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# Triage and Initial Stabilization of the Emergency Small Animal Surgical Patient

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## Introduction

One of the most challenging aspects of emergency medicine is being presented with patients who have a variety of clinical signs and disease severities to assess, prioritize, stabilize, and provide with definitive care. The veterinary clinician is reliant on information provided by the client, their physical assessment, and initial diagnostics to determine severity of illness and injury, and therefore, urgency of care. Efficient identification and treatment of respiratory, cardiovascular, urinary, and neurologic derangements is essential for successful patient outcomes. When emergency surgical intervention is required, it is crucial to appropriately stabilize the patient for anesthesia without unduly delaying surgical care.

## Triage and Initial Assessment

Triage is an essential tool in the setting of emergency medicine to assess and prioritize critically ill patients [1, 2]. This is particularly true of patients that may require emergency surgical intervention, as the time to provide appropriate stabilizing care and definitive surgical therapy likely impacts patient outcome.

In many veterinary hospitals, nurses obtain pertinent historical information and perform a basic assessment to determine whether the patient needs immediate further evaluation or is stable enough to be seen in turn. In general, over-triage is preferred to under-triage in veterinary medicine, as the severity of signs presented by the patient and observed by the pet owner may not be fully appreciated by untrained individuals. Triage and training systems in patient assessment are used routinely in human emergency medicine. A variety of triage systems exist for human patients, which when combined with education of the medical staff on the system's guidelines for prioritizing medical care, reduces inconsistencies in decision-making [3]. In veterinary medicine, no uniformly accepted triage system exists. Veterinary healthcare professionals therefore use historical information and intuition to make rapid decisions regarding the need for immediate care and order in which patients will be seen. The animal trauma triage (ATT) score was developed retrospectively and assessed prospectively to help to classify and prognosticate for a heterogenous patient group. For each patient, six categories are assessed (perfusion, cardiac, respiratory, skeletal, neurologic, and eye/muscle/integument) and scored from 0 to 3, with 0 being unaffected or only slightly affected to 3 indicating severe injury. The six scores are added together with a maximum possible score of 18. In both the retrospective and prospective populations, the mean ATT score of survivors was significantly lower than non-survivors and for each one-point increase in ATT, the likelihood of survival decreased 2.3–2.6 times [4]. Another veterinary triage system, adapted from the Manchester triage system, uses a five-category system using color-coding to indicate urgency. Examples of "red" emergencies (those which need to be seen immediately) include severe respiratory distress, decompensated shock, life-threatening hemorrhage, and active seizures. Very urgent emergencies, including moderate respiratory distress, evidence of aortic thromboembolism, and urethral obstruction, were classified as "orange." Urgent emergencies, such as mild hemorrhage, moderate dehydration and open fracture were classified as "yellow," while non-urgent disease processes such as localized inflammation, soft-tissue swelling, stranguria and recent isolated seizure were classified as "green" [5]. The study determined that the use of a veterinary triage list by nurses upon triage corresponded better to retrospectively reviewed patient status than when individual

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Companion website: www.wiley.com/go/aronson/surgical

judgment and intuition upon triage were employed [5]. Multicenter, prospective evaluation of this veterinary triage list is warranted to determine whether patient care is improved and time to be seen can be better estimated.

Irrespective of the need for a formal veterinary triage system, a brief, but thorough physical exam remains the gold standard for recognizing critical patient status. The initial triage assessment includes visual examination and assessment of four key body systems: cardiovascular, respiratory, neurologic, and urinary [6, 7]. Information regarding the patient's mentation and responsiveness, as well as respiratory rate and effort, are obtained quickly on brief visual exam, often before performing any parts of a physical examination. Thoracic and cardiac auscultation with concurrent pulse palpation and a more thorough assessment of neurologic status, if indicated, follows visual examination. After cardiovascular, respiratory, and neurologic status is determined, if the patient is stable enough for further evaluation, urinary triage can be performed. Any significant pain must be addressed urgently to improve patient comfort and so that the effects of pain do not alter interpretation of cardiovascular and respiratory findings. Additionally, aggression should not be considered a sign of patient stability, as many scared and stressed patients will be aggressive in the face of severe shock.

## Respiratory Assessment

Before any physical examination, all patients should have their respiratory rate, effort, noise, and pattern observed from afar. If a patient is showing any changes in respiratory pattern or effort, oxygen supplementation should be provided immediately. If there is any respiratory compromise, the patient should be presumed to be in hypoxemic shock until proven otherwise and oxygen supplementation should be provided. Further respiratory triage involves auscultation of the upper airway, trachea, and thorax. During abbreviated thoracic auscultation, emphasis should be placed on determining heart rate, rhythm, the presence of murmur(s) or arrhythmia(s), and lung sounds in all lung fields. Visual and auditory assessment of respiratory pattern and noise combined with thoracic auscultation should help localize the anatomic origin of the respiratory distress. The impact of pain, stress, and anxiety on respiratory rate and effort should not be underestimated (Figure 1.1).

If respiratory noise is localized to the upper airway, diseases associated with an upper airway obstruction, including laryngeal paralysis, laryngeal collapse, brachycephalic airway disease, tracheal collapse, the presence of a tracheal, laryngeal, or pharyngeal mass, and the presence of a foreign body should be ruled out. See Chapter 28 for stabilization of the patient with upper airway obstruction.

**Figure 1.1** Cat with open mouth breathing secondary to the pain associated with an aortic thromboembolism.

If decreased lung sounds relative to respiratory effort are heard dorsally, pneumothorax should be suspected. However, if decreased lung sounds are heard ventrally, pleural effusion should be considered. Lung sounds may not be completely absent in the presence of pleural effusion or pneumothorax, they may be reduced relative to the other lung fields but still present. See Chapter 34 for stabilization of the patient with pleural space disease. Diseases of the chest wall such as masses, rib fractures, and flail segments may also result in abnormal auscultation of the pulmonary parenchyma. For more information on chest wall disease, see Chapter 41.

For patients in respiratory distress with a heart murmur, arrhythmia, pleural effusion, or pulmonary crackles present on auscultation, cardiogenic and hypoxemic shock should be considered as possible differentials. This is particularly important since fluid therapy is often contraindicated in most patients with cardiac dysfunction or failure and must be ruled out, to the best of the clinician's ability on triage, prior to administering intravenous (IV) fluid therapy.

### Hypoxemic Shock

Hypoxemic shock occurs secondary to decreased arterial blood oxygen content. Common causes of hypoxemic shock include pulmonary parenchymal disease, such as pneumonia, severe anemia, and hypoventilation (Figure 1.2). Many veterinary patients in hypoxemic shock are at the limits of their physiologic reserves, and are intolerant of excessive handling, restraint, and manipulation; they should be handled carefully. Clinical signs include weakness, mental

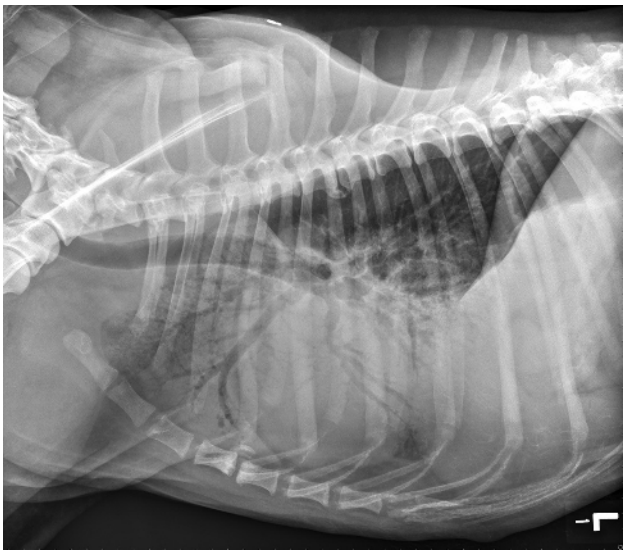


**Figure 1.2** Lateral thoracic radiograph showing cranioventral pulmonary infiltrates creating an alveolar pattern consistent with aspiration pneumonia.

depression, pale mucous membranes, dyspnea, crackles or increased bronchovesicular lung sounds, or decreased lung sounds ventrally (pleural effusion) or dorsally (pneumothorax), and cyanosis. Patients with diaphragmatic hernia may have decreased lung sounds dorsally or ventrally. Cyanosis is only seen with severe hypoxemia (at least 5 g/dl of deoxygenated hemoglobin), and thus the absence of cyanosis absolutely does not rule out hypoxemia. In anemic animals, cyanosis is unlikely to be detected due to decreased hemoglobin concentration, and therefore should not be relied upon to diagnose hypoxemia [8].

In any patient with suspected hypoxemic shock, supplemental oxygen should be provided until the ability to adequately oxygenate is confirmed. Diagnostics that can be helpful for the patient in hypoxemic shock include pulse oximetry (peripheral capillary oxygen saturation, $SpO_2$), arterial blood gas analysis, thoracic radiographs, thoracic/trauma computed tomography (CT), and thoracic ultrasound. $SpO_2$ may be less effective with bright lighting, poor perfusion, high motion, and pigmentation of the skin. It is convenient since it can noninvasively determine percentage of oxygenated hemoglobin, and, for sedentary patients, can be left in place for continuous monitoring (Figure 1.3). Many patients in respiratory distress will not tolerate the restraint necessary for arterial blood gas collection and thoracic radiographs, especially on presentation. If obtaining an arterial blood gas is feasible, findings may include decreased $SpO_2$, decreased partial pressure of carbon dioxide ($PaCO_2$) consistent with hyperventilation, increased $PaCO_2$ consistent with hypoventilation, decreased partial pressure of oxygen

**Figure 1.3** Continuous pulse oximetry assessment in a laterally recumbent dog receiving oxygen supplementation via nasal prongs.

