Complications During Anesthesia: Evaluation and Treatment

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Abstract

Physiologic complications are common during anesthesia. In an effort to decrease morbidity and mortality, good practice dictates vigilant monitoring and effective treatment of complications during anesthesia. Therefore, veterinarians must be able to interpret the data from the monitoring equipment used during anesthesia and to treat complications as they occur. The equipment that is commonly used in veterinary practice and treatment algorithms for common complications are discussed here.

Key Content

• Perianesthetic death occurs in small animals in approximately 1 in 1000 anesthetic administrations—about 10 times greater than the incidence in humans.

• Differences in mortality rates between human and veterinary patients probably result from differences in monitoring, training, cost, patient population, and reporting.

• The cardiovascular goals during anesthesia are to provide warm, oxygenated, glucose-rich blood (perfusion) to all vital organs.

• Hypotension is the most commonly reported anesthetic complication.

• Blood pressure is often used as an indirect measure of cardiac output and organ perfusion.

• The ECG is a recording of the electrical potentials generated by myocardial cells.

• The most common respiratory complication in anesthetized patients is hypoventilation (hypercapnia) associated with anesthetic drug administration.

• Assessment of oxygenation and ventilation (carbon dioxide) can be done indirectly by assessing respiratory rate, depth of ventilatory excursion, and mucous membrane color, although all three can be misleading.

• A pulse oximeter measures the amount of oxygen that is bound to hemoglobin and is reported as a percentage of hemoglobin that is saturated with oxygen.

• Hypothermia is a common but often overlooked complication of anesthesia.

"Going to sleep is easy…waking up can be a challenge."

Perianesthetic death occurs in small animals in approximately 1 in 1000 anesthetic administrations (1). This is 10 times greater than the incidence in humans (about 1/10,000) but less than in horses (about 1/100) (2).

In humans, a death associated with anesthesia is 1/1,000,000, with 10% of those deaths associated with anesthetic complications (3). In small animals, anesthetic complications were reported in about 2% of patients (1).

Differences in mortality rates between human and veterinary patients (dogs and cats) are likely multifactorial. Some of the differences in human compared with veterinary anesthesia may account for the differences in mortality, including monitoring, training, cost, patient population, and reporting.

All people who are anesthetized are continually monitored by trained healthcare professionals, either anesthesiologists or nurse anesthetists. These individuals have had advanced training in anesthesia and many have even further advanced training in particular areas (eg, pediatric cardiology, obstetrics). In addition, humans are monitored by professionals who have no other responsibilities in the operating room. Anesthetic monitoring is also quite different. The guidelines from the Anesthesiology Society of America (ASA) have specific requirements for personnel and for mechanical monitoring (4). The comparable guidelines from the American College of Veterinary Anesthesiologists (ACVA) only suggest training and mechanical monitoring (5).

Since it is reasonable that differences in patient monitoring account for some of the increased mortality in veterinary patients; greater attention should be paid to our anesthetized patients. At a minimum, all patients should be monitored for cardiovascular stability, adequate respiratory function, and temperature.

Goals

The cardiovascular (CV) goals during anesthesia are to provide warm, oxygenated, glucose-rich blood (perfusion) to all vital organs. The body uses

oxygen and glucose to make adenosine triphosphate (ATP) that fuels the body. Anaerobic metabolism cannot provide enough ATP, and its by-product is lactic acid (ie, a contributor to acidemia).

Adequate tissue perfusion requires sufficient cardiac output (CO) as well as sufficient oxygen content (the amount of oxygen carried on hemoglobin plus the oxygen dissolved in plasma). CO is difficult to measure clinically and is often inferred from blood pressure. Remember that the definition of CO is:

$$CO = \text{stroke volume} \times \text{heart rate}$$

Remember also that stroke volume is dependent on preload (volume), afterload (systemic vascular resistance), and cardiac contractility.

Circulation Monitoring

At a minimum, all patients should have pulse quality and heart rate assessed. Pulse quality is frequently misinterpreted as blood pressure. Pulse quality (pulse pressure) is the difference between systolic arterial pressure and diastolic arterial pressure. When there is a big difference, the pulse feels stronger. A strong pulse may occur with hypotension as long as there is a large enough difference systolic and diastolic pressure. Similarly, patients that are vasoconstricted (higher diastolic pressure) may have weaker pulse pressure if the difference is small.

