

A Review of Shock



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Inadequate cellular energy production, most commonly caused by significant disruption of cardiovascular homeostasis, results in the clinical development of shock. Reduced tissue perfusion leads to decreased oxygen supply to the tissues compared to the amount of oxygen that those tissues consume. Clinically, shock can vary from subtle abnormalities to cardiovascular collapse. Rapid identification of shock, followed by focused therapy and close monitoring, is essential to a successful outcome.^{1,2}

There are currently five types of shock based on the underlying cause (hypovolemic, cardiogenic, distributive, metabolic, and hypoxemic). A patient may experience more than one type of shock simultaneously, so that the classifications may have limited clinical benefit. Hypovolemic, cardiogenic, and distributive shock are the most common forms seen clinically.¹

Shock develops at the cellular level in response to a decrease in oxygen delivery (DO_2), an increase in oxygen consumption by the tissues (VO_2), or a combination of the two. Decreased oxygen delivery is the

most common cellular cause of shock; however, hypoxemia (severe anemia, pulmonary disease) and some metabolic derangements (e.g., hypoglycemia, toxin exposures) can also lead to shock.

Pathophysiology

Hypovolemic shock most commonly results from hemorrhage (internal or external) or significant loss of other body fluids (severe vomiting, diarrhea). Excessive body fluid loss leads to a reduced blood volume (hypovolemia), which results in decreased cardiac output. Initially, the body makes acute compensatory efforts to increase blood volume, such as releasing catecholamines, which causes heart rate elevation, vasoconstriction, and improved cardiac contractility, resulting in improved cardiac output. Fluid is also shifted from the interstitial space to the intravascular space to restore adequate blood volume.³ During this initial phase, the body can effectively compensate for the decreased blood volume (or “compensated shock”).

In the absence of medical intervention, the initial homeostatic efforts become ineffective (or “decompensated shock”). Prolonged reduced tissue perfusion will lead to systemically inadequate tissue oxygenation and cellular dysfunction, which triggers a series of physiologic events that eventually result in multiple organ failures and possibly death.⁴

Clinical Presentation

Given that shock develops at the cellular level, it can affect every body system. During the compensatory phase of shock, the cardiovascular and neurologic systems often reveal abnormalities. As shock progresses, the respiratory, gastrointestinal, and renal systems can develop abnormalities. Ultimately, end-organ failure (acute kidney injury, liver failure, cardiopulmonary arrest) can occur if shock is not reversed. In dogs, the gastrointestinal tract appears to be particularly sensitive to shock.¹ Dogs with shock often develop ileus, diarrhea, hematochezia, or melena. In contrast to dogs, cats commonly develop respiratory

abnormalities, as their lungs appear particularly sensitive to shock development.⁵ Changes in heart rate are also unpredictable in cats with shock and can be increased or decreased. In general, cats experiencing shock develop lethargy, hypothermia, pale mucous membranes, poor peripheral pulses, cool extremities, and generalized weakness or collapse.⁵

Patients presenting in the compensatory phase of shock may only have subtle abnormalities on physical exams, such as mild mental dullness, mild to moderate tachycardia (dogs), and mild tachypnea.¹ These patients may appear more stable than they truly are. Patients in the decompensated phase of shock exhibit more severe signs, including depressed mentation, systemic weakness, tachycardia (dogs) or bradycardia (cats), tachypnea, absent or weak peripheral pulses, pale mucous membranes, and prolonged capillary refill time (CRT > 2 seconds).¹

Diagnosing Shock

Clinical signs and patient history are most commonly used to diagnose shock; however, it can be challenging to identify in patients presenting in compensated shock. Early recognition and treatment of shock improve patient outcomes, so ideally, patients in this group would be identified and receive intervention as soon as possible.^{7, 8}

A thorough physical exam and vital parameter monitoring are essential in identifying patients in both compensated and decompensated shock, as well as those patients who are at risk for developing shock. Diagnostics that can help evaluate a patient for the



A dog exhibiting decompensated shock.

presence of shock include blood pressure measurement, lactate level, calculating the shock index, and point-of-care imaging (AFAST/TFAST). In conjunction with other clinical signs, a Doppler blood

pressure measurement of < 90 mm Hg or a mean arterial pressure (MAP) of < 65 mm Hg is consistent with shock.⁶

During shock, inadequate tissue oxygen supply causes cellular function to shift from aerobic to anaerobic metabolism, leading to lactate production.⁴ A lactate measurement greater than 2.5 mmol/L indicates anaerobic metabolism due to systemic hypoperfusion. It should increase clinical suspicion of shock in conjunction with a patient's history and other physical exam findings.^{4,6,9} With medical intervention and stabilization, an improvement in lactate over time more accurately predicts outcome as compared to a single initial measurement.^{2,4,10}

As discussed above, heart rate and systolic blood pressure may be normal during early, compensated shock, leading to a false assumption of patient stability. The shock index (SI) is a calculated ratio between heart rate and systolic blood pressure (HR/SBP).⁹ In human patients with normal heart rate and systolic blood pressure, SI has been found to more accurately identify shock and predict outcome.^{9,10} Recent veterinary literature evaluating the clinical utility of SI found dogs presenting as emergencies with an SI > 1.0 were significantly more likely to be experiencing shock as compared to dogs who were not in shock.⁹ Another canine study concluded that an SI > 0.9 was consistent with compensated shock in dogs with ongoing hemorrhage.¹¹ A third study found that in dogs suffering from vehicular trauma, an increased SI was associated with increased mortality.¹² As of the writing of this article, SI has not been extensively investigated in cats.

