

Fluid Therapy for the Trauma Patient

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KEY WORDS

- crystalloids
- isotonic crystalloids
- hypertonic crystalloids
- colloids
- synthetic colloids
- natural colloids
- fluid therapy plan

"Triage with a gurney—stat!...place a pressure wrap on that artery...we need oxygen...do a cutdown on the jugular vein and get me a large-bore catheter...push in a liter of crystalloids and a bag of hetastarch...warm up a unit of whole blood...I'll go talk with the owner...." Whether the life-threatening injury has been caused by a moving vehicle, a fall from an incredible height, malicious actions, or a big dog–little dog battle, this is the "Drama of Trauma."

Maintaining a patent airway, supporting oxygenation and ventilation, and promoting adequate circulation are crucial to stabilizing the traumatized animal. Hypovolemia, hemorrhage, cardiac arrhythmias, myocardial depression, and release of inflammatory mediators into the systemic circulation can occur due to trauma and can lead to insufficient venous return and decreased cardiac output. The shock response is initiated, which can be lifesaving in the early stages; however, ongoing tissue hypoxia and cellular energy depletion will lead to decompensation and eventual death. The intravascular volume must be rapidly but cautiously expanded to eliminate the shock response.

Knowledge of capillary dynamics and the composition of administered fluids is necessary to establish the optimal plan for posttrauma shock resuscitation. The success of the fluid resuscitation plan depends on administering the right fluid or combination of fluids at the right rate, in the right volume, and at the right time. The goal is to promote circulation without exacerbating hemorrhage or contributing to interstitial fluid overload. This requires selecting appropriate resuscitation end points and utilizing optimal administration techniques. The fluid therapy plan must be

rapidly formulated—often within seconds of presentation—with little margin for error.

CAPILLARY DYNAMICS

The majority of bodily fluids 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.

Within the intravascular compartment, the capillaries are the site of normal fluid exchange between the intravascular and interstitial spaces. The capillary "membrane" is composed of endothelial cells and a basement membrane. Lipid-soluble molecules such as oxygen and carbon dioxide are freely permeable and rapidly cross through the endothelial cells to the area of lower concentration. Non-lipid-soluble particles, including water, must diffuse through tight endothelial intercellular clefts. The size of the solute determines whether that particle can move freely across this intact capillary "membrane" (Figure 1).

Starling's Law defines the forces affecting the volume of fluid that is distributed between the intravascular and interstitial compartments. As the blood passes through the length of the capillary, the hydrostatic pressure gradient causes a continuous, dynamic movement of water and solutes into the interstitium. Approximately 90% of the volume of fluid that leaves the normal arterial capillary is taken up at the venular end of the capillary. The remaining 10% of filtered fluid volume, along with waste products and proteins, is deposited into the lymphatics to maintain volume equilibrium. When the ability of the lymphatics to remove excess fluid is overwhelmed, interstitial edema occurs.

In contrast to most of the capillaries of the body, the capillaries of the brain have tight intercellular clefts that prevent most small ions from leaving. Brain capillaries are also supported by "glial feet," which are small projections from surrounding glia that add support on all sides of the capillary. This helps prevent overstretching of the capillaries in situations of high pressure, thereby minimizing fluid extravasation into brain tissue.¹

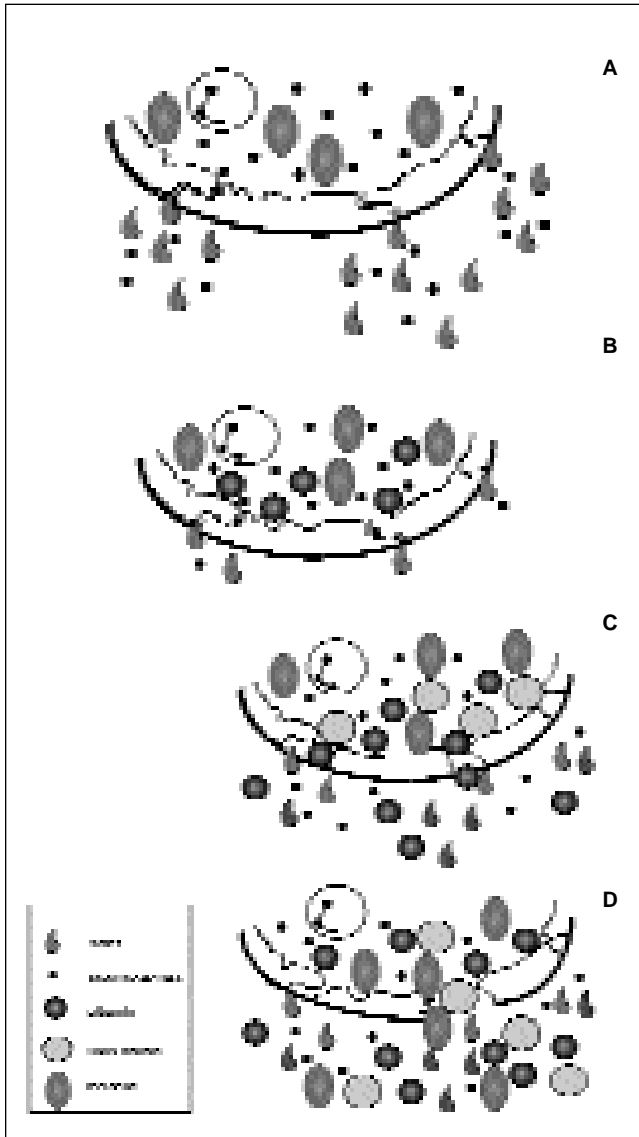


Figure 1. Cross-sectional schematics of a capillary illustrating the importance of capillary pore size. (A) The normal, tight endothelial junction allows only water and small ions to freely penetrate the capillary "membrane." (B) The administration of low-molecular weight colloids (which are too large to pass through the normal, tight junction) will help retain fluid within the vascular space. (C) When holes in the capillary "membrane" are sufficiently large to allow low-molecular weight colloids to pass, it is necessary to administer high-molecular weight (HMW) colloids to aid in retaining the fluid within the vascular space. (D) When there is rupture in the capillary that leaves a large hole, everything in the capillary will pass freely into the interstitial space at that site.

The dynamics of the various fluid compartments changes during shock. Separation of capillary endothelial junctions during reperfusion of hypoxic tissue and active transcellular-vesicular transport of proteins result in leakage of albumin and fluids out of the intravascular space.^{2,3} Intracellular and interstitial water content increases following hemorrhagic shock as a result of electrogenic pump depression.⁴⁻⁶ The resultant hypovolemia and edema affect oxygen transport

and diffusion to the cell, adding insult to the already dysfunctional cell membrane.⁷

When traumatic injuries lead to a systemic inflammatory response, permeability of the capillaries and postcapillary venules increases. Vascular leakage of albumin occurs at the postcapillary venules with diameters of 10 to 50 μm .⁸ These endothelial cells contract and pull away from each other, resulting in large interendothelial gaps.^{9,10} 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.¹¹⁻¹³ 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.

