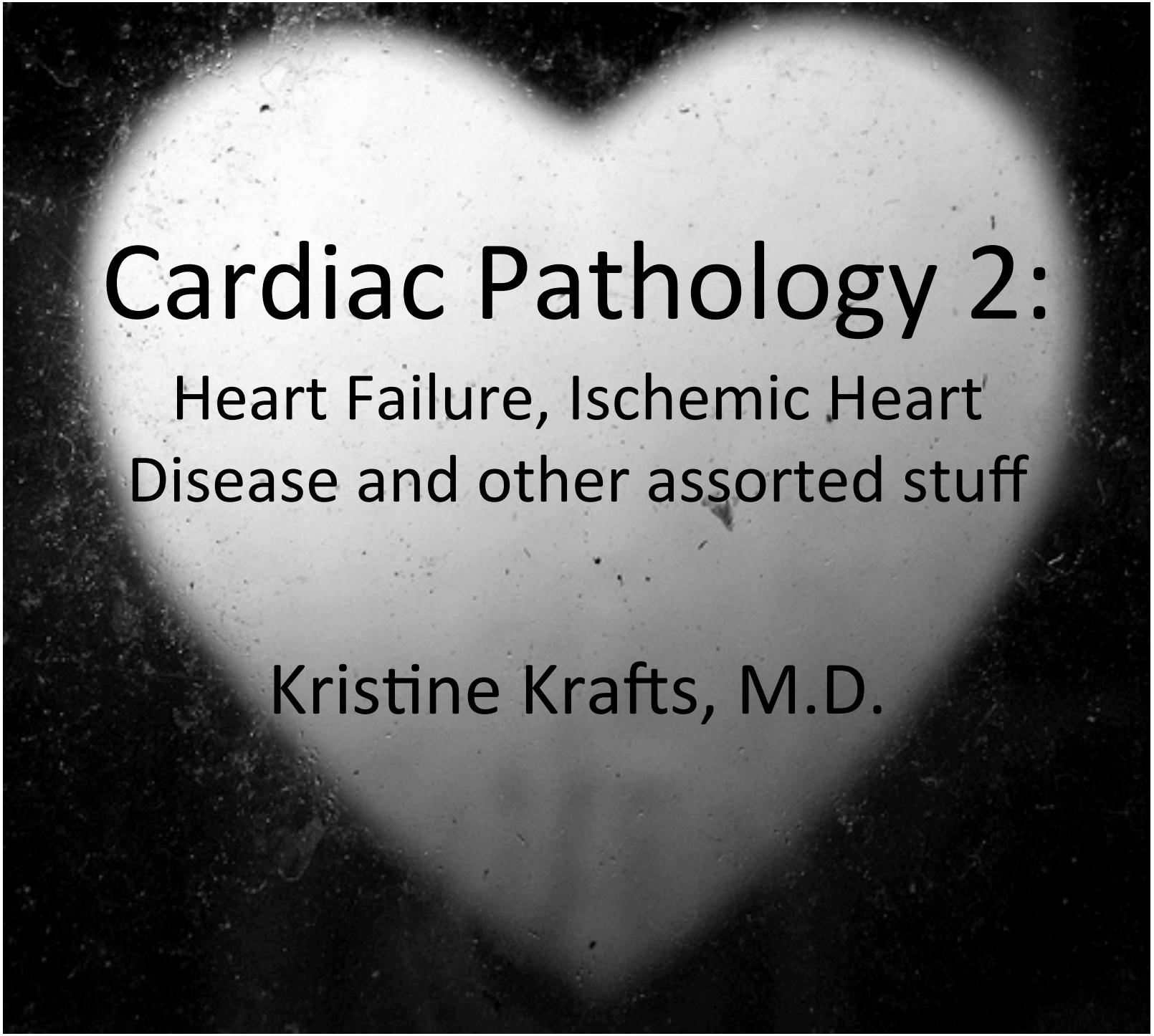


Cardiac Pathology 2:

Heart Failure, Ischemic Heart Disease and other assorted stuff

Kristine Krafts, M.D.

