MED/SURG 1

pretty much everything you need to know
Look at you! You’re amazing! And you’re soon to be even more amazing since you’re taking steps to learn as much as you can in nursing school. Yay, you!

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The cardiovascular system is one of the most interesting you’ll come across in your nursing school career...and this is just the beginning. In Advanced Med/Surg you’ll really dive into the complexities of this system and all the pathophysiology that make it a favorite of many nurses. I hope you “heart” the heart as much as I do, and enjoy these notes as you start learning about the nursing care of individuals with cardiovascular disorders. Rock on!

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Key Concept
The first thing to understand about the cardiovascular system is that the purpose of the entire system is to provide oxygenation and perfusion to the body. There are four concepts related to oxygenation and perfusion that you will see again and again. It is imperative that you understand these...they are:

1. Hypoxemia: decreased oxygen concentration of arterial blood
2. Hypoxia: oxygen deficiency in body tissues
3. Ischemia: tissue not getting enough oxygen
4. Necrosis: tissue death

Overview
Cardiovascular (CV) disease is the leading cause of death in the US. One death occurs every 33 seconds, and 25% of the population has CV disease. Yes, it’s pretty bad, folks. Most of the patients you see in the hospital will have some kind of cardiovascular disease.

Technology & Diagnostics
How is CV disease diagnosed and treated? Glad you asked! Though there are advancements occurring all the time, the mainstays of testing and treatment are:

- EKG: This is your typical electrode-based diagnostic tool. This could be as few as three leads (or electrodes), and as many as 12 (simply called a “12-lead”). The electrodes create a graphic representation of the electrical impulses that the heart generates during the cardiac cycle. Don’t worry, we’ll go into this in much more detail in Med/Surg 2. Interfering factors are electrolyte imbalances and certain drugs such as digitalis, quinidine and barbituates.

- Ultrasound (ECHO): Used to evaluate the structure and function of the heart. It’s not all that accurate in patients with COPD (due to a lot of air between the heart and chest cavity) and patients who are obese. See more here.

- Stress Test: A stress test is exactly what it sounds like...the heart is tested while under stress. The first part of the test is called the “resting” portion, which is essentially an EKG conducted while the patient is resting. The “stress” portion is done next for purposes of comparison. Most patients will undergo the stress portion by simply walking on an inclined treadmill (in some cases, they may have to actually jog a little in order to get the heart rate up), but what about patients who are too sick or disabled to ambulate safely? Those folks can still get their stress test, but the stress is chemically induced. And yes, it should make you a wee bit nervous because these are very powerful drugs! The patient is injected with a medication that will make their heart beat faster while the technician records everything on the EKG. The most commonly used medications are adenosine, dobutamine or dipyridamole (Persantine). We’ll talk about these more in the Advanced Med/Surg section.

- Angiography: Also known as a “heart cath” or, if you want to be fancy, then call it by its full name of “cardiac catherization.” Whatever you call it, this is an invasive procedure used to visualize the heart chambers, arteries and great vessels. It is used most often to
Angiography con'td

evaluate patients with chest pain and patients who have had a positive stress test to locate the region of coronary occlusion. It is also used to determine the effects of valvular heart disease. Most heart caths are performed from the left side, but right-sided heart caths are also regularly conducted to calculate cardiac output, measure right heart pressures, and identify pulmonary emboli. If your patient is really sick, they may get both.

Radioisotope studies:

Sometimes, the physician needs an even more detailed picture of what is going on in the heart...and this means you’ll be going on a road trip with your patient to Nuclear Medicine (also called “Nuc Med” by those in the know.) These are relatively non-invasive (except for the IV that’s required) and are generally well-tolerated by the patient. Like many tests, it does require that the patient hold still for extended periods of time, so if your patient has a lot of anxiety or is confused, this could be a problem!

For this test, a small amount of radioactive material is injected and the emissions from this material are detected with the imaging device to show distribution of blood flow to the heart and details about cardiac function. And yes, it’s pretty cool to watch!

Clot prevention:

In some cases, your patient may simply need to take clot-prevention drugs such as warfarin (Coumadin), heparin or one of the newer type of “blood thinners” such as Pradaxa or Xarelto.

Each has benefits and risks, but the main difference between warfarin or heparin and the newer medications is that the newer ones don’t require regular blood monitoring. Before you go and get too excited, think about it...an anticoagulant medication that doesn’t require monitoring can be extremely risky. It is also important to understand that while warfarin and heparin both have antidotes, these newer drugs do not.

Google Pradaxa or Xarelto along with the word “hemorrhage” and you’ll understand pretty quickly why these medications should not be taken lightly.

Clot lysis:

If you recall that “lysis” refers to breaking something apart, then you’ll realize this is referring to the “clot busting” drugs, also known as thrombolytics. The most commonly used medications are activase and alteplase (tPA). These drugs help to break up and dissolve clots that are occluding vessels.

Angioplasty:

This invasive procedure involves placing a balloon at the stenotic area to widen a narrowed or obstructed blood vessel.

Endarterectomy:

This is a surgical procedure to remove plaque material or blockage in the lining of an artery...usually the carotid artery in the neck.

