Hemorrhagic Shock management

SHOCK

- transition between life and death

Table 5-1
Classification of shock
Hypovolemic
Cardiogenic
Septic (vasogenic)
Neurogenic
Traumatic
Obstructive

- Shock is defined as a failure to meet the metabolic demands of cells and tissues and the consequences that ensue.

- central component of shock is **decreased tissue perfusion**. This may be a direct consequence of the etiology of shock, such as in hypovolemic/hemorrhagic, cardiogenic, or neurogenic etiologies, or may be secondary to elaborated or released molecules or cellular products that result in endothelial/ cellular activation, such as in septic shock or traumatic shock.

BOX 3.1 Five Categories of Shock According to Primary Treatment of Causes and Problems

Primarily Infusion of Volume

Hemorrhagic shock
- Traumatic
- Gastrointestinal
- Body cavity

Hypovolemia
- Gastrointestinal losses
- Dehydration from insensible losses
- Third-space sequestration from inflammation

Volume Infusion and Vasopressor Support

Septic shock
Anaphylactic shock
Central neurogenic shock
Drug overdose

Improvement in Pump Function by Infusion of Inotropic Support or Reversal of the Cause of Pump Dysfunction

Myocardial ischemia
- Coronary artery thrombosis
- Arterial hypotension with hypoxemia

Cardiomyopathy
- Acute myocarditis
- Chronic diseases of heart muscle (ischemic, diabetic, infiltrative, endocrinologic, congenital)

Cardiac rhythm disturbances
- Atrial fibrillation with rapid ventricular response
- Ventricular tachycardia
- Supraventricular tachycardia

Septic shock with myocardial failure (hypodynamic shock)
Overdose of negative inotropic drug
- Beta blocker
- Calcium channel antagonist

Structural cardiac damage
- Traumatic (e.g., flail mitral valve)
- Ventriculoseptal rupture
- Papillary muscle rupture

Immediate Relief from Obstruction to Cardiac Output

Pulmonary embolism
Cardiac tamponade
Tension pneumothorax
Valvular dysfunction
- Acute thrombosis of prosthetic valve
- Critical aortic stenosis

Congenital heart defects in newborn (e.g., closure of patent ductus arteriosus, with critical aortic coarctation)
Critical idiopathic subaortic stenosis (hypertrophic obstructive cardiomyopathy)

Specific Antidotes Due to Cellular or Mitochondrial Poisons

Carbon monoxide
Methemoglobinemia
Hydrogen sulfide
Cyanide

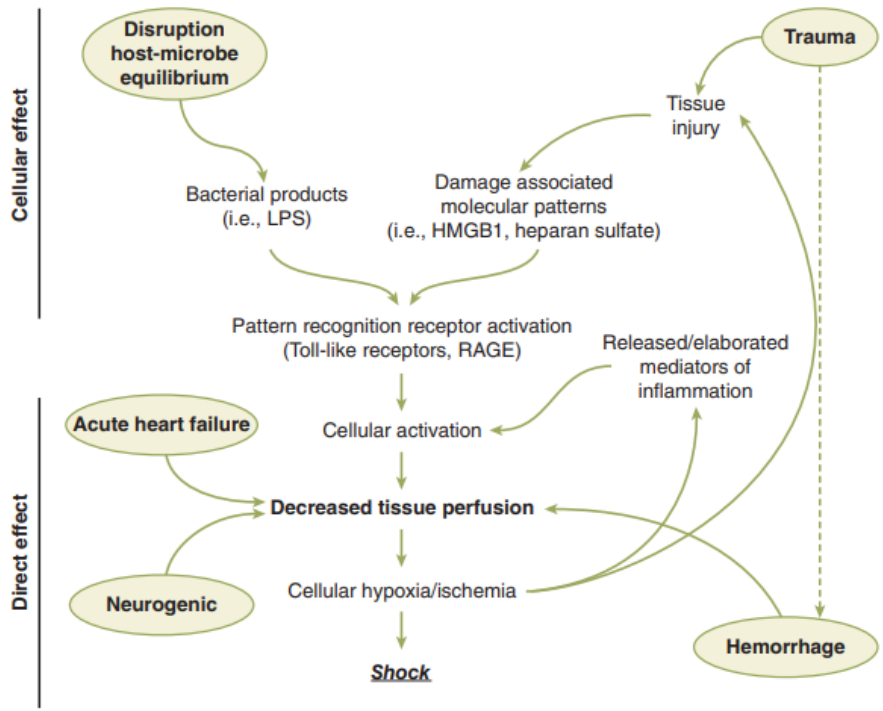


Figure 5-1. Pathways leading to decreased tissue perfusion and shock. Decreased tissue perfusion can result directly from hemorrhage/hypovolemia, cardiac failure, or neurologic injury. Decreased tissue perfusion and cellular injury can then result in immune and inflammatory responses. Alternatively, elaboration of microbial products during infection or release of endogenous cellular products from tissue injury can result in cellular activation to subsequently influence tissue perfusion and the development of shock. HMGB1 = high mobility group box 1; LPS = lipopolysaccharide; RAGE = receptor for advanced glycation end products.

