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How Do I Know that My Patient is Alive? Mini Series

Session 1: Pulse Oximetry

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PULSE OXIMETRY & HYPOXAEMIA

Introduction

In 1990, Clarke and Hall reported a survey of anaesthetic mortality in U.K. small animal practice. This revealed an overall mortality of approximately 0.24% for dogs and 0.29% for cats. Further analysis of their figures sub-divided mortality rates into 0.12% and 0.18% for healthy dogs and cats, respectively, and 3.13% and 3.33% for 'sick' dogs and cats, respectively. More recently, Dr. David Brodbelt of the RVC has completed a much more detailed prospective Confidential Enquiry into Peri-operative Small Animal Fatalities (CEPSAF), reporting overall mortality figures of 0.17% and 0.24% for dogs and cats respectively. Healthy dogs and cats had mortality rates of 0.05% and 0.11%, respectively, while 'sick' animals had figures of 1.33% and 1.4% for dogs and cats, respectively. Thus, these figures show that small animal anaesthetic mortality has decreased in the last 20 years since the Clarke and Hall study, but this may be put into perspective when one considers that overall anaesthetic mortality in humans (including sick patients) is of the order of 0.001%.

For the purposes of CEPSAF, an anaesthetic-related death was defined as "perioperative death within 48 hours of termination of the procedure, except where death was due solely to inoperable surgical or pre-existing medical conditions". Perhaps one of the most alarming issues to arise from the study was the timing of anaesthetic-related death: almost 50% of dogs and >60% of cats that died, did so in the recovery period, with around 50% of them succumbing within the first 3 hours after termination of the anaesthetic, and many animals were unattended at this time. This suggests that greater attention should be made to continued observation, monitoring and support of animals in the post-anaesthetic period.

Although a number of factors influencing anaesthetic mortality are outwith the anaesthetist's control (e.g. body weight, age), others may be subject to manipulation and may allow a reduction in anaesthetic-related deaths. CEPSAF identified a number of areas where improvements may easily be made:

a) the high death rate in both dogs and cats during the anaesthetic recovery period suggests that improved observation and support may be required throughout this time, with greater attention paid to continued monitoring of the vital body systems, and supplementation of inspired oxygen in higher risk animals,

b) the association between endotracheal intubation and mortality in cats implies that greater care must be taken when performing this procedure,

c) fluid therapy should be carried out cautiously in cats, with both the volume and rate of administration carefully controlled

d) monitoring of pulse quality and use of pulse oximetry should be advocated

Interestingly, aside from the use of pulse oximetry, of the 117 centres included in the CEPSAF study:
-95 could monitor ECG - *but only 20 routinely used it*
-56 had non-invasive blood pressure (NIBP) monitoring - *but only 23 routinely used it*
-26 had capnography - *but only 24 routinely used it*

This is of concern, as analysis of serious peri-operative incidents in human anaesthesia has demonstrated that:
-pulse oximetry alone would have detected 40-82%
-pulse oximetry + capnography would have detected 88-93%
-pulse oximetry + capnography + NIBP would have detected ~93%

Consequently, the take-home message should be that, if anaesthetic monitoring equipment is available, it should be utilised in all anaesthetised patients, but particularly.
Dave Brodbelt's PhD thesis can be accessed and downloaded at:
http://www.rvc.ac.uk/Staff/Documents/dbrodbelt_thesis.pdf
so in the high risk groups.

Oxygen and its measurement

Oxygen is an essential requirement for cell function, and must be delivered in adequate quantities to all tissues throughout the body. Oxygen is carried in 2 forms in the blood:

1. dissolved in the plasma (~2%)
2. bound to haemoglobin (~98%)

There are 3 main stages in movement of oxygen from inspired gas into the blood: firstly, the inspired gas enters the alveoli; secondly, oxygen moves down its concentration gradient from an area of high partial pressure (inspired gas) to an area of lower partial pressure (pulmonary capillary blood) by crossing the alveolar-capillary membrane. The oxygen that crosses over from the alveoli then dissolves in the pulmonary capillary plasma. Oxygen then diffuses down a further concentration gradient from the plasma into the red blood cell and binds to the haemoglobin (Hb). This latter concentration gradient is maintained, as the Hb avidly binds oxygen, with each molecule of oxygen that binds inducing a conformational change in the Hb molecule that encourages uptake of further oxygen molecules. Eventually, a point is reached where there is equilibrium in oxygen movement between the Hb in the erythrocyte and the plasma, and between the plasma and the alveoli, and no further oxygen is taken up. This all happens very quickly - within a fraction of a second.

