

Cardiac Pathology 2:

Congenital and Ischemic
Heart Disease

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Cardiac Pathology Outline

- Blood Vessels
- Heart I
- Heart II

Cardiac Pathology Outline

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

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
- Most due to systolic dysfunction
- Some due to diastolic dysfunction, valve failure, or abnormal load
- Heart can't pump blood fast enough to meet needs of body

Heart Failure

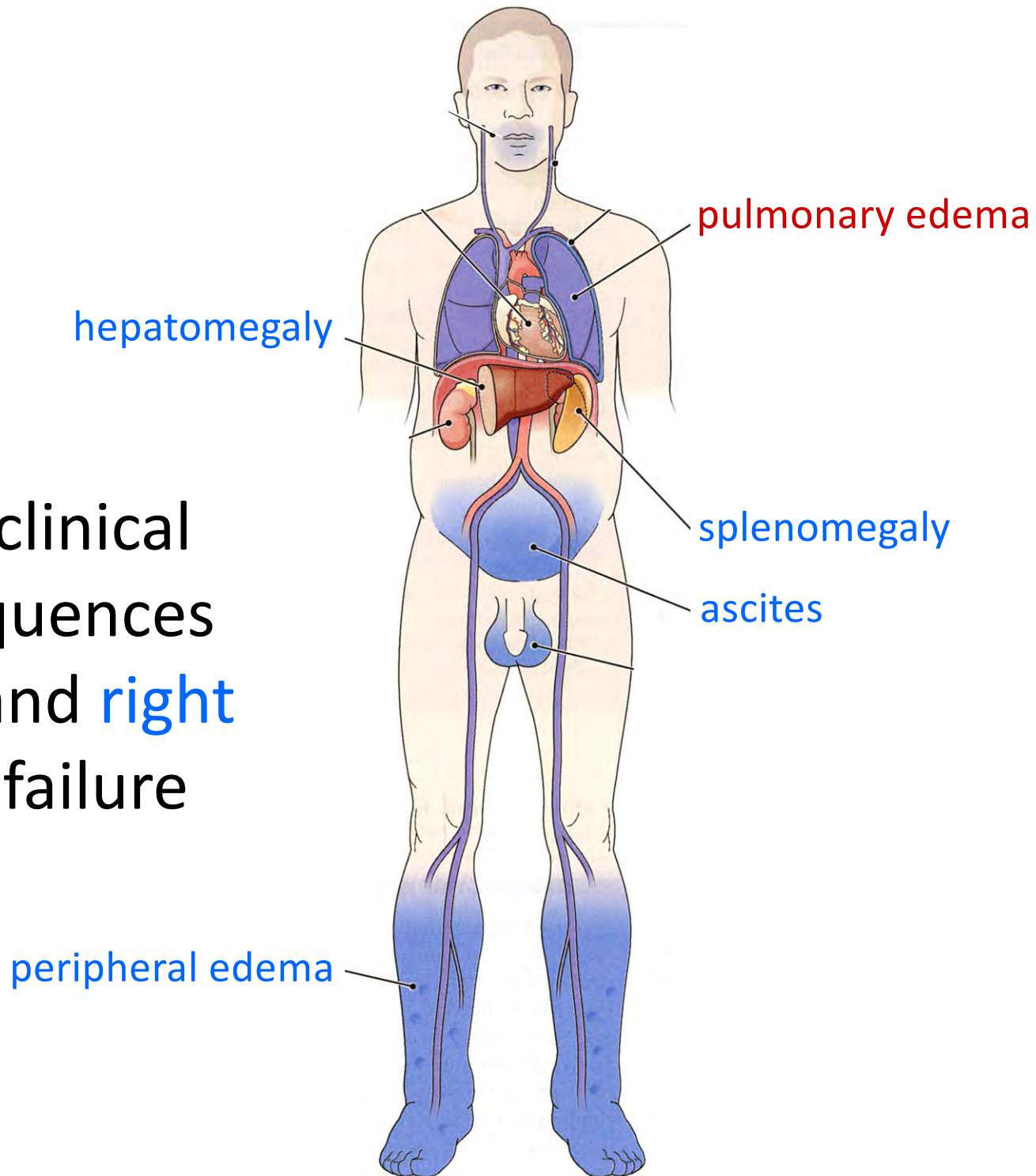
- System responds to failure by
 - Releasing hormones (e.g., norepinephrine)
 - Frank-Starling mechanism
 - Hypertrophy
- Initially, this works
- Eventually, it doesn't
 - Myocytes degenerate
 - Heart needs more oxygen
 - Myocardium becomes vulnerable to ischemia

R



L

Main clinical consequences of **left** and **right** heart failure



Left Heart Failure

- Left ventricle fails; blood backs up in lungs
- Commonest causes
 - Ischemic heart disease (IHD)
 - Systemic hypertension
 - Mitral or aortic valve disease
 - Cardiomyopathy
- Heart changes
 - LV hypertrophy, dilation
 - LA may be enlarged too (risk of atrial fibrillation)

Left Heart Failure

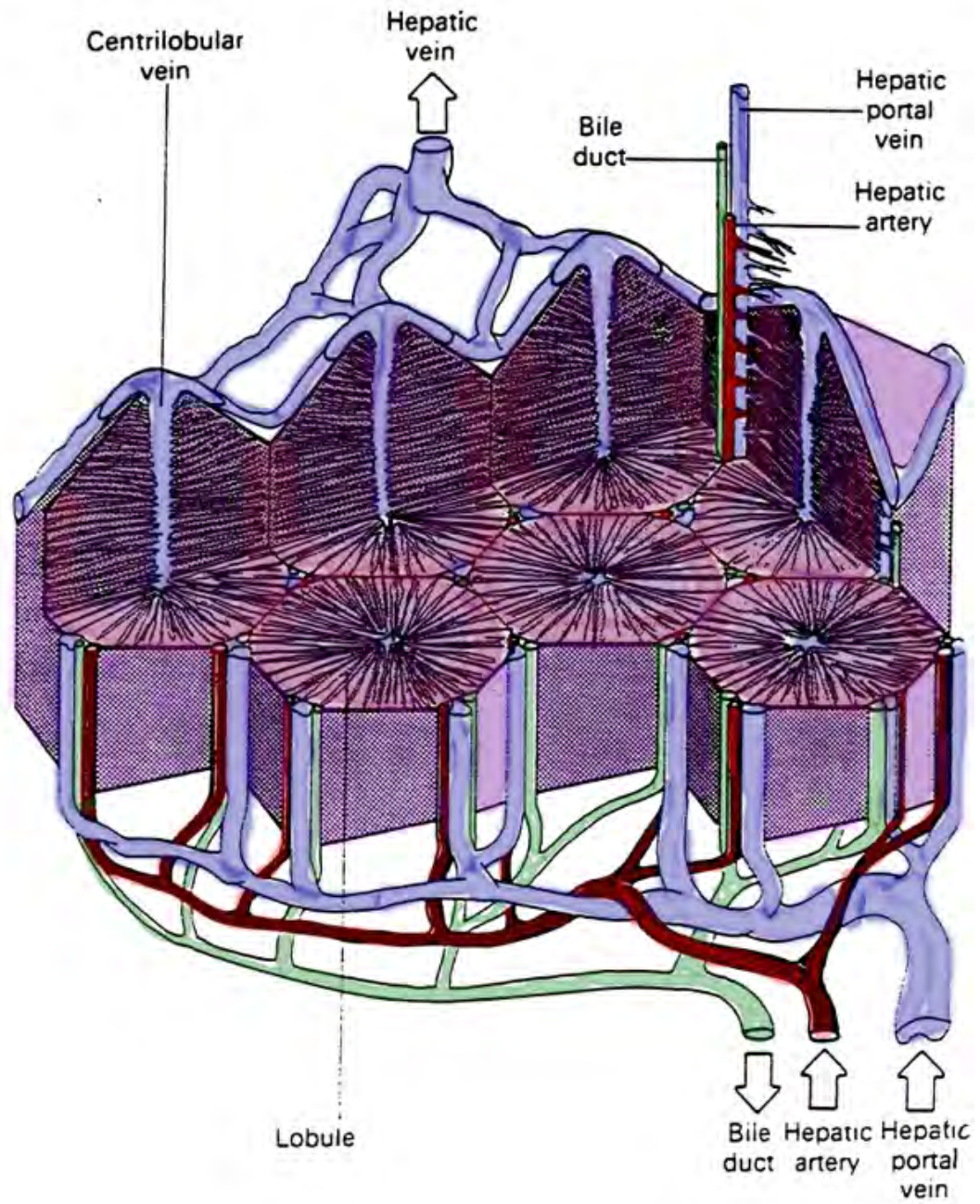
- Dyspnea
- Orthopnea and paroxysmal nocturnal dyspnea
- Enlarged heart, increased heart rate, fine rales at lung bases
- Later: mitral regurgitation, systolic murmur
- If atrium is big, “irregularly irregular” heartbeat

