**Alterations in Cardiovascular Function**

Celeste Armenta, RN
Nurs 210

**Diseases of Arteries and Veins**

- Atherosclerosis
  - Not a single disease entity, can take several forms, depending on individuals age and genetic and physiologic status, and the risk factors to which each individual may be exposed
  - Leading contributor to coronary artery and cerebrovascular disease

**Pathophysiology**

- Lesions progress from endothelial injury to fatty streak to fibrotic plaque to complicated lesion
- Endothelial injury occurs and a series of pathologic events occur
  - Endothelial cells stop making normal antithrombotic and vasodilatory substances
  - Macrophages start sticking to the damaged endothelial surface

**Clinical Manifestations**

- S/S that result from inadequate perfusion of tissues because of obstruction of the vessels that supply them
- Partial vessel obstruction may lead to transient ischemic events, often associated with exercise or stress
- Increased obstruction with thrombosis may result in tissue infarction

**Evaluation and Treatment**

- Complete Health History
- Risk Factors
- PE
- X-ray films
- ECG
- US
- Angiography (identify affected vessel)

**Prevention/ Health Promotion**

- BP checked annually
  - Begin annual check of BP at 18
  - Pediatric BP per well child visit periodic screening
  - Total Cholesterol w/ HDL/LDL fraction
    - All adults Q 5 years starting at age 20
    - HDL Target 40+ mg/dL
    - Triglycerides Target: 150mg/dL or less
  - *These Guidelines are per the Adult Treatment Panel III (ATP III) recommendations of the National Cholesterol Education Program (NCEP)
  - Blood sugar checked beginning at age 40 unless high risk per USPSTF

**Hypertension**
**HTN: Incidence, Prevalence**
- Ubiquitous in US: 50 million Americans have HTN
- Prevalence increases steadily with age: greater in blacks than whites in both sexes, all ages
- HTNsive men outnumber women in young adulthood and early middle age; women outnumber men post-menopause
- Nearly 65% of people aged 65 and older have high BP (>140 systolic, >90 diastolic)
- Relationship between BP and risk of CVD events is continuous, consistent, independent of other risk factors. The higher the BP, the greater the risk of MI, heart failure, stroke, and kidney disease

**Cardiovascular Risk Factors**
- Smoking
- Dyslipidemia
- DM
- Age > 60 years
- Gender: Male, Female (Post Menopause)
- Family HX of premature CV disease in Women < age 65; Men < age 55

**Accurate BP Measurement**
- Make sure cuff is accurate and the right size
- Pt. seated quietly 5 minutes, feet on floor, arm supported at heart level
- 2 measurements
- Ambulatory monitoring indicated for evaluation of white coat HTN
- Home measurement - Accuracy? If home BP averages > 135/85 pts generally considered hypertensive

**Patient Evaluation**
- Assess lifestyle and CV risk factors
- Assess for identifiable causes of high BP
- Assess for presence of target organ damage and CVD

**Hypertension**
- Essential Hypertension
  - 90% of HTN
  - Unsure of actual cause
  - Fluid retention and Renin may be involved
- Hypertension with identifiable causes is called:
  - Secondary HTN

**HTN with identifiable cause is called Secondary HTN**
- Renal Causes: Chronic kidney & renovascular disease
- Endocrine causes: Thyroid and parathyroid disease, Cushings, primary aldosteronism, pheochromocytoma
- Pregnancy
- Drug related

**Secondary HTN**
- Drugs that can cause HTN
  - Antidepressants
  - Appetite suppressants
  - Cyclosporine
  - Erythropoetin
Mineral and corticosteroids
Nasal decongestants
Oral contraceptives
Sympathomimetics

22 Causes: Secondary HTN
Toxins, Social Drugs, Drugs of Abuse
Alcohol (excess, chronic use)
Amphetamines
Anabolic steroids
Caffeine
Cocaine
Lead

23 Diagnosis: History
General: age, race, sex, marital status
HPI: Last BP level if known, yrs of known HTN, TX (current & past), current meds, allergies
Past Med: MI, angina, HF; CVA, TIA’s; DM; asthma; renal, hepatic, thyroid diseases, rheumatic heart disease, surgeries, etc.
Social HX: Occupation, living situation, use of ETOH, tobacco, exercise, diet, life stress
Family HX: especially CVA, MI, HTN, angina DM

