Hypovolemic Shock

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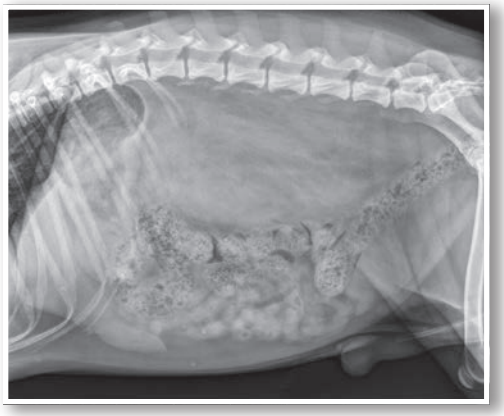

Retroperitoneal effusion in a dog

You have asked…

What is hypovolemic shock, and how should I manage it?

The expert says…

Shock, a syndrome in which clinical deterioration can occur quickly, requires careful analysis and rapid treatment. Broad definitions for shock include inadequate cellular energy production or the inability of the body to supply cells and tissues with oxygen and nutrients and remove waste products. Shock may result from a variety of underlying conditions and can be classified into the broad categories of septic, hemorrhagic, obstructive, and hypovolemic shock.[1-3] Regardless of the underlying cause, all forms of shock share a common concern: inadequate perfusion.[1,2] Perfusion (ie, flow to or through a given structure or tissue bed) is imperative for nutrient and oxygen delivery, as well as removal of cellular waste and byproducts of metabolism. Lack of adequate perfusion can result in cell death, morbidity, and, ultimately, mortality.

All forms of shock share a common concern: inadequate perfusion.

Hypovolemic shock is one of the most common categories of shock seen in clinical veterinary medicine.[4] In hypovolemic shock, perfusion is impaired as a result of an ineffective circulating blood volume. During initial circulating volume loss, there are a number of mechanisms to compensate for decreases in perfusion, including increased levels of 2,3-Bisphosphoglycerate, resulting in a rightward shift in the oxyhemoglobin dissociation curve and a decreased blood viscosity. When approximately 30% of blood volume is lost, however, compensatory mechanisms may fail, resulting in organ dysfunction. Causes of hypovolemia include hemorrhage (surgery, trauma, neoplasia, anticoagulant rodenticide ingestion); fluid loss from vomiting, diarrhea, or renal disease; severe burns; and third-space losses (eg, edema, ascites).[1,2]

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To Compensate, or Not to Compensate…

In states of hypovolemia, the body has a compensatory neuroendocrine response to improve circulating blood volume and metabolic demands. The body senses these changes in several ways, including[1,2]:

- When blood flow to tissue is decreased, oxygen extraction from the blood delivered to the microcirculation is increased.
- Decreased filling of vessels activates stretch receptors and baroreceptors in the aortic arch, carotid body, and splanchnic vessels.

The body responds to these changes in several ways:

- Hormonal mediators are released, including epinephrine, norepinephrine, angiotensin, renin, and aldosterone. This subsequently results in water and salt retention; increased heart rate; and increased cardiac contractility, vasodilation, and redistribution of flow to vital organs.
- Peripheral vagal stimulation and increased sympathetic stimulation result in vasoconstriction of the precapillary arteriolar sphincters, increased heart rate, and increased cardiac contractility.
- Movement of fluid from the interstitial space into the intravascular space is caused by an altered transcapillary pressure gradient (increased intravascular colloid oncotic pressure [COP], decreased intravascular hydrostatic pressure).
- Increased fluid retention by the kidneys results from an up-regulation of the renin–angiotensin–aldosterone system.

When perfusion becomes compromised in spite of these mechanisms, decompensatory hypovolemic shock ensues.

Clinical Clues

Clinical signs of hypovolemic shock include tachycardia (in cats, bradycardia; in dogs, decompensated shock [ie, when compensatory mechanisms cannot respond to ongoing blood loss]), altered mentation, pale pink mucous membranes (**Figure 1**), prolonged capillary refill time, weak femoral pulses, cold extremities, tachypnea, and hyperlactatemia. Lactate levels rise in response to lactic acidosis caused by the anaerobic metabolism secondary to decreased tissue perfusion and hypoxia. These may be distinguished from other causes of shock, including signs of cardiogenic shock (may include loud heart murmur, tachycardia or bradycardia, arrhythmias, weak heart sounds) or distributive shock (eg, bright red mucous membranes [**Figure 2**], bounding femoral pulses).[1,2]

