Pathophysiology

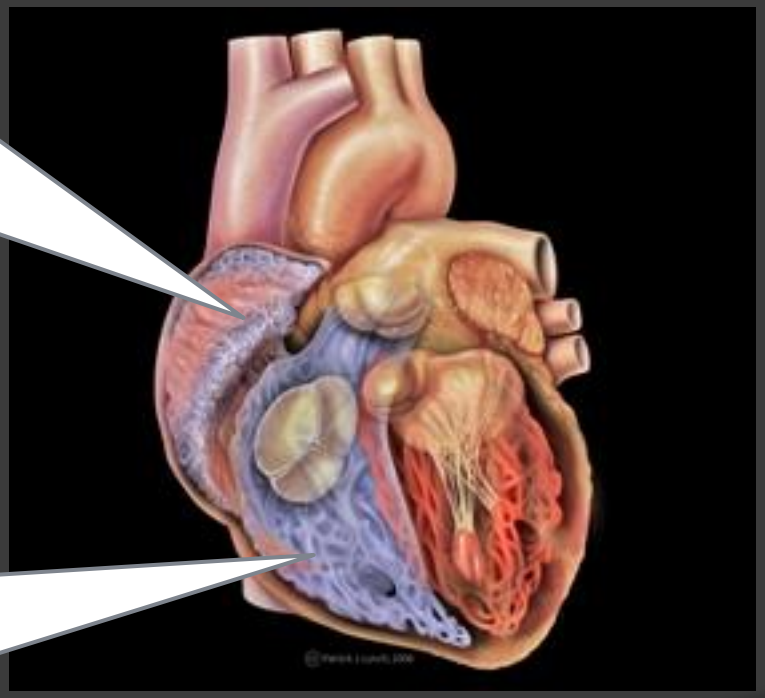
Cardiovascular disorders

Arrhythmia

Arrhythmia /dysrhythmia: abnormality in the site of origin of impulse, rate, or conduction

1. Bradycardia - Cardiac beats below 60 beats per minute .
2. Tachycardia – Cardiac beat above 100 beats per minute.

If the arrhythmia arises from atria, SA node, or AV node it is called supraventricular arrhythmia



If the arrhythmia arises from the ventricles it is called ventricular arrhythmia

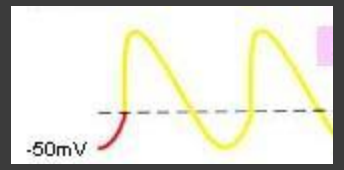
Causes of arrhythmia

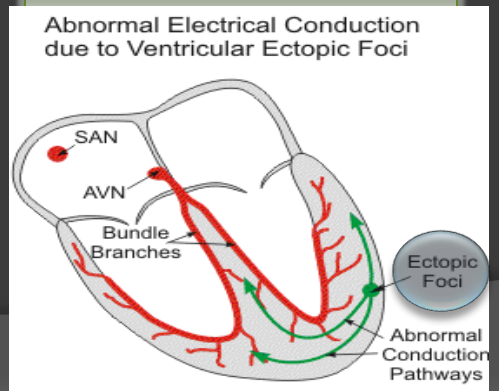
- arteriosclerosis
- Coronary artery spasm
- Heart block
- Myocardial ischemia

