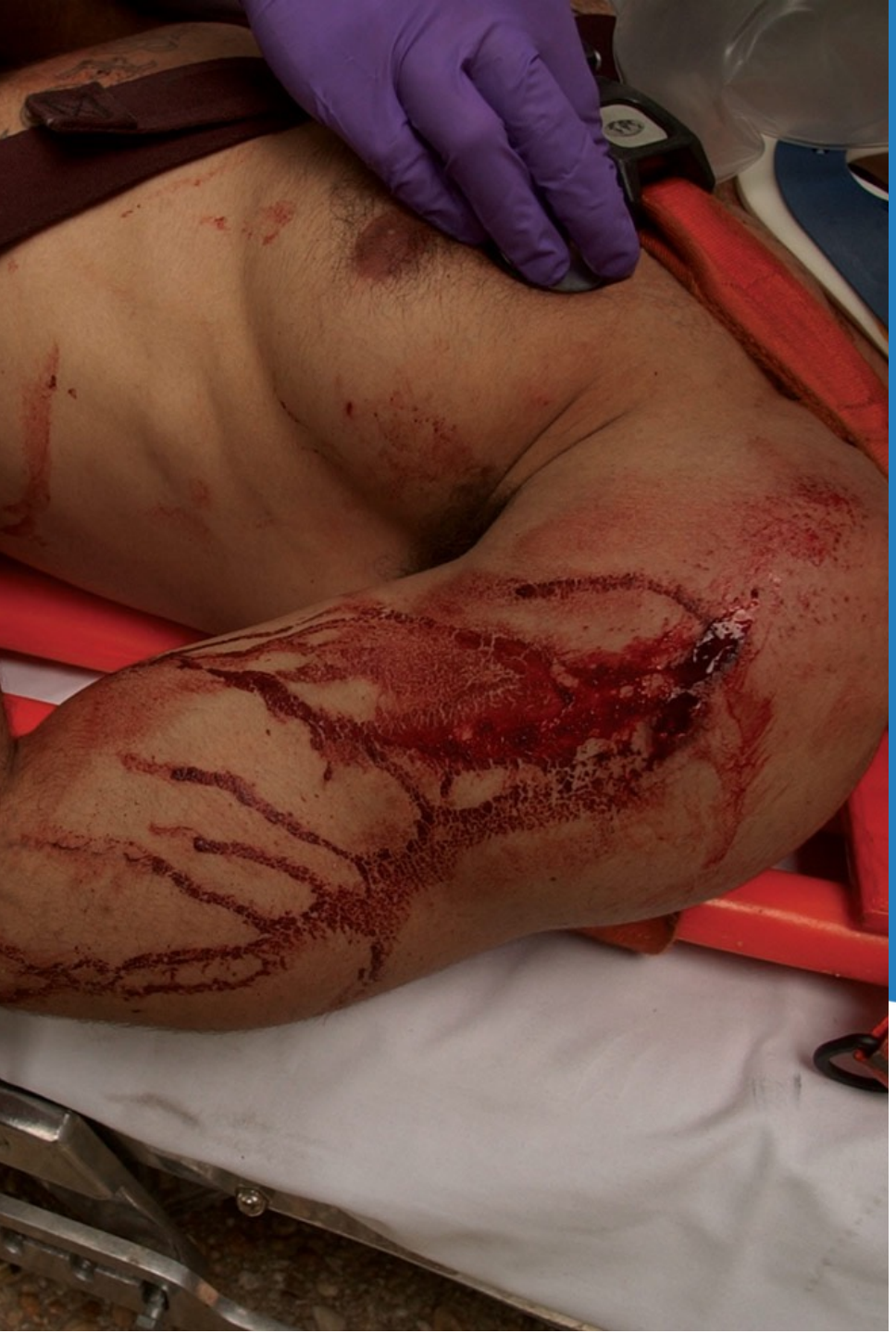

Shock

Management and Resuscitation of the Critical Patient

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National EMS Education Standard Competencies

Shock and Resuscitation

Integrates a comprehensive knowledge of the causes and pathophysiology into the management of shock, respiratory failure or arrest with an emphasis on early intervention to prevent arrest.

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Introduction

When working with a critical patient:

- Conduct a rapid assessment

- Provide life saving treatment

- Develop a differential field diagnosis

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Introduction

If a patient is in critical condition, you must be well prepared to:

- Make the right decision
- Use time appropriately
- Provide care

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Critical Patients

While caring for critical patients you will come across:

- Premorbid conditions
- Major trauma
- Patients in the peri-arrest period

Table 1 Adult Premorbid Conditions Directly Affecting EMS

Condition	Healthy Adult	Unhealthy Adult
Congestive heart failure	Unlikely	X
Coronary Artery Disease	Unlikely	X
Drug toxicity	X	X
Electrolyte imbalance	X	X
Obesity	Unlikely	X
Pulmonary embolus	X	X
Renal failure	Unlikely	X
Stroke	Unlikely	X
Uncontrolled hypertension	Unlikely	X
Uncontrolled diabetes	Unlikely	X

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The EMS Approach to Diagnosis

Follow a standard approach when determining a field diagnosis

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Shock: The Critical Patient Evolving in Front of You

Shock: state of collapse and failure of the cardiovascular system

- Leads to insufficient perfusion of organs/tissues

- Normal compensatory mechanism

- Untreated shock will lead to death

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Anatomy and Physiology of Perfusion

