THE PERICARDIUM

- The 2 layers surrounding the heart.
- 2 Layers:
  - 1) Visceral Pericardium.
  - 2) Parietal Pericardium.
PERICARDITIS

- Acute inflammation of the pericardium.
- Acute inflammatory response involved.

Results in:

1) Resolution, or

2) Formation of scarred, thickened pericardium.
ACUTE PERICARDITIS

CLASSIFIED ACCORDING TO:

1) CAUSE - infection, trauma, rheumatic fever.

2) NATURE OF THE EXUDATE: serous, fibrinous, purulent, hemorrhagic.
ACUTE PERICARDITIS

- Associated w/ ↑ capillary permeability.

- VIRAL- Most common causes- coxsackie, echovirus, influenza, EBV, mumps, hepatitis, HIV. Often is “post-viral.”

- Other causes: post-cardiotomy syndrome, trauma, rheumatic fever, irradiation; metabolic disorders: uremia, connective tissue diseases (lupus, RA, etc.).
ACUTE VIRAL PERICARDITIS

- Men > Women.
- Viral prodrome.
ACUTE PERICARDITIS

- MANIFESTATIONS: TRIAD OF:
  - 1) Chest pain.
  - 2) Pericardial friction rub.
  - 3) EKG changes.
ACUTE PERICARDITIS

CHEST PAIN

- Abrupt, sharp.
- Precordial, radiating to the neck, back, abdomen.
- Worse w/ inspiration, cough, swallowing.
- Relieved by sitting up, leaning forward- pain likely comes from inflammation of the surrounding pleura, as the parietal pericardium is only pain-sensitive below the 5th & 6th intercostal spaces.
ACUTE PERICARDITIS

EKG CHANGES

- Similar to MI but w/out the T-wave inversion.
CHRONIC PERICARDITIS W/ EFFUSION

- Increase in the inflammatory exudate beyond the acute period.
- Often no cause is found.
- Assoc. w/ other forms of heart disease: RHD, congenital anomalies, hypertension; OR other systemic diseases: lupus, RA, scleroderma, myxedema, uremia.
- MANIFESTATIONS: minimal, often found incidentally on chest X-Ray.
CHRONIC PERICARDITIS W/ EFFUSION

- As it progresses, the exudate can restrict motion and compromise cardiac filling.
CONSTRUCTIVE PERICARDITIS

- Development of fibrous scar between the visceral and parietal pleura.
- Scar → scar retraction → compromises diastolic filling → ascites, DOE, pedal edema, fatigue, JVD.
- KUSSMAUL’S SIGN: Inspiration → ↑ in venous return → but it can’t be accommodated by the right atrium → JVD
CORONARY HEART DISEASE

AKA CORONARY ARTERY DISEASE (CAD), ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE (ASCVD).
CORONARY HEART DISEASE

BLOOD FLOW THROUGH THE HEART

- L & R CORONARY ARTERIES.
- L (LEFT MAIN) → L.A.D., CIRCUMFLEX-SUPPLY THE L VENTRICLE AND SEPTUM.
- R → R VENTRICLE → GOES POSTERIOR TO BECOME THE POSTERIOR DESCENDING.
CORONARY HEART DISEASE

COLLATERAL CIRCULATION

- Early on in atherosclerosis, collaterals develop along with advancing disease.
- See Pg. 541.
- Eventually, the collaterals can’t keep pace, symptoms develop after approx. 70-75% occlusion.
CORONARY ATHERSCLEROSIS

- Sx’s develop at 75% occlusion.
- Most common location: 1st several cm’s of the LAD and Left Circumflex.

2 TYPES OF LESIONS:
- 1) FIXED / STABLE LESION: seen in stable angina.
- 2) UNSTABLE LESION: seen in unstable angina, MI.
CORONARY HEART DISEASE

DETERMINANTS OF PLAQUE RUPTURE:

1) Size of the lipid-rich core and stability and thickness of the fibrous cap.

2) The presence of inflammation w/ plaque degeneration.