Right Heart Failure

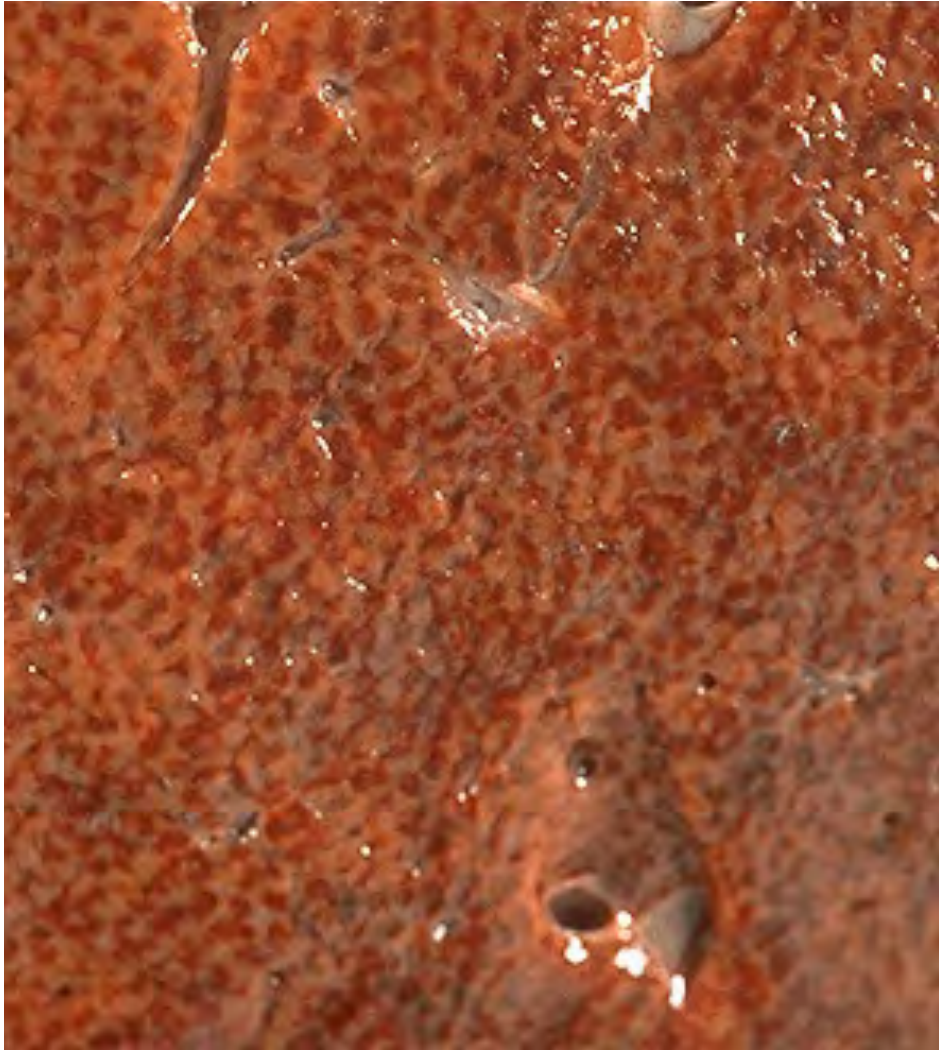
- Right ventricle fails; blood backs up in body
- Commonest causes
 - Left heart failure
 - Lung disease (“cor pulmonale”)
 - Some congenital heart diseases
- Heart changes
 - RV hypertrophy, dilation
 - RA enlargement

