CARDIOGENIC SHOCK

DONE BY: ABDULLAH BAYADAH
KHALED BAYAYDAH
YOUSEF MAHADEIN

SHOCK
“(circulatory failure)

-Shock is life-threatening circulatory disorder characterized by **generalized inadequate blood flow** (**HYPOPERFUSION**) through the body → that lead to **tissue hypoxia** → tissue cells cannot meet their metabolic needs → multiple organ failure

-**HYPOTENSION** is a common presentation of shock but the terms are not synonymous

CIRCULATORY SHOCK

WITH decreased cardiac output

- Due to cardiac abnormalities that decrease pumping of blood (ex. MI, arrhythmia, valvular dysfunction)
- Due to decrease in venous return = decrease CO , mostly due to diminish blood volume, obstruction of flow

WITHOUT decreased cardiac output

- Increase tissues metabolic rate → increase oxygen demands → normal cardiac output is inadequate to meet the tissue needs

➢ Inadequate tissue perfusion despite normal or elevated cardiac output

- abnormal tissue perfusion patterns, so most of the cardiac output is passing through blood vessels besides those that supply the local tissues with nutrition

•**Extreme Physical Exertion**: During high-intensity exercise, metabolic needs can spike. If oxygen delivery can't keep pace due to peripheral vasodilation or other factors, it can lead to shock.

•**Hyperthermia**: Elevated body temperature can significantly increase metabolic demands. If the body cannot effectively increase blood flow to dissipate heat, it can result in shock.

Hypovolemic shock

- Due to poor intake or excessive loss of fluids
- According to etiology subdivides:

a) Haemorrhagic: Trauma – Upper GI bleeding – Postpartum hemorrhage

b) Non-haemorrhagic fluid loss: Diarrhea – Vomiting – Burns – 3rd space loss (bowel obstruction – pancreatitis)

- Pathophysiology: Loss of intravascular fluid volume → ↓ preload and SV → ↓ CO → compensatory ↑ SVR + HR

Obstructive shock

- Due to blocking of blood flow to or from the heart
- Causes:

1) ↓ Diastolic filling: Cardiac tamponade - Constrictive pericarditis - Restrictive cardiomyopathy

2) ↓ Venous return: Tension pneumothorax

3) ↑ Ventricular afterload: Massive pulmonary embolism (PE) - Aortic stenosis

- Pathophysiology: obstruction of the heart or its great vessels → inability of the heart to circulate blood → ↓ CO → compensatory ↑ SVR

Distributive shock

- Due to redistribution of body fluids
- Pathophysiology: vasodilation with or without capillary leakage → redistribution of fluid from the intravascular to the extravascular compartment
- Types:

I. Septic (aka. Warm shock: warm extremities – normal capillary refill – strong peripheral pulses)

II. Neurogenic

III. Anaphylactic

- low SVR and normal or high cardiac output state

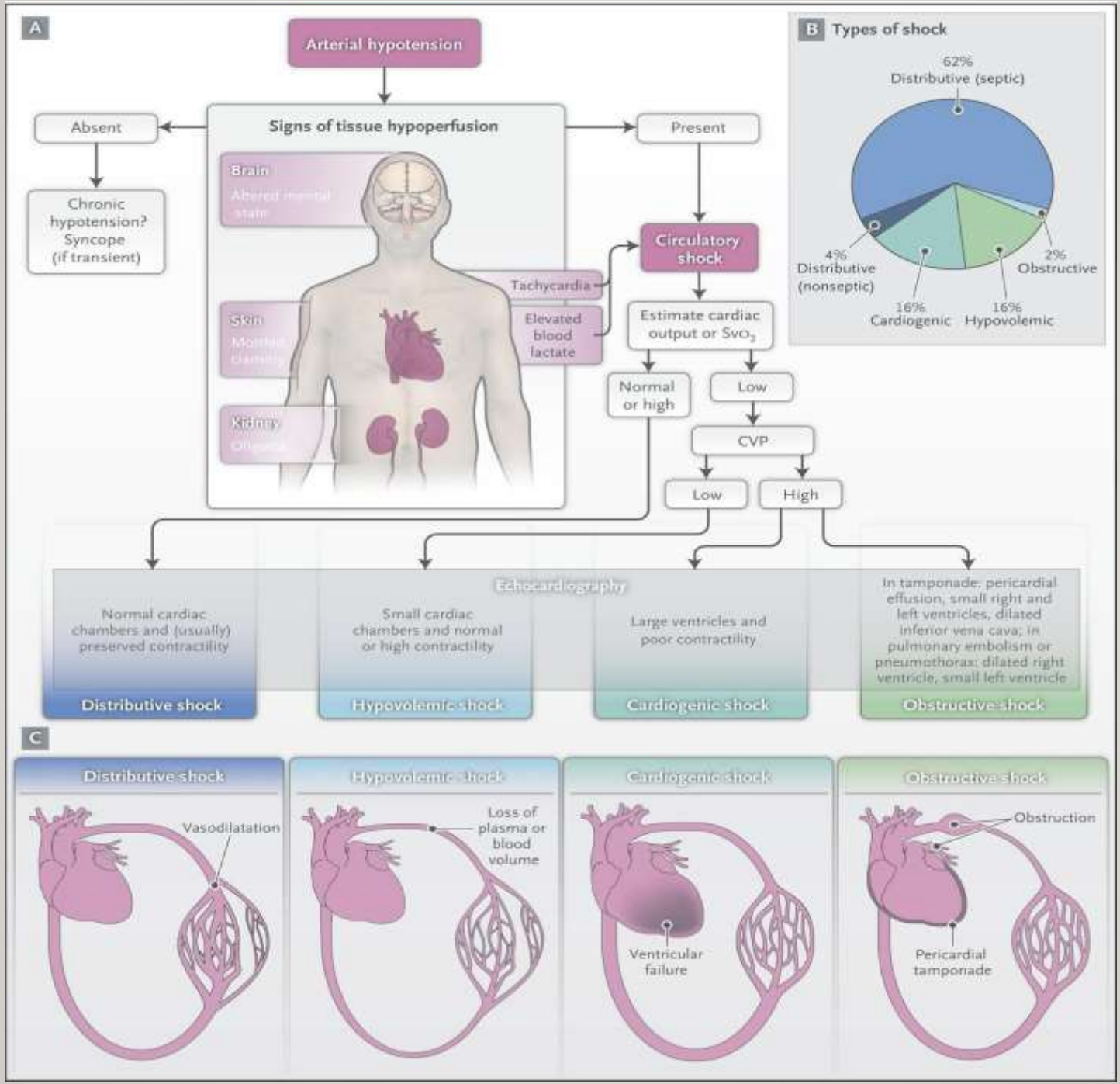
Cardiogenic Shock

- Due to poor pumping function or circulatory overload

SIGNS & SYMPTOMS OF ALL FORMS OF SHOCK

1) Hypotension
2) Tachycardia
3) Clinical signs of malfunction of underperfused organs:

- Lactic acidosis [hyperlactatemia] (due to anaerobic metabolism)
- Renal (Oliguria / anuria)
- CNS (Altered mental status; disorientation and confusion)
- Cutaneous (skin that is cold and clammy, with vasoconstriction and cyanosis)


A
Arterial hypotension
Absent
Chronic hypotension? Syncope (if transient)
Signs of tissue hypoperfusion
Brain
Altered mental state
Skin
Mottled, clammy
Kidney
Oliguria
Present
Tachycardia
Elevated blood lactate
Circulatory shock
Estimate cardiac output or SvO_2
Normal or high
Low
CVP
Low
High
Normal cardiac chambers and (usually) preserved contractility
Distributive shock
Echocardiography
Small cardiac chambers and normal or high contractility
Hypovolemic shock
Large ventricles and poor contractility
Cardiogenic shock
In tamponade: pericardial effusion, small right and left ventricles, dilated inferior vena cava; in pulmonary embolism or pneumothorax: dilated right ventricle, small left ventricle
Obstructive shock
B Types of shock
62% Distributive (septic)
4% Distributive (nonseptic)
2% Obstructive
16% Cardiogenic
16% Hypovolemic
C
Distributive shock
Vasodilatation
Hypovolemic shock
Loss of plasma or blood volume
Cardiogenic shock
Ventricular failure
Obstructive shock
Obstruction
Pericardial tamponade

HEMODYNAMIC CHANGES

-Shock is characterized by its effect on:

1. Cardiac output
2. SVR
3. Volume status → volume status is assessed via:

    -Jugular venous pressure

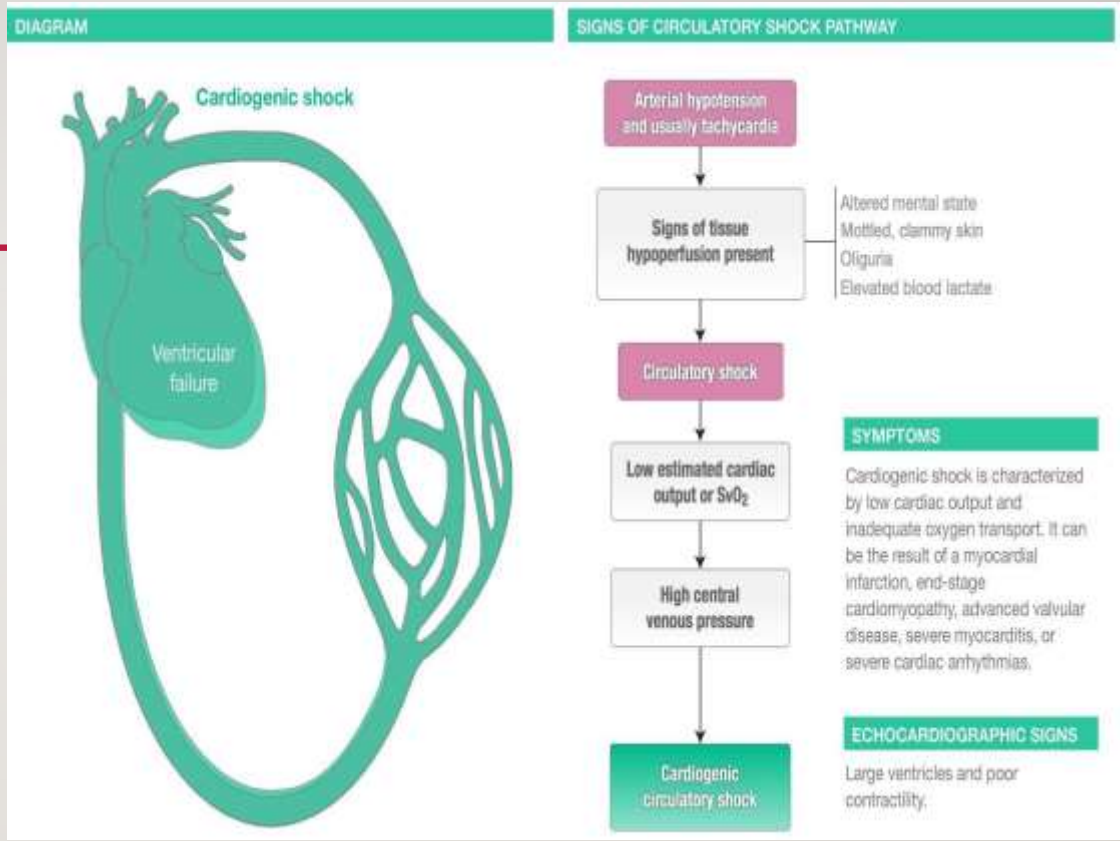
    -Pulmonary capillary wedge pressure [PCWP]

