PEER REVIEWED

ANESTHETIC MONITORING
Therapeutic Actions

Jeff Ko, DVM, MS, Diplomate ACVA, and Rebecca Krimins, DVM

This article is the last one in our ***Anesthetic Monitoring Series****, which has covered the goals and reasons for anesthetic monitoring, the physiologic parameters for anesthetized dogs and cats, and anesthetic monitoring equipment and its purpose.*

Read the first two articles in the series, ***Anesthetic Monitoring: Your Questions Answered*** *(January/February 2012) and* ***Anesthetic Monitoring: Devices to Use & What the Results Mean*** *(March/April 2012), at* ***todaysveterinarypractice.com****.*

When monitoring an anesthetized patient, two simple questions should always be asked:

1. Is the patient well oxygenated?
2. Is the patient well perfused?

An essential goal of monitoring is to prevent adverse events; it is much easier to prevent an adverse event than to treat one. Anesthetic monitoring provides an early warning of cardiorespiratory decompensation, allowing vital therapeutic intervention.

It is important to realize that monitoring anesthesia is not a therapeutic action. When an abnormality is detected in an anesthetized patient, correct interpretation is necessary in order to institute appropriate therapy. Then, comprehensive monitoring of the patient's progress is continued until the abnormality resolves. If the appropriate action occurs too late, anesthetic injury or fatality may occur.

This article addresses the recognition of specific abnormalities detected through anesthetic monitoring of dogs and cats as well as the therapeutic actions taken to correct them.

OVERVIEW OF MONITORING EQUIPMENT

Devices used to monitor anesthetized dogs and cats include, but are not limited to:

- Electrocardiography (ECG)
- Pulse oximetry
- Capnometry
- Direct and indirect arterial blood pressure monitors
- Blood gas analysis.

For a complete list of anesthetic monitoring equipment and its uses, see **Table 1** in **Anesthetic Monitoring: Devices to Use & What The Results Mean** (March/April 2012), available at **todaysveterinarypractice.com.**

ELECTROCARDIOGRAPHY

Definition of Abnormalities

Normal ECG morphology and conduction pathway includes the:

- **P wave**: Electrical activity is initiated by the sinoatrial (SA) node, signaling both atria to contract.
- **P–R interval**: Electrical impulses travel from the SA node to the atrioventricular node; because the stimulus of depolarization slows within the atrioventricular (AV) node, there is a pause.
- **QRS complex**: Electrical impulses travel from the bundle of His to the ventricles, causing ventricular depolarization.
- **T wave**: Ventricular repolarization.

ECG abnormalities are an irregularity in electrical impulse formation or electrical conduction; both may lead to various types of cardiac arrhythmias (**Figure,** page 30).[1]

- The normal electrical activity of the heart originates in the SA node (primary pacemaker), spreads through the atria to the AV node, travels to the bundle of His, and finally reaches the myocardial Purkinje fibers.

