



Heart I:

Heart Failure,
Congenital Heart Disease,
and Ischemic Heart Disease

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Cardiac Pathology Outline

- Blood Vessels
- Heart I
 - Heart Failure
 - Congenital Heart Disease
 - Ischemic Heart Disease

Heart I Lecture Objectives

- Describe in simple terms what heart failure is.
- Explain how the heart tries to adapt to heart failure and list three consequences that can result when adaptation fails.
- To make it easier on yourself (so you can understand more and memorize less), review the basics of heart blood flow (i.e., where does blood from the left side vs. the right side of the heart go?)
- Look at slides 9-12 and make sure you understand why the symptoms in left and right sided heart failure make sense.

Heart I Lecture Objectives

- Define the following terms: pulmonary edema, ascites, peripheral edema, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cor pulmonale, nutmeg liver.
- Explain what's wrong with the heart in the four congenital heart diseases covered in lecture.
- Explain what can happen clinically in atrial and ventricular septal defect.
- Compare and contrast left-to-right and right-to-left shunts. Define Eisenmenger syndrome and know which congenital defects it occurs in.

Heart I Lecture Objectives

- What does the ductus arteriosus do in the fetus? What normally happens to it after birth? Make sure you understand the direction of blood flow in the fetus and how that changes after birth. Check out the posts online from Path Student (posted on our home page) if this seems confusing.
- Make sure you know the four features of Tetralogy of Fallot. There is a way to understand the disease so you don't have to just memorize four seemingly random features – we'll talk about that in class.

Heart I Lecture Objectives

- Define ischemic heart disease, and list the three syndromes that are associated with it.
- Define angina pectoris, and describe the symptoms and underlying causes of both typical and unstable angina.
- Define myocardial infarction and explain what makes it different from angina. Describe the usual series of events that lead up to myocardial infarction.
- Describe the histologic findings seen in myocardial infarction on day 1, day 3, and day 7.

Heart I Lecture Objectives

- List the clinical symptoms of myocardial infarction (MI), and compare and contrast how troponins and CK-MB change over time after an MI.
- List some potential complications of MI, and describe in general what the prognosis is for someone who has had an MI.

Okay – these objectives are LONG. But they cover EVERYTHING you need to know. I decided to err on the side of a little too wordy rather than skip things.

Cardiac Pathology Outline

- Blood Vessels
- Heart I
 - Heart Failure

Heart Failure

- End point of many heart diseases
- Common!
 - 5 million affected each year
 - 300,000 fatalities
- Heart can't pump blood fast enough to meet needs of body
- Most due to systolic dysfunction
- Some due to diastolic dysfunction, valve failure, or abnormal load

Heart Failure

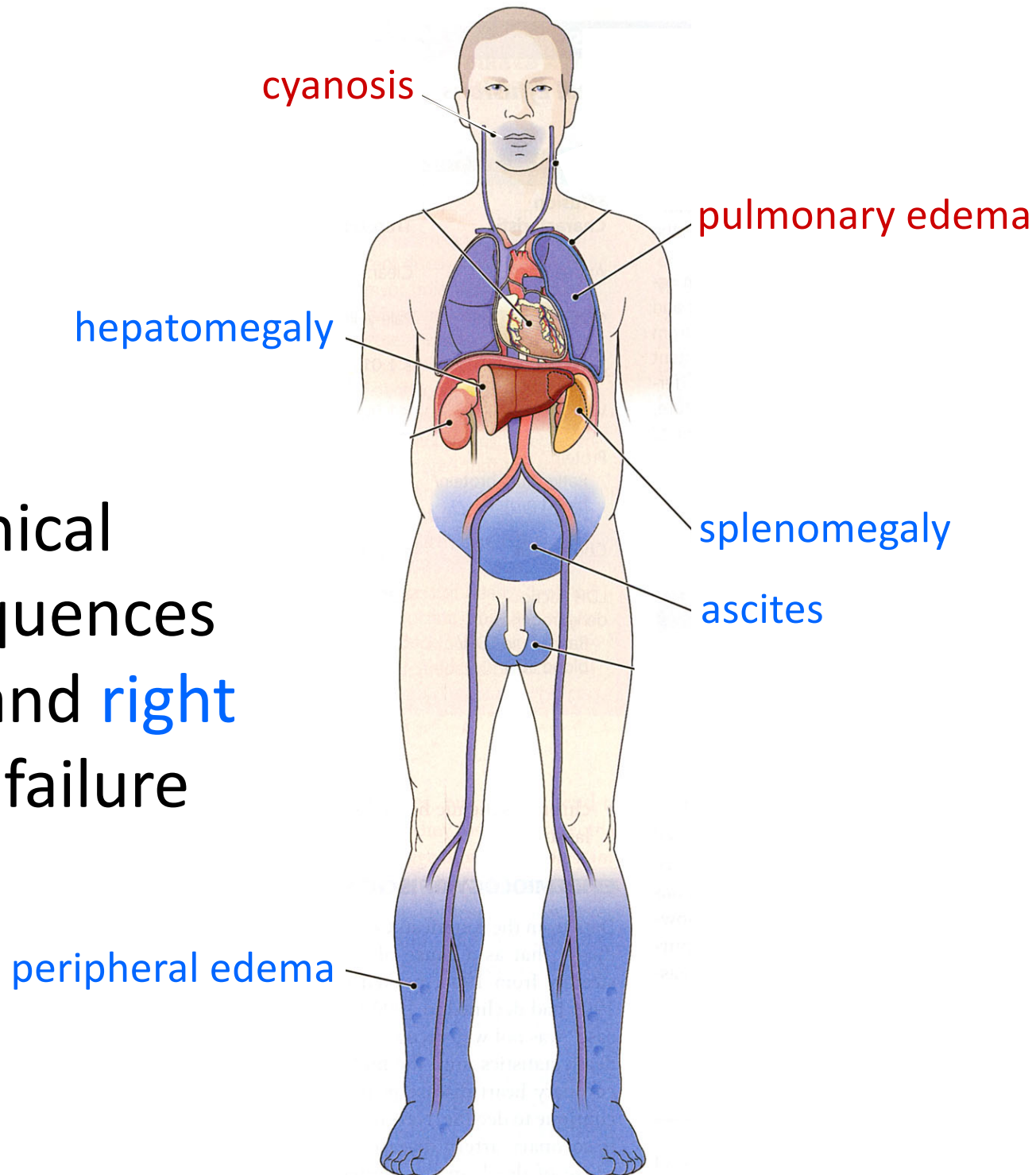
- System responds to failure by
 - Releasing hormones (e.g., norepinephrine)
 - Frank-Starling mechanism (increased stretching of heart muscle cells)
 - Hypertrophy
- Initially, this works
- Eventually, it doesn't
 - Myocytes degenerate
 - Heart needs more oxygen
 - Myocardium becomes vulnerable to ischemia

