LET'S TALK SHOCK

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Overview

Shock is considered a dynamic and complex clinical syndrome. It is a condition that is common in our patients and often difficult to define and understand. Early identification of shock and implementation of therapy is paramount to having a successful patient outcome.

What is Shock?

Shock can be defined as inadequate cellular energy production. It can also be defined as a condition in which tissue and cellular oxygen delivery does not meet oxygen demand. What this means is that shock occurs secondary to poor tissue perfusion, which causes poor oxygen delivery to vital tissues. Shock isn't a single entity, but rather the result of an underlying insult.

Types of Shock

Shock has been classified into many different types based on the different pathophysiologies. The three main types of shock are hypovolemic, distributive and cardiogenic, with hypovolemic shock being the most common type seen in small animal medicine. Depending on the disease process that is the cause of shock, it is not uncommon for patients to experience more than one type of shock. Regardless of the type, the goal of addressing shock is to optimize oxygen delivery to tissues.

Pathophysiology of the Different Shock States

Hypovolemic shock occurs when there is a decrease in circulating blood volume. Hypovolemia occurs most commonly from blood loss (hemorrhage), gastrointestinal loss (vomiting, diarrhea), urinary loss (polyuria), burn wounds, third-spacing of fluids or decreased intake of fluids. The loss of circulating blood volume results in decreased venous return to the heart (preload), which decreases cardiac output.

Distributive shock occurs when there is ineffective or inappropriate circulation and distribution of blood volume. This means there is adequate blood volume, but inadequate perfusion of said blood volume. This leads to a maldistribution of blood flow, in which vessels dilate and create peripheral blood pooling. During vasodilation, the vessels expand, making the normal blood volume insufficient and causing the blood to be displaced away from the heart and central circulation. Distributive shock occurs most commonly from vasodilatory states, such as sepsis, systemic inflammatory response syndrome (SIRS), anaphylaxis, heatstroke, or adverse drug reactions.

Cardiogenic shock occurs when there is pump failure causing failure of forward blood flow. This occurs when there is adequate blood volume but reduced cardiac output from cardiac dysfunction. Forward flow failure refers to decreased venous return to the aorta and systemic circulation. Without a normal functioning heart pump, tissue ischemia results from lack of blood perfusion and circulation. Causes of cardiac dysfunction occurs from dysrhythmias, poor contractility, valvular disease, structural or anatomical defects and congestive heart failure. Cardiac dysfunction results in an increased heart rate, decreased stroke volume, decreased cardiac output, decreased blood pressure, increased systemic vascular resistance and increases in pulmonary pressures. The main sign associated with cardiogenic shock is respiratory in nature.

Phases of Shock

The first stage of shock is known as the compensatory phase. During this time, the body has received an insult and is initiating a systemic response to compensate for decreased oxygen delivery to tissues. In this early stage it may not be as clinically obvious that shock is occurring, as compensatory mechanisms are working to maintain cardiac output and tissue perfusion. Clinical signs of this phase include mild changes in mentation, tachycardia (heart working harder to maintain cardiac output), prolonged capillary refill time (from vasoconstriction), tachypnea (from decreased oxygen), adequate to bounding pulse quality (heart trying to maintain perfusion), normal blood pressure (from vasoconstriction and tachycardia).

The second stage of shock is known as early decompensatory shock. The animal enters this phase after compensatory mechanisms have failed, and the body begins to succumb to prolonged poor oxygen delivery to tissues. Clinical signs include a moderate to severe depression in mentation, moderate to severe tachycardia, poor pulse quality, pale mucous membranes, hypotension and cool extremities.

The third and final stage of shock is known as late decompensatory shock. Progression to this phase indicates prolonged hypoperfusion lack of oxygen to tissues; this leads to organ failure and ultimately death. This phase is characterized by an obtunded or stuporous mentation, bradycardia, severe hypotension, and absent pulse quality.

