I. Anatomy and physiology review
   A. Layers
   B. Chambers
   C. Heart valves
   D. Flow of blood
   E. Blood supply of myocardium
      1. RCA
      2. L Main
      3. LAD
      4. Circumflex
   F. Cardiac cycle
      1. Systole
      2. Diastole
   G. Cardiac output and cardiac index - SV x HR. CI = CO /body surface area.
      1. Preload
      2. Afterload
   H. Cardiac pressures p. 1557 of Black and Hawks
      1. R atrium
      2. R ventricle
      3. Pulmonary artery
      4. L atrium
      5. L ventricle
   I. Electrophysiology
      1. Properties of heart
         a. Excitability - ability to respond - Na and K
         b. Automaticity (rhythmicity) - ability to initiate impulse spontaneously and repetitively without neurohormonal influence
            (1) SA node  60-100 bpm
            (2) atria  60-100
            (3) AV node  40-60
            (4) Ventricles 20-40
         c. Contractility - extracellular calcium required! Then triggers more calcium from sarcoplasmic reticulum. Significance: Ca channel blockers alter cardiac rate but not skeletal muscle contraction.
         d. Refractoriness - inability to respond to new stimulus while still in state of depolarization - can shorten as HR increases
(1) ventricles - absolute refractoriness - .25 to .3 sec
(2) ventricles - relative refractoriness - .05 sec
e. Conductivity - move electrical impulses along and across cell membranes of muscle mass but not through fibrous bands.

2. SA node -
3. AV node - delay is .07
4. Bundle of His and bundle branches
   a. R bundle
   b. L bundle - 2 fascicles (branches)
5. Purkinje fibers

II. Monitor waves - p. 120 in Urden.
A. P
B. QRS
C. T
D. PR Interval (PRI)
E. ST segment
   1. Depressed
   2. Elevated

III. Rate
A. Using monitor paper
B. Six second strip

IV. Rhythms - p. 123, Urden, Stacy, and Lough
A. Normal sinus rhythm
   1. Rate
   2. Rhythm
   3. P wave
   4. PRI
   5. QRS complex
      a. Shape
      b. Width (duration)
   6. Etiology
   7. Treatment
B. Sinus bradycardia
   1. Rate
   2. Rhythm
   3. P wave
   4. PRI
   5. QRS complex
      a. Shape
      b. Width
   6. Etiology
7. Treatment - atropine, pacer (see p. 1691 in Black) - look for pacer spike (p 1696), Oxygen

C. Sinus tachycardia
1. Rate
2. Rhythm
3. P wave
4. PRI
5. QRS complex
   a. Shape
   b. Width
6. Etiology - FEVER, shock, pain, meds (including dopamine), hormones (epi)
7. Treatment - varies by cause. Channel blockers, beta blockers, oxygen!

D. Sinus dysrhythmia - variance of NSR with respiratory cycle

E. Premature Atrial Contraction (PACs)
1. P wave - shape varies
2. QRS complex - < .12

F. Paroxysmal supraventricular tachycardia (PSVT) - begins and ends suddenly
1. Rate - 150 - 250
2. Rhythm - regular
3. P waves - rate may be too fast to see P wave
4. PRI - normal
5. QRS - normal
6. Etiology - caffeine, nicotine, digitalis, mitral prolapse, CHF
7. Treatment - Adenocard (adenosine) - antiarrhythmic (slows the conduction in the AV node), vagal maneuver, Ca channel blockers, digitalis, and cardioversion.

G. Atrial flutter - several P waves called flutter waves for each QRS

H. Atrial fibrillation
1. Rate
   a. Atrial- 350 to 600 fibrillatory waves
   b. Ventricular - 60 to 100 = controlled; >100 = uncontrolled
2. Rhythm - irregularly irregular ventricular complex
3. P wave - fibrillations
4. PRI - absent
5. QRS - normal
6. Etiology - various sites in atria
7. Treatment
   a. Convert electrically or chemically (amiodarone, calcium channel blockers, beta blockers, dig)
   b. Control the rate with medication
   c. NOTE: mural thrombi
I. Premature ventricular contractions (PVCs)
   1. Rate - underlying rhythm
   2. Rhythm - Early QRS complex
   3. P wave - absent or after PVC
   4. PRI - absent
   5. QRS - > 0.12 - wide with bizarre shape
      a. Unifocal
      b. Multifocal
   6. Etiology - myocardial ischemia (may be secondary to MI, especially anterior), electrolyte (especially K & Ca) imbalance, hypoxia, acidosis, heart disease, meds.
   7. Physiology - ectopic focus, impulse spreads through unusual pathways

J. Ventricular tachycardia
   1. Rate - > 100
   2. Rhythm - usually regular
   3. P wave - unrelated to QRS
   4. PRI - NA
   5. QRS - > 0.12, bizarre shape
   6. Etiology - see PVCs. NOTE: some antidysrhythmics can cause more serious dysrhythmias than those the drugs were intended to treat. E.G.: amiodarone.
   7. Physiology - repeating ectopic focus. Usual conduction pathway bypassed
   8. Treatment - pharmacologic or electrical defibrillation. Pulseless VT requires immediate defib, epi, and CPR. For pts. with a pulse but may have symptoms such as decreased level of consciousness or hypotension, lidocaine (emergency Rx), amiodarone (sustained V tach), and/or synchronized cardioversion may be utilized. Chronic VT is treated with antiarrhythmics (see NOTE above) or an ICD.

K. Ventricular fibrillation
   1. Rate - NA
   2. Rhythm - irregular - no recognizable QRS
   3. P Wave - absent
   4. QRS - NA
   5. Etiology - frequently preceded by VT.
   6. Physiology - may be fine or coarse. Results from single or multiple ventricular foci. Ventricles only quiver.
   7. Treatment - DEFIBRILLATION. May use epi and antidysrhythmics and repeat defib. All supportive CPR measures are instituted. 25 lbs. pressure on defibrillator paddles. New policy at BHMC. AEDs by laymen.

