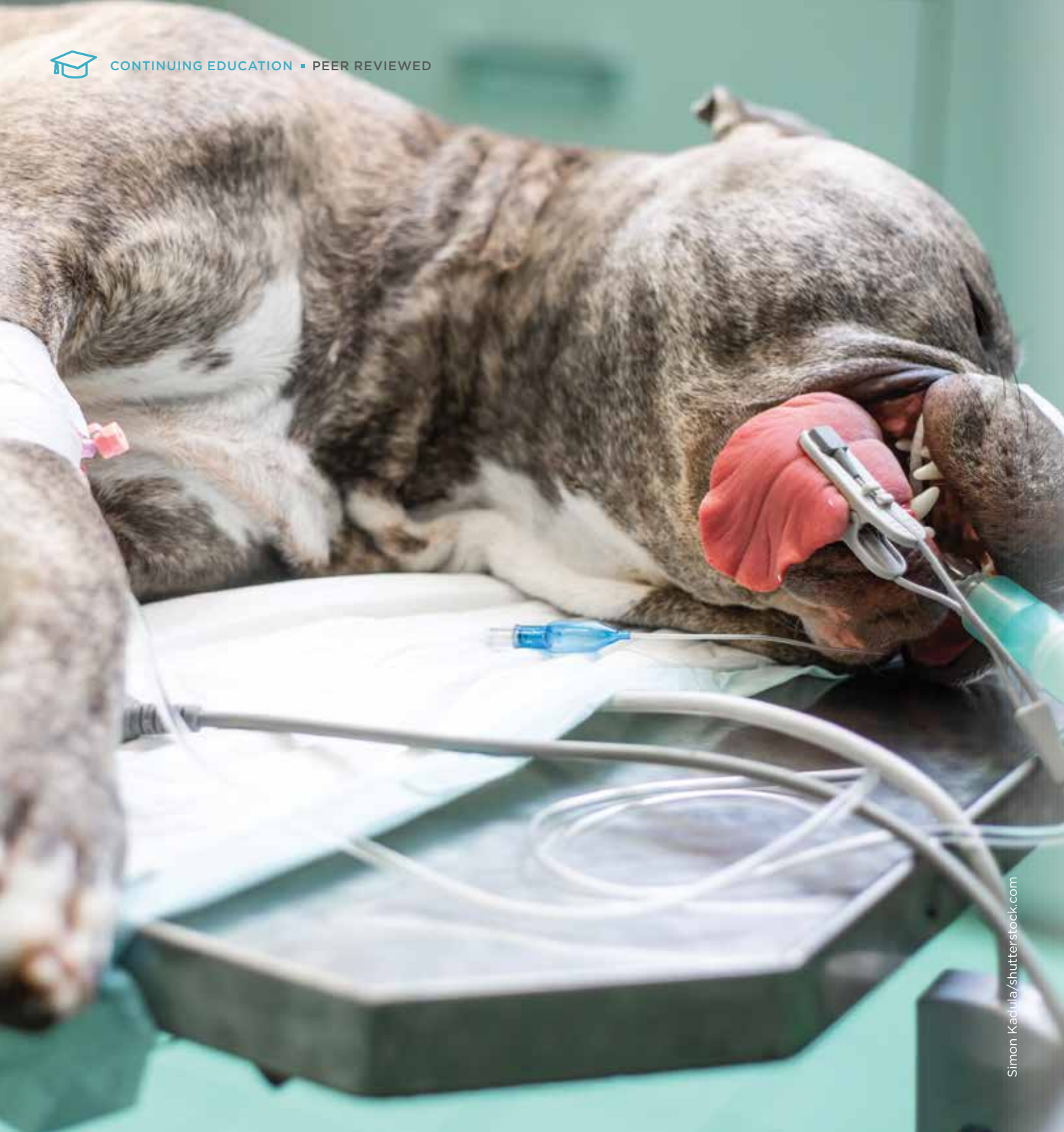




Abstract

Anesthetic complications can rapidly become emergencies if not identified and rectified. The most effective way to deal with anesthetic complications is to prevent them through (1) appropriate patient stabilization; (2) anesthetic and analgesic drug and dosage selection; (3) anesthetic equipment preparation; (4) pre-, post-, and intraoperative patient support; and (5) physiologic monitoring. This article provides an overview of the most common and/or critical anesthesia-related complications, with tips for complication identification, prevention, and treatment or correction.

CONTINUING EDUCATION

ANESTHESIA/ANALGESIA

How to Identify, Prevent, and Treat or Correct Anesthetic Complications

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Many anesthetic drugs can impair the physiologic function of vital organ systems, potentially leading to anesthesia-related complications. Patient factors, including extremes of age (i.e., neonatal, geriatric), presence of disease, and extremes of body weight and size, can also contribute to complications during anesthesia, as can duration of anesthesia.¹ Surgical procedure, approach, and invasiveness, along with patient positioning, can exacerbate

some anesthesia-related complications. Although most complications generally have minimal impact on the patient when recognized and corrected early, the ultimate complication is anesthesia-related death, the rate of which is higher in dogs and cats than in humans.¹

The most effective way to deal with anesthetic complications is to prevent them through appropriate patient stabilization based on the

Take-Home Points

- The most effective way to deal with anesthetic complications is to prevent them.
- The most effective anesthesia monitor is a trained veterinary professional (veterinary nurse or veterinarian) who can respond to abnormal physiologic changes before they become complications.
- Excessive anesthetic depth is a somewhat common, but often unrecognized, complication that can cause or contribute to most other complications, including hypotension and hypoventilation.
- Analgesia and appropriate premedication are critical components of an optimal anesthetic event.
- The most common/critical complications generally involve the central nervous, cardiovascular, and respiratory systems; thus, physiologic monitoring and support are generally focused on those systems.
- Supporting the central nervous, respiratory, and cardiovascular systems supports other organ systems (e.g., renal, hepatic) by promoting adequate tissue oxygen delivery.
- The potential for anesthetic complications does not end when the vaporizer is turned off. Most unexpected anesthetic deaths occur in the recovery phase of anesthesia. Vigilant monitoring in the recovery period is critical for anesthetic safety and should continue until the patient is awake and responsive with normal physiologic parameters.



