# احیای آب و الکترولیت و اسیدوز در تروما

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- **Shock Classification and Initial Fluid Resuscitation**

Classic signs and symptoms of shock are :

Tachycardia, hypotension, tachypnea, altered mental status, diaphoresis, and pallor.

In general, the quantity of acute blood loss correlates with physiologic abnormalities.

The goal of fluid resuscitation is to re-establish tissue perfusion.

Fluid resuscitation usually begins with isotonic crystalloid, typically Ringer's lactate.

**Table 7-4**

## Signs and symptoms of advancing stages of hemorrhagic shock

| | CLASS I | CLASS II | CLASS III | CLASS IV |
|---|---|---|---|---|
| Blood loss (mL) | Up to 750 | 750–1500 | 1500–2000 | >2000 |
| Blood loss (% BV) | Up to 15% | 15%–30% | 30%–40% | >40% |
| Pulse rate | <100 | >100 | >120 | >140 |
| Blood pressure | Normal | Normal | Decreased | Decreased |
| Pulse pressure (mmHg) | Normal or increased | Decreased | Decreased | Decreased |
| Respiratory rate | 14–20 | >20–30 | 30–40 | >35 |
| Urine output (mL/h) | >30 | >20–30 | 5–15 | Negligible |
| CNS/mental status | Slightly anxious | Mildly anxious | Anxious and confused | Confused and lethargic |

BV = blood volume; CNS = central nervous system.

- However, for patients arriving in shock (persistent SBP <90 mmHg in an adult), instead of crystalloid the current practice is to activate a massive transfusion protocol (MTP) in which red blood cells (RBC) and fresh frozen plasma (FFP) are administered.

- Patients who have a good response to fluid infusion (i.e., normalization of vital signs, clearing of the sensorium) and evidence of good peripheral perfusion (warm extremities with normal capillary refill) are presumed to have adequate overall perfusion.

- Urine output is a reliable indicator of organ perfusion but requires time to quantitate.

- Adequate urine output is 0.5 mL/kg per hour in an adult, 1 mL/kg per hour in a child, and 2 mL/kg per hour in an infant <1 year of age.

- Because measurement of this resuscitation-related variable is time dependent, it is generally more useful in the OR and intensive care unit (ICU) setting, than in initial evaluation in the trauma bay.

- There are several caveats to be considered when evaluating the injured patient for shock.
- Tachycardia (HR >110 bpm) is often the earliest sign of ongoing blood loss, but the critical issue is change in HR over time.
- Individuals in good physical condition with a resting pulse rate in the 50s may manifest a relative tachycardia in the 90s; although clinically significant, this does not meet the standard definition of tachycardia.

- Conversely, patients on cardiac medications such as β-blockers may not be capable of increasing their heart rate to compensate for hypovolemia.

- Bradycardia can occur with rapid severe blood loss; this is an ominous sign, often heralding impending cardiovascular collapse.

- Other physiologic stresses, aside from hypovolemia, may produce tachycardia, such as hypoxia, pain,anxiety, and stimulant drugs (cocaine, amphetamines).

- As noted previously, decreased SBP is not a reliable early sign of hypovolemia because blood loss must exceed 30% before hypotension is evident.

- Additionally, younger patients may maintain their SBP due to sympathetic tone despite severe intravascular deficits until they are on the verge of cardiac arrest.

- Pregnant patients have a progressive increase in circulating blood volume over gestation; therefore, they must lose a relatively larger volume of blood before manifesting signs and symptoms of hypovolemia .

- Based on the initial response to fluid resuscitation, hypovolemic injured patients can be separated into three broad categories: responders, transient responders, and nonresponders.

- Individuals who are stable or have a good response to initial fluid therapy as evidenced by normalization of vital signs, mental status, and urine output are unlikely to have significant ongoing hemorrhage, and further diagnostic evaluation for occult injuries can proceed in an orderly .

- At the other end of the spectrum are patients classified as “nonresponders” who have persistent hypotension despite aggressive resuscitation. These patients mandate immediate identification of the source of hypotension with appropriate intervention to prevent a fatal outcome.

- Transient responders are those who respond initially to volume loading with improvement in vital signs, but subsequently deteriorate hemodynamically.

# Transfusion Practices

- Injured patients with life-threatening hemorrhage develop acute coagulopathy of trauma.

- The mechanism for inadequate clot formation remains uncertain, but it is believed to involve activation of protein C, which impairs Va and VIIa, glycocalyx breakdown, which releases heparin sulfate, immune activation with the releases of DAMPs, DNA, histone, polyphosphate, and PMN elastase, and complement activation.

- Fibrinolysis is an important component of the ACOT; hyperfibrinolysis and fibrinolysis shutdown are both associated with increased mortality.

- Rather, its component parts, packed red blood cells (PRBCs), fresh frozen plasma, platelets, and cryoprecipitate, are administered.

- Specific transfusion triggers for individual blood components remain debated. Although current critical care guidelines indicate that :

- PRBC transfusion should occur once the patient's hemoglobin level is <7 g/dL,64 in the acute phase of resuscitation a hemoglobin of 10 g/dL is suggested to facilitate hemostasis via platelet margination.