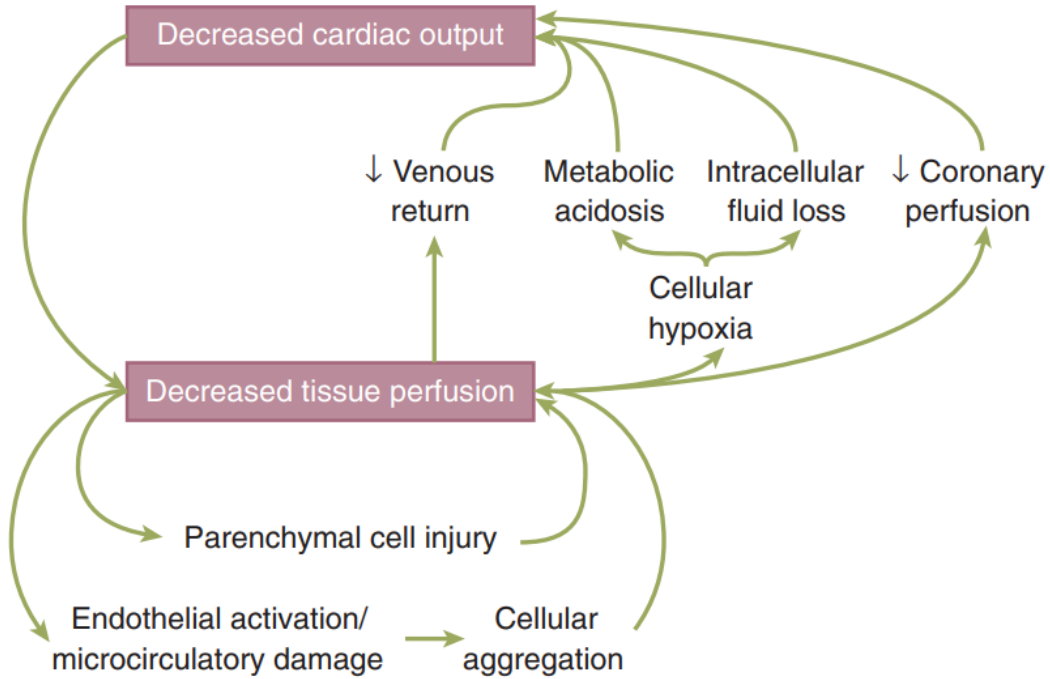


Figure 5-2. The "vicious cycle of shock." Regardless of the etiology, decreased tissue perfusion and shock results in a feed-forward loop that can exacerbate cellular injury and tissue dysfunction.

Table 5-2

Hemodynamic responses to different types of shock

TYPE OF SHOCK	CARDIAC INDEX	SVR	VENOUS CAPACITANCE	CVP/PCWP	Svo_2	CELLULAR/METABOLIC EFFECTS
Hypovolemic	↓	↑	↓	↓	↓	Effect
Septic	↑↑	↓	↑	↑↓	↑↓	Cause
Cardiogenic	↓↓	↑↑	→	↑	↓	Effect
Neurogenic	↑	↓	→	↓	↓	Effect

The hemodynamic responses are indicated by arrows to show an increase (↑), severe increase (↑↑), decrease (↓), severe decrease (↓↓), varied response (↑↓), or little effect →). CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; Svo_2 = mixed venous oxygen saturation; SVR = systemic vascular resistance.

- The emergency clinician identifies shock by linking the qualitative clinical impression, synthesized from the patient's history of present illness, age, health status, and general appearance, to quantitative data, including vital signs, laboratory tests, urine output, and measurements of systemic oxygenation. When the clinical impression and quantitative data suggest widespread organ hypoperfusion, emergent resuscitation is used to restore tissue oxygenation and substrate delivery to prevent deterioration into a vicious cycle of systemic inflammation, organ dysfunction, and death

- hemodynamic parameters such as blood pressure and heart rate are relatively insensitive measures of shock, and additional considerations must be used to help aid in early diagnosis and treatment of patients in shock. The general approach to the management of patients in shock has been empiric: **assuring a secure airway with adequate ventilation**, **control of hemorrhage** in the bleeding patient, and **restoration of vascular volume and tissue perfusion.**

BOX 3.2 Empirical Criteria for Diagnosis of Shock

- Ill appearance or altered mental status
- Heart rate >100 beats/min
- Respiratory rate >20 breaths/min or $Paco_2$ <32 mm Hg
- Arterial base deficit <–4 mEq/L or lactate level >4 mM/L
- Urine output <0.5 mL/kg/h
- Arterial hypotension >30 min duration, continuous

Regardless of cause, majority should be met in patients with shock.

