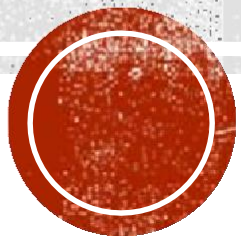
SHOCK



- Outline
  - Definitions
  - Types of Shock
  - Stages of shock
  - General clinical features
  - Initial approach
  - Management principles

DEFINITION

- The state of widespread reduction of effective tissue perfusion and delivery of oxygen and nutrients to vital organs leads first to reversible and then if prolonged to irreversible cellular injury. (circulatory failure)

↓ Tissue perfusion (o2 delivery < o2 demand (

- The clinical manifestations of shock are the result of stimulation of the sympathetic and neuroendocrine stress response, inadequate oxygen delivery , end organ dysfunction

- Blood pressure is often used as an indirect estimator of tissue perfusion .
- BP = C O * TPR
- Low cardiac output

  loss of contractility

  Low intravascular volume
- Peripheral vasodilation

TYPES OF SHOCK

- Cardiogenic shock

  cardiac disorder ⟶ fall in cardiac output

- Hypovolemic shock

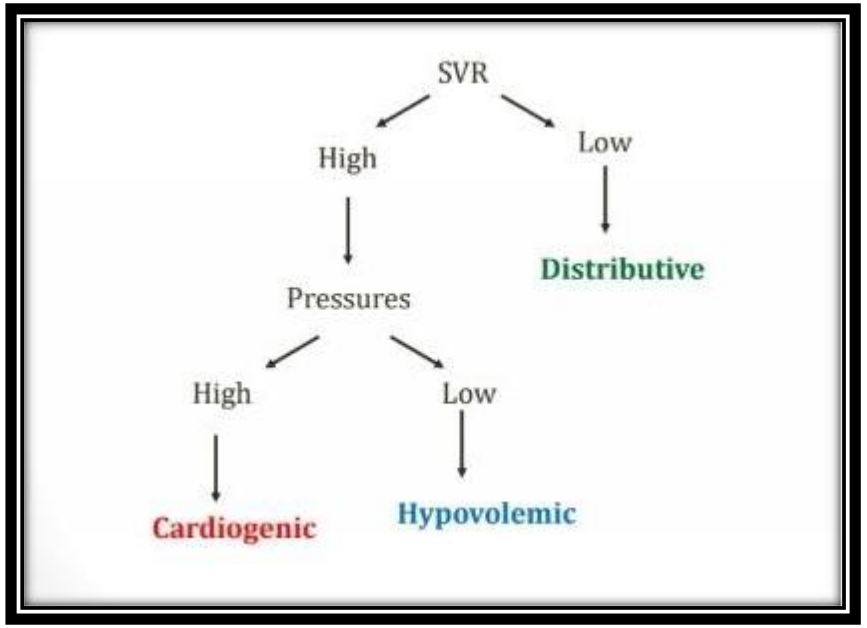
  fall in intravascular volume ⟶ fall in cardiac output

- Distributive shock

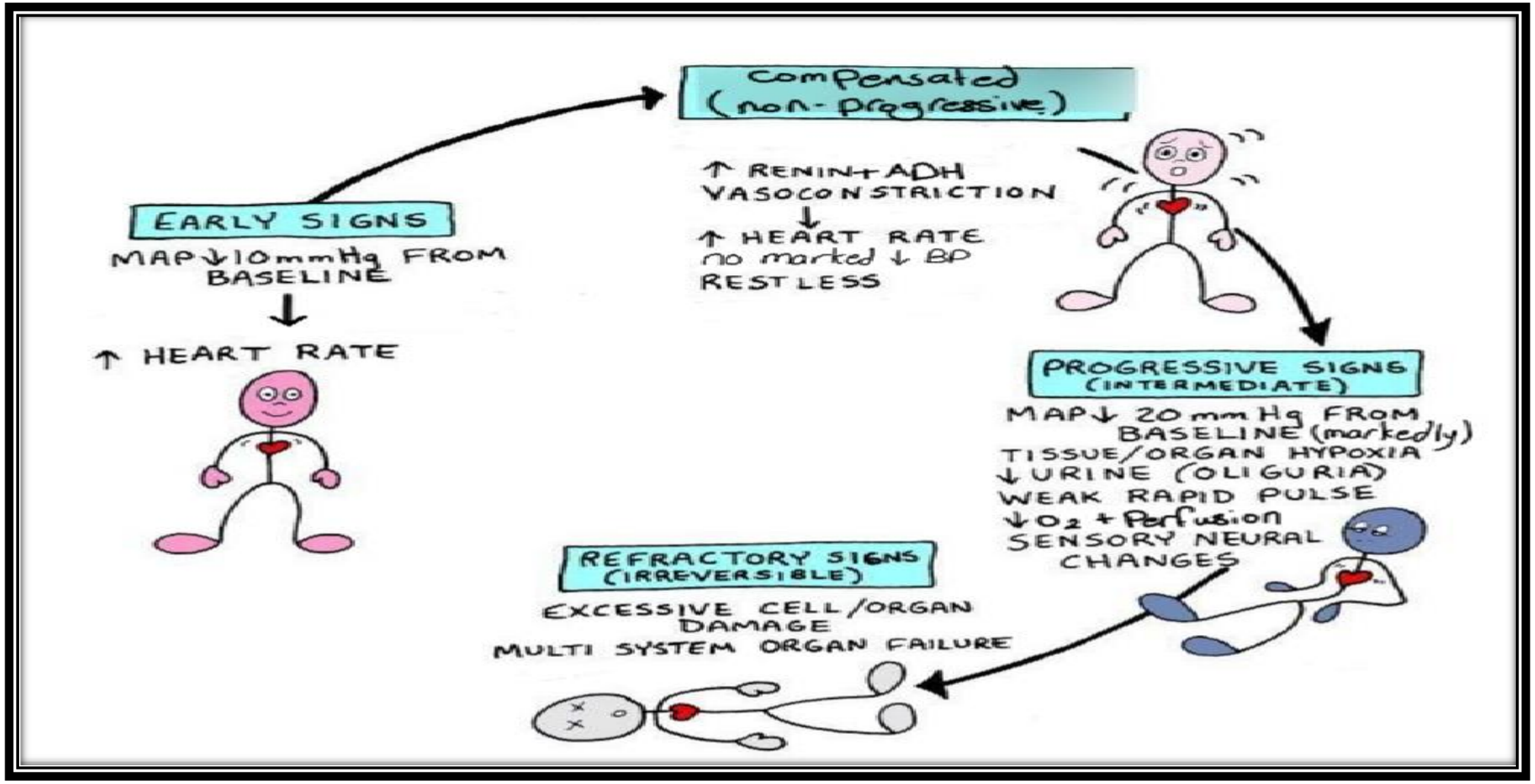
  septic , anaphylaxis, neurogenic

  (peripheral vasodilation)

- Obstructive shock



STAGES OF SHOCK



GENERAL CLINICAL FEATURES

- Metabolic acidosis (due to anaerobic metabolism)*
- Hypotension, tachycardia*
- Increase respiratory rate (shallow), pulmonary edema*
- C old clammy skin , pallor
- Drowsiness, confusion, irritability
- Oligourea,*
- multi-organ failure

INITIAL APPROACH IN SHOCK

- Focus history and physical examination to determine possible cause of shock

  hemorrhage …… Hyopvolumic shock

  Myocardial infarction …….. Cardiogenic Shock

  spinal cord injury or neurological deficit ……. Neurogenic shock

- Lab tests directed tword possible cause

ON PHYSICAL EXAM

- Cold skin (high SVR and low C O)

  hypovolemic and cardoigenic

- Warm skin (low SVR and high C O)