3) Lack of smooth muscle cells w/ impaired healing and plaque stabilization.
PLAQUE RUPTURE

- Plaques w/ a thin fibrous cap over a large lipid core at increased risk of rupture.

WHEN DOES RUPTURE HAPPEN:
- 1) Spontaneously.
- 2) W/ change in hemodynamics.
- 3) Diurnal variation: increased the 1st hour after arising.
THROMBOSIS & OCCLUSION

- See text.
- In essence, when plaque rupture happens, the coagulation cascade is activated by the usual suspects: thromboxane A2, thrombin, platelets, etc.
- A thrombus develops acutely obstructing the artery.
ISCHEMIC HEART DISEASE

- ANGINA = Ischemia w/out infarction.
- 3 FLAVORS:
  1) STABLE ANGINA.
  2) UNSTABLE ANGINA.
  3) M.I.
- Unstable Angina and MI also classified under “Acute Coronary Syndromes.”
STABLE ANGINA

- Fixed coronary obstruction of 75% or more, leading to a disparity between demand for oxygen and nutrients and their delivery.

- Precipitated by ↑ metabolic demands of the heart: exertion, cold, emotional stress.
STABLE ANGINA

MANIFESTATIONS

- Pain: sub-sternal, precordial.
- Steady, squeezing, crushing, suffocating.
- May radiate to L shoulder, arm, jaw.
- Confused w/ arthritic / musculoskeletal pain, heartburn / indigestion.
- Relieved by NTG or rest w/in 5-10 minutes → if not, is not ischemic, or is severe ischemia.
PRINZMETAL’S ANGINA

- “Variant,” “Vasospastic.”
- Due to vasospasm.
- Can occur at rest.
- Can occur in absence of atherosclerosis.
- Nocturnal, as opposed to diurnal.
ACUTE CORONARY SYNDROMES

- INCLUDES:
  - 1) UNSTABLE ANGINA.
  - 2) NON-ST SEGMENT ELEVATION MI.
  - 3) ST-SEGMENT ELEVATION MI.

- ST SEGMENT- SEE TEXT.
ACUTE CORONARY SYNDROMES

- SERUM MARKERS:
  - “Cardiac enzymes.”
  - Enzymes released upon cell death.
  - Includes, Myoglobin, CPK / CK MB (creatine phosphokinase / creatine kinase), Troponin I and T.
  - “Isoenzymes.” The MB band of CPK.
ACUTE CORONARY SYNDROMES

- SERUM MARKERS:
  - CRP- C-REACTIVE PROTEIN: An inflammatory marker; assoc. w/ increased risk for recurrent events across the spectrum of acute coronary syndromes.
UNSTABLE ANGINA / NON-ST SEGMENT ELEVATION MI.

- Syndrome of ischemia ranging between stable angina and MI.
- May occur as:
  1) Primary disorder- progression of stable angina.
  2) Secondary disorder- non-coronary, cocaine, anemia.
  3) Within weeks after an MI.
UNSTABLE ANGINA / NON-ST SEGMENT ELEVATION MI.

MANIFESTATIONS

- Pain is more: 1) intense, and 2) persistent.
- Whether it is unstable angina or MI based on serum markers / enzymes.
ST-SEGMENT ELEVATION MI

- The classic “heart attack.”
- Assoc. w/ ischemic death (infarction) of myocardium.

- 40-50% LAD.
- 30-40% R CORONARY.
- 15-20% L CIRCUMFLEX.
ST-SEGMENT ELEVATION MI

- Abrupt onset of pain.
- But more prolonged, not relieved by rest or NTG.
- ACCOMPANIED BY: S.O.B., weakness, fatigue; GI Sx’s: N / V; sympathetic stimulation → anxiety, restlessness, impending doom. Skin: pale, cool, moist (diaphoresis).
- NO ST-SEGMENT ELEVATION
ST-SEGMENT ELEVATION MI

SUDDEN DEATH

- WITHIN 1 HOUR OF Sx’S.
- DUE TO ARRHYTHMIA.
- 30-50% DIE WITHIN 1ST HOUR.
CARDIOMYOPATHY

- Disorders of heart muscle.
- Primary- heart muscle disorders of unknown origin.
- Secondary- due to CVD, MI, etc.
PRIMARY CARDIOMYOPATHY

- Sx’s develop when disease is advanced.
- Suspect when young, previously healthy patient develops cardiomegaly and heart failure.