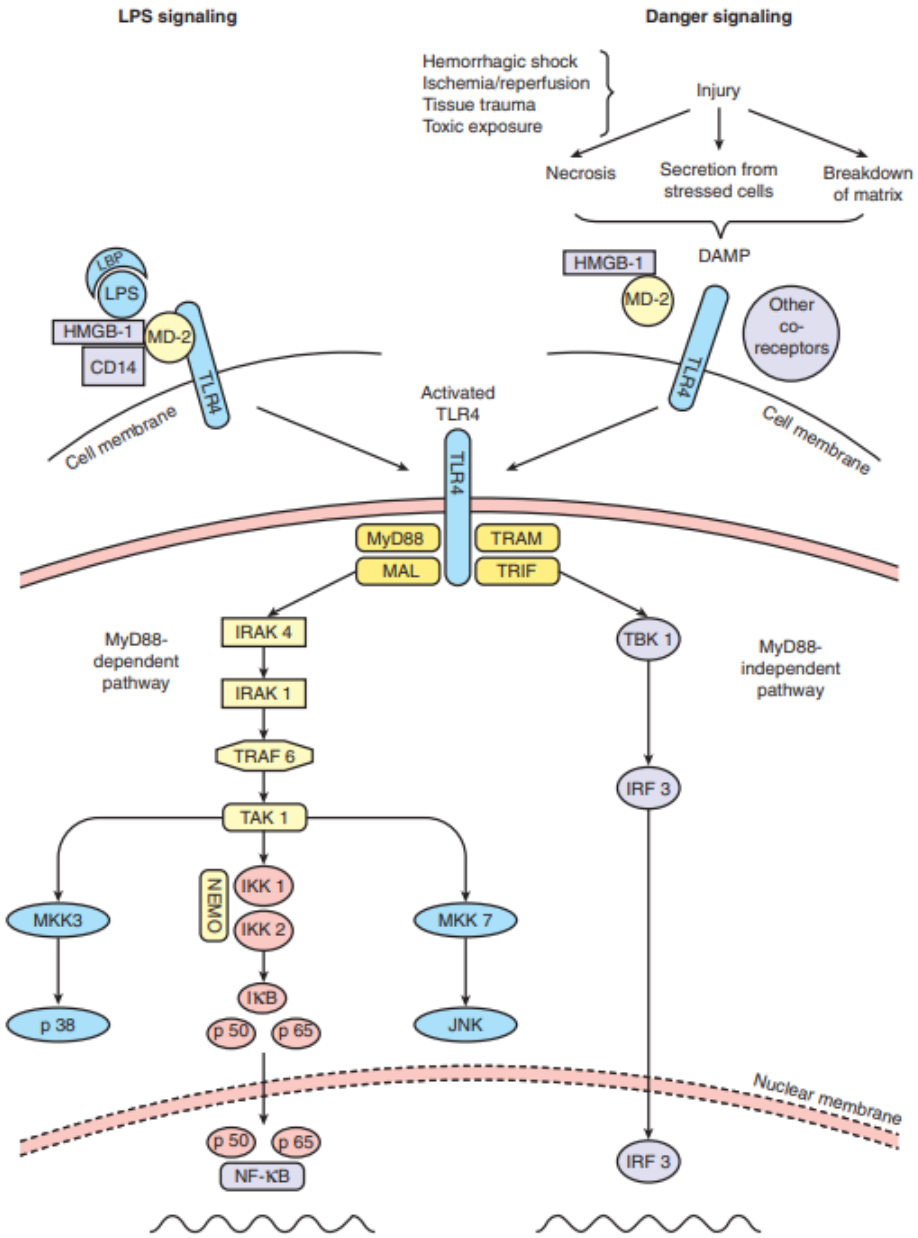
BOX 3.4 Variables Indicating Tissue Hypoperfusion

Hypotension
Tachycardia
Low cardiac output
Dusky or mottled skin
Delayed capillary refill
Altered mental state
Low urine output
Low central venous oxygen saturation
Elevated lactate level

In ***traumatic shock***, soft tissue and bony injury

lead to the activation of inflammatory cells and the release of circulating factors, such as cytokines and intracellular molecules that modulate the immune response. Recent investigations have revealed that the inflammatory mediators released in response to

tissue injury (damage-associated molecular patterns [DAMPs]) are recognized by many of the same cellular receptors (pattern recognition receptors [PRRs]) and activate similar signaling pathways as do bacterial products elaborated in sepsis(pathogenassociated molecular patterns), such as lipopolysaccharide.These effects of tissue injury are combined with the effects of hemorrhage, creating a more complex and amplified deviation from homeostasis.



