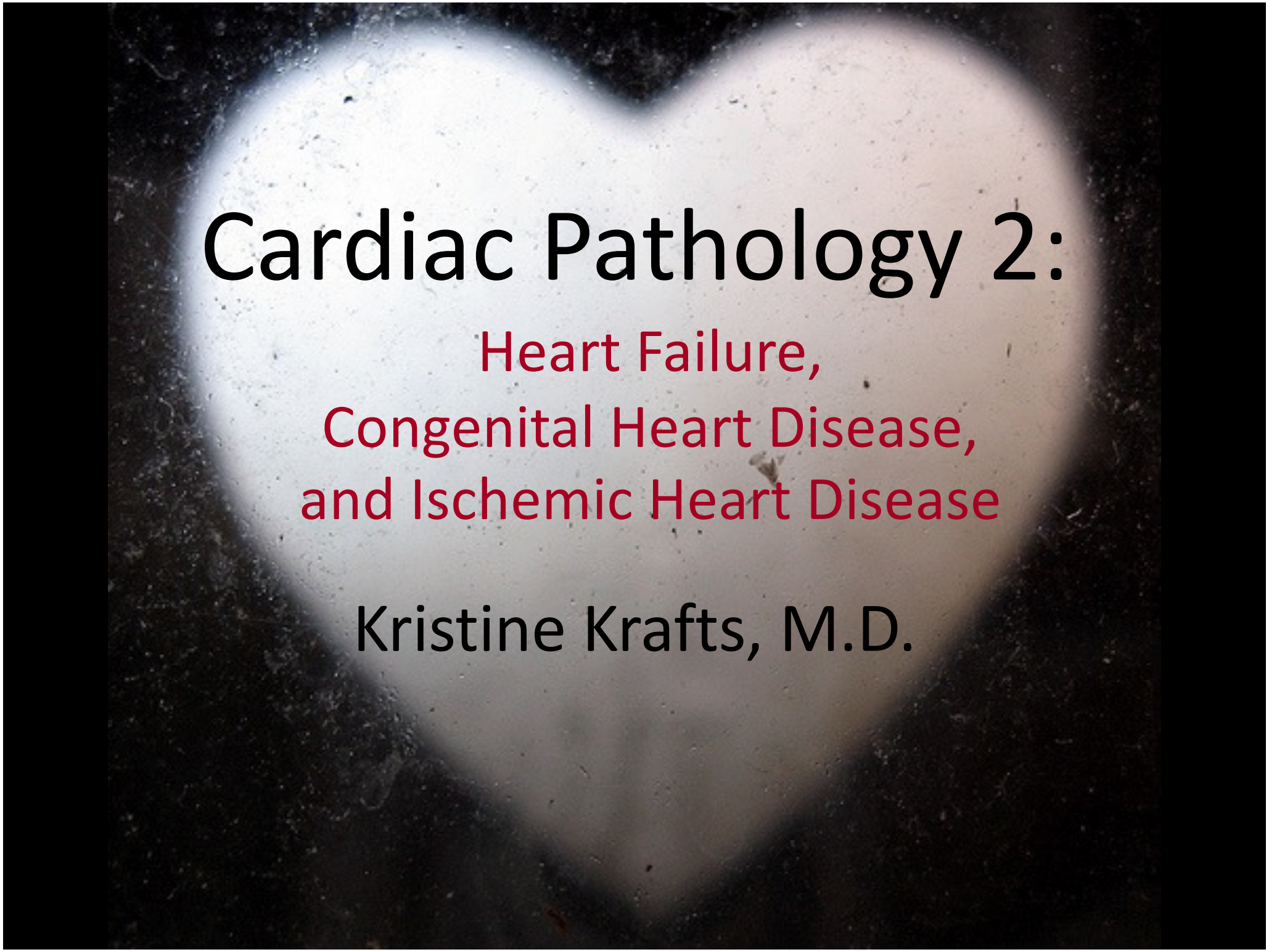


Cardiac Pathology 2:

Heart Failure, Congenital Heart Disease, and Ischemic Heart Disease

Kristine Krafts, M.D.

