MED SURG SUCCESS PACK

brought to you by

STRAIGHT A NURSING
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. You rock!

By purchasing and downloading this e-book you agree to the terms and conditions, you understand that this is a reference guide and nothing here should be viewed as set in stone as research and medical practices change regularly.

© 2019 Digital Health Media LLC. All content is the property of the Straight A Nursing website (www.straightnursingstudent.com) and Digital Health Media LLC. Replication and distribution of this material is prohibited by law. All digital products (PDF files, e-books, resources, online content and videos) are subject to copyright protection. Each product sold is licensed to a single user and customers are not allowed to copy, distribute, share or transfer the products (and any associated user names or passwords) purchased to any other individual or entity. Fines of up to $10,000 may apply and individuals will be reported to the BRN and their school of nursing if known. In some cases, Digital Health Media LLC may encrypt, force password and/or stamp license details which include customer name and e-mail into its digital products to ensure safety.

This study guide is not intended to be used a medical advice; it is for educational purposes only. When details differ, please defer to specifics as outlined by your own facility or nursing program.

Please send corrections, updates and comments to hello@straightnursingstudent.com
CARDIOVASCULAR SYSTEM

brought to you by

STRAIGHT "A" NURSING
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. Some estimates show that one death occurs every 33 seconds, and 25% of the population has CV disease. Most of the patients you see in the hospital will have some kind of cardiovascular disease (and the sad part is, it is often preventable).

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:

EGG: This is your typical electrode-based diagnostic tool. This could be as few as three leads (or electrodes), and as many as 10 (which provides 12 views, so it’s 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 barbiturates (also, if your patient is brushing his teeth, this looks like V-tach! Always assess your patient BEFORE you freak out!).

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.

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 EGG 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 EGG. 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 catheterization.” 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 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 anticoagulants 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. UPDATE: There is now a reversal agent for Factor Xa Inhibitors called Andexanet alfa. It’s a good thing too. Google Eliquis 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 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.

Bypass surgery: CABG (coronary artery bypass graft) and peripheral bypass are very 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 and so are the treatments. More details on this in a bit.

**Examples of arterial disorders:**

- **Atherosclerosis:** 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:** Stage 1 HTN is SBP 130-139 or DBP 80-89; Stage 1 is SBP > 139 or DBP > 89

- **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 is staged as elevated, stage 1 hypertension and stage 2 hypertension. Any elevations in BP (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:**

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

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

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 and blood pressure.

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.

Studies show that the incidence of myocardial infarction 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 highly encouraging, but requires patient participation.

Each lifestyle modification can decrease SBP by 4-5 mmHg and DBP by 2-4 mmHg:
- weight loss
- limit alcohol intake (1 drink for the ladies, no more than 2 for the gentlemen)
- increase physical activity

Adhering to a heart healthy diet can decrease SBP by approximately 11 mm Hg:
- low in sodium
- low in saturated fat
- eat more fruits, vegetables and whole grains

The treatment for hypertension is tailored to the patients blood pressure and other risk factors for cardiovascular disease.

Low-risk patients with elevated BP or stage 1 HTN with low risk for cardiovascular disease typically start with lifestyle modification and have their blood pressure re-checked in 3-6 months. They should be counseled on smoking cessation, dietary changes, exercise, and alcohol use.

Patients with stage 1 HTN who are at higher risk for cardiovascular disease are treated with lifestyle modifications and antihypertensive medication. The initial first-line medication is either a thiazide diuretic, CCB, ARB or ACEi. BP is checked again after one month of starting medication.

Patients with stage 2 HTN are treated with lifestyle modification and two or more antihypertensive medications from different drug classes; they should see their primary care provider one month after starting the medication.

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.

According to the CDC's 2018 data:
- The prevalence of obesity among adults was 39.8%
- The annual medical costs associated with obesity was $147 billion
- Obesith-related conditions account for some of the leading causes of premature death in the US
- Hispanics and non-Hispanic blacks have higher prevalence of obesity than non-Hispanic whites and non-Hispanic Asians

Smoking
According to the CDC, as of 2015 15.1% 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 atherosclerosis...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 denudation, initiating the smooth muscle proliferation that leads to atherosclerotic 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 a myocardial infarction:
- If the person quits smoking now, they have a 50% reduction in the risk of re-infarction, sudden cardiac death and total mortality
- This group is highly receptive to teaching, so teach!
- When MI patients are given information about quitting, there is a 50% long-term cessation rate, which 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 have 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 (Chantix).
- Chart all smoking cessation teaching
- Smoking is the cause of more than 10% of CV deaths.
- Smoking costs the US $90 billion year.

Environmental smoke has about 34% of the impact on atherosclerotic progression that occurs with active smoking.

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.

The NIH recommends that adults partake in 30 mins of moderate activity five days a week, for a total of 2.5 hours. Adding moderate to vigorous activity for 60 minutes most days of the week is recommended to help adults avoid gradual weight gain.

**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 150-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 the cardiovascular system 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 serious 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 severe 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 let’s do a quick overview.
Left or Right?

In order to understand the concept of left vs right-sided heart failure, you must think about the pathway of blood flow through the body.

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.

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 is present, 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 (such as 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 ventricle 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.”

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

Classification of CHF (based on assessment of patient)

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

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, which stresses 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.

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 therapy

Treatments for CHF
The main goal with CHF is to correct aggravating factors:
- Pregnancy (symptoms often resolve after giving birth)
- Arrhythmias such as atrial fibrillation
- Infections
• Hyperthyroidism
• Thromboembolism
• Endocarditis
• Obesity
• Hypertension
• Physical activity
• Dietary excess
• Medications (more on this in a moment)

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 patient 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 heart attack. Her BP is 151/84 and she has an EF of 35%. She exhibits no signs of dyspnea and does yoga three times a week. What meds would you anticipate this patient is prescribed? (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. Lisinopril is one of the more common ones you’ll see these days.

The advantages of ACEi are:
- Inhibit left ventricular remodeling post myocardial infarction (remember that ACEi block the “neurohormonal 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 neurohormonal 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 patients (those with diabetes, hypertension, atrioventricular septal defect, or anyone who’s had a myocardial infarction).

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. Notice they end in “-sartan”.

Diuretics (Thiazides, Potassium-Sparing, and Loop)
Thiazides act on the cortex of the kidney. They inhibit active exchange of \( \text{Cl} \) and \( \text{Na} \) 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 reabsorption of Na in the distal convoluted and collecting tubule. They also save K, so you will want to make sure K is 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 decreases 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 of 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, patients begin to experience nitrate tolerance. This is 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 likely going to hold the dose. 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 careful that you don’t touch the paste as you will get an intense headache (often referred to as a “nitro 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 want to talk to the MD about holding the drug if your patient becomes hyperkalemic.

**Beta-Blockers**
Carvedilol is a common beta blocker used to treat heart failure. As the name suggests, beta blockers block beta-1 (myocardial) and beta-2 (pulmonary, vascular, uterine) 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 patients 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
- 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 you are 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, which are pain, pallor, pulselessness, paresthesia and paralysis. If your patient has any of these signs, notify the MD STAT as the patient may be losing blood flow to the limb and possibly needs immediate intervention to restore perfusion.

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-life back pain that radiates...this is a big one you’ll see on tests!)
Manifestations of AAA cont’d

• 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 is 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 extremities 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 and drugs don’t work very well.

DVT - Deep Vein Thrombosis
This is a very common disease that happens most often in the lower extremities, but can also occur in the upper extremities (especially if a PICC line is in place). DVT can often be asymptomatic, but you may see unilateral edema, 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 Anti-thrombotic 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 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 or TPA
  • If can’t tolerate heparin or TPA, 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 that you learned in 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.
Respiratory Disorders - The Lower Airway

Respiratory disorders of the lower airway involve everything from the tracheobronchial tree and below. This includes the trachea, the right and left mainstem bronchi, the segmental bronchi and the terminal bronchioles and lung tissues. Common diseases of the lower airway include:

- asthma
- chronic obstructive pulmonary disease (COPD)
- tracheobronchitis
- pneumonia

Asthma

Asthma is a disorder of the bronchial airways characterized by periods of reversible bronchospasm, mucosal edema, increased mucus production, and airway inflammation. Though the spasm is reversible, people still die of asthma attacks, so it’s important that your patients with asthma understand their treatment plans (more on this later). Asthma can be inherited and is often a result of environmental factors (pollen, dust mites, mold, animal dander, smoke, pollution, and even cockroaches), exercise, changes in temperature, strong odors, excitatory states, beta blockers and aspirin.

Pathophysiology: Early-Phase Reaction vs. Late-Phase Reaction

In an early-phase reaction (meaning the reaction occurs soon after the exposure) the beta lymphocytes produce immunoglobulin E. These IgE antibodies attach to mast cells and basophils in the bronchial walls and mediators are released. The mediators cause the edema and bronchoconstriction which are the hallmark of an asthma attack. When the bronchioles are narrowed, expiratory air expulsion is decreased, air is trapped in the airways and the alveoli hyperinflate.

A late-phase reaction is a delayed reaction that presents the same clinically as the early-phase reaction, only it occurs 4-8 hours after the exposure.

Clinical Manifestations

Dyspnea (shortness of breath)
Increased respiratory effort - accessory muscle use, nasal flaring
Expiratory wheezes
Coughing
Cyanosis (late sign)

How is Asthma Diagnosed?

Asthma is diagnosed based on several different things: spirometry results, the patient's clinical manifestations, chest x-ray, IgE blood tests, allergy tests and CBC with differential.

- Spirometry is a pulmonary function test that measures how much air moves in and out after the patient has taken a deep breath. A patient with asthma will have
  - decreased PEFR (peak expiratory flow rate)
  - decreased FEV (forced expiratory volume)
  - decreased FVC (forced vital capacity)
  - increased FRC (functional residual capacity)
  - increased TLC (total lung capacity)
  - increased RV (residual volume).
  - Note that the increase in FRC, TLC and RV is due to air being trapped in the lungs.
- A chest x-ray could show areas of hyperinflation and is often used to monitor the progress of the disease.
• IgE blood tests will show a rise in IgE levels when the patient is having an allergic reaction.
• Allergy tests can pinpoint what triggers the asthma attack.
• CBC with differential will show increased eosinophils in allergic reactions.

Asthma Classifications
Asthma is classified by the severity and frequency of symptoms.
• Mild intermittent: patient has symptoms < 2 times per week.
• Mild persistent: patient has symptoms more than 2x per week, but never more than once per day.
• Moderate persistent: patient has symptoms daily and activity is definitely affected.
• Severe persistent: patient has symptoms continually and frequent attacks.
• Status asthmaticus: patient is having a severe, life-threatening reaction that is not responding to medication.

Asthma Treatment
Asthma treatment is based on preventing exacerbations and reversing airway spasm when it occurs. Effective treatment enables the patient to maintain normal activity levels, normal lung function with as few side effects from the medication as possible. Sounds easy, right? Actually, it’s pretty straightforward, which is great because asthma medication administration is one of those things that require really good teaching. So how do we do it? Basically, it involves a two-pronged approach: reversing the spasm and controlling inflammation. We do this with a variety of medications:

Reversing the Spasm: Beta Agonists - Beta agonists stimulate the beta adrenergic receptors, dilating the airways (Albuterol).

Reversing the Spasm: Nebulized Atropine - Atropine is an anticholinergic that blocks the parasympathetic system.

Controlling Inflammation: IV Steroids - Used to decrease inflammation

Controlling Inflammation: Inhaled Corticosteroids - Prevents mast cells from emptying, reduces edema and spasm (Fluticasone, Flovent, Pulmicort).

Controlling Inflammation: Mast Cell Stabilizers - Suppress the release of bronchoconstrictive substances (Cromolyn)

Controlling Inflammation: Leukotriene Modifiers - Blocks the action of leukotrienes (Singulair)

Typical Medication Regimen for Asthma Patients
Asthma patients will usually have more than one type of inhaler to use at home. It is important that the patient understand when to use each type AND when to seek medical care. In general terms, the regimen consists of a long-term medication and one that provides immediate relief when the condition flares up.