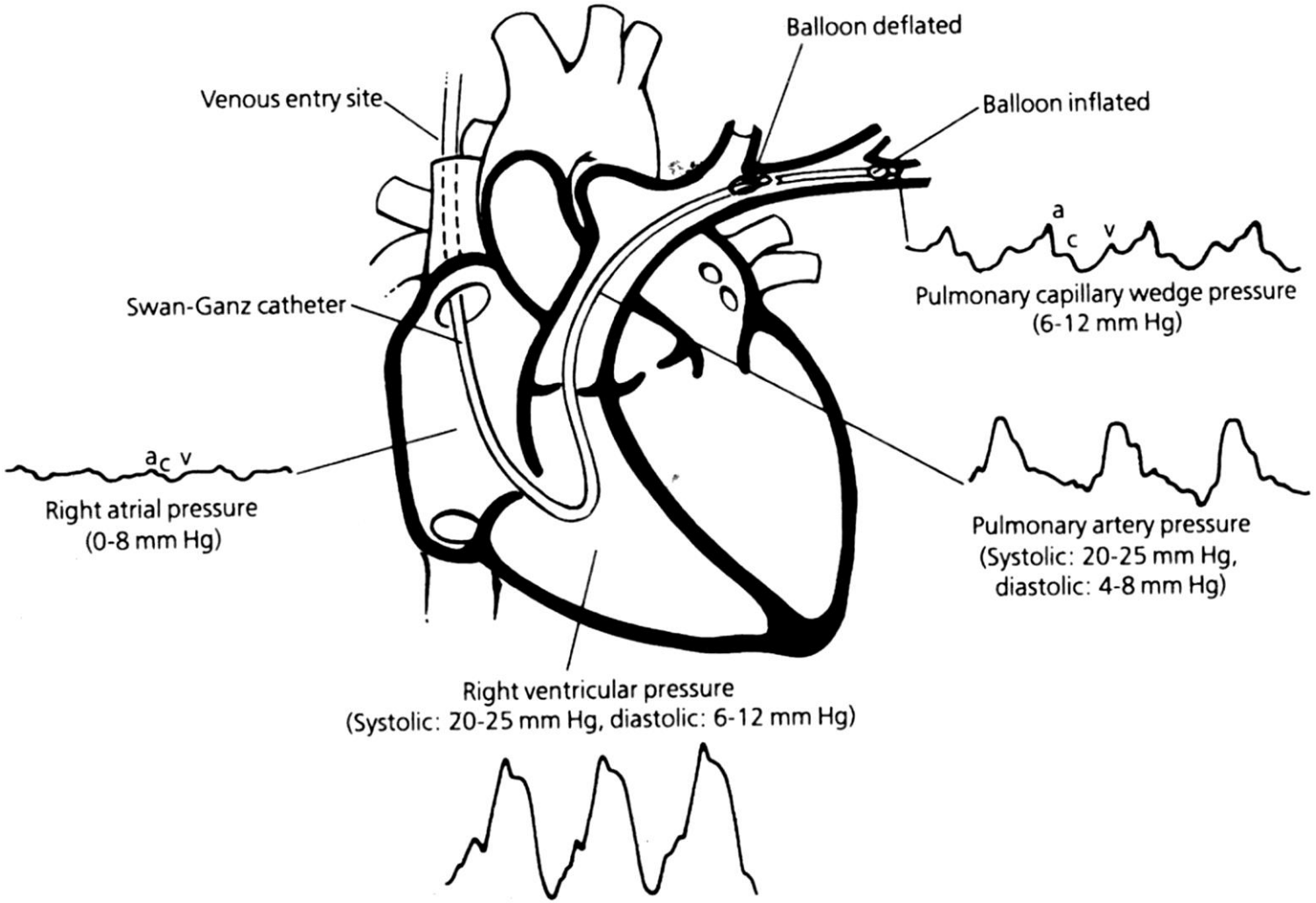
  Distributive

- High JVP and Pulmonary rales

  cardiogenic

If still unknown cause …..
Swan Ganz catheter

SWAN GANZ CATHETER



SWAN GANZ DATA

- Direct
  - RA Pressure (Normal ~ 5mmHg)
  - RV Pressure (20/5)
  - PA Pressure (20/10)
  - PCWP Pressure (10)
  - Mixed venous O2 sat
- Calculated
  - Cardiac output
  - Systemic Vascular Resistance

- We can calculate SVR from flow equation

$$\text{Cardiac Output} = \frac{\text{O2 Consumption}}{(\text{Art O2} - \text{Ven O2})}$$

- We can determine cardiac output from fick equation

$$\text{SVR} = \frac{\text{MAP} - \text{RAP}}{\text{CO}}$$

Used to determine whether any hemodynamic or blood flow related abnormalities exist in the heart and lung

HEMODHNAMIC CHANGES OF SHOCK

Type of shock	Cardiac output	BP	SVR	PCWP	RA .P	RV.P
Cardiogenic	↓	↓	↑	↑	↑	↑
Hypovolemic	↓	↓	↑	↓	↓	↓
Distributive	↑	↓	↓	↓	↓	↓

MANAGEMENT PRINCIPLES

- Initial stabilisation of patient :
- ABC (airway , breathing ,circulation)
- Specific treatment for each type

Cardiogenic shock

Yasmin Mohammad Almughrabi

Definition:

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

(decreased CO > tissue hypoxia)

In this case, unlike the hypovolemic shock, we don’t have loss in volume.

Cardiogenic Shock

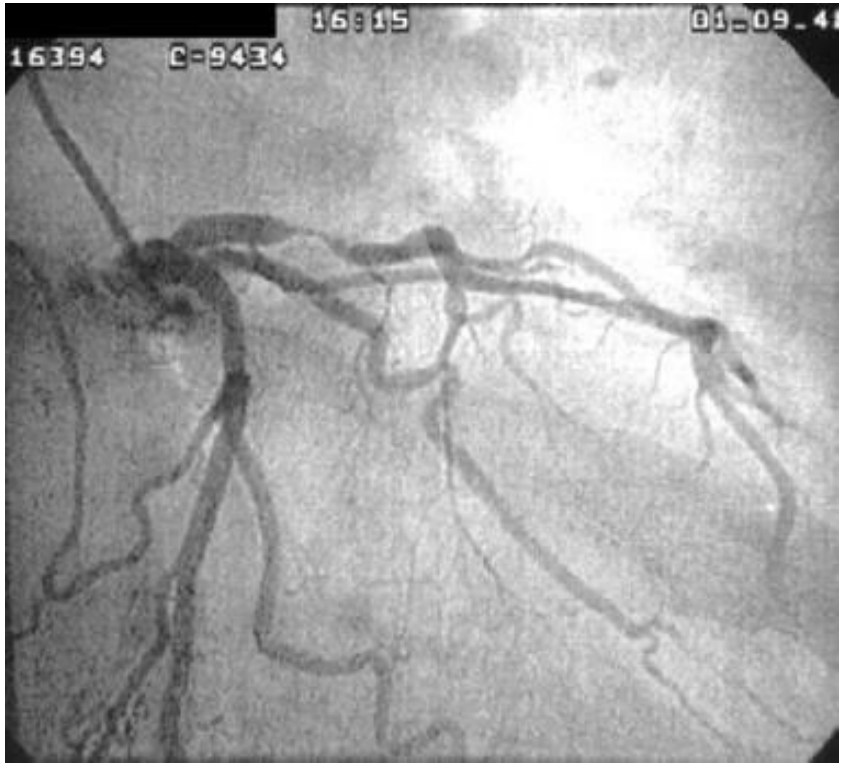
as defined in the SHOCK trial

1. Persistent hypotension (SBP < 90 mmHg or MAP 30 mmHg below baseline)
2. Cardiac Index < 1.8 L/min/m^2 without support (< 2.2 L/min/m^2 with support)
3. Adequate or elevated filling pressure (LVEDP > 18 mmHg, RVEDP > 10 mmHg)

Etiology:

Various forms of cardiac dysfunction can cause cardiogenic shock:

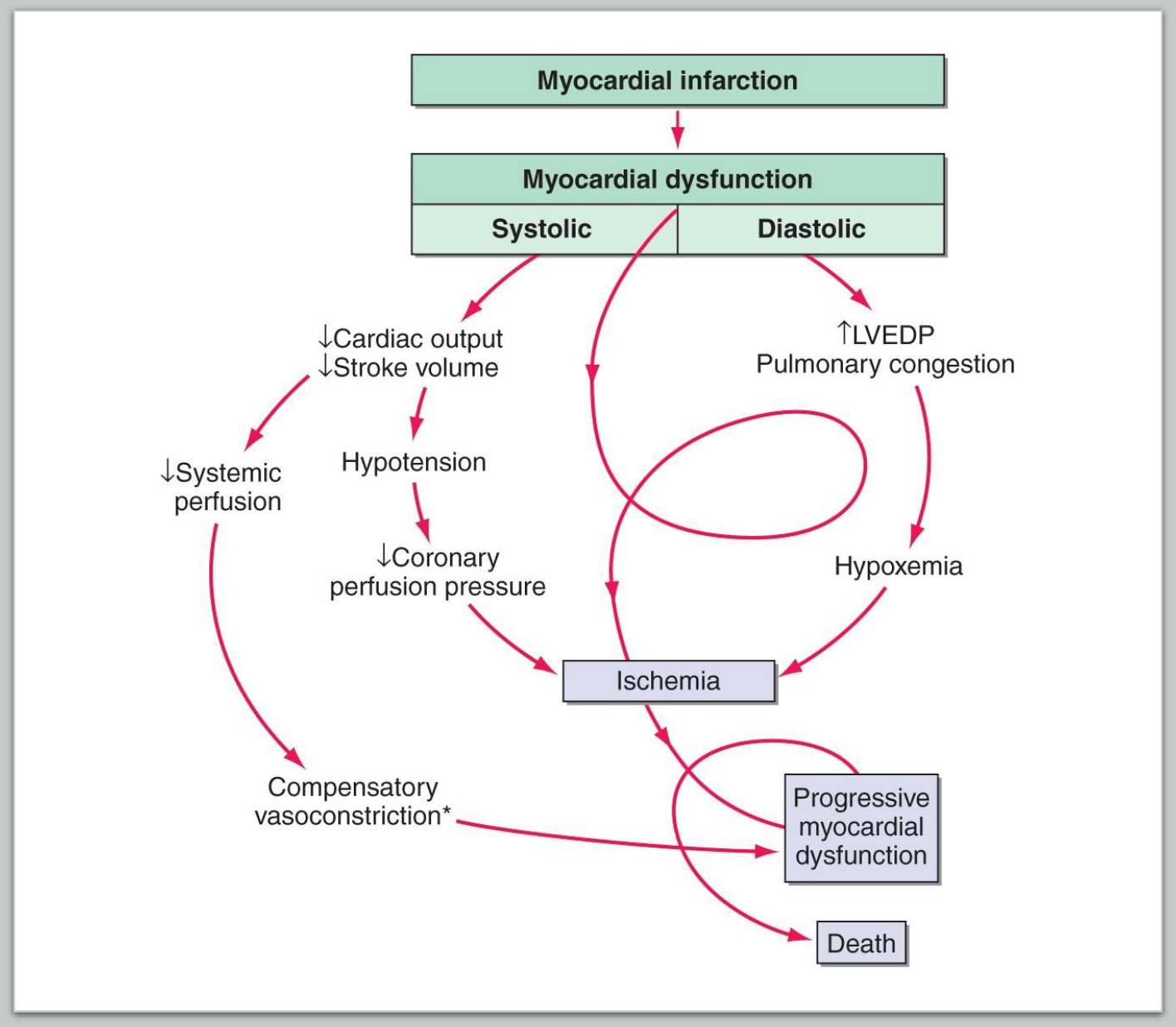
- MI (most common)
- Mechanical defects: acute mitral regurgitation (papillary muscle rupture), ventricular wall rupture, cardiac tamponade, left ventricular outflow obstruction(AS, HOCM), left ventricular inflow obstruction(AS, atrial myxoma)



- Contractility defect: myocarditis, arrhythmias, ischemic and non-ischemic cardiomyopathy)
- Right ventricular failure
- Pulmonary embolus (right ventricular with or without left ventricular failure)
- Other causes include cardiotoxic drugs (doxorubicin), myocardial depressant drugs(BBs,CCBs), metabolic derangements(acidosis, hypocalcemia)

Pathophysiology:

•cardiogenic shock is generally associated with the loss of more than 40% of the LV myocardial muscle.

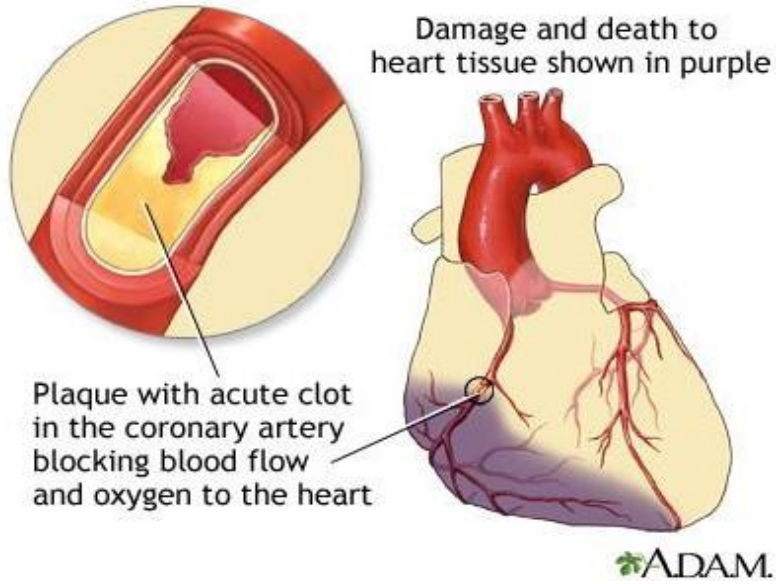


Myocardial pathology

- Cardiogenic shock is characterized by systolic and diastolic dysfunction leading to end organ hypoperfusion. The interruption of blood flow in an epicardial coronary artery causes the zone of myocardium supplied by that vessel to lose the ability to shorten and perform contractile work. If a sufficient area of myocardium undergoes ischemic injury, LV pump function become depressed and systemic hypotension develops.
- Patients who develop cardiogenic shock from acute MI consistently have evidence of progressive myocardial necrosis with infarct extension. Decreased coronary perfusion pressure and cardiac output as well as increased myocardial oxygen demand play a role in the vicious cycle that leads to cardiogenic shock and potentially death.
- Patients suffering from cardiogenic shock often have multivessel coronary artery disease with limited coronary blood flow reserve. Ischemia remote from the infarcted zone is an important contributor to shock. Myocardial diastolic function is also impaired, because ischemia decreases myocardial compliance and impairs filling, thereby increasing LV filling pressure and leading to pulmonary edema and hypoxemia.

Risk factors:

- Old age
- History of HF or heart attack
- Have blockages (CAD) in several of the heart's main arteries
- Have DM or high high BP
- Existing cardiac disease



Diagnosis:

1. History and physical examination :-

History plays a very important role in understanding the etiology of the shock and thus helps in the management of cardiogenic shock :

The presenting symptoms of CS are variable include:

- Hypotension (systolic BP<90mmHg)
- Angina/ chest pain
- Altered mental status
- Low urine output (oliguria)
- Cool clammy skin (due to peripheral VC)
- Tachycardia

Physical examination findings:

- Altered mental status
- Peripheral pulses are faint, rapid and sometimes irregular if there is underlying arrhythmia
- Jugular venous dissension
- Pulmonary vascular congestion may be associated with rales
- Murmurs in the presence of valvular disorder such as mitral regurgitation or aortic stenosis
- Peripheral edema

2. **Laboratory studies:**

- CBC
- Lactate level
- Arterial blood gases
- BNP
- Cardiac enzyme test(troponins and CK-MB)

3. **Imaging studies:**

- CXR:for pulmonary congestion
- Echocardiogram
- Coronary angiography

4. **ECG:**for acute MI or any arrhythmia

Management:

- Cardiogenic shock is an emergency requiring immediate resuscitative therapy before the irreversible damage of vital organs. Rapid diagnosis with prompt initiation of pharmacological therapy to maintain blood pressure and to maintain respiratory support along with a reversal of underlying cause plays a vital role in the prognosis of patients with cardiogenic shock.

Emergency life support

- Most people who have cardiogenic shock need extra oxygen. If necessary, you'll be connected to a breathing machine (ventilator). You'll receive medications and fluid through an IV line in your arm.
- Fluid resuscitation to correct hypotension, unless pulmonary edema is present

Revascularization

- Early and definitive restoration of coronary blood flow; at present, this represents standard therapy for patients with cardiogenic shock due to myocardial ischemia

Pharmacological therapy

- Inotropic agents

-eg, dobutamine / milrinone

-to improve the pumping function of the heart and lower LVEDP

- Vassopressors

-eg, NE / epinephrine/ dopamine

-to treat low BP

-should be used with caution as it can cause tachycardia and increase myocardial oxygen demand in patients with recent MI

- Diuretic: eg, furosemide

-if signs of volume overload are present (pulmonary edema)

- Aspirin and heparin

-pts with MI and ACS

Surgical and other procedures

a. Balloon pump

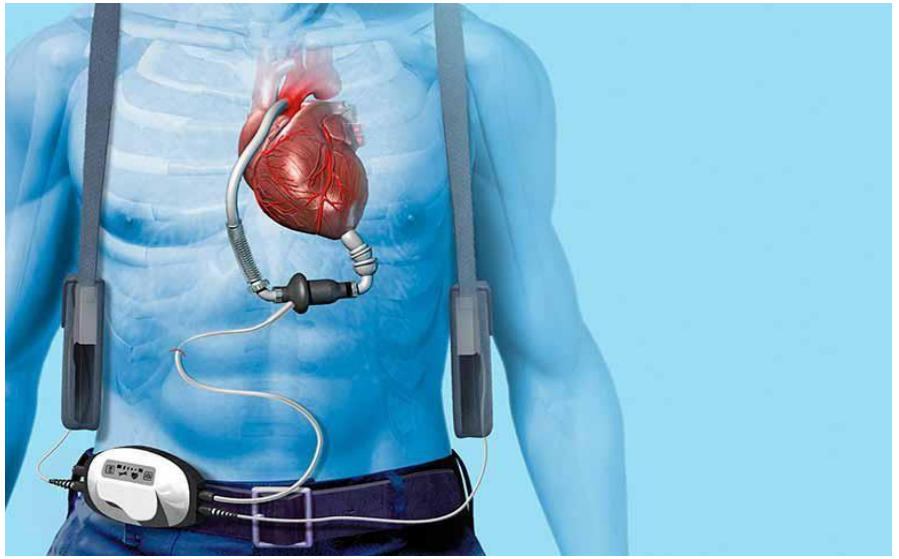
Balloon pump is inserted in the main coronary artery of the heart. The pump inflates and deflates within the aorta, helping blood flow and taking some of the workload off the heart.

