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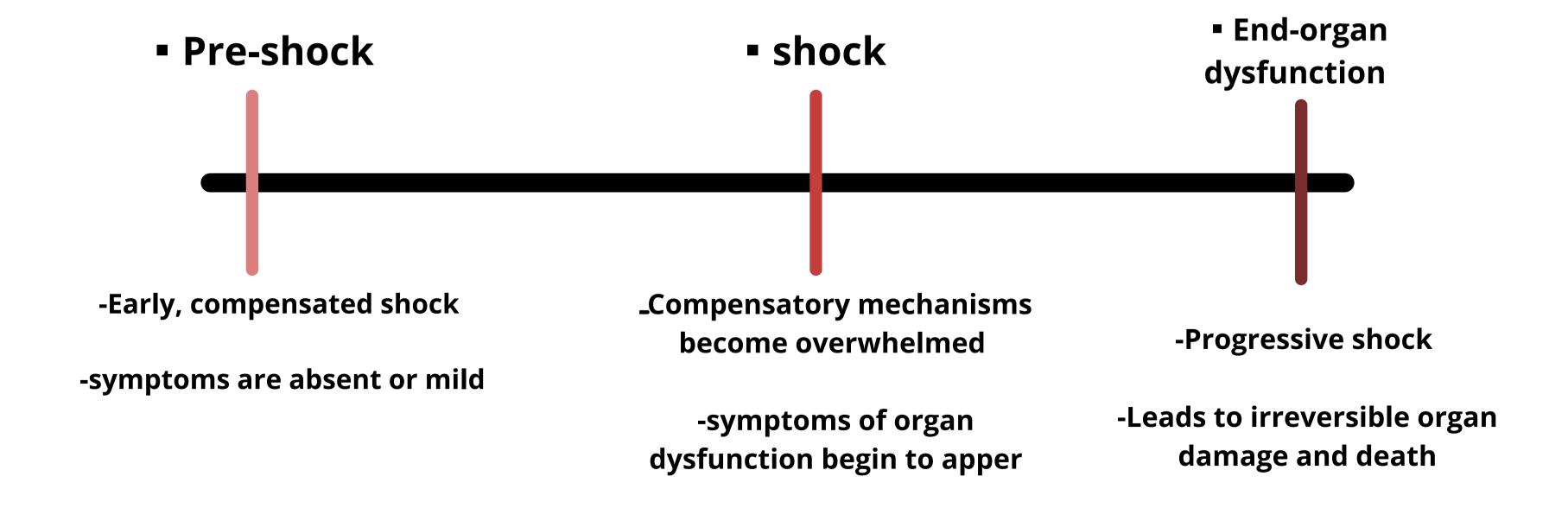
4 TYPES

Defnetion

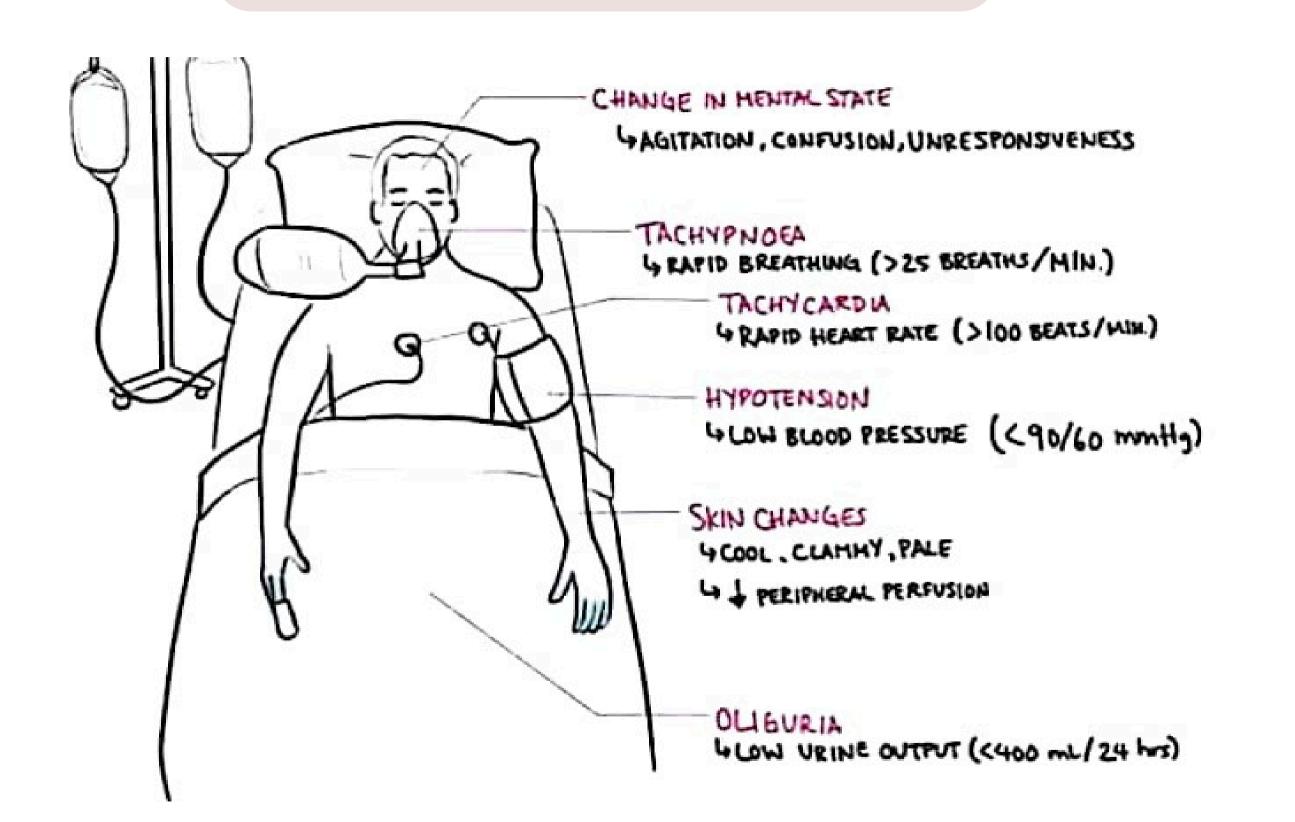
- Shock is a life-threatening manifestation of circulatory failure that leads to cellular and tissue hypoxia resulting in cellular death and dysfunction of vital organs.
- The effects of shock are initially reversible, but rapidly become irreversible, resulting in multi-organ failure (MOF) and death.



STAGES OF SHOKE



Clinical features



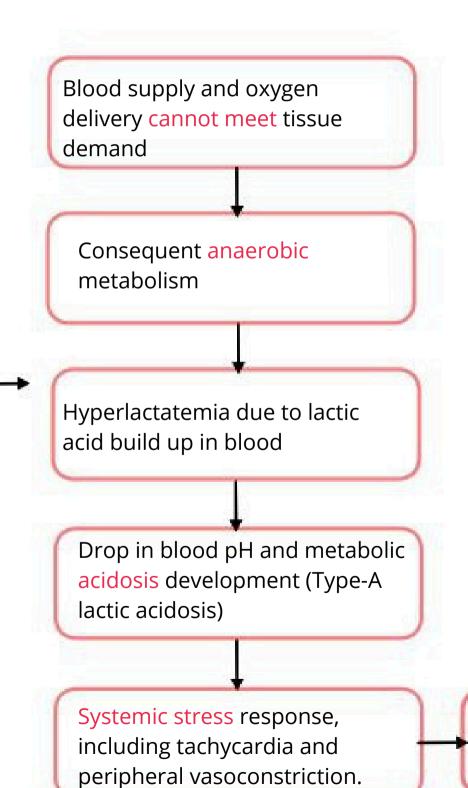
Pathophysiology

Decreased Intravascular volume
Decreased Myocardial function
Increased Systemic vascular resistance
Changes in distribution of blood flow