DAILY: Long-acting medications such as leukotriene modifiers (Singulair), cromolyn sodium, inhaled corticosteroids (Symbicort, Flovent, Qvar) and combo drugs like Advair that consist of a corticosteroid and long-acting bronchodilator. These meds help keep the inflammation that causes asthma attacks under control.

QUICK RELIEF: When an asthma attack strikes, the drugs to reach for are short-acting beta 2 agonists (Albuterol, Xopenex, Terbutaline) and anticholinergics (Atrovent).

How do beta 2 agonists work? Great question! They work by relaxing smooth muscle that lines the airways and the effects typically last for about 3-6 hours. Note that these meds do NOT decrease inflammation.

Important Note!
The absence of wheezes in a patient actively having an asthma attack is a VERY BAD SIGN! It means the airways are so constricted NO air is moving. This patient needs help STAT!
When anticholinergics are used, it is typically in conjunction with a beta 2 agonist as they do not work as quickly.

Note that if your patient has exercise-induced asthma, they may take their quick-relief medications prior to any activity.

**Nursing Care of the Patient with Asthma**
As always, nursing care begins with assessment. You are watching to see how short of breath your patient is and how their lungs sound. You can ask them to rate their shortness-of-breath on a scale of 0-10. Another way is by evaluating if they are speaking in full or partial sentences (2-3 word sentences indicate pretty significant shortness of breath). Listen to their lungs... do you hear wheezes?

You’ll also want to get a medical history on these patients. What meds do they take? What allergies do they have? What were they doing when the attack occurred? What has triggered attacks in the past?

**Nursing Diagnoses and Interventions for Asthma**

**Ineffective breathing pattern**
- Nebulizer treatment
- Possibly addition of heliox ([read about it here](#))
- Oxygen
- Position of comfort (semi-fowlers)

**Impaired gas exchange**
- Oxygen
- Monitor lung sounds and SpO2
- Assess skin signs; watch for cyanosis

**Ineffective airway clearance**
- Suction and oral care
- Culture sputum as needed
- Encourage fluids to help thin secretions
- Sit upright if able

**Risk for anxiety r/t air hunger**
- Encourage pt to stay calm
- Provide a reassuring presence
- Oxygen
- Convey assessment findings to pt; explain plan of care

**COPD - Chronic Obstructive Pulmonary Disease**
COPD is a combination of disorders that affect the movement of air in and out of the lungs: asthma, emphysema and chronic bronchitis. Current thinking states that it is caused by long-term exposure to particulate matter and irritants, mainly from cigarette smoke. We’ve already looked at asthma, so let’s look at these two other conditions in a little more detail.

**Emphysema**
In emphysema, the walls of the alveoli and the space between the walls are destroyed. The patient ends up with permanent over-inflation, obstructed air

**The 50-50 Club**
You might hear another nurse or a respiratory therapist state that your patient is in the “50-50 Club.”

This means that on their ABG, the PaO2 is around 50 (normal is 80-100) and the CO2 is also around 50. (normal is 35-45).

This is due to chronic hypoxemia and hypercapnia. You’ll also note on the ABG that the HCO3 will also be elevated as a way to compensate... so as long as your pH is within normal limits, your ABG will typically show values consistent with compensated respiratory acidosis. You’ll learn all about ABGs in advanced M/S :-)
passages, loss of recoil, increased ventilatory dead space, bleb formation and partial airway collapse. In other words, it’s not good.

Your patient with emphysema will show the following:
- Progressive dyspnea on exertion
- Increased AP diameter of the chest (barrel chest)
- Hyper-resonance of chest when percussed
- Over-inflation and flattened diaphragm on CXR
- Enlarged heart and right ventricle
- Cyanosis and/or clubbed fingers
- Peripheral pitting edema due to increased pressures in the lungs and fluid backing up into the periphery

**Chronic Bronchitis**
Chronic bronchitis involves inflammation of the airways combined with increased mucus production, which makes it harder for air to travel effectively into and out of the lungs. The main cause is...wait for it...cigarettes.

Your patient with chronic bronchitis will have the following signs/symptoms:
- Shortness of breath
- Productive cough and copious sputum
- Exercise intolerance
- Prolonged expiration and wheezing
- Hypoxemia & hypercapnia
- Frequent lung infections

**COPD Complications**
Your patient with COPD will have numerous complications and likely battle frequent lung infections that result in acute respiratory failure requiring hospitalization and possibly even a stay in the ICU. Because of bleb formation and the hyperinflated state of the lungs, note that spontaneous pneumothorax can occur.

It is also important to note that COPD patients often have high anxiety. Imagine always feeling like you’re not getting enough air. When they are having an exacerbation, their anxiety is sometimes just as much of an issue as their airway (ok, not quite...but you get the idea.) Now add to that the fact that hypoxia causes agitation, so you could have a pretty “active” patient. However, what usually happens is the patient will be anxious and agitated during the hypoxic phase, then become hypercapnic as their CO2 rises, at which point they become somnolent. Providing a calm, reassuring presence will help immensely!

**Care of the COPD patient**
Let’s say you have a COPD patient (a member of the “50-50” club) who comes in through your ED and is admitted to your unit. The medical team is going to prescribe interventions to improve ventilation, remove secretions and prevent complications.

- **Improve ventilation**: bronchodilators, anticholinergics, corticosteroids, theophylline (old school treatment) and oxygen (often through BiPAP if CO2 is dangerously high). Many times the patient will not want to wear BiPAP as it can be uncomfortable, and having anxiety or hypoxia-induced agitation will make it worse. If your patient is refusing BiPAP and becomes somnolent, it’s because their CO2 is now too high...get that BiPAP on while you have the chance! When their CO2 comes down, they’ll wake up, and possibly take the BiPAP off again.
- **Remove secretions**: bronchodilators, pulmonary hygiene, chest physiotherapy and positioning. Basically the patient is going to get medications/treatments that loosen up the secretions and promote coughing all that gunk up.
- **Preventing complications**: breathing exercises and pursed-lip breathing, treat edema, prevent infection, oxygen as needed.
- **DON’T WITHHOLD OXYGEN!** You have learned by now that giving a COPD patient TOO MUCH oxygen can
depress their respiratory drive. That’s because the patient with COPD will be driven to breathe by low O2 levels, not rising CO2 levels (which is what a “healthy” patient does). So, if you give too much oxygen, this can “wipe out” the COPD patient’s respiratory drive. HOWEVER, this does not mean you WITHHOLD OXYGEN. What you do want to do, is give oxygen VERY conservatively. Even small increases in FIO2 can have dramatic impact on your COPD patient, so give as little oxygen as you need to in order to achieve an O2 saturation of typically around 87-88% (the MD will specify).

**Nursing Diagnoses and Interventions for COPD**

**Ineffective airway clearance**

- Administer medications to loosen secretions
- Encourage fluids to loosen secretions
- Pulmonary hygiene, cough/deep breathe
- High-fowler’s position

**Impaired gas exchange**

- Administer bronchodilators
- Low-flow oxygen or BiPAP as needed
  - BiPAP safety tips - Patient must be able to remove BiPAP mask on their own; patient cannot be restrained! Avoid BiPAP if patient is nauseous. If the patient vomits while wearing BiPAP it will just force the emesis down their airway!!
- Monitor pulse oximetry
- Monitor lung sounds

**Anxiety**

- Provide reassuring presence (if you stay calm it will help the patient stay calm)
- Teach effective breathing techniques such as pursed-lip breathing (assess readiness for learning first!)
- Explain care; encourage questions

**Activity intolerance**

- Provide rest periods
- Cluster care
- Monitor for increased respiratory effort and decreased oxygen saturation levels with exertion

**Imbalanced nutrition, less than body requirements**

- Small, frequent, high-calorie meals
- Encourage nutritional intake
- Instruct patient to take rest periods while eating

**Pneumonia**

One of the most common medical diagnoses you’ll see in Med/Surg or a Medical ICU is pneumonia. Pneumonia is an inflammatory process in the lung parenchyma which results in increased fluid in the interstitial space and the alveoli. It can be caused by bacteria, fungi, protozoa, viruses and aspiration of a foreign substance (typically emesis or food). Risk factors include smoking (or a history of smoking), lung cancer, COPD, upper respiratory infections, immobility, intubation and being of advanced age.

**Pneumonia Pathophysiology**

The first step in pneumonia is invasion by the offending agent (let’s say it’s a virus). The lungs then initiate an inflammatory response which disrupts the ability to cough effectively while reducing ciliary motility. In addition, the inflamed alveolar sacs
(which, as you recall, are full of fluid) can’t participate in normal gas exchange. And, to top it all off, the exudate in the alveoli consolidate which further impairs gas exchange.

---

### Signs and Symptoms of Pneumonia

- Fever
- Chills & sweats
- Chest pain with breathing
- Headache
- Productive cough
- Hemoptysis
- Dyspnea
- Fatigue

---

### Care of the Pneumonia Patient

Depending on the cause of the pneumonia, the patient will receive antibiotics, antifungals, or antiviral medications. Most patients who are hospitalized with pneumonia are going to need some kind of oxygen support. This could be as simple as a nasal cannula or as intense as a mechanical ventilator. This patient will also receive nebulized bronchodilators and you’ll want to keep their fluid status and electrolytes optimized. Patients with a weak cough may require nasotracheal suctioning, and intubated patients will need suctioning to help keep the lungs clear.

### Nursing Diagnoses and Interventions for Pneumonia

**Ineffective airway clearance**

- Administer medications to loosen secretions
- Encourage fluids to loosen secretions
- Pulmonary hygiene, cough/deep breathe
- High-fowler’s position

**Ineffective breathing pattern**

- Teach patient to splint chest wall when coughing to help reduce pain
- Monitor for increased WOB, SOB and dropping O2 sats

**Activity intolerance**

- Provide rest periods
- Cluster care
- Monitor for increased respiratory effort and decreased O2 sat with exertion
- Gradually increase activity as patient’s respiratory function improves

---

If you’ve got a patient who’s AT RISK for pneumonia, there are plenty of things you can do to help prevent it. It’s actually one of those hospital acquired conditions that we try really hard to avoid. Here’s what you can do:

- Good hand hygiene
- Encourage fluid intake to help keep the patient’s secretions loose so they can cough them up effectively
- Reposition every 2 hours
- Facilitate coughing/deep breathing (this often means controlling pain!)
- Reduce aspiration risk (upright position for eating/drinking). If your patient’s ability to swallow is questionable, get a swallow evaluation!
- Administer the pneumonia vaccine

If your patient is on a ventilator, you’ll be doing a collection of interventions to reduce ventilator associated pneumonia (also simply referred to as VAP). Note that the official VAP bundle is actually: HOB 30-45 degrees, daily sedation vacation and assessment for readiness to extubate, peptic ulcer prophylaxis and DVT prophylaxis. The problem with this bundle is that peptic ulcer prophylaxis and DVT prophylaxis aren’t directly related to pneumonia prevention. However, the things you do to actually reduce VAP, are listed above in the blue box.
Atelectasis
A term you will hear a lot in your clinicals/courses is “atelectasis.” Atelectasis is a condition in which the lungs don’t fully expand and the alveoli are essentially collapsed. This is typically due to a lack of deep breathing (pain, immobility, respiratory-depressing drugs, mechanical ventilation, paralysis) or a blockage of the airway (think mucus plugs, foreign body aspiration and even lung cancer).

Signs/Symptoms of Atelectasis
• Mild to significantly low O2 sats
• Crackles upon deep inspiration (will sound like rice crispies cereal). This occurs when the patient FINALLY takes a deep breath and those little alveoli pop open and re-inflate.
• Low-grade fever (though this may not be supported by official studies, it seems to be the case some of the time!)

Treating Atelectasis
In most cases, atelectasis is going to be a result of surgery, pain or a lack of coughing & deep breathing. So, the simple treatment is to encourage coughing and deep breathing (the Incentive Spirometer is great for this!). If the patient is in pain after surgery, you’ll need to treat the pain in order to get the patient to cough/deep breathe.

