Physiotherapy in Respiratory and Cardiac Care

PART ONE – PHYSIOLOGY AND PATHOLOGY

Chapter 1  THE PHYSIOLOGICAL BASIS OF CLINICAL PRACTICE

Learning objectives

On completion of this chapter the reader should be able to:

- understand how the respiratory system defends the body against the elements
- recognize how the control of respiration adapts to different situations
- understand the mechanics of breathing and its relation to the work of breathing
- use clinical reasoning to relate ventilation and perfusion to clinical practice
- interpret arterial blood gases and apply them to a problem-based approach to treatment
- understand how the respiratory and cardiovascular systems contribute to oxygen delivery and its relationship to oxygen consumption
- recognize how the cardiorespiratory system responds to a variety of individual circumstances.

INTRODUCTION

*Breathing is a forbidden fruit on the tree of knowledge*  
Gilbert 1999a

Breathing is unique. Most of us give it little thought, yet it can be voluntary or automatic and is preserved in unconsciousness. It is associated with a respiratory system of remarkable ingenuity, being responsible for gas being pumped into the lungs, diffused into the circulation, transported around the body and passed into cells. It is also involved in speaking and laughing, balancing pH and controlling
metabolism.

An understanding of how this system works creates the foundation for evidence-based practice. This chapter covers not just the textbook lungs of hefty young healthy males but also those of people who may smoke or be stressed or overweight.

Cardiovascular physiology is in Chapter 4, and the ‘oxygen cascade’ section in this chapter brings the workings of the respiratory and cardiovascular systems together.

**DEFENCE**

*Imagine wearing your insides on the outside*

The lung is an ‘outdoor’ organ that has to interact with the environment while facilitating ventilation, and is the body’s primary route of infection (Waterer 2012). Every day, up to 500 million alveoli in the adult lungs (West 2013) allow a surface area the size of a tennis court to be exposed to a volume of air and pollutants that could fill a swimming pool (Hanley & Tyler 1987). It is only by means of a sophisticated biological barrier that the body does not succumb to this onslaught.

Defense against the outside world is based on a network of filters, reflexes, secretions and specialized cells. Physiotherapists treat patients whose defenses are breached when the nose is bypassed by mouth-breathing or an artificial airway, cilia are damaged by smoking or disease, and cough is inhibited by pain or weakness.

**Nose**

The nasal passages are the gatekeeper of the respiratory tract, providing the first line of defense by means of:

- sensing suspicious smells
- sneezing in response to irritating substances
- filtering large particles
- insulating against swings in temperature and humidity.
Mouth
The oral cavity, from the lips to the junction of the hard and soft palates, is home to over 700 species of microbe, whose purpose is to support the immune system (Gupta 2011). They are harmful only if they reach sites to which they do not normally have access, leading to most hospital-acquired pneumonias being caused by endotracheal tubes or nebulizers (Guggenbichler et al 2011).

Pharynx
The pathways for air and food converge in the pharynx. When a person chews, breathing continues through the nose, but during swallowing the pharynx can only deal with food and the airway is closed off.

The nasopharynx exposes inspired and expired gas to a large surface area of highly vascular, moist mucous membrane (Fig 1.1). This nasal mucosa delivers warmth and humidity to the inspiratory breath and recovers a third of it on the expiratory breath (Richards et al 1996).

The oropharynx extends from the oral cavity to the tonsils. The tonsils are lymphoid tissue which defend against foreign pathogens. Surgical removal of tonsils and adenoids renders children more vulnerable to passive smoking (Chen et al 1998).

The hypopharynx is responsible for the tricky process of swallowing, for which 56 muscles are required to ensure airway protection while giving food the right of way (Higashijima 2010).

The epiglottis is a leaf-like lid which snaps shut over the larynx during swallowing to prevent aspiration into the trachea. The functions of the larynx are primarily to elevate during swallowing in order to protect the airway, secondarily to stabilize the transition between inhalation and exhalation (p.8), and only as an afterthought to provide speech.

Cortical, subcortical and brainstem neural control centres help co-ordinate breathing and swallowing and ensure that swallowing is followed by exhalation, which further helps to prevent aspiration. This tight respiratory–swallowing coupling is compromised by neurological impairment or hypercapnia (Nishino
Airways

Inhaled irritant particles increase bronchoconstrictor tone in order to narrow the airways. This is normally protective, but becomes exaggerated and counterproductive in asthma, when it is called bronchospasm.

Other particles are trapped on a layer of sticky mucus lining the airways from the nasopharynx to the terminal bronchi. This mucous blanket is gripped from underneath by tiny hooks on the tips of hair-like cilia attached to the epithelium. These move the mucus up to the throat, from where it is swallowed or expectorated. This ‘mucociliary escalator’ can cleanse the lungs in 20 minutes, moving particles at an average 1-2 cm/min, most rapidly in the trachea and decreasing with each airway generation as the total cross section widens (Morris & Affi 2010, p.163).

The cilia beat in a ‘sol’ layer of watery fluid, reaching up to penetrate the ‘gel’ layer of mucus, hooking onto it with the onward stroke and diving beneath it into the sol layer on the recovery stroke at 20 beats a second (Fig 1.1). Balanced systemic hydration keeps the fluid in the sol layer the same height as the cilia. If the sol layer is too deep, or the cilia length shortened by smoking (Leopold 2013), the hooks cannot reach the mucus. If the sol layer is too shallow, as in cystic fibrosis, mucus clogs up the delicate cilia. Systemic hydration is also relevant to the gel layer because water constitutes 95% of respiratory mucus and helps maintain mucociliary clearance (Nakagawa et al 2004).

Fig 1.1
The humidifying effects of nose breathing on inspiration (left) and expiration (Fisher & Paykel)
Other protective functions of the mucus are insulation, antibacterial action, and preventing the patient from drying out (Button & Boucher 2008). Moisture in inspired air helps maintain optimum ciliary beat frequency and acts as a buffer against extremes of temperature because water requires four times more energy to change temperature than air (Williams et al 1996).

This finely co-ordinated mechanism is compromised by smoking, disease, age (Fig 1.2), immobility, hypoxia, inflammation, dehydration and prolonged coughing which narrows the airways (Wanner et al 1996).

Fig 1.2
Mucous velocity with different ages and conditions (Wanner et al 1996)
KEY POINT
Optimizing systemic hydration should be the first intervention for people with sputum retention

An extra function of the sol layer is its antimicrobial property, without which inhaled bacteria could double in number every 20 minutes. Failure to clear excess mucus may disable this chemical shield. An extra function of the gel layer is to engulf particles as well as carry them along on its surface (Knowles & Boucher 2002).

Airway epithelial cells contain pattern recognition receptors which warn downstream immune cells of approaching bacteria. Further protection is afforded by some interesting taste bud cells which have relocated to the airway and respond to noxious particles by stimulating the cilia (Kinnamon 2011).

Cough

A cough is only as effective as the deep breath preceding it

Sobush (2008)
'The cough is the body’s strongest physiological reflex and has been known to fracture brittle ribs, cause a pneumothorax (Khajotia 2012) and in extremis rupture the diaphragm (Reper et al 2012). Its protective function is to expel secretions and debris when mucociliary clearance is damaged, as in bronchiectasis, or overwhelmed, as with some chest infections. Like swallowing and belching, it is subject to higher cortical control, either as cough inhibition or a voluntary cough. A reflex cough occurs if irritants stimulate inflammatory, chemical, mechanical or thermal receptors. These are located in the pleura, upper airways and, unexpectedly, the external auditory canal (Polverino 2012), as those who have Arnold’s nerve cough reflex know when they clean their ears. In the airways, the receptors are most sensitive at the glottis and carina. Vagal afferents then transmit the messages to the brainstem, and the phrenic and spinal motor nerves transmit impulses to the respiratory musculature (Homnick 2007). Stimulation of the pharynx causes a gag rather than a cough.

A cough comprises:

• A deep inspiration to near total lung capacity
• Snapping shut of the glottis (which requires intact bulbar function)
• A short pause to allow distribution of air past secretions
• Sharp contraction of the expiratory muscles to create intrathoracic pressures of at least 100 mmHg (Levitzky 2007, p.219),
• Sudden opening of the epiglottis, exploding the trapped gas outwards at up to 800 km/hr (Polverino 2012).

The resulting shear force overcomes viscous, frictional and gravitational resistance so that secretions are cleared from the upper airway, while deeper secretions are squeezed from the lower airway (Pitts et al 2013). Coughing is accompanied by violent swings in pleural pressure, which cause dynamic airway compression (p.). This is initiated in the trachea during the cough and extends peripherally as lung volume decreases, ensuring that the full length of the tracheobronchial tree is involved. The airways normally re-open with a subsequent deep breath, but for people unable to take a deep breath, they stay closed for lengthy periods. Coughing may be inhibited by pain, and the mechanism is less efficient in some people with obstructive airways disease if they have poor expiratory flow or airways that collapse on expiration. It is weakened
with neurological disease or if the glottis is bypassed by intubation or tracheostomy.

Complications of coughing include bronchospasm, exhaustion and stress incontinence. Despite the high pressures, barotrauma is normally avoided by the support of the rib cage and by contraction of intercostal and abdominal muscles which buttress the chest wall and prevent overdistension of alveoli.

**Other lung defenses**

Pollutants which manage to reach the alveoli are met with scavenger macrophages and proteolytic enzymes which would be powerful enough to destroy the alveoli themselves except for the presence of an inhibitor from the liver called alpha₁-antitrypsin (AAT), lack of which causes AAT deficiency (Ch.3) and predisposes to HIV (Ferreira et al 2014). Pathogens which survive this still have to overcome a barrage of inflammation (Eisele & Anderson 2011). Asbestos particles circumvent all these defenses because of their peculiar shape.