Cardiac Pathology Outline

- Blood Vessels
- Heart I
- Heart II

Cardiac Pathology Outline

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

Cardiac Pathology Outline

- Blood Vessels
- Heart I
  - Heart Failure

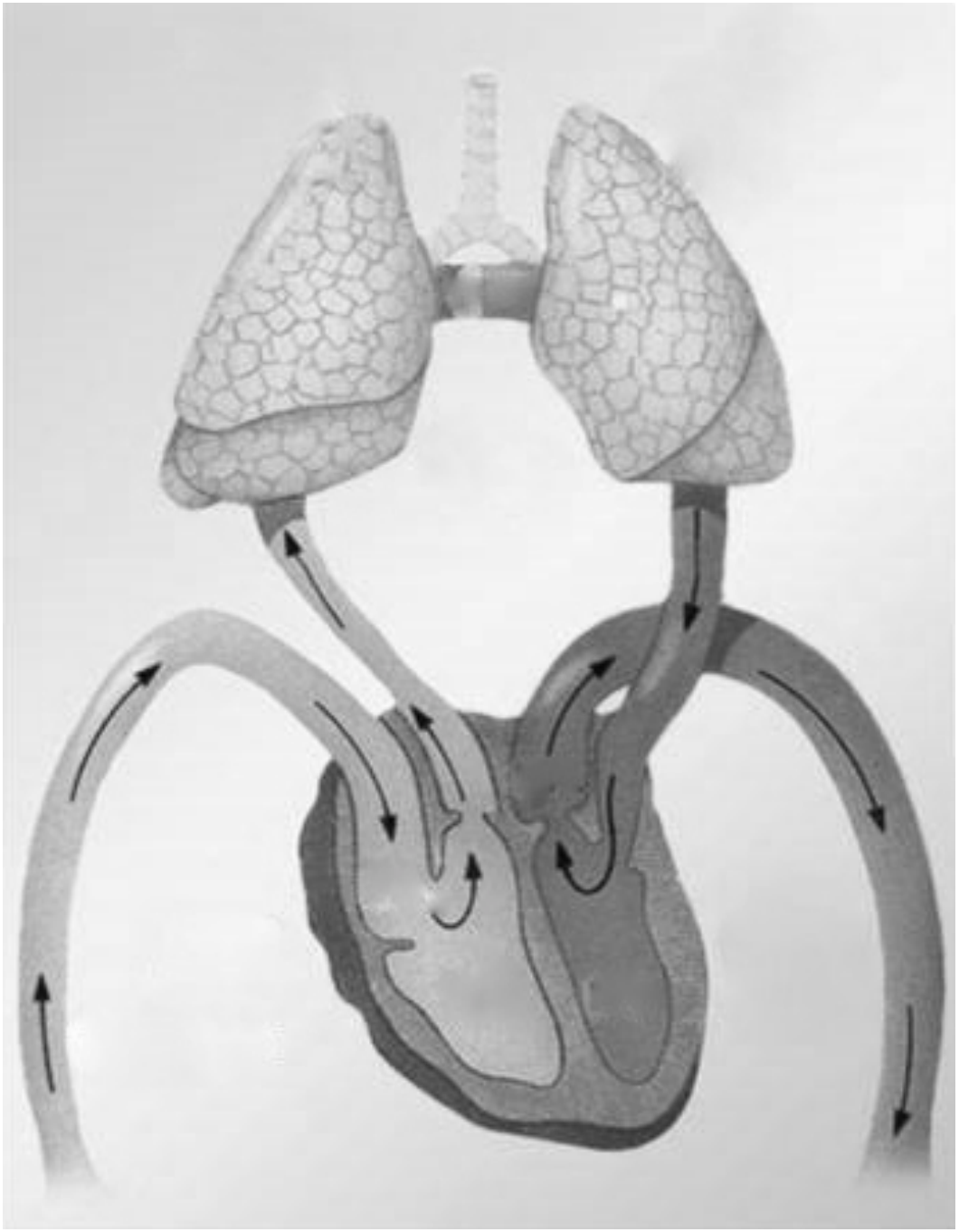
Heart Failure

- 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

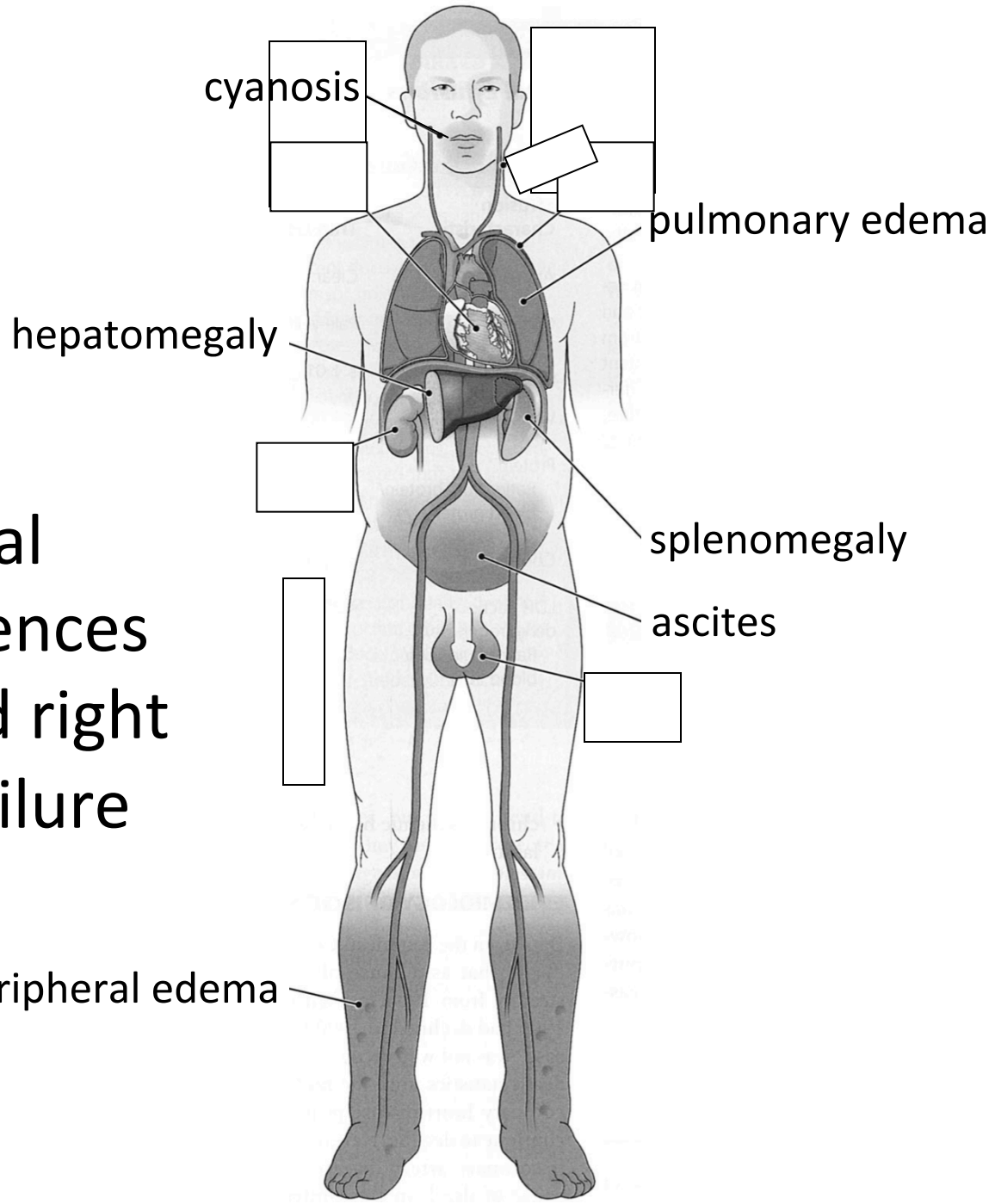
- System responds to failure by
  - Releasing hormones (e.g., norepinephrine)
  - Frank-Starling mechanism
  - Hypertrophy
- Initially, this works
- Eventually, it doesn't
  - Myocytes degenerate
  - Heart needs more oxygen
  - Myocardium becomes vulnerable to ischemia

R



L

Clinical consequences of left and right heart failure



Left Heart Failure

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

Left Heart Failure

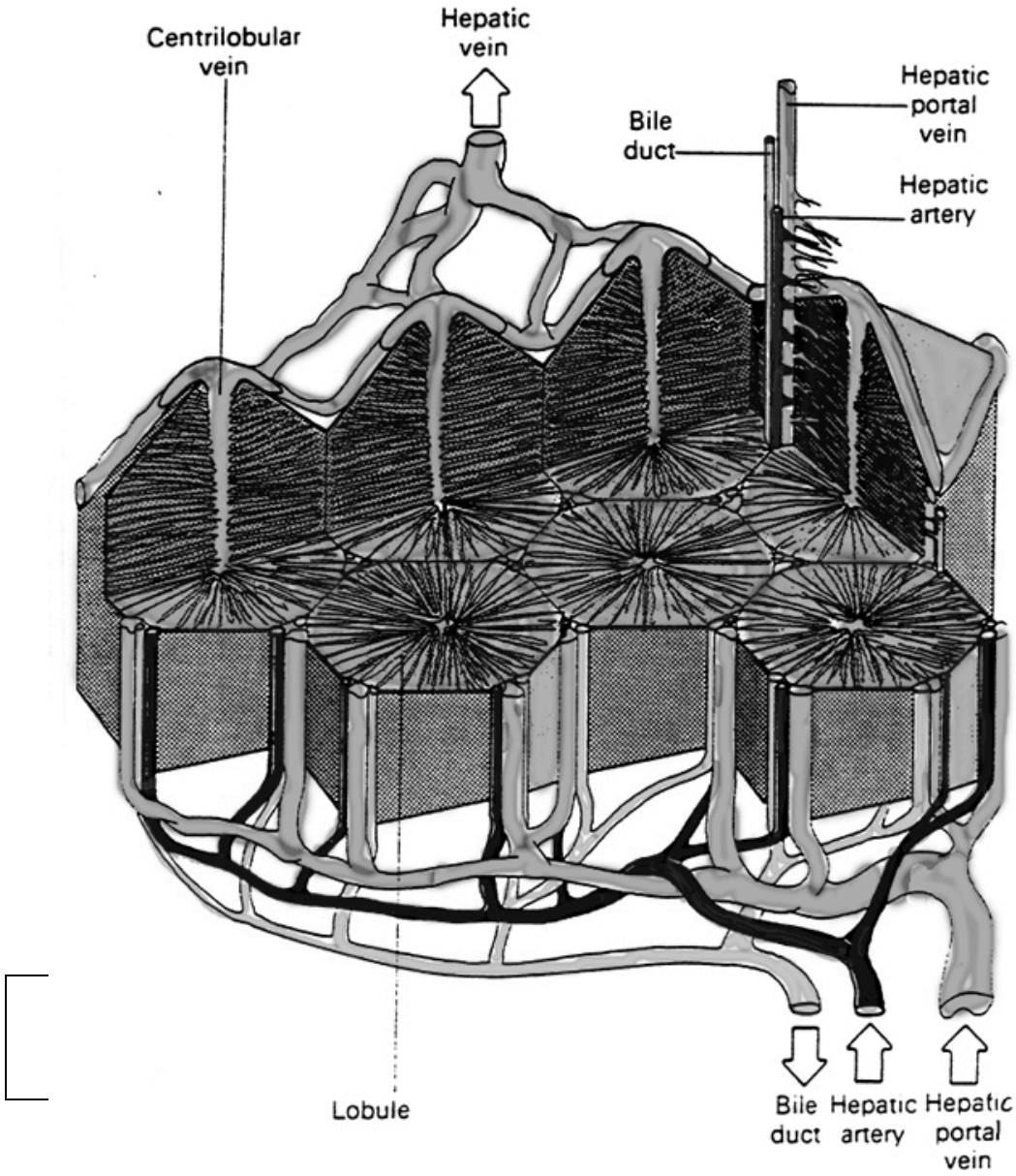
- Dyspnea
- Orthopnea, paroxysmal nocturnal dyspnea too
- Enlarged heart, increased heart rate, fine rales at lung bases
- Later: mitral regurgitation, systolic murmur
- If atrium is big, irregularly irregular heartbeat

