Shock

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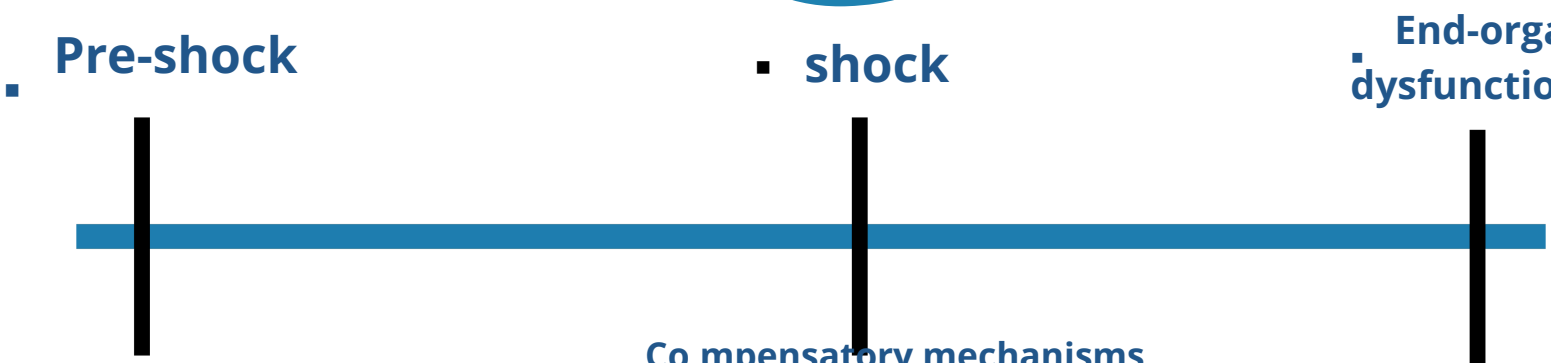
CONTENTS:

- DEFINITION
- DIAGNOSTIC FACTOR& INVESTIGATION
- STAGES & COMMON FEATURES
- TYPES

Defnetion

- **Shock is a life-threatening manifestation of circulatory failure that leads to cellular and tissue hypoxia cause by inadequate oxygen delivery that is unable to meet cellular metabolic needs and oxygen consumption requirements**
- **The effects of shock are initially reversible, but rapidly become irreversible, resulting in multi-organ failure (MOF) and death.**

STAGES OF SHOKE



- **Pre-shock**
- **shock**
- **End-organ dysfunction**

-Early, compensated shock

-symptoms are absent or mild

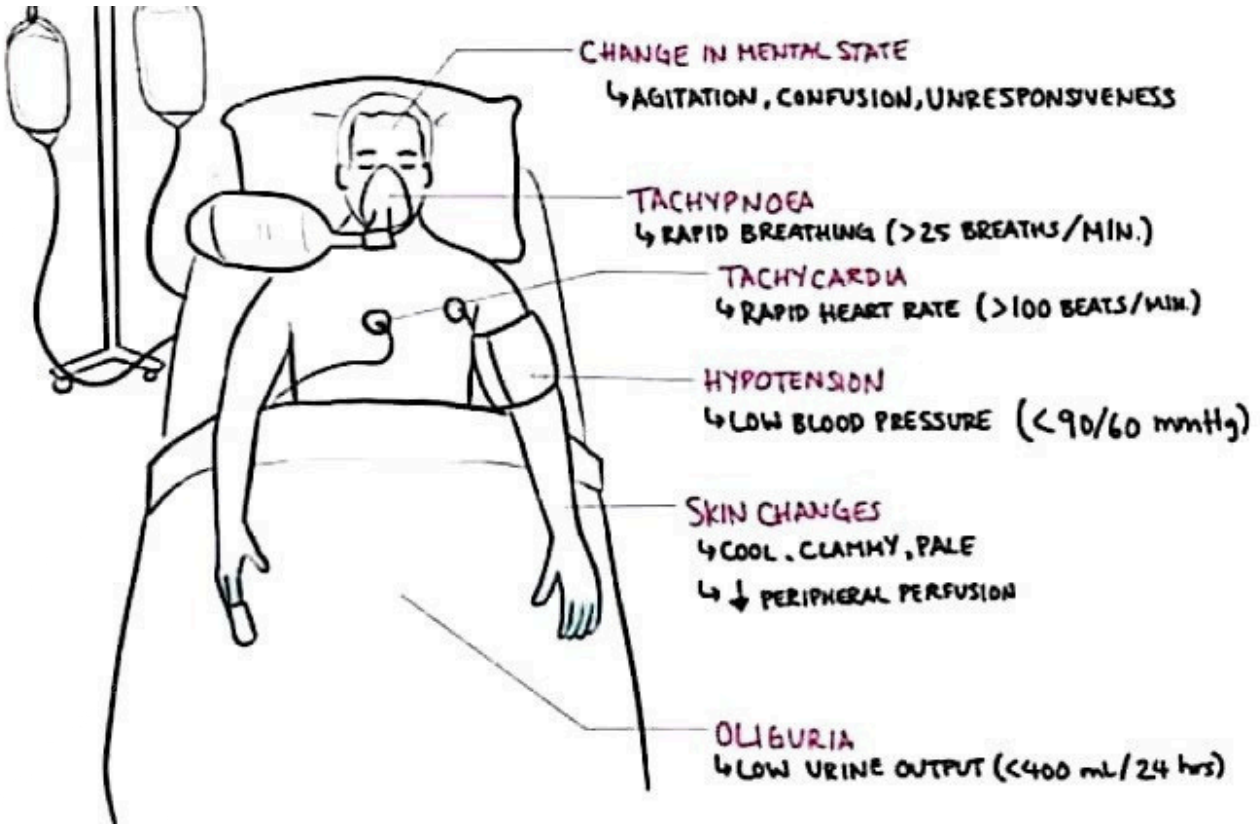
Co_mpensatory mechanisms become overwhelmed