Hypotension is the most commonly reported anesthetic complication. It can occur in healthy patients in response to vasodilatory drugs (eg, inhalants, acepromazine), loss of sympathetic tone due to excessive anesthetic depth, and use of local anesthetics. Renal perfusion is best associated with mean arterial blood pressure; mean arterial pressure below 60 mm Hg is considered hypotensive and should be addressed.

Better monitoring includes blood pressure assessment and ECG.

Blood Pressure
Blood pressure is often used as an indirect measure of CO and organ perfusion. Clinically, it can be measured in three ways: Doppler, oscillometric, and direct (invasive).

How Does It Work?
Doppler. Use the Doppler probe to find an audible peripheral pulse, inflate a sphygmomanometer cuff to occlude flow to that vessel, and slowly deflate the cuff until the audible pulse is heard again. The pressure at which the pulse resumes is the systolic pressure. There is some evidence that in cats, blood pressure measured this way is somewhere between mean and systolic pressure (6).

Oscillometric. Automated blood pressure monitors use a cuff that measures changes in oscillations. The cuff is inflated until the pulse is occluded (no oscillations) and is slowly deflated until oscillations resume (ie, systolic pressure). The point of maximum oscillations is the mean blood pressure, and the diastolic pressure is usually calculated from the other two values. Anything that can change oscillations (eg, shivering) can negatively affect accuracy.

Direct. For invasive (direct) blood pressure. A catheter is placed in a peripheral artery and connected to a pressure transducer. The transducer can then generate an arterial wave (graphically) and measure systolic, mean, and diastolic pressure.

What Does It Tell You?
Depending on the method, at a minimum blood pressure monitoring will determine systolic blood pressure and often a mean and diastolic blood pressure. Although somewhat oversimplified, systolic pressure is most reflective of CO and diastolic pressure is most reflective of vessel tone. Mean blood pressure is the best indicator of organ (especially renal) perfusion.

Limitations
Higher blood pressure is usually associated with increased CO and increased perfusion; however, that does *not* occur with increases in systemic vascular resistance. For example, a patient administered medetomidine will initially have vasoconstriction and decreased CO. In that patient, blood pressure is increased with decreased CO.

Accuracy is best with direct pressure measurement, followed by Doppler and then by oscillometric methods. Accuracy of the oscillometric method is based on many things--appropriate size of cuff, shape of leg, heart rate, and rhythm. It is not surprising that accuracy could be different when applied to a Bulldog leg vs a Retriever leg.

Treatment
After determining that your patient is hypotensive, direct treatment at the underlying problem. Recall that CO is dependent on stroke volume and heart rate, and that stroke volume is affected by preload, systemic vascular resistance, and cardiac contractility.

Healthy patient. For illustrative purposes, consider a healthy hypotensive dog presenting for an orthopedic procedure. Following are the steps involved in treating this patient.

• Assess anesthetic depth and determine whether the dog could be vasodilated. If you suspect vasodilation, treat with a fluid bolus (crystalloids at 10-20 ml/kg).
• Assess heart rate. If the rate is slow and the patient is hypotensive, treat with an anticholinergic (eg, glycopyrrolate).

• If still hypotensive, consider treating with a colloid (eg, hetastarch), especially if the patient has low protein (to increase oncotic pressure).
• If the other treatments did not resolve the hypotension, administer a positive inotrope (dopamine 5-7 mcg/kg/min).

Compromised patient. For illustrative purposes, consider a patient with sepsis. We can assume that there is inappropriate vasodilation (due to sepsis) and likely hypoproteinemia. These patients are also often volume-depleted. The approach to treating hypotension in this patient includes 1) using as little inhalant as possible (to avoid exacerbating vasodilation); 2) providing oncotic support (plasma or hetastarch); 3) providing volume resuscitation (crystalloids); and 4) administering a vasoconstrictor, such as norepinephrine. Norepinephrine has been shown to be beneficial in treatment of septic shock (7).

ECG
How Does It Work?
The ECG is a recording of the electrical potentials generated by myocardial cells. The potentials produced are small (low voltage), so using conductive gel lowers the skin's electrical resistance, but artifacts and a poor signal can still be a problem. A lead records the electrical signals of the heart from electrodes that are placed at specific points on the patient's body. When a depolarization wavefront moves toward a positive electrode, it creates a positive deflection on the ECG; when a depolarization wavefront moves away from a positive electrode, it creates a negative deflection.