The function of the wafer-thin alveolar-capillary membrane is to allow efficient gas exchange, but this also allows carbon monoxide and chemical warfare to cause their mischief.

The entire blood volume passes through the lungs, which help detoxify foreign substances that have made it into the circulation. The pulmonary circulation also performs a range of metabolic functions and acts as a filter to help protect the systemic 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 (Ch.16) include a filter to perform some of these functions.

**CONTROL**

*Breathing is the basic rhythm of life*  
Hippocrates

Breathing is normally controlled with such exquisite sensitivity that pH in the blood is maintained within precise limits despite unpredictable metabolic demands.
Carbondioxide (CO$_2$) and oxygen are also controlled, but less tightly.

Clusters of neurons in the pons and medulla receive and integrate stimuli from the rib cage, lungs, chemoreceptors and cortex. They then discharge impulses to the respiratory muscles, triggering contraction.

The respiratory centres also perceive and respond to posture, coughing, hiccupping, defaecating, stepping into a cold shower and the lactic acid produced by intense exercise (de Souza 2010). Respiratory control occurs at a subconscious level but can be overridden by breathing exercises and modified by emotion, pain and some pathological states. Chronic obstructive pulmonary disease (COPD), for example, shows less precise control over breathing during sleep and exercise (Dempsey 2002), and some COPD patients have an altered chemoreceptor response, becoming dependent on low oxygen (hypoxic respiratory drive) rather than the more usual high CO$_2$ (hypercapnic respiratory drive) as a stimulus to breathe (p.).

Yawning is controlled by the hypothalamus and is thought to relate to thermoregulation of the brain. The open mouth promotes pharyngeal cooling and the deep inspiration increases flow down the internal jugular vein. Boredom and the evening are associated with higher brain temperature. Contagious yawning appears to have evolved in order to coordinate arousal and vigilance in budgerigars and some mammals (Gallup & Eldakar 2012).

MECHANICS

When you can’t breathe, nothing else matters.

Patient

The respiratory muscles

The lungs hang from whichever part of the chest wall is uppermost at the time and are attached to each other medially by their roots, but do not directly touch any muscle. The chest wall comprises the rib cage and diaphragm, which are expandable. The diaphragm contracts downwards against the abdominal contents, which are themselves incompressible (but push out the abdominal wall), and the pelvic bowl, which is rigid. The respiratory muscles extend from the mastoid
process, tongue and nose (Lalley 2013) to the pubic symphysis, and are the only skeletal muscles vital to life, providing the power for the respiratory pump and delivering oxygen to the distal airways while removing CO₂.

**Inspiration**

The diaphragm, the seat of the soul according to the ancient Greeks, is a dome-shaped sheet of muscle on which the upright lungs rest, separated by the pleura. It is innervated from C3-5 via the phrenic nerves and generates 70% of tidal volume. Attached to the bottom of the rib cage, it separates two compartments of markedly different densities, the thorax and abdomen (Mangera et al 2012).

At rest, or if paralysed, the diaphragm extends upwards almost to nipple level (see Fig 19.8). Contraction flattens it, pressing down against the fulcrum of the abdominal contents, displacing the abdominal viscera by 5-7 cm, protruding the abdominal wall (unless prevented voluntarily or by tight clothing) and levering the lower rib cage upwards and outwards in a bucket handle action, causing expansion of the lower chest. This displacement downwards and outwards creates negative intrathoracic pressure which sucks air into the lungs.

Muscles assisting this process are:

- the external intercostals which stabilize the chest wall so that diaphragmatic contraction can create these pressure changes
- the scalenes which stabilize the upper rib cage to prevent it being pulled downwards
- pharyngeal muscles which prevent collapse of the upper airway from the negative pressure.

These and other accessory muscles become major inspiratory muscles when there is increased work of breathing (WOB) such as with airflow obstruction or exercise.

During upper limb activity, intercostal and accessory muscles are obliged to stabilize the torso, forcing the diaphragm to take a greater load. People with COPD may find daily activities daunting because of the sustained arm movements required, especially in standing when the diaphragm also contributes to postural stability.

*The diaphragm’s role in core stability is by controlling intra-abdominal pressure and reducing the stress on the spine through cooperative action with the*
abdominal and pelvic floor muscles (Noh et al 2014). The postural functions of the respiratory muscles have led to links between increased WOB and impaired balance (David et al 2012) and between diaphragm fatigue and recurrent back pain (Janssens et al 2013).

**Expiration**

The transition between inhalation and exhalation is smoothed by a brake on expiratory flow caused by airflow resistance, especially at the larynx, and by continued low-grade diaphragmatic activity. When the larynx is bypassed by intubation, positive end-expiratory pressure (PEEP) is applied to the airway to support this function, thus avoiding fatigue of the diaphragm during exhalation. This function of the larynx is called ‘physiological PEEP’.

Normal exhalation is largely passive, lung elastic recoil providing the driving force. This recoil is created firstly by surface tension acting throughout the vast gas/liquid interface lining the alveoli, and secondly by elasticity of the lung tissue which has been stretched during inspiration. Elastic recoil is reduced at low lung volumes, like a slack elastic band, and in emphysema because of damaged alveolar septa.

Active expiration becomes stronger with speech, exercise, coughing, sneezing, giving birth and obstructive airways disease. Abdominal, internal intercostal and pelvic floor muscles may then be recruited to augment passive recoil. Latissimus dorsi is enlisted during singing, and in COPD during forced exhalation, which may explain the benefit found by some patients by joining a choir (Watson et al 2012).

Speech is created when the expiratory column of air is interrupted by vibrating vocal cords which break it into sound waves. The sound is then modified by the oropharynx, nasopharynx and oral cavity. A person with COPD may have a weak voice because of inability to generate sufficient expiratory pressure, and a person with neurological disease may have poor articulation because of weakness of the oropharyngeal musculature.

Respiratory muscle dysfunction can occur with COPD, thoracic deformity, critical illness or neurological disease (Gea 2012).
PRACTICE TIP
Drop your pen and then pick it up. Did you hold your breath? This illustrates the postural work of the diaphragm.

Pressures

**Alveolar pressure**: pressure inside the lung.

**Pleural (intrapleural/intrathoracic) pressure**: pressure in the pleural space.

**Pressure equivalents**
- 1 mmHg = 1.36 cmH₂O
- 1 kPa = 7.5 mmHg
- 1 torr = 1 mmHg.

Alveolar pressure is negative on inspiration and slightly positive on expiration. Pleural pressure needs to be constantly negative to keep the lungs open. This negative pressure is achieved by inward pull from lung elastic recoil and outward pull from rib cage recoil, which attempts to spring outwards at all but the highest volumes (Dominelli & Sheel 2012). Outward recoil is assisted by the pumping out of pleural fluid by the lymphatics, leading to a total negative pleural pressure of about -6 cmH₂O, modulated by the breathing pattern (Negrini 2013). Inward and outward forces are in equilibrium at the end of a quiet exhalation, this resting lung volume being termed the functional residual capacity (FRC). Outward recoil assists inspiration, especially from low lung volumes.

These pressures are disturbed by:
- a large 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 less opposed and the lung hyperinflates.

Increasing the expiratory pressure by coughing or forceful expiration can only generate extra flow in the early stages because, as airways narrow and frictional resistance increases, pleural pressure exceeds airway pressure and compresses
the airways. This ‘dynamic airway compression’ occurs at about 40 cmH₂O (De Beer 2013), presaging the effort-independent portion of the flow-volume relationship (see Fig 2.33) and can make forceful coughing counterproductive for people with obstructive airways disease.

**Resistance**

\[
\text{Resistance} = \text{force that must be overcome during breathing} = \frac{\text{pressure change}}{\text{flow change}}
\]

**Airflow resistance**

Resistance to airflow is caused by friction, which is created in the airways when gas slides against the walls and over itself. Airflow resistance depends on the speed of airflow and calibre of the airway.

Peripheral airflow resistance is low because at the level of the terminal bronchioles the large number of small airways creates a wide total cross-sectional area of 180 cm², causing laminar flow. The cross section reduces to 2.5 cm² in the trachea, where there is higher total resistance, creating turbulent and disorganized airflow (Fig 1.3). Fig 1.4a shows increased resistance due to airflow obstruction.

**Fig 1.3**

Increase in total cross section of the airways as they divide, creating less frictional resistance as airflow becomes more laminar and streamline

Frictional resistance must also be overcome in the chest and abdomen as organs
are displaced by the moving lung.

**Elastic resistance**

Lung tissue, alveolar surface liquid and the chest wall contribute to elastic resistance, which is increased by conditions such as pulmonary oedema, lung fibrosis (Fig. 1.4b), rib cage deformity, obesity or a slumped posture.

**Fig 1.4**

(a) Both alveoli are normal, but the airway supplying alveolus B shows airflow obstruction which is causing frictional resistance to airflow.

(b) Both airways are normal, but alveolus B shows reduced compliance caused by a thickened alveolar-capillary membrane or raised surface tension, which increases elastic resistance.

![Diagram](image)

**Compliance**

\[
\text{Compliance} = \frac{\text{volume change}}{\text{pressure change}}
\]

Compliance reflects the willingness of the lungs to distend, and elastance the willingness to return to their resting position. Compliance is represented by the relationship between volume and pressure, i.e. how much pressure (work of breathing) is required to expand the lungs. This relationship is curved rather than linear due to a variation in the work of breathing at different lung volumes (Fig 1.5).

