Objectives

- Define Shock
- Discuss basic physiology of shock.
- List the components of a shock state.
- Discuss and describe the categories of shock.
- Correlate possible management interventions with assessment findings.

Brief History

- First described symptoms in 1575
- 1743 first used term “Shock”
- WW I reduction of O2 to tissues, lactic acidosis
- Renal failure Korean War
Shock
A state of generalized cellular hypoperfusion leading to inadequate cellular oxygenation to meet metabolic needs

Basic Mechanisms
• Oxygen supply meets demand = aerobic metabolism, glucose metabolized
• Usually only 25% oxygen in capillaries utilized by tissues for aerobic metabolism
• Tissues can up this to 50-60% when stressed, up to a point, then...........

Basic Mechanisms
• Problem with anaerobic metabolism is less energy yield, lactate produced. Lactic acidosis ensues, as cant be cleared as fast as CO2 from tissues.
• Lactic Acid impairs at cell level then organ level
• Impaired organ means poor cellular perfusion
• Shock is a self perpetuating condition.
Metabolism

• All cells require energy to function
• Aerobic metabolism
  ▫ Oxygen is required for efficient production of the energy molecule ATP and converting pyruvate to carbon dioxide and water through the Kreb's cycle
• Anaerobic metabolism
  ▫ Inadequate oxygen results in decreased ATP (energy molecule) production and accumulation of lactic acid

Consequences

• Decreased ATP (energy) for cell membrane function
  ▫ Potassium and lactic acid enter the blood
    ▪ Low pH results in release of cellular enzymes that autodigest cells
    ▪ Cellular death, organ failure result
  ▫ Sodium and water enter the cell
    ▪ Cellular edema
    ▪ Further loss of intravascular (blood) volume

Pathophysiology of Shock

• The heart must be an effective pump
  \[ CO = SV \times HR \]
• Stroke volume depends on adequate return of blood to the heart
• If blood volume decreases, cardiac output will decrease unless the body alters the heart rate
Pathophysiology of Shock

• Hemodynamics
  ▫ Perfusion of the body tissues requires
    • An effective pump
    • An adequate volume of blood
    • Vascular resistance

• Adequate blood pressure is required for perfusion
• Cardiac output is one factor in maintaining blood pressure
  \[ BP = CO \times SVR \]
• Vasoconstriction occurs to increase systemic vascular resistance if cardiac output falls

Pathophysiology of Shock

• Microvascular changes
  ▫ Early: precapillary and postcapillary sphincters constrict causing ischemia
  ▫ As acidosis increases: precapillary sphincters relax but postcapillary sphincters remain constricted causing stagnation
  ▫ Finally: postcapillary sphincters relax causing washout, releasing microemboli and aggravating acidosis
Pathophysiology of Shock

- Vasoconstriction
  - Ischemic phase of shock

- Ischemic sensitivity
  - Brain: 4 to 6 minutes
    - Altered LOC occurs early
  - Organs: 45 to 90 minutes
    - Acute renal failure, ARDS
  - Skin and skeletal muscle: hours

Oxygen Delivery

- Amount of oxygen that a given volume of blood carried multiplied by the speed at which it is carried to the cell.
- Content of arterial O$_2$ is CaO$_2$
- CaO$_2$=1.34 x Hb x SaO$_2$ + (.003xPaO$_2$)
- Delivery: DO$_2$= CO x CaO$_2$
- CO= SV x HR
- SV is a consequence of preload, afterload, and contractility.

Cardiac Output

- Number of beats per minute multiplied by amount of blood per beat.
- A decrease in one necessitates an increase in the other.
- Tachycardia earliest and most effective
- Drops out at over 150 beats per minute
- Diastolic filling drops off with tachycardia
Preload

- Starling:1915. The stretch on myocardial fibers. Clinically its Left Ventricular End Diastolic Volume or LVEDV.
- An increase of LVEDV produces increased CO up to a point, then falls off. This is the flat part of the “curve.”
- At this point is failure and pulmonary edema

Starling Curve

Afterload

- Resistance against which the heart muscle contracts.
- SVR: systemic vascular resistance
- Clinically SVR measures afterload.
- \[ SVR = \frac{MAP - CVP}{CO} \times 80 \]
**Contractility**

- Force of contraction of the myocardium independent of preload and afterload.
- Ability of the heart to squeeze at a constant preload and afterload.
- Most important factor governing contractility is perfusion of the myocardium.

