Cardiac Pathology 2: Congenital and Ischemic Heart Disease
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Cardiac Pathology Outline
- Blood Vessels
- Heart I
  - Heart Failure
  - Congenital Heart Disease
  - Ischemic Heart Disease

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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
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)
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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

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  - 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

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Left-to-right shunts

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- Ventricular septal defect
- 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
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- Patent ductus arteriosus

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

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 septal defect
- Ventricular septal defect
- 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 septal defect
- Ventricular septal defect
- 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

- 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
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.

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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

Vessel <70% occluded by plaque
No symptoms

How atherosclerosis leads to angina and MI
How atherosclerosis leads to angina and MI

Vessel >70% occluded by plaque
Stable angina

Disrupted plaque
Uh oh

Thrombus partially occluding vessel
Unstable angina

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

Week 2
Granulation tissue forms

Weeks 3-8
Granulation tissue replaced by collagen, forming a scar

Myocardial Infarction

Clinical features
- Severe, crushing chest pain ± 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.

Complications
- contractile dysfunction
- arrhythmias
- rupture
- chronic progressive heart failure

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