Hypoxaemia is defined as low oxygen in the blood - usually a haemoglobin saturation less than 90% or a partial pressure of oxygen in arterial blood (PaO_2) of less than 60 mmHg (8 kPa).and must be distinguished from **hypoxia**. Hypoxia is defined as decreased oxygen at tissue level and may be a result of hypoxaemia. However, hypoxia is not always associated with hypoxemia. Cellular hypoxia results in a switch to anaerobic metabolism leading to intracellular acidosis, lactic acid production and cell damage.

The amounts of oxygen dissolved in plasma and bound to haemoglobin (Hb) are related through the oxyhaemoglobin dissociation curve (Fig. 1). The curve is 'sigmoid' shaped because of the characteristics of haemoglobin.

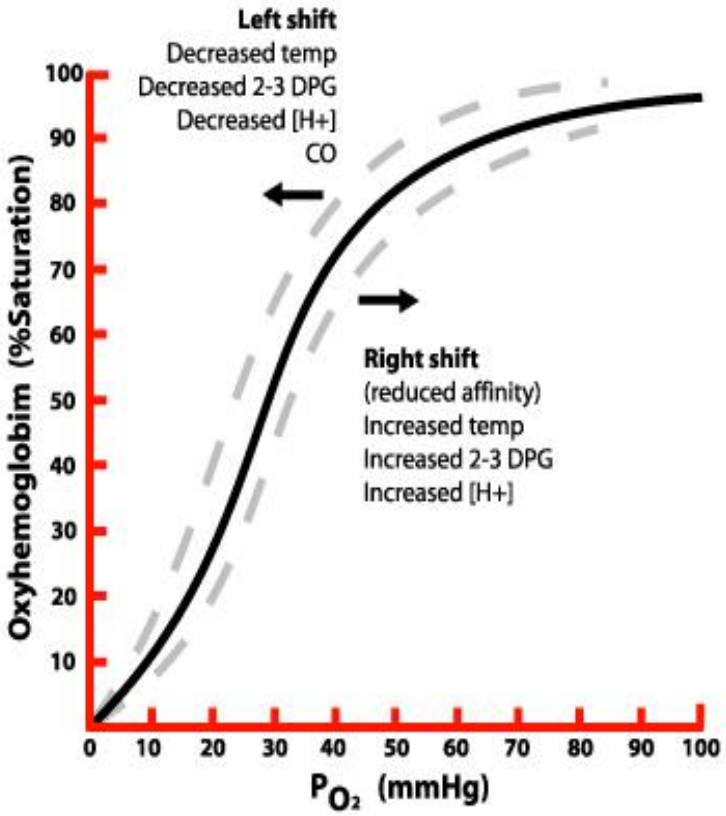

Fig 1 - Oxyhaemoglobin dissociation curve
(Image reproduced courtesy of Anaesthesia UK

PO_2 represents the partial pressure of free oxygen dissolved in the plasma i.e. freely dissolved oxygen in the plasma, and it can be seen from Fig. 1 that oxyhaemoglobin saturation decreases sharply (the so-called 'shoulder' of the curve) once PO_2 drops below about 60mmHg. Fig. 1 also highlights that, as the PO_2 increases, the oxyhaemoglobin saturation (SO_2) also increases, although a point is reached at a PO_2 of around 90mmHg where, despite further increases in PO_2, there is very little increase in SO_2, i.e. the curve reaches a plateau. A PaO_2 (the 'a' subscript here indicates we are talking about arterial blood) of 90-100mmHg is the value normally seen in patients with healthy lungs breathing room air, and this equates to a SaO_2 of approximately 97-98%. The PaO_2 can be measured directly by taking an arterial blood sample and passing it through a blood gas analyser, while the SaO_2 is usually measured with a pulse oximeter and given the abbreviation SpO_2 (Sp = **S**aturation measured by **p**ulse oximetry).