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

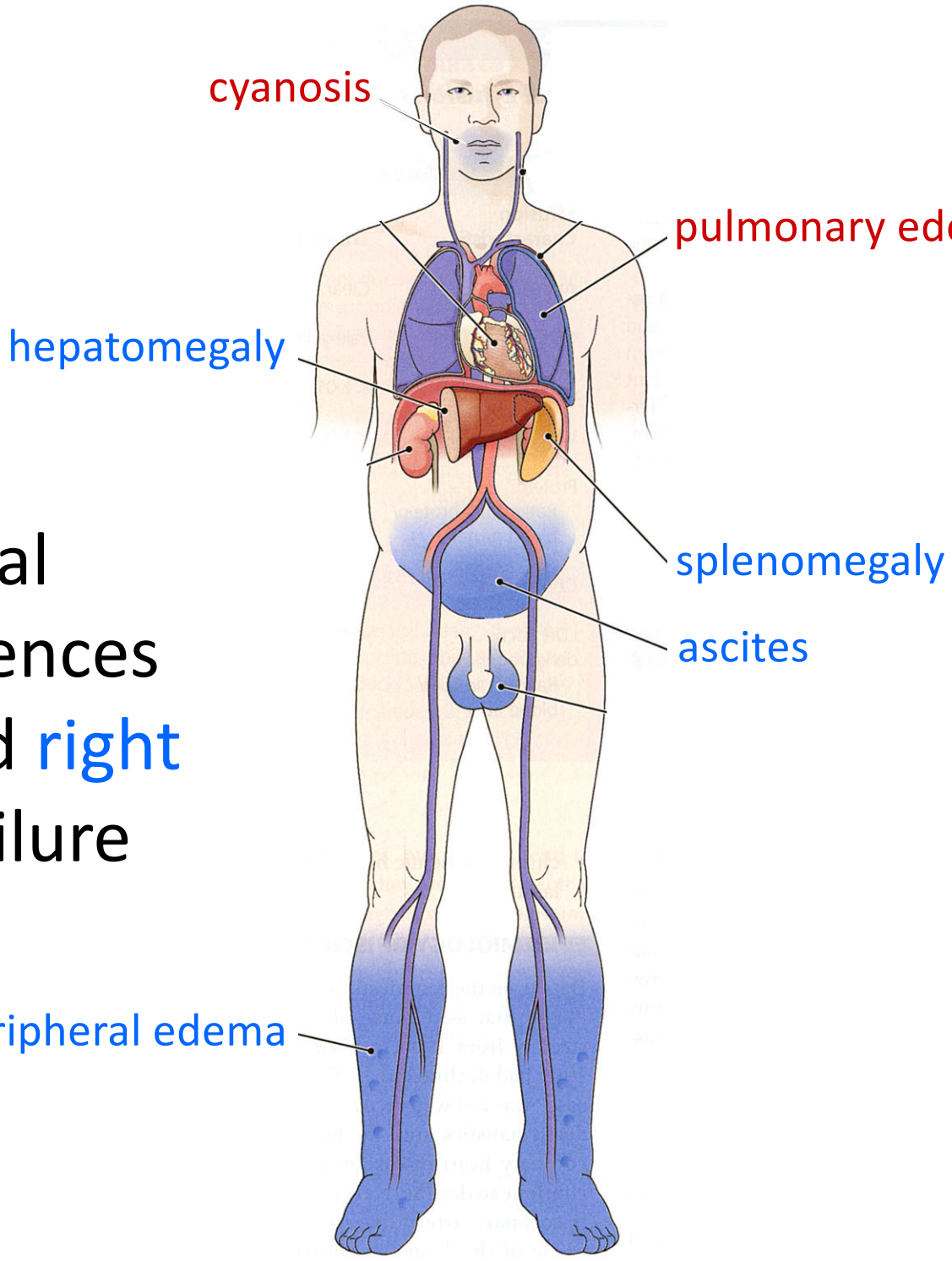
Heart Failure

- 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

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

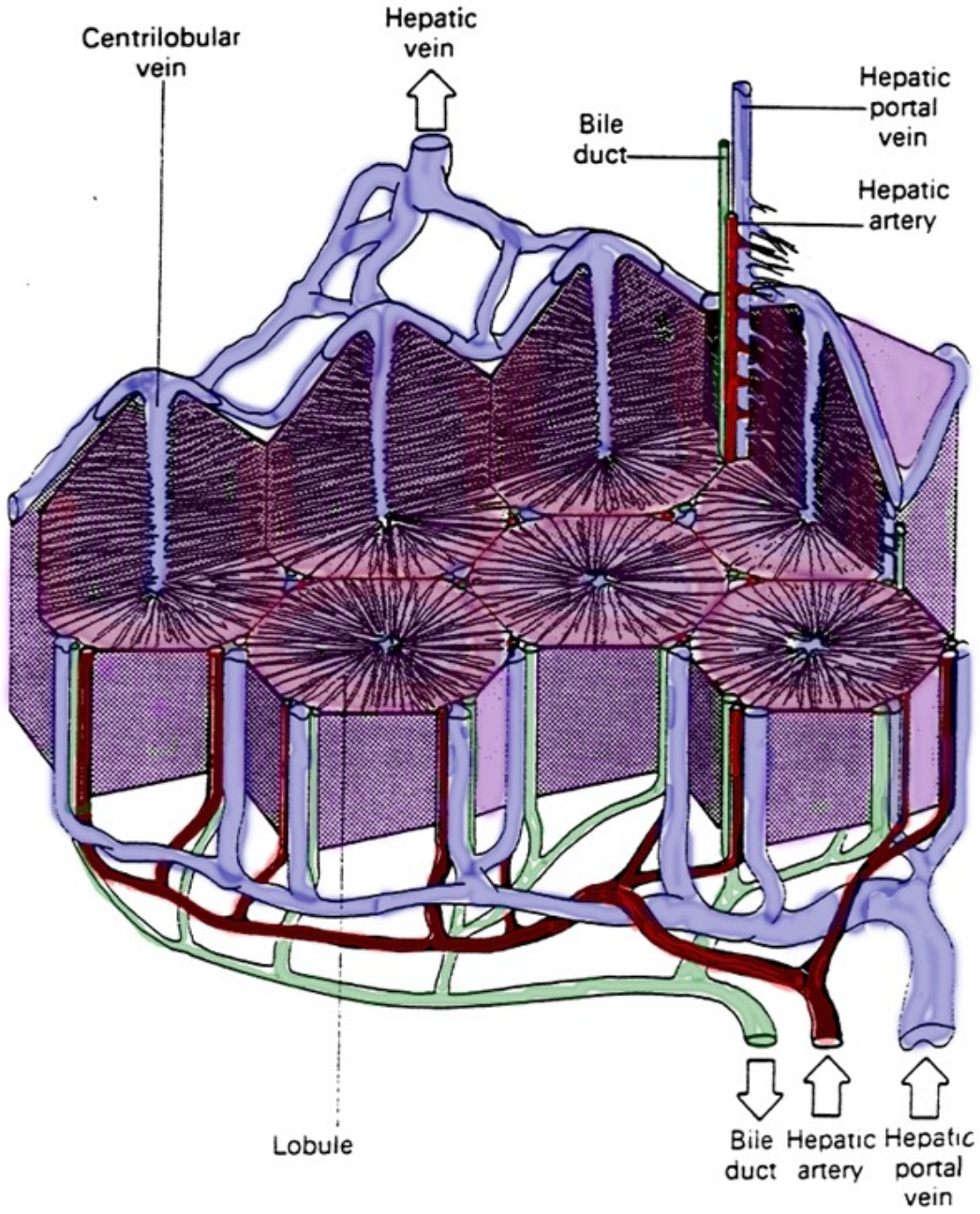
- Right ventricle fails; blood backs up in body
- Commonest causes
  - Left heart failure
  - Lung disease ("cor pulmonale")
  - Some congenital heart diseases
- Heart changes
  - right ventricular hypertrophy, dilation
  - right atrial enlargement