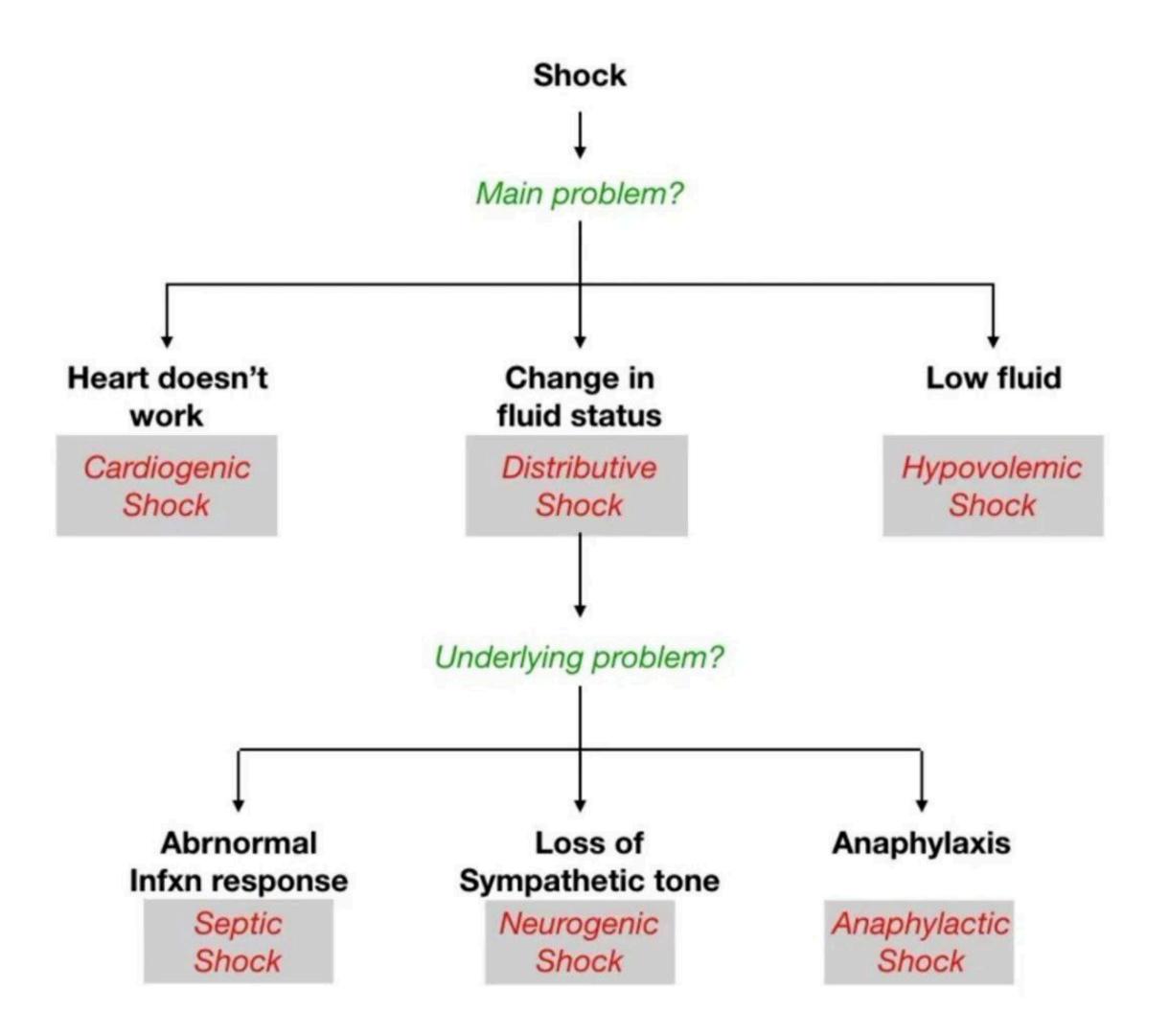
Global tissue hypo-perfusion; decreased blood flow and O2 delivery to, and consequently O2 utilization by, the cells.

OR

Increased tissue oxygen demand without homeostatic increase in blood flow



Once physiological compensation mechanisms are overwhelmed, organ dysfunction ensues, followed by organ failure, irreversible organ damage, and death





1-Hypotension (defined as decrease of ≥40 mmhg from baseline).

Occurs in most patients but a normal BP doesn't rule out shock.

2-Tachycardia (may be an earlier sign of shock than hypotension as compensatory mechanisms can maintain cardiac output).

3-Skin (cold sweaty skin, clammy peripheries, mottled, ashen appearance, skin cyanosis (besides lips and tongue cyanosis))

4-oligourea(consider inserting a urinary catheter, oliguria is defined as <0.5 ml/kg/hr.)

5. Hypoxemia

6-mental state:(use GCS/Agitation, confusion, and distress occur early). Unresponsiveness indicates severe and advanced shock).

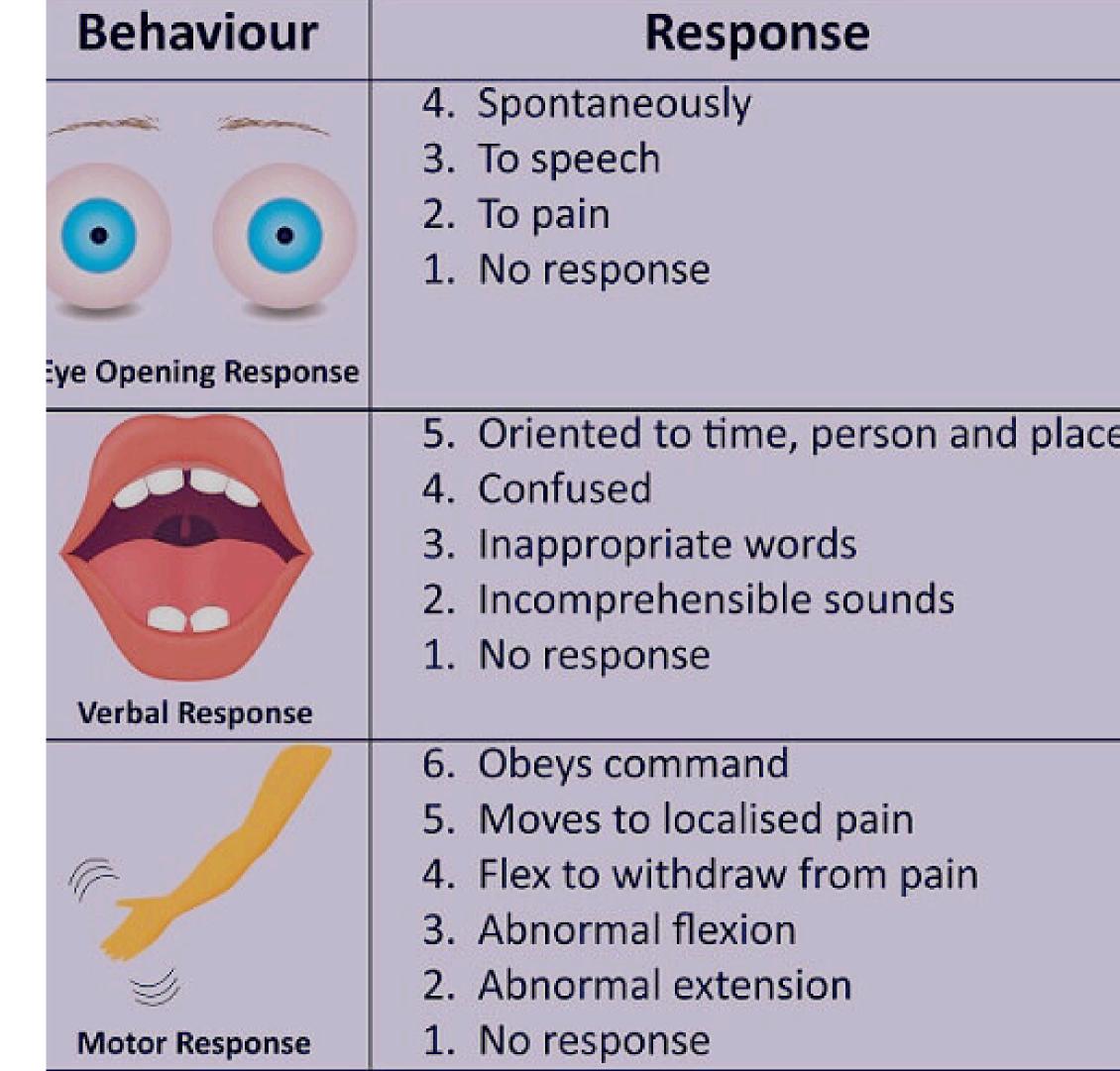
7-positive risk factor (history of sepsis, recent MI, history of hemorrhage, trauma, surgery, exposure to known allergen, change in medications, significant co-morbidities)

8-Dyspnea (Respiratory rate may be increased because of hypoxia (e.g., in pneumonia) but will often remain elevated despite correction of PaO2 due to the need of compensatory hyperventilation of the generated metabolic acidosis)

9-Fever (suggests septic shock). 10-Chest pain (suggest MI).

11-Hypothermia (it is the most obvious clinical sign of end-stage irreversible shock of any cause).

Glasgow coma scale



Diagnostic investigations

- 1. Lactate (From arterial blood gas)
 (result: >2mmol/L)
- 2.Arterial blood gas or venous blood gas (result: Metabolic acidosis; pH < 7.35, bicarbonate <22)
- 3.Glucose (result: > 7mmol/L or > 126mg/dL in non diabetic patien.

