Shock

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Introduction

1. Definition of shock
2. Types of shock
3. Causes of shock
4. Signs and symptoms of shock
5. Body’s response to shock
6. Treatment of shock
Definition of Shock

In 1852, shock was defined as “a rude unhinging of the machinery of life.” Probably no better definition exists to describe the devastating effects of this process on a patient, but a more recent definition calls shock “the collapse and progressive failure of the cardiovascular system.”

Shock left untreated may be fatal. It must be recognized and treated immediately, or the patient may die.
Definition of Shock

The definition of shock does not involve low blood pressure, rapid pulse or cool clammy skin - these are merely the signs. Simply stated, shock results from inadequate perfusion of the body’s cells with oxygenated blood.
Types of Shock

- Hypovolemic shock
  - Hemorrhage
  - Burns
  - Diarrhea
  - Vomiting
  - Peritonitis
Types of Shock

Cardiogenic

Cardiomyopathy
Pulmonary Embolism
Heart Disease
Myocardial Infarction

Cardiac Contusion
Cardiac Tamponade
Aortic Aneurysm
Arrhythmia
Types of Shock

- Vasogenic
  - Psychogenic
  - Septic
  - Anaphylactic
Review of Body Fluids: Distribution of Body Water

**Intracellular**
- K+
- Protein
- PO₄⁻

**Extracellular**

- Na⁺
- Cl⁻

**Interstitial**

**Blood**

**RBC**
Distribution of Body Water

- Varies with
  - Age
  - Sex
  - Nutrition
  - Hydration
  - Disease
  - Lean body mass (usual blood volume varies... 50, 60, 70, 80 cc per kgm of LBM)
Types of Shock
Hypovolemic Shock

- Hypovolemic shock refers to a medical or surgical condition in which rapid fluid loss results in multiple organ failure due to inadequate perfusion.
  - Trauma
  - Hemorrhage
  - Vomiting / diarrhea
  - Burns
Hypovolemic Shock

- The human body responds to acute hemorrhage by activating 4 major physiologic systems: the hematologic system, the cardiovascular system, the renal system, and the neuroendocrine system.
Hypovolemic Shock: Hematologic System

- Activating the coagulation cascade and contracting the bleeding vessels (via local thromboxane A2 release)
- Platelets are activated which form an immature clot on the bleeding source
- The damaged vessel exposes collagen, which subsequently causes fibrin deposition and stabilization of the clot.
Hypovolemic Shock: Cardiovascular System

- Increases the heart rate, increasing myocardial contractility, and constricting peripheral blood vessels.
- This response occurs secondary to an increase in release of norepinephrine and a decrease in baseline vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels).
- The cardiovascular system also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.
Hypovolemic Shock: Renal System

- The kidneys respond to hemorrhagic shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus.

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Renin

Angiotensinogen

Angiotensin II

Lungs and Liver

Angiotensin I
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Angiotensin II
Hypovolemic Shock: Renal System

- Angiotensin II has 2 main effects, both of which help reverse hypovolemic shock, vasoconstriction of arteriolar smooth muscle and stimulation of aldosterone secretion by the adrenal cortex.
Hypovolemic Shock: Neuroendocrine System

- Causes an increase in circulating antidiuretic hormone (ADH)
- ADH is released from the posterior pituitary gland in response to a decrease in blood pressure (as detected by baroreceptors) and a decrease in sodium concentration.
- ADH indirectly leads to an increase in reabsorption of water and salt (NaCl) by the distal tubule, the collecting ducts, and the loop of Henle.
Vasogenic Shock

- Septic Shock
  - Hyperdynamic
  - Hypodynamic
- Toxic Shock
- Neurogenic Shock
- Psychogenic Shock
Psychogenic Shock

- Also known as “fainting spells”
- Caused by sudden dilation of blood vessels which temporarily halts blood flow to the brain
Neurogenic Shock

- Failure of the nervous system to control diameter of blood vessels
- Causes pooling of blood and there is generally no actual blood loss
- Classic signs of shock may not be present
Septic Shock vs. SIRS

- Systemic Inflammatory Response Syndrome (SIRS)
  - physiologic alternations and organ dysfunction seen with bacterial infections
  - 2 or more changes in these 4 factors, as follows: body temperature, heart rate, respiratory function, and peripheral leukocyte count.
Septic Shock vs. SIRS

- **Sepsis**
  - systemic host response to infection with SIRS plus a documented infection

- **Severe Sepsis**
  - sepsis plus end-organ dysfunction or hypoperfusion

- **Septic Shock**
  - sepsis with hypotension, despite fluid resuscitation with evidence of inadequate tissue perfusion
Septic Shock: Pathophysiology