Definition:
Right heart failure due to some intrinsic lung disease

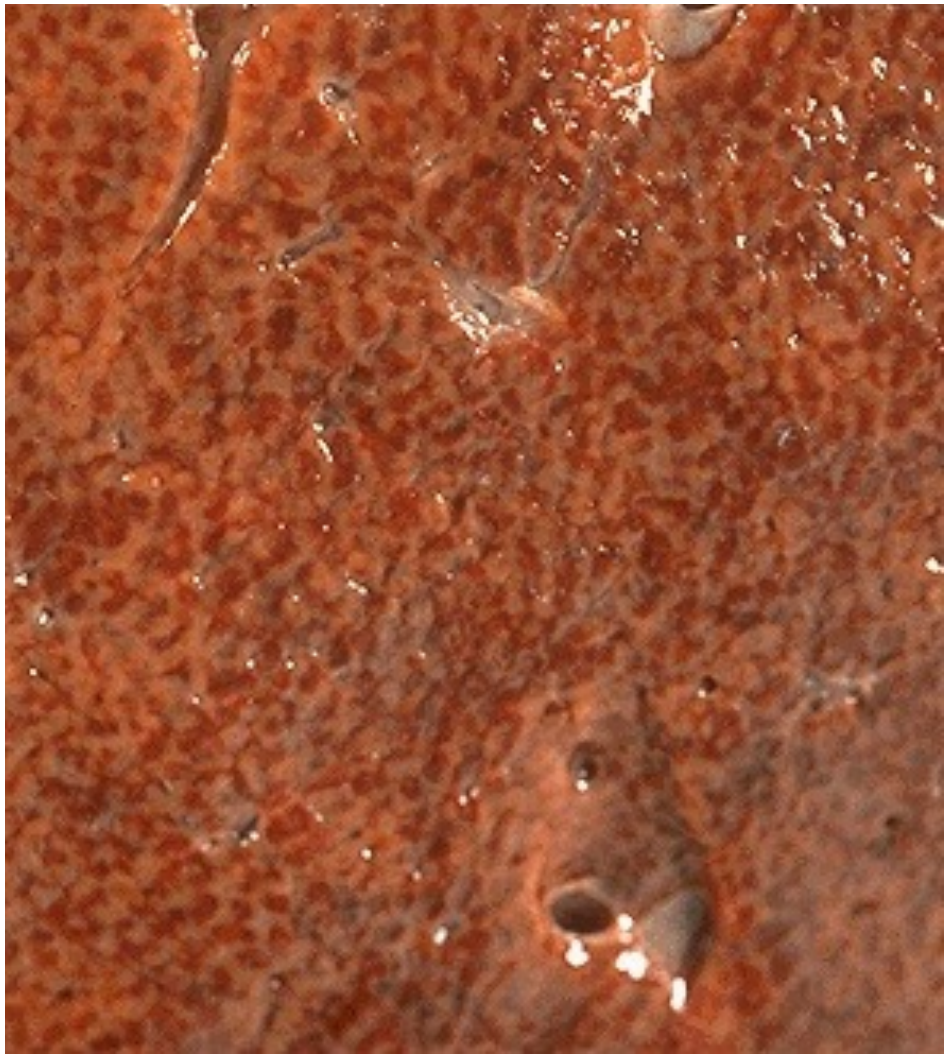
Right Heart Failure

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

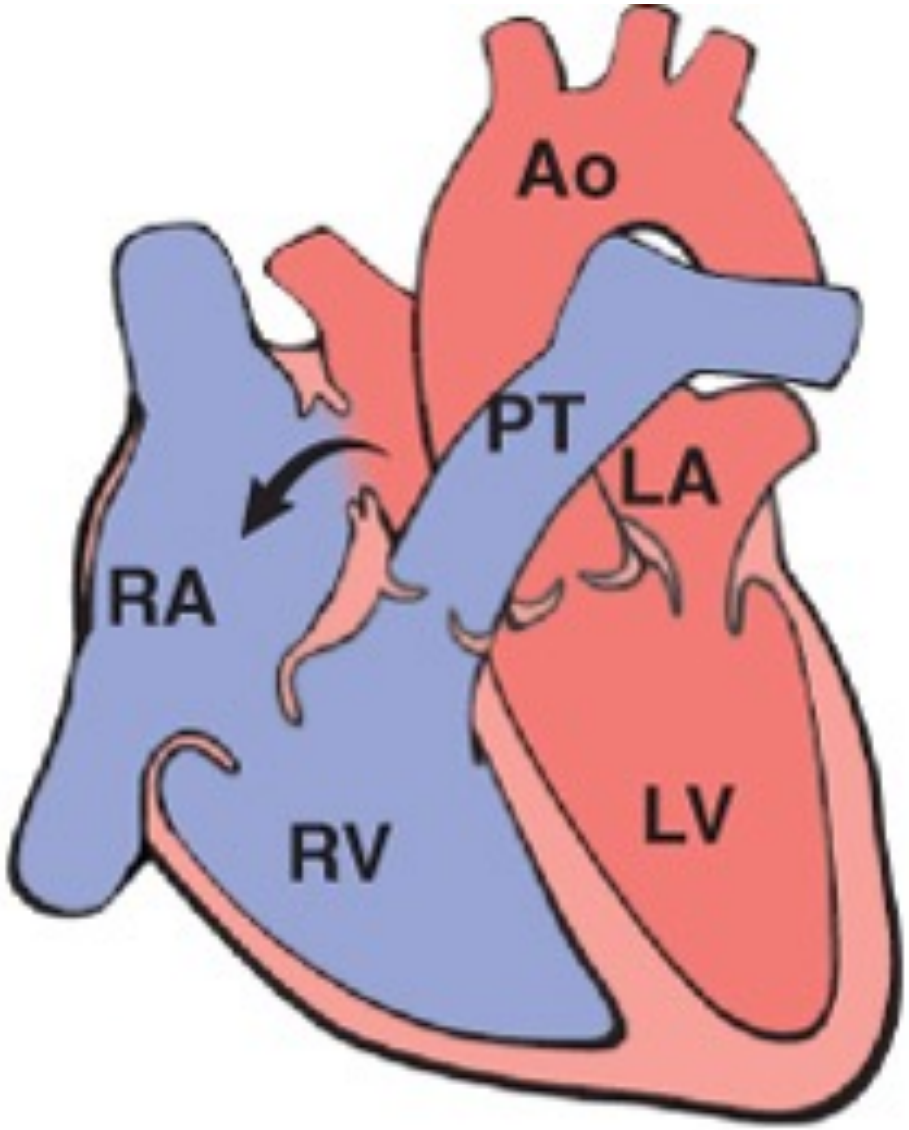
- Blood Vessels
- Heart I
  - Heart Failure
  - Congenital Heart Disease

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

