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Controversies in Fluid Resuscitation

Remember when you first had the opportunity to write orders as a medical student? How careful and even deliberate you were? When it came to ordering intravenous fluids, how much thought you would give to the choice of fluid and the rate? Should it be half normal or quarter normal?

I have a story from my intern year. I had carefully written out fluid orders — ones precise enough for the space program. It must have caught the attention of one of the older physicians at our county hospital. This physician had initially started as a medical student in the 1930s. After looking at my detailed fluid orders, he said that was largely a waste of time. He further said that dumb kidneys beat a smart doctor any day. The point being that the body has mechanisms to regulate its fluid volume, and that the primary function of fluid resuscitation was to restore normal kidney function, and then let them do the rest.

Well, like a lot of what he told us, there was both some simplification and some truth in it. So it was with much of what I learned during that year; it is the simple principles that remain constant and not the detailed knowledge — which changes in its nuances — that have proved useful these past 30 years.

—J. Stephan Stapczynski, MD, FACEP, Editor

Case Examples

It's a Saturday afternoon, and your first patient in an already crowded department is a 45-year-old male who is brought into the hospital by ambulance complaining of nausea, vomiting, and abdominal pain. The patient has a significant history of coronary artery disease and aortic stenosis. The ECG reveals a sinus tachycardia without any ST abnormality. The nurse calls you to the bedside because the patient has a blood pressure (BP) of 80/30 mmHg with a pulse of 120 beats per minute (bpm). His skin is cold and clammy. The nurse asks you what fluids you want.

At the same time, a 70-year-old man who has been coughing with a fever for the past 2 days is upgraded to the critical area of the ED, with a temperature of 104.1°F, a BP of 92/40, a heart rate of 102, and a lactate of 6. The nurse again asks, "What fluid and how fast?"

Introduction

Fluid resuscitation has always been an integral part of the resuscitation of unstable or critically ill patients. Fluid use has been the subject of controversy because there is little class I data to guide clinicians.

Trials in the past 10 years have clarified some of the controversies surrounding fluid management. For instance, early aggressive fluid resuscitation guided to certain end goals decreases morbidity and mortality and has been supported by recent trials. In contrast, both overaggressive and exceedingly conservative fluid resuscitation have resulted in adverse outcomes. This article will review physiology, goals of fluid therapy, and the differences between crystalloid and artificial colloid. Blood products will not be discussed, as they are beyond the scope of this article. We hope to clarify situations in which crystalloid and colloid are most beneficial.

Executive Summary

- Crystalloids and colloids are equivalent for fluid resuscitation in the vast majority of seriously ill or injured patients.
- Colloids may adversely impact patients with sepsis, traumatic brain injury, and on cardiopulmonary bypass.
- Colloids have a role in treating hypotension induced by hemodialysis or paracentesis.
- Hypertonic saline may have a limited role in the resuscitation of patients with traumatic brain injury.

Pathophysiology of Shock

Shock is defined as a condition of inadequate delivery of oxygen and substrates for normal cellular function. Whether the cause of shock is hypovolemia, pump failure, or vasodilatory, the oxygen (O_2) delivery to tissue is impaired, causing global tissue ischemia. Hypotension is often defined as a systolic pressure value of 90 mmHg or less. Hypotension often is an ominous sign of shock, but by itself does not equate to shock. While shock and hypotension often occur together, shock also involves signs and symptoms of end organ hypoperfusion. The conventional signs include but are not limited to altered level of consciousness, decreased urine output, tachycardia, mottled skin, delayed capillary refill (greater than 2 seconds), and metabolic acidosis.

Shock can be present with a normal blood pressure and can be referred to as "cryptic shock" or "normotensive shock." Cryptic shock, frequently associated with early severe sepsis and septic shock, is characterized by global tissue hypoxia and can be quantified by increased blood lactate levels and decreased central venous oxygen saturation ($ScvO_2$) despite the presence of normal hemodynamic criteria.[1] Cryptic shock often leads to increased morbidity and mortality due to global ischemia and a delay in diagnosis.

To understand shock, one must understand the concepts of oxygen delivery. Oxygen delivery (DO_2) is the total amount of oxygen saturated to hemoglobin and delivered to the peripheral tissues per minute. It is calculated according to the equation shown in Figure 1.

Cardiac output is the most important determinant of oxygen delivery. As illustrated by Figure 1, cardiac output has the ability to compensate for increases in metabolic needs or decreases in O_2 carrying capacity. However, cardiac output is a product of both heart rate and stroke volume. Stroke volume is affected by a number of factors. Thus, cardiac output is difficult to predict and manipulate. Hemoglobin is the next biggest contributor to oxygen delivery. As hemoglobin is easily manipulated through transfusion, it becomes the most important factor in regulating oxygen delivery. To more easily visualize this, imagine there are two towns separated by a train. Town A is rich and has all the supplies, and town B is starving. There is only one train, and the following choices exist to effectively feed the people of the towns: 1) speed up the train by increasing cardiac output, which is best done by increasing preload with fluids or adding an inotropic medication; 2) increase the number of boxcars so more food can be transported (transfusing to a higher hemoglobin); and lastly and less efficiently, increase the amount of fuel (increase the Fio_2).

The heart is essentially a dual-chamber hydraulic pump made of muscle and valves. When a weight or force is applied to one end of a resting muscle, the muscle stretches to a new length. The force or weight applied to the resting muscle prior to the onset of contraction is called preload. The increase in the length of the muscle leads to a more forceful contraction by the muscle itself.

