Cardiogenic Shock

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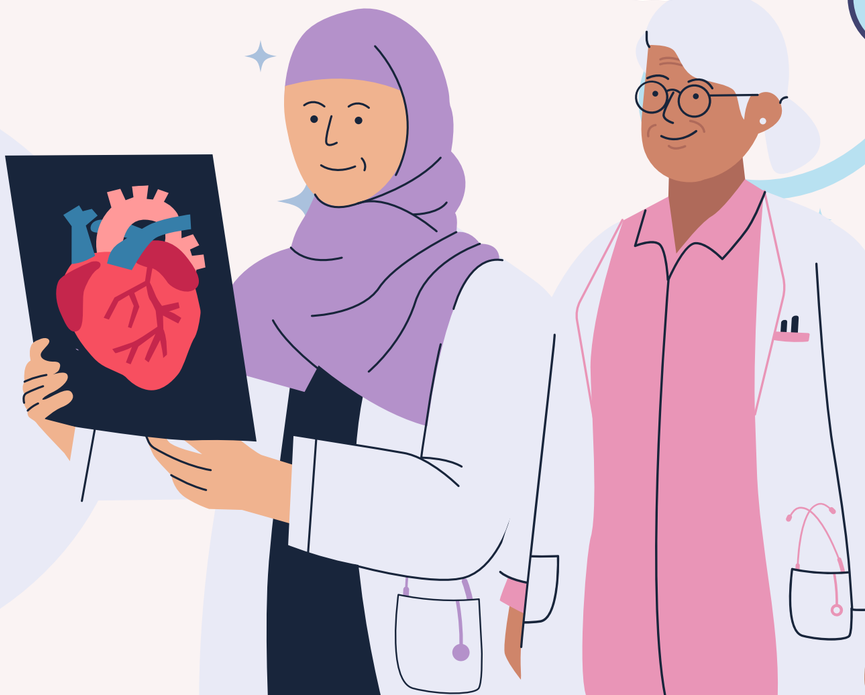
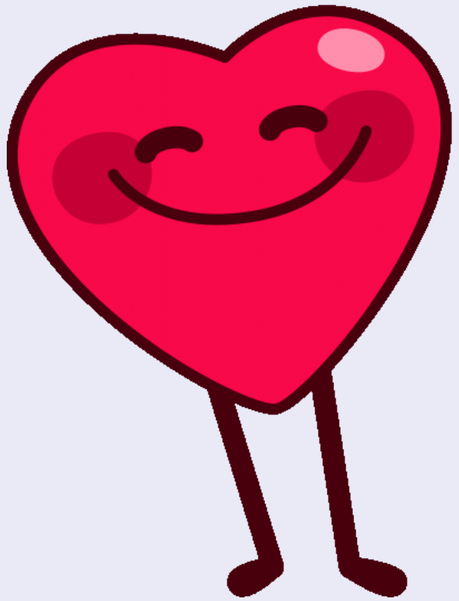


Basic of Shock

► Shock is a life-threatening disorder of the circulatory system that results in inadequate organ perfusion and tissue hypoxia, leading to metabolic disturbances and, ultimately, irreversible organ damage.

► Shock index = pulse rate/systolic blood pressure

- Normal range: 0.4–0.7
- > 1 (positive shock index): consistent with circulatory shock



► Shock is characterized by its effect on cardiac output, SVR, and volume status (volume status is assessed via jugular venous pressure or pulmonary capillary wedge pressure [PCWP]).The hemodynamic changes associated with different types of shock. Circulatory shock can occur either by Decrease cardiac output or Without decrease cardiac output.

Basic of Shock

There are four main categories of shock:

• **Hypovolemic** (poor intake or excessive loss of fluids).

• **Cardiogenic** (poor pumping function or circulatory overload).

• **Distributive** (low SVR and high cardiac output states which include septic, anaphylactic,neurogenic shock, and severe hepatic failure).

• **Obstructive** (i.e., massive PE, cardiac tamponade, tension pneumothorax)

► **Management** of shock involves ABCs (airway, breathing, and circulation) should be addressed for all patients in shock and treatment of the underlying cause.

