





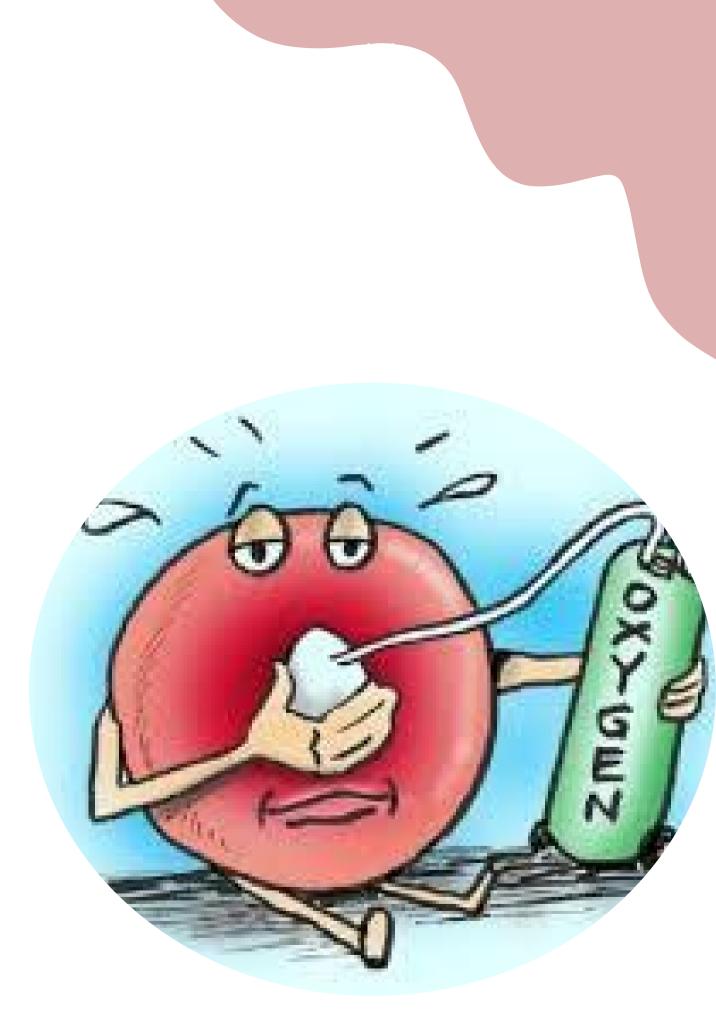


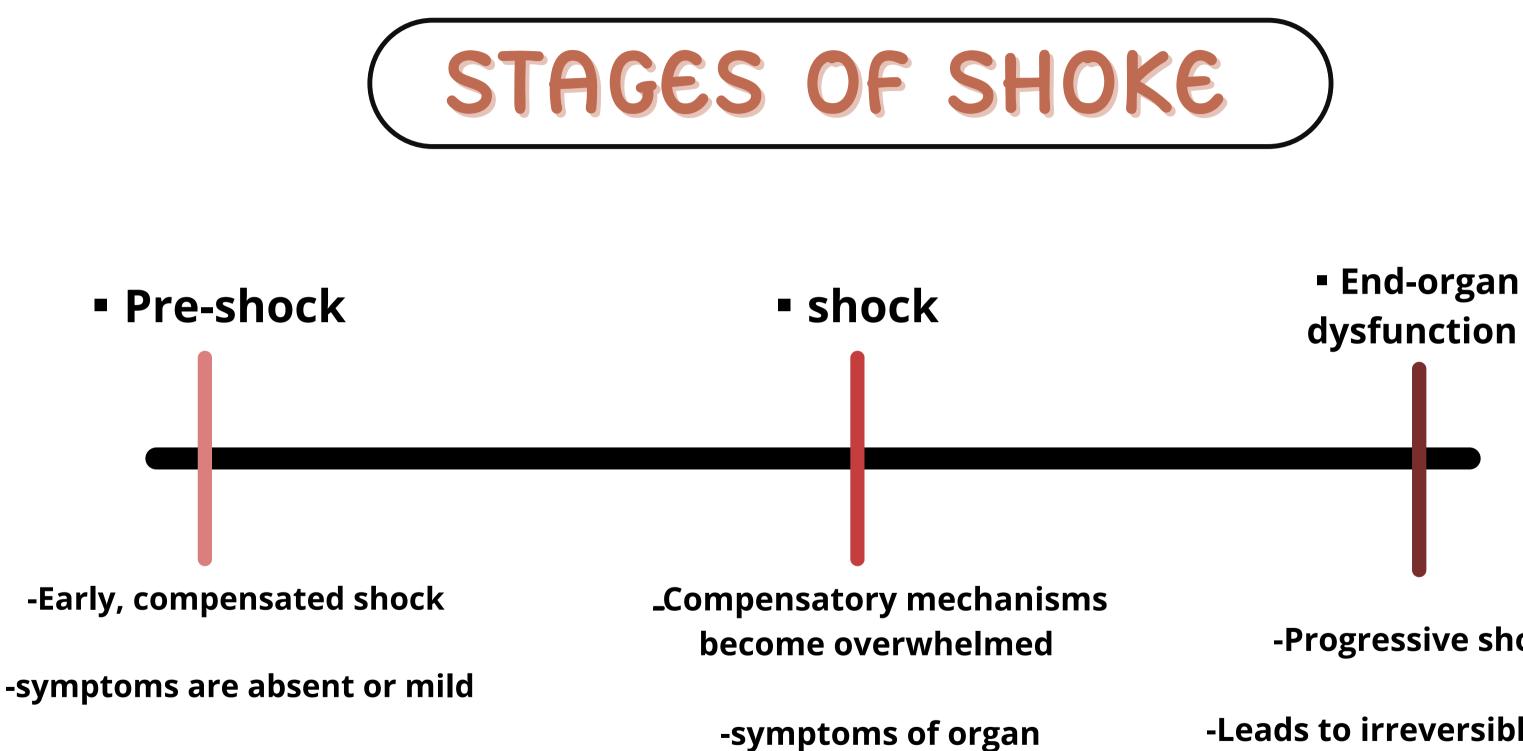






- Shock is a life-threatening manifestation of circulatory failure that leads to cellular and tissue hypoxia resulting in cellular death and dysfunction of vital organs.
- The effects of shock are initially reversible, but rapidly become irreversible, resulting in multi-organ failure (MOF) and death.