The amount of stretch or preload applied to the cardiac muscle at rest is determined by the volume in the ventricles at the end of diastole. This is termed ventricular end-diastolic volume (EDV). A greater EDV correlates directly to a more forceful contraction. The stroke volume (SV) of the normal heart is primarily a function of the preload. Although the compliance of the muscle and afterload also may affect the SV, their significance dwindles as the patient becomes hypovolemic. This emphasizes the importance of fluid resuscitation in the hypotensive or hypovolemic patient. The most effective means of preserving the cardiac output is to maintain adequate EDV by correcting volume deficits and minimizing hypovolemia. Thus, fluid resuscitation increases EDV or preload and cardiac output, and therefore restores tissue perfusion and oxygenation, and often blood pressure.

With hypovolemia, the body compensates to maintain perfusion and O_2 delivery using both the micro- and macrocirculatory system. This is in response to insufficient stretch sensed by carotid and aortic baroreceptors. Mechanisms include adrenal release of catecholamines which induce tachycardia, increased cardiac contractility leading to increased SV, and peripheral vasoconstriction. These mechanisms compensate for decreased CO in accordance with the equation depicted in Figure 1. Venoconstriction supports intrathoracic volume and produces an increase in preload, whereas arterial constriction will increase perfusion pressure, which in turn directly increases organ blood flow. Perfusion may be shunted toward the organs essential to life such as the heart

Figure 1: Calculation

1) Oxygen Delivery:

$DO_2 = CO \times CaO_2$, where $CaO_2 = (Hbg \times 1.39 \times SaO_2)$*

* CO: cardiac output; Hbg: hemoglobin; CaO_2: oxygen content; SaO_2: O_2 saturation

2) Cardiac Output:

CO = HR x SV

* CO: cardiac output; HR: heart rate; SV: stroke volume

and the brain while non-vital organs such as the skeletal muscle receive less than optimal blood flow. This maintains mean arterial pressure (MAP) under the conditions of hypovolemia and, ultimately, organ hypoperfusion.

Signs and Symptoms of Shock

Ideally, signs and symptoms of shock would be directly related to the degree of hypovolemia and have a progressive symptomatic pattern. However, this is unreliable in the clinical setting. Signs and symptoms of hypovolemia, in fact, are markedly variable and are a function of the disease state, progression of the condition, and the capacity of the individual to compensate for the specific disease.

As an example, sepsis produces a much more complex set of signs and symptoms than does hemorrhage due to pathologic vasodilation and exaggerated organ dysfunction in addition to the loss of preload. In addition, children and otherwise healthy individuals maintain the compensatory ability to endure a substantial loss in volume without developing clinical symptoms. Patients with pre-existing co-morbid disease such as coronary artery disease or heart failure may not be able to withstand even a negligible loss of fluid.

Typical, albeit unreliable signs of hypovolemia include delayed capillary refill, dry mucous membranes, abnormal skin turgor, and sunken eyes. Although neither specific nor sensitive, reduced cardiac output has been known to produce fatigue, dyspnea, and near syncope. Organ dysfunction may be an early recognizable sign of hypovolemia. These include oliguria, electrolyte and/or acid-base disruptions such as lactic acidosis.

Blood Pressure

Hypotension is a phenomenon considered to be absolutely pathologic. A MAP of less than 65 mmHg, systolic BP of less than 90 mmHg, and/or systolic BP decrease by more than 40 mmHg from the patient's baseline must always be considered pathological unless proven otherwise. However, sufficient O_2 delivery is not guaranteed by the existence of a normal BP. Oftentimes, critically ill patients will maintain normal or near-normal BP due to compensation. In the absence of prompt diagnosis and resuscitation, these patients may well deteriorate into hypotension. Discrete momentary periods of hypotension are significant indicators of hypoperfusion and may be precursors to a hemodynamic crisis and are often the initial symptom of uncompensated shock.

Neither automated blood pressure cuffs nor direct auscultation methods are adequate gauges of accurate BP. Respectively, these techniques have been known to significantly overestimate and underestimate BP measurements. The risk of substantial errors in BP measurements of hemodynamically unstable patients supports the use of invasive arterial pressure monitoring.

Heart Rate

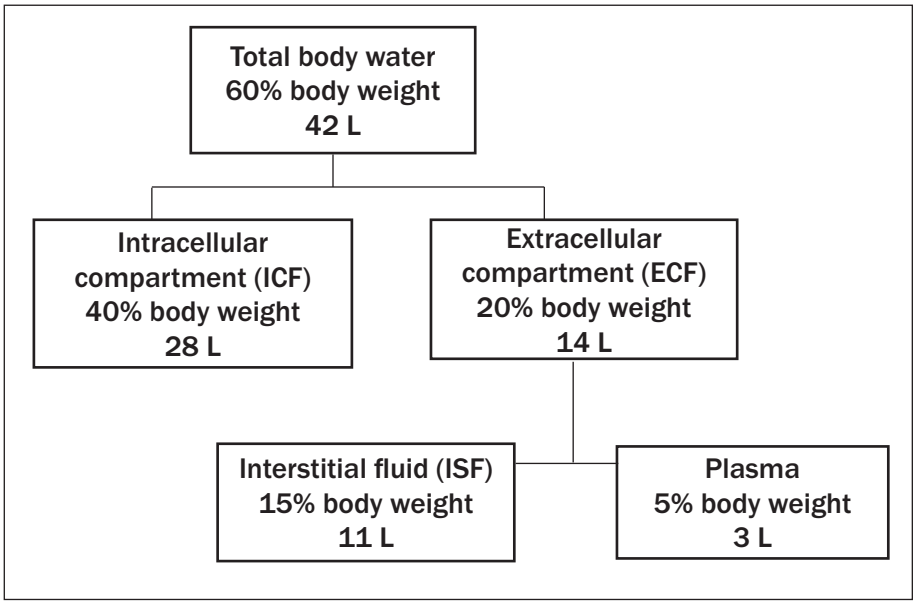
Sinus tachycardia is an inherently non-specific indicator of autonomic response to hypovolemia, hemorrhage, and other causes of disease states. Heart rate will increase as hypovolemia worsens. (*See Figure 1.*) Increases in heart rate help to maintain an adequate cardiac output in the presence of a depleted stroke volume. Unfortunately, the reaction of heart rate to acute volume loss is quite erratic. In average patients, even a 20% loss of volume will fail to elicit tachycardia. A compensatory tachycardia may be additionally muted in the presence of co-morbidities and/or medication, such as beta-blockers. Paradoxical and relative bradycardia also may be reported in up to 30% of patients experiencing both traumatic and non-traumatic hemoperitoneum.