Atrial Septal Defect

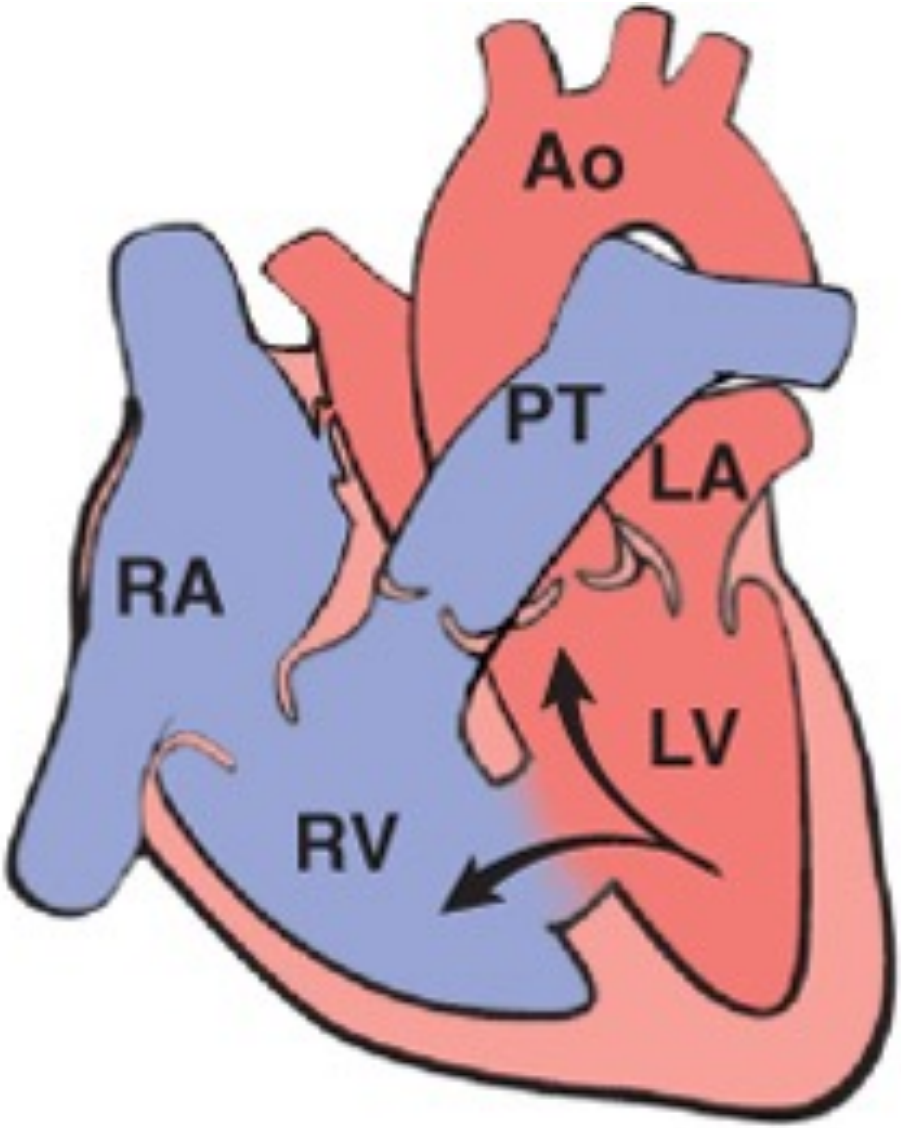
- Initially, shunt is left-to-right (asymptomatic)
- Sometimes, pulmonary vessels become constricted (pulmonary hypertension)
- Eventually, shunt can reverse, becoming right-to-left (this is called Eisenmenger syndrome)
- Surgical repair prevents irreversible pulmonary changes and heart failure



