

CARDIOVASCULAR SYSTEM DISORDERS



Chapter contents

Three major contents of the chapter are:-

Cardiac and valve disorders (of heart)

Vascular disorders (of blood vessels)

Hematologic disorders (of blood)

Outlines

- ✓ Arrhythmia/dysrhythmia
- ✓ Angina pectoris/myocardial ischemia
- ✓ Myocardial infarction(MI)
- ✓ Infective endocarditis
- ✓ Pericarditis
- ✓ Rheumatic heart disease
- ✓ Valvular heart disease
- ✓ Pulmonary edema
- ✓ Hypertension
- ✓ Shock (different types)
- ✓ Coronary artery disease (CAD)
- ✓ Phlebitis
- ✓ Deep vein thrombosis (DVT)
- ✓ Varicose vein
- ✓ Blood disorders (different types)

Anatomic & physiologic overview of CVS

- The cardiovascular system consists of:
 - ⊕ The heart
 - ⊕ Blood
 - ⊕ Blood vessels (including arteries, capillaries, and veins).
- ▶ Heart is a hollow, muscular organ located in the center of the thorax (mediastinum) and rests on the diaphragm. About two-thirds of it lies to the left of the median plane
- The heart pumps blood to the tissues, supplying them with oxygen and other nutrients.

Layers of the heart

- ✓ **Endocardium** - lines the inside of the heart and valves.
- ✓ **Myocardium**- the thickest layer, composed of cardiac muscle and performs the work of the heart (responsible for the pumping).
- ✓ **Epicardium** - exterior layer of the heart.
- ✓ **Pericardium**- a double walled sac or membrane (parietal & visceral) encase the heart. Between the parietal and visceral membranes is a space called the **pericardial cavity**.
- **It contains 5 to 30 ml of pericardial fluid** that lubricates the membranes and allows the heart to beat almost without friction.

Four chambers

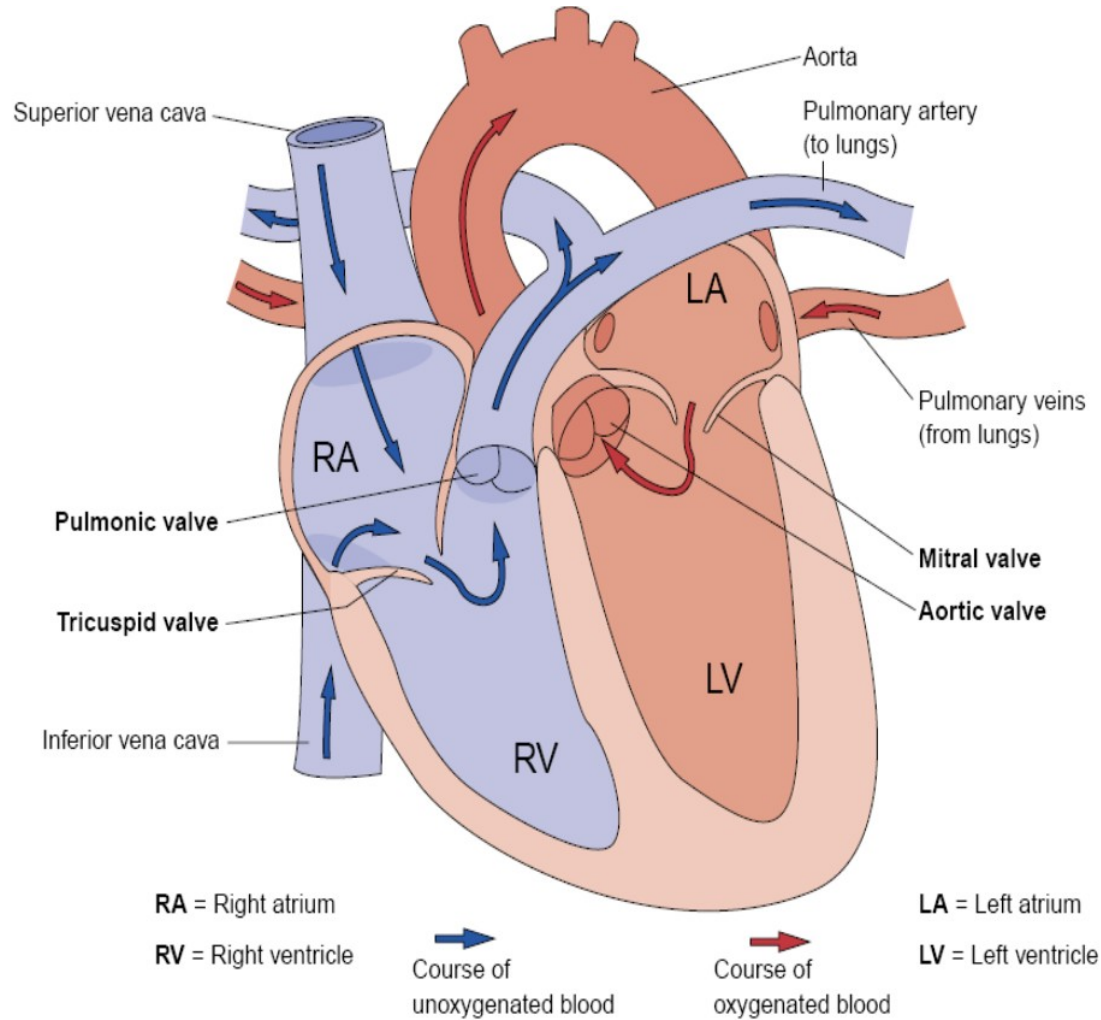
- **Right atria** - receives blood returning from the superior vena cava (head, neck, and upper extremities), inferior vena cava (trunk and lower extremities), and coronary sinus (coronary circulation).
- **Right ventricle** - receive blood from right atrium and pump it to the lungs for oxygenation through pulmonary artery.
- **Left atria** - receives **oxygenated blood** from the pulmonary circulation via four pulmonary veins.
- **Left ventricle** - receive blood from left atrium and pump it to the rest of the body.

Four valves

Definition: Permit blood to flow in only one direction.

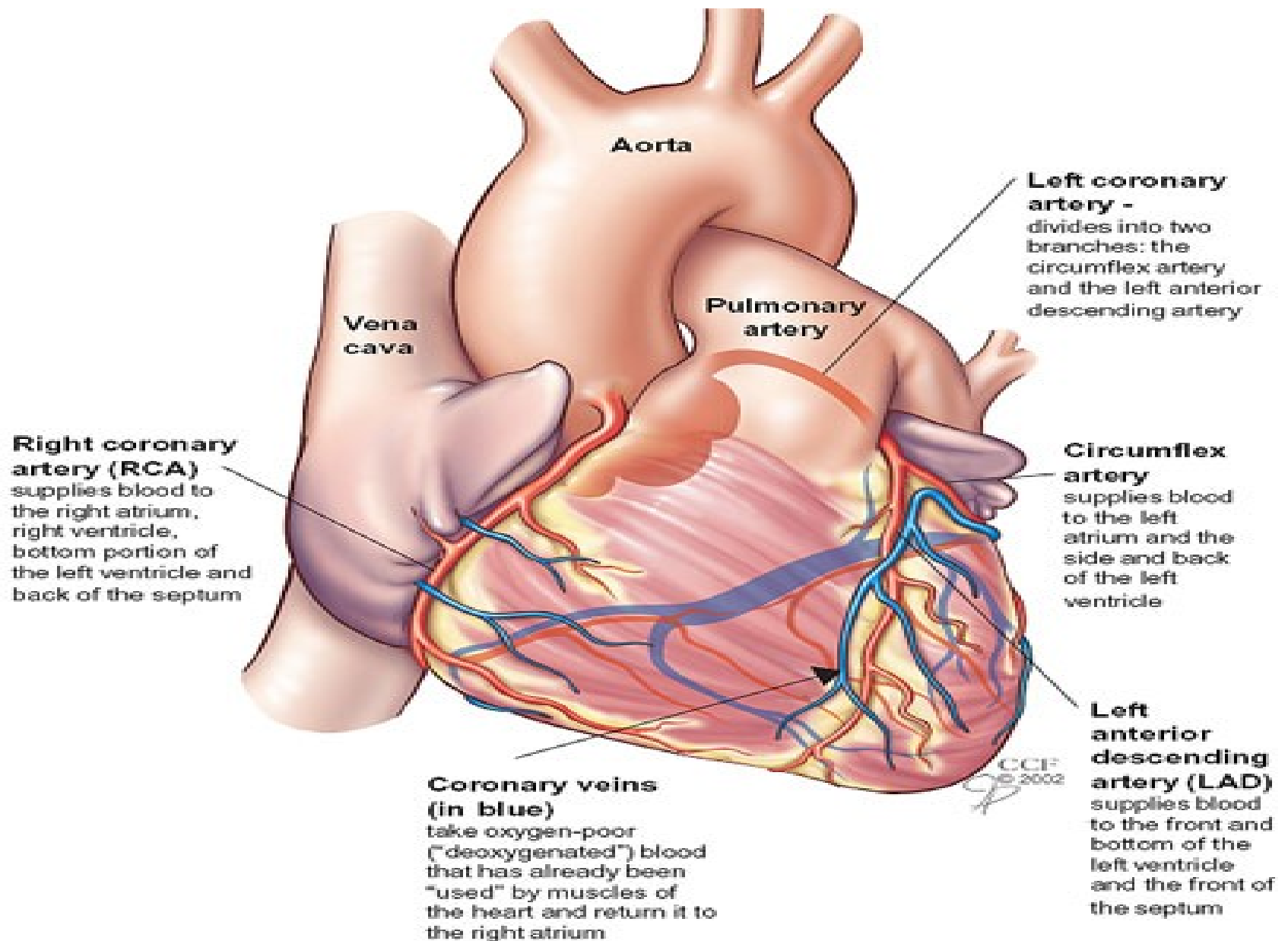
- **Two atrioventricular valves** (separate the atria from the ventricles)
 - ✓ Tricuspid valve (separates the right atrium from the right ventricle).
 - ✓ Mitral or bicuspid valve (Separate left atrium and the left ventricle)
- **Two semilunar valves**
 - ✓ Pulmonic valve (separate right ventricle and the pulmonary artery)
 - ✓ Aortic valve (between left ventricle and the aorta)

Anatomy & physiology of the Heart

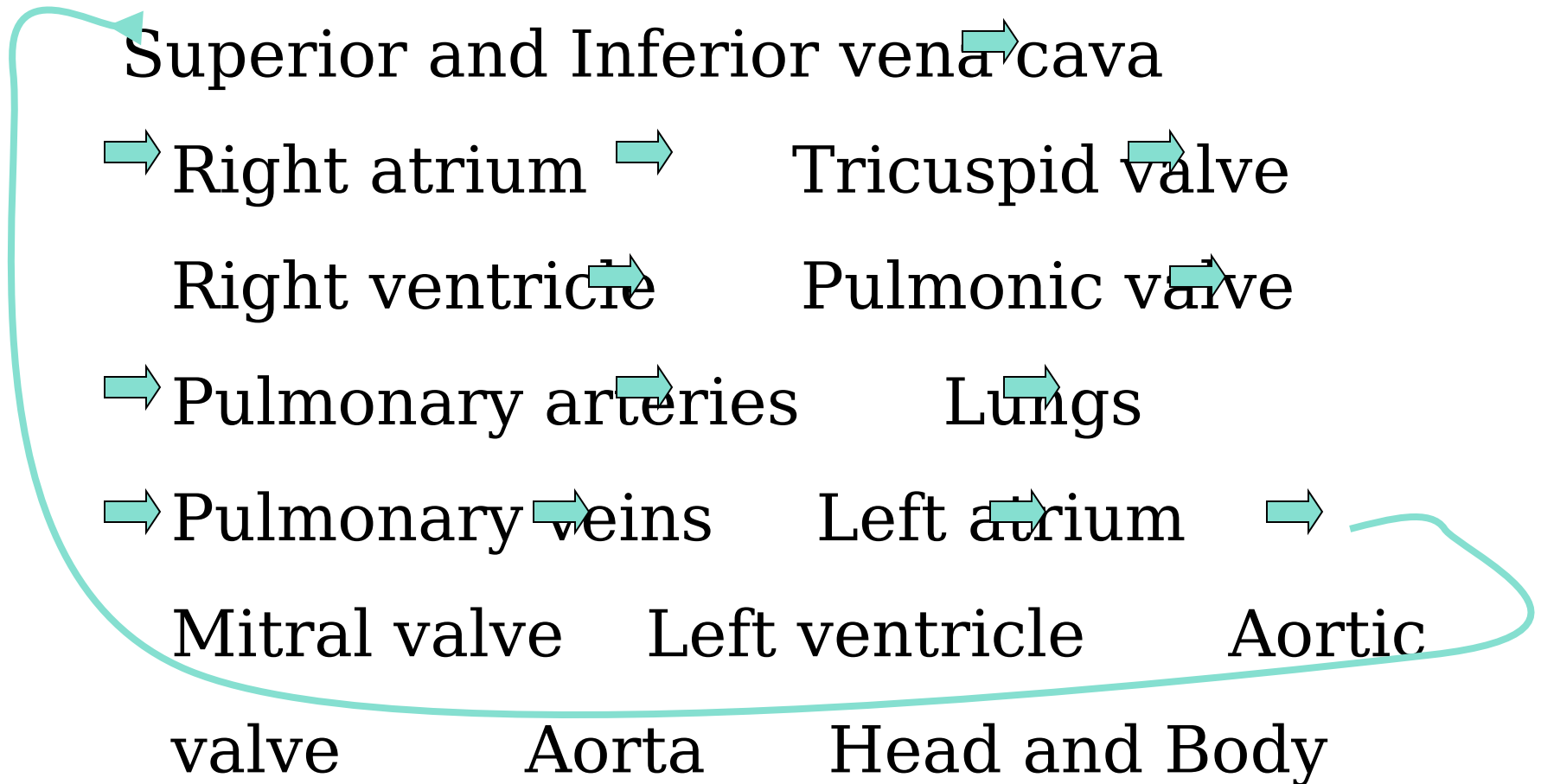


Coronary arteries

- ✓ Supply **arterial blood** to the heart.
- ✓ Heart requires high metabolic rate
- ✓ The right side of the heart is supplied by the right coronary artery, which progresses around to the bottom or inferior wall of the heart.
- ✓ The posterior wall of the heart receives its blood supply from posterior descending artery.



Circulation of the blood in the heart



Circulation

- ▶ **Coronary circulation** – the circulation of blood within the heart.
- ▶ **Pulmonary circulation** – the flow of blood between the heart and lungs.
- ▶ **Systemic circulation** – the flow of blood between the heart and the cells of the body.

Common sign & symptoms of cardiovascular disorders

- ❖ **Chest pain or discomfort** (angina pectoris, MI, valvular heart disease). Angina pectoris is a cardiac pain. It arises in the precordial area and radiates to the left neck, shoulder and left upper arm.
- ❖ **Edema and weight gain** (right ventricular failure, HF) which usually starts from the leg.
- ❖ **Shortness of breath or dyspnea** (MI, left ventricular failure, HF)
- ❖ **Orthopnea**, difficulty in breathing when lying flat or during recumbent position.

- ❖ Some patients have sudden attacks of orthopnea at night, a condition known as **paroxysmal nocturnal dyspnea (PND)**.
- ❖ **Palpitations** - Is a subjective unpleasant perception of one's own heartbeat. Occurs in dysrhythmias resulting from myocardial ischemia , valvular heart disease,
- ❖ **Cough** :Which usually occurs at night(nocturnal).
- ❖ **Fatigue**: Earliest symptom associated with several cardiovascular disorders



❖ **Dizziness and syncope or loss of consciousness:-**

Occurs in postural hypotension, dysrhythmias, cerebrovascular disorders.

❖ ***Symptoms of Arterial occlusion:***

- ✓ **Acute:** pain, loss of function, altered cutaneous sensation, gangrene.
- ✓ **Chronic:** Intermittent claudication (pain around calf muscle on walking) which gets relieved with rest.

❖ ***Symptoms of Venous insufficiency:***

- ✓ Swelling and pain of the affected limb



Assessing cardiovascular functions

Health history

- ❑ Nursing history should focus on the following areas:
 - ✓ Family history of incidence and age of heart disease
 - ✓ High cholesterol levels
 - ✓ High blood pressure, stroke, obesity
 - ✓ Congenital heart disease
 - ✓ Rheumatic fever
 - ✓ Heart murmur, heart attack or heart failure

History.....

- ✓ Present symptoms indicative of heart disease (e.g. Fatigue, dyspnea, orthopnea, edema, cough, pain, palpitations, syncope, hypertension, wheezing, hemoptysis)
- ✓ Presence of diseases that affect heart (e.g. Obesity, diabetes, lung disease, endocrine disorders)
- ✓ Life style habits that are risk factors for cardiac disease (e.g. Smoking, alcohol intake, eating & exercise patterns, areas & degree of stress perceived)

Risk factors for cardiovascular problems

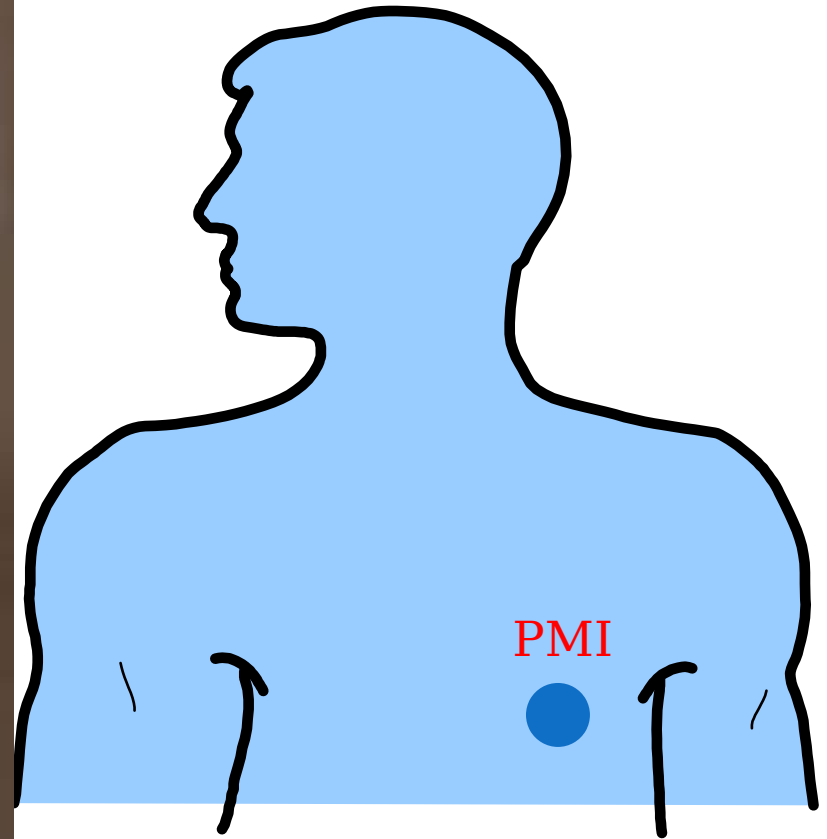
- ✓ Smoking
- ✓ Diabetes mellitus
- ✓ Age older than 60 years
- ✓ Gender (men & postmenopausal women)
- ✓ Family History of cardiovascular disease
- ✓ Family history of high BP & heart attack
- ✓ Over weight & obesity
- ✓ High cholesterol level in the blood and lipidemia
- ✓ Sedentary life style (no exercises)
- ✓ Stress at home or at work

Physical examination

1. Inspection:

- Observe for general appearance and cognition - indicate the heart's ability to propel oxygen to the brain (cerebral perfusion).
- Inspect the carotid arteries carefully for equal appearance.
- Check for pulsations in the jugular veins => tricuspid stenosis.
- Watch for jugular venous distention => a possible sign of right side heart failure

Figure: Funnel chest



Inspect the skin for:

- ❖ **Central Cyanosis**(lips, tongue and buccal mucosa) & **Peripheral Cyanosis** (nails beds, skin of the nose, earlobes, and extremities)
- ❖ **Jaundice:** yellowish discoloration of the skin or sclera due to destruction of red blood cells or congestion of liver
- ❖ **Pallor:** a decrease in the color of the skin—is caused by lack of oxyhemoglobin (due to low hemoglobin).
- ❖ **Petechiae:** Pinpoint hemorrhages into the skin usually around hair follicles.

Inspect the extremities for:

- **Edema:** typically occurs in extremities and sacrum (dependent areas), but may also be found behind scapula or periorbital area.
- **Clubbing of Finger Nails:** the obliteration of the normal angle (160°) between the base of the nail and the skin. Clubbing(concavity)of finger nail means the angle is $> 180^\circ$.
- **Varicose Veins:** Enlarged, dilated and swollen *veins* just beneath the skin.

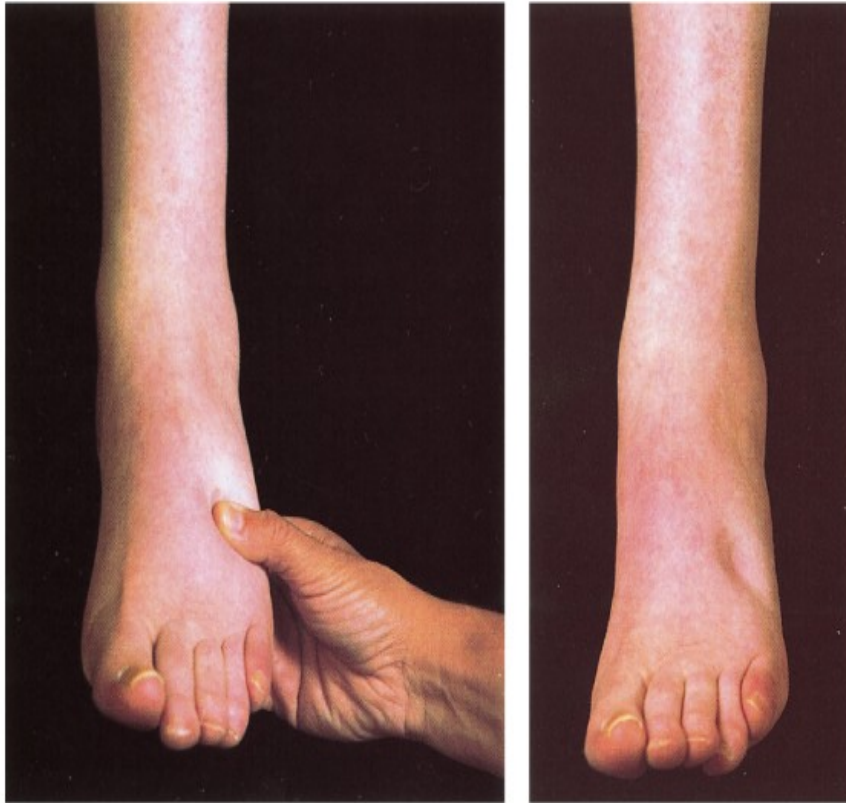


Figure 33-6 3+ pitting edema of the left foot. (Used with permission from Bates B. [1995]. *Bates' guide to physical examination and history taking* [6th ed., p. 438]. Philadelphia: Lippincott Williams & Wilkins)

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3. Palpate the heart for:

- PMI: normally located in the 5th intercostal space just medial to the mid clavicular line.
- **Thrills (a palpable murmur):** a loud murmurs (vibration can be felt with hands) due to turbulent blood flow through valve, or constricted vessels
- **Heart sounds**
- **Parasternal heave** (lifting the palm or pen when put on the parasternal area) - due to hypertrophy

Palpate the extremities

for:

- **Capillary refilling time:** Sluggish return (> 2 seconds) of color up on releasing pressure suggests decreased peripheral circulation.
- **Temperature and moisture:** Moist and calm hand may occur in decreased peripheral circulation. Cool or cold skin may indicate poor cardiac output and tissue perfusion
- **Arterial Pulses:** The arterial pulse should be examined for its rate, rhythm, volume (amplitude), configuration

Take Blood pressure readings

- ❖ From both arms (Lying, sitting & standing)
- Normal adult values ranging from 90/60 to 140/90 mm Hg with average of 120/80 mm of Hg.
- Compare B/P between the right and the left arms, and between the arms and legs, if necessary.
- Normally, there may be a difference of **5-10 mm Hg** on right and left. For follow up purposes, take from the arm with the higher value.
- But if the difference is more than **10-15 mm Hg**, it suggests arterial compression or obstruction on the side with lower pressure.

3. Auscultation

- ❑ Auscultate for normal : S_1 and S_2 **AND**
- ❑ Extra or additional heart sounds : S_3 and S_4 and murmur.

Coronary Artery Disease (CAD)

- ▶ Coronary arteries supply blood to the heart muscle.
- ▶ Our heart muscle needs oxygen-rich blood to function.
- ▶ Small branches dive into the heart muscle to bring it blood.

Coronary Artery Disease (CAD)

- A **narrowing** of the coronary arteries that prevents adequate blood supply to the heart muscle.
- Usually caused by **atherosclerosis**, it may progress to the point where the heart muscle is damaged due to **lack of blood supply**. Such damage may result in infarction, arrhythmias, and heart failure.
- Coronary heart disease (CHD) is the leading cause of death
- Coronary Artery Disease (CAD) more common in men
- MI in young, healthy women is rare

Cause of CAD

1. Atherosclerosis :- Is an abnormal **accumulation of lipid**, or fatty, substances and fibrous tissue creating blockages or narrowing of the vessel.

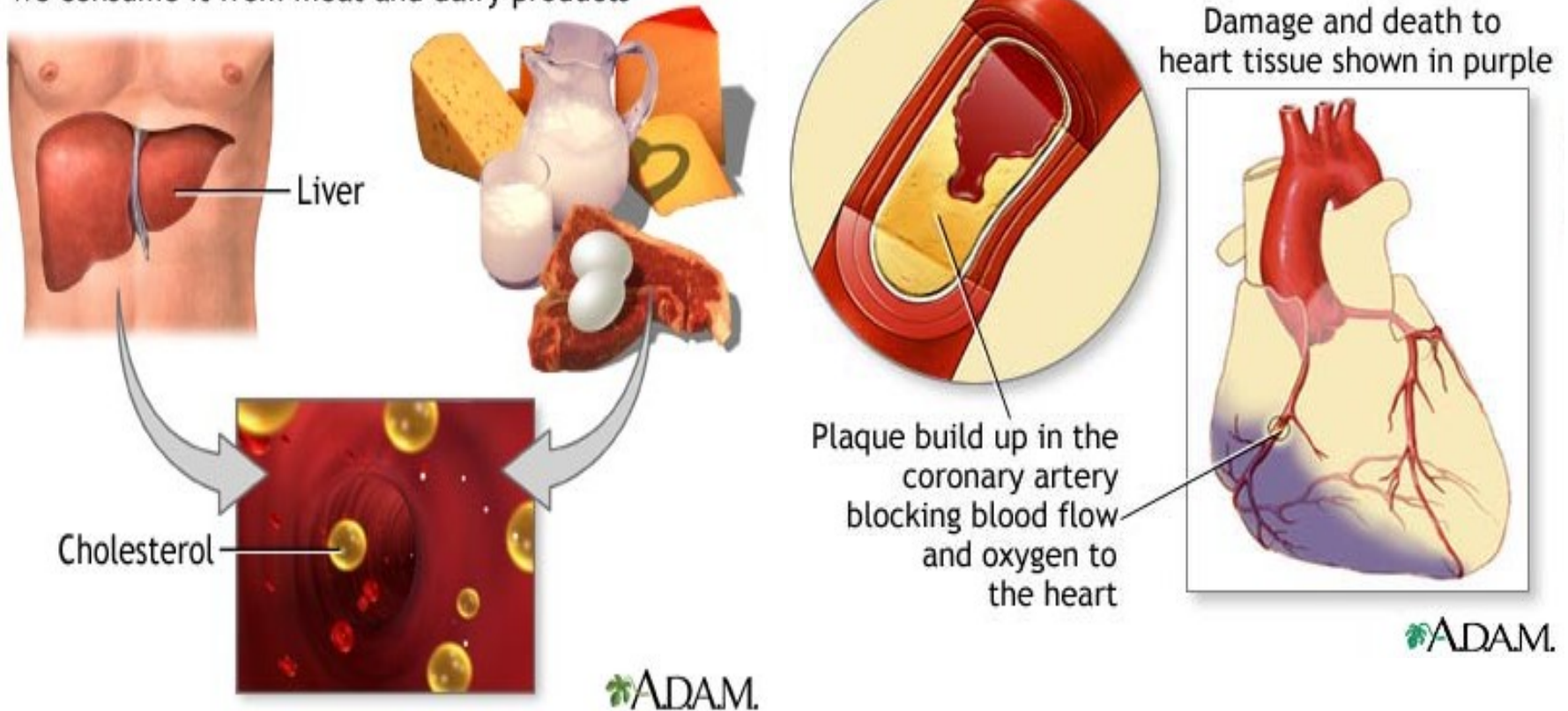
- ✓ These accumulations are referred to as atheromas or plaques
- ✓ The **Atheromas** (plaque) protrude into the lumen of the vessel, narrowing it and obstructing blood flow.

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- Presence of atheroma (Plaques consist of lipids, cells, fibrin, cell debris)
 - ✓ These substances **block and narrow** the coronary vessels in a way that reduces blood flow to the myocardium leading to **myocardial infarction or sudden death.**

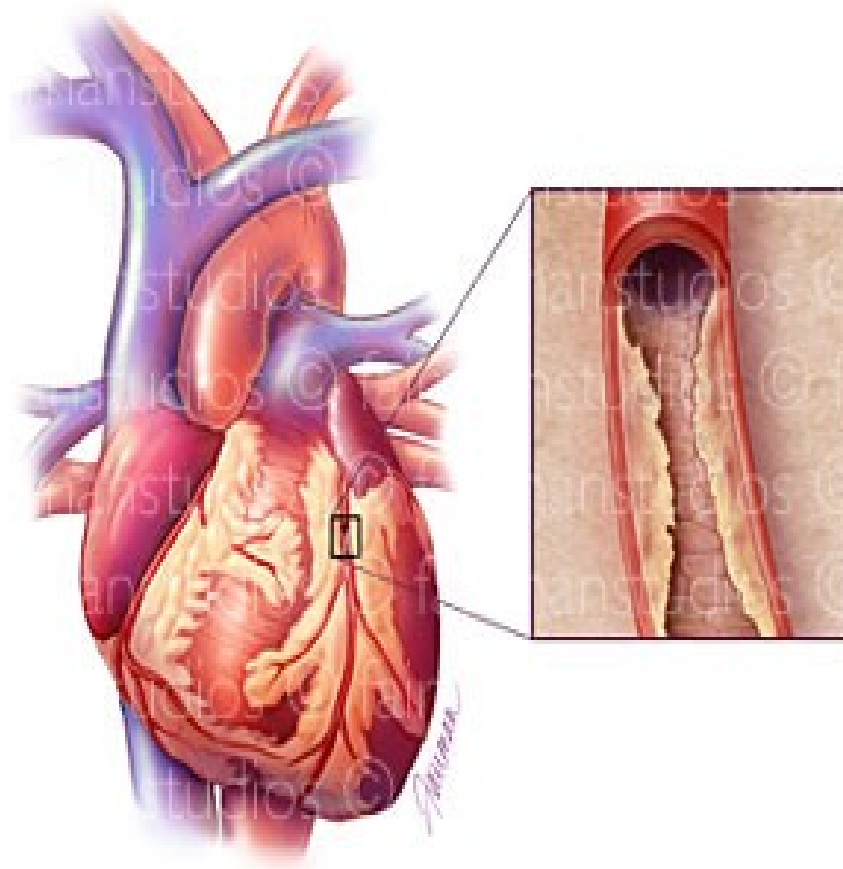
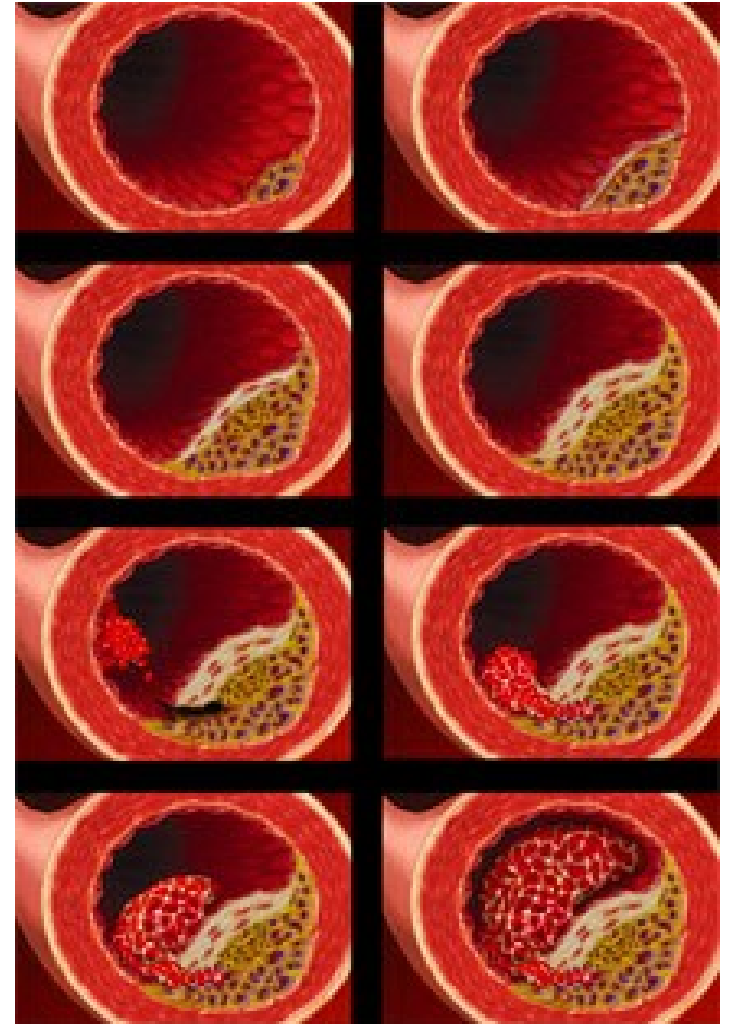


PP of CAD

Cholesterol is produced by the liver and we consume it from meat and dairy products



PP of CAD ...