**“Going on a road trip”**

refers to any time you have to take the patient out of the room for a diagnostic test or procedure. Critically ill patients in ICU require a nurse to be present at all times, so if you work in intensive care, and your patient needs a CT scan, MRI or Nuc Med study, guess what? You are going on a road trip! And yes, it is as scary as it sounds...but never fear, prepare well and you’ll do great!
Stent: The placement of a **stent** holds an artery open. Patients who receive stents will have to take anticoagulant medication for life as clots like to form on foreign objects.

Valve surgery: When the heart valves are faulty, they can be repaired or replaced. The types of surgeries refer to the valve in question...for example, an AVR is an aortic valve replacement while an MVR refers to the mitral valve. Valves are repaired or replaced when they are not operating optimally as in regurgitation or stenosis. More on these issues later on!

Bypass surgery: CABG (coronary artery bypass) and peripheral bypass are super serious surgical procedures. In a **CABG** (pronounced “cabbage”), arteries or veins from elsewhere in the body are grafted to the coronary arteries in order to bypass atherosclerotic narrowings and improve the blood supply to the heart. Peripheral arterial bypass refers to treating blockages in the vessels of the legs.

Amputation: In cases of severe vascular disease, sometimes amputation is the only available treatment.

**Arterial & Venous Disorders**

It is important to understand that the cardiovascular system involves two types of blood vessels... arteries and veins. You absolutely must know the implications of an arterial disease vs. a venous disease as the signs/symptoms are different as are the treatments. More details on this further on.

**Examples of arterial disorders:**

- **Artherosclerosis:** Both coronary artery disease (CAD) and peripheral artery disease (PAD) involve thickening and hardening of arterial walls. When these vessel walls harden and become thick, blood flow is impaired, so you will see problems downstream of the artery. More on the difference between PAD and PVD later on.

- **Hypertension:** BP over 140/90

- **Aneurysm:** A localized abnormal dilation of a blood vessel (usually artery).

- **Raynaud’s Disease:** A primary vasospastic disease of small arteries, presents as an overly exaggerated response of vasomotor controls to cold or emotion. This is most easily observed in the hands, which will turn pale as the tiny blood vessels limit flow to the periphery.

- **Buerger’s:** This is a chronic, recurring, inflammatory, vascular occlusive disease, chiefly of the peripheral arteries and veins of the extremities. Be careful that you do not confuse this with “Berger’s Disease” which is something else altogether! **Buerger’s** is strongly associated with tobacco use, and treatment often involves amputation of the affected body part.

**Examples of venous disorders:**

- **Thrombophlebitis:** Inflammation of a vein in conjunction with the formation of a thrombus.
Emboli: Masses of undissolved matter present in a blood or lymphatic vessel and brought there by the blood or lymph.

Venous Stasis Ulcers: These are ulcers in the lower leg (usually inner part of leg just above ankle). They can get pretty intense, leaving shallow wounds in the leg that are extremely difficult to heal. They are common in patients who have a history of leg swelling, varicose veins or blood clots.

Lymphedema: An abnormal accumulation of tissue fluid in the interstitial spaces.

**Cardiovascular Disease**

Though CV disease is the leading cause of death in the U.S. the really sad thing is that there are many risk factors associated with this disease that are modifiable:

- Hypertension
- Obesity
- Smoking
- Hyperlipidemia
- Stress
- Lack of Exercise
- Diabetes
- Excess Sodium
- Alcohol

**Hypertension**

Hypertension (HTN) affects about 25% of the US population (50 million folks), and represents any BP above 140/90. However, it is important to note that ANYTHING above 120/80 is considered risky because there is a direct relationship to CV disease as blood pressure rises. Note that elderly people can have an increase in systolic pressure only...this is because atherosclerosis causes a loss of elasticity in the large arteries and diastolic pressure does not increase in correlation with systolic.

What does HTN do that’s so damaging? For starters, it damages the intima of the vessel wall. Recall from your anatomy course that the intima is a delicate interior lining of the vessel. As it becomes damaged, macrophages are attracted to the area causing an increase in inflammation. This, in turn, creates more work for the heart to pump against (afterload), and can lead to CHF and cardiovascular “events” such as heart attack and stroke.

**Pathophysiology of Hypertension:**

1. **Step 1:** The kidneys release renin into the bloodstream, which travels to the liver where it converts aniotensinogen to angiotensin I.

2. **Step 2:** Angiotensin I goes to the lungs where it is converted to angiotensin II, which is a powerful vasoconstrictor!

3. **Step 3:** Angiotensin II then goes to the kidneys which causes aldosterone to be released. Aldosterone causes sodium and water retention. This retained sodium and water both increase blood volume.

4. **Step 4:** Arteriolar constriction increases peripheral vascular resistance (remember that angiotensin II is a potent vasoconstrictor!). This increase in blood volume combined with the vascular resistance work together to cause hypertension.