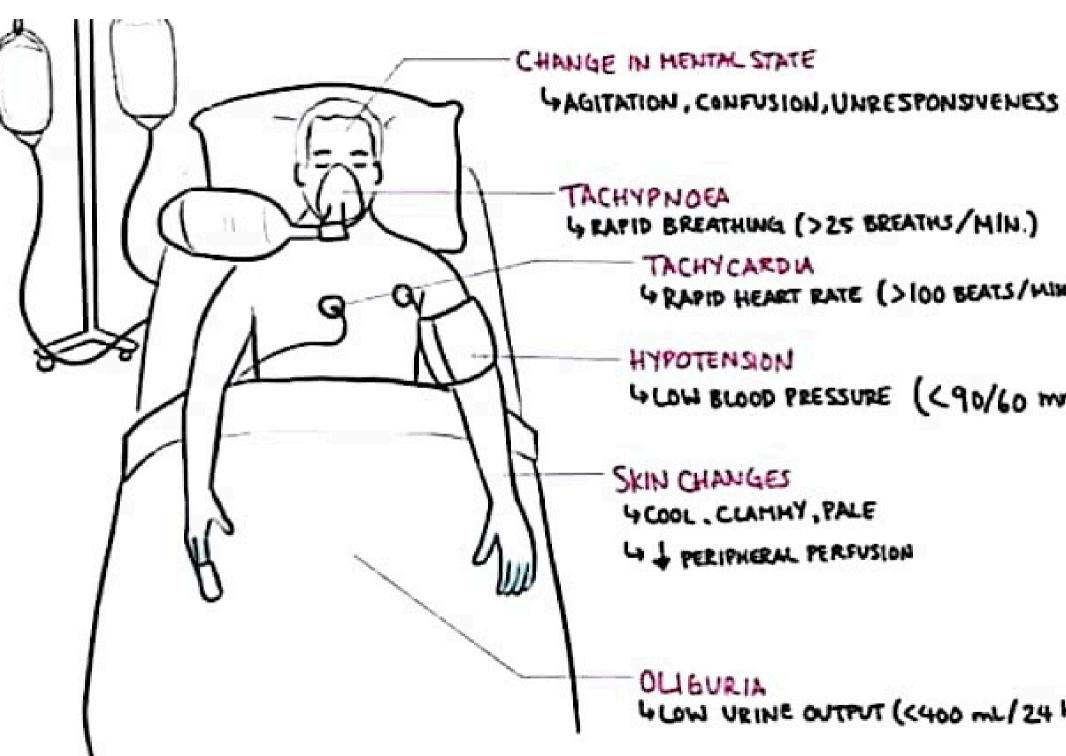


dysfunction begin to apper

-Progressive shock

-Leads to irreversible organ damage and death

Clinical features

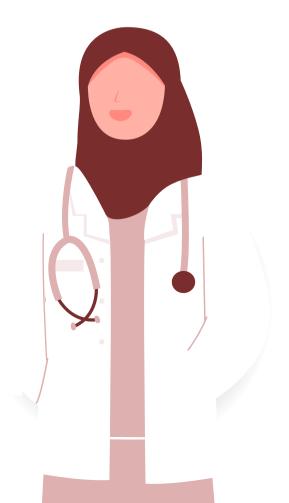




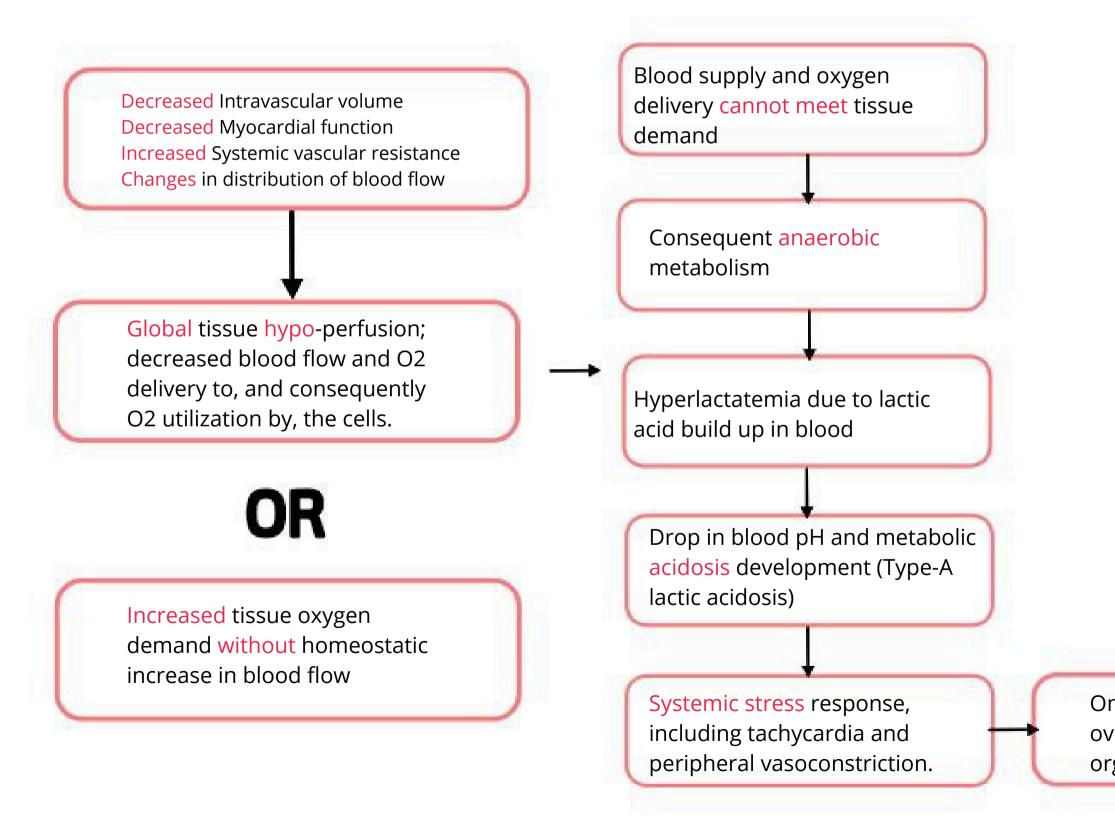
4 RAPID HEART RATE (>100 BEATS/MIN.)

4 LOW BLOOD PRESSURE (<90/60 mmHg)

4 LOW URINE OUTPUT (<400 ml/24 hrs)

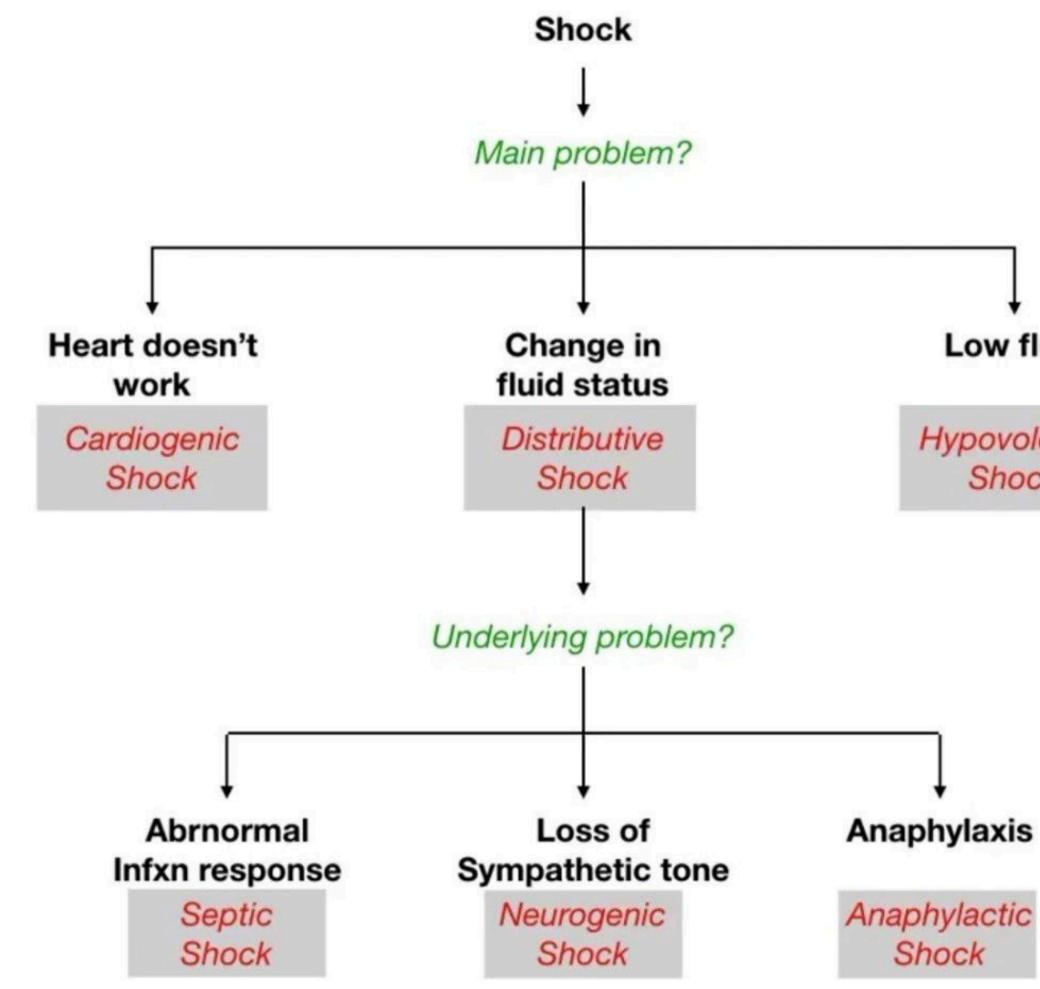


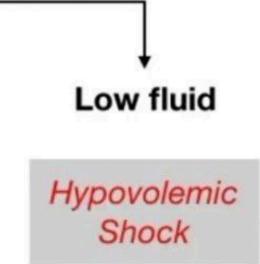
Pathophysiology





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)

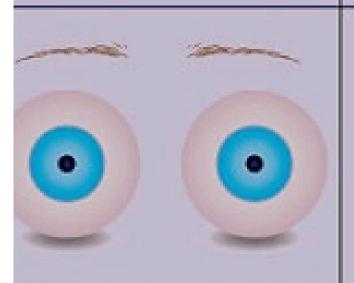
8-Dyspnea (Respiratory rate may be increased because of hypoxia (e.g., in pneumonia) but will often remain elevated despite correction of PaO2 due to the need of compensatory hyperventilation of the generated metabolic acidosis)

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

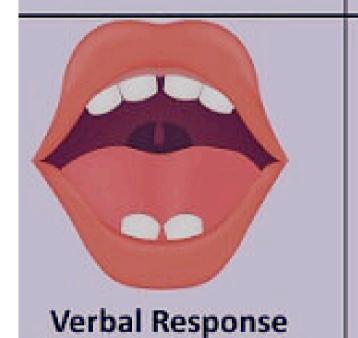
11-Hypothermia (it is the most obvious clinical sign of end-stage irreversible shock of any cause).

