Cardiovascular
01/06/15

Heart disease: more prevalent on the southeast coast, this is very similar to the distribution of diabetes. CV disease is the leading cause of death in men and women in the US.

- Major risk factors: Smoking status, BMI, physical activity, healthy diet, total cholesterol, BP and fasting plasma glucose. What is the #1 thing you can address that can have the greatest impact? Diet!

Healthy diet (4/5 of the following):

- Fruits and veggies >4.5 cups/day
- Fish more than two 3.5 oz servings per week (http://www.seafoodwatch.org)
- Fiber rich whole grains >1.1g of fiber/10g carbohydrate at 3 servings per day
- Sodium <1500 mg/d
- Sugar sweetened beverages <36 oz per week

Prevention is key!

- Primordial: I have no risk factors, how do I maintain my lifestyle to not have risk in the future? (No risk yet)
- Primary: action to preclude the development of a disease
- Secondary: what you do when you have early symptoms of a disease to preclude the development of possible irreparable medical conditions.
- Tertiary: I had a heart attack, let's make sure I don't have another one

CV disease is higher in men but there is no difference in race.
Overall, decreasing risk factors decreases risk of CV disease.
One study found significant risk reduction in patients that saw naturopaths that provided counseling about nutritional and physical activity compared to standard medical care. (Seely, D et al. CMAJ 2013 Jun11; 185(9): E409-16).

Case: 40 old woman with elevated high sensitivity CRP. She was physically active, eating a diet with no soy/gluten/dairy and she meditated and did yoga everyday. When calculating her risk she had a 2% risk of a cardiac event in the next 10 years. If we made all the changes possible her risk would only drop to 1%. After discussing that with the patient she was relieved.

Risk assessment:
ASCVD risk calculator: this is what we use for asymptomatic patients (healthy patients). This is primary prevention. Once the patient has a disease this can't be used. Note that LDL is not part of this risk calculator.

Heart anatomy: Group 1
The flow of life: Right atrium receives blood from both the SVC and IVC→ right ventricle → pulmonary artery→ lungs→ pulmonary veins→ left atrium→ left ventricle→ aorta→ rest of the body

Receptors: Aorta: chemo and baroreceptors; chemoreceptors sense PCO2/decreases in pH, baroreceptors sense changes in arterial pressure.
Tissue layers: myocardium, pericardium (actually can survive without the pericardium)
Arteries: Coronary arteries fill during diastole (with atherosclerosis there is angina)

Cardiac Cycle: Group 2
P wave: corresponds to atrial depolarization
QRS wave: ventricular depolarization
T wave: Ventricular repolarization
During contraction atrial pressure is higher than ventricular pressure. With aortic opening there is an ejection of blood and aortic pressure is below ventricular pressure.
Vagus nerve: decrease in parasympathetic increases sympathetic output, which increases contractility, increases SV and increases output; communicates with SA node communicates with AV node to the bundle of His to the purkinje fibers.

Physiology: Group 3
CO=SV x HR
Pre load: amount of volume in ventricle at end of diastole; equal to end diastolic volume
After load: aortic pressure under normal conditions
Resistance=change in pressure/change Q (rate of flow)
Starling: ability to adapt to changes in volume, increased EDV leads to increase in CO
Ejection fraction= SV/EDV x 100; normally it is >60%. With what condition can this be normal? Left sided ventricular hypertrophy. Ejection fraction is the most prognostic test for CHF.

Cardiac exam: Group 4
1. Vitals
2. BP sitting and reclining
3. Inspection: skin/pallor, cavity, PMI, looking at JVP specifically of right internal JVP while reclined at 45 degrees (<5 is normal). The RJVP goes directly to the right atrium, which is why it is a better determinant.
4. Palpation: for apex, look for thrills (murmurs)
5. Percussion: left axillary line, intercostal spaces, measure apex for possible hypertrophy
6. Auscultation: 5 areas, looking for split S2, erbs point, mitral, bruit check, pulse check (vascular exam). On deep inspiration there can be a split S2 due to pressure change. If heard without inspiration it can be pathologic.
7. Special maneuvers/tests: valsalva, (MVP), friction rub (pericarditis), hair on toes, clubbing, etc.

Peripheral Vasculature: Group 5
Smooth muscle in arteries
Valves to prevent backflow
Receptors in aorta and carotid arteries
Angiotensin-renin system: angiotensin II increases aldosterone, leads to arterial vasoconstriction, increases sympathetic activity, etc. juxtaglomerular cells sense low sodium increasing renin production progressing the creation of angiotensin.

PAD symptoms: intermittent claudication in the calves/quads/glutes, loss of hair on toes, scaly skin.

Diagnostic testing/Risk Assessment: Group 6

Tools: There are multiple tools that you can use to assess risk including the ASVCD risk calculator but keep in mind that different calculators calculate different risk-for example one may look at risk of an MI in ten years, another may look at risk of atherosclerosis over a lifetime, etc. Coronary calcium score does not look at the individual, you need to think critically.

Acute testing for MI: Troponin, myoglobin, creatine kinase (re-infarction), ECG

Acute testing for DVT: Homan's? Ultrasound depending on Well's score

Lab tests options: VAP panel, cholesterol, HDL, LDL, TG, BNP, hs-CRP, Apo B; keep in mind some of these are more indicated than others. For example hs-CRP is not very specific and is more indicated in women. Also note that these labs aren't indicated unless there is risk.

Imaging for risk: CIMT

CAD and Atherosclerosis:

Ischemia: lack of oxygen

Angina: symptom of chest pain most likely due to ischemia

3 cardiac causes of chest pain: angina, MI, pericarditis

3 non-cardiac causes of chest pain: muscle pain, Costochondritis, Herpes zoster, GERD

2 cardiac conditions with symptom of SOB: pericarditis, CHF, ischemia, GERD

CAD:

- Risk factors: similar to those for CV events, family history event <55 in men or <65 in women, elevated Homocysteine (treated in childhood has been helpful but not in adults), chlamydia exposure, Type A personality, etc.
- "Fatty meal + sex = killer of older men"-Dr. Smith
- Pathogenesis: fatty streaks, plaque formation. A calcified plaque is stable, the problem is soft plaque ("it's like a scar versus a pimple.") However, soft plaques aren't distinguishable on imaging.
- Angina: the amount of pain does not correspond to the level of ischemia.
    - Stable angina: It can be stable if it lasts less than 20 minutes and is provoked by exertion and relieved by rest.
    - Prinzmetal angina: This is a spasm, no clot causing a blockage; it usually happens at night.
    - Unstable angina: symptoms occur when walking less than 2 blocks or walking 1 flight of stairs. They are unresponsive to rest. MI and death in 30% of patients within 3 months.
- Making a diagnosis: you can assume someone has CAD if they have an MI. Other tests include ECG, stress test, CT/EBCT, MRI. The stress test is first line testing-it is non invasive. However, the gold standard is angiography since doctors can look and treat simultaneously.
- What kills most people: Pain free disease; there only event is the MI that kills them