Measurement of respiratory function: gas exchange

Iain Campbell
James Waterhouse

Blood normally spends about 0.75 s in the alveolar capillary. Equilibration of carbon dioxide occurs in about 0.25 s and of oxygen in about 0.4 s. Therefore, equilibrium occurs rapidly and is normally complete when the blood has traversed only about half the capillary. Within the pulmonary capillary there is thus a large reserve for gas transfer. Although pulmonary blood flow may rise fivefold with exercise, gas transfer (at sea level) is still normally complete. The pulmonary circulation also has a large reserve. With increases in cardiac output, pulmonary capillaries have the capacity to expand (distension) and capillaries that are closed at rest or have no blood in them, open up (recruitment).

The carbon monoxide transfer test is used in chest medicine as a noninvasive method of quantifying gas transfer. Carbon monoxide is more soluble in blood than carbon dioxide or oxygen. When it is present in inspired air, equilibrium is not reached by the time the blood has perfused the alveoli. No appreciable loss of diffusion gradient develops in the alveolus and the gas continues to move rapidly into the bloodstream, therefore its uptake is normally independent of blood flow, and thus limited by diffusion. Its rapid uptake from the alveolus makes it a sensitive test of gas transfer. It is considered principally as an index of diffusion, but probably is also affected by ventilation–perfusion abnormalities. The carbon monoxide transfer test is not widely used in anaesthetic practice.

Measurement of ventilation ($\dot{V}_A$) and perfusion ($\dot{Q}$) involves the inhalation or injection of radioactive isotopes, respectively. These methods are in common use in chest medicine to assess localized abnormalities. Multiple gas elimination is a research technique that allows ventilation–perfusion abnormalities to be quantified precisely. It involves injection of tracer gases of varying solubilities and allows distribution curves to be plotted for pulmonary blood

Iain Campbell *is Consultant Anaesthetist at the University Hospitals of South Manchester NHS Trust and Visiting Professor of Human Physiology at Liverpool John Moores University. He qualified from Guy's Hospital Medical School, London, and trained in anaesthesia in Zimbabwe, Southend, Montreal and Leeds.*

James Waterhouse *is Professor of Biological Rhythms at the Research Institute for Sport and Exercise Sciences, Liverpool John Moores University. He qualified as an animal physiologist at the University of Oxford and gained his DPhil there. His research interests are circadian rhythms in humans, with particular reference to jetlag and assessing the clock-driven component of a measured circadian rhythm.*

flow and ventilation in relation to $\dot{V}_A$:$\dot{Q}$ ratios. Such techniques are not in routine use in anaesthetic practice and are beyond the scope of this article, which focuses on the common clinical techniques used to measure ventilation and gas exchange, and the derivation of parameters that can be calculated from them.

Blood gases

The ultimate test of how well ventilation is matched to perfusion is the measurement of blood gases. Inadequate ventilation (hypoventilation) is always accompanied by a raised partial pressure of carbon dioxide in arterial blood ($PaCO_2$). Mismatch of ventilation to perfusion results in hypoxaemia. $PaCO_2$ does not rise with ventilation–perfusion abnormalities unless ventilation is inadequate, because of depression of the respiratory centre (e.g. opioids) or pathology of the ventilatory apparatus (e.g. muscle paralysis, crush injury of the chest).

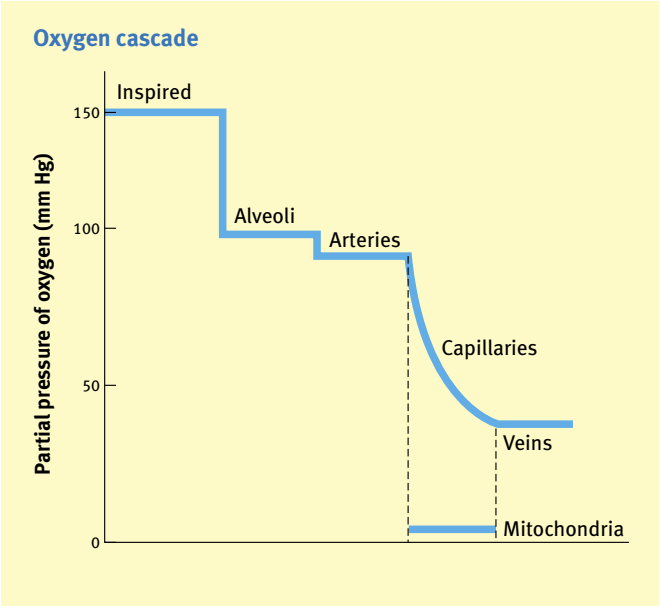
$PaCO_2$ is a function of CO_2 production ($\dot{V}CO_2$) and alveolar ventilation ($\dot{V}_A$) and is given by the relationship:

$$PaCO_2 = \dot{V}CO_2/\dot{V}_A \times K$$

where K is a constant. Thus, halving $\dot{V}_A$ doubles $PaCO_2$.