24 HX (cont): Review of Systems
HEENT: HA, Blurring/diplopic vision, vertigo, dizziness
Chest: Chest pain, exertional dyspnea, SOB, orthopnea, palpitations, claudication, syncope
GI: Ulcer HX, abd pain, N/V/diarrhea, melena, hematochezia
GU: Dysuria, urgency, frequency, hesitancy, nocturia, incontinence, loss of libido, loss of erection, prostate dx
Neuro: Seizures, pain, compression states, gait problems
Psych: Depression states, suicidal ideation, manic depressive

25 Physical Exam
HT, WT, BP (measure both arms, calculate BMI)
Eyes: Fundi: nicking, hemorrhage, exudate
CV: carotids for bruits, complete heart exam
Pulmonary: Inspir/expir breath sounds, rate wheezes, SOB
Abdomen: abd. and femoral bruits, liver span, masses
Legs/feet: pulses, edema, bruising, arterial/venous impairment, sensation

26 Lab Workup
CBC
Urinalysis
Serum potassium
Creatinine
Blood Glucose
Lipid Profile after 9-12 hr fast
EKG

27 Target Organ Damage/Clinical Cardiovascular Disease (TOD/CCD)
Heart Disease (LVH, Angina/prior MI, Prior CABG, Health failure)
● Stroke or TIA
● Chronic kidney disease
● Peripheral Arterial Disease
● Retinopathy

28  **JNC VII Guidelines**

- **Blood Pressure Systolic**  Diastolic
- Normal: below 120  below 80
- Prehtn  120-139  or 80-89

- **Hypertension**
  - Stage 1: 140-159 or 90-99
  - Stage 2: Above 160 or>100

29  **Recommended Follow up BP’s**

30  **JNC VII Risk Stratification**

- Determine blood pressure stage
- Determine risk group by major risk factors and TOD/CCD
- Determine treatment recommendations
- Determine goal blood pressure
- Refer to specific treatment recommendations

31  **JNC VII Risk Groups**

- **Risk Group A**
  - No major risk factors
  - No TOD/CCD

- **Risk Group B**
  - At least one major risk factor, not including DM
  - No TOD/CCD

- **Risk Group C**
  - TOD/CCD and/or DM, with or without other risk factors

32  **Goal of Treatment**

- The goal of prevention/management of HTN is to reduce CV and renal morbidity/mortality
- Most people with HTN will reach Diastolic BP goal once Systolic is met, primary focus should be on achieving Systolic BP goal
- Achieving targets that are <140/90 are associated with decrease in CV complications
- In patients with HTN and DM or renal disease the goal is <130/80

33  **Goal Blood Pressures**

34  **Lifestyle Modifications for HTN**

- Quit smoking
- Lose weight
- Restrict Na+
- Limit ETOH to 1-2 drinks/day
- Get 35-44 mins aerobic exercise/most days
- Maintain adequate intakes K+, Ca, Mg

35  **HTN Drug Treatment**

- Decision to begin drug treatment considers: the degree of BP elevation, presence of TOD, and presence of clinical CAD or other risk factors
- Drug RX clearly decreases cardiovascular morbidity/mortality
- Start if 6-12 mos of Lifestyle Mod has not lowered BP, or if BP.160/100, or if BP in
multi risk patient 140-159/90-99
- Start with low dose of long acting once daily drug, and titrate dose
- Low dose combinations may be useful

37 Initial Drug Treatment:
Uncomplicated HTN
- Diuretics - have been virtually unsurpassed in preventing CV complications of HTN. Should be the first drug of choice unless there is a compelling indication to choose another drug and there are lot of other drugs out there!!!

38 Thiazide Diuretics
- Hydrochlorothiazide, indapamide
- Prevent sodium from being reabsorbed in the kidney. As sodium excreted, it pulls water along with it. Also increases excretion of chloride, potassium and bicarb

39 Loop Diuretics
- Lasix, Bumex
- Most potent diuretics leading to greatest volume of diuresis
- Act primarily on the ascending loop of Henle to increase secretion of sodium, chloride and water
- Most common SE's are hypotension, hyponatremia and hypokalemia

40 Potassium Sparing Diuretics
- Dyazide
- Work on distal tubule of kidney to increase the excretion of sodium and water and decrease the excretion of potassium and hydrogen

41 Aldosterone Receptor Blockers
- Spironolactone
- A potassium sparing diuretic that is structurally similar to aldosterone and act as aldosterone antagonist
- Spironolactone competes with aldosterone for receptor sites and as a result sodium, chloride and water are excreted sodium, chloride and water are excreted and potassium retained
- Typically used for cirrhosis and hyperaldosteronism

