing order of efficacy, administration is by intermittent blockade, continuous infusion (Cook, 1997) or PCA (Mann *et al.*, 2000).

Advantages of epidurals are legion: ↓ pulmonary complications and intubation time (Frost, 1996), ↓ opioid side effects, (Massard and Wihlm, 1998), ↓ stress response and paralytic ileus, ↑ diaphragmatic function and mobilization (Cook, 1997), ↓ oxygen consumption, ↓ incidence of DVT and wound infection, hospital stay shortened by an average of a week (Smedstad, 1992), and ↑ graft blood flow after vascular surgery (Cousins, 1989).

Disadvantages of epidurals are local infection risk, especially with repeated top-ups, partial sensory or motor loss and blockade of sympathetic outflow which is especially noticeable in hypovolaemic patients. Patients receiving intermittent dosage should lie flat for 30 minutes after a top-up to avoid hypotension. High blocks are mainly associated with hypotension, while low blocks may cause urine retention. Respiratory depression is found in less than 1% of patients, usually occurring within ½–1 hour of a top-up or 6–12 hours later (Jacques, 1994), and is reversible with naloxone. Other side effects are nausea and paralytic ileus. Dislodgement can be prevented by subcutaneous tunnelling (Burstal *et al.*, 1998).

Intrathecal
The subarachnoid space can be directly targeted, producing profound analgesia without motor, sensory or sympathetic block. Complications include 'spinal headache' in nearly half the patients (Nilsson, 1997) due to leak of cerebrospinal fluid (CSF) through a punctured dura and loss of the intracranial CSF 'cushion'. If this occurs during mobilization, the patient should be returned to bed to lie still.

Oral
Drugs can be administered orally several days after surgery if acute pain has subsided. Effects are variable.

Transdermal
For a local painful procedure, EMLA (eutetic mixture of local anaesthetics) skin patches create superficial anaesthesia when applied to the skin an hour beforehand. No child should be submitted to venipuncture, lumbar puncture or indeed, any injection without prior application of their 'magic cream'. Needle-phobic adults also benefit (Biro, 1997).

Transmucosal
Mucous membranes impose less of a barrier than skin, as cocaine abusers have discovered. Drugs such as buprenorphine administered sublingually are speedily absorbed.

Other routes
If the paravertebral, intrapleural, extrapleural or extradural routes are used, pneumothorax is a risk.

Cryoanalgesia

Pain after thoracotomy or rib fracture can be eliminated by cryoanalgesia, an open procedure that freezes the intercostal nerves, creating total pain relief until they regenerate over a period of weeks or months. Its use is limited because 20% of patients develop neuralgias (Kavanagh, 1994).

Entonox

Short-lived analgesia can be achieved by inhaling a 50% mix of nitrous oxide and oxygen (Entonox or laughing gas), delivered from a cylinder by face mask and demand valve (Figure 10.5), or an IPPB machine or ventilator. Entonox is not metabolized, but eliminated unchanged by the lungs so that side effects such as light-headedness, drowsiness or nausea are minimal (Sacchetti, 1994). Entonox is also suitable for children able to understand its use (Lawler, 1995).

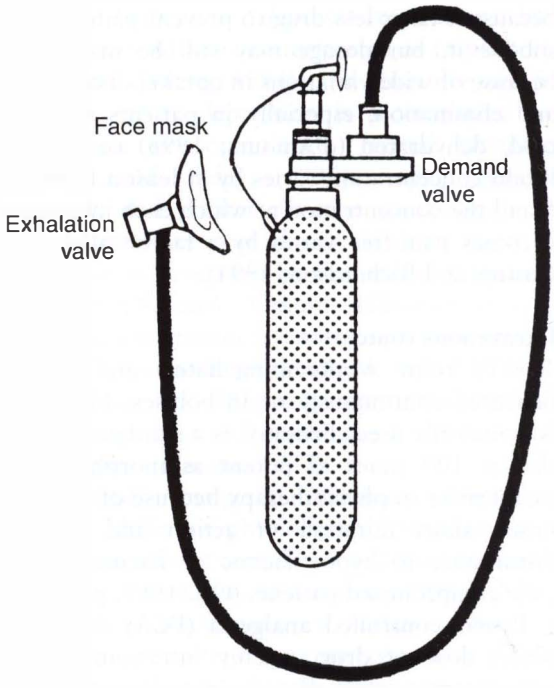


Figure 10.5 Entonox cylinder for rapid pain relief.

Patients need to hold the mask firmly over their face and inhale with sufficient force to activate the demand valve. It is usually effective in 2–3 minutes but occasionally up to 10 minutes is needed. The gas must be self-administered so that drowsiness would cause the mask to drop away and prevent overdosing. Patients must be under observation until fully alert.

If used continuously for over 32 hours, bone marrow changes occur (Oh 1997, p. 683), but this is not a problem with intermittent use. Entonox is not utilised for the many minor but

distressing hospital procedures for which it is ideal. Local protocols govern its use in individual workplaces.

Precautions are:

- Sealed pockets of air (e.g. surgical emphysema, bullae, pneumothorax, bowel obstruction, ear surgery, tracheal tube cuffs or balloon-tipped catheters) because nitrous oxide is 32 times more soluble than air and diffuses into air-filled spaces. The X-ray of a patient with fractured ribs should be examined for a pneumothorax.
- The first 16 weeks of pregnancy.
- Acute head injury because of increased cerebral blood flow.
- Severe heart failure because of a risk of pulmonary oedema (Hahn, 1997).

Entonox is unsuited to patients who need more than 50% oxygen. Conversely, hypercapnic COPD patients dependent on a hypoxic drive to breathe require a lower proportion of oxygen and may not be suitable because less than 40% nitrous oxide does not provide analgesia (Tobias, 1994). Cylinders must be stored on their side when not in use, as nitrous oxide liquefies and falls to the bottom.

Transcutaneous electrical nerve stimulation (TENS)

Analgesics are normally adequate, but there are occasions when TENS is useful. It is time-consuming for the physiotherapist, but does not depress the respiratory system, is non-invasive, non-toxic and produces mobile and happy patients. TENS can decrease narcotic requirements, reduce pulmonary complications and improve mobility (Wang *et al.*, 1997). Agreeable side effects include reduction in nausea and paralytic ileus (Akyüz, 1993). The following are practical points additional to conventional TENS.

Skin sensation is checked because it is ineffective to stimulate anaesthetic areas, and stimulation of areas of hyperaesthesia worsens pain. Two or four electrodes are applied, half-way along the incision or close to each corner, as soon as possible after surgery. Alternatively, acupuncture points can be stimulated. If sterile electrodes are used, two long electrodes are applied in theatre alongside the wound under the dressing, with controls set at a level determined before surgery.

Re-adjustment is necessary as the patient adapts to the sensation or becomes more awake. Patients can use the pulsed or boost mode to reduce accommodation or when moving or coughing. They can adjust their own controls, decide how many days to use it, and whether to continue at night. Liaison with nursing staff and daily skin washes are needed.

TENS near the chest is contraindicated for people using a synchronous pacemaker, and TENS near the head is inadvisable for people with epilepsy (Scherder, 1999). The electrodes must be placed away from ECG electrodes to minimize interference (Sliwa and Marinko, 1996). Stimulation should not be applied over a pregnant uterus or damaged skin.

Phantom limb pain has shown a response to TENS applied to sites on the remaining limb corresponding to painful sites on the amputated limb as identified by the patient (Kawamura, 1999).

Other techniques

Acupressure may reduce postoperative pain (Felhendler, 1996) and hypnosis can be useful if analgesics are inadequate (Ohrbach *et al.*, 1998).

POSTOPERATIVE PHYSIOTHERAPY

Is physiotherapy necessary for postoperative patients? Research has shown positive outcomes (Chumillas *et al.*, 1998; Hall *et al.*, 1996; Ntoumenopoulos and Greenwood, 1996; Warren, 1980). But individual techniques need scrutiny to identify which are appropriate for which patient.

Postoperative physiotherapy is based on techniques to increase lung volume and clear secretions (Chapters 6 and 8). Modifications specific to surgery are discussed here.

Mobility

Bed mobility and independence following abdominal surgery are encouraged with a rope attached to the end of the bed by which patients can pull themselves up (Figure 10.6). As soon as possible they are encouraged to sit with their legs dangling over the edge of the bed. Post-operative mobilization out of bed helps increase lung volume, improve $\dot{V}_A/\dot{Q}$ matching and mobilize secretions (Cockram *et al.*, 1999). If surgically and medically acceptable, this should be on the first postoperative day. Intravenous equipment that is plugged into the mains can usually be temporarily unplugged for ambulation but the standby battery must be functioning. For patients with a urinary catheter, leg bags are more dignified than loose catheter bags. Posture correction is incorporated as soon as discomfort has eased. For safety aspects, see page 148.

Positioning

If pain, surgical procedure or instability delay mobilization, emphasis should be on accurate and comfortable positioning, alternating from side to side and if possible sitting out of bed. Positioning can re-expand atelectatic lung (Westbrook and Sykes, 1992), but regular position change is needed to prevent atelectasis reappearing in dependent zones. If lying well forward on the side (p. 150) pulls on the wound, the position may need to be modified. Clinical assessment and the X-ray (Figure 10.7) will assist in decisions about positioning.

Breathing exercises

Breathing exercises are not usually necessary even after major surgery (Stiller *et al.*, 1994) but, if mobilization is delayed, positioning limited, or respiratory complications develop,

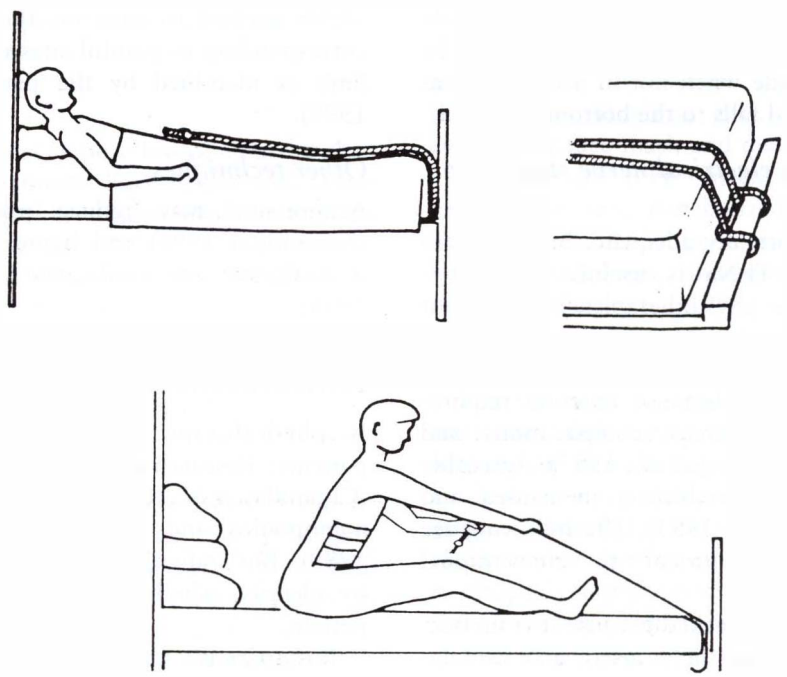


Figure 10.6 Rope to assist independent bed mobility.

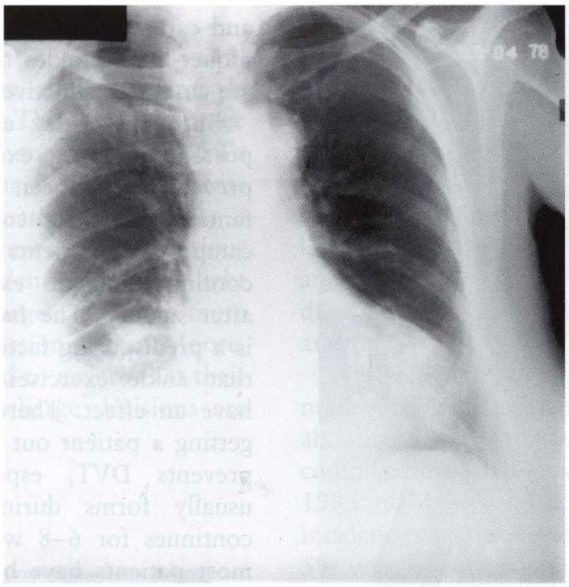


Figure 10.7 Opacity in right lower zone suggests consolidation. Upward shift of right hemidiaphragm suggests some lower lobe collapse. Thin angled line on the right suggests segmental collapse rather than displaced horizontal fissure because it does not attach to the hilum. Opacity in left lower zone is probably breast shadow but auscultation is required to rule out consolidation. Positioning should be in alternate side-lying, with extra time in left-side-lying to encourage expansion of collapsed lung tissue.

deep breathing is required. After oesophagectomy, or upper abdominal surgery in high risk patients, regular prophylactic deep breathing is advisable.

Deep breathing is done in a position that achieves a balance between comfort and optimal ventilation, usually well-forward-side-lying. For patients who cannot achieve this, upright sitting is the next option.

Accumulated secretions are usually cleared postoperatively as mucociliary transport recovers. Superficial secretions in the throat can be dispersed by throat-clearing. Stronger expiratory manoeuvres are used if necessary, but expiration beyond FRC causes airway closure, which is not easily reversible in patients who are weak and in pain (Craig, 1981). Unnecessary forced expiration, coughing, percussion and vibrations cause pain and splinting, and may simply create the lesion that they are supposed to cure.

If there is subjective or objective evidence of accessible secretions, and coughing is necessary, patients may prefer to remain in side-lying, but if they are willing, sitting over the edge of the bed is mechanically efficient and allows maximum support (Figure 10.8). Pressing on the incision with a pillow is less effective than sensitive and accurately timed manual support but, when patients are alone, they may find a pillow, towel or cough belt (see Figure 10.15) helpful. If patients are too weak, fatigued or drowsy to co-operate, mechanical aids may be indicated.

Prevention of deep vein thrombosis

Half the deaths from pulmonary embolism could be avoided by prophylactic guidelines for DVT prevention (Forbes, 1994). Some examples are:

- intermittent or sequential pneumatic leg compression devices during and after surgery, so long as there is no arterial disease (Oakley *et al.*, 1998)

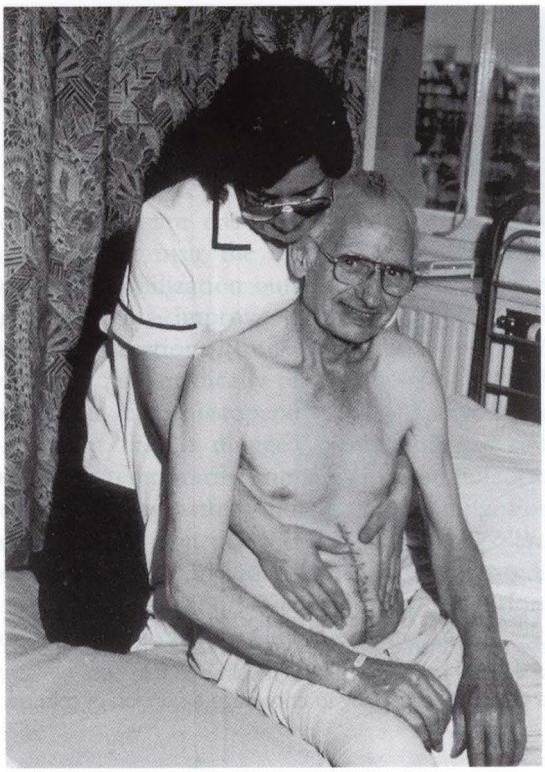


Figure 10.8 Manual support for postoperative coughing after laparotomy. Gentle firm pressure is directed at holding the wound edges together. After thoracotomy, the physiotherapist can sit beside the patient on the opposite side to the wound so that s/he can support the wound while giving counterpressure to the patient's body.

- leg elevation before, during and after surgery, to avoid calf compression (Ashby, 1995)
- avoidance of leg-crossing
- for high risk patients, drugs such as dextran or heparin
- graduated thromboembolism (TED) stockings to facilitate deep venous blood flow.

TED stockings must apply graduated compression that is greater in the lower calf and diminishes up the leg, and must be individually fitted, otherwise they are of no value (Moser, 1990). Excessive pressure may reduce deep venous flow (Lawrence and Kakker, 1980). The physiotherapist's main task is to advise patient and carers to smooth out the omnipresent tourniquet-like wrinkles that reduce blood flow and are counterproductive (Sigel, 1973).

But whither leg exercises? The theory that postoperative leg exercises have any place in prevention has been relegated to the realms of fantasy, unless someone can be found to set up camp beside patients and prod them into near-continuous ankle exercises both during and after surgery. The fact that sluggish circulation is a predisposing factor for DVT does not mean that ankle exercises after the operation will have an effect. There is also no evidence that getting a patient out of bed several times a day prevents DVT, especially as the thrombus usually forms during surgery and the risk continues for 6–8 weeks after discharge when most patients have been fully mobile for some time (Forbes, 1994). It is also remotely possible that exercising the ankles could trigger an asymptomatic DVT to form a pulmonary embolus, as has been reported following application of a sequential compression devise (Siddiqui, 2000).

Two decades-old studies have been unsuccessful in proving the benefits of ankle exercises for DVT prevention:

- Flanc (1969) found that a combination of elastic stockings, elevation of the foot of the bed and six-times-daily physiotherapy-supervised leg exercises reduced the incidence of postoperative DVT in elderly people, without indicating which component was effective
- Scurr (1981) found that DVT formation during surgery was reduced by a mechanical device that continually moved the ankle during the operation.

Exercises are, however, necessary after orthopaedic surgery or if bed rest is prolonged, in order to prevent joint stiffness and muscle weakness. If DVT has been confirmed, leg exercises and mobilization are contraindicated until several days after anticoagulation therapy (usually heparin) has been established, or after discussion with medical staff.

Discharge

Advice on self-management after discharge is often the main intervention. This is to encourage progressive activity suited to the individual's lifestyle, along with regular rest. For patients who have been doing breathing exercises, a reminder to stop them prevents conscientious patients continuing them indefinitely. In the light of evidence that early discharge can lead to more complications and re-admission rates (Moore, 1994), physiotherapists need to ensure that their voices are heard when discharge decisions are taken.

ABDOMINAL SURGERY

> ***The effect of an upper abdominal incision seems to strike at the root of normal respiration.***
>
> Bevan, 1964

Abdominal surgery impinges less on respiration now that most abdominal organs are amenable to the laparoscope, but procedures such as laparoscopic cholecystectomy take longer than laparotomy and entail tilting the head down and pumping CO_2 into the peritoneum. This impairs diaphragmatic function (Lindgren, 1997) and can refer pain to the right shoulder. However, compared to laparotomy, laparoscopy causes less pain, lung dysfunction and mood depression and allows patients to be discharged and return to work sooner (Freeman and Armstrong, 1994).

Full-incision surgery of the upper abdomen is more problematic than either chest or lower abdomen. It is associated with more pulmonary complications than chest surgery (Luce *et al.*, 1993, p. 300), and is followed by a 20–40% incidence of chest complications compared to 2–5% with lower abdominal surgery (Celli, 1993). Patients having lower abdominal surgery, e.g. via the transverse incision in Figure 10.9, do not require formal physiotherapy (Giroux *et al.*, 1987) unless they fall into a high-risk category, but it is advisable to check pain control and mobility.

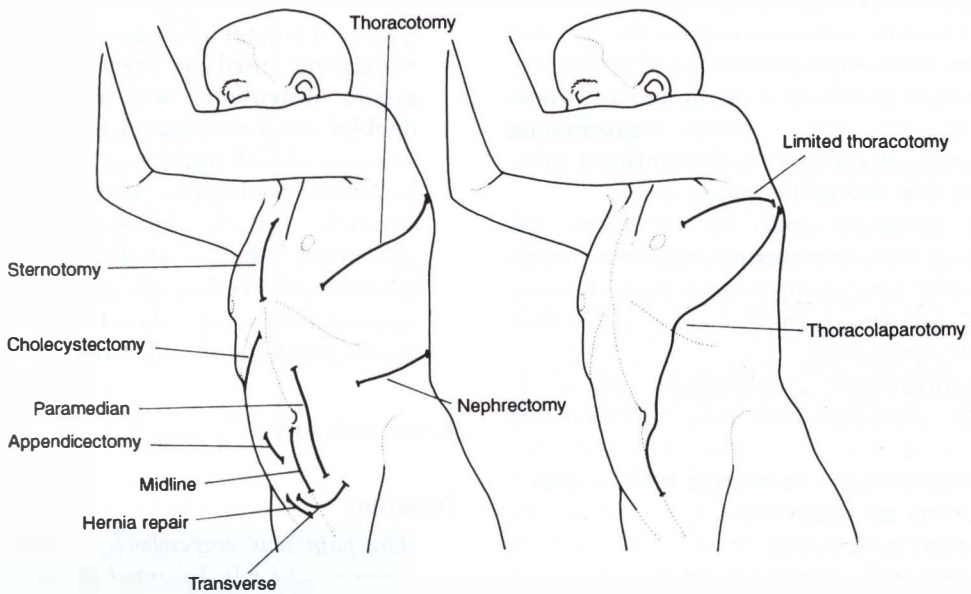


Figure 10.9 Conventional surgical incisions.

Bowel resection leads to significant loss of exercise tolerance. Up to 40% maximal workload is lost, which directly relates to loss of employment (Brevinge, 1995), thus reinforcing the need for rehabilitation.

An *abdominal aortic aneurysm* (AAA) means that the vessel has bulged to twice its normal size (Sternbergh, 1998). Severe abdominal pain and backache suggests a contained rupture, which leads to complete rupture after 2–3 hours. Mortality from surgical repair is 5% before the vessel leaks and 50% afterwards (Bell, 1996). A long midline or flank incision leads to prolonged paralytic ileus, large fluid shifts and significant pain (Cerveira *et al.*, 1999). There is a risk of cardiovascular instability, and patients are not usually mobilized for at least 2 days.

Patients who have had an **aortofemoral graft** should avoid hip flexion on the affected side for 3 days. They may mobilize before they are allowed to sit.

Complications of abdominal surgery, in addition to those previously described, are the following:

- **Paralytic ileus**: Loss of gut activity silences bowel sounds and is normal for the first day or two. It becomes problematic if prolonged, and causes distension if swallowed air is held up in the atonic colon, exacerbating diaphragm dysfunction and requiring a nasogastric tube and nil-by-mouth order.
- **Pain**: Analgesia tends to be taken less seriously than after sternotomy, even though pain after laparotomy is often worse because most activity requires some abdominal muscle contraction.
- **Malnourishment**: Abdominal surgery is usually associated with poor nutrition due to:
  - malabsorption associated with pre-existing gut pathology
  - preoperative fasting
  - the catabolic effects of surgery
  - intestinal handling which affects the delicate mucosal lining (Anup, 1999)
  - postoperative nausea and precarious appetite
  - unfamiliar food.

Poor nutrition reduces mobility, predisposes to infection, depression, muscle weakness, pressure sores and prolonged hospital stay, and delays wound healing (Edington *et al.*, 1997). It may or may not be coincidental that while most of the 1980s Maze hunger strikers died after 60 days, the one who had sustained a prior fracture died at 45 days (Leary *et al.*, 2000)

To facilitate rehabilitation, the following are suggested:

- routine nutritional assessment for every patient facing major surgery and a course of preoperative feeding for the 27% of general surgery patients who are malnourished on admission (Edington *et al.*, 1997)
- avoidance of preoperative fasting in excess of 6 hours (Thomas, 1987)
- early postoperative nutrition to reduce complications, length of hospital stay and mortality (Baublys *et al.*, 1997), including enteral feeding in the recovery room for patients at risk, using small bowel access if there is paralytic ileus (Babineau, 1994).

Routes of enteral feeding are the following:

- *nasogastric*: used to prevent vomiting and gastric distension, but hinders coughing, disables the oesophagogastric sphincter and increases risk of aspiration (Smithard, 1995)
- *gastrostomy*: delivers food directly to the stomach via a catheter through the abdominal wall, known as a PEG (percutaneous endoscopic gastrostomy tube)
- *jejunostomy*: also placed percutaneously, avoids problems of acidic stomach secretions.

LUNG SURGERY

Incisions

'The pain was everywhere. I couldn't get across what I felt. I wanted to move myself but they were insistent on moving me. I lost the ability to control the situation. I felt

very alone. The whole thing was one of the most personal experiences of my life. It took a long time to be able to talk about it.'

Katie, a nursing sister, following her thoracotomy

A standard posterolateral *thoracotomy* involves an incision below the scapula, the division of latissimus dorsi and spreading or resection of the ribs. This leads to restricted shoulder and chest wall movement and, if analgesia is not adequate, 'one of the most intense postoperative pain experiences known' (Kavanagh, 1994). Pain is best controlled by regional anaesthesia rather than intravenous opioids (Brodner, 1997). *Muscle-sparing thoracotomy* is less disabling (Landreneau *et al.*, 1996). *Thoracoscopy* can be used for pulmonary, pleural, cardiac and oesophageal surgery (Figure 10.10).

Procedures

If a large bulla is restricting function in a patient with emphysema, it can be removed by thoracoscopic laser *bullectomy*. This shows an average 29% improvement in exercise capacity and sometimes discontinued need for oxygen therapy (Hazelrigg *et al.*, 1996).

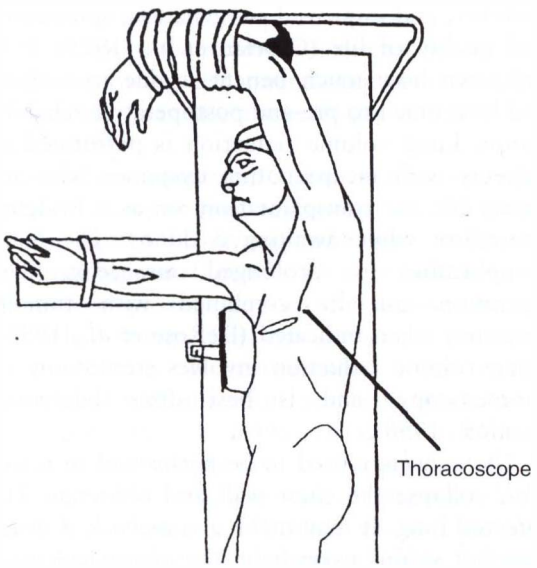


Figure 10.10 Patient in position for minimal-access lung surgery. The shoulder joint ligaments are vulnerable to overstretch. (Redrawn from Benetti, F. et *al.* (1996) Video-assisted minimally invasive coronary operations without cardiopulmonary bypass. *Journal of Thoracic and Cardiovascular Surgery*, 112, 1478–1484, with permission.)

Small peripheral lesions can be removed by *lung biopsy* or *wedge resection* (Giacomo, 1999). A lung segment is occasionally removed by *segmentectomy*.

A patient with a preoperative FEV_1 of more than 2 L is considered to have adequate postoperative pulmonary reserve to undergo *lobectomy*. This is usually to remove a cancerous lobe and may be curative if mediastinal lymph nodes are not involved. The vacated space is accommodated by expansion of the rest of the lung and occasionally shift of the hemidiaphragm and mediastinum (Klein, 1999). With thoracoscopy and meticulous attention to patient education, pain control and mobilization, one-day admissions are possible (Tovar *et al.*, 1998).

A *sleeve resection* is removal of the T-junction of a bronchus with its lobe. This causes twice as much atelectasis as a lobectomy, plus oedema around the anastomosis and ciliary impairment from nerve damage (Massard and Wihlm, 1998). Modified postural drainage may be needed. Positive pressure techniques such as manual hyperinflation may damage the anastomosis or cause a pneumothorax in the immediate postoperative period.

A complete lung is removed by *pneumonectomy*. The lung space is filled with air, blood and fibrin (Figure 10.11), the quantity of which is regulated by one of the following:

- a chest drain, which is kept clamped except when drainage is required
- a temporary small thoracic catheter
- needle aspiration (Deslauriers, 1999).

A chest drain allows recognition of haemorrhage and, if the suture breaks down, prevents a tension pneumothorax, but it increases the risk of empyema and bronchopleural fistula. These drains must never be attached to suction, nor clamped or unclamped by anyone other than the surgeon. Excess drainage of the vacated space

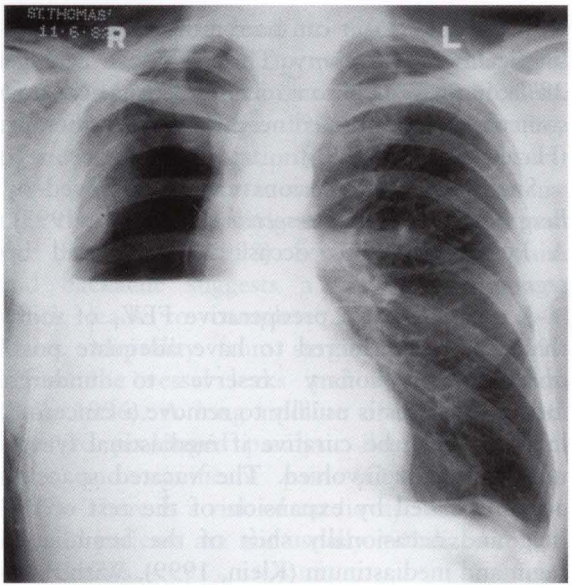


Figure 10.11 X-ray one day after right pneumonectomy, showing a horizontal fluid line separating the exudate from the air-filled space , which shows no lung markings. The trachea is shifted towards the vacated space. Speckled appearance in the soft tissue outside the rib cage on the right is surgical emphysema.

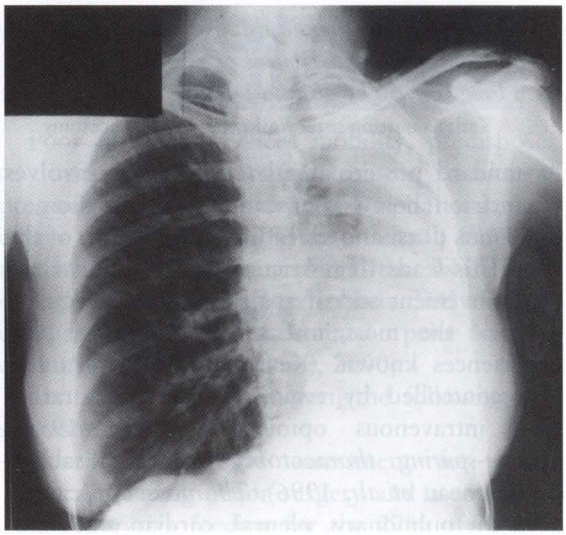


Figure 10.12 Image of a chest some months after left pneumonectomy, showing the vacated space now filled and opaque Also visible are the deviated trachea, healed rib and stitches. The remaining lung shows the cystic appearance of bronchiectasis.

pulls the remaining lung into the space, and too little drainage leads to the bronchial stump becoming soggy (Valji, 1998).

A few days after chest drain removal, the pneumonectomy space has filled with inflammatory exudate. Once the level is above the stump and all is well, the patient goes home. The air is absorbed in 4–6 weeks (Klein, 1999) and, in the ensuing months, the space shrinks by upward shift of the hemidiaphragm, lateral shift of the mediastinum and crowding of the ribs (Figure 10.12). Mortality after pneumonectomy is on average 6.9%, compared to 2.9% after lobectomy (Bisson *et al.*, 1998).

Lung volume reduction is palliative resection of useless lung in patients whose emphysema is characterized by regions of destruction and large bullae that take up space and compress relatively normal underlying tissue. The diaphragm returns to a functional dome shape, which results in decreased work of breathing, greater energy and mobility, and improved oxygenation, spirometry and quality of life (O'Brian *et al.*, 1999). It is unknown how much benefit is due to surgery and how much to pre-and postoperative rehabilitation. Lung volume reduction is performed in patients with incapacitating dyspnoea who are unsuitable for transplantation, or as a bridging procedure while awaiting a donor. The main complication is prolonged air leak. The operation can be combined with tumour resection when indicated (DeRose *et al.*, 1998). Lung volume reduction involves sternotomy or thoracoscopy, and is best done bilaterally (Kotloff, 1998).

Thoracoplasty used to be performed to resect ribs, collapse the chest wall and obliterate TB-infected lung. It may make a comeback if drug-resistant strains overwhelm the pharmacological industry.

Complications

As well as the complications described previously, the following are specific to lung surgery.

Diaphragm dysfunction occurs if the phrenic nerve is injured. Extended resection of hilar or mediastinal tumours may sacrifice the phrenic nerve unavoidably, leading to hemidiaphragm paralysis. During pneumonectomy, some surgeons deliberately cut the phrenic nerve to diminish the residual space.

Following lobectomy, *contusion* in adjacent lung may cause $\dot{V}_A/\dot{Q}$ mismatch and hypoxaemia. Following pneumonectomy, hypoxaemia often occurs on exercise.

Escape of air into the pleura is to be expected after lobectomy. This *air leak* manifests as bubbling in the underwater seal drainage bottle. The chest drain is not removed until bubbling stops.

Escape of air into subcutaneous tissue may occur, causing *surgical emphysema.* This is rarely of more than cosmetic significance, but patients need reassurance that it is temporary. If secretions are a problem, ACB/AD (p. 198) is preferable to coughing. If a swollen face has forced the eyes shut, relatives can be shown how to massage the eyelids to allow temporary vision.

Damage to the recurrent laryngeal nerve, especially following left pneumonectomy or upper lobectomy, may affect speech and cough (Carew *et al.*, 1999).

Problems associated with malignancy (the usual reason for lung resection) include malnutrition and COPD (Wong and Shier, 1997).

Bronchopleural fistula is a breach between lung and pleura, due to breakdown of the bronchial stump. This has the same effect as a pneumothorax. It is a dreaded event, usually associated with infection, and with mortality of over 30% (Varoli, 1998). It is most likely if mechanical ventilation is required, and is suspected if there is a spiking temperature, X-ray evidence of a decreasing fluid level post-pneumonectomy, or expectoration of bloody-brown secretions, especially when lying down with the fistula upwards (p. 152). Empyema usually follows (Deschamps *et al.*, 1999). Spread of infected material is minimized by the patient sitting up or lying on the thoracotomy side. Small fistulae close naturally but large defects need chest drainage, sealing via bronchoscopy (Varoli, 1998), surgery to resuture the bronchial stump or, as a last resort, long-term open drainage. Positive pressure physiotherapy techniques are inadvisable. Patients requiring mechanical ventilation might benefit from a high-frequency system (Campbell *et al.*, 2000).

Average *functional effects* include the following:

- Lobectomy leads to 10% reduced spirometry values but negligible loss of exercise capacity.
- Pneumonectomy leads to 33% reduced spirometry, 20% loss of exercise capacity and some dyspnoea on exercise (Bolliger, 1998).

Long-term pain occurs in 50% of patients but can be reduced by aggressive pain control immediately after surgery (Katz *et al.*, 1996).

Physiotherapy

Following uncomplicated thoracoscopic surgery, patients can sit out 4–6 hours postoperatively (Nicholson, 1993) but chest assessment must continue.

Following thoracotomy, many patients require respiratory care. Shoulder and postural exercises begin once pain allows. This maintains range of movement and is sometimes seen to improve S_aO_2. Some patients suffer pain from thoracic joints stretched during surgery, which may be eased by mobilizations of the joints at the spine. Progressive exercise should proceed apace, and some weeks after discharge, patients benefit from outpatient rehabilitation.

Points to note in relation to pneumonectomy are the following:

- If sputum clearance is necessary, ACB/AD is preferred to coughing, in order to protect the stump.
- Following a normal pneumonectomy, patients should not lie on the *non-operated* side, to prevent fluid spilling onto the stump, and some surgeons demand that this be lifelong.

- Patients should not lie on *either* side after radical pneumonectomy, which entails entering the pericardium, in case of cardiac herniation.
- There should be no head-down tip and, for some patients, no lying flat.
- Suction, if necessary, should be shallow.
- Normally the remaining lung is able to accommodate the entire resting pulmonary blood flow but, during rehabilitation, pulmonary hypertension may occur on exercise.

PLEURAL SURGERY

The commonest indication for pleural surgery is recurrent, bilateral or persistent pneumothorax. Other indications are problematic pleural effusions or bronchopleural fistulas. Pleural surgery leaves a long-term mild restrictive defect.

A *pleurodesis* introduces irritant chemicals, tetracycline, fibrin glue or laser pulses into the pleura via thoracoscopy, setting up a sterile inflammation, fibrosis and adherence of the two layers of the pleura. If sterile talcum powder is the irritant chemical, it should be confined to elderly people because it is carcinogenic and systemic distribution may occur over time (Werebe, 1999). Procedures relying on an inflammatory response should not be followed by anti-inflammatory analgesia. Kindly surgeons instil local anaesthetic into the pleura before closure.

A *pleurectomy* strips off the parietal pleura so that a raw surface is left at the chest wall, to which the visceral pleura adheres. This may require a thoracotomy but is less painful than the acute pleurisy set up by a pleurodesis.

Chronic empyema may be managed by debridement via thoracoscopy, but if infection is not contained, *decortication* may be required, which involves peeling off the thickened fibrosed visceral pleura. The parietal pleura is spared unless long-standing empyema and deformity mean that it will impair lung expansion.

CHEST DRAINS

Simple wound drains are adequate to remove blood from the affected site after most forms of surgery, but if the operation has interfered with pleural pressures, underwater-seal drains are usually required. The airtight system becomes an extension of the patient's pleura and allows air and blood to escape from the pleural space while preventing their re-entry. It restores normal negative pleural pressure and allows the lung to re-expand. Chest drains are also used after heart surgery, although some cardiac surgeons find that simple wound drains are sufficient.

An alternative to the underwater seal system is the flutter valve (Waller, 1999) which allows release of air on expiration but collapses shut by negative pressure on inspiration. They are cheap, safe, effective unless suction is required, and allow earlier mobility (Graham *et al.*, 1992).

Procedure

All tissues down to the pleura are infiltrated with local anaesthetic and, after this has taken effect, the drains are inserted above the rib where there are fewest vessels and nerves. Following heart surgery, one drain is usually placed inside the pericardium to prevent cardiac tamponade, and one outside the pericardium to drain blood from the mediastinum. After lung, pleural or oesophageal surgery, two drains are placed in the pleura, traditionally one in the apex to remove air and the other in the base to remove blood, but both air and blood will find the drain, especially when suction is applied. Pleural drains may also be required if the pleura has been cut after heart, kidney or upper abdominal surgery. Following pneumonectomy, a single drain may be left in the vacated space. For a pneumothorax, a single chest drain in the pleura restores negative pressure and allows reinflation of the lung.

Mechanism

One bottle may be adequate for minimal drainage, or more commonly, two or three chambers are used (Figure 10.13). The underwater seal, comprising the distal end of a tube

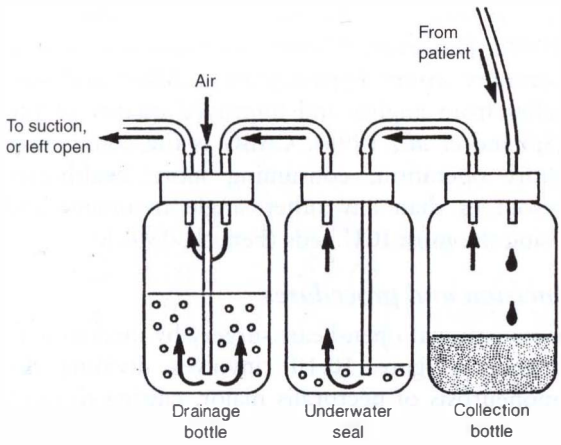


Figure 10.13 Classic 3-bottle drainage system.

submerged in 2 cm of sterile water, acts as a one-way valve. Fluid drains by gravity, and air is expelled through the water into the atmosphere. Fluid cannot return so long as the system is below the level of the patient's chest. Air cannot return because the water acts as a seal while offering minimal resistance to drainage. The length of tube below the water in the underwater seal bottle determines the pressure required to expel the unwanted contents from the chest, so is kept short without breaking the underwater seal.

Drainage depends on gravity or suction. Free drainage occurs when the exit tube is open to the atmosphere. This allows the water level in the underwater seal tube of a pleural drain to swing, reflecting the change in pleural pressure with breathing. If the negative pressure of the patient's breathing is inadequate to reinflate the lung, e.g. with excess fluid drainage or air leak, suction is applied at pressures of 10–20 cmH_2O.

Management

Patients with a chest drain need advice on activity, posture correction and shoulder mobility. Some need respiratory care.

Chest drain clamping was rife in the past on the assumption that dire consequences would follow disconnection. In practice, disconnection of the drain with entry of a small volume of air into the chest is trivial and reversible. Clamping is best avoided except in an emergency or when lifting the bottle above the patient when fluid could be siphoned into the chest. The drains should be clamped close to their exit from the chest.

The principles of safe handling of chest drains are the following:

- Before treatment, the location of the clamps should be checked so that they can be found in case of need.
- Junctions in the tubing should not be taped, otherwise a disconnection might be missed.
- If the bottle needs to be lifted above the level of the patient's chest when the patient turns, the tubing should be clamped. Clamping must be avoided if there is an air leak.
- Drainage is assisted by deep breathing, coughing or mobility, but care should be taken to avoid disconnection.
- When handling patients, the tubing should be held in alignment with the patient's chest to minimize discomfort.
- The bottles and tubing should be kept exposed throughout, to avoid accidental knocking or kinking.
- The system should be observed before and after treatment to check for any change in drainage, air leak or swing in the water level. Extra drainage is expected after treatment, but excessive loss suggests haemorrhage and should be reported.

An air leak is present if air is bubbling through the water, this air having passed through the visceral pleura from the lung at each breath. The hole should seal in time but positive pressure techniques would hinder this process and are to be avoided unless essential. If there is any change in air leak after treatment, this should be reported. Clamping is dangerous with an air leak and might precipitate a tension pneumothorax. If there is no pressure swing, this means that:

- the tube is kinked or blocked (sudden)
- the lung has successfully re-expanded,

thereby occluding the pleural end of the tube (gradual)
- the system contains a collection bottle, which obliterates the swing
- the system is on suction, which overrides the swing.

The procedure of 'milking' chest drains to dislodge clots and maintain patency can cause pulmonary trauma. Gentle squeezing is preferred (Kam *et al.*, 1993).

Chest drains are removed by nursing staff after the lung has fully expanded and drainage is complete. The distress of the procedure has been described by nearly half the patients in one survey as the worst memory of their hospital stay (Carson, 1994). This can be avoided by topical anaesthetic cream applied to the site three hours before removal (Valenzuela and Rosen, 1999), or Entonox. The patient is asked to take and hold a deep breath during removal to prevent air being drawn into the chest.

Problems

If any junction in the system becomes disconnected, the ends must be cleaned and reconnected, the patient asked to cough a few times to force out any air that has been sucked into the chest, and the incident reported.

If the tubing becomes disconnected from the patient, the following steps should be taken:

1. Ask the patient to exhale and, at the same time, press gauze, sheet or a hand against the wound at end-exhalation, speed taking precedence over sterility.
2. Ask the patient to breathe normally.
3. Summon assistance but stay with the patient and maintain pressure on the wound.
4. Observe breathing rate and chest symmetry.
5. If the patient is distressed, give reassurance and oxygen.

HEART SURGERY

For many patients, heart surgery carries a particular fear because of the significance attributed to the heart as the source of life (Maguire, 1998). However, 97–98% of them will survive coronary artery bypass graft (CABG) and find relief from angina and improved quality of life (Sjöland *et al.*, 1996). CABG is the commonest heart operation, consuming more health-care resources than any other single treatment and using the most ICU beds (Feneck, 1996).

Incision and procedures

Conventional open-heart surgery by median sternotomy (Figure 10.10) involves dividing the aponeurosis of pectoralis major, cutting through and retracting the sternum, then instigating cardiopulmonary bypass to allow surgery on a quiescent heart in a bloodless field. Cardiopulmonary bypass, or extracorporeal circulation, involves the heart being stopped, the aorta cross-clamped to clear the coronary arteries of blood, then the circulating blood being removed from the right atrium, filtered and oxygenated outside the body, and pumped back into the ascending aorta. Neither heart nor lungs are functioning during this period and the lungs are partially or totally collapsed. Some surgeons fill the pericardial sac with chilled saline to reduce tissue oxygen demand, protect the brain and other vital organs and reduce the flow required for bypass. This is no longer considered essential but moderate cooling to 30° may provide a safety margin.

Less invasive procedures use a 'beating heart' technique that avoids cardiopulmonary bypass and cross-clamping (Oz *et al.*, 1997).

Patients are mechanically ventilated until rewarmed and haemodynamically stable, then extubated and transferred to a cardiothoracic ward.

For CABG, the saphenous veins and/or internal mammary artery (IMA) are used to replace diseased coronary arteries (Figure 10.14). Patency rates for IMA grafts are 20 years compared to an average 8 years for saphenous vein grafts (Scarlett, 1998). However, the IMA is harvested from the chest wall, punctures the pleura, causes greater shoulder and chest wall pain (El-Ansary *et al.*, 2000) and greater impair-

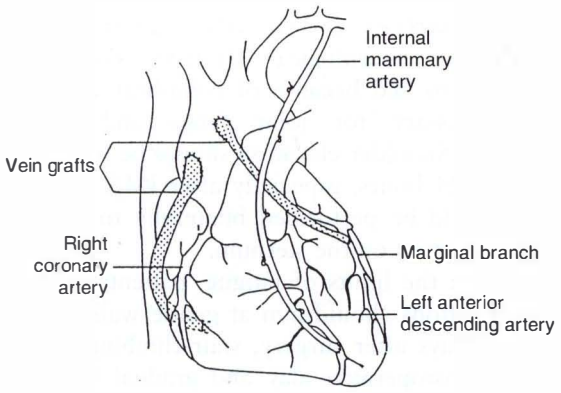


Figure 10.14 Grafts to the heart, including internal mammary artery. (From Adam, S. K. and Osborne, S. (1997) *Critical Care Nursing*, Oxford Medical, Oxford)

ment of lung function than the saphenous veins (Rolla, 1994).

Angioplasty is invasive but non-surgical revascularization. A balloon-tipped catheter is passed through the femoral artery and threaded up into the blocked coronary artery, where the balloon is intermittently inflated to clear the lumen. After several hours of lying flat to prevent bleeding, and then a period of observation, patients are discharged with minimal activity restrictions. Re-stenosis is commoner with angioplasty than CABG, but complications are fewer.

Complications specific to heart surgery

- Postoperative cardiovascular instability, although minimized by control of pain, fluids and oxygenation (Sonksen, 1998), may restrict turning or other forms of physiotherapy.
- Lower lobe atelectasis, mainly on the left, occurs in 30–88% of patients (Jindani, 1993), due to either compression of the left lower lobe, or trauma or cold injury to the phrenic nerve. This is so common as to be hardly considered a complication unless it progresses, and indeed many patients leave hospital with radiological changes not yet resolved.
- Some phrenic nerve damage is common. Regeneration occurs over a number of months. Severe damage may lead to diaphragmatic paralysis, sometimes bilateral and occasionally persistent (Katz *et al.*, 1998).
- Haemorrhage is particularly dangerous if blood is trapped in the pericardium, causing tamponade (p. 384).
- Pulmonary oedema or pleural effusion may be caused by aggressive fluid replacement or the effect of cardiopulmonary bypass in releasing vasoactive substances and altering capillary permeability (Wehberg *et al.*, 1996). Pleural effusion is usually left-sided and not significant.
- Disorientation is due to impaired cerebral perfusion during bypass, the alien environment in which the patient wakes up, hypoxia and sleep deprivation. It is especially common in elderly people.
- Retraction of the sternum and ribs may cause diaphragm impairment (Dickey, 1989), musculoskeletal pain (Stiller *et al.*, 1997), or first rib fracture (Wiener, 1992).
- Hypotension may impair perfusion to vital organs, as indicated by acute renal failure. Neurological defects range from a 25–30% incidence of cognitive defect at 1 year to a 2–3% incidence of stroke (Taylor, 1998). Impaired gut perfusion is reported to occur in 60% of patients and is a better predictor of complications than BP (Welsby and Mythen, 1997). Cardiac output is supported as necessary with inotropic drugs or mechanical devices (Chapter 12).
- Cardiopulmonary bypass contributes to neurological defects, and exposure of blood to any surface other than vascular endothelium may upset the clotting cascade or generate a systemic inflammatory response (Dodson *et al.*, 1997).
- Pain increases cardiac workload by sympathetic drive, and can lead to respiratory failure by breathing inhibition (Nelson *et al.*, 1998).
- Sternal dehiscense may occur 2–3 weeks postoperatively. It is usually associated with

infection and is suspected if the X-ray shows broken sutures or a gradually widening lucent line at the sternotomy site.
- In the ensuing years, atherosclerosis may progress in the grafted vessels and re-operation may be necessary, which carries a higher mortality than the first operation.

Physiotherapy

After CABG, blood pressure should be observed before, during and after treatment because hypertension increases cardiac work and can cause bleeding. Diastolic pressure is more significant than systolic pressure because coronary artery perfusion is highest during diastole. The operation notes may indicate the limits within which BP should be maintained.

For a stable patient on IPPV, manual hyperinflation is acceptable in the immediate postoperative period (Patman *et al.*, 1998) so long as it is indicated.

If hypoxaemia persists after extubation, CPAP can be helpful if comfortable, and may prevent the need for re-intubation (Romand and Donald, 1995). Until the patient can mobilize, regular positioning on alternate sides reduces postoperative complications (Tidwell, 1990), so long as this can be achieved with comfort and safety. Right-side-lying may be associated with better gas exchange than left-side-lying (Banasik and Emerson, 1996). Some surgeons request that the patient not be turned immediately after IMA graft.

A proportion of patients will appear euphoric on the first day, possibly reflecting delight at their survival, but then sink into depression for some days afterwards. When identified, these patients should be encouraged to take things gently on the first day to avoid debilitating mood swings which interfere with rehabilitation. The relationship between pain and anxiety is strongest on day 2 (Nelson *et al.*, 1998).

If breathing exercises or incentive spirometry are necessary for respiratory complications, manual support of the wound on inspiration may improve comfort and allow greater excursion.

Bed exercises provide the opportunity to check for neurological damage. For those confined to bed because of complications, they are necessary for joint range and muscle strength. Shoulder elevation should be limited to 90° for 24 hours, especially after IMA grafting, and should be performed bilaterally to avoid a shearing stress on the sternum.

Within the limits of fatigue, patients without complications should aim at gentle walking one or two days after surgery, stair-climbing on the fourth postoperative day and gradual increases thereafter. Mobilization without supplementary oxygen begins when the patient's S_aO_2 is > 92% while breathing oxygen at less than 6 L/min at rest.

Contraindications to mobilization are:

- ↑ or ↓ BP
- complete heart block reliant on external cardiac pacing
- atrial fibrillation with compromise of cardiovascular stability
- sinus tachycardia above 120 bpm
- heart failure requiring inotropic drugs
- IV vasodilator drugs
- Swan–Ganz catheter
- new myocardial infarct or symptomatic angina
- neurological event.

Contraindications to stair-climbing are the above plus heart rate at rest over 100 bpm or HR prior to stair-climbing over 110 bpm (Cockram *et al.*, 1999). After less invasive cardiac procedures, patients can often sit out 4 hours after surgery and walk later the same day.

If the sternum is heard or felt to click on movement, a cough belt (Figure 10.15) or towel is needed to stabilize the chest wall. One handle of the cough belt is passed through the other and pulled on coughing. Other patients may have continuing chest wall pain and benefit from joint mobilizations (Dickey, 1989).

Patients are usually ready for discharge within a week. It is useful to check the breathing pattern and posture to make sure that there are no lingering signs of tension that could become a

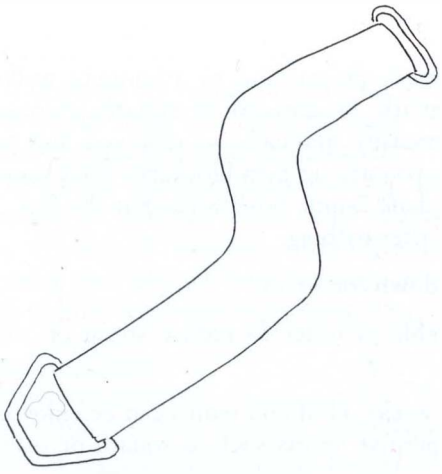


Figure 10.15 The 'Cough lok'.

habit. Patients need to understand the distinction between incisional pain and anginal pain, receive advice on rest/exercise balance, and be given written information, an example of which is provided in Box 10.1 for patients after a full-incision operation. They require a comprehensive exercise programme such as that described by Ungeman-deMent *et al.* (1986), and/or assessment at an outpatient physiotherapy clinic to check for musculoskeletal problems (El-Ansary *et al.*, 2000) and/or cardiac rehabilitation.

CABG is effective in reducing angina but functional impairment often continues after surgery (Allen, 1990), especially without rehabilitation, and atherosclerosis continues. Patient and family are well advised to cultivate a lifestyle that retards the disease process in the grafted vessels.

TRANSPLANTATION

> ***'Each new day is welcomed with open arms come sun, rain or snow. Gone are the excuses for putting off activities until the weather is better or the time more opportune.'***
>
> Marsh (transplant patient), 1986

Receiving a transplanted heart, lung or both sometimes causes profound change in a patient's attitude to life. Postoperative feelings of resurrection are not unusual and the patient's mood may swing between depression and euphoria (Ellis, 1995). For those who survive the waiting list, operation and complications, each day is precious and life is usually sweet.

The indication for transplantation is end-stage organ disease. Patients must be well enough to cope with the operation and aftermath. Examples are:

- cardiomyopathy for heart transplant
- cystic fibrosis (CF) for double lung or heart–lung transplant
- non-infective lung disorders such as pulmonary fibrosis and emphysema for single lung transplant (which makes efficient use of the donor pool).

The operations are no longer technically awesome, and now that immunosuppressive therapy is better able to prevent organ rejection, the main limiting factor is lack of donor organs. Lobar transplants are easing this problem in relation to lungs, and xenotransplantation from other species is currently being examined by ethicists.

Recipients are matched with donors for factors such as blood type and organ size. Strict criteria are applied to donor and recipient (Maurer *et al.*, 1998). Recipients must be free of HIV infection, recent malignancy, tobacco and other addictions, be optimally nourished and on minimal steroids. Delays to heart transplant can sometimes be bridged by a left ventricular assist device (Koul *et al.*, 1998). One-year survival is 85–90% for heart, 75–85% for heart–lung and 60–90% for lung transplantation (Oh, 1997, p. 818).

Procedures

Cardiopulmonary bypass is used for heart and occasionally double-lung transplant. For single lung transplant, a thoracotomy incision and bronchial anastomosis are used. For bilateral lung transplants, a massive trans-sternal bilateral thoracotomy (clamshell incision) allows sequential single-lung procedures, creating two separate

Box 10.1 Home advice following heart surgery, to be modified for individual needs

1. On returning home, take regular walks around the house, progressing to 10-minute walks outside within a week. In the second week, do light housework. Progress to 30 minutes exercise daily, possibly split into two walks a day. Increase the intensity gradually so that you aim to produce a rise in pulse rate to over 100 per minute (unless you are on beta-blockers). Find some enjoyable exercise that you can maintain indefinitely, e.g. a daily 2-mile brisk walk. For the first 2 months, delay exercise for 1 hour after meals or 30 minutes after bathing.

2. For the next 6 weeks, allow yourself an hour a day to lie down for a nap.

3. Women are advised to wear a bra as soon as comfortable in order to reduce strain on the wound.

4. Swimming, fishing and cycling can be taken up after 6–8 weeks. Golf and tennis can be enjoyed so long as unilateral arm movements are not forced. Competitive sports such as squash or team games are not recommended for at least 3 months, and then only with the doctor's advice.

5. Sexual activity can be started after about 2 weeks or when you can manage everyday activities such as climbing stairs. It is advisable to remain in the dependent position in the early stages to avoid strain on the incision. Your partner may be more anxious than you about resuming sex, and may need reassurance. Some drugs such as beta-blockers can affect sexual performance.

6. Avoid driving for at least 6 weeks, or 1 week following angioplasty. Do not drive if it brings on angina. In the UK it is not necessary to notify the DVLA, but advise your insurance company.

7. Sedentary work can be resumed after 4 weeks.

8. Do not lift, push or pull anything heavy, especially with breath-holding, for 6 weeks. This includes mowing, digging and vacuum-cleaning.

9. Expect a degree of breathlessness, tiredness, poor concentration, memory loss and aches and pains across the shoulders and chest. These are normal and should improve over 6 weeks. Take painkillers for as long as you need them. Breathlessness that hinders speaking should be reported to the doctor. Some arm movements cause a clicking feeling in the breast bone. If this continues after 3 months, let the doctor know. Depression or anxiety may come and go for several weeks.

10. It is safe to fly after 10 days, and airports will supply a wheelchair if necessary. However you are unlikely to get the best out of a holiday for at least 6 weeks.

11. The wires in your breast bone will not rust or set off security alarms.

12. Make a list of questions to ask the doctor at your follow-up appointment.

13. No smoking.

14. Keep happy!

bronchial anastomoses and preserving the coronary-to-bronchial circulation. A sternotomy is used for other procedures.

A heart–lung transplant (HLT) provides heart and lungs *en bloc* and retains the collateral circulation. HLT may incorporate a domino procedure in which the recipient's healthy heart is used as a donor for a second recipient.

Complications

All transplants are complicated by the *side effects of life-long drugs* to inhibit organ rejection, including increased risk of malignancy. Cyclosporin causes immunosuppression by inhibiting lymphocytes and can cause renal damage. The side effects of steroids include opportunist infection and altered body image due to extra body hair or cushinoid appearance.

Heart transplant

Acute heart *rejection* is suspected if there are temperature or ECG changes, and confirmed by biopsy. Chronic heart rejection accelerates atherosclerosis of the transplanted arteries and is identified by annual angiography. The only treatment for chronic rejection is re-transplantation.

Lung transplant

Transplanting the lung has proved difficult because it is the only organ in contact with the atmosphere and has evolved a strong protective immunity to anything foreign. Many complications may emerge, as described below.

Infection is the main cause of death in the early stages. Contributing factors are immunosuppression, lymphatic interruption and hilar stripping during surgery. Denervation of the lung impairs mucociliary transport and the patient's awareness of the presence of secretions, an awareness that people with CF have developed to a fine art. Patients are reminded to expect some secretions in the immediate postoperative period because it takes a few days for the debris to clear. Secretions also continue to be produced from the native airway of CF patients above the anastomosis. A further hindrance to secretion clearance in the short term is postoperative oedema around the anastomosis. Denervation is often permanent but the bronchial arteries and lymphatic system are thought to regenerate in some weeks (Oh, 1997, p. 816). An advantage of denervation is that reduced neural drive may contribute to decreased breathlessness (Brath *et al.*, 1997).

Pulmonary oedema can be caused by loss of lymphatic drainage or the ischaemic insult of surgery followed by reactive reperfusion. 'Reperfusion pulmonary oedema' peaks 8–12 hours postoperatively, causing hypoxia and reduced lung compliance. Treatment with fluid restriction and diuretics may thicken secretions, dry the mouth and inhibit expectoration.

Hypercapnia may develop in people with preoperative CO_2 retention because the new normal oxygenation suppresses their hypoxic respiratory drive. This normalizes within a week.

Acute or chronic lung *rejection* can occur from a few days to several years postoperatively. FVC and FEV_1 should rise to a plateau some months after surgery and then remain stable, but a subsequent reduction of 10–15% is a warning of possible rejection. Suspicions are confirmed if there is fever, breathlessness, hypoxaemia and fine crackles on auscultation. X-ray signs are similar for both rejection and opportunistic infection, and gentle bronchoscopy is needed to distinguish the two. Patients are given a diary in which to record spirometry readings, temperature, weight and medication. Treatment of rejection is by increased steroids. Confirmation of rejection indicates that physiotherapy should be modified as follows:

- suction must be minimal because it may damage the anastomosis and exacerbate oedema from surgery and erythema from rejection
- exercise training is temporarily ceased or modified.

Obliterative bronchiolitis is a devastating complication following repeated episodes of rejection. It occurs in 10–50% of recipients at around 6–18 months after surgery and has a mortality of 30–50% (Nathan, 1995). It creates a combined obstructive and restrictive defect, the small airways becoming obstructed by inflammation and then obliterated by granulation tissue, which then fibroses. It may be complicated by extending into alveoli and developing into pneumonia (see 'BOOP' in the Glossary). Monitoring bronchodilator response

in the small airways may identify the process so that augmented immunosuppression can be started (Rajagopalan *et al.*, 1996). PEP or CPAP give symptomatic relief. Re-transplantation may be required but carries a high mortality.

Stenosis may occur at the anastomosis site. Treatment is by balloon dilation or insertion of a silastic stent, but the condition is usually associated with obliterative bronchiolitis.

Physiotherapy

Preoperative physiotherapy takes place when the patient attends the surgeon's assessment clinic, because there is little time once a donor has been found. An exercise programme should be adhered to for those who are not deteriorating rapidly. Noninvasive ventilation may be required while awaiting a donor organ.

Postoperative respiratory care is similar to other forms of chest surgery, with extra attention to prevention of infection, plus other considerations described below.

Following lung transplant or HLT, endobronchial suction if needed should be undertaken without the catheter reaching the anastomosis, which in ventilated patients is just below the end of the endotracheal tube. Continuous humidification and sometimes modified postural drainage may be needed because of impaired mucociliary clearance. Long-term chest clearance is not needed, and some innervation may occur over time. Manual hyperinflation and IPPB should be used with caution because of the risk of pneumothorax or bronchial dehiscence.

Many patients are debilitated and need extensive rehabilitation. In the early stages, the following is a guide to a progressive exercise regime:

- day 1–2: sitting out in a chair
- day 2: upper limb exercises, static pedals
- day 2–3: walk round room
- day 4: walk outside room
- day 5: exercise bike, gym, stairs.

After discharge, patients pursue an exercise programme at home or in the gym, with the following precautions:

- Transplanted hearts can show a training effect but are denervated so that the transmission of angina is impaired, the pulse is not a reliable monitoring tool and there is a delayed heart rate response to exercise, requiring ample warm-up and cool-down periods. After 6–12 months of exercise, responses may become near-normal, although the intensity and duration of exercise is limited.
- Steroids may cause osteoporosis, myopathy or delayed healing.

Attention is focused on the recipient, and the donor and donor's relatives are vulnerable at this time. Respiratory physiotherapy may be requested to maintain oxygenation to the organ to be transplanted. A brain-dead donor must be cared for as any moribund patient and not talked over. Conversation with relatives must avoid terminology such as 'harvesting' the organs, or comments on the importance of maintaining vital signs.

REPAIR OF COARCTATION OF THE AORTA

Stricture of the aorta raises BP due to impaired renal perfusion. Patients may be symptomless, the condition often being picked up on routine X-ray, but surgery is advisable before hypertension wreaks damage in later life. Repair is by resection of the narrowed segment and anastomosis or insertion of a graft.

The following precautions are needed postoperatively to avoid a sudden rise in BP that might strain the anastomosis:

- The head-down tip should be avoided. Some surgeons prefer the patient not to lie flat.
- Mobilization should be slow and fatigue avoided. Extra care is needed when patients are beginning to feel well enough to exert themselves.
- Vigorous exercise should be discouraged for several months.

OESOPHAGECTOMY

Oesophageal cancer leaves only 10–20% of patients alive 1 year from diagnosis and 5–10% alive after 5 years (Mills and Sullivan, 2000). It is usually diagnosed too late for successful resection, but surgery can relieve the distressing symptom of dysphagia and sometimes effect a cure. Oesophagectomy is a harrowing operation, with access by thoracolaparotomy, thoracotomy and laparotomy/laparoscopy, or thoracotomy with neck incision, depending on the location of the tumour.

Complications are reduced with early extubation, adequate pain control and meticulous preoperative preparation (Caldwell, 1993), but the following may occur:

- significant atelectasis as the stomach is pulled up into the chest to be anastomosed to the oesophageal stump
- leakage or dehiscence at the anastomosis
- empyema (Figure 10.16) or abscess (Klein, 1999)
- pleural effusion
- weakness after protracted preoperative malnourishment.

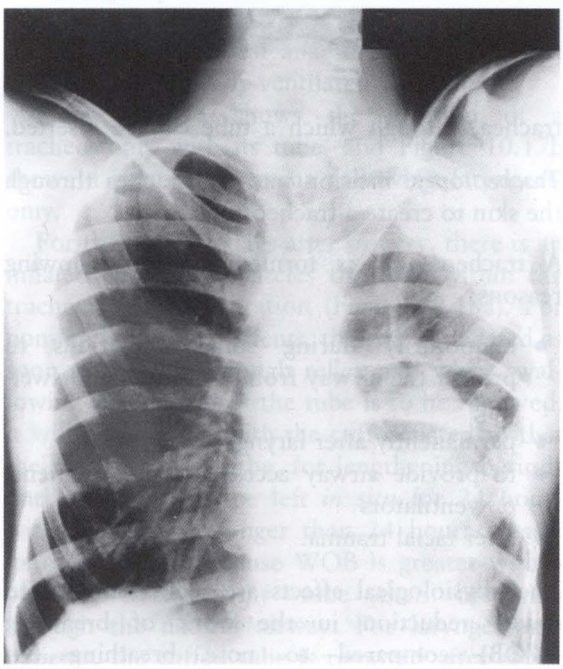


Figure 10.16 X-ray showing an empyema in the left upper zone that developed in a 17-year-old youth after oesophagectomy. In the left chest can be seen the cut rib of the thoracotomy and the colon transplant that replaced the oesophagus, partially filled with fluid.

Postoperative precautions are:

- avoid the head-down tilt in case reflux of gastric contents damages the anastomosis (some surgeons prefer patients to maintain head elevation)
- avoid neck movements that might stretch the anastomosis
- with a high resection, avoid naso- or oropharyngeal suction because the catheter might accidentally enter the oesophagus.

Sputum retention is common and a request for early minitracheostomy is advisable in selected patients. Discharge advice is detailed by Savage (1992).

BREAST SURGERY

Complications of mastectomy include lymphoedema, joint stiffness, muscle weakness and shoulder pain, especially after axillary node dissection (Kelley and Jull, 1998). Patients require advice on upper limb movement and posture correction, but should not elevate the shoulder joint beyond 90° until the drains have been removed. An example of an exercise sheet is given in Box 10.2. After lumpectomy, patients may have no significant complications but will need a reminder on arm movement.

HEAD AND NECK SURGERY

Swallowing dysfunction and aspiration are common complications of head and neck surgery. Head and neck cancers are among the most emotionally traumatic because of disfigurement, loss of natural speech, impaired taste, mucus discharge, complications of radiotherapy, limited ability to express feelings, and difficulties with swallowing, breathing and nose-blowing (Monga *et al.*, 1997). Frustration, social with-

Box 10.2 Examples of exercises after breast surgery, to be tailored to the individual

For the first day or two after the operation you may have one or two drains to allow fluid to escape into a sealed bottle or bag.

Days 1–3:
Shrug shoulders up and down.
Roll shoulders back and down.
Walk round the ward taking your drains with you, allowing your arm to swing gently as you walk.
Avoid standing with shoulders hunched.
Use both hands as normal.

Days 2–3 onwards:
Do the following exercises, trying to go a little further each time and ensuring a gentle stretch but no pain.

- Clasp hands together, lift hands up, first with elbows bent, then gradually straighten elbows as it gets easier.
- Clasp hands behind neck and move your elbows apart.
- Clasp hands behind back with elbows bent, lift them up towards bra strap level.
- Clasp hands behind back with elbows straight, stretch arms backwards.

Continue these exercises three times each, twice a day, for about 3 weeks or until you have full movement. If movement is not regained in 6 weeks, ask your doctor to refer you to a physiotherapist.

drawal, depression and problems with close relationships are common, and 50% of patients may develop psychiatric morbidity (McQuellon and Hurt, 1997).

Laryngeal cancer

Cancer of the larynx is highly curable in its early stages, and laryngectomy has been practised for 100 years. However, there has been little improvement in cure rate (Levine, 1997), and the main aim is early diagnosis, or preferably prevention. Smoking is the main risk factor (Koufman and Burke, 1997). Partial or total laryngectomy is required if radiation treatment fails. Partial or radical neck dissection is performed for malignant invasion of other tissues. A more extensive commando procedure resects part of the mandible, tongue, neck structures or floor of the mouth.

Tracheostomy

Tracheostomy: artificial opening into the trachea, through which a tube can be inserted.

Tracheotomy: incision into the trachea through the skin to create a tracheostomy.

A tracheostomy is formed for the following reasons:

- temporarily during some operations to protect the airway from aspiration and swelling
- permanently after laryngectomy
- to provide airway access for some patients on ventilators
- after facial trauma.

The physiological effects are reduction in dead space, reduction in the work of breathing (WOB) compared to nose breathing but increased WOB compared to mouth breathing (Prichard, 1994).

Percutaneous tracheotomy is a dilational technique that causes less trauma and scarring than a full surgical tracheotomy. It can be

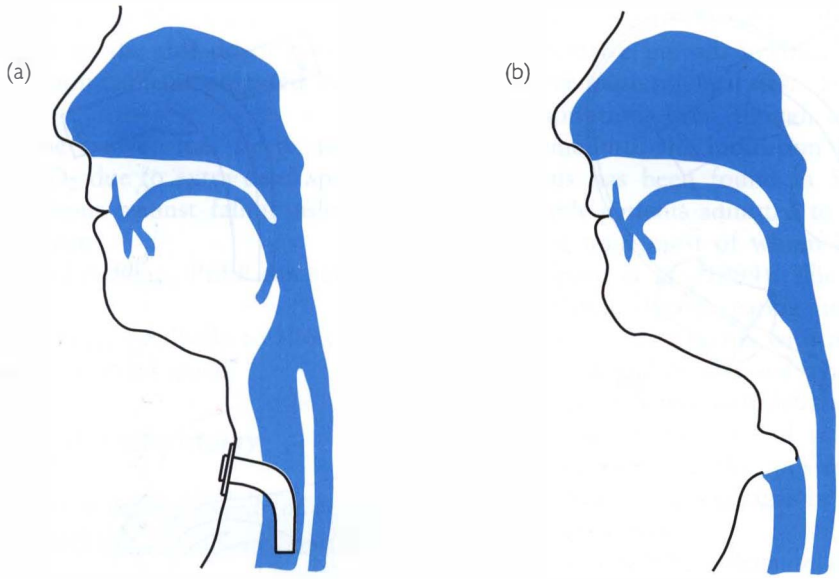


Figure 10.17 (**a**) Tracheostomy tube *in situ*. (**b**) Tracheostomy and laryngectomy.

performed under local anaesthesia and is often used for mechanically ventilated patients.

Figure 10.17a shows the location of a tracheostomy with its tube, and Figure 10.17b shows a permanent stoma following laryngectomy.

For the first few days after surgery, there is an inflated cuff that encircles the tube within the trachea to limit aspiration (Figure 10.18a). For non-laryngectomy patients, the cuff is deflated as soon as there is a cough reflex and when swallowing is adequate. If the tube is to be removed, it will be plugged, with the cuff deflated so that the patient can breathe, for lengthening periods until the plug can be left *in situ* for 24 hours without distress. Longer than 24 hours creates its own distress because WOB is greater with a plugged tracheostomy tube than breathing through the natural airway. For laryngectomy patients, the cuffed tube is removed after about 48 hours when haemorrhage is no longer a risk, and replaced with an uncuffed tube (Figure 10.18b).

Uncuffed tubes are used for patients requiring permanent tracheostomies. They incorporate an inner cannula (Burns *et al.*, 1998) whose function is to prevent trauma from repeated tube changes and as a safety factor in case of blockage. It is left *in situ* for suction, but may need to be removed and cleaned beforehand, as follows:

- Support the neck plate with one gloved hand.
- Unscrew, unlock or unclip the inner tube with the other gloved hand, then remove outwards and downwards.
- Clean with a brush and sterile water, dry inside and out with gauze.
- Put it back in, screw, lock or clip into place.

The tapes are best secured with Velcro and should be loose enough to fit one finger between the tape and neck.

Variations are the following:

- A **silver tube** (Figure 10.18c) is uncuffed and helps maintain the stoma.
- For people without a laryngectomy but requiring a long-term tracheostomy, speech is possible with a **fenestrated tube** (Figure 10.18d). This has inner and outer cannulae

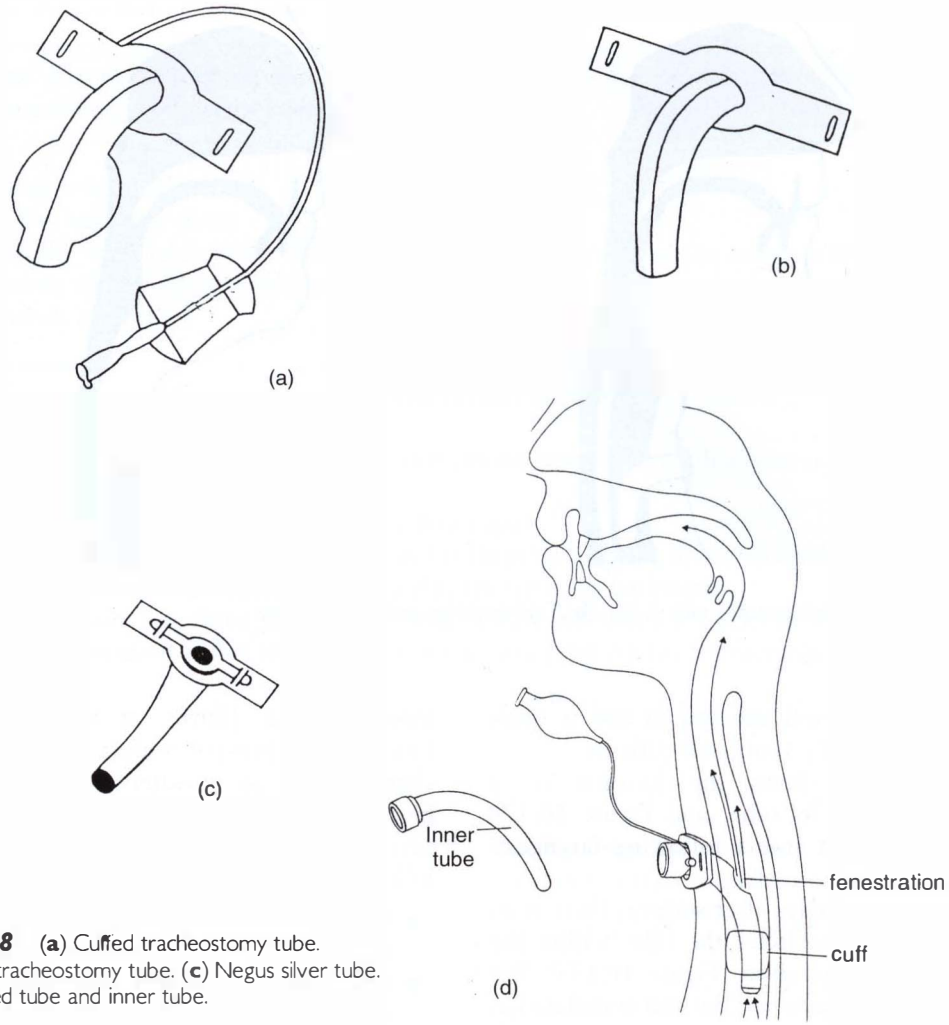


Figure 10.18 (**a**) Cuffed tracheostomy tube. (**b**) Uncuffed tracheostomy tube. (**c**) Negus silver tube. (**d**) Fenestrated tube and inner tube.

with matching windows (fenestrations) on their posterior curves. With the cuff deflated and stoma occluded by a plug or gloved finger on expiration, the patient can speak by breathing out through the windows, around the tube and up through the larynx. An unfenestrated inner cannula is used for suction, eating and drinking, but a new model is available that can be left in place for suction because it has many tiny windows (Mallingcrodt, Appendix C). The patient must sit upright when eating and for 20 minutes afterwards. Fenestrated tubes are not to be used with mechanical ventilation.

- A **speaking valve** such as the Passy–Muir allows speech, in patients who have not had a laryngectomy, without occluding the stoma with a finger. The valve opens on inspiration and closes on expiration to force air through the vocal cords (Manzano, 1993), and can be free-standing or in-line with a ventilator circuit. Improved sense of smell and appetite are added advantages.

Practical points when using a speaking valve are:

- Before connecting the valve, suction the airway, then let the cuff down slowly, with another suction catheter prepared in case of need.
- Remove the valve for sleep to avoid impaired S_aO_2 due to extra dead space, and take precautions against falling asleep with the valve *in situ*
- For ventilated patients, PEEP can sometimes be reduced.
- Increase tolerance gradually to allow accommodation to the dead space.

Complications of tracheostomy

> *'We can never make the sounds of crying, shouting or laughter.'*
>
> Ulbricht, 1986

Unavoidable complications of a tracheostomy tube are the following:

- impaired cough, because there is no closed glottis behind which air can build up
- impaired swallow because of reduced muscle co-ordination, upset pressure gradients and anchoring of the larynx, especially with an inflated cuff
- damage to the trachea, increased by the pull of ventilator tubing or mishandling
- loss of the modest natural PEEP normally maintained by the larynx (p. 357).

Complications that may occur are the following:

- Obstruction due to a blood clot, or if poorly managed, due to thick secretions.
- Haemorrhage. This may be obvious, or indicated by pulsation of the tracheostomy tube synchronously with the pulse. If suspected, the airway should be suctioned and cuff inflated. This will temporarily limit aspiration of blood into the lungs, until medical attention arrives. The head-down position also inhibits aspiration.
- Surgical emphysema (Douglas and Flabouris, 1999).
- Displacement, especially if there is excessive movement of the tube in the immediate post-operative period, uncontrolled coughing or over-loose tracheal ties.
- Aspiration, even though aspiration itself is sometimes the indication for tracheostomy. This has been found in 50% of medically stable patients admitted to a chronic ventilation unit, most of whom had no symptoms (Elpern *et al.*, 1994). The cuff needs to be deflated during eating to facilitate swallowing, but patients with neurological disorders should be assessed by a speech-language therapist before cuff deflation.
- Dysphagia, nausea and vomiting if pressure is exerted on the posterior wall of the trachea and oesophagus by the wrong size or shape of tube.
- Infection, partly because the oropharynx is teeming with bacteria and partly because poor suction technique is widespread. Prolonged hospitalization or poor nutrition encourage infection.
- Weight loss due to dysphagia and reduced appetite. Food is less appetizing because of impaired taste and smell, which are never fully restored.
- Fistula formation, which is suspected if suctioned secretions contain food and drink.
- Erosion of the trachea due to excess movement of the tracheostomy tube, and late onset stricture due to granulation tissue as eroded areas heal.
- Stenosis, which may take months or years to develop. Signs are cough, retained secretions and increasing dyspnoea. Laser treatment, or resection and anastomosis, may be required.

Management

It takes about a week after tracheostomy for fascia and muscle to fuse and form a tract, during which time tracheal dilators are on hand in case the tube becomes dislodged. Two spare tracheostomy tubes must be available, one a size smaller in case a change is needed urgently. At the bedside there is also an obturator, a solid insert that, when inside the tracheostomy tube, assists its insertion and which is removed imme-

diately afterwards so that the patient can breathe. Tracheostomy tubes should normally only be changed by a specifically trained nurse, physiotherapist or doctor.

The greatest fear of tracheostomy patients during their early postoperative days is the inability to summon help, and a bell must always be within reach. For patients unable to call for help immediately, a bell is not sufficient and they should be in sight of the nurses' station, not in a side room.

Continuous hot water humidification is needed for the first 48 hours. If the tracheostomy is permanent, the airway acclimatizes to its new exposure to the environment. Patients will then need to maintain adequate fluid intake, and are supplied with sterile saline and a syringe to moisten the airway if necessary. An 'artificial nose' can be used as a heat-moisture exchanger (p. 190), or a bib over the stoma can filter out large particles. Humidification is restarted if infection occurs, and people with chronic lung disease may need intermittent humidification. Gloves should be worn for all contact with the tracheostomy area.

Liberal mouthwashes are required after head and neck surgery, even for patients with excess salivation. Suction of the mouth requires low pressures and avoidance of areas of anastomosis. If the sternomastoid muscle has been excised, the patient's head will need support postoperatively when s/he is moving around the bed. If the spinal accessory nerve has been transected, there might be shoulder pain and limited abduction. Connecting tubes must be supported during movement so that they do not drag on the wound. Staff need to allow time for lip-reading and deciphering written requests. Patients need questions that require only a yes or no answer. They do not need a raised voice.

For disfiguring surgery, a mirror should be given to patients only if someone is available to be with them for their first view. Visitors need preparation before their first visit. Women may have more difficulty adapting to their new self-image because of a low-pitched voice and more exposed cosmetic changes. Self-esteem often improves when patients are encouraged in activities ranging from cleaning their own inner tube to helping other patients. With a long-term tracheostomy, patient or carer will need to be taught to do their own suction (although many patients can huff out their secretions) and deal with a blocked tube (see next page).

Patients require postural correction and advice such as avoiding traction on the brachial plexus and supporting the neck plate when coughing or sneezing. An exercise regime begins with gentle range of movement exercises on day 1. Respiratory care is provided as required, and an incentive spirometer, attached with a connector, has been shown to improve lung function (Tan, 1995). Major head and neck surgery should be followed by a comprehensive exercise programme such as that described by Herring *et al.* (1987).

The speech-language therapist assists with swallowing and, after laryngectomy, voice restoration by methods such as the following.

- An electrolarynx held at the neck produces a tone that is shaped into mechanical-sounding speech as the user mouths the words.
- Oesophageal speech involves compressing air into the oesophagus and releasing it, causing a vibration. The technique is difficult to learn but creates a more normal sound than the electrolarynx.
- A tracheo-oesophageal puncture can be created and a valve inserted through which patients can be trained to generate oesophageal speech.

Isolated laryngeal transplants have been performed and provide hope for the future.

Decannulation of the tracheostomy tube is described on page 359. Discharge advice for patients with an ongoing tracheostomy includes:

- sterile suction
- protecting the stoma from water
- keeping the home dust-free
- avoiding people with chest infections
- winter flu vaccination

- instructions for carers on mouth-to-stoma resuscitation
- replacement or removal of a blocked tube in an emergency
- a contact number in case of problems
- for those with facial disfigurement, information on support groups (Appendix C).

Respiratory arrest with a tracheostomy

If a tracheostomied patient suffers respiratory arrest due to obstruction, the following steps should be taken:

1. Press the crash button or ask a colleague to call the crash team.
2. Suction the airway.
3. If there is an inner cannula, remove it. If this was obstructed, removal may be sufficient. Otherwise, continue as below.
4. Move the patient's head in case this relieves the obstruction.
5. Remove the tracheostomy tube as follows:
   - Deflate cuff
   - Cut securing tape
   - Slide out tube, using tracheal dilators to maintain the patency of stoma
6. Continue ventilation via tracheal dilators, either encouraging the patient to breathe spontaneously or blowing down the stoma.

Experienced physiotherapists can insert a new tracheostomy tube, or a nasopharyngeal tube can be used if necessary.

CASE STUDY: MR LS

Identify the problems of this 74-year-old man on his first day after left upper lobectomy for small cell carcinoma.

Subjective

Pain on coughing.
Bringing up thick green phlegm.
Unable to sleep.

Objective

Notes: *Pseudomonas* chest infection.
Charts: intermittent intramuscular analgesia, pyrexia, S_aO_2 94%.
Patient slumped in bed, on 40% dry oxygen.
Rapid shallow breathing pattern.
Auscultation: ↓ BS LLL, coarse crackles.
PA and lateral films show two fluid-filled cavities in left upper zone (Figures 10.19a and b) Scan at tracheal level identifies largest cavity (Figure 10.19c). Radiology report states that cavities may be abscesses, empyema or bronchopleural fistula.

Questions

1. Analysis?
2. Patient's problems?
3. Precaution?
4. Goals?
5. Plan?

BS = breath sounds; LLL = left lower lobe; PA = postero-anterior.

RESPONSE TO CASE STUDY

1. Analysis

Inadequate analgesia contributing to shallow breathing and probable LLL atelectasis.
LLL atelectasis suggested by ↓ BS in L lower zone and raised hemidiaphragm on left (too large a shift to be attributable to loss of the upper lobe).

2. Problems

Pain.
Poor sleep.
↓ lung volume LLL, contributing to poor gas exchange.
Retention of infected bronchial secretions, contributing to poor gas exchange.

3. Precaution

Avoid right-side-lying to prevent infected fluid spreading from abscess or bronchopleural fistula.

4. Goals

Increase lung volume and clear secretions.
Rehabilitate.

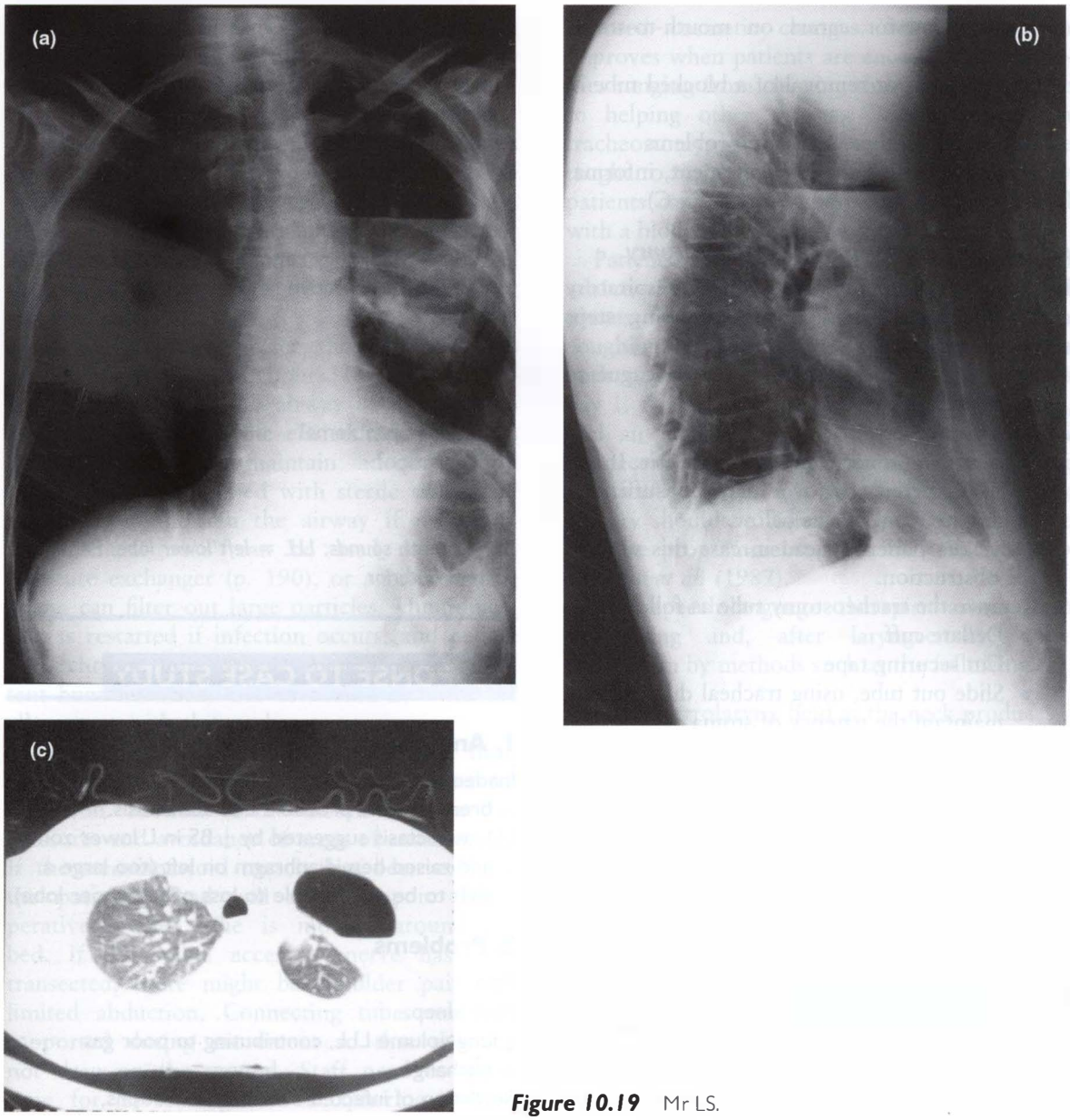


Figure 10.19 Mr LS.

5. Plan

- Liaise with team to clarify location of infected fluid.
- Obtain adequate analgesia.
- Identify cause of poor sleep, then remedy if able.
- Check that flow of oxygen meets patient's subjective requirements.
- Humidify oxygen.
- Controlled mobilization.
- Sitting in chair or upright in bed: deep breathing with end-inspiratory hold, then

incentive spirometry, then identify which is most effective.
- Sitting in chair or upright in bed: ACB/AD.
- If secretions do not clear, choose another method that is simpler and less tiring, e.g. gentle vibrations or flutter.
- If secretions still do not clear, request minitracheostomy.
- Ask patient, with reminders from nurses or patient's visitors if possible, to practise either deep breathing or incentive spirometry, then ACB/AD if able, preferably for a few minutes every waking hour.
- Visit patient little and often: remind him about exercises, check position/comfort/humidification, treat as required.
- Upper limb exercises and posture correction.
- Progress.

ACB/AD: active cycle of breathing and/or autogenic drainage

LITERATURE APPRAISAL

Can you criticize the following title before reading the rest of the article? 'Are incentive spirometry, intermittent positive pressure breathing and deep breathing exercises effective in the prevention of postoperative pulmonary complications?' (*Phys. Ther.* (1994), 74, 3–16)

RESPONSE TO LITERATURE APPRAISAL

1. The single word 'and' indicates that several modalities are being tested. This does not help us to identify which.
2. IS, IPPB and deep breathing are indicated for different levels of patient ability and cooperation, and are not interchangeable in this way.

RECOMMENDED READING

American Society of Anesthesiologists (2000) Practice advisory for the prevention of perioperative peripheral neuropathies. *Anesthesiology,* **92,** 1168–82.

Basse, L. (2000) A clinical pathway to accelerate recovery after colonic resection. *Ann.Surg,* **232,** 51–57.

Bartsch, M. H., (2000) Exercise capacity and extent of resection as predictors of surgical risk in lung cancer. *Eur.Resp.J.,* **15,** 828–832.

Burke D. T. (2000) Prevention of deep venous thrombosis: overview of available therapy options for rehabilitation patients. *Am.J.Phys.Med.Rehab.,* **79** (suppl.), S3–S8.

Chua,K. S. G., Reddy, S. K. and Lee, M. C. (1999) Pain and loss of function in head and neck cancer survivors. *J.Pain Symptom Man.,* 18, 193–202.

Cockram, J., Jenkins, S. K., and Clugston, R. (1999) Cardiovascular and respiratory responses to early ambulation and stair climbing following coronary artery surgery. *Physiother.Theory Pract.,* **15,** 3–15.

Eliachar, I. (2000) Unaided speech in long-term tube-free tracheostomy. *Laryngoscope,* **110,** 749–760.

Good, M. and Stanton, M. (1999) Relief of postoperative pain with jaw relaxation, music and their combination. *Pain,* **81,** 163–172.

Harden, B. (1997) Lung volume reduction surgery for emphysema. *Physiotherapy,* **83,** 136–140

Hogue, S. L., Reese, P. P. and Colopy, M. (2000) Assessing a tool to measure patient functional ability after outpatient surgery. *Anesth.Analg.,* **91,** 97–106.

Huerta-Torres, V. (1998) Preparing patients for early discharge after CABG. *Am.J.Nurs.,* 98, 5, 49–51.

Hyde, J. (2000) Reducing morbidity from chest drains. *Br.Med.J,* **314,** 914.

Moores, L. K. (2000) Smoking and postoperative pulmonary complications. *Clin.Chest* Med., 21, 139-146.

Orringer, M. K. (1999) The effects of tracheostomy tube placement on communication and swallowing. *Respir.Care,* **44,** 845–853

Pandit, S. K., Loberg, K. W. and Pandit, U. A. (2000) Toast and tea before elective surgery? *Anesth.Analg.,* **90,** 1348–1351.

Rosenberg, A. D. (2000) Reducing post-traumatic morbidity with pain management. *Curr.Op.Anaesthesiol.,* **13,** 181–184.

Schwaiblmair, M. (1999) Cardiopulmonary exercise testing before and after lung and heart-lung transplantation. *Chest,* **159,** 1277–1283.

Siafakas, N. M., Mitrouska, I. and Bouros, D. (1999) Surgery and the respiratory muscles. *Thorax,* **54,** 458–465

Stevensen, C. J. (1994) The psychophysiological

effects of aromatherapy massage following cardiac surgery. *Complem.Ther.Med.*, **2**, 27–35.

Street, D. (2000) A practical guide to giving entonox. *Nurs.Times*, **96**, 34, 47–48.

Thompson, M.N. and Bell, P.R.F. (2000) Arterial aneurysms. *Br.Med.J*, 320, 1193–1196.

Warner, D.O. (2000) Preventing postoperative pulmonary complications. *Anesthesiology*, **92**, 1467–1472.

Weissman, C. (2000) Pulmonary function after cardiac and thoracic surgery. *Curr.Op.Anaesthes.*, 13, 47-51

Wong, D. H. (1997) Chronic obstructive pulmonary disease and postoperative pulmonary complications. *Curr.Opin.Anaesth.*, 10, 254-258

Young, P. J. and Matta, B. F. (2000) Anaesthesia for organ donation in the brainstem dead – why bother? *Anaesthesia*, **55**, 105–106.

11 Physiotherapy for specific groups of people

Summary

Overview of cardiac rehabilitation

Coronary heart disease is the commonest cause of death in the UK (DoH, 1993). Hospitalization continues to shorten and rehabilitation is taking on a higher profile. It would be ideal for rehabilitation to be initiated before heart surgery, but it usually starts after the operation or after a myocardial infarction (MI), and increasingly for people with chronic heart failure.

Once arrhythmias, acute heart failure and ischaemia have been stabilized after surgery, rehabilitation begins with a balance of rest and gentle progressive exercise from sitting out, to walking, to stair-climbing. After discharge, a convalescent programme of steady but not progressive exercise is maintained. An outpatient rehabilitation programme involving physical reconditioning begins 3–6 weeks after surgery or angioplasty and 4–8 weeks after MI. The following benefits of cardiac rehabilitation have been demonstrated:

- ↑ work capacity
- ↑ myocardial perfusion
- ↓ angina
- ↓ fatigue, depression and anxiety
- ↓ in mortality by 20% (Finlayson, 1997)
- ↓ hospital re-admission rates by 38% (Huang *et al.*, 1990).
- ↓ risk of sudden death with vigorous exercise 50-fold (Bärtsch, 1999).

The exercise component does not affect the atherosclerotic process but can enlarge coronary arteries to provide protection by increased flow (Morris, 1991). Benefits have also been shown if patients are very elderly (Hellman, 1994), have congestive cardiac failure (Cahalin, 1996) or intermittent claudication, for which it can be an alternative to surgery (Hunt *et al.*, 1999).

Education, exercise and relaxation are similar to pulmonary rehabilitation (Chapter 9), with the differences outlined below.

Education

Education is a central component of the programme because, firstly, distress hampers the recovery process (Melamed, 1999) and, secondly, morbidity and mortality caused by angina are not necessarily proportional to the number of vessels involved (King and Nixon, 1988), indicating the importance of factors outside the coronary system. Post-MI anxiety is likely to be related to fear of a repeat heart

attack, which is associated with ischaemia and arrhythmias (Moser and Dracup, 1996). Depression occurs in an average 20% of patients after MI, usually after discharge from hospital, and this itself increases the risk of cardiovascular mortality (Seiner, 1999).

Education should include information on medication, especially as beta-blockers are associated with depression, and 30% of patients taking amiodarone experience neurological problems that may appear as symptoms of depression (Seiner, 1999). Information on lifting techniques are included for those who have had a sternotomy. Hyperventilation is common after a cardiac event, and once identified is usually manageable by education (p. 299) before the new breathing pattern becomes established and the full hyperventilation syndrome develops. Fatigue is also common and needs to be explained as a normal response.

Safety

The role of the cardiologist is to screen patients, arrange an exercise ECG (stress test) to detect ischaemic changes on graded exercise, and assist with risk assessment, summarized as follows:

- low risk: uncomplicated recovery from a small MI, no angina
- medium risk: previous MI or angina, satisfactory recovery from recent MI, no medication for heart failure
- high risk: recent large MI, angina on moderate exercise, medication for heart failure (these patients do their exercises in sitting).

If a stress test is not available, heart rate (HR) should be maintained at 10 beats below angina threshold level and exercise should be symptom-free (Hertanu and Moldover, 1996).

Exercise training is contraindicated if there is:

- unstable angina
- uncompensated or symptomatic heart failure
- resting hypertension over 200/100
- orthostatic BP drop of more than 20 mmHg with symptoms
- aortic stenosis, third-degree heart block or uncontrolled arrhythmias
- acute disorder such as pulmonary embolism, pericarditis or systemic illness (Jones and West, 1995).

Unsupervised exercise training is contraindicated if there is:

- exercise-induced hypotension
- exertional angina uncontrolled by drugs
- complex arrhythmias
- inability to self-monitor (Brannon *et al.*, 1998, p. 5).

Participants should bring their anti-angina medication with them. They must not exercise after a large meal or in extremes of heat or cold. Systolic BP should not rise more than 20 mmHg or fall more than 10–20 mmHg during exercise (Brannon *et al.*, 1998, p. 4), and dynamic exercise is preferred to isometric exercise (Figure 11.1).

Patients on anticoagulation drugs after valve surgery should avoid high-impact activity or contact sports in case of bruising or bleeding, and those on diuretics should avoid prolonged exercise in the heat in case of reduced potassium and fluid volume (Cahalin, 1996). For post-sternotomy patients, exercise should not place a shearing stress on the sternum because union takes 8–12 weeks.

Exercise training

The physiotherapist learns to identify the characteristics of each patient's angina and the feel of their individual pulse. Exercise prescription is based on exercising to a percentage of rate of perceived exertion (RPE), maximum HR (MHR) or modifications of MHR (Johnson and Prins, 1991). For MHR, 75% maximum is usually the aim, although improvements have been shown at intensities as low as 40% maximum (Lavie *et al.*, 1992). Excessive HR is inadvisable because a brief diastole prevents blood nourishing cardiac muscle. Beta-blocker or calcium channel blocker drugs dampen the heart's response to exercise.

Borg's RPE scale (Box 11.1) correlates with HR, oxygen uptake, ventilation and blood lactate.

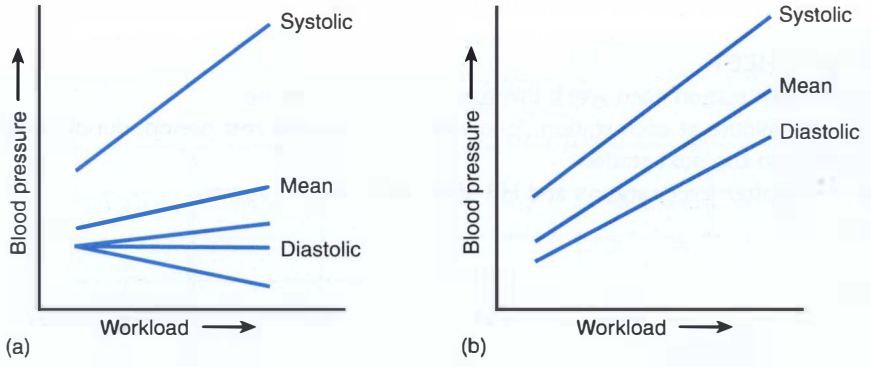


Figure 11.1 Comparison of (a) dynamic and (b) isometric exercise, showing a lesser increase in BP during dynamic work compared to isometric work. (Modified from Laslett, L. *et al.* (1987) Exercise training in coronary artery disease. *Cardiology Clinics*, 5, 211–225, with permission.)

Most patients exercise to levels 11–13 but aerobic fitness can be improved at levels 10–11, corresponding to 55–60% MHR or brisk walking, an intensity to which patients are likely to adhere (Paley, 1997). Perceived exertion is explained to patients as the total inner feeling of exertion, not leg ache, breathlessness or other perception. People with a predominantly type A personality (Ferguson, 1992) may underestimate their RPE.

If patients do not enjoy the intensity of exercise training, or if risk assessment is not available, improvement in quality of life is possible with gentle exercise for which risks do not need to be comprehensively assessed (Worcester *et al.*, 1993).

If HR is used for monitoring, participants take their pulse before, immediately after and 5 minutes after exercise, noting the time it takes to return to normal. A more comprehensive method is shown in Box 11.2. Exercise sessions last about 30 minutes, preferably three times a week. When maintaining the programme at home, three 10-minute bursts of exercise are as effective and may be preferable for some patients (DeBusk, 1990). Less intensive exercise should be continued between sessions.

Anaerobic exercise can strain the left ventricle. Patients should not exercise through angina or excessive breathlessness. If a participant gets angina during exercise, they should stop exercising and take their medication. If symptoms are unrelieved after 15 minutes of repeated medication, the doctor should be called. Meanwhile, the patient can be asked if they forgot to take their regular tablets.

A check ECG should be requested if the pulse behaves abnormally, if exercise tolerance declines over two or three sessions or if patients feel that their heart is not 'right'. Patients often detect that something is amiss before it becomes obvious, and

Box 11.1 Borg's RPE scale (Borg, 1982)

6	
7	very very light
8	
9	very light
10	
11	fairly light
12	
13	somewhat hard
14	
15	hard
16	
17	very hard
18	
19	very very hard
20	

Box 11.2 Example of documentation for circuit exercises. A new record sheet is used each week

INFORMATION SHEET

- Start with the same station each week throughout the programme
- Exercise for one minute at each station, followed by 30 seconds rest period, during which proceed with your group to the next station
- Record your RPE after every station and HR after every second station

Level A: weeks 1–2
Level B: weeks 3–5
Level C: weeks 6–8

Station	Exercise	Level
1	Shuttle walks	A: 10–14 lengths B: 20–25 lengths C: 28–32 lengths
2	Arm raises	A: out to side × 50 B: out to side × 70 C: plus 1 kg × 70
3	Sit-to-stand	A: 25–50 B: 35–40 C: 45–50
4	Step ups	A: 32–36 B: 45–50 C: 52–56
5	Floor mats	A: 20 bridges, hold for 5 B: 40 sit ups, hand on thigh C: 40 sit ups plus twist
6	Marching	A: knee high 100 paces B: arm swing 200 paces C: elbow to knee 120 paces
7	Punching Push ups	A: 125 punches forward B: 150 punches overhead C: 70 on parallel bars
8	Calf exercise	A: up/down on toes × 70 B: up/down multigym × 70 C: step marching 60 paces
9	Trampet	A: march 120 paces B: slow jog 240 paces C: fast jog 300 paces
10	Cycle	A: gently, minimum resistance B: moderately, some resistance C: brisk with moderate resistance

Continued overleaf

Box 11.2 continued

RECORD SHEET

MAXIMUM HEART RATE:
(220 - AGE)

TRAINING HEART RATE:
(220 - AGE) X 0.75

WEEK 1			WEEK 2			WEEK 3			WEEK 4		
DATE	HR at rest		DATE	HR at rest		DATE	HR at rest		DATE	HR at rest	
START STATION (circle)	HR	RPE		HR	RPE		HR	RPE		HR	RPE
1			1			1			1		
2			2			2			2		
3			3			3			3		
4			4			4			4		
5			5			5			5		
6			6			6			6		
7			7			7			7		
8			8			8			8		
9			9			9			9		
10			10			10			10		

reports of 'impending doom' need to be taken seriously. Referral for drug review is required if angina occurs, side effects increase or if exercise tolerance is reduced by pulmonary oedema.

If patients say they do not feel well or feel unusually tired, they should be advised to sit down. They can have a drink if they feel thirsty. If patients develop dizziness or palpitations, change colour or develop an irregular pulse that takes more than 1 minute to recover, they should lie down. Observation of their breathing should be maintained. When one participant feels unwell and is being attended to, the rest of the class should be kept occupied to reduce their anxiety.

Details of exercise specific to cardiac patients can be modified from page 232 or found in Broad (1991) or Brannon *et al.* (1998). Improvement normally levels off in about three months, after which a maintenance programme is followed.

Relaxation

Relaxation is an essential component of the programme. When compared to exercise training alone, relaxation and exercise training have shown a greater reduction in re-admission rates, mortality, recurrent infarctions and need for surgery (van Dixhoorn *et al.*, 1987). Stress management should be included because patients have a tendency to tolerate stress without being aware of it. Angina provoked by stress is thought to last longer than exertional angina (Brannon *et al.*, 1998, p. 95).

HYPERVENTILATION SYNDROME

> ***Hyperventilation can fairly claim to have replaced syphilis as the great mimic.***
>
> Lum, 1981

Introduction

Breathing in excess of metabolic requirements has been used for centuries by religious sects in order to achieve trance-like states of consciousness. These states incorporate some of the symptoms of acute hyperventilation but without the fear associated with the disorder of hyperventilation syndrome (HVS).

We are all familiar with the light-headedness associated with acute hyperventilation at times of excitement or stress, an adaptive response preparing for fight or flight. Hyperventilation syndrome, however, is a chronic condition associated with habitual overbreathing and somatic or psychological symptoms (Malmberg *et al.*, 2000). HVS has no known organic basis, but it depletes the body's stress-coping mechanisms and produces an array of alarming symptoms, usually intermittently.

HVS is a diagnosis begging for recognition, perhaps because it occupies the boundary between body and mind. Most sufferers have their diagnosis unrecognized, sometimes descending into chronic invalidism, but it is a surprisingly common condition that occurs to some degree in 6–11% of the population and contributes to nearly 40% of symptoms in general practice (Duncan, 1987). It appears more commonly in women (often in early adulthood) than in men (often in middle age).

Attempts to understand the condition have led to successive definitions such as irritable heart, autonomic imbalance, cardiovascular neurosis, effort syndrome, neurocirculatory asthenia, soldiers' heart, designer jeans syndrome and, as a last resort, 'factor X syndrome'. It was first identified during the American Civil War, and again exacerbated during the 20th-century world wars, when many thousands of young soldiers were invalided out with so-called heart disease. It was still not fully recognised during the Falkland and Gulf wars, although the symptoms overlap with those of Gulf War syndrome.

The lucky few who are correctly diagnosed form the tip of an iceberg of disability. Widespread failure to identify the syndrome is because:

- there is cursory coverage in medical texts
- physiological adaptation has taken place so that few patients are conspicuously breathless
- symptoms are non-specific and widely variable
- there is no unequivocal diagnostic test.

If HVS is not identified, patients trek fruitlessly from clinic to clinic, accruing ever-fatter case files, being labelled as depressive or neurotic or hysterical, and submitting to invasive investigations and sometimes years of debilitating medication. Without treatment, the condition persists or worsens in 75% of people. Some 40% of children with HVS have symptoms into adulthood (Timmons and Ley, 1994, p. 113), which is unsurprising as 86% of children appear to go undiagnosed and are often dismissed as having growing pains (Joorabchi, 1997). But HVS is eminently treatable, with symptoms being abolished in 75% of patients (Timmons and Ley, 1994, p. 113). Relaxation and breathing re-training are found to be significantly more effective than psychological methods or drugs (Kraft, 1984).

Causes

> ***The thread of the breath is woven throughout the tapestry of a person's entire life experience.***
>
> Harris, 1996

Physiologically, HVS is simply an abnormality of respiratory control, but the original factor that sets off the pattern is often difficult to identify. Body memory is thought to be held particularly in the breathing pattern (Harris, 1996), and patients may have experienced a bereavement or other loss. Contributing factors are chronic pain, withdrawal from drugs such as opiates (Brashear, 1983), liver cirrhosis because of intracellular acidosis (Karetzky, 1967) and hypermobility syndrome because of mobile thoracovertebral joints (Innocenti, 1998). Although there is usually a combination of factors, there may be one single cause (Brashear, 1983), e.g. viral illness.

Breathing occupies a central role in translating psychological changes into somatic changes (Gilbert, 1999). A vicious cycle develops in which hyperventilation, symptoms and anxiety reinforce each other (Figure 11.2), with sympathetic dominance further driving ventilation. Symptoms are brought on by a trigger such as

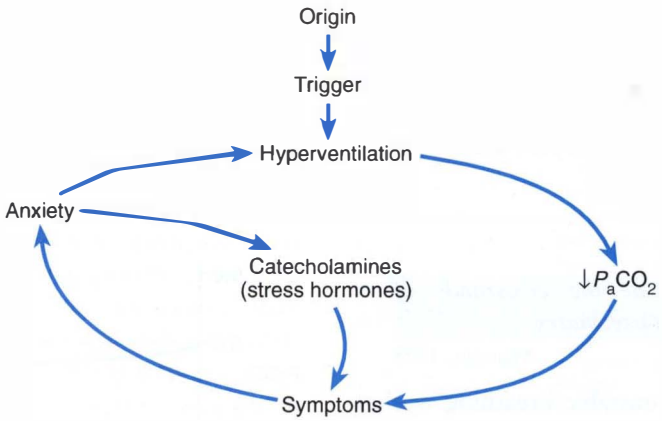


Figure 11.2 Vicious cycle into which people with hyperventilation become trapped.

anxiety or prolonged conversation. Once the cycle is established, cause and effect are interchangeable. The question that continues to tease researchers is whether hyperventilation or anxiety come first, but in practice they both augment each other. Either way, the syndrome is associated with:

- food intolerance, premenstruation (Widmer *et al.*, 1997) because progesterone is a respiratory stimulant, drugs (e.g. caffeine, nicotine, aspirin), menopausal hot flushes, spastic colon (Gilbert, 1999), prolonged mechanical ventilation
- emotional factors such as fear, suppressed anger, depression, laughter, orgasm
- occupations such as singing, which encourage deep inhalation, call centre work, which entails prolonged telephone speaking, and musicians (Widmer *et al.*, 1997)
- restrictive clothes, hence tightly corseted Victorian women taking the vapours after collapsing on sofas, and a century later the alternative name 'designer jeans syndrome'
- conditions that cause overbreathing and may predispose to HVS by reprogramming the respiratory centres, e.g. heart failure, pain, interstitial lung disease, long-term low grade fever or asthma
- in children, family discord or anxieties, e.g. school sports, with the common consequence of fainting in assembly or possibly 'mass psychogenic illness' (Jones *et al.*, 2000)
- conditions such as hypertension, chronic fatigue syndrome, 'fibromyalgia', sleep disorders, Raynaud's disease, migraine, panic attacks and agoraphobia.

Panic disorder is characterized by unpredictable and overwhelming feelings of fear accompanied by symptoms of sympathetic nervous system arousal such as sweating, palpitations, tremor and angina-like chest pain (Mansour, 1998).

Agoraphobia has been found in 60% of people with HVS, and HVS has been found in 60% of people with agoraphobia (Garssen, 1983). Other phobias can be related to HVS and may be its presenting complaint.

Although hyperventilation is a recognized stress response, it is not known why some people respond to stress with chronic hyperventilation while others develop, say, backache or a skin disorder. It may be related to past events around fear and breathing, such as a hypoxaemic birth, a ducking in the school swimming pool, a forcefully applied anaesthetic mask in childhood or a history of abuse in which crying out is physically prevented. Personality plays a part because people who respond to stress in this way

tend to suppress their emotions and are often conscientious, perfectionist and enthusiastic. It is these qualities, along with the high motivation that accompanies their relief at finding constructive help, which makes people with HVS a delight to treat.

Effects

> ***These patients sit at the crossroads of cardiology and neuropsychiatry.***
>
> Mansour, 1998

Overbreathing and/or unstable breathing washes out the body's CO_2 stores of about 120 L (Gardner, 1996), causing low and/or fluctuating P_aCO_2 levels and raising the pH of cerebrospinal fluid and blood. This reduces plasma calcium and potassium, excites neuromuscular junctions and causes the sensory aberrations characteristic of HVS. Autonomic instability of blood vessels (Figure 11.3) and nerves causes symptoms in almost any system of the body, sometimes one-sided (Gardner, 1996).

Cerebral vasoconstriction causes dizziness, faintness, blackouts, headaches, visual disturbance and sometimes a dissociated state of unreality that feels like a floating sensation or a barrier against the real world as if s/he is an outsider looking in (Lazarus, 1969). Children are often misdiagnosed with epilepsy (Enzer *et al.*, 1967).

Coronary vasoconstriction, compounded by decreased oxygen yield to the tissues due to left shift of the oxygen dissociation curve, may cause angina (Magarian, 1982) or atypical chest pain. Up to 90% of non-cardiac chest pain is thought to be associated with HVS (DeGuire *et al.*, 1992) but misdiagnosis with heart disease is frequent, especially as patients may have tachycardia, arrhythmias and adrenaline-induced ECG changes. A flattened T-wave and depressed ST segment is typical, but compared to heart pathology, these tend to disappear with exercise (Missri and Alexander, 1978). As well as its independent existence, HVS is thought to be both a risk factor and a complication of ischaemic heart disease (Weiner, 1991).

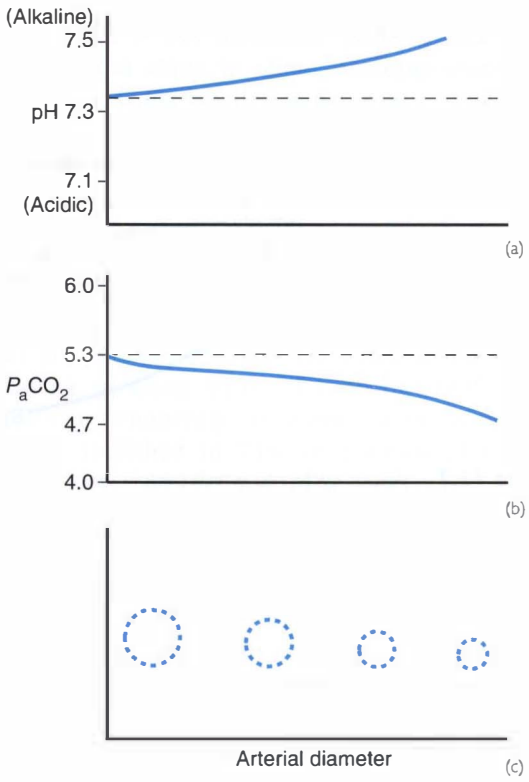


Figure 11.3 Hyperventilation causing (a) respiratory alkalosis, (b) hypocapnia and (c) vasoconstriction. (From Gilbert, V. E. (1989) Detection of pneumonia by auscultation of the lungs in the lateral decubitus positions. *American Review of Respiratory Disease*, **140**, 1012–1016)

Left shift of the dissociation curve, caused by respiratory alkalosis, depresses phosphate levels and leads to disturbed glucose metabolism, fatigue, disorientation, paraesthesia and muscle cramps (Widmer *et al.*, 1997). The kidneys try to offset the alkalosis by excreting bicarbonate. An increased drive to breathe re-sets the respiratory centres in order to maintain normal pH, obliging the patient to continue low-grade hyperventilation despite a persistently low P_aCO_2.

Misdiagnosis of asthma is common because the disorders commonly overlap (Morice, 1998). They augment each other's symptoms and hypo-

capnia can cause bronchoconstriction (van Doorn, 1982). Demeter (1986) identified asthma in 80% of patients seen for HVS. Patients diagnosed with asthma may bring on their HVS symptoms every morning when they test to see if they need their inhaler by taking a 'nice deep breath'.

The breathlessness of HVS is distinctive. It is disproportionate, fluctuating, poorly correlated with exercise, greater with inspiration than expiration and exacerbated by crowds, conversation or social situations. It usually improves with rest but occasionally worsens at night, mimicking asthma and the orthopnoea of heart disease (Magarian, 1992). The breathlessness of HVS is sometimes described as air hunger, heaviness on the chest or smothering. Negative trials of nitroglycerine or bronchodilators help to eliminate a diagnosis of heart disease or asthma, and a normal peak flow reading when breathless can be reassuring.

Activity may relieve symptoms, but sometimes loss of fine tuning means that breathing may not adjust to activity, and occasionally symptoms worsen on exercise. Even though fatigue is a common symptom, some patients choose to work out in a gym because it provides the opportunity to take deep breaths. Occupations that heighten arousal without an accompanying increase in activity, e.g. driving or watching TV, can worsen symptoms.

Other signs and symptoms are shown in Table 11.1.

Table 11.1 A selection of the clinical features associated with hyperventilation syndrome

System	Manifestation
Neurological	Tingling and numbness (especially extremities mouth)
	Faintness
	Weakness
	Blurred vision, migraine
	Poor memory and concentration
	Tremor, tetany
Emotional	Panic attacks
	Phobias (especially claustrophobia, agoraphobia)
	Anxiety
	Suppression of emotion
	Depression
	Mood swings
	Depersonalization
Gastrointestinal	Oesophageal reflux
	Difficulty swallowing
	Nausea
	Indigestion
	Wind
	Irritable bowel
Musculoskeletal	Myalgia
	Stiffness, cramps
	Tetany in severe cases
General	Air hunger
	Insomnia
	Hypoglycaemia
	Blurred body image
	Exercise intolerance

Assessment

People with HVS may or may not arrive for physiotherapy having been screened to exclude organic disease. They may have received a selection of diagnoses from peripheral neuropathy or neurosis to myocardial infarction or multiple sclerosis. Some have been dismissed as malingerers or told that it is all in their mind. Others have been told that it is 'only hyperventilation', somehow disqualifying further consideration. The first priority therefore is to ensure that patients feel welcome and know that their problem is taken seriously. They need space, time, privacy and an attentive ear.

The case notes are checked for disorders that cause breathlessness. Low haemoglobin should be identified because breathing retraining can exacerbate symptoms of anaemia. If patients are on beta-blockers, these can either exacerbate HVS by causing bronchospasm, or ameliorate autonomic symptoms and help break the vicious cycle of sympathetic stimulation and hyperventilation. If patients are being weaned off sedatives, relaxation will be difficult unless treatment coincides with the peak effect of the drug. Withdrawal from medication can itself trigger anxiety and panic attacks. β_2-agonists given for asthma can provoke palpitations and agitation (Criner and Isaac, 1995).

Subjective

> *'Feelings of flying apart, absolute terror, falling down through the world, spinning through the universe....'*
>
> Patient quoted by Bradley, 1994

Feelings vary from anxiety to fear of impending madness. Fear of dying is common (Timmons and Ley, 1994, p. 142), as is fear of flying, being trapped in a lift or feeling unable to escape from a crowded supermarket. Patients may complain of an inability to take a satisfying breath, or may in fact be unaware of any breathing abnormality. If symptoms have worsened while they are on a waiting list, this may be because a common response to a diagnosis of a breathing disorder is to practise deep breathing exercises.

It is useful to identify factors that precede symptoms and the patient's interpretation of them. Patients are often puzzled as to why symptoms affecting so many parts of the body can be caused by a breathing disorder, and may not report 'irrelevant' symptoms. Specific questions about symptoms that are likely to correspond to their experience help elicit these, and also facilitate acceptance of the diagnosis.

Questions on lifestyle may reveal a hyperactive trait and a pattern of rushing to meet deadlines. Some 63% of patients show a tendency to perform tasks quickly, immediately, impatiently, often several simultaneously and with a tendency to think ahead, whereas only 20% of normal subjects show these characteristics (van Dixhoorn, 1986). Other commonly encountered factors are:

- general hyperresponsiveness (Garssen, 1980), as shown in breathing, emotions, and sometimes allergy to food or medication
- light-headedness, sometimes leading to 'postural sway', which is similar to feelings produced by standing on foam (Sakellari, 1997)
- exercise deconditioning due to avoidance of activities that cause dyspnoea, as occurs with people who have asthma (Trooster, 1997).

Patients should say all they want at this stage because it relieves tension and reduces their need to talk during treatment, which upsets the breathing pattern.

Observation

It is normal to sigh when sad, breath-hold when frightened, say 'phew' when relieved and breathe fast when stressed, but people who chronically hyperventilate often have a habitually labile breathing pattern, with disturbance continuing after the stress is withdrawn. Minute ventilation is more likely to be observed by tidal volume in men and RR in women. The rhythm shows a variety of patterns:

- shallow, fast and apical
- sighing and yawning
- irregular
- prolonged inspiration and curtailed expiration
- excessive thoracic movement, sometimes with abdominal paradox
- 'cogwheel' breathing, as if the patient dare not let the air out
- breath-holding.

Despite the variety of possible breathing patterns, changes may be subtle and not evident. Breathlessness and hyperventilation are not necessarily synonymous. The breathing required to maintain hypocapnia is less than that required to induce it, and resting CO_2 levels may be halved with only a 10% increase in minute ventilation (Gardner, 1996).

Other signs are a stiff posture and gait, lack of coordination between talking and breathing, rapid speech as if the patient is trying to cram several sentences into one, excessive hand movements or other indication of tension, and strategies to sneak in more air such as a dry cough, throat clearing or chest heaving before speaking. Belching may be caused by air swallowing, cold hands by vasoconstriction, and moistening of the lips by a dry mouth. If chest wall tenderness is present, palpation can reassure patients that it is not heart pain. Patients are further reassured if thoracic mobilizations ameliorate this tenderness.

Questionnaires

Suspicions of HVS are raised when any person demonstrates an unusual mix of clinical features that include some of those described. The diagnosis can be confirmed by a score above 23 out of 64 on the Nijmegen questionnaire (Figure 11.4). This has been validated by Vansteenkiste *et al.* (1991) and shows a positive and negative predictive power of 94% and 92% respectively (van Dixhoorn, 1986). Further relevant information and outcome data can be gleaned by a questionnaire such as that in Figure 11.5.

Tests

Objective tests can be distressing and are limited in accuracy because of the absence of normal precipitating factors. The following are available but none have been validated.

1. Breath-holding time of less than 30 seconds is suggestive of HVS.
2. A provocation test entails rapid breathing for 1 minute, which, in patients with a chronically low P_aCO_2, may bring on familiar symptoms, not just the dizziness that is a normal response to acute hyperventilation. The test may reassure patients of the validity of their symptoms and show them that they have some control, but it is neither sensitive nor specific (Malmberg *et al.*, 2000). Vasospasm can be hazardous, and cerebral vascular disease, epilepsy and sickle cell disease are contraindications (Brashear, 1983).
3. A low P_aCO_2 is not in itself diagnostic because the syndrome is intermittent, but if patients have a series of blood gas results, a value below 4.3 kPa (32 cmH_2O) raises suspicions of HVS. A more specific test is capnography to measure expired CO_2 at the mouth, this 'end-tidal CO_2' being 4–6% in normal subjects. The mouthpiece alone can upset the CO_2 level (Han *et al.*, 1997), but if the resting level is low or erratic, HVS is implicated (Timmons and Ley, 1994, p. 109). Capnography is not distressing and can be used to provide feedback for patients and outcome measures for physiotherapists (Figure 11.6).

	Never 0	Rarely 1	Sometimes 2	Often 3	Very often 4
Chest pain					
Tension					
Blurred vision					
Dizzy spells					
Confusion					
Faster or deeper breathing					
Shortness of breath					
Tight feelings in chest					
Bloated feelings in stomach					
Tingling fingers					
Unable to breathe deeply					
Stiff fingers or arms					
Tight feelings around mouth					
Palpitations					
Anxiety					

Figure 11.4 Nijmegen questionnaire. Patients mark with a tick how often they suffer from the symptoms listed.

Name.....		
	First session date	**Last session date**
Subjective		
Headache		
Chest pain		
Abdominal pain		
Muscle pain		
Other pains		
Stiffness		
Weakness		
Faintness		
Fatigue		
Dry mouth		
Depression		
Anxiety		
Panic attacks		
Sleep		
Fitness level		
Digestion		
Swallowing		
Concentration		
Feelings of unreality		
Objective		
Hand movements		
Eye contact		
Posture/gait		
Cough		
Throat clearing		
Chest heaving		
Speech rate/rhythm		
Chest mobility		
Nose/mouth breathing		
Respiratory rate		
Breathing pattern		

Figure 11.5 Supplementary questionnaire.

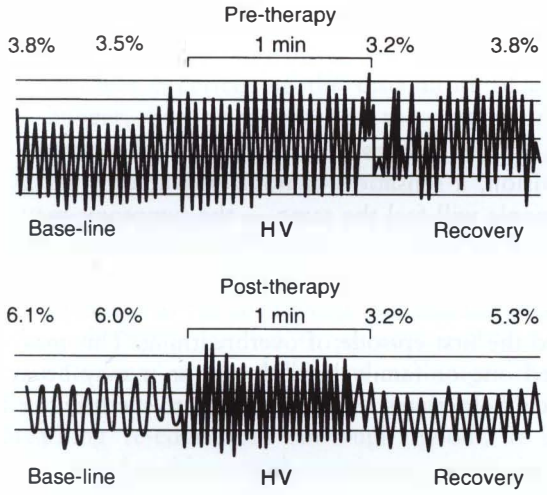


Figure 11.6 End-tidal CO_2 trace (%) before and after physiotherapy. (From Rowbottom, I. and Lothian Respiratory Function Service, City Hospital, Edinburgh). *HV = voluntary hyperventilation.*

Education

If a waiting list precludes prompt treatment, education can begin in advance. Sending an advice sheet (Box 11.4) or information about a patient-friendly book such as that by Bradley (1994) will do much of the physiotherapist's work, and sending a questionnaire to be filled out (Figure 11.4) saves time and brings some relief to the patient if they identify familiar symptoms.

