Pathophysiology of Shock

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Overview

- Hypoperfusion
- Components of the Circulatory System
- Pathophysiology of Hypoperfusion
- Types of Shock
- Evaluation and Treatment Strategies
- Multiple Organ Dysfunction Syndrome
Hypoperfusion
Hypoperfusion (shock) is inadequate perfusion of body tissues. Inability of the body to meet its metabolic demands.
Hypoperfusion

- Shock occurs first at the cellular level and progresses to the tissues, organs, organ systems, and ultimately the entire organism.
- Final common pathway
  - Systemic Acidosis
Components of the Pulmonary and Circulatory System

- Normal tissue perfusion is dependent upon:
  - Adequate respiratory system
  - The pump (heart)
  - The fluid (blood)
  - The container (blood vessels)

Any problem with the components can lead to inadequate tissue perfusion.
The movement and utilization of oxygen in the body is dependent upon the following conditions:

- Adequate concentration of inspired oxygen.
- Appropriate movement of oxygen across the alveolar/capillary membrane into the arterial bloodstream.

Onloading
The Fick Principle

- Adequate number of red blood cells to carry the oxygen.
- Proper tissue perfusion.
- Efficient off-loading of oxygen at the tissue level.
Components of the Circulatory System
The Pump

- The heart is the pump of the cardiovascular system.
- Receives blood from the venous system, pumps it to the lungs for oxygenation, and then pumps it to the peripheral tissues.
First component = blood flow through right heart

- Unoxygenated blood flows from inferior and superior vena cavae into the right atrium, through the tricuspid valve, into the right ventricle and through the pulmonic valve
The second component of blood flow through the pulmonary circulation continues:

- the blood travels from PULMONARY ARTERIES into the LUNGS, through the PULMONARY ALVEOLAR-CAPILLARY NETWORK and into the PULMONARY VEINS
BLOOD FLOW THROUGH THE HEART

The third and final component of blood flow through the pulmonary circulation continues:

when the blood travels from the PULMONARY VEINS into the LEFT ATRIUM, through the MITRAL VALVE, into the LEFT VENTRICLE, through the AORTIC VALVE and out to the rest of the body.
**CARDIAC CYCLE**

- **Cardiac Cycle** - represents the actual time sequence between ventricular contraction and ventricular relaxation.

- **SYSTOLE** = simultaneous contraction of the ventricles.

- **DIASTOLE** = synonymous with ventricular relaxation - ventricles fill with 70% of blood passively from atria.
STROKE VOLUME

**Stroke Volume**
- volume of blood ejected out of one ventricle of heart during single beat or contraction
- Estimated at approximately 70 cubic centimeters per beat

**Heart Rate**
- number of contractions / beats per minute
- Normal Heart Rate = 60 to 100 beats per minute
Factors affecting stroke volume:

- **PRE-LOAD** = pressure in the ventricles at the end of diastole
  - Amount of blood delivered to the heart during diastole
  - Volume and pressure available to the ventricles for cardiac contraction
  - Dependent upon or equal to venous return
  - Left ventricular end diastolic pressure (LVEDP)

- Cardiac contractile force—the strength of contraction of the heart.

- Afterload—the resistance against which the ventricle must contract.
This concept is a law of physiology which states that up to a limit, the more myocardial fibers are stretched by chamber filling, the greater will be the force of contraction.

- The “RUBBER BAND THEORY”, the farther you stretch a rubber band, the harder it snaps back to original size.
Contractile Force

- Is affected by circulating hormones known as catecholamines.
  - Epinephrine
  - Norepinephrine
    - Neurotransmitters of sympathetic nervous system
Cardiac Output = amount of blood pumped by left ventricle in 1 minute

**FORMULA** to determine cardiac output:

\[
\text{Cardiac output [CO]} = \text{Stroke Volume [SV]} \times \text{Heart Rate [HR]}
\]

Compensation
Blood Pressure

- Peripheral vascular resistance is the pressure against which the heart must pump.
- Determined by vasoconstriction and vasodilation
- Measured as diastolic pressure
- Formula: Blood Pressure = Cardiac Output \times Peripheral Vascular Resistance Compensation
Circulation

- **Systolic Pressure**
  - Strength and volume of cardiac output

- **Diastolic Pressure**
  - More indicative of the state of constriction of the arterioles

- **Mean Arterial Pressure**
  - 1/3 pulse pressure added to the diastolic pressure
  - Tissue Perfusion Pressure
Cardiac Physiology

Nervous Control of the Heart (ANS)

- Sympathetic
- Parasympathetic
- Autonomic Control of the Heart

Chronotropy
Inotropy
Dromotropy
Autonomic Nervous System

- Regulates functions of the body that are involuntary or are not under conscious control
- Heart rate and blood pressure are regulated by this component of nervous system
Two Major Divisions of Autonomic Nervous System

- **Sympathetic Nervous System** = preparation of body for physical activity [“fight or flight”]
- **Parasympathetic Nervous System** = regulates the calmer [“rest and digest”] functions of our existence
Cardiovascular System Regulation

Parasympathetic Nervous System

- **Decrease**
  - Heart rate
  - strength of contractions
  - blood pressure

- **Increase**
  - Digestive system
  - Kidneys

Sympathetic Nervous System

- **Increase**
  - Body activity
  - Heart rate
  - Strength of contractions
  - Vascular constriction
    - Bowel & Digestive Viscera
    - Decreased urine production
  - Respiration
  - Bronchodilation

