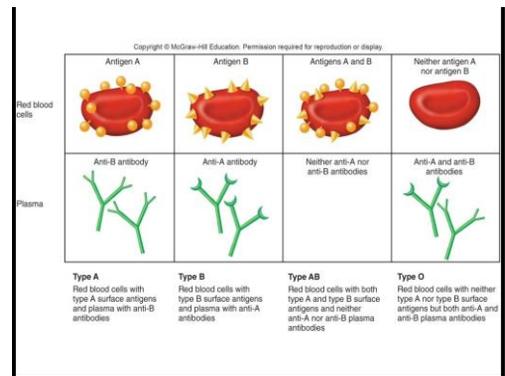


chapters 12, 13, 15, 16

Review

1

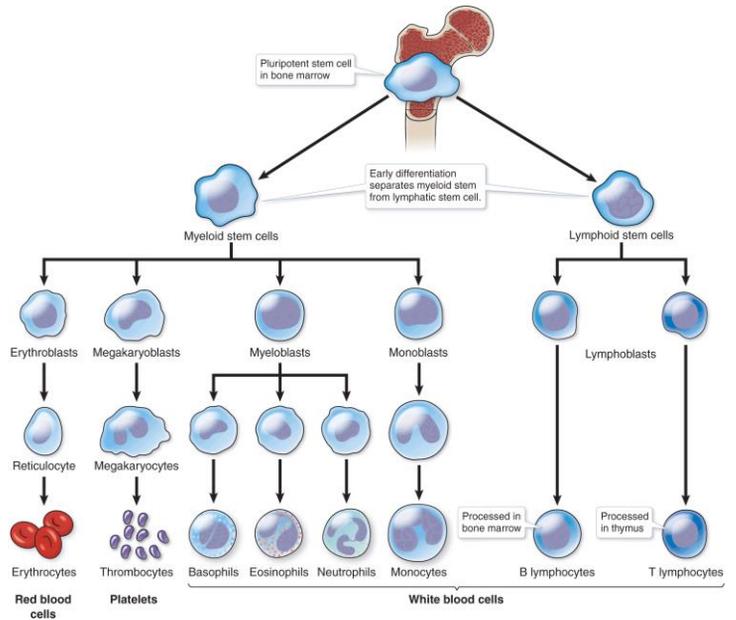
- All body cells, contain surface antigens.
- Specific types of antigens on the surface of RBCs are called **agglutinogens**.
- TYPES OF AGGLUTINOGENS:
 - type A
 - type B.
- FOUR TYPES according to these agglutinogens: type A, B, AB (**universal recipient**), and O (**universal donor**).
 - O → no antigens
- Another antigen on the surface of RBCs is **Rh factor**, also called D antigen.
 - If Rh factor → positive (Rh+)
 - If no Rh factor → negative (Rh-)



2

Thrombocytopenia: decreased platelets

- Resulting in increased susceptibility to bleeding and ecchymosis (bruising), nosebleeds (epistaxis), gingival bleeding, or subcutaneous hemorrhaging resulting in purpura or petechiae.

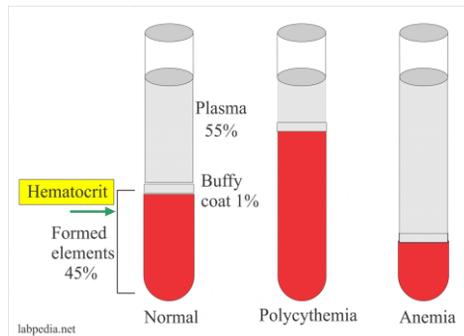
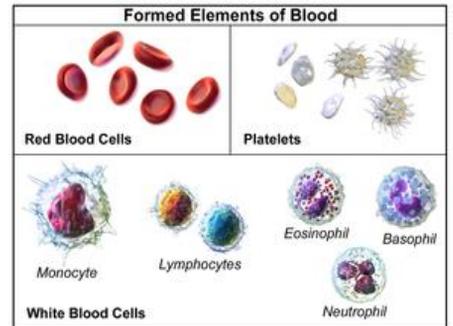


3

Lab tests

• **CBC With Differential.**

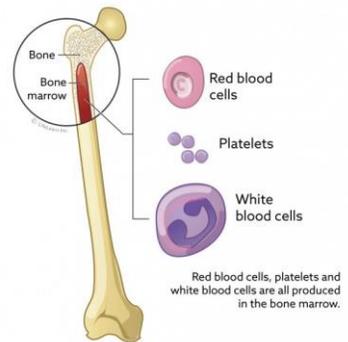
- identifies RBC number, Hgb, Hct, platelet number, and proportions of specific WBCs in the bloodstream.
- important initial diagnostic test used to identify which specific type of WBC.