2. Arteriosclerosis:- Is thickening of the walls of the arterioles, **with loss of elasticity and contractility**

- Loss of elasticity, walls thick and hard, lumen narrows (the term means “**hardening of the arteries.**”)
- It is a diffuse process whereby the muscle fibers and the endothelial lining of the walls of small arteries and arterioles become **thickened.**



Arteriosclerosis

Risk Factors for CAD

Modifiable risk factors:

- Increasing cholesterol levels or increased LDL (bad cholesterol)
- Cigarette smoking
- Hypertension
- Obesity, diabetes mellitus
- Sedentary life style

Non-modifiable risk factors:

- ❑ Increasing age - Your risk for CHD goes up the older you get.
- ❑ Gender:- Male > Female s, because female have hormone estrogen that increase high lipoprotein when increase in HDL it decrease action of LDP or liver become less faction become it beak down and deposit it
- ❑ Family history:- You are more likely to develop the condition if someone in your family has had a history of heart disease -- especially if they had it before age 50.

Clinical manifestations

- ✓ May be asymptomatic until a major portion of the lumen of a blood vessel is occluded.
- ✓ Angina /chest pain or dizziness caused by decreased blood supply and oxygen to the heart (most common)
- ✓ Shortness of breath
- ✓ Nausea, vomiting
- ✓ Diaphoresis
- ✓ Weakness and fatigue
- ✓ Cool, clammy skin
- ✓ ECG changes

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- ⊕ Peripheral pulses may be diminished or absent.
- ⊕ Pallor in the nail beds due to reduced blood flow in the extremities
- ⊕ Skin temperature in the extremities is cooler, and there is prolonged capillary refill (greater than 3 seconds).
- ⊕ A carotid bruit (abnormal sound on auscultation) indicates atherosclerosis in the carotid artery.
- ⊕ Sometimes symptoms occur only with exertion. In some people, symptoms occur at rest.

Diagnostic Tests

- ◆ Elevation of **cholesterol and triglycerides** (total cholesterol levels above 200 mg/Dl)
- ◆ Low-density lipoproteins (**LDLs**) increase, at risks for CAD
- ◆ Radiological studies of the arteries can be performed to show narrowed or occluded vessels.

Eg. **Coronary angiography/arteriography** -- an invasive procedure designed to evaluate the heart arteries for narrowing or occlusion under x-ray-----most accurate.

- ◆ Exercise stress test
- ◆ Electrocardiogram (ECG)

Treatment

➤ Lifestyle changes

▶ Dietary measures

- Low fat diet because the formation of plaque within arteries is primarily caused by fatty deposits
- Low cholesterol

- ◆ Increased physical activity - Increased activity raises HDL levels (the desirable cholesterol).
- ◆ Increasing physical activity may also lower insulin resistance and facilitate weight loss.

- ◆ Cessation of tobacco - the risk of developing CAD is two to six times higher in cigarette smokers than in non-smokers.
- Smoking contributes to a loss of high-density lipoproteins (HDLs).
- The rate of progressive damage to blood vessels is increased with smoking.
- Smoking also causes vasoconstriction, which leads to **angina pectoris** and cardiac dysrhythmias.
- ◆ Managing hypertension / decrease sodium ion intake
- ◆ Stress reduction

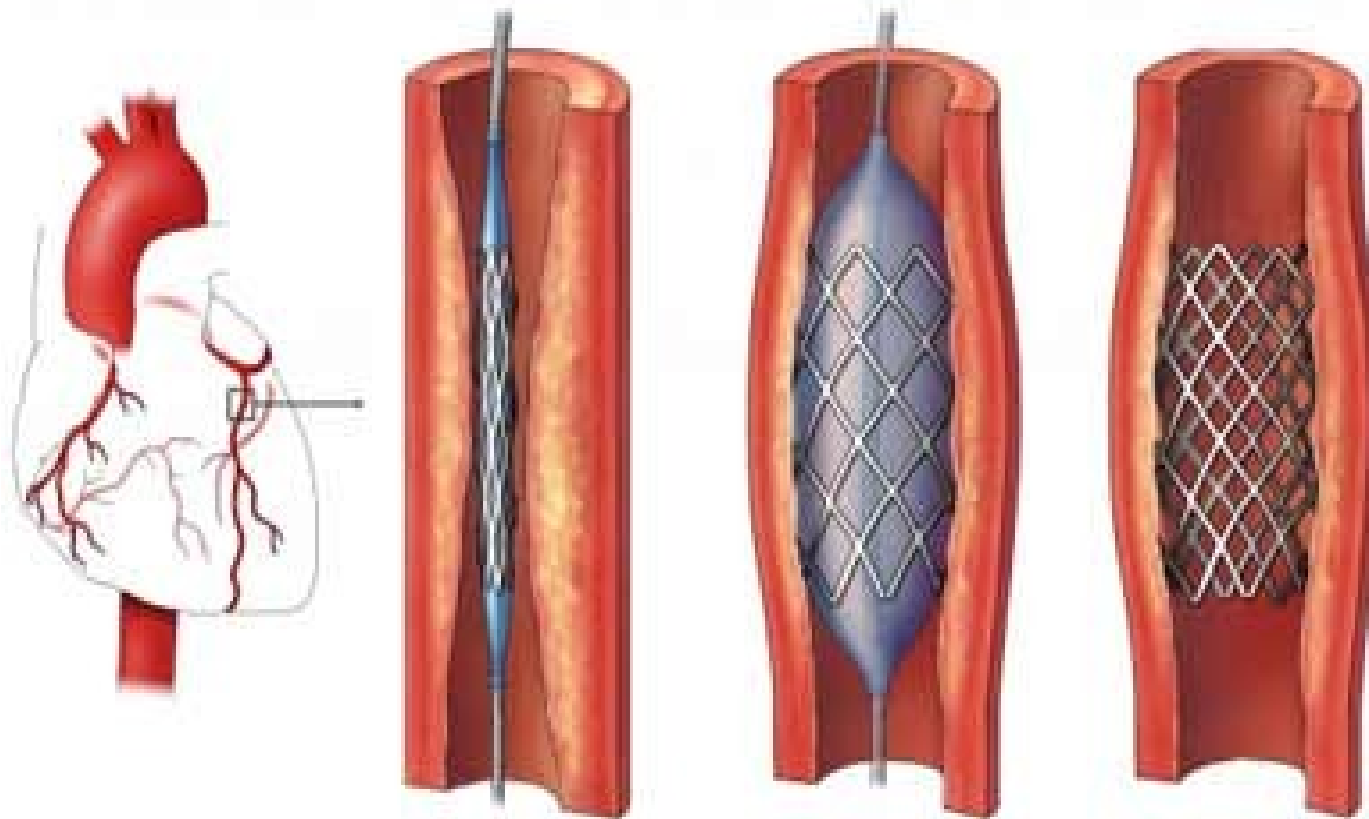
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- **Medication:** When dietary control is not effective, medication is also used.
 - ✓ Lipid lowering agents - High doses of **statins**, which lower lipid levels. It may take 4 to 6 weeks before lipid levels respond to drug therapy.
 - ✓ Nitrates & Nitroglycerine
 - ✓ Antiplatelet / Oral anticoagulant
 - ✓ Beta blockers
 - ✓ Calcium channel blockers
 - ✓ Diuretics

➤ **Surgical interventions**

- ✓ **Stent insertion**
- ✓ **Percutaneous transluminal coronary angioplasty (PTCA)** - is procedure in which a long tube with a small balloon is passed through blood vessels into the narrowed artery. The balloon is inflated, causing the artery to expand
- ✓ **Coronary artery bypass grafting (CABG)**- is a surgical procedure in which an artery from an arm or the chest is removed and graphed to aorta, bypassing the blockage connected to the diseased coronary artery at a point distal to the region of atherosclerotic plaque

Stent insertion



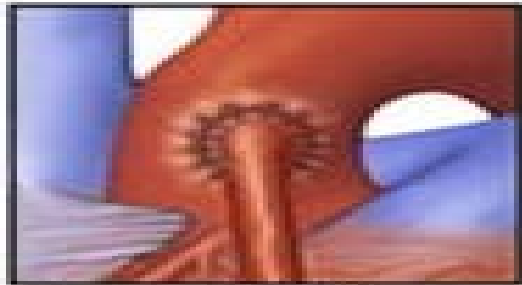
A splint is left
inside the
lumen

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CVD,2023

Coronary Artery Bypass Graft

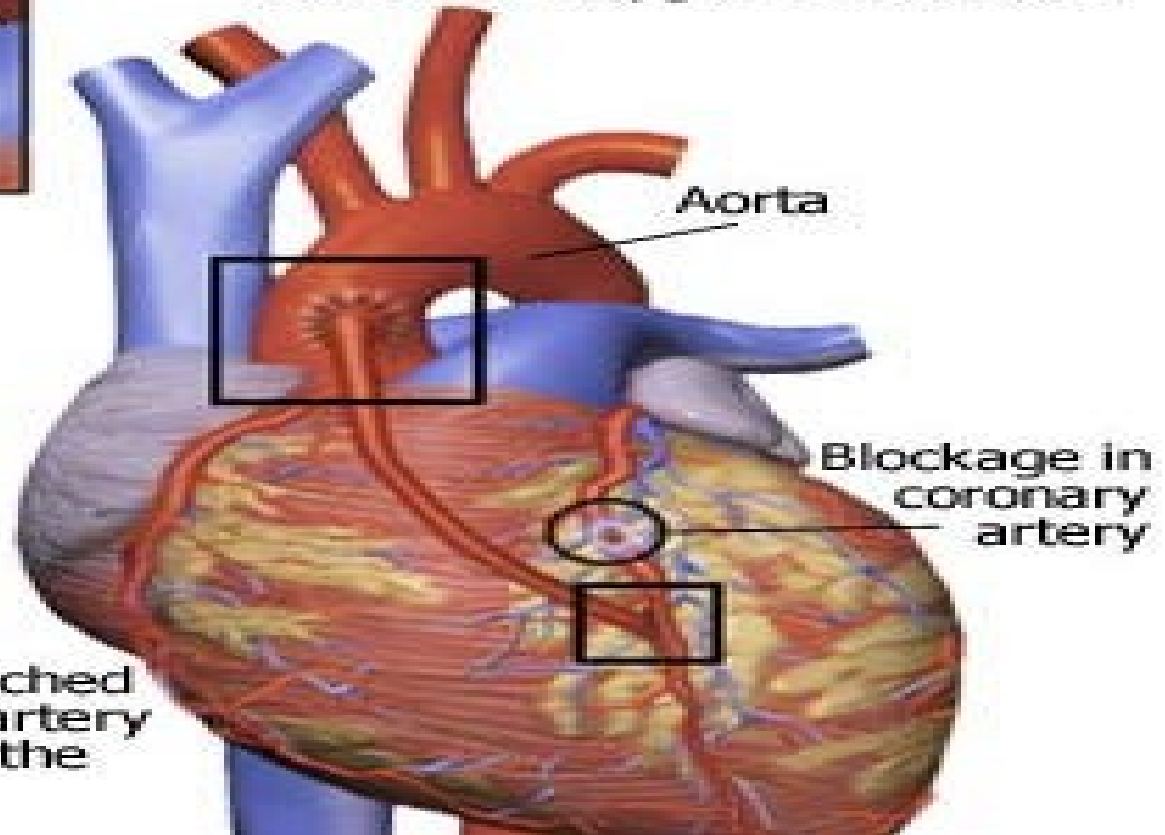
A procedure to bypass a blocked section of a coronary artery and to deliver oxygen to the heart



One end of the blood vessel is attached to the aorta



Other end is attached to the coronary artery at a point below the blockage



Prevention

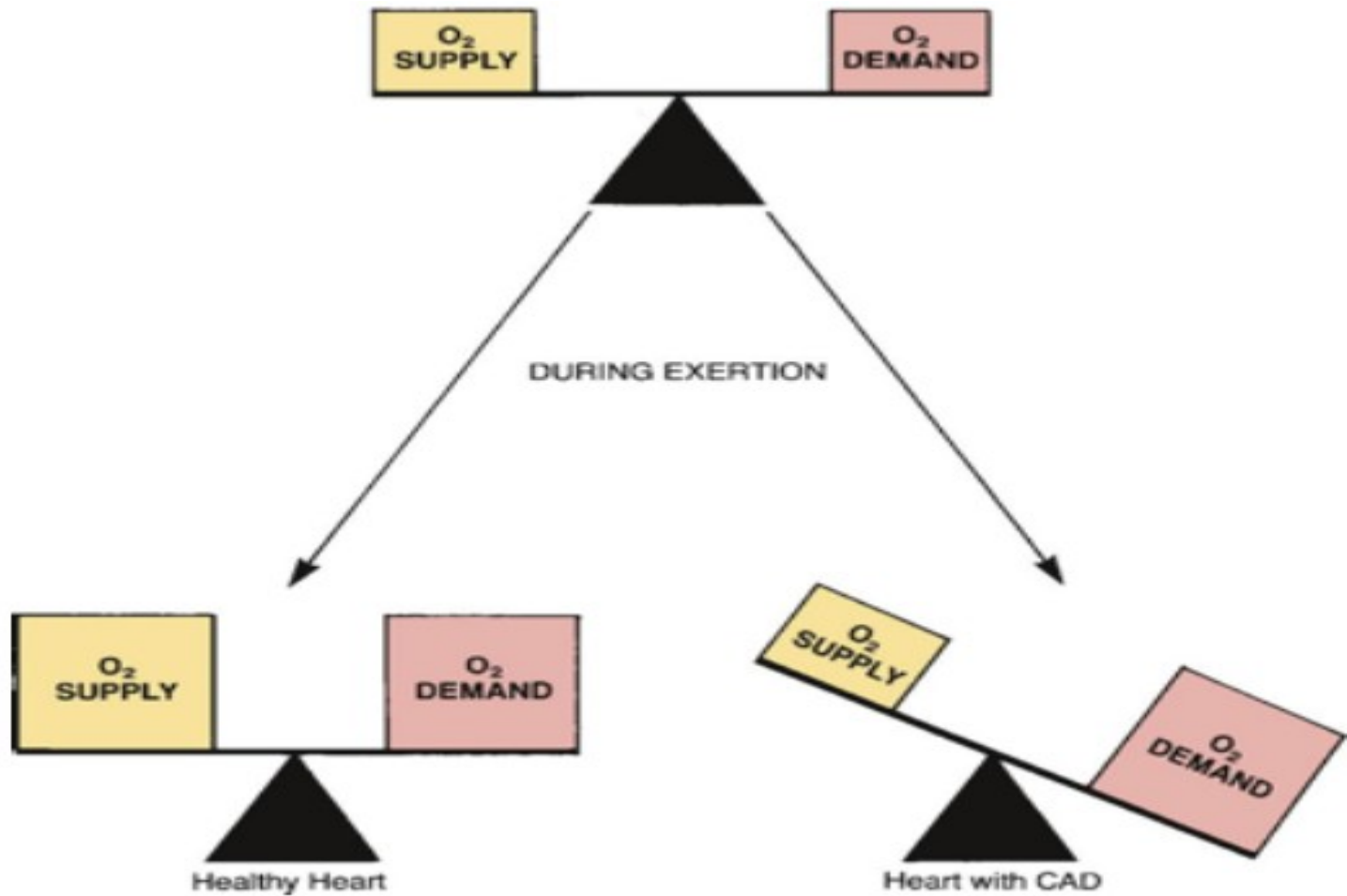
- ✓ Avoid or reduce stress as best as you can.
- ✓ Don't smoke.
- ✓ Eat well-balanced meals that are low in fat and cholesterol and include several daily servings of fruits and vegetables.
- ✓ Get regular exercise. If your weight is considered normal, get at least 30 minutes of exercise every day. If you are overweight or obese, you should get 60 - 90 minutes of exercise every day.
- ✓ Keep your blood pressure below 130/80 mmHg if you have diabetes or chronic kidney disease, and below 140/90 otherwise
- ✓ Keep your cholesterol and blood sugar under control.

Angina pectoris

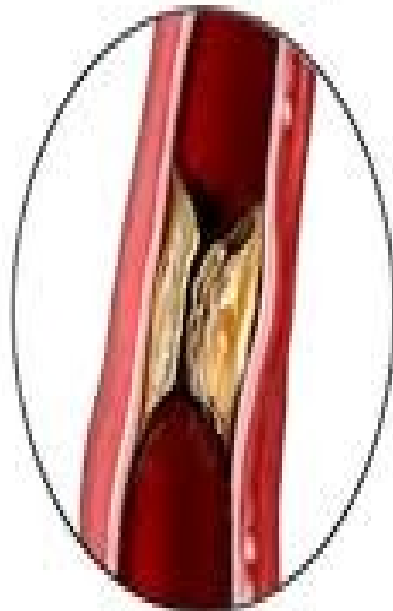
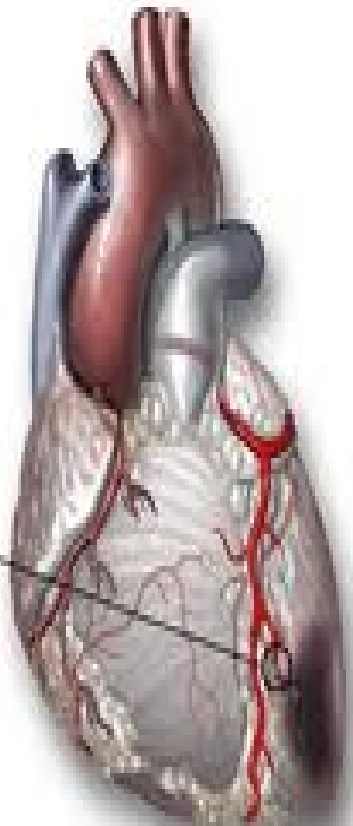
- Angina pectoris is a **clinical syndrome** usually characterized by episodes of **pain or pressure** in the anterior chest .
- Resulting from **narrowing** of blood vessels to the **coronary artery**.
- The cause is usually insufficient coronary blood flow which results in a **decreased oxygen supply** to meet an increased myocardial **demand for oxygen** in response to physical

Causes:

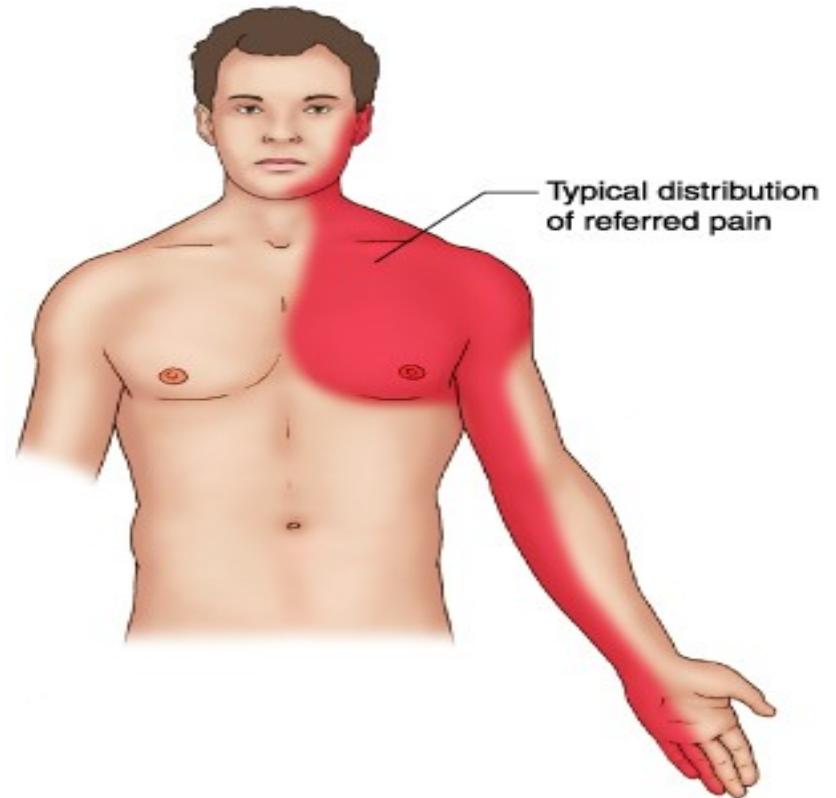
- ❑ Imbalance between the supply (perfusion) and demand of the heart for oxygenated blood.
- ❑ Reduced blood flow due to obstructive atherosclerotic lesions in the coronary arteries.
- ❑ The obstructive atherosclerotic lesions often cause lumen narrowing.
- ❑ Coronary lumen narrowing causes ischemic cardiac pain/angina pectoris when the demand for oxygen and nutrients increases.
- ❑ When the **demand** for oxygen by the heart muscle exceeds the available **supply**, **chest pain occurs**.



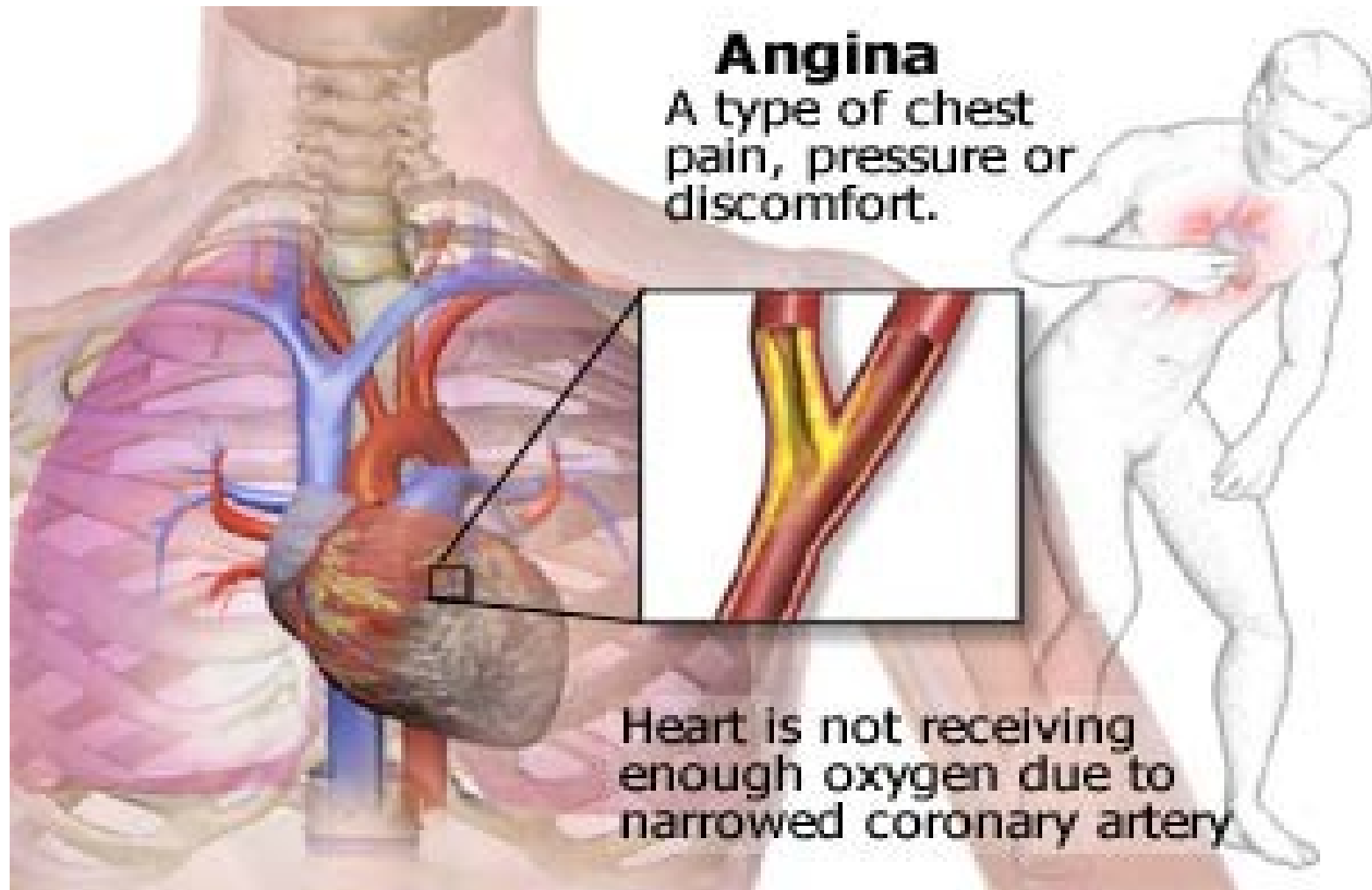
Blocked Lumen in Branch
of Left Coronary Artery



Anterior infarct



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Factors associated with typical angina pain

1. Physical exertion (increase myocardial O₂ demand).
2. Exposure to cold (cause elevation of BP increase oxygen demand)
3. Eating heavy meals
4. Stress or any emotion-provoking situation

NB: An episode of angina is typically precipitated by physical activity, excitement, or emotional stress.

Type of angina

A. Stable angina: predictable & consistent pain that occurs on exertion and relieved by rest.

- ✓ Also called “**Effort Angina**”
- ✓ Discomfort is **precipitated by activity**
- ✓ Minimal or no symptoms at rest
- ✓ Symptoms disappear after rest OR
- ✓ Cessation of activity



B. Unstable angina or crescendo angina:

Is a medical emergency, poses a much greater risk of death than stable angina, but a smaller risk of death than MI.

- ✓ Symptoms occur more frequently and stable angina.
- ✓ **Pain occurs at rest**; is of new onset; is of intensity **(Crescendo)**, force, or duration;
- ✓ **Isn't relieved by rest**; and is slow to subside in response to **nitroglycerin**



C. Variant angina or Prinzmetal's angina

Cause: Brief Vasospasm of coronary Vessels

- ✓ Hence, as in stable angina, pain is secondary to insufficient oxygenation of the heart.
- ✓ Can produce pain at any time, even during **rest and sleep**.

D. Silent ischemia

- ✓ **Objective evidence of ischemia** (as ECG changes with a test), but patient reports no symptom.
- ✓ Not all ischemic events are perceived by patients

Clinical manifestations

- ✓ May produce pain lasting 3 to 5 minutes vary in severity from feeling of indigestion to chocking in retrosternal area , radiate to neck , jaw shoulders , inner aspects of upper arms:- **angina pain is not always felt in the chest.**
- ✓ **Leviene's Sign** (Patients clench their fist over their sternum)
- ✓ **Restrosternal** discomfort that may radiate down both arms but usually the left sometimes radiates to mandible maxilla, teeth

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- ✓ Feeling of weakness or numbness in the arms , wrists and the hands
- ✓ Shortness of breath:- increased heart rate, increases respiratory rate and increases oxygenation
- ✓ Pallor, Diaphoresis due to increased work of body to meet basic physiologic needs; anxiety.
- ✓ Dizziness or lightheadedness
- ✓ Tachycardia—heart pumping faster trying to meet oxygen needs as anxiety increases.
- ✓ **Anxiety—not** getting enough oxygen to heart muscle, the patient becomes nervous.

Diagnostic findings

- ❑ **History** : Often made by evaluating the clinical manifestations of ischemia and the patients history
- ❑ 12-Lead ECG helps in making diagnosis
 - ✓ **T-wave inverted** with initial ischemia, which is reduced blood flow due to an obstructed vessel, usually first sign.
 - ✓ ST-segment changes occur with injury to the myocardium (heart muscle).
- ❑ **Coronary arteriography:**
 - ✓ To determine plaque build-up in coronary arteries.
- ❑ **Chest x-ray** :
 - ✓ To determine signs of heart failure.

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❑ **Cardiac enzymes:**

- ✓ Are cellular proteins released in to the blood as a result of cardiac cell membrane injury.
- ✓ **Troponins, C- reactive kinase (CK)MB**, which is an enzyme released by damaged cardiac tissue 2 -6 hours following an infarction

There are three isoenzymes of CK

1. Ck-MM (skeletal muscle)
2. Ck-MB (myocardial muscle)
3. Ck-BB (Brain)

- ❑ **Elevated Ck-MB** indicates myocardial damage

Management

❑ **Objective:**

- ✓ Decrease oxygen demand of myocardium and to increase oxygen supply to meet its need
- ✓ Relief of symptoms
- ✓ Slowing progression of the disease
- ✓ Reduction of future events like myocardial infarction

❑ **Methods:** non pharmacological, pharmacological & surgical therapy

Non pharmacological Therapy of Angina

- ❖ Patients should attempt to avoid factors that can precipitate angina.
- ❖ **These include:**
 - ✓ Overexertion
 - ✓ Heavy meals
 - ✓ Emotional stress
 - ✓ Exposure to cold
 - ✓ Smoking, obesity
 - ✓ Hypertension
 - ✓ Hyperlipidemia and
 - ✓ Sedentary lifestyle

Pharmacological therapy of angina

Nitroglycerin :

- Oldest and most frequently used antianginal drugs
- Prodrugs that are **sources of nitric oxide (NO)**
- They release NO and helps **dilate or relax** coronary arteries and get more oxygen to heart muscle.
 - ↓ Myocardial work load (↓ se in O₂ demand).
 - Improve perfusion of ischemic myocardium.
 - Inhibition of platelet aggregation
- **Adverse effects:** - mainly due to excessive vasodilatation.