- Increases skeletal muscle perfusion
PNS & SNS always act in balance

Baroreceptors: Monitor BP

- Location
  - Aortic Arch
  - Carotid Sinuses

- Send Impulses to the Medulla
  - Cardioacceleratory Center
    - SNS: controls release of E and NE
  - Cardioinhibitory Center
    - PNS: controls the vagus nerve

Vasomotor Center
- Arterial and Venous tone
Chemoreceptors
- Located in carotid bodies and aortic arch
- Central and peripheral chemoreceptors
- Monitors level of CO2 in CSF
- Monitors level of O2 in blood
Adrenergic Receptors and Effect on Heart Rate

- **Adrenergic** - sympathetic nerve fibers that use epinephrine or epinephrine-like substances as neurotransmitters
- **Receptor** - a reactive site or cell surface within that combines with molecule to produce physiological effect
- **Cholinergic** - parasympathetic nerve fibers that use acetylcholine as neurotransmitter
Blood is thicker and more adhesive than water.

Consists of plasma and the formed elements.
- Red cells, white cells, platelets

Transports oxygen, carbon dioxide, nutrients, hormones, metabolic waste products, and heat.

An adequate amount is needed for perfusion, and must be adequate to fill the container.
Blood vessels serve as the container of the cardiovascular system. Under control of the autonomic nervous system they can adjust their size and selectively reroute blood through microcirculation. Microcirculation is comprised of the small vessels: arterioles, capillaries, and venules.
Capillaries have a sphincter between the arteriole and capillary called the pre-capillary sphincter.

The pre-capillary sphincter responds to local tissue demands such as acidosis and hypoxia, and opens as more blood is needed.
Post-capillary Sphincter

- At the end of the capillary between the capillary and venule is the post-capillary sphincter.
- The post-capillary sphincter opens when blood needs to be emptied into the venous system.
Sphincter Functioning

- CO₂ increases
- O₂ falls
- Drop in pH
- Sphincters Constrict
- MAST Cells Stop Releasing HISTAMINE
- CO₂ decreases
- O₂ returns
- pH normal
- Sphincters Dilate
The Pathophysiology of Hypoperfusion
Causes of Hypoperfusion

- Inadequate pump
  - Inadequate preload.
  - Inadequate cardiac contractile strength.
  - Excessive afterload.
Causes of Hypoperfusion

- Inadequate fluid
  - Inability to adequately fill the container
  - Hypovolemia
    Blood and/or fluid (water) losses
Causes of Hypoperfusion

- Inadequate container
  - Dilated container without change in fluid volume (inadequate systemic vascular resistance).
    - Relative hypovolemia
  - Leak in the container.
Shock at the Cellular Level

Shock causes vary, however the ultimate outcome is impairment of cellular metabolism.
  - Acidosis
Impaired Use of Oxygen

- When cells don’t receive enough oxygen or cannot use it effectively, they change from aerobic to anaerobic metabolism.
Cellular Metabolism

Two Step Process

- Glycolysis
  Cell utilizing energy source
  Releases energy
- Aerobic Metabolism: 95% of cellular Energy
  Requires oxygen and glucose
  Kreb’s cycle (citric acid cycle)
    • Uses carbohydrates, proteins and fats to release energy

Other Processes

- Anaerobic Metabolism
  Inadequate oxygen pathway
  Byproducts: Pyruvic Acid ➔ Lactic Acid
  Cellular death eventually occurs due to inadequate perfusion
Glucose breakdown. (A) Stage one, glycolysis, is anaerobic (does not require oxygen). It yields pyruvic acid, with toxic by-products such as lactic acid, and very little energy. (B) Stage two is aerobic (requires oxygen). In a process called the Krebs or citric acid cycle, pyruvic acid is degraded into carbon dioxide and water, which produces a much higher yield of energy.
Usually the body is able to compensate for any changes. However when the various compensatory mechanisms fail, shock develops and may progress.
Compensation Mechanisms

- The catecholamines epinephrine and norepinephrine may be secreted.
- The renin-angiotensin system aids in maintaining blood pressure.
- Another endocrine response by the pituitary gland results in the secretion of anti-diuretic hormone (ADH).
Catecholamines

- Epinephrine
- Norepinephrine

Actions

- **Alpha 1**
  - Vasoconstriction
  - Increased peripheral vascular resistance
  - Increased preload

- **Alpha 2**
  - Regulates release of NE

- **Beta 1**
  - Positive inotropy
  - Positive chronotropy
  - Positive dromotropy

- **Beta 2**
  - Bronchodilation
  - Smooth muscle dilation in bowel

Cardiovascular System Regulation

Hormone Regulation
Antidiuretic Hormone (ADH)

- aka: Arginine Vasopressin (AVP)
- Released
  - Posterior Pituitary
  - Drop in BP or Increase in serum osmolarity
- Action
  - Increase in peripheral vascular resistance
  - Increase water retention by kidneys
  - Decrease urine output
  - Splenic vasoconstriction
    - 200 mL of free blood to circulation
Angiotensin II

- Released
  - Primary chemical from Kidneys
  - Lowered BP and decreased perfusion

