SHOCK

Supervision: Dr.Ashraf Al-dmor

Presentation:

Yaqut AL-Madadhah

Miami Hlalat

Farah Jaafrah

Yousef Abdallah

Yazan sawalhah

DEFINITION

•Shock is a life-threatening condition that occurs when the body is not getting enough blood flow due to acute circulatory failure that lead to inadequate Organ perfusion and cellular hypoxia.

(Oxygen demand>> > Oxygen Consumption)

•The effects of shock are initially

reversible, but rapidly becom

irreversible resulting in multi-organ

failure (MOF) and death.

Stages of shock

Initial stage:

Cellular, not clinically apparent

Compensatory stage:

Neural, hormonal, biochemical compensation to maintain homeostasis;

inadequate perfusion → autonomic nervous

system attempts to compensate

- Sympathetic nervous system vasoconstriction, ↑ contractility
- Release of catecholamines, vasopressin, angiotensin II

→ ↑ vasoconstriction, ↑ retention water, sodium

→ ↑ SVR, ↑ blood volume → ↑ BP → ↑ perfusion

Progressive stage:

Compensation fails,

requires aggressive interventions to prevent multiple organ dysfunction syndrome

Irreversible stage:

Decreased perfusion (vasoconstriction, decreased cardiac output) → anaerobic metabolism; profound hypotension, hypoxemia, organ failure; recovery unlikely

Common Features of Shock

- Hypotension (not an absolute requirement)
  - SBP < 90mm Hg, not seen in “preshock”
- Cool skin
  - Vasoconstrictive mechanisms to redirect blood from periphery to vital organ
  - Exception is warm skin in early distributive shock
- Oliguria (↓kidney perfusion)
- Altered mental status (↓brain perfusion)
- Metabolic acidosis

Key diagnostic factor

- • 1. Hypotension (defined as decrease of ≥40 mmHg from baseline more significant
- when the systolic blood pressure less than 90.
- • •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 changes (cold sweaty skin, clammy peripheries, mottled, ashen appearance
- skin cyanosis (besides lips and tongue cyanosis))
- • 4. Oliguria :(consider inserting a urinary catheter, oliguria is defined as <0.5
- ml/kg/hr.
- • 5. Mental state changes (use GCS/Agitation, confusion, and distress occur early.
- Unresponsiveness indicates severe and advanced shock.)

- • 6.Positive Risk factors (history of sepsis, recent MI, history of hemorrhage,
- trauma, surgery, exposure to known allergen, change in medications, significant
- co-morbidities)
- • 7. Fever (suggests septic shock)
- • 8. Chest pain (suggests MI)
- • 9. 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.)
- • 10. Hypoxemia
- • 11. Hypothermia (it is the most obvious clinical sign of end-stage irreversible
- shock of any cause.)

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Glasgow Coma Scale

EYE OPENING

Spontaneous	4
To sound	3
To pressure	2
None	1

VERBAL RESPONSE

Oriented	5
Confused	4
Words	3
Sounds	2
None	1

MOTOR RESPONSE

Obeys commands	6
Localising	5
Withdrawal	4
Abnormal flexion	3
Extension	2
None	1

Glasgow coma scale scoring

Mild	Moderate	Severe
13-15	9-12	3-8

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

(>7mmol/L or >126mg/dL in non-diabetic patients)

4. Blood test:

✓ **CBC** :

(result: Hb < 100g/L suggests hemorrhages, 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 edema, pneumonia, pneumothorax, widened mediastinum (e.g., due to aortic dissection).

Use an ABCDE approach to manage shock empirically:

ABCDE	Assessment	Treatment
A – Airways	◦ Voice changes ◦ Breath sounds (stridor, snores, or increased breathing effort)	◦ Airway opening manoeuvre ◦ Airway suction ◦ Consider inserting an oropharyngeal or nasopharyngeal airway in deeply unconscious patients <8 GCS
B – Breathing	◦ Respiratory rate ◦ Chest wall expansion ◦ Chest percussion ◦ Lung auscultation ◦ Pulse oximetry	◦ Seat comfortably ◦ Inhaled medications ◦ Bag-mask ventilation ◦ Decompress tension pneumothorax (needle thrococentesis)
C – Circulation	◦ Skin color, sweating ◦ Capillary refill time (normally <2 s) ◦ Palpate pulse rate (60–100/ min) ◦ Heart auscultation ◦ Blood pressure (systolic 100–140 mmHg) ◦ ECG monitoring	◦ Stop bleeding ◦ Elevate legs ◦ intravenous access with crystalloid fluid administration
D – 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 > 10 g/dL) ◦ Serial lactate levels or central venous oxygen saturations to assess tissue oxygen extraction
E – Exposure and End points of resuscitation	◦ Exposure and temp assessment ◦ Assess goal values	◦ Make sure your approach is gaol-directed : Urine output > 0.5 mL/kg/hr CVP 8-12 mmHg MAP 65 to 90 mmHg Central venous oxygen concentration > 70%