L. Ventricular asystole

M. AV blocks
N. Bundle branch blocks

V. Assessment
A. History - present illness, cardiovascular status, family history, and lifestyle.
B. Safety issues
   1. It’s considered cardiac in origin until proven otherwise. SEE PAGE 1564, BLACK, FOR DIFFERENTIAL DIAGNOSIS.
   2. Little correlation between severity of pain and severity of cause.
   3. Poor correlation between location of pain and the source due to referred pain.
C. Physical Exam
   1. General appearance: color, skin temp, diaphoresis, position, facial expression. NOTE: tongue now thought to be most sensitive site for observation of central cyanosis.
   2. Jugular vein distention: noted with fluid volume overload and right ventricular dysfunction. LEARN Box 10-1 for procedure.
   3. PMI - apical impulse - a normal pulsation visualized on the chest wall. See figure 10-3 in Urden.
   4. Peripheral examination - cyanosis may indicate decreased cardiac output or PVD.
   5. Palpation:
      a. Arterial pulses - diminished or absent may indicate decreased CO or PVD. Doppler ultrasound to be used if pulse not palpable. Once found, MARK it.
      b. Capillary refill - alteration indicates arterial insufficiency.
      c. Edema - peripheral, lumbar, body. See scale: Table 10-2 (Urden).
      d. Ck. for deep vein thrombosis - bedrest and MI are risk factors.
      e. Evaluate thoracic and abdominal pulsations - a prominent aortic pulsation (vs. a forward movement felt by using firm fingertip pressure just above the umbilicus).
   6. Ausculatation:
      a. Blood pressure!!!!!!!
      b. Bruits
      c. Heart sounds - sounds are loudest “downstream” from the valve.
         (1) S1
         (2) S2
         (3) S3 and S4
         (4) murmurs - p. 111, Table 10-3, Urden.
         (5) pericardial friction rub
   7. Laboratory study assessment
      a. Potassium
         (1) hyperkalemia - peaked T wave, widening QRS, v fib or standstill.
(2) hypokalemia - PVCs, V tach, F fib, prominent U wave.

b. Calcium
(1) Hypercalcemia - bradys, AV blocks, BB blocks. Potentiates dig.
(2) Hypocalcemia - (common in many critical pts.) - decreased myocardial contractility with decreased CO and hypotension, decreased responsiveness to dig. ST segment lengthens. Sometimes brady, VT, asystole.

c. Cardiac enzymes
(1) creatine kinase - MB
(2) troponin I and troponin T

d. WBCs - rise with inflammatory process.

e. Coag studies

f. ECG evaluation - see p. 1715 in Black.

VI. Acute Myocardial Infarction - p. 160, Urden; p. 1707, Black. AKA heart attack, MI, coronary occlusion, a “coronary.” Characterized by the formation of localized necrotic areas (cell death) within the myocardium.

A. Etiology and risk factors - most commonly caused by the complete or nearly complete occlusion of a coronary artery from coronary artery spasm, coronary artery thrombosis, or hemorrhage into a plaque. Risk factors: same as angina and other CAD.

B. Pathophysiology - irreversible damage to the myocardium as a result of ischemia. Ischemia (noted by pain) can be noted within 10 seconds after occlusion; cellular death occurs about 15-20 minutes after occlusion. In addition, in the presence of the lactic acid (the byproduct of anaerobic metabolism) that is seen in ischemia, the cardiac cells become less functional. As the acidosis increases, the function, including pumping ability, decreases. The acidosis also allows the cells to be more vulnerable to the lysosomal enzymes within the cell, causing intracellular digestion, and leads to conduction system disorders and dysrhythmias. As the cells necrose, intracellular enzymes such as Troponin and CPK are introduced into the bloodstream.

1. Types - subendocardial, intramural, subepicardial and transmural.
2. Zones -
   a. Zone of infarction and necrosis
   b. Zone of hypoxic injury 0 potential for recovery with oxygen
   c. Zone of ischemia - Recovers easily.

3. Sites
   a. Most common - anterior wall of the left ventricle near the apex. Fed by the LAD.
   b. Posterior wall of the left ventricle near the base - Circumflex
   c. Inferior surface of the heart. RCA
C. Clinical manifestations and diagnostic findings


2. ECG - changes in Q wave, ST segment, and T wave. **The first changes in an MI occur in the ST segment.** Then the T wave, then the Q. ST segment and T wave changes usually resolve, Q changes remain evident. See p. 160 (Urden) and 1711 (Black). 12 lead on p. 1711.

3. Laboratory tests -
   a. Troponin-I - see attachment. Absolutely positive at 0.9 ng/mL. 7 hrs. to 5 days.
   b. CK-MB - Above 2.5. 3 hrs. - 3 days
   c. CPK - Above 374
   d. WBC - leukocytosis of 10-20,000 shows the second day after MI, lasts approx 1 week.

4. Echo and TEE

D. Emergency Care

1. EMS or ER - keep client calm, give oxygen, start IV, connect to heart monitor. CPR if needed. Thrombolytic therapy may be started in ER. Begins with Aspirin, 325 mg., chewed. Reopro (Integrelin, Agristat), TPA. Dobutamine or dopamine to elevate pressure. Lopressor (metoprolol) may be ordered to lower rate and pressure. Port chest. 12 lead ECG. TNG SL. Chest pain lab. COMMUNITY EDUCATION NOTE: An aspirin - chewed - at the onset of symptoms decreases the mortality rate by 23%. Pain meds administered - see below.

2. Cath lab - frequently pts. sent to cath lab before being sent to CCU - differential diagnosis for severe pain. Immediate treatment of clot or other blockage. Stent placement. Diagnose need for immediate surgery.

3. CCU - First 24 hrs is the time of highest risk of sudden death. First 6 hrs is the crucial time frame for salvage of the myocardium.
   a. **Pain control** is a priority because pain indicates ischemia - pain increases preload and, therefore, myocardial oxygen demands.
      (1) MORPHINE relieves pain, provides euphoria for the feeling of doom (thus helping with anxiety), and dilates bronchioles.
      (2) Nitroglycerin drip - decreases preload and afterload.
      (3) Beta blockers, ACE inhibitors, and statins may be used.
   b. ECG monitoring for arrhythmias.
   c. Oxygen - treat tissue hypoxia
   d. Anticoagulation - TPA, aspirin, ReoPro, Plavix, Lovinox (1mg/Kg.), heparin. BLEEDING is the death-producing, most common side-effect of the anticoagulants. Other complications include allergic reactions and stroke.
   e. Antiarrhythmics - arrhythmias cause 40 to 50% of deaths from
AMI. Lidocaine, etc. for dysrhythmias. V-tach, V-fib.

f. Goals of treatment - successful reperfusion; relief of chest pain; prevention of dysrhythmias.