R



L

Clinical
consequences
of **left** and **right**
heart failure



Left Heart Failure

- Left ventricle fails; blood backs up in lungs
- Most common causes
 - Ischemic heart disease (IHD)
 - Hypertension
- Heart changes
 - LV hypertrophy, dilation
 - LA may be enlarged too (risk of atrial fibrillation)

Left Heart Failure

Symptoms

- Dyspnea, orthopnea, paroxysmal nocturnal dyspnea
- Crackles or rales in lungs (pulmonary edema)
- Later: mitral regurgitation, systolic murmur
- If atrium is big, “irregularly irregular” heartbeat

Right Heart Failure

- Right ventricle fails; blood backs up in body
- Commonest causes
 - Left heart failure
 - Lung disease (“cor pulmonale”)
 - Some congenital heart diseases
- Heart changes
 - right ventricular hypertrophy, dilation
 - right atrial enlargement

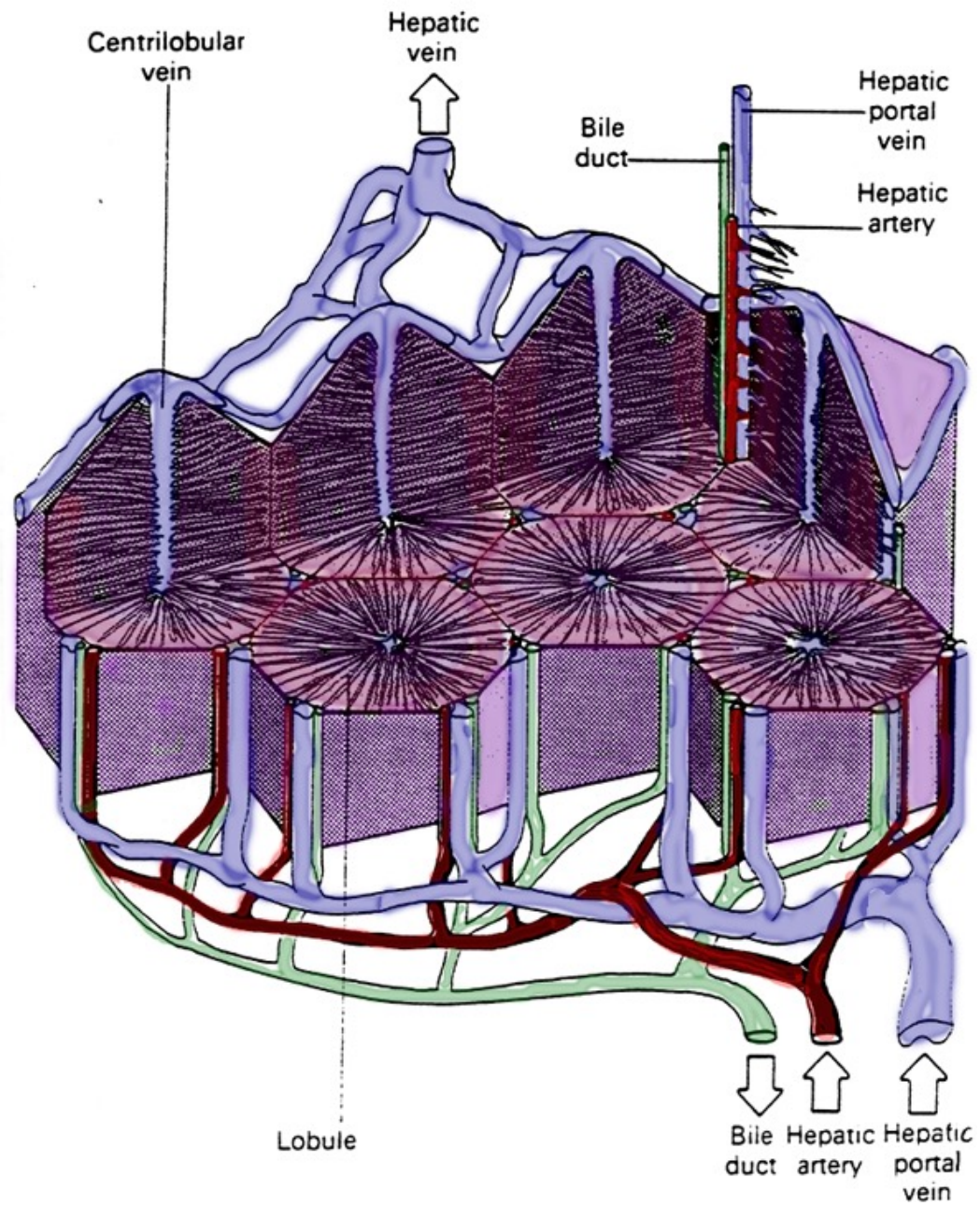


Definition:
Right heart failure
due to some intrinsic
lung disease

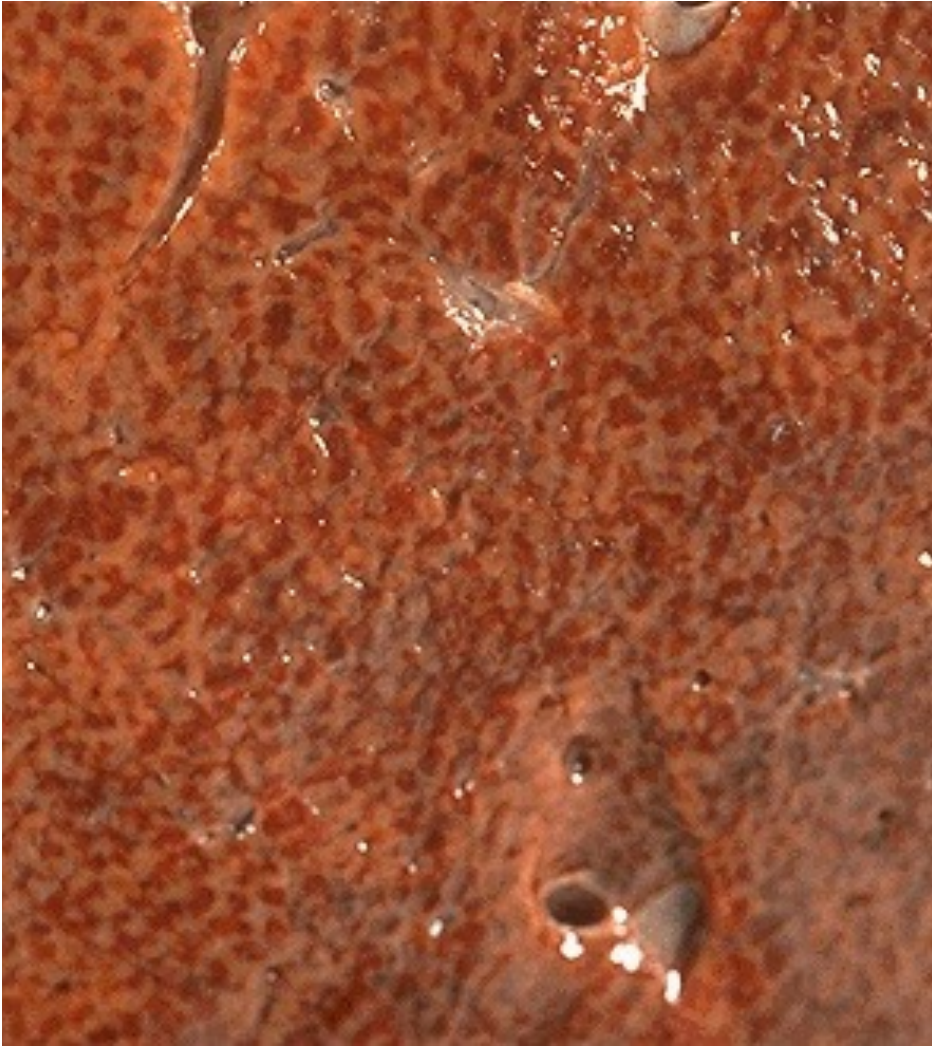
Right Heart Failure

Symptoms

- Peripheral edema
- Big, congested liver (“nutmeg liver”)
- Big spleen
- Most chronic cases of heart failure are bilateral