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*Studies show that the incidence of MI increases proportionately with increases in systolic pressure over 120. Studies also show that diabetics are less at risk of “CV events” when diastolic pressure is <80.*
Treating Hypertension:
Lifestyle modifications are fantastic, but they won’t fix everyone’s HTN. Sadly, many people find lifestyle changes difficult to adhere to, and the consequences of leaving HTN untreated are devastating. So, most people need to be on more than one type of BP drug as each type lowers the blood pressure in a different way. If someone is going to implement lifestyle changes, they can drop their BP 5-20 mmHg for every 10kg they lose...this is great IF they can stick to it!

Pre-hypertension: Systolic 120-139 / Diastolic 80-89
The plan here is lifestyle modification (diet, smoking cessation, increase activity, cut back on sodium).

Stage I hypertension: Systolic 140-159 / Diastolic 90-99
The typical treatment at this stage is a thiazide diuretic +/- beta blocker

Stage II hypertension: Systolic 160+ / Diastolic 100+
The typical treatment will utilize multiple medications...usually a thiazide diuretic plus an angiotensin-converting enzyme inhibitor (ACEi), angiotensin receptor blocker (ARB), a beta blocker (BB) or a calcium channel blocker (CCB). In some cases, patients will be on three + meds.

Obesity
Obesity is associated with increased risk of CV disease. This is especially true for “central obesity” which is when the extra weight is carried around the abdominal area and the vital organs of the trunk. Note that obesity is an independent risk factor for HTN, but it is often associated with other risk factors such as Type 2 diabetes, inactivity and hyperlipidemia. The dangers of obesity are very real...one study followed 1 million Americans for 14 years and found the risk of CV death to be 2x higher in obese individuals.

Some scary obesity statistics:
- 60% of adults are overweight or obese
- The percentage of overweight young people has more than doubled in the past 30 years
- Between 10-15% of Americans age 6-17 are overweight
- The annual cost for the US is $100 billion

Smoking
As of 2013, 17.8% of adults in the US continue to smoke, but this is down from 42% in 1965, when the CDC started tracking the data. So while we are definitely doing better, we’re not there yet!

Smoking is the strongest promoter for artherosclerosis...how does this happen, you ask? In general terms, smoking increases peripheral vascular resistance (PVR), increases LDL (“lethal” cholesterol) and decreases HDL (“healthy” cholesterol). The arterial endothelium is damaged, and platelets (which have increased aggregation due to smoking), are thought to adhere to subendothelial connective tissue exposed by endothelial denutation, initiating the smooth muscle proliferation that leads to artherosclerotic plaque formation. Whew! That’s a mouthful! The short answer is...smoking leads to atherosclerosis!

What’s more, as many as 30% of coronary heart disease (CHD) deaths in the US each year are attributed to smoking and the risk is strongly dose-related. It also nearly doubles the risk of ischemic stroke because the carbon monoxide more readily binds to hemoglobin than does oxygen and tissues suffer from lack of oxygen. What’s the takeaway here? Encourage your patients to stop smoking!
Some facts regarding smokers who have had an myocardial infarction:
- If the person quits smoking now, there is a 50% reduction in the risk of reinfarction, sudden cardiac death and total mortality
- This group is highly receptive to teaching, so teach!
- When MI pts are given information about quitting, there is a 50% long-term cessation rate...this is awesome!
- Modest telephone-based counseling can increase this percentage to 70% and is cheap!

**Smoking Cessation**
- Approximately 1.3 million quit each year
- After 1 year off cigarettes, the excess risk of heart disease is reduced by half.
- After 15 years of abstinence, the risk is similar to that of people who never smoked.
- In 5-15 years, the risk of stroke returns to the level of those who've never smoked.
- Only 50% of smokers seen in primary care were spoken to about smoking.
- It is better to have a “quit day” than to taper.
- Hypnosis is not supported by sufficient evidence.
- Vaccines have been tested, perhaps someday!
- Counseling works!
- Drugs work! Nicotine patch or gum, bupropion, varenicline.
- Chart all smoking cessation teaching!
- Smoking is the cause of more than 10% of CV deaths.
- Smoking costs the US $90 billion year.

**Hyperlipidemia**
Generally, low density lipoproteins (LDL) are the “bad” kind of lipid or cholesterol...an easy way to remember this is that L stands for “lethal.” In general, each 1% drop in LDL confers to a 2% reduction in CV events. Note that there are actually two types of LDL...the small dense type (which is actually what contributes to CV disease) and the large “fluffy” type. This probably won’t come up, but more research is being done in this area, so I thought I’d put it out there. High density lipoproteins (HDL) are the “healthy” ones. When you look at someone’s total cholesterol level, you want to take into account what the ratio is between their HDL and the LDL. If LDL is low and HDL is high, then you are good to go!

**Treating Hyperlipidemia**
The chosen treatment depends on several factors including: lipid profile/levels, other CV risks and the cost of therapy. Generally, patients are advised to change their diet (low-fat, low cholesterol), adopt healthy lifestyle changes, and take drugs (statins, resins, fibrates, niacin).

**Stress**
Stress plays a huge role in cardiovascular risk. In fact, high stress can DOUBLE the risk of CV morbidity. That’s huge! This is a tough one because it’s not enough to just tell patients to “chill out” or relax. Many people will need help learning healthy ways to cope with stress...this can be through things such as meditation, exercise, deep breathing, behavioral therapy or improved communication. What you want to avoid is patients de-stressing in unhealthy ways such as smoking, drinking, binge eating, or using drugs.