- Action
  - Converted from Renin into Angiotensin I
  - Modified in lungs to Angiotensin II
    - 20 minute process
    - Potent systemic vasoconstrictor
    - 1 hour duration
    - Causes release of ADH, Aldosterone and Epinephrine
Cardiovascular System Regulation
Hormone Regulation

- **Aldosterone**
  - **Release**
    - Adrenal Cortex
    - Stimulated by Angiotensin II
  - **Action**
    - Maintain kidney ION balance
    - Retention of sodium and water
    - Reduces insensible fluid
Cardiovascular System Regulation
Hormone Regulation

Glucagon

- Release
  Alpha Cells of Pancreas
  Triggered by Epinephrine

- Action
  Causes liver and skeletal muscles to convert glycogen into glucose
  - Gluconeogenesis
  - Cellular fuel source
Cardiovascular System Regulation
Hormone Regulation

- **Insulin**
  - Release
    - Beta Cells of Pancreas
  - Action
    - Facilitates transport of glucose across cell membrane

- **Erythropoietin**
  - Release
    - Kidneys
    - Hypoperfusion or hypoxia
  - Action
    - Increases production and maturation of RBC’s in the bone marrow
Stages of Shock

- **Compensated Shock**
  - Minimal Change

- **Decompensated Shock**
  - System beginning to fail

- **Irreversible Shock**
  - Ischemia and death imminent
Compensated shock is the early stage of shock during which the body’s compensatory mechanisms are able to maintain normal perfusion.
Decompensated shock is an advanced stage of shock that occurs when the body’s compensatory mechanisms no longer maintain normal perfusion.
Shock Variations

- Irreversible shock is shock that has progressed so far that the body and medical intervention cannot correct it.
### Table 4-2  The Stages of Shock

<table>
<thead>
<tr>
<th><strong>Compensated Shock</strong></th>
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<tbody>
<tr>
<td>Initial stage of shock in which the body progressively compensates for continuing blood loss.</td>
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<tr>
<td>• Pulse rate increases</td>
<td></td>
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<td>• Pulse strength decreases</td>
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<td>• Skin becomes cool and clammy</td>
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<tr>
<td>• Progressing anxiety, restlessness, combativeness</td>
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<tr>
<td>• Thirst, weakness, eventual air hunger</td>
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</table>

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<tr>
<th>** Decompensated Shock **</th>
<th></th>
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<tbody>
<tr>
<td>Begins when the body's compensatory mechanisms can no longer maintain preload.</td>
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<tr>
<td>• Pulse becomes unpalpable</td>
<td></td>
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<tr>
<td>• Blood pressure drops precipitously</td>
<td></td>
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<tr>
<td>• Patient becomes unconscious</td>
<td></td>
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<tr>
<td>• Respirations slow or cease</td>
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<tr>
<th><strong>Irreversible Shock</strong></th>
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<td>Shortly after the patient enters decompensated shock, the lack of circulation begins to have profound effects on body cells. As they are irreversibly damaged, the cells die, tissues dysfunction, organs dysfunction, and the patient dies.</td>
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Types of Shock

- Cardiogenic
- Hypovolemic
- Distributive
  - Neurogenic
  - Septic
- Anaphylactic
Cardiogenic shock occurs in 6% to 8% of patients with ST-segment elevation MI (STEMI)

Overall mortality rate between 50% and 90%

Median time from infarction to development of shock is eight hours.
Pathophysiology

Cardiogenic shock

- State of decreased cardiac output (CO) producing inadequate tissue perfusion despite adequate or excessive circulating fluid volume
- Cardiogenic shock can occur secondary to left-sided or right-sided heart failure.
Cardiogenic shock usually results from AMI that affects at least 25% to 40% of the myocardium.

A relatively small AMI can precipitate cardiogenic shock in a patient with previous heart failure.
Other causes of cardiogenic shock

- Systolic
  - Beta-blocker or calcium-channel blocker OD
  - Myocardial contusion
  - Acidosis

- Diastolic
  - Ventricular hypertrophy
  - Restrictive cardiomyopathies
Other causes of cardiogenic shock

- **Afterload**
  - Outflow obstructions
  - Malignant hypertension
  - Hypertrophic cardiomyopathy

- **Structural/valvular**
  - Endocarditis, mitral or atrial regurgitation, mitral stenosis, tamponade
Reduction in cardiac output (CO) after AMI results in hypotension. Tachycardia ensues to compensate for reduced CO.

- Increased work = increased myocardial oxygen demand
- Decreased diastolic phase = decreased coronary artery filling

Further ischemia and myocardial dysfunction result
Cardiogenic Shock

- The heart loses its ability to supply all body parts with blood.
- Usually the result of left ventricular failure secondary to acute myocardial infarction or CHF.
- Many patients will have normal blood pressures.
- Usually indicative that 40% of the myocardium is injured
- Shock that persists after other forms have corrected
- High mortality (80%)
The major difference between cardiogenic shock and other types of shock is the presence of pulmonary edema causing:

- Respiratory distress.
- As fluid levels rise, crackles or rales may be heard on pulmonary auscultation.
- There may be a productive cough with white or pink-tinged foamy sputum.
- Cyanosis, altered mentation, and oliguria.
Treatment

- Assure an open airway.
- Administer oxygen.
- Assist ventilations as necessary.
- Keep the patient warm.
Treatment

- Elevate the patient’s head and shoulders.
- Establish IV access
  - Controversial
  - Patient dependent
  - Fluid resuscitation
- Monitor the heart rate.
- Dopamine or dobutamine may be administered.
Case Study
0445 hours

You, a paramedic, and your EMT partner are dispatched to a private residence for “difficulty breathing.”

