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Shock: An Overview

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Shock is a sequela of trauma and diseases commonly seen in emergency practice, such as heart failure, inflammatory conditions (e.g., pancreatitis), or sepsis. The common deficiency that shock patients share is decreased delivery or utilization of oxygen.

Shock is defined as *inadequate cellular energy production* or *decreased cellular oxygen utilization related to decreased blood flow that leads to cell death and organ failure.*[1] Inadequate energy production is exemplified by decreased oxygen delivery. In traumatized patients, this may be due to hemorrhage; in patients with heart failure, it may be related to decreased cardiac output; and in patients with inflammatory conditions, it may be secondary to maldistribution and inappropriate vascular resistance. Decreased cellular oxygen utilization is seen in septic shock and with certain toxins.[1]

PATHOPHYSIOLOGY OF SHOCK

To understand the pathophysiology of shock, one must understand how oxygen is delivered to, and used by, cells.

Oxygen Delivery

Hemoglobin, the body's oxygen carrier, is found within red blood cells. Each molecule of hemoglobin is able to bind up to 4 molecules of oxygen. This oxygen is then off-loaded to cells for use in energy production.

Normally, the amount of oxygen delivered to the cell is 2 to 4 times the amount required, depending on the tissue, which ensures an adequate supply. However, oxygen delivery depends on adequate perfusion of tissue. If tissues are not perfused with blood, then oxygen is not delivered to the cells, regardless of the oxygen content of the blood.[2]

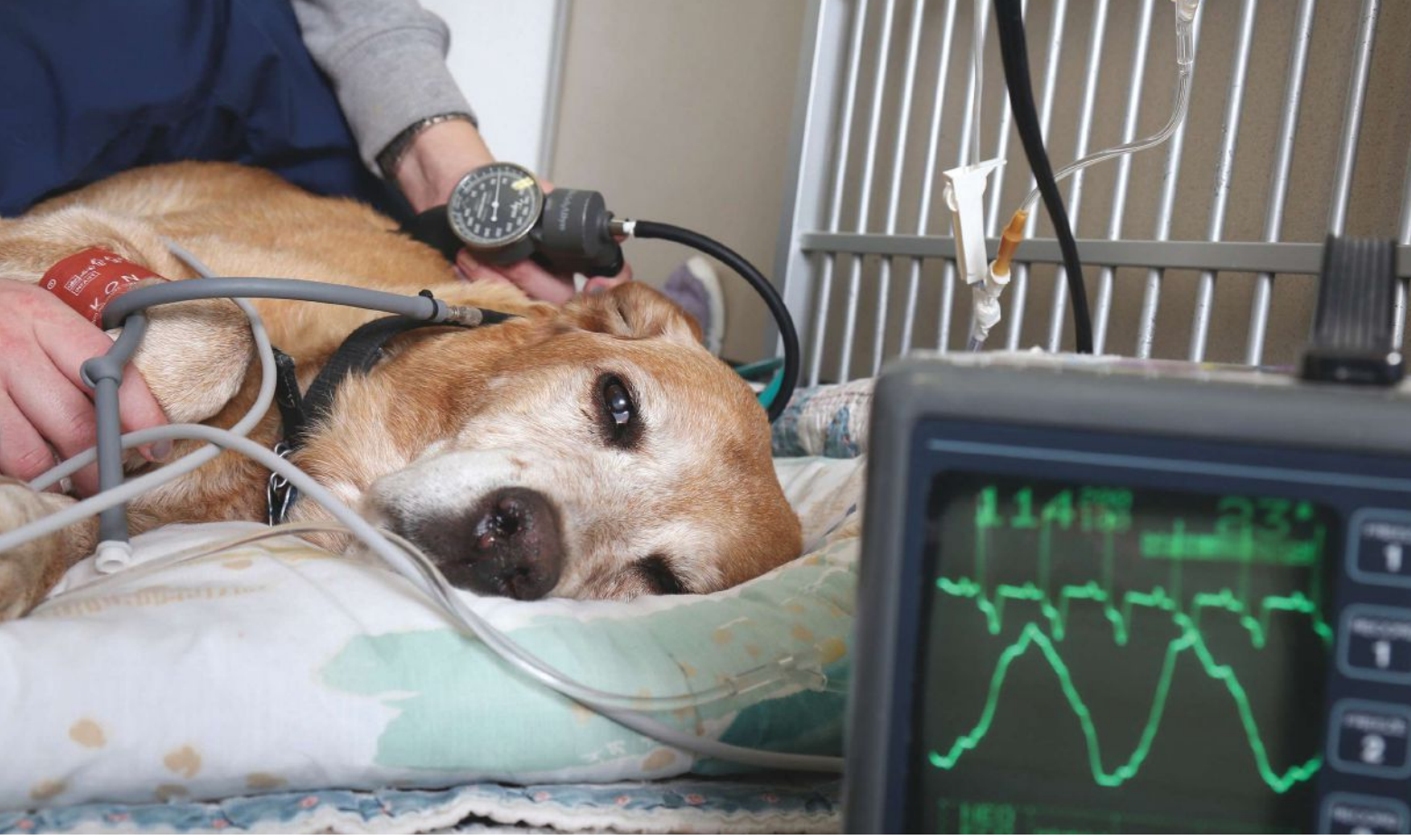
Based on the formulas in **BOX 1**, a decrease in oxygen delivery can be secondary to an increase in afterload, as well as a decrease in heart rate, stroke volume, and the concentration or saturation of hemoglobin.[2]