At the first appointment, expectations are checked and goals agreed e.g. in the short term to cope with panic attacks and in the long term to integrate a normal breathing pattern into everyday life, as identified by elimination of symptoms. The mechanism of HVS can be explained using the vicious cycle (Figure 11.2), and this explanation can reduce anxiety and improve symptoms. The explanation includes reassurance that HVS does not cause harm, nor does it indicate any physical damage. It is a normal response to stress or other trigger, not a

Box 11.4 (from Veronica Bastow, Physiotherapy Dept, Kings Lynn Hospital)

HYPERVENTILATION SYNDROME

What is hyperventilation syndrome?

'Hyperventilation' means overbreathing, which may be taking too many breaths or breathing too deeply. 'Syndrome' means a collection of symptoms.

Our breathing normally happens without us having to think about it. The number of breaths and size of breath is controlled by our 'breathing regulator' system. This system causes us to take a breath of air into our lungs so that oxygen can move into our bloodstream and be used by the different parts of our body. As the oxygen is used up, we produce carbon dioxide which is our 'exhaust gas'. This moves back to the lungs in the bloodstream, and as we breathe out, some of this carbon dioxide is breathed out into the air. However, we need to keep some carbon dioxide — it plays an important role in our blood.

If we breath faster or more deeply than our body needs, we may breathe out too much carbon dioxide. This can give us some funny feelings varying from quite mild symptoms in some people to quite severe symptoms in others.

All of us at some time in our life will probably overbreathe and have some of the symptoms caused by this — this is nothing to worry about. However, a few people will go on to have Hyperventilation Syndrome, where the symptoms happen more often and help is needed to stop them.

How do I know if I have Hyperventilation Syndrome?

It is sometimes spotted by your doctor, although some people feel so unwell during an attack that they go to the casualty department.

Box 11.4 continued

What are the symptoms likely to be?

The symptoms are quite variable and may include tingling fingers, tingling around the mouth, dizziness, fainting, chest pain, tiredness, disturbed vision, a sensation of not being able to get a deep enough breath, sighing and yawning. No two people will feel the same — the symptoms may be quite different.

What causes it to start?

Quite often there has been an event which has caused the first episode of overbreathing. This may be an emotional happening such as death of a loved one or family breakdown, or it may be a physical cause such as intense pain or an asthma attack. Sometimes it is not possible to identify the first event.

What tests might be done?

Sometimes a blood test is done, but normally the history and symptoms alone identify the problem. The doctor or physiotherapist will probably listen to your chest and may ask you to blow into a peak flow meter to check how well your lungs work.

What treatment can be given?

A physiotherapist will work with you to help you to be more aware of your breathing and to teach you to breathe in a more normal way. You will probably be given breathing control exercises to practice at home.

Will I get completely better?

Most people do get better. However there is always a chance that you may have another attack if something triggers it. However, hopefully you will have learned how to cope and will be able to control it with the breathing exercises.

How long will I need to have physiotherapy?

This will be different for each patient. If may take many weeks for some to learn the breathing techniques and for their body to adjust to the higher (more normal) level of carbon dioxide. Your physiotherapist will want to be sure that you are much better and have learned how to cope if a relapse should occur.

psychiatric illness. When patients understand that their symptoms stem from chemical changes in the blood, they can feel exonerated from the stigma of not having had their complaints validated by a diagnosis. The following advice may be found helpful:

- Physiotherapy will not eliminate the cause nor remove precipitating factors, but a collaborative approach helps to identify triggers, break out of the vicious cycle and gain control over breathing.
- Commitment is required in the early stages when learning to control breathing, and in the later stages when incorporating practice into everyday life.
- It is useful, and for some people essential, to integrate a small but fundamental shift in attitude and lifestyle that allows time for relaxation and reflection.

- A nice deep breath does not help relaxation.
- Re-learning the new lower level of breathing involves experiencing the discomfort of air hunger, but after practice the respiratory centres will become re-tuned to registering a more normal breathing pattern.

To anticipate the feelings experienced with breathing re-education, 'bad breathlessness' can be explained as the distressing and uncontrolled symptom that is familiar to patients, and 'good breathlessness' as the feeling of air hunger that they initiate and control as part of their breathing re-education. Although 'good', it is not comfortable, and patients appreciate acknowledgement of this.

Patients are advised that symptoms are occasionally worse in the first week of treatment. This is thought to be due to a paradoxical but transient increase in minute volume. The respiratory centres may interpret breathing re-education as a form of suffocation and find subconscious ways of increasing ventilation temporarily. For people with long-standing HVS, symptoms sometimes worsen during the first session when they attempt to reduce the minute volume, possibly as the body's reaction to an apparently threatening intervention.

Education enables patients to step out of their vicious cycle and begin to take control of their breathing. Figure 11.7 shows the ventilatory response to graded exercise before and after gaining insight into the nature of the condition.

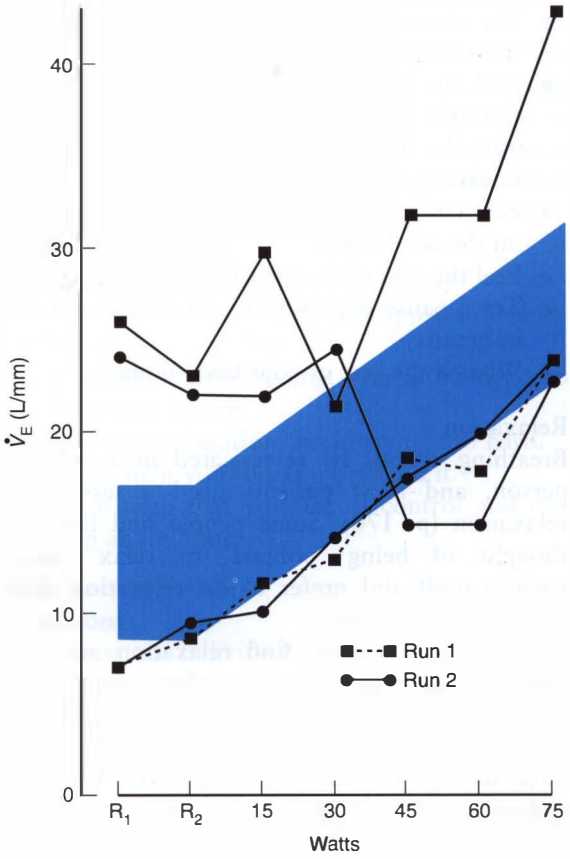


Figure 11.7 Ventilatory response to graded exercise in a patient before (higher lines) and after (lower lines) education on the process of HVS. Two measurements were made 30 min apart on each occasion. The stippled area represents the range of response seen in normal subjects. (From Howell, J. B. L. (1997) The hyperventilation syndrome under threat. *Thorax*, **52**(suppl. 3), S30–S34)

Breathing re-education

According to psychoanalyst Wilhelm Reich, changing a person's breathing pattern is tantamount to emotional surgery. The safe environment of a physiotherapy department is unlikely to excavate the depth of emotion that is expected from the analyst's couch, but feelings may surface and if this brings tears, a proffered box of tissues lets patients know that this is acceptable. A quiet room is required, with an open window or fan. The patient settles comfortably into half-lying, with a pillow under their knees.

Awareness of breathing

Patients can learn the feel of their breathing, using some (but not all!) of the following:

- Rest one hand on the upper chest and one on the abdomen to distinguish upper chest and abdominal breathing.
- Experiment with slight alterations in depth and rate of breathing, to distinguish the two concepts.

- Try alternate nose and mouth breathing to feel the difference.
- Feel the passage of the breath as it passes through the nose, down the windpipe and into the lungs, then visualize the air gently returning along the same route.
- Feel cool air on the in-breath and warm air on the out-breath.
- Feel the size of the breath, feel its shape.
- Try a pause between the out-breath and the in-breath.
- What is the rest of your body doing?

Relaxation

Breathing cannot be re-educated in a stressed person, and most patients need a session of relaxation (p. 170). Some people find that the thought of being 'obliged' to relax causes tension itself and prefer to do relaxation after the breathing session.

Tense people often find relaxation an alien concept, and it may be easier after a brief neck massage, during which it is helpful for patients to focus on the experience and not feel that they have to talk or 'do' anything. Even the old-fashioned infrared lamp can be helpful prior to relaxation, usually to the back with the patient prone. Lying prone may facilitate relaxation, possibly because this is a less vulnerable position. A highly developed sense of success and failure is often prevalent with HVS, and the relaxation session is an opportunity to reinforce that there is no 'right' or 'wrong' way.

Focusing on the breath itself helps relaxation, especially if patients are encouraged to very gently 'breathe in the good air' and 'breathe out the tension', as if freeing the breath. A relaxed state can be maintained by bringing the patient's awareness to areas of tension throughout treatment, including the jaw and throat. Physiotherapists should ensure that they themselves are relaxed.

Settling the breathing

> ***Man should no more breathe through his mouth than take food through his nose.***
>
> Clifton-Smith, 1999, p. 30

A habit of nose breathing can be established during this session, by explanation and role modelling. To settle the breathing pattern, the physiotherapist encourages regular and gentle speed, depth and synchrony, using a rhythmic voice. Abdominal breathing (p. 154) is useful, taking care to maintain small gentle breaths. Abdominal breathing may be facilitated with the elbows above waist level, or hands behind the head or back, and/or by lying prone, while maintaining relaxation. Occasionally abdominal breathing is easier after reducing the minute volume.

Reducing the breathing

A combination of education, relaxation and abdominal breathing has shown improvement in 94% of patients (Pinney *et al.*, 1987). But patients with established HVS may require further intervention to reduce minute volume. Patients can be asked simply to 'breathe less', very gently to avoid tension and exacerbation of abnormal breathing patterns. Some patients need the explanation that this means reducing the rate or depth of breathing, or both, but thinking too hard about an automatic process may be counterproductive. So long as the patient understands that the aim is to achieve the slight discomfort of 'air hunger', s/he can often achieve this independently without too much control by the physiotherapist.

'Low and slow' is the key. Some patients need only a pause at end-exhalation, so long as this does not go further than air hunger and cause tension. If patients tense up, they should focus on returning to smooth, gentle, rhythmic breathing.

To help patients maintain their rhythm, it is best not to ask for verbal feedback during the practice, although they can nod or shake in answer to quiet questions. Observations of the breathing pattern will indicate tension, suggesting that they have gone too far and need a reminder not to allow more air hunger than is 'slightly uncomfortable'. They are advised to then gently get their breath back.

The physiotherapist watches closely and may

need to give selective advice on rate or depth. Manoeuvres to slip in a covert deep breath include a subtle change in breathing pattern, body movement, shift in position, or preceding speech with a sharp intake of breath. The physiotherapist and patient can compete as to who notices these first! However, much of the time is spent in silence as the patient focuses on their breath and achieving air hunger.

In the first session, when patients feel air hunger, they are congratulated and advised to start getting their breath back by allowing themselves slightly deeper and/or slightly faster breathing, without gasping. When they are able to tolerate the air hunger, they are asked if they can experience it for a few moments so that their respiratory centres can begin receiving normal messages. The patient gradually learns the right balance for themselves of 'slight discomfort but no tension'. It is similar to the 'slight breathlessness' taught to respiratory patients when desensitizing to breathlessness. The periods of air hunger can be gradually extended.

If this is too nebulous for the patient, more structured support can be given by pacing the patient's breathing to the physiotherapist's voice. The patient is asked to breathe in time with the physiotherapist's words, the rate of which is slightly slower than the patient's rate. Counting or pacing may be used e.g.:

- 'In-and-out, in-and-out....'
- 'In-and-out-two-three, in-and-out-two-three....'
- 'In-and-relax-out, in-and-relax-out....'
- 'In-and-let-it-out, in-and-let-it-out....'

Words and timing should be flexible to suit the patient, but words need to be repeated rhythmically. Some patients find that this pacing brings a sense of security in the early stages, the words acting as a 'breathing pacemaker'. Progression is aimed at independent control without the physiotherapist's voice.

If patients feel an irresistible need for air, they can take a conscious and controlled deeper breath, then get back gently into rhythm, sometimes with a preliminary breath-hold as compensation (but not if this causes tension). The concept of control is important for people who hyperventilate because they have felt out of control of their most fundamental physiological function. Advice can be given at intervals if necessary:

- Keep it smooth/shallow/slow.
- Swallow if you need to suppress a deep breath.
- Keep the rhythm going, you don't need to hold your breath.
- Maintain relaxation, avoid trying too hard.
- Don't fight your breath, befriend it.
- Be assured that you are in control and can stop at any time.

Variations

A process as individual as breathing needs a flexible approach. Suggested variations are the following.

- Physiotherapists can use themselves or a mirror to demonstrate the patient's breathing pattern and different options.
- Patients can slow down by 'breathing in' to areas of muscle tension, then 'breathing out' the tension, or they can visualize inhalation as if going up a hill and exhalation as if coming down the other side.
- The simple yoga technique described on page 172 suits the most hardened workaholic because it is so brief.
- Some patients slow down if the physiotherapist moves physically away and asks them to 'breathe from where I am'.
- Humming may slow the breath.
- Putting the tip of the tongue between the lips encourages nose breathing.
- Neurophysiological facilitation (p. 154) may have beneficial results.

By the nature of the syndrome, it is essential that patients are not hurried, and an undisturbed hour should be set aside for the first session.

Progression and home management

As control is established, the process of modified relaxation, rhythmic breathing and breathing re-education is repeated in sitting, standing, walking and activities that might cause breath-holding such as bending, stair-climbing or eating. Particular attention is required for jobs that involve speech such as teaching or using the phone. If prolonged talking brings on symptoms, slowing down speech can be practised by reading aloud, starting with poetry in order to use the natural pauses, then reading stories to children. Tips for maintaining control during speech are:

- check shoulder relaxation and breathing pattern
- take small breaths and inhale through the nose between sentences instead of gulping through the mouth
- add mental commas (Bradley, 1994).

Pacing functional activities is often beneficial, although difficult for people who are hyperactive. Conditioning is improved by exercise that is steady, rhythmic and enjoyable, with the patient discouraged from indulging in either anticipatory hyperventilation or obsessive overachieving.

Posture and breathing may be affected by tense abdominal muscles, which may compress the abdomen on inspiration, especially in people who need to appear in public or are obese. A balance can be negotiated between allowing outward abdominal movement while feeling comfortable with their appearance. Tight clothes and belts should be avoided.

In the early stages, some people with severe HVS find that wearing a large-volume oxygen mask (minus oxygen) at home helps to retain P_aCO_2 at night or during ADL, so long as they do not become emotionally dependent on it. Paper bags, or plastic bags with the corners cut out, can be used as a 'bandaid' for acute episodes.

Patients are advised to maintain a steady blood sugar by having breakfast (including protein which is slow to metabolize), avoiding going without a snack for more than 3 hours, and avoiding carbohydrate binges, which may lead to reactive hypoglycaemia. This should be emphasized for patients who are too busy to eat during the day and eat heavily at night, which can produce night-time or early-morning symptoms. Meals should be slow and enjoyable, with patients avoiding excessive coffee, cola or chocolate. If patients must smoke, deep inhalations are to be avoided.

To assist sleep, some patients find it helpful to follow the yoga practice of spending the first 5–10 minutes lying on the left side, to warm the body, then turning to the right side to assist relaxation (Clifton-Smith, 1999, p. 30).

Panic attacks occur in 50% of people with HVS (Cowley, 1987). Once started, they may be as impossible to stop as a firework, but they become less prevalent once patients begin to gain control. Coping strategies include identifying trigger factors, talking through the process, relaxing the shoulders, swallowing, behavioural techniques such as rehearsals or distraction, acupressure to Lu1 two thumb-widths lateral to the nipple line in the second intercostal space (Ellis, 1994, p. 49) and techniques to manage breathlessness (p. 173) or coughing fits (p. 204). If out in crowds, window shopping can be used while focusing on coping strategies. An internal dialogue can be initiated at the onset of panic, e.g.: 'these symptoms are frightening but this has happened before and I know it's simply over-breathing and I'm not going mad or dying'. Breathing gently into cupped hands held over the nose and mouth helps to retain CO_2.

Much encouragement is needed to help patients integrate their new breathing pattern and attitude into the distractions of everyday life. If progress is slow, attention can be given to identifying individual fears and precipitating factors. Reassessment of the abnormally high demands to which patients often subject themselves may be fruitful.

A handout helps to correlate this mass of information, especially as poor concentration is a common symptom. Audio tapes of advice and relaxation can be helpful, sometimes including

the physiotherapy session. If counting is used, this can be taped at fast, medium and slow speeds. If commercial relaxation tapes are used, patients are reminded to ignore any instructions to breathe deeply.

It is worth motivating patients to work hard during the first crucial week. Some are able to set aside 20 minutes a day for relaxation. Others find individual ways of relaxing such as a rocking chair, personal stereo or fishing. An extended hot bath is not advised because excessive heat stimulates the respiratory centres.

Practice in breathing re-education should take place little and often, after brief relaxation and with the patient's mobile phone switched off. This could be around three times a day for 15 minutes, or mini-sessions of 3 minutes every 1–2 hours. Times can be flexible to suit the individual. Spot checks throughout the day can be assisted by memory aids and use of opportunities such as red traffic lights, coffee breaks, queues and 'grey bar time' while the computer is processing. Computers can have beeps programmed in at set intervals to act as reminders. Gradually the practice sessions become less defined as the correct minute volume becomes automatic.

Patients who tend to become preoccupied with a daily programme should not be burdened with excessive homework. After the first hard-working week of raising awareness and developing new habits, it is best to use education as the basis for individuals to make their own decisions, with advice as required. Flexibility is particularly necessary for mothers, who find a tight routine impossible.

Physiotherapy is needed weekly until self-management is stabilized, usually after a few sessions, then sometimes monthly for adjustment and encouragement, followed by advice that patients can ask for a review session if required. Once learned and reinforced, the new breathing pattern can be maintained automatically because there has been no physical damage, as there is in emphysema. Self-awareness and stress management however must last a lifetime. Patients are advised that hyperventilation may never return, or it may return at stressful times such as Christmas or a wedding, but they will recognize it and should be able to control it. A hint of symptoms can become a useful barometer.

If patients ask for advice on complementary therapies, many techniques such as reflexology and aromatherapy are helpful for relaxation, as are meditative practices that exclude deep breathing techniques. Some osteopaths and Alexander teachers specialize in HVS. Hypnotherapy is unwise for people who suffer episodes of depersonalization.

If patients do not improve after several sessions, and it becomes apparent that they are not practising at home, or if they exhibit a 'yes, but . . .' tendency, it is possible that they subliminally 'need' their hyperventilation to block out memories, in the same way that some patients with chronic pain express their emotional distress on a physical level (Bruera, 1997). This is not a conscious process and makes the disorder no more tolerable, but if it is the case, physiotherapy is unhelpful and may just 'feed' the somatization.

Outcomes

The simplest outcome is reduced RR, aiming at 12 breaths per minute, which Sakakibara (1996) has reported as alleviating panic attacks. Subjective report of reduced symptoms is relevant to the patient. Outcomes can include the breathing pattern, breath-holding time, a Nijmegen questionnaire or capnography. The following outcomes have been documented:

- doubling of breath-holding times (Maskell *et al.*, 1999)
- improvements in capnogrophy, anxiety, depression and other symptoms after 3–14 weeks of treatment (Tweeddale *et al.*, 1994)
- reduction in Nijmegen scores and Hospital Anxiety and Depression Scale in all patients audited after 2 treatment sessions and a phone call (Williams, 2000).

Discharge letters to both GP and consultant help raise awareness of the syndrome and effectiveness of treatment.

To be wholly alive is to breathe freely, move freely and to feel fully.

Lowen, 1991

ELDERLY PEOPLE

Work is now urgently needed to rehabilitate rehabilitation for elderly people.

Young, 1998

Age is becoming less acceptable as an explanation for ill-health. The majority of respiratory patients are elderly, and they need to start rehabilitation as soon as they are admitted to hospital. This helps to reduce the dependency, anxiety and depersonalization to which they are susceptible in this environment. Rehabilitation for elderly people is cost-effective (Young, 1998). It can reduce death rates by 35%, re-admissions by 12% (Kings Fund, 1998) and maintain independence. Without rehabilitation, one study found that as many as 75% of independent elderly people were no longer independent on discharge from an acute hospital (Hamilton and Lyon, 1995).

Physiological changes associated with ageing are discussed on page 18. Some distinctions between clinical changes due to ageing and those due to avoidable factors are discussed below.

1. Confusion can be caused by hypoxaemia, dehydration, infection, pain, over-medication (especially sedatives), disturbed sleep, depressive illness, lack of hearing aid or glasses, or disorientation resulting from admission to hospital. Several of these factors contribute to postoperative confusion (Moller, 1998). Confusion should not be accepted without investigation unless dementia has been diagnosed by a specialist.
2. Depression is an independent risk factor for mortality in institutionalized older people (Schulz, 2000). It is a common outcome of the helplessness associated with hospitalization or the move to an institution, and often goes unrecognized, especially in people who have got out of the habit of asserting themselves. This underlines the importance of consulting patients throughout. Millard (1983) asks 'When autonomy is removed, is death the only personal choice?' Depression should always be considered if health staff comment that 'she's forgetful, she's a wanderer, she's beginning to dement'.
3. Postural hypotension is a drop in systolic BP of 20 mmHg or more on standing, and is present in a third of people over age 65. It is related to vascular insufficiency, dehydration or the side effects of certain drugs including tricyclic antidepressants, and it causes 5% of falls (Lubel, 1989).
4. Impaired absorption, distribution, metabolism and elimination of drugs is common, and side effects are often missed. Some 60% of serious adverse drug reactions in elderly people have been blamed on diuretics, which are often given for oedema even though oedema in the elderly is usually caused by immobility (Valacio and Lie, 1994).
5. Regulation of body temperature may be unstable, causing impaired response to a cold environment or inability to develop a pyrexia in response to pneumonia (Irwin *et al.*, 1998).
6. Silent aspiration of stomach contents may occur, especially at night.
7. Aches and pains are pathological and not to be accepted as part of ageing.
8. Some reduction in exercise tolerance is expected, as demonstrated by a linear reduction in $\dot{V}O_{2\ max}$ with age (Paterson, 1992), but poor mobility is more likely to be due to treatable conditions such as anaemia, painful feet, fear of exercise or lack of vitamin D which is common in institutionalized elderly people (Bischoff *et al.*, 1999). People in their ninth decade have shown increased exercise tolerance with a low-frequency training programme (Hamdorf, 1999). Box 11.5 is an example of a mobility progress sheet kept at the bedside and used by all members of the team.
9. An assumption that incontinence is

Box 11.5 Progress chart. Dates are added to the left column. Mobility aids are added as relevant. +2 = *with two assistants*, +1 = *with one assistant.*

Mobility				
Baseline	none	+2	+1	independent
at D/C				
Ex.tolerance (yds)				
Baseline				
at D/C				
Stairs (no.)				
Baseline	none	+2	+1	independent
at D/C				
ADL				
toilet:	none	+2	+1	independent
wash:				
dress:				
other:				

inevitable may lead to mopping-up taking precedence over preventive action such as maintenance of mobility, ensuring access to the bathroom and emptying the bladder before peak flow readings.

10. Constipation may be due to an inefficient thirst mechanism (Hyde, 1999), medication, change of diet, dementia, immobility, lack of privacy or feeling hurried when on the commode. As well as addressing the cause, exercise and abdominal massage show positive outcomes (Resende and O'Neill, 1992), and massage has the added benefit of restoring the benefits of touch, which are sometimes lost to elderly people (Fraser and Kerr, 1993).
11. Breathlessness is considered common (Boezen, 1998) which may be why respiratory disease is often overlooked (Patterson *et al.*, 1999), and reversible components may not be treated even if a diagnosis is made (Sherman *et al.*, 1992). Self-imposed ageism prevents some patients reporting symptoms (Luce, 1996).
12. Misery is not an inevitable accompaniment to old age.

Unsteady gait can be affected by changes in any of the three main afferents of the posture and balance control mechanism: vision, vestibular input and proprioception. Other possible causes of falls are:

- poor eyesight
- poor balance
- poor footwear
- weakness
- lack of confidence
- postural hypotension
- medication
- transient ischaemic attacks
- breathlessness
- pain.

Practical ways to help maintain orientation in elderly people are the following:

- Avoid using first names uninvited (Gordon, 1994), because for older generations this tends to be seen as a sign of disrespect rather than a sign of friendliness.
- Encourage patients to wear normal clothing when possible.
- Ensure patients are kept fully informed throughout.
- Encourage patients to bring to hospital their budgerigars (Jones, 2000) and as much clutter of personal possessions as allowable.
- Avoid physical restraints.

Physical restraints slow rehabilitation, decrease mental functioning, cause fear and discomfort and can increase rather than decrease the risk of injury (Schieb *et al.*, 1996).

Untreated pain can reduce mobility, disrupt sleep and lead to malnutrition, social isolation and depression. Some of these effects may result in yet more prescription of drugs (Closs, 1996). A variety of pain scales have been developed for assessing pain in the elderly (Morrison *et al.*, 1998; Herr, 1998).

Other problems which can hinder rehabilitation are malnutrition, to which elderly patients are particularly susceptible (Tierney, 1995), and memory loss. Rastall *et al.* (1999) advise writing down physiotherapy advice and exercise programmes. Exercise programmes not only improve ADL but have been shown to reduce daytime agitation and night-time restlessness in nursing home residents (Alessi, 1999).

Autonomy is central to rehabilitation. It has been found that the less residents of institutions have control of their lives, the more they lose control over the use of their faculties (Bach and Haas, 1996, p. 448). Autonomy can be facilitated by respecting patients' senior status, experience and wishes regarding management. This means, for example, allowing them to return to bed when they request, rather than enforcing unhappy hours slumped in hospital chairs. Discomfort reduces the depth of breathing, and the zeal with which patients are hauled out of bed for lengthy periods has led to 'chairsores' becoming more prevalent than bedsores in some hospitals (Mulley, 1993).

Box 11.6 Daily exercise programme for Mr/Ms .....

TO BE DONE ONCE/TWICE A DAY

Sitting up in bed or sitting in your chair:

(1) Circle your shoulders: shrug both shoulders up slowly, then pull them forwards, then down, then backwards, then relax.

(2) While breathing in, lift both arms up forwards until over your head. while breathing out, bring them down sideways slowly.

TO BE DONE ..... TIMES A DAY

(3)
IF IN YOUR CHAIR:
Tighten your thigh muscle and straighten your knee slowly. Hold for a count of 3, then let it down slowly. Repeat with other leg.

Repeat ..... times

IF IN BED:
Pull your toes up, push your knee into the mattress, lift up your straight leg slowly. Hold for a count of 3, then let it down slowly. Repeat with other leg.

Repeat ..... times

Standing

(1) Stand up, take a relaxed breath, sit down.

Repeat ..... times

(2) Walk to .....,
Rest and get your breath back.

Walk back to your chair.

Respiratory health is best maintained by a personally tailored programme of little-and-often mobility, and return to a home environment as soon as possible.

Box 11.6 is an example of an exercise sheet that can be customized to each patient and brightened up with pictures from PhysioTools.

PEOPLE WHO ARE DYING

'It begins with an easy voice saying,
Just a routine examination;
as October sunlight
pierces the heavy velvet curtains.
Later it is the friends who write but do not visit....
it is boiled fish....
it is doctors who no longer stop by your bed....
it is terror every minute of conscious night and day to a background of pop music.'

Wilkes, 1983.

Physiotherapists are suited to working with people who are dying because of their skill with physical contact, which can communicate what words cannot, and their experience with disabled people, who often have similar needs to people who are dying (Purtilo, 1976). An area as subjective as death requires more of us as humans than as 'experts', and working with dying people can be enriching and painful.

End-stage disease is not a time to withdraw physiotherapy, because there is much that can be done to ease the passage towards a good death.

Palliative care is the aim for patients with advanced, progressive and ultimately fatal disease, e.g. metastatic malignancy, some neurological conditions and end-stage AIDS or respiratory failure.

Reactions of patients

> *'Now and then the whole thing becomes unreal. Out of the middle of the night's darkness, or bringing me to a sudden, chilling halt during the day, the thought comes: this can't be happening to me.* Me *with only a few months to live? Nonsense. And I stare up at the darkness, or out at the sunlit street, and try to encompass it, to feel it. But it stays unreal.'*
>
> Bell, 1961

When told that they are dying, most people feel overwhelmed and experience a variety of reactions. Fear is usually predominant at first, although not primarily fear of death itself (Murray-Parkes, 1998). There is fear of the dying process, fear of isolation, fear of being a burden, fear of symptoms and disintegration, fear of the unknown, and reflected fear in the eyes of those around them and the questions that are not asked.

For a person dying from a smoking-related disorder, guilt is an extra burden. Anger is another ever-ready emotion that may arise from feelings of helplessness, or act as a defence against the experience of grief. Grief is a normal response but if suppressed can develop into depression, which amplifies pain, distresses relatives and erodes the patient's ability to do the emotional work of separating and saying goodbye (Block, 2000). Patients should be allowed to express sorrow, anger, guilt, unusual humour or any other feeling, for which they should not have to apologize.

Many patients deny reality in order to avoid the pain of grief or fear, acting and talking as if they expect to get better. Denial is a defence mechanism to be respected because it is a natural response and a necessary cushion. When and if patients are ready to confront the truth, they may sink into a dark place that can paradoxically be a creative process by which they begin to take responsibility for the way they respond to change. Only then can they accept their loss and allow fear to dissolve.

These reactions are not stages that occur with defined boundaries but they weave in and out of a patient's awareness, so that a moment of anger may open into acceptance, followed by the mind curling back into the darkness of fear. Time is needed, and patients with cancer or emphysema have time, which, with support, can be used wisely.

Reactions of relatives

> *'When someone you love dies, you pay for the sin of outliving them with a thousand piercing regrets.'*
>
> Simone de Beauvoir, 1966

Support for relatives assists the patient. Families and friends can feel a kaleidoscope of emotions such as remorse, relief, impotence at being unable to help, and similar reactions to those of the patient. Bereavement can be eased before death by involving relatives in decisions about levels of support for patients who are unable to make their own decisions (Billings, 2000). Risk factors for the more difficult emotions include:

- prior ambiguous or dependent relationship with the dying person
- in Western societies, advanced age
- minority groups unable to follow their own customs
- those who have learning difficulties or are confused (Sheldon, 1998).

A demented person who loses a partner may repeatedly forget, and can feel shock and grief each time they are told. They need much support through their bereavement, including

involvement in rituals such as the funeral and visiting the grave.

Relatives need the opportunity to share the truth with the person who is dying. Patient and spouse are often given contradictory information (Thomsen, 1993) which leaves them out of step with each other. Both might try to 'protect' the other, sometimes with the collusion of health staff. Just when they need each other the most, they are separated by a conspiracy of silence.

Talking is helpful (Timmons and Ley, 1994, p. 246) as is assistance in providing comforts for the dying person. Children also benefit from open communication and need the opportunity to be close to their dying relative. They know their own limitations and may simply want to pop in and out of the sick room. Children often fantasize that they are to blame for the death of a parent or sibling, or they may feel they must not distress their parent and so avoid talking about it. Telephone contact with bereaved relatives after the death has been found helpful (Stone *et al.*, 1999).

Reactions of staff

> *'The Sister was very cross with me and told me to pull myself together because the Consultant was coming.'*
>
> Blanckenhagen (cancer patient), 1986

Once a patient's condition is known to preclude recovery, this can be interpreted as a failure by health staff. Reactions may include avoidance, heroic measures to prolong life, unsuitable bonhomie, the use of drugs to suppress patients' expression of emotion, inaccurate optimism (Billings, 2000) or inappropriate reassurance. Reassurance has been criticized as 'social bromide' aimed at making staff feel better rather than the patient (Fareed, 1996).

Health staff working with people at the end of life need support themselves. They need access to their own feelings because expression of feelings by staff, when appropriate, has been found to be therapeutic for patients, who find professional detachment unhelpful and even offensive (Fallowfield, 1993).

Communicating with dying people

> *'His yellow eyes watched us being taught at the bedside of each patient and when we came to his bed we all walked directly past him to the patient on his other side. Not a word was said. Not a greeting. Not even a nod. ... Dismay turned to guilt with the thought that I, too, had no idea how to approach or comfort a dying patient.'*
>
> Carmichael, 1981

It is not easy to find the right words to say to people who are facing death. The key is to listen. Patients find relief if they feel that it is acceptable to talk, and the astute listener can pick up indirect questions. Patients may drop hints that they would like to talk by ploys such as mentioning other people who have died, joking about their future or asking how long before they get better. We can indicate a willingness to listen by asking if we can sit on the bed, maintaining eye contact and asking non-threatening questions such as 'How do you feel in yourself?' While patients are talking, they can be encouraged by prompts such as 'Go on' or simply 'Mm?'.

During and after talking, patients need time to process their thoughts, and silence can be used constructively. It is not helpful to rationalize patients out of their feelings, tell them what to do, or say that we know how they feel (we do not). It is however helpful to provide information that reduces anxiety, and discussion itself helps to divest death of its power. Uncertainty is one of the hardest things to bear (Bortoluzzi, 1994), and patients who are left in ignorance feel a loss of control that shackles their coping strategies.

We might also find it useful to ponder our own reactions: 'Am I feeling uncomfortable? Am I helping or hindering her flow of thought? Am I responding to his needs or mine? Am I frightened of death myself?'

Honesty is essential for this form of communication. The majority of patients want to be told their diagnosis and feel they should have a

say in who else is told (Buckman, 1996). Information increases the ability to cope (Falk, 1997). Fear of the unknown is a heavier burden than the truth. Those who do not want to hear have their safety strategy of denial. It is thought that most patients realize eventually that they are dying (Barbato, 1998) and then may be alone to face the truth from which they were being sheltered.

When patients ask questions about their prognosis, however indirect, it is unethical to avoid giving information, and keeps patients in a subordinate position. Lying to patients may stem from a false assumption that distress equals harm, or uncertainty about who should take the initiative. In theory physiotherapists have as much right and responsibility to inform patients as other health staff (Sim, 1986), and UK guidelines give physiotherapists discretion (Barnitt, 1994). In practice, it is often another member of the team who communicates difficult information; while the physiotherapist's role is to ensure that patients' questions are answered, and that issues of power about who 'owns' the truth do not hinder this. These problems are not new; in 1672 a French physician considered the idea of telling the truth to patients, but concluded that it would not catch on (Buckman, 1996).

Honesty should be tempered by sensitivity, with an emphasis on what medical science has to offer, and a check that patients do not associate emotive words like 'cancer' with misconceptions about an inevitable and distressing death.

To reveal the diagnosis to the family without the patient's knowledge creates tension and mistrust, and is unethical. Family requests, e.g. to suction a patient, should be respected and discussed, but should not take precedence over the rights of the patient (Snider, 1995).

Reaction to bad news is varied and sometimes irrational, including regression to child-like behaviour, relief, despair at the loss of fulfilment, or projection of hostility. Patients may choose to face in a different direction from that which we intend, but defence mechanisms are rarely maladaptive. Patients should be left with some realistic hope, even if directed towards a minor achievement. And it is always worth casting a backward glance when leaving the bedside, because it is sometimes necessary to return and pick up the pieces.

> ***'Bearing the agony of knowing one has a life-threatening condition is not as problematic as not being given adequate information.'***
>
> Dewar, 1995

Management of symptoms

As soon as it is known that patients are in need of care rather than cure, the emphasis is on allowing them to choose both the method and timing of their treatment. Palliative care does not preclude rehabilitation, including setting goals and maximizing independence.

Breathlessness

Breathlessness is the most common severe symptom in the last days of life (Molen, 1995) and is present in up to 90% of people with a variety of advanced cancers (Dudgeon and Lertzman, 1998). Patients are less likely to have developed coping strategies than with slowly progressive COPD. The pharmacological and physical management of breathlessness is discussed in Chapters 5 and 7 but further measures can be taken for people who are dying.

Dyspnoea may be caused by a tumour, lung fibrosis following radiotherapy, cachexia, or a coexisting condition. Treatable causes of breathlessness should be identified, e.g. pleural effusion, ascites, anxiety, obstruction or compression of the lung, or anaemia. Steroids may relieve breathlessness associated with diffuse malignant lung involvement. The normal constraints on using these drugs are unnecessary at the end of life. The cannabinoids are licensed in the UK for treating nausea, vomiting and lack of appetite (Sharpe, 2000). Patients being managed at home often feel relieved if they can self-administer, for example, nebulized morphine or lignocaine for breathlessness. The reassuring presence of a nebulizer may reduce

respiratory panic, but nebulized drugs vary in effectiveness and require collaboration with the local nebulizer service. Carers can use mechanical or fine manual vibrations over the chest to provide some relief. Patients vary in their response to oxygen. It is usually of no value and simply sets up a barrier between patient and family (Burford and Barton, 1998), but it is worth a trial in those with hypoxaemia (Bruera *et al.*, 1993), so long as a dry mouth is avoided.

Cough

Cough occurs in 30% of people with cancer and 80% of people with lung cancer (Twycross and Lack, 1984). If pulmonary oedema, infection or bronchospasm contribute, they can be dealt with pharmacologically. Smoking cessation will ease the cough but this takes 2–4 weeks. Nebulized lignocaine is useful for a terminal cough caused by pooling of saliva, but may increase the risk of aspiration and sometimes causes bronchospasm (Ahmedzai and Davis, 1997). Excessive secretions can be controlled by inhaled indomethacin (Homma *et al.*, 1999). Physical management depends on whether the cough is productive (Chapter 8).

Nicotine withdrawal

Once smokers are unable to take oral fluids, they are also unable to smoke and may become agitated. Nicotine patches can be applied by carers and can relieve patients' distress (Gallagher, 1998).

Difficulty swallowing

Hyoscine is useful to dry the saliva of people who cannot swallow, delivered by patch, subcutaneous injection or nebulization (Criner, 2000). A speech–language therapist provides support.

Dehydration

> ***When it comes to dying, arms are for hugging not for intravenous infusions.***
>
> Potts, 1994

Some patients may escape symptoms associated with dehydration such as headache, nausea and cramps, but almost all experience thirst (Blower, 1997), and in the late stages when patients can no longer communicate, they may suffer dehydration-related delirium (Bruera, 1998). The best option is often physical assistance to drink, according to the patient's response, using a spoon or feeding cup, with if necessary advice to the patient about when to swallow. This often requires the time and patience of a relative.

If oral fluids cannot be tolerated because of, for example, nausea, dysphagia or bowel obstruction, subcutaneous fluids are often acceptable to patients and manageable at home. Occasional patients prefer rectal hydration (Bruera, 1998). Dehydration symptoms of a dry mouth can be relieved by the measures on p. 202, and a coated tongue with unsweetened pineapple chunks (Reynard, 1997).

Pain

> ***Omitting to attempt to provide cancer pain relief amounts to a type of bodily harm.***
>
> Zenz, 1997

Some 80% of cancer patients experience pain (Bruera, 1997) and over 90% of it can be controlled (Paice, 1998). If pain is allowed to fill the patient's field of consciousness, it can lead to distress, withdrawal, indifference to personal appearance and degeneration of personality.

Drug management for palliation of pain in acute hospitals is often characterized by underestimation of symptoms and overestimation of side effects. Prescribers can be reassured that physical dependence on opioids is rare in terminal care, and not an issue unless medication is withheld. 'Opiophobic' prescribers need to understand that dose requirements vary 1000-fold, and that there is no upper limit (Hanks, 1996). Constipation must be prevented, but other side effects are usually transient. Since the Dr Shipman case in Britain, patients may need reassurance that diamorphine is not a polite way to kill patients.

Other pain-relieving strategies are TENS for localized pain, pain meditations (Levine, 1988), massage (Wilkinson, 1996) and any of the

patient's own strategies such as ointments and hot water bottles. Pellino (1998) found that feeling in control of pain played a larger role in adjustment than belief that cancer could be cured.

Discomfort

Regular turning and positioning to suit each individual eases the discomfort of immobility. Some patients like to be propped up with their head well supported, while others like to be curled up on their side with generous quantities of pillows. Skin needs care.

If movement eases the discomfort of immobility, simple brief exercises may tempt patients who feel that activity is unnecessary. Osteoporosis is common and exacerbated by radiation, chemotherapy, poor nutrition and immobility. In advanced malignancy, there is a risk of bony deposits and the clinician should be alert to any new pain.

Depression

Depression is underrecognized but can usually be identified by the simple question 'Are you depressed?' (Billings, 2000). Whether management is by talking, medication or a combination should be decided by the patient. The majority of patients who express a wish to hasten their death are depressed, often due to feelings of helplessness and being a burden on their family rather than symptoms such as pain (Billings, 2000). This may be manageable by maximizing independence, providing emotional support and/or use of rapid-onset psychostimulants.

Death rattle

People who are too weak to expectorate may collect excessive secretions in their throat. The resulting 'death rattle' is distressing for visitors and neighbouring patients. The noise may ease with repositioning. Secretion formation can be prevented by a hyoscine patch or syringe pump at the first indication of moist breath sounds (Ahmedzai, 1988). Low doses of a tricyclic antidepressant help to dry up mouth and throat secretions. Chest percussion is not indicated and unsafe because of the risk of fracture. Suction is not indicated.

On dying well

> ***All I want to know is that there will be someone there to hold my hand when I need it. I am afraid. Death may be routine to you, but it is new to me. ... I've never died before.***
>
> Gallagher and Trenchar, 1986

This message from a dying student nurse advises her colleagues how they can best help her towards a good death. When patients are free from fears, they can live their remaining life to the full. Conscious dying is possible when a pain-free state without undue sedation has been achieved, so that patients are not trapped between perpetual pain and perpetual somnolence. Death can be a positive achievement when patients are not consumed by anxiety about symptoms, and have stopped fighting for life. Through the many little deaths of dying, they have plumbed the depths of their being, but fear has dissolved, there is peace without defeatism and they are free to look for some meaning in the experience.

Working with dying people is demanding and requires us to be emotionally healthy. It means sharing anguish, absorbing misdirected anger and providing comfort and dignity for people who are totally dependent. It is about emotional involvement, wherein lies its challenge and reward.

CASE STUDY: MS SJ

Identify the problems of this 70-year-old woman with a 48-year history of fatigue and non-specific symptoms.

Background

RMH
Investigated for multiple sclerosis: NAD.
Some depressive symptoms, labelled as 'abnormal illness'.
Barium swallow NAD, awaiting endoscopy.
Migraine with certain foods.
Many other investigations but NAD.
SH
Lives with husband, does not use stairs.
Spends most of the time sitting down.
HPC
Since age 22: overwhelming chronic fatigue.
4–5 years: dysphagia.
2 weeks: ↑ SOB.

Subjective

Fatigue since started work, only ever able to work part-time, worse with stress, everything is a great effort, feels like battery going down.
Tend to drop things.
Difficulty in shops, go dizzy, need someone with me in case I fall, use a stick to keep me steady and to keep people at a distance.
Worse since reading book on relaxation and trying deep breathing exercises.
Always been anxious, e.g. taking the iron with me in the car when I go out to ensure I've not left it on.
Difficulty sleeping.
Aches and pains since teenager, medication unhelpful.
It's like I can feel all my muscles.
Reflexology has helped.
Fed up with hospitals.

Objective

Nervous posture including excess hand movements.
Breathing pattern normal in sitting, tense and rapid in lying.
Sighs before speaking.
Nijmegen score 28.

Questions

1. Analysis?
2. Patient's problems?
3. Goals?
4. Plan?

RMH = relevant medical history; NAD = nothing abnormal discovered.

RESPONSE TO CASE STUDY

1. Analysis

Subjective and objective signs of hyperventilation syndrome. Patient using excess energy to maintain breathing pattern and avoid falling.

2. Problems

Fatigue.
Anxiety.
Poor sleep.

3. Goals

Shop without anxiety.
Visit friends.

4. Plan

- Control breathing.
- Pacing and energy conservation.
- Stairs.
- ↑ exercise tolerance.

POSTSCRIPT TO CASE STUDY

Sequence of progress

1. No change in symptoms, but 'husband says less huffing and puffing'.
2. No change in symptoms, but 'I'm a little more in control of my breathing'.

3. Able to control nightly chest pain with shallow breathing.
4. Improved symptoms.
5. Improved function including stairs.
6. Visiting friends and distant family.

Discharge summary

Some fatigue still present but not preventing activities.
Nijmegen score 12.
Christmas card 9 months later indicated that improvement was maintained.

LITERATURE APPRAISAL

Could the following signs and symptoms indicate anything else?

> ***Signs and symptoms which indicate a need for suctioning include: patient restlessness or anxiety, diaphoresis, increased BP and HR.***
>
> *Accident Emerg. Nurs.* (1997) 5, 92–98

Diaphoresis = sweating.

RESPONSE TO LITERATURE APPRAISAL

There are many possible causes of restlessness, anxiety, sweating and increased vital signs. If not identified and remedied, these could be increased by suctioning.

RECOMMENDED READING

Billings, J. A. (2000) Palliative care. *Br. Med. J.*, 321, 555–558.

Collins, F. (2000) Selecting the most appropriate armchair for patients. *J. Wound Care.*, 9(2), 73–76.

Corner, J. (1996) Non-pharmacological intervention for breathlessness in lung cancer. *Palliat. Med.*, 10, 299–305.

Felhendler, D. and Lisander, B. (1999) Effects of non-invasive stimulation of acupoints on the cardiovascular system. *Comp. Ther. Med.*, 7, 231–234.

Fraser, S. I. (2000) Death – whose decision? *J. Med. Ethics*, 26, 121–125.

Gilbert, C. (1999) Hyperventilation and the body. *Accident Emerg. Nurs.*, 7, 130–140.

Gilbert, C. (1999) Yoga and breathing. *J. Bodywork Mov. Ther.*, 3, 44–54.

Han, J. N. (1996) Influence of breathing therapy on complaints, anxiety and breathing pattern in patients with HVS and anxiety disorders. *J. Psychosom. Res.*, 41, 481–493.

Hayes, R. (1995) Pain assessment in the elderly. **r. J. Nurs.**, 4, 1199–1204.

Molloy, D. W., Guyatt, G. and Russo, R. (2000) Systematic implementation of an advance directive program in nursing homes. *J. A. M. A.*, 283(11), 1437–1444.

Nolan, M. and Nolan, J. (1999) Cardiac rehabilitation following myocardial infarction. *Br. J. Ther. Rehab.*, 60, 142–148.

Paley, C. A. (1997) A way forward for determining optimal aerobic exercise intensity? *Physiotherapy*, 83, 620–624.

Pounsford, J. C. (1997) Nebulisers for the elderly. *Thorax*, 52(suppl. 2), S53–55.

Roberts, D. K., Thorne, S. E. and Pearson, C. (1993) The experience of dyspnea in late-stage cancer. *Cancer Nurs.*, 16, 310–320.

Rowbottom, I. (1992) The physiotherapy management of chronic hyperventilation. *ACPRC Journal*, 21, 9–12.

Tierney, A. J. (1996) Undernutrition and elderly hospital patients. *J. Adv. Nurs.*, 23, 228–236.

Watson, R. (1993) Thirst and dehydration in elderly people. *Elderly Care*, 5(4), 41–44.

Wolf, S. I. (1996) Reducing frailty and falls in older persons: an investigation of Tai Chi. *J. Am. Geriatr. Soc.*, 44, 489–497.

Zimmermann, P. G. (1998) Effective communication with patients with dementia. *J. Emerg. Nurs.*, 24, 412–415.

12 Intensive care, monitoring and support

Summary

Introduction

Patients are admitted to an intensive care unit (ICU) for intensive therapy, intensive monitoring or intensive support. They are not necessarily critically ill, but are at risk of failure of one or more major organs. Their needs range from observation of vital signs after major surgery to total support of physiological systems. Admission usually depends on expectation of recovery.

The importance of rehabilitation in the ICU is emphasized by evidence of the after-effects. Follow-up clinics have revealed:

- impaired mobility, continuing pain, 15% incidence of posttraumatic stress disorder (Waldmann and Gaine, 1996)
- compression neuropathies (Jones and Griffiths, 1998)
- prolonged weakness, fear of falls, panic attacks (Griffiths and Jones, 1999)
- significant anxiety and depression a year after discharge (Shelly, 1998).

A high dependency unit (HDU) is for patients who require support for a single organ system or who need closer monitoring than provided on a general ward (DoH, 1996).

The environment

> *'When the link to life seems tenuous the immediate world is clung to desperately. . . . I had a passionate need to make that corner of the world a home.'*
>
> Moore, 1991, p. 12

Effects on the patient

It is ironic that seriously ill people find themselves in an environment that exacerbates stress. This saps energy and is a major contributor to slowing a patient's recovery (Swann, 1989). The physiological damage inflicted by stress is described on page 25. It is not an optional extra to give attention to this aspect of patient management, but an integral part of physiotherapy.

The severity of the stress response varies with the patient's ability to control the situation. Sensory and sleep deprivation, psychotropic drugs, immobility, isolation, reduced communication and re-regulation of the biological clock have been classified as psychological torture by Amnesty International (Dyer, 1995). These conditions are found in the ICU, albeit without intent. Examples are described below.

1. *Communication problems*: Inability to communicate has been found the most stressful experience in the ICU (Pennock, 1994; Villaire, 1995).
2. *Sleep fragmentation*: Lack of sleep leads to death faster than lack of food (Wood, 1993). A full 90-minute cycle is needed to achieve the restorative benefits of sleep, yet this is rare in the ICU (Wood, 1993). The more ill the patient, the more sleep they need and the less they are likely to get it. Lack of sleep increases morbidity, mortality, confusion (Pilbeam, 1992, p. 260) and reduces respiratory drive (Shneerson, 1996b). Confused patients are more likely to self-extubate (Nuttal, 1998) and are unable to co-operate with physiotherapy. Sleep disruption is caused by noise, interruptions, anxiety, pain, reversal of the day–night cycle, difficulty in finding a comfortable position or fear of falling asleep and not waking again.
3. *Fear*: Patients face previously unmet fears for which they have not developed coping strategies. Fear is compounded by helplessness. Patients try to assess their progress by watching staff and family reactions and comparing themselves to others on the unit. Agitation can lead to myocardial arrhythmias, ischaemia and sometimes infarction (Nuttal, 1998).
4. *Sensory deprivation*: Social isolation, loss of comforting touch, immobilization, certain drugs, a limited visual field and removal of hearing aid or glasses lead to a form of emotional solitary confinement that can leave patients felling intense loneliness despite constant attention. These stress factors are amplified threefold in the absence of windows (Criner and Isaac, 1995).
5. *Sensory overload* (Figure 12.1): Patients find themselves lost in a sea of electronic wizardry, bombarded by unfamiliar beeping, overhead lights, telephones, confining equipment, painful procedures (sometimes without warning), tubes in various orifices and incomprehensible conversation over their heads. Most ICU conversations are between staff rather than with the patient (Wood, 1993). Noise is consistently above internationally recommended levels, leading to physiological damage (Kam, 1994) including hearing loss (Halpern, 1999). A combination of sensory deprivation and overload can cause disorientation, often after the first two or three lucid days. A third of postoperative patients develop delirium (Smith *et al.*, 1997), which more than doubles the risk of death (Nuttal, 1998).
6. *Sensory monotony and loss of time sense*: Patients struggle to keep track of time through a tranquillized haze, which is worsened when there is no day–night sequence in lighting or routine. This compounds disorientation, or, for more alert patients, causes boredom. Boredom is usually a negative experience, but occasionally the empty time gives patients an opportunity for reflection, especially if they have been close to death. Some may emerge with a sharpened perception of what is important in their life.
7. *Discomfort*: Patients experience immobility, gagging on the endotracheal tube, dribbling, sweating, a dry mouth, distended abdomen, unscratchable itches and lack of synchrony with the ventilator. Discomfort is increased with paralysis or other form of restraint. Physical restraints have been found to increase rather than decrease the risk of self-extubation (Taggert, 1994).
8. *Helplessness, dependency and depression*: The less patients are able to do for themselves, the more frustrated they feel. This may become internalized as depression, especially as they are inhibited in expressing feelings

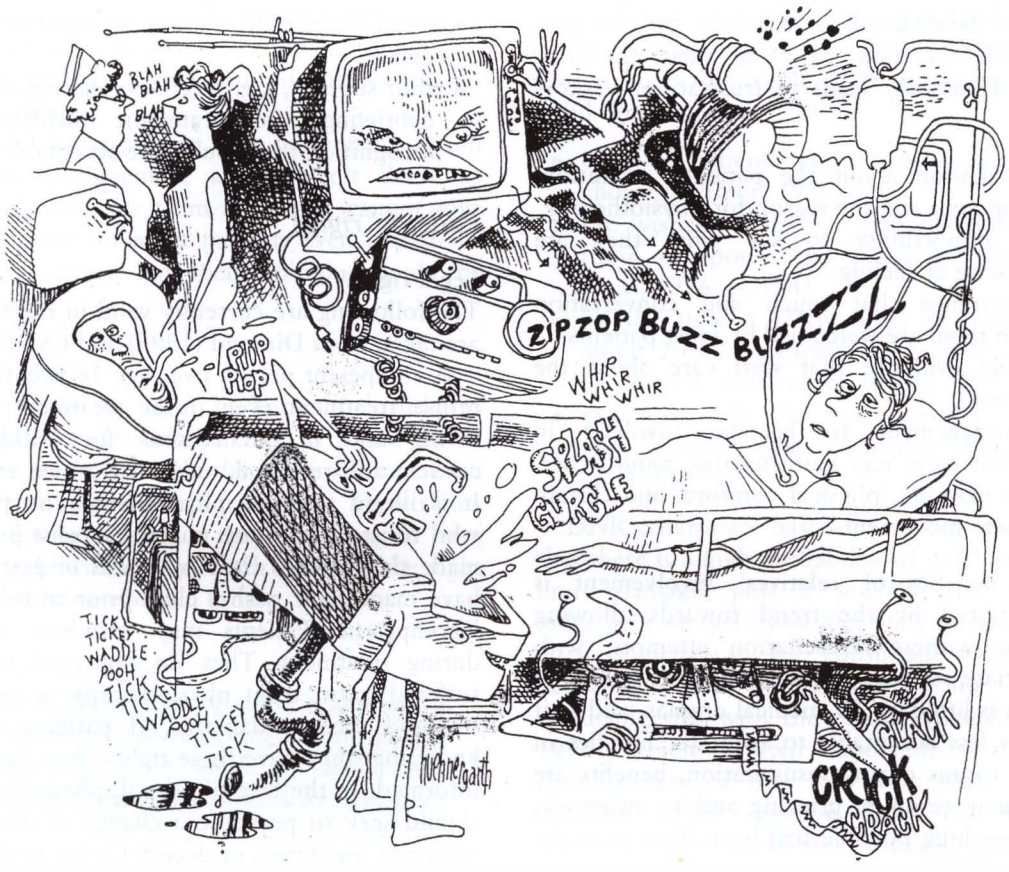


Figure 12.1 Sensory overload. (From Lindenmuth, J. E., Breu, C. S. and Malooley, J. A. (1990) Sensory overload. *Am. J. Nurs.*, **80**, 1456)

when dependent on the goodwill of those who care for them. One patient commented that 'it is the helplessness of illness that is humiliating' (Moore, 1991, p. 12). Depression is particularly apparent in the recovery period and can hinder rehabilitation.

9. *Loss of privacy, dignity and identity*: It is easy for us to forget how people feel when they lose their autonomy, clothes, teeth, personal space and surname. Patients who are elderly or from a different culture are particularly vulnerable to this form of depersonalization. Sometimes patients want privacy from their own relatives, and they should be asked before visitors are ushered in willy-nilly.

> ***He may cry out for rest, peace, dignity, but he will get infusions, transfusions, a heart machine ... he will get a dozen people around the clock, all busily preoccupied with his heart rate, pulse, secretions or excretions, but not with him as a human being.***
>
> Kübler-Ross, 1973

Effects on relatives

Relatives can do much to ease a patient's stress, so long as they in turn are given support. They

may feel bewildered, daunted by the environment and reluctant to voice their concerns. Enforced passivity leads to frustration. Visitors need:

- information about the patient's condition, equipment and the reason for physiotherapy
- the opportunity to say what they are thinking or feeling
- reassurance that touch and conversation from them are welcomed by most patients
- visible evidence that staff care about the patient
- encouragement to become involved in patient care, e.g. hairbrushing, mouth care, foot massage, physical comforts and certain passive movements.

The benefits of relatives' involvement is demonstrated by the trend towards allowing them to witness resuscitation attempts, with appropriate support. As the veil of mystery and heroism is lifted by TV medical dramas, and as it becomes less acceptable to sequester families in waiting rooms during resuscitation, benefits are apparent in terms of grieving and an awareness that everything possible had been done (van der Woning, 1997).

Effects on staff

Emotional responses can become dulled by the frequency with which they are elicited. People working in an ICU need defences against the suffering around them, but these are not incompatible with sensitive patient care.

If we become stressed, we are not only less able to identify with the experience of the patient, but are more likely to make mistakes. Reactions to working in the ICU include overdetachment, anxiety due to the responsibility, frustration at communication difficulties or inability to relieve suffering, and inappropriate joking with other staff which can be misunderstood by patients or relatives. Strategies to reduce staff stress include:

- involvement of all staff in decision-making
- multidisciplinary training to increase confidence
- staff support, e.g. feedback, sharing of ideas, debriefing after traumatic incidents and recognition that doubts are acceptable.

Patients' rights

Legal rights

The following are currently valid in the UK and are taken from Dimond (1999) and CSP (1995).

A competent person over age 16 is entitled to refuse treatment even if the treatment is life-saving and if the reasons for withholding consent are irrational, unknown or non-existent. It is illegal to force physiotherapy on patients who resist or who are unable to resist but have made their wishes clear by words or gesture, or have made their wishes clear prior to becoming incompetent. Patients may withdraw consent during treatment. They do not need to have suffered harm from physiotherapy in order to sue and recover damages. If patients do not know that they have these rights, they should be informed. In the face of refusal, physiotherapists should seek to persuade a change of mind, but must not use duress or deceit. Giving inadequate information can lead to litigation (Bury and Mead, 1998, p. 32). The following allow treatment without consent:

- common law power to act out of necessity
- statutory authorization, e.g. Mental Health Act 1983
- patient incompetence.

A patient is considered incompetent to give or withhold consent if s/he is incapable of one of the following:

- comprehending and retaining treatment information
- believing such information
- weighing such information and arriving at a choice.

Panic, indecisiveness, irrationality and mental illness in themselves do not amount to incompetence. However, if mental illness renders the

patient incapable of understanding or retaining information so that s/he is unable to make a decision and assess risks, this constitutes incapacity to consent.

Refusal of treatment and subsequent action must be documented, and difficult decisions discussed with the team. Relatives cannot give valid consent for adult patients even if patients are unconscious, but their opinion should be considered.

Moral rights

Patients have the right to know the truth, to participate in decision-making, to refuse to be used for teaching, and to be given full care even when their choice differs from ours. Their rights should not be violated if they are young or have learning difficulties. In North America the right to the truth is enshrined in law.

End-of-life decisions

> ***Ethics is the exercise of moral reasoning in circumstances where strong feeling is not always the surest guide to action nor procedural powers the surest way to justice.***
>
> Dunstan, quoted by Branthwaite, 1996

Making a choice between undesirable alternatives is the task facing those who make end-of-life decisions. Unfortunately few patients at present get the opportunity to discuss this in advance of a situation when treatment may be extending death rather than prolonging life (Hofman *et al.*, 1997). When this has not been discussed early enough, discontinuation of treatment is considered by the relevant team members, with the consultant taking the final decision. The views of the family are considered, but if they are burdened with taking the final decision, it can leave lasting guilt (Phelan, 1995).

Advance directives, or living wills, allow individuals, when competent, to express a wish to be spared life-sustaining treatment in case of intractable or terminal illness. These are not legally binding in many countries, and are often not available, retrieved or honoured during acute hospital care. The British Medical Association directs that 'do not resuscitate' (DNR) orders be made in consultation with the patient unless this is impossible. This should be done in good time because:

- 85% of patients prefer to be offered the choice (Heffner, 1996)
- the wishes of 40% of patients differ from those of spouse and physician (Bach and Haas, 1996, p. 184)
- by the time a DNR order is made, only about 20% of patients are capable of being involved in the decision (Ryan, 1998)
- most patients do not consider advance consultation to be insensitive (Kerridge *et al.*, 1998).

DNR criteria are based on medical judgement that there is a high probability of death or severe brain damage, plus if possible the patient's judgement on quality of life. Only in exceptional circumstances can the consultant make DNR decisions based on quality of life without discussing this with the patient (Blackie *et al.*, 1999), which is often impossible in the ICU. Patients may or may not want their families involved in the decision. DNR decisions are reviewed at intervals and documented in the medical and nursing notes. DNR status does not preclude physiotherapy, or in some cases, surgery (Clemency, 1997).

A decision to withdraw mechanical ventilation is followed by 'terminal weaning', which is weaning in the knowledge that it will be followed by death. This should be accompanied by titrated narcotics and non-invasive monitoring. Extubation should be avoided if this could cause respiratory distress (Krishna, 1999).

Teamwork

Interpersonal factors are the main causes of stress in high-dependency areas (Biley, 1989), and poor communication is the chief cause of errors (Gosbee, 1998). Teamwork is enhanced by mutual respect and assertiveness, mutual teaching and learning, shared coffee breaks, flexibility and above all good communication.

Problems may arise over boundaries and

autonomy. If physiotherapists would like to ask for review of a medical therapy that is not their direct responsibility, they can raise the subject diplomatically by asking for advice about it, or by making the link between medical management and rehabilitation. If physiotherapy is medically prescribed, physiotherapists can thank the doctor for his/her advice and clarify that the patient will be assessed and treated as appropriate. Results are likely to be positive when communicating in a way that makes it easy for others to agree.

Communication between physiotherapists and nurses is facilitated by the physiotherapist offering to help change sheets when it fits in with turning the patient during treatment, and the nurse incorporating regimes such as hourly incentive spirometry into the nursing plan. Turning for physiotherapy should be co-ordinated with turning for pressure area care.

Infection control

Hospitals are curious places and ICUs even curiouser. Immunocompromised patients are crowded together and bombarded with ICU-hardened bacteria which flourish in the invasive machinery. Widespread broad-spectrum antibiotics are then added, which encourages superinfection by resistant organisms. Loss of upper airway defences in mechanically ventilated patients leaves them vulnerable to colonization. Cross-infection by the hands of staff contributes to 30% of pathogens (Weinstein, 1991). ICU patients are 5 to 10 times more likely than other patients to acquire nosocomial infection (Weber *et al.*, 1999), and overall hospital-acquired infections cost the NHS £1 billion a year (Rennie, 2000). Measures to prevent infection include:

- most importantly, handwashing or glove-changing between patients (Rossoff, 1995)
- avoidance of hot-air hand-dryers which leave hands still moist (Gould, 1994)
- plastic aprons, colour-coded to ensure they are changed between patients (Gill and Slater, 1991)
- removal of watches and avoidance of long sleeves (Singer and Webb, 1997, p. 78)
- fastidious attention to sterile suction technique, including resting the disconnected catheter-mount on the glove paper to avoid touching the sheets
- care of tracheostomies as the surgical wounds that they are
- minimizing the time that patients spend supine, which increases the risk of aspiration of gastric contents (Torres, 1992)
- avoidance of frequent changes of ventilator tubing (Fink *et al.*, 1998), tracheal tubes (Feldman *et al.*, 1999) or pulmonary artery catheters (Saint and Matthay, 1998).

The methicillin-resistant staphylococcus aureus (MRSA) bacterium is often found on the skin of the general population, but MRSA and other antibiotic-resistant bugs create havoc in hospitals, where they are easily spread by staff hands to compromised hosts.

MONITORING

> ***'Frankly it feels quite awful to be connected to machines through every available orifice, plus several new medically made ones, in spite of feeling thankful for all the life-sustaining help and healing ministrations.'***
>
> Brooks, 1990

From the patient's point of view, monitors bring both anxiety and reassurance. From the staff point of view, they are useful to record sudden or subtle changes in a patient's status. They are complementary to clinical observation and not a substitute. Monitoring differs from measuring: it implies regular observation and a systematic response if there is deviation from a specified range.

Ventilator interactions

Ventilator graphics demonstrate flows, pressures and volumes that represent the patient's response to the ventilator. Details are given in Pilbeam (1998, p. 42) or the manufacturer's handbook. Below is an outline.

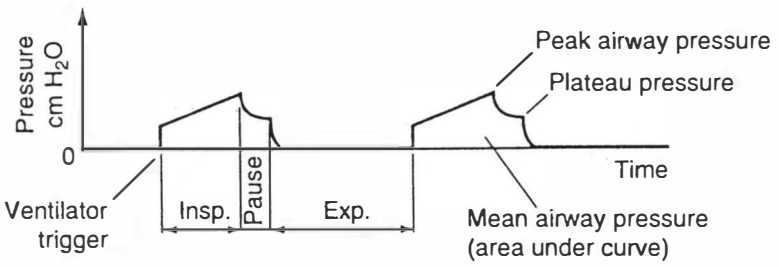


Figure 12.2 Pressure–time curve representing controlled mandatory ventilation.

Pressure–time curve

Airway pressures throughout the respiratory cycle are shown in Figure 12.2. *Peak airway pressure* is equivalent to peak inspiratory pressure. *Mean airway pressure* is associated positively with oxygenation and negatively with the haemodynamic side effects of intermittent positive pressure ventilation (IPPV). End-inspiratory *plateau pressure* reflects peak alveolar pressure, and is kept below 30–40 cmH_2O if possible to reduce the risk of lung injury (MacIntyre, 1996).

Flow–time curve

The flow–time curve is useful to verify the presence of intrinsic PEEP, as shown by inadequate expiratory time, and the effect of bronchodilators in a patient with reversible airways obstruction (Figure 12.3).

Pressure–volume loop

Lung compliance is represented by the pressure–volume loop (Figure 12.4), which is related to the pressure–volume curve (p. 6). Spontaneous inspiration is shown by negative pressure to the left, the area of which represents the patient's work of breathing. The work done by the ventilator is shown by positive pressure plotted to the right, the area becoming larger when the ventilator has to work harder. Work of breathing (WOB) can be calculated from this loop (Banner *et al.*, 1996).

Flow–volume loop

Figure 12.5 shows a flow-volume loop, which is

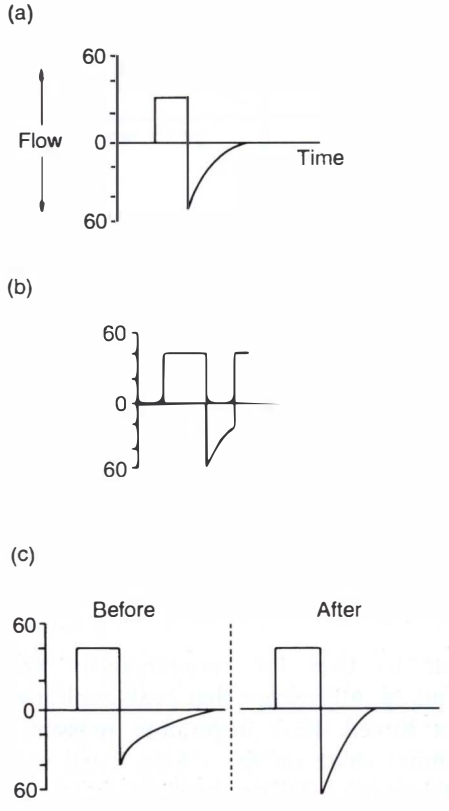


Figure 12.3 Flow–time curve. (a) Normal: inspiratory flow above the *x* axis and expiratory flow below. (b) Intrinsic PEEP: expiratory flow unable to return to zero before the next inspiration begins. (c) Before and after bronchodilator: prolonged and normal expiratory flow.

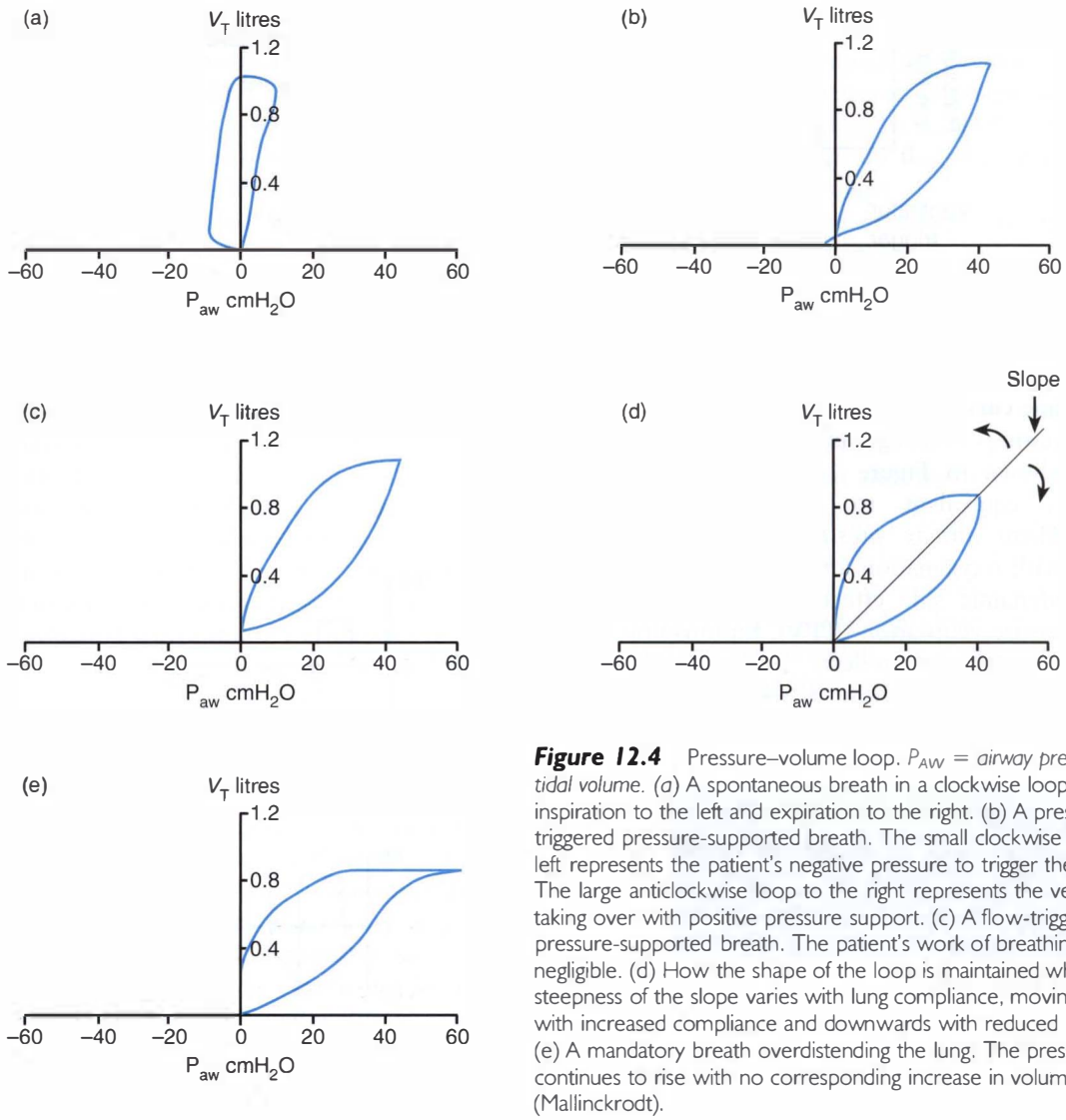


Figure 12.4 Pressure–volume loop. P_{AW} = *airway pressure;* V_T = *tidal volume.* (*a*) A spontaneous breath in a clockwise loop, with inspiration to the left and expiration to the right. (b) A pressure-triggered pressure-supported breath. The small clockwise loop to the left represents the patient's negative pressure to trigger the breath. The large anticlockwise loop to the right represents the ventilator taking over with positive pressure support. (c) A flow-triggered pressure-supported breath. The patient's work of breathing is negligible. (d) How the shape of the loop is maintained while the steepness of the slope varies with lung compliance, moving upwards with increased compliance and downwards with reduced compliance. (e) A mandatory breath overdistending the lung. The pressure continues to rise with no corresponding increase in volume (Mallinckrodt).

similar to that for spontaneously breathing patients (p. 60) except that peak expiratory flow is not forced. Peak inspiratory pressure is the maximum value on the *x* axis. Tidal volume is the maximum value on the *y* axis.

Gas exchange

Arterial oxygen gases

Arterial blood samples from an indwelling arterial catheter are subject to spontaneous variability, and patients should be undisturbed for 20 minutes before each measurement and stay in the same position. These conditions are not always achieved in the hurly-burly of the ICU.

Arterial oxygen saturation

Oximetry is the physiotherapist's friend. It gives instant feedback on arterial oxygen saturation. The different absorption of light by saturated and unsaturated haemoglobin is detected by the

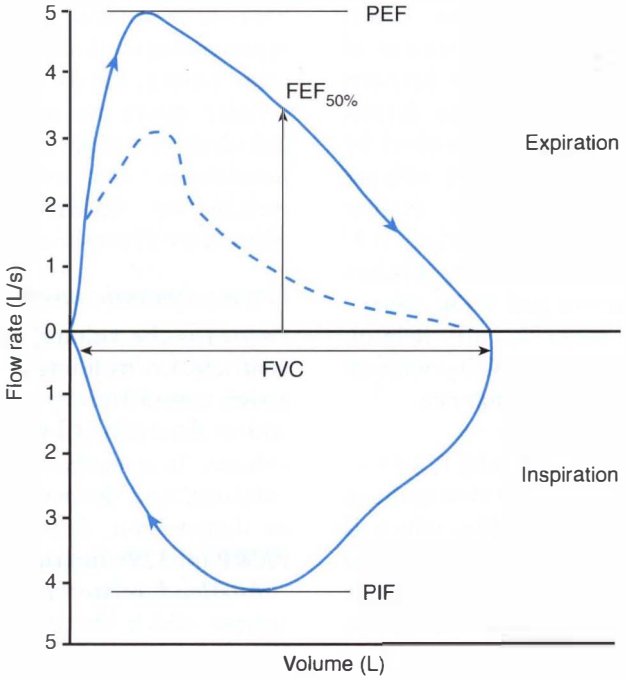


Figure 12.5 Flow–volume loop. The scooped-out dashed line of the expiratory curve indicates obstructed airways. *PEF = peak expiratory flow (peak flow); $FEF_{50\%}$ = forced mid-expiratory flow, FVC = forced vital capacity; PIF = peak inspiratory flow.*

oximeter, which continuously displays the percentage of haemoglobin that is saturated with oxygen. A sensor is attached close to a pulsating arteriolar bed such as the ear, finger or toe, with its position changed regularly to protect the skin.

A level of 92% indicates adequate arterial oxygenation (Komara and Stoller, 1995). S_aO_2 below 90% corresponds to a P_aO_2 of less than 8 kPa (60 mmHg) under normal conditions, and obliges patients to increase their cardiac output to maintain oxygen delivery (Ahrens, 1999b). During severe or rapid desaturation, the oxygen dissociation curve is shifted to the right and a below-normal S_aO_2 is recorded. S_aO_2 is always interpreted in relation to the F_IO_2.

Oximetry is less accurate than arterial blood gas analysis, but is acceptable at values above 75%. S_aO_2 is dependent on perfusion, and accuracy is compromised by cold peripheries, vasopressor drugs, hypotension, hypovolaemia, hypothermia, peripheral vascular disease and anaemia. In low perfusion states, a finger probe is advised (Goodfellow, 1997), preferably one of the middle two fingers. The sensors are affected by movement, nicotine stains, bruising, clubbing, jaundice, nail polish or, for an ear probe, pierced ears. Carbon monoxide poisoning falsely elevates S_aO_2 (Hampson, 1998).

Physiotherapy can upset gas exchange, and if desaturation occurs, treatment should normally stop and the '100% oxygen' knob on the ventilator activated if appropriate. If S_aO_2 does not return to its baseline value within minutes, remedial action should be taken such as repositioning the patient, increasing F_IO_2 in consultation with the nurse or manual hyperventilation (p. 373).

Capnography

A capnograph displays expired CO_2 concentra-

tion as a waveform called a capnogram, which provides continuous non-invasive assessment of the adequacy of ventilation. A sensor between the tracheal tube and ventilator tubing detects the amount of infrared radiation absorbed by expired CO_2. Values at end-exhalation indicate 'end-tidal CO_2', which approximates alveolar PCO_2 unless there is $\dot{V}_A/\dot{Q}$ mismatch. Many ICU patients have $\dot{V}_A/\dot{Q}$ mismatch, and CO_2 production also varies with nutrition and WOB (Napolitano, 1999), but the trend is still helpful. Normal value is 1 mmHg below P_aCO_2, with an acceptable range up to 5 mmHg difference.

Transcutaneous monitoring ($P_{tc}O_2$ and $P_{tc}CO_2$)
Oxygen and CO_2 diffuse across the skin and can be measured by a sensor on the skin, which is heated to increase gas permeability across the skin barrier. In haemodynamically stable patients, values relate to respiratory status, but measurements vary with cardiac output, skin metabolism or capillary blood flow, and values are thought to reflect tissue gas tension rather than arterial gas tension. Skin burns are avoided by rotating sites every 4 hours and incorporating a temperature alarm.

Neonates show a correlation between $P_{tc}O_2$ and arterial oxygenation, and accuracy is greater than with end-tidal CO_2 (Tobias, 1997). Adults have varying skin thicknesses and results are less reliable. $P_{tc}O_2$ is at least 10% below P_aO_2, and $P_{tc}CO_2$ is similarly higher than P_aCO_2, but mathematical correction can neutralize the error (Rosner *et al.*, 1999).

Tidal volume

If tidal volume is not continuously monitored and displayed, it can be measured by attaching a Wright spirometer to the tracheal tube and taking the average of 5 breaths.

Fluid status

The fluid balance chart gives an overview of fluid status. Fluids in the intravascular space affect pulse, BP, cardiac output, left and right atrial pressures (p. 327, 9), the difference between peripheral and central temperature and, representing the kidney's sensitivity to perfusion, urine output. Fluids in the interstitial and intracellular spaces are more difficult to assess, but dehydration is suggested by thirst and dry mucus membranes, and overhydration may increase weight or oedema, either peripheral or pulmonary (Twigley and Hillman, 1985).

Haemodynamic monitoring

Preload is the volume of blood returning to the ventricle, i.e. its filling pressure at end-diastole. It assists contraction by stretching the myocardium and is determined by venous return and blood volume. It is increased in heart failure or fluid overload, and decreased in hypovolaemic shock or dehydration. Left preload is monitored by PAWP (p. 329) and right by CVP (p. 327).

Afterload relates to the amount of pressure against which the ventricle must work during systole, as if opening a door against a wind. It is increased with systemic/pulmonary hypertension, peripheral vasoconstriction or aortic/pulmonary valve disease, and decreased with vasodilation, e.g. in septic or neurogenic shock. Left ventricular afterload is reflected by systolic BP and systemic vascular resistance. Right ventricular afterload is reflected by pulmonary artery pressure and pulmonary vascular resistance.

The heart and vascular systems act as a continuous loop in which constantly shifting pressure gradients keep the blood moving. In many patients, cardiovascular function can be gauged from clinical signs such as BP, HR, urine output and mental status. However these may be unreliable in critical illness and invasive haemodynamic monitoring is then required.

Blood pressure
BP can be measured by an automated cuff that intermittently compresses the limb and senses arterial pulsations. Continuous monitoring of BP by an indwelling catheter gives a beat-to-beat waveform display and provides more accurate and instant feedback than cuff pressure. The most relevant reading is mean arterial pressure,

representing the perfusion pressure over the cardiac cycle.

Right atrial pressure

The *central venous pressure* is monitored by creating an extension of the patient's vascular system via a central line and measuring the pressure within it by a transducer, a device that converts pressures to electrical signals, or a water manometer (Figure 12.6). A radio-opaque catheter is passed through a large central neck or arm vein until it is just outside the right atrium, through which all venous blood passes. The central venous pressure (CVP) within this system is equivalent to the right atrial pressure (RAP), which reflects the preload of the right ventricle.

The CVP (i.e. RAP) indicates circulating blood volume and the ability of the heart to handle that volume. It is affected by the interaction between blood volume, right heart function, peripheral venous tone and posture. CVP is equivalent to JVP (p. 34).

Normal values are 3–12 cm H_2O (measured by manometer) or 0–8 mm Hg (by transducer). Single values are less relevant than the trend, but a high value might indicate heart failure, pulmonary embolus, COPD, pneumothorax or over-transfusion of fluid. The CVP provides early warning of cardiac tamponade (p. 384), which causes a sudden rise in CVP, or haemorrhage, which causes a sudden drop. CVP is more sensitive to haemorrhage than BP, because BP is maintained for longer by vasoconstriction.

Dynamic CVP measurements are used to assess fluid status if the cardiac status is stable. A fluid challenge of 50–200 mL colloid is infused over 10 minutes, and a rise in CVP of 3 mmHg indicates an adequate circulating volume (Singer and Webb, 1997, p. 262). It it does not rise significantly, the patient is relatively hypovolaemic.

Multiple functions are serviced by multilumen catheters. Central venous catheterization is now a routine procedure used not just for CVP measurements but also for infusing fluids, drugs, blood and hyperosmolar feeds. These thick feeds are required for patients who need nutrition without too much fluid volume, which cannot be given through peripheral veins. However, one study found that central venous lines create a 64-fold higher risk of catheter-related sepsis than peripheral lines (Collignon, 1994). Implications for physiotherapy are the following:

- Cannulation of a large vein near the pleura may cause a pneumothorax, haemothorax or surgical emphysema. After placement of a central line, the X-ray should be examined before any positive pressure treatment such as manual hyperinflation.
- The supine position is often used for measurement because the transducer must be level with a zero reference point. If the patient is not repositioned afterwards, prophylactic chest care is compromised. Despite the tradition of supine measurement, Wilson (1996) shows that readings are accurate when sitting up, and claims that supine is illogical because in this position intrathoracic pressure is sensitive to pressure from abdominal contents.
- A high CVP may indicate pulmonary oedema, which impairs gas exchange. A low CVP may indicate hypovolaemia, which can lead to adverse haemodynamic response to manual hyperinflation.

The CVP directly measures right atrial pressure but usually reflects filling pressures for both sides of the heart. However, left atrial pressure may need to be measured separately for two reasons:

- it may take 24–48 hours for the CVP to rise in response to left ventricular failure because the pressure has to back up through the pulmonary circulation and the right ventricle may initially compensate
- CVP does not reflect left atrial pressure if the compliance of either ventricle is affected by septic shock, ischaemia, vasopressors or vasodilators
- CVP does not reflect left atrial pressure if

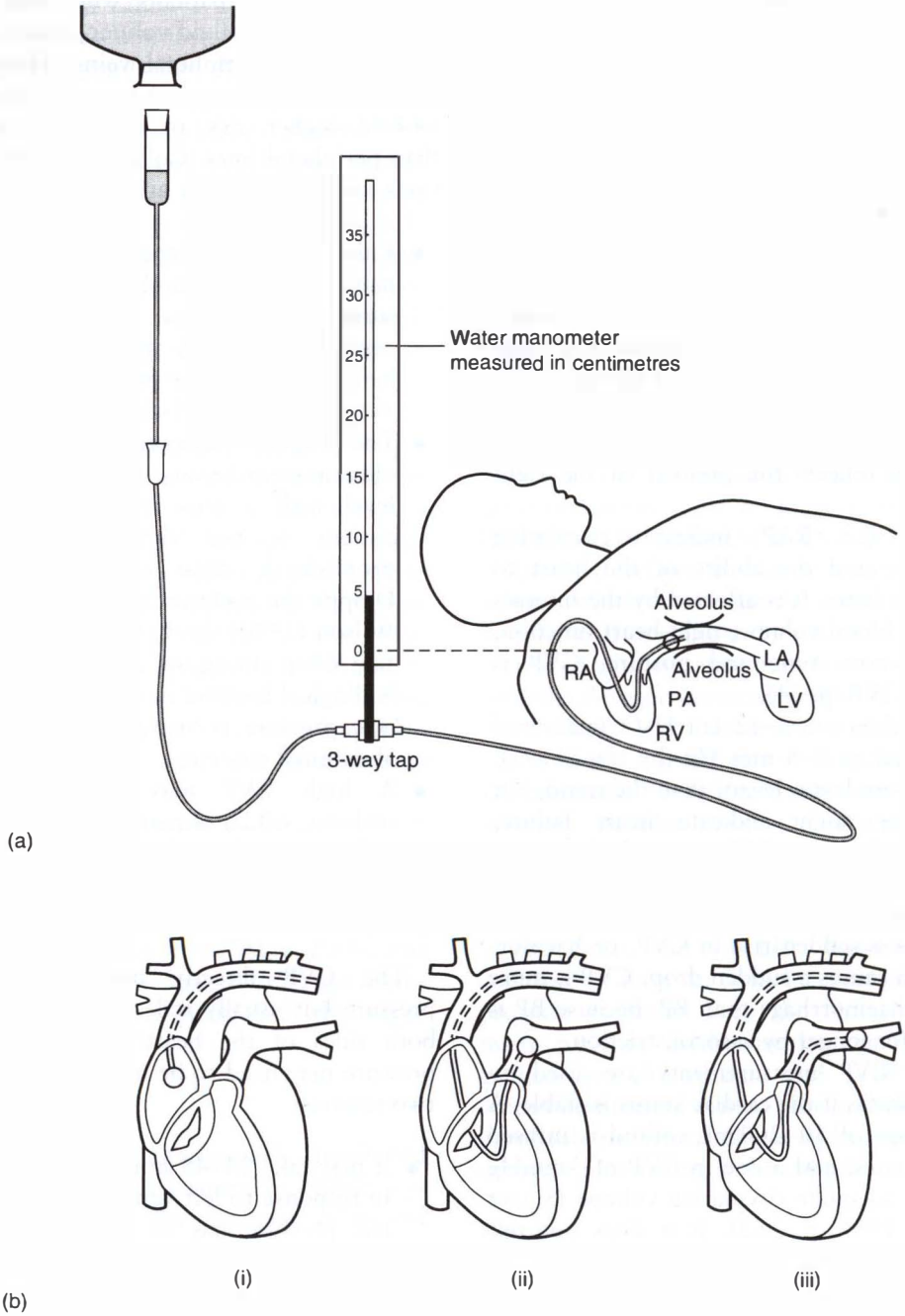


Figure 12.6 (a) Haemodynamic monitoring using a manometer. A multilumen catheter monitors CVP from the right atrium, PAP from the pulmonary artery, and PAWP from the pulmonary vasculature. Zero point on the manometer is at the level of the right atrium. The CVP reading is 5 cmH_2O. *RA = right atrium; RV = right ventricle; PA = pulmonary artery; LA = left atrium; LV = left ventricle.* (b) Passage of catheter as it measures (i) CVP, (ii) PAP, (iii) PAWP.

pulmonary hypertension pushes up the CVP even when the patient is systemically hypovolaemic.

Left atrial pressure

If left atrial pressure measurement is required, a pulmonary artery catheter incorporating three to five lumens is used. Sometimes called a Swan–Ganz catheter, it is passed along the CVP catheter route, then floated through the right ventricle into the pulmonary artery, drawn by an inflated balloon at its tip (Figure 12.6). The pulmonary artery catheter monitors cardiac output (p. 330) and the two following vascular pressures.

Mean *pulmonary artery pressure* (PAP) reflects the pressure that needs to be generated by the right ventricle to pump blood through the pulmonary vasculature. Normal values are 10–20 mmHg. A raised PAP indicates pulmonary hypertension, pulmonary embolism or fluid overload. People with advanced COPD show a PAP of up to 40 mmHg, rising further during sleep and exercise.

The catheter can then be carried further by the flow of blood until it wedges in a branch of the pulmonary vasculature and occludes it. The catheter tip is isolated from pressure fluctuations in the right side of the heart and is in direct communication with the left atrium via the pulmonary capillary bed, so long as there is a continuous column of blood between the two. The pressure monitored at this point is the *pulmonary artery wedge pressure* (PAWP) or left atrial pressure. It reflects pressure in the left ventricle via the left atrium and lung vasculature. The balloon acts as a form of pulmonary embolus so is deflated between measurements to prevent ischaemia.

The continuous column of blood in the pulmonary vascular bed is tenuous if the catheter is in the upper zone (Zone 1, p. 10) of the lungs where there is no perfusion under the positive pressure of IPPV. Measurements are compromised if the patient is severely hypovolaemic, changes position, or is subject to high lung inflation pressures (Brandstetter *et al.*, 1998). Values are also affected by valve stenoses, floppy ventricles following serial myocardial infarcts or sepsis, or stiff ventricles following sympathetic stimulation caused by hypovolaemic shock. The more ill the patient, the less accurate are single measurements, but the trend is helpful.

PAWP shows left ventricular changes more promptly than CVP and gives a more accurate indication of fluid status because it is affected by fewer variables. This allows fine tuning when establishing optimum PEEP (p. 353), helps to rationalize fluid and drug therapy and distinguishes between hypovolaemia (↓ PAWP) and left ventricular failure (↑ PAWP). Normal values are 5–15 mmHg. Implications for physiotherapy are similar to those for CVP readings.

PAWP is also known as pulmonary artery occlusion pressure, pulmonary capillary wedge pressure or, on ward rounds, simply wedge pressure. The glamour of this expensive technology has led at times to misuse of a system that demonstrates a 24% complication rate (Cooper, 1996), including thrombosis, sepsis, arrhythmias, trauma to the delicate pulmonary vessels (resulting in bloodstained secretions) and pulmonary ischaemia or infarction. Its use is best reserved for haemodynamically unstable patients who are refractory to medical treatment after scrupulous assessment.

Tissue oxygenation

The efficiency of a bus journey is best measured when it arrives at its destination rather than when it leaves the garage. Similarly, oxygen delivery to the tissues is more relevant than oxygen in arterial blood.

Mixed venous oxygenation

Oxygen levels in the pulmonary artery indicate the extent to which oxygen supply (cardiac output, haemoglobin, S_aO_2) has met demand (oxygen extraction at tissue level). Both haemodynamic and gas exchange components of the oxygen cascade can be monitored in the pulmonary artery.

Mixed venous blood in the pulmonary artery comprises individual streams from a multitude of

capillary beds which have been mixed in the right ventricle before returning to the lungs. The oxygen in this pooled blood is what is left after its journey, and reflects events anywhere from alveoli to mitochondria. It is especially useful in identifying problems at tissue perfusion and extraction level, beyond the reach of arterial blood gas measurements.

The mixed venous oxygen saturation of haemoglobin in pulmonary artery blood ($S\bar{v}O_2$) is on average 65–75%, and should be more than 10% below S_aO_2. A low $S\bar{v}O_2$ reflects:

- ↓ oxygen delivery, e.g. suction, anaemia, low cardiac output, hypoxaemia, haemorrhage
- ↑ oxygen demand, e.g. suction, exercise, pain, fever, anxiety, agitation, laboured breathing or hypermetabolic states.

$S\bar{v}O_2$ does not pinpoint which of the variables is responsible for any change, and acts more as an early warning system to advise on further investigation. Cardiac output is simultaneously monitored so that it can be distinguished from other variables.

Values below 50% are normally associated with anaerobic metabolism, and values below 40% are incompatible with life following myocardial infarction (Edwards, 1997). People with chronic heart failure are more tolerant of low levels. $S\bar{v}O_2$ can be improved by increasing F_IO_2 or cardiac output, reducing stress or addressing other relevant factors. Excessively high values above 85% indicate 'luxury perfusion', in which oxygen cannot be extracted by tissues that have been damaged by global ischaemia caused by, for example, hypothermia or severe sepsis.

During physiotherapy, if $S\bar{v}O_2$ varies by more than 10% from the baseline for more than 3 minutes, treatment should be stopped (Hayden, 1993). If it has not recovered 3 minutes after suctioning, extra F_IO_2 is required.

Gastric tonometry

The gut is the crystal ball of tissue hypoxia. It provides advance warning because it can be starved of oxygen when other tissues are well supplied. Its susceptibility to hypoperfusion is because of:

- the mucosa's high metabolic demand
- its tendency to vasoconstrict because of a rich innervation by sympathetic nerves
- the intense ischaemia to which the narrow villus tips are prone.

Gastric tonometry entails passing a saline-filled balloon into the stomach and measuring the PCO_2 that passes across the membrane. Gastric mucosal pH can also be measured directly, or a fibreoptic sensor can be used (Knichwitz *et al.*, 1998). Acidosis indicates hypoperfusion, which if not corrected may contribute to multisystem failure (Ruffolo, 1998).

The splanchnic circulation is the largest regional circulation, containing 25–40% of systemic blood volume (Ruffolo, 1998; Grounds, 1997). Its vulnerability is demonstrated by the following:

- A 20% reduction in systemic blood flow reduces gut blood flow by an average 55%.
- 20% hypovolaemia causes a 60% reduction in gastric blood flow (Ricour, 1989).

Cardiac output (CO)

Invasive measurement of CO is by thermodilution. A known quantity of a cold sterile solution is rapidly injected into a channel of the pulmonary artery catheter, which exits into a central vein near the right atrium. The temperature of blood when it reaches the pulmonary artery indicates the speed with which the solution has been warmed, providing a measurement of cardiac output (Figure 12.7). CO can also be assessed non-invasively by transoesophageal Doppler ultrasound to measure aortic blood flow (Baillard *et al.*, 1999). $S\bar{v}O_2$ can be a surrogate for CO if oxygen consumption is stable. Reduced urine output is the simplest indicator of reduced CO.

Cardiac output usually reflects BP but they do not always change in harmony. If the myocardium is poorly contractile, peripheral vasoconstriction may maintain BP in the face of falling

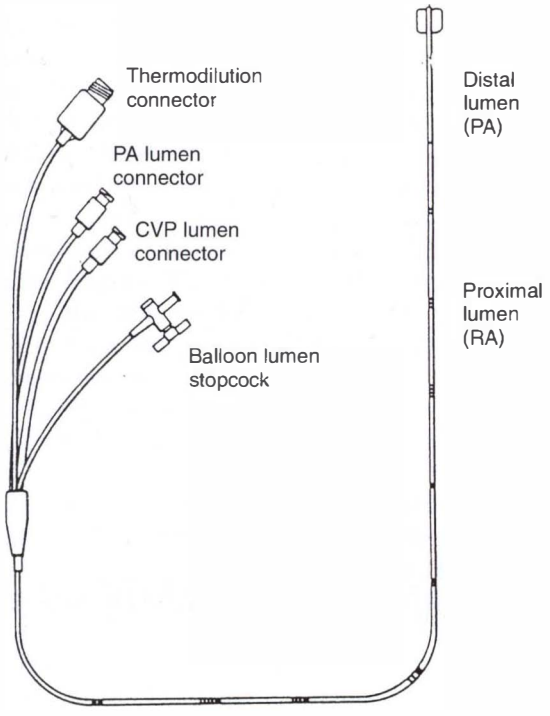


Figure 12.7 Four-lumen pulmonary artery catheter. *CVP = central venous pressure; PA = pulmonary artery; RA = right atrium.*

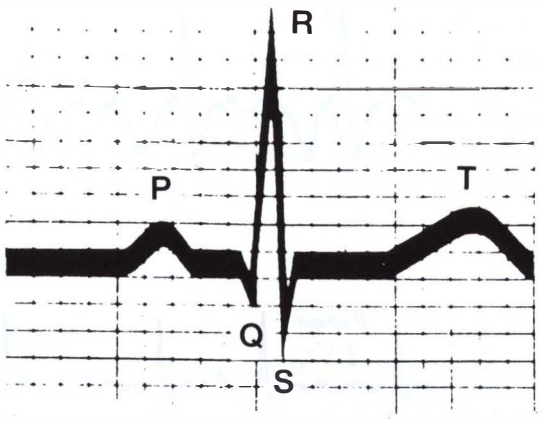


Figure 12.8 Normal ECG trace of one heart beat. *P wave = atrial depolarization; PR interval = atrioventricular conduction time; Q = ventricular depolarization; R = first positive deflection during ventricular depolarization, S = first negative deflection during ventricular depolarization, QRS complex = total ventricular depolarization, T = ventricular repolarization (recovery period) in preparation for next cycle.*

CO. A septic patient in a hyperdynamic state may have a high CO, but vasodilation will drop the BP. Accurate measurements require the patient to be in a steady state.

Electrocardiography (ECG)

Disturbances such as hypoxia, physiotherapy, electrolyte imbalance, myocardial ischaemia or anxiety can cause disorders of heart rate (HR) or rhythm. The effects are significant if they affect cardiac output. They are picked up on the ECG, which is a recording of electrical activity in the heart comprising waves, complexes and intervals.

Sinus rhythm is normal rhythm originating from the sinoatrial (SA) node (Figure 12.8). Supraventricular arrhythmias originate from above or in the atrioventricular (AV) node and are known as atrial and nodal arrhythmias respectively. *Sinus tachycardia* or *supraventricular tachycardia* is HR over 100 bpm, recognized by rapid rate, regular rhythm and normal QRS complex. Causes include sympathetic activity, electrolyte imbalance or excess β_2-agonist medication. Cardiac output is rarely compromised. *Sinus bradycardia* is HR under 60 bpm with normal rhythm.

Ventricular tachycardia is distinguished from supraventricular tachycardia by a lost P wave and broad and bizarre QRS complex. It usually impairs cardiac output, BP and tissue perfusion, and can lead to pulmonary oedema or ventricular fibrillation.

Nodal rhythm occurs when the AV node takes over from a non-functioning or slow SA node. This causes lost P waves and a variable or absent PR interval. Cardiac output may fall because atrial contraction is out of synchrony with the ventricle, which loses its 'atrial kick'.

The SA node is the natural pacemaker, but if it does not initiate an impulse at correct intervals, an ectopic (abnormal) focus outside the SA node may take the initiative. These

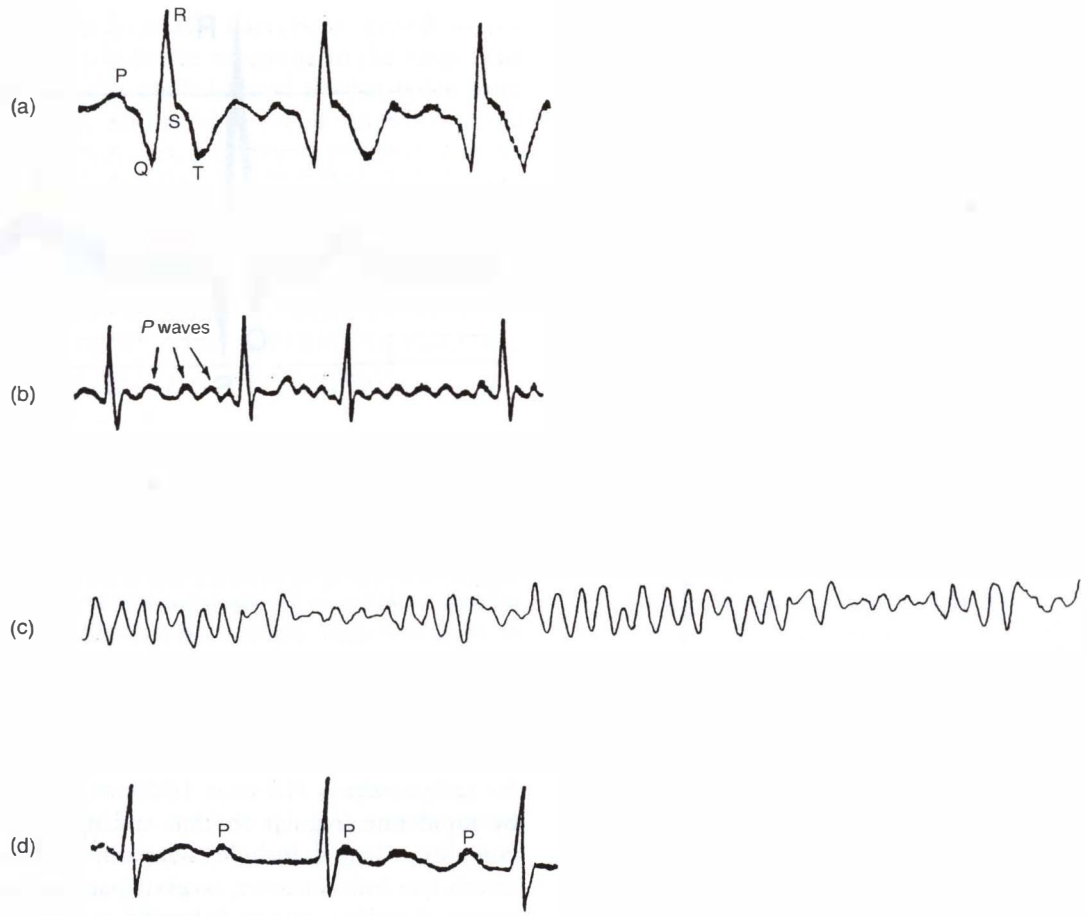


Figure 12.9 ECG traces indicating (a) myocardial infarction, (b) atrial fibrillation, (c) ventricular fibrillation, (d) complete (third-degree) heart block.

ectopic beats are seen as premature beats followed by a compensatory pause, sometimes felt as missed heart beats by the patient. They are common and do not contraindicate physiotherapy unless they increase in number or cause haemodynamic disturbance. However, they may signal the onset of a more serious arrhythmia.

Atrial ectopics manifest as occasional abnormal P waves or an early normal beat, and are of little significance unless frequent. *Nodal ectopics* are the main cause of nodal rhythms. *Ventricular ectopics* are caused by an irritable focus in the ventricle, producing an absent P wave, wide and wayward QRS complex and inverted T wave. They occur following heart surgery or myocardial infarction (Figure 12.9a), in smokers or in those suffering from hypoxia or low potassium levels. *Bigeminy* means that every other heart beat is ectopic and *trigeminy* means that every third beat is ectopic.

ST segment elevation suggests pericarditis, coronary artery spasm or acute myocardial infarction (MI) which will respond to thrombolytic drugs. ST segment depression (Figure 12.9a) indicates myocardial ischaemia or infarction that does not respond to thrombolytic therapy.

Atrial fibrillation (AF) occurs when ectopic foci throughout the atria discharge too fast for

the atrial muscle to respond other than by disorganized twitching out of sequence with ventricular activity. It appears as a rapid rate, irregular rhythm and the replacing of P waves with a chaotic baseline (Figure 12.9b). It can be worsened by sympathetic stimulation, hypoxia, hypokalaemia, over- or underhydration, pulmonary embolism, myocardial ischaemia or heart surgery. The ventricles lose their support from the atria and may be unable to sustain normal cardiac output. Patients may have no symptoms or suffer palpitations, dyspnoea, fatigue or stroke. Treatment is by anti-arrhythmic drugs or cardioversion by DC shock. Slow AF does not necessarily contraindicate physiotherapy. AF is a common arrhythmia in the general population, affecting 4% of people over 70 years (Kamalvand and Sulke, 1999) due to heart failure or advanced age.

Atrial flutter is less common than AF and short-lived. It causes regular sawtooth undulations on the ECG and either deteriorates to AF or spontaneously recovers.

Ventricular fibrillation (VF) is the commonest cause of cardiac arrest. Breakdown of ordered electrical activity causes an ineffectual quivering of the ventricles, appearing as a chaotic line and providing no cardiac output (Figure 12.9c). *Asystole* is ventricular standstill that also leads to cardiac arrest. It is caused by VF that has 'burnt itself out' or a bradyarrhythmia that has become so slow that asystole occurs. It shows as a straight line with occasional minor fluctuations. VF and asystole can be misinterpreted when similar traces are produced by manual techniques to the chest or disconnected electrodes respectively.

Heart block (HB) is an anatomic or functional interruption in the conduction of an impulse, shown as a disrupted relationship between P wave and QRS complex. Causes are hypoxia, MI, digoxin therapy, heart disease or complications after heart surgery. First-degree HB shows a prolonged PR interval, but there are no symptoms or need for treatment. Second-degree HB shows dropped beats, and if it causes dizziness, fainting or reduced CO, a pacemaker is indicated. In third-degree HB, atrial and ventricular rhythms are independent of one another (Figure 12.9d). This requires a pacemaker to avoid a form of syncope called a Stokes Adams attack. *Bundle branch block* disturbs intraventricular conduction and widens the QRS complex.

SUPPORT

Fluids

Dehydration: intracellular and interstitial water deficit stemming from hypertonicity and disturbed water metabolism

Hypovolaemia or *volume depletion*: extracellular fluid depletion which affects vascular circulating volume and haemodynamic status.

An adequate circulating volume is the primary consideration before drugs or other forms of support are given. Blood volume determines preload and is the single largest contributor to cardiac function (Wilkins *et al.*, 1995, p. 322). A well-filled patient is less likely to suffer haemodynamic compromise with manual hyperinflation and suction (Schwartz, 1987). Fluid homeostasis normally operates to preserve tissue perfusion first and tonicity second (Mange *et al.*, 1997) but unstable patients may have difficulty achieving this balance.

The fate of administered fluids depends on which type of fluid is chosen. *Colloids* are thick fluids such as plasma, blood and dextran that contain large molecules and are retained in the circulation. Colloids are normally unable to escape through the capillary endothelium and therefore help keep water in the vascular compartment by exerting oncotic (colloid) pressure from within. Transfused colloid therefore stays in the intravascular compartment and influences circulatory function (Golster, 1995). Colloids that have an oncotic pressure greater than plasma are called plasma expanders. Blood is sometimes classified separately because it has oxygen-carrying capacity. Albumin is a colloid that has recently been condemned until further research measures its safety (Roberts 1998a).

Crystalloids are thin fluids with small molecules such as dextrose and saline. They have less effect on intravascular volume because over 70% is lost from the plasma into the interstitial space (Helmqvist, 2000). Excess crystalloid transfusion, when escaping from the circulation, may cause interstitial oedema which impairs oxygen transport from blood to tissue cells, or pulmonary oedema which impairs gas exchange in the lungs (Choi *et al.*, 1999). Crystalloid is used to provide the daily requirements of water and electrolytes. Too much crystalloid is risky in patients with ARDS because of leaky capillaries and impaired compensating mechanisms.

There is some controversy over which to choose for fluid resuscitation in hypovolaemic patients. Colloid is usually recommended but it is more expensive and has more side effects. In general, hypovolaemia is often treated rapidly with normal saline, and dehydration slowly with 5% dextrose (Mange *et al.*, 1997).

Nutrition

> ***Recovery is often dependent upon ability to complete the healing process prior to the exhaustion of fuel.***
>
> Shikora, 1996

The mythical comparison between the nutritional status of prisoners of war and ICU patients comes perilously close to the truth at times. Patients who require the most nutrition are those least likely to be adequately fed (Shikora, 1996). Physiotherapists may watch their patients waste away on the empty calories of a dextrose infusion while they are struggling uphill to maintain the condition of lungs and limbs. Rehabilitation is directly affected: one study showed how extra postoperative nutrition in orthopaedic patients enabled them to be independently mobile 5 days earlier than controls (Bastow *et al.*, 1983).

Causes of malnutrition

Patients may be disadvantaged by a pre-existing nutritional deficit, gut problems, glucose intolerance or impaired perfusion to the liver and kidney. Further obstacles common in the ICU are:

- inadequate feeding in an attempt to limit weight gain following fluid overload during surgery (Lowell, 1990)
- lack of recognition of a process as undramatic as malnourishment
- limited understanding of the importance of nutrition in the healing process
- the patient's lack of hunger, ability to express hunger or capacity to eat normally.

On top of this is hypermetabolism. Sykes and Young (1999, p. 230) calculate that a postoperative patient might require 10% extra calories, someone with peritonitis 25% extra calories, and a person with severe burns 60% extra. Increased WOB may demand another 20% extra calories overall. Rennie (1998) considers that neglect of nutritional support could be construed as malpractice.

Some degree of malnutrition is unavoidable in septic patients if the inflammatory response causes protein breakdown and obligatory nitrogen loss.

Effects of malnutrition

Malnutrition leads to muscle wasting, delayed weaning, impaired healing, atelectasis due to depleted surfactant, pulmonary oedema due to low albumin, increased risk of oxygen toxicity (Durbin, 1993), prolonged effect of paralysing drugs (Sinha, 1998) and the effects described on page 131.

Management

If patients are able, they should sit out and eat at normal times. If the gut is functioning, it should be used. If patients cannot swallow, PEG feeding (p. 264) is suitable for prolonged enteral nutrition. Patients on continuous morphine have reduced upper gut motility and may be better fed via the duodenum or jejunum than via the stomach (Bosscha *et al.*, 1998). Enteral feeding may be easier in right-side-lying because of the shape of the stomach.

Intravenous (IV) feeding is used only if necessary. It is deficient in certain essential nutrients, trebles the incidence of pneumonia compared to enteral feeding and causes atrophy of the gut lining (Reiland, 2000). The gut must keep moving to prevent stasis, bacterial overgrowth, permeability and invasion of intestinal flora into the systemic circulation, whence to wreak havoc in the rest of the body and risk multisystem failure (p. 409).

Problems can also arise from rapid feeding, overfeeding or inappropriate feeding. Starvation for over 48 hours can lead to 'refeeding syndrome' if subsequent feeding is too quick, with possible cardiopulmonary and neurological complications (Marik 1996b). Rapid administration of high carbohydrate feeds causes hyperglycaemia, which favours bacterial growth, may increase mortality (Elia, 1995) and increases CO_2 production. IV feeds with glucose providing more than 50% of the non-protein calories can increase CO_2 production two- to eightfold (Tobin, 1991, p. 4). Extra WOB is needed to increase minute ventilation and blow off the CO_2, and for patients with marginal respiratory reserve, this can precipitate respiratory failure (Liposky, 1994). Slow administration of high-fat low-glucose feeds may assist patients with hypercapnic COPD, especially during weaning. Immune-enhancing formulas show benefit for patients at risk (Singer and Little, 1999).

Medication

Infusion pumps are required to titrate drug dosage to the individual patient because:

- response varies due to complex interactions of multiple drugs
- ICU drugs are potent and some have a narrow window between effective and toxic doses
- physiological processing may be affected by the stress response or impaired circulatory, renal or liver function. (Drugs should preferably not rely on a failed organ for excretion).

Cardiovascular drugs

The relationship between heart function, vascular tone and fluid volume can be manipulated to augment cardiac output, reduce myocardial oxygen demand or redistribute blood flow to vital organs.

Diuretics

Reduction in blood volume and preload are the aims of diuretics (p. 138). They are used as the first-line drug for hypertension, heart failure and pulmonary oedema. When mobilizing a patient who is taking diuretics, a wary eye should be kept for signs of postural hypotension.

Systemic vasodilators

Hypertension, heart failure and angina are treated by venodilators such as the **nitrates**, which predominantly reduce preload, and arterial dilators such as **hydralazine**, which reduces afterload. Side effects include postural hypotension, especially in the early stages, and headache. Calcium channel blockers such as **verapamil**, and ACE inhibitors such as **captopril**, decrease vascular resistance and can lower BP. ACE inhibitors can cause a dry cough and, with the first dose, a drop in BP. If the cough is a problem, angiotensin II antagonists may be substituted.

Inotropes

A failing heart can be assisted by inotropes, which augment the force of cardiac contraction. Before giving inotropes, fluid status should be sufficient to ensure that the drug will not be stimulating an empty heart.

Adrenaline is an inotrope that stimulates the sympathetic system and increases the speed and force of cardiac contraction. At low doses it raises systolic pressure and at high doses it raises both systolic and diastolic pressures. It increases HR, cardiac output and myocardial oxygen demand. It dilates coronary and skeletal muscle vessels, constricts peripheral vessels and at high dose constricts renal blood vessels. **Noradrenaline** causes generalized vasoconstriction and increases systemic vascular resistance.

Dopamine is used in low doses because side effects include tachycardia, arrhythmias and increased pulmonary shunt. A small 'renal dose' may increase urine volume but benefit to the kidney is doubtful (Nolan, 1996). At high doses, dopamine causes vasoconstriction. *Dobutamine* gives a greater boost to oxygen delivery, has less adverse effects on shunt and does not cause vasoconstriction. *Dopexamine* is a weak inotrope, an arterial vasodilator and specifically increases renal and splanchnic blood flows. It is sometimes used perioperatively in high-risk patients as prophylaxis against renal failure.

Inotropic agents are used for circulatory failure and to offset the depressant effects of PEEP on cardiac and renal function. They do so at the expense of increasing myocardial oxygen demand. In patients with tachycardia, inotropes may cause myocardial ischaemia and actually decrease oxygen delivery. They are therefore only used after regulation of fluid and vasodilator therapy. Side effects mimic the effects of over-stimulating the sympathetic nervous system.

Digoxin is a cardiac glycoside with mild inotropic effects that has been in and out of fashion for two centuries. It helps control atrial fibrillation by strengthening and slowing HR, but may cause other arrhythmias in the presence of hypoxia.

Beta-blockers

Beta-blockers such as *propranolol* and *atenolol* are 'negative inotropes' which inhibit sympathetic action, block the action of adrenaline, slow the HR, reduce cardiac work and relieve hypertension, angina and arrhythmias. They are also used for anxiety, migraine and glaucoma. Beta-blockers may induce bronchospasm and tingling and numbness of the extremities, and are contraindicated in asthma. Other side effects are fatigue, hypotension, breathlessness and the blunting of cardiac exercise responses.

Anti-arrhythmic drugs

These include *amiodarone*, which can cause pulmonary fibrosis (Goldschlager *et al.*, 2000).

Pulmonary vasodilators

Patients with acute pulmonary hypertension may benefit from pulmonary vasodilators such as prostacyclin, nitric oxide or a combination (Hill and Pearl, 1999). *Prostacyclin* is administered intravenously or by nebulizer, has a half-life of 5 minutes and is less toxic than nitric oxide (Scheeren, 1997) but can affect the systemic vasculature and cause hypotension.

Nitric oxide has an unpromising background as a corrosive gas that is found in bus exhausts, cigarette smoke, smog and welding fumes. But it plays a role in controlling blood vessel wall tone, and its Janus-faced nature allows it to be exploited to dilate vessels adjacent to ventilated alveoli, decreasing shunt, reducing $\dot{V}_A/\dot{Q}$ mismatch and benefiting 50% of patients in severe respiratory failure (Singer and Webb, 1997, p. 180). When inhaled, its effects are limited to the pulmonary vasculature because it has a 1500 greater affinity than carbon monoxide for haemoglobin (Greenough, 1995) and is inactivated by the time it reaches the systemic circulation. Side effects include loss of surfactant and platelet function, toxicity with high F_IO_2 levels and rebound pulmonary hypertension if the dose is reduced too quickly. Heat moisture exchangers are safer than hot-water baths because excessive humidification can combine with nitric oxide to form nitric acid (Singer and Webb, 1997, p. 180).

Patients should not be removed from their nitric oxide during physiotherapy. If manual hyperinflation is necessary, the gas can be filtered into a rebreathing bag. However, many of these patients are critically ill and dependent on PEEP to maintain gas exchange, in which case manual hyperinflation is relatively contraindicated.

Sedatives

Sedation is required for most patients on IPPV, but should not be used as first line management of anxiety or asynchrony with the ventilator. Drugs that cloud consciousness cause delusions if anxiety stems from patients' realistic perception of their situation. Sedatives are no substitute

for the primary task of explanations, relief of discomfort and accurate ventilator management.

Commonly prescribed anxiolytics are:

- *diazepam* or *midazolam*, which have a long half-life
- *chlormethiazole*, which can increase bronchial secretions
- *isoflurane*, which if prolonged can cause liver failure
- *propofol*, which stabilizes HR and BP.

All sedatives reduce oxygen consumption but can cause respiratory depression, loss of time sense and misinterpretation of voices and noises which can paradoxically increase anxiety. Midazolam can cause dizziness and hiccups. Propofol carries a high lipid load, which can increase CO_2 production (Sykes and Young, 1999, p. 229), but its quick onset makes it useful as a bolus 2 minutes before physiotherapy. It attenuates metabolic as well as haemodynamic disturbance, stabilizing excess oxygen consumption and CO_2 production (Cohen *et al.*, 1996). Both midazolam and propofol can reduce BP.

Analgesia

Prior to physiotherapy, a bolus of intravenous analgesia is often indicated, using a short-acting drug such as *fentanyl* or *alfentanil*. If this is not adequate, *Entonox* may be used before and during treatment. This can be administered through some ventilators either by the intensivist or, after training, by a respiratory technician or physiotherapist.

Muscle relaxants

> *'You can't scratch your arm if it itches. You can't do nothing. Except lay there in one position. That's very, very uncomfortable.'*
>
> Patient quoted by Jablonski, 1994

Neuromuscular blocking agents such as *atracurium* or *pancuronium* are paralysing agents. They are used if it is detrimental to allow patients to move after acute head injury, or as a means to reduce oxygen consumption in severely hypoxic patients. They may be used as a last resort if patients are resisting ventilation and deep sedation is inadequate. Neuromuscular blockade should not be used to keep a patient quiet. They act as a form of chemical restraint, and for patients this can feel frightening, especially if they are not told that they are being given a drug that will make them feel weak. Patients feel, hear and think normally, and need regular explanation and orientation.

The induction of weakness, rather than paralysis, is normally sufficient (Marino, 1995). Patients should be sedated to the point of unrousability beforehand (Oh, 1997, p. 675) and appropriate analgesia administered because paralysing drugs obliterate the only means by which patients can indicate discomfort. Accurate monitoring can be provided by a peripheral nerve stimulator (Rowlee, 1999), but the drug should be withdrawn every 48 hours to assess the need for continued paralysis (Marcy, 1994).

Disadvantages are risk of pneumonia (Cook, 1998) and persistent myopathy in patients who are taking steroids (Behbehani, 1999) especially if those in renal failure are taking pancuronium, which is excreted by the kidney.

Treatment for pain, anxiety and ventilator asynchrony is interwoven, but medication for each should be distinguished. Harvey (1996) has shown that up to 25% of ICU staff believe paralysing drugs reduce anxiety, and up to 80% believe that diazepam has analgesic properties despite evidence that sedatives can increase sensitivity to pain. Withdrawal syndromes can develop when analgesics, sedatives or muscle relaxants are stopped after prolonged use (Cammarano *et al.*, 1998).

Drugs for airflow obstruction

Airflow obstruction increases airway pressure and heightens the risk of barotrauma and haemodynamic disturbance. Bronchodilators or steroids can be delivered to ventilated patients by metered dose inhaler (MDI) or small-volume nebulizer. The effect of aerosolized drugs is variable and should be monitored, e.g. by decreased wheeze on auscultation, a drop in

peak airway pressure, a normalized flow curve (Figure 12.3a) or reduced intrinsic PEEP (Wollam, 1994).

Nebulizers should be removed and cleaned after each use. Craven *et al.* (1984) found that bacterial aerosol was produced by 71% of in-line medication nebulizers. The hot-water humidifier or HME should be removed during administration.

Nebulizers are more commonly used than MDIs, but can increase the effort to trigger a breath. An MDI with spacer delivers more drug than a nebulizer for the same dose (Marik *et al.*, 1999). The MDI is placed at the Y-connector and fired just after the beginning of inspiration, using double the dose compared to spontaneously breathing patients (Hess, 1994). Dry powder inhalers cannot be used in ventilator circuits.

Plasmapheresis

Plasma exchange is used to remove circulating toxins or replace missing plasma factors in people with immune-mediated diseases such as Guillain–Barré syndrome or myasthenia gravis (Appleyard and Sherry, 1998). Blood is separated into its components in a centrifuge, plasma is discarded and a plasma replacement fluid is infused in equal volume.

Pacemaker

When the heart's conducting pathways are damaged, an artificial pacemaker can deliver an electrical stimulus to the myocardium. For temporary use, pacing wires connect the patient's myocardium to an external pacing box. For permanent support, the energy source is implanted under the skin.

Indications are third-degree heart block, arrhythmias refractory to medication, and prophylactic support in the first days after heart surgery. Insertion of a permanent pacemaker requires the patient to rest afterwards, but they can mobilize fully in 24 hours. A cardioverter defibrillator may be implanted into patients at risk of VF (Collins, 1994).

Advanced cardiac support

For patients in profound heart failure, temporary assistance by an *intra-aortic balloon pump* can increase survival in high-risk patients (Arafa *et al.*, 1998). The pump, housed in a console, is connected to a catheter with a deflated balloon at its tip. This is threaded through the patient's femoral artery and into the aorta (Figure 12.10). Here it is triggered into action by the patient's ECG. Diastole causes balloon inflation, which assists aortic valve closure and augments perfusion of myocardium, brain and kidneys. In systole, the balloon deflates, decreasing afterload and assisting the ventricle to empty.

The effect is similar to combined inotropic and vasodilator therapy, increasing myocardial perfusion and reducing workload. Complications include vascular damage, embolism and lower limb ischaemia; peripheral perfusion should be checked hourly (Bentall, 1998). Heparinization lessens the risk of thrombosis but increases the risk of bleeding.

Indications for the baloon pump are critically impaired cardiac output, e.g. cardiogenic shock or inability to wean from cardiopulmonary bypass. As patients recover, assistance is reduced gradually from every beat (1:1) to every 4th beat (1:4). Implications for physiotherapy are the following:

- The augmented BP, visible on the console, should be monitored throughout.
- Hip flexion should be avoided on the cannulated side.
- Patients are often too unstable to turn, but if turning is indicated, care is required to avoid disconnection of the catheter.
- If manual hyperinflation is necessary, cardiac output should be monitored throughout.
- Manual percussion or vibrations are unwise because of interference with the ECG, and mechanical percussors and vibrators are contraindicated. If vibrations are needed, one supporting hand underneath the patient minimizes movement.

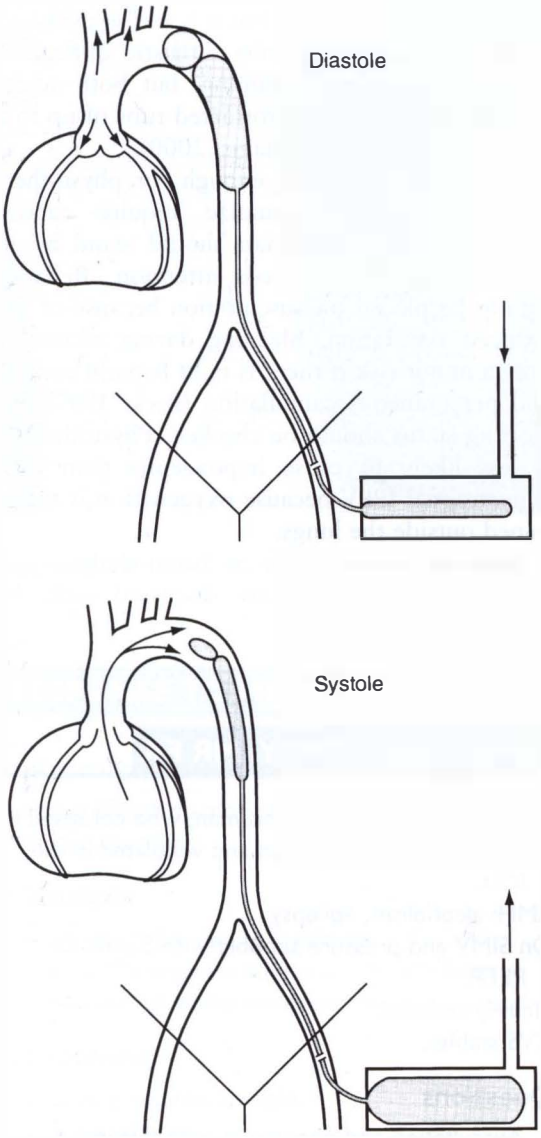


Figure 12.10 Intra-aortic balloon pump. An external balloon indicates the inflation or deflation of the intra-aortic balloon. (From Collier, P. S. and Dohoo, P. J. (1980) The intra-aortic balloon pump. *Physiotherapy*, **66**, 156–157, with permission.)

- To reduce the risk of bleeding, coughing should be avoided for some hours after removal of the balloon.

A *ventricular assist device* is a supplementary pump implanted in the abdomen. Developed as a bridge to heart transplantation, mortality can be reduced by 55% for patients awaiting a donor (Tector, 1998). It has also been developed for permanent circulatory support (Jarvik *et al.*, 1998).

Advanced pulmonary support

IPPV rests the respiratory muscles but does not rest lung tissue itself. If potentially damaging volumes and pressures are being delivered by the ventilator, they can be reduced by augmenting gas exchange with *intravascular oxygenation* (IVOX). Gas exchange occurs via a mop-like, 2-foot-long bundle of hollow fibres lying free in the vena cava. These are permeable to gases but not fluids and are flushed continuously with oxygen, while CO_2 is discharged through a double-lumen catheter. The device can provide 10–25% of the patient's oxygen requirements but obstruction to venous return may offset the benefits and it is little used at present.

