Management of Ischemic Heart Disease (IHD)

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442 course

Introduction

• Coronary heart disease (CHD) is the most common form of heart disease

Most common cause of death

• An estimated 330,000 people have a myocardial infarct each year

• Approximately 1.3 million people have angina each year

Here in SA we are 10 years younger in terms of incidence of ischemic heart disease. In western society it’s 65 years old. In SA society it’s 55 years old. Due to emergence of sedentary life style, smoking, DM.

Myocardial Ischemia

Symptoms develop here

• Disease of the coronary arteries is almost always due to atheroma and its complications – particularly thrombosis

• Results when there is an imbalance between myocardial oxygen supply and demand

• Most occurs because of atherosclerotic plaque with in one or more coronary arteries

• Limits normal rise in coronary blood flow in response to increase in myocardial oxygen demand

Atheroma: low density lipoprotein engulfed with microcytes, lymphocytes Deposited in the wall and forms what’s known as fatty streaks With progression its causes lumen narrowing
Defining myocardial infarction: ischemia means imbalance between O2 demand and blood supply

Whenever you have reduction of blood flow to the heart and with the patient exercising, ischemia and chest pain will develop. This happens with every organ, ischemia of the legs > claudication, ischemia of the brain > stroke.

Oxygen Carrying Capacity

- The oxygen carrying capacity relates to the content of hemoglobin and systemic oxygenation
- When atherosclerotic disease is present, the artery lumen is narrowed and vasoconstriction is impaired
- Coronary blood flow cannot increase in the face of increased demands and ischemia may result

Angina: IMPORTANT
1) chest discomfort: heaviness, tightness and rarely described as pain and its diffuse (visceral) difficult to localized
2) comes with exertion and/or emotional distress (following hearing bad news, loss of loved ones, loss of money
3) relieved by stress

If all three components present > **stable** angina
If only 2 > **atypical** angina
If only 1 > **myocardial** pain
Stable angina: When the patient gets pain at certain known level of exertion for 1 year e.g. someone lives in the 1st floor, he climbs the 1st floor normally but when he climb the 2nd floor he gets the pain

Unstable angina: When the patient has a new onset, symptoms at rest or progression in stable angina e.g. the patient starts feeling the pain with climbing the 1st floor

Note that 30% of adult visits to the ER is due to chest pain While pediatrics it’s due to cough and diarrhea

Stable angina:
- Character of pain: Exertional pain
- Relievers: Responds to GTN
- Enzymes: Normal
- ECG: Often normal

Unstable angina:
- Character of pain: Rest pain
- Relievers: No GTN effect
- Enzymes: Elevated
- ECG: Often ST depression

Acute Coronary Syndromes:

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<td>ST segment elevation</td>
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Angina:

- When ischemia results it is frequently accompanied by chest discomfort: Angina Pectoris

- In the majority of patients with angina, development of myocardial ischemia results from a combination of fixed and vasospastic stenosis
Chronic Stable Angina

- May develop sudden increase in frequency and duration of ischemic episodes occurring at lower workloads than previously or even at rest
- Known as unstable angina: up to 70% patients sustain MI over the ensuing 3 months

Angina: cont

- Patients with mild obstruction coronary lesions can also experience unstable angina
- >90% of Acute MI result from an acute thrombus obstructing a coronary artery with resultant prolonged ischemia and tissue necrosis

Treatment of Angina

- Treatment of Chronic Angina is directed at minimizing myocardial oxygen demand and increasing coronary flow
- Where as in the acute syndromes of unstable angina or MI primary therapy is also directed against platelet aggregation and thrombosis

Epidemiology

- Modifiable Factors: hyperlipidemia- ^ LDL (<130 normal) or low HDL (>60 normal), Hypertension, cigarette smoking and diabetes, obesity, BMI of >30
- Non-Modifiable Factors: advanced age, male sex, family medical history: male <55 y/o, female <65 y/o
- Other: sedentary lifestyle and stressful emotional stress  Very important factor we should not neglect
### Quality

- Tightness, squeezing, heaviness, pressure, burning, indigestion or aching sensation
- It is rarely “PAIN”
- Never: sharp, stabbing, prickly, spasmodic, or pleuritic
- Lasts a few seconds < 10 minutes
- Relieved by NTG s/l
- Levine Sign: clench fist to sternum