ASD

Ventricular Septal Defects

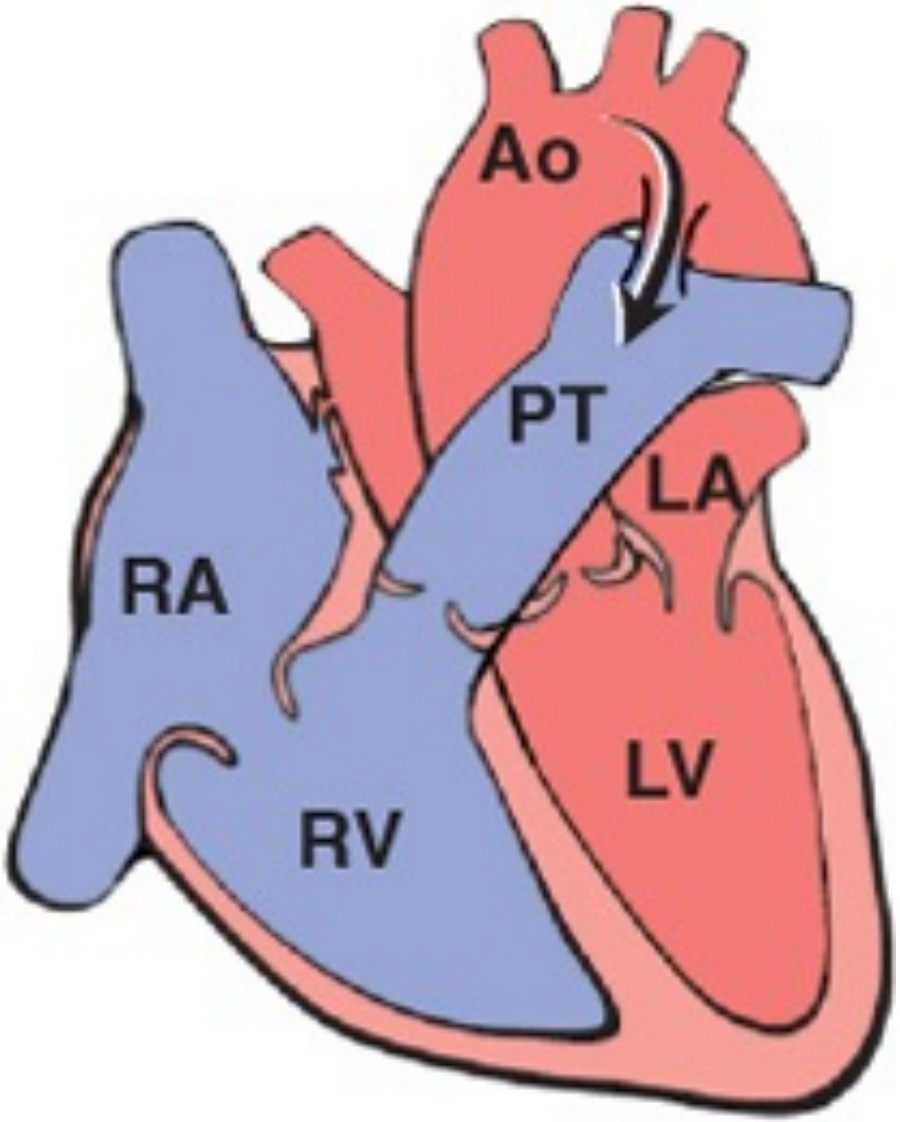
- Most common congenital cardiac anomaly
- Most close spontaneously in childhood
- Small VSD: asymptomatic
- Large VSD: big left-to-right shunt which may eventually become right-to-left