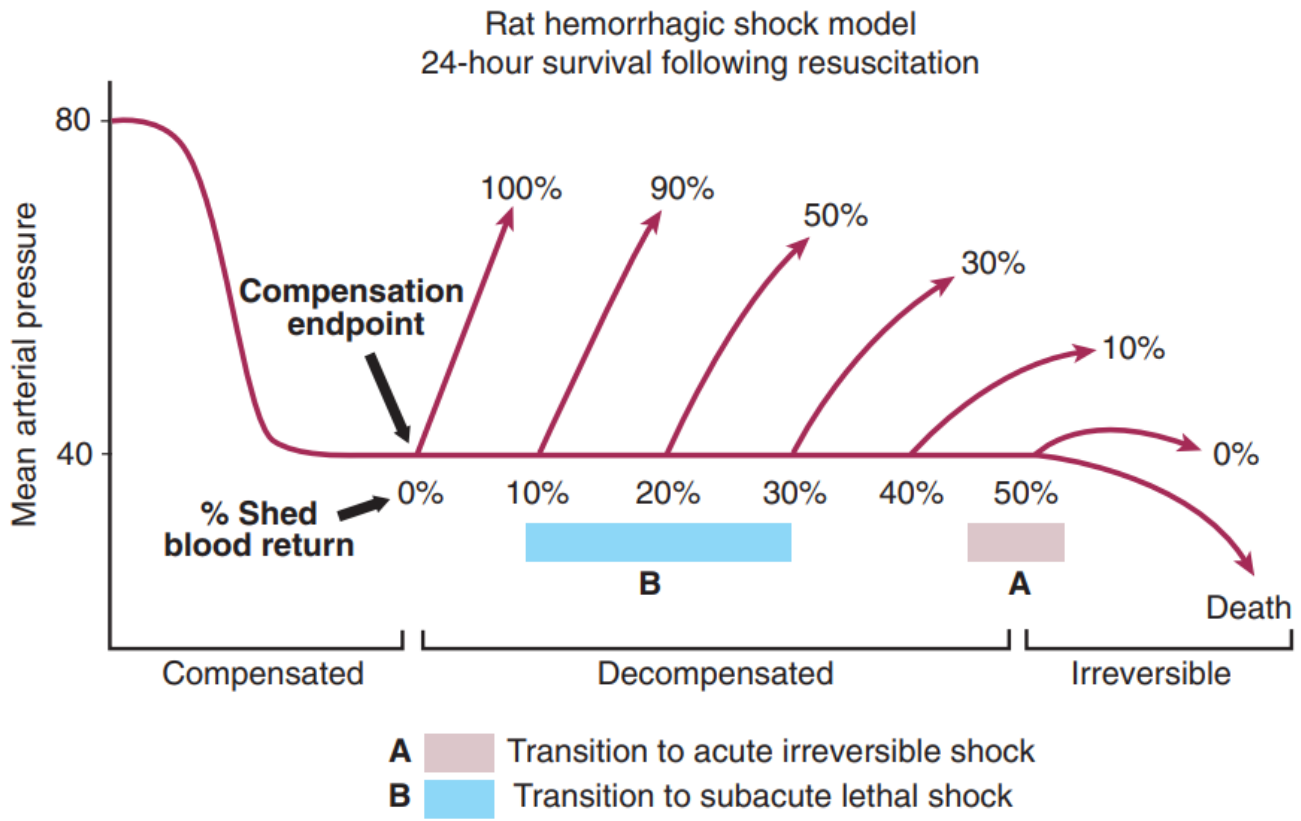


Figure 5-3. Rat model of hemorrhagic shock through the phases of compensation, decompensation, and irreversibility. The percentages shown above the curve represent survival rates. *(Reproduced with permission from Shah NS, Kelly E, Billiar TR, et al. Utility of clinical parameters of tissue oxygenation in a quantitative model of irreversible hemorrhagic shock*, Shock. *1998 Nov;10(5):343-346.)*

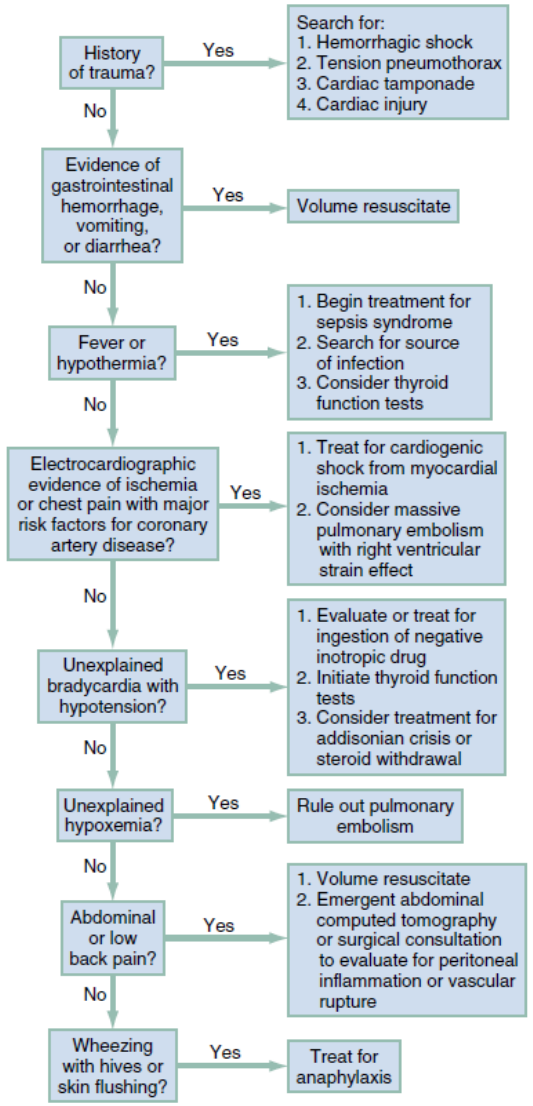


Fig. 3.1 Flow Diagram to Classify Undifferentiated Shock.