**Lack of Exercise**
Sadly, 60% of Americans are sedentary! Physical inactivity is another one of those independent risk factors for CV disease. Improving activity reduces weight, improves lipid profiles and reduces BP... yay for exercise!

A 1996 statement from the NIH recommends that adults accumulate at least 30 mins of moderate activity on most days (and yes, walking to class counts so park far far away!), and more vigorous activity 3-4x a week for 30-60 minutes. Your heart and lungs will thank you!
**Diabetes**
Interestingly, the goal is not to keep patients’ blood glucose at “the right level” as this historically lead to too many incidents of hypoglycemia. Instead, the goal is to keep patients closer to 70-130 when they’re not sick, and usually under 160-ish when they’re in the hospital...this is because blood sugar levels rise when the body is under stress...and since overshooting the goal can be dangerous, we just let it ride a little higher. One of the problems diabetics have as it relates to cardiovascular is with arterial perfusion, which leads to necrotic tissues in the periphery. You will see that many diabetics have poor tissue perfusion, poor wound healing and fragile skin.

**Sodium Intake**
In obese patients, each 2g increase in sodium intake is associated with a 61% increase in CV mortality.

**Alcohol Intake**
And now for some good news! Moderate alcohol consumption (identified as 1 glass of wine for women and 2 glasses for men) increases HDL, may decrease coagulation and lower blood pressure. However, moderation is the key. Large alcohol intake increases blood pressure, risk of stroke, cardiomyopathy and non-cardiac disorders. Note that binge drinking is a real problem, especially among college aged males.

**Non-Modifiable Risk Factors**
Unfortunately, there are a few risk factors that are outside of our control...these are gender, age and genetics. Males are more likely to have CVD than females, and the older you get the higher the chance. Genetics is simply the luck (or lack of it) of the draw.

**Atherosclerosis**
Atherosclerosis occurs when fatty plaques accumulate within the vessel wall causing the opening to narrow. This decreases the elasticity of the vessel and reduces the blood volume that can flow through the vessels. This leads to ischemia and, in sever cases, necrosis. In chronic atherosclerosis, the individual develops collateral circulation (think of this as branches of vessels) that enable the tissues to get blood flow via an alternate route. Note that this takes time to develop. In cases of acute atherosclerosis, the effects are often more pronounced as collateral circulation has not yet developed.

**Congestive Heart Failure**
Congestive heart failure (CHF) occurs when the heart is not able to pump adequately to meet metabolic demands. As the heart pump fails, blood pressure drops, cardiac output drops, blood backs up into places where it shouldn’t be and the patient feels fatigued and/or short of breath. There are two kinds of CHF...right-sided and left-sided. Understanding the pathophysiology and signs/symptoms of each is important, so pay attention!

**Left or Right?**
In order to understand the concept of left vs right-sided heart failure, you must think about the pathway of bloodflow through the body...so let’s review!

1) Blood is pumped out of the left ventricle into the body.
2) Blood travels to the periphery and back around to the right atrium, then right ventricle.
3) Blood is pumped out of the right ventricle into the lungs.
4) Oxygenated blood flows into the left atrium and then the left ventricle.
5) Blood is pumped out of the left ventricle into the body....and around and around we go.

So think about what would happen in left heart failure. The blood is supposed to be pushed out to the body, but instead it
backs up. Where is it going to back up to? Well, what's on the pathway BEFORE the blood goes into the left side of the heart? The lungs! You got it! So, in left heart failure, the blood backs up to the lungs causing pulmonary edema. Because fluid gets into the alveoli and reacts with the surfactant, you get foamy sputum...the foam is basically the detergent (surfactant) mixed with fluid. You won’t see this all the time, so don’t discount left heart failure if you don’t see it. The most common symptom you will see is shortness of breath with decreased O2 saturation levels, and you’ll probably be able to hear that the lungs sound “wet.”

Now, for right heart failure...again, think about the pathway. Blood travels from the periphery to the right side of the heart. So, if the right heart fails, you get peripheral edema and a tender/enlarged liver (called “congestive hepatomegaly”) that occurs when the hepatic veins become engorged.

Not to get too tricky on you or anything, but the most common cause of right-sided heart failure is actually left-sided heart failure...so it’s always possible that your patient has both. If they do, they are probably in a pretty bad way. This occurs because when left-sided heart failure occurs, blood backs up so much that it backs all the way into the right ventricle, and so on and so forth. Think about the pathway and it all makes absolute perfect sense!