The first responding engine arrives at the same time you do.
Initial Impression

- Patient’s wife meets you at the door.
  - You note that she looks worried.
- She lets you in and leads you to the living room while telling you that her husband “is really sick.”
- You hear labored, gurgling respirations as you approach the living room.
Initial Impression

56 y/o male
- Is sitting upright on couch
- Has severe respiratory distress
- Has pale, diaphoretic skin
- Shows accessory muscle use
- Looks tired
Discussion

- What is your initial impression of the patient’s status?
- What are your next, most immediate actions?
Initial Assessment

- Your partner administers oxygen via nonrebreather mask.
- A firefighter EMT takes the patient’s blood pressure as you listen to lung sounds.
Initial Assessment

You note

- Patient alert and oriented
  Extremely anxious
- Bibasler Rales extending up to the mid lobes
- Skin cold, diaphoretic, pale
- Radial pulse rapid, weak
Initial Assessment

- When asked, the patient is only able to reply in two- to three-word sentences but says:
  - Had onset of chest pain about 12 hours ago
  - Has substernal, dull, radiating pain to left arm and neck
  - Pain was a “7” at first but now subsided to a “2”
  - Has been sitting in chair ever since
  - Has had progressive difficulty breathing over past six hours
Do you immediately treat the patient's chest pain with sublingual (SL) nitroglycerin?

Why or why not?

You first need to be sure that the patient has an adequate BP to support nitroglycerin administration.
Initial Assessment

- **Patient history**
  - Hypertension
  - Numerous slipped discs

- **Medications**
  - Propranolol
  - Elavil
  - Naproxen

- **Allergy to codeine and hydrocodone**
Initial Assessment

- **Vital signs**
  - HR = 118, regular
  - RR = 30 shallow
  - BP = 72/48
  - SaO$_2$ = 91% on 15 Lpm
Is this patient in shock?
- What classification?
- Compensated or decompensated?
  Yes, the patient is in shock, specifically in cardiogenic shock, decompensated.

What are your additional assessment priorities?
- Assessment priorities include obtaining a 12-Lead ECG and deciding if intubation is necessary.
You reassess the patient’s airway, noting that
- It is open
- Patient can keep it open himself

However
- Patient is getting noticeably tired
- He is using accessory muscles
- He is occasionally bobbing his head
- SaO$_{2}$ 91% on 15 Lpm
Do you attempt to intubate?

- There is not a right or wrong answer here. A lot depends on local protocol, the availability to use sedatives or neuromuscular blockade agents (NMBAs), and personal experience and preference.
You decide not to intubate, and place the patient on the cardiac monitor as your partner initiates large-bore IV access.
Assessment

- Cardiac rhythm

Interpretation?
You perform a 12-Lead ECG.
Assessment

12-Lead ECG

Interpretation?
- Sinus Tachycardia with an anteroseptal MI
- Old inferior MI
Assessment

- Blood glucose = 110 mg/dL
- No jugular vein distention (JVD) or peripheral edema noted

Vital signs

- HR = 120 irregular
- RR = 30 shallow
- BP = 70/42
- SaO₂ = 90% on 15 Lpm
Based on the physical exam findings, does the patient have left- or right-sided heart failure?

- Left-sided heart failure.

What is your treatment plan?

- Treatment plan should include dopamine to correct hypotension and allow use of MONA for the AMI.
Dopamine is administered at 5 mcg/kg/min and titrated to a systolic BP of 90 mmHg.
What must the dopamine drip rate be to achieve a 5 mcg/kg/min administration?

- Patient weight estimated at 190 pounds
- Microdrip set (60 drops/mL)
- Dopamine concentration 400 mg in 250 mL of fluid
Assessment/Treatment

- BP rises to 102 systolic after administration of dopamine.
- Aspirin, nitro SL, furosemide administered
How much morphine should be administered?

- NONE! Patient has allergy to codeine and hydrocodone.
Treatment

- Patient is placed in a stair chair and moved to the stretcher outside the front door.
- Patient suddenly becomes very agitated and is in respiratory arrest by the time he reaches the stretcher.
- Patient is quickly moved to the ambulance with BVM ventilations provided.
An OPA is inserted and the patient hyperventilated while the paramedic prepares for intubation.
Treatment

- Patient is intubated.
Tube placement is confirmed.