FLUID COMPOSITION

The number, charge, and size of the particles in water, together with capillary dynamics, determine the movement of that fluid throughout the different fluid compartments. Water moves across the capillary membrane by osmosis. Remember that the cell membrane is permeable to water but not to small ions; thus the ability of water to move across the cellular membrane depends on the osmolality of the administered fluid compared with that of intracellular fluid. This characteristic of administered fluids is termed *tonicity*.

In addition to the number and charge of particles in administered fluids, the size of the particle helps determine its destination. Proteins are the only dissolved substances in normal plasma that do not diffuse readily through the capillary membrane, which maintains the protein concentration in plasma at approximately three times that in interstitial fluid. The proteins are responsible for the osmotic pressure at the capillary membrane. To distinguish osmotic pressure at the capillary membrane from that exerted at the cell membrane, the term *colloid osmotic pressure* or *colloid oncotic pressure* (COP) is used. The Gibbs-Donnan effect causes COP to be about 50% greater than that caused by the proteins alone.

There are two major categories of administered fluids: crystalloids and colloids. 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 among the fluid compartments. The most commonly administered crystalloids and their specific characteristics are listed in Table 1.

A *colloid* is a water-based solution with both small

Table 1
Characteristics and Contents of Commonly Used Crystalloids and Colloids

Name	Fluid Compartment	Osmolarity (mOsm/L)	pH	Na ⁺ (mEq/L)	Cl ⁻ (mEq/L)	K ⁺ (mEq/L)	Mg ⁺⁺ (mEq/L)	Ca ⁺⁺ (mEq/L)	Dextrose (g/L)	Buffer	COP (mmHg)
CRYSTALLOIDS											
Replacement											
0.9% saline	Extracellular	308 (isotonic)	5.0	154	154	0	0	0	0	None	0
Lactated Ringer's solution	Extracellular	275 (isotonic)	6.5	130	109	4	0	3	0	Lactate	0
Plasmalyte-A [®] pH7.4 ^a	Extracellular	294 (isotonic)	7.4	140	98	5	3	0	0	Acetate, gluconate	0
Normosol-R ^{®b}	Extracellular	295 (isotonic)	5.5–7	140	98	5	3	0	0	Acetate, gluconate	0
7.0% saline	Extracellular	2396 (hypertonic)	—	1197	1197	0	0	0	0	None	0
5% dextrose in water	Intracellular	252 (hypotonic)	4.0	0	0	0	0	0	50	None	0
Maintenance											
2.5% dextrose in ½ strength lactated Ringer's solution	Extracellular	264 (isotonic)	4.5–7.5	65.5	55	2	0	1.5	25	Lactate	0
ProcalAmine ^{®c}	Extracellular	735 (hypertonic)	6–7	35	41	24	5	0	30	Acetate, phosphate	0
3% Freamine III ^{®c}	Extracellular	405 (hypertonic)	6–7	35	41	24	5	0	0	Acetate, phosphate	0
COLLOIDS											
Natural											
Whole blood	Extracellular	300 (isotonic)	Variable	140	100	4	0	0	0–4	None	20
Frozen plasma	Extracellular	300 (isotonic)	Variable	140	110	4	0	0	0–4	None	20
Stroma-free hemoglobin	Extracellular	300 (isotonic)	7.8	150	118	4	0	1.4	0	Lactate	—
Synthetic											
6% hetastarch	Extracellular	310 (isotonic)	5.5	154	154	0	0	0	0	None	70
10% pentastarch	Extracellular	326 (isotonic)	5.0	154	154	0	0	0	0	None	25
Dextran 40	Extracellular	311 (isotonic)	3.5–7.0	154	154	0	0	0	0	None	40
Dextran 70	Extracellular	310 (isotonic)	3–7	154	154	0	0	0	0	None	60
Oxypolygelatin	Extracellular	200 (hypotonic)	7.4	155	100	0	0	1	0	None	45–47

From: Rudloff E, Kirby R: Fluid therapy: Crystalloids and colloids. *Vet Clin North Am Small Anim Pract* 28(2):297–328, 1998. Used with permission.

^aBaxter Healthcare, Corp.

^bAbbott Laboratories.

^cMcGaw Inc.

molecules that are permeable to the capillary membrane and large molecules that cannot 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. Syn-

thetic colloids are man-made, large molecules dissolved in normal saline. The saline's sodium content determines the osmolarity of the solution, and the size of the synthetic molecule determines the distribution of the solution. Natural and synthetic colloids and their specific characteristics are listed in Table 1.

Crystalloids

Starling's forces favor the movement of intravascular crystalloids from the capillary into the interstitial space. For example, in the normal vasculature only approximately 20% of lactated Ringer's solution administered intravenously remains in the intravascular space; 80% moves into the interstitium after 1 hour.¹⁴ Crystalloids are therefore used primarily as interstitial volume replacement and maintenance fluids.

The critical role of interstitial fluid in the cell's metabolism and survival makes crystalloid administration an important part of the trauma resuscitation and maintenance fluid therapy plan. Selection of the specific crystalloid to administer is primarily based on the tonicity of the fluid.

Isotonic Crystalloids

In most traumatic situations, resuscitation with crystalloids is best accomplished using an isotonic, balanced electrolyte solution such as lactated Ringer's, Plasmalyte-A[®], or Normosol-R[®]. These solutions provide electrolytes and buffers in concentrations similar to normal plasma and are called *replacement fluids*. Isotonic saline is also a replacement fluid but is not "balanced" because it contains only sodium, chloride, and water. The selection of a specific crystalloid is based on the electrolyte (Na⁺, K⁺, Mg⁺⁺, Ca⁺⁺) concentrations, osmolarity, and pH of the fluid to be administered compared to the needs of the patient. For example, normal saline is often the crystalloid of choice for animals with head trauma because of the slightly higher sodium concentration.

Hypertonic Crystalloids

Crystalloid solutions are made hypertonic by adding sodium or glucose (e.g., 3.0%, 7.0%, and 7.5% saline and 5% glucose added to balanced electrolyte solutions or maintenance solutions). The hypertonicity draws water into the intravascular space after administration. If there is ongoing loss of plasma water, or if there is already interstitial dehydration, these fluids should be avoided.

The use of hypertonic saline in trauma resuscitation leads to rapid intravascular volume expansion after administration of a small volume of this crystalloid. However, this increase is transient because the diffused water dilutes the intravascular hypertonic sodium solution. This diluted solution then redistributes back into the interstitial spaces, often taking additional sodium. Cellular dehydration is a sequela, which may be advantageous in some resuscitative situations (brain in cerebral edema; myocardium in shock) but

generally is not beneficial. Hypertonic saline is often combined with colloids to retain more of the water and sodium within the intravascular space.

Colloids

Whole blood, plasma products, and concentrated albumin contain natural colloids in the form of proteins, primarily albumin. Oxypolygelatin, dextran 40 and 70, and hydroxyethyl starches (hetastarch and pentastarch) are synthetically derived colloids. Stroma-free hemoglobin is a natural hemoglobin product that has been synthetically polymerized.