-symptoms of organ dysfunction begin to apper

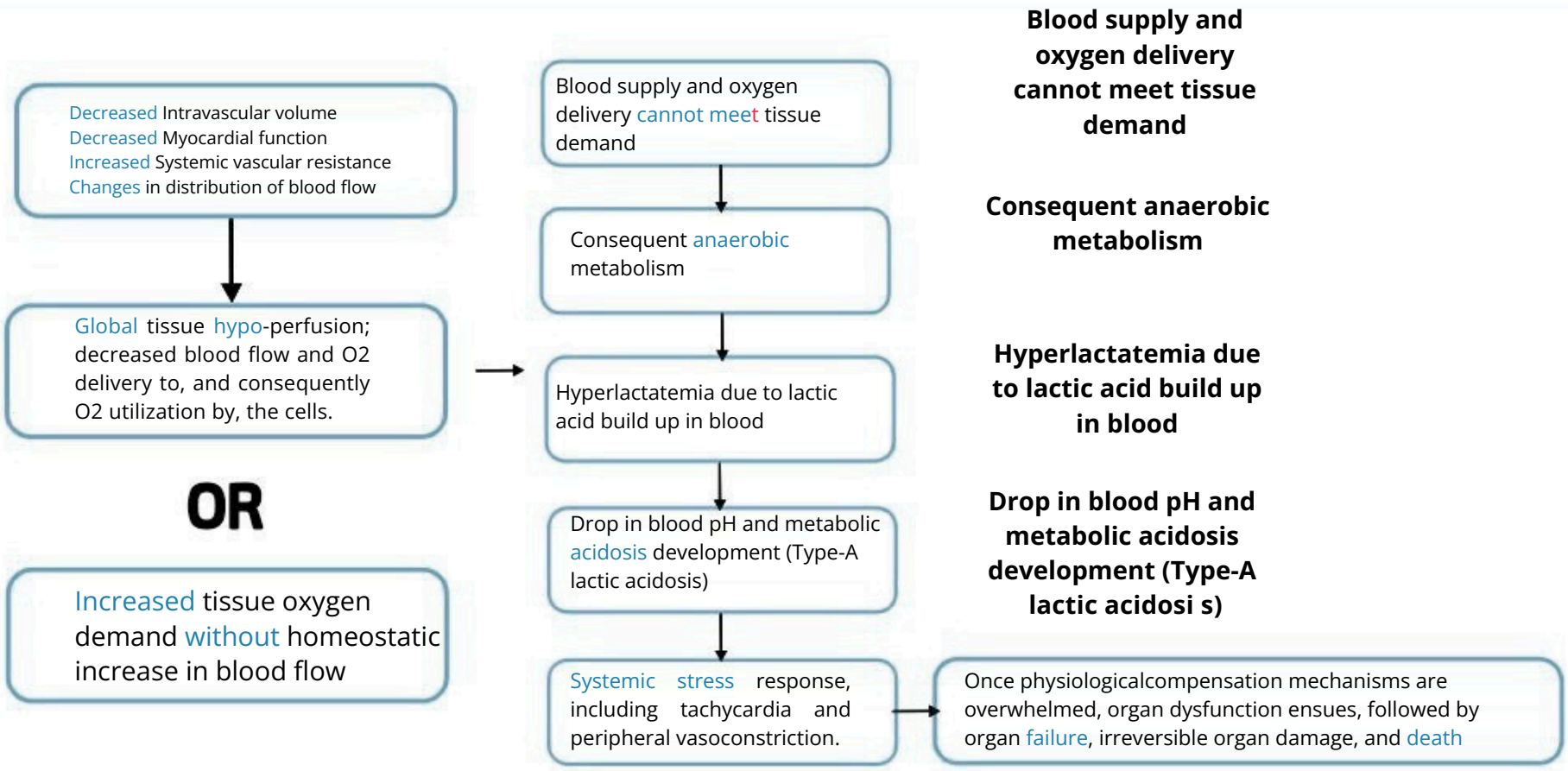
-Progressive shock

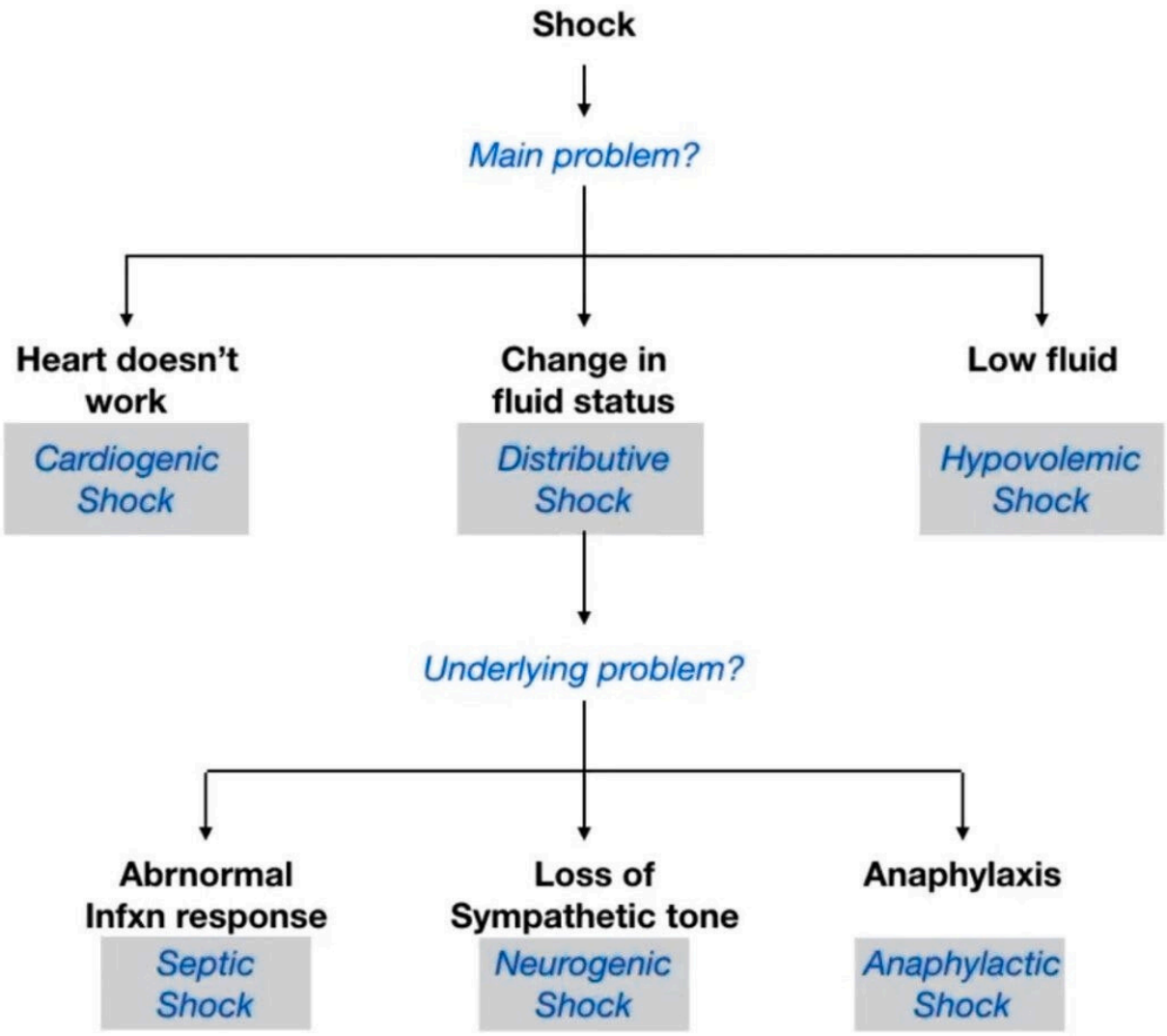
-Leads to irreversible organ damage and death

Clinical features



Pathophysiology




Shock
Main problem?
Heart doesn't work
Cardiogenic Shock
Change in fluid status
Distributive Shock
Low fluid
Hypovolemic Shock
Underlying problem?
Abrnormal Infxn response
Septic Shock
Loss of Sympathetic tone
Neurogenic Shock
Anaphylaxis
Anaphylactic Shock

Key diagnostic factor

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-positiverisk factor(historyofsepsis,recentMI, historyofhemorrhage, 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 PaO_2 due to the need of compensatory hyperventilation of the generated metabolic acidosis)

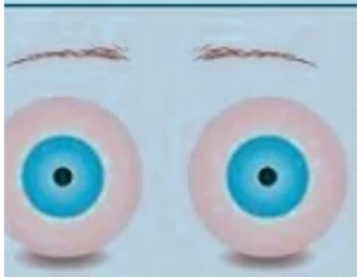
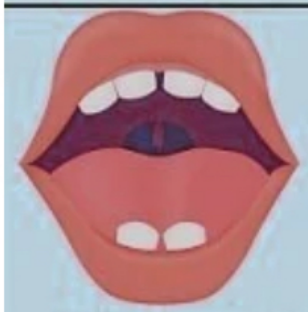
9-Fever (suggests septic shock).

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

Total GOS = E+V+M
Maximum = 15 (fully conscious)
Minimum = 3(deep shock)
13-15 —> mild brain injury
9-12 -> moderate brain injury
<=8 —> severe brain injury (coma, usually requires airway protection)

Behaviour	Response
 Eye Opening Response	4. Spontaneously 3. To speech 2. To pain 1. No response
 Verbal Response	5. Oriented to time, person and place 4. Confused 3. Inappropriate words 2. Incomprehensible sounds 1. No response
 Motor 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.
- 4.Blood test:
  - CBC (result: Hb < 10g/dL suggests hemorrhage, WBC >12 x 10^3/ macro-liter if sepsis is present.)
  - Urea and electrolytes (evidence of renal impairment if kidney perfusion is compromised for 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)
- 5.ECG (evidence of MI, arrhythmias, electrolyte abnormalities)
- 6.CXR: look for pulmonary oedema, pneumonia, pneumothorax, widened mediastinum (e.g., due to aortic dissection).

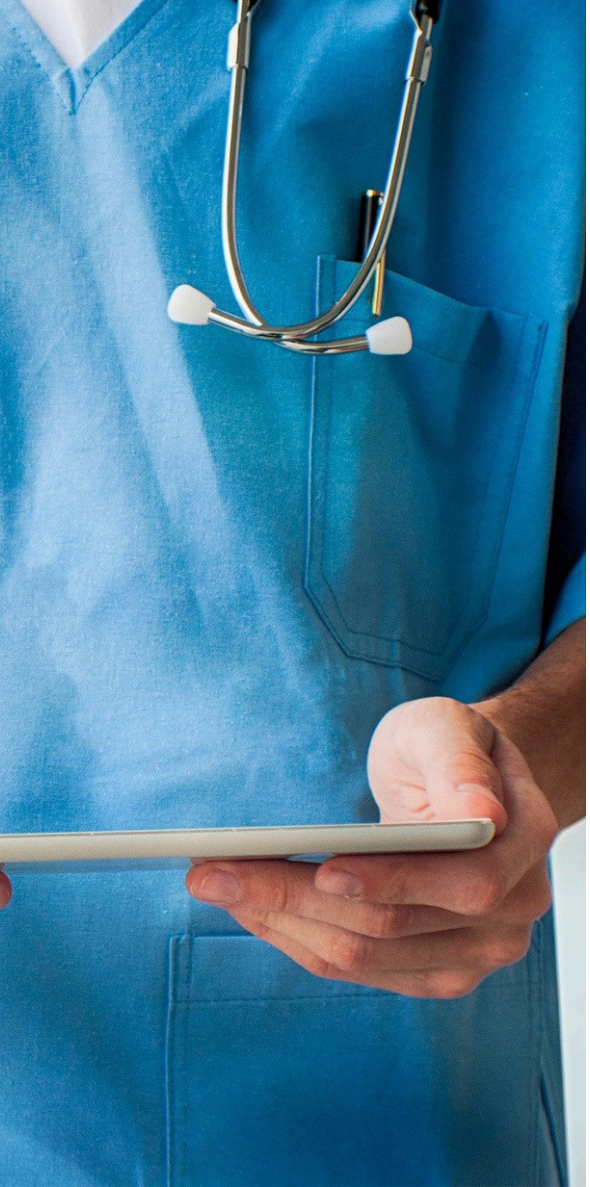
NB:

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	Assessment	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 .
Breathing	• 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
Disability	+ Temperature + Blood sugar + Pupils + Pain	+ Glucose supplements + Temperature management + pain management
Exposure	+ Perform head to toe examination , front and back	+ Manage abnormal finding appropriately

TYPES



Anaphylactic Shock

Anaphylactic shock is a severe, life-threatening allergic reaction that happens when the immune system overreacts to an allergen
It causes the sudden release of chemicals like histamine leading to widespread vasodilation (low blood pressure), leaky blood vessels (swelling), and airway constriction (breathing difficulty).