What Does It Tell You?
ECGs are useful to assess the rate, rhythm, and conduction patterns of the heart. Each complex can also be inspected to look at individual waves (P, QRS, T) and to see whether the association is normal and that the waves are a normal size. Abnormal waves can also be identified, which can aid in clinical diagnoses (eg, premature ventricular contractions, hyperkalemia, or ST-segment depression). Although the ECG only measures electrical activity, such activity is generally associated with a beating heart (except electromechanical dissociation).

Limitations
Generation of ECG waves does not give *any* information about CO. Normal rhythms may be associated with poor CO, and abnormal rhythms (eg, atrial fibrillation) may be associated with adequate CO. Many ECG monitors autocalibrate the wave size; therefore, wave size may be distorted and misleading. Pulseless electrical activity (previously called electromechanical dissociation) can produce a fairly normal-looking ECG rhythm in the absence of a heartbeat or circulation.

Treatment

The decision to treat arrhythmia should be based on the physiologic effect of the arrhythmia and the potential to degenerate into a lethal rhythm. The most commonly accounted abnormal rhythms under anesthesia include the following.
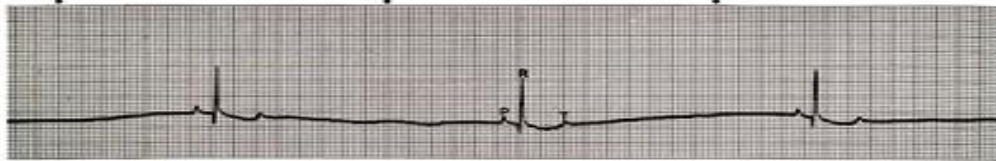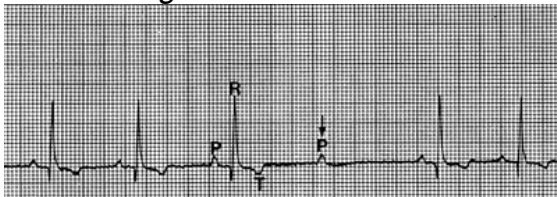
Sinus bradycardia

Fig. 6–8. Sinus bradycardia at a rate of 40/min in a dog with acute renal failure. The rhythm is regular with only a slight variation in the R-R intervals.

The decision to treat sinus bradycardia should be made globally. Is the rate inappropriate for the patient? Are there escape beats? Is CO being affected? Is the bradycardia in reflex to hypertension (think alpha-2 agonists)? Treat with an anticholinergic (eg, glycopyrrolate 0.01mg/kg IV).

*Second-degree heart block*

Second-degree heart block is commonly seen in anesthetized patients that have been given opioids (eg, morphine, hydromorphone). Opioids cause slowing of firing at the sinoatrial node (bradycardia) as well as slowed conduction through the atriventricular node (second-degree heart block). The decision to treat should be based on CV stability (or instability). Treat with an anticholinergic (eg, glycopyrrolate 0.01 mg/kg IV).

Premature ventricular contractions
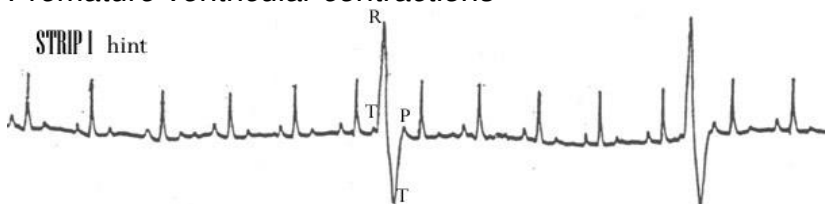
Anesthetized patients occasionally have benign premature ventricular contractions; however, these contractions generally indicate some myocardial irritability (hypoxemia, acidemia, electrolyte abnormalities). Treatment should be directed at fixing the underlying problem. However, if the premature contractions are negatively affecting CO or are multifocal, they should be treated with lidocaine.