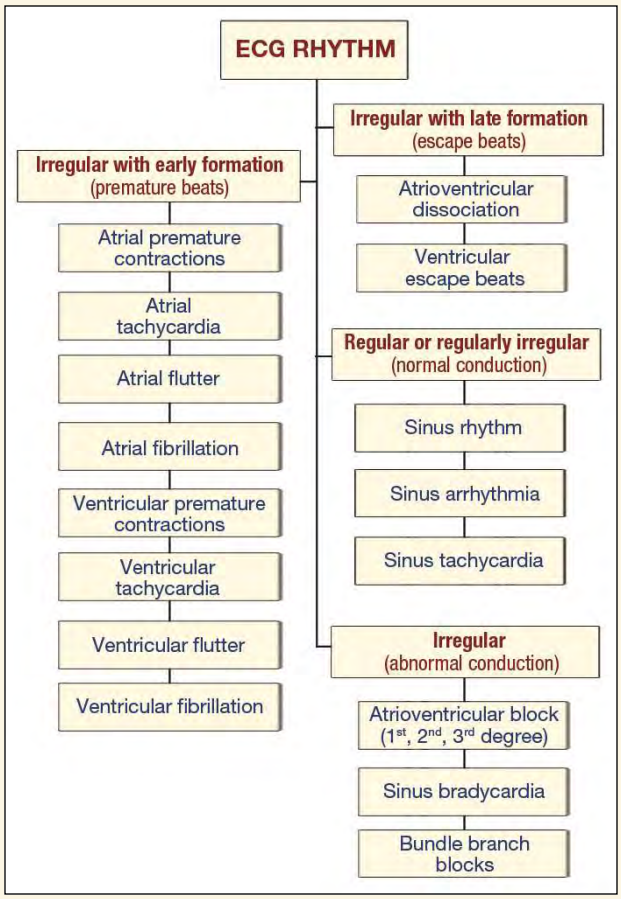

Figure. *Electrocardiographic diagnosis of cardiac arrhythmias based on electrical impulse formation and conduction*

- There are other subsidiary pacemakers besides the SA node, such as the AV node, Purkinje fibers, and other myocardial cells, particularly the ventricular tissue, which can initiate impulse formation and form escape or premature beats.

Recognition of Abnormalities

The following questions can help determine whether an abnormality is present on an ECG:

- What is the patient's heart rate? Is it too slow (bradycardia), normal (sinus rhythm), or too fast (tachycardia)?
- What is the patient's heart rhythm? Is it regular or irregular?
- When examining the electrocardiogram (either from a frozen screen image on the monitor or a printed rhythm strip), the anesthetist should address these additional questions:
  - » Is there an impulse formation or impulse conduction problem?
  - » Are P waves present and are they occurring regularly?
  - » Is there a P wave for each QRS complex and does each QRS complex have a corresponding P wave?
  - » Is the P–R interval consistent?
  - » Do the QRS complexes appear similar across the ECG strip?

Management of Abnormalities

Cardiac arrhythmias should be monitored closely and treated appropriately in order to prevent more serious forms of arrhythmias from occurring. A serious cardiac arrhythmia is considered any condition that affects the rate or rhythm to the point where blood pressure is affected.

The balance between heart rate and blood pressure is a key consideration when determining whether or not to treat a cardiac arrhythmia. The purpose of heart rate is to maintain blood pressure. Four possible scenarios regarding the relationship between heart rate and blood pressure are depicted in **Table 1**.

Following are possible arrhythmias that may or may not affect blood pressure under general anesthesia as well as their appropriate treatments.

Heart Blocks & Sinus Bradycardia

Causes

- Atrioventricular heart blocks of various degrees (first, second, or third degree) are frequently associated with sinus bradycardia, sinus arrhythmia, and increased vagal tone and can stem from various causes.
- Brachycephalic breeds and athletic dogs often have high vagal tone.
- Opioids (morphine, hydromorphone, fentanyl, and sometimes butorphanol) and alpha-2 agonists (xylazine, medetomidine, and dexmedetomidine) are associated with activation of vagal tone and reduced sympathetic outflow.

Treatment

- Anticholinergic drugs, such as atropine (0.02 mg/kg IV or 0.04 mg/kg IM) or glycopyrrolate (0.005 mg/kg IV or 0.01 mg/kg IM) can be administered to either prevent (IM) or treat (IV) bradycardia and heart blocks in the anesthetized animal.
- *Note that the use of anticholinergics to treat medetomidine- or dexmedetomidine-induced bradycardia is not recommended due to expectation of minimal improvement of cardiac output and the potential to induce even more serious arrhythmias.*
- Some dogs with third-degree heart block do not respond to atropine; a cardiac workup is recommended before performing general anesthesia on these patients. Placement of a temporary or permanent pacemaker may be required for safe anesthesia.

Ventricular Premature Contractions (VPCs)

Causes

- VPCs may be induced by high sympathetic tone associated with pain, stress, excitement, hypoxia, hypercapnia, electrolyte and acid-base disturbances, autonomic disturbances, and drugs.
- Anesthetic drugs, such as dissociatives (ketamine, tiletamine-zolazepam), thiobarbiturates, propofol, and halothane, are known to induce VPCs. Atropine and some inotropes can also induce VPCs.