### Ischemic Heart Disease

- Imbalance between Myocardial oxygen supply and demand = Myocardial hypoxia and accumulation of waste metabolites due to atherosclerotic disease of coronary arteries

### Signs & Symptoms accompany Angina

- Dyspnea, nausea, diaphoresis resolve quickly after cessation of angina
- Angina is a diffuse sensation rather than discrete

### Stable Angina

- Stable Angina: chronic pattern of transient angina pectoris precipitated by physical activity or emotional upset, relieved by rest with in few minutes
- Temporary depression of ST segment with no permanent myocardial damage
Angina Pectoris

- Angina Pectoris: uncomfortable sensation in the chest or neighboring anatomic structures produced by myocardial ischemia

Variant Angina

- Typical anginal discomfort usually at rest
- Develops due to coronary artery spasm rather than increase myocardial oxygen demand
- Transient shifts of ST segment – ST elevation

Unstable Angina

- Increased frequency and duration of Angina episodes, produced by less exertion or at rest = high frequency of myocardial infarction if not treated

Silent Ischemia

- Asymptomatic episodes of myocardial ischemia
- Detected by electrocardiogram and laboratory studies

These are asymptomatic repeated MI frequently with patients with long standing DM due to autonomic neuropathy
Myocardial Infarction

- Region of myocardial necrosis due to prolonged cessation of blood supply
- And chronic inflammatory process
- Results from acute thrombus at side of coronary atherosclerotic stenosis
- May be first clinical manifestation of ischemic heart disease or history of Angina Pectoris

Precipitants

- Exertion: walking, climbing stairs, vigorous work using arms, sexual activity
- Vasoconstriction: extremities, increased systemic vascular resistance, increased in myocardial wall tension and oxygen requirements
- Myocardial Ischemia displays a circadian rhythm threshold for Angina it is lower in morning hours.

Physical Examination

- Arcus senilis, xanthomas, funduscopic exam: AV nicking, exudates
- Signs and symptoms: hyperthyroidism with increased myocardial oxygen demand, hypertension, palpitations
- Auscultate carotid and peripheral arteries and abdomen: aortic aneurysm
- Cardiac: S4 common in CAD, increased heart rate, increased blood pressure

Ischemia

- Myocardial ischemia may result in papillary muscle regurgitation
- Ischemic induced left ventricular wall motion abnormalities may be detected as an abnormal precordial bulge on chest palpation
- A transient S3 gallop and pulmonary rales = ischemic induced left ventricular dysfunction

50% of patients have carotid artery disease
90% of patients who have peripheral disease have heart disease not yet manifested
Diagnostic Tests

• **Blood tests** include serum lipids, fasting blood sugar, Hematocrit, thyroid (anemias and hyperthyroidism can exacerbate myocardial ischemia)

• **Resting Electrocardiogram**: CAD patients have normal baseline ECGs
  — pathologic Q waves = previous infarction
  — minor ST and T waves abnormalities not specific for CAD

Electrocardiogram

• Electrocardiogram: is useful in diagnosis during cc: chest pain

• When ischemia results in transient horizontal or downsloping ST segments or T wave inversions which normalize after pain resolution

• ST elevation suggest severe transmural ischemia or coronary artery spasm which is less often

Exercise Stress Test

• Used to **confirm diagnosis** of angina

• Terminate if hypotension, high grade ventricular disrhythmias, 3 mm ST segment depression develop

• (+): reproduction of chest pain, ST depression

• Severe: chest pain, ST changes in 1st 3 minutes, >3 mm ST depression, persistent > 5 minutes after exercise stopped

• Low systolic BP, multifocal ventricular ectopy or V-tach, ST changes, poor duration of exercise (<2 minutes) due to cardiopulmonary limitations

Other Diagnostic Tests

• Radionuclide studies

• Myocardial perfusion scintigraphy

• Exercise radionuclide ventriculography

• Echocardiography

• Ambulatory ECG monitoring

• **Coronary arteriography**

A patient comes to the clinic and we exercise / stress the patient to induce ischemia, but it's important to check patient's fitness and ability to perform exercise. We observe for St segment changes, if it changes we label the test **+ve**

If we want to stress an old patient we give Dobutamine (physiological stress)
Management Goals to reduce Anginal Symptoms