VSD

Patent Ductus Arteriosus

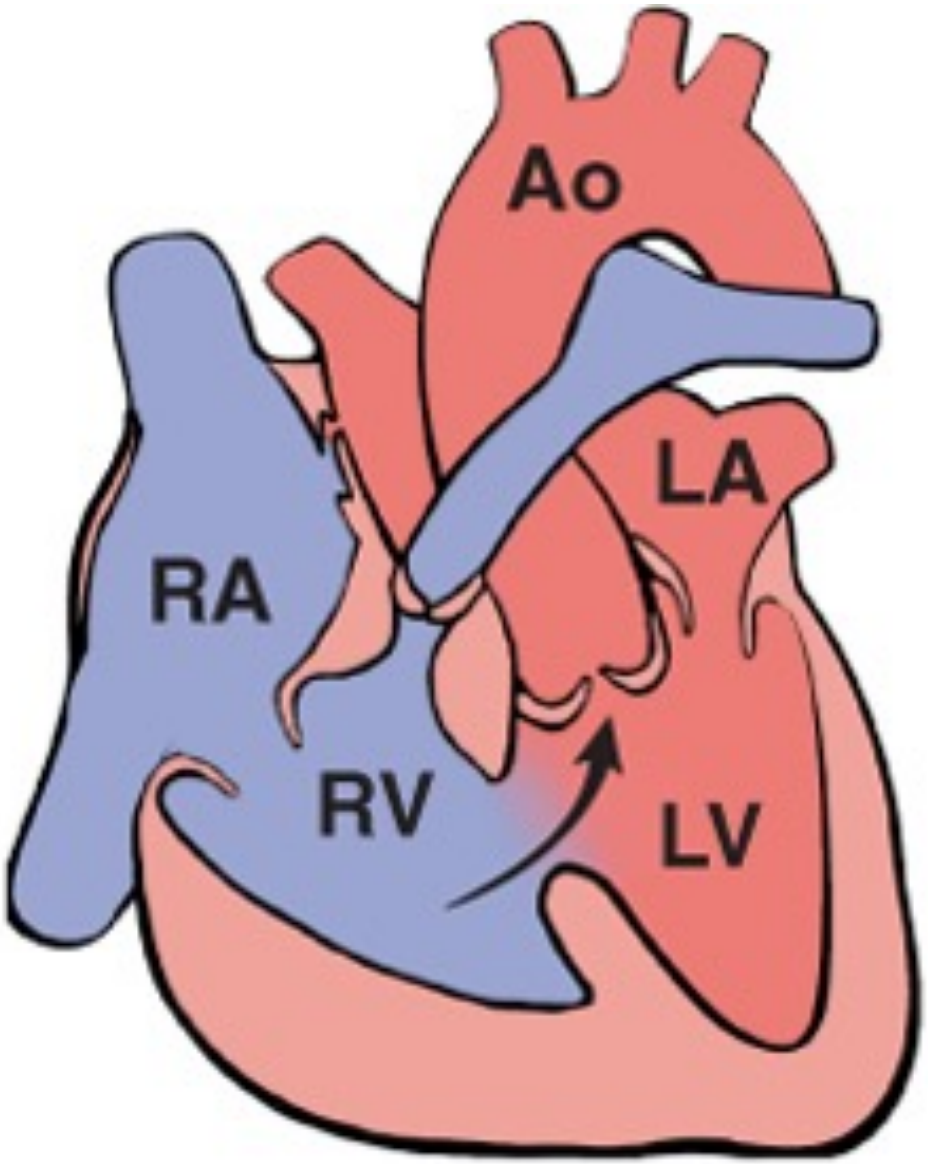
- Ductus: allows flow from PA to aorta in fetus
- Closes spontaneously by day 1-2 of life
- Small PDA: asymptomatic
- Large PDA: big left-to-right shunt which may eventually become right-to-left