The most common symptoms of CHF are dyspnea and fatigue

Risk Factors and Etiology of CHF
Heart failure may result from a primary abnormality of the heart muscle (like an infarction) that impairs ventricular function and prevents the heart from pumping enough blood. It can also be caused by other problems:
- Mechanical disturbances in ventricular filling during diastole (due to blood volume that’s too low for the ventricle to pump) occur in mitral stenosis secondary to rheumatic heart disease or constrictive pericarditis and in atrial fibrillation.
- Systolic hemodynamic disturbances (excessive cardiac workload caused by volume overload or pressure overload) limit the heart's pumping ability. This can result from mitral or aortic insufficiency, which leads to volume overload. It can also result from aortic stenosis or systemic hypertension, which causes increased resistance to ventricular emptying and decreased cardiac output.
- Myocardial infarction.
- Lung disease decreases oxygen saturation levels, so the body compensates by vasoconstricting the vessels in the lungs. This causes a lot of resistance for the right side of the heart to pump against, and it gets into trouble, leading to right-sided heart failure.

Ventricular Function
How well the ventricle pumps depends on several factors:
1) Contractility: How well does the heart contract? Contractility directly affects stroke volume, which affects cardiac output. Recall that heart rate x stroke volume = cardiac output!
2) Preload: How much blood is delivered to the heart (venous return)? If you are dehydrated, this will cause low preload, so you need to give that person fluids. Preload also affects stroke volume (and thus cardiac output).
3) Afterload: Is the heart pumping against a lot of pressure? If so, stroke volume will go down.
4) Heart rate: Elevated heart rates mean less time for ventricular filling, so cardiac output will be reduced.
5) Left ventricular wall integrity, synergistic left ventricle contraction and valvular competence are all related to cardiac output. If any of these factors are not operating well, cardiac output will be reduced.
Classification of CHF (based on symptoms)

CHF can range from asymptomatic left ventricular (LV) dysfunction all the way to refractory CHF (“refractory” simply means anything that is resistant to treatment). If we treat CHF early before symptoms appear, we have a better chance of treating the disease overall. You may hear these classes referred to as the patient’s “functional capacity.”

Normal heart: Has no symptoms, has normal exercise ability and normal left ventricle function.
Class I: Asymptomatic. Has no symptoms, normal exercise response and abnormal left ventricle function. The ejection fraction (EF) may be down, but you’d only know this if you did an ECHO.
Class II: Compensated. Has no symptoms unless exercising, left ventricle function is abnormal.
Class III: Decompensated. Has symptoms when not exercising, left ventricle function is abnormal.
Class IV: Refractory. Has symptoms that are not controlled with treatment. This guy needs a heart transplant.

Classification of CHF (based on assessment of patient)

Class A: No evidence of disease, no symptoms, and no limits on physical activity.
Class B: Minimal cardiovascular disease. Mild symptoms and slight limitation during ordinary physical activity, but patient is comfortable while resting.
Class C: Moderately severe cardiovascular disease. Very limited in regards to activity (even light activity), however patient is comfortable while resting.
Class D: Severe cardiovascular disease. Severely limited in regards to activity, patient has symptoms even while resting.

CHF Treatment Objectives

The goals with treating CHF are to increase survival, decrease morbidity, increase exercise capacity, increase quality of life, decrease neurohormonal changes, halt progression of the disease (or at least slow it) and decrease symptoms. The reason you want to decrease neurohormonal changes is because when blood pressure is low, the neurohormonal response activates the SNS…this stresses the heart (think about what the SNS does to the heart). So, the heart remodels which means it gets fibrous tissue. Unfortunately, fibrous tissue does not work as well so we will turn off the neurohormonal response with medications to prevent fibrosis in the heart. What meds do we use to accomplish this? ACE-inhibitors and Beta Blockers.

Treatments for CHF

The main goal with CHF is to correct aggravating factors:

- Pregnancy
- Arrhythmias such as atrial fibrillation
- Infections
- Hyperthyroidism
- Thromboembolism
- Endocarditis
- Obesity
- Hypertension
- Physical activity
- Dietary excess
- Medications (more on this in a moment)

Increased Risk for CHF

- arrhythmias
- bradycardia
- pregnancy
- thyrotoxicosis
- pulmonary embolism
- infections
- anemia
- increased physical activity
- increased salt intake
- increased water intake
- emotional stress
- failure to comply with Tx

In terms of treatment, it depends on the severity of the disease. It is classified by the New York Heart Association as Class I-IV, based on the ability of the pt to exercise without symptoms. It is staged based on evolution of the disease on a letter scale A through D (see above). How we treat it combines both of these assessments together:
Class A is the individual who is at high risk, but not actually showing signs. The goal here is to lower the risk and we do this by giving an ACEi.

B is for anyone who has asymptomatic LV dysfunction, an EF < 40% (class I). This person will get an ACEi and a B-Blocker in an effort to reduce risk.

C is for symptomatic CHF (class II and III). This person will get an ACEi, a B-Blocker, diuretics and be on a low sodium diet...all in an effort to reduce risk. They may also get an ARB (angiotensin II receptor blocker) and digoxin.

D is for the most at risk person...symptomatic CHF (class IV). This person will get specialized therapy and get in line for a heart transplant.

Common Clinical Findings of CHF
- dyspnea
- fatigue
- cheyne stokes respirations
- orthostatic hypotension
- liver tenderness
- liver enlargement
- peripheral edema
- pulmonary crackles
- weak pulses

QUIZ TIME! A 62 year old tests negative for a heat attack. Her BP is 151/84 and she has an EF of 35%. She exhibits signs of dyspnea and does yoga three times a week. Whameds would you anticipate she should have? (see answer at the end of this document!)