patient's American Society of Anesthesiologists status; anesthetic and analgesic drug and dosage selection (**BOX 1**); anesthetic equipment preparation; pre-, post-, and intraoperative patient support; and physiologic monitoring.⁴

Complications involving the central nervous system (CNS), respiratory system, and cardiovascular system are among the most common anesthetic complications and are generally the most immediately life-threatening. Thus, physiologic monitoring and support are focused on these systems. However, anesthesia can directly or secondarily affect all organ systems, primarily through decreased oxygen delivery. Supporting the CNS, respiratory system, and cardiovascular system supports other organ systems (e.g., renal, hepatic) by ensuring adequate oxygen delivery. Physiologic monitoring greatly improves anesthetic safety, and the best monitor is a trained

veterinary nurse who watches the patient and responds to concerning physiologic trends—often before they become complications. Vigilant monitoring in the recovery period is critical for anesthetic safety and should continue until the patient is awake and responsive with normal physiologic parameters. (**BOX 2**).

CENTRAL NERVOUS SYSTEM COMPLICATIONS

Excessive Anesthetic Depth

Excessive anesthetic depth is a dangerous complication that is often not recognized until it results in other complications, primarily hypotension and hypoventilation. Excessive anesthetic depth during the maintenance phase of anesthesia can also contribute to a prolonged anesthesia recovery.

Causes of excessive anesthetic depth include absolute overdose of anesthetic drugs, but relative overdose can also occur. Inhalant anesthesia dose, defined roughly as the minimum alveolar concentration (MAC) required

BOX 1 Tips for Choosing and Using Analgesic and Sedative Drugs

Appropriate use of analgesic and sedative drugs tends to decrease the necessary dose of induction and inhalant drugs, which improves anesthetic safety since most adverse effects of anesthetic drugs are dose dependent.^{2,3} Depending on the degree and source of pain, analgesic considerations should include opioids, local anesthetic drugs, and nonsteroidal anti-inflammatory drugs, as well as options such as ketamine infusions.^{4,5} Sedatives to consider include α_2 agonists, acepromazine, and the benzodiazepines.⁴ Preanesthetic anxiolytics (e.g., gabapentin, trazodone) should be considered in patients with high fear/anxiety/stress (FAS), as decreased FAS also generally decreases the required dose of sedative drugs and, potentially, induction and inhalant drugs.⁴

To decrease the likelihood of both sudden arousal and the tendency to lean toward excessive anesthetic depth, administer preanesthetic sedatives and provide robust analgesia. This allows a more stable plane of anesthesia at a lower inhalant mean alveolar concentration.³ It is also recommended to have a dose of an injectable induction drug available so that if the patient undergoes arousal, an appropriate plane of anesthesia can be regained more rapidly and safely instead of “bagging down” with inhalants until the patient stops moving.

BOX 2 Avoiding Complications in Recovery

Anesthetic complications are often categorized by organ system, but their timing should also be considered. There are 4 separate but continuous and equally important phases of anesthesia: preanesthesia, induction, maintenance, and recovery. Recovery is often forgotten, and yet many anesthetic complications, including most unexpected anesthetic deaths, occur in recovery.¹ Patients that have an American Society of Anesthesiologists (ASA) status of III or higher and patients of any ASA status that had complications during anesthesia—even if they seemed minor—likely need monitoring and support into the recovery period.

Along with any of the physiologic concerns discussed in this article, common recovery complications include “rough” recoveries. Any patient vocalizing or with uncoordinated movement (thrashing) for more than 1 to 2 minutes (less, if violent) should receive sedatives, analgesics, or, most commonly, both to prevent self-injury. Pain is often a major contributor.

to keep a patient anesthetized, is lower in neonates, geriatric patients, and many compromised patients (e.g., patients with anemia, hypercarbia, hypoxia) than in middle-aged, healthy patients.³ Thus, the inhalant dose (and often the dose of other anesthetic drugs) should be decreased in these patients. Hypothermia also decreases the MAC, potentially by as much as 4% to 5% with every decrease in degree Celsius.³

Inadequate Anesthetic Depth

Inadequate anesthetic depth and/or inadequate analgesia to support the level of anesthetic depth is less common but can result in sudden arousal. This complication can also lead to detrimental outcomes, and the anesthetist should always be prepared with an injectable anesthetic drug and intravenous access to quickly reanesthetize an animal if it is aroused to the point of moving or injuring itself and/or personnel. Additional analgesia (e.g., opioid bolus, local anesthetic block, infusion of analgesic drugs) should be added if initial analgesia is deemed inadequate to prevent arousal.

Prevention of Central Nervous System Complications

- Anticipate patients that might need lower drug dosages and stabilize those with underlying disease if possible.
- Administer preoperative sedatives and analgesic drugs to decrease the risk of sudden arousal.
- Calculate drug dosages carefully. If a patient is overconditioned (body condition score > 5/9), calculate dosages based on lean body weight.
- Continually assess anesthetic depth using parameters such as response to noxious stimulation (i.e., pain), eye position, jaw tone, respiratory rate and rhythm, heart rate and rhythm, and arterial blood pressure, and challenge the anesthetic plane by decreasing the inhalant dose (i.e., “turning down the vaporizer”) and reassessing the patient’s responses.
- Treat each patient as an individual rather than maintaining the same inhalant dose for all patients.
- Record the inhalant concentration (or infusion drug dose) on the anesthetic record to ensure that the anesthetist is continuously aware of the drug dose being delivered to the patient. An example of this anesthetic record form can be viewed and downloaded for use at go.navc.com/3u3yv0y.