Perfusion: circulation of blood in adequate amounts to meet the cells' needs

- Requires working cardiovascular system

- Requires adequate gas exchange, glucose, and waste removal

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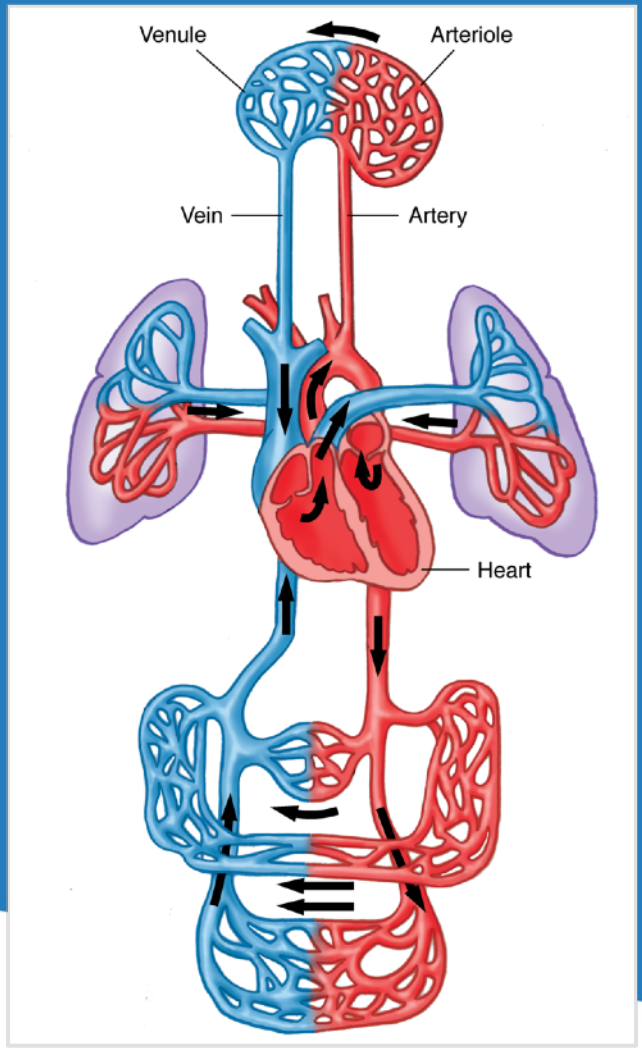
Anatomy and Physiology of Perfusion

Cardiovascular system requires three components:

1) Functioning pump

2) Adequate fluid

3) Intact system of tubing



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Anatomy and Physiology of Perfusion

The heart's contractility allows it to increase or decrease the volume of blood pumped

Cardiac output (CO): volume of blood that the heart can pump per minute

- Heart must have adequate strength
- Heart must receive adequate blood

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Anatomy and Physiology of Perfusion

* **Blood pressure is generated by:**

  - Contractions of the heart

  - Dilation and constriction of blood vessels

* **Blood pressure varies directly with cardiac output, systemic vascular resistance, and blood volume**

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Anatomy and Physiology of Perfusion

* **Cardiac output = Heart rate × Stroke volume**

* **Blood pressure = Cardiac output × systemic vascular resistance**

* **Mean arterial pressure: blood pressure.**

MAP = DBP + 1/3 (SBP – DBP)

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Anatomy and Physiology of Perfusion

The body is perfused via the cardiovascular system, which is controlled by the autonomic nervous system

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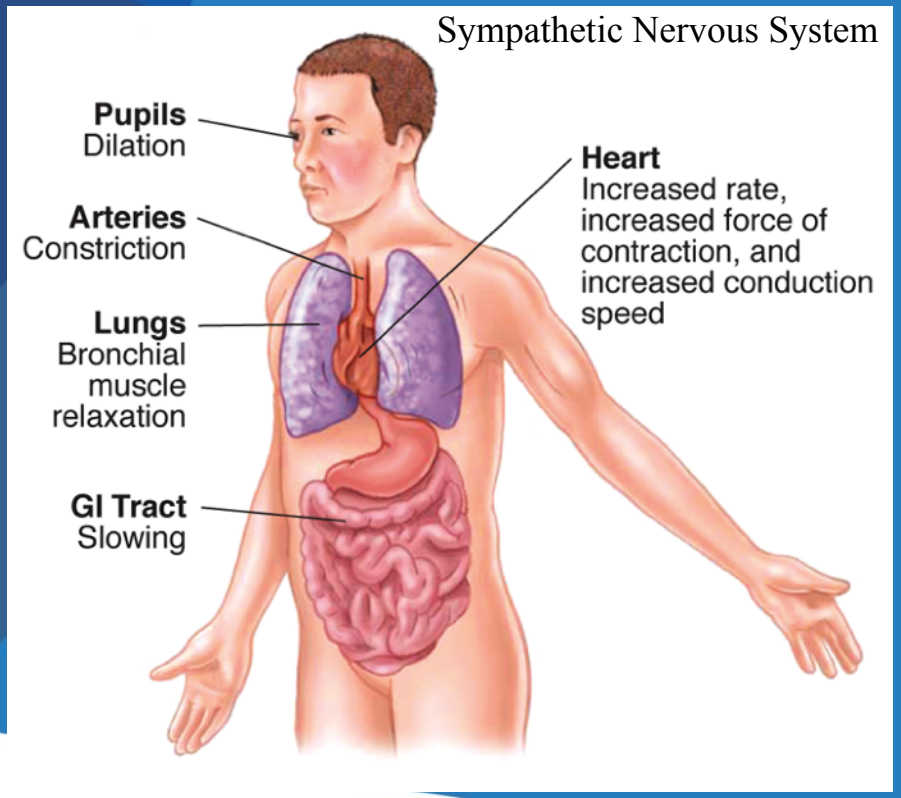
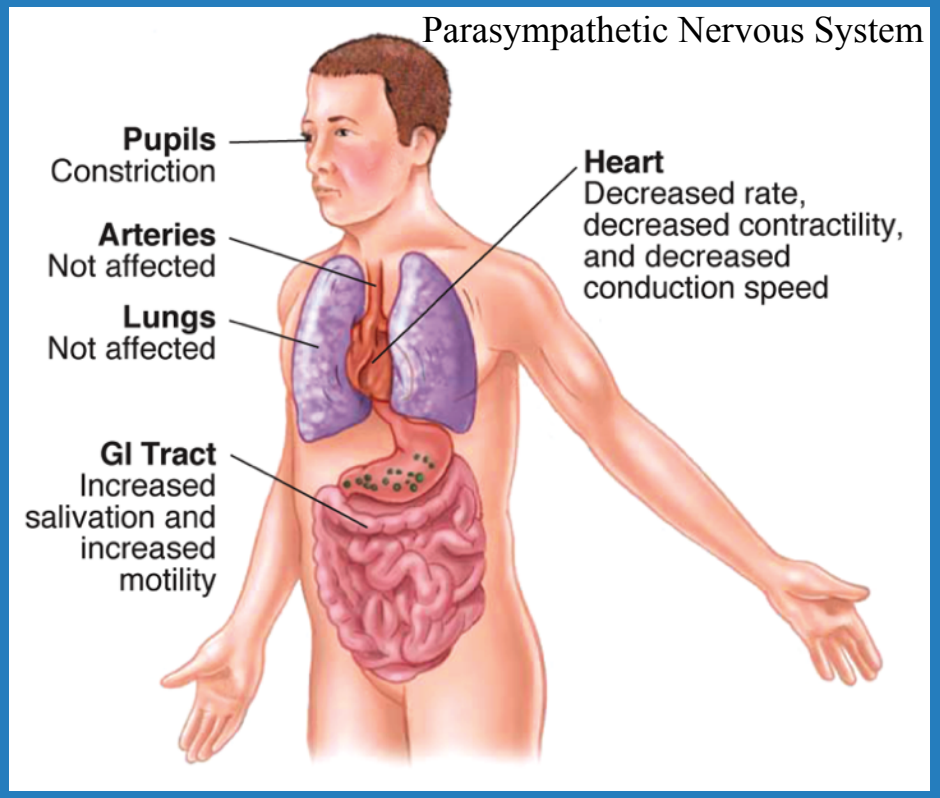
Anatomy and Physiology of Perfusion

