FLUID RESUSCITATION IN TRAUMA

Dr. S.A. Rajkumar,
Tirunelveli

Introduction

Trauma is fast becoming a major cause for increased mortality in India. Haemorrhage accounts for 40% of trauma-related deaths. It is well established that, appropriate fluid replacement is an essential component of trauma resuscitation. There are many issues regarding trauma fluid resuscitation. When to start the fluid resuscitation, how to assess the severity of injury, which type of fluid to be resuscitated. Since uncontrolled hemorrhage is one of the leading causes of early death after trauma, the treatment of these casualties at the site of the injury or during transportation to the hospital remains an important issue. Therefore, after ensuring an adequate airway, oxygenation and ventilation, the focus for resuscitation of the severely injured patient switches to approach to appropriate and effective fluid replacement to reverse hemorrhagic shock and to restore perfusion to vital organs. Once hemorrhage is controlled, restoration of normovolemia is a priority. In the presence of uncontrolled haemorrhage, aggressive fluid management may be harmful. Though in the initial stages of trauma resuscitation the precise fluid used is probably not important as long as an appropriate volume is given, we should have a clear idea about the need of the patient, once we are able to infuse any type of fluid we want.

Each decade has brought improvements in our understanding of shock following trauma. With more understanding of the underlying mechanisms, we have been able to define more clearly optimal resuscitation strategies.

Historical perspective:

In 1628, with the discovery of circulation of blood by William Harvey led to further development. In 1662, first injection intravenously was made in man by J.D. Major. In 1665 a dying animal was successfully transfused with blood. In 1667, a boy was transfused with lamb blood. Eventually he died. In 1900, Dr. Karl Landsteiner identified various blood groups. The first IV fluid was administered in 1832 for a cholera victim. NS was used successfully intravenously in the early 1900s. Blalock in 1932 demonstrated in canine experiments that plasma fluid could escape into injured tissue in volumes sufficient to produce hypotension. In 1935 continuous method of IV transfusion published.
**Physiology:**

Shock is an imbalance in the oxygen supply to tissues relative to the needs of the tissues. It leads eventually to multi organ failure as endpoint (fig. 1). Loss of intravascular volume causes increased vascular tone. Blood flow is redistributed among the organ systems of the body and perfusion to the heart and brain is maintained at the expense of cutaneous, splanchnic and renal vascular beds. Intravascular volume deficits cause increase in sympathetic tone. Catecholamine levels are increased to increase cardiac output and blood pressure. Release of prostaglandins cause local vasodilatation, whereas thromboxane A2 release, cause vasoconstriction. Platelet-activating factor causes coronary vasoconstriction and increases platelet aggregation. Capillary hydrostatic pressure is decreased and the extracellular fluid is depleted as transcapillary refill occurs. Acidosis initially facilitates the unloading of oxygen to the tissues due to shift of oxygen dissociation curve. Urine output is diminished as water and sodium are retained. Uncontrolled haemorrhage leads to acutely fatal shock, characterized by complete failure of the cardiovascular system: loss of contractile power in the heart and great vessels, inappropriate vasodilation, loss of response to catecholamines and, eventually, brain death.

![Figure 1](image.png)

**Pathophysiology**

In simple terms, shock is failure of adequate oxygen delivery to the tissues of the body. Hypoperfusion leads to cellular ischaemia, leading in turn to ‘hibernation’ (the cell reduces its level of metabolic activity), anaerobic metabolism (absent oxygen, the cell must produce lactic acid as it generates energy), apoptosis (the cell begins a programmed shutdown process) and outright necrosis (cell death) [Figure 2]. The ischaemic cell takes up interstitial fluid (perhaps to dilute accumulating metabolic poisons) and swells, reducing perfusion to its neighbours. Lactate, and other toxic metabolites, affect the cells. The net effect is a biological cascade that, if unchecked, can be fatal.
Clinically, shock is characterized by the symptoms of the body's response to hypoperfusion. Low blood pressure, tachycardia, decreased urine output, pale skin and diaphoresis are all characteristic.

Acute hemorrhage causes a decreased cardiac output and decreased pulse pressure. These changes are sensed by baroreceptors in the aortic arch and atrium. With a decrease in the circulating volume, neural reflexes cause an increased sympathetic outflow to the heart and other organs. The response is an increase in heart rate, vasoconstriction, and redistribution of blood flow away from certain nonvital organs, such as the skin, gastrointestinal tract and kidneys.

