Performing anesthesia is a task that most veterinary technicians undertake on a daily basis. Intra-operative monitoring is imperative for optimizing all anesthetic procedures. In addition to allowing informed, flexible, and well-timed responses to changes in the patient’s status, it can also serve as a database for comparison prior to subsequent anesthetic episodes. A variety of equipment is available to monitor the patient’s physiologic parameters, including but not limited to stethoscopes, blood pressure monitors, electrocardiograph (ECG) tracings, pulse oximeters, end-tidal carbon dioxide monitors, and temperature probes. Is one monitor better than the others? One must first consider the overall effects of general anesthesia before evaluating monitors that would be ideal for assessing patients under general anesthesia. It is well known that inhalant anesthetics are potent respiratory depressants, and anesthetized animals may breathe up to 50% less as compared to the conscious state. They are potent vasodilators, readily causing hypotension at increased levels of anesthesia. Decreased cardiac output, central nervous depression, and muscle relaxation are also direct effects of inhalant anesthetics. With this information in mind, let’s examine the various monitoring modalities and exactly what they tell us about the anesthetized patient.

**Pulse Quality and Stethoscope Use**

Pulse palpation is useful in evaluating the heart rate (unless an arrhythmia is present), and is determined by the difference between the systolic and diastolic phase. Pulse quality will feel similar whether the blood pressure is 120/80 with a mean arterial pressure (MAP) of 90mmHg or 100/60 (MAP 70mmHg). Therefore, pulse quality is NOT a reliable indication of adequate blood pressure. Although it can be presumed that weak pulses likely represent hypotension, normal pulses do not necessarily represent normotension. Additionally, hypertension cannot be diagnosed by pulse palpation. Normal pulse rates can vary greatly based on species and breed. The normal heart rate for an anesthetized dog should be 50–180 (depending on body size) and the normal heart rate for cats should be 100–220.

The stethoscope is used to auscult the heart rate and rhythm. Although heart murmurs, arrhythmias, and other cardiac pathology (e.g., tamponade) can often be identified with a stethoscope, auscultation alone does not provide relevant information in regard to cardiac function or output. Hence, the information obtained from pulse quality and chest auscultation tells little about anesthetic depth in the anesthetized patient.

**Electrocardiography (ECG)**

ECG monitoring is commonplace during general anesthesia. It is important to ensure good contact of leads to skin by using either ECG paste or alcohol when placing ECG leads. Avoid wetting large areas of the skin and direct contact with the table. Exact lead locations are not as important as ensuring that all waves are present (even if they are inverted). The P-wave represents atrial depolarization. The QRS-wave represents ventricular depolarization. The T-wave indicates ventricular repolarization. It is important to realize that an ECG tracing does not provide information about chamber size, or how efficiently the heart is ejecting blood. Therefore, the ECG should be used strictly for the detection of dysrhythmias during the perianesthetic period.

Induction agents and disease processes may predispose patients to cardiac arrhythmias. Other potential causes of cardiac arrhythmias may include an inadequate or excessive anesthetic depth, pain, hypoxia, hypercapnia, heart or lung disease, and traumatic myocarditis. Electrolyte imbalances and acidosis may also be a source of cardiac arrhythmias. It is not always necessary to treat arrhythmias unless they are causing adverse affects to the patient. Bradycardia is commonplace in patients undergoing general anesthesia. Bradycardia can be defined as a heart rate of <80 beats per minute in a small dog, or <60 beats per minute in large dogs. In cats, bradycardia is defined as <100 beats per minute. There are numerous causes for bradycardia, which may include drug side effects, excessive vagal tone, hypertension, hyperkalemia, uremia, hypothermia, increased intracranial pressure, profound hypoxemia, and deep-level inhalants, among others. Moreover, when underlying factors such as hypoxemia or hypovolemia are present, bradycardia can be a sign of impending cardiac arrest. Tachycardia is defined as >180 beats per minute in a dog, and >200 beats per minute in cats. Tachycardic states can lead to hypotension. There are many causes of tachycardia, which may include but are not limited to, drugs, an inadequate plane of anesthesia, hyperthermia, anaphylactic reactions, hypovolemia, early-stage hypercarbia, and numerous disease states.
Pitfalls associated with interpretation of ECG tracings can include lead malpositioning due to broken clips or loose connections to the monitor. Electrical interference caused by cautery or other operating room equipment can also be problematic. Rate inaccuracies can occur based on the size of the waveform, resulting in either double-counting or non-counting issues. Patient motion secondary to shivering or increased respiratory rates can cause blurred or erratic tracings. Additionally, some patients may demonstrate pulse-less electrical activity (normal to near-normal ECG tracing but with very weak or nonexistent palpable peripheral pulses) or electromechanical dissociation—normal or near normal ECG tracing with very poor (or zero) cardiac contractility. As a final caveat, electrical activity is often the last aspect to completely disappear prior to the pronouncement of death.