“

TABLE 1-6 Hemodynamic Changes in Shock States

Shock	Cardiac Output	SVR	PCWP
Cardiogenic	↓	↑	↑
Hypovolemic	↓	↑	↓
Distributive			
Neurogenic	↓	↓	↓
Septic	↑	↓	↓
Obstructive	↓	↑	Variable

CARDIOGENIC SHOCK



- Occurs when heart is unable to generate a cardiac output sufficient to maintain tissue perfusion

  (Tissue demands > tissue perfusion)

- Can be defined as a:
- systolic BP <90 with urine output <20 mL/hr and adequate left ventricular filling pressure (LV filling pressure usually elevated in cardiogenic shock)

**Cardiogenic shock is the top cause of death in people who had a previous heart attack

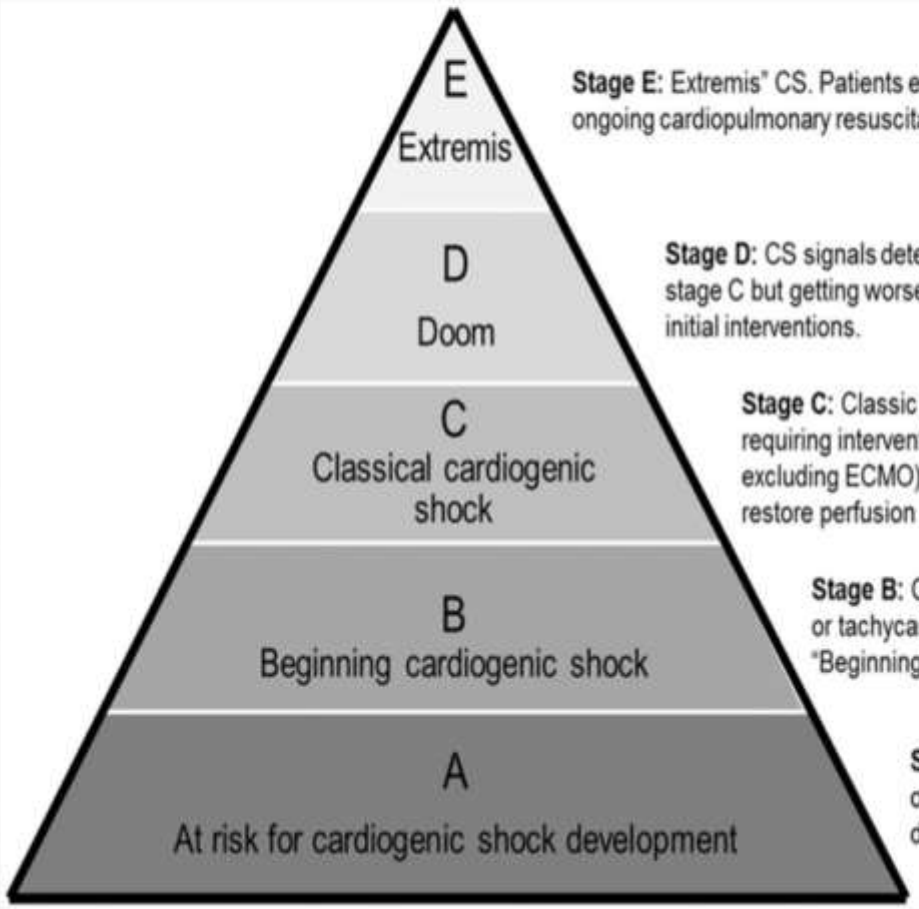
CARDIOGENIC SHOCK CAN BE DEFINED BY THE FOLLOWING:

1. Cardiac output is decreased, and SVR is increased (Because the body tries to increase the BP by vasoconstricting the vessels, so the BP becomes normal again)

2. Systolic BP is less than 80 mmhg without inotropic or vasopressor support, or less than 90 mmhg with inotropic or vasopressor support, for at least 30 mins or Mean arterial pressure 30 mmhg below baseline

3. A cardiac index of <2.2 L/min

Cardiogenic shock pyramid according to **recent** proposal



Stage E: Extremis" CS. Patients experiencing cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) and/or ECMO.

Stage D: CS signals deteriorating or "Doom". Similar to stage C but getting worse and failing to respond to initial interventions.

