Monitoring of the Emergent Small Animal Patient

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KEYWORDS
- Emergency • Monitoring • Triage • Diagnostics • Small animals

KEY POINTS
- Careful monitoring of the emergent patient is crucial in assessment and treatment of potentially life-threatening conditions.
- Monitoring equipment does not replace the clinical evaluation of the patient. Hands-on serial patient assessment can recognize patient changes before clinical deterioration.
- Major body systems assessed include the respiratory system (eg, airway, breathing), cardiovascular system (eg, circulation), and neurologic system (eg, dysfunction).
- Assessment of the cardiovascular system begins with hands-on patient assessment, followed by timely serial assessments, and is supplemented with diagnostics including electrocardiography and monitoring of blood pressure.
- Assessment of the respiratory system begins with observation of the patient from afar, followed by hands-on assessment. It can then be supplemented with diagnostics including radiographs, pulse oximetry, and arterial blood gases.

Monitoring of the emergent patient is a challenging aspect in both the emergency room and intensive care unit (ICU). When people think of “monitoring,” the immediate reaction is monitoring with equipment. Monitoring equipment does not replace the clinical evaluation of the patient. Hands-on monitoring is an important aspect of patient evaluation and patient care, and allows one to better focus the diagnostics and treatment plan. While useful, there are limitations of monitoring systems that are important to be cognizant of, as invasive monitoring may have potential negative consequences in a critically ill patient.

The condition of an emergent patient can dramatically change on a minute-to-minute basis, not only making the initial assessment valuable, but emphasizing the importance of serial assessment as part of thorough patient management. Although diagnostics can be performed to supplement the physical examination, diagnostics do not replace a thorough examination. Moreover, with the increasing costs of veterinary medicine, clients may have significant limitations, preventing advanced diagnostics.
As a result, the use of physical examination findings to help fine-tune appropriate diagnostics will allow for overall better patient care.

In the patient that presents on emergency, the initial focus is on triage and the primary survey. Triage is the art of giving priority to patients and their problems on presentation to the hospital. Triage involves a concise history, including the patient signalment, primary complaint, and time of onset. The signalment (eg, age, breed, sex) helps provide a differential diagnosis list, as younger patients may have a different differential list (eg, trauma, poisoning) in comparison with older patients (eg, neoplasia, metabolic disease), and intact dogs (eg, pyometra, prostatic abscess) may have a differential list different to that of spayed or neutered patients. A triage examination to assess the major body systems is then completed. In veterinary medicine, emergent findings which warrant immediate care may include trauma, toxin exposure, urethral obstruction, seizures, bleeding, heat prostration, shock, wounds, anemia, or reproductive emergencies.1

Following the triage examination, a primary survey is performed. This survey is designed to rapidly identify abnormalities associated with life-threatening conditions. Major body systems assessed include the respiratory system (eg, airway, breathing), cardiovascular system (eg, circulation), and neurologic system (eg, dysfunction). Failure to recognize an abnormality in any of these systems can result in immediate life-threatening deterioration of the patient. There are several pneumonics used to aid in primary survey evaluation, including ABC-LOC (airway, breathing, circulation, level of consciousness) and ABCD (airway, breathing, circulation, drug use/exposure/dysfunction).

**RESPIRATORY (AIRWAY AND BREATHING)**

Assessment of the respiratory system begins with simple observation of the patient from afar, followed by hands-on assessment. Visual assessment of the respiratory system includes making sure the patient has a patent airway and is ventilating adequately. Because the patient may be most comfortable with the owner, the respiratory visual examination is often performed while taking a triage history from the owner to obtain a sense of the respiratory status before handling, as stress, pain, or anxiety may alter the respiratory pattern and make the respiratory assessment difficult.

During the normal respiratory cycle, the primary work of breathing is through contraction and relaxation of the diaphragm. During inspiration, contraction of the diaphragm results in the chest wall and abdomen moving outward in a coordinated manner. During expiration the diaphragm relaxes, and the chest and abdominal walls move inward. 2 In patients with respiratory distress, clinical signs of increased work of breathing, tachypnea, cyanosis, orthopnea, open-mouth breathing, restlessness, or an inability to lie down may be seen. Other abnormalities may include short and shallow breathing with absent chest wall motion or flaring of the nares. With severe respiratory distress, paradoxic respiration may be seen (eg, when the chest wall and abdominal wall move in opposite directions). Paradoxic abdominal movement is seen in conditions preventing adequate lung inflation, including upper respiratory tract obstruction, diaphragmatic injury, decreased lung compliance, and pleural effusion.

Posture is another visual cue when assessing the respiratory system. A patient in respiratory distress may display orthopnea, most commonly seen as head and neck extension and abducted elbows. Elevation of the head and extension of the neck allows straightening the trachea while abduction of the elbows minimizes compression of the chest wall. Clinical signs of orthopnea are often associated with advanced, severe respiratory fatigue and potentially imminent respiratory arrest or failure.
Anatomic localization of the origin of the respiratory disease may be determined based on respiratory patterns. There are 5 breathing patterns commonly seen in patients that present for respiratory distress.

1. An increased respiratory rate (eg, tachypnea) does not always indicate pathologic pulmonary disease (eg, panting, healthy canine patient). When presented with a tachypneic patient, work of breathing, auscultation, and ancillary testing (eg, pulse oximetry) should be assessed; if normal, other causes of tachypnea such as “non-respiratory lookalikes” (eg, anemia, pain, anxiety, fever, metabolic acidosis) should be considered.

