FLUIDS AND HEMODINAMIC MANAGEMENT
IN TRAUMA PATIENTS

Luis H. Tello, MV, MSc, DVM
Lead Dr. Portland Hospital
International Medical Advisor
Banfield Pet Hospital, USA

The hemodynamic management of the patients after a multiple trauma requires a comprehensive approach and the usage of multiple monitoring and therapeutics. These patients may respond initially to a regular symptomatically therapy, but in more complicated cases it is imperative for the clinicians to understand the physiopathology and the progression of the signs to interpret their clinical data and adjust the treatment accordingly.

Current definition for shock is an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation, regardless of cause. The lack of an adequate tissue perfusion leads to a decreased oxygen delivery to cells, tissues and organs, leading to anaerobic metabolism and development of acidosis. Tissue acidosis and increasing oxygen debt eventually progress to organ and cell dysfunction, multiple organ dysfunction and patients death.

For many years the statement: “The fluid therapy is one of the cornerstones in the trauma patients” has been accepted with no restrictions. While more evidence is available, questions rise about the best way to do it. There are many goals for the fluid therapy in trauma patients: treating the shock and hemorrhage effects, reversing the effects of the fluid shift from the vascular to the interstitial space, improvement of blood pressure and tissue perfusion. All of these events are primarily cause of death in those patients.

General Physiology:

Body fluid distribution

The total body water ranges from 55 – 70% of the lean body weight. In the average adult dog the total body water is about 65%. Thus in a 15 Kg dog the total body water will equal approximately 9 liters. This data is not clear in the cat, and some evidence suggest a number closer to 55%.
The body water is distributed mainly into 2 compartments:
A) the intracellular fluid space, and
B) the extracellular fluid space.
About 66% of the total body water stay in the intracellular fluid space and 33% in the extracellular fluid space.

The extracellular fluid space is further subdivided into two fluid containing compartments:
1) – the interstitial space (containing 75% of the extracellular fluid space water) and
2) – the intravascular space (containing 25% of the extracellular fluid space water).

When water is added to one compartment, it distributes evenly across the total body water and the amount of volume added to any given compartment, is proportional to its fractional representation of the total body water. Thus, if one liter of free water is placed in the intravascular space, there will be a minimal increase in the intravascular volume after equilibrium takes place. In fact, approximately 30 minutes after rapid volume infusion of free water, only 1/10th of the volume infused remains in the intravascular space.

TRAUMA PATIENTS

Blood loss is one the most common consequences in a trauma patients, therefore it is not well tolerated in the animal. Although loss of important sections of organs as adrenal glands, liver, kidneys, lungs does not lead to the death of an animal, loss of 35% of the blood volume can be fatal. The dangers of hemorrhage are related to a cardiovascular system that operate with a relatively small volume and respond to the Frank – Starling curve in the heart with the possible purpose of to limit cardiac work and conserve energy.

Clinically, water deficits can be divided into perfusion deficits (intravascular space) and dehydration (interstitium). A tissue perfusion deficit is associated with decreased oxygen delivery for the cells, less production of energy and therefore is considered a live-threatening condition. Based on the cause and physiologic processes that lead to hypoperfusion, the type of shock may be classified as: hypovolemic, distributive, obstructive and/or cardiogenic. All of them except the pure cardiogenic, requires a fluid therapy plan and therapy.

In an average and healthy animal, until 15% loss of blood volume does not require intervention with intravenous fluids. With a loss of this volume, there is a three phase compensatory response to mild hemorrhage:

Stage I. Within one hour of a mild to moderate hemorrhage, interstitial fluid begins to move into the capillaries. This fluid shift continues for 36 – 40 hours. The movement of fluid from the interstitial space leaves an interstitial fluid deficit that could be interpreted as “dehydration”. That dehydration are rapidly paid when fluids are replaced into the vascular space.

Stage II. The ongoing loss of blood activates the renin/angiotensin/aldosterone system, which leads to sodium conservation by the kidneys. Because sodium distributes primarily in the interstitial space (80% of sodium is extravascular), the retained sodium replenishes the fluid deficit in the interstitial space. There is an active variation in the hematocrit during the fluid therapy in these patients. As any fluid is infused, crystalloid or
colloid, a immediate fall in the Hct can be expected. As the intravenous resuscitation fluids redistribute, the PVC rises again. Serum protein shows similar trends as Hct.