Heart Rate

Several factors that affect cardiac output and blood pressure, including the stretch of the vascular walls, partial pressures of oxygen and carbon dioxide in the blood, and pH, play important roles in regulation of heart rate in shock patients.

Changes in the mean arterial pressure (MAP) trigger changes in heart rate. An increase in MAP causes bradycardia and vasodilation, while a decrease causes tachycardia and vasoconstriction.[2] These changes are mediated by baroreceptors in the heart and great vessels. Although baroreceptors do not affect heart rate directly,[2] they are sensitive to the stretch of vascular walls and provide feedback that can promote or inhibit vasoconstriction.

- **High-pressure vascular baroreceptors**, located in arterial walls, sense increases in stretch when MAP rises and the reduction when it falls. When MAP is low, the activity of these baroreceptors decreases, prompting an increase in sympathetic nervous system activity and vasoconstriction.[3]
- **Low-pressure baroreceptors** sense decreases in effective circulating volume due to decreased blood volume, with similar results.



Angel, a patient at Animal Emergency & Specialty Center, Parker, CO, receives care from Alex Botkin, a technician in training. *Image by Brian Walski/Colorado Visions.*

When blood volume is low, vasopressin is released, which decreases sodium secretion. Residual sodium in the vascular space causes an increase in plasma osmolarity, which helps hold water in the vascular lumen.[4] Depending on the type of shock, affected patients may have a low MAP, low blood volume, or both. However, depending on the stage of shock and the ability of the patient to compensate through these mechanisms, the heart rate may be increased, normal, or decreased.[3]

A decrease in cardiac output results in an increase in partial pressure of carbon dioxide ($PaCO_2$) and a decrease in pH and partial pressure of oxygen (PaO_2). Chemoreceptors (found primarily in the brain) sense changes in blood $PaCO_2$, PaO_2, and pH secondary to a drop in cardiac output, resulting in tachycardia in an attempt to increase cardiac output. If a change in cardiac output does not alter these values, there is no reflex tachycardia.[2]

BOX 1 Formulas for Oxygen Delivery

- Oxygen delivery (Do_2): Product of cardiac output (CO) and oxygen content of arterial blood (Cao_2)

$$Do_2 = CO \times Cao_2$$

- Cardiac output (CO): Product of stroke volume (SV) and heart rate (HR)

$$CO = SV \times HR$$

- Oxygen content of blood (Cao_2): Product of hemoglobin concentration in blood (Hb), a factor of 1.39, and oxygen saturation of hemoglobin (Sao_2)

$$Cao_2 = ([Hb \times 1.39] \times Sao_2)$$

Hemoglobin Saturation and Concentration

The affinity of hemoglobin for oxygen is relatively low, but it increases as each molecule of oxygen binds (i.e., as hemoglobin saturation with oxygen increases). As oxygen is off-loaded to tissue, the affinity decreases again, facilitating further off-loading of oxygen. Hemoglobin can also bind carbon dioxide, carbon monoxide, and nitric acid.[2] When these are bound to hemoglobin, they prevent the binding of oxygen, causing decreased oxygen saturation and leading to tissue hypoxia and shock despite normal oxygenation of the blood.