E. Medical management - goal of treatment is to balance the heart's ability to function with the risk of developing hypovolemia, hypoxemia, muscle atrophy, and pulmonary embolus.

1. Rehabilitation
a. Physical activity - bedrest less than 24 hrs. usually. Don't let heart rate rise more than 25% above resting level. Blood pressure must not rise more than 25 mm Hg above normal. Passive exercises for those in whom complete bed rest required. Educate the client: as pt. improves, they must observe for signs of overexertion: dyspnea, chest pain, tachycardia, sense of exhaustion. Teach about combination of cold and exertion.
c. Education: A&P of heart, CAD, risk factors of CAD, behavioral counseling, sometimes home activities.
d. Sexual activity - sexual intercourse may resume 4-8 weeks after an MI if physician agrees. (Activity equivalent to climbing 2 flights of stairs.) NO eating before sex. May need TNG beforehand.
e. Smoking - stop.
f. Anticoagulation - ASA

F. Complications
1. Dysrhythmias - ventricular ectopy, SVT, heart block, bradycardia.
2. Cardiogenic shock - 9% of deaths; 80% of those who develop cardiogenic shock EXPIRE.
   a. Symptoms: low BP, diaphoresis, rapid pulse, restlessness, cold clammy skin, ashen skin color.
   b. Treatment - IV fluids, relief of pain, treat dysrhythmias, Levophed (norepinephrine), dopamine, dobutamine. Dobutamine, epi, isoproterenol increase cardiac contractility and cardiac output. Vasodilators (nitroprusside) may be used to increase circulation to improve tissue perfusion. (TNG).
   c. Monitor ECG and pulmonary artery catheter!!! PA cath used to monitor cardiac pressures, cardiac index or output, and determine amount of fluids that can be administered to the client.!!
3. Heart failure and pulmonary edema - disables 20% of clients and is responsible for 33% of deaths after an MI.
4. Pulmonary embolism - PE - secondary to phlebitis or thrombus from leg, pelvis, heart (due to A. fib). Occurrence: 10-20% of MIS.
5. Recurrent MI -
6. Complications due to myocardial necrosis
   a. V. aneurysm, myocardial rupture, v. septal defect (VSD), and
ruptured papillary muscle. Occur 7-10 days post MI, usually.

(1) Symptoms: CHF. Severe mitral insufficiency develops secondary to papillary muscle rupture. Ventricular dysrhythmias occur secondary to v. aneurysm. Symptoms of cardiac rupture are those of cardiac tamponade.

(2) Treatment: decrease workload of heart, increase oxygen supply, surgery, pericardiocentesis for tamponade.

7. Pericarditis - pericardial friction rub - c/o chest pain that is aggravated with movement, deep inspiration, cough. Pain is relieved by sitting up and leaning forward. Treat with NSAIDS or acetaminophen.

8. The pts. that do the worst are elderly (over 80), previous history of heart disease or uncontrolled diabetes mellitus, anterior location of MI (30% mortality), and/or hypotension.

G. Nursing management:
1. Treatment of pain:
   a. Assessment
   b. 12-lead ECG
   c. Drug therapy
   d. Be calm, reassure client
   e. Limit visitors

2. Dysrhythmias
   a. Monitor
   b. Keep alarms set at all times
   c. Document rhythm strip when rhythm changes and/or q shift.
   d. Report 6 or more PVCs to MD
   e. Administer drug therapy and MONITOR FOR APPROPRIATE EFFECTS AND SIDE EFFECTS
      (1) lido: widening QRS, confusion
      (2) procainamide: hypotension, widening QRS
      (3) Nitro gtt: hypotension, headache
      (4) isoproterenol: hypotension, brady
   f. Monitor K level - know interaction of K and digoxin
   g. Maintain patent IV or hep lock.

3. Nausea - Clear liq diet

4. Cardiac failure
   a. Assess for cardiac failure: mental status, lung sounds, heart sounds, urinary output, peripheral perfusion, vital signs, jugular vein distention, dependent edema (sacral), weakness, fatigue, SOB with activity, change in ABGs
   b. Record hemodynamic pressures of PA cath q 2-4 hrs and pm; watch for wedge pressure greater than 18 mm Hg, cardiac output less than 4 L/min, cardiac index less than 2.5 L/min.
   c. Monitor effects of meds
5. Impaired gas exchange
   a. Oxygen
   b. ABGs
   c. Assess skin color, capillary refill, LOC, VS q 2-4 hrs and prn.
   d. Prepare for intubation and vent for hypoxia.

6. Feeling of powerlessness
   a. Let pt. talk
   b. Educate client
   c. Allow choices when possible

7. Anxiety and fear
   a. Limit # of nursing personnel - stability
   b. Do not avoid questions
   c. Allow verbalization of fears
   d. Educate - frequent assessments are routine
   e. Repeat info for family, allow family in when possible
   f. Comfortable, quiet environment

8. Constipation
   a. High fiber diet
   b. Encourage fluids as much as possible
   c. Stool softeners or laxatives
   d. Use BSC rather than bedpan if possible

9. Activity intolerance
   a. Monitor VS before and after activity
   b. Watch for tach, dysrhythmias, dyspnea, diaphoresis, pallor after activity
   c. Encourage pt to verbalize fatigue
   d. Provide assistance with ADL as necessary; provide rest p meals.
   e. Increase activity as ordered

10. Heart failure - see CHF
11. Fluid volume excess
    a. Monitor I and O - both.
    b. Semi-Fowler’s position
    c. Weight daily
    d. Watch for jugular vein distention, edema, presence of anasarca
    e. Breath sounds: adventitious. Monitor for dyspnea or tachypnea, sudden extreme shortness of breath, feelings of panic.
    f. Note increased lethargy, hypotension, muscle cramping
    g. Evaluate effectiveness of diuretics and potassium supplements
    h. Dietary consult for diet rich in potassium if needed

12. Life-style modifications - education begins on admission
    a. Discuss A & P, define “heart attack,” CAD
    b. Have client identify own risk factors
    c. Discuss how risk factors, which ones can be changed, such as diet,
exercise, cholesterol, smoking, treatment of hypertension, treatment of D.M.

d. Medications and effects and side-effects.

