Distributive Shock

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Clinical Clues
Distributive shock is generally associated with altered vasomotor tone—notably inappropriate vasodilation (eg, sepsis, systemic inflammatory response syndrome), excessive vasoconstriction (eg, following trauma or anaphylaxis), or abnormalities in normal blood flow (eg, obstructive diseases such as gastric dilatation-volvulus [GDV] or pericardial effusion)—resulting in maldistribution of blood flow.

Patients with septic distributive shock often have hyperemic mucous membranes caused by uncontrolled vasodilation from inflammatory mediators and cytokine release (see References, page 96). Patients with anaphylactic or obstructive distributive shock show

YOU HAVE ASKED ...
What is distributive shock, and how do I treat it?

THE EXPERT SAYS ...
Shock (ie, inadequate cellular energy production or the body’s inability to supply cells and tissues with oxygen and nutrients and remove waste products) can cause quick clinical deterioration and requires rapid identification and treatment. Distributive shock is a general classification for syndromes that cause massive maldistribution of blood flow (see References, page 96). Anaphylactic, obstructive, and septic shock are common forms of distributive shock.
clinical signs such as tachycardia, pale mucous membranes, delayed capillary refill time, poor pulse quality, and altered mentation; cats may be presented with bradycardia rather than tachycardia. Additional clinical signs of anaphylactic shock in cats and dogs may include vomiting, diarrhea, pruritus, and signs consistent with hypovolemic shock (see Signs of Hypovolemic Shock). Cats in anaphylactic shock may be presented in respiratory distress because of laryngeal or pharyngeal edema, bronchoconstriction, and mucus production; this can occur in dogs but is less common.

Diagnostic Testing & Evaluation
Diagnostic testing for distributive shock depends on signalment, history, and physical examination findings. There are many diagnostics to consider, but the importance of the physical examination cannot be overemphasized. For example, a patient presented on emergency after vaccine administration should prompt the clinician to consider treatment for anaphylactic shock, whereas a large-breed dog presented with a distended abdomen and nonproductive retching suggests GDV. The clinician should identify the need for immediate point-of-care diagnostics and second- or third-tier diagnostics that are valuable but not immediately needed.

Point-of-care diagnostic testing may include a basic minimum database (ie, packed cell volume, total protein, blood glucose, AZO test or blood urea nitrogen/creatinine test), followed by more advanced testing (eg, CBC with blood smear to assess WBCs for toxic changes and pathogens associated with leukocytes or erythrocytes, serum chemistry profile, urinalysis, coagulation parameters, serum lactate, blood pressure measurement). Thoracic and abdominal radiography as well as thoracic and abdominal ultrasonography may also be indicated.

A lactate test is important to assess tissue perfusion. With adequate perfusion, patients in shock often develop hyperlactatemia caused by anaerobic metabolism. The normal lactate concentration

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in adult dogs and cats is <2.5 mmol/L. Persistently elevated lactate concentrations in dogs may help predict mortality in specific diseases, notably GDV. Although single lactate readings were initially used to predict mortality, trends in serial lactate concentration measurements may be more beneficial during resuscitation and have been found to be a better predictor of outcome.

**Treatment**

When a patient is presented with presumptive distributive shock, a typical triage assessment including ABC (airway, breathing, circulation) should be performed. If warranted, IV access and fluid resuscitation should be performed to correct malperfusion and hypotension. Isotonic crystalloids at a shock bolus rate (dogs, 20-40 mL/kg; cats, 10-20 mL/kg over 15-30 minutes) should be administered. Supplemental oxygen should be administered via flow-by methods (50-150 mL/kg/min) or nasal oxygen cannulas (50-100 mL/kg/min).

**Anaphylactic Shock**

If anaphylaxis is suspected, epinephrine, a potent α- and β-adrenergic agonist, is recommended (0.01 mg/kg IV or IM, repeated every 5 minutes as needed). Stimulation of α₁-adrenergic receptors results in vasoconstriction. H₁-receptor antagonists (eg, diphenhydramine 2-4 mg/kg IM) can also be considered, notably to reduce pruritus, erythema, urticaria, hives, and angioedema. In conjunction with H₁-antagonists, H₂-antagonists (eg, famotidine 1-2 mg/kg/IV) decrease erythema and decrease gastric acid production. These medications should not be substituted for epinephrine but rather used adjunctively with epinephrine.

Glucocorticoids are often administered for allergic or hypersensitivity reactions; however, in states of anaphylactic shock, glucocorticoids do not have immediate effect and are not a first-line therapy. The anti-inflammatory effects may not occur for several hours after administration, and, although they may help reduce the severity of a biphasic reaction, they are not a first-line therapy for anaphylaxis. If needed for inflammation or in cases of respiratory distress caused by oropharyngeal edema, dexamethasone sodium phosphate at 0.05-0.1 mg/kg IV once or twice a day is recommended. A constant rate infusion of a vasopressor or positive inotrope may be required in patients unresponsive to fluid and epinephrine therapy.

See *Table* (next page) for drugs and doses used for treating anaphylactic shock.

**Obstructive Shock**

Obstructive shock occurs when there is an obstruction to flow, such as with GDV or pericardial effusion. Resolution is directed at correcting the cause of shock. As with anaphylactic shock patients, patients presented with presumptive obstructive shock should receive triage assessment. If warranted,
IV access and fluid resuscitation should be performed to correct malperfusion and hypotension. Isotonic crystalloids at a shock bolus rate (dogs, 20-40 mL/kg; cats, 10-20 mL/kg over 15-30 minutes) should be administered. Supplemental oxygen should be administered with flow-by oxygen methods (50-150 mL/kg/min) or nasal oxygen cannulas (50-100 mL/kg/min).

In patients with GDV, gastric distension obstructs venous return in the caudal vena cava, leading to decreased cardiac preload and cardiac output. Poor perfusion is treated with IV fluid boluses given to effect, followed by gastric decompression and surgical return of the stomach to its normal position.

Pericardial effusion, another common cause of obstructive shock, can lead to pericardial tamponade resulting from increased intrapericardial pressure, which equals or exceeds right atrial/ventricular pressure and results in decreased ventricular filling. This decreases stroke volume, leading to decreased cardiac output and finally decreased blood pressure. Poor perfusion is treated with IV fluid boluses given to effect, followed by pericardiocentesis and resolution of pericardial tamponade. The goal of treatment is to improve preload and reduce ischemia and reperfusion.

Conclusion
Distributive shock, whether anaphylactic or obstructive, should be carefully evaluated and quickly treated, as it often results in severe cardiovascular abnormalities, multiple organ failure, and/or death. Aggressive fluid resuscitation alone to promote adequate tissue perfusion may be unsuccessful, and additional therapy is often needed.

**References**

**TABLE**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Route</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>0.01 mg/kg</td>
<td>IV or IM</td>
<td>potent α- and β-adrenergic agonist</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>2-4 mg/kg</td>
<td>IV or IM</td>
<td>H&lt;sub&gt;1&lt;/sub&gt;-antihistamines</td>
</tr>
<tr>
<td>Famotidine</td>
<td>1-2 mg/kg</td>
<td>IV or IM</td>
<td>H&lt;sub&gt;2&lt;/sub&gt;-antihistamines</td>
</tr>
<tr>
<td>Dexamethasone SP</td>
<td>0.05-0.1 mg/kg</td>
<td>IV or IM</td>
<td>Glucocorticoid</td>
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