➢ Hypovolemic Shock

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

** **Causes:**

Non-hemorrhagic	hemorrhagic
✓ Vomiting ✓ Diarrhea ✓ Bowel obstruction ✓ Burns ✓ Dehydration	✓ GI bleed ✓ Trauma ✓ Massive hemoptysis ✓ post-partum bleeding

SIGNS & SYMPTOMS:

- ↓CARDIAC OUTPUT
- ↓PAOP (PULMONARY ARTERY OCCLUSION/ WEDGE PRESSURE)
- ↑SVR (SYSTEMIC VASCULAR RESISTANT)
- HYPOTENSION
- TACHYCARDIA, TACHYPNEA
- COOL, CLAMMY SKIN <COLD SHOCK>

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**

• Evaluation of Hypovolemic Shock

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

Management of hypovolemic shock

✓ 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

➢ ANAPHYLACTIC SHOCK:

Anaphylactic shock

Anaphylactic shock is a severe, generalised or systemic hypersensitivity reaction, characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes

IgE mediated

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

Non IgE mediated

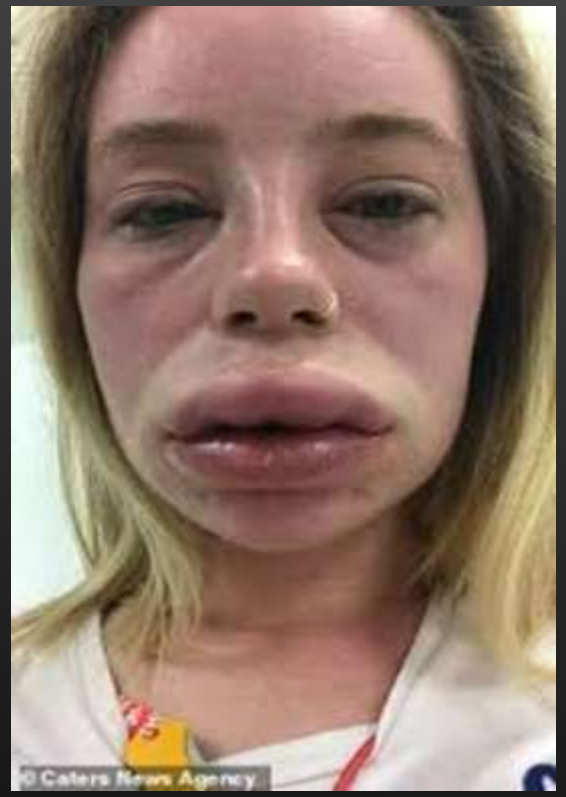
1. Activation of complement system
1. The complement peptides (anaphylatoxins) such as C3a and C5 directly act on mast cells and basophils leading to mediator release

Idiopathic

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

- **Possible causative agent of Anaphylactic shock:**
  1) **IgE mediated:** Food, Airborne allergens, Latex, Venom, insect sting, Medication, Semen
  2) **Immunologic non IgE mediated:** Immune aggregate, Intravenous immunoglobulin, Medication (NSAIDs), Radiocontrast media
  3) **Non immunologic:** Opiates, Physical factors (e.g., exercise, cold, heat)
  4) **Idiopathic**

- **Clinical presentation of Anaphylactic shock:**
  - ✓ **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.
  - ✓ Booth **poorly controlled asthma** and **previous anaphylaxis** are **risk factors for fatal anaphylaxis.**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- **Treatment algorithm of Anaphylactic shock:**

A) In Cardiorespiratory arrest

1. **Start CPR** 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)

Treatment is complex because:

- 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

➢ Obstructive Shock

- **General Characteristics**

  ✓ Caused by an **obstruction of circulation**. Most commonly due to **massive** pulmonary embolism (**PE**) or limited filling of the heart due to extrinsic pressure from pericardium (**cardiac tamponade**) or thoracic cavity (**tension pneumothorax**).