**CLASSIFIED AS:**
- 1) DILATED.
- 2) HYPERTROPHIC.
- 3) RESTRICTED.
- 4) PERIPARTUM.
HYPERTROPHIC CARDIOMYOPATHY

- Most common cause of sudden cardiac death in the young, athletes.
- IHSS, Asymmetric septal hypertrophy.
- Familial, autosomal dominant.
- Myofibril disarray → uncoordinated contractions & impaired relaxation → fatal arrhythmias.
- Sudden death often the 1st symptom, atrial fib.
- DX: echocardiography.
PERIPARTUM CARDIOMYOPATHY

- LV dysfunction, occurs 1 mo. prior to delivery to 5 mos. post-partum.
- Rare.
- Seen in: advance maternal age, multi-fetal pregnancies, pre-eclampsia, gestational hypertension.
- Mortality: 18-56%.
- Most likely cause: myocarditis.
VALVULAR HEART DISEASE

1) MITRAL STENOSIS.
2) MITRAL REGURG.
3) AORTIC STENOSIS.
4) AORTIC REGURG.

R-SIDED VALVULAR DISEASE IS USUALLY CONGENITAL.
MITRAL STENOSIS

- Replacement of valvular tissue w/ fibrous scar.
- Most commonly from rheumatic fever.
- Incomplete opening during diastole → impaired filling of LV → distention of the LA → pulmonary congestion → R-sided failure.
MITRAL STENOSIS

MANIFESTATIONS

- ↓ LV filling, ↓ C.O.
- ↑ blood in the LA → Pulmonary congestion.
- ↑ blood in the LA → atrial arrhythmias (atrial fib in 30%) → mural thrombi → embolic stroke, peripheral arterial emboli.
MITRAL STENOSIS

MANIFESTATIONS

- Sx’s: those of pulmonary congestion: SOB, PND, orthopnea.
- Palpitations, chest pain, weakness, fatigue, PAC’s, atrial fib.
- THE MURMUR: in diastole during v. filling; low-pitched rumble; apex of the heart.
MITRAL REGURGITATION

- Incomplete closure of the mitral valve.
- L ventricular stroke volume divided between the aorta and back into the L atrium.
- FROM: RHD, ruptured chordae tendonae or papillary muscle from an MI; mitral valve prolapse (next); acute: from MI; chronic: RHD.
MITRAL REGURGITATION

MANIFESTATIONS

- Well-tolerated for years until the regurg makes LV enlarge → ↑ LA →
pulmonary congestion, atrial fib.
- THE MURMUR: pansystolic.
MITRAL VALVE PROLAPSE

- 2-5% of the population.
- “Floppy mitral valve syndrome.”
- CAUSE: from unknown to assoc. w/ other diseases: Marfan’s, osteogenesis imperfecta, metabolic disorders.
- PATHOLOGY: myxedematous degeneration of the valve leaflets $\rightarrow$ enlarged, floppy $\rightarrow$ prolapse back into the L atrium during systole.
MITRAL VALVE PROLAPSE MANIFESTATIONS

- Most are asymptomatic.
- Sx’s: vague chest pain, dyspnea, fatigue, anxiety, palpitations, lightheadedness.
- AUSCULTATION: mid-systolic click, late-systolic murmur.
- Variety of EKG changes.
- Arrhythmias.
AORTIC STENOSIS

- **CAUSES:** Rheumatic fever, congenital malformation.

- Resistance to ejection of blood from the LV $\rightarrow$ $\uparrow$ work demand on the LV.