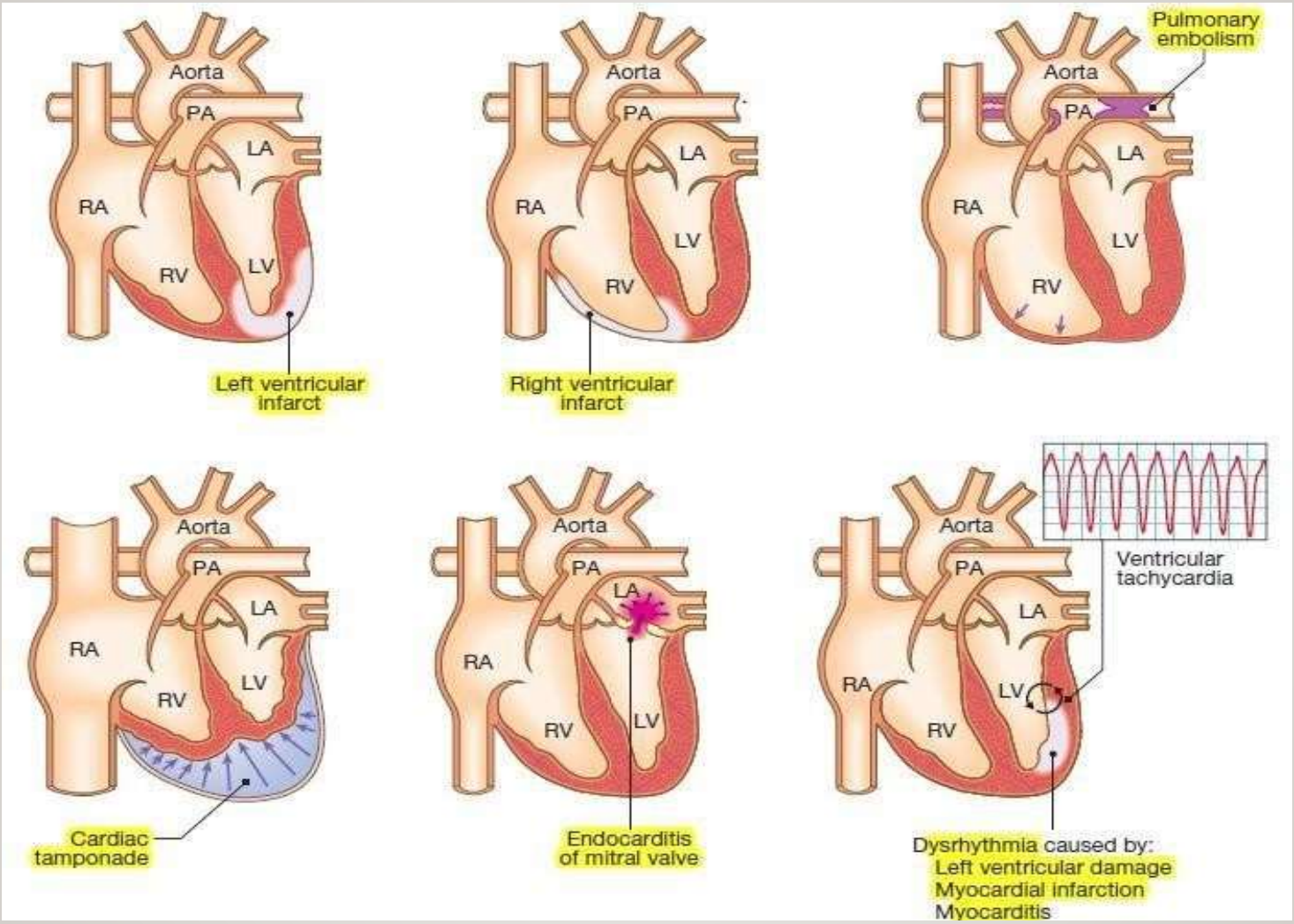
Stage C: Classic CS. Manifest CS with hypoperfusion requiring intervention (inotropes, vasopressors or MCS, excluding ECMO) beyond volume resuscitation to restore perfusion

Stage B: Clinical evidence of relative hypotension or tachycardia without hypoperfusion being at "Beginning" of CS (pre-shock).

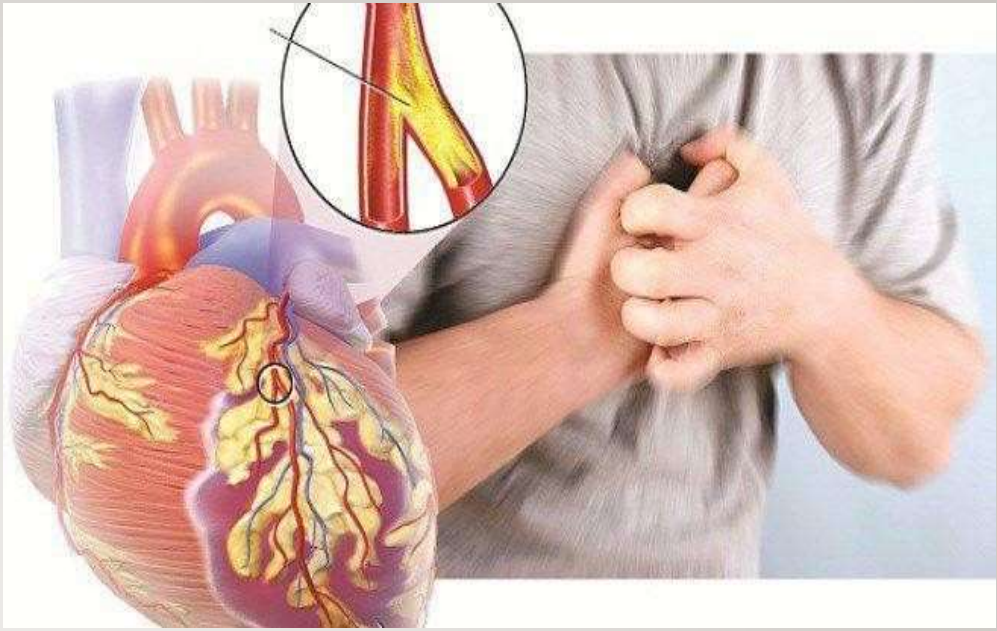
Stage A: Currently no signs/symptoms of CS, but being "At risk" for its development.