Some conditions can increase the affinity of hemoglobin for oxygen, thereby decreasing off-loading of oxygen to tissue. These include alkalosis, hypocapnia, hypothermia, and methemoglobinemia. They may be caused by respiratory disease (e.g., decreased fraction of inspired oxygen, shunt, pneumonia, asthma, hypoventilation) or cardiac disease (e.g., pericardial effusion, cardiac tamponade, congestive heart

failure).[2] The common end result is a decrease in oxygen delivery at the cellular level, again leading to shock.

A decrease in oxygenated hemoglobin content in the blood may also be caused by a decrease in hemoglobin concentration, as seen in anemia.

Stroke Volume

Stroke volume is determined by preload, afterload, and cardiac contractility.[2]

Preload is determined by the stretching of ventricular cardiac cells in response to the presence of blood in the ventricle (venous return).

Contractile response is related to the magnitude of the stretch; therefore, the greater the stretch, the stronger the contraction. Any decrease in ventricular filling (e.g., decreased venous return) decreases preload and, as a result, cardiac output and blood pressure.[4]

Afterload is the force required by the heart to eject blood. The pressure generated by the ventricle must exceed the pressure on the aortic valve in order for blood to be ejected. An increase in afterload (e.g., an elevated diastolic blood pressure) decreases stroke volume.[4] If stroke volume decreases, both cardiac output and oxygen delivery decrease, leading to shock.

Oxygen Utilization

Normally, mitochondria are responsible for consumption of 98% of the body's oxygen.[1] Through aerobic metabolism, they use oxygen to produce the bulk of energy used in the body in the form of adenosine triphosphate (ATP).[1] A decrease in oxygen delivery to the cell leads to anaerobic metabolism, which is a relatively inefficient method of energy production, producing 2 ATP for every molecule of glucose rather than the 36 ATP produced through aerobic metabolism.[3] In addition, anaerobic metabolism produces lactate, which can be monitored (SEE MONITORING).[4]

In shock patients, prolonged anaerobic metabolism and the resulting decrease in ATP causes sodium and calcium to accumulate in energy-depleted cells. This increases the osmotic pull and water enters the cell, causing cellular swelling and death.[2] Ischemia of the cell causes production of inflammatory mediators, leading to an increase in capillary permeability, vasodilation, leukocyte activation, and mitochondrial dysfunction.

Shock also manifests when a cell is unable to utilize oxygen. One example is cyanide toxicosis: this process disrupts the electron transport chain of cellular respiration, leading to a decrease in energy production, which prevents use of oxygen by the cell and causes hypoxia and shock despite a normal arterial oxygen concentration.[1]

When oxygen is reintroduced to the cell, reperfusion injury can occur. In this situation, radical oxygen species (HO^-, O_2^-, H_2O_2) are produced. These compounds cause further cellular dysfunction, an increase in cellular permeability, damage to the DNA, and the breakdown of proteins.[5]

TABLE 1 Clinical Signs Associated with Each Stage of Shock in Dogs

PHYSICAL EXAMINATION FINDINGS	COMPENSATORY SHOCK	EARLY DECOMPENSATORY SHOCK	LATE DECOMPENSATORY SHOCK
Temperature	Normal to low normal (98°F–99°F)[a]	Slight to moderate hypothermia (96°F–98°F)	Moderate to marked hypothermia (<96°F)
Heart rate	Tachycardia (>180 bpm)	Tachycardia (>150 bpm)	Bradycardia (<140 bpm)
Mucous membrane color	Normal to pale (hyperemic in distributive shock)	Pale	Pale to gray/muddy
Capillary refill time	Normal to slightly prolonged (<1 sec; rapid in distributive shock)	Prolonged (<2 sec)	Prolonged (≥2 sec)
Respiratory rate	Tachypnea (>50 breaths/min)	Tachypnea (>50 breaths/min)	Bradypnea
Blood pressure	Slight hypotension to normal (70–80 mm Hg)	Mild to moderate hypotension (50–70 mm Hg)	Marked hypotension refractory to fluid therapy (<60 mm Hg)
Mentation	Responsive	Obtunded	Obtunded to stuporous

Adapted with permission from Thomovsky E, Johnson PA. Shock pathophysiology. *Compend Contin Educ Pract Vet* 2013;35(8):E1-E9.
[a]Values in parentheses are approximate.