4

Myelodysplastic Syndrome

- A **myelodysplastic syndrome (MDS)** is a broad term used for **disorders of the stem cells in the bone marrow**.
- Bone marrow hematopoiesis is dysfunctional and ineffective
- Resulting in deficient numbers of all blood cells or one type of blood cell: WBCs, RBCs, or platelets.
- 60 and 75 years (few patients are younger than age 50, and rare in children.
 - 10,000 to 20,000 new cases per year in the United States
- CAUSES: environmental exposures (radiation and benzene)
- Clinical presentation: similar to that of hematologic neoplasms.



5

Myelodysplastic Syndrome

- MDS can lead to complete bone marrow failure or leukemia.
- TREATMENT: involves transfusion of the deficient type of blood cells and chemotherapy.
- Lack of RBCs causes anemia,
- Lack of WBCs causes infections,
- Lack of platelets causes increased susceptibility to bleeding and bruising.

Leukemia:

- cancer of developing WBCs within the bone marrow.
- The cancerous WBCs are arrested at an early stage of development, proliferate uncontrollably, and do not function.

6

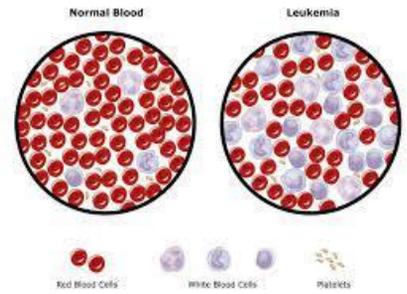
Leukemias

- Neoplastic proliferation involving WBCs other than lymphocytes.

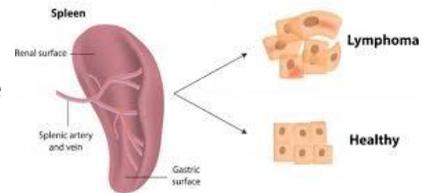
Lymphomas

- Neoplasms of lymphocytes and are commonly solid tumors found in lymphoid tissue.

- Each specific type of leukemia or lymphoma has a specific pathophysiologic process.



Lymphoma



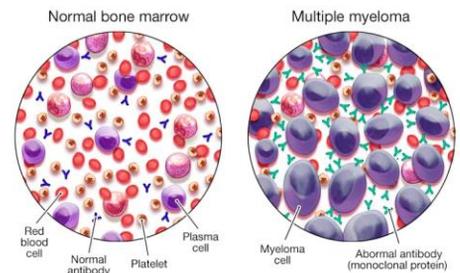
Leukemias and Lymphomas cause similar pathophysiologic conditions in the body.

- **Nonfunctional, cancerous WBCs proliferate** and overwhelm the bone marrow and other lymphoid tissue.
- The cancerous WBCs increase to excess numbers, and they crowd and **suppress development of the other blood cells (healthy WBCs, RBCs, and platelets) in the bone marrow.**

7

Multiple myeloma (MM)

- Hematologic neoplasm that arises in B lymphocytes.
- In normal conditions B lymphocytes transform into plasma cells, which then secrete Igs.
- In MM, the B lymphocytes that become plasma cells proliferate uncontrollably within the bone marrow.
- As a consequence, an excessive number of Igs are secreted by the plethora of plasma cells.
- There are **abnormally large numbers of abnormal Igs and Ig fragments.**



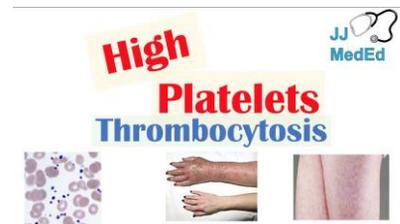
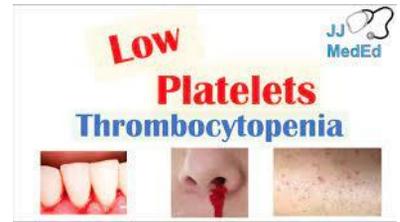
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Hematologic Neoplasms