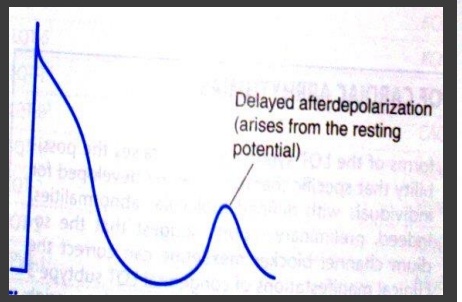
Mechanisms of Arrhythmogenesis

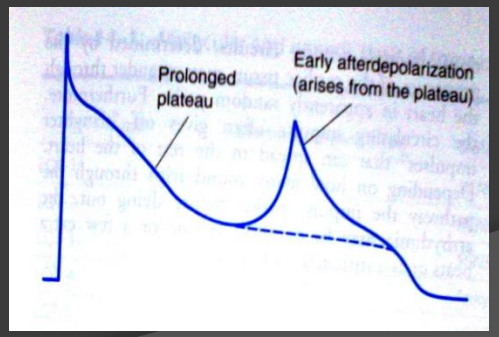
1- Abnormal impulse generation

- Automatic rhythms
  - Enhanced normal automaticity
    - ↑AP from SA node
  - Ectopic focus
    - AP arises from sites other than SA node
- Triggered rhythms
  - Delayed afterdepolarization
  - Early afterdepolarization







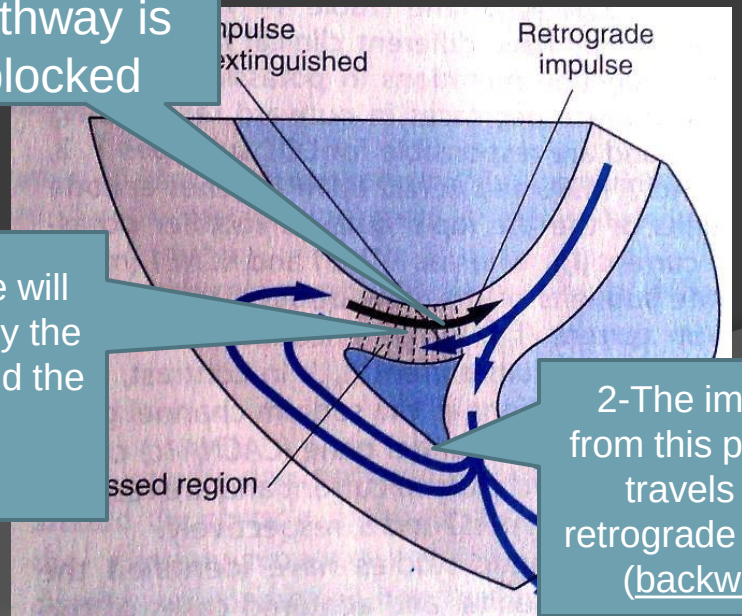


2-Abnormal conduction

- Conduction block
  - 1st degree
  - 2nd degree
  - 3rd degree
- Reentry
  - Circus movement
  - Reflection