- $\text{ETCO}_2$
- Lung sounds noted bilaterally
Ongoing Assessment

- Lung sounds still noted with rales in all fields.
- Patient fighting tube, opens eyes to verbal stimuli

Vital signs
- HR = 118
- RR = 12 shallow, assisted
- BP = 112/60
- \( \text{SaO}_2 = 93\% \) on 20 Lpm
Ongoing Assessment

You provide a report to the receiving facility.
Ongoing Assessment

Vital signs at time of arrival
- HR = 118
- RR = 12
- BP = 116/72
- SaO₂ = 95%
Endotracheal tube placement confirmed
Patient sedated, placed on ventilator
12-L ECG obtained
  AMI confirmed
Central venous access obtained
Dopamine discontinued, dobutamine drip initiated
ED Treatment and Beyond

- IV nitrates and beta-blockers administered

- Laboratory studies
  - CBC, coagulation profile
  - Electrolytes
  - Cardiac marker levels: Troponin, CK MB

- Imaging studies
  - Chest radiograph
  - Echocardiogram
**ED Treatment and Beyond**

- **Good News**
  - Patient receives emergent cardiac catheterization
- **Bad News**
  - Arrests during procedure, and cannot be resuscitated
  - Autopsy reveals complete occlusion of left coronary artery (LCA) and widespread infarction of left ventricle and septum
Hypovolemic Shock

- Shock due to loss of intravascular fluid.
  - Internal or external hemorrhage.
  - Trauma.
  - Long bones or open fractures.
  - Dehydration.
  - Plasma loss from burns.
  - Excessive sweating.
  - Diabetic ketoacidosis with resultant osmotic diuresis.
Greater Loss

- ↑ Cellular Ischemia
- ↓ Capillary Microcirculation
- ↑ Possibility of Capillary Washout
  - Buildup of lactic acid and CO₂
  - Relaxation of post capillary sphincters
  - Release of byproducts into circulation
    - • PROFOUND METABOLIC ACIDOSIS
Evaluation

- Altered level of consciousness.
- Pale, cool, clammy skin.
- Blood pressure may be normal, then fall.
Evaluation

- Pulse may be normal then become rapid, finally slowing and disappearing.
- Urination decreases.
- Cardiac dysrhythmias may occur.
Treatment

- Airway control.
- Control severe bleeding.
- Keep the patient warm.
- Fluid resuscitation
  - Bolus or boluses of crystalloid solution
  - 3cc for 1cc of blood loss
Neurogenic Shock

- Results from injury to brain or spinal cord causing an interruption of nerve impulses to the arteries.
- The arteries dilate causing relative hypovolemia.
- Sympathetic impulses to the adrenal glands are lost, preventing the release of catecholamines with their compensatory effects.
Blunt trauma is the cause of about 80% of all spinal-cord injuries.

- Auto collisions most frequent cause
- Falls, sports injuries

Cervical region most commonly injured

- Other regions in descending order of frequency are
  - Thoracolumbar junction, thoracic, lumbar
Parasympathetic nervous system stimulation results in:

- Bradycardia
- Bradycardia and reduced cardiac contractile force
- Vasodilation
  - Reduced systemic vascular resistance (SVR)
- Bronchoconstriction
- Increased GI motility, pupillary constriction
Sympathetic nervous system stimulation results in:

- Diaphoresis
- Vasoconstriction
- Tachycardia
- Bronchodilation
- Increased blood flow to skeletal muscles
- Decreased blood flow to abdominal organs
- Energy production/glucose release
Neurogenic shock and spinal shock are NOT the same!

The terms are NOT interchangeable!
Spinal shock

- Refers to numerous different neurologic manifestations of spinal-cord injury
  - Initial areflexia, flaccidity
  - Gradual onset of hypertonia, exaggerated reflexes, spasticity
- Manifestations occur over varying time periods after injury.
Neurogenic shock

- Type of distributive shock resulting from loss or suppression of sympathetic tone
  Rarest form of shock

- Most common cause is spinal-cord injury above T1.
Sympathetic nervous system originates from T1 – L6.

- Disrupt spinal cord above T1, remove sympathetic influence
- Parasympathetic (vagal) influence still exists, since neurons originate from brainstem.
“Balance” between sympathetic and parasympathetic nervous systems disrupted

Condition of unopposed vagal tone exists.

- Bradycardia
- Vasodilation
Vasodilation also results in warm, dry skin and hypothermia

- Warm blood floods peripheral vasculature.
- Heat is lost to environment.
Bradycardia and vasodilation result in:

- Decreased SVR
- Decreased venous return to heart
- Decreased preload
- Decreased stroke volume
- Decreased cardiac output = hypotension
- Decreased perfusion of peripheral tissues
- Impaired cellular metabolism
Simply put, a condition of *relative hypovolemia* exists.

The amount of fluid (blood) remains the same, but the size of the container (vasculature) gets larger.
Airway control.

Maintain body temperature.

Immobilization of patient.

Consider other possible causes of shock.

IV access (fluid challenge) and medications that increase peripheral vascular resistance.

- Alpha agonists (norepinephrine)
- Dopamine
Case Study
You and your partner, both paramedics, are dispatched to a residential address for an injury from a fall.
Initial Impression

- Fire service EMTs are at the scene when you arrive.
- Patient’s son meets you at the curb and directs you to the garage.
- He tells you that his father “fell down a flight of stairs.”
Patient presents

- Nasal airway in place
- Inadequate respirations
  - Firefighter ventilating with a BVM
- Fully exposed
- Fully immobilized
Discussion

- What is your initial impression of the patient’s status?

- What do you note about the EMT care?

- What are your first, most immediate actions?
Knowing it’s your turn to be in charge of patient care, your partner says, “I’ll go back to the ambulance and set up.”