The Physiology of Fluid Therapy

Total body water (TBW) for the average adult male (70 kg) comprises approximately 42 liters. This is dependent upon the fat content of the individual. There is a range of three fluid compartments in which TBW is dispersed. There is an extracellular compartment (ECF), which itself is divided into plasma fluid and interstitial fluid, and there is a fluid-filled intracellular compartment (ICF). Between the ECF and ICF exists the semi-permeable cell membrane that functions to regulate the passage of materials into and out of the cell. This membrane is highly permeable to water while remaining impermeable to electrolytes. A sodium (Na+)-potassium (K+) pump enveloped in the cell membrane and associated with both the ICF and ECF serves to transport Na+ out of the cell in exchange for the transfer of K+ into the cell. (*See Figure 2.)* Na+ is the predominating ion of the ECF and, as such, the Na+ content is the major factor determining the volume of the ECF.

The Starling Equation defines net

Figure 2: Distribution of Body Water



fluid movement between compartments as the difference between capillary hydrostatic pressure and interstitial hydrostatic pressure subtracted by the difference between capillary oncotic pressure and interstitial oncotic pressure ($J_v = K_f\{[P_c - P_i] - \sigma[\pi_c - \pi_i]\}$, where J_v is fluid movement, K_f is filtration coefficient, σ is reflection coefficient, P_c and P_i are capillary and interstitial hydrostatic pressure, respectively, and π_c and π_i are capillary and interstitial oncotic pressure, respectively). Net fluid motion is determined by the hydrostatic pressure differences of interstitial and capillary fluids (pressure exerted by fluid due to gravity) and the oncotic pressure differences between interstitial and capillary contents (osmotic pressure derived from the presence of compartmental proteins drawing in water in the interest of equilibrium).

The total osmolarity corresponds to the number of osmotically active particles. In physiologic conditions, it is approximately 280 mOsm/L spanning across the three fluid compartments. The number of osmotically active particles in a given solution is directly responsible for the osmotic pressure of said solution. Consequently, a solution with an osmolarity of 280 mOsm/L is defined as isotonic, less than 280 mOsm/L is termed hypotonic, and greater than 280 mOsm/L is distinguished as hypertonic.

As the plasma and the interstitial proteins do not penetrate the capillary walls, they are responsible for the osmotic pressure. If introduced to a hypertonic solution, in vitro cells will shrink as water moves out through the cellular membrane in an attempt to equilibrate the outside environment with the interior of the cell. Conversely, when placed within a hypotonic solution, in vitro cells will swell up and subsequently burst as water pours into the cell through the cellular membrane in an effort to equilibrate the interior of the cell with the exterior of the cell.

Intravenous (IV) solutions will have varying effects on fluid volume of compartments based on osmolarity of the solution and the extent of capillary leakage and pre-existing water deficits in specific fluid compartments. As an example, one liter of normal saline (NS) will increase the plasma volume by only 200 cc after equilibration.

All fluids must pass through the plasma compartment, which is also known as the first space. The interstitial fluid compartment is defined as the second space. The **third space** is a pathologic expansion of the interstitial compartment at the cost of plasma volume. This is often due to inflammation and results in edema. Unfortunately, this accumulation of fluid cannot be removed by diuresis, dialysis, or by any other means. Once the primary condition and the inflammation resolve, fluids shift from the pathological space and reintegrate into the plasma volume.

In addition to administration of IV fluids, humoral factors also affect the fluid status of the body, such as anti-diuretic hormone (ADH), also known as vasopressin or argipressin. ADH is a peptide hormone whose end goal is to restore the body's osmolality to 280 mOsm/L. It increases the water permeability of collecting ducts in the kidney, thereby allowing for a greater amount of water reabsorption. ADH secretion occurs in response to excessive plasma osmolality and/or a decreased plasma volume.