Treatment

- If VPCs are frequent, multifocal, or occur in a series, they may affect blood pressure and treatment is necessary.
- Treatment is directed toward the specific cause of the VPCs.
- Severe runs of VPCs can be treated with:

Table 1. Four Scenarios: Balance Between Heart Rate & Blood Pressure in Maintaining Tissue Perfusion

Circulation Issue (Heart Rate)	**Perfusion Issue** (Blood Pressure)	**Potential Treatments**
High: > 180 bpm	**Low:** MBP < 60 mm Hg	• Reduce inhalant concentration • Administer fluids and inotropic agent • Provide 100% oxygen supplementation • Provide proper ventilation
Low: ≤ 50 bpm*	**High:** MBP > 90 mm Hg	• Ensure BP is maintained • Further treatment is not necessary if BP is maintained • If MBP > 100 mm Hg, consider evaluating and treating for increased intracranial pressure
Low: ≤ 50 bpm*	**Low:** MBP < 60 mm Hg SBP < 90 mmHg DBP < 65 mmHg	• Reduce inhalant concentration • Administer anticholinergic, inotropic, and fluids
High: > 180 bpm	**High:** MBP > 90 mm Hg	• If anesthetic plane is too light, increase inhalant concentration • Administer opioids or analgesics

**Heart rates that are low enough to affect BP are usually ≤ 50 bpm.*
BP = blood pressure; DBP = diastolic blood pressure; MBP = mean blood pressure; SBP = systolic blood pressure

- » 2 mg/kg of lidocaine IV given as a single bolus; this dose can be repeated up to a max of 8 mg/kg IV over a course of 5 to 10 minutes.
- » A constant rate of infusion of lidocaine between 25 to 80 mcg/kg/min may be used.

Atrial Premature Contractions (APC) & Bundle Branch Blocks (BBB)

- APCs and BBBs may not require treatment if the hemodynamic status of the patient (ie, blood pressure) is not compromised.
- Providing appropriate ventilation with 100% oxygen and appropriate depth of anesthesia are frequently sufficient to prevent further deterioration of APCs or BBBs under general anesthesia.

BLOOD PRESSURE MEASUREMENT

Definition of Abnormalities

Systolic Blood Pressure (90–140 mm Hg)

- Systolic blood pressure (BP) can be viewed as an indicator that summarizes cardiac contractility.[2]
- When systolic BP is lower than 90 mm Hg (some textbooks use 80 mm Hg as a cutoff for hypotension), myocardial depressive effects may be profound and cardiac output reduced.[3]
- Lack of venous return can reduce systolic BP.
- When systolic BP exceeds 140 mm Hg, the animal may be in an excessively light plane of anesthesia, and sympathetic tone and cardiac output may be high.

Diastolic Blood Pressure (65–90 mm Hg)

- Diastolic BP reflects peripheral vascular resistance.[2]
- When diastolic BP is lower than 60 to 65 mm Hg, it indicates vasodilation or hypovolemia.[2]
- When diastolic BP is higher than 90 mm Hg, it indicates peripheral vasoconstriction.[2]
- Profound increases or decreases in peripheral vascular resistance may reduce cardiac output.[2]

Mean Arterial Blood Pressure (70–90 mm Hg)

- Mean arterial BP should be at least 60 to 70 mm Hg to maintain adequate tissue and organ perfusion.[2]
- When mean arterial BP is less than 60 mm Hg, organ perfusion suffers.
- When mean arterial BP is greater than 90 mm Hg, the animal is usually in a lighter plane of anesthesia, unless the BP is increased due to other pharmacologic agents (ie, vasopressors or inotropes).

