Anaesthetic Monitors in Small Animal Anaesthesia: Beyond the Numbers

Dr Roz Machon BVSc MS MANZCVSc Dipl. ACVA

New Zealand

Vet Education Pty Ltd
ANAESTHETIC MONITORING DEVICES
Looking Beyond the Numbers
Roz Machon BVSc (Hons), MVSc, MS, MANZCVS, DACVA

Introduction

The over-riding role of an anaesthetist is to watch over the patient: to assess anaesthetic depth, monitor vital organ function and obtain information that can be used to maximise patient safety and minimise risk. The term “monitor” comes from the Latin “to warn”, and means to check systematically or keep watch over. Good patient monitoring requires an understanding of physiology, pharmacology – and in particular – how anaesthetic agents adversely affect vital organ function. It also requires some knowledge of the technology under-pinning various monitoring devices, and an understanding of what these devices can and can’t tell you about patient wellbeing. Anaesthetic depth and vital organ function are intimately linked in the sense that anaesthesia affects vital organ function in a dose-dependent manner, while vital organ function is itself a measure of anaesthetic depth: both factors must be evaluated when monitoring anaesthetized patients.

While there is no question that simple monitoring techniques provide essential “baseline” information about anaesthetic depth and vital organ function, “hands on” methods are limited in the sense that they lack subtlety: parameters may indicate cardiopulmonary function is inadequate but do little to quantify the degree of impairment. In contrast, monitoring devices such as non-invasive blood pressure (NIBP) monitors, pulse oximeters and capnometers, provide quantitative data about cardiopulmonary function that is difficult, if not impossible, to gain using physical assessment alone. These monitors are non-invasive, can be used in a wide range of patients, and often – although not always – provide continuous information about cardiopulmonary function. Knowledge of the “numbers” (i.e. normal values) is essential for appropriate interpretation of this information. However, when this quantitative data is supplemented with an understanding of the relevant physiology, pharmacology and device technology (as outlined above), monitors can provide information about anaesthetic depth and physiological status that extends beyond “the numbers”, providing a broader perspective of patient wellbeing and the potential to reduce morbidity and mortality.

Can the use of anaesthetic monitoring devices improve patient outcome?

Support for the usefulness of monitoring devices with respect to outcome comes from anaesthetic morbidity and mortality studies in both the human and veterinary literature. Anaesthetic complications may range in nature from minor through to catastrophic, and may be classed as preventable or unpreventable. Studies investigating critical incidents in anaesthetized people have conclusively demonstrated the use of minimum monitoring standards (i.e. a set of published guidelines or recommendations that document the baseline level of acceptable care) to significantly reduce patient morbidity and mortality. Routine monitoring of arterial blood pressure (ABP), arterial saturation (SaO\textsubscript{2} or SpO\textsubscript{2} when measured via pulse oximetry) and end-tidal carbon dioxide (ETCO\textsubscript{2}) values has long been considered a minimum standard in human anaesthesia. An Australasian study of 2000 critical incidents in anaesthetized people (Webb, 1993) showed a monitor of some sort was the first indicator of a problem in 52% of reported incidents and complications. The authors of this study used a model
to predict the theoretical “usefulness” of each monitor in a typical anaesthetic scenario. Based on these predictions, pulse oximetry would have detected 82% of all problems and would have warned of nearly 60% of problems prior to the potential for organ damage. The addition of capnometry would have raised these figures to 88% and 65% respectively, while the addition of ABP monitoring would have resulted in detection of 93% of complications – providing warning of 65% of these before the potential for organ damage. Based on this analysis and after consideration of costs, the authors created a “priority sequence for monitor acquisition” list. Ranking of the monitors (in descending order) was as follows: - (1) stethoscope, (2) NIBP monitor, (3) pulse oximeter, and (4) capnometer, suggesting ABP monitoring was particularly useful in detecting complications and reducing risk in anaesthetized people.

Few studies report the incidence of common, “every day” anaesthetic complications in cats and dogs. Dyson et al’s (1998) anaesthetic morbidity and mortality study of > 16,800 small animals in 66 private practices in Canada, reported complications – including respiratory depression, apnoea and arrhythmias – in 2.1% of dogs and 1.3% of cats. Patients in this study were monitored at least intermittently in about 85% of cases, although monitoring was limited to simple techniques (i.e. physical assessment, stethoscope and/or apnoea monitor) with ABP measurement performed in < 0.2% of cases. In contrast, a study examining 2556 dogs and 683 cats anaesthetized by the anaesthesia service of a large University Veterinary Teaching Hospital (Gaynor et al, 1999), reported various complications – including hypotension, hypoxaemia and hypoventilation – in 12% of dogs and 10.5% of cats, when patients were monitored with more advanced devices. While it could be argued the higher incidence of complications in the teaching hospital-, versus the private practice-based patients was the result of a sicker patient population (studies suggest ASA III-V patients represent only 5-10% of the caseload in private practice versus 20-40% of that in referral practice), a higher incidence of complications was also noted in private practice patients that were monitored more intensively, highlighting the link between vigilant monitoring and problem recognition. A recent retrospective study (Redondo et al, 2007) of cardiopulmonary complications in 1281 dogs undergoing a variety of diagnostic and surgical procedures supports this view. Hypoxia, bradycardia, hypotension and hypoventilation were identified in approximately 16%, 36%, 38% and 63% of dogs, respectively, when monitoring devices were employed.