- 5.ECG (evidence of MI, arrhythmias, electrolyte abnormalities)
- 6.CXR: look for pulmonary oedema, pneumonia, pneumothorax, widened mediastinum (e.g., due to aortic dissection).
- Urea and electrolytes (evidence of renal impairment if kidney perfusion is compromised for
 4.Blood test: example hypokalemia and
 - hypernatremia with diarrhea and vomiting (hypovolemic shock)
 - ☐ Coagulation studies (result: PT, PTT, fibrinogen; prolonged with DIC in septic shock)

☐ CBC (result: Hb < 10g/dL suggests hemorrhage, WBC >12 x 10³/ macro-liter if sepsis is present.)

☐ C-reactive protein (result: high values suggest sepsis)

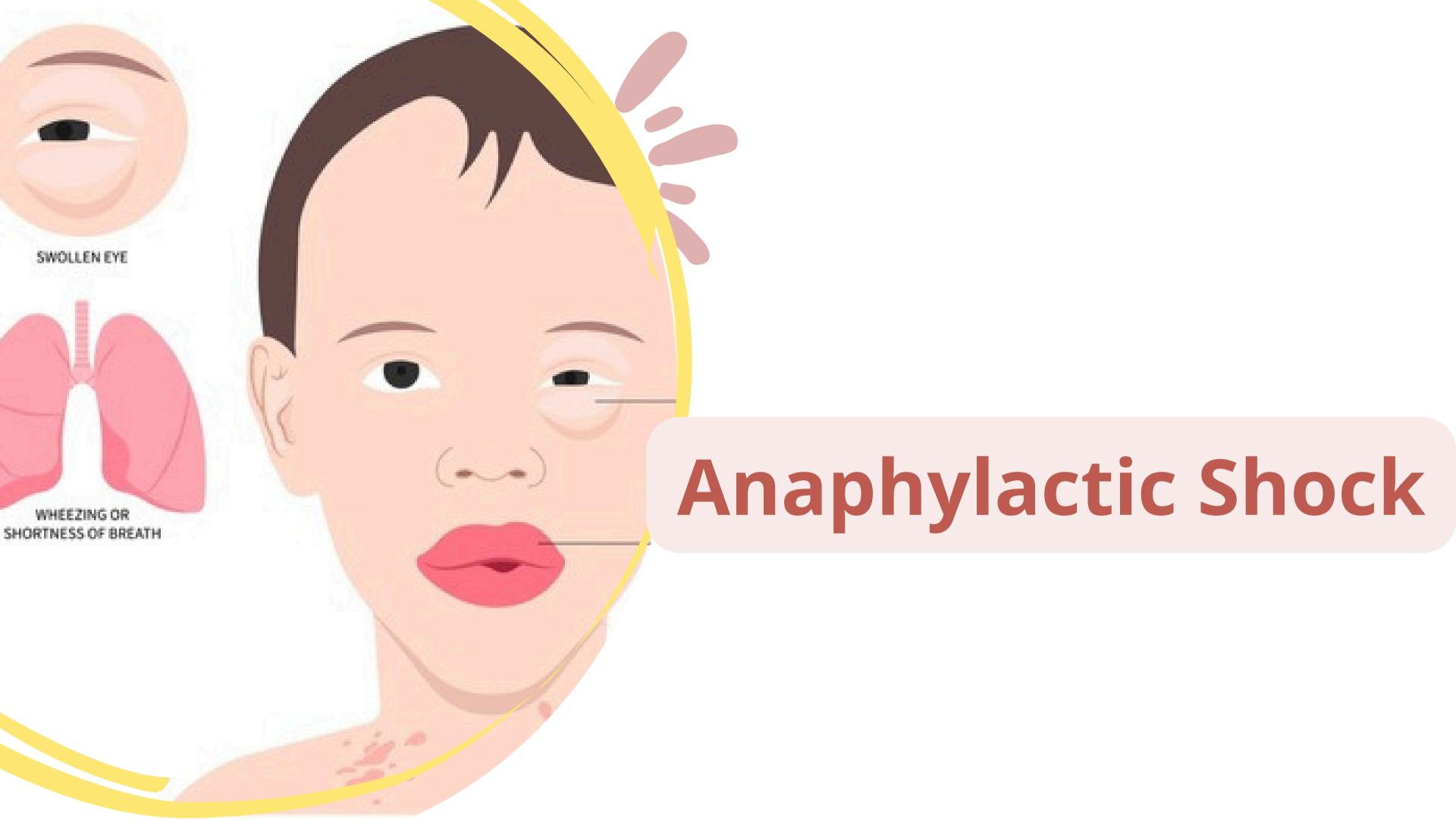
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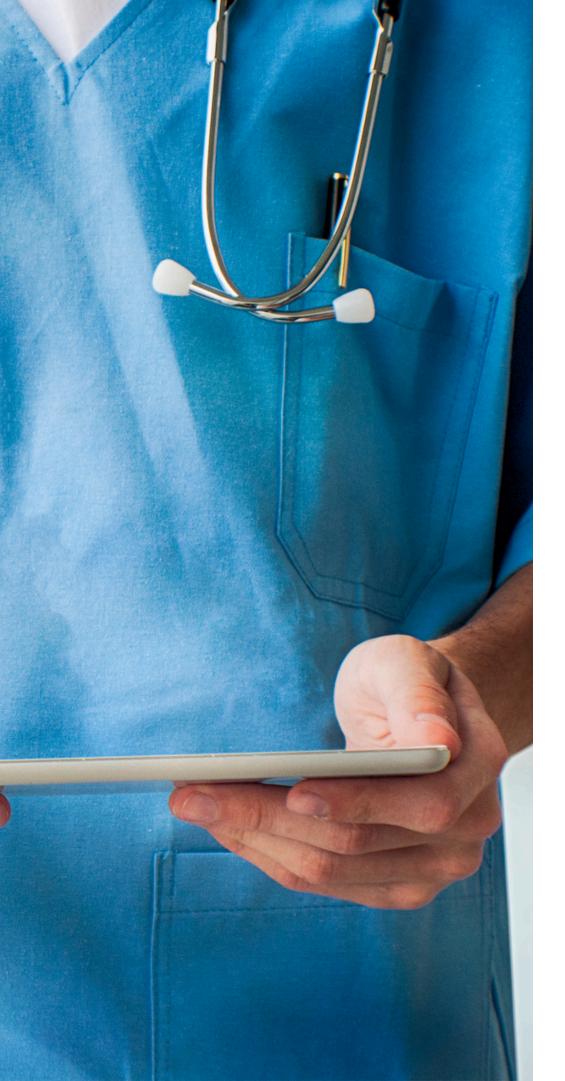
Resuscitation should not delay • while investigating the etiology of undifferentiated shock. Use an ABCDE approach to .manage shock empirically

Use an ABCDE approach to manage shock empirically

ABCDE	Assissment	Treatment
Airway	Voice changes Breath soundes(stridor, snores, increased breathing effort).	 Airway opening manoeuver Airway suction Consider inserting an oropharyngeal or nasopharyngeal airway in deeply unconscious patients <8 GCS .
B reathing	 Respiratory rate Chest wall expansion Chest percussion Lung auscultation Pulse oximetry 	 Seat comfortably Inhaled medications Bag –mask ventilation Decompress tension pneumothorax (needle thoracentesis)
Circulation	 Skin color ,sweating Capillary refill time (normally <2s) Palpate pulse rate (60-100/min) Heart auscultation Blood pressure (systolic 100-140mmHg) ECG monitoring 	Stop bleeding Elevate legs Intravenous access with crystalloid fluid administration
Delivery of oxygen /Disability	 Assess arterial oxygen saturation Mixed venous oxygenation Cardiac index For disability: assess consciousness level, mental status, movement, reflexes 	 Decrease oxygen demands (provide analgesia and anxiolytics to relax muscles and avoid shivering) Maintain arterial oxygen saturation (give supplemental oxygen ,maintain hemoglobin >10g/dl) Serial lactate levels or central venous oxygen saturations to assess tissue oxygen extraction
Exposure /End points of resuscitation	Exposure and tempreture assessment Assess goal values	Make sure your approach is goal —directed: Urine output >0.5ml/kg/hr CVP 8-12 mmHg MAP 65 to 90 mmHg Central venous oxygen concentration >70%

TYPES



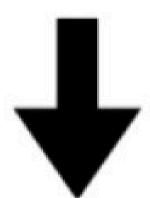


Anaphylactic Shock

 Anaphylactic shock is a severe, generalized or systemic hypersensitivity reaction, characterized by rapidly developing life-threatening airway and/or

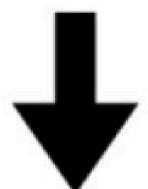
breathing and/or circulation problems usually associated with skin and mucosal changes.