Initial Assessment & Recognition of Shock

In addition to historical information, physical exam findings can also help in diagnosing shock. When performing a physical assessment to determine if a patient is in shock, focus should be on the six perfusion parameters. These parameters include mentation, heart rate, pulse quality, mucous membrane color, capillary refill time, and extremity temperature.

Mentation is the first parameter you will assess as you approach the patient and begin your assessment. During shock, decreased cerebral function from lack of oxygen supply is perhaps the earliest outward sign. A change in cerebral perfusion alters an animal's mentation within seconds. When classifying a patient's mentation, it can be classified as normal/alert, dull/depressed/obtunded, stuporous or comatose.

Heart rate is usually the first physical parameter you evaluate when going through your physical assessment. It is expected for an animal in shock to be tachycardic, as tachycardia is the body's compensatory response to hypoperfusion and inadequate oxygenation. During auscultation, it should also be noted if there are any irregularities in the heart rate, which can indicate an arrhythmia and would warrant performing an electrocardiogram (ECG).

Pulse quality should be taken in conjunction with heart rate auscultation when performing your initial assessment. Pulse quality refers to the difference between the systolic and diastolic arterial blood pressure. Pulse quality is reflective of adequate stroke volume, (amount of blood pumped by the heart each beat). As stroke volume is one of the determinants of cardiac output, a change can indicate compromised cardiac output, which occurs in shock. An animal's pulse quality can be described as normal, weak, bounding, or absent.

Mucous membrane color provides information about peripheral capillary perfusion. Normal mucous membrane color is pink, which indicates normal oxygenated hemoglobin in red blood cells present in the capillary beds. During the circulatory problems associated with shock, mucous membrane color changes in response to changes in perfusion. Pale mucous membranes are indicative of blood loss and

vasoconstriction, which can occur from hypovolemic or cardiogenic shock states. Injected (reddened) mucous membranes are indicative of vasodilation, which can occur from distributive shock states.

Capillary refill time (CRT) provides further information about peripheral perfusion. A prolonged CRT suggests poor perfusion from peripheral vasoconstriction, as with hypovolemic and cardiogenic shock states. A rapid CRT suggests a hyperdynamic (systolic-diastolic difference) state, which is associated with distributive shock states.

Extremity temperature is evaluated by feeling the paws and distal limbs of a patient, which should normally be warm to the touch. Cool extremities indicate poor perfusion, as cardiac output diverts blood flow to the central circulation.

In addition to the six perfusion parameters, you can also use blood pressure measurements. Blood pressure is sometimes the first thought-of parameter to measure in cases where shock is a concern. Too often, shock definitions have been associated around hypotension, which is misleading as to what shock actually is. Blood pressure measurements can be taken either indirectly, using oscillometric or Doppler methods, or directly, using an arterial catheter. To ensure consistency, the same limb and same cuff size should be used each time a blood pressure reading is taken.

Hypovolemic & Distributive Shock Treatment

The goal in treating hypovolemic and distributive shock states is to administer fluid resuscitation to increase intravascular volume, improve systemic perfusion, and restore oxygen delivery to tissues. During each therapeutic intervention, the patient should be reassessed to determine the next steps in treatment.

Venous access in a shock patient can sometimes be difficult to obtain due to compromised perfusion from cardiovascular compromise. However, it is most ideal to place a large gauge, short length IV catheter in the cephalic vein. Smaller gauge IV catheters create increased resistance to fluid flow, which is counterproductive as shock fluid resuscitation involves providing rapid, large volume fluid therapy.

Intravenous isotonic crystalloids are the mainstay fluid type for treating these shock states, as they have the most similar composition to the patient's extracellular fluid compartment. In canines, the shock dose of IV crystalloids is 60 to 90ml/kg. In felines, the shock dose of IV crystalloids is 45 to 60ml/kg. When delivering crystalloids, you should start with aliquots, such as ¼ or 1/2 the shock dose, and then reassess the patient. Ideally, IV crystalloids should be administered rapidly, over 10-15 minutes. As mentioned earlier, assessment of perfusion parameters and resuscitation endpoints should be used to guide fluid therapy.

