

Medical Practice Improvement

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Shock II

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Disclosures

- I have no disclosures



Learning Objectives

1. Treatment of shock

2. Recognizing different types of shock

3. Learning to differentiate the different physiology of shock



Oxygen Delivery

- $DO_2 = Q(1.33(Hgb_{(g)})(SaO_2) + (.003PaO_2))$
 - $Q = \text{cardiac out put} = HR \times SV$
 - $CaO_2 = \text{Oxygen Carrying Capacity of Blood} = 1.33(Hgb_{(g)})(SaO_2) + (.003PaO_2)$
- What does this mean?
- How can I use this to fix shock?
- Why am I even listening to lectures?



Oxygen Carrying Capacity of Blood

- HgB is in grams
- SaO₂ in percent
- Normal range about 20.1 mL O₂ / dL blood

Total Oxygen Content Calculation

The total oxygen content (CaO₂) in the blood is calculated by summing the oxygen bound to hemoglobin and the small amount of oxygen dissolved in plasma:

$$CaO_2 = (Hb \times 1.34 \times SaO_2) + (0.003 \times P aO_2)$$



Breaking it down by Piece

- Q is the cardiac out put
 - Heart rate x stroke volume
 - Evaluate pump function
 - May need inotropes
 - May need balloon pump
 - May need impella
 - May need LVAD
 - Cardiac transplant/death



Breaking it down by Piece

- $(1.33(\text{Hgb}_{(g)})(\text{SaO}_2))$
 - Biggest factor to the oxygen carrying capacity is Hgb!
 - Followed by SaO_2
 - SaO_2 is improved by ventilator (See VENT I and II Lectures)
 - Flo- Trac or vigileo can help with volume status



Dissolved Oxygen Component

- (.003PaO₂)
 - Amount of Oxygen dissolved in the Blood
 - At 1 atm and standard temperature =negligible effect
- Fun Fact
 - At 3 atm and 100% fio2 the serum can oxygenate the body



SCV02

What is it?

Central Venous Oxygen Saturation

SCV02, or **Central Venous Oxygen Saturation**, is a measure of the oxygen saturation in venous blood returning to the heart, specifically from the head and upper body. It is typically **2-3% lower than SvO2 (mixed venous oxygen saturation)**, as it reflects the oxygen content from these areas, which generally consume less oxygen than the lower body and organs. SCV02 is often used in critical care to assess oxygen delivery and consumption balance, providing a surrogate measure of oxygen flux.

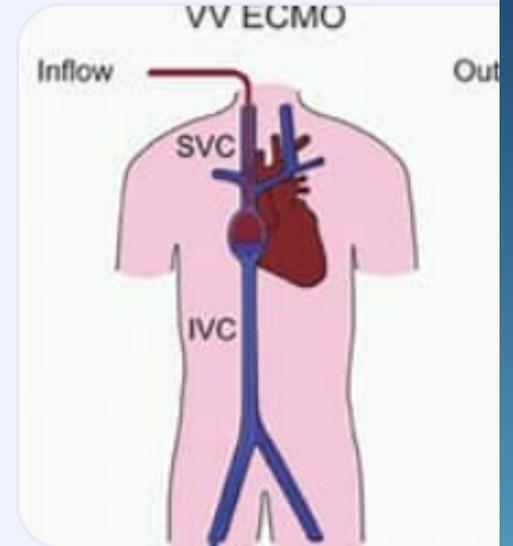


SV02

To obtain **ScVO₂ (Central Venous Oxygen Saturation)**, you typically need to use a **central venous catheter**. Here are the steps:

- **Insert a central venous catheter (CVC)** into a central vein, such as the internal jugular, subclavian, or axillary vein. ↻ 2
- **Measure the ScVO₂** using a device that reads the oxygen saturation of the blood returning from the right side of the heart. ↻ 2
- **Monitor the ScVO₂** to assess oxygen extraction and delivery in critical care settings. ↻ 2

This measurement is crucial for evaluating oxygenation status in patients, especially in critical illness or shock states.



O₂ Consumption

O₂ CONSUMPTION

- The amount of oxygen extracted by the peripheral tissues during the period of one minute is called oxygen consumption or VO₂.
- (N- 200-300ml/min)
- **$VO_2 = Q \times (CaO_2 - CvO_2) \times 10$**
- **$VO_2 = Q \times 1.34 \times Hb \times (SaO_2 - SvO_2) \times 10$**
- O₂ consumption is commonly indexed by the patients body surface area (BSA) and calculated by:
 - VO₂ / BSA
 - Normal VO₂ index is between 110-160ml/min/m².