Right Heart Failure

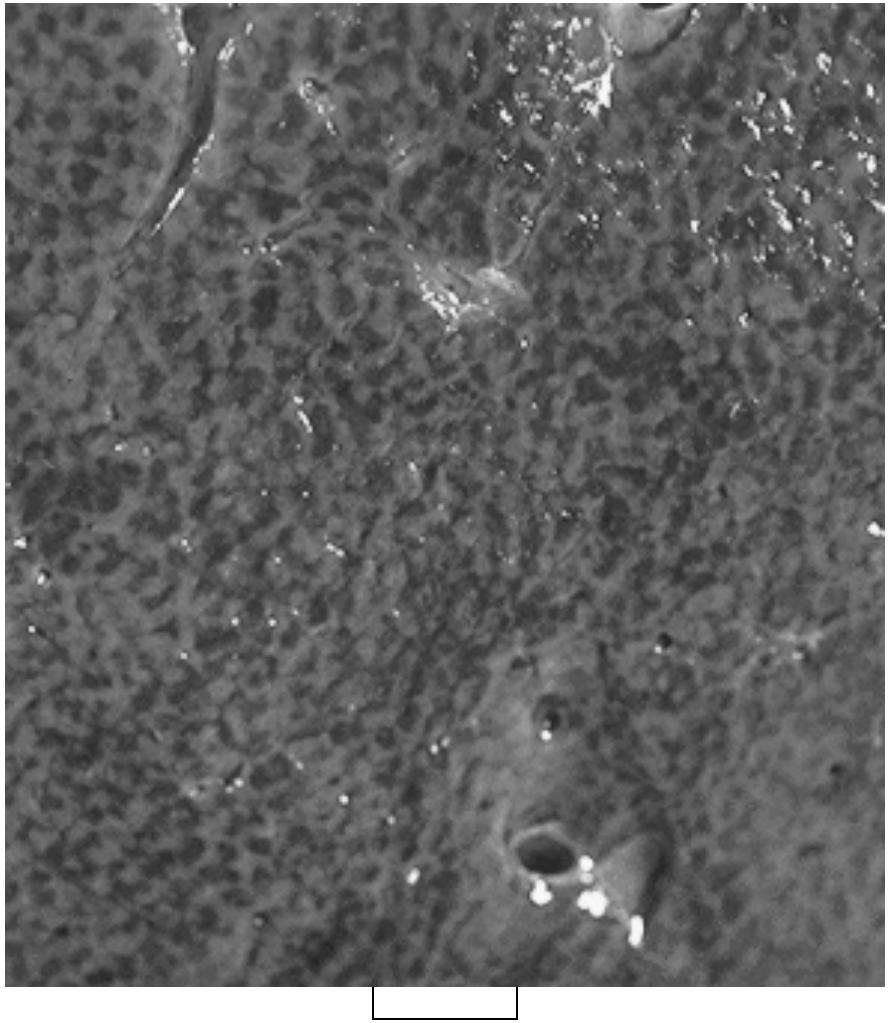
- Right ventricle fails; blood backs up in body
- Commonest causes
  - Left heart failure
  - Lung disease (cor pulmonale")
  - Some congenital heart diseases
- Heart changes
  - right ventricular hypertrophy, dilation
  - right atrial enlargement

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

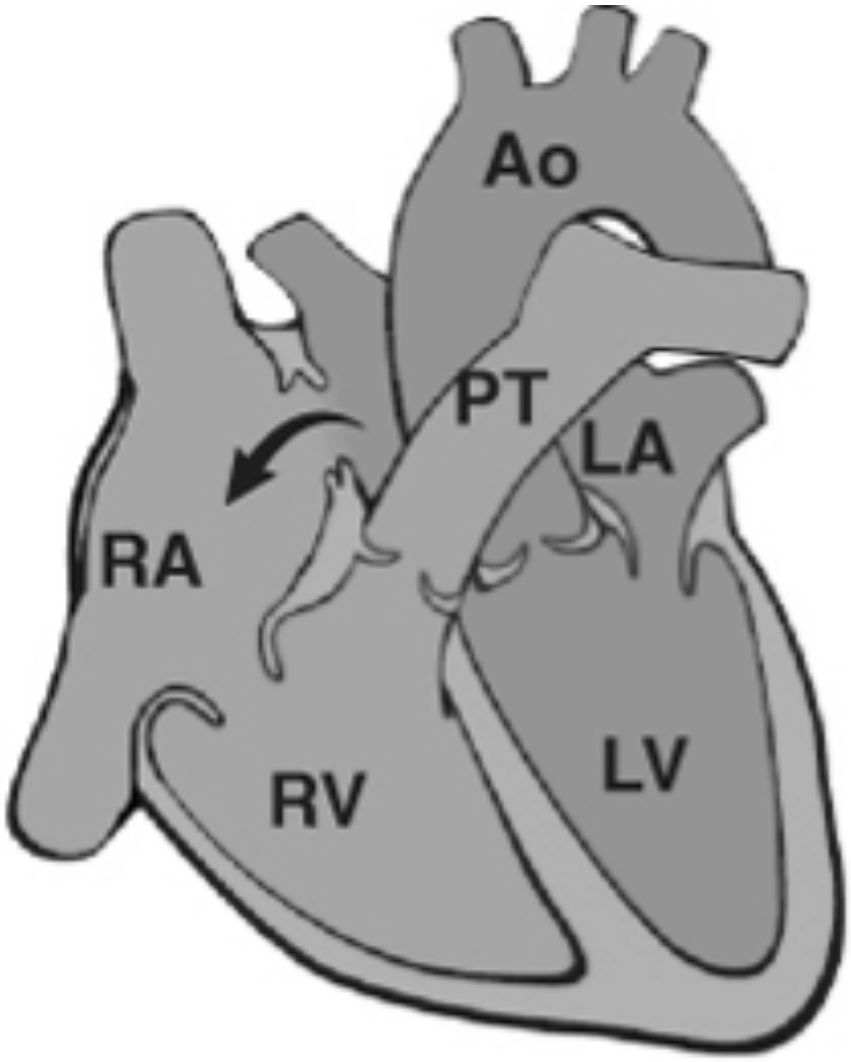
- Abnormalities of heart/great vessels present from birth
- Faulty embryogenesis, weeks 3-8
- Broad spectrum of severity
- Cause unknown in 90% of cases

Congenital Heart Disease

- Left-to-right shunts
  - atrial septal defects
  - ventricular septal defects
  - Patent ductus arteriosus
- Right-to-left shunts
  - tetralogy of fallot
  - transposition of the great arteries
- Obstructions
  - aortic coarctation