b. Coronary artery bypass surgery

c. Ventricular assist device

Mechanical device implanted into the abdomen and attached to the heart to help it pump.

b. Heart transplant

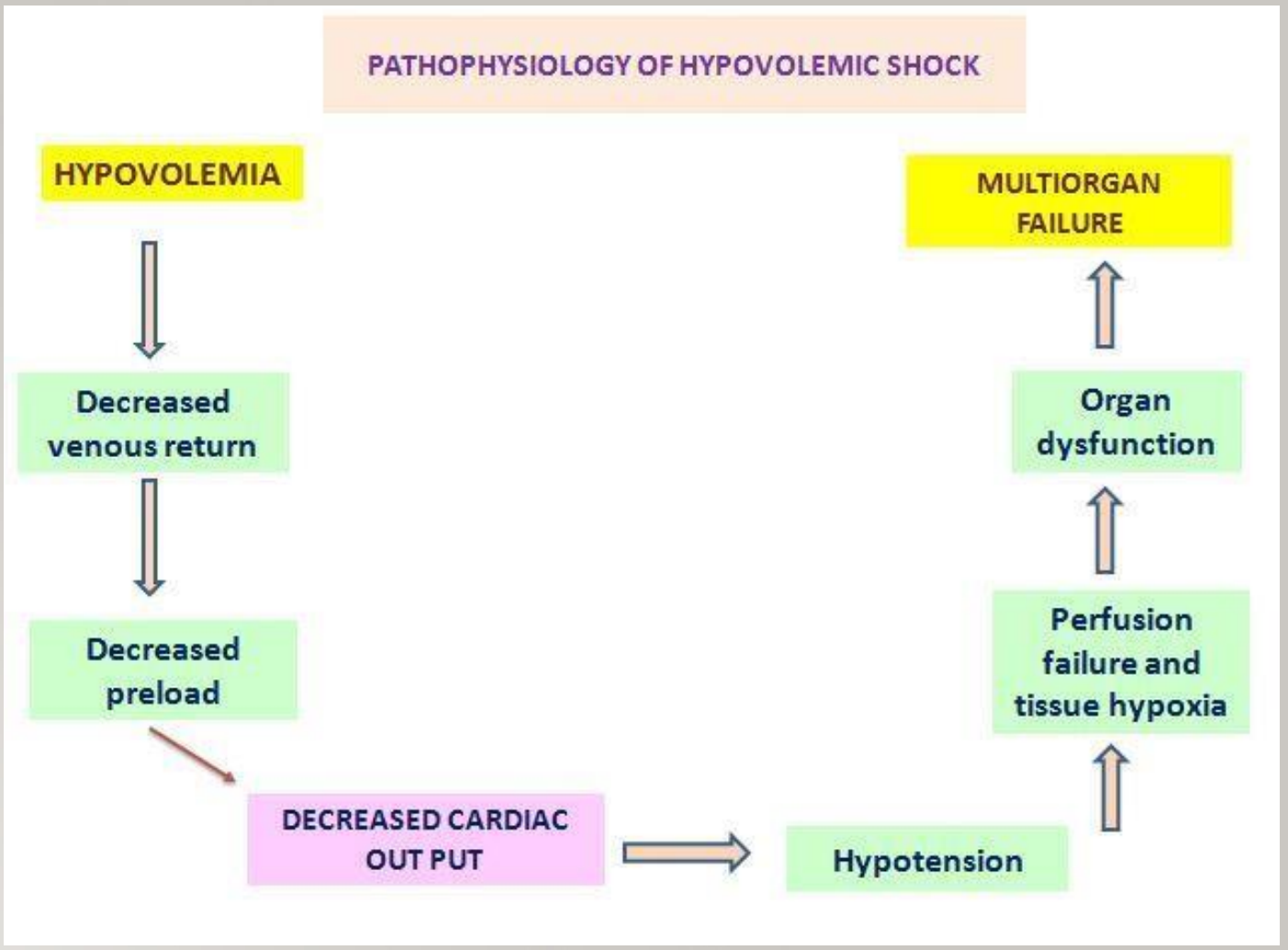


Thank you

HYPOVOLEMIC SHOCK

Hypovolemic shock is an emergency condition in which severe blood and fluid loss make the heart unable to pump enough blood to the body due to decreased preload.

It leads to multiple organs failure.


PATHOPHYSIOLOGY OF HYPOVOLEMIC SHOCK
HYPOVOLEMIA
Decreased venous return
Decreased preload
DECREASED CARDIAC OUT PUT
Hypotension
Perfusion failure and tissue hypoxia
Organ dysfunction
MULTIORGAN FAILURE

CAUSES :

1) Hemorrhagic

- Trauma
- GI bleeding
- Retroperitoneal

2) Nonhemorrhagic

- vomiting
- Severe diarrhea
- Severe dehydration for any reason
- Burns

PRESENTATION OF HYPOVOLEMIC SHOCK

- Hypotensive
- flat neck veins
- clear lungs
- cool, cyanotic extremities
- evidence of bleeding
  - Anticoagulant use
  - trauma, bruising
- oliguria

CLASSES OF HYPOVOLEMIC SHOCK

TABLE 1-6 Hemodynamic Changes in Hypovolemic Shock

Class	Blood Volume Lost (%)	Pulse (↑)	Systolic BP (↓)	Pulse Pressure (↓)	Capillary Refill (↓)	Respiratory Rate (↑)	CNS	Urine Output (↓)
I	10–15	Normal	Normal	Normal	Normal	Normal	Normal	Normal
II	20–30	>100	Normal	Decreased	Delayed	Mild tachypnea	Anxious	20–30 mL/hr
III	30–40	>120 weak	Decreased	Decreased	Delayed	Marked tachypnea	Confused	20 mL/hr
IV	>40	>140 non-palpable	Marked decrease	Marked decrease	Absent	Marked tachypnea	Lethar-gic, coma	Negligible

MANAGEMENT

- 1.Airway and breathing—patients in severe shock and circulatory collapse generally require intubation and mechanical ventilation.

  2. Circulation

  a.If hemorrhage is the cause,apply direct pressure.

  b.IV hydration

  •Patients with class I shock usually do not require fluid resuscitation. Patients with class II shock benefit from fluids, and patients with classes III and IV require fluid resuscitation.

  •Give fluid bolus followed by continuous infusion and reassess.

  •The hemodynamic response to this treatment guides further resuscitative effort.

  c. For nonhemorrhagic shock, blood is not necessary. Crystalloid solution with appropriate electrolyte replacement is adequate.

OBSTRUCTIVE SHOCK

Obstíuctive shock is one of the fouí types of shock, caused by a physical obstíuction in the flow of blood.. Obstíuction can occuí at the level of the gíeat vessels oí the heaít itself.

