

Oxygen saturations and pulse oximetry

Edited by the perioperative CPD team

The pulse oximeter provides an indication of oxygen status in the body. However, the link between a pulse oximeter reading and the events occurring in the body can be complex and difficult to evaluate. We all require an understanding of factors associated with oxygen uptake and delivery to be able to use pulse oximetry in a knowledgeable way.

Introduction

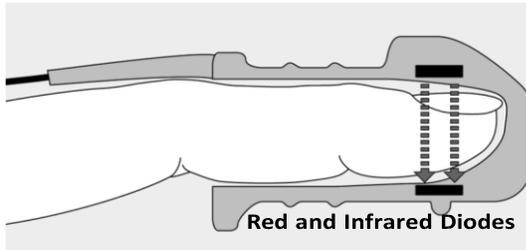
The pulse oximeter allows cost-effective monitoring of arterial oxygen saturation, reducing the need for invasive arterial blood gas measurement, and allowing rapid, easy assessment of oxygen status. Early warning of low blood-oxygen levels (hypoxaemia) has enhanced practice in a variety of clinical settings and provides reassurance in regards to oxygenation status.

Critical evaluation of the information being provided by the pulse oximeter is essential for safe practice. Unlike the thermometer, pulse oximeters are complex machines and the physiological processes they assess are also complex.

Often there is a lack of understanding of the underlying physiology of oxygenation that can affect evaluation of saturation readings and thus the care delivered.

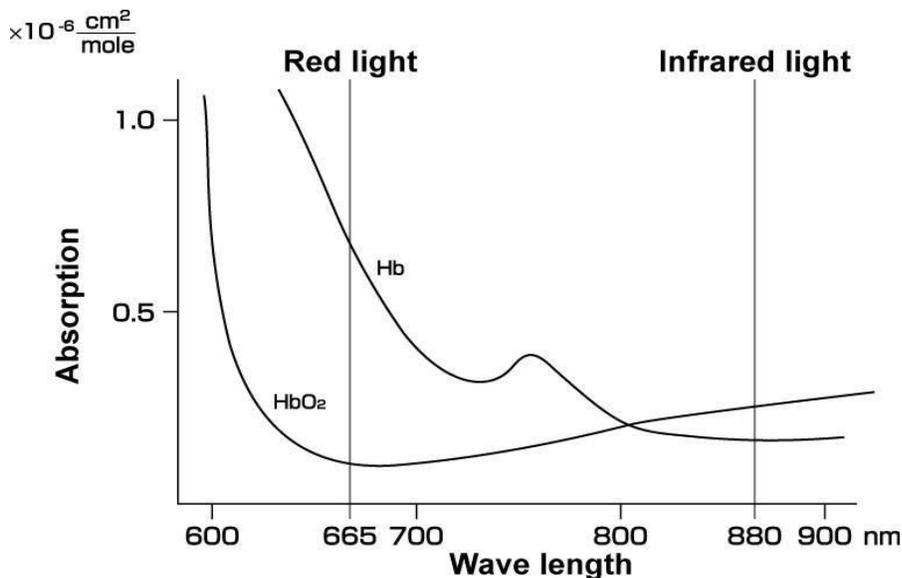
Pulse oximeters

The pulse oximeter was developed in Japan in the mid-1970s but only entered clinical practice a decade later. It projects light in two wavelengths, measuring absorption in pulsatile blood by saturated haemoglobin (visible red) and desaturated haemoglobin (infrared wavelength). This information is then compared with a database of saturation values taken from healthy volunteers given hypoxic gas concentrations to breathe, and a saturation percentage is provided. Since there are ethical issues in forcing desaturation below 70% in volunteers, accuracy of machines below this level is questionable.



Accuracy of pulse oximeters depends on how frequently they are calibrated, but is around [+ or -] 2% for values over 90 percent. Values under 80% may be less accurate. This is also true for pulse oximetry in the paediatric and neonatal populations.

Because pulse oximeters use light and colour to determine saturation, their accuracy can be affected by a number of external factors: black, green or blue nail polish on the patient's finger will affect readings, as does the presence of dyes in the blood, but jaundice does not. Bright ambient or fluorescent light may also affect readings. Other factors affecting accuracy of pulse oximetry will be discussed further below.