Concurrently, a multisystem hormonal response to acute hemorrhage occurs. Corticotropin-releasing hormone is stimulated directly. This eventually leads to glucocorticoid and beta-endorphin release. Vasopressin from the posterior pituitary is released, causing water retention at the distal tubules. Renin is released by the juxtamedullary complex in response to decreased mean arterial pressure, leading to increased aldosterone levels and eventually to sodium and water resorption. Hyperglycemia commonly is associated with acute hemorrhage. This is due to a glucagon and growth hormone--induced increase in gluconeogenesis and glycogenolysis. Circulating catecholamines relatively inhibit insulin release and activity, leading to increased plasma glucose.
In addition to these global changes, many organ-specific responses occur. The brain has remarkable autoregulation that keeps cerebral blood flow constant over a wide range of systemic mean arterial blood pressures. The kidneys can tolerate a 90% decrease in total blood flow for short periods of time. With significant decreases in circulatory volume, intestinal blood flow is dramatically reduced by splanchnic vasoconstriction. Early and appropriate resuscitation may avert damage to individual organs as adaptive mechanisms act to preserve the organism.

**Trauma associated coagulopathy:**

Coagulopathy is an inevitable consequence of massive bleeding. The coagulopathy of trauma appears to be the sum of the effects of hypothermia, acidosis, and clotting element consumption, loss, and dilution. Hypothermia acts predominantly on platelet activation and adhesion but it also slows the metabolic rates of the coagulation factor enzymes. Acidosis predominantly affects activities of the enzyme complexes on lipid surfaces. Embolization of brain substance, marrow fat, amniotic fluid, and other strong thrombo-plastins causes disseminated intravascular coagulation, with consumption of coagulation factors. Extensive soft tissue trauma with multiple disruptions of endothelial surfaces has a similar effect. The direct loss of clotting factors through hemorrhage rapidly reduces the body's small normal stores of 10 g of fibrinogen and 15 ml of platelets. Resuscitation, even with blood components, causes further dilution (fig. 3).

**Assessment of traumatic shock:**

When the patient is received in the emergency department, assessing whether the patient is in shock or at the verge of shock is important. If the patient is in shock, it is also important to assess whether the shock is due to hemorrhage or other causes. Table 1 shows differential diagnosis for shock.
Table 1. Differential Diagnosis for Shock in Trauma

<table>
<thead>
<tr>
<th>No.</th>
<th>Etiologies of Shock in Trauma</th>
<th>Symptoms &amp; Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Hemorrhage / volume loss</td>
<td>Narrow pulse pressure, slowing of external bleeding without intervention, bleeding from trauma.</td>
</tr>
<tr>
<td>2.</td>
<td>Tension pneumothorax</td>
<td>Deviated trachea, absent unilateral breath sounds, distended neck veins, narrow pulse pressure, pulsus paradoxus.</td>
</tr>
<tr>
<td>3.</td>
<td>Pericardial tamponade</td>
<td>Distended neck veins, muffled heart sounds, narrow pulse pressure, pulsus paradoxus.</td>
</tr>
<tr>
<td>4.</td>
<td>Myocardial contusion</td>
<td>Tachycardia out of proportion to other injuries, abnormal ECG, elevated cardiac enzymes.</td>
</tr>
<tr>
<td>5.</td>
<td>Neurogenic shock</td>
<td>Spinal injury above T6, bradycardia, warm extremities.</td>
</tr>
</tbody>
</table>

Whatever may be the cause, unless otherwise proved, the patient must treated as in hemorrhagic shock. Once the hemorrhagic shock is identified, treatment should begin immediately. Classic teaching from ATLS divides hemorrhagic shock into categories based on the percentage of blood volume lost and expected accompanying vital signs and physiologic features. (See Table 2)

Table 2. Classes of Shock by ATLS (in Adult)

<table>
<thead>
<tr>
<th></th>
<th>Class 1</th>
<th>Class 2</th>
<th>Class 3</th>
<th>Class 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (%)</td>
<td>&lt; 15 %</td>
<td>15 – 30 %</td>
<td>30 – 40 %</td>
<td>&gt; 40 %</td>
</tr>
<tr>
<td>Heart rate (beats / min.)</td>
<td>&lt; 100</td>
<td>100 - 120</td>
<td>120 – 140</td>
<td>&gt; 140</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal / increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Respiratory rate (breaths / minute)</td>
<td>14 – 20</td>
<td>20 – 30</td>
<td>30 – 40</td>
<td>&gt; 35</td>
</tr>
<tr>
<td>Mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious, confused</td>
<td>Confused, lethargic</td>
</tr>
</tbody>
</table>
**Diagnosis:**

Hemorrhagic shock patient may be subjected to the following tests not only to identify the amount of shock or bleeding but also to help in managing the patient.