Sympathetic Nervous System



Parasympathetic Nervous System



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Respiration and Oxygenation

*** Alveoli receive oxygen-rich air from each breath**

*** Oxygen and carbon dioxide pass across tissue layers through the process of diffusion**

Molecules move from an area of higher concentration to an area of lower concentration

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Respiration and Oxygenation

Carbon dioxide is dissolved in plasma and attaches to the blood's hemoglobin

- Combines with water to create carbonic acid
  * Breaks down at the lungs and carbon dioxide is exhaled

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Regulation of Blood Flow

Blood flow through capillary beds is regulated by the capillary sphincters

- Under control of the autonomic nervous system
- Regulation is determined by cellular need

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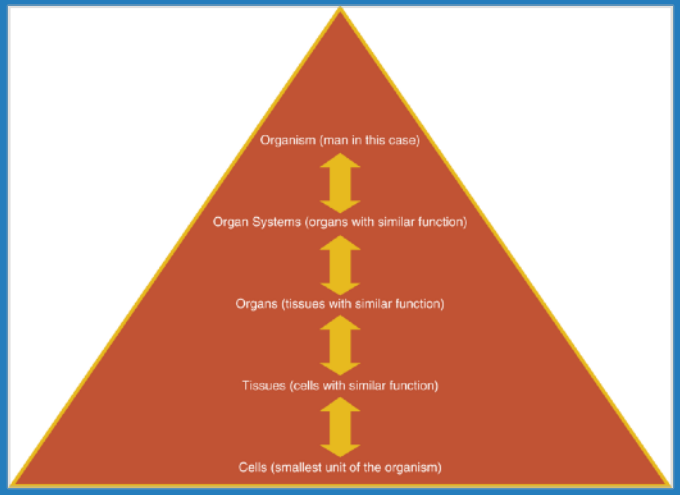
Pathophysiology of Shock

*** Shock results from:**

- Inadequate CO (Cardiac Output)
- Decreased SVR (Systemic Vascular Resistance)
- Inability of RBCs to deliver oxygen



*** The body shunts blood flow to vital organs**

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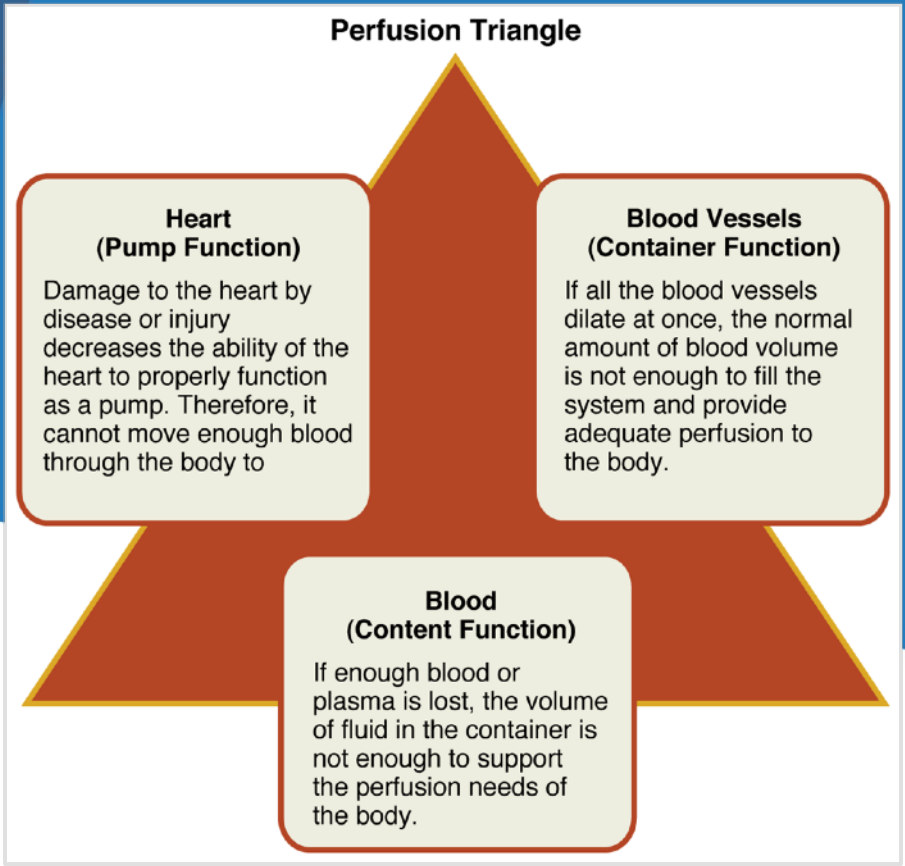
Pathophysiology of Shock

The cardiovascular system consists of the "perfusion triangle."