- **Signs and Symptoms:**
 - Anemia
 - Thrombocytopenia
 - Fever, chills

8

- **Normal range of platelets = 150,000 to 400,000 cell/uL.**
- **Thrombocytopenia = low number of platelets**
 - fewer than 100,000/uL
 - can cause bleeding.
 - A wide number of medications can trigger an antibody reaction to platelets, causing lysis of platelets and thrombocytopenia. A viral or bacterial infection can also stimulate antibody destruction of platelets for unknown reasons.
- **Thrombocytosis = excessive number of platelets**
 - more than 750,000/uL
 - can cause excessive clotting.
 - In disorders such as polycythemia, a hyperactive disorder of the bone marrow, too many platelets can lead to development of thrombi (clots); as a result, occlusion of blood vessels can occur, and this can give rise to stroke, myocardial infarction, deep venous thrombosis, pulmonary emboli, and peripheral arterial disease.



9

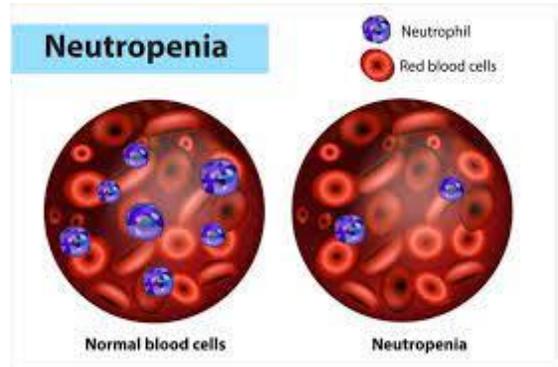
- A client is at risk of bleeding because the client's platelet count is 80,000/uL.
 - Thrombocytosis or Thrombocytopenia ?



10

Neutropenia

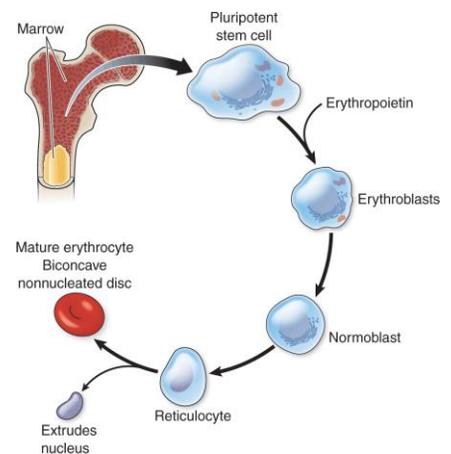
- Neutropenia is a decrease in neutrophil number
- CAUSE:
 - ✓ Diseases that damage the bone marrow (deficient marrow function)
 - ✓ Infections
 - ✓ Certain medications.
- Neutropenia can occur rapidly.
- Neutropenia increases risk of infection, decreases signs of infection, and diminishes healing ability.



11

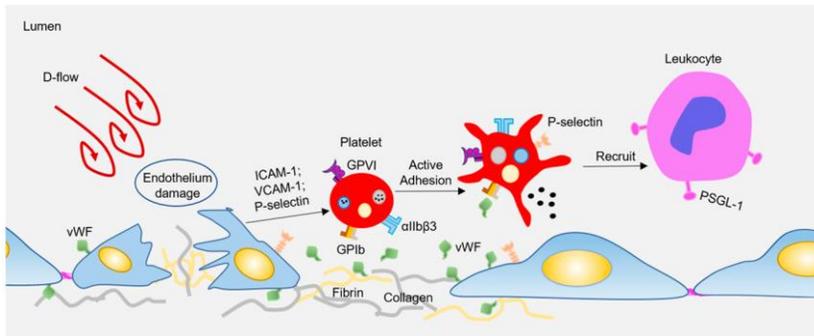
Synthesis of Erythrocytes

- **Erythropoiesis** → the specific steps in the bone marrow that leads to the synthesis of mature RBCs.
- Hypoxia is the major stimulus for erythropoiesis.
 - The kidney senses hypoxia in the bloodstream and releases the hormone erythropoietin (EPO), which stimulates the bone marrow.
 - When stimulated by erythropoietin, the bone marrow synthesizes RBCs from stem cell erythroid precursors.
 - The resulting increase in the number of RBCs reverses the hypoxia
- **Most common cause of erythropoietin deficiency:**
 - Renal failure