1. **Laboratory investigations:**
   - Complete blood count
   - Blood type and crossmatch
   - Coagulation profile, including prothrombin time (PT), partial thromboplastin time (PTT), and international normalized ratio (INR)
   - Blood gas analysis and lactate level.
   - Toxicology studies, including alcohol level and drug screen, as appropriate
   - Pregnancy test, as appropriate

2. **Ultrasound**
   - FAST (Focused Assessment with Sonography for Trauma)
   - eFAST (extended FAST – includes thorax)

3. **X-ray, CT and MRI**
   - In emergencies MRI has no role and CT has minimal role.

**Treatment of traumatic shock:**

Severe hypovolemia is associated with cardiovascular decompensation, reduced cellular perfusion and oxygen delivery and the development of profound lactic acidosis. If oxygen delivery is not restored quickly, cell membrane pumps fail and cellular function will not recover even if adequate oxygen delivery is restored. Depending on the number of cells sustaining irreversible damage organ failure or death may ensue. Therefore, the rationale behind fluid replacement in the trauma patient is to minimize the number of irreversibly damaged cells by restoring adequate tissue perfusion and oxygen delivery as rapidly as possible.

Few would argue that the best resuscitation fluid is blood. Yet in emergencies, due to limited availability and other practical reasons as cross matching push us to go for suitable alternative fluids.

**Resuscitation fluids:**

The resuscitation fluids available for IV infusion are broadly divided into three categories. They are crystalloids, colloids and blood / blood products. Let us see them in detail:
1. Crystalloids:

A crystalloid is a solution of nonionic or ionic particles. The contents of a number of commonly used crystalloids are listed in Table 3. Most crystalloid intravenous fluids are isotonic with plasma. They do not contain larger oncotic particles and will, therefore, pass freely across the microvascular membrane. Their precise distribution will be determined by their sodium concentration. Solutions containing approximately isotonic concentrations of sodium (0.9% saline or Ringer’s solution) will distribute rapidly across most of the extracellular space. Crystalloids containing less than isotonic concentrations of sodium will be increasingly distributed to the intracellular space. Dextrose 5% is distributed throughout the total body water and is ineffective for replacing intravascular fluid loss. Hypertonic solutions are distributed less freely into the interstitial space and stay longer in plasma.

Some basic characters of different crystalloids merit to be mentioned here are:

a. NS and RL are the only crystalloids that do not have dextrose (calorie value).
b. RL is the most physiological solution (similar constitution to ECF).
c. Only fluid that corrects metabolic alkalosis is Isolyte G (due to ammonium chloride content).
d. Fluids that correct metabolic acidosis are Isolyte M, Isolyte P and Isolyte E.
e. Isolyte M has highest potassium concentration. It is a maintenance fluid.
f. Isolyte G is gastric replacement fluid and is useful in vomiting and metabolic alkalosis.
g. Isolyte P is paediatric maintenance solution with high potassium content and high water content. It is also indicated in diabetes insipidus.
h. Isolyte E is extracellular fluid replacement solution used in early trauma management.

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Dextrose</th>
<th>Na mEq/l</th>
<th>K mEq/l</th>
<th>Cl mEq/l</th>
<th>Lactate mEq/L</th>
<th>Acetate mEq/L</th>
<th>Ca mEq/L</th>
<th>NH₄Cl mEq/L</th>
<th>HPO₄ mEq/L</th>
<th>Citrate mEq/L</th>
<th>Osmolarity mOsm/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>5% D</td>
<td>50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>278</td>
</tr>
<tr>
<td>NS</td>
<td>-</td>
<td>154</td>
<td>-</td>
<td>154</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>308</td>
</tr>
<tr>
<td>DNS</td>
<td>50</td>
<td>130</td>
<td>195</td>
<td>109</td>
<td>28</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>586</td>
</tr>
<tr>
<td>RL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>130</td>
<td>109</td>
<td>28</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>274</td>
</tr>
<tr>
<td>Isolyte M</td>
<td>50</td>
<td>40</td>
<td>35</td>
<td>40</td>
<td>-</td>
<td>20</td>
<td>-</td>
<td>-</td>
<td>15</td>
<td>-</td>
<td>410</td>
</tr>
<tr>
<td>Isolyte P</td>
<td>50</td>
<td>25</td>
<td>20</td>
<td>22</td>
<td>-</td>
<td>23</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>3</td>
<td>368</td>
</tr>
<tr>
<td>Isolyte G</td>
<td>50</td>
<td>63</td>
<td>17</td>
<td>22</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>70</td>
<td>-</td>
<td>-</td>
<td>580</td>
</tr>
</tbody>
</table>

| Plasma  | 5       | 142      | 4.5     | 103      | 1.5           | -             | 2.5      | -           | 1           | -              | 291              |
Some of the commonly available crystalloids and their characteristics:

1) 5% Dextrose solution:
   a. It contains 50 g of glucose in 1 litre.
   b. It does not contain any electrolytes.
   c. The glucose in 5D is metabolised and water is distributed into all compartments of the body.
   d. Stay in the plasma volume is very poor for 5D.
   e. It is used in correction of hypernatremia.
   f. Causes cerebral edema – so not to be used in CNS surgeries and head injuries.
   g. It is not compatible with blood.