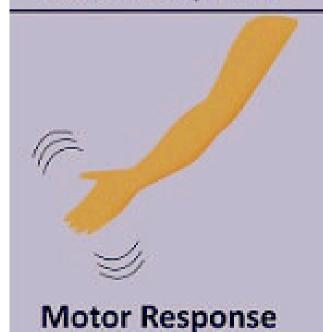
Glasgow coma scale

Behaviour



ye Opening Response





Response

- 4. Spontaneously
- 3. To speech
- 2. To pain
- 1. No response

- 5. Oriented to time, person and place
- 4. Confused
- 3. Inappropriate words
- 2. Incomprehensible sounds
- 1. No response
- 6. Obeys command
- 5. Moves to localised pain
- 4. Flex to withdraw from pain
- 3. Abnormal flexion
- 2. Abnormal extension
- 1. No response

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).

□ CBC (result: Hb < 10g/dL suggests hemorrhage, WBC >12 x 10³/ macro-liter if sepsis is present.) **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) C-reactive protein (result: high values suggest sepsis)**

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

delay • ology of oach to ally

Use an ABCDE approach to manage shock empirically

ABCDE	Assissment	Treatment	
Airway	 Voice changes Breath soundes(stridor, snores, increased breathing effort). 	 Airway opening many Airway suction Consider inserting an patients <8 GCS . 	
Breathing	 Respiratory rate Chest wall expansion Chest percussion Lung auscultation Pulse oximetry 	 Seat comfortably Inhaled medications Bag –mask ventilation Decompress tension 	
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 w 	
Delivery of oxygen /Disability	 Assess arterial oxygen saturation Mixed venous oxygenation Cardiac index For disability : assess consciousness level ,mental status ,movement ,reflexes 	 Decrease oxygen den shivering) Maintain arterial oxy >10g/dl) Serial lactate levels o extraction 	
Exposure /End points of resuscitation	 Exposure and tempreture assessment Assess goal values 	 Make sure your approved on the series of the	

noeuver

n oropharyngeal or nasopharyngeal airway in deeply unconscious

s on n pneumothorax (needle thoracentesis)

with crystalloid fluid administration

emands (provide analgesia and anxiolytics to relax muscles and avoid

sygen saturation (give supplemental oxygen , maintain hemoglobin

or central venous oxygen saturations to assess tissue oxygen

roach is goal –directed : hr

oncentration >70%

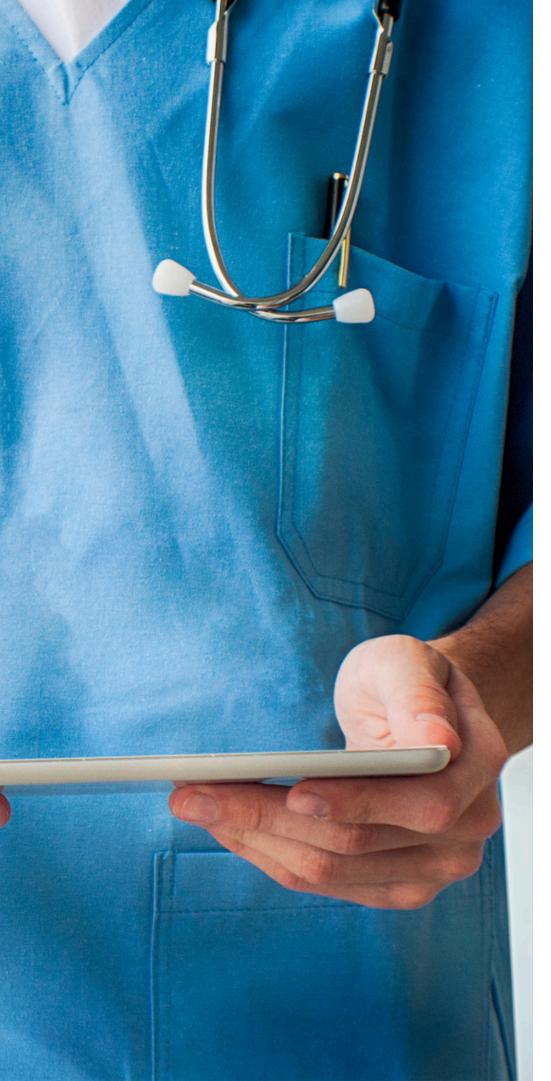
TYPES



SWOLLEN EYE

WHEEZING OR SHORTNESS OF BREATH

Anaphylactic Shock

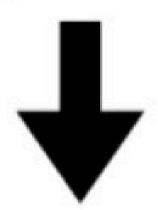


Anaphylactic Shock

• Anaphylactic shock is a severe, reaction, characterized by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

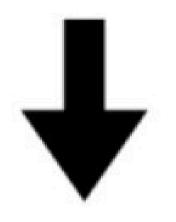
generalized or systemic hypersensitivity

IgE mediated

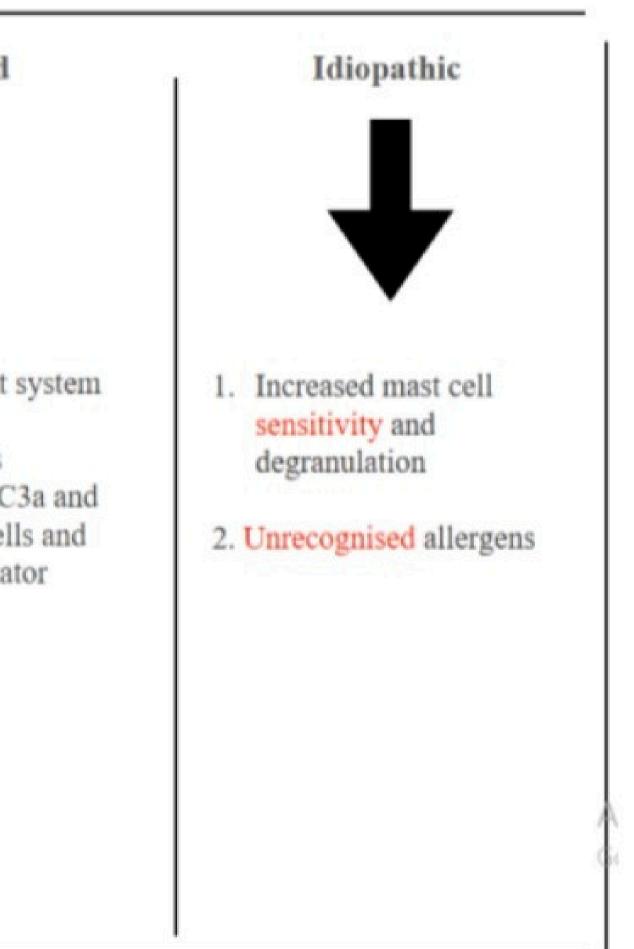


- Allergen entry: ingestion, inhalation, parenteral, or skin contact.
- Formation of immunoglobulin E (IgE) antibodies specific to the antigen presented on first exposure.
- 3. IgE antibodies attach to high-affinity Fc receptors on basophils and mast cells.
- On subsequent exposure, binding of antigen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