This is when the impulse is not conducted from the atria to the ventricles

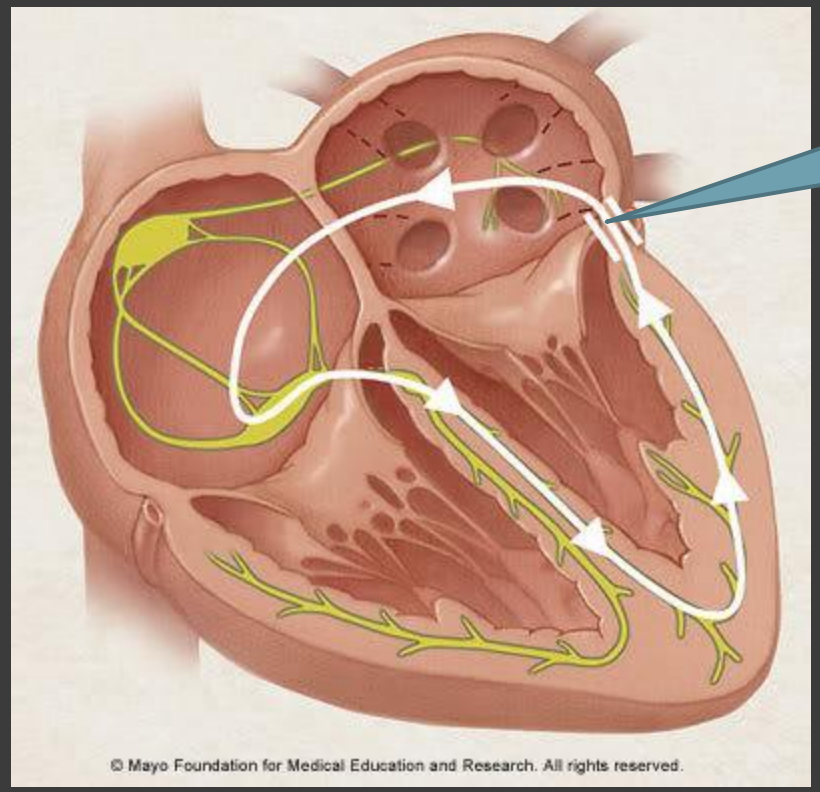
1-This pathway is blocked



3-So the cells here will be reexcited (first by the original pathway and the other from the retrograde)

2-The impulse from this pathway travels in a retrograde fashion (backward)

Abnormal anatomic conduction



Here is an accessory pathway in the heart called Bundle of Kent

- Present only in small populations
- Lead to reexcitation → Wolf-Parkinson-White Syndrome (WPW)

Types of Arrhythmia

Supraventricular Arrhythmias

- ✓ Sinus Tachycardia: high sinus rate of 100-180 beats/min, occurs during exercise or other conditions that lead to increased SA nodal firing rate
- ✓ Atrial Tachycardia: a series of 3 or more consecutive atrial premature beats occurring at a frequency >100/min
- ✓ Paroxysmal Atrial Tachycardia (PAT): tachycardia which begins and ends in acute manner
- ✓ Atrial Flutter: sinus rate of 250-350 beats/min.
- ✓ Atrial Fibrillation: uncoordinated atrial depolarizations.

AV blocks

A conduction block within the AV node , occasionally in the bundle of His, that impairs impulse conduction from the atria to the ventricles.

ventricular Arrhythmias

- **Ventricular Premature Beats (VPBs):** caused by ectopic ventricular foci; characterized by widened QRS.
- **Ventricular Tachycardia (VT):** high ventricular rate caused by abnormal ventricular automaticity or by intraventricular reentry; can be sustained or non-sustained (paroxysmal); characterized by widened QRS; rates of 100 to 200 beats/min; life-threatening.
- **Ventricular Flutter** - ventricular depolarizations >200/min.
- **Ventricular Fibrillation** - uncoordinated ventricular depolarizations

Arrhythmia take home message?

- Bradycardia?
- Tachycardia?
- Conduction velocity?
- Refractory period?

Coronary Artery Disease

■ The term coronary artery disease refers to disorders of myocardial blood flow due to stable or unstable coronary atherosclerotic plaques.

■ Unstable atherosclerotic plaques tend to fissure or rupture, causing platelet aggregation and potential for thrombus formation with production of a spectrum of acute coronary syndromes of increasing severity, ranging from unstable angina, to non-ST-segment elevation myocardial infarction, to ST-segment elevation myocardial infarction.

■ Stable atherosclerotic plaques produce fixed obstruction of coronary blood flow with myocardial ischemia occurring during periods of increased metabolic need, such as in stable angina.

Unstable Angina/Non-ST Elevation Myocardial Infarction

- Unstable angina/non-ST elevation myocardial infarction (UA/NSTEMI) is considered to be a clinical syndrome of myocardial ischemia ranging from angina to myocardial infarction.
- The pain associated with unstable angina typically has a persistent and severe course and is characterized by at least one of three features:
- (1) it occurs at rest (or with minimal exertion), usually lasting more than 20 minutes (if not interrupted by nitroglycerin);
- (2) it is severe and described as frank pain and of new onset (i.e., within 1 month); and
- (3) it is more severe, prolonged, or frequent than previously experienced

Unstable Angina/Non-ST Elevation Myocardial Infarction

- Unstable angina/NSTEMI is classified as low, intermediate, or high risk for acute MI, based on the clinical history, ECG pattern, and serum biomarkers. The ECG pattern in NSTEMI may demonstrate normal or St segment depression (or transient ST-segment elevation) and T-wave changes. The degree of ST-segment deviation has been shown to be an important measure of ischemia and prognosis.