Synthetic colloids are also a fluid option during shock resuscitation. Colloid solutions contain large molecules suspended in crystalloid solutions that help maintain intravascular volume because they don't as readily cross the blood vessel barrier. In recent years, there has been controversy and debate about the use of synthetic colloids in fluid resuscitation.

The use of hypertonic solutions can also be lifesaving in the emergency setting. Hypertonic saline is an excellent choice for rapid, small-volume resuscitation. Although short-lived (typically 30 minutes), the transient cardiovascular effects of administration may provide enough time for other therapies, such as crystalloids, to take full effect. Use of hypertonic saline is also desirable as you can deliver a much smaller volume to obtain the wanted restoration of intravascular volume effect.

In the case of hypovolemic shock from severe hemorrhage, blood component therapy is often used in addition to IV crystalloid resuscitation. Giving pRBCs increases oxygen content and giving FFP addresses coagulopathies. Ideally, blood products are administered slowly, through a filter, over 1-4 hours to be able to monitor for signs of a transfusion reaction and to prevent volume overload from rapid infusion.

Patients who are nonresponsive to shock doses of fluid resuscitation may require additional pharmacologic intervention. Other agents that can be used include vasopressors (norepinephrine, vasopressin), catecholamines (epinephrine) and sympathomimetics (dopamine, dobutamine). These agents work on receptors throughout the body to promote arterioconstriction and vasoconstriction (increasing blood pressure and heart rate), as well as improved heart contractility.

Cardiogenic Shock Treatment

The goal of treating the cardiogenic shock state is to improve oxygenation and restore adequate tissue perfusion. Treatment of cardiogenic shock differs from the other types of shock, and these patients are more susceptible to rapid decompensation.

Providing supplemental oxygen therapy is first and foremost. Oxygen can be delivered by flow-by face mask initially and can continue supportively as either nasal cannula or oxygen cage delivery system.

It is also very important to limit stress as much as possible. These animals can very easily decompensate; therefore, utilization of low-stress and minimal handling techniques should be performed. In these cases, stabilization and treatment often has to be performed in steps.

Obtaining venous access is also important, however, unlike the other shock states, a smaller gauge IV catheter is more acceptable and better tolerated in these patients.

The mainstay medication used to treat cardiogenic shock is furosemide. Furosemide is a loop diuretic that exerts it effects on the loop of Henle within the nephron. Furosemide increases urine production by increasing renal excretion of water and electrolytes; in doing so, it reduces intravascular volume and venous pressures. Furosemide is most commonly used to treat congestive heart failure, which is one of the most common causes of cardiogenic shock.

It is also important to remember to treat the underlying cause attributing to the cardiogenic shock state. Other pharmacologic intervention is most likely warranted depending on whatever heart condition is also present.

Nursing Care

Patients in shock present in an unstable condition, and the importance of the role of the veterinary technician cannot be overstated. Dedicated nursing care and close monitoring are essential during the stabilization and hospitalization periods of a shock patient.

Monitoring often involves one-on-one care for at least the first hour from time of presentation. During stabilization therapies, it is primarily the veterinary nurse assessing the patient and recording vital signs. As the patient becomes less critical, monitoring can decrease in frequency to every one to four hours.

There are also some special considerations to take into account depending on the type of shock a patient is experiencing. In the case of severe hemorrhage, administering a blood transfusion would

require additional frequent monitoring. In the case of severe GI losses, maintaining patient cleanliness as well as quantifying losses to be replaced will be necessary. In the case where vasopressor agents are used, more diligent monitoring of heart rate and blood pressure is required. In the case of congestive heart failure, monitoring of respiratory parameters (respiratory rate, respiratory effort, pulse oximeter) will also be essential.

Conclusion

Early recognition and prompt emergency therapy is essential to the successful outcome of a patient in shock. It is also important to serially examine and reevaluate perfusion parameters in shock patients and to determine end points of resuscitation.

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