Right Heart Failure

- 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

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

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

Congenital Heart Disease

Left-to-right shunts

- Atrial septal defect
- Ventricular septal defect
- Patent ductus arteriosus

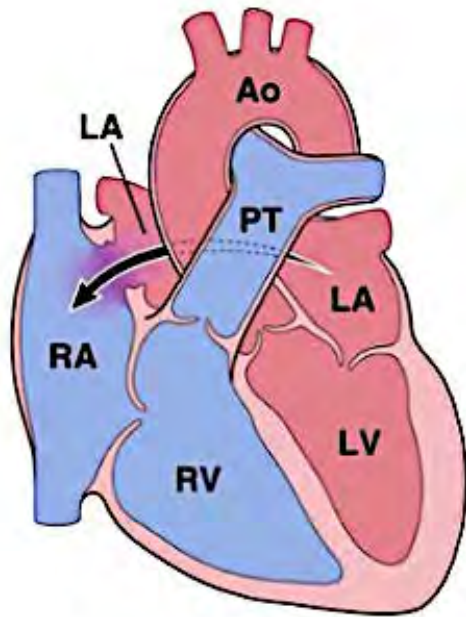
Right-to-left shunts

- Tetralogy of Fallot

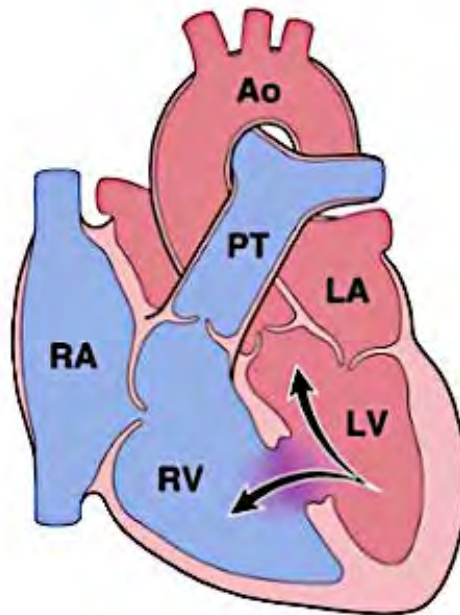
Obstructions

- Aortic coarctation

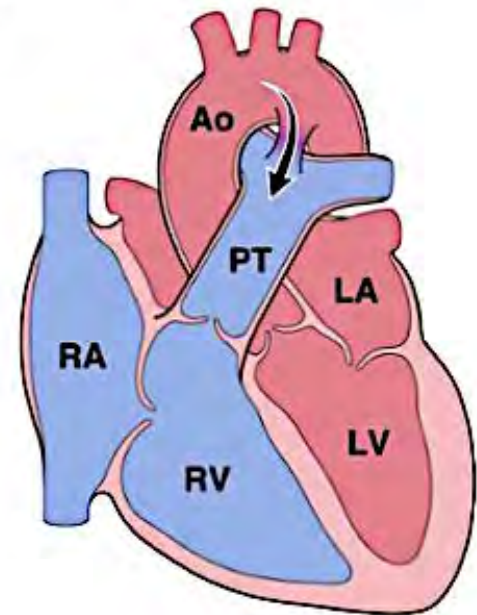
Left-to-right shunts



Atrial
septal defect

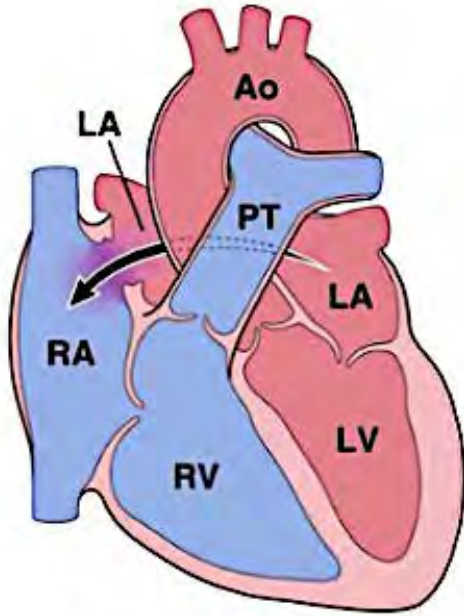


Ventricular
septal defect

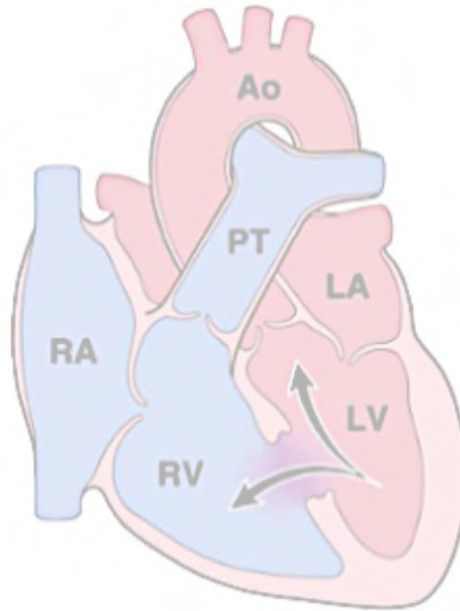


Patent
ductus arteriosus

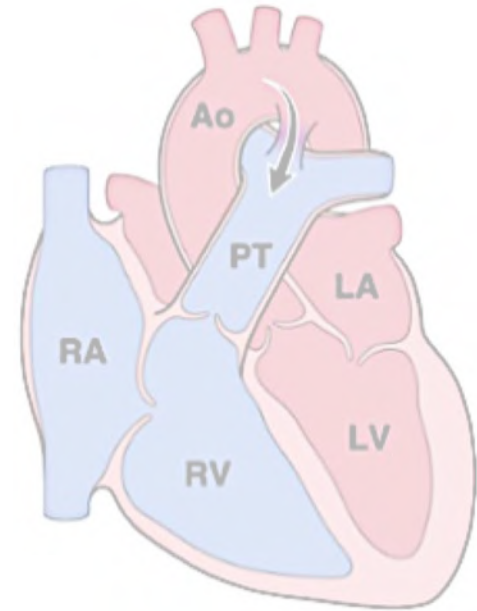
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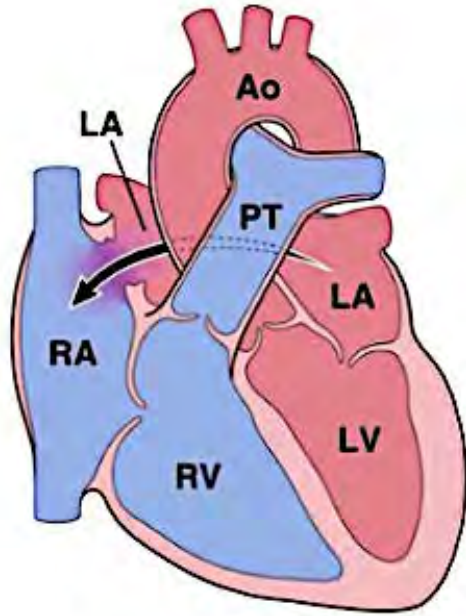
Ventricular
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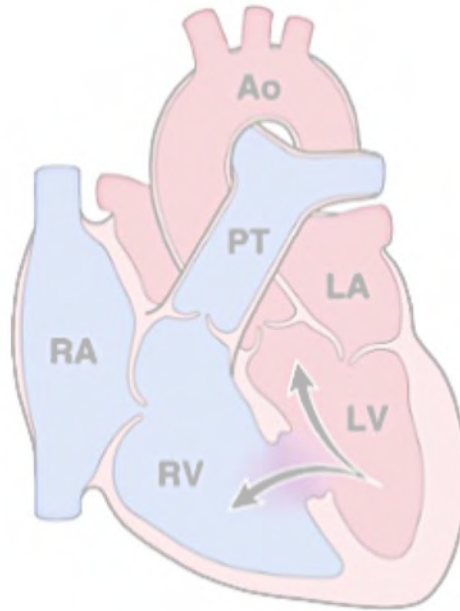
Patent
ductus arteriosus

- Small ASD: asymptomatic
- Large ASD: big left-to-right shunt
- Eventually, may develop Eisenmenger syndrome

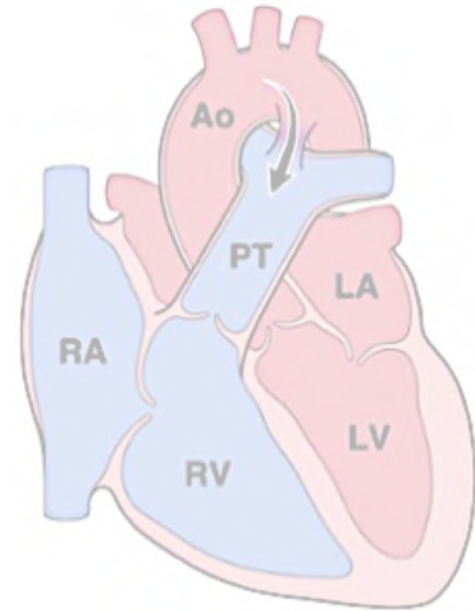
Left-to-right shunts



Atrial septal defect



Ventricular septal defect



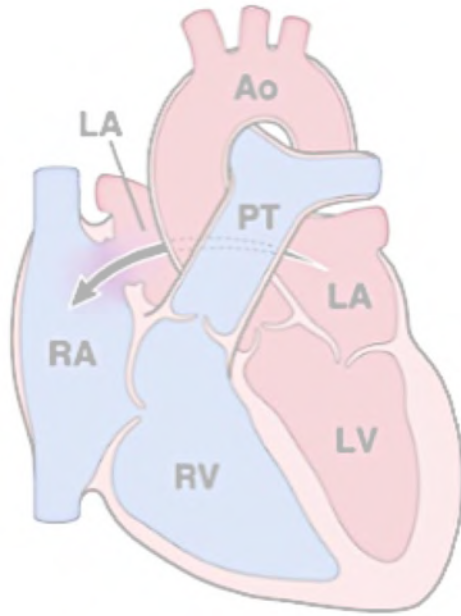
Patent ductus arteriosus

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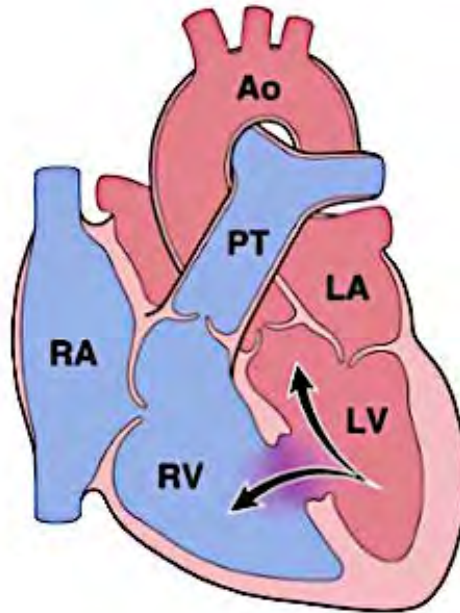
Eisenmenger syndrome:

Big left-to-right shunt puts extra pressure on pulmonary circulation. Eventually, pulmonary vessels constrict, and the **shunt reverses** (becomes right-to-left).