One of the firefighter EMTs sees you approaching and gives you a report.
The EMT states

- Patient fell about 12 feet, landing on back and shoulder
- Patient initially responsive to deep pain only
- Snoring respirations were corrected with nasal airway
- Patient was initially hypotensive, bradycardic, and bradypneic
- Large bruise and abrasion across back
  C7–T2 level
Initial Assessment

While receiving the report, you are able to note:

- Patient responds to pain with moaning
- Good chest rise and fall with ventilation
- Patient appears to be “belly breathing”
- Lungs sounds present and clear bilaterally
- No movement of extremities noted
What might account for the patient's “belly breathing”?

- With the mechanism of injury and the bruise across the C7-T2 area, it is possible that the patient has suffered a spinal fracture with spinal cord insult. With a cord insult to this area, the patient would still have innervation of his diaphragm via the phrenic nerve (originates in the brainstem) but will have lost innervation of his intercostal muscles (nerves originate from the thoracic spinal cord). The result is continued diaphragm contraction without intercostal muscle contraction, leading to “belly breathing.”
One of the EMTs finishes taking vital signs and reports

- HR = 52, regular
- RR = <6, shallow
  Assisted respirations 12/minute
- BP = 68/40
Is the patient in shock?

- If so, what classification?
  The patient is in neurogenic shock, a subclassification of distributive shock
  - Compensated or decompensated?

What are your immediate priorities?

What are your next, most immediate actions?
Patient is moved into the ambulance.
You ask one of the firefighters to drive, so you and your partner can provide care.
Another jumps in back to assist with patient care.
Once in the ambulance, you immediately repeat an initial assessment while your partner ALSs the patient.
You note

- Airway clear and open
- Lung sounds clear and equal bilaterally
  Patients respiratory rate of 10–12/minute assisted with BVM
- No trauma noted to anterior or lateral chest
- Skin warm, dry
- No radial pulse, carotid pulse rate = 50
Assessment

- Continued assessment reveals
  - Head, abdomen, pelvis, and extremities atraumatic
  - No spontaneous movement of extremities noted
    - No response to painful stimuli

- Your partner reports a blood glucose of 142 mg/dL.
Your partner reports vital signs

- HR = 50 regular
- RR = being assisted
- BP = 64/unobtainable
- SaO$_2$ = 100 % on 20 Lpm while ventilated
Cardiac monitor

Interpretation?
What is your treatment plan for this patient?
Your partner initiates large-bore IV access and begins fluid resuscitation.
Your partner contacts medical control and gives a report.

Medical control orders you to administer one liter of fluid rapidly and start dopamine if hypotension is not resolved.
You initiate large-bore IV access on the other arm as your partner prepares a dopamine drip.
Patient remains profoundly hypotensive after one liter of fluid.

Dopamine administration is initiated at 5 mcg/kg/min.
What must the dopamine drip rate be to achieve a 5 mcg/kg/min administration?

- Patient weight estimated at 200 pounds
- Microdrip set (60 drops/mL)
- Dopamine concentration 400 mg in 250 mL of fluid
Patient is prepared for transfer to the ED.

Vital signs at time of arrival
- HR = 50 regular
- RR = 10
- BP = 78/38
- SaO₂ = 100%
- Lungs are clear
The dopamine dose is increased to 10 mcg/kg/min and the patient is transferred to the ED.
ED Treatment and Beyond

- ABCs are assessed.
- RSI is performed.
- Central venous access is obtained.
- Additional one liter of normal saline administered without effect
  - Dopamine administration continued
  - Dobutamine administration initiated
Imaging studies

- Trauma radiograph series obtained
  Fractures to C6, T1, and left scapula identified

Laboratory studies

- Full trauma panel

Methylprednisolone administered

Patient sent to CT scan for further evaluation, trauma service takes over care
CT reveals:
- Spinal column fractures to C7 and T1
- Spinal cord injury at T1

Swelling over next 2 days extends injury

Patient permanently loses function below injury

Transferred 28 days later to rehabilitation center
Anaphylactic Shock

- A severe immune response to a foreign substance.
- Signs and symptoms most often occur within a minute, but can take up to an hour.
- The most rapid reactions are in response to injected substances:
  - Penicillin injections.
  - Bees, wasps, hornets.
Because immune responses can affect different body systems, signs and symptoms vary widely:

- Signs and symptoms due to changes in capillary permeability
  - Skin: Flushing, itching, hives, swelling, cyanosis.
  - Respiratory system: Breathing difficulty, sneezing, coughing, wheezing, stridor, laryngeal edema, laryngospasm.
Evaluation

- **Cardiovascular system:**
  - Vasodilation, increased heart rate, decreased blood pressure.

- **Gastrointestinal system:**
  - Nausea, vomiting, abdominal cramping, diarrhea.