12

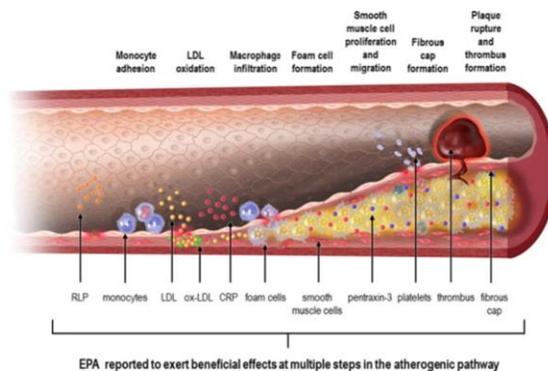
- **Hyperlipidemia** → elevated levels of lipids in the bloodstream.
- → Major condition that lead to atherosclerosis
- Hypercholesterolemia → elevated cholesterol levels
- Hypertriglyceridemia → elevated triglyceride levels
- Atherosclerosis → contributing factor to all forms of CVD.
- LDL: cholesterol which contributes to atherosclerosis
- High levels of LDL cholesterol and low levels of HDL cholesterol predispose to CVD.



13

If your client has a high low-density lipoprotein level (LDL), you understand that your client has a risk factor for coronary artery disease because you know that LDL promotes atheromas and plaque development

- **Optimally, LDL levels should be less than 100 mg/dL.**
- Several steps characterize the development of atherosclerosis.
 - endothelial injury → foam cells produced → atherosclerotic plaques develop → plaques calcify → fissures cause them to break off

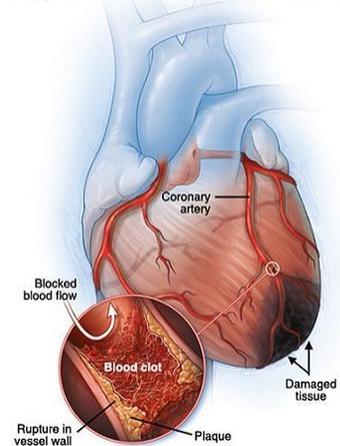


14

Ischemic Heart Disease (Myocardial ischemia)

- **Ischemic heart disease** is a disorder caused by the deprivation of blood to the myocardium of the heart.
- When coronary blood flow is interrupted or obstructed → lack of tissue perfusion (circulation), or ischemia.
- can be temporary → perfusion of the myocardium is reestablished.
- can be prolonged → lead to MI (death of heart muscle tissue)

Myocardial Ischemia



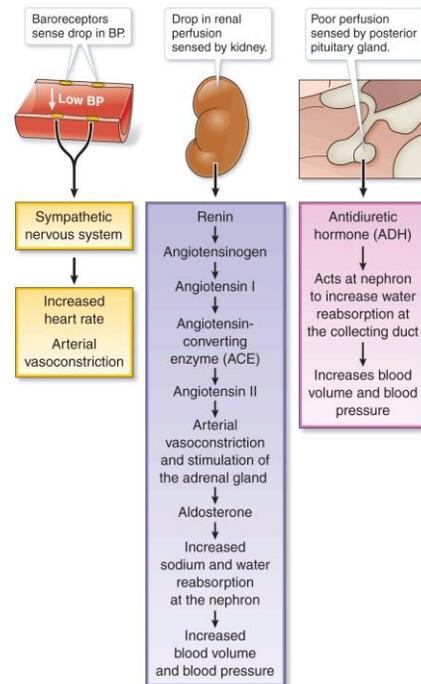
15

Several systemic mechanisms are in place to regulate BP and, subsequently, blood flow.

Mechanisms that regulates arterial blood pressure (BP) include:

- Baroreceptors
- rennin–angiotensin–aldosterone system (RAAS)*
- posterior pituitary–antidiuretic hormone (ADH) mechanism
- Natriuresis*