IgE mediated

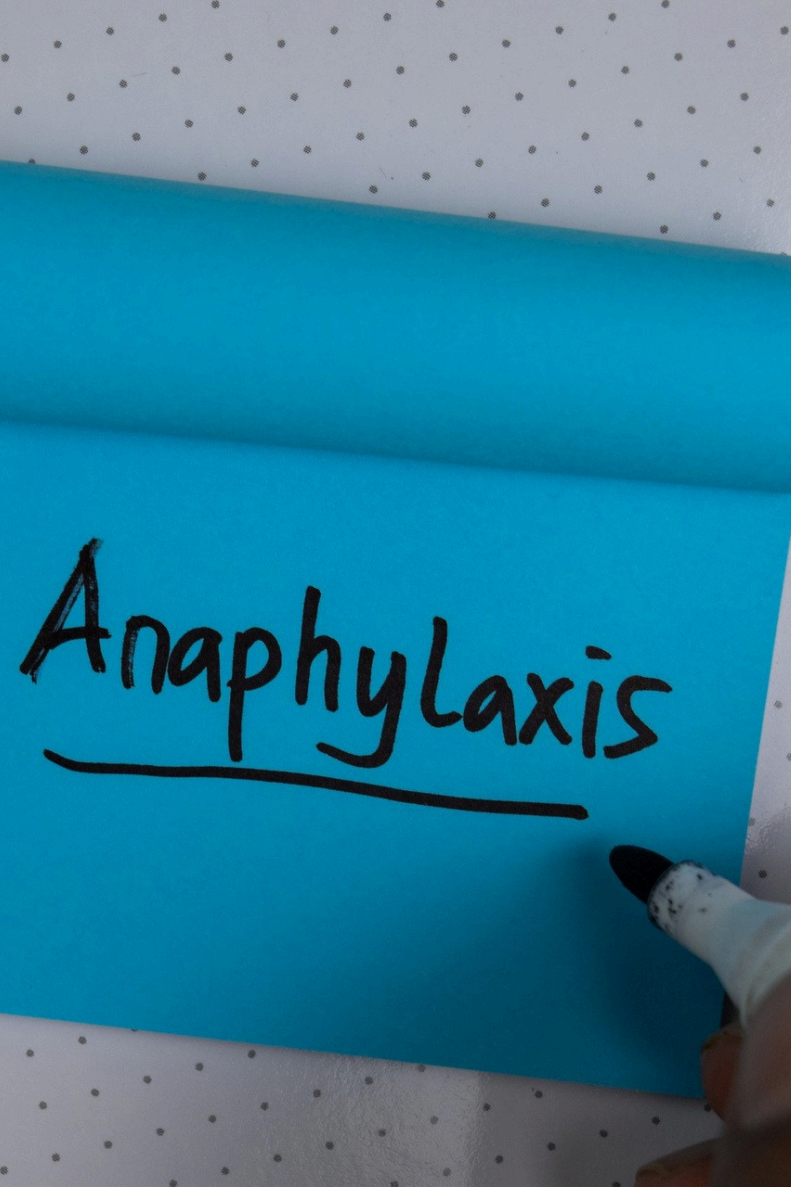
1. Allergen entry: ingestion, inhalation, parenteral, or skin contact.
2. Formation of immunoglobulin E (IgE) antibodies specific to the antigen presented on first exposure.
3. IgE antibodies attach to high-affinity Fc receptors on basophils and mast cells.
4. On subsequent exposure, binding of antigen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

Non IgE mediated

1. Activation of complement system
2. The complement peptides (anaphylatoxins) such as C3a and C5 directly act on mast cells and basophils leading to mediator release

Idiopathic

1. Increased mast cell sensitivity and degranulation
2. Unrecognised allergens



Signs and 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**

Possible causative agent

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

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

🔷 **2. Anaphylaxis with cardiac arrest (cardiorespiratory arrest)**

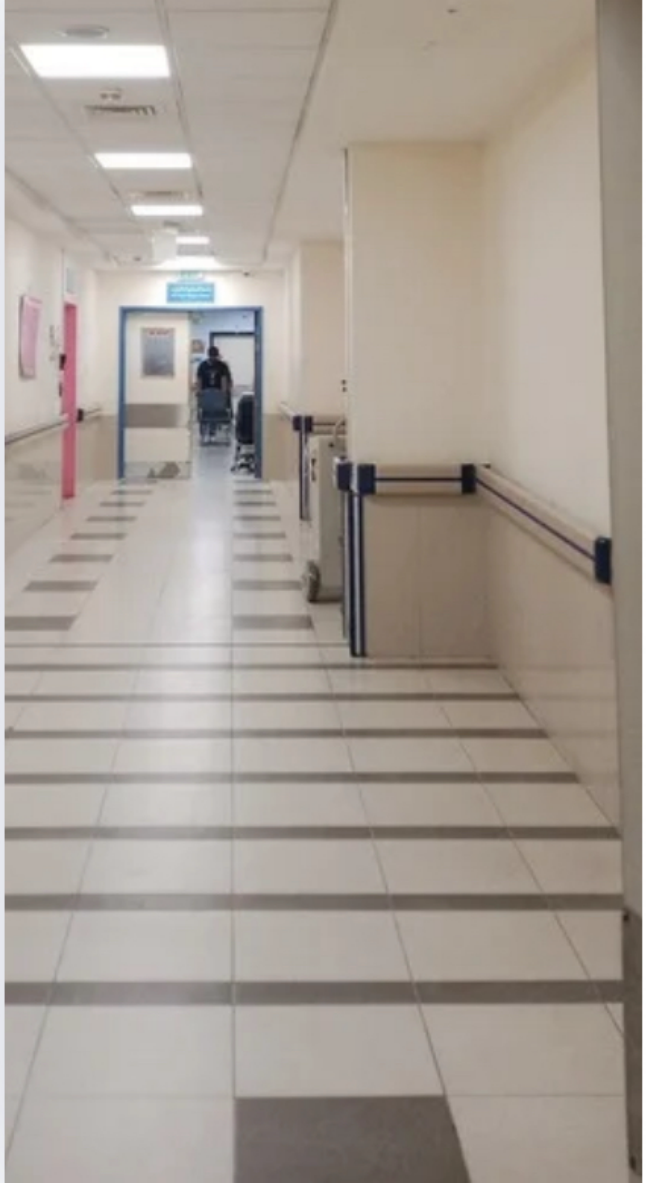
- Follow **Advanced Life Support (ALS/ACLS)** protocols.
- **IV adrenaline (1:10,000 = 0.1 mg/mL solution)**
- **Adult dose:**
  - **1 mg IV/IO every 3–5 minutes** during CPR
- **Child dose:**
  - **0.01 mg/kg IV/IO** (0.1 mL/kg of 1:10,000 solution)
  - Repeat every **3–5 minutes**

💡 Here IV is used because circulation is absent, so IM won’t distribute.

🔷 **1. Anaphylaxis without cardiac arrest (non–cardiorespiratory arrest)**

- **IM adrenaline is first-line.**
- **Adult dose:**
  - **0.5 mg IM** (0.5 mL of 1:1000 = 1 mg/mL solution)
  - Into mid-anterolateral thigh
  - Repeat every **5 minutes** if needed
- **Child dose:**
  - **0.01 mg/kg IM** (max 0.3–0.5 mg per dose)
  - Repeat every **5 minutes** if needed

💡 IM route is preferred because it gives rapid absorption and is safer than IV outside ICU.