TYPES OF SHOCK

There are many ways to categorize shock. Three main categories of shock are described here: circulatory, metabolic, and hypoxic.[1]

Circulatory Shock

Circulatory shock occurs when there is a decrease in effective circulating volume, as perceived by the baroreceptors. To have an adequate effective circulating volume, the body must have both adequate blood volume and adequate blood pressure. This category of shock is divided into 3 subcategories: cardiogenic, hypovolemic, and distributive.[4]

Cardiogenic Shock

Cardiogenic shock occurs when effective circulating volume decreases despite normal or increased blood volume and appropriate systemic resistance. This type of shock is caused by decreased stroke volume due to a decrease in contractility and can be seen in patients with heart failure, such as those with congestive heart failure, cardiac tamponade, or cardiac arrhythmias.[1]

Clinical signs are similar to those of other types of shock, with the addition of one or all of the following:

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cardiac murmurs, arrhythmias, bloody froth coming from the mouth or nose, orthopnea, and cyanosis.

Hypovolemic Shock

Hypovolemic shock occurs when blood volume is decreased through hemorrhage, third space fluid distribution, or dehydration. Loss of whole blood may be caused by an external wound or internal bleeding, such as that seen with an intraabdominal mass. Trauma may result in a hemoperitoneum or hemothorax. Inflammation (such as that seen with pancreatitis) causes capillaries to become "leaky," leading to fluid loss into body cavities (third spacing). Dehydration may be due to vomiting or diarrhea.[1]

Distributive Shock

Distributive shock occurs when the body is unable to maintain vasoconstriction of blood vessels. This causes systemic vasodilation, leading to hypotension despite normal cardiac function and effective circulating volume. This condition occurs with severe anaphylaxis or any other disease process that causes severe inflammation (e.g., pancreatitis, pyelonephritis, hepatitis).[1]

Sepsis (the presence of infection with systemic signs of inflammation) is a common cause of distributive shock. Septic shock is diagnosed when hypotension secondary to sepsis is nonresponsive to adequate fluid resuscitation.[6] Several factors contribute to septic shock, including bacterial endotoxins, cytokines (tumor necrosis factor α, multiple interleukins) that act as proinflammatory mediators, radical oxygen species released from leukocytes (increasing permeability of the capillaries), and nitric oxide (causing prolonged vasodilation).[7]

Clinical signs associated with distributive shock are different than those seen with other classifications of shock. Patients often present with strong pulses, hyperemic mucous membranes, rapid capillary refill time, and elevated temperature.

Metabolic Shock

Metabolic shock is seen when oxygen delivery to the cell is normal, but the cell is unable to utilize oxygen for energy production. Causes of metabolic shock include hypoglycemia, cyanide poisoning, or mitochondrial dysfunction.[1]

Hypoxic Shock

Hypoxic shock results from impaired oxygen delivery to cells. It may be secondary to a decrease in the oxygen content of blood, as seen in anemia (decreased hemoglobin concentration), decreased hemoglobin saturation, or respiratory disease. Alternatively, the oxygen content of the blood may be normal, but oxygen off-loading may be inadequate.

Patients may have coexisting types of shock. As an example, sepsis can cause widespread inflammation and vasodilation, leading to distributive shock, while simultaneously decreasing the cells' ability to utilize oxygen, causing metabolic shock.

STAGES OF SHOCK

Clinical signs associated with each stage of shock in dogs and cats are summarized in TABLES 1 AND 2.

Compensatory

As previously mentioned, baroreceptors in the heart and

TABLE 2 Clinical Signs Associated with Each Stage of Shock in Cats

PHYSICAL EXAMINATION FINDINGS	COMPENSATORY SHOCK	EARLY DECOMPENSATORY SHOCK	LATE DECOMPENSATORY SHOCK
Temperature	Normal to low normal (<97°F)[a]	Slight to moderate hypothermia (<95°F)	Moderate to marked hypothermia (<90°F)
Heart rate	Severe tachycardia (>240 bpm) or mild bradycardia (160–180 bpm)	Moderate tachycardia (>200 bpm) or bradycardia (120–140 bpm)	Mild tachycardia (>180 bpm) or severe bradycardia (<120 bpm)
Mucous membrane color	Pale (hyperemic in distributive shock)	Pale to white	Pale to gray/muddy
Capillary refill time	Normal to slightly prolonged (<1 sec; rapid in distributive shock)	Prolonged (<2 sec)	Prolonged (≥2 sec)
Respiratory rate	Tachypnea (>60 breaths/min)	Tachypnea (>60 breaths/min)	Bradypnea
Blood pressure	Slight hypotension to normal (80–90 mm Hg)	Mild to moderate hypotension (50–80 mm Hg)	Marked hypotension refractory to fluid therapy (<50 mm Hg)
Mentation	Responsive	Obtunded	Obtunded to stuporous