► Shock is associated with a very high mortality rate.

Basic of Shock

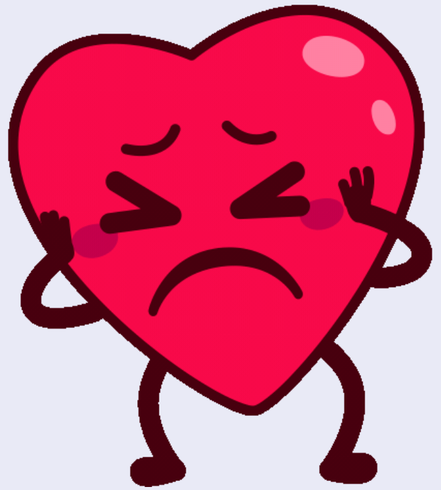
TABLE 1-6 Hemodynamic Changes in Shock States

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

• 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

Etiology

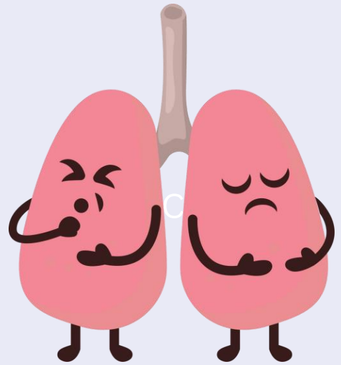
- **Myocardial Infarction (MCC)**
- • Arrhythmias
- Cardiomyopathy
- • Blunt cardiac trauma
- • Ventricular septal defect, ventricular rupture
- • Valve defects: severe aortic or mitral regurgitation
- • Certain drugs (e.g., beta blockers, calcium channel blockers)
- • Myocarditis
- • After cardiac arrest
- • Heart failure

Patho physiology

•Underlying event causes dysfunction of the heart cardiac contractility and/or SV → ↓ CO

• Systemic circulation: ↓ CO and ↓ BP → ↑ catecholamines → vasoconstriction and ↑ myocardial oxygen demand → ↑ renin-angiotensin-aldosterone system → further ↑ vasoconstriction and retention of sodium and water → shunting of blood to the brain and vital organs → insufficient perfusion of peripheral organs

• Pulmonary circulation: ↓ cardiac contractility and/or ↓ SV → ↑ pulmonary hydrostatic pressure → pulmonary edema