($PaO_2$) consistent with hypoxemia, and an increased partial pressure of alveolar–arterial oxygen gradient $P(A–a)O_2$). Calculation of the $P(A–a)O_2$ gradient provides objective information on pulmonary function by removing the influence of ventilation on $PaO_2$. When a patient is breathing 21% oxygen, the $P(A–a)O_2$ should be less than 10–15 mmHg. When a patient is breathing 100% oxygen, the $P(A–a)O_2$ should be less than 150 mmHg. If the $P(A–a)O_2$ gradient is greater than 15 mmHg while breathing 21% oxygen, it is consistent with pulmonary dysfunction. For A–a gradient calculation, see the formula in Box 1.1.

Preliminary evaluation of the ratio of $SpO_2$ to fraction of inspired oxygen ($F_iO_2$) to the partial pressure of oxygen in arterial blood to $FiO_2$ ($P_aO_2/FiO_2$) showed good correlation between the two values in dogs. It is possible that with further investigation, the $SpO_2/FiO_2$ may become a reliable, less invasive alternative to determining $P_aO_2/FiO_2$ [9].

Thoracic radiographs may show pulmonary parenchymal infiltrates ventrally consistent with pneumonia, caudodorsally consistent with non-cardiogenic pulmonary edema, and in the perihilar region consistent with congestive heart failure. In the trauma patient,

**Box 1.1 Formula Used for A–a Gradient Calculation**

The partial pressure of alveolar–arterial oxygen ($P(A–a)O_2$) gradient provides objective information on pulmonary function by removing the influence of ventilation on $PaO_2$.

$$P(A\text{-}a)O_2 = P_AO_2 - P_aO_2$$

$$P_AO_2 = (F_iO2 \times (P_{atm} - P_{H2O})) - (P_aCO_2 / R)$$

$P_AO_2$ = alveolar gas equation

$F_iO_2$ = percentage of inspired $O_2$

$P_{atm}$ = atmospheric pressure (760 mmHg used at sea level)

$P_{H_2O}$ = water vapor pressure (53 mmHg 39°C for dogs/cats; 47 mmHg at 37°C in humans)

R = respiratory quotient (approximately 0.8–0.9)

**Example Blood Gas**

$PaCO_2$ = 24.2

$PaO_2$ = 59.5

$P_AO_2$ = (0.21 × (760 – 53)) – (24.2/0.9)

$P_AO_2$ = 121.6

$P(A–a)O_2$ = 121.6–59.5

$P(A–a)O_2$ = 62.1 (indicates hypoxemia is due to pulmonary dysfunction)

pulmonary contusions, which can be present in any lung field(s), may not become radiographically apparent for up to 48 hours, although peak opacification has been shown to occur at 6 hours in human trauma patients [10]. Additionally, up to 30% of human trauma patients do not have radiographic evidence of contusions on initial thoracic radiographs, which is why CT is often proposed as the preferred method of thoracic imaging [10–12]. In a study of dogs that had succumb to vehicular trauma, thoracic radiographs underestimated the presence of contusions, while also overestimating their severity. The same study also noted that thoracic radiographs were less sensitive than CT for detecting rib fractures [13]. Initial investigation in the use of thoracic ultrasound for detection of pulmonary contusions in dogs with vehicular trauma showed a high sensitivity for diagnosing contusions compared with CT, and even noted improved sensitivity compared with thoracic radiographs [14]. Therefore, cautious respiratory monitoring and repeat thoracic imaging may be indicated in any patient with a history of known or suspected trauma.

Thoracic ultrasound, also known as thoracic focused assessment with sonography for trauma, triage, and tracking (TFAST), allows clinicians to assess for pleural and pericardial effusion, pneumothorax, and pulmonary parenchymal infiltrates [15–20]. It is particularly useful in patients that are not stable enough for thoracic radiographs, as well as a monitoring tool to assess for response to therapy. Thoracic ultrasound may be performed with the patient in sternal or lateral recumbency. Pleural effusion is generally visible in the cranial and/or caudoventral pleural space. Ultrasound guidance to localized fluid pockets can be helpful to guide thoracocentesis. When evaluating for the presence of pneumothorax, the caudodorsal thorax is evaluated for the lack of a "glide" sign, which is diagnostic for pneumothorax. A glide sign is created by the normal back and forth respiratory motion of the interface between the visceral and parietal pleura (Video 1.1). Free air in the thoracic cavity obliterates the glide sign [15–17]. Cellular or fluid infiltrate into the pulmonary parenchyma, as with edema, hemorrhage, and pneumonia can be assessed using ultrasound in four windows in each hemithorax (caudodorsal, cranial, middle lung lobe regions, and perihilar) for the presence of increased penetration of ultrasound, which manifest as hyperechoic lines (B-lines) in parallel with the ultrasound beam, that can be individual or coalescing (Figure 1.4 and Video 1.2) [18–22].

**Video 1.1** TFAST showing a normal glide sign, which is created by the respiratory motion of the visceral and parietal pleural interface sliding back and forth.


**Video 1.2** TFAST showing coalescing B-lines created by marked pulmonary infiltrates allowing ultrasound penetration into the pulmonary parenchyma.

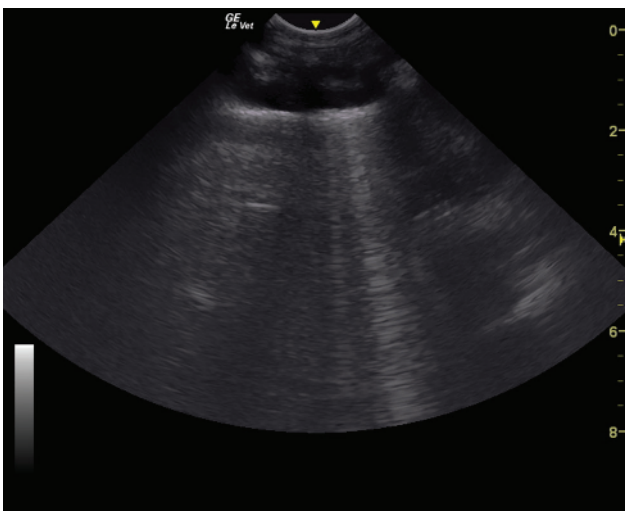




**Figure 1.4** TFAST ultrasonographic appearance (still image) of a B-line, which is created by increased infiltrates in the pulmonary parenchyma allowing ultrasound penetration.

## Cardiovascular Assessment

The most important part of the cardiovascular assessment during emergency patient triage is the determination whether the patient is in shock. If shock is suspected, the type of shock and need for fluid therapy must then be determined. The common feature in all shock patients is inadequate cellular energy metabolism, which is most commonly due to poor perfusion. However, metabolic and hypoxic shock can occur with normal perfusion, so one must be careful to not rule out shock on the basis of normal perfusion parameters alone [23, 24].

For cardiovascular triage, mucous membrane color, temperature, and capillary refill time (CRT) can be used to assess perfusion. Signs of poor perfusion during mucous membrane assessment include pale pink to white mucous membranes, cool temperature, and prolonged to absent CRT (> 2 seconds). Bright pink or red mucous membranes, injected capillaries, and rapid CRT can be seen with distributive shock. However, depending on the patient's stage of cardiovascular compromise and the degree of compensation, even patients with shock can have normal mucous membrane appearance. Heart rate and rhythm should be assessed simultaneously with pulse palpation to determine pulse pressure quality and for deficits. Both femoral and dorsal metatarsal artery palpation is preferred to appreciate discrepancies in proximal and distal perfusion. Extremity temperature on limb palpation and rectal temperature should be noted to complete the patient's perfusion clinical picture [23–26].