Another such humoral feature that regulates fluid shifts is the Renin-Angiotensin-Aldosterone System. This system is primarily activated in order to raise blood pressure, which it does by constricting blood vessels and increasing the intravascular fluid content. When blood volume is low, the kidneys will release renin, which cleaves angiotensinogen (a prohormone synthesized by the liver) into angiotensin I. Angiotensin I is then converted into angiotensin II in the lungs by angiotensin converting enzyme. Angiotensin II then binds to mesangial cell receptors inside the glomerulus to induce renal vasoconstriction and the release of aldosterone from the adrenal glands. Aldosterone will bind to receptors in the renal tubules, stimulating a greater amount of Na+ reabsorption from the urine. With the increased Na+ reabsorption, water follows, resulting in a greater amount of water reabsorption.[2]

End Points of Therapy

The goal in the treatment of shock is to restore oxygen delivery at a level that is sufficient to meet metabolic need and repay any oxygen debt that may have accumulated. This is accomplished by supporting all of the components of oxygen delivery: cardiac output, hemoglobin,

and oxygen saturation. Depending on the disease state, this often also includes control of the primary metabolic insult. For instance, in trauma patients who are bleeding, control of hemorrhage, including surgical control if necessary, is paramount, as no therapy will be successful if hemorrhage is ongoing. In sepsis, this may include use of antibiotics and/or drainage to achieve source control. However, even after control of the primary problem, many patients will have evidence of inadequate tissue perfusion or ongoing cellular dysfunction. This may be true in the presence of seemingly normal vital signs and adequate target organ function such as urine output.

There is no clinically available measurement to ensure that cellular metabolism has been normalized. Normally followed vital signs often underestimate the depth of shock and are normal even in the face of inadequate oxygen delivery. Compensatory vasoconstriction will result in a normal blood pressure even if cardiac output is still markedly depressed. Insults to the renal tubules will allow kidneys to produce adequate volumes of dilute urine even in the face of partially compensated shock. Finally, global measurements such as serum lactate or venous oxygen saturations represent the mixing of blood from a number of different vascular beds.

Thus, vascular systems such as the splanchnic circulation may be inadequately perfused, yet adequate circulation in other vascular beds may mask this. Tools exist to measure specific vascular beds such as measuring sublingual tissue CO_2 levels or gastric intramucosal pH. More recently, transcutaneous measures of blood flow have been developed. However, none of these have achieved sufficient accuracy and reliability to be used in clinical medicine.[3] Thus, we must rely on surrogate measurements to guide therapy. These measurements include estimates of preload, tissue oxygen extraction, and whole body metabolic parameters.

Central Venous Pressure

Central venous pressure (CVP) is measured by inserting a catheter in the central circulation, placing the tip of a catheter into the superior vena cava, near the right atrium. CVP calculates the pressure in the right atrium and is a surrogate measurement for right ventricular end-diastolic pressure, which is a marker of volume status or preload. CVP is affected by the amount of blood in the central venous compartment and also by the compliance of the right side of the heart, and the intrathoracic pressure. If the CVP measurement is to be used, the end-diastolic pressure must be measured at end expiration. Normal CVP ranges from 2 to 7 mmHg. Target values used in resuscitation are 8-12 mmHg. If the CVP is low, the patient is assessed to be volume depleted and requires volume. Normal or elevated CVPs imply adequate preload. However, this can be problematic as the relationship between CVP and volume status may be quite dynamic.[4]

The veins contain approximately 70% of total blood volume and are 30 times more compliant than arteries. Therefore, changes in blood volume within the veins are associated with relatively small changes in venous pressure. Also, disease states like obstructive lung disease, positive pressure ventilation, and right heart strain will lead to falsely elevated CVP despite significant intravascular hypovolemia.

Instead of using an absolute CVP value as a surrogate for intravascular volume, changes in the CVP in response to fluid boluses can be used instead. The principle is that the vascular system is "full" when it reaches the limits of easy distensibility. If a fluid bolus does not significantly increase the CVP, then the vascular system has more space for more fluid and is "not full." When the CVP rises significantly to a bolus of fluid, then there is less space for more fluid and the vascular system is "full." Empirical guidelines used for this method are an increase of greater than 5 mmHg after a fluid bolus of 250 to 500 mL suggests the vascular system is full and intravascular volume is at its maximum.

Mixed Venous Oxygen Saturation and Central Venous Oxygen Saturation

Tissue oxygen extraction is one of the main compensatory mechanisms used to maintain oxygen consumption in the face of inadequate oxygen delivery. The most complete mixing of venous return occurs in the pulmonary artery. Therefore, mixed venous oxygen saturation ($SMVO_2$) traditionally was the measurement used to calculate oxygen consumption. A normal value is generally considered to be approximately 70%, translating into a 30% oxygen extraction ratio, assuming normal oxygenation on the arterial side. Sampling mixed venous oxygen involves slowly withdrawing 5 mL of blood from the pulmonary artery and sending it for blood gas analysis. If excess tension is placed on the syringe during the sampling process, blood can be pulled back from the pulmonary vein, artificially raising the oxygen saturation.

As the use of pulmonary artery catheters has decreased, there has been interest as to whether central venous oxygen saturation ($SCVO_2$) would be an adequate substitution for $SMVO_2$. $SCVO_2$ ideally is measured in the right atrium but can also be measured in the superior or inferior vena cava. This has the advantage of not requiring a pulmonary artery catheter to obtain a sample. Data exist that suggest that $SCVO_2$ is a useful clinical measurement, at least following acute hemorrhage.[5] In addition, in a landmark study done by Rivers et al,[6] ED patients with severe septic shock were randomized to standard care or to early goal-directed therapy (EGDT). In the EGDT group, $SCVO_2$ was an important parameter and was targeted to 70% utilizing fluids and inotropic support as well as maintaining a hematocrit of greater than 30%. There was a highly statistical survival advantage to receiving EGDT

that persisted out to three months. This study strongly argues for utilizing oxygen transport as a goal in critical illness with measurement of venous extraction as an important component.

