

TYPES OF SHOCK	Hypovolemic	Cardiogenic	Distributive	Obstructive	Neurogenic or Spinal
<p>Assessment findings</p>	<p>- Trauma: MOI -> internal / external loss of blood (hemorrhage)</p> <p>- Medical: - Hx of fluid loss, i.e. vomiting, diarrhea, dehydration... - Bleeding from mouth or rectum - bright or dark red - coffee-ground emesis (vomiting) - melena (black tarry stools)</p> <p>s/s: - dizziness or syncope w/sitting or standing - orthostatic changes in vital signs</p>	<p>Caused by inadequate function of the heart or pump failure. The backup of blood into the lungs (pulmonary edema) is a major effect leading to impaired ventilation.</p> <p>- Chief complain of: - chest discomfort - dyspnea - syncope/near syncope associated w/altered HR.</p> <p>- Abnormal hearth rhythms/rate: - bradycardia - excessive Tachycardia - dysrhythmias</p> <p>- Signs of acute CHF: - pulmonary edema - jugular venous distention - orthopnea</p>	<p>Occurs with any medication suggesting vasodilation & "leaky" vessels.</p> <p>Examples: - Anaphylaxis - Septic or infection - Certain drug overdose</p> <p>s/s: - signs of a sympathetic nervous system response: - ↑ HR - ↑ RR - clammy or diaphoretic skin - warm, flushed skin, specially in dependent areas - systemic swelling & brochoconstriction - hives</p>	<p>Physical obstruction of blood flow or ventilation.</p> <p>Insufficient O₂ in the blood will produce shock.</p> <p>Examples: - Significant chest wall trauma: - Rib or sternal fractures - Tension pneumothorax - Pericardial tamponade - Medical cause: - Pulmonary embolus</p> <p>s/s: - mechanism of injury - signs of poor perfusion/shock - sympathetic nervous system response (↑ HR + ↑ RR) - dyspnea (SOB) and/or chest discomfort</p>	<p>- Associated specifically with spinal trauma, vessels below the spinal injury dilate.</p> <p>- Rare occurrence</p> <p>- MOI - Falls: landing feet-first or head-first - Penetrating trauma to the back with neurological deficits.</p> <p>s/s: Loss of function below the site of spinal cord injury: - flaccid paralysis distal to injury site - loss of sympathetic NS function: - relative bradycardia - hypotension - vasodilation: warm, pink, dry skin - loss of bladder control - priapism (persistent, and often painful, penile erection)</p>
<p>Dysfunctions in perfusion</p>	<p>- Loss of plasma/fluid</p> <p>- Loss of red blood cells (=> inability to deliver enough O₂ to the cells)</p>	<p>- Altered HR - Reduced preload in tachycardia - Reduction in timely cardiac output</p> <p>- Myocardial trauma - Damage to contractile & electrical cells - Ineffective pumping</p> <p>- CHF or severe AMI - Damage to contractile cells - Fluid & pressure backs up into the lungs (=> SOB)</p>	<p>Slow movement of red blood cells to the tissues: - dilated vessels are unable to move fluid as effectively to the cells - "leaky" vessels encourage fluid to move out of the vascular system (=> pulmonary edema, pedal edema)</p>	<p>- Chest wall trauma - Inability to ventilate adequately</p> <p>- Tension pneumothorax - Compression of lung tissue & kinking of vena cava</p> <p>- Cardiac tamponade - Pressure against ventricles that ↓ cardiac output.</p> <p>- Pulmonary embolus - Obstruction of the pulmonary artery - Inefficient loading of red blood cells at the lungs</p>	<p>- Similar to distribute shock</p> <p>- Loss of sympathetic tone - ↓ HR, - ↓ cardiac output - ↓ peripheral vascular resistance.</p> <p>Parasympathetic nervous system kicks in.</p>
<p>Treatment</p>	<p>- High flow O₂</p> <p>- Secure & maintain an airway & provide respiratory support as needed.</p> <p>- Minimize the loss of red blood cells in uncontrolled bleeding</p> <p>- Increase the amount of circulating fluid.</p> <p>- Splint any bone & joint injuries</p> <p>- If no fracture extremities, raise legs 6" to 12".</p> <p>- Conserve body heat w/blankets</p> <p>- Rapid Transport</p>	<p>Base treatment on the particular dysfunction: - Symptomatic Bradycardia: Atropine - Tachycardia: - vagal maneuver - O₂ - fluid bolus if no signs of pulmonary edema (keep in mind that by given fluids you will increase the workload of a sick heart) - Myocardial trauma: - High flow O₂ - fluid bolus is no signs of pulmonary edema - MI/CHF: - O₂ - other drugs if BP allows for their admin. - Rapid Transport</p>	<p>- High flow O₂</p> <p>- Secure & maintain an airway & provide respiratory support as needed.</p> <p>- Fluid boluses – septic shock</p> <p>- Specific treatments for: - anaphylaxis: - Epi 1:1,000 - 0.1-0.5mg - Benadryl - 10-50mg - overdose: - Narcan 0.4-2mg</p> <p>- Conserve body heat w/blankets</p> <p>- PASG</p> <p>- Rapid Transport</p>	<p>- Early recognition and transport to a trauma or critical care hospital. - Recognition is often difficult - Use of MOI or patient history may assist in recognition</p> <p>- Aggressive ventilation w/high flow O₂</p> <p>- Fluid boluses</p> <p>- Rapid Transport</p>	<p>- High flow O₂</p> <p>- Secure & maintain an airway & provide respiratory support as needed.</p> <p>- Large-bore IV with fluid boluses</p> <p>- Atropine with slow rates</p> <p>- PASG</p> <p>- Rapid Transport</p>