**Fig 1.5.**
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. Clinical reasoning indicates that prevention of atelectasis would therefore be more sensible than treating it once it has occurred.

**PRACTICE TIP**
Blow up a balloon and feel your work of breathing at low, mid and high volumes

Compliance is lower on inspiration, i.e. it is harder to breathe in than out, due mostly to alveolar fluid surface tension. This process is known as hysteresis and is demonstrated by the pressure-volume loop (Fig 1.6).
Alveoli are vulnerable to injury at excessively high or low volume, and mechanical ventilation (MV) aims to avoid either extreme, especially in patients with damaged lungs, e.g. acute respiratory distress syndrome.

The contribution of airways to compliance relates to their calibre, resistance being increased and compliance decreased if airways are narrowed by bronchospasm, oedema, the collapsing airways of emphysema, and sometimes secretions in the large airways where there is greater overall resistance and less collateral ventilation.

The surface tension of alveolar fluid is partially counteracted by surfactant, a constituent in the fluid that acts like detergent, preventing the alveolar walls sticking together. Small alveoli are therefore less likely to collapse and empty their contents into large alveoli.

**PRACTICE TIP**
Pour some water into a plastic bag, empty it and then pull the bag open. The forces of surface tension are what makes this difficult. Repeat with a few drops of washing-up liquid added.

The contribution of extra-pulmonary structures to compliance relates to the ease with which the chest wall can be pushed away on inspiration. Kyphoscoliosis or a distended abdomen reduce compliance.

**Static compliance** is measured during a breath-hold so 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 (WOB)**

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

- the pressure required to move a volume of gas, i.e. transpulmonary pressure \( \times \) tidal volume
- oxygen consumed by the respiratory muscles, i.e. the oxygen cost of breathing.

Elastic resistance contributes 80% of the WOB and airflow resistance the remaining 20% (Levitzky 2007 p.32). Relative contributions to airflow resistance are:

- nasal passages: 50%
- larynx: 25%
- trachea to 8th generation: 20%
- peripheral airways: 5% (Eriksson 1996).

Normally, breathing is surprisingly efficient, helped by fluid coating the moving surfaces of the pleura, assuring a tight lung–chest wall coupling and allowing lung volume to faithfully follow changes in chest wall volume during the respiratory cycle (Negrini 2013). The pleural cavity however does not appear to be essential: people who have had a pleurectomy, and elephants (West 2001), have no functioning pleura but are able to breathe quite happily. However, it is handy for thoracic surgeons who would have difficulty if the lung was attached directly to the chest wall.

A change in alveolar pressure of only 1 cmH\(_2\)O is usually enough for airflow (Negrini 2013) and WOB uses just 2–5% of total oxygen consumption at rest, but this may be increased to 40% in people with obstructive airways disease (Cairo 2012, p.194).

Deep breathing increases the work performed against elastic resistance, while rapid breathing increases the work against airways resistance. Most patients
find the right balance, but some need breathing re-education to find the optimal breathing pattern to minimize their WOB.

**Inspiratory muscle fatigue**
Fatigue reduces the capacity to develop force in response to a load. When acute, it is reversible by rest. 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 in the muscle is called peripheral fatigue.

The diaphragm differs from other skeletal muscles in that it has to provide a lifetime of sustained action against elastic and resistive loads rather than irregular action against inertial loads. It is equipped for this by its comparative resistance to fatigue (Polla et al. 2004) and the unusual way in which perfusion increases during contraction (Anzueto 1992). Fatigue occurs if energy demand exceeds supply, as when WOB is increased by airflow obstruction, or when the ability of the respiratory muscles to contract efficiently is impaired by hyperinflation, scoliosis or a flail chest.

Fatigue serves a protective function to avoid depletion of enzymes. Procedures that force patients to overuse fatigued muscles can cause muscle damage (Kallet 2011), which is most likely to occur when weaning patients from MV.

**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 encouraging activity. Weakness of the respiratory muscles may be caused by:

- neuromuscular disorder
- disuse atrophy
- malnutrition
- hypoxia, hypercapnia or acidosis
- low calcium, potassium or phosphate levels
- steroids
• inflammation, as occurs with COPD, sepsis or multisystem failure.

Weakness predisposes a muscle to fatigue. Fatigue differs from weakness in that a normal muscle can become fatigued if faced with excess WOB. Fatigue and weakness often coexist, especially in respiratory failure or during weaning from MV. The clinical features of fatigue and weakness are similar (Ch.2). Both are experienced as breathlessness.

**VENTILATION**

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

| **Respiration:** | (a) exchange of gases between the 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 defense functions of the respiratory system. |
| **Ventilation:** | gas movement between the outside and the alveoli, i.e. inspiration and expiration (the terms ventilation and respiration are sometimes used interchangeably). |
| **Breathing:** | the process by which the respiratory pump creates ventilation. |
| **Minute ventilation or minute volume (VE):** | amount of gas breathed per minute, i.e. tidal volume x respiratory rate. |
| **Tidal volume (VT):** | volume of air inhaled and exhaled at each breath. |
| **Alveolar ventilation:** | (tidal volume – physiologic dead space) x respiratory rate. |

A healthy spontaneously breathing adult maintains an approximate $V_E$ of 5-9 litres, moving a $V_T$ of 450-600 ml with a respiratory rate of 10-15 bpm.

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, defined above
- dead space ventilation ($V_D$), which does not contribute to gas exchange.

Most dead space is made up of anatomical $V_D$ (Fig 1.7) 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 $V_T$ in a human, more in a giraffe. Alveolar $V_D$, representing air that reaches the alveoli but does not get into the blood, is minimal
in normal lungs. The sum of anatomical and alveolar $V_D$ is called physiological $V_D$. The presence of $V_D$ is one reason why it is more efficient to increase $\dot{V}_E$ by breathing deeper than by breathing faster. Dead space is most usefully expressed in relation to tidal volume ($V_D/V_T$) and is normally 30% of $V_T$ (Gott & Dolling 2013).

Ventilation is not distributed evenly in the lungs (Fig 1.8). In most healthy young adults, dependent regions are better ventilated for two reasons:

1. In the upright position, alveoli in upper regions are more inflated, but mostly with $V_D$ gas. Gas travels more easily at first to the open spaces of these non-dependent regions, but they are rapidly filled and gas then travels to the dependent regions below. Alveoli in dependent regions are partially compressed by the weight of the lungs, heavy with blood, above and around them. They therefore have more potential to expand, i.e. they are more compliant, allowing greater ventilation with fresh gas. This reasoning carries through to whatever position a young adult is in. If the alveoli are collapsed or heavily compressed, e.g. if the patient is slumped in a chair, they will be lower on the compliance curve (Fig 1.5) and less easy to inflate.

2. In the horizontal position, dependent fibres of the diaphragm contract from a position of mechanical advantage because they are more stretched. Side-lying augments this because the shape of the chest creates a greater vertical distance. Although fresh gas in the lower lung provides a greater contribution to gas exchange, the upper lung is more expanded and therefore responds earlier to deep breathing exercises for increasing lung volume. For patients with atelectasis, therefore, and indeed for most clinical problems, patients are placed with the affected lung upwards (Ch.6).

![Fig 1.7](image1)
Fig 1.7
Average volumes and flows of gas and blood in the lungs. Frequency: average breaths per minute (West 2011)

![Fig 1.8](image2)
Fig 1.8
Effect of gravity on the distribution of ventilation and perfusion (left), with position of alveoli on compliance curves (right).
(a) Upright lungs in a healthy young non-obese person, showing slight downward ventilation gradient and strong downward perfusion gradient
(b) In supine, pressure from the abdominal contents stretches the dependent portion of the diaphragm, compressing dependent alveoli but facilitating more efficient diaphragm contraction.

(c) Side lying allows greater volume change in the dependent lung due to pressure from the abdominal contents stretching this side of the diaphragm.

\[ V: \text{lung volume, } P: \text{pressure required to expand lung.} \]
However, unlike the perfusion gradient, the ventilation gradient is only slight and responds to minor fluctuations, so that:

- it is reversed in elderly people (Lowe 2005), who have a higher closing volume (Fig 1.9), and most patients are elderly,
- it is reversed in obese people (Pedoto 2012), those on some modes of MV, those who spend time in slumped positions without taking deep breaths, and sometimes in children,
- in prone, pressure from the abdominal contents obliterates the gradient so that the lungs are uniformly inflated (Marcucci 2001).

**Fig 1.9**
Factors that shift tidal breathing into the closing volume range, leading to airway closure in the lung bases during quiet breathing. $V_T$: tidal volume

**PRACTICE TIP**
Auscultate colleagues’ lungs in standing, sitting, slumped sitting, supine, side lying and prone

If airways are obstructed, ventilation can continue through collateral channels. These are unimportant in normal lungs and ventilating through them is less efficient than using the main airways because airflow resistance is 50 times greater. This difference is eliminated in emphysema, when collateral ventilation promotes more homogeneous ventilation (Cetti et al 2006).

Normal breathing creates a $V_T$ 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.

**DIFFUSION**

*No other capillaries in the body are shielded from the outside environment by such a minute amount of tissue*
The wide total cross section of the peripheral airways means that airflow here essentially ceases, and gas movement from the respiratory bronchioles to the alveoli continues by gaseous diffusion. In the alveoli, Oxygen dissolves in alveolar fluid and is driven across the alveolar-capillary membrane by the partial pressure difference, with CO₂ coming the other way. The alveolar-capillary membrane is 50 times thinner than airmail paper (Weibel 2013), or 0.2 μm (West 2013), comprising two sheets of endothelium held together by wisps of connective tissue support. It is semipermeable, preventing plasma proteins and water leaking into alveoli but allowing gas exchange. Oxygen tension is equalized in one-third of the time that the blood takes to pass each alveolus, while CO₂ diffuses 20 times more quickly (Goodman 2001).