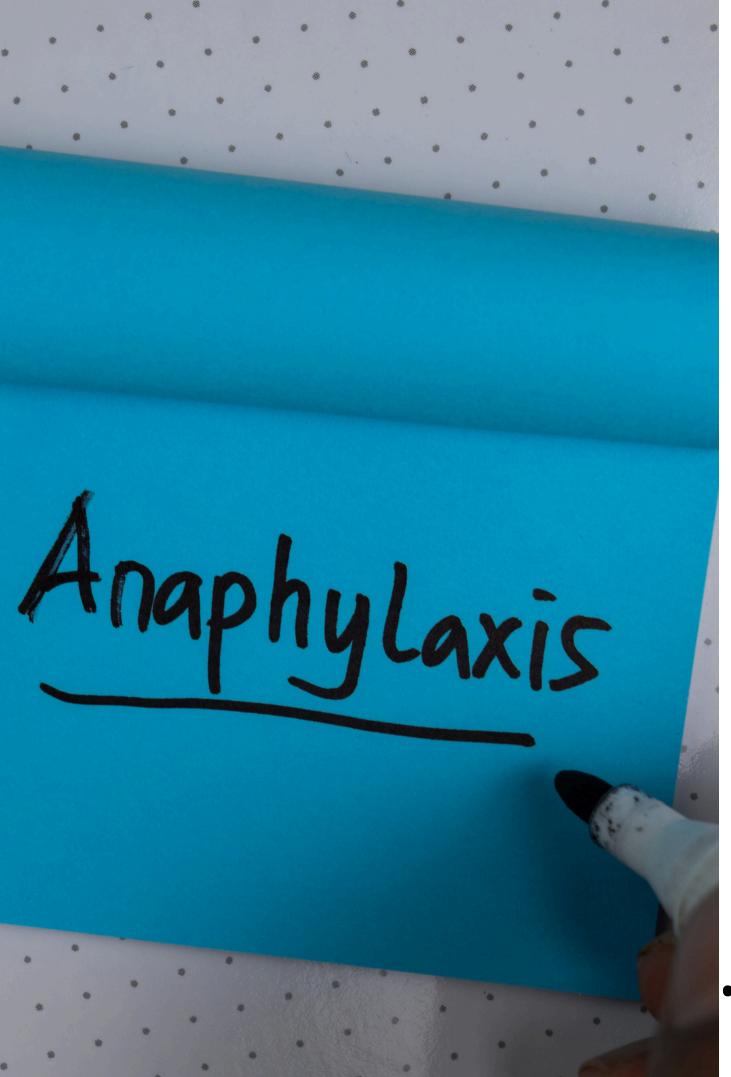




- aggregate, (NSAIDs),
 - **Radiocontrast media**
- Non immunologic: Opiates, Physical factors (e.g., exercise, cold, heat)
- Idiopathic

• IgE mediated: Food, Airborne allergens, Latex, Venom, insect sting, Medication, Semen • Immunologic non IgE mediated: Immune

Intravenous immunoglobulin, Medication



- 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.

Sudden onset and rapid progression of

 poorly controlled asthma and previous anaphylaxis are risk factors for **fatal anaphylaxis**

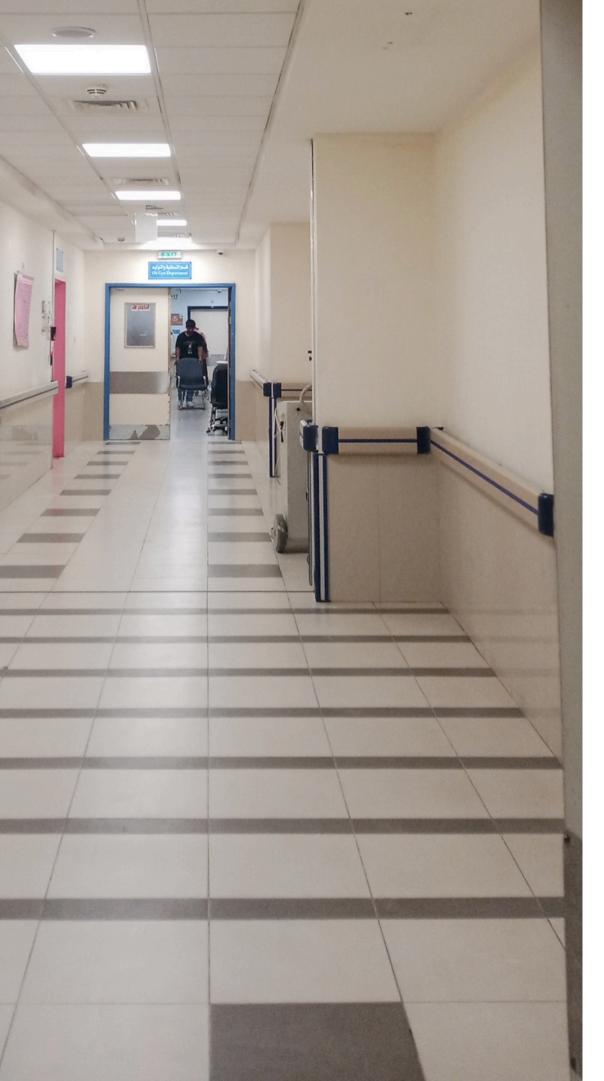


Treatment

A) In Cardiorespiratory arrest advanced life support 2. Call for help

1. Start CPR in a secure location and

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)
- adrenaline)

• IV glucagon (if pt is on B blocker and not responding to

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.

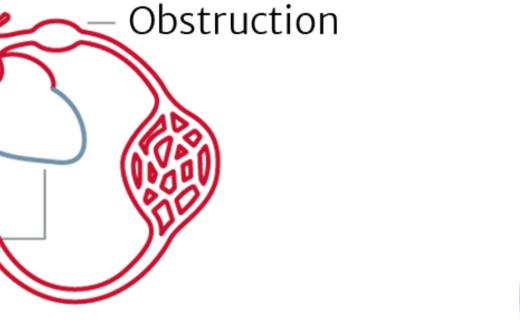
- 4. Review by a senior clinician.
- **After all: Before discharge from**
- hospital, give clear instructions to patients to return to hospital if
- symptoms recur.

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.