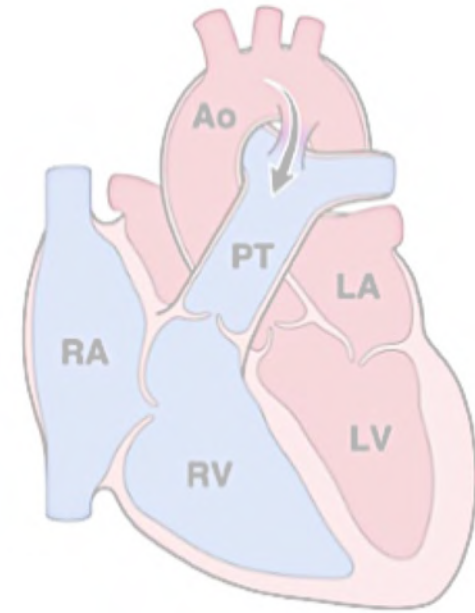
Left-to-right shunts



Atrial
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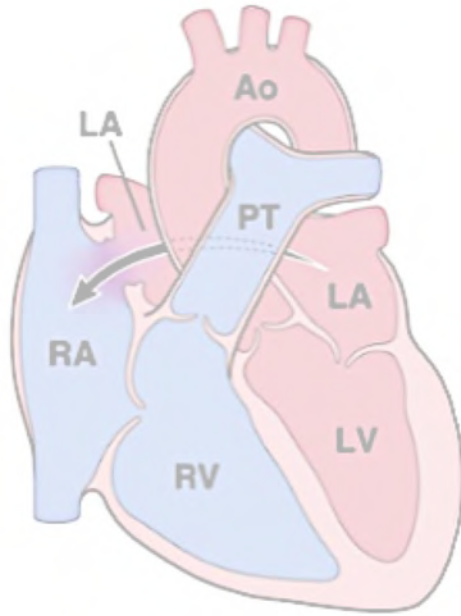
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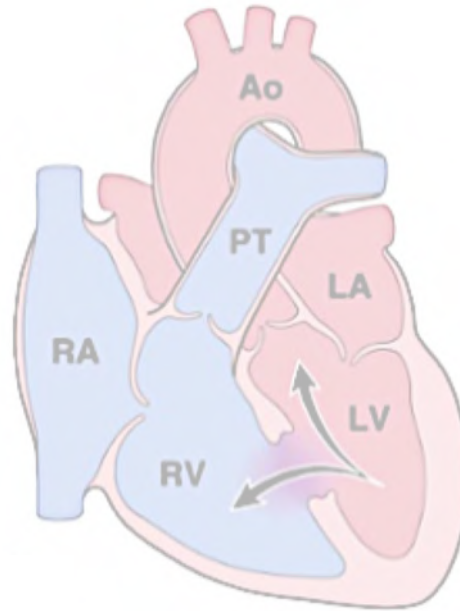
Patent
ductus arteriosus

- Most common congenital heart anomaly
- Small VSD: asymptomatic
- Large VSD: big left-to-right shunt
- Eventually, may develop Eisenmenger syndrome

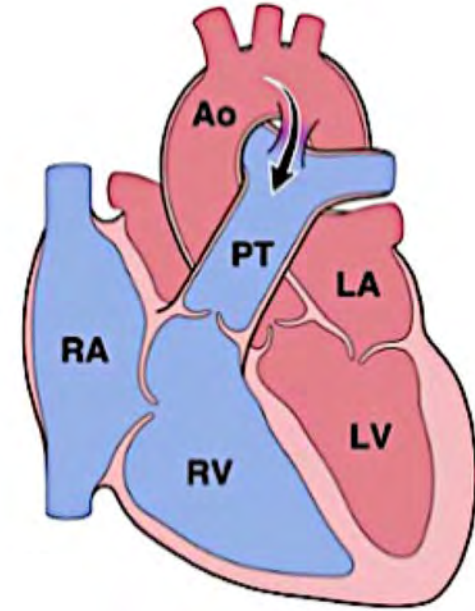
Left-to-right shunts



Atrial
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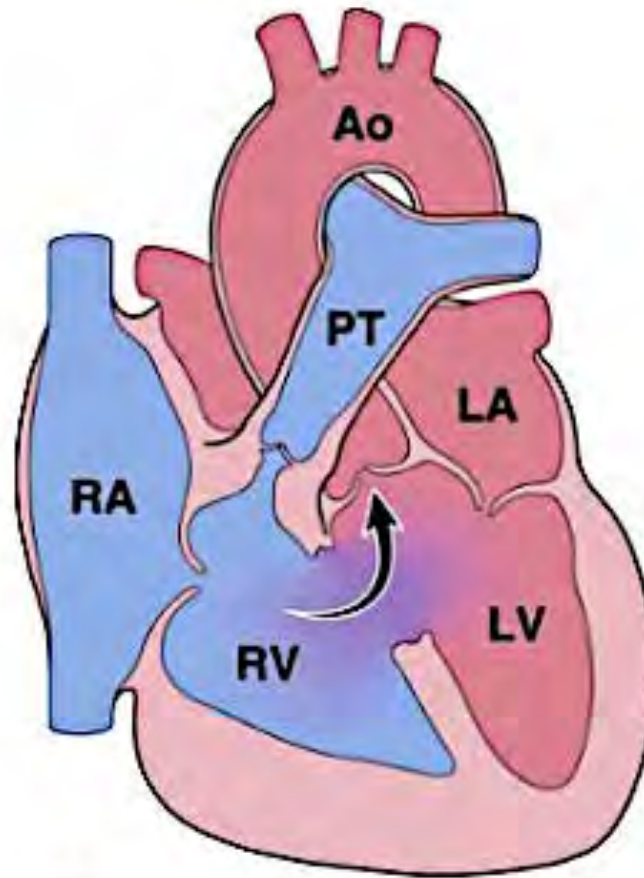
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Patent
ductus arteriosus

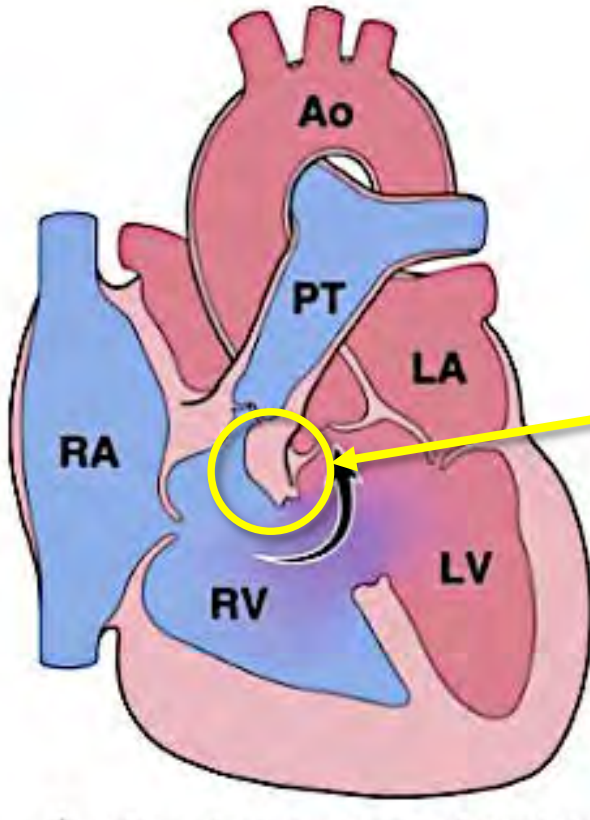
- In utero: ductus lets blood flow from PA to aorta
- After birth: ductus closes
- Small PDA: asymptomatic
- Large PDA: big left-to-right shunt
- Eventually, may develop Eisenmenger syndrome