Oxygen & Hypoxia

Pulse oximetry readings are used to try to determine how much, and how well, oxygen is being delivered to body cells. Oxygen is essential for the manufacture of adenosine triphosphate (ATP), the energy source in all body cells. In the absence of oxygen, some body tissues, e.g. skeletal muscle, are able to continue making ATP through anaerobic metabolism. This leads to a build-up of lactic acid that, if prolonged, causes cell damage and death (ischaemia and necrosis).

The brain and myocardium are most vulnerable to hypoxia because the cells in these tissues are unable to effectively utilise anaerobic pathways for ATP generation.

Signs of systemic hypoxia include fatigue, restlessness, lethargy and confusion, as oxygen to the central nervous system is reduced. Other signs indicate compensatory mechanisms: increased heart rate, pallor, and increased rate and depth of respirations. It should be noted that these signs are not always present and can be lost as hypoxia worsens.

Cyanosis

The most frequently mentioned sign of hypoxia is cyanosis. Cyanosis is an unreliable, and late, indicator of hypoxia, generally occurring where saturation of haemoglobin (Hb) falls below 80%. The darker colour of unsaturated Hb gives rise to a bluish-purple hue in affected tissues, most visibly nail beds, skin and mucous membranes (gums, tongue and under the eyelids). Central cyanosis indicates low arterial oxygen content. Peripheral cyanosis occurs where circulation has slowed and/or there is increased extraction of oxygen from Hb. This can be as a result of poor circulation to a specific region, eg with peripheral vascular disease. Other causes are shock, cord exposure or heart failure.

Areas of the body that remain warm, or retain circulation in shock states (mouth, eyes) should be checked when looking for signs of cyanosis. If extremities are observed, they should be warmed to increase blood flow. There may be difficulty in detecting cyanosis in people with darker skins or jaundice, or in poor or fluorescent light. Someone with severe anaemia may not have enough Hb to appear cyanotic even with low saturations. Overall, the pulse oximeter will detect low oxygenation more reliably and earlier than observation for signs such as cyanosis.

Oxygen delivery

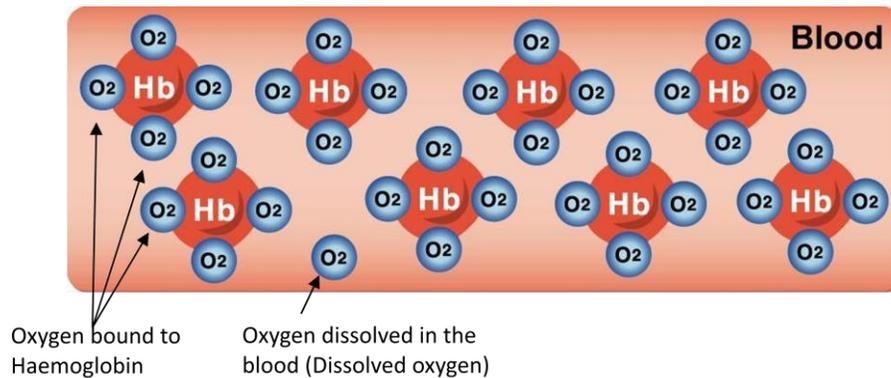
It is easy to forget that while pulse oximeters measure the amount of oxygen being carried by Hb in arterial blood, the real purpose of this assessment is to determine how much oxygen is reaching the body cells. However, the amount of oxygen being delivered to the tissues cannot be determined solely using pulse oximetry.

Delivery of oxygen, to body cells for use in aerobic metabolism is essential for life. It is determined by three factors:

- * The amount of oxygen being carried by the Hb (oxygen saturation).
- * The amount of Hb present in the blood (Hb concentration).
- * How well the Hb is being transported around the body (cardiac output).

Oxygen concentration

A single red blood cell contains more than 600 million Hb molecules. Each Hb molecule contains four iron atoms, each of which binds a single molecule of oxygen. The concentration of free oxygen in the plasma determines the binding of oxygen to Hb. Arterial plasma oxygen concentration (measured by the arterial blood gas test) is reported as a pressure in either millimetres of mercury (mmHg) or kilopascals (kPa).