What is the value of one over the other? Well, obtaining an arterial blood sample is invasive (and sometimes tricky), and the equipment required to run the sample is expensive. In addition, inappropriate sample handling may introduce errors into the results. Pulse oximetry, on the other hand, is non-invasive, and the equipment required is now relatively cheap. Why then do we still bother with arterial blood gases if pulse oximetry is so useful? The answer lies mainly in the shape of the oxyhaemoglobin dissociation curve. The flat upper part of the curve means that there is very little change in SpO_2 for quite dramatic changes in PaO_2. Patients breathing oxygen enriched gas - for example, during anaesthesia - may have PaO_2 values of upwards of 500 mmHg, and, at this point on the curve, the SpO_2 will be approximately 100%. If a problem were to occur (e.g. a pulmonary embolus) that impaired their oxygenation, a drop in their PaO_2 from a value of 500mmHg down to a value of say 100mmHg would have very little effect on the pulse oximeter reading: it may drop from an initial value of 100% down to around 98% - something that would be unlikely to provoke concern, and therefore recognition, in the person monitoring the patient. Thus, arterial blood gas analysis gives a more accurate picture of adequacy of oxygenation over the range of PaO_2 values observed in clinical practice. The pulse oximeter, however, gives us adequate **clinical** information, because, even although oxygenation is impaired in the animal described above (i.e. the PaO_2 is lower than it should be), the patient is not in any real danger until SpO_2 drops below about 93% (the shoulder of the oxyhaemoglobin dissociation curve). The comparison between measuring PaO_2 and SpO_2 is often described as a person approaching a cliff edge: the PaO_2 gives you a warning that you are approaching the edge of the cliff when you are still a mile off, and will continue warning you as you get closer; the SpO_2, on the other hand, only tells you when you are right at the edge of the cliff and about to fall off. For anaesthetists, the problem comes when the anaesthetic ends, the oxygen enriched mixture is turned off and the animal subsequently demonstrates hypoxaemia. At this point, monitoring often ceases and may be part of the reason why anaesthetic related deaths commonly occur in the recovery period.

How does a pulse oximeter work?

Without going into too much detail of the physics behind pulse oximetry, the simplified answer is based on the fact that oxyhaemoglobin and deoxyhaemoglobin absorb different wavelengths of light.

The pulse oximeter consists of, on one side, a light emitting diode, and, on the other side, a photodetector. The two wavelengths of light used in the diode are 660nm and (usually) 940nm (although 910nm is illustrated in Fig. 2), and the diode turns these 2 wavelengths on and off in sequence approximately a thousand times per second. Patient tissue (commonly, tongue, toe or ear in animals; finger or ear lobe in humans) is placed between the light emitting diode and the photodetector.

Some of the light from the diode will be absorbed by the tissue itself, some will be absorbed by venous blood, and some will be absorbed by non-pulsatile arterial blood (i.e. the blood that just sits in the artery between pulses). The pulse oximeter is able to 'ignore' these static forms of absorption of light, and focus on the change in absorption during pulsatile flow. By analysing the relative proportions of each of the two wavelengths of light that pass through the tissue (Beer-Lambert Law), the oximeter is able to display the percentage oxyhaemoglobin saturation value (SpO_2), and also the pulse rate.

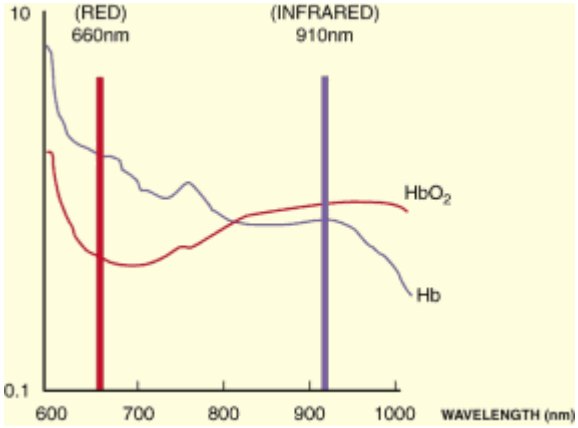

Fig. 2. Absorption spectra of oxyhaemoglobin (HbO_2) and deoxyhaemoglobin (Hb). Image reproduced courtesy of AnaesthesiaUK.

Normal SpO_2 should be above 95% in patients with adequate oxygenation capacity (and will usually be 98-100% in patients receiving supplemental oxygen). Decreases below approximately 93% should be investigated urgently, since - as discussed above - this is the shoulder of the oxyhaemoglobin dissociation curve, and even small further decreases in saturation are associated with marked decreases in oxygenation. The commonest cause of decreased SpO_2 is usually that the pulse oximeter probe has been in the same place too long and is compressing the tissue bed, and simply removing and re-positioning the probe will generally sort the problem (while simultaneously observing the patient to check they're not cyanotic!). If saturation remains low, the next stage would be check for equipment faults (e.g. failure in oxygen supply; nitrous oxide turned on instead of oxygenation etc.); if no fault can be found here, arterial blood gas analysis should (ideally) be performed to confirm whether oxygen levels are genuinely low.