If the cause is a tumor, then removing the tumor is the treatment. If it’s due to an inhaled peanut, then removing the peanut is the treatment. Get it? Basically you just want to figure out what’s causing it, and fix the underlying problem. But, for the most part, atelectasis is going to be caused by a failure to cough and take deep breaths. Find out WHY the patient isn’t taking deep breaths and go from there.

Respiratory Disorders - The Upper Airway
• Strep
• Epiglottitis
• Rhinitis
• Sinus infection
• Common cold
• Influenza

Disorders of the upper airway rarely cause hospitalization so we won’t go into detail about them here. However, as with all things in nursing, there are a couple of exceptions: strep and influenza. Strep, in and of itself doesn’t usually cause someone to be so ill they need to be hospitalized. But a complication of strep, epiglottitis, does. You’ll learn loads about epiglottitis in your pediatric course because it’s MUCH more common in kids. Here’s a quick snapshot of what you need to know now:

Epiglottitis Snapshot
Epiglottitis is a swelling of the epiglottis which, if it gets big enough, will completely occlude the airway. So yes, it’s pretty scary! The patient with epiglottitis will present with the following signs & symptoms: hoarse or muffled voice, inability to swallow, drooling (refer back to “inability to swallow”), and sore throat.

The very FIRST thing you want to do to treat epiglottitis is to avoid further inflammation! Encourage the patient not to talk and allow them to assume any posture or position that facilitates breathing. You will also:
• Administer steroids to decrease inflammation
• Take a strep culture if it’s safe to do so (irritating the throat with a culture swab could cause more swelling!) If it were
epiglottitis cont’d

me, I’d ask the MD to do it, or at least be standing at the bedside while I’m swabbing the throat, in case the patient
gets into trouble.
• Obtain blood cultures
• Start antibiotics just in case the cause is bacterial
• Oxygen if needed
• And, most importantly, get an emergency tracheostomy kit and have it nearby. If the airway occludes, you won’t be
able to intubate this patient. They will have to get an emergency tracheostomy.
• Try to stay calm and promote a calm environment. If this means kicking out rowdy visitors, do it.

Influenza
Usually when people get the flu, they feel awful and then recover over the coarse of a few days. Common signs/symptoms of
the flu are: sudden onset of fever/chills, feeling tired, headache, cough, runny nose and possibly a hoarse voice.

When influenza gets serious, it typically turns into pneumonia, and if that gets even MORE serious, it turns into sepsis. The
takeaway here is that the flu can be fatal, even if otherwise young, healthy individuals.

The Flu Vaccine
The flu is actually a result of three types of viruses: A, B and C. The Type A flu is the most common form and is typically
what hits us every year during “flu season” (in the U.S. this is October to May.) However, it is important to know that this Type
A flu changes a bit every three years or so, which causes higher outbreaks of flu that we’re essentially “not ready for” (mean-
ing our vaccine wasn’t geared toward it yet). Type B also comes around each year, but it only causes big problems every five
years or so. And Type C is pretty much no big deal. It only causes a few cases so we don’t worry about it too much.

Each year, researchers analyze the available data and develop a vaccine to the flu strains they expect to be most prevalent
in a given area. The vaccine causes the body to produce antibodies to the flu virus, but this takes about two weeks. So, to all
those people who say, “I got the flu vaccine and was sick the next day with the flu,” please explain to them that this does not
mean the flu vaccine doesn’t work. It simply means they were exposed BEFORE their body had time to produce antibodies.

The vaccine is available in both injection and inhalation formats. The injection formats can be either inactivated or recombinant
vaccines) and the inhalation type is a live attenuated vaccine.

Who gets the flu vaccine?
Well, if you’re an RN working with the public you’ll likely “have” to get the flu vaccine or have to wear a mask the entire flu
season. Most people choose to get the vaccine vs wear the mask. Who else? Basically all individuals six months and older
(except for those for whom it is contraindicated).

Who shouldn’t get the flu vaccine?
• Anyone with a life-threatening allergy to the flu vaccine or any of its ingredients.

Who should MAYBE not get the flu vaccine?
• Individuals with an egg allergy (options are available)
• Anyone with a history of Guillain-Barre (a truly awful disease)

For more information about the flu, the [CDC website](https://www.cdc.gov) has a lot of really great information.
Functions of the Kidney
- Eliminate toxins and waste
- Maintain blood pressure (in response to Aldosterone and ADH. Think of the whole renin - Ang II pathway)
- Stimulate RBC production. Remember that the kidneys sense a drop in hemoglobin & hematocrit and this stimulates the release of erythropoietin.
- Activate Vitamin D
- Regulate acid-base and electrolyte balance

Failing kidneys can lead to high BP, because they are not able to do their job in regards to the maintenance of blood pressure. Also, if your patient’s kidneys don’t work well, you will need to adjust their dosages and meds, and you’ll have to watch fluid intake too! Lots to keep an eye on with these guys.

Diagnostics: KUB and CT
A KUB is an x-ray of the kidneys, ureter, bladder. A CT-scan shows cross-sections of the anatomy and, in some cases involves the use of contrast dye that could ultimately cause kidney failure. Note that you’ll often see a KUB ordered for feeding tube placement. Though the x-ray is of the kidneys, ureter and bladder, this image will also show where the feeding tube is. For this reason it is often used to verify position.

Random Kidney Facts to Amaze Your Friends
- H+ is secreted in exchange for bicarb. This will come in handy later when you get deep into acid/base balance.
- When the bladder is full of urine it backs up to the loop of Henle.
- We have about 2 million nephrons.
- AAA (abdominal aortic aneurysm) can block off the blood supply to the renal artery... that's bad news.
- The afferent arterioles go IN to the kidney, the efferent arterioles go OUT.
- The peritubular capillaries provide nourishment and oxygen to the kidneys. Problems with blood supply (either due to low BP or obstruction at the glomerulus) lead to oxygenation issues of the tubule and you get renal failure.
- The glomerulus is a high-pressure capillary system that pushes fluid into the filtrate. The higher the pressure exerted at the efferent arteriole, the higher the pressure in the glomerulus. This leads to MORE filtrate.
- If the filtrate has protein and blood cells in it, this is more bad news for your patient and indicates a problem with the glomerulus.
- Each glomerulus filters 0.1 ml which equates to 180 L per day with all of them working together.
- The capillaries here are 25x more permeable than systemic capillaries, and they have a huge surface area.

Kidney Anatomy & Physiology Review
The PCT (Proximal Convoluted Tubule)
This is the workhorse of the kidney. 60-80% of the “stuff” that is absorbed by the kidneys is absorbed at the PCT. If you give your patient an osmotic diuretic (such as glucose or mannitol), it will prevent reabsorption of Na in the PCT, so watch those Na levels! As for glucose, it is reabsorbed on a transport system which is fully loaded at blood sugar levels of about 250, so anything above that is spilled into the urine. In the old days, they used to monitor glucose with urine testing, but that definitely is not very accurate compared to the techniques available today.

The Loop of Henle
The main thing you need to know about the Loop of Henle has to do with how diuretics affect the loop and what this does
for your patient’s electrolyte levels. Note that diuretics that work on the loop are POTENT! 25% of Na is absorbed here, so keep that in mind if your patient is on a loop diuretic. You’ll also want to monitor potassium levels, as loop diuretics are NOT potassium-sparing and your patient can easily develop hypokalemia. The main one you’ll see is Lasix/furosemide.

**The Distal Tubule:**
This is where your potassium-sparing diuretics are going to work. 3-5% of Na is reabsorbed here, and Na is exchanged for K under the influence of aldosterone. The MAIN thing to know here is that it’s where potassium-sparing diuretics like Aldactone/spironolactone do their magic. These type of diuretics will not cause the drastic drop in K that you can see with Loop diuretics. However, you do need to monitor for hyperkalemia, especially in patients with renal dysfunction.

**The Collecting Duct:**
If the pores are open (ADH opens those pores!), then water moves from the dilute tubule fluid into the concentrated interstitial space. Urine becomes smaller in volume and gets as concentrated as the interstitial space.

**Misc A&P Things to Know:**
- Peristalsis moves urine forward along the ureters (don’t want it backing up into kidneys!)
- The ureters are innervated, so it’s very painful to have a kidney stone.
- Valves in the bladder do their best to keep the urine from backing up into kidneys
- The bladder holds 1000-1800 mls
- Voiding occurs at around 200-400 mls, and hopefully it completely empties. If not, this is called urinary retention and it leads to UTIs.
- Flushing (via voiding) removes bacteria, and macrophages search out any that are remaining.

**PRO TIP!** Patients generally don’t love taking diuretics because urinating all the time is a pain in the neck. If a patient’s urine is dilute, then they probably took their diuretic. If it’s amber-colored and they tell you they took their Lasix…they are trying to pull a fast one!

**Comparison of Diuretics**

- **With no diuretic:** The tubules do their thing as outlined above
- **With furosemide:** Lasix/furosemide blocks loop reabsorption of Na, and more Na gets in to the distal tubule for exchange with K. As a result the patient loses K.
- **With spironolactone:** Aldactone/spironolactone inhibits aldosterone, so there is no exchange of K for Na in the tubule fluid. It is not as potent of a diuretic, but it does spare K.

**Urinary Tract Infections**
The very first treatment for a UTI is prevention! Catheterization always carries with it a risk of a UTI, so we need to do as much as we can to keep this from happening. UTIs are painful, can cause sepsis, and cost the hospital a lot of money.
- Some catheters are impregnated with antibacterial substances.
- Remove the cath as soon as you can. If there is EVER another option, you will always want to go with that option as opposed to leaving in a catheter or inserting one in the first place. External collection devices are great options for both men (condom catheters) and women (PureWick™).
- In & out catheters have less risk of infection, so do those if you can (AKA “I/O” or a “straight cath”).
- No urine output or decreased output? Pt complaining of bladder fullness after voiding? Bladder scanning shows you if they have any retained urine. If the bladder is full and they are unable to void, you’ll need to use a catheter to drain the bladder. If they’ve just voided and the bladder is not empty, they are retaining urine. If there’s nothing in there, then the patient has other problems that need to be addressed (dehydration, renal failure...
come to mind). Sometimes the patient will have a catheter in place and still complain of bladder fullness, or you'll see very little output. Occasionally the catheters will be clogged or kinked. Before you panic and think your patient is going into acute renal failure, assess the catheter for patency and flush with sterile saline if necessary to dislodge mucus, sediment or a blood clot.

Things that cause a UTI besides catheterization are:

- Obstruction: For example, patients with BPH essentially have an obstruction caused by the prostate
- Hypotonic bladder: Bladder doesn’t contract adequately
- Female: Due to the short urethra and proximity to anus.
- Older patients
- Immune suppressed individuals
- Uncircumcised patients: Keep things clean, just be sure to put foreskin back where it belongs!
- Poor hygiene/sexual activity
- Some medications can even put your patient at risk for UTI, so be sure to ask about home meds!

FYI: When your patient’s urine is positive for leukocyte esterase then they have a UTI. Cystitis (inflammation of the bladder) can be asymptomatic, but is typically painful. If the infection goes to the kidneys, then you'll have systemic effects such as fever and increased WBC.

**The Micturition Reflex**

As the bladder fills with urine it will stretch and send a message to the brain. The brain, however, is more patient than the bladder and says "wait!" So, the bladder waits and fills some more. It sends another message to the brain which still says “wait!” So the bladder waits some more. Finally, the brain decides to listen to the bladder (the brain must be really stubborn!) and the brain activates the PNS which causes the bladder to contract, and blocks the SNS which relaxes the sphincter and urination occurs.

But what if this whole system isn’t working the way it should?

- **Neurogenic bladder** can be either hyper-reflexive or hyporeflexive.
- **Hyper-reflexive bladder** often occurs with spinal cord injury and it involves the patient having NO CONTROL of their bladder contraction. The contraction is not coordinated with the relaxation of the sphincter and the bladder does not completely empty. There will be a small bladder volume and reflux of urine back up to the kidney, which can lead to pyelonephritis. Check for residual after the patient voids to check for residual.
- **Hyporeflexive bladder** happens when there is damage to the nerve and the messages do not go where they are supposed to go. The bladder fills and does not empty (ouch!). There will be a large bladder volume and reflux back up to the kidneys, plus a large amount of residual (use that bladder scanner!)