Impaired diffusion across the membrane does not play a major role in gas exchange abnormalities except sometimes during exercise (Garvey et al 2012) or when sepsis causes high cardiac output and shortened transit times (Townsend & Webster 2000).

**PERFUSION**

The lungs have a dual circulation. The high-pressure (100 mmHg) bronchial circulation, from the aorta, supplies the lung tissue itself but is not essential to survival, as shown after lung transplant when the bronchial vessels are tied. However, the lungs are awash with blood from the dominant low-pressure (~15 mmHg) pulmonary circulation, which bathes the surfaces of the alveoli in order to exchange gases (West 2013). The pulmonary vasculature is equivalent to 7000 km of capillaries (Denison 1996), through which up to 6 l of blood passes per minute (Ivanov 2013). This can act as a blood reservoir in case of need such as haemorrhage.

The pulmonary circulation can respond to changes in flow with little change in pressure, reducing resistance by dilating, recruiting closed capillaries or shifting blood to the systemic circulation.

The effect of gravity on the low pressure pulmonary circulation is to create a
perfusion gradient with more blood in dependent regions (Fig 1.8). This is steeper than the ventilation gradient because of the density of blood. The perfusion gradient is represented by zones (West 2012 p.45):

- Zone I is in the upper non-dependent lung, where alveolar pressure exceeds pulmonary arterial pressure so that capillaries are squashed and no blood flows.
- Zone II is in the middle, where pulmonary arterial pressure exceeds alveolar pressure, which in turn exceeds venous pressure.
- Zone III is in dependent lung, where venous pressure exceeds alveolar pressure.

Zone I in health is small or nonexistent. However, if hypovolaemic shock reduces arterial pressure, or MV increases alveolar pressure, Zone I becomes significant. MV also pushes blood downwards (Ch.17) and the pressure of this blood may lead to airway closure in Zone III.

In addition, lung perfusion is affected by:

- lung volume, the vessels being stretched in the hyperinflated state
- disease, e.g. alveolar destruction in emphysema causes disruption of perfusion as well as ventilation
- position, e.g. perfusion is more uniform in prone than supine (Nyren 1999) and better matched with ventilation (Henderson et al 2013).

VENTILATION/PERFUSION RATIO

It is no good having a well-ventilated alveolus if it is not supplied with blood, nor 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 is expressed as the ratio of alveolar ventilation to perfusion ($\dot{V}_{A}/\dot{Q}$).

$\dot{V}_{A}/\dot{Q}$ matching is assisted by the mixing of expired and inspired gases through the common deadspace (Glenny et al 2013), but a degree of $\dot{V}_{A}/\dot{Q}$ mismatch is normal because of dissonance between ventilation and perfusion gradients, the lung bases receiving 18 times more blood and 3.5 times more gas than the apices in the upright lung (Thomas 1997).
Pathological $\dot{V}_A/\dot{Q}$ mismatch is due to a high or low $\dot{V}_A/\dot{Q}$ ratio. A high ratio occurs when the alveoli are ventilated but perfusion is impaired so there is nowhere for the oxygen to go, causing increased dead space. A low $\dot{V}_A/\dot{Q}$ ratio occurs where lung units are perfused but not adequately ventilated, which is the condition most frequently dealt with by physiotherapists. This creates a shunt, defined as the fraction of cardiac output that is not exposed to gas exchange in the lung. A shunt over 20% limits the utility of oxygen therapy because added oxygen cannot reach the shunted blood. The shunt is measured by comparing arterial and mixed venous blood (p.329), expressed as a percentage of cardiac output. A small shunt is normal because part of the bronchial circulation mingles with pulmonary venous drainage. The mixing of shunted venous blood with oxygenated blood is known as venous admixture, which is normally 5% of cardiac output (Takala 2007).

Systemic hypoxia stimulates selective vasodilation to assist perfusion of vital tissues. Pulmonary hypoxia stimulates the opposite response: if a fall in alveolar oxygen tension is detected, an ingenious mechanism called hypoxic vasoconstriction helps maintain gas exchange by constricting capillaries adjacent to these alveoli, thus limiting wasted perfusion and improving $\dot{V}_A/\dot{Q}$ match (Ariyaratnam et al 2013). When the lung bases are affected, e.g. in pulmonary oedema, obesity (Pedoto 2012) or the early stages of COPD, local shutdown of vessels forces blood to the better ventilated upper regions, shown radiologically as enhanced vascular markings towards the apices, or ‘upper lobe diversion’. Hypoxic vasoconstriction becomes counterproductive when alveolar hypoxia occurs throughout the lung, as occurs in advanced COPD, leading to generalized vasoconstriction and pulmonary hypertension.

**ARTERIAL BLOOD GASES**
**PO**2: partial pressure or tension of oxygen

**PaO**2: partial pressure of oxygen in arterial blood, i.e. oxygen dissolved in plasma (normal 11-14 kPa or 82.5-105 mmHg)

**SaO**2: extent to which haemoglobin in arterial blood is saturated with oxygen, i.e. capacity of blood to transport oxygen

**SpO**2: as above, but described in terms of measurement by pulse oximetry (the distinction is rarely important and normal values are the same)

**Haemoglobin** (Hb): molecule in erythrocytes that binds to and transports oxygen and some CO₂ around the body

**PaCO**2: partial pressure of CO₂ in arterial blood; basis of respiratory acid-base balance, (normal 4.7-6.0 kPa or 35-45 mmHg)

**F**I**O**₂: fraction of inspired oxygen (normal values in the Glossary)

Arterial blood gas (ABG) analysis identifies if breathing is effective by giving an indication of gas exchange, ventilation and acid-base status. Readings should be related to the concentration of inspired oxygen (F_I^O_2) and previous values.

Arterial blood samples are taken either from an indwelling arterial catheter or by intermittent puncture of the radial artery using local anaesthesia. Local anaesthesia is stipulated in the UK Guidelines (BTS 2008), not just for humanity but also because the pain of a needle going through the arterial wall may lead to hyperventilation or apnoea, which can invalidate the results (Lee 2012).

Table 1.1 relates the different measurements of arterial oxygenation.

<table>
<thead>
<tr>
<th>SaO₂ %</th>
<th>PaO₂ (kPa)</th>
<th>PaO₂ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>97</td>
<td>12.7-14.0</td>
<td>95-105</td>
</tr>
<tr>
<td>94</td>
<td>9.3-10.0</td>
<td>70-75</td>
</tr>
<tr>
<td>92</td>
<td>8.9-9.7</td>
<td>67-73</td>
</tr>
<tr>
<td>90</td>
<td>7.7-8.3</td>
<td>58-62</td>
</tr>
<tr>
<td>87</td>
<td>6.9-7.7</td>
<td>52-58</td>
</tr>
<tr>
<td>84</td>
<td>6.1-6.9</td>
<td>46-52</td>
</tr>
</tbody>
</table>

**PaO₂** describes the 2% of oxygen that is dissolved in plasma, and reflects the pressure needed to push oxygen from blood into tissue cells. **SaO₂** describes the
98% of oxygen that is bound to Hb for transport. These do not give a measure of oxygenation at tissue level, and resting levels do not reflect oxygenation during exercise, nor predict nocturnal oxygenation.

Gas exchange entails:

- oxygen dissolving in alveolar lining fluid,
- diffusion of oxygen through the alveolar-capillary membrane into plasma,
- oxygen binding to Hb for transport around the body,
- at its destination, oxygen from Hb dissolving back into plasma,
- diffusion of oxygen across the capillary wall and delivery to the tissues.

The reverse happens to CO₂, but much is carried dissolved in blood and it slips back and forth with ease. The movement of CO₂ traditionally does not come under the term ‘gas exchange’.

**Oxygen dissociation curve**

The relationship between SaO₂ and PaO₂ is not direct but expressed by the oxygen dissociation curve (Fig 1.10). Its S-shape illustrates the protective mechanisms that function in both health and disease.

**Fig 1.10**

Oxygen dissociation curve relating oxygen saturation to oxygen tension. 2.3.DPG is an enzyme in red blood cells which increases in chronic hypoxaemia and allows easier unloading of O₂ to hypoxic tissues. P₅₀ is the PaO₂ at which Hb is 50% saturated and is the most sensitive indicator of a shift in the curve, a high value suggesting poor affinity of Hb for O₂. The shaded area represents critical tissue hypoxia.

**Upper flat portion of the curve**

At the plateau of the curve, the combining of oxygen with Hb is favoured by a high PO₂, and its stability is not unduly disturbed by changes in PaO₂. In health, this encourages loading of oxygen in the high PO₂ environment of the lung, and discourages unloading of oxygen before blood reaches the capillary bed. In disease, there can be a significant change in PaO₂, e.g. a reduction to 10.7 kPa (80 mmHg), with little change in SaO₂.

**Steep portion of the curve**
The dissociation of Hb becomes proportionately greater as PO$_2$ falls, so that small changes in PaO$_2$ greatly affect SaO$_2$. In health, this means that Hb can offload quantities of oxygen at cellular level while maintaining oxygen tension in the blood. In disease, large amounts of oxygen can be unloaded when tissues are hypoxic. A PaO$_2$ < 7.3 kPa (55 mmHg) tips the patient into a slippery slope where further small drops in PaO$_2$ 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 onto its oxygen, as with hyperventilation, hypometabolism or a cold environment. Pink ears and noses on frosty mornings are due to the reluctance of Hb to unload oxygen.