13. Impaired skin integrity
a. Inspect skin all over at least daily!
b. Assist with active or passive range of motion exercises
c. Reposition q 2 hrs in bed or chair
d. Use sheepskin, elbow and heel protectors, other devices as necessary (including special beds)

CHAPTER 56, or p. 1649 or p. 166

VII. Heart Failure - state in which the heart is unable to pump enough blood to meet the metabolic needs of the body at rest or during exercise. Is not a disease but is a group of symptoms. May be called congestive heart failure, cardiac decompensation, cardiac insufficiency or ventricular failure
A. Etiology and risk factors - conditions that damage the heart include atherosclerosis (responsible for 2/3 of cases), congenital heart defect, high blood pressure, pulmonary hypertension, MI or valvular disorders.

1. Abnormal loading conditions
a. Preload - refers to the stretch of the ventricular myocardial fibers just before v. contraction. Determined by the condition of the heart valves, blood volume, ventricular wall compliance, and venous tone. Too much preload lessens the force and efficiency of ventricular contraction (Starling’s Law), cardiac output decreases, and the heart fails. Conditions that will cause this include: regurgitation of any of the 4 valves; hypervolemia; congenital defects (left to right shunt); VSD; ASD; PDA (patent ductus arteriosus); heart failure
b. Afterload - the amount of tension that the heart must generate to overcome systemic pressure and allow adequate ventricular emptying. I.E.: how hard the heart must pump to force blood into circulation. Affected by tone of systemic arterioles, elasticity of aorta and large arteries, size and thickness of the ventricle, presence of aortic stenosis, viscosity of blood. When subjected to prolonged high pressures, ventricle will fail. Conditions that result in increased afterload include: aortic valvular stenosis, mitral valvular stenosis, pulmonic valvular stenosis, systemic hypertension, pulmonary hypertension, high peripheral vascular resistance.

2. Abnormal muscle function
a. Intrinsic conditions: MI, myocarditis, cardiomyopathy, and v. aneurysm. These disorders impair contractile function of
myocardial fibrils.

b. Extrinsic conditions: constrictive pericarditis, cardiac tamponade.

3. Precipitating or exacerbating conditions
   a. Physical or emotional stress - increased sympathetic nervous tone with catecholamine release
   b. Dysrhythmias - tachycardia - ventricular filling time decreased and workload and oxygen requirements increased.
   c. Infections - every degree of fever increases metabolism (and oxygen needs) by 7%. Pulmonary infections decrease oxygen available.
   d. Anemia
   e. Thyroid disorders - thyrotoxicosis - accelerates heart rate and increases workload of heart. Hypothyroidism leads to heart failure by predisposing client to atherosclerosis.
   f. Pregnancy - increased metabolic needs
   g. Nutritional deficiency - Thiamine - reduces myocardial contractility, causes tachycardia and ventricular dilation
   h. Pulmonary disease - increased pressure in pulmonary system due to obstructive lung disease, emboli can produce heart failure
   i. Hypervolemia - results from poor renal function, cardiac disease, meds (such as steroids), excessive intake of sodium. Also, too much IV fluids and volume expanders (blood, dextran, albumin).

B. Pathophysiology - the compromised heart has a limited ability to respond to the body’s needs for increased output in situations of stress. The failing heart uses 3 compensatory mechanisms: (1) ventricular dilation, (2) ventricular hypertrophy, and (3) increased sympathetic nervous system stimulation (tachycardia). P. 172
   1. Vent. dilation - muscle fibers of heart lengthen to increase preload and thus cardiac output but get too long and ineffective. A dilated (enlarged) heart requires more oxygen. This results in hypoxia of the cardiac muscle, further compromising the heart’s effectiveness.
   2. Vent. hypertrophy - an increase in the diameter of the muscle fibers to increase contractility. This also increases the demand for oxygen. As the heart hypertrophies, it can outgrow the coronary blood supply and become hypoxic and ineffective. Beyond a certain point, the hypertrophy begins to impede ventricular emptying as it blocks the valve areas.
   3. Increased sympathetic stimulation - causes venous and arteriolar constriction, which increases afterload and the myocardial workload. It reduces renal blood flow, which causes water and sodium retention. Tachycardia occurs secondary to the increased circulating catecholamines and as a result of stretching of the vena cava. Tach eventually results in further failure.

   1. Left ventricular failure - causes pulmonary congestion or disturbances in
respiratory control mechanisms, such as Cheyne-Stokes respirations.
a. dyspnea - degree of SOB can vary - find out exactly how much exertion is required to cause symptoms.
b. cough - common symptom - may produce frothy, blood-tinged sputum. Bilateral crackles usually heard when frothy sputum present.
c. orthopnea - more advanced dyspnea. Client sits up with both hands on knees, sleeps on pillows at night, or sits up in reclining chair to prevent edema in legs from absorbing and causing acute distress.
d. PND - paroxysmal nocturnal dyspnea - clients awaken with feeling of severe suffocation. Respirations are labored, may wheeze. May take 30 minutes for relief to occur. Primary cause is the absorption of edema from lower extremities at night when pt. lies flat in bed.
e. acute pulmonary edema - medical emergency - capillary pressure in lungs becomes so elevated that fluid is pushed from circulating blood into interstitium and then into alveoli, bronchioles and bronchi. If not relieved, the client suffocates - drowns in own fluid. Symptoms are terrifying:
(1) severe dyspnea, orthopnea
(2) pallor, possibly cyanosis
(3) tachycardia
(4) expectorate large amts. frothy, blood-tinged sputum
(5) wheezing and bubbling respirations
(6) fear
(7) sweating
(8) nasal flaring and use of accessory breathing muscles
(9) tachypnea
(10) vasoconstriction
(11) hypoxia
(12) gallop heard
(13) pulsus alternans
(14) cerebral hypoxia - further anxiety, irritability, restlessness, confusion, impaired memory, bad dreams, insomnia result. Respirations become inadequate due to hypercapnia.
(15) fatigue and muscle weakness
(16) renal changes - nocturia occurs due to increased formation of urine at night as more fluid is absorbed and blood flow to kidneys improves. Nocturia then increases fatigue. Oliguria is late sign of failure.
(17) if renal artery pressure falls, sodium and water are retained and renin-angiotensin-aldosterone mechanism is activated. These mechanisms result in 30% increase in blood volume
and further edema.