What is my pulse oximeter not telling me?

The physiological equation describing the <u>oxygen content of the arterial blood</u> (CaO_2) is:

CaO_2 = {[Hb] x 1.34 x SpO_2} + {0.003 x PaO_2}

Where:

1.34 = oxygen carrying capacity of Hb (also known as Huffners constant)

SpO_2 = % oxygen saturation measured by pulse oximeter as a decimal

PaO_2 = partial pressure of oxygen in blood measured by arterial blood gas analysis

In the case of anaemic patients, the SpO_2 will be completely normal - although there will be less Hb in the blood - that which is present will still be fully saturated with oxygen, so the pulse oximeter will give 'normal' readings. However, the oxygen **content** of the blood (CaO_2) will be decreased due to the reduced Hb concentrations (anaemia). Thus, it is important to recognise that the presence of a normal SpO_2 on the pulse oximeter does not tell you that there is sufficient oxygen in the blood, it only tells you whether the Hb that is present is saturated or not.

Tissue oxygen delivery is defined by:

$$\mathbf{DO_2 = Q \times CaO_2}$$

where DO_2 = oxygen delivery

Q = cardiac output

CaO_2 = oxygen content of arterial blood

Thus, again, even if we have normal oxygen content in the blood, this does not necessarily imply that we have adequate oxygen delivery to the tissues, because cardiac output could be low. This is not a failing of the pulse oximeter, it merely emphasises the limitations that the equipment has.

So, what does a pulse oximeter not tell us? It doesn't tell us whether there is adequate oxygen in the arterial blood, and it doesn't tell us whether adequate oxygen is being delivered to the tissues.

Limitations of pulse oximetry

There are several situations where pulse oximeters may fail to give a reading, or may give inaccurate ones:

1. Vibration / patient movement - It is much more difficult (though still possible) to obtain accurate pulse oximeter readings on conscious patients than those that are unconscious, simply due to movement artefact, as this interferes with the machine's ability to detect pulsatile blood flow in the tissues. This is especially true with shivering patients in the recovery room
2. Vasoconstriction - Marked peripheral vasoconstriction as may be seen following administration of alpha-2 agonists, such as medetomidine, may prevent some pulse oximeters from obtaining readings. Better quality devices can generally still obtain a signal, although this cannot be guaranteed. Vasoconstriction will also occur in cold patients following general anaesthesia.

3. Visible light - Light sources, particularly if pulsatile (e.g. fluorescent strip lights), can interfere with the ability of the pulse oximeter to get a signal.

This is less common with newer machines, as shielding of the probe has improved over the years, but may still occur if the probe is partially placed.

4. Calibration- Achieved by using human volunteers and progressively desaturating them by making them breathe increasingly hypoxic gas mixtures. Because it is considered unethical to desaturate these volunteers to SpO_2 of < 80%, readings below this value on the pulse oximeter are extrapolated, i.e. accuracy may be imprecise. However, potentially it doesn't matter too much if the patient's SpO_2 is 49% or 67%, because both values imply a serious problem.

5. Pigmentation - It may be difficult to obtain a good signal if the pulse oximeter probe is placed on a heavily pigmented area. This can be problematic in breeds such as Shar Peis and Chows, where the tongue and oral mucosa are often black. A signal can sometimes be obtained in these animals by attaching the probe to the vulva or prepuce, as this is often less heavily pigmented.

6. Pulsatile veins - Although not a common problem, the pulse oximeter can be confused if there is pulsatile flow within veins, and may give falsely low saturation readings. This can sometimes occur with severe tricuspid regurgitation or 3° (complete) atrio-ventricular blockade.

7. Dyshaemoglobinaemias - There are 2 main abnormal forms of Hb that can lead to inaccurate pulse oximeter readings. **Methaemoglobinaemia** may occur following local anaesthetic toxicity (particularly prilocaine), but is also seen in cats with paracetamol intoxication. This form of dyshaemoglobin tends to make the pulse oximeter read towards 85%, and no conclusions regarding oxygenation can be made using pulse oximetry in this setting. The second significant dyshaemoglobin is **carboxyhaemoglobin**, which may be seen in patients following carbon monoxide intoxication (e.g. animals trapped in house fires). Carboxyhaemoglobin tends to push pulse oximeter values towards 100%, as it has a similar light absorption spectrum to oxyhaemoglobin. Again, pulse oximetry is an unsuitable means of assessing patient oxygenation if carboxyhaemoglobin is present.