The oxygen cascade

Figure 1 illustrates how the partial pressure of oxygen (PO_2) falls as gas is moved from the inspired air to the tissues. Inspired air is humidified in the upper airways and gas exchange with the alveoli is a function of ventilation. From the alveoli, oxygen moves into the arteries. The difference between PO_2 in the alveoli (P_AO_2) and in the arteries (PaO_2) is a function of ventilation–perfusion relationships and, in some chest diseases, diffusion across the alveolar capillary membrane. From the arteries, gas exchange with the peripheral tissues occurs in the capillaries. O_2 and CO_2 diffuse through the tissues. Most oxygen is used, and most CO_2 produced, in the mitochondria where PO_2 is very low (1–4 mm Hg).



1

Inspired PO_2 (P_IO_2) is calculated from barometric pressure (760 mm Hg at sea level) and the proportion of O_2 in inspired air (F_IO_2; for atmospheric air 20.9%) allowing for humidification of the inspired air; the saturated vapour pressure of water at 37°C is 47 mm Hg. Thus:

$$P_IO_2 = (760 - 47) \times 20.9/100 = 149 \text{ mm Hg}$$

The decrease in PO_2 from the inspired air to the alveolar air is due to dilution by the CO_2 in the alveoli, but the body produces less CO_2 than it uses O_2. The ratio of CO_2 produced to O_2 consumed is given by the respiratory quotient or respiratory exchange ratio (R). This is normally 0.7–1. It is seldom measured and a value of 0.8 is normally assumed. Based on this, P_AO_2 can be calculated from the simplified form of the alveolar air equation:

$$P_AO_2 = P_IO_2 - P_ACO_2/R$$

P_ACO_2 can be taken as end-tidal CO_2, though it is more usual to use $PaCO_2$. In terms of the figures quoted above:

$$P_AO_2 = 149 - 40/0.8 = 99 \text{ mm Hg}$$

P_AO_2 can be influenced by raising or lowering the F_IO_2 or by changing ventilation, which changes $PaCO_2$ and thus alters the P_AO_2 in line with the alveolar air equation. O_2 uptake from the alveoli may also have an effect, particularly in patients with respiratory impairment and a raised oxygen consumption caused by shivering, pain, anxiety or fever.

The difference between P_AO_2 and PaO_2 (the alveolar–arterial oxygen difference or gradient) is due mainly to ventilation–perfusion mismatch, specifically alveoli over-perfused relative to their ventilation (i.e. $\dot{V}_A$:$\dot{Q}$ < 0.8) ranging from alveoli with a $\dot{V}_A$:$\dot{Q}$ ratio only slightly less than 0.8 to alveoli that are perfused and not ventilated at all (i.e. $\dot{V}_A$:$\dot{Q}$ = ∞; atelectasis, lobar collapse, pneumonia). In health (in the upright position), the alveoli with $\dot{V}_A$:$\dot{Q}$ ratios less than 0.8 occur in the lower part of the lung. There is a gradation of $\dot{V}_A$:$\dot{Q}$ ratios from about 3.3 at the top (over-ventilated and under-perfused) to 0.63 at the base (under-ventilated and over-perfused). This distribution of $\dot{V}_A$:$\dot{Q}$ ratios leads to a spread of P_AO_2 values, with those at the top of the lung being high (about 130 mm Hg) and those at the base being low (about 80 mm Hg). With such a spread of P_AO_2 values how can one justify using a single figure? The figure given by the alveolar air equation is conventionally taken to be an average value for P_AO_2 over the whole lung. However, the figure is heavily weighted by basal alveoli, which is where most of the ventilation (and perfusion) takes place. It is also known as the ideal P_AO_2.

The principal factor affecting ventilation and perfusion in the lung is gravity in relation to the weight of the lung and the low pressures in the pulmonary circulation. All of the above arguments about the distribution of ventilation and perfusion have applied to the conscious individual in the upright position. When the individual lies down gravity still applies, so the areas of high $\dot{V}_A$:$\dot{Q}$ ratios move to the anterior aspect of the chest and of low $\dot{V}_A$:$\dot{Q}$ to the posterior. Airway closure then takes place posteriorly rather than at the base.