Distributive Shock

Distributive shock, also known as vasodilatory shock, is one of the four broad classifications of disorders that cause inadequate tissue perfusion.

Systemic vasodilation leads to decreased blood flow to the brain, heart, and kidneys causing damage to vital organs

There are three subtypes are

- Septic,
- Anaphylactic/anaphylactoid,
- Neurogenic shock.

The most common etiology of distributive shock is sepsis.



Distributive Shock

Distributive shock typically presents with:

- tachypnea
- tachycardia
- hypotension.
- Other symptoms include an altered mental status, shortness of breath, cough and fever.

In anaphylactic shock low blood pressure is related to decreased systemic vascular resistance (SVR).

- triggered primarily by a massive release of histamine by mast cells activated by antigen-bound immunoglobulin E
- Increased production and release of prostaglandins.



Distributive Shock

Patient present with low

Low SVR

High CO

Low BP

Treatment is volume first then

- 1st line
 - Levophed(Norepinephrine)
 - Vasopressin (0.04units/min)
- 2nd line
 - Epinephrine
 - Angiotensin 2 (in the face of renal failure)
 - Neosynephrine
- 3rd line
 - Dopamine

Refractory Shock → consider steroids, T4, methylene blue



Distributive Shock

- Treat underlying cause
 - Infection → source control and abx
 - Anaphylaxis → remove allergen and epi and volume, steroids, antihistamines
 - Neurogenic → volume, rule out hemorrhagic shock, then pressers



Distributive Shock

- Methylene blue (MB) is used as a **rescue therapy in refractory septic shock**, particularly when profound vasoplegia persists despite adequate fluid resuscitation and high-dose vasopressors. Its mechanism targets the **nitric oxide (NO)–cyclic GMP pathway**, which is central to sepsis-induced vasodilation.
- **Why It Works When Vasopressors Fail**
- In severe septic shock:
 - Vascular smooth muscle becomes refractory to catecholamines
 - Excess NO signaling overwhelms α -adrenergic vasoconstriction
 - Simply increasing norepinephrine may be ineffective
- Methylene blue:
 - Blocks the downstream NO pathway
 - Restores the effectiveness of norepinephrine and vasopressin



Methylene Blue

- **Important Risks**
 - Serotonin syndrome (if on SSRIs, SNRIs, linezolid)
 - Methemoglobinemia (at high doses)
 - Hemolysis in G6PD deficiency
 - Pulmonary vasoconstriction (caution in ARDS)
 - Interference with pulse oximetry
-
- **Typical Dosing:**
 - 1–2 mg/kg IV over 20–30 minutes
 - Sometimes followed by infusion (0.25–1 mg/kg/hr)
 - Used early in vasoplegia appears more effective than late salvage therapy.



Distributive Shock

- Lab tests
 - CBC
 - CMP
 - Lactate
 - UA
 - Chest X-ray
- Treat underlying cause
 - Infection → source control and abx
 - Anaphylaxis → remove allergen and epi and volume, steroids, antihistamines
 - Endocrine → volume, hydrocortisone 100mg IV q8 hours or 50 mg q 6 hours