**Incontinence**

There are several types of incontinence:

- **Stress**: weakened muscles and support structure d/t multiple pregnancies, jogging and obesity
- **Urge**: this is caused by infection and irritable bladder
- **Overflow**: this is the result of hypotonic bladder
- **Functional**: this is the result of not being able to get to the bathroom...maybe the person is in restraints, or has a physical limitation

The treatments for incontinence are:

- Strengthen muscles and tighten the sphincters by doing Kegals (50/day), electric stimulation, implants and surgery.
• Timed voiding. This trains the bladder and ensures someone doesn’t get so full that they have to rush to the bathroom and possibly not make it (urgency is also a HUGE risk factor for falls, especially in the elderly).

• Medications
  • Oxybutynin (Ditropan): decreases contractility
  • Bethanechol (Urecholine): increases tone
  • Antibiotics
  • B+O (Belladonna and Opium): antispasmodic
  • Phenazopyridine (Pyridium): analgesic; note that it turns the urine bright orange

• Pads, external catheters and I/O catheterizations are also another treatment. The goal is to avoid Foley catheterization as much as possible. Incontinence in and of itself is not a reason to put in a Foley.

**Prerenal Failure**

**Pathophysiology of Prerenal Failure**

When we talk about renal failure being “prerenal” we are talking about WHERE the problem occurs. In this case, the problem occurs BEFORE the kidneys (as opposed to within the kidney or in the ureter). In pre-renal failure, the kidney system works normally but is excreting a small volume of concentrated urine. Low blood flow leads to decreased GFR leading to decreased urinary output. Decreased urinary output leads to increased concentration, decreases the amount of urine sodium, and leads to an increased BUN and creatinine. The most common etiology of this is a low blood pressure or low cardiac output, which is essentially “before” the kidney, so it’s called PRErenal failure.

Some causes of low blood pressure or low cardiac output are dehydration, heart failure, sepsis and hemorrhage. You can also see it in cases where the patient has low blood flow due to atherosclerosis or chronic liver disease.

**Signs & Symptoms of Prerenal Failure**

• Dizziness
• Dry mouth
• Low blood pressure (hypotension)
• Rapid heart rate
• Slack skin, decreased skin turgor
• Thirst
• Weight loss
• Urine output is usually low in people with prerenal ARF (acute renal failure)
• The patient also may have symptoms of heart or liver disease

**Prerenal ARF Complications**

Many prerenal ARF patients are critically ill and require admission to an intensive care unit. They may suffer from severe infection, such as sepsis. Decreased perfusion can cause acute organ failure, such as cardiac or liver failure.

Symptoms of heart failure include:

• Dyspnea (shortness of breath)
• Edema (fluid retention and swelling)
• Venous engorgement

Symptoms of liver failure include:

• Confusion, disorientation, stupor
• Sweet, ammonia odor
• Ascites
• Yellow sclera and jaundice
• Shortness of breath (due to ascites)

**Prerenal ARF Diagnosis**
A complete physical examination and a medical history help the physician diagnose prerenal ARF. In addition, laboratory studies often reveal a high BUN to Cr ratio (BUN:Cr > 20:1), along with abnormal urine chemistry.

**Prerenal ARF Treatment**
The goal of treatment is to improve kidney perfusion (blood circulation). This usually involves treating the underlying condition (e.g., infection, heart failure, hypotension, liver failure). Intravenous fluids are administered to most patients to treat dehydration and improve kidney perfusion. You will sometimes hear the term “fluid challenge.” What this means is you will give your patient a fluid bolus to see if it raises their blood pressure (and thus their urine output). Depending on the patient, the amount will range from 250ml to 1-2 liters. Patients with heart failure typically can only handle small amounts of fluids, while most will be able to handle a liter with no problems. If the patient “fails” the challenge, this could mean they need vasoactive medications, which you will learn about in your advanced Med/Surg class.

**Prerenal ARF Prognosis**
In general, patients with prerenal ARF improve dramatically with intravenous fluids. Urine output increases and renal function improves as the extra fluids raise the blood pressure.

**Intrarenal Failure: Glomerulonephritis**
Because the problem occurs WITHIN the kidney, glomerulonephritis is considered a type of INTRArenal failure. Glomerulonephritis can occur due to Ab-Ag complexes after a patient has strep throat, an autoimmune disease such as Goodpasture’s or lupus, and diabetes. It is a type of kidney disease that damages the kidneys’ ability to remove waste and excess fluids. Also called glomerular disease, glomerulonephritis can be acute or chronic. If it occurs on its own, it’s known as primary glomerulonephritis. If another disease, such as lupus or diabetes is the cause, it’s called secondary glomerulonephritis. Treatment depends on the type of glomerulonephritis the patient has.

**Signs & Symptoms of Glomerulonephritis**
Signs and symptoms of glomerulonephritis may depend on whether you have the acute or chronic form, and its cause. Your first indication that something is wrong may come from symptoms or from the results of a routine urinalysis. Signs and symptoms may include:

- **Cola-colored or diluted, iced-tea-colored urine** from hemolysed red blood cells in the urine
- **Foamy urine** due to excess protein (proteinuria)
- **High blood pressure** (hypertension)
- **Fluid retention** (edema) with swelling evident in the face, hands, feet and abdomen
- **Fatigue from anemia or kidney failure**
- **Less frequent urination** than usual

*Cola or Tea-Colored Urine + Foam + Hypertension = Glomerulonephritis!*

**Physiology of Glomerulonephritis**
Each of the kidneys contains approximately 1 million tiny filters (glomeruli), which attach to the opening of a small fluid-collecting tubule. Each glomerulus and tubule form a nephron, which is the functional unit of the kidneys. The glomeruli filter the blood as it passes through the kidneys, and the filtered blood returns to the bloodstream. The tubules modify what the glomeruli filter by saving needed substances, such as protein. The waste goes to the bladder through a tube from each kidney.
(the ureter) and passes out of the body when you urinate. Glomerulonephritis — an inflammation of the glomeruli — can damage the kidneys so that they lose their filtering ability, allowing dangerous levels of fluid and waste to accumulate in the body. Often the cause of glomerulonephritis is unknown. Known causes include:

- **Infections**
  - Post-streptococcal glomerulonephritis: Glomerulonephritis may develop after a strep infection in the throat or, rarely, on the skin (impetigo). Post-infectious glomerulonephritis is becoming less common in the United States, most likely because of rapid and complete antibiotic treatment of most streptococcal infections.
  - Bacterial endocarditis: Bacteria can occasionally spread through the bloodstream and lodge in the heart, causing an infection of one or more of the heart valves. Those at greatest risk are people with a heart defect, such as a damaged or artificial heart valve. IV drug users are also at high risk for endocarditis.
  - Viral infections: Among the viral infections that may trigger glomerulonephritis are HIV, and the hepatitis B and hepatitis C viruses, which primarily affect the liver.

- **Immune diseases**
  - Lupus: A chronic inflammatory disease, lupus can affect many parts of the body, including the skin, joints, kidneys, blood cells, heart and lungs.
  - Goodpasture’s syndrome: A rare immune lung disorder that may mimic pneumonia, Goodpasture’s syndrome causes bleeding (hemorrhage) into the lungs as well as glomerulonephritis.
  - IgA nephropathy: Characterized by recurrent episodes of blood in the urine, this primary glomerular disease results from deposits of immunoglobulin A (IgA) in the glomeruli. IgA nephropathy can progress for years with no noticeable symptoms. The disorder seems to be more common in men than in women.
  - Polyarteritis: This form of vasculitis affects small and medium blood vessels in many parts of the body, such as the heart, kidneys and intestines.
  - Wegener’s granulomatosis: This form of vasculitis affects small and medium blood vessels in the lungs, upper airways and kidneys.

- **Conditions that cause scarring of the glomeruli**
  - High blood pressure: Damage to the kidneys and their ability to perform their normal functions can occur as a result of high blood pressure. Glomerulonephritis can also cause high blood pressure because it reduces kidney function, so it's a vicious cycle.
  - Diabetic kidney disease: Diabetic kidney disease (diabetic nephropathy) can affect anyone with diabetes, and typically takes years to develop. Good control of blood sugar levels and blood pressure may prevent or slow kidney damage.
  - Focal segmental glomerulosclerosis: Characterized by scattered scarring of some of the glomeruli, this condition may result from another disease or occur for no known reason.

**Chronic glomerulonephritis** sometimes develops after a bout of acute glomerulonephritis. In some people there’s no history of kidney disease, so the first indication of chronic glomerulonephritis is chronic kidney failure. Infrequently, you'll find that chronic glomerulonephritis runs in families. One inherited form, Alport syndrome, may also involve hearing or vision impairment.

**Complications of Glomerulonephritis**

- **Acute kidney failure:** Loss of function in the filtering part of the nephron may cause waste products to accumulate too rapidly. The patient will need emergency dialysis.
- **Chronic kidney failure:** This is a VERY serious complication where the kidneys lose function over time. Kidney function at less than 10% of normal capacity indicates end-stage kidney disease, which requires dialysis or kidney transplant.
- **High blood pressure:** Damage to the kidneys and the resulting buildup of wastes in the bloodstream can raise BP.
Treatments and Meds for Glomerulonephritis
Treatment varies based on whether it’s the acute or chronic form of the disease. Some cases of acute glomerulonephritis (especially those that follow strep), can improve on their own. Other treatments include:
• Controlling high blood pressure
• Control salt intake
• Diuretics
• ACE inhibitors
• ARBs (angiotensin receptor blocker)
• Treat the underlying cause
• Antibiotics to treat strep or bacterial infection
• Corticosteroids and immune-suppressing drugs for lupus or vasculitis
• Fish oil supplements for IgA nephropathy (if you’re interested in autoimmune, look this one up!)
• Plasmapheresis sometimes used to treat Goodpasture’s
• Treat kidney failure
• Temporary dialysis may be needed for acute forms of the disease
• The only long-term therapies are dialysis and transplant

Intrarenal Failure: Nephrotic Syndrome
Nephrotic syndrome is another type of intrarenal failure. It is a disorder caused by damage to the small blood vessels in the kidneys that filter waste and excess water from the blood. When healthy, these small vessels keep protein from seeping into the urine and out of the body. When damaged, they don’t do this very well and protein leaks out of the blood and can lead to edema. Nephrotic syndrome can increase the patient’s risk for infections and blood clots, so be watchful!

Signs & Symptoms of Nephrotic Syndrome
• Edema, particularly around eyes, ankles, feet
• Foamy urine d/t protein in the urine
• Weight gain d/t excess fluid retention
• Loss of appetite and/or vomiting
• Low levels of serum albumin and elevated triglycerides and cholesterol

Foamy Urine + Edema = Nephrotic Syndrome!

Causes of Nephrotic Syndrome
Nephrotic syndrome is caused by damage to the tiny blood vessels (glomeruli) of the kidneys. Many disorders can cause glomerular damage and lead to nephrotic syndrome.
• Minimal change disease: The most common cause of nephrotic syndrome in children. The cause of the abnormal function typically can’t be determined.
• Focal segmental glomerulosclerosis: Characterized by scattered scarring of some of the glomeruli, this condition may result from another disease, a genetic defect, or occur for no known reason.
• Membranous nephropathy: This kidney disorder is the result of thickening membranes within the glomeruli. The exact cause of the thickening isn’t known, but it’s sometimes associated with other medical conditions, such as hepatitis B, malaria, lupus and cancer.
• Diabetic kidney disease: Diabetes can lead to kidney damage (diabetic nephropathy) that affects the glomeruli, particularly in people with diabetes that’s poorly controlled or people who also have high blood pressure.
• Systemic lupus erythematosus: This chronic inflammatory disease can lead to serious kidney damage.
• Amyloidosis: This disorder occurs when substances called amyloid proteins accumulate in your organs. Amyloid buildup often affects the kidneys, damaging the filtering system.
What Happens in Nephrotic Syndrome?
Healthy glomeruli keep blood protein (mainly albumin) — which is needed to maintain the right amount of fluid in your body — from seeping into your urine. When damaged, glomeruli often lose this ability leading to the condition's hallmark symptom of proteinuria (protein in the urine). Without albumin contributing to oncotic pressure, third-spacing occurs and results in edema.

