

## **Shock and Shock Resuscitation: Parts 1 and 2**

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### **Introduction**

The most commonly encountered life-threatening problem in the critical patient is poor oxygen distribution and utilization by tissues. Oxygen has the highest extraction ratio of all blood constituents, making it the most flow-dependent blood component. Oxygen utilization ( $V_{O_2}$ ) is the measure of the body's overall metabolism. Inadequate  $V_{O_2}$  is the major pathogenic mechanism in the development of shock syndromes. The  $V_{O_2}$  can be rate limited by reduced supply (i.e. hemorrhage or cardiac failure) or flow maldistribution (i.e. trauma, surgical operations, anesthetics, sepsis, metabolic disorders). The pattern of oxygen delivery and utilization ( $D_{O_2}$  and  $V_{O_2}$ ) has been found to be a strong determinant of survival in humans.

The purpose of tissue oxygenation is to facilitate the production of adenosine triphosphate (ATP), the body's energy source. In the presence of oxygen, one glucose molecule is responsible for producing 38 ATP. In an anaerobic environment, only 2 ATP are produced per glucose molecule, with lactic acid as a detrimental end-product. Energy is required to power the sodium-potassium and calcium pumps within the cell. Tissue hypoxia causes depletion of energy stores and subsequent excessive amounts of sodium and calcium retained within the cell. The sodium draws water from the extracellular compartments and severe cellular swelling results. As intracellular calcium accumulates, lysosomal membranes breakdown, releasing enzymes which can destroy cell membranes. These enzymes activate kinins and prostaglandins which cause local vasodilation and increased capillary permeability. Interstitial edema, maldistribution of blood flow, and organ dysfunction result.

### **Capillary Dynamics**

The majority of fluids within the body are located within the intravascular, interstitial and intracellular compartments. The interstitial and intracellular compartments make up the extravascular space. The intracellular compartment is contained within a cell membrane that is freely permeable to water but not to charged particles. The interstitial space is composed of collagen fiber bundles, proteoglycan filaments and lymphatics and is located between the cells and the vasculature. The intravascular space is contained within the vascular network of arteries, arterioles, capillaries, venules, and veins.

Starlings Law defines the forces that affect the volume of fluid that is distributed between the intravascular and interstitial compartments. As the blood passes throughout the length of the capillary, the hydrostatic pressure gradient causes a continuous, dynamic movement of water and solutes into the interstitium. The dynamics of the various fluid compartments changes during shock. When the underlying pathology leads to a systemic inflammatory response, there is increased permeability of the capillaries and post capillary venules of the body. Albumin (69,000 daltons) will flux across the capillary membrane into the interstitium at and remote from the injured site as a result of cytokine action. Hypoalbuminemia associated with this systemic inflammatory response implies that the capillary pore size is at least 69,000 daltons in diameter when there is adequate liver function and no evidence of significant renal or intestinal albumin loss.

### **Pathophysiology**

Hypovolemic shock is a condition which results from a reduction in blood volume to the extent that ventricular filling, arterial blood pressure, peripheral blood flow, and  $V_{O_2}$  are inadequate to maintain cellular integrity. The most common causes are hemorrhage, wound fluid loss, burns, and loss of fluid into a third body fluid space. Within the walls of the aortic arch and at the bifurcation of the internal and external carotid arteries (carotid sinus), there are special pressure receptors, called baroreceptors, with their respective afferent nerve fibers (buffer nerves). When there is adequate pressure exerted on the baroreceptors, buffer nerves discharge at a slow rate, sending afferent impulses to the brain stem to inhibit the tonic discharge of the vasoconstrictor nerves and excite the cardio-inhibitory center.

In the early stages of hypovolemic shock, inadequate cardiac output leads to insufficient stretch of the baroreceptors, hypotension, and hypoperfusion. The inhibitory discharge in the buffer nerves is decreased and norepinephrine is released from the nerve endings and norepinephrine and epinephrine are released into the circulation from the adrenal gland. The overall result is an elevation in heart rate, increase in myocardial contractility, and vasoconstriction of the arteries and veins. These early changes represent the **compensatory stage** of shock and are represented clinically by an elevation in heart rate, normal or increased arterial pressure, normal or increased flow (bounding pulses, hyperemic mucous membranes, and rapid capillary refill time) and increased  $V_{O_2}$ . This stage is easily overlooked by the clinician since the patient may appear within normal limits. The heart rate is a key physical sign. Volume replacement is necessary, generally leading to favorable results if the cause is arrested. This phase is not seen in the cat except when there is extreme pain.