Shock means that one part is not working properly



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Pathophysiology of Shock

*** Blood carries oxygen and nutrients through vessels to the capillary beds to tissue cells**

*** Blood clots control blood loss**

Form depending on:

- *Retention of blood because of blockage*
- *Changes in a vessel wall*
- *Blood's ability to clot*

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Pathophysiology of Shock

When pressure is failing, neural and hormonal mechanisms are triggered

- Epinephrine and norepinephrine causes changes in pulse rate, cardiac contractions, and vasoconstriction
- Body fluids shift to maintain pressure

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Compensation for Decreased Perfusion

The body responds to any event that leads to decreased profusion

- Baroreceptors activate vasomotor center to begin constriction of the vessels
- Chemoreceptors measure shifts in carbon dioxide in the arterial blood

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Compensation for Decreased Perfusion

Stimulation normally occurs when the systolic pressure is between 60–80 mm Hg

- Drop in pressure causes baroreceptor stimulation to decrease
- Sympathetic nervous system is stimulated
- The renin-angiotensin-aldosterone system is activated and antidiuretic hormone is released

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Compensation for Decreased Perfusion

* The overall response is to increase preload, stroke volume, and pulse rate

* Myocardial oxygen demand increases if hypoperfusion persists

Cells switch to anaerobic metabolism

$$C_6H_{12}O_6 + 6O_2 \xrightarrow[\text{many steps}]{\text{aerobic respiration}} 6CO_2 + 6H_2O + 38\,ATP$$

glucose (stored energy) + oxygen → carbon dioxide + water + (usable energy)

$$C_6H_{12}O_6 \xrightarrow[\text{fewer steps}]{\text{anaerobic respiration}} 2\,\text{Lactic Acids} + 2ATP$$

glucose (stored energy) → 2 Lactic Acids + (usable energy)

(in humans)

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Compensation for Decreased Perfusion

Table 5 Effects of Epinephrine and Norepinephrine

Epinephrine	
Alpha-1	Vasoconstriction Increase in peripheral vascular resistance Increased afterload from arteriolar constriction
Alpha-2	Inhibit insulin release Relax gastrointestinal smooth muscle
Beta-1	Positive **chronotropic effects** (increase in the heart's rate of contraction) Positive **inotropic effects** (increase in the contractility of the heart muscle) Positive **dromotropic effects** (increase in the heart's velocity of conduction)
Beta-2	Bronchodilation Gastrointestinal smooth muscle dilation
Norepinephrine	
Alpha-1 and alpha-2	Vasoconstriction Increase in peripheral vascular resistance Increased afterload from arteriolar constriction

The release of epinephrine and norepinephrine improves CO and increases SVR.

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Compensation for Decreased Perfusion

Failure to preserve perfusion leads to decreases in preload and cardiac output

- Myocardial blood supply decreases
- Coronary artery perfusion decreases
- Liver and pancreas functions are impacted
- Gastrointestinal motility is decreased
- Urine production decreases

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Shock-Related Events at the Capillary and Microcirculatory Levels

Decreased perfusion leads to cellular ischemia

- The body can tolerate anaerobic metabolism for only a short time
  - Leads to systemic acidosis
  - Ischemia stimulates increased carbon dioxide

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Shock-Related Events at the Capillary and Microcirculatory Levels

Sodium-potassium pump normally sends sodium back out against the concentration gradient

- Reduced ATP results in dysfunctional pump
  * Excessive sodium diffuses into the cells.

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Shock-Related Events at the Capillary and Microcirculatory Levels

Intracellular enzymes are usually bound in an impermeable membrane

- Cellular flooding explodes the membrane.
  * Leads to last phase of shock
  * Decreases venous return and diminishes blood flow.

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Shock-Related Events at the Capillary and Microcirculatory Levels

*** Reduced blood supply results in slowing of sympathetic nervous system activity**

*** The buildup of lactic acid and carbon dioxide acts as potent vasodilators.**

- Accumulation washes into the venous circulation

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Shock-Related Events at the Capillary and Microcirculatory Levels

White blood cells and blood clotting systems are impaired

- May lead to:
  * Decreased resistance to infection
  * Disseminated intravascular coagulation (DIC)

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Multiple-Organ Dysfunction Syndrome

Progressive condition characterized by failure of two or more organs that were initially unharmed

- Each tissue has its own warm ischemic time.
- Patients have a mortality rate of 60-90%
- Classified as primary or secondary

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Multiple-Organ Dysfunction Syndrome

Occurs when injury or infection triggers a massive systemic response

- Results in the release of inflammatory mediators and activation of the:

* Complement system
* Coagulation system
* Kallikren-kinnin system

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Multiple-Organ Dysfunction Syndrome

Overactivity results in a maldistribution of systemic and organ blood flow

- Body accelerates tissue metabolism
- Progression causes organs to malfunction

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Multiple-Organ Dysfunction Syndrome

Typically develops within hours or days after resuscitation

Affects specific organs and organ systems:

- Heart
- Lungs
- Central nervous system
- Kidneys
- Liver
- GI tract

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Causes of Shock

Normal tissue perfusion requires an intact heart, fluid volume, and tubing

- Damage to any one disrupts tissue perfusion
- Shock results from many conditions
- Have a high index of suspicion in emergency medical situations

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Causes of Shock

A **Pump failure**
Causes: Heart attack, trauma to heart, obstructive causes

B **Low fluid volume**
Causes: Trauma to vessels or tissues, fluid loss from GI tract (vomiting/diarrhea can also lower the fluid component of blood)

C **Poor vessel function**
Causes: Infection, drug overdose (narcotic), spinal cord injury, anaphylaxis

*** Three basic causes of shock:**

- Pump failure
- Low fluid volume
- Poor vessel function

*** Certain patients are more at risk**

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The Progression of Shock

Shock occurs in three phases: compensated, decompensated, and irreversible

- Also called four grades of hemorrhage or four classes of shock

American College of Surgeons Classes of Acute Hemorrhage

Factors	I	II	III	IV
Blood loss	<15% (<750ml)	15-30% (750-1500ml)	30-40% (1500-2000ml)	>40% (>2000ml)
Pulse	>100	>100	>120	>140
B.P.	Normal	Normal	↓	↓↓
Pulse pressure	N or ↓	↓	↓↓	↓↓
Capillary refill	<2s	2-3s	3-4s	>5s
Resp. rate	14-20	20-30	30-40	>40
Urine output ml/hr	30 or more	20-30	5-10	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious & confused	Confused Lethargic

* Class I and II = compensated shock
* Class III = decompensated shock
* Class IV = irreversible shock

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The Progression of Shock

* Recognize signs and symptoms early on
* Begin immediate treatment before damage occurs

Table 6 Compensated Versus Decompensated Shock

Compensated Shock	Decompensated Shock
▪ Agitation, anxiety, restlessness ▪ Sense of impending doom ▪ Weak, rapid (thready) pulse ▪ Clammy (cool, moist) skin ▪ Pallor with cyanotic lips ▪ Shortness of breath ▪ Nausea, vomiting ▪ Delayed capillary refill in infants and children ▪ Thirst ▪ Normal blood pressure	▪ Altered mental status (verbal to unresponsive)* ▪ Hypotension ▪ Labored or irregular breathing ▪ Thready or absent peripheral pulses ▪ Ashen, mottled, or cyanotic skin ▪ Dilated pupils ▪ Diminished urine output (oliguria) ▪ Impending cardiac arrest

*Mental status changes are late indicators.