IgE mediated



- Allergen entry: ingestion, inhalation, parenteral, or skin contact.
- Formation of immunoglobulin E (IgE)
 antibodies specific to the antigen presented on first exposure.
- IgE antibodies attach to high-affinity Fc receptors on basophils and mast cells.
- On subsequent exposure, binding of antigen to the IgE antibodies leads to bridging and triggers the degranulation of mast cells.

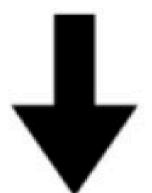
Non IgE mediated



- 1. Activation of complement system
- The complement peptides

 (anaphylatoxins) such as C3a and
 C5 directly act on mast cells and
 basophils leading to mediator
 release

Idiopathic

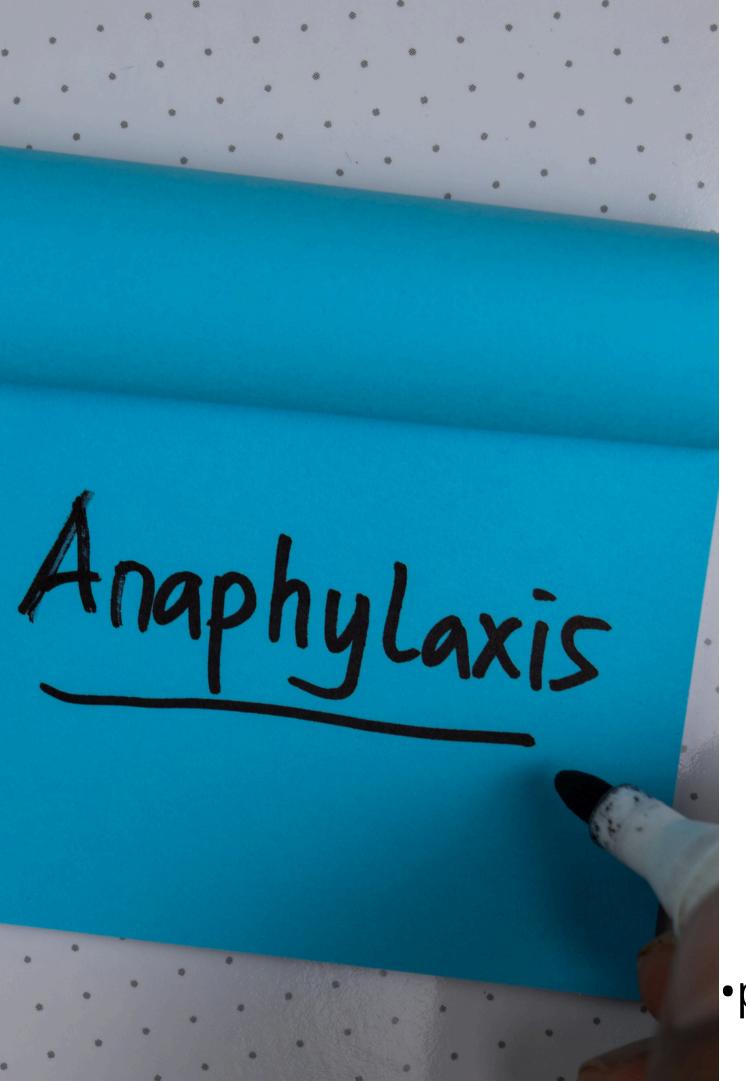


- Increased mast cell sensitivity and degranulation
- Unrecognised allergens



Possible causative agent

- IgE mediated: Food, Airborne allergens, Latex, Venom, insect sting, Medication, Semen
- Immunologic non IgE mediated: Immune aggregate, Intravenous immunoglobulin, Medication (NSAIDs), Radiocontrast media
- Non immunologic: Opiates, Physical factors (e.g., exercise, cold, heat)
- Idiopathic



- Sudden onset and rapid progression of symptoms.
- Firstly: Pruritus, flushing, urticaria (hives)
- Next: swelling, angioedema, trouble swallowing, trouble breathing/shortness of breath, wheezing, hoarse voice, stridor.
- Finally: Altered mental status, respiratory distress, bradycardia followed by respiratory failure and cardiac arrest.
- poorly controlled asthma and previous anaphylaxis
 are risk factors for fatal anaphylaxis



Treatment

- A) In Cardiorespiratory arrest
- 1. Start CPR in a secure location and advanced life support
- 2. Call for help
- 3. Don't give intramuscular adrenaline



- **B)** Not in Cardiorespiratory arrest:
- 1. ABCDE principles
- 2. Position the patient and remove the trigger
- 3. IM adrenaline, repeat if not responding after 5min
- 4. High-concentration oxygen
- 5. IV crystalloid fluid to counteract fluid shifts associated with

vasodilation

- 6. Vital signs monitor.
- 7. Consider the following:
- Nebulized adrenaline (if marked stridor)
- Nebulized short acting B2 agonist (if bronchoconstriction and wheezing)
- IV atropine (if bradycardic)
- IV glucagon (if pt is on B blocker and not responding to adrenaline)



Hypotension, tachycardia, and adrenaline may cause myocardial ischemia by reducing perfusion during diastole. The alpha-1 agonist action of adrenaline can lead to severe hypertension/hypertensive crisis.

Biphasic reaction: Potential second reaction that can occur between 4 and 12 hours after the initial reaction.

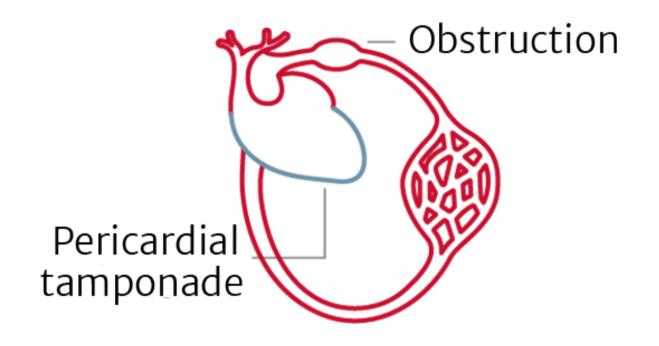
- To prevent biphasic reaction:
- 1. Antihistamine
- 2. Corticosteroids
- 3. If symptoms recur manage the pt as you would for an initial anaphylactic reaction.
- 4. Review by a senior clinician.

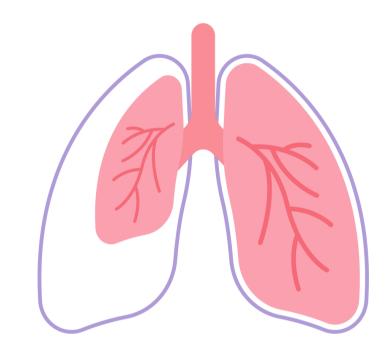
 After all: Before discharge from

hospital, give clear instructions to patients to return to hospital if symptoms recur.