Atrial Septal Defects

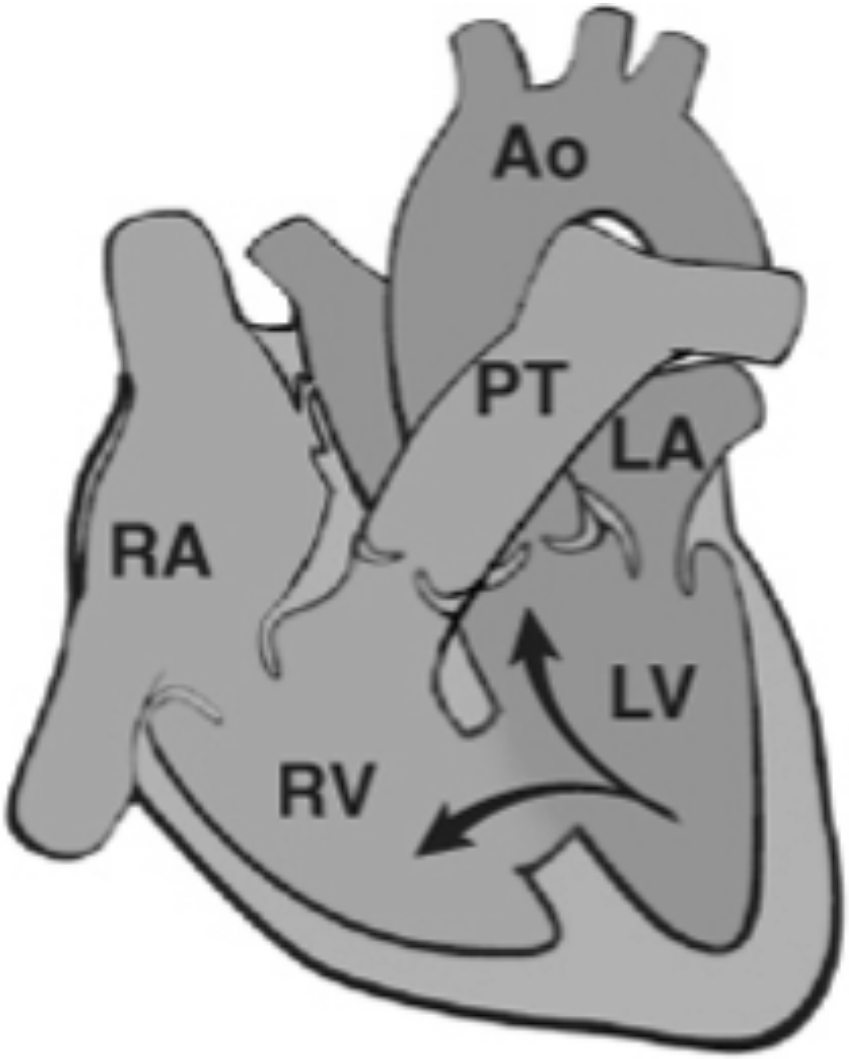
- Initially, left-to-right shunt (asymptomatic)
- Eventually, pulmonary vessels may become constricted (pulmonary hypertension), leading to right-to-left shunt (Eisenmenger syndrome)
- Surgical repair prevents irreversible pulmonary changes and heart failure



ASD

Ventricular Septal Defects

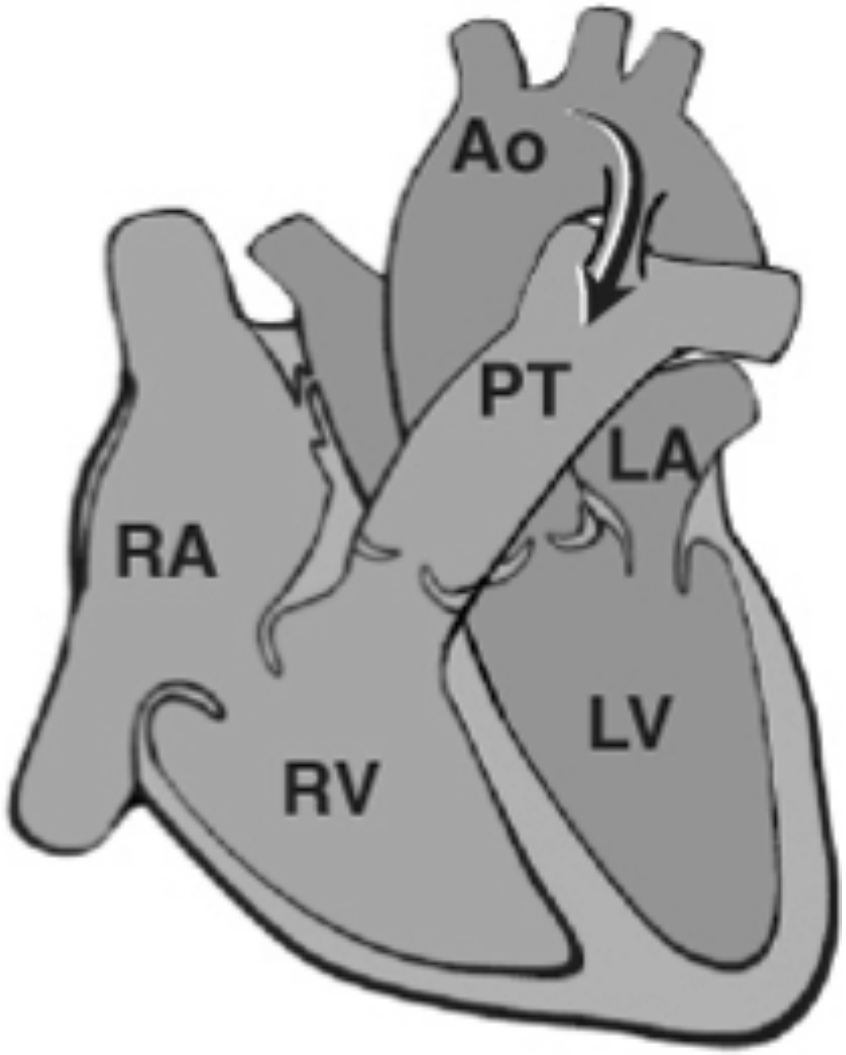
- Most common congenital cardiac anomaly
- Most close spontaneously in childhood
- Small VSD: asymptomatic
- Large VSD: big left-to-right shunt, may become right-to-left