Endogenous restoration of depleted intravascular volume occurs through the movement of interstitial fluids into the intravascular space. Catecholamines mediate arteriolar vasoconstriction which diminishes capillary bed hydrostatic pressure favoring influx of interstitial fluid into the vascular tree distal to the arteriolar constriction. Subsequently, the lymphatic flow pattern returns the plasma proteins to the intravascular space. Increases in interstitial pressures caused by crystalloid distribution into the interstitial space may augment lymphatic flow thus the “protein - refill” mechanism. This process combined with increased albumin synthesis and spontaneous diuresis secondary to volume repletion explains the return of serum protein levels after crystalloid resuscitation.

Stage III. Within a few hours after mild/moderate hemorrhage, the bone marrow begins to increase production of erythrocytes. Unfortunately, their replacement is slow with only 15 - 20 ml of cell volume being produced daily and complete replacement requiring a couple of months.

**FLUID THERAPY IN TRAUMA PATIENTS**

Intravascular fluid deficits due to trauma can be replaced with isotonic crystalloid solutions, colloids, hypertonic saline or combinations of these. Isotonic crystalloid solutions such as Normosol –R, lactated ringer's solution (LRS) or 0.9% NaCl will be distributed over the complete extracellular space and only ¼ of the administered fluid will remain intravascular. This may lead to edema formation, especially in patients with a SIRS o sepsis.

The usage of colloid solutions in combination with a crystalloid are widely spread in veterinary practice, as colloids contain large molecules that will stay intravascular and will additionally exert an oncotic effect that keeps the crystalloid solution in the intravascular compartment. However some evidence released lately in human patients, seems to point toward more side effects and complications with the combination that with pure crystalloids. Further research is necessary on this subject to clarify protocols and recommendations.

**USING CRYSTALLOID FLUIDS**

Crystalloid fluids are mixtures of sodium chloride and other physiologically active solutes. They are generally isotonic with plasma and have sodium as their major osmotically active particle. The distribution of sodium determines the distribution of infused crystalloid fluids. Sodium is the major solute in the extravascular space and 75% of the extracellular space is extravascular. Therefore, infused sodium will reside primarily outside the vascular compartment.

Aggressive fluid resuscitation with crystalloid solutions as Normosol-R, LRS or normal saline) with an end goal of 3 volumes of crystalloid for each volume of blood lost have been widely accepted as the standard management of hemorrhagic shock secondary to trauma. However some questions rise about the safety in such approach due to the risk of overload, elevated CVP, reduction of the myocardium perfusion, pulmonary and peripheral edema and dilution coagulopathy.

In feline patients there are more doubts and red flags because, the intravascular blood volume is much
smaller than in dogs and the total shock bolus of crystalloid is suggested around 45-60 ml/kg. A protocol for cats is initially infusing 10-30 ml/kg rapidly over 10-20 minutes, while the animal is carefully observed for a response, or for evidence of complications. This dose can then be repeated if necessary. If the hemodynamic values (MM, CRT, BP, HR and mentation) in the patient begin to improve, administration may be decreased down before the total bolus has been given. Clinical values as Packed cell volume (PCV), total solids (TS), electrolytes and blood glucose should be monitored before, during, and after such therapy.

During phase I there is an interstitial fluid deficit which needs to be replaced during early fluid therapy. In fact, the goal of fluid therapy for mild hemorrhage is to fill the interstitial space, not the vascular space. This is the rationale for using crystalloid (sodium-containing) fluids for the resuscitation of mild hemorrhage.

Sodium-containing fluids are well suited for the replacement of extracellular fluid losses (dehydration) and for replacement of blood volume. Their use is directed to replacement of the interstitial fluid deficits seen in hemorrhage. The significance of the deficit has been questioned but latest evidence seem to support the usage of such fluids.

Nevertheless, crystalloid solutions have proven to be effective in the resuscitation of animals with acute hemorrhage and they continue to be popular resuscitation fluids for trauma victims.