Complications of Nephrotic Syndrome
- Blood clots: The inability of the glomeruli to filter blood properly can lead to loss of blood proteins that help prevent clotting. This increases the risk of developing a blood clot (thrombus).
- High blood cholesterol and elevated blood triglycerides: When the level of albumin in the blood falls, the liver makes more albumin in response. At the same time, the liver releases more cholesterol and triglycerides. (critical thinking tip: what would happen in a patient who ALSO has liver disease?)
- Poor nutrition: Loss of too much blood protein can result in malnutrition. This can lead to weight loss, but it may be masked by edema, which causes weight gain.
- High blood pressure: Damage to the glomeruli and the resulting buildup of wastes in the bloodstream (uremia) can raise blood pressure.
- Acute kidney failure: If the kidneys lose their ability to filter blood due to damage to the glomeruli, waste products may build up quickly in the blood. If this happens, the pt may need emergency dialysis — an artificial means of removing extra fluids and waste from the blood — typically with an artificial kidney machine (dialyzer).
- Chronic kidney failure: Nephrotic syndrome may cause the kidneys to gradually lose their function over time. Kidney function at less than 10-15 percent of normal capacity is considered end-stage kidney disease, which usually requires dialysis or a kidney transplant.
- Infection: Although it’s not clear why, people who have nephrotic syndrome are at an increased risk of infection, such as pneumonia.

Intrarenal Failure: Tubular Dysfunction
Tubular dysfunction is caused by damage to the tubular cells, which causes the osmolarity of the urine to then equal the osmolarity of the plasma. Because there is no concentration gradient to pull fluid into the tubule, the result is oliguria. The patient will present with increased BUN/creatinine. Clinical causes of tubular dysfunction are nephrotoxins such as gentamicin, contrast dye (be careful of those CT scans!), ischemia (be watchful after ANY surgery where the renal arteries are clamped... such as AAA repairs), and pyelonephritis where back up of urine goes into the kidneys (this hits the long loops of Henle first).

Pyelonephritis is a common “intrarenal” disease of the kidney. It is associated with cystitis and reflux. The signs and symptoms are flank pain, fever, WBC/bacteria in urine. The predisposing factors are neurogenic bladder (hypotonic and hypertonic) and obstructive uropathies such as BPH (benign prostatic hyperplasia), a kinked catheter, foreign objects, tumors and stones.

Speaking of kinked catheters, if your patient has a Foley and you notice a marked decrease in urine output, check for kinks, flush with 30ml NS (toward and away from the bladder, so keep it sterile!) and reposition. If the urine backs up, they are at risk for pyelonephritis! Plus, it saves you an embarrassing call to the doc when the patient doesn’t actually have decreased urine output.

Postrenal Failure
Renal failure that occurs due to problems “after” the kidneys, is considered POSTrenal. The most common cause is some kind of anatomical problem in the ureter, a kidney stone that’s blocking the flow or, BPH (when the prostate grows too large and compresses the ureter).
As for kidney stones, the most common calculi (kidney stones) are calcium oxalate or calcium phosphate stones and magnesium stones (also called “struvite”). The stones are very painful to pass, but most can dissolve or pass with increased fluid intake and by altering the pH and electrolytes of body fluids/urine. You can also limit oxalate, Na, Ca and purines. If you have uric acid stones, then you’d alkalize urine to get rid of them by consuming a diet high in fruits and vegetables, drinking alkaline mineral water and taking citrate.

Foods high in purines include: organ meats, bacon, beef, pork, lamb, game meats, anchovies, sardines, herring, mackerel, scallops, gravy and beer.

The short version is meat, seafood, gravy and beer. Yum!

**Taking Care of Patients in Renal Failure**
Regardless of what CAUSED the renal failure, you are going to do pretty much the same things for every patient.

**Monitor I/O**
Monitoring urine output is very important when you’re dealing with a patient in renal failure. Note that renal failure doesn’t always mean the patient is going on dialysis. Many patients continue to produce urine, just probably not enough overall. What you are looking for is any sign that urine output is dropping off.

Normal urine output is 0.5ml/kg/hr. Yes, we’ve all heard that 30ml/hr is sufficient, but what if your patient is 6’4” and weighs 285 pounds? Well, his desired urine output is actually 64ml/hr. If your patient is a teeny tiny little gal who weighs 92 pounds, her urine output goal is actually closer to 21ml/hr. If your urine output is decreased for more than 3-4 hours, then this warrants a call to the doc, especially if it’s causing problems for the patient or you suspect something even scarier is going on (one of the hallmark signs of sepsis is decreased urine output!).

**Monitor Lung Sounds**
When the kidneys aren’t working properly, the body’s fluids aren’t expelled as they should be. In many cases, this fluid backs up into the lungs causing coarse, wet lung sounds and decreased oxygen saturation. If your patient’s lungs sound wet and their urine output is down, you should probably speak to the MD about diuretics.

**Monitor Electrolytes**
Because the kidneys filter some key electrolytes, you can expect these to be altered when renal function is impaired. The MOST important electrolyte you are going to monitor is potassium because of the potential for hyperkalemia to cause cardiac arrhythmias. If you are taking care of a renal patient who starts having runs of V-tach or shows tall, peaked T-waves on their ECG…check a K level and call the MD for a stat order of the hyperkalemia cocktail. This is a standard treatment given to patients with elevated potassium levels in order to get them down to the safes zone. It consists of insulin, glucose, Kayexalate and the inhaled medication, Albuterol.

How does this work exactly? For starters, the insulin opens the cell so that glucose can enter, and potassium “hitches a ride” on that sugar molecule, causing the serum levels of K to decrease. Kayexalate binds potassium in the GI tract (basically the patient poops it out). And, Albuterol decreases potassium by also shifting potassium into the cell. Another medication you’ll see given in symptomatic hyperkalemia is calcium gluconate. The calcium protects the myocardium from the elevated levels of potassium and reduces myocardial excitability, but note it does nothing to decrease the K level itself.

**Monitor ECG**
As you just read, hyperkalemia causes arrhythmias, elevated T waves and yes, even cardiac arrest. If your patient’s K levels are high, you’re going to make sure they are on a cardiac monitor at all times. Same if the patient has low potassium levels as well...monitor that ECG!
The immune system is complex with a lot of moving parts that work together to fight off infection or invasion by a foreign body. When this works well, it's fantastic! But, as you'll see further on, sometimes the immune response can go haywire, and this is where conditions like sepsis and autoimmune disease come into play. We'll talk about sepsis in Med/Surg 2.

**Innate Immunity vs. Acquired Immunity**

Innate immunity: does not require previous exposure to a pathogen

Acquired immunity: the body "learns" from previous exposure and "remembers" how to fight off a pathogen

**Cells of the Immune System**

It all starts with stem cells, which are those that can essentially form into any other type of cell. This makes it an "undifferentiated" cell, and it can form into a variety of different cells.

**White Blood Cells (AKA leukocytes)**

- **Neutrophils (40-75% of WBCs):** Most WBCs are neutrophils. They are the first to arrive in a bacterial infection, then they call in other cells to help through the process of chemotaxis. Note that neutrophils also activate complement, which is discussed here further on.

- **Lymphocytes (20-40% of WBCs):** Two main types = T-cells and B-cells
  1) T-cells are a type of lymphocyte that originates in the bone marrow and matures in the thymus. They differentiate into a few different type of T-cells, including:
    - Cytotoxic T-cells: fight off viruses and cancer; are key in organ transplant rejection
    - Helper T-cells: coordinate the immune response, in other words they "help!"
    - Regulatory/Suppressor T-cells: help modulate the immune response (though this doesn’t always work as in the case of autoimmune disease.)
    - Memory T-cells: when normal T-cells have learned how to fight off a pathogen, they become "memory T-cells". They play a key role in long-term immunity...think of chicken pox or mumps.
  2) B cells (make antibodies). B-Cells are trained in the bone marrow and each B-lymphocyte makes one shape of antibody. Memory B-cells circulate throughout the body and cause stronger, more rapid antibody responses when they detect an antigen that their parent cells had previously reacted to. This is why you might have a moderate reaction to shellfish when you have it in March, but have an anaphylactic reaction when you eat it again on your birthday in June. The response is faster and stronger with each subsequent exposure, thanks to memory B-cells.

- **Monocytes (2-10% of WBCs):** Produced in the bone marrow and migrate to tissues and mature into macrophages.
- **Eosinophils (1-6% of WBCs):** You’ll see eosinophils in parasitic infection and allergic responses.
- **Basophils (<1% of WBCs):** Similar to mast cells; they are key in immediate hypersensitivity reactions and release histamine as part of the allergic response.

When WBCs are immature, the nucleus is just a small band, so you will see these called “band cells” or just “bands.” If you see a lot of bands, then you know the infection is new. You will also hear this referred to as a “left shift.” This is a holdover term from the old days when lab reports were written by hand. Bands were notated on the left side of the reports, so it became known as a “shift to the left” indicating that the bands are elevated and the infection is new.

**Microphages and Macrophages**

Some of the WBCs are phagocytes, which means they are the warrior cells of the body. They are a key player in innate immunity and are the cells that surround, consume and essentially digest invading microbes through the process of phagocytosis (watch this amazing video [here](#)). There are two main types of phagocytes: microphages and macrophages.

- Microphages are circulated in the blood constantly and there are large reserves of them in the bone marrow. Neutrophils are considered microphages.
• Macrophages, on the other hand, are monocytes that have been called into action. Rather than circulating and always being “on the hunt” like microphages, they are located at strategic locations in the body such as the alveoli, intestines and liver (Kupffer cells). The body does not have large reserves of macrophages, but they do live longer than microphages.

**Megakaryocytes**
Megakaryocytes live in the bone marrow and are responsible for the production of platelets.

**The CBC Differential**
Though each facility/hospital/school will have slightly varying differences in what is considered a normal lab value, the following is a good general guide:
• RBC 3.9 -5.7
• Hgb (hemoglobin) 12-16
• Hct (hematocrit) 37-47%
• WBC 5,000-10,000
  • Neutrophils 40-60% (range from immature band cells to mature PMNs)
  • Lymphocytes 20-40%
  • Monocytes 4-8%
  • Eosinophils 1-3%
  • Basophils 0-1%
• Platelets 150,000 to 400,000

**What is an ANC (Absolute Neutrophil Count)?**
Because neutrophils are THE most important PMN for fighting off infection, we conduct what is called an “absolute neutrophil count” or an ANC. More than just a percentage of the neutrophils, this is an actual count. Normal values are about 2,500 to 7,000-ish.

**What to do when the ANC Is abnormal**
If the ANC is below 500, then the patient is at huge risk for infection. Your patient will be on neutropenic precautions, which includes a whole host of interventions:
• Use alcohol-based hand wash.
• Prohibit fresh flowers in the room because the standing water is a breeding ground for bacteria.
• Triple wash fruits and veggies IF they are allowed. Many hospitals will not serve a neutropenic patient ANY fresh or raw fruits or vegetables. Some will allow fruits with removable skins such as bananas. Canned fruit might be ok.
• Bottled water only (tap water has potential pathogens, even filtered water in the hospital).
• PPE includes gown, mask and gloves to avoid contaminating patient (though this can vary by facility).

**Other Important Players in Immunity**
**Complement** is a system of about 30 proteins that work together to fight off microorganism invaders. It has many functions in the inflammatory response. The short version is that complement causes lysis of cells, phagocytosis of cells and inflammation of the surrounding tissues.

**Platelets** do more than just clot the blood. They also release substances that increase vessel wall permeability and activate complement, allowing for chemotaxis of WBC.

**Lymph nodes** play an important role in immunity. When they are removed, patients are at higher risk for infection. Patients with mastectomy typically have lymph nodes removed and are at risk for infection on the side of the mastectomy, so you would
never start an IV line on that arm. You would also want to watch for edema on that arm as it is very likely to occur (elevation is helpful and some arms may need to be wrapped in a compression-style bandage). We also avoid BPs on that side as well.