f. Cheyne-Stokes respirations - apparently due to alteration in blood flow to brain

2. Right ventricular failure - peripheral edema and venous congestion of organs (all organs that drain into vena cava) occur.
   a. Liver engorgement - hepatomegaly and abd. pain result. The pain eventually disappears in chronic heart failure. Cirrhosis also eventually occurs, manifested by ascites and jaundice.
   b. Gi tract engorgement - anorexia, nausea, bloating. Metabolic needs may be up, caloric intake is down, tissue wasting and cachexia result. (Anorexia and nausea may also result from dig. toxicity.)
   c. Dependent edema - results from increased hydrostatic pressure - when clients are ambulatory, edema begins in feet and ankles and ascends up the lower legs. Often subsides after a night’s rest. Concurrent jugular vein distention differentiates the edema of heart failure from that of lymphatic obstruction, cirrhosis, and hypoproteinemia.
   d. Anasarca - late sign of failure
   e. Cyanosis may result as edema reduces peripheral blood flow.
   f. Anxiety, fright, depression.

3. High-output vs. low-output failure
   a. High output means heart putting out normal amount - requirements are just too much - e.g. Paget’s disease.
   b. Low output - from heart disease.

D. Diagnostic tests
   1. Chest x-ray - may see enlarged heart, pulmonary and venous congestion, interstitial edema, even pleural effusions
   2. ABGs - results vary according to degree of failure and response of client. Late failure shows acidosis, hypoxemia, hypercapnia.
   3. Liver enzymes - document degree of liver failure
   4. ECG - may show cause of failure
   5. Echo - shows cardiac chamber size, ventricular function, and helps assess disease involved.

E. Medical management - goals are to improve pump performance and reduce cardiac workload
   1. Positioning - high Fowler’s. Keep the legs dependent in acute failure.
   2. Oxygen - high concentrations - nonrebreathing mask achieves highest oxygen concentrations of masks. May require entubation and vent.
   3. Bronchodilators
   4. Digitalis - is most effective in failure associated with low cardiac output caused by ischemic, rheumatic, hypertensive, or congenital heart disease. Used for a. fib, the most common dysrhythmia seen in heart failure.
Should be used with caution in acute MI due to increased myocardial oxygen demand. Dig. levels can become elevated as a result of medication interactions with quinidine, verapamil, and amiodarone. Also, some antibiotics and anticholinergic agents. Elderly and clients with advanced cardiac disease are therefore the most prone to toxicity. Watch K and Mg.

a. Increases ventricular contractility (inotropic effect)
b. Increases ventricular emptying
c. Slows conduction of impulses through AV node and Purkinje fibers
d. Augments stroke volume
e. Increases cardiac output

5. Dopamine, dobutamine, amrinone, milrinone - inotropic agents - used to treat severe low-output heart failure. Dopamine in low doses causes vasodilation in the renal, mesenteric, cerebral and coronary vascular beds, primarily resulting in increased renal blood flow, glomerular filtration rate, and sodium excretion. At moderate doses (4-8 microgm/Kg/min), dopamine causes an increase in heart rate, stroke volume, and cardiac output. Intense vasoconstriction (alpha-adrenergic effect) occurs at rates above 8 mics. Increase in heart rate does increase oxygen usage in myocardium. Dobutamine is a synthetic derivative of dopamine; it increases heart rate, AV conduction, and myocardial contractility. The heart does not require as much additional oxygen when dobutamine is used vs. dopamine. Amrinone and milrinone - used on short term basis (therefore in the short-stay outpatient cardiac treatment unit) - acts much like dopamine but has no alpha-adrenergic stimulating effect - has cardiac inotropic effects, increases renal blood flow.

6. Reducing preload - diuresis - can produce hypovolemia and hypotension.
7. Reducing afterload - vasodilating agents - nitroglycerin causes venous dilation. Hydralazine (Apresoline) is a frequently used arterial dilator. Sodium nitroprusside dilates both veins and arterioles. ACE inhibitors block vasoconstriction and increase renal blood flow, enhancing diuresis. Some beta-blockers are also in use.

8. Diet - sodium restrictions, potassium supplements are frequent. In advanced failure, oral fluid intake may need to be limited to 1000 ml/day.
9. Rest - bedrest may be ordered temporarily to decrease cardiac workload. However, bedrest should not be so long that the complications of immobility begin. Passive leg exercises and anticoagulant therapy may be utilized to prevent some complications.

F. Nursing management
1. Vital signs every 15 min to 1 hr.
2. Monitor for dysrhythmias
3. Monitor heart and lung sounds q 2-4 hrs. Listen for crackles and gallop.
4. Intake and output at least q 2 hrs. Hourly urinary output usually. Watch
5. Assess mental status at least q 4 hrs.
6. Small meals - rest period following meals
7. High Fowler’s position for dyspnea.
8. Frequent oral care if client mouth-breathing
9. Daily weight to monitor response to diuretic therapy.
10. Monitor for signs of increasing peripheral edema
11. Low sodium diet.
12. Fluid restrictions if necessary
13. Turn, cough, deep-breathe q 2 hrs. for respiratory effects. Provide pressure reduction mattress for skin if necessary.
14. Oxygen
15. ABGs and pulse oximetry
16. Skin color, temperature, keep extremities warm (promotes vasodilation), watch for thrombophlebitis, encourage active and perform passive ROM exercises.
17. Enforce frequent rest periods. Monitor client’s response to activity, noting the development of dyspnea, tachycardia, angina, hypotension, diaphoresis and dysrhythmias. Check vital signs prior to major activity, immediately afterward and 3 min. later. Increase activity as ordered.
18. Monitor dig and potassium levels.

