Update in Shock Resuscitation

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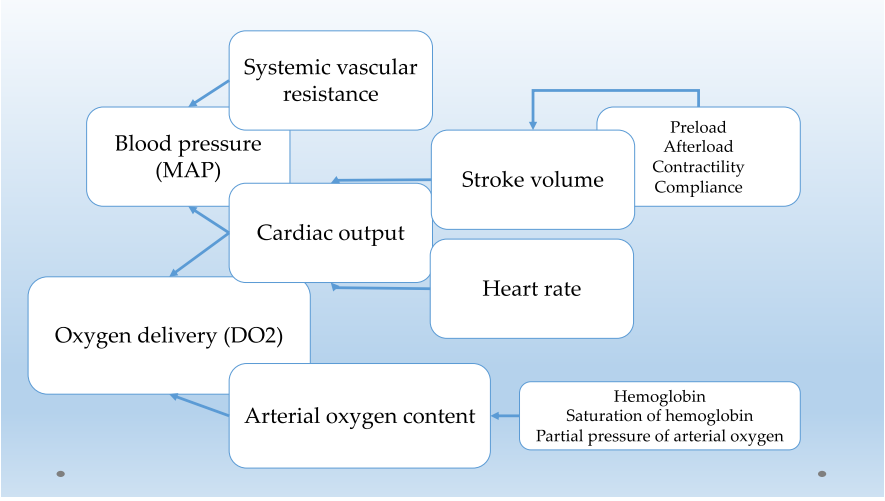
Shock pathophysiology review

Shock is the result of decreased oxygen delivery resulting in anaerobic metabolism, acidosis, organ dysfunction, and death. Initially, the body is able to compensate for shock in multiple ways, but as compensation is exhausted, hypotension results. Oxygen delivery is determined by cardiac output and arterial oxygen content. Blood pressure is a fine balance between cardiac output and systemic vascular resistance. Cardiac output is determined by stroke volume and heart rate. Multiple factors affect stroke volume including cardiac preload, afterload, contractility, and compliance. Arterial oxygen content is determined by hemoglobin, oxygen saturation of hemoglobin, and the partial pressure of arterial oxygen present.

Based on these components, there are three mechanisms necessary for normal tissue oxygen delivery: an effective cardiac pump, adequate pumping volume in the intravascular space, and an adequate response of the vascular tree to both endogenous and exogenous stimulants. Shock can be compounded by metabolic disturbances or hypoxemia.

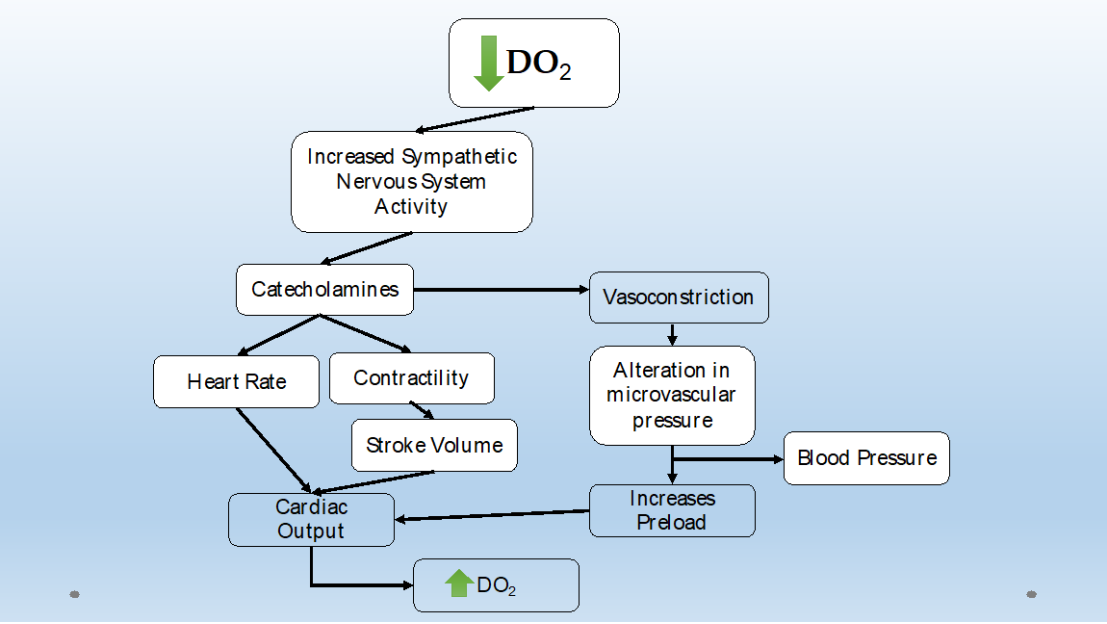


Compensatory mechanisms of shock

The normal physiologic response to shock is to address these three mechanisms. Fluid can redistribute from the interstitium to the intravascular space and the body has humoral responses to maintain blood pressure (and effective circulating volume). This helps the body maintain normal perfusion when afflicted with shock, but does make the initial stages of shock more difficult to recognize.