Eg:- Headache, Flushing, Orthostatic hypotension, Syncope

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Beta-adrenergic blocking agents :
propranolol ,metoprolol , atenolol

- Appear to reduce myocardial oxygen consumption, result in a reduction in **heart rate, decreased BP** , and reduced myocardial contractility.
- They **decrease cardiac workload** (-ve inotropic and chronotropic effects)
- Should be avoided by patients with **asthma**. If an asthmatic individual absolutely must use a **beta₁ -selective agent** (eg, metoprolol) should be chosen
- This is because ; blockade of beta₂ receptors in the lung can promote **bronchoconstriction**

Calcium channel blocking agents : Verapamil and diltiazem

- ✓ Drugs that prevent calcium ions from entering cells($\downarrow$ Ca^{2+} influx)
- ✓ Some decrease sinoatrial node **automaticity** and node conduction ,resulting in slower heart rate
- ✓ Peripheral **vasodilatation**
 - ▶ CCBs act selectively on peripheral arterioles and arteries and arterioles of the heart
 - ▶ The result is arteriolar dilation and reduction of peripheral resistance (**afterload**)

Antiplatelet and anticoagulant medications:

- Decrease platelet aggregation and thereby decrease the risk of **thrombus** formation in coronary arteries
- **Aspirin**: prevents platelet activation and reduces the incidence of MI and death with CAD
 - Patients with stable angina are recommended to take 75 to 162 mg of aspirin daily, unless there is a specific reason not to.
- **Heparin** : prevents the formation of new blood clots

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Oxygen administration :

- ✓ Usually initiated at the onset of chest pain to increase the amount of oxygen delivered to the myocardium and to decrease the pain

Analgesic:

- ✓ Typically morphine intravenously during acute pain

Nursing interventions

- ✓ **Treating angina** : when patient experiences angina the nurse should direct patient to stop activities and sit or rest in bed in semi-fowler position
- ✓ **Reducing anxiety** : The nurse should explore and implicate that the diagnosis has for the patient providing information about the illness, treatment and methods of preventing its progression
- ✓ **Preventing pain** : when the patient has pain with minimal activity, the nurse alternates the patients activities with **rest** periods

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- ✓ Monitor vital signs—look for change in BP, PR; irregular pulse; pulse deficit
- ✓ Notify physician if systolic blood pressure is less than 90 mmHg & heart rate is less than 60 beats per minute.
- ✓ You may have an order to hold **nitrates** if SBP < 90 mmHg to reduce risk of patient passing out from lack of blood flow to brain. Nitrates dilate arteries to the heart and increase blood flow.
- ✓ You may have an order to hold **beta blockers** if heart rate goes below 60; you should continuously monitor the patient's pulse rate.

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- ✓ Assess chest pain each time the patient reports and describe angina pectoris as **PQRST**:-
 - Place, (chest, shoulder, back...)
 - Quality (describe as—stabbing, squeezing, etc.),
 - Radiation (does the pain travel anywhere else?),
 - Severity (on a scale of 1 to 10), and
 - Timing (when it started and how long it lasts and what preceded the pain).

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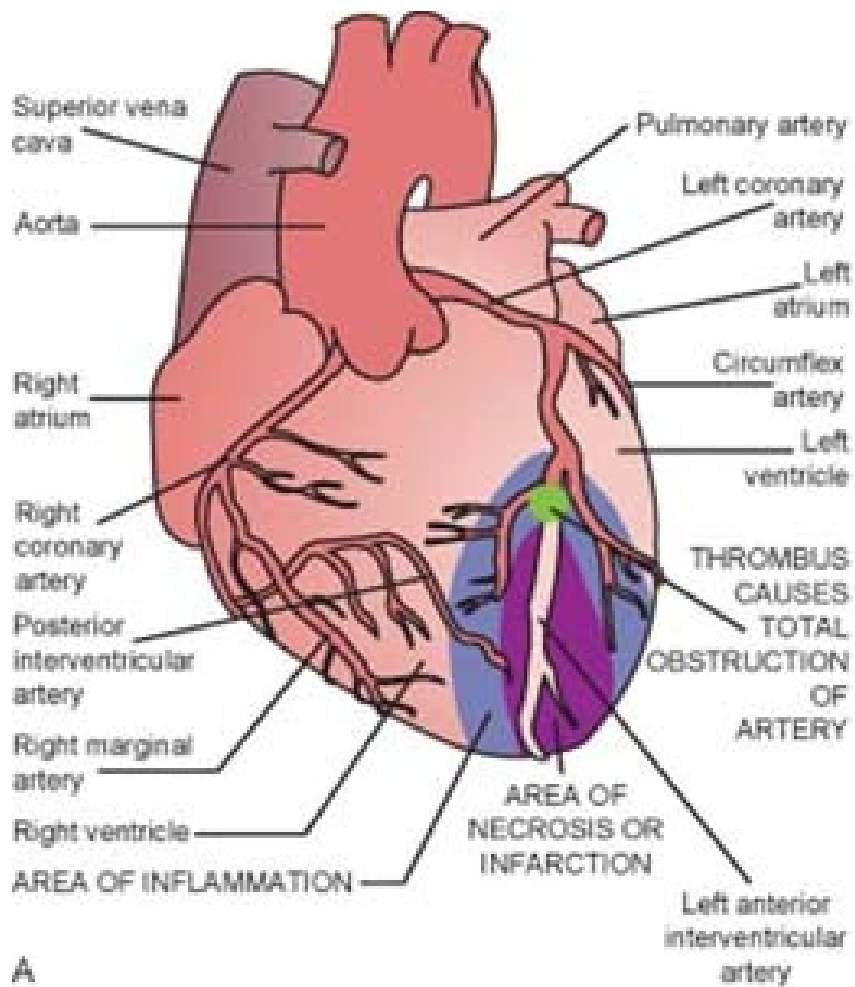
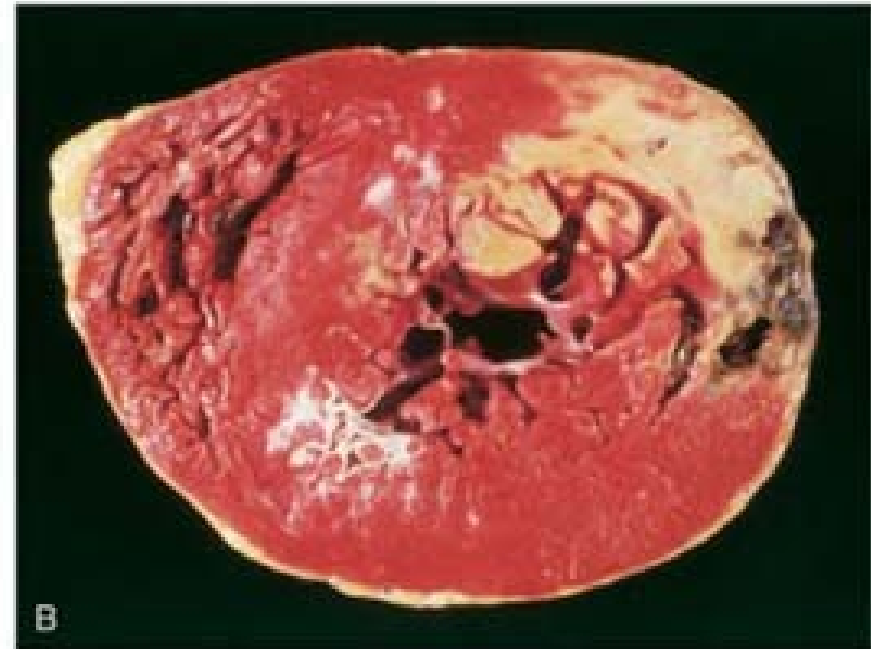
- ✓ Monitor cardiac status using a 12-lead electrocardiogram (EKG)
- ✓ Record fluid intake and output & assess for renal function.
- ✓ Explain to patient: Rest when pain begins to decrease oxygen demands.
- ✓ Avoid stress and activities that bring on an angina attack.
- ✓ Stop smoking! Smoking is associated with heart disease.
- ✓ Slowly increase exercise to build up activity tolerance & possibly exercise with cardiac rehabilitation.

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- ✓ Adhere to the prescribed diet and exercise plan.
- Lower cholesterol and fat intake to decrease further plaque build-up, and decrease excess salt intake to help BP control.
- Diet (small, frequent, easily digested meals, no caffeine)
- ✓ To prevent myocardial ischemia from progressing to infarction the nurse teaches precipitating physiological factors such as activity during cold weather, during heavy meal, or with sexual intercourse and psychological factors such as anger or grief.

Myocardial infarction (MI)

- It is the **interruption** of blood supply to part of the heart, causing some heart cells to **die**.
- MI, also known as “**heart attack**”.
- Death of cardiac muscle due to prolonged severe ischemia/interrupted blood supply
- MI occurs when blood supply to the myocardium is interrupted for a prolonged time due to the blockage of coronary arteries or by complete blockage of one of the coronary arteries
- This results in insufficient blood/oxygen reaching cardiac muscle, causing cardiac muscles/cells become **ischemic, die, and form an infarct**



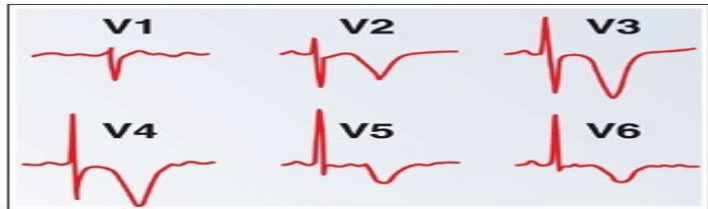
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- MI may result in sudden death, or the infarct undergoes a healing process and is replaced with **connective tissue**
- Patients are typically (not always) symptomatic, but some patients will not be aware of the event; they will have what is called a ***silent MI***.
- After an MI the heart may be weakened and develop **congestive failure** or **cardiac arrhythmias**
- Frequency rises progressively with **increasing age** in presence of risk factors to atherosclerosis.
- **Men** are at significantly greater risk than women.

Clinical features

- Maybe asymptomatic, known as a **silent MI**, which is more common in diabetic patients
- **Chest pain** that is unrelieved by rest or nitroglycerin and radiates to arms, jaw, back and/or neck
- Dyspnea/**shortness of breath**, especially in the elderly or women
- **Heart rate >100 (tachycardia)** rapid and weak pulse (low CO) because of sympathetic stimulation
- Profuse sweating (diaphoresis), cool, pale, moist skin
- Nausea, vomiting, Hypotension/shock
- Anxious, restless & **Sudden death due to arrhythmia**



Electrocardiographic abnormalities

Increased total
CK, CK-MB, and
cardiac troponins

Nausea and
vomiting

Cyanosis

Sweating

Dyspnea

Steady, crushing
substernal chest
pain—may radiate
to left jaw, shoulder
or arm

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Complications

Vascular Complications

- ✓ Recurrent ischemia & infarction

Mechanical Complications

- ✓ Left ventricular wall & septal rupture

Myocardial Complications

- ✓ Diastolic & Systolic dysfunction
- ✓ Congestive heart failure
- ✓ Hypotension/cardiogenic shock

Electrical Complications

- ✓ Arrhythmias of different types (25% patients sudden death after MI)

Assessment And Diagnostic Findings

- History (clinical features)
- ECG
 - ✓ T-wave inversion—sign of ischemia.
 - ✓ ST-segment elevated or depressed—sign of injury.
 - ✓ Significant Q-waves—sign of infarction.
- Echocardiogram may show cardiac damage
- Less than 25 ml/hr of urine output due to lack of renal blood flow (blood urea nitrogen increases)
- Complete blood count (WBC increases due to inflammatory process)
- Cholesterol levels & lipid profile (they will be raised)

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- Measuring the blood levels of proteins that leak out of fatally injured myocytes
- **Elevated creatine kinase MB (CK-MB)**—usually done serially, the numbers will rise along a predetermined curve to signify myocardial damage and resolution.
- **Elevated troponin I- and troponin T-proteins** elevated within one hour of myocardial damage.
- The **myoglobin** level can elevate within 1 to 2 hours of acute MI and peaks within 3 to 15 hours.
- Because myoglobin is also present in skeletal muscle, an elevated myoglobin level is not specific for the diagnosis of MI.

Management

Goal of management:

- ✓ Minimize myocardial damage
- ✓ Preserve myocardial function
- ✓ Prevent complications
- ✓ Reperfusion of heart muscle
- ✓ Resolution of pain and ECG changes
- ✓ Reversing and preventing further damage to the myocardium.

Non-drug treatment (General measures)

- ✓ Bed Rest: Strict in the 1st 12hrs. If no complications, sit in 24hrs , and Ambulate in 48-72hrs.
- ✓ Diet: Low fat, low Sodium , high fiber diet.
- ✓ Bowel: Bed rest + Narcotics→ Constipation, Stool softener & Laxatives
- ✓ Sedation: ↓Anxiety & ensures adequate sleep
 - **Diazepam** 5mg 3-4x/day , Additional dose at bed time

- ❖ **Supplemental oxygen** is used to help meet myocardial oxygen demand. Dose: 2-4 l/min, via facemask, if patient is hypoxic (low O2 saturation) **PLUS**
- ❖ Administer sublingual **nitroglycerin** (unless the systolic blood pressure is less than 90 mm Hg).
Dose: 0.5mg, sublingual, every 5 min up to 3 doses
- ❖ Administer **analgesics** to relieve pain, reduce pulmonary congestion, and decrease myocardial oxygen consumption.

Eg. Morphine.

Dose: 2-4 mg IV. every 5 min until the desired level of analgesia is achieved or until unacceptable side effects occur.

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- ❖ Administer **thrombolytic** therapy within 3 to 12 hours of onset because thrombolytic drugs lyse coronary thrombi by converting plasminogen to plasmin.

Eg. Alteplase, Streptokinase, Anistreplase & Reteplase.

- ❖ Heparin should be given following thrombolytic therapy. Dose: 7500 units subcutaneously every 12 hours BID until the patient is ambulatory.

- ❖ **Contraindications**

- Previous hemorrhagic stroke at any time
- Active internal bleeding (does not include menses)
- Active peptic ulcer

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- ❖ Administer **antiarrhythmics** because arrhythmias are common as are conduction disturbances.

Eg. Amiodarone, Lidocaine & Procainamide.

- ❖ Administer **calcium channel blockers** as they appear to prevent reinfarction and ischemia. Eg. Verapamil, Diltiazem.

- ❖ Angiotensin-converting enzyme (ACE) inhibitors are administered to patients with MI with heart failure in the absence of significant hypotension.

- ✓ ACE inhibitors help prevent ventricular remodeling (dilation) and preserve ejection fraction.

Example: Enalapril, 5 - 40 mg P.O. once or divided into two to three doses daily.

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- ❖ Administer **beta-adrenergic blockers** because they reduce the duration of ischemic pain and the incidence of ventricular fibrillation; decreases mortality.
- ✓ They reduce oxygen demand by decreasing the heart rate and contractility.
- ✓ They also increase coronary artery filling by prolonging diastole.

Eg. Propranolol, Metoprolol.

- Metoprolol dosage: 5 mg I.V. every 2 to 5 min for a total of 3 doses
- Atenolol dosage: 50-100mg P.O. QD

Nursing interventions

- ✓ Administer opioids as prescribed (morphine: decreases sympathetic activity and reduces heart rate, respirations, BP, muscle tension, and anxiety).
- ✓ Offer support and reassurance to patient that relief of anxiety is a priority.
- ✓ EKG continuous monitoring for arrhythmias.
- ✓ Administer sublingual **nitroglycerin** as directed; recheck BP, heart rate, and respiratory rate before administering nitrate therapy and 10 to 15 minutes after dose.

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- ✓ Vital signs—check for changes in BP, pulses.
- ✓ Pulse-oximetry monitoring. Maintain oxygen saturation greater than 92%. Administer oxygen by nasal cannula if prescribed
- ✓ **Monitor:**
 - Cardiovascular—look for changes or instability in pulse, heart sounds, murmur.
 - Respiration—look for changes, fluid in lung fields, shortness of breath.

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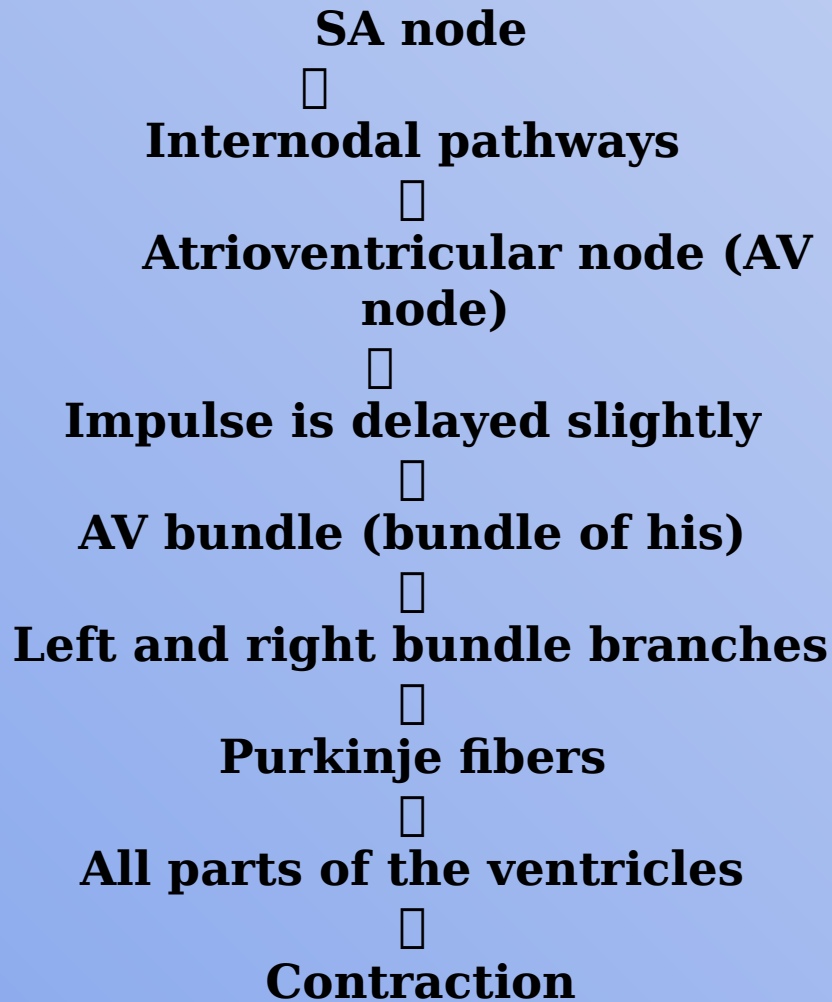
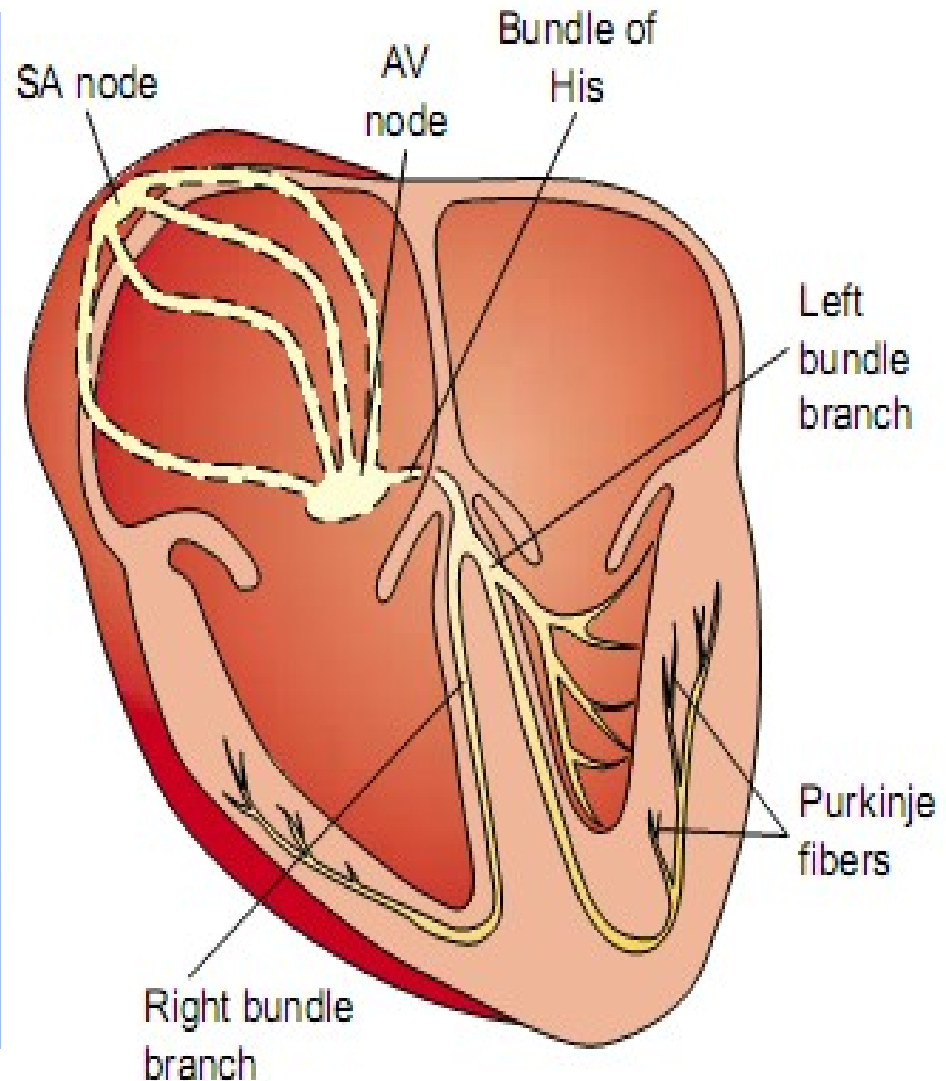
- ✓ Explain to the patient:
- Change to a low-fat, low-cholesterol, low-sodium diet.
- Smoking cessation.
- Limit activities.
- Stress reduction.
- Lifestyle changes such as diet changes

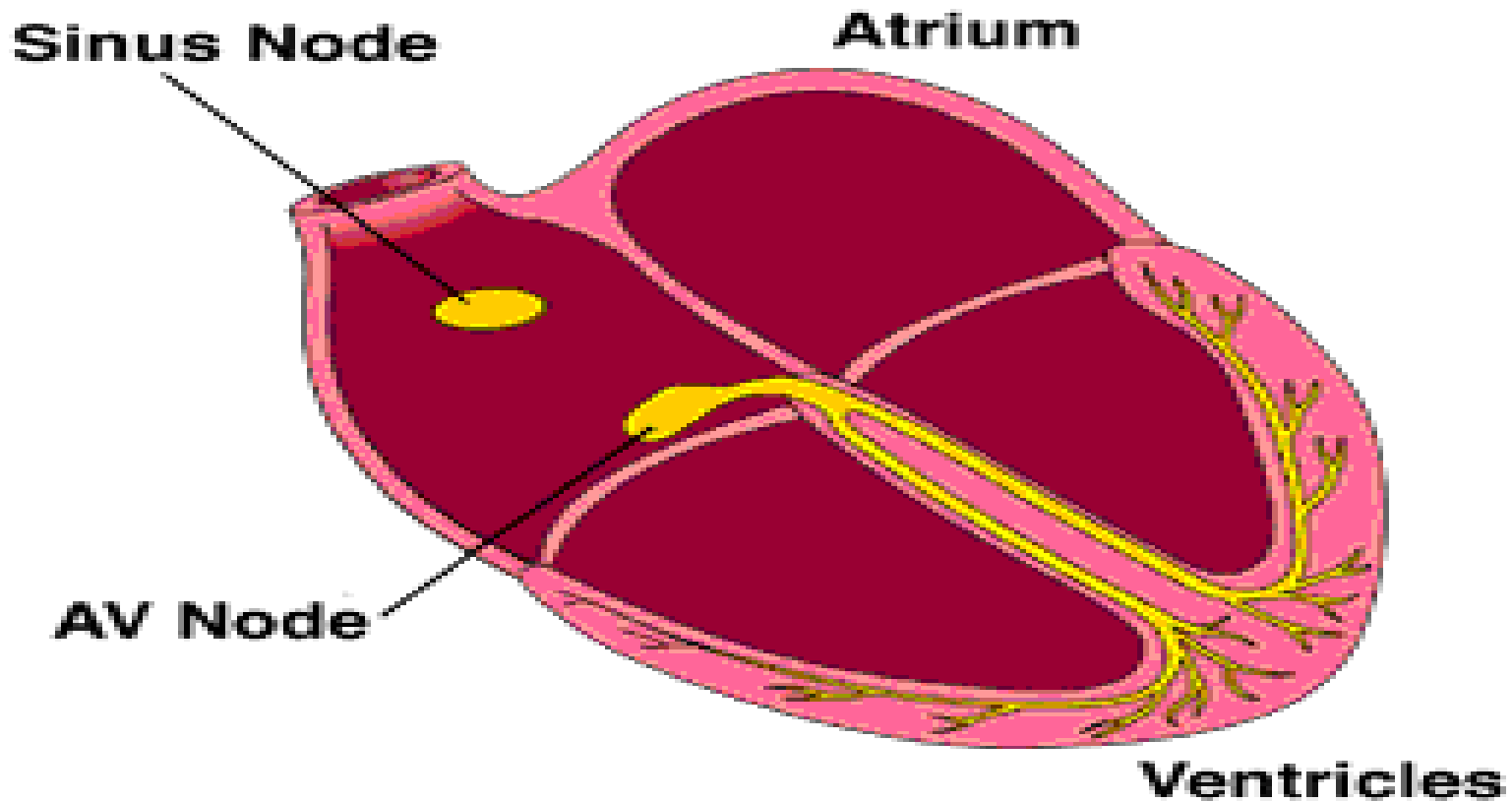
Electrical disorders

Arrhythmias/Dysrhythmias

- ✓ **Heart generates and transmits** electrical impulses that stimulate contraction of the myocardium.
- ✓ In the heart there are pathways for action potential that triggers each contraction of the heart muscle.
- This conduction system starts at the sinoatrial node (SA node) in the wall of the ***right atrium*** near the entrance of the superior venacava.
- The electrical impulse quickly travels from the sinus node through the atria to the atrioventricular (AV) node , then to purkinje fibers, located in the ventricular muscle

The Impulse Travel

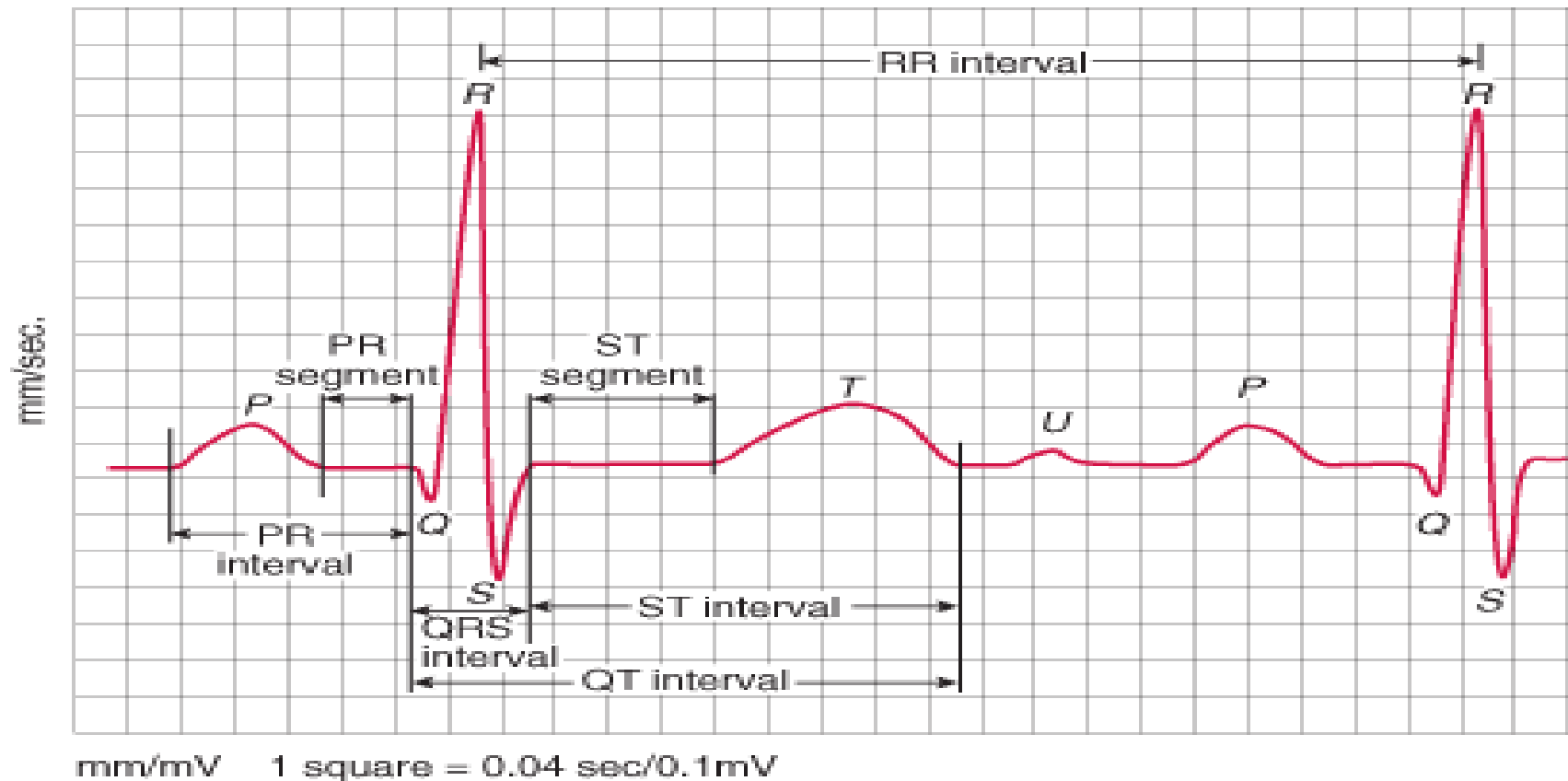




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ECG

- **ECG** provides a graphic representation of cardiac electrical activity



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- Arrhythmia/Dysrhythmia is an **abnormal** electrical activity of the heart.
- It is disorders of the **formation or conduction** (or both) of the electrical impulse within the heart resulting in disturbances of the **heart rate, the heart rhythm**, or both.
- May cause the heart to beat too slowly (**bradycardia**) or too rapidly (**tachycardia**).

