



## CrackCast Episode 6 – Shock

### Episode overview:

- 1) List, define and explain the 5 causes of shock
- 2) What is the utility of lactate and base deficit in the management of shock?
- 3) Define: SIRS, Sepsis, Severe Sepsis, and Septic Shock
- 4) List 5 empirical criteria for the diagnosis of circulatory shock
- 5) Describe Early Goal Directed Therapy
- 6) Describe the management goals in cardiogenic shock

**Wisecracks:** A stepwise approach to cardiogenic shock

### Rosen's in Perspective: What is SHOCK?

- “a transition between life and death”
- mitochondria are first to be affected in shock
  - The “canaries in the coalmine”
- Widespread failure of the circulatory system to oxygenate and nourish the body leading to systemic inflammation, organ dysfunction, and death

### 1) List, define and explain the 5 causes of shock

- Hypovolemic
- Obstructive
- Distributive
- Cardiogenic
- Cellular Toxins

### Hypovolemic Shock

Various Causes: hemorrhage, GI losses, third spacing (burns, albumin deficient space, septic shock)

A strict definition of hemorrhagic shock can be debated

- 1) It can be esoterically classified by: simple hemorrhage causing hypovolemia vs. hemorrhage with hypo-perfusion vs. hemorrhagic shock
- 2) Simply put hemorrhagic shock can be “suspected bleeding with at least 4 signs of shock”
- 3) Rosen's also mentions hypovolemic shock can be placed into various stages: early, compensated, uncompensated but these are not clinically useful



- Clinically, the body's response with HR and BP to volume loss are variable depending on age, medications, co-morbid conditions etc.
- Rosen's wants us to think through how hypovolemia leads to two phases of organ injury
  - 1) First it decreases perfusion to non-critical organs, and manifests as
    - a) acidemia
    - b) declining base deficit (more negative)
    - c) increased alveolar ventilation
  - After 1/3 of total blood volume is lost, cardiovascular reflexes can no longer adapt, leading to hypotension and then activation of the H-P-A axis and stress hormone release, stimulating glycogenolysis, lipolysis, and hypokalemia in an effort to bolster SVR and perfusion of vital organs
  - 2) The second phase of organ injury is induced by resuscitation!!!
    - Resuscitation can lead to liver injury, ARDS, ATN, so just because they have been resuscitated from severe hypovolemic shock, doesn't mean they are out of the woods!!

## **Obstructive Shock**

Obstructive shock can be defined as shock resulting from any physical obstruction that limits cardiovascular flow

All main causes are intrathoracic:

- a) pulmonary embolism
- b) cardiac tamponade
- c) tension pneumothorax
- d) valvular dysfunction
  - 1) critical aortic stenosis
  - 2) acute thrombosis of a prosthetic valve
- e) congenital heart defects (eg. closure of PDA)
- f) critical idiopathic subaortic stenosis (HOCM)
- g) air embolism

## **Distributive Shock**

Distributive shock has 5 main subtypes:

- 1) septic shock
- 2) anaphylactic shock



- 3) central neurogenic shock
- 4) drug overdose
- 5) adrenal crisis

### Septic Shock:

Most commonly caused by a bacterial organism, although only 50% of the time an organism is found

- The LPS of gram negative, or other gram positive bacteria are most common causes
- Thought to be responsible for three major systemic effects:
  - 1) Relative hypovolemia
    - a. increased venous capacitance, which leads to decreased RV filling (and thus decreased CO)
    - b. GI, skin losses, and capillary leak (third spacing)
    - c. Impaired thirst response in brain
  - 2) Cardiovascular depression
    - a. Impaired cardiac mechanical function by cytokines and direct injury to heart muscles by cellular toxins
  - 3) Induction of systemic inflammation
    - a. Leads to ARDS, renal dysfunction, MODS etc.

We will visit the consensus definition in question #3!!

### Anaphylactic Shock

- a type I hypersensitivity reaction to an antigen, which is often life threatening if not treated urgently (SEVERE form of an allergic reaction)

### Central Neurogenic Shock

- this is NOT the same as spinal shock (more to come in future episodes)
- usually a diagnosis of EXCLUSION
  - 1) caused by an injury to the cervical or thoracic vertebrae causing peripheral sympathetic denervation
  - 2) look for the **classic TRIAD**: hypotension, bradycardia, and warm extremities

### Drug Overdose

- Due to a sympatholytic or sedative drug
  - Eg. clonidine, TCA, mixed narcotic/benzo



## Adrenal Crisis

- acute mineralocorticoid deficiency – usually precipitated by something that increases body's demand for steroids
  - autoimmune, adrenal hemorrhage, infectious etc.

You can use the acronym “S.N.ACK. D.AD.” for distributed shock if that helps!!

## Cardiogenic Shock

Case Definition:

- 1) Cardiac failure (impaired forward blood flow of the heart) causing systemic hypoperfusion/shock
  - a. results when more than >40% of the myocardium is injured (direct or not)
  - b. more common in patients with underlying cardiac disease

Causes:

- a) ACS (main cause)
- b) Cardiomyopathy/myocarditis/chronic heart diseases
- c) Cardiac dysrhythmias
- d) Negative inotropic drug ingestion
- e) Structural heart damage: flail mitral valve, septal wall rupture, papillary muscle rupture.