G. Medical management in severe situations
1. Intractable heart failure - may be treated with prolonged bed rest, severe sodium restriction (as much as 250 mg. sodium restricted), fluid restriction of 500 ml/dy, diuresis, and IV dobutamine therapy. The clients may be transferred from an acute unit to a subacute unit on this therapy.
2. Counterpulsation (aortic balloon pump) may be used to decrease afterload.
3. Ventricular assist devices may be utilized in selected individuals - those that stand a good chance of recovery in 5 to 7 days and those awaiting heart transplant. VADs may support circulation either partially or totally. Some battery powered VADs are now available so that the patient can go home.
4. ECMOs (extracorporeal membrane oxygenation systems) are used for short-term (48 hr) hemodynamic stabilization. This system bypasses the entire heart.

H. Surgical management - heart transplant and cardiomyoplasty - the use of the latissimus dorsi to provide the muscle for the heart. Stimulation of the latissimus dorsi muscle begins 2 weeks after surgery. It is at least 7 weeks before the muscle is trained adequately to support the heart.
DISORDERS OF CARDIAC STRUCTURE

VIII. Acute pericarditis  
A. Pathophysiology - can follow common viral infection, myocardial infarction, tuberculosis, bacteremia, or renal failure. Adhesions form along pericardial space, pericardial sac may eventually be obliterated.  
B. Manifestations - primarily chest pain. May mimic MI or pleurisy. Sitting up frequently relieves the pain!!! Auscultation reveals scratchy, leathery, creaky sound heard predominantly and most frequently at the 3\textsuperscript{rd} intercostal space left of the sternal border. Heard best if client holding his breath and it may be necessary for the client to sit up. Fever, chills, malaise may also be noted. And anxiety.  
C. Diagnosis - some changes may be noted on ECG. Lab studies show elevated ESR, elevated WBC.  
D. Medical management - treat the cause, if possible, and treat pain and fever. Treat the anxiety by assuring the client that he is not having an MI.

IX. Acute pericarditis with effusion - fluid may accumulate in the pericardial sac. If it accumulates slowly, 1-2 L. may be present although normally the pericardial sac will only accommodate 80-200 ml fluid. Symptoms as above, but will include muffled heart sounds. Pulsus paradoxus may be present. Echo is diagnostic. Treatment is pericardiocentesis if cardiac compression is evident.

X. Cardiac tamponade - life-threatening complication. Cardiac emergency. Fluid accumulates in pericardial sac rapidly, resulting in decreased venous blood flow to heart, decrease in cardiac output and arterial blood pressure, and narrowing pulse pressure. Tachycardia is a compensatory mechanism. Jugular venous distention, cyanosis of lips and nails, dyspnea, muffled heart sounds, diaphoresis and paradoxical pulse are present as well as the hypotension and tachycardia. Echo shows increasing fluid, may be some non-specific ECG changes. Treatment is pericardiocentesis. STAT.

XI. Hypertrophic cardiomyopathy  
A. Etiology - genetically transmitted.  
B. Pathophysiology - disproportionate thickening of intraventricular septum. Left ventricular wall may encroach on left ventricular chamber. Septal hypertrophy may obstruct left ventricular outflow. Left ventricle becomes stiff, inhibiting flow of blood into chamber, with resultant elevation of pressure in left atrium, pulmonary venous system, and pulmonary wedge pressure.  
C. Manifestations - death may be first symptom. Dyspnea most common symptoms. Angina pectoris, fatigue, syncope, cardiac dysrhythmias with palpitations, PND, and heart failure may be noted. Exertion exacerbates symptoms. 4\textsuperscript{th} heart sound may be noted.  
D. Diagnosis - ECG, chest x-ray, echo, scans useful in diagnosis.  
E. Management - beta-adrenergic blocking agents such as propranolol used to decrease vigor and frequency of contraction and myocardial workload. Calcium
channel blockers may also be used. * Surgical alteration of septum may be helpful.

F. Nursing management - monitor for symptoms of
1. Peripheral edema
2. Pulmonary edema
3. Decreased renal perfusion
4. Decreased CO
5. Diaphoresis
6. Dyspnea, orthopnea
7. Anxiety
8. Frothy, pink sputum

XII. Mitral valve disease - includes stenosis, regurgitation, and prolapse
A. Etiology - rheumatic heart disease is most common.
B. Pathophysiology
1. Mitral stenosis - obstruction of flow of blood from left atrium to left ventricle with resulting increases in pulmonary venous and pulmonary capillary pressures. Left atrium hypertrophies to accommodate the increase in pressure and volume; right ventricle hypertrophies because of the chronic pulmonary hypertension. Decrease in cardiac output can occur due to lower than normal preload.
2. Mitral regurgitation - there is flow of blood through both the aortic and mitral valves during systole, resulting in both atrial and ventricular enlargement. Eventually failure of left ventricle results with an increase in left atrial pressure and an increase in pulmonary pressures. Eventually, right-sided failure can develop.
3. Mitral prolapse - cusps billow upward during systolic contraction. Cusps may enlarge and thicken. Mitral regurgitation may result. INFECTION is a major problem.

C. Manifestations
1. Stenosis - diastolic murmur, atrial fib with irregular pulse and subsequent mural thrombi and systemic embolization.
2. Regurg - when cardiac output falls, pts. experience fatigue and dyspnea first, followed by orthopnea, PND, and peripheral edema. Blowing, high-pitched systolic murmur with radiation to left axilla may be heard. Severe regurg is associated with 3rd heart sound. Atrial fib is common.
3. Prolapse - may be asymptomatic although regurgitant murmur or midsystolic click may be heard. Symptoms, when present, may include tachycardia, lightheadedness, syncope, fatigue, weakness, dyspnea, chest discomfort, anxiety, and palpitations.

D. Diagnosis - echo, x-ray, and perhaps cardiac cath.
E. Medical management
1. Stenosis - diuretics and Na restriction. Dig to treat a fib. Beta-blockers to
decrease heart rate, and anticoagulants unless surgery planned soon.

2. Regurg - restrict physical activities that produce fatigue and dyspnea. Reduce sodium intake, use diuretics, nitrates, and ACE inhibitors.

3. Prolapse - beta-blockers to relieve syncope, palpitations, chest pain. Antibiotics prophylactically before ANY invasive procedure. To include dental.