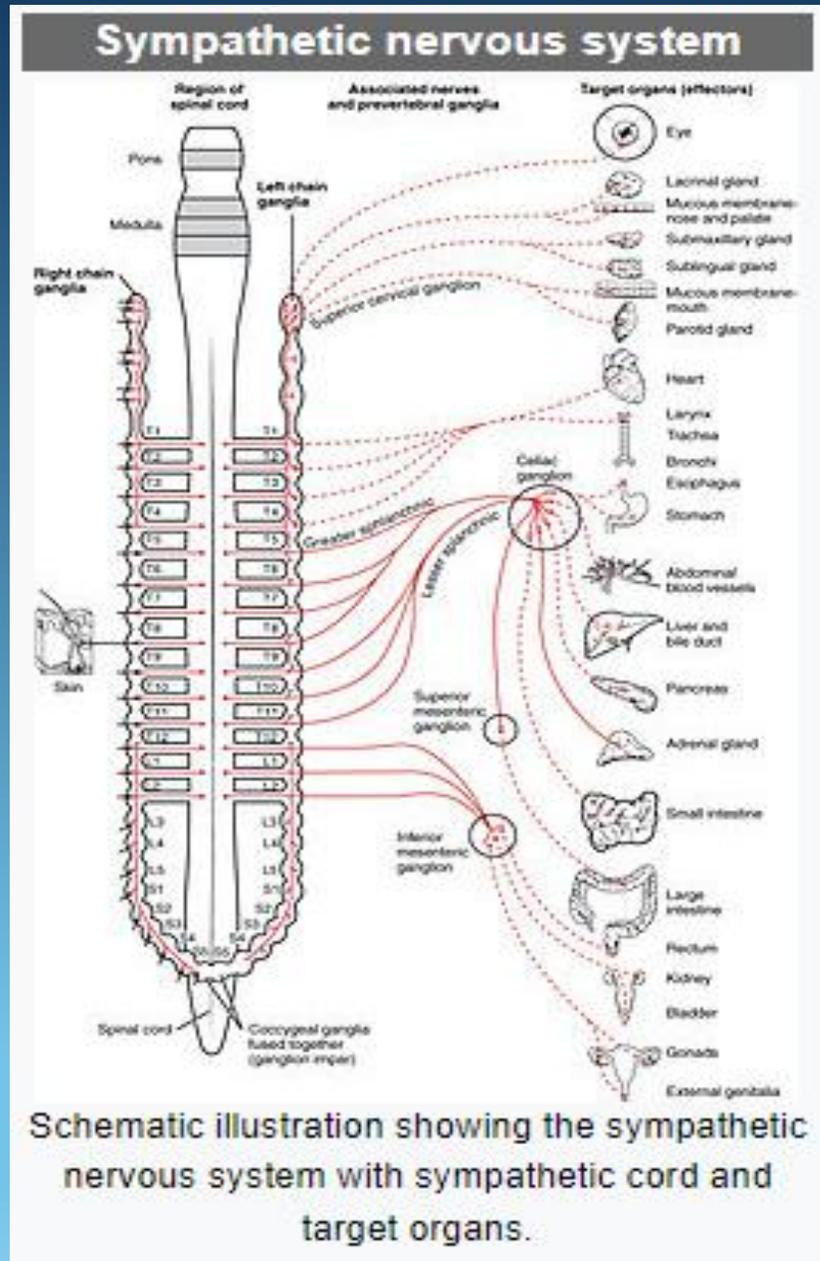


Neurogenic Shock

- Neurogenic Shock is caused by the loss of vascular tone normally supported by the sympathetic nervous system due to injury to the central nervous system especially spinal cord injury above the level of T4.



Neurogenic Shock



Neurogenic Shock

- Patient will be bradycardic
- Hypotensive
- Peripherally warm
- Low CO
- Low SVR
- In trauma hemorrhagic shock until proven otherwise
- Treatment is alpha and Beta agonist
 - Dopamine
 - Norepinephrine
 - Epinephrine
 - Not neosyneprhine all alpha and will cause worsening bradycardia



Hypovolemic Shock

- Hypovolemic shock is an emergency condition in which severe blood or other fluid loss makes the heart unable to pump enough blood to the body.



Hypovolemic Shock

- Symptoms include the following:
 - Marked tachycardia,
 - Decreased systolic BP,
 - Narrowed pulse pressure (or immeasurable diastolic pressure),
 - Decreased urinary output,
 - Depressed mental status (or loss of consciousness),
 - Cold and pale skin.



Hypovolemic Shock

- A narrow pulse pressure in a hypovolemic shock patient indicates a decreasing cardiac output and an increasing peripheral vascular resistance
- In trauma, a low or narrow pulse pressure suggests significant blood loss (insufficient preload leading to reduced cardiac output)



Hypovolemic Shock

- The top number (systolic) minus the bottom number (diastolic) is the pulse pressure. For example, if the resting blood pressure is 120/80 millimeters of mercury (mm Hg), the pulse pressure is 40 — which is considered a healthy pulse pressure
- Pulse pressure less than 30mmHg shows significant vasoconstriction
- Pulse pressure 20 mmHG or less is peri arrest in trauma
 - If you attempt to intubate without resuscitation the patient will code.