Liquid ventilation eliminates the gas–liquid interface in the lungs by filling them with an

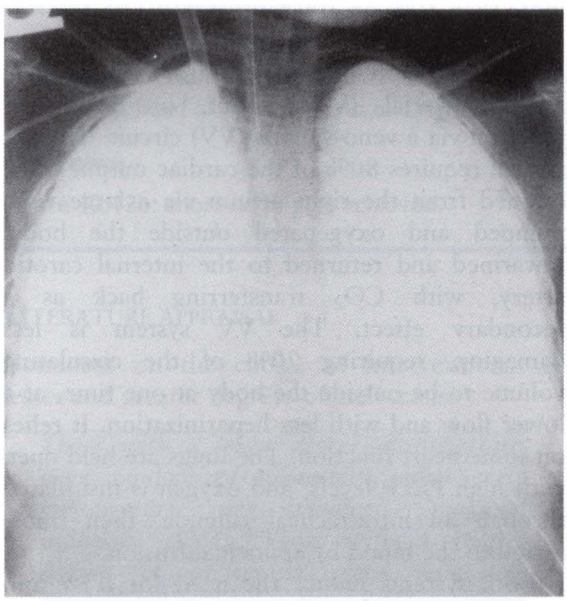


Figure 12.11 Radiograph of liquid ventilation.

inert colourless liquid called perfluorocarbon. This is twice as heavy as water, highly soluble to respiratory gases and opaque to X-rays (Figure 12.11). **Partial liquid ventilation** fills the lungs to FRC so that conventional IPPV can continue. This support system eliminates surface tension, allows ventilation at lower pressures, and helps alveolar recruitment in dependent areas by a 'liquid PEEP' effect. Disadvantages are that spontaneous breathing is hard work, and radio-opacity makes densities such as consolidation impossible to detect on X-ray, although pneumothoraces are crystal-clear. Mucus cannot mix with perfluorocarbon and tends to float on top, from where it can be debrided by saline lavage (Fuhrman *et al.*, 1998). Suction is forbidden. Liquid ventilation has so far shown benefits in paediatrics only.

Advanced cardiopulmonary support

As a last resort for people with severe but potentially reversible cardiopulmonary failure, extracorporeal gas exchange is a modified form of cardiopulmonary bypass and buys time for an injured lung to recover. Typical criteria include P_aO_2/F_IO_2 ratio of less than 11.2 and shunt of more than 30% on 100% oxygen.

Extracorporeal membrane oxygenation (ECMO) supports cardiorespiratory function via a veno-arterial (VA) circuit, or respiratory function via a veno-venous (VV) circuit. The VA system requires 80% of the cardiac output to be drained from the right atrium via a large vein, pumped and oxygenated outside the body, rewarmed and returned to the internal carotid artery, with CO_2 transferring back as a secondary effect. The VV system is less damaging, requiring 20% of the circulating volume to be outside the body at one time, at a lower flow and with less heparinization. It relies on some heart function. The lungs are held open with high PEEP levels, and oxygen is insufflated through an intratracheal cannula, then transferred to the blood by apnoeic diffusion.

Both systems reduce the need for IPPV and facilitate lung healing. There is concern about neurological damage from cannulation of the carotid artery in infants, but it is well established in specialist neonatal units. Logistic difficulties for adults are more daunting, but both adults and children can have protracted runs of up to a month (Fiddler and Williams, 2000).

If patients are stable enough for physiotherapy, the ECMO cannulae require careful handling and a technician should stand by in case the machinery needs attention. Reliance cannot be placed on auscultation because of the reduced ventilation. Bleeding during suction is only a minor risk if there is tight heparin control and percutaneous cannulation (Peek, 1997) but clotting status should be checked. Physiotherapy is less likely to cause hypoxaemia than with conventional IPPV because oxygenation is maintained outside the lungs.

Support systems such as haemodialysis and surfactant replacement are discussed with the relevant pathologies.

MINI CASE STUDY: MR FA

Identify the problems of this man, who collapsed in A&E, then was intubated and ventilated in the ICU.

RMH: alcoholism, epilepsy.

On SIMV and pressure support with 5 cmH_2O PEEP.

Heavily sedated.

CVS stable.

Questions

1. Auscultation and percussion note (Figure 12.12a)?
2. Analysis?
3. Problems?
4. Goals?
5. Plan?
6. Passive movements?
7. Outcome (Figure 12.12b)?

CVS = cardiovascular system, SIMV = synchronized intermittent mandatory ventilation, RMH = relevant medical history.

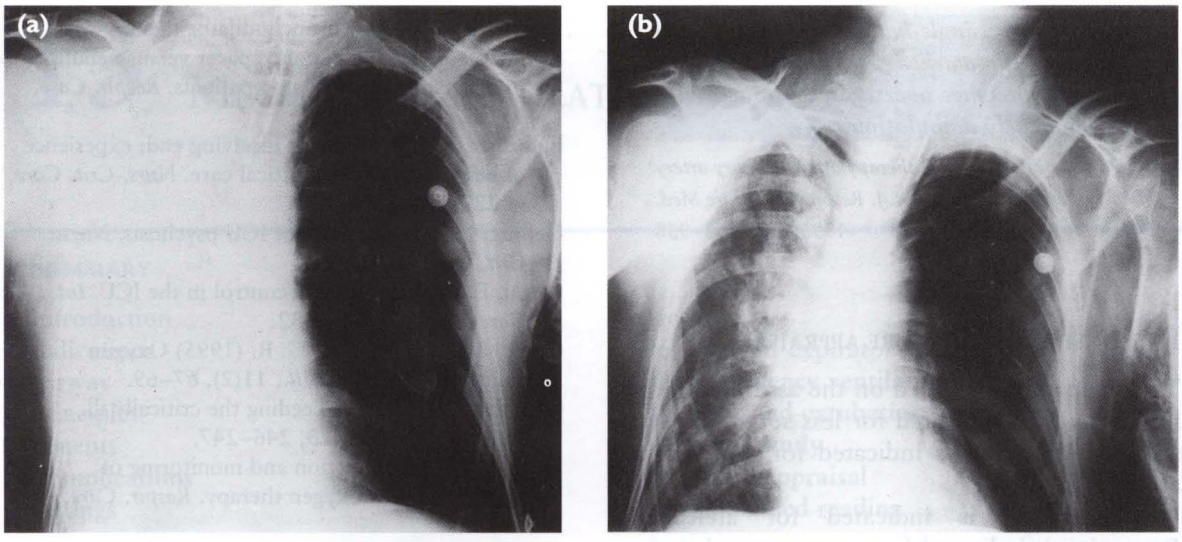


Figure 12.12 X-ray of Mr FA before and after physiotherapy.

RESPONSE TO MINI CASE STUDY

1. Auscultation and percussion note

Reduced breath sounds on right, percussion note dull on the right.

2. Analysis

Collapse probably due to a seizure.
Figure 12.12a suggests aspiration to right lung.
Possible alcohol intake increases risk of aspiration.

3. Problems

Loss of lung volume on right.
Poor gas exchange.
Possible pain.

4. Goals

Short term: restore functioning lung volume.
Medium term: restore patient mobility.
Long-term: team management of follow-up support and rehabilitation.

5. Plan

Review radiologist report in case of hidden rib fractures. If all clear:

- Optimize analgesia
- Position in left-side-lying
- Manual hyperinflation
- Percussion and vibrations
- Suction.

6. Passive movements

Unsafe until patient is able to report pain and orthopaedic team has assessed fractured left humerus, head of right humerus and right clavicle.

Outcome

Figure 12.12b: short-term goal achieved.

LITERATURE APPRAISAL

Comment on the choice of interventions for lesser and greater degrees of atelectasis.

> *Those with a lesser degree of atelectasis were randomized to receive either early mobilization or sustained maximal inflations (SMI). Those with a greater degree of atelectasis were separately randomized to receive either SMI or single-handed percussions.*

... adding single-handed percussions to patients with marked atelectasis does not improve outcomes over those obtained with SMI and early ambulation.

Postoperative physical therapy after coronary artery bypass surgery. Am. J. Respir. Crit. Care Med. 1995; **152**: 953–958

RESPONSE TO LITERATURE APPRAISAL

This appears to be based on the assumption that mobilization is indicated for less severe atelectasis and percussion is indicated for more severe atelectasis.

Mobilization is indicated for atelectasis. Percussion is indicated for sputum retention.

A glance at the physiology might have saved the authors a bit of time.

RECOMMENDED READING

Ahrens, T. (1999) Continuous mixed venous monitoring. *Crit. Care Nurs. Clin. North Am.*, **11**, 33–48.

Ahrens, T. (1999) Capnography. *Crit. Care Nurs. Clin. North Am.*, **11**, 49–62.

Ahrens, T. (1999) Pulse oximetry. *Crit. Care Nurs. Clin. North Am.*, **11**, 87–98.

Barnitt, R. and Partridge, C. (1997) Ethical reasoning in physical therapy and occupational therapy. *Physiother. Res. Int.*, **2**, 178–192.

Cowie, M. R. (1994) Understanding electrocardiograms. *Student Br. Med. J.*, ii, 229–232.

Duarte, A. G. (2000) Bronchodilator therapy with metered-dose inhaler and spacer versus nebulizer in mechanically ventilated patients. *Respir. Care*, **45**, 817–823.

Fulbrook, P. (1999) on the receiving end: experience of being a relative in critical care. *Nurs. Crit. Care*, **4**, 222–230.

Gelling, L. (1999) Causes of ICU psychosis. *Nurs. Crit. Care*, **4**, 22–26.

Girou, E. (2000) Infection control in the ICU. *Int. Care Med.*, **26**, 131–132.

Grant, I. S. and Nimmo, G. R. (1995) Oxygen transport. *Care Crit. Ill.*, **11**(2), 67–69.

Griffiths, R. D. (1997) Feeding the critically ill. *Intens. Care Med.*, **23**, 246–247.

Hess, D. (2000) Detection and monitoring of hypoxemia and oxygen therapy. *Respir. Care*, **45**, 65–80.

Kahn, D. M., Cook, T. E. and Carlisle, C. C. (1998) Identification and modification of environmental noise in an ICU setting. *Chest*, **114**, 535–540.

Kavanagh, R. J. (1995) Crystalloids and colloids in the critically ill patient. *Care Crit. Ill*, **11**, 114–119.

Lawrence, M. (1995) The unconscious experience. *Am. J. Crit. Care*, **4**, 227–232.

Quirk, J. (2000) Malnutrition in critical ill patients in ICUs. *Br. J. Nurs.*, **9**, 537–541.

Schallom, L. and Ahrens, T. (1999) Using oxygenation profiles to manage patients. *Crit. Care Clin. N. Am.*, **11**, 437–446.

Shelly, M. P. (1998) Sedation in the ITU. *Care Crit. Ill.*, **14**, 85–88.

Theaker, C., Mannan, M., Ives, N. and Soni, N. (2000) Risk factors for pressure sores in the critically ill. *Anaesthesia*, **55**, 221–224.

Webb, J. M. (2000) Delirium in the ICU. *Crit. Care Nurse*, **22**, 47–60.

13 Mechanical ventilation

Summary

Introduction

> *Patients often had difficulty understanding where the machinery ended and their bodies began ... [others] resented health professionals touching their ventilators ... [others] perceived the surrounding machinery as reassuring. Patients reported a need for repeated explanations.*
>
> Jablonski, 1994

Intermittent positive pressure ventilation (IPPV) augments or replaces the function of the inspiratory muscles by delivering gas under positive pressure to the lungs. This substitutes for the respiratory pump but is not necessarily beneficial for lung tissue, which is vulnerable to the shear forces of repetitive opening of alveoli. There is a narrow range of pressures and volumes within which the lungs are safe from either overdistension or atelectasis.

The traditional philosophy of ventilator management was the aggressive pursuit of textbook blood gas values, with secondary concern for complications. Now the primary aim is to minimize complications even if this leads to a degree of respiratory acidosis. IPPV is less about the application of a machine to a passive patient, and more about the complex interaction between patient and machine.

Indications

Patients may not have primary respiratory disease but are in impending or established respiratory failure. Indications are the following:

- Patients who are unable to ventilate adequately, oxygenate adequately or both. Examples are respiratory depression due to post-anaesthesia or drug overdose, inspiratory muscle fatigue due to exacerbation of COPD, inspiratory muscle weakness due to neurological impairment, or severe hypoxaemia due to lung parenchymal disease.
- Patients who are able to breathe adequately but for whom this is deemed inadvisable, e.g. those with acute head injury.
- Patients who require intubation for airway protection or to overcome upper airway obstruction. They require some ventilatory support to compensate for the work of breathing (WOB) through the tubing.

Airway

> *'like a toilet paper roll ... a hard rubber tube ... a soggy cigar ... like you were gagging on something.'*
>
> Patient describing his endotracheal tube (Jablonski, 1994)

Patient and ventilator are connected through a sealed tracheal tube (endotracheal or tracheostomy tube Figure 13.1), which reaches sufficiently beyond the vocal cords to safely allow some head movement.

An *endotracheal tube* (ETT) through the mouth or nose can be used for up to 2 weeks, but causes discomfort, distress and sometimes panic (Stauffer, 1999). A nasal tube is marginally better tolerated than an oral tube and causes less movement-related injury to the larynx, but creates more airflow resistance and increases the risk of sinusitis (Juniper, 1999b). Average internal diameter sizes are 8 mm for oral and 7 mm for nasal tubes.

A *percutaneous tracheostomy tube* is more comfortable than an ETT, causes less resistance, is easier for suctioning, may allow the patient to eat and is used when longer-term ventilation is anticipated. A newly created tracheostomy signifies the need for extra care during patient handling, especially suctioning.

A cuff (p. 279) prevents escape of ventilating gas past the tracheal tube and inhibits large volume aspiration. It is not watertight and does not compensate for the small-volume aspiration caused by inability of the vocal cords to close. All patients on IPPV therefore have some gastro-oesophageal reflux (Carter and Hornick, 1999). The optimum cuff pressure to maintain mucosal perfusion but minimize risk of aspiration is 25 mmHg, checked by nursing staff regularly with a manometer.

The problems of tracheal tubes are:

- disrupted communication
- swallowing dysfunction in up to 50% of patients (Tolep, 1996)
- risk of chest infection because of the damage described above, loss of defence mechanisms and an invitation for bacteria to breed in the pool of secretions that collects above the cuff and then trickles down past the cuff into the lungs
- with a tracheostomy, the complications described on page 281
- with an ETT: discomfort, gagging, retching, over-salivation, airflow resistance and damage to the trachea and larynx.

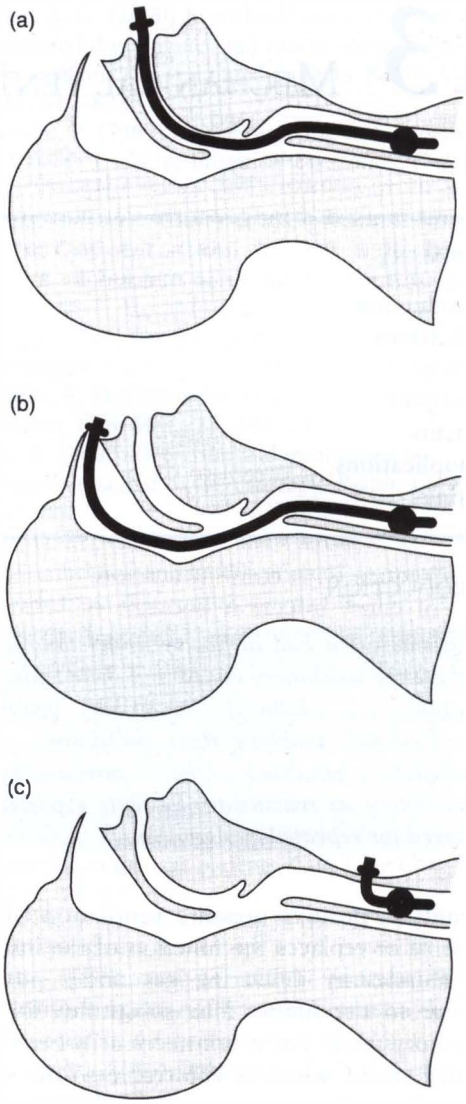


Figure 13.1 Tracheal tubes. (a) Oral endotracheal tube. (b) Nasal endotracheal tube. (c) Tracheostomy tube.

Airway damage is exacerbated by mishandling, traction from the weight of the ventilator circuit and excessive neck movement which can cause 2 cm excursions of the cuffed tube on either flexion or extension (Weiner *et al.*, 1991).

Humidification is provided by a hot-water humidifier, which is the most effective device and only needs to be changed between patients (Dreyfuss, 1995). Temperature alarms are set at maximum 37°C and minimum 30°C. An alternative is a heat–moisture exchanger (HME), which is adequate for short-term use in well-hydrated patients who do not have excessive or thick secretions. HMEs pose a lesser infection risk, and incorporate antibacterial properties. However, their use should be limited to between 24 hours (Marcy, 1994) and 5 days (Branson *et al.*, 1993) because they have not been shown to reduce the incidence of pneumonia (Dreyfuss, 1995) and can cause obstruction with thick secretions (Marcy, 1994).

PRINCIPLES

A bewildering array of all-singing all-dancing ventilators are flooding the market, leading to a terminology jungle which becomes more complex as their versatility increases. A ventilator breath can, however, be classified according to how it is triggered into inspiration, controlled (generated) during the inspiratory phase, and cycled into expiration.

Trigger

Either the patient or ventilator can trigger inspiration. For *patient-triggered breaths*, older machines require patients to trigger their breaths by a preset pressure of typically between –1 and –2 cmH_2O. This entails activation of a demand valve and a degree of effort, because an inbuilt insensitivity is necessary to prevent artefacts setting off inspiration. New machines use a more comfortable flow trigger, which senses a drop in the patient's flow at a sensitivity of, say, 3 L/min. For *ventilator-triggered breaths*, the inspiratory trigger is set according to time so that inspiration occurs automatically at a set rate.

Control

The method of control is the driving mechanism that delivers the inspiratory breath. It remains constant despite changes in ventilatory load.

Volume control (or volume-limited ventilation) delivers a specific minute volume at a constant flowrate, using preset variables such as respiratory rate (RR), tidal volume (V_T) and inspiratory:expiratory (I:E) ratio. Airway pressure rises slowly during inspiration to a peak value that varies with airway resistance and lung compliance. A pressure limit is set for safety. *Pressure control* (or pressure-limited ventilation) delivers a preset pressure during inspiration, resulting in a variable V_T that depends on the preset pressure, airway resistance, lung compliance, patient effort and inspiratory time.

Volume control is commonly used for adults for three reasons:

- It can be relied on to deliver a consistent minute volume regardless of airway resistance and lung compliance.
- It maintains steady P_aCO_2 levels when this is imperative, e.g. in acute head-injured patients.
- Inspiratory pressure increases gradually and may cause lesser shear forces on the alveoli.

Pressure control is usually considered to be safer for patients with stiff lungs (indicated by peak airway pressure above 35 cmH_2O on volume control), especially for those with ARDS, for whom it reduces the work of breathing (Kallet, 2000). For babies, it limits alveolar pressures and may reduce the risk of barotrauma.

Cycle

Inspiration cycles into expiration when a preset time has elapsed (time-cycled) or a preset pressure has been reached (pressure-cycled) or when a preset volume has been delivered (volume-cycled). Time cycling is the commonest and is adjusted via flow and/or I:E ratio. Pressure control is different from pressure cycling, a pressure-controlled breath being usually cycled into expiration after a preset time.

BENEFITS

- IPPV acts as an accessory muscle to supplement patient effort. This is hindered if there

is a narrow ETT, obstructed airways, stiff lungs, poor trigger sensitivity or inappropriate settings so that patient and machine are not synchronous.

- IPPV allows control of gas exchange and acid–base homeostasis by manipulating inspired oxygen, minute volume, airway pressure, I:E ratio and PEEP.

COMPLICATIONS

The negative effects of IPPV are of particular interest to the physiotherapist because some complications are worsened by the extra positive pressure of manual hyperinflation.

Impaired cardiac output

Positive pressure in the chest impedes venous return (preload) to the heart, which tends to decrease cardiac output, which in turn reduces renal, hepatic and splanchnic blood flow. Compensatory peripheral vasoconstriction normally maintains blood pressure, but this mechanism may not be viable in patients who are elderly, who suffer autonomic neuropathy such as Guillain–Barré syndrome, or are hypovolaemic, either absolutely, or functionally due to vasodilation e.g. in septic shock. These patients may drop their BP, especially when first ventilated.

Haemodynamic compromise is most likely if mean airway pressure is high, inspiratory time prolonged or mean expiratory pressure raised as with PEEP. These effects can be modified by fluids, inotropic drugs (p. 335) or reduced I:E ratios so that the heart has time to fill during the longer expiratory phase. Patients with poor lung compliance suffer less haemodynamic compromise because the alveolar pressure is transmitted less easily through the stiff lung.

Barotrauma

Barotrauma is extra-alveolar air, e.g. pneumothorax. The name arose from assumptions that the cause was excess pressure, because affected patients tend to have high peak pressures. It is now understood that excess volume is the cause, because sustained alveolar distension can rupture the delicate alveolar–capillary membrane (Heulitt, 1995). This explains why coughing, when pressure increases massively but volume is stable, rarely causes barotrauma. The term 'volutrauma' rather than 'barotrauma' is sometimes used to describe extra-alveolar air in this context.

Air first extrudes from distended alveoli, then tracks centrally along bronchovascular sheaths (Figure 13.2), causing *pulmonary interstitial emphysema*, i.e. air in interstitial lung

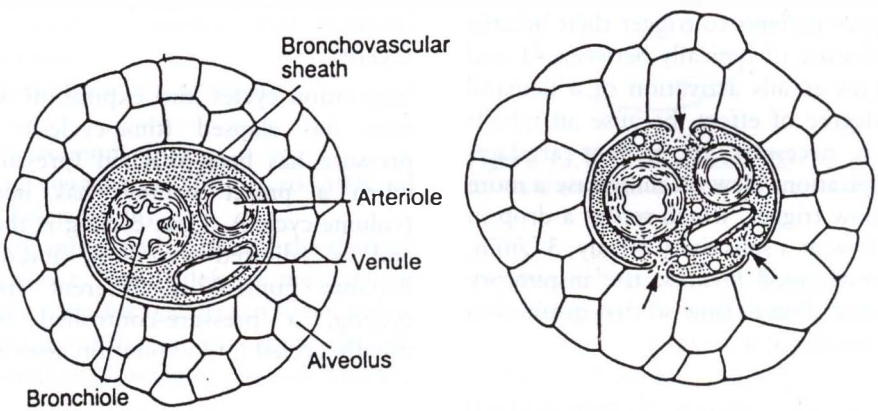


Figure 13.2 Development of barotrauma. Second picture shows overdistended alveoli leading to rupture of delicate alveolar–capillary membranes. (From Maunder, R. T., Pierson, D. J and Hudson, L. D. (1984) Subcutaneous and mediastinal emphysema: pathophysiology, diagnosis and management. *Arch. Int. Med.*, **144**, 1447–1453)

spaces. This can lead to *pneumomediastinum*, sometimes detectable on X-ray and usually evident on CT scan (Wiener *et al.*, 1991). A pneumomediastinum tends to vent into subcutaneous tissue and cause *surgical emphysema* (Figure 13.3), which may herald a *pneumothorax* (Jantz and Pierson, 1994). **Pneumopericardium** is not easily distinguishable from pneumomediastinum but is rare and usually occurs only after heart surgery or in neonates with stiff lungs.

Barotrauma is a significant risk in lungs that are stiff, hyperinflated or unevenly damaged. X-ray identification is discussed on page 49.

Displaced perfusion

The positive pressure of controlled mandatory ventilation (p. 350) displaces blood from the thorax and accentuates the perfusion gradient from upper to lower regions, leaving non-dependent lung regions virtually without blood flow (Figure 13.4). The degree to which perfusion is affected depends on the proportion of positive pressure created by the mode of ventilation.

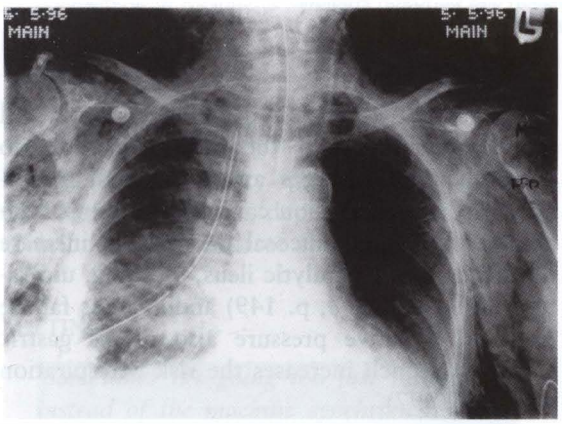


Figure 13.3 Soft tissue shadowing outside the rib cage in the subcutaneous tissues of the upper trunk, indicating surgical emphysema. The patient is intubated, has a chest drain suggesting a recent pneumothorax, and a calcified aorta suggesting advanced age.

Displaced ventilation

Spontaneous breathing draws ventilation down to dependent lung regions, causing a downwards ventilation gradient (p. 9). If the patient is receiving full ventilator-triggered IPPV in the form of controlled mechanical ventilation, this gradient is reversed (Figure 13.14) and ventilation is abolished in dependent lung regions (Hedenstierna, 1997). Reasons are the following:

- Positive pressure gas takes the path of least resistance which is to the more open upper regions.
- The diaphragm is inactive and it is irrelevant that its dependent fibres are more stretched by abdominal viscera.
- The lower region is compressed by the increased perfusion gradient and is less compliant.

Dependent areas therefore receive the least ventilation and are vulnerable to progressive atelectasis. The ventilation gradient is less disturbed when a mode is used in which spontaneous breathing is encouraged.

Increased dead space

Dead space increases because of reduced overall perfusion, and to a lesser extent because of distension of ventilator tubing.

$\dot{V}_A/\dot{Q}$ mismatch

Disturbed ventilation and perfusion gradients, and increased dead space, result in $\dot{V}_A/\dot{Q}$ mismatch, which would lead to hypoxaemia if not offset by ventilator strategies such as PEEP, inspiratory pause and supplemental oxygen.

Fluid imbalance

Fluid retention can be caused by decreased renal perfusion, redistribution of blood flow within the kidneys, and stress-mediated ADH secretion (Pilbeam, 1998, p. 148). Fluid maldistribution can occur with large lung volume changes, causing overdistension of alveoli, depletion of surfactant, microvascular damage, leakage of fluid and pulmonary oedema (Heulitt, 1995).

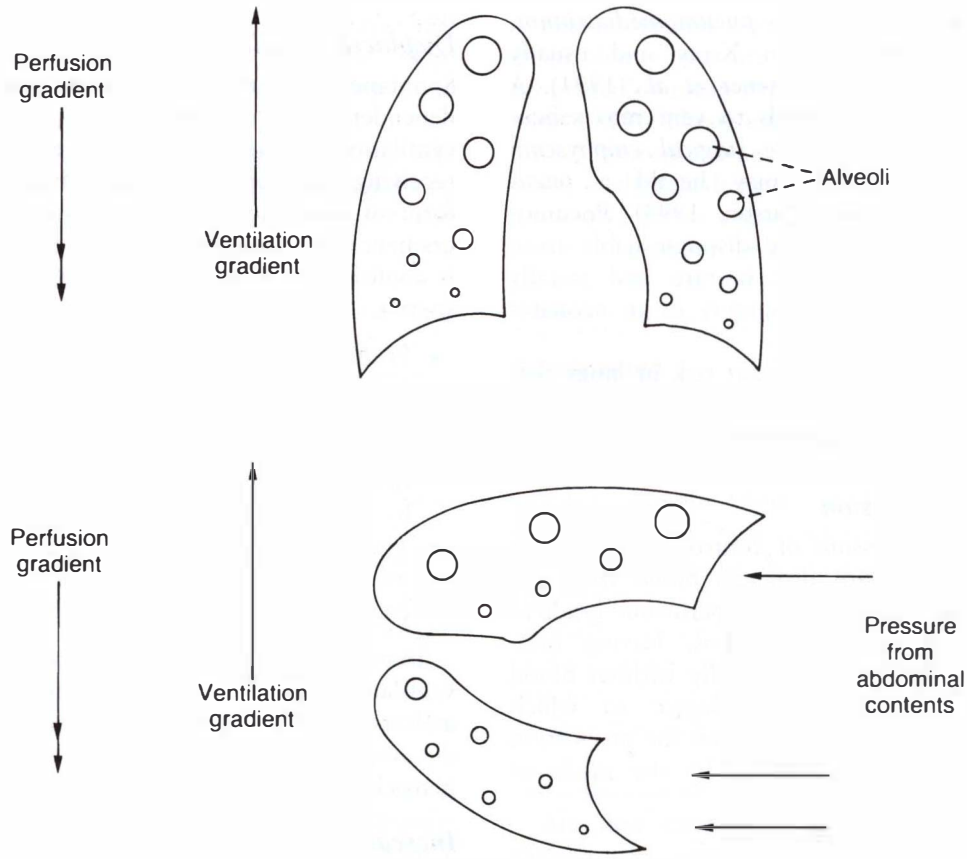


Figure 13.4 Effect of controlled mandatory ventilation on ventilation and perfusion gradients. In contrast to spontaneous respiration, the perfusion gradient increases downwards and the ventilation gradient is reversed. Compare with Figure 1.6.

Discomfort

IPPV can be uncomfortable if full explanations are not given and if ventilation is not matched synchronously to the patient.

Breathlessness

Even when minute volume is adequate, patients can feel breathless because of asynchronous ventilation, loss of control of their breathing, increased airflow resistance in the tracheal tube and abnormal stimulation of lung stretch receptors.

Gut dysfunction

Splanchnic blood flow does not have autoregulation capabilities and is dependent on arterial blood pressure. It is therefore vulnerable to haemodynamic changes and abdominal distension. Reduced perfusion can increase the permeability of the gut mucosal barrier and increase the incidence of paralytic ileus, bleeding, ulceration (Pilbeam, 1998, p. 149) and systems failure (p. 409). Positive pressure also delays gastric emptying, which increases the risk of aspiration (Rennie, 1997).

Complications of high oxygen levels

Oxygen toxicity (p. 120) can occur with prolonged high F_IO_2 levels. Absorption atelectasis (p. 120) can occur when a high F_IO_2 is used with low tidal volumes.

Excess secretions

Bronchial secretions are increased, partly by irritation from the tracheal tube and partly because secretion clearance is impaired. Secretion volume increases with length of intubation (Palmer and Smaldone, 1998).

Gas trapping

Intrinsic PEEP (p. 68) occurs unintentionally when exhalation has not finished before the next breath starts. It is more likely with volume control ventilation and commonest with hyperinflation conditions such as emphysema or acute asthma, when air is trapped in the lungs by obstructed airways and a narrow tracheal tube. The result is overdistended alveoli and risk of barotrauma.

Signs of hyperinflation such as reduced breath sounds and hyperresonant percussion note suggest that intrinsic PEEP is present. Confirmation is by a flow–time curve showing persistent flow at end-expiration (see Figure 12.3b). The effects of this unwanted PEEP can be mitigated by ventilator manipulations and airway clearance to reduce airways resistance. Extrinsic PEEP (p. 353) can be deliberately imposed to counterbalance intrinsic PEEP, provided it is below the intrinsic PEEP level (Patel and Yang, 1995).

Weak inspiratory muscles

Resting the respiratory muscles causes atrophy, the degree of which depends on the relative contribution of ventilator and patient.

SETTINGS

> ***'Sometimes it's going too fast for you, so instead of the machine synchronizing with you, you have to synchronize with the machine.'***
>
> Patient quoted by Jablonski, 1994

Ventilation and oxygenation are matched to the patient according to P_aCO_2 and P_aO_2 respectively. A healthy spontaneously breathing adult maintains adequate ventilation with a V_T of 450–600 mL and RR of 10–15/min to give an approximate *minute volume* of 5–9 L. Minute volume is adjusted according to P_aCO_2, V_T being adjusted for a small change in P_aCO_2, and RR adjusted for a larger change. Normal range for minute volume on IPPV varies widely; a COPD patient with chronic hypercapnia requires a great deal less than a hypermetabolic septic patient.

Inspired oxygen concentration (F_IO_2) is adjusted according to P_aO_2, although the relationship between F_IO_2 and P_aO_2 is less direct than that between minute volume and P_aCO_2 (Chatburn, 1991) because P_aO_2 is subject to more variables.

Inspiratory flow rate is related to the *I:E ratio*. I:E ratio is normally 1:2 to allow adequate expiratory time for CO_2 clearance and venous return. It can be set as low as 1:4 to prevent intrinsic PEEP or as high as 4:1 (inverse-ratio ventilation) in severely hypoxaemic patients in order to recruit alveoli. For a patient with emphysema and prolonged expiration who normally has a spontaneous I:E ratio of 1:5 or 1:6, inspiratory flow is increased for rapid inspiration and long exhalation. For a patient with fibrotic lungs who works hard to inhale and whose high recoil pressure speeds exhalation, the flow rate is slowed for prolonged inspiratory time and short expiratory time.

Inspiratory pause (plateau) provides an end-inspiratory-hold, which enhances gas distribution by allowing time for recruitment of poorly ventilated alveoli. *Trigger sensitivity* dictates the patient's negative pressure that is required to initiate the breath if pressure-triggering is used. A *sigh mechanism* is available with some ventilators but little used; in most patients the risk of atelectasis is better reduced by PEEP (Chatburn, 1991), but patients with acute respiratory distress syndrome (ARDS) have shown improved gas exchange with this 'automatic sigh' facility (Pelosi *et al.*, 1999).

MODES

IPPV can take over the WOB by controlled mandatory ventilation, or the work can be shared between ventilator and patient using a variety of ventilatory modes. These create the pressure, flow and volume patterns that allow ventilatory support to be adjusted to the individual so that less sedation is required and less complications ensue. Modes have to be matched skilfully to the patient because all are less efficient than spontaneous breathing (Shelledy, 1995). Too much support leads to muscle atrophy, and too little overworks the patient. Terminology varies according to country and manufacturer but the following are common parlance.

Controlled mandatory ventilation (CMV)

Fully controlled mandatory ventilation is a ventilator-triggered mode that is only needed for patients who are unable to breathe at all or for whom complete control is necessary (see Figure 12.2). CMV is an unforgiving mode that dictates the depth and frequency of each breath and time-cycles into exhalation. Patients do not like to be controlled and sedation is always required. Risks of intrinsic PEEP and other complications are significant. Minute volume is set high enough to maintain a mild respiratory alkalosis so that spontaneous breathing is inhibited.

Assist-control

Assist-control is patient-triggered CMV. Breaths are triggered or imposed according to patient effort. In some ventilators the only difference from CMV is the trigger sensitivity, while in others there are more sophisticated differences. Hyperventilation and respiratory alkalosis are risks.

Intermittent mandatory ventilation (IMV)

The IMV mode allows patients to breathe spontaneously between a preset number of mechanical breaths, without regard for the patient's breathing pattern.

Synchronized intermittent mandatory ventilation (SIMV)

If patients do not breathe after a preset time interval, the SIMV mode delivers a mandatory breath. Breaths are mandatory or spontaneous according to the stage of the SIMV cycle (Figure 13.5a). SIMV has superseded IMV because synchrony with inspiratory effort is more comfortable, thus avoiding breath stacking and excess WOB. Preset variables include RR, V_T, inspiratory time and pause time. Cycling is by pressure or time, whichever comes first.

Pressure support

Pressure support (PS) is a patient-triggered flow-cycled mode which provides a preset pressure boost to each inspiratory effort. This pressure continues on a plateau until inspiratory flow is less than 25% peak, when cycling into expiration occurs. Patients can determine their own RR, V_T and I:E ratio (Figure 13.5b). The preset variables are trigger sensitivity and pressure support level. PS reduces WOB in proportion to the pressure delivered. A pressure of 5–8 cmH_2O overcomes the work imposed by the ventilator circuit and ETT, and is therefore equivalent to spontaneous breathing. A pressure of 25 cmH_2O effectively eliminates the patient's need to do more than trigger the breath.

PS is relatively comfortable and ensures synchrony because patients have control. It acts like IPPB (p. 159) but inspiration is terminated according to flow rather than pressure, thus discouraging the unhelpful expiratory effort that can impair the effectiveness of IPPB. PS is used for patients who can reliably trigger the ventilator. A modification of PS is called proportional assist. This acts as a form of 'power steering' by responding to the patient in proportion to inspiratory effort.

Assist mode

This is similar to the PS mode, and the term is sometimes used interchangeably, but the breathing pattern is fixed by the characteristics

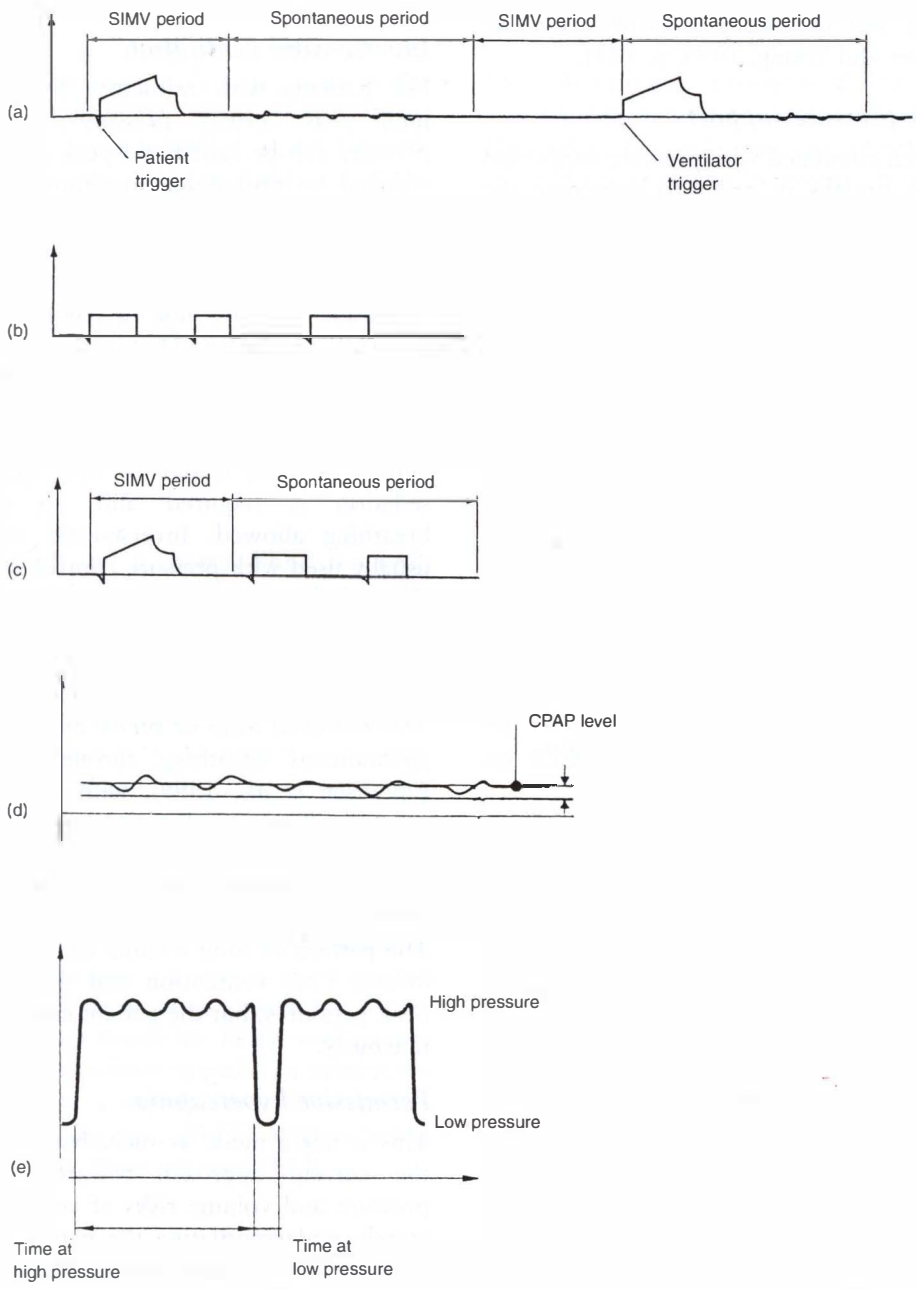


Figure 13.5 Pressure–time curves. Negative deflections indicate patient-triggered breaths. The baseline would normally be raised above zero to indicate PEEP. (a) SIMV. The first cycle shows a mandatory breath synchronized with the patient's inspiration, then spontaneous breaths. The second cycle shows an apnoeic period, then a mandatory breath triggered by the ventilator after a preset time interval. (b) Pressure support. Breaths vary according to the patient's breathing pattern. (c) SIMV with pressure support. Spontaneous breaths are supported by inspiratory pressure. (d) CPAP. All breaths are spontaneous breaths at an elevated pressure level. (e) Airway pressure release ventilation. All breaths are spontaneous and at an elevated pressure level, but the pressure is released at timed intervals.

of the lung and the machine rather than the patient (Sykes and Young, 1999, p. 153).

SIMV with pressure support

SIMV is often combined with pressure-supported spontaneous breaths in order to overcome the resistance of the tubing or, at higher pressures, to provide extra support (Figure 13.5c).

Continuous positive airway pressure (CPAP)

CPAP (Figure 13.5d) is used with spontaneously breathing patients. For intubated patients, it follows the same principle as non-invasive CPAP by mask. It carries the same disadvantage of increased expiratory WOB, but when delivered through a ventilator, it is more beneficial than mask CPAP because:

- it imposes less inspiratory WOB because the trigger is by a demand-flow system
- it carries no mask complications
- it substitutes for the bypassed larynx by creating a form of physiological PEEP (p. 357) to prevent alveolar collapse.

CPAP is specifically suited to patients who have poor gas exchange or intrinsic PEEP due to airflow obstruction. When combined with pressure support, it is similar to BiPAP.

Bilevel positive airways pressure (BiPAP)

BiPAP through the ventilator is equivalent to the support provided by non-invasive ventilation for the spontaneously breathing patient (p. 179). Beware the terminology 'BIPAP' with a capital I, which is a different and obscure mode (Silver, 1998) similar to airway pressure release ventilation (see below).

Mandatory minute ventilation (MMV)

MMV is a little-used mode in which the ventilator provides a guaranteed preset minute volume if the patient's spontaneous breathing drops below a preset level. Pressure support is sometimes added to ensure an adequate V_T for patients with rapid shallow breathing.

Inverse-ratio ventilation

For patients with refractory hypoxaemia and high peak airway pressures, mean airway pressure can be raised and peak airway pressure reduced by prolonging inspiratory time to the point of reversing the I:E ratio. The inspiratory flow rate is slowed or the inspiratory pause increased so that a longer inspiration recruits collapsed alveoli and a shorter expiration prevents recollapse. Disadvantages are risk of distended alveoli, intrinsic PEEP, compromised cardiac output and the discomfort of an unnatural breathing pattern during which the patient is often unable to fully exhale. Heavy sedation is required and no spontaneous breathing allowed. Inverse-ratio ventilation is usually used with pressure control to reduce the risk of barotrauma for people with ARDS (Ludwigs, 1998).

Airway pressure release ventilation

This modified form of BiPAP allows unrestricted spontaneous breathing throughout the cycle (Putensen *et al.*, 1999), with intermittent 1–2 second releases so that CO_2 can be eliminated and fresh gas can fill the alveoli (Figure 13.5e). Preset parameters are the high and low pressures, and the times at each pressure level. The pattern of lung volume change is similar to inverse ratio ventilation and therefore reduces peak pressures, but the patient can breathe spontaneously.

Permissive hypercapnia

This is not a mode as such, but an outcome of the current approach towards limiting the pressure and volume risks of ventilation. Deliberately underventilating the patient causes CO_2 retention but helps mitigate some of the complications of IPPV. Blood gas targets are modified and a low minute volume allows P_aCO_2 to rise up to 8 kPa (60 cmH_2O), with pH and oxygenation closely monitored. Compensation restores pH in the brain and myocardium towards normal over several hours (Allan, 1998). If there is no cerebral or cardiac injury, hypercapnia is

normally well tolerated and pH might be allowed to drop to 7.2, at which level compensating mechanisms are usually adequate if the kidneys are functioning normally (Freichels, 1998). Permissive hypercapnia is used for people with damaged lungs such as ARDS, or those sustaining high airway pressures e.g. acute asthma.

POSITIVE END-EXPIRATORY PRESSURE (PEEP)

There are several ways to boost S_aO_2:

- ↑ F_IO_2
- prolong the plateau
- ↑ I:E ratio
- apply PEEP.

To all intents and purposes, PEEP is CPAP, but the term is used for ventilated patients only. PEEP maintains constant positive pressure in the lungs throughout exhalation so that airway pressure does not fall to atmospheric pressure at end-exhalation. Like CPAP, it prevents alveoli from collapsing and aims to improve oxygenation. Unlike CPAP, it does not require the patient to breathe.

PEEP can be used with any mode of ventilation. Pressures vary from 3 cmH_2O to over 20 cmH_2O and are shown when the manometer of the ventilator returns to this value instead of zero at end-exhalation. For pressure-triggered breaths, the machine is set to cycle into inspiration a few cmH_2O below the PEEP level so that the patient does not have to make an inspiratory effort all the way down to zero. This applied PEEP is termed 'extrinsic PEEP' when it needs to be distinguished from intrinsic PEEP.

Benefits

Positive effects of PEEP include:

- stability of alveoli and conservation of surfactant (Verbrugge, 1998)
- resting lung volume raised out of the range of airway closure
- increased alveolar availability for gas exchange.

In healthy adults, 5 cmH_2O of PEEP raises FRC by 400–500 mL (Wilkins *et al.*, 1995, p. 251). At optimum pressures, $P_{A-a}O_2$ is reduced and oxygen saturation improved. The risk of atelectasis is decreased at pressures above 10 cmH_2O, as shown by reduction in radiological lung densities, but these reappear within a minute of removing the PEEP (Brooks-Brunn, 1995).

Complications

Excess PEEP can cause hyperinflation, which is risky and does not increase recruitment for gas exchange (Peruzzi, 1996). High levels of PEEP are associated with complications that are exaggerations of the complications of mechanical ventilation, especially the following:

- The continuous positive pressure impairs venous return and cardiac output. This can offset the beneficial effects of PEEP by causing a net decrease in oxygen delivery to the tissues. Haemodynamic compromise usually occurs at over 15 cmH_2O PEEP in normovolaemic patients, at lower pressures in hypovolaemic patients and at higher pressures in patients with stiff lungs. Fluid administration may compensate by boosting intravascular volume and stabilizing cardiac output, but this may incur pulmonary oedema when PEEP is discontinued. Haemodynamic monitoring is required, especially if perfusion to the gut and kidney is at risk (Azar, 1996). PEEP should be applied in small increments and titrated against oxygen delivery.
- PEEP increases the risk of barotrauma in patients who have lung disease, especially in hyperinflation conditions, unless carefully controlled extrinsic PEEP is being used specifically to reduce intrinsic PEEP
- Raised pressure within the thoracic cage increases CVP and PAWP readings (p. 327 and 329) at the same time as the ventricular filling pressure that they represent is declining because of impaired venous return.
- High-level PEEP may disrupt the alveolar–

capillary barrier and redistribute alveolar fluid, leading to pulmonary oedema.
- When disconnecting the ventilator circuit for suction, pressure from PEEP increases blow-back, with risks to staff and other patients of cross-contamination. This risk is eliminated with an in-line suction catheter.

Precautions

High-level PEEP should be avoided with an undrained pneumothorax and used with caution in patients who have surgical emphysema, bulla or bronchopleural fistula. Hypovolaemia is a relative contraindication, but if PEEP is necessary, measures can be taken to support cardiac output with fluids and inotropes. At levels above 10 cmH_2O, manual hyperinflation requires certain precautions (p. 375).

Best PEEP

While effective PEEP increases lung compliance and boosts S_aO_2, excessive PEEP decreases compliance by over-distending alveoli (see Figure 1.3), and reduces cardiac output. Best PEEP means optimum oxygen delivery. If tissue oxygenation monitoring is not available, PEEP is titrated against the optimum balance of S_aO_2 and cardiac output. The effect on oxygen delivery is measurable within 15 minutes of initiating PEEP (Patel, 1993). Figure 13.6 shows how best PEEP improves ventilation to the lung bases.

Indications

'Physiological' PEEP at 3–5 cmH_2O is routinely applied in order to maintain alveolar stability, and is especially useful in low lung volume states to prevent progressive parenchymal injury. Higher levels of PEEP promote gas exchange and reduce the necessity for toxic levels of inspired oxygen. Occasionally, differential ventilation with selective PEEP is used for targeting specific atelectatic areas (Klingstedt *et al.*, 1991).

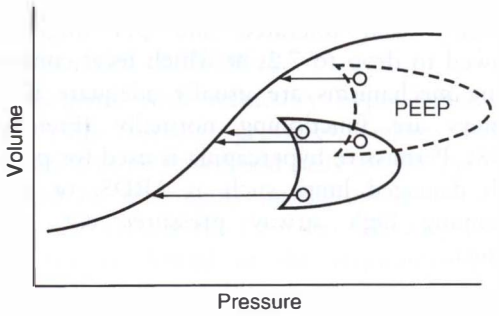


Figure 13.6 Effect of PEEP on regional pressure and volume relationships, showing how it improves ventilation in dependent lung. Compliance is greater (the curve steeper) in the upper part of the lung without PEEP, and in the base of the lung with PEEP. (From Lumb, A. B. (2000) *Nunn's Applied Respiratory Physiology*, 5th edn. Butterworth-Heineman, London)

HIGH FREQUENCY VENTILATION

How does the Himalayan mountain shrew maintain oxygenation during copulation? With a RR up to 600/min, its V_T is less than its dead space but it still manages to achieve gas exchange by a mechanism similar to the intriguing phenomenon of high frequency ventilation (HFV).

There are three basic methods of this form of mechanical ventilation:

- High frequency positive pressure ventilation uses time-cycled conventional ventilation at a RR of 50–100/min.
- High frequency jet ventilation, the most widely available method, directs short rapid jets of gas through a nozzle into the airways and entrains air by the Venturi principle. Expiration is by passive recoil and a RR of 100–600/min is achieved.
- High frequency oscillation (HFO) forces mini-bursts of gas in and out of the airway. Both inspiration and expiration are active, which avoids the gas trapping that often occurs with passive exhalation (Hardinge, 1995). This can be superimposed on spontaneous breathing to reduce WOB or mobilize secretions (pp. 181, 202) or it can be the sole method of ventilation. Rates up to 4000/min are possible.

Mechanism

With such a meagre V_T, gas exchange is complex and cannot rely on bulk flow of gas. The classic concept of 'dead' space is no longer applicable, and this space is in fact thought to play an active part in gas exchange by the following mechanisms (Bower, 1995):

- High velocity flow creates turbulent mixing in the central airways, which is propagated peripherally by convective inspiratory flow.
- Gas mixing may occur by asynchronous filling and emptying of alveoli independent of each other, an effect known merrily as 'disco lung'.
- Molecular diffusion, the primary mechanism of normal gas exchange in terminal lung units, is augmented, especially by the vibrating gas of HFO.

Advantages

- Lung tissue is protected because alveoli are subject to minimal volume and pressure changes (Durbin, 1993).
- Spontaneous respiration is inhibited and little sedation is needed. Most patients find the sensation comfortable, as if being massaged from the inside.
- HFV provides an even distribution of ventilation because diffusion is independent of regional compliance and gas flow does not take the path of least resistance.
- Jet ventilation via minitracheostomy allows spontaneous respiration through the normal airway.

Disadvantages

- Shallow breaths hinder lung recruitment for oxygenation (Herridge and Slutsky, 1996).
- With jet ventilation, humidification is difficult, although possible with a hot-plate vaporizer.
- Except with HFO, high inspiratory flows and limited exhalation time create the risk of intrinsic PEEP (Herridge and Slutsky, 1996).
- Except with HFO, secretion clearance may be adversely affected (McEvoy *et al.*, 1982).
- HFV is noisy.

Indications

HFV tends to be used as a rescue mode when other techniques have failed. Enthusiasts consider the following to be indications:

- vulnerable lungs such as occur with ARDS
- bronchopleural fistula, large air leak, flail chest, acute head injury or unstable cardiovascular status, so long as low airway pressure is assured
- patients with an inordinate respiratory drive, or a need for minimum sedation
- patients with unilateral lung stiffness, using differential ventilation to each lung
- neonates, especially HFO.

Physiotherapy

Jet ventilation through a minitracheostomy allows patients to take deep breaths and cough. Suction can be performed without interruption of ventilation. Suction has less adverse effects on oxygenation or heart rhythm than IPPV. Manual hyperinflation is not possible and F_IO_2 must be increased by about 20% for 3 minutes before and after suction.

WEANING AND EXTUBATION

> ***'I was sure I would not be able to breathe on my own. The machine was put to a setting which gave me a couple of breaths and the rest was up to me. I hated that, I never knew when to take my breaths.'***
>
> Ludwig, 1984

Weaning should be a seamless process throughout the period of mechanical ventilation. The physiotherapist starts on the process straight away. After a preliminary rest, an exercise programme is initiated as the patient is able: on the bed, in sitting, standing or walking connected to a re-breathing bag. Systemic exercise has shown the following benefits:

- shorter ventilatory time
- improved weaning success
- better ADL status on discharge (Koll *et al.*, 1999).

The physiotherapist's contribution to the team management of weaning is to advise on the balance of rest and exercise, contribute to weaning decisions by assessing the breathing pattern, and occasionally to extubate the patient.

Weaning may be protracted for patients with chronic lung disease, neuropathies or for anyone after prolonged ventilatory support. Difficulties in weaning may be managed by transitory noninvasive ventilation or multidisciplinary rehabilitation (Merveille *et al.*, 1999). Liberation from the ventilator requires:

- progressive reduction in support until the patient is able to sustain spontaneous breathing
- a trial of spontaneous breathing through the tracheal tube
- extubation.

Criteria for weaning

Patients need the neuromuscular ability to breathe and adequate ability to oxygenate arterial blood (Bruton *et al.*, 1999). This can be identified by various criteria:

- correction of the underlying reason for IPPV
- maximum ventilatory reserve and optimum gas exchange, i.e. pH and P_aCO_2 related to the individual's premorbid state, plus vital capacity > 10 mL/kg, maximum inspiratory pressure > 20 cmH_2O, shunt < 15%, dead space < 60% of tidal volume, P_aO_2 > 11 kPa on F_IO_2 of 0.4 (P_aO_2/F_IO_2 ratio > 27.5).
- optimum nutrition, fluid, metabolic and cardiovascular status, including adequate haemoglobin levels, and no fever
- maximum, endurance, mobility and ability to cough
- optimum bronchodilation and clear airways
- restoration of normal diurnal rhythm
- previous night's uninterrupted sleep
- haemodynamic stability
- no abdominal distension
- reversal of sedation
- minimal pain
- absence of abdominal paradox and rapid shallow breathing (Howie, 1999).

The trend is away from set values and towards indices based on the breathing pattern. Patients who fail to wean tend to have a breathing pattern similar to that of acute respiratory failure (Rosario *et al.*, 1997). If a disturbed breathing pattern is ignored, exhaustion sets in (Figure 13.7). Pilbeam (1998, p. 327) has suggested the following test to identify if

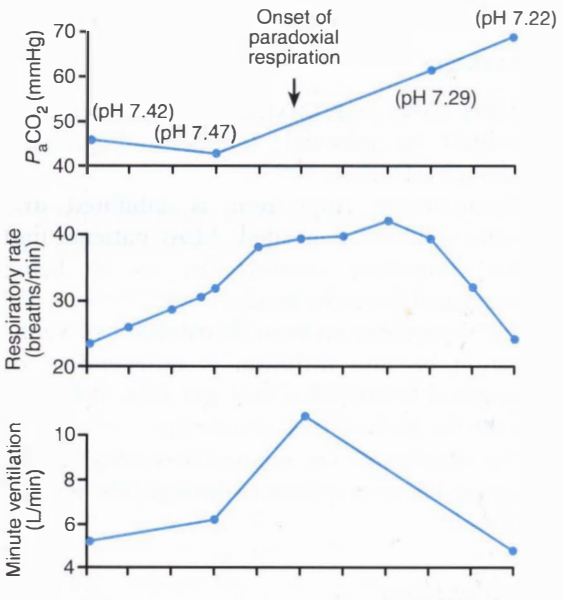


Figure 13.7 Sequence of changes as the diaphragm tires during a failed 20-minute attempt at weaning. Fatigue is represented at first by a ↓ P_aCO_2 and alkalosis, caused by ↑ respiratory rate and minute ventilation. As breaths become shallower, P_aCO_2 progressively rises and acidosis develops. As exhaustion sets in, respiratory rate drops rapidly. (From Cohen, C., Zagelbaum, G., Gross, D. *et al.* (1982) Clinical manifestations of inspiratory muscle fatigue. *Am. J. Med.*, **73**, 308–316)

patients are ready to breathe on their own:

- The patient is disconnected from the ventilator.
- Spontaneous breathing is maintained on the same F_IO_2, with CPAP equivalent to the previous PEEP, for 5 minutes.
- RR and V_T are measured, and a RR:V_T ratio of less than 100 suggests that weaning will be successful.

Weaning

Reduction in ventilatory support takes the form of periods of decreased number of breaths in SIMV mode or decreased pressure in PS mode. The following steps are then taken:

1. Explanations are given, with assurance that it is only a trial.
2. The patient takes up his or her preferred posture, usually sitting upright.
3. Humidified oxygen is connected to the tracheal tube by a T-piece, which allows high flows of oxygen without entrainment of room air. For breathless people, 30 cm of extension tubing attached to the exhalation side is required to prevent entry of room air. Oxygen flow should be high enough, and extension tubing long enough, to prevent interruption of the stream of mist that exits, even during inspiration.
4. The airway is suctioned if necessary.
5. The patient is disconnected from the ventilator, given oxygen, encouraged to breathe, and monitored for signs of laboured breathing, anxiety, desaturation, rising P_aCO_2, fatigue or drowsiness. A breathlessness visual analogue scale allows the patient to contribute to weaning decisions (Bouley, 1992). If the diaphragm tires, it may need 24 hours to recover, and it is better to return the patient to respiratory support than to await respiratory distress (Sykes and Young, 1999, p. 238).

Continuing problems may be due to weaning strategies that provide neither sufficient muscle work nor sufficient rest. This leads to muscle atrophy and/or perpetual muscle fatigue. Patients undergoing protracted weaning are best given adequate ventilatory support at night and short periods of significant work during the day (MacIntyre, 1995). The principles of balanced rest and exercise are illustrated by the following studies:

- Schönhofer *et al.* (1996) found that individually adjusted CMV, interrupted by spontaneous breathing, was superior to IMV or PS weaning.
- Esteban (1995) found that intermittent spontaneous breathing led to extubation three times as quickly as gradually decreasing IMV and twice as quickly as gradual PS weaning.

Other factors to consider are:

- Spending over 30 minutes on a T-piece alone can lead to atelectasis (Singer and Webb, 1997, p. 14) because of high WOB and loss of 'physiological PEEP' provided by the larynx, whose resistance prevents exhaled air rushing out too quickly.
- The inspiratory resistance caused by an HME may be significant, and weak patients may be more weanable with a hot-water humidifier (Bourdelles *et al.*, 1996).

Inadequate nutrition is a common cause of weaning failure. Basili (1981) showed how 93% of adequately nourished people could be weaned compared to only 55% of inadequately nourished people. Some patients require a period of 'nutritional restitution' in which weaning is postponed while they are fed up to 1½ times their normal requirements (DeMeo, 1992), so long as overloading with carbohydrates is avoided (p. 335).

Other difficulties may be due to undetected diaphragmatic paralysis, obstructive sleep apnoea (Noureddine, 1996), claustrophobia or fear of suffocation. Fears are managed by providing information and truthful reassurance that the patient can return to the ventilator by request at any time.

Weaning success can be improved with biofeedback using oximetry (Holliday, 1999),

tidal volume monitoring (Jacavone, 1998) or relaxation (Acosta, 1988). Another form of biofeedback is to connect the patient briefly to a re-breathing bag with an open valve, which patients watch to reassure them that they can breathe and are in control. A bedside fan may decrease breathlessness.

Extended use of a CPAP mode is only helpful if there are narrow airways or poor gas exchange, but is not indicated routinely (Bailey, 1995) because it tends to impose an additional workload (Patel *et al.*, 1999).

There is some evidence that strategies to enhance respiratory muscle endurance may facilitate weaning (Rosario *et al.*, 1997), and inspiratory muscle training can be used to provide brief periods of exertion alternating with rest (Figure 13.8).

Criteria for extubation

The ETT should be removed as soon as possible, so long as the following criteria are met:

- The patient can maintain a patent airway.
- The patient can protect the airway from aspiration.
- The patient can maintain a clear chest.
- The reason for intubation has been alleviated.

The ability to sustain a head lift has been suggested as indicating sufficient strength to protect the airway (Tobin and Yang, 1990). The cough response can be assessed by asking the patient to cough or by gently stimulating the upper airway with a catheter. Patients at risk need testing to ensure they have a peak cough flow above 3 L/s (Bach and Haas, 1996, p. 423) and a speech–language assessment. If there is no leak when the cuff is deflated, post-extubation stridor is a danger (Marik 1996a). Algorithms and details of criteria can assist extubation decisions (Maxam-Moore, 1996; Campbell 1999b).

A sustainable 30–60 minutes of spontaneous ventilation suggests that the patient is ready for extubation (Laghi, 1995), unless IPPV has been prolonged.

Extubation

If sputum retention is anticipated, it may be better to request a minitracheostomy as prophylaxis rather than await respiratory distress. The steps for extubation are the following:

1. Give physiotherapy if indicated, or simply suction the airway. Check for a cough reflex.
2. Ensure that re-intubation equipment and personnel are available.

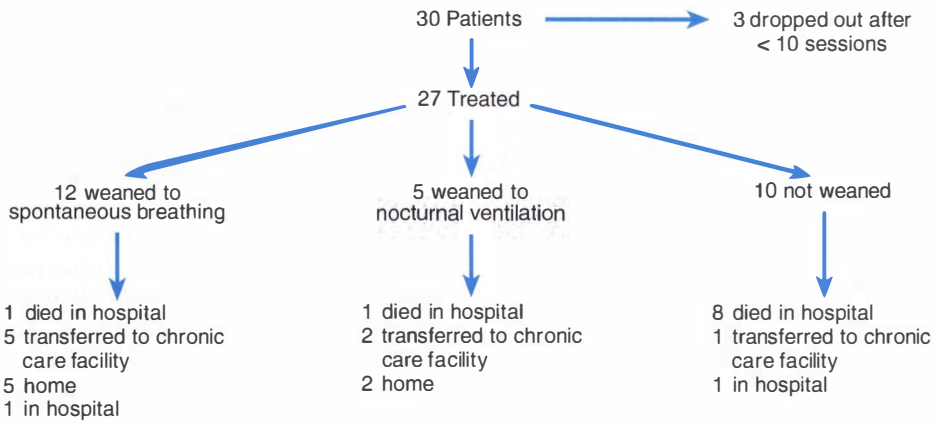


Figure 13.8 The effect of inspiratory muscle training in 30 ventilator-dependent patients. (From Aldrich, T. K. (1989) Weaning from mechanical ventilation. *Crit. Care Med.*, **17**, 143–147, with permission.)

3. Sit the patient upright.
4. Explain how the tube will be removed and that some hoarseness is commonplace afterwards.
5. Suck out the mouth and throat to clear secretions that have pooled above the inflated cuff.
6. Cut the tape holding the tube in place, insert a fresh catheter to reach just distal to the tip of the tube, deflate the cuff, slide the tube out in a gentle downward curve, at peak inspiration when the vocal cords are dilated, suctioning during withdrawal.
7. Encourage the patient to cough out secretions that have accumulated around the end of the tube. If this is impossible, bag-squeeze while deflating the cuff, which forces secretions into the mouth from above the cuff.
8. Give oxygen, non-invasive ventilation (Girault, 1999) or other support, observe monitors and breathing pattern, listen for stridor.
9. Enjoy patient's delight at their renewed voice.

Decannulation of tracheostomy

Weaning for tracheostomied patients can incorporate an intermediate step of replacing the cuffed tube with an uncuffed or fenestrated tube, which can be plugged for increasingly longer periods to test for adequate breathing and coughing.

When the tube has been removed, the patient is taught to hold a sterile dressing over the stoma when coughing. Delayed decannulation increases the risk of exacerbation in COPD patients (Clini *et al.*, 1999). For those leaving the ICU with a tracheostomy, a removable inner tube is essential in case of blockage.

MINI CASE STUDY: MS CM

Identify the problems of this 58-year-old woman who has been admitted for mechanical ventilation because of apnoea of unknown cause.

Background

RMH: Several admissions for IPPV.

Nurse report

Patient needs regular reminders to breathe.

Subjective

I hate this tube in my throat.

Objective

Intubated, on CPAP.
Patient alert, in side-lying.
Vital signs, auscultation and X-ray normal.

Day 2

Diagnosed with Ondine's curse.

Questions

1. Does the patient have a problem with impaired oxygenation?
2. Does the patient have a problem with impaired ventilation?
3. Does the patient have a problem with her inspiratory muscles?
4. Does the patient have a problem with her respiratory pump?
5. Is the mode of ventilation suitable?
6. Goals?
7. Plan?

Ondine's curse = apnoea caused by loss of automatic control of respiration, usually due to defective chemoreceptor responsiveness secondary to neurological or other disorder.

RESPONSE TO MINI CASE STUDY

1. No, the lungs are clear, observations and X-ray normal and there is no predisposing history such as surgery or respiratory disease.
2. Yes, spontaneous breathing is inadequate.
3. No, she is not complaining of fatigue or breathlessness and she is able to breathe when prompted.

4. Yes, the diagnosis implicates a component of her respiratory pump.
5. No, CPAP supports oxygenation, not ventilation. Ms CM needs a mode that provides mandatory breaths if she does not breathe, e.g. SIMV.
6. Goals: maintain function while short- and long-term management is organized, rehabilitate.
7. Plan:

- Liaise with team re mode of ventilation.
- Check patient's understanding of diagnosis.
- Mobilize patient fully, including outside if possible, with nurse and equipment.
- Negotiate with patient and nurse a daily exercise programme of getting dressed, sitting out and walking.
- Liaise with physiotherapist in referral centre to which patient will be sent for long-term home ventilatory planning.

LITERATURE APPRAISAL

The following statements were made to justify therapeutic percussion over rib fractures, thoracic abrasions and lung contusion. Comment on the logic (1–4) and the conclusion (5).

> *1. ... coughing causes more pain and greater alterations in intrathoracic pressure than properly performed percussion ....*
> *2. ... more than 406 patients have received chest wall percussion ....*
> *3. ... lung abscess and lung contusion are indications for chest physical therapy.*
> *4. A 42% mortality is reported following lung contusion with a flail chest.*
> *5. ... there was no statistically significant difference between the patients who did and did not receive manual percussion ....*
>
> *Phys. Ther. Pract.* 1994; 3: 92–108

RESPONSE TO LITERATURE APPRAISAL

1. Comparing percussion to coughing is not relevant and does not itself justify chest percussion.
2. Oh well, that's OK then.
3. Lung abscess and lung contusion are different. The first may be an indication for physiotherapy and the second may be a contraindication.
4. The fact that a condition has a high mortality does not itself justify physiotherapy, unless there is evidence that physiotherapy could reduce this mortality.
5. Well, well.

RECOMMENDED READING

AARC Clinical Practice Guidelines (1999) Removal of the endotracheal tube. *Respir. Care*, **44**, 85–90.

Alvisi, R. and Volta, C. A. (2000) Predictors of weaning outcome in COPD patients. *Eur. Resp. J.*, **15**, 656–662.

Bruton, A., Conway, J. and Holgate, S. T. (1999) Weaning adults from mechanical ventilation. *Physiotherapy*, **85**, 652–661.

Hawker, F. F. (1996) PEEP and CPAP. *Curr. Anaesth. Crit. Care*, **7**, 236–242.

Jantz, M. A. and Pierson, D. J. (1994) Pneumothorax and barotrauma. *Clin. Chest Med.*, **15**(1), 75–92.

Juniper, M. (1999) Ventilator associated pneumonia. *Care Crit. Ill*, **15**, 198–201.

Tonelli, M. R. (1999) Withdrawing mechanical ventilation: conflicts and consensus. *Respir. Care*, **44**, 1383–1387.

14 Physiotherapy for patients in intensive care

Summary

- Assessment
- Charts
- Patient
- Monitors
- Ventilator
- Imaging

Handling patients who are critically ill

- Minimizing oxygen consumption
- Turning
- Handling unconscious or paralysed people
- Pressure area care

Techniques to increase lung volume

- Positioning
- Deep breathing on the ventilator
- Manual hyperinflation

Techniques to clear secretions

- Postural drainage
- Manual techniques
- Suction

Exercise and rehabilitation

- Exercises
- Mobilization
- Transfer from ICU

Recognition and management of emergencies

- Cardiac arrest
- Respiratory arrest
- Seizure
- Haemorrhage
- Massive haemoptysis
- Cardiac tamponade
- Tension pneumothorax
- Traumatic pneumothorax
- Pulmonary embolism
- Air embolism
- Equipment malfunction or disconnection
- Patient distress on IPPV

On calls

Mini case study

Literature appraisal

Recommended reading

Assessment

> *'No-one explained ... all they said was not to worry about it.'*
>
> Thomson, 1973

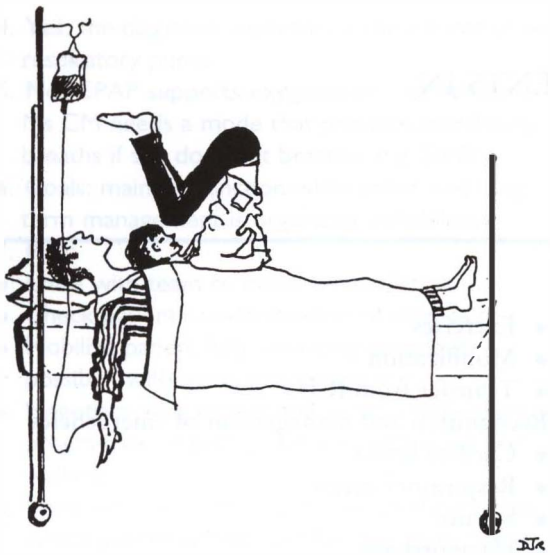
Assessment is required before, during and after treatment, especially if patients are unable to complain of new symptoms. The sequence of assessment described in Chapter 2 is used, with additions described below.

Charts

Sedation may be charted (Box 14.1) but there is no reliable means of assessing awareness (Coursin and Coursin, 1998). If the chart indicates pyrexia, the patient is consuming extra oxygen, but moderately pyrexial patients should not be actively cooled unless they have acute head injury or significant hypoxaemia (Gozzoli,

Box 14.1 Addenbrooke's Sedation Score (Shelly, 1998)

0	Agitated
1	Awake
2	Roused by voice
3	Roused by suction
4	Unrousable
5	Paralysed
6	Asleep



'The physiotherapist will come and do exercises on his chest'. From *ACPRC Newsletter*, 11, 1987, with permission.

2001). Pulmonary artery blood temperature is the gold standard for measurement of core body temperature. Peripheral temperature is measured by a probe on the toe, and if it is more than 5° lower than the core temperature, poor circulation is implicated. Survival is unlikely if peripheral temperature is less than 27°C (Joly and Weil, 1969).

Blood pressure should be checked for any response to previous sessions of manual hyperinflation (MH). If BP is low, unstable or sags on inspiration, or if mean arterial pressure is less than 60 mmHg, the patient may be unable to maintain cardiac output during MH.

Fluid status may be measured by daily weight, a change of more than 250 g/day suggesting fluid gain or loss (Parker and Middleton, 1993). The fluid balance chart is affected by a multitude of factors but is still a useful guide. Electrolyte and haematocrit values are decreased with fluid excess and increased with fluid loss. Fluids and electrolytes are disturbed by diuretics, diabetes, vomiting, diarrhoea, heart or kidney failure, burns, ascites or large open wounds. The signs of hypovolaemia are:

- ↑ HR
- ↑ RR
- ↓ vascular pressures (CVP, PAWP)
- ↓ systolic BP
- pallor
- ↓ urine output
- ↓ pulse pressure.

Hypovolaemia reduces pulse pressure because compensation by reactive vasoconstriction assists venous return and helps maintain diastolic pressure, so that in the early stages systolic pressure drops faster than diastolic pressure.

Extended clotting time is apparent for patients on anticoagulants or with DIC (p. 408) and increases the risk of bleeding during suction. Conversely, hypercoagulable states increase the risk of deep vein thrombosis (DVT) in a group of patients already at high risk (Saint and Matthay, 1998). Hypercoagulation is increased with malignancy, dehydration and pain (Shapiro *et al.*, 1995). Details of clotting parameters are in the Glossary.

Low serum albumin is common in ICU patients because of fluid and permeability problems. This is associated with reduced surfactant, poor wound healing and a drop in osmotic pressure, leading sometimes to peripheral and pulmonary oedema. Potassium levels below 4 mmol/L predispose patients to arrhythmias, contraindicating most forms of physiotherapy. Neutropenia can be caused by poor nutrition, immune disorders or anticancer drugs, and is associated with vulnerability to infection and sepsis.

Patient

> ***'I could think and I could hear, but I could not move and I could not talk or open my eyes.'***
>
> Lawrence, 1995b

Is the patient conscious, confused, agitated, sedated, paralysed? Paralysis, whether pathological or pharmacological, indicates the importance of clarity in communication because patients

may be trying to make sense of sounds and sensations but cannot give feedback. Unconscious patients hear and understand more than we expect (Lawrence 1995b; Sisson, 1990). What channels of communication are available? Is perception or interpretation of information altered by drugs or cerebral damage?

Agitation may be due to the endotracheal tube, fear, lack of information, incorrect ventilator settings, restraints, awkward positioning, pain or gut distension. For ventilator-related distress, see page 386. Other points to note before treatment are:

- Accessory muscle activity suggests excess work of breathing (WOB), and laboured breathing may indicate an obstructed airway.
- Lines and tubes, including femoral lines, haemofiltration lines, pacing wires and lines in the feet, should be kept in view throughout treatment.
- Hydration is difficult to assess clinically because oedema or overhydration can coexist with intravascular depletion in critically ill people (Dobb and Coombs, 1987).
- Vasoconstriction or low cardiac output is indicated clinically by cold hands.

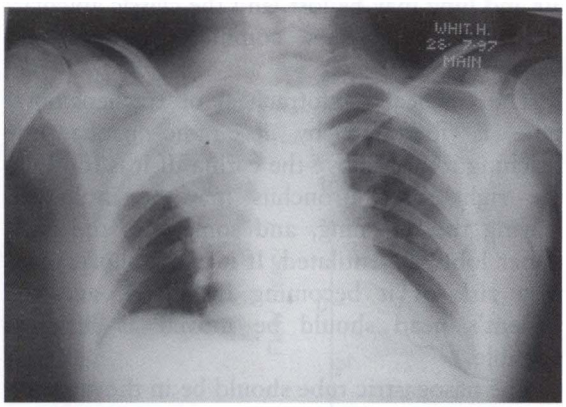


Figure 14.1 This endotracheal tube has passed into the right main bronchus and beyond the right upper lobe bronchus. The right upper lobe is not being ventilated and is collapsing as its air is absorbed. Breath sounds would be reduced and the percussion note dull. Physiotherapy is contraindicated until the tube has been repositioned.

- In side-lying, the dependent compressed lung generates more dullness to percussion than the upper lung (Gilbert, 1989).
- Apical breath sounds should be scrutinized to pick up problems such as a pneumothorax, haemothorax or intubated right main bronchus (Figure 14.1).
- The stethoscope may pick up the wheeze-like sound of air leaking around the cuff of the tracheal tube, or the bubbling of condensed humidifier water in the tubing.
- If manual ventilation is undertaken, breath sounds can be heard more clearly, and sometimes crackles can be elicited with a sharp release on expiration.

Clinical signs of decreasing cardiac output are the following:

- pale or dusky colour
- cold extremities
- sweating
- dizziness with position change
- confusion or altered consciousness
- ↑ HR
- ↑ RR
- ↓ P_aCO_2
- ↓ urine output.

Monitors

Hypoxaemia can precipitate arrhythmias. If S_aO_2 falls, treatment should be halted until it stabilizes, and/or F_IO_2 should be increased. $S_{\bar{v}}O_2$ can be used to monitor the effect of procedures such as suction, which reduces oxygen supply.

Changes in BP and HR reflect factors as diverse as septicaemia, pain, drugs or fluid status. Monitors should be observed during treatment in order to identify responses relating to physiotherapy. Systolic pressures as low as 80 mmHg may be adequate so long as this is normal for the patient and the patient is warm and passing sufficient urine, but caution must be exercised if MH is necessary.

Spontaneous arrhythmias occur in 78% of patients (Artucio and Pereira, 1990), but those caused by physiotherapy can be identified by ECG changes during treatment.

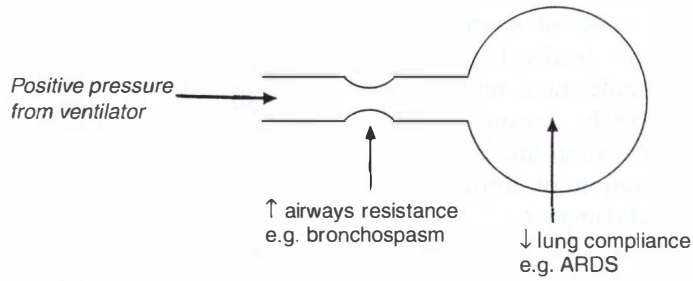


Figure 14.2 Causes of increased peak airway pressure for patients on volume-control ventilation. *ARDS = acute respiratory distress syndrome.*

Ventilator

Charts indicate ventilator settings and trends in the patient's response, while the machine indicates what is occurring from moment to moment. In volume control, airway pressure provides the following information:

- Peak pressure is normally 20 cmH_2O.
- Values above 30 cmH_2O may be due to airflow obstruction, stiff lungs, pulmonary oedema, pneumothorax or obstruction by upper airway secretions, a kinked tube or clenched teeth (Figure 14.2).
- Peak pressure below normal is due to a leak in the circuit.
- Oscillation in airway pressure signifies spontaneous breaths between ventilator breaths.
- Erratic readings indicate a patient fighting the ventilator or coughing, which can be confirmed by observation.
- Readings that dip substantially below the end-expiratory baseline suggest excess WOB.
- The complete cycle is raised in proportion to the level of PEEP.

Alveolar pressure is not equal to airway pressure readings, unless there is zero flow, because resistance to gas flow in the airways creates a pressure gradient. Alveolar pressure is more negative than airway pressure during patient triggering and more positive during a positive-pressure breath. A high level of PEEP means that patients are at risk of severe hypoxaemia if they are disconnected from the ventilator. A saw-tooth pattern on the flow–volume curve may indicate excess secretions (Figure 14.3).

Imaging

Portable X-rays are taken with the patient supine or sitting up as able. A supine or slumped position causes a pleural effusion to lose its clear boundary (Figure 14.4). Pleural effusions are common and not well tolerated in the ICU, contributing to hypoxaemia and sometimes cardiovascular instability (Mattison *et al.*, 1997). A third of pneumothoraces are not clearly seen in a supine film because the boundary between air and lung may be lost, and the classic apicolateral location is less common (Juniper and Garrard, 1997).

The tracheal (endotracheal or tracheostomy) tube is identifed by its radio-opaque line reaching to just above the carina. If it is too long the right main bronchus may be intubated, leaving the left lung, and sometimes the right upper lobe, unventilated. If it is too short, there is a risk of it becoming dislodged, and the patient's head should be moved as little as possible.

The nasogastric tube should be in the stomach and not the lung. A central venous line is usually traceable to the vena cava. A pulmonary artery catheter passes through the heart in a loop, with its tip in a branch of the pulmonary artery.

Films before and after initiation of high levels of PEEP may show an apparent clearing of infil-

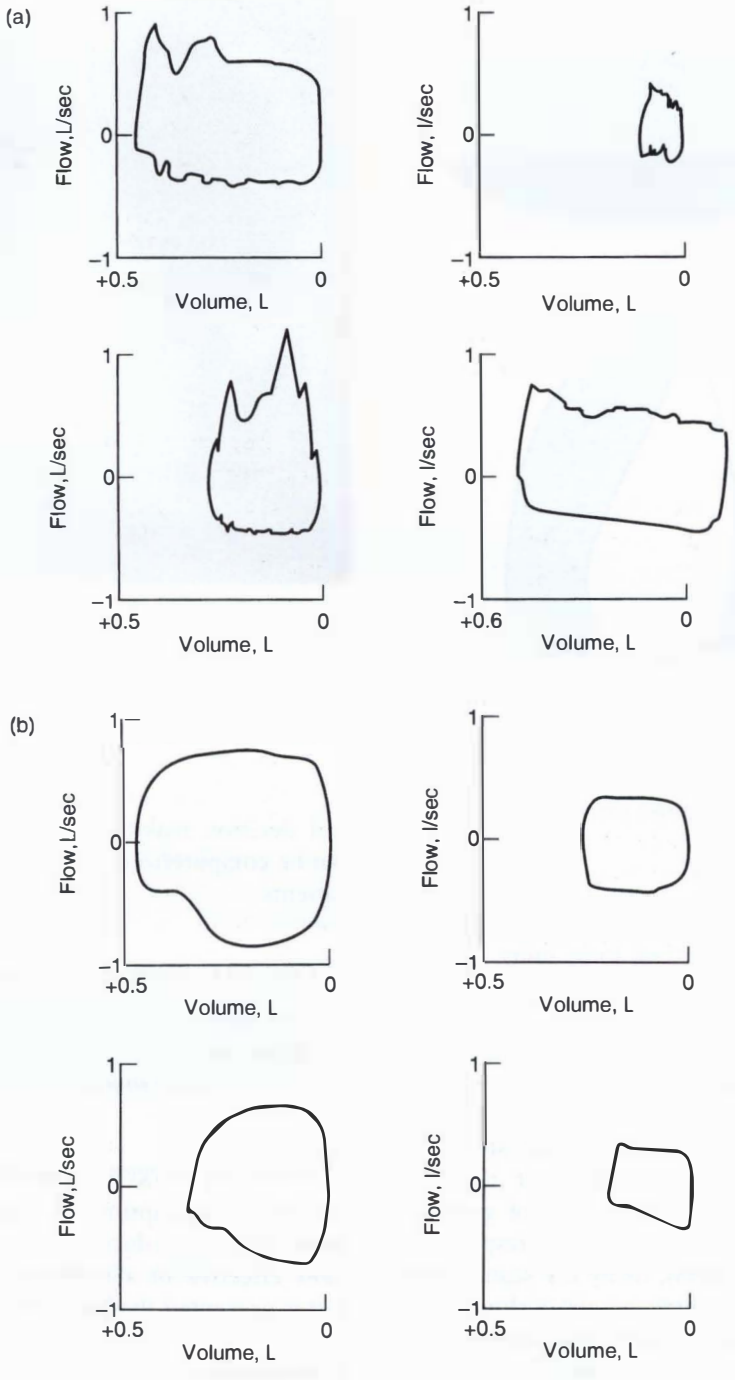


Figure 14.3 Flow–volume loops. (a) Jagged curves indicate the presence of secretions. (b) Smooth curves indicate clear airways. (From Jubran, A. (1994) Use of flow–volume curves in detecting secretions in ventilator dependent patients. *American Journal of Respiratory and Critical Care Medicine*, **150**, 766–769)

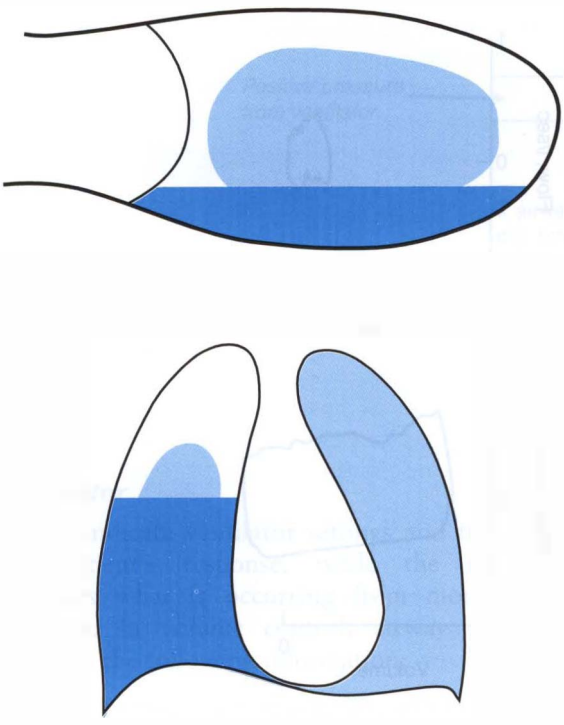


Figure 14.4 Representation of a pleural effusion on x-ray. Top picture of a supine patient would show a diffuse ipacity. Bottom picture of an upright patient would show a clear fluid line

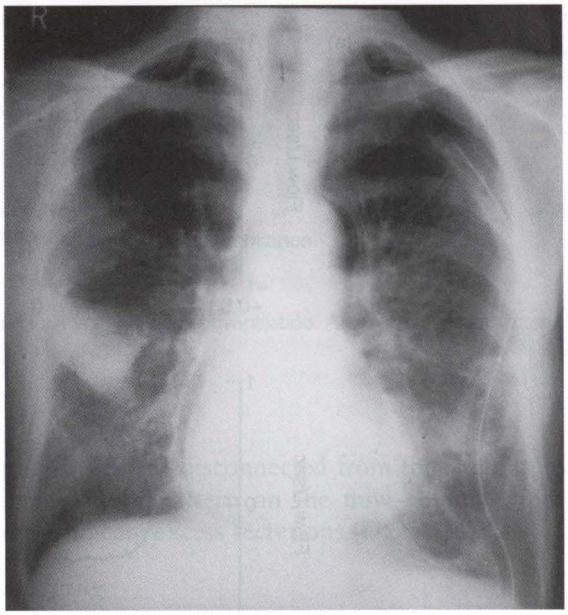


Figure 14.5 Evidence of barotrauma in a ventilated patient. Air is evident in the mediastinum and pericardium, and the chest drain on the patient's left indicates that there has been a pneumothorax. The opacity on the patient's right is a loculated pleural effusion.

trates through redistribution of pulmonary oedema and recruitment of lung units (Ely *et al.*, 1996).

Early signs of barotrauma are difficult to detect radiologically. Pulmonary interstitial emphysema can usually be identified only in neonates (small cystic lucencies, perivascular cuff of air around vessels and linear streaking towards the hilum), or in adults if there is contrast provided by a background of generalized opacification such as acute respiratory distress syndrome (ARDS), or by CT scan. Pneumomediastinum may show as air outlining the mediastinum or a 'continuous diaphragm' extending from one hemidiaphragm to the other below the heart. Pneumopericardium presents as a lucency or halo around the heart (Figure 14.5).

Box 14.2 provides guidance on assessment and decision making for the ICU patient. This can be computerized for adaptation to individual patients.

HANDLING PATIENTS WHO ARE CRITICALLY ILL

> ***Who am I?***
> ***Where am I?***
> ***Why do I hurt so much?***
>
> *Nursing Times*, 1981

Minimizing oxygen consumption

Oxygen consumption is increased by stress. Motivation is reduced by stress. Treatment is most effective in a motivated patient. Stress is better prevented than treated.

Preliminaries

> ***'Someone would come near me and would just be working and not saying anything to me. That would be frightening because I***

Box 14.2 ICU clinical reasoning model (V. Bastow, S. Randall and A. Ludlow, Queen Elizabeth Hospital, Kings Lynn, with modifications)

Precautions
MRSA status: +ve/–ve/unknown in sputum Y/N
Clotting status
Other

Assessment
Nurse comments

Subjective assessment

Charts
- Pain score Sedation score GCS
- Temp BP HR RR S_aO_2
- CVS stable Y/N
- ABGs on F_IO_2 ____ : pH P_aO_2 P_aCO_2 HCO_3^- BE
  Acidosis/alkalosis/
  Respiratory/metabolic
  Acute/compensated
- Fluid balance
- Relevant medication
- Other

Ventilation
- F_IO_2
- Self-ventilating Y/N Breathing pattern
- NIV Y/N Mode
- IPPV Y/N VC/PC Peak pressure if on VC
- Tidal volume
- Mode SIMV PS SIMV+PS Other
- PEEP
- Patient triggering Y/N
- Humidifier/HME

CXR date Radiology report/own interpretation

Clinical assessment
Appearance
Auscultation breath sounds added sounds
Abdominal distension Y/N
Percussion note
Other

Continued overleaf

Additional respiratory information

Additional non-respiratory information

Indication(s) to treat	**Precautions/reasons not to treat**

Treatment
Positioning
Breathing techniques: DB IS ACB/AD Cough Other
MH
Vibs/shaking/percussion
Saline instillation Y/N mL
Suction
Ex: PMs Act/assisted Active SOOB Mob
Other

Outcomes (document changes only)
Auscultation: Breath sounds Added sounds
S_aO_2
Secretions
Peak pressure (VC)
CVS stability
ROM
Mob
CXR
Other

Plan (including instructions for patient/family/staff)

ABGs = arterial blood gases; ACB = active cycle of breathing; AD = autogenic drainage; CVS = cardiovascular system; DB = deep breathing; F_IO_2 = fraction of inspired oxygen; GCS = Glasgow Coma Scale; HCO_3^- = bicarbonate; HME = heat-moisture exchanger; IS = incentive spirometry; MH = manual hyperinflation; NIV = non-invasive ventilation; PC = pressure control; PMs = passive movements; ROM = range of movement; SOOB = sit out of bed; VC = volume control.

> ***didn't know what they were going to do next.***
>
> Parker *et al.*, 1984

The traditional protocol of turning, percussion, vibration and suction can release stress hormones, destabilize cardiac output, increase oxygen consumption by over 50%, raise BP and HR, increase $P_{A-a}O_2$ and reduce P_aO_2 (Weissman, 1993). This varies with how patients are handled, but it is a reminder to avoid any intervention that is unnecessary. All patients need explanations, and some need sedation (Horiuchi, 1995), increased F_IO_2 before treatment, or extra pressure support (Kemper, 1993). All patients, including those who are unconscious or paralysed, need warning before all procedures and any physical contact. Without this, fear can further increases oxygen consumption (Turnock, 1997). Fluids and medication may be required to ensure adequate cardiac output and minimum pain.

Orientation

Patients need a visible clock, calendar, family photographs, personal belongings in an area that they can control, information on progress, interpretation of noises and voices, attendance to alarms promptly, explanations of neighbours' alarms, a personal telephone if they are able to talk, trips outside the unit when possible, and treatment with the same physiotherapist before, during and after admission to the ICU when feasible. We should enter the patient's space gently, introduce ourselves and explain our purpose.

Sleep and rest

> *Sleep ...*
> *Balm of hurt minds, great Nature's second course,*
> *Chief nourisher in life's feast.*
>
> William Shakespeare, *Macbeth*

Patients should not, if possible, be woken when asleep, especially if flickering eyelids indicate that they are in the REM phase of the sleep cycle when tissue regeneration is at its maximum (Shelly, 1992).

Family

If visitors are present, they can either be invited to stay or asked to leave during treatment, depending on the patient's wish. The presence of relatives means that they can become involved in patient care and are reassured that physiotherapy is not distressing.

Communication

> *'So many believe that because you are unable to talk, you either can't or don't want to listen.'*
>
> Holden, 1980

The priority in stress reduction is to establish communication. Understanding and memory may be affected by anxiety or drugs, but patients who are unable to speak are neither deaf nor mentally impaired. Aids to communication include:

- the patient's understanding of why they cannot speak and how long this is expected to last, as they have often forgotten or may not have been told
- clear and explicit explanations, repeated as necessary, including why physiotherapy is necessary, what it will feel like, how long it will last and instructions on how to ask for it to stop
- hearing aid or glasses if used
- if lip-reading proves inadequate, communication aids such as word or picture charts, pencil and paper, or for greater privacy a magic slate
- when appropriate, a speaking tracheostomy tube for adults (p. 280) or children (Tucker, 1991)
- referral to a speech–language therapist when appropriate
- if unable to write or use charts, yes-or-no questions asked one at a time, e.g.: 'Are you hot? cold? itchy? worried? tired? sleepy? nauseous? in pain? Is your mouth dry? the tube bothering you? Do you want to turn? raise or lower your head? Do you need more air? less light? less noise? more information? bottle or bedpan?'

Communication should be aimed *at* patients rather than *over* them. Chatting over patients can increase stress more than suction (Lynch, 1978). One patient said 'It didn't matter what they talked about, so long as they talked to me' (Villaire, 1995). Other patients found that their attempts at communication were met by being told to relax or being given a sedative (Jablonski, 1994). If a patient wishes not to communicate, this should also be respected.

> *'The most important thing for me was the human contact, the communication.'*
>
> Villaire, 1995

Helplessness

> *'What do you do when you can't bear it? There is only one thing to do: bear it ...*

what else are you going to do? What are the alternatives?'

Rollin, 1976

Helplessness can lead to depression (Jones and O'Donnell, 1994). The more helpless the patient, the more important is autonomy. Patients can choose whether they would like treatment now or later (if possible), whether the bedhead is the right height. They can have charge of the TV remote and radio channel, if required, and decide whether they would like to regain their day/night rhythm by being woken in the day or having a sleeping pill at night. Autonomy is particularly important in this situation of unequal power. Depression is eased by allowing expression of emotion and encouraging independence and decision-making. Anxiety is reduced by combining factual information with advice that enables patients to be proactive (Jones and O'Donnell, 1994). Benefits have been found with imaginative interventions such as a pet visitation scheme (Giuliano *et al.*, 2000).

Touch

'It surprised me how much I valued human touch.'

Redfern, 1985

ICU patients are extra-sensitive to human physical contact as a contrast to the cold clinical procedures to which they are frequently subjected. Therapeutic touch assists relaxation and sleep (Cox and Hayes, 1999) and foot massage is accessible for the ICU patient and can reduce tension and lower RR (Stevensen, 1994). The benefits of even brief massage can be confirmed by watching the monitors. As always, it should be remembered that individuals vary and some dislike touch.

Comfort

Measures to alleviate physical discomfort include regular position change, before the allotted time if the patient requests.

Self-esteem

Patients should not be expected or coerced into

being 'popular' at a time when they least need such a burden. Praise is a potent motivator and enhances self-esteem.

'Hearing remained acute and was the primary means of receiving information from the environment. "I heard a lot more than I think they think I heard".'

Jablonski, 1994

Turning

'To be talked frankly through a complete procedure, particularly its estimated length, would help curb the deadly effects of uninformed anticipation.'

Brooks, 1990

During turning, transient changes in vital signs are acceptable, but if S_aO_2 drops and does not return to its usual value within 5 minutes, HR increases or decreases by over 10 bpm and does not settle, or $S_{\bar{v}}O_2$ varies as described on page 330, the patient should be returned gently to the previous position.

A suggested sequence for turning is the following.

1. Inform patient
2. Turn off continuous tube feedings.
3. Ensure sufficient slack in lines and tubes.

4. Clear ventilator tubing of any accumulated water that could spill into the patient's airway.
5. Ensure that glide sheets are in place, the team is following the same manual handling protocol and individuals are responsible for the airway and vulnerable lines.
6. Ensure that the team is co-ordinated in care of the skin and joints (e.g. protect heels from friction, prevent hip strain by avoiding use of the leg as a passive lever).
7. Support the tracheal tube. Some trusted patients can hold an endotracheal tube briefly with their teeth during the turn.
8. Say clearly, so that the team and patient can hear, agreed instructions, e.g. 'Ready, steady, turn'.
9. Turn smoothly.
10. Check lines, patient comfort and monitors.

Handling unconscious or paralysed people

> *'You can't do nothing except lay there in one position. That's very very uncomfortable.'*
>
> Jablonski, 1994

We need to act as the consciousness of the unconscious. It is easy to depersonalize patients who cannot respond to us, especially if we have not had the opportunity to get to know them when they were alert. When handling paralysed or unconscious people, attention should be given to:

- protecting the eyes
- supporting the head in a neutral position
- reassuring the patient that s/he will not fall off the bed
- when positioned, aligning the limbs and spine in neutral positions, with special care of the shoulder joint (which has no stability without muscle tone) and ulnar nerve (stretched with combined elbow flexion and forearm pronation)
- in supine, positioning the upper limbs with palms downwards
- in side-lying, checking that the ear is not twisted under the head
- clearing objects or creases from under the patient.

Pressure area care

Pressure sores are found in a third to a half of ICU patients (Peerless *et al.*, 1999) and cost the NHS a billion pounds a year plus litigation expenses (Allen, 1998). Each grade 4 sore causes on average 17 other patients to forgo a bed. Pressure sores distress people, kill people and are avoidable (Kiernan, 1998). Risk factors are malnutrition, obesity, steroids, vasopressor drugs, diabetes, advanced age and restricted movement due to traction, support systems or patient instability.

Anything can be put on a pressure sore except the patient. Hospitals are full of concoctions for treating pressure sores, but better still is prevention, by means of:

- adequate nutrition (Russell, 2000), especially vitamin C and protein (Barratt, 1989)
- frequent turning and judicious positioning (Davies, 1994)
- pressure-reducing cushions on chairs (Collins, 1999) and specialized beds (Willis, 1996)
- keeping pressure areas dry
- turning without friction
- avoidance of excessive washing or rubbing with talc, cream or towels
- prevention of hypotension or hypovolaemia.

Pressure sore risk is reduced by a rotating bed, a low-air-loss bed or a thermoreactive mattress with squidgy foam. Some of these mattresses allow the patient to sink into a moulded well, which is unhelpful for mobility and respiratory care.

Most effective for skin care is an air-fluidized bed such as the Clinitron, whose silicon beads float the patient like a semi-submerged iceberg in a current of warm air. This lowers skin contact pressure to below capillary occlusion pressure, controls temperature and absorbs exudate into the beads. Points to note are the following:

- Good teamwork is needed to ensure regular

turning for preventive respiratory care, because turning is no longer required for pressure area care.
- The dry air current may contribute to dehydration.
- A hoist may be needed for mobilizing the patient out of bed.
- The bed should be switched off and unplugged for cardiopulmonary resuscitation.

A sacral pressure sore that has developed in supine does not preclude sitting out in a chair, so long as a pressure cushion is used and an upright position is maintained to prevent pressure on the sacrum.

TECHNIQUES TO INCREASE LUNG VOLUME

For spontaneously breathing patients, lung volume can be increased by the techniques discussed in Chapter 6. For ventilated patients, the following modifications can be incorporated.

Positioning

> ***There may be a fine irony in the observation that our advanced knowledge and technology by themselves cannot save the patient. Instead, something so simple as turning the patient from supine to lateral to prone to lateral, at least hourly, may make the difference between living and dying for the intensive care patient.***
>
> Bendixen, quoted by Ray, 1974

Positioning is the main physiotherapy treatment for patients in intensive care, and may be the only intervention for some patients. By preventing the abdominal contents encroaching on lung volume (p. 149), positioning restores ventilation to dependent lung regions more effectively than PEEP or large tidal volumes (Froese and Bryan, 1974). Simply turning from supine to side-lying can clear atelectasis from dependent regions (Brismar, 1985).

Side-lying increases FRC (Ibañez *et al.*, 1981) and enhances gas exchange (Lewandowski, 1992) compared to supine. It also allows easier movement against gravity for weak patients (Chung, 1992). The prone position is useful for some severely hypoxaemic patients (p. 415). As with spontaneously breathing patients, ventilated patients with unilateral lung pathology usually show optimal gas exchange when lying with the affected lung uppermost (Wong, 1999).

Factors that modify positioning are head trauma, abnormal muscle tone, pain, spinal cord injury, fractures, pressure sores, unstable BP and invasive support systems such as haemofiltration. Semi-recumbent positions protect against aspiration but not gastro-oesophageal reflux (Orozco-Levi *et al.*, 1995). Right-side-lying is more likely to impair cardiac output than left-side-lying in unstable patients (Bein *et al.*, 1996).

Kinetic rotating beds turn patients continually along the longitudinal axis, and are useful if they can rotate a full 180°. There are inconsistent reports of their benefits to the respiratory system. MacIntyre *et al.* (1999) found that they had little effect on respiration and increased patient anxiety, but reduced urine infections. Raoof *et al.* (1999) found them beneficial when combined with mechanical percussion.

Deep breathing on the ventilator

If patients are on a mode of ventilation that incorporates spontaneous breathing, they may be able to take deep breaths voluntarily. Deep breathing is particularly successful when patients are motivated by watching the results of their endeavours on the tidal volume monitor.

Manual hyperinflation

> ***'It was by far the most frightening thing that happened to me. I'll never forget it.'***
>
> Patient quoted by Rowbotham, 1990

Manual hyperinflation delivers extra volume and oxygen to the lungs via a bag such as a rebreathing bag. Compared to positioning, which is accepted as preventive care for most ICU patients, manual hyperinflation is not used routinely because prophylaxis has not been substantiated.

Terminology

- Manual ventilation means squeezing gas into the patient's lungs at tidal volume, e.g. when changing ventilator tubing.
- Manual hyperventilation delivers rapid breaths, e.g. if the patient is breathless, hypoxaemic or hypercapnic.
- Manual hyperinflation provides deep breaths in order to increase lung volume, e.g. when treating a person with atelectasis or sputum retention.

Physiotherapy is associated with manual hyperinflation (MH). The words 'bag-squeezing' or 'bagging' are also used, although it is best to avoid saying 'bagging' with patients as it can be misinterpreted (Waldmann and Gaine, 1996).

Effects

Beneficial effects of MH are:

- reversal of atelectasis (Lumb, 2000, p. 122)
- sustained improvement in lung compliance and oxygen saturation (Patman *et al.*, 1999)
- improved sputum clearance (Hodgson *et al.*, 2000).

Disadvantages are:

- haemodynamic and metabolic upset
- risk of barotrauma for certain patients
- discomfort and anxiety if done incorrectly.

Technique

A rebreathing bag is a rubber or plastic 2 or 3 L bag such as a Water's bag (Mapelson's C), connected by an adjustable expiratory valve to an oxygen supply (Figure 14.6). Its compliance allows the clinician to feel the ease of inflation. Non-rebreathing units such as the Ambu and Laerdal bag consist of semi-rigid material which self-inflate from room air with added oxygen. These prevent excessive pressures being reached, but are less responsive to modifications in technique.

The following method is recommended:

1. Ensure the patient's fluid and cardiovascular status are optimum to minimize any drop in cardiac output.

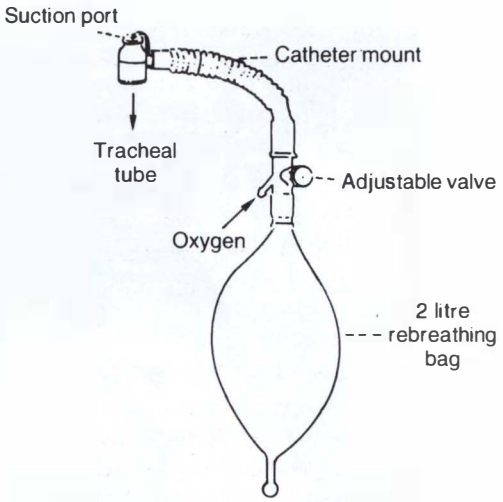


Figure 14.6 Water's bag system.

2. Ensure adequate analgesia and/or sedation.
3. Position the patient in well-forward-side-lying (Figure 14.7). The positive pressure of MH hyperinflates the more compliant upper regions so that in supine the bases are largely ignored. For patients who cannot turn, close attention to technique (see 8–9 below) may deliver some extra volume to the lung bases in supine. If a different area is to be targeted, it is placed uppermost.
4. Check monitors after the turn. MH should not be started until cardiovascular stability is assured in the new position.
5. Observe chest expansion.
6. Tell the patient that s/he will feel a deep breath. They will feel sleepy if Entonox is used. They should be free of distractions or nursing interventions.
7. Connect the bag, with heat-moisture-exchanger, to the oxygen with a flow rate of 15 L/min, turn off the low-pressure alarm, disconnect patient from the ventilator and connect him/her to the bagging circuit.
8. Rest tubing on the sheet to avoid tugging on the tracheal tube, tell the patient when to expect ordinary breaths and when deep breaths. Squeeze the bag a couple of times at tidal volume to acclimatize the patient and to

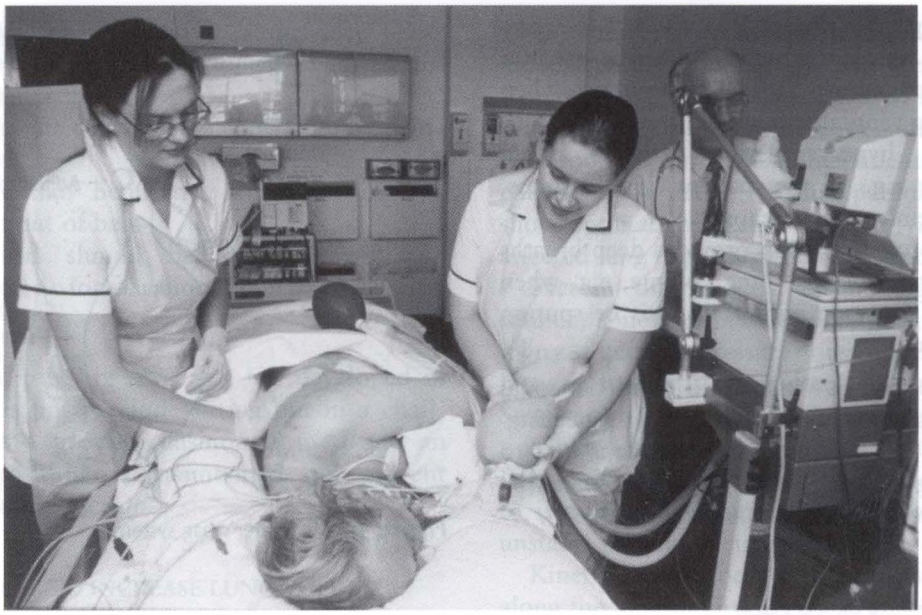


Figure 14.7 Manual hyperinflation targeting the left lower lobe, which is being palpated to check for optimum expansion. (Photograph: Nicholas Taylor.)

assess lung compliance, then give slow smooth deep breaths, adjusting the valve to increase pressure until expansion is greater than on IPPV and the manometer indicates a safe and effective pressure (see below). Slow inspiratory flows minimize turbulence and the risk of generating intrinsic PEEP (Reick, 1996).

9. Hold maximum pressure at end-inspiration for 1–2 seconds to encourage filling of poorly ventilated alveoli, especially if atelectasis is the problem. Haemodynamically unstable patients should not receive this end-inspiratory hold, and are best given one deep breath interspersed with several tidal breaths, or if the patient is able, interspersed with spontaneous breaths.
10. Release the bag sharply to simulate a huff, especially if secretion retention is the problem.
11. Watch the chest for expansion, the face for distress and the abdomen for signs of unwanted active expiration. The more alert the patient, the greater the need to co-ordinate the procedure with his/her breathing. Stop MH if the patient's facial expression or monitors indicate distress, or if crackles indicate that secretions have been mobilized and suction is required. If crackles are heard, give tidal volume breaths until the patient is suctioned. If MH causes no change, stop after 6–8 breaths for re-assessment.
12. After MH, inform the patient and reconnect to the ventilator.
13. Check that the alarm is on, observe chest movement and monitors, auscultate the chest.
14. To maintain the benefits of MH, the side-lying position should be continued so long as it is comfortable for the patient and convenient for nursing procedures.

Pressures

Each bed space should be supplied with its own manometer to ensure effective and safe pressures (Ellis *et al.*, 1999). The following are guidelines:

- For MH to be effective in normal lungs: a sustained inflation to 40 cmH_2O is required to reverse atelectasis (Novak *et al.*, 1987; Rothen, 1993).
- For MH to be safe in normal lungs: maximum pressures are 60 or 70 cmH_2O (Sommers, 1991; Eaton, 1984).
- For MH to be safe in diseased or damaged lungs: there is no safe limit, so if MH is necessary, the minimum effective pressure is used.

Mini literature appraisal

King and Morrell (1992) are frequently quoted as advising 40 cmH_2O for the maximum safe MH pressure. The authors did not explain that their sources referred to patients with damaged lungs. These findings should not be extrapolated to patients with normal lungs.

A useful teaching aid is to set up a test lung with a spare ventilator, set on spontaneous mode, high flow, and with a pressure–volume loop (p. 324) displayed. This will give feedback on the tidal volume and pressure attained with MH, including maintenance of manual PEEP. The screen can be frozen to identify details.

Complications

The complications of MH are an exaggeration of the complications of IPPV, particularly barotrauma and haemodynamic compromise. BP and cardiac output may rise (Stone, 1991) or fall (Singer *et al.*, 1994). Complications are greatest if MH uses large tidal volumes or is vigorous, but if too gentle it may lead to underventilation and hypoxaemia.

Contraindications

- Extra-alveolar air, e.g. undrained pneumothorax, bullae, surgical emphysema.
- Bronchospasm causing peak airway pressure above 40 cmH_2O.

Precautions and modifications

- Pneumothorax with a chest drain.
- Air leak as demonstrated by air bubbling through a chest drain bottle.
- BP that is low, high or unstable. If MH is essential in a hypotensive patient, the patient should be maximally stabilized first and the technique should be brief, with prolonged expiration and no end-inspiratory hold, in order to facilitate venous return.
- Hypovolaemia as demonstrated by low CVP/PAWP.
- Recent pneumonectomy because of the risk of bronchopleural fistula. The fifth to the 10th postoperative days are when the healing stump is at its most vulnerable (Pierson and Lakshminarayan, 1984).
- Acute head injury.
- Patients at risk of barotrauma, e.g. those with emphysema, acute asthma, fibrosis, Pneumocystis pneumonia or ARDS (Jantz and Pierson, 1994).
- Rib fracture because a covert pneumothorax might be present. If MH is essential, the X-ray should be scrutinized or a radiologist's opinion sought.
- During renal dialysis, which tends to destabilize BP.
- Arrhythmias or frequent ectopics.
- Hyperinflated lungs with intrinsic PEEP. If essential, a longer expiratory time might be required.
- During weaning if patients with hypercapnic COPD are dependent on their hypoxic drive to breathe. If MH is essential and the patient is not severely hypoxaemic, the bag can be connected to air instead of oxygen, the procedure kept brief and the monitors watched.
- Severe hypoxaemia with PEEP above 10 mmHg, because disconnection of the patient from the ventilator entails loss of PEEP. If MH is essential, desaturation can be minimized by:
  - incorporating a PEEP valve in the circuit, a method that has no evidence

base (Wainright and Gould, 1996) and can depress BP but which may benefit individuals
- manually preventing the bag fully deflating at end-expiration
- increasing the flow rate, and bagging faster to prevent deflation and augment oxygenation, but briefly, and only if this is safe in relation to the patient's haemodynamic status
- using the 'manual sigh' or inspiratory pause button, which may encourage some alveolar recruitment.

TECHNIQUES TO CLEAR SECRETIONS

The secretions of patients on IPPV can usually be cleared by humidification, regular position change, suction as required and MH if necessary. Jet nebulizers have shown a high instance of infection risk (p. 142–3) and greater compromise to oxygenation than a hot-water humidifier (Kuo *et al.*, 1991), so before 'saline nebs' are considered, it would be wise to ensure that continuous hot-water humidification is optimal.

Postural drainage

The head-down tilt is rarely suitable for patients on IPPV. Haemodynamics are compromised and abdominal contents weigh heavily on an inactive diaphragm. Side-to-side positioning, which is used for maintenance of lung volume, is usually adequate as modified postural drainage.

Manual techniques

Manual techniques are not required routinely and have no effect on resolving atelectasis (Denehy, 1999), but excessive or thick secretions may be an indication for percussion or vibrations, so long as there is no risk of arrhythmias (Hammon *et al.*, 1992). There are reports of atelectasis being caused by vibrations beyond FRC (Laws and McIntyre, 1969) but this should not occur if manual techniques are routinely followed by strategies to restore FRC, e.g. positioning or MH. Vibrations cause less airflow than MH (Figure 14.8), but the oscillations may

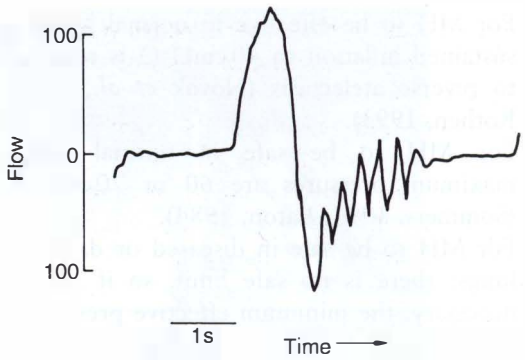


Figure 14.8 Airflow during manual hyperinflation and vibrations. (From MacLean, D., Drummond, G. and Macpherson, C. (1989) Maximum expiratory airflow during chest physiotherapy on ventilated patients before and after the application of an abdominal binder. *Int. Care Med.*, **15**, 396–369)

be helpful, and effectiveness varies with individuals. Neurophysiological facilitation or rib springing are sometimes beneficial. Monitors should be observed closely because some patients cannot meet the extra metabolic demand by increasing their cardiac output.

Suction

> ***Many dreaded both the suction procedure and the instillation of normal saline.***
>
> Jablonski, 1994

Suction is complicated by:

- ↓ lung volume by an average 27% (Brochard *et al.*, 1991)
- ↑ oxygen demand also by an average 27% (White *et al.*, 1990)
- ↓ oxygen supply, transient bronchospasm (Guglielminotti, 1998)
- destabilized haemodynamics and repeated inoculation of the lungs with bacteria from the tracheal tube (Sottile, 1986).

Suctioning secretions rarely reduces respiratory resistance, indicating that secretions contribute little to airways obstruction, but may clear airways sufficiently to reduce intrinsic PEEP (Guglielminotti, 1998). Occasionally, there is a worthwhile reduction in resistance when secre-

tions are blocking the upper airways (Chatila *et al.*, 1995). It is assumed that stasis of secretions encourages infection.

Suction should be carried out when indicated and not routinely (Judson, 1994). Indications, contraindications and technique for nasopharyngeal suction are described in Chapter 8. Modifications for intubated patients are described below.

Preliminaries

For people with cardiovascular instability, administration of a narcotic analgesic before suction can attenuate haemodynamic disturbance (Klein *et al.*, 1988). Patients are advised that they will feel more in control if they let the catheter pass and avoid coughing until it is irresistible. Self-ventilating patients may find it helpful subjectively to hyperventilate beforehand.

For access to the left main bronchus, Judson (1994) recommends turning the head to the left. An angled (coudé-tipped) catheter facilitates this when the tip is directed to the left. This is used more by anaesthetists because physiotherapists usually mobilize secretions sufficiently beforehand.

Technique for open suction

Catheter size should be no more than half the internal diameter of the tracheal tube. A size 12 catheter is normally used, but size 14 is acceptable with large tubes. Aseptic technique should be pristine. The catheter must not touch the rim of the tracheal tube on insertion, and sterile gloves are mandatory. Boxed gloves are not recommended because half have been found to be contaminated (Rossoff, 1995).

Vibrations are unnecessary during suction because, unless the patient is paralysed, enforced coughing overrides outside influences. Occasionally apical vibrations can be used to stimulate a cough. During catheter withdrawal, some patients voluntarily hold their breath longer than necessary, in which case they can be told, when the catheter has been withdrawn back to the tracheal tube, when to breathe again.

If more than one suction pass is necessary, S_aO_2 or $S_{\bar{v}}O_2$ must first return to baseline. The same catheter should not be used for repeated suction because of the bacteria-laden inner surface of the tracheal tube (Sottile, 1986). The patient's mouth may need suction afterwards with a clean catheter or Yankauer sucker. Patients who are able will prefer to do this themselves.

Monitors should be observed, and suction terminated if HR slows by 20 or increases by 40 bpm, if BP drops or arrhythmias develop.

Technique for closed-circuit suction

An in-line catheter (Figure 14.9) avoids disconnection from the ventilator and can cause less

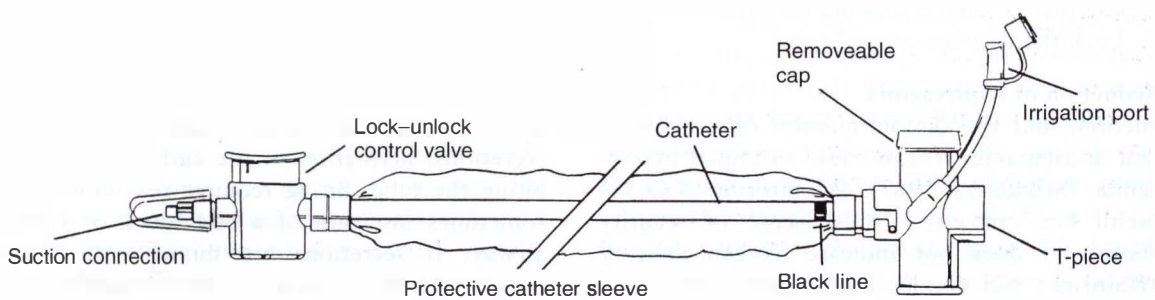


Figure 14.9 Components of a closed circuit catheter. The control valve locks the vacuum on or off. The catheter is protected inside an air-tight sleeve. A T-piece connects the device to the tracheal tube. The irrigation port allows saline instillation for irrigating the patient's airway or for cleaning the catheter.

desaturation (Wainright and Gould, 1996). The catheter is sealed in a protective sleeve and becomes part of the ventilator circuit via a T-piece. Gloves are not necessary and the same catheter is used for 24 hours.

For people who need high F_IO_2 and PEEP, there is less physiological disturbance (Johnson, 1994), and neonates have shown improved S_aO_2 compared to open suction (Castling, 1995). In-line suction should reduce cross-infection but there is no evidence that it influences nosocomial infection for the patient using it. Disadvantages are reduced manual sensitivity, and the obligation to use intermittent suction to relieve suction pressure rather than the rocking thumb technique. Appendix C lists manufacturers who provide videos on techniques. The following is a suggested procedure for the Trach-Care system:

1. Ensure black marker is visible except when catheter is in use.
2. Support T-piece throughout.
3. Unlock and depress vacuum control valve, check suction pressure.
4. Release valve to close off suction.
5. Advance catheter fully, withdraw slightly, depress valve to apply suction, withdraw catheter slowly until black marker is visible, i.e. catheter is out of the patient's airway.
6. Clean catheter by opening the side port, depressing vacuum control valve, injecting 5–10 mL sterile saline into the side port while continuing suction and keeping the black marker in view, releasing valve when completed, then recapping the port.
7. Lock the vacuum control valve.

Reduction of hypoxaemia

Suction, and the discontinuation of ventilation that accompanies it, can cause sustained hypoxaemia (Schwartz, 1987). Monitoring S_aO_2 is useful but can give a false sense of security because it does not indicate oxygen delivery (Wainright and Gould, 1996), and suction can be accompanied by a significant rise in oxygen consumption without a corresponding rise in cardiac output (Walsh *et al.*, 1989). If available, $S_{\bar{v}}O_2$ should be used.

Returning the patient to the ventilator at normal settings between suction passes is not adequate to prevent desaturation (Baun, 1984) and a variety of measures are available to prevent hypoxaemia:

- Manual hyperventilation and hyperinflation help to reverse hypoxaemia and atelectasis respectively. This has been shown to prevent desaturation in the majority of patients (Chulay, 1988) and is described as one of the most effective techniques (Goodnough, 1985).
- The '100% oxygen' button can be used, or the nurse asked to raise the ventilator F_IO_2 2–3 minutes prior to suction then return it to normal 2–3 minutes afterwards, so long as S_aO_2 has stabilized. A few minutes is normally considered adequate for the extra oxygen to be delivered from the ventilator, but washout time varies from several breaths (Ciesla, 1996) to 15 minutes (Sasse, 1995). This is another reason for physiotherapists to become acquainted with their ventilator handbook.
- A maximum of 10 seconds should be allowed for each suction. If longer is needed, this can be accommodated by removing the thumb from the catheter port to release the vacuum, occluding the catheter mount opening (with the catheter still *in situ* but withdrawn sufficiently to prevent coughing), then giving the patient 100% oxygen by MH or the ventilator. Suction is resumed when ready.

Problems

Difficulty passing the catheter may be due to kinking of the tracheal tube, obstruction by thick secretions, herniation of the cuff or the patient biting the tube. Biting requires reassurance and sometimes insertion of a bite block or Guedel airway. If secretions are mixed with blood, liaison with the nurse is required. Possible causes are recent tracheostomy change, trauma from a pulmonary artery catheter, clotting disorder, heparinization or suction that is rough, frequent, too deep or carried out with dry airways.

Saline instillation

Normal saline is sometimes instilled into the lungs with the intention of mobilizing thick secretions. Disadvantages are interference with gas exchange and risk of bronchospasm and infection. Even with a sterile technique, bacteria can be dislodged from a colonized ETT and seeded into the lower airway (Hagler and Traver, 1994). There are also doubts about its efficacy because mucus does not incorporate water easily (Dulfano, 1973). The need for saline suggests that humidification may be inadequate. However, saline may help dislodge encrusted secretions or encourage coughing (Gray *et al.*, 1990), and limited data suggest that it can be beneficial (Judson, 1994). If instillation is used, the following points are suggested:

- Warm the saline first if there is a risk of bronchospasm.
- Administer it slowly to prevent patients feeling as if they are drowning.
- Do not wet the tracheostomy dressing.
- If the aim is to loosen secretions (rather than dislodge debris at the end of the tracheal tube), the patient can be turned after instillation, so that the instilled side is uppermost for treatment.
- A volume of 5 mL has been advised (Bostick and Wendelgass, 1987), but 10 mL or more can be used if it is trickled in slowly and interspersed with manual ventilation or a ventilator manual sigh to prevent desaturation.

If this does not clear secretions, saline can be delivered more distally by injecting it through a suction catheter (p. 443).

With an in-line catheter, hold the T-piece upwards so that gravity assists passage of saline, unlock the vacuum control valve, advance the catheter and inject saline through the side port just before inspiration so it is carried distally with the next breath.

The amount of material recovered has been described as 41% following a 20 mL lavage (Lam, 1985), but this will depend on timing, which is aimed at retrieving the maximum quantity of mucus. If too quick, only the saline will be retrieved. If too slow, the saline will have been absorbed. Optimal time is usually found to be equivalent to the time it takes to instil the saline, turn the patient so that the instilled side is uppermost, then perform a few MH breaths before suctioning.

Afterwards

After suction, patients should not be moved until stable (Riegel, 1985). After treatment, the following steps should be taken as appropriate:

- Check alarms.
- Ensure that the call bell and other requirements are within reach of the patient.
- Reassure the patient that s/he is not being left alone and that their lines are safe so that they do not feel inhibited from moving.
- Tell the patient the time.
- If a rest is required, liaise with the nurse about dimming the light or using eye shades.
- Check any individual concerns, e.g. anxiety about facing a wall.

EXERCISE AND REHABILITATION

> ***'I gained greater comfort when the positions of my legs were varied.... Hearing was acute: every sound seemed magnified.'***
>
> Gandy, 1968

Exercises

Activity is required to maintain sensory input, comfort, joint mobility and healing ability (Frank *et al.*, 1994), and minimize the weakness caused by loss of up to half the patient's muscle mass (Griffiths and Jones, 1999). Patients confined to bed need active or passive exercises, with special attention to the Achilles tendon, hip joint, joints around the shoulder, two-joint muscles and, for long-term patients, the jaw and spine. The longer a patient is bedridden, the more time is needed for exercise, including stretches and mobilizations to thoracic joints (Barker, 1998). A stiff chest wall may respond to manual rotation of the thorax in time with the ventilatory cycle.

Precautions include care with passive movements for paralysed people whose joints are unprotected by muscle tone, ensuring that movement is functional and takes account of trunk position, and avoiding a long lever. Vigorous active exercise should be avoided for patients who are on inotropic support because they have limited cardiovascular reserve, or those on CMV because they are unable to increase their minute volume.

Patients with fractures, burns or altered muscle tone need input from specialist colleagues. Patients with neurological problems, or damage to peripheries due to impaired perfusion, may need splinting to optimize function and circulation.

Mobilization

> ***The physician must always consider complete bed rest as a highly unphysiological and definitely hazardous form of therapy, to be ordered only for specific indications and discontinued as early as possible.***
>
> Dock, 1944

Bed exercises, even when strenuous, are unable to prevent deconditioning (Sandler, 1988). An attempt should be made to stand and walk all patients for whom there is no contraindication such as cardiovascular instability, relevant fracture or paralysis. Attachment to a ventilator does not preclude getting out of bed, but solicitous attention to lines and tubes is required. A rebreathing bag provides ventilatory support if the patient walks more than a few steps from the ventilator. Walking should be brief to prevent fatigue.

The patient needs to spend time sitting with their legs dangling over the edge of the bed before standing (Winslow, 1995). Patient and monitors should be scrutinized when the patient is upright. Pallor, or HR reduction by 5–10, indicates that the patient should sit down again (Winslow, 1995).

For patients unable to stand, sitting in a chair helps prevent hypovolaemia (Wenger, 1982), redistribute skin pressure, change resting muscle length, assist orientation and load vertebrae to limit calcium loss and promote cartilage nutrition. Tipping chairs are useful. A tilt-table may be helpful so long as there is no postural hypotension due to loss of the calf pump.