**Blood Pressure**

There are 2 factors that affect blood pressure, and 2 factors that affect cardiac output. *Blood pressure* is determined by the cardiac output and total peripheral resistance. Total peripheral resistance is defined as the resistance to blood flow created by the peripheral arterial system as well as capillary beds. *Cardiac output* is determined by a combination of the heart rate and stroke volume. The heart rate is the speed at which the heart is pumping, or the number of contractions per minute. The stroke volume is the amount of blood ejected with one contraction of the heart. *Blood pressure* itself may be defined as “the lateral force on the arterial wall.” Additionally, blood volume is a major determinant of blood pressure. Baroreceptors located in the carotid and aorta help regulate blood pressure through the central nervous system control centers.

It is important to realize that all patients experience some degree of hypotension during general anesthesia, and if the patient has preexisting conditions that decrease blood pressure, hypotension will be exacerbated during anesthesia. Normal arterial blood pressure values for canines are systolic 110–119mmHg and diastolic 55–110mmHg, and for felines, systolic 120–170mmHg and diastolic 70–120mmHg.

Although *direct* blood pressure monitoring is considered the “gold standard,” it is highly impractical when it comes to routine blood pressure monitoring in most privately owned veterinary facilities due to the advanced skill level required to place arterial catheters and the need for 24-hour care. Therefore, only indirect methods of blood pressure monitoring will be discussed. There are 2 methods to measure blood pressure indirectly—by using either a Doppler or an oscillometric device (e.g., Dinamap, Cardell, petMAP). Regardless of the method used, selection of the correct sized blood pressure cuff is imperative for providing the most accurate results. The width of the cuff should extend 40% around the circumference of the limb. When the cuff is determined to be too small, the next wider size should be selected. In cats, it is acceptable to use a cuff that is only 30% of the circumference of the limb. The cuff should be snug, but not too tight. It is acceptable to use a piece of tape to keep it from becoming dislodged during cuff inflation. **Selection of an inappropriate cuff size is the most common source of errors.** If the cuff is too narrow or too loose, the reading will be falsely high. If the cuff is too wide or too tight, the reading will be falsely low. Additionally, cuffs placed over a joint are less likely to compress the artery, and a hole in the cuff may result in pressure leaks that occur too quickly for the machine to record. Acceptable cuff locations include the forelimb, tail, and hindlimbs, where the areas proximal to the carpus and tarsus work best. The ventral tail is a good choice in cats and short-legged breeds such as the bassett hound and dachshund.

**Oscillometric methods** detect intracuff changes caused by the pulse wave. They calculate the systolic, diastolic, and mean arterial pressure (MAP) as well as the heart rate. They frequently can be programmed to obtain readings at various time intervals (e.g., once per minute, once per hour.) Most of these monitors are designed to detect heart rates of greater than 30–40 beats per minute.

**Doppler methods** use a “return-to-flow” principle to detect the systolic blood pressure. Doppler measurements are most accurate when the systolic blood pressure is within normal limits and when the patient has good peripheral perfusion. Using a Doppler to measure blood pressure is very simple, and all technicians should be able to proficiently perform this simple task. In cats it is hypothesized that the resultant reading probably represents the MAP; therefore a correction factor of 14mmHg is added to the reading to more accurately reflect actual feline systolic pressures. Because the “white coat” phenomenon has been well documented in humans, the patient should be calm and as well-acclimated as possible to avoid an inadvertent false diagnosis of hypertension or hypotension. Perform several readings in a conscious patient and average the results. Be warned that a Doppler can mistake heavy respirations for blood flow. Profound arrhythmias, hypothermia, patient motion, low batteries, and electrical interference can also impede obtaining good readings.
There are drawbacks associated with indirect methods of blood pressure monitoring. In general they all tend to underestimate the actual blood pressure, and all work best when the MAP is 60–100mmHg. Patient movement, smaller patient size (< 5.0kg), cold or vasoconstricted patients, and patients with short legs or excessive skin will all adversely affect results. Additionally, measurements may be difficult to obtain in patients with limb edema.