2. Pleural space disease is visually characterized by shallow, rapid respirations. Auscultation may reveal the presence of dull lung sounds and muffled heart sounds. Differentials for pleural space disease include pneumothorax, pleural effusion (eg, hemothorax, pyothorax, chylothorax), and diaphragmatic hernia.

3. Upper airway obstruction is characterized by inspiratory stridor or stertor. With upper airway obstruction the respiratory rate may be normal; however, respiratory effort may be dramatic owing to ventilation through a narrowed airway. Differentials include laryngeal paralysis, tracheal collapse, nasopharyngeal polyp, neoplasia, granulomatous disease, coagulopathy (resulting in bleeding into the upper airway), and brachycephalic airway syndrome.

4. Lower airway disease is typically characterized by tachypnea with a prolonged expiration and an expiratory push. Expiratory wheezing may also be found on auscultation. Common differentials include feline allergic airway disease, pulmonary fibrosis, and chronic bronchitis.

5. Pulmonary parenchymal disease is characterized by both labored inspiration and expiration. On auscultation, common findings may include harsh lung sounds, pulmonary crackles, and wheezes. Common differentials include infectious pneumonia (eg, bacterial, fungal, viral, protozoal, parasitic), aspiration pneumonia (eg, infectious and chemical pneumonitis), interstitial lung diseases, pulmonary edema (cardiogenic and noncardiogenic), coagulopathies (eg, long-acting anticoagulants), and neoplasia.

Following visual examination and hands-on examination (eg, auscultation), diagnostic tools may be used to supplement the examination of the respiratory system. In general, diagnostics such as thoracic radiographs are not considered a first line of defense when assessing the respiratory system, as this poses a stressful threat to a compromised patient. Once stabilized, however, advanced diagnostics can be performed. Such methods include pulse oximetry, arterial blood gas (ABG) or venous blood gas (VBG) analysis, thoracic radiography, and Thoracic Focused Assessment with Sonography for Trauma (TFAST). Please see the article “The Use of Ultrasound for Dogs and Cats in the Emergency Room (AFAST and TFAST)” elsewhere in this issue for more information.

Pulse oximetry (SpO2) is a noninvasive, readily available diagnostic monitoring tool that can be used to evaluate oxygenation, and is often considered the first objective method for assessing severity of hypoxemia in a patient. Pulse oximetry works by spectrophotometry and measures 2 forms of hemoglobin that circulate in arterial blood: oxyhemoglobin (saturated hemoglobin) and deoxyhemoglobin (unsaturated hemoglobin). A combination of light reflectance and absorption is used to measure the concentrations of oxyhemoglobin and hemoglobin present within red blood cells (RBC). The measurements are placed in a formula to calculate the percentage of saturation (SaO2 = [HbO2/HbO2 + Hb] × 100). Pulse oximetry allows clinicians to measure SpO2 and therefore extrapolate the partial pressure of oxygen (PaO2) based on the oxyhemoglobin dissociation curve (Fig. 1). Although pulse oximeters estimate
hemoglobin saturation, they do not assess oxygen delivery or tissue perfusion. The pulse oximeter can be used for intermittent monitoring of oxygen saturation, or alternatively to provide a continuous real-time assessment, which is particularly useful for monitoring general anesthesia or sedation.

Common testing sites for using the pulse oximeter include the mucous membranes (eg, tongue, buccal membrane) or thin, nonpigmented skin found on the pinnae, back of the hock, prepuce, vaginal fold, or interdigital hairless areas of the paw. In healthy patients, the SpO₂ when breathing room air (fraction of inspired oxygen [FiO₂] 21%) is expected to be at least 96%. SpO₂ measurements of less than 93% to 94% require further evaluation, and typically require oxygen supplementation. An SpO₂ of 90% indicates severe hypoxemia, and is consistent with a PaO₂ of 60 mm Hg (normal 80–100 mm Hg, at sea level).

One must keep in mind, however, that the pulse oximeter cannot differentiate between oxyhemoglobin, carboxyhemoglobin (eg, from carbon monoxide toxicosis), methemoglobin (eg, from acetaminophen toxicosis), or cyanide toxicity. Of note, cyanide poisoning may result in a falsely elevated SpO₂ because it reduces the oxygen extraction from the arterial blood.⁴,⁵ As a result, this tool has limited use for patients with carbon monoxide poisoning or methemoglobinemia. Severe anemia, hypotension, or vasoconstriction may also result in a falsely low SpO₂. Dark skin pigmentation will result in light interference and be difficult to interpret. On the pulse oximeter the signal strength, detected heart rate, and waveform oscillation should always be assessed (eg, matching) when evaluating the reading to ensure accurate results.

ABG analysis is the gold standard for direct assessment of pulmonary function (eg, oxygenation and ventilation) and provides information about the metabolic acid-base status of the body.⁶ The most commonly evaluated parameters include pH, PaO₂, partial pressure of carbon dioxide (PaCO₂), and bicarbonate (HCO₃). Normal PaO₂ is expected to be 80 to 100 mm Hg (FiO₂ 21%).⁷ Normal PaCO₂ is 35 to 45 mm Hg. Advanced blood gas machines may also assess electrolytes, blood urea nitrogen (BUN), creatinine, and blood lactate concentrations.