Obstructive Shock

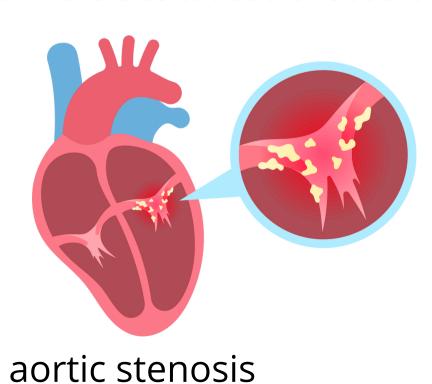


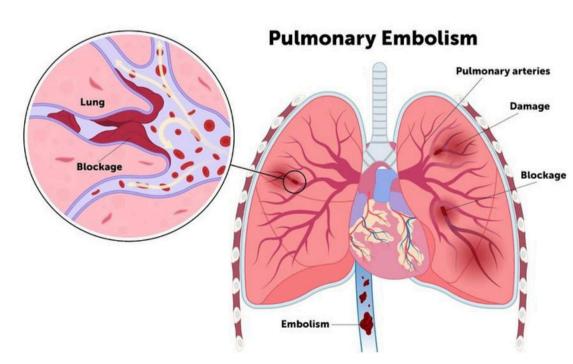




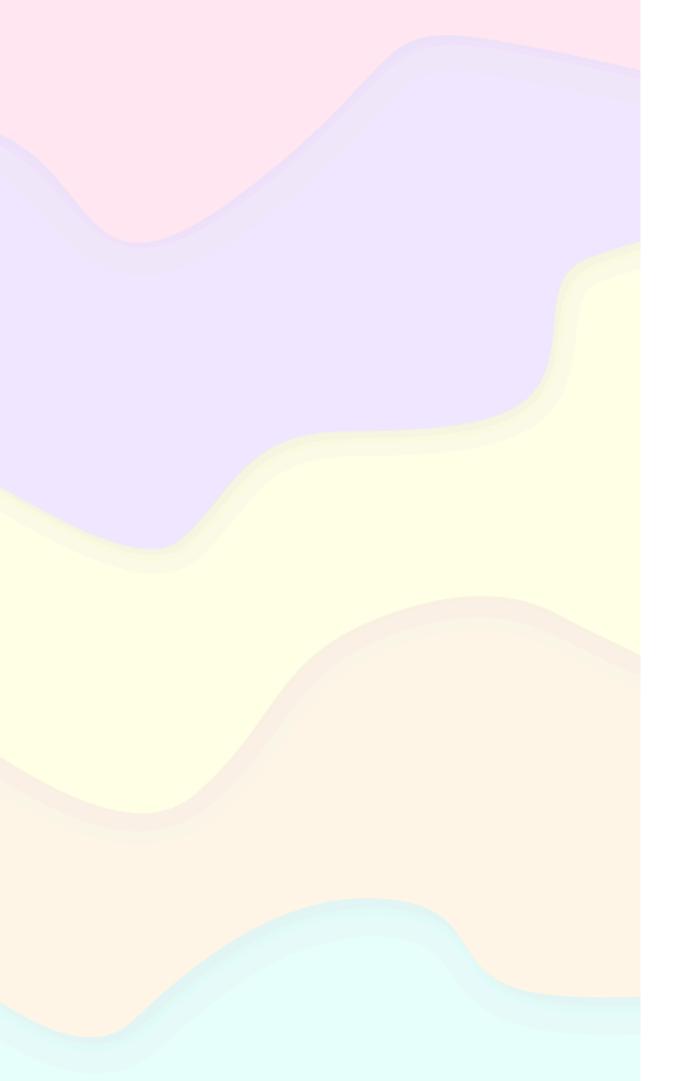
*Usually manifests with elevated JVP

2% Obstructive shock



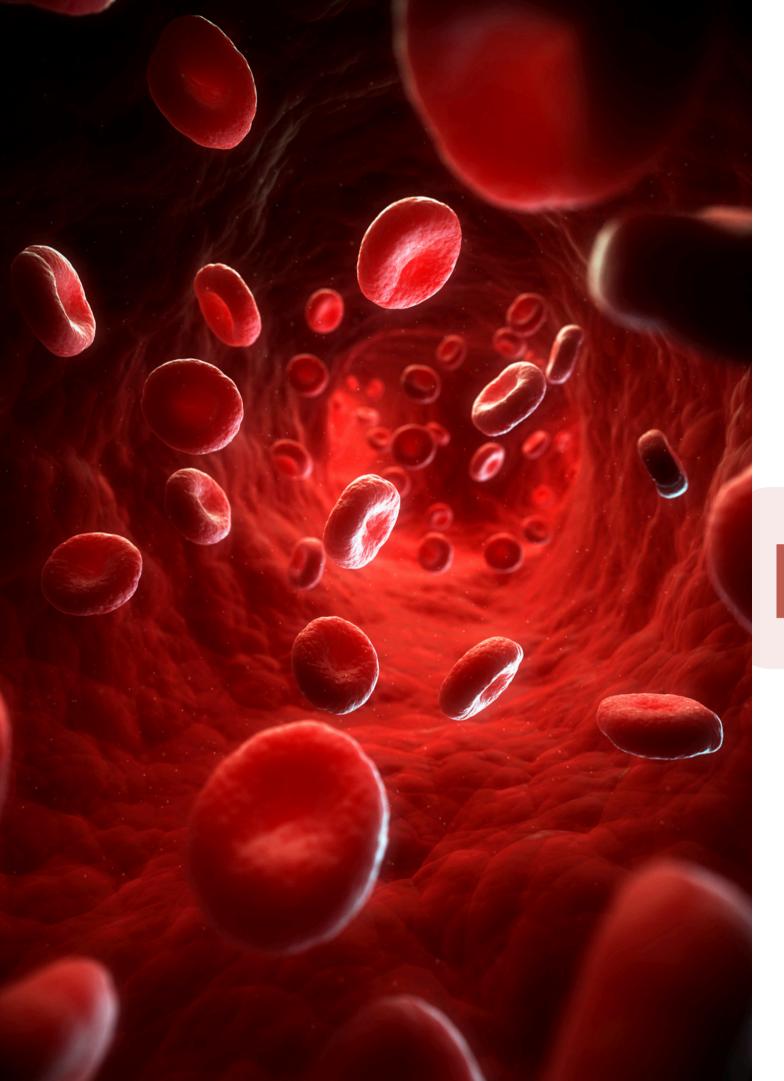






Treatment requires diagnosis of underlying disease.

- Supportive treatment usually involves giving fluid and/or vasopressors to maintain blood pressures until definitive therapy can be given.
- Treatment:
- 1. Cardiac tamponade: Requires pericardiocentesis or pericardial window.
- 2. Aortic stenosis: valve replacement
- 3. Massive PE: Heparin, Apixaban, Rivaroxaban considerthrombolytic



HYPOVOLEMIC Shock

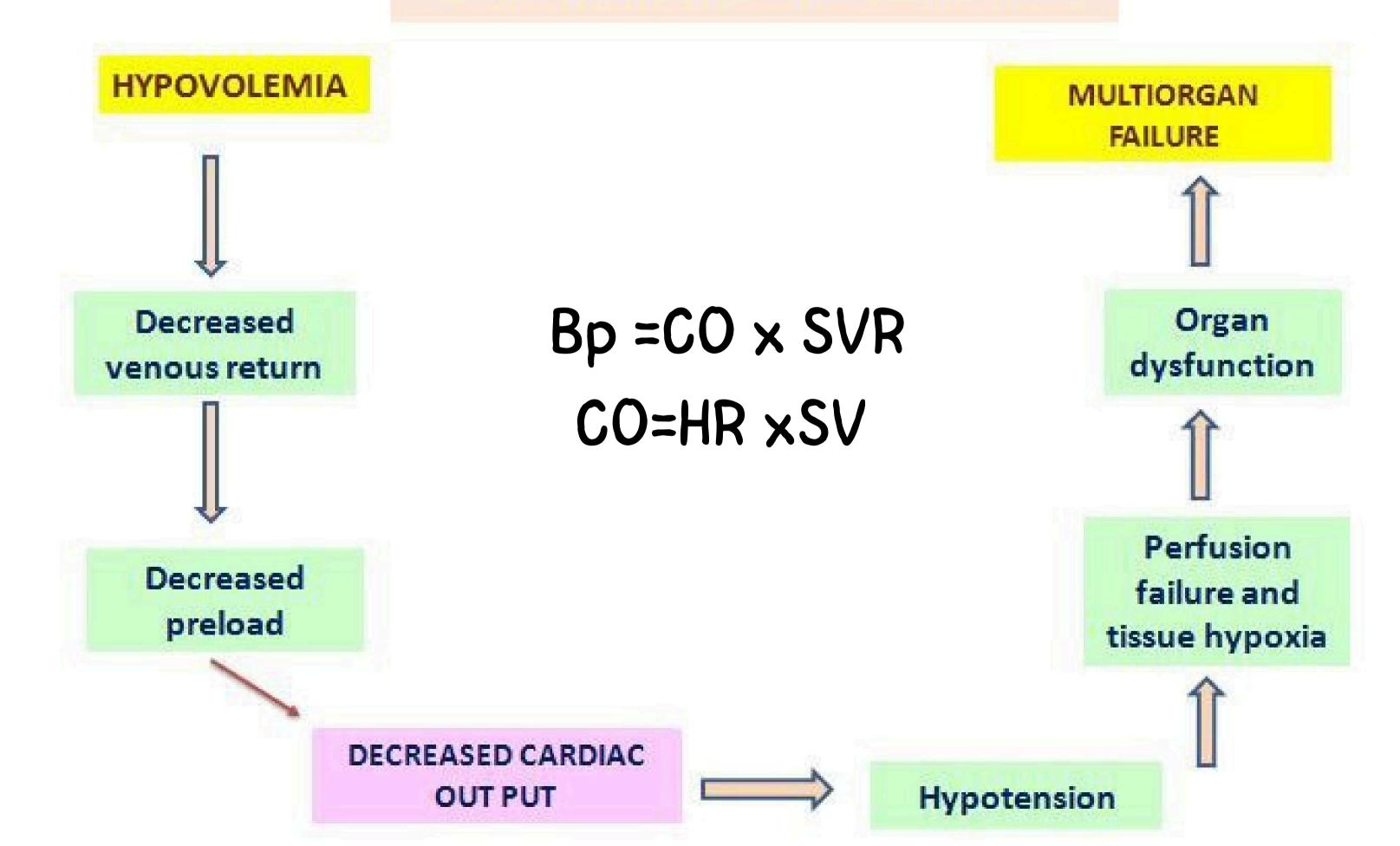
Definition: Reduced circulating blood volume with secondary decreased cardiac .output