These findings support the hypothesis that monitoring devices aid in the detection of anaesthetic complications in veterinary patients, just as they do in the human arena. But does this translate to an improvement in outcome? The importance of being able to rapidly detect, interpret, and take appropriate steps to correct, an abnormality in an anaesthetized patient was highlighted in an anaesthetic survey of small animal practitioners performed in the late 1980’s (Dodman et al, 1992). In this survey, 46% of the 39 respondents reported “zero” anaesthetic complication rates over the two-year retrospective investigation period, although four of these practitioners went on to report seven anaesthetic-related deaths during this same period. In fact, cardiopulmonary arrest (CPA) was the most commonly reported complication in this study, outranking all other cardiopulmonary problems and suggesting a failure to recognise and respond to earlier indicators of excessive anaesthetic depth or cardiopulmonary compromise. In comparison, Dyson et al were able to demonstrate that monitoring – by a trained veterinary technician/nurse – significantly reduced serious morbidity and anaesthetic-related mortality, presumably because minor problems were detected and corrected before they escalated. As
outlined above, few of the 66 practices participating in Dyson et al’s survey employed advanced monitoring devices at the time of the study, preventing an assessment of whether or not the use of monitors could further reduce risk.

CEPSAF (the Confidential Enquiry into Perioperative Small Animal Fatalities) - a recent, large scale, multi-centre, cohort investigation of the risks of anaesthetic and sedation-related mortality in > 98,000 dogs and 79,000 cats - is the first veterinary trial that goes some way towards answering the question of whether the use of monitoring devices can reduce the risks of anaesthetic-related death in small animals. The authors defined anaesthetic and sedation-related death as “perioperative death within 48 hr of termination of the procedure, except where death was due solely to inoperable surgical or pre-existing medical conditions” i.e. all cases in which anaesthesia and sedation could not reasonably be excluded from contributing to death, including those deaths that occurred in the recovery period. CEPSAF showed a significant (p < 0.001), 3-4-fold reduction in the odds of death when pulse rate and pulse oximetry were routinely monitored in anaesthetized cats. Assessment of the success (or failure) of other monitoring devices to also reduce mortality was not possible, because strategies such as ABP monitoring were performed in < 10% of patients. Current evidence therefore supports the view - but does not conclusively demonstrate - that intelligent employment of monitoring devices such as capnometers, pulse oximeters and NIBP monitors, aid in problem recognition and reduce the risk of anaesthetic-related death in cats and dogs.

Looking Beyond the Numbers

1. Anaesthetic monitoring devices: Capnography

Ventilation: reviewing the physiology

Minute respiratory volume (V_m) is the amount of air drawn into the lung on a per minute basis. It can be calculated as follows (Equation 1) and is a rough estimate of the amount of air available for gas exchange. The actual volume of air participating in gas exchange - alveolar ventilation (V_A) - is less than V_m because 20-35% of tidal volume (V_T) is “lost” to physiological dead space (V_D) as shown in Equation 2.

- **Equation 1:** Minute respiratory volume (V_m) = respiratory rate (RR) x tidal volume (V_T)
- **Equation 2:** V_A = (V_T - V_D) x RR

Ventilation and gas exchange are therefore intimately linked: normal ventilation is necessary for normal gas exchange, while abnormal ventilation results in abnormal gas exchange. Compensatory responses allow V_m to be maintained in the face of small changes in RR or V_T – a change in one parameter usually being offset by a change in the opposite direction of the other. Compensatory mechanisms have their limits however, and hypoventilation occurs when V_A falls below normal, while the opposite is true for hyperventilation. Any change in V_A directly affects the partial pressure of carbon dioxide in arterial blood (PaCO_2). V_A and PaCO_2 are inversely related: if V_A falls by 50%, PaCO_2 will double; while a 100% increase in V_A (e.g. vigorous intermittent positive pressure ventilation) will halve PaCO_2. PaCO_2 therefore acts as the hallmark of the adequacy of ventilation. If patients are breathing room air (21% oxygen), hypoventilation may also result in hypoxaemia; however, hypoventilation will always result in hypercapnia.
The effects of anaesthesia on normal ventilation

Under normal circumstances, ventilation is itself tightly regulated by PaCO$_2$: a very small increase in PaCO$_2$ stimulates the respiratory control centre to increase V$_m$ while an equally small reduction decreases the drive to breathe, returning PaCO$_2$ to normal. This relationship is represented graphically by the PaCO$_2$/ventilatory response curve. PaCO$_2$ levels are maintained within a very small range via this mechanism, only varying by about 3 mmHg during the course of a normal day’s activities. Anaesthetic agents depress the ventilatory response to PaCO$_2$ resulting in reduced respiratory drive. Most agents produce dose-dependent depression, flattening the PCO$_2$/ventilatory response curve and pushing this to the right. Apnoea may be seen in patients with PaCO$_2$ levels insufficient to trigger breathing (i.e. values below the apnoeic threshold); while respiratory arrest will occur once the depth of anaesthesia is such that the normal response to PaCO$_2$ is abolished (usually Stage III, Plane 4 or Stage IV).