Common locations for ABG sampling include the dorsal pedal artery, the auricular artery, or the femoral artery. Owing to physical restraint, an ABG analysis may not
be possible because of the added stress of a patient already in respiratory distress (eg, feline patient). Rather, a VBG analysis may be a substitute when evaluating the pH, P\textsubscript{CO\textsubscript{2}}, electrolytes, and blood lactate. As P\textsubscript{venousCO\textsubscript{2}} is typically within 5 mm Hg of P\textsubscript{arterialCO\textsubscript{2}} when perfusion is adequate, ventilation—not oxygenation—can be adequately assessed on VBG analysis.\textsuperscript{8} Increases in P\textsubscript{CO\textsubscript{2}} indicate compromised ventilatory function and may result in increasing acidosis, mental depression, and vasodilation. Decreases in P\textsubscript{CO\textsubscript{2}} may be seen in hyperventilating patients or may be related to poor gas exchange. Oxygenation cannot be adequately assessed on VBG analysis, and should only be assessed with ABG. However, the combined use of a VBG analysis and pulse oximetry reading can be used to provide information similar to that obtained with ABG, based on extrapolation from an oxygen hemoglobin dissociation curve (see \textbf{Fig. 1}).

When assessing an ABG with oxygen supplementation, the P\textsubscript{aO\textsubscript{2}} should be 5 times the inspired oxygen concentration with normal pulmonary function. For example, when breathing room air (Fi\textsubscript{O\textsubscript{2}} 21%), the P\textsubscript{aO\textsubscript{2}} should be 100 mm Hg (21 \times 5 = 105 mm Hg). Under anesthesia (Fi\textsubscript{O\textsubscript{2}} 100%), the P\textsubscript{aO\textsubscript{2}} should be 500 mm Hg (100 \times 5 = 500 mm Hg). Another way of assessing oxygenation is by calculating the alveolar-arterial (A-a) gradient (P\textsubscript{aCO\textsubscript{2}} = [(P\textsubscript{B} – P\textsubscript{H\textsubscript{2}O}) \times Fi\textsubscript{O\textsubscript{2}}] – P\textsubscript{aco\textsubscript{2}} (1/RQ)) (where RQ is respiratory quotient), or P\textsubscript{aCO\textsubscript{2}}:Fi\textsubscript{O\textsubscript{2}} ratio. These formulations provide a more objective assessment of respiratory function. Patients with an A-a gradient greater than 10 to 15 mm Hg\textsuperscript{9,10} or a P\textsubscript{aCO\textsubscript{2}}:Fi\textsubscript{O\textsubscript{2}} ratio less than 300 mm Hg have respiratory compromise, and may require further intervention ranging from supplementation oxygen administration to mechanical ventilation. These latter 2 formulations should only be assessed based on an ABG analysis.

\textbf{CARDIOVASCULAR SYSTEM (CIRCULATION)}

Assessment of the cardiovascular system also begins with hands-on patient assessment, followed by timely serial assessments. Patient assessment should include heart rate and rhythm, pulse quality, mucous membrane color, and capillary refill time. Ancillary monitoring devices for the emergent patient include electrocardiography (ECG) and blood pressure monitoring.

ECG records the electrical activity generated by the heart from electrodes attached to the skin of the patient. It allows one to assess if the electrical activity is normal, thus providing information on the expected contraction of the heart. The ECG assesses heart rate and rhythm, but does not measure or assess cardiac output or tissue perfusion.\textsuperscript{11} Monitoring of the critically ill patient with initial, intermittent, or even continuous ECG evaluation is important, particularly in patients with underlying cardiac disease, metabolic disease, neoplasia, or at risk for development of arrhythmias. Arrhythmias result from abnormalities of impulse generation that alter the heart rate, heart rhythm, origin of excitation, or atrial and ventricular depolarization owing to interference with electrical conduction. If the electrical current is abnormal, the clinician must determine if the abnormality is a result of primary cardiac abnormalities resulting in abnormal impulse generation and conduction (eg, dilated cardiomyopathy), a systemic illness (eg, ventricular premature contractions [VPCs] seen following splenectomy), or other underlying conditions such as electrolyte abnormalities, hypoxia, trauma, or those secondary to medications.\textsuperscript{11,12} The ECG should be used to look for the presence of dysrhythmias, bradycardia, or tachycardia. Both tachyarrhythmias and bradyarrhythmias may result in decreased cardiac output (because of poor ventricular delivery or poor ventricular filling),
potentially resulting in congestive heart failure, secondary end-organ failure (eg, acute renal failure, “shock gut”) and sudden death. Table 1 lists examples of tachyarrhythmias and bradyarrhythmias. The overall prognosis for a malignant arrhythmia depends on the underlying cause or disease and response to treatment. In general, the following parameters should warrant immediate intervention if detected on ECG:

- Dog: heart rate (HR) less than 50 beats/min or greater than 180 beats/min
- Cat: HR less than 120 beats/min or greater than 240 beats/min
- Presence of severe VPCs including R-on-T phenomenon (often predisposing to serious ventricular arrhythmias such as ventricular fibrillation)
- Ventricular tachycardia greater than 180 beats/min
- Pulse deficits
- Hypotension
- Clinical symptoms of poor perfusion (eg, prolonged capillary refill time, dull mentation)

When evaluating an arrhythmia, common questions that must be answered to determine if the arrhythmia requires therapy include:

1. Is the arrhythmia hemodynamically significant (eg, causing HR changes, absent pulses, weakness, collapse, blood pressure changes, and resulting overall in clinical signs associated with poor perfusion)?
2. Is the arrhythmia one that can lead to further morbidity and mortality (eg, R-on-T phenomenon, sustained ventricular tachycardiac, HR >180 beats/min)?
3. Can you identify an underlying cause for the arrhythmia (eg, T waves may be large relative to the R waves during hyperkalemia)?
4. What are the risks of beginning therapy? Do the benefits outweigh the risks (keeping in mind that antiarrhythmic therapies have the potential to be proarrhythmogenic)?