Obstructive Shock

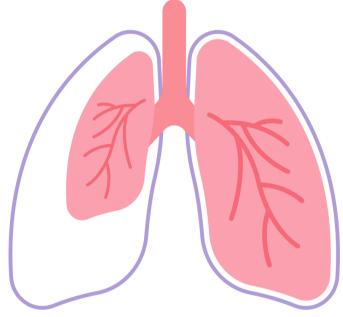
tension pneumothorax

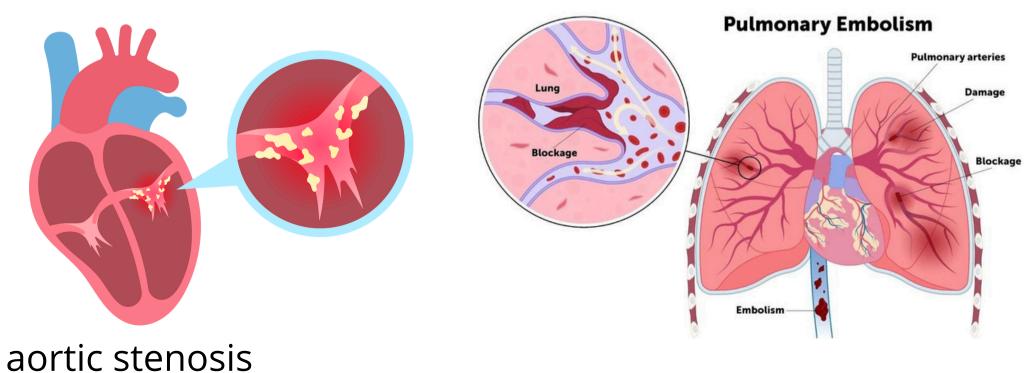


2% Obstructive shock

Pericardial

tamponade







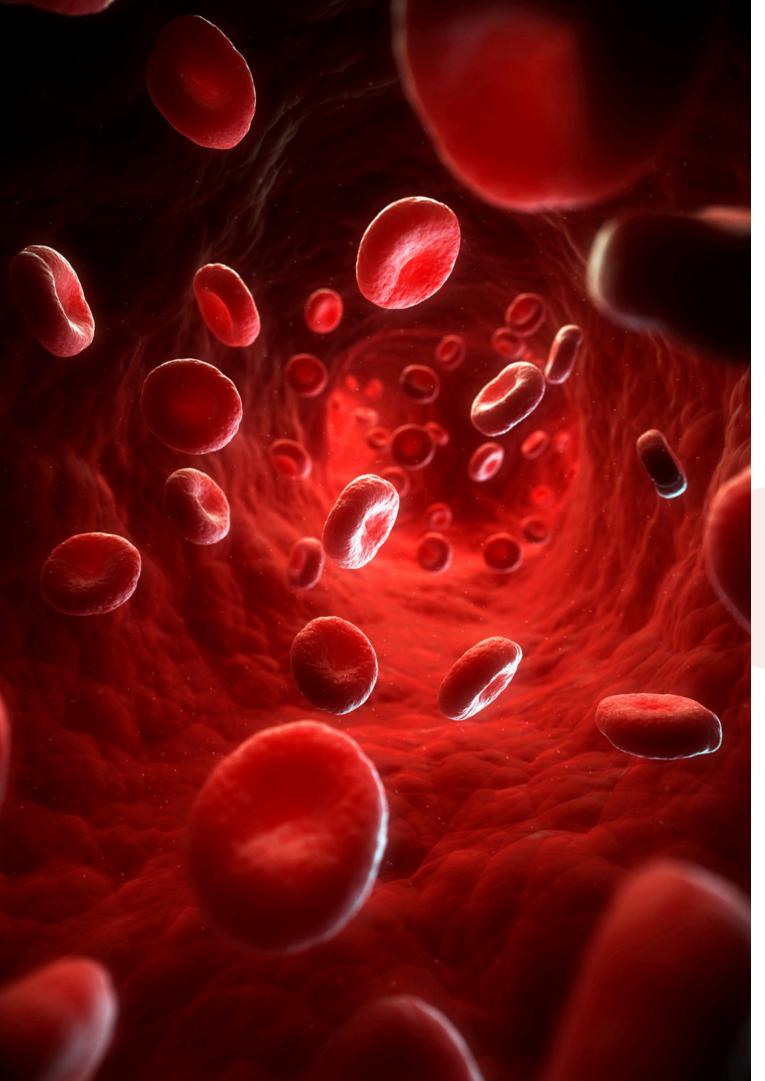


Treatment requires diagnosis of underlying disease. giving fluid and/or until definitive therapy can be given. • Treatment: **1. Cardiac tamponade: Requires** pericardiocentesis or pericardial window.

Rivaroxaban considerthrombolytic

- Supportive treatment usually involves
- vasopressors to maintain blood pressures

- 2. Aortic stenosis: valve replacement 3. Massive PE: Heparin, Apixaban,



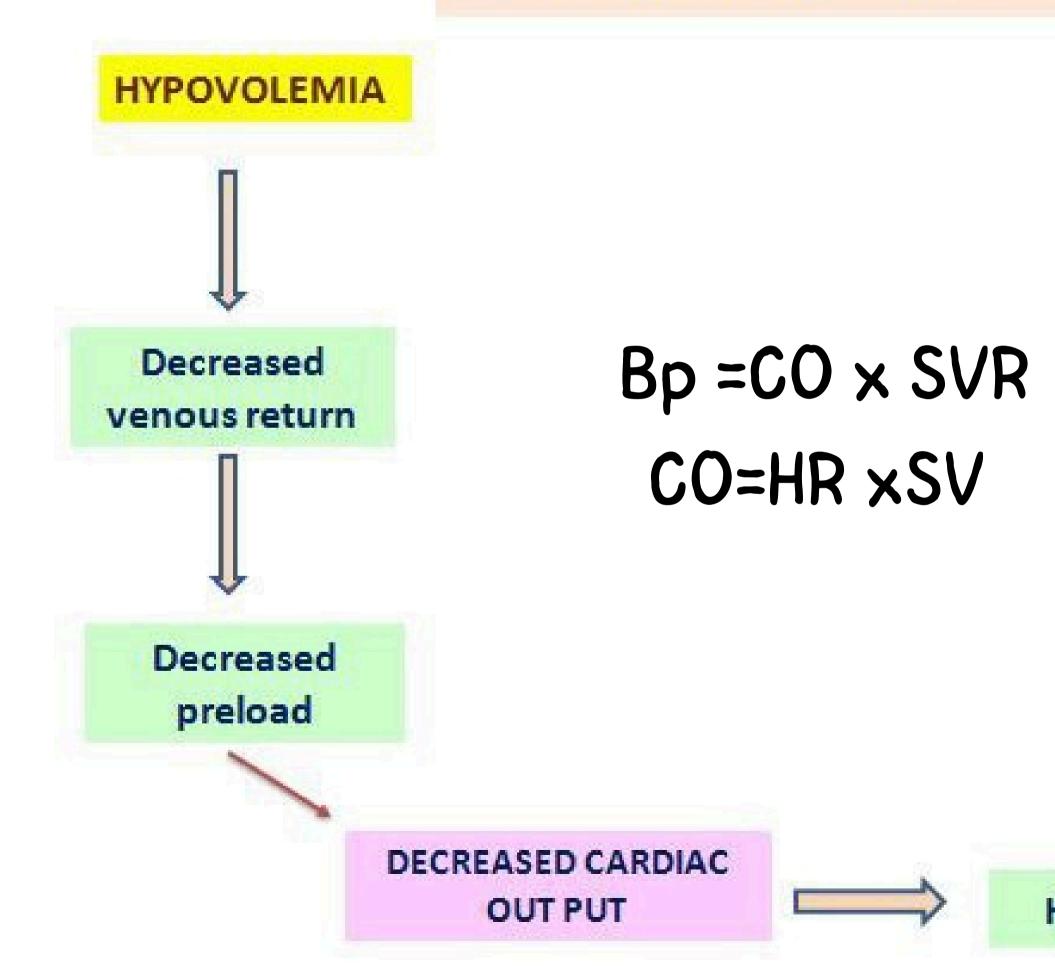
HYPOVOLEMIC Shock

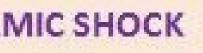
Definition: Reduced circulating blood volume with secondary decreased cardiac .output

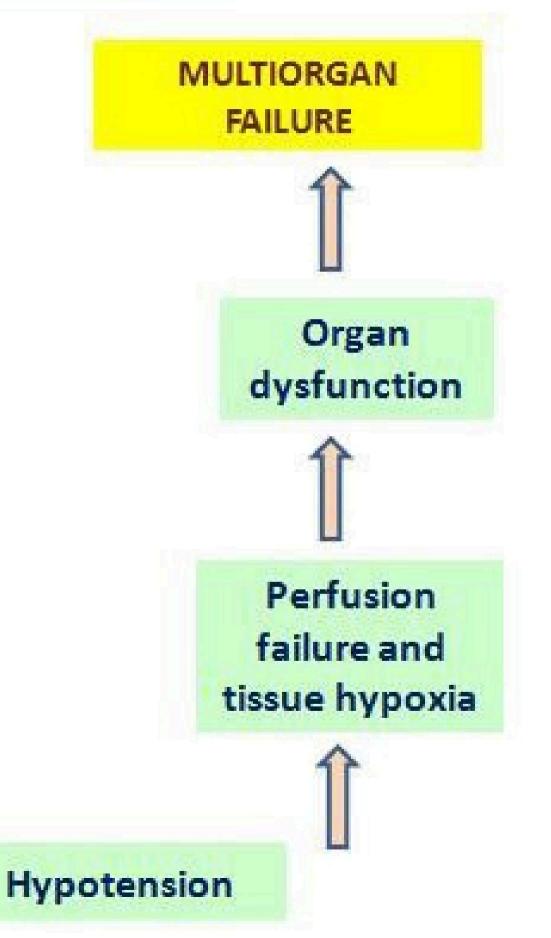
HYPOVOLEMIC Shock

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

PATHOPHYSIOLOGY OF HYPOVOLEMIC SHOCK



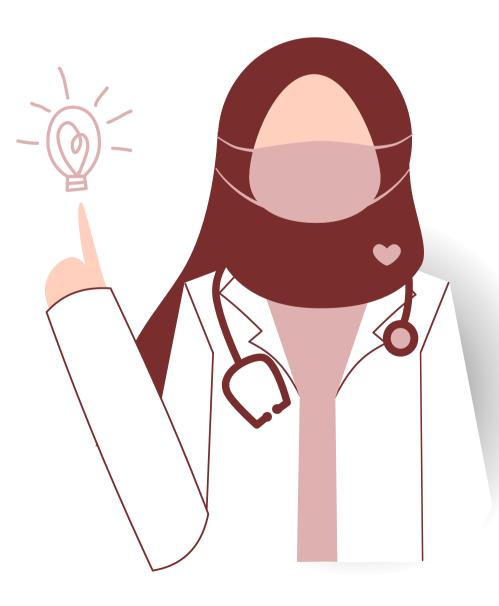






- CBC
- ABG
- Electrolytes
- Coagulation studies
- Type and cross-match
 - As indicated: CXR,
 - Pelvic x-ray, CT, GI endoscopy, Vascular radiology