CAUSES

1. Myocardial infarction (MI): most common cause
2. Arrhythmias
3. Decompensated Heart failure
4. Cardiomyopathy
5. Myocarditis
6. Ventricular septal defect, ventricular rupture
7. Valve defects: severe aortic or mitral regurgitation
8. Blunt cardiac trauma
9. Certain drugs (e.g., beta blockers, calcium channel blockers)


Aorta
PA
LA
RA
RV
LV
Left ventricular infarct
Aorta
PA
LA
RA
LV
RV
Right ventricular infarct
Pulmonary embolism
Aorta
PA
LA
RA
LV
RV
Aorta
PA
LA
RA
RV
LV
Cardiac tamponade
Aorta
PA
LA
RA
RV
LV
Endocarditis of mitral valve
Ventricular tachycardia
Aorta
PA
LA
RA
RV
LV
Dysrhythmia caused by:
Left ventricular damage
Myocardial infarction
Myocarditis

RISK FACTORS



1. **Age: old age**
   - **Delayed response to treatment**
   - **Myocardial dysfunction**
2. **Gender; females .**
3. **Race & ethnicity .**
4. **History of heart disease .**
5. **Medical conditions; (diabetes, hypertension, obesity, pneumothorax, sepsis).**
6. **Medical procedure .**

In relation to race and ethnicity : asian people tends to have the hightest risk for CS
Because :

1- Many Asians tend to have higher levels of visceral fat
2- Genetic Factors: Genetic variations in Asians may affect cholesterol metabolism
3- Higher Rates of Coronary Artery Disease (CAD)

SIGNS & SYMPTOMS OF CARDIOGENIC SHOCK

- Tachycardia .
- Decrease Blood pressure (sys BP<90mmHg).
- Lactic acidosis (anaerobic metabolism).
- Oliguria .
- Peripheral pulses are rapid and faint and may be irregular if arrhythmias are present .
- heart sounds are distant, and third and fourth heart sounds may be present .
- Chest pain .

SIGNS & SYMPTOMS OF CARDIOGENIC SHOCK

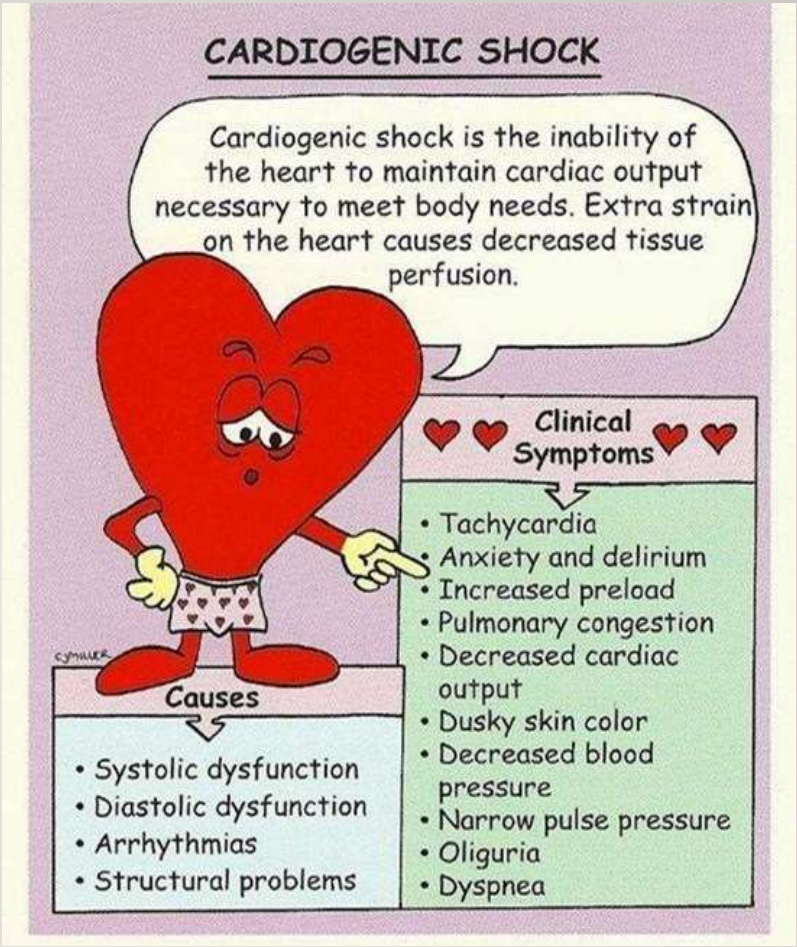
- signs of volume overload : (edema) .
- Pulmonary congestion manifested as rales on pulmonary examination .
- Engorged neck veins (JVP elevated) .
- Cool, clammy extremities .(COLD SHOCK)
- Autonomic symptoms, including nausea, vomiting, and sweating.
- Delirium and altered mental status

NOTE : that Jugular Venous Pulse is only elevated in cardiogenic & obstructive shock.