Treatment/Correction of Central Nervous System Complications

- If the patient is slightly to moderately deep, turn down the vaporizer and give the patient a few breaths to eliminate some of the inhalant. Increasing the flow of oxygen through the flowmeter will result in a more rapid change in anesthetic depth.
- If the patient is profoundly deep, it may be necessary to turn the vaporizer completely off, fill the rebreathing bag with oxygen using the flowmeter, and manually ventilate the patient. Administer breaths at a normal rate and depth. Overventilation can cause respiratory alkalosis.
- If the patient is too light, increase the vaporizer setting and, if needed, administer a dose of injectable induction drug for a more rapid response. Once the patient is at an appropriate plane of anesthesia, assess pain management. Addition of analgesia may be more appropriate, effective, and safe than maintaining the patient at an increased dose of anesthetic drug.

RESPIRATORY SYSTEM COMPLICATIONS

Hypoventilation

Hypoventilation, defined as inadequate gas exchange (i.e., removal of CO₂/uptake of O₂), is a common complication that often occurs secondary to excessive anesthetic depth. Hypoventilation due to inadequate respiratory rate and/or tidal volume (the volume of air entering the lungs during inhalation) can cause hypercarbia (which may lead to respiratory acidosis) and potentially hypoxemia (which can lead to decreased tissue oxygen delivery).

Causes of hypoventilation include anesthetic drugs, physical or physiologic abnormalities or changes in the patient, and equipment malfunction.

Almost all anesthetic drugs can cause some degree of respiratory compromise, and the degree of respiratory depression is largely dose dependent. Propofol, alfaxalone, and all of the inhalant drugs can cause dose-dependent respiratory depression, whereas ketamine is unlikely to cause this complication.

Physical or physiologic abnormalities or changes that can cause hypoventilation include those that impair the respiratory drive initiated in the CNS or gas exchange in the airways. Diseases that cause metabolic alkalosis or directly affect the CNS (e.g., primary CNS disease,

cranial trauma, brain tumors) can impair the respiratory drive. Gas exchange may be impaired by upper or middle airway conditions that impede airflow to the lungs (e.g., laryngeal dysfunction, tracheal collapse) or lower airway diseases (e.g., pneumonia) and physiologic changes such as ventilation/perfusion (V/Q) mismatch (e.g., pulmonary consolidation, tumors). Other factors such as obesity, pregnancy, thoracic trauma, and patient positioning can impede ventilation by impairing normal thoracic and/or diaphragmatic movement(s).

Equipment malfunction that impairs gas exchange can involve any component from the endotracheal (ET) tube to the oxygen supply. Common equipment complications include kinked, plugged, malpositioned, or excessively long ET tubes; inadequate fresh gas flow; malfunctioning inspiratory/expiratory valves in a rebreathing system; inadequate oxygen flow in a nonrebreathing system; and exhausted CO₂ absorbent.

A closed pop-off valve is a crisis complication, as the breathing system—and the patient's respiratory system—will rapidly become pressurized, causing collapse of the intrathoracic blood vessels and resulting in inadequate oxygen delivery to the myocardium and subsequent cardiac arrest. Adding a pop-off button just behind the pop-off valve can help avoid this complication (**FIGURE 1**).

Hypercarbia

In conscious patients, the normal values for arterial



FIGURE 1. Pop-off button placed between the pop-off valve and the scavenging system. When pressed, the button closes the system, allowing a positive-pressure breath to be administered by the anesthetist. When the button is no longer pressed, it automatically opens; thus, the system is never closed for longer than it takes to give a breath.

partial pressure of carbon dioxide (Paco₂) and end-tidal carbon dioxide (ETCO₂) are 35 to 45 mm Hg.

However, mild respiratory depression under anesthesia is acceptable and values up to 55 mm Hg are tolerable in most patients. Allowing CO₂ to increase above this limit usually results in respiratory acidosis.

The main cause of hypercarbia is hypoventilation. Other causes include failure to eliminate CO₂ due to exhausted CO₂ absorbent (rebreathing system) or inadequate oxygen flow (nonrebreathing system) and excessive production of CO₂, as can occur with moderate to profound hyperthermia or hypermetabolic states such as malignant hyperthermia. Failure to eliminate CO₂ from the system results in “rebreathing” of CO₂, which can be recognized on a capnograph curve or wave by a high (>45 mm Hg) peak and failure to return to 0 baseline (**FIGURES 2 AND 3**). All other causes of hypercarbia result in a high peak with a 0 baseline in the inspiratory pause. Hyperventilation would result in a curve with a low peak (<35 mm Hg).

Hypoxemia

The ideal arterial partial pressure of oxygen (Pao₂) as measured by arterial blood gas is 5 times the fraction of

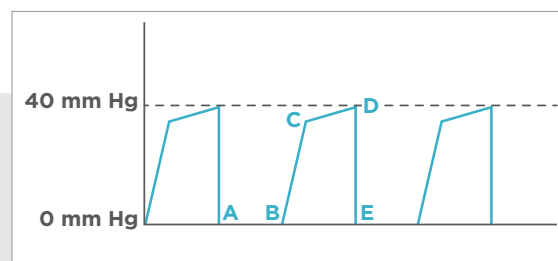


FIGURE 2. Normal capnograph waveform. **(A–B)** Zero baseline representing the respiratory “pause” between breaths. **(B–C)** Rapid, sharp rise reflecting the exhalation of mixed dead space and alveolar gas. **(C–D)** Alveolar plateau representing exhalation of mostly alveolar gas. **(D)** End-tidal CO₂ value at end of exhalation. **(D–E)** Rapid, sharp downstroke representing inhalation.