* *Why glomerulonephritis leads to high BP*

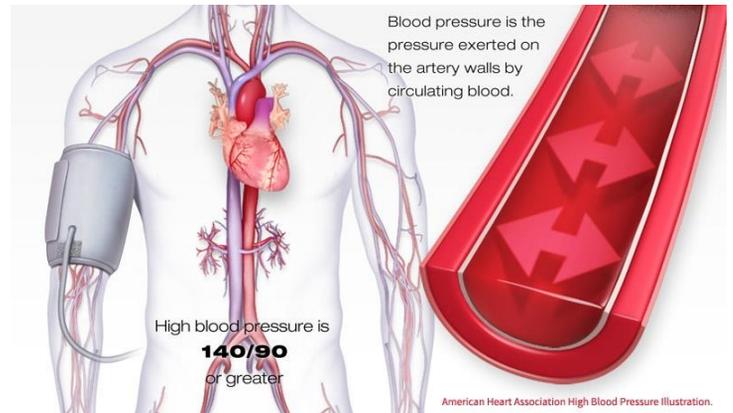


16

Hypertension (HTN) - elevated arterial BP

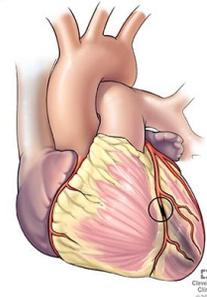
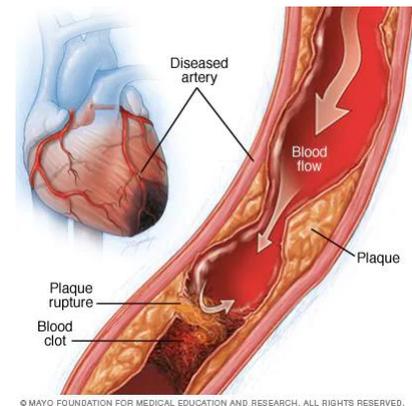
causes direct and indirect cardiovascular damage:

- Increased pressure from within the arterial vessel causes endothelial injury
 - Endothelial injury initiates atherosclerosis:
 - The damaged area of the endothelium decreases release of NO (chemical of vasodilation), and increases release of endothelin (potent vasoconstrictor).
 - The net result is arterial vasoconstriction and initiation of atherosclerosis in the area subjected to high BP.



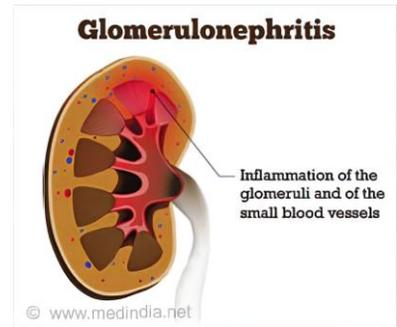
17

- Once **atherosclerotic plaque** is fully developed, elevated pressure within the blood vessel increases the stress on the area of plaque and commonly causes **plaque rupture**.
- A piece of plaque travels to a more distal, narrower branch of the artery and creates an **obstruction** at this site.
- **Ischemia** of tissue is the end result. When this occurs **in a coronary artery, it causes myocardial ischemia**.



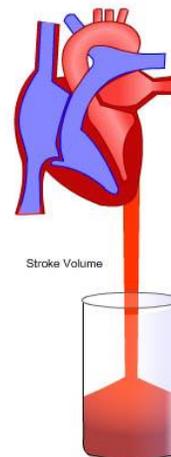
18

- There are two main categories of HTN:
 - primary HTN (essential HTN)
 - most common type
 - unknown etiology
 - secondary HTN
 - result of some pathology in another system or organ
 - *Example:* Glomerulonephritis.



19

- **CARDIAC OUTPUT (CO)** → the amount of blood that flows from the heart's left ventricle per minute.
- The pressure of blood = arterial BP
 - force against the walls of arteries as the heart pumps blood through the body.
 - Resistance = total peripheral vascular resistance (PVR) caused by the blood vessels within the circulatory system.



To increase cardiac output

Increase stroke volume
or

Increase heart rate
or
increase both

- Average heart rate (HR) = 70 beats per minute.
- The volume of blood ejected by the left ventricle per minute is the cardiac output (CO) → $CO = BP/PVR$

20

Glucose

- Chronically high circulating levels of glucose in the bloodstream cause harm to the endothelial lining of arteries.
- Glucose reacts with the cell membranes of endothelial cells in a process called glycosylation (also called glycation).
 - The glucose reacts with vessel wall proteins and forms advanced glycosylation end products (AGEs), → injure the smooth endothelial surface.
 - → endothelial inflammation and formation of atherosclerotic plaque.
 - → stimulation by AGEs causes endothelium to secrete endothelin (potent vasoconstriction).
- Continuous high glucose levels, like those that occur in uncontrolled diabetes, predispose to arteriosclerosis, atherosclerosis, and vasoconstricted arteries.
- These changes occur throughout the body, which is the reason diabetes is a risk factor for acute cardiovascular events such as stroke and myocardial infarction (MI).



