Successful treatment of shock can be a difficult achievement in the best of circumstances. The ultimate treatment of shock is to correct the underlying cause. In some cases, such as simple hypovolemia, this may be more somewhat straightforward. Yet, for many of the patients in shock, we need the stopgap measure of optimizing delivery of oxygen while the underlying cause is investigated. While in theory, this seems simple, the complexity of actual patient conditions make this a challenging puzzle.

Early goal Directed Therapy (EGDT)

When we use traditional resuscitative parameters in shock (normalization of blood pressure, capillary refill time, heart rate), we have not actually proven correction of the shock state. In humans, it was found that up to 85% of patients had ongoing tissue hypoxia despite normalization of these classic vital parameters. Since the 1980’s, therapeutic approaches to the treatment of true shock (tissue hypoxia) have been debated, studied and refined in human medicine. The landmark studies by Rivers et al proved the positive effect of treatment aimed at rapidly optimizing oxygen delivery in septic shock patients. Early goal directed therapy (EGDT) is a therapeutic approach aimed at optimizing the delivery of oxygen (DO2) to the consumption of oxygen (VO2).

Example of an EGDT algorithm used for human patients presenting in shock.
Veterinary EGDT

While the data unequivocally shows a positive effect in initial outcomes of human shock patients using EGDT, the veterinary evidence is still in its infancy. Determining optimal end-points in our resuscitative efforts are still mostly unknown. Zacher et al (JAVMA 2010) found a strong survival correlation between GDV patients that had hyperlactemia reductions of >50% with intravenous fluids preoperatively (100% vs 15%). Conti-Patara et al (JVECC 2012) evaluated tissue perfusion parameters (lactate, BD, ScvO2) in dogs with pyometra and shock to guide ICU therapy. They found that continued evidence of oxygen debt was associated with non-survivors.

4 Phases of Shock Treatment

1. Salvage: The goal initially is to restore a minimum blood pressure and cardiac output for immediate survival.
2. Optimization: The goal is to increase tissue oxygen availability.
3. Stabilization: After restoring oxygen availability, supporting the damaged organs becomes the priority.
4. De-escalation: Once patient homeostasis begins to return, decreasing the vasoactive medications and fluids to permit endogenous control to resume.

Fluid Resuscitation

Crystalloids are the mainstay of initial shock therapy for the vast majority of cases. The type of crystalloid chosen initially will depend on several factors. Although, almost any isotonic fluid (normosol, plasmalyte, saline) is appropriate for volume replacement. The volume is to start with depends on the blood volume of the patient (dogs have approximately 80-90 ml/kg, while cats have 50-60 ml/kg).

- The fluid challenge
  - Start with ¼ to ½ of the patients blood volume of an isotonic crystalloid. You will give it over 15-20 minutes and then reassess the cardiovascular status. If the heart rate did not decrease or the blood pressure is still low, this can repeated.
  - If low oncotic pressure, adding a synthetic colloid would be indicated. Typically, start with 2ml/kg IV for cats and 5ml/kg IV for dogs over 30 minutes. Current products include Hetastarch and the newer Vetstarch.
    - Synthetic colloids have been shown to cause some coagulation changes and should be used only as needed.
  - Hypertonic saline (7.5% NaCl) is useful for small volume resuscitation and traumatic brain injury patients. Typically start with 2-4 ml/kg IV.
  - Intravenous fluids can lead to severe life threatening conditions, therefore frequent reassessment is necessary.
  - The endpoint of fluid resuscitation would be correction of macro-parameters (heart rate, pulse quality, blood pressure) and micro-parameters (lactate, base excess).
Blood component therapy is often indicated in many disease states. If available, it can be extremely useful to help get a patient out of shock when oxygen carrying ability is ineffectual (such as anemia). Starting with a pRBC transfusion at 10-20 ml/kg, plasma at 10-20 ml/kg, or fresh whole blood at 20-30 ml/kg.

Vasoactive Agents

In some shock states, fluid volume alone will not be enough. If the patient continues to be in shock as evidenced by either macro or micro perfusion parameters, vasoactive drugs may be necessary. Since oxygen delivery to the tissue is dependent upon both cardiac output and systemic vascular resistance, enhancing cardiac function or vascular tone may be required.

- Vasopressors
  - Dopamine
    - Used to enhance cardiac contractility via beta receptors
    - Start with 5 mcg/kg/min IV CRI
    - You can increase the dosage every few minutes to maximize its effect, although watch for tachycardia.
  - Norepinephrine
    - Used to cause vasoconstriction to increase blood pressure via alpha receptors
    - Start with 0.1 mcg/kg/min IV CRI
    - Caution with vasoconstrictors as they can cause ventricular dysrhythmias and impair tissue perfusion.
  - Vasopressin
    - Used to cause vasoconstriction via V1 receptors
    - Start with 0.5 mU/kg/min IV CRI
    - This may be more useful than other catecholamines in acidotic patients

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