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Compensated Shock

* **Earliest stage of shock**
  - The body can still compensate for blood loss
* **Level of responsiveness is the best indication of tissue perfusion**
* **Blood pressure is maintained**

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Decompensated Shock

* **Blood volume drops more than 30%**
* **Compensatory mechanisms begin to fail**
  - Signs and symptoms become obvious
* **Sometimes treatment will result in recovery**

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Decompensated Shock

Once blood pressure drop is detected, shock is well developed

- Consider an emergency and start transport to closest appropriate facility as quickly as possible

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Irreversible (Terminal) Shock

*** Last phase of shock**

*** Life-threatening reductions in cardiac output, blood pressure, tissue perfusion**

Cells begin to die and vital organ damage cannot be repaired.

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Scene Size-Up

* Size up the scene for hazards
* Follow standard precautions
* Determine the number of patients and the need for additional resources
* Quickly assess the MOI or nature of illness.

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Primary Assessment

Form a general impression

- How does the patient look?
- Assess mental status using AVPU
- Introduce yourself and ask their name, location and day of the week

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Primary Assessment

Airway and breathing

- If you suspect cardiac arrest, use CAB approach
  * Otherwise, asses the ABCs
- Manage immediate threats
- Assess airway patency and examine the chest
- Assess the adequacy of the patient's ventilatory status

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Primary Assessment

Circulation

- Take CAB approach if you suspect the patient does not have a pulse
    * In patients with a pulse, determine if it is adequate
- In conscious patients, assess the radial pulse
    * In unconscious patients, check the carotid pulse

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Primary Assessment

Circulation (cont'd)

- If you know the patient is hypotensive, provide immediate transport to the ED

- Also note the patient's skin color, temperature, and condition

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Primary Assessment

Transport decision

- All patients need to be prioritized

  * If shock is from a medical problem, fast-track to an assessment based on body systems

  * If shock is from trauma, let the MOI guide your assessment

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History-Taking

Can be done en route to the ED in a high-priority patient

- Unless patient is pinned, and you suspect a delay in extrication, delay establishing IV/IO access until you are en route

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Secondary Assessment

Drop in systolic blood pressure or altered mental status indicates the body can no longer compensate

- Other indicators include end-tidal carbon dioxide (lowered in hypoperfused states) and lactic acid buildup

Courtesy of EKF Diagnostics www.ekfdiagnostics.com

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Reassessment

*** Re-visit the primary assessment, vital signs, chief complaint, and treatment performed**

*** Determine what interventions are needed**

- Patients in decompensated shock will need rapid intervention

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Special Considerations for Assessing Shock

Healthy, fit, young adults are equipped to combat life-threatening blood loss

- Resilient cardiovascular system
- Not smoking increases oxygenation

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Pediatric Considerations

*** Pediatric patients can compensate until a 30–35% blood loss**

*** Ability to compensate relies on increasing heart rate and systemic vascular resistance**

*** Lesser glucose stores lead to hypoglycemia**

*** Treat aggressively and early with significant MOI or indication of shock**

- Never wait to see a drop in blood pressure.

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Geriatric Considerations

* **Ability to manage blood loss is diminished**
* **Manage fluid therapy carefully**
* **Thermoregulation is less effective**
* **Cardiovascular disorders or diabetes affect ability to compensate**
* **Medications may prevent clot formations**

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Emergency Medical Care of a Patient With Suspected Shock

Airway and ventilatory support take priority when treating a patient with shock

- Maintain an open airway and suction as needed
- Administer high-flow supplemental oxygen
- Consider early, definitive airway management if not maintained

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Emergency Medical Care of a Patient With Suspected Shock

* Control any external hemorrhage and try to estimate the amount of blood lost

* Look for signs of internal hemorrhage

* Goal of volume replacement is to maintain perfusion without increasing internal or uncontrollable external hemorrhage

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Emergency Medical Care of a Patient With Suspected Shock

*** If signs of tension pneumothorax, perform the needle chest decompression**

***With suspected cardiac tamponade, recognize the need for pericardiocentesis**

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Emergency Medical Care of a Patient With Suspected Shock

Non-pharmacologic interventions include:

- Proper positioning of the patient
- Prevention of hypothermia
- Rapid transport

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IV Therapy

IV lines (2 large bore or IO w/pressure bag) are inserted to provide:

- Immediate replacement of fluids in patient who have already lost significant volumes of fluid or blood
- Potential replacement in patients who are at risk of losing significant volumes of fluid or blood
- Provide route for administration of medication

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IV Therapy

*** IV lines should be inserted to keep a vein open for emergency administration of drugs**

- Patients who need a vein kept open include:
  - * Those at risk of cardiac arrest
  - * Those needing parenteral medication

***IV flow rate is determined by local protocol, presumptive diagnosis, and condition of patient's lungs**

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Crystalloids

* Solutions that do not contain proteins or other large molecules
* Rapidly equilibrates into tissues
* Fluids of choice when only salt and water have been lost

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Crystalloids

***Commonly used crystalloids are:**

- Normal saline
- Lactated Ringer's solution

*** Either fluid will benefit the patient**

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EMERGENCY MEDICAL RESPONDER/EMT

- Routine Medical -or- Trauma Care
- Secure and maintain airway
- Perform non-visualized advanced airway, if indicated
- Administer oxygen per non-rebreather mask at 15 LPM
- Control external hemorrhage
- Keep patient flat with lower extremities elevated (if possible)
- Conserve body temperature, and reassure patient

AEMT

- IV/IO 0.9% NS through large bore catheter
- Initiate IV/IO wide open to maintain SBP>100, recheck lung sounds frequently
- Adult fluid bolus: 30ml/kg, for max of 3L
- Pediatric fluid bolus: 20ml/kg up to 3 times, for max of 60ml/kg
- If trauma patient see *Routine Trauma Care*