Causes:

- Ⓢ Factor that stimulate the heart (stress, caffeine, alcohol, nicotine etc)
- Ⓢ Certain drug that contain caffeine
- Ⓢ Congenital defect of the heart
- Ⓢ Coronary Artery Disease (CAD)
- Ⓢ Myocardial Infarction (MI)
- Ⓢ Rheumatic Heart Disease (RHD)
- Ⓢ Heart block (1st, 2nd and 3rd degree AV block)

Types of Dysrhythmias

Sinus bradycardia

- Occurs when the **sinus node** creates an impulse at a **slower**-than-normal rate(<60/min)
- Causes
 - MI
 - Lower metabolic rate, Hypothyroidism, Hypothermia
 - Increase vagal tone (eg, from vomiting, suctioning, severe pain, extreme emotions)
 - Increase ICP
 - Medications (eg, calcium channel blockers, amiodarone, beta-blockers)

Sinus bradycardia



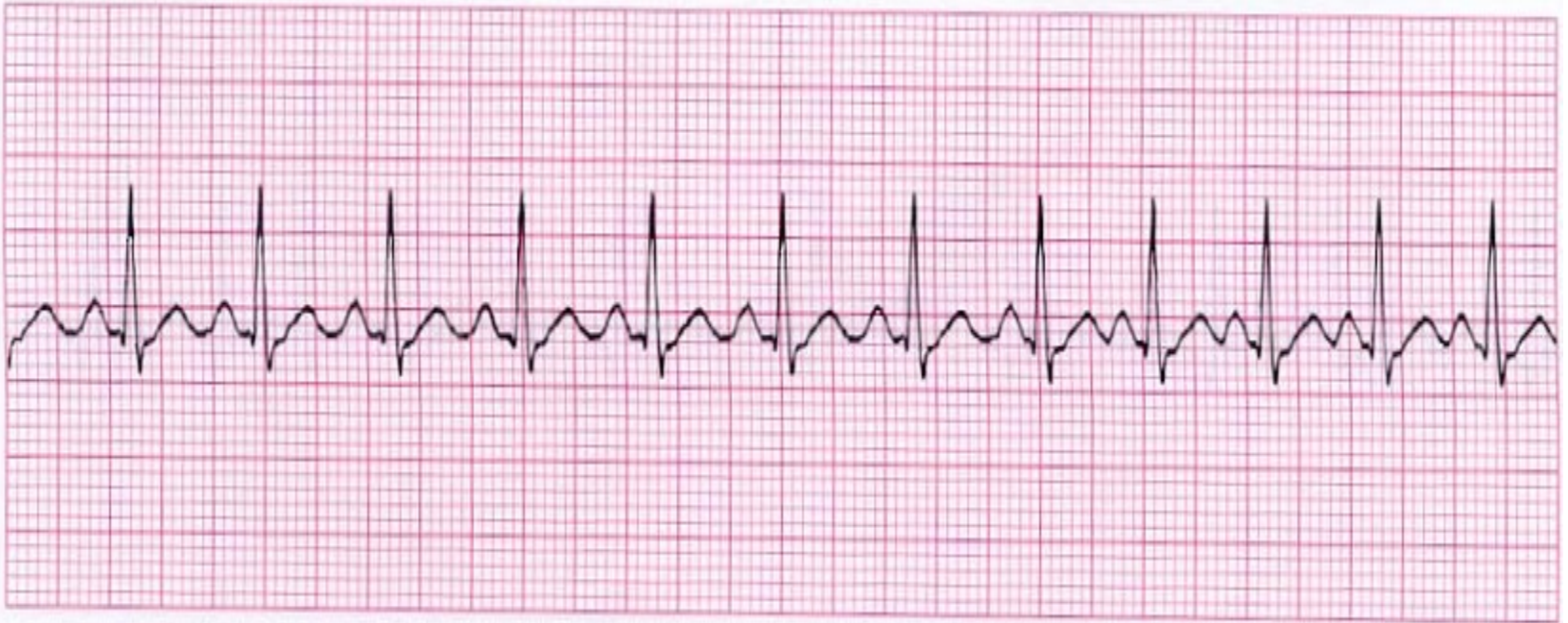
Sinus tachycardia

- Occurs when the sinus node creates an impulse at a faster-than-normal rate(>100/min)

Causes

- ✓ Thyrotoxicosis, Drugs – epinephrine
- ✓ Acute blood loss, Hypotension, Hypovolemia
- ✓ Anemia
- ✓ CHF
- ✓ Sympathomimetic medication
- ✓ Physical activity; hemorrhage; shock, dehydration; fever; electrolyte imbalance; fear; and anxiety

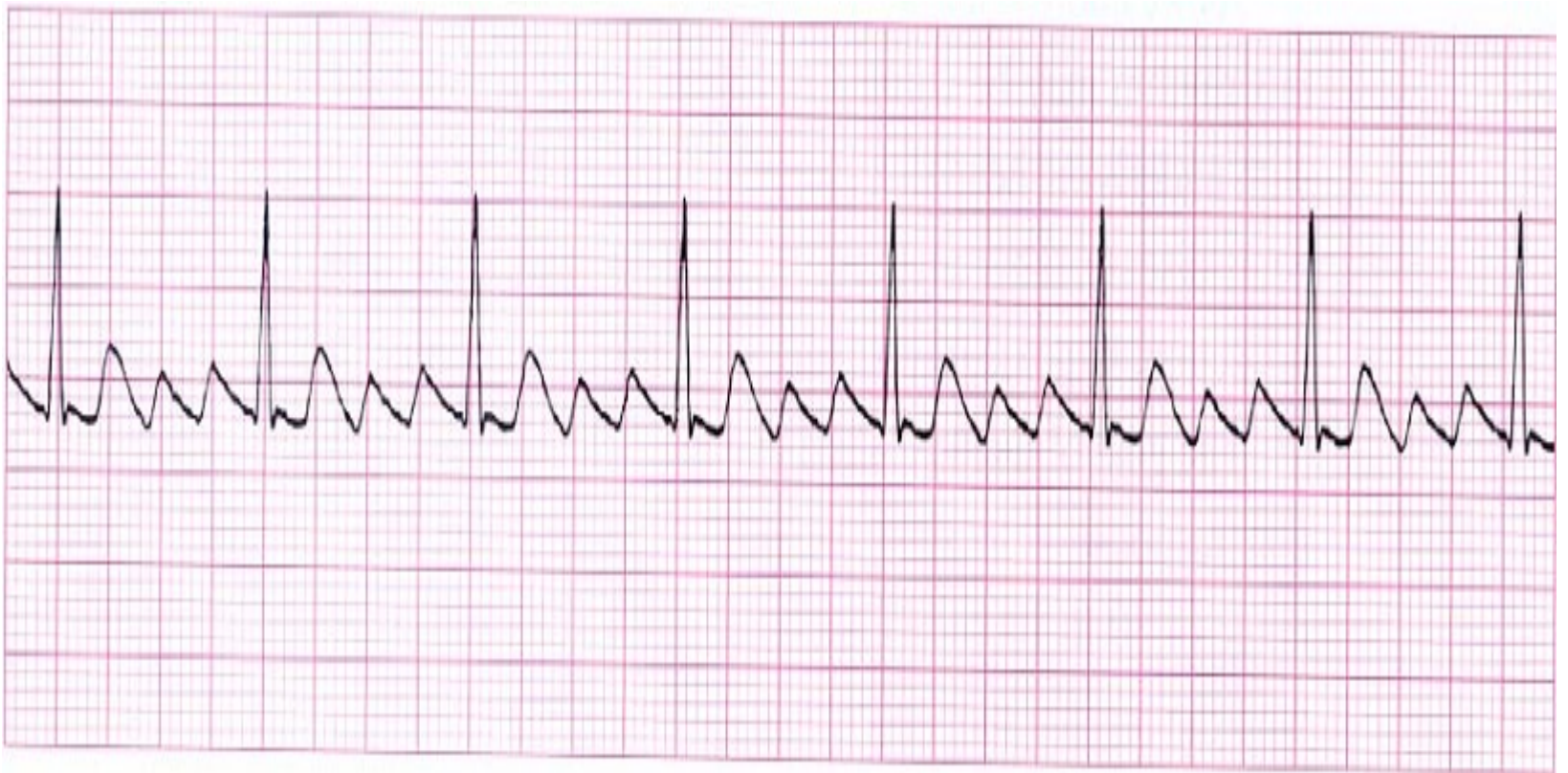
Sinus tachycardia



Atrial flutter

- When atrium create impulse at atrial rate between 250-400b/minutes
- Ventricular and atrial rate: Atrial rate ranges between 250 and 400; ventricular rate usually ranges between 75 and 150
- Occurs because of a ***conduction defect in the atrium*** and causes a rapid, regular atrial rate, usually between 250 and 400 times per minute.

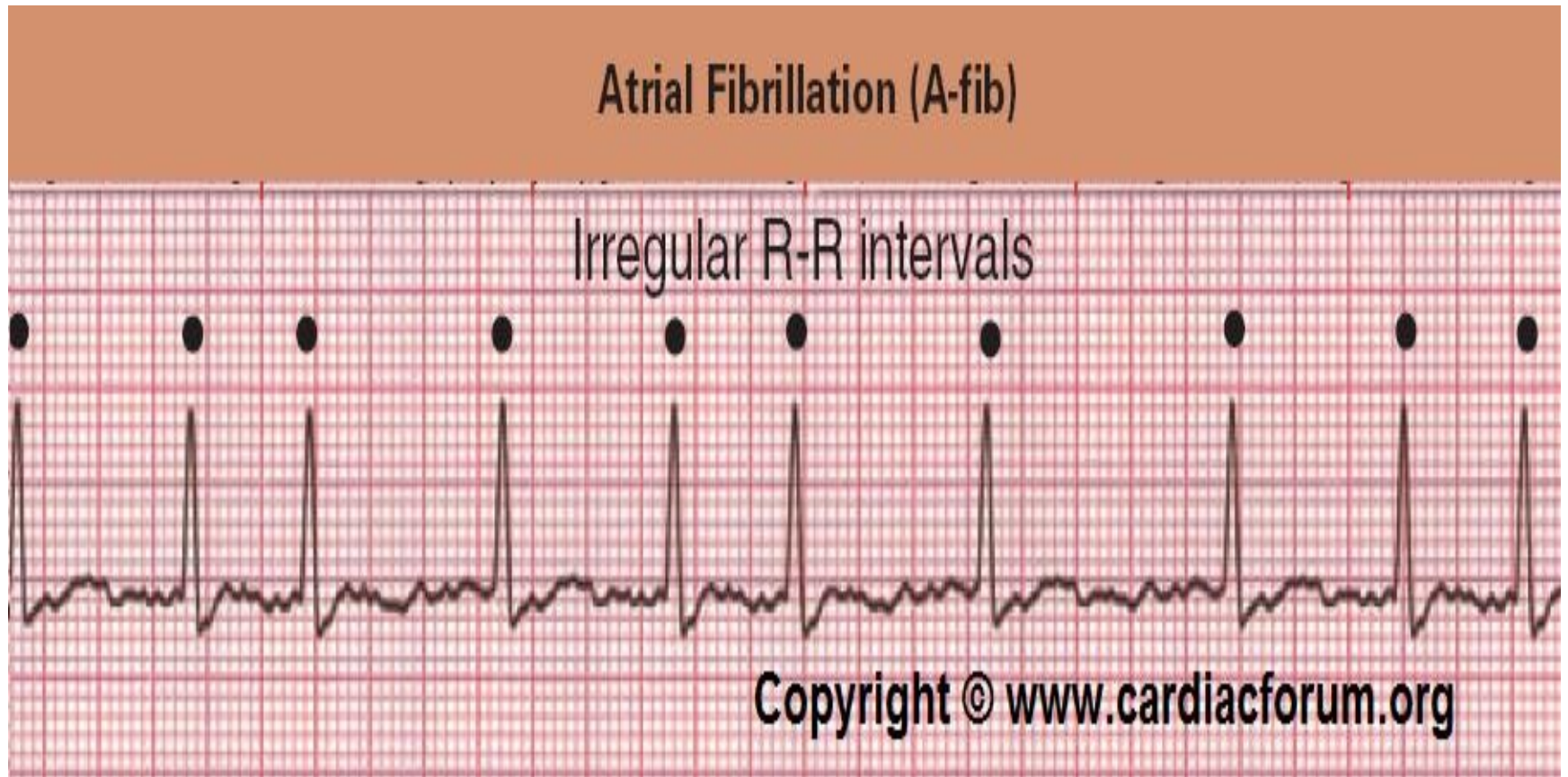
Atrial flutter



Atrial fibrillation

- Is an uncoordinated atrial electrical activation that causes a rapid, disorganized, and uncoordinated twitching of atrial musculature.
- Severe derangement of the heart rhythm, that represents the firing of multiple ectopic foci in the ventricle. HR is not measurable.
- Usually occurs in people of advanced age with structural heart disease, such as valvular heart disease, coronary artery disease, hypertension, congenital disorder (especially atrial septal defect), and heart failure (diastolic or systolic).
- Absence of discrete P waves & irregular QRS complex

Atrial fibrillation



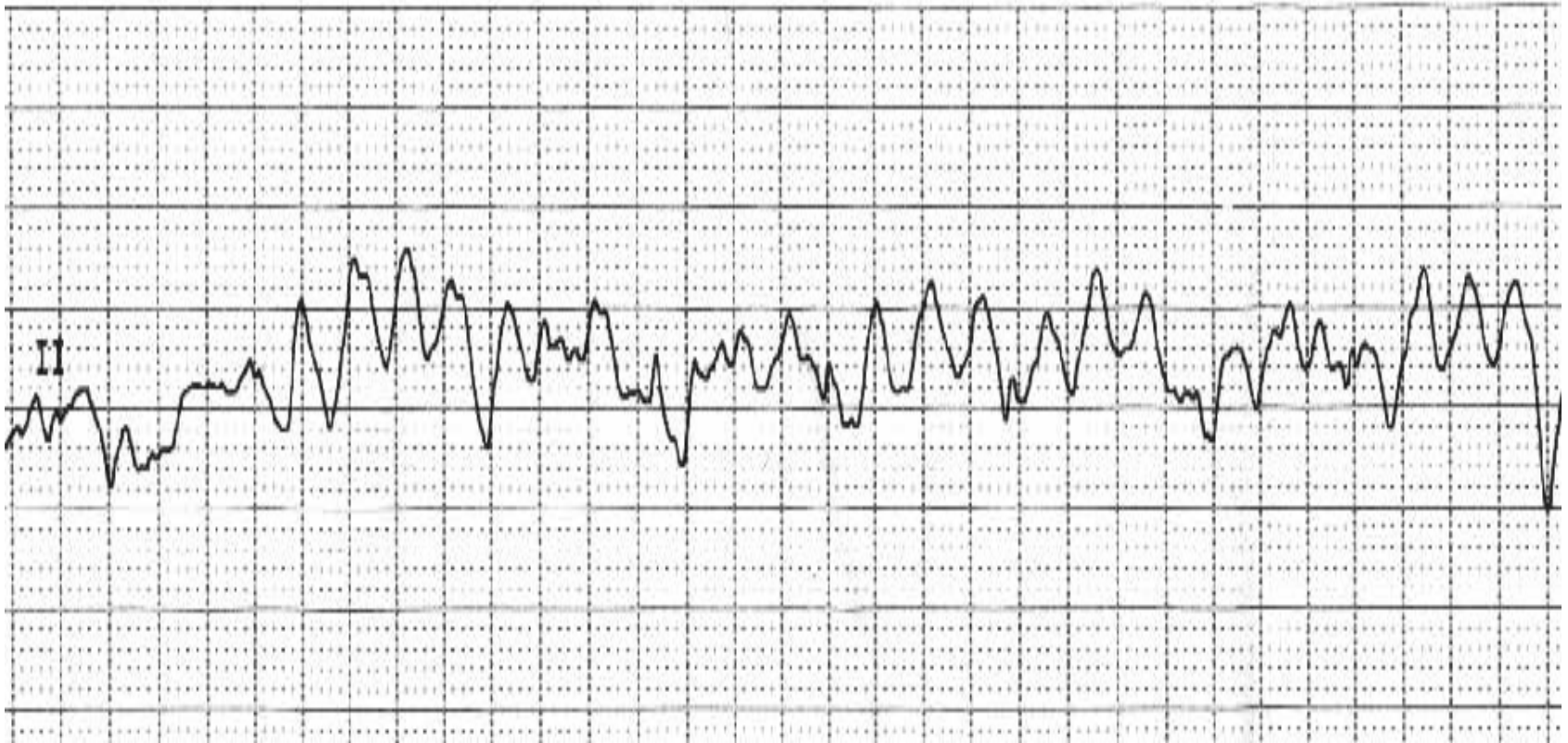
Ventricular tachycardia

- Occurs at rate $>100/\text{min}$
- It is suggested by a wide-complex QRS tachycardia at a rate exceeding 100 beats per minute.
- **Causes**
 - ☀ Cardiac ischemia/infracton
 - ☀ Digital toxicity
 - ☀ Electrolyte imbalance/hypokalemia
 - ☀ Work load on the heart (exercise, HF)

Ventricular fibrillation

- Is a rapid but disorganized ventricular rhythm that causes in effective quivering of the ventricle(Ventricular rate >300/min)
- Ventricular fibrillation is caused by **numerous waves of depolarization** spreading throughout the **ventricles** simultaneously, leading to disorganized ventricular contraction and immediate loss of cardiac function.
- Absent P waves
- Disorganized electrical activity, Deflections continuously change in shape, magnitude and direction

Ventricular fibrillation



Atrioventricular block

First degree: prolonged, constant PR interval (>0.20 s). May be secondary to increased vagal tone or digitalis; no treatment required.

Second degree: Narrow QRS, progressive increase in PR interval. Seen with drug intoxication (digitalis, beta blockers), increased vagal tone, MI. Usually transient, no therapy required; if symptomatic, use atropine (0.6 mg IV, repeated 3-4)

Third degree (complete AV block): No atrial impulse propagates to the ventricles. Seen with MI, digitalis toxicity, or degenerative conduction system disease.

Clinical manifestation of arrhythmia

- Chest pain
- SOB
- Light headiness
- Dizziness and fainting
- Brady cardia
- Tachycardia

Diagnosis

C/M, P/E & ECG

Treatment of arrhythmias

Sodium channel blockers

- Largest group of anti-arrhythmic drugs.
- Block voltage gated **sodium** channel,
- Decrease in **excitability** of cardiac cells and impulse conduction **velocity**, reduced **heart rate**.
- Subdivided into class IA, IB & IC based on their different effect on the **duration of AP** even though they have the same basic mechanism of action (Na channel blockade).

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Class IA, e.g, Quinidine, Procainamide

- ✓ Quinidine : atrial fibrillation and flutter and to prevent recurrent tachycardia and fibrillation
- ✓ Procainamide: acute treatment of ventricular arrhythmias

Class IB, e.g, Lidocaine, Phenytoin

- ✓ Used in Ventricular tachycardia and fibrillation
- ✓ Not used in atrial arrhythmias or AV junctional arrhythmias

Class IC, e.g, Flecainide, Propafenone

- ✓ Used for supraventricular fibrillation and flutter

β - blockers: Propranolol, Acebutolol, Metoprolols

- **Sympatholytic** activity (they reduce beta adrenergic activity in the heart).
- They decrease **sympathetic tone** in the heart, reduce automatic discharge in the SA node (they reduce automaticity) & AV nodal conduction.
- Increase/prolong the PR interval – from SA node to AV node.
- Used in treating **sinus tachycardias**, protecting the ventricles from high atrial rates (slow AV conduction)

Potassium CB: Amiodarone, Sotalol, Ibutilide

- Delay repolarization (blocking of K channel)
- Prolongation of Action Potential duration.
- Used for most arrhythmias

CCBs: Verapamil, Diltiazem

- Blocks cardiac calcium currents (depress slow inward of Ca current).
- **Prolong conduction time**, therefore PR interval increase (from SA node to AV node).
- Used to treat supraventricular tachycardia

Miscellaneous

Adenosine → inhibits AV conduction & increases AV refractory period. It decreases conduction velocity, and decreases automaticity in the AV node.

Digoxin (cardiac glycosides): enhances vagal activity, inhibits Na/K ATPase □ refractory period, slows AV conduction.

Uses: treatment of atrial fibrillation and flutter

Atropine: selective muscarinic antagonist

Cardiac effects: blocks vagal activity to **speed AV** conduction and increase HR

Uses: treat vagal **bradycardia**

Nursing Interventions

- ☞ Monitoring and Managing the Dysrhythmia
 - ▶ Antidysrhythmic drugs
 - Assess carefully for side effects
 - Ensure continuous ECG monitoring
 - ☞ Minimizing Anxiety (reassurance)
 - ☞ Be aware any precaution before provide the drug
- example: -PR < 60/min => don't give digoxin

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- ✓ Monitor PR, BP, consciousness, urinary output, poor peripheral perfusion and report abnormal finding
- ✓ Initiate CPR as indicated
- ✓ Monitor fluid and electrolyte imbalance
- ✓ Provide O₂ if necessary
- ✓ Provide all safety measures during the procedures

Infective endocarditis

- A **microbial infection** of the endothelial surface of the heart or valves.
- It is an inflammatory process of the endocardium, especially the **valves**.
- Often associated with destruction of underlying cardiac tissue.

Etiology

- Usually Bacterial
 - Staphylococcus aureus (30-40%)
 - **Streptococcus** viridans (most common cause (35-60%))
 - Enterococci
- Sometimes Fungal
 - Candida albicans

Predisposing factors

- ❖ Microorganisms enter the blood stream in many ways:
 - Dental disease (caries, abscess)
 - Through the oral cavity after dental procedures, mouth or tooth abscess
 - Oral irrigations, or oral irritations from dental floss
 - URT surgery, intubations, prosthetic heart valves, and structural heart disease
 - Prolonged IV catheters, IV drug use
 - GI & genitourinary tract procedures

Clinical Manifestations

Early

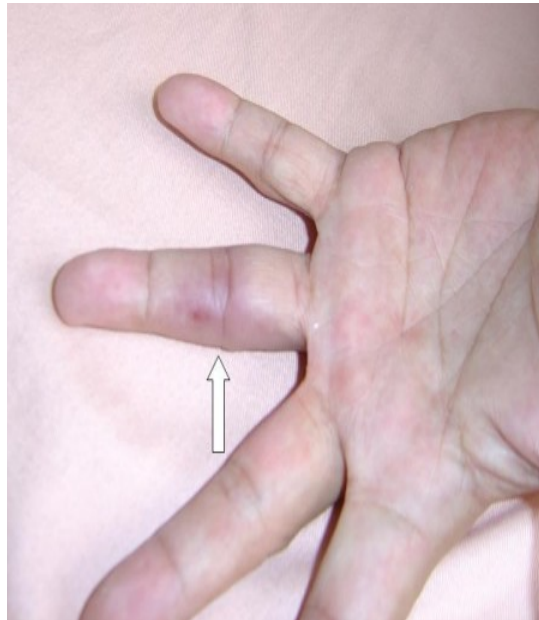
- ✓ Weakness
- ✓ Anorexia
- ✓ Fever (most common)
- ✓ Fatigue
- ✓ Arthralgia(Pain in a joint or joints)
- ✓ Myalgia
- ✓ Murmur (80-85% patients)
- ✓ Weight loss
- ✓ Night sweats

Late

- ❖ Embolization can occur in any part of the body (clinically in 11 - 43%)
- ❖ Stroke, transient ischemic attacks
- ✓ Loss of vision from embolization to the brain or retinal artery
- ✓ Myocardial infarction, due to coronary artery embolism
- ✓ Pulmonary embolism in > 50% cases
- ✓ **Osler's nodes** - painful, erythematous, pea sized nodules on tips of fingers & toes resulting from inflammation around a small, infected embolus
- ✓ Hematuria, proteinuria due to immunologic reaction to infection leads to renal failure

Embolic Lesions

Osler's Nodes: Are Small, Painful Petechiae in Extremities

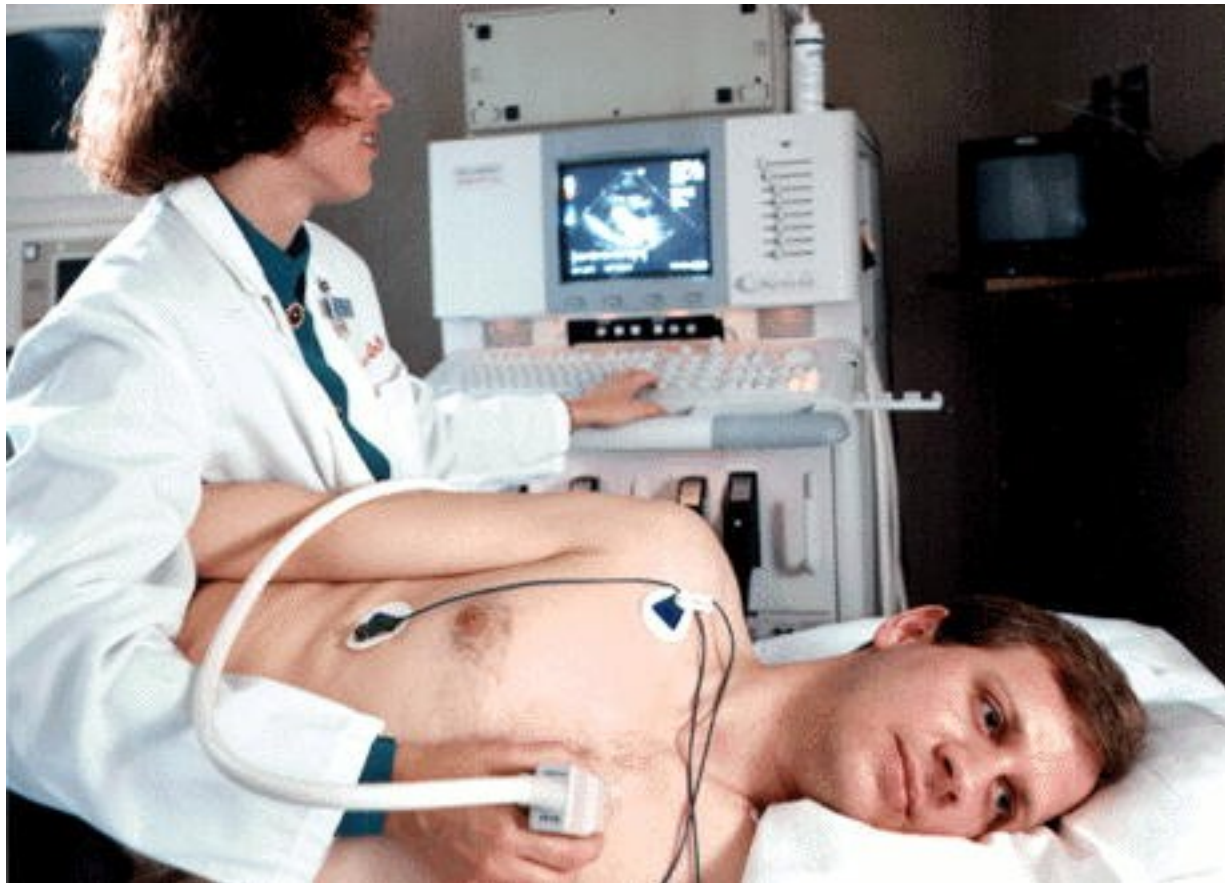


Diagnostic procedures

- ✓ Clinical (based on sign and symptoms)
- ✓ Three or more blood cultures with in 24-48hrs (Isolation of microorganism)
- ✓ CBC: Increase WBC
- ✓ Increase ESR(The rate at which red blood cells settle out in a tube of blood under standardized conditions; a high rate usually indicates the presence of inflammation)
- ✓ Microscopic hematuria, Urea and Creatinine may be elevated due to glomerulonephritis
- ✓ Bacteremia (Aerobic and anaerobic)

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- ✓ Electrocardiogram: To detect complications like MI, conduction abnormalities.
 - Conduction delays
 - Ischemia or infarction
- ✓ Chest X-ray
- ✓ Echocardiography
 - Detection/characterization of valvular lesions
 - Detection of associated abnormalities



Management

Medical management

- ❖ Crystalline penicillin 2-3 million IU every 4 hours for 4 weeks OR
- ❖ Gentamycin 1 mg/kg TID with crystalline penicillin for 02 weeks OR
- ❖ Ceftriaxone 2 gm once daily IV or IM for 04 weeks

Prophylaxis

- ❖ Antibiotics before undergoing:
 - ✓ Surgery
 - ✓ Dental treatment
 - ✓ Instrumentation
 - ✓ Gynecology or obstetric manipulations

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- ❖ Antibiotic regimen for infective endocarditis
 - **Streptococci**
 - ✓ Benzyl penicillin (1.2g IV 4 hourly) 4-6 weeks **PLUS**
 - ✓ Gentamicin (1mg/kg IV 8-12 hourly) 4-6 weeks **OR**
Vancomycin 15 mg/kg IV BID for 4 weeks
 - **Enterococci**
 - **Ampicillin sensitive**
 - ✓ Ampicillin (2 g 4 hourly) for 4-6 weeks **PLUS**
 - ✓ Gentamicin (1mg/kg IV 8-12 hourly) for 4-6 weeks
 - **Ampicillin resistant**
 - ✓ Vancomycin(1g IV12hourly) for 4-6 weeks

Complications

- ❖ Congestive heart failure (~60% of IE patients)
 - **Most common complication**
 - Main indication to surgical treatment
- ❖ Uncontrolled infection
 - Persisting infection
 - Perivalvular extension and cause pericarditis
- ❖ Systemic embolism (30% of IE patients)
 - Brain, spleen, kidney and lungs
- ❖ Acute renal failure & Neurologic events (embolic stroke, purulent meningitis)

Nursing interventions

- Treat fever when present with rest, cooling measures, forced fluids, and analgesics
- Do not enforce complete bed rest unless fever or signs of heart damage develop
- Assess for rapid heartbeat, easy fatigability, dyspnea, restlessness, signs of heart failure
- Warn for signs of embolization:
 - Hematuria
 - Pleuritic chest pain
 - LUQ pain

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- Provide reassurance for prolonged therapy
- Avoid excessive physical exertion
- Make sure susceptible patients to get prophylactic antibiotic
- Continue taking your IV antibiotics
- Floss every day, and brush with a soft tooth brush
- Monitor yourself for manifestations of infective endocarditis. Take your T⁰ everyday, report any fever, chills malaise, loss of appetite, weight loss, or increased fatigue to your nurses.

Myocarditis

- Myocarditis is an inflammatory disorder of the myocardium.
- It is usually caused by a **viral infection**, particularly adenovirus and enterovirus infections (eg, **coxsackie virus**).
- It results in a decrease in myocardial function resulting in increasing **hydrostatic forces**. These increased forces lead to both **pulmonary edema** and **congestive heart failure**.
- Complete recovery of ventricular function has been reported in as many as 50% of patients.
- Some patients develop chronic myocarditis (ongoing or resolving) and/or dilated cardiomyopathy and may eventually require cardiac transplantation.

Causes

Infectious

- ✓ Viruses (adeno, coxsackie)
- ✓ Bacterial
- ✓ Fungal
- ✓ Protozoal (Chagas' disease)

Non-infectious

- ✓ Ischemic damage & Mechanical trauma
- ✓ Toxins/Drugs (alcohol, anthracyclines)
- ✓ Systemic disorders (sarcoid, lupus, scleroderma)
- ✓ Internal triggers
 - Autoimmune activation against self antigens

Clinical manifestations

- ✓ **Chest pain** (often described as "stabbing" in character)..
- ✓ Symptom of **CHF** (edema, breathlessness and hepatic congestion). This is the most common presenting picture in all ages.
- ✓ **Arrhythmia:** Atrioventricular conduction disturbances, Sinus tachycardia is typical & Junctional tachycardia.
- ✓ Palpitations (due to arrhythmias).
- ✓ Dyspnea on exertion

- ✓ **Fever** (especially when infectious), Chills, Fatigue, Myalgias
- ✓ Since myocarditis is often due to a viral illness, many patients give a history of **symptoms consistent with a recent viral infection**, including fever, diarrhea, joint pains, and easy fatigueability.
- ✓ Myocarditis is often associated with **pericarditis**, and many patients present with signs and symptoms that suggest concurrent myocarditis and pericarditis. Pericardial friction rub (Associated with Pericarditis) due to pericardial effusion.
- ✓ **Sudden death** (in young adults, myocarditis causes up to 20% of all cases of sudden death).

Physical examination

- ✓ Tachycardia, weak pulse, cool extremities, decreased capillary refill, and pale or mottled skin may be present.
- ✓ Heart sounds may be muffled, especially in the presence of pericarditis.
- ✓ Jugular venous distention and edema of the lower extremities may be present.

Diagnosis

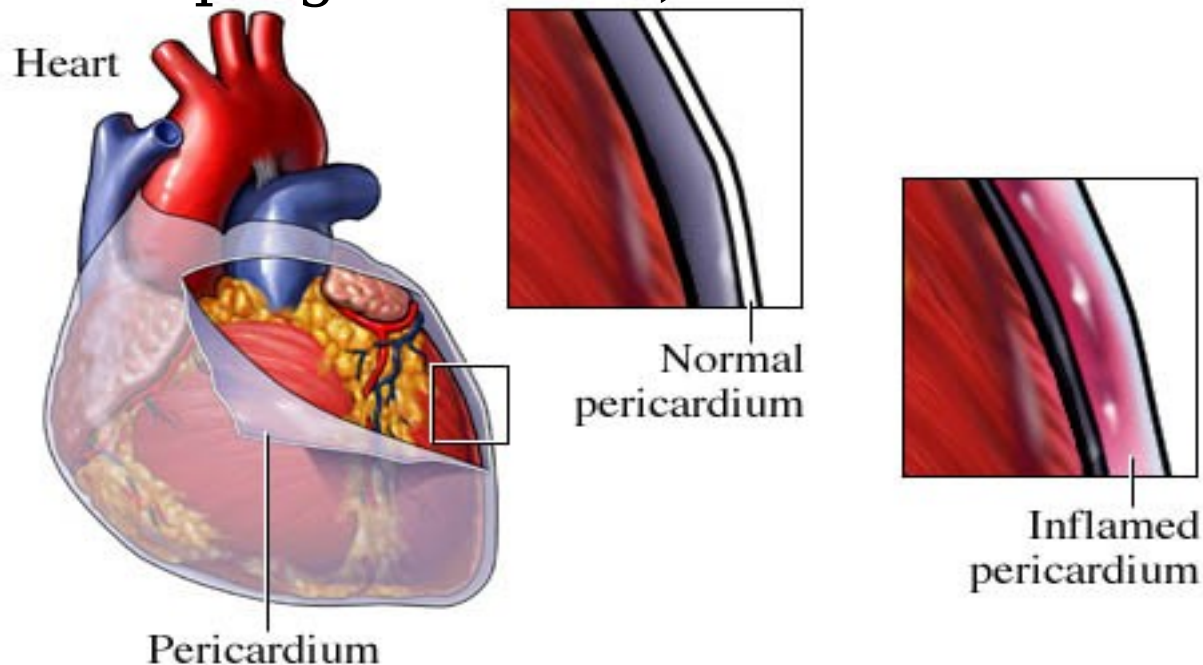
- ✓ Cultures from blood , stools and throat.
- ✓ ECG: T wave inversions, ST-segment elevation
- ✓ Echocardiogram
 - ventricular dysfunction
 - Chamber dilation & Regional hypertrophy
 - Regional wall motion abnormalities
- ✓ CBC (WBC raise), ESR increases
- ✓ Renal function (BUN & creatinine will rises)
- ✓ Cardiac enzymes (CK-MB and Troponin may be elevated) due to myocardial injury.