**Hypoxaemia and hypoxia**

**Hypoxaemia**

Hypoxaemia is reduced oxygen in arterial blood, defined as PaO$_2$ < 8 kPa (60 mmHg) or SaO$_2$ < 90%. Causes are:

- low $V_A/Q$ ratio or wasted perfusion (↑ shunt)
- high $V_A/Q$ ratio or wasted ventilation (↑ dead space)
- hypoventilation
- diffusion abnormality
- ↓ FIO$_2$.

Low $V_A/Q$ occurs when blood is shunted through consolidated, collapsed or damaged lung without seeing any oxygen, somewhat attenuated by hypoxic vasoconstriction. High $V_A/Q$ occurs, for example, when a pulmonary embolus blocks perfusion, thus increasing alveolar dead space and causing $V_A/Q$ mismatch at the other end of the spectrum (Fig 1.11).
Hypoventilation can be caused by respiratory depression (e.g. from oversedation), respiratory muscle weakness (e.g. with neuromuscular disorder), or sometimes with COPD if patients chronically hypoventilate in order to rest the diaphragm (Ch.3).

Diffusion abnormalities occur in disorders such as pulmonary oedema or fibrosing alveolitis, when a thickened alveolar-capillary membrane increases PAO$_2$-PaO$_2$.

↓FIO$_2$, is due to inadequate oxygen therapy, high altitude or fire entrapment.

**Hypoxia**

The term hypoxia is sometimes used interchangeably with hypoxaemia but it means oxygen deficit at tissue level, when demand exceeds supply, leading to anaerobic metabolism once PaO$_2$ reaches 4.5 kPa (33.8 mmHg) (Townsend & Webster 2000). It is more relevant to body function than hypoxaemia but more difficult to measure.

**Hypoxaemic** hypoxia occurs when hypoxia is caused by hypoxaemia.

**Anaemic** hypoxia is when Hb levels are reduced or abnormal Hb cannot carry enough oxygen. **Ischaemic** hypoxia indicates impaired oxygen transport, e.g. haemorrhage, myocardial infarct or peripheral arterial disease. **Histotoxic** hypoxia occurs when cells cannot extract or utilize oxygen, e.g. following cyanide poisoning or in septic shock.

**Effects of hypoxaemia and hypoxia**

Acute hypoxaemia induces vasodilation of the peripheral vascular beds, increasing cardiac output and improving oxygen delivery. Chronic hypoxaemia thickens the blood and may strain the right heart (see Fig 3.4) and even transient hypoxaemia may shorten life (Criner 2013).

Hypoxia progressively causes the following:

PaO$_2$ <7.3 kPa (55 mmHg): memory defect, impaired judgment

<5.3 kPa (40 mmHg): tissue damage
<4 kPa (30 mmHg): unconsciousness
<2.7 kPa (20 mmHg): death, except in migrating geese and hibernating turtles (Nikinmaa 2013).

The brain is the first organ to be affected, followed by the gut lining. The kidney is also sensitive to hypoxia and manifests its distress more obviously, reducing urine output and increasing potassium, creatinine or urea levels. Cardiac arrhythmias may occur when SaO₂ drops below 80%.

**Hypercapnia**

Hypercapnia is high PaCO₂ and reflects hypoventilation, leading to respiratory acidosis or compensated metabolic alkalosis. An acutely rising PaCO₂ is a danger sign if it presages exhaustion (see Fig 3.27), and with acidosis is an indication for mechanical ventilatory assistance. But chronic hypercapnia is neither dangerous nor damaging and may accompany advanced stable lung disease. It has also been associated with neck pain (Dimitriadis 2013). A certain level of CO₂ is required to transmit nerve impulses, control blood flow to the brain and shift the oxygen dissociation curve when required.

The clinical signs of hypoxaemia and hypercapnia are insensitive and non-specific, but Table 1.2 indicates some similarities and differences.

<table>
<thead>
<tr>
<th>Table 1.2 Clinical features of hypoxaemia and hypercapnia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypoxaemia</strong></td>
</tr>
<tr>
<td>Cyanosis</td>
</tr>
<tr>
<td>↑ Respiratory rate</td>
</tr>
<tr>
<td>↑ Heart rate</td>
</tr>
<tr>
<td>Peripheral vasoconstriction</td>
</tr>
<tr>
<td>Respiratory muscle weakness</td>
</tr>
<tr>
<td>Arrhythmias or bradycardia</td>
</tr>
<tr>
<td>Restlessness → confusion → coma</td>
</tr>
</tbody>
</table>

**Interpretation**
Examples of ABG abnormalities are:

- $\downarrow \text{PaO}_2$ with $\uparrow \text{PaCO}_2$: exacerbation of lung disease in a patient who is becoming too exhausted to ventilate adequately
- $\downarrow \text{PaO}_2$ with $\downarrow \text{PaCO}_2$: hypoxaemia in a patient who is breathless, e.g. pneumonia, fibrosing alveolitis, pulmonary oedema, pulmonary embolus
- normal $\text{PaO}_2$ with $\downarrow \text{PaCO}_2$: emotion causing hyperventilation, e.g. painful arterial puncture or hyperventilation syndrome.

Hypercapnia is likely to be longstanding if pH is >7.35 (BTS 2008).

Reduced minute volume raises PaCO$_2$ and lowers SaO$_2$, but the reverse is not true. Increased minute volume blows off CO$_2$, but SaO$_2$ does not rise above normal because Hb cannot be supersaturated. However mechanical ventilation with a high FiO$_2$ can raise PaO$_2$ above normal when the extra oxygen dissolves in the plasma.

**Other indices of oxygenation**

Gas exchange is best documented in relation to the inspired oxygen (Karbing et al 2007), described as the $\text{PaO}_2$:FiO$_2$ ratio (see Glossary). Less easy to measure, but useful in identifying the cause of hypoxaemia, is the difference between alveolar and arterial oxygen, known as the alveolar-arterial gradient, the ‘A-a gradient’, or $\text{PAO}_2$-$\text{PaO}_2$. A raised gradient indicates greater difficulty in getting oxygen across the alveolar-capillary membrane, as occurs in diffusion impairment. Hypoventilation or reduced FiO$_2$ does not affect the $\text{PAO}_2$-$\text{PaO}_2$.

**Acid-base balance**

Normal pH in the human body: 7.35-7.45

The degree of acidity or alkalinity is measured by pH, which measures hydrogen ions in solution, but negatively, i.e:

- low pH means more hydrogen ions and greater acidity
- high pH means fewer hydrogen ions and greater alkalinity.
Neutral is 6.8, but body functions occur on the alkaline side of neutral. **Acidosis** occurs when arterial pH falls below 7.35. The term means increased acidity in the body, while **acidaemia** means increased acidity of blood plasma, but the former is normally used for both. Acidosis can lead to myocardial depression, arrhythmias and hypotension, while hypercapnic acidosis weakens the respiratory muscles and can increase inflammation (Bruno 2012). **Alkalosis** occurs at pH over 7.45. The pH responds to metabolic and respiratory change but cannot differentiate between them.

The following identify acid-base imbalances:

- **Respiratory acidosis** occurs when the drop in pH is caused by increased PaCO$_2$. It is caused by hypoventilation, leading sometimes to ventilatory failure.
- **Respiratory alkalosis** occurs when a patient hyperventilates, which pushes down PaCO$_2$. This always accompanies a raised minute volume and often accompanies breathlessness, though the rapid shallow breathing pattern of a breathless person is not necessarily efficient, nor synonymous with hyperventilation.
- **Metabolic acidosis** occurs when the body produces too much acid or the kidneys cannot remove enough acid. Metabolic acids include all the body acids except dissolved CO$_2$. They are not respirable and have to be neutralized, metabolized, or excreted by the kidney. Lactic acidosis is a form of metabolic acidosis caused by the build up of lactic acid if oxygen supply is inadequate.
- **Metabolic alkalosis** raises pH out of proportion to changes in PaCO$_2$, a common occurrence in critically ill patients due to volume loss, diuretics or low potassium (Oh 2010).

Patients can have mixed acid-base disorders, e.g. a patient with acute hypercapnic COPD may have CO$_2$-induced respiratory acidosis, but comorbidities such as diabetes or the side effects of diuretics can induce a metabolic alkalosis, with the knock-on effects of a depressed respiratory drive and increased airway resistance (Terzano et al 2012).

**Buffers**

A buffer is a weak acid or base which mitigates a disturbed pH by mopping up or squeezing out hydrogen ions like a chemical sponge. **Bicarbonate** (HCO$_3^-$) is a buffer whose value provides an estimate of the metabolic component of acid-base balance, although it is affected by both metabolic and respiratory components. Values for HCO$_3^-$ are:

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

The effect of PaCO$_2$ on pH is in App C.

**Standard bicarbonate** (SBE) is the bicarbonate concentration under standard conditions, being adjusted as if PaCO$_2$ were 5.3 kPa, i.e. it is similar to bicarbonate in a person with normal acid-base status.

**Base excess** (BE) is a calculated value from SBE, being positive in metabolic alkalosis and negative in metabolic acidosis. It represents the quantity of acid required to restore pH to normal if PCO$_2$ were adjusted to normal. Like HCO$_3^-$, it measures metabolic acid-base balance, but takes buffering by red blood cells into account and provides a more complete analysis of metabolic buffering than HCO$_3^-$. BE is calculated from pH, PaCO$_2$ and haematocrit. Values are:

- normal: minus 2 to plus 2 mmol/l
- metabolic acidosis: < minus 3 mmol/l
- metabolic alkalosis: > plus 3 mmol/l.