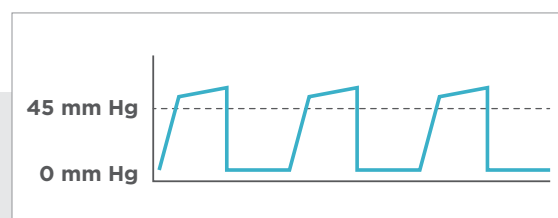


FIGURE 3. Capnograph waveform indicating rebreathing of CO₂. The waveform baseline stays elevated and does not return to 0.

inspired oxygen (F_{iO_2}). Therefore, the P_{aO_2} of a patient breathing room air (21% oxygen) would be roughly 105 mm Hg and that of a patient breathing 100% oxygen from the anesthesia machine would be roughly 500 mm Hg. Although values are rarely this high, the patient is not considered hypoxemic until the P_{aO_2} with room air decreases below approximately 80 mm Hg (absolute hypoxemia = $P_{aO_2} < 60$ mm Hg) and below 200 to 300 mm Hg on 100% oxygen (“relative” hypoxemia, but not truly hypoxemic).

Hypoxemia is defined as oxygen saturation (SpO_2) $< 90\%$ in all patients, but SpO_2 should be maintained at 95% to 100% if the patient is breathing 100% oxygen (**FIGURE 4**).

Hypoventilation is the primary cause of hypoxemia. Others include:

- Low F_{iO_2} , which could mean that the patient is breathing room air but needs supplemental oxygen, that oxygen is not flowing through the system, or that the nitrous oxide or air to oxygen ratio is too high (if nitrous oxide or air/oxygen mix is being used)
- Physiologic changes that cause V/Q mismatch
- Impaired diffusion of oxygen across the alveolar/arterial membrane

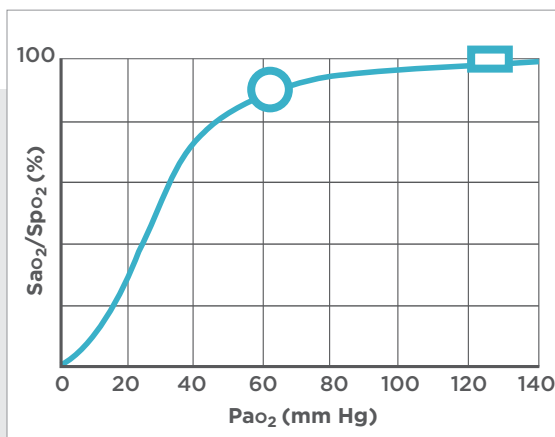


FIGURE 4. Oxygen-hemoglobin dissociation curve, used in pulse oximetry. The **circle** represents a hypoventilating patient breathing room air. The steep slope of the curve shows that continued hypoventilation would cause rapid oxygen desaturation. The **rectangle** represents a hypoventilating patient breathing 100% oxygen. This patient will remain $> 95\%$ saturated even with decreasing P_{aO_2} until the P_{aO_2} reaches the steep slope of the curve. Although this patient is not technically hypoxemic, it will likely be hypercarbic. P_{aO_2} = arterial partial pressure of oxygen; SaO_2 = arterial blood oxygen saturation; SpO_2 = oxygen saturation, pulse oximeter

- Anatomical right-to-left shunting of blood (e.g., from a reversed patent ductus arteriosus)

Hyperventilation

Hyperventilation (P_{aCO_2} or $ETCO_2 < 25$ to 30 mm Hg) in patients under anesthesia is generally due to an underlying cause such as pain or inadequate anesthetic depth. However, excessive anesthetic depth with subsequent increased CO_2 can also cause hyperventilation in its initial phases; therefore, anesthetic drugs should not be automatically administered to hyperventilating patients.

Hyperventilation can lead to respiratory alkalosis.

Iatrogenic causes of hyperventilation include aggressive positive-pressure ventilation, which can be avoided by the use of capnography. Treatment/correction of the underlying cause is required.

Prevention of Respiratory System Complications

- Stabilize the patient. Stabilization could be acute (e.g., placing a patient in respiratory distress in an oxygen cage) or chronic (e.g., providing treatment, allowing time for pneumonia to resolve).
- Anticipate patients that may be or become hypoxemic.
- Preoxygenate the patient. Three minutes of breathing 100% oxygen increases the time to desaturation ($SpO_2 < 90\%$) from 1 minute (room air) to 5 minutes.⁶
- Monitor and support the following:
 - Respiratory rates (e.g., 5 to 10 breaths/min in cats and dogs, size dependent); supplement as necessary to maintain normal $ETCO_2$ and SpO_2
 - Tidal volume (10 to 15 mL/kg body weight), not often measured unless advanced equipment is available. Tidal volume can be very roughly assessed by watching the patient’s thoracic expansion during inhalation.
 - Mucous membrane color (pink versus “dusky”; late sign of hypoxemia if dusky)
 - SpO_2 ($> 90\%$ room air; $> 95\%$ on 100% oxygen)
 - $ETCO_2$ (35 to 55 mm Hg)
 - Arterial blood gas, if needed and available
- Triple-check that all anesthesia equipment is functioning normally.

Treatment/Correction of Respiratory System Complications

Treatment and/or correction of respiratory complications depends on the cause. Generally, ensuring an appropriate anesthetic depth and



Practice Case: Hypercarbia During Surgery

During a routine ovariohysterectomy in a healthy 1-year-old, 10-kg (22-lb) female mixed-breed dog, you notice the patient's end-tidal CO₂ (ETco₂) is 70 mm Hg and its breathing is rapid and very shallow, as evidenced by minimal movement of the thorax during inhalation/exhalation. What do you do?