- **MANIFESTATIONS:** asymptomatic for years $\rightarrow$ angina, syncope, heart failure, fainting, exertional hypotension.

- $\downarrow$ stroke volume $\rightarrow$ $\downarrow$ systolic pressure and pulse pressure.
AORTIC STENOSIS

CLINICAL FINDINGS

- Slow heart rate, ↓ pulse amplitude.
- Soft, absent, or paradoxically split S2.
- Harsh systolic ejection murmur L sternal border.
AORTIC REGURGITATION

- **CAUSES:** Rheumatic fever, congenital, aortic dissection, endocarditis, prosthetic valve.

- **ACUTE:** Large regurg. volume acutely in a ventricle that has not adapted to this volume → pulmonary edema; ↓ C.O. → ↑ peripheral resistance → worsening regurg. → death from pulmonary edema.

- **CHRONIC:** gradual development of L ventricular failure → dyspnea, orthopnea, PND.
AORTIC REGURGITATION

- PHYSICAL FINDINGS:
- Widening of the pulse pressure, elevated systolic, lower diastolic.
- High-pitched, blowing murmur during diastole.
CARDIAC CONDUCTION & RHYTHM DISORDERS

CHAPTER 27
CARDIAC CONDUCTION & RHYTHM DISORDERS

See text re the conduction system, depolarization, repolarization, etc.
PREMATURE VENTRICULAR CONTRACTIONS

- “PVC’s”
- Ectopic ventricular pacemaker.
- Ventricular filling not complete, insufficient ejection of blood into the arterial system.
- Pulse absent or greatly diminished.
- Clinically insignificant in the absence of heart disease.
- In a patient w/ cardiac disease, predispose to more serious arrhythmias.
VENTRICULAR TACHYCARDIA

- “V-TACH”
- Ventricular rates to 250 bpm.
- Onset can be sudden.
- The heart is pumping, sort of, except there is reduced filling during diastole which can compromise C.O. severely.
- Also, can convert to more serious and life-threatening arrhythmias, so treatment needed, quickly.
VENTRICULAR FIBRILLATION

- “V Fib.”
- The ventricle “quivers” but does not contract; no cardiac output.
- Fatal if not reversed within minutes by cardioversion / defibrillation.
HEART BLOCK

- Disruption of conduction along the path from the AV node thru the Bundle of His thru the Perkinje fibers.
- 3 FLAVORS:
  - 1) 1$^{\text{ST}}$ DEGREE.
  - 2) 2$^{\text{ND}}$ DEGREE.
  - 3) 3$^{\text{RD}}$ DEGREE OR COMPLETE HEART BLOCK.
HEART BLOCK

- 1\textsuperscript{st} degree heart block asymptomatic, no treatment needed.
- 2\textsuperscript{nd} and 3\textsuperscript{rd} degree potentially fatal, require placement of a pacemaker.
HEART FAILURE AND CIRCULATORY SHOCK

CHAPTER 28
HEART FAILURE

**INVOLES:**

1) Decreased pumping ability of the heart (cardiac output).
2) Decrease in cardiac reserve.

Cardiac reserve = the ability of the heart to increase C.O.
CARDIAC OUTPUT

- STROKE VOLUME = the amount of blood ejected per cardiac cycle.
- C.O. = STROKE VOLUME X HEART RATE
- Heart rate- regulated by the A.N.S.
- Stroke Volume- regulated by:
  - 1) Pre-load.
  - 2) After-load.
  - 3) Contractility.
YER “LOADS”

- **PRE-LOAD** = the loading condition of the heart at the end of diastole = end-diastolic volume.
- **AFTER-LOAD** = the force the heart must generate to eject the blood.
- **AFTER-LOAD IS AFFECTED BY:**
  - 1) Systemic vascular resistance.
  - 2) Ventricular wall tension.
Therefore, after-load may impair ventricular ejection and increase wall tension if the ventricles can’t generate sufficient pressure.