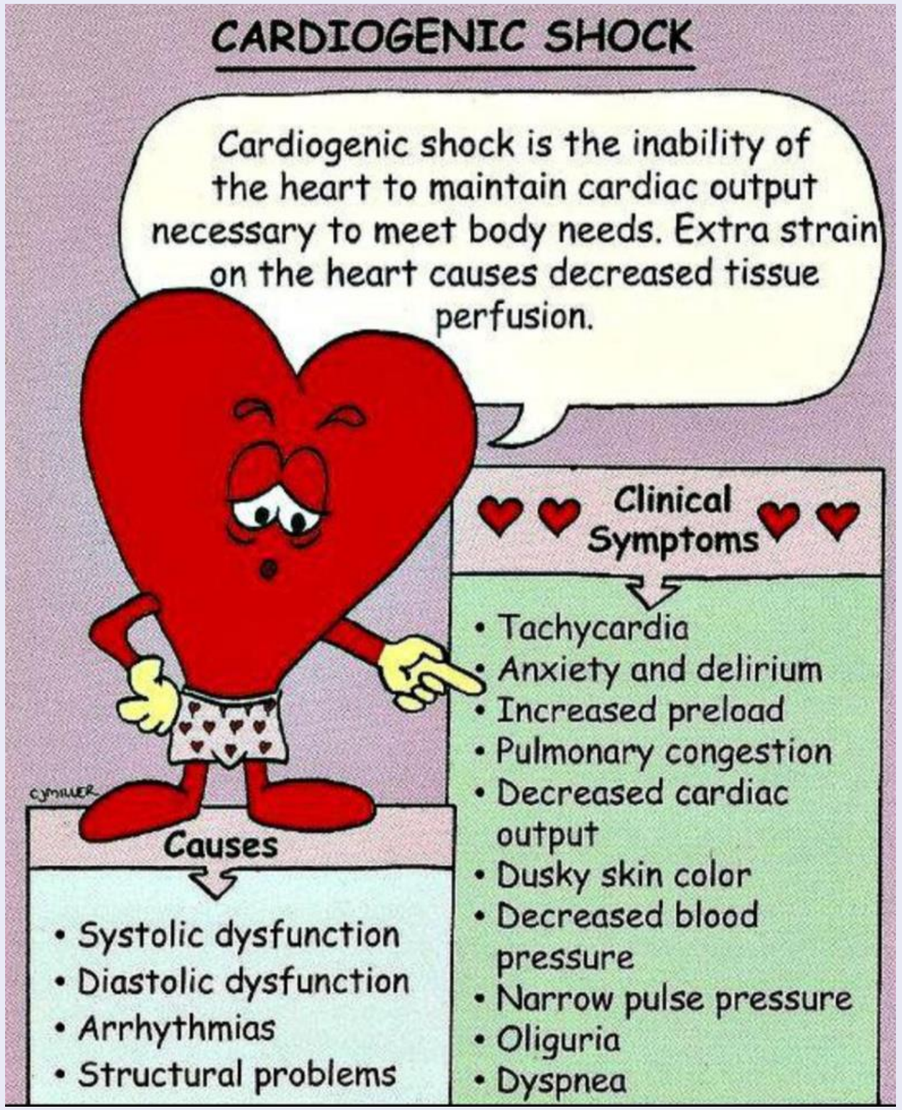
Clinical Features

1. Tachycardia
2. Hypotension
3. Decrease pulse pressure
4. Tachypnea
5. Altered consciousness: Agitation, confusion, disorientation, Lethargy, stupor, coma (e.g., low GCS score)
6. Oliguria: urine output < 0.5 mL/kg/hour
7. Cold extremities and Slow capillary refill time
8. Signs of congestive heart failure alongside shock (e.g., ↑ JVP, crackles on lung auscultation)

Clinical Features

- Hypotension may be absent in some patients with preexisting hypertension in which end-organ perfusion may be impaired without hypotension.

- Tachycardia may be absent in patients on beta blockers



Diagnosis

1. ECG—look for signs of ischemia (i.e., ST-segment changes), arrhythmia (ventricular or atrial tachyarrhythmia) or cardiomyopathy.

2. Echocardiogram—can diagnose a variety of mechanical complications of MI, identify valve disease,estimate EF, look for pericardial effusion, etc.

3. Hemodynamic monitoring with a Swan–Ganz catheter may be indicated: PCWP, pulmonary artery pressure, cardiac output, cardiac index, SVR—keep cardiac output >4 L/min, cardiac index >2.2, PCWP <18 mm Hg.

4. ABG— Acidosis as↑ Lactate (> 2 mEq/L) due tissue hypoperfusion and is associated with poorer outcomes.

5- Chest X-Ray - Pulmonary edema, cardiac enlargement, or pleural effusions.