Hepatic blood flow



“Nutmeg” liver



Nutmeg

Cardiac Pathology Outline

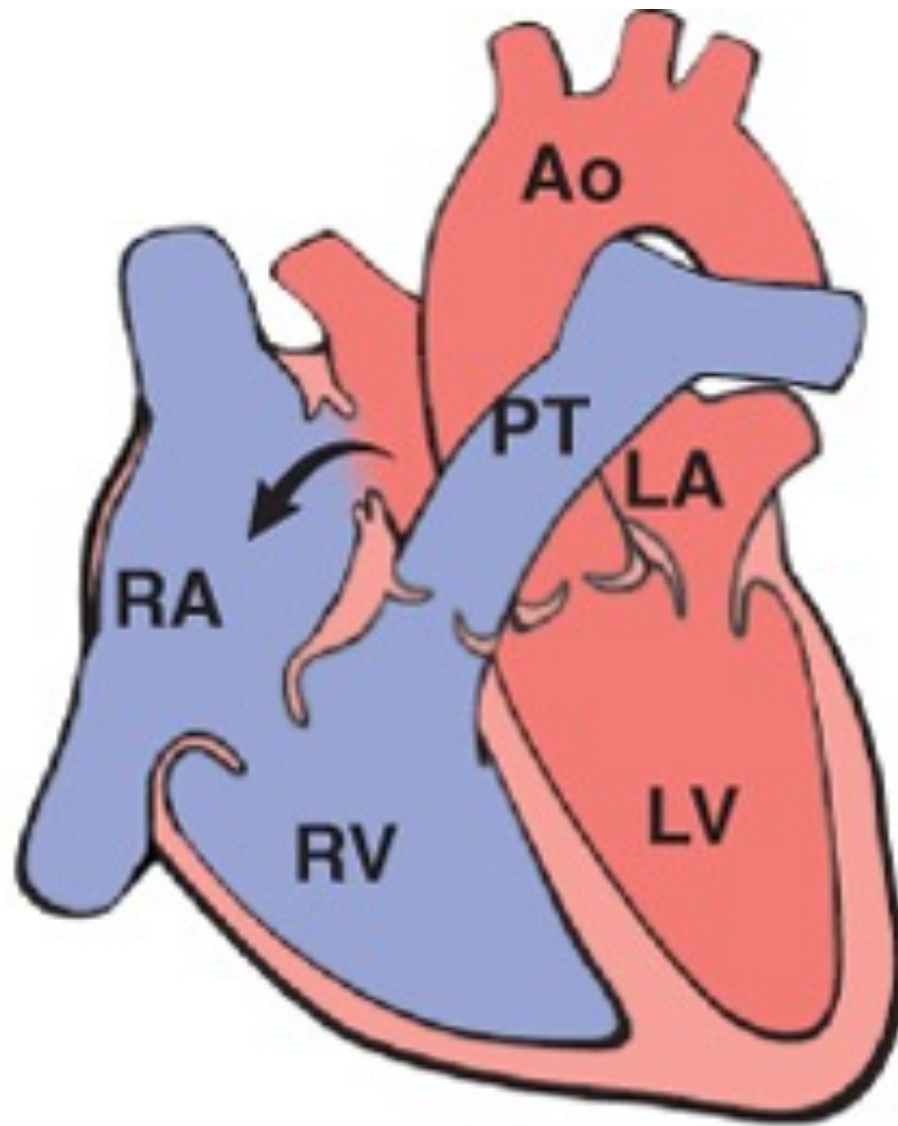
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Congenital Heart Disease

- Abnormalities of heart/great vessels present from birth
- Faulty embryogenesis, weeks 3-8
- Broad spectrum of severity
- Cause unknown in 90% of cases

Atrial Septal Defect

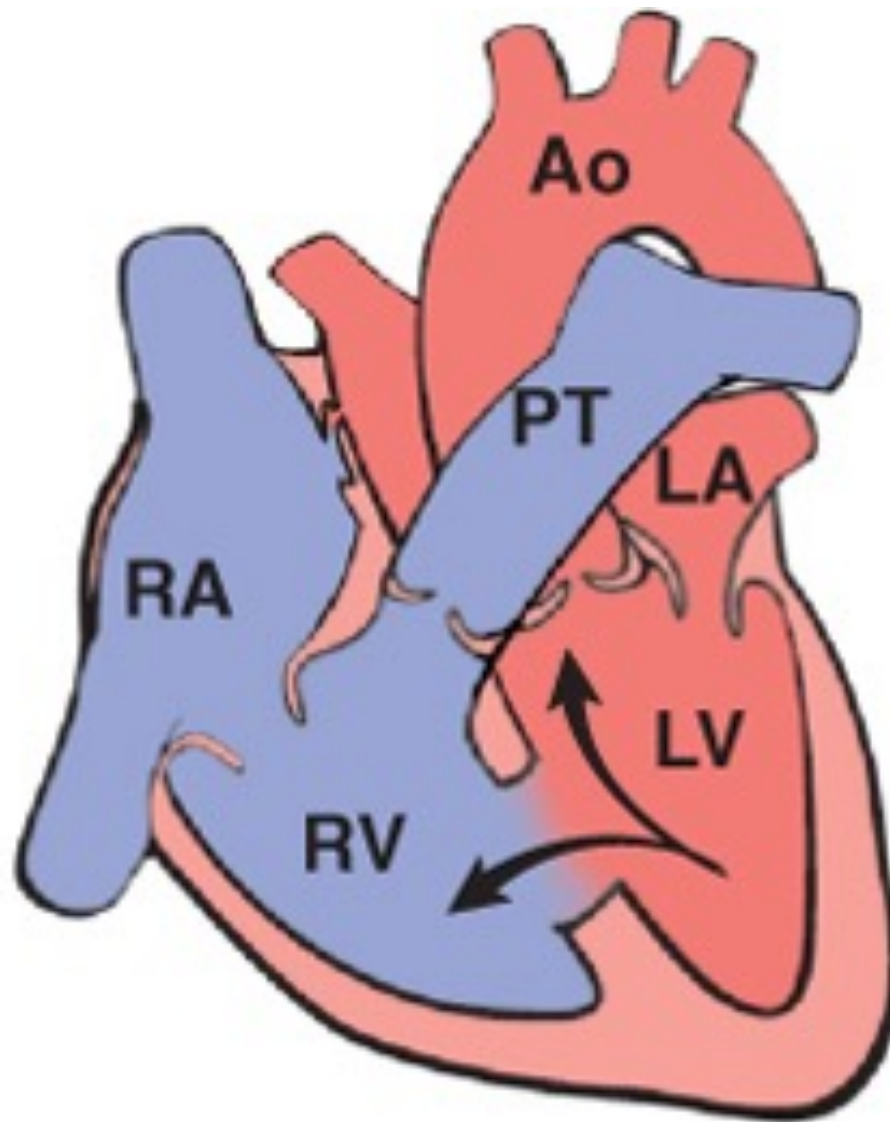
- Initially, shunt is left-to-right (asymptomatic)
- Sometimes, pulmonary vessels become constricted (pulmonary hypertension)
- Eventually, shunt can reverse, becoming right-to-left (this is called Eisenmenger syndrome)
- Surgical repair prevents irreversible pulmonary changes and heart failure