Recognition of Abnormalities

In a review study of 1281 dogs undergoing general anesthesia, 38% of dogs experienced episodes of hypotension.[3]

- Is the proper cuff size being used for monitoring BP? Improper cuff size may cause artifact-induced hypotension or hypertension.
- Determine if cardiac rhythm and rate are normal. The following can affect blood pressure:[2]
  - » Cardiogenic shock (heart failure, deep plane of anesthesia)
  - » Hypovolemic shock (acute blood loss, severe dehydration)
  - » Distributive shock (profound vasodilation due to anesthetics, maldistribution of blood flow due to sepsis)
  - » Obstructive shock (pericardial effusion and tamponade, large pleural effusions, and tension pneumothorax) can affect blood pressure.[3]
- Verify the appropriate depth of anesthesia by checking the patient and other vital signs.
- Verify that acid-base and electrolyte status are within normal limits.

Management of Abnormalities

- A combination of various actions to manage abnormalities includes:
  - » Reducing depth of anesthesia
  - » Treating with appropriate fluid administration
  - » Treating with vasopressor/inotrope administration.

Table 2. Inotropes & Vasopressors: Clinical Use in Dogs & Cats

Drug	Mechanisms	Pros/Cons	Side Effects	Dosage
Ephedrine (50 mg/mL)	• Stimulates alpha and beta receptors • Indirectly stimulates norepinephrine release	• Improves cardiac contractility and vascular resistance • Can be used as bolus injection • Has longer duration than others (10–15 min) • Inexpensive	• Tachycardia • Bradycardia • VPCs • Hypertension	• **Bolus:** 0.15–0.25 mg/kg IV; can be repeated • **CRI:** 5–10 mcg/kg/min
Dobutamine (12.5 mg/mL)	• Stimulates beta receptors	• Improves cardiac contractility • Less likely to induce peripheral vasoconstriction • Short duration of action	• Tachycardia • Bradycardia • VPCs • Hypertension	• **CRI: 2–15 mcg/kg/min**
Dopamine	• Low dose (1–2 mcg/kg/min) stimulates dopaminergic receptors • Mid dose (3–8 mcg/kg/min) stimulates beta receptors • High dose (> 9 mcg/kg/min) stimulates alpha receptors	• Low doses stimulate dopaminergic receptors • More likely to induce tachycardia than dobutamine • Short duration of action	• Bradycardia • VPCs • Hypertension • Tachycardia • Vasoconstriction	• **CRI:** 1–15 mcg/kg/min
Norepinephrine	• Stimulates alpha and beta receptors • Vasopressor effect on vasculature	• Increases MAP • Increases peripheral vascular resistance • Short duration of action	• Tachycardia • Vasoconstriction	• **CRI:** 0.05–2 mcg/kg/min
Phenylephrine	• Stimulates alpha receptors	• Increases MAP • Increases peripheral vascular resistance • Short duration of action	• Tachycardia • Vasoconstriction	• **CRI:** 1–10 mcg/kg/min

CRI = constant rate infusion; MAP = mean arterial pressure; VPC = ventricular premature contraction

Depth of Anesthesia & Fluid Administration

- If the patient's anesthetic plane is too deep, then the anesthetist should reduce the inhalant concentration followed by ventilation of the patient in order to efficiently washout the higher inhalant concentration.
- If packed cell volume and total plasma protein are within normal limits, the anesthetist should increase the balanced electrolyte fluid administration.
- Colloid fluids can be provided if necessary for intravascular volume expansion.

Medical Management

- Specific vasopressors or inotropes may improve vascular tone and/or myocardial contractility (**Table 2**).
- In dogs and cats, IV boluses of ephedrine, 0.15–0.25 mg/kg, can provide temporary relief of hypotension, and pending circumstances, this dose may be repeated until surgery is completed.
- Using a noninvasive blood pressure monitor at an interval of 1 to 2 minutes during the hypotensive stage will provide the proper guide for titration of inotropes (either by constant rate infusion or bolus injections).

Monitoring Equipment

- Severe hypotension may result in an improper readout from a noninvasive blood pressure monitor,[2] in other words, the values on the monitor may be incorrect.
- Using reliable indirect blood pressure monitoring, ultrasonic Doppler, or invasive blood pressure monitoring may be necessary in critically ill patients under general anesthesia.