**Pulse Oximetry**

Pulse oximeters provide continuous and noninvasive monitoring of pulse and an estimate of arterial hemoglobin saturation (SpO₂), but do not provide data on the amount (partial pressure) of oxygen in arterial blood, as dissolved in plasma (PaO₂). Pulse oximeters can be used on the lip, tongue, ear pinna, prepuce, vulva, toe web or digits, metacarpus, tail, rectal mucosa, or flank skin folds. If a skin-fold site is selected, it should ideally be hairless, nonpigmented, and fairly thin-skinned (but not overly so). In large animals consider using the nostril/nasal septum as well.

There are 5 main types of hemoglobin: oxyhemoglobin, reduced hemoglobin (deoxyhemoglobin), methemoglobin, carboxyhemoglobin, and fetal hemoglobin. Since 95% of oxygen delivery to tissues is by oxyhemoglobin, saturation is of high clinical significance. Not all types of hemoglobin are capable of transporting oxygen, and as such are termed “dysfunctional hemoglobins.” The presence of other light-absorbing types of hemoglobin such as methemoglobin (e.g., resulting from the use of Cetacaine® [benzocaine] sprays prior to intubation, or from the use of nitrates and nitrites, metoclopramide, or sulfa drugs) and carboxyhemoglobin (as a result of carbon monoxide exposure) will cause the pulse oximeter to overestimate arterial oxygen saturation. Conversely, extraneous blood-borne dyes (such as methylene blue) are known to potentially lower SpO₂ readings to 85%, regardless of the true saturation value. Pigmented substances such as bilirubin lipids (hyperbilirubinemia) may also affect arterial blood light absorption and alter SpO₂ values. Other causes for erroneous SpO₂ values include severe anemia or hemodilution. Moreover, the pulse oximeter may display an SpO₂ reading of 100%, in spite of the considerable decrease in arterial blood oxygen content secondary to low hemoglobin values.

Further pitfalls of pulse oximetry use include erroneous and unreliable results or potential complete loss of function when peripheral pulsations are reduced or absent, as in the case of hypotension, hypothermia, or hypovolemia. Other conditions that can contribute to unreliable pulse oximeter readings include arrhythmias and tachycardia, increased venous pulsations (e.g., right heart failure, tricuspid regurgitation, etc.), and movement artifacts (e.g., shivering). Erroneous pulse oximeter readings may also occur when using certain Xenon arc surgery lights (resulting in an SpO₂ reading of 100% and a pulse rate of 180–225), without the probe being attached to a patient! Using rectal or esophageal probes can eliminate interference from external light sources such as fluorescent lights, heat lamps, and surgical lamps.

Finally, be aware that the pulse oximeter is surrounded by controversy in regard to its use as a monitoring device—it is either prized or despised. This is due, in part, to the oxyhemoglobin dissociation curve, which describes the nonlinear relationship between PaO₂ and SpO₂. For example; patients breathing 100% oxygen may have a PaO₂ that is 5 times greater than the SpO₂ (e.g., PaO₂ = 500 mmHg; SpO₂ = 100%). Since the oxyhemoglobin dissociation curve is sigmoid shaped, the hemoglobin saturation would demonstrate only a very slight increase—going from 98% to 100%. Pulse oximeters are most beneficial when evaluating desaturation, such as when the reading drops to below 90%, which corresponds with a PaO₂ that is less than 60mmHg. Pulse oximeters are most accurate within 2% to 6%, and within the 80 to 100 percentile.

**End Tidal Carbon Dioxide (ETCO₂)**

End tidal carbon dioxide (ETCO₂) is the result of expired gases from the alveoli. End tidal carbon dioxide analysis can be used to help assess acid/base status as well as the adequacy of patient ventilation in a variety of clinical situations, such as during anesthesia when effects of drugs and inhalants can decrease tidal volumes by more than 25%, or during long-term ventilatory assistance as with the use of a mechanical ventilator. An abrupt decrease in ETCO₂ can be an early and reliable indication of an impending cardiovascular collapse or cardiac arrest. Consequently, ETCO₂ production can be used to assess the effectiveness of cardio-pulmonary-cerebral-resuscitation (CPCR) techniques, since delivery of carbon dioxide from the lungs requires blood flow, cellular metabolism, and alveolar ventilation.

