I. Heart Failure
   a. Heart unable to maintain sufficient cardiac output to meet tissue metabolic demands
   b. Increasing incidence
   c. Most common reason for hospitalization in people over 65 years of age
   d. Etiological factors
      i. Cardiac disorders: myocardial ischemia, HT, dilated cardiomyopathy
   e. Pathogenesis
      i. Impaired ability of myocardial fibers to contract (systolic failure)/ relax (diastolic failure)/ both
      ii. Results in blood flow congestion in systemic/pulmonary venous circulations
   f. Compensatory Mechanisms
      i. Restore cardiac output to normal
      ii. Mechanisms
         1. SNS activation
         2. Increased preload
         3. Myocardial hypertrophy (remodeling)
         4. Sympathetic nervous system activation
            a. Due to baroreceptor reflex stimulation (baroreceptors detect falls in blood pressure)
            b. CNS increases sympathetic nerve activity to the heart causing venoconstriction
            c. Renal juxtaglomerular cells release renin to activate the RAAS cascade, leading to increase Na and water retention
         5. Increased preload
            a. Due to reduced EF with resultant increase in residual ESV
            b. Decreased CO to kidney will reduce glomerular filtration causing fluid conservation
            c. Activates RAAS cascade leading to elevated blood volume
         6. Myocardial hypertrophy and remodeling
            a. Due to chronic elevation of myocardial wall pressure
            b. High systolic pressure in the ventricle is needed to overcome a high afterload
            c. Neurohormonal factors: hypertrophy
            d. Angiotensin II: remodeling
      iii. Detrimental to cardiac tissue in the long term
         1. Current heart failure management is aimed at reducing long-term effects
   g. Clinical Manifestations
      i. LV failure is most common

ii. Often leads to RV failure

h. Left Sided
    i. Backward effects: blood accumulates in pulmonary circulation
        1. Pulmonary congestion, dyspnea
    ii. Forward effects: insufficient CO with diminished oxygen and nutrient delivery to peripheral tissues and organs

i. Right Sided
    i. Pulmonary disorders result in increased pulmonary vascular resistance. This leads to high afterload and resultant right ventricular hypertrophy.
    ii. Backward effects: congestion in systemic venous system
    iii. Forward effects: low output to left ventricle leads to low CO

j. Biventricular
    i. Most often due to primary left-sided HF which progresses to right-sided HF
    ii. Clinical manifestations
    iii. Reduced CO
    iv. Pulmonary congestion: LHF
    v. Systemic venous congestion: RHF

k. Diagnosis
    i. Classification and staging
    ii. According to symptom severity
    iii. FACES: fatigue, activity limitation, congestion, edema, shortness of breath
    iv. B-type natriuretic peptide level
    v. X-ray and echocardiography

l. Treatment
    i. Improve Co while minimizing congestive symptoms and cardiac workload
        1. Decrease preload
    ii. Reduce preload: reduce intravascular volume
        1. Diuretics, ACE-I
    iii. Reduce afterload
        1. β-blockers
    iv. Increase contractility → Digoxin
        1. Digitalis, other cardiac glycosides
    v. Pacemakers: synchronize ventricular contraction

II. Cardiac Dysrhythmia

a. Arrhythmia
b. Abnormal cardiac rhythm: impulse generation or conduction
c. 3 major types
    i. Abnormal sinus rhythm rates → tachy, brady
    ii. Abnormal impulse generation sites: ectopic sites
    iii. Disturbed conduction pathways
d. Can indicate underlying pathophysiological disorders
e. Can impair CO

f. Treatment
    i. When symptomatic or progressive
    ii. Anti-arrhythmic drugs → can cayse arrhythmias too
    iii. Increase CO: pacemakers, pro-contractile drugs
    iv. Ablation procedures → get rid of other nodes to retain to normal PQRST
    v. Heart Attack → give patient nitroglycerin under the tongue, vasodialate coronary arteries
        1. Give aspirin (blood thinner) break up any small blood clots