Hypovolemic Shock

Factors	I	II	III	IV
Blood loss	<15% (<750ml)	15-30% (750-1500ml)	30-40% (1500-2000ml)	>40% (>2000ml)
Pulse	>100	>100	>120	>140
B.P.	Normal	Normal	↓	↓↓
Pulse pressure	N or ↓	↓	↓↓	↓↓
Capillary refill	<2s	2-3s	3-4s	>5s
Resp. rate	14-20	20-30	30-40	>40
Urine output ml/hr	30 or more	20-30	5-10	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious & confused	Confused Lethargic



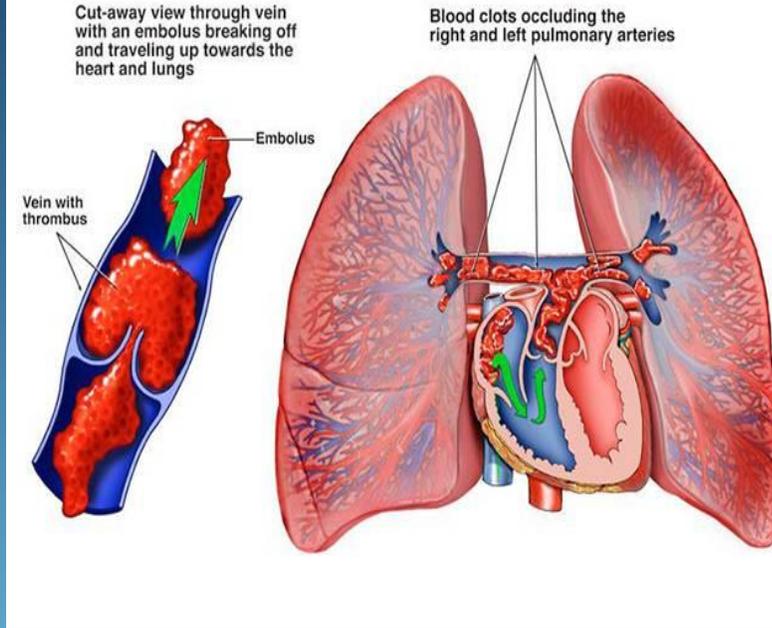
Hypovolemic Shock

- Treatment
 - Stop bleeding
 - Give fluid if dehydration
 - Give blood products if bleeding
 - Pressors as necessary
- Remember patient do not do better if they code!
- Shock III
 - → evolution of data collected, devices for goal directed therapy