Long-term patients may be excited at the prospect of their much-awaited first expedition out of bed, and some are then disillusioned by the extent of their weakness and fatigue. This is when they most need encouragement. A visit outside is helpful, 'to maintain my sanity' according to one patient (Clark, 1985). Lack of outside windows has been shown to double the incidence of delirium (MacKellaig, 1990).

ICUs are now considered to be early rehabilitation units. Assistants can help with exercises and mobilization, under supervision. ICU rehabilitation has been shown to accelerate recovery (O'Leary and Coakley, 1996), and this should start with the first treatment, even if modestly.

Transfer from the ICU

> ***'When I was moved from intensive care to the general ward I felt desolate ... as if my life support system had been ripped away.'***
>
> Moore, 1991, p. 12

For patients who have been under constant supervision for some time, transfer to the ward can mean anxiety and dread (Jones and O'Donnell, 1994) as well as relief at reaching a milestone. One study found that a quarter of patients died after leaving the ICU, half of the deaths being unexpected (Goldhill and Sumner, 1998). This is more than would be expected from erroneous discharge decisions, and 'relocation stress' is thought to be the culprit, due to the change in environment, staff and routine, the sudden reduction in technical and human support (Jones and O'Donnell, 1994) and 'inadequate protection against surprise' (Bowes, 1984).

Suggestions to remedy this include early information to the patient, reduction of equipment at the bedside, a visit from the patient's named

ward nurse, an exit interview (Sawdon *et al.*, 1995) and a booklet for patients and families (Jones and O'Donnell, 1994). Follow-up clinics have been found not only useful for patients, families and staff, but also cost-effective (Waldmann and Gaine, 1996). Physiotherapists should involve themselves in these clinics to identify musculoskeletal problems left over from extended immobility.

After discharge from the ICU, patients need ongoing rehabilitation to reduce common problems of imbalance, fear of falling and panic attacks (Griffiths and Jones, 1999).

After discharge from hospital, patients need continuation of physiotherapy at home when necessary because this is often when they realize how debilitated they are, and few know how and when to start exercising and how to pace themselves (Griffiths and Jones, 1999).

> ***'I've tried to help by doing the washing up but I keep dropping the crockery.***
>
> ***When I went home I climbed the stairs on my hands and knees and came down on my bottom.'***
>
> Patients quoted by Griffiths and Jones, 1999

RECOGNITION AND MANAGEMENT OF EMERGENCIES

The key to the successful management of emergencies is informed anticipation and recognition. Physiotherapists are not immersed in life-threatening events every day, so it is advisable to review protocols regularly in order to maintain confidence and avoid the indecision that is often evident at the scene of an emergency.

Some emergencies are covered in the text:

- tracheostomies, page 281
- chest drains, page 270
- fat embolism, page 407
- shock, page 410.

Cardiac arrest

Cardiac arrest is the sudden cessation of heart function. It is the commonest mechanism of the old-fashioned process of death, but is potentially reversible. It is followed within seconds by loss of consciousness and then by loss of respiration.

Anticipation

Before starting work in any new ward or unit, the first task is to locate the crash trolley. Before seeing a new patient, the medical history will provide evidence of risky conditions such as ischaemic heart disease, severe respiratory disease, drug overdose, metabolic disturbance, arrhythmias or shock.

All staff are updated regularly on basic life support, and local training should take precedence over the following guidelines.

Recognition

Warning signs are change in breathing, colour, facial expression or mental function. Hypoventilation with altered consciousness is an ominous combination. Loss of consciousness is the first obvious, though non-specific, sign.

The patient's colour may be pale, ashen or blue, depending on the cause. No carotid pulse can be felt in the groove between the larynx and sternomastoid muscle.

Respiration may become gasping and then stops (unless respiratory arrest has been the primary event).

An ECG may show ventricular fibrillation (VF), ventricular tachycardia, asystole or electromechanical dissociation (EMD).

Action

The time between collapse and initiation of resuscitation is critical, and a false alarm is better than a dead patient. If suspicions are raised by a change in consciousness and colour, feel for the pulse if skilled in this. Call out to the patient, and if s/he is unresponsive, follow the basic life support stage of cardiopulmonary resuscitation (CPR):

1. Summon help by pressing the crash button and by bellowing 'Cardiac arrest!'. If no-one is available and a telephone is nearby, call the crash team or ambulance.

2. Position the patient supine and remove pillows.
3. Establish a patent airway. Use one hand to tilt the head back and the other to lift the chin forward to pull the base of the tongue away from the back of the throat. (If neck injury is suspected, substitute the head tilt by a jaw thrust: with index fingers under the angles of the jaw, pull jaw forwards without moving the neck, open mouth with the thumbs). If necessary, suction mouth and throat with a Yankauer sucker. If an obstruction can be seen, extract it if accessible, but if there is a risk of pushing it further down, use the choking protocol (see below). Leave well-fitting dentures in place to improve the mouth seal.
4. Keeping the airway open by correct head positioning, look, listen and feel for breathing for 10 seconds. If breathing is absent or gasping, ventilate with 100% oxygen using a face mask, resuscitation bag and oxygen at 15 L/min. Pull the mask edges apart before positioning over the face, while maintaining a patent airway. Do not lose the patient airway position achieved. Bag-mask ventilation is easier with two people, but if only one person is available, it is easier to resuscitate with a Laerdal face mask or, if necessary, mouth-to-mouth with a pinched nose. The first two breaths should be slow to minimize risk of aspiration.
5. Kneel on the bed and apply external chest compression, using body weight through straight arms: with the heels of both hands two fingerbreadths above the xiphoid process, depress the lower sternum 4–5 cm at 100 compressions a minute. Apply pressure smoothly and evenly to minimize fracture risk. Effectiveness of chest compression is suspected by return of a healthy colour and confirmed by palpation of a spontaneous pulse when a spare person is available.

For two-people CPR, the last two stages are performed concurrently, with chest compressions and ventilation at a ratio of 5:1. If only one person is available, the compression-to-ventilation ratio is 15:2. For physiotherapists working in the community or out of reach of a crash trolley, it is advisable to carry a pocket mask for mouth-to-mask ventilation.

Do not interrupt CPR to make further checks unless there are signs of life. If the patient regurgitates, turn the head to the side, suction or wipe out the mouth, and continue CPR. If recovery occurs, turn the patient into the semi-prone recovery position (with the patient's flexed arm supporting the head to keep the neck in alignment) so that the tongue falls safely to the side of the mouth and unwanted material can drain out.

Common errors are:

- not maintaining a patent airway throughout, e.g. inadequate neck extension
- not allowing chest deflation between breaths
- not compressing the chest with sufficient force
- compressing one side of the sternum rather than mid-sternum.

When the crash team arrives, they will instigate advanced life support, i.e. ECG monitoring, intubation, medication, defibrillation. Staff should stand clear while defibrillation is applied.

When no longer needed, the physiotherapist can give attention to other patients who may be distressed at witnessing the event. If the patient survives, s/he will need debriefing by an appropriate member of staff. If the patient dies, the physiotherapist and those involved will need some support.

Respiratory arrest

As cardiac arrest leads to respiratory arrest, so does respiratory arrest, if untreated, lead to cardiac arrest.

Anticipation

Predisposing factors include exacerbation of COPD, airway obstruction (e.g. foreign body, swelling or bleeding from trauma, smoke inhalation) or aspiration (especially following drug

overdose). Warning signs are inability to speak, and either violent respiratory efforts, laboured breathing or drowsiness.

Recognition
Respiratory arrest is indicated by absence of movement of the chest, loss of airflow from the mouth and nose, and sometimes cyanosis. This progresses to loss of consciousness.

Action
1. Call for help.
2. Establish a patent airway as described for CPR. If there is no airflow, continue as below.
3. If a foreign body is the likely culprit, e.g. the victim is choking, attempt to dislodge it from the throat by suction or, if it can be seen, by hand. If unsuccessful, administer up to five piston-like Heimlich manoeuvres: strong inward and upward thrusts to the abdomen applied below the rib cage and above the navel, providing the patient is not pregnant or very fat. This can be done from behind a standing victim or kneeling astride a supine victim. If the patient is still not breathing, continue as below.
4. Ventilate by bag-mask ventilation, Laerdal face mask or mouth-to-mouth, as described above. Inspiration time is 1.5–2 seconds. Repeat once every 6 seconds. Continue for 1 minute, then re-assess.

If cardiac arrest ensues, instigate full CPR. If the patient starts breathing, turn him/her into the recovery position because vomiting is common as consciousness lightens.

Seizure

Anticipation
The medical notes indicate whether a patient has a history of epilepsy. Other causes of fitting are head injury, alcohol intoxication, or in children, fever. Some patients sense an aura in advance.

Recognition
Seizures vary from transient loss of consciousness to major muscle activity, followed by drowsiness.

Action
1. Patients subject to frequent seizures should have the bed kept low, side rails up and padded, and oxygen and suction available.
2. If there is advance warning, insert an airway. Do not attempt this once the seizure is under way.
3. Protect the patient's head and body from injury. Loosen tight clothing, especially around the neck. Do not use restraints or hold the victim down. Keep in side-lying if possible.
4. Afterwards, ensure the patient is in the recovery position. Reassure him/her as consciousness returns. Request medical assessment.

Haemorrhage

Anticipation
Uncontrolled bleeding can follow surgery, arterial line disconnection or trauma.

Recognition
External bleeding is not easily missed. Internal bleeding is suspected if there are signs of severe hypovolaemia (p. 362). BP and HR are the least reliable of these signs because BP can be maintained until 40% of blood volume is lost, and HR is responsive to other variables. Bleeding into a closed space causes extreme pain.

Action
1. Position the patient supine.
2. Apply pressure to the bleeding point if accessible.
3. Elevate the affected part if possible.
4. Request assistance.
5. Explain to the patient what is being done.

Massive haemoptysis

Massive haemoptysis can be defined in relation to the volume expectorated or the magnitude of the effect, e.g. 100–1000 mL blood over 24

hours or sufficient to be life-threatening by virtue of blood loss, hypotension, or, most commonly, asphyxiation (Dweik and Stoller, 1999).

Anticipation
Predisposing factors are lung cancer, bronchiectasis, abscess or TB.

Action
The patient should be positioned head down, and if the side of the haemorrhage is known, laid on the affected side to prevent aspiration into the healthy lung. Cough suppressants and sedatives should not be given. Patients with depressed consciousness or risk of asphyxiation need intubation and suction. Bronchial artery embolization may be required (Mal *et al.*, 1999).

Cardiac tamponade

Cardiac tamponade is accumulation of gas or fluid, usually blood, in the pericardium. The pericardium is not distensible and can only accommodate 100 mL fluid rapidly without affecting cardiac output, after which an additional 40 mL doubles pericardial pressure, compressing the heart and damming back blood in systemic veins (Hyde *et al.*, 1996). If increasing pressure is not relieved, cardiac arrest is inevitable.

Anticipation
Tamponade can occur in the first 24 hours after heart surgery. Other predisposing factors are trauma, dissecting aneurysm, infection or malignancy.

Recognition
Progressive compression of the heart leads to precipitate loss of cardiac output and rise in filling pressures. Hypovolaemia masks some of these signs, but the following may be evident.

- ↓ BP, $S_{\bar{v}}O_2$, urine output
- ↑ CVP and JVP
- ↑ PAWP, HR
- CVP and PAWP approximately equal
- sudden change in pericardial drain output
- distended neck veins
- pulsus paradoxus (see Glossary)
- narrowed pulse pressure
- enlarged heart on X-ray.

Action
Alert the doctor, who will aspirate fluid direct from the pericardium.

Tension pneumothorax

Gas entering the pleural space on inspiration but unable to escape on expiration causes tension pneumothorax. Cardiac arrest will follow within about 20 minutes.

Anticipation
In ventilated patients, pneumothoraces are likely to be under tension especially at the following times:

- immediately after intubation, if inadvertent tube placement into the right main bronchus causes hyperinflation of the right lung
- in the hours following initiation of mechanical ventilation, when air is forced through a previously unknown leak in the pleura.

Predisposing factors are emphysema, and surgery or other trauma to the chest. Surgical emphysema in the neck can be a warning sign.

Recognition
Tension pneumothorax is sufficiently rare to be sometimes mistaken for bronchospasm. Both of these conditions cause respiratory distress, wheeze, increased airway pressure and laboured breathing. The added features of tension pneumothorax are:

- ↓ amplitude in ECG (often the first sign)
- unequal chest movement
- hyperresonant percussion note on the affected side
- ↓ breath sounds on the affected side, or both sides if severe
- ↓ S_aO_2
- cyanosis

- distended neck veins and ↑ CVP (unless patient is hypovolaemic)
- displaced apex beat
- in self-ventilating patients, dyspnoea and tracheal deviation away from the affected side
- ventilator evidence of high airway pressure, and expired minute volume less than preset minute volume
- ↓ BP, ↑ HR, progressing to cardiovascular collapse
- radiograph as in Figure 14.10 (this is an X-ray we should never see because there is no time to waste).

Action

Alert the doctor, who will insert a 14G cannula into the pleura at the second intercostal space in the midclavicular line to release the pressure. While waiting, an experienced physiotherapist can disconnect the patient from the ventilator and manually ventilate with 100% oxygen, using high flow and low pressure. Others should maximize F_IO_2 through the ventilator.

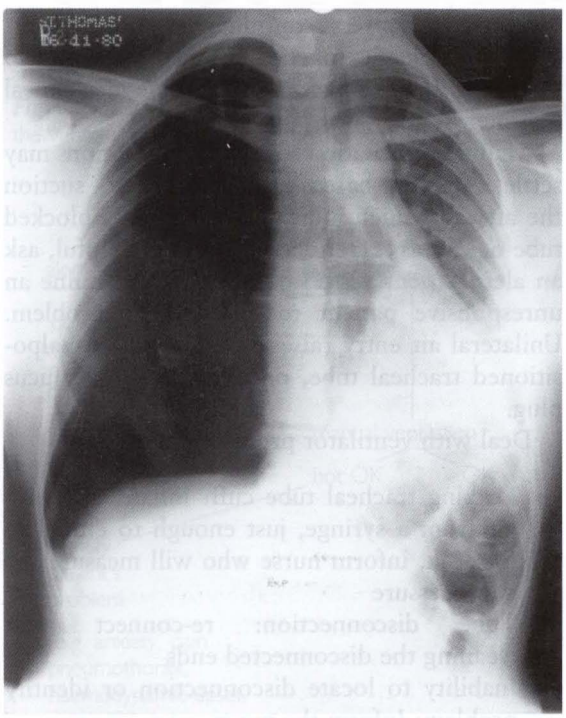


Figure 14.10 Right tension pneumothorax, as indicated by a black area devoid of lung markings on the right and mediastinal shift to the left away from the affected side.

Pulmonary embolism

Sudden patient distress and the signs described on page 113 raise suspicions of pulmonary embolism. Disconnection from the ventilator and manually ventilating the patient do not help. Monitors will show ↑ HR, ↑ BP ↑ RR, ↓ S_aO_2, ↑↑ CVP, ↓ cardiac output. See page 113 for management.

Air embolism

Anticipation

Air may enter the circulation after cardiac or neurosurgery, or occasionally from a pneumothorax or during insertion or removal of a pulmonary artery catheter or vascath.

Recognition

A large air embolus causes respiratory distress, palpitations, dizziness, weakness and pallor or cyanosis.

Action

Summon help. Place the patient head down in left-side-lying, which diverts air away from the pulmonary artery and pulmonary circulation. Give high-percentage oxygen. An embolus larger than 100 mL may cause cardiac arrest, which requires cardiac compression with heavy and deep pressure to disperse air bubbles to peripheral segments of the pulmonary artery.

Equipment malfunction or disconnection

Astute eyes and ears help pick up the slight hiss of an air leak, identify from an orchestra of alarms which is the offending malfunction, or notice the subtle change in a drowsy patient's demeanour that signifies that something is amiss. Prevention includes reading the manufacturer's handbook in order to understand the workings of the ventilator, and familiarity with the alarms to help distinguish what each signifies.

Alarms

The most relevant alarms for the physiotherapist are the high-pressure alarm, low-pressure alarm, and alarms for BP, F_IO_2 and humidifier heater. The high-pressure alarm is set at 5–10 cmH_2O higher than peak airway pressure and it is activated if there is:

- major atelectasis
- sputum retention
- condensed water in the tubing
- patient coughing or fighting the ventilator
- bronchospasm
- pneumothorax
- partial extubation
- right main bronchus intubation
- cuff herniation over the end of the tracheal tube
- patient biting the endotracheal tube.

If the patient bites the ETT, this requires dissuasion, sedation or change to a nasal tube. For a displaced ETT, the doctor will deflate the cuff, reposition the tube, inflate the cuff, listen for equal breath sounds and request a check X-ray.

The low-pressure alarm indicates that pressure has fallen more than 5–10 cmH_2O below the desired limit and means a leak in the system, confirmed by reduced expired minute volume and airway pressure. A disconnected circuit should be reconnected after a quick alcohol wipe if it has touched anything. The patient's condition should be checked, the cause determined and appropriate adjustments made or the nurse informed.

Alarms are fallible. Patient observation comes first.

Arterial line or vascular catheter disconnection

Firm pressure to the site is required if an arterial line or vascath becomes disconnected from the patient. Reassure the patient, who may be frightened at the amount of blood. Observe patient and monitors for signs of hypovolaemia. Inform the nurse.

Patient distress on IPPV

Patient-related problems (Figure 14.11) include:

- pain
- fear
- pneumothorax, pulmonary oedema, abdominal distension, bronchospasm or mucus plug
- biting the tube.

Ventilator-related problems include:

- kink or leak in the circuit
- intrinsic PEEP
- inappropriate settings for flow rate, tidal volume, I:E ratio or trigger sensitivity.

While awaiting assistance from other members of the team, check airway pressure and monitors. Ask the patient if s/he wants more air. If the answer is a nod, or the patient is unable to respond, disconnect the patient from the ventilator and connect to the bag with oxygen. Either manually ventilate or allow the patient to self-ventilate through the bag with the valve open for minimal resistance and a high flow rate for comfort. If distress continues, it is a patient-based problem, to be sorted with yes/no questions. If it resolves, there is some mechanical mischief.

Manual ventilation or hyperventilation may settle a patient-based problem. If not, suction the airway, which indicates if there is a blocked tube or excess secretions. If this is unhelpful, ask an alert patient yes/no questions, or examine an unresponsive patient to identify the problem. Unilateral air entry raises suspicions of a malpositioned tracheal tube, pneumothorax or mucus plug.

Deal with ventilator problems as follows:

- Leaking tracheal tube cuff: inflate cuff with air from a syringe, just enough to eliminate the leak, inform nurse who will measure the cuff pressure
- Tube disconnection: re-connect after cleaning the disconnected ends
- Inability to locate disconnection or identify problem: Inform the nurse
- Tracheal tube malfunction, bronchospasm, ventilator asynchrony unresolved by talking to patient: inform the doctor.

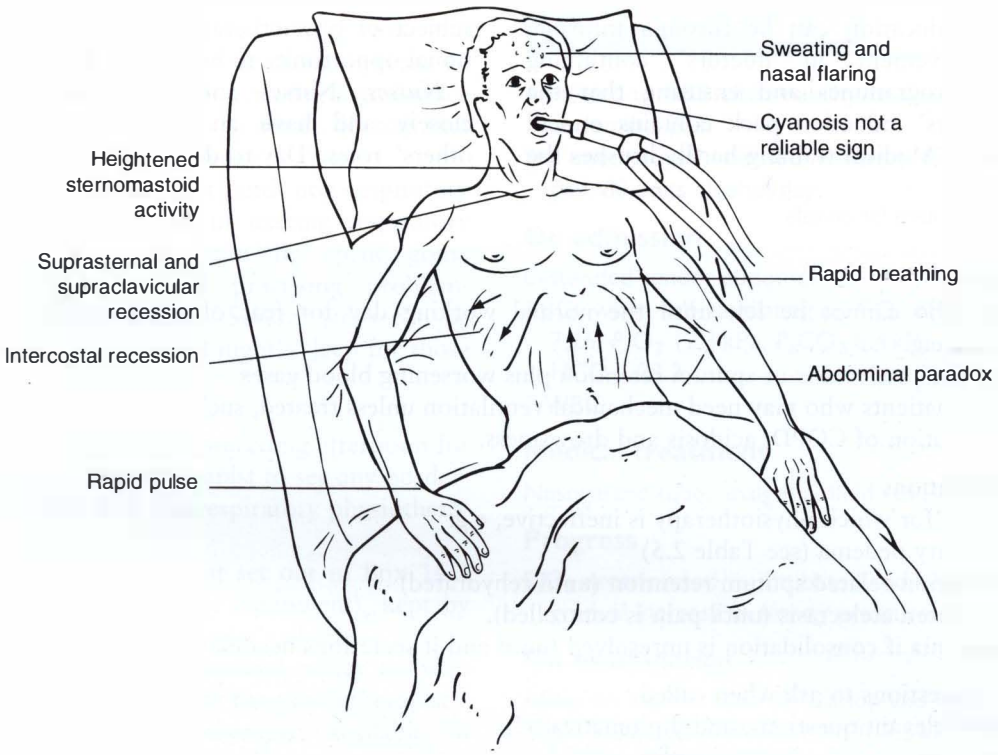


Figure 14.11 Physical signs of patient distress. (From Tobin, M. J. (1991) What should the clinician do when a patient fights the ventilator? *Resp. Care*, **36**, 395–406)

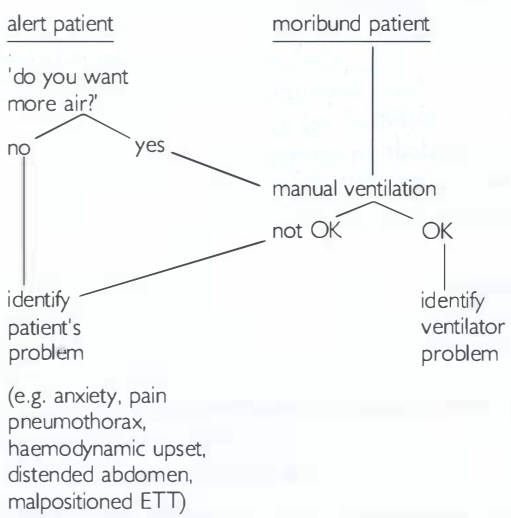


Figure 14.12 Flow chart for relieving patient distress

Figure 14.12 provides a quick-check guide, after discussion with other members of the team.

ON CALLS

A well managed on-call system can sustain many a sick patient through a difficult night. The key to success is education so that all parties understand the scope and limitations of physiotherapy. Education should be targeted at relevant staff, as follows.

Doctors. All levels of medical staff need advice on the indications for out-of-hours physiotherapy, with particular attention to juniors starting a new rotation. Young doctors in a new environment can become anxious with an unfamiliar event and may call out the physiotherapist unnecessarily or not call when

indicated. Education can be through informal talks, involvement in doctors' continuing education programmes and ensuring that the house officers' induction pack contains on-call information. Medical training hardly brushes the subject of physiotherapy and this is an educational opportunity to be grasped gladly.

Nurses. Nurses and physiotherapists work closely and have an understanding of each others' roles. Day-to-day exchange of informa-

Box 14.3 Criteria for on-calls

Indications

Patients who cannot be left until the normal working day for fear of deterioration in their condition, e.g.

- those with atelectasis or sputum retention plus worsening blood gases
- certain patients who may need mechanical ventilation unless treated, such as those with exacerbation of COPD, acidosis and drowsiness.

Non-indications

Conditions for which physiotherapy is ineffective, e.g.

- pulmonary oedema (see Table 2.5)
- dehydration-related sputum retention (until rehydrated)
- pain-related atelectasis (until pain is controlled).
- pneumonia if consolidation is unresolved (until and if secretions become a problem).

General questions to ask when called

(ask only relevant questions, and diplomatically!)

- doctor's name and contact details
- patient's name, diagnosis and location
- auscultation
- hydration status
- trend in temperature
- trend in blood gases
- X-ray results
- cardiovascular status
- pain, and outcome of analgesia
- patient's problem, e.g. atelectasis, exhaustion, sputum retention
- if productive of sputum, is the patient able to clear it unaided?
- if non-productive of sputum, is there sputum retention, or no sputum?
- availability of X-ray, medical notes and necessary equipment.

If physiotherapy is not indicated:

- politely explain indications for emergency physiotherapy
- say that if the situation changes, please call again.

If physiotherapy is indicated:

- before coming in, ask for analgesics, anti-emetics or humidification if necessary
- ask for patient to be positioned appropriately.

After treatment

Liaise with nursing staff re: ongoing management, e.g. positioning, rest, reminders on incentive spirometry.

tion lays the foundation for co-operation, and this can be developed into teaching sessions so that some nursing staff can perform maintenance chest care and know when to advise doctors that the physiotherapist be called.

Physiotherapists. Junior and non-respiratory seniors need confidence in making respiratory decisions. Useful time can be spent going through equipment and practising problem-solving with case studies. Several steps can be taken to facilitate a sound night's sleep for those on duty:

- time set aside on the preceding afternoon for the on-call physiotherapist to see any borderline patient with the respiratory physiotherapist
- a handout such as that set out in Box 14.3 (to include location of equipment), kept by the on-call physiotherapist's phone
- clarification of departmental policy on who is authorized to call out the physiotherapist
- a respiratory physiotherapist available to inexperienced on-call staff at the end of a bleep for advice.

If called to Accident and Emergency, it is advisable to check that the patient is not immersed in tests and investigations, and to identify when s/he will be available for physiotherapy.

The interests of the patient and good relations with other disciplines can be fostered by the physiotherapist taking responsibility for pre-arranging call-outs when appropriate. The physiotherapist can also act as advisor and consultant over the phone.

Many departments organize evening physiotherapy shifts because there is evidence that this can stem deterioration in patients after major surgery (Ntoumenopoulos and Greenwood, 1996) or patients with excessive secretions (Wong, 2000).

Guidelines for different on-call problems may be helpful, e.g. Box 14.4.

CASE STUDY: MR AP

Admitted to A&E after inability to sleep due to abdominal pain

SH: unemployed, married, children, 15 cigarettes/day, 80 units alcohol/day.

On admission

Distended tender abdomen.

ABGs when self-ventilating on F_IO_2 of 0.6: pH 7.26, P_aO_2 12.3 kPa, P_aCO_2 8.4 kPa, BE 1.5, HCO_3^- 23.2.

↑ WBC.

Medical treatment

Nasogastric tube, analgesia, fluid resuscitation.

Progress

S_aO_2 deteriorated → intubated and ventilated → gradual increase in airway pressure.

On examination

ABGs on inverse ratio ventilation and F_IO_2 of 0.85: pH 7.20, P_aO_2 7.9 kPa, P_aCO_2 7.5 kPa, BE 1.0, HCO_3^- 25.2.

Breath sounds absent bibasally.

Diagnosis

Acute pancreatitis.

Questions

1. What is causing the high airway pressure?
2. What is obliterating the breath sounds?
3. Is the acidosis respiratory or metabolic?
4. What syndrome may develop?
5. Problem?
6. Goals?
7. Plan?

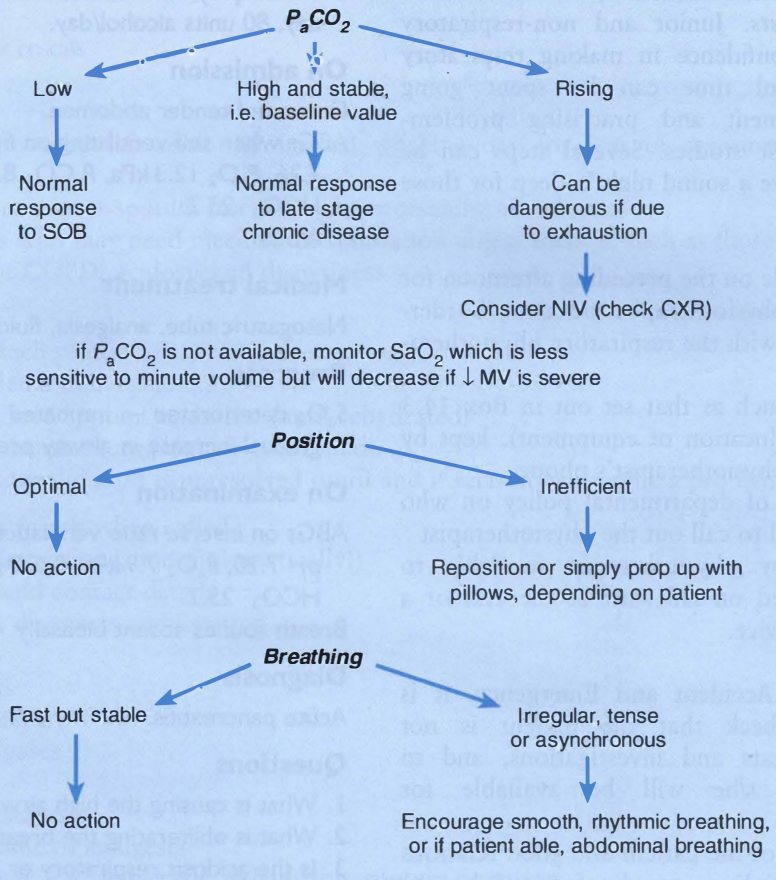
ABGs = arterial blood gases; BE = base excess; WBC = white blood cell count.

RESPONSE TO CASE STUDY

1. High airway pressure is caused by pressure against diaphragm from distended abdomen.
2. Breath sounds reduced by compression of lungs from distended abdomen.

Box 14.4 Management of the acutely breathless patient

Management of objective signs



- *Bronchospasm*
  Check drug chart and PF chart.
  Liaise with team.
- *Secretions*
  Hydration.
  Humidification (warm if bronchospasm).
  Slow rhythmic percussion.
  AD or modified ACB.
  Cough suppression until secretions accessible, then cough facilitation.

Continued opposite

Management of symptoms

- *Fatigue*
  Positioning and some SOB strategies (p. 169–175)
- *Feeling out of control*
  Identify patient's coping strategies, suggest any others.
- *Lack of sleep*
  Liaise with team re. environment, check anxiety.
- *Anxiety*
  Identify cause, provide information.
- *Pain*
  Identify cause. If due to coughing, educate on selective cough facilitation and suppression as and when appropriate. If due to muscle tension, relieve by positioning, relaxation. Suggest or show massage to relatives.
- *Exhaustion*
  Monitor P_aCO_2 and pH.

Tips for handling the acutely breathless patient

- Avoid noise, bright lights, crowding
- Do not enter patient's personal space until after introductions
- Avoid chatter, be specific, talk gently
- Offer questions with yes/no answers
- Identify patient's view of cause of breathlessness
- Patient or relatives may have information on individual relieving strategies
- Patient may find curtains claustrophobic, may need window open.

ACB = active cycle of breathing; AD = autogenic drainage; CXR = chest X-ray; MV = minute volume; NIV = non-invasive ventilation; PF = peak flow; RR = respiratory rate; SOB = shortness of breath.

3. Respiratory acidosis due to CO_2 retention.
4. Multisystem failure.
5. Progression of compression atelectasis, with deteriorating gas exchange.
6. Reverse and prevent further atelectasis, rehabilitate.
7. Plan:

- Avoidance of supine.
- Initiate kinetic rotation bed.
- If there is localized atelectasis: try MH in side-lying, re-assess for benefits or adverse effects.
- If there are audible secretions: suction.
- Identify if S_aO_2 responds to above techniques, or whether deteriorating gas exchange is due to developing ARDS. (NB distended abdomen may contraindicate prone).
- Care of musculoskeletal system.
- Progress.

LITERATURE APPRAISAL

(Title: Endotracheal suctioning: ventilator vs manual delivery of hyperoxygenation breaths)

> ***The researcher delivered a [manual] tidal volume equal to the patient's ventilator tidal volume ....***
>
> ***...these findings support the use of the patient's ventilator for hyperoxygenation during suctioning.***
>
> *Am. J. Crit. Care* 1996; 5: 192–197

RESPONSE TO LITERATURE APPRAISAL

If the tidal volume delivered by manual ventilation is no more than the ventilator tidal volume, there is nothing to compare.

RECOMMENDED READING

AARC Clinical Practice Guidelines (1993) Endotracheal suctioning of mechanically ventilated adults and children with artificial airways. *Respir. Care*, 38, 500–504.

Anzueto, A. (1999) Muscle dysfunction in the ICU. *Clin.Chest Med.*, **20**, 435–452.

Bergbom-Engberg, I. (1989) Assessment of patients' experiences of discomfort during respirator therapy. *Crit. Care Med.*, **17**, 1068–1072.

Cassa, S., Costar, S. and Tracey, C. (1998) Patient handling aids: prescribing guidelines. *Br. J. Ther. Rehabil.*, **5**, 7–9.

Copnell, B. and Fergusson, D. (1995) Endotracheal suctioning. *Am.J.Crit.Care*, 4, 100–105.

Epstein CD, Henning RJ (1993) Oxygen transport variables in the identification and treatment of tissue hypoxia. *Heart Lung*, 22, 328–348.

Gamrin, L. (2000) Protein-sparing effect in skeletal muscle of growth hormone treatment in critically ill patients. *Ann.Surg.*, 231, 577–586.

Glass, C. A. and Grap, N. J. (1995) Ten tips for safer suctioning. *Am. J. Nurs.*, 95(5), 51–53.

Guglielminotti, J. (2000) Bedside detection of retained tracheobronchial secretilns in patients receiving mechanical ventilation. *Chest*, **118**, 1095–1099.

Juniper, M. C. and Garrard, C. S. (1997) The chest X-ray in intensive care. *Care Crit. Ill*, **13**, 198–200.

Lapinsky, S.E. (1997) Safety and efficacy of a sustained inflation maneuver for alveolar recruitment. *Chest*, **112**, 126S.

McEleney, M. (1998) Endotracheal suction. *Prof. Nurse*, **13**, 373–376.

Marcy, T. W. (1994) Respiratory distress in the ventilated patient. *Clin. Chest Med.*, **15**(1), 55–74.

Raymond, S.J. (1995) Normal saline instillation before suctioning. *Am.J.Crit.Care*, 4, 267–271.

Selsby, D. and Jones, J. G. (1990) Some physiological and clinical aspects of chest physiotherapy. *Br. J. Anaesth.*, **64**, 621–631.

Voggenreiter, G. (1999) Intermittent prone positioning in the treatment of severe and moderate posttraumatic lung injury. *Crit.Care.Med*, 27, 2375–2382.

Whittaker, J. and Ball, C. (2000) Discharge from intensive care. *Int.Crit.Care Nurs.*, **16**, 135–143.

15 Disorders in intensive care patients

Summary

Lung disease

Chronic Obstructive Pulmonary Disease

Mechanical ventilation is not indicated for people with COPD who are suffering an irreversible deterioration in their condition, but it may be needed to buy time during an exacerbation. It is useful to know in advance whether patients would prefer to be ventilated in the event of serious exacerbation.

Intermittent positive-pressure ventilation (IPPV) is usually by pressure support, with extrinsic (applied) PEEP to counterbalance intrinsic PEEP (Rossi, 1994). Patients with chronic hypercapnia must have their minute ventilation titrated to pH rather than P_aCO_2 so that compensatory renal bicarbonate retention will be adequate for buffering during weaning. A person who has acclimatized to complex acid–base compensations may find that the sudden change to IPPV has a destabilizing effect, leading to arrhythmias, hypotension and the unmasking of hypovolaemia. If physiotherapy is needed within 30 minutes of starting IPPV, close monitoring is necessary. Manual hyperinflation (MH) is inadvisable unless essential because of overdistended alveoli and the uneven distribution of extra positive pressure within the damaged lungs.

Weaning can be tiring, protracted and frightening for patients. Physiotherapy is crucial in preparing for this in advance. Rest and sleep are required for 48 hours after initiation of IPPV (Corris, 1990) interspersed with nutritional support and exercises. Rest is also required before the first weaning attempt. Otherwise time should be organized around short periods of exercise alternating with rest. Bed exercises should be demonstrated to the patient, nurse and family, written down and left with the nursing notes. Daily standing and walking are required unless contraindicated. Early rehabilitation has shown:

- ↑ lung function
- ↓ breathlessness
- ↑ exercise tolerance (Nava, 1998).

If problems after extubation are encountered, non-invasive ventilation has been found to reduce the need for reintubation by two-thirds (Hilbert *et al.*, 1998).

Asthma

Mechanical ventilation is a perilous venture for people with acute severe asthma, carrying an average mortality of 20% (McFadden and Warren, 1997). IPPV is indicated if patients become exhausted from the effort of maintaining hyperinflation above TLC in order to keep obstructed airways open, leading to an intractably rising P_aCO_2 with acidosis and impaired consciousness.

Positive pressure volumes above TLC risk intrinsic PEEP, barotrauma, reduced venous return, hypotension, arrhythmias and right heart failure due to compressed pulmonary capillaries. Dehydrated patients are particularly vulnerable.

High levels of oxygen are required. Permissive hypercapnia (p. 352) may be used in an attempt to maintain airway pressures below 40 mmHg. Hyperinflation and intrinsic PEEP can be controlled by:

- extrinsic PEEP
- high inspiratory flow rate (e.g.100 L/min) to prolong expiratory time
- brief disconnection from the ventilator and allowing the trapped gas to escape through the airway
- bilateral expiratory manual compressions of the chest during several successive expirations while disconnected from IPPV, using two people in synchrony, or, for one person, over ribs 8–10 (van der Touw *et al.*, 1998).

Intractable bronchospasm may require heliox (Manthous, 1997), inhalation agents (Bellomo, 1994), hypothermia (Browning, 1992), a continuous IV bronchodilating anaesthetic such as ketamine (Levy *et al.*, 1998) or extracorporeal support. Prolonged paralysis should be avoided unless essential because of its association with myopathy when combined with steroids (Behbehani, 1999).

Physiotherapy is inadvisable immediately after initiation of IPPV because the combination of anaesthesia, hypovolaemia and high airway pressures may cause profound hypotension. β_2-agonists may reduce potassium and further destabilize the cardiovascular system. Rapid infusion of fluids, sometimes with vasopressors, usually restores BP, but physiotherapy is still best limited to stress reduction. If thick mucus plugs are present, instillation of warmed sterile saline, e.g. 2 mL every 15 minutes, may be indicated (Branthwaite, 1985). When airway pressures have settled to normal, other treatment is given as necessary, but MH is unwise while the chest is hyperinflated.

Any sudden deterioration should raise suspicions of tension pneumothorax. The usual signs (p. 384) can be obliterated in a hyperinflated patient on IPPV.

NEUROMUSCULAR DISORDERS

Severe respiratory muscle weakness may lead to hypercapnic ventilatory failure. The physiotherapist's role is to maintain chest, muscles, joints and morale, while being alert to inadequate sedation and analgesia which may occur because of difficulty in assessment and a need for regular neurological examination.

Guillain–Barré syndrome

> ***'The physiotherapist was a most welcome person, as, despite the discomfort endured to have 'dead' limbs stretched and repositioned, this left me comfortable for several more hours.'***
>
> Clark, 1985

Guillain–Barré syndrome is an autoimmune demyelinating peripheral neuropathy. It causes a predominantly motor deficit with some autonomic and sensory components. The syndrome develops after an infection in 75% of patients (Winer, 1994). Presenting features vary widely and include backache, paraesthesia and weakness. Weakness progresses for up to a month, and ventilatory failure develops in 20% of patients, sometimes with alarming speed. This

can be predicted by difficulties with speech or swallowing, but vital capacity (VC) measurements are the most reliable warning sign. Intubation should be considered if VC decreases to 18 mL/kg (Meythaler, 1997).

Medical treatment is mainly supportive, but plasma exchange hastens recovery by removing antibodies from the blood (Appleyard and Sherry, 1998).

Physiotherapy is mainly prophylactic, especially to avoid contractures that can develop and become major components of disability (Soryal *et al.*, 1992). Muscle pain occurs in up to 72% of patients (Pentland, 1994). This is exacerbated at initiation of movement but eased after a few moments of mobility exercises. Exercise should therefore be:

- regular and frequent
- preceded if necessary by anti-inflammatory drugs or Entonox
- gentle at the start
- precise, to ensure full range without risking the damage that can occur with hypotonia and sensory impairment (Soryal *et al.*, 1992)
- when active, brief to avoid fatigue.

Spinal movements should be included, e.g. double knee-and-hip flexion, knee rolling, and neck movements with due care of the tracheal tube. Relatives can assist with some routine exercises. Extremities may be hypersensitive, and a cradle eases the weight of bedclothes.

Autonomic involvement leads to unstable BP and heart rate (HR), and sustained hypertension may alternate with sudden hypotension. The risk of hypotension is reduced by ensuring that turning is gentle, avoiding any intervention if CVP is below 5 cmH_2O, and acclimatization to the upright posture with a tilt table. Risk of bradycardia is reduced by oxygenation before and after suction.

Progress can be hindered by anaemia or prolonged depression with mental fatigue (Meythaler, 1997). Physiotherapy can incorporate trips outside the ICU and, later, hydrotherapy. Recovery takes weeks or months. Some 10% of patients die and 20% are left disabled (Meythaler, 1997). Self-help groups provide support from the ICU stage onwards (Appendix C).

Acute quadriplegia

> ***'You can't appreciate what it is to be paralysed unless you are. The big things you get used to easier, like not getting up and walking around. The trivial things – like not being able to scratch your nose or feed yourself – they hurt.'***
>
> Patient quoted by Stewart and Rossier, 1978

People whose lives have been devastated by disease or trauma to the cervical spine are overwhelmed at first and find it difficult to comprehend how savagely their life has been limited. Physiotherapists who care for people with acute quadriplegia need to allow them to work through their grief at their own pace, while endeavouring to prevent the respiratory complications that are the leading cause of death.

Quadriplegia is paralysis of the limbs and trunk. Tetraplegia is paralysis of the limbs.

Pathophysiology and clinical features

Spinal damage following trauma is greater if there is mishandling at the scene of the accident, e.g. helmet removal. Normal neurological function and normal radiology does not exclude cervical spine injury, and up to 25% of the damage is thought to occur after the initial trauma due to inadequate protection of the spine (Walker, 1998).

Lesions above T6 to L1 paralyse the abdominal muscles and impair coughing. Higher thoracic lesions paralyse the intercostals and destabilize the rib cage, causing paradoxical inward motion on inspiration. Lesions above C4 denervate the diaphragm, leaving only the sternomastoid and trapezius muscles to shift a trace of air into the lungs. Ascending oedema of the traumatized spinal cord may further compromise respiratory function shortly after admission.

Paralysed abdominal muscles reduce venous return and risk an exaggerated response to hypovolaemia. A complete cervical injury is equiva-

lent to a total sympathectomy, reducing tone in blood vessels, denervating the cardiac sympathetic nerve supply and leaving parasympathetic tone (mainly the vagus) unopposed. Hypotension and bradycardia can result, especially during suction of the mouth or airways, and during exertion. A lesion above T6 removes sympathetic control to the splanchnic bed, which is a major reservoir for controlling BP. Cardiac monitoring is required for the first 2 weeks, and oximetry is advisable to detect nocturnal desaturation. Poor circulation creates a high DVT risk (Alderson, 1999), especially with multiple trauma. Diminished venous return and interrupted sympathetic outflow blunt the heart's response to exercise, which can limit exercise capacity (Haas and Axen, 1991, p. 255).

Physiotherapy

The third to fifth crucial days after injury are when lung complications are commonest. Assessment must be meticulous because hypoxia or hypotension may cause secondary damage to the spinal cord. McMichan *et al.* (1980) have shown how preventive measures can reduce the need for IPPV by two-thirds, using regular position change, hourly incentive spirometry, percussion and assisted coughing.

The head-down position is best avoided, but if it is essential for postural drainage, care is needed to ensure that tipping is done slowly and not fully, that cervical traction is maintained, that observation is maintained in case of sudden sputum mobilization, and that arterial and venous pressures are monitored because of the loss of compensatory cardiovascular reflexes.

Coughing is assisted by manual pressure using a hand on each side of the lower ribs and one forearm exerting strong pressure upwards and inwards against the abdomen, in synchrony with any expiratory force that the patient can muster. Some patients require two physiotherapists for this. Care should be taken to avoid disturbing neck traction, jarring the fracture site, exacerbating associated injuries, or pushing towards the spine instead of the diaphragm. Cough assistance should not be attempted if there is a paralytic ileus, which is common for several days after injury. Quadriplegia at C5 or below allows patients to learn self-assisted coughing when able, leaning forward in their chair and using any strength in their arms against the abdomen (Bach and Haas, 1996, p. 407).

Early minitracheostomy is advisable if there is a hint of sputum retention, especially as the neck cannot be extended for effective nasopharyngeal suction. If suction is required, whether for an intubated or spontaneously breathing patient, it should be accompanied by measures to minimize hypoxia, monitoring of S_aO_2 and BP, and availability of IV atropine or other drug in case of profound bradycardia.

Treatment of the limbs involves close attention to positioning and range of movement (Bromley, 1998). A third to a half of patients develop shoulder pain, which often leads to depression (Goldstein, 2000), and hinders wheelchair rehabilitation (Curtis *et al.*, 1999). The key is prevention by early and frequent full-range movement, scapular stretches and education to all team members on the need for care in positioning, especially if there is cervical traction or a rotating bed. In side-lying, direct pressure on the shoulder should be prevented. In supine, the 'crucifixion' position has been advocated, using padded arm boards on each side, but not to the extent of discouraging active arm movement (Crow *et al.*, 2000). This may be useful for other patients with paralysed shoulders, e.g. those with Guillain–Barré syndrome.

Patients with complete lesions are mobilized as soon as possible. Those with incomplete lesions are usually maintained on bedrest for about 6 weeks to ensure optimum perfusion to the spinal cord. Mobilization takes the form of elevation gradually with a tilt table, monitoring BP with every 10° increase in height. Standing is less comfortable than supine because the floppy abdominal muscles allow bulging of the abdomen and loss of vital capacity by an average 14% (Chen *et al.*, 1990). The application of an abdominal binder is helpful for standing or sitting (Goldman, 1986). Physiotherapy in the

acute stage should be little and often to prevent fatigue, and active exercise may require ventilator adjustments to maintain stable blood gases. Environmental temperature needs controlling because impaired sympathetic outflow hinders thermoregulation. If a halo vest is used to stabilize the fracture, all those involved must know how to open or adjust it in case of cardiac arrest.

Muscle tone and strength have a complicated relationship in spinal injuries. After the 'spinal shock' period, which varies from a few days to several weeks, the spinal cord below the lesion begins to transmit reflexes. Lung function may improve as flaccidity changes to spasticity and stiffening thoracic joints provide some compensation for loss of intercostal muscle function. People with a lower cervical injury may have a vital capacity of 1.2–1.5 L at first, rising to 2 L or more, which allows 80% of patients with a lesion at or below C4 to be weaned from IPPV (Sykes and Young, 1999, p. 94). Reflexes can also be beneficial by reducing muscle wasting and osteoporosis, but if disabling spasms are provoked by minor stimuli, baclofen may be required. Osteoporosis is minimized by weight-bearing and the drug pamidronate (Nance *et al.*, 1999). Bronchodilators may be required for parasympathetic-induced bronchospasm. High-dose steroids administered within 8 hours of injury may improve neurological recovery (Bracken *et al.*, 1997).

Respiratory rehabilitation is lengthy for people with high lesions and may be hindered by a 48% incidence of depression (Krause, 2000). This is eased by promoting communication, sometimes with the help of a speech–language therapist, and ensuring that patients have as much control over their environment and treatment as feasible. Depression increases platelet aggregation and DVT risk (Seiner, 1999). Antidepressants that alter serotonergic transmission are not contraindicated but have been known to increase spasticity (Stolp-Smith, 1999).

Ventilator-dependent patients can gain some degree of independence with biofeedback (Morrison, 1988), glossopharyngeal breathing (p. 180 and Appendix C), short periods on a portable ventilator with a mouthpiece, or possible use of β_2-stimulants to improve muscle strength (Signorile, 1995). Exercise training in the form of arm ergometry and incentive spirometry has shown a 24% increase in FVC (Crane, 1994). There is enough evidence on the benefits of inspiratory muscle training for it to be attempted with all patients, either with a mouth-trainer or weights on the abdomen (K.H. Lin, 1999), possibly with the addition of expiratory muscle training (Ehrlich *et al.*, 1999). Training the surviving expiratory muscles may improve coughing (Gounden, 1993). Non-invasive ventilation can provide a fuller and more enjoyable life than invasive ventilation (Bach and Haas, 1996, p. 450).

Phrenic nerve pacing can coax the diaphragm to life and gain freedom from the ventilator, allow near-normal speech, travel, employment and greater independence (Creasey, 1996). Intercostal electrodes can be added (Dunn *et al.*, 1995). Magnetic stimulation of expiratory muscles may assist coughing (Lin, 1998).

Management of problems such as pain (Bryce, 2000), spasticity and sleep apnoea (Burns, 2000) is central to rehabilitation because these can impair lifestyle more than the extent of the injury (Westgren, 1998). The importance of these aspects of quality of life is shown by a study that found marginally disabled patients to have a higher suicide rate than those with functionally complete lesions (Hartkopp *et al.*, 1998). The importance of accepting patients' feelings is underscored by Laskiwski (1993) who found that expressions of despair were necessary for successful rehabilitation; they represented the abandonment of impossible hopes and the formulation of realistic goals.

Exercise training should be initiated early in the rehabilitation process, to minimize the cardiovascular deconditioning common in wheelchair-users. After the first 2 years, mortality parallels that of the general population, and although some patients develop maladaptive lifestyles, support and encouragement in the early

stages enable most to find the determination to rebuild their lives, including the ability to enjoy sex and have children (Linsenmeyer, 2000). Many patients report that their initial response was that death was better than living with such a disability, but one study found 92% glad to be alive, and it is a tribute to the human spirit that 'those who have a WHY to live will put up with almost any HOW' (deLateur, 1997).

Acute head injury

Nowhere is accurate assessment and finely tuned clinical judgement more vital than in the early stages of acute head trauma. Methods to control intracranial pressure and prevent lung problems may be in conflict, and are often complicated by other trauma.

The effect of most injuries is maximal at onset, but head trauma may precipitate a process that sometimes converts a mild injury into a life threatening condition. Primary damage sustained at the time of impact is irreversible, but secondary damage, which encompasses every mishap that befalls thereafter, can double mortality by reducing oxygen delivery to the brain (Wald, 1993). Secondary damage varies with the quality of management.

Effect of head injury on the respiratory system (Figure 15.1)

Chest infection is second only to intracranial hypertension as a cause of death following head injury (Rudy, 1991). Reasons for this and other respiratory complications are legion:

- Damage to the respiratory centre may cause abnormal breathing. Hyperventilation and lowered P_aCO_2 cause tissue hypoxia (Figure 15.2). Hypoventilation and raised P_aCO_2 cause vasodilation and raised intracranial pressure, usually as a terminal event. Cheyne–Stokes or ataxic breathing are signs of severe damage.
- Loss of protective pharyngeal reflexes in an unconscious patient may cause acute aspiration.
- Associated trauma such as facial injury, fractured ribs, haemopneumothorax or lung contusion compromise the airway or impair gas exchange.
- Immobility, recumbency and depressed consciousness cause shallow breathing and impaired cough.
- Over-enthusiastic fluid restriction, in an attempt to reduce cerebral oedema, can lead to hypotension and reduced oxygen delivery.

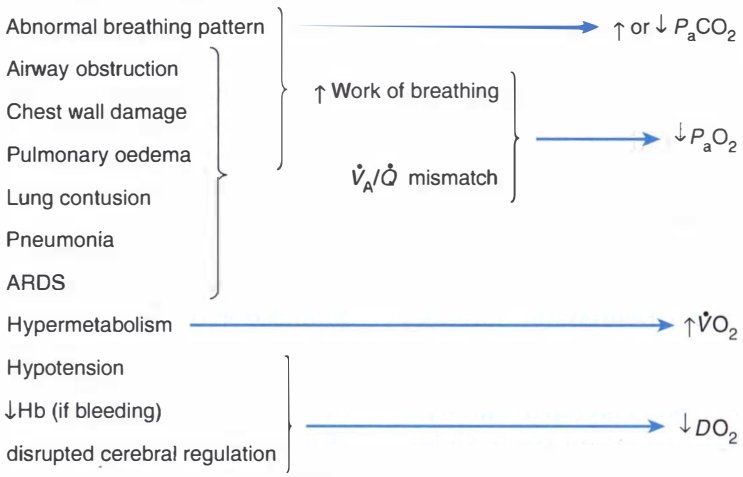


Figure 15.1 Effect of acute head injury on gas exchange and oxygen delivery. Some factors overlap. *ARDS = acute respiratory distress syndrome; DO_2 = oxygen delivery; $\dot{V}O_2$ = oxygen consumption.*

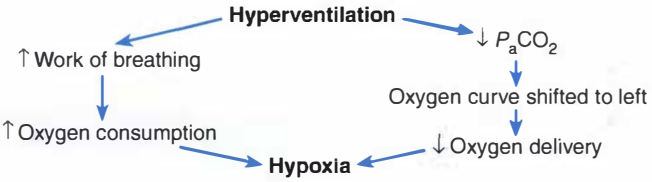


Figure 15.2 Effect of hyperventilation in acute head injury.

- Over-enthusiastic fluid administration, in an attempt to maintain cerebral perfusion, can cause pulmonary oedema. In addition, severe head injury can release catabolic hormones such as noradrenaline at up to seven times the normal level (Bruder, 1998), creating a massive sympathetic discharge, vasoconstriction and a surge of fluid into the pulmonary circulation to cause neurogenic pulmonary oedema (Kerr, 1998).
- Inflammatory mediators delivered from the injured central nervous system into the systemic system predispose to multisystem failure (Kochanek, 1999).
- Pneumonia is common in the early stages if acute aspiration has occurred at the time of injury or emergency intubation. It is less common in later stages because many patients are young and few have underlying medical illness (Hsieh *et al.*, 1992).
- Later problems may arise, e.g. DIC (p. 408), because of fluid imbalance, multisystem failure or fat embolism.

Effect of head injury on the brain

CSF: cerebrospinal fluid
ICP: intracranial pressure (normal 0–15 mmHg, critical > 20 mmHg)
CPP: cerebral perfusion pressure (normal > 70 mmHg, critical < 60 mmHg)
MAP: mean arterial pressure (normal 90 mmHg, critical < 80 mmHg)

Primary injury is caused by bleeding, contusion, and shearing forces in which the oscillating brain distracts nerve fibres from their bodies. Secondary damage is caused by cerebral oedema, raised ICP, hypoxaemia, anaemia due to bleeding, hypotension, hypertension and infection.

The skull contains 80% brain tissue, 10% CSF and 10% blood volume (Fisher, 1997). Like other tissue, the brain swells when damaged, reaching a maximum 24–48 hours after injury. Initially, swelling can be accommodated by displacement of CSF and venous blood into the spinal subarachnoid space and jugular veins. When these compensating mechanisms have reached their limit, a small increase in cerebral oedema within the rigid container of the skull causes a disproportionate upsurge in ICP, as shown in Figure 15.3. Extreme intracranial hypertension may cause coning, in which the brainstem herniates through the foramen magnum.

A decrease in CPP is the principal mechanism

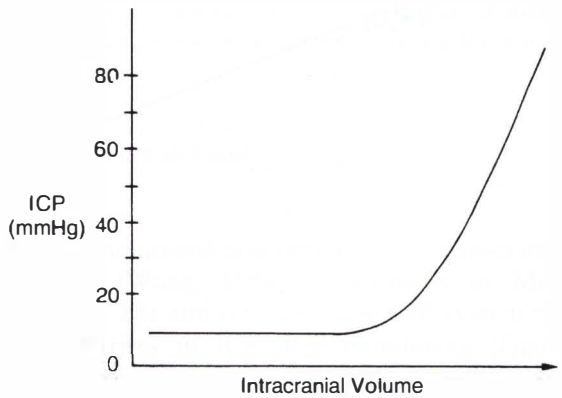


Figure 15.3 Intracranial pressure–volume curve. ICP is stable at first, but spatial compensation is exhausted at the inflection point and further swelling causes a steep rise.

by which elevated ICP exerts secondary damage (Chitnavis, 1998). CPP is the driving force of cerebral circulation and must be kept above 70 mmHg to perfuse the brain (Punt, 1989). This requires an adequate blood pressure but is compromised by a high ICP. MAP (which is related to blood pressure) and ICP are in effect competing for space:

CPP = MAP – ICP.

The picture can be further complicated if autoregulation is affected. Normally cerebral blood flow remains constant over a CPP range of 50–150 mmHg due to compensatory vasodilation as a response to hypoxia or hypotension. If this mechanism is damaged by brain injury, ICP follows MAP passively rather than remaining independent, and BP must therefore be tightly controlled. Medical intervention may be needed to regulate cerebral vascular resistance (Zhuang *et al.*, 1992).

Intracranial dynamics are reflected in a vicious cycle that exacerbates the secondary effects of head trauma (Figure 15.4). Lung complications can cause hypoxia, to which brain tissue is particularly sensitive because of its high oxygen requirements and dependence on aerobic glucose metabolism. Hypoxia causes cerebral oedema, and disturbances in P_aCO_2 add to this woeful picture.

Cerebral haemodynamics and oxygen metabolism are also affected by vascular resistance and oxygen extraction variables (Cruz, 1995). It is no wonder that head injuries have a reputation for being treacherous.

The effect on the brain, and the emotional effect on the patient, cause 42% of patients to show evidence of major depression from the acute stage onwards. This interferes with recovery (Rosenthal *et al.*, 1998).

Factors that increase ICP

ICP is keenly sensitive to a multitude of factors.

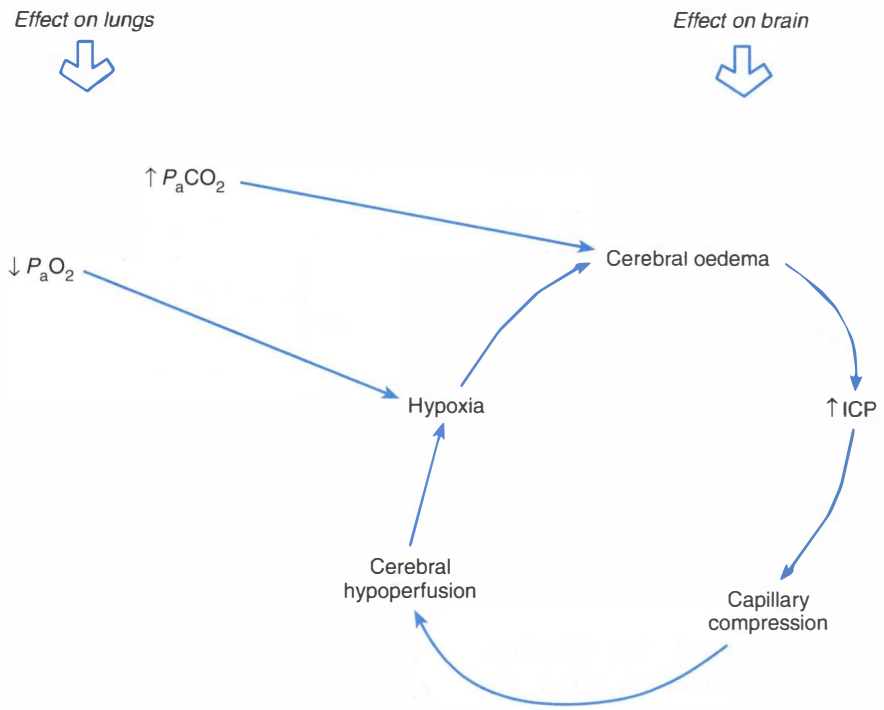


Figure 15.4 Vicious cycle set up by acute head injury.

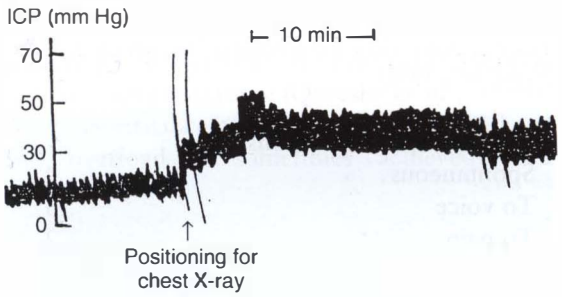


Figure 15.5 ICP tracing in a severely head-injured patient, showing prolonged elevation after position change. (From Shalit, M. N. and Umansky, F. (1977) Effect of routine bedside procedures on ICP. *Israel J. Med. Sci.*, **13**, 881–886, with permission.)

- Head-down postural drainage increases arterial, venous and intracranial pressures because cerebral veins have no valves. It also impairs compensatory venous outflow and is contraindicated in the acute stage (Lee, 1989).
- Turning the patient increases ICP (Chudley, 1994), much of this being due to head movement obstructing drainage from the brain (Figure 15.5).
- Head movement, coughing, suction, manual hyperinflation, vibrations and percussion can impede compensatory outflow from the brain and raise ICP (Paratz, 1993). Outflow is also obstructed by extreme hip flexion (Mitchell and Mauss, 1978), a tracheal tube tied rather than taped, or a poorly applied cervical collar (Dodd *et al.*, 1995).
- Hypertension increases ICP, and hypotension reduces CPP. Reduction in brain perfusion by hypotension is one of the most important predictors of mortality (Lannoo, 1998). It can double death rates (Pietropauli *et al.*, 1992), but is not widely appreciated and occurs particularly at the following times:
  - on admission, when a patient may be quietly bleeding into the abdomen and losing consciousness because of hypotension rather than brain injury
  - during surgery, when BP may be deliberately kept low (Pietropauli *et al.*, 1992).
- Even deeply comatosed patients show a surprising sensitivity to conversation over their beds. Discussion about their condition increases ICP more than general discussion (Mitchell and Mauss, 1978). However, when relatives talk to patients, a reduction in ICP may be seen (Chudley, 1994).
- ICP is increased by pain and discomfort, including injections, BP measurements (Ersson *et al.*, 1990), noise, restraints, movement of the tracheal tube, Yankauer in the mouth, arousal from sleep or emotional upset (Mitchell *et al.*, 1981). Head-injured people show an exaggerated response to the pain of associated injuries (Mirski, 1995). Passive movements can transiently increase ICP (Brimioulle *et al.*, 1997).
- Transport between hospitals causes an adverse event in 40% of patients and increased ICP in 51% (Waldmann, 1998). Within-hospital transfer is also hazardous.
- Delayed intracranial hypertension may occur after removal of monitoring devices and can be anticipated by a raised WBC count (Souter *et al.*, 1999). Systemic infection may exacerbate brain damage (Kochanek, 1999).

Most of these factors warn physiotherapists to keep their distance, but the importance of maintaining gas exchange is a cogent reminder not to stray too far.

General management

Monitoring

CPP is monitored non-invasively by transcranial Doppler (Wong, 1994) or indirectly by MAP and ICP. The aim is to optimize CPP even at the cost of rises in ICP. ICP monitoring (Figure 15.6) is needed for comatosed patients if the Glasgow Coma Scale score falls below 8, or if cerebral oedema is identified on CT scan (White, 1992). Impending elevations of ICP should be predicted and managed while still within the

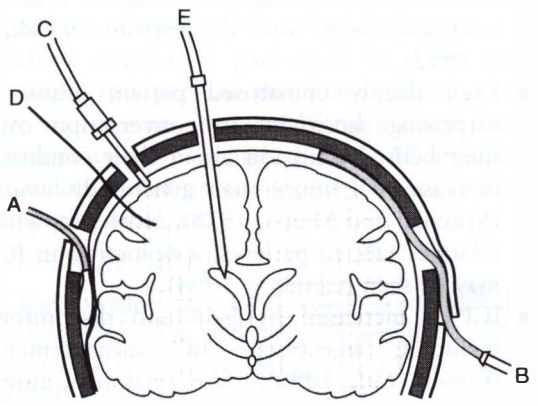


Figure 15.6 ICP monitoring: A = epidural sensor, which leaves the dura intact; B = subdural bolt, inserted into the subdural space; C = subarachnoid bolt, inserted into the subarachnoid space; D = parenchymal catheter, implanted in brain substance; E = intraventricular catheter, implanted into non-dominant lateral ventricle, can be used to withdraw CSF for diagnosis or therapy, risks ventricular displacement. (From Vos, H. R. (1993) Making headway with intracranial hypertension. *Am. J. Nur.*, **93**, 28–36)

normal range (Chitnavis, 1998). Clinical manifestations of neurological dysfunction are not apparent in a paralysed patient, and for others they simply indicate that secondary damage has already occurred (Harrington, 1993). Reliance on clinical evidence of raised ICP can lead to blind and inappropriate interventions (Jeevaratnam and Menon, 1996). Monitoring is assisted by the following:

- bedside cerebral blood flow measurement (Wietasch *et al.*, 2000)
- end-tidal CO_2 ($ETCO_2$), which gives a continuous indication of P_aCO_2
- jugular venous oximetry, which indicates cerebral oxygenation (Gopinath *et al.*, 1999)
- EEG, which measures spontaneous brain activity and is used to detect seizure focus or to localize the source of irritation
- CT scan, which can be transmitted electronically from a general ICU to a neurosurgical unit for advice (Waldmann, 1998)
- Glasgow Coma Scale, which indicates the degree of injury (Box 15.1).

Box 15.1 The Glasgow Coma Scale; a score below 8 indicates severe injury and anticipates mortality up to 36% (Waldmann, 1998)

Best eye-opening response	
Spontaneous	4
To voice	3
To pain	2
None	1
Best verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incoherent	2
None	1
Best motor response	
Obeys commands	6
Localizes pain	5
Withdraws from pain	4
Flexes to pain	3
Extends to pain	2
None	1

Head elevation

It is common practice to elevate the bed-head 15–35° in order to reduce ICP and encourage CSF outflow. However, this causes hypotension in hypovolaemic patients (Arbour, 1998) and may compromise CPP (Feldman, 1992). Head position should be established individually rather than routinely (Ropper *et al.*, 1982) and not raised above 60° (Durward *et al.*, 1983). The head should always be kept aligned with the body to allow compensatory outflow.

Fluid management

Normovolaemia is the target (Waldmann, 1998). Excess fluid rushes into injured brain cells and increases cerebral oedema, and dehydration reduces brain perfusion. Small changes in blood osmolality exert a strong effect on brain water, and initial fluid resuscitation is probably best achieved with hypertonic saline (Prough, 1996) whose sodium ions do not cross the blood–brain

barrier, do not risk brain swelling, and do not cause renal dysfunction as seen with repeated mannitol administration (Qureshi *et al.*, 1998). Rapid maintenance of cerebral perfusion without fluid overload is sometimes achieved with inotropes.

Nutrition

Nutritional support is needed because energy expenditure may be doubled for up to 4 weeks (Borzotta, 1994) and a hypermetabolic state may persist for a year (Woodward, 1996). Swallowing problems require a speech–language referral. Paralytic ileus may be a hindrance during the first fortnight but enteral feeding should be started when possible because IV feeds can increase cerebral oedema and cause neuronal damage from hyperglycaemia and lactic acidosis (Woodward, 1996). A stable blood sugar is required to prevent hypoglycaemia (Adam and Osborne, 1997, p. 250).

Temperature control

Temperature must be tightly regulated. A pyrexia of 1° can raise cerebral metabolism by up to 10% (Vos, 1993) and increase fluid requirements. Hypothermia causes arrhythmias, shifts the oxygen dissociation curve to the left, and if it causes shivering, increases oxygen consumption up to fivefold (Frost, 1996).

Drug therapy

Judicious doses of the osmotic diuretic mannitol decrease blood viscosity and enhance cerebral blood flow (Waldmann, 1998) but can cause dehydration. Sedatives reduce brain metabolism, but monitoring is required to ensure that they do not reduce CPP (Papazian, 1993), especially in hypovolaemic patients. For pain, opioids can be used so long as MAP is maintained (Suarez, 1999), but Entonox is contraindicated (Moss and McDowall, 1979). Anaesthetic agents may be used to reduce cerebral metabolism. Phenytoin is used to control seizures which could otherwise cause cerebral hypoxia. Vasoconstrictor drugs increase CPP but may impair oxygenation to areas around contused brain tissue and other vital organs. Prophylactic antibiotics may increase the virulence of infection and subsequent mortality (Demling, 1990). Although steroids such as dexamethasone reduce vasogenic oedema around brain tumours, they are unable to reduce traumatic cerebral oedema because intracellular fluid is inaccessible (Punt, 1989).

Mechanical ventilation

Intubation may be needed to maintain a clear airway. IPPV may be needed to regulate an unstable breathing pattern, ensure oxygenation, control ICP or manage chest complications. Paralysis reduces oxygen consumption but its routine use has been questioned since Hsiang (1993) found that it can increase complications.

Hyperventilation is sometimes imposed to lower P_aCO_2, induce cerebral vasoconstriction and lower intracranial blood volume. The effect is instant but not sustained because over the next 6–24 hours buffering of respiratory alkalosis relaxes the cerebral vessels. Over-enthusiastic hyperventilation can cause cerebral ischaemia (Ruta, 1993), as signalled by slowing of the EEG. P_aCO_2 must not be allowed to drop below 3.3 kPa (25 mmHg).

High PEEP risks hypotension and further impairment of venous outflow from the brain. High frequency ventilation may be beneficial in eliminating BP fluctuations and maintaining venous return.

Physiotherapy

The hallmark of physiotherapy is maximum involvement and minimum intervention. Involvement is by:

- frequent assessment to assist delicate risk/benefit decisions
- supervision of handling to minimize ICP disturbance.

Intervention is unwise in the presence of cardiovascular instability, hypotension or ICP above 15 mmHg (Paratz, 1993). If it is essential, a drug to moderate ICP should be given beforehand. Treatment can be timed to follow withdra-

wal of CSF. Quiet explanations are required for all patients, however deeply comatose. Stress reduction strategies have shown beneficial effects (Chudley, 1994).

Assessment

S_aO_2 is the main guideline because its assessment requires no handling and is continuous and instant. Any sign of impending chest complications requires preventive action. Observation of BP, ICP and $ETCO_2$ is required prior to and throughout treatment. In the absence of an ICP monitor, signs of a raised ICP are:

- ↑ pupil size
- pupil unresponsive to light
- ↓ consciousness
- change in vital signs, breathing pattern or muscle tone
- vomiting.

If the patient is not intubated, a minitracheostomy is advisable if suction is necessary. Nasal suction is contraindicated in the presence of:

- watery CSF leaking from the nose or ear, indicating a connection between the subarachnoid space and nasal passages, thus risking infection
- severe epistaxis, indicating tissue disruption and risking the catheter entering the sinuses.

Positioning

Turning is safe with ICP < 15 (Chudley, 1994). Patients should be log-rolled slowly using a turning sheet, with one person solely responsible for maintaining head alignment. A kinetic bed may be used (Tillett, 1993). Accurate positioning in side-lying with neutral head position aids prophylactic chest care. However, for patients with unstable ICP and low risk of lung complications, it is best to leave the patient supine in the early stages if there is a suitable mattress for preventing pressure sores. Neck flexion must be prevented by using a thin pillow or none at all (Jones, 1995b). Calf pressure should be avoided because DVT is a significant risk (Gersin, 1994).

Manual hyperinflation

Ersson *et al.* (1990) found that patients are exposed to equal risks of impaired CPP with both MH and suction, and should always be sedated beforehand. If MH is essential, it should be brief and avoid disturbance to P_aCO_2.

Manual techniques

If these are essential, percussion should be rhythmic, smooth and gentle, and vibrations should be fine and avoid affecting intrathoracic pressure. A vibrator can be used.

Suction

Elevation in ICP during suction can be dramatic (Rudy, 1991), mild (Brucia, 1996), transient, or, in a third of patients, prolonged beyond 15 minutes (Kerr *et al.*, 1996). Variations in response are due to the degree of hypoxia and impairment of venous outflow from coughing and mechanical stimulation. If suction is indicated, the following precautions are advised:

- rest from previous activity
- 100% oxygen before and afterwards
- head kept strictly in alignment
- tracheal tube stabilized throughout
- contact with the carina avoided (Brucia, 1996)
- avoidance of more than one suction pass at a time, and use of manual hyperventilation to reduce ICP when necessary (Crosby and Parsons, 1992).

Exercise

Extreme hip flexion should be avoided in the acute stage. If flaccidity is present with no altered muscle tone, it may be best to avoid any movements in the first few days. If spastic patterning develops, appropriate positioning must be maintained and factors that increase tone avoided, e.g. pain, anxiety, infection, pressure under the feet and the weight of bedclothes. Staff and carers should be taught positioning and handling to encourage inhibitory control over spasticity. If increased tone or clonus is identified, immediate splinting and

stretching to maintain dorsiflexion has been recommended (Moseley, 1997), before range of movement is lost. Early attention to range of movement is essential because the majority of head-injured patients have been found to have long-term contractures (Watkins, 1999).

Teamwork

Pre-planning is needed to avoid a cumulative rise in ICP. Most teams arrange physiotherapy, nursing and other interventions as far apart as possible, although some find it best to do everything at once and then let the patient settle. Co-ordinated teamwork is especially important in the first vulnerable week.

Head injury can impede identification of pain. Pain limits rehabilitation, increases depression and if neglected may become chronic (Lahz, 1996). The physiotherapist is the most likely team member to detect and report pain.

Rehabilitation starts in the ICU. MacKay (1992) has shown that length of coma can be cut to a third by a multidisciplinary programme after the immediate acute period, using orientation, sensory stimulation, exercise and family involvement. Long-term rehabilitation allows cognitive improvement to continue for up to 10 years (Prough, 1996).

The persistent vegetative state cannot be diagnosed for at least a year after injury, but patients are subject to considerable misdiagnosis (Watson *et al.*, 1999).; Potential for improvement remains, and our limited ability to find evidence of a functioning mind does not preclude its existence.

Myasthenia gravis

This progressive autoimmune disorder affects the neuromuscular junction and weakens muscles in proportion to their use. It is confined to the eye muscles in 20% of patients (Oh, 1997, p. 434), and for others the limb and trunk muscles are usually affected asymmetrically. The patient may complain of fatigue rather than weakness. Treatment is by anticholinesterase drugs, steroids, occasionally plasmapheresis, and thymectomy via sternotomy (Nilsson, 1997).

The disease is punctuated by myasthenic and cholinergic crises, which are treated by increased drugs for the former, and atropine to counteract overadministration of drugs for the latter. Intensive care is required after thymectomy, during crises or if bulbar weakness threatens the airway. Physiotherapy includes clearance of the excess bronchial secretions stimulated by anti-cholinesterase.

Botulism

Botulism, affects the neuromuscular junction. It is an infection derived from contaminated food or surgical wound infection. Bulbar and respiratory muscles may become paralysed, and sometimes IPPV is required for several months.

Tetanus

The tetanus bacillus produces one of the most lethal poisons known. It is a common resident of superficial soil and enters the body through a wound. It infects any dead tissue and spreads to the central nervous system, leading to muscle rigidity, autonomic instability and sometimes convulsions. Patients experience pain, stiffness and inability to open their mouth (lockjaw). Spasms of the larynx or diaphragm are life-threatening and require intubation and IPPV respectively. Sedation and sometimes muscle relaxants are needed. Risks are contractures, aspiration, DVT and cardiovascular complications. Recovery occurs over 6 weeks, but residual stiffness is common.

Critical illness neuropathy

A self-limiting neuropathy occurs to varying degrees in some patients, precipitated by metabolic upset, paralysing and steroid drugs, and immobility (O'Leary and Coakley, 1996). In particular, it has been found in 70–80% of patients with sepsis or multisystem failure (Hund, 1999). Failure to recognize the condition leads to misjudgement of weaning ability. Recovery is usually complete but residual peroneal weakness may occur. Physiotherapy is needed little and often to optimize musculoskeletal function without exhausting the patient.

CHEST TRAUMA

Penetrating injuries can cause blood loss, infection and haemopneumothorax (blood and air in the pleura). Haemoglobin in a trauma patient needs to be above 10 g/100 mL for adequate oxygen delivery (Nolan, 1996).

Penetrating injuries, e.g. stab wounds, require a programme of chest mobility and exercise, as described by Senekal and Eales (1994). Before mobilizing patients who have a haemothorax or haemopneumothorax, they should be asked to clear some of the blood from the pleural space by positioning themselves with the chest drain dependent for a few minutes.

Blunt injuries, e.g. from road traffic accidents, spread force over a wide area from compression, shearing and tension, often causing rib fractures and lung contusion. Full cervical protection should be maintained after blunt trauma until comprehensive radiographs have been examined by a senior orthopaedic surgeon (Nolan, 1997).

If the abdomen sustains blunt trauma, the diaphragm may rupture, usually on the left because the liver buttresses the right side. Diaphragmatic rupture causes abdominal viscera to herniate into the chest, and surgical repair is required. The diagnosis is often missed but the X-ray shows a displaced hemidiaphragm.

Rib fracture

A third of patients with traumatic rib fractures develop pulmonary complications (Ziegler, 1994). Pain causes a restrictive defect, exacerbated if there is chest wall derangement. Complications include pneumothorax (sometimes under tension), surgical emphysema and haemothorax. Each pleural space can hold about 2 L of blood, so an average adult, whose blood volume is 5–6 L, can exsanguinate into the thorax if haemorrhage is uncontrolled.

The commonest locations are the third to tenth ribs, often laterally where there is no muscle protection (Figure 15.7). Fractures of the well-protected first three ribs indicate great force and are often accompanied by intrathoracic injury. Lower rib fractures may be accompanied by intra-abdominal injury.

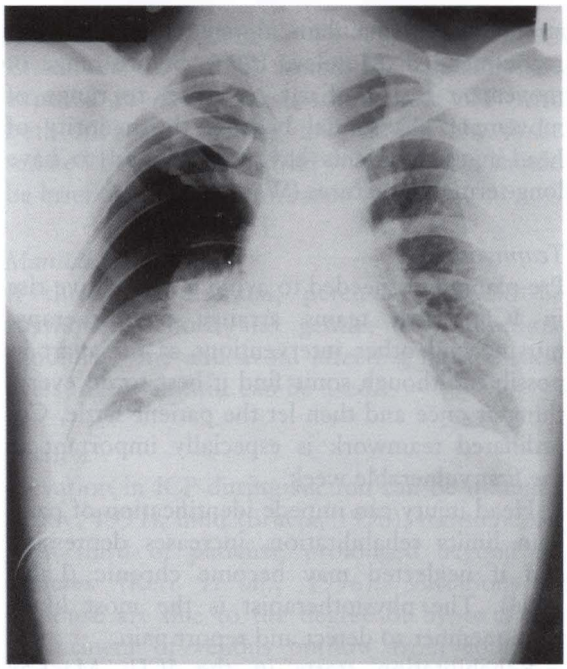


Figure 15.7 Multiple rib fractures following a road traffic accident. On the right, ribs 4–6 are fractured laterally and ribs 6–11 posteriorly. On the left, the first rib is fractured. There is a right pneumothorax and chest drain.

An incompetent segment of chest wall that is large enough to cause paradoxical breathing and impair respiration is called a flail segment (Figure 15.8). This requires at least two ribs to be fractured in more than one place. Paradoxical movement may not be apparent in the first day or two if muscle spasm stabilizes the chest wall.

Assessment

Palpation elicits exquisite pain and is not necessary, but crepitus may be felt by laying the hand gently over the tender area. The X-ray may underestimate the presence and extent of rib fractures by 50% (Mayberry, 1997), especially if they are anterior. A line of fractures suggests single trauma whereas scattered fractures suggest repeated injury as with alcoholism, or bony weakness as with malignancy. Cough fractures

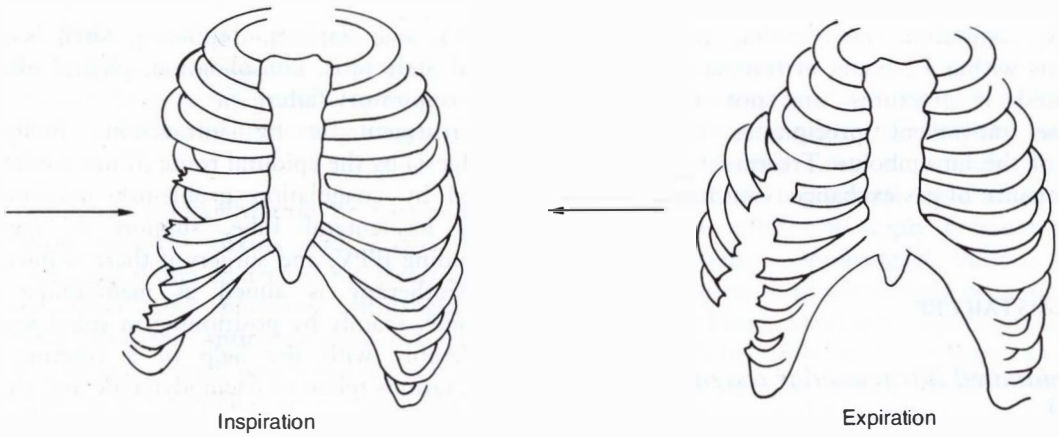


Figure 15.8 Flail chest caused by fractured ribs. The unstable segment is sucked in on inspiration and pushed out on expiration.

may occur in frail patients or those with osteoporosis. Hyperflexion over a seat belt may cause transverse sternal fracture.

Treatment

Early regional pain control is essential, usually by a thoracic epidural. If chest drains are used for pneumothorax or haemothorax, local anaesthetic can be administered through the drain. TENS may be helpful (Sloan *et al.*, 1986) and Entonox can be administered if there is no pneumothorax. A cough belt or towel supports coughing.

Once pain is controlled, regular incentive spirometry is advisable. If gas exchange is impaired, CPAP or BiPAP provides pneumatic stabilization. Minitracheostomy may be required for sputum retention. Soft tissue injuries are usually present and will need attention. Early mobility is to be encouraged.

Lung contusion

Shearing or crushing forces lead to pulmonary laceration and a form of 'blood pneumonia' known as contused lung. Blood-filled alveoli cause shunt, $\dot{V}_A/\dot{Q}$ mismatch and hypoxaemia. Lung compliance is reduced and oedema develops over 48 hours.

Assessment

Signs of contusion are dyspnoea and bloody secretions. Peripheral ground-glass mottling develops on X-ray over 12–48 hours, or immediately if severe, followed by absorption of the infiltrates after 3–5 days or progression to ARDS. Perfusion of unventilated lung leads to shunt and hypoxaemia.

Treatment

IPPV may be needed if hypoxaemia is refractory to oxygen therapy or CPAP. If secretions are present, contused lungs do not take kindly to percussion and vibrations. Mechanical vibrators may help, and an oscillating bed has been found to reduce chest infections (Fink *et al.*, 1990). If frank bleeding is present, suction is contraindicated unless breathing is obstructed by secretions.

Fat embolism

Trauma or orthopaedic surgery may cause fat to be released from bone marrow into the circulation, leading to capillary inflammation and occlusion. Organs with a high blood flow are particularly affected, including the lungs, which may develop ARDS. Warning signs are breath-

lessness, agitation, tachycardia, pyrexia and cyanosis within 72 hours of trauma. The risk is increased if fractures are not immobilized because movement precipitates intravascular entry of the fat embolus. Treatment is aimed at maintenance of gas exchange, vital functions and hope.

SYSTEMS FAILURE

Disseminated intravascular coagulation (DIC)

The normal response to tissue damage is a contained explosion of thrombin to initiate coagulation and limit blood loss. This can become uncontained after severe damage such as burns, brain or spinal cord injury, fat embolism, any form of shock, and sometimes the drug Ecstasy. This leads to DIC, in which liberated thromboplastin activates uncontrolled coagulation and blocks vessels with clumps of platelets and fibrin, causing ischaemia and organ damage. When clotting factors and platelets have been depleted, bleeding can occur from the slightest trauma, including suction. Fourrier (1992) describes multisystem failure and death as common outcomes of DIC, sometimes referred to as 'Death Is Coming'.

Acute pancreatitis

An inflamed pancreas can be caused by gallstones, alcoholism, drug reaction or eating disorder. A fifth of patients with acute pancreatitis develop a severe attack with 25% mortality (Reece-Smith, 1997). Activated pancreatic enzymes autodigest pancreatic tissue and set off a cascade of ischaemia, inflammation, vasodilation, increased capillary permeability and DIC. Progressive liquefaction of the pancreas may occur, leading to abscess formation and sepsis. Patients suffer paralytic ileus, which increases the risk of aspiration because of delayed gastric emptying, a rigidly distended abdomen and continuous epigastric pain, worse in supine. Diaphragmatic dysfunction is compounded by its proximity to the inflamed pancreas (Matuszczak, 1998), and diaphragm splinting often leads to basal atelectasis, consolidation, pleural effusion and respiratory failure.

Treatment is by intravenous hydration, analgesia by the epidural route (if not contraindicated by coagulation problems), nutrition by long nasoenteral tube, support as required including IPPV, and surgery if there is infection. Physiotherapy is aimed at maintaining lung volume, mainly by positioning in side-lying and preferably with the help of a rotating bed. Precautions relate to haemodynamic and electrolyte upset.

Collagen vascular disease

The following autoimmune disorders may lead to multisystem failure:

- Goodpasture's syndrome, which can cause nephritis and lung haemorrhage
- Wegener's granulomatosis, which is a triad of upper respiratory tract lesions, pulmonary disease and glomerulonephritis
- polyarteritis nodosa, which causes inflammation and necrosis of arteries, leading to ischaemia of any organ
- systemic lupus erythematosus (SLE), which involves chronic inflammation of many systems including skin, nervous system, kidney and blood vessels.

Severe collagen vascular disease is suspected if there are blood-stained secretions on suction, spontaneous bleeding or abnormal clotting studies. It will not resolve until the trigger mechanism is removed, and treatment is aimed at the underlying condition, along with modification of the immune response with steroids, restoration of haemostasis and support of failing systems. Nasopharyngeal suction is contraindicated, and tracheal suction requires extra caution because of the risk of bleeding.

Kidney failure

The kidney fails acutely in response to hypotension, hypoxia or multisystem failure, and is a measure of severity of the underlying condition. Acute renal failure occurs in 30% of critically ill

patients (Galley, 2000) and is associated with complex multisystem problems. It is suspected if urine output drops or urea and creatinine levels rise. Patients in renal failure can be supported in several ways:

- Continuous haemofiltration or haemodiafiltration removes toxins and excess fluid slowly and allows control of BP, electrolytes, medication and nutrition (Kutsogiannis, 2000). Moderate anticoagulation is required but a specialist renal unit is not necessary.
- Intermittent haemodialysis is faster but can cause BP disturbance, pulmonary and systemic inflammatory changes, wheezing, hypoxaemia due to capillary blockage and bleeding due to anticoagulation. Vascular access is commonly by an arteriovenous fistula at the wrist.
- Peritoneal dialysis risks infection, impairs basal ventilation and is now little used. Physiotherapy should coincide with the end of the emptying cycle to ensure free diaphragmatic movement.

Physiotherapists must develop a healthy respect for the renal vascular catheter or 'vascath', as disconnection leads to major blood loss. Other precautions are to be watchful of fluid volume changes or hypertension and to be aware of the risk of bleeding as patients are anticoagulated. Details of kidney disease are on page 114.

Liver failure

Liver cells are vulnerable to hypoxia. Acute liver failure leads to multisystem involvement but support of these systems may allow the liver to recover or permit survival until a donor organ is available for liver transplant. DIC often occurs because impaired clearance function of the liver allows activated factors to rampage through the body. Kidney failure occurs in 50% of patients with liver failure, although blood urea is not raised because of reduced urea production by the failing liver.

Cirrhosis may obstruct the portal vein and create portal hypertension, transmitting back pressure throughout the portal system. Dilated surface blood vessels in the lower oesophagus may cause oesophageal varices. Severe bleeding from the varices requires insertion of a Sengstaken tube via the nose or mouth into the stomach, usually for several days, from which balloons are inflated to apply pressure to the bleeding points. Oesophageal varices are a contraindication to physiotherapy because of the tendency to bleed. More precautions for treating patients with liver disease are on page 114.

Fulminant hepatic failure occurs in people with previous normal liver function and is most commonly caused by paracetamol overdose. After a quiescent 24 hours, patients develop a raised ICP, low potassium and blood sugar, and present a hyperdynamic picture of high cardiac output and low systemic vascular resistance. Handling and suction should be minimal and the patient usually remains supine. Transplant is the treatment of choice, otherwise patients may die from cerebral oedema, hypotension or sepsis.

Liver transplantation requires a 'Mercedes-Benz' double subcostal incision and laparotomy, necessitating close attention to pain relief. Postoperative complications include right basal atelectasis, pleural effusion, liver rejection and the effects of immunosuppressive drugs. Rehabilitation is surprisingly rapid once the toxin-producing liver has been removed. Some patients are so poisoned by their own liver that it is removed even if no donor is immediately available.

MULTISYSTEM FAILURE

Bacteraemia: viable bacteria in blood.
Infection: invasion of normally sterile host tissue by microorganisms.
Septicaemia: systemic infection in which pathogen is present in blood.
Endotoxin: toxin released by Gram-negative bacteria as they disintegrate.
Sepsis: systemic response to infection, manifest by two or more of the following:
temperature > 38° or < 36°C

HR > 90/min
RR > 20/min or P_aCO_2 < 4.3 kPa (32 mmHg)
WBC > 12 000

Systemic inflammatory response syndrome (SIRS): generalized inflammatory response, manifest by two or more of the above:

Sepsis syndrome: SIRS caused by infection.

Multisystem failure: systems failure caused by direct insult or SIRS, in which homeostasis cannot be maintained without intervention. Also known as multiple organ failure or multiple organ dysfunction syndrome.

Shock: failure of oxygen supply to meet oxygen demand.

Most deaths in surgical ICUs are due to multisystem failure (Deitch, 1999). Once three or more organs have failed, mortality is over 90% (Molnar and Shearer, 1998).

Shock

In contrast to the layperson's shock–horror understanding of the term, shock occurs when the reserve capacity of tissue respiration is exhausted. Once oxygen delivery (DO_2) can no longer satisfy oxygen consumption ($\dot{V}O_2$), a cascade of damaging events ensues.

Hypovolaemic shock is caused by loss of fluid, e.g. haemorrhage or burns. Early physiological compensation is by redistribution of fluid from extravascular to intravascular space, and selective vasoconstriction to non-vital systems. A young person can lose 30% of his/her blood and still maintain BP and HR (Adam and Osborne, 1997, p. 335). Hypovolaemic shock is identified by the signs on page 362. Cardiac output is compromised, (pp. 113, 381).

Cardiogenic shock is caused by sudden heart failure, as in severe myocardial infarction. It is characterized by high CVP, low cardiac output and pulmonary oedema.

Septic shock occurs when sepsis-induced hypotension is unresponsive to fluid resuscitation. Sepsis causes a fever which resets the hypothalamic thermostat, leading to peripheral vasodilation in an attempt to lose heat, thus depleting perfusion to the viscera. Endotoxins stimulate excess nitric oxide production which augments uncontrolled vasodilation and in effect reduces circulating blood volume, creating a 'functional haemorrhage'. High cardiac output therefore cannot sustain an adequate BP. Hypoxia-damaged tissues cannot extract sufficient oxygen, as shown by $S\bar{v}O_2$ rising to 85% or more. Patients are pyrexial, flushed, tachypnoeic, hypotensive and have a bounding pulse. Vasopressors may compromise regional blood flow.

Other types of shock are *anaphylactic shock*, an allergic reaction by more than one system, and *neurogenic shock* following nervous system damage and loss of sympathetic tone. Both are characterized by widespread vasodilation and hypotension.

Shock follows a characteristic sequence:

1. inadequate tissue perfusion
2. anaerobic metabolism
3. lactic acidosis
4. metabolic acidosis
5. cellular damage
6. organ failure.

Causes of multisystem failure

Shock either causes multisystem failure directly, or becomes part of the process of a catastrophic event that makes excessive demands on oxygen consumption, e.g.:

- prolonged hypotension
- sepsis
- aspiration
- over-transfusion
- smoke inhalation
- head injury
- near-drowning
- fat embolism
- pulmonary embolism
- lung contusion
- poisoning/drug abuse
- peritonitis
- acute pancreatitis
- cardiopulmonary bypass
- multisystem disease

- DIC (p. 408)
- immunosuppression following trauma or surgery (Wichmann *et al.*, 1998).

Interaction of these predisposing factors can blur cause and effect, e.g. the inflammatory response can activate the coagulation process, and shock can stir up immunochaos. Multisystem failure is usually established within 24 hours of injury (Cryer *et al.*, 1999).

Pathophysiology of multisystem failure

If an amputated limb is reimplanted after a delay, it releases endotoxins which invade the body and set off an inflammatory domino effect. Re-amputation is required to prevent the rest of the body becoming poisoned. This analogy represents multisystem failure, but treatment is not so simple.

Hypoperfusion and reperfusion activate a deadly cascade of mediators from damaged cells, creating 'rogue inflammation'. This subverts the normal healing function of inflammation, escapes the usual control mechanisms and exacerbates rather than repairs injury. Autodestruction leads to increased permeability of epithelium.

The main culprit is the gut, whose vulnerability to hypoperfusion has earned it the name 'engine of multisystem failure' (Botterill and MacFie, 2000). Just 1 hour of gut ischaemia can cause cell necrosis, loss of mucosal integrity and leakage of gut bacteria into the circulation (Brown, 1994). Patients are in effect poisoning themselves, which explains why 50% of patients have no identifiable septic focus (Deitch, 1999). The main victim is the lung, because of its large vascular component, now poisoned and permeable.

Hypoxia is caused by:

- refractory hypoxaemia
- reduced gas diffusion at tissue level because of interstitial oedema
- impaired oxygen extraction due to damaged cells
- excess oxygen consumption due to a twice-normal metabolic rate.

Circulating catecholamines may increase cardiac output and total body blood flow but deranged autoregulation sends the circulating blood to resilient tissue such as skin and muscle at the expense of needy systems such as the gut and liver. Maldistribution of the circulation and defective microvasculature leads to progressive failure of other systems. Those most related to mortality are the kidney and liver. Those most relevant to the physiotherapist are the haematological and respiratory systems, leading to DIC and acute respiratory distress syndrome (ARDS).

Medical management of multisystem failure

Any potential septic focus needs treatment to prevent further stimulation of the inflammatory response, e.g. removal of dead bowel or stabilization of fractures. The main aim is then to restore normal homeostasis and sustain tissue perfusion rather than focus on a single system (a tyre blowout that wrecks a car is not corrected by replacement of the tyre). Ventilatory and haemodynamic support aims at maintaining DO_2 so that gastric intramucosal pH remains above 7.35 (Oh, 1997, p. 735). The balance between beneficial and damaging interventions is a fine one, and a formidable array of options can improve oxygen delivery but cannot directly assist oxygen extraction and has not notably reduced mortality.

DO_2 is promoted by respiratory support, inotropic support and vasodilators. $\dot{V}O_2$ is reduced by respiratory support, sedation, paralysis and avoidance of stress or pyrexia. Support may include packed red blood cell transfusion or haemofiltration to wash out circulating mediators (Lingnau, 1995). Lactate-induced metabolic acidosis can usually be corrected by manipulation of IPPV in order to affect P_aCO_2, but a pH of less than 7.2 requires haemofiltration. Severe sepsis can increase energy expenditure by 50% (Bruder, 1998) and nutritional support is essential, preferably enterally to stabilize the gut lining. However, the septic response hinders the utilization of nutrition, as shown by high nitrogen excretion and relentless muscle wasting (Green *et al.*, 1995).

Skilled fluid management is required because transfused fluid tends to escape into the leaky lung, while inadequate circulating volume hinders DO_2. Renal impairment complicates the picture. Adequate volume is guided by a CVP of 10–12 cmH_2O and PAWP of about 18 cmH_2O for optimum cardiac output.

Antibiotics are usually prescribed, but may exacerbate symptoms because destroyed bacteria release more endotoxin (Tangredi, 1998). Steroids may be helpful if initiated early and if there is Gram-negative septicaemia (Lefering, 1995).

Physiotherapy

Critical illness neuropathy develops in 70% of patients (O'Leary and Coakley, 1996). Meningococcal septicaemia can have severe musculoskeletal and neurological consequences, and hypoperfusion to the peripheries can lead to necrosis and gangrene. If fingers are affected, the hand needs to be carefully splinted in a functional position to optimize circulation and prevent contractures. Passive movements require extreme care to protect the skin.

ACUTE RESPIRATORY DISTRESS SYNDROME

Acute lung injury is parenchymal lung injury leading to alveolar–capillary membrane leak and non-cardiogenic pulmonary oedema. P_aO_2:F_IO_2 ratio is less than 40 kPa (300 mmHg). The severest form is acute *respiratory distress syndrome* (ARDS), also known as leaky lung syndrome, shock lung, white lung or, because it was first described in soldiers salvaged from the Vietnam battlefields, Da Nang lung. ARDS shows a P_aO_2:F_IO_2 ratio of less than 26 kPa (200 mmHg).

Pathophysiology

Lung tissue can be injured directly, e.g. by aspiration, contusion or smoke inhalation. It can be injured indirectly by toxins let loose by multisystem failure. As a result, both alveolar and vascular functions of the lung are ravaged by inflammatory mediators. The resulting sieve-like alveolar–capillary membrane allows flooding of the alveoli, leading to massive pulmonary oedema, which almost drowns the patient. A lung up to quadruple its normal weight causes compression atelectasis (Pelosi and Gattinoni, 1996) in dependent regions (Figure 15.9). Invading plasma proteins deplete surfactant, exacerbate atelectasis, increase shunt and widen $P_{A-a}O_2$. Vascular injury leads to pulmonary hypertension, which exacerbates oedema formation and inhibits right ventricular function.

The waterlogged and inflamed lungs become progressively and unevenly damaged. Some lung tissue is necrotic and consolidated, with the consistency of liver. Some is collapsed but potentially recruitable. Some is still undamaged and compliant, but this functioning tissue may only make up one-third of the normal lung capacity (Slutsky, 1993).

This so-called 'baby lung' creates a restrictive defect that worsens as the basic framework of

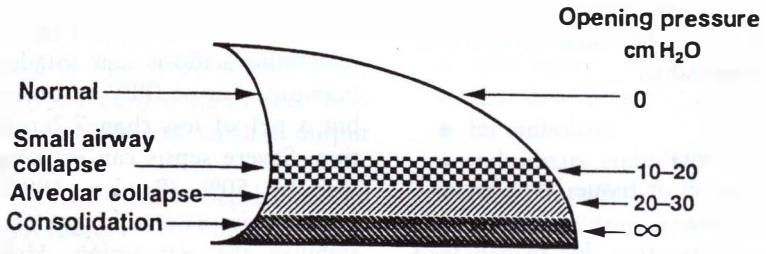


Figure 15.9 Diagram illustrating how the greater weight of dependent lung causes progressively greater opening pressures downwards. (From Gattinoni, L., quoted by Sykes, K. and Young, G. D. (1999) *Respiratory Support in Intensive Care*, BMJ Publishing, London.)

the lung, which has been intact during the florid oedema phase, is remodelled and weakened by the inflammatory process. Fibrosis sets in within 2 weeks (Ryan and Higgins, 1998). Non-homogenous loss of elasticity causes uneven distribution of expiratory time as alveoli empty at different speeds, contributing to intrinsic PEEP, overdistension of alveoli and a 12% incidence of barotrauma (Du *et al.*, 1997).

When oxygen delivery (DO_2) decreases to a critical level, oxygen extraction cannot increase to compensate, and oxygen consumption ($\dot{V}O_2$) drops linearly with DO_2 (Wong, 1998).

Clinical features

Following the provoking insult, there is a latent period of 1–3 days before signs become apparent. Respiratory distress develops over the next 24 hours as patients struggle to breathe through lungs that feel like a wet sponge. Both P_aO_2 and P_aCO_2 drop. Diagnosis is usually when virulent hypoxaemia develops and, in the spontaneously breathing patient, P_aCO_2 rises as the patient tires. Development of the syndrome is less obvious if IPPV is already in place.

X-ray signs lag behind clinical signs by 24–48 hours. Then diffuse bilateral 'snowstorm' infiltrates develop, with sparing of the costophrenic angles (Figure 15.10). After a week, a finer consolidation becomes apparent, corresponding to fibrosis. Recovery brings resolution, except in 20% of patients who manifest residual opacities, fibrosis and hyperinflation (Wiener, 1991). CT scanning shows the opacity of atelectasis in dependent regions if the patient's position has not been regularly changed, and sometimes barotrauma in non-dependent regions (Figure 15.11).

Breath sounds are surprisingly normal, with just a harsh edge to them. Pulmonary artery catheterization shows a high PAP, reflecting pulmonary hypertension. PAWP (p. 329) is normal because ARDS is not a condition of generalized overhydration, in contrast to cardiogenic pulmonary oedema, which causes a high PAWP. $S_{\bar{v}}O_2$ is usually reduced as a result of hypermetabolism, but may be increased if hypoxic cells are unable to extract oxygen.

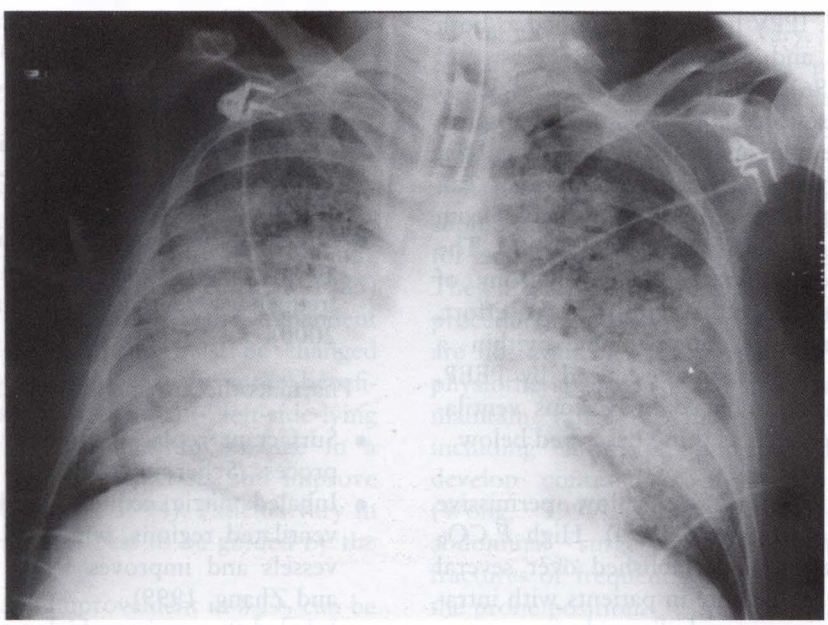


Figure 15.10 Image of lungs affected by ARDS.

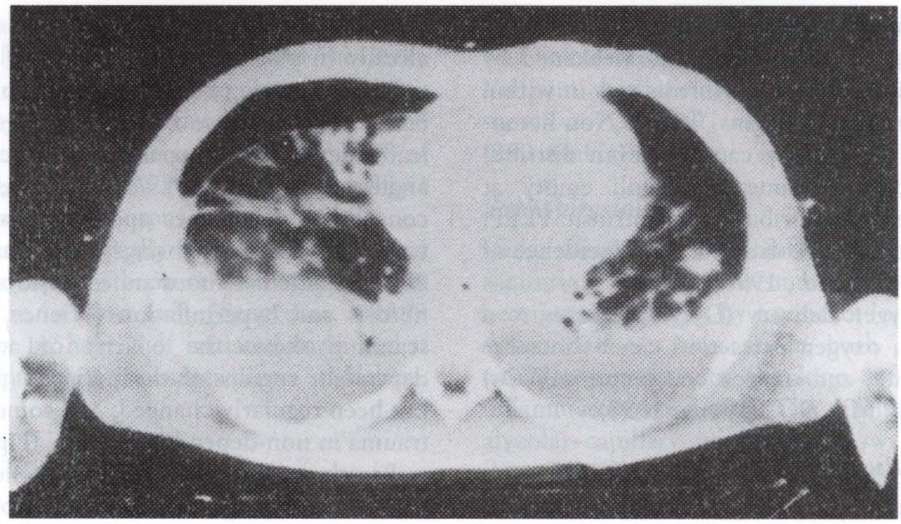


Figure 15.11 CT scan of a patient with ARDS, showing dense areas of atelectasis in dependent regions and barotrauma represented by a pneumothorax in the right anterior region.

Medical treatment

The cornerstone of management is meticulous supportive care. Non-invasive ventilation is helpful in the early stages (Rocker *et al.*, 1999), but intubation and mechanical ventilation are often necessary. IPPV has been described as both a good friend and a secret killer (Pelosi and Gattinoni, 1996). It reduces the work of breathing but squeezes the bulk of the inspiratory gas into healthy and fragile functioning lung tissue. This creates stretching forces that can cause structural damage indistinguishable from the disease process itself (MacIntyre, 1996). The risk is reduced by finely tuned synchrony of ventilator-assisted breaths with patient effort, and maintenance of lung volume within a narrow range. Atelectasis is reduced by PEEP, and overdistension reduced by various ventilatory manoeuvres such as those described below.

- Low tidal volumes can allow permissive hypercapnia (Hickling, 1994). High P_aCO_2 is well tolerated if established over several days but is inadvisable in patients with intracranial lesions or metabolic acidosis.
- Judiciously applied PEEP stabilizes alveoli, improves gas exchange, reduces intrinsic PEEP and distributes inspired air more evenly. Levels over 15 cmH_2O prevent progressive loss of lung compliance associated with low-volume ventilation (Cereda, 1996). However, if overdistension occurs and compliance worsens, it can damage rather than protect the lung. High PEEP is less helpful as fibrosis sets in.
- Pressure control ventilation limits peak pressure and may prevent over-distension of compliant areas of lung.
- Other options are inverse-ratio ventilation, airway pressure release ventilation and high-frequency ventilation (Campbell *et al.*, 2000).

Pharmacotherapy includes the following:

- Surfactant replacement early in the disease process (Schermuly, 2000).
- Inhaled nitric oxide, distributed to well-ventilated regions, which dilates pulmonary vessels and improves $\dot{V}_A/\dot{Q}$ match (Stewart and Zhang, 1999).
- Inhaled β_2-agonists which may improve lung compliance (Moriña, 1997).

- Steroids are sometimes given in the later stages but tend to encourage infection in critically ill patients (Bass *et al.*, 1997).
- Supplemental oxygen is limited to 60% if possible, to prevent oxygen toxicity. Hypoxaemia is more responsive to PEEP than high F_IO_2 levels.

Extracorporeal gas exchange, a last resort, allows reduced minute volume and lower F_IO_2 to be delivered but causes bleeding in 75% of patients (Anderson, 1994).

Physiotherapy

The physiotherapist may not be informed that ARDS is developing because it is a syndrome rather than a disease and the patient already has a diagnosis. The condition is suspected if a patient with relevant predisposing factors shows tachypnoea and severe hypoxaemia, or if a ventilated patient develops high airway pressures or the ventilator is changed from volume control to pressure control.

Like the medical management, physiotherapy aims to maximize DO_2 while causing the least harm. Gratuitous increase in stress and energy expenditure must be avoided. The main respiratory problem is reduced lung volume. Secretions are usually of little note.

Positioning

Positioning has a marked influence on gas exchange because of the unevenly damaged lungs (Tobin, 1994). Side-lying reduces lung densities in the uppermost lung (Brismar, 1985) but reinforces compression in the dependent lung (Du *et al.*, 1997), and must be changed regularly. Right-side-lying may be more beneficial for cardiac output than left-side-lying (Wong, 1998). Regular position change in a kinetic bed can reduce atelectasis and improve gas exchange (Hormann, 1994). Patients vary in their response and it is best to be guided by the monitors.

A more dramatic improvement in S_aO_2 can be found in two-thirds of patients by gently turning them prone (Lim *et al.*, 1999). There are more alveoli posteriorly, because of the configuration of the chest and position of the heart, and in prone these alveoli are uppermost and recruitable. Ventilation becomes more even because the heart is supported by the sternum and there is less parenchymal distortion (Yang, 1991). Perfusion is also more uniform in prone (Marini, 1999). $\dot{V}_A/\dot{Q}$ is better matched and oxygenation improved. The following claims have been reported after proning patients with ARDS:

- ↑ lung volume by 57% (Rialp *et al.*, 1997)
- ↓ need for PEEP (Lim *et al.*, 1999)
- ↑ P_aO_2 by between 2.7 kPa (20 mmHg) and 6.2 kPa (47 mmHg)
- ↑ P_aO_2:F_IO_2 ratio by between 6.7 kPa (50 mmHg) and 13.3 kPa (100 mmHg)
- ↑ normal $\dot{V}_A/\dot{Q}$ units by 12%
- ↓ shunt by 11% (Wong, 1999)
- ↓ barotrauma (Du *et al.*, 1997)
- ↑ drainage of secretions (Kesecioglu, 1997)
- ↓ length of ICU stay (Gosheron, 1998).

Oxygenation is greater if pressure on the abdomen is minimized by using an air-fluidized bed or supporting the chest and pelvis with pillows. Improvement is usually apparent within 15 minutes but responses vary. If there is no benefit within an hour, the patient is best returned to the previous position (Pilbeam, 1998, p. 159). A positive response is most likely before fibrosis begins developing.

Benefits may be partially lost on returning to supine and some patients are best left in prone for extended periods on a skin-friendly mattress. They are returned supine if required for various procedures, and at night if sufficient skilled staff are not available. During periods in supine, the physiotherapist can make a full assessment and maintain range of movement to all joints, including elbow, hip and shoulder which can develop contractures if prone is prolonged (Wong, 1999). Practical obstacles such as abdominal surgery, spinal injury, unstable fractures or frequent seizures can contraindicate the prone position.

Three or four staff are required for the turn, including an intensivist or experienced nurse at

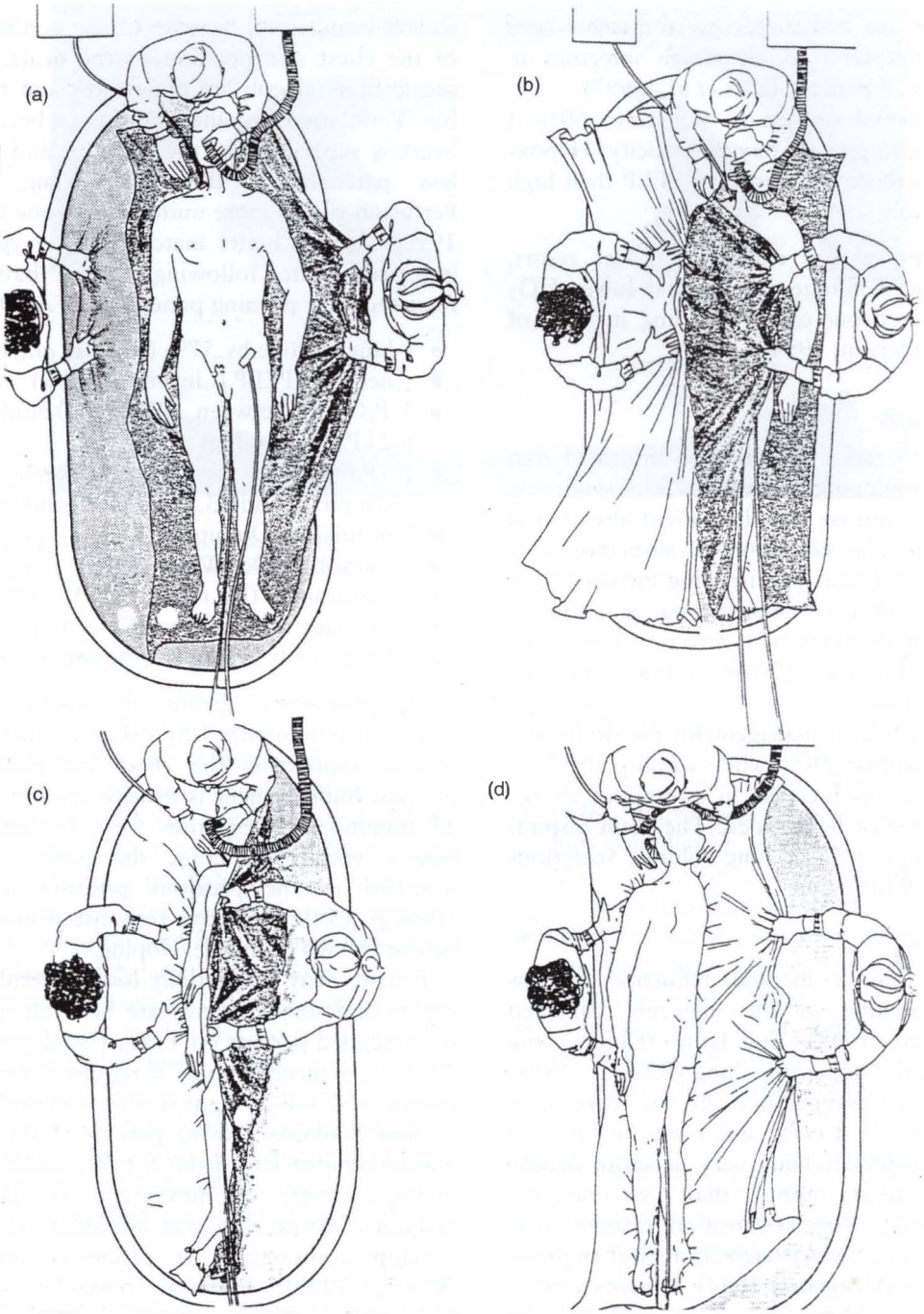


Figure 15.12 Turning a patient prone. (**a**) The patient is pulled to the side of the bed on the old sheet over a glide sheet. (**b**) A clean sheet is laid under the patient. (**c**) The patient is rolled prone over his/her neutrally-positioned arm. (**d**) The patient is pulled to the middle of the bed on the clean sheet over the glide sheet. (From Kesecioglu, J. (1997) Prone position in therapy-refractory hypoxaemia. *Curr. Opin. Anaesthesia*, **10**, 92–100)

the head of the bed to protect the airway and neck lines (Figure 15.12). A suggested procedure is described below.

1. Inform patient, with reassurance that s/he will be safe.
2. Secure eye protection.
3. Disconnect and plug lines as feasible, redirect others in axis of body. Chest drains will need long tubing.
4. Check that team members understand the vulnerability of the shoulder joint.
5. Slide the patient to the edge using a glide sheet.
6. Place pillows at pelvis and chest level, but close enough to avoid lumbar lordosis.
7. Place patient's hand under the hip, with elbow straight and shoulder in neutral.
8. Roll patient over this arm and into prone, so that pelvis and chest rest on pillows and abdomen is free of pressure.
9. Reconnect lines.
10. Ensure that no joint is at end-range, especially the lumbar spine and neck. The neck must be slightly flexed and only half rotated. Slight neck flexion can be facilitated by overhanging the head at the end of the bed, supported on a cushioned table. A pillow or horseshoe headrest allows the tracheal tube to be unrestricted but secure.
11. Check that the ulnar nerve is not overstretched and shoulder joint remains near-neutral (Figure 15.13). Either the arm to which the head is turned can be semi-flexed, or both elbows extended and shoulders internally rotated.
12. Ensure that women's breasts and men's genitals are not compressed.
13. Place pillow under shins to prevent peroneal nerve stretch, positioning the pillow to avoid knee and toe pressure from mattress.
14. Tilt the bed head-up to about 20° in order to prevent facial oedema and potential eye damage.
15. Suction airway because the turn often mobilizes secretions.

Head and arm positions are alternated 2-hourly. Volume of feed may need to be reduced in case of regurgitation. Pressure areas now

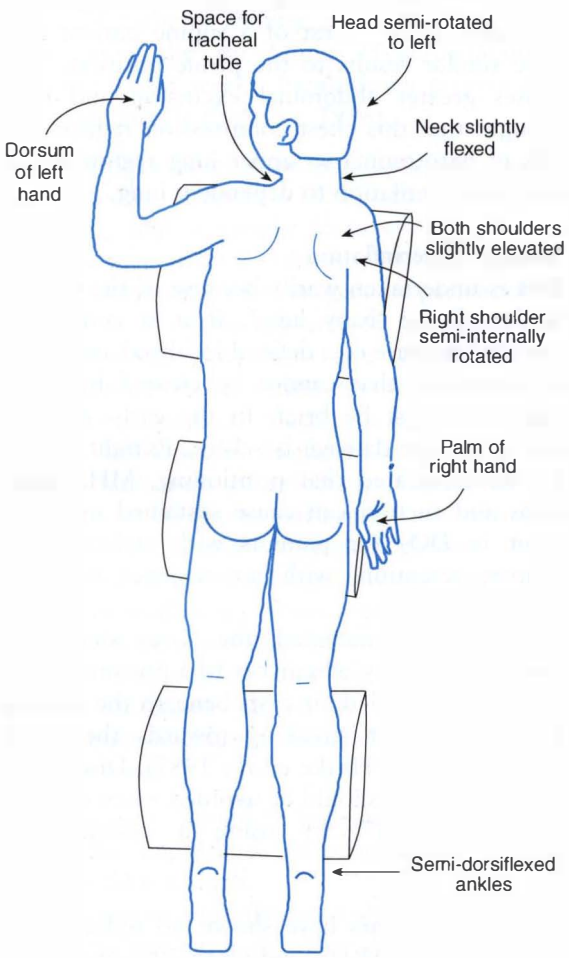


Figure 15.13 Suggested position for a proned patient. Pillows are arranged around the tracheal tube to maintain a clear airway and prevent obstruction. Pillows support (a) upper chest to ensure support for head of humerus and slight protraction of shoulder girdle, (b) pelvis to prevent extension of lumbar spine and (c) shins to prevent pronation. The head of the left humerus may need extra support, e.g. by a flattened rolled bandage or corner of a pillow. Right dorsum and left palm are rested against the bed. To change position, the upper part of the patient is raised, with due consideration for the patient's joints, airway and lines, and with manual handling technique agreed in advance. The head is then semi-turned to the opposite side and arms re-arranged.

include ears, cheeks, knees and toes. Potential cardiac arrest must be planned for and a protocol prepared for rapid return to supine (Sun, 1992).

Some clinicians find that placing 5 kg sandbags on the chest of a supine patient may have similar results to the prone position. This causes greater abdominal excursion and it is thought that this chest compression reduces the risk of barotrauma to upper lung regions while directing ventilation to dependent lung.

Manual hyperinflation

MH is undertaken warily because of the vulnerability of the 'baby lung'. If it is considered essential because of a defined localized atelectasis or secretions that cannot be cleared by other means, it must be brief. In the early stage of disease before damage is severe, Enright (1992) has demonstrated that positioning, MH, vibrations and suction can cause sustained improvement in DO_2 for patients with atelectasis or sputum retention, with no evidence of barotrauma.

If MH is anticipated, the X-ray should be examined for any precursors to a pneumothorax such as thin-walled air cysts beneath the visceral pleura or linear streaking towards the hilum (Albelda, 1983; Haake *et al.*, 1987). Disconnection for suction should be avoided when possible (Schwartz, 1987) by using a closed-circuit catheter.

The past 20 years have shown no reduction in mortality from ARDS, and up to 70% of patients still perish, half from sepsis and a tenth from respiratory failure (Wong, 1998). People with trauma-induced ARDS are most likely to survive, while those with an infective aetiology do less well (Wyncoll and Evans, 1999). Survivors show a remarkable capacity to regenerate lung tissue and many become symptom-free over time, but it is recommended that rehabilitation is continued because 50% experience respiratory symptoms, sometimes including severe breathlessness (Smith and Sinclair, 1996) and depression (Bowton, 1999). Post-traumatic stress has been found in 27% of survivors due to distressing experiences in the ICU (Schelling, 1998). This is a reminder to keep stress reduction at the centre of ARDS management.

POISONING AND PARASUICIDE

Complications of poisoning include arrhythmias due to the toxin or metabolic upset, fluid depletion due to vomiting or diarrhoea, and respiratory compromise due to ventilatory depression, upper airways obstruction or pulmonary oedema. If gastric lavage is attempted, it can cause aspiration, distress and laryngeal spasm, and it may increase drug absorption by forcing gastric contents into the small bowel (Valladares, 1996).

Deaths from poisoning remain steady at 4000 a year in the UK (Jones and Volans, 1999). Some poisoning is accidental but most is self-inflicted. Health staff have shown negative attitudes to parasuicide patients (Guylay, 1989), including judgements about attention-seeking. However, these patients are often at the extremes of depression or desperation and over 90% have diagnosable psychiatric illness (Urbaitis, 1999). The professional approach is to withhold personal judgement and care for the patient in such a way that s/he believes life to be worth living after all. This may help reduce the 1% of patients who will go on to kill themselves within a year (Kapur *et al.*, 1998).

Successful suicides are 'a permanent solution to a temporary problem' (Guylay, 1989) and relatives bereaved by suicide find recovery more difficult than from non-suicide bereavements. The care they receive in the first hours can have a profound impact on their grief (Odell, 1997).

SMOKE INHALATION

Smoke inhalation is the primary cause of fire-related deaths (Flynn, 1999). The addition of smoke inhalation to a burn increases mortality by 20%, or 60% if pneumonia develops (Papini, 1999). The heat from inhaled smoke is filtered by the upper airways at the expense of bronchos-

pasm, mucosal swelling, pulmonary oedema, paralysis of cilia and ulceration. Toxins, steam and crack cocaine can overwhelm the filtering properties of the airways and penetrate to alveoli, where they destroy surfactant and burn lung tissue (Haponik, 1992). Upper airway obstruction is the most treatable respiratory complication, but if intubation is delayed, asphyxia may occur from face and neck oedema.

Secondary damage arises from the inflammatory response to injured tissue which increases vascular permeability. This exacerbates pulmonary oedema, which can halve lung compliance and quadruple respiratory resistance (Papini, 1999). Pulmonary oedema is difficult to control because of the fluid requirements of surface burns. Oxygen delivery is impaired by shock, inflammatory mediators and inhaled carbon monoxide. Carbon monoxide famously binds to haemoglobin 200 times more strongly than oxygen, and also shifts the oxygen dissociation curve to the left, which hinders loading of oxygen from the lungs and interferes with unloading of oxygen to the tissues. Infection is commonly transmitted to the denuded airways from the hospital environment, infected burns or endogenous sepsis. The stages of lung injury are:

1. bronchospasm (first 12 hours)
2. pulmonary oedema (6–12 hours post-burn)
3. bronchopneumonia (> 60 hours post-burn)

Wheeze and sooty sputum may not appear for 24 hours, and X-ray signs of pulmonary oedema are not apparent for some days. Severe injury is indicated by dyspnoea and cyanosis. Later developments may include restricted expansion due to a tight armour of scarring around the chest, and various effects of epithelial damage such as long-term hyperreactivity, tracheal stenosis or bronchiectasis (Tasaka *et al.*, 1995).

Medical treatment is based on:

- pain management (Wu *et al.*, 1999)
- judicious fluid administration, both crystalloid to resuscitate the interstitial space and colloid for the intravascular space
- humidified oxygen, at 100% if carbon monoxide has been inhaled
- CPAP if the face is not burned, or IPPV with PEEP, followed by extubation over a fibreoptic bronchoscope in case of oedema
- supplementary feeding, preferably enteral to preserve the gut lining. Hypermetabolism can last for weeks, break down protein and waste muscle (Nguyen, 1996).

Other options are hyperbaric oxygen, inhaled nitric oxide (Papini, 1999) to assist gas exchange, and a simple form of hypnosis to augment pain relief (Ohrbach, 1998). Pain relief is a priority, especially as pain experienced in hospital is a stronger predictor of adjustment after discharge than burn size (Ptacek *et al.*, 1995). Prophylactic antibiotics are not recommended but bacterial infection is likely after day 2 or 3, and cultures should be obtained at the earliest indication of infection (Papini, 1999). Oximetry is falsely normal because the oximeter cannot distinguish oxyhaemoglobin from carboxyhaemoglobin. Large mucus casts may require bronchoscopy or occasionally lavage.

Respiratory physiotherapy is aimed at maintaining lung volume and clearing thick and prolific secretions caused by airway damage. Lavish humidification is needed. Precautions are the following.

- Treatment should be little and often because of the importance of prophylaxis and the inevitable fatigue.
- Percussion and vibrations should be avoided over chest burns, whether dressed or not. If manual techniques are essential, a vibrator is reasonably comfortable.
- If suction is necessary, it should be gentle, minimal and scrupulously aseptic to prevent further mucosal damage.
- Patients need extra attention to communication if facial oedema affects vision or speech.
- If there is oedema around the head or neck, postural drainage is contraindicated and patients are often nursed upright.
- If hoarseness, voice change or stridor develops, nasopharyngeal suction is contrain-

dicated and the patient's condition should be reported because intubation will be required.

Two-hourly exercises are required for burned limbs, especially the hands (Keilty, 1993), using Entonox or other analgesia. Provision of a 'Burn Intensive Care Gym' provides the opportunity for patients to improve their functional status and take responsibility for self-management (Gripp *et al.*, 1995).

NEAR-DROWNING

Near-drowning is defined as submersion followed by survival for 24 hours, then deterioration. Death from pulmonary complications can occur, especially with 'wet drowning', which leads to pulmonary oedema, inactivation of surfactant, bronchospasm, hypoxaemia and cerebral oedema. If water is swallowed, there is a high incidence of vomiting, sometimes followed by further aspiration. Frequent physiotherapy to clear the airways may be needed for at least 48 hours in order to prevent atelectasis.