**Along with clinical assessment, arterial blood gas** analysis helps identify the main causes of acidosis or alkalosis, although one may dominate and the other partially compensate (Fig 1.12). The prime mover is the one on which to focus and it is a mistake to treat a compensation.

**Fig 1.12**
Four examples of acid-base imbalance showing the process of compensation. Thick arrows in each box indicate which way a value has gone in order to create the acidosis or alkalosis. Thin arrows indicate which way the other value is going in order to compensate.
A quick tip, as seen above is that if the primary acid-base disturbance is metabolic, pH and bicarbonate/BE change in the same direction, while if the primary problem is respiratory, pH and PaCO$_2$ change in opposite directions.

**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). Any deviation of pH from normal is fiercely resisted, at whatever cost, by three homeostatic mechanisms:

1. The buffer system neutralizes acids or bases by giving up or absorbing hydrogen ions, all within seconds. The following equation represents the dissociation of carbonic acid in solution, acting as a sink for hydrogen ions:

   \[
   \text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^- 
   \]

   An increase in PaCO$_2$ shifts the equilibrium to the right, increasing H$^+$ and causing respiratory acidosis. A decrease in PaCO$_2$ shifts it to the left.

2. If buffering is not adequate, the lungs then present an avenue for regulating CO$_2$. Hyper- or hypoventilation can stabilize acid-base balance in 1-15 minutes.

3. If this is still not adequate, the kidneys take over, but they need 3-5 days to do so (Ayers & Warrington 2008).

These mechanisms work to dispose of the acids that are continually produced by the body's metabolic processes, caused mostly by the hydration of CO$_2$ to create carbonic acid.

**Interpretation**

Step 1: look at the pH:
- ↓ pH means acidosis
- ↑ pH means alkalosis.

Step 2: look at the PaCO$_2$: does it account for an abnormal pH? If breathing is the prime mover:
- ↑ PaCO$_2$ means respiratory acidosis
- ↓ PaCO$_2$ means respiratory alkalosis.

Step 3: look at the HCO$_3^-$ or BE: does it account for an abnormal pH? If breathing is not the prime
mover:
- $\downarrow \text{HCO}_3^-$ or BE means metabolic acidosis
- $\uparrow \text{HCO}_3^-$ or BE means metabolic alkalosis.

To help decide if a change in pH is respiratory or metabolic (if this is not obvious clinically), any change outside the following is likely to be metabolic in origin (Williams 1998):
- for every increase in PaCO$_2$ of 2.6 kPa (20 mmHg) above normal, pH falls by 0.1
- for every decrease in PaCO$_2$ of 1.3 kPa (10 mmHg) below normal, pH rises by 0.1.

When pH is restored to normal, full compensation has occurred. The stages can be identified as follows:
- abnormal pH + change in PaCO$_2$ or bicarbonate/BE = non compensation, i.e. an acute process
- abnormal pH + change in PaCO$_2$ and bicarbonate/BE = partial compensation
- normal pH + change in PaCO$_2$ and bicarbonate/BE = full compensation.

Table 1.3 clarifies the causes, effects and recognition of arterial blood gas imbalances. Examples are in App C

Table 1.3 Interpretation of arterial blood gas trends (N = normal)

<table>
<thead>
<tr>
<th>Status</th>
<th>Causes</th>
<th>Effects</th>
<th>Recognition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory acidosis</td>
<td>Hypoventilation, e.g. exhaustion, weakness</td>
<td>PaCO$_2$ $\uparrow$, pH $\downarrow$, HCO$_3^-$ (no renal compensation yet)</td>
<td>Shallow breathing, drowsiness, severe acute respiratory disease.</td>
</tr>
<tr>
<td>Chronic (compensated) respiratory acidosis</td>
<td>Chronic hypoventilation</td>
<td>PaCO$_2$ $\uparrow$, pH N, HCO$_3^-$ $\uparrow$ (retention of HCO$_3^-$ to restore pH, i.e. full compensation)</td>
<td>Severe chronic respiratory disease, e.g. COPD.</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Acute hyperventilation, e.g. anxiety, pain, acute cardiorespiratory disease, fever, CNS injury</td>
<td>PaCO$_2$ $\downarrow$, HCO$_3^-$ $\downarrow$, pH $\uparrow$ (partial compensation)</td>
<td>Breathlessness.</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Shock, lactic acidosis, diabetic acidosis, severe diarrhoea or dehydration, kidney failure</td>
<td>HCO$_3^-$ $\downarrow$, pH $\downarrow$, PaCO$_2$ $\downarrow$, BE &lt; -2 (partial compensation)</td>
<td>Hyperventilation (respiratory compensation to blow off PCO$_2$).</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Sepsis, heart failure, diuretics, severe vomiting, $\downarrow$ albumin</td>
<td>HCO$_3^-$ $\uparrow$, pH $\uparrow$, PaCO$_2$ $\uparrow$, BE &gt; +2 (partial compensation)</td>
<td>Delirium</td>
</tr>
</tbody>
</table>
Table 1.4 shows how two respiratory disorders can affect arterial blood gas readings. PaCO$_2$ values reflect breathlessness in acute asthma and hypoventilation in COPD. HCO$_3^-$ and pH values reflect an acute non-compensated condition in acute asthma, and full compensation in COPD.

**Table 1.4** Examples of arterial blood gas values in two disorders (numbers in brackets indicate mmHg).

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Acute asthma</th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO$_2$</td>
<td>12.7 (95)</td>
<td>9.3 (70)</td>
<td>7.3 (55)</td>
</tr>
<tr>
<td>PaCO$_2$</td>
<td>5.3 (40)</td>
<td>3.3 (25)</td>
<td>8 (60)</td>
</tr>
<tr>
<td>pH</td>
<td>7.4</td>
<td>7.5</td>
<td>7.4</td>
</tr>
<tr>
<td>HCO$_3^-$</td>
<td>24</td>
<td>24</td>
<td>29</td>
</tr>
</tbody>
</table>

**THE OXYGEN CASCADE**

The *raison d'être* of the cardiorespiratory system is to get oxygen to the tissues by means of oxygen cascading from the outside to the subcellular environment. Even when ventilation, diffusion and perfusion are in order, oxygen still has to reach and get into the tissues.

Oxygen transport is the passage of oxygen to the tissues via the arteries and capillaries. The arterial circulation also acts as a cushion to soften the pulsations generated by the heart so that capillary blood flow is stable. The term ‘oxygen transport’ is often used synonymously with, and is virtually the same as, oxygen delivery, which is the oxygen presented to the tissues. Oxygen consumption or uptake by the tissues is usually equivalent to oxygen demand, determined by the metabolic needs of the tissues.

Oxygen availability to the tissues depends on:

- SaO$_2$, PaO$_2$ and Hb
- cardiac output (CO)
- distribution of CO and local perfusion
- oxygen extraction
- the oxygen dissociation curve.

Tissue oxygenation reflects the balance between supply (oxygen delivery or DO$_2$) and demand (oxygen consumption or VO$_2$). The cardiorespiratory system, as with most other systems, has
plenty of reserve capacity, and \( \text{DO}_2 \) is normally 4 times greater than \( \dot{\text{VO}}_2 \), creating an oxygen extraction ratio \( \left( \dot{\text{VO}}_2/\text{DO}_2 \right) \) of 25% (Fawcett et al 2006).

\( \dot{\text{VO}}_2 \) varies with metabolic rate. An increase is usually met without difficulty by increased \( \text{DO}_2 \) (mostly through increased CO, partly through increased minute ventilation) and increased oxygen extraction by the tissues. Once maximum oxygen extraction is reached, consumption no longer drives delivery but becomes dependent on it. Further increase in demand, or reduced supply, leads to hypoxia.

Some organs are greedier than others, e.g:

- The brain comprises 2.2% of body weight and receives 1.5% of CO
- The myocardium comprises 0.5% body weight and receives 5% of CO
- The kidneys comprise 0.5% body weight and receive 20% of CO
- Skeletal muscle and skin comprise 50% body weight and receive 10% of CO (Epstein 1993).

Tolerance to hypoxia also varies:

- The brain suffers irreversible damage within 3 minutes
- The kidneys and liver can tolerate 15–20 minutes of hypoxia
- Skeletal muscle withstands 60–90 minutes and vascular smooth muscle 24–72 hours
- Hair and nails continue to grow for some days after death (Leach & Treacher 2002).

Septic patients may need 50-60% extra oxygen delivered to their tissues, while patients with multiple trauma, septic shock or burns may require 100% extra (Epstein 1993). At the same time, critically ill patients may be affected by:

- impaired \( \text{DO}_2 \) because of cardiorespiratory dysfunction
- cellular oxygen extraction hindered by toxins associated with sepsis, leading to mitochondrial dysfunction (Van Boxel et al 2012)
- loss of autoregulation, leading to disordered regional distribution of blood flow, both between and within organs
- hypoxic kidneys and a liver unable to detoxify byproducts of the shocked state.

If the body is not able to acquire, transport, extract and utilize sufficient oxygen, lactic acidosis occurs.

Compared to gas exchange in the lung, which is easily monitored in arterial blood, tissue oxygenation is usually estimated indirectly from the leftover oxygen in pulmonary artery blood,
where it is at the end of its journey before being reoxygenated in the lungs (Ch.17).