All general anaesthetic agents produce at least some degree of muscle relaxation and therefore impair diaphragmatic-, intercostal- and chest wall function. As anaesthetic depth increases, muscle relaxation becomes so profound that patients are unable to coordinate normal inspiratory activity, resulting in characteristic “tracheal tug” ventilation (a sign of Stage III, Plane 4 anaesthesia) and ultimately, respiratory arrest. Anaesthetic-induced muscle relaxation increases the work of breathing and usually reduces V$_t$ – a problem compounded by surgical positioning or the placement of instruments etc on the patient’s chest. The effects of relatively small, anaesthetic-induced reductions in V$_t$ may produce greater effects on V$_A$ than initially expected because a proportion of V$_t$ must always be used to “fill” physiological dead space (V$_D$). At best, V$_D$ remains constant following the induction of anaesthesia but often increases considerably (e.g. in the face of progressive atelectasis). Either way, V$_D$ now consumes a relatively greater proportion of V$_t$ than normal – studies in anaesthetized dogs suggest up to 50% – significantly reducing the volume of gas available to participate in gas exchange (i.e. V$_A$). This effect is exacerbated by excessively large facemasks or overly long ET tubes, which add additional dead-space, further reducing V$_A$.

Monitoring the adequacy of ventilation: reviewing the technology

PaCO$_2$ is traditionally measured via arterial blood gas analysis with normal ranges reported as 28-49 mmHg (3.7-6.4 kPa) in dogs and 35-49 mmHg (4.6-6.4 kPa) in cats (but 35-45 mmHg is a reasonable estimate for both species). Although highly accurate, arterial blood gas analysis is invasive, technically demanding, relatively expensive, and cannot easily provide continuous information. However, PaCO$_2$ can also be estimated simply and non-invasively by measuring the partial pressure of CO$_2$ in alveolar gas (P$_A$CO$_2$) using a capnometer. This is possible because in normal animals, P$_A$CO$_2$ closely approximates PaCO$_2$ – the hallmark of the adequacy of ventilation – due to the ability of CO$_2$ to readily diffuse across the alveolar wall, allowing blood and alveolar tensions to rapidly approach equilibrium. Most veterinary capnometers use infrared absorption to measure CO$_2$ levels. With this technique, a specific wavelength of infrared light (4.28 μm) is shone through a gas sample containing an unknown concentration of CO$_2$ and the subsequent absorption measured. The actual CO$_2$ concentration is determined by the Beer-Lambert law (using a technique similar to that used in pulse oximetry)
and is reported in various units e.g. millimetres of mercury (mmHg), kilopascals (kPa) or percent (%).

Capnometers are basically simple gas analysers and usually report a maximal CO\(_2\) value (presumed to be endtidal CO\(_2\) (ET\(\text{CO}_2\)), a measure of \(P\text{\textsubscript{a}}\text{CO}_2\)) and a minimal value (the concentration of CO\(_2\) in inspired gases). More sophisticated monitors also display a graphic portrayal of the concentrations of CO\(_2\) in respired gases over time. This results in a characteristic waveform – the capnogram – that reflects the various phases of the respiratory cycle. Capnometers that sample airway gases continuously and display a capnogram in addition to digital readouts of ET- and inspired CO\(_2\) levels are best for clinical practice and have become increasingly affordable.

There are two main types of infrared capnometer: - (1) mainstream, in which a small sensor is placed within the endotracheal tube, and (2) side-stream, in which a sample of gas is continuously drawn from a side-port connected to the patient’s endotracheal tube and transported to the analyser via a narrow-bore tube. Side-stream capnometers are more practical in veterinary practice because they are generally cheaper and less cumbersome than mainstream devices; however, improvements in mainstream design offer some advantages. Side-stream monitors continuously sample gas from a port attached to the machine-end of the endotracheal tube at a rate of 150-300 ml/min, providing continuous information but also resulting in two potential problems. Firstly, gas is continuously lost from the patient breathing circuit – fresh gas flow rates may have to be increased to compensate for this, particularly when using non-rebreathing circuits. Secondly, in patients maintained with inhalational techniques, the sampled gas will contain traces of inhalant that must be scavenged or returned to the breathing circuit. Because the sampled gas also contains CO\(_2\), the “return tube” must be connected to the scavenging system when using a non-rebreathing circuit, or the expiratory side of the circuit (i.e. up-stream from the soda lime canister) when using a rebreathing circuit.

**Problems of ventilation: Hypoventilation and hypercapnia**

Anaesthetic-induced hypoventilation impedes normal gas exchange by reducing the overall volume of gas delivered to the alveoli each minute (i.e. \(V_A\)). This may result in hypoxia if patients are breathing room air, or impede the delivery of inhalant to the alveoli resulting in unstable anaesthetic depth, but will always induce hypercapnia. Severe respiratory depression was the most commonly reported anaesthetic complication in two surveys of anaesthetic morbidity and mortality in small animal practice (Clarke and Hall, 1990; Dyson *et al.*, 1998), and is considered the most common forerunner to CPA in anaesthetized patients. Simple hypoventilation is the most common cause of hypercapnia in anaesthetized patients; however, hypercapnia may also arise as a result of rebreathing (e.g. insufficient fresh gas flow rates, faulty one-way valves, exhausted soda lime or other machine/circuits faults) – a problem readily detected with capnometry – or rare metabolic disorders (e.g. malignant hyperthermia). The effects of hypercapnia are varied and include peripheral vasodilation, sympathetic nervous system stimulation and acidosis. Hypercapnia also increases myocardial sensitivity to circulating catecholamines (increasing the likelihood of arrhythmias) and exacerbates hyperkalaemia. CO\(_2\)-induced vasodilation leads to changes in cerebral blood flow, increased intracranial pressure, increased bleeding from incisions, and the appearance of characteristic brick-red mucous membranes. CO\(_2\) is a known CNS depressant and has been investigated as both an anaesthetic and euthanasia agent in a number of species. Respiratory acidosis causes widespread cellular and organ dysfunction and may be partly responsible for the central depressant effects of CO\(_2\). Mild hypercapnia is actually sympathomimetic, producing small increases in heart
rate, myocardial contractility and ABP; however, the more potent effects of acidosis and CNS depression soon overwhelm this potential benefit once PaCO₂ rises to > 65 mmHg or pH falls below 7.2.