lies atch (R, GI lar



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

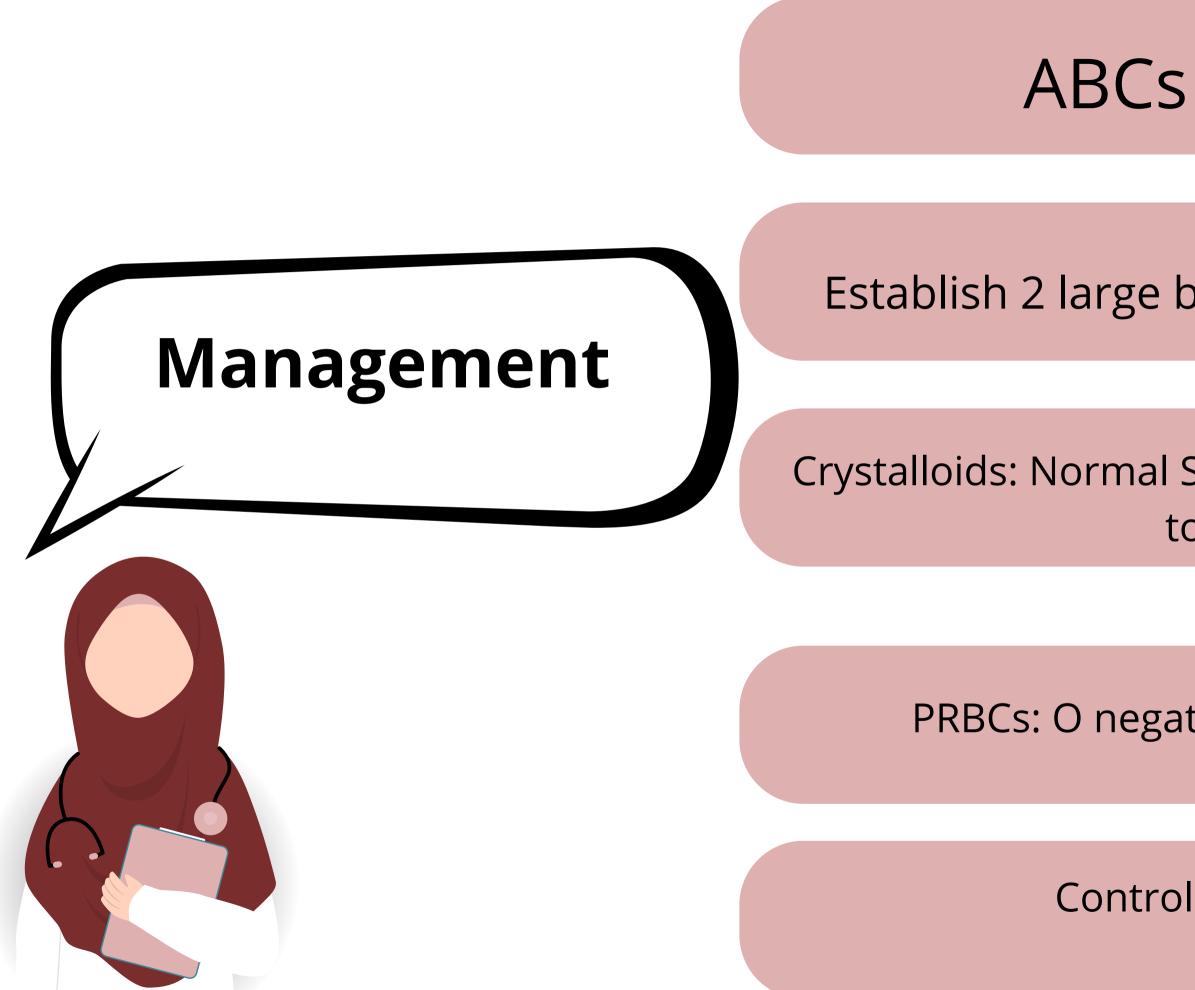
Arterial Blood Gas (ABG)