Treatment

- ✓ Bedrest or limitation of activity in the acute phase. Exercise especially during the acute phase of virus myocarditis enhances viral replication rate, enhances immune mechanisms and increases inflammatory lesions and necrosis.
- ✓ Resumption of physical activity can take place within 2 months of the acute disease.
- ✓ Intubation & ventilation
- ✓ Treatment of PHTN, Diuretics
- ✓ *Digoxin* should be **avoided** in patients suffering from acute HF induced by viral myocarditis
- ✓ ACE inhibitors
- ✓ Restrict salt intake to 2-3g of sodium per day

Pericarditis

- ✓ Pericardial layer covers the heart and protect it from any infection.
- ✓ **Pericarditis** is an inflammation of the pericardium (the membranous sac enveloping the heart).



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- Pericarditis can lead to an accumulation of fluid in the pericardial sac (**pericardial effusion**) and increased pressure on the heart, leading to cardiac tamponade.
- Cardiac tamponade = Mechanical compression of the heart resulting from large amounts of fluid collecting in the pericardial space and limiting the heart's normal range of motion.
- Frequent or prolonged episodes of pericarditis may also lead to **thickening & decreased elasticity** that restricts the heart's ability to fill properly with blood (**constrictive pericarditis**) leading to decreased cardiac output & signs & symptoms of heart failure.

Etiology

Infectious

- ✓ Viral: Coxsackie influenza
- ✓ Bacterial : staphylococcus, streptococcus, meningococcus, pneumococcus
- ✓ Parasitic, Fungal

Myocardial injury

- ✓ Myocardial infarction
- ✓ Cardiac trauma: blunt or penetrating
- ✓ Cardiac catheterization & Post cardiac surgery

Drug reaction

- ✓ Procainamide
- ✓ Hydralazine

Metabolic disorders

- ✓ Uremia= Accumulation in the blood of nitrogenous waste products (urea) that are usually excreted in the urine
- ✓ Myxedema= Hypothyroidism marked by dry skin and swellings around lips and nose as well as mental deterioration

Chronic anemia

Neoplasm: lymphoma

Radiation therapy

Clinical manifestations

- ❖ Chest pain (Pleuritic and sharp in nature) located beneath:
 - ✓ The clavicle
 - ✓ In the neck
 - ✓ In the left scapula region
 - The pain may worsen with deep inspiration & when lying down or turning
 - The pain is relieved with a forward leaning or sitting position.

- ❖ Pericardial friction rub (Present in 85% of cases of pericarditis). Pericardial friction rub is audible throughout the respiratory cycle, whereas the **pleural friction rub disappears when respirations are on hold.**
- ❖ Mild fever
- ❖ Leukocytosis = an abnormal increase in the number of white blood cells in the blood as a result of infection.
- ❖ Increase ESR
- ❖ Dyspnea
- ❖ Sign of heart failure as a result of pericardial compression due to constrictive pericarditis or cardiac tamponade

Diagnostic procedures

- ✓ Patient history (Clinical manifestation)
- ✓ **CBC** – leukocytosis ($\text{WBC} > 20,000$), increase ESR
- ✓ Echocardiogram (99% diagnostic to pericardial effusion) reveals: Inflammation, Fluid build-up, Heart failure.
- ✓ ECG indicate: new widespread ST elevation or PR depression, Bradycardia, Atrial fibrillations
- ✓ **Chest x-ray** — typically normal

Complications

- ❑ Pericardial effusion (accumulation of fluid between the pericardial linings)
- ❑ Cardiac tamponade (a life threatening complication caused by accumulation of fluid in the pericardium). Tamponade occurs when all cardiac chambers are compressed as a result of increased pressure
- ❑ **Constrictive pericarditis** -is the result of scarring and consequent loss of the normal elasticity of the pericardial sac.
- ❑ Heart failure
- ❑ Fibrosis
- ❑ Peripheral oedema
- ❑ Increase systemic venous pressure

Management

- Treat the underlying causes: eg. If bacteria give antibiotics.
- Administer analgesics & NSAIDS such as:
 - Ibuprofen for pain relief during the acute phase & hasten reabsorption of fluid in the patient with rheumatic pericarditis
 - Corticosteroids (e.g. prednisolone) if the pericarditis is severe or if the patient does not respond to NSAIDS
- For patients with a large pericardial effusion:
 - Pericardiocentesis (removal of pericardial fluid)
 - Pericardiectomy (surgical removal of the tough encasing pericardium) is used to release both ventricles from the constrictive & restrictive inflammation.

Nursing process

Nursing diagnosis

- Ineffective breathing pattern related to chest pain
- Altered thermoregulation , hyperthermia, related to infection and inflammation as evidenced by temp of 37.5 to 38 degrees Celsius
- Altered comfort /pain related to inflammation of the pericardium
- Risk for cardiogenic shock related to decreased cardiac output
- Ineffective tissue perfusion related to decreased blood flow
- Anxiety related to therapeutic interventions and uncertainty of prognosis

Nursing Management

- ✓ Position patient in semi fowlers to relieve pain and allow expansion of chest for effective breathing
- ✓ Monitor vital signs closely to detect infections
- ✓ Bed rest during acute conditions
- ✓ Encourage gradual increase in physical activity
- ✓ Assess for symptoms of cardiac tamponade & report immediately if the following manifestations occur:
 - Tachycardia
 - Hypo tension
 - Jugular venous distension

Rheumatic heart disease(RHD)

- Rheumatic heart disease is an **autoimmune** disease affecting the heart causing chronic progressive damage to the heart and its valves.
- The disease follows upper respiratory tract infection with Group A Beta hemolytic streptococci.
- It is a sequel of a previous group A streptococcal infection, usually of the upper respiratory tract. One beta-streptococcal serotype (e.g., M types 3, 5, 18, 19, 24) is linked directly to acute rheumatic heart disease.
- Antigen of GABHS induces **antibody** response w/c leads to autoimmune damage to heart valves, subcutaneous tissue, tendons, joints & basal ganglia of brain.

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- ❖ Etiology: almost always occurs as a delayed sequence (usually 2-3 weeks) to a group A Beta hemolytic streptococcal infection of the URT, usually a pharyngeal infection.
- ❖ The disease affects children and young adults (**5-15years**). 90% are between 5 and 15 years of age.
- ❖ In adult the risk of developing of RHD is about 3% following **group A Beta hemolytic Streptococcal (GAS)** Pharyngitis with a recurrence rate of 1/6.
- ❖ Ineffective treatment of infection results in delayed reaction and inflammation of the cardiac tissues and CNS, joints, skin, and subcutaneous tissue.

- The disease involves the heart, joints, central nervous system (CNS), skin, and subcutaneous tissues.
- The incidence of RHD has been lowered in the developed countries but is still high in poor communities
- The dramatic decline in the incidence of rheumatic fever in the developed world is thought to be largely owing to antibiotic treatment of streptococcal infection, though it started to decline before the era of antibiotic, probably due to improvement of **socioeconomic status**.

Pathophysiology

- ✓ Manifestation of RHD appear to be related to an abnormal immunological response to an URTI with a group A Beta hemolytic streptococci.
- ✓ An abnormal humoral and cell-mediated immune response to group A streptococci cell membrane antigens cross-react with other tissues and bind to receptors on:
 - Heart
 - Muscle
 - Joint and brain cells triggering immune and inflammatory responses

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➤ These responses results in:-

- Cardiac lesions
- Carditis involving all layers of the heart
 - Valvular deformity - mitral and aortic valves are most commonly affected but less Commonly → tricuspid and rarely pulmonic valves.

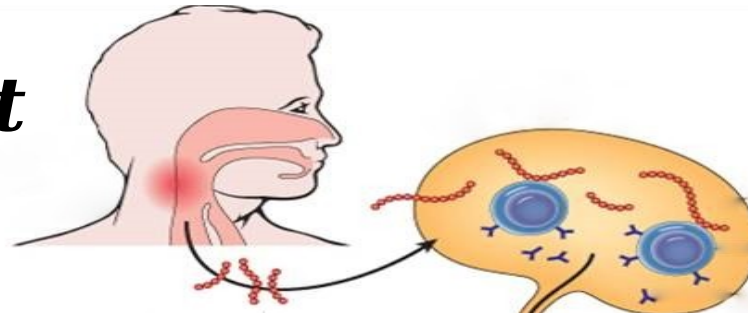
→ it results in valvular:-

- Stenosis → narrowing of the valves
- Regurgitation → failure of the leaflets to oppose

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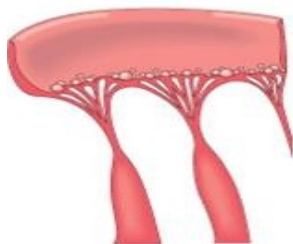
- Extra cardiac lesions:- it is a systemic disease involving especially the connective tissues:-
 - The joints → polyarthrititis
 - The skin → subcutaneous nodules
 - CNS → chorea - (inflammation of CNS) only when brain and spinal cord is involved
 - Lungs → fibrinous pleurisy, pneumonitis

**Strep
throat**

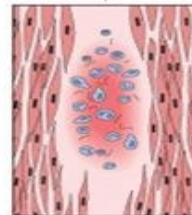
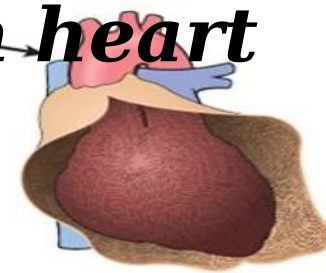


**Antibod
y
product
ion**

**Antibody cross-
reaction
within heart**



vegetation



pericarditis

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DAMEE

CVD, 2023



Predisposing factors

- ❖ Socioeconomic Factors
 - ✓ Common in low socioeconomic groups
 - ✓ Major public health problem in the poor countries because the contributing factors are:
 - Crowded living
 - Inadequate Rx.
 - Poor nutrition
 - Lowered state of health
- ❖ Genetic factor
- ❖ Altered immune response
- ❖ Living in urban areas than rural communities

Clinical Manifestation

- Symptoms of streptococcal pharyngitis may precede rheumatic symptoms e.g.
 - Sudden onset of sore throat; throat reddened with exudate
 - Swollen, tender lymph nodes at angle of jaw
 - Headache and fever >38 degrees celsius
 - Some cases of streptococcal throat infection are relatively **asymptomatic**

- ✓ **Carditis** occurs in as many as 40- 60% of patients and may manifest as: New murmur, Cardiomegaly & Congestive heart failure.
- ✓ **Pericarditis** with or without a pericardial rub
- ✓ **Valvular disease:** mitral and aortic valves are commonly affected.
- ✓ Heart murmurs; pleural and pericardial rubs.
- ✓ **Migratory poly arthritis** (S/S - swelling, heat, Tenderness, redness). Location: knees, ankles, Elbows, wrists. It occurs in 75% of cases
- ✓ **Chorea** -(irregular, jerky, involuntary, unpredictable muscular movements especially affecting the head, face or limbs)

- ✓ **Subcutaneous nodules** (S/S firm small, hard, painless swelling found most commonly over knees, elbows, spine, scapulae). It occur in 10% of patients and are edematous fragmented collagen fibers.
- ✓ **Erythema marginatum** (S/S - nonpruritic, non painful and neither indurated or raised bright). Location: Inner aspects of the upper arm and thigh but never on the face. It occurs in about 5% of cases.
- ✓ **Arthralgia** (pain in joint) and limitation of motion
- ✓ Fever >38 degrees celsius.
- ✓ Prolonged Pulse Rate (heart beat) interval demonstrated by ECG.

Diagnosis

- Jones criteria developed by the American Heart Association is used to make the diagnosis.

Major Criteria

- ✓ Carditis
- ✓ Migratory poly arthritis
- ✓ Subcutaneous nodules
- ✓ Erythema marginatum

Minor Criteria

- ✓ History of previous attack
- ✓ Fever, Arthralgia (pain in joint) and limitation of motion

NB: 2 major Jones criteria or 1 major plus 2 minor Jones criteria
PLUS positive throat culture or rapid antigen antibody test.

Laboratory Studies

- ✓ Streptococcal antibody tests disclose preceding streptococcal infection
 - ASO titer: positive in 80% of cases
 - Anti DNAase β & Anti hyaluronidase is positive in 95 % of cases
- ✓ Isolate group A streptococci via throat culture which has 20-40% yield (***positive throat culture***)
- ✓ Acute phase reactants (raised ESR, C-reactive protein)
- ✓ Leukocytosis
- ✓ Anemia usually is caused by suppression of erythropoiesis.

ECG

- ✓ PR interval prolongation is seen in 25% of all cases but is neither specific to nor diagnostic.

Echocardiography

- ✓ Helps evaluate valvular damage, chamber size, ventricular function and the presence of a pericardial effusion.

Treatment

Medical therapy

- ❑ Treat group A streptococcal (GAS) infection regardless of organism detection with appropriate antibiotic.
- ✓ Benzathin penicillin 1.2 million IU IM for 10 days

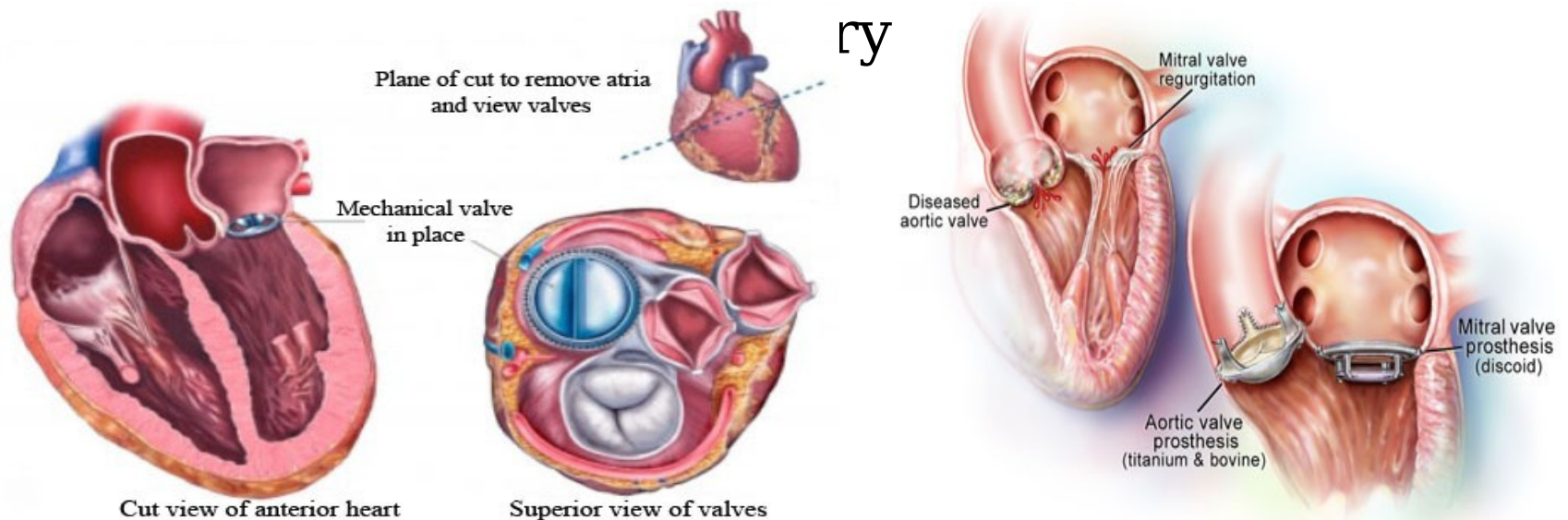
Alternatives:

- ✓ Erythromycin, 250mg four times P.O. daily for 10 days. PLUS
- ✓ Corticosteroids → 40-60 mg. PO/day with tapering dose for 2 week → for carditis and joint symptom. Eg. Prednisolone

- ❑ Treat CHF by conventional therapy such as digoxin and diuretics

Surgical therapy

- ✓ The definitive treatment in patients with significant symptoms not responding to conservative treatment is surgical valve replacement.
- ✓ Severe mitral or aortic valve dysfunction



Complications

- ✓ Heart failure
- ✓ Atrial fibrillation
- ✓ Infective endocarditis
- ✓ Atrial and ventricular arrhythmias

Nursing assessment

History:

- ✓ Recent streptococcal infection
- ✓ Chest pain
- ✓ Migratory joint pain
- ✓ Tenderness (especially large joints)

Objective data:

- ✓ Fever
- ✓ Subcutaneous nodules & Erythema marginatum
- ✓ pericardial friction rub
- ✓ Gallop rhythm, Murmurs &
- ✓ Peripheral edema

Nursing diagnosis

- Acute Pain related to migratory inflammation of the joints.
- Activity Intolerance related to joint pain.
- Decreased cardiac output related to valve dysfunction of heart failure as evidenced by low blood pressure
- Altered comfort pain(joint pain) related to swollen joint as evidenced by patient's verbalisation and inability to stretch joints well.

Nursing interventions

❖ **Reducing fever**

- ✓ Do tepid sponging to reduce fever through conduction and evaporation and administer antipyretic e.g. ASA.

❖ **Maintaining Adequate Cardiac Output**

- ✓ Monitor for tachycardia (usually persistent when patient sleeps) or bradycardia.

❖ **Pain management**

- ✓ Total bed rest /quiet environment for the comfortability of the patient.
- ✓ Administer prescribed analgesic drugs eg. PCM
- ✓ Diversion therapy- avoid the patient's mind concentrating on his/her pain

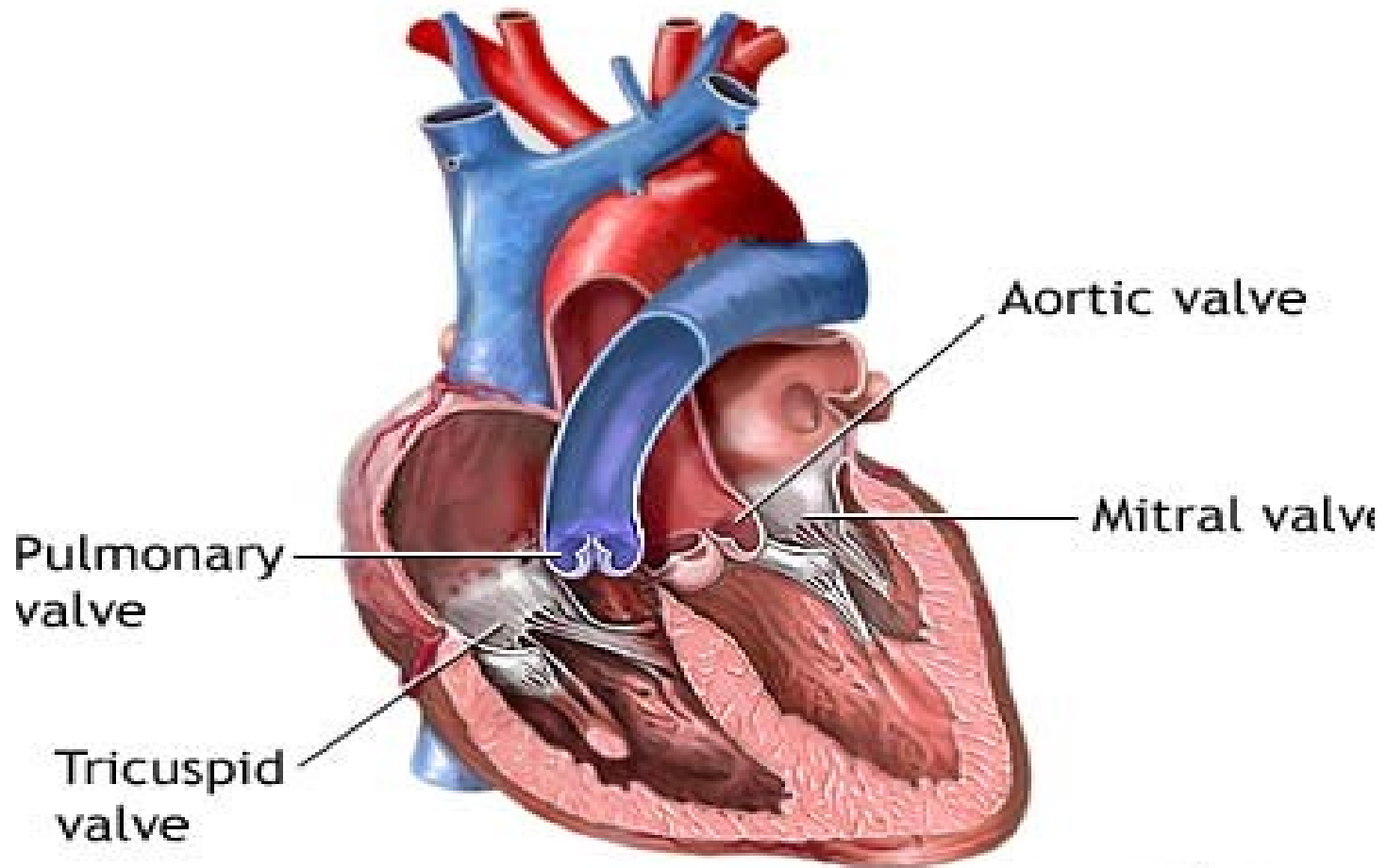
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- ✓ Maintain bed rest for duration of fever or if signs of active carditis are present → strict bed rest → until the fever , ↑ ESR, ↑ WBC, ↑ PRI return normal.
- ✓ Allow the patient to do the physical exercises which he/she can manage to do due to his/her easily fatigue.
- ✓ Counsel patient to maintain good nutrition.
- ✓ Counsel patient on importance of receiving adequate rest.
- ✓ Advice patients in long-term antibiotic therapy to prevent relapse (5 years for most adults).
- ✓ Promote good dental hygiene to prevent gingival infection

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- ✓ Instruct patient to seek treatment immediately when sore throat occur.
- ✓ Warn the patient and the parents to report any signs and symptoms of recurrent streptococcal infections:
 - sudden sore throat
 - Diffuse throat red
 - Oropharyngeal exudates
 - Swollen and tender cervical lymph node
 - $\uparrow T^0$ (38.3 - 40°C)

Valvular Heart Diseases (VHD)



ADAM

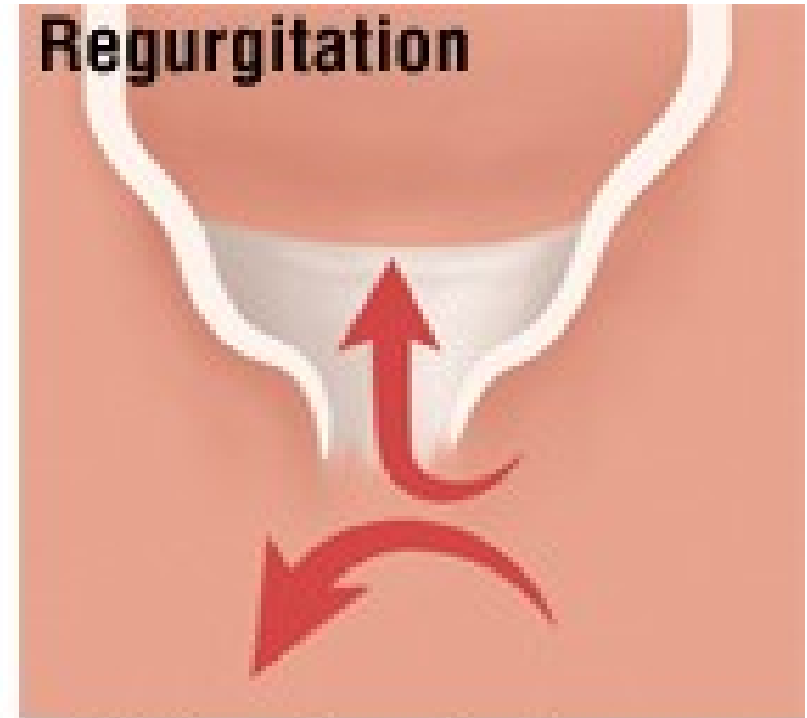
- Heart valves maintain one-way blood flow through the heart.
- The four heart valves make sure that blood always flows freely in a forward direction and that there is no backward leakage.
- When any of the heart valves do not close or open properly, blood flow is affected.
- **Stenosis**:- When valves do not open completely, results reduced flow of blood through the valve.
- **Regurgitation**:- When valves do not close completely, blood flows backward through the valve.

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Stenosis

Valve doesn't open all the way, not enough blood passes through



Regurgitation

Valve doesn't close all the way so blood leaks backwards

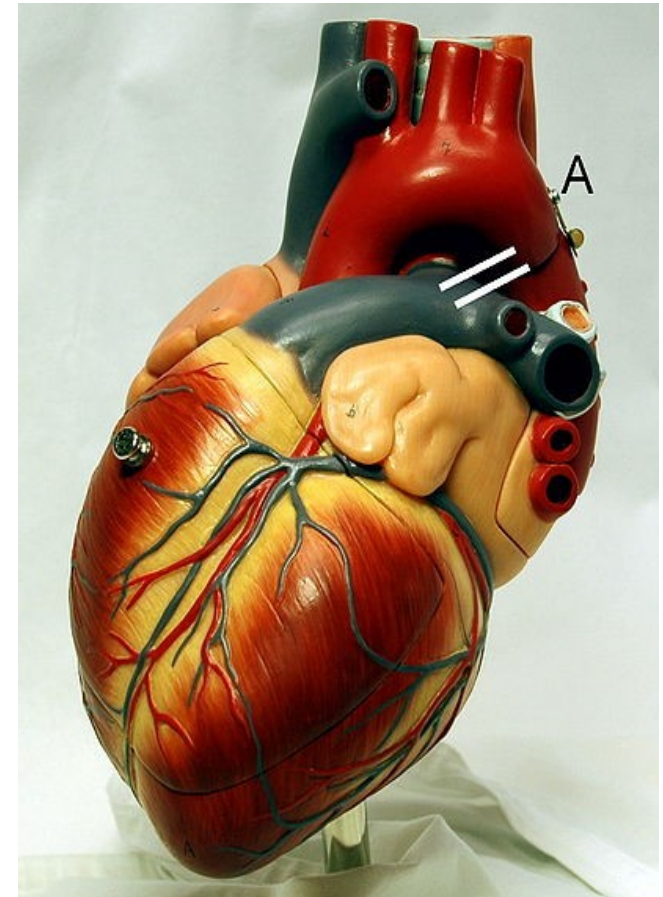
- ❖ Valvular heart disease is the commonest cardiac disease in the developing world, occurring at the younger age.
- ❖ It causes significant morbidity and mortality due to lack of appropriate preventive and therapeutic intervention.
- ❖ It accounts for 42 % of cardiac patients attending hospitals in Ethiopia.
- ❖ Aside from antibiotic prophylaxis, very little medical therapy is available, surgery is the treatment for most symptomatic lesions.
- ❖ However; surgical management is unavailable for most patients who are suffering from valvular heart diseases in Ethiopia.

➤ ***Common valvular heart disease(VHD) includes:-***

- ✓ *Aortic Stenosis (AS)*
- ✓ *Aortic Regurgitation (AR)*
- ✓ *Mitral Stenosis (MS)*
- ✓ *Mitral Regurgitation (MR)*
- ✓ *Tricuspid Regurgitation (TR)*

Congestive Heart Failure(CHF)

- **CHF** is inability of the heart to **pump** adequate amount of blood to all vital organs.
- It is the inability of the heart to pump sufficient blood to meet the needs of the tissues for **oxygen** and **nutrients**.
- Is a clinical syndrome characterized by **inadequate systemic perfusion** to meet the body's metabolic demands as a result of abnormalities of cardiac structure or function.



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- CHF is endpoint for coronary atherosclerosis, HTN, valve disease, Cardiomyopathy.
- Most common cause is cardiac muscle damage usually due to CAD & Less commonly due to valve defects.
- Responsible for 5-10% of all hospital admissions in America.
- Causes or contributes to approximately 250,000 deaths per year.
- More than half a million deaths annually in the U.S.
- Carries a 1-year mortality rate of more than 50% in patients with advanced forms of the condition, and
- Nearly \$27 billion annually in healthcare costs.

Compensatory Mechanisms in CHF

- As HF develops, the body activates neuro-hormonal compensatory mechanisms to cope with the HF and are responsible for the signs and symptoms that eventually develop and lead to clinical deterioration.

- Neurohormonal system

Stimulated by decreased perfusion □ secretion of hormones

- ✓ Epi— Increases contractility, heart rate and pressure
- ✓ Vasopressin— Mild vasoconstriction, renal water retention

- Renin-angiotensin-aldosterone system

176 Renin secretion by the kidneys causes

Classification of CHF

- ❑ CHF can be subdivided into different types:
 - ✓ Systolic heart failure
 - ✓ Diastolic heart failure
 - ✓ Left- sided (left ventricular) - forward failure
 - ✓ Right- sided (right ventricular) - backward failure
- ❖ **Systolic heart failure:** there is reduced cardiac contractility result in failure of ejecting function of the heart.
- ❖ **Diastolic heart failure :** there is impaired cardiac relaxation and abnormal ventricular filling. Result in failure of filling the ventricles.

❖ **Left- sided (left ventricular) - forward failure**

- ✓ Occurs when the left ventricle fails as an effective forward pump/Cannot eject all of the blood delivered from the right heart → back pressure of blood into the pulmonary circulation → When pressure becomes too high, the fluid portion of the blood is forced into the alveoli → pulmonary edema → decreased oxygenation capacity of the lungs.
- ✓ Left systolic dysfunction secondary to CAD is the most common cause of HF, accounting for nearly 70 percent of all cases.

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❖ **Right- sided (or right ventricular) - backward failure**

- ✓ Results from weakened right atrium which leads to venous congestion in systemic circulation. This results in fluid accumulation in the body.
- ✓ Symptoms result from inability of the heart to accept the blood coming from periphery
- ✓ Congestion is in the peripheral tissues and viscera.
- ✓ The most common cause of right heart failure is left heart failure.

▶ 179 NB: Right & Left ventricles can fail independently but usually fail together. DAMEE CVD, 2023

Etiology of CHF

- ❑ Coronary Heart Disease (60%) : Atherosclerosis of the coronary arteries is the primary cause of HF. When cholesterol and fatty deposits build up in the heart's arteries, less blood reaches the heart muscle. This damages the muscle and cause HF.
- ❑ Acute systemic hypertension -- With HTN, the chambers of the heart enlarge and weaken because they pump against high pressure.
- ❑ Valvular heart disease (especially aorta and mitral disease) --- Don't open and/or close completely □ increased workload □ Heart failure

- ❑ Infections -- Myocarditis, Bacterial endocarditis, Constrictive pericarditis
- ❑ Dysrhythmias--Tachycardias □ decreased diastolic filling time □ decreased SV □ CHF.
Example: Atrial dysrhythmias □ as much as 30% reduction in stroke volume
- ❑ Alcohol- chronic
- ❑ MI --The ischemic tissue is basically taken out of the equation, leaving a portion of the heart to do the work of the entire heart □ decreased SV □ CHF.
- ❑ Diabetes—Tend to be overweight, HTN, Hyperlipidemia & CAD

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- ❑ Pulmonary disease. Example: COPD, Pulmonary emboli
- ❑ Congenital heart diseases (Aortic stenosis & regurgitation, Mitral stenosis & regurgitation, tricuspid regurgitation)
- ❑ Thyrotoxicosis (increase BMR)
- ❑ Cor pulmonale (right sided heart failure caused by chronic lung disease)
- ❑ Cardiomyopathy (disease of heart muscle that weakens contraction)
- ❑ Anemia (increase HR as compensatory mechanism)
- ❑ Rheumatic heart disease

AHA Heart Failure Classification

- ❖ **Stage A:** Those at risk for heart failure, but who have not yet developed structural heart changes (diabetics, those with coronary disease without prior infarct).
- ❖ **Stage B:** Individuals with structural heart disease (i.e. left ventricular hypertrophy, chamber enlargement), however no symptoms of heart failure have ever developed.
- ❖ **Stage C:** Patients who have developed clinical signs & symptoms of heart failure.
- ❖ **Stage D:** Patients with refractory heart failure requiring advanced intervention (biventricular pacemakers, left ventricular assist device, or transplantation).