**Capnometry: looking beyond the numbers**

Three factors determine ETCO₂: (1) cellular metabolic rate, (2) the rate of CO₂ exchange between the blood and alveoli, and (3) alveolar ventilation (VA). Because CO₂ diffuses across the alveolar wall so readily, we would normally expect little difference between ETCO₂ and PaCO₂ (i.e. PaCO₂ minus ETCO₂ should equal about zero). In reality, ETCO₂ is generally 3-8 mmHg less than PaCO₂ due to the presence of physiologic shunt (about 2-3% of cardiac output - rich in CO₂ bypasses the lungs under normal conditions) and because alveolar CO₂ concentrations are diluted by inspired gas (which is essentially CO₂-free). ETCO₂ therefore closely approximates PaCO₂, at least in patients with normal lung function and appropriate matching of alveolar ventilation and perfusion. Normal ETCO₂ values in anaesthetized dogs and cats range from about 30-45 mmHg; however, values up to 55 mmHg are considered acceptable in most patients. The inspired CO₂ value should always be zero: values greater than this suggest rebreathing. ETCO₂ values > 55 mmHg are indicative of clinically significant hypercapnia and warrant correction.

Capnography provides irrefutable evidence the trachea is intubated and warns of problems such as accidental extubation, disconnection from the circuit, faulty one-way valves and insufficient fresh gas flow rates. Monitoring of ETCO₂ also provides basic information about cellular metabolism and the adequacy of cardiovascular function: malignant hyperthermia results in marked increases in ETCO₂, while significant falls in cardiac output (e.g. CPA) are reflected by abrupt reductions in ETCO₂. The shape of the capnogram can also provide useful information with deviations suggesting a number of problems including circuit leaks or partial obstruction of the endotracheal tube. However, interpretation requires experience and must be considered in association with other indicators of anaesthetic depth and vital organ function as the waveform for simple, easily corrected technical faults can mimic life-threatening problems (e.g. disconnection from the circuit mimics CPA).

As with any technology, reported numbers are not always accurate. As outlined above, the PaCO₂-ETCO₂ gradient is very small in patients with reasonably normal lung function: ETCO₂ is therefore a good estimate of PaCO₂. However, factors that widen the PaCO₂-ETCO₂ gradient reduce the ability of ETCO₂ to accurately reflect PaCO₂. ETCO₂ is a measure of PA CO₂. Because only perfused alveoli can participate in gas exchange, non-perfused alveoli have a PCO₂ of zero, which effectively dilutes ETCO₂ and increases the PaCO₂-ETCO₂ gradient. This effect is exacerbated by any condition that increases alveolar dead space (e.g. V/Q mismatch, hypotension, pulmonary vasoconstriction and pulmonary thrombo-emboli). In contrast, conditions that specifically increase shunt produce minimal changes in the PaCO₂-ETCO₂ gradient. Capnometry has been shown to be an unreliable indicator of the adequacy of ventilation in dogs undergoing thoracotomy, and may be inaccurate in patients with problems that significantly increase Vₐ (as outlined above). Inaccuracies have also been reported in association with high respiratory rates or when using non-rebreathing systems with high fresh gas flow rates – scenarios common in small veterinary patients. A high ETCO₂ value is always indicative of hypercapnia (but may still
underestimate the degree); however, the converse is not always true depending on the size of the PaCO₂-ETCO₂ gradient in an individual patient.

2. Anaesthetic monitoring devices: Pulse oximetry

Oxygenation, and the relationship between PaO₂ and SaO₂: reviewing the physiology

The partial pressure of oxygen in arterial blood (PaO₂) is said to be the hallmark of the adequacy of gas exchange. As mentioned previously, CO₂ is a very diffusible gas. PaCO₂ is a sensitive indicator of changes in ventilation, but is relatively insensitive to changes in gas exchange because large changes are needed to impair the passage of such a readily diffusible substance. In contrast, oxygen (O₂) is about 20 times less diffusible than CO₂: reductions in gas exchange are rapidly reflected by a corresponding fall in PaO₂.

Under normal circumstances, about 98% of the O₂ diffusing across the alveoli binds to haemoglobin while the remaining 2 or 3% dissolves in the blood. Arterial saturation (SaO₂) is a measure of the amount of haemoglobin in the oxygenated or “saturated” form expressed as a percentage, while PaO₂ is a measure of the amount of dissolved O₂ expressed in millimetres of mercury (mmHg) or kilopascals (kPa). Normal SaO₂ and PaO₂ values in cats and dogs breathing room air are about 97% and 80-100 mmHg (10.7-13.3 kPa), respectively. The relationship between PaO₂ and SaO₂ is described by the oxyhaemoglobin dissociation curve and summarized by the following number sequence:

<table>
<thead>
<tr>
<th>PaO₂</th>
<th>40</th>
<th>50</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>SaO₂</td>
<td>70</td>
<td>80</td>
<td>90</td>
</tr>
</tbody>
</table>

PaO₂ has a variable but important effect on SaO₂ as shown by the sigmoid-shape of the oxyhaemoglobin dissociation curve. Normal patients operate on the plateau of the curve. SaO₂ cannot increase substantially in this portion of the curve – even when patients inspire 100% O₂ – because most of the haemoglobin leaving the lung is already fully saturated: SaO₂ can only increase by 2 or 3% although PaO₂ may increase five-fold. However, once the shoulder of the curve is reached (a situation that occurs commonly in anaesthetized patients), small decreases in PaO₂ have profound effects on arterial saturation and may result in life-threatening hypoxia.