The pressure of oxygen in the plasma is determined by the movement of oxygen from the lungs into the plasma via diffusion, as the blood passes through the pulmonary capillaries. The rate at which oxygen diffuses from alveoli into the blood is determined by three main factors:

- 1) Concentration gradient for oxygen.
- 2) Surface area available for diffusion.
- 3) Thickness of the alveolar-capillary membrane.

These factors are subject to alteration through disease processes and can also be changed by clinical interventions.

Concentration gradient: For a gas or liquid, diffusion occurs where there is a difference in pressure or concentration between two different areas. It helps to think of the gas or liquid as a ball rolling down a slope. The larger the difference in concentration between the two areas, the steeper the slope, and so the faster the ball will roll. A smaller difference in concentration means a shallower slope and so slower diffusion.

There is normally a steep concentration gradient for oxygen between the alveoli and the blood in the pulmonary capillaries. When we inhale, air moves into the exchange portion of the lungs (the alveoli). This inhaled air contains 21% oxygen. The pressure of the oxygen in the alveoli can be calculated thus:

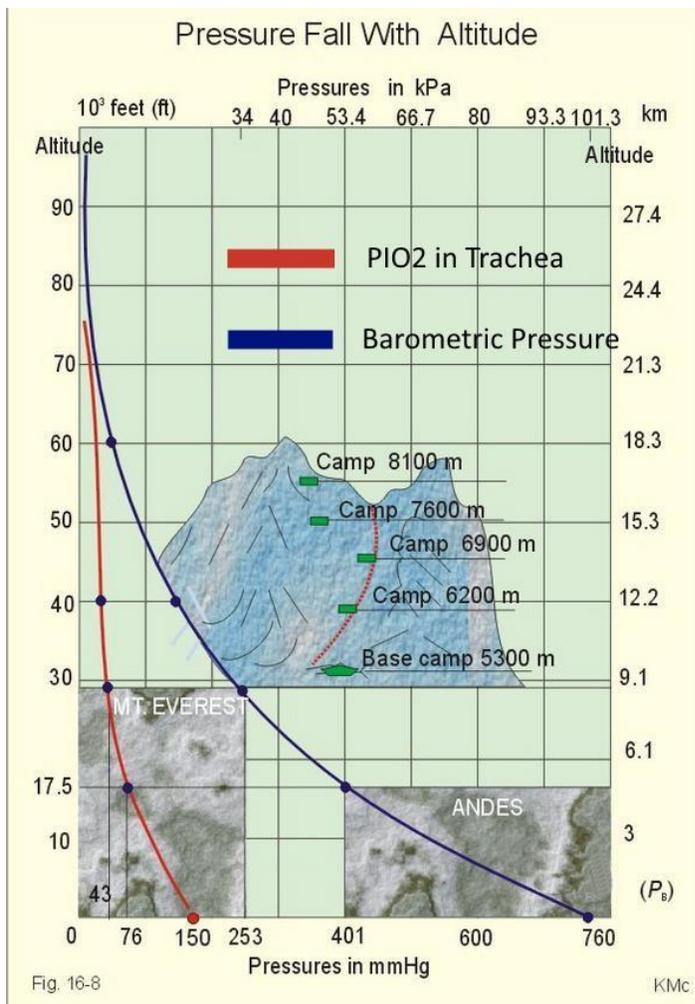
- * At sea level, atmospheric pressure is around 760mmHg (101kPa).
- * When we inhale, air gets mixed with about 46mmHg (6kPa) of water vapour as it is humidified. This alters the available oxygen to 21% of 713mmHg (95kPa), giving an oxygen concentration of around 150mmHg (20kPa).
- * By the time the air reaches the alveoli, this figure has fallen to around 100mmHg (13kPa). This is because the inhaled air mixes with dead space air (air in the trachea and bronchi where gas exchange does not occur) and residual air that was not exhaled after the previous breath.

Blood coming into the lungs via the pulmonary arteries has an oxygen concentration of about 40mmHg (5kPa)--the same as venous blood. There is a steep concentration gradient for oxygen to diffuse from the alveoli (100mmHg) to the pulmonary capillaries (40mmHg). In a healthy adult, at rest, where blood passes through the pulmonary capillaries in about three-quarters of a second, an equilibrium is easily reached by rapid diffusion, so that blood leaving the lungs has an oxygen concentration of about 100mmHg.