New York Heart Association HF Classification

- ❖ **Class I:** Ordinary physical activity does not cause fatigue, breathlessness or palpitation (includes asymptomatic left ventricular dysfunction).
- ❖ **Class II:** Patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, breathlessness or angina pectoris (symptomatically 'mild' heart failure).
- ❖ **Class III:** Although patients are comfortable at rest, less than ordinary activity will lead to symptoms (symptomatically 'moderate' heart failure).
- ❖ **Class IV:** Symptoms of congestive cardiac failure are present even at rest. Increased discomfort with any physical activity

Clinical Manifestations of CHF

Right-Sided Heart Failure

- ✓ Dependent pitting edema (peripheral edema) - hydrostatic pressure exceeds that of interstitial pressure. Fluid leaks from the capillaries into the surrounding tissues causing peripheral edema
- ✓ Jugular vein distention - Comes from back pressure building from right heart into venous circulation
- ✓ Hepatomegaly result from venous engorgement of the liver.
- ✓ Ascites (fluid in abdomen)
- ✓ Anorexia, and nausea (due to venous engorgement in abdominal organ) & Weight gain due to fluid retention
- ✓ Lungs are clear due to left ventricular pressures are normal.

Left-Sided Heart Failure

- ✓ Dyspnea upon exertion, paroxysmal nocturnal dyspnea or orthopnea.
- ✓ Cough (dry and nonproductive) due to alveolar irritation, but later productive of pink, frothy sputum
- ✓ An extra heart sound, the S3, or “ventricular gallop”
- ✓ Pale, cool extremities due to decreased tissue perfusion
- ✓ Tachycardia, decreased blood pressure, decreased peripheral pulses.
- ✓ Pulmonary edema, pleural effusion

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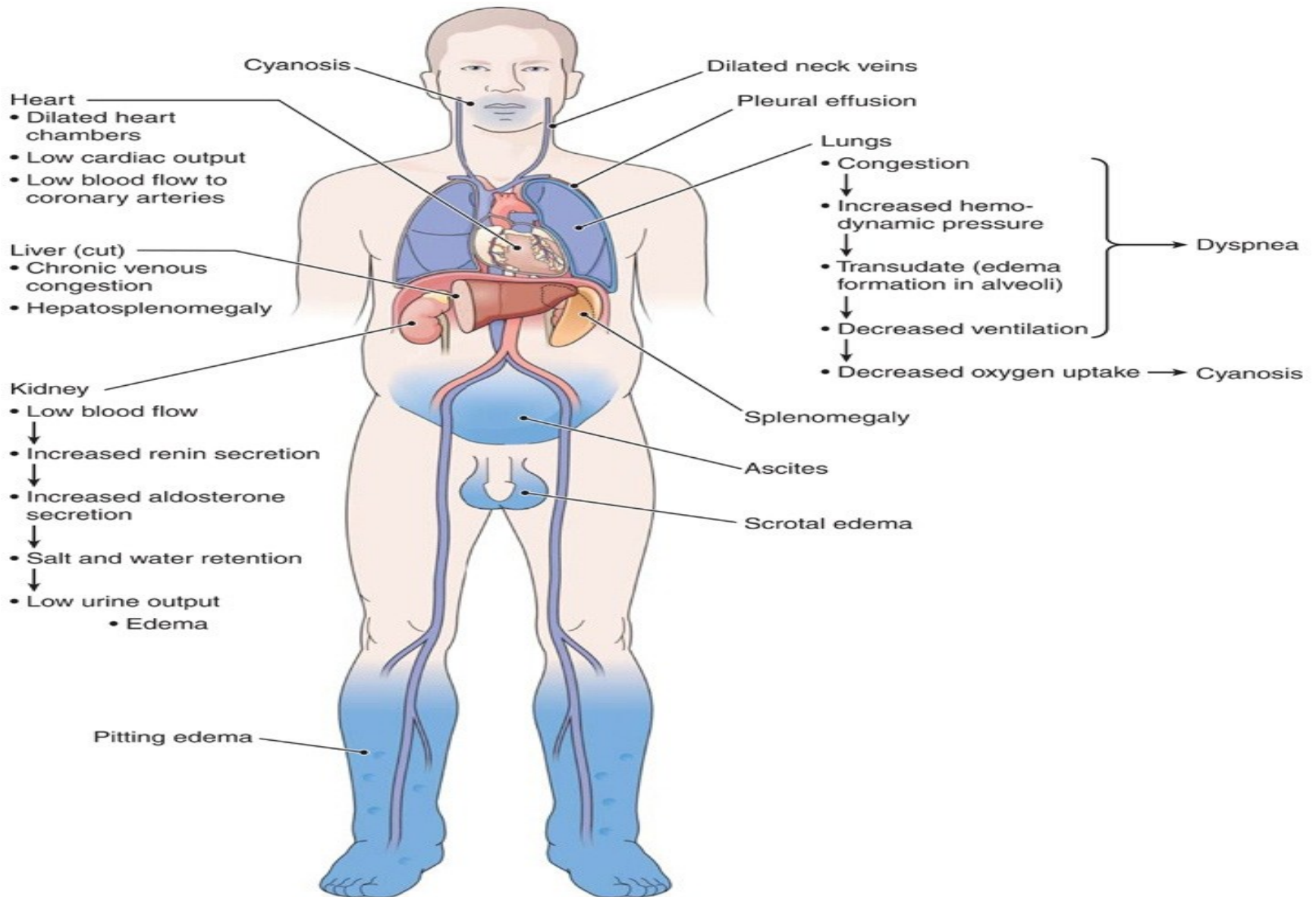
- ✓ Oliguria(<30 ml/hour) due to reduction in CO decreases blood flow to the kidneys.
- ✓ Behavioral changes (Restlessness, confusion, impaired memory, Severe apprehension, agitation, confusion due to decreased cerebral perfusion).
- ✓ Insomnia due to PND, respiratory distress, coughing, and nocturia.
- ✓ Fatigue also results from the increased energy expended in breathing.
- ✓ Remember: Left ventricular failure is a true life threatening emergency.

Physical findings of CHF

- ✓ Tachycardia and Tachypnea and signs of respiratory distress including use of accessory muscles of respiration
- ✓ Jugular venous distention (JVD) frequently is present and engorged neck veins
- ✓ Pulsus alternans (alternating weak and strong pulse indicative of depressed left ventricle [LV] function)
- ✓ Wheezing or rales may be heard on lung auscultation due to pulmonary congestion
- ✓ Apical impulse frequently is displaced laterally due to LV hypertrophy.



- ✓ Skin may be diaphoretic due to sympathetic stimulation and energy expenditure
- ✓ Cyanosis—due to low oxygen saturation levels.
- ✓ Lower extremity edema also may be noted
- ✓ Vital Signs:
 - ▶ BP—elevated (due to compensatory mechanism)
 - ▶ Pulse rate—elevated to compensate for decreased stroke volume
 - ▶ Respirations—rapid and labored



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Diagnostic Tests

- History and PE, symptoms of CHF (Framingham major and minor criteria)

Major criteria

- PND
- JVD
- Cardiomegally
- Hepatojugular reflex
bpm)
- Acute pulmonary edema
- S3 gallop & Rales

Minor criteria

- Extremity edema
- Night cough
- Dyspnea on exertion
- Tachycardia(>120 bpm)
- Pleural effusion
- Hepatomegaly

NB: At least 1 major and 2 minor criteria are required for diagnosis

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- **Chest x-ray:** may show cardiomegally, pulmonary edema, and pleural effusion.
- **Electrocardiogram (ECG/EKG):** is a nonspecific tool but may be useful in diagnosing concomitant cardiac ischemia, prior myocardial infarction (MI), cardiac dysrhythmias, chronic hypertension, and other causes of left ventricular hypertrophy.
- **Echocardiography:** may help identify valvular abnormalities, ventricular dysfunction, cardiac tamponade, pericardial constriction, and pulmonary embolus.

➤ **Laboratory studies:**

- ⊕ Serum electrolytes (Na & K)
- ⊕ RFT (BUN & Creatinine levels)
- ⊕ Thyroid function test (Thyroid-stimulating hormone)
- ⊕ Complete blood cell count, Hemoglobin levels
- ⊕ Routine urinalysis
- ⊕ Cardiac stress testing or cardiac catheterization (for coronary artery disease and cardiac ischemia are causing the HF).

Differential diagnosis

- ✓ Pulmonary disease with dyspnea
- ✓ Other conditions leading to peripheral edema like varicose vein, renal disease.
- ✓ Hepatic cirrhosis
- ✓ Enlargement of liver
- ✓ Ascites

Management of Heart Failure

A. General measures :

- ✓ Dietary sodium restriction to < 3 g/day
- ✓ Avoidance of drinking excessive amounts of fluid
- ✓ Reduce anxiety and emotional stress
- ✓ Avoid excess physical exertion (NB: exercise may be advised within the limit of the patient's cardiac function)
- ✓ Weight reduction is encouraged in obese patients
- ✓ Avoiding alcohol & Cessation of smoking
- ✓ Supplemental Oxygen if trouble breathing

Diuretics:

- ✓ First-line drugs for all patients
- ✓ Are useful in relieving congestion and reduce or prevent edema by removing fluid
- ✓ Decrease the body's retention of salt and water
- ✓ Reduces blood pressure
- ✓ However, excessive diuresis must be avoided:
- ✓ During diuresis, obtain daily weights, aiming for loss of 1–1.5 kg/d.

Thiazide (Hydrochlorothiazide):

- ✓ Initial dose 25 mg PO/day, Maximum dose 100 mg PO/day
- ✓ NB: observe for electrolyte imbalances

Loop (Furosemide or Lasix):

- ✓ Loop diuretics are most potent and unlike thiazides remain effective when GFR < 25 mL/min
- ✓ Initial dose 20-40 mg PO 1-2 X daily or 20 mg IV.
- ✓ Maximum dose 400 mg PO/day or 80 mg IV /day
- ✓ In most patients with HF, loop diuretics are preferred
- ✓ Combine loop diuretic with thiazide for augmented effect

Potassium-Sparing Diuretics:

- ✓ Is an aldosterone inhibitor/ Aldosterone antagonist
- ✓ Reduces mortality in patients with advanced heart failure.
- ✓ This drug should be reserved for patients with moderately severe or severe heart failure
- ✓ Potassium sparing diuretics are useful adjunct to reduce potassium loss; should be used cautiously when combined with ACE inhibitor to avoid hyperkalemia.
- ✓ Example: Spirinolactone
- ✓ Initial dose: 25 mg PO/day or every other day
- ✓ Maximum dose: 50 mg PO BID or higher

Cardiac glycosides (Digoxin):

- ✓ Inotropic effects on the heart (enhancement of myocardial contractility) & Negative chronotropic effects.
- ✓ Acts by inhibiting the Na^+/K^+ ATPase and increase intracellular calcium. □ This increases myocardial contractility.
- ✓ Increases the contractility of the heart □ increasing the cardiac output
- ✓ Digoxin is useful in heart failure due to marked systolic dysfunction and heart failure associated with atrial fibrillation and rapid ventricular rate.

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- ✓ Not indicated in CHF due to pericardial disease, restrictive cardiomyopathy, or mitral stenosis (unless atrial fibrillation is present).
- ✓ Digoxin is contraindicated in hypertrophic cardiomyopathy and in pts with AV conduction blocks.
- ✓ Digoxin loading dose is administered over 24 h (0.5 mg PO/IV, followed by 0.25 mg every 6 hour to achieve total of 1.0–1.5 mg). Subsequent dose (0.125– 0.25 mg QD) depends on age, weight, and renal function.
- ✓ Initial dose : 0.125 mg PO/day

▶²⁰⁰ Maximum dose : 0.25 mg PO/day DAMEE CVD,2023

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Adverse Effects:

- ❖ Digitalis toxicity which includes anorexia, nausea, and lethargy.
- ❖ Cardiac toxicity/Causes cardiac dysrhythmias of different types which are precipitated by: Hypokalemia, hypercalcemia, hypomagnesemia, hypothyroidism, or myocardial ischemia.
- ❖ Treatment of cardiac glycosides toxicity is achieved by:
 - ✓ Oral K^+ supplements as hypokalemia may increase digoxin toxicity.
 - ✓ Antiarrhythmic drugs procainamide and phenytoin

Vasodilators:

- ✓ They cause vasodilatation and reduce the peripheral resistance and after load and improve cardiac performance.

Eg. Hydralazine:

- ✓ Widens the blood vessels allowing more blood flow
Initial dose: 25 mg PO TID, Max. dose: 150 mg PO QID.

Eg. Nitrates (Isosorbide dinitrate):

- ✓ Relaxation of smooth muscle , Widens blood vessels, lowers systolic blood pressure

Initial dose: 10 mg PO TID, Max. dose: 80 mg PO TID

NB: Hydralazine and nitrates in combination are effective afterload reducing agents.

Angiotensin Converting Enzyme (ACE) Inhibitors:

- ✓ Prevent the production of the chemicals that causes blood vessels to narrow hence reduces blood pressure.
- ✓ Resulting in blood pressure decreasing and the heart pumping easier.
- ✓ ACE inhibitors may result in significant hypotension, so start at lowest dosage & the dose should be titrated to the maximum that can be tolerated.
- ✓ Initial dose: Captopril 6.25 mg PO/day or every other day
Enalapril 2.5 mg PO BID
- ✓ Maximum dose: Captopril 50-100 mg PO QID
Enalapril 10- 20 mg PO BID

Beta Adrenoreceptor blockers

- ✓ Useful by blocking the beta- adrenergic receptors of the sympathetic nervous system, the heart rate and force of contractility are decreased □could actually worsen CHF
- ✓ They are indicated to moderately severe heart failure.
- ✓ To avoid adverse effect, therapy is started with a low dose that is gradually increased over a period of weeks or months.
- ✓ Metoprolol: Initial dose 6.25 mg PO BID

Maximum dose: 75 mg PO BID

NB: Monitor for bradycardia and hypotension.

Calcium channel blockers:

- ✓ Used to dilate blood vessels & used in the presence of ischemia
- ✓ Includes: Nifedipine, Diltiazem & Verapamil
- ✓ Not found to be useful because they may precipitate or exacerbate CHF

Other medications:

- ✓ Anticoagulant if thromboembolism
- ✓ Statins if hyperlipidemia

Surgical procedures.

- ✓ Example: Mechanical heart pump such as left ventricular assist device, heart transplantation.

Treatment approach by disease stage

Stage A:

- ✓ Prescribe ACEIs especially in hypertension & Encourage regular exercise, smoking cessation, etc.

Stage B:

- ✓ All measure under stage A & add beta blockers

Stage C:

- ✓ All measures under stage A&B & Add diuretic, spironolactone, digitalis in systolic HF, Restrict dietary salt to $< 2\text{g/day}$

Stage D:

- ✓ All measures under stage A, B, and C & IV inotropic drug, mechanical assist device, heart transplantation.

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First line

- ✓ Digoxin, 0.125 -0.375 mg, P.O. QD PLUS
- ✓ Furosemide, 40-240mg, P.O. divided in to 2-3 doses QD PLUS
- ✓ Potassium chloride, 600 mg P.O. once QD OR BID

AND/OR

- ✓ Enalapril, 5-40mg P.O. once or divided into two doses QD AND/OR
- ✓ Spironolactone, 25 mg P.O.QD

Nursing diagnoses of CHF

- ✓ Impaired gas exchange
- ✓ Decreased cardiac output
- ✓ Excess fluid volume related to retention of fluid
- ✓ Activity intolerance related to imbalance between oxygen supply and demand
- ✓ Anxiety related to breathlessness

Nursing Management of CHF

- ✓ Administer prescribed medications, diuretics, digitalis, anticoagulants, vasodilators, oxygen as prescribed.
- ✓ Monitor fluid volume (Check intake and output)
- ✓ Weigh daily
- ✓ Provide a low- sodium diet.
- ✓ Auscultate lung sounds.
- ✓ Determine degree of JVD.
- ✓ Assess dependent edema.
- ✓ Monitor vital signs, FVO and dehydration.
- ✓ Psychological support/controlling anxiety and emotional support

- ✓ Position patient in semi-Fowler's position to ease breathing/provide pillow.
- ✓ Call the physician or nurse practitioner if experiencing fluid retention, such as a weight gain in 1 to 2 days.
- ✓ Monitoring blood volume (measure intake and output).
- ✓ Promotion of rest/don't make these patient's walk (try to get them to sit still if they appear agitated and hypoxic).
- ✓ Tell the patient:
 - Eat foods low in sodium to avoid fluid retention (low dietary intake of sodium (<1500 mg/day)).
 - Raise legs when sitting to lessen dependent edema.

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- ✓ Monitoring and managing potential complication:

- ❖ **Vasodilators:**

- ✓ Observe for hypotension

- ❖ **Angiotensin-Converting Enzyme (ACE) Inhibitors:**

- ✓ Observe for symptomatic hypotension, increased serum K, cough, and worsening renal function

- ❖ **Beta-adrenergic Blocking Agents (beta-blockers):**

- ✓ Observe for decreased heart rate, symptomatic hypotension and fatigue

❖ **Diuretics:**

- ✓ Observe for electrolyte abnormalities, renal dysfunction, diuretic resistance, and decreased BP. Carefully monitor I&O and daily

❖ **Digitalis:**

- ✓ Observe for digitalis toxicity (dysrhythmia), blurred vision, bradycardia, confusion, and

❖ **Calcium Channel Blockers:**

- ✓ Observe for symptomatic hypotension, drowsiness, or dizziness.

- ❖ **Others:** Cardiogenic shock, thromboembolism, pericardial effusion and cardiac tamponade.

Hypertension

- Blood pressure is a pressure exerted **by the blood** on **the blood vessels** as it flows through the blood vessels.
- Blood pressure is the product of **cardiac output** multiplied by **peripheral resistance**.
- **Cardiac output** is the product of the HR X stroke volume.
- In normal circulation, pressure is exerted by the flow of blood through the heart and blood vessels.



- Hypertension is intermittent or sustained elevation in systolic or diastolic blood pressure.
- A systolic blood pressure greater than 140 mm Hg and diastolic pressure greater than 90 mm Hg based on the average of two or more accurate blood pressure measurements taken during two or more contacts with a health care provider.
- HTN=(SBP>140mmHg)& (DBP>90mmHg)
- Hypertension is sometimes called the “**silent killer**” because people who have it are often symptom free

Blood pressure classification for adults

Blood pressure category	Systolic blood pressure (mmHg)		Diastolic blood pressure (mm Hg)
Optimal	< 120		< 80
Normal	120–129	and	80–84
High normal	130–139	and	85–89
Stage 1 HPN	140–159	or	90–99
Stage 2 HPN	160–179	or	100–109
Stage 3 HPN	≥ 180	or	≥ 110

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- If there is a disparity in the category between the systolic and diastolic pressures, the higher value determines the severity of hypertension
- The prevalence dramatically increases in patients older than 60 years.
- The age-adjusted prevalence of hypertension is slightly higher in men than in women.
- The prevalence in women is closely related to age, with substantial increase occurring after age 50.
□ This may be related to hormonal changes associated with menopause

Hypertensive crisis

- It is as severe hypertension characterized by diastolic blood pressure (DBP) greater than **130 mmHg**.
- Blood pressure elevation to such degree can cause vascular damage, encephalopathy, retinal hemorrhage, renal damage and death.
- About 1 -2% of the hypertensive population develop this complication.
- May occur in patients whose hypertension has been **poorly controlled**, whose hypertension **has been undiagnosed**, or in those who have abruptly **discontinued their medications**.
- It is categorized into two: **hypertensive emergency & hypertensive urgency**.

Hypertensive emergency

- ✓ A situation in which blood pressure is severely elevated and there is **evidence of actual or probable target organ damage**.
- ✓ Acute, life-threatening blood pressure elevations that require prompt treatment
- ✓ Blood pressure in which there is acute impairment of an organ system (CNS, CVS, Renal).
- ✓ The blood pressure should be lowered aggressively **over minutes to hours to** prevent or limit target organ damage.

Hypertensive urgency:

- ✓ A situation in which blood pressure is severely elevated but there **is no evidence of target organ damage**
- ✓ Hypertension in which BP is high and there is potential risk but not yet caused acute end-organ damage.
- ✓ The rapidity with which BP must be reduced is controversial but, these patients require BP control **over several days to weeks.**

□ **Hypertension can result from :-**

- Change (increase) in cardiac output
- Change (increase) in peripheral resistance, or both.
- **Based on the etiology/risk factors, HTN can be divided in to two major types:**

□ **Primary Hypertension/essential hypertension** is elevation of blood pressure from an unknown cause (accounts 90%).

□ **Secondary Hypertension** -- has a known cause (10%)

□ **Isolated systolic hypertension** (systolic >160,

220
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diastolic <90) most common in elderly patients.

Risk factors for primary hypertension

❑ **Non-modifiable risk factors**—those that cannot be changed and includes:

- ♣ Family history of hypertension
- ♣ Age (As age increases risk increases)
- ♣ Gender (Male > Female)
- ♣ Race
- ♣ Diabetes mellitus

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- ❑ **Modifiable risk factors—those** that can be changed— include:-
 - ☛ Decreased activity levels
 - ☛ Smoking
 - ☛ High sodium dietary intake
 - ☛ Prolonged, recurrent stress
 - ☛ Obesity
 - ☛ Excessive caffeine, alcohol
 - ☛ Saturated fats
 - ☛ Oral contraceptives use

Clinical Manifestations

- ✓ Usually asymptomatic / **"Silent killer"**. Disease symptoms and organ damage caused by hypertension are not evident **until 10-15 years after the disease has started**
- ✓ Headache, Bloody nose (epistaxis)
- ✓ Severe anxiety, or shortness of breath
- ✓ Dizziness (a feeling of unsteadiness), Blurred vision
- ✓ **Pathologic** changes in the kidneys such as
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increased Blood Urea Nitrogen (BUN) and

■ **Retinal changes such as:-**

- ✓ Hemorrhages
- ✓ Exudates (fluid accumulation)
- ✓ Arteriolar narrowing, and
- ✓ Cotton-wool spots (small infarctions) occur.
- In severe hypertension, papilledema (swelling of the optic disk) may be seen.

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■ Cerebrovascular involvement may lead to a stroke or transient ischemic attack (TIA), manifested by:

- ✓ Alterations in vision or speech
- ✓ Dizziness
- ✓ Weakness, a sudden fall, or
- ✓ Transient or permanent paralysis on one side (hemiplegia).

Diagnostic findings

History: Patient's Relevant History

- Duration of hypertension
- Previous therapies: responses and side effects
- Family history of hypertension and cardiovascular disease
- Dietary and psychosocial history
- Other risk factors: weight change, dyslipidemia, smoking, diabetes, physical inactivity
- Sign and symptoms

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- Blood pressure readings higher than 140/90 mmHg on **at least three occasions.**
- **EKG or chest x-ray** -- Ventricular hypertrophy
- Blood test to look for associated cardiovascular risks.
- **High cholesterol**—often associated with hypertension.
- **Check electrolytes for imbalance**—sodium, potassium, chloride
- Monitor BUN and creatinine for renal function,
- Fasting blood glucose, total cholesterol, HDL and LDL (often computed) cholesterol, triglycerides

Treatment of Hypertension

Objectives:-

- Detection and management of other cardiovascular risk factors
- Detection and management of target organ damage
- Prevention of target organ damage
- Decreasing the risk of CVA, CAD, heart failure, renal disease, and other long-term sequelae of hypertension
- Decrease the side effects of medications
- **Achieve target blood pressure** ($< 140/90\text{mmHg}$), but in patients having diabetes with proteinuria and chronic kidney disease ($< 130/80\text{ mmHg}$)

Non pharmacologic management

- **Smoking cessation:** Complete cessation of smoking
- **Physical activity:** At least 30 minutes of moderate intensity activity 5-7 days per week
- **Weight reduction:** BMI 18-24kg/m², waist circumference < 102cm men, < 88cm women.
- **Dietary recommendations:** emphasize fruits, vegetables, low-fat dairy products, fiber, whole grains, and protein sources that are reduced in saturated fats and cholesterol
- Reduce caffeine intake
- Avoid stress, emotion and smoking

-
- **Reduce salt intake:** about 1 tsp of table salt.
 - **Alcohol consumption:** limited to two drinks or less per day (one standard drink)
 - 1 bottle (341 mL) of 5% beer or,
 - 1 glass (150 mL) of 12% wine or,
 - 1.5 oz (45 mL) of 40% spirits
 - Health-promoting lifestyle modifications

10 commandments of Hypertension

- ✓ Reduce salt intake
- ✓ Minimize stress
- ✓ Reduce obesity
- ✓ Control alcohol intake
- ✓ Exercise regularly
- ✓ Stop smoking
- ✓ Eat more fruits and vegetables
- ✓ Control cholesterol level
- ✓ Enjoy in recreational activity
- ✓ Consult your health care provider

Medical management

1. Diuretics
2. Sympatholytic drugs
3. Vasodilator drugs
4. Calcium antagonist drugs
5. Angiotensin-converting enzyme inhibitor

- ❑ **Diuretic Treatment:** furosemide, spironolactone, HCT
- Should be cornerstone of most antihypertensive regimes.
- Thiazides are preferred in patients with adequate renal function.
- Thiazides preferred over loop diuretics because of longer duration of action; however, loop diuretics are more potent and are used in patients with reduced renal function (when GFR < 25 mL/ minutes)
- Diuretics can be used alone or in combination with other antihypertensive drugs
- **S/E:** Excessive loss of fluid, sodium, and potassium

Sympathetic Blocking Drugs:

- ✓ Alpha blockers lower BP by vasodilation. Eg. Prazosin
- ✓ Beta blockers lower BP by decreasing heart rate and cardiac output: eg. propranolol, metoprolol, atenolol
- ✓ Beta blockers need to be used cautiously—fetal hypoglycemia and low birth weights have been reported.
- ✓ Contraindicated in bronchial asthma, COPD, bradycardia & heart block.

□ Vasodilators:

- ✓ Decrease the muscular tone and contractile function of blood vessels
- ✓ Hydralazine is a potent vasodilators that must be used with diuretics and sympathetic blocking drugs

Calcium Antagonists:

- ✓ Block the influx of calcium into the heart and arterial blood vessels, reduce heart rate and relax blood vessels.
- ✓ Verapamil and diltiazem act on both the heart and blood vessels to lower blood pressure.
- ✓ Nifedipine and other calcium blockers lower blood pressure only by vasodilation.
- ✓ All have negative inotropic effects and should be used cautiously if LV dysfunction is present.
- ✓ Verapamil, and to a lesser extent diltiazem, can result in bradycardia and AV block, so combination with beta blockers is generally avoided.

Angiotensin-Converting Enzyme Inhibitors (ACEIs):

- ✓ ACEIs inhibit the formation of angiotensin which is a potent vasoconstrictor.
- ✓ ACEIs decrease the release of aldosterone which retains sodium and water : eg. enalapril, captopril
- ✓ May be used as monotherapy or in combination with beta blockers, calcium antagonists, or diuretics
- ✓ The ACEIs can be used with thiazide and loop diuretics, but not potassium-sparing diuretics or should be used cautiously to prevent hyperkalemia.
- ✓ ACE inhibitors are contraindicated in pregnancy and advanced renal failure.

Selection of antihypertensive drugs

Mild case:

- ✓ Usually respond to a single drug. Eg. Diuretics, beta blockers, ACEIs or Ca channel blockers.

Moderate case:

- ✓ Diuretics plus one of the above drugs or If 3 drugs required, diuretics + sympatholytics + ACEIs or Ca channel blockers can be used.

Severe cases:

- ✓ Use loop diuretics, hydralazine, In elderly use thiazide and Ca blockers.

Treatment approach

Non -Emergency conditions

❑ First line

- ✓ Hydrochlorothiazide, 12.5- 25 mg/day, P.O. QD

❑ Alternatives

- ✓ Nifedipine, 10 – 40 mg, P.O., .BID or
- ✓ Atenolol, 25-100mg P.O.QD or
- ✓ Enalapril, 2.5- 40mg P.O., once OR divided into two doses QD

Treatment of hypertensive crisis

Hypertensive Urgency

- Start either a low dose of a calcium channel blocker (Nifedipine) OR
- ACE inhibitor (captopril or Enalapril) or Beta blocker .
- Furosemide 20 - 40mg (PO or IV) can be added to the above agents.
- If patient is reliable follow up can be **made every one to two days**. If not reliable, admit the patient.
- Avoid rapid drop in blood pressure

Hypertensive Emergencies

- ✓ Use rapidly acting antihypertensive intravenously.

Example: Hydralazine, 5-10 mg initial dose, repeated every 20 to 30 minutes (with maximum dose of 20 mg) should be given until the mean arterial blood pressure is reduced by 25% (within minutes to 2 hours), then towards **160/100** mm Hg within 2-6 hours.

- ✓ N.B. Additional Furosemide, 40 mg I.V. can be used according to blood pressure response.

Potential Complications

- ✖ Left ventricular hypertrophy
- ✖ Myocardial infarction
- ✖ Heart failure
- ✖ Cerebrovascular accident (CVA, stroke, or brain attack)
- ✖ Renal insufficiency and failure
- ✖ Retinal hemorrhage

Nursing process

Nursing Diagnosis

- ✓ Excess fluid volume
- ✓ Deficient knowledge regarding the relation between the treatment regimen and control of the disease process
- ✓ Noncompliance with therapeutic regimen related to side effects of prescribed therapy

Nursing Interventions

- ✓ Monitor blood pressure with multiple readings:- lying, sitting, and standing, bilateral both arms.
- ✓ Record fluid intake and output.
- ✓ Reduce stress by providing a quiet environment.
- ✓ Provide patient and family teaching:-
- ✓ No smoking
- ✓ Change to a low-sodium and low-cholesterol diet
- ✓ Reduce alcohol intake

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- ✓ Reduce weight—decreased risk for obesity, better BP control with better regular exercise
- ✓ Discuss the Side effects of medications
- ✓ Discuss the importance of regular blood pressure monitoring
- ✓ Discuss the importance of lifelong medical follow up examination
- ✓ Teaching patients self-care
- ✓ Monitoring and managing potential complications

Shock

- Inadequate perfusion and oxygenation of cells
- “A significant reduction in tissue perfusion, Resulting in poor oxygen delivery to these tissues”
- Without treatment, inadequate blood flow to the cells results in poor delivery of oxygen and nutrients, cellular hypoxia, and cell death that progresses to organ dysfunction and eventually death.
- Adequate blood flow to the tissues and cells requires an

- A. Adequate cardiac pump,
- B. Effective vasculature or circulatory system, and
- C. Sufficient blood volume.

↓ Cardiac output /ineffective circulating, blood volume



hypotension



systemic hypo-perfusion



cellular hypoxia/anoxia



reversible cell injury

(continued anaerobic glycolysis & lactic acid
accumulation)



irreversible cell injury



cell death



Stages of Shock

Non-Progressive Stage:

- ✓ Also referred to as warm shock or compensated shock.
- ✓ It is characterized by rapid compensation for diminished tissue perfusion by various homeostatic mechanisms.
- ✓ Reflex neurohumoral mechanisms activated □ maintain CO & BP.

Examples:

Catecholamines;

- ✓ Renin-angiotensin-aldosterone system;
- ✓ Sympathetic stimulation,
- ✓ Hypothalamic release of adrenocorticotrophic hormone (ACTH).