Diagnosis

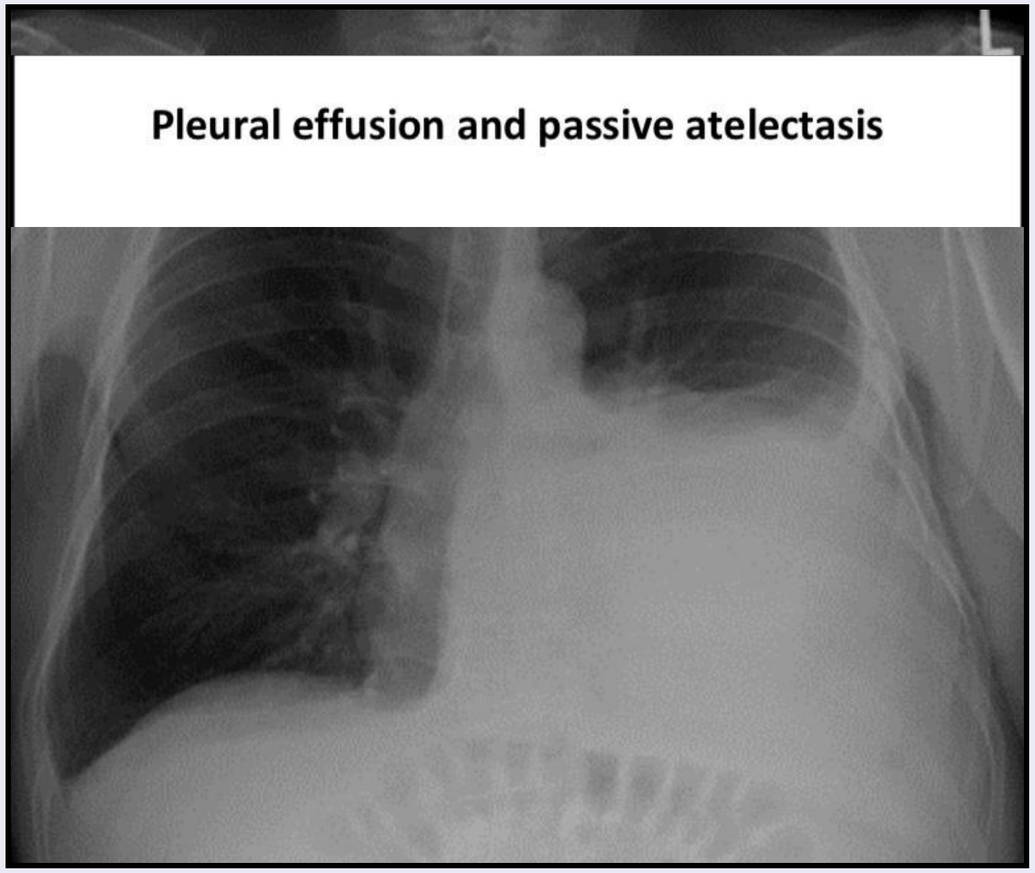
6- Complications or end-organ dysfunction:

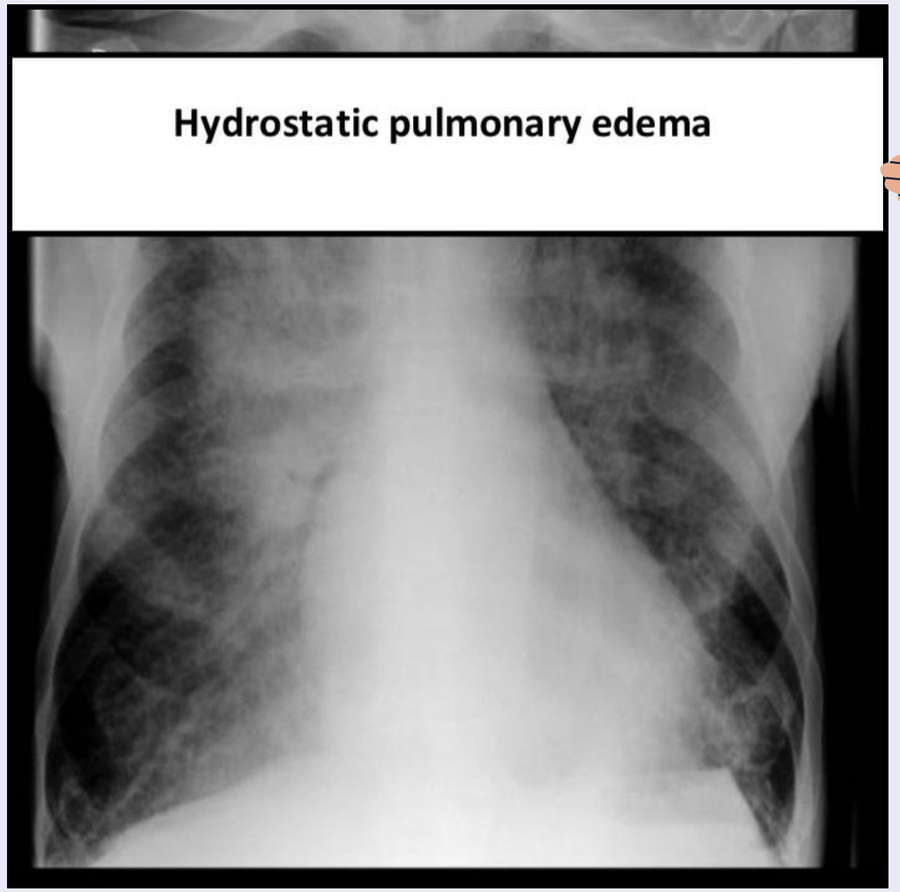
- Hypoglycemia or hyperglycemia.
- Electrolyte abnormalities.
- Renal function tests**:** ↑ BUN, ↑ creatinine, other signs of AKI or ATN (e.g., on urinalysis).
- Liver chemistries**:** elevated in shock liver.
- Coagulation panel**:** suggestive of DIC, acute traumatic coagulopathy, or acute liver failure.