ASD

Ventricular Septal Defects

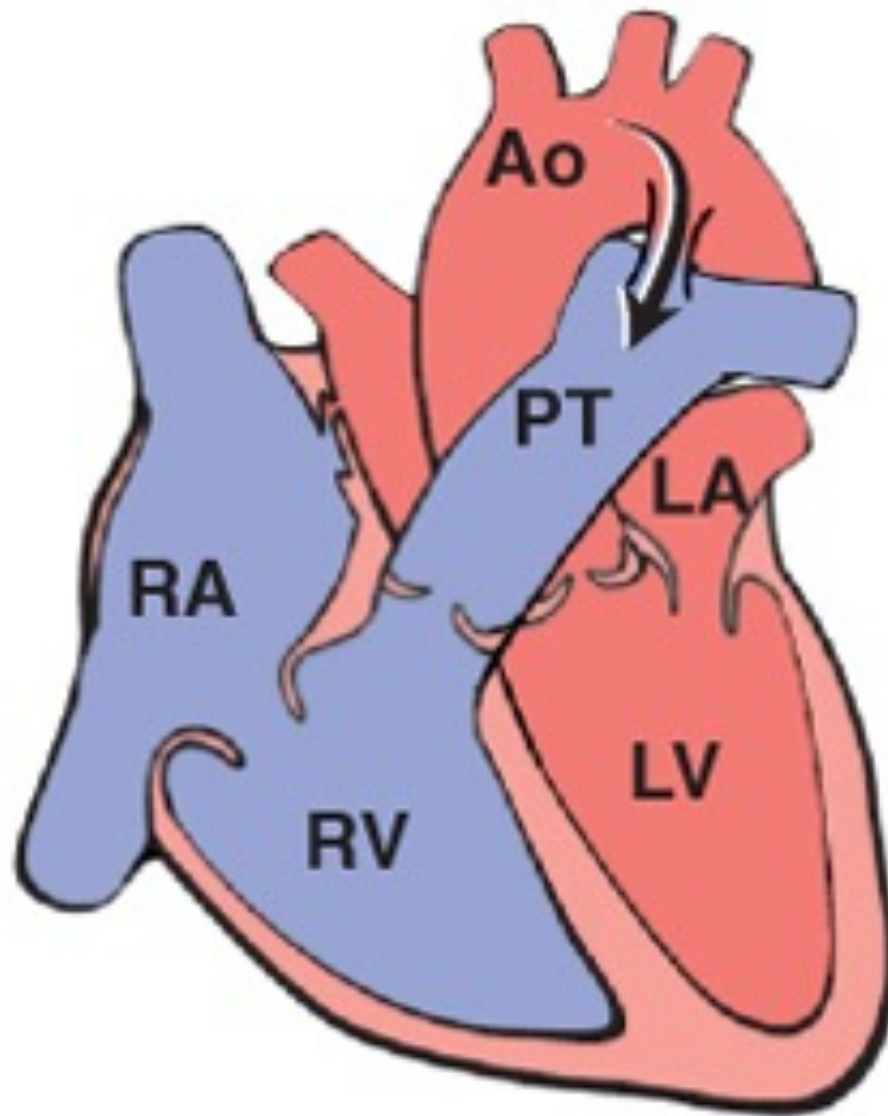
- Most common congenital cardiac anomaly
- Most close spontaneously in childhood
- Small VSD: asymptomatic
- Large VSD: big left-to-right shunt which may eventually become right-to-left



VSD

Patent Ductus Arteriosus

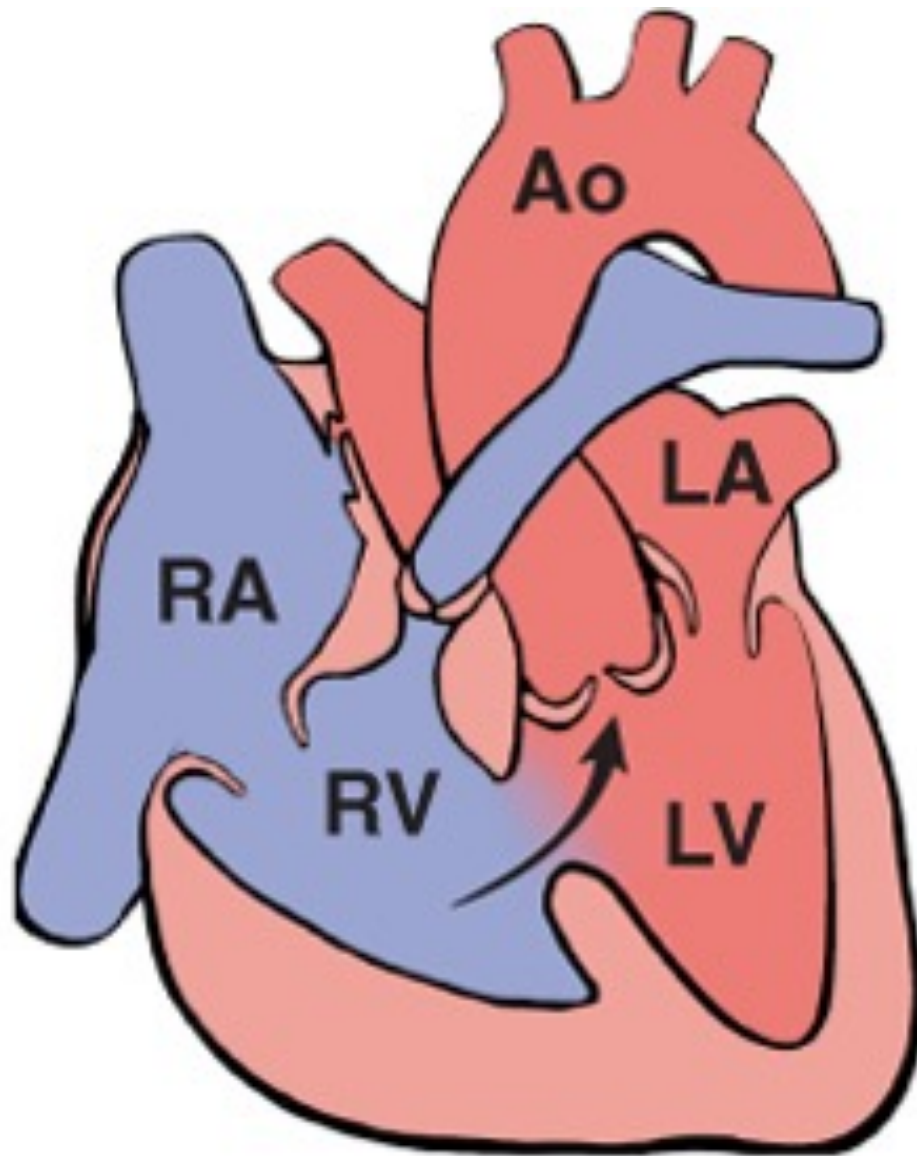
- Ductus: allows flow from PA to aorta in fetus
- Closes spontaneously by day 1-2 of life
- Small PDA: asymptomatic
- Large PDA: big left-to-right shunt which may eventually become right-to-left