The readers are referred to a cardiology resource for additional information of treatment of cardiac arrhythmias. Emergency cardiac drug dosing can be found in Table 9 of the article elsewhere in this issue on emergency management and treatment of the poisoned small animal patient.

**BLOOD PRESSURE MONITORING**

Blood pressure monitoring in the emergent or critically ill patient is an important monitoring tool in the diagnosis of hypotension or hypertension. Hypotension is more commonly seen in the emergency room or ICU setting following hypovolemia,

<table>
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<tr>
<th>Table 1</th>
<th>Types of cardiac arrhythmia</th>
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<tr>
<td>Tachyarrhythmias</td>
<td>Atrial tachycardia</td>
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<td></td>
<td>Atroventricular (AV) nodal tachycardia</td>
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<td>Ventricular tachycardia</td>
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<td>Ventricular fibrillation</td>
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<td>Atrial fibrillation</td>
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<td>Bradyarrhythmias</td>
<td>Sick sinus syndrome</td>
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<td>Severe second-degree AV block</td>
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<td>Third-degree AV block</td>
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<td>Atrial standstill</td>
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<td>Asystole</td>
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hemorrhage, sepsis, and so forth, and must be treated aggressively and rapidly to ensure adequate tissue perfusion. However, when assessing blood pressure, it is important that it is not directly correlated with perfusion; rather, it is an assessment of overall global tissue perfusion. The assumption is that if the patient is hypotensive, blood flow in tissue will be inadequate, leading to decreased tissue perfusion.\textsuperscript{13,14}

The primary treatment for hypotension should be directed at the underlying cause, as there are many variables that affect blood pressure (Fig. 2). Hypotension as a result of hypovolemia is treated with intravenous fluid therapy. Isotonic crystalloid boluses of 10 to 30 mL/kg or colloid boluses with hydroxyethyl starch (eg, Hetastarch) of 5 to 10 mL/kg over 15 to 30 minutes can increase the circulating fluid volume and improve the blood pressure. Patients with underlying cardiac disease (eg, chronic valvular heart disease, dilated or hypertrophic cardiomyopathy) that require fluid resuscitation warrant the use of judicious fluid therapy (eg, smaller volumes such as 5–10 mL/kg of an isotonic crystalloid or 2–5 mL/kg of a colloid over 30–60 minutes) followed by careful reassessment to determine if additional fluid boluses are warranted. Large volume resuscitation is not appropriate for every hypovolemic patient with normal cardiac function.

Hypotensive resuscitation is a form of fluid resuscitation for stabilization of the presurgical patient with uncontrolled hemorrhage. The most commonly practiced form of limited-volume resuscitation in veterinary medicine is permissive hypotension. This form of fluid resuscitation consists of administration of conservative volumes of intravenous fluids before definitive control of hemorrhage, with the target of a systolic blood pressure of 60 to 80 mm Hg. The goal is to provide adequate perfusion to the vital organs (eg, brain, kidneys, heart) without an increased risk of further massive hemorrhage.\textsuperscript{15,16} Conversely, hypotension is not always a product of hypovolemia, and when hypotension exists despite an adequate circulating fluid volume, other therapies may be necessary. Inotrope therapy with blood pressure medications such as dopamine (5–20 \( \mu \)g/kg/min), dobutamine (2–20 \( \mu \)g/kg/min), or norepinephrine (0.05–0.4 \( \mu \)g/kg/min) may be needed.

In hypertensive patients (mean arterial pressure [MAP] >160 mm Hg or systolic pressure >200 mm Hg), several factors must be addressed, including toxicant ingestion (eg, selective serotonin reuptake inhibitor [SSRI] antidepressants, amphetamines), agitation, pain, underlying metabolic disease (eg, heart disease, renal disease, hyperadrenocorticism, immune-mediated hemolytic anemia), and neoplasia (eg, pheochromocytoma), among others. The use of judicious sedation/analgesia, anxiolytics, vasodilators, or angiotensin-converting enzyme inhibitors may be necessary. If the hypertension is toxicant related and concurrent agitation is simultaneously observed, the repeated use of sedatives may be required in cardiovascularly stable patients.

<table>
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<th>Blood pressure = CO x Systemic Vascular Resistance</th>
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<tr>
<td><strong>Heart Rate</strong></td>
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<tr>
<td><strong>Stroke Volume</strong></td>
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<tr>
<td><strong>Preload</strong></td>
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<tr>
<td><strong>Afterload</strong></td>
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<td><strong>Contractility</strong></td>
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**Fig. 2.** Summary of factors controlling cardiac output: Central determinants of blood pressure are stroke volume and heart rate, both of which influence cardiac output. Changes in either will affect cardiac output.
(eg, phenothiazines). In patients failing to respond to sedative/anxiolytic therapy, the use of antihypertensives or other cardiac medications may be indicated to prevent vascular injury or secondary complications (eg, retinal detachment).