BOX 3.3 Definitions and Criteria for Septic, Hemorrhagic, and Cardiogenic Shock

Septic Shock

Sepsis—Suspected or confirmed infection with a new or increased Sequential Organ Failure Assessment (SOFA) score of 2 from baseline (see Table 3.1)

Septic shock—Sepsis plus hypotension requiring vasopressors after fluid loading plus lactate >2 mmol/L (Patients requiring vasopressors with a normal lactate should still be treated as having shock)

Hemorrhagic Shock

Simple Hemorrhage

Suspected bleeding with pulse rate <100 beats/min, normal respiratory rate, normal blood pressure, and normal base deficit

Hemorrhage with Hypoperfusion

Suspected bleeding with base deficit <–4 mEq/L *or* persistent pulse rate >100 beats/min

Hemorrhagic Shock

Suspected bleeding, with at least four criteria listed in Box 3.2

Cardiogenic Shock

Cardiac Failure

Clinical evidence of impaired forward flow of the heart, including presence of dyspnea, tachycardia, pulmonary edema, peripheral edema, and/or cyanosis

Cardiogenic Shock

Cardiac failure plus four criteria listed in Box 3.2

Hemorrhagic shock/PATHOPHYSIOLOGY

❑ The most common cause of shock in the surgical or trauma patient

- decreased baroreceptor stimulation from stretch receptors in the large arteries --->
- decreased inhibition of vasoconstrictor centers in the brain stem increased chemoreceptor stimulation of vasomotor centers
- diminished output from atrial stretch receptor

- Hypovolemia also induces sympathetic stimulation, leading to epinephrine and norepinephrine release, activation of the renin-angiotensin cascade, and increased vasopressin release

➢ Peripheral vasoconstriction is prominent while lack of sympathetic effects on cerebral and coronary vessels and local autoregulation promote maintenance of cardiac and CNS blood flow.

The clinical and physiologic response to hemorrhage has been classified:

Table 5-5

Classification of hemorrhage

PARAMETER	CLASS I	CLASS II	CLASS III	CLASS IV
Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic	Hypotension	Severe hypotension
CNS symptoms	Normal	Anxious	Confused	Obtunded

bpm = beats per minute; CNS = central nervous system.

Young vs Elderly patients?

Hemorrhagic shock/DIAGNOSIS

- The clinical signs of shock may be evidenced by **agitation**, **cool clammy extremities**, **tachycardia**, **weak or absent peripheral pulses**, and **hypotension**. Such apparent clinical shock results from at least 25% to 30% loss of the blood volume.

- substantial volumes of blood may be lost before the classic clinical manifestations of shock are evident. Thus, when a patient is significantly tachycardic or hypotensive, this represents both significant blood loss and physiologic decompensation.

In addressing the sensitivity of vital signs and identifying major thoracoabdominal hemorrhage, a study retrospectively identified patients with injury to the trunk and an abbreviated injury score of 3 or greater who required immediate surgical intervention and transfusion of at least 5 units of blood within the first 24 hours:

- 95% percent of patients had a heart rate greater than 80 bpm at some point during their post injury course.
- **only 59% of patients achieved a heart rate greater than 120 bpm.**
- 99% percent of all patients had a recorded blood pressure of less than 120 mmHg at some point.
- 93% percent of all patients had a recorded SBP of less than 100 mmHg.

- A more recent study corroborated that **tachycardia was not a reliable sign of hemorrhage following trauma** and was present in only 65% of hypotensive patients.

- Several studies have demonstrated that the initial serum lactate and serial lactate levels are reliable predictors of morbidity and mortality with hemorrhage following trauma
- Similarly, base deficit values derived from arterial blood gas analysis provide clinicians with an indirect estimation of tissue acidosis from hypoperfusion