  ✓ Cardiac output limited by obstruction, SVR makes a compensatory increase to maintain systemic blood pressure.

  ✓ Usually manifests with elevated JVP due to limited filling of right ventricle or obstruction of pulmonary circulation by PE.

  ✓ 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, consider thrombolytic

➢ Cardiogenic Shock

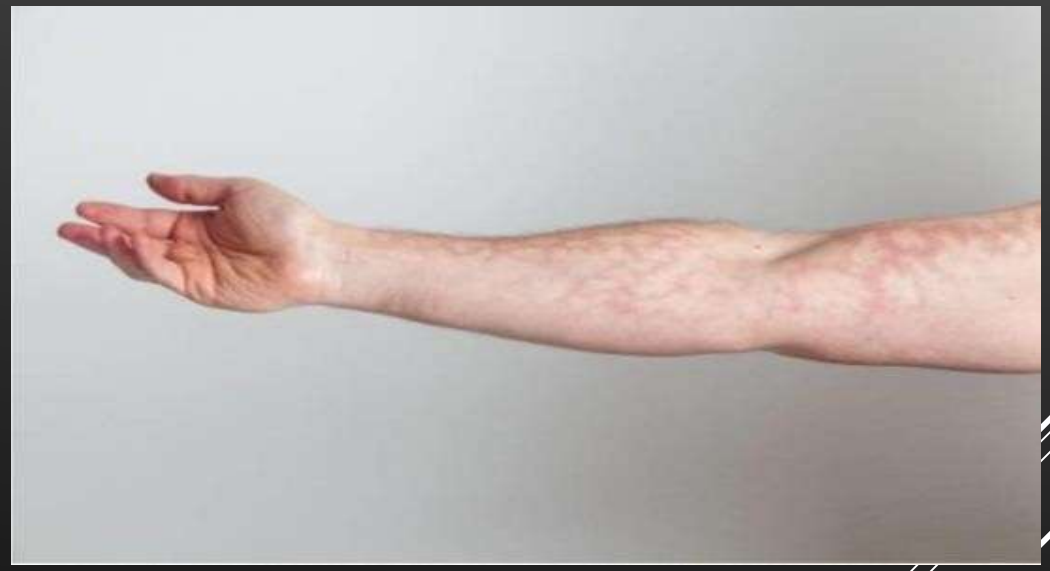
- Definition: is a life-threatening condition in which your heart suddenly can't pump enough
- • blood to meet your body’s needs
- • Caused by a defect in :
- • ❖1. Filling : diastolic heart failure
- • ❖2. Contraction : Acute MI
- • Congestive heart failure
- • cardiomyopathies
- • ❖3. Arrhythmias: both bradycardia & tachycardia
- • ❖4. Valvular diseases
- • ❖5. Congenital diseases like VSD

CARDIOGENIC SHOCK CHARACTERISTICS

- • Cardiogenic shock characterized by :
- • ❖Decrease cardiac output
- • ❖Increase PAOP “pulmonary artery occlusion/wedge pressure”
- • ❖Increase SVR “as compensation to maintain drop in B.P”
- • ❖Decrease left ventricular stroke work “ LVSW “

CARDIOGENIC SHOCK SIGNS

- • Angina
- • • Hypotension
- • • Tachypnea
- • • Cold & clammy skin (mottled skin)
- • • Pulmonary edema
- • • Peripheral edema
- • • Altered mental status
- • • Murmurs