Following physical examination of the cardiovascular system, emergency diagnostic tools that can aid cardiovascular triage include indirect blood pressure, electrocardiogram (ECG), venous or arterial blood gas, packed cell volume/total solids (PCV/TS), lactate, and left atrial to aortic root ratio on ultrasound. Indirect blood pressure methods, such as Doppler or oscillometric technologies, provide rapid noninvasive determination of arterial blood pressure. Doppler is particularly useful in small patients, cats, and those with cardiac arrhythmias. Oscillometric methods are convenient as they can be programmed to cycle at predetermined intervals, such that repeated measurements can be obtained automatically. For both methods, cuff size selection in relation to limb diameter is essential for accurate results. Cuff diameter should be approximately 40% of the limb circumference in dogs and 30% in cats. Cuffs that are too large will generate falsely low blood pressure results, and falsely high results will be obtained from a cuff that is too small [27]. In hypotensive patients, noninvasive methods have been shown to have the greatest variability compared with direct measurements [28]. Direct arterial blood pressure is considered the gold standard for blood pressure determination, and offers the additional benefits of continuous, real-time results that are accurate with arrhythmias and decreased perfusion. However, placement of an arterial catheter is technically challenging, especially in distressed or hypotensive patients and cats, uncomfortable for the patient during placement, and requires constant monitoring to ensure the catheter is not inadvertently dislodged. ECG is helpful to evaluate for the presence of cardiac arrhythmias, which can be the primary cause of shock (cardiogenic), or secondary complications of hypovolemic, metabolic, hypoxic, or distributive shock. Venous blood gas monitoring, particularly for pH, partial pressure of carbon dioxide in venous blood ($PvCO_2$), partial pressure of oxygen in venous blood ($PvO_2$), electrolytes, and base excess/deficit is important to help determine the underlying cause of cardiovascular compromise, assess cellular oxygen delivery and metabolism, and response to therapy. Similarly, PCV/TS are essential to evaluate for blood and/or protein loss, dehydration, and appropriate hemodilution response if fluid therapy is used. Lactate can be a marker of anaerobic metabolism and is often increased in shock patients (type A lactic acidosis), although less reliably in cats. It can support clinical assessment of poor perfusion and trended over time with treatment of the primary cardiovascular disturbance. It has been associated with outcome in gastric dilatation and volvulus, pyometra, and immune-mediated hemolytic anemia [29–35]. It is important to remember that type B lactic acidosis, which is hyperlactatemia in the face of normal perfusion, does not resolve with fluid therapy. Causes of type B lactic acidosis include liver failure, neoplasia (especially hematopoietic), diabetes mellitus, sepsis/systemic inflammatory response syndrome, and various drugs and toxins [30].

Comparing the left atrium diameter with the root of the aorta (LA : Ao), when using a short axis view from the right parasternum, is helpful to assess left atrial volume. The LA : Ao ratio was originally developed to support a diagnosis of congestive heart failure, but it can also be used to evaluate for left atrial volume underload, which can occur with hypovolemic shock. In dogs, the normal LA : Ao ratio is 1.3, whereas the ratio is 1.5 in cats [36, 37]. Baseline LA:Ao ratio on triage can help support a diagnosis of cardiogenic shock secondary to congestive heart failure if the LA : Ao ratio is increased. When the ratio is decreased, hypovolemic shock may be present, especially if found in conjunction with other parameters that support hypoperfusion. Changes in the ratio with treatment, whether intravenous fluids or diuretics, can be useful for monitoring response to therapy.

The use of point of care ultrasound techniques in dogs has been used to objectively assess vascular status and may be used as an adjunct diagnostic to guide fluid and/or vasopressor therapy. In normal dogs with furosemide induced hypovolemia, the caudal vena cava to aorta ratio measured from the right intercostal space in left lateral recumbency corresponded with decreasing body weight consistent with volume loss [38]. This ratio was also decreased after blood donation in healthy dogs [39]. In greyhounds, the iliac location for this ratio did not detect volume status changes before and after blood donation [40]. While additional work is needed in canine clinical cases of hypovolemia as well as normal and critically ill cats, this technique appears to be a promising method for non-invasive volume assessment.

## Hypovolemic Shock and Fluid Therapy

Hypovolemic shock, which is the most common type of shock seen in veterinary medicine, can be due to blood loss, fluid loss, or inadequate intake, and results in decreased tissue delivery of oxygen. Clinical signs consistent with hypovolemic shock are mental depression, weakness, pale mucous membranes, prolonged CRT, tachycardia, weak peripheral pulses, cool extremities, and tachypnea.

To treat hypovolemic shock, intravascular volume replacement is essential and generally accomplished with intravenous crystalloids, colloids, blood products, or a combination of the fluid replacement options. "Shock" doses of fluid therapy are based on the blood volume for a given species, and amounts for replacement are based on the percentages of volume loss to cause cardiovascular changes secondary to hypovolemic shock. Blood volume is approximately 90 ml/kg in dogs and 45–60 ml/kg in cats. Generally, patients are given portions of their shock dose of fluids, such as 10–30 ml/kg of balanced isotonic crystalloid solutions or 5–10 ml/kg of colloid solutions as a bolus over 15–20 minutes and assessed for improvement in perfusion parameters. The bolus is repeated if indicated. Hypertonic saline (7.5%, 3–5 ml/kg IV over 15–20 minutes in dogs, 2–3 ml/kg IV over 15–20 minutes in cats) is also effective for rapid volume expansion in hypovolemic shock but should only be used in patients with normal hydration. The decision about whether crystalloids or colloids should be chosen as the initial resuscitation fluid is controversial and has yet to be determined in both human and veterinary medicine [41–47]. In veterinary patients, the decision is often based on availability, cost, and whether there are concerns about the patient's colloid osmotic pressure and the ability to maintain fluid within the intravascular space. In June 2013, a boxed warning was placed on hydroxyethyl starch (HES) solutions, such as hetastarch, due to concerns for increased mortality, severe renal injury, and risk of bleeding associated with their use in critically ill adults, including those with sepsis and admitted to the intensive care unit. VetStarch® (Abbott Laboratories, Chicago, IL), a veterinary specific HES solution, is commercially available as a synthetic colloid for plasma volume expansion. Preliminary veterinary studies have conflicting evidence on association between the use of synthetic colloids and acute kidney injury in dogs; they should be used with caution until further research is available [48, 49].

Hypovolemic resuscitation or controlled intravascular volume replacement titrated to a mean arterial blood pressure (MAP) of 60 mmHg is widely used for human and veterinary patients with hemorrhagic shock [50–54]. The goal is to preserve perfusion to the vital organs, particularly the kidneys and cerebral circulation, without supranormalizing blood pressure, to prevent disruption of any clots tempering further hemorrhage. Experimental evidence in a swine model shows that rebleeding occurs when MAP is greater than 60 mmHg, while maintaining the MAP at approximately 60 mmHg maintains renal and cerebral blood flow [54]. Recommendations for decreased volume fluid resuscitation for crystalloid boluses are between 20 and 30 ml/kg and 5 ml/kg for colloid boluses titrated to effect and target blood pressure [52].

Transfusion with packed red blood cells (pRBC; 5–10 ml/kg), fresh frozen plasma (FFP; 10–20 ml/kg), or whole blood (10–20 ml/kg) may be indicated for patients with anemia and/or coagulopathy. While there is no absolute PCV below which a transfusion is required, consideration of the chronicity of anemia, cardiovascular stability, continuing losses, anticipated surgical intervention, and pulmonary function all impact the decision of whether or not to transfuse a patient. It is also important to remember, that in many critically ill patients, even after control of hemorrhage, coagulopathy may persist due to dilution, consumption, delayed liver production of clotting factors, and liver dysfunction, so repeated dosing of FFP may be needed even once coagulation parameters have normalized.

Regardless of the fluid type chosen for cardiovascular resuscitation, it is imperative that frequent reassessment of the patient's cardiovascular parameters in response to treatment be performed. That same physical exam parameters and initial diagnostics used to diagnose shock should be reevaluated. Additional diagnostics that may be helpful for determining whether a patient is appropriately or maximally fluid resuscitated, especially if shock persists, include central venous pressure (CVP) and central venous oxygen saturation ($S_{CV}O_2$). CVP, which is a measure of the hydrostatic pressure within the intrathoracic (cranial or caudal) vena cava, is used to approximate right atrial pressure, or preload.

Normal CVP is 0–5 cm $H_2O$. CVP values less than 0 cm $H_2O$ are consistent with hypovolemia or decreased venous tone secondary to vasodilation [55]. Increased CVP (> 7–10 cm $H_2O$) can be seen with volume overload, pleural space disease (pneumothorax, pleural effusion), pericardial disease (restrictive pericarditis, pericardial effusion), tricuspid valve disease, myocardial disease, and intraabdominal hypertension [55, 56]. The value of CVP monitoring for guiding fluid resuscitation has been questioned in both human and veterinary critical care in recent years. In addition to CVP, central catheters can also be used to measure $S_{CV}O_2$, which is an assessment of global tissue oxygenation and is the percentage of saturated hemoglobin within the cranial or caudal vena cava or right atrium. Alterations in $S_{CV}O_2$ reflects imbalance between oxygen delivery and consumption. Decreased $S_{CV}O_2$ is seen with increased oxygen consumption relative to delivery, as with hypovolemia, anemia, cardiac dysfunction, pulmonary dysfunction, fever, and hyperthermia. Increased $S_{CV}O_2$ is seen with decreased oxygen consumption relative to delivery, as with hypothermia and mitochondrial dysfunction [57–59]. In critically ill dogs, a decrease in $S_{CV}O_2$ below 68% within the first 24 hours of hospitalization was associated with poor outcome with progressive increase in mortality with decrease in $S_{CV}O_2$ [59]. In septic dogs that underwent surgery for pyometra, survivors had lower lactate, base deficit and their average $S_{CV}O_2$ was 74.6%. Non-survivors in this study had an average $S_{CV}O_2$ of 62.4% [33]. Co-oximetry, which is not widely available, is needed for $S_{CV}O_2$ determination; this is likely the reason for its limited clinical use in veterinary patients.