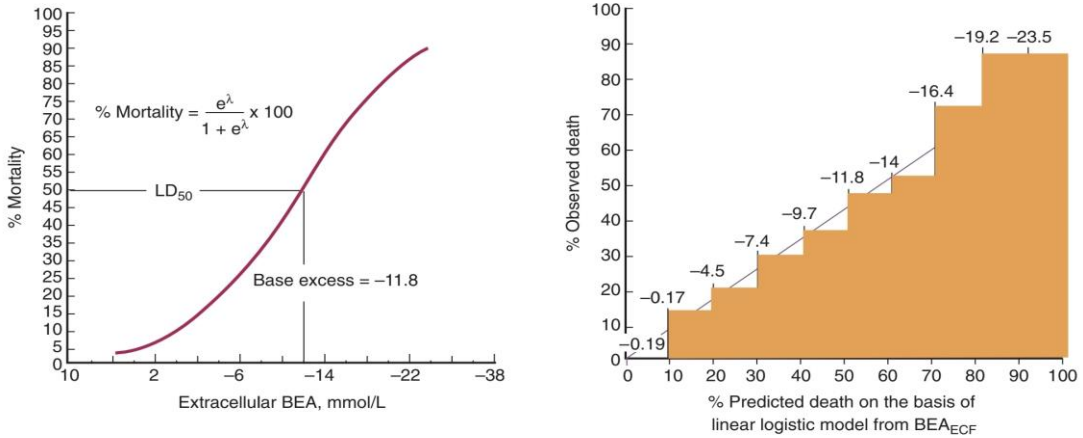


Figure 5-9. The relationship between base deficit (negative base excess) and mortality in trauma patients. BEA = base excess arterial; ECF = extracellular fluid. *(Reproduced with permission from Siegel JH, Rivkind AI, Dalal S, et al: Early physiologic predictors of injury severity and death in blunt multiple trauma,* Arch Surg. *1990 Apr;125(4):498-508.)*

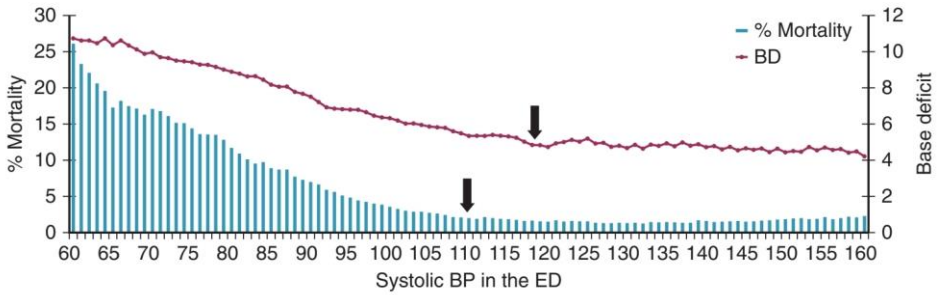


Figure 5-7. The relationship between systolic blood pressure and mortality in trauma patients with hemorrhage. These data suggest that a systolic blood pressure of less than 110 mmHg is a clinically relevant definition of hypotension and hypoperfusion based upon an increasing rate of mortality below this pressure. Base deficit (BD) is also shown on this graph. ED = emergency department. *(Reproduced with permission from Eastridge BJ, Salinas J, McManus JG, et al. Hypotension begins at 110 mm Hg: redefining "hypotension" with data,* J Trauma. *2007 Aug;63(2):291-297.)*

- Although hematocrit changes may not rapidly reflect the total volume of blood loss, admission hematocrit has been shown to be associated with 24 hour fluid and transfusion requirements and more strongly associated with PRBC transfusion than either tachycardia, hypotension, or acidosis. It must be noted that lack of a depression in the initial hematocrit does not rule out substantial blood loss or ongoing bleeding.

- In management of trauma patients, understanding the patterns of injury of the patient in shock will help direct the evaluation and management.

1. Identifying the sources of blood loss in patients with penetrating wounds is relatively simple because potential bleeding sources will be located along the known or suspected path of the wounding object.
2. Patients with penetrating injuries who are in shock usually require operative intervention.

3. Patients who suffer multisystem injuries from blunt trauma have multiple sources of potential hemorrhage. Blood loss sufficient to cause shock is generally of a large volume, and there are a limited number of sites that can harbor sufficient extravascular blood volume to induce hypotension (e.g., external, intrathoracic, intra-abdominal, retroperitoneal, and long bone fractures).

4. In the non-trauma patient, the GI tract must always be considered as a site for blood loss.

5. Substantial blood loss externally may be suspected from prehospital medical reports documenting a substantial blood loss at the scene of an accident, history of massive

6. visible brisk bleeding, or presence of a large hematoma adjacent to an open wound. Injuries to major arteries or veins with associated open wounds may cause massive blood loss rapidly. Direct pressure must be applied and sustained to minimize ongoing blood loss.

7. Tourniquets should be used for extremity bleeding stopped by direct pressure and applied in

Hemorrhagic shock/TREATMENT

- Control of ongoing hemorrhage is an essential component of the resuscitation of the patient in shock.

- treatment of hemorrhagic shock is instituted concurrently with diagnostic evaluation to identify a source.

- Patients who fail to respond to initial resuscitative efforts should be assumed to have ongoing active hemorrhage from large vessels and require prompt operative intervention.

- Based on trauma literature, patients with ongoing hemorrhage demonstrate increased survival if the elapsed time between the injury and control of bleeding is decreased.

The appropriate priorities in these patients are as follows:

- **(a) control the source of blood loss,**
- **(b) perform IV volume resuscitation with blood products in the hypotensive patient, and**
- **(c) secure the airway.**

damage control resuscitation

- This strategy begins in the emergency department, continues into the operating room, and into the intensive care unit (ICU). Initial resuscitation is limited to keep SBP around 80 to 90 mmHg. This prevents renewed bleeding from recently clotted vessels. Resuscitation and intravascular volume resuscitation is accomplished with blood products and limited crystalloid.