Right-to-left shunt



Tetralogy of Fallot

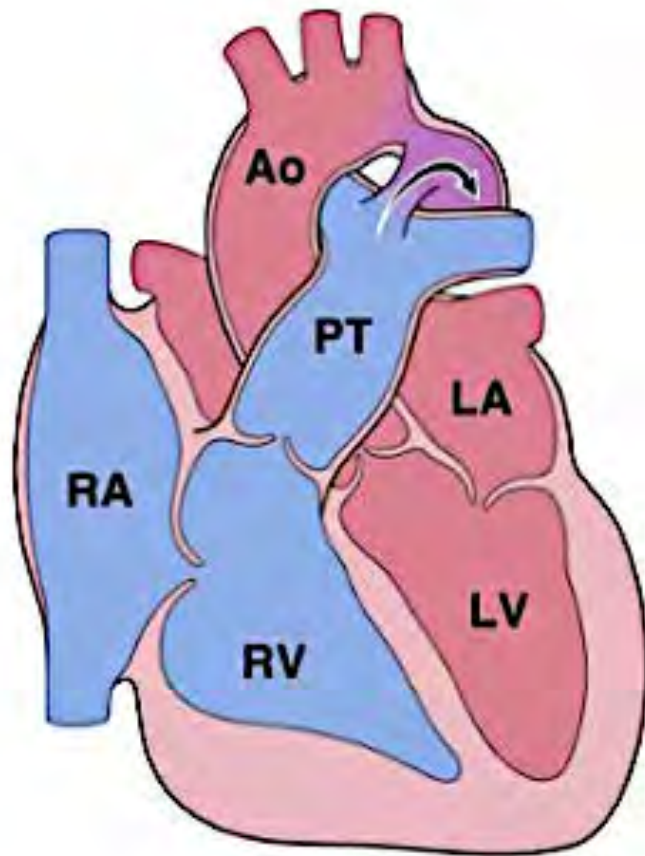
Right-to-left shunt



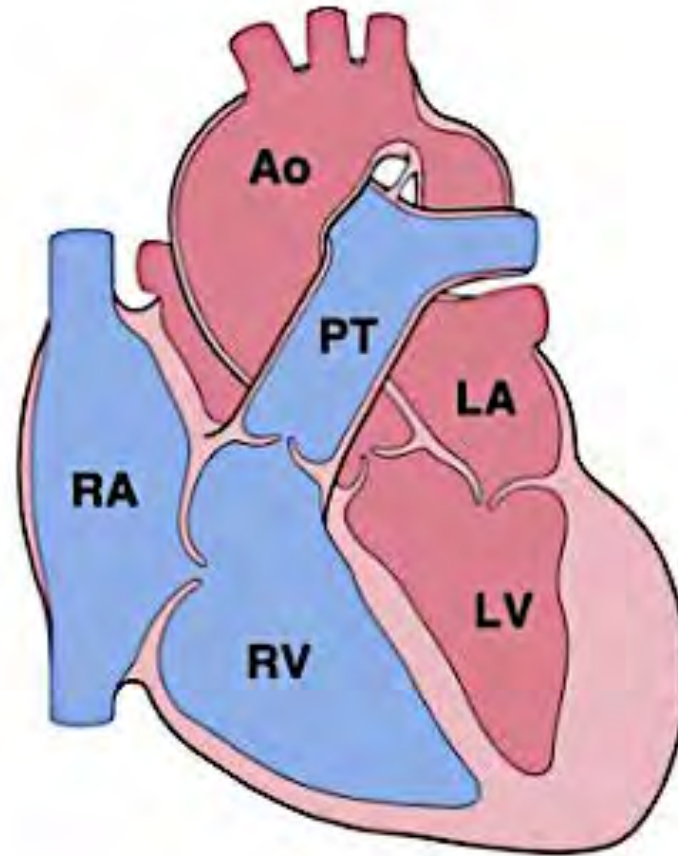
- Most common cyanotic congenital heart disease
- Main problem: infundibular septum is pushed up and to the right
- Four features:
 1. VSD
 2. Overriding aorta
 3. RV outflow obstruction
 4. RV hypertrophy

Tetralogy of Fallot

Obstruction



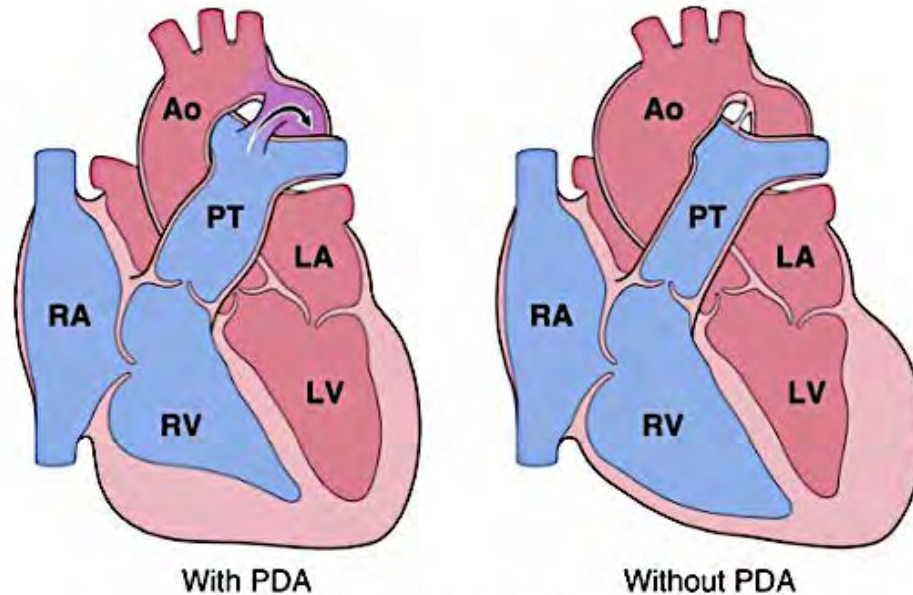
With PDA



Without PDA

Coarctation of the aorta

Obstruction



Coarctation of the aorta

- Coarctation = narrowing
- With PDA: unoxygenated blood gets dumped into aorta, causing cyanosis of lower extremities shortly after birth.
- Without PDA: hypertension of upper extremities, hypotension of lower extremities; usually asymptomatic until adulthood.