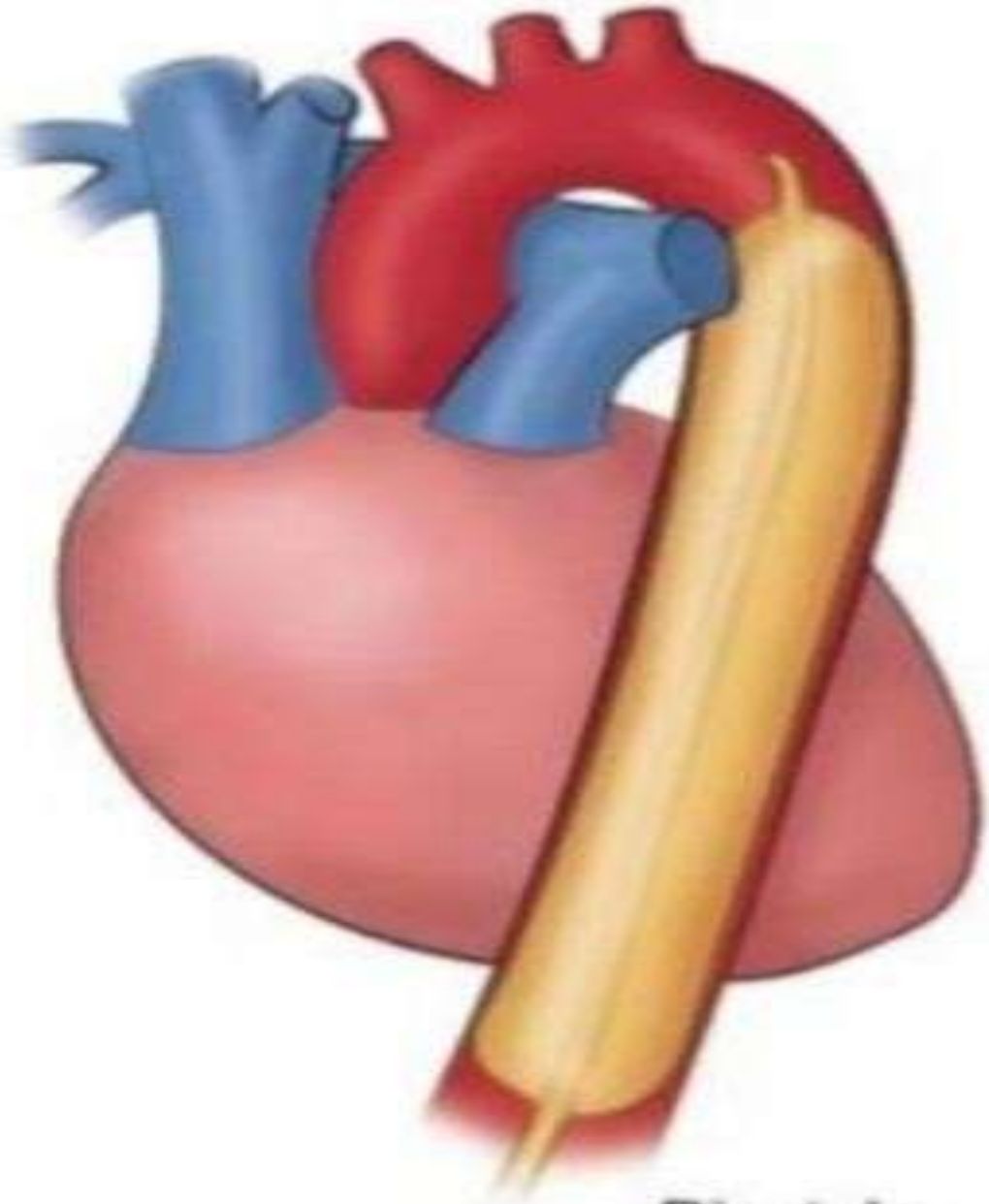


PATHOPHYSIOLOGY OF CARDIOGENIC SHOCK :