- In septic shock due to bacterial infection, circulatory insufficiency occurs when bacterial products interact with host cells and serum proteins to initiate a series of reactions that may ultimately lead to cell injury and death.
- Not only are these bacterial products harmful themselves, but the widespread and unregulated host response to these substances also results in the elaboration or an extensive array of chemical mediators that lead to further cell damage.
Septic Shock: Pathophysiology

- Septic shock develops in less than one half of patients with bacteremia. It occurs in about 40% of those patients with gram-negative bacteremia and about 20% of those patients with *Staphylococcus aureus* bacteremia.
Septic Shock: History

- Fever
- Chills
- Sweating
- Altered mental status
  - Apprehension
  - Anxiety
  - Agitation
Septic Shock: History

- Some localizing symptoms
  - Head and neck infections - earache, sore throat, sinus pain or congestion, nasal congestion or exudate, swollen lymph glands
  - Chest and pulmonary infections - cough (especially if productive), pleuritic chest pain, dyspnea
  - Abdominal and GI infections - abdominal pain, nausea, vomiting, diarrhea
  - Pelvic and genitourinary infections - pelvic or flank pain, vaginal or urethral discharge, dysuria, frequency, urgency
  - Bone and soft tissue infections - focal pain or tenderness, focal erythema, edema
Cardiogenic Shock

- Cardiogenic shock is characterized by a decreased pumping ability of the heart causing a shock-like state with inadequate perfusion to the tissues.
- It occurs most commonly in association with, and as a direct result of, acute ischemic damage to the myocardium.
Cardiogenic Shock

- Intrinsic
  - Myocardial injury
  - Tachycardia
  - Bradycardia
  - Valvular defect

- Extrinsic
  - Pericardial tamponade
  - Tension pneumothorax
  - Large pulmonary embolus
Cardiogenic Shock: History

- Most patients presenting with cardiogenic shock do so in conjunction with an AMI and, therefore, present with the constellation of symptoms of acute cardiac ischemia (e.g., chest pain, shortness of breath, diaphoresis, nausea and vomiting).
- Patients experiencing cardiogenic shock may also present with pulmonary edema and presyncopal or syncopal symptoms.
Cardiogenic Shock: Physical

- Physical examination will often reveal a patient in the middle of an AMI.
- Patients appear in frank extremis, profoundly diaphoretic and complaining of severe shortness of breath and chest pain.
- Clinical assessment begins with attention to the ABCs and vital signs.
Cardiogenic Shock: Physical

- Neck examination may reveal jugular venous distention. This is evidence of right ventricular failure and may be prominent.
  - With increasing degrees of ventricular dysfunction, florid pulmonary edema and severe hypotension may develop.
  - Auscultation of the chest may reveal varying degrees of congestive heart failure (CHF).
Cardiogenic Shock: Physical

- Careful attention should be directed toward the cardiac examination, as there are mechanical causes of cardiogenic shock which are readily amenable to surgical intervention, and without which the mortality is dismal.
  - These include papillary rupture, valvular dysfunction, myocardial wall or septal rupture, cardiac tamponade and aortic aneurysm.
  - Loud murmurs may indicate a valvular dysfunction while muffled heart tones with JVD and pulsus paradoxus may suggest tamponade.
## Signs and Symptoms of Shock

### Hard-core measurements
- Blood pressure changes ↑ or ↓
- Pulse rate ↑ or ↓
- Hemoglobin / hematocrit ↓ or NL
- Urine output ↓
- Electrocardiogram
- Arterial blood gas
- **Pulmonary artery wedge pressure**
- **Cardiac output**
- **Cardiac index**
- **Central venous pressure**

### Soft-core measurements
- Skin changes (cool, pale or damp)
- Altered Sensorium (depressed or apprehensive)
- Thirst
- Vein changes
- Hyperventilation
- Obvious or occult blood loss

** requires invasive monitoring
Mechanisms of Response to Severe Stress

- Distribution of regional blood flow
- Oxygen transport mechanisms
  - Temperature
  - Anaerobic metabolism
  - Ventilation
  - Oxygen extraction
  - Blood hemoglobin concentration
Composite of Reported Responses to Hemorrhage in Man