PARAMEDIC

- Consider RSA see *Respiratory Distress Guidelines*
- **Push Dose 1:100,000 Epinephrine per section 5.42** titrated to maintain a SBP > 90 mmHg
- If hemorrhagic shock refer to *2.42 Routine Trauma Care* for TXA indications and dosing

This page updated 3/22/18

Pathophysiology, Assessment, and Management of Specific Types of Shock

Classifications of shock:

1) Cardiogenic

2) Obstructive

3) Distributive

4) Hypovolemic

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Pathophysiology, Assessment, and Management of Specific Types of Shock

Initial management of shock:

1) Manage the airway

2) Administer supplemental oxygen

3) Put the patient in a position of comfort

4) Obtain vital signs, SpO2, lung sounds, and a 12-lead EKG

5) Obtain IV access

6) Maintain body heat

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Cardiogenic Shock

* Occurs when the heart cannot circulate sufficient blood to maintain adequate peripheral oxygen delivery

* Most commonly caused by an AMI accompanied by dysfunction of left ventricle (40% loss)

* Also caused by right ventricular failure, valvular disorders, cardiomyopathies, ventricular septal defects, papillary muscle rupture, myocardial insufficiency, and sustained dysrhythmias

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Cardiogenic Shock

Populations at the greatest risk include:

- Elderly
- Patients with a history of diabetes mellitus
- Patients with a history of AMI with an ejection fraction of less than 35%

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Cardiogenic Shock

Prolonged efforts to stabilize the patient are not recommended

- Expedite transport as quickly as possible
- Secure the airway and administer oxygen (Consider CPAP/BiPaP)
- Obtain a 12-lead ECG
- Administer crystalloid solution
- Pressors (push dose epinephrine and drip)
- Auscultate the lungs.

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Obstructive Shock

*** Causes are not directly associated with loss of fluid, pump failure, or vessel dilation**

*** Occurs when blood flow in the heart or great vessels becomes blocked**

*** Tension pneumothorax, cardiac tamponade, pulmonary embolism**

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Obstructive Shock

Tension pneumothorax

- Caused by damage to the lung tissue
- Air accumulates within the chest cavity and applies pressure to the mediastinum, kinking vena cava
    * Life-threatening condition
- Tachycardia, hypotension, decreased lung sounds, JVD, tracheal deviation
- Treatment is needle decompression and chest tube

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Obstructive Shock

Cardiac tamponade

- Caused by blunt or penetrating trauma, tumors, or pericarditis
- Occurs when blood leaks into the pericardium and compresses the heart
- Tachycardia, hypotension, narrow pulse pressure, muffled heart sounds, JVD, electrical alternans on EKG
- Treatment is pericardiocentesis

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Distributive Shock

* Occurs when there is widespread dilation of the resistance vessels (small arterioles), the capacitance vessels (small venules), or both
* Circulating blood volume pools in vascular beds
* Septic, Neurogenic, Anaphylactic, and Psychogenic

Distributive Shock

Septic shock

* Occurs from a widespread infection
  - Activation of inflammatory-immune response
  - Increased microvascular permeability, vasodilation, third-space fluid shifts, and micro thrombi formation
  - An uncontrolled response results in hypoperfusion to cells
  - Untreated, the result multiple-organ dysfunction and death

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Distributive Shock

Septic shock (cont'd)

1) Insufficient volume of fluid in the container due to leakage

2) Fluid leaks out and collects in the respiratory system, affecting ventilation

3) Larger-than-normal vascular bed must contain the smaller-than-normal volume of fluid

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Distributive Shock

Septic shock (cont'd)

* Presents similarly to hemorrhagic shock
  - Weak, thready pulse; shallow, rapid respirations; AMS
  - Usually have warm or hot skin
* Ventilatory support, IV fluids, Pressors
* Complex hospital management with antibiotics

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Distributive Shock

Neurogenic shock

* Usually results from spinal cord injury
* Results in loss of normal sympathetic nervous system tone and vasodilation
* Perfusion of organs and tissues becomes inadequate, even though no blood or fluid has been lost
* Hypotension, bradycardia, skin is pink, warm, and dry, absence of sweating below the level of the injury

Distributive Shock

Neurogenic shock (cont'd)

* Spinal shock: occurs after a spinal injury produces motor and sensory losses (may not be permanent)

- Commonly at the upper cervical levels
- Injury to autonomic nervous system
- Characterized by flaccid paralysis, flaccid sphincters, absent reflexes, absence of pain, temperature, touch, proprioception, and pressure below the level of the injury; impaired thermoregulation, bowel distention, and loss of peristalsis
- Level of injury is related to severity of shock

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Distributive Shock

Neurogenic shock (cont'd)

* Care is similar to general management
  - Patient should be immobilized and kept warm
  - Determine the necessity for IV fluids
  - Pressors if needed

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Distributive Shock

Anaphylactic shock

- Occurs when a person reacts violently to a substance to which he or she has been sensitized (to develop a heightened reaction)

- Patient experiences widespread vascular dilation (normal blood volume in larger container)

- Combination of poor oxygenation and poor perfusion may prove fatal

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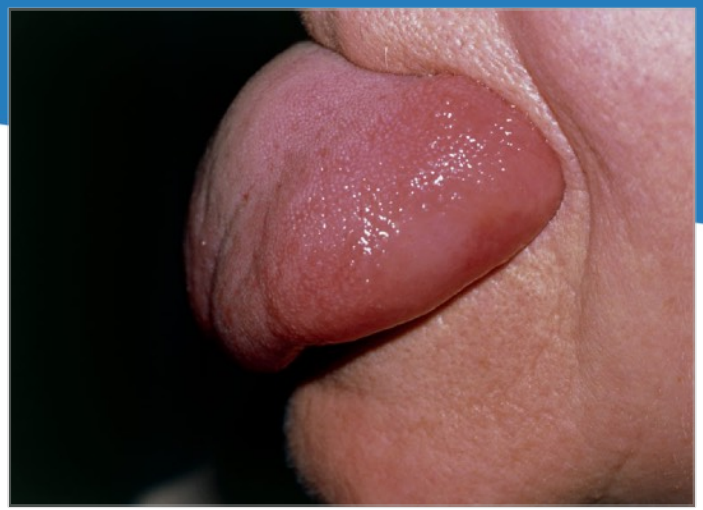
Distributive Shock

Anaphylactic shock (cont'd)

- Histamines and other vasodilator proteins are released, which causes severe bronchoconstriction and wheezing