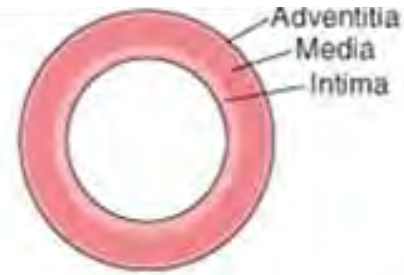
Cardiac Pathology Outline

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Ischemic Heart Disease

- Myocardial perfusion can't meet demand
- Usually caused by decreased coronary artery blood flow due to atherosclerosis (“coronary artery disease”)
- Two main clinical syndromes: angina and myocardial infarction

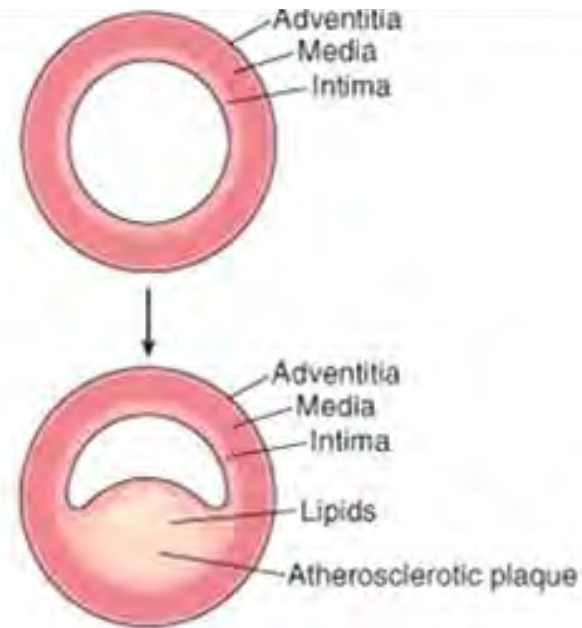
How atherosclerosis leads to angina and MI



Normal vessel

No symptoms

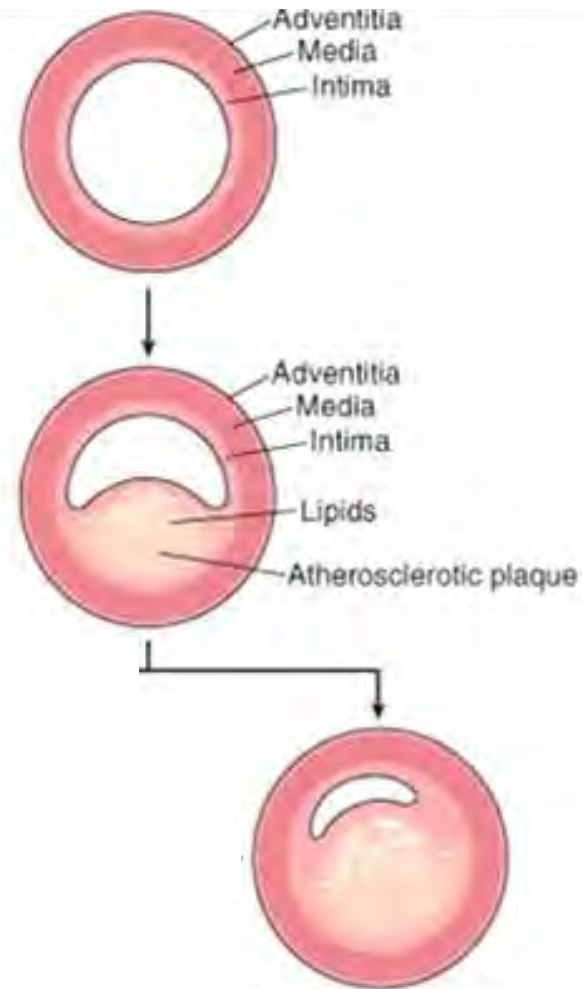
How atherosclerosis leads to angina and MI



Vessel <70% occluded by plaque

No symptoms

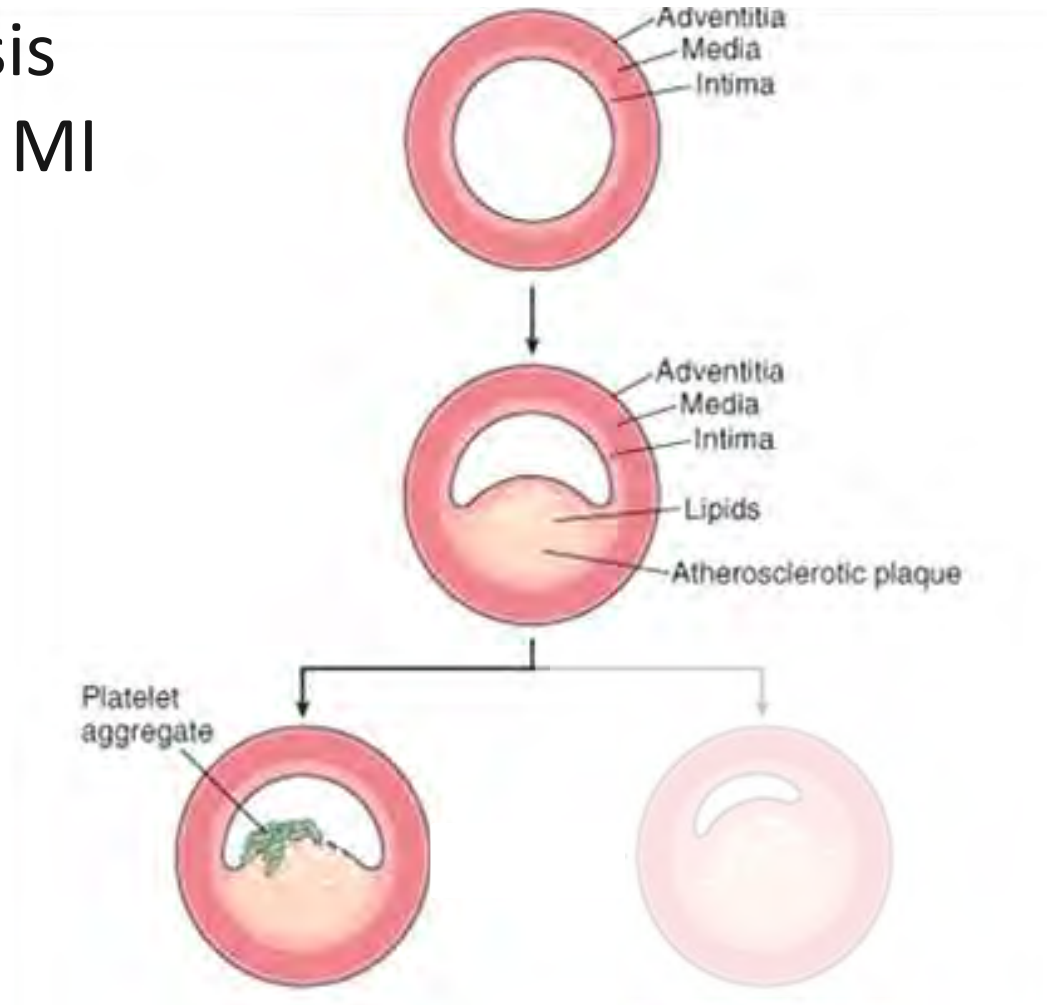
How atherosclerosis leads to angina and MI