'Dry drowning' accounts for 10% of near-drowning admissions and is caused by laryngospasm in a panicking victim, leading to apnoea and hypoxaemia. Fluid is not aspirated and the airways rarely need clearance by physiotherapy.

Hypothermia, defined as core temperature below 35°C, commonly occurs with near-drowning. Resuscitation attempts should be prolonged and nobody considered dead until they are warm and dead. Patients are given warmed humidified oxygen, warmed IV fluids, warm blankets (not space blankets, which simply prevent heat loss) and sometimes cardiopulmonary bypass.

CASE STUDY: MR CA

Identify each day's problems and plans for this 25-year old male hit by scaffolding (Figure 15.14), which fractured his 6th and 7th ribs on the right (imperceptible on X-ray).

Day 1

Subjective:
pain and breathlessness.

Objective:
pale, sweaty, rapid breathing, flail segment on R, other respiratory observations normal.

Questions

1. Problems?
2. Plan?

Day 2

X-ray shows white-out on R.
pH 7.48, P_aO_2 6.7 kPa, P_aCO_2 3.9 kPa, HCO_3^- 24.
Intubated and ventilated → blood gases normalized.

Questions

1. Percussion note?
2. Breath sounds?
3. Explain P_aCO_2.
4. Explain white-out on R.
5. Explain P_aO_2.
6. Why was the patient mechanically ventilated when he still had one fully functioning young lung?
7. Problems?
8. Plan?

Day 3

Epidural in place.
Weaned and extubated.

Subjective:
Fatigue.

Objective:
Crackles on auscultation.

Questions

1. Problems?
2. Plan?

Day 4

Questions

1. Consolidation? atelectasis?

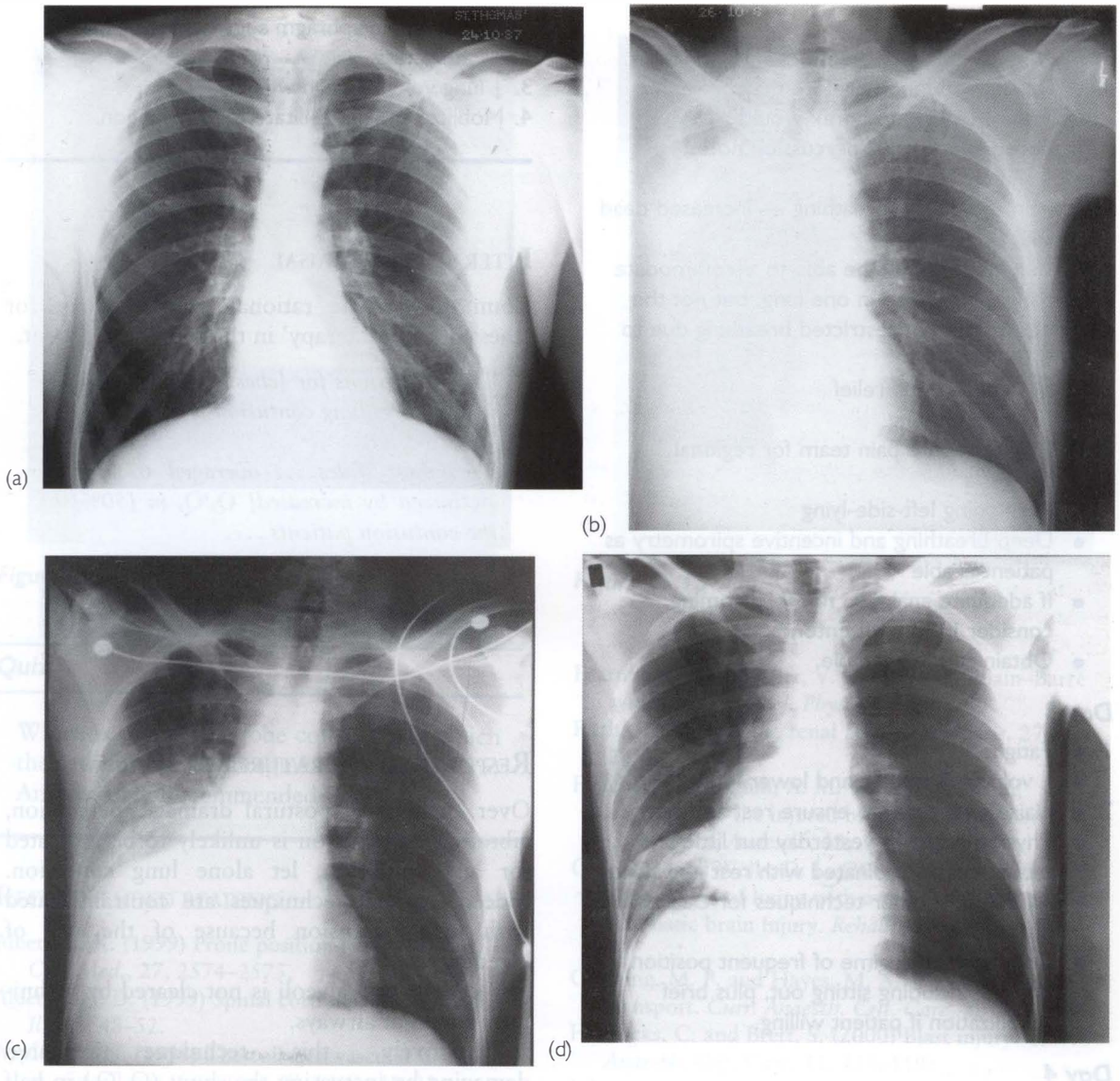


Figure 15.14 X-rays of Mr CA. (a) Day 1. (b) Day 2. (c) Day 3. (d) Day 4.

2. Lobe(s) affected?
3. Problems?
4. Plan?

RESPONSE TO CASE STUDY

Day 1

1. Pain → potential for respiratory complications
2. • Liaise re. analgesia.
   • Position for optimum pain relief and respiratory function.
   • Regular incentive spirometry.
   • Assess frequently until analgesia adequate, then initiate more active measures, e.g. mobilization.

Day 2

1. Percussion note dull on R.

2. Breath sounds ↓ on R.
3. P_aCO_2 ↓ because of breathlessness.
4. White-out sudden, therefore may be due to aspiration. (A haemothorax would have produced a stony dull percussion note.)
5. • ↑ shunt on R.
   • Pain → shallow breathing → increased dead space.
6. A young man should be able to accommodate loss of gas exchange in one lung, but not the added problem of restricted breathing due to pain.
7. • Inadequate pain relief
   • ↓ volume R lung
8. • Refer to acute pain team for regional analgesia
   • Positioning left-side-lying
   • Deep breathing and incentive spirometry as patient is able
   • If adequate analgesia not forthcoming, consider IPPB with Entonox
   • Obtain sputum sample.

Day 3

1. • Fatigue
   • ↓ volume R middle and lower lobes.
2. • Liaise with team to ensure rest and sleep
   • Physiotherapy as yesterday but little and often, and co-ordinated with rest and sleep.
   • ACB/AD + other techniques for clearance of secretions
   • Daily written regime of frequent position change, including sitting out, plus brief mobilization if patient willing.

Day 4

1. and 2.
   • Middle lobe consolidation.
   • ↑ R hemidiaphragm suggests lower lobe atelectasis.
3. ↓ lung volume as above.
4. Mobilize and rehabilitate to full function.

LITERATURE APPRAISAL

Comment on the rationale and evidence for 'chest physical therapy' in the following patient.

> *The indications for [chest physical therapy] included ... lung contusion ....*
>
> *Treatment times ... averaged 67 min ... [followed by increased] Q_s/Q_t in [50%] of the contusion patients ....*
>
> *The long-term clinical effect of these changes is unknown.*
>
> *Crit. Care Med.* 1985; 13: 483–486

RESPONSE TO LITERATURE APPRAISAL

Over an hour of postural drainage, percussion, vibration and suction is unlikely to be indicated for any condition, let alone lung contusion. Indeed, manual techniques are contraindicated with lung contusion because of the risk of bleeding.

Bleeding into alveoli is not cleared by techniques aimed at airways.

Objectively, this technique appeared damaging by increasing the shunt (Q_s/Q_t) in half the patients with contusion.

Subjectively, one can only guess.

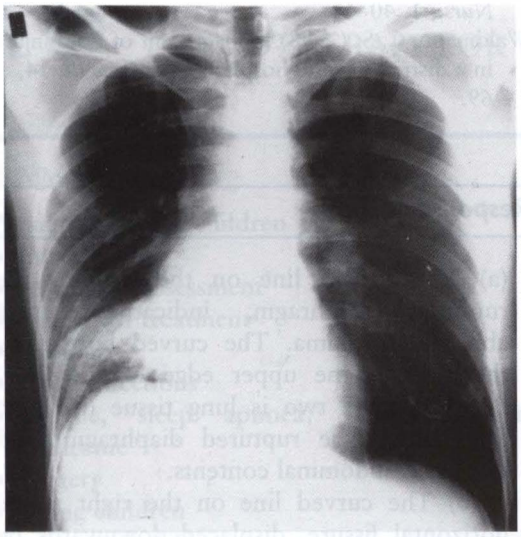


Figure 15.15 (a)

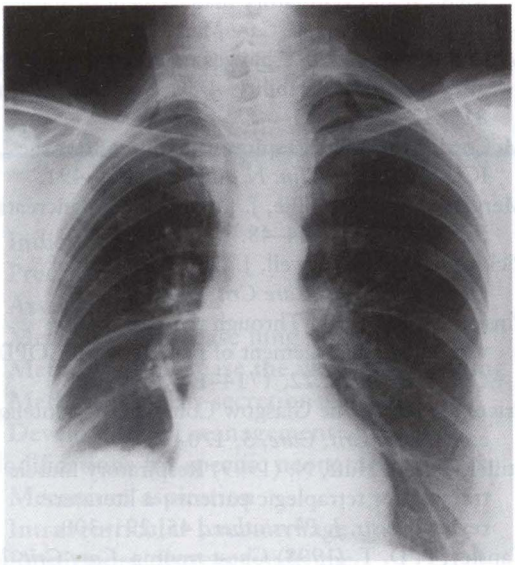


Figure 15.15 (b)

Quiz

Which is the middle lobe collapse and which the ruptured diaphragm? (Figure 15.15) Answer after Recommended Reading.

RECOMMENDED READING

Albert, R. K. (1999) Prone position in ARDS. *Crit. Care Med.*, **27**, 2574–2575.

Alderson, J. D. (1999) Spinal cord injuries. *Care Crit. Ill*, **15**, 48–52.

Appleyard, N. (1998) The collagen vascular diseases in critical care. *Care Crit. Ill*, **14**(1), 29–33.

Bernal, W. and Wendon, J. (2000) Acute liver failure. *Curr. Opin. Anaesth.*, **12**, 113–118.

Bulger, E. M. (2000) Rib fractures in the elderly. *J. Trauma*, **48**, 1040–1043.

Cubukcu, A., Paksoy, M. and Gönüllü, N. N. (2000) Traumatic rupture of the diaphragm. *Int. J. Clin. Pract.*, **54**, 19–21.

Dhond, G. R. and Dob, D. P. (2000) Critical care of the obstetric patient. *Curr. Anaesth. Crit. Care.*, **11**, 86–91.

Edwards, J. D. (1997) Oxygen transport in the critically ill. *Br. J. Intens. Care*, 7, 23–29.

Fearnhead, L. and Fritz, V. U. (1996) Guillain–Barré syndrome. *S. Afr. J. Physiol.*, **52**, 85–87.

Firth, J. (1999) Acute renal failure. *Medicine*, **27**(5), 24–31.

Fletcher, S. and Lam, A. M. (2000) Improving outcome for the injured brain and spinal cord. *Curr. Opin. Anaesth.*, **13**, 155–160.

Gill, D. J. and Wells, D. L. (2000) Forever different: experiences of living with a sibling who has a traumatic brain injury. *Rehabil. Nurs.*, **25**, 48–52.

Gunning, M. P. and Hayes, M. A. (1999) Oxygen transport. *Curr. Anaesth. Crit. Care*, **10**, 319–324.

Horrocks, C. and Brett, S. (2000) Blast injury. *Curr. Anaesth. Crit. Care*, **11**, 113–119.

Johnson, J. and Silverberg, R. (1995) Serial casting of the lower extremity to correct contractures during the acute phase of burn care. *Phys. Ther.*, **75**, 262–266.

Keilty, S. E. J. (1993) Inhalation burn injured patients and physiotherapy management. *Physiotherapy*, **79**, 87–90.

Kennedy, D. D., Fletcher, N. and Hinds, C. (2000) Neuromuscular dysfunction in critical illness. *Curr. Opin. Anaesth.*, **13**, 93–98.

Kulkarni, V. and Webster, N. (1996) Management of sepsis. *Care Crit. Ill*, **12**, 122–127.

Levy, B. D., Kitch, B. and Fanta, C. H. (1998)

Medical and ventilatory management of status asthmaticus. *Intens. Care Med.*, **24**, 105–117.
McColl, M. A. (2000) Changes in spiritual beliefs after traumatic disability. *Arch. Phys. Med. Rehab.*, **81**, 817–823.
McCord, M. (1999) Respiratory failure: after the ICU. *Crit. Care Clin. N. Am.*, **11**, 481–491.
Mergener, K. and Baillie, J. (1998) Acute pancreatitis. *Br. Med. J.*, **316**, 44–48.
Pickford, A. and Criswell, J. (2000) Review of gunshot injuries. *Care Crit. Ill*, **16**, 28–32.
Pinsky, M. R. (1994) Through the past darkly: ventilatory management of patients with COPD. *Crit. Care Med.*, **22**, 1714–1717.
Price, P. (2000) The Glasgow Coma Scale in intensive care. *Nurs. Crit. Care*, **5**, 170–173.
Stiller, K. and Huff, N. (1999) Respiratory muscle training for tetraplegic patients: a literature review. *Austr. J. Physiother.*, **45**, 291–300.
Tansley, P. D. T. (1998) Chest trauma. *Care Crit. Ill*, **14**, 260–265.
Vandevoort, M. (1999) Nutritional protocol after acute thermal injury. *Acta. Chir. Belg.*, 99, 9–16.
Wake, D. (1995) Near drowning. *Intens. Crit. Care Nurs.*, 1, 40–43.
Waldmann, C. S. (1998) Management of head injury in a district general hospital. *Care Crit. Ill*, **14**, 65–69.

Response to quiz

(a) The curved line on the right is the ruptured diaphragm, indicating severe abdominal trauma. The curved border on the right is the upper edge of the liver. Between these two is lung tissue (in front and behind the ruptured diaphragm) and displaced abdominal contents.

(b) The curved line on the right is the horizontal fissure, displaced downwards by the collapsed middle lobe. The increased density reflects loss of middle lobe volume.

16 Physiotherapy for children and infants

Summary

Physiotherapy for children
- Introduction
- Aspects of assessment
- Aspects of treatment
- Asthma
- Chest infections
- Fatigue, sleep apnoea, hyperventilation syndrome
- Surgery
- Dying children

The neonatal ICU
- Introduction
- Care of the parents
- Management of pain and stress
- Temperature and fluid regulation
- Oxygen therapy
- Feeding
- Humidification
- Mechanical ventilation
- Neonatal support systems

Physiotherapy for neonates
- Indications
- Precautions
- Assessment
- Methods to increase lung volume
- Methods to decrease the work of breathing
- Methods to clear secretions
- Developmental management

Modifications for specific neonatal disorders
- Meconium aspiration
- Intraventricular haemorrhage
- Respiratory distress syndrome
- Chronic lung disease of prematurity

Emergencies in the neonatal unit
- Sudden hypoxaemia
- Apnoeic attacks
- Pneumothorax
- Cardiorespiratory arrest

Mini case study
Literature appraisal
Recommended reading

Physiotherapy for children

Introduction

Adult patients can say to themselves: 'I understand that I am not in hospital for the rest of my life, that my family will visit, that the nasty things they are doing to me are for my own good'. Young children do not have these resources of reasoning and may be overwhelmed by bewilderment, uncertainty about the behaviour expected of them and sometimes feelings that they are abandoned or being punished. Despite progress in humanizing children's experience in hospital, long-term emotional disturbance can still occur. Children need to be listened to, believed and given some control over what is done to them. Teenagers in particular need autonomy because they are extra-sensitive to peer pressure and often feel they have outgrown the paternalistic environment of paediatric units.

Children appreciate having the same physiotherapist throughout their stay. Those over 3 years old should be included when their treatment in discussed in their presence. Children need their own toys and belongings, and all but the sickest are best dressed in their day clothes. Hospitalized children, like adults, commonly adopt the sick role and may show an exaggeration of the behaviour patterns that they normally use to cope with stress.

Parents require confidence in their own competence, and acknowledgement that they are

the experts on their children, especially as they may be more effective than health workers in the identification of their child's problems (Roberts, 1996). Siblings require involvement because they may feel a variety of responses, including jealousy, anxiety, isolation and guilt. And if their brother or sister is disabled, they are likely to suffer bullying at school (Miller, 1996).

Aspects of assessment

Parents should be welcomed during assessment and treatment. If this causes the child to express anxiety more noisily than when unaccompanied, this is healthier than withdrawal. For any anxious patient, but particularly the young, it is advisable to avoid touch until a modicum of trust has been established. If the child's favourite toy, TV programme, food or game is listed above the bed, these can be used to engage the child's interest. Assessment can be done on the parent's lap, with a description of what is being examined and why. If an intravenous needle is present, it can inhibit children from moving and they should be reassured that it will be supported throughout. If a nil-by-mouth sign is present, the child may be distressed and not understand why s/he is so thirsty.

Children have a high respiratory rate (RR) because of the extra metabolism needed for growth and the relatively large metabolically active brain and viscera. Vital signs are shown in Table 16.1.

In young children and babies, laboured inspiration is shown by chest recession (retraction) because of the compliant chest wall (Figure 16.1). Laboured expiration is shown by grunting, which acts as a form of CPAP to splint open the narrow airways. Other signs of respiratory distress are:

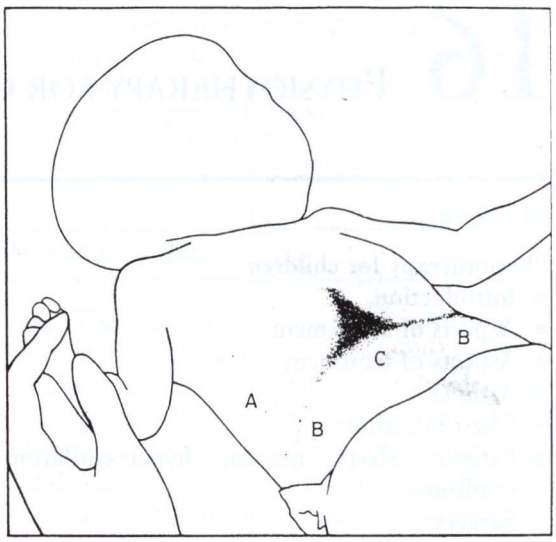


Figure 16.1 Retractions indicating respiratory distress. A = intercostal; B = subcostal. C = substernal. (From Wilkins, R. L., Krider, S. J. and Sheldon, R. L. (1995) *Clinical Assessment in Respiratory Care*, Mosby, Toronto, with permission.)

- ↑ RR
- asynchronous breathing, shown by a seesaw motion between chest wall and abdomen
- nasal flaring
- apnoea associated with bradycardia or pallor.

Before auscultation, children can be given the opportunity to see and feel the stethoscope, and use it to listen to themselves or a doll. The diaphragm or bell should be warmed before use.

Breathlessness can hamper communication, interfere with sleep and affect eating or

Table 16.1 Age-related vital signs (Prasad and Hussey, 1995)

	Newborn	1–3 years	3–7 years	Over 7 years
RR	40–60	20–30	20–30	15–20
P_aO_2	60–90	80–100	80–100	80–100
HR	100–200	100–180	70–150	80–100
BP	60/30–90/60	75/45–130/90	90/50–140/80	90/50–140/80

drinking. Alert children in respiratory distress assume a position that promotes airway patency and they should be allowed to maintain this position. Deterioration in gas exchange may be indicated by pallor, sweating, restlessness, agitation, glazed eyes and, in ventilated children, fighting the ventilator. Hypoxaemia must be excluded before sedating an unsettled child. Cyanosis is a severe sign. Atelectasis of the left lower lobe may be missed on a portable X-ray film because it is hidden by the heart.

Aspects of treatment

Clear, honest and simple explanations should be given to the child before treatment, including demonstrations on a teddy, descriptions of what the treatment will feel like, how long it will last and reassurance that it can be stopped temporarily by request at any time. Resistance to treatment can often be overcome by giving the child a choice, e.g. whether to keep the TV on or off, have incentive spirometry or a walk outside, have curtains open or closed. Requests to stop treatment must be respected, and if further treatment is refused despite cajoling, distraction, joking and enlisting the help of parents or a play therapist, serious thought should be given to whether the benefits of continuing treatment outweigh the effects of enforcement.

Babies can be given rattles and toys to watch during treatment. For children, hand puppets can be used to give instructions, story-telling can be used so that the child looks forward to the next instalment in the next physiotherapy session.

In the UK, a child's consent to treatment is required if s/he is of 'sufficient understanding to make an informed decision' (Children Act, 1989). Children of school age are presumed competent for this purpose (Rylance, 1996) unless there is a specific reason otherwise. Children should not simply be deemed to be competent if they agree, and incompetent if they disagree. Hall (1994) claims that health workers now have a legal obligation to believe their younger patients. After age 16, full adult rights apply and parental consent is not required. Some modifications of the techniques described in Chapters 6–8 are discussed below. Treatment should not be straight after a meal.

Methods to increase lung volume

Young children need particular attention to maintenance of lung volume because lack of elastic tissue in immature lungs means that they share with elderly people a tendency for airway closure at low lung volumes.

Two-year-olds can do breathing exercises if taught imaginatively. The use of paper mobiles, bubble-blowing, blowing through a straw or blowing a tissue will utilize the deep breath that is taken before blowing out. Paediatric incentive spirometers are often popular. Abdominal breathing can be taught by placing a favourite toy on the abdomen, 'like a boat on the sea'. Crying upsets the flow rate without increasing volume (Figure 16.2) and should be avoided, particularly in children with stridor.

When positioning for gas exchange, the distribution of ventilation is opposite to the adult pattern. The compliant chest wall and lack of connective tissue support for the small airways inhibits flow to the more compressed dependent regions, directing ventilation preferentially to upper regions. Airway closure occurs above resting lung volume until the age of 6 or 7 (James, 1991) and poor ventilation to lower regions may predominate for the first 10 years of life (Davies *et al.*, 1990). In the presence of unilateral lung pathology, gas exchange varies and oximetry is the best guide.

For spontaneously breathing children, CPAP (p. 156) is used if adequate oxygenation cannot be maintained with oxygen therapy. CPAP is suited to children to compensate for their floppy chest walls. Administration can be by face mask, which is not easy to seal, or a short soft nasal prong (Morley, 1999). A starting pressure of 5 cmH_2O is used, and gradually increased until grunting stops or oxygenation is optimum. Pressures above 10 cmH_2O bring a risk of gastric distension or possible pneumothorax. For intubated children, CPAP in the form of PEEP is always required because the tracheal tube prevents grunting.

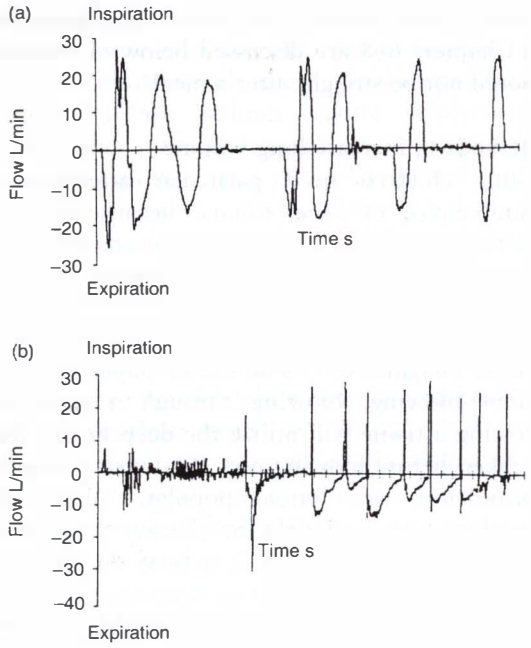


Figure 16.2 Breathing pattern of (a) a 2-year-old child at rest and (b) a 20-month old child while crying (left half) and sobbing (right half). (From Nikander, K. (1997) Adaptive aerosol delivery. *Eur. Resp. Rev.*, **7**, 385–387)

Methods to clear secretions

Postural drainage can be enjoyable over a bean bag or on the helper's lap in a rocking chair. For infants, who spend much time supine, the sitting position is included to drain the apical segments of the upper lobes, with particular attention to the right upper lobe.

Many young children have undiagnosed gastro-oesophageal reflux, which usually clears spontaneously within 12 months (Nelson, 1998). For children in whom this is suspected, prone and left-side-lying give some protection (Ewer *et al.*, 1999) because the greater curvature of the stomach provides an enhanced reservoir capacity. The head should generally be raised to 30° and the head-down tilt is usually contraindicated (Button *et al.*, 1997). However, children vary in the position at which reflux occurs, and symptoms should be checked. Feeds are given little and often, and physiotherapy must be avoided after eating.

Percussion is sometimes more soothing than vibrations. Huffing can be encouraged by blowing cotton wool or using the story of the big bad wolf who 'huffed and puffed and blew the house down'. From age 4–5 years children can do aspects of ACB and AD (p. 194), but are unable to put it together consistently until 6–7 years and may not actually do it when on their own.

Wheelbarrow races and games incorporate exercise and position change. Wind instruments use the principles of PEP (p. 198). Families can be encouraged to use swimming and biking as treats.

Coughing can be encouraged by laughter and rewarded by earning a star on a cough score sheet. Coughing with a protruded tongue helps to prevent swallowing. In the first year of life, coughing can sometimes be stimulated at the neck by finger pressure sideways during exhalation against the trachea below the thyroid cartilage. This must be gentle to avoid bradycardia.

Nasopharyngeal suction is unnecessary if the child is coughing effectively, even if secretions are swallowed. When suction is necessary, the procedure on page 205 is followed, using water-soluble jelly or the child's saliva as lubricant, and catheter sizes as in Table 16.2.

It is advisable to advance the catheter only until a cough is stimulated, or, for intubated patients, not more than 1 cm beyond the end of the tracheal tube (Riston, 2000). Excessive suction pressure causes greater mucosal damage

Table 16.2 Suggested catheter sizes for the non-intubated child

Age	Catheter size (FG)
Neonate	5
6 months	6
1 year	8
2 years	10
6 years	12

(Czarnik *et al.*, 1991) and does not appear to remove more secretions (Howard, 1994). There is little research evidence of the pressure at which damage occurs, but Parker (1998, p. 338) recommends 10–20 kPa (75–150 mmHg). Mini-tracheotomy has been used for children from 12 years old who need repeated suction (Allen and Hart, 1988).

If a sputum specimen is required, children under the age of 4 can rarely expectorate. A cough swab may be successful, in which the child coughs and secretions are collected from the back of the throat by a swab and sent to microbiology in a sterile container. A specimen of nasopharyngeal aspirate may be requested in order to obtain epithelial cells for diagnostic purposes. This entails nasal suction to the post-nasal pathway, as judged by using a length of catheter that has been measured from nose to ear. Specimens are often unhelpful because of oral contamination, and even secretions from intubated children usually carry pathogenic bacteria from the larynx and trachea (Hjuler, 1995).

Asthma

Asthma is the most frequent cause of respiratory symptoms in childhood (Battistini *et al.*, 1993). Some 10% of children are affected (Deaves, 1993), twice as many as any other chronic illness, and morbidity and mortality are increasing worldwide (Fawcett, 1995). The disease is both under- and overdiagnosed. One study showed that children consulted a general practitioner on average 16 times before asthma was diagnosed (Levy and Bell, 1984). Another showed that self-limiting infections are often misdiagnosed as asthma and lead to unnecessary medication (Stein, 1999). A simple screening method is to take the peak flow before and after a 6-minute exercise test, a drop of more than 15% within 10 minutes indicating asthma (Jones and Bowen, 1994). Details of asthma are given in Chapter 3, with aspects related to children described below.

Rehabilitation programmes have shown positive results (Baan-Slootweg, 1997), and the need for education is underlined by the child's fear about the disease. Two-thirds of children outgrow their asthma (Sears, 1994), and it is thought to be more than coincidence that this is at a time when they outgrow their fears (Gillespie, 1989). Education is therefore the key, including the following suggestions:

- use of colourful diaries and stickers, available from asthma organizations or manufacturers (Appendix C)
- practice in using inhalers, preferably in front of a group to improve confidence at school, and, for children over 6 years, practice in monitoring peak flow
- hard, enjoyable, controlled physical activity because the majority of children with asthma are deconditioned and associate exercise with anxiety (Clark, 1999)
- healthy eating, especially fresh fruit (Forastiere *et al.*, 2000)
- prevention, including environmental and diet modification from infancy in the case of atopic families (Brewin, 1998).

Some couples need to begin prevention before birth. Children born to an atopic couple have a 45% chance of developing asthma, sensitization beginning from 22 weeks gestation (Brewin, 1998). Avoidance of smoking by pregnant mothers is essential (Young, 2000).

Chromones (p. 133) are effective in 70% of children, in which case steroids can often be avoided (Korhonen *et al.*, 1999). Theophylline is not advisable for children (Goodman, 1996), and β_2-agonists, including the slow-acting varieties, should be used intermittently and not regularly (Bisgaard, 2000). Inhalers need to cope with children's lack of co-ordination, short inspiratory time, reduced ability to breath-hold and low inspiratory flow rate. Infants inspire at a flow rate below 10 L/min (Wildhaber, 1996). The following are suitable:

- inhaler with spacer and mask: 0–2 years
- inhaler with spacer: 3 years upwards
- dry powder inhaler (which needs a higher inspiratory flow rate): 5 years upwards

- metered dose inhaler: 10 years upwards (Cogswell, 1994).

Large-volume spacers may need to be tipped, mask downwards, during inhalation to allow the valve to open. If the mask is frightening, the parent can support it on the child's face, or the cheek can first be stroked gently with the mask. In countries where spacers are not available, a sealed 500 mL plastic bottle is a substitute (Zar *et al.*, 1999). If nebulizers are used, they can be tried on a teddy first, and some children can use them when asleep. Comprehensive instructions and regular checks are needed to ensure reliable technique (Kamps, 2000). Absorption of aerosolized drugs is reduced with crying (Iles *et al.*, 1999).

Chest infections

Risk of infant infections is reduced by breast feeding (Wright, 1989) and increased by parental smoking (Couriel, 1994). Young children react more severely to respiratory infection than adults because of their narrow airways, but if segmental atelectasis occurs, it usually clears spontaneously (Simoes, (1999). Some infections are associated with a higher incidence of COPD in adult life (Shaheen *et al.*, 1994). Inappropriate use of antibiotics is particularly condemned in children because of the association with multiresistant infections (Arason, 1996). Urban children have on average 5–8 respiratory infections a year, each lasting about a week (Horton, 1996). The more severe infections are described below.

Croup and epiglottitis

Croup is an acute syndrome of upper airways obstruction usually caused by laryngotracheobronchitis. Humidification is commonly applied but it is unclear how a mist might reduce an inflammatory obstruction. Croup is usually benign and self-limiting. Severe disease accompanied by stridor requires hospitalization, fluids and steroids (Griffin, 2000).

Epiglottitis is a less common but more vicious form of upper airway obstruction caused by fulminant infection of the soft tissues around the entrance to the larynx. The child presents with tachypnoea and severe sore throat so that swallowing saliva is difficult and drooling occurs. The child usually sits upright or in a 'tripod' position with neck extended and arms providing support for the accessory muscles. Prompt treatment is required, including intubation if steroids are unable to maintain a patient airway. Table 16.3 charts the differences.

Table 16.3 Croup and epiglottitis

	Croup	Epiglottitis
Age	6 months to 3 years	2–6 years
Aetiology	Viral	Bacterial
Onset	Over days	Over hours
Temperature	< 38.5°C	> 38.5°C
Cough	Barking	Minimal
Stridor	If severe only	Yes
Voice	Hoarse	Weak
Can drink	Yes	No
Active	Yes	No

Physiotherapy is not indicated in the acute phase for non-intubated babies, but may be required if symptoms persist, or for intubated babies if there are excessive secretions.

Bronchiolitis

Bronchiolitis is inflammation of the bronchioles due to viral colonization of the bronchiole mucosa. It is the commonest respiratory tract illness of infancy (Cade *et al.*, 2000), and is most usual in those born prematurely. Ciliary damage, excess mucus and mucosal oedema lead to airway obstruction and hyperinflation. Signs are excess oral secretions, wheeze, fine crackles, and breathlessness with chest retractions, indicating a fivefold or more increase in the work of breathing (Milner and Murray, 1989). Gas trapping may prevent sternal recession, unlike croup or pneumonia. Admission to hospital is required if the infant is too breathless to manage a cough or has a RR above 50/min (Isaacs, 1995).

Treatment is by hydration, humidity, oxygen, maintenance of the head-up position, minimal

handling and occasionally heliox (Paret *et al.*, 1996). Bronchodilators are rarely helpful, but if prescribed should be nebulized with oxygen because they can worsen hypoxaemia in wheezy infants (Rakshi, 1994). Antibiotics (Roosevelt, 1996) and steroids (Simoes, 1999) are considered ineffective, but benefits have been claimed for Chinese herbs (Kong *et al.*, 1993). The acute illness subsides suddenly in about a week, with recovery over 2–3 weeks, but 70% of infants experience recurrent cough and wheeze (Cade *et al.*, 2000).

Physiotherapy is not recommended routinely (Nicholas *et al.*, 1999) and tends to cause desaturation and increased wheeze in the acute stage. If sputum retention becomes a problem, percussion in modified side-to-side positions is indicated, with suction if necessary. Close evaluation is required by assessing post-treatment wheeze, oxygen saturation and how the child settles.

Pneumonia

The clinical course of pneumonia is more acute than in adults, with chest recession and RR above 50 sometimes seen. Rapid breathing may be the only sign (Ralafox, 2000), but slow laboured breathing can be an indication of very severe pneumonia. Physiotherapy is occasionally needed in the later stages if the child is unable to clear airway debris.

Pertussis (whooping cough)

This is a lower respiratory tract infection characterized by coughing spasms that terminate in a 'whoop' as air is gasped into the lungs, or in younger children by vomiting and apnoea. The disease can be lengthy (hence the nickname '100-day cough') and coughing severe, leading sometimes to airway damage and bronchiectasis in later life. Physiotherapy is only required if there is sputum retention, and treatment must avoid any stimulus that irritates the sensitive airways and sets off more coughing.

Cystic fibrosis

For cystic fibrosis, see page 87.

Fatigue, sleep apnoea, hyperventilation syndrome

Sleep apnoea and hyperventilation syndrome can both occur in children, and either may manifest as chronic fatigue or attention-deficit disorder. Enlarged tonsils can cause obstructive sleep apnoea, leading to snoring, failure to thrive, enuresis and behavioural problems (Lamm *et al.*, 1999). Management of sleep apnoea is as for adults, including CPAP when necessary (McNamara, 1999). Hyperventilation syndrome is commonly undiagnosed (Enzer, 1967).

Surgery

Preoperative management

Preoperative information reduces a child's distress (LaMontagne, 1996), and advice on breathing and mobility leads to more rapid recovery (Carmini *et al.*, 2000). Parents have not always explained the operation to the child. Without explanation, the boundary between reality and fantasy can be blurred, e.g. the distinction between anaesthesia and death. Children have been known to mistake a bone marrow test for a 'bow-and-arrow test' or a dye injection for a 'die injection'. Preoperative stress is greatest in younger children (Aono *et al.*, 1997).

Preoperative explanations are helped by pictures, rehearsal of procedures, visits to hospital facilities and encouragement to discuss the experience with children who have had the same operation. Physical sensations and their reasons should be explained. Truth is essential because if the child's trust is shaken, co-operation is lost. Parents require highly detailed explanations (Kain *et al.*, 1997).

Children should be allowed oral fluids 2 hours before surgery to reduce the risks of dehydration, nausea and hypoglycaemia (Phillips *et al.*, 1994). Excessive food starvation can be harmful (Veall, 1995). Early milk-feeding post-operatively helps reduce crying (Gunawardana, 2000).

Separating a screaming child from its parent

at the door of the operating room is no longer acceptable, and a parent should be present during induction of, and emergence from, anaesthesia (Hall *et al.*, 1995).

Pain management

Postoperative pain management for children is characterized by doctors under-prescribing and nurses under-administering (Hall, 1994). Young children can undergo intubation and chest drain insertion without medication, and older children have described the pain of medical procedures as the worst aspect of their condition (Yaster, 1995). Fulton (1996) has named this 'institutional violence' and describes children's subsequent behaviour as similar to that after non-accidental injury. Untreated pain has detrimental effects on short- and long-term clinical outcomes (Chambliss, 1997), and could bring prosecution if applied to animals. Children with neurological impairment are at particular risk (Pederson and Bjerke, 1999). The causes of poor pain management in children are legion:

- Children's subjective complaints may not be taken seriously. Health staff tend to rely on assumptions and personal beliefs when assessing children's pain (Beyer and Byers, 1985).
- Distinguishing pain from agitation is challenging in young children, and children may not express pain in terms that are easily understood by adults. Absence of crying does not indicate absence of pain.
- Doctors' anxieties about drug side effects have led to children being described as 'therapeutic orphans' (Yaster, 1995). Opiates cause no more respiratory depression than in adults (Twycross, 1998), but meticulous prescription is required.
- Children may minimize complaints if analgesia is administered by the dreaded needle.
- Children are easily held down by force.
- Some health staff do not realize that pain is experienced from birth, and indeed before birth (McCullagh, 1996). There is evidence of synapses in a fetus as young as 8.5 weeks' gestation, and response to tactile stimuli at 5 weeks gestation (McCullagh, 1996). Invasive procedures during the first trimester of pregnancy have been linked to impaired lung function and increased respiratory symptoms after birth (Greenough and Yüksel, 1997).

The low priority given to children's pain is reinforced by some medical textbooks, e.g. '... these patients vigorously object to having an arterial puncture done even if they are relatively sick. Because of this, more than one person is usually required to obtain the sample' (Deming, 1995, p. 213). This demonstrates little awareness of the ethical, legal and humane considerations in relation to forcibly holding down a child to inflict pain.

Parents' opinions must be actively sought because they tend to assume that everything to minimize pain is done automatically. For older children, it is better to ask the child because parents may underestimate their child's pain (St-Laurent-Gagnon, 1999). Self-report also gives an indication of the associated fear (Manne *et al.*, 1992). Children over 7 can use a visual analogue scale. Those over 3 can use colour intensity scores, face scales (Figure 16.3) or charts with

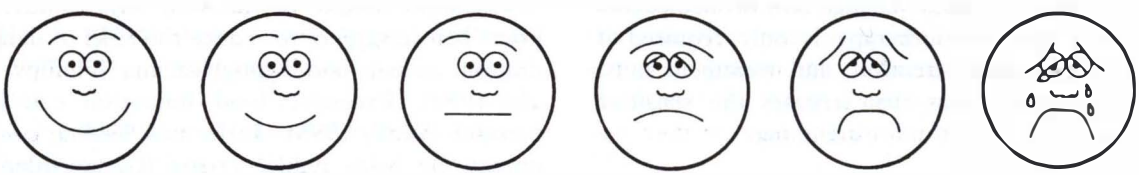


Figure 16.3 Faces scale for pain assessment in children. (From Carter, B. (1994) *Child and Infant Pain*, Nelson Thornes.)

body outlines. Prelingual and non-verbal children can be observed for signs of withdrawal, face and body reactions, irritability, pallor, momentary breath-holding, prolonged sleeping, or in older babies who are experienced in hospital procedures, an expression of frozen watchfulness. Physiological measures such as changes in RR, heart rate, BP and S_aO_2 can be used as adjuncts but are not specific as indicators of pain and not sustained with continued pain.

Children fear injections more than anything else in hospital (Twycross, 1998) and a painless route of administration is required. An exception is the rectal route because absorption is slow and variable, it can be perceived as abusive, and there has been one known fatality (Gourlay and Boas, 1992). Children can use patient-controlled analgesia from the age of 4, TENS (Lander, 1993), and 'fentanyl lollipops' (Chambliss, 1997).

Nausea needs treating because it can lead to dehydration, wound disruption and later re-admission (Paxton, 1996). If sedation is required, doses should normally be greater than recommended (Murphy, 1997). Both sedation and opioids need to be reduced gradually (Fonsmark *et al.*, 1999).

Postoperative management

Children waking up in an intensive care unit are subject to extra fears, especially if explanations are not full and clear. Nasogastric tubes can increase the work of breathing and increase apnoeas (Oberwaldner, 2000) and for long-term enteral feeding, gastrostomy feeds are more effective (Cosgrove, 1997). Apnoeas are common in preterm infants (Levin, 1999).

Children like to be touched as little as possible after surgery. If coughing is necessary, they prefer to splint the incision themselves by leaning forward with their arms crossed or hugging a teddy bear. Children must not be discouraged from crying nor told to be brave. If they are 'difficult', it is usually because they are frightened.

Child survivors of road accidents show a high incidence of long-term effects, including depression, attempted suicide and post-traumatic stress disorder (Yule, 1999). Children tend to mistake flashbacks for reality and keep their feelings to themselves so as not to upset their parents.

Dying children

Children have a right to grieve. They have the capacity to do so, and begin to develop an understanding of death from the age of 2–3 years (Sheldon, 1998). They may be prevented from this necessary process because of a natural desire by others to protect them from suffering. Children understand more than they can articulate and usually know if they are going to die (Purssell, 1994). Evasion can leave them with a sense of bewilderment, betrayal and fantasies that are more frightening than reality.

Many children are able to take decisions about whether to have active or supportive therapy (Purssell, 1994). Communication with dying children should be based on honesty. If death is compared to sleep, for example, they may develop an unhealthy fear of bedtime.

As well as experiencing the adult responses to dying, children carry the burden of their parents' grief. Parents may carry the burden of being avoided by their friends. Siblings have been identified as the most unhappy of the family members (Harding, 1996) and show double the risk of psychological disturbance (Black, 1998). They may be shunned by friends, worry about their own vulnerability, and be confused by a mixture of what they have been told, overheard, observed and imagined. Siblings should not be fed 'Susie-is-going-away-on-a-long-trip' euphemisms or they may wait for her return. When asked about their own needs, siblings have requested information, open family communication and active involvement in the dying child's care (Harding, 1996). Sheldon (1998) details storybooks and workbooks that assist communication.

The quality of care for the child has a major impact on the family's bereavement (Stead, 1999). Symptoms such as fatigue may not be treated, even when the cause is anaemia, depres-

sion or malnutrition. The fact that 75% of children with cancer now survive has led to a tendency for aggressive treatment to take precedence over palliation even when there is little hope of cure (Wolf *et al.*, 2000). One study found that half the children who died in hospital were mechanically ventilated for their final 24 hours (Wolf *et al.*, 2000).

Comprehensive support for the family, including contact after the death, helps reduce the high incidence of distress, divorce and sibling neglect that tends to accompany the death of a child. It is unhelpful to tell parents that they will get over their child's death because it is rarely true. Parents may find some ease in reflecting that it may have been better to have loved and lost a child than not to have had the child at all.

THE NEONATAL ICU

Introduction

> ***The emergence of the baby into the outside world is perhaps the most cataclysmic event of its life.***
>
> West, 1995

The sharp intake of breath that adults take in response to sudden cold is thought to be a physiological memory of the first breath. All babies have undergone the trauma of birth and the complex transition from respiration via the placenta to gas exchange in the lung. Premature babies have the added shock of being displaced foetuses. They are delivered into a world against which they have limited defence mechanisms, and sometimes without the basic capacity for respiration, kidney function or temperature control.

The lower the gestational age, the more keenly sensitive are premature babies to their environment. The immature cochlea is particularly sensitive to noise levels, and a noisy environment can cause hypertension, raised intracranial pressure, hearing loss (Mishoe, 1995) and disturbed breathing (Figure 16.4). A neonatal intensive care unit (NICU) provides the technology and skill to care for sick babies, preterm or term. It is not always their ideal environment, with its bright lights, chorus of noises, frequent disturbances and resistant bacteria (Man *et al.*, 2000).

Central to a baby's universe is his/her mother, and infants recognize physical separation from birth (Christensson, 1995). Bonding between child and mother is hindered by the barrier of the incubator and the mother's reticence in disturbing equipment. NICU 'graduates' run an above-average risk of language delay (Jennische and Sedin, 1999), educational handicap (Saigal, 2000) and abuse in later life (Anon, 1985). Attention has now focused on optimizing the environment and the bond between parents and child. Parents need to be involved in the care and comfort of their baby, and babies need to

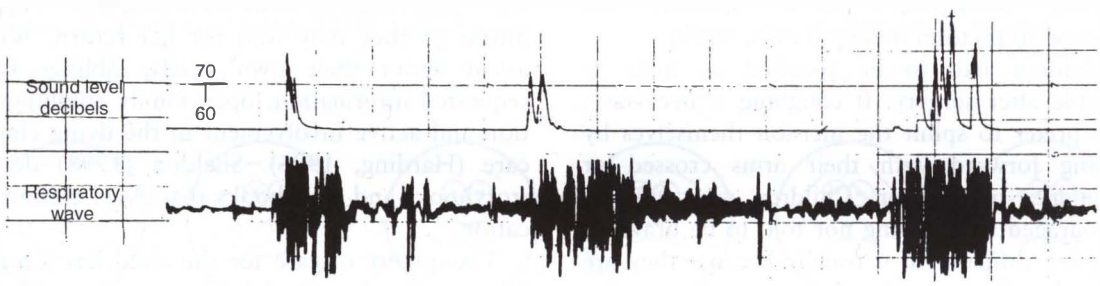


Figure 16.4 The effect of noise on the breathing pattern of a premature infant. (From Long, J. G., Lucey, J. F. and Philip, A. G. S. (1980) Noise and hypoxemia in the intensive care nursery. *Pediatrics*, **65**, 143–145, with permission.)

hear and feel their mother. Handling by health staff can destabilize preterm infants (Murphy, 1991), but stroking and gentle handling have shown beneficial effects (Harrison, 1996) and handling by the mother can reduce stress and oxygen consumption (Ludington, 1990). Monitoring provides feedback.

Babies are known as neonates for the first month of life, and neonates born before 37 weeks' gestation are considered preterm. Respiratory problems are the commonest cause of morbidity and mortality (Wilson, 1992). Problems are more abrupt than in adults because of their different respiratory systems, as described below.

- The immature respiratory centre causes irregular breathing patterns and occasional periods of apnoea.
- For the first year of life, the intercostal muscles are immature, the ribs cartilaginous and horizontal, and the rib cage nearly three times as compliant as the lung (Papastamelos, 1995). The diaphragm does most of the work of breathing despite containing less fatigue-resistant fibres than adults and working at a disadvantage because of its horizontal angle of insertion. Work of breathing is 2–3 times that of adults (Hoffman, 1995). By 2 years old, the bucket handle action has developed and the rib cage and lung become equally compliant (Papastamelos, 1995). By 3 years old, when more time is spent upright, rib cage configuration is oblique rather than horizontal.
- Response to heavy work loads is an increased rate rather than increased depth of breathing.
- Hypoxaemia tends to cause bradycardia rather than tachycardia. Immature myocardium has less capacity to increase stroke volume, and bradycardia may reduce cardiac output.
- Collateral ventilation is not established until age 2–3 years, leaving the lungs vulnerable to atelectasis. Maturity of the surfactant system occurs at about 35 weeks' gestation, and preterm infants are at extra risk of atelectasis.
- Peripheral airways are narrow and contribute up to 40% of total airways resistance from birth to about age 5, leaving young children prone to obstructive diseases of the small airways such as bronchiolitis (James, 1991).
- Blood pressure regulation is unrefined.
- The normal respiratory rate in infants and children has an extensive range that is more responsive to disease and emotion than that of adults.
- Most babies are able to breathe through their mouths but are preferential nose-breathers because their large tongues narrow the oropharynx (Sporik, 1994).

Care of the parents

I longed as I have never longed for anything so badly, to hold her ... to put her face against mine, whisper that I was here, that it was all right .... I stroked her hand with my finger ... and feeling her minute pink fingers holding so hard to mine, I was hit sideways and bowled over by the purest, tenderest, most passionately committed love I have ever felt ....

I couldn't bear to ... not be able to help her myself at all .... It hurt me so much, to see her weak, in distress and apparently struggling so hard.... I wanted to drag everything away from her, every bit of machinery, to pick her up and hold her to my breast, even if it meant that she died in a few minutes. That would be better than having her suffer this, the pain, fear, noise, bewilderment, being surrounded and handled by strangers....

Every time I touched her, she relaxed and the monitors showed it, her heart rate settled, her limbs were calmer, her eyes searched less frantically about....

I had never held her to me and I ached to do so, she seemed so alone in there amongst all the wires and drips and tubes and monitors.

Hill, 1989

Management of pain and stress

Evolution has not yet caught up with the survival of premature babies. Sky (1990) suggests that they are hyperconscious and only gradually develop mechanisms for filtering the sensory inputs that await them in life. Pain causes greater long-term harm to infants than to older children, and even greater damage to preterm infants (Larsson, 1999), increasing morbidity and mortality (Lancet, 1992), and can cause hypersensitivity to subsequent pain (Barker, 1995). At the same time, the capacity to communicate appears not to have evolved at this stage (Hadjistavropoulos, 1997), which is why specific assessment tools are necessary (Blauer, 1998).

Premature infants exist in a precarious metabolic milieu, and loud voices, knocking the incubator or even rearranging a limb can lead to bradycardia, disorganized breathing and hypoxia. Pain or stress causes hypertension, hypoxaemia and hypercapnia (Wessel, 1993). Suggestions to reduce stress during physiotherapy are the following.

- Light and noise can be reduced by partially covering the incubator during treatment and keeping sounds to a minimum (Cole *et al.*, 1990), apart from talking to the infant. Equipment should not be put on the incubator. Heat loss must be avoided, especially from the head.
- Restraints should be avoided unless essential (Sparsholt, 1989).
- Procedures that cause crying should be minimized because they predispose to irregular breathing, apnoeic episodes, pulmonary hypertension and hypoxaemia (Murphy, 1991).
- If sedation is required, propofol can be used for brief procedures (Erb and Frei, 1996).

A team approach to stress reduction results in a more stable cardiopulmonary system, with benefits lasting for months after discharge (Mann, 1986). Examples are described below.

- Dimmed lights at night encourage sleeping and weight gain. Ear muffs decrease episodes of desaturation, crying and intracranial hypertension. Other beneficial influences are the mother's voice, a soft blanket or sheepskin (Sparsholt, 1989), 'silent' rubbish bins, quiet radios, swaddling in 'cosy nests' or other positioning aids, and 'I am sleeping till ... pm' signs (Hutchon, 2000). Periods of undisturbed sleep reduce risks of hypoxaemia, hypertension and apnoea (Cole *et al.*, 1990).
- Skin contact with the mother and hearing the mother's voice lead to a reduction in oxygen consumption (Ludington, 1990) and pain (Gray *et al.*, 1999).
- Visiting should be unrestricted and include grandparents and siblings (Johnstone, 1994).
- Intubation upsets BP, HR and S_aO_2, and the child must be premedicated (Bhutada et al., 2000). Heelsticks are thought to be more painful than venepuncture, especially the heel squeeze (Lindh, 1999) and they should be accompanied by skin anaesthesia (Jain, 2000). One blood sample should be used for multiple measurements as well as blood gases, and a long intravenous line is preferable to frequent attempts at venous access (Chiswick, 1999). When required, neonates benefit from epidural analgesia (Ochsenreither, 1997), and premature neonates can be given intravenous opioids (Franck, 1998).
- A stressed parent means a stressed baby, and parents benefit from help to cuddle their child when attached to awesome equipment, advice on baby massage, and a rocking chair and other comforts for them and their child.

Rocking beds have been advocated for babies because of their positive effects on ventilation, feeding, crying and length of hospitalization (Sammon, 1994).

The relevance of mother–baby contact was highlighted by Kennell (1999), when an episode of staff shortage obliged mothers to provide 24-hour care for their premature infants. Outcomes were reduced infection, improved weight gain,

increased breast-feeding, quicker development and, for the mothers, more rapid recovery from childbirth. So keen were the babies on the low-technology approach that they even preferred unwashed to washed nipples. Gale and Vanden-Berg (1998) took this a step further with 'kangaroo care', when equipment shortage led to mothers incubating preterm babies next to their skin. Outcomes were similar to the first study, plus improved sleep, oxygenation and muscle tone, greater stability and reduced mortality. Snuggling next to the mother's breasts is a baby's ideal ecological niche.

Temperature and fluid regulation

The more immature the baby, the less efficient is heat conservation because of scant subcutaneous fat, fragile skin, inability to sweat or shiver and a large surface area in relation to body mass. Up to 50% of calorie intake may be used for maintaining body temperature. Warmth from overhead radiant heat allows easier access to the baby than an incubator, but promotes water loss. Both dehydration and fluid overload are risky for an immature kidney.

Oxygen therapy

Supplemental oxygen is given via a Perspex head box or, for longer-term use, a nasal cannula or catheter (Coffman and McManus, 1984). Hyperoxia or fluctuations in F_IO_2 can lead to retinopathy of prematurity (Cunningham, 1995) due to excessive oxygen pressures reaching the retinal artery. This causes constriction, proliferation and fibrosis of the delicate retinal capillaries, leading to blindness. Oximetry cannot detect hyperoxia, and S_aO_2 should be kept between 87% and 92% in preterm infants to allow a margin of safety. The oximeter probe site must be changed 4-hourly. With transcutaneous monitoring, $P_{tc}O_2$ should be kept at 6.7–10.7 kPa (50–80 mmHg) and $P_{tc}CO_2$ at 5.3–7.3 kPa (40–55 mmHg). Control of oxygen therapy is essential because lack of oxygen can also worsen retinopathy of prematurity (Stephenson, 2000).

Feeding

The effort to co-ordinate breathing, suckling and swallowing can reduce S_aO_2 in premature infants (Pickler, 1996), and extra oxygen may be required during feeds even if a feeding tube is in place (Shiao, 1995). Preterm infants need preterm formula rather than standard formula in order to maintain brain growth and prevent long-term cognitive dysfunction (Lucas *et al.*, 1998).

Humidification

Hot-water humidification is required for intubated infants, and often for spontaneously breathing infants because their small nasal passages and airways block easily. Heat–moisture exchangers may increase the work of breathing and at present do not supply the warmth to prevent loss of body heat. Ultrasonic humidification can be hazardous in intubated babies (Tamer, 1970).

Mechanical ventilation

If neither oxygen nor CPAP are able to maintain oxygenation, mechanical ventilation may be needed. Tracheal tubes are uncuffed until up to 10 years of age, allowing a slight air leak and less risk of mucosal damage. The mucosa is vulnerable because the subglottic area is the narrowest part of the airway and young children tend to move their necks more than adults. Elaborate systems for endotracheal tube fixation are required to prevent such a heavy contraption from becoming disconnected from such a tiny nose. Pressure-controlled ventilators are used for infants up to 1 year old, so that high peak airway pressures are avoided and flow can increase automatically to compensate for the cuff leak.

The high compliance of the chest wall and low collagen and elastic content of lung tissue afford little protection against overdistension, and a quarter of ventilated babies develop some form of barotrauma (Parker and Hernandez, 1993). The following may occur.

- Pulmonary interstitial emphysema (PIE) is identifiable radiologically as lucent streaks radiating from the hila, representing air in the interstitium. Unlike air bronchograms, the streaks do not branch or taper. Extension of PIE to the periphery can lead to pneumothorax.
- Pneumothorax is suspected if there is rapid deterioration without obvious cause.

Babies with respiratory distress syndrome are particularly at risk of PIE because of their lack of surfactant. Babies with more compliant lungs are more at risk of pneumothorax (Chatburn, 1991). A pneumothorax is treated by chest tube drainage, but PIE is difficult to treat and causes up to a third of ventilated low-birth-weight babies to develop chronic lung disease of prematurity (Coghill *et al.*, 1991). These complications may be reduced with a low tidal volume, or by using high-frequency ventilation or negative pressure ventilation (Samuels and Southall, 1989).

PEEP (p. 353) is used in all neonates at 2–5 cmH_2O but is specifically required if P_aO_2 is < 6.7 kPa (50 mmHg) at $F_IO_2 > 0.6$ (Pilbeam, 1998, p. 360). Following extubation, subglottic oedema may develop immediately or over 24 hours. In premature infants on prolonged IPPV, airway damage from the tracheal tube and suction can lead to tracheobronchial stenosis (Brownlee, 1997).

Neonatal support systems

If bronchodilation is required, preterm and term infants on IPPV can respond to inhaled bronchodilators delivered by nebulizer or inhaler and spacer (Sivakumar *et al.*, 1999). Advanced support systems (Chapter 12) are more established for neonates than for adults (Barrington *et al.*, 1999).

PHYSIOTHERAPY FOR NEONATES

The main role of the physiotherapist is to judge if and when intervention is appropriate. Treatment itself may be carried out by the physiotherapist, neonatal nurse or in part by the parent. The maxim that routine treatment is taboo is never more apt than in the NICU. The approach is to assess, identify any problems, and balance up the benefits and risks of intervention.

Indications

Physiotherapy may be needed if there are excess secretions that cannot be cleared by humidification and suction, or if there is poor gas exchange, increased work of breathing or radiological evidence of atelectasis. Treatment may be indicated after extubation if airway irritation has created excess secretions. Neonates who have aspirated meconium need treatment unless contraindicated.

Precautions

Because of the poorly developed defences of neonates, all health workers should wash their hands meticulously and avoid the NICU if they have an infection. Physiotherapy is contraindicated for neonates who are hypothermic, show cardiac instability (unless this is due to hypoxia), have an undrained pneumothorax or are producing fresh bloodstained secretions. Treatment should be scheduled before feeds or over 1 hour afterwards. If physiotherapy is necessary within these times, the gastric contents can be aspirated by syringe via the nasogastric tube before treatment, and replaced afterwards. If the infant is receiving phototherapy for jaundice, it can be removed temporarily for treatment; when replaced, protective eye shields must be put back.

Infant and monitors should be observed before, during and after treatment. Large swings in BP are particularly damaging for preterm paralysed infants (Bohin, 1995). Casual handling should be avoided and physiotherapy sessions structured so that the infant is rested before and afterwards. Cumulative procedures or major disturbances such as suction can reduce P_aO_2 by as much as 5.3 kPa (40 mmHg) (Speidel, 1978). Lights should be no brighter than needed for safe observation. Oxygen desaturation may be caused by suction, or may be an indication for

suction. It can also be caused by the head falling into flexion and obstructing the upper airway.

Assessment

Not all neonates need hands-on assessment. The nurse will have information on secretions and response to interventions. Intubated neonates need a check on their humidifiers. Other details are below.

Notes, charts and reports give information on birth and other history, weight gain or loss, response to handling and suction, results of the last suction, mode and frequency of feeds and whether the baby has rested since the last intervention. The Apgar score gives an indication of birth asphyxia through heart rate, respiratory effort, reflex irritability, muscle tone and colour. A recent history of self-limiting bradycardia or periods of apnoea suggests that suction might be needed. Much reliance is placed on this information because of the limitations of clinical and subjective assessment. Arterial blood gas values correlate with those of capillary blood that has been warmed to 'arterialize' it, usually taken from the heel.

Monitors indicate physiological distress, shown by bradycardia (HR < 90/min), tachypnoea or apnoea. A respiratory rate of more than 60 may predict hypoxia in infants (Rajesh *et al.*, 2000). Worsening oxygenation may be a sign of accumulating secretions or infection. Normal S_aO_2 is 97–100% and must not fall below 93%. Preterm infants have a left-shifted dissociation curve because of foetal haemoglobin, and desaturation may reflect a lower P_aO_2 than in adults.

Breath sounds at the mouth of an intubated infant include the slight hiss of the intentional air leak. Upper airway obstruction with a mucus plug increases this sound when air is forced out past the tracheal tube. If this is reduced after physiotherapy, it may indicate a good outcome.

Auscultation of the chest seems to pick up every sound in the NICU. Rapid shallow breathing, or ventilator noise and other referred sounds, can thwart the listener. Wheezes and crackles are transmitted throughout the chest, and may be easier to feel than hear. *Palpation* is necessary not just over the chest but also to check for abdominal distension, which can be disabling in a baby dependent on the diaphragm to breathe.

X-ray findings may indicate atelectasis or consolidation, with the right upper and middle lobes needing special attention because of their tendency to collapse (Figure 16.5). Neonates have a large thymus, which looks similar to right upper lobe consolidation. Air bronchograms projected through the heart shadow may not be significant but are pathological when seen peripherally. Any sign of PIE (p. 438) contraindicates manual hyperinflation.

Methods to increase lung volume

Positioning

Spontaneously breathing neonates, especially when premature, benefit from raising the head of the mattress to ease the load on the diaphragm and lessen the risk of gastrooesophageal reflux.

Side-lying allows greater diaphragmatic excursion than supine. If there is a pneumothorax or unilateral PIE, side-lying with the affected lung dependent may assist absorption of the unwanted air (Swingle *et al.*, 1984), under cover of monitoring. When infants are in side-lying, they respond best when the trunk and limbs are supported in a flexed position.

The prone position puts unmonitored neonates at risk of sudden infant death syndrome or cot death (Hallsworth, 1995). However, prone is not contraindicated in the supervised environment of the ICU, and in preterm infants has been shown to stabilize the chest wall and increase S_aO_2 (Dimitriou, 1996), so long as the endotracheal tube is long enough to avoid displacement (Marcano, 2000). Prone neonates require the head of the mattress to remain raised.

Precautions to observe when positioning a neonate are to monitor the effects of handling, avoid any pull on the tracheal tube and check for change in air leak around the tracheal tube after position change.

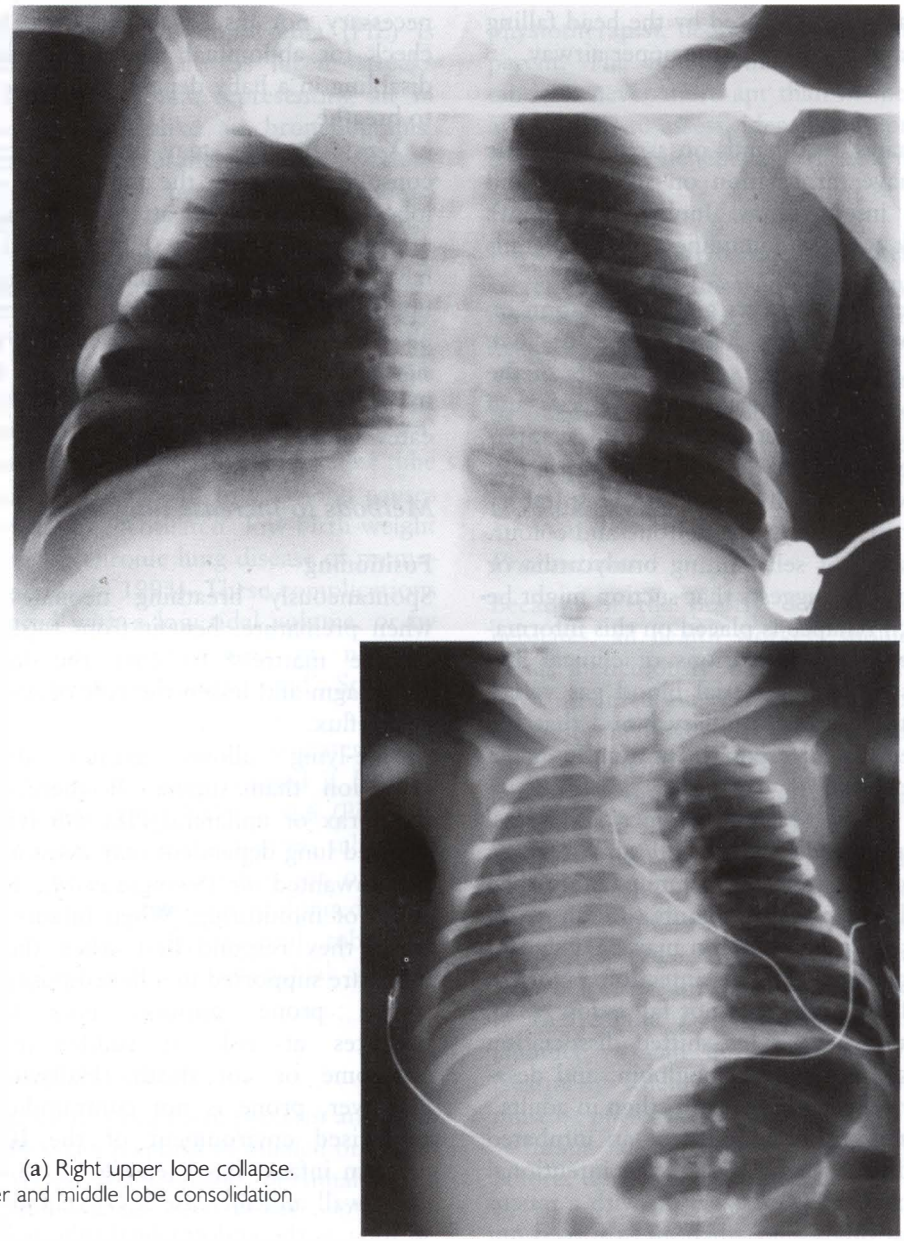


Figure 16.5 (a) Right upper lope collapse. (b) Right upper and middle lobe consolidation and collapse.

Manual hyperinflation

The younger the child, the less advisable it is to use manual hyperinflation (MH) because of the risk of pneumothorax. Contraindications are similar to adults, with three additions:

- hyperinflation conditions such as meconium aspiration and bronchiolitis
- prematurity, because the risk of pneumothorax is too great
- infants whose RR is over 40/min, because it is impossible to achieve an effective hyperinflation.

The indication for MH is loss of lung volume that does not respond to positioning or clearance of a mucus plug. A 500 mL bag is used for infants and a 1 L size for children. These bags have an open tail which is squeezed between finger and thumb to regulate the pressure more sensitively than a valve. A suggested technique is as follows:

1. Incorporate a manometer in the circuit to check pressures (Howard and Koniak, 1990).
2. Check monitors.
3. Turn oxygen flow to 6 L/min (although gas flow to the infant is controlled manually).
4. Bag-squeeze using fingers rather than the whole hand, interspersing each hyperinflation with three or four tidal breaths.
5. Control pressure so that the chest rises only slightly more than during IPPV, and the manometer indicates a rise of no more than 5 cmH_2O above the peak airway pressure for infants and 10 cmH_2O for older children (Parker, 1998).
6. Maintain some positive pressure at the end of expiration to mimic PEEP and prevent airway collapse.
7. Between watching the manometer and monitors, observe the patient.

Methods to decrease the work of breathing

Work of breathing is increased by stress (Wessel, 1993). Measures to reduce stress are described on page 436. Positioning head up, as described above, decreases the work of breathing.

Methods to clear secretions

Postural drainage

Unstable preterm neonates who cannot tolerate handling should not have their position changed for treatment. Other babies can be treated in alternate side-lying. If the head-down tip is necessary, observation and monitoring should be continuous because of the baby's reliance on diaphragmatic function.

Percussion and vibrations

Manual techniques are fruitful in neonates because of the compliant rib cage. Percussion is usually well tolerated and may be soothing. It can be performed with a soft-rimmed face mask, using firm pressure directly on the skin and taking care to stay within the surface markings of the little lungs. Vibrations with the finger tips can be applied on every second or third expiration.

Contraindications include those on page 193 plus risk of intraventricular haemorrhage and rickets. Monitors should, as always, be observed throughout because some neonates respond poorly. If manual techniques are essential in preterm babies, they must be as delicate as possible while maintaining effectiveness, and the head must be supported throughout. It is advisable to liaise with a paediatric respiratory physiotherapist before using manual techniques with premature babies because of the risk of causing a form of brain damage similar to 'shaken baby syndrome' (Harding *et al.*, 1998).

Suction

Secretions in the endotracheal tube can double airflow resistance (Chatburn, 1991), and shallow suction is indicated for intubated neonates as required. Deeper suction should only be used if necessary because it can cause bradycardia, arrhythmias, atelectasis, abrupt peaks in blood pressure, raised intracranial pressure (Durand *et al.*, 1989) and pneumothorax (Vaughan *et al.*, 1978). Figure 16.6 shows how the blood pressure of a baby increased by 20 mmHg during endotracheal suction.

For non-intubated infants, positioning and percussion may shift secretions so that they are swallowed. If not, suction may be necessary. The techniques described on pages 205 and 429, are modified by the following:

- Have the baby in side-lying and wrapped up comfortably but firmly.
- For preterm infants, preoxygenate by no more than 10% to avoid retinopathy of prematurity (Parker, 1998).
- Set the vacuum pressure (p. 429).

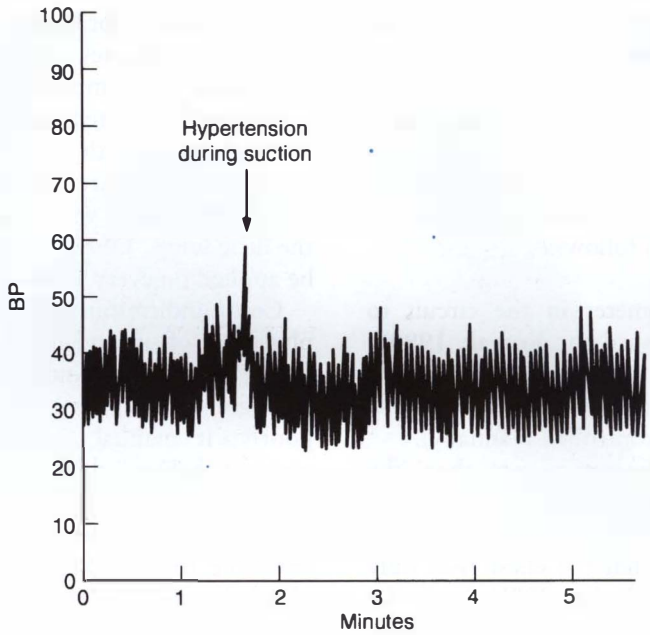


Figure 16.6 Hypertension in an infant during suction. (From McIntosh, N. (1989) MARY – a computerised neonatal monitoring system. *Int. Care Clin. Monit.*, **10**, 272–282)

- Use a size 5–6 FG catheter.
- Observe monitors.
- Suction the nasopharyngeal airway, passing the catheter only as far as stimulates a cough.
- Suction the nostrils afterwards.
- Invite the parent to cuddle the baby.

For ventilated infants, tracheal suction is required if the recent history suggests that suction is productive, or if monitors indicate hypoxaemia. It may not be possible to feel or auscultate for the presence of secretions. Modifications to the above protocol are described below.

1. Avoid suction if the temperature is below 36°C or there are signs of decreased cardiac output or shock (pallor or cyanosis, sweating, decreased peripheral temperature).
2. Lubrication is not necessary in intubated babies.
3. Liaise with medical staff about the advisability of predosing with a drug to blunt the stress response (Hickey *et al.*, 1985).
4. Preoxygenate by increasing the ventilator F_IO_2 by 10–20%, but observe oximeter to monitor the response (Hodge, 1991).
5. Avoid deep suction. The catheter should not advance more than 1–2 cm beyond the end of the tracheal tube (Vaughan *et al.*, 1978). Its length can be checked against the length of an equivalent-sized ETT, which is usually taped to the outside of the incubator for emergencies, or a calibrated catheter can be used (Kleiber *et al.*, 1988).
6. Ensure catheter diameter is less than half the diameter of the airway (Bertone, 1988):
   5 FG for ETT size 2.5 mm
   6 FG for ETT size 3 mm
   8 FG for ETT size 3.5 mm.
7. For postoperative infants, ensure that someone supports the wound.
8. Draw up normal saline into a syringe, disconnect the infant from the ventilator, instil saline down the tracheal tube (0.5 mL

for preterm neonates, 1–3 mL for term babies), reconnect for a few breaths, then disconnect and suction.
9. After reconnection to the ventilator, suction mouth and nostrils.
10. When S_aO_2 has returned to baseline, turn the F_IO_2 back to its previous value slowly. If the oximeter or other monitors do not show that values have returned to baseline, or the child does not settle, further suction may be indicated, or another problem investigated in liaison with the nurse.

Two alternatives have been suggested for instilling saline. Downs (1989) describes more accurate saline delivery, so long as hypoxaemia is not a significant risk:

1. Inject saline into an unconnected sterile suction catheter until the catheter is filled and a drip is visible at the tip.
2. Disconnect the ventilator and insert the catheter into the tracheal tube.
3. Instil saline directly to the distal end of the tracheal tube, using the syringe.
4. Withdraw saline, along with some of the mucus, using the syringe.
5. Disconnect catheter from the syringe, connect to the suction circuit, suction.
6. Monitor.

Prasad and Hussey (1995, p. 88) describe lavage for mucus plugging, to be carried out under close monitoring and in association with the anaesthetist:

1. Preoxygenate and sedate.
2. Position the baby in the reverse postural drainage position, i.e. the affected part downwards.
3. Instil saline, 2–5 mL for infants and 10–15 mL for older children.
4. Manually ventilate with tidal volumes.
5. Reposition into postural drainage position.
6. Apply manual techniques and MH.
7. Suction.
8. Monitor.

Caution should be observed after extubation, because suction-induced bronchospasm may cause obstruction.

Developmental management

Nearly 50% of extemely premature babies grow up disabled (Christie, 2000), and all premature babies require assessment by specialist colleagues.

Extended time in prone may lead to a flattened frog position because of hypotonia, and this can be avoided by raising the pelvis on a roll (Downs *et al.*, 1991). Passive movements are normally unnecessary, but for very-low-birth-weight babies, daily gentle exercise can improve weight and bone mineralization (Moyer-Mileur, 2000). Monitors should be observed because handling may destabilize the infant.

Low-birth-weight babies enjoy waterbeds, water pillows, rocking beds and air mattresses (Long, 1995), and very-low-birth-weight babies appear to benefit from womb-like swaddling in flexion (Short *et al.*, 1996).

MODIFICATIONS FOR SPECIFIC NEONATAL DISORDERS

Meconium aspiration

Full-term babies who suffer asphyxia during prolonged labour may pass meconium (faecal material) before birth, then gasp and suck it into their mouth. It stays safely there until delivery, but emergence of the chest causes it to be drawn deep into the lungs by the first breath. This results in acute obstruction of small airways, which if complete causes atelectasis, and if incomplete causes hyperinflation. The sticky meconium sets up a chemical pneumonitis which provides an ideal medium for infection.

If labour is prolonged, or monitoring suggests that the baby is in distress, airway suction during birth, before delivery of the thorax, reduces the risk. If aspiration has occurred, IPPV should be withheld until the airways have been suctioned so that particulate material is not forced into distal airways. If mechanical ventila-

tion is initiated, it is like ventilating through treacle.

Immediate and intensive physiotherapy is needed in the form of postural drainage, percussion, vibrations and suction. Treatment is continued until secretions are free from the dark colour of meconium. Midwives should be taught this technique unless physiotherapy is available straight after birth.

Intraventricular haemorrhage

Bleeding into the cerebral ventricles may occur in the first week of premature life, when swings in BP or arterial blood gases can cause the fragile capillaries in the ventricles to burst. Precipitating factors are pain (Larson, 1999), manual techniques, suction, endotracheal obstruction or intubation without sedation (Wren, 1989). If it occurs, the head-down tip is contraindicated and any physiotherapy is to be avoided unless essential.

Respiratory distress syndrome

Respiratory distress syndrome (RDS) occurs in premature babies, especially when delivered by caesarean section, and is the commonest cause of death in the preterm infant (Wilson, 1992). Lack of surfactant leads to patchy atelectasis, stiff lungs and increased work of breathing. Alveoli inflate with difficulty and collapse between respiratory efforts.

Signs of respiratory distress develop in the first hours of life. Breath sounds are reduced and diffuse fine crackles are heard. The radiograph shows hyperinflation with mottling and air bronchograms, indicating areas of atelectasis. Distress persists for 24–48 hours, then the condition either stabilizes and improves over several days as surfactant is produced, or the disease is protracted and severe. Mortality is 50%, but survivors show little morbidity (Heulitt, 1995).

Prevention is by prophylactic instillation of artificial surfactant on delivery of preterm babies, administered in different positions to ensure even distribution (Willson, 1998). Management is by regulation of temperature, fluid and nutrition (Tang, 1997) and respiratory support using oxygen, CPAP, IPPV or high-frequency oscillation (Plavka *et al.*, 1999). Normal ventilation pressures for infants are 15–20 cmH_2O, but infants with RDS require 20–40 cmH_2O. This must be reduced during recovery to avoid haemodynamic compromise.

Physiotherapy is limited to advice on positioning in the early stages, anything more energetic being unnecessary and sometimes destabilizing. Periods in alternate side-lying assist secretion clearance and midline orientation, and help prevent postural abnormalities. Intubation irritates the airways and may stimulate excess secretions, which need to be cleared in the recovery phase when the infant is stable, usually with suction and occasionally with percussion.

Chronic lung disease of prematurity

The more premature an infant, the more likely s/he is to suffer a continuum of lung injury progressing from RDS, PIE, oxygen toxicity and finally to chronic lung disease of prematurity, also known as bronchopulmonary dysplasia. This results from the interaction of immature lungs with high-volume mechanical ventilation, and is considered present if the infant reaches term age and cannot be discharged from hospital without oxygen or ventilatory support (Verklan, 1997).

Inflammation interferes with surfactant production and leads to scarring, disordered lung growth, stiff lungs and pulmonary hypertension. Signs are persistent respiratory distress and high oxygen requirements. X-ray changes range from 'grey' lungs to widespread cystic areas interspersed with regions of collapse. Lengthy intubation leads to erosion of the upper airway in up to half of patients, often undiagnosed (Doull, 1997).

Prevention is by ventilator management to minimize inflation pressures (Greenough, 1990). Treatment is by diuretics, preferably inhaled (Prabhu, 1997), bronchodilators and, ironically, increasing levels of oxygen and higher inflation pressures as the disorder progresses. Failure to thrive is minimized by adequate nutrition to

compensate for the high metabolic rate (Martin and Shaw, 1997). Long-term hospitalization may be required.

Physiotherapy is indicated if secretions are present because the lungs are prone to recurrent atelectasis and infection. Treatment is avoided or modified if the child is wheezy or has pulmonary hypertension. When it is necessary, treatment is preceded by bronchodilators and stopped if wheezing is precipitated. Percussion in alternate side-lying and sometimes suction can be given, with extra attention to the upper lobes. Physiotherapy may be required after discharge, either directly or through parent education.

After long hospitalization, parents need comprehensive preparation for discharge so that they build up confidence and do not feel that they have 'borrowed' their baby to take home. Domiciliary oxygen or non-invasive ventilation may be required (Teague, 1997). For prelingual children who require a tracheostomy, delayed communication, including hearing loss, can be prevented by speech–language therapy (Orringer, 1999). To assist communication, an uncuffed tube can be occluded with a gloved hand on expiration if the child is attempting to talk, laugh or cry. Occlusion must be brief and explained to the child. Toddlers learn to drop their chin to occlude the tube when they want to talk.

Chronic lung disease of prematurity is survived by 70% of children, but they are left with a risk of cot death and sometimes neurological problems. The lungs can repair as they grow, but it is thought that adult chronic lung disease is in store for many (Cano and Payo, 1997). Details of home oxygen for children are given in RCP (1999).

EMERGENCIES IN THE NEONATAL UNIT

Sudden hypoxaemia

A drop in S_aO_2 may be followed by bradycardia and fighting the ventilator. Manual ventilation with gentle pressure should be carried out until the cause is found. If sudden, hypoxaemia could be caused by a displaced or blocked tracheal tube (unchanged CVP), or barotrauma (↑ CVP).

Apnoeic attacks

Respiratory pauses are physiological lulls in respiration. They may presage *pathological apnoea*, which lasts more than 20 seconds and may be associated with hypoxaemia. Pathological apnoea may be due to brain-stem immaturity, upper airway and chest wall instability or gastro-oesophageal reflux (Ewer *et al.*, 1999). If this does not resolve spontaneously, the baby will need gentle stimulation (Holditch, 1994) or intubation.

Pneumothorax

Any sudden deterioration in the condition of a ventilated infant raises suspicions of barotrauma. A pneumothorax is evident on X-ray, but clinical signs can be elusive. Breath sounds may still be present because sound is transmitted from the unaffected lung. A tension pneumothorax causes bradycardia and a plunge in cardiac output.

Cardiorespiratory arrest

Most cardiorespiratory arrests in infants and children are of respiratory origin. Establishing a patent airway by head positioning may prevent progress of the event. Care should be taken to avoid pressing on the soft tissues under the chin or over-extending the neck because this may occlude the trachea.

If an oropharyngeal airway is required, it is not turned upside down for insertion, as in the adult. The correct size reaches from the corner of the mouth to the angle of the jaw. In the NICU, oxygen by bag and mask is available. If mouth-to-mouth breathing is necessary, both mouth and nose should be covered with the rescuer's mouth, and gentle puffs given. Parents are best taught to use the nose only, as they may obstruct the airway when attempting to seal the mouth (Wilson-Davis, 1997). The Heimlich manoeuvre is contraindicated in children under the age of 3.

Cardiac arrest is usually systolic and due to

respiratory arrest. When assessing for responsiveness, shaking should be avoided. If artificial ventilation does not restore the heart beat, chest compression is started by encircling the chest with both hands and squeezing the mid-sternum to a depth of 2 cm, with the thumbs at one finger's breadth below an imaginary line joining the nipples, and ensuring that the chest fully re-expands between compressions. The ratio of breaths to compressions is 1:5 in infants and children, repeated 20 times per minute. The easiest pulse to locate is the brachial pulse on the inside of the upper arm (Zideman, 1994).

MINI CASE STUDY: JW

This 7-year-old was admitted after fitting. Identify what has happened and answer the questions.

HPC: seizure this afternoon → fall on to head → vomited → respiratory distress → intubated and ventilated.
CT scan and clinical assessment shows no neurological damage.
Ventilated on SIMV with F_IO_2 of 0.4.
P_aO_2 22.27 kPa (167 mmHg), P_aCO_2 4.1 kPa (30.8 mmHg), pH 7.44, HCO_3^- 21.8.
Sedated on midazolam.
Extubation planned for tomorrow morning.
Suction non-productive.

Questions

1. Auscultation (Figure 16.7a)?
2. Percussion note?
3. Analysis?
4. Problems?
5. Goal?
6. Plan?
7. Outcome (Figure 16.7b)?

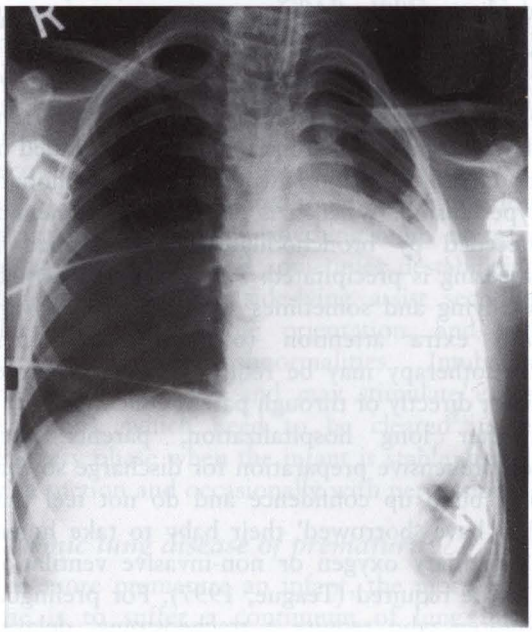


Figure 16.7a JW.

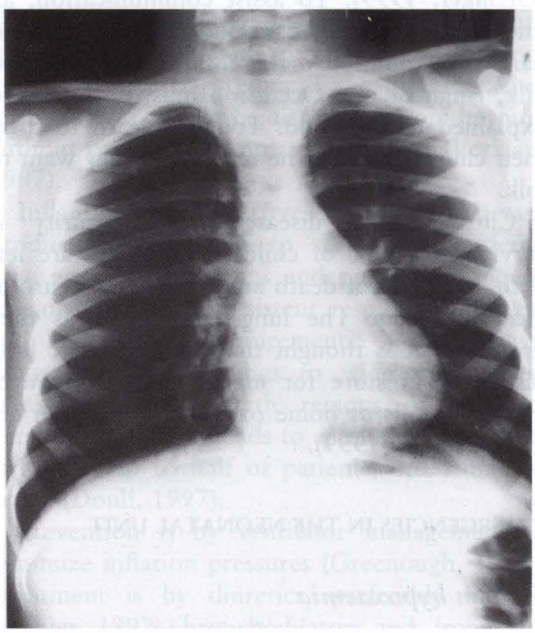


Figure 16.7b JW.

RESPONSE TO MINI CASE STUDY

1. Auscultation

↓ breath sounds L midzone and L lower zone, bronchial breathing L midzone and L lower zone.

2. Percussion note

Dull L midzone and L lower zone.

3. Analysis

Atelectasis and consolidation L lung, probably due to aspiration.

4. Problems

↓ lung volume on L,
Inability to clear secretions.

5. Goal

L lung cleared and inflated by morning.

6. Plan

Saline instillation to left lung
Postural drainage
Manual hyperinflation
Percussion
Suction.

7. Outcome

Goal achieved.

LITERATURE APPRAISAL

Do you agree?

> ***Unless it can be shown that the fetus has a conscious appreciation of pain ... the responses to noxious stimulation must still essentially be reflex.***
>
> Do fetuses feel pain? *Br. Med. J.* (1996), 313, 795–799.

RESPONSE TO LITERATURE APPRAISAL

It is not scientific to make an assertion of 'fact' by assumption.

It is not scientific to make an impossible statement. A foetus by definition cannot be demonstrated to consciously appreciate a sensation.

Perhaps unreferenced facts and impossible statements enable the author (and reader?) to feel more comfortable in not having to contemplate a foetus in pain.

RECOMMENDED READING

Alderson, P. (2000) The rise and fall of children's consent to surgery. *Paediatr. Nurs.*, 12(2), 6–8.

Arthur, R. (2000) Interpretation of the paediatric chest x-ray. *Paed. Resp. Rev.* 1, 41–50.

Askin, D. F. (1997) Interpretation of neonatal blood gases. *Neonat. Network*,16(5), 17–21.

Beeby, P. J. (1998) Short- and long-term neurological outcomes following neonatal chest physiotherapy. *J. Pediatr. Child Health*, 34, 60–62.

Briassoulis, G. C. (2000) Air leaks from the respiratory tract in mechanically ventilated children with severe respiratory disease. *Pediatr. Pulmonol.*, 29, 127–134.

Cantagrel, S. and Cloarec, S. (1999) Consequences of pulmonary inflations (sighs) on cerebral haemodynamics in neonates ventilated by high-frequency oscillation. *Acta Paediatr.*, 88, 1004–1008.

Chaisupamongkollarp, T. (1999) Prone position in spontaneously breathing infants with pneumonia. *Acta Paediatr.*, 88, 1033–1034.

Dalton, H. J. and Heulitt, M. J. (1998) Extracorporeal life support in pediatric respiratory failure. *Respir. Care*, 43, 966–977.

Eber, E. (1997) Bronchoalveolar lavage in children. *Eur. Respir. Monogr.*, 5, 136–161.

Grasso, M. C., Button, B. M. and Allison, D. J. (2000) Benefits of music therapy as an adjunct to chest physiotherapy in infants and toddlers with CF. *Ped. Pulmonol*, 29, 371–381.

Gross, G. W. (1999) Chest imaging in the neonatal and pediatric ICU. *Respir. Care*, 44, 1095–1126.

Hemmila, M. R. and Hirschl, R. B. (1999) Advances in ventilatory support of the pediatric surgical patient. *Curr. Opin. Pediatr.*, 11, 241–248.

Heulitt, M. J. and Bohn, D. (1998) Lung-protective strategy in pediatric patients with ARDS. *Respir. Care*, 43, 952–960.

Jacobson, B. (1998) Obstetric care and proneness of offspring to suicide as adults. *Br. Med. J.*, 317, 1346–1349.

Joad, J. P. (2000) Smoking and pediatric respiratory health. *Clin. Chest Med.*, **21**, 37–46.

Lam, W. H. (1999) Mechanism and management of paediatric head injury. *Care Crit. Ill*, 15(3), 95–98.

Lemons, J. A., Blackmon, L. R. and Kanto, W. P. (2000) Prevention and management of pain and stress in the neonate. *Pediatrics*, **105**, 454–461.

Long, T. (1995) A critical appraisal of positioning infants in the neonatal intensive care unit. *Phys. Occup. Ther. Pediatr.*, **15**(3), 17–29.

Marcus, C. L. (2000) Pathophysiology of childhood obstructive sleep apnoea. *Respir. Physiol.*, **119**, 143–154.

Oberwaldner, B. (2000) Physiotherapy for airway clearance in paediatrics. *Eur. Respir. J.*, 15, 196–204.

O'Callaghan, C. and Barry, P. W. (2000) Asthma drug delivery devices for children. *Br. Med. J.*, 320, 664.

Rennie, J. M. and Bokhari, S. A. (1999) Recent advances in neonatology. *Arch. Dis. Child. Fetal Neonat. Ed.*, **81**, F1–F4.

Russell, R. I. R. (1998) Weaning from mechanical ventilation in children. *Care Crit. Ill.*, **14**, 121–123.

Shekerdemian, L. (1999) Cardiovascular effects of mechanical ventilation. *Arch. Dis. Child.*, 80, 475–480.

Sweeney, J. K., Heriza, C. B., Reilly, M. A. and Smith, C. (1999) Practice guidelines for the physical therapist in the neonatal intensive care unit. *Pediatr. Phys. Ther.*, **11**, 119–132.

Törnage, C. J. (1999) First week kangaroo care in sick very preterm infants. *Acta Paediatr.*, 88, 1402–1404.

Weir, E. C., Burrow, J. G. and Bell, F. (2000) Existing methods of pain evaluation in children. *Br. J. Ther. Rehab.*, 7, 418–423.

Young, A. E. R., Manara, A. R. and Burd, D. A. R. (1995) Intensive care management of the child with severe burns. *Care Crit. Ill.*, **11**(3), 93–97.

17 Evaluation of respiratory physiotherapy

Summary

Introduction

> *Respiratory therapy is one of those technical orphans that grew up eluding the cold eye of scientific enquiry.*
>
> Hughes, 1980

If a patient who is receiving physiotherapy gets better, is this due to the physiotherapy, the physiotherapist or divine intervention? The credibility of respiratory physiotherapy is being much challenged in the pages of learned journals and on the shop floor. This we must welcome.

With tongue in cheek, Stiller and Munday (1992) commented that 'Some may question the need for studies, given the generally accepted and extensive use of chest physiotherapy'. When this approach is taken seriously, it inhibits progress.

Evaluation is notoriously difficult because literature is scarce and ambiguous, 'chest physiotherapy' is poorly defined, and variables such as simultaneous medical input and the placebo effect upset results. It is also complicated by evidence that:

- one-third of patients adhere to treatment
- one-third reject it outright
- one-third accept it but get it wrong (Lloyd, 1998).

Effective physiotherapy requires scrutiny of the research, development of standards from the research, audit to integrate this and other evidence into practice, and a system of continuous evaluation so that the process does not become a luxury to be tagged on at the end if there is time.

> *Only about 15% of all contemporary clinical interventions are supported by objective scientific evidence that they do more good than harm.*
>
> White, 1988

Definitions

Benchmark: agreed criterion by which a practice can be judged (Barnard and Hartigan, 1998, p. 119).

Clinical governance: framework to improve patient care using evidence-based guidelines produced by the National Institute for Clinical Effectiveness. Includes audit, accountability and patient satisfaction (DoH, 1999). If breached, litigation can be instigated. All breaches

should be reported to the line manager in writing.

Competence: presense of the skills and abilities required for safe and effective practice.

Criteria: list of what should happen for a standard to be achieved. This is checked against clinical records during a criterion-based audit (Barnard and Hartigan, 1998, p. 27).

Evidence-based practice: clinical decision-making based on the systematic search for, appraisal of and use of current evidence. Where there is lack of objective evidence, clinical expertise can be included in the definition (Partridge, 1996; Parmar, 1998). Clinical expertise is a tool to be nurtured mindfully, not used as anecdotal justification.

Guideline: written framework in which scientific knowledge is analysed and translated into a usable format to assist clinical decision-making (Harrison, 1998). Requires references, explanation of reasoning, and grading of recommendations and quality of the evidence. Sometimes suffers from lack of consensus.

Outcome measure: subjective or objective change due to physiotherapy input. Examples of positive outcomes are reduced breathlessness or improved exercise tolerance. Outcome measures must be appropriate, reliable, valid and responsive (Barnard and Hartigan, 1998, p. 30).

Peer review: review of the work of an individual by those who are equal in grade and speciality (Barnard and Hartigan, 1998, p. 28).

Protocol: precise, legally binding instructions developed from a guideline (Jacyna, 1992).

Standards: see p. 452.

RESEARCH

> ***Understand information you hear with the reasoning of responsibility, not the reasoning of the reporter.***
>
> Hadrat Ali, 598–661

Definitions

- *Case reports* provide anecdotal evidence to generate new ideas (Ernst, 1995).
- *Descriptive research* combines description of a certain patient population, discussion of physiotherapy management and presentation of the associated literature.
- *Pilot studies* are required in advance of more ambitious projects in order to refine methodology and uncover potential flaws.
- *Single case studies* are a quantitative approach in which treatment periods are alternated with non-treatment periods. Patients act as their own controls and a large homogeneous group of patients is not needed (Sim, 1995).
- *Controlled trials* include a group of subjects who do not receive the treatment under investigation so that the outcome is attributable to the topic being investigated.
- *Randomized controlled trials* allocate subjects randomly so that any difference in outcome can be explained only by the treatment (Roberts, 1998).
- *Blind trials* keep subjects in the dark about which side of the study they are participating in, so that the placebo effect is minimized.
- *Double-blind trials* prevent investigators knowing the subjects' allocation.

Secondary research comprises (Bury and Mead, 1998, p. 146):

- *reviews*, which summarize the results from several studies and draw conclusions
- *systematic reviews*, which select and critically appraise relevant research and analyse the data
- *meta-analysis*, which pools studies of similar design.

Problems for respiratory physiotherapy

When putting results of research into practice, absence of evidence is not evidence of absence. Research in respiratory physiotherapy comes with several obstacles:

- a host of confounding factors including input from other disciplines
- lack of defined categorization in physiotherapy compared to medicine

- shortage of time, money or support
- ethics.

Ethical considerations have, by definition, no neat answer. As suggested in the *British Medical Journal*: 'Can we insist on evidence of effectiveness from randomized controlled trials for support services which are of such evident human desirability as to render their deliberate withholding difficult or unethical?' (Keeley, 1999).

> ***Why does irrational clinical behaviour, such as clinging to practices shown to be of little value, persist in the face of ample contrary evidence?***
>
> Carr, 1996

LITERATURE APPRAISAL

> ***Why do kamikaze pilots wear helmets?***

A questioning and indeed a suspicious mind is necessary when reading articles because research can prove or disprove almost anything. The most prestigious journals publish articles based on false premises, poor design and with inaccurate conclusions. Researchers may be biased towards proving their own ideas. Editors may be reluctant to publish negative results. Beware of literature that contains:

- extrapolation of results from medical research, e.g. lengthy manual ventilation used by anaesthetists is not the same as the brief manual hyperinflation used by physiotherapists
- extrapolation of results from healthy young volunteers to people who are ill, e.g. dynamic hyperinflation simulated by using CPAP on normal subjects
- extrapolation of results from animals to humans, e.g. dogs have a different chest shape and their pleural space communicates bilaterally
- assessment of more than one technique in one study
- lack of distinction between correlation and causation
- unreferenced factual statements
- interpretation without consideration of alternative explanations
- uncontrolled variables
- jargon obscuring clarity
- lack of a clear aim
- inadequate definitions, e.g. 'conventional chest physiotherapy', so that reproducibility is impossible
- physiotherapists used as handmaidens to collect data rather than as designers of the study
- conclusions that 'chest physio-therapy was of no value', instead of the inelegant but accurate: 'postural drainage with percussion in this way for this amount of time for these patients showed no evidence of improved mucociliary clearance/greater quantity of sputum/reduced airflow resistance'.

Examples are given below.

- Torrington *et al.*, (1984) imposed 4-hourly IPPB, 4-hourly incentive spirometry, 2-hourly deep breathing and 2-hourly nebulization on obese postoperative patients. The authors expressed surprise that additional 4-hourly PD and percussion increased discomfort, fever and cost. Nor did this onslaught reduce atelectasis. The study has been much quoted to claim that postoperative physiotherapy is unnecessary.
- Shapiro *et al.* (1992) took a leap of faith when they concluded that 'inspiratory muscle rest confers no benefit' after encasing patients in negative-pressure body suits overnight. Patients were too uncomfortable to sleep and found a visit to the toilet an ordeal. Perhaps they should have tried a positive-pressure non-invasive ventilator.
- Ng and Stokes (1992) attempted to evaluate respiratory muscle activity during 'unilateral' chest expansion, but did not distinguish inspiratory and expiratory muscles, used 'subjective observation' to judge this notoriously ambiguous manoeuvre, and did not

explain the physiology or implications in relation to aeration of the lung.

- Misuse of references is common. Chuter (1990) claimed that 'diaphragmatic movements ... improve ventilation of the lower lung zones', quoting a reference that was using deep breathing rather than diaphragmatic breathing.
- Weissman *et al.* (1984) did not define chest physical therapy in a paper associating it with major haemodynamic and metabolic stress.
- Researchers sometimes succumb to the temptation to use a plethora of interventions. Alison *et al.* (1994) claimed triumph for physical therapy from an uncontrolled trial in which people with an exacerbation of cystic fibrosis benefited from 'rest, intravenous antibiotics, physical therapy, high-calorie diet and regular medical review'. Maybe it was the rest that was the beneficial agent, maybe the medical review, maybe just natural recovery from an exacerbation.

All who drink of this remedy recover in a short time, except those whom it does not help, who all die. Therefore it is obvious that it fails only in incurable cases.

Galen, 2nd century

STANDARDS

The perception is, if chest physiotherapy doesn't help, it won't hurt.

Eid, 1991

Evaluation needs standards against which outcome can be measured. Standards define the expected level of performance. They must be measurable, understandable, desirable and achievable. They are usually subject to staffing levels. Standards are only useful if audited and if audit lends to appropriate change in practice. Staff are motivated by setting their own standards. Tables 17.1 and 17.2 give examples.

Other standards could include identification of which surgical patients are to be assessed, time between referral and assessment for acute and non-acute patients, agreement with patients of plans and goals, explanation to patients about limitations and risks of treatment, provision for patient self-management and follow-up, liaison with the multidisciplinary team, and maintenance of evidence-based practice by training, supervision, case presentations, sharing of information from courses and a journal club.

Table 17.1 Standards for mobility: all inpatients

1. All patients mobilize daily unless:
   - it is unsafe,
   - it is impossible for practical reasons, e.g. unnavigable lines, uncontrolled pain.
2. For patients who do not mobilize, the reason is documented, e.g. practical or safety contraindication, patient refusal (land action taken), staff shortage.
3. The daily exercise programme is documented:
   - in notes, if given verbally to patient,
   - as handout for patient, copied in notes.
4. Documentation demonstrates progression.

OUTCOME EVALUATION

The distinguishing characteristic of the professional is that he does what he does intelligently, not routinely.

Ten Hove, 2001

The reference point for evaluation is the outcome of treatment. Employers now require physiotherapists to justify their time in terms of outcomes and cost (Dalley, 1999). Subjective evaluation is by listening to patients and using questionnaires. Objective evaluation is by a selection of the methods used in assessment. Like research, the tools for outcome evaluation are based on reliability, validity and attribution. Unlike research, clinical evaluation can include the complete package of treatment as well as individual components (Dalley, 1999).

Subjective measurement

Patient and physiotherapist may have different views of success. Reduced symptoms may not affect patient wellbeing, or treatment may improve function but not affect symptoms

Table 17.2 Some respiratory standards, criteria and measurement

Standard	Criteria	Measurement
1. Each physiotherapy session is evaluated	Written evidence of assessment and evaluation in physiotherapy notes, including response to treatment and appropriate changes in treatment. Outcomes recorded. Discharge summary written.	4-monthly notes audit.
2. Treatment plan is suited to the patient's problems	There is written evidence that the treatment plan relates to the patient's assessment.	4-monthly notes audit.
3. Each member of the respiratory team is responsible for clinical evaluation.	There is evidence of regular evaluation of clinical practice.	For juniors, 1 hour review with senior weekly. For seniors, peer support quarterly.
4. All patients requiring emergency physiotherapy out of normal working hours receive safe and effective treatment.	Relevant staff have ongoing training.	Twice-yearly training by senior staff.
	Staff have access to written guidelines for respiratory problems commonly encountered out of hours.	On-call information in induction pack.
	Staff work a minimum of one Sat/Sun three times a year.	Audit of rotas.
	Staff have access to advice from specialist staff.	Yearly on-call audit.
	Service use is documented.	Yearly audit.
	Referrers are provided with written protocol.	6-monthly provision of on-call protocol to referrers.
	Inappropriate referrals are followed up.	Yearly audit.

(Duckworth, 1999). Subjective outcomes are based on the patient's priorities. Patient surveys can be created from quality of life scales or assessment questionnaires (e.g. pp. 218). They should guarantee anonymity and confidentiality.

Objective measurement

Obstacles to measuring outcome include the following:

- S_aO_2 and other measurements vary with factors other than physiotherapy.
- Postoperative atelectasis may be self-limiting.
- Mouthpieces may interfere with what they are measuring.
- Patients and other members of the health team may be seduced by mechanical aids that make exotic noises.
- Quality of treatment cannot be assessed from the number and length of treatments.
- Patients with chronic respiratory disease are notoriously vulnerable to suggestion.
- Respiratory disease is often complicated by multipathology.

Some measurements are valid if taken in the context of the full clinical picture:

- ↑ oxygenation, i.e. ↑ P_aO_2, S_aO_2 or $S\bar{v}O_2$
- ↑ ventilation, i.e. ↓ P_aCO_2
- increase or maintenance of exercise tolerance, e.g. shuttle test or functional activities
- ↓ pain
- ↑ independence, e.g. ADL
- ↑ well-being, e.g. quality of life scores
- increase or maintenance of lung volume (p. 163)
- clearance of secretions (p. 209)
- ↓ work of breathing (p. 181)
- lack of deterioration.

Box 17.1 is an example of measuring the outcome for on-calls. This provides guidance for on-call staff, an indication of the adequacy of training and a means of monitoring the appropriateness of the call. Lack of improvement in the patient does not necessarily mean an inappropriate call out.

Box 17.1 On call record

Patient ..... Date .....

Physiotherapist .....

Called by .....

Diagnosis .....

Reason for referral .....

Problems encountered .....

	Pretreatment	Post-treatment	N/A
Breathing pattern BS AS S_aO_2 CXR Other			

Action	✔ or ✖		
Did not attend		**Attended**	
Inappropriate (state reason)	Advice only	Appropriate	Inappropriate (state reason)

BS = breath sounds; AS = added sounds; N/A = not applicable.

Successes folder

Good research on respiratory physiotherapy is scarce, and it is wise to build up a record of objective proof of success, e.g. before-and-after auscultation, S_aO_2 or copies of X-rays. This is a crude measure that does not reflect the effects of education and rehabilitation on quality of life, but it comes in handy if challenged by budget-holders.

COST EFFECTIVENESS

> *Do no harm – cheaply.*
>
> Hughes, 1980

Cost effectiveness is allied to clinical effectiveness because time is freed up for further input. Measures to save time include:

- handouts for patients to reinforce education
- avoiding treatment that is not evidence-based
- highlighting relevant events in the physiotherapy notes to assist weekend and handover staff (this is also a safety factor for busy times)
- mobility charts to help involve nursing staff with rehabilitation
- assistants supported and valued
- journal club to screen more journals than one person can read

Box 17.2 Physiotherapy assessment referrals (from Suzanne Roberts, as used at Whittington Hospital, London)

Week *Ward* *Physiotherapist* *Bleep*

Date	Name of patient	Referrer (print name and designation)	Physiotherapy problem for which assessment is requested

- educational material, homemade or from organizations (Appendix C), sent to respiratory outpatients before their first appointment
- to assist the ward report, written referral sheet on wards, pinned up at the nurses' station, to be filled out by referring staff and checked daily by the physiotherapist, e.g. Box 17.2
- information for nursing and medical staff about appropriate referrals, by problem or by condition, e.g. Box 17.3
- follow-up telephone calls to outpatients for motivation and support when face-to-face contact is not essential. Telephone consultation is becoming increasingly part of the training of hospital staff in North America (Oberklaid, 1998).

Extended care practitioners improve efficiency by being trained in skills such as taking capillary blood gases, thus being able to progress treatment without waiting for a doctor. Physiotherapists do not improve efficiency by learning techniques that are not physiotherapy skills and do not save physiotherapy time.

It is cost-effective to spend a few moments writing individual daily programmes for patients if this motivates them to do their daily practice. It is cost-effective to reduce a patient's need for medication. It is cost-effective to reduce the need for other services. Heijerman (1992) has shown that, following rehabilitation, some people with cystic fibrosis are no longer oxygen-dependent nor need consideration for transplantation.

An on-call service is cost-effective if it prevents deterioration or avoids the need for more time-consuming intervention. It is not cost-effective if non-respiratory physiotherapists have not developed the competencies to deal with critically ill patients. Nicholls (1996) puts the case for the credibility of respiratory physiotherapy: 'We cannot claim to offer 24-hour care for patients while working only eight of them'.

Short-termism must not intrude on cost-effectiveness. Prevention and rehabilitation are central to efficient respiratory care.

THE AUDIT CYCLE

> ***People do not resist change. They resist being changed.***
>
> Lloyd, 1998

Research and patient feedback tell us the right thing to do. Audit tells us if we are doing the right thing right. It entails clinically led peer review, which systematically analyses practice and outcome against agreed standards, then modifies practice where indicated (Sealey, 1999).

Protected time, simple topics and minimal paperwork are advised. Liaison with the hospital's clinical audit department is a useful first step. The topic chosen should have the potential for improvement and be largely responsive to physiotherapy, e.g.:

- percentage of problems resolved
- percentage of patients receiving discharge advice

Box 17.3 Criteria for respiratory physiotherapy

CRITERIA BY PROBLEM

Sputum retention
Patients who have sputum but are unable to clear their chests independently, e.g. due to weakness, drowsiness, exhaustion.
Note 1: if a patient is productive of sputum, this may be a good sign (they can clear their own chests) or a bad sign (they have excess secretions with potential for infection, e.g. bronchiectasis).
Note 2: if a patient is non-productive, this may be a good sign (no secretions) or a bad sign (sputum retention).

Loss of lung volume
Patients who have atelectasis, e.g. post-op.

Breathlessness
Patients who have acute or chronic breathlessness.

Worsening gas exchange
Patients who have deteriorating blood gases or oxygen saturation.

CRITERIA BY CONDITION

Urgent referral
- person who has *aspirated*.

Necessary referrals
- person with *fractured ribs* (adequate analgesia required).
- person with *COPD*, *bronchiectasis*, *cystic fibrosis* or *pneumonia*.
- person with *restrictive disease*, e.g. fibrosing alveolitis.

Usual referrals
- person with *asthma*, unless s/he is mobile, breathing comfortably and has access to an education programme.
- person with *lung abscess*, unless abscess is responding to antibiotics and does not require postural drainage for clearance.
- person with a *chest infection*, unless s/he is mobile and does not have difficulty clearing secretions.
- person with *pleural effusion* or *pneumothorax*, unless s/he is mobile, has adequate gas exchange and no chest drain.

Unnecessary referral
- person with *pulmonary oedema*, unless s/he has another physiotherapy problem.

- percentage of referrals or call-outs considered appropriate
- percentage of surgical patients discharged with preoperative function.

Methods of measurement can be chosen from the outcome or assessment sections of this book. If the full audit cycle is not completed, the exercise is wasted. A typical notes audit would comprise the cycle in Figure 17.1.

The following is an example of a biannual postoperative audit:

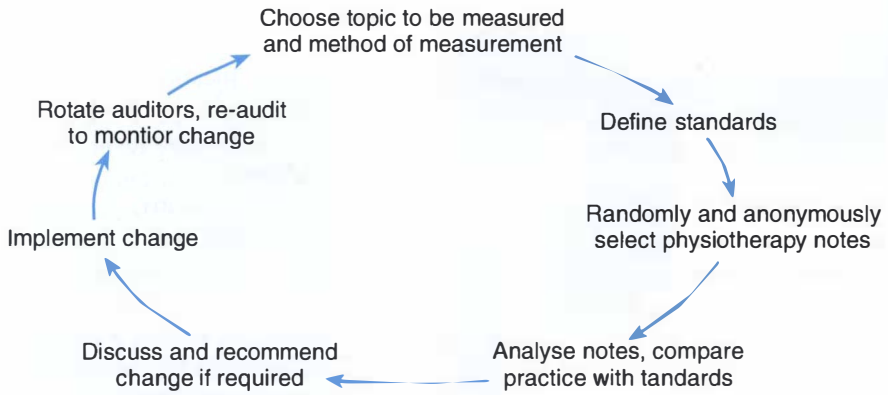


Figure 17.1 Notes audit cycle.

Standard: Patients will be able to climb one flight of stairs on their fourth postoperative day.
Patients: all surgical patients admitted in March and September who are able to climb one flight of stairs preoperatively.
Method: stairs assessment on fourth postoperative day.
Audit meeting: assess notes, identify cause of any shortfall, recommend change, agree who is to be next auditor and date of the next meeting to monitor change.

If it is felt that staff shortage is slowing patient discharges, this could be audited in consultation with the hospital discharge officer. Box 17.4 shows a method of collecting this information.

Box 17.4 Inpatient delayed discharges

Patient . . . . . .

No . . . . . .

Ward . . . . . .

Date considered due for discharge . . . . . .

Cause of delayed discharge:
- social services/home circumstances: Y/N
- staff shortage: Y/N

If yes, which discipline. . . . . .

- other. . . . . .

Date discharged . . . . . .

EDUCATION AND CONTINUING EDUCATION

> ***There are no known facts, only the present theory of the day***
>
> Howell, cited byConway, 1992a

Continuing competency

Juniors abd non-respiratory seniors doing out-of-hours work require updating of their technical, handling and problem-solving skills. Documentation should demonstrate regular training and assessment in techniques and clinical reasoning.

Needs of students and juniors

> ***When educators are more humanistic in their training of students, the students become more humanistic in their care of patients.***
>
> Williams and Deci, 1998

Learning is improved by autonomy. An autonomous environment improves problem-solving and achieves a more humanistic approach in learners (Williams and Deci, 1998). It is the human qualities of supervisors that are often considered of equal or more importance than clinical skills (Neville and French, 1991). Students and juniors need:

- clarification of expectations
- assistance in setting feasible objectives and assessing whether these are met
- encouragement to work creatively and not become a clone of their senior
- regular contact with their senior (CSP, 1998) for feedback, case discussions, trouble-shooting and assessing the balance of guidance and responsibility
- praise when due (Jackson, 1999)
- correction in a way that does not undermine confidence or belittle them in front of patients
- space for reflection
- enjoyment in their work
- for senior students, consultation on how closely they want to be supervised (Onuoha, 1994)
- encouragement to learn from patients, e.g. Appendix D
- an information folder, e.g. Box 17.5.

Box 17.5 Respiratory team information

- Wards: locations, specialities, phone numbers, crash trolleys, meetings
- Medical and surgical teams and bleep numbers
- Referral systems
- Team meetings
- Pre-tutorial reading material
- Junior responsibilities
- Guidelines, e.g. tracheostomy care, use of equipment, infection control
- Assessment sheets, mobility charts
- Patient handouts, e.g. relaxation, pre/post op information, ACB/AD, breathless positions, exercise sheets
- Previous junior projects
- End-of-rotation questionnaires

Seniors need to ensure competency in the respiratory service. Weekend teams should contain a mix of experienced and inexperienced staff, if numbers allow. Novices need the opportunity to shadow the respiratory senior before weekend duties, and access to a mentor during on-call duties. Learning is facilitated by positive role modelling from a supervisor with the following qualities:

- clear setting of priorities
- enthusiasm, honesty and commitment
- respect for juniors so that they in turn respect their patients
- tolerance of a wide range of normality
- avoidance of labelling patients as difficult or not liked
- willingness to say 'I don't know'
- constructive relationships with medical and other staff
- ability to coax the nervous patient, soothe the fearful and encourage the weary.

When asking a patient's permission for student attendance, it is advisable that the student is not present, so that the patient feels free to refuse. Permission should also be sought for the student to read the medical notes

(Wilkie, 1997), and patients advised, when appropriate, of the student's gender.

Clinical practice is not enough to develop empathy (Thomson *et al.*, 1997) and sensitivity needs to be facilitated rather than blunted, e.g. if there has been a ward round in which a patient's needs have been ignored, debriefing is required rather than acceptance. Supervisors need to maintain awareness lest juniors feel obliged to conform.

Our finest clinicians should analyse their intuitive process so that they can pass on how they recognize subtle changes in a breathing pattern, sense a patient's motivation or adjust their treatment in response to barely perceptible clues.

Self assessment

> ***You may get used to hearing about pain, so don't let yourself get immune to it . . . when you get to that point, you have to stop and re-evaluate yourself.***
>
> Askew *et al.*, 1998

We are the most available and abiding judges of our own work, aided by self-questioning:

- Am I allowing myself to get swamped with acute respiratory work and not tackling prevention or rehabilitation?
- Do I favour patients who are appreciative and co-operative while neglecting those who are demanding or depressed?
- Have I achieved the appropriate balance between patients' needs and my professional development?
- How do I handle my mistakes?

Continuing education lays the foundation for lifelong self-evaluation. It also provides the opportunity for seniors to show that compassion is fundamental to effective respiratory care, not an old-fashioned, unscientific luxury reserved for the naive and uninitiated.

> ***Sometimes learning requires courage. To become a learner is to become vulnerable.***
>
> Berwick, 1991

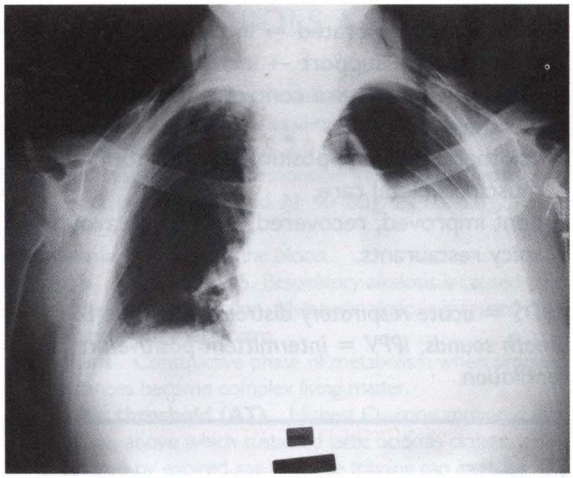


Figure 17.2 Mr FF.

MINI CASE STUDY: MR FF

What has happened to the lungs of this 33-year-old man admitted with food poisoning after eating a spicy Mexican meal?

HPC: Vomiting → back pain → collapse.
Subjective: SOB.

Questions (Figure 17.2)

1. Auscultation?
2. Percussion note?

RESPONSE TO MINI-CASE STUDY

Answers

Vomiting → ruptured oesophagus → empyema → development of gas-forming organism → pneumothorax.

1. **Auscultation**: BS ↓ L upper zone
2. **Percussion note**: hyperresonant L upper zone.

Progress

Chest tube drained foul-smelling liquid.

Blood gases deteriorated → intubation + IPPV with pressure support → increasing airway pressure → pressure control initiated.
ARDS developing.
Physiotherapy: prone positioning, monitoring, musculoskeletal care.
Patient improved, recovered, decided to avoid spicy restaurants.

ARDS = acute respiratory distress syndrome; BS = breath sounds; IPPV = intermittent positive pressure ventilation.

LITERATURE APPRAISAL

At last, logic:

> ***... when children share a room (which implicates both exposure to other children and lower socioeconomic status)....***
>
> *Br. Med. J.* (1989), 299, 949

RESPONSE TO LITERATURE APPRAISAL

To cheer us up, this shows lateral thinking in interpreting results.

RECOMMENDED READING

Bithell, C. (2000) Evidence-based physiotherapy. *Physiotherapy*, 86, 58–60.

Case, K., Harrison, K. and Roskell, C. (2000) Differences in the clinical reasoning process of expert and novice cardiorespiratory physiotherapists. *Physiotherapy*, 86, 14–21.

CSP (1997) *Guidelines for Good Practice for the Education of Clinical Educators*, Information paper CPD14, Chartered Society of Physiotherapy, London.

CSP (1999) Clinical effectiveness. *Frontline*, March supplement, Chartered Society of Physiotherapy, London.

CSP (1999) How to get a research grant. *Frontline*, **Suppl.1**(3), 8–9.

CSP (1999) Setting up a research programme. *Frontline*, **Suppl.**1(3), 16–17.

Culham, E. (1998) Evidence-based practice and professional credibility. *Physiother. Ther. Pract.*, 14, 65–67.

Goldhill, D. (2000) Clinical guidelines in intensive care. *Care Crit. Ill*, 16(1), 2–4.

Hartigan, G. (1995) Choosing a method for clinical audit. *Physiotherapy*, 81, 187–188.

Johnson, S. C., Dodd, M. E. and Evans, W. (1998) Improving the efficiency of medical respiratory referrals in a DGH. *Physiotherapy*, 84, 584–589.

Kitchen, S. S. (2000) Audit: does it really improve care? *Physiotherapy*, 86, 226–228.

McGovern, M. C. and Stewart, M. C. (1999) Drawing up clinical guidelines. *Int. J. Clin. Pract.*, 53, 118–120.

Morrel, C. and Harvey, G. (1999). *The Clinical Audit Handbook,* Baillière Tindall, London.

Murray, P. (1999) Fundamental issues in questionnaire design. *Accid. Emerg. Nurs.*, 7, 148–153.

Partridge, C. J. (1998) Research in cardiopulmonary physiotherapy, in *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, (eds B. A. Webber and J. A. Pryor), Churchill Livingstone, Edinburgh.

Pieri-Davies, S., Ewart, K. and Boden, K. (1994) On-call respiratory issues threaten patients (letter). *Frontline*, July 7.

Price, A. M. and Chalker, M. (2000) Our journey with clinical supervision in an intensive care unit. *Intens. Crit. Care Nurs.*, 16, 51–56.

Rogers, R. and Perren, P. (1999) Truth or illusion: evidence-based practice in the real world. *Br. J. Ther. Rehab.*, 6, 275–280.

Shekelle, P. G., Woolf, S. H. and Eccles, M. (1999) Developing guidelines. *Br. Med. J.*, 318, 593–596.

Sim, J. (1989) Methodology and morality in physiotherapy research. *Physiother.*, 75, 237–243.

Sim, I. and Hlatky, M. A. (1996) Growing pains of meta-analysis. *Br. Med. J.*, 313, 702–703.

Tobin, A. and Judd, M. (1998) Understanding the barriers. *Physiotherapy*, 84, 527–529.

Wallis, C. and Prasad, A. (1999) Who needs chest physiotherapy? Moving from anecdote to evidence. *Arch. Dis. Child.*, 80, 393–397.

Williams, S. and Harrison, K. (1999) Physiotherapeutic interactions: a review of the power dynamic. *Phys. Ther. Rev.*, 4, 37–50.

GLOSSARY OF ABBREVIATIONS, DEFINITIONS, SYMBOLS AND VALUES

Values in [square brackets] are American. Most values are approximate.

If the definition is not here, see the index.

L = *litre.*
N = *normal.*
Δ = *change.*
Dot over symbol = *value per unit time, e.g.* $\dot{V}O_2$ = *oxygen consumption.*
Bar over symbol = *mean value, e.g.* $\bar{v}$, *mixed venous.*
μ–Micro- prefix indicating 10^{-6}, i.e. $\times 0.000001$.
μm See Micrometer.
$\Delta V/\Delta P$ Change in volume in response to change in pressure, i.e. compliance.
A Alveolar, e.g. P_AO_2.
a Arterial, e.g. P_aO_2.
AAA Abdominal aortic aneurysm.
ABG Arterial blood gas.
ABPA Allergic bronchopulmonary aspergillosis.
Absorption atelectasis Alveolar collapse due to either resorption of air distal to a bronchial obstruction or excess administration of O_2, which is then absorbed.
ACB Active cycle of breathing.
ACE inhibitors Angiotensin converting enzyme inhibitor drugs.
Acidaemia Acidosis of the blood.
Acidosis pH below 7.35. Respiratory acidosis is caused by malfunctioning respiration. Metabolic acidosis is caused by malfunctioning metabolism.
Acinus Portion of lung participating in gas exchange, supplied by a primary respiratory bronchiole.
ACPRC Association of Chartered Physiotherapists in Respiratory Care.
AD Autogenic drainage.
Adherence Degree to which patient behaviour coincides with clinical recommendations, also known as patient compliance but with less implication of obedience.
ADL Activities of daily living.
Adult respiratory distress syndrome Old terminology for acute respiratory distress syndrome.
Adventitious sounds Added sounds on auscultation.
Aetiology Cause.
Aerophagia Gas in the stomach.
Aerosol Suspension of solid or liquid particles, e.g. pollen, dust, smoke, mist, viruses, therapeutic aerosol for humidification and drug delivery.
Air trapping Retention of inspired gas in poorly ventilated areas of lung.
Airway (1) Path that air travels from atmosphere to alveoli; (2) device to hold natural airway open for relief of obstruction or to allow IPPV.
Airway closure Closure of small airways, common in dependent lung regions at end-expiration.
Airways resistance N: 0.5–3.0 $cmH_2O/L/s$.
Albumin Plasma protein responsible for providing most osmotic pressure in blood. N: 40–60 g/L, [4.0–6.0 g/100 mL].
Alkalaemia Alkalosis of the blood.
Alkalosis pH above 7.45. Respiratory alkalosis is caused by malfunctioning respiration. Metabolic alkalosis is caused by malfunctioning metabolism.
Anabolism Constructive phase of metabolism when simple substances become complex living matter.
Anaerobic threshold (AT) Highest O_2 consumption during exercise, above which sustained lactic acidosis occurs. Measured by expired gas. Exercise training can increase AT by 25–40%.
Anastomosis Surgical union of tubular structures.
Angina Crushing dull chest pain due to impaired blood supply to the myocardium, worsened with exertion or stress.
Angioplasty Invasive but non-surgical dilation of coronary artery stenosis, using catheter via femoral puncture, or laser.
Anoxia Synonymous with hypoxia, but implying a more complete O_2 lack.
Antibody Substance produced by interaction of antigen with body's defences. Also known as immunoglobulin.
Antigen Allergen, i.e. irritant that elicits an immune response.
AP Anteroposterior.
APACHE Acute Physiology And Chronic Health Evaluation: scoring system to measure severity of illness, using predictor variables such as diagnosis, age and physiological measures.
APTT See Clotting studies.
Apgar score Scoring system to measure birth asphyxia, using a combination of heart rate, respiratory effort, muscle tone, reflex irritability and colour.
Apneustic breathing Prolonged inspiration, usually due to brain damage.
Apnoea Absence of breathing for more than 10 seconds.
Arteriovenous oxygen difference Difference between O_2 delivered to and returning from tissues. Calculated from arterial and mixed venous blood samples. Related to metabolic rate.
Ascites Fluid in the abdominal cavity.
Aspiration (1) Inhalation of unwanted substances (e.g. gastric acid, sea water) into lungs; (2) therapeutic removal of fluid or gas from a cavity such as the pleural space.
Asystole No heartbeat.
Auscultation Use of stethoscope to hear sounds from a body cavity.
Base deficit Negative base excess.