Complications of cardiogenic shock

If not treated immediately, cardiogenic shook can lead to :

Liver damage , kidney damage , paralysis & it can lead to sudden death.



DIAGNOSIS

laboratory studies:

- complete blood cell (CBC) count
- biochemical profile: electrolytes, renal function (eg, urea and creatinine levels), and liver function tests (eg, bilirubin, [AST],[ALT])
- Cardiac enzymes which include creatine kinase (CK) and its subclasses, troponin, myoglobin, and LDH
- arterial blood gas (ABG): acidosis as↑ Lactate (> 2 mEq/L) due tissue hypoperfusion and is associated with poorer outcomes.

Electrolytes : hypokalemia, hypomagnesemia, acidosis.

DIAGNOSIS

ECG

- look for signs of ischemia (i.e., STsegment changes) or arrhythmia(ventricular or atrial tachyarrhythmia)

Echocardiogram

- can diagnose a variety of mechanical complications of MI, identify valve disease, estimate EF, look for pericardial effusion, etc

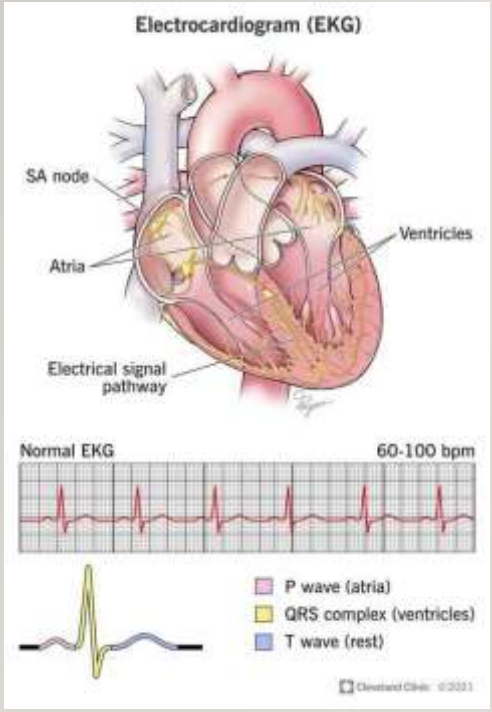
Invasive hemodynamic monitoring (Swan-Ganz catheterization)

- is very useful for helping to exclude other causes and types of shock (eg, volume depletion, obstructive shock, and septic shock)
- Indicate : PCWP, pulmonary artery pressure , cardiac output, CI, SVR
- KEEP cardiac output >4L/min

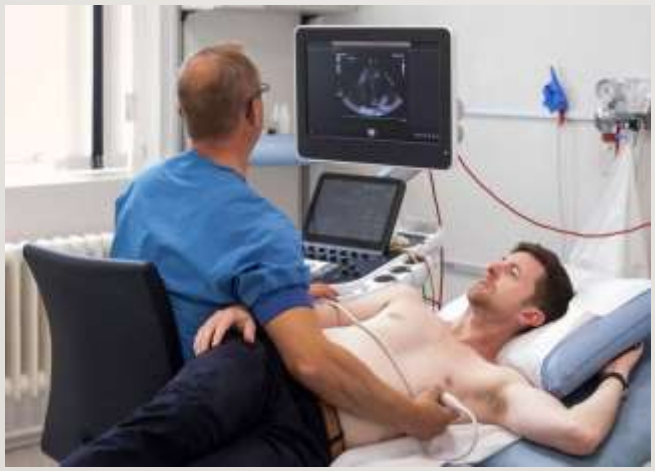
  CI >2.2, PCWP <18mmHg.