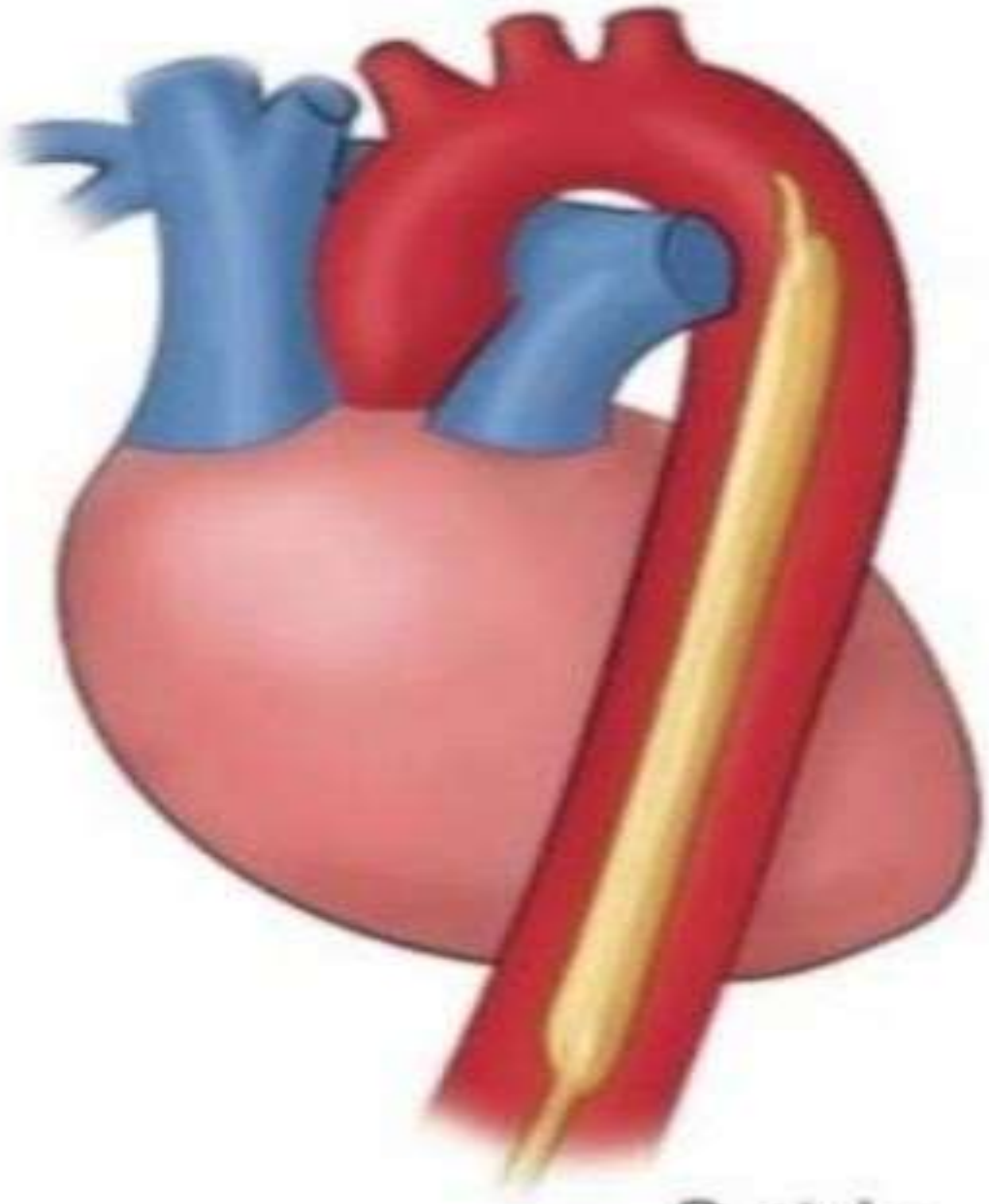
- • Often after ischemia there will be loss of left ventricle function “
- • considered clinical shock when there is loss of 40% of left ventricle “
- • ❖decrease Cardiac output → hypoxia , lactic acidosis
- • ❖Decrease stroke volume & as a compensation

TREATMENT

- ❖Goals : airway stability & improving myocardial pump function
- ❖Cardiac monitor, pulse oximetry
- ❖Supplemental oxygen, IV fluid
- ❖Catheterization if ongoing ischemia
- ❖Preload augmentation: consider fluids
- ❖Contractility: dopamine , dobutamine
- ❖Afterload reduction: nitroglycerin, dobutamine
- ❖If inotropes & vasopressors fail , do an intra-aortic balloon pump



Diastole



Systole

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, k2SVR low, PCWP low to normal