2) NS:
   a. It contains 154 mEq of Na\(^+\) and 154 mEq of Cl\(^-\) in 1 litre of NS.
   b. It does not contain dextrose hence no calorie value.
   c. It increases only ECF volume.
   d. It is used in dehydration due to vomiting, diarrhoea, diuresis or excessive sweating.
   e. It is also used in the treatment of alkalosis and initial stages of DKA (diabetic ketoacidosis).
   f. It is compatible with blood transfusion.

3) DNS:
   a. It contains 50 g of glucose and 154 mEq of Na\(^+\) and 154 mEq of Cl\(^-\) in 1 litre.
   b. It corrects dehydration & electrolyte deficiency and so used in or nasogastric aspiration induced alkalosis and hypochloremia.
   c. It is not preferred in severe hypovolemic shock (due to osmotic diuresis by dextrose and further hypovolemia).
   d. It is compatible with blood transfusion.

4) RL:
   a. It is the most physiological solution available.
   b. It is very much useful in hypovolemia.
   c. The lactate in RL is metabolised in liver into bicarbonate and so mild alkalosis occurs. (hence preferred in many surgical conditions where slight metabolic acidosis prevails)
   d. It is preferred in burns, trauma or other fluid loss conditions.
   e. RL is not compatible with blood transfusion.

5) Hypertonic saline solution:
a. 3% NS with 1000 mOsm/L osmolality as opposed to 0.9% NS is hypertonic in nature and retain in the plasma for longer time. Smaller volumes are required (10-12 ml/Kg) to resuscitate.
b. 7.5% with osmolality 2400 mOsm/L is also available. The smaller quantity requirement (4-5 ml/Kg) makes it attractive for use during prehospital resuscitation.
c. They elevate mean arterial pressure and cardiac output. They also increase renal, mesenteric, total splanchnic and coronary blood flow.
d. By mixing with colloid the plasma stay can be increased substantially.
e. They are very much useful in controlled hemorrhage. But if the hemorrhage is uncontrolled, they are not advised (because of the increased blood pressure and rebleeding).

2. Colloids:

A colloid is a fluid, containing particles that are large enough to exert an oncotic pressure across the microvascular membrane. In comparison to the crystalloids, they have greater intravascular persistence. The duration of intravascular persistence depends on molecular size, shape and ionic charge. Albumin is the only colloid containing particles of uniform molecular weight (monodisperse). The other colloids are polymers and contain particles with a wide range of molecular weights. Albumin and dextran are naturally occurring colloids. (Blood is also a colloid in technical sense.)

Semisynthetic colloids include modified gelatins, hetastarch (hydroxyethyl starch) and hemoglobin solutions. Colloids allow a more rapid resuscitation with lower volume of fluid. For every 100 of colloid infusion, it expands plasma volume as given in the table 4. The table also depicts about the duration of stay in the plasma of a particular colloid.

**Characteristics of IV colloid fluids per 100 ml infusion:**

<table>
<thead>
<tr>
<th>Colloid</th>
<th>Effective Plasma Volume expansion</th>
<th>Duration of stay</th>
</tr>
</thead>
<tbody>
<tr>
<td>5% Albumin</td>
<td>70 – 130 ml</td>
<td>16 hrs.</td>
</tr>
<tr>
<td>25% Albumin</td>
<td>400 – 500 ml</td>
<td>16 hrs.</td>
</tr>
<tr>
<td>10% Dextran – 40</td>
<td>100 -150 ml</td>
<td>6 hrs.</td>
</tr>
<tr>
<td>6% Dextran – 70</td>
<td>80 ml</td>
<td>12 hrs.</td>
</tr>
<tr>
<td>Gelatin polymer (Haemaccel)</td>
<td>50 ml</td>
<td>4 - 5 hrs.</td>
</tr>
<tr>
<td>6% Hetastarch (HES)</td>
<td>100 – 130 ml</td>
<td>24 hrs.</td>
</tr>
<tr>
<td>10% Pentastarch</td>
<td>150 ml</td>
<td>8 hrs.</td>
</tr>
</tbody>
</table>

Table 4
When colloids are infused serum protein levels are maintained at more normal levels; fluid entering into interstitial space is also limited; therefore, the peripheral edema, usually manifest after massive resuscitation, is less seen.