ST Elevation Myocardial Infarction

- Acute ST elevation MI (STEMI), also known as heart attack, is characterized by the ischemic death of myocardial tissue associated with atherosclerotic disease of the coronary arteries. The area of infarction is determined by the coronary artery that is affected and by its distribution of blood flow.
- The onset of STEMI usually is abrupt, with chest pain as the significant symptom. The pain typically is severe and crushing, often described as being constricting, suffocating, or like "someone sitting on my chest." It usually is substernal, radiating to the left arm, neck, or jaw, although it may be experienced in other areas of the chest. Unlike that of angina, the pain associated with MI is more prolonged and not relieved by rest or nitroglycerin, and narcotics frequently are required.

ST Elevation Myocardial Infarction

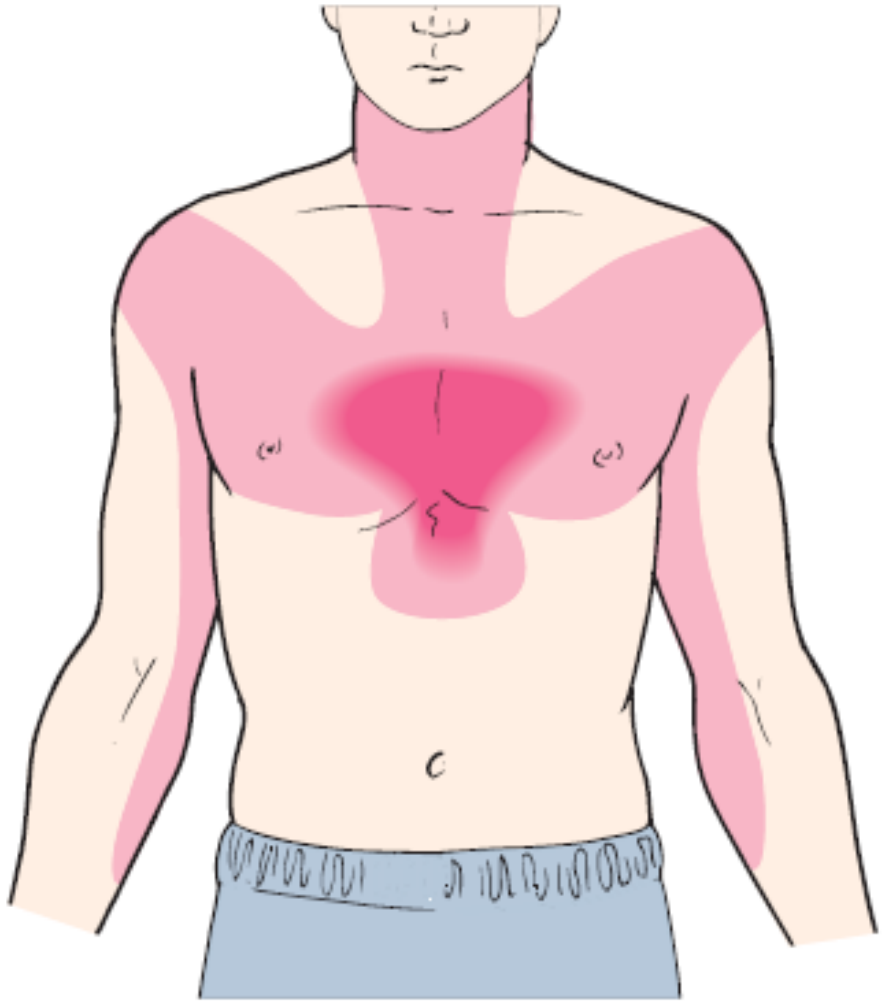
- Gastrointestinal complaints are common with acute MI. There may be a sensation of epigastric distress; nausea and vomiting may occur. These symptoms are thought to be related to the severity of the pain and vagal stimulation.
- The epigastric distress may be mistaken for indigestion, and the person may seek relief with antacids or other home remedies, which only delays getting medical attention.
- Sudden death from acute MI is death that occurs within 1 hour of symptom onset. It usually is attributed to fatal arrhythmias, which may occur without evidence of infarction.