42 Beta Blockers
(beta adrenergic antagonists)
- Atenolol, metoprolol, propranolol
- Block beta adrenergic receptor sites in the heart, muscle and conduction system decreased heart rate and reduced strength of contraction (negative chronotropy and negative ionotroph)
- May cause bradycardia, fluid retention, heart failure
- If non selective (can lead to bronchiolar constriction)

43 ACE Inhibitors
- Captopril, lisinopril, enalapril
- Act by interfering with the renin angiotensin aldosterone system preventing the conversion of Angiotensin I to angiotensin II

44 ACE’s
- Enhance the effects of diuretics. If they are used with potassium sparing diuretics or
potassium containing salt substitutes hyperkalemia may occur
- Less effective when used with NSAID’s
- SE’s: headache, cough, angioedema, throat tickle

### Calcium Channel Blockers
- Diltiazem, verapamil
- Act as vasodilators: Prevent passage of calcium ions across myocardial cell membrane & vascular smooth muscle cells
- Decrease force of contraction and reduces workload of heart
- Prevent arterioles from constricting; reduce systemic vascular resistance

### Calcium Channel Blockers (Dihydropyridines)
- Nifedipine (Procardia, Adalat), nicardipine, felodipine, amiodipine (Norvasc)
- Differ from Verapamil, Diltiazem by producing greater arteriolar vasodilation, but fewer cardiac actions; Minimal effects on sinoatrial & AV node conduction
- Used in moderate to severe HTN, with angina

### Alpha (Adrenergic Receptor) Blocker
- Prazosin (Minipres), Terazosin (Hytrin), Doxazosin (Cardura)
- Venodilators: Antagonize vasoconstrictor effects of neurogenic stimulation & catecholamines
- BP lowered more in upright position than supine; orthostatic hypotension common

### Centrally Acting Drugs
- Imidazolines (Clonidine or Catapres), Aldomet
- Central sympathetic drugs act at level of brainstem; interfere with tonic output of CNS vasomotor control centers
- Produce both arterial and venous vasodilation, & reduced cardiac responses to external stimuli

### Direct Vasodilators
- Hydralazine, minoxidil, nitroprusside
- Relax peripheral vascular smooth muscles causing vasodilation
- SE’s may include headache, rash, fainting

### Indications for Other Drugs
- DM type I (IDDM): Start with Ace inhibitor if proteinuria present
- Heart Failure: Start with ACE inhibitor or diuretic
- MI: Beta-Blocker after MI; ACE inhibitor for LV dysfunction after MI
- Isolated Systolic HTN (older patients); diuretics (preferred) and beta blockers or calcium antagonists

### All People with HTN
- People with any degree of HTN need to adopt lifestyle modifications
- Only those with the mildest of elevations, no CV risk factors and without DM or kidney disease should be treated with lifestyle modifications alone
- It is better to start with drug treatment and then later dc it then to wait around too long for those lifestyle modifications to take hold!

### Ischemic Heart Disease
- In patients with HTN and stable angina beta blocker, calcium channel blocker also used
- With unstable angina or MI, treat initially with BB’s and ACE’s, adding other drugs needed
- Intensive lipid management and ASA therapy

### Heart Failure
- Demonstratetable ventricular dysfunction, use ACE and BB’s
With symptomatic ventricular dysfunction and end stage heart disease, ACE’s, BB’s, ARB’s and aldosterone blockers are recommended with loop diuretics

**Diabetic Hypertension**
- Usually need combo of two or more drugs
- ACE or ARB’s favorably affect the progression of diabetic nephropathy and reduce albuminuria
- Thiazides, BBs, ACE’s, ARB’s, and CCB’s reduce CVD and stroke incidence

**Chronic Kidney Disease**
- Almost all these patients will have HTN and need aggressive rx
- ACE is and ARB’s both have favorable effects on disease progression
- With advance disease loop diuretics are usually needed

**HTN & Renal artery stenosis: Before and after angiography**

**Obesity and Metabolic syndrome**
- Risk for development of HTN and CVD
- Metabolic syndrome is the presence of three or more of the following conditions:
  - Waist circum>40”men, >35”women
  - Glucose intol (FBS>110)
  - BP>130/85
  - Triglycerides>150
  - Low HDL <40men <50 women

**Postural Hypotension**
- Decrease in standing SBP>10mm when associated with dizziness or fainting is more frequent in older patients and those taking diuretics
- Avoid volume depletion and rapid dose titration of drugs

**Hypertensive Crisis**
- Critical elevation in BP, diastolic exceeds 120mm Hg; with acute or ongoing end organ disease, constitutes “emergency”