There are several methods for measuring blood pressure, including measurement of direct arterial blood pressure (DABP), Doppler blood pressure, and oscillometric blood pressure; these are listed in order of accuracy.

**Monitoring of Direct Arterial Blood Pressure**

Monitoring of direct arterial blood pressure is considered the gold standard for blood pressure measurement in both veterinary and human patients, and allows the clinician to assess trends in pressure changes. It requires placement of an arterial catheter, typically in the dorsal pedal artery. Other sites for arterial catheter placement include the femoral artery and the auricular artery.\(^{17,18}\) Placement of an arterial catheter allows for rapid blood sampling and ABG analysis in the critically ill patient.

To monitor DABP, a catheter must be connected to a pressure transducer and monitor; this transducer then converts the pressure wave to an electrical impulse, which is transmitted to the monitor for display. This process allows for continuous monitoring of systolic pressure, diastolic pressure, and MAP. One must keep in mind that false results may be seen with compliant tubing, clot formation within the catheter, air bubbles in the tubing, or malfunction of catheter or tubing. Additional complications seen with arterial catheter placement include catastrophic hemorrhage (eg, from accidental disconnection of the arterial line), hematoma formation at the site of arterial puncture, thrombosis of the artery (more common in cats), necrosis of the tissues distal to the catheter (notably in cats that have an indwelling catheter for more than 6–12 hours), and infection.\(^{19}\) Despite many advantages, DABP monitoring is often limited to critically ill patients or those patients under anesthesia (eg, intraoperative or receiving mechanical ventilation), where there is little movement and the benefits outweigh the risks of arterial catheter placement.

**Monitoring of Noninvasive Blood Pressure**

Monitoring of noninvasive blood pressure is based on inflation of a cuff to levels greater than systolic pressure to occlude arterial flow, followed by measurement of the pressure at which flow returns; this is typically measured by Doppler or oscillometric measurement. Doppler blood pressure measures systolic pressure, and is preferred because of its accuracy in smaller animals such as cats and small dogs. The Doppler unit consists of a probe, amplifier, speaker, and rechargeable battery. The probe consists of two piezoelectric crystals, one transmitting a continuous ultrasonic wave at a set frequency while the other crystal acts as a receiver. The Doppler method is preferred in patients with hypotension or in those with arrhythmias or tachycardia, which make the oscillometric results inaccurate. An appropriately sized cuff (measuring about 40%–60% of the limb or tail circumference) is placed proximal to the Doppler probe. This cuff is inflated to a pressure greater than the expected systolic pressure, resulting in absence of the sound signal. Once the signal is lost, the pressure in the cuff is slowly reduced. When the flow in the artery returns, the return of the audible pulse signal indicates the systolic arterial pressure.\(^{20}\)

Oscillometric blood pressure devices measure oscillations within a cuff bladder using a microprocessor. Examples of oscillometric devices used in veterinary medicine include brands such as the Dinamap (ie, Device for Indirect Noninvasive Automatic Mean Arterial Pressure) and Cardell. Common locations for blood pressure measurement and cuff placement include just below the elbow, above the hock, below the hock, or on the tail base. Following cuff placement, the cuff is pressurized. The
pressure is held constant while the microprocessor samples pressure oscillations and is incrementally deflated. During cuff deflation the microprocessor measures the pressure, averaging the amplitude of pressure oscillations.\textsuperscript{21,22} The peak amplitude of oscillations equals the MAP. Systolic pressure equals the pressure at which oscillations are first detected, and diastolic pressure equals the pressure at which oscillations decrease rapidly. The HR calculated by the blood pressure machine should be compared with the patient’s pulse rate on examination or heart rate on auscultation to ensure accuracy; one that does not match is generally considered inaccurate.\textsuperscript{21} Many factors can adversely affect oscillometric blood pressure measurement. Common causes for erroneous readings include movement of the patient, arrhythmias, tachycardia, and inappropriate cuff width. A cuff that is too wide will result in an artifactual lower reading, and a cuff that is not wide enough will result in a falsely elevated reading.

**NEUROLOGIC ASSESSMENT (DYSFUNCTION)**

Primary neurologic evaluation should include both an extracranial and intracranial assessment. Extracranial assessment for life-threatening injuries includes hemorrhage, respiratory distress, and trauma. Triage of the ABCDs should initially be cursory to allow rapid identification and stabilization of a problem; therefore, although a full neurologic examination is not immediately performed, dysfunction is still considered a priority in initial monitoring of the emergency patient. Patients in severe hypovolemic shock may have decreased mentation, and should be frequently assessed during volume resuscitation to evaluate if the neurologic dysfunction improves. Likewise, patients should be assessed for appropriate neurologic dysfunction before the administration of analgesics, which may skew further assessment. Although the initial examination is important, serial patient assessment will allow for detection of changes in mental status. If detected, this should prompt a more in-depth patient evaluation and investigation of underlying causes. Once life-threatening issues are assessed, intracranial priorities include maintaining cerebral perfusion pressure (CPP) and ensuring oxygen delivery to the brain.\textsuperscript{23} CPP, which is the difference between intracranial pressure (ICP) and MAP, can be enhanced by decreasing ICP (eg, mannitol, 15°–30° head elevation) and maintaining MAP (eg, fluid therapy, oxygen therapy).