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[a]Values in parentheses are approximate.

vasculature sense decreases in systemic blood pressure. Initially, in response to decreased oxygen delivery, the body increases blood flow to the tissues. This is accomplished by increasing cardiac output via an increase in heart rate.[8] As a result, blood pressure can be normal due to compensatory mechanisms in this stage.[3] Pale mucous membranes and an increased capillary refill time are due to peripheral vasoconstriction, while decreased temperature is due to vasoconstriction in the gastrointestinal tract. Respiratory rate and effort are normal or increased to compensate for oxygen deficiency. Decreased mentation secondary to decreased blood flow or oxygenation in the brain may be observed.

Decompensatory

Decompensatory shock occurs when the body is no longer able to compensate for the decrease in oxygen delivery. In this stage, the respiratory rate drops owing to a decrease in function of the respiratory muscles. Blood pressure decreases despite tachycardia and may be nonresponsive to fluid resuscitation. As blood gas abnormalities worsen, the patient may become obtunded and hypothermic.[3]

Late Decompensatory

Patients may demonstrate bradycardia and hypotension that does not respond to aggressive fluid therapy. The capillary response time becomes more prolonged, and mucous membranes often appear pale or gray/muddy. The respiratory rate and effort continue to fall owing to failure of the respiratory muscles in response to hypoxia and hypercapnia. The partial pressure of carbon dioxide increases and, in the absence of a reflex tachycardia response, causes a decrease in myocardial force and, subsequently, a decrease in cardiac output.[8]

TREATMENT

Treatment should focus on increasing oxygen delivery to, and extraction by, the tissues. This can be accomplished by providing supplemental oxygen, increasing effective circulating volume with crystalloids or colloids, increasing hemoglobin concentration via blood products, and increasing cardiac output with medications.[9]

An intravenous catheter should be placed to allow vascular access. To deliver a large amount of fluids in a short amount of time, use a large-bore, short catheter. If venous access is not possible, place an intraosseous catheter for use until a peripheral catheter is placed.

Oxygen Supplementation

Patients experiencing shock will benefit from oxygen supplementation. This can be accomplished using several methods, including flow-by oxygen, a mask, nasal cannulas, or an oxygen cage.

Flow-by Oxygen

Flow-by oxygen is a quick and easy method of providing oxygen when a patient is presented. The tubing is placed at the patient's nose and provides oxygen at a concentration of 25% to 45%.[10] While quick and easy, this method is not ideal because it requires a high flow rate and staff to hold the tubing in place. Some patients do not like the feeling of the oxygen flow in their face and become agitated.[10]

Facemask

Oxygen can also be delivered via a facemask, which allows a lower oxygen flow rate and delivers a higher percentage of oxygen (35%–55% if delivered at a rate of 6–10 L/min) compared with flow-by oxygen. It is important that the oxygen mask be the right size and fit for the patient.[10] If it is too loose, oxygen will escape; if it is too tight, the patient will rebreathe carbon dioxide.[10]

Again, some patients may not tolerate the mask and may become agitated. These patients may accept the mask more readily if the diaphragm is removed. While this decreases the chance of the patient rebreathing carbon dioxide, it allows oxygen to escape, decreasing the percentage of oxygen provided.

Nasal Cannula

Once the patient is stable and hospitalized, nasal cannulas can be placed in one or both nostrils to provide long-term oxygen supplementation. A single cannula can deliver 30% to 50% oxygen at a flow rate of 100 to 150 mL/kg/min, while 2 cannulas can increase the delivered oxygen concentration to as much as 70%.[10]

Oxygen Cage

An oxygen cage is an excellent choice for a patient that may not tolerate placement of nasal cannulas (e.g., cats, brachycephalic breeds). Most oxygen cages can provide 40% to 50% oxygen.[10]

Fluid Resuscitation

Crystalloids

While they are the most important aspect of resuscitation in critical patients, crystalloids should be used with caution because aggressive administration can cause a positive fluid balance (fluid overload), which can be detrimental to the patient.[11] Within 30 to 60 minutes of administration,

TECHPOINT

With rapid recognition, appropriate treatment, and vigilant monitoring, many patients that suffer from shock can survive.