Oxygen Delivery

As the majority of metabolic states that comprise critical illness require increased oxygen needs, it is reasonable to think that oxygen delivery would be an important parameter to follow in such patients. Critical determination of oxygen delivery includes cardiac output and hemoglobin, as well as oxygen saturation. A landmark study by Tuschmidt and Rackow demonstrated that survival was better in patients with higher oxygen deliveries in a mixed ICU population of septic shock.[7] Calculating oxygen delivery requires measuring cardiac output, which in the past required the use of a pulmonary artery catheter. However, newer technology is available that calculates cardiac output from a peripheral arterial line, obviating the need for a pulmonary artery catheter. This may be important as there has been a recent increase in concern that use of PA catheters may be associated with increased complications and higher mortality.[8,9]

Base Deficit

Base deficit is defined as a decrease in the total concentration of base compared to acid. During states of inadequate oxygen delivery, anaerobic metabolism is used to generate high-energy phosphates as substrate. While this is an inefficient system, it is the only option in the face of inadequate oxygen supply. One byproduct is the development of acidosis. Base deficit (BD) is a good measure of global tissue acidosis and has been demonstrated to be a good predictor of fluid requirements, transfusion requirements, and outcome following trauma. While the most common cause of acidosis early in critical illness is lactic acidosis, other types of acidosis can contribute, such as those from toxic ingestion, renal failure, or seizure activity.[10-11] All of those also impact BD. Normal base deficit is between +2 and -2 mmol/L. The more negative the number, the greater the amount of acidosis. Abnormalities can be stratified into mild (BD -2 to -5), moderate (BD -6 to -14), and severe (BD greater than -14).

Serum Lactate

Lactate is a byproduct of anaerobic glycolysis and is formed by pyruvate via the action of the enzyme lactate dehydrogenase. As with base deficit, lactate is a measure of global acidosis but is specific to tissue hypoperfusion. Blood lactate is a balance between production and clearance of lactate, so states that impede lactate clearance will increase serum lactate in the face of relatively normal production. As lactate is a global measurement, microvascular hypoperfusion can exist in a single vascular bed and not be reflected in total body lactate until later. Therefore, early on, lactate levels should be checked frequently. Lactate levels are generally less than 2 mmol/L. Any significant elevation should be considered abnormal. A single lactate may not be sufficient to guide therapy. However, the ability to normalize lactate has been shown to be the most accurate predictor of outcome following acute injury.[12] Despite this, there are some limitations to use of lactate levels as the sole gauge of end organ perfusion, and serum lactate should be interpreted in a global context.[13]

Fluid Resuscitation

Intravenous access is a vital part of fluid resuscitation. The determinants of flow through a rigid catheter follows Poiseuille's law, which states that laminar flow rates are indirectly proportional to the catheter length and directly proportional to the fourth power of the radius of the catheter. Therefore, a catheter radius doubled in size will increase the flow 16 fold. Doubling the length of the catheter will decrease flow by half. Thus, the line of choice for fluid resuscitation is a short large-bore catheter, either a 16 g or 14 g peripheral IV. If central venous access is required, a 9 French introducer is preferred. Triple-lumen catheters should be avoided for rapid volume resuscitation due to their increased length and smaller radius. IV tubing size is also a factor in fluid delivery, and the use of pressure bags increases delivery.

Hypovolemia may be due to external fluid loss in the form of bleeding, diarrhea, sweating, etc., or by internal fluid loss due to extravasation, exudation, or transudation. Volume expansion is best achieved by infusing boluses of the desired fluid and evaluating the appropriate hemodynamic response or lack thereof. A 20 mL/kg bolus is usually recommended if a crystalloid is used. A 10 mL/kg bolus is recommended when using colloid.

Volume expansion is key to reversing inadequate oxygen delivery. Maximizing cardiac preload is the most sufficient way to support cardiac output and delivery of oxygen to the tissues. Thus, fluid therapy should be considered inotropic support and should be titrated to effect. If oxygen delivery has been maximized, and there is evidence of adequate peripheral perfusion, in most settings there is no advantage to additional fluid therapy. In addition, if cardiac output and oxygen delivery are still not normal after significant volume expansion, one must question whether additional fluid would be beneficial. In those cases, increasing cardiac contractility by means of pharmacologic inotropic support may be a better choice. Overzealous fluid administration creates interstitial edema, which may prove to be particularly injurious in the pulmonary circulation. This clearly is a dynamic situation and may require minute-to-minute fine-tuning. In such cases, a direct measurement of cardiac performance using invasive or noninvasive technology may be helpful.

There is a wide range of options for the resuscitation fluid. We will not discuss blood products, as they are beyond the scope of this article, but will concentrate on crystalloids,

Table 1: Crystalloid Solution Electrolytes Composition

Solution	NA (mEq/L)	Cl (mEq/L)	K (mEq/L)	Destrose (g/L)	Lactate (mEq/L)	Ca (mEq/L)	pH	mOsm/L	Calories (Kcal/L)	Other
0.45% Saline	77	77	0	0	0	0	5.7	155	0	
D5 water	0	0	0	50	0	0	5.0	252	170	
Lactated Ringer's	130	109	4	0	28	3	6.7	273	9	
Normosol (P-lytes)	140	98	5	0	0	0	7.4	295	15	Gluconate 23 mEq/L Acetate 27 mEq/L
0.9% Saline	154	154	0	50	0	0	5.7	308	0	
Ringer's	147	156	4	0	0	4.5		309	0	
D5 0.45% saline	77	77	0	50	0	0	5.2	406		
D5 Lactated Ringer's	130	109	4	50	28	3	6.7	525	170	
3% saline	513	513	0	0	0	0	5.7	1025	0	
5% saline	855	855	0	0	0	0	5.7	1710	0	
* Na = Sodium; Cl = Chloride; K = Potassium										

colloids, and hypertonic solutions. The fluid choice most often reflects the personal belief of the attending physician and/or availability of one fluid or another, rather than evidence-based medicine. However, recently performed large randomized clinical trials have shown that there is no clear benefit of colloid over crystalloid.[10] The choice of fluid does have considerable cost implications in that colloids are significantly more expensive than crystalloids.