PULSE OXIMETRY

Definition & Recognition of Abnormalities

Initial Examination

- Rule out a false alarm by checking the patient's mucous membrane color.
- Rule out poor contact of pulse oximeter probe with the tissue.
- Determine if motion from the patient or artifact or optical interference from fluorescent lighting is causing a problem.
- Determine if coloration (pigmentation) or vasoconstriction (from hypothermia, hypovolemia, or use of an alpha-2 agonist) of the tissue is causing a problem.
- Recent development of the Radical-7 pulse oximeter (**masimo.com**) employs new algorithm software that allows more reliable readings of SpO_2 in case of motion, pigmentation, hypotensive, and vasoconstrictive stage in animals.

Five Causes of Hypoxemia

A PaO_2 < 60 mm Hg (pulse oximetry reading < 92%) is indicative of hypoxemia. The following causes of hypoxemia may result in low pulse oximetry readings:[4]

- Low inspiratory fraction of oxygen (FiO_2)
- Low respiratory rate (due to hypoventilation or apnea)
- Low cardiac output due to excessively low or high heart rate or low venous return to the heart
- Pathological right-to-left shunt, physiologic intrapulmonary

right-to-left shunt, and/or ventilation/perfusion mismatch (pulmonary issue)
- Oxygen diffusion barrier through the alveolar wall induced by inflammatory lung tissue, such as pneumonia, or physical barriers, such as pulmonary edema or interstitial lung disease. Intraplural foreign bodies, such as plural effusion and viscera (due to diaphragmatic hernia), can lead to severe hypoxemia.

Additional Causes of Hypoxemia

Low FiO_2

Rule out or check:
- Lack of oxygen supply from the oxygen source in an anesthetic machine/breathing circuit
- Oxygen flow meter for a crack or leak
- Oxygen tank to verify it is not empty
- Oxygen connections to the anesthetic machine, breathing circuit, endotracheal tube, and inspired oxygen concentration (if so equipped).

A MONITOR ALARM IS SOUNDING: IS IT A MECHANICAL OR PHYSIOLOGIC PROBLEM?

While monitoring devices truly help, some devices create so many false alarms that the vigilance of the anesthetist diminishes. The anesthetist needs to be able to differentiate between malfunction of the monitor versus true physiological abnormality.

1. When an alarm sounds from ANY monitoring device, it is important to visually inspect and examine the patient FIRST. This involves examining the patient's depth of anesthesia, including:
   - » Eye position
   - » Palpebral or corneal reflexes
   - » Toe pinch withdrawal response
   - » Pulse/heart rate
   - » Color and CRT of mucous membranes
   - » Chest excursion observation
   - » Heart and lung auscultation.

   These responses or lack of responses help determine whether the patient is in distress or if there is a problem with the monitoring device.
2. False electrocardiography can be caused by background electrical noise signals, loose leads, or artifacts due to patient (or operator) movement. Artifacts and misplacement of ECG leads can be misinterpreted as arrhythmias, which is especially true with esophageal ECG when the electrodes may have poor contact with the mucosal area.
3. False alarms frequently occur with pulse oximetry due to the combination of a malpositioned probe and vasoconstriction of the vascular bed.
4. Blood pressure cuffs that are too small or large can create false BP readings.

Low Respiration/Airway Patency Issues

Rule out:
- Esophageal intubation
- Airway obstruction due to kinking of the endotracheal tube
- Obstruction of the airway due to vomitus
- Mucus plug
- Foreign body
- Overinflation of the endotracheal tube
- A deep plane of anesthesia may cause apnea and low cardiac output.

Additional Nonpulmonary Issues

- Severe bleeding or anemia may cause a loss of oxygen-carrying capacity (due to a low hemoglobin concentration).
- Occasionally, regional ischemia due to malposition of the tongue over the endotracheal tube may cut off local blood circulation, resulting in local tissue hypoxemia and/or lack of pulsation to the probe.
- Pulse oximeter monitoring is inaccurate in patients with carbon monoxide poisoning.