Figure 1 gives examples of changes to oxygen concentration in inspired air with increasing altitude. For each of these altitudes, the calculation is the same: barometric pressure less water vapour (46mmHg/6kPa) multiplied by 21%.

The proportion of oxygen in the air remains the same no matter how high you go (21%), but the pressure changes with atmospheric pressure.

Figure 1:



At the top of Mount Everest, the pressure of inspired oxygen is 42mmHg (33kPa). This drops as the inspired air mixes with dead space and residual air, although not as much as during normal breathing at sea level. Blood coming to the lungs in the pulmonary arteries will also have lower oxygen than at sea level, as more is extracted by hypoxic tissues but, as you can see, there is very little gradient for diffusion of oxygen. At this altitude most climbers use oxygen, and those who don't, spend considerable time at slightly lower altitudes getting acclimatised.

If you look at the figures for a depressurised jet plane, you will see why they recommend putting your own oxygen mask on before tending to others around you. The sudden drop of inspired oxygen levels to 30mmHg (4kPa) gives a person about 30 seconds before losing consciousness.

Another way to alter the concentration gradient for the diffusion of oxygen is to increase the percentage of inspired oxygen. We do this when we provide oxygen therapy.

For example, if a patient was prescribed oxygen at 28% (2 litres per minute via nasal cannula), the pressure of inspired oxygen (at sea level) becomes:

$760\text{mmHg (101kPa)} - 46\text{ mmHg (6kPa)} = 714\text{mmHg (95kPa)}$

Multiply by 28% = 199mmHg (26.7kPa)

Compare with 250mmHg/20kPa at 21% (room air).

Can you calculate the increase in concentration gradient and thus the rate of diffusion for a person receiving oxygen at 40% (6 L/min via simple face mask) and 24% (1 L/min via nasal cannula)? Essentially, by providing oxygen, we are increasing the steepness of the slope for oxygen to roll down, so it enters the pulmonary capillaries faster and in larger amounts than if breathing room air. This can compensate for changes in other factors that affect diffusion rates and thus provides the most basic treatment for hypoxia and hypoxaemia.

Surface area: The surface area available for diffusion of oxygen is determined by the number of alveoli in the lungs receiving both air and blood flow. In adults this area is between 50 and 75 square metres.

Surface area for diffusion is affected by normal physiological factors, such as posture, and by pathological conditions. When standing upright, upper alveoli receive good airflow but poor circulation, whereas the alveoli in the base of the lungs have good blood supply but are less well ventilated. Increasing the pulmonary arterial pressure (eg during exercise) will increase blood flow to capillaries of the upper alveoli, increasing the surface area for diffusion.

Diseases or conditions that decrease air or blood flow in the lungs will decrease the surface area for diffusion and so reduce the amount of oxygen diffusing into the blood and binding to Hb.

Examples of conditions that reduce blood flow to the alveoli are pulmonary embolism, tumours compressing vessels in a specific area and, in neonates, patent ductus arteriosus. Reduced airflow occurs with obstructive airway disease, e.g. chronic bronchitis or bronchiectasis. It also occurs with pneumothorax or surgical removal of a lung or lobes.

Thickness of the alveolar-capillary membrane: This membrane separates the air in the alveoli from the blood in the pulmonary capillaries. It is normally very thin (1 micron), allowing such rapid diffusion of oxygen that blood is fully saturated before it has traversed one-third of the pulmonary capillary. Conditions which increase the thickness of the barrier, or the distance between air and blood, reduce the rate of diffusion. The accumulation of fluid in the alveoli caused by pulmonary oedema is one example. Sarcoidosis and asbestosis also cause thickening of the membrane due to scarring.

Blood leaving the pulmonary capillaries will normally have equilibrated with alveolar oxygen concentrations. Pressure of oxygen in the blood will be about 100mmHg . As blood enters the pulmonary veins to return to the left side of the heart, it mixes with blood from unventilated portions of the lungs and the oxygen falls to around 95mmHg . At this concentration, oxygen will bind to about 97% of the Hb in the red blood cells.