Some of the commercially available colloids and their characteristics:

1) Albumin:
   a. Commercially available albumin are in two types: 5% albumin and 25% albumin.
   b. 5% albumin expands plasma volume equal to its volume. 25% albumin expands the plasma volume 4 to 5 times its volume.
   c. Albumin stays in the plasma volume for 16 hrs.
   d. It is used in acute hypovolemic shock, correction of hypoproteinemia or in plasmapheresis.
   e. It is contraindicated in cardiac failure.
   f. Albumin is not suitable for parenteral nutrition.

   Human albumin is a single polypeptide with a molecular weight of 65-69 kDa and a strong negative charge of minus 17. It has transport functions, free radical scavenging and anticoagulant properties and may have a role in preserving microvascular integrity. Human albumin solution is generally considered to be free from any risk of transmitting infection. However, a single bottle of albumin represents exposure to many thousands of donors and, there is a concern about the theoretical risk of transmission of the prion causing new variant Creutzfeldt Jakob disease.

2) Dextran:
   a. Dextran 40 (means molecular weight 40,000) stays in plasma for 6 hrs (due to small molecule size and rapid excretion) whereas dextran 70 (molecular weight 70,000) stays for 12 hrs.
   b. Used in acute hypovolemic shock, thrombo embolism or slow microcirculation.
   c. Dextran is contraindicated in bleeding disorders / CCF.
   d. It may interfere with blood grouping and cross matching.

3) Gelatin polymer (Haemaccel):
   a. It is more commonly available and used. It remains in blood for 4-5 hrs. and expand plasma volume by 50% of its volume.
   b. It is used in acute hypovolemic shock, burns, trauma, surgical blood loss etc.
   c. One disadvantage is hypersensitivity reactions do occur.
   d. It does not interfere with blood grouping and cross matching.

4) Hetastarch (HESstarch):
   a. It remains in blood for 24 hrs. and expands plasma volume by equal to its volume. (i.e.) 100 ml of HES infused will raise the PV 100 ml.
   b. It is used in acute hypovolemic shock, burns, trauma, surgical blood loss etc.
c. It should be used cautiously in CCF patients and patients with bleeding disorders.
d. It is non-antigenic and so hypersensitivity reactions are rare. So it is better than Haemaccel.
e. It does not interfere with blood grouping and cross matching.

3. Blood and blood products:

1) Blood:
   It offers the advantages of volume expansion and also transports oxygen. It also remains in the intravascular space for prolonged periods. There are, however, many disadvantages to blood as a resuscitation fluid. It must be cross-matched, which requires a specimen from the patient and time to be prepared by the blood bank. Massive transfusion can produce dilutional coagulopathy, hypocalcemia and hypomagnesemia. Blood-borne viral pathogens may be transfused, causing hepatitis or HIV.

2) FFP:
   FFP contains the labile as well as stable components of the coagulation, fibrinolytic and complement systems; the proteins that maintain oncotic pressure. It is very useful in coagulation deficiencies caused by massive hemorrhage. It is indicated during massive blood transfusion. This should be transfused only after ABO compatibility testing.

3) Platelet concentrate:
   Prepared from single donar, usually given when bleeding is due to thrombocytopenia or in massive hemorrhage when the platelet is <10,000 cells/cmm.

4) Cryoprecipitate:
   It is used along with blood and FFP during massive transfusion. It can be infused without cross matching. Usually given 4 to 5 units combined.

5) Hemoglobin solutions:
   In an effort to satisfy the need for a nonantigenic, disease-free, oxygen-carrying fluid a number of hemoglobin solutions are now at advanced stages of development. Free hemoglobin causes severe renal injury. Polymerization of the hemoglobin overcomes this problem and improves intravascular persistence. Human hemoglobin-based preparations are naturally occurring and have been studied extensively; however, there is a limited availability of outdated units of blood that are needed for production. There are three main sources for the hemoglobin solutions currently under development: (a) bovine blood, (b) out-of-date human blood and (c) recombinant hemoglobin. The products currently under investigation do not require cross-matchings, have similar dissociation curve to blood and are apparently free from risk of transmitting viral or bacterial infections. They have an intravascular half-life of about 24h. Diaspirin cross-linked hemoglobin [DCLHb (Hem Assist, Baxter)] and bovine hemoglobin
solution (Hemopure, Biopure) have a significant vasopressor effect, which is thought to result from scavenging endothelial nitric oxide.