F. Surgical management - may be necessary at some point in the disease process to prevent failure.

XIII. Aortic valve disease - includes stenosis and regurgitation

A. Etiology
1. Stenosis - congenital defect, degeneration or calcification in the elderly, and rheumatic fever.
2. Regurg - rheumatic fever, syphilis, infective endocarditis. Also connective tissue disorders.

B. Pathophysiology
1. Stenosis - orifice narrows, left ventricular pressures rise, ventricle hypertrophies and dilates, later cardiac output falls and pulmonary hypertension occurs.
2. Regurg - results in volume overload of left ventricle and gradually a decrease in left ventricular stroke volume. Left ventricle dilates and hypertrophies and eventually fails.

C. Manifestations
1. Stenosis - symptoms appear late in course of disease. Angina pectoris is common and is brought on by exertion. PND and pulmonary edema eventually occur. Other late symptoms include palpitations, fatigue, and visual disturbances. Sudden death can result from dysrhythmias and myocardial ischemia. Systolic murmur is audible, systolic thrill over aorta may be noted.
2. Regurg - eventually left ventricle enlarges to point that pts. complain of being aware of heartbeat and palpitations (due to large left ventricular stroke volume. Prominent pulsations in the neck and head-bobbing with each heart beat may be noted. Sinus tach and PVCs can cause palpitations. Increase BP, exaggerated carotid artery pulsations, and water-hammer pulse may be noted. Auscultation: soft, high-pitched, blowing decrescendo diastolic murmur.

D. Diagnosis - echo, ECG, chest x-ray.

E. Medical management
1. Stenosis - avoid activities that cause symptoms. Prophylactic antibiotics for invasive procedures. dysrhythmia treated with antiarrhythmics.
2. Regurg - relieve symptoms - advise only tolerated activity. Prevent infection.

F. Surgical management -
1. Stenosis - clients that are symptomatic have poor prognosis without surgery.
2. Regurg - surgical replacement of the incompetent valve is the only effective long-term intervention. Timing: for acute, severe aortic regurg and left vent. failure, early valve replacement can be life-saving.

XIV. Tricuspid and pulmonic valve disease - relative rare. Tricuspid disorders usually develop in combination with other structural disorders of the heart. Pulmonic valve disorders are commonly due to congenital defects.

XV. Nursing management for valvular heart disease - main focus is to help the client maintain a normal cardiac output. Patients in an acute care unit are monitored closely for a drop in BP, rise in heart rate, or a decrease in urinary output. The chest is auscultated at least q 4 hr for adventitious breath sounds or gallop. Valvular heart disease requires lifelong management. Planning for discharge includes instruction to clients regarding medications, exercise, including the ability to recognize fatigue, and dietary restrictions.

XVI. Cardiac surgery - performed when the probability of survival with a useful life is greater with surgery than without.

A. Valvular surgery
1. M. stenosis - valve commissurotomy, reconstruction or replacement.
2. M. regurg - valve reconstruction or replacement
3. A stenosis - valve replacement or balloon aortic valvuloplasty.
4. A regurg - replacement
5. Types of valves - mechanical; tissue. Picture: p. 1352
6. Mechanical valves are very durable but these clients must be on anticoagulants their entire lives!!! They must be evaluated preop for whether they will take the anticoagulants.
7. Tissue (pig or human) don’t last as long but don’t have as much risk of thrombus formation.

B. Heart transplant
1. Pathophysiology - use of cyclosprine makes transplants possible. Approx. 70% survive 10 years. Infection, cardiac failure, or rejection are the usual causes of death.
2. Technique - usual technique is orthotopic (see p. 1353).
3. Special post-op considerations - recognition and treatment of REJECTION. Higher risk of acute rejection soon after transplantation that decreases dramatically p 3 months. 84% of transplant clients have at least 1 episode of rejection during 1st 3 months. Methylprednisolone is the treatment for these acute rejection periods. (Then we have to observe for infection.)

C. Assisted circulation and mechanical hearts - cardiac failure can lead to multi-
system failure. ECMO or mechanical hearts can be used for a period of time until donor heart available. Artificial hearts are rubber, silicone, and Teflon and are air-powered. Major complications of use include hemorrhage, infection, ATN, and neuro disturbances and permanent use is not recommended.

D. Open heart surgery

1. ECC machine MUST be utilized. This (1) diverts circulation from heart and lungs, (2) performs gas exchange, (3) filters, rewarms or cools the blood, as necessary, (4) pumps oxygenated, filtered blood back into systemic circulation. ECC pump is primed with 3-4 L. RL. Risks are that the pump can crush blood cells, leading to thrombus formation; or create air emboli. Other complications include shock, hemorrhage, fluid overload, hemolysis, and kidney or lung damage.

2. Medical management before surgery
   a. Thorough cardiology workup
   b. Usual anesthesia workup

3. Nursing management before surgery - records baselines for everything. This includes preop medications. Preop teaching, when possible, is extremely helpful. Thorough preop teaching makes a real difference in post-op cooperation and understanding of the routine of both the client and the family. Explanations include all tubes (NG, endo, chest tube, vent, IVs, art line, PA line, foley cath), the type of room (sometimes even a tour) the client will be in post-op, the post-op sched., to include the fact that VS will be q 15 minutes initially post-op; and information about the rules and guidelines of unit and waiting room. Other pre-op care includes antibiotic wash and NAIR or shave in operating room.

4. Post-op nursing management
   a. VS - q 15 min. to q 2 hrs.. All pressures, such as art. line vs. BP cuff, peripheral pulses, filling time, PA pressures if PA line in. (Picture of arterial line and the proper waveform on p. 1263.)
   b. Ck. lung sounds frequently (at least q 1 hr initially). Wheezing may indicate pulmonary edema, bronchospasm, airway obstruction. Bronchodilators and suctioning may be necessary. Atelectasis may be noted. Observe sputum for color and quantity and thickness. Watch vent for inspiratory effort and tidal vol.
   c. Frequent ABGs. - watch for acidosis, “fighting” the vent, etc. Post extubation, observe for signs the pt. needs to be re-intubated.
   d. Observe for hemorrhage, shock, cardiac tamponade, or infection.
   e. Ck. heart sounds frequently (with lung sounds). Listen for pericardial rubs, murmurs, gallops.
   f. ECG - initially post-op, the next AM, and then as ordered. Monitor will be continuous. Pacer wires may be in place.
   g. Chest tubes - ck. for drainage. 1 may be in mediastinum to drain pericardium. May be 100 ml. drainage first hr, up to 500 ml. the
first 24 hrs. Be aware that sudden cessation of drainage may indicate clot in tube or other obstruction.