If fluid loss continues, the sympathetic stimulation will continue with more intensity, reducing the blood supply to the skin, muscles, viscera, and

kidneys, in an effort to maintain an effective circulating volume and to promote brain and heart perfusion. Uneven blood flow is the key event in this **middle or early decompensatory stage** of shock. ATP is utilized and glucose stores are depleted, resulting in free fatty acids being metabolized for energy. Prostaglandins, thromboxane A<sub>2</sub>, and leukotrienes lead to vasoconstriction, platelet aggregation, cardiac depression, leakage of lysosomal enzymes, and chemotaxis of white blood cells. Clinical signs associated with the middle stage of shock are low rectal temperature, poor pulses, pale mucous membranes, prolonged capillary refill time, and coolness of the limbs and skin. The heart rate is elevated and the animal usually has depressed mentation. Vigorous fluid therapy is required, and the prognosis becomes guarded.

In the cat, there is hypotension, hypothermia and normal or slow heart rate. The Hypothermia plays a significant role in the compensatory response that the cat is able to mount. Low rectal temperatures (< 98F) are associated with poor response of the adrenergic receptors to catecholamines. This can lead to inadequate compensatory vasoconstriction and poor cardiac response.

The terminal stages of shock are similar regardless of the etiology. Prolonged sympathetic overactivity leads to severe tissue hypoxia and decompensation of the vital organs (brain and heart). Heart rate slows, peripheral veins and arteries dilate, and blood pools, reducing blood volume and cardiac output further. This vicious cycle is the **terminal decompensatory stage** of shock which carries with it a grave prognosis. Clinical signs include heart failure, pulmonary edema, alteration of consciousness, severe hypotension, and abnormal respiratory patterns. Cardiopulmonary arrest is a common sequel.

### **Aggressive Resuscitation Procedures**

The primary objective of therapeutics in each case is to open up unevenly vasoconstricted microcirculatory networks and provide oxygen to the tissues.

1. **Oxygen administration:** At least a 40-60% oxygen concentration in inspired air should be maintained. This can be administered by endotracheal tube, transtracheal catheter, flow-by technique, mask, bag, or nasal cannula.
2. **Fluid Selection** Poor perfusion and dehydration are different problems, requiring different therapeutic strategies. Perfusion deficits are due to a loss of intravascular fluid volume (though heart failure must be ruled out as the cause). Replacement of these deficits should occur rapidly and involves giving enough solution to expand and maintain the intravascular space. Dehydration is an extravascular (primarily interstitial) volume deficit, replaced with crystalloids.. It is quite possible to have perfusion deficits without significant dehydration and dehydration without significant perfusion

deficits.

A *crystalloid* is a water based solution with small molecules that are permeable to the capillary membrane. The sodium and glucose concentrations of these fluids determine the osmolality and tonicity of the fluid and the distribution between the fluid compartments. A *colloid* fluid is a water based solution with both small molecules that are permeable to the capillary membrane as well as large molecules that can not cross the capillary membrane. Natural colloids consist of plasma proteins from donor animals and are administered as fresh frozen plasma, frozen plasma, whole blood, albumin concentrate, and stroma free hemoglobin. Synthetic colloids are man-made large molecules dissolved in normal saline. During initial resuscitation, a combination of synthetic colloid and crystalloids will be utilized.

**3. Select resuscitation end-points.** Successful resuscitation therapy depends upon administering quantities of fluids sufficient to reach specified end-points. This process is termed *end-point resuscitation*. Physical and hemodynamic parameters are the mainstay of monitored end-points. The need to avoid volume overload and increased capillary hydrostatic pressure in traumatized animals with closed cavity hemorrhage, on-going hemorrhage, and brain or lung trauma will dictate the end-points selected and end-point resuscitation technique employed.

*Supranormal end-point resuscitation:* For hypovolemic and SIRS shock, resuscitation of perfusion to supranormal values to increase oxygen delivery is recommended. Restoration of physical perfusion parameters (lowering of heart rate, stronger pulses, normal capillary refill time, pink mucous membranes) are used in conjunction with hemodynamic parameters. This technique is not to be used in the presence of closed cavity hemorrhage and lung or brain edema or hemorrhage. The sudden increase in capillary hydrostatic pressure can cause exacerbation of hemorrhage or edema.

*Hypotensive end-point resuscitation:* Traumatic shock with closed cavity hemorrhage or brain or lung edema warrants hypotensive resuscitation. The animal is resuscitated to end-points of improved physical perfusion parameters, but blood pressures remain in the low normal range rather than utilizing supranormal values. This is to avoid dislodging clots that may be providing life-saving hemostasis and to avoid a significant increase in hydrostatic pressure and worsening of brain or lung edema.

#### **4. Resuscitation Techniques**

Techniques for resuscitation with colloids include rapid volume intravascular resuscitation for dogs and small volume resuscitation for the dog and cat. Selection of either resuscitation technique will depend upon the species of the animal, the presence of brain or lung pathology, and the probability of ongoing or closed cavity hemorrhage. (see figures below)