**Vascular Access:**

In trauma patients presenting with multiple injuries and hemorrhagic shock, vascular access is important and urgent. The need of the placement and the size and number of intravenous catheters are dictated by the degree of shock, the duration and the rate of bleeding and type of injury. Access to the vascular system must be obtained promptly. This is best done by the insertion of two large-caliber peripheral intravenous catheters before any consideration is given to a central venous line. The rate of flow is proportional to the fourth power of the radius of the catheter and the pressure gradient between the catheter opening and venous system and is inversely related to its length and fluid viscosity (Poiseuille's Law). Hence, short-length, large-caliber catheters are preferred for rapid infusion of large volumes of fluid.

Location of the injury must be considered when choosing a site for venous access. One should avoid venous access in injured limbs. In patients with injuries below the diaphragm, at least one intravenous catheter should be placed in the tributary of the superior vena cava because vascular disruption of the inferior vena cava may be present.

If circumstances prevent the use of peripheral veins, large-caliber, central venous (femoral, jugular or subclavian vein) access using the Seldinger technique or saphenous vein cutdown is indicated depending on the skill and experience level of the attending physician. In children younger than six years, the placement of an intraosseous needle should be attempted before central line insertion.

**Fluid Warming:**

All intravenous fluids given to the injured patient should be prewarmed to 39°C (102.2°F) before using. Fluids can be warmed in specific warmers or microwave ovens (not for blood products). Hypothermia following a major trauma increase mortality and has a number of adverse effects:

1. Oxyhemoglobin dissociation curve is shifted to the left which impairs peripheral oxygen unloading.
2. Shivering will compound the lactic acidosis that accompanies hypovolemia.
3. Hypothermia increases bleeding by dilutional coagulopathy.
4. Hypothermia increases the risk of infection.
5. Hypothermia increases the risk of cardiac morbidity events.

**When to give fluid:**

Fluid administration is the cornerstone of acute resuscitation. As per the golden hour concept, the mortality increases proportional to the time of treatment from injury.
Golden hour concept:

<table>
<thead>
<tr>
<th>Time from injury</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 hr</td>
<td>10 %</td>
</tr>
<tr>
<td>2 hr</td>
<td>11 %</td>
</tr>
<tr>
<td>3 hr</td>
<td>12 %</td>
</tr>
<tr>
<td>4 hr</td>
<td>33 %</td>
</tr>
<tr>
<td>5 hr</td>
<td>36 %</td>
</tr>
<tr>
<td>6 hr</td>
<td>41 %</td>
</tr>
<tr>
<td>8 hr</td>
<td>75 %</td>
</tr>
<tr>
<td>More than 8 hr</td>
<td>75 %</td>
</tr>
</tbody>
</table>

Intravascular volume is lost because of hemorrhage, uptake by ischemic cells and extravasation into the interstitial space. Administration of intravenous fluids will predictably increase cardiac output and blood pressure in a hypovolemic trauma patient. Hence, it might seem logical to start rapid fluid infusion as soon as possible after trauma so that adequate perfusion is restored as quickly as possible. This implies starting fluid replacement at the scene. However, attempts to replace fluid may delay the patient's arrival in the hospital more than 30 minutes and leads to less chance of improvement in outcome in patients with severe trauma. Moreover, under some circumstances, increasing the patients blood pressure before control of hemorrhage may disrupt the hemostatic thrombus by increase in blood pressure, producing secondary hemorrhage.

Animal experiments have demonstrated decreases in mortality when animals were resuscitated to a mean arterial pressure (MAP) of 40 mm Hg versus those resuscitated to a more normal MAP. Animals resuscitated to the higher MAP had more blood loss and increased hemodilution. However, in the absence of radial pulse (or a central pulse for penetrating torso injuries), consideration should be given to administration of intravenous fluid en route to hospital.

**Which fluid for volume replacement?**

Once hemorrhage has been controlled, fluid resuscitation is important. Intravascular volume should be restored as quickly as possible in an effort to restore an adequate cardiac output and reverse tissue ischemia.

Despite the fact that most trauma patients presents the signs and symptoms of hypovolemia due to moderate-to-severe blood loss, packed red cells and blood derivatives are uncommonly transfused on the scene of the accident. It is important to carefully scrutinize the pros and cons associated with their use even in more advanced phases. Today, many different classes of fluids (other than blood and blood-derivatives) are available and their actions are best
understood by examining the relationship between the fluids used in the resuscitation of trauma victims and the body's water compartments.