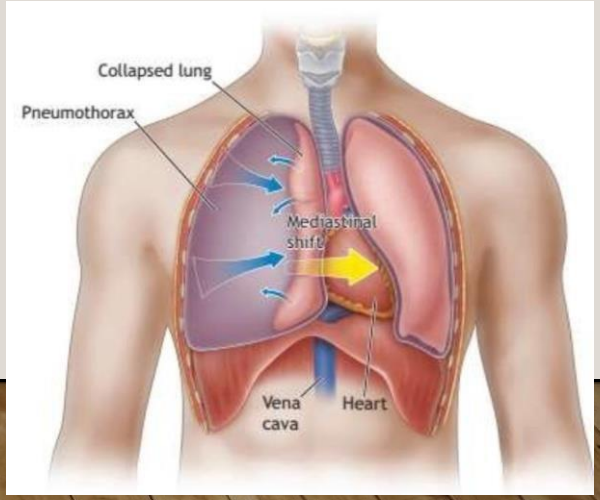
- If blood flowing INTO the heart is obstructed, it causes a decrease in cardiac output because of impaired diastolic filling.
- If blood flowing OUT of the heart is obstructed, it causes a decrease in cardiac output because of excessive afterload

CAUSES

- Causes include any obstruction of blood flow to and from the heart , including:

1.TENSION PNEUMOTHORAX

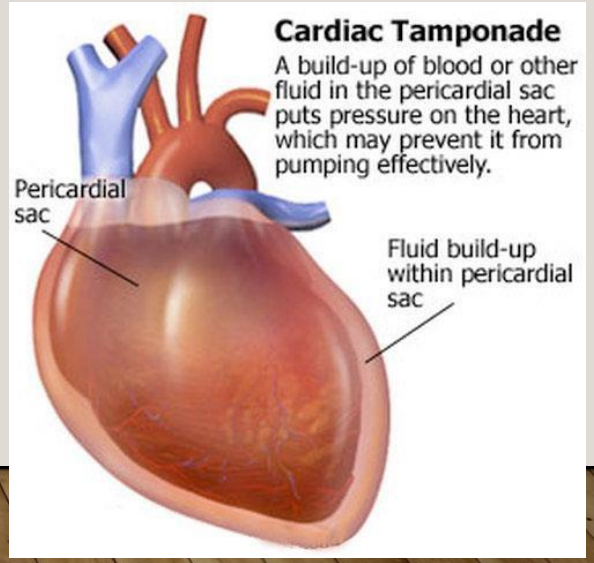
The accumulation of air within the pleural space increases pressure and ultimately obstructs venous blood return to the heart. **The obstruction results in decreased diastolic filling.**



CAUSES

2.CARDIAC TAMPONADE

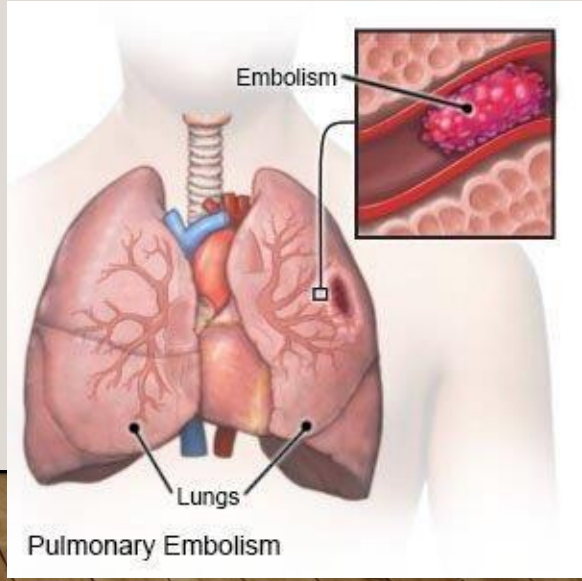
- As fluid accumulates, the increased pressure decreases venous return to the heart and causes right ventricular compression which results in a progressive decline in right ventricular end-diastolic volume. The decreased end- diastolic volume compromises cardiac output which results in shock symptoms.



CAUSES

3.PULMONARY EMBOLISM

- This obstruction increases the pulmonary vascular resistance. If large enough, the clot increases the load on the right side of the heart. The right ventricle must work harder to pump blood to the lungs. With back-up of blood, the right ventricle can begin to dilate. Right heart failure can ensue, leading to shock and death.



SIGNS AND SYMPTOMS

Signs and symptoms seen depending on the underlying cause:

- Tension pneumothorax

decreased breath sounds and chest mobility, Tracheal deviation

- cardiac tamponade

hypotension, jugular vein distension, pulsus paradoxus

- Pulmonary embolism

shortness of breath and hypoxia, Chest pain, syncope, hemoptysis

MANAGEMENT

1. Control airway

Intubation

2. Treat the underlying cause

Tension pneumothorax : chest tube

Pericardial tamponade : pericardiocentesis

Pulmonary embolism : anticoagulation

3. Isotonic fluids

Septic shock

- جاسم الظفيري

Septic shock: is a clinical state that accompanies infection either confined to a local site from which toxin are absorbed or associated with invasion of organism in to the blood stream.

Causes of Sepsis:

- **Bacterial** :

- Staph. aureus, coagulase-negative staphylococci in all ages
- Strep. Pneumoniae in all ages.
- Neisseria Meningitidis, in children and young adults
- E.coli, with UTI (urosepsis) and biliary tract infections
- Pseudomonas aeruginosa and MRSA are Nosocomial infections
- Group B strep – in neonates

- **Fungal**: Candida spp. and Histoplasma capsulatom can be a complication of Post-operation or in immunocompromised hosts

- **Parasitic**: Falciparum malaria, Babesia microti.

- **Viral**: Influenza (flu) and others

Risk Factors:

1. Age - elderly and very young are at high risk.
2. Instrumentation or surgery (including illegal abortion occurring in unhygienic circumstances).
3. Alcohol abuse.
4. Burns or injuries.
5. Immunosuppression (eg.DM).
6. Medications, eg. high dose corticosteroids, chemotherapy.
7. Black race.
8. Males are more prone , although the mortality in females is higher.

Pathophysiology:

- Immune-system activation
- The endothelium and coagulation system
- Organ dysfunction

Immune-system activation:

- Innate immune system activated by bacterial products (lipopolysaccharide)→host receptors including (TLRs) on leukocytes, macrophages and endothelial cells.
- After activation, the WBCs will release:

  *1- Pro-inflammatory **cytokines** ((TNF)-alpha, IL1, 6) release →activate immune cells/recruitment.*

  *2- Reactive oxygen species, **nitric oxide** (NO), proteases, and pore-forming molecules → bacterial killing.*

  NO is responsible for systemic vasodilatation & increased capillary permeability → hypoperfusion & hypotention

  3- Complement system activation → activation and migration of leukocytes →attack the organism.

The endothelium and coagulation system:

- Activated endothelium allows the adhesion and migration of immune cells, and becomes permeable to proteins, resulting in **oedema**.
- Alterations in the coagulation systems by:

*Increase in procoagulant factors (plasminogen activator inhibitor type I , tissue factor).
*reduced circulating levels of natural anticoagulants (antithrombin III and activated protein C (APC)), which also carry anti-inflammatory and modulatory roles.

altered coagulation, frequently leading to **disseminated intravascular coagulation (DIC)**

Inflammation and organ dysfunction:

- **vasodilatation** and increased capillary **permeability** leads to reductions in circulating volume and **hypotension**.
- pericapillary **oedema** & mural oedema (reduce capillary diameter) and **microthrombus** formation leads to **Impaired O2 delivery**
- Disordered blood flow through capillary beds, resulting from shunting of blood through collateral channels and an increase in blood viscosity secondary to loss of red blood cell flexibility so organs may become **hypoxic**. These abnormalities may lead to **lactic acidosis**, **cellular dysfunction, and multi-organ failure**.
- The **mitochondrial function** is also affected by the systemic inflammatory processes, resulting in impaired oxidative phosphorylation and aerobic energy generation which is called cytopathic hypoxia. This will lead to further lactic acidosis and the development of multiple organ failure.

Clinical Features:

The signs and symptoms are arranged in two groups:
A. Signs and Symptoms that are related to infection :

1. Fever [More than 38.3º], chills and/or sweating.
2. Tachycardia
3. Tachypnea

B. Signs and Symptoms that are due to hypoperfusion ‘Shock’:

1. Confusion
2. Malaise
3. Hypotension
4. Oliguria
5. Poor capillary refill [In severe cases]
6. Palpable skin purpura if associated with DIC.

+ Non-Specific Symptoms:

1. Nausea
2. Vomiting
3. Diarrhea
4. Abdominal Pain

Progression of Shock

A. Warm / Early shock:

The initial release of inflammatory cytokines causes a drop in systemic vascular resistance (SVR) and vasodilatation which leads to low diastolic blood pressures.

The patient is warm, pink, well-perfused with tachycardia and tachypnea

B. Cold shock:

The blood vessels are constricted due to the activation of the sympathetic nervous system in an attempt to maintain blood pressure by increasing the systemic vascular resistance.

The patient presents with cold peripheries, slow capillary refill and hypotension.

Table 3. Features of warm and cold shock

	WARM shock	COLD shock
Peripheries	warm, flushed	cold, clammy, cyanotic
Capillary refill	< 2 sec	> 2 sec
Pulse	bounding	weak, feeble
Heart rate	tachycardia	tachycardia or bradycardia
Blood pressure	relatively maintained	hypotension
Pulse pressure	widened	narrowed

Complications:

1. Disseminated Intravascular Coagulation (DIC); Occurs in 40% of patients with septic shock
2. Acute Respiratory Distress Syndrome (ARDS)
3. Acute Kidney Disease
4. Myocardial Ischemia
5. Liver Failure

Investigations:

1. CBC: .
2. Urine analysis,.
3. Renal function –
4. Liver function tests -
5. Clotting screen,

7. **Blood cultures** -
8. **Radiology** - including CXR, abdominal ultrasound looking for a collection, and CT scan looking for source.
9. **Measures of lactate and oxygen saturation of venous blood**
10. **Arterial blood gases.**
11. **More invasive investigations looking for a source of infection** - lumbar puncture, bronchoscopy, laparoscopy, lymph node biopsy, etc.