21

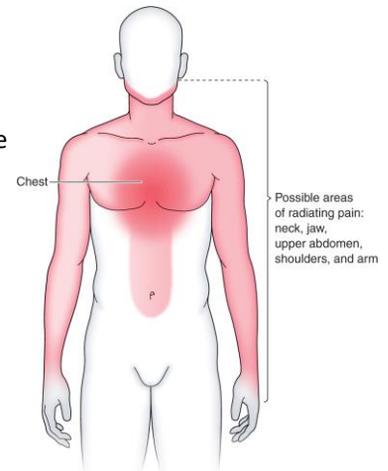
- **ANGINA PECTORIS** → squeezing pain in the chest when there is lack of blood flow to the myocardium (myocardial ischemia).
 - **Stable angina**
 - chronic chest pain that the patient **has experienced in the past and feels similar to past episodes.**
 - Prescribed medication for episodes of stable angina and self-medicates for treatment.
 - Pain usually occurs during exertion or other conditions that increase the heart's work demands.
 - Has a predictable pattern that has been experienced by the patient.
 - The patient is usually on a daily medication that aims to prevent angina episodes.
 - **Unstable angina**
 - Chest pain that is occurring **for the first time** in a patient.
 - Is an **emergency situation** requiring immediate medical attention.
 - Worsening of angina pain, sudden-onset angina at rest, and angina lasting more than 15 minutes are symptoms of UA.



22

ANGINA

- → history of chest pain brought on by exertion or stress.
- Patients report retrosternal chest discomfort and use terms such as pressure, choking, squeezing, or heaviness on the chest
- **Classic cardiac chest pain:** crushing sensation felt on the left side of the chest, radiating into the left shoulder down the left arm.
 - Alternatively, cardiac pain can radiate to the jaw, back, neck, right arm, or epigastric region
- **precipitated by exertion or stress** (Example: exposure to cold, eating a large meal, and emotional stress)
- Duration of the pain → **approximately 1 to 5 minutes.**
- **Can be relieved by:**
 - Rest
 - Medications (nitrates)
 - Intensity of pain does not change with respirations, cough, or change in position.
 - Additional stressors that can bring on angina



23

ANGINA PECTORIS

- Administer Oxygen if $\text{SaO}_2 < 95\%$.
- Medications: nitrates, aspirin, others.
 - Nitrates: potent vasodilators that widen the coronary arteries to deliver optimal circulation to the myocardial muscle.
 - reduce preload and afterload by stimulating dilation of veins and arteries.
 - sublingual tablet, nasal spray, paste, patch, or IV preparation.
 - Short-acting nitroglycerin (NTG) sublingual tablets → immediately stimulate coronary vasodilation during episodes of acute angina.
 - Long-acting nitrates, (Example: isosorbide)
- **Sublingual glyceryl trinitrate tablets or NTG spray remain the treatment of choice for rapid relief of acute symptoms and anticipated angina.**

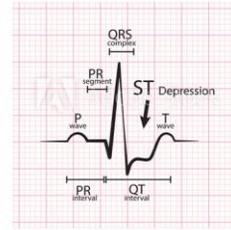
24

Acute Coronary Syndrome (ACS) :

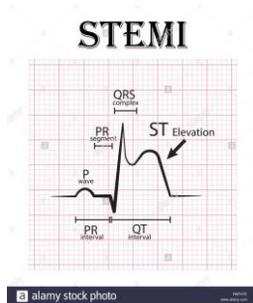
→ Unstable angina (UA), or MI.

- There are two main types of MI:
 - **ST segment elevation myocardial infarction (STEMI)**
 - **non-ST elevation myocardial infarction (NSTEMI).**

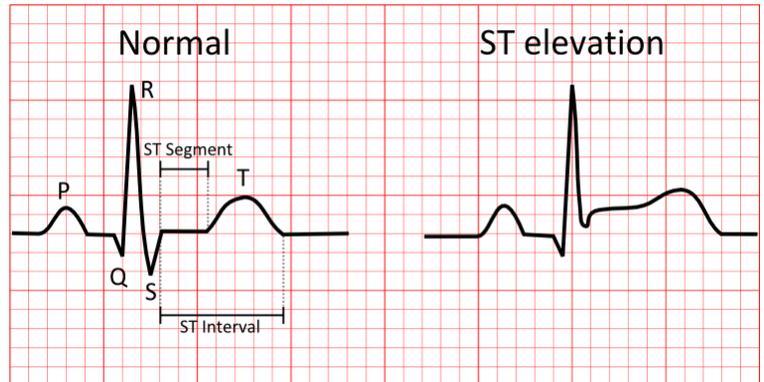
NSTEMI



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25

A client has had an acute myocardial infarction (MI) and his wife has a history of angina. She asks you how she will know if the pain is angina or if it is actually an MI. How would you answer?