60% to 80% of crystalloids have diffused out of the vascular space and into the interstitial space.[11] Because of this, multiple fluid boluses may be required.

Isotonic Crystalloids

Studies in human patients have shown that, when given in high volumes and at rapid rates, 0.9% saline has the potential to cause hypernatremia and hyperchloremia because of the high sodium and chloride concentrations.[11] An isotonic crystalloid that more closely resembles plasma levels of sodium and chloride is recommended, making lactated Ringer's solution, Normosol-R, and Plasma-Lyte the preferred choices for resuscitation.[11] These fluids have been shown to cause fewer complications as well as decrease the risk of mortality.[11]

Hypertonic Saline

Hypertonic saline is a crystalloid solution that contains a higher concentration of sodium and chloride relative to plasma[12]; however, it is administered in smaller volumes than isotonic crystalloids. Hypertonic saline increases plasma osmolarity, pulling water into the vascular space from the interstitial space, thereby expanding plasma volume. The resulting volume expansion is greater than the volume infused and quickly increases cardiac output and contractility as well as the MAP. This effect lasts anywhere from 20 minutes to 3 hours.[12] The smaller volume required makes this an ideal fluid choice for patients that may not tolerate large volumes, such as those with head trauma or cardiac disease.[9]

Hypertonic saline is is available in several concentrations, ranging from 7% to 23%. Hypertonic saline with a concentration of 7% is safe to use in a peripheral vein, is administered at a dose of 2.5 to 5 mL/kg, and should be given no faster than 1 mL/kg/min. If a higher concentration is used (e.g., 23%), it should be diluted before injecting or it will cause hemolysis. The effects of any concentration of hypertonic saline last longer if it is used in conjunction with a colloid. A 23% concentration of hypertonic saline can be mixed with a colloid at a ratio of 1:2.5.[12]

As with all medications, hypertonic saline has unwanted side effects. A transient, dose-dependent increase in sodium and chloride will occur. Pulling fluid from the interstitial space will worsen any dehydration; therefore, hypertonic saline should be avoided in dehydrated patients. If used in the face of dehydration, it must be accompanied by an isotonic crystalloid to restore interstitial fluid.[12]

Colloids

The use of hydroxyethyl starch (HES) has been controversial for many years. In human studies, prolonged usage has been shown to increase the risk of acute kidney injury, coagulopathies, and mortality.[11] Later-generation colloids (tetrastarches) have been shown to be safer owing to their increased clearance, meaning there is less tissue and plasma retention, which decreases the risk of adverse effects.[11]

Human studies have shown that while HES restores the effective circulating volume more quickly than crystalloids, overall, there is no difference in benefit between the two with regard to end-point hemodynamic stabilization.[11] In these studies, those receiving HES required a lower vasopressin dose and maintained a higher central venous pressure, which is an indicator of blood volume. They also had a more rapid restoration of hemodynamic stability. Improvements in blood lactate, heart rate, and blood pressure were similar to those receiving crystalloids.[11]

Human studies have shown that acute kidney damage secondary to use of HES is multifactorial. The hyperviscosity of the colloid causes ischemia leading to acute kidney injury; stasis of flow through the kidneys during filtration causes obstruction of the tubular lumen; and osmotic nephrosis causes swelling of the proximal renal tubular cells. Later-generation colloids are less nephrotoxic, but the same complications still occur.[11]

Human studies have also shown that HES has negative, dose-dependent affects on coagulation. It inhibits platelet adhesion and aggregation by binding to the platelet surface and also decreases the expression of glycoprotein receptors on the surface of the platelet (an important step in platelet adhesion).[11] As with acute kidney damage, these complications are more severe with early-generation colloids.[11]

HES also binds to von Willebrand factor and factor VIII, accelerating their clearance. Clinical bleeding associated with HES administration has not been confirmed in studies specific to veterinary medicine[11]; however, bleeding complications have been observed in veterinary patients. For this reason it is important to consider potential complications when using HES in patients with a coagulopathy or renal disease.