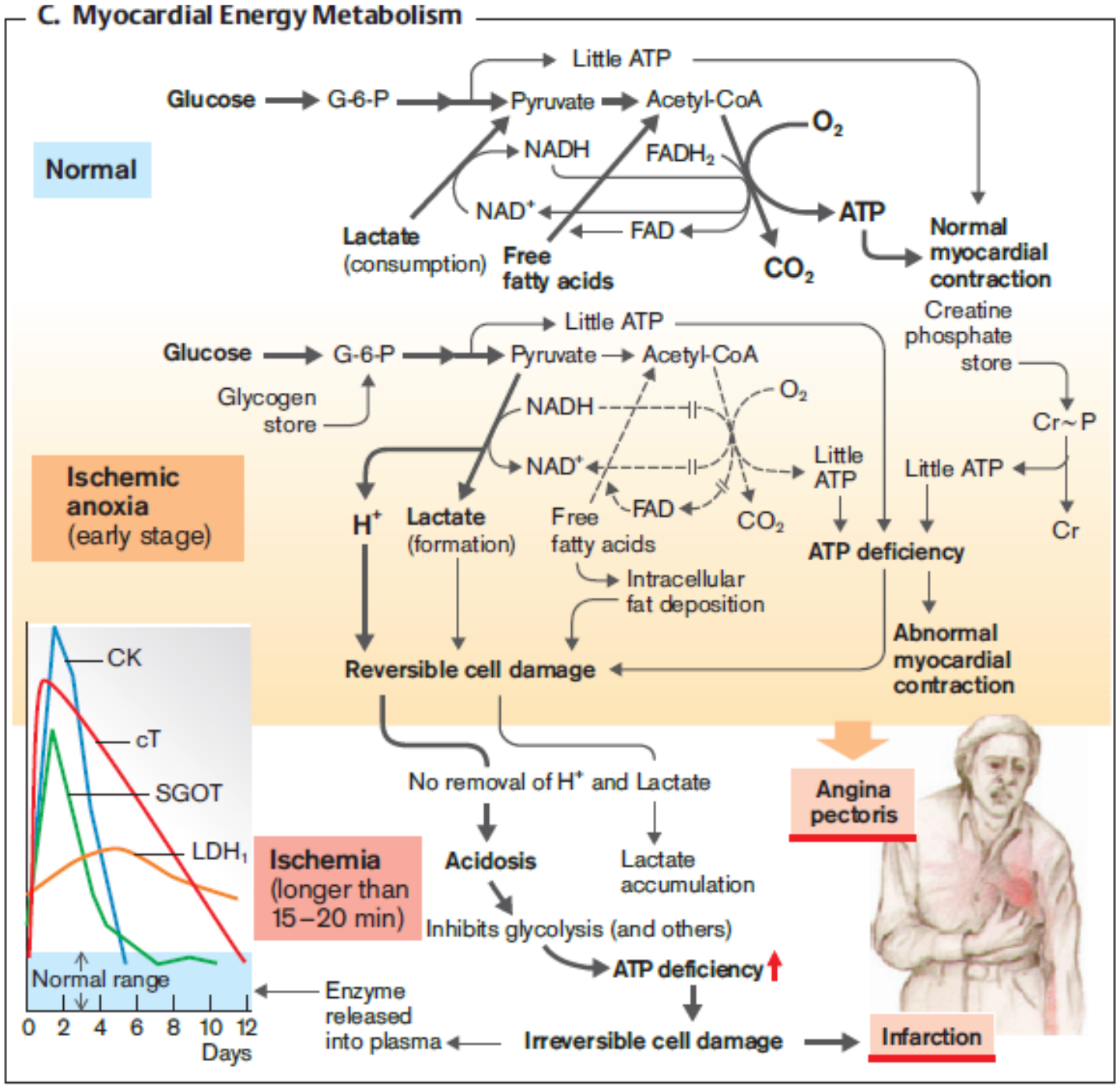
ST Elevation Myocardial Infarction

- The principal biochemical consequence of MI is the conversion from aerobic to anaerobic metabolism with inadequate production of energy to sustain normal myocardial function.
- As a result, a striking loss of contractile function occurs within 60 seconds of onset.
- Changes in cell structure (i.e., glycogen depletion and mitochondrial swelling) develop within several minutes.
- These early changes are reversible if blood flow is restored.

Chronic Ischemic Heart Disease
Chronic Stable Angina

- Angina pectoris is a symptomatic paroxysmal chest pain or p
myocardial ischemia.
- Chronic stable angina is associated with a fixed coronary obs
blood flow and metabolic demands of the myocardium.
- Stable angina is the initial manifestation of ischemic heart di
- chronic stable angina is provoked by exertion or emotional st
of nitroglycerin.




C. Myocardial Energy Metabolism
Normal
Little ATP
Glucose
G-6-P
Pyruvate
Acetyl-CoA
O_2
NADH
$FADH_2$
NAD^+
FAD
ATP
CO_2
Lactate
(consumption)
Free
fatty acids
Normal
myocardial
contraction
Ischemic
anoxia
(early stage)
Glycogen
store
Creatine
phosphate
store
Cr~P
Cr
H^+
Lactate
(formation)
Free
fatty acids
Intracellular
fat deposition
Little
ATP
ATP deficiency
Abnormal
myocardial
contraction
Reversible cell damage
CK
cT
SGOT
LDH_1
Normal range
0 2 4 6 8 10 12
Days
Enzyme
released
into plasma
Ischemia
(longer than
15–20 min)
No removal of H^+ and Lactate
Acidosis
Lactate
accumulation
Inhibits glycolysis (and others)
ATP deficiency
Irreversible cell damage
Angina
pectoris
Infarction

Heart failure

■ The function of the heart is to move deoxygenated blood from the venous system through the right heart into the pulmonary circulation, and oxygenated blood from the pulmonary circulation through the left heart and into the arterial circulation.

