Physiology for practice: the mechanisms controlling respiration

Breathing, or respiration, is something we do not have to think about – it just happens; indeed, if we do think about it, our breathing pattern changes. Nurses count and record patients’ respiration every day with perhaps little thought as to the physiological mechanisms that maintain this vital function, or to the wealth of information available from careful assessment of a patient’s breathing. Knowledge of these processes is important to be able to understand the symptoms, treatment and management of respiratory disease.

The medullary respiratory centre

Rhythmic breathing (normal, quiet breathing at rest or during sleep) is initiated by the respiratory centre in the medulla oblongata of the brainstem. This centre has two groups of neurones: a ventral group and a dorsal group. The dorsal group is sometimes referred to as the inspiratory centre because it acts as the respiratory ‘pacemaker’. The neurones may be self-excitatory (working automatically without the need for stimulus from nerve pathways) in a similar way to the cardiac cells at the sinoatrial node, although this is not certain (Marieb, 2003). Without other influences, these neurones switch on for approximately two seconds and off for three in a constant, rhythmic pattern. A simple calculation provides the number of breaths per minute which are generated by the inspiratory centre:

One minute = 60 seconds
One respiratory cycle = five seconds (two on, three off)
Respiratory rate per minute = 60/5
= 12 breaths per minute

In practice, the normal respiratory rate is 12–18 breaths per minute in adults and 18–20 breaths per minute in children (Martini and Bartholomew, 2003).

For breathing to happen, there must be a connection from the medullary inspiratory centre to the muscles of the lungs. The active inspiratory centre neurones stimulate the nerves to the inspiratory muscles, the phrenic nerve to the diaphragm and intercostal nerves to the external intercostal muscles. These muscles contract leading to expansion of the thorax so that air is drawn into the lungs. When the inspiratory centre neurones are not active, the stimulus to the muscles stops, the muscles relax and expiration occurs passively. This rhythmic pattern continues unless some other influence affects the inspiratory centre neurones and either increases or decreases breathing rate.

Respiratory rate is determined by the length of time the inspiratory centre is active before it is switched off. The depth of each inspiration is determined by the strength of the nerve stimulus to the muscles of respiration – the greater the stimulus, the greater the depth of respiration.

The ventral group of respiratory neurones in the medulla become more active during forced respiration, especially forced expiration (Marieb, 2003). Impulses from the ventral neurones travel via nerves to the muscles involved in forced expiration, notably the internal intercostal muscles (supplied by intercostal nerves) and abdominal muscles (supplied by the vagus nerve). This group of neurones appears to play a role in inspiration as well as expiration, but the precise mechanism is unclear (Marieb, 2003) (Fig 1).
In the absence of other stimuli, respiration would continue in this same rhythmic pattern throughout life. This would not be a problem if people sat quietly every day but respiratory rates alter with activities such as running, climbing and singing, laughing or crying. In order to help us to tailor our breathing to meet the needs of everyday life, a number of physiological mechanisms influence this basic breathing pattern.

**Influence from the pons** Centres in the pons (close to the medulla in the brain stem) influence the respiratory neurones in the medulla (Fig 1). The pontine respiratory group of neurones (once known as the pneumotaxic centre) is responsible for the ‘fine-tuning’ of our breathing and for preventing overinflation of the lungs. It achieves this by sending constant inhibitory impulses to the inspiratory centre in the medulla to limit the period of inspiration.

**Influence from the lungs** The lungs contain stretch receptors (or baroreceptors) which also appear to influence respiration. When the lungs expand during inspiration, stretch receptors in the lung walls are activated and act via the vagus nerve to inhibit the inspiratory centre in the medulla oblongata and allow reflex expiration to occur (Bourke, 2003). These receptors are particularly important in animals and in young babies who have a poorly organised brainstem (Stocks, 1996) but their role in adults remains uncertain, especially during quiet respiration. Marieb (2003) suggests that this mechanism is probably protective rather than regulatory.

Other receptors in the lungs are sensitive to irritants such as gases, debris, inhaled foreign bodies and excess mucus. When they are activated, these receptors influence the respiratory centre via the vagus nerve so that coughing can occur to clear the irritant.

**Influence from the higher brain centres** The higher centres of the brain are the areas where we understand and manipulate information and experience thoughts, feelings and emotions. These centres can also influence respiration.

Respiratory rate and depth alter when the centres of the limbic system involved with emotions such as pain, anger or excitement are activated, though this effect is involuntary and outside our control. Centres in the hypothalamus are activated and influence both the rate and the depth of respiration via the pons (Martini and Bartholomew, 2003) and the medullary inspiration centre. Respiration can be increased or decreased via this pathway. Examples of this mechanism in action include gasping with fear or cold, a rise in respiratory rate when the body temperature is high, and breath-holding during times of anger.