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) example is Salbutamol

Dose (Nebulized):

- **Adult:** 2.5–5 mg via nebulizer every 20 minutes for up to 3 doses, then as needed
- **Child:** 2.5 mg via nebulizer every 20 minutes for up to 3 doses, then as needed
- Delivered with **oxygen-driven nebulizer** (6–8 L/min flow)

• 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:**

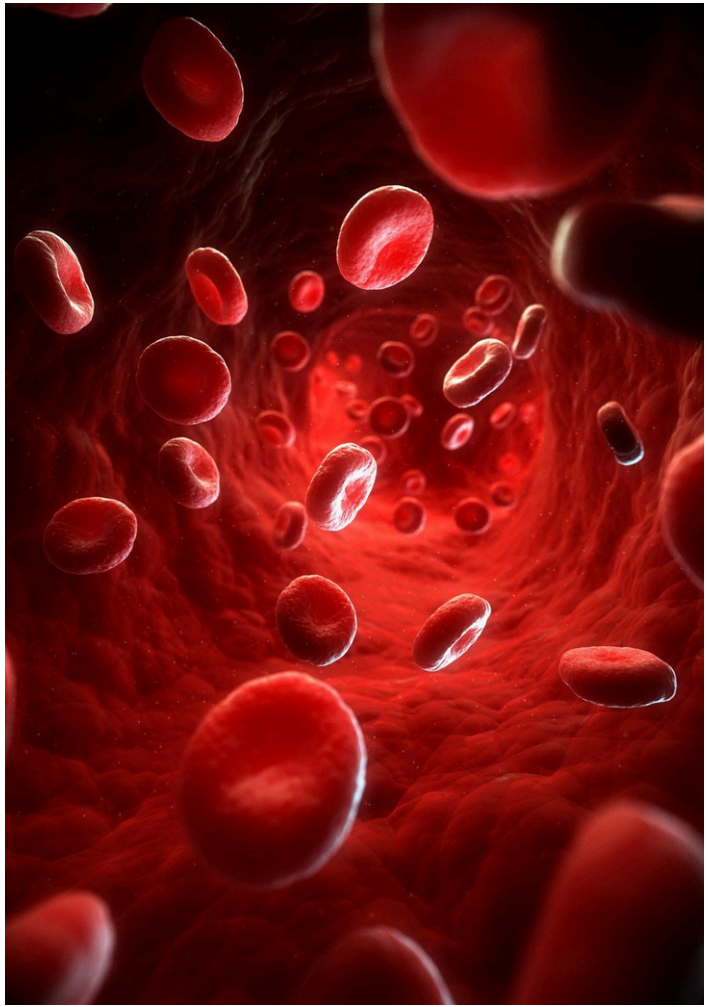
1. Antihistamine (diphenhydramine 25-50 mg IV or IM,)

2. Corticosteroids (hydrocortison 200mg IV every 6 hours for adult)

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.



HYPOVOLEMIC Shock

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

HYPOVOLEMIC Shock

***Causes:**

a) Non-hemorrhagic

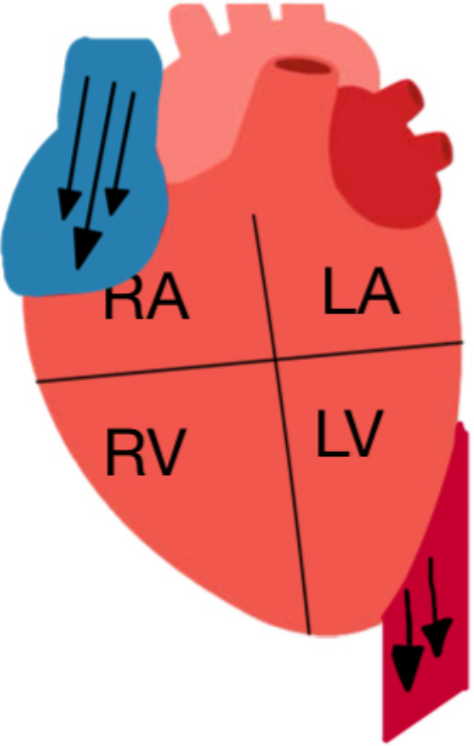
- ❖Vomiting
- ❖Diarrhea
- ❖Bowel obstruction
- ❖Burns
- ❖Dehydration

b) Hemorrhagic

- ❖GI bleed
- ❖Trauma
- ❖Massive hemoptysis
- ❖post-partum bleeding

***sign :**

1)Decrease CO
2)Decrease SV
3)increase HR.
4)increase SVR



~Co = SV * HR	~BP=SVR * SV
-increase HR	-increase SVR
-decrease SV	-decrease BP
-Decrease Co	-decrease SV
	—

Why?

-decrease co and SV ?

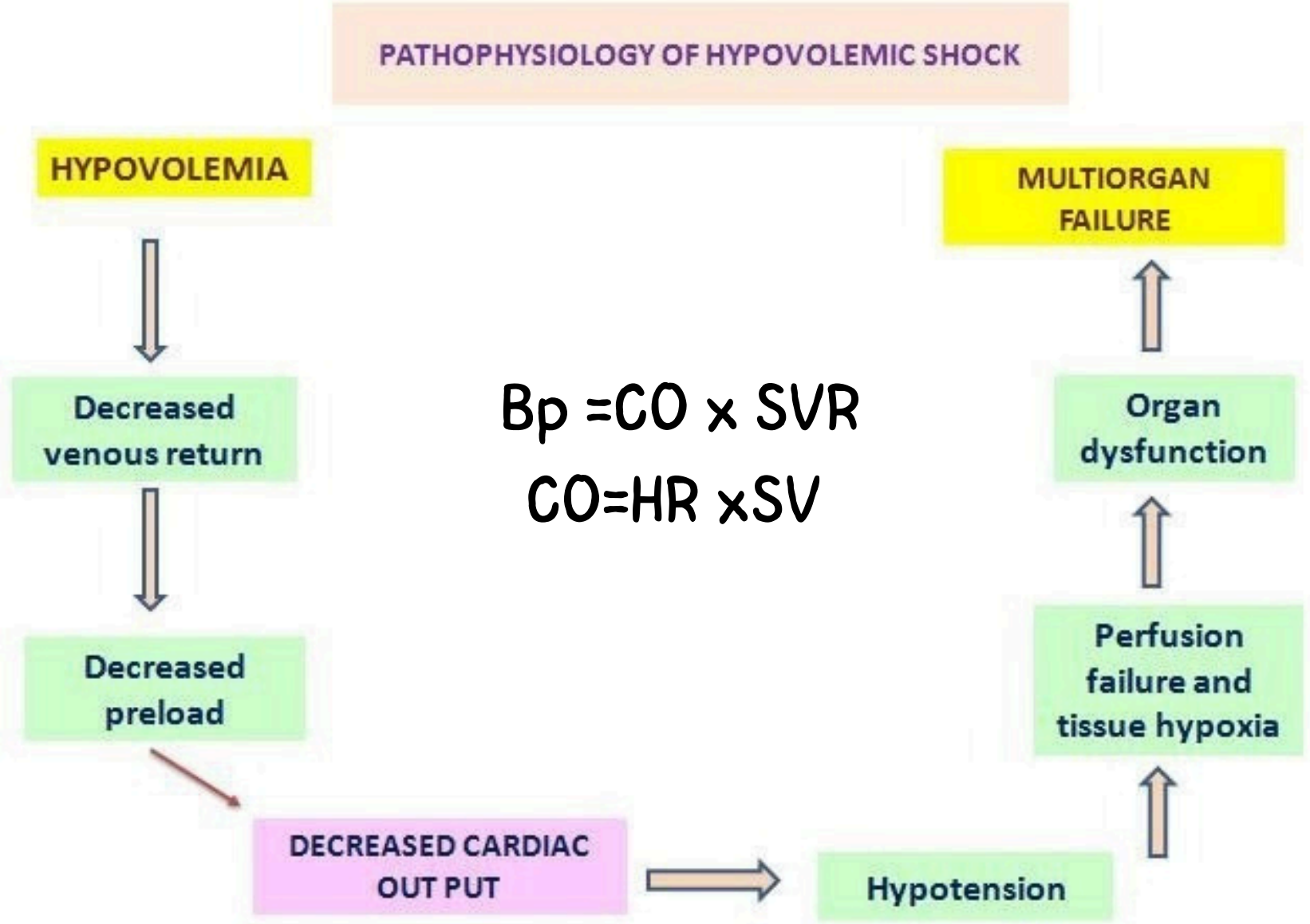
It results due to decrease VR
that will lead to decreased LV Filling
decrease preload leads to decrease SV

-increase HR?

1)Compensation to maintain Co

increase SVR?