PDA

Tetralogy of Fallot

- Most common cause of cyanotic congenital heart disease
- Four features:
 - VSD
 - obstruction to RV outflow tract
 - overriding aorta
 - RV hypertrophy
- Right-to-left shunt causes cyanosis, paradoxical emboli



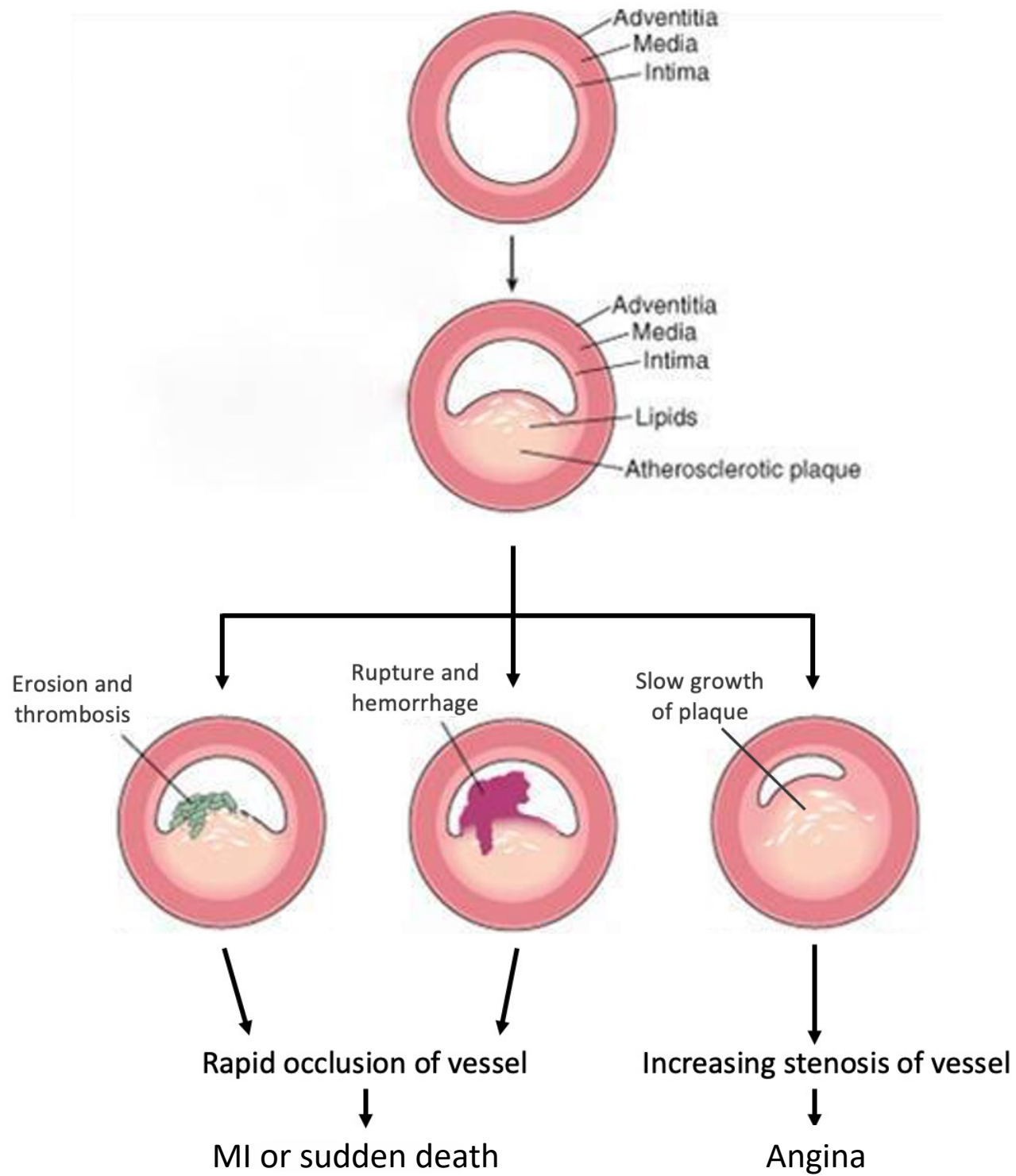
Tetralogy of Fallot

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Ischemic Heart Disease

- Myocardial perfusion can't meet demand
- Usually caused by decreased coronary artery blood flow (“coronary artery disease”)
- Three syndromes:
 - angina pectoris
 - acute MI
 - sudden cardiac death