Colloids are primarily intravascular volume-replacing fluids. In general, the greater the number of small molecules that exist per unit volume, the greater the initial oncotic effect and plasma volume expansion. Colloids with the greatest plasma volume expansion (listed in descending order) are 25% albumin, oxypolygelatin, dextran 40, pentastarch, hetastarch, stroma-free hemoglobin, 5% albumin, whole blood, and plasma.

The degree of molar substitution, the size of the molecule, and the enzymatic processes required for breakdown determine the half-life of the molecule in the body. Rapid renal elimination of polymeric material with a size well below the glomerular threshold (<70,000 daltons) occurs 1 to 3 hours after injection. Larger molecules are degraded into smaller pieces and voided. Extravasated polymeric material, lost from the bloodstream but not voided in the urine, accounts for 20% to 30% of the total dose of macromolecules (>70,000 daltons) administered intravenously. Commercially available synthetic colloids (listed in order of longest half-life) are hetastarch, dextran 70, pentastarch, dextran 40, and oxypolygelatin.

Natural Colloids

Whole blood, plasma, and concentrated albumin are natural colloids, and albumin has the largest number of oncotic molecules. The rest of COP is attributed to fibrinogen (average molecular weight of 320,000 daltons) and globulins (average molecular weight of 140,000 daltons).¹⁵

Concentrated human albumin solutions are available for rapid low-volume resuscitation. In humans, concentrated 5% albumin is used during hypovolemic resuscitation and 25% albumin has been administered to edematous patients requiring volume resuscitation. Unfortunately, concentrated canine and feline albumin solutions are not commercially available.

Natural colloids contain more than just oncotic pro-

teins. Fresh whole blood contains red blood cells, coagulation factors, platelets, albumin, fibrinogen, globulins, white blood cells, and antithrombin. Fresh frozen plasma contains coagulation factors, albumin (3.5% to 5% concentration), fibrinogen, globulins, and antithrombin, whereas frozen plasma contains these substances with virtually no concentration of coagulation factors V and VIII.

Dogs receiving whole blood or a combination packed red blood cell–colloid transfusion should be blood-typed and cross-matched if time permits. If time is a limiting factor, a universal donor (dog erythrocyte antigen [DEA] 1.1 negative) should be chosen for a canine patient. Blood-typing or cross-matching is always recommended for the cat. Blood products should be warmed to patient temperature prior to infusion and administered via an 18 micron micropore filter.

Fresh frozen or frozen plasma transfusion may be appropriate for animals that acutely lose albumin from the vasculature due to an increase in capillary membrane pore size. When the serum albumin level is less than 2.0 g/dl, we administer plasma products in an effort to support the body's requirement of albumin as a buffer and carrier molecule.¹⁶⁻¹⁹

Patients receiving plasma transfusions do not require cross-matching. Plasma should be defrosted in a warm water bath to patient temperature and administered via an 18 micron micropore filter.

Potential concerns with administering natural colloids include the risk of a transfusion reaction, expense, and availability. Time is a factor when treating the acutely hemorrhaging patient. Waiting for blood-typing and cross-matching of whole blood products or for a blood product to warm prior to administration is not always feasible. Transmission of blood-borne illnesses is a possibility with natural colloid infusion, although the risk is minimized with proper screening of blood donors. Serum calcium should be monitored during multiple transfusions. Administration of large volumes of blood products can also cause a dilutional coagulopathy.

Synthetic Colloids

Synthetic colloids were developed to provide timely and convenient fluid resuscitation while avoiding the problems encountered with rapid natural colloid infusions. They provide an increase in COP beyond what is attainable with natural colloids and can be used in conjunction with whole blood or plasma. They are not, however, to be considered a substitute for blood products when albumin, red blood cells, antithrombin, or coagulation proteins are needed.

Oxypolygelatin

Oxypolygelatin is produced from cattle-bone marrow gelatin that is gradually heated under controlled conditions and oxidized with hydrogen peroxide. Excretion of oxypolygelatin is primarily by glomerular filtration and secretion into the feces.²⁰ Oxypolygelatin administration results in an intravascular volume two times the amount administered. Simultaneous administration of crystalloids for interstitial volume replacement is recommended. Because of its rapid and dramatic volume-expanding capabilities, postinfusion vascular volumes should be closely monitored to avoid volume overload or exacerbation of hemorrhage. An osmotic diuresis occurs through the excretion of the small gelatin molecules.²¹ Oxypolygelatin has not been associated with renal failure.²⁰ There are no direct effects on coagulation proteins or platelets. A dilutional coagulopathy has been reported,²¹ similar to that seen with dextran 40, and the clotting time was significantly increased after equal volume oxypolygelatin infusion.²² Because gelatins have been reported to initiate tetany by lowering serum calcium, the oxypolygelatin product for veterinary use is supplemented with calcium.

A clinically relevant risk of allergic reaction, mediated by histamine and complement activation, can occur with oxypolygelatin.^{20,23} The reported incidence of anaphylactic reactions for all species of gelatins is higher than that for either dextran or hetastarch. The manufacturer of oxypolygelatin recommends using it with extreme caution in animals with coagulation disorders, hypoproteinemia, cardiac and pulmonary insufficiency, and renal diseases.

Dextran

Dextrans are polysaccharides composed of linear glucose residues.²⁴ They are produced by the enzyme dextran sucrose during growth of various strains of the bacterium *Leuconostoc* in media containing sucrose. Different molecular weight dextrans can be produced by acid hydrolysis of the parent macromolecule. Survival of dextrans in plasma is determined by the molecular weight. Large polymers are retained in blood until they are metabolized to a size that is able to penetrate the endothelial cell barrier. The smallest dextran molecules are rapidly filtered by the kidneys and induce a mild diuresis. Because of their linear structure, many molecules easily enter through the capillary membranes into the interstitial space and eventually return to the bloodstream via the lymphatics. Larger molecules are briefly stored in hepatocytes, renal tubular cells, and the reticuloendothelial system without producing toxicity.²⁵ Dextran

is broken down completely to CO₂ and H₂O by dextranase (present in spleen, liver, lung, kidney, brain, and muscle) at a rate approaching 70 mg/kg every 24 hours.^{26,27} The plasma half-life is 2.5 hours for dextran 40 and 25 hours for dextran 70.

The reported degree of dextran-induced volume expansion varies based on the type and concentration of solution used and the experimental setting. A 500 ml bolus of dextran 40 produces a 750 ml expansion in intravascular volume at 1 hour and 1050 ml at 2 hours.²⁸ In normal dogs, dextran 70 increased the plasma volume 1.38 times (138%) the volume infused.²⁹

Dextran is not an anticoagulant but has antithrombotic effects due to hemodilution of blood, temporary change in the function of factor VIII:Ag, and decreasing platelet aggregability and thrombus stability, especially at a lower molecular weight.^{30,31} Dextran copolymerizes with the fibrin monomer, destabilizing clot formation.³² Blood glucose levels may be elevated as dextran metabolizes to its glucose residues. This may be a response to rapid degradation of the glucose polymers or a catecholamine response to shock. Bilirubin values can be falsely increased for unknown reasons. Dextran 70 may cause a change in the total solids value that does not reflect actual protein content. Clinical significance of these changes is not known.³³ Dextran 70 may interfere with blood cross-match due to red blood cell cross-linking.³⁴⁻³⁶

Dextran 40 has been associated with acute renal failure,³⁷ anaphylaxis, and bleeding diathesis. Dextran 70 has rarely been associated with acute renal failure.²⁵ Clinical and experimental experiences with dextran 70 in the dog indicate that moderate to life-threatening allergic reactions are rare.