## Cardiogenic Shock

It is important to differentiate hypovolemic shock from cardiogenic shock, as many of the physical exam findings can overlap but the treatment is usually vastly different. Fluid therapy is generally contraindicated in most patients with cardiogenic shock. Cardiogenic shock can be due to forward (left-sided) or backward (right-sided) failure of blood flow. Common causes of cardiogenic shock include congestive heart failure, systolic dysfunction, as with dilated cardiomyopathy, diastolic dysfunction, as with hypertrophic cardiomyopathy, and arrhythmias [25, 26]. Clinical signs of cardiogenic shock include pale mucous membranes, heart murmur and/or arrhythmias, poor or variable pulse quality, pulse deficits, and tachycardia or bradycardia. Findings consistent with right-sided heart failure include decreased ventral lung sounds consistent with pleural effusion, jugular venous distension, ascites, and hepatomegaly. Clinical signs seen with left-sided dysfunction and left-sided heart failure include increased respiratory rate or effort, respiratory distress, pulmonary crackles (pulmonary edema), and decreased lung sounds ventrally consistent with pleural effusion (cats).

In addition to history and physical examination findings, other diagnostics often needed to diagnose cardiogenic shock include ECG, blood pressure, pulse oximetry ($SpO_2$), thoracic radiography, and TFAST. TFAST can be used to determine cardiac contractility, myocardial thickness, and cardiac chamber (atria and ventricle) size. Focused echocardiography training for emergency veterinarians has been shown to improve their diagnostic capabilities for determination of several cardiac abnormalities [59]. Treatment may involve oxygen supplementation, pericardiocentesis, diuretic therapy, antiarrhythmics, vasopressors, or vasodilators depending on the etiology of cardiogenic shock.

## Distributive/Septic Shock

Distributive shock is defined as a maldistribution of blood flow, most commonly due to altered systemic vascular resistance (SVR). Decreased SVR is the most common SVR alteration, and vasodilatory shock secondary to sepsis is one of the most readily recognized forms of distributive shock. Distributive shock can also be secondary to obstructive disease processes, such as gastric dilation and volvulus, pericardial effusion, and neoplasia causing vascular obstruction (such as adrenal tumors with invasion into the vena cava) [25]. The clinical signs of distributive shock in dogs are often very different from other forms of shock. In dogs, the mucous membranes are often bright pink (Figure 1.5), CRT is decreased (< 2 seconds), and peripheral pulses can be bounding or more prominent than normal. Cats with septic shock generally do not demonstrate the hyperdynamic signs seen in dogs and instead have pale mucous membranes, bradycardia, and decreased rectal temperature [60].

Many patients with distributive shock also have a component of hypovolemic shock (absolute or relative), so fluid therapy to correct intravascular volume deficit is essential. In humans with severe sepsis and septic shock, early goal-directed therapy is shown to improve patient outcome when compared to traditional management strategies. In two landmark human studies, hemodynamic parameters such as direct arterial blood pressure, CVP, and $S_{CV}O2$ measurement, and treatment with crystalloids, colloids, pRBC, and catecholamines to improve cardiac contractility and/or vasomotor tone were used until prescribed endpoints were achieved [61, 62]. Standardized goal-directed therapy does not yet exist for veterinary patients, therefore, normalization of routinely monitored cardiovascular and perfusion parameters, including heart rate and

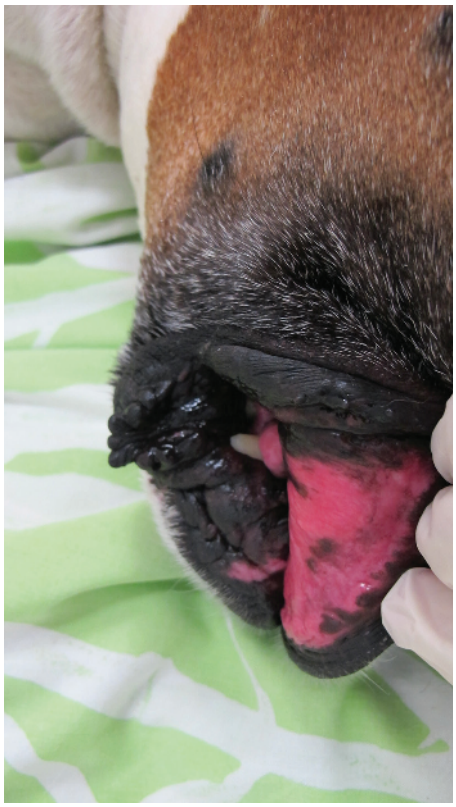


**Figure 1.5** Bright pink mucous membranes in a dog with septic peritonitis.

rhythm, rectal temperature, mucous membrane color and CRT, blood pressure, CVP, PCV/TS, lactate and base deficit are recommended [31, 63, 64].

When fluid therapy fails to normalize hemodynamic parameters, particularly blood pressure in septic patients, vasoactive catecholamines may be necessary [65, 66]. Commonly used vasoactive catecholamines used in critical care are dopamine, dobutamine, and norepinephrine (Table 1.1). Vasopressin, also known as antidiuretic hormone, is a peptide synthesized in the pituitary that binds vasopressin specific receptors on vascular smooth muscle. Vasopressin stores can become depleted with prolonged shock or sepsis resulting in vasoplegia despite intravenous fluid and vasoactive catecholamine therapy. Vasopressin deficiency has been documented in people with refractory hypotension, and positive benefit has been shown with the addition of intravenous administration of vasopressin. Experience with vasopressin is growing in veterinary medicine [67, 68].

Early administration of broad-spectrum antibiotics has been shown to improve survival in human patients with sepsis and septic shock when combined with early goal-directed therapy. When antibiotics were given within one hour of triage in combination with early goal-directed therapy, mortality decreased from 33.3% to 19.5% [69]. In veterinary patients with septic shock, antimicrobials should be given as soon as reasonably possible, especially for those that will undergo emergency anesthesia and surgery. In critically ill septic patients anticipated to undergo surgery, perioperative first- and second-generation cephalosporins likely do not provide adequate antimicrobial coverage and should not be used in favor of more broad-spectrum medications. In the absence of confirmatory culture and sensitivity testing, broad-spectrum therapy should be used until a diagnostic culture result is obtained and antimicrobial therapy can be de-escalated. Intravenous administration is preferred in all cardiovascularly unstable and critically ill patients as oral, intramuscular, and subcutaneous absorption may not be predictable. Antibiotics that can be considered for first-line broad-spectrum therapy include ampicillin and clavulanate (Unasyn®, Pfizer, 22–30 mg/kg IV every 8 hours); ampicillin (18–22 mg/kg IV every 8 hours) combined with enrofloxacin (10–15 mg/kg IV every 24 hours), cefoxitin (30 mg/kg IV every 6 hours), and clindamycin (10 mg/kg IV every 12 hours) combined with cefotaxime (40–50 mg/kg IV every 6 hours), or ceftazidime (30–50 mg/kg IV every 6–8 hours with dosing at the lower end of the range for cats and higher end for dogs).

### Metabolic Shock

Metabolic shock is defined as dysfunction of cellular metabolism, which generally occurs in the face of adequate perfusion and oxygenation. Examples include severe pH derangements, hypoglycemia, adrenal insufficiency, and certain toxicities such as cyanide. The clinical signs seen with metabolic shock closely resemble those in hypovolemic shock and are related to the underlying etiology. Mental depression is the most universally recognized sign of metabolic shock. The same diagnostics used for patients in hypovolemic shock are indicated for the patient in metabolic shock. Treatment may include correction of acid–base derangements with IV fluids and/or bicarbonate, dextrose supplementation, and steroid administration, if adrenal dysfunction is demonstrated or highly suspected [70–74].

## Dehydration

The management of hypovolemic shock focuses on restoration of intravascular volume for improvement in cardiovascular function, rather than normalization of hydration. Dehydration is a reflection of interstitial fluid balance, and while imperative to assess, treat, and monitor for improvement, treating hypovolemic shock must take

**Table 1.1** Commonly used vasopressors used in the emergency room and intensive care unit.

| | CRI dose | Effect | Additional information |
|---|---|---|---|
| Dopamine | 1–4 μg/kg/minute | Vasodilation (renal) | Mixed data for renal effects |
| | 5–10 μg/kg/minute | Increased contractility, some vasoconstriction | |
| | 10–20 μg/kg/minute | Vasoconstriction, variable contractility effects | |
| Dobutamine | 2–20 μg/kg/minute (dogs) | Increased contractility, little vasoconstriction | |
| | 2–5 μg/kg/minute (cats) | Increased contractility, little vasoconstriction | Can cause seizures in cats |
| Norepinephrine | 0.05–2 μg/kg/minute | Potent vasoconstriction | |
| Vasopressin | 0.5–2 mU/kg/minute (dogs) | Potent vasoconstriction (even in acidosis) | Limited clinical experience in dogs, no dose established for cats |

*Source:* Adapted from Simmons and Wohl [65]. CRI = constant rate infusion.

priority. Interstitial fluid losses are generally gradual and are therefore corrected over time. Prolonged or severe dehydration can lead to hypovolemic shock. The percentage of dehydration (5–12%) is estimate based on physical exam (skin turgor, sunken eyes, urine output) and objective criteria, such as PCV/TS, loss of body weight, and urine specific gravity. The fluid deficit is determined using the percentage of dehydration and the patient's lean body weight. Estimation of lean body weight is imperative when determining the fluid prescription for obese patients, especially cats, as significant overhydration can result if overweight or obese body weight is used. The deficit is then corrected over a period of 12–48 hours depending on chronicity, patient's tolerance to fluid therapy, maintenance fluid needs, and any continuing fluid losses (Box 1.2). In small animal patients, maintenance fluid rates are generally 2–3 ml/kg/hour in dogs and 1–2 ml/kg/hour in cats [75].