When assessing the emergent patient neurologically, the Modified Glasgow Coma Scale (GCS) may be used. The modified GCS is a quantitative assessment of neurologic function assessing level of consciousness, brainstem reflexes, and motor activity and posture. Each category has a scale ranging from 1 to 6, with 6 being normal and 1 being recumbent, nonresponsive, and areflexic with dilated, unresponsive pupils and an absent oculocephalic reflex. Once the assessment is complete, the scores for each of the 3 systems are cumulated. The total score provides a 48-hour survival prognosis, with 3 to 8 being grave, 9 to 14 being guarded, and 15 to 18 yielding a good prognosis. One must exercise caution when prognosticating a neurologically impaired patient based on an initial assessment, as the trend over time is more valuable when predicting the final outcome.\textsuperscript{24,25} The reader is referred to the article elsewhere in this issue on updates in the management of the neurologic trauma small animal patient for further information.

In the veterinary patient, common variables assessed in the neurologic examination/dysfunction include:

- Mentation:
  - Alert
  - Obtunded (dull, but can be aroused by nonnoxious stimuli)
- Stuporous (semiconscious/somnolent, rousable with a noxious stimulus)
- Comatose (unconscious, cannot be roused by a noxious stimulus)
- Brain dead (no cerebrocortical electrical activity, no brainstem reflex function)

- Pupils: size, symmetry, pupillary light reflexes
- Presence and direction of nystagmus
- Menace response
- Facial asymmetry
- Posture
- Pain sensation, conscious proprioception, and withdrawal reflexes in limbs

In patients suspected of having increased ICP, careful and frequent monitoring of HR and systemic blood pressure are imperative to rule out the rapid onset of the Cushing reflex. The Cushing reflex (also referred to as the vasopressor response) is a physiologic nervous system response to increased ICP, and is clinically detected based on an acute onset of bradycardia and severe hypertension. Physiologically an increased ICP results in an increase in the cerebrospinal fluid pressure. When the ICP exceeds the MAP, the arterioles in the cerebrum become compressed. Compression results in diminished blood supply to the brain, leading to cerebral ischemia. Cerebral ischemia activates the parasympathetic and sympathetic nervous systems; activation of the sympathetic nervous system results in arterial vasoconstriction, increasing the total resistance of blood flow and elevating the systemic blood pressure. Systemic hypertension is produced in an attempt to restore blood flow to the ischemic cerebral arterioles. Baroreceptors in the carotid arteries detect the hypertensive compensation and trigger a parasympathetic response via the vagus nerve, resulting in bradycardia.26,27 When clinical signs of Cushing reflex are seen, imminent herniation may occur; therefore, the patient should be treated aggressively and immediately (eg, with mannitol).

TEMPERATURE

Monitoring a patient’s body temperature provides valuable information and does not require expensive equipment, and is most commonly performed rectally or via placement of the probe in the axilla. If using a rectal thermometer in the axilla, it is recommended to add 1 Fahrenheit degree for an equivalent approximate rectal temperature, as this modality is generally not considered to be as accurate.28

When an elevated rectal temperature is detected, it is important for the clinician to differentiate hyperthermia from a true fever. Heat balance occurs through the actions of heat-gaining and heat-dissipating mechanisms. Heat gain is seen with hypermetabolism, exercise, increased muscle activity (eg, tremors, seizures), and elevated ambient temperature (eg, heat stroke, when locked in a car). Heat-dissipating methods include behavioral changes such as finding a cool location, panting, and peripheral vasodilation. When heat gain exceeds the ability of the body to dissipate heat, hyperthermia occurs. Risk factors for developing hyperthermia include upper airway obstruction, laryngeal paralysis, brachiocephalic airway syndrome, high ambient humidity, and collapsing trachea.29,30 With fever, differentials such as underlying infectious, inflammatory, or neoplastic processes should be considered. As fever is an endogenous source of heat, patients presenting with fever should not be cooled, in contrast to exogenous sources of heat (eg, hyperthermia), which should be cooled.

When hyperthermia is present, rapid cooling measures should be instituted when body temperatures exceed 104°F to 105°F/40°C to 40.5°C in the dog or 106°F/41.1°C in the cat.29–31 Hyperthermia may result in decreased perfusion to the mesentery
and thermal injury to the gastrointestinal tract, hematemesis, hematochezia, bacterial translocation, hypoglycemia, disseminated intravascular coagulation (DIC), neuronal damage, cerebral edema, hemorrhage, and seizures.

Convection is the most effective cooling method, which can be accomplished by wetting the patient with cool (not cold) water and using a fan to disperse the heat. Applying ice or cold water is discouraged, as this may cause peripheral vasoconstriction and delay heat loss. To prevent rebound hypothermia, cooling measures should be discontinued when body temperature reaches 103°F/39.4°C.

Although hyperthermia is more common in emergent patients, hypothermia is equally concerning. Again, rule-outs include an exogenous versus endogenous cause for hypothermia. An exogenous source (eg, a fall through ice, heat loss by anesthesia) warrants active warming, in contrast to an endogenous source (eg, severe dehydration or hypovolemia resulting in poor blood flow to the rectal area, resulting in hypothermia). With endogenous sources of hypothermia, warming should only be performed with resolution of the cause of the hypothermia; in other words, the concurrent use of intravenous fluid therapy to help perfuse the patient while simultaneously warming the patient is warranted.