h. Fluid balance - daily weights

i. Renal function - hrly urines. May be some blood due to hemolysis of ECC initially, then clearing. Fluid challenge may be given for low urinary output. Diuretics, dopamine, or dobutamine may be used if fluid challenge does not work. The additional 3-4 L fluid the patient was given during surgery will gradually absorb and be excreted post-operatively.

j. Electrolytes - immediately post-op, then daily. Observe potassium closely. Potassium may be rechecked if arrhythmias begin. Other blood studies include H & H, protimes or other clotting studies.

k. Hrly neuro cks. - pts. should awaken in 1 to 2 hrs. and be sleepy but otherwise neurologically as intact as they were pre-op. Inability to awaken may result from poor cerebral capillary perfusion during ECC. Disorientation and restlessness may anoxia or embolization. Fatigue or fear can produce confusion. Hemiplegia can result from emboli to brain. Disorientation, delusions, and psychosis may result from (1) being in ICU, (2) sensory deprivation or overload, (3) lack of rest and sleep, (4) fear and anxiety, (5) impersonal atmosphere and feeling of being only a case, (6) desynchronization of circadian rhythm, and (7) side-effects of medications. Post-op depression can occur.

l. Diet post-op will be as tol. Start with sips of water and advance.

m. Pain will be treated as necessary.

n. Some methods of preventing or to treating confusion post-op
   (1) always address client by name - introduce self
   (2) calendar and clock in room
   (3) treat the client, not the machines
   (4) turn cardiac monitor so patient can’t easily see it
   (5) schedule alternating activity and rest throughout day
   (6) allow clients to freely discuss fears and anxieties
   (7) prepare S/O’s for changes in client’s sensorium after surgery.
   (8) explain all interventions to clients and allow time for questions.

o. To prevent the complications of bed rest, plan activity!

p. Teach post-op wound care - delayed wound healing in the leg is common.

q. While rejection is the most common complication post-op heart transplant, the most common cause of death is infection. They need to be taught and be alert to signs of infection.

r. Discharge teaching - begun on admission -
(1) takes approx. 6 wks. for sternum to heal. Driving not recommended, should not lift anything over 5 lbs.  
(2) inspect incisions. Apply Betadine and clean dressings daily. Call MD if infection noted.  
(3) low sodium and low cholesterol diet.  
(4) exercise rehab  
(5) learn to ck pulse, ck it daily for rate and regularity. Call MD if it rises by 20 beats per minute or if new irregularity present.  
(6) also teach client to report dizziness or increased fatigue, sudden weight gain or peripheral edema, or new shortness of breath.

PERIPHERAL VASCULAR DISEASE

XVII. Chronic arterial occlusion
     A. Etiology  
     B. Pathophysiology - Lower limbs vs. upper limbs  
         1. Aorto-iliac bifurcation  
         2. Femoral bifurcation  
     C. Clinical manifestations and diagnostic findings  
         1. Intermittent claudication  
         2. Rest pain!  
         3. Dependent rubor and white pallor  
         4. Reproducible pain  
         5. Coldness  
         6. Aortoiliac disorder Sx  
         7. Femoropopliteal disorder sx  
         8. Popliteal artery sx  
         9. Arteriogram  
     D. Medical management  
         1. Nonpharmacologic  
         2. Pharmacologic  
     E. Nursing management  
         1. Positioning  
         2. Provide warmth  
         3. Prevent vasoconstriction  
         4. Prevent injuries to lower extremeites  
         5. Avoid pain  
         6. Avoid exercise only when arterial ulcer. Bedrest for rest pain.  
     F. Endovascular interventions  
         1. PTA  
         2. Stents
3. Thrombolytic therapy - streptokinase and low dose heparin
   a. Contraindications
   b. Complications
   c. Nursing management

G. Surgical interventions
1. **Arterial bypass**
   a. Femoral artery bypass graft
      1. locations
      2. graft material
      3. preop care
         a) record pulses
         b) no infection
      4. postop care
         a) postop edema - why?
         b) anticoagulants
            i) for thrombosed graft
            ii) longer term
         c) oxygen sat
         d) antibiotics
         e) elastic wraps only when ambulating
      5. Nsg diagnoses
      6. complications
         a) bleeding
         b) reclotting
         c) compartment syndrome

XVIII. Acute arterial occlusion
A. Pathophysiology - trauma, embolism, thrombosis
B. Signs and symptoms - 6 P’s
   1. pain
   2. paresthesia
   3. poikilothermia
   4. paralysis
   5. pallor
   6. pulselessness
C. Treatment
   1. Amputation
   2. Embolectomy
   3. Reconstruction
   4. Meds - anticoagulants, fibrinolytics
D. Nursing management - preop
   1. Comfortable and warm
   2. No pressure on limb
3. Position - level or slightly dependent

E. Aneurysms - definition - 50% increase in size of vessel
1. Causes - atherosclerosis, congenital, trauma, infection, age and nutrition
2. Most common locations
3. Classifications

F. Abdominal aortic aneurysms
1. Symptoms and diagnosis
2. Complication - rupture - usually retroperitoneal - EMERGENCY
   a. Symptoms
   b. Treatment for rupture
3. Elective surgery - midline incision
   a. Complications
      (1) MI
      (2) renal failure
      (3) emboli
      (4) spinal cord ischemia
      (5) changes in sexual function
4. Preop nursing management
   a. Educate patient and family
   b. Careful assessment of pulses
5. Postop
   a. Monitor for hemorrhage
   b. Oxygen saturation
   c. Peripheral tissue perfusion - pulses
   d. Pain
   e. Ischemia of bowel
   f. Spinal cord ischemia

XIX. Aortic dissections - longitudinal splitting of muscle of aorta
A. Etiology and classification
B. Clinical manifestations
C. Complications
D. Management

XX. Thoracic
XXI. Peripheral
XXII. Subclavian Steal Syndrome
XXIII. Thoracic Outlet Syndrome
A. Types
B. Treatment
C. Diagnosis