Hemostatic changes in healthy experimental dogs given dextran 70 include an increase in the buccal mucosal bleeding time and partial thromboplastin time and a decrease in von Willebrand's factor antigen and factor VIII coagulant activity without clinical bleeding.³³ Fibrinogen concentration decreases in excess of what can be explained by dilution in dogs.

Hydroxyethyl Starch

Hydroxyethyl starch is the parent name of a polymeric molecule made from a waxy species of either maize or sorghum and is composed primarily of amylopectin (98%). It is a highly branched polysaccharide closely resembling glycogen.²⁴ Two species of hydroxyethyl starch are currently commercially available for fluid resuscitation and maintenance: 6% hetastarch and 10% pentastarch.^{24,34,38-40} The disappearance of hydroxyethyl starch molecules from the body depends on their rate of absorption by tissues (liver,

spleen, kidney, and heart), gradual return to circulation, uptake by the reticuloendothelial system, enzymatic degradation to smaller particles by amylase, and clearance through the urine and bile. Blood alpha-amylase-mediated hydrolysis reduces the molecular weight to less than 72,000 daltons. A rise in serum amylase is to be expected without alteration in pancreatic function. The metabolism of hydroxyethyl starch retained in tissue probably occurs by the action of cytoplasmic lysosomes.⁴¹

There are conflicting reports of hydroxyethyl starch-induced coagulopathies. The molecular weight range appears to play a role, as does the amount administered.^{24,42-44} Dilutional effects on coagulation factors and proteins are produced in response to the volume expansion of the plasma. Bleeding time and volume of blood lost were most significant in dogs receiving more than the recommended dose.

When hetastarch is infused at 25 ml/kg in normal dogs, the initial increase in plasma volume is 1.37 times (137%) the volume infused.²⁹ The intravascular persistence of hetastarch is significantly greater than that of dextran 70, with 38% of hetastarch remaining 24 hours after infusion compared with 19% of dextran.²⁵ Hetastarch favors retention of intravascular fluid and prevents washout of interstitial proteins.⁴⁵⁻⁴⁷ Administration of hetastarch by constant rate infusion provides a constant supply of higher molecular weight particles, thereby maintaining and augmenting plasma COP.

The incidence of anaphylaxis with hetastarch infusion has been reported in humans as 0.0005% to 0.085%.^{48,49} Hetastarch is nontoxic and nonallergenic to dogs at doses up to 100 ml/kg.⁵⁰ In our experience a large number of cats have had a moderate reaction, displaying nausea and occasionally vomiting when hetastarch is infused rapidly. However, when the drug is administered slowly (over 15 to 30 minutes) to the cat, this side effect is eliminated.

There is a notable increase in activated prothrombin time in patients receiving hetastarch, attributable to precipitation of factor VIII by hetastarch.⁵¹⁻⁵⁴ This effect is reportedly less than that associated with dextran. We have administered hetastarch to more than 500 dogs and 150 cats over a 6 year period. Activated clotting times (ACTs) in these animals were prolonged without clinical evidence of hemorrhage. It is our impression that the ACTs may increase to 180 seconds in the dog (normal ACT = 90 to 120 seconds) and 120 seconds in the cat (normal ACT = 75 to 90 seconds) due to the direct and dilutional effects of hetastarch, without concern for clinical hemorrhage. However, when hetastarch has been administered in

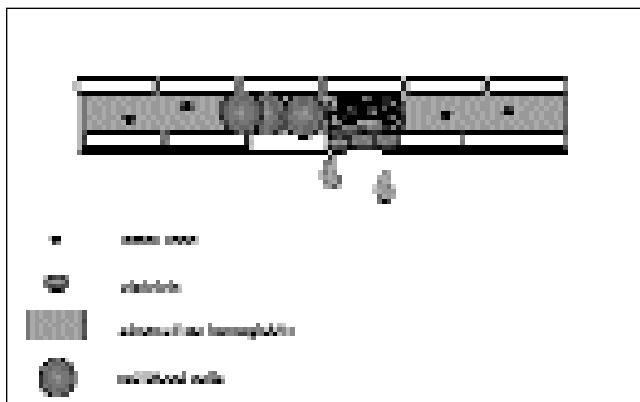


Figure 2. Administration of stroma-free hemoglobin. Hemostasis within an injured capillary can prevent the passage of red blood cells into the vessels distal to the thrombus. The administration of stroma-free hemoglobin solution has the potential to bring oxygen-carrying hemoglobin as a high-molecular weight colloid to the vasculature distal to the clot and provide oxygen to these distal tissues.

quantities that exceed 20 ml/kg/day during resuscitation of dogs with massive trauma, an occasional increase in incisional bleeding has occurred. This may be due to an increase in microcirculatory flow and blood pressure as well as dilutional and direct effects of the hetastarch on coagulation.

Pentastarch is being used in humans as an adjunct for leukopheresis and in Europe and Canada as a hemodiluting and volume-expanding agent.^{55,56} Pentastarch is more rapidly degraded and excreted than hetastarch is due to a lower molar substitution. Infusion of 500 ml of pentastarch results in a 700 ml increase in plasma volume within 30 minutes.⁵⁷ Pentastarch has been investigated for inducing hypervolemia in hemodilution treatment of acute stroke victims,⁵⁸ as well as for volume expansion in human cardiac patients undergoing surgery,^{59,60} septic patients,⁶¹ and burn resuscitation.⁶² Pentastarch's similar effects on coagulation parameters are similar to those of hetastarch; however no significant changes are noted for urokinase-activated clot lysis and bleeding times with pentastarch.⁶³ In addition, pentastarch's effects on factor VIII moieties are a result of hemodilution alone.

Stroma-Free Hemoglobin

It has been proposed that hemoglobin solution might be a temporary substitute for red blood cells.⁶⁴ Hemoglobin has the ability to bind oxygen, with 1 g of hemoglobin chemically binding 1.3 ml of oxygen. Hemoglobin can become fully saturated for oxygen at ambient oxygen pressures, and the oxygen is normally unloaded at the capillary at pressures of approximately 40 mmHg.

The hemoglobin solution is made stroma free to

avoid complications of nephrotoxicity. The molecule is modified by the addition of pyridoxal phosphate, giving the molecule an increased capacity for oxygen saturation. Additionally, the molecule is polymerized so that approximately 50% of the molecules have a molecular weight of 65,000 to 130,000 daltons and less than 10% have a molecular weight greater than 500,000 daltons. The size of the molecules helps to maintain the COP within normal (20 to 25 mmHg). Administration of stroma-free hemoglobin improves the rheology of blood flow compared to whole blood transfusions. It has the potential to carry oxygen to ischemic, traumatized tissues where red blood cell transport is impaired due to microvascular thrombosis (Figure 2).