VSD

Patent Ductus Arteriosus

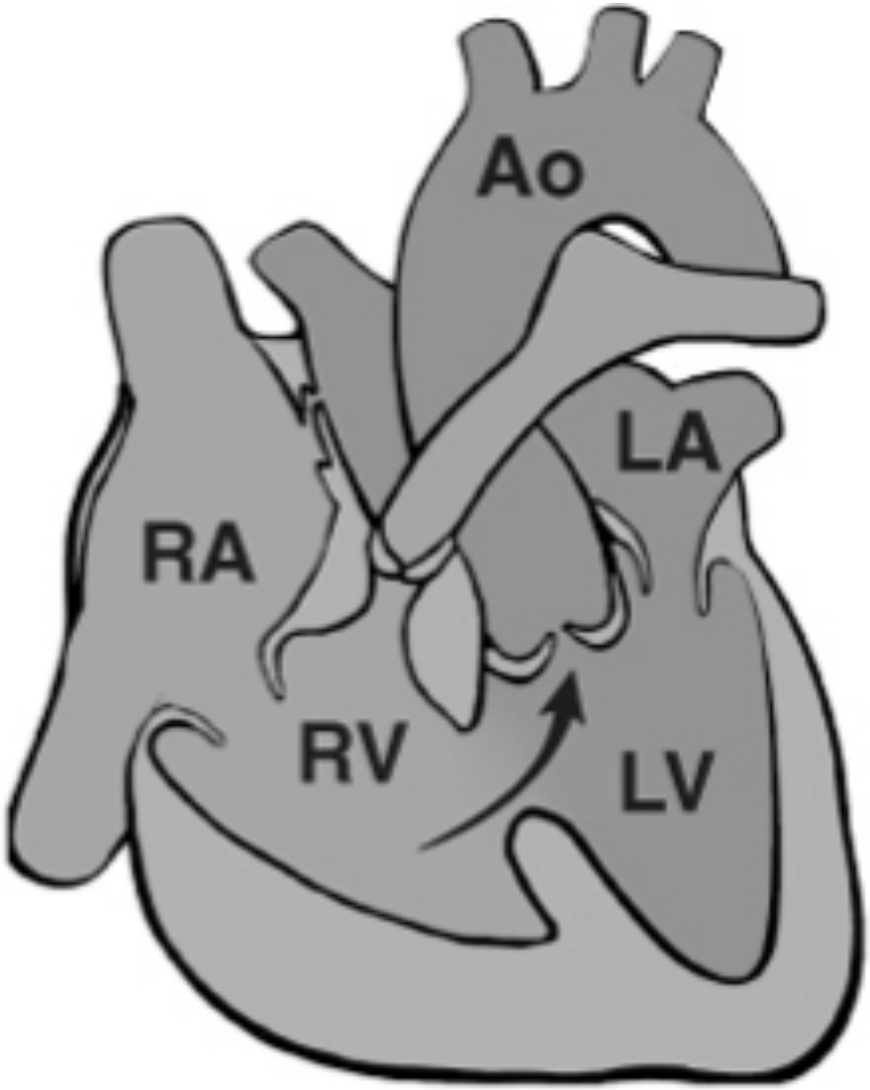
- Ductus: allows flow from PA to aorta
- Closes spontaneously by day 1-2 of life
- Small PDA: asymptomatic
- Large PDA: shunt becomes right-to-left



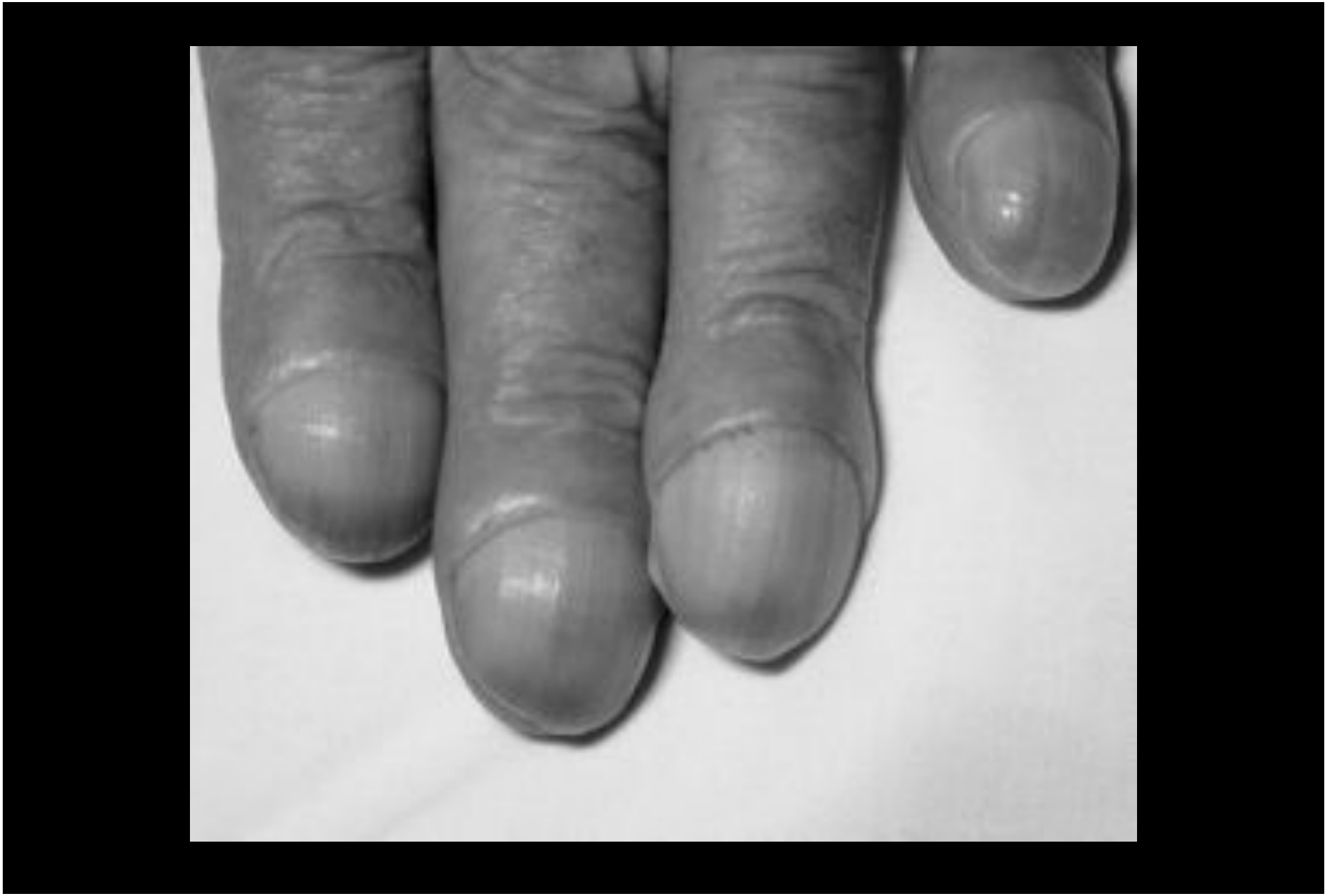
PDA

Tetralogy of Fallot

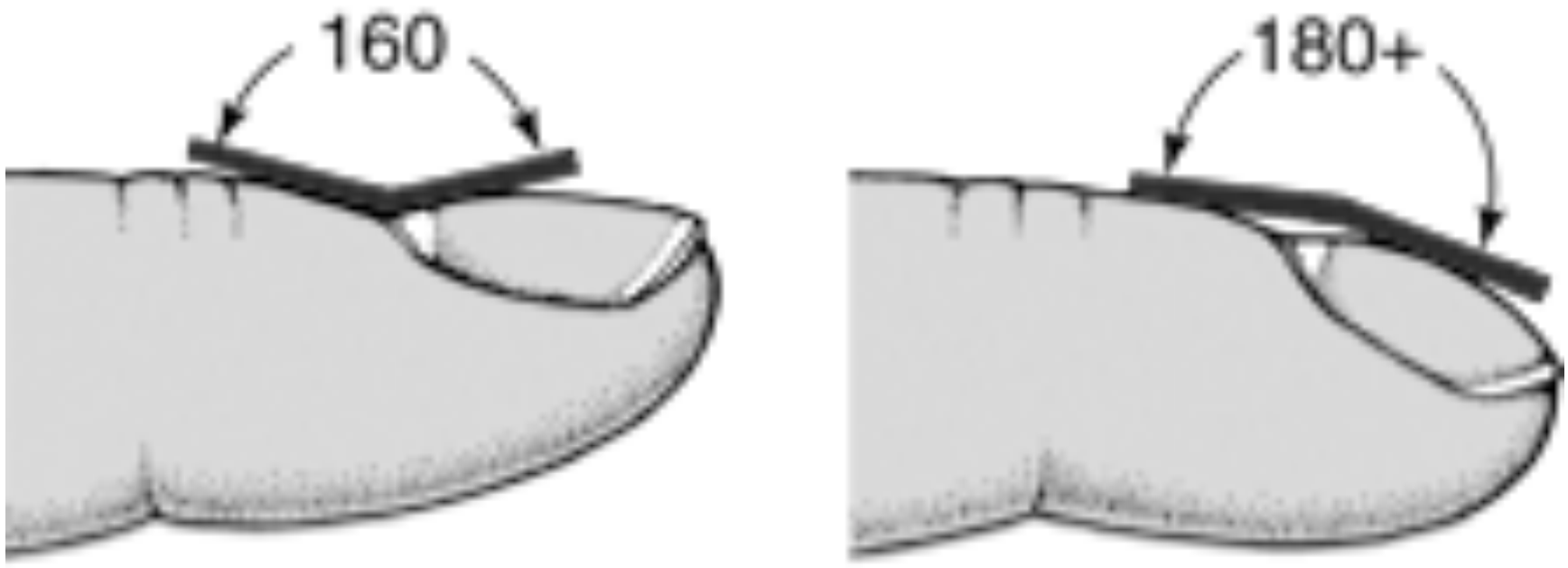
- Most common cause of cyanotic congenital heart disease
- Four features:
  - VSD
  - obstruction to RV outflow tract
  - overriding aorta
  - RV hypertrophy
- Cyanosis, erythrocytosis, clubbing of fingertips, paradoxical emboli