• Prevent complications – myocardial infarction, and to prolong life
• No smoking, lower weight, control hypertension and diabetes
• Patients with CAD – LDL cholesterol should achieve lower levels (<100)
• HMG-CoA reductase inhibitors are effective

Management goal (very imp question in every case, It doesn’t mean treatment only, so:
1) say you will treat as an outpatient or inpatient? In this case admit the patient
2) we start medical therapy. Primary prevention medications
3) thorough clinical investigations like echo, ecg, blood work, then start medications accordingly

Pharmacologic Therapy

• Therapy is aimed in restoring balance between myocardial oxygen supply and demand

• Useful Agents: nitrates, beta-blockers and calcium channel blockers

Nitrates

• Reduce myocardial oxygen demand
• Relax vascular smooth muscle
• Reduces venous return to heart
• Arteriolar dilators decrease resistance against which left ventricle contracts and reduces wall tension and oxygen demand

By activation of cyclic GMP
Nitrates: cont

- Dilate coronary arteries with augmentation of coronary blood flow
- Side effects: generalized warmth, transient throbbing headache, or lightheadedness, hypotension
- ER if no relief after X2 nitro’s: unstable angina or MI

Problems with Nitrates

- Drug tolerance
- Continued administration of drug will decrease effectiveness
- Prevented by allowing 8 – 10 hours nitrate free interval each day.
- Elderly/inactive patients: long acting nitrates for chronic antianginal therapy is recommended
- Physical active patients: additional drugs are required

Beta Blockers

- Prevent effort induced angina
- Decrease mortality after myocardial infarction
- Reduce Myocardial oxygen demand by slowing heart rate, force of ventricular contraction and decrease blood pressure

Beta -1

- Block myocardial receptors with less effect on bronchial and vascular smooth muscle-patients with asthma, intermittent claudication

They act on B1 receptor and therefore decrease contractility and slow down heart rate, basically relax the heart
Induce coronary vasodilation > improve coronary blood flow
**Beta-Agonist blockers**

- With partial B-agonist activity:
- Intrinsic sympathomimetic activity (ISA) have mild direct stimulation of the beta receptor while blocking receptor against circulating catecholamines
- Agents with ISA are less desirable in patients with angina because higher heart rates during their use may exacerbate angina
- not reduce mortality after AMI

**Beta-blockers**

- Duration of beta-blockers depends on lipid solubility
- Accounts for different dosage schedules

**Contraindications**

- Symptomatic CHF, history of bronchospasm, bradycardia or AV block, peripheral vascular disease with s/s of claudication

**Side Effects**

- Bronchospasm (RAD), CHF, depression, sexual dysfunction, AV block, exacerbation of claudication, potential masking of hypoglycemia in IDDM patients
Calcium Channel Blockers

- Anti-anginal agents prevent angina
- Helpful: episodes of coronary vasospasm
- Decreases myocardial oxygen requirements and increase myocardial oxygen supply
- Potent arterial vasodilators: decrease systemic vascular resistance, blood pressure, left ventricular wall stress with decrease myocardial oxygen consumption

Calcium Channel Blockers

- Secondary agents in management of stable angina
- Are prescribed only after beta blockers and nitrate therapy has been considered
- Potential to adversely decrease left ventricular contractility
- Used cautiously in patients with left ventricular dysfunction

Drug Selection

- Chronic Stable Angina: beta blocker and long acting nitrate or calcium channel blocker (not verapamil: bradycardia) or both.
- If contraindication to BB a CCB is recommended (bronchospasm, IDDM, or claudication) any of CCB approved for angina are appropriate.

Drugs

- Verapamil and Cardizem is preferred because of effect on slowing heart rate
- Patients with resting bradycardia or AV block, a dihydropyridine calcium blocker is better choice
- Patients with CHF: nitrates preferred amiopidine should be added if additional therapy is needed

2nd or 3rd line not recommended to every patient
Negative ionotropic effect, thus decreases contractility
Vasodilation effect
If patient is not controlled with BB give CCB still not controlled? Nitrates
Drugs

• Primary coronary vasospasm: no treatment with beta blockers, it could increase coronary constriction
• Nitrates and CCB are preferred
• Concomitant hypertension: BB or CCB are useful in treatment
• Ischemic Heart Disease & Atrial Fibrillation: treatment with BB, verapamil or Cardizem can slow ventricular rate

Combination Therapy

• If patients do not respond to initial antianginal therapy – a drug dosage increase is recommended unless side effects occur.