Biot's respiration Irregular cycles of deep gasps and apnoea.
BiPAP Bilevel positive airways pressure.
BIPAP Biphasic positive airways pressure.
Bleb Collection of extra-alveolar air under visceral pleura (see *also* Bulla).
Blood culture Blood taken from a pyrexial patient to identify microorganisms.
Bohr effect Reduced availability of O_2 from oxyhaemoglobin in regions of low PCO_2.
Bohr equation Difference between end-tidal and arterial PCO_2. Calculates physiological dead space.
BOOP Bronchiolitis obliterans organizing pneumonia. Occurs secondary to lung or bone marrow transplant, drug reaction, aspiration or collagen vascular disease.
Bradypnoea Slow breathing.
Bronchiole Airway unsupported by cartilage.
Bronchomalacia Disorder characterized by degeneration of elastic and connective tissue of trachea and bronchi.
Bronchopleural fistula Communication between lung and pleura, caused by thoracic surgery, trauma, mechanical ventilation or pulmonary disease.
Bronchorrhoea Excess bronchial secretions.
Bronchospasm Abnormal contraction of smooth bronchial muscle, causing narrowing of airway.
BTS British Thoracic Society.
Buffer Substance that binds or releases hydrogen ions in order to keep pH constant.
Bulla Collection of air in lung tissue which is more than 1 cm in diameter, caused by alveolar destruction.
CABG Coronary artery bypass graft.
Cachexia Emaciation due to catabolism of body protein and fat, caused by malnutrition, malignancy, some chronic diseases.
Calcium N: 2.2–2.6 mmol/L.
Catabolism Metabolic process releasing energy and CO_2.
C_aO_2 See Oxygen content.
Cardiac enzymes Enzymes released from damaged heart muscle after myocardial infarction.
Cardiac index Cardiac output/body surface area. N: 2.5–3.5 L/min/m^2. Highest at age 10, then decreasing with age.
Cardiac output (CO or $\dot{Q}$) Blood ejected by left ventricle per minute, i.e. heart rate x stroke volume. N: 4–8 L/min at rest, up to 25 L/min on exercise.
Cardioplegia Cooling of heart to reduce metabolic rate during surgery.
Catabolism Destructive phase of metabolism, when large molecules are converted into small molecules.
Catecholamine Compound having sympathomimetic action, e.g. adrenaline.
CCF Congestive cardiac failure.
CF Cystic fibrosis.
CFA Cryptogenic fibrosing alveolitis.
Chest wall Structures outside lung that are involved in breathing movements, i.e. rib cage, diaphragm and abdominal wall.
Chylothorax Effusion of chyle from thoracic duct into pleural space.
CI See Cardiac index.
Circadian Over 24 hours.
Closing capacity Lung volume at which airway closure begins, as a result of narrowing of dependent airways as lung deflates. Rises with age as small airways narrow, equalling FRC at an average 44 years in supine and 66 years in standing.
Closing volume Closing capacity minus residual volume. Increases (becomes a greater proportion of FRC) with small airways disease, smoking, obesity and extremes of age. N: 10% of VC in young people with normal lungs. Age 65: 40% of VC.
Clotting studies
- *ACT (activated clotting time)* N: 100–140 s.
- *APTT (activated partial thromboplastin time)* N: 30–40 s.
- *Bleeding time* N: 3–9 min.
- *FDPs (fibrinogen degradation products)* N: < 10 mg/mL.
- *Fibrinogen level* N: > 150 mg/dL.
- *INR (international normalized ratio)* N: 1–1.4. Higher means increased clotting time and risk of bleeding.
- *KPTT (kaolin partial thromboplastin time)* N: < 7 s above control.
- *Platelet count* N: 140 000–400 000/mm^3. Risk of bleeding with suction: < 50 000/mm^3. Spontaneous bleeding: < 20 000/mm^3.
- *PT (prothrombin time)* N: 12–15 s.
- *PTT (partial thromboplastin time)* N: 12–30 s.

CMV (1) Controlled mandatory ventilation or conventional mechanical ventilation, (2) cytomegalovirus (virus in the herpes group).
CO (1) Cardiac output, (2) carbon monoxide.
CO_2 Carbon dioxide.
COAD Chronic obstructive airways disease.
Coagulation See Clotting studies.
Collateral ventilation Exchange of inspired gas between adjacent lung units.
Colonization Presence and multiplication of microorganisms without necessarily causing tissue damage.
Colostomy Surgical creation of opening into large bowel.
Coma Unrousable unconsciousness.
Compliance of lung Change in volume in response to change in pressure. N static compliance: 200 mL/cmH_2O. N dynamic compliance: 180 mL/cmH_2O. N compliance in patient on IPPV: 35–50 mL/cmH_2O.
Compliance of lung measured on IPPV Tidal volume/(plateau airway pressure – PEEP).
Consolidation Replacement of alveolar air by substance of greater density than air.
Contralateral Opposite side.
COPD Chronic obstructive pulmonary disease.
Cough syncope Loss of consciousness for a few seconds following cough. Sometimes occurs in middle-aged men with COPD. Benign except while driving.

CPAP Continuous positive airways pressure.
CPR Cardiopulmonary resuscitation.
Creatinine Electrolyte in plasma or urine, formed from muscle breakdown and excreted by kidneys. N in plasma: 50–100 nmol/L [0.6–1.2 mg/100 mL]. ↑ in hypovolaemia or kidney failure, ↑↑ in septic shock.
Cryptogenic Of unknown cause.
Cryptogenic organizing pneumonia See BOOP.
CSF Cerebrospinal fluid.
CT Computed tomography.
CVP Central venous pressure. N: 1–7 mmHg or 5–12 cmH_2O.
C_vO_2 See Mixed venous oxygen content.
CXR Chest X-ray.
Cytokine Inflammatory product.
Cytomegalovirus Virus to which immunocompromised people are susceptible.
DB Deep breathing.
Deglutition Swallowing.
Delirium Condition of disorientation, fear, misperception, hallucination.
DH Drug history.
DIC Disseminated intravascular coagulation.
DNR Do not resuscitate.
DO_2 See Oxygen delivery.
Doppler Use of changes in frequency of light and sound waves to monitor relative motion.
Duty cycle (T_I/T_{TOT}) Ratio of inspiratory time to total respiratory cycle time. Short T_I in relation to T_{TOT} indicates ↓ V_T and ↑ dead space, suggesting inspiratory muscle fatigue. *See also* Tension-time index.
DVT Deep vein thrombosis.
Dysphagia Difficulty swallowing.
$ECCO_2R$ extracorporeal CO_2 removal.
ECG Electrocardiogram.
ECMO Extracorporeal membrane oxygenation.
-ectomy removal.
EEG Electroencephalogram: electric potential of brain cells recorded as brain waves.
EIA Exercise-induced asthma.
Eisenmenger's syndrome Pulmonary vascular disease with cyanosis due to extrapulmonary shunt in which congenital heart disease causes a systemic-to-pulmonary circulation connection.
Elastance Opposite of compliance.
EMD Electromechanical dissociation: dissociation of electrical and mechanical activity of the heart, represented by clinical signs of cardiac arrest with normal ECG. Causes include tension pneumothorax, cardiac trauma or severe electrolyte or acid–base imbalance. Leads to cardiac arrest.
Encephalopathy Damage to the structure or function of brain tissue.
Endotoxic shock Septic shock.
Endurance Capacity of muscle to sustain contraction.
Enteral Via the gut.
Entrainment Dilution of a gas stream or aerosol with external gas such as room air.
Eosinophilia Excess eosinophils. Indicates allergic state, e.g. extrinsic asthma.
Eosinophils Inflammatory cells associated with hypersensitivity reactions. ↑ in allergy, e.g. extrinsic asthma.
Epiglottis Cartilage that diverts food to oesophagus by closing over trachea.
ERCP Endoscopic retrograde cholangiopancreatography: procedure for detection and treatment of gallstones.
ERO_2 Oxygen extraction ratio.
Erythrocytosis Polycythaemia.
$ETCO_2$ End-tidal CO_2. N: 4–6%.
ETT Endotracheal tube.
Eucapnia Normal P_aCO_2.
Extracorporeal Outside the body.
Fatigue Abnormal tiredness after normal activity, or decreased energy that prevents activity that needs sustained effort.
FBC Full blood count.
FDPs See Clotting studies.
FEF_{25-75} Forced expiratory flow in middle half of expiration.
FEF_{50} As above.
FET Forced expiration technique.
FEV_1 Forced expiratory volume in 1 second.
FFP Fresh frozen plasma. Frozen within 6 hours of donation. Contains all clotting factors at normal concentration.
FH Family history.
F_IO_2 Fraction of inspired oxygen.
First-pass metabolism Metabolism of a drug other than systemically, due to insufficient absorption or metabolism by gut or liver before drug enters systemic circulation.
Fluid compartments Extracellular space (14 L) and intracellular space (28 L). Extracellular space is subdivided into vascular (4 L) and interstitial (10 L) spaces.
Fluid overload 10% or greater increase in weight due to fluid.
FRC Functional residual capacity.
FUO Fever of unknown origin.
FVC Forced vital capacity.
GCS Glasgow Coma Scale.
Generations Branches of tracheobronchial tree, numbering between 10 and 23.
Glottis Opening between vocal cords.
Glucose level in blood N: 3.0–5.5 mmol/L. ↑ in stress, ↑↑ in diabetes mellitus, ↓ in liver failure or starvation.
GOR Gastro-oesophageal reflux.
Gram-negative pathogenic bacteria Virulent organisms that thrive in moist environments, e.g. *Haemophilus*, *Klebsiella*, *Pseudomonas*, *Salmonella*.
Haematocrit Concentration of red blood cells in blood. N in women: 36–46%, N in men: 40–50%. Anaemia: < 36%. Polycythaemia: > 55%.
Haemoglobin Respiratory pigment in red blood cells that combines reversibly with oxygen. N in women: 11.5–15.5 g/100 mL, N in men: 14.0–18.0 g/100 mL. ↓ in anaemia, ↑ in polycythaemia.

Hamman's sign Crunching sound synchronous with heart beat, indicating pneumomediastinum.
Harrison's sulcus Deformity of the thorax in children caused by pull of diaphragm on ribs that are either not yet calcified or weakened by rickets.
Hartmann's solution Fluid and electrolyte replenisher.
Hb Haemoglobin.
HCO_3^- Bicarbonate ion concentration.
HDU High dependency unit.
Hepatomegaly Enlarged liver.
HFJV High-frequency jet ventilation.
HFO High-frequency oscillation.
HFPPV High-frequency positive pressure ventilation.
HFV High-frequency ventilation.
Hiccup Involuntary clonic spasm of intercostals and diaphragm followed by abrupt glottic closure, of unknown aetiology.
Hickman line Central venous access line, e.g. for chemotherapy.
Histotoxic Tissue-poisoning.
HIV Human immunodeficiency virus.
H:L ratio ratio of power in high and low frequency bands of electromyogram of respiratory muscle. ↓ with respiratory muscle fatigue.
HLT Heart lung transplant.
HME Heat–moisture exchanger.
HR Heart rate. N: 60–100/min.
HRCT High resolution computed tomography.
Humoral Non-cellular.
Hyperdynamic Status signalling onset of septic shock: galloping pulse, pyrexia, shaking chill, flushing of skin, high cardiac output, unstable BP.
Hyperkalaemia ↑ potassium.
Hypermetabolism ↑ basal energy expenditure by more than 10%.
Hypernatraemia ↑ serum sodium.
Hyperosmolar Containing a high concentration of osmotically active ingredients.
Hyperoxia Abnormally high oxygen tension in blood.
Hyperreactivity Heightened sensitivity to variety of stimuli. Present in airways with asthma. Sometimes present with COPD, bronchiectasis, CF, sarcoidosis, LVF.
Hyperthermia Core temperature > 40.5°C.
Hyperventilation CO_2 removal in excess of CO_2 production, causing P_aCO_2 < 4.7 kPa (35 mmHg).
Hypogammaglobulinaemia ↓ gamma globulin in blood, associated with ↓ resistance to infection.
Hypokalaemia ↓ potassium.
Hyponatraemia ↓ sodium.
Hypopnoea Shallow slow breathing.
Hypoventilation CO_2 production in excess of CO_2 removal, causing P_aCO_2 > 6.0 kPa (45 mmHg).
Hysteresis Difference in compliance between inspiration and expiration.
IABP Intra-aortic balloon pump.
Iatrogenic Causing or exacerbating harm by medical intervention.
ICP Intracranial pressure.
ICU Intensive care unit.
Idiopathic Of unknown cause.
I:E See Inspiratory:expiratory ratio.
Ileostomy Surgical creation of opening into ileum.
Ileus Gut obstruction, e.g. due to paralytic ileus.
Immotile cilia syndrome Primary ciliary dyskinesia.
Immunoglobulin Antibody. Examples in respiratory secretions: IgA, IgE, IgG, IgM.
IMV Intermittent mandatory ventilation.
Infarct Death of tissue due to lack of blood supply.
Infection Presence of microorganisms or their products in normally sterile tissue (see *also* Sepsis).
Infiltrate Fluid, cells or other substance in fluid or tissue space, e.g. pulmonary interstitial infiltrate = fluid between capillary and alveolus, showing on X-ray as diffuse shadowing.
INR See Clotting studies.
Inspiratory capacity Volume inspired during maximum inspiration from resting end-expiratory position.
Inspiratory:expiratory ratio Duration of inspiration relative to expiration.
Inspiratory force See MIP.
Inspissated Thick.
Intracranial hypertension ↑ ICP.
Intrapulmonary pressure Alveolar pressure.
Intrathoracic pressure Pleural pressure.
IPPB Intermittent positive pressure breathing.
IPPV Intermittent positive pressure ventilation, i.e. mechanical ventilation.
Ipsilateral Same side.
IRT Immune reactive trypsin: antibody identified in CF screening.
IRV Inspiratory reserve volume.
IS Incentive spirometry.
Isotonic Exerting the same osmotic pressure, for example, isotonic saline contains salt equal to that in body.
Isotonic exercise Active exercise with movement, force of contraction remaining constant.
IV Intravenous.
JVP Jugular venous pressure.
K Potassium.
Kartagena's syndrome Triad of bronchiectasis, sinusitis and situs inversus, associated with primary ciliary dyskinesia.
Kerley B lines Thin 1–2 cm horizontal lines on X-ray that abut the visceral pleural surface, representing engorged lymphatics and thickened interlobular septa, indicative of pulmonary oedema. Become visible when the pulmonary artery wedge pressure exceeds 25 mmHg.
kPa kilopascal.
KTPP See Clotting studies.
Kussmaul breathing Deep sighing breathing often seen in patients with metabolic acidosis.

Kyphoscoliosis Anteroposterior and lateral curvature of spine.
Lactate (in blood) Serum lactate. N: < 1 mmol/L. Severe oxygen debt and poor prognosis: 2.5–3.0.
Lactic acidosis Elevation of blood lactate due to insufficient oxygen in tissues for body's needs, causing metabolic acidosis. *See also* Anaerobic threshold.
Laparoscopy Minimal access incision through abdominal wall.
Laparotomy Full surgical incision through abdominal wall.
Laplace's law Pressure developed by diaphragmatic contraction directly proportional to the tension developed in its fibres and inversely proportional to its radius of curvature.
Laminar Streamline.
Larynx Cylindrical tube connecting pharynx and trachea, formed by cartilages and containing vocal cords.
Leukocyte White blood cell.
Leukocytosis ↑ white blood cells.
LFT (1) Lung function test (2) liver function test.
Locked-in state Total paralysis below third cranial nerve, with patient partially or fully aware.
LTOT Long-term oxygen therapy.
LVEDP Left ventricular end-diastolic pressure.
LVEDV Left ventricular end-diastolic volume. Determinant of preload. Depends on venous return to L ventricle, circulating blood volume and efficiency of left atrial contraction. Measured, by assumption, from PAWP, which relates to LVEDP.
LVF Left ventricular failure.
Lymphocyte Small leukocytes comprising 25% total white blood cell count.
-lysis Breakdown.
MAP See Mean arterial pressure.
Mast cells Connective tissue cells involved in hypersensitivity reactions. Release histamine in response to specific stimuli. ↑ in asthma.
MCV See Mean corpuscular volume.
MDI Metered dose inhaler.
Mean arterial pressure Average pressure pushing blood through systemic circulation, i.e.: (systolic BP + (diastolic BP × 2))/3. N: 80–100 mmHg. Compromised circulation to vital organs: < 60 mmHg. Compromised circulation to injured brain: < 80 mmHg.
Mean corpuscular haemoglobin Amount of Hb in red blood cells.
Mean corpuscular volume Size of red blood cells. ↓ (small RBCs): iron deficiency. ↑ (large RBCs): vitamin B_{12} or folate deficiency.
Mediastinum Part of thoracic cavity between the pleural sacs containing the lungs. Contains all the thoracic viscera except the lungs.
MEF_{50} Maximum expiratory flow in mid-expiration.
Mendelson's syndrome Aspiration of gastric contents into lungs.
MEP Maximal expiratory pressure. N: 100 cmH_2O. Inadequate cough: < 40 cmH_2O.
mEq Milliequivalent, i.e. one-thousandth of molecular weight of substance.
mEq/L Milliequivalents per litre of solution. Electrolyte concentration, also expressed as mmol/L.
MET Metabolic energy expenditure. Metabolic unit representing oxygen consumed at rest. Basal metabolism consumes 1 MET, eating consumes 2 METs, dressing consumes 3 METs, vacuuming consumes 5 METs, brisk 30–60 min walk consumes 9–10 METs. N: 3.5 ml oxygen/kg body weight/min.
Metabolism Chemical processes of body, comprising anabolism and catabolism. Varies with healing, activity level and temperature.
Metastasis Ability of cells to spread through bloodstream or lymphatics.
MH Manual hyperinflation.
MI See myocardial infarction.
Micrometre (μm) One millionth of a metre.
Micron Old term for micrometre (see *above*).
Miliary TB Homogenous TB of lung manifest by mottling on X-ray.
MIP Maximum inspiratory pressure: assessment of respiratory muscle strength. N: −100 to −130 cmH_2O (men), −70 to −100 cmH_2O (women). Typical value for hypercapnic COPD: −55 (men), −40 (women). Poor weaning outcome: −20. Inadequate cough: −0–20.
Mixed venous blood Blood in pulmonary artery.
Mixed venous oxygen content $(Hb \times S_{\bar{v}}O_2 \times 1.39) + (P_{\bar{v}}O_2 \times 0.023)$.
Mixed venous oxygen saturation N: 75%.
MMEF Maximum mid-expiratory flow.
mmHg Millimetres of mercury.
mmol Millimole: One one-thousandth (0.001) of molecular weight of substance.
MMV Mandatory minute ventilation.
MODS Multiple organ dysfunction syndrome (multisystem failure).
Mucoactive Affects quality or quantity of mucus.
Mucokinetic Accelerates mucus transport.
Mucolytic Destroys mucin in mucus gel.
Mucoviscidosis Cystic fibrosis.
MV Minute ventilation or volume (see V_E)
Mycoplasma pneumonia Atypical pneumonia which affects otherwise healthy people rather than the chronically ill or elderly.
Myocardial infarction Death of a portion of heart muscle due to myocardial ischaemia.
Na See Sodium.
Neutropenia ↓ neutrophils, i.e. $< 1.5 \times 10^9$.
Neutrophil White blood cell used for phagocytosis of bacteria but which in excess releases tissue-damaging enzymes as part of uncontrolled inflammation.
Neutrophilia ↑ neutrophils.

NFR Not for resuscitation.
NHS National Health Service.
NICE National Institute for Clinical Excellence.
NICU Neonatal intensive care unit.
NIPPV Nasal (or non-invasive) intermittent positive pressure ventilation, i.e. non-invasive ventilation.
NIV Non-invasive ventilation.
NO Nitric oxide.
Nociceptive Painful.
Normocapnia Normal P_aCO_2.
O_2 Oxygen.
Occupational lung disease Disease due to inhalation of dust, particles, fumes or gases while working.
OER See Oxygen extraction ratio.
OHFO Oral high frequency oscillation.
Oliguria ↓ urine output, i.e. < 20 mL/h (normal 50–60 mL/h).
Operation Surgery plus anaesthesia.
Orthostatic Relating to the erect position.
Orthotopic transplantation Replacement of recipients' organ with that of donor.
-oscopy Visual examination of the interior of an organ.
Osmolality Number of osmotically active particles per kilogram of solvent.
Osmolar load Osmolality of solution × volume infused.
Osmolarity Number of osmotically active particles per litre of solution.
-ostomy Formation of artificial opening to skin surface.
-otomy Incision.
Oxygen consumption Amount of oxygen consumed by tissues each minute, i.e. $CI \times (C_aO_2 - C_{\bar{v}}O_2) \times 10$ mL/min/m^2. N at rest: 200–250 mL/min (if contributing values normal, i.e. CO 5 L/min, Hb 15 g/100 mL, S_aO_2 97%, $S_{\bar{v}}O_2$ 75%). Critical illness: 600 ml/min. Maximum on exercise in unfit male: 3600 mL/min. Maximum on exercise in fit male: 5000 mL/min.
Oxygen content Total amount of oxygen in blood, i.e. $(Hb \times S_aO_2 \times 1.39) + (P_aO_2 \times 0.023)$. N in arterial blood: 17–20 mL/100 mL.
Oxygen cost of breathing Energy requirements of respiratory muscles. Provides indirect measure of work of breathing. N: 1 mL/L of ventilation.
Oxygen delivery (DO_2) Volume of oxygen presented to tissues, i.e. $CI \times C_aO_2$. N: 550–650 mL/min/m^2. May be elevated in hyperdynamic states.
Oxygen demand Oxygen needed by cells for aerobic metabolism, estimated by $\dot{V}O_2$.
Oxygen extraction Oxygen transferred from blood to tissues, i.e. C_aO_2 difference between arterial and mixed venous blood, equivalent to $\dot{V}O_2/DO_2$.
Oxygen extraction ratio Ratio of oxygen consumption to oxygen delivery ($\dot{V}O_2/DO_2$), indicating efficiency of tissues in extracting oxygen. Calculation: $(C_aO_2 - C_{\bar{v}}O_2)/C_aO_2$. N: 25%. High oxygen extraction to meet excess metabolic needs: > 35%. Maximum OER: 60–70% for most tissues.
Oxygen flux Percentage of oxygen that reaches tissues.
Oxygen transport Oxygen transported from lungs to mitochondria.
Oxygen uptake Oxygen consumption.
Ozone Gas that provides a protective layer to earth's atmosphere but at ground level causes inflammation in hyperreactive airways.
P_{50} PO_2 at which 50% of haemoglobin in blood is saturated with oxygen. N: 27–28 mmHg.
PA Posteroanterior.
$P_{A-a}O_2$ Alveolar–arterial oxygen gradient, i.e. difference in partial pressures of P_AO_2 and P_aO_2. Indicates gas exchange efficiency but varies with F_iO_2. N on room air: 0.7–2.7 kPa (5–20 mmHg), reflecting normal anatomical shunt. ↑ with $\dot{V}_A/Q$ mismatch or diffusion impairment, ↑ on exercise, ↑ in the elderly, ↑ on supplemental oxygen; for example, on 100% oxygen $P_{A-a}O_2$ = 3.3–8.6 kPa.
Pack years Average number of packs smoked daily multiplied by years smoked, e.g. smoking 1 pack/day for 30 years = 30 pack-year history.
Packed cell volume Equivalent to haematocrit. N: 0.36–0.46 (women), 0.40–0.50 (men). ↑ in polycythaemia, ↓ in anaemia.
P_aCO_2 Partial pressure of CO_2 in arterial blood. N: 4.7–6.0 kPa (35–45 mmHg).
Palliation Alleviation of symptoms.
P_AO_2 Partial pressure of oxygen in alveoli.
P_aO_2 Partial pressure of oxygen in arterial blood. N: 11–14 kPa (80–100 mmHg).
P_aO_2:F_iO_2 Ratio of P_aO_2 to inspired oxygen. Estimates shunt; similar to $P_{A-a}O_2$ but easier to use. N: 40 kPa (300 mmHg).
PAOP Pulmonary artery occlusion pressure (= PAWP).
PAP Peak airways pressure (= peak inspiratory pressure).
PAP Pulmonary artery pressure. N: 10–20 mmHg (systolic 22, diastolic 10, mean 15). Pulmonary hypertension: 25 mmHg.
Paralytic ileus Decrease or absence of peristalsis.
Parenchyma Foamlike substance comprising the gas exchanging part of lung, made up of alveoli, small airways, capillaries and supporting tissue.
Parenchymal lung disease Disease affecting parenchyma, e.g. interstitial lung disease, pneumonia, TB, ARDS.
Parenteral Other than through the gut.
Pathogen Bacteria, viruses, etc. that can cause disease.
PAWP Pulmonary artery wedge pressure. N: 5–15 mmHg. Pulmonary congestion: 20 mmHg. Pulmonary oedema: 25 mmHg.
$\bar{P}_{aw}$ Mean airway pressure.
PC Pressure controlled ventilation.
PCA Patient controlled analgesia.
PCIRV Pressure-controlled inverse-ratio ventilation.
PCP *Pneumocystis carinii* pneumonia.
PCV See packed cell volume.
PCWP Pulmonary capillary wedge pressure (= PAWP).
PD Postural drainage.
P_{di} Transdiaphragmatic pressure.

PE Pulmonary embolus.
PEEP Positive end-expiratory pressure.
PEFR Peak expiratory flow rate.
p_{Emax} Maximum expiratory pressure at mouth. See MEP.
PEP Positive expiratory pressure.
Percussion (therapeutic) Clapping chest wall to loosen secretions; (diagnostic) tapping chest wall to identify density of underlying tissue.
Percutaneous Through the skin.
Petechiae Small skin haemorrhages.
pH Inverse of log of hydrogen ion concentration. Measure of hydrogen ions in solution.
Phlebotomy Therapeutic withdrawal of blood.
PICU Paediatric intensive care unit.
PIE Pulmonary interstitial emphysema.
PIF Peak inspiratory flow. N: 40–50 L/min. Breathlessness or exercise: up to 200 L/min.
PIFR Peak inspiratory flow rate (= peak inspiratory flow).
P_{Imax} Maximum inspiratory pressure at the mouth. See MIP.
PIP Peak inspiratory pressure.
Plasma colloid osmotic pressure N: 3.4 kPa (26 mmHg). Risk of pulmonary oedema: 1.45 kPa (11 mmHg).
Plasma osmolarity N: 280–300 mosmol/L.
Plasmapheresis Plasma exchange.
-plasty Reconstruction.
Platelet count See Clotting studies.
Platypnoea Difficulty breathing while sitting up.
Plethoric Florid complexion due to excess red blood cells.
PM Passive movements.
PN Percussion note.
Pneumectomy Lung volume reduction surgery (cf. pneumonectomy, see index).
Pneumomediastinum Air in mediastinum.
Pneumonitis Inflammation of lung tissue due to chemical or physical insult.
Pneumopericardium Air in pericardium.
PO_2 Partial pressure or tension of oxygen.
Polycythaemia Excess red blood cells due to late-stage lung disease, cyanotic congenital heart disease, high-altitude living or sleep apnoea.
Polysomnography Recording of physiological parameters during sleep.
Polyuria ↑ urine output, i.e. > 100 mL/h.
POMR Problem oriented medical record.
Postural hypotension Drop in BP of more than 5 mmHg on moving to upright position.
Potassium (K) Electrolyte in plasma or urine. N in plasma: 3.5–5.0 mmol/L.
Poudrage Pleurodesis.
PS Pressure support ventilation.
Psittacosis Infectious disease of birds transmitted to humans, causing atypical pneumonia.
PT See Clotting studies.
PTCA Percutaneous transluminal coronary angioplasty.
$P_{tc}CO_2$ Transcutaneous CO_2 tension.
$P_{tc}O_2$ Transcutaneous oxygen tension.
PTT See Clotting studies.
Pulmonary hypertension ↑ pulmonary artery pressure, i.e. > 25 mmHg (mean) at rest or 30 mmHg on exercise in presence of cardiac output below 5 L/min.
Pulmonary osteoarthropathy Pain and swelling of joints associated with lung, liver and congenital heart disease.
Pulmonary vascular resistance N: 25–125 dyn.s.cm^{-5}
Pulse pressure Difference between systolic and diastolic pressures: indicates blood flow. N: 40–70 mmHg. ↑ with hypertension, ↓ with poor stroke volume. Dangerously low tissue perfusion: 20 mmHg.
Pulsus paradoxus Weaker pulse on inspiration than expiration caused by expansion of pulmonary vascular bed on inspiration. i.e. excess negative pressure in chest, e.g. severe acute asthma, hypovolaemic patient on IPPV, cardiac tamponade. N: 10 mmHg, higher value indicating laboured breathing.
Pump, ventilatory/respiratory Components of breathing mechanism, comprising respiratory centres, muscles and nerves of respiration, chest wall.
Purulent Containing pus.
PVD Peripheral vascular disease.
$P_{\bar{v}}CO_2$ Mixed venous CO_2 tension. N: 6.1 kPa (46 mmHg).
$P_{\bar{v}}O_2$ Mixed venous oxygen tension. N: 4.7–5.3 kPa. Minimum acceptable: 3.7 kPa (28 mmHg).
Pyothorax Large empyema.
Q Volume of blood.
QALY Quality-adjusted life-year.
QOL Quality of life.
$\dot{Q}$ Volume of blood per unit time.
$\dot{Q}O_2$ Oxygen delivery (alternative abbreviation: DO_2).
$\dot{Q}_s$ Shunted blood.
$\dot{Q}_T$ Cardiac output.
$\dot{Q}_s/\dot{Q}_t$ Shunt fraction.
Radiolabelling Monitoring mucus clearance by inhalation of radiolabelled aerosol and following up its clearance by gamma camera.
RAP Right atrial pressure.
Raynaud's phenomenon of the lung Vasospasm in the lungs associated with Raynaud's syndrome.
RCV Respiratory syncytial virus.
RDS Respiratory distress syndrome.
Relative humidity N in ambient air: 25–50%. N in upper trachea: 95%.
REM Rapid-eye-movement phase of sleep cycle.
Resection Surgical cutting out.
Resistance to gas flow through airways Spontaneous ventilation: 0.6–2.4 cmH_2O/L/s. COPD: 13–18 cmH_2O/L/s. During IPPV: 6 cmH_2O/L/s.
Respiratory inductive plethysmography Spirometry for ventilated patients, including measurement of lung volume to detect intrinsic PEEP.
Respiratory quotient (RQ) Ratio of CO_2 produced to O_2 consumed. Provides a measure of energy consumption.

Usually < 1.0 because expired minute volume is slightly less than inspired minute volume, less CO_2 being excreted than O_2 absorbed. N: 0.7–1.0, depending on food eaten before test. RQ of carbohydrate oxidation: 1.0. RQ of fat oxidation: 0.7.

RFT Respiratory function test.

Rhinitis Inflammation of mucus membrane of nose, either seasonal (hayfever) or perennial.

Rhonchi Low-pitched snoring-like wheeze on auscultation, often related to airway secretions.

Rigors Bone-shaking, teeth-rattling chills.

ROM Range of motion

RPE Rate of perceived exertion

RR Respiratory rate N: 10–20/min.

RTA Road traffic accident.

RV Residual volume.

S_aO_2 Saturation of haemoglobin with oxygen in arterial blood. N: 95–98%.

SGAW Specific airways conductance.

SH Social history.

Shunt N: 2% of cardiac output.

Silent lung zone Small airways where airflow resistance is difficult to measure so that damage may not be detectable in early obstructive airways disease.

SIMV Synchronized intermittent mandatory ventilation.

Sinus arrhythmia Acceleration of pulse with respiration, common in children.

Sinusitis Inflammation of sinus cavities of face. If chronic, may cause or exacerbate some pulmonary diseases.

Situs inversus Transposition of organs in chest and abdomen to the opposite side, e.g. heart on right side.

SLE Systemic lupus erythematosus.

Small airways Terminal and respiratory bronchioles, i.e. < 1 mm diameter. Unsupported by cartilage, therefore influenced by transmitted pleural pressures.

SOB Shortness of breath.

SOOB Sit out of bed.

Sodium (Na) Electrolyte in plasma or urine. N in plasma: 135–147 mmol/L [135–147 mEq/L].

Somatization Distress expressed as a physical symptom.

Splanchnic Related to viscera.

S_pO_2 Oxygen saturation by pulse oximetry, equivalent to S_aO_2.

Stent Device for maintaining patency of obstructed structure.

Sternotomy Surgical cutting through the sternum.

Strength Ability of muscle to create force.

Stroke volume Volume ejected from ventricle with each beat. Dependent on preload, afterload and contractility. Normally the same for each ventricle. N: 60–130 mL.

Subcutaneous emphysema Surgical emphysema.

Surfactant Phospholipid protein complex that lines alveoli.

SVR See Systemic vascular resistance.

S_vO_2 See Mixed venous oxygen saturation.

Syncope Transient loss of consciousness, e.g. faint.

Systemic vascular resistance (MAP – CVP/cardiac output) x 79.9. N: 800–1400 dyn.s.cm^{-5}. Septic shock: < 300.

Tamponade Fluid in the pericardium.

TED Thromboembolic disease.

Tension-time index (TT) Measurement of muscle fatigue. Quantifies relationship between load and capacity. Indicates proportion of muscle's maximum capacity that can be sustained indefinitely. Diaphragmatic TT (TT_{di}) = (mean P_{di} per breath/max P_{di}) x T_I/T_{TOT}. TT beyond which fatigue occurs: 0.15.

Thoracentesis Thoracocentesis, i.e. withdrawal of fluid from pleural cavity.

Thoracoplasty Surgery used historically for pulmonary TB, involving rib resection and localized lung collapse to allow healing.

Thoracoscopy Minimal access incision through chest wall.

Thoracotomy Full surgical incision through chest wall.

Thrombocytopenia ↓ platelet count.

Thrombolysis Dissolution of thrombus.

TIA Transient ischaemic attack.

T_I/T_{TOT} See Duty cycle

TLC Total lung capacity.

TLCO Total lung transfer capacity for carbon monoxide.

TPN Total parenteral nutrition, i.e. food administered intravenously.

Tracheal sounds Sounds heard on auscultation at suprasternal notch or side of neck.

Tracheal tube Endotracheal or tracheostomy tube.

Trendelenburg position Head down tilt.

Tonicity Osmotic equivalence of fluids. Isotonic fluids have same osmolality as serum or plasma, hypotonic fluids have less, hypertonic fluids have more.

Torr Measurement of pressure used in USA, equivalent to mmHg.

TT_{di} Diaphragmatic tension-time index. See tension-time index.

T_{TOT} Total respiratory cycle.

Turbulent flow Flow that is not smooth or laminar, resulting in eddy currents which reduce the volume of gas moved per unit of time per pressure gradient.

Tusk mask Oxygen delivery system in which two 20 cm lengths of corrugated tubing are fitted to exhalation ports of oxygen mask to trap oxygen during exhalation and recycle it to patient during next inhalation (Hnatiuk, 1998).

Tussive Related to cough.

Us & Es Urea and electrolytes.

Upper respiratory tract Nose (or mouth), pharynx and larynx.

Urea Electrolyte in plasma or urine, formed from protein breakdown and excreted by kidneys. N in plasma: 3–7 mmol/L. Dehydration: > 8. Hypovolaemia: > 18. Kidney failure: 55.

Urine output N: 1 mL/h/kg, average 50–60 mL/h. Renal failure: less than half normal.

V Volume of gas.

v Venous.

Valsalva manoeuvre Forced expiration against closed glottis. Accompanies heavy resistance exercise requiring stabilization of the thorax.

VAP Ventilator-associated pneumonia.

VAS Visual analogue scale.

Vascath Vascular catheter, usually connecting patient to renal support.

Vasopressor Drug that causes vasoconstriction of capillaries and arteries.

VATS Video-assisted thoracoscopic surgery.

VC (1) Vital capacity; (2) volume-controlled ventilation.

VCIRV Volume-controlled inverse-ratio ventilation.

V_D Volume of dead space gas. N: for anatomical V_D: 2 ml/kg body weight.

V_D/V_T Dead space in relation to tidal volume, calculated by the Bohr equation. N: 0.3–0.4, i.e. 30–40%, depending on position. Critical increase: 0.6.

Venous return Blood returning to right atrium.

VF Ventricular fibrillation.

V_T Tidal volume. N: 7 mL/kg for spontaneously breathing people, 7–10 mL/kg for ventilated patients, up to 12 mL/kg in acute respiratory failure.

Venesection See Phlebotomy.

Venous admixture Mixing of shunted venous blood with oxygenated blood, i.e. mixture of 'true' shunt, which completely bypasses the pulmonary capillary bed, and 'effective' shunt due to $\dot{V}_A/\dot{Q}$ mismatch. N: 5% of cardiac output.

Venous thromboembolism Combined PE and DVT.

$\bar{v}$ Mixed venous.

$\dot{V}$ Volume of gas per unit time, i.e. flow.

$\dot{V}_{50}$ Flow rate half-way through expiration.

$\dot{V}_A/\dot{Q}$ Ratio of alveolar ventilation to perfusion. N: 0.8 (4 L/min for alveolar ventilation, 5 L/min for perfusion).

$\dot{V}CO_2$ CO_2 production. Mean fraction of expired CO_2 x VE. N: 200 mL/min at rest, increasing by 7% for each 1°C rise in body temperature.

VE (Expired) minute volume/ventilation. Also abbreviated as MV. Expired gas is usually measured (more CO_2 is produced than O_2 consumed).

$\dot{V}O_2$ Oxygen consumption.

$\dot{V}O_2/DO_2$ See Oxygen extraction ratio.

$\dot{V}O_{2max}$ Oxygen consumption at maximum exertion, reflecting aerobic capacity. Increases with fitness, declines with advancing age but rate of decline is slower in physically active people. N: > 25 mL/kg/min, or 25 times resting level. *See also* Anaerobic threshold.

WBC See White blood cell count.

WCPT World Confederation of Physical Therapy.

Well-year of life Outcome measure incorporating morbidity and mortality; for example, if disease halves quality of life for 2 years, patient has lost one full well-year.

White blood cell count N: $4–10 \times 10^9$/L [4000–10 000/mm^3]. Bacterial infection: > 10 000/mm^3. Vulnerability to infection: < 4000/mm^3.

WOB See Work of breathing.

Work of breathing N: 0.3–0.5 kg.m/min.

Xenotransplant Cross-species transplant.

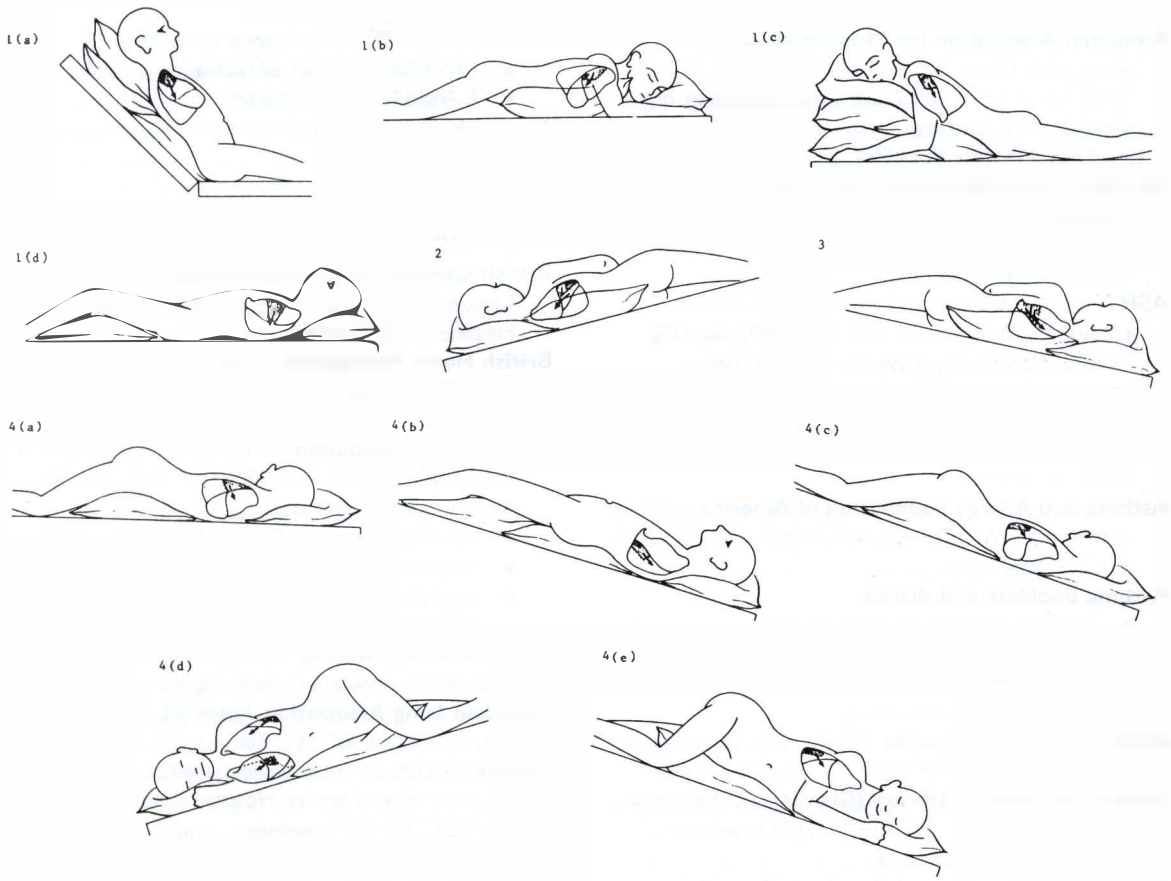
Appendix A: Transatlantic Dictionary

British	North American
1st floor	2nd floor
Accident and Emergency (A&E)	Emergency Room (ER)
Adrenaline	Epinephrine
ASAP (as soon as possible)	Stat
Cardiac arrest	Code
Chest drains	Chest tubes
Community care	Home care
Consultant	Staff person
ECG	EKG
Entonox	Nitronox
Frame/Zimmer	Walker
Frusemide	Furosemide
General practice	Primary care/family practice
Hospital	Health Sciences Centre/Facility
Houseman/woman	Intern
Hyoscine	Scopolamine
Lignocaine	Lidocaine
mmHg (unit of pressure)	torr
Nil by mouth	NPO (*nil per os*)
Paracetamol	Acetaminophen
Passive movements	Range of motion
Patient's notes	Patient's chart
Peak expiratory flow rate	Maximum expiratory flow rate
Pethidine	Meperidine
Queue	Line up
Referral	Consult
Registrar	Resident
Respiratory physiotherapist	Part physical therapist, part respiratory therapist
RTA (road traffic accident)	MVA (motor vehicle accident)
Salbutamol	Albuterol
Sluice	Utility room
Speech–language therapist	Speech pathologist
Splint	Cast
Stick	Cane
Theatre	Operating room (OR)
Trachy (tracheostomy)	Trache
Walk or mobilize	Ambulate
Ward	Floor

Appendix B: Postural Drainage Positions

1. **Upper lobes**
   1(a) Apical segments of both upper lobes – sitting upright
   1(b) Posterior segment of right upper lobe – left-side-lying, turned 45° towards prone
   1(c) Posterior segment of left upper lobe – right-side-lying, turned 45° towards prone, shoulders raised 30 cm
   1(d) Anterior segments of both upper lobes – supine
2. **Middle lobe**
   Lateral and medial segments – supine, quarter turned to left, foot of bed raised 35 cm
3. **Lingula**
   Superior and inferior segments – supine, quarter turned to right, foot of bed raised 35 cm
4. **Lower lobe**
   4(a) Apical segments of both lower lobes – prone, head turned to side
   4(b) Anterior basal segments of both lower lobes – supine, foot of bed raised 46 cm
   4(c) Posterior basal segments of both lower lobes – prone, head turned to side, foot of bed raised 46 cm
   4(d) Medial basal segment – right-side-lying, foot of bed raised 46 cm
   4(e) Lateral basal segment – left-side-lying, foot of bed raised 46 cm

(Reproduced with permission from Downie, P. A. (ed.) (1987) *Cash's Textbook of Chest Heart and Vascular Disorders for Physiotherapists*, Faber, London.)



Appendix C: Resources

ACPRC (Association of Chartered Physiotherapists in Respiratory Care), c/o Chartered Society of Physiotherapy, *see below*

Action for Sick Children, Argyle House, 300 Kingston Rd, Wimbledon, London SW20 8LX. Tel: 020 8542 4848; fax: 020 8542 2424; e-mail: action-for-sick-children-edu@msn.com

Action for Victims of Medical Accidents, 44 High St, Croydon, Surrey CRO 1YB. Tel: 020 8686 8333; fax; 020 8667 9065; website: www.avma.org.uk

Air pollution information, tel: 0800 556677.

Alzheimer's Disease Society, Gordon House, 10 Greencoat Place, London SW1P 1PH. Tel: 020 7306 0606; fax: 020 7306 0808; email: info@alzheimers.org.uk; website: www.alzheimers.org.uk

American Association for Respiratory Care, 11030 Ables Lane, Dallas, TX 75229, USA. Tel: +1 972 243 2272; fax: +1 972 484 2720; e-mail: info@aarc.org; website: www.aarc.org

- *Clinical Practice Guidelines*, $35

American Lung Association and American Thoracic Society, 1740 Broadway, New York, NY 10019-4374, USA. Tel: +1 212 315 8700; e-mail: info@lungusa.org; website: www.lungusa.org

ASH (Action on Smoking and Health), 102 Clifton St, London EC2A 4HW. Tel: 020 7739 5902; fax: 020 7613 0531; website: www.ash.org.uk; e-mail: action.smoking.health@dial.pipex.can (campaigning organization, not for smoking cessation advice). Government official helpline for those needing advice: 0800 169 0169

Asthma and Allergy Foundation of America, 1233 20th Street NW, Suite 402, Washington, DC, USA. Tel: +1 202 2650265.

Asthma booklets and diaries:

- National Asthma Campaign *see below*.
- Allen & Hanburys, Uxbridge, Middlesex UB11 1BT. Tel: 020 8990 9888; fax: 020 8990 4321; website: www.glaxowellcome.co.uk.

Asthma Society of Canada, PO Box 213, Station K, Toronto, Ontario M4P 2G5. Tel: +1 416 977 9684.

Audiovisual Medical Library (Graves), 201 Felixstowe Rd, Ipswich IP3 9BJ. Tel: 01473 726012; website: www.namron.demon.co.uk

Autogenic drainage specialist physiotherapists:

- Christine Atkinson, Hull Royal Infirmary. Tel: 01482 674539
- Alison Gates, Churchill Hospital, Oxford. Tel: 01865 741841, bleep 5058
- Lynne Gumery, Birmingham Heartlands Hospital. Tel: 0121 424 2000, bleep 2742
- Melanie Liley, Nottingham University. Tel: 0115 840 4880
- Paula McNaughton, Solihull Hospital. Tel: 0121 711 4455, page 1039
- Maureen Rowatt, Glasgow Victoria Infirmary. Tel: 0141 201 6000, bleep 3435

Bird ventilator: EME, 60 Gladstone Place, Brighton BN2 3QD. Tel :01273 645100; fax: 01273 645101; website: www.eme-med.co.uk

Breathe Easy Clubs

- SW England/S Wales: susan@blfsw.fsnet.co.uk
- South: blf85@yahoo.com
- London: blf@britishlungfoundation.com
- E/W Midlands: 0121 6272260
- E Anglia/N Home Counties: blf@britishlungfoundation.com
- NW England/N Wales: daphne@blfnw.freeserve.co.uk
- NE England/Yorks: margaretbn@currantbun.com
- Scotland: redballoon@blfscotland.org.uk

British Cardiac Patients Association (BCPA Zipper Club, 6 Rampton End, Willingham, Cambs CB4 5JB). Helpline 01223 846845

British Heart Foundation, 14 Fitzhardinge St, London W1H 4DH. Tel: 020 7935 0185; website: www.bhf.org.uk

British Lung Foundation, 78 Hatton Gardens, London EC1N 8JR. Tel: 020 7831 5831; fax: 020 7831 5832; website: www.lunguk.org

- self-help groups, *see* Breathe Easy Clubs, above
- travel insurance
- Lung and Asthma Information Agency – epidemiological database for health staff

British Thoracic Society Tel: 020 7831 8778; fax 020 7831 8766; www.brit-thoracic.org.uk.

Canadian Lung Association, Suite 908, 75 Albert St, Ottawa, Ontario KIP 5E7. Tel: +1 613 237 1208.

Cancer booklets on breathlessness: Institute of Cancer Research, Royal Marsden Hospital, London SW3 6JJ. Tel: 020 7352 8171; website: www.royalmarsden.org.uk/patientinfo/index.asp

Cancer support groups

- BACUP (British Association of Cancer United Patients), 3 Bath Place, London EC2A 3DR. Tel:

020 7613 2121; fax: 020 7696 9002; freeline no.: 0808 800 1234; e-mail: info@cancerbacup.org.uk; website: www.cancerbacup.org.uk
- Cancerlink, 11–21 Northdown St, London N1 9BN. Tel: 020 7833 2818; fax: 020 7833 4963; helpline: 0808 2080000 (Mon, Wed, Fri 0900–1700); e-mail: cancerlink@cancerlink.org; website: www.cancerlink.org

Cancer websites
- www.cancerfatigue.org
- www.webhealing.com
- www.scu.edu/Hospice
- www.langara.bc.ca/vnc/suffer2.htm

Carers National Association, 20 Glasshouse Yard, London EC1A 4JS. Tel: 020 7490 8818; fax: 020 7490 8824

Chartered Society of Physiotherapy, 14 Bedford Row, London WC1R 4ED. Tel: 020 7306 6666; fax: 020 7306 6611; e-mail: csp@csphysio.org.uk; website: www.csp.org.uk

Chest drain booklet Sherwood Davies Geck. Tel: 01329 224114; fax 01329 224390.

Cinnamon Trust (residential homes that welcome pets), Foundry House, Foundry Square, Hayle, Cornwall TR27 4HE. Tel: 01736 757900; fax 01736 757010; website: www.cinnamontrust.co.uk

Continence Foundation, 307 Hatton Square, 16 Baldwins Gardens, London EC1N 7RJ. Tel: 020 7404 6875; helpline: 020 7831 9831 (Mon–Fri 0930–1630); website: www.continence.foundation@dial.pipex.com

Cough lok (coughing aid for postoperative patients) Hawksley & Sons, Marlborough Road, Lancing, West Sussex BN15 8TN. Tel: 01903 752815; fax: 01903 766050; e-mail: enquiries@hawksley.co.uk; website: www.hawskley.co.uk

Cystic Fibrosis Guidelines *Clinical Guidelines for Physiotherapy Management of Cystic Fibrosis* (2001). Eds Gumery LB, Pryor, J., Prasad, S. A., Dodd, M. Available from Chartered Society of Physiotherapy.

Cystic Fibrosis Research Trust and Association of Cystic Fibrosis Adults, 11 London Rd, Bromley, Kent BR1 1BY. Tel: 020 8464 7211; fax: 020 8313 0472; website: www.cftrust.org.uk.

Elder Abuse helpline, 0800 7314141 (1000–1630).

Facial disfigurement support network (Let's Face It), 14 Fallowfield, Yateley, Hants GU46 6LW. Tel/fax: 020 8952 4990; www.letsfaceit.force9.co.uk

Flutter video and information: Clement Clarke, Edinburgh Way, Harlow, Essex CM20 2TT. Tel: 01279 414969; fax: (general) 01279 635232, (respiratory) 01279 456304; website: www.clement-clarke.com

Footdrop silicone orthoses: Dorset Orthopaedic Co. Ltd, 11 Headlands Business Park, Salisbury Road, Ringwood, Hants BH24 3PB. Tel: 01425 480065; fax: 01425 480083; website: www.dorset-ortho.co.uk

Glossopharyngeal breathing video: B. A. Webber, Sunnybank, The Platt, Amersham, Bucks HP7 OHX. Tel: 01494 725724. £20

Guillain–Barré Syndrome support group, LCC Offices, Eastgate, Sleaford, Lincs NG34 7EB. Helpline 0800 374803 (24 hours). Tel/fax: 01529 304615; website: www.gbs.org.uk

Headway (head injuries organization), 4 King Edward Court, King Edward St, Nottingham NG1 1EW. Tel: 0115 9240800; fax: 0115 9584446

Hospices www.hospiceinformation.co.uk

Humidifiers:
- Sunrise Medical, Wollaston, W.Midlands DY8 4PS. Tel: 01384 446688; fax: 01384 446699; website: www.sunrisemedical.com
- Fisher & Paykel, 16 Cordwallis Park, Clivemont Rd, Maidenhead, Berks SL6 7BU. Tel: 01628 626136; fax: 01628 626146; website: www.FPHcare.com
- Intersurgical, Crane House, Molly Millars Lane, Wokingham, Berks, RG41 2RZ. Tel: 0118 9795579; fax: 0118 9656356; website: www.intersurgical.co.uk.
- Kendall, 154 Fareham Road, Gosport, Hants PO13 OAS. Tel: 01329 224114; fax: 01329 224390; website: www.tyco.com (also video)
- Medicaid, Heath Place, Bognor Regis, West Sussex PO22 9SL. Tel: 01243 846111; fax: 01243 846100; website: www.medic-aid.com

Incentive spirometers:
- Kendall, see Humidifiers
- Intersurgical, see Humidifiers (also filters for incentive spirometers)

Information for patients:
- J. A. M. A. Patient Pages website: www.ama-assn.org/consumer.htm
- see Support groups

Inhaler tester for assessing inspiratory flow: Clement Clarke, see Flutter

Inspiratory muscle trainers:
- Clement Clarke, see Flutter
- Medicaid, see Humidifiers
- Henleys, 39 Brownfields, Welwyn Garden City, Herts AL7 1AN. Tel: 01707 333164; fax: 01707 334795; website: www.henleysmed.com.

- Sunrise Medical, see Humidifiers
- Tangent Healthcare, 74 Roman Bank, Long Sutton, Lincs PE12 9LB. Tel/fax: 01945 880008; website: www.ultrabreathe.com.

Kolaczkowski video: Waldemar Kolaczkowski, Physiotherapy Dept, St Michael's Hospital, Toronto, Ontario, Canada. £50

Laryngectomy support group: National Association of Laryngectomee Clubs, 6 Rickett St, London SW6 1RU. Tel: 020 7381 9993; fax: 020 7381 0025.

Listening books for housebound people: 12 Lant St, London SE1 1QH. Tel: 020 7407 9417; fax: 020 7403 1377; website: www.listening-book.org.uk

Liver patients support group: The British Liver Trust, Central House, Central Avenue, Ransomes Europark, Ipswich IP3 9QG. Tel: 01473 276326; fax: 01473 276327; website: www.britishlivertrust.org.uk.

Mallinckrodt (tracheostomy tubes, suction catheters, ventilators, oximeters, heat-moisture exchangers), 10 Talisman Business Centre, London Rd, Bicester, Oxfordshire OX26 6HR. Tel: 01869 322700; fax: 01869 321890; website: www.mallinckrodt.com.

Manometer for testing bag-squeezing pressures: Vital Signs, The Sussex Business Village, Lake Lane, Farnham, W.Sussex PO22 0AL. Tel: 01243 555300; fax: 01243 555400; website: www.vital-signs.com.

Minitracheostomy: SIMS Portex Ltd, Hythe, Kent, CT21 6JL. Tel: 01303 260551; fax: 10303 265560; website: www.portex.com (also videos)

Motor Neurone Disease Association, PO Box 246, Northampton NN1 2PR. Tel: 01604 250505; fax: 01604 624726, helpline 08457 626262; 24 hrs 0164 22269; e-mail: enquiries@mndassociation.org; website: mndassociation.org

- Breathing Space Kit to reduce fears in final stages of disease
- leaflets on communication, swallowing, physiotherapy, dying
- Resource file £10

Myasthenia Gravis Association Tel: 01332 290219.

National Asthma Campaign, Providence House, Providence Place, London N1 0NT. Tel: 020 7226 2260, helpline 08457 010203 (Mon–Fri 0900–2100); fax: 020 7704 0740; website: www.asthma.org.uk.

National Heart, Lung and Blood Institute, Building 31, Room 4A18, National Institutes of Health, Bethesda, MD 20982, USA. Website: nhlbi.nih.gov

NICE National Institute for Clinical Excellence: www.nice.org.uk

Non-invasive ventilator videos (for staff training and patient education): Lane Fox Unit, St Thomas' Hospital, London SE1 7EH. £12 each

Non-invasive ventilators:

- AirMed, 33 Half Moon Lane, Herne Hill, London SE24 9JX. Tel: 020 7737 5881; fax: 020 7737 5234; website: www.airmedltd.com
- Medicaid, see Humidifiers
- PneuPAC, Bramingham Business Park, Enterprise Way, Luton, Beds LU3 4BU. Tel: 01582 430000; fax: 01582 430001; website: www.pneupak.co.uk.
- ResMed, 67B Milton Park, Abingdon, Oxon OX14 4RX. Tel: 01235 862997; fax: 01235 831336; website: www.resmed.com.au

Oscillator: Hayek, Breasy Medical Equipment Ltd, 9 Burroughs Gardens, London NW4 4AU. Tel: 020 8203 6877; fax: 020 8203 4758.

Oxygen equipment:

- Oxygen Therapy Company, Shearwater House, Ocean Way, Cardiff CF1 5HF. Tel: 0800 373580; fax: 01222 488255; e-mail: bucklei@oxygentherapy.co.uk (*Living with Oxygen* booklet, liquid oxygen, concentrators, holiday oxygen service)
- BOC Customer Service Centre, Priestley Road, Worsley, Manchester ME28 2UT. Tel: 0800 111 333.
- ATS Medirent. Tel: 01344 477777; fax: 01344 477789 (liquid oxygen rental)
- Life Support Ltd, 4 Stavely Road, Dunstable, Beds LU6 3QQ. Tel: 01582 662616; fax: 01582 665289 (portable oxygen-conserving cylinders)
- Omnicare. Tel: 0500 823773.
- Sunrise Medical, see Humidifiers

Oximeters (portable) for exercise:

- Life Support Ltd, see Oxygen equipment finger oximeter
- Stowood Scientific Instruments, Royal Oak Cottage, Beckley, Oxford OX3 9UP. Tel/fax: 01865 358860; website: www.stowood.co.uk (wrist oximeter).
- Sunrise Medical, see Humidifiers
- see Mallinckrodt (belt oximeter)

PEP devices:

- Astra Tec, Brunel Way, Stonehouse, Glos GL10 3SX. Tel: 01453 791763; fax: 01453 791001; website: www.astratecuk.com
- Astra Tec Inc, 1000 Winter St, Suite 2700, Waltham, MA 02154, USA
- Henleys, see Inspiratory muscle trainers
- Medicaid, see Humidifiers

Polio support group: British Polio Fellowship, Eagle Office

Centre, South Ruislip, Middlesex HA4 6SE. Tel: 020 8842 1898; fax: 020 8842 0555; e-mail: british.polio@dial.pipex.com/info@britishpolio.org
- list of articles on exercise and post-polio syndrome

Postoperative pain video and booklet: Institute of Health and Community Studies, UK. £45

Primary ciliary dyskinesia family support group, 67 Evendons Lane, Wokingham, Berks RG41 4AD; www.p-c-d.org.uk (also video).

Quality of Life assessment: www.qlmed.org.

Quit (smoking cessation), Victory House, 170 Tottenham Court Road, London W1T 7NR,. Tel: 020 7388 5775, helpline 0800 002200; fax: 020 7388 5995; website: www.quit.org.uk

Shuttle audio tape and booklet, Sally Singh, Pulmonary Rehabilitation Dept, University Hospital of Leicester, Groby Road, Leicester LE3 9QP. Tel: 01162 871471

Spinal Injuries Association, Newpoint House, 76 St James Lane, London N10 3DF. Tel: 020 8444 2121; fax: 020 8444 3761; e-mail: sia@spinal.co.uk; website: www.spinal.co.uk

Stroke Association, Stroke House, 123–137 Whitecross St, London EC1Y 8JJ. Tel: 020 7566 0300; fax: 020 7490 2686; e-mail: strok@stroke.org.uk; website: www.stroke.org.uk.
- booklet on swallowing

Suction videos:
- Maersk Medical, Thornhill Rd, North Moons Moat, Redditch, Worcs B98 9NL. Tel: 01527 64222; fax: 01527 592111; website: www.maersk-medical.com
- Vygon, Bridge Road, Cirencester, Glos GL7 1PT. Tel: 01285 657051; fax: 01285 650293; e-mail: vygon@vygon.co.uk

Support groups website (2000 UK groups): www.surgerydoor.co.uk

Swallowing booklets: see Motor Neurone Disease Association and Stroke Association

Thermocyclopad (back pad), Niagara Therapy UK Ltd, Middleton House, 43--49 High Street, Horley, Surrey, RH6 7BN. Tel: 01293 787040; fax: 01293 782 006; e-mail: Cctherapy@niagaraholdings.co.uk

Tracheostomies in children: Aid for Children with Tracheostomies, 215a Perry St, Billericay, Essex CM12 ONZ. Audiotape from Mallinckrodt

Tracheostomy audiotapes and booklet: see Mallinckrodt

Yankauer substitute with soft tip – Meddis. Tel: 01491 825500; fax 01491 826600, ngough@meddis.co.uk.

APPENDIX D: ARTICLES ON PATIENTS' EXPERIENCES

Bevan, P. G. (1964) Cholecystectomy in a surgeon. *Lancet*, **i**, 214–215.

Bevan, J. R. (1969) Polyneuropathy. *Lancet*, **i**, 1310.

Bowes, D. (1984) The doctor as patient: an encounter with Guillain–Barré syndrome. *Can. Med. Assoc. J.*, **131**, 1343–1348.

Brooks, D. H. M. (1992) Living with ventilation: confessions of an addict. *Care Crit. Ill*, **8**, 205–207.

Carter, B. (1995) An asthmatic in 'Wonderland': a patient's perspective. *Accid. Emerg. Nurs.*, **3**, 139–141.

Castledine, G. (1993) A personal view of asthma. *Br. J. Nurs.*, **2**, 919.

Clark, K. J. (1985) Coping with Guillain–Barré syndrome. *Intens. Care Nurs.*, **1**, 13–18.

Cornall, A. (1995) Pulmonary rehabilitation: a patient's experience. *ACPRC J.*, **26**, 10–11.

Farrow, J. (1995) Cystic fibrosis – it's a funny name. *Respir. Dis. Pract.*, **12**(1), 5–6.

Gandy, A. P. (1968) An attack of tetanus. *Lancet*, **ii**, 567–568.

Gordon, S. (1998) Letter to a patient's doctor. *Ann. Intern. Med.*, **129**, 333–334.

Heath, J. V. (1989) What the patients say. *Intens. Care Nurs.*, **5**, 101–108.

Holden, T. (1980) Patiently speaking. *Nurs. Times*, **76**(24), 1035–1036.

Houston, J. E. (1988) Physiotherapy from a patient's point of view. *S. Afr. J. Physiother.*, **44**, 38–41.

Jablonski, R. S. (1994) The experience of being mechanically ventilated. *Qual. Health Res.*, **4**, 186–207.

Khadra, M. H. (1998) What price, compassion? *Med. J. Austr.*, **169**, 42–3.

Kinnear, W. J. M. (1994) A patient's view of living with a ventilator, in *Assisted Ventilation at Home – a Practical Guide*, Oxford Medical Publications, Oxford, ch. 8, pp. 85–93.

Lancet (1969) At the receiving end. *Lancet*, **ii**, 1129–1131.

Lancet (1969) Cardiac arrest. *Lancet*, **ii**, 262–264.

Lawrence, M. (1995) The unconscious experience. *Am. J. Crit. Care*, **4**, 227–232.

Marsh, B. (1986) A second chance. *Br. Med. J.*, **292**, 675–676.

Moore, T. (1991) *Cry of the Damaged Man*. Picador, Sydney.

Neuberger, J. (1991) The patient's viewpoint. *Respir. Med.*, **85**(suppl. B), 53–56.

Nicholson, E. M. (1975) Personal notes of a laryngectomee. *Am. J. Nurs.*, **75**(12), 2157–2158.

Nursing Times (1981) Who am I? Where am I? Why do I hurt so much? *Nurs. Times*, **77**(15), 633–635.

Oermann, M. H. (1983) After a tracheostomy: patients describe their sensations. *Cancer Nurs.*, **6**, 361–366.

Peloquin, S. M. (1995) The depersonalization of patients. *Am. J. Occup. Ther.*, **47**, 830–837.

Pisetsky, D. S. (1998) Doing everything. *Ann. Intern. Med.*, **128**, 869–870.

Redfern, S. (1985) Taking some of my own medicine. *Care Crit. Ill*, **1**, 6–7.

Rosen, A. (1998) Last on the list. *Br. Med. J.*, **316**, 1324–1325.

Ruiz, P. A. (1993) The needs of a patient in severe status asthmaticus. *Intens. Crit. Care Nurs.*, **9**, 28–39.

Shovelton, D. S. (1979) Reflections on an intensive therapy unit. *Br. Med J.*, **ii**, 737–738.

Standing, C. (1987) The treatment of our son with cystic fibrosis. *J. Roy. Soc. Med.*, **80**(suppl. 15), 2–4.

Stead, C. E. (1999) Sudden infant death syndrome (SIDS) on the 'other side'. *Accid. Emerg. Nurs.*, **7**, 170–174.

Thomson, L. R. (1973) Sensory deprivation: a personal experience. *Am. J. Nurs.*, **73**, 266–268.

Ulbright, G. F. (1986) Laryngectomy rehabilitation: a woman's viewpoint. *Women Health*, **11**, 131–136.

Villaire, M. (1995) ICU from the patient's point of view. *Crit. Care Nurse*, **15**(1), 80–87.

While, A. (1985) Personal view. *Br. Med. J.*, **291**, 343.

Wilkinson, J. (1987) The experience and expectations of parents of a child with cystic fibrosis. *J. Roy. Soc. Med.*, **80**(suppl. 15), 7–8.

Zimmerman, M. D., Appadurai, K. and Scott, J. G. (1997) Survival. *Ann. Intern. Med.*, **127**, 405–408.

Appendix E: References on outcome measures for pulmonary rehabilitation

Outcomes bulletted

ACCP/AACVPR (1997) Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. *Chest*, **112**, 1363–1396.
- ↓ SOB, ↓ hospital days and ↓ total no. hospitalizations

Ambrosino, N. and Foglio, K. (1996) Selection criteria for pulmonary rehabilitation. *Respir. Med.*, **90**, 317–322,
- elderly people: ↑ flexibility, strength, fitness

Bax, J. (1997) Long-term effects of an out-patient pulmonary rehabilitation programme in patients with asthma and COPD. *Eur. Respir. J.*, 10, 458S.
- ↑ ET and ↑ QoL, still maintained after 3 years

Bendstrup, K. E. (1997) Out-patient rehabilitation improves activities of daily living, quality of life and exercise tolerance in COPD. *Eur. Respir. J.*, 10, 2801–6.
- ↑ ADL, QoL, ET

Cambach, W., Wagenaar, R. C. and Koelman, T. W. (1999) The long-term effects of pulmonary rehabilitation in patients with asthma and COPD. *Arch. Phys. Med. Rehabil.*, **80**, 103–111.
- ↑ ET, ↑ QoL

Celli, B. R. (1995) Pulmonary rehabilitation in patients with COPD. *Am. J. Respir. Crit. Care Med.*, **152**, 861–864.
- ↑ ET, ↑ QoL

Clark, C. J., Cochrane, L. and Mackay, E. (1996) Low intensity peripheral muscle conditioning improves exercise tolerance and breathlessness in COPD. *Eur. Respir. J.*, **9**, 2590–2596.
- ↑ endurance, ↑ ADL, ↓ SOBOE

Didour, M. (1997) Effects of lung cycle ergometer training in elderly patients with bronchial asthma. *Eur. Respir. J.*, **10**, 458S.
- elderly patients with asthma: ↑ ET, ↑ QoL

Flanigan, K. S. (1991) Outcome of a 3-day pulmonary rehabilitation programme. *Respir. Care*, **36**, 1271.
- short programme: ↑ ADL, ↑ confidence.

Foglio, K., Bianchi, L. and Bruletti, G. (1999) Long-term effectiveness of pulmonary rehabilitation in patients with CAO. *Eur. Respir. J.*, **13**, 125–132.
- ↑ QoL, maintained 1 year after rehabilitation

Fulambarker, A., Lund, D. and Chandok, S. (1995) Effect of pulmonary rehabilitation on hospital days of COPD patients. *Chest*, 114S.
- ↓ admissions, ↓ length of stay

Gallefoss, F. and Bakke, P. S. (1999) How does patient education and self-management among asthmatics and patients with COPD affect medication? *Am. J. Respir. Crit. Care Med.*, **160**, 2000–2005.
- ↓ β_2-agonist medication.

Garrod, R. (1998) The pros and cons of pulmonary rehabilitation at home. *Physiotherapy*, **84**, 603–607.
- ↑ ET by 24%

Giddings, D. J. (1994) Outcome evaluation of a respiratory rehabilitation program. *Physiother. Can.*, **46**(2suppl.), 81
- ↑ ET, ↑ ADL

Griffiths, T. L., Gregory, S. E. and Ward, S. A. (1996) Effects of a structured domiciliary exercise training programme on quality of life and walking tolerance in patients with severe COPD. *Eur. Respir. J.*, **9**(23), 144s.
- severely impaired patients: ↑ ET, ↑ QoL

Griffiths, T. L., Burr, M. L. and Campbell, I. A. (2000) Results at 1 year of outpatient pulmonary rehabilitation: a randomised controlled trial. *Lancet*, **355**, 362–368.
- ↓ hospitalisations by half

Haas, F. and Axen, K. (1991) *Pulmonary Therapy and Rehabilitation*, Williams & Wilkins, Baltimore, MD, p. 336.
- cost savings > $2600 per patient/year

Haggerty, M. C. (1999) Functional status in pulmonary rehabilitation participants. *J. Cardiopulmonary Rehabil.*, **19**, 35–42.
- ↑ ET, ↑ functional status

Heijerman, H. G. M. (1992) Long-term effects of exercise training and hyperalimentation in adult CF. *Int. J. Rehabil. Res.*, **15**, 252–257.
- CF: transplantation no longer needed in some patients

Hodgkin, J. E., Bartolome, R., Celli, B. R., Connors, G. L. (2000) *Pulmonary Rehabilitation: Guidelines to Success*, 3rd edn. Lippincott Williams & Wilkins, Philidelphia, PA.
- ↓ costs, ↓ hospitalisations

Lacasse, Y., Wong, E. and Guyatt, G. H. (1996) Meta-analysis of respiratory rehabilitation in COPD. *Lancet*, **348**, 1115–1119.
- 14 trials showed ↑ ET, ↑ QoL

Make, B. (1990) Pulmonary rehabilitation – what are the outcomes? *Respir. Care*, **35**, 329–331.
- literature review of outcomes

Make, B. (1994) Collaborative self-management strategies for patients with respiratory disease. *Respir. Care*, **39**, 566–577.
- ↑ workload by 30%, ↑ $\dot{V}O_{2\ max}$ by 9.2%, ↑ 6-minute distance by 21%, ↑ ADL, ↑ social and emotional function

Murray, E. (1993) Anyone for pulmonary rehabilitation? *Physiotherapy*, **79**, 705–710.
- ↑ ET, ↓ hosp, ↓ symptoms, ↓ anxiety and depression, ↑ QoL

Novitch, R. S. (1995) Pulmonary rehabilitation in patients with interstitial lung disease. *Am. J. Respir. Crit. Care Med.*, **151**, A684.
- interstitial lung disease: ↑ ET and arm ergometry.

Ojanen, M. (1993) Psychosocial changes in patients participating in a COPD rehabilitation program. *Respiration*, **60**, 96–102.
- ↑ well-being, ↓ symptoms

Parker, L. and Walker, J. (1998) Effects of a pulmonary rehabilitation program on physiologic measures, quality of life and resource utilization. *Respir. Care*, **43**, 177–182.
- ↑ QoL, ↓ costs

Revill, S. M., Morgan, M. D. L. and Singh, S. J. (1999) The endurance shuttle walk. *Thorax*, **54**, 213–222.
- ↑ endurance

Ries, A. L., Kaplan, R. M. and Limberg, T. M. (1995) Effects of pulmonary rehabilitation on physiologic and psychosocial outcomes in patients with COPD. *Ann. Intern. Med.*, **122**, 823–832.
- ↓ symptoms, ↑ ET

Schleifer, T. J. (1994) Patient responsibility in an innovative COPD therapy program. *Physiother. Can.*, **46**(2suppl.), 81.
- for severe disease: ↑ ADL, ↓ exacerbations, ↓ anxiety

Sridhar, M. K. (1997) Pulmonary rehabilitation. *Br. Med. J.*, **314**, 1361.
- ↑ QoL, ↑ ET, ↓ SOB, ↑ control

Tanaka, M. (1999) Paced breathing exercises for patients with chronic respiratory failure. *WCPT Proc.*, p. 312.
- RR ↓ from 21/min to 16/min average

Votto, J., Bowen J. and Scalise, P. (1996) Short-stay comprehensive inpatient pulmonary rehabilitation for advanced COPD. *Arch. Phys. Med. Rehabil.*, **77**, 1115–1118.
- ↑ 12-minute distance by 66%, ADL ↑ by 39%, SOB ↓ by 65%

Young, P., Dewse, M. and Fergusson, W. (1999) Improvements in outcomes for COPD attributable to a hospital-based respiratory rehabilitation programme. *Austr. NZ J. Med.*, **29**, 59–66.
- ↓ need for steroids, ↓ hospitalisations

ADL = activities of daily living; ET = exercise tolerance; QoL = quality of life; RR = respiratory rate; SOB = shortness of breath; SOBOE = shortness of breath on exertion.

APPENDIX F: CONVERSIONS

Conversion of kilopascals (kPa) to millimetres of mercury (mmHg)
Multiply by 7.5, i.e. mmHg = kPa × 7.5.

mmHg	kPa
3.8	0.5
7.5	1.0
15	2.0
22.5	3.0
26.3	3.5
30	4.0
33.8	4.5
37.5	5.0
45	6.0
48.8	6.5
52.5	7.0
60	8.0
67.5	9.0
75	10.0
82.5	11.0
90	12.0
97.5	13.0
100	13.3
105	14.0
112	15.0
120	16.0
128	17.0
135	18.0
142	19.0
150	20.0
225	30.0
300	40.0

Conversion of millimetres of mercury (mmHg) to centimetres of water (cmH_2O):
Divide by 0.133.

mmHg	cmH_2O
5	6.8
10	13.6
20	27.2
30	40.7
40	54.3

ANNOTATED BIBLIOGRAPHY

AACVPR (1998) *Guidelines for Pulmonary Rehabilitation Programs*, 2nd edn, American Association of Cardiovascular and Pulmonary Rehabilitation, Illinois.
- Comprehensive, detailed documentation

Adam, S. K. and Osborne, S. (1997) *Critical Care Nursing: Science and Practice*, Oxford University Press, Oxford.
- Detailed anatomy and physiology, flow charts on pathology, definitions, nursing procedures explained

Aloan, C. A. and Hill, T. V. (1995) *Respiratory Care of the Newborn Child*, 2nd edn, J. B. Lippincott, Philadelphia, PA, 592 pp.
- Diseases, assessment, physiotherapy, oxygen, IPPV, home care

Anderson, I. D. (1999) *Care of the Critically Ill Surgical Patient*, Edward Arnold, London, 224 pp.
- Respiratory failure, shock, sepsis, multiple injuries, nutrition, pain management

Bird, B., Smith, A. and James, K. (1998) *Exercise Benefits and Prescription.* Stanley Thornes, Cheltenham.
- Application to different conditions

Bourke, S. J. and Brewis, R. A. L. (1998) *Lecture Notes on Respiratory Medicine*, Blackwell Science, Oxford, 216 pp.
- Accessible style, clearly written.

Bradley, D. (1998) *Hyperventilation Syndrome*, revised edn, Kyle Cathie, London.
- Readable and informative, essential reading for patients

Brostoff, J. and Gamlin, L. (1999) *Asthma: The Complete Guide.* Bloomsbury, London.
- Comprehensive guide for patients

Cairo, J. M. and Pilbeam, S. P. (1999) *McPherson's Respiratory Care Equipment*, 6th edn, Mosby, St Louis, MO.
- Clinically oriented

Corne, J., Carroll, M., Brown, I. and Delany, D. (1997) *Chest X-ray Made Easy*, Churchill Livingstone, Edinburgh.
- Pocket-sized, simple, clearly written

Croft ,T. M., Nolan, J. P. and Parr, M. J. A. (1999) *Key Topics in Critical Care*, Bios Scientific Publications, Oxford.
- Disorders, symptoms, treatments

Economou, S. G. and Economou, T. S. (1999) *Instructions for Surgical Patients*, Saunders, London.
- Information sheets to photocopy for patients

French, W. A. (2000) *Case Profiles in Respiratory Care*, 2nd edn, Delmar, Canada.
- Aimed at respiratory therapists: case studies, thought-provoking questions

Fried, R. (1999) *Breathe Well and Be Well*, John Wiley, New York
- Populist guide to mind–body interaction

Frownfelter, D. L. and Dean, E. (1996) *Principles and Practice of Cardiopulmonary Physical Therapy*, 3rd edn, Mosby, Chicago, IL.
- American approach, detailed

Goetzman, B. W. and Wennberg, R. P. (1999) *Neonatal Intensive Care Handbook*, 3rd edn, Mosby, St Louis, MO.
- Diseases, procedures, oxygen, CPAP, IPPV

Grace, P. A. and Borley, N. R. (1999) *Surgery at a Glance*, Blackwell, Oxford
- Two pages per topic, includes diagrams

Habel, A. and Scott, R. (1998) *Notes on Paediatrics: Cardiorespiratory Disease*, Butterworth-Heinemann, London.
- Concise

Hodgkin, J. E., Bartolome, R., Connors, G. L. and Celli, B. R. (2000) *Pulmonary Rehabilitation: Guidelines to Success*, 3rd edn, Lippincott Williams & Wilkins, Philadelphia, PA.

Jefferies, A. and Turley, A. (1999) *Crash Course: Respiratory System*, Mosby, London.
- Bullet lists, flow charts, condensed information, easy reading, few references

Kinnear, W. J. M. (1997) *Lung Function Tests: A Guide to their Interpretation*, Nottingham University Press, Nottingham.
- Commonly used tests plus blood gases, exercise testing, respiratory muscle function

Kinnear, W. J. M., Johnston, I. D. A. and Hall, I. P. (1999) *Key Topics in Respiratory Medicine*, Bios Scientific Publications, Oxford.
- Diseases, symptoms, treatments, in alphabetical order

Lehrer, S. (1993) *Understanding Lung Sounds*, 2nd edn, W. B. Saunders, London.
- Book and audiotape

Levitzky, M. G. (1999) *Pulmonary Physiology*, 5th edn, McGraw Hill, New York.

- Short, logical, clear

Luce, J. M., Pierson, D. J. and Tyler, M. L. (1993) *Intensive Respiratory Care*, 2nd edn, W. B. Saunders, London.
- Basic level, well written, poorly referenced

Lumb, A. B. (2000) *Nunn's Applied Respiratory Physiology*, 5th edn, Butterworth-Heinemann, London.
- Comprehensive respiratory physiology

Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford.
- Packed with ammunition to dissuade smokers

Morgan, M. and Singh, S. (1997) *Practical Pulmonary Rehabilitation*, Edward Arnold, London.
- Practical manual

Nicholls, A. and Wilson, I. (2000) *Perioperative Medicine*, Oxford University Press, Oxford.
- High-risk surgical patients with medical diseases, pocket size

Oh, T. E. (1997) *Intensive Care Manual*, 4th edn, Butterworth-Heinemann, London.
- large, international perspective, clearly written

Orenstein, D. M. (1997) *Cystic Fibrosis: A Guide for Patient and Family*, 2nd edn, Lippincott-Raven, New York.
- Pathology, airway clearance (emphasis on PD and manual techniques), organizations

Paw, H. G. W. and Park, G. R. (2000) *Handbook of Drugs in Intensive Care*, Greenwich Medical Media, London.

Payne, R. (1995) *Relaxation Techniques*, 2nd edn, Churchill Livingstone, Edinburgh.
- Practical approach covering 18 techniques

Pilbeam, S. P. (1998) *Mechanical Ventilation: Physiological and Clinical Applications*, Mosby, St Louis, MO.
- Clear descriptions, easy reading, American terminology

Pryor, J. A. and Webber, B. A. (eds) (1998) *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, Churchill Livingstone, Edinburgh.
- Particularly good on ventilators, monitors, paediatrics, hyperventilation syndrome

Ries, A. L. and Moser, K. M. (2000) *Shortness of Breath: A Guide to Better Living and Breathing*, 6th edn, Mosby, St Louis, Missouri.
- For patients, practical, lively pictures

Simonds, A. K., Muir, J. F. and Pierson, D. J. (1996) *Pulmonary Rehabilitation*, BMJ Publications, London.
- Clearly written, well-referenced, some overlap between chapters, little information on breathlessness management or ADL

Singer, M. and Webb, Q. (1997) *Oxford Handbook of Critical Care*, Oxford University Press, Oxford.
- Pocket-size, packed with facts and figures including drugs; useful to keep in the ICU

Smith, M. and Ball, V. (1998) *Cardiovascular/Respiratory Physiotherapy*, Mosby, London.
- Emphasis on the acute patient, well illustrated, good coverage of research and audit

Smith, T. (1994) *Coping with Bronchitis and Emphysema*, Sheldon, London.
- For patients, UK approach

Stillwell, S. B. (1998) *Quick Critical Care Reference*, 3rd edn, Mosby, St Louis, MO.
- Tiny book with formulae, normal values, criteria, diagrams

Sykes, K. and Young, D. (1999) *Respiratory Support in Intensive Care*, 2nd edn, BMJ Publishing, London.
- Invasive and non-invasive ventilation

Timmons, B. H. and Ley, R. (1994) *Behavioral and Psychological Approaches to Breathing Disorders*, Plenum Press, New York.
- Comprehensive information on hyperventilation syndrome

Vincent, J. L. (2000) *Yearbook of Intensive Care and Emergency Medicine*, Springer, New York.
- Latest ICU research

Weinstock, D. (ed.) (1990) *Normal and Abnormal Breath Sounds*, Springhouse Audio, Pennsylvania.
- Includes audiotape

West, J. B. (1999) *Respiratory Physiology*, 6th edn, Williams & Wilkins, Baltimore, MD.
- Detailed and clearly written

Wilkins, R. L., Hodgkin, J. E. and Lopez, B. (1996) *Lung Sounds*, 2nd edn, Mosby, Toronto
- Includes audiotape or CD

Wilkins, R. L., Krider, S. J. and Sheldon, R. L. (2000) *Clinical Assessment in Respiratory Care*, 4th edn, Mosby, Toronto.
- Physiology, pathology, assessment and monitoring, strong on clarity and definitions

Williams, S. J. (1993) *Chronic Respiratory Illness*, Routledge, London.
- Social and practical problems, symptom management

Wood, K. J. (1998) *Critical Thinking Cases in Respiratory Care*, F. A. Davies, Philadelphia, PA.
- Questions and answers, 'thought prompts', flowcharts, problem-based

References

AARC (1992) Clinical Practice Guideline: exercise testing for evaluation of hypoxaemia. *Respir. Care*, 37, 907–912.

AARC (1993a) Clinical Practice Guideline: intermittent positive pressure breathing. *Respir. Care*, 38, 1189–1194.

AARC (1993b) Clinical Practice Guideline: directed cough. *Respir. Care*, 38, 495–499.

Abbott J., Dodd, M. and Bilton, D. (1994) Treatment compliance in adults with CF. *Thorax*, 49, 115–120.

Abelson, J. L. (1996) Respiratory psychophysiology and anxiety. *Psychosomatic Med.*, 58, 302–313.

Aboussouan, L. S. (1997) Effect of noninvasive positive-pressure ventilation on survival in amyotrophic lateral sclerosis. *Ann. Intern. Med.*, 127, 450–454.

ACCP/AACVPR (1997) Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. *Chest*, **112**, 1363–1396.

Acosta, F. (1988) Biofeedback and progressive relaxation in weaning the anxious patient. *Heart Lung*, **17**, 299–301.

ACPCF (Association of Chartered Physiotherapists in Cystic Fibrosis), personal communication.

Adam, S. K. and Osborne, S. (1997) *Critical Care Nursing*. Oxford Medical.

AHFS (1999) Drug information. American Hospital Formulary Service, p. 2378.

Ahmedzai, S. (1988) Respiratory distress in the terminally ill patient. *Respir. Dis. Pract.*, 5(5), 20–26.

Ahmedzai, S. (1997) Palliation in non-malignant disease, in *Practical Pulmonary Rehabilitation*, (eds M. Morgan and S. Singh), Chapman & Hall, London.

Ahmedzai, S. and Davis, C. (1997) Nebulised drugs in palliative care. *Thorax*, 52(suppl. 2), S75–S77.

Ahrens, T. (1999a) Continuous mixed venous monitoring. *Crit. Care Nurs. Clin. North Am.*, **11**(1), 33–48.

Ahrens, T. (1999b) Pulse oximetry. *Crit. Care Nurs. Clin. North Am.*, **11**(1), 87–98.

Aiping, J. (1994) Analysis of therapeutic effects of acupuncture on abstinence from smoking. *J. Trad. Chinese Med.*, 14, 56–63.

Aitkenhead, A. R. (1989) Analgesia and sedation in intensive care. *Br. J. Anaesth.*, 63, 196–206.

Akyüz, G. (1993) Transcutaneous nerve stimulation in the treatment of pain and prevention of paralytic ileus. *Clin. Rehabil.*, 7, 218–221.

Albelda, M. (1983) Ventilator-induced subpleural air cysts. *Am. Rev. Respir. Dis.*, **127**, 360–365.

Alderson, J. D. (1999) Spinal cord injuries. *Care Crit. Ill*, **15**, 48–52.

Alessi, C. L. (1999) A randomised trial of a combined physical activity and environmental intervention in nursing home residents. *J. Am. Geriatr. Soc.*, 47, 784–791.

Alison, J. A., Donnelly, P. M. and Lennon, M. (1994) The effect of a comprehensive intensive inpatient treatment program on lung function and exercise capacity in patients with CF. *Phys. Ther.*, 74, 583–593.

Allan, A. J. (1998) Permissive hypercapnia. *Care Crit. Ill*, **14**, 233–236.

Allen, J. K. (1990) Physical and psychosocial outcomes after coronary artery bypass graft surgery. *Heart Lung*, **19**, 49–54.

Allen, S. (1996) Management of the patient with asthma. *Curr. Opin. Anaesth.*, 9, 254–258.

Allen, S. (1998) The use of the Respicair bed. *Care Crit. Ill*, **14**, 127–131.

Allen, P. W. and Hart, S. M. (1988) Minitracheotomy in children. *Anaesthesia*, 43, 760–761.

Allen, G. M., Hickie, I. and Gandevia, S. C. (1994) Impaired voluntary drive to breathe: a possible link between depression and unexplained ventilatory failure in asthmatic patients. *Thorax*, 49, 881–884.

Allen, C., Glasziou, P. and Mar, C. D. (1999) Bed rest: a potentially harmful treatment needing more careful evaluation. *Lancet*, 354, 1229–1233.

Allison, S. (1995) A study of the sociological consequences of COAD. *Physiotherapy*, **81**, 457.

Allison, S. S. and Yohannes, A. M. (1999) Consequences of COPD in community-based patients. *Physiotherapy*, 85, 663–668.

Almirall, J., Gonzalez, C. A. and Balanzo, X. (1999) Proportion of community-acquired pneumonia cases attributable to tobacco smoking. *Chest*, 116, 375–379.

Ambrosino, N., Nava, S. and Torbicki, A. (1993) Haemodynamic efects of pressure support and PEEP ventilation by nasal route in patients with stable COPD. *Thorax*, 48, 523–528.

d'Amours, R. H. (1996) Postoperative pain management. *J. Orth. Sports PT*, 24, 227–234.
Amyes, S. G. B. (1995) Antibiotic resistance in the ICU. *Br. J. Intensive Care*, 5, 263–270.
Anand, K. J. S. (1998) Analgesia and sedation for ventilated children and infants. *Respir. Care*, 43, 942–951.
Andersen, J. B., Qvist, J. and Kann, T. (1979) Recruiting collapsed lung through collateral channels with PEEP. *Scand. J. Respir. Dis.*,60, 260–266.
Andersen, J. B., Olesen, K. P. and Eikard, B. (1980) Periodic CPAP by mask in the treatment of atelectasis. *Eur. J. Respir. Dis.*, 61, 20–25.
Anderson, H. L. (1994) Extracorporeal life support for respiratory failure after multiple trauma. *J. Trauma*, 37, 266–274.
Anderson, H. R., Spix, C. and Medina, S. (1997) Air pollution and daily admissions for COPD. *Eur.Respir.J.*, 18, 1064–71.
Andersson, H. I. (1998) Widespread musculoskeletal chronic pain associated with smoking. *Scand. J. Rehabil. Med.*, 30, 185–191.
Andréasson, B., Jonson, B. and Kornfält, R. (1987) Long-term effects of physical exercise on working capacity and pulmonary function in CF. *Acta Paediatr. Scand.*, 76, 70–75.
Angus, R. M. (1996) Comparison of the acute effects on gas exchange of nasal ventilation and doxapram in exacerbations of COPD. *Thorax*, 51, 1048–1050.
Anon (1985) Neonatal intensive care, *Care Crit. Ill*, 1, 3.
Antman, K. and Chang, Y. (2000) Kaposi's sarcoma. *New. Eng. J. Med.*, 342, 1027–1038.
Anto, J. M. and Sunyer, J. (1997) Thunderstorms: a risk factor for asthma attacks. *Thorax*, 52, 669–670.
Anup, R., Aparna, V. and Pulimood, A. (1999) Surgical stress and the small intestine. *Surgery*, 125, 560–569.
Anzueto, A. (1992) Resistive breathing activates the glutathione redox cycle and impairs performance of rat diaphragm. *J. Appl. Physiol.*, 72, 529–534.
Anzueto, A. (1999) Muscle dysfunction in the ICU. *Clin. Chest Med.*, 20, 435–452.
Aono, J., Ueda, W. and Kataoka, Y. (1997) Differences in hormonal responses to preoperative emotional stress between preschool and school children. *Acta Anaesthesiol. Scand.*, 41, 229–231.
App, E. M., Kieselmann, R. and Reinhardt, D. (1998) Sputum rheology changes in CF lung disease following two different types of physiotherapy. *Chest*, 114, 171–177.
Appleyard, N. and Sherry, K. (1998) Plasmapheresis in the ICU. *Care Crit. Ill*, 14, 207–211.
Arafa, O. E., Pedersen, T. H. and Svennevig, J. L. (1998) Intraaortic balloon pump in open heart operations. *Ann. Thorac. Surg.*, 65, 741–747.
Arason, V. A. (1996) Do antimicrobials increase the carriage rate of penicillin resistant pneumococci in children? *Br. Med. J.*, 313, 387–391.
Arbour, R. (1998) Aggressive management of intracranial dynamics. *Crit. Care Nurse*, 18(3), 30–40.
Arens, R., Gozal, D. and Omlin, K. J. (1994) Comparison of high frequency chest compression and conventional chest physiotherapy in hospitalized patients with CF. *Am. J. Respir. Crit. Care Med.*, 105, 1154–1157.
Argyropoulou, P. (1993) Buspirone effect on breathlessness and exercise in patients with COPD. *Respiration*, 60, 216–220.
Aris, R. M. (1998) Increased rate of fractures and severe kyphosis: sequelae of living into adulthood with CF. *Ann. Intern. Med.*, 128, 186–193.
Aronsson, B., Perk, J. and Torstensson, O. (1996) Activities of daily living, an important target in pulmonary rehabilitation. *Eur. Respir. J.*, 9(23) 380s.
Arthur, H. M., Daniels, C. and McKelvie, R. (2000) Effect of a preoperative intervention on preoperative and postoperative outcomes. *Ann. Int. Med.*, 133, 253–262.
Artucio, H. and Pereira, M. (1990) Cardiac arrhythmias in critically ill patients. *Crit. Care Med.*, 18, 1383–1388.
Arunabh, M. D. (2000) Management of acute exacerbations in COPD. *Curr. Opin. Pul. Med.*, 6, 122–126.
Ashby, E. C. (1995) Posture, blood velocity in femoral vein and prophylaxis of DVT. *Lancet*, 345, 419–421.
Ashour, M. (1996) Hemodynamic alterations in bronchiectasis. *J. Thorac. Cardiac Surg.*, 112, 328–334.
Ashutosh, K. (1997) Clinical and personality profiles and survival in patients with COPD. *Chest*, 111, 95–98.
Askew, R., Kibelstis, C. and Overbaugh, S. (1998) Physical therapists' perception of patients' pain and its effect on management. *Physiother. Res. Int.*, 3, 37–57.

Atkinson, G. (1997) Air pollution and exercise. *Sports Exercise Injury*, 3, 2–8.

Austan, F. (1996) Heliox inhalation in status asthmaticus. *Heart Lung*, 25, 155–157.

Austin, J. H. M. and Ausubel, P. (1992) Enhanced respiratory muscular function in normal adults after lessons in proprioceptive musculoskeletal education. *Chest*, 102, 486–490.

Averns, H. L. (1996) Smoking and outcome in ankylosing spondylitis. *Scand. J. Rheumatol.*, 25, 138–142.

Axen, K. (1991) Training for strength and endurance, in *Pulmonary Therapy and Rehabilitation*, (eds F. Haas and K. Axen) Williams & Wilkins, Baltimore, MD, pp. 211 and 336.

Azar, G. (1996) Neither dopamine nor dobutamine reverses the depression in mesenteric blood flow caused by PEEP. *J. Trauma*, 40, 679–686.

Baan-Slootweg, O. H. van der (1997) Evaluation of the effects of an out-patient pulmonary rehabilitation programme for asthmatic children. *Eur. Respir. J.*, 10, 459s.

Babineau, T. J. (1994) Time to consider early gut feeding. *Crit. Care Med.*, 22, 191–192.

Bach, J. R. (1993) Airway secretion clearance by mechanical exsufflation. *Arch. Phys. Med. Rehabil.*, 74, 170–177.

Bach, J. R. (1994) Update and perspectives on noninvasive respiratory muscle aids. *Chest*, 105, 1230–40.

Bach, J. R. and Haas, F. (1996) Pulmonary rehabilitation. *Phys. Med. Clin. North Am.*, 7, 205–462.

Bach, J. R. and Moldover, J. R. (1996) Cardiovascular, pulmonary and cancer rehabilitation. *Arch. Phys. Med. Rehabil.*, 77, S45–S51.

Bachrach, L. K., Loutit, C. W. and Moss, R. B. (1994) Osteopenia in adults with cystic fibrosis. *Am. J. Med.*, 96, 27–34.

Badr, M. S., Grossman, J. E. and Weber, S. A. (1994) Treatment of refractory sleep apnea with supplemental CO_2. *Am. J. Respir. Crit. Care Med.*, 150, 561–564.

Baharloo, F. (1999) Tracheobronchial foreign bodies. *Chest*, 115, 1357–1362.

Bailey, C. R. (1995) The role of CPAP during weaning in cardiac surgical patients. *Anaesthesia*, 50, 677–681.

Baillard, C., Cohen, Y. and Fosse, J. P. (1999) Haemodynamic measurements in critically ill patients. *Anaesth. Intensive Care*, 27, 33–37.

Baldwin, D. R. (1994) Effect of addition of exercise to chest physiotherapy on sputum expectoration and lung function in adults with CF. *Respir. Med.*, 88, 49–53.

Baldwin, D. R. (1997) Non-invasive ventilation for acute exacerbations of COPD. *Br. Med. J.*, 314, 163–164.

Balfour, C. (1993) Physiotherapists and smoking cessation. *Physiotherapy*, 79, 247–250.

Balfour-Lynn, I. (1999) Difficult asthma. *Arch. Dis. Child.*, 80, 201–6.

Balfour-Lynn, I. M., Prasad, S. A. and Laverty, A. (1998) A step in the right direction: assessing exercise tolerance in CF. *Pediatr. Pulmonol.*, 25, 278–284.

Banasik, J. L. and Emerson, R. J. (1996) Effect of lateral position on arterial and venous blood gases in postoperative cardiac surgery. *Am. J. Crit. Care*, 5, 121–126.

Banner, A. S. (1994) Theophylline: should we discard an old friend? *Lancet*, 343, 618.

Banner, M. J., Kirby, R. R. and Blanch, P. B. (1996) Differentiating total work of breathing into its component parts. *Chest*, 109, 1141–1143.

Barbato, M. P. (1998) Death is a journey to be undertaken. *Med. J. Austr.*, 168, 296–297.

Bargon, J., Dauletbaev, N., Kohler, B. *et al.* (1999) Prophylactic antibiotic therapy is associated with an increased prevalence of *Aspergillus* colonization in adult cystic fibrosis patients. *Respir. Med.* 93, 835–838.

Barker, D. P. (1995) Exposure to invasive procedures in neonatal ICU admissions. *Arch. Dis. Child.*, 72, F47–F48.

Barker, M. (1998) Who are we? What are we moving? Cardiorespiratory Conference, National Heart and Lung Institute, London.

Barnard, S. and Hartigan, G. (1998) *Clinical Audit in Physiotherapy*, Butterworth-Heineman, Oxford.

Barnes, T. A. (1994) *Core Textbook of Respiratory Care Practice*, Mosby, London.

Barnes, P. J. (1995) Chronic obstructive pulmonary disease. *Update*, 51, 91–97.

Barnes, P. J. and Pauweis, R. A. (1994) Theophylline in the management of asthma. *Eur. Respir. J.*, 7, 579–591.

Barnitt, R. (1994) Truth telling in occupational

therapy and physiotherapy. *Br. J. Occup. Ther.*, 57, 334–340.
Barr, S. I. (1999) Effects of dehydration on exercise performance. *Can. J. Appl. Physiol.*, 24, 164–172.
Barratt, E. (1989) The management of pressure sores. *Intensive Ther. Clin. Monitoring*, 10, 255–259.
Barratt, T. E. (1995) Inhaled β-adrenergic receptor agonists in asthma: more harm than good? *Am. J. Respir. Crit. Care Med.*, 151, 574–577.
Barrington, K. J., Singh, A. J. and Etches, P. C. (1999) Partial liquid ventilation with and without inhaled nitric oxide in a newborn piglet model of meconium aspiration. *Am. J. Respir. Crit. Care Med.*, 160, 1922–1927.
Barry, P. W. (1994) Inhaled steroid therapy. *Curr. Paediatr.*, 4, 114–117.
Barry, P. W. (1996) Inhalational drug delivery. *Thorax*, 51, 835–840.
Bartlett, R. H., Gazzaniga, A. B. and Gerachty, T. R. (1973) Respiratory maneuvers to prevent postoperative pulmonary complications. *J. A. M. A.*, 224, 1017–1021.
Barton, J. (2000) Mother knew best. *Breathe Easy*, 35, 2.
Bartter, T. (1994) The evaluation of pleural effusion. *Chest*, 106, 1209–1214.
Basili, H. R. (1981) Effect of nutritional support on weaning patients off ventilators. *J. Parenter. Enter. Nutr.*, 5, 161–163.
Basmajian, J. V. (1998) The elixir of laughter in rehabilitation, *Arch. Phys. Med. Rehabil.*, 79, 1597.
Bass, T. L., Miller, P. K. and Campell, D. B. (1997) Traumatic adult respiratory distress syndrome. *Chest Surg. Clin. North Am.*, 7, 429–443.
Bastow, V. (1996) Personal communication.
Bastow, M. D., Rawlings, J. and Allison, S. (1983) Benefits of supplementary tube feeding after fractured neck of femur. *Br. Med. J.*, 287, 1589–1592.
Bateman, J. R. M., Newman, S. P. and Daunt, K. M. (1979) Regional lung clearance of excessive bronchial secretions during chest physiotherapy. *Lancet*, i, 294–297.
Bateman, J. R. M., Newman, S. P. and Daunt, K. M. (1981) Is cough as effective as chest physiotherapy in the removal of excessive tracheobronchial secretions? *Thorax*, 36, 683–687.
Baterman, D. and McLay, J. S. (1999) Clinical pharmacology: the basics. *Medicine*, 27(3), 1–4.
Battistini, E., Gianiorio, P. and Girosi, D. (1993) Bronchial asthma in infancy and childhood. *Eur. Respir. J.*, 3, 373–376.
Baublys, A., Kekstas, G. and Vilkelis, S. (1997) Early enteral versus parenteral nutrition. *Intensive Care Med.*, 23(suppl. 1), S139.
Baudouin, S. V. (1997) Oedema and cor pulmonale revisited. *Thorax*, 52, 401–402.
Bauer, M. L., McDougal, J. and Schoumacher, R. A. (1994) Comparison of manual and mechanical chest percussion in hospitalized patients with CF. *J. Pediatr.*, 124, 250–254.
Baun, M. M. (1984) Cumulative effects of 3 sequential endotracheal suctioning episodes. *Heart Lung*, 13, 148–154.
Baxter, J. N. (1995) Pathophysiology of laparoscopy. *Br. J. Surg.*, 82, 1–2.
Baxter, K., Nolan, K. M. and Winyard, J. A. (1993) Are they getting enough? Meeting the oxygen needs of postoperative patients. *Prof. Nurse*, 8, 310–312.
Bärtsch, R. (1999) Platelet activation with exercise and risk of cardiac events. *Lancet*, 354, 1747–1748.
Beards, S. C. and Nightingale, P. (1994) Aspiration. *Care Crit. Ill*, 10, 198–202.
Behbehani, N. A. (1999) Myopathy following mechanical ventilation for acute severe asthma. *Chest*, 115, 1627–1631.
Behera, D., Bernard, S. and Butler, J. (1995) Positive end expiratory pressure reduces bronchial blood flow after aspiration. *Respiration*, 62, 10–15.
Bein, T., Metz, C. and Keyl, C. (1996) Effects of extreme lateral posture on hemodynamics and plasma atrial natriuretic peptide levels in critically ill patients. *Intensive Care Med.*, 22, 651–655.
Bell, P. (1996) Leaking abdominal aortic aneurysms. *Care Crit. Ill*, 12, 59–63.
Bell, T. (1961) *In the Midst of Life*, Atheneum, New York.
Bellamy, D. (1997) COPD moves into the limelight. *Update*, 55, 772.
Bellet, P. S. (1995) Incentive spirometry to prevent acute pulmonary complications in sickle cell disease. *N. Engl. J. Med.*, 333, 699–703.
Bellia, V. (1993) Nocturnal asthma. *Eur. Respir. Rev.*, 3, 383–386.
Bellomo, R. (1994) Asthma requiring mechanical ventilation. *Chest*, 105, 891–896.
Belman, M. J., Brooks, L. R. and Ross, D. J. (1991) Variability of breathlessness measurement in patients with COPD. *Chest*, 99, 566–571.

Benetti, F., Mariani, M. A. and Sani, G. (1996) Video-assisted minimally invasive coronary operations without cardiopulmonary bypass. *J. Thorac. Cardiovasc. Surg.*, **112**, 1478–1484.

Bégin, P. (1991) Inspiratory muscle dysfunction and chronic hypercapnia in COPD. *Am. Rev. Respir. Dis.*, **143**, 905–912.

Benhamou, D., Muir, J. F. and Radpaud, C. (1997) Long-term efficiency of home nasal mask ventilation in patients with diffuse bronchiectasis and severe chronic respiratory failure. *Chest*, **112**, 1259–1266.

Bennett, N. T. (1991) Treatment of status asthmaticus with continuously aerosolized albuterol. *Respir. Care*, **36**, 1323.

Bennicke, K. (1995) Cigarette smoking and breast cancer. *Br. Med. J.*, **310**, 1431–1433.

Bentall, S. (1998) Assessing the risk of lower limb ischaemia in patients undergoing intraaortic balloon counterpulsation. *Care Crit. Ill*, **14**, 228–232.

Beratis, N. G. (1996) Increased BP in neonates and infants whose mothers smoked during pregnancy. *J. Pediatr.*, **128**, 806–812.

Berge, M. (1999) DNase treatment of primary ciliary dyskinesia. *Pediatr. Pulmonol.*, **27**, 59–61.

Berti, M., Fanelli, G. and Casati, A. (2000) Patient supplemented epidural analgesia after major abdominal surgery. *Can. J. Anesth.*, **47**, 27–32.

Bertone, N. (1988) The role of physiotherapy in a neonatal intensive care unit. *Austr. J. Physiother.*, **34**, 27–34.

Berwick, D. M. (1991) The double edge of knowledge. *J. A. M. A.*, **266**, 841–842.

Bestall, J. C., Paul, E. A., Garrod, R. and Garnham, R. (1999) Usefulness of the MRC dyspnoea scale as a measure of disability in patients with COPD. *Thorax*, **54**, 581–586.

Bevan, P. G. (1964) Cholecystectomy in a surgeon. *Lancet*, **i**, 214–215.

Biley, F. C. (1989) Stress in high dependency units. *Intensive Care Nurs.*, **5**, 134–141.

Billings, J.A. (2000) Palliative care. *Br. Med. J.*, **321**, 555–558.

Bilton, D., Dodd, M. E. and Abbot, J. V. (1992) The benefits of exercise combined with physiotherapy in the treatment of adults with CF. *Respir. Med.*, **86**, 507–511.

Bio, M. (1998) Positive pressure ventilation in amyotrophic lateral sclerosis [letter]. *Ann. Intern. Med.*, **128**, 601.

Bird, J. (1997) Intensive care problems in obstetric patients. *Care Crit. Ill*, **13**, 241–244.

Biro, P. (1997) Comparison of topical anaesthesia methods for venous cannulation in adults. *Eur. Resp. J.*, **1**, 37–42.

Bischoff, H. A., Stahelin, H. B. and Urscheler, N. (1999) Muscle strength in the elderly: its relation to vitamin D metabolites. *Arch. Phys. Med. Rehabil.*, **80**, 54–58.

Bisgaard, H. (2000) Long-acting β_2-agonists in management of childhood asthma. *Ped. Pulmonol.*, **29**, 221–234.

Bisson, A., Stern, M. and Caubarrere, I. (1998) Preparation of high-risk patients for major thoracic surgery. *Chest Surg. Clin. North Am.*, **8**, 541–555.

Black, D. (1998) The dying child. *Br. Med. J.*, **316**, 1376–1378.

Blackie, K. A., Davidson, A. C. and Marsh, M. J. (1999) 'Not for resuscitation': guidelines for decision making and documentation. *Int. J. Clin. Pract.*, **53**, 213–216.

Blair, P. S. (1996) Smoking and the sudden infant death syndrome. *Br. Med. J.*, **313**, 195–198.

Blanckenhagen, J. (1986) Unheard victims of breast cancer. *The Independent*, 7 October.

Block, S. D. (2000) Assessing and managing depression in the terminally ill patient. *Ann. Intern. Med.*, **132**, 209–218.

Blower, A. C. (1997) Is thirst associated with disability in hospital inpatients? *J. Hum. Nutr. Dietetics*, **10**, 289–293.

Bluman, L. G., Mosca, L. and Newman, N. (1998) Preoperative smoking and postoperative pulmonary complications. *Chest*, **113**, 883–889.

British Medical Journal (1997) Passive smoking [editorial], *Br. Med. J.*, **315**, 961–962.

Boezen, H. M. (1998) Breathlessness in elderly individuals is related to low lung function and reversibility of airway obstruction. *Eur. Respir. J.*, **12**, 805–810.

Boghdadi, M. S. (1997) Cocaine: pathophysiology and clinical toxicology. *Heart Lung*, **26**, 466–483.

Bohin, S. (1995) Circulatory effects of ventilator and end-expiratory pressure in unparalysed preterm infants. *Acta Paediatr.*, **84**, 1300–1304.

Bolgiano, C. S. (1990) Administering oxygen therapy. *Nursing*, **20**, 47–51.

Bolliger, C. T. (1998) Functional evaluation of the lung resection candidate. *Eur. Respir. J.*, **11**, 198–212.

Bonnet, R. (1995) Intractable cough associated with the supine body position. *Chest*, **108**, 581–585.
Bonita, R. (1999) Passive smoking as well as active smoking increases the risk of acute stroke. *Thorax*, 8, 156–160.
Boots, R. J., Howe, S. and George, N. (1997) Clinical utility of hygroscopic heat and moisture exchangers in intensive care patients. *Crit. Care Med.*, 25, 1707–1712.
Borak, J., Sliwinski, P. and Piasecki, Z. (1991) Psychological status of COPD patients on long term oxygen therapy. *Eur. Respir. J.*, 4, 59–62.
Borg, G. A. V. (1982) Psychophysiological bases of perceived exertion. *Med. Sci. Sports Exercise*, 14, 377–381.
Bortoluzzi, G. (1994) Facing death: a personal perspective. *NZJ. Physiother.*, 22, 6–9.
Borzotta, A. P. (1994) Enteral versus parenteral nutrition after severe head injury. *J. Trauma*, 37, 459–468.
Bosley, C. M. (1996) Psychological factors associated with use of home nebulized therapy for COPD. *Eur. Respir. J.*, 9, 2346–2350.
Bosscha, K., Niewenhujis, V. B. and Vos, A. (1998) Gastrointestinal motility and gastric tube feeding in mechanically ventilated patients. *Crit. Care Med.*, 26, 1510–1517.
Bostick, J. and Wendelgass, S. T. (1987) Normal saline instillation as part of the suctioning procedure. *Heart Lung*, **16**, 532–536.
Bosworth, D. G. and Nielson, D. W. (1997) Effectiveness of home versus hospital care in the routine treatment of CF. *Pediatr. Pulmonol.*, 24, 42–47.
Botman, M. J. (1987) Contamination of small-volume medication nebulizers. *J. Hosp. Infect.*, **10**, 204–208.
Bott, J., Keilty, S. E. J. and Brown, A. (1992) Nasal intermittent positive pressure ventilation. *Physiotherapy*, 78, 93–96.
Botterill, I. and MacFie, J. (2000) Bacterial translocation in the critically ill. *Care Crit. Ill*, **16**, 6–10.
Boulet, L.-P., Milot, J. and Gagnon, L. (1999) Long-term influence of inhaled corticosteroids on bone metabolism and density. *Am. J. Respir. Crit. Care Med.*, 159, 838–844.
Bouley, G. H. (1992) The experience of dyspnea during weaning. *Heart Lung*, **21**, 471–476.
Böllert, F. G. E., Paton, J. Y. and Marshall, T. G. (1999) Recombinant DNase in CF. *Eur. Respir. J.*, 13, 107–113.
Bourdelles, G. L., Mier, L. and Fiquet, B. (1996) Comparison of the effects of heat and moisture exchangers and heated humidifiers. *Chest*, **110**, 1294–1298.
Bourke, S. and Boyd, G. (1997) Pigeon fancier's lung. *Br. Med. J.*, **315**, 70–71.
Bower, L. K. (1995) Extracorporeal life support and high-frequency oscillatory ventilation. *Respir. Care*, 40, 61–73.
Bowes, D. (1984) The doctor as patient: an encounter with Guillain–Barré syndrome. *Can. Med. Assoc. J.*, **131**, 1343–1368.
Bowler, S. D., Green, A. and Mitchell, C. A. (1998) Buteyko breathing techniques in asthma: a randomised controlled trial. *Med. J. Austr.*, **169**, 575–578.
Bowton, D. L. (1999) On patients and patience. *Crit. Care Med.*, **27**, 2573.
Boyd, S. (1994) Evaluation of the literature on the effectiveness of physical therapy modalities in the management of children with CF. *Pediatr. Phys. Ther.*, 6, 70–74.
Boylan, A. M. (2000) Mesothelioma. *Curr. Opin. Pulm. Med.*, 6, 157–163.
Bracken, M. B., Shepard, M. J. and Holford, T. R. (1997) Administration of methylprednisolone for acute spinal cord injury. *J. A. M. A.*, 277, 1597–1504.
Bradding, P., Rushby, I. and Scullion, J. (1999) As-required versus regular nebulized salbutamol for the treatment of acute severe asthma. *Eur. Respir. J.*, **13**, 290–294.
Bradley, T. D. (1993) Unexpected presentations of sleep apnoea. *Br. Med. J.*, **306**, 1260–1262.
Bradley, D. (1994) *Hyperventilation Syndrome*, Kyle Cathie, London.
Bradley, J., Howard, J. and Wallace, E. (1999) Validity of a modified shuttle test in adult cystic fibrosis. *Thorax*, 54, 437–439.
Brandsma, J. W., Robeer, B. G. and van den Heuvel, S. (1998) The effect of exercises on walking distance of patients with intermittent claudication. *Phys. Ther.*, 78, 278–288.
Brandstetter, R., Grant, G. R. and Estillo, M. (1998) Swan-Ganz catheter: misconceptions, pitfalls, and incomplete user knowledge. *Heart Lung*, 27, 218–222.
Brannon, F. J., Foley, M. W. and Starr, J. A. (1998) *Cardiopulmonary Rehabilitation: Basic Theory and Application*, F. A. Davis, Philadelphia, PA.
Branson, R. D. (1993) The nuts and bolts of

increasing oxygenation. *Respir. Care*, 38, 672–689.
Branson, R. D. (1996) Comparison of three humidification techniques during mechanical ventilation. *Respir. Care*, **41**, 809–816.
Branson, R. D. (1999) Humidification for patients with artificial airways. *Respir. Care*, 44, 630–641.
Branson, R. D., Davis, K. and Campbell, R. S. (1993) Humidification in the ICU. *Chest*, **104**, 1800–1805.
Branthwaite, M. A. (1985) The intensive care of asthma. *Br. J. Hosp. Med.*, 34, 1–8.
Branthwaite, M. A. (1996) Witholding or withdrawing intensive care. *Br. J. Intensive Care*, 6, 51–54.
Brashear, R. E. (1983) Hyperventilation syndrome. *Lung*, **161**, 257–273.
Brath, H., Lahrmann, H. and Wanke, T. (1997) The effect of lung transplantation on the neural drive to the diaphragm in patients with severe COPD. *Eur. Respir. J.*, 10, 424–429.
Braun, N. M. T., Faulkner, J. and Hughes, R. L. (1983) When should respiratory muscles be exercised? *Chest*, 84, 76–84.
Breslin, E. H. (1992) The pattern of respiratory muscle recruitment during pursed lip breathing. *Chest*, **101**, 75–78.
Breslin, E. H. (1996) Respiratory muscle function in patients with COPD. *Heart Lung*, 25, 271–285.
Breslin, E. H., Garoutte, B. C. and Carrieri-Kohlman, V. (1990) Correlations between dyspnea, diaphragm and sternomastoid recruitment during inspiratory breathing. *Chest*, 98, 298–302.
Breslin, E., van der Schans, C. and Breukink, S. (1998) Perception of fatigue and quality of life in patients with COPD. *Chest*, **114**, 958–964.
Brett, A. and Sinclair, D. G. (1993) Use of continuous positive airway pressure in the management of community acquired pneumonia. *Thorax*, 48, 1280–1281.
Brevinge, H. (1995) Exercise capacity in patients undergoing proctocolectomy and small bowel resection for Crohn's disease. *Br. J. Surg.*, 82, 1040–1045.
Brewin, A. (1998) Allergy avoidance. *Nurs. Stand.*, **12**(32), 49–53.
Briars, G. and Warner, J. (1993) Cystic fibrosis. *Practitioner*, 237, 765–770.
Bright, L. D. (1995) Deep vein thrombosis. *Am. J. Nurs.*, 95, 48–49.
Bright, P. (1996) The diagnosis of occupational asthma. *Thorax*, **51**, 857–863.
Brimioulle, S., Moraine, J.-J. and Norrenberg, D. (1997) Effects of positioning and exercise on ICP in a neurosurgical ICU. *Phys. Ther.*, 77, 1682–1689.
Brismar, B. (1985) Pulmonary densities during anesthesia with muscular relaxation. *Anesthesiogy*, 62, 422–428.
Britton, J. (2000) Interferon gamma-1b therapy for cryptogenic fibrosing alveolitis. *Thorax*, 55 (suppl.1), 537–540.
Broad, S. H. (1991) An holistic approach to cardiac rehabilitation. *NZJ. Physiother.*, 19, 6–10.
Brochard, L., Mion, G. and Isabey, D. (1991) Constant-flow insufflation prevents arterial oxygen desaturation during suctioning. *Am. Rev. Respir. Dis.*, 144, 395–400.
Brodner, G. (1997) Pain management in patients undergoing thoracic surgery. *Curr. Opin. Anaesth.*, 10, 54–59.
Bromley, I. (1998) *Tetraplegia and Paraplegia*, Churchill Livingstone, Edinburgh.
Brooks, D. H. M. (1990) The route to home ventilation. *Care Crit. Ill*, 6, 96–97.
Brooks-Brunn, J. A. (1995) Postoperative atelectasis and pneumonia. *Heart Lung*, 24, 94–112.
Broomhead, C. J. (1995) Physiology of postoperative nausea and vomiting. *Br. J. Hosp. Med.*, 53, 327–330.
Brown, S. E. (1983) Prevention of suction-related arterial oxygen desaturation. *Chest*, 83, 621–628.
Brown, K. K. (1994) Septic shock. *Am. J. Nurs.*, 94, 20–27.
Brown, M. A. and Halonen, M. (1999) Perinatal events in the development of asthma. *Curr. Opin. Pulmonary Med.*, 5, 4–9.
Browning, D. (1992) Treatment of acute severe asthma assisted by hyperthermia. *Anaesthesia*, 47, 223–225.
Brownlee, K. G. (1997) Paediatric bronchoscopy. *Arch. Dis. Child.*, 77, 272–275.
Brucia, J. (1996) The effect of suction catheter insertion and tracheal stimulation in adults with severe brain injury. *Heart Lung*, 25, 295–303.
Bruder, N. (1998) Influence of body temperature, with or without sedation, on energy expenditure in severe head-injured patients. *Crit. Care Med.*, 26, 568–572.
Bruera, E. (1997) Cancer pain management. *Acta Anaesth. Scand.*, 41, 146–153.

Bruera, E. (1998) Proctolysis for dehydration of terminally ill cancer patients. *J. Pain Symptom Man.*, **15**, 216–219.

Bruera, E., deStoutz, N. and Velasco, A. (1993) Effects of oxygen on dyspnoea in hypxaemic terminal cancer patients. *Lancet*, **342**, 13–14.

Brusasco, V. and Fitting, J. W. (1998) Lung hyperinflation in airway obstruction. *Eur. Respir. J.*, **9**, 2440.

Bruton, A., Conway, J. and Holgate, S. T. (1999) Weaning adults from mechanical ventilation. *Physiotherapy*, **85**, 652–661.

Bryce, T. N. (2000) Pain after spinal cord injury. *Phys. Med. Rehab. Clin. N. Am.*, **11**, 157–168.

BTS (1990) Guidelines for management of asthma in adults. *Br. Med. J.*, **301**, 797–800.

BTS (1993) Guidelines for management of asthma: a summary. *Br. Med. J.*, **306**, 776–782.

BTS (1994) Guidelines for the measurement of respiratory function. *Respir. Med.*, **88**, 165–194.

BTS (1997) Instructions for patients using home nebulizers. *Thorax*, **52**(suppl. 2), S17.

Buccheri, G. (1998) Depressive reactions to lung cancer are common and often followed by poor outcome. *Eur. Respir. J.*, **11**, 173–178.

Buckley, F. P. (1997) Anesthetic risks related to obesity. *Curr. Opin. Anaesth.*, **10**, 240–243.

Buckman, R. (1996) Talking to patients about cancer. *Br. Med. J.*, **313**, 699–700.

Buff, D. D., Bavli, S. Z. and Bloch, M. H. (1995) Circadian patterns of emergency asthma presentations. *Respir. Care*, **40**, 624–630.

Burdet, L. (1997) Thermogenic effect of bronchodilators in patients with COPD. *Thorax*, **52**, 130–135.

Burdon, J. G. W. (1994) Chronic lung disease and the perception of breathlessness. *Eur. Respir. J.*, **7**, 1342–1349.

Burford, B. W. and Barton, S. J. (1998) Care of the dying patient, in *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, (eds B. A. Webber and J. A. Pryor), Churchill Livingstone, Edinburgh.

Burge, P. S. (1992) Peak flow measurement. *Thorax*, **47**, 903.

Burke, R. P. (1998) Minimally invasive pediatric cardiac surgery. *Curr. Opin. Pediatr.*, **10**, 527–531.

Burns, S. P. (2000) Sleep apnea syndrome in chronic spinal cord injury. *Arch. Phys. Med. Rehab.*, **81**, 1334–1339.

Burns, S. M., Spillman, M. and Wilmoth, D. (1998) Are frequent inner cannula changes necessary? *Heart Lung*, **27**, 58–62.

Burstal, R., Wegener, F. and Hayes, C. (1998) Subcutaneous tunnelling of epidural catheters. *Anaesth. Intensive Care*, **26**, 147–151.

Bury, T. J. and Mead, J. M. (1998) *Evidence-based Healthcare: A Practical Guide for Therapists.* Butterworth-Heineman, London.

Bush, A., Cole, P. and Hariri, M. (1998) Primary ciliary dyskinesia: diagnosis and standards of care. *Eur. Respir. J.*, **12**, 982–988.

Busse, W. W. (1995) Stress and asthma. *Am. J. Respir. Crit. Care Med.*, **151**, 249–252.

Butland, B. K., Strachan, D. P. and Anderson, H. R. (1999) Fresh fruit intake and asthma symptoms in young British adults. *Eur. Respir. J.*, **13**, 744–750.

Butler, S. G. and Sutherland, R. J. (1998) Current airway clearance techniques. *NZ Med. J.*, **111**, 183–186.

Button, B. M., Heine, R. G. and Catto, A. G. (1994) Postural drainage exacerbates gastroesophageal reflux in patients with lung disease. *Pediatr. Res.*, **36**, 2.

Bye, P. T. P., Alison, J. A. and Regnis, J. A. (1997) Exercise performance and rehabilitation in cystic fibrosis. *Crit. Rev. Phys. Rehabil. Med.*, **9**, 1–33.

Bynum, L. J., Wilson, J. E. and Pierce, A. K. (1976) Comparison of spontaneous and positive-pressure breathing in supine normal subjects. *J. Appl. Physiol.*, **41**, 341–346.

Cade, A., Brownlee, K. G. and Conway, S. P. (2000) Randomised placebo controlled trial of nebulized corticosteroids in acute respiratory syncytial viral bronchiolitis. *Arch. Dis. Child.*, **82**, 126–130.

Cahalin, L. P. (1996) Heart failure. *Phys. Ther.*, **76**, 516–533.

Cahalin, L. P. (1997) Inspiratory muscle training in patients with chronic heart failure awaiting cardiac transplantation. *Phys. Ther.*, **77**, 830–838.

Cahalin, L. P. and Sadowsky, H. S. (1995) Pulmonary medications. *Phys. Ther.*, **75**, 397–414.

Caldwell, M. T. P. (1993) Timing of extubation after oesophagectomy. *Br. J. Surg.*, **80**, 1537–1539.

Callaghan, S. (1998) Evaluating two dressings for the prevention of nasal bridge pressure sores. *Prof. Nurse*, **13**, 361–364.

Callegari, G. (1994) Clinical effects of oscillating positive expiratory pressure. *Respir. Care*, **39**, 1104.