Capnometers and capnographs monitor ETCO₂ by evaluating samples of the patient’s exhaled gases taken from the anesthetic circuit via an adapter placed on the end of the patient’s endotracheal tube. This adapter must be placed
precisely at the end of the patient’s nose to eliminate excessive dead space and prevent rebreathing of carbon dioxide. Capnometers provide only minimum and maximum ETCO₂ values, while capnographs provide a graphic display of exhaled carbon dioxide as each breath is taken. Diagnosing abnormalities in ventilation or anesthetic circuit function is easier using the graphical data provided by a capnograph.

Normal ETCO₂ values are 35–45mmHg. Under normal circumstances, ETCO₂ typically underestimates the arterial carbon dioxide partial pressure (PaCO₂) by a clinically insignificant 2–5mmHg. End tidal carbon dioxide values above 45mmHg indicate inadequate ventilation, necessitating ventilatory assistance via manual or mechanical means. Conversely, by allowing modest increases in ETCO₂ (up to 50mmHg), the anesthetist can bolster arterial blood pressure via endogenous catecholamine release. Nonetheless, the highest ETCO₂ permissible should be 60mmHg.

There are caveats to ETCO₂ monitoring: Esophageal intubation, occlusion of the endotracheal tube, inadequate seal on the endotracheal tube, anesthetic circuit dysfunction/disconnects, moisture within the sampling line, hyperventilation, or respiratory and/or cardiac arrest are all potential causes of failure to detect carbon dioxide. Elevated ETCO₂ levels may occur as a result of hypoventilation due to airway obstruction, pneumothorax, body positioning, or lung disease, or during periods of acutely increased metabolism (e.g., thyroid storm or catecholamine release). Significant disparities between PaCO₂ and ETCO₂ indicate an inefficiency of gas exchange (e.g., dead space ventilation), which may be secondary to pulmonary embolism, thromboembolism, decreased cardiac output, or perhaps as a result of mechanical ventilation (intermittent positive pressure ventilation). Explanations for elevated ETCO₂ and inspiratory carbon dioxide may include anesthetic machine malfunction (e.g., malfunctioning valves within the breathing circuit), unsuitable fresh gas flow rates (e.g., non-rebreathing circuits), or exhausted carbon dioxide granules. Therefore, end tidal carbon dioxide is best analyzed in conjunction with an arterial blood gas sample to yield the most complete status of respiratory function.

**Temperature**

Hypothermia is not only one of the most common anesthetic complications, but also the easiest to document without special equipment. The hypothalamus closely regulates core body temperature. However, this regulation can be impaired in pediatric and geriatric patients; lean breeds; and those with organ failure, large wounds, or infections. Almost all anesthetized or sedated patients will lose body heat under general anesthesia, with the exception of adult Nordic breeds (i.e., samoyed, Siberian husky, Alaskan malamute), which can actually become hyperthermic. Small patients are at the greatest risk, due in large part to their small body-surface-to-mass ratio. Hypothermia is exacerbated in prolonged surgical procedures, especially those that expose open body cavities or use cold irrigation solutions. Hypothermia-induced bradycardia is typically nonresponsive to anticholinergics. Hypothermia contributes to delayed drug metabolism and decreased hepatic metabolism, resulting in prolonged recovery and potential drug toxicity. Clotting times can be prolonged due to impaired platelet function and hemoconcentration with sludging. Hypothermia also suppresses immune function and may lead to increased infection rates.

Obviously, prevention is key when addressing hypothermia. Rewarming should be considered when the patient’s temperature drops to ≤ 97.6°F. There are a variety of ways to maintain an envelope of warm air around perioperative patients. Convection-type warm air devices (e.g., BAIR Huggers®) are the most effective, followed by circulating warm water blankets. At least 60% of the body surface area must be in contact with the external heat source for rewarming efforts to be most effective. If latex gloves or bottles of warm water are to be used for smaller patients, it is essential that they be initially warmed to a temperature of ≤107°F and removed once they cool to the temperature of the patient. Commercially available wire electric heating-pads and heat lamps have been associated with uneven heating, thermal injury, and electrocution and should be avoided.

In conclusion, the information provided by anesthetic monitors is only as valuable as the person interpreting the data. Therefore, always remember to “Treat the patient, NOT the monitor.”

**References**