These humoral responses include catecholamine release, anti-diuretic hormone (ADH) release, renin-angiotensin-aldosterone system activation, and blood flow preferentially shunted to the brain and heart.

Catecholamines like epinephrine and norepinephrine promote vasoconstriction and improve cardiac contractility. Antidiuretic hormone, also known as vasopressin, provides additional vasoconstriction and aid in water retention. RAAS system activation causes further vasoconstriction via angiotensin II, and further water retention with aldosterone release.



Traditional treatment

The hallmarks of a decompensated shocky patient are identified on initial physical examination via perfusion parameters (tachycardia, weak pulses, cool extremities, dull mentation, prolonged CRT, and pale mucous membranes). As previously mentioned, the initial stages of shock are often missed because of compensation. Preliminary diagnostics may reveal hypotension or hyperlactatemia.

Traditional resuscitation would have one give a quarter shock dose (~20 ml/kg) of crystalloids over fifteen minutes and then re-evaluate. This process would be repeated until normotension is achieved or give stronger consideration to synthetic colloids, blood products, or vasopressors. The benefits of traditional resuscitation are that they are readily available, inexpensive, and quick and easily administered. However, there is an increased risk of dilutional coagulopathy, increased interstitial edema, and lack of oxygen carrying capacity.

The goals of resuscitation are to restore intravascular volume, improve cardiac output and oxygen delivery, restore end organ perfusion, and prevent hypoxic tissue injury. Historically, this is achieved with aggressive fluid therapy early in presentation. This is based on models from the 1930s and 50s of controlled hemorrhage. There are new studies that better mimic real hemorrhage more closely, and showed that immediate large volume resuscitation resulted in increased rate, volume, and duration of bleeding. Rapid infusion of intravenous fluids can lead to damage to the endothelial glycocalyx, an increase in intravascular permeability, worsened tissue edema, and potential fluid overload. Delayed resuscitation also improved hemodynamic parameters and improved survival rates.

Newer thoughts in shock therapy

Over the past few years, there has been a trend to giving smaller fluid boluses over longer periods of times, i.e. the “slolus”. This portends giving closer to 10-15 ml/kg in dogs over 30-45 minutes, or 5-10 ml/kg in cats. This process similarly still requires constant reassessment to determine efficacy in each patient. Giving a smaller amount of fluid over a longer period of time leads to less edema, less hypervolemia, and better identification of cause of shock. This lends to better long term outcomes and less morbidity. Interstitial edema will be slower to form because there is decreased shear stress upon the vascular walls causing less disruption to the endothelial glycocalyx. This has been hypothesized to help prevent fluid redistribution. Crystalloids typically redistribute from the intravascular space in 30-60 minutes.

In patients that are septic, this approach helps to reduce volume overload associated with aggressive fluid therapy in septic patients. Often times, sepsis causes distributive shock, not hypovolemic shock, and also increases vascular permeability and blunts catecholamine response. So this approach helps to limit causing patients to become hypervolemic. Hypervolemia can lead to worsened metabolic acidosis, worsened hypothermia, and worsened coagulopathies. Remember this is different from permissive hypotension, which is a concept used to treat hemorrhagic shock. The goal of this model is life support with avoidance of a rapid increase in intravascular hydrostatic pressure and mean arterial blood pressure that could potentially dislodge newly formed clots. However, it is still applicable, as there will be a reduced risk of decreased blood viscosity and dilution of coagulation factors which can worsen hemorrhage. It also helps to decrease the use of unnecessary blood products which in veterinary medicine is a limited resource.

Subnormal blood pressures are beneficial for both hypovolemic and hemorrhagic shock as there is less of a risk of bleeding and decreased formation of interstitial edema. The end goals are the same as previously.

Take home points

Remember that in many newer studies, more aggressive fluid therapy does not result in vital organ perfusion, but may result in higher mortality. Our goals remain to restore effective circulating volume, optimize oxygen delivery to tissues, and ensure end organ perfusion. Despite all of the studies, it is unrealistic to assume that there is ever one fluid therapy protocol that will fit every situation. However, more and more evidence shows that too much fluid can be harmful rather than helpful.

References upon request