Tetralogy of Fallot



Clubbing of fingertips



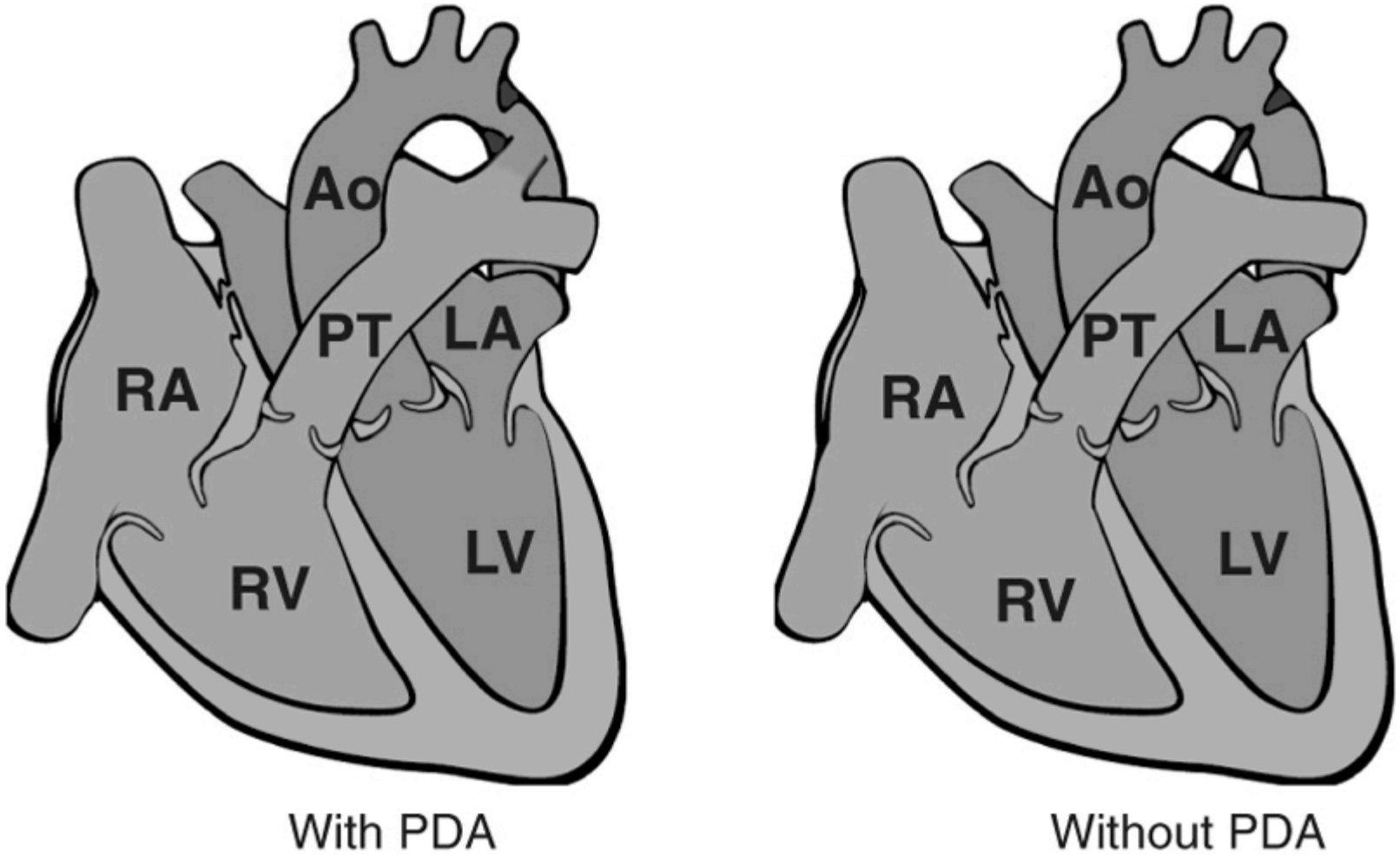
Normal (L) and clubbed (R) fingertips

Transposition of Great Arteries

- Aorta arises from R ventricle; pulmonary artery arises from L ventricle
- Outcome: separation of systemic and pulmonary circulations
- Incompatible with life unless there is a big shunt (VSD)

Aortic Coarctation

- Coarctation = narrowing
- "Infantile" (preductal) and "adult" (postductal) forms
- Cyanosis and/or low blood pressure in lower extremities
- Severity depends on degree of coarctation