- Too little volume allowing persistent severe hypotension and hypoperfusion is dangerous, yet too vigorous of a volume resuscitation may be just as deleterious. Control of hemorrhage is achieved in the operating room (or angiography suite once surgical causes of hemorrhage have been ruled out), and efforts to warm patients and to prevent coagulopathy using multiple blood products and pharmacologic agents are used in both the operating room and ICU.

hypotensive resuscitation

- Cannon and colleagues first made the observation that attempts to increase blood pressure in soldiers with uncontrolled sources of hemorrhage is counterproductive, with increased bleeding and higher mortality.
- This work was the foundation for the “hypotensive resuscitation” strategies. Several laboratory studies confirmed the observation that attempts to restore normal blood pressure with fluid infusion or vasopressors was rarely achievable and resulted in more bleeding and higher mortality. A prospective, randomized clinical study compared delayed fluid resuscitation (upon arrival in the operating room) with standard fluid resuscitation (with arrival by the paramedics) in hypotensive patients with penetrating torso injury. The authors reported that delayed fluid resuscitation resulted in lower patient mortality.
- Further laboratory studies demonstrated that fluid restriction in the setting of profound hypotension resulted in early deaths from severe hypoperfusion. These studies also showed that aggressive crystalloid resuscitation attempting to normalize blood pressure resulted in marked hemodilution, with hematocrits of 5%.

❑ Reasonable conclusions in the setting of uncontrolled hemorrhage include

- any delay in surgery for control of hemorrhage increases mortality;
- with uncontrolled hemorrhage, attempting to achieve normal blood pressure may increase mortality, particularly with penetrating injuries and short transport times;
- a goal of SBP of 80 to 90 mmHg may be adequate in the patient with penetrating injury;
- profound hemodilution should be avoided by early transfusion of red blood cells.
- For the patient with blunt injury, where the major cause of death is a closed head injury, the increase in mortality with hypotension in the setting of brain injury must be avoided. In this setting, a SBP of 110 mmHg would seem to be more appropriate.

- **Volume Replacement**

1. ***Crystalloids***
2. ***Colloids and hypertonic saline.***
3. ***Blood products***

- **Vasopressors**

- ***Coagulation factor***

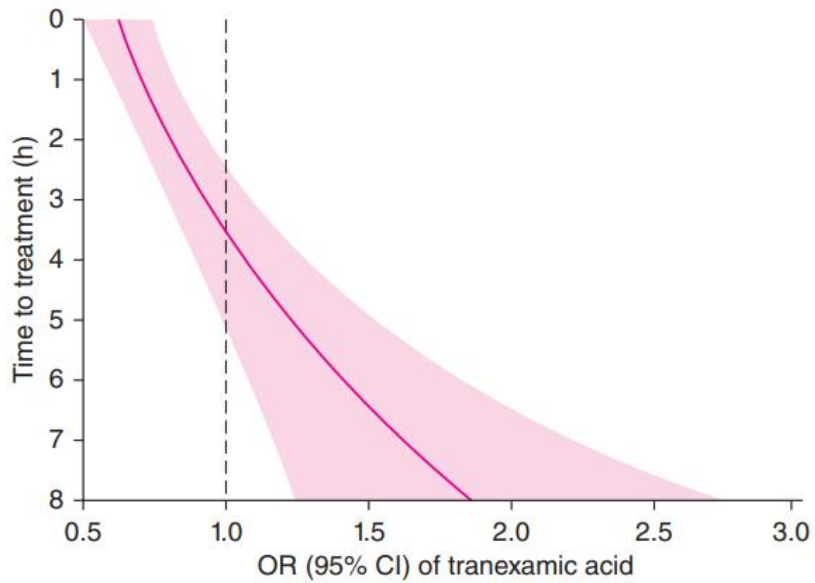


Figure 5-10. Early treatment (within 3 hours) of trauma patients with tranexamic acid reduces mortality. However, later treatment exacerbated outcome. *(Reproduced with permission from Roberts I, Shakur H, Afolabi A, et al. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial,* Lancet. *2011 Mar 26;377(9771):1096-1101.)*

BOX 3.5 Clinical Management Guidelines for Three Common Causes of Shock

Hemorrhagic Shock

- Ensure adequate ventilation and oxygenation.
- Provide immediate control of hemorrhage, when possible (e.g., traction for long bone fractures, direct pressure, REBOA), and obtain urgent consultation as indicated for uncontrollable hemorrhage.
- Initiate judicious infusion of isotonic crystalloid solution (10–20 mL/kg).
- With evidence of poor organ perfusion and 30-min anticipated delay to hemorrhage control, begin packed red blood cell (PRBC) infusion (5–10 mL/kg).
- With suspected massive hemorrhage, immediate PRBC transfusion may be preferable as the initial resuscitation fluid, with balanced transfusions of PRBCs, fresh frozen plasma, and platelets.
- Treat coincident dysrhythmias.