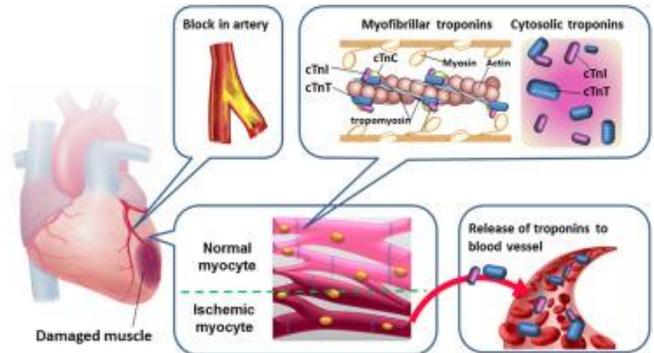


Rest and intake of **nitroglycerin** relieves chest pain from angina but not from MI

26

Angina Diagnosis – similar to arteriosclerosis

- Blood pressure (BP) measurement,
- total blood cholesterol, LDL, HDL
- triglycerides
- Additional tests → ECG, lipoprotein (a),
- serum electrolytes, homocysteine, cardiac enzymes, fibrinogen, cardiac troponins (cTn), chest x-ray, high sensitivity C-reactive protein (hs-CRP), calcium computerized tomography (CT) scan, cardiac angiogram, cardiac catheterization, and intravascular ultrasonography.

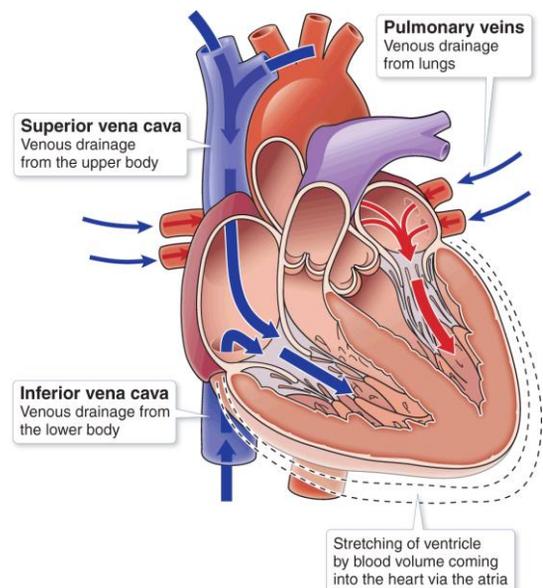


It is important to recognize that **cTn**, not an ECG, is the confirmatory test of MI.

27

Cardiac preload

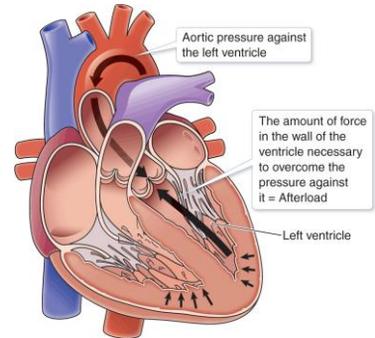
- **Preload** = volume of blood that stretches the right or left ventricle of the heart.
- Also referred as ventricular end-diastolic volume → achieved by the passive filling of the ventricle from the atria.
- In clinical setting: the volume of blood entering the right atrium is most often referred to as preload.



28

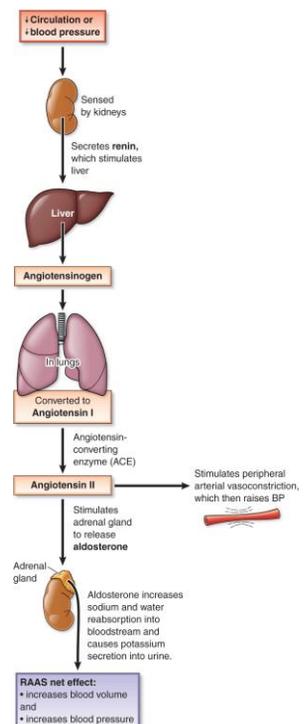
Cardiac afterload

- **Afterload** = force that the ventricle must overcome in order to eject its contents.
- It is the tension or stress developed in the wall of the ventricle during ejection.
- In clinical setting: afterload is most commonly measured as the aortic pressure against the left ventricle.
 - Aortic pressure reflects the systemic arterial pressure.
 - Therefore, if there is high systemic arterial pressure (also referred to as HTN), this is considered high afterload.