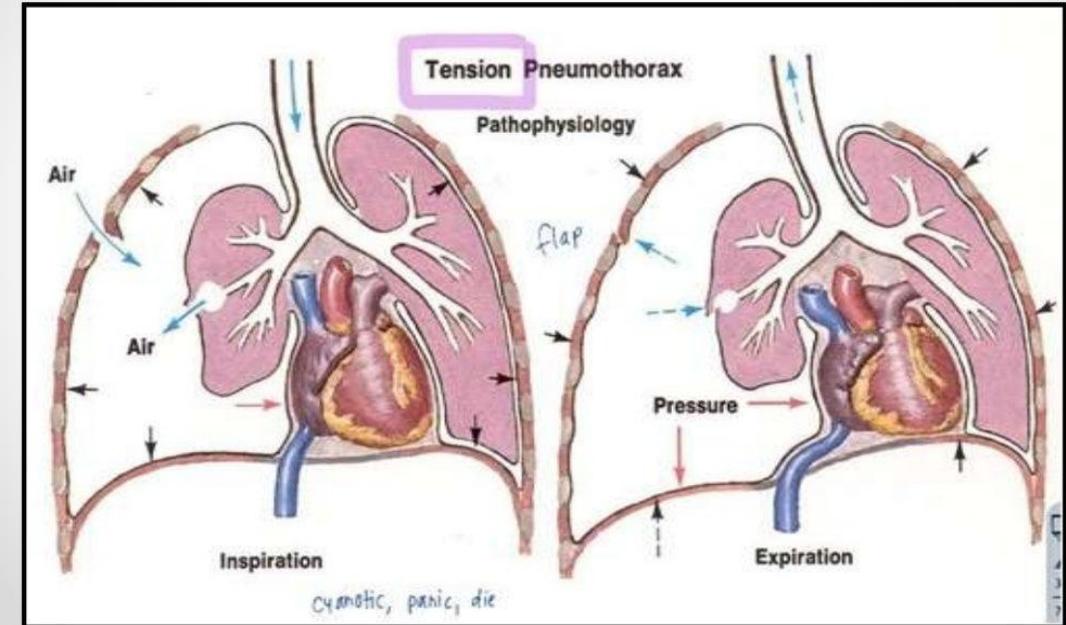


Obstructive Shock

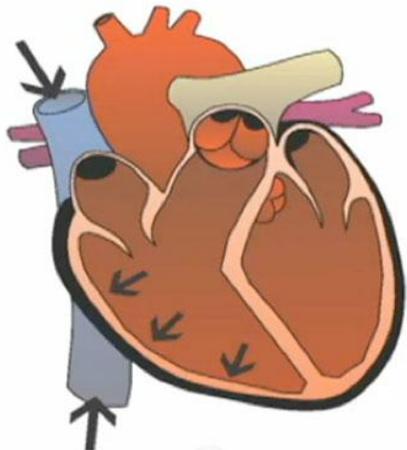
Pulmonary Embolism