Focused assessment with sonography (FAST) examination has gained wide acceptance in the emergency/critical care setting as a noninvasive and rapid screening test that utilizes ultrasound to identify the presence of free fluid in the abdominal (AFAST), thoracic (TFAST), and pericardial spaces.^{13,14} The technique is rapid and easy to learn and provides non-radiologists helpful information within minutes of a

patient's presentation.¹³ AFAST exams can be useful to rapidly and non-invasively identify free fluid in patients following trauma.^{13, 15} In a recent study, AFAST/TFAST were shown to have high agreement with CT findings for the diagnosis of peritoneal and pleural effusion; TFAST was less reliable in accurately diagnosing pneumothorax.¹⁸ AFAST/TFAST can be combined with fluid sampling to provide a rapid and accurate diagnosis, directing further stabilization, diagnostics, and therapy.

Treating Shock

Successful treatment of shock requires aggressive and early intervention to restore adequate circulating blood volume, tissue perfusion, and oxygen delivery to tissues. Fluid therapy is the mainstay of therapy in all forms of shock except for cardiogenic shock. Aggressive correction of the physiologic derangements often requires the rapid delivery of large volumes of fluids. Use of a large bore, short catheter(s) in peripheral or central veins is ideal to allow delivery of fluids as rapidly as possible. Intraosseous catheters can be used as well if vascular access is unobtainable.

Replacement isotonic crystalloids (Lactated Ringers Solution, Normosol R, 0.9% Sodium Chloride) are most commonly administered as a bolus (given as fast as possible). The recommended crystalloid bolus dose ("shock dose") is based on a patient's blood volume, where the blood volume of dogs is 60-90 mL/kg, and cats is 45-60 mL/kg. It is recommended to start with a bolus of 10-20 mL/kg (alternately ¼ of blood volume/"shock dose") of crystalloid intravenously over 15 to 30 minutes and then reevaluate the patient.¹⁶ Fluid therapy should be tailored to the individual patient based on clinical response to avoid fluid overload. After 30 minutes, only 25% of administered crystalloid will remain in the vasculature so that repeat doses may be required.¹⁷ Animals with decompensated shock often require further therapies, such as additional fluid therapy (hypertonic crystalloids, colloid therapy, blood products), vasopressor therapy, and supplemental oxygen therapy, in addition to therapies addressing the underlying cause of shock.

In animals with hypovolemic shock due to hemorrhage, hypotensive resuscitation (to maintain a MAP of 60 mm Hg, instead of > 90 mm Hg) may be beneficial until surgery can be performed to control the hemorrhage (damage control surgery).^{1, 16, 18} This is a temporary strategy to be used between the time of presentation and surgical intervention or other means of hemostatic control.¹⁶

Cardiogenic shock develops due to decreased cardiac function in the presence of adequate intravascular volume; reduced forward blood flow leads to inadequate tissue oxygen delivery. Poor contractility (such as in dilated cardiomyopathy or sepsis), as well as inadequate ventricular filling (such as with pericardial tamponade or arrhythmias), can result in cardiogenic shock. Patients in cardiogenic shock should not be aggressively treated with fluid therapy. Instead, therapies such as supplemental oxygen, diuretics, positive inotropic drugs (dobutamine), anti-arrhythmic agents should be considered as indicated. Sometimes patients may benefit from low-dose fluid therapy to enhance forward blood flow and maximize cardiac output. Severe cases of cardiogenic shock may require sedation, endotracheal intubation, and mechanical ventilation until the underlying cardiac disease can be controlled.¹

Resuscitation therapy should continue until physiologic end goals are achieved (normalization in heart rate/rhythm, respiratory rate, mucous membrane color and CRT, mentation, body temperature).⁷ More recent studies suggest that normalization of these parameters may not correlate with a resolution of decreased tissue perfusion.¹⁶ Thus, continued close monitoring of physical exam findings and other diagnostics are recommended until the patient shows signs of consistent recovery.

Identifying patients in compensated shock can be challenging as it often involves rapid decision-making in the face of a limited physical exam and incomplete medical history. Early, aggressive therapy to correct fluid losses and restore tissue perfusion often results in a positive outcome. If untreated, shock will progress to multi-organ failure and death.

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