**Determinants of Oxygen Uptake**

- We measure Oxygen content in arteries going in cellular environment, measure it going out in the venous side. The difference is the uptake, or VO2.
- VO2 = CO x (CaO2 - CvO2)
- Arterial Sat (95-100%)
- Venous Sat (70-75%)
- So, normally we only utilize 20-25% of O2

**Oxygen Extraction**

- So, quite a bit of O2 left in reserve.
- I said before tissues can up it to 50-60% under stress.
- This is called oxygen extraction O2ER.
- O2ER = VO2/DO2 or consumption/delivery.
- Reaches a critical point as usual....
Shock Categories

- Hypovolemic
- Cardiogenic
- Distributive
  - neurogenic
  - septic
  - anaphylactic

Hypovolemic

- Reduction of intravascular volume and preload
- Bleeding, diarrhea, vomiting......
- Most common category and typically dealt with by surgeons.

Shock Classifications

<table>
<thead>
<tr>
<th></th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount of blood loss (% total blood volume)</td>
<td>&lt;700 mL (15%)</td>
<td>700-1500 mL (15%-30%)</td>
<td>1500-2000 mL (30%-40%)</td>
<td>&gt;2000 mL (40%-60%)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>Normal or minimally increased</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min)</td>
<td>Normal</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Greatly decreased</td>
</tr>
<tr>
<td>Urine output (mL/hr)</td>
<td>Normal</td>
<td>20-30</td>
<td>&lt;10</td>
<td>Minimal</td>
</tr>
</tbody>
</table>
Clinical Signs

Class I (15%): Like giving blood
Class II (15-30%): 750-1500cc. Tachycardic, orthostatic, decreased pulse pressure, cool clammy.
Class III (30-40%): 1500-2000cc. Hypotension, urine drop, anxiety, combative.
Class IV: >40%. >2000cc. Obtunded, oliguria, ready to code.

Compensatory Mechanisms

- Increase in sympathetic activity by receptors in atrium, aorta, carotid.
- Norepinephrine and Epinephrine released with increase in HR and contractility, increasing CO.
- PVR increased as well. Shunting.
- Skin, skeletal muscle, splanchnic circulation first, then kidneys.
- Keep brain and heart alive.

Compensations

- Renal perfusion drops off, renin kicks in, angiotensinogen to angiotensin1, to angiotensin 2.
- Angiotensin 2 increases vascular tone, reabsorption of Na, stimulates aldosterone release which also augments Na reabsorption.
- ADH release from Pituitary to retain water, vasoconstriction
Compensations

- Shift of fluid from extravascular space to intravascular space. Hematocrit falls.
- Mechanisms overwhelmed.
- Pre-capillary sphincters relax leading to increase in capillary hydrostatic pressure. Driving fluid back into interstitium.

Compensations

- Decrease in coronary perfusion pressure, myocardial ischemia.
- Acidosis leads to arteriolar vasodilatation, negative inotropic effects.
- If all this is not interrupted.....shock state is irreversible, leads to death.
- Lead to damage control surgery thinking.
Treatment

- ABCs
- Urine output 0.5ml/kg/hr
- Lactate measurements
- Base Deficit is a measure of number of millimoles of base necessary to correct the pH of a liter of whole blood to 7.40.
- If remains elevated, cellular perfusion poor.

Cardiogenic Shock

- Failure of the heart to function effectively as a pump to provide adequate blood flow.
- Intrinsic or extrinsic causes
- Low systolic BP

Cardiogenic Shock

- Before treating, must exclude hypovolemia. Once excluded, decide if it is level of heart muscle or in thoracic area.
- Most common cause is MI, with loss of 40% LV function. Leads to elevated preload, transmitted to lungs, pulmonary edema ensues.
Cardiogenic Shock

- Arrhythmias
- Tachycardia
- Reduced Diastole, decreases coronary perfusion
- Bradycardia
- MI also produces mechanical defects, ie valves, aneurysm, septal defects.

Cardiogenic Shock

- Extrinsic causes include tension pneumothorax, vena caval obstruction, pulmonary embolism, pericardial tamponade.

Treatment of Cardiogenic

- ABCs
- Augment low pressures with volume
- Hypotension with low SVR best treated with agent that increases contractions, and dilates periphery. Dobutamine.
Dobutamine

• Acts primarily on Beta 1 receptors in myocardium.
• Not many EMS agencies carry or use Dobutamine
• What do we have that can help?