Medications for Treating CHF

ACE inhibitors (ACEi)
ACE inhibitors inhibit the renin-angiotensin-aldosterone system. Recall that this system is activated in response to hypotension, decreased sodium concentration in the distal tubule, decreased blood volume and renal sympathetic nerve stimulation. The kidneys release renin which cleaves angiotensinogen into angiotensin I. Angiotensin I is then converted into angiotensin II via the angiotensin converting enzymes (ACE) in the lungs (also in the endothelium of blood vessels in many parts of the body). Angiotensin II causes vasoconstriction and the release of ADH (among other things). Both of these work to increase blood pressure. If this pathway is inhibited, then the increase in blood pressure is thus inhibited. That's what an ACEi does.

ACEi drugs end in the word “pril”. Quinapril was studied in 1993 and the study shows that patients who received quinapril did not require any additional treatment, as compared to the group who took quinapril for a while and then took a placebo.

The advantages of ACEi are:
- Inhibit left ventricular remodeling post myocardial infarction (remember that ACEi block the “neurohormal pathway” introduced earlier)
- Modify the progression of chronic CHF (increased survival and decreased hospitalizations; improved quality of life)
- In contrast to other vasodilators, do not produce neurohormal activation or reflex tachycardia
- Tolerance to its effects does not develop
- Studies show that the probability of death decreases when taking ACEi vs a placebo

ACEi indications are:
- Clinical cardiac insufficiency (all patients)
- Asymptomatic ventricular dysfunction (LVEF < 35%)
- High risk CHF pts (those with diabetes, hypertension, atrioventricular septal defect, or anyone who’s had a myocardial infarction).

What is an Ejection Fraction?
An “ejection fraction” or “EF” is a measure of the percentage of blood leaving the heart each time it pumps...it shows how well the heart pumps with each beat. A normal LVEF is 55-70%.
Angiotensin I Inhibitors (also known as AT1 receptor blocker, or ARB)
These drugs block vasoconstrictor and aldosterone-secreting effects of angiotensin II at various receptor sites including vascular smooth muscle and the adrenal glands. It leads to vasodilation and lowers blood pressure! Some common ARBs are Losartan, Valsartan, Irbesartan and Candesartan.

Diuretics (Thiazides, Potassium-Sparing, and Loop)
Thiazides act on the cortex of the kidney. They inhibit active exchange of Cl and Na in the cortical diluting segment of the ascending loop of Henle. They increase the excretion of sodium and water by inhibiting sodium reabsorption. In the process, hydrogen ions are also excreted, which can contribute toward metabolic alkalosis. Remember that water follows salt, so if we excrete sodium, we also excrete water. The most common one you will see is Hydrochlorothiazide.

Potassium-sparing diuretics act on the medulla and inhibit resportion of Na in the distal convoluted and collecting tubule...they also save K, so you will want to make sure K not too high! They also save hydrogen ions, so metabolic alkalosis is less of an issue. Spironolactone is a common one you’ll see.

Loop diuretics act on the medulla and inhibit the exchange of Cl, Na, K in the thick segment of the ascending loop of Henle. Note that hydrogen ions are also lost with loop diuretics as well. A common loop diuretic is Lasix (furosemide).

With both thiazide and loop diuretics, you absolutely must watch for hypokalemia...if your patient is given one of these medications and excreting a lot of urine (and with it a lot of potassium), their levels can drop leading to cardiac arrhythmias.

Digoxin
Digoxin binds to the Na/K/ATPase pump in the membranes of heart cells and decrease its function. This causes an increase in the level of sodium ions in the myocytes which then leads to a rise in the level of calcium ions. This causes an increase in the length of Phase 4 and Phase 0 of the cardiac action potential, which when combined with the effects of digoxin on the CNS, leads to a decrease in heart rate. Increased amounts of calcium are then stored in the sarcoplasmic reticulum and released by each action potential, which is unchanged by digoxin. This leads to increased contractility of the heart. Digoxin also increases vagal activity via its action on the CNS, decreasing the conduction of electrical impulses through the AV node. Whew! The short version is that digoxin makes the heart beat more slowly and stronger:

• Increases the force of myocardial contraction
• Prolongs refractory period of the AV node
• Decreases conduction through the SA and AV nodes
• Causes increased cardiac output and slowing of the heart rate.