<table>
<thead>
<tr>
<th>% Loss of Circulating blood volume</th>
<th>Pulse Rate</th>
<th>Systolic pressure</th>
<th>Pulse pressure</th>
<th>Capillary refill</th>
<th>Respirations</th>
<th>Central nervous system</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 15%</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>15 – 30%</td>
<td>&gt; 100</td>
<td>Normal</td>
<td>↓</td>
<td>Delayed</td>
<td>Mild tachypnea</td>
<td>Anxious</td>
<td>20-30 ml/hr</td>
</tr>
<tr>
<td>30 – 40%</td>
<td>&gt; 120, weak</td>
<td>↓</td>
<td>↓</td>
<td>Delayed</td>
<td>Marked tachypnea</td>
<td>Confused</td>
<td>20 ml/hr</td>
</tr>
<tr>
<td>&gt; 40%</td>
<td>&gt; 140, non-palpable</td>
<td>Marked ↓</td>
<td>Marked ↓</td>
<td>Absent</td>
<td>Marked tachypnea</td>
<td>Lethargic</td>
<td>Negligible</td>
</tr>
</tbody>
</table>
Factors Effecting Response to Shock and Treatment

- Magnitude of insult
- Health of patient
- Age
- Temperature extremes (hot or cold)
- Altitude
- Humidity
- Fear
- Pain
- Hydration status
- Nutrition
- Pre-existing diseases
- Drugs taken by patient (especially cardiac meds)
Normal Changes With Age Affecting Response to Shock

- **Cardiac**
  - Stiffer myocardium
  - Valvular damage
  - Poorly functioning AV node and Bundle of His

- **Lungs**
  - Pulmonary fibrosis
  - Loss of alveoli
  - Inadequate rib movement
Normal Changes With Age Affecting Response to Shock

- **Liver**
  - Decreased function
  - Especially in those with history of alcohol abuse

- **Kidneys**
  - Decreased number of glomeruli
  - Decreased function of tubules

- **Blood vessels**
  - Stiff atherosclerotic arteries
  - Ectatic arteries and veins
Treatment of Shock: Goal

TO RESTORE NORMAL TISSUE PERFUSION

- Blood pressure
- Pulse
- Respirations
- Skin Appearance
- Sensorium
- Urine output (30-50 cc per hour)
- Hemoglobin 8-10 gm or Hematocrit 24-30
Treatment of Shock

- Surgery: immediate vs. delayed vs. none
- Establish airway and deliver O$_2$
- Insert 2 large bore IVs with relatively short length of tubing; infuse Normal saline or Lactated Ringer’s
- Treat mechanical causes of shock if they are present
  - Tension pneumothorax
  - Pericardial tamponade
  - Exsanguinating hemorrhage
Treatment of Shock

- While inserting IVs, draw blood for laboratories and for blood typing
- Relieve pain with IV narcotics
- Reassess
- Blood transfusion: think twice
- Vasopressors
- Antibiotics?
Treatment of Shock

- Maintenance IV fluids
- Inotropic support?
- Early removal of septic focus (i.e. dead bowel or large abscess) or other definitive surgery
Blood transfusions

- Good effects
  - Restores blood volume
  - Stays in vessels
  - Increases $O_2$ delivery to tissues
Blood Transfusions

- Bad effects
  - **Transfusion reactions**
    - Febrile & allergic: 1/100
    - Hemolytic
      - Fatal: 1/100,000
      - Non-fatal: 1/6000
  - **Transmission of disease**
    - Hep B: 1/200,000
    - Hep C: 1/103,000
    - HIV: 1/450,000
    - Malaria, Chagas, Yersinia: < 1/1,000,000
    - Overall 3/10,000
  - **Decreases immunity to cancer and infection**
Transfusion Algorithm

Injury

Hemodynamically unstable

Initial Assessment ABCs

Hemodynamically stable

Establish diagnosis and priorities

Crystalloid administration

Fails to Respond
HR > 120
BP < 90

Ongoing hemorrhage

Hemodynamically stable

Crossmatched blood or PRBC

Remains stable
HCT > 24%

Remains stable
HCT > 24%

Ongoing hemorrhage

Transfuse crossmatched blood

No transfusion

Responds

Remains stable
HCT > 24%

Remains stable
HCT > 24%

No transfusion

Responds transiently

Ongoing hemorrhage

Transfuse type-specific PRBC or Type O

Transfuse crossmatched blood
Pitfalls

- Failure to recognize occult hemorrhage
- Do not assume hypotension after trauma is due to head injury.
- Always perform a rectal examination.
- Inadequate resuscitation
Don’t forget…

- **Pregnancy** - Optimization of perfusion in the mother is the treatment of choice for the fetus.

- **Pediatric** - Compensatory mechanisms may be quite effective in children. Hypotension is a late finding and represents significant hemorrhage.

- **Geriatric** - Medications and underlying diseases may modify responses to cause or even therapy.