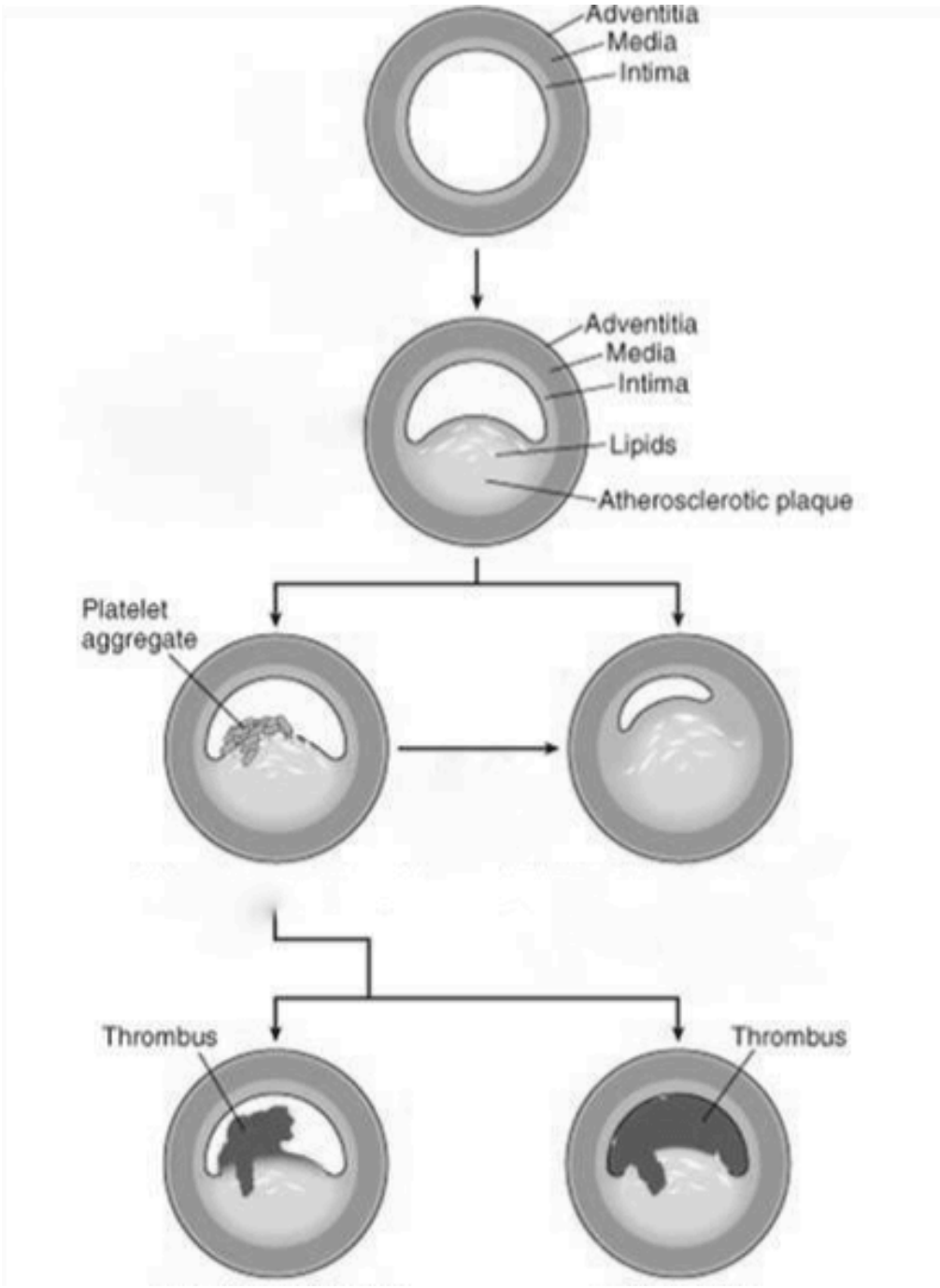
Coarctation of the aorta

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")
- Four syndromes:
  - angina pectoris
  - acute MI
  - chronic IHD
  - sudden cardiac death


Adventitia
Media
Intima
Adventitia
Media
Intima
Lipids
Atherosclerotic plaque
Platelet aggregate
Thrombus
Thrombus

Angina Pectoris

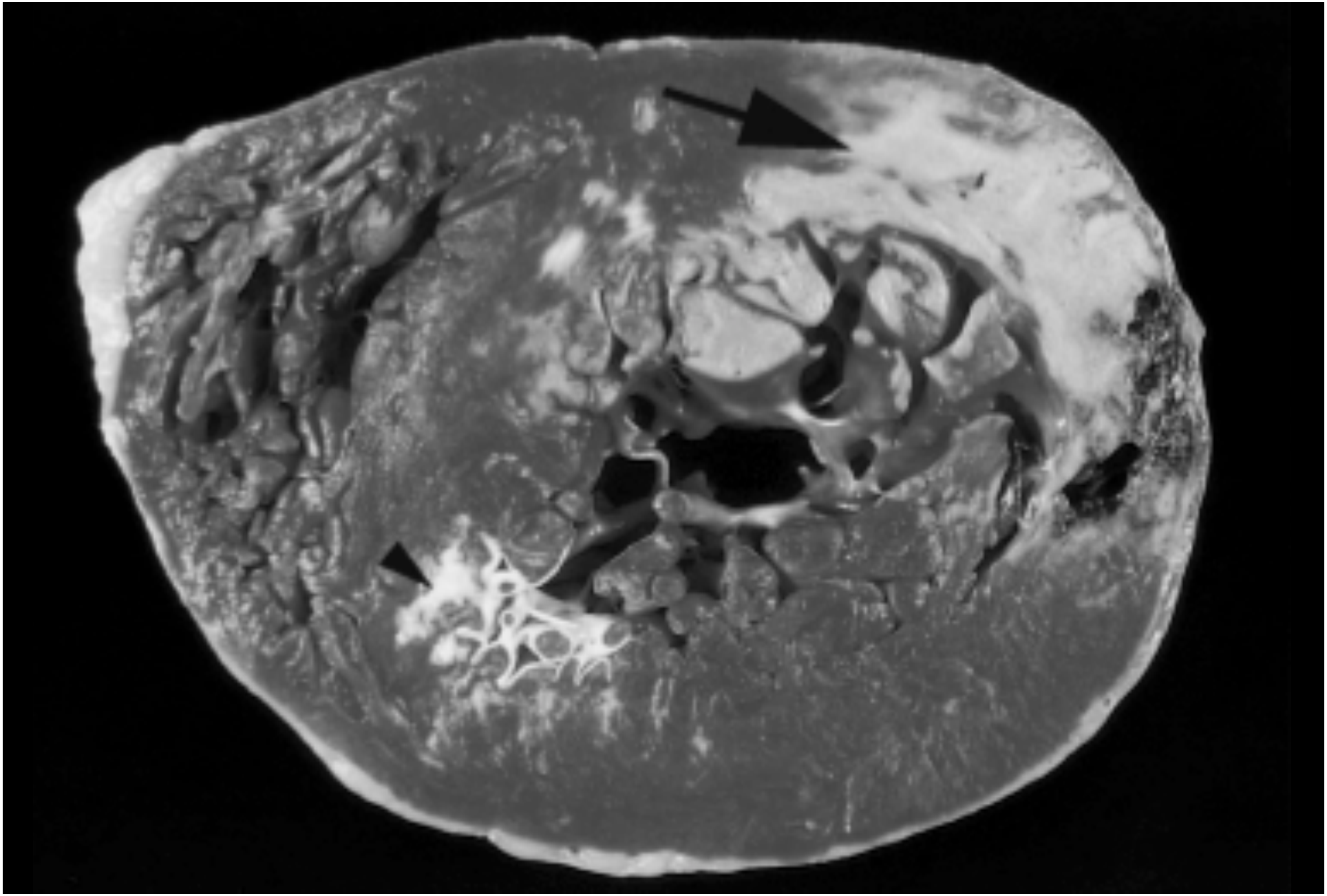
- Intermittent chest pain caused by transient, reversible ischemia
- Typical (stable) angina
  - pain on exertion
  - fixed narrowing of coronary artery
- Prinzmetal (variant) angina
  - pain at rest
  - coronary artery spasm of unknown etiology
- Unstable (pre-infarction) angina
  - increasing pain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