Management:

- Early recognition
- adequate antibiotic therapy (broad spectrum) as early as possible
- source control - Identify the source of infection, and treat with antimicrobial therapy, surgery, or both
- Early hemodynamic resuscitation and continued support using supportive measures to correct hypoxia, hypotension, and impaired tissue oxygenation (hypoperfusion)

- Proper ventilator management with low tidal volume in patients with acute respiratory distress syndrome (ARDS)
- Maintain adequate organ system function, guided by cardiovascular monitoring, and interrupt the progression to multiple organ dysfunction syndrome (MODS)

Thank You

NEUROGENIC SHOCK

- Neurogenic shock is a medical condition which occurs as a result of disturbance in the sympathetic outflow causing loss of vagal tone
- Experiences neurogenic shock after injury to the spinal cord and when there is disruption in the blood circulation throughout the body due to injury/ illness.
-

- It is a serious and life-threatening condition, which requires prompt medical attention without any delay. If the treatment is delayed, then it causes irreversible tissue damage and even death.
- Out of the different types of the shocks, neurogenic shock is the most difficult to manage, mainly because of the irreversible damage to the tissues.
- Neurogenic shock mainly affects the spinal cord; the function of which is transmitting neural signals from the brain to the entire body and back.

causes of neurogenic shock include:

car accidents

sport injuries

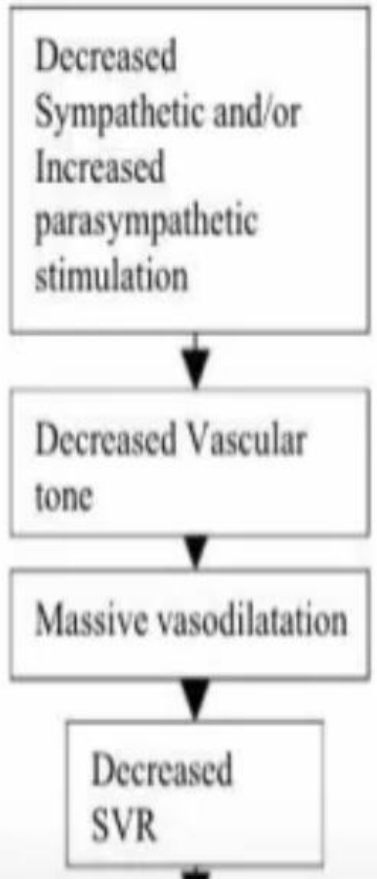
gunshot wounds to the spine

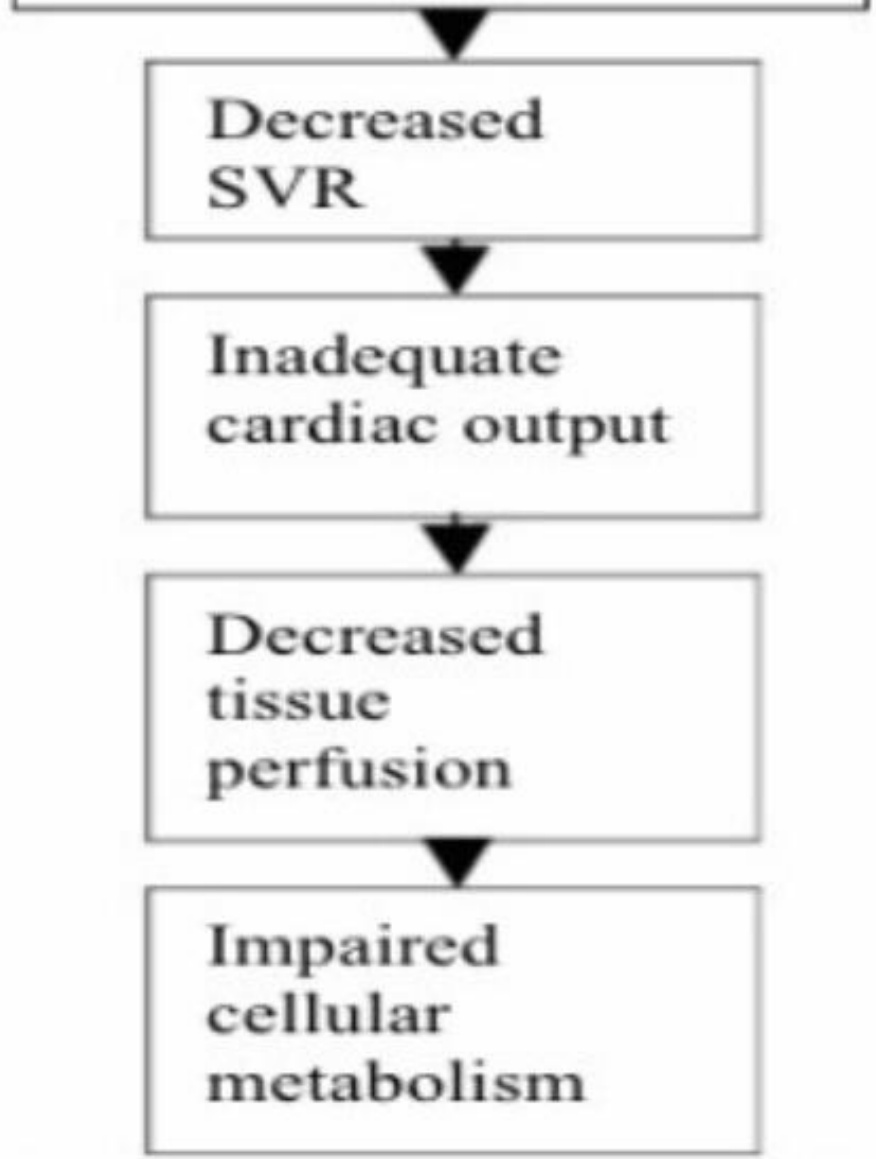
medications that affect the autonomic nervous system, which regulates breathing and other automatic bodily functions

PATHOPHYSIOLOGY

Predisposing Factor:
-Age
-gender

Precipitating Factor:
-trauma to the spinal cord resulting in the sudden loss of autonomic and motor reflexes below the injury level.




Decreased SVR
Inadequate cardiac output
Decreased tissue perfusion
Impaired cellular metabolism

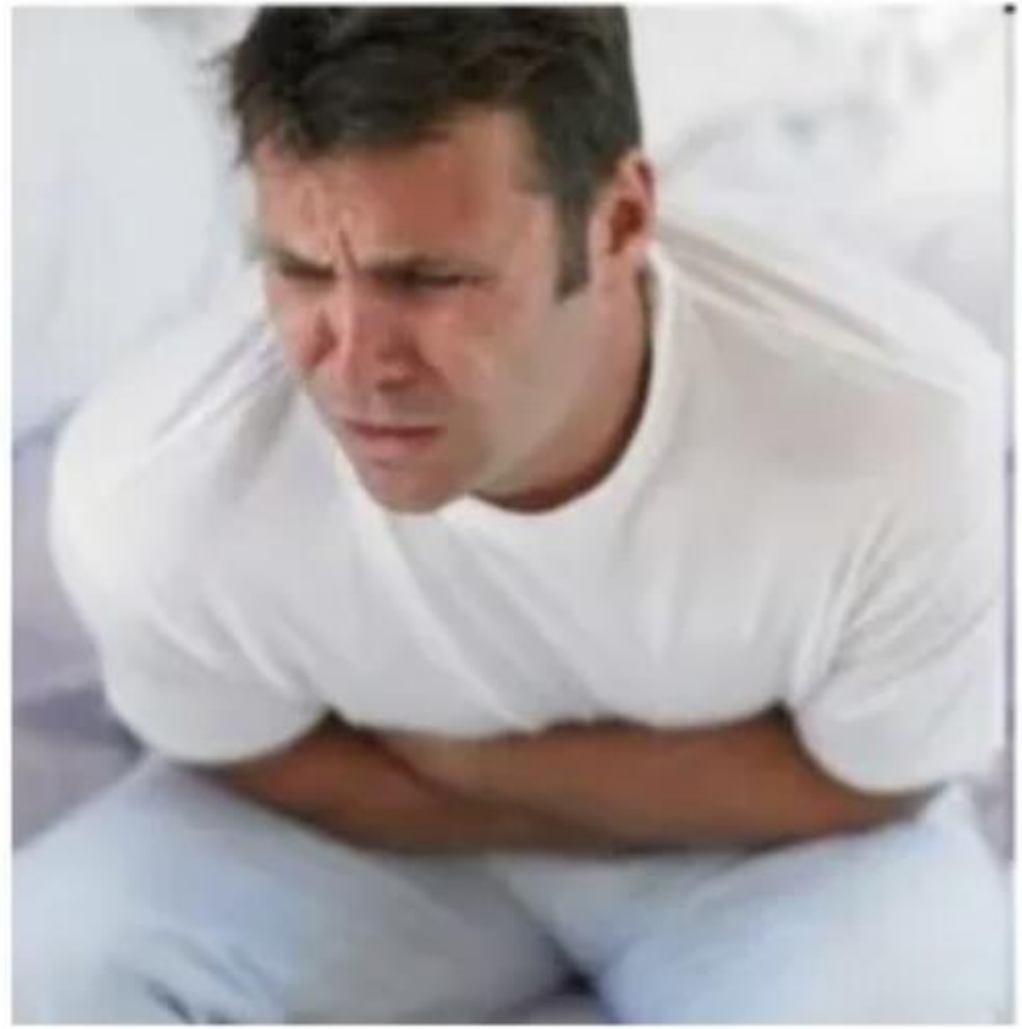
SYMPTOMS:

Emergency signs and symptoms

- Extreme back pain
- Weakness, incoordination or paralysis in any part of your body
- Numbness, tingling or loss of sensation in your hands, fingers, feet or toes
- Loss of bladder or bowel control
- Difficulty with balance and walking
- Impaired breathing after injury

Others symptoms….

- Dizziness
- Nausea
- Vomiting
- Blank stares
- Fainting
- Increased sweating
- Anxiety
- Pale skin



In more severe cases of neurogenic shock, you may experience:

- Difficulty breathing
- Chest pain
- Weakness from irregular blood circulation
- Bradycardia, or a slower heart rhythm
- Faint pulse
- Cyanosis, or discolored lips and fingers
- Hypothermia, or decreased body temperature

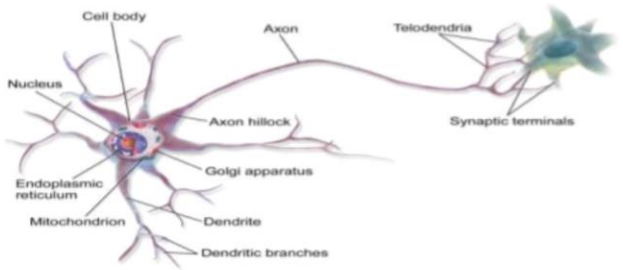
sign

- Hypothermia, hypotension, decreased CO, bradycardia, flaccid paralysis

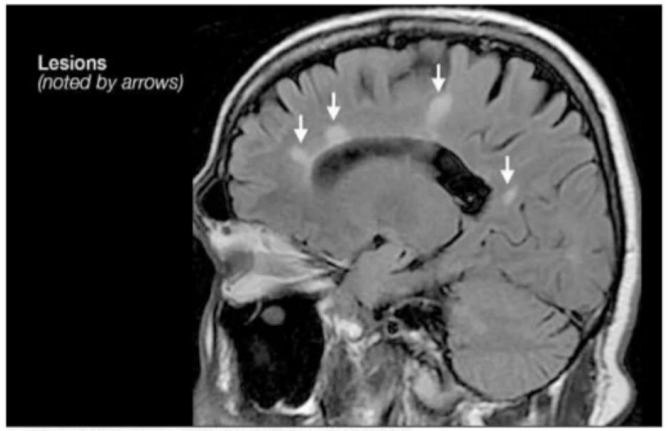
- Most common causes is spinal injury above T 6.
- Most rare form of shock

DIAGNOSTIC EVALUATION:

- Physical examination
- History collection

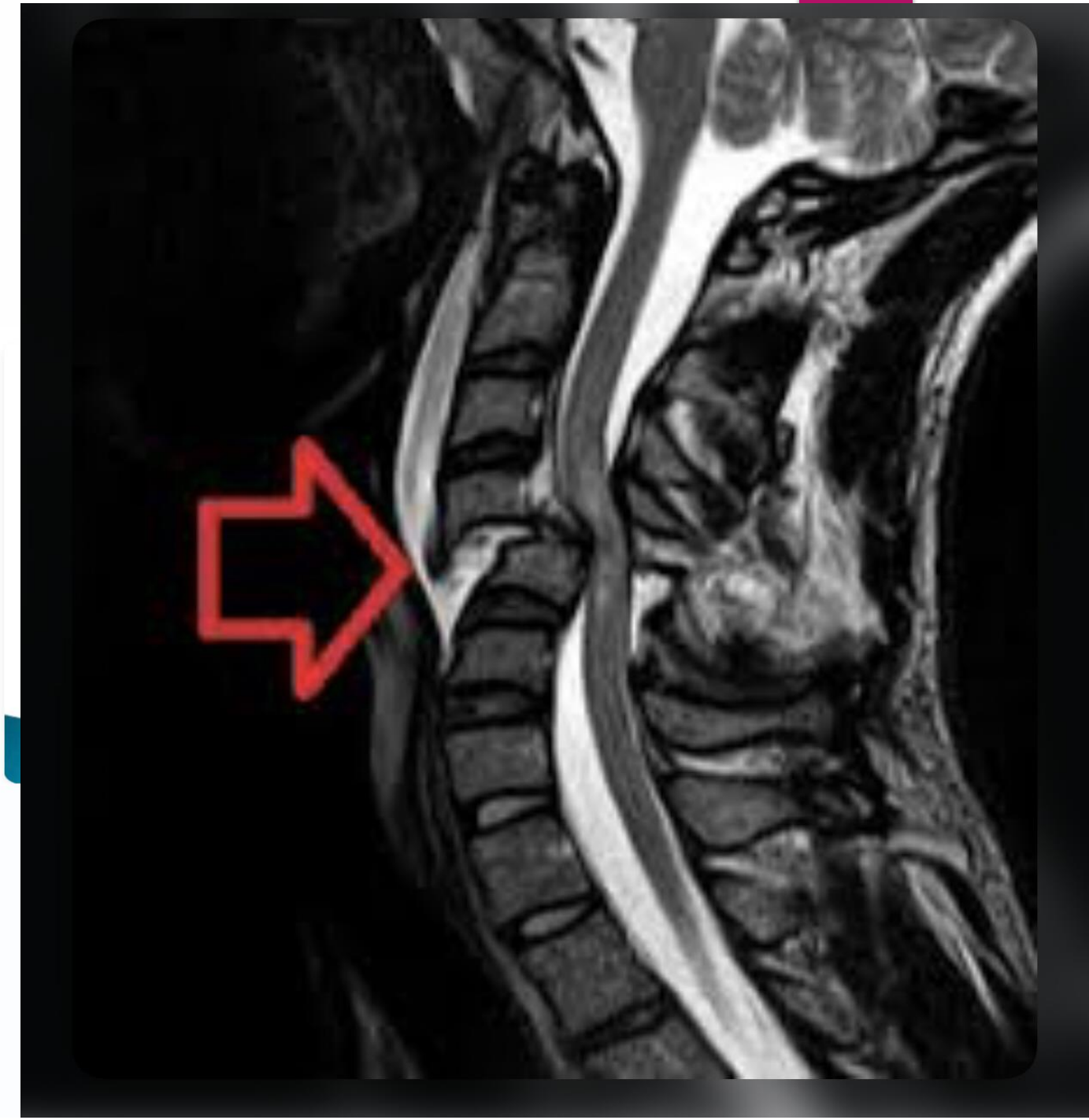


CT scan:



MRI scan:

Spinal injury or hemorrhage



MEDICAL MANAGEMENT:

Early (acute) stages of treatment:

In the emergency room, focus on:

- Maintaining the ability to breathe
- Preventing shock
- Immobilizing neck to prevent further spinal cord damage
- Avoiding possible complications, such as stool or urine retention, respiratory or cardiovascular difficulty, and formation of deep vein blood clots in the extremities

Management

- Main aim is to prevent complication and maintain perfusion

- Hypovolemic- with fluid
- Observe for fluid overload
- Vasopressors
- Hypothermia
- Treat hypoxia
- Maintain ventilator support