- *Causes:
- a) Non-hemorrhagic
 - Vomiting
 - Diarrhea
- *Bowel obstruction
 - **Burns
 - Dehydration

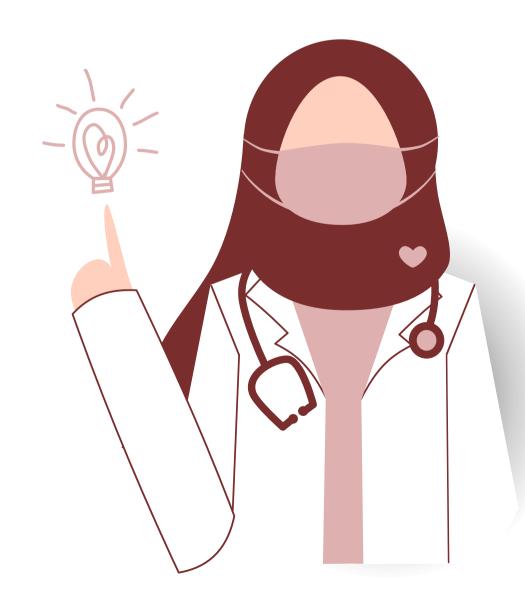
- b) Hemorrhagic
 - **GI** bleed
 - **Trauma
- Massive hemoptysis
- *post-partum bleeding

PATHOPHYSIOLOGY OF HYPOVOLEMIC SHOCK





- CBC
- ABG
- Electrolytes
- Coagulation studies
- Type and cross-match
 - As indicated: CXR,
 Pelvic x-ray, CT, GI
 endoscopy, Vascular
 radiology



Classes of Hypovolemic Shock

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
Blood Vol. %	< 15%	15-30%	30-40%	> 40%
Pulse	< 100	> 100	> 120	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Resp. Rate	14-20	20-30	30-40	> 40
UOP	> 30	20-30	5-15	negligible
Mental Status	sl. Anxious	mildly anx	confused	lethargic
Fluid	crystalloid	crystalloid	blood	blood

Arterial Blood Gas (ABG)

ABG	Normal range
O ₂ CT	15-23% per 100 mL of blood
рН	7.35-7.45
PaCO ₂	35-45 mmHg
PaO ₂	80-100 mmHg
HCO ₃	22-26 mEq/L
O ₂ Sat	95-100%

Complete Blood Count Normal Range* WBCs 3,500-11,000 cells/mcL 34.9%-44.5% in women Hematocrit 38.8%-50% in men **Platelets** 150,000-450,000/mcL 4.3-5.7 million cells/mcL in men RBCs 3.9-5.1 million cells/mcL in women 13-17 g/dL in men Hemoglobin 11.5-15.5g/dL in women

ABCs

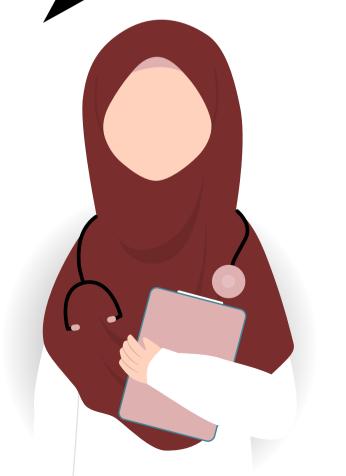
Management

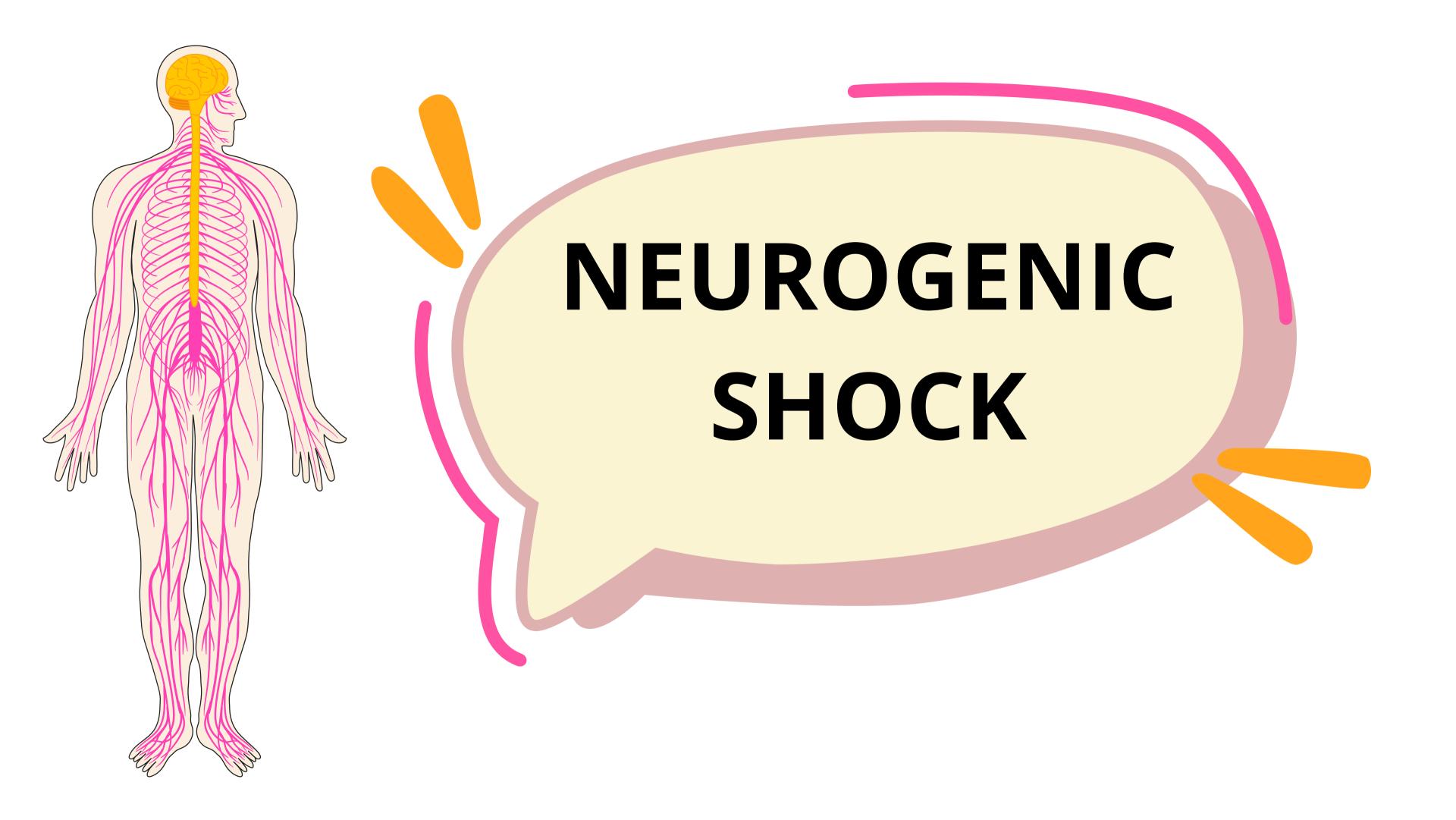
Establish 2 large bore IVs or a central line

Crystalloids: Normal Saline or Lactate Ringers: Up to 3 liters

PRBCs: O negative or cross matched

Control any bleeding





General Characteristics:

- Neurogenic shock results from a failure of the sympathetic nervous system to maintain adequate vascular tone (sympathetic denervation)
- Causes include spinal cord injury, severe head injury, spinal anesthesia, pharmacologic sympathetic blockade , Characterized by peripheral vasodilation with decreased SVR



1. Warm, well-perfused skin.

- 2. Urine output low or normal
- 3. Bradycardia and hypotension (but tachycardia can occur)
 - 4. Cardiac output is decreased, SVR low, PCWP low to normal

Treatment





Cardiogenic Shock

Cardiogenic Shock

•Cardiogenic shock is a life-threatening condition in which your heart suddenly can't pump enough blood to meet your body's needs.