**Causes of HTN Crisis**
- Abrupt increase BP in chronic HTN
- Renovascular HTN; parenchymal renal disease (chronic)
- Scleroderma other collagen vascular disease
- Cocaine, amphetamines, PCP
- Pre-eclampsia eclampsia
- Pheochromocytoma;Renin or aldosterone secreting tumor

**Cardiovascular Disease**

**Myocardial Ischemia**
- Narrowing of a major coronary artery by more than 50% impairs blood flow sufficiently to hamper cellular metabolism under conditions of increased myocardial demand
- Most common cause of myocardial ischemia is atherosclerosis

**Myocardial Ischemia**
- Supply is reduced by the following factors:
  - Hemodynamic factors, such as increased resistance in coronary vessels, hypotension, decreased blood flow
  - Cardiac factors such as decreases of diastolic filling time, increases in HR, or valvular incompetence
  - O2 in blood
  - Systemic disorders that reduce blood flow or the availability of oxygen (shock)

Continued
Demand is increased by the following factors:
- High systolic blood pressure
- Increased ventricular volume
- Increased thickness of the myocardium (left ventricular hypertrophy caused by increased systemic resistance, such as with aortic valve stenosis and hypertension
- Increased heart rate resulting from exercise, stress, hyperthyroidism, anemia, or hyperviscosity of the blood (polycythemia)

Continued
- Myocardial cells become ischemic within 10 seconds of coronary occlusion
- After several minutes the heart cells lose the ability to contract, thus hampering pump function and depriving the myocardium of a glucose source necessary for aerobic metabolism
- Anaerobic processes take over, and lactic acid accumulates
- Cardiac cells remain viable for app. 20 min under ischemic conditions.

Angina
- Stable-ischemic pain regularly produced by a given amount of exercise, episodes stable
- Cause is an imbalance b/w O2 supply and demand
- Unstable-pain occurs with less exertion, at rest, more prolonged
- Variant (Prinzmetal) reversible vasospasm

Stable Angina
- Caused by luminal narrowing and hardening of the arterial walls so that the affected vessels cannot dilate in response to increased myocardial demand associated with physical exertion or emotional stress

Symptomatology
- Dyspnea, precordial pressure, squeezing, radiation to anterior chest, neck, torso
- Aggravating factors-decreased exercise intolerance, stress, large meal
- Pattern-physical or emotional activity

Prognosis Determined by
- Level of Left Ventricular functioning
- Extent of CAD

History
- Risk factors
- Family History
- Smoking
- Hyperlipidemia
- GI Symptoms
- Dyspepsia
- GERD
- Heme positive stools

Comorbid conditions
- COPD
- Peripheral vascular disease
- BPH
- Orthostatic hypotension
Physical examination may disclose extra, rapid heart sounds (left ventricular gallop or S3), indicating impaired left ventricular function during the ischemic attack.

**Inspection...extra tips**
- Earlobe: Lichten’s sign
- Eyes
  - Xanthelasma-eyelids
  - Arcus senilis in persons <40
  - Corneal opacities
- Mouth
  - Palatal petechiae-associated

**Xanthelasma**

**Xanthomata: Telltale signs**

**Laboratory Studies**
- CBC, Chem panel
- TSH
- EKG
- Stress Test

**Pharmacologic TX**
- Correlate with pathophysiologic process
- Atherosclerotic process
- Decrease myocardial oxygen demand
- Tx with nitroglycerine, beta blockers, calcium channel blockers
- Control lipids

**Thrombus formation:**
- Treat with anti-platelet therapy
- ASA 325mg QD
- Abnormal vasomotor tone:
  - Treat with Calcium Channel Blocker

**Pharmacologic Tx**
- Individualize treatment
- Most cardiologists recommend sublingual nitroglycerine
- Beta blockers
  - Efficacious, but serious SE bradycardia, bronchial asthma, impotency, lethargy
  - Important to taper off

**Pharmacologic**
- Nitrates
  - Decrease myocardial O2 demand via vasodilation, decreasing preload, afterload
  - Forms: sublingual, oral, topical
  - SE: headache, flushing, blurred vision
  - Therapeutic trial- may be diagnostic

**Calcium Channel Blockers**
- Vasodilate like nitrates
Adalat, Procardia are potent vasodilators
Diltiazem and verapamil block AV and slow conduction
Decrease contractility—may increase CHF
SE leg edema, constipation

Unstable Angina
New onset, progressive or prolonged angina without prompt relief by nitro
Associated with ST and T wave changes, without enzyme elevations
Thrombi are seen on angiography 85%