Ventricular tachycardia
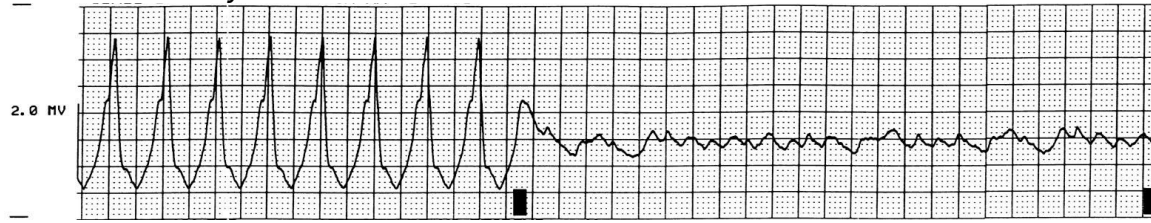
Ventricular tachycardia generally affects CO by a decrease in diastolic filling time (fast rate) and can degenerate into ventricular fibrillation. It is treated with lidocaine.

Hyperkalemia
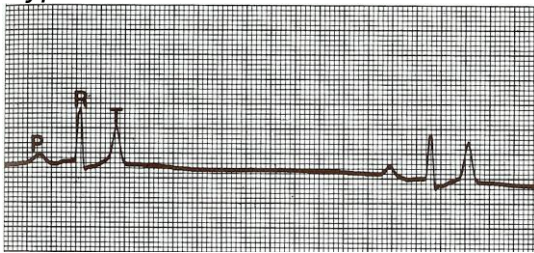
Hyperkalemia can alter ECG appearance and cause bradyarrhythmia. Characteristic changes associated with hyperkalemia include low or lost p waves and tall, spiked T waves. Treatment should be directed at lowering extracellular potassium (dextrose, insulin, bicarbonate), decreasing arrhythmias (calcium), and eliminating the underlying problem (unblock the cat!).

Respiratory Monitoring

Respiratory complications are frequent anesthetic problem. The most common complication is hypoventilation (hypercapnia) associated with drug administration. Most premedications, induction agents, and inhalants cause hypoventilation. Accumulation of carbon dioxide in small amounts is benign (especially when the patient is on 100% oxygen), but remember that it acts as an acid, and hypoventilation by definition causes respiratory acidosis.

If hypoventilation results in apnea or if the patient is not given supplemental oxygen, hypoxemia may occur. Recall that the body needs oxygen and glucose to make ATP.

Assessment of oxygenation and ventilation (carbon dioxide) can be done indirectly by assessing respiratory rate, depth of ventilatory excursion, and mucous membrane color, although all three can be misleading. Better monitoring would include a pulse oximeter and capnometer/capnograph.

Pulse Oximeter
How Does It Work?

A pulse oximeter measures the amount of oxygen that is bound to hemoglobin and is reported as a percentage of saturation (SpO_2). Since hemoglobin with oxygen and hemoglobin without oxygen absorb red and infrared light wavelengths differently, the percentage of one form versus the other can be measured. Hemoglobin absorbs more visible red light, which is why it appears blue. Complex computer programs help identify pulsatile movement (to differentiate from a vein) and calculate the percentage of saturation. In room air (21% oxygen), most mammals have an SpO_2 between 95% and 100%.

What Does It Tell You?
Pulse oximeters are valuable because they provide information on whether the patient has access to oxygen (eg, oxygen tank turned on), whether the patient is breathing, whether oxygen is exchanging in the alveoli, and whether CO is adequate for oxygen to reach the peripheral circulation (ie, the generation of a pulse at a peripheral site).

Limitations
The number generated by a pulse oximeter does *not* provide information about whether the amount of hemoglobin is adequate to support the patient but whether hemoglobin has oxygen attached to it. For example, a geriatric cat with a packed cell volume of 9% (hemoglobin approximately 3%) probably does not have enough oxygen-carrying capacity to supply the body with sufficient oxygen, but the pulse oximeter could easily read 100% saturated. Since many animals are close to 100% saturated on room air, it can be difficult to assess 100% saturation when higher percentages of oxygen are administered. This means that the difference between arterial oxygen of 100 mm Hg or 500 mm Hg cannot be determined with a pulse oximeter. Although a pulse is required for a reading, the pulse oximeter does not indicate adequate perfusion. Altered hemoglobin (methemoglobin or carboxyhemoglobin) will alter the accuracy of SpO_2 readings. Methemoglobin will cause the SpO_2 to read 85%, and carboxyhemoglobin generally gives a falsely high reading in patients with carbon monoxide poisoning.