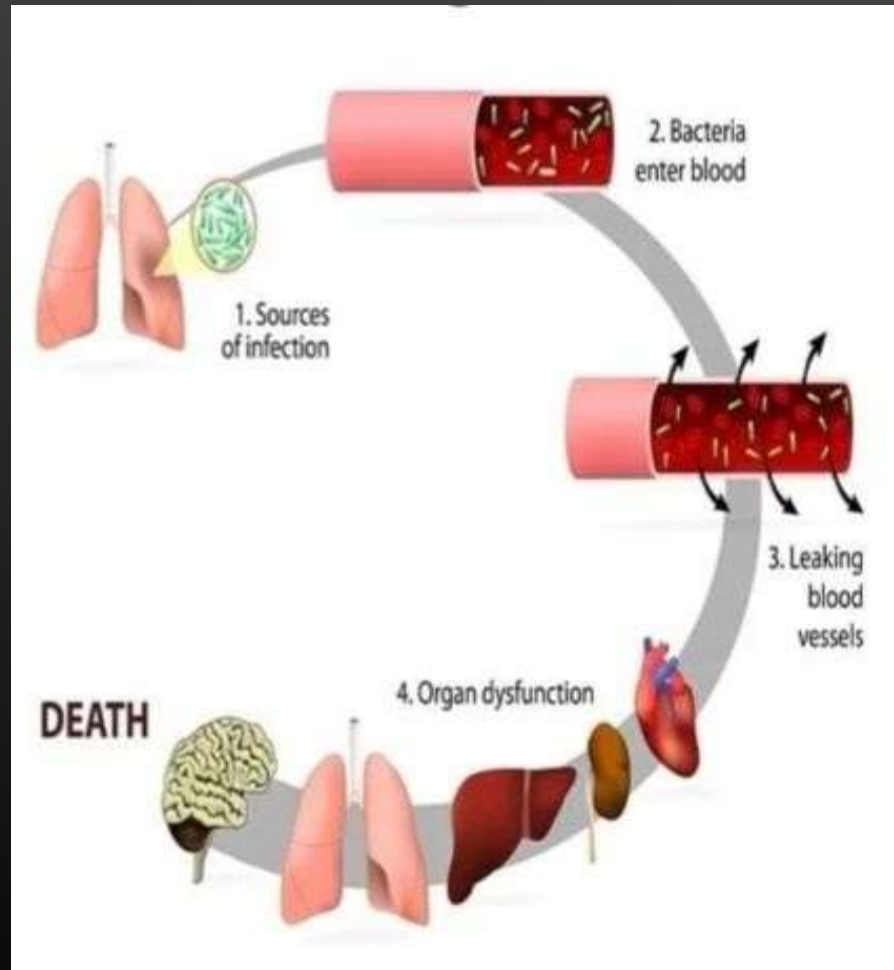
- **Treatment**
- 1. Judicious use of IV fluids as the mainstay of treatment
- 2. Vasoconstrictors to restore venous tone, but cautiously

➢ 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

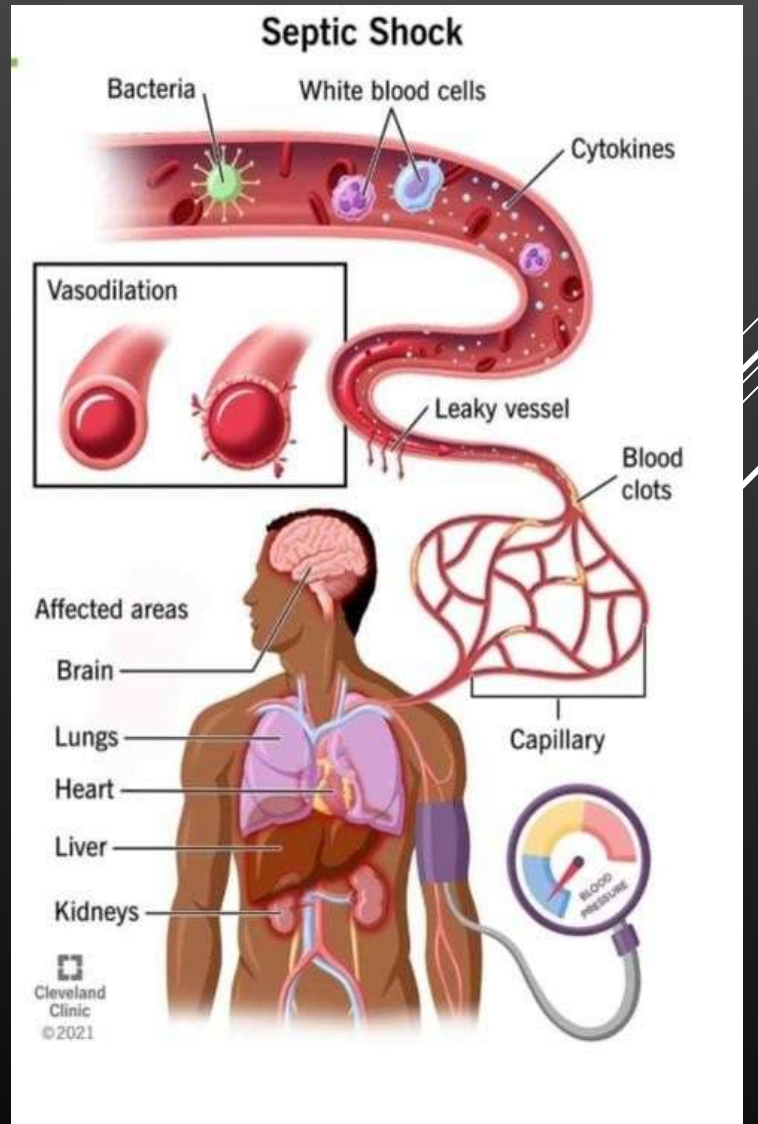
Common causes :
include pneumonia, urinary tract infection, meningitis, abscess formation, cholangitis, cellulitis, and peritonitis.