- Urticaria

- Widespread vasodilation and blood vessel leakage, leading to hypovolemia and significant swelling, which may occlude the upper airway



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Distributive Shock

Anaphylactic shock (cont'd)

- Management needs to occur quickly
  - Remove the inciting cause and resolve life threats to the ABCs
  - Evaluate the patient's ventilatory status
  - Provide cardiovascular support with IV fluids and/or pressors
  - Bronchodilator
  - Anti-histamine
  - Steroid
  - Epinephrine

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Distributive Shock

Psychogenic shock

- Sudden reaction of the nervous system that produces a temporary vascular dilation, resulting in syncope

- Blood pools in the dilated vessels, reducing the blood supply to the brain, ceasing normal function

- Life-threatening causes include:

    * Irregular heartbeat

    * Brain aneurysm

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Distributive Shock

Psychogenic shock (cont'd)

- If the patient has fallen, check for injuries
- Assess the patient for any other abnormalities
- Record your initial observations
- Obtain an ECG

Hypovolemic Shock

* Occurs because of inadequate blood volume
* Hemorrhagic, non-hemorrhagic, and third-space causes
* Hemorrhagic = internal and external sources of blood loss
* Non-hemorrhage = vomiting, diarrhea, sweating
* Third-space losses = fractures and burns

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Hypovolemic Shock

Hemorrhagic Shock

- Major life threat to most trauma patients is blood loss
- Hemorrhage is most prevalent due to blunt or penetrating injuries to vessels or organs, long bone or pelvic fractures, major vascular injuries, and multi system injury
- Can be internal or external

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Hypovolemic Shock

Hemorrhagic Shock (cont'd)

- Key is to recognize the clinical signs and symptoms of shock in its earliest phase and begin immediate treatment before permanent damage occurs

- Clinical suspicion based on MOI and patient assessment

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Hypovolemic Shock

Hemorrhagic Shock (cont'd)

- 2 large bore IVs
- IVF with goal to maintain systolic blood pressure between 80-90 mmHg systolic and MAP around 65 mmHg (permissive hypotension)
- Pelvic binder
- Wound packing/dressing/hemostatic agents
- Splints
- Tourniquet application
- TXA
- Pressors?

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Hypovolemic Shock

Non-hemorragic Shock

- Occurs when fluid loss is contained within the body, as in dehydration, burn injury, crush injury, and anaphylaxis

  * GI losses, especially through vomiting and diarrhea

  * Loss as a consequence of fever, hyperventilation, or high environmental temperature

  * Increased and excessive sweating

  * “Third-spacing” losses

  * Plasma losses from burns, drains, and granulating wounds

/

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Hypovolemic Shock

Non-hemorragic Shock (Cont'd)

- Signs and symptoms

  * Restlessness and anxiety

  * Thirst

  * Skin is pale, cold, clammy, and mottled; poor turgor

  * Rapid, weak pulse, rising more than 15 beats/min when raised from recumbent to sitting

  * Furrowed tongue and sunken eyes

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Hypovolemic Shock

Non-hemorragic Shock (Cont'd)

- Testing and Treatment
  * Labs
  * EKG
  * ABCs
  * IVF
  * Anti-emetic
  * Thermal regulation

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EVENT MEDICINE
EDUCATION PLANNING OPERATIONS

SIGNS & SYMPTOMS OF SHOCK

	RR	HR	BP	SKIN	TEMP	URINE	OTHER S&S
ANAPHYLACTIC Severe allergic reaction.	↑↓	↑	↓	Flushed Swollen Itchy	NO CHANGE	↓	Urticaria, Pruritus, Decreased LOC, Bronchoconstriction
CARDIOGENIC Failing pumping ability of the heart.	↑	↑	↓	Pale Cool Clammy	NO CHANGE	↓	Chest Discomfort, Syncope, JVD, Pulmonary Edema, Orthopnea
HYPOVOLEMIC Reduced circulating blood volume.	↑	↑	↓	Pale Cool Clammy	NO CHANGE	↓	Anxiety, Thirst, Syncope, Weakness, Confusion, Dizziness, Syncope, Weak Pulse
OBSTRUCTIVE Physical obstruction of great vessels or the heart.	↑	↑	↓	Extremities: Pale Cool	↓	↓	Muffled Heart Sounds, JVD, Decreased LOC, Signs of Poor Perfusion
NEUROGENIC Severe central nervous system damage.	↑	↓	↓	Warm Flushed Dry	OR	No Bladder Control	Paralysis Distal to Injury Site, Priaprism
SEPTIC Extreme immune system response to an infection.	↑	↑	↓	Flushed then Pale & Cool	≥38°C OR <36°C	↑	Bounding Pulse, Altered LOC

		Pre-load PCWP JVP	Pump Fn CO	After-load SVR	Perfusion O2 Sat	Tx
Hypovolemic	- Intravascular vol loss - hemorrhagic - fluid loss	↓	↓	↑	↓	Fluids
Cardiogenic	- Arrhythmia - AMI, valve failure - cardiomyopathy - pericarditis/PE	↑	↓	↑	↓	Dobutamine (5-20mcg/kg/min)
Distributive	Vasodilatory-↓↓ SVR -septic shock/SIRS/TSS - Anaphylaxis - neurogenic shock - Drug/toxin - Addisonian crisis	↓/-	↑	↓	-/↑	Norepi (neurogenic, septic) Epi (anaphylaxis) Phenyl (neurogenic) Dopamine
Obstructive	- Tension PTX - Tamponade - PE	↑	↓	-/↑	-/↓	Thoracostomy, pericardiocentesis

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5.42 Epinephrine Push Dose and Drip

Epinephrine has alpha and beta effects so it is an inopressor.
Do not give cardiac arrest doses (1mg) to patients with a pulse.
Do not utilize as first line treatment for shock, initiate only after appropriate other resuscitation measures.
Refer to Broselow or pediatric reference for pediatric blood pressure ranges.
BP must be monitored frequently, no less than every 5 minutes.