**VARIATIONS**

**Effects of obesity**

Obesity is the most common metabolic disease worldwide (Sood 2009) and the commonest chronic disorder in the US (O’Donnell et al 2010). The condition has more than doubled globally since 1980, is the fifth leading mortality risk (WHO 2012) and second only to tobacco as the leading preventable cause of death. For adolescents and children, the incidence of overweight has tripled since 1980 (DeTurk & Cahalin 2011, p.477).

Obesity loads the respiratory system, pushing up the diaphragm so that FRC approaches residual volume (Fig 1.13), leading to closure of dependent airways and $V_{A}/Q$ mismatch (Salome et al 2010). Breathing patterns tend to be rapid, shallow and apical.

**Fig 1.13**

Effect of obesity on lung volumes, showing how FRC can approach RV. Compare with Fig 2.33, p. TLC: total lung capacity, IC: inspiratory capacity, FRC: functional residual capacity, RV: residual volume

Obesity may also cause:

- a mixed obstructive/restrictive respiratory defect (Reynolds 2011)
- ↑ risk of diabetes, liver disease and heart disease (McGown et al 2014) including in children (Balakrishnan 2014)
- ↑ work of breathing, ↑ $\dot{V}O_2$ (Murphy & Wong 2013) and breathlessness (Bernhardt et al 2013)
- attenuated response to hypoxic and hypercapnic ventilatory drives, ↓ work capacity, exaggerated blood pressure (BP) response to exercise (Dipla et al 2012) and, with exercise, stress on the heart (Vella et al 2012)
- systemic inflammation which reduces muscle mass (King et al 2013), worsens airway inflammation, increases infection risk (Almond 2013) and reduces response to steroids and bronchodilators (Sismanopoulos 2013)
- urinary incontinence (Krause 2010), osteoporosis, sleep apnoea (Capodaglio 2013), many cancers (Vanni & Bugianesi 2014) and barotrauma during mechanical ventilation (Pedoto 2012)
- 38 -

- ↓ life expectancy (Jiang et al 2013)
- bias in the health care system (O’Brien et al 2012).

On the surgical wards, an obese patient should barely have emerged from anaesthesia before the physiotherapist becomes involved in pain control and positioning, closely followed by incentive spirometry and early mobilization (Reynolds 2011). Morbidly obese people show a greater propensity for thrombosis and wound infections and demonstrate a 30% likelihood of atelectasis or pneumonia after abdominal surgery (Licker et al 2007).

On the medical wards, obesity is associated with asthma (Divekar 2014) and COPD (O’Donnell et al 2010). For patients with lung disease, steroid medication can augment obesity, and obesity can disrupt pharmacokinetics (Cooper 2011).

On the intensive care unit, obesity increases the risk of sepsis and the complications of immobilization, the latter being moderated by early rehabilitation (Genc et al 2012). Oddly but pleasingly, obesity is also associated with improved survival in septic shock (Wacharasint 2013).

Multidisciplinary rehabilitation is required for comorbidities (Capodaglio 2013). Oxygenation may increase during exercise because of the effect of deep breathing on expansion of collapsed lung units (Sood 2009).

Effects of smoking

*Tobacco is the single most preventable cause of death in the world today… it kills up to half of those who use it*  
WHO 2008

Approximately 20% of the world population smokes tobacco (Rom 2013), and each smoker loses at least a decade of life (Jha et al 2013). Causes are the 5000 chemicals in tobacco smoke (Rom 2013), including cyanide, carbon monoxide, arsenic and 43 known carcinogens (Aslani & Rafiei 2012). The smoke causes mucous obstruction (Randall et al 2006) and inflammation (Rom 2013). Carnage to the cardiorespiratory systems is well known (Ch.3/4), but virtually every system is affected and the litany of destruction is outlined below.

Smoking is associated with back pain, other musculoskeletal pains (Pignataro 2012), osteoarthritis (Amin et al 2007), muscle weakness and osteoporosis (Pignataro 2012). Smoking compromises immunity (Bauer et al 2013) and plays a major role in autoimmune diseases, e.g. rheumatoid arthritis, SLE (Ch.3) (Pignataro 2012) and ankylosing spondylitis (Ward et al 2008). It impairs cognition, increases
the risk of developing some neurological diseases and neuropathic pain (Pignataro 2012), wrinkles the skin (Patel 2006), disturbs sleep (Ezzie 2011), increases depression (Berk et al 2013), doubles the risk of dementia (Pignataro 2012) and shortens life by 11 minutes for each cigarette smoked (Warren 2001). It depletes vitamin C, which would otherwise repair some of the lung damage (Banerjee et al 2008), and damages the diaphragm and quadriceps (Ramirez-Sarmiento 2004). It ulcerates the colon, rots teeth (Rom 2013) and causes cataract (Glynn et al 2009) and ulnar neuropathy (Richardson 2009). It impairs bone and wound healing, and increases the risks of cancer and postoperative complications, although it eases postoperative nausea and vomiting (Talbot & Palmer 2013) in patients who can escape the ward. Smoking doubles the risk of developing macular degeneration (Evans et al 2005) and increases the risk of subarachnoid haemorrhage 6-fold (Partridge 1992) and pneumothorax 13-fold (Light 1993). Drug metabolism is impaired (Licker et al 2007), opioid requirements increased (Talbot & Palmer 2013), and the effects can even jump a generation, the grandchildren of smokers being more likely to develop asthma (Leslie 2013).

Smoking is neither virile nor sexy, and tobacco is toxic to both testes and ovaries. Smoking during pregnancy increases preterm birth (Cox et al 2013), and causes as much damage to the foetus as if it was smoking itself (Le Souëf 2000). The BMA (2004) has found the following:

- Fertility is reduced in men and women, women smokers being twice as likely to be infertile as non-smokers, and both sexes showing reduced response to fertility treatment.
- For men, damage to blood vessels leads to male smokers being at least 50% more likely to be impotent, while those who succeed are more likely to produce damaged, malformed or mutated sperm.
- In women, smoking can cause dysmenorrhoea and a 25% increased risk of miscarriage.
- Smoking is the largest preventable cause of infant ill health and death, as well as risking birth defects. Damage persists into later life, and children whose mothers smoked during pregnancy are more likely to develop obesity, COPD and possibly cancer.
- The risk of cot death is trebled in infants whose mothers smoke, and increased 2½ times if the father smokes. Babies of smokers cry more, and later have poorer performance at school.
Second hand smoke increases postoperative complications (Licker 2007), risk of heart and lung disease (Talbot & Palmer 2013), and, in children, dental caries (Tanaka et al 2010), asthma and glue ear (Rouch et al 2010). Passive smoking also increases the risk of stroke (Cao et al 2013), eye disease (Kahn 2006) and exercise limitation (Arjomandi et al 2012). Third-hand smoke is a risk to children who play on contaminated surfaces or ride in cars where others have smoked (Talbot & Palmer 2013).

Nicotine is more addictive than heroin and seven times as addictive as alcohol (Haas & Haas 2000). Its one redeeming feature is that, according to patients, it aids relaxation and acts as a social crutch (Lancet 2013).

**A custom loathsome to the eye, hateful to the nose, harmful to the brain and dangerous to the lungs**

King James I

**Effects of stress**

All ill people suffer stress, usually as a result of and sometimes as a predisposing factor to illness. The following effects of stress have been identified:

- ↑ respiratory rate, heart rate and BP (Schwartz et al 2011)
- ↑ sympathetic and platelet activation, which impairs vascular function, progresses heart disease (Staniute et al 2013) and predisposes to arrhythmias (Brame & Singer 2010)
  - ↑ circulating catecholamines (Friedrich 2013)
  - hyperglycaemia, immunosuppression, ↑ catabolism (Fawcett et al 2012)
  - inflammation (Maslanik 2013), possibly explaining a link with asthma (Lehrer et al 2008) and Gulf War syndrome (Broderick et al 2013)
  - in children, impaired immune function which can last into adult life (Slopen et al 2013)
  - before birth, inflammatory changes that may permanently alter the function of the nervous and immune systems (Diz-Chaves et al 2013).

**Effects of sleep**
Sleep is something of a mystery. 

Primhak & Kingshott, 2012

The effects of sleep on breathing include (Thompson 2001):

- ↓ respiratory drive and ↑ PaCO₂, especially during rapid eye movement (REM) sleep (Fig 1.14), the reduced minute ventilation being out of proportion to the lower metabolic rate
- dips in SaO₂ to 90% or less (BTS 2008) which may drive SaO₂ down the steep part of the dissociation curve
- ↓ lung volume due to the horizontal position, ↓ muscle tone, leading to \( \dot{V}_{\text{A}}/\dot{Q} \) mismatch, and a doubling of airway resistance (Xie 2012)
- bronchoconstriction, which is only of consequence with asthma (Kamdar et al 2012)
- ↓ mucociliary clearance.

Most people can accommodate all this quite happily, but those with little cardiorespiratory reserve can suffer dramatic nocturnal desaturation (Ch.5) as well as nights that are disturbed by breathlessness or coughing.

Sleep fragmentation brings further tribulation. A full 90 minute cycle is needed to gain the benefits of REM sleep, which comprises about a quarter of the sleep cycle and is associated with dreaming and perceptual learning. The brain is highly active during this restorative phase and consumes more oxygen than when awake at rest. It is the time when memories are processed (Cipolli et al 2013) and is particularly important for critically ill patients to prevent memory distortion.

Sleep deprivation contributes to toxin accumulation (Xie et al 2013), stress (Kerkhofs 2012), stroke, multiple sclerosis, Alzheimer's disease, epilepsy, pain (Palma 2013) impaired cognition, inflammation, depression, diabetes, obesity and cardiovascular disease (Porkka-Heiskanen 2013). Cardiac complications can develop from too much or too little sleep (Ramos et al 2013).