Infusion of a balanced salt solution is the current standard of care with the initial bolus, as per Advanced Trauma Life Support guidelines, given "as rapidly as possible" - the usual dose is 1 L or 2 L for adults and 20 ml / Kg for children. The main controversy surrounds which fluid or fluids are the most appropriate for achieving these goals.

**Crystalloids vs colloids:**

Our body contains 60% water. Of 60%, 40% is intra cellular fluid (ICF). Remaining 20% is extra cellular fluid (ECF). ECF contains plasma volume (PV) 5% and interstitial space (ISC) 15%. The wide-ranging pharmacological and pharmacodynamic properties of the colloids emphasize the significant differences between these fluids. Crystalloids differ from colloids in its osmolarity. Crystalloids diffuse freely into the interstitial space compared to colloids (Fig. 3).

Advantages of crystalloids includes the lesser cost compared with colloids, although larger volumes are needed to reach similar endpoints. There is also generalized agreement that colloids, but not crystalloids, can cause anaphylactoid reactions. In states of increased vascular permeability, colloids tend to leak into the extravascular space, which leads to increases in colloid oncotic pressure in the interstitium, potentially increasing total lung water and pulmonary dysfunction. This increased oncotic pressure also may hamper the mobilization of third space fluid. The major disadvantages of isotonic crystalloids are their limited ability to remain within the intravascular space. Lactated Ringer's solution by the end of a 1 liter infusion expands the intravascular compartment by only 194 ml, the remaining 80% of fluid is lost to the interstitial space. Generally, two to four times as much crystalloid as colloid is required to achieve the same physiologic endpoints. The better intravascular retention of colloids in comparison with crystalloid may make it easier to interpret the results of a colloid fluid challenge.
The popular approach to fluid resuscitation of trauma patient is to use both crystalloid and colloid. After hemorrhage there will be some movement of interstitial fluid into the intravascular space while intracellular volume remains unchanged. The replacement of interstitial fluid as well as intravascular fluid would seem logical. However, patients with severe injuries will quickly develop systemic inflammatory response syndrome (SIRS) and with it, a leaky microcirculation.

In patients without capillary leak, the gelatin solutions will exert a reasonable plasma oncotic pressure but, in comparison with other colloids, this effect is short-lived (about 2 hours). Intravascular retention of dextran and hydroxyethyl starch (HES) is significantly better than gelatin. However, the potential for dextran and HES to impair coagulation, limits their use for very high volume resuscitation. This problem limits the maximum safe dose of dextran to about 1500 ml or 70 Kg weight/day. It has been recommended to restrict HES use to around 2 L / day in the average weight patient. There is some evidence that HES may improve microcirculatory perfusion, possibly by reducing endothelial swelling or by modifying leukocyte adhesion. It may inhibit some components of acute. Albumin is used in pediatrics, partly because HES is not licensed for use in children.

Hypertonic saline solutions are attractive as they provide small volume resuscitation and rapid restoration of hemodynamics with laboratory evidence of improved microcirculatory hemodynamics. The side-effects of hypertonic saline are related to their action and include hypernatremia, hyperchloremic metabolic acidosis and the risk of pulmonary edema, especially in patients with limited cardiac reserve. Another theoretical risk derives from an excessive increase of the volemia and / or arterial pressure, which could enhance bleeding. Hypertonic solutions require further clinical investigation and, as yet, are not in common use.

In comparison with blood, hemoglobin solution may provide oxygen delivery to ischaemic tissue; the acellular fluid may perfuse capillaries that are compressed by edema that would prevent the passage of red cells and hemoglobin polymer is able to filter from the circulation. Advantages of hemoglobin solutions include longer self-life, lower cost, no need for cross-matching and minimal risk of viral transmission. Concerns with hemoglobin solution include the potential for inappropriate vasoconstriction and hypertension as a result of increased scavenging of nitric oxide and potentiation of coagulopathy because of platelet impairment. Although the long-term safety of massive transfusion with any of the hemoglobin solutions has yet to be demonstrated, it is highly likely that in the future at least one of these fluids will be a routine therapy for trauma patient resuscitation.

**When to stop resuscitation:**

The conventional endpoints - heart rate, blood pressure and urine output are crude assessment of the adequacy of resuscitation following severe trauma. Hypoperfusion can coexist with normotension until severe derangements occur, causing multiple organ dysfunction syndrome. The following indicators can be used to assess the resuscitation:
1. **Base deficit** reflects the hemodynamic and tissue perfusion changes associated with hemorrhagic shock. It reflects the severity of shock, the oxygen debt, changes in oxygen delivery, the adequacy of fluid resuscitation and the likelihood of multiple organ failure and survival with reasonable accuracy in previously healthy adult and pediatric trauma patients. A base deficit between 2 and 5 mmol/L suggests mild shock, between 4-14 mmol/L is a sign of severe shock. On admission base deficit in excess of 5 to 8 mmol/L correlates with increased mortality.