1. Quickly give the patient a breath by holding down the pop-off button and squeezing the rebreathing bag. As the breath is delivered, "feel" for resistance in the system (i.e., assess how easy it is to administer a breath to the patient) while watching the machine and the thoracic excursion of the patient to make sure that there are no obstructions or other causes of impaired flow.
2. Assess anesthetic depth, look at the vaporizer setting, and think about the drugs/dosages that the patient has

received for premedication and induction. Is the patient too deeply anesthetized? If so, decrease the vaporizer setting and continue to breathe for the patient.

3. Check for correct intubation and patency of tube. Reintubate if needed.
4. If the cause is not yet elucidated, check the equipment. Is the CO₂ absorber exhausted? Is the ETco₂ monitor functioning normally? Electronic monitors sometimes give false information. If equipment issues are suspected, change to a different monitor and/or fix the equipment problems on the anesthetic machine you are using or switch to a different machine.
5. Finally, is there a cause of increased CO₂ production rather than decreased CO₂ elimination? Uncommon

anesthetic emergencies like hyperthermia (and anything that causes hypermetabolism) can cause an increase in CO₂ production that may overwhelm the patient's ability to eliminate CO₂.

If no cause can be found, this patient might require ventilator assistance throughout the anesthetic episode. This is uncommon in young, healthy patients but very common in aged or compromised patients. Ventilator assistance does not necessarily mean that a mechanical ventilator is required, since a person can easily ventilate the patient. Ventilator assistance also does not necessarily mean that total control of ventilation is necessary. Many patients can breathe on their own but need some support—often 1 to 2 additional breaths per minute or even every 3 to 5 minutes will suffice. This is patient dependent, and appropriate support will be determined by monitoring.

supporting ventilation will correct hypoventilation, hypercarbia, and hypoxemia. In the case of primary hypercarbia, check equipment function and body temperature. In the case of primary hypoxemia, ensure that the patient is intubated correctly (i.e., ET tube in airway with aboral tip in the trachea and not in a bronchus) and that oxygen is flowing through the machine and through the breathing system that is connected to the patient. A deep sigh or recruitment maneuver may be necessary to re-expand atelectatic alveoli. Repositioning patients may relieve impairment of thoracic and/or diaphragmatic movement.

Other respiratory complications include a kinked or occluded ET tube, aspiration pneumonia, and inability to intubate, the last of which may require advanced techniques such as a temporary tracheostomy.

CARDIOVASCULAR SYSTEM COMPLICATIONS

The most common cardiovascular complications are presented below. Others include the most serious

complication, cardiac arrest. Cardiopulmonary resuscitation (CPR) is covered in detail elsewhere (RECOVER [Reassessment Campaign on Veterinary Resuscitation] Guidelines),⁷ and CPR training for the anesthesia team is recommended.

Hypotension

Hypotension (mean arterial pressure [MAP] <60 mm Hg; systolic arterial pressure [SAP] <90 mm Hg if monitoring with Doppler ultrasonography) is a common effect of many anesthetic drugs and can be exacerbated by comorbidities. As with hypoventilation, hypotension is often secondary to excessive anesthetic depth. Hypotension leads to decreased blood flow (and therefore decreased oxygen delivery) to the tissues. However, because blood flow is more difficult to measure than blood pressure, blood pressure is used as a surrogate metric for monitoring.

Causes

Factors that affect any part of the cardiovascular

system—the heart, blood vessels, and circulating blood and plasma—can cause hypotension. Such factors include anesthetic drugs and physical or physiologic issues in the patient.

Many anesthetic drugs can cause some degree of cardiovascular depression or dysfunction by affecting both heart rate and stroke volume. The degree of cardiovascular depression is dose dependent for most drugs, and overdose of an anesthetic drug is a common cause of cardiovascular depression. Propofol, alfaxalone, and the inhalant drugs can cause dose-dependent hypotension, whereas ketamine is less likely to cause this complication.

Physical or physiologic abnormalities or changes in the patient that cause hypotension include anything that impairs pump function, vascular tone, or circulating volume.

- **Pump function** (heart rate and myocardial contractility) can be impaired by some anesthetic drugs, cardiac disease (e.g., hypertrophic cardiomyopathy, dilated cardiomyopathy, mitral insufficiency) and many systemic diseases (e.g., hypothyroidism).
- **Vascular tone** is affected by anesthetic drugs (especially inhalant drugs and acepromazine); some cardiac medications; and systemic diseases such as septicemia, which often cause vasodilation. Profound vasodilation results in decreased circulating fluid volume.
- **Circulating volume** is affected by any form of intravascular fluid loss (e.g., dehydration, hemorrhage, “third-spacing” of fluid, evaporation of fluid from open body cavities and the respiratory tract).

BOX 3 Normal Heart Rates

These ranges can be used as guidelines; rates outside of these limits are common and are acceptable if they are not mediated by an underlying cause that should be corrected and not affecting blood pressure.