Calverley, P. and Bellamy, D., (2000) The challenge

of providing better care for patients with COPD. *Thorax*, 55, 78–82.
Calvo, V., Borro, J. M. and Morales, P. (1999) antifungal prophylaxis during the early postoperative period of lung transplantation. *Chest*, 115, 1301–1304.
Cambach, W. (1997) The effects of a community-based pulmonary rehabilitation programme on exercise tolerance and quality of life. *Eur. Respir. J.*, 10, 104–113.
Cameron, I. R. and Bateman, N. T. (1990) *Respiratory Disorders*, Edward Arnold, London, p. 75.
Cammarano, W. B., Pittet, J.-F. and Weitz, S. (1998) Acute withdrawal syndrome related to the administration of analgesic and sedative medications. *Crit. Care Med.*, 26, 676–684.
Campbell, J. (1999a) Cannabis: the evidence. *Nurs. Stand.*, 13, 45–47.
Campbell, R. S. (1999b) Extubation and the consequences of reintubation. *Respir. Care*, 44, 799–803.
Campbell, E. J. M. and Howell, J. B. L. (1963) The sensation of dyspnoea. *Br. Med. J.*, ii, 868.
Campbell, E. J., Baker, M. D. and Crites, P. (1988) Subjective effects of humidification of oxygen for delivery by nasal cannula. *Chest*, 93, 289–293.
Campbell, D., Steinmann, M. and Porayko, L. (2000) Nitric oxide and high frequency jet ventilation in a patient with bilateral bronchopleural fistulae and ARDS. *Can. J. Anaesth.*, 47, 53–57.
Cano, A. and Payo, F. (1997) Lung function and airway responsiveness in children and adolescents after hyaline membrane disease. *Eur. Respir. J.*, 10, 880–885.
Carella, M. J. (1999) Improvement in pulmonary and exercise performance in obese patients after weight loss. *Respir. Care*, 44, 1458–1464.
Carew, J. F., Kraus, D. H. and Ginsberg, R. J. (1999) Early complications: recurrent nerve palsy. *Chest Surg. Clin. North Am.*, 9, 597–608.
Carmichael, R. (1981) Personal view. *Br. Med. J.*, 282, 1388.
Carmini, V., Damignani, R. and Brooks, D. (2000) Preoperatie physiotherapy teaching in paediatric cardiac patients. *Physiother. Can.*, 42, 312–314.
Carone, M. (1997) Comparison of invasive and noninvasive saturation monitoring in prescribing oxygen during exercise. *Eur. Respir. J.*, 10, 446–451.
Carr, L. (1993) Manual therapy for patients with respiratory disease. *ACPRC J.*, 23, 13–15.
Carr, D. B. (1996) Pain therapy. *Curr. Opin. Anaesth.*, 9, 415–420.
Carr, L., Pryor, J. A. and Hodson, M. E. (1995) Self chest clapping. *Physiotherapy*, 81, 753–757.
Carrieri, V. (1992) Exercise may result in greater decreases in anxiety and distress associated with dyspnea than in dyspnea intensity. *Am. Rev. Respir. Dis.*, 145, A765.
Carroll, S. (1989) Antibiotics and respiratory infection. *Respir. Dis. Pract.*, 6, 29.
Carson, M. M. (1994) Managing pain during mediastinal chest tube removal. *Heart Lung*, 23, 500–505.
Carter, B. (1995) An asthmatic in 'Wonderland': a patient's perspective. *Accident Emerg. Nurs.*, 3, 139–141.
Carter, A. B. and Hornick, D. B. (1999) Therapy for ventilator-associated pneumonia. *Clin. Chest Med.*, 20, 681–691.
Casaburi, R. (1992) Principles of exercise training. *Chest*, 101, 263S–267S.
Cassel, W., Plock, T. and Becker, C. (1996) Risk of traffic accidents in patients with sleep-disordered breathing. *Eur. Respir. J.*, 9, 2606–2611.
Castledine, G. (1993) A personal view of asthma. *Br. J. Nurs.*, 2, 919.
Castling, D. (1995) Neonatal endotracheal suction. *Br. J. Int. Care*, 5, 218–221.
Cazzola, M. (1998) Cardiac effects of formoterol and salmeterol in patients suffering from COPD with preexisting cardiac arrhythmias and hypoxemia. *Chest*, 114, 411–415.
Celli, B. (1993) Respiratory muscle strength after upper abdominal surgery. *Thorax*, 48, 683–684.
Celli, B. R. (1994) Physical reconditioning of patients with respiratory diseases. *Respir. Care*. 39, 481–499.
Celli, B. R., Rodriguez, K. S. and Snider, G. L. (1984) A controlled trial of IPPB, incentive spirometry and deep breathing exercises after abdominal surgery. *Am. Rev. Respir. Dis.*, 130, 12–15.
Cereda, M. (1996) Positive end-expiratory pressure prevents the loss of respiratory compliance during low tidal volume ventilation. *Chest*, 109, 480–485.
Cerveira, J. J., Halpern, V. J., Faust, G. and Cohen, J. R. (1999) Minimal incision abdominal aortic aneurysm repair. *J. Vasc. Surg.*, 30, 977–984.
Chambliss, R. (1997) Pain management in the pediatric ICU. *Curr. Opin. Pediatr.*, 9, 246–253.
Chan, C. S. (1990) Eucapnia and hypercapnia in

patients with chronic airflow limitation. *Am. Rev. Respir. Dis.*, **141**, 861–865.
Chang, S.-C., Shiao, G.-M. and Perng, R.-P. (1989) Postural effect on gas exchange in patients with unilateral pleural effusions. *Chest*, **96**, 60–63.
Charbonneau, M. (1994) Changes in obstructive sleep apnea characteristics through the night. *Chest*, **106**, 1695–1701.
Charlton, A. and White, D. (1996) Smoking and menstrual problems in 16-year-olds. *J. Roy. Soc. Health*, **89**, 193–195.
Chatburn, R. L. (1991) Principles and practice of neonatal and pediatric mechanical ventilation. *Respir. Care*, **36**, 569–595.
Chatham, K. (1995) Respiratory muscle training. *Br. J. Ther. Rehabil.*, **2**, 31–36.
Chatham, K. (2000) Individualised fixed load inspiratory muscle training responses in a patient with severe restrictive lung disease and an elite sportsman. *Physiotherapy*, **86**, 28–30.
Chatham, K., Berrow, S. and Beeson, C. (1994) Inspiratory pressures in adult cystic fibrosis. *Physiotherapy*, **80**, 748–752.
Chatila, W., Hall, J. B. and Manthous, C. A. (1995) The effect of pulmonary secretions on respiratory mechanics in intubated patients. *Respir. Care*, **40**, 1048–1051.
Cheever, K. H. (1999) Reducing the effects of acute pain in critically ill patients. *Dimens. Crit. Care Nurse*, **18**, 14–23.
Chen, Y., Horne, S. L. and Dosman, J. A. (1993) Body weight and weight gain related to pulmonary function decline. *Thorax*, **48**, 375–380.
Chen, Y., Rennie, D. C. and Lockinger, L. A. (1998) Effect of environmental tobacco smoke on cough in children with a history of tonsillectomy or adenoidectomy. *Eur. Respir. J.*, **11**, 1319–1323.
Cherny, N. I. and Foley, K. M. (1997) Nonopioid and opioid analgesic pharmacotherapy of cancer pain. *Otolaryngol. Clin. North Am.*, **30**, 281–304.
Chiswick, M. L. (1999) Assessment of pain in neonates. *Lancet*, **355**, 6–7.
Chitnavis, B. P. (1998) Intracranial pressure monitoring. *Care Crit. Ill*, **14**, 80–84.
Choi, P. T. L., Yip, G. and Quinonez, L. G. (1999) Crystalloids vs. colloids in fluid resuscitation. *Crit. Care Med.*, **27**, 200–210.
Christen, W. G. (1992) A prospective study of cigarette smoking and risk of cataract in men. *J. A. M. A.*, **268**, 989–993.
Christen, W. G. (1996) A prospective study of cigarette smoking and risk of age-related macular degeneration in men. *J. A. M. A.*, **266**, 1147–1151.
Christensen, T. and Kehlet, H. (1984) Postoperative fatigue and changes in nutritional status. *Br. J. Surg.*, **71**, 473–476.
Christensen, E. F., Nedergaard, T. and Dahl, R. (1990) Long-term treatment of chronic bronchitis with PEP mask and chest physiotherapy. *Chest*, **97**, 645–650.
Christensson, K. (1995) Separation distress call in the human neonate in the absence of materal body contact. *Acta Paediatr.*, **84**, 468–473.
Christie, B. (2000) Premature babies have high death and disability rate. *Br. Med. J.*, **321**, 467.
Chudley, S. (1994) The effect of nursing activities on ICP. *Br. J. Nurs.*, **3**, 454–459.
Chulay, M. (1988) Arterial blood gas changes with a hyperinflation and hyperoxygenation suctioning intervention. *Heart Lung*, **17**, 654–661.
Chumillas, S., Ponce, J. L. and Delgado, F. (1998) Prevention of postoperative pulmonary complications through respiratory rehabilitation. *Arch. Phys. Med. Rehabil.*, **79**, 5–9.
Chung, D. C. (1992). Partially paralyzed: a personal experience [letter]. *Anesthesiology*, **77**, 1053–1054.
Church, S. E. (1991) Bronchoconstricton caused by humidified oxygen in asthmatic patients. *Thorax*, **46**, 4.
Chuter, T. A. M. (1990) Diaphragmatic breathing maneuvers and movement of the diaphragm after cholecystectomy. *Chest*, **97**, 1110–1114.
Cichero, J. A. Y. and Murdoch, B. E. (1998) The physiologic cause of swallowing sounds. *Dysphagia*, **13**, 39–52.
Ciesla, N. D. (1994) Chest physical therapy for the adult ICU trauma patient. *Phys. Ther. Pract.*, **3**, 92–108.
Ciesla, N. D. (1996) Chest physical therapy for patients in the ICU. *Phys. Ther.*, **76**, 609–625.
Clague, J. E. and Calverley, P. M. A. (1990) Management of COPD. *Hosp. Update*, **16**, 20–32.
Clark, K. J. (1985) Coping with Guillain–Barré syndrome. *Intensive Care Nurs.*, **1**, 13–18.
Clark, C. J. (1996) Is pulmonary rehabilitation effective for patients with COPD? *Lancet*, **348**, 1111.
Clark, C. J. (1999) Physical activity and asthma. *Curr. Opin. Pulmonary Med.*, **5**, 68–75.

Clark, C. J. and Cochrane, L. (1999) Physical activity and asthma. *Curr. Opin. Pulmonary Med.*, 5, 68–75.

Clark, C. J., Cochrane, L. and Mackay, E. (1996) Low intensity peripheral muscle conditioning improves exercise tolerance and breathlessness in COPD. *Eur. Respir. J.*, 8, 2590–2596.

Clarke, S. W. (1989) Rationale of airway clearance. *Eur. Respir. J.*, 2(suppl. 7), 599S-604S.

Clarke, S. W. (1991) Chronic bronchitis in the 1990s. *Respiration*, 58(suppl.), 43–6.

Clarke, J. P., Schuitemaker, M. N. and Sleigh, J. W. (1998) The effect of intraoperative ventilation strategies on perioperative atelectasis. *Anaesth. Intensive Care*, 26, 262–266.

Claxton, A. R. (1997) Evaluation of morphine versus fentanyl. *Anesth. Analg.*, 84, 509–514.

Cleland, J. G. F. (1996) Heart failure. *Update*, 52, 658–663.

Clemency, M. V. (1997) Do not resuscitate orders in the perioperative period. *Anesth. Analg.*, 84, 859–864.

Clifton-Smith, T. (1999) *Breathe to Succeed*, Penguin, New Zealand.

Clini, E., Vitacca, M. and Bianchi, L. (1999) Long-term tracheostomy in severe COPD patients. *Respir. Care*, 44, 415–420.

Closs, S. J. (1996) Pain and elderly patients. *J. Adv. Nurs.*, 23, 237–242.

Coates, A. L. (1992) Oxygen therapy, exercise and cystic fibrosis. *Chest*, 101, 2–4.

Cobb, J. M. T. (1996) Psychosocial stress and susceptibility to upper respiratory tract illness. *Psychosomatic Med.* 58, 408–412.

Cochrane, G. M. (1995) Acute severe asthma. *Thorax*, 50, 1–2.

Cochrane, G. M. (1997) Compliance with nebulized therapy. *Eur. Respir. Rev.*, 7, 383–384.

Cochrane, G. M., Webber, B. A. and Clarke, S. W. (1977) Effects of sputum on pulmonary function. *Br. Med. J.*, ii, 1181–1183.

Cockcroft, D. W., McParland, C. P. and Britto, S. A. (1993) Regular inhaled salbutamol and airway responsiveness to allergen. *Lancet*, 342, 833–836.

Cockram, J., Jenkins, S. and Clugston, R. (1999) Cardiovascular and respiratory responses to early ambulation and stair climbing following coronary artery surgery. *Physiother. Theory Pract.*, 15, 3–15.

Coffman, J. A. and McManus, K. P. (1984) Oxygen therapy via nasal catheter for infants with BPD. *Crit. Care Nurse*, 4, 22–23.

Coghill, C. H., Haywood, J. L. and Chatburn, R. L. (1991) Neonatal and pediatric high-freguency ventilation. *Respir. Care*, 36, 596–603.

Cogswell, J. J. (1994) Epidemiology of asthma. *Curr. Paediatr.*, 4, 139–142.

Cohen, D., Horiuchi, K. and Kemper, M. (1996) Modulating effects of propofol on metabolic and cardiopulmonary responses to stressful ICU procedures. *Crit. Care Med.*, 24, 612–617.

Coirault, C. (1999) Relaxation of the diaphragm muscle. *J. Appl. Physiol.*, 87, 1243–1252.

Cole, P. (1999) Cardiorespiratory physiotherapy conference, London.

Cole, J. G., Begish, A. and Judas, M. L. (1990) Changing the NICU environment. *Neonat. Netw.*, 9, 15–23.

Collignon, P. (1994) Intravascular associated sepsis. *Med. J. Austr.*, 161, 374–378.

Collins, J. (1976) Blood gases, rational oxygen therapy, air flow limitations. *Physiotherapy*, 62, 48–52.

Collins, M. A. (1994) When your patient has an implantable cardioverter defibrillator. *Am. J. Nurs.*, 94, 34–39.

Collins, F. (1999) The contribution made by an armchair with integral pressure-reducing cushion in the prevention of pressure sore incidence in the elderly acutely ill patient. *J. Tissue Viability*, 9, 133–137.

Confalonieri, M. (1994) Severe exacerbations of COPD treated with BiPAP. *Respiration*, 61, 310–316.

Connor, D. F. J. and Muir, J. (1998) Balanced analgesia for the management of pain in an opioid addict. *Anaesth. Intensive Care*, 26, 459–460.

Connors, A. F. (1996) Outcomes following acute exacerbation of severe COPD. *Am. J. Respir. Crit. Care Med.*, 154, 959–967.

Connors, A. F., Hammon, W. E. and Martin, R. J. (1980) Chest physical therapy: the immediate effect on oxygenation in acutely ill patients. *Chest*, 78, 559–564.

Consroe, P., Musty, R. and Rein, J. (1997) The perceived effects of smoked cannabis on patients with multiple sclerosis. *Eur. Neurol.*, 38, 44–48.

Conway, J. (1992a) Chest physiotherapy – the way forward. *Physiotherapy*, 78, 77–78.

Conway, J. (1992b) The effects of humidification for patients with chronic airways disease. *Physiotherapy*, 78, 97–101.

Conway, S. P. (1997) Recombinant human DNase in CF. *Arch. Dis. Child.*, 77, 1–3.

Conway, S. P. and Littlewood, J. M. (1997) rhDNase in CF. *Br. J. Hosp. Med.*, 57, 371–372.
Cook, T. M. (1997) Epidural analgesia following upper abdominal surgery. *Acta Anaesthesiol. Scand.*, 41, 18–24.
Cook, D. J. (1998) Incidence of and risk factors for ventilator-associated pneumonia. *Ann. Intern. Med.*, 129, 433–440.
Cooper, C. (1995) Rheumatoid arthritis, corticosteroid therapy and hip fracture. *Ann. Rheum. Dis.*, 54, 49–52.
Cooper, A. B. (1996) Pulmonary artery catheters in the critically ill. *Crit. Care Clin.*, 12, 777–793.
Copestake, A. J. and McConnell, A. (1994) Inspiratory muscle training reduces exertional breathlessness in healthy elderly men and women. II Egrepa Conference Proceedings, Scotland.
Cormier, Y., Laviolette, M. and Atton, L. (1991) Influence of lung volume on collateral resistance. *Respir. Physiol.*, 83, 179–188.
Corrado, A. (1996) Intermittent negative pressure ventilation in the treatment of hypoxic hypercapnic coma in chronic respiratory insufficiency. *Thorax*, 51, 1077–1082.
Corris, P. A. (1990) Respiratory failure in COPD. *Int. Ther. Clin. Monit.*, 11, 2, 50–54.
Corris, P. A. and Dark, J. H. (1993) Aetiology of asthma. *Lancet*, 341, 1369–1371.
Corsico, R. (1993) Anti-asthma drugs and quality of life. *Eur. Respir. Rev.*, 3, 366–368.
Cosgrove, M. (1997) Experience of percutaneous endoscopic gastrostomy in children with Crohn's disease. *Arch. Dis. Child.*, 76, 141–143.
Costello, C. M. (1996) Effect of nebulized recombinant DNase on neutrophil elastase load in CF. *Thorax*, 51, 619–623.
Coultas, D. B. (1998) Passive smoking and risk of adult asthma and COPD. *Thorax*, 53, 381–387.
Couriel, J. M. (1994) Passive smoking and the health of children. *Thorax*, 49, 731–734.
Coursin, D. R. and Coursin, D. B. (1998) Survivors, beware of posttraumatic stress disorder. *Crit. Care Med.*, 26, 834–835.
Cousins, N. (1981) *Human Options*, WW Norton, New York, p. 210.
Cousins, M. J. (1989) Acute pain and the injury response. *Regional Anesthes.*, 14, 162–179.
Covey, L. S. (2000) Advances in non-nicotine pharmacotherapy for smoking cessation. *Drugs*, 59, 17–31.
Cowan, S. (1998) Bisphosphonates and glucocorticoid-induced osteoporosis: implications for patients with respiratory diseases. *Thorax*, 53, 331–332.
Cowley, D. S. (1987) Hyperventilation and panic disorder. *Am. J. Med.*, 83, 929–937.
Cowley, H. C. (1993) Management of liver disease on ICU. *Care Crit. Ill*, 9, 122–127.
Cowling, M. G. (2000) Stenting in the oesophagus. *Hosp. Med.*, 61, 33–36.
Cox, C. and Hayes, J. (1999) Physiologic and psychodynamic responses to the administration of therapeutic touch in critical care. *Int. Crit. Care Nurse*, 15, 363–368.
Craig, D. B. (1981) Postoperative recovery of pulmonary function. *Anesth. Analg.*, 60, 46–52.
Crane, L. (1994) The effect of exercise training on pulmonary function in persons with quadriplegia. *Paraplegia*, 32, 435–441.
Craven, D. E., Lichtenberg, D. A. and Goularte, T. A. (1984) Contaminated medication nebulizers in mechanical ventilator circuits. *Am. J. Med.*, 77, 834–838.
Creasey, G. (1996) Electrical stimulation to restore ventilation. *J. Rehabil. Res. Develop.*, 33, 123–132.
Cregan, P. (1999) Surgery in the information age. *Med. J. Austr.*, 171, 514–516.
Criner, G. J. (2000) Effects of long-term oxygen therapy on mortality and morbidity. *Respir. Care*, 45, 105–118.
Criner, G. J. and Isaac, L. (1995) Psychological issues in the ventilator-dependent patient. *Respir. Care*, 40, 855–865.
Crockett, A. W. B. (1997) Symptom-free asthma. *Update*, 55, 463–468.
Crombie, I. K. (1998) Cut and thrust: antecedent surgery and trauma among patients attending a chronic pain clinic. *Pain*, 76, 167–171.
Crosby, L. J. and Parsons, L. C. (1992) Cerebrovascular response of closed head-injured patients to a standardized endotracheal suctioning and manual hyperventilation procedure. *J. Neurosci. Nurs.*, 24, 40–49.
Crowe, J., MacKay, M. and Morris, H. (2000) A multi-centre, randomized controlled trial of the effectiveness of positioning on quadriplegic shoulder pain. *Physiother. Can.*, 42, 266–272.
Cruickshank, J. P. and Lumley, K. (1999) Immediate management of acute severe asthma in adults. *Br. J. Nurs.*, 8, 103–108.
Cruickshanks, K. J. (1998) Cigarette smoking and hearing loss. *J. A. M. A.*, 279, 1715–1719.

Cruz, J. (1995) Cerebral blood flow, vascular resistance and oxygen metabolism in acute brain trauma. *Crit. Care Med.*, 23, 1412–1417.

Cryer, H. G., Leong, K. and McArthur, D. L. (1999) Multiple organ failure: by the time you predict it, it's already there. *J. Trauma*, 46, 597–606.

CSP (1995) *Rules of Professional Conduct*, Chartered Society of Physiotherapy, London.

CSP (1998) *Standards for Newly-qualified Physiotherapists*. Chartered Society of Physiotherapy, London.

Cunningham, S. (1995) Transcutaneous oxygen levels in retinopathy of prematurity. *Lancet*, 346, 1464–1465.

Cupples, S. A. (1991) Effects of timing and reinforcement of pre-operative education for patients having coronary artery bypass graft surgery. *Heart Lung*, 20, 654–660.

Currie, A. E. and Gallagher, P. J. (1988) The pathology of clubbing. *Br. J. Dis. Chest*, 82, 382–385.

Currie, D. C., Gaskell, M. D. and Cole, P. J. (1986) Practice, problems and compliance with postural drainage. *Br. J. Dis. Chest*, 80, 249–253.

Curtis, J. R. (1994) Emergent assessment and management of acute respiratory failure in COPD. *Clin. Chest Med.*, 15, 481–500.

Curtis, K. A., Drysdale, G. A. and Lanza, R. D. (1999) Shoulder pain in wheelchair users with tetraplegia and paraplegia. *Arch. Phys. Med. Rehabil.*, 80, 453–457.

Czarnik, R. E., Stone, K. S. and Everhart, C. C. (1991) Differential effects of continuous versus intermittent suction on tracheal tissue. *Heart Lung*, 20, 144–151.

D'Alonzo, G. E. (1995) Measurements of morning and evening airflow. *Am. J. Respir. Crit. Care Med.*, 152, 1097–1099.

D'Alonzo, G. E., Steinijans, V. W. and Keller, A. (1995) Measurements of morning and evening airflow grossly underestimate the circadian variability of FEV_1 and peak expiratory flow in asthma. *Am. J. Respir. Crit. Care Med.*, 152, 1097–1099.

D'Amours, R. H. (1996) Postoperative pain management. *J. Orthop. Sports PT*, 24, 227–234.

Dalley, J. (1999) Evaluation of clinical practice. *Physiotherapy*, 85, 491–497.

Dallimore, K., Jenkinsm S. and Tucker, B. (1998) Respiratory and cardiovascular responses to manual chest percussion in normal subjects. *Austr. Physiother. J.*, 44, 267–274.

Dantzker, D. R. (1983). The influence of cardiovascular function on gas exchange. *Clin. Chest Med.*, 4, 149–159.

Dar, K. (1995) Arterial versus capillary blood sampling. *Br. Med. J.*, 309, 24–25.

Dargie, H. J. (1994) Diagnosis and management of heart failure. *Br. Med. J.*, 308, 321–328.

Darin, J. (1982) The need for rational criteria for the use of unheated bubble humidifiers. *Respir. Care*, 27, 945–947.

Dark, J. H. (1997) Lung: living related transplantation. *Br. Med. Bull.*, 53, 892–903.

David, A. (1991) Positive expiratory pressure, in *Respiratory Care*, (ed. J. A. Pryor), Churchill Livingstone, Edinburgh, pp. 51–63.

Davies, K. (1994) Prevention of pressure sores. *Br. J. Nurs.*, 3, 1099–1104.

Davies, C. W. H. (1999) Intrapleural streptokinase in the management of malignant multiloculated pleural effusions. *Chest*, 115, 729–733.

Davies, R. J. O. and Hopkin, J. M. (1989) Nasal oxygen in exacerbations of ventilatory failure. *Br. Med. J.*, 299, 43–44.

Davies, H., Helms, P. and Gordon, I. (1990) The effect of posture on regional ventilation in children and adults. *Thorax*, 45, 313–314.

Davies, L., Angus, R. M. and Calverley, P. M. A. (1999) Oral corticosteroids in patients admitted to hospital with exacerbations of COPD. *Lancet*, 354, 456–460.

Davis, P. B., Drumm, M. and Konstan, M. W. (1996) Cystic fibrosis. *Am. J. Respir. Crit. Care Med.*, 154, 1229–1256.

Davis, G. and Glaser, R. (1990) Cardiorespiratory fitness following spinal cord injury. In: *Key Issues in Neurological Physiotherapy* (Eds. Ada, L., Canning, C.). Butterworth-Heinemann, London.

Daviskas, E. (1997) Inhalation of dry-powder mannitol increases mucociliary clearance. *Eur. Respir. J.*, 10, 2449–2454.

Daviskas, E. (1999) Inhalation of dry powder mannitol improves clearance of mucus in patients with bronchiectais. *Am. J. Respir. Crit. Care Med.*, 159, 1843–1848.

Davy, K. P. and Seals, D. R. (1994) Total blood volume in healthy young and older men. *J. Appl. Physiol.*, 76, 2059–2062.

Dean, E. (1994) Oxygen transport: a physiologically-based conceptual framework for the practice of cardiopulmonary physiotherapy. *Physiotherapy*, 80, 347–354.

Dean, E. (1997) Oxygen transport deficits in systemic

disease and implications for physical therapy. *Phys. Ther.*, 77, 187–201.
Dean, E. and Ross, J. (1992) Mobilization and exercise conditioning, in *Pulmonary Management in physical therapy*, (ed. C. C. Zadai), Churchill Livingstone, Edinburgh.
Dean, N. C., Brown, J. K. and Himelman, R. B. (1992) Oxygen may improve dyspnea and endurance in patients with COPD. *Am. Rev. Respir. Dis.*, **146**, 941–945.
Deaves, D. M. (1993) An assessment of the value of asthma education in the prevention of childhood asthma. *J. Adv. Nurs.*ing, **18**, 354–363.
DeBusk, R. F. (1990) Training effects of long versus short bouts of exercise in healthy subjects. *Am. J. Cardiol.*, **65**, 1010–1013.
Dechman, C., Sato, J. and Bates, J. H. T. (1993) Effect of pleural effusion on respiratory mechanics and the influence of deep inflation, in dogs. *Eur. Respir. J.*, 6, 219–224.
Decramer, M. (1994) Corticosteroids contribute to muscle weakness in CAO. *Am. J. Respir. Crit. Care Med.*, **150**, 11–16.
Decramer, M. (1997) Hyperinflation and respiratory muscle interaction. *Eur. Respir. J.*, **10**, 934–941.
DeGuire, S., Gevirtz, R. and Kawahara, Y. (1992) Hyperventilation syndrome and the assessment of treatment for functional cardiac symptoms. *Am. J. Cardiol.*, 70, 673–677.
Deitch, E. A. (1999) Prevention of multiple organ failure. *Surg. Clin. North Am.*, 79, 1471–1488.
Dekker, F. W., Schrier, A. C. and Sterk, P. J. (1992) Validity of peak expiratory flow measurement in assessing reversibility of airflow obstruction. *Thorax*, 47, 162–166.
DeLateur, B. J. (1997) Quality of Life: a patient-centred outcome. *Arch. Phys. Med. Rehabil.*, 78, 237–299.
Delaunay, L. (1995) Laparoscopic cholecystectomy minimally impairs postoperative cardiorespiratory and muscle performance. *Br. J. Surg.*, 82, 373–376.
Delk, K. K., Gevirtz, R. and Hicks, D. A. (1993) The effects of biofeedback retraining on lung function in patients with CF. *Chest*, **105**, 23–28.
DeLucas, P. (1996) Exercise test in the early diagnosis of interstitial pulmonary diseases. *Eur. Resp.J.*, 9(23), 1415.
DeMeo, M. T. (1992) Nutrition in acute pulmonary disease. *Nutr. Rev.*, 50, 320–328.
Demeter, S. L. (1986) Hyperventilation syndrome and asthma. *Am. J. Med.*, 81,89–94.
Deming, D. D. (1995) Respiratory assessment of neonatal and pediatric patients, in *Clinical Assessment in Respiratory Care*, (eds R. L. Wilkins, R. L. Sheldon and S. J. Krider), Mosby, Toronto.
Demling, R. (1990) Pulmonary dysfunction after cerebral injury. *Crit. Care Med.*, **18**, 768–774.
Denehy, L. (1999) The use of manual hyperinflation in airways clearance. *Eur. Respir. J.*, **14**, 958–965.
Denholm, S. W. and Goodwin, H. A. (1995) Aspiration of foreign bodies. *Hosp. Update*, **21**, 562–564.
Denison, D. M. (1996) Physiological principles, in *Occupational Lung Disease*, (ed. W. Raymond Parkes), 2nd edn, Butterworth-Heinemann, London.
Dennison, A., Oakley, N. and Appleton, D. (1996) Local anaesthesia for major general surgical procedures. *Postgrad. Med. J.*, 72, 105–108.
DeRose, J. J., Argenziano, M. and El-Amir, N. (1998) Lung reduction operation and resection of pulmonary nodules. *Ann. Thorac. Surg.*, 65, 314–318.
Deschamps, C., Pairolero, P. C. and Allen, M. S. (1999) Early complications. *Chest Surg. Clin. North Am.*, 9, 587–595.
Deslauriers, J. (1999) Techniques of pneumonectomy. *Chest Surg. Clin. North Am.*, 9, 437–448.
DeVito, A. J. (1990) Dyspnea during hospitalization for acute phase of illness as recalled by patients with COPD. *Heart Lung*, **19**, 186–191.
Dewar, A. L. (1995) Unbearable incidents: failure to endure the experience of illness. *J. Adv. Nurs.*, 22, 957–964.
Dexter, F. and Hindman, B. J. (1997) Recommendations for hyperbaric oxygen therapy of cerebral air embolism. *Anesth. Analg.*, 84, 1203–1207.
Dhand, R. and Fink, J. (1999) Dry powder inhalers. *Respir. Care*, 44, 940–951.
Dickey, J. L. (1989) Postoperative osteopathic manipulation of median sternotomy patients. *J. Am. Osteopath. Assoc.*, 89, 1309–1322.
Dicpinigaitis, P. V. (1998) Treatment of chronic refractory cough with baclofen. *Respiration*, 65, 86–88.
Didour, M. (1997) Effects of lung cycle ergometer training in elderly patients with bronchial asthma. *Eur. Respir. J.*, 10, 458S.
Diflorio, T. (1999) The effect of midazolam on persistent postoperative nausea and vomiting. *Anaesth. Intensive Care*, 27, 38–40.
Dilworth, J. P. and White, R. J. (1992) Postoperative

chest infection after upper abdominal surgery. *Respir. Med.*, 86, 205–210.
Dimitriou, G. (1996) A comparison of supine and prone positioning in oxygen dependent and convalescent premature infants. *Br. J. Intensive Care*, 6, 254–259.
Dimond, B. C. (1999) *Legal Aspects of Physiotherapy*, Blackwell, Oxford.
Dimopoulou, I. (1999) Respiratory function in patients with thalassaemia and iron overload. *Eur. Respir. J.*, 13, 602–659.
Dinwiddie, R. (2000) Pathogenesis of lung disease in CF. *Respiration*, 67, 3–8.
Dixhoorn, J. van (1986) Behavioral characteristics predisposing to hyperventilation complaints. *Gedrag Gezondheid.*, 14, 169–174.
Dixhoorn, J. van, Duivenvoordent, H. J. and Staal, J. A. (1987) Cardiac events after mycardial infarction: possible effect of relaxation therapy. *Eur. Heart J.*, 8, 1210–1214.
Dobb, G. J. and Coombs, L. J. (1987) Clinical examination of patients in the ICU. *Br. J. Hosp. Med.*, 38, 102–8.
Dobbs, P. and Coad, N. (1999) The pathophysiology and ICU treatment of patients following use of ecstasy. *Br. J. Intensive Care*, 9, 9–13.
Dock, W. (1944) The evil sequelae of complete bed rest. *J. A. M. A.*, 125, 1083–1085.
Dodd, F. M., Simon, E. and McKeown, D. (1995) The effect of a cervical collar on the tidal volume of anaesthetised adult patients. *Anaesthesia*, 50, 961–963.
Dodd, M. E. (1991) Exercise in cystic fibrosis adults, in *Respiratory Care*, (ed. J. A. Pryor), Churchill Livingstone, Edinburgh, pp. 27–50.
Dodd, M. E. (1996) Nebuliser therapy: what nurses and patients need to know. *Nurs. Stand.*, 10(31), 39–42.
Dodd, M. E., Howarth, C. S. and Webb, A. K. (1998) A practical approach to oxygen therapy in cystic fibrosis. *J. Roy. Soc. Med.*, 91(suppl. 34), 30–39.
Dodge, J. (1999) Gastro-oesophageal reflux in infants. *Acta Paediatr.*, 88, 359–370.
Dodson, L. A., Nathan, N. S. and D'Ambra, M. N. (1997) New concepts in right heart failure. *Curr. Opin. Anaesth.*, 10, 21–28.
DoH (1993) *The Health of the Nation*, Department of Health, London.
DoH (1995) *Health of the Nation: Nutritional Guidelines for Hospital Catering*, Department of Health, London.
DoH (1996) *Guidelines of Admission to and Discharge from Intensive Care and High Dependency Units.* Department of Health, London.
DoH (1999) *Clinical Governance.* NHS Executive, Leeds.
Donahoe, M. and Rogers, R. M. (1990) Nutritional assessment and support in COPD. *Clin. Chest Med.*, 11, 487–504.
Donahoe, M., Rogers, R. M. and Cottrell, J. J. (1992) Is loss of body weight in COPD patients with emphysema secondary to low tissue oxygenation? *Respiration*, 24 (suppl. 2), 33–39.
Donald, I. (1977) Pain – a patient's view, in *Pain: New Perspectives in Measurement and Management*, (ed. A. W. Harcus), Churchill Livingstone, Edinburgh, p. 1.
Donald, K. J., Robertson, V. J. and Tsebelis, K. (2000) Setting safe and effective suction pressure. *Intensive Care Med.*, 26, 15–19.
Donaldson, G. C. (1999) Effect of temperature on lung function and symptoms in COPD. *Eur. Respir. J.*, 13, 844–849.
Donner, C. F. and Carone, M. (1998) Determining quality of life in COPD patients. *RT Int.*, 7, 37–43.
Donner, C. F. and Howard, P. (1992) Pulmonary rehabilitation in COPD with recommendations for its use. *Eur. Respir. J.*, 5, 266–275.
Dorinsky, P. M., Reisner, C. and Ferguson, G. T. (1999) The combination of ipratropium and albuterol optimizes pulmonary function testing in COPD. *Chest*, 115, 966–971.
Douglas, N. (1993) Nocturnal asthma. *Respir. Dis. Pract.*, 10, 7–9.
Douglas, W. E. and Flabouris, A. (1999) Surgical emphysema following percutaneous tracheostomy. *Anaesth. Intensive Care*, 27, 69–72.
Doull, I. J. M. (1997) Tracheobronchomalacia in preterm infants with chronic lung disease. *Arch. Dis. Child.*, 76, F203–F205.
Dow, L. (1996) Does dietary intake of vitamins C and E influence lung function in older people? *Am. J. Respir. Crit. Care Med.*, 154, 1401–1404.
Downs, J. A. (1989) Endotracheal suction: a method of tracheal washout. *Physiotherapy*, 75, 454.
Downs, J. A., Edwards, A. D. and McCormick, D. C. (1991) Effect of intervention on development of hip posture in very preterm babies. *Arch. Dis. Child.*, 66, 797–801.
Dresler, C. M. (1996) Smoking cessation and lung cancer resection. *Chest*, 110, 1199–1202.
Dreyfuss, D. (1995) Mechanical ventilation with

heated humidifers or heat moisture exchangers. *Am. J. Respir. Crit. Care Med.*, 151, 986–992.
Drugs and Therapeutics Bulletin (1997) Long-acting β_2-stimulants in asthma. *Drugs Ther. Bull.*, 35, 1–4.
Du, H. L., Yamada, Y. and Suzuki, R. O. S. (1997) Beneficial effects of the prone position on the incidence of barotrauma in oleic acid-induced lung injury. *Acta Anaesthesiol. Scand.*, 41, 701–707.
Duckworth, M. (1999) Outcome measurement selection and typology. *Physiotherapy*, 85, 21–27.
Dudgeon, D. J. and Lertzman, M. (1998) Dyspnea in the advanced cancer patient. *J. Pain Symptom Manage.*, 16, 212–219.
Dudley, D. L., Glover, E. M., and Jorgenson, B. N. (1980) Psychosocial concomitants to rehabilitation in COPD. *Chest*, 77, 544–551.
Dulfano, M. J. (1973) Physical properties of sputum. *Am. Rev. Respir. Dis.*, **107**, 130–133.
Duncan, S. R. (1987) Hyperventilation syndrome. *Hosp. Med.*, 23, 86–98.
Dunlevy, C. D. and Tyl, S. E. (1992) The effect of oral versus nasal breathing on oxygen concentrations from nasal cannulas. *Respir. Care*, 37, 357–360.
Dunn, R. B., Walter, J. S. and Walsh, J. (1995) Diaphragm and accessory respiratory muscle stimulation using intramuscular electrodes. *Arch. Phys. Med. Rehabil.*, 76, 266–271.
Durand, M., Sangha, B. and Cabal, L. A. (1989) Cardiopulmonary and intracranial pressure changes related to endotracheal suctioning in preterm infants. *Crit. Care Med.*, 17, 506–510.
Duranti, R. (1995) Mechanical loading and control of breathing in patients with severe COPD. *Thorax*, 50, 127–133.
Durbin, C. G. (1993) Oxygen toxicity in the critically ill patient. *Respir. Care*, 38, 739–753.
Durward, Q. J., Amacher, A. L. and Maestro, R. F. (1983) Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension. *J. Neurosurg.*, 59, 938–44.
Dweik, R. A. and Stoller, J. K. (1999) Role of bronchoscopy in massive hemotpysis. *Clin. Chest Med.*, 20, 89–105.
Dyer, I. (1995) Preventing the ITU syndrome. *Intensive Crit. Care Nurs.*, 11, 130–139.
Eakin, E. G., Resnikoff, P. M., Prewitt, L. M. and Ries, A. L. (1998) Validation of a new dyspnea measure. *Chest*, 113, 619–624.
Eaton, J. M. (1984) Adult manual resuscitators. *Br. J. Hosp. Med.*, 31, 67–703.
Eaton, D., Bannister, P. and Mulley, G. P. (1994) Axillary sweating in clinical assessment of dehydration in ill elderly patients. *Br. Med. J.*, 308, 1271.
Edington, J., Kon, P. and Martyn, C. N. (1997) Prevalence of malnutrition after major surgery. *J. Hum. Nutr. Diet.* 10, 111–116.
Edmondson, R. (1994) The causes and management of pulmonary embolism. *Care Crit. Ill*, 10, 26–9.
Edwards, J. D. (1997) Oxygen transport in the critically ill. *Br. J. Intensive Care*, 7, 23–29.
Efthimiou, J. and Barnes, P. F. (1998) Effect of inhaled corticosteroids on bones and growth. *Eur. Respir. J.*, 11, 1167–1177.
Egbert, L. D., Battit, G. E. and Welch, C. E. (1964) Reduction of postoperative pain by encouragement and instruction of patients. *N. Engl. J. Med.*, 270, 825–827.
Ehrlich, M., Manns, P. J. and Poulin, C. (1999) Respiratory training for a person with C3–C4 tetraplegia. *Austr. J. Physiother.*, 45, 301–307.
Eid, N. (1991) Chest physiotherapy in review. *Respir. Care*, 36, 270–282.
Eigen, H., Clark, N. M. and Wolle, J. M. (1987) Clinical-behavioral aspects of CF. *Am. Rev. Respir. Dis.*, 136, 1509–1513.
El-Ansary, D., Adams, R. and Ghandi, A. (2000) Musculoskeletal and neurological complications following CABG. *Austr. Physiother. J.*, 46, 19–25.
Elborn, J. S. (1996) Nutrition and survival in CF. *Thorax*, 51, 971–972.
Elborn, J. S., Shale, D. J., Britton, J. R. (1991) Cystic fibrosis: current survival and population estimates to the year 2000. *Thorax*, 46, 881–885.
Elborn, J. S., Johnston, B. and Allen, F. (1992) Inhaled steroids in patients with bronchiectasis. *Respir. Med.*, 86, 121–124.
Elborn, J. S., Prescott, R. J. and Stack, B. H. R. (2000) Elective versus symptomatic antibiotic treatment in cystic fibrosis patients. *Thorax*, 55, 355–358.
Elia, M. (1995) Changing concepts of nutrient requirements in disease. *Lancet*, 345, 1279–1282.
Elliott, M. W. (1995) Nocturnal assisted ventilation using BiPAP. *Eur. Respir. J.*, 8, 436–440.
Elliott, M. W., Aquilina, R. and Green, M. (1994) A comparison of different modes of noninvasive ventilatory support. *Anaesthesia*, 49, 279–283.
Ellis, N. (1994) *Acupuncture in Clinical Practice*, Chapman & Hall, London.

Ellis, B. (1995) Cardiac transplantation. *Physiotherapy*, **81**, 157–162.

Ellis, E. R., Redfern, J. and Rusterholtz, B. (1999) The effects of feedback on the performance of manual hyperinflation by physiotherapists. *WCPT Proceedings*, p. 31.

Elpern, E. H., Scott, M. G. and Petro, L. (1994) Pulmonary aspiration in mechanically ventilated patients with tracheostomies. *Chest*, **105**, 563–566.

Ely, W. E., Johnson, M. M. and Chiles, C. (1996) Chest X-ray changes in air space disease are associated with parameters of mechanical ventilation. *Am. J. Respir. Crit. Care Med.*, **154**, 1543–1550.

Emergency Care Research Institute (1977) Suction catheters. *Health Devices*, **6**, 132–141.

Empey, D. (1992) International consensus on diagnosis and treatment of asthma. *Respir. Dis. Pract.*, **9**, 13–20.

Empey, D. (1993) Tuberculosis – a killer returns. *Respir. Dis. Pract.*, **10**, 4–5.

Emtner, M. (1998) A 3-year follow-up of asthmatic patients participating in a 10-week rehabilitation program. *Arch. Phys. Med. Rehabil.*, **79**, 539–544.

Emtner, M., Herala, M. and Stålenheim, G. (1996) High-intensity physical training in adults with asthma. *Chest*, **109**, 323–330.

Emtner, M., Hedin, A., Stålenheim G (1998) Asthmatic patients' views of a comprehensive asthma rehabilitation programme. *Physiother. Res. Int.*, **3**, 175–193.

Enderby, P. (1995) The effect of dairy products on the viscosity of saliva. *Clin. Rehabil.*, **9**, 61–64.

Eng, P. (1998) Flexible fiberoptic bronchoscopy. *Respir. Ther. Int.*, **7**, 33–35.

Eng, P. A., Morton, J. and Douglass, J. A. (1996) Short-term efficacy of ultrasonically nebulized hypertonic saline in CF. *Pediatr. Pulmonol.*, **21**, 77–83.

Engdahl, O. (1993) Chest radiograph – a poor method for determining the size of a pneumothorax. *Chest*, **103**, 26–29.

Engström, C. P., Persson, L. O., Larsson, S. *et al.* (1996) Functional status and well-being in COPD. *Thorax*, **51**, 825–830.

Enqvist, B. (1997) Preoperative hypnosis reduces postoperative vomiting after surgery of the breasts. *Acta Anaesth. Scand.*, **41**, 1028–1032.

Enright, S. (1992) Cardiorespiratory effects of chest physiotherapy. *Intensive Care Britain*, Greycoat, Harley St, London, 118–123.

Enzer, N. B. and Walker, P. A. (1967) Hyperventilation syndrome in childhood. *J. Pediatr.*, **70**, 521–532.

Epstein, C. D. and Henning, R. J. (1993) Oxygen transport variables in the identification and treatment of tissue hypoxia. *Heart Lung*, **22**, 328–348.

Erb, R. and Frei, F. J. (1996) Sedation in the paediatric ICU. *Curr. Opin. Anaesth.*, **9**, 239–243.

Eriksson, L. (1996) Pulmonary physiology relevant to chest physiotherapy. *Israel J. Med.*, **32**, S110–S114.

Ernst, E. (1995) What is wrong with anecdotal evidence? *Eur. J. Phys. Med. Rehabil.*, **5**, 145–146.

Ernst, E. (1998) Complementary therapies for asthma. *J. Asthma*, **35**, 667–671.

Ersson, U., Carlson, H. and Mellström, A. (1990) Observations on intracranial dynamics during respiratory physiotherapy in unconscious neurosurgical patients. *Acta Anaesthesiol. Scand.*, **34**, 99–103.

Esteban, A. (1995) A comparison of four methods of weaning. *N. Engl. J. Med.*, **332**, 345–348.

Everard, M. L., Clark, A. R. and Milner, A. D. (1992) Drug delivery from holding chambers with attached facemask. *Arch. Dis. Child.*, **67**, 580–585.

Everard, M. L., Evans, M. and Milner, A. D. (1994) Is tapping jet nebulizers worthwhile? *Arch. Dis. Child.*, **70**, 538–539.

Everly, G. S. (1989). *A Clinical Guide to the Treatment of the Human Stress Response*, Plenum Press, New York.

Ewer, A. K., James, M. E. and Tobin, J. M. (1999) Prone and left lateral positioning reduce gastro-oesophageal reflux in preterm infants. *Arch. Dis. Child. Fetal Neonat. Ed.*, **81**, F201–F205.

Fabregas, N. (1996) Diagnosis of ventilator-associated pneumonia. *Curr. Opin. Anaesthesiol.*, **9**, 157–161.

Fahrenfort, M. (1987) Patient emancipation by health education. *Patient Education Council, USA*, **10**, 25.

Faling, L. J. (1986) Pulmonary rehabilitation – physical modalities. *Clin. Chest Med.*, **7**, 599–618.

Falk, S. (1997) Lung cancer. *Update*, **55**, 706–712.

Fallowfield, L. (1993) Giving sad and bad news. *Lancet*, **341**, 476–478.

Fan, C.-F., Tanhui, E. and Joshi, S. (1997) Acupressure treatment for prevention of postoperative nausea and vomiting. *Anesth. Analg.*, **84**, 821–825.

Fanta, C. H. (1992) Emergency management of acute severe asthma. *Respir. Care*, 37, 551–563.
Fareed, A. (1996) The experience of reassurance. *J. Adv. Nurs.*, 23, 272–279.
Fawcett, D. D. (1995) Inhaled anti-inflammatory agents in childhood asthma. *Respir. Care*, 40, 108–113.
Faymonville, M. E. (1997) Psychological approaches during conscious sedation. *Pain*, 73, 361–367.
Fehrenbach, C. (1998) Chronic obstructive pulmonary disease. *Prof. Nurse*, 13, 771–777.
Feldman, Z. (1992) Effect of head elevation on head-injured patients. *J. Neurosurg.*, 76, 207–211.
Feldman, C., Kassel, M. and Cantrell, J. (1999) The presence and sequence of endotracheal tube colonization in patients undergoing mechanical ventilation. *Eur. Resp. J.*, 13, 546–551.
Felhendler, D. (1996) Pressure on acupoints decreases postoperative pain. *Clin. J. Pain*, 12, 326–329.
Feneck, R. O. (1996) Cardiac surgery and intensive care. *Br. J. Intensive Care*, 6, 155–161.
Feng, W., Deng, W. W. and Huang, S. G. (1998) Short-term efficacy of RC-Cornet in improving pulmonary function and decreasing cohesiveness of sputum in bronchiectasis patients. *Chest*, 114, 320.
Ferdinand, B. (1998) Postoperative pneumonia. *Chest Surg. Clin. North Am.*, 8, 529–541.
Ferguson, J. A. (1992) Pain following coronary artery bypass grafting. *Intens. Crit. Care Nurs.*, 8, 153–162.
Ferguson, M. K. (1999) Preoperative assessment of postoperative risk, *Chest*, 115, 58S–63S.
Ferguson, A. D., Prescott, R. J. and Selkon, J. B. (1996) The clinical course and management of thoracic empyema. *Q. J. Med.*, 89, 285–289.
Ferrer, M. (1997) Chronic obstructive pulmonary disease stage and health-related quality of life. *Ann. Intern. Med.*, 127, 1072–1079.
Fessler, H. E. (1995) CPAP reduces inspiratory work more than dyspnea during hyperinflation. *Chest*, 108, 432–440.
Fiddler, H. and Williams, N. (2000) ECMO: a physiotherapy perspective. *Physiotherapy*, 86, 203–208.
Fiel, S. B. (1993) Clinical management of pulmonary disease in CF. *Lancet*, 341, 1070–1074.
Field, S. K. (1998) Does medical antireflux therapy improve asthma in asthmatics with GOR? *Chest*, 114, 275–283.
Fillmore, C. M. (1999) Nutrition and dietary supplements. *Phys. Med. Rehab. Clin. N. Am.*, 10, 673–697.
Fink, J. and Dhand, R. (1999) Bronchodilator resuscitation in the emergency department. *Respir. Care*, 44, 1353–1374.
Fink, M. P., Helsmoortel, C. M. and Stein, K. L. (1990) The efficacy of an oscillating bed in the prevention of lower respiratory tract infection in critically ill victims of blunt trauma. *Chest*, 97, 132–137.
Fink, J. B., Krause, S. A. and Barrett, L. (1998) Extending ventilator circuit change interval beyond 2 days. *Chest*, 113, 405–411.
Finlayson, J. M. (1997) The role of exercise in rehabilitation after uncomplicated myocardial infarction. *Physiotherapy*, 83, 517–523.
Fiorentini, A. (1992) Potential hazards of tracheobronchial suctioning. *Intens. Crit. Care Nurs.*, 8, 217–226.
Fisher, M. D. (1997) Pediatric traumatic brain injury. *Crit. Care Nurse*, 20, 36–51.
Fisher, E. B., Haire-Joshu, K. and Morgan, G. D. (1990) Smoking and smoking cessation. *Am. Rev. Respir. Dis.*, 142, 702–720.
Fitting, J.-W. (1992) Nutritional support in chronic obstructive lung disease. *Thorax*, 47, 141–143.
Fitzgerald, J. M. (1988) Use of oxygen therapy for adult patients outside the critical care areas of a university hospital. *Lancet*, i, 981–983.
Fixley, M. S. (1978) Flow dependence of gas distribution and the pattern of inspiratory muscle contraction. *J. Appl. Physiol.*, 45, 733–741.
Flanc, C. (1969) Postoperative DVT, effect of intensive prophylaxis. Lancet, i, 477–478.
Flaxman, J. (1978) Quitting smoking now or later. *Behav. Ther.* 9, 240–270.
Fletcher, E. C. (1992) A double-blind trial of nocturnal supplemental oxygen for sleep desaturation in patients with COPD. *Am. Rev. Respir. Dis.*, 145, 1070–1076.
Flood, P. T. and Partridge, M. R. (1996) Asthma: a changing perspective on management. *Curr. Anaesth. Crit. Care*, 7, 260–265.
Flynn, M. B. (1999) Identifying and treating inhalation injuries in fire victims. *Dimens. Crit. Care Nurs.*, 18, 18–23.
Foglio, K., Bianchi, L. and Bruletti, G. (1999) Long-term effectiveness of pulmonary rehabilitation in patients with CAO. *Eur. Respir. J.*, 13, 125–132.
Foley, M. P., Prax, B. and Crowell, R. (1996) Cardiorespiratory demands in older adults. *Phys. Ther.*, 76, 1313–1319.

Fong, S. L. C. (1994) Compliance among adults with CF. *Ann. Pharmacother.*, 24, 689–691.
Fonsmark, L., Rasmussen, Y. H. and Carl, P. (1999) Occurrence of withdrawal in critically ill sedated children. *Crit. Care Med.*, 27, 196–199.
Forastiere, F., Pistelli, R. and Sestini, P. (2000) Consumption of fresh fruit rich in vitamin C and wheezing symptoms in children. *Thorax*, 55, 283–288.
Forbes, C. (1994) Management of deep vein thrombosis. *Practitioner*, 238, 168–175.
Fourrier, F. (1992) Septic shock, multiple organ failure and DIC. *Chest*, 101, 816–823.
Francis, K. (1996) Physical activity in prevention and rehabilitation of breast cancer. *Crit. Rev. Phys. Rehabil. Med.*, 8, 323–341.
Franck, L. S. (1998) The use of intravenous opioids to provide analgesia in critically ill premature neonates. *J. Pain Symptom Manage.*, 15, 41–69.
Frank, C., Akeson, W. H., Woo, S. L. Y. and Amiel, D. (1994) Physiology and therapeutic value of passive joint motion. *Clin. Orthoped. Related Res.*, 185, 113–124.
Fraser, J. and Kerr, J. R. (1993) Psychophysiological effects of back massage on elderly institutionalized patients. *J. Adv. Nurs.*, 18, 238–245.
Frazer, C. A. (1999) Effect of transcutaneous electrical nerve stimulation on emesis associated with PCA. *WCPT Proceedings*, p. 61.
Freedberg, P. D., Hoffman, L. A. and Light, W. C. (1987) Effect of progressive muscle relaxation on asthma. *Heart Lung*, 16, 24–30.
Freedman, S. (1987) Breathlessness and respiratory mechanics during reflex or voluntary hyperventilation in patients with CAL. *Clin. Sci.*, 73, 311–318.
Freeman, J. A. and Armstrong, I. R. (1994) Pulmonary function tests before and after laparoscopic cholecystectomy. *Anaesthesia*, 49, 579–582.
Freichels, T. A. (1998) Managing mechanical ventilation in status asthmaticus. *Dimens. Crit. Care Nurs.*, 17, 2–7.
Fried, R. (1993) *The Psychology and Physiology of Breathing*, Plenum Press, New York.
Froese, A. B. and Bryan, C. (1974) Effects of anesthesia and paralysis on diaphragmatic mechanics in man. *Anesthesiology*, 41, 242–255.
Frost, E. A. M. (1996) Management of post-operative complications. *Curr. Opin. Anaesth.*, 9, 481–487.
Frownfelter, D. L. and Dean, E. (1996) *Principles and Practice of Cardiopulmonary Physical Therapy*, Mosby, Chicago, IL.
Fuhrman, B. P., Hernan, L. J. and Steinhorn, D. M. (1998) Partial liquid ventilation and the challenges of randomized controlled trials. *Respir. Care*, 43, 1086–1091.
Fuller, D., Sullivan, J. and Fregosi, R. F. (1996) Expiratory muscle endurance performance after exhaustive submaximal exercise. *J. Appl. Physiol.*, 80, 1495–1502.
Fulmer, J. D. and Snider, G. L. (1984) ACCP-NHLBI National conference on oxygen therapy. *Chest*, 86, 234–247.
Fulton, Y. (1996) Children's rights and the role of the nurse. *Paed. Nurs.*, 8, 29–31.
Gale, G. and VandenBerg, K. A. (1998) Kangaroo Care. *Neonat. Netw.*, 17, 69–71.
Gallagher, R. (1998) Nicotine withdrawal as an etiologic factor in delirium [letter]. *J. Pain Symptom Manage.*, 16, 26–27.
Gallagher, R. and Trenchard, J. (1986) *Movements of Life*, Redemptionist Publications, Alton.
Gallego, J. and Perruchet, P. (1991) Effect of practice on the voluntary control of a learned breathing pattern. *Physiol. Behav.*, 49, 315–319.
Galley, H. F. (2000) Can acute renal failure be prevented? *J. Roy. Coll. Surg. Edin.*, 45, 44–50.
Gandy, A. P. (1968) An attack of tetanus. *Lancet*, ii, 567–568.
Ganea, G. R. and Bogue, C. W. (1999) Pain management and weaning from narcotics and sedatives. *Curr. Opin. Pediatr.*, 11, 207–212.
Gardner, W. N. (1996) The pathophysiology of hyperventilation syndrome. *Chest*, 109, 516–530.
Garrod, R. (1998) The pros and cons of pulmonary rehabilitation at home. *Physiotherapy*, 84, 603–607.
Garrod, R., Bestall, J. and Garnham, R. (1996) Factors affecting attendance at an outpatient pulmonary rehabilitation programme. *Eur. Respir. J.*, 9(23), 382s.
Garrod, R., Bestall, J. C. and Paul, E. (1999) Evaluation of pulsed dose oxygen delivery during exercise in patients with severe COPD. *Thorax*, 54, 242–244.
Garssen, B. (1980) Role of stress in the development of the hyperventilation syndrome. *Psychother. Psychosom.*, 33, 214–225.
Garssen, B. (1983) Agoraphobia and the hyperventilation syndrome. *Behav. Res. Ther.*, 21, 643–649.

Gaultier, C. and Crapo, R. (1997) Effects of nutrition, growth hormone disturbances, training, altitude and sleep on lung volumes. *Eur. Respir. J.*, **10**, 2913–2919.

Gauwitz, D. F. (1995) How to protect the dysphagic stroke patient. *Am. J. Nurs.*, **95**, 34–38.

Gaynor, J. W. (1998) Update on lung transplantation in children. *Curr. Opin. Pediatr.*, **10**, 256–261.

Geller, M. (2000) Marked peripheral edema associated with montelukast and prednisone. *Ann. Int. Med.*, **132**, 924.

Gershman, N. H. (1996) Comparison of two methods of collecting induced sputum. *Eur. Respir. J.*, **9**, 2448–2453.

Gersin, K. (1994) The efficacy of sequential compression devices in multiple trauma patients with severe head injury. *J. Trauma*, **37**, 205–208.

Ghoneim, M. M. (2000) Awareness during anesthesia. *Anesthesiology*, **92**, 597–602.

Giacomo, T. D. (1999) Thoracoscopic resection of solitary lung metastases from colorectal cancer is a viable therapeutic option. *Chest*, **115**, 1141–1143.

Giannini, D. and Bacci, E. (1999) Inhaled beclomethasone dipropionate reverts tolerance to the protective effect of salmeterol. *Chest*, **115**, 629–634.

Gibson, G. J. (1995) Perception, personality and respiratory control in life-threatening asthma. *Thorax*, **50**(suppl. 1), S2–S4.

Gibson, G. J. (1996) Pulmonary hyperinflation. *Eur. Respir. J.*, **9**, 2640–2649.

Gidding, S. (1994) Active and passive tobacco exposure. *Pediatrics*, **94**, 750–751.

Gift, A. G. (1992) Relaxation to reduce dyspnea and anxiety in COPD patients. *Nurs. Res.*, **41**, 242–246.

Gilbert, A. (1996) Anxiety: a risk factor in open heart surgery. *ACPRC J.*, **29**, 60.

Gilbert, C. (1999) Breathing and the cardiovascular system. *J. Bodywork Mov. Ther.*, **3**, 215–224.

Gilbert, V. E. (1989) Detection of pneumonia by auscultation of the lungs in the lateral decubitus positions. *Am. Rev. Respir. Dis.*, **140**, 1012–1016.

Giles, D. R. (1995) Short-term effects of postural drainage with clapping vs autogenic drainage on oxygen saturation and sputum recovery in patients with CF. *Chest*, **108**, 952–954.

Gill, J. and Slater, J. (1991) Building barriers against infection. *Nurs. Times*, **87**(50), 53–54.

Gillespie, D. (1989) Let's listen to the children. *Respir. Dis. Pract.*, **6**, 23.

Gillespie, D. J. and Rehder, K. (1987) Body position and $\dot{V}_A/\dot{Q}$ relationships in unilateral pulmonary disease, *Chest*, **1**, 75–79.

Girard, J. P. and Terki, N. (1994) The Flutter as an adjunct to drug therapy in the management of bronchial asthma. *J. Invest. Allergy Clin. Immunol.*, **4**, 23–27.

Girault, C. (1997) Physiological effects and optimisation of nasal assist-control ventilation for patients with COPD. *Thorax*, **52**, 690–696.

Girault, C. (1999) Noninvasive ventilation as a systematic extubation and weaning technique in acute-on-chronic respiratory failure. *Am. J. Respir. Crit. Care Med.*, **160**, 86–92.

Girault, C., Muir, J.-F. and Mihaltan, F. (1996) Effects of repeated administration of zolpidem on sleep, diurnal and nocturnal respiratory function, vigilance and physical performance. *Chest*, **110**, 1203–1211.

Girodo, M., Ekstrand, K. A. and Metivier, G. J. (1992) Deep diaphragmatic breathing for the asthmatic patient. *Arch. Phys. Med. Rehabil.*, **73**, 717–720.

Giroux, J. M., Lewis, S. and Holland, L. G. (1987) Postoperative chest physiotherapy for abdominal hysterectomy patients. *Physiother. Can.*, **39**, 89–93.

Giuliano, K. K., Bloniasz, E. and Bell, J. (2000) Implementation of a pet visitation program in critical care. *Crit. Care Nurse*, **19**, 43–49.

Goldhill, D. R. and Sumner, A. (1998) Outcome of intensive care patients in a group of British ICUs. *Crit. Care Med.*, **26**, 1337–1345.

Golding, J. (1994) The consequences of smoking in pregnancy. Smoking in Pregnancy Conference, University of Bristol, Bristol, UK.

Goldman, J. M. (1986) Effect of abdominal binders on breathing in tetraplegic patients. *Thorax*, **41**, 940–945.

Goldman, J. M., Teale, C. and Muers, M. F. (1992) Simplifying the assessment of patients with chronic airflow limitation for home nebulizer therapy. *Respir. Med.*, **86**, 33–38.

Goldschlager, N., Epstein, A. E. and Naccarelli, G. (2000) Practical guidelines for clinicians who treat patients with amiodarone. *Arch. Int. Med.*, **160**, 1741–1748.

Goldstein, R. S. (1995) Home mechanical ventilation. *Chest*, **108**, 1581–1586.

Goldstein, R. (2000) Musculoskeletal conditions after spinal cord injury. *Phys. Med. Rehab. Clin. N. Am.*, **11**, 91–108.

Goldstone, J. and Moxham, J. (1991) Weaning from mechanical ventilation. *Thorax*, **46**, 56–62.

Golster, M. (1995) Blood volume and colloid osmotic pressure. *Crit. Care Med.*, **23**, A88.

Goodfellow, L. M. (1997) Application of pulse oximetry and the oxyhemoglobin dissociation curve. *Crit. Care Nurs. Q.*, **20**, 22–27.

Goodman, D. C. (1996) Theophylline in acute childhood asthma. *Pediatr. Pulmonol.*, **21**, 211–218.

Goodnough, S. K. (1985) The effects of oxygen and hyperinflation on arterial oxygen tension after suction. *Heart Lung*, **14**, 11–17.

Gopinath, S. P., Valadka, A. B. and Uzura, M. (1999) Comparison of jugular venous oxygen saturation and brain tissue PO_2 as monitors of cerebral ischemia after head injury. *Crit. Care Med.*, **27**, 2337–2345.

Gordon, S. (1994) What's in a name? *J. Emerg. Nurs.*, **20**, 170–171.

Gordon, K. and Child, A. (2000) Heart failure. *Nurs. Times*, **96**, 45–47.

Gorini, M. (1996) Breathing pattern and CO_2 retention in severe COPD. *Thorax*, **51**, 677–683.

Gosbee, J. (1998) Communication among health professionals. *Br. Med. J.*, **316**, 642.

Gosselink, R. (1996) Reliability of a commerciially available threshold loading device in healthy subjects and in patients with COPD. *Thorax*, **51**, 601–605.

Gosselink, R., Kovacs, L. and Decramer, M. (1999) Respiratory muscle involvement in multiple sclerosis. *Eur. Respir. J.*, **13**, 449–454.

Gosselink, R. A. A. M. (1995) Diaphragmatic breathing reduces efficiency of breathing in patients with COPD. *Am. J. Respir. Crit. Care Med.*, **151**, 1136–1142.

Gould, D. (1994) The significance of hand-drying in the prevention of infection. *Nurs. Times*, **90**, 33–335.

Gounden, P. (1993) The effect of posture on pectoralis major and latissimus dorsi in expiration in tetraplegia. *S. Afr. J. Physiother.*, **49**, 25–27.

Gourlay, G. K. and Boas, R. A. (1992) Fatal outcome with use of rectal morphine for postoperative pain control in an infant. *Br. Med. J.* **304**, 766–767.

Gozzoli, V. (2001) Is it worth treating fever in ICU patients? *Arch. Int. Med.*, **161**, 121–123.

Graff, T. D. and Benson, D. W. (1969) Systemic and pulmonary changes with inhaled humid atmospheres. *Anesthesiology*, **30**, 199–207.

Graham, A. N. J., Cosgrove, A. P. and Gibbons, J. R. P. (1992) Randomised clinical trial of chest drainage systems. *Thorax*, **47**, 461–462.

GRASSIC: Grampian Asthma Study of Integrated Care (1994) Integrated care for asthma. *Br. Med. J.*, **308**, 559–564.

Grasso, M. C., Button, B. M. and Allison, D. J. (2000) Benefits of music therapy as an adjunct to chest physiotherapy in infants and toddlers with CF. *Pediatr. Pulmonol.*, **29**, 371–381.

Gravil, J. H., Al-Rawas, O. A. and Cotton, M. M. (1998) Home treatment of exacerbations of COPD by an acute respiratory assessment service. *Lancet*, **351**, 1853–1855.

Gray, H. H. (1992) Management of pulmonary embolism. *Thorax*, **47**, 825–832.

Gray, E. (1999) Do respiratory patients receive adequate analgesia? *Nurs. Stand.*, **13**(47), 32–35.

Gray, J. F., MacIntyre, N. R. and Kronenberger, W. G. (1990) The effects of bolus normal saline instillation with endotracheal suctioning. *Respir. Care*, **35**, 785–790.

Gray, L., Watt, L. and Blass, E. M. (1999) Skin-to-skin contact is analgesic in healthy newborns. *Pediatrics*, **105**, e14.

Green, M. and Moxham, J. (1993) Respiratory muscles in health and disease, in *Respiratory Medicine: Recent Advances*, (ed. P. J. Barnes), Butterworth-Heineman, London.

Green, C. J., Campbell, I. T. and McClelland, P. (1995) Energy and nitrogen balance and changes in midupper-arm circumference with multiple organ failure. *Nutrition*, **2**, 739–746.

Greenfield, S. M. (1997) Drinking before sedation. *Br. Med. J.*, **314**, 162.

Greenough, A. (1990) Bronchopulmonary dysplasia. *Arch. Dis. Child.*, **65**, 1082–1088.

Greenough, A. (1995) Nitric oxide – clinical aspects. *Care Crit. Ill*, **11**, 143–147.

Greenough, A. and Yuksel, B. (1997) First trimester invasive procedures: effects on symptom status and lung volume in very young children. *Ped. Pulmonol.*, **24**, 412–422.

Greenwald, B. M. (1993) CPAP to treat profound hypoxaemia associated with acute severe asthma. *Crit. Care Med.*, **21**(suppl.) S154.

Greif, R., Laciny, S., Rapf, B. and Hickle, R. S. (1999) Supplemental oxygen reduces the incidence of postoperative nausea and vomiting. *Anesthesiogy*, **91**, 1246–1252.

Greif, R., Akca, O. and Horn, E. P. (2000)

Supplemental perioperative oxygen to reduce the incidence of surgical wound infection. *N. Engl. J. Med.*, 342, 161–167.
Gribbin, H. R. (1993) Management of respiratory failure. *Br. J. Hosp. Med.*, 49, 461–477.
Grief, R. (2000) Supplemental perioperative oxygen to reduce the incidence of surgical wound infection. *New Eng. J. Med.*, 342, 161–167.
Griffin, S. (2000) Nebulised steroid in the treatment of croup. *Br. J. Gen. Pract.*, 50, 135–141.
Griffiths, R. (2000) Breathing circuits and their uses. *Br. J. Periop. Nurs.*, 10, 55–59.
Griffiths, R. D. and Jones, C. (1999) Recovery from intensive care. *Br. Med. J.*, 319, 427–429.
Griffiths, T. L., Gregory, S. E. and Ward, S. A. (1996) Effects of a structured domiciliary exercise training programme on quality of life and walking tolerance in patients with severe COPD. *Eur. Respir. J.*, 9(23), 144s.
Griffiths, T. L., Burr, M. L. and Campbell, I. A. (2000) Results at 1 year of outpatient pulmonary rehabilitation: a randomised controlled trial. *Lancet*, 355, 362–368.
Gripp, C. L., Salvaggio, J. and Fratianne, R. B (1995) Use of burn intensive care unit gymnasium as an adjunct to therapy. *J. Burn Care Rehabil.* 16, 160–1.
Groen, J. J. (1979) The psychosomatic theory of bronchial asthma. *Psychother. Psychosom.*, 31, 38–48.
Gross, D. and King, M. (1984) High frequency chest wall compression. *Physiother. Can.*, 36, 137–139.
Grounds, M. (1997) Can enhancement of cardiovascular physiology reduce surgical mortality and morbidity? *Br. J. Intensive Care*, 7, 97–105.
De Guia, T. S. (1995) Inhaled corticosteroids in COPD. *Chest*, 108, 1486–1489.
Guérin, G. (1997) Facial mask noninvasive mechanical ventilation reduces incidence of nosocomial pneumonia. *Intensive Care Med.*, 23, 1024–1032.
Guernelli, J., Wainapel, S. F. and Pack, S. (1999) Morbidly obese patients with pulmonary disease. *Am. J. Phys. Med. Rehabil.*, 78, 60–65.
Guglielminotti, J. (1998) Effects of tracheal suctioning on respiratory resistances. *Chest*, 113, 1335–1338.
Guia, T. S. (1995) Inhaled corticosteroids in COPD. *Chest*, 108, 6, 1486–1489.
Gumery, L. B., Pryor, J., Prasad, S. A. and Dodd, M. (2001) *Clinical Guidelines for Physiotherapy Management of Cystic Fibrosis*, CSP, London.
Gunawardana, R. H. (2000) Postoperative crying in infants [letter]. *Anaesthesia*, 55, 197.
Guylay, J. (1989) What suicide leaves behind. *Issues Comp. Pediatr. Nurs.*, 12, 103–118.
Haake, R., Schlichtig, R. and Ulstad, D. R. (1987) Barotrauma. *Chest*, 91, 608–613.
Haas, F. and Haas, S. S. (1990) *The Chronic Bronchitis and Emphysema Handbook*, John Wiley, Chichester.
Hadfield, P. J. (1997) Treatment of otitis media with effusion in children with primary ciliary dyskinesia. *Clin. Otolaryngol.*, 22, 302–306.
Hadjistavropoulos, H. D. (1997) Judging pain in infants. *Pain*, 73, 319–324.
Hagarty, E. M. (1996) Use of pulse oximetry to determine oxygen prescription. *Respir. Care*, 41, 30–36.
Hagler, D. A. and Traver, G. A. (1994) Endotracheal saline and suction catheters. *Am. J. Crit. Care*, 3, 444–447.
Hahn, R. G. (1997) Haemodynamics during inhalation of a 50% N_2O/O_2 mixture. *Acta Anaesthesiol. Scand.*, 41, 485–491.
Hahn, A., Bach, J. R. and Delaubier, A. (1997) Clinical implications of maximal respiratory pressure determinations for individuals with Duchenne muscular dystrophy. *Arch. Phys. Med. Rehabil.*, 78, 1–6.
Hall, O. (1984) Physical and social problems of CF in the young adult. *ACPRC Newsl.*, 7, 29–33.
Hall, P. A. and Bowden, M. I. (1996) Introducing an acute pain service. *Br. J. Hosp. Med.*, 55, 15–17.
Hall, P. A., Payne, F. F. and Stack, C. G. (1995) Parents in the recovery room. *Br. Med. J.*, 310, 163–164.
Hall, J. C., Tarala, R. A. and Tapper, J. (1996) Prevention of respiratory complications after abdominal surgery. *Br. Med. J.*, 312, 148–153.
Hallsworth, M. (1995) Positioning the pre-term infant. *Paed. Nurs.* 7, 18–20.
Halpern, N. A. (1999) Hearing loss in critical care. *Crit. Care Med.*, 27, 211–219.
Hamdorf, P. A. (1999) Walking with its training effects on the fitness and activity patterns of 79–91 year old females. *Austr. NZ J. Med.*, 29, 22–28.
Hamilton, L. and Lyon, P. S. (1995) A nursing-driven program to preserve and restore functional ability in hospitalized elderly patients. *J. Nurs. Admin.*, 25, 30–37.
Hamm, H. and Light, R. W. (1997) The pleura: the outer space of pulmonary medicine. *Eur. Respir. J.*, 10, 2–3.

Hammon, W. E., Connors, A. F. and McCaffree, D. R. 1992 Cardiac arrhythmias during postural drainage and chest percussion of critically patients. *Chest*, 102, 1836–1841.

Hampson, N. B. (1988) Pulse oximetry in severe carbonmonoxide poisoning. *Chest*, 114, 1036–41.

Han, J. N., Stegen, K. and Simkens, K. (1997) Unsteadiness of breathing in patients with HVS and anxiety disorders. *Eur. Respir. J.*, 10, 167–176.

Hanks, G. W. (1996) Morphine in cancer pain. *Br. Med. J·*, 312, 823–826.

Hanley, M. V. and Tyler, M. L. (1987) Ineffective airway clearance related to airway infection. *Nurs. Clin. North Am.*, 22, 135–149.

Hanning, C. D. (1992) Prolonged oxygen therapy. *Br. J. Anaesth.*, 69, 115–116.

Hannon, J. C. (2000) The physics of Feldenkrais. *J. Bodywork Mov. Ther.*, 4, 114–122.

Hansell, D. M. (1998) Imaging pulmonary embolism. *Br. Med. J.*, 316, 490–491.

Haponik, E. F. (1992) Smoke inhalation injury. *Respir. Care*, 37, 609–617.

Haponik, E. F., Aquino, S. L. and Vining, D. J. (1999) Virtual bronchoscopy. *Clin. Chest Med.*, 20, 201–220.

Haraldsson, P.-O. (1990) Clinical symptoms of sleep apnea syndrome and automobile accidents. *ORL*, 52, 57–62.

Harcus, A. W. (1977) *Pain – New Perspectives in Measurement and Management*, Churchill Livingstone, Edinburgh, p. 39.

Harding, J. E., Miles, F. K. I. and Becroft, D. M. O. (1998) Chest physiotherapy may be associated with brain damage in extremely premature infants. *J. Pediatr.* 132, 440–444.

Harding, R. (1996) Children with cancer. *Prof. Nurse*, 11, 588–590.

Harding, S. M., Guzzo, M. R. and Richter J. E. (1999) 24-h esophageal pH testing in asthmatics. *Chest*, 115, 654–659.

Hardinge, F. M. (1995) Effects of short term high frequency negative pressure ventilation. *Thorax*, 50, 44–49.

Hardy, K. A. (1994) A review of airway clearance techniques. *Resp. Care*, 39, 440–455.

Hargreaves, M. R. (1995) Inhaled sodium cromoglycate in ACE-inhitor cough. *Lancet*, 345, 13–15.

Harmer, M. (1991) Postoperative pain relief. *Anaesthesia*, 46, 167–168.

Harper, R., Brazier, J. E., Waterhouse, J. C. and Walters, S. J. (1997) Comparison of outcome measures for patients with COPD in an outpatient setting. *Thorax*, 52, 879–887.

Harrington, G. R. (1993) Noninvasive monitoring. *Am. J. Med.*, 95, 221–227.

Harris, J. D. (1996) Rib cage and thoracic spine. *Phys. Med. Rehabil. Clin. North Am.*, 7, 761–771.

Harris, P. (1989) Are pulmonary haemodynamics of importance to survival in chronic obstructive lung disease? *Eur. Respir. J.*, 2(suppl. 7), 674s–677s.

Harris, M. L. and Moxham, J. (1998) Measuring respiratory and limb muscle strength using magnetic stimulation. *Br. J. Intensive Care*, 8, 21–26.

Harris-Eze, A. O. (1996) Role of hypoxemia and pulmonary mechanics in exercise limitation in interstitial lung disease. *Am. J. Respir. Crit. Care Med.*, 154, 994–1001.

Harrison, D. A. (1993) The performance of two intensive care humidifiers at high gas flows. *Anaesthesia*, 48, 902–905.

Harrison, P. (1995) The use of heliox to wean from mechanical ventilation. *Chest*, 108, 134S.

Harrison, L. (1996) Effects of gentle human touch on preterm infants. *Neonat. Netw.*, 15, 35–41.

Harrison, L. (1998) Clinical guidelines and protocols in ITU. *Care Crit. Ill*, 14, 155–157.

Harrison, T. W. (1999) Randomised placebo controlled trial of β-agonist dose reduction in asthma. *Thorax*, 54, 98–102.

Hartkopp, A., Hansen, H. and Seidenschnur, A.-M. (1998) Suicide in a spinal cord injured population. *Arch. Phys. Med. Rehabil.*, 79, 1356–1361.

Harvey, M. A. (1996) Managing agitation in critically ill patients. *Am. J. Crit. Care*, 5, 7–15.

Hasani, A., Pavia, D. and Agnew, J. E. (1994) Regional lung clearance during cough and FET. *Thorax*, 49, 557–561.

Hatipoglu, U. S., Laghi, F. and Tobin, M. J. (1999) Does inhaled albuterol improve diaphragmatic contractility in patients with COPD? *Am. J. Respir. Crit. Care Med.*, 160, 1916–1921.

Hayakawa, H., Sato, A. and Imokawa, S. (1996) Bronchiolar disease in rheumatoid arthritis. *Am. J. Respir. Crit. Care Med.*, 154, 1531–1536.

Hayden, R. A. (1993) Trend-spotting with the $S\bar{v}O_2$ monitor. *Am. J. Nurs.*, 93, 26–33.

Hazelrigg, S., Boley, T. and Henkle, J. (1996) Thoracoscopic laser bullectomy. *J. Thorac. Cardiovasc. Surg.*, 112, 319–327.

HEA (1995) *Passive Smoking*. Health Education Authority, London.

Hecht, S. (1993) A tobacco-specific carcinogen in the urine of men exposed to cigarette smoke. *N. Engl. J. Med.*, 329, 1543–1546.

Hedenstierna, G. (1997) Lung function during anesthesia. *Curr. Opin. Anaesthesiol.*, 10, viii–xi.

Hedenstierna, G. (1999) Invited editorial on kinetics of absorption atelectasis during anesthesia. *J. Appl. Physiol.*, 86, 1114.

Heffner, J. E. (1990) Pulmonary reactions from illicit substance abuse. *Clin. Chest Med.*, 11, 151–161.

Heffner, J. E. (1996) Advance directive education during pulmonary rehabilitation. *Chest*, 109, 373–379.

Heijden, H. F. M. van der, Dekhuijzen, P. N. R. and Folgering, H. (1996) Pharmacotherapy of respiratory muscles in COPD. *Respir. Med.*, 90, 513–522.

Heijdra, Y. F. (1994) Effects of body position, hyperinflation and blood gas tensions on maximal respiratory pressures in patients with COPD. *Thorax*, 49, 453–458.

Heijdra, Y. F. (1996) Nocturnal saturation improves by target-flow inspiratory muscle training in patients with COPD. *Am. J. Respir. Crit. Care Med.*, 153, 260–265.

Hellman, E. A. (1994) Outpatient cardiac rehabilitation in elderly patients. *Heart Lung*, 23, 506–512.

Henderson, R. C. (1999) Bone mineral content and body composition in children and young adults with CF. *Pediatr. Pulmonol.*, 27, 80–84.

Henzi, I. (2000) Dexamethasone for the prevention of postoperative nausea and vomiting. *Anesth. Analg.*, 90, 186–194.

Hernandez-Reif, M. (2000) High blood pressure and associated symptoms reduced by massage therapy. *J. Bodywork Mov. Ther.* 4, 31–38.

Herr, K. A. (1998) Evaluation of the faces pain scale for use with the elderly. *Clin. J. Pain.*, 14, 29–38.

Herridge, M. S. and Slutsky, A. S. (1996) High-frequency ventilation. *Respir. Care*, 41, 385–393.

Herring, D., King, A. I. and Connelly, M. (1987) New rehabilitation concepts in management of radical neck dissection syndrome. *Phys. Ther.*, 67, 1095–1099.

Hertanu, J. S. and Moldover, J. R. (1996) Cardiovascular, pulmonary and cancer rehabilitation. 1. Cardiac rehabilitation. *Arch. Phys. Med. Rehabil.*, 77, S38–S44.

Heslop, A. and Shannon, C. (1995) Assisting patients living with long-term oxygen therapy. *Br. J. Nurs.*, 4, 1123–1128.

Hess, D. (1994) Inhaled bronchodilators during mechanical vantilation *Respir. Care*, 39, 105–121.

Heulitt, M. J. (1995) Acute respiratory distress syndrome in pediatric patients. *Respir. Care*, 40, 74–85.

Hickey, P. R., Hansen, D. D. and Wessel, D. L. (1985) Blunting of stress responses in the pulmonary circulation of infants by fentanyl. *Anesth. Analg.*, 64, 1137–1142.

Hickling, K. G. (1994) Low mortality in ARDS using permissive hypercapnia. *Crit. Care Med.*, 22, 1568–1578.

Hilbert, G., Gruson, D. and Portel, L. (1998) Noninvasive pressure support ventilation in COPD patients with postextubation hypercapnic respiratory insufficiency. *Eur. Respir. J.*, 11, 1349–1353.

Hill, L. L. and Pearl, R. G. (1999) Combined inhaled nitric oxide and inhaled prostacyclin. *J. Appl. Physiol.*, 86, 1160–1164.

Hill, S. (1989) *Family*, Michael Joseph, London.

Hindle, T., Dhoot, R. and Georgieva, C. (1996) Clinical nutrition in NHS hospitals. *Br. J. Intensive Care*, 6, 61–65.

Hirschberg, A. J. (1994) Use of doxapram hydrochloride as an alternative to intubation for COPD patients with hypercapnia. *Ann. Emerg. Med.*, 4, 701–703.

Hjelmqvist, H. (2000) Colloids versus crystalloids. *Curr. Anaesth. Crit. Care*, 11, 7–10.

Hjuler, I. M. (1995) Bacterial colonization of the larynx and trachea in healthy children. *Acta Paediatr.*, 84, 566–568.

Hnatiuk, O. W. (1998) Delivery of high concentrations of inspired oxygen via Tusk mask. *Crit. Care Med.*, 26, 1032–1035.

Hodge, D. (1991) Endotracheal suctioning and the infant. *Neonat. Netw.*, 9, 7–14.

Hodgkin, J. E. and Petty, T. L. (1987) *Chronic Obstructive Pulmonary Disease*, W. B. Saunders, London.

Hodgkin, J. E., Connors, G. L. and Bell, C. W. (1993) *Pulmonary Rehabilitation: Guidelines to Success*, Butterworth, London.

Hodgkin, J. E., Connors, G. L. C. and Celli, B. R. (2000) *Pulmonary Rehabilitation: Guidelines to Success*, 3rd edn. Lippincott, Philadelphia.

Hodgson, C., Denehy, L. and Ntoumenopoulos, G.

(1996) The acute cardiorespiratory effects of manual lung hyperinflation in ventilated patients. *Eur. Respir. J.*, 9(suppl. 23), 37s.
Hoffman, G. M. (1995) Airway complications of instrumentation. *Respir. Care*, 40, 97–107.
Hoffmann, B. and Welte, T. (1999) The use of noninvasive pressure support ventilation for severe respiratory insufficiency due to pulmonary oedema. *Intensive Care Med.*, 25, 15–20.
Hofman, J. C., Wenger, N. S. and Davis, R. B. (1997) Patient preferences for communication with physicians about end-of-life decisions. *Ann. Intern. Med.*, 127, 1–12.
Hogan, W. J. (2000) Life after antireflux surgery. *Am. J. Med.*, 108, 181s–191s.
Holden, T. (1980) Patiently speaking. *Nurs. Times*, 76(24), 1035–1036.
Holditch, D. (1994) Pathologic apnea and brief respiratory pauses in preterm infants. *Nurs. Res.*, 43, 293–299.
Holgate, S. T. (1996) Inhaled sodium cromoglycate. *Respir. Med.*, 90, 387–390.
Holleman, D. R. (1995) Does the clinical examination predict airflow limitation? *J. A. M. A.*, 273, 313–319.
Holliday, J. F. (1999) Reduction in ventilator response to CO_2 with relaxation feedback. *Chest*, 115, 1285–1292.
Holmes, P. W., Barter, C. E. and Pierce, R. J. (1992) Chronic persistent cough: use of ipratropium bromide. *Respir. Med.*, 86, 425–429.
Homma, S., Kawabata, M. and Kishi, K. (1999) Successful treatment of refractory bronchorrhea by inhaled indomethacin. *Chest*, 115, 1465–1468.
Hoozen, B. E. V. (1997) Marijuana. *Clin. Rev. Allerg. Immunol.*, 15, 243–268.
Hopp, L. J., Kim, M. J. and Larson, J. L. (1996) Incremental threshold loading in patients with COPD. *Nurs. Res.*, 45, 196–202.
Horiuchi, K. (1995) Propofol attenuates the metabolic and cardiopulmonary responses to stressful ICU procedures. *Crit. Care Med.*, 23, A36 (suppl.).
Hormann, C. (1994) Effect of kinetic therapy in patients with severe ARDS. *Crit. Care Med.*, 22, A87.
Horton, R. (1996) How often do children cough? *Lancet*, 348, 699–670.
Hosker, H. S. R. (1995) Assessment and provision of home nebulizers for COPD. *Respir. Med.*, 89, 47–52.
Hospital Update (1995) An epidemiological overview of asthma. *Hosp. Update*, 21, 369.
Houtmeyers, E. (1999) Effects of drugs on mucus clearance. *Eur. Respir. J.*, 14, 452–467.
Houtmeyers, E., Gosselink, R. and Gayan-Ramirez, G. (1999) Regulation of mucociliary clearance in health and disease. *Eur. Respir. J.*, 13, 1177–1188.
Howard, F. (1994) Endotracheal suctioning and the neonate. *Paediatr. Nurs.*, 6, 14–17.
Howard, L. and Koniak, D. (1990) Evaluation of manometer use in mechanical ventilation of infants. *Heart Lung*, 19, 620–627.
Howes, T. Q., Deane, C. R. and Levin, G. E. (1995) The effects of oxygen and dopamine on renal and aortic blood flow in COPD. *Am. J. Respir. Crit. Care Med.*, 151, 378–383.
Howie, A. (1999) Rapid shallow breathing as a predictive indicator during weaning. *Nurs. Crit. Care*, 4, 171–178.
Hsia, C. C. W. (1993) Respiratory muscle limitation in patients after pneumonectomy. *Am. Rev. Respir. Dis.*, 147, 744–752.
Hsiang, J. (1993) Paralysis for ICP control in severe head injury. *Crit. Care Med.*, 21, S236.
Hsieh, A. H., Bishop, M. J. and Kubilis, P. S. (1992) Pneumonia following closed head injury. *Am. Rev. Respir. Dis.*, 146, 290–294.
Huang, D., Ades, P. A. and Weaver, S. (1990) Cardiac rehospitalizations and costs are reduced following cardiac rehabilitation. *J. Cardiopulm. Rehabil.*, 10, 108.
Hubbard, R., Johnston, I. and Britton, J. (1998) Survival in patients with cryptogenic fibrosing alveolitis. *Chest*, 113, 396–400.
Hudes, E. T. (1989) Recovery room oxygenation. *Can. J. Anaesth.*, 36, 20–24.
Hughes, R. L. (1980) Do no harm – cheaply. *Chest*, 77, 582–583.
Hugli, O. (1996) The daily energy expenditure in stable COPD. *Am. J. Respir. Crit. Care Med.*, 153, 294–300.
Huib, A. M. K. (1999) Stable chronic obstructive pulmonary disease. *Br. Med. J.*, 319, 495–500.
Hund, E. (1999) Myopathy in critically ill patients. *Crit. Care Med.*, 27, 2544–2547.
Hung, C.-H., Chu, D.-M. and Wang, C.-L. (1999) Hypokalemia and salbutamol therapy in asthma. *Pediatr. Pulmonol.*, 27, 27–31.
Hunt, D., Leighton, M. and Reed, G. (1999) Intermittent claudication: implementation of an exercise programme. *Physiotherapy*, 85, 149–153.

Hurewitz, A. N. (1985) Obesity alters regional ventilation in lateral decubitus position. *J. Appl. Physiol.*, 59, 774–783.

Hutchon, B. (2000) Cradle philosophy. *Ther. Weekly*, 26, 6.

Hyde, J. A., Rooney, S. J. and Graham, T. R. (1996) Fluid management in thoracic trauma. *Hosp. Update*, 22, 448–452.

Hyde, V. (1999) Constipation and laxative use in older community-dwelling adults. *Clin. Effect. Nurs.*, 3, 180–80.

Ibañez, J., Raurich, J. M. and Abinanda, R. (1981) The effect of lateral positions on gas exchange in patients with unilateral lung disease during mechanical ventilation. *Intensive Care Med.*, 7, 231–234.

Iles, R., Lister, P. and Edmunds, A. T. (1999) Crying significantly reduces the absorption of aerosolised drug in infants. *Arch. Dis. Child.*, 81, 163–165.

Ing, A. J., Ngu, M. C. and Breslin, A. B. X. (2000) Obstructive sleep apnea and GOR, *Am. J. Med.*, 108, 120s–125s.

Ingram, R. H. (1967) Effect of pursed lips expiration on the pulmonary pressure-flow relationship in obstructive lung disease. *Am. Rev. Respir. Dis.*, 96, 381–388.

Inman, M. D. (1996) The effect of regular inhaled albuterol on exercise-induced bronchoconstriction. *Am. J. Respir. Crit. Care Med.*, 153, 65–69.

Inman, C. L., Warren, G. L. and Hogan, H. A. (1999) Mechanical loading attenuates bone loss due to immobilization and calcium deficiency. *J. Appl. Physiol.*, 87, 189–195.

Innocenti, D. M. (1966) Breathing exercises in the treatment of emphysema. *Physiotherapy*, 52, 437–441.

Innocenti, D. M. (1974) Physiotherapy in the management of acute asthma. *Nurs. Mirror*, 139, 77–79.

Innocenti, D. M. (1997) Breathing re-education [letter]. *Physiotherapy*, 83, 269.

Innocenti, D. M. (1998) Hyperventilation, in *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, (eds B. A. Webber and J. A. Pryor), Churchill Livingstone, Edinburgh.

Ioli, F., Casaburi, R., Patessio, A. and Donner, C. F. (1991) Exercise prescription. *Eur. Respir. Rev.*, 1, 486–489.

Irwin, R. S., Boulet, L.-P. and Cloutier, M. M. (1998) Managing cough as a defense mechanism and as a symptom. *Chest*, 114, 133S–179S.

Isaacs, D. (1995) Bronchiolitis. *Br. Med. J.*, 310, 4–5.

Jacavone, J. (1998) Use of pulmonary rehabilition stretegies to wean a difficult-to-wean patient. *Crit. Care Nurse*, 18, 29–37.

Jackson, G. (1999) Recognising and rewarding good performance: the importance of being valued. *Int. J. Clin. Pract.*, 53, 3.

Jackson, M., Flower, C. D. R. and Shneerson, J. M. (1993) Treatment of symptomatic pulmonary aspergillomas. *Thorax*, 48, 928–930.

Jacobsson, C. (2000) How people with stroke and healthy older people experience the eating process. *J. Clin. Nurs*, 9, 255–264.

Jacox, A., Carr, D. and Chapman, C. R. (1992) Clinician's quick reference guide to postoperative pain management in adults. *J. Pain Symptom Manage.*, 7, 214–228.

Jacques, A. (1994) Epidural analgesia. *Br. J. Nurs.*, 3, 734–738.

Jacyna, M. R. (1992) Pros and cons of medical audit. *Hosp. Update*, 18, 512–516.

Jaffé, A., Bush, A. and Geddes, D. M. (1999) Prospects for gene therapy in CF. *Arch. Dis. Child.*, 80, 286–289.

Jain, A. (2000) Local anaesthetic effect of topical amethocaine gel in neonates. *Arch. Dis. Child. Fetal Neonat. Ed.*, 82, F42–F45.

Jalleh, R., Fitzpatrick, M. F. and Jan, M. A. (1993) Alcohol and cor pulmonale in chronic bronchitis and emphysema. *Br. Med. J.*, 306, 374.

James, I. (1991) Respiratory management in paediatrics. *Care Crit. Ill*, 7, 47–50.

James, A., Williams, J. and Wilkins, W. E. (1998) A pre-discharge ward for acute medical patients. *J. Roy. Coll. Phys.*, 32, 231–234.

Jansen, H. M. (1995) Predisposing conditions to bacterial infections in COPD. *Am. J. Respir. Crit. Care Med.*, 151, 2073–2080.

Janson, C. (2000) What characterizes patients who are unable to tolerate CPAP? *Respir. Med.*, 94, 145–149.

Janssens, J. P. (1998) Transcutaneous PCO_2 to monitor noninvasive mechanical ventilation. *Chest*, 113, 768–773.

Janssens J. P. (2000) Management of dyspnea in severe COPD. *J. Pain Symptom. Man.* 19, 378–392.

Janssens, J. P., Pache, J. C. and Nicod, L. P. (1999) Physiological changes in respiratory function associated with ageing. *Eur. Respir. J.*, 13, 197–205.

Jantz, M. A. and Pierson, D. J. (1994) Pneumothorax and barotrauma. *Clin. Chest Med.*, **15**, 75–92.

Jarvik, R., Westaby, S. and Katsumata, T. (1998) LVAD power delivery. *Ann. Thorac. Surg.*, 65, 470–473.

Jarvis, M. J. (1986) Low cost carbon monoxide monitor in smoking assessment. *Thorax*, **41**, 886–887.

Jarvis, M. J. (1995) Smoking cessation. *Thorax*, 50(suppl. 1), S22–S24.

Jederlinic, P., Muspratt, J. A. and Miller, M. J. (1984) Inspiratory muscle training in clinical practice. *Chest*, 86, 870–873.

Jefferies, A. and Turley, A. (1999) *Crash Course: Respiratory System*, Mosby, London.

Jenkin, A. (1996) The nasopharyngeal airway. *Accident Emerg. Nurs.*, 4, 16–20.

Jenkins, S. C., Heaton, R. W. and Fulton, T. J. (1987) Comparison of domiciliary nebulized salbutamol and salbutamol from a metered dose aerosol in stable COPD. *Chest*, **91**, 804–807.

Jenkins, S. C. and Moxham, J. (1991) The effects of mild obesity on lung function. *Respir. Med.*, 85, 309–311.

Jenkins, S. C., Soutar, S. A. and Moxham, J. (1988) The effects of posture on lung volumes in normal subjects and in patients pre- and post-coronary artery surgery. *Physiotherapy*, 74, 492–496.

Jennings, P. (1992) Coping mechanisms. *Pediatr. Nurs.*, 4, 13–15.

Jennische, M. and Sedin, G. (1999) Speech and language skills in children who required neonatal intensive care. *Acta Paediatr.*, 88, 975–982.

Jette, A. M. (1993) Tobacco use; a modifiable risk factor for dental disease among the elderly. *Am. J. Publ. Health*, 83, 1271–1274.

Jette, D. U., Manago, D. and Medved, E. (1997) The disablement process in patients with pulmonary disease. *Eur. Respir. J.*, 77, 385–394.

Jindani, A. (1993) Postoperative cardiac surgical care. *Br. Heart J.*, 69, 59–64.

Jobst, K., McPherson, K. and Brown, V. (1986) Controlled trial of acupuncture for disabling breathlessness. *Lancet*, ii, 1416–1418.

Johnson, K. L. (1994) Closed versus open endotracheal suctioning. *Crit. Care Med.*, 22, 658–666.

Johnson, J. H. and Prins, A. (1991) Prediction of maximal heart rate during a submaximal work test. *J. Sports Med. Phys. Fitness*, 31, 44–47.

Johnson, M. K. (2000) Large lung bullae in marijuana smokers. *Thorax*, 55, 340–342.

Johnston, I. D. A., Prescott, R. J., Chalmers, J. C. and Rudd, R. M. (1997) British Thoracic Society study of cryptogenic fibrosing alveolitis. *Thorax*, 52, 38–44.

Johnstone, M. (1994) Children visiting members of their family receiving treatment in ICUs. *Intens. Crit. Care Nurs.*, 10, 289–292.

Jokic, R. (1998) Continuous positive airway pressure requirement during the first month of treatment in patients with severe OSA. *Chest*, 114, 1061–1069.

Jolliet, P. (1999) Beneficial effects of helium:oxygen versus air:oxygen noninvasive pressure support. *Crit. Care Med.*, 27, 2422–2429.

Joly, H. R. and Weil, M. H. (1969) Temperature of the great toe as an indication of the severity of shock. *Circulation*, 39, 131–138.

Jones, A. Y. M. and Jones, R. D. (2000) The effect on sound generation of varying both gas flow rate and the viscosity of sputum-like gel. *Lung*, 178, 31–40.

Jones, P. D. (1985) Microbial contamination of domiciliary nebulizer equipment. *Austr. NZ J. Med.*, 15, 585–589.

Jones, M. J. (1998) Neurophysiological facilitation of respiration. Cardiorespiratory physiotherapy conference, London.

Jones, J. (2000) Pet therapy for elderly wins growing recognition. *Br. Med. J.*, 320, 602.

Jones, C. and Barrett, C. (1995) The role of the pharmacist in asthma care. *Respir. Dis. Pract.*, 12, 25–26.

Jones, A. and Bowen, M. (1994) Screening for childhood asthma using an exercise test. *Br. J. Gen. Pract.*, 44, 127–131.

Jones, C. and Griffiths, R. (1998) Intensive care follow-up workshop. *Br. J. Intensive Care*, 8, 139–140.

Jones, C. and O'Donnell, C. (1994) After intensive care – what then? *Intens. Crit. Care Nurs.*, 10, 89–92.

Jones, A. L. and Volans, G. (1999) Management of self-poisoning. *Br. Med. J.*, 319, 1414–1417.

Jones, D. and West, R. (1995) *Cardiac Rehabilitation*, BMJ Publishing, London.

Jones, A., Tse, E. and Cheung, L. (1997) Restoration of lung volume using the flutter or breathing exercise. *Austr. Physiother. J.*, 43, 183–189.

Jones, A., Jones, R. D., Kwong, K. and Burns, Y. (1999) Effect of positioning on recorded lung sound intensities in subjects without pulmonary dysfunction. *Phys. Ther.*, 79, 682–690.

Jones, T. R., Allen, A. and Hoy, D. (2000) Mass

psychogenic illness attributed to toxic exposure at a high school. *N. Engl. J. Med.*, 342, 96–100.

Joorabchi, B. (1997) Expressions of the hyperventilation syndrome in childhood. *Clin. Pediatr.*, **16**, 1110–1115.

Jorm, A. F., Rodgers, B. R. and Jacomb, P. A. (1999) Smoking and mental health. *Med. J. Austr.*, 170, 74–77.

Jounieaux, V. (1995) Oxygen cost of breathing in patients with emphysema or chronic bronchitis in acute respiratory failure. *Am. J. Respir. Crit. Care Med.*, 152, 2181–2184.

Joynt, G. M. and Lipman, J. (1994) The use of heat and moisture exchangers in critically ill patients. *Care Crit. Ill*, 10, 271–274.

Jubran, A. (1994) Use of flow-volume curves in detecting secretions in ventilator-dependent patients. *Am. J. Respir. Crit. Care Med.*, 150, 766–769.

Judson, M. A. (1994) Mobilization of secretions in ICU patients. *Respir. Care*, 39, 213–225.

Judson, M. A. (1998) Lung transplantation for pulmonary sarcoidosis. *Eur. Resp. J.*, 11, 738–744.

Juniper, E. F. (1999a) Validation of a standardized version of the asthma qualiity of life questionnaire. *Chest*, 115, 1265–1270.

Juniper, M. (1999b) Ventilator associated pneumonia. *Care Crit. Ill*, 15, 198–201.

Juniper, M. C. and Davies, C. W. H. (1998) New guidelines for the management of life threatening asthma. *Care Crit. Ill*, 14, 61–63.

Juniper, M. C. and Garrard, C. S. (1997) The chest X-ray in intensive care. *Care Crit. Ill*, 13, 198–200.

Justins, D. M. and Richardson, P. H. (1991) Clinical management of acute pain. *Br. Med. Bull.*, 47, 561–583.

Kain, Z. N., Wang, S. M. and Caramico, L. A. (1997) Parental desire for perioperative information and informed consent. *Anesth. Analg.*, 84, 299–306.

Kakizaki, F., Shibuya, M. and Yamazaki, T. (1999) Preliminary report on the effects of respiratory muscle stretch gymnastics on chest wall mobility. *Respir. Care*, 44, 409–414.

Kallet, R. H. (2000) The effects of pressure control versus volume control asisted ventilation on patient work of breathing. *Respir. Care*, 45, 1085–1096.

Kam, P. C. A. (1994) Noise pollution in the anaesthetic and intensive care environment. *Anaesthesia*, 49, 982–986.

Kam, A. C., O'Brien, M. and Kam, P. C. A. (1993) Pleural drainage systems. *Anaesthesia*, 48, 154–161.

Kamalvand, K. and Sulke, N. (1999) Recent advances in the non-surgical management of atrial fibrillation. *Int. J. Clin. Pract.*, 53, 44–46.

Kamps, A. W. A. (2000) Poor inhalation technique, even after inhalation instructions, in children with asthma. *Pediatr. Pulmonol.*, 29, 39–42.

Kandioler, D. (1998) Long-term results after repeated surgical removal of pulmonary metastases. *Ann. Thorac. Surg.*, 65, 909–912.

Kapur, N., House, A. and Creed, F. (1998) Management of deliberate self poisoning in adults. *Br. Med. J.*, 316, 831–832.

Karetzky, M. S. (1967) The cause of hyperventilation and arterial hypoxia in patients with cirrhosis of the liver. *Am. J. Med.*Sci, 254, 797–804.

Karper, W. B. and Boschen, M. B. (1993) Effects of exercise on acute respiratory tract infections and related symptoms. *Geriatr. Nurs.*, 14, 15–18.

Katz, J., Clairoux, M. and Kavanagh, B. P. (1994) Preemptive lumber epidural anaesthesia reduces postoperative pain. *Pain*, 59, 395–403.

Katz, J., Jackson, M. and Kavanagh, B. P. (1996) Acute pain after thoracic surgery predicts long-term post-thoracotomy pain. *Clin. J. Pain.*, 12, 50–55.

Katz, M. G., Katz, R. and Schachner, A. (1998) Phrenic nerve injury after CABG. *Ann. Thorac. Surg.*, 65, 32–35.

Kauffmann, F. (1993) The ageing lung. *Respir. Med.*, 87, 5–7.

Kavanagh, B. P. (1994) Pain control after thoracic surgery. *Anesthesiology*, 81, 737–759.

Kawagoe, Y. (1994) Hyperinflation with intrinsic PEEP. *J. Appl. Physiol.*, 77, 2440–2448.

Kawamura, H. (1999) The effects of pre-and-postoperative contralateral TENS for the phantom limb pain in amputees. *WCPT Proceedings*, p. 61.

Keeley, D. (1999) Rigorous assessment of palliative care revisited. *Br. Med. J.*, 319, 1447–1448.

Keenan, S. P. (1997) Effect of noninvasive positive pressure ventilation on mortality in patients with acute respiratory failure. *Crit. Care Med.*, 25, 1685–1692.

Keilty, S. E. J. and Bott, J. (1992) Continuous positive airways pressure. *Physiotherapy*, 78, 90–92.

Keilty, S. E. J. and Moxham, J. (1995) Noninvasive ventilation in acute-on-chronic airways disease. *Hosp. Update*, 21, 165–171.

Keistinen, T. (1997) Bronchiectasis: an orphan disease with a poorly-understood prognosis. *Eur. Respir. J.*, **10**, 2784–2787.

Kelley, S. and Jull, G. (1998) Breast surgery and neural tissue mechanosensitivity. *Austr. Physiother.*, 44, 31–37.

Kellner, R., Samet, J. and Pathak, D. (1992) Dyspnea, anxiety and depression in chronic respiratory impairment. *Gen. Hosp. Psychiat.*, **14**, 20–28.

Kemper, M. (1993) The effects of adding pressure support during chest physical therapy. *Respir. Care*, 38, 1245.

Kennell, J. H. (1999) The humane neonatal care initiative. *Acta Paediatr.*, 88, 367–370.

Kerr, G. W. (1998) Neurogenic pulmonary oedema. *J. Accident Emerg. Med.*, **15**, 275–277.

Kerr, K. (1999) Exercise – no easy option. *Physiotherapy*, 85, 114–115.

Kerr, M., Sereika, S. and Rudy, E. (1996) The effect of neuromuscular blocking agents on ICP response to endotracheal suctioning in severe head injured patients. *Crit. Care Med.*, 24, A24.

Kerridge, I. H., Pearson, S. A., Rolfe, I. E. and Lowe, M. (1998) Decision making in CPR. *Med. J. Austr.*, **169**, 128–131.

Kesecioglu, J. (1997) Prone position in therapy-refractory hypoxaemia. *Curr. Opin. Anaesth.*, **10**, 92–100.

Khadra, M. H. (1998) What price, compassion? *Med. J. Austr.*, **169**, 42–43.

Khajotia, R. R. (1991) Induced sputum and cytological diagnosis of lung cancer. *Lancet*, 338, 976–977.

Kiely, J. L. (1999) Subjective efficacy of nasal CPAP therapy in OSA. *Eur. Respir. J.*, **13**, 1086–1090.

Kiernan, M. (1998) Pressure sores. *Br. J. Ther. Rehabil.*, **5**, 402–405.

Kikuchi, R., Watabe, N. and Konno, T. (1994) High incidence of silent aspiration in elderly patients with community-acquired pneumonia. *Am. J. Respir. Crit. Care Med.*, **150**, 251–253.

Kimmel, P. L., Miller, G. and Washington, W. B. (1989) Sleep apnoea syndrome in chronic renal disease. *Am. J. Med.*, 86, 308–314.

King, M. (1985) Clearance of mucus by simulated cough. *J. Appl. Physiol.*, 58, 1776–1782.

King, M. (1997) Mucoactive therapy. *Pediatr. Pulmonol. Suppl.*, **14**, 122–123.

King, M. (1998) Experimental models for studying mucociliary clearance. *Eur. Respir. J.*, **11**, 222–228.

King, D. and Morrell, A. (1992) A survey on manual hyperinflation as a physiotherapy technique in intensive care units. *Physiotherapy*, 78, 747–750.

King, J. C. and Nixon, P. G. F. (1988) A system of cardiac rehabilitation: psychophysiological basis and practice. *Br. J. Occup. Ther.*, **51**, 378–383.

King, M., Dasgupta, B. and Tomkiewicz, R. P. (1997) Rheology of cystic fibrosis sputum after in vitro treatment with hypertonic saline alone and in combination with rhDNase. *Am. J. Respir. Crit. Care Med.*, **156**, 173–177.

Kings Fund (1998) *Trends in Rehabilitation Policy and Effective Practice in Rehabilitation*, Kings Fund, London.

Kinsman, R. A., Yaroush, R. A. and Fernandez, E. (1983) Symptoms and experiences in chronic bronchitis and emphysema. *Chest*, 83, 755–761.

Kirk, A. J. B. (1986) Minitracheostomy in cardiothoracic practice. *Care Crit. Ill*, 2, 104–110.

Klech, H. H. (1998) Reduced smoking – an acceptable goal for the hopeless heavy smoker? *Eur. Respir. J.*, **11**, 263–264.

Klefbeck, B. (1999) Combination of inspiratory muscle training and assisted ventilation in patients with prior polio. *WCPT Proceedings*, 141.

Kleiber, C., Krutzfield, N. and Rose, E. F. (1988) Acute histologic changes in the tracheobronchial tree associated with different suction catheter insertion techniques. *Heart Lung*, 17, 10–14.

Klein, J. S. (1999) Chest imaging in the surgical ICU. *Respir. Care*, 44, 1078–1094.

Klein, P., Kemper, M. and Weissman, C. (1988) Attenuation of the hemodynamic responses to chest physical therapy. *Chest*, 93, 38–42.

Klingstedt, C., Hedenstierna, G. and Lundquist, H. (1991) The influence of body position and differential ventilation on lung dimensions and atelectasis. *Respir. Care*, 36, 12.

Kluft, J., Beker, L. and Castagnino, M. (1996) A comparison of bronchial drainage treatments in CF. *Pediatr. Pulmonol.*, 22, 271–274.

Knichwitz, G., Rötker, J. and Möllhoff, T. (1998) Continuous intramucosal PCO_2 measurment allows the early detection of intestinal malperfusion. *Crit. Care Med.*, 25, 1550–1557.

Kochanek, P. M. (1999) Subarachnoid hemorrhage, systemic immune response syndrome and MODS. *Crit. Care Med.*, 27, 454–455.

Kolaczkowski, W. (1989) Improvement in oxygen saturation after chest physiotherapy in patients with emphysema. *Physiother. Can.*, 41, 18–23.

Koll, U., Matsuda, Y. and Takayanagi, K. (1999) Assessment of health-related quality of life in patients with chronic respiratory failure. *WCPT Proceedings*, p. 318.

Koltyn, K. F. and Morgan, W. P. (1992) Efficacy of perceptual versus heart rate monitoring in the development of endurance. *Br. J. Sports Med.*, 26, 132–134.

Komara, J. J. and Stoller, J. K. (1995) The impact of a postoperative oxygen therapy protocol. *Respir. Care*, 40, 1125–1129.

Kong, X. T., Fang, H. T. and Jang, G. Q. (1993) Treatment of acute bronchiolitis with Chinese herbs. *Arch. Dis. Child.*, 68, 468–471.

Konrad, F. X. (1993) Bronchial mucus transport in chronic smokers and nonsmokers during general anesthesia. *J. Clin. Anesth.*, 5, 375–380.

Konstan, M. W. (1994) Efficacy of the flutter device for airway mucus clearance in patients with CF. *J. Pediatr.*, 124, 689–693.

Konstan, M. W., Byard, P. J. and Hoppel, C. (1995) Effect of high dose ibuprofen in patients with cystic fibrosis. *N. Engl. J. Med.*, 332, 848–854.

Korhonen, K., Korppi, M. and Remes, S. T. (1999) Lung function in school-aged asthmatic children with inhaled cromoglycate, nedocromil and corticosteroid therapy. *Eur. Respir. J.*, 13, 82–86.

Kotloff, R. M. (1998) Comparison of short-term functional outcomes following unilateral and bilateral lung volume reduction surgery. *Chest*, 113, 890–895.

Koufman, J. A. and Burke, A. J. (1997) The etiology and pathogenesis of laryngeal carcinoma. *Otolaryngol. Clin. North Am.*, 30, 1–5.

Koul, B., Solem, J.-O. and Steen, S. (1998) HeartMate left ventricular assist device as bridge to heart transplantation. *Ann. Thorac. Surg.*, 65, 1625–1630.

Kraft, A. R. (1984) The hyperventilation syndrome. *Br. J. Psychiat.*, 145, 538–542.

Krämer, B. K., Schweda, F. and Riegger, G. A. J. (1999) Diuretic treatment and diuretic resistance in heart failure. *Am. J. Med.*, 106, 90–96.

Krause, J. S. (2000) Depression after spinal cord injury. *Arch. Phys. Med. Rehap.*, 81, 1099–1109.

Krishna, G. (1999) Terminal weaning from mechanical ventilation. *Crit. Care Med.*, 27, 9–10.

Kritz, H. (1995) Passive smoking and cardiovascular risk. *Arch. Intern. Med.*, 155, 1942–1948.

Kübler-Ross, E. (1973) *On Death and Dying*, Tavistock, London.

Kuo, C. D., Lin, S. E. and Wang, J. H. (1991) Aerosol, humidity and oxygenation. *Chest*, 99, 1352–1356.

Kuo, C. D., Shiao, G. M. and Lee, J. D. (1993) The effects of high-fat and high-carbohydrate diet loads on gas exchange and ventilation in COPD patients and normal subjects. *Chest*, 104, 189–196.

Kutsogiannis, D. J. (2000) Continuous venovenous hemodiafiltration for renal failure and sepsis. *Can. Med. Assoc. J.*, 162, 537–538.

Kuwahira, I. (1993) Changes in regional blood flow distribution and oxygen supply during hypoxia in conscious rats. *J. Appl. Physiol.*, 74, 211–214.

Kuyvenhoven, M. M. (2000) Antimicrobial agents in lower respiratory tract infections. *Br. J. Gen. Pract.*, 50, 133–134.

Laatikainen, A. K. (1999) Bone mineral density in perimenopausal women with asthma. *Am. J. Respir. Crit. Care Med.*, 159, 1179–1185.

Lacasse, Y. (1996) Meta-analysis of respiratory rehabilitation in COPD. *Lancet*, 348, 1115–1119.

Lacasse, Y., Rousseau, L. and Maltais, F. (1998) Prevalence and impact of depression in patients with severe COPD. *Chest*, 114, 319.

LaChapelle, D. L., Hadjistavropoulos, T. and Craig, K. D. (1999) Pain measurement in persons with intellectual disabilities. *Clin. J. Pain.* 15, 13–23.

Lagergren, J., Bergström, R. and Lindgren, A. (1999) Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. *N. Engl. J. Med.*, 340, 825–831.

Laghi, F. (1995) Weaning from mechanical ventilation. *Curr. Opin. Crit. Care*, 1, 71–76.

Lahz, S. (1996) Incidence of chronic pain following traumatic brain injury. *Arch. Phys. Med. Rehabil.*, 77, 889–891.

Lakhani, M. K. (1996) The dizzy patient. *Update*, 52, 383–386.

Lalloo, U. G. (1998) Increased sensitivity of the cough reflex in progressive systemic sclerosis patients with interstitial lung disease. *Eur. Respir. J.*, 11, 702–705.

Lam, S. (1985) Effect of bronchial lavage volume of cellular and protein recovery. *Chest*, 88, 856–859.

Lamm, C., Mandeli, J. and Kattan, M. (1999) Evaluation of audiotapes as an abbreviated test for OSA in children. *Pediatr. Pulmonol.*, 27, 267–272.

LaMontagne, L. L. (1996) Children's preoperative coping and its effects on postoperative anxiety. *Nurs. Res.*, 45, 141–146.

Lan, C. (1996) Cardiorespiratory function, flexibility

and body composition among geriatric Tai Chi Chuan practitioners. *Arch. Phys. Med. Rehabil.*, 77, 612–616.
Lancet (1992) Pacifiers, passive behaviour and pain. *Lancet*, 339, 275–276.
Lancet (1995a). Pathophysiology and management of sickle cell pain crisis. *Lancet*, 346, 1408–1411.
Lancet (1995b) Deglamorising cannabis. *Lancet*, 346, 1241.
Landa, J. F., Kwoka, M. A. and Chapman, G. A. (1980) Effects of suctioning on mucociliary transport. *Chest*, 77, 202.
Landbo, C. (1999) Prognostic value of nutritional status in COPD. *Am. J. Respir. Crit. Care Med.*, 160, 1856–1861.
Lander, J. (1993) TENS for children's procedural pain. *Pain*, 52, 209–216.
Landreneau, R. J., Pigula, F. and Luketich, J. D. (1996) Acute and chronic morbidiy differences between muscle-sparing and standard lateral thoracotomies. *J. Thorac. Cardiovasc. Surg.*, 112, 1346–1351.
Lane, D. J. (1991) Chronic persistent asthma. *Respir. Med.*, 85, 359–363.
Lang, C. (1991) Using relaxation and exercise for people living with HIV/AIDS. *Physiotherapy*, 79, 379–384.
Lannefors, L. (1992) Mucus clearance with three chest physiotherapy regimes in CF. *Eur. Respir. J.*, 5, 748–753.
Lannoo, E. (1998) Cerebral perfusion pressure and intracranial pressure. *Intensive Care Med.*, 24, 236–241.
Laporte, D. M., Chan, D. and Sveistrup, H. (1999) Rising from sitting in elderly people, part 1. *Br. J. Occup. Ther.*, 62, 36–42.
Lareau, S. C. (1999) Dyspnea in patients with COPD. *Heart Lung*, 28, 65–73.
Larsson, B. A. (1999) Pain management in neonates. *Acta Paed.*, 88, 1301–1310.
Lascelles, B. D. X. (1997) Post-operative central hypersensitivity and pain. *Pain*, 73, 461–471.
Laskiwski, S. (1993) The patient with spinal cord injury: modifications of hope and expressions of despair. *Can. J. Rehabil.*, 6, 143–153.
Laursen, S. B. (1998) Bi-level positive airway pressure treatment of OSA. *Respiration*, 65, 114–119.
Lavie, C. J., Milani, R. V. and Squires, R. W. (1992) Exercise and the heart. *Postgrad. Med.*, 91, 130–150.
Lavies, N. (1992) Identification of patient, medical and nursing staff attitudes to postoperative opioid analgesia. *Pain*, 48, 313–319.
Lawler, K. (1995) Entonox: too useful to be limited to childbirth? *Prof. Care Mother Child*, 5, 19–21.
Lawrence, M. (1995) The unconscious experience. *Am. J. Crit. Care*, 4, 227–232.
Lawrence, D. and Kakker, V. V. (1980) Graduated static external compression of the lower limb. *Br. J. Surg.*, 67, 119–121.
Laws, A. K. and McIntyre, R. W. (1969) Chest physiotherapy: a physiological assessment during IPPV in respiratory failure. *Can. Anaesth. Soc. J.*, 16, 487–492.
Lazarus, H. R. and Kostan, J. J. (1969) Psychogenic hyperventilation and death anxiety. *Psychosomatics*, 10, 14–22.
Leach, R. M. (1992) Portable liquid oxygen and exercise ability in severe respiratory disability. *Thorax*, 47, 781–789.
Leach, M. (1998) Signs and symptoms of systemic lupus erythematosus. *Nurs. Times*, 94(13), 50–52.
Leach, R. M. and Bateman, N. T. (1993) Acute oxygen therapy. *Br. J. Hosp. Med.*, 49, 637–644.
Leach, R. M. and Bateman, N. T. (1994) Domiciliary oxygen therapy. *Br. J. Hosp. Med.*, 51, 47–54.
Leach, R. M. and Treacher, D. F. (1998) Oxygen transport 2 – tissue hypoxia. *Br. Med. J.*, 317, 1370–1373.
Leary, R., Fellows, I. and Fletcher, S. (2000) Enteral nutrition part 1. *Care Crit. Ill*, 16, 22–26.
Ledson, M. J. (1998) Cross infection between cystic fibrosis patients colonised with *Burkholderia cepacia*. *Thorax*, 53, 432–436.
Lee, S. T. (1989) Intracranial pressure changes during positioning of patients with severe head injury. *Heart Lung*, 18, 411–414.
Lee, L., Friesen, M. and Lambert, I. R. (1998) Evaluation of dyspnea during physical and speech activities in patients with pulmonary diseases. *Chest*, 113, 625–632.
Lee, B. G., Presneil, J. J. and Cade, J. F. (1999) Tidal volume alteration during mechanical ventilation with heliox. *Anaesth. Intensive Care*, 27, 96.
Lefering, R. (1995) Steroid controversy in sepsis and septic shock. *Crit. Care Med.*, 23, 1924–1303.
Leger, P. (1994) Nasal intermittent positive pressure ventilation. *Chest*, 105, 100–105.
Leino, K., Mildh, L. and Lertola, K. (1999) Time course of changes in breathing pattern in morphine- and oxycodone-induced respiratory depression. *Anaesthesia*, 54, 835–840.

Leisure, G. S., Stone, D. J. and Spiekermann, B. F. (1995) Airway management of the chronically intubated patient. *Respir. Care*, 40, 1279–1286.

Leite, J. J. (1995) Role of pulmonary restriction in patients with ankylosing spondilitis *Chest*, **108**, 157S.

Lenhardt, R. (1997) Assessment of perioperative hypothermia. *Curr. Opin. Anaesthesiol.*, 10, 454–458.

Lennard-Jones, J. E. (1992) *Positive Approach to Nutrition as a Treatment*, Kings Fund, London.

Lennie, T. A. (1999) Anorexia in response to acute illness. *Heart Lung*, 28, 386–401.

Lera, S. (1997) Psychological dimensions of coping in respiratory rehabilitation. *Eur. Respir. J.*, 10, 459s.

Leslie, K. and Sessler, D. I. (1999) Peri-operative hypothermia. *Best Pract. Res. Clin. Anesthesiol.*, **13**, 349–361.

Levin, A. (1999) Humane neonatal care initiative. *Acta Paediatr.*, 88, 353–355.

Levine, P. A. (1997) Management of advanced-stage laryngeal cancer. *Otolaryngol. Clin. North Am.*, 30, 101–113.

Levine, S. (1988) *Who Dies?* Gateway, Bath, p. 134.

Levitzky, M. G. (1995) *Pulmonary Physiology*, McGraw-Hill, New York.

Levy, M. and Bell, L. (1984) General practice audit of asthma in childhood. *Br. Med. J.*, 289, 1115–1118.

Levy, B. D., Kitch, B. and Fanta, C. H. (1998) Medical and ventilatory management of status asthmaticus. *Intensive Care Med.*, 24, 105–117.

Lewandowski, K. (1992) approaches to improve survival in severe ARDS. In: Vincent, J.L. (ed) *Yearbook of Int, Care Emerg. Med*, Springer-Verlag, Berlin.

Lewis, M. I., Sieck, G. C. and Fournier, M. (1986) Effect of nutritional deprivation on diaphragm contractility and muscle fiber size. *J. Appl. Physiol.*, 60, 596–603.

Li, X. Y. (1996) Mechanisms of cigarette smoke induced increased airspace permeability. *Thorax*, **51**, 465–471.

Liebeskind, J. C. and Melzack, R. (1987) The international pain foundation. *Pain*, 30, 1–2.

Light, R. W. (1989) Effects of oral morphine on breathlessness and exercise tolerance in patients with COPD. *Am. Rev. Respir. Dis.*, 139, 126–133.

Light, R. W. (1993) Management of spontaneous pneumothorax. *Am. Rev. Respir. Dis.*, 148, 245–248.

Lightowler, J. V. J. (1996) Local anaesthetic infiltration prior to arterial puncture. *Thorax*, **51**(suppl. 3), A67.

Lim, T. K., Ang, S. M. and Rossing, T. H. (1989) The effects of deep inhalation on maximal expiratory flow during spontaneous asthmatic episodes. *Am. Rev. Respir. Dis.*, 140, 340–343.

Lim, C. M., Koh, Y. and Chin, J. Y. (1999) Respiratory and haemodynamic effects of the prone position. *Eur. Respir. J.*, 13, 163–168.

Lin, K. H. (1999) Abdominal weight and inspiratory resistance. *Arch. Phys. Med. Rehab.*, 80, 741–745.

Lin, V. W. H., Singh, H. and Chitkara, R. K. (1998) Functional magnetic stimulation for restoring cough in patients with tetraplegia. *Arch. Phys. Med. Rehabil.*, 79, 517–522.

Lindeman, C. A. (1971) Nursing intervention with the presurgical patient. *Nurs. Res.*, 20, 319–332.

Lindgren, L. (1997) Pain after laparoscopic cholecystectomy. *Acta Anaesthesiol. Scand.*, **41**, 191–192.

Lindh, V. (1999) Heel lancing in term new-born infants. *Pain*, 80, 143–148.

Lindley, C. M., Dalton, J. A. and Fields, S. M. (1990) Narcotic analgesics. *Cancer News*, 13, 28–38.

Lingnau, W. (1995) Continuous hemofiltration improves multiple organ failure outcome. *Crit. Care Med.*, 23, A106.

Link, W. J., Spaeth, E. E. and Wahle, W. M. (1976) The influence of suction catheter tip design on tracheobronchial trauma and fluid aspiration. *Anesth. Analg.*, 55, 290–297.

Linsenbardt, S. T., Thomas, T. R. and Madsen, R. W. (1992) Effect of breathing techniques on blood pressure response to resistance exercies. *Br. J. Sports Med.*, 26, 97–100.

Linsenmeyer, T. A. (2000) Sexual function and infertility following spinal cord injury. *Phys. Med. Rehab. Clin. N. Am.*, 11, 141–163.

Liposky, J. M. (1994) Ventilatory response to high caloric loads. *Crit. Care Med.*, 22, 796–802.

Lipworth, B. J. (1995) New perspectives on inhaled drug delivery. *Thorax*, 50, 105–110.

Lisboa, C., Villafranca, A. and Leiva, E. (1997) Inspiratory muscle training in CAL: effect on exercise performance. *Eur. Respir. J.*, 18, 537–542.

Little, S. A., Elkholy, M. M. and Chalmers, G. W. (1999) Predictors of nocturnal oxygen desaturation in patients with COPD. *Respir. Med.*, 93, 202–207.

Liu, R. (1994) Effects of background stress and

anxiety on postoperative recovery. *Anaesthesia*, 49, 382–386.
Liu, C. and Douglas, R. M. (1998) Chinese herbal medicines in the treatment of acute respiratory infections. *Med. J. Austr.*, 169, 579–582.
Ljubic, S., Metelko, Z. and Car, N. (1998) Reduction of diffusion capacity for carbon monoxide in diabetic patients. *Chest*, 114, 1033–1035.
Lloyd, G. (1987) Heat and moisture exchangers. *Care Crit. Ill*, 3, 110–113.
Lloyd, S. (1998) Education and adherence to therapy in a clinical setting. *Eur. Respir. Rev.*, 58, 333–337.
Lock, S. H. (1992) Comparison of liquid and gaseous oxygen for domiciliary portable use. *Thorax*, 47, 98–100.
Locke, C. R., Talley, N. J. and Fett, S. L. (1999) Risk factors associated with symptoms of GOR. *Am. J. Med.*, 106, 642–649.
Logemann, J. A. (1986) Treatment for aspiration related to dysphagia. *Dysphagia*, 1, 34–38.
Lomholt, N. (1982a) The new NL tracheal suction catheter. *Acta Anaesth. Scand.*, 26, 4–8.
Lomholt, N. (1982b) Design and function of tracheal suction catheters. *Acta Anaesth. Scand.*, 26, 1–3.
Long, T. (1995) The effect of positioning prematurely born infants in the neonatal ICU. *Phys. Occup. Ther. Pediatr.*, 15, 17–31.
Lowell, J. A. (1990) Postoperative fluid overload. *Crit. Care Med.*, 18, 728–733.
Lowen, A. (1991) *Breathing, Movement and Feeling*, International Institute for Bioenergetic Analysis, New York.
Lubel, D. (1989) Falls and the older person. *Care Elderly*, 1, 66–67.
Lucas, A., Morley, R. and Cole, T. J. (1998) Randomised trial of early diet in preterm babies and later IQ. *Br. Med. J.*, 17, 1481–1487.
Luce, P. J. (1996) Asthma in the elderly. *Br. J. Hosp. Med.*, 55, 118–124.
Luce, J. M., Pierson, D. J. and Tyler, M. T. (1993) *Intensive Respiratory Care*, W. B. Saunders, Philadelphia, PA.
Ludington, S. M. (1990) Energy conservation during skin-to-skin contact between infants and mothers. *Heart Lung*, 19, 445–451.
Ludwig, C. (1984) When you learn that you are a myasthenic. *Remedial Ther.*, 24 February, 8.
Ludwigs, U. (1998) Pulmonary epithelial permeability and gas exchange. *Chest*, 113, 459–466.
Lum, L. C. (1981) Hyperventilation and anxiety state. *J. Roy. Soc. Med.*, 74, 1–4.
Lumb, A. B. (2000) *Nunn's Applied Respiratory Physiology*, 5th edn. Butterworth-Heinemann, London.
Lynch, J. J. (1978) The simple art of touching. *Nursing*, 8, 32–36.
Ma, Y., Li. J. and Lui, Y. (1997) Short wave diathermy for small spontaneous pneumothorax. *Thorax*, 52, 561–562.
McCaffery, M. and Ferrell, B. R. (1992) Does the gender gap affect your pain control decisions? *Nursing*, 92, 49–51.
McCallum, T. J. (2000) Fertility in men with cystic fibrosis. *Chest*, 118, 1059–1062
McCarren, B. (1992) Dynamic pulmonary hyperinflation. *Austr. Physiother. J.*, 38, 175–179.
McConnell, A. K., Caine, M. P. and Donovan, K. J. (1998) Inspiratory muscle training improves lung function and reduces dyspnoea in mild/moderate asthmatics. *Clin. Sci.*, 95, 4.
McConnochie, K. and Chatham, K. (1991) Double blind controlled study of inspiratory muscle training. *Thorax*, 46, 755P.
McCool, F. D. and Tzelepis, G. E. (1995) Inspiratory muscle training in the patient with neuromuscular disease. *Phys. Ther.*, 75, 1006–1014.
McCowan, C., Neville, R. G. and Thomas, G. E. (1998) Effect of asthma and its treatment on growth. *Br. Med. J.*, 316, 668–772.
McCullagh, P. (1996) Determining foetal sentience. *Hosp. Update*, 22, 5–6.
McDonald, C. F. (1995) Exertional oxygen of limited benefit in patients with COPD and mild hypoxaemia. *Am. J. Respir. Crit. Care Med.*, 152, 1616–1619.
McEvoy, R. D., Davies, J. H. and Hedenstierna, G. (1982) Lung mucociliary transport during high-frequency ventilation. *Am. Rev. Respir. Dis.*, 126, 452–456.
McFadden, E. R. (1995) Asthma. *Lancet*, 345, 1215–1260.
McFadden, E. R. and Warren, E. L. (1997) Observations on asthma mortality. *Ann. Intern. Med.*, 127, 142–147.
McGrath, P. J. and Frager, G. (1996) Psychological barriers to optimal pain management in infants and children. *Clin. J. Pain*, 12, 135–41.
McIlwaine, M., Davidson, A. G. F. and Wong, L. T. K. (1991) The effect of chest physiotherapy by postural drainage and autogenic drainage on oxygen saturation in CF. *Pediatr. Pulmonol.*, 6, 291.