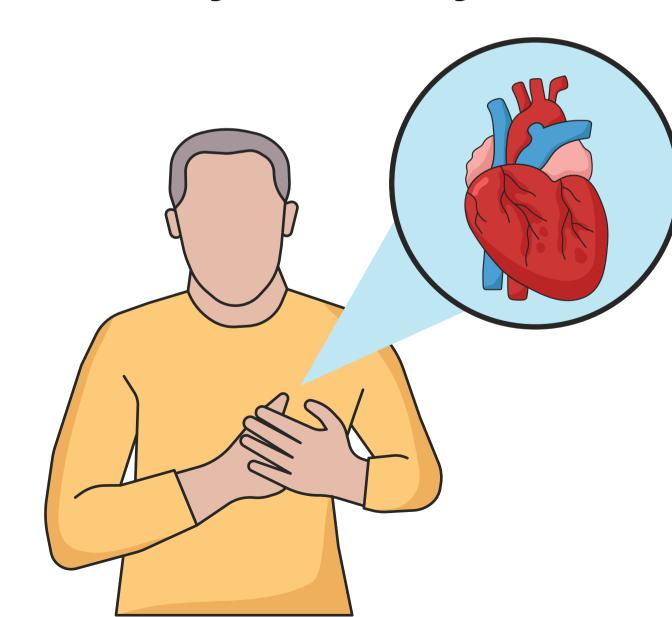
• It may be caused by:

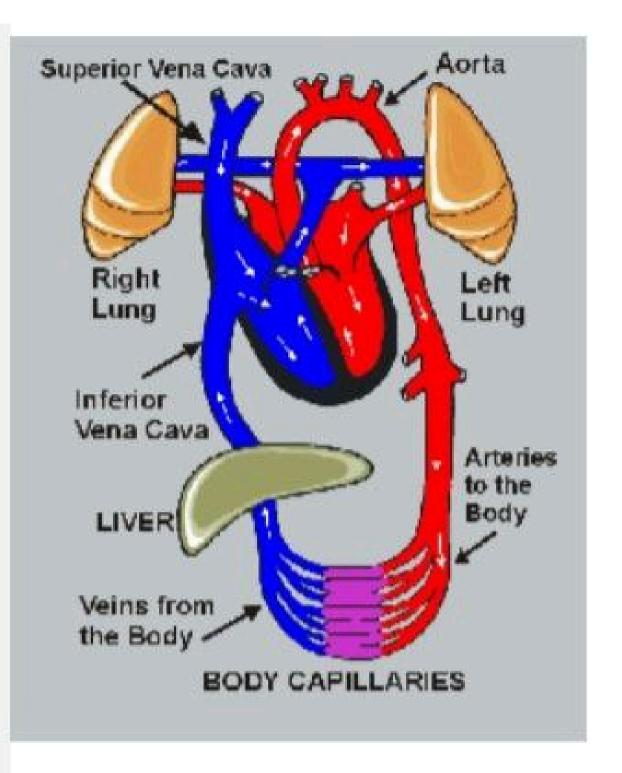
myocardial damage (infarction).

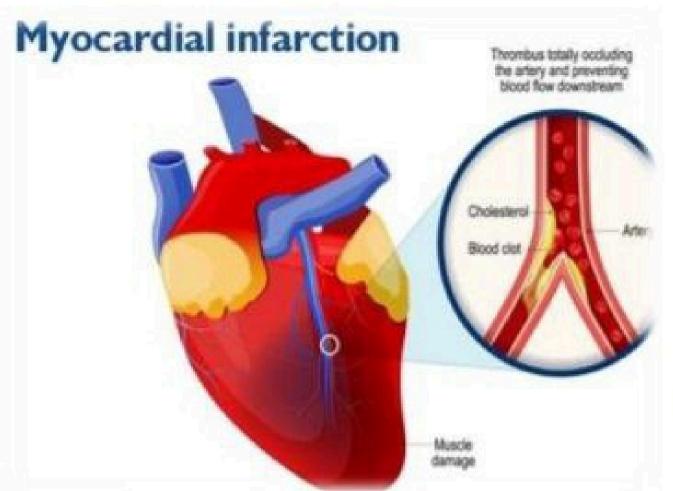
extrinsic compression (cardiac tamponade).

ventricular arrhythmias.

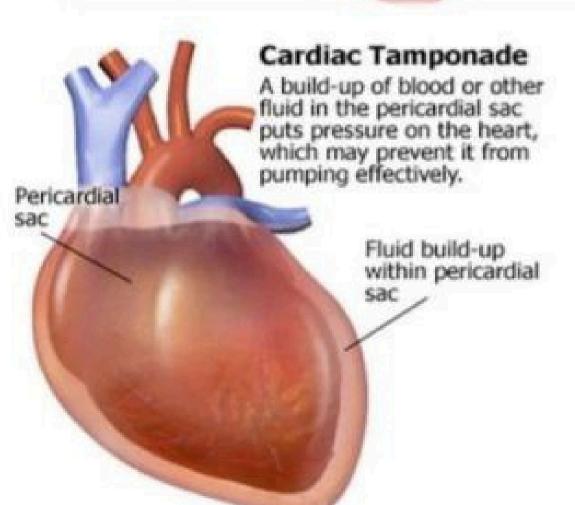
outflow obstruction (e.g., pulmonary embolism).