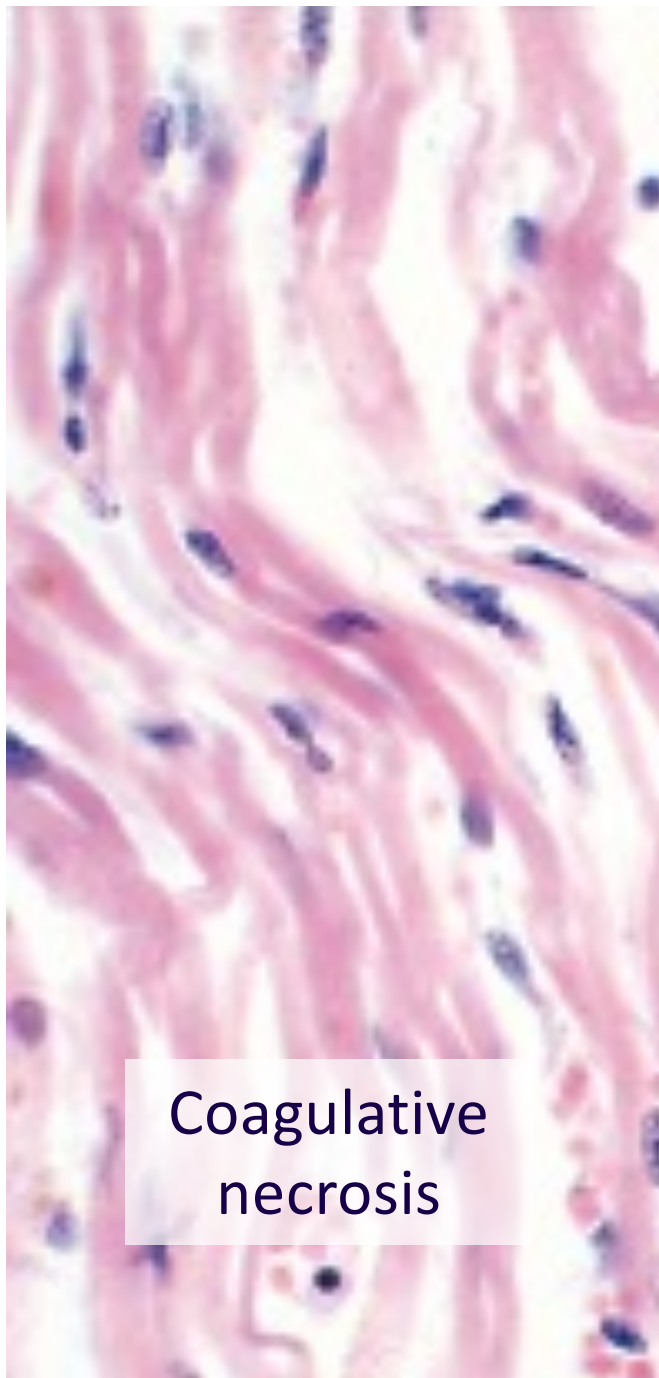


Angina Pectoris

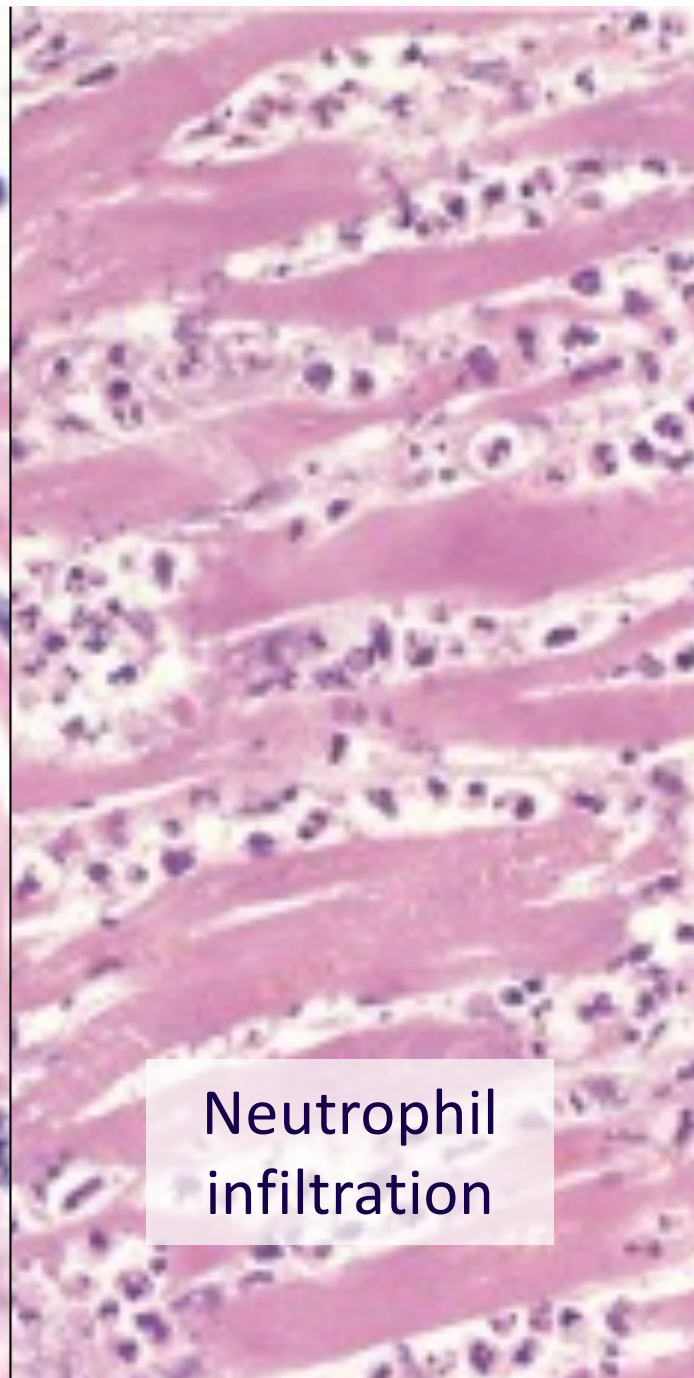
- Intermittent chest pain caused by transient, reversible ischemia
- Typical (stable) angina
 - pain on exertion
 - fixed narrowing of coronary artery
- Unstable (pre-infarction) angina
 - increasing pain with less exertion
 - plaque disruption and thrombosis