1)Sympathetic nervous system (SNS) → norepinephrine release.
2)Renin–angiotensin–aldosterone system (RAAS) → angiotensin II (vasoconstriction).
3)Redirect blood flow to vital organs


PATHOPHYSIOLOGY OF HYPOVOLEMIC SHOCK
HYPOVOLEMIA
Decreased venous return
Decreased preload
DECREASED CARDIAC OUT PUT
Hypotension
Perfusion failure and tissue hypoxia
Organ dysfunction
MULTIORGAN FAILURE
Bp =CO x SVR
CO=HR xSV

Evaluation

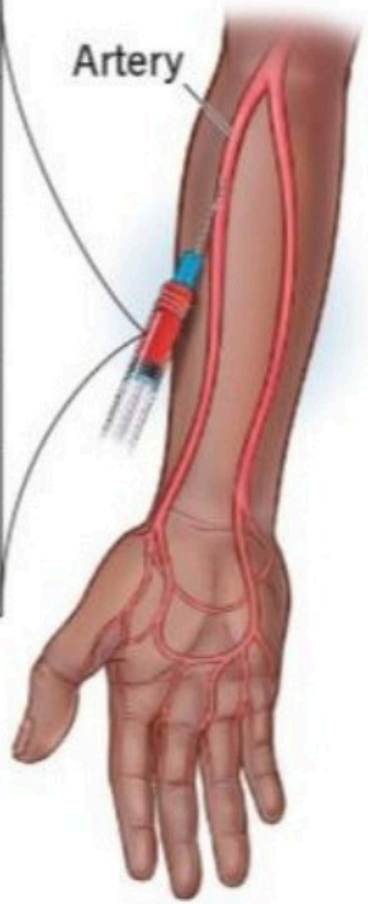
- CBC
- ABG
- Electrolytes
- Coagulation studies
- Type and cross-match
- As indicated: CXR, Pelvic x-ray, CT, GI endoscopy, Vascular radiology

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_2CT	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
O_2Sat	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

Management

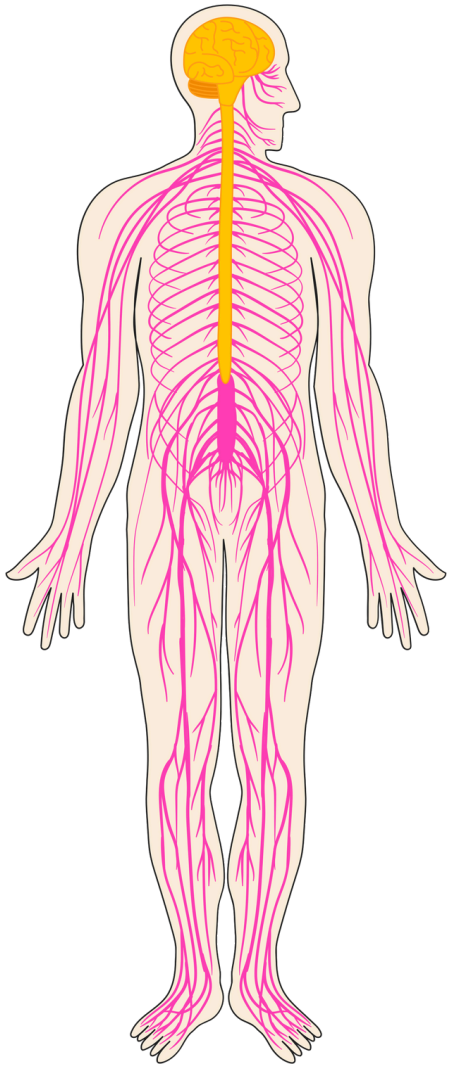
ABCs

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**

Clinical Features

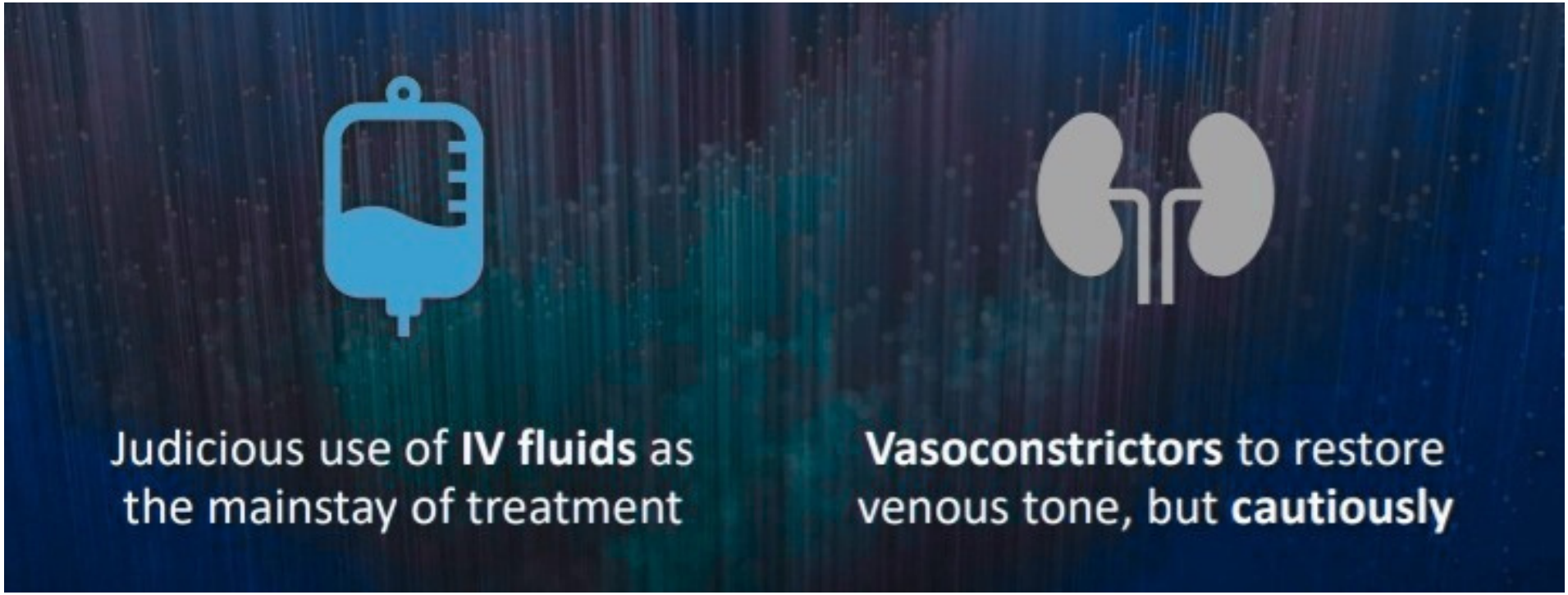
1. Warm, well-perfused skin. 2. Urine output low or normal

3. Bradycardia and hypotension (but tachycardia can occur)

4. Cardiac output is decreased, SVR low, PCWP low to normal

PCWP: pulmonary capillary widge pressure

Treatment


Judicious use of **IV fluids** as the mainstay of treatment
Vasoconstrictors to restore venous tone, but **cautiously**

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.

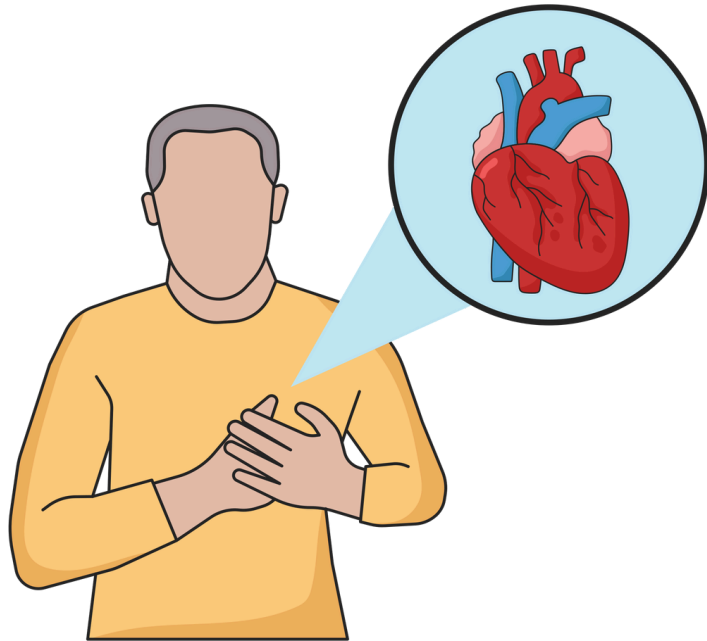
- **It may be caused by:**

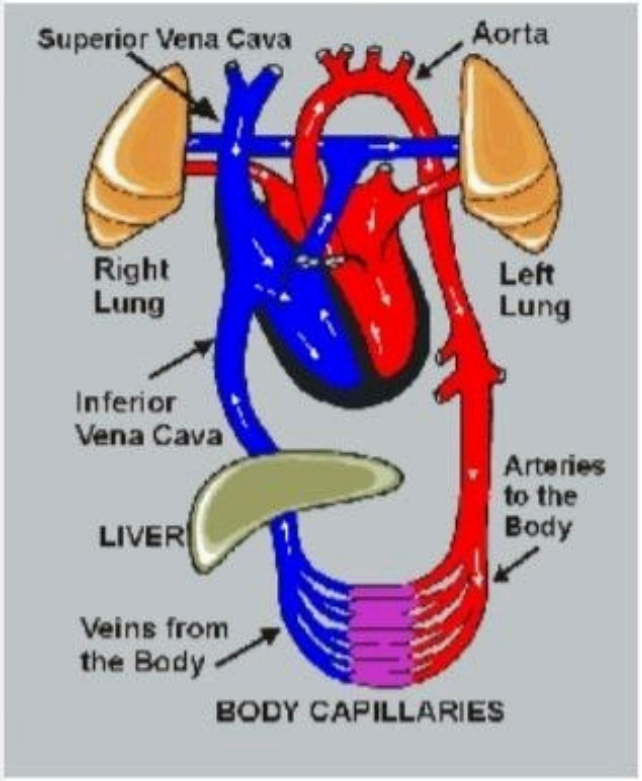
myocardial damage (infarction).