Treatment

Rapid fluid administration is the mainstay of therapy. Isotonic crystalloid fluids (eg, lactated Ringer's solution, Normosol-R [hospira.com], Plasma-Lyte A [abbottanimalhealth.com], 0.9% saline) are often used initially.[5,6] Shock doses of fluids are 90 mL/kg for dogs and 44–60 mL/kg for cats.[7] The entire shock dose is not administered initially; instead, 0.25 to 0.33 of the calculated shock dose is administered as rapidly as possible, usually over 10 to 15 minutes, followed by patient reassessment (eg, heart rate, capillary refill time, mucous membrane color, core

Table 1 **Vasopressors**

Vasopressor	*Dose Range*	*Frequency*
Dobutamine	1–20 µg/kg/min (dogs) 1–5 µg/kg/min (cats)	CRI
Dopamine	5–15 µg/kg/min	CRI
Norepinephrine	0.05–0.3 µg/kg/min	CRI
Vasopressin	0.01–0.04 mU/kg/min	CRI

COP = colloid oncotic pressure

1

Pale pink mucous membranes in a canine patient.

Injected (ie, bright red) mucous membranes in a canine patient.

body temperature, blood pressure). In dogs, a simple method to calculate one-quarter shock volume is to take the patient's weight in pounds and add a zero, indicating the amount of fluid in milliliters to administer as a bolus over 10 to 15 minutes.

Crystalloids alone may be insufficient to restore intravascular and interstitial volume; or, there may be underlying or compounding diseases, including head trauma, pulmonary trauma, and/or hypoproteinemia, which may lead to edema formation. Synthetic colloid fluids may be advantageous if used *with caution* in these cases. Hetastarch or VetStarch (abbottanimalhealth.com) are the most common synthetic colloid choices, with bolus dose recommendations of 2 to 5 mL/kg for cats and 3 to 10 mL/kg for dogs.[8]

For cases with head trauma or pulmonary injury, hypertonic saline may be considered, as it draws fluid from the interstitial spaces to the intravascular space, resulting in a rapid but transient increase in effective circulating volume; 7.5% hypertonic saline is commonly used at 4 to 8 mL/kg via bolus.[9] Hypertonic solutions should be avoided in a dehydrated or hypernatremic patient, as they may lead to undesirable effects (eg, cellular dehydration, stimulating diuresis before adequate plasma volume expansion has been achieved).

When hemorrhage is the underlying cause of hypovolemic shock, additional considerations include definitive control of bleeding and blood product administration. When treating anemia, administration of approximately 10 mL/kg packed RBCs or 30 mL/kg fresh whole blood[8,9] raises the hematocrit by about 10%.

When Is Enough, Enough?

For hypovolemic shock, fluids are administered to reach certain endpoints of resuscitation. The endpoint typically reflects an improved or restored perfusion status of the patient, including normalizing the heart rate, blood pressure, body temperature, mucous membrane color, capillary refill time, and pulse quality.

If signs of hypovolemic shock persist despite the perception of adequate fluid administration, the clinician must consider reasons for endpoint failure, including inadequate volume

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Table 2 Common Crystalloids & Their Chemical Properties

Solution (mEq/L)	*Lactated Ringer's Solution*	*Plasma-Lyte A, Normosol R*	*0.9% NaCl*
Acetate	0	27	0
Ca	3	0	0
Cl	109	98	154
Gluconate	0	23	0
K	4	5	0
Lactate	28	0	0
Mg	0	3	0
Na	130	140	154
Osmolarity (mOsml/L)	272	294	310

administration, ongoing hemorrhage, third-spacing of fluid (ie, movement of fluid into the interstitial space or body cavities), heart disease, inappropriate vasomotor tone, or metabolic illness (eg, metabolic acidosis, hypoglycemia).

If an appropriate shock fluid volume has been administered, appears adequate, and the patient is still hypotensive, vasopressors may be considered.[10] ■ cb

Find More

Look for future articles on other types of shock in upcoming issues.

Table 3 Colloids & Their Chemical Properties[11]

Colloid	*Mean MW (kDa)*	*Molar Substitution*	*Colloid Oncotic Pressure (mm Hg)*
5% Human albumin	69	N/A	23.2 ± 0.1
6% Hetastarch in 0.9% NaCl	600	0.75	32.7 ± 0.2
6% Hetastarch in balanced electrolyte solution (Hextend)	670	0.75	37.9 ± 0.1
6% VetStarch	130	0.4	40
25% Human albumin	69	N/A	>200
Canine fresh frozen plasma	69	N/A	17.1 ± 0.6

See **Aids & Resources**, back page, for references & suggested reading.