• Combination therapy: successful use of lower dosages of each agent while minimizing individual drug side effects

Combination Therapy Include:

• Nitrate and beta blocker
• Nitrate and verapamil or cardizem for similar reasons
• Long acting dihydropyridine calcium channel blocker and beta blocker
• A dihydropyridine and nitrate is often not tolerated without concomitant beta blockade because of marked vasodilatation with resultant head ache and increased heart rate

Combinations

• Beta blockers should be combined only very cautiously with verapamil or cardizem because of potential of excessive bradycardia or CHF in patients with left ventricular dysfunction
Other methods

- Patients with 1 – 2 vessel disease with normal left ventricular function are referred for catheter based procedures.
- Patients with 2 and 3 vessel disease with widespread ischemia, left ventricular dysfunction or DM and those with lesions are not amendable to catheterization based procedures and are referred for CABG.

PCI: primary coronary intervention

PCI vs CABG

Metal stents
Acute Coronary Syndromes

- Unstable Angina
- Non-ST-Segment Elevation MI (NSTEMI)
- ST-Segment Elevation MI (STEMI)

Similar pathophysiology
Similar presentation and early management rules
STEMI requires evaluation for acute reperfusion intervention

Diagnosis is simply by cardiac markers and ECG

- ST segment elevation > STEMI > immediately cath lab and PCI (Time is muscle) less than <90 minutes
- ST depression > chest pain > unstable angina
  +ve cardiac markers > NSTEMI
  -ve cardiac markers > unstable angina

Both treated the same but not necessarily within 90 min but within 24 hrs of admission
Diagnosis of Acute MI
STEMI / NSTEMI

- At least 2 of the following
  - Ischemic symptoms
  - Diagnostic ECG changes
  - Serum cardiac marker elevations

Diagnosis of Angina

- Typical angina—All three of the following
  - Subternal chest discomfort
  - Onset with exertion or emotional stress
  - Relief with rest or nitroglycerin

- Atypical angina
  - 2 of the above criteria

- Noncardiac chest pain
  - 1 of the above

Diagnosis of Unstable Angina

- Patients with typical angina - An episode of angina
  - Increased in severity or duration
  - Has onset at rest or at a low level of exertion
  - Unrelieved by the amount of nitroglycerin or rest that had previously relieved the pain

- Patients not known to have typical angina
  - First episode with usual activity or at rest within the previous two weeks
  - Prolonged pain at rest

Unstable Angina

NSTEMI
- Non occlusive thrombus
- Non specific ECG
- Normal cardiac enzymes
- Occluding thrombus sufficient to cause tissue damage & mild myocardial necrosis
- ST depression +/- T wave inversion on ECG
- Elevated cardiac enzymes

STEMI
- Complete thrombus occlusion
- ST elevations on ECG or new LBBB
- Elevated cardiac enzymes
- More severe symptoms
Acute Management

- Initial evaluation & stabilization
- Efficient risk stratification
- Focused cardiac care

Evaluation

- Efficient & direct history
- Initiate stabilization interventions

Plan for moving rapidly to indicated cardiac care

Chest pain suggestive of ischemia

Immediate assessment within 10 Minutes

Initial labs and tests
- 12 lead ECG
- Obtain initial cardiac enzymes
- electrolytes, cbc, lipids, bun/cr, glucose, coags
- CXR

Emergent care
- IV access
- Cardiac monitoring
- Oxygen
- Aspirin
- Nitrates

History & Physical
- Establish diagnosis
- Read ECG
- Identify complications
- Assess for reperfusion

Focused History

- Aid in diagnosis and rule out other causes
  - Palliative/Provocative factors
  - Quality of discomfort
  - Radiation
  - Symptoms associated with discomfort
  - Cardiac risk factors
  - Past medical history - especially cardiac

Reperfusion questions
- Timing of presentation
- ECG c/w STEMI
- Contraindication to fibrinolysis
- Degree of STEMI risk

Very important any Patient with MI
Aspirin
Statin
BB

3/13/2020

And treat accordingly
STEMI > cath lab
NSTEMI > icu, stabilize and next day send to cath lab
Targeted Physical Examination

- Examination
  - Vitals
  - Cardiovascular system
  - Respiratory system
  - Abdomen
  - Neurological status