7- BNP and NT-proBNP compared to baseline

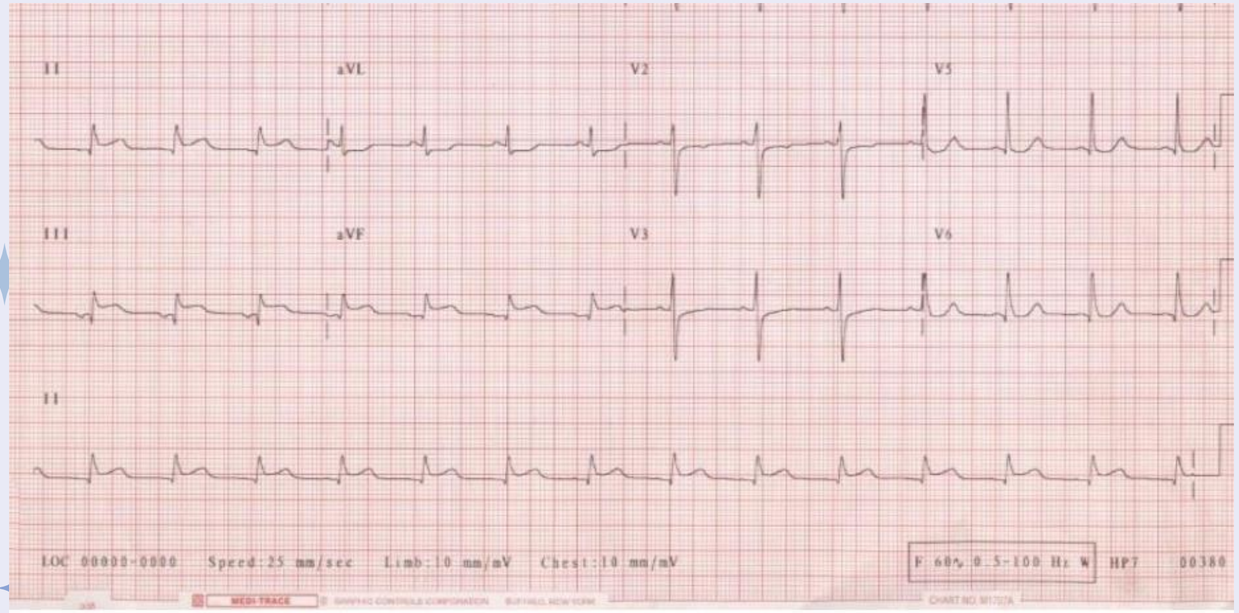
8- Cardiac markers: ↑ troponin I and troponin T in acute coronary syndrome