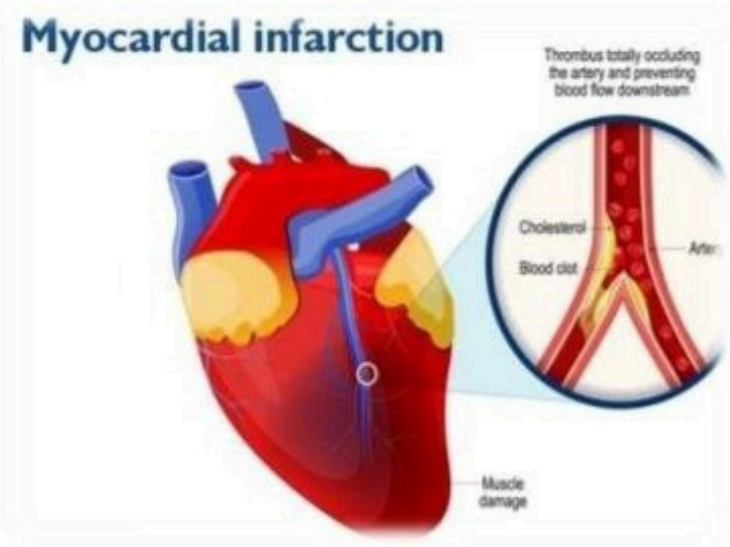
extrinsic compression (cardiac tamponade).

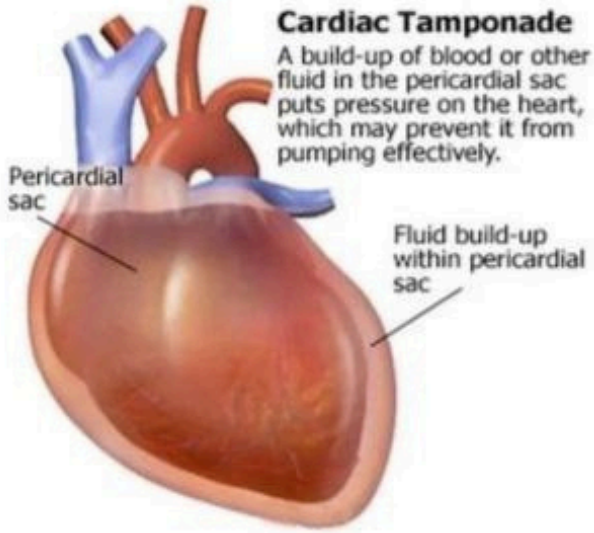
ventricular arrhythmias.

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

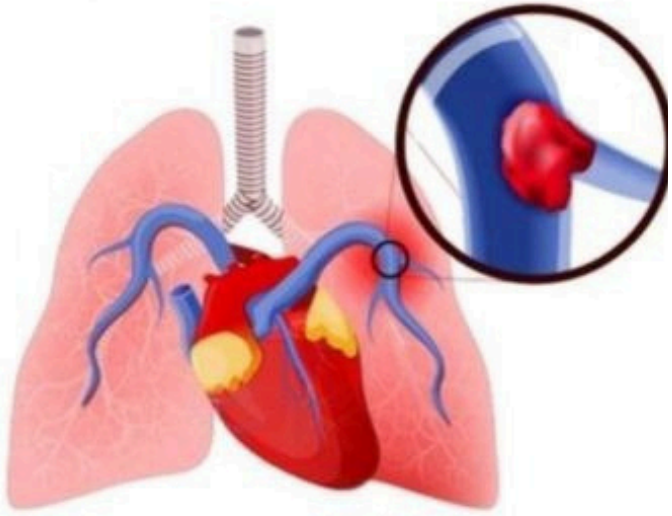








Pulmonary embolism



Physiology/Pathophysiology

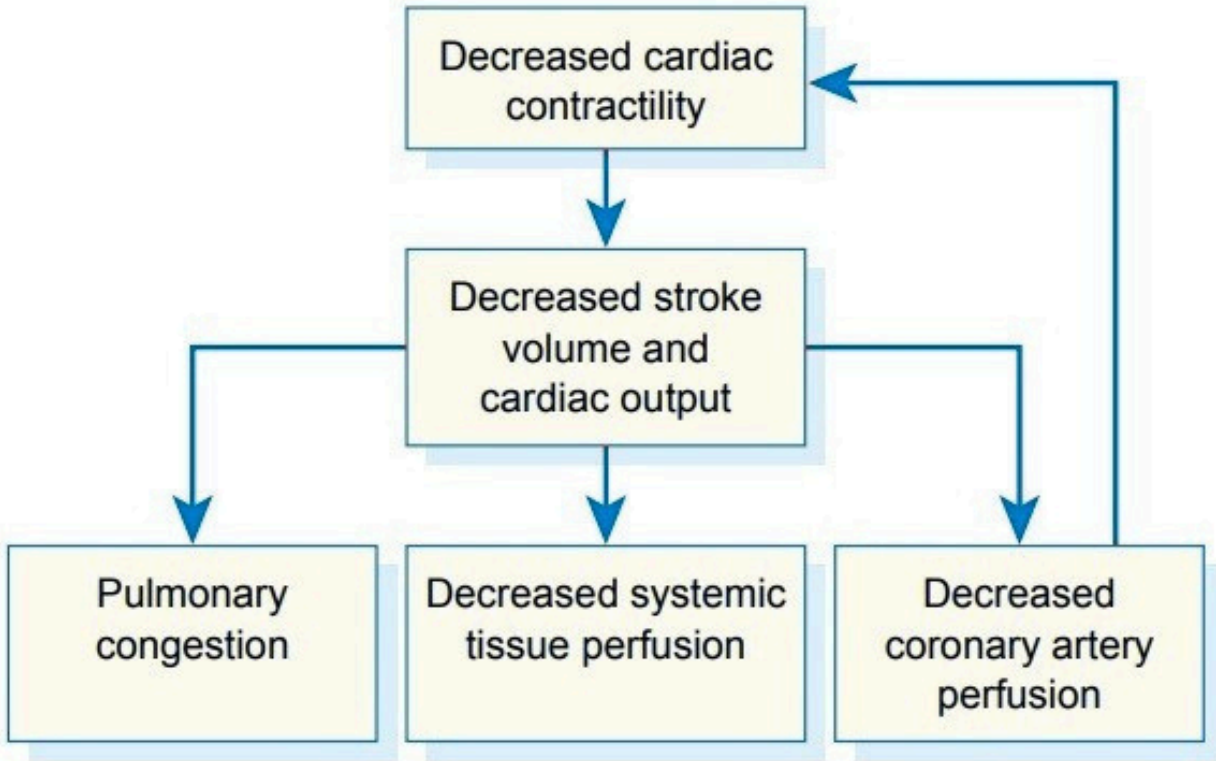
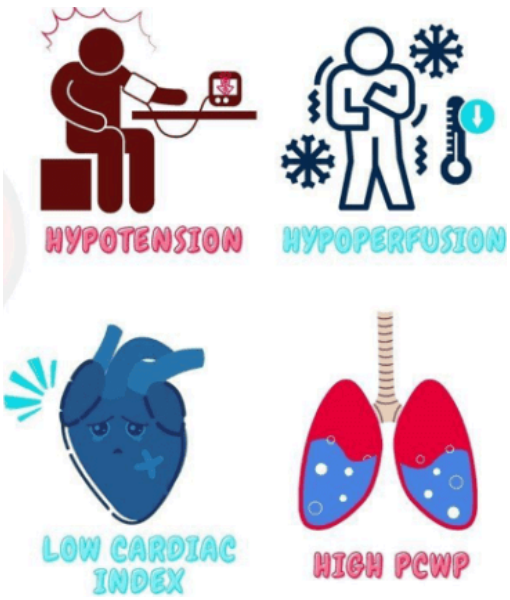
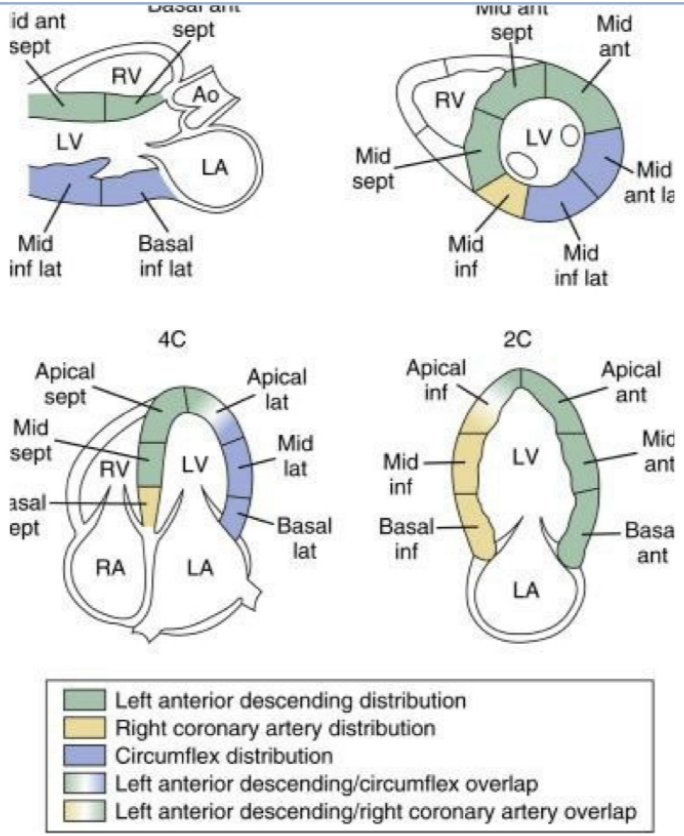