Crystalloids

A crystalloid solution is a non-ionic or ionic particle solution. Crystalloid solutions are classified as hypotonic, isotonic, and hypertonic based on their osmolarity properties. In resuscitation, solutions that are closer to physiological osmolarity are favored because they predominantly remain intravascularly. Since they do not contain protein or large particles, they do not alter osmotic pressure; therefore, water can freely cross the microvascular membrane. Hypotonic solutions will shift water into the ICF, whereas hypertonic solutions tend to shift water into the ECF. Normal saline, lactated Ringer's, Ringer's, and Normosol (P-Lytes) are the main varieties of isotonic solutions for resuscitation. Normal saline is actually slightly hypertonic since its osmolarity is 308 mOsm/L. Lactated Ringer's is close to physiological osmolarity, but contains lactate.

To date, there is no trial assessing the benefits of one isotonic solution compared to another. Isotonic solution will rapidly distribute itself across the ECF. The kinetics of crystalloid vary depending on volume status. However, one can predict that 75% of the volume will go to the interstitial space and 25% will remain within the intravascular space. Some crystalloid solutions come with dextrose, which mainly serves a caloric purpose. However, dextrose is ineffective at replacing intravascular fluid loss. (*See Table 1 for electrolyte composition of crystalloids.*)

Colloids

Colloids are fluid solutions containing particles large enough to cause an osmotic pressure change across the microvascular membrane. They are either a protein or a synthetic protein. In comparison to crystalloids, colloids stay in the intravascular space longer. Their duration depends on the molecular size and ionic charge. Protein colloids include human serum albumin, which is derived from pooled human serum in 5% and 25% concentrations. Albumin is the only colloid that maintains a uniform molecular weight, whereas the synthetic colloids are polymers with a wide range of molecular weights. This means the molecular weight of the synthetic colloid is the total weight of all molecules divided by the total number of molecules.

The often-mentioned theoretical

Table 2: Colloid Solution Electrolytes Composition

Solution	NA (mEq/L)	Cl (mEq/L)	Lactate (mEq/L)	Albumin (g/L)	Dextran (g/L)	HES (g/L)	pH	mOsm/L	Oncotic Pressure (mmHg)	Other
Albumin 5%	154	154	0	50	0	0	6.6	290	20	
Albumin 25%	154	154	0	250	0	0	6.9	310	100	
Hetastarch: Hespan Hextend	154 130	154 109	0 28	0 0	0 0	60 60		310 310	30 30	K 4 Meq/L
Dextrans: Dextran 40 Dextran 70	154 154	154 154	0 0	0 0	100 60	0 0	6.7 6.3	320 320	68 70	
Gelatins: Gelofusine Haemaccel	154 145	125 145	0 0	0 0	0 0	0 0	7.4 7.4	325 325	30 30	Ca 6.25 mmol/L K 5.1 mmol/L

* Na = Sodium; Cl = Chloride; HES = Hetastarch

disadvantage of lactated Ringer's solution is that the infusion of additional lactate will contribute to the existing metabolic acidosis. This hypothesized concern has not be validated in human trials and, in fact, the problem with lactated Ringer's is that of any other crystalloid fluid, under-resuscitation. If adequate amounts of crystalloid are used to restore circulating volume and oxygen delivery, the additional lactate is metabolized by the liver and does not contribute to perpetuating or exacerbating the metabolic acidosis.

Synthetic protein solutions come in various formulations. Hydroxyethyl starch (HES) is made from amilopectin, which, itself, is derived from cornstarch with branched glucose polymers. In plasma, however, they are rapidly hydrolyzed and destroyed within 10 minutes. Adding a hydroxyethyl group to the glucose subunits extends the duration of action within the plasma. The renal system is unable to eliminate starches due to the size of the molecule; therefore, they must be cleared in the liver. All starches affect the coagulation system by altering the hepatic-dependent cofactors in the coagulation cascade. Starches include Hetastarch (Hespan® and Hextend®), Pentastarch (HAES-steril 6%, HAES-steril 10%), Pentaspan 10%, and Hexastarch (EloHAES 6%).

Dextrans (dextran-40® and dextran 70®) are naturally occurring glucose polymers. Unlike other synthetic colloids, they are eliminated by the kidneys and, thus, will not affect coagulation. Unfortunately, there is an inherently higher renal failure complication rate associated with their use.

Gelatins (Gelofusine® and Haemacell®) are either succinylate gelatins or urea-linked gelatins that are degradations of animal collagens. Since gelatins are from animal products, there is a rare but real possibility of anaphylactic shock when compared to other colloids. With gelatins, histamine release increases the microvascular endothelial pore size, which further decreases the volume-expanding effect. Gelatin also will interfere with fibrin polymerization, reducing the ability to form clots.