X-RAY

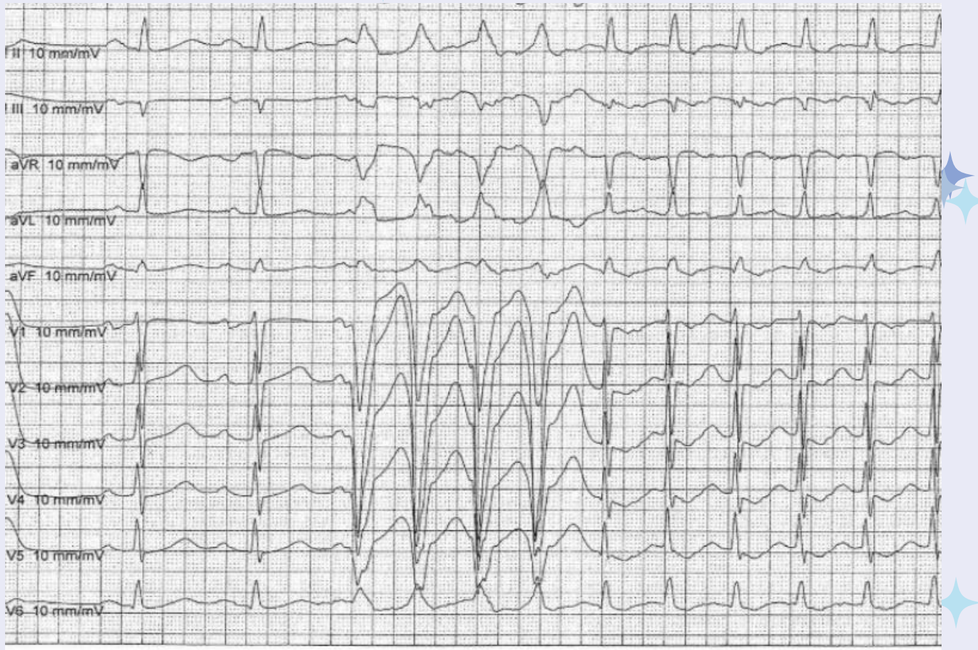

Pleural effusion and passive atelectasis


Hydrostatic pulmonary edema

ECG



Acute inferior ST-segment elevation myocardial infarction (STEMI)



Ventricular and supraventricular tachyarrhythmia

Treatment of Cardiogenic Shock

1- ABCs

2- Identify and treat underlying cause

a. Acute MI

Standard treatment (see MI section) Emergent revascularization with PCI (or CABG) has been shown to improve survival.

b. If cardiac tamponade

pericardiocentesis/surgery

c. valvular abnormalities

Surgical correction

d. Treatment of **arrhythmias**

Treatment of Cardiogenic Shock

3. Preload reduction for patients with **volume overload**: Loop diuretics, dialysis if renal failure, nitroglycerin (though this can worsen hypotension)

4. Inotropy

a. Dobutamine (inotrope) can be used to increase inotropy and simultaneously decrease afterload (SVR), enhancing cardiac output

b. Dopamine or norepinephrine can be used. The SOAP II trial found that for patients with cardiogenic shock, dopamine had higher 28-day mortality compared to norepinephrine.

c. Milrinone is a phosphodiesterase inhibitor which increases inotropy and decreases SVR. It is often used in conjunction with other inotropes.

Treatment of Cardiogenic Shock

5. 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.