Mild hypothermia is defined as a body temperature anywhere from 96°F to 98°F/35.6°C to 36.7°C, whereas moderate hypothermia is 94°F to 96°F/34.4°C to 35.6°C and severe hypothermia is 90°F to 94°F/32.2°C to 34.4°C. A temperature lower than 90°F/32.2°C must be addressed rapidly, as this is considered life-threatening hypothermia. Severe hypothermia can lead to cardiac abnormalities, vasodilation, and decreased blood flow. Heat support is provided with gloves or bottles filled with warm water, or blankets with circulating warm water. To prevent rebound hyperthermia, warming measures should be discontinued when body temperature reaches 99°F to 100°F/37.2°C to 37.8°C.

**BODY WEIGHT**

Hospitalized patients should be weighed at least once a day (ideally on the same scale). Recording body weight in the metric scale is advantageous, as 1 kg = 1000 mL. Azotemic, oliguric, or anuric patients should be weighed at least 3 to 6 times per day. This simple monitoring tool can be used to assess changes in hydration status based on weight gain or weight loss. Acute changes in body weight are caused by changes in fluid balance rather than body mass. Once hydrated, an acute gain in body weight would be a concern for excess fluid accumulation (eg, edema). Conversely, acute weight loss may indicate ongoing fluid losses or continued dehydration. For example, a 30-kg dog that is 10% dehydrated requires 3 L to replace dehydration. As 1 L equates to 1 kg, this patient is expected to gain approximately 3 kg with appropriate fluid resuscitation. A 0.1-kg change in body weight translates to 100 mL of fluid gained or lost.

**URINE OUTPUT**

Urine output (UOP) is often an underused tool in the critically ill patient or even in the patient with urethral obstruction. Along with body weight, UOP can be an important tool in assessing hydration status. Normal UOP ranges from 1 to 2 mL/kg/h. A decreased UOP despite adequate hydration and perfusion, reported as less than 0.5 mL/kg/h, is referred to as oliguria. Total lack of UOP is referred to as anuria. Fluid intake (“ins”) measurement is often easy, quantified by the digital fluid pump. By contrast, urine output (“outs”) can be monitored grossly (eg, quantified either during walks or measurement of absorbent pads within the cage) or via measurement with
a closed-system urinary catheter collection. A closed urinary collection system allows continuous urine collection and measurement to compare with fluid intake (ins), to ultimately assess fluid balance within the body, as ins should match the outs to ensure adequate hydration.37,38

The placement of a urinary catheter and a closed urinary collection system is not without risk. Nosocomial and ascending bacterial urinary tract infections, along with sedation risks and possible urethral injury, can occur, albeit rarely. Urinary catheter placement should be performed using a strict aseptic technique, and removed as soon as clinically possible to help prevent secondary infection.39

Aside from volume measurement, the urine-specific gravity (USG) can provide information regarding hydration. First, the USG should ideally be assessed before any administration of fluid; this will allow adequate evaluation of underlying renal function. Once a patient has been treated with intravenous fluids USG can be evaluated, as patients with ongoing fluid deficits will concentrate their urine, resulting in a hypersthenuria (cat >1.040, dog >1.025). Ideally, patients on intravenous fluids should be isosthenuric (eg, USG 1.015–1.018), as this indicates appropriate hydration.

Measurement of ins and outs, UOP, urine volume, and USG should all be assessed together to evaluate the clinical picture of the patient. An increased USG with a lower UOP (eg, USG 1.038, UOP 0.5 mL/kg/h) often indicates dehydration, and warrants an increase in fluid therapy. Conversely, a decreased UOP with concurrent isosthenuria or hyposthenuria (eg, USG 1.015, UOP 0.5 mL/kg/h) in an adequately hydrated and euvoletic azotemic patient may warrant diuretics such as furosemide or vasopressor support (to increase renal blood flow), if appropriate. Patients should be adequately hydrated based on additional assessment measures (eg, physical examination, evidence of hemodilution, weight gain, central venous pressure [CVP]) before instituting medications to increase UOP (eg, furosemide, mannitol); otherwise these drugs may result in increased UOP at the expense of dehydrating the patient.

MINIMUM DATABASE MONITORING

A basic emergency minimum database should include packed cell volume (PCV), total solids (TS), BUN, blood glucose (BG), and a blood smear. Depending on the capabilities of the clinic and the stability of the patient, further diagnostics may include electrolytes, creatinine, lactate, VBG, pulse oximetry, ECG, and blood pressure. Patients on intravenous fluids should have a daily minimum database (eg, PCV/TS/BG/BUN) and electrolytes (eg, Na\(^+\), K\(^+\)) performed. Additional types of point-of-care machines are becoming more common in private practice settings, offering rapid assessment of variables that once took 24 hours to obtain.