McIlwaine, P. M., Wong, L. T. K. and Peacock, D. (1996) Long term comparative trial of conventional postural drainage and percussion versus PEP in the treatment of CF. *Israel J. Med. Sci.*, 32(suppl.), S275.

McIlwaine, P. M., Wong, L. T. W. and Peacock, D. (1997) Flutter versus PEP. *Pediatr. Pulmonol.*, **14**(suppl.), 299.

McIntosh, A. (1989) Sleep deprivation in critically ill patients. *Nursing*, **3**, 44–45.

MacIntyre, N. R. (1995) Psychological factors in weaning from mechanical ventilation. *Respir. Care*, 40, 277–281.

MacIntyre, N. R. (1996) Minimizing alveolar stretch injury during mechanical ventilation. *Respir. Care*, **41**, 318–326.

MacIntyre, N. R., Helms, M., Wunderink, R. and Schmidt, G. (1999) Automated rotational therapy for the prevention of respiratory complications during mechanical ventilation. *Respir. Care*, 44, 1447–1451.

MacKay, L. E. (1992) Early intervention in severe head injury. *Arch. Phys. Med. Rehabil.*, 73, 635–641.

MacKellaig, J. M. (1990) A review of the psychological effects of intensive care on the isolated patient and family. *Care Crit. Ill*, 6, 100–102.

McKenzie, D. K. (1994) Dynamic changes in the zone of apposition and diaphragm length during maximal respiratory efforts. *Thorax*, 49, 634–638.

McLaren, P. (1991) The right to know. *Br. Med. J.*, 303, 937–938.

Macklem, P. T. and Eidelman, D. (1990) Reexamination of the elastic properties of emphysematous lungs. *Respiration*, 57, 187–192.

McMichan, J. C., Michel, L. and Westbrook, P. R. (1980) Pulmonary dysfunction following traumatic quadriplegia. *J. A. M. A.*, 243, 528–529.

McNamara, R. M. (1992) Utility of the peak expiratory flow rate in the differentiation of acute dyspnea. *Chest*, **101**, 129–132.

McNamara, F. (1999) Obstructive sleep apnea in infants and its management with nasal CPAP. *Chest*, **116**, 10–16.

Macnaughton, P. D. (1995) Posture and lung function in health and disease. *Br. J. Intensive Care*, **1**, 133–137.

MacNee, W. (1995) Oxidants/antioxidants in idiopathic pulmonary fibrosis. *Thorax*, 50, 553–558.

MacNeish, C. F. (1997) A comparison of pulmonary availability between Ventolin nebules and respiratory solution. *Chest*, **111**, 204–208.

McNicholas, W. T. (1997) Impact of sleep on respiratory failure. *Eur. Respir. J.*, 10, 920–933.

McQuellon, R. P. and Hurt, G. J. (1997) The psychosocial impact of laryngeal cancer. *Otolaryngol. Clin. North Am.*, 30, 231–241.

Mador, M. J. (1991) Respiratory muscle fatigue and breathing pattern. *Chest*, 100, 1430–1435.

Mador, M. J., Kufel, T. J. and Pineda, L. A. (2000) Diaphragmatic fatigue and high-intensity exercise in patients with COPD. *Am. J. Respir. Crit. Care Med.*, **161**, 118–123.

Magarian, G. J. (1982) Hyperventilation syndromes. *Medicine*, **61**, 219–235.

Magnussen, H. and Holz, O. (1999) Monitoring airway inflammation in asthma by induced sputum. *Eur. Respir. J.*, **13**, 5–7.

Maguire, P. (1998) Surgery and loss of body parts. *Br. Med. J.*, 316, 1086–1088.

Mahadeva, R., Webb, K. and Westerbeek, R. C. (1998) Clinical outcome in relation to care in centres specialising in CF. *Br. Med. J.*, 316, 1171–1175.

Mahler, D. A. (1990) *Dyspnea*, Futura Publishing, New York.

Mahler, D. A. (1995) Changes in dyspnea, health status and lung function in chronic airway disease. *Am. J. Respir. Crit. Care Med.*, 151, 61–65.

Mahon, J. L. (1999) Theophylline for irreversible chronic airflow limitation. *Chest*, **115**, 38–48.

Majid, A. A. (1992) Pain control after thoracotomy. *Chest*, **101**, 981–984.

Mak, V. H. F., Bugler, J. R. and Roberts, C. M. (1993) Effect of arterial desaturation on 6-minute walk, effort and breathlessness in patients with airflow limitation. *Thorax*, 48, 33–38.

Mak, V. H. F., Harvey, C. and Treacher, D. F. (1995) Non-invasive assisted ventilation for respiratory failure for acute severe asthma. *ACPRC J.*, 26, 22–23.

Make, B. M. (1994) Collaborative self-management strategies for patients with respiratory disease. *Respir. Care*, 39, 566–577.

Mal, H., Rullon, I. and Mellot, F. (1999) Immediate and long-term results of bronchial artery embolization for life-threatening hemoptysis. *Chest*, **115**, 996–1001.

Malfroot, A. and Dab, I. (1991) New insights on gastro-oesophageal reflux in CF. *Arch. Dis. Child.*, **66**, 1339–1345.

Malmberg, L. P., Tamminen, K. and Sovijärvi, A. R. A. (2000) Orthostatic increase of respiratory gas exchange in hyperventilation syndrome. *Thorax*, 55, 295–301.

Man, P., Verhoeven, B. A. N. and Verbrugh, H. A. (2000) An antibiotic policy to prevent emergence of resistant bacilli. *Lancet*, 355, 973–978.

Mange, K., Matsuura, D., Cizman, B. and Soto, H. (1997) Language guiding therapy: the case of dehydration versus volume depletion. *Ann. Intern. Med.*, 127, 848–853.

Mann, N. P. (1986) Effect of night and day on preterm infants. *Br. Med. J.*, 293, 1265–1267.

Mann, C., Pouzeratte, Y. and Boccara, G. (2000) Comparison of intravenous or epidural patient-controlled analgesia in the elderly. *Anesthesiology*, 92, 433–441.

Manne, S. L., Jacobsen, P. B. and Redd, W. H. (1992) Assessment of acute pediatric pain. *Pain*, 48, 45–52.

Manning, H. L. (1995) Pathophysiology of dyspnoea. *N. Engl. J. Med.*, 333, 1547–1553.

Manning, H. L. (2000) Bronchodilator therapy in COPD. *Curr. Opin. Pulm. Med.*, 6, 99–103.

Mansour, V. M. (1998) Panic disorder: coronary spasm as a basis for cardiac risk. *Med. J. Austr.*, 168, 390–392.

Manthous, C. A. (1994) Administration of therapeutic aerosols to mechanically ventilated patients. *Chest*, 106, 560–571.

Manthous, C. A., Hall, J. B. and Melmed, A. (1995) Heliox improves pulsus paradoxus and peak expiratory flow in nonintubated patients with severe asthma. *Am. J. Respir. Crit. Care Med.*, 151, 310–314.

Manzano, J. L. (1993) Verbal communication of ventilator-dependent patients. *Crit. Care Med.*, 21, 512–517.

Marcano, B. (2000) Cephalad movement of tracheal tubes due to prone positioning of pediatric patients with ARDS. *Chest*, 118, 179S.

Marcus, C. L. (1992) Supplemental oxygen and exercise performance in patients with CF. *Chest*, 101, 52–57.

Marcy, T. W. (1994) Respiratory distress in the ventilated patient. *Clin. Chest Med.*, 15, 55–74.

Marik, P. E. (1996a) The cuff-leak test as a predictor of postextubation stridor. *Respir. Care*, 41, 509–511.

Marik, P. E. (1996b) The refeeding syndrome in ICU patients. *Crit. Care Med.*, 24, A84.

Marik, P., Hogan, J. and Krikorian, J. (1999) A comparison of bronchodilator therapy delivered by nebulization and metered-dose inhaler in mechanically ventilated patients. *Chest*, 115, 1653–1657.

Marinho, P. E. M., Brito, E. C. S. and Gusmao, A. L. (1999) Comparative study of the change in inspiratory and expiratory muscle strength between young and elderly subjects by the use of volumetric incentive spirometer. *WCPT Proceedings*, p. 385.

Marini, J. J. (1999) PEEP in the prone position. *Crit. Care Med.*, 27, 1–2.

Marini, J. J., Tyler, M. L. and Hudson, L. D. (1984) Influence of head-dependent positions on lung volume and oxygen saturation in chronic air-flow limitation. *Am. Rev. Respir. Dis.*, 129, 101–105.

Marino, J. (1995) Neuromuscular blockade in the ICU. *Crit. Care Med.*, 23, A240.

Marino, W. D., Nyarko, W. C. and Kiyici, N. (1995) Helium-oxygen mixtures in adult patients with COPD and CO_2 retention. *Chest*, 108, 107S.

Marsh, B. (1986) A second chance. *Br. Med. J.*, 292, 675–676.

Marshall, S. M. (1996) The perioperative management of diabetes. *Care Crit. Ill*, 12, 64–69.

Marshall, R. and Holden, W. S. (1963) Changes in calibre of the airways in man. *Thorax*, 18, 54–58.

Martin, M. and Shaw, N. J. (1997) Feeding problems in infants and young children with chronic lung disease. *J. Hum. Nutr. Diet.*, 10, 272–275.

Martin, C. J., Ripley, H. and Reynolds, J. (1976) Chest physiotherapy and the distribution of ventilation. *Chest*, 69, 174–178.

Martinez, F., Epstein, S. and Roe, J. (1991) Determinants of respiratory muscle strength in CAO. *Am. Rev. Respir. Dis.*, 143, A161.

Martinez-Moragon, E. (1998) Malignant pleural effusion. *Respiration*, 65, 108–113.

Maskell, N., Wells, S. and Meecham Jones, D. J. (1999) The value of an intensive physiotherapy course in patients with hyperventilation. *ALA/ATS Conference*, California (abstract), p. 832.

Mason, M. F. (1993) *Speech Pathology for Tracheostomized and Ventilator Dependent Patients*, Voicing, California.

Massard, G. and Wihlm, J.-M. (1998) Postoperative atelectasis. *Chest Surg. Clin. North Am.*, 8, 503–528.

Mastro, T. D., Fields, B. S. and Breiman, R. F. (1991) Nosocomial legionnaires' disease and use of

medication nebulizers. *J. Infect. Dis.*, 163, 667–671.
Mathewson, H. S. (1993) Combined drug therapy in asthma. *Respir. Care*, 38, 1340.
Mathewson, H. S. (1997) Post-myocardial infarction drugs. *Respir. Care*, 42, 517–519.
Mathur, R. (1999) Cerebral bioenergetics in stable COPD. *Am. J. Respir. Crit. Care Med.*, 160, 1994–1999.
Mattison, L. E., Coppage, L. and Alderman, D. F. (1997) Pleural effusions in the medical ICU. *Chest*, 111, 1018–1023.
Matuszczak, Y. (1998) Diaphragmatic function is markedly altered in cerulein-induced pancreatitis. *Crit. Care Med.*, 26, 1327–1331.
Maurer, J. R., Frost, A. E. and Estenne, M. (1998) International guidelines for the selection of lung transplant candidates. *Heart Lung*, 27, 223–229.
Maxam-Moore, V. A. (1996) The development of an early extubation algorithm for patients after cardiac surgery. *Heart Lung*, 25, 61–68.
Mayberry, J. C. (1997) The fractured rib in chest wall trauma. *Chest Surg. Clin. North Am.*, 7, 239–256.
Mayor, A. H. (1997) Magnetic resonance imaging of the heart during CPAP via a facemask in normal subjects [abstract]. *Anesth. Analg.*, 84, 104.
Medical Monitor (1992) Maternal smoking increases squint risk in children. *Med. Monitor*, 27 November, 33.
Meduri, G. U. (1990) Ventilator-associated pneumonia. *Chest*, 97, 1208–1219.
Meduri, G. U. (1996) Noninvasive positive pressure ventilation via face mask. *Chest*, 109, 179–193.
Mehta, A. C. (1999) Airway stents. *Clin. Chest Med.*, 20, 139–152.
Mejia, R. (1999) Target dyspnea ratings predict expected oxygen consumption as well as target heart rate values. *Am. J. Respir. Crit. Care Med.*, 159, 1485–1489.
Melamed, S. (1999) Emotional reactivity and debilitating beliefs during hospitalization predict future adjustment to first MI in men. *Scand. J. Rehabil. Med.*, 31, 23–30.
Melendez, J. A. (1992) Postthoracotomy respiratory muscle mechanics during incentive spirometry. *Chest*, 101, 432–436.
Mellins, R. B., Zimmerman, B. and Clark, N. M. (1992) Patient compliance. *Am. Rev. Respir. Dis.*, 146, 1376–1377.
Menier, R. J. (1994) Benefits of a multidisciplinary pulmonary rehabilitation program [letter]. *Chest*, 105, 640–641.
Menkes, H. and Britt, J. (1980) Rationale for physical therapy. *Am. Rev. Respir. Dis.*, 122, 127–132.
Merveille, O. C., Childers, M. K. and Kreimid, M. M. (1999) Weaning from mechanical ventilation in a general rehabilitation center. *Am. J. Phys. Med. Rehabil.*, 78, 85–86.
Meythaler, J. M. (1997) Rehabilitation of Guillain–Barré syndrome. *Arch. Phys. Med. Rehabil.*, 78, 872–879.
Middleton, P. G., Geddes, D. M. and Alton, E. W. (1993) Effect of amiloride and saline on nasal mucociliary clearance and potential difference in cystic fibrosis and normal subjects. *Thorax*, 48, 812–816.
Mier, A., Laroche, C. and Agnew, J. E. (1990) Tracheobronchial clearance in patients with bilateral diaphragmatic weakness. *Am. Rev. Respir. Dis.*, 142, 545–546.
Milgrom, H. and Bender, B. (1997) Nonadherence to asthma treatment and failure of therapy. *Curr. Opin. Pediatr.*, 9, 590–595.
Milla, C. E. (1998) Risk of death in CF patients with severely compromised lung function. *Chest*, 133, 1230–1234.
Millard, P. H. (1983) Depression in old age. *Br. Med. J.*, 287, 375.
Miller, S. (1996) Living with a disabled sibling. *Paed. Nurs.*, 8, 21–24.
Miller, M. R. and Ouanjer, P. H. (1994) Peak flow meters. *Br. Med. J.*, 308, 548–549.
Miller, K. M., Perry, P. A. (1990) Relaxation techniques and postoperative pain in patients undergoing cardiac surgery. *Heart Lung*, 19, 136–146.
Miller, R. F. and Semple, S. J. G. (1991) Continuous positive airways pressure for pneumocystis carinii pneumonia. *Respir. Med.*, 85, 133–138.
Miller, S., Hall, D. O. and Clayton, C. B. (1995) Chest physiotherapy in cystic fibrosis: a comparative study of autogenic drainage and the active of breathing with postural drainage. *Thorax*, 50, 165–169.
Miller, T. P. and Barbers, R. G. (1999) Management of the severe asthmatic. *Curr. Opin. Pulmonary Med.*, 5, 58–62.
Mills, M. E. and Sullivan, K. (2000) Patients with operable oesophageal cancer: their experience of information-giving in a regional thoracic unit. *J. Clin. Nurs.*, 9, 236–246.

Milne, A. (1998) *Smoking: The Inside Story*, Woodside, Stafford.
Milner, A. D. (1997) The role of corticosteroids in bronchiolitis and croup. *Thorax*, 52, 595–597.
Mirski, M. A. (1995) Sedation for the critically ill neurologic patient. *Intensive Care Med.*, 23, 2038–2053.
Mishoe, S. C. (1995) Quiet – hospital zone. *Respir. Care*, 40, 1116–1117.
Misset, B. (1991) Heat and moisture exchanger vs. heated humidifier during long-term mechanical ventilation. *Chest*, 100, 160–163.
Mitchell, A. (1998) Steroids and depression. *Br. Med. J.*, 316, 244–245.
Mitchell, P. H. and Mauss, N. K. (1978) Relationship of patient-nurse activity to ICP variations. *Nurs. Res.*, 27, 4–10.
Mitchell, P. H., Ozuna, J. and Lipe, H. P.(1981) Moving the patient in bed: effects on ICP. *Nurs. Res.*, 30, 212–218.
Miyahara, Y. (1995) Monitoring of nocturnal oxygen desaturation. *Respiration*, 62, 348–352.
Mogan, J., Wells, N. and Robertson, E. (1985) Effects of preoperative teaching on postoperative pain. *Int. J. Nurs. Stud.*, 22, 267–285.
Mohsenifar, Z., Horak, D. and Brown, H. V. (1983) Sensitive indices of improvement in a pulmonary rehabilitation program. *Chest*, 83, 189–192.
Mohsenin, V. (1994) Daytime oxygen saturation does not predict nocturnal desaturation in patients with COPD. *Arch. Phys. Med. Rehabil.*, 75, 285–289.
Molen, B. (1995) Dyspnoea: a study of measurement instruments. *J. Adv. Nurs.*, 22, 948–956.
Moller, J. T. (1998) Long-term postoperative cognitive dysfunction in the elderly. *Lancet*, 351, 857–861.
Molnar, Z. and Shearer, E. (1998) Venovenous haemofiltration in the treatment of sepsis and multiple organ dysfunction syndrome. *Br. J. Intensive Care*, 8, 12–18.
Monga, U., Tan, G. and Ostermann, J. (1997) Sexuality in head and neck cancer patients. *Arch. Phys. Med. Rehabil.*, 78, 298–304.
Moore, T. (1991) *Cry of the Damaged Man*, Picador, Sydney.
Moore, F. D. (1994) The effect of length of stay on complications. *Ann. Surg.*, 220, 738–739.
Morgan, A. D., Peck, D. F. and Buchanan, D. R. (1983) Effect of attitudes and beliefs on exercise tolerance in chronic bronchitis. *Br. Med. J.*, 286, 171–173.
Morgan, M. D. L. (1992) Detection and quantification of pulmonary emphysema by computed tomography. *Thorax*, 47, 1001–1004.
Morice, A. (1996) Inhaled therapy for asthma. *Update*, 52, 592–596.
Morice, A. H. (1998) Asthma. *Update*, 56, 44–49.
Moriña, P. (1997) Effects of nebulized salbutamol on respiratory mechanics in ARDS. *Intensive Care Med.*, 23, 58–64.
Morley, C. (1999) Continuous distending pressure. *Arch. Dis. Child. Fetal Neonat. Ed.*, 81, F52–F56.
Morrell, N. W. (1994) Collateral ventilation and gas exchange in emphysema. *Am. J. Respir. Crit. Care Med.*, 150, 635–641.
Morris, C. K. (1991) Cardiovascular benefits to increased activity. *Herz*, 16, 222–236.
Morris, J. (1999) The effects of immobilization on the musculoskeletal system. *Br. J. Ther. Rehabil.*, 6, 390–393.
Morrison, S. A. (1988) Biofeedback to facilitate unassisted ventilation in individuals with high-level quadriplegia. *Phys. Ther.*, 68, 1378–1380.
Morrison, R. S., Ahronheim, J. C. and Morrison, G. R. (1998) Pain and discomfort associated with common hospital procedures. *J. Pain Symptom Manage.*, 15, 91–101.
Morrison, R. S. and Siu, A. L. (2000) A comparison of pain and its treatment in advanced demential and cognitively intact patients with hip fracture. *J. Pain Symptom. Man.* 19, 240–248.
Mortensen, J., Falk, M. and Groth, S. (1991) The effects of postural drainage and PEP in cystic fibrosis. *Chest*, 100, 1350–1357.
Moseley, A. M. (1997) The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. *Phys. Ther.*, 77, 240–249.
Moser, K. M. (1990) Venous thromboembolism. *Am. Rev. Respir. Dis.*, 141, 235–249.
Moser, D. K. and Dracup, K. (1996) Is anxiety early after myocardial infarction associated with subsequent ischemic and arrhythmic events? *Psychosomatic Med.*, 58, 395–401.
Moss, E. and McDowall, D. G. (1979) ICP increases with 50% nitrous oxide in severe head injuries. *Br. J. Anaesth.*, 51, 757–760.
Mowatt-Larssen, C. A. (1993) Specialized nutritional support in respiratory disease. *Clin. Pharm.*, 12, 278–292.
Moyer-Mileur, L. J. (2000) Daily physical activity

program increases bone mineralization and growth in preterm very low birth weight infants. *Pediatrics*, 106, 1088–1092.

Moniche, S., Bülow, S. and Hesselfeldt, P. (1995) Convalescence and hospital stay after colonic surgery with balanced analgesia, enforced oral feeding and mobilisation. *Eur. J. Surg.*, 161, 283–288.

Muers, M. (1993) Understanding breathlessness. *Lancet*, 342, 1190–1191.

Muir, J.-F. (1993) Home mechanical ventilation. *Thorax*, 48, 1264–1273.

Muir, J.-F., Girault, C. and Cardinaud, J.-P. (1994) Survival and long-term follow-up of tracheostomized patients with COPD treated by home mechanical ventilation. *Chest*, 106, 201–209.

Muldoon, M. F. (1998) What are quality of life measurements measuring? *Br. Med. J.*, 316, 542–545.

Mulholland, C., Lennon, S. and Graham, R. (1994) Does prone positioning improve oxygen saturation in a patient with CF? *Physiother. Theor. Pract.*, 10, 223–233.

Mulley, G. (1993) Nursing elderly patients out of bed. *Br. Med. J.*, 307, 80.

Mulloy, E. (1996) Ventilation and gas exchange during sleep and exercise in severe COPD. *Chest*, 109, 387–394.

Munday, I. T., Desai, P. M. and Marshall, C. A. (1993) The effectiveness of preoperative advice to stop smoking. *Anaesthesia*, 48, 816–818.

Munro, N. C. (1992) Radiological evidence of progression of bronchiectasis. *Respir. Med.*, 86, 397–401.

Munro, N. C., Currie, D. C. and Garbett, N. D. (1989) Chest pain in chronic sputum production. *Respir. Med.*, 83, 339–341.

Murch, S. H. (1995) Cellular mediators of lung damage. *Br. J. Intensive Care*, 5, 27–30.

Murphy, F. M. (1991) The high-risk infant. *Physiotherapy*, 77, 367–370.

Murphy, M. S. (1997) Sedation for invasive procedures in paediatrics. *Arch. Dis. Child.*, 77, 281–286.

Murray-Parkes, C. (1998) The dying adult. *Br. Med. J.*, 316, 1313–1315.

Muscolino, G. (1997) Pancoast tumours. *Thorax*, 52, 284–286.

Mutatkar, S. S. (1999) Comparison between effect of diaphragmatic breathing exercise with purse lip expiration with and without an end-inspiratory hold. *WCPT Proceedings*, p. 36.

Myles, W. S. and Maclean, D. (1986) A comparison of response and production protocols for assessing perceived exertion. *Eur. J. Appl. Physiol.*, 55, 585–587.

Nagler, A. (1996) Reduction in pulmonary fibrosis in vivo by halofuginone. *Am. J. Respir. Crit. Care Med.*, 154, 1082–1086.

Nance, P. W., Schryvers, O. and Leslie, W. (1999) Intravenous pamidronate attenuates hone density loss after acute spinal cord injury. *Arch. Phys. Med. Rehabil.*, 80, 243–251.

Napolitano, L. M. (1999) Capnography in critical care. *Crit. Care Med.*, 27, 862–863.

Nathan, S. D. (1995) Bronchiolitis obliterans in single-lung transplant recipients. *Chest*, 107, 967–972.

Nava, S. (1998a) Noninvasive mechanical ventilation in the weaning of patients with respiratory failure. *Ann. Intern. Med.*, 128, 721–728.

Nava, S. (1998b) Rehabilitation of patients admitted to a respiratory intensive care unit. *Arch. Phys. Med. Rehabil.*, 79, 849–854.

Neilly, J. B. (1992) Effects of selective sleep deprivation on ventilation during recovery sleep in normal humans. *J. Appl. Physiol.*, 72, 100–109.

Nelson, H. S. (1995) β_2-adrenergic bronchodilators. *N. Engl. J. Med.*, 333, 499–504.

Nelson, S. P. (1998) One-year follow-up of symptoms of gastroesophageal reflux during infancy. *Pediatrics*, 102, 67–74.

Nelson, F. V., Zimmerman, L. and Barnason, S. (1998) The relationship and influence of anxiety on postoperative pain in the CABG patient. *J. Pain Symptom Manage.*, 15, 102–109.

Nelson-Piercy, A. (1996) Asthma in pregnancy. *Br. J. Hosp. Med.*, 55, 115–117.

Neville, S. and French, S. (1991) Clinical education. *Physiotherapy*, 77, 351–354.

Neville, E., Gribbin, H. and Harrison, B. D. W. (1991) Acute severe asthma. *Respir. Med.*, 85, 463–474.

Newell, S. Z. (1989) Inspiratory and skeletal muscle strength and endurance and diaphragmatic activation in patients with CAL. *Thorax*, 44, 903–912.

Newman, K. B., Lynch, D. A. and Newman, L. S. (1994) Quantitative computed tomography detects air trapping due to asthma. *Chest*, 106, 105–109.

Ng, G. Y. and Stokes, M. J. (1992) EMG recordings

of respiratory muscles during unilateral and bilateral chest expansion. *Austr. Physiol. J.*, 38, 203–208.
Nguyen, T. T. (1996) Current treatment of severely burned patients. *Ann. Surg.*, 223, 14–25.
Nicholas, T. E. (1997) Surfactant replacement therapy in ARDS. *Thorax*, 52, 195–197.
Nicholas, K. J., Dhouieb, M. O. and Marshall, T. G. (1999) An evaluation of chest physiotherapy in the management of acute bronchiolitis. *Physiotherapy*, 85, 669–674.
Nicholls, D. (1996) The crisis in undergraduate respiratory care. *ACPRC J.*, 29, 51.
Nicholson, C. (1993) Are you ready for video thoracoscopy? *Am. J. Nurs.*, 93, 54–57.
Nield, M. A. (1999) Inspiratory muscle training protocol using a pressure threshold device. *Arch. Phys. Med. Rehabil.*, 80, 100–103.
Nield, M. (2000) Dyspnea self-management in African Americans with chronic lung disease. *Heart Lung*, 29, 50–55.
Nikander, K. (2000) Evaluation of pulsed and breath-synchronized nebulization. *Pediatr. Pulmonol.*, 29, 120–126.
Nikolajsen, L. (1997) The influence of preamputation pain on postamputation stump and phantom pain. *Pain*, 72, 393–405.
Nilsson, E. (1997) Intrathecal morphine for post-sternotomy pain. *Acta Anaesthesiol. Scand.*, 41, 549–556.
Ninane, V. (1997) Intrinsic PEEP. *Eur. Respir. J.*, 10, 516–518.
Ninane, V., Rypens, F. and Yernault, J.-C. (1992) Abdominal muscle use during breathing in patients with CAO. *Am. Rev. Respir. Dis.*, 146, 16–21.
Ninane, V., Yernault, J.-C. and deTroyer, A. (1993) Intrinsic PEEP in patients with COPD. *Am. Rev. Respir. Dis.*, 148, 1037–1042.
Nishimura, K. (2000) Clinical course and prognosis of patients with COPD. *Curr. Opin. Pulm. Med.*, 6, 127–132.
Nolan, J. P. (1996) Care for trauma patients in the ICU. *Curr. Anaesth. Crit. Care*, 7, 139–145.
Noppen, M., Dhondt, E. and Mahler, T. (1994) Successful management of recurrent pneumothorax in CF by localized apical thorascopic talc poudrage. *Chest*, 106, 262–264.
Northridge, D. (1996) Frusemide or nitrates for acute heart failure? *Lancet*, 347, 667–668.
Norton, B. (1996) A randomised prospective comparison of percutaneous endoscopic gastrostomy and nasogastric tube feeding after acute dysphagic stroke. *Br. Med. J.*, 312, 13–16.
Nosworthy, J., Barter, C. and Thomas, S. (1992) An evaluation of three elements of pulmonary rehabilitation. *Austr. Physiother.*, 38, 189–193.
Noureddine, S. N. (1996) Sleep apnea: a challenge in critical care. *Heart Lung*, 25, 37–42.
Novak, R. A., Shumaker, L. and Snyder, J. V. (1987) Do periodic hyperinflations improve gas exchange in patients with hypoxemic respiratory failure? *Crit. Care Med.*, 15, 1081–1085.
Novitch, R. S. (1995) Pulmonary rehabilitation in patients with interstitial lung disease. *Am. J. Respir. Crit. Care Med.*, 151, A684.
Ntoumenopoulos, F. and Greenwood, D. (1996) Effects of cardiothoracic physiotherapy on intrapulmonary shunt in abdominal surgical patients. *Austr. J. Physiother.*, 42, 297–303.
Nunn, J. F., Coleman, A. J. and Sachithanandan, T. (1965) Hypoxaemia and atelectasis produced by forced expiration. *Br. J. Anaesth.*, 37, 3–11.
Nuorti, J. P. (2000) Cigarette smoking and invasive pneumococcal disease. *N. Engl. J. Med.*, 342, 681–689.
Nursing Times (1981) Who am I? Where am I? Why do I hurt so much? *Nurs. Times*, 77(15), 633–635.
Nuttal, G. A. (1998) No difference exists in the alteration of circadian rhythm between patients with and without ICU psychosis. *Crit. Care Med.*, 26, 1351–1355.
Nygren, J., Thorell, A. and Jacobsson, H. (1996) Preoperative gastric emptying. *Ann. Surg.*, 222, 728–734.
Nyren, S. (1999) Pulmonary perfusion is more uniform in the prone than in the supine position. *J. Appl. Physiol.*, 86, 1135–1141.
Oakley, M. J., Wheelwright, E. F. and James, P. J. (1998) Pneumatic compression boots for prophylaxis against DVT: beware occult arterial disease. *Br. Med. J.*, 316, 454–455.
Oberklaid, F. (1998) Twenty-four hour access to health information and advice. *Med. J. Austr.*, 169, 125–126.
Oberwaldner, B. (2000) Physiotherapy for airway clearance in paediatrics. *Eur. Respir. J.*, 15, 196–204.
O'Brian, G. M., Furukawa, S. and Kuzma, A. M. (1999) Improvements in lung function, exercise and quality of life in hypercapnic COPD patients after lung volume reduction surgery. *Chest*, 115, 75–84.

Ochsenreither, J. M. (1997) Epidural analgesia in infants. *Neonat. Netw.*, **16**, 79–84.
O'Connor, B. J. (1997) Premenstrual asthma: still poorly understood. *Thorax*, **52**, 591–592.
Odderson, I. R. (1995) Swallow management in patients on an acute stroke pathway. *Arch. Phys. Med. Rehabil.*, **76**, 1130–1133.
Odell, M. (1997) Suicide: a study of grief in intensive care. *Care Crit. Ill*, **13**, 238–240.
O'Donnell, D. E. (1994) Breathlessness in patients with chronic airflow limitation. *Chest*, **106**, 904–912.
O'Donnell, D. E. (1995) The impact of exercise reconditioning on breathlessness in severe CAL. *Am. J. Respir. Crit. Care Med.*, **152**, 2005–2013.
O'Donnell, D. E. and Webb, K. A. (1992) Breathlessness in patients with severe chronic airflow limitation. *Chest*, **102**, 824–831.
O'Driscoll, B. R. (1997) Nebulisers for COPD. *Thorax*, **52**(suppl. 2), S49–S52.
Oh, T. E. (1997) *Intensive Care Manual*, 4th edn, Butterworth-Heinemann, London.
Ohrbach, R., Patterson, D. R. and Carrougher, G. (1998) Hypnosis after an adverse response to opioids in an ICU burn patient. *Clin. J. Pain*, **14**, 167–175.
Ojanen, M. (1993) Psychosocial changes in patients participating in a COPD rehabilitation program. *Respiration*, **60**, 96–102.
O'Kroy, J. A. (1993) Effects of flow and resistive training on respiratory muscle endurance and strength. *Respiration*, **60**, 279–283.
Olds, D. L. (1994) Intellectual impairment in children of women who smoke cigarettes during pregnancy. *Pediatrics*, **93**, 221–227.
O'Leary, M. J. and Coakley, J. H. (1996) Weakness and wasting in the critically ill patient. *Curr. Anaesth. Crit. Care*, **7**, 81–86.
Olivier, F. L. (1998) Suggested guidelines for the use of exercise with adults in acute care settings. *Physiother. Can.*, **50**, 127–135.
Olsen, M. F., Hahn, I. and Nordgren, S. (1997) Randomised controlled trial of prophylactic chest physiotherapy in major abdominal surgery. *Br. J. Surg.*, **84**, 1535–1538.
Olséni, L. (1994) Chest physiotherapy in COPD: FET combined with either postural drainage or PEP. *Respir. Med.*, **88**, 435–440.
Onuoha, A. R. A. (1994) Effective clinical teaching from the perspectives of students, supervisors and teachers. *Physiotherapy*, **80**, 208–214.
Orenstein, D. M. and Kaplan, R. M. (1991) Measuring the quality of well-being in CF and lung transplantation. *Chest*, **100**, 1016–1018.
Orozco-Levi, M., Torres, A. and Ferrer, M. (1995) Semirecumbent position protects from pulmonary aspiration but not completely from gastroesophageal reflux in mechanically ventilated patients. *Am. J. Respir. Crit. Care Med.*, **152**, 1387–1390.
Orringer, M. K. (1999) The effects of tracheostomy tube placement on communication and swallowing. *Respir. Care*, **44**, 845–853.
Osman, L. M. (1997) Quality of life and hospital re-admission in patients with COPD. *Thorax*, **52**, 67–71.
Ott, A., Slooter, A. J. C. and Hofman, A. (1998) Smoking and risk of dementia and Alzheimer's disease. *Lancet*, **351**, 1840–1843.
Oz, M. C., Argenziano, M. and Rose, E. A. (1997) What is 'minimally invasive' coronary bypass surgery? *Chest*, **112**, 1409–1416.
Paice, J. A. (1998) Barriers to cancer pain relief. *J. Pain Symptom Manage.*, **16**, 1–9.
Pal, B. (1998) Following up outpatients by telephone. *Br. Med. J.*, **316**, 1647.
Palange, P., Forte, S. and Onorati, P. (1998) Effect of reduced body weight on muscle aerobic capacity in COPD. *Chest*, **114**, 12–18.
Palevsky, P. M. (1996) Hypernatremia in hospitalized patients. *Ann. Intern. Med.*, **124**, 197–203.
Paley, C. A. (1997) A way forward for determining optimal aerobic exercise intensity? *Physiotherapy*, **83**, 620–624.
Palmer, L. B. and Smaldone, G. C. (1998) Secretions in mechanically ventilated patients and duration of intubation. *Crit. Care Med.*, **26**, A112.
Pankow, W. (1997) Influence of noninvasive positive pressure ventilation on inspiratory muscle activity in obese subjects. *Eur. Respir. J.*, **10**, 2847–2852.
Panos (1994) *The Smoke Blows South*, Briefing Paper 13, Panos, London.
Papastamelos, C. (1995) Developmental changes in chest wall compliance in infancy and early childhood. *J. Appl. Physiol.*, **78**, 179–184.
Papazian, L. (1993) Effect of bolus doses of midazolam on ICP and CPP in patients with severe head injury. *Br. J. Anaesth.*, **71**, 267–271.
Papini, R. P. G. (1999) Current concepts in the management of burns with inhalation injury. *Care Crit. Ill*, **15**, 61–65.

Paratz, J. (1993) The effect of respiratory physiotherapy on ventilated neurosurgical patients. *Physiother. Theor. Pract.*, 9, 3–11.

Paret, G., Dekel, B. and Vardi, A. (1996) Heliox in respiratory failure secondary to bronchiolitis. *Pediatr. Pulmonol.*, 22, 322–323.

Parker, A. (1998) Paediatrics, in *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, (eds J. A. Pryor and B. A. Webber), Churchill Livingstone, Edinburgh.

Parker, A. and Middleton, P. G. (1998) Assessment*Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, (eds J. A. Pryor and B. A. Webber), Churchill Livingstone, Edinburgh.

Parker, J. C. and Hernandez, L. A. (1993) Mechanisms of ventilator-induced lung injury. *Crit. Care Med.*, 21, 131–142.

Parker, M. M., Schubert, W. and Shelhamer, J. H. (1984) Perceptions of a critically ill patient experiencing therapeutic paralysis in an ICU. *Intensive Care Med.*, 12, 69–71.

Parmar, M. (1998) Evidence based medicine. *Care Crit. Ill*, 14, 218.

Partridge, M. R. (1992) Smoking and the young. *Br. Med. J.*, 305, 2.

Partridge, M. R. (1994) Asthma: guided self-management. *Br. Med. J.*, 308, 547–548.

Partridge, C. (1996) Evidence based medicine – implications for physiotherapy? *Physiother. Res. Int.*, 1, 69–73.

Parvez, L., Vaidya, M. and Sakhardande, A. (1996) Evaluation of antitussive agents in man. *Pulm. Pharmacol.*, 9, 299–308.

Pasero, C. L. (1994) Avoiding opioid-induced respiratory depression. *Am. J. Nurs.*, 94, 25–31.

Pasterkamp, H. (1997) Respiratory sounds. *Am. J. Respir. Crit. Care Med.*, 156, 974–987.

Patel, M. (1993) Optimal time for measuring the cardiorespiratory effects of PEEP. *Chest*, 104, 139–142.

Patel, H. and Yang, K. L. (1995) Variability of intrinsic PEEP. *Crit. Care Med.*, 23, 1074–1079.

Patel, R. G., Petrini, M. F. and Dwyer, T. M. (1999) Work of breathing during weaning from ventilation. *Respir. Care*, 44, 421–427.

Paterson, D. H. (1992) Effects of ageing on the cardiorespiratory system. *Can. J. Sport Sci.*, 17, 171–177.

Patman, S., Jenkins, S. and Bostock, S. (1998) Cardiovascular responses to manual hyperinflation in postoperative coronary artery surgery patients. *Physiother. Theor. Pract.*, 14, 5–12.

Patman, S., Jenkins, S. and Stiller, K. (1999) Manual hyperinflation: effects on respiratory parameters. *Anaesth. Intensive Care*, 27(suppl.), 96.

Patterson, R., Mazur, N. and Roberts, M. (1998) Hypersensitivity pneumonitis due to humidifier disease. *Chest*, 114, 931–933.

Patterson, C., Teale, C. and Dow, L. (1999) Do we overlook respiratory symptoms and airflow obstruction in elderly medical admissions [letter]? *Age Ageing*, 28, 326.

Pavia, D. (1991) Bronchoalveolar clearance. *Respiration*, 58(suppl.), 13–17.

Pavy, T. J. G. and Doyle, D. L. (1996) Prevention of phantom limb pain by infusion of local anaesthetic into the sciatic nerve. *Pain*, 24, 599–600.

Paxton, D. (1996) Treating postoperative nausea in children. *Hosp. Update*, 22, 428–430.

Payne, R. (1995) *Relaxation Techniques*, Churchill Livingstone, Edinburgh.

Peach, H. and Pathy, M. S. (1981) Follow-up study of disability among elderly patients discharged from hospital with exacerbations of chronic bronchitis. *Thorax*, 36, 585–589.

Pearce, A. C. (1984) Smoking and anaesthesia. *Anesthesiology*, 61, 576–584.

Pearson, M. G. (1996) Managing asthma. *ACPRC J.*, 29, 53.

Peche, R. (1996) Sternomastoid muscle size and strength in patients with severe COPD. *Am. J. Respir. Crit. Care Med.*, 153, 422–425.

Pedersen, S. (1996) Inhalers and nebulizers. *Respir. Med.*, 90, 69–77.

Pederson, C. and Bjerke, T. (1999) Pediatric pain management. *Dimens. Crit. Care Nurs.*, 18, 42–51.

Peek, G. J. (1997) Extra-corporeal membrane oxygenation. *Br. Med. Bull.*, 53, 745–746.

Peerless, J. R., Davies, A., Klein, D. and Yu, D. (1999) Skin complications in the ICU. *Clin. Chest Med.*, 20, 453–467.

Pehrsson, K. (1994) Quality of life of patients treated by home mechanical ventilation due to restrictive ventilatory disorders. *Respir. Care*, 88, 21–26.

Pellegrino, R., Rodarte, J. R. and Brusasco, V. (1998) Assessing the reversibility of airway obstruction. *Chest*, 114, 1607–1612.

Pellegrino, R., Villosio, C., Milanese, U. and Garelli, G. (1999) Breathing during exercise in subjects with mild-to-moderate airflow obstruction. *J. Appl. Physiol.*, 87, 1697–1704.

Pellino, T. A. (1998) Perceived control mediates the relationship between pain severity and patient satisfaction. *J. Pain Symptom Manage.*, 15, 110–116.

Pelosi, P. and Gattinoni, L. (1996) Mechanical ventilation in ARDS. *Curr. Opin. Anaesth.*, 9, 515–522.

Pelosi, P., Cadringher, P. and Bottino, N. (1999) Sigh in acute respiratory distress syndrome. *Am. J. Respir. Crit. Care Med.*, 159, 872–880.

Pennock, B. E. (1994) Distressful events in the ICU. *Heart Lung*, 23, 323–327.

Penson, R. T. and Rudd, R. M. (1997) Commentary: octreotide and hypertrophic pulmonary osteoarthropathy. *Thorax*, 52, 297–298.

Pentland, B. (1994) Pain in Guillain–Barré syndrome. *Pain*, 59, 159–164.

Peper, E. (1992) Fifteen-month follow up with asthmatics utilizing EMG/incentive inspirometer feedback. *Biofeedback Self-reg.*, 17, 143–151.

Peruzzi, W. T. (1996) The current status of PEEP. *Respir. Care*, 41, 273–81.

Petersen, G. M., Pierson, D. J. and Hunter, P. M. (1979) Arterial oxygen saturation during nasotracheal suctioning. *Chest*, 76, 283–287.

Peto, R., Speizer, E. and Cochrane, A. L. (1983) The relevance in adults of airflow obstruction but not mucus hypersecretion to mortality from chronic lung disease. *Am. Rev. Respir. Dis.*, 128, 491–500.

Petty, T. L. (1990) The national mucolytic study. *Chest*, 97, 75–83.

Petty, T. L. and O'Donohue, W. J. (1994) Further recommendations for prescribing and research in long-term oxygen therapy. *Am. J. Respir. Crit. Care Med.*, 150, 875–877.

Phelan, D. (1995) Hopeless cases in intensive care. *Care Crit. Ill*, 11, 196–197.

Phillips, S. (1993) Zingiber officinale (ginger): an antiemetic for day surgery. *Anaesthesia*, 48, 715–717.

Phillips, G. D. and Millard, F. J. C. (1994) The therapeutic use of ultrasonic nebulizers in acute asthma. *Respir. Med.*, 88, 387–389.

Phillips, S., Hutchinson, S. and Davidson, T. (1993) Preoperative drinking does not affect gastric contents. *Br. J. Anaesth.*, 70, 6–9.

Phillips, S., Daborn, A. K. and Hatch, D. J. (1994) Preoperative fasting for paediatric anaesthesia *Br. J. Anaesth.*, 73, 529–536.

Phillips, G. E., Thomas, S. and Heather, S. (1998) Airway response of children with primary ciliary dyskinesia to exercise and β_2-agonist challenge. *Eur. Respir. J.*, 11, 1389–1391.

Picardo, C. (1996) Classification of severe asthma exacerbations. *Eur. Respir. J.*, 9, 1175–1178.

Pickler, R. H. (1996) Effects of nonnutritive sucking on behavioral organisation and feeding in preterm infants. *Nurs. Res.*, 45, 132–135.

Piérart, F., Wildhaber, J. H. and Vrancken, I. (1999) Washing plastic spacers in household detergent reduces electrostatic charge. *Eur. Respir. J.*, 13, 673–678.

Pieri-Davies, S., Ewart, K. and Boden, K. (1994) On-call respiratory issues threaten patients (letter). *Frontline*, 7 **July**.

Pierson, D. J. and Lakshminarayan, S. (1984) Postoperative ventilatory management. *Respir. Care*, 29, 603–611.

Pietropauli, J. A., Rogers, F. B. and Shackford, S. R. (1992) The deleterious effects of intraoperative hypotension in patients with severe head injuries. *J. Trauma*, 33, 403–407.

Piirilä, P., Sovijärvi, A. R. A. and Kaisla, T. (1991) Crackles in patients with fibrosing alveolitis, bronchiectasis, COPD and heart failure. *Chest*, 99, 1076–1083.

Pilbeam, S. P. (1998) *Mechanical Ventilation*, Mosby, St Louis, MO.

Pin, I. (1992) Use of induced sputum to investigate airway inflammation in asthma. *Thorax*, 47, 25–29.

Piña IL (1996) Exercise and heart failure. *Chest*, 110, 1317–27.

Pina, J. S., Behrens, M. L. and Moffit, D. R. (1997) Are nose clips necessary in adults and children performing forced expiratory spirometry? *Respir. Care*, 42, 492–497.

Pinney, S., Freeman, L. J. and Nixon, P. G. F. (1987) Role of the nurse counsellor in managing patients with the hyperventilation syndrome. *J. Roy. Soc. Med.*, 80, 216–218.

Pitkin, A. (1997) Hyperbaric oxygen therapy in intensive care. *Br. J. Intensive Care*, 7, 107–113.

Pitkin, R. M. (1998) Listen to the patient. *Br. Med. J.*, 316, 1252.

Plavka, R., Kopecky, P. and Sebron, V. (1999) A prospective randomized comparison of conventional mechanical ventilation and very early high frequency oscillatory ventilation. *Intensive Care Med.*, 25, 68–75.

Playfair, W. (1881) Notes on the systematic treatment of nerve prostration. *Lancet*, i, 8–9.

Plaza, V., Serra-Batlles, J. and Ferrer, M. (2000)

Quality of life and economic features in elderly asthmatics. *Respiration*, 67, 65–70.
Plebani, A. (1997) Usefulness of chest physiotherapy with PEP in HIV-infected children. *Acta Paediatr.*, 86, 1195–1197.
Poi, P. J. H., Chuah. S. Y. and Srinivas, P. (1998) Common fears of patients undergoing bronchoscopy. *Eur. Respir. J.*, **11**, 1147–1149.
Pollack, C. (1999) *Cardiorespiratory Physiotherapy Conference*, London.
Pollock, M. L. and Wilmore, J. H. (1990) *Exercise in Health and Disease*. W. B. Saunders, London.
Poponick, J. M. (1999) Use of a ventilatory support system (BiPAP) for acute respiratory failure. *Chest*, **116**, 166–171.
Postma, D. S. (1991) Inhaled therapy in COPD. *Respir. Med.*, 85, 447–449.
Postma, D. S. (1998) When can an exacerbation of COPD be treated at home? *Lancet*, 351, 1827–1828.
Potempa, K. M., Folta, A. and Braun, L. T. (1991) The relationship of resting and exercise blood pressure in subjects with essential hypertension before and after propranolol. *Heart Lung*, **21**, 509–514.
Potts, M. (1994) Grief has to be. *Lancet*, 343, 279–280.
Powell, K. E. and Pratt, M. (1996) Physical activity and health *Br. Med. J.*, 313, 126–127.
Powell-Tuck, J. (1997) Penalties of hospital undernutrition. *J. Roy. Soc. Med.*, 90, 8–11.
Powell-Tuck, J. (2000) Malnutrition and pressure ulcers. *Br. J. Nurs.*, 9, 194–204.
Power, C. and Li, L. (2000) Cohort study of birthweight, mortality and disability. *Br. Med. J.*, 320, 840–841.
Prabhu, V. G. (1997) Pulmonary function changes after nebulized and IV frusemide in ventilated premature infants. *Arch. Dis. Child.*, 77, F32–F35.
Prabhu, M. B., Mink, J. T. and Graham, B. L. (1990). Effect of a deep breath on gas mixing and diffusion in the lung. *Respir. Physiol.*, 79, 195–204.
Prasad, S. A. (1993) Current concepts in physiotherapy. *J. Roy. Soc. Med.*, 86(suppl. 20), 23–29.
Prasad, S. A. and Hussey, J. (1995) *Paediatric Respiratory Care*, Chapman & Hall, London.
Prescott, E. (1998) Smoking and risk of myocardial infarction in women and men. *Br. Med. J.*, 316, 1043–1047.
Prescott, E. (1999) Socioeconomic status, lung function and admission to hospital for COPD. *Eur. Respir. J.*, 13, 1109–1114.
Preston, I. M., Matthews, H. R. and Ready, A. R. (1986) Minitracheotomy. *Physiotherapy*, 72, 494–6.
Prichard, A. J. N. (1994) Tracheostomy. *Care Crit. Ill*, 10, 64–69.
Prough, D. S. (1996) Head trauma and brain resuscitation. *Curr. Opin. Anaesth.*, 9, 188–193.
Pryor, J. A. and Webber, B. A. (1998) *Physiotherapy for Respiratory and Cardiac Problems*, 2nd edn, Churchill Livingstone, Edinburgh.
Ptacek, J. T., Patterson, D. R. and Montgomery, B. K. (1995) Pain, coping and adjustments in patients with burns. *J. Pain Symptom Manage.*, 10, 446–455.
Punt, J. (1989) Head injury. *Care Crit. Ill*, 5, 233–237.
Purssell, E.. (1994) Telling children about their impending death. *Br. J. Nurs.*, 3, 119–120.
Purtilo, R. B. (1976) Similarities in patient response to chronic and terminal illness. *Phys. Ther.*, 56, 279–284.
Putensen, C., Mutz, N. F. and Putensen, G. (1999) Spontaneous breathing during ventilatory support improves ventilation-perfusion distributions in patients with ARDS. *Am. J. Respir. Crit. Care Med.*, 159, 1241–1248.
Quanjer, P. H. (1993) Standardised lung function testing. *Eur. Respir. J.*, 6(suppl. 16), S1.
Qureshi, A. I., Suarez, J. I. and Bhardwaj, A. (1998) Use of hypertonic (3%) saline/acetate infusion in the treatment of cerebral edema. *Crit. Care Med.*, 26, 440–446.
Radford, R. (1982) A rational basis for percussion-augmented mucociliary clearance. *Respir. Care*, 27, 556–563.
Rajagopalan, N., Maurer, J. and Kesten, S. (1996) Bronchodilator response at low lung volumes predicts bronchiolitis obliterans. *Chest*, 109, 405–407.
Rajesh, V. T., Singhi, S. and Kataria, S. (2000) Tachypnoea is a good predictor of hypoxia in acutely ill infants. *Arch. Dis. Child.*, 82, 46–49.
Rakshi, K. (1994) Management of acute bronchiolitis. *Arch. Dis. Child.*, 71, 463–469.
Ralafox, M. (2000) Diagnostic value of tachypnoea in pneumonia defined radiologically. *Arch. Dis. Child.*, 82, 41–45.
Ramonatxo, M., Boulard, P. and Préfaut, C. (1995)

Validation of a noninvasive tension-time index of inspiratory muscles. *J. Appl. Physiol.*, 78, 646–653.

Rampulla, C. (1992) Dyspnea on exercise. *Chest*, **101**, 248S–252S.

Ramsay, J. (1994) The psychology of childhood asthma. *Paed. Nurs.*, 6, 17–18.

Ramsey, B. W. (1996) Management of pulmonary disease in patients with CF. *N. Engl. J. Med.*, 335, 179–188.

Rankin, N. (1998) What is optimum humidity? *Respir. Care Clin. North Am.*, 4, 321–328.

Raoof, S., Chowdhrey, N. and Raoof, S. (1999) Effect of combined kinetic therapy and percussion therapy on the resolution of atelectasis in critically ill patients. *Chest*, **115**, 1658–1666.

Ras, G. J., Staden, M. V. and Schultz, C. (1994) Respiratory manifestations of rigid spine syndrome. *Am. J. Respir. Crit. Care Med.*, 150, 540–546.

Rastall, M., Brooks, B. and Klarneta, M. (1999) An investigation into younger and older adults' memory for physiotherapy exercises. *Physiotherapy*, 85, 122–128.

Rawal, N. (1997) Survey of postoperative analgesia following ambulatory surgery. *Acta Anaesthesiol. Scand.*, 41, 1017–1022.

Ray, J. F. (1974) Immobility, hypoxemia, and pulmonary arteriovenous shunting. *Arch. Surg.*, 109, 537–541.

RCP (1996) *Clinical Guidelines for Cystic Fibrosis Care*, Royal College of Physicians, London.

RCP (1999) *Domiciliary Oxygen Therapy Services*, Royal College of Physicians, London.

Reardon, J. (1994) The effect of comprehensive outpatient pulmonary rehabilitation on dyspnea. *Chest*, 105, 1046–1052.

Redfern, S. (1985) Taking some of my own medicine. *Care Crit. Ill*, 1, 6–7.

Reece-Smith, H. (1997) Pancreatitis. *Care Crit. Ill*, 13, 135–138.

Reed, J. C. (1996) *Chest Radiology*, Mosby, St Louis, MO, p. 217.

Rees, J. (1987) Cough. *Update*, 34, 1110–1119.

Regnard, C. (1997) Mouth care, skin care and lymphoedema. *Br. Med. J.*, 315, 1002–1005.

Regnis, J. A. (1994) Benefits of nocturnal nasal CPAP in patients with CF. *Chest*, 106, 1717–1724.

Reick, C. W. (1996) Auto-PEEP associated with excessive oxygen flow through five manual resuscitators. *Respir. Care*, 41, 1009–1012.

Reid, P. T. and Rudd, R. M. (1993) Management of malignant pleural effusion. *Thorax*, 48, 779–780.

Reid, W. D. and Loveridge, B. M. (1983) Physiotherapy management of patients with COAD. *Physiother. Can.*, 35, 183–195.

Reiland, K. E. (2000) Gastrointestinal surgical patients' outcomes influenced by nutrition. *AORN J.*, 71, 199–204.

Reina-Rosenbaum, R., Bach, J. R. and Penek, J. (1997) The cost/benefits of outpatient-based pulmonary rehabilitation. *Arch. Phys. Med. Rehabil.*, 78, 240–244.

Reisman, J. J. (1988) Role of conventional physiotherapy in CF. *J. Pediatr.*, 113, 632–636.

Renfroe, K. L. (1988) Effect of progressive relaxation on dyspnea and state anxiety in patients with COPD. *Heart Lung*, 17, 408–413.

Rennie, M. (1997) Central dogma unproven. *Br. J. Intensive Care*, 7, 125.

Rennie, M. (1998) The gut, central as always. *Br. J. Intensive Care*, 8, 5.

Rennie, M. (2000) A rotten culture. *Br. J. Int. Care*, 10, 41.

Renston, J. P. (1994) Respiratory muscle rest using nasal BiPAP ventilation in patients with stable severe COPD. *Chest*, 105, 1053–1060.

Resende, T. L. and O'Neill, P. A. (1992) Effects of exercise and abdominal massage on constipation and faecal incontinence. *Physiotherapy*, 78, 589.

Restrick, L. J., Clapp, B. R. and Mikelson, C. (1997) Nasal ventilation in pregnancy. *Eur. Respir. J.*, 10, 2657–2658.

Revill, S. M., Morgan, M. D. L. and Singh, S. J. (1999) The endurance shuttle walk. *Thorax*, 54, 213–222.

Rezvani, K. (1996) Ecstasy induced pneumomediastinum. *Thorax*, 51, 960–961.

Rhodes, K. (1993) Diuretics in clinical practice. *Practitioner*, 237, 49–52.

Rialp, G., Betbes A. J. and Ormaechea, E. (1997) Changes in pulmonary mechanics with change from supine to prone in ARDS. *Intensive Care Med.*, 23, S12.

Ricour, C. (1989) Stress-induced disturbances of the gastro-intestinal tract in children. *Intensive Care Med.*, 15, S32–S36.

Riedler, J. (1996) Inhaled hypertonic saline increases sputum expectoration in CF. *J. Pediatr. Child Health*, 32, 48–50.

Riegel, B. (1985) A review and critique of the literature on preoxygenation for endotracheal suctioning. *Heart Lung*, 14, 507–518.

Ries, A. L. (1994) The importance of exercise in pulmonary rehabilitation. *Clin. Chest Med.*, 15, 327–337.

Ries, A. L. and Moser, K. M. (1996) *Shortness of Breath: a Guide to Better Living and Breathing*. Mosby, Missouri.

Rimm, E. R. (1995) Prospective study of cigarette smoking, alcohol use and the risk of diabetes. *Br. Med. J.*, 310, 555–559.

Ringbaek, T. J. (2000) Rehabilitation of patients with COPD. Exercise twice a week is not sufficient! *Respir. Med.*, 94, 150–154.

Ritson, P. (2000) Manual bagging and shallow suction. *ACPRC Conference*, E. Midlands.

Roa, J. (1991) Work of breathing and ventilatory muscle recruitment during pursed lip breathing. *Am. Rev. Respir. Dis.*, 143, 4.

Roberts, H. (1996) Listen to the parents. *Br. Med. J.*, 313, 954–955.

Roberts, I. (1998a) Human albumin administration in critically ill patients. *Br. Med. J.*, 317, 23.

Roberts, C. J., Parke, T. J. and Sykes, M. K. (1993) Effects of intraoperative inspired gas mixtures on postoperative nocturnal oxygen saturation. *Br. J. Anaesth.*, 71, 476–480.

Rochester, D. F. (1992) Nutritional repletion. *Sem. Respir. Med.*, 13, 44.

Rocker, G. M., Mackenzie, M.-G. and Williams, B (1999) Noninvasive positive pressure ventilation: successful outcome in patients with ALI/ARDS. *Chest*, 115, 173–177.

Roizen, M. F. (1997) Anesthesia and medical disease. *Curr. Opin. Anaesth.*, 10.

Rolla, G. (1994) Effect of pleurotomy on pulmonary function after CABG with IMA. *Respir. Med.*, 88, 417–420.

Rollin, B. (1976) *First You Cry*, New American Library, New York.

Romand, J.-A. and Donald, F. (1995) Physiological effects of CPAP in the critically ill. *Care Crit. Ill*, 11, 239–243.

Roosevelt, G. (1996) Dexamethasone in bronchiolitis. *Lancet*, 348, 292–295.

Ropper, A. H., O'Rourke, D. and Kennedy, S. K. (1982) Head position, ICP and compliance. *Neurology*, 32, 1288–1291.

Rosario, N. D., Sassoon, C. S. H., Chetty, K. G. and Gruer, S. E. (1997) Breathing pattern during acute respiratory failure and recovery. *Eur. Respir. J.*, 10, 2560–2565.

Rosenberg, M. (1992) Patient-controlled analgesia. *J. Oral Maxillofacial Surg.*, 50, 386–389.

Rosenthal, M., Christensen, B. K. and Ross, T. P. (1998) Depression following traumatic brain injury. *Arch. Phys. Med. Rehabil.*, 79, 90–103.

Rosenthal, M. H. (1999) Intraoperative fluid management. *Chest*, 114, 106S–112S.

Rosner, V., Hannhart, B. and Chabot, F. (1999) Validity of transcutaneous oxygen/carbon dioxide pressure measurement. *Eur. Respir. J.*, 13, 1044–1047.

Rossi, A. (1994) Effects of PEEP on $\dot{V}_A/\dot{Q}$ mismatching in ventilated patients with chronic airflow limitation. *Am. J. Respir. Crit. Care Med.*, 149, 1077–1084.

Rossi, A., Appendini, L. and Poggi, R. (1993) Acute bronchial asthma: indications for intensive care. *Eur. Respir. J.*, 3, 400–403.

Rossoff, L. J. (1995) Is handwashing really needed in an intensive care unit? *Crit. Care Med.*, 23, 1211–1216.

Roth, G. (1990) *Maps to Ecstasy*, Crucible, London.

Rothen, H. U. (1993) Re-expansion of atelectasis during general anaesthesia. *Br. J. Anaesth.*, 71, 788–795.

Roussos, C. (1996) Fatigue of the respiratory muscles. *Int. Care Med.*, 22, 134–155.

Rowbotham, E. (1990) ACPRC Study Day, London.

Rowe, B. H. (1993) Performance of an asthma quality of life questionnaire. *Am. Rev. Respir. Dis.*, 148, 675–681.

Rowlee, S. C. (1999) Monitoring neuromuscular blockade in the ICU. *Heart Lung*, 28, 352–362.

Royse, C. F. (1997) The effect of supplemental oxygen on the incidence of hypoxaemia after premedication in patients undergoing cardiac surgery. *Anaesth. Intensive Care*, 25, 347–349.

Rubin, B. K. (1997) The classification and use of muco-active medications. *Pediatr. Pulmonol.*, 14, 118.

Rudkin, S. T. and White, R. J. (1995) Use of respiratory specialist nurse in the education and supervision of patients receiving LTOT. *Respiratory Nurses Conference*, Warwick.

Rudy, E. B. (1991) Endotracheal suctioning in adults with head injury. *Heart Lung*, 20, 667–674.

Ruffolo, C. (1998) Gastric tonometry. *Crit. Care Nurs. Q.*, 21, 26–32.

Ruggier, R., Bailey, C. R. and Grounds, R. M. (1994) A simple method for maintenance of oxygen

saturation following intravenous induction of anaesthesia. *Anaesthesia*, 49, 759–761.
Ruiz, P. A. (1993) The needs of a patient in severe status asthmaticus. *Intens. Crit. Care Nurs.*, 9, 28–39.
Russell, L. R. (2000) Malnutrition and pressure ulcers. *Br. J. Nurs.*, 9, 194–204.
Russell, P. O., Epstein, L. H. and Johnston, J. J. (1988) The effects of physical activity as maintenance for smoking cessation. *Addict. Behav.*, 13, 215–218.
Russo, E. (1998) Cannabis for migraine treatment. *Pain*, 76, 3–8.
Russo, P. (1990) Cardiovascular responses associated with activity and inactivity. In: *Key Issues in Neurological Physiotherapy* (Eds. Ada, L., Canning, C.). Butterworth-Heinemann, London.
Rusznak, C. (1991) Influence of salbutamol and salmeterol on ciliary beat frequency. *Thorax*, 46, 782P.
Ruta, T. S. (1993) The effect of acute hypocapnia on local cerebral blood flow. *Anesthesiogy*, 78, 134–140.
Ryan, C. J. (1998) Matters of life and death. *Med. J. Austr.*, 169, 124.
Ryan, D. and Higgins, B. (1998) Corticosteroid therapy in late ARDS. *Care Crit. Ill*, 14, 2–5.
Ryan, C. F. and Love, L. L. (2000) Unpredictable results of laser assisted uvulopalatoplasty. *Thorax*, 55, 399–404.
Ryan, C. F., Road, R. D. and Buckley, P. A. (1993) Energy balance in stable malnourished patients with COPD. *Chest*, 103, 1038–1044.
Rylance, G. (1996) Making decisions with children. *Br. Med. J.*, 312, 794.
Saag, K. G. (1997) Cigarette smoking and rheumatoid arthritis severity. *Ann. Rheum. Dis.*, 56, 463–469.
Sacchetti, A. (1994) Pediatric analgesia and sedation. *Ann. Emerg. Med.*, 23, 237–249.
Sahebjami, H. (1993) Anthropometric and pulmonary function test profiles of outpatients with stable COPD. *Am. J. Med.*, 94, 469–474.
Sahn, S. A. and Heffner, J. E. (1994) *Pulmonary Pearls II*, Mosby, St Louis, MO, p. 162.
Sahn, S. A. and Heffner, J. E. (2000) Spontaneous pneumothorax. *New. Eng. J. Med.*, 342, 868–874.
Saigal, S. (2000) School difficulties at adolescence in a regional cohort of children who were extremely low birth weight. *Pediatrics*, 105, 325–331.
Saint, S. and Matthay, M. A. (1998) Risk reduction in the ICU. *Am. J. Med.*, 105, 515–523.
Sakakibara, M. (1996) Effect of slowed respiration on cardiac parasympathetic response to threat. *Psychosomatic Med.*, 58, 32–37.
Sakellari, V. (1997) Hyperventilation effect on postural sway. *Arch. Phys. Med. Rehabil.*, 78, 730–736.
Salazar, J. (1991) Exercise physiology, in *Pulmonary Therapy and Rehabilitation*, (eds F. Haas and K. Axen), Williams & Wilkins, Baltimore, MD, pp. 58,63.
Salmon, B., Wilson, N. M. and Silverman, M. (1990) How much aerosol reaches the lungs of wheezy infants and toddlers? *Arch. Dis. Child.*, 65, 401–403.
Salmon, P. (1992) Surgery as a psychological stressor. *Stress Med.*, 8, 193–198.
Saltin, B., Blomqvist, G. and Mitchel, J. H. (1968) Response to bed rest after exercise and after training. *Circulation*, 38(suppl. 7), 1–76.
Sammon, M. P. (1994) Entrainment of respiration to rocking in premature infants. *J. Appl. Physiol.*, 77, 1548–1554.
Samuels, M. P. and Southall, D. P. (1989) Negative extrathoracic pressure in infants and young children. *Br. Med. J.*, 299, 1253–1257.
Samuels, S., Samuels, M. and Dinwiddie, R. (1995) A survey of physiotherapy techniques used in specialist clinics for cystic fibrosis. *Physiotherapy*, 81, 279–283.
Sandler, H. (1988) The hemodynamic effects of repeated bed rest exposure. *Aviat., Space Environ. Med.*, 54, 1047–1054.
Sapira, J. D. (1995) About egophony. *Chest*, 108, 865–867.
Sasse, S. A. (1995) Arterial oxygenation time after an FiO_2 increase in mechanically ventilated patients. *Am. J. Respir. Crit. Care Med.*, 152, 148–152.
Sato, P., Sargur. M. K. and Schoene, R. B. (1986) Hypnosis effect on carbon dioxide chemosensitivity. *Chest*, 89, 828–831.
Saudny, H. (1997) Impact of nutritional support on functional status. *Am. J. Respir. Crit. Care Med.*, 156, 794–799.
Savage, J. (1992) Advice to take home. *Nurs. Times*, 88(38), 24–27.
Savci, S., Ince, D. I. and Arikan, H. (2000) A comparison of autogenic drainage and the active cycle of breathing in patients with COPD. *J. Cardiopulm. Rehab.* 20, 37–43.
Sawdon, V., Woods, I. and Proctor, M. (1995) Post-

intensive care interviews. *Intens. Crit. Care Nurs.*, 11, 329–332.
Sawyer, E. H. and Clanton, T. L. (1993) Improved pulmonary function and exercise tolerance with inspiratory muscle conditioning in children with cystic fibrosis. *Chest*, 104, 1490–1497.
Scalvini, S., Porta, R. and Zanelli, E. (1999) Effects of oxygen on autonomic nervous system dysfunction in patients with COPD. *Eur. Respir. J.*, 13, 119–124.
Scarlett, J. (1998) Minimally invasive cardiac surgery. *Crit. Care Nurse*, 21, 16–23.
Scheeren, T. (1997) Prostacyclin (PGI2). *Intensive Care Med.*, 23, 146–158.
Scheinkestel, C. D., Bailey, M. and Myles, P. S. (1999) Hyperbaric or normobaric oxygen for acute carbon monoxide poisoning. *Med. J. Austr.*, 170, 203–210.
Schelling, G. (1998) Health-related quality of life and posttraumatic stress disorder in survivors of ARDS. *Crit. Care Med.*, 26, 651–659.
Schenkel, S. (1996) Oxygen saturation during daily activities in COPD. *Eur. Respir. J.*, 9, 2584–2589.
Scherder, E. (1999) Epilepsy: a possible contraindication for TENS [letter]. *J. Pain Symptom Manage.*, 17, 152–153.
Schermuly, R. T. (2000) Differential impact of ultrasonically nebulized versus tracheal-instilled surfactant on $\dot{V}_A/\dot{Q}$ mismatch. *Am. J. Respir. Crit. Care Med.*, 161, 152–159.
Schieb, D. A., Protas, E. J. and Hasson, S. M. (1996) Special feature: implications of physical restraint and restraint reduction of older persons. *Top. Geriatr. Rehabil.*, 12, 70–83.
Schleifer, T. J., Gleason, J. and Grady, L. (1994) Patient responsibility in an innovative COPD program. *Physiother. Can.*, 46(suppl.), 81.
Schlichtig, R. and Sargent, S. C. (1990) Nutritional support of the mechanically ventilated patient. *Crit. Care Clin.*, 6, 767–777.
Schneiderman, J. (2000) A randomized controlled trial of a 3-year home exercise program in CF. *J. Pediatr.*, 136, 304–310.
Schönhofer, B., Mang, H. and Haidl, P. (1996) Controlled ventilation in conscious patients. *Eur. Respir. J.*, 9, 330s.
Schönhofer, B., Geibel, M. and Sonneborn, M. (1997) Daytime mechanical ventilation in chronic respiratory insufficiency. *Eur. Respir. J.*, 10, 2840–2846.
Schulz, R. (2000) The association between depression and mortality in older adults. *Arch. Int. Med.*, 160, 1761–1768.
Schwartz, S. Z. (1987) Effects of blood volume and discontinuance of ventilation on pulmonary vascular pressures. *Crit. Care Med.*, 15, 671–675.
Schwartzstein, R. M. (1992) Asthma: to run or not to run? *Am. Rev. Respir. Dis.*, 145, 739–740.
Sciaky, A. J. (1994) Mobilizing the intensive care unit patient. *Phys. Ther. Pract.*, 3, 69–80.
Scum (1981) Simple mechanical method for decreasing the incidence of thromboembolism. *Am. J. Surg.*, 141, 582–585.
Scurr, J. H., Robbe, I. J., Ellis, H. and Goldsmith, H. S. (1981) Simple mechanical method for decreasing the incidence of thromboembolism. *Am. J. Surg.*, 141, 582–585.
Sealey, C. (1999) Two common pitfalls in clinical audit: failing to complete the audit cycle and confusing audit with research. *Br. J. Occup. Ther.*, 62, 238–243.
Sears, M. R. (1994) Growing up with asthma. *Br. Med. J.*, 309, 72–73.
Seiner, S. J. (1999) Treating depression in patients with cardiovascular disease. *Harvard Rev. Psychiat.*, 7, 85–93.
Senekal, M. (1994) The optimal physiotherapeutic approach to penetrating stab wounds of the chest. *S. Afr. J. Physiother.*, 50, 29–36.
Senekal, M. and Eales, C. (1994) The optimal physiotherapeutic approach to penetrating stab wounds of the chest. *S. Afr. J. Physiother.*, 50, 29–36.
Shah, P. L., Scott, S. F. and Hodson, M. E. (1994) Lobar atelectaiss in CF and treatment with recombinant human DNase. *Respir. Med.*, 88, 313–315.
Shaheen, S. O., Barker, D. J. P. and Shiell, A. W. (1994) The relationship between pneumonia in early childhood and impaired lung function in late adult life. *Am. J. Respir. Crit. Care Med.*, 149, 616–619.
Shapiro, S. H., Ernst, P. and Gray-Donald, K. (1992) Effect of negative pressure ventilation in severe COPD. *Lancet*, 340, 1425–1429.
Shapiro, B. A., Warren, J. and Egol, A. B. (1995) Practice parameters for intravenous analgesia and sedation. *Crit. Care Med.*, 23, 1596–1600.
Sharkey, R. A. (1997) Renal functional reserve in patients with severe COPD. *Thorax*, 52, 411–415.
Sharma, O. P. (1998) Many faces of pulmonary aspergillosis. *Eur. Respir. J.*, 12, 795–715.

Sharpe, M. (1998) Putting the rest cure to rest – again. *Br. Med. J.*, 316, 796.
Sharpe, P. (2000) Cannabis: time for scientific evaluation of this ancient remedy? *Anesth. Analg.*, 90, 237–240.
Sharpe, P. and Hanning, C. (1999) Neurological sequelae. *Best Pract. Res. Clin. Anaesth.*, 13, 451–463.
Sheldon, F. (1998) Bereavement. *Br. Med. J.*, 316, 456–458.
Shelledy, D. C. (1995) A comparison of the effects of assist-control, SIMV and SIMV with pressure support. *Heart Lung*, 24, 67–75.
Shelly, M. P. (1992) Intensive care sedation, in Vincent, J. L. (ed) *Yearbook of Intensive Care and Emergency Medicine*, Springer-Verlag, Berlin.
Shelly, M. P. (1998) Sedation in the ITU. *Care Crit. Ill*, 14, 85–88.
Shelly, M. P., Lloyd, G. M. and Park, G. R. (1988) A review of the mechanisms and methods of humidification of inspired gases. *Intensive Care Med.*, 14, 1–9.
Shepherd, R. W. (1988) Increased energy expenditure in young children with CF. *Lancet*, i, 1300–1303.
Sherman, C. B., Hubert, M. and Fogel, B. S. (1992) Unrecognized respiratory disease in the elderly. *Am. Rev. Respir. Dis.*, 145, A763.
Shiao, S. Y. P. K. (1995) Nasogastric tube placement. *Nurs. Res.*, 44, 82–88.
Shikora, S. A. (1996) Enteral nutrition and the critically ill. *Postgrad. Med. J.* 72, 395–402.
Shivaram, U., Donath, J. and Khan, F. A. (1987) Effects of continuous positive airway pressure in acute asthma. *Respiration*, 52, 157–162.
Shneerson, J. M. (1996a) Motor neurone disease. *Br. Med. J.*, 313, 244.
Shneerson, J. M. (1996b) Techniques in mechanical ventilation. *Thorax*, 51, 756–761.
Short, M. A., Brooks-Brunn, J. A. and Reeves, J. (1996) The effects of swaddling versus standard positioning on neuromuscular development in very low birth weight infants. *Neonat. Netw.*, 15, 25–31.
Shum, M. I. D., Lauder, I. and Lam, W. K. (1993) Effect of antibiotics on sputum inflammatory contents in acute exacerbations of bronchiectasis. *Respir. Med.*, 87, 449–454.
Siafákas, N. M., Mitrouska, I. and Bouros, D. (1999) Surgery and the respiratory muscles. *Thorax*, 54, 458–465.
Sibuya, M., Yamadam, M. and Kanamaru, A. (1994) Effect of chest wall vibration on dyspnoea. *Am. J. Respir. Crit. Care Med.*, 149, 1235–1240.
Siddiqui, A. U. (2000) Pulmonary embolism as a consequence of applying sequential compression device on legs in a patient asymptomatic of deep vein thrombosis. *Anesthesiology*, 92, 880–882.
Sigel, B. (1973) Compression of the deep venous system of the lower leg. *Arch. Surg.*, 106, 38–44.
Signorile, J. F. (1995) Increased muscle strength in paralyzed patients after spinal cord injury. *Arch. Phys. Med. Rehabil.*, 76, 55–58.
Silkoff, P. E. and Martin, R. J. (1998) Chronobiology of asthma. *Respir. Ther. Int.*, 7, 21–26.
Silver, J. A. and Foster, W. M. (1990) Effect of short term vitamin C therapy on mucociliary function. *Am. Rev. Respir. Dis.*, 141, A523.
Silver, M. R. (1998) BIPAP: useful new modality or confusing acronym? *Crit. Care Med.*, 26, 1473–1474.
Sim, J. (1986) Truthfulness in the therapeutic relationship. *Physiother. Pract.*, 2, 121–127.
Sim, J. (1995) The external validity of group comparative and single system studies. *Physiotherapy*, 81, 263–270.
Similowski, T. (1991) Contractile properties of the human diaphragm during chronic hyperinflation. *N. Engl. J. Med.*, 325, 917–923.
Simoes, E. A. F. (1999) Respiratory syncytial virus infection. *Lancet*, 354, 847–851.
Simmonds, P. (1999) Managing patients with lung cancer. *Br. Med. J.*, 319, 527–528.
Simonds, A. K. (1996) *Non-invasive Respiratory Support*, Chapman & Hall, London.
Simonds, A. K. (1998) Discharging the ventilator-dependent patient. *Br. J. Intensive Care*, 8, 47–51.
Simonds, A. K., Muir, J. F. and Pierson, D. J. (1996) *Pulmonary Rehabilitation*, BMJ Publications, London.
Simpson, F. G. (1993) Home nebulizers – do we know what we are doing? *ACPRC J.*, 22, 36–37.
Simpson, K., Killian, K. and McCartney, N. (1992) Randomised controlled trial of weight-lifting exercise in patients with chronic airflow limitation. *Thorax*, 47, 70–75.
Sims, S. E. R. (1987) Relaxation training as a technique for helping patients cope with cancer. *J. Adv. Nurs.*, 12, 583–591.
Sinclair, D. G. (1997) Gastrointestinal permeability following cardiopulmonary bypass. *Int. Care Med.*, 23, 510–516.

Singer, M. and Little, R. (1999) Cutting edge. *Br. Med. J.*, 319, 501–305.

Singer, M. and Webb, Q. (1997) *Oxford Handbook of Critical Care*, Oxford Medical Publications, Oxford.

Singer, M., Vermaat, J. and Hall, G. (1994) Hemodynamic effects of manual hyperinflation in critically ill mechanically ventilated patients. *Chest*, 106, 1182–1187.

Singh, S. J., Morgan, M. D. L., Hardman, A. E. and Rowe, C. (1994) Comparison of oxygen uptake during a conventional treadmill test and the shuttle walking test in chronic airflow limitation. *Eur. Respir. J.*, 7, 2016–2020.

Singh, V. (1990) Effect of yoga breathing exercises on airway reactivity in subjects with asthma. *Lancet*, 335, 1381–1383.

Sinha, S. (1998) Effect of nutritional status on vecuronium-induced neuromuscular blockade. *Anaesth. Intensive Care*, 26, 392–395.

Sisson, R. (1990) Effects of auditory stimuli on comatose patients with head injury. *Heart Lung*, 19, 373–378.

Sivakumar, D., Bosque, E. and Goldman, S. L. (1999) Bronchodilator delivered by metered dose inhaler and spacer. *Paediatr. Pulmonol.*, 27, 208–212.

Sivasothy, P., Smith, I. E. and Shneerson, J. M. (1998) Mask intermittent positive pressure ventilation in chronic hypercapnic respiratory failure due to COPD. *Eur. Respir. J.*, 11, 34–40.

Sjöland, H., Wiklund, I. and Caidahl, K. (1996) Improvement in quality of life and exercise capacity after coronary bypass surgery. *Arch. Intern. Med.*, 156, 265–271.

Sjöström, B., Haljamäe, H. and Dahlgren, L.-O. (1997) Assessment of postoperative pain. *Acta Anaesthesiol. Scand.*, 41, 339–344.

Sky, M. (1990) *Breathing*. Bear, New Mexico, p. 49.

Slavin, R. G. (1996) ABPA in CF. *Pediatr. Pulmonol.*, 21, 1–2.

Sliwa, J. A. and Marinko, M. S. (1996) Transcutaneous electrical nerve stimulator-induced ECG artifact. *Am. J. Phys. Med. Rehabil.*, 75, 307–309.

Sliwinski, P. (1994) The adequacy of oxygenation in COPD patients undergoing long-term oxygen therapy. *Eur. Respir. J.*, 7, 274–278.

Sloan, J. P., Muwanga, C. L. and Waters, E. A. (1986) Multiple rib fractures: TNS versus conventional analgesia. *J. Trauma*, 26, 1120–1122.

Slotman, G. J. (1998) Hyperbaric oxygen in systemic inflammation. *Crit. Care Med.*, 26, 1932.

Slutsky, A. S. (1993) Mechanical ventilation. *Respir. Care*, 38, 1389–1319.

Small, S. P. and Graydon, J. E. (1992) Perceived uncertainty, physical symptoms and negative mood in hospitalized patients with COPD. *Heart Lung*, 21, 568–574.

Smalldone, G. C. (1993) Regional impairment of mucociliary clearance in COPD. *Chest*, 103, 1390–1396.

Smedstad, K. G. (1992) Postoperative pain relief and hospital stay after total esophagectomy. *Clin. J. Pain*, 8, 149–153.

Smeltzer, S. C. (1996) Expiratory training in multiple sclerosis. *Arch. Phys. Med. Rehabil.*, 77, 909–912.

Smith, M. A., Mathewson, J. J. and Ulert, I. A. (1996) Contaminated stethoscopes revisited. *Arch. Intern. Med.*, 156, 82–84.

Smith, C. E. and Mayer, L. S. (1998) Continuous positive airway pressure. *Heart Lung*, 27, 99–108.

Smith, H. R. and Sinclair, D. G. (1996) Severe pulmonary dysfunction following acute respiratory distress syndrome. *Postgrad. Med. J.*, 72, 555–556.

Smith, S., Garnett, P. and Buckley, P. (1997) Delirium in the critically ill. *Care Crit. Ill*, 13, 175–178.

Smith, I. E., King, M. A. and Siklos, P. W. L. (1998) Treatment of ventilatory failure in the Prader–Willi syndrome. *Eur. Respir. J.*, 11, 1150–1152.

Smith, J. A., Redman, P. and Woodhead, M. A. (1999) Antibiotic use in patients admitted with exacerbations of COPD. *Eur. Respir. J.*, 13, 835–838.

Smithard, D. G. (1995) Dysphagia assessment after acute stroke. *Hosp. Update*, 21, 555–560.

Smy, J. (1995) How asthma drains the patient's purse. *Asthma J.*, 1, 8.

Snider, G. L. (1995) Withholding and withdrawing life-sustaining therapy. *Am. J. Respir. Crit. Care Med.*, 151, 279–281.

Sommers, M. S. (1991) Potential for injury: trauma after cardio-pulmonary resuscitation. *Heart Lung*, 20, 287–295.

Sonksen, J. (1998) Safer non-cardiac surgery for patients with coronary artery disease. *Br. Med. J.*, 317, 1400–1401.

Soryal, I., Sinclair, E., Hornby, J. (1992) Impaired joint mobility in Guillain–Barré syndrome. *J. Neurol. Neurosurg. Psychiat.*, 55, 1014–1017.

Sottile, F. D. (1986) Nosocomial pulmonary infection. *Crit. Care Med.*, 14, 265–270.

Sorahan, T. (1997) Childhood cancer and parental use of tobacco. *Br. J. Cancer*, 75, 134–138.

Soutar, A. (1997) Bronchial reactivity and dietary antioxidants. *Thorax*, 52, 166–170.
Souter, M. J., Andrews, P. J. D. and Pereirinha, M. R. (1999) Delayed intracranial hypertension. *Crit. Care Med.*, 27, 177–181.
Spahija, J. A. (1996) Effects of pursed-lips breathing and expiratory resistive loading in healthy subjects. *J. Appl. Physiol.*, 80(5), 1772–1784.
Sparsholt, M. (1989) Pain and the special care baby unit. *Nurs. Times*, 85(41), 61–64.
Speidel, B. D. (1978) Adverse effects of routine procedures on preterm infants. *Lancet*, i, 864–866.
Spence, D. P. S. (1991) Effect of nebulized salbutamol on walking distance. *Thorax*, 46, 305P.
Spooner, C. (2000) Nedocromil sodium in the treatment of exercise-induced asthma. *Eur. Resp. J.*, 16, 30–37.
Sporik, R. (1994) Why block a small hole? *Arch. Dis. Child.*, 71, 393–394.
Spring, C. (1999) The fibre-optic bronchoscope in the ICU. *Br. J. Intensive Care*, 9, 14–22.
Squier, H. C., Ries, A. L. and Kaplan, R. M. (1995) Quality of well-being predicts survival in lung transplantation candidates. *Am. J. Respir. Crit. Care Med.*, 152, 2032–2036.
Sridhar, M. K. (1995) Nutrition and lung health. *Br. Med. J.*, 310, 75–76.
Sridhar, K. S., Lobo, C. F. and Altman, R. D. (1998) Digital clubbing and lung cancer. *Chest*, 114, 1535–1537.
Stanescu, D. C. (1981) Pattern of breathing and ventilatory response to CO_2 in subjects practising hatha-yoga. *J. Appl. Physiol.*, 51, 1625–1629.
Stapleton, J. A., Lowin, A. and Russell, M. A. H. (1999) Prescription of transdermal nicotine patches for smoking cessation in general practice. *Lancet*, 354, 210–215.
Starke, I. D., Webber, B. A. and Branthwaite, M. A. (1979) IPPB and hypercapnia in respiratory failure. *Anaesthesia*, 34, 283–287.
Stauffer, J. L. (1999) Complications of endotracheal intubation and tracheotomy. *Respir. Care*, 44, 828–843.
Stead, C. E. (1999) Sudden infant death syndrome (SIDS) on the 'other side'. *Accident Emerg. Nurs.*, 7, 170–174.
Stein, R. T. (1999) Respiratory syncytial virus in early life and risk of wheeze and allergy by 13. *Lancet*, 354, 541–546.
Steiner, M. C., Singh, S. and Morgan, M. D. L. (2000) Stable COPD [letter]. *Br. Med. J.*, 320, 312.
Steinfort, C. L., Smith, M. J. and Harrison, N. K. (1987) Reactive arthritis in bronchiectasis. *Am. J. Respir. Dis.*, 135, A42.
Stephenson, J. (2000) Oxygen for preemies. *J. A. M. A.*, 283, .
Sterk, P. J. (1994) Improving asthma treatment. *Eur. Respir. J.*, 7, 220–222.
Sternbergh, W. C. (1998) Abdominal and thoracoabdominal aortic aneurysm. *Surg. Clin. North Am.*, 78, 827–843.
Steven, M. H., Pryor, J. A. and Webber, B. A. (1992) Physiotherapy versus cough alone in the treatment of cystic fibrosis. *NZ J. Physiother.*, 20, 31–37.
Stevensen, C. J. (1994) The psychophysiological effects of aromatherapy massage following cardiac surgery. *Complement. Ther. Med.*, 2, 27–35.
Stewart, T. D. and Rossier, A. B. (1978) Psychological considerations in the adjustment of spinal cord injury. *Rehabil. Lit.*, 39, 75–80.
Stewart, T. E. and Zhang, H. (1999) Nitric oxide in sepsis. *Respir. Care*, 44, 308–313.
Stiller, K. R. and Munday, R. M. (1992) Chest physiotherapy for the surgical patient. *Br. J. Surg.*, 79, 745–749.
Stiller, K., Montarello, J. and Wallace, M. (1994) Are breathing and coughing exercises necessary after coronary artery surgery? *Physiother. Theor. Pract.*, 10, 143–52.
Stiller, K., McInnes, M. and Huff, N. (1997) Do exercises prevent musculoskeletal complications after cardiac surgery? *Physiother. Theor. Pract.*, 13, 117–126.
Stolp-Smith, K. A. (1999) Antidepressant exacerbation of spasticity. *Arch. Phys. Med. Rehabil.*, 80, 339–342.
Stone, K. S. (1991) The effect of lung hyperinflation and endotracheal suctioning on cardiopulmonary haemodynamics. *Nurs. Res.*, 40, 76–80.
Stone, G., Huggon, A. M. and Nayeem, N. (1999) Telephone follow-up of relatives after sudden death. *Int. J. Clin. Pract.*, 53, 107–109.
Strachan, D. P. (1991) Ventilatory function and winter fresh fruit consumption. *Thorax*, 46, 624–629.
Stradling, J. R., Negus, T. W. and Smith, D. (1998) Mandibular advancement devices for the control of snoring. *Eur. Respir. J.*, 11, 447–450.
St-Laurent-Gagnon, T. (1999) Pain evaluation in preschool children and by their parents. *Acta Paediatr.*, 88, 422–427.
Strijbos, J. H. (1996) A comparison between an outpatient pulmonary rehabilitation program and

a home-care pulmonary rehabilitation programme. *Chest*, 109, 366–372.
Strollo, P. J. (1998) Sleep-disordered breathing. *Respir. Care*, 43, 421–428.
Suarez, J. I. (1999) Narcotics for brain-injured patients? *Crit. Care Med.*, 27, 362–363.
Suda, T. (1995) Hypersensitivity pneumonitis associated with home ultrasonic humidifiers. *Chest*, 107, 711–717.
Suman, O. E. (1995) Airway obstruction during exercise in asthma. *Am. J. Respir. Crit. Care Med.*, 152, 24–31.
Sun, W.-Z. (1992) Successful cardiopulmonary resuscitation in the prone position. *Am. Rev. Respir. Dis.*, 77, 202–204.
Sutcliffe, A. J. (1993) Pain relief for acutely ill and injured patients. *Care Crit. Ill*, 9, 266–269.
Sutton, P. P., Lopez, M. T. and Pavia, D. (1985) Assessment of percussion, vibratory shaking and breathing exercises in chest physiotherapy. *Eur. J. Respir. Dis.*, 66, 147–152.
Sutton, P. P., Gemmell, H. G. and Innes, N. (1988) Use of nebulized saline and nebulized terbutaline as an adjunct to chest physiotherapy. *Thorax*, 43, 57–60.
Suzuki, S. and Sato, M. (1995) Expiratory muscle training and sensation of respiratory effort during exercise in normal subjects. *Thorax*, 50, 366–370.
Swann, P. (1989) Stress management for pain control. *Physiotherapy*, 75, 295–298.
Swanston, M. (1993) Pain assessment with interactive computer animation. *Pain*, 53, 347–351.
Swartz, K. (1996) A national survey of endotracheal suctioning techniques in the pediatric population. *Heart Lung*, 25, 52–60.
Swerts, P. M. J. (1990) Exercise reconditioning in the rehabilitation of patients with COPD. *Arch. Phys. Med. Rehabil.*, 71, 570–573.
Swingle, H. M., Eggert, L. D. and Bucciarelli, R. L. (1984) New approach to management of unilateral tension pulmonary interstitial emphysema in premature infants. *Pediatrics*, 74, 354–357.
Sykes, K., Young, J. D. (1999) *Respiratory Support*, BMJ Publishing, London.
Szasz, T. S. (1968) The psychology of persistent pain, in *Pain*, (ed. A. Soulairac), Academic Press, London.
Taggert, J. A. (1994) Evaluating unplanned endotracheal extubations. *Dimens. Crit. Care Nurs.*, 13, 114–121.
Takeda, S. (1997) The effect of nasal CPAP on plasma endothelin-1 concentrations in patients with severe cardiogenic pulmonary oedema. *Anesth. Analg.*, 84, 1091–1096.
Tamaoki, J. (1992) Effect of indomethacin on bronchorrhea. *Am. Rev. Respir. Dis.*, 145, 548–552.
Tamaoki, J. (1994) Effect of long term treatment with oxitropium bromide on airway secretion in chronic bronchitis. *Thorax*, 49, 545–548.
Tamer, M. A. (1970) Hyponatremia secondary to ultrasonic aerosol therapy in the newborn. *J. Pediatr.*, 77, 1051–1054.
Tan, A. K. W. (1995) Incentive spirometry for tracheostomy and laryngectomy patients. *J. Otolaryngol.*, 24, 292–295.
Tang, W. (1997) Influence of respiratory distress syndrome on body composition after preterm birth. *Arch. Dis. Child.*, 77, F28–F31.
Tangredi, M. (1998) Septic shock. *Am. J. Nurs.*, 98(3), 46–49.
Tannenbaum E. L. and Davids L. M. (1995) Cystic fibrosis – approaches to management. *S. Afr. J. Physiother.*, 51, 2, 27–30.
Tantucci, C., Duguet, A. and Similowski, T. (1998) Effect of salbutamol on dynamic hyperinflation in COPD. *Eur. Respir. J.*, 12, 799–804.
Tariq, M. (1999) A foreign body in the bronchus still presents problems. *Int. J. Clin. Pract.*, 53(1), 81–82.
Tasaka, S., Kanazawa, M. and Mori, M. (1995) Long-term course of bronchiectais and bronchiolitis obliterans as late complication of smoke inhalation. *Respiration*, 62, 40–42.
Tashkin, D. P., Simmons, M. S., Chang, P. (1993) Effects of smoked substance abuse on nonspecific airway hyperresponsiveness. *Am. Rev. Respir. Dis.*, 147, 97–103.
Tattersall, R., Ledson, M. J. and Convery, R. P. (1997) The effect of posture and physiotherapy on reflux in adult CF patients. *Thorax*, 52(suppl. 6), A43.
Taylor, D. R. (2000) Interactions between corticosteroids and β_2 agonists. *Thorax*, 55, 595–602.
Taylor, K. M. (1998) Brain damage during cardiopulmonary bypass. *Ann. Thorac. Surg.*, 65, S20–S26.
Teague, W. G. (1997) Pediatric application of noninvasive ventilation. *Respir. Care*, 42, 414–422.
Teasdale, K. (1995) Theoretical and practical considerations on the use of reassurance in the nursing management of anxious patients. *J. Adv. Nurs.*, 22, 79–86.

Tector, A. J., Kress, D. C. and Downey, F. X. (1998) Transition from cardiopulmonary bypass to the HeartMate LVAD. *Ann. Thorac. Surg.*, 65, 643–646.

Ten Hove, R. (2001), quoting Pearson, *On-Call Conference*, Nottingham.

Thistlethwaite, J. E. (1998) Respiratory tract infections. *Update*, 56, 108–112.

Thomas, E. A. (1987) Pre-operative fasting – a question of routine? *Nurs. Times*, 83(49), 46–47.

Thomas, V. (1995) Psychological characteristics and the effectiveness of patient-controlled analgesia. *Br. J. Anaesth.*, 74, 271–276.

Thomas, H. M. (1996b) Pulmonary rehabilitation. *Chest*, 109, 299–300.

Thomas, G. A. O. (1996a) Nicotine and gastrointestinal disease. *Q. J. Med.*, 89, 485–488.

Thomas, G. (1997) Use of the prone position. *Care Crit. Ill*, 13, 96–100.

Thomas, G. A. O. (2000) Role of smoking in inflammatory bowel disease. *Postgrad. Med. J.*, 76, 273–279.

Thomas, E. O. and Manara, A. R. (1998) A review of percutaneous tracheostomy. *Br. J. Intensive Care*, 8, 122–129.

Thomas, J., Cook, D. J. and Brooks, D. (1995) Chest physical therapy management of patients with CF. *Am. J. Respir. Crit. Care Med.*, 151, 646–650.

Thomas, S., Andrews, A. M. and Hay, N. P. (1999) The anti-microbial activity of maggot secretions. *J. Tissue Viability*, 9, 127–131.

Thomsen, O. O. (1993) What do gastroenterologists in Europe tell cancer patients? *Lancet*, 341, 473–476.

Thomson, A. (1997) The role of negative pressure ventilation. *Arch. Dis. Child.*, 77, 454–458.

Thomson, D., Hassenkamp, A.-M. and Mansbridge, C. (1997) The measurement of empathy in a clinical and a non-clinical setting. *Physiotherapy*, 83, 173–180.

Thomson, L. R. (1973) Sensory deprivation: a personal experience. *Am. J. Nurs.*, 73, 266–268.

Thorax (1997) Asthma in adults and schoolchildren. *Thorax*, 52(suppl. 1) S2–S8.

Thoresen, M. (1999) Protecting the brain. *Semin. Neonatol.*, 5, 1–2.

Tidwell, S. L. (1990) Effects of position changes on mixed venous oxygen saturation in patients after coronary revascularization. *Heart Lung*, 19, 574–578.

Tiep, B. L. (1991) Reversing disability of irreversible lung disease. *W. J. Med.*, 154, 591–597.

Tierney, A. J. (1995) Undernutrition and elderly hospital patients. *J. Adv. Nurs.*, 23, 228–236.

Tillett, J. M. (1993) Effect of continuous rotational therapy on ICP in the severely brain-injured patient. *Crit. Care Med.*, 21, 1005–1011.

Timmons, B. H. and Ley, R. (1994) *Behavioral and Psychological Approaches to Breathing Disorders*, Plenum Press, New York.

Tirimanna, P. R. S. (1996) Evaluation of the suitability of weekly peak expiratory flow rate measurements. *Br. J. Gen. Pract.*, 46, 15–18.

Tobias, J. D. (1994) Pain management and sedation in the pediatric ICU. *Pediatr. Clin. North Am.*, 41, 1269–1280.

Tobias, J. D. (1997) Noninvasive monitoring of CO_2 during respiratory failure in toddlers and infants. *Anesth. Analg.*, 85, 55–58.

Tobin, M. J. (1988) Respiratory muscles in disease. *Clin. Chest Med.*, 9, 263–286.

Tobin, M. J. (1990) Dyspnea. *Arch. Intern. Med.*, 150, 1604–1611.

Tobin, M. J. (1991) *Respiratory Monitoring*, Churchill Livingstone, Edinburgh.

Tobin, M. J. and Yang, J. (1990) Weaning from mechanical ventilation. *Crit. Care Clin.*, 6, 725–746.

Todd, D. B. (1996) Prescription of exercise. *Update*, 52, 408–411.

Todisco, T. (1995) Effect of bamiphylline on tracheobronchial mucus clearance. *Respiration*, 62, 16–20.

Todisco, T. (1996) NDYAG laser therapy using a fiberoptic bronchoscope. *Eur. Respir. J.*, 9(suppl. 23), 375S.

Tolep, K. (1996) Swallowing dysfunction in patients receiving prolonged mechanical ventilation. *Chest*, 109, 167–172.

Torres, A. (1992) Pulmonary aspiration of gastric contents in patients receiving mechanical ventilation. *Ann. Intern. Med.*, 116, 540–543.

Torrington, K., Sorenson, D. and Sherwood, L. (1984) Postoperative chest percussion with postural drainage in obese patients following gastric stapling. *Chest*, 86, 891–895.

Tougaard, L., Krone, T. and Sorknaes, A. (1992) Economic benefits of teaching patients with COPD about their illness. *Lancet*, 339, 1517–1520.

Touw van de, T., Mudaliar, Y. and Nayyar, V. (1998) Cardiorespiratory effects of manually compressing

the rib cage during tidal expiration in acute severe asthma. *Crit. Care Med.*, 26, 1361–1367.
Tonnesen, P. (1999) Higher dosage nicotine patches increase one-year smoking cessation rates. *Eur. Respir. J.*, 13, 238–246.
Tovar, E. A., Roethe, R. A. and Weissig, M. D. (1998) One-day admission for lung lobectomy. *Ann. Thorac. Surg.*, 65, 803–806.
Trochtenberg, S. (1994) Nebulized lidocaine in refractory cough. *Chest*, 105, 1592–1593.
Trooster, T. (1997) Peripheral muscle weakness and deconditioning commonly occur in patients with HVS. *Eur. Respir. J.*, 10, 72s.
Trudel, G., Uhthoff, H. K. and Brown, M. (1999) Extent and direction of joint motion limitation after prolonged immobility. *Arch. Phys. Med. Rehabil.*, 80, 1542–1547.
Tsang, K. W. T., Ho, P. I. and Chan, K. N. (1999) A pilot study of low-dose erythromycin in bronchiectasis. *Eur. Respir. J.*, 13, 361–364.
Tschopp, J. M. (2000) Treatment of spontaneous pneumothorax. *Respiration*, 67, 108–111.
Tsubota, N., Yanagawa, M. and Yoshimura, M. (1994) The superiority of exercise testing over spirometry in the evaluation of postoperative lung function for patients with pulmonary disease. *Surg. Today*, 24, 103–105.
Tucker, E. Z. (1991) Tracheostomy speaking valve in pediatric ventilator-assisted patients. *Respir. Care*, 36, 1271.
Turner, R. E. (1997) NPPV: Face versus interface. *Respir. Care*, 42, 389–394.
Turnock, C. (1997) ICU syndrome. *Br. J. Intensive Care*, 7, 144–147.
Tweeddale, P. M., Rowbottom, I. and McHardy, G. J. R. (1994) Breathing retraining. *J. Psychosom. Res.*, 38, 11–21.
Twigley, A. J. and Hillman, K. M. (1985) The end of the crystalloid era? *Anaesthesia*, 40, 860–871.
Twycross, A. (1998) Perceptions about children's pain experience. *Prof. Nurse*, 13, 822–826.
Twycross, R. and Lack, S. (1984) *Therapeutics in Terminal Cancer*, Pitman, Bath.
Twycross, R. and Lack, S. (1990) *Oral Morphine in Advanced Cancer*, Beaconsfield Ltd, Beaconsfield.
Ulbricht, G. F. (1986) Laryngectomy rehabilitation: a woman's viewpoint. *Women Health*, 11, 131–136.
Ulrich, R. S. (1984) View through a window may influence recovery from surgery. *Science*, 224, 420–421.
Ungeman-deMent, P., Bemis, A. and Siebens, A. (1986) Exercise program for patients after cardiac surgery. *Arch. Phys. Med. Rehabil.*, 67, 463–466.
Urbaitis, J. C. (1999) Psychiatric treatment helps survivors of suicide attempts. *Crit. Care Med.*, 27, 458–459.
Valacio, R. and Lie, M. (1994) Diuretics and ACE inhibitors. *Care Elderly*, 6, 294–296.
Valenzuela, R. C. and Rosen, D. A. (1999) Topical lidocaine–prilocaine cream (EMLA) for thoracostomy tube removal. *Anesth. Analg.*, 88, 1107–1108.
Valji, A. M. (1998) Postpneumonectomy syndrome. *Chest*, 114, 1766–1769.
Valladares, P. (1996) Comparing clinical management of overdoses. *Emerg. Nurse*, 4, 6–8.
Van der Woning, M. (1997) Should relatives be invited to witness a resuscitation attempt? *Accident Emerg. Nurs.*, 5, 215–18.
Van Dixhoorn, J. (1986) Behavioral characteristics predisposing to hyperventilation complaints. *Gedrag Gezondh.*, 14, 169–174.
Van Dixhoorn, J., Duivenvoordent, H. J. and Staal, J. A. (1987) Cardiac events after myocardial infarction: possible effect of relaxation therapy. *Eur. Heart J.*, 8, 1210–1214.
Van Doorn, P. (1982) Control of the end-tidal PCO_2 in the hyperventilation syndrome. *Bull. Eur. Physiopathol. Res.*, 18, 829–836.
Vansteenkiste, J., Rochette, F. and Demedts, ? (1991) Diagnostic tests of hyperventilation syndrome. *Eur. Respir. J.*, 4, 393–399.
Varoli, F. (1998) Endoscopic treatment of bronchopleural fistulas. *Ann. Thorac. Surg.*, 65, 807–809.
Vaughan, R. S., Menke, J. A. and Giacoia, G. P. (1978) Pneumothorax: a complication of endotracheal tube suctioning. *J. Pediatr.*, 92, 633–634.
Veall, G. R. Q. (1995) Prolonged starvation in paediatric surgery. *Anaesthesia*, 50, 458–460.
Venables, T. (1994) Helping patients who want to quit smoking. *Respir. Dis. Pract.*, 11(2), 6–8.
Verboon, J. M. L., Bakker, W. and Sterk, P. J. (1986) The value of the forced expiration technique with and without postural drainage. *Eur. J. Respir. Dis.*, 69, 169–174.
Verbrugge, S. J. C. (1998) Lung overinflation without PEEP promotes bacteremia. *Intensive Care Med.*, 24, 172–177.
Verklan, M. T. (1997) Bronchopulmonary dysplasia. *Neonat. Netw.*, 16(8), 5–11.
Verra, F. (1995) Ciliary abnormalities in bronchial

epithelium of smokers. *Am. J. Respir. Crit. Care Med.*, **151**, 630–634.
Vilar, F. J. (1998) Management of *Pneumocystis carinii* pneumonia in intensive care. *Br. J. Intensive Care*, 8, 53–60.
Villaire, M. (1995) ICU from the patient's point of view. *Crit. Care Nurse*, **15**, 80–87.
Vitacca, M. (1998) Acute effects of deep diaphragmatic breathing in COPD patients. *Eur. Respir. J.*, **11**, 408–415.
Vos, H. R. (1993) Making headway with intracranial hypertension. *Am. J. Nurs.*, 93, 28–36.
Vos, P. J. E., Folgering, H. Th. M. and van Herwaarden, C. L. A. (1995) Predictors for nocturnal hypoxaemia in normoxic and mildly hypoxic patients with COPD. *Eur. Respir. J.*, 8, 74–77.
Wagner, P. D. (1992) Ventilation-perfusion matching during exercise. *Chest*, **101**, 192S–198S.
Wahba, R. W. M. (1991) Perioperative functional residual capacity. *Can. J. Anaesth.*, 38, 384–400.
Wainright, S. P. and Gould, D. (1996) Endotracheal suction. *J. Clin. Nurs.*, 5, 389–398.
Wald, S. L. (1993) The effect of secondary insults on mortality and disability after severe head injury. *J. Trauma*, 34, 377–381.
Waldmann, C. S. (1998) Management of head injury in a district general hospital. *Care Crit. Ill*, 14, 65–69.
Waldmann, C. and Gaine, M. (1996) The intensive care follow-up clinic. *Care Crit. Ill*, 12, 118–121.
Walker, A. (1995) Patient compliance and the placebo effect. *Physiotherapy*, 81, 120–126.
Walker, P. (1998a) Baclofen, a treatment for chronic hiccup. *J. Pain Symptom Manage.*, **16**, 125–132.
Walker, M. (1998b) Protection of the cervical spine in the unconscious patient. *Care Crit. Ill*, 14, 4–7.
Waller, D. A. (1999) A physiological comparison of flutter valve drainage bags and underwater seal systems for postoperative air leaks. *Thorax*, **54**, 442–443.
Walsh, J. M., Vanderwarf, C. and Hoscheit, D. (1989) Unsuspected hemodynamic alterations during endotracheal suctioning. *Chest*, 95, 162–165.
Wang, B., Tang, J. and White, P. F. (1997) Effect of the intensity of transcutaneous acupoint electrical stimulation. *Anesth. Analg.*, 85, 406–413.
Warren, C. P. W. (1980) Pulmonary disorders and physiotherapy in patients who undergo cholecystectomy. *Can. J. Surg.*, 23, 384–386.
Watcha, M. F. (1996) Nausea and vomiting. *Curr. Opin. Anaesthesiol.*, 9, 300–305.
Watkins, C. A. (1999) Mechanical and neurophysiological changes in spastic muscles. *Physiotherapy*, 85, 603–609.
Watson, J. M. (1997) Does influenza immunisation cause exacerbations of chronic airflow obstruction or asthma? *Thorax*, 52, 190–194.
Watson, M. J., Horn, S. A. and Wilson, B. A. (1999) Assessing a minimally responsive brain injured person. *Br. J. Ther. Rehabil.*, 6, 436–441.
Webb, A. K. and Govan, J. R. W. (1998) Burkholderia cepacia: another twist and a further threat. *Thorax*, 53, 333–334.
Webb, R. K., Ralston, A. C. and Runciman, W. B. (1991) Potential errors in pulse oximetry. *Anaesthesia*, 46, 207–212.
Weber, ?. (1999) Effect of continuously nebulized ipratropium bromide plus albuterol on emergency department length of stay. *Chest*, **115**, 937–944.
Weber, D. J., Paasch, R. and Rutala, W. A. (1999) Nosocomial infections in the ICU. *Chest*, **115**, 34s–41s.
Wedzicha, J. A. (1986) Polycythaemia in hypoxic lung disease. *Respir. Dis. Pract.*, 3, 26–30.
Wehberg, E. K. (1996) Nitric oxide mediates fluid accumulation during cardiopulmonary bypass. *J. Thorac. Cardiovasc. Surg.*, **112**, 168–174.
Weill, D., Mack, M. J. and Tennison, D. (1998) Adverse effects of medications commonly administered to thoracic surgical patients. *Chest Surg. Clin. North Am.*, 8, 681–702.
Weiner, H. (1991) Stressful experience and cardiorespiratory disorders. *Circulation*, 83(suppl. II), II-2–II-8.
Weiner, P. (1995) Inspiratory muscle training during treatment with corticosteroids. *Chest*, **107**, 1041–1044.
Weiner, P., Azgad, Y. and Ganam, R. (1992) Inspiratory muscle training in patients with bronchial asthma. *Chest*, 102, 1357–1361.
Weiner, P., Berar, N. and Davidovich, A. (2000) The perception of dyspnoea in patients with asthma, before and after inhaled glucocorticosteroids. *Respir. Med.*, **94**, 161–165.
Weinstein, R. A. (1991) Epidemiology and control of nosocomial infections. *Am. J. Med.*, **91**, 179s–184s.
Weisberg, S. C. (2000) Pharmacotherapy of asthma in children. *Pediatr. Pulmonol.*, 29, 46–61.
Weissleder, A. (1976) Physical therapeutic approach to the control of an acute asthmatic attack. *Phys. Ther.*, 56, 681–683.
Weissman, C. (1993) Stressing the critically ill patient. *J. Crit. Care*, 8, 100–108.

Weissman, C., Kemper, M. and Damask, M. C. (1984) Effect of routine intensive care interactions on metabolic rate. *Chest*, 86, 815–818.

Welsby, I. and Mythen, M. G. (1997) Gut perfusion during cardiac surgery. *Curr. Opin. Anaesth.*, 10, 34–39.

Wencker, M., Banik, N. and Buhl, R. (1998) Long-term treatment of alpha1-antitrypsin deficiency-related pulmonary emphysema with human alpha1-antitrypsin. *Eur. Respir. J.*, 11, 428–433.

Wenger, N. K. (1982) Early ambulation. *Adv. Cardiol.*, 31, 138–141.

Werebe, E. C. (1999) Systemic distribution of talc after intrapleural administration in rats. *Chest*, 115, 190–193.

Wessel, D. L. (1993) Hemodynamic responses to perioperative pain and stress in infants. *Crit. Care Med.*, 21, S361–S362.

West, J. B. (1995) *Respiratory Physiology*, 5th edn, Williams & Wilkins, Baltimore, MD.

Westbrook, J. L. and Sykes, M. K. (1992) Peroperative arterial hypoxaemia. The interaction between intrapulmonary shunt and cardiac output. *Anaesthesia*, 47, 307–310.

Westgren, N. (1998) Quality of life and traumatic spinal cord injury. *Arch. Phys. Med. Rehabil.*, 79, 1433–1439.

Wetter, D. W. (1994) Smoking as a risk factor for sleep-disordered breathing. *Arch. Intern. Med.*, 154, 2219–2224.

Wheatley, J. R. (1990) The effect of hyperinflation on respiratory muscle work in acute induced asthma. *Eur. Respir. J.*, 3, 625–632.

White, D., Stiller, K. and Roney, F. (1999) The incidence and severity of symptoms of incontinence in adult CF patients. *WCPT Proceedings*, p. 35.

White, K. L. (1988) Foreword, in *Medicine and Culture*, (ed. L. Payer), Henry Holt, New York.

White, R. J. (1992) The diagnosis and initial management of head injury. *N. Engl. J. Med.*, 327, 1507–1511.

White, K. M., Winslow, E. H. and Clark, A. P. (1990) The physiologic basis for continuous mixed venous oxygen saturation monitoring. *Heart Lung*, 19, 548–551.

Whitehead, B. F. and Leval, M. R. (1994) Paediatric lung transplantation: the agony and the ecstasy. *Thorax*, 49, 437–439.

Whitney, J. D. (1989) Physiologic effects of tissue oxygenation on wound healing. *Heart Lung*, 18, 466–476.

Whittaker, J. A. (1990) The effects of refeeding on peripheral and respiratory muscle function. *Am. Rev. Respir. Dis.*, 142, 283–288.

WHO (1996) *The Global Burden of Disease*, Harvard School of Public Health, Cambridge, MA.

Wichmann, M. W., Ayala, A. and Chaudry, I. H. (1998) Severe depression of host immune functions following closed-bone fracture, soft-tissue trauma and hemorrhagic shock. *Crit. Care Med.*, 26, 1372–1378.

Widmer, S., Conway, A., Cohen, S. and Davies, P. (1997) Hyperventilation: a correlate and predictor of debilitating performance anxiety in musicians. *Med. Probl. Performing Artists*, 12, 97–106.

Wiener, C. M., McKenna, W. J. and Myers, M. J. (1990) Left lower lobe ventilation is reduced in patients with cardiomegaly in the supine but not the prone position. *Am. Rev. Respir. Dis.*, 141, 150–155.

Wiener, J. P. (1992) Postoperative pleural and pulmonary abnormalities in patients undergoing CABG. *Chest*, 102, 1313–1314.

Wiener, M. D., Garay, S. M. and Leitman, B. S. (1991) Imaging the ICU patient. *Clin. Chest Med.*, 12, 169–198.

Wiest, G. H., Lehnert, G. and Bruck, W. M. (1999) A heated humidifier reduces upper airway dryness during CPAP. *Respir. Med.*, 93, 21–26.

Wietasch, G. J. K., Mielck, F. and Scholz, M. (2000) Bedside assessment of cerebral blood flow by double-indicator dilution technique. *Anesthesiology*, 92, 367–375.

Wiggins, J. (1991) Steroid trials in the assessment of air flow limitation. *Respir. Med.*, 85, 295–299.

Wijkstra, P. J. (1994) Relation of lung function, maximal inspiratory pressure, dyspnoea and quality of life with exercise capacity in patients with COPD. *Thorax*, 49, 468–472.

Wildhaber, J. H. (1996) Effect of electrostatic charge, flow, delay and multiple actuations on the in vitro delivery of salbutamol. *Thorax*, 51, 985–988.

Wiles, F. W. and Hnizdo, E. (1991) Relevance of airflow obstruction and mucus hypersecretion to mortality. *Respir. Med.*, 85, 27–35.

Wilkes, L. (1983) Nightmare, in *The Oxford Book of Death*, Oxford University Press, Oxford.

Wilkie, P. (1997) Teaching medical students in general practice. *Student Br. Med. J.*, 5, 445.

Wilkins, R. L., Krider, S. J. and Sheldon, R. L. (1995)

Clinical Assessment in Respiratory Care, Mosby, Toronto.
Wilkinson, S. (1996) Get the massage. *Nurs. Times*, 92(34), 61–64.
Williams, J. (2000) Audit of hyperventilation syndrome. *ACPRC Conference*, poster.
Williams, S. J. (1993) *Chronic Respiratory Illness*, Routledge, London.
Williams, M. T. (1995) Chest physiotherapy and cystic fibrosis. *Chest*, 106, 1872–1882.
Williams, S. J. (1993) *Chronic Respiratory Illness*, Routledge, London.
Williams, G. C. and Deci, E. L. (1998) The importance of supporting autonomy in medical education. *Ann. Intern. Med.*, 129, 303–308.
Williams, A. R., Hind, M. and Sweeney, B. P. (1994) The incidence and severity of postoperative nausea and vomiting in patients exposed to positive intra-operative suggestions. *Anaesthesia*, 49, 340–342.
Williamson, I. J. (1993) Role of oxygen during recovery from exercise in patients with COPD. *Thorax*, 48, 1056.
Willis, J. (1996) Flotation beds. *Nurs. Times*, 92, 45–48.
Wills, P. J. and Cole, P. J. (1996) Mucolytic and mucokinetic therapy. *Pulm. Pharmacol.*, 9, 197–204.
Wills, P. J., Hall, R. L. and Chan, W. (1997) Sodium chloride increases the ciliary transportability of CF and bronchiectasis sputum. *J. Clin. Invest.*, 99, 9–13.
Willson, D. F. (1998) Surfactant in pediatric respiratory failure. *Respir. Care*, 43, 1070–1085.
Wilson, A. E. (1996) Effect of backrest position on hemodynamic and right ventricular measurements. *Am. J. Crit. Care*, 5, 264–270.
Wilson, D. C. (1992) Respiratory problems in the newborn. *Br. J. Intensive Care.*, 2, 287–294.
Wilson, R. (1988) Secondary ciliary dysfunction. *Clin. Sci.*, 75, 113–120.
Wilson, R. (1999) Bacterial infection and COPD. *Eur. Respir. J.*, 13, 233–235.
Wilson, R. C. and Jones, P. W. (1989) A comparison of the visual analogue scale and modified Borg scale for the measurement of dyspnoea during exercise. *Clin. Sci.*, 76, 277–282.
Wilson-Davis, S. L. (1997) Air entry in infant resuscitation: oral or nasal routes? *J. Appl. Physiol.*, 82, 152–155.
Winer, J. B. (1994) Diagnosis and treatment of Guillain–Barré syndrome. *Care Crit. Ill*, 10, 23–25.
Winslow, E. H. (1995) Dangling: a review of relevant physiology, research and practice. *Heart Lung*, 24, 263–272.
Wiselka, M. (1994) Influenza. *Br. Med. J.*, 308, 1341–1345.
Wissing, D. R. (1988) Use of respiratory care procedures in the management of hospitalized asthmatics. *Ann. Allerg.*, 61, 407–420.
Wolf, J., Holcombe, E. and Grier, M. D. (2000) Symptoms and suffering at the end of life in children with cancer. *N. Engl. J. Med.*, 342, 326–333.
Wolf, S. L., Coogler, C. and Xu, T. (1997) Exploring the basis for Tai Chi Chuan as a therapeutic exercise approach. *Arch. Phys. Med. Rehabil.*, 78, 886–892.
Wollam, P. J. (1994) Prediction and assessment of bronchodilator response in mechanically ventilated patients. *Respir. Care*, 39, 730–735.
Wollmer, P., Ursing, K. and Midgren, B. (1985) Inefficiency of chest percussion in the physical therapy of chronic bronchitis. *Eur. J. Respir. Dis.*, 66, 233–239.
Wolter, J. M. (1997) Home intravenous therapy in CF. *Eur. Respir. J.*, 10, 896–900.
Wong, F. C. (1994) Transcranial doppler monitoring. *Intens. Care Britain*, Greycoat, Harley St, London, 66–69.
Wong, W. P. (1998) Acute respiratory distress syndrome. *Physiotherapy*, 84, 439–450.
Wong, W. P. (1999) Use of body positioning in the mechanically ventilated patient with acute respiratory failure. *Physiother. Theor. Pract.*, 15, 25–41.
Wong, W. P. (2000) Physical therapy for a patient in acute respiratory failure. *Phys. Ther.*, 80, 662–670.
Wong, D. H. and Shier, M. E. F. (1997) Chronic obstructive pulmonary disease and postoperative pulmonary complications. *Curr. Opin. Anaesth.* 10, 254–258.
Wong, C. A., Cole, A. A. and Watson, L. (1996) Pulmonary function before and after anterior spinal surgery in adult idiopathic scoliosis. *Thorax*, 51, 534–536.
Wood, A. M. (1993) A review of literature relating to sleep in hospital. *Intens. Crit. Care Nurs.*, 9, 129–136.
Wood, M. K. and Spiro, S. G. (2000) Pulmonary embolism. *Hosp. Med.*, 61, 46–50.
Woodward, S. (1996) Nutritional support for head-injured patients. *Prof. Nurse*, 11, 290–292.

Worcester, M. C., Hare, D. L. and Oliver, R. G. (1993) Early programmes of high and low intensity exercise and quality of life after acute myocardial infarction. *Br. Med. J.*, 307, 1244–1247.

Wren, W. S. (1989) Anaesthetic technique in the newborn. *Intensive Care Med.*, **15**, S46–S49.

Wright, A. L. (1989) Breast feeding and lower respiratory tract illness. *Br. Med. J.*, 299, 946–949.

Wright, E. C. (1993) Non-compliance – or how many aunts has Matilda? *Lancet*, 342, 909–913.

Wu, C. L., Jani, N. D. and Perkins, F. M. (1999) Thoracic epidural analgesia versus intravenous patient-controlled analgesia for the treatment of rib fracture after motor vehicle crash. *J. Trauma*, 47, 564–567.

Wuyam, B., Payen, J. F. and Levy, P. (1992) Metabolism and aerobic capacity of skeletal muscle in chronic respiratory failure related to COPD. *Eur. Respir. J.*, 5, 157–162.

Wyncoll, D. L. A. and Evans, T. W. (1999) Acute respiratory distress syndrome. *Lancet*, 354, 497–501.

Wysocki, M. (1999) Noninvasive ventilation in acute cardiogenic pulmonary edema: better than CPAP? *Intensive Care Med.*, 25, 1–2.

Yamamoto, H. (2000) Long-term effects of nasal CPAP on daytime sleepiness, mood and traffic accidents in patients with OSA. *Respir. Med.*, 94, 87–90.

Yang, Q.-H. (1991) Effect of lung inflation on regional lung expansion in supine and prone rabbits. *J. Appl. Physiol.*, 71, 76–82.

Yaster, M. (1995) Pain relief. *Pediatrics*, 95, 427–428.

Yaster, M., Tobin, J. R. and Billett, C. (1994) Epidural analgesia for severe vaso-occlusive sickle cell crisis. *Pediatrics*, 93, 310–315.

Yellon, R. F., Coticchia, J. and Dixit, S. (2000) Esophageal biopsy for the diagnosis of GOR in children. *Am. J. Med.*, 108(4A), 131S–138S.

Yellowlees, P. M. (1989) Psychological defences and coping styles in patients following a life-threatening attack of asthma. *Chest*, 95, 1298–1303.

Yildiz, F. (2000) Inhaled corticosteroids may reduce neutrophilic inflammation in patients with stable COPD. *Respiration*, 67, 71–76.

Young, I. H. (1989) Effect of low dose nebulized morphine on exercise endurance. *Thorax*, 44, 387–390.

Young, J. (1998) Rehabilitation for older people. *Br. Med. J.*, **316**, 1108–1109.

Young, S., Bitsakou, H. and Caric, D. (1991) Coughing can relieve or exacerbate symptoms in asthmatic patients. *Respir. Med.*, 85(suppl. A), 7–12.

Yuksel, B. (1994) Comparison of the effects on lung function of two methods of bronchodilator administration. *Respir. Med.*, 88, 229–233.

Yule, W. (1999) Post-traumatic stress disorder. *Arch. Dis. Child.*, 80, 107–109.

Yung, B. (1996) Totally implantable venous access devices in CF. *Respir. Med.*, 90, 353–356.

Zadai, C. (1991) Cardiopulmonary ageing and exercise, in *Respiratory Care*, (ed. J. A. Pryor), Churchill Livingstone, Edinburgh, pp. 199–226.

Zalacain, R., Sobradillo, V. and Amilibia, J. (1999) Predisposing factors to bacterial colonization in COPD. *Eur. Respir. J.*, 13, 343–348.

Zapletal, A., Stefanova, J. and Horak, J. (1983) Chest physiotherapy and airway obstruction in patients with CF – a negative report. *Eur. J. Respir. Dis.*, 64, 426–433.

Zar, H. J., Brown, G. and Donson, H. (1999) Home-made spacers for bronchodilator therapy in children with acute asthma. *Lancet*, 354, 979–982.

Zenz, M. (1997) Pain therapy. *Curr. Opin. Anaesth.*, 10, 367–368.

Zhuang, J., Schmoker, J. D. and Shackford, S. R. (1992) Focal brain injury results in severe cerebral ischemia despite maintenance of cerebral perfusion pressure. *J. Trauma*, 33, 83–88.

Zideman, D. A. (1994) Guidelines for paediatric life support. *Br. Med. J.*, 308, 1349–1355.

Zidulka, A., Braidy, T. F. and Rizzi, M. C. (1982) Position may stop pneumothorax progression in dogs. *Am. Rev. Resp. Dis.*, 126, 51–53.

Ziegler, D. W. (1994) The morbidity and mortality of rib fractures. *J. Trauma*, 37, 975–979.

Zimmerman, M. D., Appadurai, K. and Scott, J. G. (1997) Survival. *Ann. Intern. Med.*, 127, 405–408

Zimmet, P. (2000) Globalization, coca-colonization and the chronic disease epidemic. *J. Int. Med.*, 247, 301–310.

INDEX

If it is not here, see the Glossary

Numbers in **bold** are for Figures and Boxes, numbers in *italic* are for Tables.