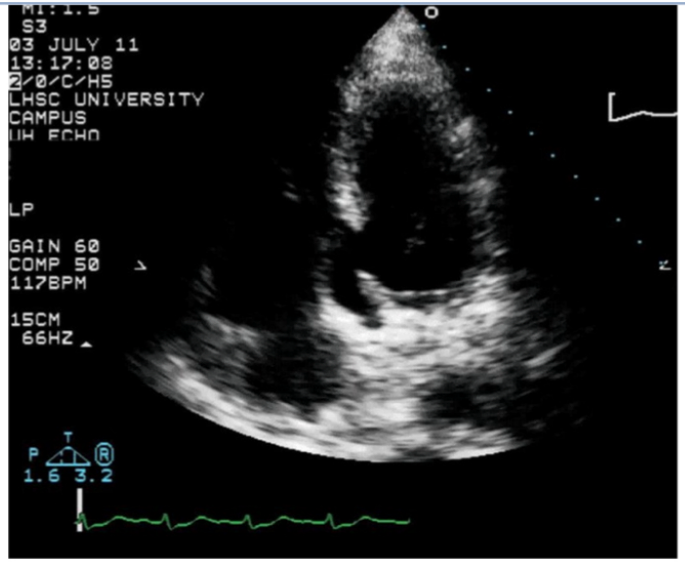
FIGURE 15-5 Pathophysiologic sequence of events in cardiogenic shock.

SYMPTOMS &SIGN

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

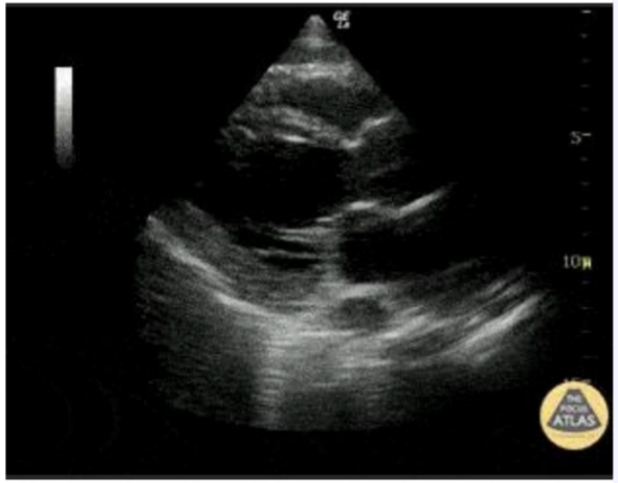






On Echo:

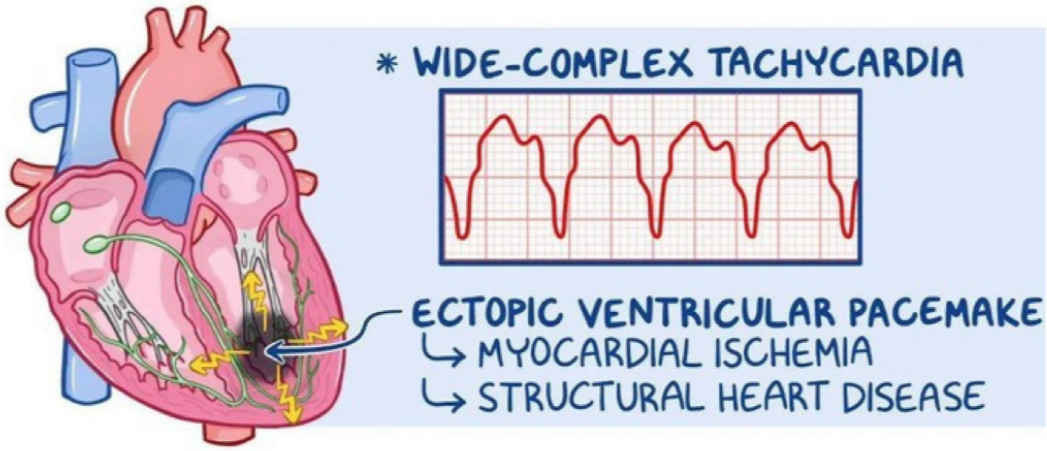
complication of MI

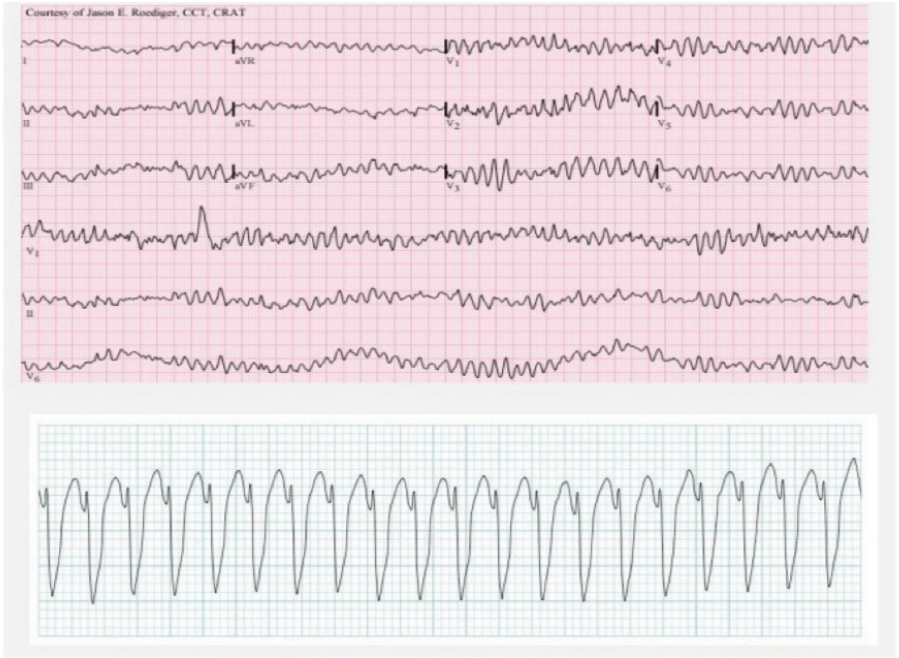


On Echo:

.We notice frank LV systolic dysfunction

VENTRICULAR TACHYCARDIA, VT, or V-TACH





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 myocardial

1.Cardiac monitor, pulse oximetry

2.Supplemental oxygen, IV access

3.Catheterization if ongoing ischemia

4.Preload augmentation: Consider Fluids

5.Contractility: Dopamine, dobutamine

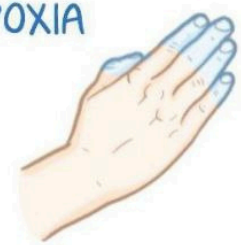
6.Afterload reduction : Nitroglycerin, Dobutamine

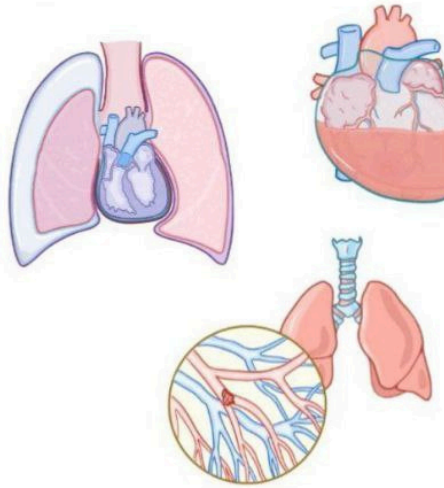
7.intra-aortic balloon pump(if inotropes&vasopressors fail