**The First Line of Defense**
The epithelium serve as the body’s first line of defense, and when these surfaces are compromised, the risk for infection goes up. Here are some examples:

**The Respiratory System**
In the respiratory system, the epithelium are damaged by smoking. The cilia become paralyzed and ineffective which means that all that gunk you breathe in gets dumped in the lungs with no filter to keep it out. This is why smokers have such higher risk for respiratory infections.

**The Gastrointestinal System**
Gastrointestinal defenses are designed to keep pathogens at bay. Lysozymes in secretions, rapid pH changes (pH of 2 in the stomach increases to a pH of 8 in small intestine), normal flora, peristalsis, secretions, exfoliation of dead cells…all work to keep the microbes from causing infection.

So what happens when the normal GI system is compromised because of things we do in the clinical setting?

- Feeding tubes. When you bypass the upper GI tract with a feeding tube, then not every defense will be in play. This is why you will change the tubing and tube feed container regularly: in an open-bag system, do not hang more than four hours of tube feeding at a time. In a closed/sterile system (where the tubing connects directly into a closed bottle), change these AND the tubing daily.
- What if you give an H2 blocker or PPI to reduce stomach acid? When the stomach is less acidic than normal, then the patient is at higher risk for infection.
- GI surgery. Before opening up the gut, it is important to decontaminate it before GI surgery. If surgery is planned, you’d have the patient do a “bowel prep” which essentially entails drinking a concoction to make them poop a lot, thereby cleaning out the bowel. In an emergency with no time for formalities, the peritoneal cavity can get contaminated, so you would give antibiotics like Flagyl post op. However, note that when you give lots of broad spectrum antibiotics, you wipe out normal flora and can get overgrowth, for example with clostridium difficile (c-diff).

**The Genitourinary System**
Females are at higher risk for UTI than males due to a shorter urethra and proximity to anus. In addition, males have the benefit of antimicrobial seminal fluid. Both males and females have acidic urine and the bladder mucosa secretes mucus, IgA and lysozymes. Both urethras have peristalsis, valves and macrophages to help reduce infection risk.

The clinical significance of this:

- Inserting a Foley catheter has a high risk of UTI.
- Better to do an in-and-out cath multiple times a day...though to be honest this is kind of a huge pain in the neck, and I don’t see it ordered that often. Instead we just try to get the Foleys out ASAP. If there’s urinary retention after that, then we’ll do an I/O cath. Other tricks are to turn on the faucet while your patient is trying to urinate. Or, if your patient is a male who can’t ambulate to the commode, get them standing at the side of the bed to use the urinal since it is often the case that men just can’t urinate sitting in bed or lying down.
- Residual urine increases the risk of UTI, so get out that bladder scanner if you’re suspicious of retention.
- Infections can tract up to the kidneys, which can be very serious!
The Second Line of Defense: Nonspecific Defenses

Inflammation

Inflammation can be caused by pathogens (ex: cellulitis), thermal sources (ex: a hot motorcycle pipe on the leg), radiation (ex: sunburn) and chemical (ex: aspiration of gastric juices into lungs). The hallmark features of inflammation are:

- Red (vasodilation)
- Hot (vasodilation)
- Swollen (leaky capillaries)
- Painful (chemical response, also swollen tissue stimulates nerves)

Pathophysiology of Inflammation

When the vessels vasodilate, the neutrophils arrive via chemotaxis. They get into the tissue because the vessels have increased permeability, which is part of the immune response. Therapies include drugs, heat & cold, elevation.

- Heat increases superficial blood flow. Use heat for muscle spasm, stiff joints, superficial thrombophlebitis.
- Cold vasoconstricts, decreases swelling, decreases metabolism, and slows nerve conduction. Use cold after a trauma, either intermittently or continuous (some studies show continuous is better). If continuous is used, note that we’re not using ice here, folks. Ice goes on for 20 and then off for 20 to 60 minutes. A common use for continuous cold therapy is after a Le Forte surgery. The patient wears a mask-type apparatus that is connected to a machine that continually pumps cool water through the mask, helping to reduce swelling and pain in the surgical area.

Phagocytosis

Phagocytosis is the domain of the neutrophils and macrophages. Pus is basically a bunch of dead neutrophils that have responded to bacterial infection.

- You will see pustules in an acne bacterial infection and folliculitis
- Vesicles are blisters and you’ll see this in herpes virus, impetigo, and contact dermatitis

Fever

Temperature is regulated by the thermostat in the hypothalamus.

- Metabolism creates heat
- Perfusion and diaphoresis dissipate heat, as does vasodilation
- Increased temps occur with pyrogenic factors (infection) or injury to the hypothalamus (head injury or CVA), or inability to dissipate (heat stroke)
- 37-degrees Celsius is normal
  - We start treating/culturing around 38.5 (possibly lower in a patient receiving chemotherapy)
- At 40-degrees, thermoregulation is impaired
- 41-degrees is lethal!
- A slight fever is beneficial because it interferes with bacterial growth

Interferons

Interferons are proteins known as cytokines, which if you recall, are the proteins that communicate to other cells telling them to trigger the immune response. They are called “interferons” because they “interfere” with viral replication, activate immune cells (the natural killer cells and macrophages), and up-regulate antigen presentation. Interferons protect nearby cells from infection or cancer, so they’re pretty darn useful! However, they come with a price. When your patient has a fever, muscle-pain and other “flu-like” symptoms, that’s the interferons talking. Like most aspects of the immune system, they can be suppressed by steroids such as prednisone.
The Third Line of Defense: Acquired Immunity

B & T Lymphocytes
Lymphocytes are “trained” in one of two places...the bone marrow (B cells) or the thymus (T cells). As lymphocytes mature, the DNA that codes for cell surface receptors is sliced and diced so that each cell makes a different shape of receptor. Cells with receptors that will fight off the bad guys and not hurt one’s own tissue are further developed and sent out into circulation. Those receptors with the potential to interact and destroy native (self) tissue are destroyed...usually. If they get out into the body, later in life they can get activated and will then attack one’s normal tissue (thus causing an autoimmune disease).

Hypersensitivity Reactions
There are four types of hypersensitivity/allergic reactions...it’s not all anaphylaxis and welts!
- Allergies/Anaphylactic (Type I)
- Autoimmune (Type II)
- Immune Complex (Type III)
- Delayed Hypersensitivity (Type IV)

Type I Hypersensitivity - Allergic Response
A Type I hypersensitivity reaction is a rapid response to an antigen against which the individual has pre-existing IgE antibodies. IgE is present in very low levels in most people and it has a half life in the serum of only 2-3 days. However, if it is bound to high-affinity receptors, then its half-life is about 3 weeks. The high-affinity receptors are found on mast cells and basophils. Anaphylaxis occurs with Type I reactions.

Type II Hypersensitivity - Autoimmunity
A Type II hypersensitivity reaction is caused by specific antibodies (IgM or IgG) binding to cells or tissue antigens. This is basically the body turning its immunity powers against its own cells. Examples are Rheumatic Fever where the body thinks its own cells are Strep, so it attacks them. Other examples are Goodpasture Syndrome, hemolytic anemia or in hemolytic disease of newborns (this occurs when a Rh-negative mom has a second Rh-positive child and the mom’s IgG antibodies attack the fetus’ red blood cells because it sees them as foreign invaders.)

Type III Hypersensitivity - Immune Complex
This type is mediated mainly by IgG antibodies. It is now thought that this form of hypersensitivity has a lot in common with Type I, except that the antibody involved is IgG and therefore not bound to mast cells. These complexes are not easily cleared by the spleen or liver and they tend to lodge in areas of high blood pressure, blood flow turbulence, and in joints where inflammation occurs...common sites of injury are the kidney, skin and mucus membranes. Some examples of Type III hypersensitivity include glomerulonephritis, rheumatoid arthritis, vasculitis and systemic lupus erythematosus.

Type IV Hypersensitivity - Delayed Hypersensitivity
These are termed “delayed” reactions because they are cell-mediated responses that typically take 2-3 days to develop. A perfect example of this is a positive skin reaction to a TB test. Other Type IV reaction examples include graft versus host disease, Hashimoto’s thyroiditis and transplanted organ rejection. Read more about it here, if you are so inclined.

Aging and Immunity
As you get older, the thymus atrophies, leading to decreased T cell function. However, note that healthy elderly who live AT HOME have nearly the same risk of infection as younger people do. The higher risk comes into play with chronic disease (such as diabetes and cancer), hospitalization and time spent in a skilled nursing facility (SNF).
How do you protect immunity in the elderly?
• Prevent injury. Ensure that fall protocols are in place and take care of the skin (pressure ulcers are HUGE causes of infection, and the elderly are very susceptible to pressure ulcers.)
• Immunizations (influenza, pneumococcal, zoster, tetanus). Note that the vaccination does not always prevent disease, but may reduce morbidity and mortality if disease occurs. One way to get vaccine rates up is for hospitals and SNFs to have standing orders for patients to be screened for vaccines and receive them when appropriate. It has helped a lot to get vaccine rates up.

Which vaccinations would a healthy 89 year old in a SNF receive?
• Pneumococcal (one dose after age 65)
• Tetanus (q 10 years)
• Influenza (once each fall)
• Zoster (once)

Cancer
When you think of cancer, you typically think of solid tumor cancers, though there are actually multiple types of cancer (shown below). In the case of solid tumor cancers, you have cells that have lost their normal ability to stop growing when they touch adjacent cells, so the tumor just keeps on growing. Cancer also de-represses genes from fetal development that code for growth factors and also de-represses genes that create cell maturity and specialization. In addition, cancer cells lose their normal adhesiveness that holds tissue together, so they break off and metastasize. With cancer, cells no longer die like they’re supposed to…they essentially become immortal (apoptosis), and they gain the ability to stimulate blood vessel growth (angiogenesis) which is what brings nutrients to the tumor. The outcome of all this is that you have immature cells that live too long, grow too fast, break off and metastasize.

Types of cancer, classified by the type of tissue in which the cells develop:
• Lymphomas (cancer of infection fighting organ): Lymphomas start in the lymphocytes and affect the ability of the lymphatic system to fight infection. The lymphatic system removes excess fluid, transports and absorbs fatty acids, AND is responsible for transporting white blood cells to and from the lymph nodes. When the lymphocytes are cancerous, they multiply and gather in the lymph nodes (and sometimes other tissues such as the liver and spleen) and hinder the body’s ability to effectively fight off infection.
• Leukemias (cancer of blood forming organs): Leukemias originate in the bone marrow and are caused by the rapid production of white blood cells that do not function properly, are not able to fight infection and prevent the bone marrow from making enough red blood cells and platelets.
• Myeloma (originates in bone marrow): Myeloma is a cancer that involves plasma cells...the white blood cells that produce antibodies. These myeloma cells keep the body from being able to fight infection.
• Carcinomas (cancer of epithelial cells such as skin, lining of lungs): Carcinomas are the most common types of cancers and can stay in their primary location or spread to other parts of the body. Basal cell carcinoma is the most common type of skin cancer, with squamous cell carcinoma coming in second. You can also have carcinoma of the renal tubules (renal cell carcinoma) and milk ducts (ductal carcinoma in situ), which is the most common type of breast cancer.
• Sarcomas (cancer of bone, muscle and connective tissue): Sarcomas can originate in both soft tissue and bone, and are classified as such. Soft tissue sarcomas develop in muscle, fascia, tendons, fat, synovial tissue and even blood vessels. Sarcomas of the bone encompass all the bone cancers including osteosarcoma and chondrosarcoma, which forms in cartilage cells.
• Adenocarcinoma (originates in glandular tissue): These cancers form in glandular tissue throughout the body, namely the lungs, prostates, colon/rectum, pancreas and esophagus.
• Blastoma (originates in embryonic tissue of organs): Blastomas arise from undifferentiated or embryonic tissue
and can occur in various parts of the body. For example, a nephroblastoma is a type of renal cancer and a retinoblastoma is a tumor of the retina. You will also see glioblastoma (brain tumor) and osteoblastomas (affecting bone).