In veterinary medicine, the use of vasopressors and inotropic drugs is preferred before risking injury with high volumes of crystalloids and HES.[11]

Blood Products

In patients in a normal resting state, anemia can be well tolerated and oxygen delivery can be maintained. However, in patients with trauma and acute loss of blood volume, the associated stress, inflammation, and pain contribute to decreased oxygen delivery.[2] Patients can tolerate a blood loss of 10% to 15%, but once blood loss reaches 20% of total blood volume, transfusion is required. Not all patients that are suffering from shock require blood products, but those that do (e.g., patients with hypovolemic shock) benefit from transfusions. If clotting values are elevated, fresh whole blood or fresh frozen plasma can be administered.[1]

Cardiovascular Support

Catecholamines are recommended if the patient is not responding to fluid therapy. Multiple catecholamine receptors are present throughout the cardiovascular system (TABLE 3). As a result, several catecholamines can be used for cardiovascular support, including dopamine, dobutamine, norepinephrine, vasopressin, and epinephrine.

Additional Therapies

Antibiotics

Antibiotics should be administered within 1 hour of suspicion or diagnosis of sepsis because delay of antibiotic administration is associated with increased mortality.[1] In the author's experience, use of broad-spectrum antibiotics is recommended because it is unlikely a specific pathogen can be identified within this time frame.

TABLE 3 Catecholamine Receptors and Locations[13]

RECEPTOR TYPE	LOCATION
Alpha-1 **Beta-2**	Vascular smooth muscle
Beta-1	Myocardium
Dopaminergic-1	Renal, coronary, and mesenteric microvasculature
Dopaminergic-2	Synaptic nerve terminals
Vasopressin-1	Vascular smooth muscle

Gastroprotectants

Mucosal damage is common in patients with shock due to stress-related mucosal disease.[1] Hypovolemia, decreased cardiac output, and vasoconstriction associated with shock lead to splanchnic hypoperfusion and reduced mucosal blood flow, gastrointestinal motility, and bicarbonate secretion, with subsequent development of an acute stress ulcer.

Options to prevent or treat stress-related mucosal disease include histamine$_2$ receptor antagonists, proton pump inhibitors, and sucralfate. Histamine$_2$ receptor antagonists (famotidine, ranitidine) and proton pump inhibitors (omeprazole, pantoprazole) decrease acid production.[14] Sucralfate is a gastrointestinal protectant that forms a paste when it comes in contact with hydrochloric acid, binding to the ulcer site and forming a barrier that prevents additional damage from gastric acids.

Antiemetics

Patients experiencing shock may also develop nausea and may benefit from antiemetics (dolasetron, maropitant citrate) to address or prevent vomiting.

MONITORING

Monitoring the patient during and after resuscitation is very important.

Physical Examination

Physical monitoring is ideal and should include palpating pulses and noting their strength, auscultating the heart while palpating the pulse and noting any asynchronous pulses, and closely watching mucous membrane color as well as the capillary refill time. Auscultating the lungs and noting any increase in respiratory rate and effort are also vital.

Hypoxia can be indicated by dyspnea, tachypnea, anxiety, or restlessness. Cyanosis also indicates hypoxia, but it is important to understand that a hemoglobin concentration >5 g/dL is necessary to detect cyanosis. If the hemoglobin concentration is lower than this, a hypoxic patient will not appear cyanotic.[10]

Blood Analysis

Packed cell volume indicates the potential oxygen-carrying capacity of blood. The hemoglobin content of the blood can be estimated as one-third of the packed cell volume; for example, a patient with a packed cell volume of 30% is expected to have a hemoglobin content of 10 g/dL. A hemoglobin concentration >8 g/dL is necessary to maintain oxygen delivery.[1] Total protein should be

evaluated in conjunction with the packed cell volume. A total protein <3.5 g/dL indicates that oncotic pull is less than adequate and colloids would be beneficial.[1]

Serial blood lactate monitoring provides information regarding the patient's perfusion status and helps guide fluid therapy. Lactate is produced when oxygen delivery is minimal. When blood lactate levels are elevated, hypoperfusion is already present. The degree of hypoperfusion can be estimated based on the blood lactate value. The normal blood lactate level is <2 mmol/L. Mild hypoperfusion is indicated by a blood lactate level of 3 to 4 mmol/L, moderate hypoperfusion by a level of 4 to 6 mmol/L, and severe hypoperfusion by a level of >6 mmol/L.[15] Once perfusion is restored, the blood lactate level should drop rapidly. Serial lactate monitoring is more useful than a single measurement.[16]