Myocardial Infarction

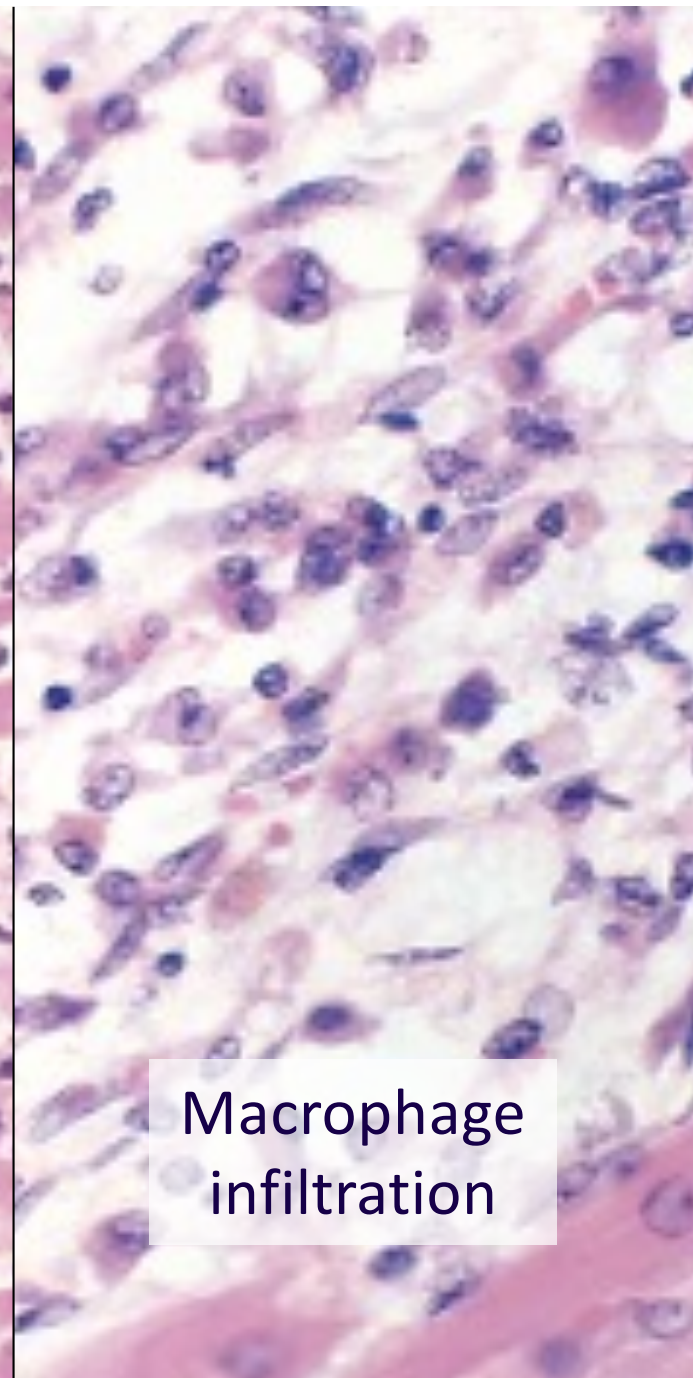
- Necrosis of heart muscle caused by ischemia
- 1.5 million people get MIs each year
- Most due to acute coronary artery thrombosis
 - sudden plaque disruption
 - platelets adhere
 - coagulation cascade activated
 - thrombus occludes lumen within minutes
 - irreversible injury/cell death in 20-40 minutes
- Prompt reperfusion can salvage myocardium



Coagulative
necrosis



Neutrophil
infiltration



Macrophage
infiltration

MI: day 1, day 3, day 7

Myocardial Infarction

- Clinical features
 - Severe, crushing chest pain $\pm$ radiation
 - Not relieved by nitroglycerin, rest
 - Sweating, nausea, dyspnea
 - Sometimes NO symptoms!
- Laboratory evaluation
 - Troponins increase within 2-4 hours, remain elevated for a week.
 - CK-MB increases within 2-4 hours, returns to normal within 72 hours.

Myocardial Infarction

- Complications
 - contractile dysfunction
 - arrhythmias
 - rupture
 - chronic progressive heart failure
- Prognosis
 - depends on remaining function and perfusion
 - overall 1 year mortality: 30%
 - 3-4% mortality per year thereafter



Rupture of papillary muscle after MI

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