Hypoxia and the effects of anaesthesia on normal oxygenation

Because ventilation and gas exchange are intimately linked, the adverse effects of anaesthesia on ventilatory function also impact gas exchange and therefore oxygenation. Hypoxia has been defined as “any state in which alveolar, blood, or tissue O₂ levels are insufficient to sustain normal organ function, resulting in real or potential damage to cells”. Hypoxia is therefore a physiologic state or a process occurring at the tissue level, where as hypoxaemia refers to an abnormally low “level” of O₂ in the blood (i.e. a PaO₂ of ≤ 60 mmHg) while desaturation refers to a fall in SaO₂ below a critical threshold (i.e. ≤ 90%). Normal PaO₂ is dependent on the inspired O₂ tension or fraction (FiO₂), Vₐ, and the matching of ventilation and perfusion (V/Q) in the lung. Reported causes of hypoxaemia in anaesthetized patients include hypoventilation, delivery of a hypoxic gas mixture, and
diffusion impairment; however, anaesthetic-induced V/Q mismatch with resultant shunt is the major impediment to gas exchange in the majority of cases.

Even under normal circumstances, alveolar ventilation and perfusion are not uniformly distributed throughout the lung. Anatomical factors, gravitational forces and the fact that the pulmonary arterial system operates under low pressure, result in preferential ventilation and perfusion of more ventral areas of the lung, producing a gradation of V/Q ratios. Appropriate matching of ventilation to perfusion in individual alveoli is optimised by hypoxic pulmonary vasoconstriction (HPV) – a homeostatic mechanism that diverts blood flow away from regions of low alveolar O\(_2\) tension (i.e. regions with poor ventilation). Anaesthesia and recumbency result in a much wider spread of V/Q ratios throughout the lung than those seen in conscious, upright patients, significantly increasing the shunt fraction (i.e. regions with poor ventilation but normal perfusion). While a range of factors including age, pulmonary disease and species determines the amount of shunt in any given patient, studies suggest that anaesthesia per se increases shunt from its normal value of 2-3% to about 10% of cardiac output. Anaesthetic-induced increases in V\(_D\) and reductions in functional residual capacity, progressive atelectasis, hypotension and impaired HPV, further compound the problem. In addition to the effects on gas exchange, anaesthesia can also contribute to tissue hypoxia via any other factor that impairs peripheral oxygen delivery (DO\(_2\)) e.g. anaemia, changes in haemoglobin O\(_2\) binding affinity, reduced cardiac output, hypotension, or peripheral vasoconstriction (see Equations 3 and 4).

**Monitoring the adequacy of oxygenation: reviewing the technology**

Oxygenation is traditionally assessed via arterial blood gas analysis (which measures PaO\(_2\)) or oximetry (which measures SaO\(_2\)). Although useful and highly accurate, both techniques are invasive, do not provide continuous information, may be associated with complications (e.g. infection), and require the purchase of expensive equipment. Traditional oximetry is based on the absorption patterns generated when different wavelengths of light are transmitted through a sample of arterial blood. In contrast, pulse oximetry provides a simple, non-invasive, and continuous means of estimating SaO\(_2\) by measuring haemoglobin saturation (known as SpO\(_2\) and expressed as a percentage) across a living tissue bed. Most models also display pulse rate and emit an audible beep for each pulse wave – the pitch varying with changes in SpO\(_2\).

Pulse oximetry is based on two physical principles: (1) oxyhaemoglobin and deoxyhaemoglobin absorb light differently and (2) arterial blood flow can be detected as a pulse wave. The pulse oximeter probe emits two different wavelengths of light (one red and one infra-red), which are shone through a tissue capillary bed and detected by a receiver. Oxyhaemoglobin absorbs infrared light well but reflects red light: this is why oxygenated blood looks red, while the opposite is true for deoxyhaemoglobin. By comparing the absorption pattern during pulsations with the underlying background absorption pattern of the tissue bed (which corresponds to venous blood, tissue and bone), the monitor is able to measure "pulsatile absorption" and calculate SpO\(_2\) (via the Beer-Lambert law). Most pulse oximeters also provide a visual display of the pulsatile signal. Known as a plethysmograph when the signal is presented as a continuous waveform – this reflects the quality of the pulse signal detected by the oximeter and is a good indicator of the likely accuracy of the reported
saturation (in-addition to acting as an indicator of tissue perfusion). A variety of different oximeters and probes are available. The human ear-lobe probes (as opposed to the finger probes) are the most useful and versatile for veterinary patients. Although a number of locations have been evaluated and shown to be reasonably accurate, the tongue is by far the most effective site for probe placement. Other useful sites include toes, the upper lip and the skin fold of the groin area. Specific veterinary probes are marketed (eg. a rectal probe); however, reports on their accuracy and performance are lacking.

In normal conscious patients breathing room air, \( \text{SpO}_2 \) is usually \( \geq 97\% \). A value of \( \geq 94\% \) is acceptable in anaesthetized patients because some degree of anaesthetic-induced cardiopulmonary depression is inevitable, and because this value still allows the patient to “operate” on the plateau of the oxyhaemoglobin dissociation curve. While marked desaturation is uncommon in normal, healthy cats and dogs receiving 100% \( O_2 \), hypoxia is common in compromised patients or those maintained with injectable agents without supplemental \( O_2 \). \( \text{SpO}_2 \) values of \( \leq 90\% \) in the presence of a strong, pulsatile signal are indicative of significant desaturation and may result from either respiratory or cardiovascular dysfunction, or equipment failure.