Progressive Stage:

- ✓ The compensatory mechanisms become overwhelmed and signs and symptoms of organ dysfunction appear.
- ✓ Characterized by tissue hypo-perfusion & onset of worsening circulatory and metabolic imbalances.
- ✓ Wide spread tissue hypoxia □ anaerobic respiration □ metabolic acidosis □ blunts vasomotor response □ peripheral pooling of blood
- ✓ Vital organs begin to fail □ confusion/decreased urinary output.

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Irreversible Stage:

- ✓ Cellular & tissue injury that is irreversible
- ✓ Despite all efforts, inevitably culminates in death
- ✓ Term only applied post-mortem
- ✓ Cardinal features of shock include hypotension, oliguria, abnormal mental status, metabolic acidosis, and, in some

Classification of shock

Cardiogenic shock

- Failure of the heart to pump sufficient blood to tissues
- Decreased pumping capability of heart
- Decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume
- **Due to:** left heart failure, ischemia, MI, arrhythmias, pulmonary embolism, myocardial/pericardial infection

Hypovolemic shock

- ✓ Loss of blood volume due to: hemorrhage, burns, profuse sweating, excessive urination, vomiting, diarrhea.
- ✓ Internal sequestration (ascites, pancreatitis, intestinal obstruction).

Anaphylactic shock

- ✓ Systemic **vasodilation** due to severe allergic reaction
- ✓ Occurs due to: severe reaction to allergens (penicillin, nuts, shellfish, animal proteins).
- ✓ IgE mediated hypersensitivity response → systemic vasodilation & ↑ vascular permeability → **Hypotension**

Septic shock

- ✓ Vasodilation due to severe systemic microbial infection - usually gram -ve bacteria (can be gram +ve or fungal)
- ✓ Multiple organ dysfunction as a result of pathogenic organisms in the blood

Sign & symptoms of shock

- Shock is a collection of signs and symptoms, many nonspecific:
- **↑ pulse but weak + ↓ BP = classic clinical signs**
- Skin: pale, cool, or clammy, Cyanosis may be apparent
- Respiratory: breathing rapid (tachypnea) and shallow
- CV: low BP, low cardiac output, Tachycardia HR > 100
- Neurologic: restlessness, anxiety, lethargy, confusion, agitation, delirium & coma
- Dry Mucous membranes
- Low urine output <0.5 ml/day and metabolic acidosis common.

- Jugular veins are flat in hypovolemic shock; jugular venous distention(JVD)suggests cardiogenic shock.
- Fever and chills usually accompany septic shock
- Skin lesion may suggest specific pathogens in septic shock

Empiric Criteria for Shock

- ❑ 4 out of 6 criteria have to be met
- ✓ Ill appearance or altered mental status
- ✓ Heart rate > 100
- ✓ Respiratory rate > 22 (or $\text{PaCO}_2 < 32 \text{ mmHg}$)
- ✓ Urine output $< 0.5 \text{ ml/d}$
- ✓ Arterial hypotension > 20 minutes duration
- ✓ Lactate > 4 (Lactate is increased in shock, predictor of mortality & can be used as a guide to resuscitation)

Treatment of shock

- Tissue perfusion/ treatment must be restored immediately.
- Initial treatment includes basic life support while identifying the underlying causes
- Maintain ABC of life support: airway, breathing, circulation
- Oxygen therapy is rapidly started at first contact with the patient

➤ Indications of endo-tracheal intubation (ETT) and

...

- ❖ There are 3 categories of drugs that play a role in treating shock:
 - ✓ Vasoconstrictors
 - ✓ Cardiotonic drugs
 - ✓ Fluid replacement agents
 - ✓ Antibiotic drugs

Vasoconstrictors

Adrenaline

- ❑ A direct myocardial stimulation that increases the strength of ventricular contraction (positive inotropic action).
- ❑ An increased heart rate (positive chronotropic action)
- ❑ Vasoconstriction in many vascular beds—especially in the precapillary resistance vessels of skin, mucosa, and kidney—along with marked constriction of the veins
- ❑ **First line for anaphylactic shock.**
- ❑ Dosage form: Injection, 0.1% in 1 ml ampoule

Cardiotonic drugs

- ❖ Cardiotonic drugs are used to ↑ the force of contraction and increase cardiac output.
- ❖ They are used in cardiogenic shock.

Digoxin

- ✓ Acts by inhibiting the Na^+/K^+ ATPase and increase intracellular calcium. □ This increases myocardial contractility.
- ✓ Increases the contractility of the heart □ increasing the cardiac output

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Dobutamine

- ✓ A β_1 adrenergic agent that is often drug of choice for short term treatment of shock.
- ✓ Has the ability to cause heart to beat more forcefully without significantly increasing heart rate.
- ✓ Increase in cardiac output assists in maintaining blood flow to vital organs
- ✓ Dosage: 2.5-15 micrograms/kg/min IV, diluted in 200mL D5W

Dopamine

- ✓ Mechanism of action is dependent on dose
- ✓ Low doses: selectively stimulates β_1 receptors in kidneys that cause vasodilation and an $\uparrow$ blood flow to kidneys (used in treating hypovolemic and cardiogenic shock).
- ✓ Moderate doses: stimulates β_1 receptors, causing heart to beat with more force and increasing cardiac output.
- ✓ Higher doses: stimulates alpha receptors causing vasoconstriction and raising blood pressure.
- ✓ Dosage: 2-20 mcg/kg/min IV diluted with dextrose 5% in water, or in sodium chloride solution 0.9%

Fluid replacement

- Used in hypovolemic shock as an initial treatment.
- Used to replace blood or other fluids lost during hypovolemic shock.
- If not due to hemorrhages: Infusion of fluid (Normal Saline or Ringer lactate) 20ml/kg fast.
- If due to hemorrhage, transfusion of packed RBC or whole blood 20ml/kg over 4 hours.
- N.B: Fluid administration in Cardiogenic shock has to be under extreme caution !!

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- Fluid replacement agents are generally placed into the 3 categories: blood, colloids, crystalloids

Blood

- ✓ Whole blood indicated for treatment of acute, massive blood loss when there is a need to replace plasma volume and supply RBC's.

Colloids

- ✓ Used when up to 1/3 of adult blood volume is lost
- ✓ Colloids are proteins that stay suspended in the blood for a long period and draw water from the body's cells and tissues into the blood vessels
- ✓ Colloids include: normal serum albumin, plasma protein fractions, dextran, etc.

Crystalloids

- ✓ IV solutions that contain electrolytes in amounts resembling those of natural plasma
- ✓ Unlike colloids, crystalloid solutions leave the blood and enter cells
- ✓ Used to replace fluids lost, and to increase urine output
- ✓ Includes: normal saline, lactated Ringer's, hypertonic saline, 5% dextrose in water,

Antibiotics agents

- ✓ Used for the patients with septic shock as initial treatment.
- ✓ Start antibiotics within an hour for those severely sick
- ✓ Don't wait for the cultures – treat empirically then change if needed.

Other drugs

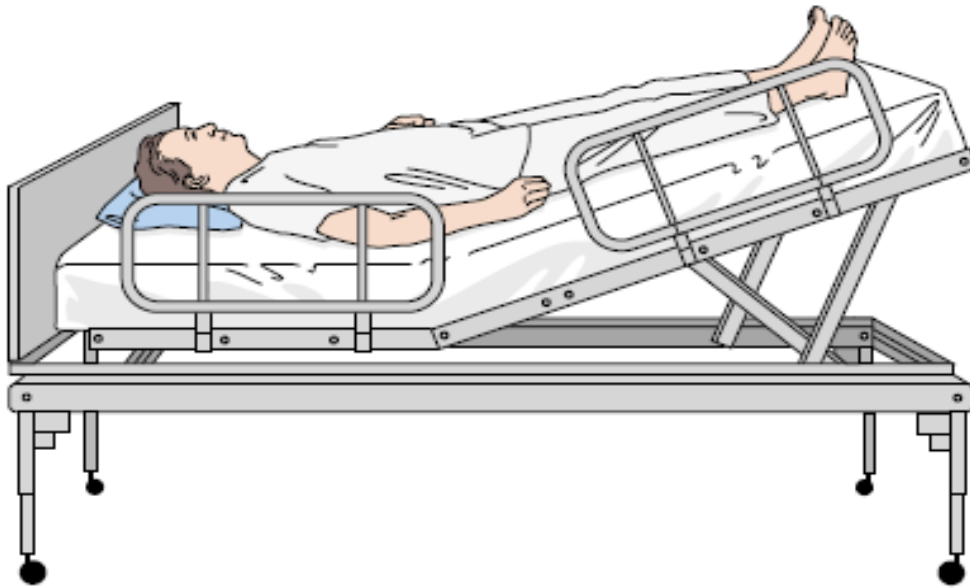
- ✓ Anti allergic for anaphylactic shock. Eg. Antihistamine may help counter histamine-mediated vasodilation and bronchoconstriction
- ✓ Corticosteroids (hydrocortisone) – may help shorten reaction
- ✓ Bronchodilators may ease breathing

Prognosis

- ❑ Prognosis varies with origin of shock & duration.
- ✓ 80% young, otherwise healthy patients survive hypovolemic shock with appropriate management
- ✓ 75% mortality rate associated with severe M.I, cardiogenic shock or gram -ve shock,

Nursing interventions

- ❖ Position the patient in shock position (T₁)



Proper positioning (Trendelenburg) for the patient who shows signs of shock. The lower extremities are elevated to an angle of about 20 degrees; the knees are straight, the trunk is horizontal, and the head is slightly elevated.

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- ❖ Make certain that there is an adequate airway. Maintain adequate oxygenation – oxygen therapy/ endo-tracheal intubation and ventilatory support.
- ❖ Start an IV infusion. Rapidly infuse IV fluids (normal saline or Ringer's lactate) initially at the rate of 1 L in 15–20 minutes; Give at least 2 L of these fluids in the first hour.
- ❖ Note: A more rapid rate of infusion is required in the management of shock resulting from bleeding. Aim to replace 2–3 times the estimated fluid loss.

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- ❖ Collect blood for estimation of hemoglobin, immediate cross-match and bedside clotting, just before infusion of fluids
- ❖ Insert a Foley catheter into the urinary bladder to obtain accurate measurements of urinary output
- ❖ Increase fluid rate if urine output is less than 30 ml/hour.
- ❖ Monitor the patient continuously
- ❖ ECG monitoring
- ❖ Relieve pain & agitation

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- ❖ Monitor every 15 minutes for blood pressure. If systolic lower than 80 mmHg, then increase oxygen flow rate.
- ❖ Be alert for signs of fluid overflow. These include, but are not limited to, crackles in the lungs and dyspnea.
- ❖ Assess for cool, pale, clammy skin, indicating hypovolemic shock.
- ❖ Explain to the patient what caused the hypovolemia and how to avoid a recurrence & the purpose of the treatment.

Assignment (10%)

1. Define shock & explain its pathophysiology (1 point)
2. Describe the three stages of shock (1 point)
3. Explain the four classification of shock (2 points)
4. Mention the common sign & symptoms of shock (1 point)
5. List & explain the diagnostic tests of shock (1 point)
6. Discuss the management of shock in terms of each classification of shock (3 points)
7. Explain nursing interventions for the patients with shock (1 point)

Vein disorders

- A. Phlebitis
- B. Deep Vein thrombosis (Thrombophlebitis)
- C. Varicose Veins

Phlebitis

Definition

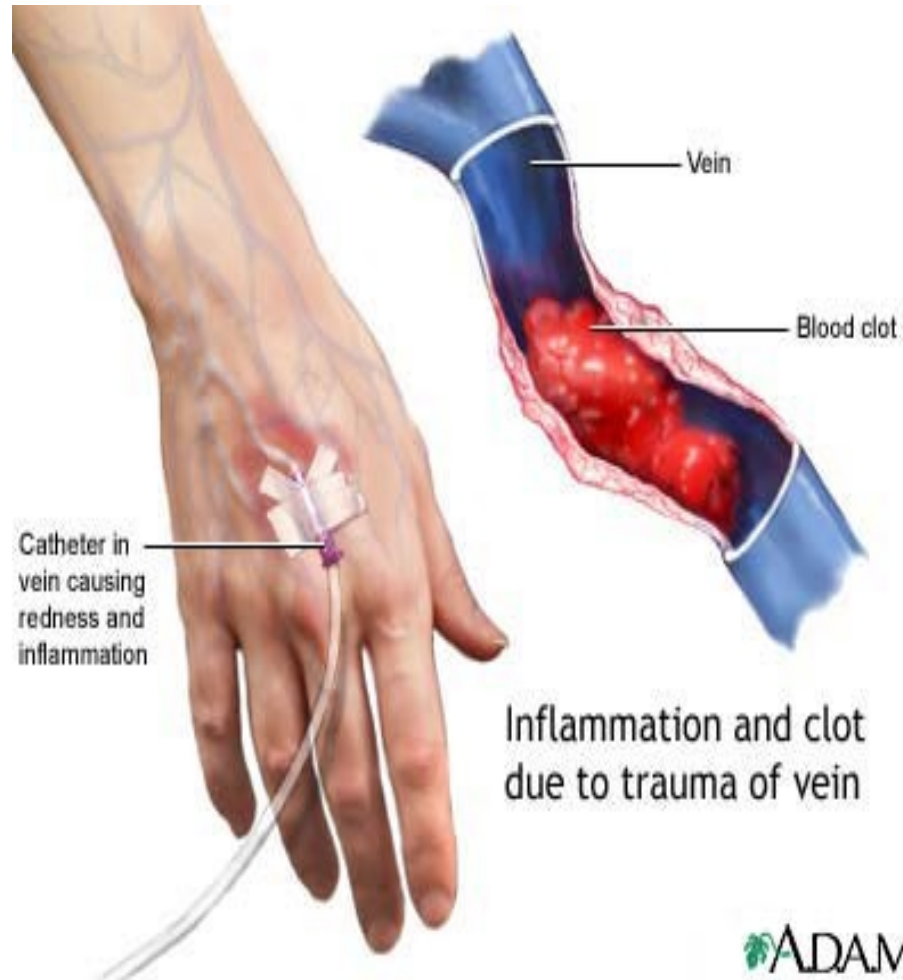
- ✓ Phlebitis is the inflammation of **a vein** usually occurs in legs, but it may occur in an arm.
- ✓ Phlebitis can occur in both the surface (superficial) or deep veins.
- ✓ **Superficial phlebitis** affects veins on the skin surface.
- ✓ The condition is rarely serious and, with proper care, usually resolves rapidly.

Etiology of phlebitis

- ✓ **Duration** of IV therapy ($\uparrow$ risk if > 72 hours)
- ✓ The type of needle access (**plastic catheter** has higher risk than stainless steel needle)
- ✓ Types of **fluid** used ($\uparrow$ osmolality and particulate matter is associated with high incidence of phlebitis)
- ✓ Insertion **site** (veins of the arm are less likely to develop phlebitis than veins of the leg)

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- ✓ **Bacterial:** Pathogenic organisms can gain access and stimulate inflammation.
- ✓ **Chemical:** caused by irritating or vesicant solutions.
- ✓ **Mechanical:** physical trauma from the skin puncture and movement of the cannula into the vein during insertion
- ✓ Medications including , Olanzapine, antidepressants
- ✓ Genetic as it is known to run in families.
- ✓ Alcohol abuse



Clinical Manifestation

- ❑ → Redness following the involved vein
- ❑ → Cord like with palpation
- ❑ → Warm to touch
- ❑ → Indurated or swollen
- ❑ → Tenderness
- ❑ → A low-grade fever may occur
- ❑ → Impaired mobility of affected part

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Diagnosis

- Patient complaint (clinical manifestation)
- Physical examination

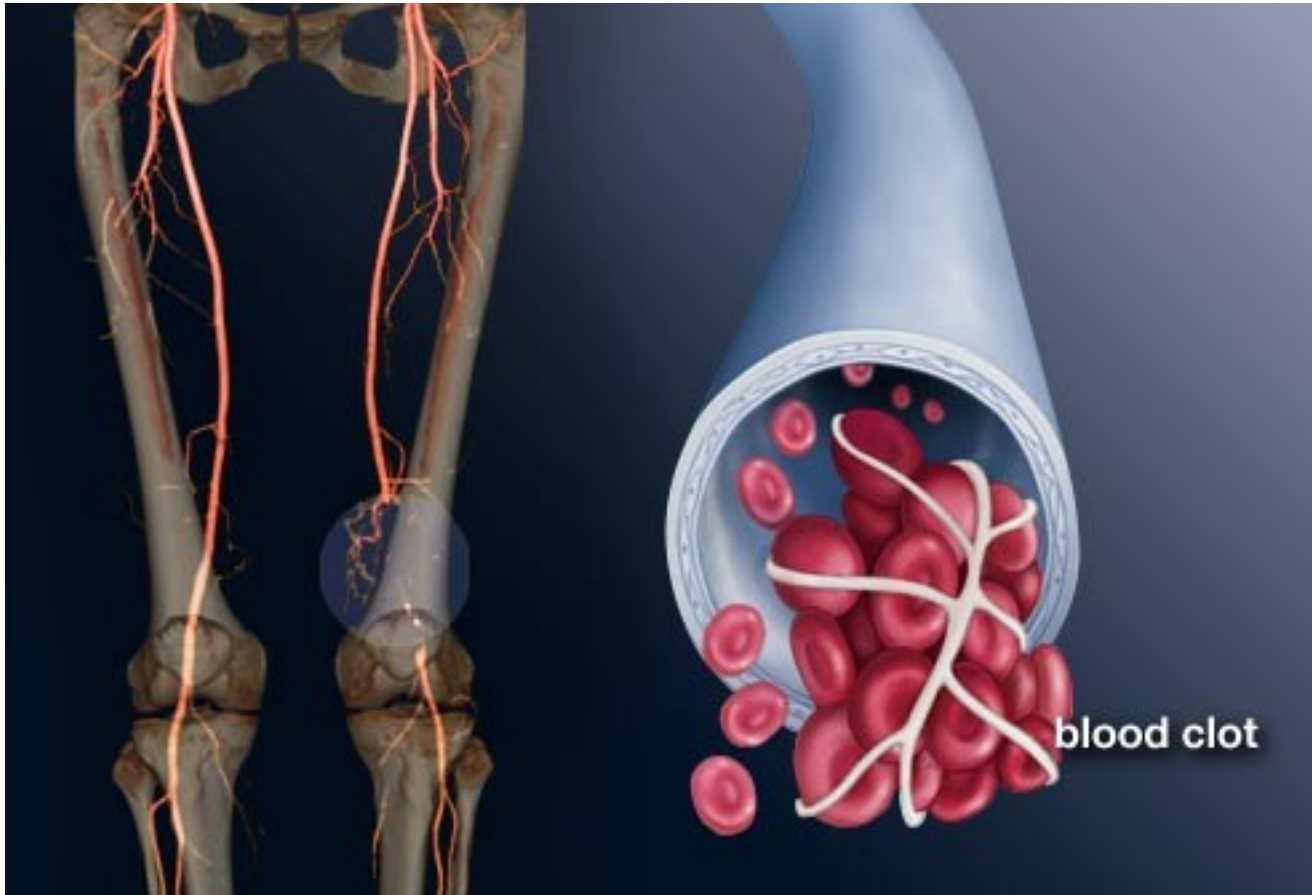
Treatment

- ▶ Moist heat application, elevating the extremity
- ▶ Analgesics & Non steroidal anti-inflammatory agents
- ▶ Antibiotics incase of infectious phlebitis
- ▶ Heparin to prevent thrombus formation
- ▶ Rarely **venoectomy**

Nursing intervention

1. Rotate IV sites every 48 - 72 hours
 - Use smallest gauge and length cannula possible
 - Use stainless steel needles
 - Use veins in upper extremities
 - Use strict aseptic technique
2. Maintain IV site
 - Fix cannula and tubing securely
 - Apply sterile dressing
3. Dilute IV push medications as much as possible

Deep Vein thrombosis

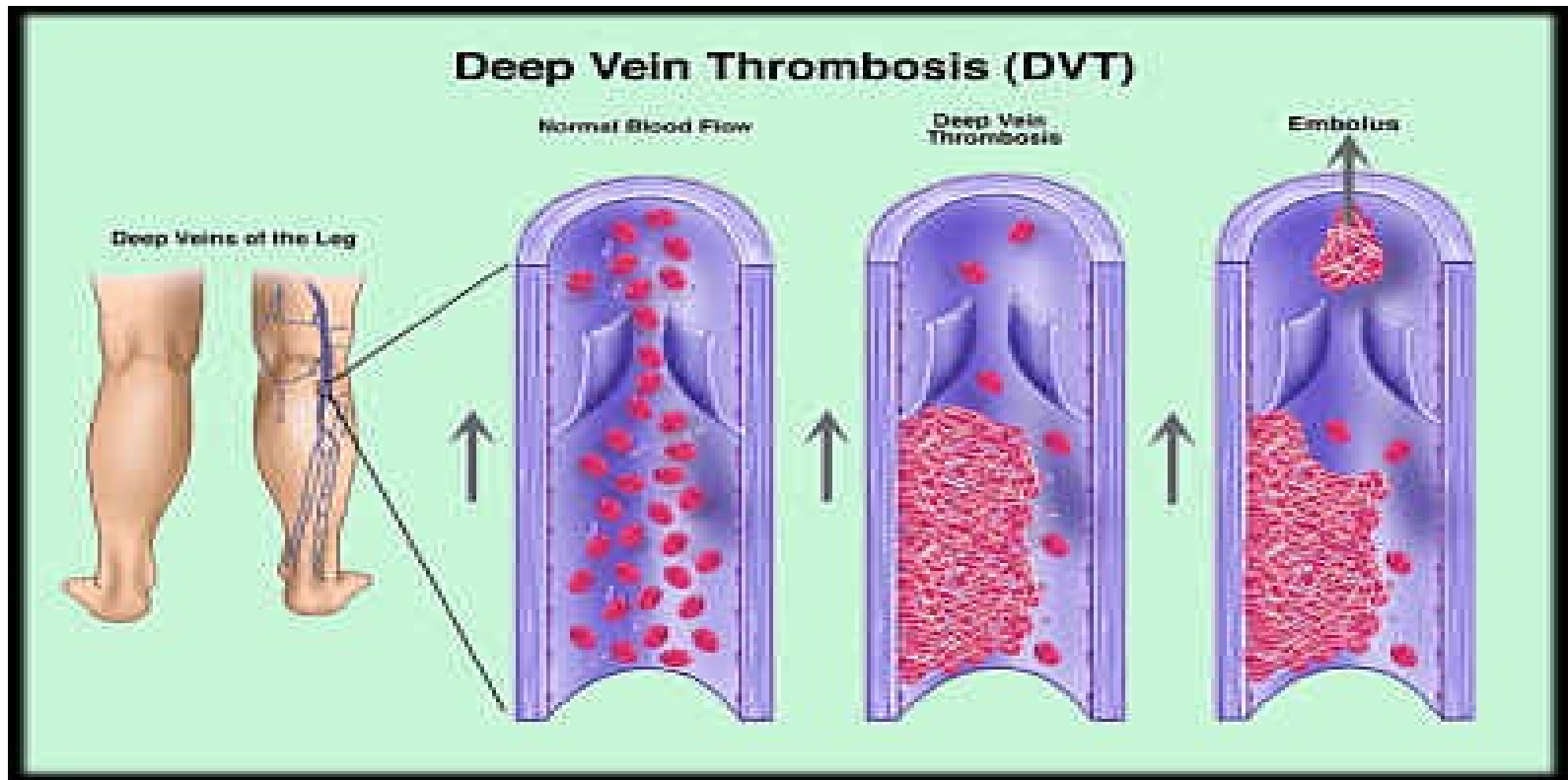


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- A **thrombus** is a clot or solid mass formed by blood components.
 - **Thrombosis** refers to the formation or presence of blood clots within a vessel. Occurs commonly in veins more than arteries.
 - Deep vein thrombosis is the formation of a blood clot in one of the deep veins of the body, usually in the leg.
 - Deep vein thrombosis affects the larger blood vessels deep in the legs.

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- Blood clots (thrombi) can form, which may break off and travel to the lungs. This is a potentially life-threatening condition



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- ✓ Venous thrombosis can occur in any vein but occurs more in the veins of the **lower extremities**. WHY????
- ✓ Upper extremity venous thrombosis is not as common as lower extremity thrombosis.
- ✓ However, upper extremity venous thrombosis is more common in patients with **intravenous catheters** or in patients with an underlying disease that causes **hypercoagulability**.

Etiology



Virchow's

1. Endothelial injury
2. Impaired blood flow
3. Hypercoagulability

- Injury to the vessel activates an inflammatory response.
- When an extremity remains immobile for any period of time, this pumping action is lost, resulting in venous stasis and pooling of blood.
- Hypercoagulability can be due to activation of circulating clotting factor and deficiency in the amount of antithrombin III.

Risk factors

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- ❑ Age (> 40 years of age)
- ❑ ↓ Antithrombin III increased factor VII
- ❑ Postoperative patient
- ❑ Varicose vein
- ❑ Previous deep vein thrombosis
- ✓ Infections or malignancies
- ✓ Patient with **cardiac disease** have
 - . Venous stasis
 - . Are less active
- ✓ Patients undergoing **estrogen therapy** due to



Clinical Manifestations

- ▶ Redness
 - ▶ Swelling
 - ▶ Heat /warm
 - ▶ Pain
- inflammatory response
- ▶ Tenderness or aching the extremity
 - ▶ 50 % are asymptomatic (Symptomatic deep vein thrombosis is "**tip of the iceberg**")

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- ❑ Positive **Homans' sign** (pain in the affected calf with forceful dorsiflexion of the foot)
- It is **not specific** because it can be elicited in any painful condition of the calf.
- Inflammation near the calf muscle can produce positive Homans' sign

NB: Caution must be taken to avoid a false positive result which can occur up to 50 % .

Homan's sign

Homan's sign: pain in the posterior calf or knee with forced dorsiflexion of the foot. It was defined by John Homan in 1941.



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- Differences in leg **circumference** bilaterally from thigh to ankle.
- Increase in the surface **temperature** of the leg, particularly the calf or ankle.
- Areas of **tenderness** or superficial thrombosis (ie, cordlike venous segment).
- In some cases, signs of a **pulmonary embolus** are the first indication of deep vein thrombosis.

Complications of Venous Thrombosis

- Chronic venous occlusions
- Pulmonary emboli from dislodged thrombi (Pieces of thrombus may break loose and travel through circulation- emboli)
- Valvular destruction
 - Chronic venous insufficiency
 - Venous ulcers
- Venous obstruction
 - Blood stasis
 - Venous gangrene

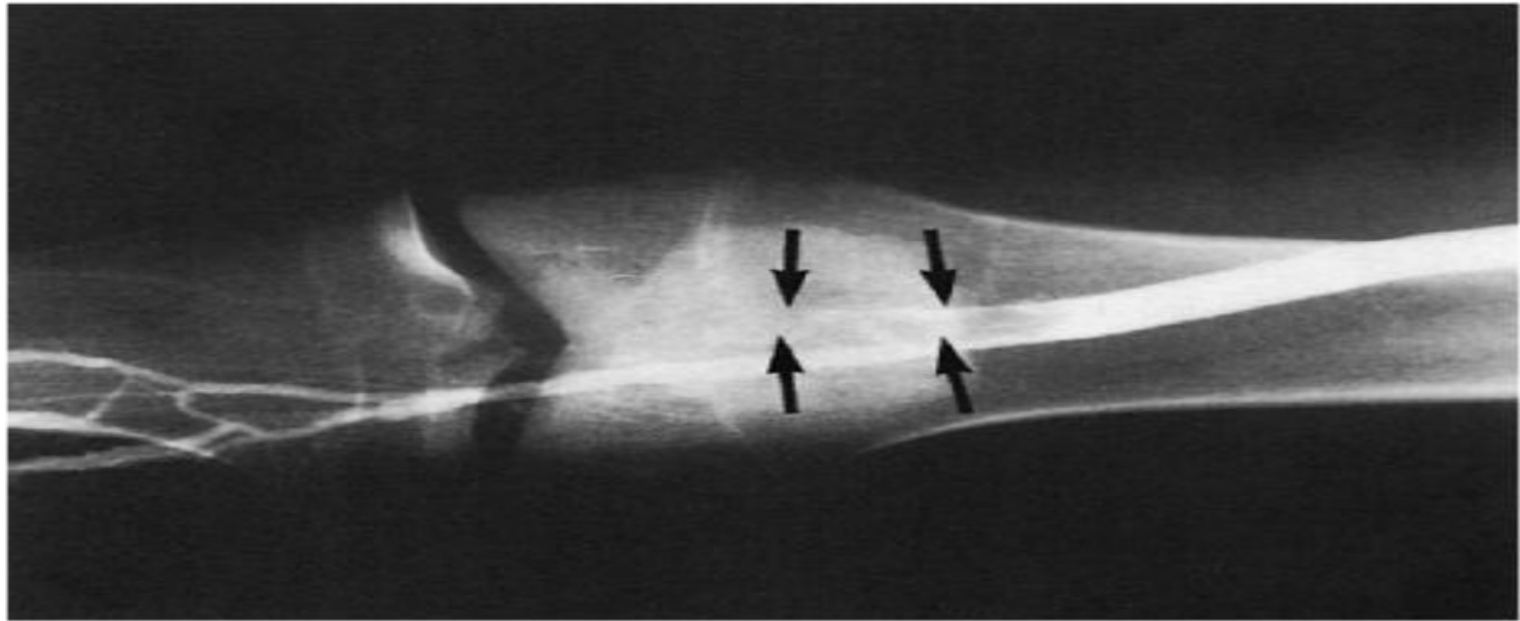


These pulmonary emboli removed at autopsy look like casts of the deep veins of the leg where they originated.



Diagnosis

- ✓ Clinical manifestations
- ✓ Nuclear venography (radiography of vein to detect thrombi in both calf and thigh)



Management

Preventive measures

- ❖ Application of elastic, graded compression stocking
- ❖ Special **body positioning** , active and positive leg exercise.
- ❖ Anticoagulants (prophylactic) for at risk patient
 - Low doses of **heparin** by activating antithrombin III decreasing production of factor X - to prevent venous thrombosis in surgical patients.
 - IV bolus dose of 5,000 to 10,000 units followed by an infusion of 1,000 units per hour.

- ❖ Dextran (glucose polymer)
 - ↓ Blood viscosity
 - Reduces platelet interactions with injured vessel walls
 - Promotes the dissolution of fibrin clots
- ❖ Dihydroergotamine mesylate (DHE)
 - ↑ venous smooth muscle tone
 - ↓ reactive vasodilatation
 - ↓ platelet aggregation

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- ✓ Embolex is more effective (Heparin + DHE)
- ✓ Warfarin (oral anticoagulant) - Prevents synthesis of Vitamin K dependent coagulation factor (II, VII, IX and X).
 - Oral dose of 2-10 mg/day.
- ✓ Analgesics (pain medications)
- ✓ Thrombolytics to dissolve an existing clot
- ✓ Antibiotics (if infection is present)

Surgical management

- ❑ Surgery is necessary for deep vein thrombosis when:
 - Anticoagulant or thrombolytic therapy is unsafe and **contraindicated**.
 - The danger of **pulmonary embolism** is extreme, or the venous drainage is so severely compromised that permanent damage to the **extremity** will probably result.
- ❑ **A thrombectomy** (removal of the thrombosis) is the procedure of choice.