DIAGNOSIS

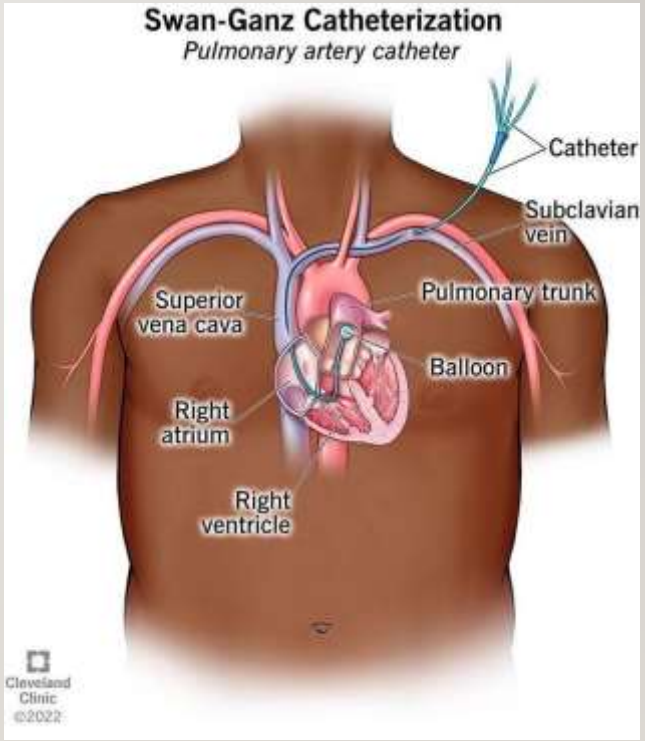
ECG



Echocardiogram

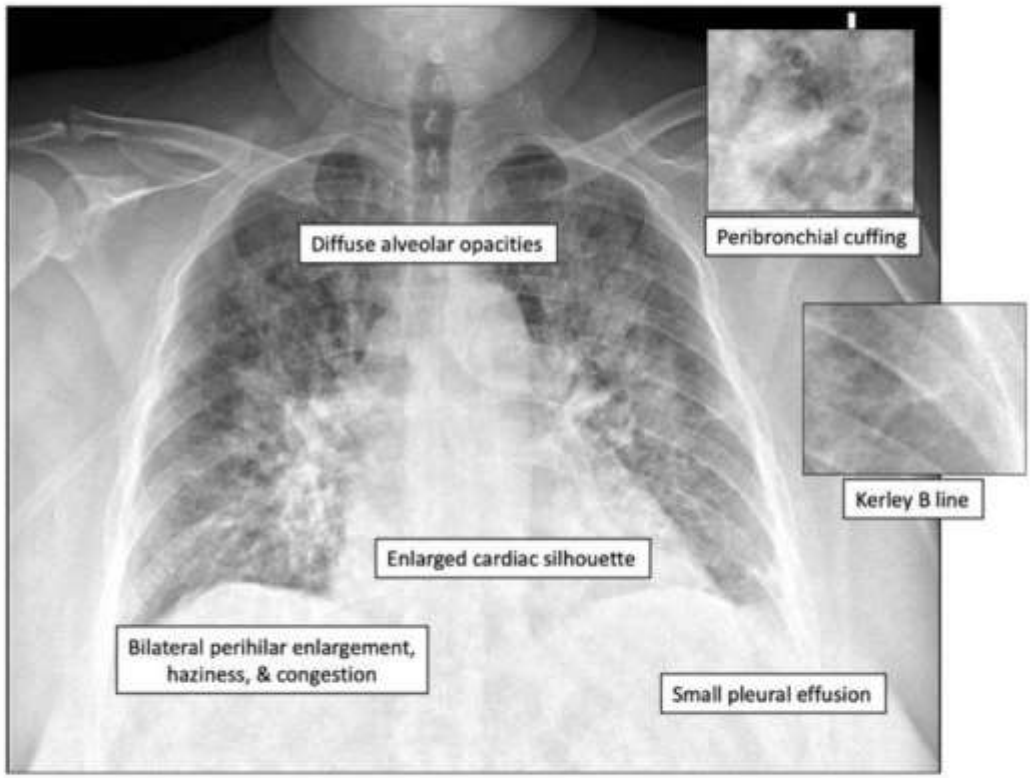


Invasive hemodynamic monitoring (Swan-Ganz catheterization)



Imaging

- Chest x-ray
- Echocardiogram



MANAGEMENT

ABC

- **Supplemental oxygen.**
- **Pain relief: Morphine.**
- **Fluid resuscitation to correct hypovolemia and hypotension, unless pulmonary edema is present.**
- **Continuous ECG monitoring.**

MAP = CO * SVR

MANAGEMENT

Cardiac support

- **Vasopressors** for hypotension which is unresponsive to fluids: **Norepinephrine and dopamine** are considered **first-line drugs**.
- **inotropic**:
  - **Dobutamine:** increase inotropy and simultaneously decrease afterload (SVR), enhancing cardiac output
  - **Milrinone:** is a phosphodiesterase inhibitor which increases inotropy and decreases SVR. It is often used in conjunction with other inotropes.
- **Loop diuretics :** for preload reduction in patients with volume overload (furosemide)

MANAGEMENT

Afterload reduction

a. IV agents like sodium nitroprusside can be used to quickly reduce afterload. There is a risk of hypotension with rapidly increasing the dose.

b. Oral agents like hydralazine and captopril can reduce afterload, are short acting.

IV fluids

are likely to be harmful if left ventricular pressures are elevated.

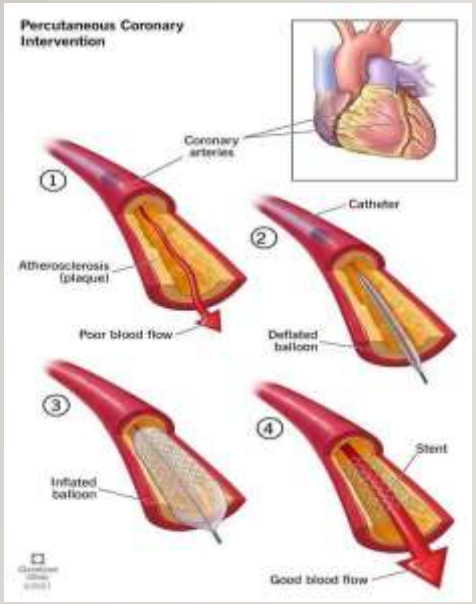
Patients usually need diuretics. Fluid bolus only in cases of hypotension and/or PCWP < 15 mm Hg.