### Table I. Composition of crystalloid solutions.

<table>
<thead>
<tr>
<th></th>
<th>Plasma 0.9%</th>
<th>Saline Ringer</th>
<th>Lactate</th>
<th>Normosol - R</th>
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<tr>
<td>Na</td>
<td>145</td>
<td>154</td>
<td>130</td>
<td>140</td>
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<td>Cl</td>
<td>103</td>
<td>154</td>
<td>109</td>
<td>98</td>
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<td>K</td>
<td>4 - 5</td>
<td>-</td>
<td>4</td>
<td>5</td>
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<td>Ca/Mg</td>
<td>5/2</td>
<td>-</td>
<td>3/0</td>
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<td>Buffer (27)</td>
<td>HCO₃ (22)</td>
<td>-</td>
<td>Lactate (28)</td>
<td>Acetate</td>
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<td>Gluconate (23)</td>
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<td>pH</td>
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<td>7.4</td>
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<tr>
<td>Osmolality</td>
<td>290</td>
<td>308</td>
<td>273</td>
<td>295</td>
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**Hypertonic Crystalloids**

Some research has suggested that the use of small volumes of concentrate crystalloid solutions is recommended because the reduced volumes of fluid required decreasing the risks of fluid overload and therefore reducing the chance for developing pulmonary edema.

Hypertonic saline (1.7%, 3%, 5%, 7.5%) is used in hypovolemic and traumatic shock with or without hyperoncotic substances. Adding the hyperoncotic solutions, the duration of effect is prolonged over their very short action. These solutions are effective and provide prompt volume expansion with significantly less volume than necessary with conventional crystalloids. Additionally, a decrease in the intracranial pressure has been observed in trauma patients.
Patients with late decompensated shock, terminal shock or concurrent head trauma may benefit from treatment with hypertonic saline at small resuscitation volume: 4 ml/kg slowly IV. The increase in intravascular osmolarity will lead to a substantial water shift from the interstitial space into the intravascular space but only for a short period of time; around 30 minutes when using NaCl 7.5%. Some research suggest that the effect may be prolonged with the addition of a colloid as Dextran or HAES.

Experimental studies show hypertonic saline solutions will improve microcirculatory flow, possibly by reducing shock - induced endothelial swelling. With endotoxic shock models, hypertonic saline is more effective than isotonic crystalloids in proving cardiac output and oxygen transport but only very transiently. In another study no benefit was seen. Controlled trials in veterinary medicine are not available.

The major drawback of hypertonic saline resuscitation is the very short duration of response. Other concerns regarding the use of hypertonic saline include producing a hypertonic state, the prompt movement of sodium to the interstitial space, the water shifts from the interstitium and intracellular space, and the potential for a rebound interstitial edema.

**COLLOID FLUIDS**

Colloids are large molecular weight substances that do not readily pass across capillary walls. The particles retained in the vascular space will exert an osmotic force that keeps fluid in the blood vessels.

Hypovolemia represents the most life - threatening aspect of acute hemorrhage. Because colloids are more effective than crystalloids for increasing vascular volume, colloid resuscitation should be more useful with severe bleeding.

While colloids remain within the vasculature they provide more consistent plasma volume expansion. The large molecules cause increases in colloid osmotic pressure that works keeping the fluid within the circulation that would otherwise be lost to the interstitium. Base on this effect has been widely accepted that small volumes of colloid need be used compared to crystalloid, for a similar cardiovascular effect.

Although the decreased interstitial fluid accumulation is difficult to quantify clinically, we have anecdotally seen increased survival and decreased morbidity in critical patients as our use of synthetic colloid solutions has increased. Synthetic colloid solutions should be considered in patients that need high rates of intravenous fluid support, particularly if hypoproteinemia is present.

The main colloid substance in the blood is a natural protein: albumin. Albumin sustain the oncotic pressure, but also albumin was shown to induce a sustained increase in the glutathione in lung epithelial cells and to inhibit some deleterious cytoquine. In hypoproteinemic patients with acute lung injury (ALI) who were given albumin, the rise in plasma thiols and antioxidant capacity was recently demonstrated. Albumin administration favorably influenced plasma thiol-dependent antioxidant status as well as levels of protein oxidative damage. Thus, it appears that albumin may have multiple physiologic effects, including boosting the antioxidant potential and modulation of redox balance, hence attenuating inflammatory processes.
In human medicine it was accepted that there would be a relationship between the albumin level and disease severity in critically ill patients as multiple trauma. However, no clear correlation has been established by the evidence.