Treatment
Hypoxemia is caused by one of five physiologic events: low inspired oxygen, hypoventilation, ventilation-perfusion (V/Q) mismatch, shunt, or diffusion impairment. When presented with a hypoxemic anesthetized patient, I address the first two quickly. Is the oxygen on and connected? Is my patient breathing? Does giving a breath resolve the problem? If not, then I consider the remaining three in light of my patient. Diffusion impairments are unusual in veterinary patients. V/Q mismatch is most common with patients with lung pathology (eg, pulmonary edema, pulmonary thromboembolism). Patients with lung pathology often need continuous positive pressure ventilation.
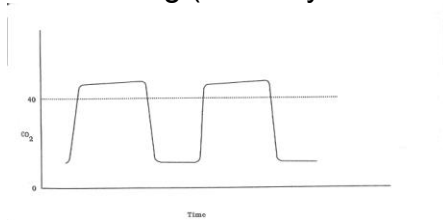
Capnograph
How Does It Work?

A capnograph (or capnogram) is an indirect measure of ventilation. It measures the amount of carbon dioxide in the breath at the end of expiration ($ETCO_2$). Since carbon dioxide easily moves across the capillaries and alveoli, the assumption is made that the amount of carbon dioxide at the end of the breath (end-tidal volume) is equivalent to the carbon dioxide in the blood. In healthy, awake animals, the difference between the arterial and end-tidal measurements is indeed close (a difference of about 5 mm Hg). Therefore, capnography allows us to estimate ventilation on the basis of the amount of carbon dioxide in the breath. This amount of carbon dioxide is measured by comparing with a reference gas for absorption of infrared light. By plotting the amount of carbon dioxide exhaled over time, a capnograph can be generated (a graphic representation). The size, upper and lower limits, and shape of the capnogram is quite useful in clinical diagnoses of both respiratory and mechanical problems.

What Does It Tell You?

Machines that just give an $ETCO_2$ number provide information about ventilation and indirectly CO. If CO is insufficient to bring carbon dioxide to the lungs, then the amount exhaled (even with normal ventilation) will decrease. Machines with a waveform readout (capnograph) provide a great deal of information about the patient (see images below). Inspection of the waveform can give information about the airways (eg, asthma), whether there is a leak in the system (machine or endotracheal tube cuff), and whether there is rebreathing of carbon dioxide (eg, carbon dioxide absorbant has expired, or one-way valves are not functioning). Capnographs are useful in cases of cardiac arrest, as they can indicate successful cardiopulmonary resuscitation as well as the return of spontaneous circulation.
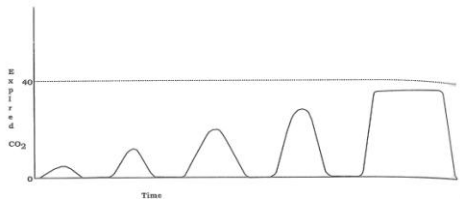
Rebreathing (one-way valves missing; carbon dioxide absorbant expired)
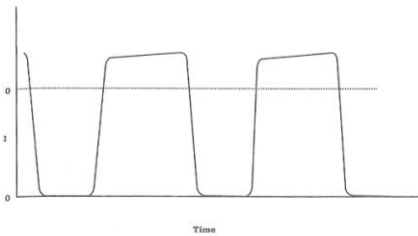

Airway obstruction (asthma, blocked tube)
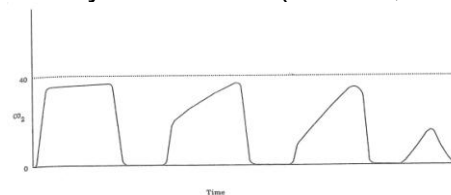
Return of circulation (postarrest)

Hypoventilation

Limitations
Capnography is based on the idea that the end-tidal carbon dioxide concentration reflects the arterial carbon dioxide concentration ($PaCO_2$). Although this is true in healthy, awake patients, the difference between the $ETCO_2$ and $PaCO_2$ can increase with disease (eg, pulmonary thromboembolism), anesthesia, or positioning. The *only* true way to know arterial carbon dioxide is to measure it (blood gas).

Treatment
Hypoventilation should be treated if carbon dioxide is greater than 60 mm Hg or if the patient is acidemic. Anesthetic depth should be assessed. If needed, ventilation should be assisted or controlled until anesthesia is discontinued.

Temperature

Hypothermia is a common but often overlooked complication of anesthesia. Anesthetic drugs affect the thermoregulator centers in the hypothalamus as well as encourage peripheral heat loss (vasodilation). Although often considered benign, hypothermia has significant physiologic consequences.