Colloid use has a theoretical advantage over crystalloids as their protein compositions prevent them from crossing the cellular membrane. This tends to lower volume requirements. One concern with the use of colloid is the cost. In addition, albumin is a serum-pooled product and, as such, it carries a relative risk of infection. New data from the recent Iraq War may suggest benefits from synthetic colloids in reducing blood product requirements, but large randomized trials are still needed. (*See Table 2 for electrolyte composition of crystalloids.*)

Hypertonic Saline

Hypertonic saline solutions (3%, 5%, and 7.5%) rapidly expand the intravascular volume by pulling water from the interstitial and intracellular space. Therefore, only small amounts of hypertonic solution are needed for the expansion of the intravascular space. Because the effects of hypertonic solutions are short lived, it has been postulated that adding a dextran base would prolong half-life.

Several small trials in hypovolemia and traumatic brain injury have studied the effect of hypertonic resuscitation on the goal of restoring blood pressure while reducing the amount of fluid volume resuscitation. Using 7.5% saline in a dextran-based solution, these trials have argued that the efficacy of hypertonic resuscitation has reduced the necessity of massive fluid resuscitation in these patients. One of the key limiting factors in all these trials is the lack of human trials at the recommended sodium levels

of 148-154 meq/L. In several trials, physicians were reluctant to administer hypertonic saline when sodium concentrations reached 147 meq/L.

In addition, the placement of hypertonic saline within a dextran-based solution has been criticized. A multicenter study on the effect of hypertonic resuscitation in pre-hospital patients had to be suspended due to the interim data analysis, which showed that patients receiving hypertonic saline in the field had increased mortality compared to the normal saline group. However, the overall death rate at 28 days did not differ between the two groups. A subset analysis of the data showed a positive trend in outcome in patients with traumatic brain injury treated with hypertonic saline. The study was modified and reinstated to enroll only traumatic brain injury patients.[14] **At this point the authors cannot recommend the use of hypertonic saline for the resuscitation of trauma patients except possibly for patients with traumatic brain injury.**[15-19]

Benefits of Colloid vs. Crystalloid

The colloid-crystalloid controversy has been going on for more than 50 years, probably due to the lack of quality studies and data to support either. There is little debate that larger volumes of crystalloid are required to restore intravascular volume than colloid. The American Thoracic Society Consensus Statement on colloid use in the critically ill[20] states that the synthetics have come under scrutiny because of their association with coagulopathy, anaphylaxis, and, on rare occasion, end organ damage. It further states that the only role of colloid is in the therapy of dialysis-related hypotension and in the management of ascites requiring paracentesis.[20]

Colloids restore intravascular volume and tissue perfusion more rapidly than crystalloids in all shock states regardless of vascular permeability.[21] All colloids affect the coagulation system, with dextran and starch solutions having the most potent antithrombotic effects. Albumin has modest anticoagulant and antithrombotic effects.[22,23] Colloids should be used with caution, particularly in cardiopulmonary bypass and in patients with sepsis.[24] There is no evidence of benefit of colloid use in treating ischemic brain injury or subarachnoid hemorrhage. Colloids may adversely impact survival in traumatic brain injury.[25]

Treatment of dialysis-related hypotension with colloids is superior to crystalloids for chronic dialysis patients.[26] Colloids are superior to crystalloids in intravascular volume replacement with large-volume paracentesis, and as adjunctive therapy to antibiotics in treating spontaneous bacterial peritonitis.[27] However, use of HES may increase the risk of acute renal failure in patients with sepsis.[28]

Although crystalloid and colloid each have advantages in a few specific clinical situations, there was no adequately powered large multicenter, randomized, double-blind trial to compare the effect of colloid vs. crystalloid in clinical patient outcomes in a heterogeneous population of all critical ICU patients until the SAFE trial was published in June 2004.[29] It compared the effects of normal saline vs. 4% albumin in 6,997 patients. This trial had insufficient power to detect small but important differences in mortality among subgroups such as the ones listed by the American Thoracic Society consensus statement,[20] but showed no difference in clinical outcomes at 28 days in the ICU among all patients. Requirements for mechanical ventilation and renal-replacement therapy, time spent in the ICU and in the hospital during the 28-day study period, and the time until death (among patients who died) were equivalent.

According to the SAFE trial, albumin and saline should be considered clinically equivalent treatments for intravascular volume resuscitation in a heterogeneous population of patients in the ICU.

Conclusion

In conclusion, we have discussed the pathophysiology and physiology of shock and the importance of fluid resuscitation in shock. While there continues to be debate among experts about the use of colloid vs. crystalloid, current data have demonstrated no clinically significant differences between the two fluid choices, with a few notable exceptions in dialysis-related hypotension and before paracentensis in treating spontaneous bacterial peritonitis.

To review the two case studies, both patients present with signs of hypoperfusion. The first patient has been vomiting and has clear signs and symptoms of hypovolemic shock. He is cold, clammy, and hypotensive. The patient is suffering from hypotension most likely related to hypovolemia, although an abdominal catastrophe cannot be ruled out. In this patient, restoration is paramount. There is no added benefit of colloid over crytalloid. Therefore, replacing volume with crystalloids is economical, efficient, and without undue complications. Until the patient is stablilized, the authors recommend two large-bore short IVs using a volume bolus of 20 mL/kg of normal saline to a maximum of 2 liters.

In the second case, the patient does not show clear signs of shock. His blood pressure is normal. His heart rate is inconsistent with his temperature, and a provider may be falsely reassured by this patient's vital signs. In actuality, this normotensive patient with an elevated lactate level likely has cryptic shock. In the context of fever and cough, one must consider sepsis. Early goal-directed therapy for sepsis recommends using two liters of crystalloid for the restoration of volume perfusion to reverse the microvascular hypoperfusion. Again, these authors recommend using small boluses of 20 mL/kg of normal saline since lactated Ringer's contains lactate and, in theory, could increase lactic acidosis. We hope that this article has given you information and evidence to more clearly guide the assessment and resuscitation of fluid status in critically ill patients.