However, don’t go thinking digoxin is the holy grail of CV medications. The long-term effects are mixed:
• Survival similar to that of the placebo
• Fewer hospital admissions
• More serious arrhythmias
• More myocardial infarctions

Vasodilator Drugs
Vasodilators affect preload and afterload. There are different kinds of vasodilators:

Arterial vasodilators reduce arterial pressure by decreasing systemic vascular resistance. This benefits patients in heart failure by reducing the afterload on the left ventricle, which enhances stroke volume and cardiac output and leads to secondary decreases in ventricular preload and venous pressure. Patients with angina benefit from arterial dilators because they decrease the oxygen demand of the heart, thereby improving the oxygen supply/demand ratio. Ex: Minoxidil, Hydralazine
Venous vasodilators reduce venous pressure, which reduces preload on the heart thereby decreasing cardiac output and the workload of the heart. This also decreases proximal capillary hydrostatic pressure, which reduces capillary fluid filtration and edema formation (edema is a result heart failure). Ex: Nitroglycerin (which is a nitrate)

To sum up...nitrates have several different effects:
- Venous vasodilation (reduce preload leading to reduced pulmonary congestion, reduced ventricular size, reduced ventricular wall stress and reduced venous oxygen saturation)
- Coronary vasodilation increases myocardial perfusion (which is why it is often given when patients are experiencing chest pain).
- Arterial vasodilation decreases afterload leading to decreased cardiac output and blood pressure.

Tolerance to Nitrates
After being administered nitrates for awhile, patients begin to experience nitrate tolerance...or a decrease in the effectiveness of the drug. This tolerance develops with all nitrates and is dose dependent. However, it decreases in 24 hours after stopping the drug. The good news is, the tolerance can be avoided if you administer nitrates at the lowest possible dose and create discontinuous plasma levels. You’ll notice that when administering nitrates you’ll often be instructed to apply the nitro paste for a period of time, remove it for a period of time, then reapply it. So, when you see that your patient has a “drug-free” period here and there...that’s why!

When giving nitrates, you want to keep an eye on the patient’s blood pressure. If the blood pressure is low, you’re going to hold the medication. If the blood pressure drops while the medication is being administered, you’re going to stop the infusion (if running through IV) or wipe off the paste (if being administered transdermally). Be super careful that you don’t touch the paste...you will get a nasty headache!

Aldosterone Inhibitors
In addition to being a potassium-sparing diuretic, Spironolactone is an aldosterone inhibitor. It’s not as powerful a diuretic as Lasix, but since it blocks aldosterone, it helps decrease fibrous tissue formation (remodeling). Note that since this drug conserves potassium, you will hold the drug if your patient becomes hyperkalemic.

Beta-Blockers
Carvedilol is a common beta blocker used to treat heart failure. As the name suggets, beta blockers block beta-1 (myocardial) and beta-2 (pulmonary, vascular, urerine) adrenergic receptor sites. It also has alpha-1 blocking activity which means it may result in orthostatic hypotension. Be careful when getting this patient out of bed!

Therapeutic effects are decreased heart rate and blood pressure, improved cardiac output, slowing of the progression of CHF and decreased risk of death. Yay!

Survival of pts on Beta-Blockers is pretty darn good. Basically, if people are on beta blockers and ACEi, the mortality rate goes down to 13.3% (a decrease from 27.7%)

Anticoagulants
Anticoagulants are given for a variety of reasons:
- Previous embolic episode
- Atrial fibrillation

Digoxin Toxicity
NCLEX (and nursing schools) love to ask questions about digoxin toxicity. The question will usually be about someone with a bradycardia who is nauseous/vomiting and seeing yellow. These are classic signs of digoxin toxicity. The treatment is a drug called Digibind, which is really really expensive...about $700 per vial.
• Identified thrombus
• Left ventricular aneurysm (3-6 months post MI)
• Class III-IV CHF in the presence of an EF less than 30%, and/or an aneurism or a very dilated left ventricle.
• Phlebitis (inflammation of a vein)
• Prolonged bed rest

Nursing Interventions for CHF
Assist with ADLs: This patient is going to be fatigued
Improve SOB: Raise HOB up, administer O2, give meds to decrease preload & afterload
Keep an eye on K: Know how the meds affect K levels, keep an eye on I&O

Peripheral Vascular Disease
Peripheral Vascular Disease can be broken down into arterial and venous diseases. Your exams will ask you to differentiate between arterial and venous disorders, so learn how to keep these straight. In general, here’s what to look for when determining arterial vs. venous:

Skin Characteristics
• Arterial: Dependent rubor, pallor with elevation, hypertrophied toenails, cool skin, hairless extremity, tissue atrophy; ulcers very painful
• Venous: Red color to skin, induration, warmth, tough skin; ulcers moderately painful and usually located on the medial ankle.

Pain
• Arterial: Typically brought on by exercise and relieved by rest (intermittent claudication); sometimes unremitting pain in foot even at rest; sharp and stabbing
• Venous: Tenderness along vein, discomfort may be relieved by applying heat; cramping and aching, walking/activity may help

Pulses
• Arterial: Weak or absent
• Venous: Typically present

Surrounding tissue
• Arterial: Gangrene, delayed wound healing
• Venous: Edematous, itchy and scaly skin, thick/coarse/brownish skin around the ankles

Assessment for PVD is to check for CSM...circulation, sensation and movement. You will monitor for the 5Ps...pain, pallor, pulselessness, paresthesia and paralysis.

To manage peripheral vascular disease we’re going to do a few things:
• Reduce risks
• Clot prevention/dissolution
• Surgery (in extreme cases)
Aneurysm

Aneurysms are classified based on how they are shaped.