Vessel >70% occluded by plaque

Stable angina

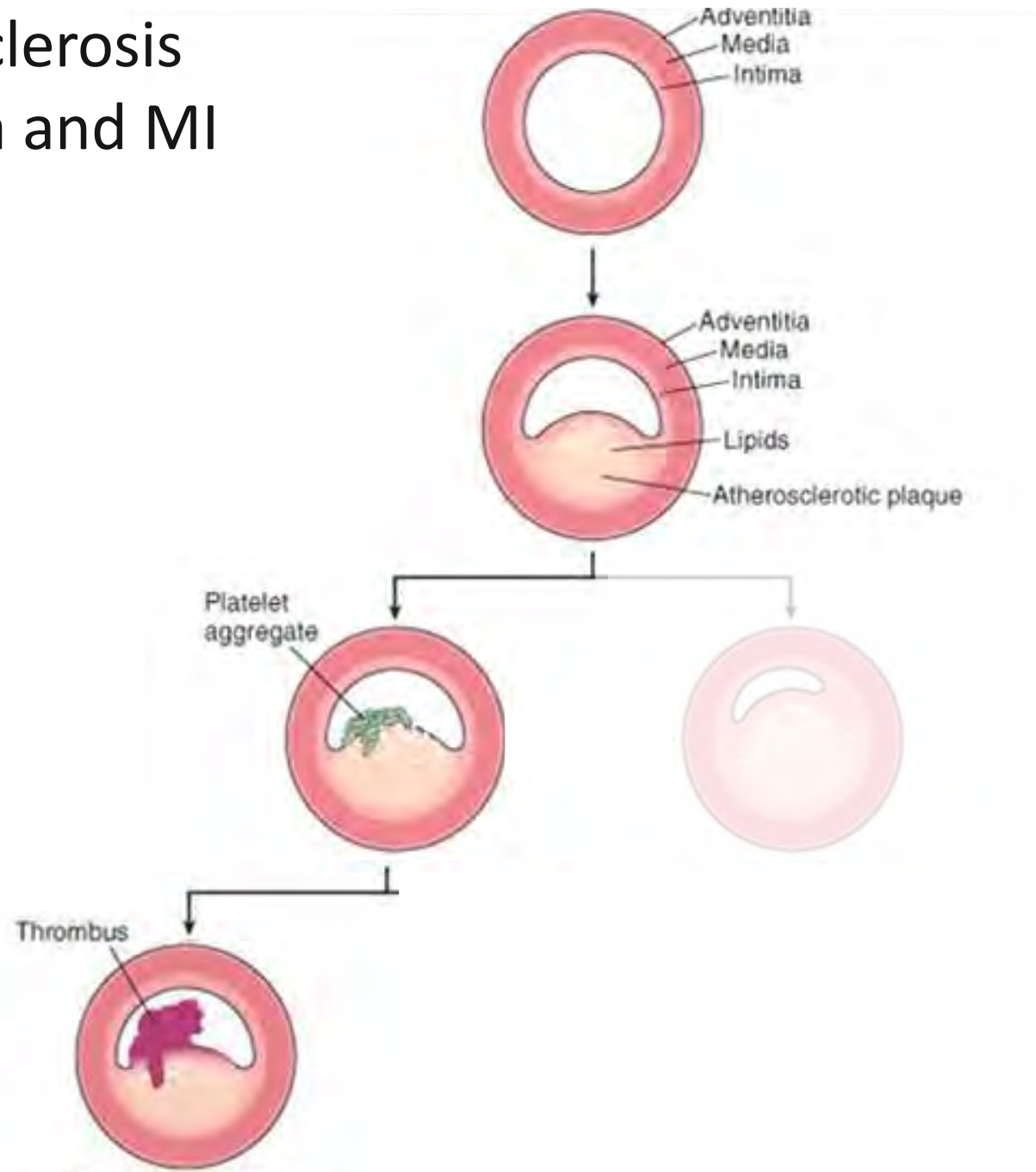
How atherosclerosis leads to angina and MI



Disrupted plaque

Uh oh

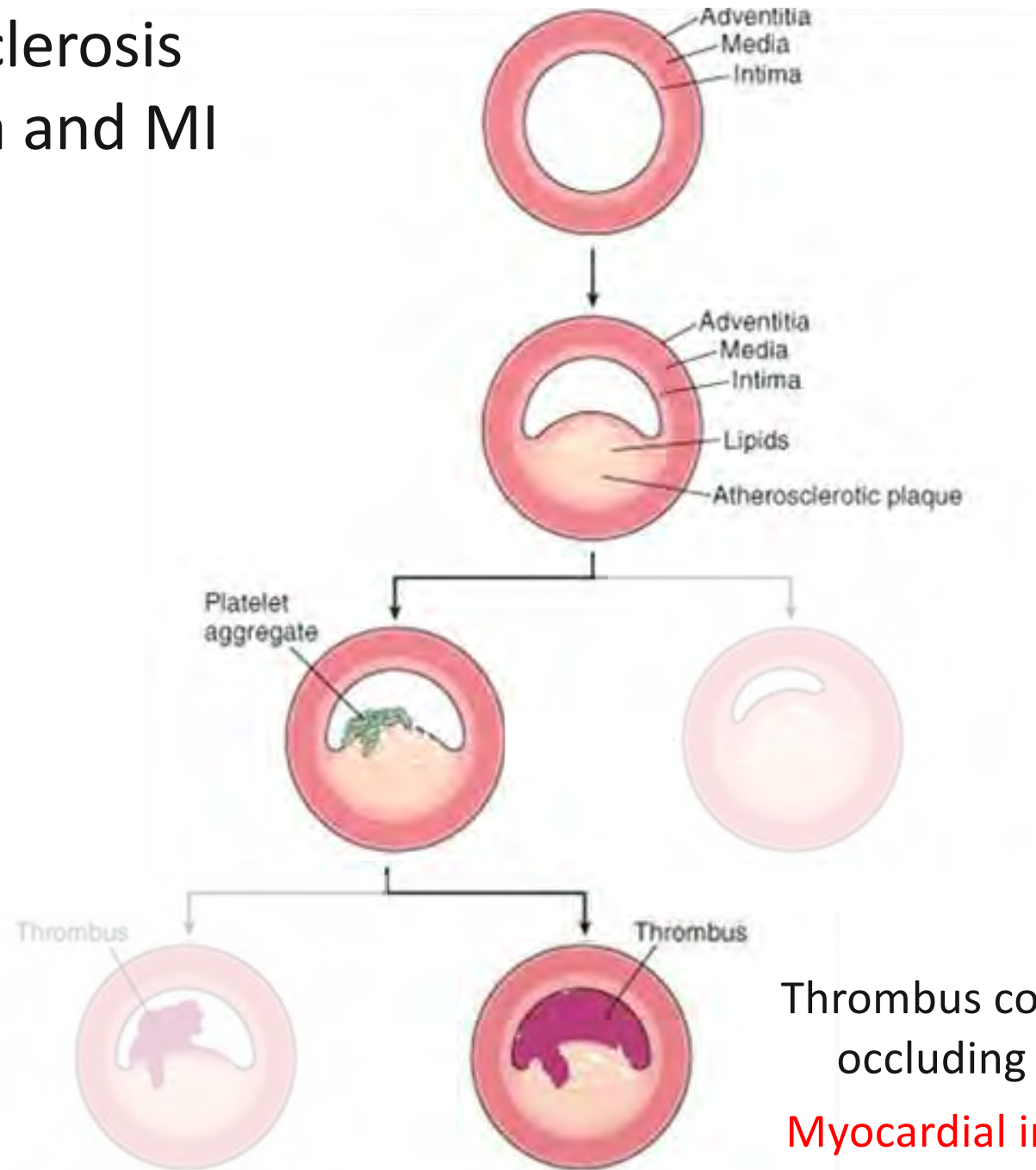
How atherosclerosis leads to angina and MI