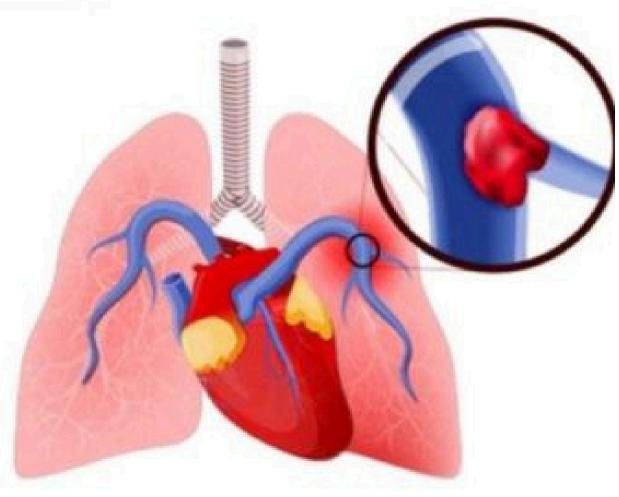






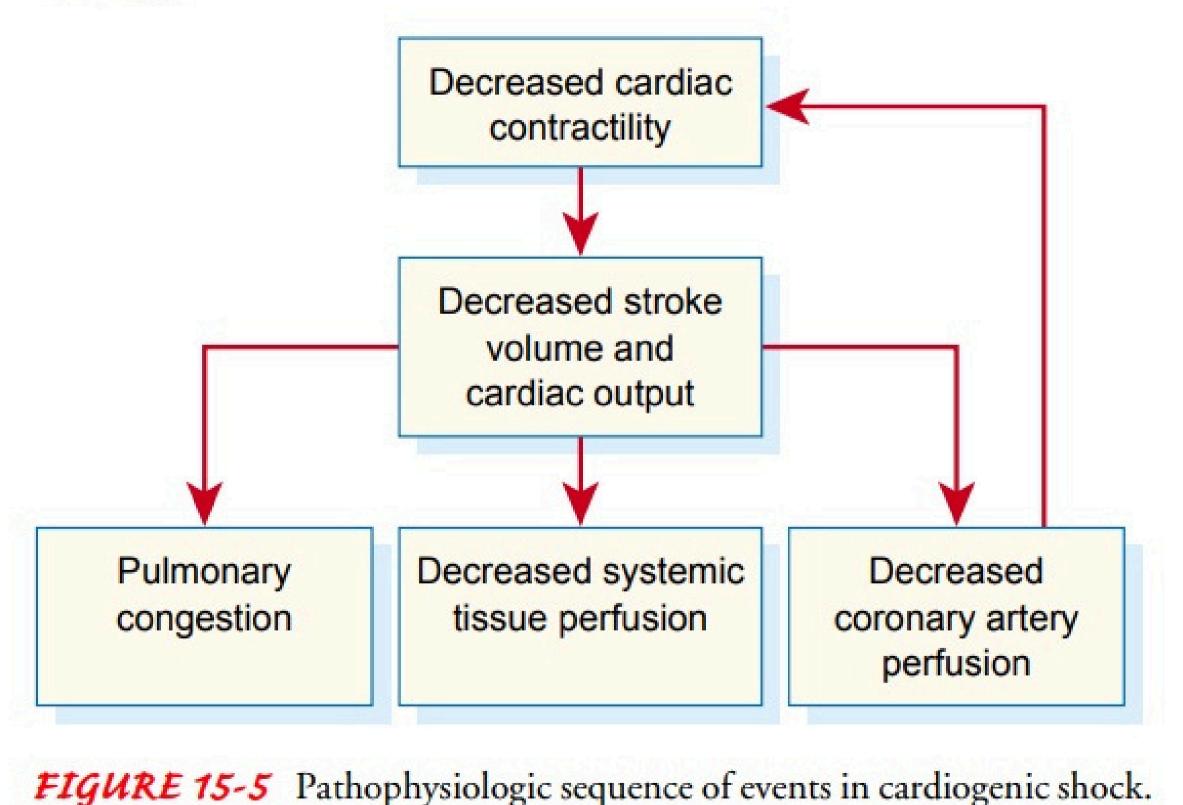
Pulmonary embolism





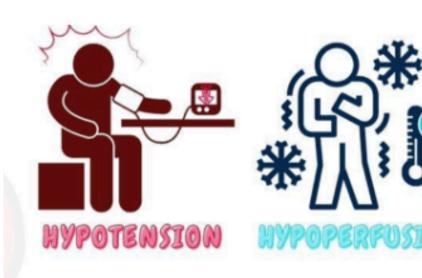


Physiology/Pathophysiology

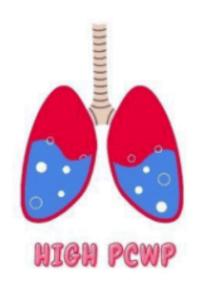


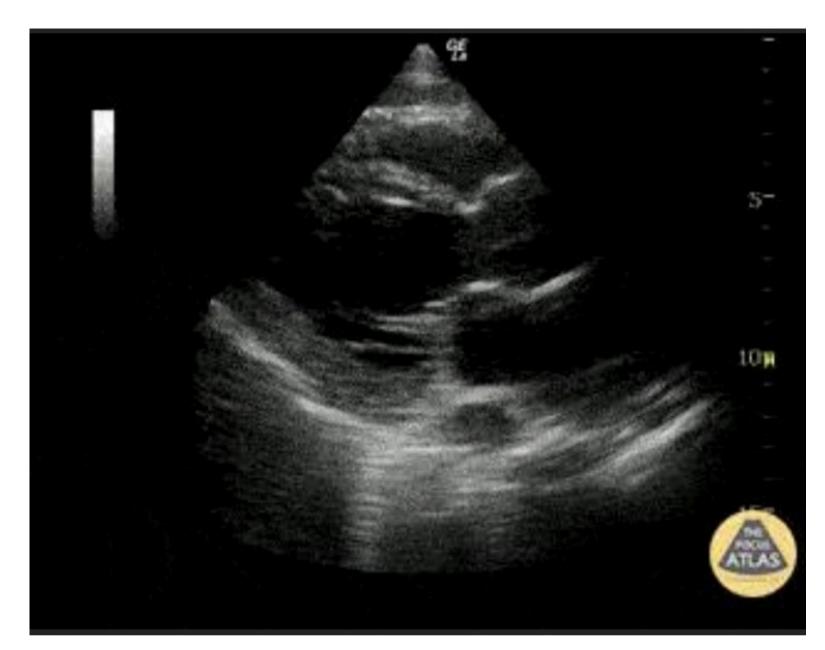
SYMPTOMS &SIGN

1. Cool, mottled skin 2. Tachypnea 3·Hypotension 4.Altered mental status 5•Murmur









On Echo:
.We notice frank LV systolic dysfunction



On ECG:

Irregular heart rates (arrhythmias), such as ventricular tachycardia or ventricular fibrillation.

These arrhythmias may be the cause of the cardiogenic shock.

Treatment

Goals:

- 1. Airway stability
- 2. improving myocardiak

1.Cardiac monitor, pulse oximetry

2.Supplemental oxygen, IV access

3.Catheterization
n if ongoing
ischemia

4.Preload augmentation: Consider Fluids

5.Contractility:
Dopamine,
dobutamine

6.Afterload reduction:
Nitroglycerin,
Dobutamine

7.intra-aortic balloon pump(if inotropes&vasopressors fail



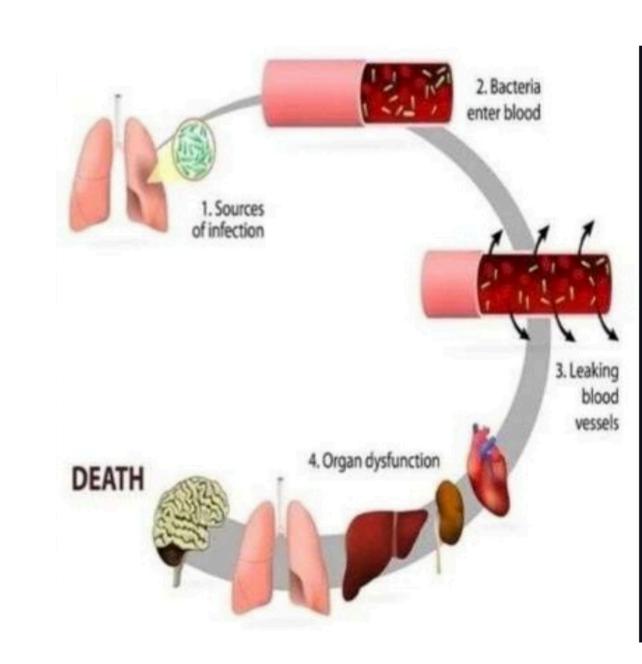
Septic shock

Septic shock

Defined as: hypotension induced by sepsis
 that persists despite adequate fluid
 resuscitation.

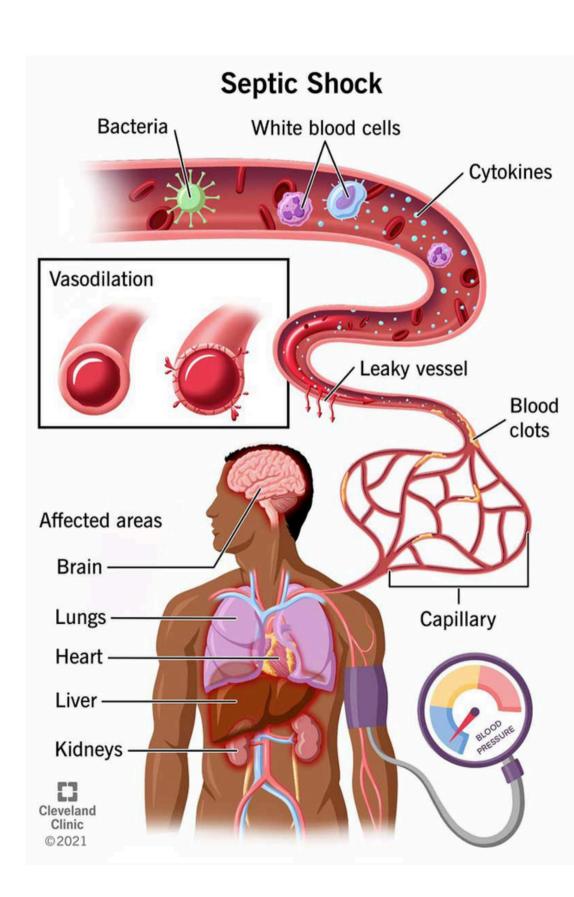
This results in hypoperfusion and can ultimately lead to multiple organ system failure and death.

 Common causes: include pneumonia, urinary tract infection, meningitis, abscess formation, cholangitis, cellulitis, and peritonitis.



Pathophysiology

- There is a severe decrease in SVR secondary
- to peripheral vasodilation. Extremities are often warm due to <u>vasodilation</u>.
- Cardiac output is normal or increased (due
- to maintenance of stroke volume and tachycardia).
- EF is decreased secondary to a reduction in contractility





- Clinical signs:
- Hyperthermia or hypothermia (hypothermia is more common in the very young, elderly, and immunocompromised)
- Tachycardia
- Wide pulse pressure
- Low blood pressure
- Mental status changes
- Diagnosis:
- Septic shock is essentially a clinical diagnosis.
- A source of infection can aid in diagnosis, but there may be no confirmed source in some cases.



Treatment:

- 1. Fluid administration to increase mean BP (Goal: MAP > 60)
- 2. Obtain cultures prior to starting antibiotics. Start IV antibiotics (broad spectrum) at maximum dosages. If cultures are positive, antibiotics can be narrowed based on sensitivity testing.
- 3. Surgical drainage if necessary.
- 4. Vasopressors (norepinephrine, vasopressin, phenylephrine) may be used if hypotension persists despite aggressive IV fluid resuscitation