- **Nervous system:**
  - Altered mental status, dizziness, headache, seizures, tearing.
Treatment

- Airway protection, may include endotracheal intubation.
- Establish an IV of crystalloid solution.
- Pharmacological intervention:
  - Epinephrine, antihistamines (H1, H2 antagonists), corticosteroids, vasopressors, inhaled beta agonists.
Septic Shock

- An infection that enters the bloodstream and is carried throughout the body.
- The toxins released overcome the compensatory mechanisms.
- Can cause the dysfunction of an organ system or result in multiple organ dysfunction syndrome.
Epidemiology

- Incidence of septic shock about 3.0 cases per 1000 population in 2005
- About 50% of patients with sepsis develop septic shock.
- 30% to 45% of patients in septic shock die within one month.
Definitions

- **Infection**: inflammatory response to the presence of microorganisms or the invasion of a normally sterile host by those organisms

- **Bacteremia**: the presence of bacteria in the blood
Sepsis: a systemic response to infection, manifested by two or more of the following:

- Temperature >38°C or <36°C
- Heart rate >90 beats/min
- Respiratory rate >20 breaths/min or PaCO₂ <32mmHg
- WBC count >12,000/µL, <4000/µL, or >10% immature (band) forms
Pathophysiology

Definitions

- **Severe sepsis**: sepsis associated with
  - Organ dysfunction
  - Hypoperfusion
    - Lactic acidosis
    - Altered mental status
    - Oliguria
  - Hypotension
Definitions

- **Septic shock**: sepsis-induced hypotension refractory to adequate fluid administration along with perfusion abnormalities including
  - Lactic acidosis
  - Altered mental status
  - Oliguria
Sepsis originates as an infection. 

- Urinary tract infection, cellulitis, indwelling device, pneumonia, abscess

Organisms invade blood stream.

- Release endotoxins, exotoxins
Host releases endogenous mediators.

- Activation of humoral defense factors
- Cytokines, interleukins, platelet-activating factor, and myocardial depressant substances

Depression of myocardial function and vasodilation occurs.

Shock and end-organ failure ensue.
Evaluation

The signs and symptoms are progressive.

- Increased to low blood pressure.
- High fever, no fever, or hypothermic.
- Skin flushed, pale, or cyanotic.
- Difficulty breathing and altered lung sounds.
- Altered mental status.
Treatment

- Airway control.
- IV of crystalloid solution.
- Dopamine to support blood pressure.
- Monitor heart rhythm.
Case Study
0730 hours

You, a paramedic, and your EMT partner are dispatched to a private residence for a “sick person.”
Initial Impression

- Patient’s daughter meets you at the door and shows you to a rear bedroom.

- She tells you that her mother “has been sick” for a few days but “looks really bad this morning.”
You are presented with
- 70-year-old (y/o) female
- Unconscious
- Snoring respirations
- Tachypneic
- Slight peripheral cyanosis
- Diaphoretic skin
How would you describe the patient's status?

What are your first, most immediate actions?
Initial Assessment

- Your partner inserts a nasal airway and applies 100% oxygen as you begin your assessment.
Initial Assessment

You note

- Patient responds to pain by opening her eyes briefly, muttering incomprehensibly, and withdrawing.
- Snoring stops with nasal airway.
- Patient’s breathing is rapid and shallow, lung sounds clear bilaterally.
- Skin is cool, peripherally cyanotic, and diaphoretic.
- Radial pulses are rapid, weak, irregular.
- Movement to all extremities is noted.
When asked, the daughter tells you that the patient

- Had a urinary tract infection last week
- Complained of increased pain with urination, then flank pain and decreased urine output
- Developed a fever two days ago
- Has been “getting worse” since
- Is normally conscious, alert, and oriented
Discussion

- Based on the information given, what is the patient’s GCS?

- What additional information would you like to have?
Your partner takes vital signs while you place the patient on the cardiac monitor.
Initial Assessment

Patient medical history

- Myocardial infarction (MI) in past
- Noninsulin-dependent diabetes
- Rheumatoid arthritis
- Recent urinary tract infection
Initial Assessment

- **Patient medications**
  - Nitroglycerin as needed
  - Aspirin daily
  - Cardizem
  - Glyburide
  - Prednisone
  - Sulfamethoxazole

- **No known drug allergies**
Initial Assessment

- Cardiac rhythm

- Interpretation?
Initial Assessment

- **Vital signs**
  - HR = 110 irregular
  - RR = 24 shallow
  - BP = 80/48
  - $\text{SaO}_2 = 95\%$ on 15 Lpm

- You note that the peripheral cyanosis has improved slightly with oxygen administration.
Discussion

- *Is this patient in shock?*
  - *If so, compensated or decompensated?*

- *What could be the cause?*

- *What additional information would you like to have?*
Treatment

- Your partner sets up for a 12-Lead ECG while you initiate large-bore IV access.
Treatment

- You administer a fluid challenge and perform a 12-Lead ECG, while your partner checks blood glucose.
Treatment

12-Lead ECG

- Vent. rate 110 BPM
- PR interval * ms
- QRS duration 92 ms
- QT/QTc 320/432 ms
- P–R–T axes * 118 -62

Interpretation?
Discussion

What do you now think is the cause of the patient’s condition?
- Septic shock should be suspected.

Does the patient have any known risk factors for sepsis?
- Patient risk factors include recent urinary tract infection, chronic corticosteroid use, and diabetes
Assessment/Treatment

- You note that 250 cc of saline have been administered with no change in BP.
- You auscultate her lungs, and there is no change.
You begin another 250 cc fluid challenge and move the patient to a Reeves stretcher.
Patient is secured to the Reeves stretcher . . .
Treatment

... and moved to the
Assessment

- Once in the ambulance, you ask your partner for another set of vitals.
- You note the second 250 cc bolus is complete and check the patient’s airway status and lung sounds.
Patient gags, opens her eyes, and takes a swing at your partner when you attempt to insert the OPA.