LACTATE

Hand-held lactate machines can be used as an inexpensive monitoring point-of-care test in the emergency room or ICU. Hyperlactatemia occurs commonly in emergent and critically ill patients, likely secondary to a lactic acidosis following poor perfusion. Hyperlactatemia may result from hypoperfusion, liver failure, sepsis, lactate-containing fluid therapy, toxins, and drug therapy.40 Hyperlactatemia is defined as a plasma lactate level above normal, commonly greater than 2.5 mmol/L.41 In the 1990s an elevation in initial lactate was used to prognosticate for certain conditions42; since then, a recent push has been made to use serial lactate measurements to assess response to therapy instead.43
COAGULATION TESTING

Coagulopathies are common in emergency patients. Diagnostics to evaluate the coagulation system include activated clotting time, prothrombin time, activated partial thromboplastin time, platelet count, and d-dimers. Evaluation of red cell morphology as part of the complete blood count and blood smear may reveal the presence of red cell fragments, schistocytes, anisocytosis, and polychromasia. The in-house use of coagulation testing is readily available in veterinary medicine, and is beneficial in the diagnostic workup (eg, long-acting anticoagulant toxicosis, evidence of DIC, response of heparin therapy) in the critically ill patient.

CENTRAL VENOUS PRESSURE

CVP, also known as right atrial pressure (RAP), describes the pressure of blood in the thoracic vena cava. The CVP reflects the amount of blood returning to the heart and the ability of the heart to pump the blood into the arterial system (ie, preload). The most common access point for a central venous catheter is the external jugular vein. A peripherally inserted central line (PICC) may also be used for CVP measurement; this can be done by placing the PICC into the medial (cat) or lateral (dog) saphenous vein. Note, however, that this can only be performed when severe intra-abdominal hypertension is not present. Moreover, accurate CVP measurements can only be evaluated when the PICC line is placed to the level of the abdomen, as close as possible to the heart, for an accurate approximation of RAP.

The use of CVP is an important, simple diagnostic monitoring tool to help guide and monitor fluid therapy. It is of particular benefit in a potentially volume overloaded, azotemic, anuric patient with cardiac disease. CVP is influenced by blood volume, venous tone and compliance, cardiac function, and intrathoracic pressure (eg, CVP measurements are inaccurate when the patient is receiving continuous positive pressure ventilation). CVP can be measured using an electronic pressure transducer or extension tubing and manometer. The normal CVP ranges between 0 and 5 cm H2O or 0 to 10 mm Hg.

A significantly elevated CVP (eg, >15 cm H2O) is suggestive of cardiac tamponade, right-sided heart failure, or potentially abnormal catheter placement. In general, caution should be used when evaluating a single CVP measurement, which does not provide as much useful information as serial measurements. Repeated measurements of CVP are valuable for establishing trends when monitoring fluid therapy, as it provides important information about the cardiovascular status. As intravenous fluids are administered the intravascular volume expands, thus both venous return and CVP will increase. When a fluid challenge is given to the patient, a euvoletic patient with normal cardiac function may demonstrate a small transient increase in CVP (2–4 mm Hg), which returns to baseline within 15 minutes. If there is an increase of less than 2 to 4 mm Hg or no increase at all in CVP, this would indicate a reduced vascular volume (potentially warranting more aggressive fluid therapy). If a fluid challenge is given and there is a large increase in CVP (eg, >4 mm Hg), the concern would be for either reduced cardiac compliance or increased venous blood volume (or both).

CAPNOGRAPHY

Capnography measures end-tidal carbon dioxide (ETCO2) concentration in exhaled gases, estimating alveolar carbon dioxide concentration. Capnometers use the absorption of infrared light at a specific wavelength projected through the gas mixture
to determine the amount of carbon dioxide present. Carbon dioxide readily diffuses across the capillary membrane and quickly equilibrates with alveolar gas, thus \( \text{ETCO}_2 \) closely approximates arterial carbon dioxide and, therefore, ventilation. However, with increased dead-space ventilation (eg, significant ventilation-perfusion mismatch from pulmonary atelectasis or lung consolidation), \( \text{ETCO}_2 \) will be measured as erroneously low. Capnometry may also be used to assess the effectiveness of cardiopulmonary resuscitation and return of spontaneous circulation.\(^{45,46} \) Unfortunately, the limitations of capnography are typically to intubated (eg, heavily sedated) patients. Nevertheless, the use of \( \text{ETCO}_2 \) is of particular use in an intubated, sedated seizure patient to ensure adequate ventilation (eg, a dog excessively sedated secondary to being loaded on anticonvulsant therapy).

**COLLOID OSMOTIC PRESSURE**

Colloid osmotic pressure (COP), otherwise known as oncotic pressure, can be measured and used to guide fluid therapy. The COP is a force created by large plasma proteins within the vascular space that do not move freely across capillaries. Normal COP values are often documented to be 18 to 25 mm Hg.\(^{47} \) A colloid osmometer is used to measure COP. When testing, the patient’s plasma is forced across a membrane on the osmometer. The molecules exert a pressure in the sample chamber, sensed by the transducer, which is then converted to electrical energy and displayed in mm Hg. A low COP is consistent with a hypo-oncotic states (eg, protein-losing nephropathy, protein-losing enteropathy, sepsis, liver failure), and warrants supplementation with synthetic colloids (eg, hydroxyethyl starch) or albumin. See the article “Fluid Therapy for the Emergent Small Animal Patient: Crystalloids, Colloids, and Albumin Products” elsewhere in this issue for additional information.

**SUMMARY**

Management of the emergent or critically ill patient requires early recognition of life-threatening disturbances (eg, ABCDs) followed by serial patient assessment and monitoring. The clinician should be ready to intervene when life-threatening changes are documented, to prevent patient compromise. Emphasis on serial patient examination and assessment, accompanied by appropriate ancillary testing, is the best way to improve outcomes in emergent patients.

**REFERENCES**