Tension Pneumothorax



Healthy



Tamponade



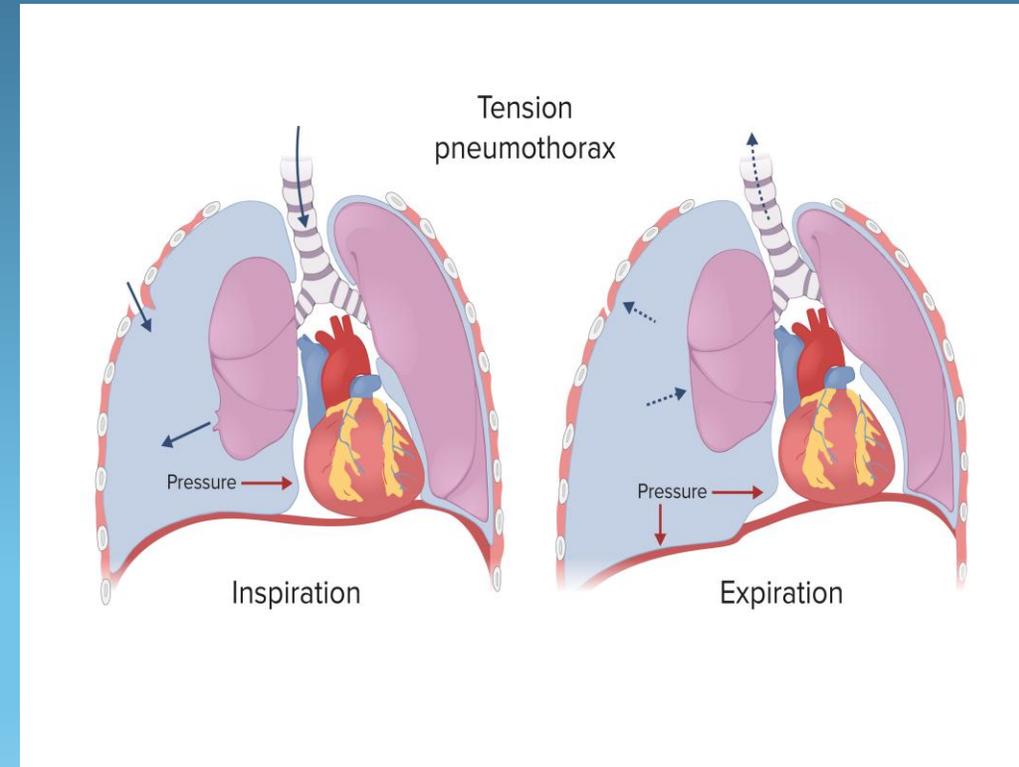
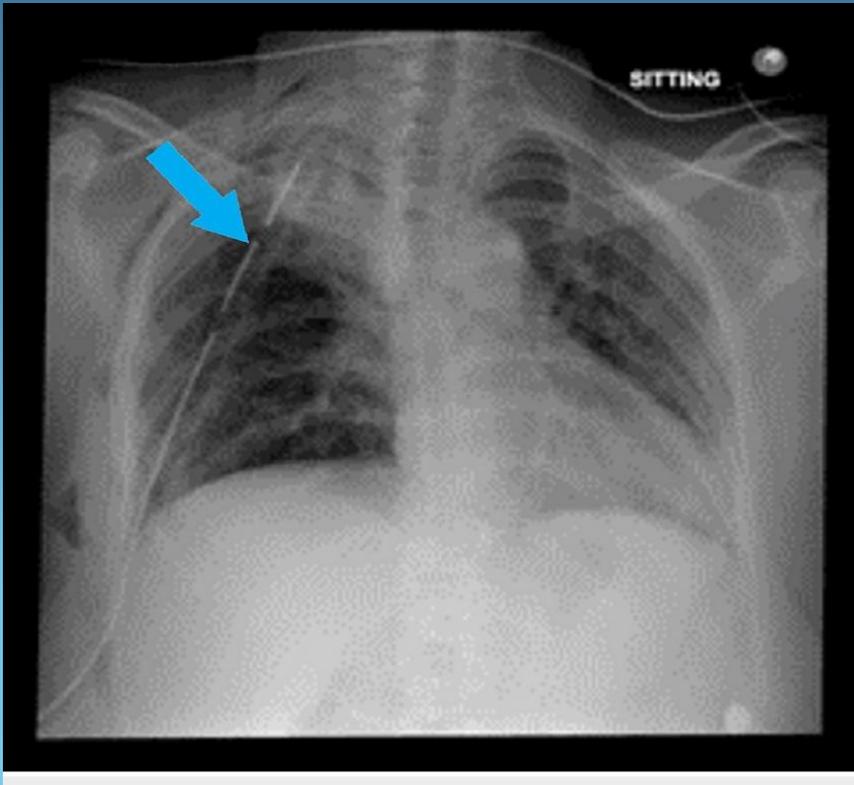
Obstructive Shock