Vital signs
- HR = 114 irregular
- RR = 24 shallow
- BP = 72/40
- SaO₂ = 94% on 15 Lpm
Would you intubate the patient at this point?

How would you treat the patient's worsening hypotension?
You begin dopamine administration at 5 mcg/kg/min and titrate to a BP of 100 systolic.
What must the dopamine drip rate be to achieve a 5 mcg/kg/min administration?

- Patient weight estimated at 110 pounds
- Microdrip set (60 drops/mL)
- Dopamine concentration 400 mg in 250 mL of fluid
You perform a reassessment and note

- Patient now responds to loud verbal stimuli
  - Opens her eyes
  - Asks, “Where am I?”
  - Does not know her name
- Weak radial pulses
- Cool, diaphoretic, pale skin
You tell the patient that you are a paramedic and that she is in an ambulance.

You then start to explain the situation to her.
Ongoing Assessment

- You contact medical control and give a report.
- Transport ends without incident.
- Vital signs at time of arrival to ED
  - HR = 100 irregular
  - RR = 24 shallow
  - BP = 102/58
  - SaO₂ = 95% on 15 Lpm
Reevaluation of ABCs
Oxygen via NRM continued
12-Lead ECG performed
Central venous access obtained
Chest radiograph obtained
- Lungs clear
ED Treatment and Beyond

- **Dobutamine administration initiated**
  - Dopamine ceased

- **Laboratory studies**
  - Blood cultures
  - Urine gram stain, urinalysis, and culture

- **Antimicrobial therapy initiated**
ED Treatment and Beyond

- Patient admitted for sepsis, develops pulmonary edema 1 day later.
- Patient intubated.
- Respiratory failure ensues; ARDS develops on day 6.
- Patient dies of respiratory failure on day 9.
Etiology of Shock
(Review)

- Hypovolemic Shock
  - Loss of blood volume

- Distributive Shock
  - Prevent appropriate distribution of nutrients and removal of wastes
    - Anaphylactic
    - Septic
    - Hypoglycemia

- Obstructive Shock
  - Interference with the blood flowing through the cardiovascular system
    - Tension Pneumothorax
    - Cardiac Tamponade
    - Pulmonary Emboli

- Cardiogenic Shock
  - Pump failure

- Respiratory Shock
  - Respiratory system not able to bring oxygen into the alveoli
    - Airway obstruction
    - Pneumothorax

- Neurogenic Shock
  - Loss of nervous control from CNS to peripheral vasculature
Shock Assessment in Review

- Scene Size-up
- Initial Assessment
- Focused H&P
  - Rapid Trauma
- Detailed H&P
- Ongoing Assessment
Shock Management in Review

- **Airway & Breathing**
  - NRB
  - PPV (overdrive respiration)
  - ET
  - Difficult Airway Devices
    - LMA, PtL, Combitube
  - Needle Decompression

- **Hemorrhage Control**

- **Fluid Resuscitation**
  - Catheter Size & length
  - Large Bore
  - 20ml/kg of NS or LR
  - STABILIZE VITALS
Shock Management in Review

- **Temperature Control**
  - Conserve core temperature
  - Warm IV Fluids

- **PASG**
  - **Action**
    - Increase PVR
    - Reduce Vascular. Volume
    - Increase central CBV
    - Immobilize lower extremities
  - **Assess**
    - Pulmonary Edema
Shock Management in Review

Pharmacology

- ONLY after Fluid Resuscitation
  
  Cardiogenic Shock
  - Fluid Challenge
  - Vasopressors: Dopamine
  - Cardiac Drugs: Epinephrine, Atropine

  Spinal & Obstructive Shock
  - IV resuscitation: NS & LR

  Distributive Shock
  - IV Resuscitation
  - Dopamine
  - PASG
Multiple Organ Dysfunction Syndrome

- MODS is the progressive impairment of two or more organ systems from an uncontrolled inflammatory response to a severe illness or injury.
MODS Stages
Primary MODS

- Organ damage results directly from a specific cause such as ischemia or inadequate tissue perfusion from shock, trauma, or major surgery.
- Stress and inflammatory responses may be mild and undetectable.
- During this response, neutrophils, macrophages, and mast cells are thought to be “primed” by cytokines.
The next time there is an injury, ischemia, or infection the “primed” cells are activated, producing an exaggerated inflammatory response.

The inflammatory response enters a self-perpetuating cycle causing damage and vasodilation.

An exaggerated neuroendocrine response is triggered causing further damage.
MODS 24 Hours After Resuscitation

- Low grade fever.
- Tachycardia.
- Dyspnea.
- Altered mental status.
- General hypermetabolic, hyperdynamic state.
MODS Within 24 to 72 Hours

- Pulmonary failure begins.
MODS Within 7 to 10 Days

- Hepatic failure begins.
- Intestinal failure begins.
- Renal failure begins.
MODS Within 14 to 21 Days

- Renal and hepatic failure intensify.
- Gastrointestinal collapse.
- Immune system collapse.
MODS After 21 Days

- Hematologic failure begins.
- Myocardial failure begins.
- Altered mental status resulting from encephalopathy.
- Death.
Summary

In a Nutshell.....

SHOCK is...

INADEQUATE

TISSUE

PERFUSION