Myocardial Infarction

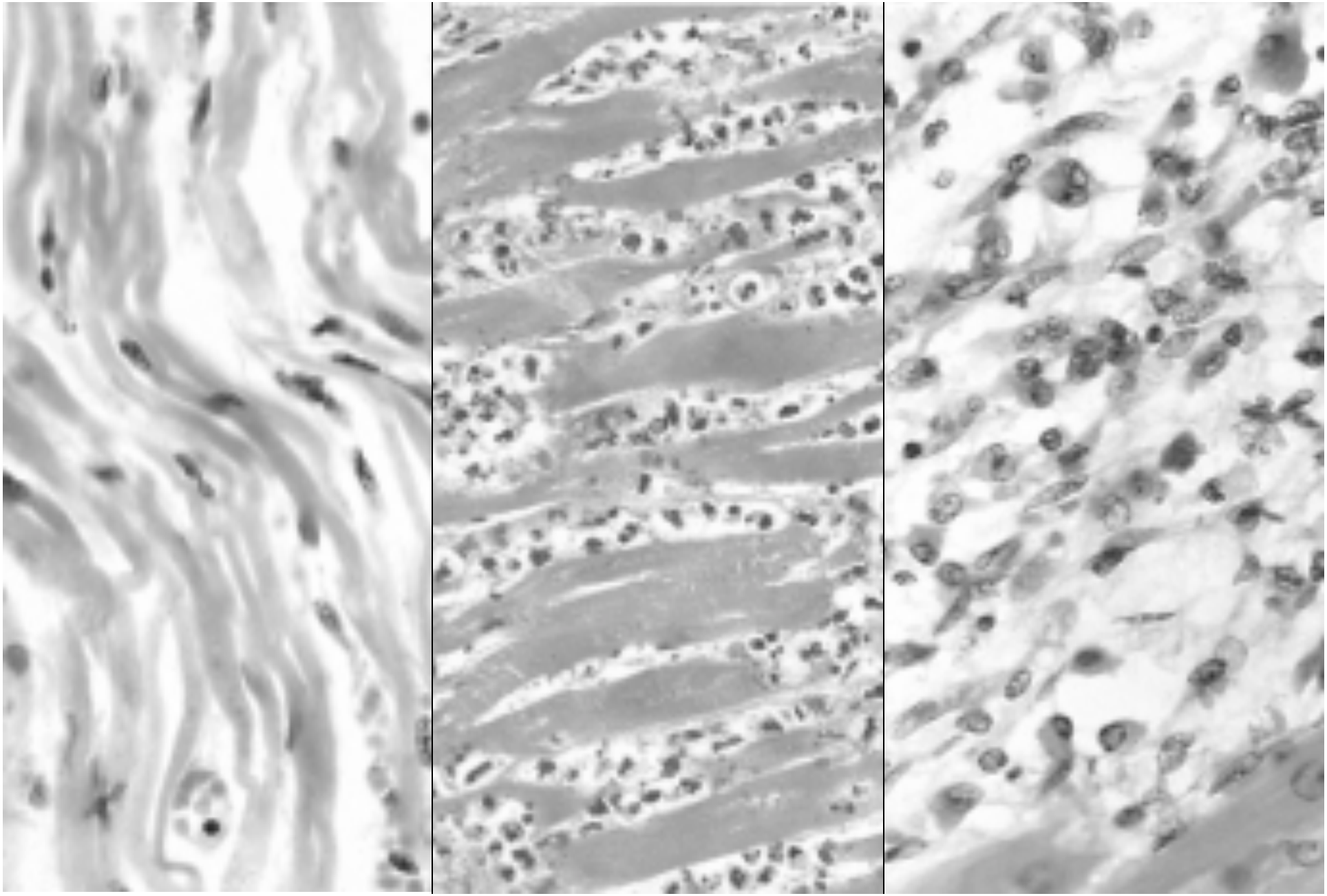
- Necrosis of heart muscle caused by ischemia
- 1.5 million people get MIs each year
- Most due to acute coronary artery thrombosis
  - sudden plaque disruption
  - platelets adhere
  - coagulation cascade activated
  - thrombus occludes lumen within minutes
  - irreversible injury/cell death in 20-40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

Morphologic Changes in Myocardial Infarction

Time	Gross changes	Microscopic changes
0-4h	None	None
4-12h	Mottling	Coagulation necrosis
12-24h	Mottling	More coagulation necrosis; neutrophils come in
1-7 d	Yellow infarct center	Neutrophils die, macrophages come to eat dead cells
1-2 w	Yellow center, red borders	Granulation tissue
2-8 w	Scar	Collagen



Acute Myocardial Infarction



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

Cardiac Pathology Outline

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- Heart I
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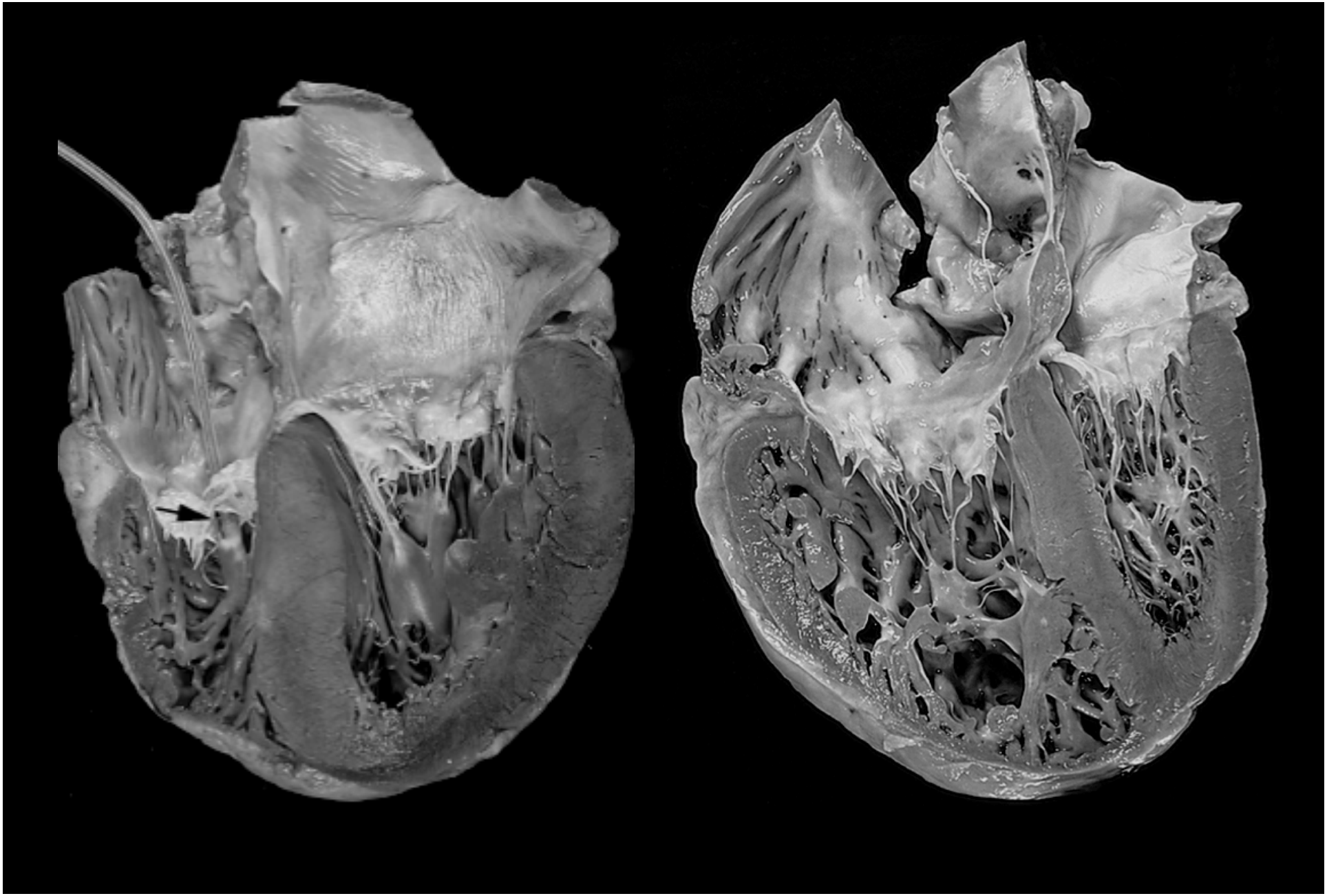
  - Ischemic Heart Disease
  - Hypertensive Heart Disease

Hypertensive Heart Disease

- Can affect either L or R ventricle
- Cor pulmonale is RV enlargement due to pulmonary hypertension caused by primary lung disorders
- Result: myocyte hypertrophy
- Reasons for heart failure in hypertension are poorly understood



Left ventricular hypertrophy (L) and cor pulmonale (R)