- **Large dogs:** 50 to 60 beats/min
- **Medium dogs:** 60 to 80 beats/min
- **Small dogs:** 80 to 100 beats/min
- **Cats and extremely small dogs:** 100 to 150 beats/min

Prevention

- Stabilize the patient. Human patients who are hypotensive prior to anesthesia are more likely to be hypotensive during anesthesia.⁸ Because of similar mammalian physiology, the same is expected for dogs and cats.
- Anticipate patients that may be or become hypotensive (e.g., patients with cardiovascular disease) and make a preanesthesia plan for treatment.
- Monitor and support:
 - Heart rate: “Normal” heart rate should be defined as normal for the size of the patient when it is relaxed (rather than excited or scared, as is common in patients being examined at the veterinary clinic) (**BOX 3**).
 - Mucous membrane color (pink versus pale), capillary refill time (<2 sec), and pulse quality/strength
 - Arterial blood pressure: MAP > 60 mm Hg, SAP > 90 mm Hg if using Doppler ultrasonography
 - SpO₂: > 90% (patient breathing room air) to 95% (patient breathing 100% oxygen)

Treatment/Correction

Exact treatment options depend on the patient and cause of hypotension. **FIGURE 5** provides a general protocol:

- Decrease anesthetic depth.
- Increase fluid rate or give a bolus of crystalloids.
- Correct the heart rate (increase or decrease).
- Continue fluid boluses or increased fluid rate if hypovolemia is the expected cause. Colloids may be necessary. Use blood products if hemorrhage is causing the hypovolemia.
- Administer positive inotropic drugs (e.g., dopamine, dobutamine; the dose of both is roughly 1 to 10 µg/kg/min) if decreased myocardial contractility is the expected cause.
- Consider vasopressors (e.g., vasopressin, norepinephrine, phenylephrine) if excessive vasodilation is the expected cause.

Arrhythmias

Although patients may present for anesthesia with a variety of preexisting arrhythmias, anesthesia itself is primarily associated with bradycardia, tachycardia, and ventricular premature contractions (VPCs).

Arrhythmias are concerning not only because they could be a result of organic cardiac disease but also because they can contribute to hypotension and decreased organ perfusion and can potentially be fatal.

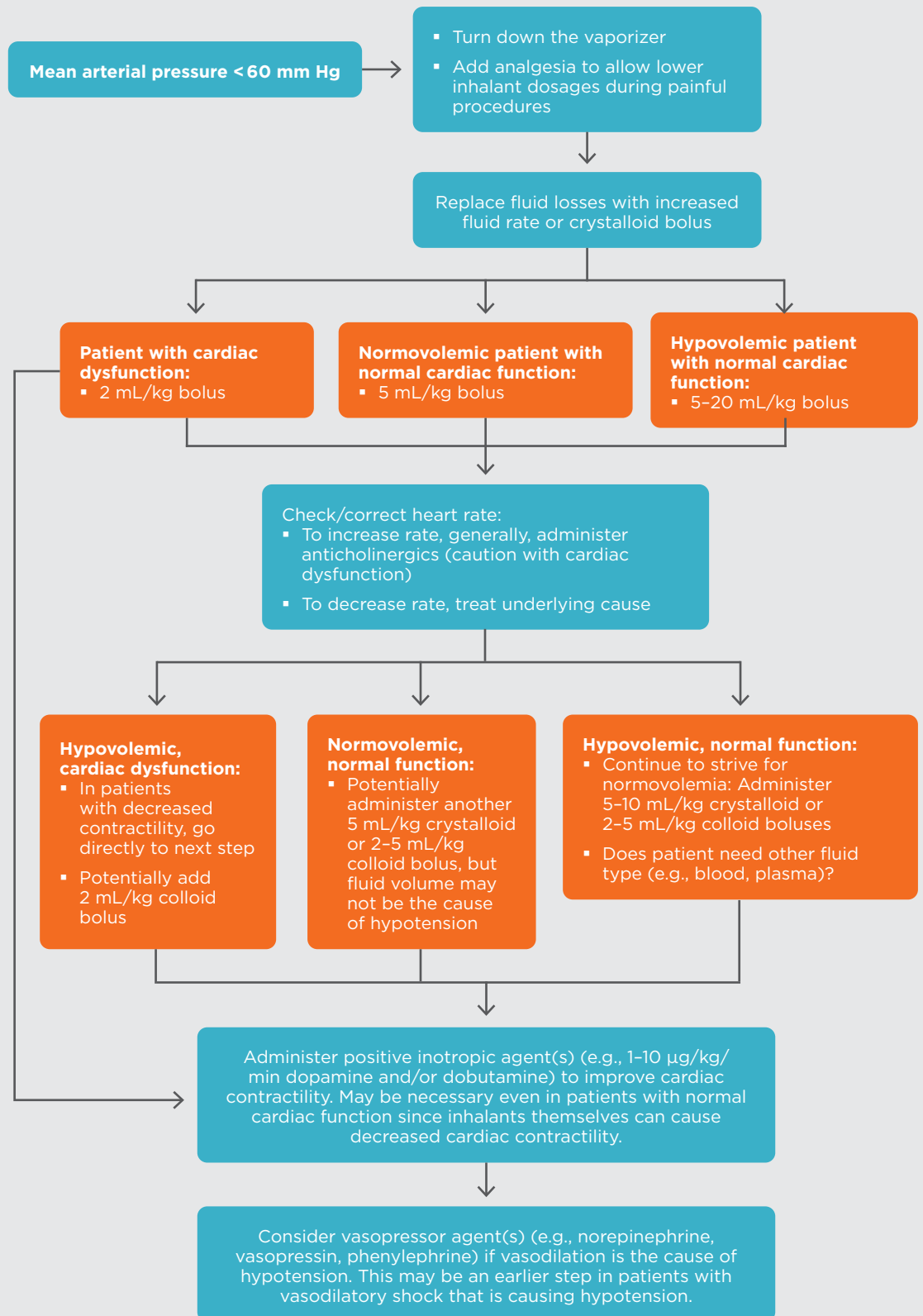


FIGURE 5. Follow the algorithm through each step of treatment until hypotension is resolved. Exact treatment options depend on the patient and cause of hypotension. Note that fluid bolus volumes are for dogs; use smaller volumes in cats (e.g., 1-3 mL) since cats have a smaller blood volume compared to dogs.