The red discoloration of the plasma after administration can interfere with biochemical analysis of the blood. It is ideal to draw blood for analysis before infusing the hemoglobin solution. The urine will demonstrate significant bilirubinuria for several days after administration.

Preliminary studies favor using stroma-free hemoglobin in the dog as a temporary substitute for red blood cells. We have used Oxyglobin^{®a} for acute resuscitation of patients with hypotension due to traumatic and intraoperative hemorrhage. The solution provides an excellent colloid, with postinfusion COPs comparable to those achieved with hetastarch. Because of its oxygen-carrying capacity, lower red cell counts can be tolerated. When it is chosen for initial resuscitation from hemorrhage, the animal can be stabilized, which provides time for obtaining, cross-matching, and warming blood products to be administered postresuscitation.

THE FLUID THERAPY PLAN

The fluid therapy plan is based on the characteristics of the specific fluids and the distribution of those fluids within the body. The resuscitation challenge begins with determining the perfusion and hydration status of the patient. Once it is known where the volume deficit lies, the best fluid or combination of fluids to reach the desired end point with the fewest complications must be selected. The route and administration technique are then chosen, with appropriate end points identified to guide the volume of each fluid administered. Determining ongoing fluid maintenance requirements after initial resuscitation can be challenging because of hemorrhage, vascular leakage, vasodilation, excessive vasoconstriction, inadequate cardiac function, alterations in fluid composition, and/or ongoing fluid loss.

^aBiopure.

There is less tolerance for error in the trauma patient than in the usual hospitalized patient. Normal kidney and cardiovascular systems can correct most fluid therapy miscalculations, but these organs may be compromised in the traumatized animal. It is often necessary to modify the resuscitation plan on a minute-to-minute basis to reach the desired end points and to compensate for dynamic changes, ongoing losses, and organ compromise.

Resuscitation

Resuscitation fluid therapy is the administration of fluids to rapidly replace a volume deficit that is causing or has the potential to cause life-threatening organ compromise. When the fluid deficit is in the intravascular space, this is manifested primarily as a *perfusion* problem. When the fluid deficit is in the interstitial or intracellular space, the physical signs reflect a *hydration* problem.

Perfusion

Fluid deficit in the intravascular space causes *poor perfusion* and inadequate tissue oxygenation. This volume deficit results in a lower vessel wall tension and a decrease in the tonic stimulation of the baroreceptors. There is blunting of vagal suppression of the sinus node and increased sympathetic stimulation. Poor perfusion is manifested by tachycardia and vasoconstriction in the dog and, most commonly, by a normal or slower than normal heart rate in the cat. The earliest stages of hypovolemic shock in the dog causes hyperemic mucous membranes and bounding pulses. This is not typically seen in the cat. Later stages in the dog and cat are manifested by poor pulse quality, prolonged capillary refill time, gray mucous membranes, and low rectal temperatures. Sufficient fluids must be rapidly administered to remain in the intravascular space, increase the vessel wall tension, and obliterate the need for the baroreceptor compensatory response. Microvascular flow must be rapidly restored for return of tissue oxygenation and cellular energy production.

Hydration

Fluid deficit in the extravascular space (interstitial and intracellular) causes *dehydration*. This results in tenting of the skin, dry mucous membranes, sunken eyes, and corneal dullness. Severe dehydration can lead to impaired perfusion due to fluid moving from the intravascular space into the interstitium in response to the increased interstitial osmolality.

To replenish the extravascular spaces, crystalloid

fluids with the same tonicity as normal plasma are administered. Over 75% to 80% of isotonic crystalloid administered intravenously will be in the extravascular space within 1 hour in a normal animal.

Fluid Selection

There will be some degree of interstitial volume depletion in any animal suffering from trauma. This makes the administration of crystalloid in some quantity a constant in the fluid therapy plan. Whether to add colloids depends on the presence of hypotension, hypertension, closed cavity hemorrhage, degree of blood loss, potential for systemic inflammatory response syndrome (SIRS), pulmonary contusions, head trauma, and/or myocardial insufficiency (Figure 3 and Table 2).

Resuscitation of perfusion deficits associated with hypovolemia requires rapid intravascular volume expansion by intravenous or intraosseous routes of administration. Crystalloids can be used alone for this purpose; however, perfusion end points may be more difficult to reach without complications of edema. In cases of shock, standard recommendations are to administer crystalloids at volumes equivalent to replacing one blood volume per hour (dog: 90 ml/kg/hr; cat: 40 to 60 ml/kg/hr). This must be titrated to effect.

When large quantities of crystalloids are rapidly administered intravenously, there is an immediate increase in hydrostatic pressure and extravasation of large quantities into the interstitial spaces. Many animals can handle the extra interstitial volume for a short period. In normal tissues, the lymphatics will return the excess fluid to the vascular space to be excreted by the kidneys. However, brain and lung failure occur when the quantity of interstitial fluid surpasses the capacity of the lymphatics. Extreme care must be taken when replacing perfusion deficits with crystalloids alone in animals with pathology in either of these organs or if there is significant impairment of renal function.

A smaller volume of colloids is needed during fluid resuscitation compared to crystalloids alone; also, their use is associated with a reduced tendency toward fluid overload, and resuscitation times are shorter.⁶⁵⁻⁶⁷ Plasma COP can be maintained near normal (20 to 25 mmHg) with synthetic colloids, favoring intravascular fluid retention.

Resuscitation and maintenance fluid therapy for shock due to trauma are often best accomplished with a synthetic colloid combined with a crystalloid (using natural colloids as indicated; Figure 3 and Table 2). When administering crystalloids with colloids, the amount must be reduced to 40% to 60% of what