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



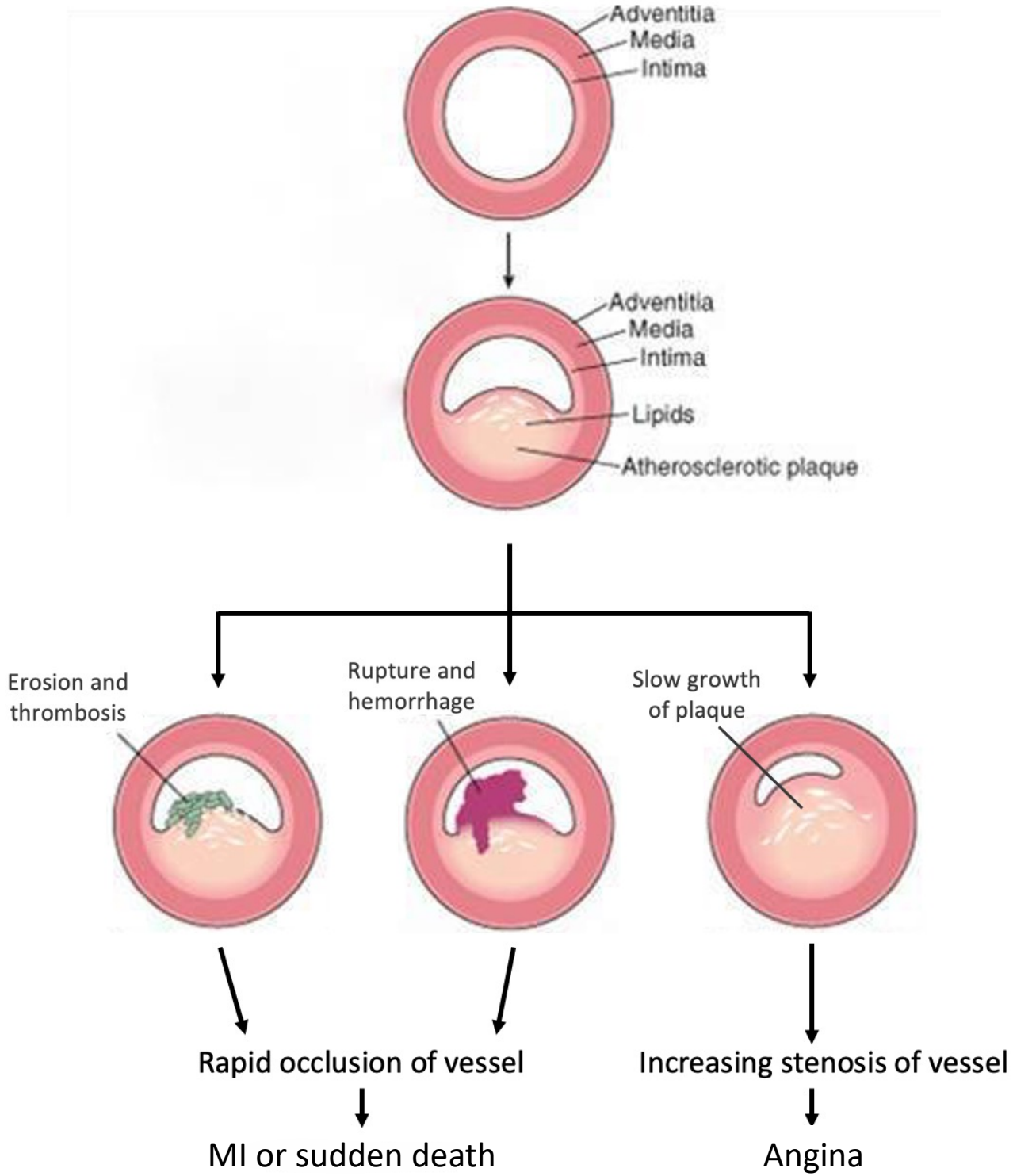
Tetralogy of Fallot

Cardiac Pathology Outline

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

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

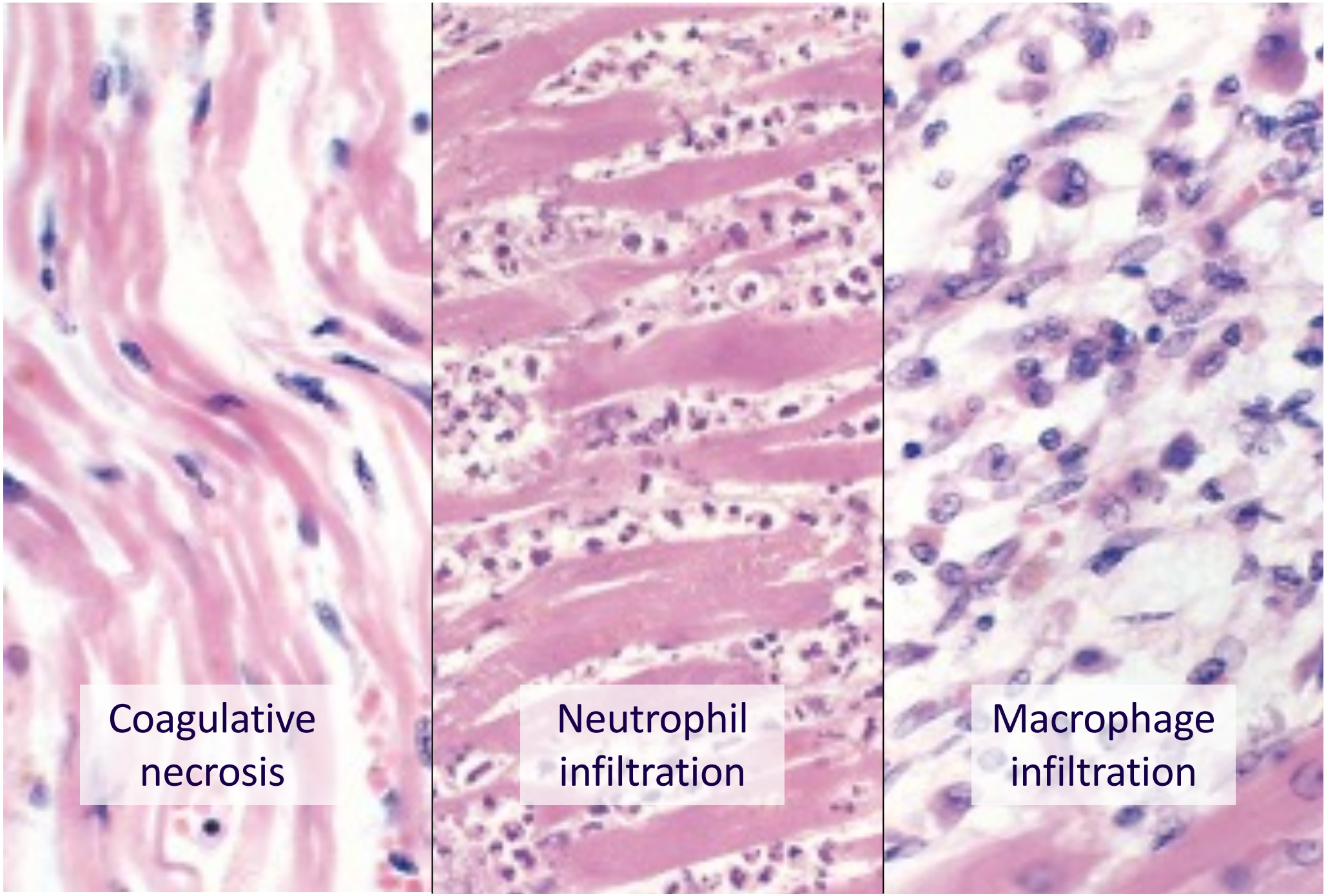

Adventitia
Media
Intima
Adventitia
Media
Intima
Lipids
Atherosclerotic plaque
Erosion and thrombosis