- The traditional thresholds for blood component replacement in the patient manifesting a coagulopathy have been INR >1.5, PTT >1.5 normal,platelet count >50,000/µL, and fibrinogen >100 mg/dl.

- However,these guidelines have been replaced by TEG and ROTEM criteria in many trauma centers.
- Such guidelines are designed to limit the transfusion of immunologically active blood components and decrease the risk of transfusion-associated lung injury and secondary multiple organ failure.

- Because complete typing and cross-matching takes up to 45 minutes, patients requiring emergent transfusions are given type O-negative RBCs.

- Similarly, without time for blood typing, AB plasma is the universal donor, although A plasma appears to be a safe option.

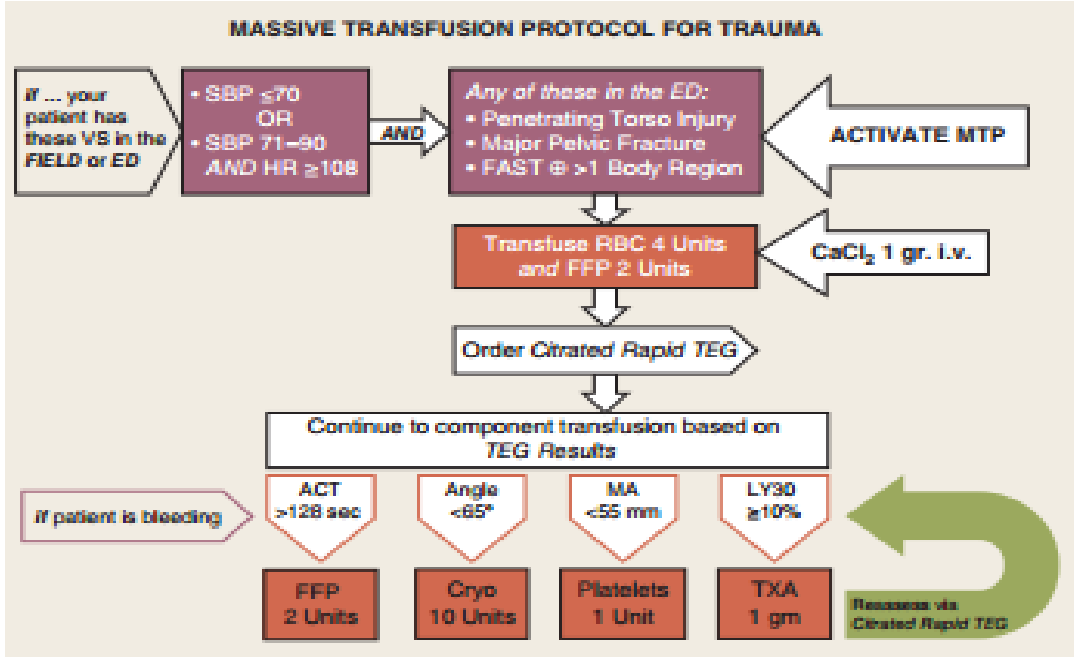
- Postinjury coagulopathy due to shock is aggravated by core hypothermia and metabolic acidosis, termed the *bloody vicious cycle*, and now commonly referred to as the lethal triad.

- The pathophysiology is multifactorial and includes inhibition of temperature-dependent enzyme-activated coagulation cascades, platelet dysfunction, endothelial abnormalities, and fibrinolytic activity.

- Such coagulopathy may be insidious, so the surgeon must be cognizant of subtle signs such as excessive bleeding from the cut edges of skin.

Point-of-care viscoelastic assays (TEG and ROTEM), which provide a comprehensive assessment of clot capacity and fibrinolysis, can provide useful information within 15 minutes.

In contrast, traditional laboratory tests of coagulation capability (i.e., INR, PTT, fibrinogen levels, and platelet count) requires at least 45 minutes.




**Massive Transfusion Protocol**

Triggers: SBP <70 with penetrating torso injury, major pelvic injury, FAST +
SBP <71-90 mmHg and HR >108 with penetrating torso injury, major pelvic injury, FAST +
** order citrated rapid TEG

**Empiric Transfusion Until Lab Results Available**

| Shipment | PRBCs | FFP | Platelets | Cryo |
|---|---|---|---|---|
| 1 | 4 | 2 | | |
| 2 | 4 | 2 | 1 | 10 |

**Continued Treatment of Shock**

Hemorrhage Control, Correct Hypothermia, Correct Acidosis
Normalize $Ca^{++}$

**TEG Based Resuscitation***

rapidTEG-ACT >128 sec → 2 units thawed plasma
rapidTEG-MA <55mm → 1 unit of apheresis platelets
rapidTEG-angle <66 degrees → 10 units pooled cryoprecipitate
rapidTEG EPL >9% → 1g tranexamic acid

## Transfusion Triggers if TEG is Unavailable

PT, PTT > 1.5 control

→2 units thawed plasma

Platelet count <50,000/mcL

→1 unit of apheresis platelets

Fibrinogen <100 mg/dL

→10 units pooled cryoprecipitate