Venous saturation: Venous blood contains about 40mmHg of oxygen. At this concentration, Hb saturation is about 70%. There is, therefore, a substantial reserve of oxygen available to meet increased tissue demand for oxygen, eg during vigorous exercise. Falling Hb saturation in the venous blood indicates that the supply of oxygen is not meeting the tissue demand.

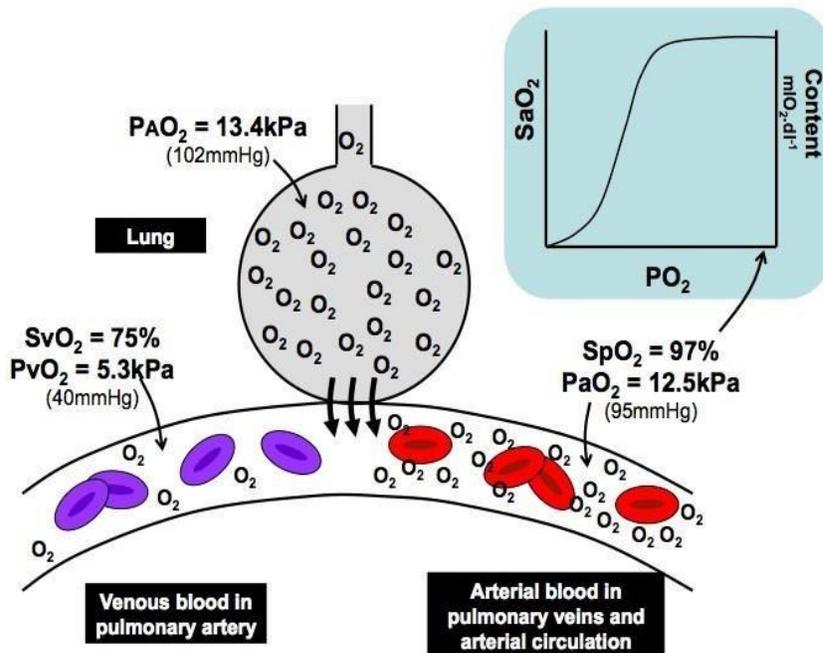
Accurate measurement of venous saturation from the whole body requires pulmonary arterial catheterisation. Venous saturation can be assessed from a central venous catheter placed in the superior vena cava, but this only provides data for blood returning from the brain and upper body. Neither of these is readily accessed outside the intensive care unit.

Concentration of haemoglobin

Normal values of Hb in the blood vary with age and sex. For adult males, concentration of Hb is between 135-175 grams per litre of blood (g/L). In females it is 115-155g/L (or 11.5-15.5 grams per decilitre). The total volume of plasma affects the results, as Hb is measured relative to this volume.

So, for example, in pregnancy there is a natural drop in measured Hb, because the volume of plasma increases. If a person is severely dehydrated, Hb concentration is increased, not because there is an increase in the number of their Hb molecules, but because there is more per litre of plasma present.

In the healthy adult, production of red blood cells occurs at a rate equal to their destruction: approximately 2.5 million red blood cells every second. This enormous turnover of cells demands an



adequate supply of nutrients: iron for haem synthesis, amino acids for globin, and folic acid and vitamin B12 for manufacture of cell nuclei.

Deficiencies in any of these will affect red blood cell production and may lead to anaemia. Anaemia also occurs as a result of increased destruction of red blood cells (eg in sickle cell disease), increased loss (arising from chronic or acute blood loss), or decreased production due to pathophysiological conditions of the bone

marrow or decreased erythropoietin as seen in chronic renal failure. The manifestations of anaemia relate largely to impaired oxygen delivery to the tissues. Fatigue, pallor, dyspnoea and tachycardia occur because of poor oxygenation and the body's attempts to compensate for this. It should be remembered that anaemia is a symptom of another condition and therefore the underlying causative process should be investigated.

The total amount of oxygen carried by Hb can be calculated. This can then be used to determine the effect of decreased Hb on oxygen transport. Each gram of Hb is capable of carrying 1.34 mL of oxygen. If we know a person's Hb concentration, we can calculate their total oxygen-carrying capacity.