**Fig 1.14**
Control of ventilation during sleep, showing reduced respiratory drive at deeper sleep stages (Owens 2013). *REM:* rapid eye movement
Nurses and doctors frequently overestimate how much sleep patients are getting, and underestimate the importance of sleep.

Gelling (1999)

**Effects of immobility**

Inactive people are contributing to a mortality burden as large as tobacco smoking.

Wen & Wu 2012

It is estimated that 60% of the world’s population is not physically active enough (Vrdoljak 2014). Immobility is known to cause:

- cardiac atrophy, ↓ blood volume, orthostatic intolerance, ↓ VO$_2$max (Fig 1.15) and cardiovascular instability during position change (Vollman 2013)
- ↓ DO$_2$, accelerated ageing and hypertension (Bortz 1984)
- for critically ill patients: pneumonia, delayed weaning, pressure sores (Vollman 2013), myopathy, delirium (Pawlik 2012), ICU psychosis and urine infection (Olivier 1998)
- ↓ cognitive function (Misak 2011) and depression (Hamer *et al* 2013)
- constipation and incontinence (Nurko & Scott 2011)
- cardiovascular deconditioning, thromboembolism, ↓ muscle mass by up to 1–5% per day (Mah 2013)
- ↓ survival and quality of life, ↑ healthcare utilization (BTS 2013)
- obesity, insulin resistance, high cholesterol, hypertension (Vrdoljak 2014)
- muscle atrophy, inflammation (Kang & Ji 2013) and joint contractures (Brower 2010).

Contractures begin immediately, especially for joints held in extension (Trudel et al 1999), although this is unlikely to be significant for a patient who is immobile for a few days.

Loss of gravitational stimulus to the cardiovascular system causes a negative fluid balance within 24 hours, impairing vasoconstrictive ability and augmenting deconditioning. Deterioration occurs more rapidly in the cardiorespiratory system than the musculoskeletal systems, and deterioration is quicker than recovery (Dean & Ross 1992).

**Fig 1.15**
The effect of immobility on maximum oxygen consumption

```
Immobility
  ↓
  ↓ blood volume
  ↑ sympathetic activity
  ↓ cardiac output
  ↓ vasoconstrictive reserve
  ↓ orthostatic intolerance
  ↓ VO2max
```

**Turning, sitting and standing up**

Regular position change as a preventive measure reduces haemodynamic instability on movement (Vollman 2013). Moving from supine to standing increases minute volume (Bahadur et al 2008) and redistributes blood from the thorax to the lower body, followed by compensatory vasoconstriction in most patients to restore BP (Fig 1.16).

**Fig 1.16** Typical circulatory response to postural change. *HR*: heart rate, *MAP*: mean arterial pressure

```
Moving from supine to standing
  ↓
```
500 ml blood migrates to pelvis and legs
\[\downarrow\]
\[\downarrow\] venous return, cardiac output and MAP
\[\downarrow\]
\[\downarrow\] BP detected by aortic arch and carotid baroreceptors
Information sent to brainstem
\[\downarrow\]
\[\uparrow\] sympathetic activity and \[\downarrow\] parasympathetic activity

\[\uparrow\] HR and myocardial contractility \[\downarrow\] arteriolar constriction \[\downarrow\] venous constriction

\[\uparrow\] cardiac output \[\uparrow\] peripheral resistance \[\uparrow\] venous return

\[\uparrow\] BP

Orthostatic intolerance occurs with inadequate haemodynamic compensation, e.g. with hypovolaemia or autonomic dysfunction, after prolonged bed rest or if the patient is elderly or dehydrated. Slow deep breathing improves orthostatic tolerance (Lucas 2013).

**Effects of exercise**

*Physical exercise is the best known training for the cardiovascular, pulmonary and musculoskeletal systems.*

Lainscak *et al* (2013)

Exercise increases oxygen delivery, consumption and extraction by several mechanisms:
- The cardiovascular response can increase cardiac output (CO) 5-fold, accomplished by a near-doubling of stroke volume and an increase in heart rate (HR) to about 220 minus the person's age (MacIntyre 2000).
- The respiratory response is represented by increased diffusion, more uniform lung perfusion, recruitment of dormant capillaries and, except with asthma, bronchodilation (Dominelli & Sheel 2012).
• Intense exercise can increase \( \dot{V}O_2 \) 20-fold (Owens 2013). Cerebral oxygenation rises during mild exercise and drops during intense exercise (Peltonen 2012). \( PaO_2 \) remains steady throughout.

• Acid-base balance is usually maintained by increased ventilation, but metabolic acidosis may develop if buffering mechanisms are unable to cope with the extra CO\(_2\) and lactic acid. Exercise enhances mucous transport (Houtmeyers 1999), aids sleep (Hargens et al 2013), induces a cascade of molecular and cellular processes that dampens chronic inflammation and pain in knee osteoarthritis (Gomes 2013) and other musculoskeletal conditions (Hagen 2012), and reduces the risk of developing chronic pain (Sluka et al 2013).

Cardiovascular performance imposes the primary limit to exercise (Trinity 2012), but for people with lung disease, dyspnoea may be the limiting factor (Stickland et al 2012). Exercise above a certain level for each individual is accompanied by inefficient anaerobic metabolism (Garvey et al 2012). Exhaustive training can impair immunity (Xiang et al 2013).

Lack of cardiorespiratory fitness is one of the strongest predictors of mortality (Smith 2013a) and regular exercise is an accepted treatment for cardiopulmonary disease (Woodard & Berry 2001). It also shows the following benefits:

• \( \uparrow \dot{V}O_2\text{max} \) (Mendes 2013), i.e. \( \uparrow \) fitness

• \( \downarrow \) cardiovascular disease and \( \uparrow \) quality of life (Vrdoljak 2014)

• \( \downarrow \) respiratory disease (Williams 2014)

• \( \downarrow \) oxidative stress and cellular ageing (Corbi et al 2012)

• \( \uparrow \) immune function (Navarro et al 2013)

• \( \uparrow \) bone mineral density to a greater degree than the drug alendronate (Macias et al 2012)

• \( \downarrow \) inflammation related to COPD (Davidson 2012), heart failure (Vlist & Janssen 2010) and sepsis (Araújo et al 2012)

• \( \downarrow \) cancer risk (p.119)

• \( \downarrow \) risk of developing stress-related disease (Puterman et al 2012)

• healthier semen in men (Vaamonde et al 2012)

• \( \uparrow \) cognition and longevity, \( \downarrow \) risk of dementia (Joyner & Barnes 2013)

• \( \downarrow \) falls (Yoo et al 2013).

Community-based exercise groups are particularly popular with elderly people (Reuter 2012).

Exercise is ‘a miracle drug’

Wen & Wu 2012
CASE STUDY: Ms LL

A 62 y.o. patient from Cape Town is admitted with an exacerbation of COPD. Answer the questions below.

Relevant medical history
- Heart failure
- Hypertension
- Increased dyspnoea for two weeks.

Subjective assessment
- Can't stop coughing
- Occasionally bring 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
- No oxygen charts
- Speaking sets off paroxysms of wheezy coughing, usually non-productive
- Clutches between legs when coughs
- Sits in chair day and night
- Can mobilize slowly
- Blood gases on air: PaO$_2$ 10.2 kPa (76.7 mmHg), PaCO$_2$ 6.4 kPa (48.1 mmHg), pH 7.4, HCO$_3^-$ 28 mmol/l.
Questions
1) Analysis?
2) Problems?
3) Goals?
4) Plan?

CLINICAL REASONING

Comment on the logic of the following conclusion to 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 CASE STUDY

1) Analysis
- Breathing pattern suggests ↑ work of breathing
- Blood gases indicate hypoxaemia, hypercapnia and compensated respiratory acidosis
- Oxygen therapy is uncontrolled
- Coughing is largely ineffective and contributes to fatigue
- Coughing, stress incontinence, immobility and fluid restriction are likely to be interrelated.

2) Problems
- Inaccurate and unmonitored oxygen
- Dyspnoea
- Uncontrolled cough
- Fatigue
- Anxiety
• Sputum retention
• Stress incontinence
• ↓ mobility.

3) Goals
• Short term: optimize oxygen, control cough, clear chest, balance rest and exercise
• Long term: educate patient and family for home management.

4) Plan
• Liaise with the team about the need for a Venturi mask, oxygen prescription and monitoring (Ch.5)
• Positioning for comfort, breathlessness and sputum clearance
• Identify cause of poor sleep e.g. dyspnoea/cough/noise/anxiety, then remedy as possible with the team
• Educate on cough suppression for use when cough is uncontrolled and nonproductive
• Educate on mucociliary clearance, including fluid intake. Patient chose manual techniques at first, then autogenic drainage
• Educate on effective cough for when secretions are accessible
• Explain connection between coughing and stress incontinence, teach preliminary pelvic floor exercises, refer to continence service
• Show breathless management strategies
• Mobilize to toilet
• Provide written daily programme for self chest management and mobility
• Liaise with ward staff re. getting dressed and mobilising
• Rehabilitate to independence, including family.

Response to clinical reasoning

It is not indicated to do an unnecessary treatment.
RECOMMENDED READING


Fronius M, Clauss WG, Althaus M (2012) Why do we have to move fluid to be able to breathe? Frontiers Physiol; 3:146


Harris RS (2005) Pressure-volume curves of the respiratory system. Respir Care; 50(1):78–99


Weibel ER (2013) It takes more than cells to make a good lung. Am J Respir Crit Care Med; 187(4):342–6