- **Saccular** = a unilateral outpouching; has a neck and mouth
- **Fusiform** = a bilateral outpouching; involves the entire circumference
- **Dissecting** = a bilateral outpouching in which layers of the vessel wall separate, creating a cavity; this is not a “true” aneurysm but rather a hematoma in the arterial wall layers
- **False** = the wall ruptures and a blood clot is retained in an outpouching of tissue…or there is a connection between a vein and an artery that does not close

Pathophysiology of Aneurysms

An aneurysm can be venous or arterial. The exact cause is unknown, but recent evidence includes atherosclerosis and hypertension. Genetics can also come into play such as with Marfan syndrome. Other causes of aneurysms include infection, mycotic infections and even trauma (though that last one is rare).

Clinical Manifestations of Aneurysms

- **AAA (Abdominal Aortic Aneurysm)**
  - Can typically be palpated in abdomen once it’s at 5 cm
  - Most common clinical manifestation is the client’s awareness of a pulsating mass in the abdomen, followed by abdominal pain and back pain.
  - Groin pain and flank pain
  - Bruits can be heard over the aneurysm…a bruit is an adventitious sound of venous or arterial origin heard on auscultation
  - Sometimes mottling of the extremities or distal emboli in the feet alert the clinician to a source in the abdomen
  - Ultrasonography and CT are diagnostic tools

- **Ruptured AAA**
  - Pulsating sensation in abdomen
  - Abrupt excruciating pain (ripping or knife-like pain that radiates…this is a big one you’ll see on tests!)
  - Manifestations of shock (pallor, tachycardia, hypotension, dry skin, excessive thirst)
  - Diminished peripheral pulses or unequal pulses
  - Abdominal rigidity
  - Differing blood pressure in arms
  - Paraplegia, hemiplegia
  - Decreased urine output or hematuria

Raynaud’s Syndrome

Raynaud’s is an arterial disease in which the small arteries and arterioles constrict in response to various stimuli. This can be caused by cold, nicotine, caffeine and stress. Obstructive Raynaud’s often seen with autoimmune diseases. The most common symptom you will see is extreme paleness in the fingers and toes, often with a very clear line of demarcation between the perfused and non-perfused areas.

Buerger’s Disease

Buerger’s Disease is an inflammatory disease of the small and medium-sized arteries and veins of the extremities… it appears to be directly related to smoking (reason #5,673 why smoking is bad for you). The main clinical manifestation is pain, digital ulcerations and ischemia. Patients may also have cold sensitivity with color changes and pain. Pulses in the posterior tibial
and dorsalis pedis are weak or absent, and in advanced cases the extremeties may be abnormally red or cyanotic. Ulceration and gangrene are frequent complications...if the patient doesn’t stop smoking they are very likely to lose fingers (or whatever part is affected). This disease is hard to treat...drugs don’t work very well.

**DVT - Deep Vein Thrombosis**  
This is a very common disease that happens most often in the lower extremities. DVT can often be asymptomatic, but you may see unilateral edema, calf pain and fever. The three factors that affect the formation of DVTs are known as Virchow’s Triad:

- Endothelial injury due to trauma or surgery
- Circulatory stasis due to immobility
- Hypercoagulable state

**Prevention and Treatment of DVTs**

- Heparin or warfarin (brand name = coumadin)
- TEDs/SCDs (prevention only)
- Elevation
- Early ambulation (prevention only)

**Mechanism of Action of Antithrombotic Agents**

- Anticoagulants prevent clot formation and extension
- Antiplatelet drugs interfere with platelet activity
- Thrombolytic agents dissolve existing thrombi

**Pulmonary Embolism**

When a thrombus breaks off and travels to the lungs (where it obstructs the pulmonary arterial bed), this is a very very bad situation. Though it can be so mild as to produce no symptoms, a massive embolism (obstructing more than 50% of the circulation) is rapidly fatal.

What to look for:

- First sign is usually dyspnea, may be accompanied by angina or pleuritic chest pain
- Tachycardia
- Air hunger
- Dropping O2 saturation
- Feeling of impending doom (one of the most common signs!)
- Productive cough (may have blood)
- Low-grade fever
- Pleural effusion

Less common signs:

- Massive hemoptysis
- Splinting of the chest
- Leg edema
- Cyanosis, syncope, distended neck veins
- Pleural friction rub
- Signs of circulatory collapse
- Hypoxia
Treatment/Management

- Resuscitate as needed
- Give O2, may need to be intubated
- Give heparin
- If can’t tolerate heparin, then surgery

**Lymphedema**

Lymphedema occurs when lymphatic flow is blocked, but can also occur with low serum protein and high venous pressure (think about the pressure gradients in the vasculature...remember that from A&P? It is treated by elevating the affected body part, compressing the affected body part, providing skin care and infection treatment, as well as administering diuretics.

**Aging and the CV system**

As we age, the efficiency of the heart’s pumping action decreases. This is related to connective tissue changes (decreased compliance), increased fat and sclerosis causing lower cardiac output, arrhythmias and valve incompetence. The vasculature changes as well, with decreased elastin and increased arteriosclerosis.

Answer to question on page 10: Anticipate an ACEi and a beta blocker.