Consequences of Heat Loss
Immune System/Healing
Hypothermia impairs the immune system by decreasing oxidative killing by neutrophils, reducing phagocytosis, suppressing leukocyte migration, causing protein wasting, and decreasing synthesis of collagen. In humans, hypothermia increases the wound infection rate three-fold compared with normothermic patients (8).

Hematology and Coagulation

Hypothermia increases blood viscosity and slows enzymatic reactions of intrinsic and extrinsic pathways. There is reduced platelet function and reversible prolongation of coagulation times (9). Hypothermic humans lose more blood and require more transfusions than equally matched normothermic patients (10).

Cardiovascular
Hypothermia decreases CO, increases the concentration of norepinephrine, and causes vasoconstriction. In dogs, arrhythmias are likely to occur at a core body temperature of about 31° C, and ventricular fibrillation is likely with temperatures below 30° C (11).

Metabolism
Hypothermia is associated with decreased liver metabolism and renal excretion of drugs. The slowed metabolism of anesthetic drugs can prolong recovery times and increase the potential for overdosing.

Consequences of Shivering
Shivering increases metabolism and can increase heat production by 500%. Although it is effective in raising body temperature, shivering increases myocardial oxygen demands, glucose needs, and raises intracranial and intraocular pressure. Aside from the metabolic demands of shivering, humans describe it as the most unpleasant memory of their perioperative experience.

Increased Morbidity
Human studies have repeatedly shown that even mild intraoperative hypothermia is associated with increased time in the ICU and total increased hospitalization times (8). Although no veterinary studies have examined the morbidity associated with hypothermia, it is reasonable to believe that similar increases might occur.

Shift in the Oxygen Dissociation Curve
Hypothermia is associated with a left shift to the oxygen dissociation curve. This shift results in greater binding of oxygen to hemoglobin and less off-loading of oxygen at the tissue level. It is possible that poor offloading in conjunction with vasoconstriction will decrease tissue perfusion.

Decrease in Minimum Alveolar Concentration
Decreased body temperature is associated with a decrease in minimum alveolar concentration of volatile inhalant anesthetics. The decreased requirement for inhalants, coupled with reduced metabolism, can increase the risk for anesthetic overdose.

Acidemia
Poor tissue perfusion, from vasoconstriction, decreased CO, and shifting of the oxygen dissociation curve, can result in anaerobic metabolism and production of

lactic acid. With rewarming, lactic acid from poorly perfused areas mixes with core blood, causing systemic metabolic acidosis.

Prevention
Prevention of perioperative hypothermia is easier than treatment. Since the skin is the major source of heat loss during anesthesia, heat loss can be decreased by 30% simply by covering the skin (12). Warm ambient temperature can help maintain normothermia but can cause discomfort for hospital staff.

Treatment
Treatment of hypothermia should be directed at preventing further heat loss as well as providing active warming. Prevention of heat loss could include covering of nonessential exposed skin, administration of warmed fluids, and exposure to a warm environment when possible. Skin covering can be accomplished with blankets, plastic, metallic/reflective sheets, or a combination of these items. Heat and humidity exchangers can be used in the anesthetic circuit to prevent the body from having to warm and humidify cold, dry anesthetic gases.

Active warming increases the total heat content by the net transfer of heat to the body via external heating source. Such heating sources can include forced warm-air blankets (Bair Hugger®), electrical heating blankets, circulating warm water blankets, and heat lamps. Circulating warm-water blankets are more effective when placed over the patient. Caution should be used with any heating source to preclude burning and dehydration.

The transfer of body heat to cold IV fluids can contribute to loss of core body temperature. The amount of heat loss is dependent on the temperature and the amount of fluid infused. Thus, warming IV fluids can minimize heat loss. In cases of extreme hypothermia, peritoneal and pleural irrigation with warm saline can effectively change core body temperature. Cardiopulmonary bypass has been used successfully in humans as a means to actively rewarm a patient with excellent long-term outcomes.

Monitoring
Although hypothermia is common perioperatively, animals can also become hyperthermic. Long-haired breeds or animals with thick undercoats can become hyperthermic when positioned with external heating sources, covered in drapes, and under warm lights. Careful monitoring of all anesthetized patients will identify animals that need more or less thermal support.

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