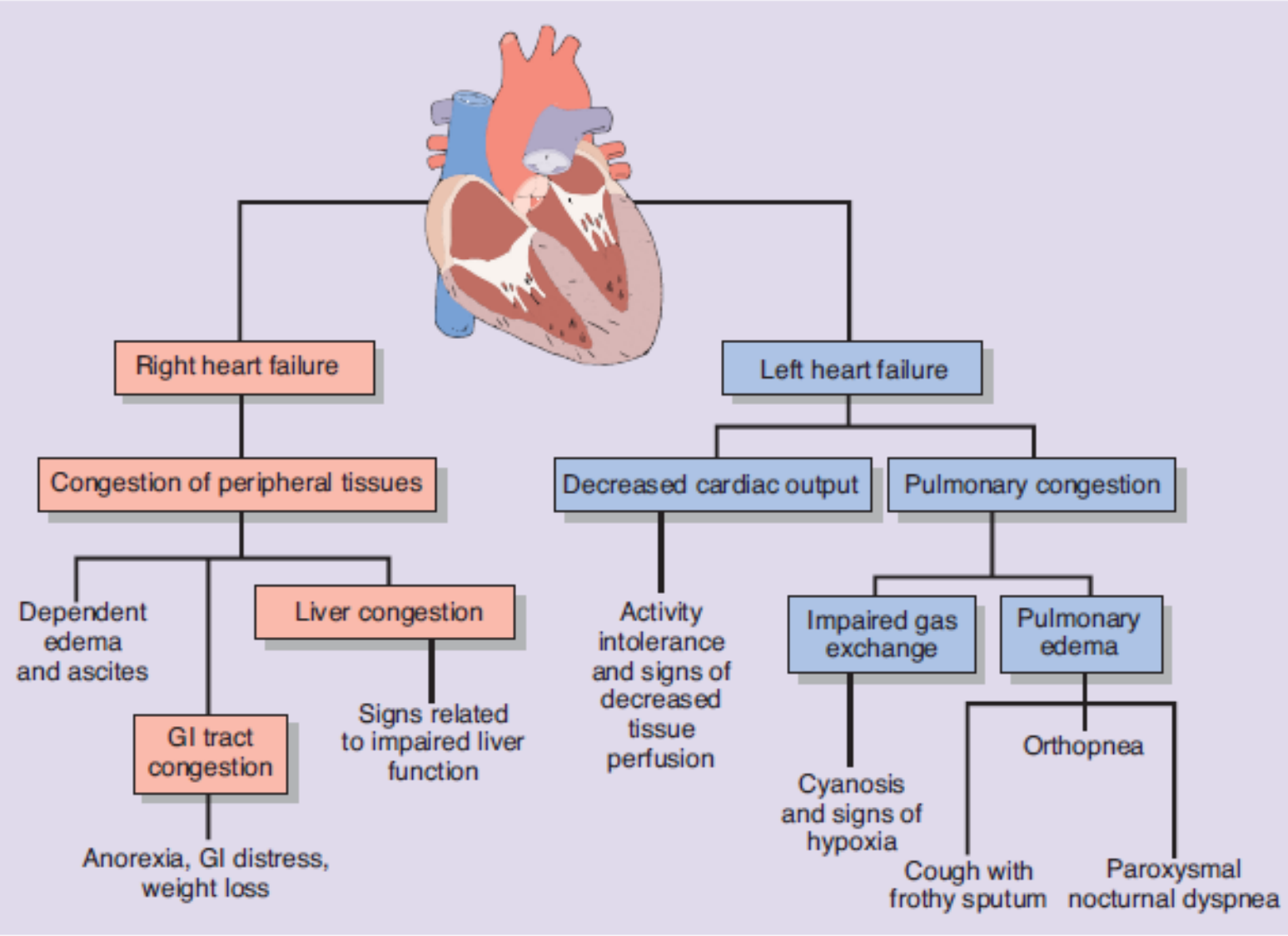
- The term heart failure denotes the failure of the heart to pump enough blood to meet the metabolic needs of the body.

■ Systolic dysfunction represents a decrease in cardiac myocardial contractility and an impaired ability to eject blood from the left ventricle; whereas, diastolic dysfunction represents an abnormality in ventricular relaxation and filling.