**Pulse oximetry: looking beyond the numbers**

Pulse oximetry is by no means infallible. Factors such as patient movement, excessive panting, high heart rates, dark pigmentation and poor peripheral perfusion (e.g. hypothermia or intense vasoconstriction) may result in grossly erroneous readings. However, \( \text{SpO}_2 \) values in the range of 70-100% are generally within 5% of actual \( \text{SaO}_2 \) values, with pulse oximetry tending to under- rather than over-estimate true saturation. From a pragmatic point of view, this translates to the delivery of supplemental \( O_2 \) to patients unnecessarily, rather than withholding \( O_2 \) from those truly in need. Pulse oximetry tends to over-estimate \( \text{SaO}_2 \) by an increasing margin of error as true \( \text{SaO}_2 \) falls below 70%; however, the measured \( \text{SpO}_2 \) value will still act as an indicator of the presence of hypoxaemia, even though it may not be an accurate reflection of the degree of desaturation at these levels. In addition, pulse oximetry is insensitive to the marked changes in oxygenation that may be seen in a patient receiving supplemental \( O_2 \), who is still operating on the “plateau” of the oxyhaemoglobin dissociation curve (i.e. \( \text{PaO}_2 \) values of 100-500 mmHg). At this point on the curve, marked changes in \( \text{PaO}_2 \) produce almost inconsequential changes in \( \text{SpO}_2 \) values - although the pulse oximeter should provide warning once these values become critical. Quite commonly, the tissue between the probe is compressed excessively, giving a reading that is accurate for the tissue bed but not a true indicator of the patient’s overall \( O_2 \) status. This problem is easily resolved by moving the probe to a new site, but as noted above, readings of \(< 90\% \) are always cause for concern and should never be ignored.

It is important to remember that \( \text{SpO}_2 \) is not a true measure of arterial \( O_2 \) content (\( \text{CaO}_2 \)) or oxygen delivery (\( \text{DO}_2 \)) – the amount of \( O_2 \) delivered to tissues on a per minute basis.

**Equation 3:** \[ \text{CaO}_2 = [Hb] \times 1.34 \times \text{SaO}_2 + (\text{PaO}_2 \times 0.003) \]

**Equation 4:** \[ \text{DO}_2 = \text{Cardiac output (CO)} \times \text{CaO}_2 \]
CaO\(_2\) is largely dependent on the amount of haemoglobin present: severely anaemic animals may therefore be critically hypoxic, even in the face of normal SaO\(_2\) and PaO\(_2\) values. Pulse oximetry is therefore not a substitute for serial measurement of PCV/Hb concentration or arterial blood gas analysis. Likewise, pulse oximetry does not provide a measure of the adequacy of ventilation. Normal SpO\(_2\) values are common in patients with marked hypoventilation, especially when the animal is receiving 100% O\(_2\): a pulse oximeter cannot detect hypercapnia. In addition, the newer pulse oximeters are capable of functioning even in the face of severe decreases in ABP – providing clinically useful readings under these conditions but reducing the usefulness of the monitor as an indirect assessor of the adequacy of peripheral perfusion (i.e. the presence of a strong signal or plethysmographic waveform does not necessarily indicate adequate perfusion as the older models tended to do).

In normal healthy conscious patients, cardiac output (CO) is continuously adjusted to meet O\(_2\) consumption (i.e. the amount of O\(_2\) taken from the microcirculation by all the tissues of the body), but if CO falls, so must DO\(_2\) (as shown in Equation 4). O\(_2\) consumption is clearly dependent on DO\(_2\) but the relationship is not linear. In the face of modest reductions in DO\(_2\), tissue oxygenation can be well maintained by drawing on physiologic reserves: blood is preferentially redistributed to vital organs and the proportion of O\(_2\) extracted from the blood increases from a normal value of 20-30% up to a maximum of about 60%. Cells continue to function aerobically at the expense of venous O\(_2\) content (which falls considerably). At some critical point however, compensatory mechanisms reach their limit and further reductions in DO\(_2\) result in an increasing imbalance between need and supply: O\(_2\) consumption now deceases as a linear function of delivery, anaerobic metabolism predominates, and tissue hypoxia ensues – often with surprising rapidity. This problem is exacerbated by factors that reduce CaO\(_2\) – particularly those that reduce haemoglobin concentration and/or SaO\(_2\) (e.g. anaemia, blood loss, V/Q mismatch and reductions in V\(_A\)).