Mechanical Issues

- Endobronchial intubation due to over insertion of an endotracheal tube may result in ventilation–perfusion mismatch.
- Other pulmonary issues may occur due to accidental closing of the pop-off valve of a breathing circuit on a patient connected to the anesthetic machine, causing pulmonary barotrauma, possible tension pneumothorax, and subsequent death.

Management of Abnormalities

In a review study of 1281 dogs undergoing general anesthesia, 16% of dogs experienced hypoxia.[3]
- Check pulse rate and blood pressure.
- Check airway patency.
- Increase oxygen flow rate.
- Reduce the depth of anesthesia.
- Ventilate the patient with 100% oxygen until a further diagnosis can be made.
- Treat the patient with a blood transfusion to increase the hemoglobin concentration.
- Continue to monitor progress of treatment.

CAPNOGRAPHY

Definition & Recognition of Abnormalities

Capnography identifies ventilation problems.

Causes of High End-Tidal CO_2 ($ETCO_2$ > 45 mm Hg)

Clinical Conditions

- The patient is hypoventilating or apneic. In a review study of 1281 dogs undergoing general anesthesia, 63% of dogs experienced hypoventilation.[3]
- The patient is experiencing malignant hyperthermia.

Medications

Common anesthetics may cause hypoventilation/apnea.
- High doses of opioids, isoflurane, sevoflurane, propofol, tiletamine-zolazepam, and ketamine can have profound respiratory depressive effects.
- Rapid administration of propofol or diazepam/ketamine can cause apnea.
- Anesthetic combinations can induce transient hypoventilation.

Mechanical Issues

- The CO_2 absorbent is exhausted and requires replacement (this can be associated with high inspiratory CO_2 concentration as well).
- The one-way valve in the breathing circuit is missing or malfunctioning (this is also associated with high inspiratory $ETCO_2$ concentration because it allows the patient to rebreathe exhaled CO_2).
- The oxygen flow rate in a nonrebreathing circuit is too low and unable to completely wash out exhaled CO_2.
- There is a malfunction of the co-axial tubing in a Bain nonrebreathing circuit. This allows rebreathing of exhaled CO_2.

Causes of Low End-Tidal CO_2 ($ETCO_2$ < 35 mm Hg):

Clinical Conditions

- The patient is in the process of becoming apneic.
- The patient is hyperventilating (due to pain or premature awakening from anesthesia).
- The patient has low cardiac output or cardiac arrest, causing low pulmonary circulation. (This can occur in critically ill patients and/or patients that are actively bleeding out.)
- A pulmonary embolus has occurred; this event can be associated with a sudden decrease in $ETCO_2$.
- Heart failure, pleural effusion, pulmonary edema, and other diseases that cause pulmonary dysfunction may cause hyperventilation.

Mechanical Issues

- An esophageal intubation has occurred (typically, $ETCO_2$ < 7 mm Hg). Depending on how long the endotracheal tube has been incorrectly placed in the esophagus, the patient may need decompression of stomach gas.
- An airway obstruction has occurred or there is partial disconnection from the capnograph.

Management of Abnormalities

Hypoventilation

- Ventilate the patient while simultaneously reducing the depth of anesthesia (by decreasing inhalant concentrations and any other anesthetic drug dosages).
- Examine the patient's airway to ensure patency. If the patency of an endotracheal tube is suspected, immediately exchange it for a new one.
- Check for abnormal waveform on the capnogram to diagnose airway obstruction.
- Ensure adequate oxygenation.
- Is the patient obese and/or placed in dorsal recumbency? Obesity and recumbency can both contribute to inappropriate ventilation.

Hyperventilation

- Is the patient too light? If so, deepen the plane of anesthesia.
- Some opioids (morphine and hydromorphone) frequently induce hyperventilation prior to hypoventilation at higher doses.