Blood Pressure

Blood pressure monitoring is important when evaluating cardiovascular status. Direct, invasive blood pressure monitoring is the gold standard but is not widely available. Indirect, noninvasive monitoring is often more practical and can be conducted via Doppler ultrasonic or oscillometric methods.[1]

Cuff size should be appropriate: in dogs, the cuff width should be 40% of the circumference of the leg at the chosen location; in cats, the circumference should be 30% to 40%.[1] A cuff that is too large will result in a falsely decreased reading, while the reading will be falsely elevated with a cuff that is too small. With the patient in lateral recumbency, the cuff should be placed over a peripheral artery at the level of the heart.[1] Readings are more accurate if the cuff is placed above the carpus (forelimb) or below the hock (hindlimb). Inaccurate results may be obtained if the patient is hypothermic or experiencing vasoconstriction.

Electrocardiography

Monitoring a continuous electrocardiogram allows technicians to evaluate the patient's heart rate closely without disturbing the patient. If the myocardium experiences hypoxia, cardiac arrhythmias in the form of ventricular premature contractions may be seen. These are not usually a concern unless the heart rate is elevated (>180 beats/min) or they are multifocal.[17]

TECHPOINT

A clear understanding of the pathophysiology, clinical signs, and treatment of shock will aid in improving nursing care.

Urinalysis

Urine output should be monitored closely: in dogs, a urinary catheter can be placed; in cats, a litterbox can be weighed. If the patient does not have a urinary catheter in place or will not use a litterbox, a peri-pad can be placed under the patient and weighed to monitor urine output. Urine output should be at least 1 mL/kg/h when a patient is receiving fluid therapy. A decrease in urine output can indicate a decrease in renal function but may also indicate inadequate fluid resuscitation.

In addition to urine output, the urine specific gravity (USG) should be monitored closely. In a dehydrated patient with normal renal function, urine will be concentrated, with a USG >1.045. In a patient that is receiving intravenous fluids and has been adequately resuscitated, USG will be in the range of 1.008 to 1.012 (isothenuria). A USG >1.014 can indicate inadequate fluid resuscitation.

CONCLUSION

Technicians play a critical role in the treatment of shock patients. A clear understanding of the pathophysiology, clinical signs, and treatment of shock will aid technicians in improving their nursing care. With rapid recognition, appropriate treatment, and vigilant monitoring, many patients that suffer from shock can survive. ■

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CE Test Article 2 Shock: An Overview

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1. Inadequate fluid resuscitation, in the early stages of shock, can be indicated by
a. Bradycardia
b. Hypertension
c. Decreased urine output
d. Hyperthermia

2. A decrease in oxygen delivery can be seen with an increase in
a. Preload
b. Afterload
c. Heart rate
d. Contractility

3. Chemoreceptors in the brain respond to a decrease in cardiac output only if it causes a change in blood
a. pH
b. Lactate
c. Pressure
d. Hemoglobin

4. _____ increases hemoglobin's affinity for oxygen.
a. Hyperthermia
b. Hypercapnia
c. Acidosis
d. Methemoglobinemia

5. A patient with a packed cell volume of 36% has an expected hemoglobin of
a. 3.6 g/dL
b. 9 g/dL
c. 12 g/dL
d. 36 g/dL

6. Adequate fluid resuscitation in a cat may be indicated by a
a. USG of 1.010
b. Blood lactate of 4.2
c. Mean arterial blood pressure of 60 mm Hg
d. Temperature of 96°F

7. A blood pressure cuff for a canine patient should measure what percentage of the circumference of the patient's leg?
a. 30%
b. 40%
c. 50%
d. 60%

8. Hydroxyethyl starches can cause a coagulopathy by binding to and accelerating clearance of
a. Factor III
b. Factor VI
c. Factor VIII
d. Factor XI

9. Severe inflammation that causes capillaries to become "leaky" can cause hypovolemic shock via
a. Blood loss
b. Third spacing
c. Dehydration
d. Increasing oncotic pull

10. Compensatory distributive shock differs from other types of shock in that patients exhibit
a. Hypothermia
b. Tachycardia
c. Prolonged capillary refill time
d. Hyperemic mucous membranes