Although V/Q mismatch and shunt result in both hypoxaemia and CO\(_2\) retention, the effects on oxygenation tend to be more profound because (1) CO\(_2\) is far more diffusible than O\(_2\), and (2) the increases in minute ventilation stimulated by any rise in PaCO\(_2\) tend to return PaCO\(_2\) to normal but can never fully compensate for the effects of venous admixture and shunt on PaO\(_2\) due of the shape of the oxyhaemoglobin dissociation curve. Provided there is at least some ventilation of a perfused alveolus (i.e. V/Q ratio is low but not zero), the administration of a FiO\(_2\) of > 35% will result in some improvement in oxygenation. However, true shunt is unresponsive to even 100% O\(_2\) because blood passing through unventilated alveoli cannot participate in gas exchange – it never “sees” the increased alveolar O\(_2\) concentrations and therefore retains its “venous” characteristics, returning this blood to the arterial side of the system. Here-in lies the problem: arterial oxygenation is determined not by the average O\(_2\) tension of the blood from the different regions of the lung (i.e. PaO\(_2\)) but by the blood’s average O\(_2\) content (i.e. CaO\(_2\)). The most important contributors to CaO\(_2\) are haemoglobin concentration and SaO\(_2\). But because blood leaving the ventilated regions of the lung is already nearly fully saturated, and because so little O\(_2\) is carried in solution in comparison to that bound to haemoglobin (even when a patient breathes 100% O\(_2\)), the administration of a high FiO\(_2\) can do little to offset the devastating effects of shunt. While a range of factors including age, pulmonary disease and species determines the amount of shunt in any given patient, studies suggest that anaesthesia per se increases shunt to about 10% of CO.
3. Anaesthetic monitoring devices: ABP monitoring

The effects of anaesthesia on cardiovascular function: reviewing the physiology

The primary role of the cardiovascular system is to maintain normal tissue perfusion and oxygen delivery ($DO_2$). Anaesthetic agents produce both direct and indirect effects on cardiovascular function, affecting such variables as heart rate and rhythm, myocardial contractility and vascular tone. In addition, general anaesthesia per se produces dose-dependent depression of the normal homeostatic control systems that regulate circulatory function, predisposing patients to intraoperative hypotension (HOTN) and reduced peripheral perfusion. Anaesthetic-induced reductions in cardiac output (CO), ABP and $V_m$ significantly reduce $DO_2$ – a problem further complicated by the inevitable “stresses” of a surgical procedure such as pain, blood loss, body position, hypothermia and tissue manipulation. Even healthy animals undergoing routine procedures may experience significant cardiovascular depression, while the effects in severely compromised patients may be profound.

Maintenance of normal ABP is perhaps the single most important function of the cardiovascular control-system because without it, the body is incapable of maintaining adequate peripheral perfusion. HOTN is defined as a systolic arterial pressure (SAP) < 90 mmHg or a mean arterial pressure (MAP) < 60-65 mmHg. Of the two, MAP is the more important measure of perfusion pressure because this is the average pressure available to “push” blood through the vascular network, over time. Although a MAP of $\geq$ 60 mmHg is usually considered necessary for adequate $DO_2$, conscious animals can employ local vascular control mechanisms to maintain blood flow to vital organs in the face of variable perfusion pressures (i.e. autoregulation) – at least on a short-term basis. Anaesthesia depresses autoregulation in a dose-dependent manner until blood flow to vital organs depends solely on MAP: when this falls below 60 mmHg, tissue perfusion is compromised and $DO_2$ becomes inadequate. Under normal circumstances, MAP is tightly regulated to ensure blood flow to vital tissues remains adequate. Deviations initiate multiple reflex responses that adjust CO and systemic vascular resistance (SVR), returning MAP to normal. Moment-to-moment adjustments are modulated primarily by baroreceptor-driven changes in autonomic activity, while variations in blood volume serve an important role in long-term (i.e. hours to days) control. Although the latter is ultimately the more dominant response, the baroreceptor reflex is crucial for maintaining MAP within normal limits and ensuring tissue perfusion remains adequate on a short-term basis.

Monitoring ABP: reviewing the technology

ABP is the most reliable and practical assessor of cardiovascular function available in every-day practice situations. Several methods for measuring ABP in anaesthetized animals are available. Although direct measurement via arterial catheterisation is the most accurate method, it is also the most invasive and technically demanding. Non-invasive techniques are appealing because they are easier and less expensive, although the information supplied is intermittent. All NIBP monitors use a similar technique to assess ABP based on a means of detecting blood flow, a means of temporarily occluding flow (usually via an inflatable cuff), and a means of measuring the pressure at which flow first returns when the occlusion is released. A number of NIBP monitors have been evaluated in dogs and cats including the Doppler ultrasonic flow detector and oscillometric-type devices such as the Dinamap™. Of these, the Doppler offers many advantages including simplicity,
portability, accuracy, relatively low cost, and the ability to be used on animals of almost any size. The Doppler provides an estimate of SAP and an audible indicator of pulse rate: SAP should always be > 90 mmHg. Disadvantages of this technique include the inability to accurately measure diastolic arterial pressure (DAP) or MAP, lack of continuous monitoring, and the fact that the monitor is not automated.

The oscillometric sphygmomanometry technique for ABP measurement was developed by Dinamap™ (Device for Indirect Non-invasive Automated Mean Arterial Pressure). Oscillometric NIBP monitors are automated and simple to use; however, readings are intermittent and may be less accurate than the Doppler in patients weighing < 7kg. Monitors of this type use a microprocessor to detect pressure oscillations arising from pulsations in an underlying artery, via an inflatable cuff. The cuff is placed around a limb or the tail base and inflated automatically to a pressure greater than SAP; then slowly deflated in a step-wise manner while simultaneously sensing the arterial pressure oscillations. The amplitude of the oscillations increases markedly at SAP, is maximal at MAP and falls away suddenly at DAP. These monitors provide a digital readout of SAP, MAP, DAP and pulse rate, and will alarm when these values fall outside preset limits. The oscillometric-type monitors cycle automatically and can be programmed to take readings at various intervals e.g. 2, 5, 10-min etc. Care should be exercised when using the STAT function as this cycles at 1-min intervals, and may result in tissue injury if used excessively as cuff-inflation will occlude distal blood flow 75% of the time. Normal values are as follows: - SAP = 110-160 mmHg (but > 90 mmHg is acceptable), MAP = 85-120 mmHg (but > 65 mmHg is acceptable), and DAP = 70-90 mmHg.