Nursing Intervention

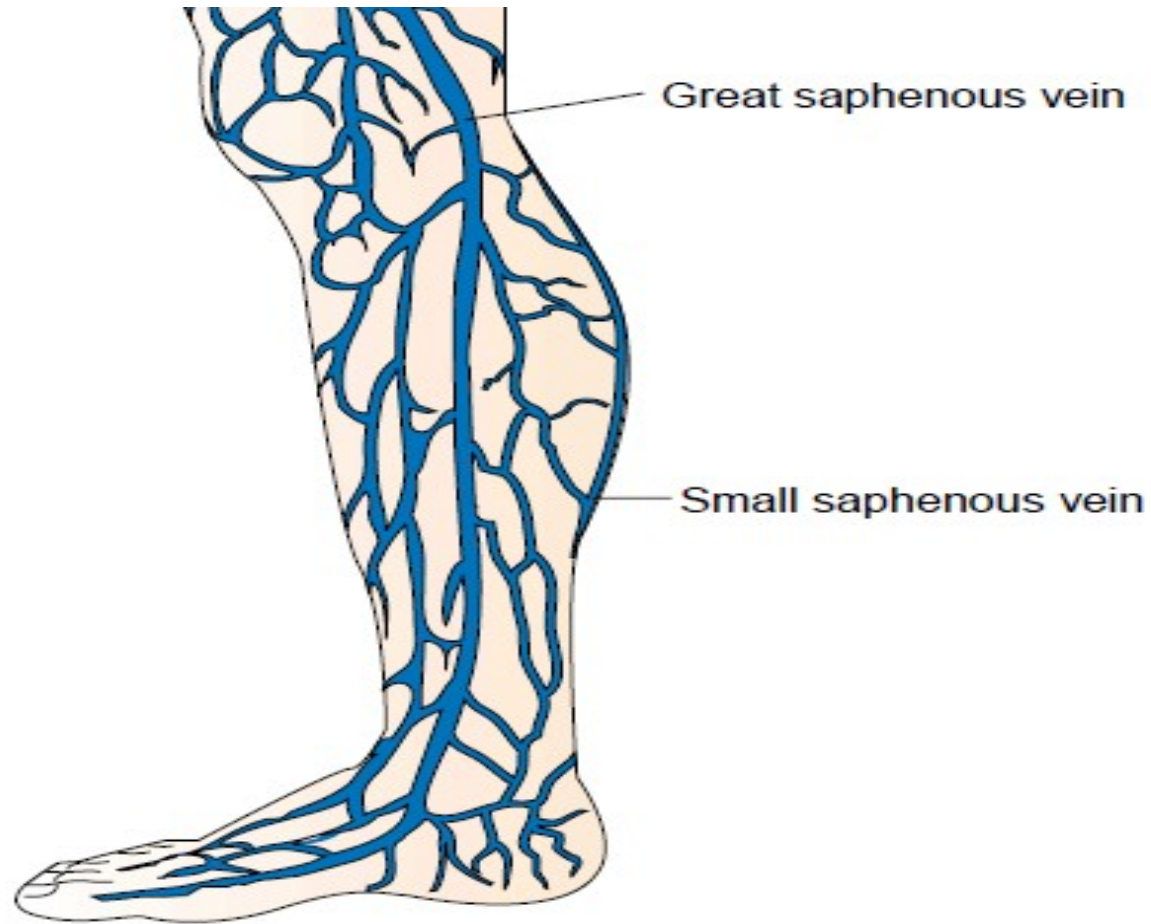
- Encourage movement of lower extremities and early ambulation.
- Encourage turning, coughing, and deep breathing at least every 2 hours
- Apply antiembolism stockings as ordered
- Elevate foot of bed 6 to 8 inches/elevate the affected area to reduce swelling.
- Restrict activity once thrombi is formed => to reduce risk of embolization.

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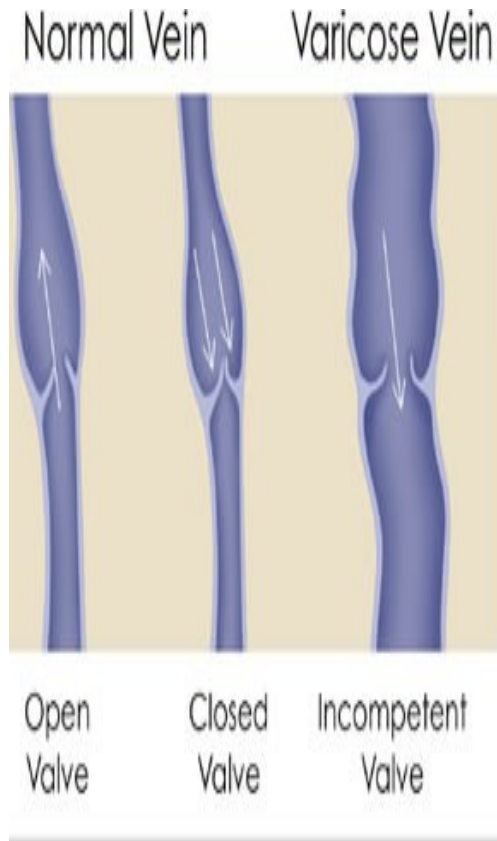
- Apply warm, moist heat to reduce inflammation and pain.
- Avoid rubbing or massaging the affected area (danger of embolizing)
- Close observation is also required to detect **bleeding**; if bleeding occurs, it must be reported immediately and anticoagulant therapy discontinued.

Varicose Veins

- ✓ Is a dilated, tortuous superficial vein that result from intrinsic weakness of the vein or by incompetent venous valves .
- ✓ Most commonly, this condition occurs in the lower extremities, the **saphenous veins**, or **the lower trunk**; however, it can occur elsewhere in the body, such as esophageal varices.
- ✓ It is a common problem affecting 10 % of the general population.
- ✓ It is more frequent in women, with increased age. WHY???



.The greater and lesser saphenous veins



- ❖ Most common in people whose occupations require **prolonged standing**, such as salespeople, hair stylists, teachers, nurses, and construction workers.
- ❖ A **hereditary** weakness of the vein wall may contribute to the development of varicosities.
- ❖ **Pregnancy** may cause varicosities. The leg veins dilate during pregnancy because of hormonal effects related to dispensability, increased pressure by the gravid uterus, and increased blood volume which all contribute to the development of varicose veins.

Risk factors

- ▶ Inherent **structural weakness** in the walls of the veins.
- ▶ Hereditary component/ Family history
- ▶ Advanced age (incompetent & weak valve)
- ▶ Prolonged standing or sitting
- ▶ Obesity & Pregnancy
- ▶ Constricting garments/clothes

Clinical Manifestations

- ✓ Prominent, enlarged, and tortuous vein on inspection
- ✓ Dull aches, muscle cramps, increased muscle fatigue in the lower legs
- ✓ The veins may appear **purplish** in color and feel hard or ropelike when palpated.
- ✓ Sensation of **heaviness or fullness** in legs
- ✓ Worsened pain after sitting or standing for a long time
- ✓ Skin color change

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Diagnosis

- History & physical examination
- CT Scan
- Doppler ultrasound
- Phlebography

Treatment

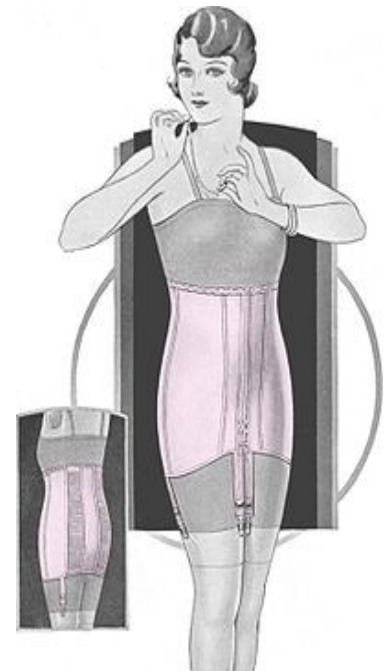
- ❖ Avoidance of standing or sitting for prolonged time.
- ❖ " " crossing the leg.
- ❖ " " wearing constricting garments
- ❖ Elevate foot of the bed 6 to 8 inches
- ❖ Wear full leg elastic support stocking, Exercising, losing weight, etc.
- ❖ Thermal Ablation
- ❖ Ligation and Stripping, Endoscopic vein surgery & Sclerosing therapy

Nursing Intervention

- ✓ Elastic compression stockings are worn continuously for about 1 week after vein stripping.
- ✓ The nurse assists the patient to perform exercises and move the legs.
- ✓ The foot of the bed should be elevated.
- ✓ Standing and sitting are discouraged

Prevention

- ✓ Avoid activities that cause venous stasis, such as wearing a constricting panty girdle or socks, crossing the legs at the thighs; and sitting or standing for long periods.
- ✓ Change position frequently, elevating the legs when they are tired.
- ✓ The patient is encouraged to walk 1 or 2 miles each day if there are no contraindications.
- ✓ Walking up the **stairs** rather than using the **elevator** or escalator is helpful, and swimming is good exercise.
- ✓ Encourage the patient to begin a weight-reduction plan.



Blood disorders

- ❑ Commonly encountered blood disorders are:
 - ♣ Anemia
 - ♣ Polycythemia
 - ♣ Leukopenia and Neutropenia
 - ♣ Leukocytosis
 - ♣ Bleeding and coagulation disorders
 - ♣ Leukemia

1. Anemia

- ❑ Blood hemoglobin (Hgb) concentration:
 - $< 130 \text{ g/L}$ ($<13 \text{ g/dl}$) or hematocrit (Hct) $< 39\%$ in adult males;
 - $<120 \text{ g/L}$ ($<12 \text{ g/dl}$) or Hct $< 37\%$ in adult females.
- ❑ As a result, the amount of oxygen delivered to body tissues is also diminished
- ❑ Anemia is **not a specific disease** state but a sign of an underlying disorder.
- ❑ It is by far the **most common** hematologic condition.

There are many different kinds of anemia

1. Bleeding (Resulting From RBC Loss)

- ✓ Bleeding from gastrointestinal tract, epistaxis (nose bleeding), trauma
- ✓ Bleeding from genitourinary tract (eg. menorrhagia)

2. Hypoproliferative (Resulting From decreased RBC Production)

- Iron deficiency , Vitamin B12 & Folate deficiency.
- Decreased erythropoietin production (eg. from renal dysfunction).

3. Hemolytic (Resulting From RBC Destruction)

- Hypersplenism (hemolysis)
- Drug-induced
- Autoimmune
- From direct injury to the erythrocyte within the circulation (mechanical heart valve-related anemia)

Common sign and symptoms includes

- Fatigue, weakness and restlessness are early manifestations.
- Dyspnea, tachycardia, irritability, decreased attention span.
- Hypovolemia; hypotension and decreased organ perfusion (In acute blood loss)
- Skin and mucous membranes may appear pale
- A smooth, sore tongue, brittle & ridged nails, & angular cheilosis in iron deficiency anemia
- Light headedness or dizziness may develops.

Normal

Pale



Diagnosis

(1) Reticulocyte Index (RI),

- RI is a measure of RBC production. RI 2-2.5% implies inadequate RBC production for the particular level of anemia.

(2) Review of blood smear and RBC indices [particularly mean corpuscular volume (MCV)].

- Cytoplasmic maturation defects such as iron deficiency or hemoglobin synthesis problems produce smaller RBCs, **MCV < 80**.

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(3) The hemoglobin, hematocrit, reticulocyte count, RBC indices

(4) Iron studies (serum iron level, total iron-binding capacity [TIBC], percent saturation, and ferritin), as well as serum vitamin B12 and folate levels, are also frequently obtained

(5) Bone marrow aspiration may be

Complications

- General complications of severe anemia include
 - Heart failure
 - Paresthesia, and
 - Confusion
- At any given level of anemia, patients with underlying **heart disease** are far more likely to have angina or symptoms of heart failure than those without heart disease.

Management

Medical Management

- ❖ Management of anemia is directed toward correcting or controlling **the cause** of the anemia and
- ❖ If the anemia is severe, the RBCs that are lost or destroyed may be replaced with **a transfusion** of packed RBCs (PRBCs).
- ❖ Bone marrow **transplantation** or peripheral blood stem cell transplantation in a aplastic anemia.

Nursing Interventions

➤ **Managing fatigue:**

- Assisting the patient to prioritize activities and to establish a balance between activity and rest.

➤ **Maintaining adequate nutrition:**

- Dietary supplements (Example: vitamins, iron, folate, protein may be prescribed).
- Advise the patient to avoid alcoholic beverages as it interferes with the utilization of essential nutrients.

➤ **Maintaining adequate perfusion:**

- Lost volume is replaced with transfusions or **intravenous fluids**

- Supplemental oxygen may be necessary, but it is rarely needed on a long-term basis unless there is underlying severe cardiac or pulmonary disease.

➤ The nurse **monitors vital signs** closely; other medications such as antihypertensive agents, may need to be adjusted or withheld.

➤ **Promoting compliance with prescribed therapy:**

- Assist patients in developing ways to incorporate the therapeutic plan into their lives, rather than merely giving the patient a list of instructions.

➤ **Monitoring and managing potential complication:**

- Patients with anemia should be assessed for signs and symptoms of heart failure, paresthesias in their lower extremities.

Reading assignment:

- ❑ Read different classification of anemias with their specific causes, clinical features, diagnosis and treatment.
- ✓ Iron deficiency anemia
- ✓ Vitamin B-12 deficiency anemia
- ✓ Vitamin B-9 (Folate) deficiency anemia
- ✓ Hemolytic anemia
- ✓ Aplastic anemia
- ✓ Sickle cell anemia
- ✓ Thalassemia
- ✓ Anemia of chronic disease

2. Polycythemia (Erythrocytosis)

- ❑ This is an increase above the normal range of RBCs in the circulation.
- ❑ Polycythemia refers to **an increased** volume of RBCs.
- ❑ Hgb level may be abnormally high at a level of 170 g/L (17 g/dl) in men and 150 g/L (15 g/dl) in women.
- ❑ It is a term used when the **hematocrit** is elevated (to more than 55% in males, more than 50% in females).
- ❑ **Hematocrit** - % of total blood volume consisting of RBCs.

Male= 42-52%

Female= 35-47%

-
- ❖ The estimated incidence of polycythemia is 2 per 100,000 people .
 - ❖ The median age at onset is 65 years.
 - ❖ With treatment, median survival exceeds 10 years.

Clinical Manifestation

- The symptoms result from the **increased blood volume** (headache, dizziness, tinnitus, fatigue, paresthesias, and blurred vision)

Diagnosis

- Elevated RBC mass (a nuclear medicine procedure),
- Elevated WBC and platelet counts.
- The **erythropoietin** level is not as low as would be expected with an elevated hematocrit; it is normal or only slightly low

Medical Management

Objective - to reduce the high blood cell mass.

- **Phlebotomy** (removing enough blood, initially 500 mL once or twice weekly) to keep the hematocrit within normal range.
- Patients need to be instructed to **avoid iron supplements**, including those within multivitamin supplements.
- Radioactive phosphorus (^{32}P) or **chemotherapeutic** agents can be used to suppress marrow function, but they may increase the risk for leukemia.

3. Leukopenia

- Normal leukocyte count = 4500 – 11,000/mm³
- **Leukopenia**, a condition in which there are fewer WBCs than normal, total leukocyte count < 4300/micro L,) results from:
 - Neutropenia (neutrophils <2000/mm³)
 - Lymphopenia (lymphocyte < 1000/micro L)
 - Monocytopenia (monocyte < 100/micro L)
 - Eosinopenia (eosinophil count < 50/micro L)

Normal Values for Leukocyte Concentration in Blood

 Cell Type	Normal Range	Percent Total
WBC		
Neutrophil	1830–7250	30–60%
Lymphocyte	1500–4000	20–50%
Monocyte	200–950	2–10%
Eosinophil	0–700	0.3–5%
Basophil	0–150	0.6–1.8%

Clinical Manifestations

- The **risk of infection** increases proportionately with the decrease in neutrophil count.
- The risk is **significant** when the ANC is less than 1000/mm³, is **high** when it is less than 500/mm³, and is almost **certain** when it is less than 100/mm³.
- The risk of developing infection also increases with the **length of time** during which neutropenia persists.
- **Fever** is the most common indicator of infection, yet it is not always present, particularly if the patient is taking **corticosteroids**.

Medical Management

- ❑ **Treatment of the neutropenia varies depending on its causes**
 - Stop medication if medication induced
 - Treatment of an underlying neoplasm
 - **Corticosteroids** may be used if the cause is an immunologic disorder.
 - Granulocyte/macrophage **colony-stimulating factor** (GM-CSF) can be effective in increasing neutrophil production when the cause of the neutropenia is decreased production.

-
- Withholding or reducing the dose of **chemotherapy or radiation therapy** may be required when the neutropenia is caused by these treatments
 - Broad-spectrum **antibiotics** are initiated after blood and other appropriate cultures are obtained
 - Risk of disseminated fungal infections; requires addition of **antifungal chemotherapy** (e.g., amphotericin B).

Nursing Management

- Assessing the severity of neutropenia and preventing and managing **infectious complications**.
- Patient teaching , particularly in the outpatient setting, so that the patient can implement appropriate **self-care measures** and know when and how to seek medical care.
- Collect blood for CBCs.
- Nurses need to be able to calculate the ANC and to assess the severity of neutropenia and the risk for infection.

4. Leukocytosis

- ❖ It refers to an **increased level of WBCs** in the circulation.
- ❖ Normal leukocyte count = 4500 - 11,000/mm³
- ❖ Although leukocytosis can be **a normal response** to increased need (eg, in acute infection), the elevation in WBCs should decrease as the need decreases.
- ❖ A prolonged or progressively increasing elevation in WBCs is abnormal and should be evaluated.
- ❖ A significant cause for persistent leukocytosis are **malignancy**

Neutrophilia

- ✓ Absolute neutrophil count $> 10,000/\text{micro L.}$

Lymphocytosis

- ✓ Absolute lymphocyte count $> 5000/\text{micro L.}$

Monocytosis

- ✓ Absolute monocyte count $> 800/\text{micro L.}$

Eosinophilia

- ✓ Absolute eosinophil count $> 500/\text{micro L.}$

Basophilia

- ✓ Absolute basophil count $> 100/\text{micro L.}$

Bleeding Disorders

- Normal hemostatic mechanisms can control bleeding from vessels and prevent spontaneous bleeding.
- The bleeding **vessel constricts** and **platelets aggregate** at the site, forming an unstable hemostatic plug.
- Circulating **coagulation factors** are activated on the surface of these aggregated platelets, forming **fibrin**, which anchors the platelet plug to the site of injury.
- The failure of normal hemostatic mechanisms can result in bleeding, which is severe at times.

➤ **Bleeding may result from abnormalities of:**

(1) Platelets, (2) Blood vessel walls, (3) Coagulation

➤ Platelet disorders characteristically produce **petechial** and **purpuric skin lesions** and **bleeding** from mucosal surfaces.

➤ Defective coagulation results in **ecchymosis**, **hematomas**, and mucosal and, in some disorders, recurrent joint bleeding (**hemarthroses**).

A. Thrombocythemia

- A marked **increase in platelet production** with the platelet count consistently greater than 600,000/mm³
- The platelet increase is accompanied by **an increase in RBCs or WBCs or both**
- **Normal platelet count: 150,000-350,000/micro L**
- The spleen typically holds about one third of the circulating platelets at any time.
- If the spleen is lost (eg, splenectomy), the platelet reservoir is also lost, and an abnormally high amount of platelets enter the circulation.

Clinical Manifestations

- ✓ Many patients are **asymptomatic**.
- ✓ Symptoms may occur more when the platelet count **exceeds 1 million/ mm³**.
- ✓ **Thrombosis** occurs in 15% to 40% of patients.
- ✓ Because these platelets can be **dysfunctional**, minor or major **hemorrhage** can also occur.
- ✓ Bleeding from the mucous membranes of the nose and mouth , and significant gastrointestinal bleeding is also possible.

- ✓ The toxic effects of platelet substances include **painful burning, warmth, and redness** in a localized distal area of the extremities.
- ✓ Neurologic manifestations may also be seen, such as numbness, tingling, and visual disturbance; these **occlusive manifestations** can progress to **stroke** and seizure and, less commonly, to **myocardial infarction**.
- ✓ Thrombotic complication may occur in age greater than 65 years, prior thrombotic events.

Diagnosis

- ❖ Examination of the **CBC** shows markedly abnormal platelets.
- ❖ **Analysis of the BM** (by aspiration and biopsy) shows a marked increase in megakaryocytes (platelet precursors).

Medical Management

- **Low-dose aspirin** therapy may be sufficient to prevent thrombotic complications; however, the use of aspirin can increase the risk for hemorrhagic complications and may be considered a contraindication in patients with a history of **GI bleeding**.
- **The neurologic symptoms** (eg, headache and erythromelalgia) and visual symptoms of thrombocytopenia can be relieved by low-dose aspirin.

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- **Hydroxyurea** (eg, Hydrea) and **anagrelide** (Agrylin) effective in lowering the platelet count.
- **Interferon-alfa-2b** (eg, Intron-A) has been shown to lower platelet counts by an unknown mechanism.

Nursing Management

✓ **Instruction about:**

- The risks of hemorrhage and thrombosis
 - Signs and symptoms of thrombosis, particularly
the neurologic manifestations, such as visual changes, numbness, tingling, and weakness.
- ✓ Patients receiving **aspirin therapy** should be informed about the increased **risk for bleeding**.

B. Thrombocytopenia

- ❑ Thrombocytopenia is defined as a platelet count $< 100,000/\text{micro L}$
- ❑ Normal platelet count is $150,000\text{--}350,000/\text{micro L}$.
- ❑ **Bleeding time**, a measurement of platelet function, is abnormally increased & injury or surgery may provoke excess bleeding.
- ❑ Spontaneous bleeding is unusual unless count $< 20,000/\text{micro L}$; platelet count $< 10,000/\text{micro L}$ is often associated with **serious hemorrhage**.

Causes of Thrombocytopenia

Decreased Production

- ❖ Toxins and Medications
- ❖ Infection (esp. septicemia, viral infection, tuberculosis)
- ❖ Alcohol and Chemotherapy

Increased Destruction

- ✓ Due to Antibodies, Lupus erythematosus
- ✓ Malignant lymphoma, Chronic lymphocytic leukemia
- ✓ Due to Infection, Bacteremia, Post viral infection

Increased Consumption

- ✓ Disseminated intravascular coagulation (DIC)

Clinical Manifestations

- **Bleeding** and **petechiae** usually do not occur with platelet counts greater than **50,000/mm³**, although excessive bleeding can follow surgery or other trauma.
- When platelet is **< 20,000/mm³**, petechiae, nose and gingival bleeding, excessive menstrual bleeding can occurs.
- When the platelet count is less than **5000/mm³**, **spontaneous**, potentially fatal central nervous system or gastrointestinal hemorrhage can occur.

Diagnosis

- Examining the bone marrow via aspiration and biopsy for **leukemia**.

Medical Management

- ❖ If platelet production is impaired, **platelet transfusions** may raise the platelet count and stop bleeding or prevent spontaneous hemorrhage.

Nursing Management

Control bleeding

- ✓ Apply direct pressure to the site.
- ✓ For epistaxis, position patient in high fowler's position; apply ice pack to back of neck & direct pressure to nose
- ✓ Notify physician for prolonged bleeding
- ✓ Administer platelets, fresh frozen plasma, packed red blood cells as prescribed.

Prevent complications

- Avoid aspirin & aspirin containing medications
- Do not give IM injections
- Do not insert indwelling catheters
- Use stool softeners, oral laxatives to prevent constipations
- Use the smallest possible needle when performing venipuncture.
- Discourage vigorous coughing or blowing of the nose.

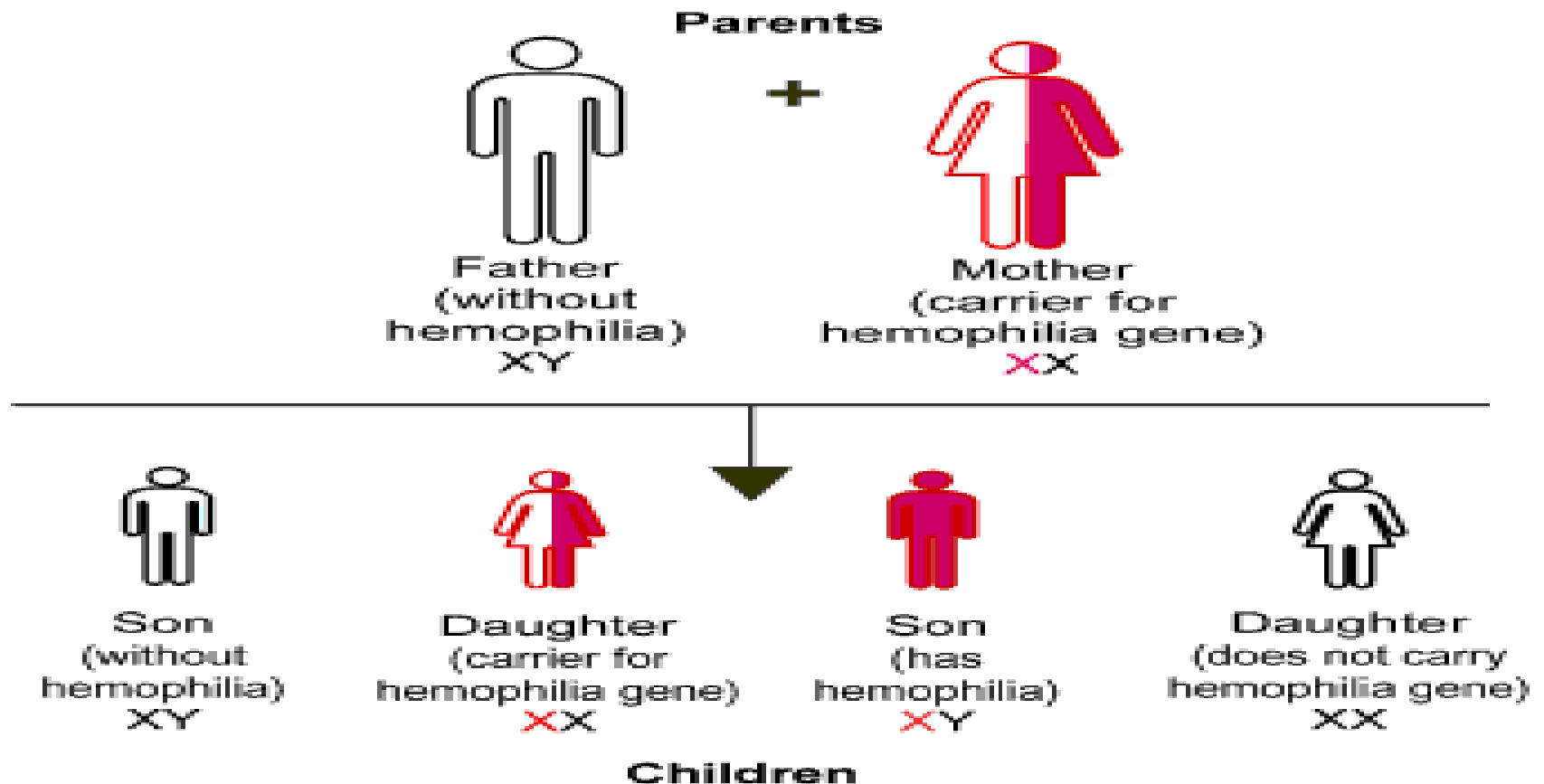
Hemophilia

- Inherited bleeding disorders in which the ability of the blood to **clot is severely reduced & cause bleeding**.
- Hemophilia A is caused by a genetic defect that results in deficient or defective **factor VIII**; hemophilia B stems from a genetic defect that causes deficient or **defective factor IX**.
- Both types of hemophilia are inherited as **X-linked traits**, so almost all affected people are males; females can be **carriers** but are almost always asymptomatic.

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- Hemophilia is a relatively rare disease; hemophilia A, which occurs in 1 of every 5000 births, is **three times more common** than hemophilia B.
- The disease occurs in all ethnic groups.
- **Hemophilia** is recognized in early childhood, usually in the toddler age group.
- However, patients with mild hemophilia may not be diagnosed until they experience severe trauma (eg, a high-school football injury) or surgery.

Inheritance of Hemophilia “Carrier” Mother and Father Without Hemophilia



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Clinical Manifestations

- **Hemorrhages** into various parts of the body even after minimal trauma.
- About 75% of all bleeding in patients with hemophilia occurs **in to joints** and Patients often note pain in a joint.
- With severe factor deficiency, they can occur **without known trauma** & progressively extend in all directions.
- Decreased sensation, weakness, & atrophy of the body.



-
- Spontaneous **Hematuria & GI bleeding** can occur.
 - Bleeding is also common in other mucous membranes, such as nasal passages.
 - The most dangerous site of hemorrhage is **in the head** (intracranial or extra cranial) .
 - Surgical procedures typically result in excessive bleeding at the surgical site.
 - **Wound healing is also poor** because clot formation is poor.

Medical Management

- Transfusion of fresh or frozen **plasma**.
- **Factor VIII & factor IX** concentrates can be administered.

As Rx or as a preventive measure before traumatic procedures (eg, lumbar puncture, dental extraction, surgery).

- Aminocaproic acid (EACA; Amicar) -slow the **dissolution of blood clots**.
- Desmopressin (eg, DDAVP), induces a transient **rise in factor VIII** levels.

Nursing management

- ✓ Most are diagnosed as children and **assist the patient to cope**.
- ✓ Teaching about activity restriction & self-care measures.
- ✓ Avoidance of use of **aspirin** , **NSAIDs** because they **interfere with clotting & inhibit platelet** function.
- ✓ Splint may be useful in patients with joint or muscular hemorrhage.
- ✓ All injection should be **avoided**; invasive procedures should be minimized or performed after administration of appropriate factor replacement.

Blood cancers - Leukemia

- Is a cancer of the blood or bone marrow and is characterized by an abnormal proliferation (production) of blood cells , usually white blood cells (leukocytes).

Classification

- Acute leukemia is characterized by the rapid increase of **immature** blood cells.
- Chronic leukemia is distinguished by the excessive build up of **relatively mature**, but still abnormal, white blood cells. Typically taking months or years to progress.
- ✓ In both cases the cells are produced **at a much higher rate** than normal cells, resulting in many abnormal white blood cells in the blood.

Causes & predisposing factors

- Although the exact cause of leukemia is **unknown** several etiologic factors have been identified including;
 - Viruses
 - Radiation
 - Exposure to drugs & chemicals
 - Familial predisposition

Clinical Features

- Onset can be abrupt or gradual,
- Usually non-specific & ill defined features at the onset.
- The most common classic manifestations are:
 1. **Anemia:** due to inadequate red blood cell production.
 2. **Bleeding:** “ “ Platelet production.
 3. **Infection:** “ “ white blood cell production.

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The above triads can be manifested with:

- ✓ Pallor
 - ✓ Fatigue & weakness
- } Due to anemia
-
- ✓ Dyspnea
 - ✓ Petechiae
 - ✓ Purpura
 - ✓ Bruise
- } Due to reduced platelets
- ✓ Lymphadenopathy & organomegally due to tissue infiltration.

Diagnosis

- ✓ Complete blood count and differential
- ✓ Bone marrow analysis (aspiration and/or biopsy)
- ✓ Chest x-ray and abdominal ultrasound studies
- ✓ An absolute lymphocyte count of $10,000/\mu\text{L}$ in the peripheral blood and
- ✓ 30% lymphocytes in the bone marrow establish the diagnosis of Chronic lymphocytic leukemia (CLL)

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Medical management

- People with leukemia have many treatment options.

General measures:

- ✓ Red blood cell transfusions to correct the anemia
- ✓ Treatments for infections caused by the lack of mature white blood cells by broad spectrum antibiotics
- ✓ Platelet transfusions to stop any bleeding

First line

- ✓ Chlorambucil 0.1 to 0.2mg/kg P.O. QD for 3 to 6 weeks **PLUS**
- ✓ Prednisolone 0.5-1mg/kg may be added, particularly in the presence of hemolytic anemia

Alternative

- ✓ Cyclophosphamide 2 to 4 mg/kg P.O. daily until systemic symptoms improve and the size of enlarged lymph nodes and organomegally are decreased **PLUS**
- ✓ Prednisolone 0.5-1mg/kg may be added, particularly in the presence of hemolytic anemia

...

Nursing management

- ✓ Supportive care with transfusions of red blood cells, white blood cells and platelets.
- ✓ Preventing or managing infection and bleeding
- ✓ Improving nutritional intake
- ✓ Maintaining fluid and electrolyte balance
- ✓ Easing pain and discomfort
- ✓ Managing anxiety and grief
- ✓ Promoting home and community based care

**Thank you for
your attention!**