Electrolyte monitoring should be performed routinely (Table 1.2) in patients with dehydration and shock. This is

**Box 1.2 Fluid Therapy Prescription Formula**

This formula incorporates fluid deficit (dehydration), ongoing losses, and maintenance fluid needs. It should be used only after intravascular volume deficits (hypovolemic shock) have been corrected.

$$\text{Fluid prescription} = \text{fluid deficit}(\text{liters}) + \text{continuing losses}(\text{including insensible losses}) + \text{maintenance fluid needs}$$

$$\text{Fluid deficit}(\text{liters}) = \%\text{dehydration} \times \text{lean body weight}(\text{kg})$$

Rate of deficit correction is generally over 12–36 hours depending on patient stability, chronicity of dehydration, and tolerance for IV fluids.

Maintenance needs are generally 2–3 ml/kg/hour for dogs and 1–2 ml/kg/hour for cats.

**Example Fluid Prescription Calculation**

25 kg mixed breed dog (lean body condition)
Estimated to be 8% dehydrated based on physical exam findings (tacky mucous membranes, prolonged skin tent, slightly sunken globes, hyperviscous saliva in the corner of the mouth).
No conditions that would make the patient fluid intolerant; plan to correct over 24 hours.
The dog is losing approximately 60 ml in vomit every hour, no excessive gastrointestinal or urinary losses.

Deficit = 0.08 × 25
Deficit = 2000 ml
Rate of deficit correction = 2000/24 = 83 ml/hour
Fluid prescription (per hour) = 83 ml (deficit) + 60 ml (losses) + 50 ml (maintenance)
Fluid prescription = 193 ml/hour

**Table 1.2** Monitoring parameter guidelines and frequencies for dehydrated patients and those in hypovolemic, distributive, and hypoxemic shock.

| | Physical assessment[a] | Blood pressure | $SpO_2$ | Urine output/ specific gravity | PCV/TS/BG/Azo Stick® | VBG/ABG/electrolytes |
|---|---|---|---|---|---|---|
| Dehydration[b] | 8–12 hours | 8–12 hours | 12–24 hours | 8–12 hours | 12–24 hours | 12–24 hours |
| Hypovolemic shock | 1–2 hours initially, then 4–6 hours once stabilized | 1–2 hours initially, then 4–6 hours | 4–6 hours | 4–6 hours | 4–6 hours initially, then 6–8 hours | 4–6 hours initially, then 6–8 hours |
| Distributive shock | 1–2 hours initially, then 4–6 hours once stabilized | 1–2 hours initially, then 4–6 hours | 4–6 hours | 4–6 hours | 6–8 hours | 6–8 hours |
| Hypoxemic shock | 1–2 hours initially, then 4–6 hours once stabilized[c] | 2–6 hours[c] | 1–4 hours[c] | 4–6 hours[c] | 12–24 hours and after pRBC transfusion[c] | 12–24 hours[c] |

[a] Physical assessment parameters include hydration evaluation, mucous membranes, capillary refill time, respiratory rate and effort, cardiac and thoracic auscultation, pulse quality, and temperature.
[b] Dehydrated patients should also be weighed every 8–12 hours.
[c] Frequency of diagnostics will depend on patient stability and amount of stress caused to the patient with handling, evaluation, and blood sampling.
ABG, arterial blood gases; BG, blood glucose; PCV, packed cell volume; SpO2, peripheral capillary oxygen saturation; TS, total solids; VBG, venous blood gases.

**Table 1.3** Analgesics, sedatives, and anxiolytics used in small animal medicine.

| Generic drug | Brand (manufacturer) | Dose | Comment |
|---|---|---|---|
| Opioids: | | | |
| Buprenorphine | Buprenex® (Reckitt & Colman) | 5–20 μg/kg IM, IV q 6–8 hours<br>Cats: 10–20 μg/kg PO q 6–8 hours | μ-partial agonist<br>Excellent oral absorption (cats)<br>Difficult to reverse |
| Butorphanol | Torbutrol®, Torbugesic-SA® (Zoetis) | 0.1–0.4 mg/kg IM, IV q 1-4hours<br>Partial μ reversal: 0.05–0.1 mg/kg IV<br>CRI loading dose: 0.1 mg/kg IV<br>CRI: 0.1–0.4 mg/kg/hours IV | κ-agonists<br>μ-antagonist<br>Variable analgesia<br>Sedative and anti-tussive |
| Fentanyl | Abstral® (Abbott Laboratories) | Dog loading dose: 1–2 μg/kg<br>Dog CRI: 2–5 μg/kg/hours<br>Cat loading dose: 1 μg/kg/hours<br>Cat CRI: 1–4 μg/kg/hours | Can cause SIADH with prolonged use |
| Fentanyl transdermal patch | Duragesic® (Janssen Pharmaceuticals) | Cat or dog < 5 kg: 25 μg patch<br>Dog 5–10 kg: 25 μg patch<br>Dog 10–20 kg: 50 μg patch<br>Dog 20–30 kg: 75 μg patch<br>Dog >30 kg: 100 μg patch | Topical heat can increase absorption<br>Caution for abuse potential/ingestion by children |
| Hydromorphone HCl | | Dog: 0.05–0.2 mg/kg IM, SQ, 0.05–0.1 mg/kg IV every q 4–6 hours<br>Cat: 0.05–0.1 mg/kg IM, S, 0.03–0.05 mg/kg IV every q 3–4 hours | IV administration can cause vomiting |
| Methadone HCl | | Dog: 0.1–0.4 mg/kg IV every q 4–6 hours<br>Dog: 0.2–2 mg/kg SQ, IM every q 4–6 hours<br>Cat: 0.05–0.2 mg/kg IV every q 4–6 hours<br>Cat: 0.1–1 mg/kg SQ, IM every q 4–6 hours | Tends to cause less sedation and vomiting than morphine |
| Morphine (preservative free) | | Dog: 0.25–1 mg/kg IM, SQ every q 4–6 hours<br>Cat: 0.05–0.5 mg/kg IM, SQ every q 4–6 hours<br>Loading dose: 0.15–0.5 mg/kg IV<br>CRI: 0.1–1 mg/kg/hour | IV administration must be done slowly to avoid histamine release, IV administration can cause vomiting |
| Morphine sulfate (with preservative) | | Dog: 0.5–2 mg/kg IM, SQ every q 4 hours<br>Cat: 0.05–0.4 mg/kg IM, SQ every q 3–6 hours | |
| Naloxone | Narcan® (DuPont Pharma) | Opioid reversal: 0.002–0.2 mg/kg IM, IV, SQ | May need to be repeated after 20–30 minutes as required |

(*Continued*)

**Table 1.3** (Continued)

| Generic drug | Brand (manufacturer) | Dose | Comment |
|---|---|---|---|
| Oxymorphone | Numorphan® (Endo Labs) | Dog: 0.02–0.2 mg/kg IV every q 1–4 hours<br>Dog: 0.05–0.2 mg/kg IM, SQ every q 2–6 hours<br>Cat: 0.01–0.05 mg/kg IV every q 2–4 hours | |
| Lidocaine: | | | |
| Lidocaine 1% preservative free | | Dog loading dose: 1–2 mg/kg IV<br>Dog CRI: 20–80 µg/kg/minute | Controversial for IV use in cats |
| NMDA antagonists: | | | |
| Ketamine | KetaFlo® (Abbott Laboratories)<br>Ketaset® (Fort Dodge Animal Health)<br>Vetamine® (Schering-Plough) | Sedation: 2–10 mg/kg IV, IM<br>Loading dose: 0.5–1 mg/kg IV<br>CRI: 0.1–0.6 mg/kg/hour | Caution with hypertension, heart disease<br>Controversial in head trauma, increased ICP/IOP, renal disease (cats) |
| Alpha-2 Adrenergics | | | |
| Dexmedetomidine HCl | Dexdomitor® (Pfizer) | Sedation: 1–10 µg/kg IV, IM<br>Loading dose: 0.5–1 µg/kg IV<br>CRI: 0.25–3 µg/kg/hour | Caution with cardiovascular disease or instability |
| Atipamezole | Antisedan® (Pfizer) | Alpha-2 Adrenergic reversal: 0.05–0.2 mg/kg IV, IM | Same volume as dexmedetomidine given IM |
| Benzodiazepines: | | | |
| Midazolam | | 0.1–0.5 mg/kg IM, IV<br>CRI: 0.1–0.5 mg/kg/hour | |
| Diazepam | | 0.1–0.5 mg/kg IV<br>CRI: 0.1–0.5 mg/kg/hour | Propylene glycol vehicle; avoid prolonged IV use or IM injection |
| Flumazenil | | Benzodiazepine reversal: 0.01–0.02 mg/kg IV | May need to be repeated after 20–30 minutes as required |
| Phenothiazines: | | | |
| Acepromazine | Aceproject® (Fort Dodge Animal Health) | 0.005–0.01 mg/kg IV every 4–6 hours<br>0.01–0.05 mg/kg IM, SQ every 4–6 hours | Caution in hypovolemia<br>Do not exceed 2 mg/kg in large dogs |
| Non-steroidal anti-inflammatory drugs: | | | |
| Carprofen | Rimadyl® (Pfizer) | Dogs: 2–4 mg/kg IV, SQ (single dose)<br>Dogs: 2 mg/kg PO 12 or 4 mg/kg PO once daily24 hours | IV or SQ should only be given when normothermic/normotensive |
| Deracoxib | Deramaxx® (Novartis) | Dogs: 1–2 mg/kg/day | |

| | | | |
|---|---|---|---|
| Meloxicam | Metacam® (Boerhringer Ingelheim)<br>OroCAM® (Abbott Laboratories) | Dogs: 0.1–0.2 mg/kg IV, SQ (single dose)<br>Dogs: 0.1 mg/kg PO or transmucosal once daily | Black box warning for cats<br>Transmucosal oral spray for dogs > 2.5 kg |
| Piroxicam | Feldene® (Pfizer) | Dogs: 0.3 mg/kg PO once daily | |
| Robenacoxib | Onsior® (Novartis) | Dogs: 2 mg/kg SQ 30 minutes before start of surgery then every q 24 hours for a maximum of 3 days<br>Dogs: 1–2 mg/kg PO once daily<br>Cats: 2 mg/kg SQ 30 minutes before start of surgery then every q 24 hours for a maximum of 3 days<br>Cats: 1 mg/kg PO once daily for a maximum of 3 days | Do not divide/break/crush feline tablets, therefore, dose range in cats of 1–2.4 mg/kg<br>Do not use in dogs or cats less 4 months of age<br>Do not use tablets in cats < 2.5 kg |