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



Complete Blood Count Normal Range*

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



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

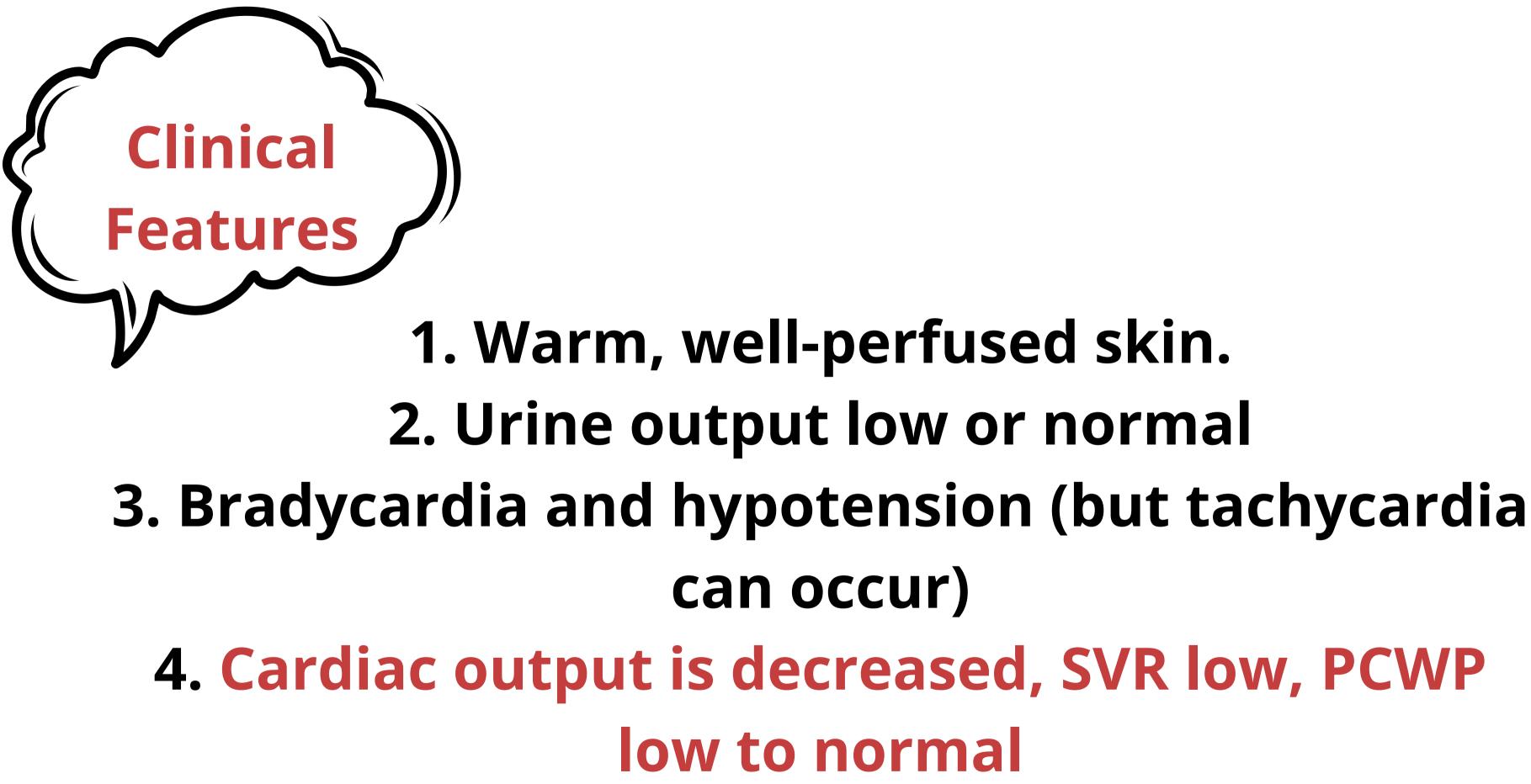
Control any bleeding

NEUROGENIC SHOCK



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



Treatment

Judicious use of IV fluids as the mainstay of treatment

Vasoconstrictors to restore venous tone, but cautiously



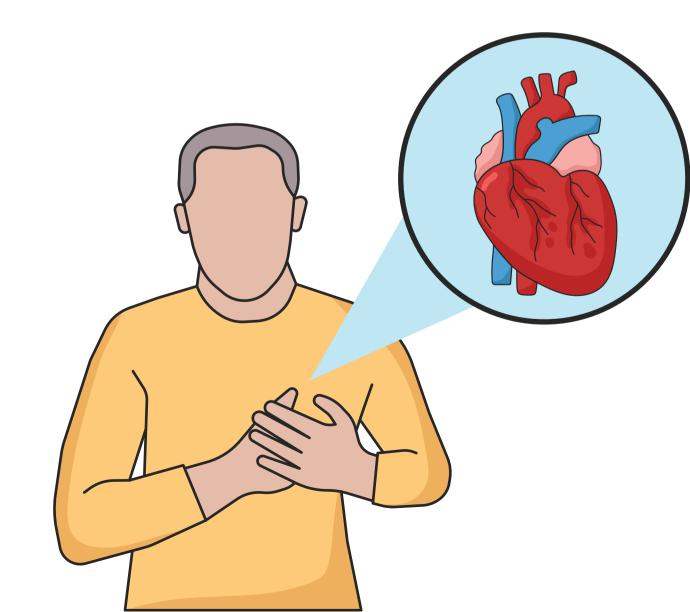


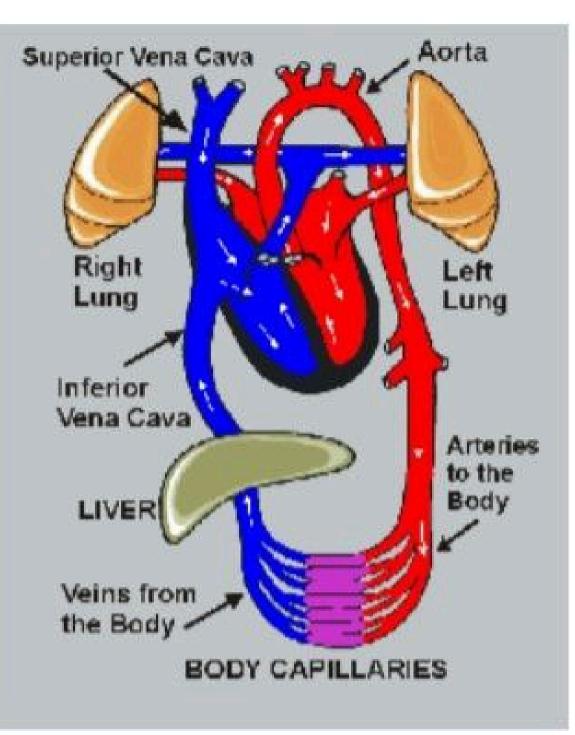
- Cardiogenic shock is a life-threatening condition in which your heart suddenly can't pump enough blood to meet your body's needs.
- It may be caused by:

myocardial damage (infarction).

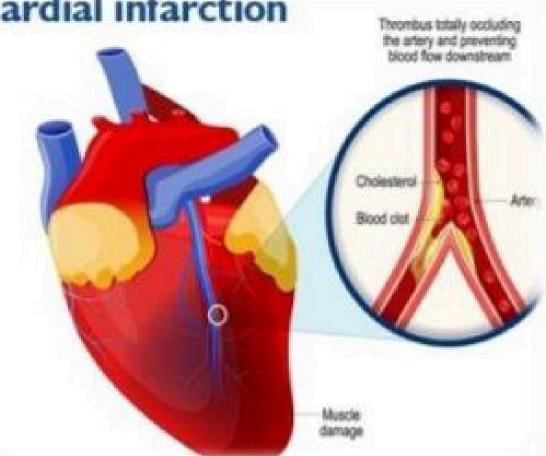
extrinsic compression (cardiac tamponade).

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





Myocardial infarction



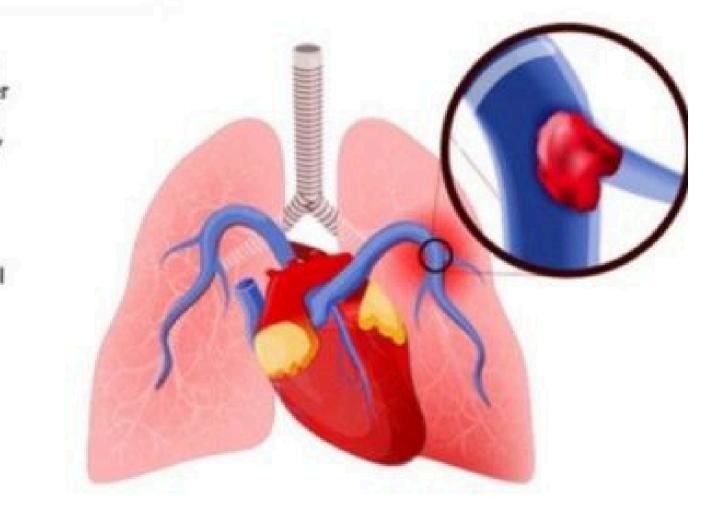
Pericardial sac

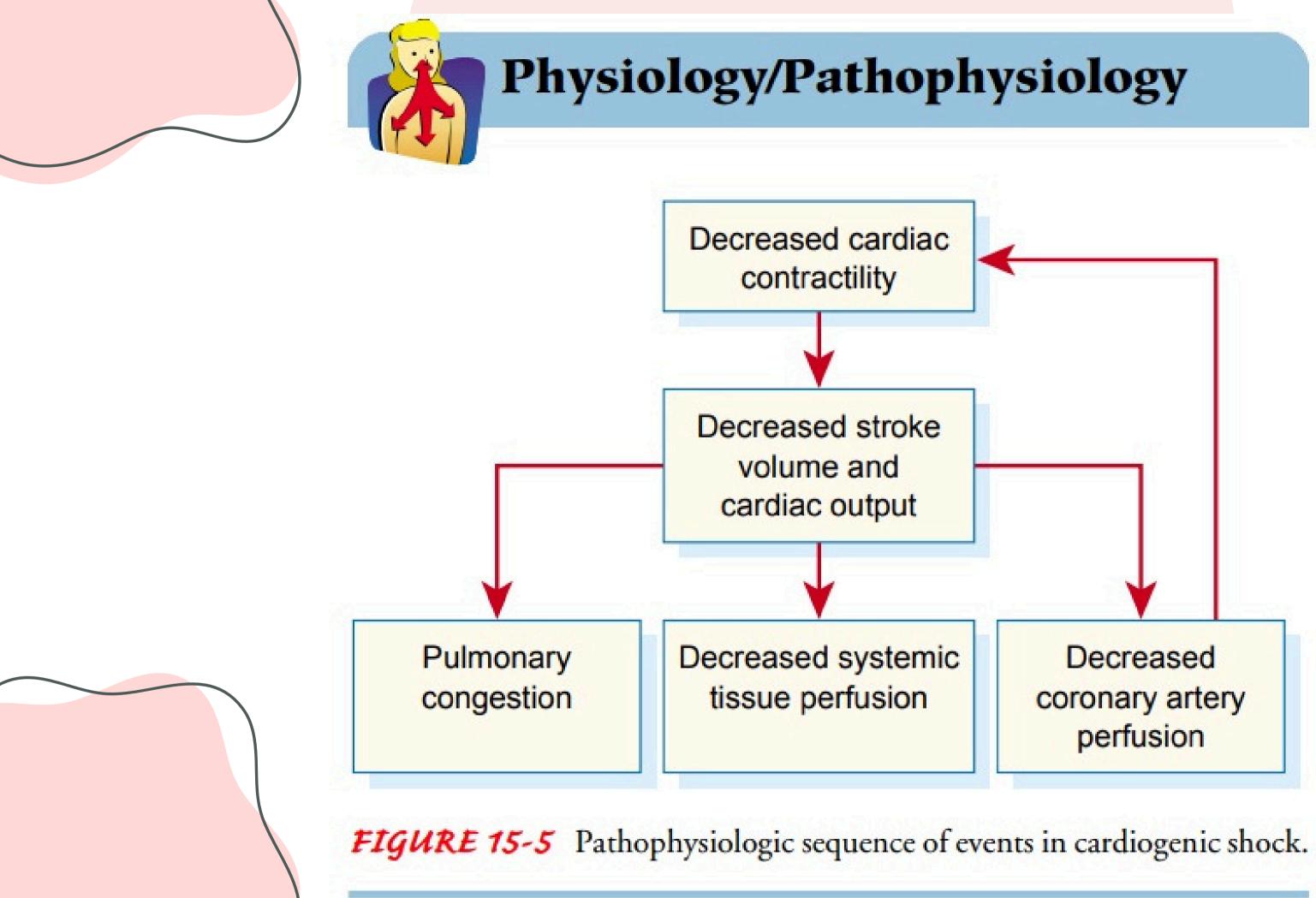
Cardiac Tamponade

A build-up of blood or other fluid in the pericardial sac puts pressure on the heart, which may prevent it from pumping effectively.