Bradycardia may be caused by some anesthetic drugs (e.g., α_2 agonists, opioids, propofol) or by surgeries/conditions that enhance vagal tone (e.g., ocular, laryngeal, gastrointestinal, urinary bladder procedures). Lower heart rates are expected in very athletic patients and large-breed dogs (**BOX 3**).

Tachycardia is usually secondary to an underlying condition. Therefore, treatment of the underlying condition (rather than treatment of the heart rate itself) is usually the correct approach. Examples of underlying conditions include pain, an inadequate plane of anesthesia, an excessive plane of anesthesia with subsequent hypotension, high CO_2 , cardiac disease, and systemic conditions such as hyperthyroidism and septicemia. Higher heart rates are expected in cats and toy-breed dogs (**BOX 3**).

VPCs can be caused or exacerbated by preexisting myocardial disease; by some anesthetic drugs; and by physiologic abnormalities such as pain, hypoxia, hypercarbia, acidosis, and electrolyte abnormalities.

Prevention

- Stabilize the patient by treating any preexisting arrhythmias.
- Monitor the electrocardiogram and correct conditions expected to cause arrhythmias.

Treatment

Bradycardia is generally vagally mediated and can be treated with anticholinergics (e.g., 0.02 to 0.04 mg/kg atropine, 0.01 mg/kg glycopyrrolate).

- Bradycardia that is unresponsive to anticholinergics may require treatment with catecholamines such as dopamine (1 to 10 mg/kg/min), epinephrine (0.1 to 1 $\mu\text{g/kg/min}$), or norepinephrine (0.05 to 1 $\mu\text{g/kg/min}$).
- Occasionally, bradycardia may be unresponsive due to cardiac disease, toxemia, profound hypothermia, profound hypoxia, or a variety of systemic diseases.
- Bradycardia should be treated any time the low heart rate is contributing to *low* blood pressure. However, α_2 agonists cause low heart rate with *high* blood pressure, and using anticholinergics in these patients will unnecessarily increase cardiac work.
 - During inhalant anesthesia, the vasoconstrictive effects of α_2 agonists often dissipate without a reflex increase in heart rate, which may result in low blood pressure. In such hypotensive patients, the effects of the α_2 agonists could be

antagonized to increase heart rate, but the author's preferred treatment is to administer an anticholinergic (e.g., atropine, glycopyrrolate), so that the sedative effects of the α_2 agonists continue to aid in maintaining a stable plane of anesthesia.

- The “rule” that anticholinergics should not be administered with α_2 agonists only applies if blood pressure is normal or high.

Tachycardia is primarily treated by eliminating the underlying cause. Occasionally, β -blockers are administered to decrease tachycardia from uncommon, uncontrollable causes such as pheochromocytoma.

The first line of treatment for VPCs is to eliminate underlying causes (e.g., administer analgesics, treat electrolyte imbalances, improve oxygenation). A low number of VPCs may be expected (e.g., patients with some gastrointestinal issues like gastric dilatation-volvulus, geriatric patients) and may not require treatment. Treatment should be initiated if the arrhythmia is life-threatening or affects the blood pressure or if the number of VPCs is >20% of the total number of ventricular beats.

- Lidocaine is generally the first choice for treatment of VPCs in dogs. The dose is 2 mg/kg IV (maximum 8 mg/kg during any 60-minute period) followed by a 25 to 75 $\mu\text{g/kg/min}$ infusion. Lidocaine in cats is also effective for treatment of VPCs but is used more cautiously and at lower doses, generally 0.2 to 0.5 mg/kg IV as an initial, slow bolus followed by a 10 $\mu\text{g/kg/min}$ infusion if needed.
- The procainamide dose in dogs is a 10 mg/kg IV bolus (range, 8 to 20 mg/kg IV) followed by a 25 to 50 $\mu\text{g/kg/min}$ infusion. The dose in cats is a 1 to 2 mg/kg IV *slow* bolus followed by a 10 to 20 $\mu\text{g/kg/min}$ infusion. Procainamide can cause profound negative inotropic effects and should not be administered to patients with impaired contractility. Arterial blood pressure should be monitored during the administration of procainamide.

THERMOREGULATORY COMPLICATIONS

Normal body temperature for dogs and cats is approximately 38.1 °C to 39.2 °C (100.5 °F to 102.5 °F). Body temperature should be kept as close to normal as possible, but temperatures down to 36.6 °C (98 °F) and up to 39.7 °C (103.4 °F) can be tolerated by most patients. Some patients may have



Small patients suffer the worst heat loss; therefore, body temperature support should be “aggressive” in these patients. Forced-air blankets tend to be most effective.

identification microchips that measure body temperature. These can be used for postanesthetic monitoring instead of rectal temperature monitoring.

Hypothermia

Hypothermia is common during anesthesia and leads to a variety of other complications, including clotting dysfunction, immunosuppression, increased risk of infection, tissue hypoxia, acidosis, abnormal cardiac electrical conduction, and myocardial ischemia.⁹ Hypothermia also has cerebral effects that decrease the patient’s anesthetic needs. Unfortunately, this decreased need is not always recognized, resulting in an overdosage of anesthetic drugs.

Importantly, hypothermia is the main cause of prolonged recovery from anesthesia. In a study of anesthetized cats that received heat support (limbs and thorax wrapped with “bubble” wrap) or no heat support, the time from extubation to standing in recovery was 26.4 ± 5.8 mins versus 47.0 ± 5.8 mins, respectively.¹⁰ Although shivering in recovery may increase body temperature, the intense muscle movements associated with shivering cause discomfort and can double or triple oxygen consumption and CO₂ production.¹¹

Causes of hypothermia include anesthesia drug–induced vasodilation (common with inhalants), muscle relaxation, and decreased thermoregulatory control. Duration of anesthesia is a major contributor, and cold surgical tables, surgery rooms, scrub solution, and inhaled air (primarily with high-flow oxygen in nonrebreathing systems), along with excessive patient hair clipping and wetting with scrub solution, all contribute to hypothermia.