STAGES OF SHOCK	Compensated	Decompensated	Irreversible
Assessment findings	<ul style="list-style-type: none"> - 15-25% of fluid loss from the vessels - Signs are subtle - Compensation by the sympathetic NS - Patient may show signs of an adrenaline rush: ↑ HR + ↑ RR (bcos the sympathetic nervous system kicks in) - Body cells begin to starve for O₂, resulting in: <ul style="list-style-type: none"> - Anxiousness - Pale skin - Complain of thirst - Complain of weakness 	<ul style="list-style-type: none"> - 25-35% of fluid loss from the vessels - The body cells are profoundly hypoxic - The sympathetic NS can NOT maintain perfusion - Classic signs of shock 	<ul style="list-style-type: none"> - > 35% fluid loss from the vessels - Low blood volume ↓ cardiac preload: <ul style="list-style-type: none"> - ↓ cardiac output - loss of peripheral pulses - very ↓ BP - Body cells die - All vital signs bottom out
HR	↑ HR (tachycardia), i.e. 120-ish	↑↑ HR (marked tachycardia), i.e. 140-ish	Slow
RR	Normal or ↑ RR (little change)	↑↑ RR: marked tachypnea	Slow / Agonal (shallow, slow (3-4 per minute), irregular inspirations followed by irregular pauses)
LOC	Anxiousness, restless	Confused, obvious alteration in mental status	Unconsciousness/Coma
SKIN	<ul style="list-style-type: none"> - Pale - Cool - Clammy - Peripheral sweating 	<ul style="list-style-type: none"> - Very pale - Cold - Sweating (diaphoretic) 	Very poor skin signs: <ul style="list-style-type: none"> - Mottled/ Waxed - Cold - No sweat
BP	Very little change (normal or high)	↓ BP	None or very ↓ BP
Pathophysiology	<ul style="list-style-type: none"> - Baroreceptors sense a drop in pressure in the arteries - Medulla is stimulated - Sympathetic NS Response: <ul style="list-style-type: none"> - Clammy and pale skin - ↑ HR + ↑ RR - Cells receive less O₂ as a result of a loss of red blood cells by the loss of fluid - Pre-capillary sphincter closes in periphery (blood shunted to the core) <p><u>Next wave of compensated shock:</u></p> <ul style="list-style-type: none"> - Sympathetic NS Response continues (adrenal hormones released) - Chemoreceptors sense changes in CO₂, and O₂ concentrations - Medulla stimulated again: <ul style="list-style-type: none"> - ↑ RR - HR continues to ↑ 	<ul style="list-style-type: none"> - Less blood flow and O₂ delivery impact more of the body: <ul style="list-style-type: none"> - Tissues in the core become hypoxic - More shifts to anaerobic metabolism - Organ function slows 	<ul style="list-style-type: none"> - The medulla stops working Sympathetic nervous system stimulation ceases - Heart function drops Drop in heart rate and contractility - Vessels dilate No more energy to constrict
Cellular Activities in Shock	<p><u>Cellular Ischemic Phase</u></p> <ul style="list-style-type: none"> - ↓ O₂ delivery cause the cells transition from aerobic to anaerobic metabolism in the periphery: <ul style="list-style-type: none"> - cells begins to build lactic acid - cellular activity begins to slow - Elevated levels of acid stimulate chemoreceptor <ul style="list-style-type: none"> - stimulation of the medulla - ↑ HR - Closure of the pre-capillary sphincter causes stagnation of blood <ul style="list-style-type: none"> - Blood begins to coagulate behind the closed pre-capillary sphincter - Post-capillary sphincter closes 	<p><u>Capillary Stagnation Phase</u></p> <ul style="list-style-type: none"> - Loss of fluid and O₂ delivery begin to impact more of the body. Organs and cells in the core begin to transition to anaerobic metabolism. - Lactic acid build in the cells, and leak into the capillaries: <ul style="list-style-type: none"> - stimulates opening of the pre-capillary sphincter - stagnated and clotted blood moves into the capillaries - Cell function drops dramatically - Continued closure of post-capillary sphincters: <ul style="list-style-type: none"> - pressure changes cause fluid shifts into the capillaries - drop in circulating blood volume =>reduce preload: <ul style="list-style-type: none"> - Cardiac output drops - Blood pressure begins to fall 	<p><u>Capillary Washout Phase</u></p> <ul style="list-style-type: none"> - Cell functions cease and cells die from lack of O₂ - Large accumulations of acid force the opening of the capillary sphincters, => sludge, clots and acid circulates throughout the entire body - Organ function fails from profound acidosis and hypoxia. <ul style="list-style-type: none"> - ↓ HR - ↓ RR - Loss in vascular constriction