- Assemble a 1:10,000 epinephrine syringe and place a double female luer lock adaptor on end
- Use an empty 20 mL syringe to draw up 2ml of the 1:10,000 epinephrine
- With same 20ml syringe, draw up 18ml of normal saline to dilute the epinephrine
- The concentration of epinephrine in the 20mL saline flush syringe is now 1:100,000, or 10 mcg/mL
- Label the syringe so it is not inadvertently given
- Administer as necessary to maintain blood pressure

Onset: 1 minute

Duration: 5-10 minutes

Goal: Maintain mean arterial pressure(MAP)>65

Dose:

Mild hypotension SBP<80

1-2ml (10-20mcg or .01-.02mg) every 2-5 minutes

Moderate hypotension SBP<70

3-5ml (30-50mcg or .03-.05mg) every 2-5 minutes

Severe hypotension(cardiovascular collapse)

6-10ml (100mcg or .06-0.1mg) per minute

In situations where a continuous infusion is needed, an epinephrine drip may be utilized.

- Draw up 1mg of either 1:1,000 or 1:10,000 epinephrine
- Inject into a 1 liter bag of Normal Saline
- The concentration of epinephrine in the 1 liter 1mcg/mL
- Label the bag so it is not inadvertently given
- Use a 10gtt/ml macrodrip tubing set and piggyback into primary infusing fluid
- Administer as necessary to maintain blood pressure

Onset: Immediate

Duration: Continuous while infused

Goal: Maintain mean arterial pressure(MAP)>65

Dose:

Mild hypotension SBP<80

1mcg/min or .001mg/min = 1gtt every 5 seconds

Moderate hypotension SBP<70

20mcg/min or .02mg/min = 4 drops per second

Severe hypotension(cardiovascular collapse)

50mcg/min or .05mg/min = 8 drops per second

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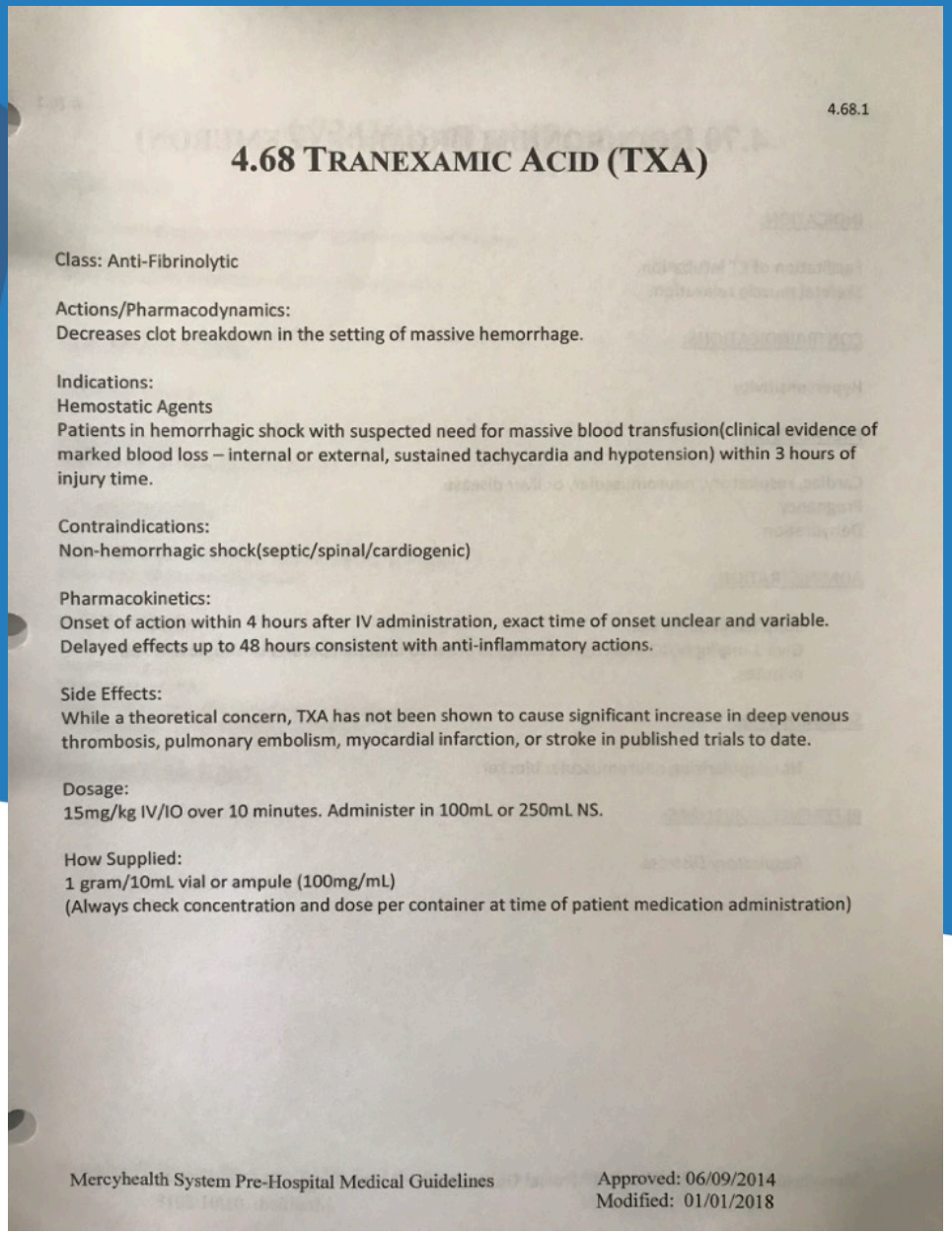

4.68.1

4.68 Tranexamic Acid (TXA)

Class: Anti-Fibrinolytic

Actions/Pharmacodynamics:
Decreases clot breakdown in the setting of massive hemorrhage.

Indications:
Hemostatic Agents
Patients in hemorrhagic shock with suspected need for massive blood transfusion(clinical evidence of marked blood loss – internal or external, sustained tachycardia and hypotension) within 3 hours of injury time.

Contraindications:
Non-hemorrhagic shock(septic/spinal/cardiogenic)

Pharmacokinetics:
Onset of action within 4 hours after IV administration, exact time of onset unclear and variable.
Delayed effects up to 48 hours consistent with anti-inflammatory actions.

Side Effects:
While a theoretical concern, TXA has not been shown to cause significant increase in deep venous thrombosis, pulmonary embolism, myocardial infarction, or stroke in published trials to date.

Dosage:
15mg/kg IV/IO over 10 minutes. Administer in 100mL or 250mL NS.

How Supplied:
1 gram/10mL vial or ampule (100mg/mL)
(Always check concentration and dose per container at time of patient medication administration)

Mercyhealth System Pre-Hospital Medical Guidelines

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