- Causes
 - Tension PTX
 - Cardiac tamponade
 - Pulmonary embolism

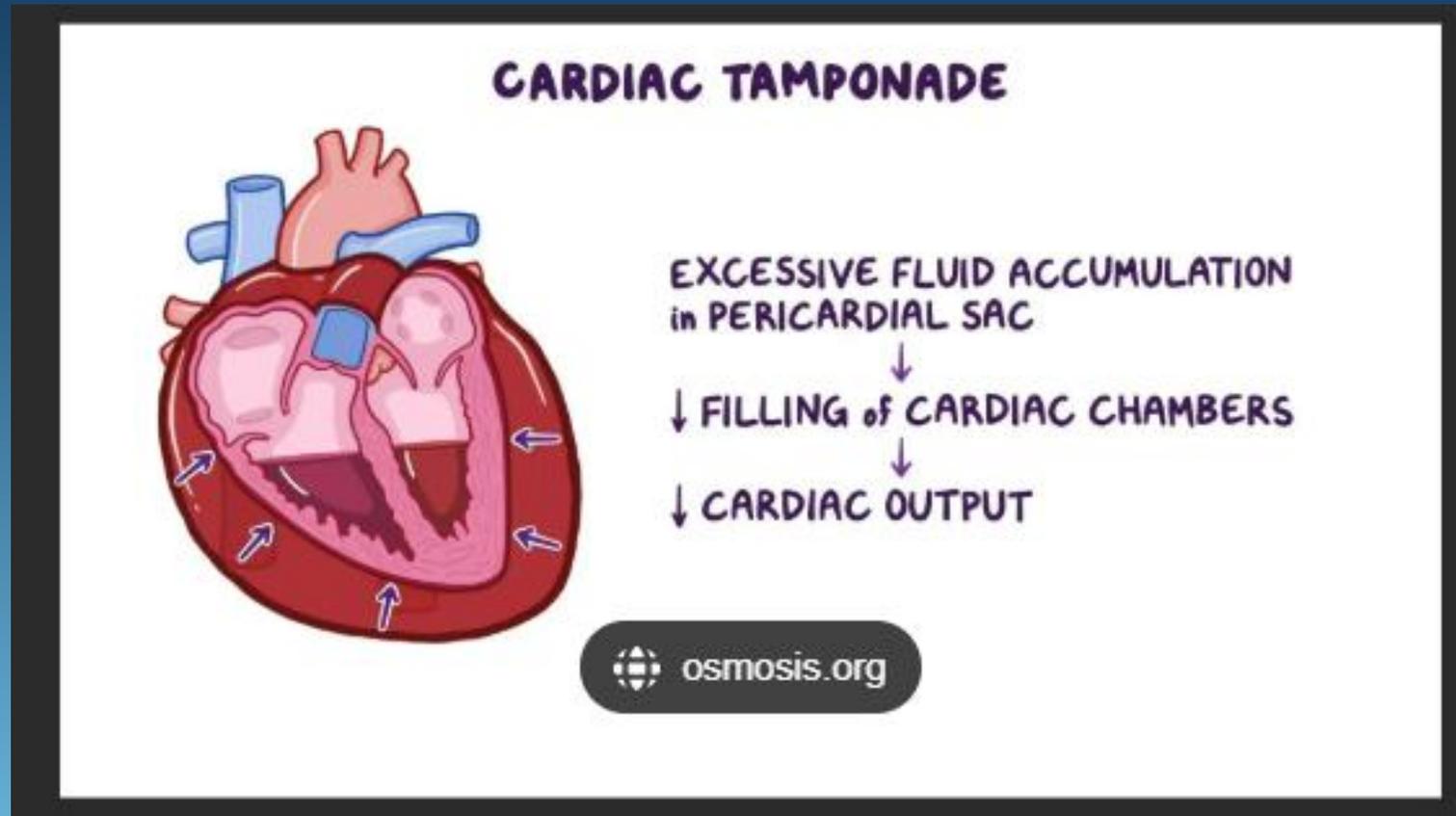


Treatment

- Tension Pneumothorax
 - Decompression of chest
 - Needle decompression
 - Chest tube



Cardiac Tamponade



needle compression
pericardial window

Decompression of
pericardium



Pulmonary Embolism

- Clot busters
 - TNK → fibrinolytic → breaks down clot
 - Pulmonary embolectomy
 - Heparin helps to prevent more clot formation
 - Heparin does not break down clots



Cardiogenic Shock

Cardiogenic Shock: Definition & Pathophysiology

- Definition: End-organ hypoperfusion due to primary cardiac pump failure
- Hemodynamics:
 - SBP <90 mmHg or MAP <65 mmHg
 - Cardiac Index <2.2 L/min/m²
 - PCWP >15 mmHg
- Clinical Findings:
 - Hypotension, oliguria, altered mentation
 - Cool extremities, pulmonary edema
- Pathophysiology:
 - ↓ Contractility or mechanical failure → ↓ CO
 - ↑ filling pressures
 - Neurohormonal activation (SNS, RAAS)



Cardiogenic Shock

Differential Diagnosis & Diagnostic Workup

Common Etiologies (4 Major Categories):

- Acute MI (most common)
- Acute decompensated heart failure / cardiomyopathy
- Mechanical complications (VSD, papillary muscle rupture, tamponade)
- Malignant arrhythmias

Initial Diagnostic Evaluation:

- ECG (STEMI/NSTEMI, arrhythmias)
- Bedside Echocardiogram (EF, wall motion, tamponade)
- CXR (pulmonary edema)

• Hemodynamic monitoring (arterial line, PA)



Cardiogenic Shock

Laboratory Evaluation

Cardiac & Perfusion Markers:

- Troponin I/T
- BNP or NT-proBNP
- Serum lactate (trend)

Metabolic & Organ Function:

- CMP (renal, hepatic function)
- ABG/VBG
- CBC
- Coagulation panel (PT/INR, PTT)
- Type & screen

Additional (as indicated):

- TSH



Cardiogenic Shock

Treatment Strategy: Stepwise Escalation

1. Chemical Augmentation (Inotropes/Vasopressors):
 - Norepinephrine (first-line vasopressor)
 - Dobutamine (inotrope for low CO)
 - Epinephrine or milrinone (selected cases)
2. Mechanical Circulatory Support:
 - IABP (afterload reduction)
 - Impella (LV unloading)
 - VA-ECMO (biventricular support)
3. Treat Underlying Cause:
 - Emergent PCI for STEMI
 - Surgical repair (VSD, papillary rupture)



Work Cited

- [Tension pneumothorax - Search Images](#)
- [chest x ray with chest tube - Search Images](#)
- [cardiac tamponade decompression - Search Images](#)
- [where to obtain the scvo2 – Search](#)
- [How to calculate the total oxygen content of arterial blood | Medmastery](#)



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