Pathophysiology

- when infectious microorganisms in the bloodstream induce a profound inflammatory response causing hemodynamic decompensation. The pathogenesis involves a complex response of cellular activation that triggers the release of a multitude of pro inflammatory mediators. This inflammatory response causes activation of leukocytes and endothelial cell, as well as activation of the coagulation system. The excessive inflammatory response

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



PREDISPOSIG FACTORS:

- very young
- elderly
- immunocompromised (HIV,systimc fibrosis,DM)

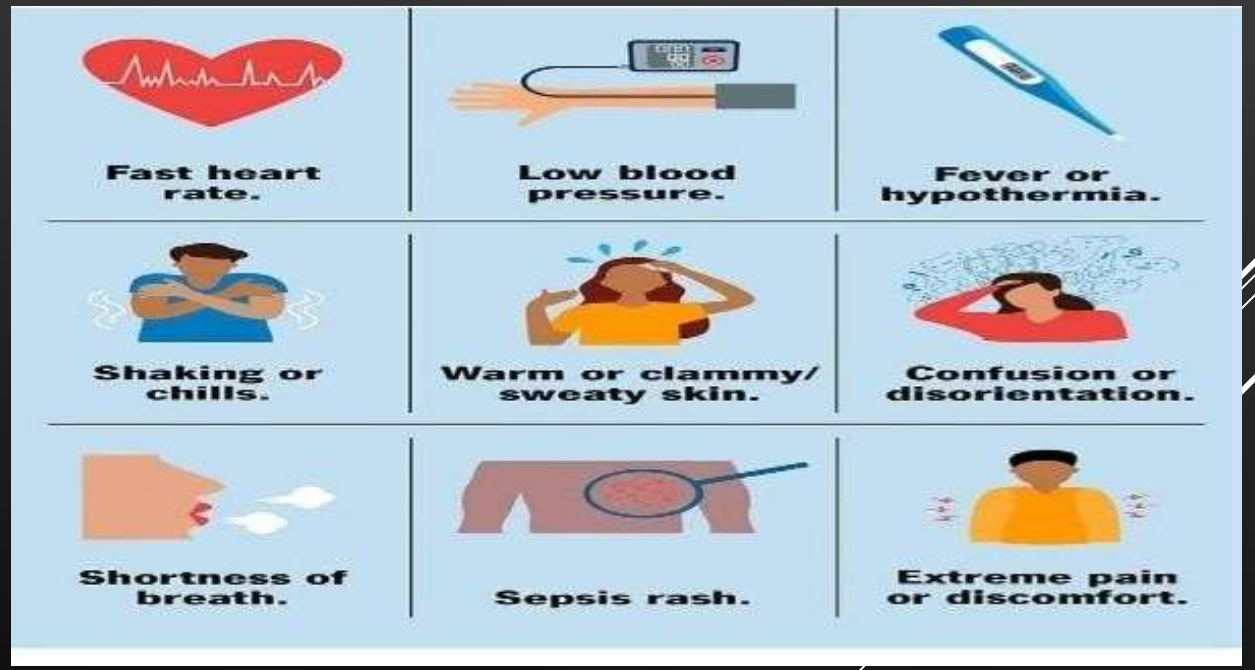
Clinical signs:

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

Tachycardia

Low blood pressure

Mental status changes



Diagnosis:

- 1. Septic shock is essentially a clinical diagnosis.

2. A source of infection can aid in diagnosis, but there may be no confirmed source in some cases.

Treatment

- 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

Thank You