Table 2
Fluid Therapy Plan for Trauma Patients^a

Status	Fluid Type	Dose/Rate/Technique	End Point	Comments ^b
Compensatory shock No internal hemorrhage Red MM, CRT < 1 sec Rapid heart rate	Isotonic, replacement crystalloid or Isotonic, replacement crystalloid with Synthetic colloid	Dog: 90 ml/kg/hr IV, IO Cat: 60 ml/kg/hr IV, IO Rapid volume resuscitation Dog: 35–55 ml/kg/hr IV, IO Cat: 10–30 ml/kg/hr IV, IO with HES/DEX/SFHb: 10–30 ml/kg IV, IO Dog: rapid volume resuscitation Cat: small volume resuscitation or GEL: 15 ml/kg in 5 ml/kg IV; small volume resuscitation	<ul style="list-style-type: none"> • Pink MM, 1–2 sec CRT • Normal heart rate • MAP ≥ 80 mmHg • CVP > 6 & < 8 cm H₂O • COP ≥ 14 mmHg • ALB > 2.0 g/dl Supranormal resuscitation if no hemorrhage	Follow with rehydration and maintenance fluid administration; watch carefully for any evidence of hemorrhage. A disproportionate PCV and TS on presentation can indicate subtle hemorrhage. Begin CRI of colloids if resuscitation with colloids was performed in the cat or if secondary SIRS is suspected in the dog or cat.
Early decompensatory shock No internal hemorrhage Pale MM, CRT > 2 sec Rapid heart rate Normal to decreased MAP Decreased CVP	Isotonic, replacement crystalloid or Isotonic, replacement crystalloid with Synthetic colloid or Hypertonic saline with synthetic colloid	Dog: 90 ml/kg/hr IV, IO Cat: 60 ml/kg/hr IV, IO Rapid volume replacement Dog: 35–55 ml/kg/hr IV, IO Cat: 24–36 ml/kg/hr IV, IO with HES/DEX/SFHb: 5–30 ml/kg IV, IO Dog: rapid volume resuscitation Cat: small volume resuscitation GEL: 15 ml/kg IV; small volume resuscitation 7% saline: Dog: 4–8 ml/kg IV, IO and HES/DEX/SFHb: 10–30 ml/kg IV, IO; rapid volume resuscitation Cat: 1–4 ml/kg IV, IO and HES/DEX/SFHb: 5–20 ml/kg IV, IO; small volume resuscitation	<ul style="list-style-type: none"> • Pink MM, 1–2 sec CRT • Normal heart rate • MAP ≥ 80 mmHg • CVP > 6 & < 8 cm H₂O • COP ≥ 14 mmHg • ALB > 2.0 g/dl Supranormal resuscitation techniques if no hemorrhage	If it is difficult to maintain resuscitation, place on a CRI of hetastarch (dog: 0.8–1.2 ml/kg/hr; cat: 1–4 ml/kg/hr). Follow with rehydration and maintenance fluid administration. Do not administer hypertonic saline to the dehydrated animal or if head injury or pulmonary injury is suspected.
Late decompensatory shock No internal hemorrhage Pale to gray MM, CRT > 2 sec Normal to slow heart rate Decreased MAP Normal/increased/decreased CVP Organ failure	Isotonic, replacement crystalloid with Synthetic colloid or Hypertonic saline with synthetic colloid	Dog: 35–55 ml/kg/hr IV, IO Cat: 24–36 ml/kg/hr IV, IO with HES/DEX/SFHb: 5–30 ml/kg IV, IO Dogs: rapid volume resuscitation Cats: small volume resuscitation or GEL: 15 ml/kg IV; small volume resuscitation 7% saline: Dog: 4–8 ml/kg IV, IO and HES/DEX/SFHb: 10–30 /kg IV, IO; rapid volume resuscitation Cat: 1–4 ml/kg IV, IO and HES/DEX: 5–20 /kg IV, IO; small volume resuscitation	<ul style="list-style-type: none"> • Pink MM, 1–2 sec CRT • Normal heart rate • MAP ≥ 80 mmHg • CVP > 6 & < 8 cm H₂O • COP ≥ 14 mmHg • ALB > 2.0 g/dl Supranormal resuscitation techniques if no hemorrhage	If it is difficult to maintain resuscitation, place on a CRI of hetastarch (dog: 0.5–1.5 ml/kg/hr; cat: 0.5–2 ml/kg/hr). May require additional cardiovascular support with positive inotropes or blood pressure support. Do not administer hypertonic saline to the dehydrated animal, or if head injury or pulmonary injury is suspected. Follow with rehydration and maintenance fluid administration.

(continued)

Table 2 (continued)
Fluid Therapy Plan for Trauma Patients^a

Status	Fluid Type	Dose/Rate/Technique	End Point	Comments ^b
Catastrophic active hemorrhage Rapidly declining PCV/TS White MM Tachycardia (cats may have bradycardia) Weak or absent peripheral pulses ± abdominal pain or distension	Whole blood or packed red blood cells mixed with isotonic saline, plasma, HES, or DEX	Rapid volume resuscitation, volume and rate determined by ongoing losses	<ul style="list-style-type: none"> • PCV > 20% • MAP = 70–80 mmHg • ALB > 2.0 g/dl • Hb > 8 g/dl Hypotensive resuscitation	Commonly associated with rupture of large vessel, liver, or spleen. No time for crossmatch. Warming of stored blood can be accomplished by running IV line through warm water. Rapid infusion can be by pump or syringe through a neonatal micropore filter. May require autotransfusion of blood from a body cavity for adequate blood supply. Watch for DIC.
	Stroma-free hemoglobin	Dog: 10–30 mg/kg IV, IO bolus; rapid volume resuscitation Cat (not approved): 5–20 ml/kg IV, IO; small volume		Can rapidly provide oxygen-carrying capabilities and maintain COP. May require transfusion once stable. May require hind limb and abdominal counterpressure to stabilize in cases of abdominal bleeding. May require thoracentesis or chest tube in cases of large volume pleural space bleeding. Surgical exploration may be only means for definitive hemostasis and stabilization.
Acute hemorrhage with hemostasis (PCV < 20%) Hypotension	Whole blood or packed red blood cells mixed with isotonic saline, plasma, HES or DEX	Dog: small volume resuscitation Cat: small volume resuscitation	<ul style="list-style-type: none"> • PCV > 25% • MAP = 70–80 mmHg • ALB > 2.0 g/dl • Hb > 8 g/dl Hypotensive resuscitation	May require active hemostasis. Must resuscitate with caution to avoid dislodging clot that is providing hemostasis. May require additional synthetic colloid dose for intravascular resuscitation. Follow with rehydration and maintenance fluid administration.
	Stroma-free hemoglobin	Dog: 10–30 mg/kg IV, IO bolus; rapid volume resuscitation Cat (not approved): 5–20 ml/kg IV, IO; small volume resuscitation		
Chronic hemorrhage or hemolysis (PCV < 15%) Blood loss is slow or has stopped	Packed red blood cells mixed with isotonic saline	Over 4–6 hours IV, IO to reach end point	<ul style="list-style-type: none"> • PCV > 25% • Hb > 8 g/dl 	Follow with rehydration and maintenance fluid administration.
	Stroma-free hemoglobin	Dog: 10–30 mg/kg IV, IO bolus; small volume resuscitation Cat (not approved): 5–20 ml/kg IV, IO; small volume resuscitation		May require blood transfusion once stabilized.

(continued)

Table 2 (continued)
Fluid Therapy Plan for Trauma Patients^a

Status	Fluid Type	Dose/Rate/Technique	End Point	Comments ^b
Hypotension with pulmonary injury, head injury, cardiac insufficiency	Hetastarch or stroma-free hemoglobin with Isotonic, replacement crystalloid	Dogs and cats: 5–20 ml/kg IV or IO; small volume resuscitation Dog: 35–55 ml/kg/hr IV, IO Cat: 24–36 ml/kg/hr IV, IO	• MAP = 70–80 mmHg • COP ≥ 14 mmHg • ALB > 2.0 g/dl Hypotensive resuscitation	Isotonic saline is crystalloid of choice for head trauma patients. Minimize colloid administration if intracranial hemorrhage is suspected (focal cranial nerve deficits, rapid decompensation). Intubation and ventilation may be required to stabilize the animal. Treat underlying heart disease or arrhythmias.
Low plasma albumin (< 2.0 g/dL) Coagulopathy (prolonged PT/aPTT) Low antithrombin (<90%)	Plasma	10–20 ml/kg IV, IO or until end point is reached over 4–6 hours	• Normal coagulation protein activity • ALB > 2.0 g/dl	Follow with rehydration and maintenance fluid administration.
Systemic inflammatory response syndrome postresuscitation	Hetastarch with Isotonic, replacement crystalloid	Dog: 0.5–1.5 ml/kg/hr IV, IO by CRI Cat: 0.5–3 ml/kg/hr IV, IO by CRI Dog: 35–55 ml/kg/hr IV, IO Cat: 24–36 ml/kg/hr IV, IO	• COP ≥ 14 mmHg • ALB > 2.0 g/dl	Must have intravascular volume replaced. Adjust maintenance fluid rates accordingly. Monitor closely for fluid overload. Wean the cat from the CRI as soon as possible.
Interstitial dehydration Decreased skin turgor Sunken eyes Dry MM Loss of eye moisture	Isotonic, replacement crystalloid	Fluid deficit (L) = % dehydration × BW(kg) IV over 1–4 hours if acute dehydration IV over 8–12 hours if chronic dehydration SQ if normovolemic, not to exceed 20 ml/kg over several injection sites	• Rehydration	Must have intravascular volume replaced. Must receive maintenance fluids during rehydration. May require adjustment based on ongoing fluid losses.