COLLOID OR CRISTALLOID

The choice of colloids v/s crystalloids for volume resuscitation in trauma patients has long been a subject of debate among critical care practitioners, primarily because there are data to support arguments for both forms of therapy.

In human medicine in 1998, the British Medical Journal published a meta-analysis on the use of albumin in the critically ill patient; 30 randomized, controlled trials involving 1,419 patients were analysed. The conclusion was that albumin may actually increase mortality.

This review had an impact on medicine practice, influencing clinicians to use less albumin but was later criticized as being flawed when subsequent reviews did not substantiate the authors' conclusions.

A recent review of 8 randomized (human) clinical trials comparing the effects of colloid versus crystalloid solutions on survival showed a 5.7% relative difference in mortality in favor of crystalloid therapy. However, a 12.3% difference in mortality rate was found in trauma patients in favor of colloids. The confidence intervals for these studies were large and one must question whether the studies were appropriately assigned to trauma or no-trauma groups.

Head trauma with hemorrhage is one of the severe restrictions in the use of colloids by the risk to colloids leaving the vascular can draw additional fluid to the area, worsening the cerebral edema and total cerebral contain of water.

In a more recent analysis of these trials the pooled date demonstrated a 13.4% mortality rate for crystalloid-treated human patients and a 21.25% mortality rate for colloid-treated patients (not statistically significant at a p = 0.01 level). In this same study, when the trials were subdivided into the apparent severity of the underlying processes, again no statistically significant difference was noted between the two treatment groups, although there was a tendency to a higher mortality in colloid-treated patients with more severe illness.

Although mortality is but one factor in assessing colloids versus crystalloids, the following recommendations have been made:

- Prompt and adequate fluid therapy is the mainstay of treatment of septic shock
- Colloid and crystalloid fluids lower hemoglobin concentration, oxygen carrying capacity and whole blood viscosity. The optimum hematocrit in septic shock has not been defined but a value of 30 - 35% seem acceptable
- The choice of fluid should take into account its effect on plasma oncotic pressure (COP). Severe decreases in COP should be avoided.
- Volume treatment should be individualized and titrated to individual needs.
HEMODYNAMIC EFFICACY IN TRAUMA PATIENTS

There is no doubt that both, crystalloids and colloids, will adequately resuscitate shock patients. With crystalloids the amount of fluid necessary to reach the same hemodynamic endpoint is usually 2 - 4 times higher with crystalloids than with colloids. This results in significant changes in body weight and induces the risk of systemic edema.

There is convincing evidence that colloid - containing fluids act more promptly than crystalloid solutions in restoring hemodynamic stability. In a study of 600 hypotensive human patients, the mean resuscitation time was shorter with colloids. a similar finding in traumatized humans showed for a given volume of fluids infused, colloid solutions expand the plasma volume to a greater extent than crystalloid solutions.

Hemodynamic and oxygen transport variables with colloids are more pronounced than with crystalloids. In a study of postoperative patients, plasma volumes before and after infusion of 1L of colloid, shows the advantage clearly to the colloids.

PULMONARY FUNCTION

One of the core issues in the colloid - crystalloid controversy is the potential difference of inducing pulmonary edema with these fluids, specially in patients that have previous complications as pulmonary contusions, common in multiple trauma. While the infusion of crystalloids does result in a significant and prolonged decline in serum albumin concentration and colloid oncotic pressure (COP), infusion of colloids help to maintain or even increase COP.

A low COP can promote the development of pulmonary edema microvascular hydrostatic pressure increases above normal. However, the evidence reveals that increases in hydrostatic pressure are more likely to result in pulmonary edema than comparable decreases in COP. Therefore, in a clinical perspective, hydrostatic pressure is more important in fluid exchange in the lung than COP, so fluid overload can cause more complications than a reduction in the level of albumin

Blood substitute call Oxyglobin should provide advantages to animals with reduced oxygen carrying capacity, but not many trials has been evaluated to support a generalized recommendation.

References available upon request