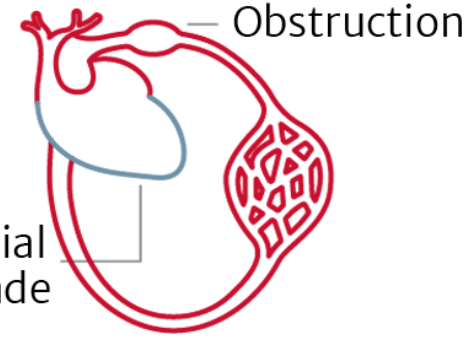
Obstructive Shock

OBSTRUCTIVE SHOCK

* MECHANICAL OBSTRUCTION PREVENTS HEART from FILLING PROPERLY or PUMPING enough BLOOD through CARDIOVASCULAR SYSTEM
  - ↓ AMOUNT of OXYGENATED BLOOD that REACHES TISSUES
    - ACUTE HYPOPERFUSION
    - TISSUE HYPOXIA



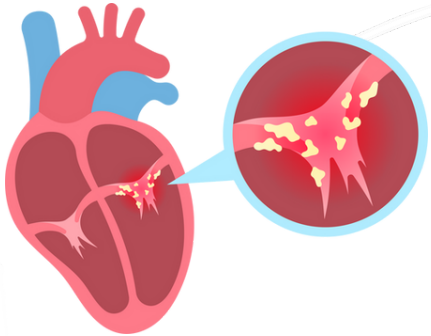




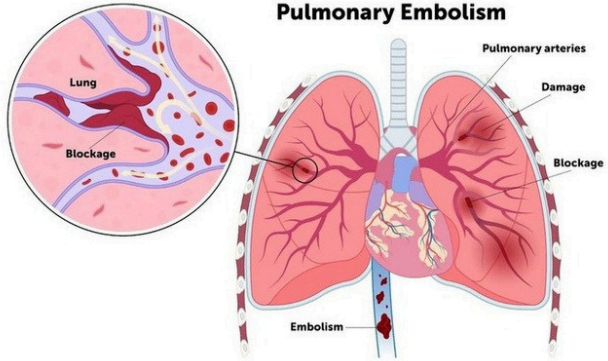
tension pneumothorax

*Usually manifests with elevated JVP

2% Obstructive shock



aortic stenosis



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

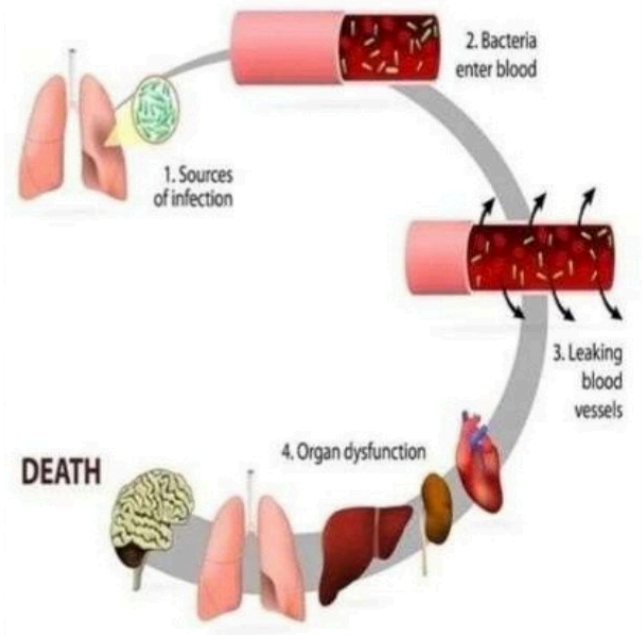
Septic shock

Septic shock

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

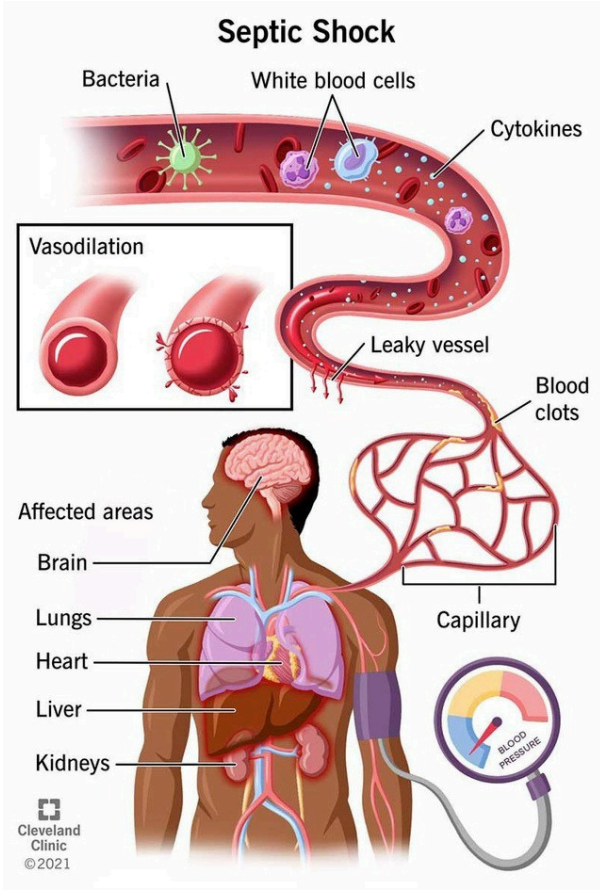
This results in hypoperfusion and can ultimately lead to multiple organ system failure and death.

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

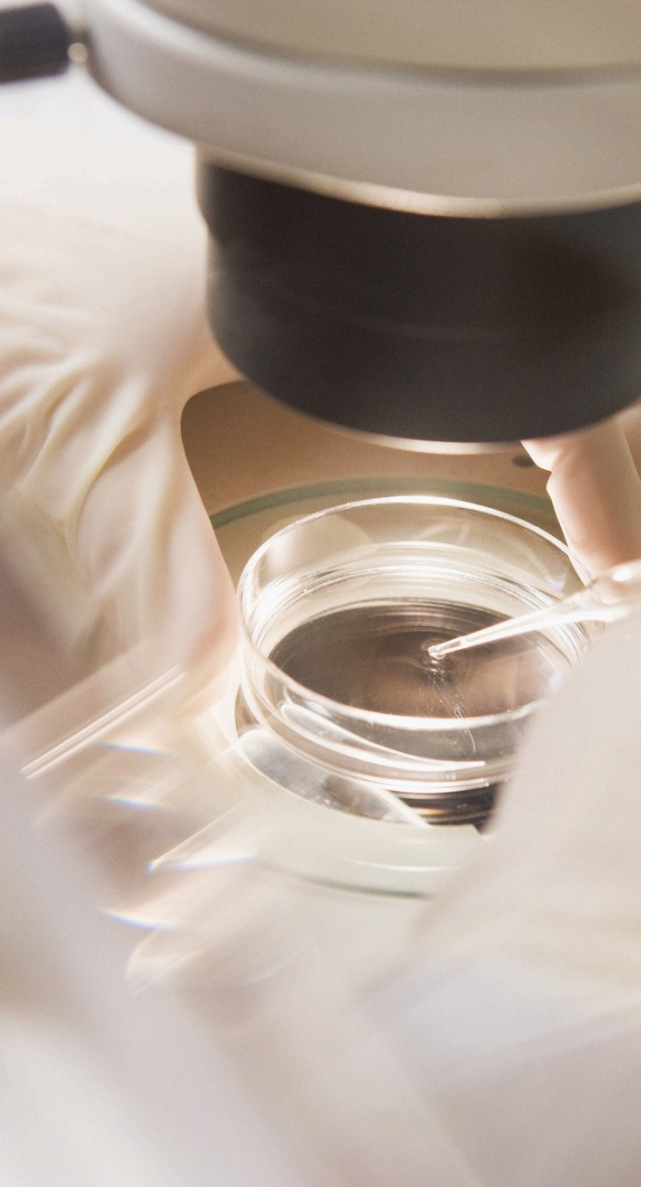


Pathophysiology

- There is a severe decrease in SVR secondary to peripheral vasodilation. Extremities are often warm due to vasodilation.
- 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:

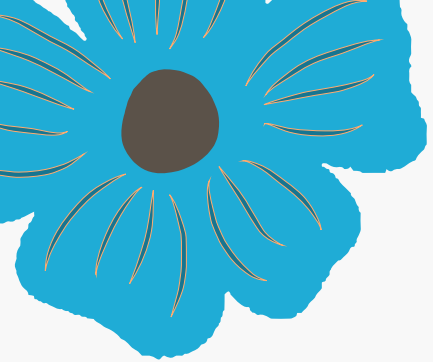
1.Fluid administration to increase mean BP (Goal:MAP >60)

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,dopamine) may be used if hypotension persists despite aggressive IV fluid resuscitation

5. Corticosteroid e.g dexamethason



سِنينُ الجَهدِ إن طَالتْ ستطوى
لها أمدٌ وللأمدِ انقضاءُ
لنَا بالله آمالٌ وسَلوى
وعندَ الله مَا خابَ الرّجاءُ
إذا اشتدّت رياحُ اليأس فينَا
سَيعقبُ ضيقِ شدّتهَا الرخَاءُ
فَبعد العتمَةَ الظَّلمِاء نورٌ
وطَول الليلِ يعقُبهَ الضياءُ

شكراً لكِ!