*Source:* Adapted from Quant and Lee JA [105] and Perkowski [106].
CRI, constant rate infusion; ICP, intracranial pressure; IM, intramuscularly; IOP, intraocular pressure; IV, intravenously; NMDA, N-methyl-d-aspartate; PO, per os (orally); SIADH, syndrome of inappropriate anti-diuretic hormone.

particularly true in anorexic patients or those with renal dysfunction, which may require supplementation with potassium and/or phosphorus. Additionally, as many fluids used in veterinary medicine are designed as "replacement" and not "maintenance" fluids, sodium values may increase in patients receiving prolonged intravenous fluid therapy, particularly in patients with continued free water loss, such as renal, gastrointestinal, skin, and respiratory loss. Fluids with lower sodium concentrations such as Normosol-M, 0.45% NaCl, and dextrose 5% in water (D5W) may be necessary to prevent or manage hypernatremia associated with prolonged fluid therapy and/or concurrent hypotonic fluid losses.

Fluid therapy in the burned veterinary patient requires special consideration, especially with respect to percentage of total body surface area affected. For information on fluid therapy for the burned patient, see Chapter 53.

Regardless of fluid type and rate used to treat shock and or dehydration, frequent patient reassessment is critical. General recommendations for patient re-evaluation are listed in Table 1.2.

## Neurologic Assessment

Initial neurologic assessment often occurs concurrently with respiratory and cardiovascular triage. Patients with normal consciousness are alert and aware of their environment. Obtunded patients have decreased responsiveness that can vary in severity. Stuporous patients are only responsive to noxious or excessive stimuli, whereas comatose patients do not respond to any stimuli. Decreased cerebral perfusion and oxygenation from hypovolemic, hypoxemic, distributive, and cardiogenic shock can have profound effects on mentation, so the patient's initial neurologic assessment must be made with patient's global perfusion status in mind. In both veterinary and human patients with traumatic brain injury, most have also sustained concurrent injuries to other major body systems that can have secondary neurologic consequences [76, 77]. Hypoglycemia (metabolic shock) can also lead to decreased mentation and must be treated before an accurate neurologic examination can be performed. As with other body systems, frequent neurologic reassessment is imperative.

While performing the neurologic evaluation, until adequate oxygenation is confirmed, supplemental oxygen should be provided by mask, flow by, or placement of the patient in an oxygen cage. In patients with head trauma, nasal prongs or nasal oxygen catheter are avoided to decrease the risk of sneezing, which can increase their intracranial pressure (ICP). Heart rate and blood pressure values can also provide important insight about the presence of increased ICP. The Cushing's reflex, which is hypertension and reflex bradycardia, is commonly seen in patients with cerebral edema, hemorrhage, skull fractures, and intracranial masses. To maintain cerebral perfusion pressure (CPP) in the face of intracranial hypertension, arterial blood pressure (MAP) is increased, since CPP equals MAP minus ICP. Pressure receptors in the aortic arch and carotid bodies trigger a decreased heart rate in response to the increase systemic blood pressure. Treatment of intracranial hypertension is imperative and is accomplished with a combination of patient positioning and pharmacologic intervention with mannitol or hypertonic saline. If there is any evidence of or concern for head trauma or ICP, the patient's head should be elevated 15–30 degrees using a flat board or other rigid surface. Pillows should not be used to elevate the patient's head, as this can cause compression of the jugular vein(s), which impairs cerebral venous outflow. Jugular venipuncture should also be avoided. Mannitol (0.25–1 g/kg IV over 15–20 minutes) is an effective osmotic diuretic to decrease intravascular volume and facilitate fluid movement from the central nervous tissue, as with cerebral edema. The resultant diuresis will lead to dehydration if mannitol administration is not followed by intravenous fluid therapy in patients that cannot or will not drink. Hypertonic saline (7.5%, 3–5 ml/kg IV over 15–20 minutes in dogs, 2–3 ml/kg IV over 15–20 minutes in cats) is also effective for treating intracranial hypertension secondary to cerebral edema and works via free water osmotic shifting out of the tissue and into the hypertonic intravascular space created by the increased sodium load [78]. Concentrated (21%) sodium chloride can be combined with 0.9% saline to create a 7.5% solution by mixing 17 ml of 21% saline with 43 ml of crystalloid. The use of corticosteroids is contraindicated in patients with head trauma, as they can contribute to gastrointestinal ulceration, especially after an episode of hypoperfusion, and precipitate hyperglycemia, which has been associated with a worsened neurologic injury in veterinary patients [79].

Veterinary patients with any history of or concern for cervical or spinal trauma should be secured to a backboard until a complete assessment of injuries is performed. In trauma patients, assessment of cranial nerves, visual examination for external signs of head trauma, and any abnormalities of body position, spinal reflexes, and the presence or absence of pain sensation should be determined before administration of drugs that may impact interpretation of findings, including analgesics and atropine. However, assessment for the presence of a head tilt, physiologic nystagmus, and postural reflexes should only be performed if it is safe to move the patient's neck and limbs. Body position changes that can be seen in patients with trauma, spinal cord lesions, or intracranial disease include Schiff–Sherrington (forelimb extensor rigidity and hindlimb flaccidity associated with a T2-L4

spinal cord lesion), decerebrate rigidity (neck extension, hyperextension of all four limbs, and decreased consciousness), and decerebellate rigidity (thoracic limb hyperextension, variable changes in the pelvic limbs, and appropriate consciousness).

A veterinary modified Glasgow Coma Scale score (mGCS) has been developed and evaluated retrospectively for assessing the severity of neurologic injury [80]. Scores are determined after assessment of level of consciousness, cranial nerve function, and motor function with higher scores (15–18) being associated with a better prognosis than lower scores (3–8 for grave prognosis and 9–14 for poor to guarded). Scoring and exact prognostication should be performed with caution however, since the mGCS has not been prospectively evaluated and patient scores may improve with therapeutic intervention and time. In a study of injured dogs and cats for whom the mGCS was used as part of trauma scoring for a veterinary trauma database, mGCS scoring system corresponded with outcome in dogs and cats with known head trauma [81, 82].

For patients with deficits in conscious proprioception, motor function, and pain sensation, spinal reflexes should be used for neurolocalization of spinal cord dysfunction to segments C1–C5, C6–T2, T3–L3, L4–S1, and S1–S3. Common causes of spinal cord disease in veterinary patients include intervertebral disc disease (IVDD), trauma, neoplasia, vascular events, and infectious/inflammatory processes. Surgical intervention could be indicated for traumatic, neoplastic, and IVDD, especially those conditions resulting in neurologic dysfunction. Patients with rapidly progressive neurologic changes and loss of deep pain may require emergency diagnostic imaging and surgical intervention, especially if IVDD is the cause. Patients with cervical lesions (C1–C5) are at risk of ventilatory failure due to phrenic and intercostal nerve involvement, particularly after surgical decompression, as there will be the added impact of secondary surgical swelling and hemorrhage. Respiratory pattern, effort, and objective measures of ventilation ($PvCO_2$ or $PaCO_2$) should be monitored very closely. Changes in oxygenation ($PaO_2$ and $SpO_2$) as a result of ventilatory failure may be late findings and should not be the sole determinant of effective ventilation and respiration. Mechanical ventilation may be necessary in patients with cervical lesions and should be anticipated in all postoperative patients with cervical neurolocalization.

## Urinary Assessment

Many injuries and abnormalities of the urinary tract are not readily apparent on initial triage and physical examination. Historical information from the pet owner regarding changes in water consumption, urine production, stranguria, pigmenturia, and recent trauma can raise suspicion for urinary tract dysfunction, however, some patients have vague and non-specific historical signs. For example, many male cats with urethral obstruction present for lethargy and/or constipation, as many owners are unable to differentiate stranguria from tenesmus.