To prevent hypothermia, ensure the patient’s preanesthesia body temperature is normal. Warm the patient if necessary. The biggest drop in body temperature occurs right at/after induction as anesthetic drugs cause vasodilatory heat loss and the thermoregulatory center becomes less responsive to body temperature changes; therefore, almost all patients will need active warming during anesthesia, starting right at induction. Small patients suffer the worst heat loss; therefore, body temperature support should be “aggressive” in these patients. Forced-air blankets tend to be most effective.

Monitor body temperature throughout the procedure. Continuous monitoring is ideal, but intermittent monitoring is acceptable. Core temperature measured by an esophageal temperature probe is generally more accurate than rectal temperature (because feces are often present).

Hyperthermia

Hyperthermia is less common during anesthesia but can be caused by overvigorous warming or drug reactions (e.g., opioid-induced hyperthermia in cats) or as a component of any disease that causes pyrexia. Malignant hyperthermia is a very rare genetic condition.

To correct hyperthermia (temperature $> 40^{\circ}\text{C}$ [104°F]), remove all heating devices and start actively cooling the patient with fans or cool water. To avoid causing hypothermia, stop active cooling when the patient’s body temperature reaches 39.7°C (103.4°F). For cats receiving opioids, expect body temperature to increase (usually to $\leq 39.7^{\circ}\text{C}$ [103.4°F]) after opioid administration and schedule more frequent (at least 3) postanesthesia temperature checks. More than 1 temperature check should also be scheduled for any patient with an abnormal (high or low) body temperature in anesthesia recovery.

SUMMARY

The “best offense is a good defense” game plan is highly appropriate for anesthesia. By carefully monitoring anesthetized patients, trained veterinary staff can recognize negative trends in patient parameters and initiate appropriate changes to keep the patient from deteriorating. Changes in the CNS, respiratory system, and cardiovascular system are the most immediately life threatening; thus, physiologic monitoring and support are focused on these systems.

Anesthetists should be trained in anesthesia monitoring and patient support. **TVP**

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CONTINUING EDUCATION

How to Identify, Prevent, and Treat or Correct Anesthetic Complications

TOPIC OVERVIEW

Anesthetic complications should be prevented if possible and addressed immediately if they do occur. This article provides an overview of the most common and/or critical anesthesia-related complications, with tips for identification, prevention, and treatment/correction of those complications.

LEARNING OBJECTIVES

After reading this article, participants will be able to:

1. Identify the most common anesthesia-related complications
2. Implement tools/techniques to prevent complications
3. Develop treatment protocols to use if complications do occur

This article has been submitted for **RACE approval for 1 hour of continuing education credit** and will be opened for enrollment upon approval. To receive credit, take the test at vetfolio.com by searching the name of the article or scanning the QR code below. Free registration is required. Questions and answers online may differ from those below. Tests are valid for 2 years from the date of approval.



1. **The most effective way to deal with anesthetic complications is to prevent them by:**
 - a. Stabilizing the patient when necessary
 - b. Vigilant physiologic patient monitoring by a trained anesthetist
 - c. Pre-, intra-, and postoperative physiologic support of the patient
 - d. All of the above are important.
2. **All organ systems can be negatively affected by anesthesia.**
 - a. True
 - b. False
3. **Anesthesia equipment is not a source of anesthesia-related complications.**
 - a. True
 - b. False
4. **Which of the following statements is true with regard to excessive anesthetic depth?**
 - a. It is a complication on its own, and it can lead to other complications like hypotension.
 - b. It is not a cause for concern.
 - c. It is the only way to keep the patient anesthetized during a painful procedure.
 - d. It only occurs when too many drugs are administered (e.g., premedications).
5. **The most effective way to prevent the potential central nervous system complication of sudden patient arousal is to:**
 - a. Keep the inhalant dose very high (e.g., $\geq 4\%$ isoflurane).
 - b. Use a very high dose of propofol or alfaxalone for induction.
 - c. Administer preanesthetic sedatives and analgesic drugs.
 - d. This complication cannot be prevented.
6. **Which of the following can cause hypotension?**
 - a. Decreased myocardial contractility
 - b. Vasodilation
 - c. Inadequate circulating blood volume
 - d. All of the above
7. **Which statement is true regarding anesthesia-related arrhythmias?**
 - a. Anesthesia causes many different arrhythmias.
 - b. Bradycardia induced by α_2 agonists is very dangerous.
 - c. Tachycardia can be caused by pain, hypovolemia, and hypoxemia.
 - d. Ventricular premature contractions must always be treated if they occur.
8. **How can high end-tidal carbon dioxide (ETCO₂) caused by patient hypoventilation be differentiated from high ETCO₂ caused by failure to remove CO₂ from the anesthetic breathing system/machine with subsequent rebreathing of CO₂?**
 - a. Retained CO₂ causes an elevated baseline on the capnograph waveform.
 - b. Hypoventilation causes an elevated baseline on the capnograph waveform.
 - c. Retained CO₂ causes a negative baseline on the capnograph waveform.
 - d. There is no way to differentiate these causes of high ETCO₂.
9. **Which statement is true regarding profound hypothermia during anesthesia?**
 - a. It is not associated with adverse effects.
 - b. It can cause delayed recovery and immunosuppression.
 - c. It is only a concern during long surgeries.
 - d. It can lead to the need for a higher dose of anesthetic drugs.