Thrombus partially occluding vessel

Unstable angina

How atherosclerosis leads to angina and MI



Thrombus completely occluding vessel

Myocardial infarction

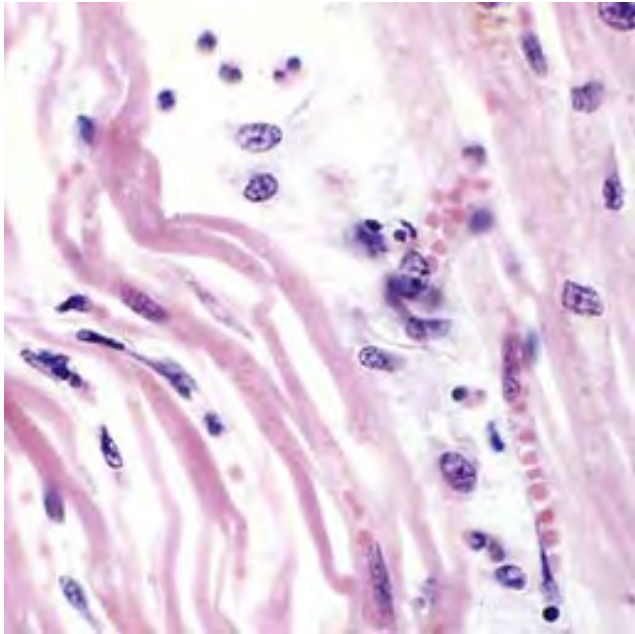
Angina Pectoris

- Intermittent chest pain caused by transient, reversible ischemia
- Stable angina
 - Pain on exertion
 - Cause: fixed narrowing of coronary artery
- Unstable angina
 - Increasing pain with less exertion
 - Cause: plaque disruption and thrombosis

Myocardial Infarction

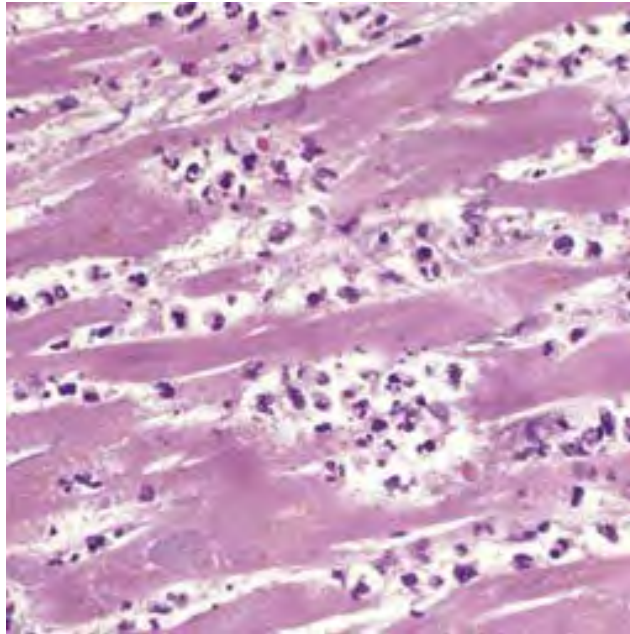
- Necrosis of heart muscle caused by ischemia
- 1.5 million people get MIs each year
- Usually due to acute coronary artery thrombosis
 - sudden plaque disruption
 - platelets adhere
 - coagulation cascade activated
 - thrombus occludes lumen within minutes
 - irreversible injury/cell death in 20-40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

What happens to the heart after an MI?



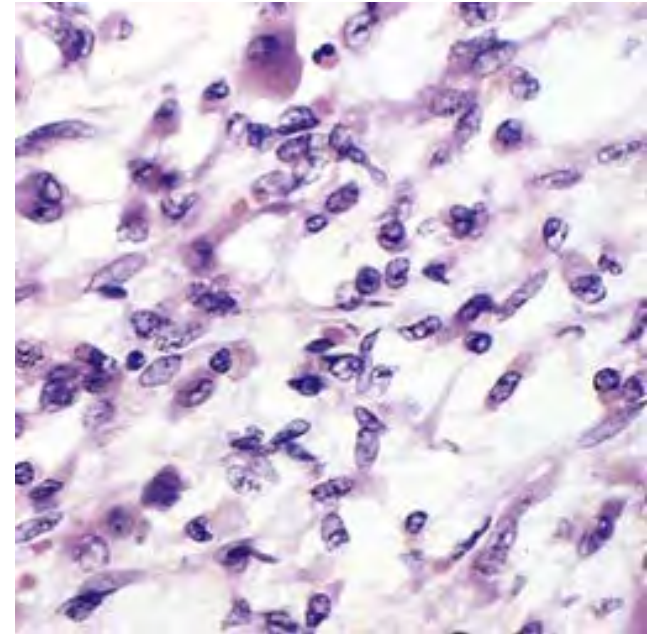
4-12 hours

Myocytes undergo
coagulative necrosis



12-24 hours

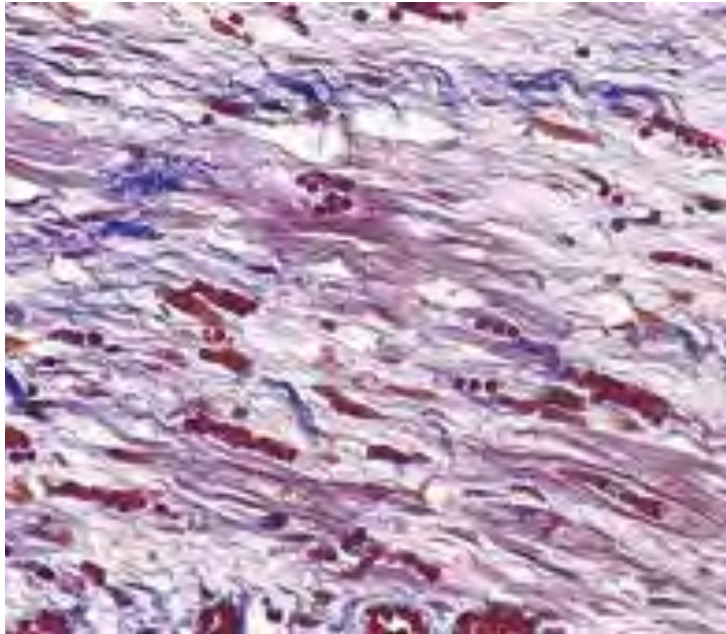
Neutrophils arrive



Days 2-7

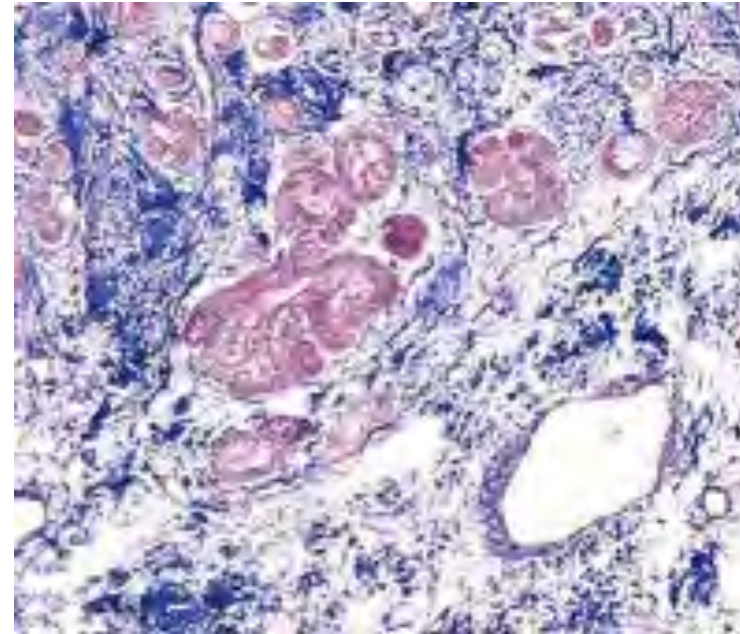
Macrophages come in
and eat dead cells;
neutrophils leave

What happens to the heart after an MI?



Week 2

Granulation tissue forms



Weeks 3-8

Granulation tissue replaced by collagen, forming a scar

Myocardial Infarction

Clinical features

- Severe, crushing chest pain \pm radiation
- Not relieved by nitroglycerin, rest
- Sweating, nausea, dyspnea
- Sometimes no symptoms

Serum markers

- 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

- overall 1 year mortality: 30%
- 3-4% mortality per year thereafter