Anaphylactic shock

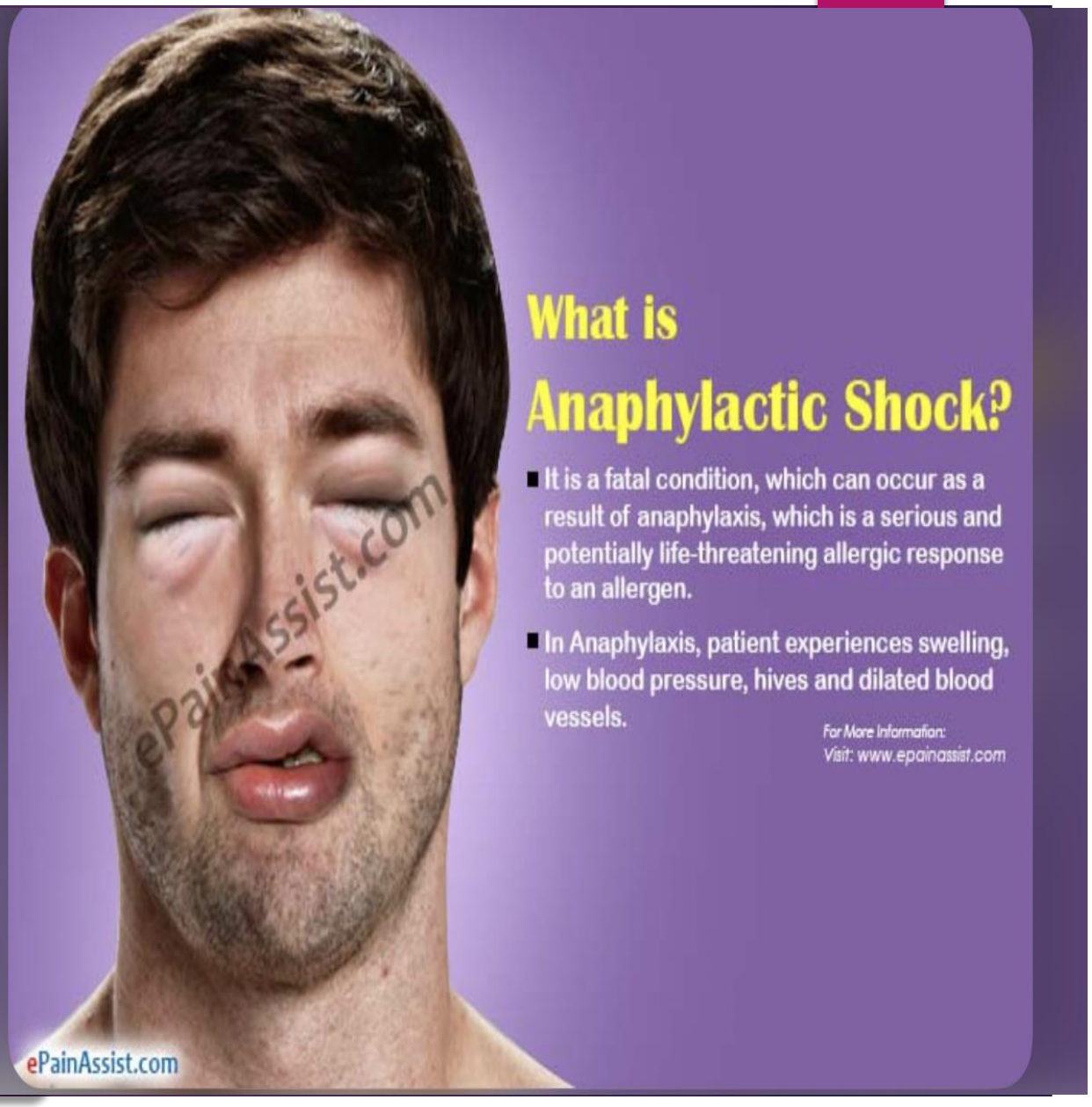
Anaphylactic Shock

Life-threatening allergic reaction

- Fast acting: within minutes
- Death usually due to swollen airway, causing respiratory distress/shock

Common causes

- Medications (ex. Penicillin)
- Food, drugs, food additives (ex. Peanuts, shellfish, nitrites, MSG)
- Insect stings
- Plant pollen



Anaphylactic shock

- A type of distributive shock that result from wide spread systemic allergic reaction to an antigen
- The hypersensitive reaction is life threatening

Stages of anaphylactic shock

- Changes in mast cell towards stimuli
- Activation of cell wall enzyme
- Meditators release
- Functional pathophysiology response
- Inflammation and release of secondary meditators

Triggers

Common causes:

- Foods
- Bee and wasp stings
- Drugs
- Latex rubber

Foods reported as triggers

- Peanuts [8]
- Fish
- Shellfish
- Eggs
- Milk
- Sesame, Pulses etc
- Others

Drugs causing anaphylaxis

- Antibiotics (especially penicillin)
- Anaesthetic agents
- Aspirin
- NSAID'S
- IV Contrast media
- Opioid analgesics

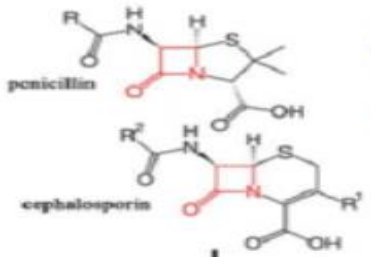
Rare Causes:

- Exercise
- Semen
- Vaccines

MECHANISMS AND TRIGGERS

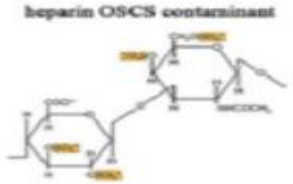
IMMUNOLOGIC: IgE/FcεRI

- Foods
- Medications
  eg. β-lactam antibiotics
- Insect stings/bites
- Natural rubber latex
- Other



IMMUNOLOGIC: OTHER

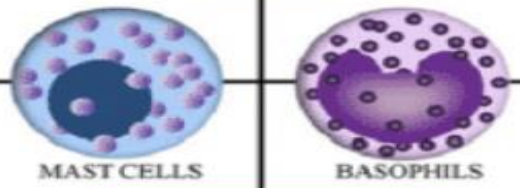
- IgG-antigen complexes
- Complement system activation
- Coagulation system activation



NON-IMMUNOLOGIC

- Exercise
- Cold air or water
- Medications, eg. opioids
- Other

CELLS



MEDIATORS

PREFORMED
HISTAMINE
TRYPTASE
CARBOXYPEPTIDASE A
CHYMASE

NEWLY GENERATED
LEUKOTRIENES
PROSTAGLANDINS
PLATELET-ACTIVATING FACTOR

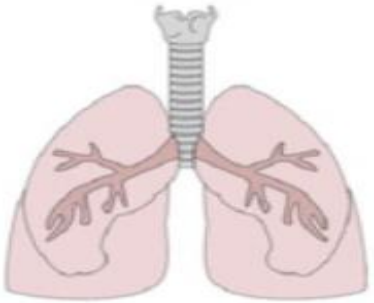
OTHER
CYTOKINES
CHEMOKINES

ORGAN SYSTEMS

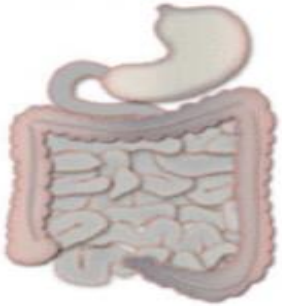
SKIN

MUCOSA

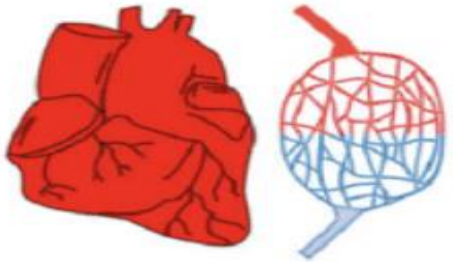
RESPIRATORY



GASTROINTESTINAL



CARDIOVASCULAR



CNS

Symptoms and signs:

- **Skin: 90 %of episodes**
  - Generalized hives.
  - Itching or flushing.
  - Swollen lips-tongue-uvula.
  - Per-orbital edema.
- **Respiratory: 70 % of episodes**
  - Nasal discharge and congestion.
  - Change in voice quality.
  - Sensation of throat closure or choking.
  - Tridor or wheeze.
  - Shortness of breath and cough.
- **Gastrointestinal: 45% of episodes**
  - Nausea.
  - Vomiting.
  - Diarrhea.
  - Crampy abdominal pain.
- **Cardiovascular: 45 % of episodes**
  - Hypotonia (collapse).
  - Syncope.
  - Dizziness.
  - Tachycardia.
  - hypotension.

Management

- Primary treatment

-Adrenaline 1:1000 with dose of 0.001ml/kg maximum 0.3 ml SC

-Can be repeated 3 times

-head extended , ventilated position

-O2 2-3 lit

- Place patient in shock position
- Pulmonic resuscitation
- Oro-pharyngeal airway
- E insertion
- Tracheostomy
- Cardiac compression

Treatment:

- Epinphrin
  - IM 0.3mg , 1:1000
  - IV 0.15mg , 1:10,000
- Antihestsmin
- Corticosteroid
- Bronchodilator
- Oxygen
- Fluids and vasopressors

Done by Haneen Yahya