From the cerebral cortex we can also voluntarily change our respiratory pattern by sending signals direct to the muscles of inspiration and bypassing the medullary centres (Marieb, 2003). The cortex is the area of the brain where we interpret and manipulate information and when we need, for example, to swim a length under water, sing or simply chat to friends, we can consciously control our breathing pattern. Many of us, in our younger days, tried to hold our breath until we collapsed, but it is impossible to alter our breathing beyond certain limits because the other respiratory control mechanisms ultimately override the influence of the higher centres.

**Chemical influences** Perhaps the most important influence on respiratory rate and depth are chemicals. Specialised receptors (chemoreceptors) respond to chemical changes in the blood and cerebrospinal fluid (CSF). Peripheral chemoreceptors in the aortic arch and the carotid bodies respond to changes in the oxygen (O₂), carbon dioxide (CO₂) and acidity (pH) levels in arterial blood.

Central chemoreceptors in the medulla oblongata respond to changes in arterial CO₂ levels and in the pH level of CSF. It is these chemoreceptors that are ultimately responsible for the homeostasis of O₂ and CO₂ levels in the blood. They ensure that there is adequate oxygen circulating for the needs of cells throughout the body and that the waste products of cellular metabolism, carried as CO₂, can be deposited in the lungs. Arterial pressures of O₂ and, especially, CO₂ are maintained within narrow limits despite large changes in consumption and production.

Normally it is a small rise in arterial CO₂ that triggers these chemoreceptors and results in a negative feedback homoeostatic response to reduce these levels. The CO₂ diffuses from the blood into the CSF where there is very little protein to mop up or ‘buffer’ the acid produced. As a result, the CSF rapidly becomes more acidic and its pH falls. This increased acidity stimulates the central chemoreceptors, which act directly upon the medullary and pontine centres to increase both the rate and depth of respiration by increasing the strength and duration of neuronal impulses from the inspiratory centre to the muscles of inspiration. The result is that the excess CO₂ is blown out of the lungs. When arterial CO₂ and CSF pH levels return to normal, the response ceases (Fig 1) and rhythmic respiration resumes.

**REFERENCES**


FIG 2. CONTROL OF RESPIRATION

Peripheral chemoreceptors, bathed as they are in newly oxygenated blood, are sensitive to arterial \( O_2 \) levels. While they are involved in the response to increased acidity (rise in arterial partial pressure of \( CO_2 \), fall in pH) they also respond to falls in the arterial partial pressure of oxygen (\( PO_2 \)). There are vast reserves of arterial oxygen bound to haemoglobin in the blood (Richardson, 2002), and a large fall in \( PO_2 \) is needed before these begin to be depleted and the peripheral chemoreceptors are stimulated. Neuronal messages via the glossopharyngeal nerves (from carotid receptors) and the vagus nerve (from the aortic receptors) stimulate the medullary inspiratory neurones. Rate and depth of respiration are increased and more \( O_2 \) is inhaled and absorbed into the blood. Once arterial \( O_2 \) levels return to normal, the stimulus ceases.

These peripheral receptors assume a vital importance in patients who retain \( CO_2 \) due to pulmonary disease such as emphysema or chronic bronchitis. In these patients, the central chemoreceptors become unresponsive to the constant stimulus of \( CO_2 \) and the peripheral chemoreceptors assume the function of driving respiration (the hypoxic drive). These patients will only breathe when arterial \( PO_2 \) is low enough to trigger the peripheral chemoreceptors. It is essential that nurses understand this physiological alteration as giving high doses of oxygen therapy to these patients will stop them breathing because \( O_2 \) levels do not fall low enough to stimulate respiration.

Influence from hormones Hormones are not only involved in the transmission of nerve impulses within the respiratory system, but recent work suggests that many are involved in the control of respiration (Saarela and Polo, 2002). Progesterone and thyroxine, for example, are known to stimulate respiration, while somostatin and dopamine have a depressant effect.

Influence from drugs and medications Many different drugs affect our respiratory rate. Barbiturates, alcohol, anaesthetics and opiates have a depressant effect, while stimulants such as caffeine and amphetamines increase respiratory rate. A variety of mechanisms are involved and readers are referred to specialist respiratory pharmacology texts.

Conclusion The simple act of breathing in and out is regulated by numerous physiological mechanisms (Fig 2). This complicated system enables us to adjust our respirations with great precision, ensuring that every cell in the body receives a constant supply of oxygen and has a means of ridding itself of waste products.