29

- to maintain fluid homeostasis → Kidney, RAAS, osmoreceptors, thirst, ADH, natriuretic peptides
- Hypotension, hypovolemia, dehydration, and low cardiac output cause low circulation throughout the body.
- Reduced circulation causes low renal perfusion, which stimulates **renin** secretion by the kidneys.
- **Renin** initiates the RAAS (a compensatory mechanism used to replenish blood volume and raise blood pressure)
 - **Renin** = enzyme released from the kidney in response to decreased renal perfusion.
 - **Renin** converts **angiotensinogen**, a large protein produced by the liver, to **angiotensin I**.
 - **Angiotensin I** is converted to **angiotensin II** in the lungs → In the lungs, angiotensin-converting enzyme (ACE) changes angiotensin I into angiotensin II, a powerful vasoconstrictor.
 - **Angiotensin II** causes release of aldosterone → Angiotensin II stimulates the synthesis and secretion of **aldosterone**, a mineralocorticoid that increases **sodium and water reabsorption** into the bloodstream.
 - **Aldosterone** also stimulates the excretion of potassium into the nephron tubules, which eventually becomes urine.
- When blood volume decreases, aldosterone begins the reabsorption of sodium from the distal tubules into the bloodstream.

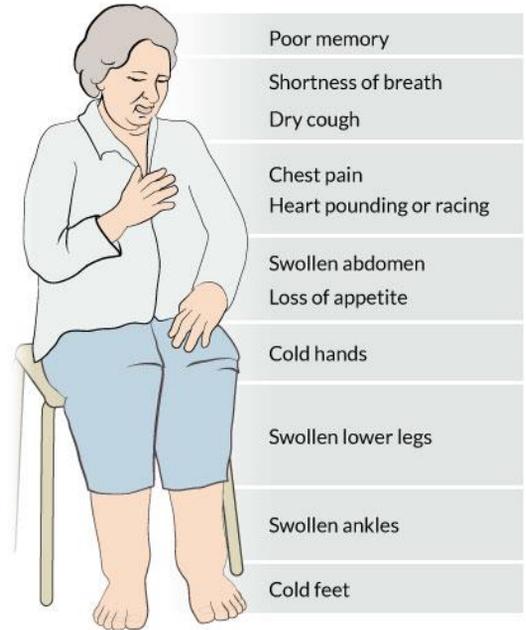


30

REVIEWING:

Manifestations of left heart failure, include:

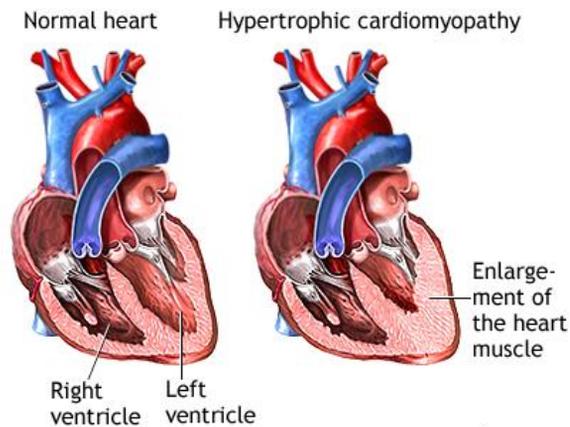
- Dyspnea
- Orthopnea
- Cough
- Edema ankles/legs



31

The increased workload on a client's heart with left heart failure has resulted in the following change to the myocardial cells:

- hypertrophy

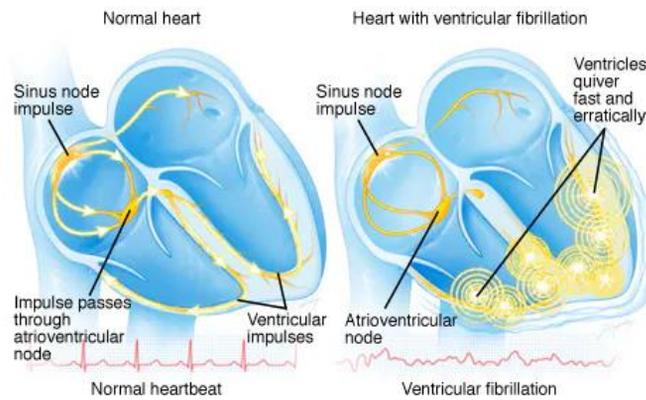


ADAM.

32

Ventricular Fibrillation:

- The heart is beating so rapidly that the ventricle is actually quivering and not ejecting any blood.



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