Cardiogenic Shock

- Ameliorate increased work of breathing; provide oxygen and positive end-expiratory pressure (PEEP) for pulmonary edema.
- Begin vasopressor or inotropic support; norepinephrine (0.5 mcg/min) and dobutamine (5 mcg/kg/min) are common empirical agents.
- Seek to reverse the insult (e.g., thrombolysis, percutaneous transluminal angioplasty).
- Consider intraaortic balloon pump counterpulsation for refractory shock.

Septic Shock

- Ensure adequate oxygenation; remove work of breathing.
- Administer 30 mL of crystalloid/kg and titrate infusion based on dynamic indices, volume responsiveness, and/or urine output.
- Begin antimicrobial therapy; attempt surgical drainage or debridement.
- Begin PRBC infusion for hemoglobin level <7 g/dL.
- If volume restoration fails to improve organ perfusion, begin vasopressor support with norepinephrine, infused at 0.5 mcg/min.

PRBCs, Packed red blood cells; *REBOA,* resuscitative endovascular balloon occlusion of the aorta.

وجود شواهد شوک هموراژیک

- افت فشار خون سیستولی به کمتر از ۹۰ میلیمتر جیوه
- شواهد بالینی وضعیت شوک نظیر نبض ضعیف، تغییر سطح هوشیاری، پوست سرد و ...
- با رعایت Permissive Hypotension

If TBI suspected: SBP> 110 mmHg, MAP> 85 mmHg

If TBI is non-suspected: SBP= 80 - 90 mmHg, MAP: 50 - 65 mmHg

خط اول درمان:

۱. تزریق کریستالوئیدهای داخل وریدی با ارجحیت رینگر لاکتات و نرمال سالین بعنوان گزینه جایگزین.
۲. در صورت وجود شواهد خونریزی شدید خارجی در سه ساعت اول بعد آسیب تزریق وریدی ترانگزامیک اسید (TXA) مد نظر قرار گیرد

1- Lactated Ringer 500 cc IV bolus up to 2 lits (peds: 10 cc/kg IV STAT up to 30 cc/kg)

2- TXA 1gr IV infusion over 10 mins then 1gr IV infusion over 8 hrs, (peds: 15 mg/kg STAT and 2 mg/kg/hr IV infusion over 8 hrs.)

خط دوم درمان:

در صورت عدم پاسخ به درمانهای خط اول
۱. شروع ترانسفیوژن خون کامل (Fresh Whole Blood) و یا پک سل (PRBCs) بدون تایپ و کراس مچ اقدام کنید (ترجیحا گروه خونی O بدون توجه به آنتی ژنRh ؛ در خانمهای باردار با Rh منفی خون Rh منفی توصیه می گردد).
۲. در صورتیکه بیمار بیش از ۲ واحد خون در این مرحله نیاز دارد به شروع اجرای پروتکل Massive Transfusion فکر کنید.

1- Transfusion of (up to) 4 bags (Peds: 20 cc/ kg) whole blood or PRBCs (group O, Rh +\-)

Non cross-matched

خط سوم درمان:

ضمن در نظر داشتن سایر علل شوک، اگر در یک ساعت اول پاسخ مناسب به درمان شوک هموراژیک ایجاد نشد

۱. شروع پروتکل Massive Transfusion: تزریق فرآورده های خونی را بصورت ۱:۱:۱
۲. برای عوارض ناشی از ترانسفیوژن شدید اقدامات لازم نظیر تزریق کلسیم، حمایت تنفسی، جلوگیری از اسیدوز و هایپوترمی، پایش فاکتورهای انعقادی و سایر اختلالات مربوطه اقدامات لازم را انجام دهید.

initiate Massive Transfusion Protocol

1- transfusion of 1 bag PRBCs and 1 bag FFP then 1 bag platelet typed and cross-matched

2- Calcium Gluconate 10% 10 cc IV STAT over 10 mins

3- check CBC, BUN, Cr, Na, K, Ca, PTT, PT, INR, serum Fibrinogen Level, ABG

KEY CONCEPTS

- Shock can occur with normal arterial blood pressure and not all patients with arterial hypotension have shock
- A base deficit more negative than −4 mEq/L or a serum lactate level greater than 4.0 mmol/L warrants a presumptive diagnosis of shock. Urine output is a reliable index of vital organ perfusion in patients with suspected shock. Normal urine output is 1.0 mL/kg/h. Output less than 0.5 mL/ kg/h indicates severe renal hypoperfusion in patients without preexisting disease
- A combination of a worsening **base deficit**, increasing **lactate level**, and low **urine output** represents persistent or worsening shock.

- Prevention of **hypothermia**, **acidemia**, and **coagulopathy** are essential in the management of patients in hemorrhagic shock.

- Prevention of hypothermia, acidemia, and coagulopathy are essential in the management of patients in hemorrhagic shock.