^aTrauma-induced clinical problems are presented. Recommended fluid choices, administration techniques, and resuscitation end-points are listed.

^bComments and concerns are added for each clinical problem. MM = mucous membranes; CRT = capillary refill time; HES = hetastarch; DEX = dextran; SFHb = stroma-free hemoglobin; GEL = gelatins; IO = intraosseous; MAP = mean arterial pressure; CVP = central venous pressure; COP = colloid oncotic pressure; ALB = albumin; CRI = constant rate of infusion; TS = total solids; DIC = disseminated intravascular coagulation. (Adapted from: Rudloff E, Kirby R: Fluid therapy: Crystalloids and Colloids. *Vet Clin North Amer Small Anim Clin* 28(2):297–328, 1998. Used with permission.)

would be administered if crystalloids were used alone. This is to prevent a dramatic increase in intravascular hydrostatic pressure and decrease in intravascular COP, which could result in interstitial edema.

In the event of catastrophic hemorrhage, reinfusion of the patient's own blood collected from a body cavity (autotransfusion) can be required to meet the ongoing demands for blood while hemostasis is being accomplished. This provides compatible blood that is at

the animal's body temperature; the blood should be given through a micropore filter to prevent contamination or administration of thrombi.

The administration of stroma-free hemoglobin instead of or in addition to autotransfused blood can reduce the risk of infusing blood that has been contaminated with bowel contents or other particulate matter. Utilizing this modified hemoglobin solution instead of large volumes of autotransfused blood can minimize

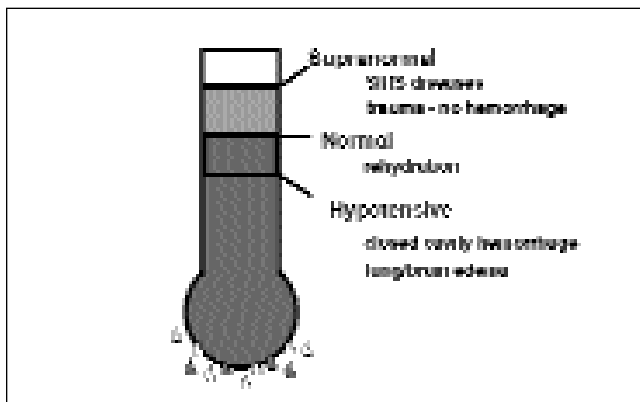


Figure 4. Resuscitation techniques for end-point resuscitation. This beaker demonstrates intravascular blood volume and blood pressure end points obtained with the supranormal and hypotensive resuscitation techniques and indications for each. SIRS = systemic inflammatory response syndrome.

or prevent the complication of disseminated intravascular coagulation that frequently accompanies large volume autotransfusions while providing a large molecular weight colloid.

A colloid that is larger than the pore size of the capillaries should be selected and administered (Figure 1C). When there is increased capillary permeability and loss of albumin through the capillary membrane, hetastarch or pentastarch is the colloid of choice. Smaller-sized colloids (e.g., dextran 40, dextran 70, oxypolygelatin, albumin) will leak through the pores and be deposited into the interstitial space (accompanied by water). Continuing to give crystalloids alone in this situation will raise the intravascular hydrostatic pressure, further dilute the COP, and accelerate fluid flow across the membrane into the interstitial fluid compartment.

Techniques for resuscitation with colloids include rapid volume intravascular resuscitation for dogs and small volume resuscitation for the dog and cat. Selection of either technique is based on the species of the animal, the presence of brain or lung pathology, and the probability of ongoing or closed cavity hemorrhage.

Rapid Volume Intravascular Resuscitation for Dogs

Unless there is closed cavity hemorrhage, pulmonary contusions, cardiac dysfunction, or head trauma, dogs experiencing traumatic shock due to hypovolemia or maldistribution of blood flow benefit from rapid volume intravascular resuscitation techniques (Table 2). Whole blood products can be administered by this technique in catastrophic hemorrhage situations; input should at least match ongoing loss. This technique is not recommended in the cat.

Crystalloid fluids are then titrated to replace interstitial fluid deficits. Additional colloids can be admin-

istered using small volume intravascular resuscitation techniques if perfusion has not improved to the desired end point after the initial bolus.

Small Volume Intravascular Resuscitation

Hypovolemic dogs with closed cavity hemorrhage, head trauma or pulmonary contusions, cardiogenic shock, and oliguric renal failure and all hypotensive hypovolemic cats can benefit from careful resuscitation using the small volume intravascular resuscitation technique for synthetic colloids. The synthetic colloid is administered in increments of 5 ml/kg over 3 to 5 minutes. The perfusion parameters are reassessed, and the 5 ml/kg bolus is repeated as needed until the end point of resuscitation is reached. The goal is to administer the smallest volume of colloid possible to successfully resuscitate the intravascular compartment. This minimizes extravasation of fluids into the brain or lungs, titrates the preload to the heart, and reduces the probability of disturbing clot formation.⁶⁸⁻⁷⁰ Animals requiring resuscitation with synthetic colloids by the technique described will be prepared for maintenance colloid infusion immediately after resuscitation.

When hypovolemia and cardiac dysfunction play a significant role in hypotension and poor perfusion from trauma, careful small volume resuscitation with a colloid can replace intravascular volume while minimizing crystalloid extravasation into the lungs.⁷¹ However, the mainstay of therapy is directed toward controlling arrhythmias, augmenting contractility as necessary, and manipulating preload and afterload.⁷²

End-Point Resuscitation

Formulas have been proposed for calculating the quantities of fluids to be delivered for successful resuscitation. However, these formulas have not proven to be consistently accurate due to individual variations such as vascular permeability, differences in cardiac, pulmonary, and renal function, and ongoing losses. Successful resuscitation therapy depends on administering quantities of fluids sufficient to reach specified end points (Table 2). This process is termed *end-point resuscitation*.