**Staging and Grading are done to classify the extent of the disease.**
- Stage 0 = Cancer in situ (limited to surface cells)
- Stage I = Cancer limited to the tissue of origin, evidence of tumor growth
- Stage II = Limited local spread of cancerous cells
- Stage III = Extensive local and regional spread
- Stage IV = Distant metastasis

**Cancer Treatments:**
- Surgery to remove the tumor and lymph nodes entirely. Can also be done to de-bulk the cancer for palliative care. For example, if the cancer is pressing on the trachea, you’d de-bulk it so the patient can breathe. We had a patient with a tumor that was pressing on the trachea, but no one would operate because the location of the tumor was just too tricky. We couldn’t put in a tracheostomy because the tumor was in the way. This patient ended up being intubated for weeks. It was heartbreaking.
- Immune therapy with exogenous interferon (“exogenous” means that we give it to you, the body isn’t making it).
- Radiation kills rapidly dividing cells
- Chemotherapy kills rapidly dividing cells and blocks new vessel growth
- Hospice is for patients for whom death is anticipated within six months. The focus is on quality of life, pain control and family support.

---

**An easy way to remember the warning signs of cancer!**

**Big Scary Bears Like Ingesting My Cookies**
- B = Bowel or bladder habit changes
- S = Sore that doesn’t heal
- B = Bleeding or discharge that’s unusual
- L = Lump or thickening of tissue
- I = Indigestion or difficulty swallowing
- M = Mole or wart changes
- C = Cough or hoarseness that won’t go away

---

**Cancer Problem List** (these are the items that would go on a care plan)
- **Potential cancer** related to exposure to carcinogen
  - Plant-based cancer prevention diet, no smoking, gloves when handling chemotherapeutic agents, avoid viruses associated with cancer such as Hep B (get vaccinated), wear lead aprons during X-ray.
- **Potential spread of cancer** related to failure of early detection
  - Screening via stool guaiac, colonoscopies, mammograms, PAP smears
- **Infection** related to side effects of chemo and/or radiation, malnutrition, surgical removal of lymphatics, immature WBCs
  - Infection is the #1 killer in cancer. WBCs are rapidly dividing cells and they get zapped by chemo and radiation. The nadir (low point) is 10 days after chemo and that’s when the WBC that got zapped would have been out in circulation, so this is the time when infection risk is the greatest.
  - Consider if lymphatics have been removed. What would happen if there was an infection in the tissue that used to be drained by those lymphatics?
  - Neutropenic precautions for ANC less than 500.
- **Bleeding** related to leukemia crowding out the platelets, causing thrombocytopenia
- **Bleeding** related to thrombocytopenia caused by chemo/radiation
  - Platelets are rapidly dividing cells, so when we give chemo/radiation to destroy rapidly dividing cancer cells, the platelets get zapped, too. What is the significance of low platelet count? Above 50,000 you are more or less ok (but patient will probably be a little “bleedy”), but less than 20,000 you could have serious bleeding problems so severe that you will institute bleeding precautions AND be watchful for spontaneous hemorrhage. The lowest platelet count I’ve seen is 3,000. Scary stuff. For example, think about your patient with very low platelets and very high blood pressure. You are going to be ON THAT blood pressure like a hawk to help avoid spontaneous hemorrhage (especially cerebral).
  - You can administer platelets but your patient will make antibodies against them making future transfusions potentially more problematic, so hold off transfusing unless you are desperate.
  - Bleeding Precautions = protect from injury, no IM injections at all (ever!), no flossing teeth, no rectal temps or rectal meds or rectal tubes (basically nothing in the rectum), no invasive procedures unless absolutely necessary. If a patient needs an invasive procedure, the doc will probably transfuse platelets first if possible. Make sure your doc knows your patient’s platelet count if it could be an issue!

- **Pain** related to tumor impinging on structures and/or inflammation

- **Malnutrition** related to cancer cachexia, obstruction in gut, N/V and anorexia with chemo
  - Antiemetics are given before chemo or before meal.
  - Check food preferences...the idea is to get the patient eating ANYTHING.
  - Perhaps avoid hot food (smell can cause nausea). Milkshakes and freshly baked bread seem to be popular on cancer units.

- **Edema** related to lymphatic removal, venous/lymphatic obstruction
  - Elevate or wrap for edema

- **Stomatitis** related to chemo/radiation
  - Dental consult encouraged prior to initiation of cancer treatment for the specific subpopulations with allogeneic bone marrow transplantation, leukemia induction/re-induction chemotherapy, or head and neck malignancies.
  - Oral assessment should be performed on a daily basis as a mandatory component of the nursing assessment.
  - Oral care regimen should consist of the measures below, and should be performed three times daily and at bedtime. In addition, the regimen should be performed one additional time (if awake) at night in patients receiving high dose methotrexate, those undergoing bone marrow or stem cell transplants, or patients with a prior history of mucositis.
    - Brush teeth with soft toothbrush and fluoride toothpaste for general hospitalized patient population; supersoft toothbrush is suggested for bone marrow transplant and hospitalized leukemia patient populations. Consideration can be given to the use of toothette swabs or Peridex mouth rinses in situations where toothbrushing may be harmful or contraindicated (for example, in individuals with periodontal disease and low platelets, or gingival hemorrhage). Dental/oral medicine consult should be considered in these situations.
    - Patients with platelets less than 50,000 should not floss.
    - Mouth rinses: Normal saline is preferred. Sodium bicarbonate, sterile water, or nonalcoholic rinses are alternatives. Nonalcoholic chlorhexadine mouthwash is not recommended for mucositis prevention, but may be considered for prevention of dental caries in individuals who cannot perform toothbrushing, or in other specific situations as indicated clinically. Dentistry or oral medicine consultation should be considered. Hydrogen peroxide mouth rinses should not be used EVER....ouch!
    - Topical fluoride for dental hygiene and caries prevention in patients undergoing head and neck or total body irradiation and in patients with significant xerostomia.
    - Mouth moisturizer (non-petrolatum) to lips and oral cavity as needed.
• Patient Education r/t oral care
  • Education should reinforce the above oral care standard
  • Provide information upon hospital discharge or at initiation of outpatient therapy (such as a computer printout or brochure if provided by the hospital)
  • Post patient education brochure in patient rooms if available. Many hospitals utilize a special TV channel to provide education...these can be a great resource!
• Saliva Substitutes
  • Mucin-based saliva substitutes are supported by the literature as being superior to carboxymethylcellulose-based substitutes for both patient preference and reduction of xerostomia.
• Topical Analgesics
  • Compounded analgesic mouth rinses such as “magic mouthwash,” consisting of various formulations of lidocaine, diphenhydramine, antacids, and/or sodium bicarbonate, are not recommended.
  • Single agent topical analgesics are preferred, such as Ulcerease® (an anesthetic mouth rinse).
• Alopecia related to chemo/radiation
  • Consider scarves or wig. Consider cutting hair or shaving head before all hair falls out as this can be traumatic for the individual. Cutting ahead of time provides the patient control over their situation.
• Diarrhea related to side effects of chemo/radiation, obstruction
  • Treat the symptoms and investigate the need to remove obstructions (if present).
• Constipation related to obstruction, opioids
  • Treat the symptoms and investigate the need to remove obstructions (if present).
• Anemia related to chemo/radiation/bleeding
  • Assist with ADLs (due to fatigue)
  • Give Epogen as ordered
• Bone fractures related to metastasis
  • Gentle mobility
• Thrombophlebitis related to increased coagulation of blood, immobility, pressure on vessels
  • Consider SCDs, compression stockings, anticoagulants, mobility
• Pressure on normal structures. What would happen if pressure was exerted by a tumor on the ureter/urethra, airway, vena cava, bowel, spine? Probably nothing good.
  • Monitor for passage of fluids/air. Consider function distal to the obstruction caused by the tumor.
• Effusion related to pleural involvement
  • Effusions could be CHF or cancer. MD will drain the effusion and examine cells to rule out cancer.
• Psychosocial adjustment (depression, anger, grief, coping)
• Infertility related to chemo/radiation treatment
  • Note that sperm are rapidly dividing cells and subject to death by chemo/radiation
  • Patients who wish to have children in the future can freeze sperm/eggs for future fertilization

**HIV Disease**
In HIV, a retrovirus (RNA) enters the cell bringing reverse transcriptase with it. It makes DNA copies of its RNA, and viral DNA integrates into the cell’s own DNA. Later, it replicates, making viral RNA and viral proteins...it gets packaged up by protease and buds out of the cell to infect others. A person may be HIV+ for many years before the virus destroys much of the immune cells leaving the person’s immune system severely compromised. When the CD4 count drops below 200, then the HIV infection is considered to be AIDS, with or without an opportunistic infection/cancer.
**Infectious Diseases Associated with HIV**

- Kaposi’s Sarcoma is a type of cancer that we don’t see much of it anymore thanks to aggressive HIV treatments
- Tuberculosis
- Cytomegalovirus
- Cryptococcal meningitis
- Toxoplasmosis
- Pneumonia, specifically pneumocystis carinii pneumonia (PCP)

**HIV Treatment**

- Multi-drug therapy
- Compliance and resistance are issues as the drug regimen can be intense; advances in pharmacology have made it less so
- General health maintenance
- Psychosocial support

**Rheumatoid Arthritis**

Rheumatoid Arthritis (RA) is a chronic autoimmune disease that causes inflammation in the joints and, in some cases, the eyes, skin, heart, blood vessels and lungs. Like all autoimmune disorders, RA occurs when the body’s immune system attacks its own tissues. The antibody complexes lodge in perivascular areas and joints, causing a painful swelling that leads to reduced mobility and deformity.

**Treatment for Rheumatoid Arthritis**

- Balance between rest and exercise
- Use splints to mobilize joints and reduce deformities
- Physical therapy
- Anti-inflammatory/immune suppressive drugs
  - NSAIDs: indomethacin, rofecoxib, etc…
  - DMARD: methotrexate, etanercept, etc…
  - Corticosteroids: prednisone
- **Prosurba** therapy
- Heat
- Sometimes cold
- Surgery to replace damaged joints

This won’t be on your exams or NCLEX, but just for your own personal knowledge...there is research being done by outside-the-box rheumatologists that RA is related to leaky gut and diet. Just google “leaky gut” and “autoimmune” and you’ll learn a ton about treating the actual cause of the disease, and not just treating the symptoms. I’ve seen it work firsthand! However, for the sake of your exams, go with the traditional medical treatments.

**Systemic Lupus Erythematosus (SLE)**

SLE involves a widespread autoimmune response that affects body tissues and organs...joints, kidneys, lungs, brain cells, heart and skin.

Drug therapy is usually targeted at reducing inflammation, the cause of most lupus symptoms. Drugs used to treat the disease...
include nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, disease modifying antirheumatic drugs (DMARDs), and cytotoxic drugs. Notice that the drugs to treat SLE are similar to those used to treat RA.

- **NSAIDs**: NSAIDs (either over-the-counter or prescription strength) are often recommended to alleviate fever, muscle pain, and joint pain and swelling. Individuals with a mild form of lupus may find an NSAID the only medication they need to relieve their symptoms, whereas individuals with more active or severe disease may require additional medications.
- **Corticosteroids**: Corticosteroids are the most common drugs used in lupus treatment because they reduce inflammation at a rapid speed and have been associated with a slowdown in the progression of the disease. The goal of corticosteroid therapy is to induce and maintain lupus remission using the lowest possible dose to avoid side effects, which can include fluid retention, muscle weakness, weight gain, increased blood sugar, and bone growth suppression (osteoporosis). Major organ involvement, however, may require higher doses of corticosteroids.
- **DMARDs**: DMARDs are frequently used in the treatment of lupus. DMARDs are effective in treating joint pain, skin rashes, fatigue, and inflammation of the lungs associated with lupus. Continuous use of DMARDs may prevent flares from recurring. In some cases, the use of DMARDs allows an individual to take lower doses of corticosteroids, decreasing the risk of side effects associated with high-dose corticosteroids.
- **Cytotoxic drugs**: Cytotoxic (cyto = cell; toxic = damage) drugs are usually prescribed for severe cases of lupus, when more than one major organ (kidneys, liver, brain, heart, lungs) is affected. These drugs work by targeting rapidly growing cells and therefore are useful in suppressing the cells involved in the hyperactive immune response.