After respiratory, cardiovascular, and neurologic assessments have been performed and treatment of urgent abnormalities initiated, assessment of the urinary tract can be performed. Palpation for a urinary bladder should be performed in all patients to assess for urethral obstruction, which causes a large, firm, painful, bladder that is unable to be expressed. Palpation of a bladder in trauma patients does not rule out injury and leakage, as small tears may not completely decompress the bladder. Additionally, lack of a palpable bladder is not always synonymous with rupture, as the bladder may not be palpable due to small size from dehydration, recent expression, or anuric or oliguric renal failure. Ultrasound is a useful tool in the emergency room, especially for urinary tract assessment. A standardized technique for abdominal ultrasonographic assessment in veterinary trauma patients has been created and validated. In human medicine, a similar technique has largely replaced the need for diagnostic peritoneal lavage in blunt abdominal trauma patients. Focused assessment sonography for trauma (FAST) has been validated to determine whether free fluid is present in the abdominal cavity after trauma [83]. It is a more sensitive diagnostic for free fluid than the presence of a palpable fluid wave, which requires at least 40 ml/kg of fluid within the peritoneal cavity. Using ultrasound in transverse and longitudinal planes, and the patient in lateral recumbency, the abdomen is evaluated at the gravity dependent and independent flanks (in the region of the kidneys), over the bladder, and below the xiphoid. If needed, ultrasound guidance or blind abdominocentesis via a one- or four-quadrant closed needle/syringe technique can be used to collect any free fluid. In many patients with significant dehydration, free fluid may not be present in the peritoneum initially, and serial monitoring as the patient is rehydrated should be performed. Once fluid is obtained, PCV/TS, glucose, lactate, creatinine, potassium, cytology, and culture can help determine the etiology of the effusion. In dogs, a fluid to blood creatinine ratio of greater than 2:1, and fluid to blood potassium ratio of greater than 1.4:1 is supportive of a diagnosis of uroabdomen [84]. In cats, a fluid to blood creatinine ratio of 2:1 and fluid to blood potassium ratio of 1.9:1 is supportive of a diagnosis of uroperitoneum [85, 86].

Initial bloodwork may reveal azotemia, which could be due to prerenal, renal, or post-renal causes. Assessment of urine specific gravity in conjunction with azotemia and PCV/TS can help to determine the etiology, but it may not be feasible or safe to obtain a urine sample during triage

and before initiation of fluid therapy. This is especially true of female dogs and cats, and male cats where sedation for catheter placement is often necessary. Cystocentesis may be contraindicated in patients where there is any concern for coagulopathy, thrombocytopenia, or thrombopathia. An assessment of prerenal azotemia is therefore often made on physical examination findings and evidence of hemoconcentation without concurrent urine specific gravity. In these cases, improvement or resolution of azotemia in response to fluid therapy and rehydration helps to support the diagnosis of prerenal azotemia. For stabilization of the uremic patient, please refer to Chapter 22.

Urine drainage techniques can be useful for exact urine production quantification and management of urine leakage. Urinary catheterization can be very helpful in patients with urinary trauma to document appropriate urine production, maintain bladder decompression in patients for whom concern over bladder or urethral trauma exists, and for patient comfort and ease of management in non-ambulatory patients. However, this use is controversial owing to the risk of ascending urinary tract infections, particularly in a hospital setting. Urinary catheterization is easy to perform in most male dogs without sedation, but placement in female dogs and male and female cats requires heavy sedation and can be more technically challenging, especially in small female dogs (Figure 1.6). Urinary drainage with locking loop or "pigtail" catheters in the peritoneum can be helpful to facilitate urine drainage and permit patient stabilization in preparation for surgical intervention when a uroabdomen is present (Figure 1.7a,b). The same catheters can be used in the bladder for decompression when transurethral catheterization is not possible, and in the renal pelvis as a nephrostomy tube when there is a ureteral obstruction or ureteral injury [86, 87].

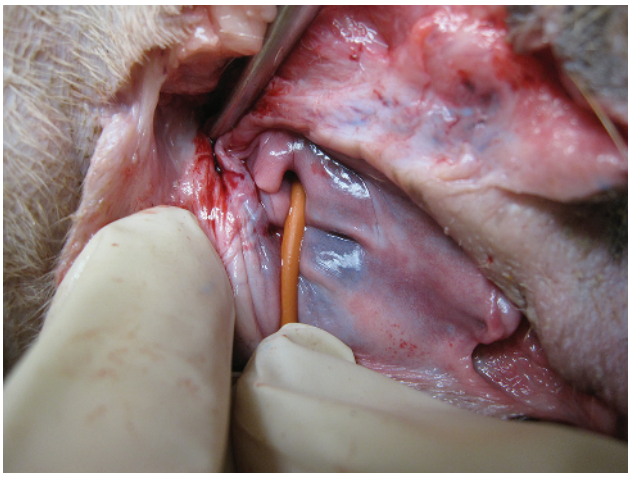
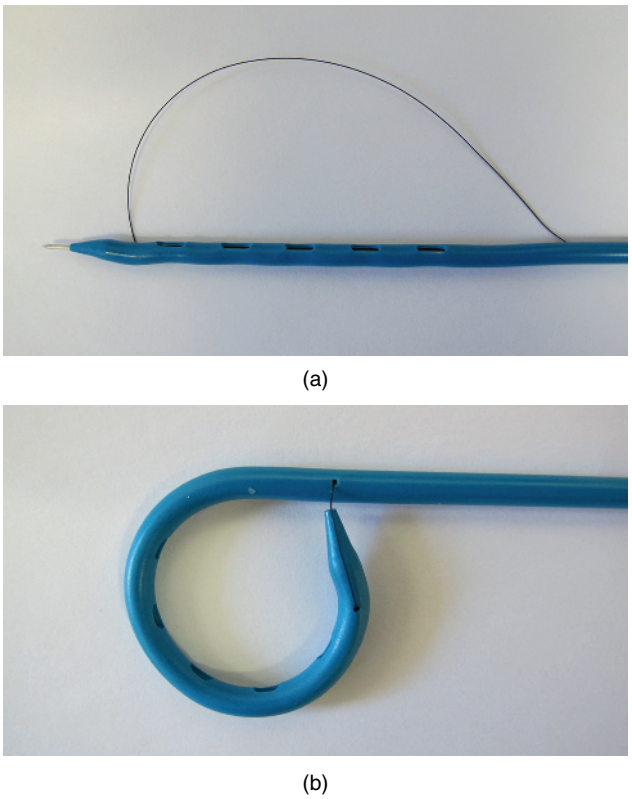


**Figure 1.6** Cadaveric dissection of the urethral papilla in a female dog, which is the major landmark used when performing urinary catheterization.



(a)

(b)

**Figure 1.7** Distal end of a locking loop, or pigtail, catheter. The catheter coil is straightened over a stylet and trocar for placement (a), and then locked into the loop configuration using the suture upon removal of the stylet and trocar (b).

## Vascular Access

Vascular access is critical in emergency patients, especially those undergoing surgery. Peripheral venous catheters are most commonly used, since they are relatively inexpensive, widely available, and can be placed quickly in emergency situations. However, they do not usually allow for repeated blood sample collection and are not appropriate for hyperosmolar fluids, including total or partial parenteral nutrition (TPN, PPN). These catheters can also be dislodged, soiled, and if a small-bore catheter is used, they will not permit rapid administration of large volumes of fluids or medications. Vessels commonly used for peripheral catheters in small animal patients are the cephalic or accessory cephalic veins, lateral saphenous vein, or distance branches the medial saphenous vein [88]. Clipping of the hair and sterile preparation of the site are preferred, but not possible in all emergency situations. Failure to adequately prepare the skin prior to catheter placement has been associated

with increased positive bacterial cultures compared with those that were aseptically placed [89]. In severely hypovolemic patients for whom these vessels cannot be cannulated percutaneously, surgical cut-down to facilitate vascular access can be performed quickly and safely in small animal patients. Catheters placed without adequate skin preparation and sterile technique or with an emergency surgical cut-down procedure should be considered temporary and removed once additional vascular access is obtained and the patient is more stable to prevent infection.

When repeated blood sample collection is anticipated, or for administration of multiple fluids or medications simultaneously, including TPN or PPN, central venous catheterization should be considered. Central catheters can have up to four lumens, which is convenient for concurrent administration of several intravenous therapies. If administration of parenteral nutrition is anticipated, one lumen should be reserved specifically for this use and labeled accordingly. Central catheters are generally placed in larger vessels, including the jugular vein and medial and lateral saphenous veins. They are most commonly placed using the Seldinger, or over the wire, technique (Video 1.3). Surgical cut-down for vascular access for Seldinger technique or venotomy and through the needle (BD Intracath, Argon Medical Devices, Franklin Lakes, NJ) catheters can also be used. Regardless of the technique for placement, since a large vessel is being accessed, adequacy of primary and secondary hemostasis must be confirmed prior to placement [88, 90]. Strict aseptic technique must be followed. It is also important to have adequate patient restraint, which generally requires sedation or anesthesia, since maintenance of positioning and discomfort of vessel dilation is not well tolerated by many awake patients. The catheter is secured to the skin with sutures, and the insertion site covered by a protective wrap.


**Video 1.3** Placement of a jugular multi-lumen catheter using Seldinger technique after a peripheral catheter has been placed in the jugular vein.