## Cellular Toxins

Causes and antidotes:

- a) Carbon Monoxide – (oxygen)
- b) Cyanide – (hydroxocobalamin)
- c) Methemoglobinemia – (IV methylene blue)
- d) Hydrogen sulfide – (oxygen/supportive/?sodium nitrite)

## 2) What is the utility of lactate and base deficit in the management of shock?

Lactate and Base Deficit are useful screening tools and monitoring parameters in a “shocky” patient

We know how patients look when they are in shock:

- The stress response makes people look ill, asthenic, pale, sweaty, and mentally altered



- Patient monitors may show hypotension, tachycardia, grunting, tachypnea, decreased urine output (<1mg/kg/hr), and mentation changes

**But** the lactate and base deficit can give us a sneak peak (“canary in the coalmine”) into what is happening at the level of the mitochondria

What do the base deficit and lactate actually measure?

- 1) Base deficit: defines the metabolic component of the body’s acid-base status and is normally -2 to +2. In simple terms it shows how much of the bicarbonate buffer system has been used up.
  - a. The more acidotic, the more negative the base deficit will be
  - b. NOTE some people choose to look at “base excess” instead of base deficit
- 2) Lactate: a by-product of anaerobic glucose metabolism and it may be elevated in both hypoxic conditions, as well as non-hypoxic conditions (catecholamines).
  - a. Lactate can help us assess how well we are resuscitating patients, although it is not a perfect test

What values are helpful for us to know?

Well anyone with a base deficit <-4 or a lactate >4, should make us worried!

### 3) Define: SIRS, Sepsis, Severe Sepsis, and Septic Shock

**SIRS – systemic inflammatory response syndrome:**

- 2 or more of:
  1. Temp < 36<sup>0</sup>C or > 38.3<sup>0</sup>C
  2. HR > 90
  3. RR > 20 or CO<sub>2</sub> < 32 (resp. alkalosis)
  4. WBC < 4k or > 12k

**Sepsis:**

- SIRS plus a documented or suspected infection

**Severe Sepsis:**

- Sepsis and 1 or MORE signs of organ dysfunction
  - Eg. high lactate, new elevated creatinine, low urine output, hepatic dysfunction, bone marrow dysfunction etc.

**Septic SHOCK**

- Severe sepsis + tissue hypoperfusion despite “adequate” fluid challenge
  - Eg. hypotension, tachycardia, low cardiac output, mottled skin, delayed cap refill, AMS, high lactate DESPITE adequate fluid challenge (usually 2L minimum).



#### 4) List 5 empirical criteria for the diagnosis of circulatory shock

ie. how can we categorize someone as being “in shock”

- \*\*\*Empirical criteria for the diagnosis of circulatory shock (>4/6)\*\*\*
  - 1) ill appearance or AMS
  - 2) HR >100 bpm
  - 3) RR >20 or PaCO<sub>2</sub> <32
  - 4) Base deficit <-4 or >4 lactate
  - 5) Urine output <0.5ml/kg/hr
  - 6) Arterial hypotension >30 min continuous duration

#### 5) Describe Early Goal Directed Therapy

- There is significant controversy over physiological targets for managing shock
- Surviving Sepsis Campaign Guidelines (and Rosen’s) use the following targets as “what should be achieved during the first 6 hours of resuscitation in sepsis”
  - a. MAP > 65 mmHg (MAP = ((2 x diastolic + systolic) / 3)
  - b. Urine output > 0.5 mL/kg/hr
  - c. CVP 8-12 or respiratory changes in the radial artery pulse pressure
  - d. central venous O<sub>2</sub> > 70% or mixed venous oxyhemoglobin saturation > 65%

\*\*\*\*side comment\*\*\*\*

- the first two goals are the best, but for the sake of the exam you should know the last two
- some people tack on the goal of normalizing the lactate in the first six hours
- Although as new trials emerge we are modifying and adapting Dr. River’s original early goal directed therapy protocol, it is useful to remember that his work redefined how we approach and manage septic shock!

#### 6) Describe the management goals in cardiogenic shock

- Cardiogenic shock is a difficult entity for us to manage in the ER because there’s not a lot we can do to directly fix the problem.
- Usually it’s a result of direct myocardial injury - i.e. myocardial infarction
- As a result the management goals centre around:
  - 1) Minimizing myocardial oxygen demand (which would cause more stress on the heart muscle)
  - 2) Maximizing cardiac output

#### WiseCracks: Our stepwise approach to cardiogenic shock

1. “Fill the tank” - goal MAP >65



- a. fluid boluses to optimize preload
2. Gently stimulate the heart
  - a. Dobutamine
    - i. 2.5 mcg/kg/min (starting dose)
3. “Squeeze the pipes”
  - a. Norepinephrine
4. Intubate - **\*\*carefully\*\***
  - we want to decrease body’s O<sub>2</sub> demand while not dropping preload too much!
5. Determine the cause if it’s not an MI and treat it
  - a. ?myocarditis ?cardiomyopathy
  - b. ?papillary muscle rupture leading to acute mitral regurgitation
  - c. ?cardiac drug overdose
6. Get the patient enroute to the place that can fix their heart (i.e. cath lab, heart transplant, ECMO center)