Rupture and hemorrhage
Slow growth of plaque
Rapid occlusion of vessel
Increasing stenosis of vessel
MI or sudden death
Angina

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



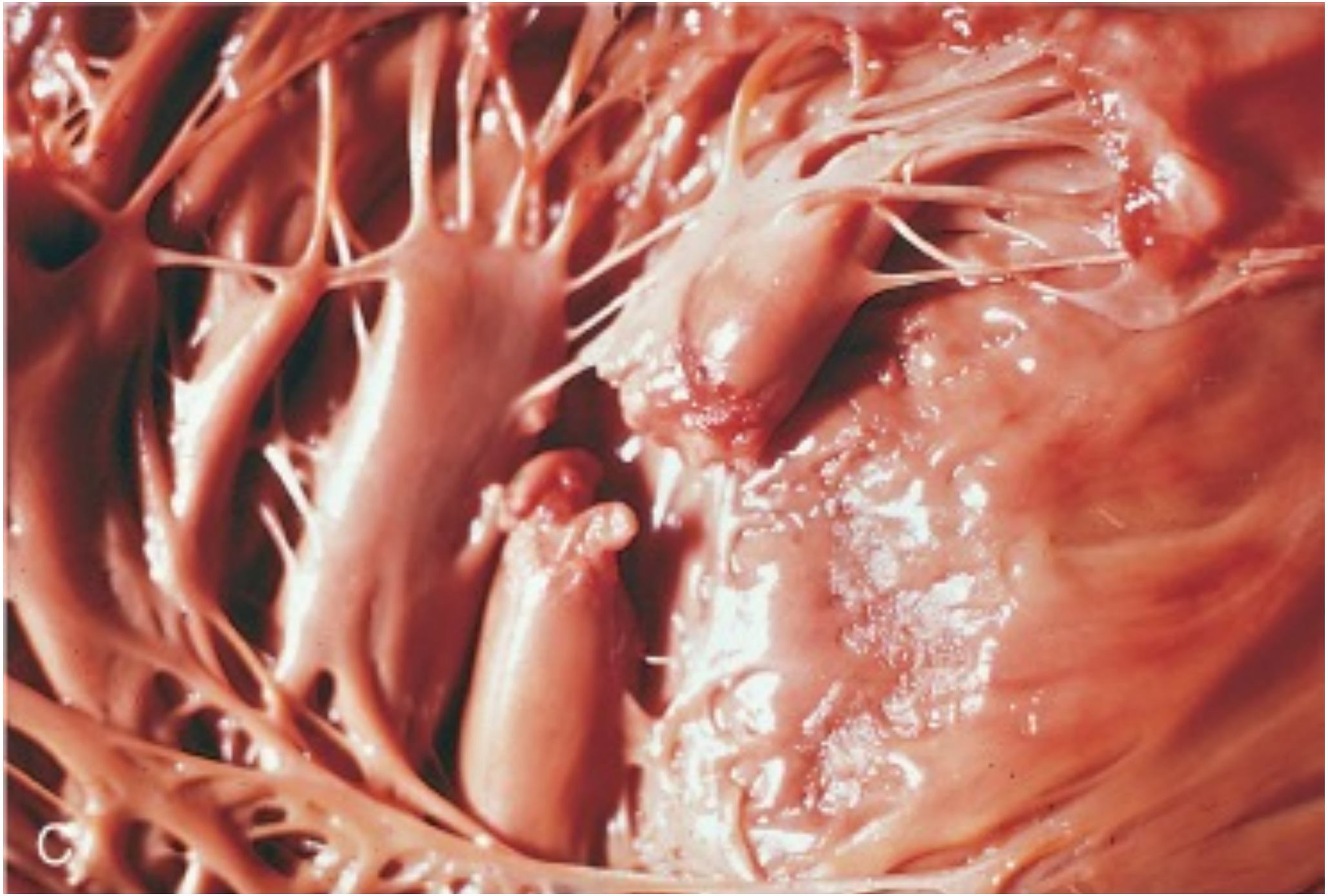
MI: day 1, day 3, day 7

Myocardial Infarction

- Clinical features
  - Severe, crushing chest pain ± 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