Prior to resuscitation the clinician must determine what end-point values for monitored parameters will indicate successful resuscitation. Physical, hemodynamic, and blood chemistry parameters are the mainstay of monitoring end points. Further investigations may lead to monitoring gastric mucosal pH and serum lactate for end-point determination. The need to avoid volume overload and increased capillary hydrostatic pressure in traumatized animals with closed cavity

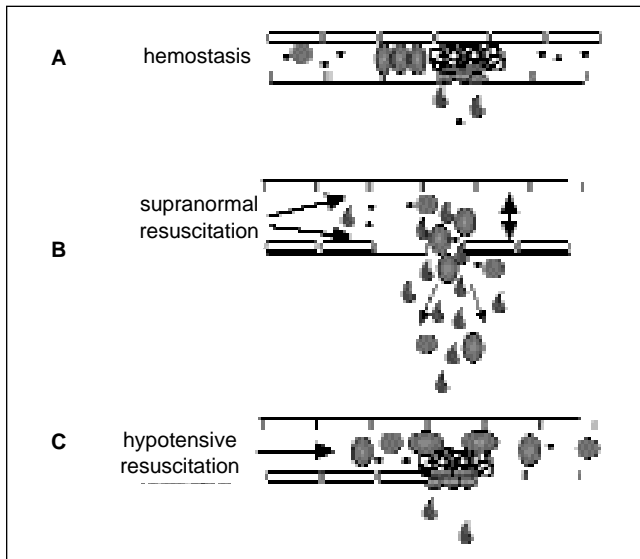


Figure 5. Different resuscitation techniques in cases of hemorrhage. (A) A ruptured capillary is repaired by the hemostatic response (platelets and a fibrin clot), which can prevent red blood cells from carrying oxygen to the vasculature distal to the thrombus. (B) Supranormal resuscitation can rapidly increase the hydrostatic pressure sufficiently to dislodge the clot and allow active hemorrhage. (C) Hypotensive resuscitation should bring the end-point values to within a low-normal range, allowing a gradual opening of the capillary while preserving the clot.

hemorrhage, ongoing hemorrhage, and brain or lung trauma will dictate the end points selected and end-point resuscitation technique employed (Figure 4).

It may not be possible to reach the desired end points of fluid resuscitation until life-threatening complications of trauma have been treated. The presence of a pneumothorax might require a rapid therapeutic thoracentesis or chest tube. Removing the positive pleural pressure allows venous return to become adequate to bring cardiac output back into an acceptable range with fluid resuscitation. Animals with severe pulmonary contusions might require anesthesia or neuromuscular blockers, intubation, and ventilation to provide adequate oxygenation of the blood during fluid resuscitation. Ongoing abdominal hemorrhage might warrant hind limb and abdominal counterpressure to provide tamponade while administering resuscitation blood, colloids, and crystalloids. Antiarrhythmic drugs may be necessary to treat tachyarrhythmias or bradyarrhythmias that are affecting cardiac output. Severe head injuries can require that the animal be ventilated with its head elevated during conservative small volume fluid resuscitation.

Supranormal End-Point Resuscitation

Below a critical level of oxygen delivery to the tissue, tissue extraction cannot increase in proportion to

the reduced delivery and oxygen consumption begins to fall. Cellular hypoxia and decreased energy production result. Clinical studies in critically ill humans have found a higher mortality rate in those who are unable to increase their oxygen extraction rate in response to decreases in oxygen transport. The highest surviving group had their oxygen consumption dependent on oxygen transport.⁷³ For hypovolemic and SIRS shock, resuscitation of perfusion to supranormal values to increase oxygen delivery is recommended (Table 2).⁷⁴ 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 (Figure 5).

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 with blood pressures remaining in the low-normal range rather than reaching supranormal values. This avoids a significant increase in hydrostatic pressure that may dislodge clots that are providing life-saving hemostasis or may cause worsening of brain or lung edema (Figure 5).

Maintenance

Often, the fluid combination chosen for initial stabilization is not the same as what is required for maintenance. Once the interstitial fluid deficit has been replaced and replacement of plasma water is no longer required, the interstitial fluid volume must be maintained. Water is used in cellular metabolism and is lost by way of the kidneys, respiration, and evaporation. Potassium is secreted in large quantities by the kidney. The cells' need for free water directs that the sodium concentration in *maintenance* fluids be approximately 50% of the plasma concentration. The need to replace lost potassium requires potassium concentration in maintenance fluids to be up to three times that in replacement fluids. These solutions are made isotonic by adding a 2.5% concentration of glucose. However, once these isotonic maintenance fluids are distributed into the interstitium, the glucose is quickly metabolized and there is additional free water to use in metabolic processes. The use of replacement fluids for maintenance therapy requires active excre-

tion of sodium by the kidney to provide the needed free water, and replacement fluids must be supplemented with potassium.

Normal maintenance fluid rates account for insensible, obligatory (urinary and fecal), and metabolic losses of water and electrolytes. Standard fluid maintenance requirements are estimated to be 40 to 60 ml/kg/day. Ongoing losses through diuresis, vomiting, diarrhea, or extravasation of fluid into third body spaces such as the peritoneum, pleural cavity, or uterus are estimated and added to the total daily fluid volume, and oral intake volumes are subtracted. The maintenance fluid therapy plan needs to be reassessed throughout the day, and adjustments should be made according to ongoing losses, metabolic requirements, oral intake of water and food, and renal and cardiac function.

It has been suggested that current methods for assessing fluid needs may overestimate the patient's actual requirements since sick patients are inappetent and inactive.¹ Water requirements have been based on formulas for maintenance energy requirements ($140 \times [\text{kg body weight}]^{0.73}$), but it is felt that this might overestimate the needs.⁷⁵ The use of calorimetry for measuring actual energy requirements has been proposed as a means for determining actual needs. Frequently, perfusion parameters must also be supported and medullary washout from aggressive resuscitation fluid administration can make the ongoing fluid requirement higher than what is required for energy needs alone. Most patients respond well to the standard fluid replacement regimen because excessive fluid and electrolytes are readily excreted by the kidney.

Crystalloids will be the mainstay of maintenance fluid therapy, with the selection made to suit the specific needs of the animal. The maintenance fluids can also provide electrolytes, COP, proteins, coagulation factors, glucose, and nutrients, depending on the fluids selected and how they are supplemented. Increased capillary permeability and hypoproteinemic states can require ongoing maintenance of COP. This can be accomplished by a constant rate infusion of a high-molecular weight colloid, such as hetastarch, combined with a maintenance crystalloid (Table 2). The crystalloid volume infused should be reduced by 40% to 60% from standard calculations to avoid intravascular volume overload.

Albumin, coagulation factors, and antithrombin III can be maintained by infusion of fresh frozen or frozen plasma. Red blood cells can be replaced and maintained by infusing packed red cells diluted in saline or whole blood. Nutrients can be provided in the form of amino acids, lipids, and glucose according

to the fluid chosen for maintenance. The composition and volumes of these nutritional supplements are based on metabolic requirements.

Alterations in sodium, potassium, calcium, magnesium, and phosphorus can cause life-threatening complications when unrecognized or uncorrected. Significant electrolyte imbalances can necessitate selection of a specific crystalloid (Table 1) or supplementation of the crystalloid to obtain and maintain electrolyte homeostasis.

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