- Recognize factors that increase risk
  - Hypotension
  - Tachycardia
  - Pulmonary rales, JVD, pulmonary edema
  - New murmurs/heart sounds
  - Diminished peripheral pulses
  - Signs of stroke

ECG assessment

- ST Elevation or new LBBB
  - STEMI

- ST Depression or dynamic T wave inversions
  - NSTEMI

- Non-specific ECG
  - Unstable Angina

ECG changes with MI

- Normal or non-diagnostic ECG

Diffuse ST segment elevation due to early repolarization, you see here a smily face
Unlike MI sad face
Unstable angina

If a patient presented with chest pain and the cardiac markers were negative don’t discharge and repeat after 6 hours

Peak of troponin 12 hrs and stays for 2 weeks
Peak for CK-mb 34 and stays for 3 to 5 days
Cardiac Care Goals

- Decrease amount of myocardial necrosis
- Preserve LV function
- Prevent major adverse cardiac events
- Treat life threatening complications

STEMI cardiac care

- **STEP 1**: Assessment
  - Time since onset of symptoms
    - 90 min for PCI / 12 hours for fibrinolysis
  - Is this high risk STEMI?
    - KILLIP classification
    - If higher risk may manage with more invasive rx
  - Determine if fibrinolysis candidate
    - Meets criteria with no contraindications
  - Determine if PCI candidate
    - Based on availability and time to balloon rx

Risk Stratification

- Assess for reperfusion
- Select & implement reperfusion therapy
- Directed medical therapy

STEMI Patient?

UA or NSTEMI

- Evaluate for Invasive vs. conservative treatment
- Directed medical therapy

Cardiac markers

Based on initial evaluation, ECG, and cardiac markers

77 78 79 80
Fibrinolysis indications

• ST segment elevation >1mm in two contiguous leads
• New LBBB
• Symptoms consistent with ischemia
• Symptom onset less than 12 hrs prior to presentation

Absolute contraindications for fibrinolysis therapy in patients with acute STEMI

• Any prior ICH
• Known structural cerebral vascular lesion (e.g., AVM)
• Known malignant intracranial neoplasm (primary or metastatic)  
  Risk of bleeding
• Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
• Suspected aortic dissection
• Active bleeding or bleeding diathesis (excluding menses)
• Significant closed-head or facial trauma within 3 months

If you’re in a hospital with no cath lab, transfer your patient. If not possible give fibrinolysis it helps in degradation of platelet plug given in the 1st hour of chest pain
And within less than 12 hours
After that its not recommended

STEMI cardiac care

• **STEP 2:** Determine preferred reperfusion strategy

  **Fibrinolysis** preferred if:
  - <3 hours from onset
  - PCI not available/delayed
  - Door to balloon > 90min
  - Door to balloon minus door to needle > 1hr
  - Door to needle goal <30min
  - No contraindications

  **PCI** preferred if:
  - PCI available
  - Door to balloon < 90min
  - Door to balloon minus door to needle < 1hr
  - Fibrinolysis contraindications
  - Late Presentation > 3 hr
  - High risk STEMI
  - Killip 3 or higher
  - STEMI dx in doubt

  **Medical Therapy**

  **MONA + BAH**

  • **Morphine** (class I, level C)
    - Analgesia
    - Reduce pain/anxiety—decrease sympathetic tone, systemic vascular resistance and oxygen demand
    - Careful with hypotension, hypovolemia, respiratory depression
  
  • **Oxygen** (2-4 liters/minute) (class I, level C)
    - Up to 70% of ACS patient demonstrate hypoxemia
    - May limit ischemic myocardial damage by increasing oxygen delivery/reduce ST elevation
• **Nitroglycerin** (class I, level B)
  - Analgesia—titrate infusion to keep patient pain free
  - Dilates coronary vessels—increase blood flow
  - Reduces systemic vascular resistance and preload
  - Careful with recent ED meds, hypotension, bradycardia, tachycardia, RV infarction

• **Aspirin** (160-325mg chewed & swallowed) (class I, level A)
  - Irreversible inhibition of platelet aggregation
  - Stabilize plaque and arrest thrombus
  - Reduce mortality in patients with STEMI
  - Careful with active PUD, hypersensitivity, bleeding disorders