2. Serum lactate determination is a reliable marker of hypoperfusion in hemorrhagic shock. Lactate levels are a measure of anaerobic metabolism secondary to inadequate oxygen supply. The amount of lactate produced is believed to correlate with the total oxygen debt, the magnitude of hypoperfusion and thus, the severity of shock. Although initial lactate levels may not correlate with outcome, the ability to clear lactate to normal seems to predict adequate resuscitation. Serum lactate levels also can be followed as the resuscitation continues. The normal plasma lactate concentration is 0.5 to 1.5 mmol/L; levels above 5 mmol/L indicate significant lactic acidosis. Failure to clear lactate within 24 hours after circulatory shock is a predictor of increased mortality.

3. Organ specific monitoring: The splanchnic bed is a region that affected earliest by hypoperfusion. Therefore, measuring intramucosal (gastric mucosal) pH may allow more rapid identification of hypoperfusion. Sustained decrease in intracellular pH correlates with systemic and intraabdominal complications, such as intraabdominal abscesses, bacteremia and intraabdominal hypertension.

4. Sublingual capnometry is a noninvasive addition to organ perfusion monitoring. In trauma patients in the early phase of shock, it detected hemorrhage as accurately as base deficit and plasma lactate. The device consists of a sublingual CO$_2$ sensor that directly measures mucosal PCO$_2$. The normal value for SLPCO$_2$ is 45 to 50 mm Hg; elevated levels suggest organ hypoperfusion.

**Summary:**

During the management of hemorrhagic shock in adults, children and infants the points to be followed can be summarized as follows:

**Prehospital scenario:**

- It is recommended that in the prehospital management of adults and older children, intravenous fluid should not be administered if a radial pulse can be felt (or, for penetrating torso injuries, if a central pulse can be felt).
- In the absence of a radial pulse (or, central pulse for penetrating torso injuries) in adults or older children, it is recommended that intravenous fluid should be administered in boluses of no more than 250 ml. The patient should then be reassessed and the process repeated until a radial pulse (or, central pulse for penetrating torso injuries) is palpable.
• The administration of intravenous fluid should not delay transportation to hospital but when given in accordance with the recommendation above, consideration should be given to administration en route to hospital.
• It is recommended that when intravenous fluid is indicated in the prehospital setting, crystalloid solutions should be the routine choice.
• Transfer to hospital should not be delayed by attempts to administer intravenous fluid.

**Hospital scenario:**

- Fluid therapy should be titrated against response to guard against over resuscitation. Blood pressure targets could be established, that will maintain better systemic perfusion and reduce the risks of causing further hemorrhage (systolic blood pressure of 80 mm Hg is appropriate for tissue perfusion and minimizing hemorrhage).
- Resuscitation is not a substitute for definitive bleeding control. So try to control the source of bleeding as early as possible.
- Try to have venous access with large bore short catheters.
- Diagnosis of source of bleeding and assessment of amount of blood loss are also important. They are done without slowing the transfusion of fluid.
- Crystalloids are used along with colloids. Crystalloids are given approximately 3 times the assessed blood loss. Colloids are given 1 to 2 times of blood. Blood is given equal volume of loss. Blood and FFP are given in the ratio of 2:1.
- Coagulation status also should be assessed frequently.
- In the presence of SIRS, when the microcirculation is 'leaky', there may be some advantages to high or medium weight colloids such as hydroxyethyl starch. At this stage, gelatins (smaller molecular weight colloid) offer little advantage over crystalloids.
- Hypertonic saline solutions may have some benefit in patients with head injuries.
- The patient should not be overresuscitated. Dangers of hyperhydration includes pulmonary edema, cardiac failure, rebleeding and dilutional coagulopathy.

**Conclusion:**

Fluid management in severely injured patients is a complicated issue and there are no high-ranked randomized, controlled trials that can help to classify the situation. Prehospital fluid replacement must not delay the patient's transfer to hospital. Once, hemorrhage is controlled, restoration of normovolemia is a priority. Initially, the precise fluid used is probably not important, as long as appropriate volume is given. Anemia is much better tolerated than hypovolemia. Future efforts to improve outcomes from haemorrhagic shock will focus on more rapid diagnosis and control of bleeding (as with recombinant Factor VIIa or various topical haemostatic agents), better monitoring of the shock state allowing more precise limitation of fluid administration during active. In the meanwhile, informed resuscitation from shock in both the early and late phases will help improve outcomes and save lives.

--------------