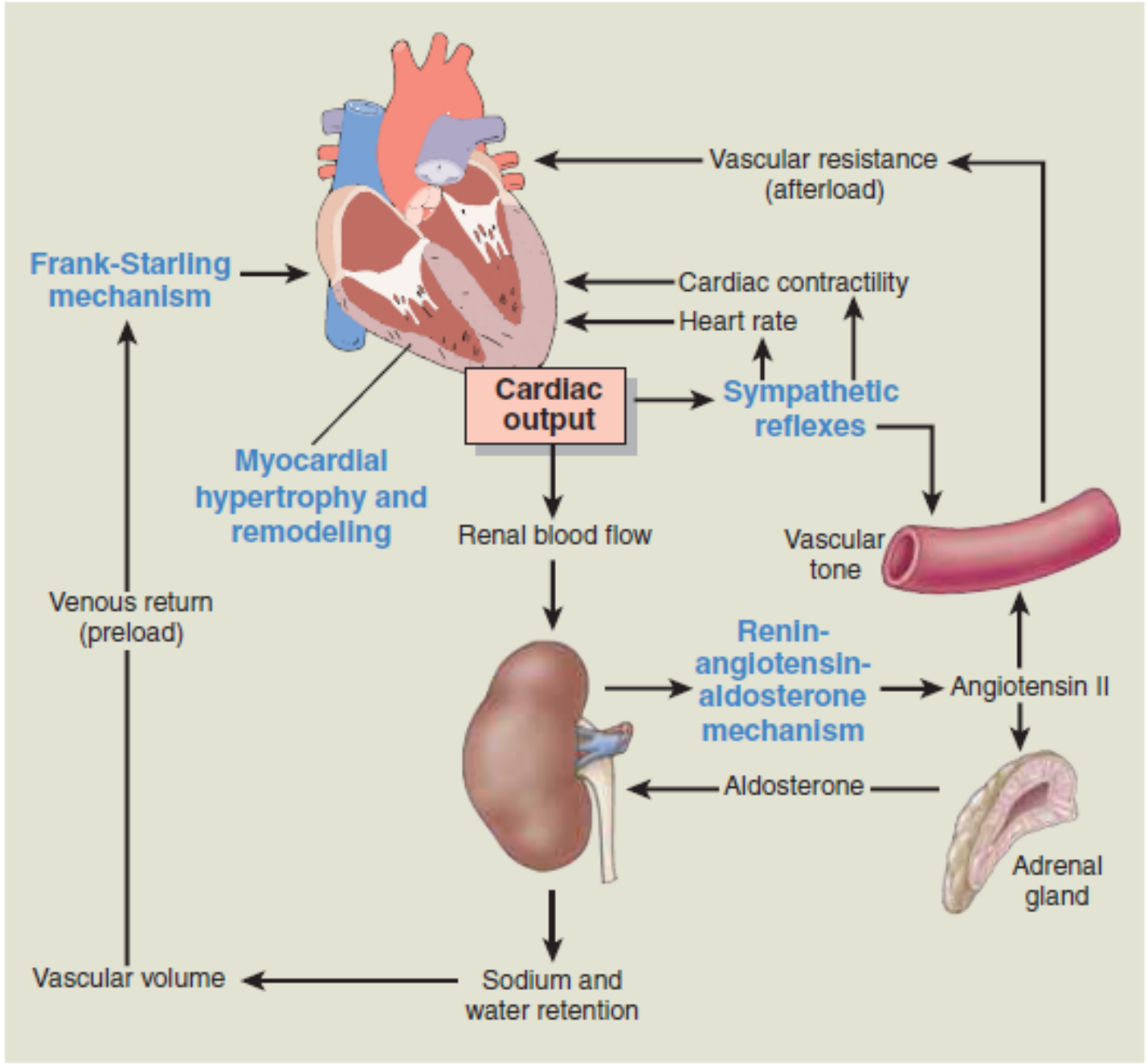
Heart failure

- Right-sided heart failure represents failure of the right ventricle to move unoxygenated blood from the venous system into the pulmonary circulation, with an eventual backup in the systemic venous circulation; whereas, left-sided heart failure represents failure of the left ventricle to move oxygenated blood from the pulmonary circulation into the arterial circulation with eventual backup of blood in the lungs. Both types result in decreased forward flow, resulting in poor circulation of oxygenated blood in the body.

Manifestations of right- and left-sided heart failure.



Compensatory mechanisms in heart failure. The Frank–Starling mechanism, sympathetic reflexes, renin–angiotensinaldosterone mechanism, and myocardial hypertrophy function in maintaining cardiac output for the failing heart.



Cardiomyopathy take home message?

- Heart?
- Blood pressure?
- Kidney?
- Life style?
- Diet?
- .....

References

- Carol Mattson Porth - Essentials of Pathophysiology_ Concepts of Altered Health States, 3rd Edition (2010, Lippincott Williams & Wilkins)
- Stefan Silbernagl, Florian Lang - Color Atlas of Pathophysiology (Basic Sciences) [Thieme] (2009, TPS)

Thank you