BMP LeukoChek®

Direct Replacement for the BD Unopette™

Single Use Test for Microscopic Counting of Leukocytes and Platelets in Whole Blood and Body Fluids

- Tested to CLIA guidelines and confirmed to produce equivalent results to the discontinued BD Unopette–EP Evaluator documentation available
- Leukocyte and platelet count procedure performed exactly the same as the BD Unopette – instructions provided

Our Customers Tell Us..."I love it! It's like the Unopette never left. The procedure is exactly the same and the performance is exactly the same". *S. G. MT (ASCP) IVCH*

"…we have used the LeukoChek samples and compared the results to our in-house CBC analyzer. We found the results to be comparable and they were easier to read than the BD Unopettes". *S. C., CVT Ash Creek Animal Clinic*

Unopette™ is a trademark of Becton, Dickinson and Company

Now available in convenient 50 packs

Free Samples Available

For more information, the name of a distributor nearest you, and to obtain your free samples call 1-800-253-3684 or visit our website at www.biomedicalpolymers.com.

Toll Free: 800/253-3684 • Tel: 978/632-2555 • Fax: 978/632-2524
E-mail: sales@biomedicalpolymers.com • www.biomedicalpolymers.com

BMP Biomedical Polymers, Inc.

- A painful patient may hyperventilate. In this case, supplemental analgesia or deepening of anesthesia is the appropriate treatment choice.
- Hyperthermia may lead patients to hyperventilate in order to try to cool their body temperature. These attempts are usually unsuccessful because thermoregulation by panting requires air movement over the surface of the tongue, which cannot happen in an intubated patient.
- Acid-base disorders can lead to hyperventilation. Treating the underlying issue will correct the problem.

Low $ETCO_2$

- Check that the patient's heart is still beating; then check the endotracheal tube for disconnects or esophageal intubation.
- Follow treatment for hypotension if the patient is experiencing low cardiac output. ■

APC = atrial premature contraction; AV node = atrioventricular node; BBB = bundle branch block; BP = blood pressure; CRT = capillary refill time; ECG = electrocardiography; $ETCO_2$ = end-tidal CO_2; FiO_2 = inspiratory fraction of oxygen; PaO_2 = partial pressure of arterial oxygen; SA node = sinoatrial node; VPC = ventricular premature contraction

References

1. Dubine D. *Rapid interpretation of EKGs*, 6th ed. Tampa: Cover Publishing Company, 2007.
2. Darovic GO (ed): *Hemodynamic Monitoring—Invasive and Non-invasive Applications*. Philadelphia: WB Sauders, 1987, pp 33-61.
3. Redondo J, Rubio M, Soler G, et al. Normal values and incidence of cardiorespiratory complications in dogs during general anaesthesia. A review of 1281 cases. *J Vet Med A Physiol Pathol Clin Med* 2007; 54(9):470-477.
4. Tremper KK. Oximetry, capnography and other non-invasive wizardry. *Anesth Analg* 1992; (Suppl):136-141.

***Jeff Ko**, DVM, MS, Diplomate ACVA, is a professor in the Department of Veterinary Clinical Sciences at Purdue University College of Veterinary Medicine. He has authored numerous articles and book chapters in the field of anesthesia and pain management. Dr. Ko lectures extensively at regional, national, and international conferences.*

***Rebecca Krimins**, DVM, is currently a third-year anesthesia resident in the Department of Veterinary Clinical Sciences at Purdue University College of Veterinary Medicine. Dr. Krimins received her DVM from Ross University School of Veterinary Medicine and her undergraduate degree from University of Chicago.*

VETLAND

Operating with Accuracy & Safety is our Priority

EX 3000
- Safest, most advanced veterinary anesthesia system in the world
- Monitors Top Risk Parameters
- Monitors drug % and limits set by user
- Monitors breath to breath ventilation and supply pressures
- Monitors for high & continuing lung pressure

V12
- Veterinary specific monitoring system
- Veterinary specific algorithms
- Most accurate NIBP monitoring
- Best value in patient monitoring

Vetland Medical
Sales & Services
Toll Free: 877-329-7775
www.vetland1.com