**ABP: looking beyond the numbers**

Normal cardiovascular function relies on a complex interaction of many variables and can be difficult to assess in terms of “overall performance”. ABP not only provides a measure of the pressure available to drive perfusion, but also acts as an indirect assessor of many cardiovascular parameters including heart rate (HR), stroke volume (SV), CO, circulating blood volume, venous return and myocardial contractility. Under normal circumstances, a change in one parameter is offset by a compensatory change in another to maintain normal tissue perfusion (e.g. CO is maintained in the face of a decrease in HR by a compensatory increase in SV). A fall in ABP is therefore indicative of a “stretching” of cardiovascular reserves: normal compensatory responses have been overwhelmed. Because most anaesthetic agents result in dose-dependent cardiovascular depression, ABP also acts as a useful indicator of anaesthetic depth. As a rule, ABP decreases as anaesthesia deepens, while surgical stimulation in a lightly anaesthetized patient results in abrupt, often dramatic elevations in ABP. However, patients at an appropriate depth of anaesthesia should always respond to a painful surgical stimulus with a small increase in ABP (i.e. they should always display a sympathetic response): failure to do so indicates excessive anaesthetic depth.

Despite its obvious importance, ABP is a relatively insensitive indicator of mild to moderate hypoperfusion because the body’s homeostatic mechanisms act to maintain ABP in the face of hypovolaemia. ABP is not synonymous with tissue perfusion: ABP may be normal in patients with significant reductions in CO and tissue blood flow (e.g. compensated hypovolaemic shock). Instead, ABP is an indicator of perfusion pressure i.e. the ability of the body to deliver blood to tissues. Measurement/calculation of CO and DO₂ would provide far better
information about the adequacy of peripheral perfusion, but are expensive, technically demanding and impractical in most small animals. However, ABP is related to CO according to the following equation, and while ABP may be normal in the face of reduced DO$_2$, HOTN is always a sign of impaired tissue perfusion.

\begin{align*}
\text{Equation 5:} & \quad ABP = \text{Cardiac output (CO)} \times \text{Systemic vascular resistance (SVR)} \\
\text{Equation 6:} & \quad CO = \text{Heart rate (HR)} \times \text{Stroke volume (SV)}
\end{align*}

Because ABP is the product of CO and SVR, a reduction in one of these factors must also reduce ABP. Many anaesthetic agents depress both myocardial contractility and systemic vascular resistance in a dose-dependent manner. While HOTN may arise from reductions in either CO or SVR, the latter seems the dominant factor in anaesthetized patients. Changes in SVR affect both ABP (vasodilation leading to HOTN) and peripheral tissue perfusion (vasoconstriction leading to reduced DO$_2$). Common causes of HOTN in anaesthetized patients include excessive anaesthetic depth, peripheral vasodilation, reduced myocardial contractility, and bradycardia; however, hypovolaemia (absolute due to blood loss or relative due to maldistribution), intraoperative haemorrhage, extreme tachycardia, arrhythmias, inappropriate positive pressure ventilation, and organ manipulation (e.g. vaso-vagal reflexes or compromised venous return) may also adversely affect ABP. HOTN is an extremely common anaesthetic complication – even in normal, healthy patients undergoing routine procedures – with reported incidences ranging from 7-38% in anaesthetized cats and dogs. The true incidence may actually be far greater, as CEPSAF showed routine intraoperative monitoring of ABP is still limited. Although normal patients can tolerate short periods of mild HOTN, prolonged or severe decreases in DO$_2$ may result in a variety of problems including acute renal failure, blindness, cardiac arrhythmias, brain damage, liver failure, failure of the gut barrier, and/or death.

As with any monitoring device, NIBP monitors are not infallible and are prone to user error. Doppler measurements have been shown to correlate well with direct measurements of systolic arterial pressure (SAP) in both conscious and anaesthetized dogs and cats, although in one study, Doppler readings in anaesthetized cats tended to underestimate direct measurements by 10-14 mmHg. Individual patient variation and small differences in “user technique” reduce absolute accuracy; none-the-less, the values measured are generally an accurate reflection of ABP. For this reason, trends in ABP may be more important than actual numbers; however, measurements indicative of hypotension always warrant further investigation. Oscillometric NIPB monitor readings have been shown to correlate well with direct ABP measurements in dogs weighing > 7 kg but are less accurate in smaller patients. Inaccuracies have also been reported in association with low heart rates (< 30 beats/minute), arrhythmias, intense peripheral vasoconstriction and HOTN.

**Conclusion**

Merril C Sosman is quoted as noting: - “You see only what you look for, you recognise only what you know”. With respect to management of anaesthetized patients, it is impossible to over-emphasize the importance of good monitoring skills combined with a sound knowledge of basic pharmacology, basic physiology and the adverse impact of anaesthesia on this. Current evidence suggests the use of anaesthetic monitoring devices reduces anaesthetic-associated mortality in small animals, just as it does in anaesthetized people. Knowledge
of correct use and expected values is essential when using these devices. However, an ability to “look beyond the numbers” may enable us to look further and recognise more problems than we might otherwise see, creating the potential to further reduce anaesthetic-induced morbidity and mortality and therefore improve patient outcome.

Key references and recommended reading