The respiratory system Part 4: breathing

This article, the last in a four-part series on respiration, examines breathing. Respiration refers to the exchange of gases between a living organism and its environment, while breathing is the process that moves air into and out of the lungs (Thibodeau and Patton, 2005). Breathing (or pulmonary ventilation) has two phases - inspiration (or inhalation) and expiration (or exhalation). It is a mechanical process that depends on volume changes in the chest cavity. The volume changes result in pressure changes, which lead to the flow of gases to equalise the pressure.

Molecules in a gas, such as air, move about freely and collide with one another and this exerts pressure. Anything that increases the number of collisions (for example, a rise in temperature increases the speed at which molecules travel) will cause a rise in gas pressure and vice versa. When gases move from an area of high pressure to an area of low pressure, this is known as ‘bulk flow’. During a breathing cycle, air moves in and out of the lungs by bulk flow. The respiratory muscles are responsible for the changes in the shape and volume of the chest cavity that cause the air movements in breathing.

At the start of a breath, pressure inside and outside the lungs is equal.

**Inspiration**

Inspiration or inhalation is an active process that occurs when the chest cavity enlarges because of the contraction of the muscles. The dome-shaped diaphragm is the most important muscle at this stage. At the start of inspiration, the diaphragm contracts and flattens, pressing down on the abdominal contents and lifting the ribcage. This increases the vertical height of the thoracic cavity (Fig 1).

At the same time, the external intercostal muscles between the ribs contract and lift the ribcage and pull the sternum forward, thus increasing the front-to-back and side-to-side dimensions (Figs 1–2).

The outer (parietal) layer of the pleura is attached to the diaphragm and the inside of the chest wall,
and so moves with those structures. This causes the interpleural pressure (NT Systems of Life, 6 June, p22) to fall from -5cmH₂O to about -9cmH₂O. As a result, the inner (visceral) layer of the pleura, which is attached to the surface of the lungs, follows and the lungs expand, that is their volume increases. The air in the lungs now has a larger space to fill and so its pressure falls. This produces a partial vacuum, which sucks air into the lungs by bulk flow. Air continues to move into the lungs until the intrapulmonary pressure is the same as atmospheric pressure.

During forced inspiration, the accessory muscles in the neck may also be used to elevate the sternum and first two ribs (Fig 1). This, combined with the maximal contraction of the inspiratory muscles, leads to the generation of a much more negative intrapleural pressure (for example, -30cmH₂O) and more rapid airflow (McGeown, 2002).

Normally about 500ml (1 pint) of air is moved in and out per breath – this is known as the tidal volume.

Expiration

In healthy people quiet expiration or exhalation is passive and relies on elastic recoil of the stretched lungs as the inspiratory muscles relax, rather than on muscle contraction. The diaphragm and external intercostal muscles return to their resting position and the volume of the chest cavity and of the lungs decreases. This ‘squashes’ the air in the lungs and raises its pressure above atmospheric air. Thus, air is driven out of the lungs by bulk flow, until the atmospheric pressure and pressure within the alveoli are equal.

Normally, expiration is effortless but if the respiratory passageways are narrowed by spasm of the bronchioles (for example, in asthma) or clogged with mucus or fluid (for example, in chronic bronchitis or pneumonia), expiration becomes an active process (Law and Watson, 2005).

In forced expiration, when it is necessary to empty the lungs of more air than normal, the abdominal muscles contract and force the diaphragm upwards and contraction of the internal intercostal muscles actively pulls the ribs downwards. This generates higher air pressures within the lungs and forces the air out more rapidly.

Quiet expiration normally ends when alveolar pressure is again equal to atmospheric.

Control of respiration

Although breathing is simple mechanically, its control is complex. The respiratory control centre is situated in the medulla oblongata of the brain. This sets the rhythm of breathing and contains neurons that are self-excitatory (rather like the cells in the sino-atrial node in the heart) and which fire off in a cycle. This maintains the normal respiratory rate of 12–15 breaths a minute.

When the inspiratory neurons in the medulla fire, they excite the muscles of inspiration – the phrenic nerve to the diaphragm and the intercostal nerves to the intercostal muscles – causing them to shorten and enlarge the volume of the chest cavity. When the medullary neurons stop firing, the muscles recoil and the chest cavity returns to its resting size.

During exercise we need to deliver more oxygen than normal to the tissues. The brain centres send more impulses to the respiratory muscles and we breathe more deeply and quickly. During forced expiration, areas in the medulla fire off impulses that contract the muscles of forced expiration – abdominal muscles and the internal intercostals.

A number of factors influence the rate and depth of respiration:

- We have a limited amount of voluntary control over respiration. For example, we can control expiration while talking or singing;
- If the lungs begin to overinflate, stretch receptors in the bronchioles and alveoli are triggered and switch off the respiratory centre so that air is expelled and the lungs return to normal;
- Chemical factors play a very important role. Blood pH and levels of oxygen and carbon dioxide are constantly monitored by specialised chemoreceptors. A rise in CO₂ and a resultant fall in pH will increase the rate and depth of breathing, so that CO₂ is blown off and the levels return to normal. These changes seem to act directly on the medullary centres. A fall in blood oxygen levels also sends impulses to the medulla to increase the rate and depth of breathing but usually only when they are very low.

REFERENCES


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