• **Beta-Blockers** (class I, level A)
  - 14% reduction in mortality risk at 7 days at 23% long term mortality reduction in STEMI
  - Approximate 13% reduction in risk of progression to MI in patients with threatening or evolving MI symptoms
  - Be aware of contraindications (CHF, Heart block, Hypotension)
  - Reassess for therapy as contraindications resolve

• **ACE-Inhibitors / ARB** (class I, level A)
  - Start in patients with anterior MI, pulmonary congestion, LVEF < 40% in absence of contraindication/hypotension
  - Start in first 24 hours
  - ARB as substitute for patients unable to use ACE-I

• **Heparin** (class I, level C to class IIa, level C)
  - LMWH or UFH (max 4000u bolus, 1000u/hr)
  - Indirect inhibitor of thrombin
  - Less supporting evidence of benefit in era of reperfusion
  - Adjunct to surgical revascularization and thrombolytic / PCI reperfusion
  - 24-48 hours of treatment
  - Coordinate with PCI team (UFH preferred)
  - Used in combo with aspirin and/or other platelet inhibitors
  - Changing from one to the other not recommended

• **Clopidodrel** (class I, level B)
  - Irreversible inhibition of platelet aggregation
  - Used in support of cath / PCI intervention or if unable to take aspirin
  - 3 to 12 month duration depending on scenario

• **Glycoprotein IIb/IIIa inhibitors** (Class IIa, level B)
  - Inhibition of platelet aggregation at final common pathway
  - In support of PCI intervention as early as possible prior to PCI

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For diabetics or EF <40

Combine it with aspirin for the first year then continue on aspirin alone

Additional medication therapy

**Clopidodrel** (class I, level B)
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**Glycoprotein IIb/IIIa inhibitors**
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Indicated for all patient with ACS

Only for acute management until discharge or after 8 weeks, whatever comes first
Additional medication therapy

- **Aldosterone blockers** (class I, level A)
  - Post-STEMI patients
    - no significant renal failure (cr < 2.5 men or 2.0 for women)
    - No hyperkalemia > 5.0
    - LVEF < 40%
    - Symptomatic CHF or DM

STEMI care CCU

- Monitor for complications:
  - recurrent ischemia, cardiogenic shock, ICH, arrhythmias
- Review guidelines for specific management of complications & other specific clinical scenarios
  - PCI after fibrinolysis, emergent CABG, etc...
- Decision making for risk stratification at hospital discharge and/or need for CABG

Unstable angina/NSTEMI cardiac care

- Evaluate for conservative vs. invasive therapy based upon:
  - Risk of actual ACS
  - TIMI risk score
  - ACS risk categories per AHA guidelines

Diagram:

- Low risk
- Intermediate risk
- High risk

Conservative therapy

Invasive therapy

Chest Pain Center
Invasive therapy option
UA/NSTEMI

- Coronary angiography and revascularization within 12 to 48 hours after presentation to ED
- For high risk ACS (class I, level A)
- MONA + BAH (UFH)
- Clopidogrel
  - 20% reduction death/MI/Stroke – CURE trial
  - 1 month minimum duration and possibly up to 9 months
- Glycoprotein IIb/IIIa inhibitors

Conservative Therapy for UA/NSTEMI

- Early revascularization or PCI no planned
- MONA + BAH (LMW or UFH)
- Clopidogrel
- Glycoprotein IIb/IIIa inhibitors
  - Only in certain circumstances (planning PCI, elevated TnI/T)
- Surveillance in hospital
  - Serial ECGs
  - Serial Markers

Secondary Prevention

- Disease
  - HTN, DM, HLP
- Behavioral
  - Smoking, diet, physical activity, weight
- Cognitive
  - Education, cardiac rehab program

Secondary Prevention
disease management

- Blood Pressure
  - Goals < 140/90 or <130/80 in DM /CKD
  - Maximize use of beta-blockers & ACE-I
- Lipids
  - LDL < 100 (70); TG < 200
  - Maximize use of statins; consider fibrates/niacin first line for TG>500; consider omega-3 fatty acids
- Diabetes
  - A1c < 7%
Secondary prevention behavioral intervention

- Smoking cessation
  - Cessation-class, meds, counseling

- Physical Activity
  - Goal 30 - 60 minutes daily
  - Risk assessment prior to initiation

- Diet
  - DASH diet, fiber, omega-3 fatty acids
  - <7% total calories from saturated fats

Thank You