MANAGEMENT

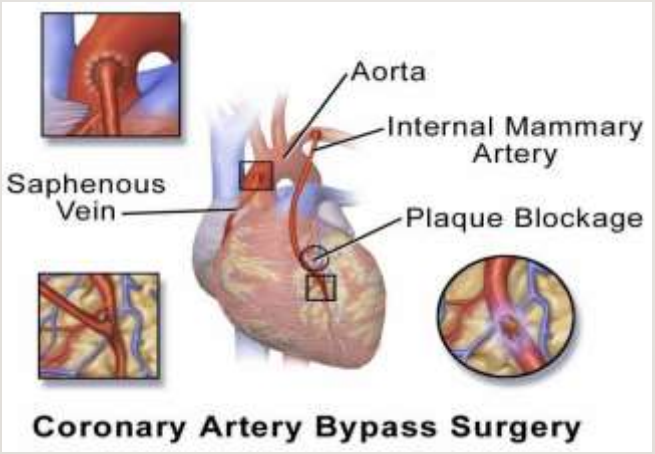
- Identify and treat underlying cause
- Acute MI: Emergent revascularization with PCI (or CABG) has been shown to improve survival.
- If cardiac tamponade :pericardiocentesis/surgery
- valvular abnormalities: Surgical correction
- Treatment of arrhythmias
- Intra-aortic Balloon Pump
- surgical valve repair or replacement
- Decrease SVR : Nitro-glycerine ,Nitroprusside

MANAGEMENT

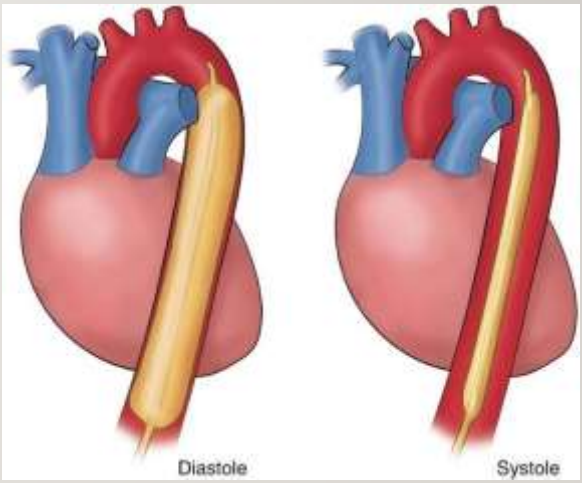
PCI



CABG



Intra-aortic Balloon Pump



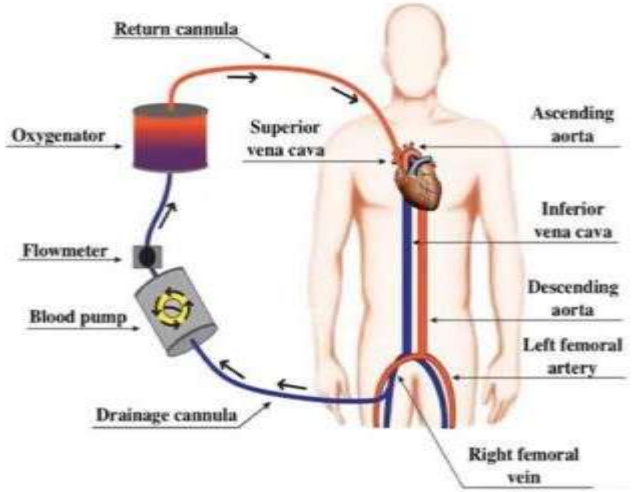
a. Decreases afterload
b. Increases cardiac output
c. Decreases myocardial oxygen demand

Coronary artery bypass grafting (CABG) and percutaneous cardiac intervention (PCI)

- Either PCI or CABG is the treatment of choice for cardiogenic shock
- PCI should be initiated within 90 minutes after presentation
- PCI remains helpful, as an acute intervention, within 12 hours after presentation to open coronary arteries that are narrowed or blocked by the buildup of plaque
- CABG improve blood flow to the heart. This procedure is usually done as soon as possible after a diagnosis of cardiogenic shock
- Thrombolytic therapy is second best but should be considered if PCI and CABG are not immediately available

reduces the likelihood of subsequent development of cardiogenic shock after the initial event.

Extracorporeal membrane oxygenation (ECMO)



It pumps and oxygenates a patient's blood outside the body, allowing the heart and lungs to rest

Acute Cardiogenic Pulmonary Edema

The "L-M-N-O-P's"

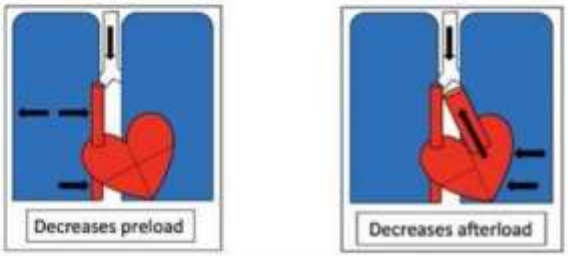
Lasix (Furosemide) – reduce preload, remove fluid

Morphine – pulmonary vasodilator, decrease dyspnea

Nitrates – reduce preload, pulmonary vasodilator

Oxygen – treat acute hypoxemic respiratory failure

Positive pressure ventilation



PROGNOSIS OF CARDIOGENIC SHOCK

- **Mortality Rates & Outcomes**
- **High mortality rate**: **40-50%** despite advanced treatment.
- **Early intervention improves survival**: **PCI, mechanical support, and vasopressors** increase survival rates.
- **Worse prognosis** if:
  - **Delayed treatment (>6 hours)**
  - **Multiorgan failure** (renal, hepatic, cerebral dysfunction)
  - **Severe left ventricular dysfunction (EF <20%)**
  - **Elderly patients & comorbidities (diabetes, CKD)**