Management
Need to be hospitalized
ASA
IV Heparin
IV Nitro
Beta blockers, CCB
Low molecular weight heparin
Diagnostic angiography

Percutaneous Transluminal Coronary Angioplasty
Coronary Artery Bypass Graft

CAD Leads to MI
Most common cause of death in pts >65
Clinical presentation may be altered by age
Prevalence increases with age in both men and women
Increased incidence in women after menopause
Atypical presentation—syncope, stroke, confusion

CAD Leads to MI
After age 70 M:F is almost equal

Levine’s Sign
Heart Attack
Continued
Continued

Pathophysiology
Cellular injury
Cardiac cells withstand ischemic conditions for about 20 minutes before cellular death takes place
After only 30 to 60 seconds of hypoxia, ECG changes are visible
After 8 to 10 seconds of decreased blood flow, the affected myocardium becomes cyanotic and cooler
Myocardial oxygen reserves are used very quickly after complete cessation of coronary flow

Continued
Glycogen stores decrease as anaerobic metabolism begins
Hydrogen ions and lactic acid accumulate
Myocardial tissues have poor buffering capabilities and very sensitive to low pH levels so further compromise is evident
Acidosis can make the heart more vulnerable to the damaging effects of lysosomal enzymes and suppress impulse conduction and contractile function, leading to heart failure

Continued
Oxygen deprivation is also accompanied by electrolyte disturbances, loss of K, Ca, Mg
Myocardial cells lose contractility and diminishes the pumping ability of the heart
Arterial occlusion causes the myocardial cells to release catecholamines, predisposing the person to serious imbalances of sympathetic and parasympathetic function, irregular heartbeats (dysrrhythmia), and heart failure

Continued
Myocardial infarction can result in functional changes of cardiac tissue
- Decreased cardiac contractility with abnormal wall motion
- Altered left ventricular compliance
- Increased left ventricular end diastolic pressure
- SA node malfunction
- Life threatening dysrhythmias and heart failure follow MI

Clinical Manifestations
- Sudden severe chest pain
- Heavy crushing, radiation to neck, jaw, back, shoulder or left arm common
- Not relieved by nitrates
- Indigestion, N/V
- Catecholamine release producing diaphoresis and peripheral vasoconstriction skin becomes cool and clammy
- Fever can develop first 24 hours and persist for 1 week

Continued
- Abnormal extra heart sounds (S3,S4), reflect left ventricular dysfunction
- Inflammation can cause pericardial friction rub, along with murmurs
- Leukocytes increased, elevated sedimentation rate, both indicate inflammation
- Blood sugar elevated, glucose tolerance remains abnormal for couple weeks

Labs
- EKG
- Cardiac Enzymes
- Cardiac Troponin
- C-reactive Protein
- BNP

Evaluation and Treatment
- Oxygen
- ASA 81-325mg Qday
- Thrombolytic therapy (TPA, streptokinase) dissolve clot, vasodilate
- Beta-blockers/ACE inhibitors
- Improved survival
- Underused

Complications
- Dysrhythmias disturbance in cardiac rhythm most common complication of acute MI affects more than 90% of individuals
- May originate from the atria, ventricles, nodal regions, or conduction tissues
- Pericarditis- anterior chest pain worsens with respiratory effort (tx: corticosteroids)
- Organic brain syndrome (blood flow to brain impaired)

Cardiomyopathies
- Causes:
  - Secondary to infectious disease
  - Exposure to toxins
  - Systemic connective tissue disease
Nutritional deficiencies
Most are idiopathic

Continued

Dilated Cardiomyopathy
- Ventricular dilation and grossly impaired systolic function, leading to heart failure
- Diminished contractility
- Diminished systolic performance of the heart
- Decreased ejection fraction
- Increased end-diastolic and residual volume

S&S
- Dyspnea
- Fatigue
- Pulmonary Congestion
- Palpitations associated with dysrhythmias may cause syncope
- Increased blood pressure
- Extra heart sounds, murmurs present

Treatment
- Salt restriction
- Diuretics
- Digoxin
- Anticoagulants
- Corticosteroids
- Vasodilators

Hypertrophic Cardiomyopathy

Clinical Manifestations
- Angina
- Syncope
- Palpitations
- Left heart failure
- TX
  - Beta blockers (reduce left vent. Stiffness and reduce heart rate to permit with ventricular filling)

Echocardiogram

Chronic Venous Insufficiency
Continued

Venous Insufficiency
2.

Venous Insufficiency
2.

Arterial Insufficiency

Continued
1.

Continued
2. Pulses