Dopamine

• If continued hypotension, an inotrope that tightens periphery up is needed.
• Low doses, renals dilate
• 5-10ug/kg/min stimulates Beta 1 (inotrope)
• Above 10ug/kg/min Alpha stimulation with increase in SVR.
• Watch for tachycardia however.

Distributive Shock

• Condition where SVR drops to the point that regardless of CO, the periphery is underperfused.
• Neurogenic, Septic, Anaphylactic are the common ones.
**Neurogenic Shock**

- Interruption of sympathetic tracts responsible for vascular tone. Loss of vasoconstriction results in blood pooling, decreased venous return, decreased ventricular filling.
- Peripheral pulses weak, cap refill brisk, UO adequate, bradycardia from unopposed vagal stimulation

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**Septic Shock**

- Body’s response to an overwhelming infection.
- Bacterial wall components like endotoxin released from G- bacteria.
- Inflammatory mediators are activated, which result in hemodynamic derangements.

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**Changes in Septic Shock**

- Hyperdynamic in early stages, hypodynamic in late stages.
- Hyperdynamic stage marked by low SVR, increased CO, warm extremities.
- Eventually leads to pooling, leakage, poor perfusion.
- Myocardial depression, hypodynamic state
Changes in Septic Shock

- Hallmark is inefficient extraction of O2, or utilization of O2 from microcirculation.
- Anaphylactic shock presents similar to septic, low SVR, capillary leakage.

Treatment of Distributive

- ABCs
  - Fill the tank first. This is often all that is necessary.
  - If not, then use a vasopressor such as Dopamine
  - Usually not needed for a long time.
  - Anaphylaxis treated with antihistamines, epinephrine in addition to volume.

The Inflammatory Response

- Death from traumatic injury follows a trimodal distribution.
  - 1st subset die at scene
  - 2nd within 6hr from unsuccessful resuscitation, brain injuries, PTX not treated.
  - 3rd within 30 days from MOF
Inflammatory Response

- The first two have decreased greatly.
- The third has not. The insult is a body wide inflammatory state, later coined SIRS.
- At low levels SIRS is helpful, unchecked it leads to MOF.

Multiorgan Failure or MOF

- Vasoconstriction moves blood to vital organs.
- GI tract can become ischemic, followed by reperfusion which causes cell injury.
- Breakdown of gut mucosal injury, mucosa becomes permeable in the septic state.
- All a theory of course.
- Two hit phenomena as another explanation

Signs of Shock

- Tachypnea
  - Hypoxia and acidosis stimulate the respiratory center
  - 20 to 30 breaths per minute
  - More than 30 breaths per minute
  - Intolerance of oxygen face mask
Signs of Shock

• Circulation
  ▫ Assessment for hemorrhage
  ▫ Level of consciousness
  ▫ Heart rate
  ▫ Pulse
  ▫ Skin color and temperature
  ▫ Capillary refill
  ▫ Blood pressure

Signs of Shock

• Disability
  ▫ Decreased cerebral perfusion results in altered LOC
  ▫ Other causes of altered LOC will not kill the patient as rapidly as shock
  ▫ Assume altered LOC is due to shock and treat

Decision Making

• What factors may affect a patient’s presentation in shock?
  ▫ Pregnancy
  ▫ Medications
  ▫ Age
  ▫ Preexisting medical conditions
Shock Management

• Four questions guide resuscitation
  ▪ What is the cause of shock in this patient?
  ▪ What is the care of this type of shock?
  ▪ Where can the patient get this care?
  ▪ What can be done between now and the time the patient reaches definitive care?

Fluid Therapy

• Current recommended practice
  ▪ Classes II, III, and IV shock
  ▪ Initial rapid bolus of 1000 to 2000 mL of warmed fluids
  ▪ Maintain systolic BP at 85 to 90 mm Hg

Complications of Shock

• Untreated, shock progresses
• Prehospital care can make a difference in the patient’s eventual outcome
  ▪ Acute renal failure
  ▪ Acute respiratory distress syndrome
  ▪ Hematologic failure
  ▪ Multiple organ dysfunction syndrome
Minimizing Complications

- Assess for shock
- Remember: cardiac output and tissue oxygenation are impaired early
- Restore/maintain: airway, ventilation, oxygenation, circulation
- Hypothermia creates a cycle of worsening shock and hypothermia
- Transport without delay

Contact Information

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