Shock

Oxygen delivery to tissues is one of the primary functions of the cardiopulmonary system and of primary importance to the patient manifesting signs of circulatory failure. Oxygen delivery is a function of cardiac output and oxygen content of arterial blood

DO_2 (oxygen delivery) = CaO_2 (oxygen content) x CO (cardiac output)

In health, blood flow (cardiac output) is adjusted to meet the oxygen demands of the individual. This occurs primarily through changes in heart rate and vasomotor control of perfusion to maintain oxygenation of active tissues. Many acute disease states result in inadequate oxygen delivery to the tissues and tissue hypoxia. Initially, this drop in oxygen delivery can be overcome by compensatory increases in oxygen delivery variables and increases in oxygen extraction. When these mechanisms fail to restore oxygen homeostasis, global tissue hypoxia (shock) results. If the defect in the transport of oxygen to the vital tissues can be identified and corrected while the patient is undergoing supportive care, recovery is possible. Failure to correct poor perfusion will lead to decreased oxygen consumption in the tissues, organ dysfunction, and death.

Classification of Shock

Shock may be classified in many ways, including by common pathway or specific cause. It is helpful to think of shock in terms of broad categories before further defining the type of shock within each category. The broadest classification system includes three major and exceedingly different causes of shock:

Hypovolemic Shock

The primary defect in hypovolemic shock is an inadequate circulating volume. This can be from sudden massive blood loss as in surgery or trauma, or fluid loss from vomiting, diarrhea, or renal disease. Neurohormonal pathways detecting a drop in blood pressure will stimulate the renin-angiotensin-aldosterone system to conserve water through the action of antidiuretic hormone. Stimulation of the sympathetic nervous system leads to epinephrine and norepinephrine release from the adrenal gland. These hormones increase vascular tone in

an attempt to shunt circulation from the periphery to vital tissue beds and result in cool extremities and prolonged capillary refill time. Myocardial contractility is also increased through their action. As the patient begins to decompensate, tachycardia is a common finding, allowing maintenance of oxygen delivery in the face of diminished stroke volume. Concurrently, fluid shifts from interstitial fluid reserves in an attempt to preserve vital perfusion of the brain, heart, and kidneys while stealing supplies from other organs and tissues such as skeletal muscle and the gastrointestinal tract.

Cardiogenic Shock

Cardiogenic shock occurs when the pumping function of the heart is severely impaired, leading to circulatory failure. As with hypovolemic shock, the patient will be tachycardic, weak, oliguric, have cool extremities and weak pulses. The patient with cardiac failure may also have evidence of primary cardiac disease such as an auscultable murmur, ascites, jugular venous distention, pulmonary edema, or cardiac arrhythmias. The primary defect in oxygen delivery is a reduced cardiac output.

Cardiac Output = Heart Rate x Stroke Volume

Stroke volume is determined by preload, after-load, and contractility. Within limits, cardiac output increases as heart rate increases. High heart rates will eventually decrease cardiac output by impairing cardiac filling and subsequent stroke volume. Tachycardia may be the result of cardiac arrhythmia or simply a physiologic response to low volume. Specific antiarrhythmic therapy and correction of underlying causes of tachycardia should be used to normalize heart rate. Clinically significant bradyarrhythmias are less common. Hyperkalemia or decompensated shock (especially in the feline) can result in clinical bradycardia. Specific arrhythmias include sick sinus syndrome, second- and third-degree atrioventricular block. It is uncommon for these slow heart rates to require emergency treatment. Often these patients have already compensated with increased stroke volume and can be referred for pacemaker treatment.

Distributive Shock

Distributive shock is probably the most challenging of the shock syndromes and one of the most difficult to reverse. The primary defect with distributive shock is an abnormal systemic vasomotor response leading to peripheral vasodilation and maldistribution of blood flow.

Increases in vascular permeability can further exacerbate this shock syndrome. Both peripheral vasodilation and increased vascular permeability result in decreased perfusion of vital tissues. The many causes of distributive shock are summarized in

Causes of Distributive Shock

1-Sepsis and endotoxemia
2-Metabolic (renal or hepatic failure or severe acid
base imbalances)
3-Toxic (anesthetic overdose, heavy metal toxicosis)
4-Endocrinologic (adrenocortical insufficiency,
diabetic ketoacidosis)
5-Neurogenic (cerebral and spinal disease)

Components of other forms of shock may contribute to poor tissue oxygenation in distributive shock. Fluid loss into body cavities and interstitial spaces results in relative hypovolemia. The release of inflammatory mediators in septic shock can depress the myocardium, resulting in a cardiogenic component. Therapy must be directed at the underlying systemic defect. In sepsis, therapy consists of drainage and control of the infected focus. Because systemic inflammation resulting from sepsis and other inflammatory disease can affect oxygen delivery in many vital tissues, serial monitoring of many variables becomes necessary to treat the variety of problems an individual may face.

Treatment

Treatment of shock should be directed at the primary problem(s) while correcting the fluid deficit. Crystalloid fluids can be used initially to restore circulating volume. Crystalloids improve cardiac output and should not be withheld for fear of diluting the red blood cell mass]. Oxygen delivery is a function not only of oxygen content, but also of cardiac output. Improved stroke volume should offset the initial drop in packed cell volume as the patient's true level of anemia becomes apparent. If signs of shock persist as the patient becomes more anemic, a hemoglobin-containing fluid (whole blood, packed red blood cells, should be administered . Volumes of fluid for resuscitation should be tailored to the individual patient. It has long been recommended that the initial goal with crystalloid fluids is to give a blood volume (approximately 90 ml/kg dog, 60 ml/kg cat) in an hour.

management of shock

New technology is providing interesting new choices in the diagnosis and management of shock. Each intervention deserves investigation and, if proven practical and effective, will help clinicians manage this devastating syndrome. In the meantime, circulatory shock needs multimodal treatment with a basic goal: to improve oxygen delivery (DO_2) (*Table*). Improved oxygen delivery is achieved by optimizing oxygen content (CaO_2) and increasing oxygen saturation (SaO_2). If the packed cell volume is limiting DO_2, we can increase hemoglobin concentration with whole blood transfusion, packed red blood cell transfusion, or hemoglobin-based oxygen-carrying fluids (HBOC).

Table **Monitoring Variables, Goals, and Therapeutic Interventions used to Address Impaired Oxygen Delivery and Circulatory Shock**

Physiologic Variable	Optimal Values	Therapeutic Interventions
Systolic arterial pressure	> 90 mmHg	Fluids, vasoactive/inotropic
Mean arterial pressure	> 70 mmHg	Fluids, vasoactive/inotropic
Central venous pressure	< 3 cm H20 - normal 5-10 cm H2O when loading	Fluids when low, diuretics and venodilators when high
Urine output	> 1 ml/kg/hr	Fluids, diuretics, dopamine
Blood glucose	70 - 200 gm/dl	Nutrition, dextrose if low Regular Insulin if high
Total serum solids	3.5 - 5.5 gm/dl	Plasma, colloids, nutrition
Albumin	> 1.5 g/dl	Plasma, 25% HSA, nutrition
Arterial blood gasses	PaO2 > 70 mmHg	Supplemental oxygen
	PaCO2 < 35 mmHg	Ventilatory support
	HCO3 14 - 24	Fluids, bicarbonate
	pH 7.35 - 7.45	Fluids, ventilator therapy, bicarbonate
	Base deficit -2 to 2 mEq/l	Fluids, vasoactive/inotropic
	Lactate < 2 mmol/l	Fluids, vasoactive/inotropic
Heart rate	70 - 150 BPM	Fluids, analgesics, antiarrhythmics