Myocardial oxygen demand is proportional to the wall tension → increased wall tension → increased demand for oxygen.
CONTRACTILITY

- The ability of the contractile elements of the heart muscle to interact with and shorten against a load.
ADAPTIVE MECHANISMS

- Affect cardiac reserve.
- See text.
- FRANK-STARLING MECHANISM
FRANK-STARLING MECHANISM

- ↑ stroke volume by ↑ ventricular end-diastolic volume.

- w/ ↑ diastolic filling → ↑ stretching of the myocardial fibers → ↑ force of contraction.

- In heart failure → normal C.O. at rest, BUT → becomes ineffective when the heart becomes overfilled and the muscle fibers are over-stretched.
FRANK-STARLING MECHANISM

- w/ exertion → less ↑ in C.O at any given increase in LV EDV → produces an elevation of LV and pulmonary capillary congestion → pulmonary congestion → Sx’s.

- Further increases in ventricular filling may produce a decrease in C.O.
CONGESTIVE HEART FAILURE

- Heart failure acc. by congestion of body tissues (pulmonary, systemic venous).
- **CAUSED BY:**
  - Acute MI, hypertension, cardiomyopathies.
  - Excessive work demands- hypermetabolic states (thyrotoxicosis), and fluid overload (renal failure)
  - Valvular stenosis, regurg.
- See table 28-1.
HIGH-OUTPUT VS. LOW-OUTPUT FAILURE

- CHF IS LOW-OUTPUT FAILURE.

- HEART FAILURE CAUSED BY THINGS SUCH AS THYROTOXICOSIS AND AVM’s ARE HIGH OUTPUT, AT LEAST AT FIRST.
SYSTOLIC VS. DIASTOLIC FAILURE

- **SYSTOLIC**- IMPAIRED EJECTION DURING SYSTOLE.

- **DIASTOLIC**- IMPAIRED FILLING DURING DIASTOLE.

- CHF HAS ELEMENTS OF BOTH.
MANIFESTATIONS OF CHF

- Fluid retention, edema.
- Fatigue, ↓ exercise tolerance.
- Cachexia, malnutrition, cyanosis.
- Diaphoresis, tachycardia.
- Pulmonary congestion: SOB, dyspnea, orthopnea, PND; chronic, dry, non-productive cough → bronchospasm → wheezing = “cardiac asthma.”
MANIFESTATIONS OF CHF

- Congestion of the viscera- esp. the liver, spleen → ascites. Can interfere w/ digestion and absorption → malnutrition, cachexia.

- Findings: pitting edema, weight gain, ascites, JVD, hepatosplenomegaly.
RIGHT HEART FAILURE VS. LEFT HEART FAILURE

LEFT-SIDED FAILURE

- CAUSES: MI, cardiomyopathy. Also seen in stenosis and regur. of the aortic and mitral v.

- ↓ C.O. from the LV → ↑ LA & LV EDP → pulmonary congestion → pulmonary edema
RIGHT HEART FAILURE VS. LEFT HEART FAILURE

RIGHT-SIDED FAILURE

- **CAUSES:** THE MOST COMMON CAUSE OF RIGHT HEART FAILURE IS IS L HEART FAILURE.
- Also seen in: cardiomyopathy, stenosis or regurg. of the pulmonic or tricuspid v., RV infarction (not common), and in pulmonary hypertension, pulmonary embolus = COR PULMONALE.
CARDIOGENIC SHOCK

“Pump failure.”

Most common cause is MI. Also arrhythmias, disruption of valvular function (ruptured chordae tendonae), and in end-stage coronary artery disease and cardiomyopathy.
CARDIOGENIC SHOCK

- THE RESULT

- ↓ C.O. → HYPOTENSION → ↑ SYSTOLIC VASCULAR RESISTANCE → ↑ AFTER-LOAD.

- VENOUS RETURN ADDED TO BLOOD NOT EJECTED → ↑ PRE-LOAD.

- ALL WORK TO FURTHER COMPROMISE CARDIAC FUNCTION.