As an example, in a person with 140g/L of Hb:

$140 \times 1.34 = 187.6\text{ml/L}$ maximum oxygen carrying capacity

Once you have calculated this data, you will see that maximum oxygen-carrying capacity varies considerably between patients. However, not every molecule of Hb in the blood is fully saturated, ie carrying its maximum four oxygen molecules. You can determine how much oxygen is actually being carried in the Hb by multiplying the maximum capacity by the percent saturation.

Carbon monoxide: Falsely high readings can be obtained from pulse oximeters in the presence of carbon monoxide (CO). Sources of CO include car exhaust, prolonged exposure to heavy traffic environments, gas heating and cooking. A single cigarette can increase CO levels in the blood for up to four hours. Heavy smokers (more than 20 cigarettes per day) can have up to seven percent of their Hb occupied by CO rather than oxygen. CO binds to Hb about 250 times more strongly than oxygen. Once in place, it prevents oxygen from binding and, because it binds so strongly, it is very slow to be removed. Carboxyhaemoglobin is bright red and most pulse oximeters cannot distinguish between it and oxyhaemoglobin, producing falsely high readings. Newer, multiple wavelength oximeters can distinguish carboxyhaemoglobin from oxyhaemoglobin, as well as give an indication of the total amount of Hb present.

Cardiac output

Even with normal Hb levels and saturation, oxygen cannot be delivered to the tissues if circulation is compromised. Cardiac output is the third factor that should be taken into account when monitoring oxygen status. It is uncommon to see direct monitoring of cardiac output outside the intensive care unit, but it is possible to assess cardiac function to some degree, using a patient's pulse and blood pressure readings.

Cardiac output is the amount of blood ejected from the ventricle each minute. It is determined by measuring the volume of blood ejected at each contraction (stroke volume) and multiplying this by the number of beats per minute (heart rate). The average adult male has a cardiac output of about 5 litres per minute (L/min), based on a stroke volume of around 70ml per beat and a heart rate of about 70 beats per minute.

We cannot measure stroke volume at the bedside but it can be estimated by looking at the difference between the diastolic and systolic blood pressure readings (the pulse pressure). A person with a blood pressure of 120/80 has a pulse pressure of 40 mmHg. A smaller pulse pressure indicates a lower stroke volume. Higher pressures indicate larger stroke volumes as with exercise. High pulse pressure can occur as a result of decreased aortic compliance due to arteriosclerosis, in which case it cannot be used as an indicator of cardiac output.

Stroke volume can also be evaluated when taking a person's radial pulse measurement. A firm or strong pulse indicates good stroke volume, while a weak or thready pulse indicates low stroke volume. An elevated pulse rate may indicate falling stroke volumes because the heart rate needs to increase to maintain cardiac output. Factors affecting stroke volume can be divided into two main categories: those involving changes in the amount of blood returning to the heart (venous return), and those affecting the ability of the heart to contract and pump blood back into the circulation.

If peripheral arterial flow is poor, accuracy of the pulse oximeter may be affected. Pulse oximeters, particularly older ones, may not distinguish between finger movement and poor pulsatile blood flow, giving inaccurately low saturation readings. Modern oximeters will also provide some indication of pulse amplitude (plethysmography) that allows the nurse to determine if flow is adequate to provide correct data to the oximeter.

Oxygen delivery to the tissues

Of the three factors that determine oxygen delivery to the tissues--oxygen saturation, haemoglobin concentration and cardiac output--cardiac output is the most vital.

At rest, the healthy body uses 250-300ml/min of oxygen. This can increase to 3000-4000ml/min during exercise. Conditions that increase the metabolic rate increase tissue oxygen demand. Examples are pregnancy, growth, infection and fever, stress or shock

Conclusion

The delivery of adequate oxygen to the tissues is vital to cell function. The processes involved are complex and cannot be assessed solely by pulse oximetry. In addition to oxygen saturation, assessment of oxygen delivery to tissues requires us to evaluate Hb levels and cardiac output for individual patients. It is important to understand the underlying physiology of oxygen transport and indicators of poor oxygen delivery to tissues to make best use of pulse oximetry in the clinical setting. While pulse oximetry is a useful indicator of saturation of Hb, it cannot be relied on as the sole source of information to determine how much, and how well, oxygen is being delivered to body cells.

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