Fluid build-up within pericardial sac

Pulmonary embolism

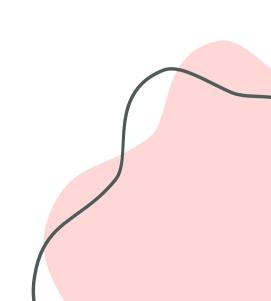








Decreased coronary artery perfusion



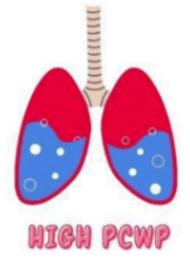
SYMPTOMS & SIGN

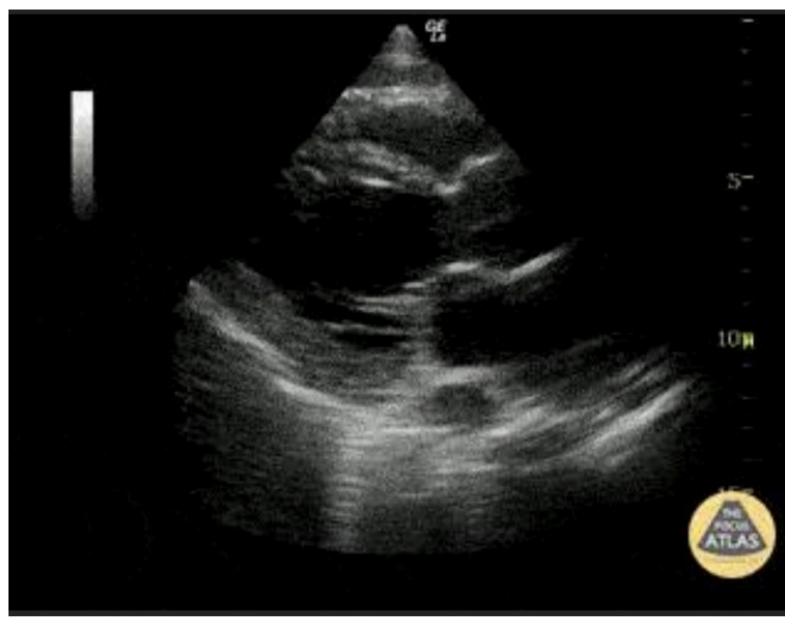
1. Cool, mottled skin 2•Tachypnea **3**•Hypotension **4**•Altered mental status 5•Murmur











On Echo:

.We notice frank LV systolic dysfunction

ventricular fibrillation. of the cardiogenic shock.

Irregular heart rates (arrhythmias), such as ventricular tachycardia or These arrhythmias may be the cause



On ECG:

Treatment

1.Cardiac monitor, pulse oximetry

2.Supplemental oxygen, IV access 3.Catheterizatio n if ongoing ischemia

4.Preload augmentation: Consider Fluids 5.Contractility: Dopamine, dobutamine

6.Afterload reduction : Nitroglycerin, Dobutamine

1. Airway stability 2. improving myocardiak

Goals:

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

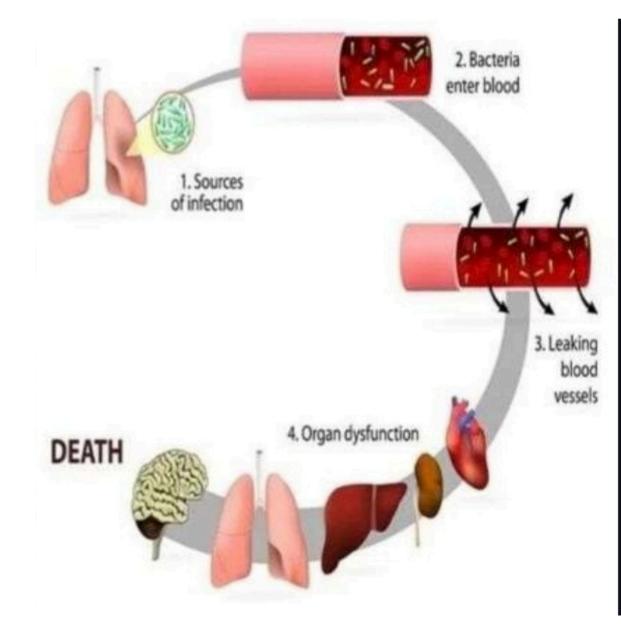


Septic shock

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.

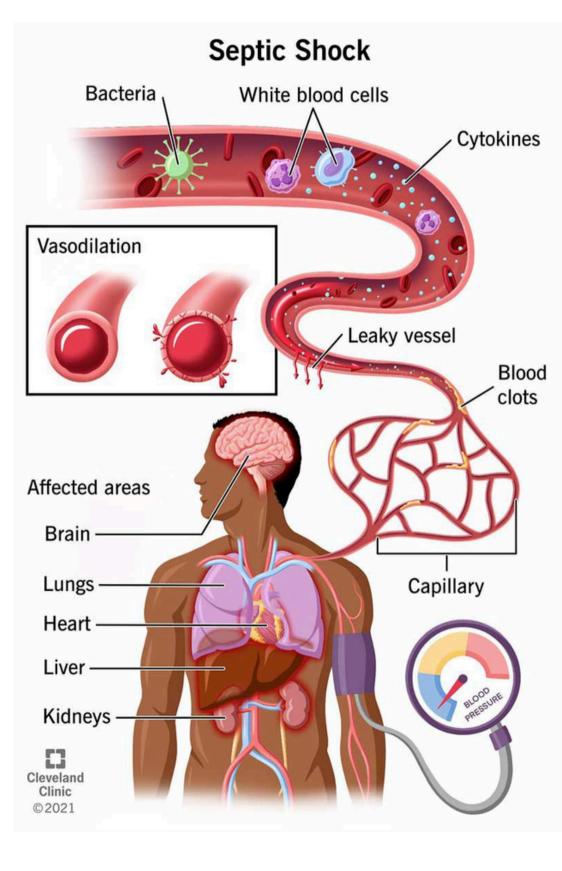




Pathophysiology

- 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





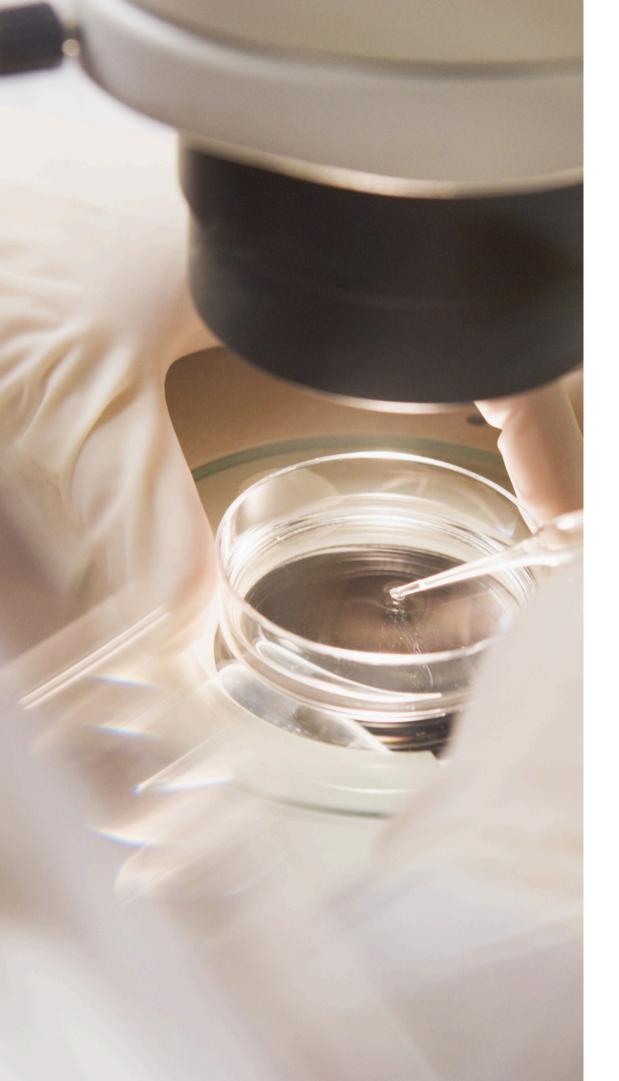


- Clinical signs:
- immunocompromised)
- Tachycardia
- Wide pulse pressure
- Low blood pressure
- Mental status changes
- Diagnosis:

cases.

• Hyperthermia or hypothermia (hypothermia is more common in the very young, elderly, and

• Septic shock is essentially a clinical diagnosis. • A source of infection can aid in diagnosis, but there may be no confirmed source in some



Treatment:

(Goal: MAP > 60) Start IV antibiotics (broad spectrum) at 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



1. Fluid administration to increase mean BP

2. Obtain cultures prior to starting antibiotics. maximum dosages. If cultures are positive,