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Physician CME Questions

1. Dextrans are:
   A. eliminated by the liver and associated with coagulopathy
   B. eliminated by the kidney and associated with increased renal failure rates
   C. eliminated by both liver and kidneys
   D. eliminated by neither liver, nor kidneys

2. In reference to the oxygen delivery calculation, what is the single most important determinant of oxygen delivery?
   A. cardiac output
   B. hemoglobin
   C. oxygen saturation
   D. increased barometric pressure

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

3. Another term for normotensive shock is:
   A. hypovolemic shock
   B. septic shock
   C cryptic shock
   D. cardiogenic shock

4. In a non-pathological scenario, lactate levels are generally less than:
   A. 5 mMol/L
   B. 4 mMol/L
   C. 3 mMol/L
   D. 2 mMol/L

5. Which of the following statements regarding intravenous colloid solutions derived from starches is *incorrect*?
   A. The renal system is unable to eliminate starches due to their molecular size.
   B. All starches potentially affect the coagulation system.
   C. Starches have a small molecular size.
   D. All starches are cleared by the liver.

6. When adding 1 L of saline intravenously to a patient, it can be expected to increase the extracellular volume by:
   A. 500 mL
   B. 400 mL
   C. 300 mL
   D. 200 mL

7. According to Poiseuille's law, which of the following statements regarding laminar flow rates is true?
   A. They are directly proportional to the catheter length.
   B. They are indirectly proportional to the 4th power of the catheter radius.
   C. They are directly proportional to the 4th power of the catheter length.
   D. They are directly proportional to the 4th power of the catheter radius.

8. According to the SAFE trial:
   A. Albumin is considered superior to saline for IV resuscitation in heterogeneous ICU populations.
   B. Saline is considered superior to albumin for IV resuscitation in heterogeneous ICU populations.
   C. Aggressive use of fluid management in ARDS shortens duration of mechanical ventilation.
   D. Albumin and saline are considered clinically equivalent for IV resuscitation in heterogeneous ICU populations.

9. A multicenter study on the effect of hypertonic resuscitation in pre-hospital patients:
   A. was modified and reinstated to enroll only traumatic chest injury patients
   B. was modified and reinstated to enroll only traumatic brain injury patients
   C. showed decreased mortality compared to the normal saline group
   D. showed a negative trend in patients with traumatic brain injury

10. Pulmonary artery catheters have been associated with which of the following?
   A. increased complications and mortality
   B. decreased complications and mortality
   C. recommendations for extended use
   D. increasing popularity

In Future Issues

ENT Emergencies

CME Answer Key

1. B; 2. A; 3. C; 4. D; 5. C; 6. D; 7. D; 8. D; 9. B; 10. A

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Controversies in Fluid Resuscitation

Calculation

1) Oxygen Delivery:

$DO_2 = CO \times CaO_2$, where $CaO_2 = (Hbg \times 1.39 \times SaO_2)$*

* CO: cardiac output; Hbg: hemoglobin; CaO_2: oxygen content; SaO_2: O_2 saturation

2) Cardiac Output:

CO = HR x SV

* CO: cardiac output; HR: heart rate; SV: stroke volume

Distribution of Body Water

Crystalloid Solution Electrolytes Composition

Solution	NA (mEq/L)	Cl (mEq/L)	K (mEq/L)	Destrose (g/L)	Lactate (mEq/L)	Ca (mEq/L)	pH	mOsm/L	Calories (Kcal/L)	Other
0.45% Saline	77	77	0	0	0	0	5.7	155	0	
D5 water	0	0	0	50	0	0	5.0	252	170	
Lactated Ringer's	130	109	4	0	28	3	6.7	273	9	
Normosol (P-lytes)	140	98	5	0	0	0	7.4	295	15	Gluconate 23 mEq/L Acetate 27 mEq/L
0.9% Saline	154	154	0	50	0	0	5.7	308	0	
Ringer's	147	156	4	0	0	4.5		309	0	
D5 0.45% saline	77	77	0	50	0	0	5.2	406		
D5 Lactated Ringer's	130	109	4	50	28	3	6.7	525	170	
3% saline	513	513	0	0	0	0	5.7	1025	0	
5% saline	855	855	0	0	0	0	5.7	1710	0	

* Na = Sodium; Cl = Chloride; K = Potassium

Colloid Solution Electrolytes Composition

Solution	NA (mEq/L)	Cl (mEq/L)	Lactate (mEq/L)	Albumin (g/L)	Dextran (g/L)	HES (g/L)	pH	mOsm/L	Oncotic Pressure (mmHg)	Other
Albumin 5%	154	154	0	50	0	0	6.6	290	20	
Albumin 25%	154	154	0	250	0	0	6.9	310	100	
Hetastarch: Hespan Hextend	154 130	154 109	0 28	0 0	0 0	60 60		310 310	30 30	K 4 Meq/L
Dextrans: Dextran 40 Dextran 70	154 154	154 154	0 0	0 0	100 60	0 0	6.7 6.3	320 320	68 70	
Gelatins: Gelofusine Haemaccel	154 145	125 145	0 0	0 0	0 0	0 0	7.4 7.4	325 325	30 30	Ca 6.25 mmol/L K 5.1 mmol/L

* Na = Sodium; Cl = Chloride; HES = Hetastarch

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