Causes of hypoxemia

There are 5 main causes:

1. Reduced inspired oxygen fraction (FIO_2) – can be a result of human error – empty cylinders, incorrectly fitted gas supplies, anaesthetic machine and breathing system faults, or simply breathing system disconnection.

2. Hypoventilation- leads to hypercapnia (high arterial partial pressure of CO_2 ($PaCO_2$)) and this reduces the concentration of oxygen within the alveoli. Hypoventilation will not result in hypoxemia if the inspired gas mixture is enriched (e.g. 100% during inhalational anaesthesia) but may contribute to hypoxemia if hypoventilation is not occurring in isolation (e.g. in combination with ventilation perfusion inequality). Hypoventilation can be exacerbated by bodyweight and positional influences – a large fat Labrador in dorsal recumbency will hypoventilate more than a fit spaniel in lateral recumbency. The degree of hypoventilation will also be influenced by anaesthetic drugs administered. Hypoventilation can be identified by measuring $PaCO_2$ using a blood gas analyser or end tidal pressures of CO_2 ($ETCO_2$ or P_ECO_2) using a capnometer or a capnograph – see later notes. Hypoventilation should be addressed by adjusting anaesthetic depth first if this is feasible. Intermittent positive pressure ventilation can be used to treat hypoventilation, but carries certain side-effects and risks.

3. Ventilation perfusion inequality – extremes of mismatching can result in shunt. A ratio of ventilation and perfusion within the lung describes how well (or how poorly) an area of lung is ventilated in relation to its perfusion with pulmonary blood. The ideal V/Q ratio is equal to 1.0.

   a. High V/Q ratio - An area of lung that is well ventilated but poorly perfused and will result in alveolar dead space. However, the small amount of pulmonary blood leaving this part of the lung will be well oxygenated.

   b. Low V/Q ratio - An area of lung that is poorly ventilated (atelectic) but well perfused will result in pulmonary shunt (blood that has a low PaO_2 and a high $PaCO_2$ (i.e. has not taken part in gas exchange) being mixed with blood with a high PaO_2 and a low $PaCO_2$ (i.e. blood that has taken part in gas exchange)).

   In the conscious animal, ventilation and perfusion ratios are optimised by an elegant physiological adaption known as hypoxic pulmonary vasoconstriction (HPV). Blood passing through poorly ventilated areas of lung is diverted away by vasoconstriction to better ventilated areas. HPV is obtunded to varying degrees during anaesthesia, being much more affected by volatile agents.
   Absorption atelectasis occurs in lungs ventilated with enriched oxygen mixtures. As oxygen rapidly diffuses across the alveolar endothelium, the alveolus subsequently empties, collapses and contributes to low V/Q ratios.

   Any disease state where either the alveolus and/or its capillary can become completely or partially obstructed, or where hypoventilation/hypoperfusion can occur by other means can result in V/Q mismatching.

4. Diffusion abnormalities and impairment – thickening of the blood gas barrier by disease (e.g. pulmonary oedema).

5. Arterio-venous shunting – e.g. a congenital right to left intra-cardiac shunt.

Treatment of hypoxaemia

Hypoxemia caused by decreased inspired oxygen concentration, hypercapnia, or diffusion defects may be corrected by increasing the inspired oxygen concentration.

If hypoventilation is occurring, always attempt to lighten the depth of general anaesthesia if this is feasible, to facilitate less depression of respiratory drive.

Intermittent positive pressure ventilation (IPPV) can be instituted either by manually squeezing the bag, or using a mechanical ventilator if available. Caution should be exercised when resorting to IPPV and attention should be paid to inspiratory pressures, blood pressure and $PaCO_2$.

Alveolar atelectasis during recumbency is difficult to treat once it has occurred

The application of ventilatory strategies that utilise PEEP may help to maintain alveolar inflation but may increase the risk of side effects (e.g. reduced cardiac output). PEEP maintains a set amount of positive intrathoracic pressure (determined by the clinician) during the expiratory pause. This can be achieved using commercially available PEEP valves, or mechanical ventilators which have this capability.

Conclusions

Pulse oximetry is a useful monitoring tool in both awake and anaesthetised patients. However, it does have limitations, and these must be understood to appreciate the value of the information it is providing. A low SpO2 reading will alert the anaesthetist to potential hypoxaemia of which there are many causes, and may facilitate rapid treatment, reducing morbidity and mortality in anaesthetised and critical patients.