6. 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

Treatment of Cardiogenic Shock

7. While controversial, Intra-Aortic Balloon Pump can be used for hemodynamic support Effects include:

a. Decreased afterload

b. Increased cardiac output

c. Decreased myocardial oxygen demand

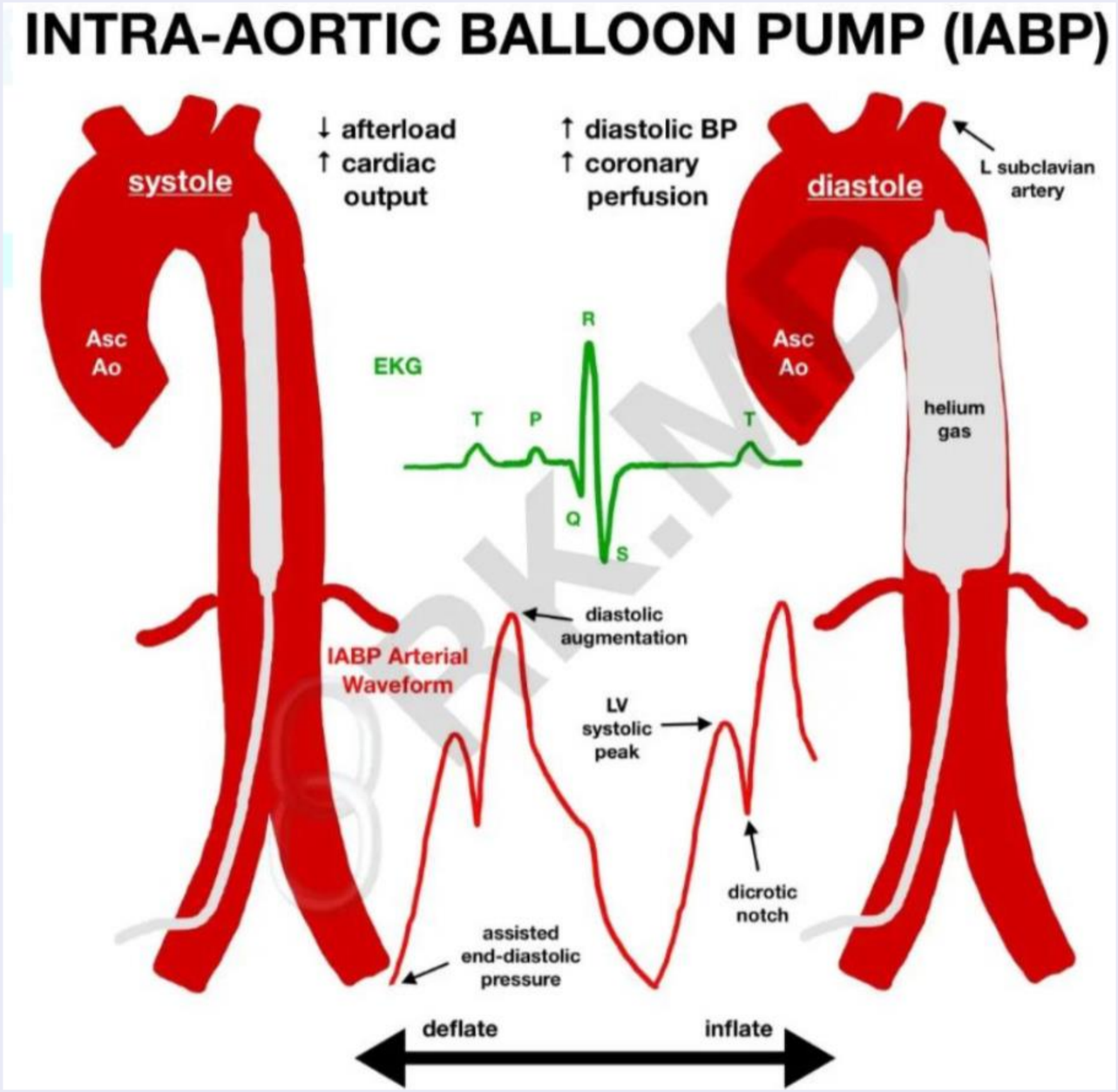
8. More advanced mechanical support devices such as extracorporeal membrane oxygenation and left ventricular assist devices (percutaneously or surgically implanted) can be used for patients with more severe shock.

Intra-aortic balloon pump

IABPs is a device that gives “mechanical support”to a failing heart—it works opposite to the normal pumping action of the heart, that is, it serves to“pump” during diastole and “relax” during systole.

• A balloon catheter is positioned in the descending thoracic aorta just distal to the subclavian artery. It facilitates ventricular emptying by deflating just before the onset of systole (reducing afterload) and increases coronary perfusion by inflating at the onset of diastole (increasing diastolic pressure). The net effect is enhanced myocardial oxygenation and increased cardiac output.

Indications are angina refractory to medical therapy, mechanical complications of MI, cardiogenic shock, low cardiac output states, and as a bridge to surgery in severe AS.


INTRA-AORTIC BALLOON PUMP (IABP)
systole
↓ afterload
↑ cardiac output
↑ diastolic BP
↑ coronary perfusion
diastole
L subclavian artery
Asc Ao
EKG
R
T
P
T
Q
S
Asc Ao
helium gas
diastolic augmentation
IABP Arterial Waveform
LV systolic peak
dicrotic notch
assisted end-diastolic pressure
deflate
inflate

Thank you for your attention

References:

- Step-up to medicine 5th edition
- Amboss medical learning platform