When intravenous catheterization is not possible, which is often the case in neonatal and small pediatric patients, intraosseous (IO) catheterization provides a rapid, safe method for delivery of fluid therapy and medications. This is because the capillary network within the marrow cavity is in direct communication with the nutrient and emissary veins that drain into the central circulation. Crystalloids, colloids, blood products and medications, including those for cardiopulmonary resuscitation, can be administered via the IO route and can be absorbed rapidly enough to be effective for the treatment of hypovolemic shock and cardiopulmonary arrest [91–96]. Sites commonly used for IO catheterization include the trochanteric fossa of the femur, proximal tibia, tibial tuberosity, wing of the ileum, the ischium and greater tubercle of the humerus [88, 92], with the trochanteric fossa and tibia used most commonly. Contraindications of placement of an IO catheter include fracture of the bone intended for cannulation, pneumatic bones in birds, and evidence of infection near the intended catheter site. Bone growth is not impacted by IO catheterization [95]. IO catheterization can be achieved with a variety of techniques, including standard hypodermic needles and spinal needles, IO infusion needles, a spring-loaded penetration injection gun (Vet B.I.G Bone Injection Gun (15-G), WaisMed Ltd, Houston, TX) and an automatic rotary insertion drill (EZ-IO (15-G Pediatric Needle Set), Vidacare Corporation, San Antonio, TX.). In a cat cadaveric study comparing these devices, the injection gun was found to be faster and easier to use, but there were no differences detected between insertion site (humerus or tibia), complications or success between the injection gun, rotary drill or manual IO catheter [96].

Direct arterial blood pressure measurement should be considered in any hemodynamically unstable patient. It allows for continuous, accurate pressure determination in the face of hypotension, hypertension, and arrhythmias. Indwelling arterial catheters can also be used to obtain blood samples, particularly for arterial blood gas analysis. Arteries generally accessible for percutaneous placement of an arterial catheter include the dorsal metatarsal artery (most commonly used), the coccygeal artery in the tail, the auricular artery in the dorsal pinna, the femoral artery, and the radial artery [88, 97–99]. Maintenance of arterial catheters in all locations, except for the dorsal metatarsal artery, is difficult in mobile patients and is generally reserved for use in sedated or anesthetized patients. Femoral, dorsal metatarsal, and coccygeal artery catheters can also become contaminated with urine and/or feces, so consideration of these issues is important prior to catheter placement. Cats tend to have poor collateral circulation. It is not recommended to leave arterial catheters in cats for longer than six to eight hours because of concern for ischemic injury to the tissues distal to the catheter [97]. Contraindications for arterial catheterization include lack of close monitoring capabilities, thrombocytopenia, thrombocytopathia, and coagulopathy.

Once the course of the artery is determined by palpation and the site is aseptically prepared, an over-the-needle catheter is used to puncture the artery from an angle of 15–30 degrees above the vessel. Pulsatile blood flow will be observed in the hub of the catheter upon successful arterial cannulization. If percutaneous placement of an arterial catheter is not possible, access to the dorsal metatarsal or femoral artery can be achieved with a surgical cut-down or ultrasound guidance [98]. Care must be taken to avoid damaging the artery, femoral vein, or sciatic nerve during the initial

skin incision and approach to the femoral artery. The catheter is secured with tape and/or sutures depending on the catheter type placed and placement method use. Once the arterial catheter is in place, it can be used to collect arterial blood samples after an adequate pre-sample of blood is taken (generally 3–6 ml of blood into syringes with small amounts of heparinized saline). Clear labeling of an arterial catheter is imperative to ensure that only heparinized saline is injected into the artery. Blood collected from the artery as a pre-sample and medications should never be injected into the arterial catheter. The catheter can also be connected to non-compliant tubing with heparinized saline and a pressure transducer for continuous arterial blood pressure monitoring.

## Analgesia, Sedation and Anxiolytics

The need for sedation and anxiolytics in the stressed or scared veterinary patient and timely analgesics for those in pain cannot be overstated. This is particularly true in patients needing emergency surgery, as many conditions requiring emergency surgical intervention create significant discomfort or pain. Anxiety and stress are present in many patients with respiratory compromise, especially those with upper airway obstruction, and should be addressed immediately to provide relief for the patient and more accurate patient assessment. As with other body systems, frequent reassessment is necessary to ensure adequate analgesia and patient wellbeing. Pain can be challenging to assess accurately in hospitalized feline patients, as they tend to be quieter and more reserved than canine patients [100, 101].

If a patient is assessed to be in pain, analgesics should be administered as soon as possible. It is not appropriate to withhold analgesia because of concerns about creating cardiovascular or respiratory instability or masking changes in patient status. When titrated doses of cardiovascular sparing analgesics and anxiolytics are used, primary cardiovascular or respiratory depression should not result. Instead, if hypotension or respiratory changes are seen after drug administration, it is more likely that the patient's cardiopulmonary instability was uncovered by relief of pain-induced tachycardia, tachypnea, and catecholamine release with secondary vasoconstriction [100].

Multimodal analgesia is preferred in many patients, especially those with marked pain, as the complexity of pain pathways often renders single agent therapy ineffective, irrespective of dose escalation [101]. The addition of local analgesics is often beneficial to decrease the systemic dose. The addition of anxiolytics may also be helpful to decrease the stress and agitation associated with hospitalization, recumbency, and activity restriction present in many surgical patients, and therefore decrease systemic analgesic dosing.

Opioids are often the first choice of analgesia in critical veterinary patients as they have rapid onset of action, can be titrated to an individual's needs, are reversible, and are cardiopulmonary sparing medications. They can be used alone or in combination with other analgesics and anxiolytics and administered as a bolus or constant rate infusion (CRI). Common adverse effects include initial excitatory phase, nausea, vomiting, bradycardia, decreased gastrointestinal motility, and respiratory depression at high doses. Cats tend to be more prone to developing an excitatory period, so the dose of opioids in cats is generally half the canine dose. Rapid intravenous administration of morphine or meperidine can cause histamine release, vasodilation, and hypotension. If adverse effects result from opioid administration, naloxone is a pure antagonist for opioid reversal. Naloxone will reverse both the positive and negative effects of opioids, which may result in pain, excitement, and agitation. Fentanyl transdermal patches are effective ways to provide potent analgesia in an outpatient setting, but they can become displaced or ingested, and have the potential for misuse and abuse by owners, including ingestion by small children.

Lidocaine is effective as a local anesthetic and epidural analgesic. It can also be administered as an intravenous CRI, generally combined with an opioid such as morphine or fentanyl, with or without the addition of a ketamine CRI. When given intravenously, it should be used cautiously and titrated carefully in cardiovascularly unstable patients, as it can cause cardiac arrhythmias, tachycardia, and seizures. This is especially true in cats, and some debate exists as to whether this medication should be given intravenously to cats for analgesia.

N-methyl-d-aspartate (NMDA) antagonists such as ketamine are very effective, owing to their multiple sites of action and effects, including analgesia, neuroprotection, and sedation. Ketamine has limited cardiopulmonary depression but can increase cardiac output and myocardial oxygen consumption, and it can cause muscle tremor activity. Controversy exists over its use in patients with head trauma as there is concern that ketamine contributes to increased ICP. It should be used with caution in patients with hypertension and cardiovascular disease. In cats, it is renally excreted so consideration for renal function with use and dose should be given.

Alpha 2 ($\alpha_2$) adrenergic agonists, such as dexmedetomidine, bind central $\alpha_2$ receptors to result in sedation, muscle relaxation, and analgesia. Additional effects include vasoconstriction, reflex bradycardia, and diuresis. Dexmedetomidine should be used cautiously in critically ill patients and should be reserved only for patients without cardiovascular disease or compromise. At low doses (1–5 μg/kg IV), dexmedetomidine is synergistic with opioids for analgesia and can be used as a bolus or

CRI. Its effects can be reversed with the $\alpha_2$ receptor antagonist atipamezole. The volume of atipamezole used for reversal is the same volume as the administered dexmedetomidine. Intramuscular administration of atipamezole is preferred to prevent rapid drug reversal, which can cause hypotension or aggression [102–104].

Benzodiazepines are effective for mild sedation and anxiolysis with minimal cardiovascular compromise. They are commonly combined with opioids for analgesia and sedation and can decrease the dose of opioids needed to achieve the desired effect. Midazolam and diazepam can be given intravenously, but only midazolam can be given intramuscularly and is preferred for CRI therapy due to the propylene glycol vehicle of diazepam. Reversal of both agents can be accomplished with intravenous dosing of flumazenil.

Phenothiazines, such as acepromazine, provide no analgesia but are potent anxiolytics in veterinary medicine. They must be used cautiously in cardiovascularly unstable patients as they can cause profound vasodilation and hypotension. They are especially useful in patients with respiratory distress, particularly upper airway obstruction. However, intravenous acepromazine takes approximately 15 minutes to achieve maximal effect, so this delay in onset of action should be anticipated in patients [101]. This is important in patients in respiratory distress, for whom this delay may not be tolerated, and more rapidly acting medications should be selected.

Non-steroidal anti-inflammatory drugs (NSAIDs) have a limited role in treating pain and inflammation in many emergency patients, especially those with gastrointestinal and renal disease or cardiovascular compromise. Even NSAIDs that can be administered parenterally should be used with extreme caution in patients with perfusion abnormalities, as they can increase the risk of gastrointestinal ulceration, hepatic insult, and kidney injury. NSAIDs are generally not recommended for cats, unless given as a single dose in healthy, hydrated, and normovolemic cats. An NSAID designed for safer use in cats (robenacoxib) is available, but extensive clinical experience is lacking. However, postoperatively, when perfusion is restored and normalized, many surgical patients benefit from control of inflammation and the analgesia achieved with NSAID therapy.