**Scleroderma**
Pathophysiology of this disease involves a hardening of the skin and connective tissue. Scleroderma is one of a group of chronic autoimmune diseases including systemic lupus erythematosus and Sjögren’s syndrome. These diseases form a spectrum, and feature various combinations of the same set of symptoms. Skin thickening is the hallmark of the disease. Traditionally, the term “scleroderma” has encompassed two groups: “localized scleroderma”, where problems are confined to the skin, and “systemic sclerosis”, where internal organs and vessels are involved as well as the skin.

**Localized Scleroderma (Morphoea)**
Localized scleroderma includes a group of conditions characterized by circumscribed, patchy, or linear scleroderma without the typical serological and visceral manifestations of systemic sclerosis (see below). This condition should not be confused with limited cutaneous scleroderma (CREST), which is a systemic variety of scleroderma. Typical signs are one or more erythematous or violaceous areas of the skin which worsen to the point that they become sclerotic and waxy. During the active phase, plaques can grow up to several centimeters in diameter. This form of scleroderma primarily affects children and young adults, especially females.

**Linear Scleroderma**
In linear scleroderma, sclerotic lesions appear as linear streaks or bands, usually on the upper or lower extremities and less commonly on the trunk or forehead. If the frontoparietal scalp is involved (“en coup de sabre”), disfiguring facial asymmetry and hemiatrophy may occur, while joint contractures and neurovascular involvement may occur if it crosses a joint. Inflammation and fibrosis may extend to the deep fascia, muscle, and sometimes, underlying bone. During the active phase of disease, blood tests may show elevated eosinophils, positive ANA and positive rheumatoid factor.

No treatment has been uniformly successful for these conditions. Transition from the localized variety to systemic sclerosis is very rare, and it is rare for patients to suffer from both.
Systemic Scleroderma
Systemic scleroderma is a connective tissue autoimmune disease characterized by thickening of the skin. Limited cutaneous scleroderma involves the face, feet, hands. Diffuse cutaneous scleroderma covers more surface area and can progress to organs such as heart, kidneys, lungs and the GI tract.

Clinical Presentation
Typically, patients present with tight skin, painful joints and Raynaud’s phenomenon. Swallowing difficulties and gastroesophageal reflux may also occur early in the disease. Severity of the disease ranges from relatively benign to a more aggressive presentation.

More serious problems include esophagitis, malabsorption, renal failure, pseudo-obstruction, cardiac arrhythmias, pulmonary hypertension and pulmonary fibrosis.

Disease Management
Scleroderma is a chronic disease that affects many aspects of the patient’s body systems and overall quality of life, therefore a holistic approach is very important.

- Raynaud’s (which most patients with scleroderma have) may be controlled by maintaining warmth, especially of the central body, as well as avoiding smoking, β-blockers, and other exacerbating factors. Sometimes calcium blockade, topical nitrates, and even parenteral vasodilators are required. Sympathectomy (a procedure in which one or more sympathetic ganglion are removed) offers temporary relief in many cases.
- Scrupulous skin care and moisturization is essential.
- ACE inhibitors can be effective in the management of scleroderma-related hypertension
- Anti-fibrotic agents (though the research shows this may not be significantly effective with the drugs currently available...more development is needed in this area)
- Immunosuppressive therapy with cyclophosphamide and mycophenolate mofetil (methotrexate studies showed no significant benefit when compared with a placebo: source hopkinsscleroderma.org, 2017).
- Anti-inflammatory meds: NSAIDs and corticosteroids

Chemical Agents and Scleroderma
Exposure to chemical agents is purported to be a cause of scleroderma:
- organic chemicals
- aliphatic hydrocarbons
- aromatic hydrocarbons
- epoxy resins
- aniline-treated rapeseed oil
- silica
- foam insulation (urea-formaldehyde)
- cocaine & appetite suppressants

Malnutrition
- Patients with malnutrition are more susceptible to infection
- Wounds heal slowly
- Catabolism of muscles, including the muscles of respiration (think about your patient on a ventilator who isn’t eating)
- Think nutrition on every patient! How long do you anticipate it will be before normal diet can be taken?
- Balance the risks/costs of nutritional assistance from NG, PEG, TPN. TPN should be considered a “last-resort” form of nutrition. It has very high infection risk and when the gut is bypassed, then the healthy gut flora can’t do their job which leads to even higher risk of infection. Plus, TPN must be administered through a central line which carries risk of infection and, in the case of PICC lines, risk for DVT.
- More than 4-5 days NPO is serious even in healthy patients!
- Megestrol is a synthetic form of the hormone progesterone. When used for treatment, a side effect was weight gain. This has led to its use for wasting syndromes and weight loss such as seen in AIDS and cancer.
- Cachexia is different from anorexia. Even with “adequate” nutrients, cachectic patients may lose weight. It is hard to treat!
- Anorexia is a lack of appetite, and is very common in cancer patients. Visit cancer.gov for ideas on how to manage it.
**Diabetes**

Patients with diabetes have a high risk for infection for a variety of reasons:
- Decreased blood flow
- Poor chemotaxis and phagocytosis
- Hyperglycemia is substrate for microbes...they love sugar!

Studies show that glucose control is important in patient outcomes:
- *Glucose control lowers the risk of wound infection in diabetics after open heart operations.* (Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A.)
  - Elevated blood glucose levels in the postoperative period are associated with an increased risk of deep wound infection in diabetic individuals undergoing open heart operations at Providence St. Vincent Hospital.
  - The incidence of infection in diabetic patients was reduced by keeping mean blood glucose level less than 200 mg/dL in the immediate postoperative period.
- *Poor glycemic control is associated with increased mortality in critically ill trauma patients.* (Gale SC, Sicoutris C, Reilly PM, Schwab CW, Gracias VH.)
  - Glycemic control improves outcomes in cardiac surgical patients and after myocardial infarction or stroke.
The Organs of the Gastrointestinal System
When we talk about the GI system, the first thing that comes to mind is the stomach and intestines. But actually, there’s a lot more going on.

Key Organs
The main players in the GI system are the ones responsible for the direct breakdown and absorption of nutrients.

- Esophagus - transports food from mouth to stomach
- Stomach - initial breakdown of nutrients occurs here
- Small intestine - main absorption of nutrients
- Large intestine - absorption of water and some nutrients
- Rectum - absorption of electrolytes, indigestible matter broken down by bacteria, stool thickened
- Anus - passage of waste

Associated Organs
- Oral (teeth, tongue, salivary glands) - initial breakdown of food
- Liver - process nutrients, secrete bile
- Pancreas - production of enzymes that aid in digestion
- Gallbladder - storage and concentration of bile

Pain Assessment of the GI Tract
Assessing pain related to the GI tract can be tricky for a variety of reasons. First of all, abdominal pain is often “diffuse” meaning it’s felt all over the abdomen, so it’s hard to pin down. Other times, the pain radiates to another part of the body (this is known as “referred pain.”) Plus, many GI disorders will cause pain, so hearing that the patient has abdominal pain just tells you that SOMETHING is going on...but to determine what it is, you and your physician colleagues will have to do a fair amount of investigative work.

Common Assessment Findings
When your patient has something going on in the GI tract, they’ll often present with abdominal pain and nausea/vomiting. Abdominal pain can be superficial (sharp pain that is localized to a particular area and exacerbated by physical stimuli such as pressing on a certain part of the abdomen), visceral (usually a “dull” or “achy” pain) or non-visceral (a mixture of sharp and dull pain, but usually well localized).

Pain Location
Abdominal pain is typically described as being in one of three areas:

- Epigastric pain - stomach, gall bladder, pancreas, liver; can often be confused with cardiac pain so the patient may be worked up for both to rule out an MI. If the pain radiates to the back, it could be ruptured aortic aneurysm
- Umbilical pain - ovary, appendix, distal duodenum, jejunum, ileum, testes, upper ureter
- Lower abdominal pain - colon, rectum, bladder, uterus, ovaries

Referred Pain
Many times problems in the GI tract will result in “referred pain.” This is when the pain is felt elsewhere in the body thanks to the goofy way our nervous system works (check out “dermatomes” if this topic interests you!).

- Gallbladder - right shoulder blade
- Duodenum - right neck/shoulder
- Stomach - mid-back, between shoulder blades
- Liver - right upper back/neck/shoulder, right flank
Pain History
Anytime your patient has pain, you want to take what is known as a “pain history.” You can remember how to do this with the acronym PQRST:

- **P** = provocation and palliation: “What were you doing when the pain started?” “What makes it worse and better?”
- **Q** = quality: “Describe the pain...is it sharp, dull, achy, burning, tingling, constant, tearing, shooting, etc....”
- **R** = region and radiation: “Where is the pain located?” “Does the pain radiate anywhere?” “Does the location of the pain change?”
- **S** = severity: “Rate your pain on a 0-10 scale.”
- **T** = timing: “How long does the pain last?” “How often does it occur?”

A great example of taking a pain history can be seen with appendicitis. Typically what you will see is:

- **P** = sudden onset
- **Q** = cramping (often leading to nausea, vomiting and diarrhea/constipation)
- **R** = right lower quadrant that changes to umbilical area as it progresses
- **S** = 7-10 (note that sudden relief typically means the appendix has ruptured)
- **T** = constant

Nausea, Vomiting and Diarrhea

**Nausea and Vomiting**
N/V goes hand-in-hand with GI tract illnesses, but can have other causes as well.

- Increased tension on walls of the stomach, duodenum and even the lower end of the esophagus.
- Decreased gastric motility
- Toxins (think food poisoning!)
- Blood in the stomach is very irritating and can cause N/V
- Bacteria (bacterial gastroenteritis)
- Head injury or anything that causes increased intracranial pressure
- Noxious odors or sights

**Diarrhea**
Diarrhea is a result of increased gastric motility (peristalsis), which can be caused by various triggers: C.diff is a big one, as is any kind of toxin, blood in the lower GI tract, viruses like Rotavirus, bacterial infections, irritable bowel disease, Crohn’s Disease and ulcerative colitis. It’s important to note that certain medications can cause diarrhea, such as antibiotics.

If your patient has diarrhea you want to be watchful for dehydration, electrolyte imbalances and hypotension. Sometimes anticholinergic medications are used to decrease gastric motility, but if the diarrhea is a result of a toxin/infection then you want the body to get rid of it as fast as possible!

**GI Bleeds**
GI bleeds can occur in the upper or lower tract and have a variety of causes.

**Upper GI Bleeds**
If the patient vomits blood (hematemesis) then it’s an upper GI bleed, meaning it’s above the duodenum. If the blood is bright red, then it’s new and means the patient is bleeding RIGHT NOW! If the emesis looks like coffee grounds, then this means the blood is partially digested (gross!) and it is from an older bleed. Remember, blood is irritating to the lining of the stomach, so it’s going to cause vomiting and/or diarrhea. Stools that include blood from an upper bleed will be dark and tarry (called “melena”). Upper GI bleeds are typically due to peptic ulcer disease, esophageal varices and cancer.
**Lower GI Bleeds**
If the bleeding results in red, bloody stools then it’s most likely a lower GI bleed originating from the intestine (large or small), rectum or anus (including hemorrhoids). However, they can occasionally be black and tarry, though this is usually the case with an upper GI bleed. The patient will often complain of diarrhea, and this is because the blood is irritating to the bowels. Some common causes are diverticulitis, inflammatory bowel disease, gastroenteritis, colitis, hemorrhoids and neoplasms. FYI: blood passed through the anus is called “hematochezia.”

**Assessments for GI Bleeds**
- Monitor H/H, will often be Q4 or Q6 hours
- Watch for signs of bleeding (emesis and stool)
- Monitor BP and check for orthostatic hypotension
  - 20 mmHg decrease in SBP
  - 10 mmHg decrease in DBP
  - may or may not involve a ≥20 bpm increase in HR
- Perform occult blood test on stool
- Assess abdominal pain

**Nursing Management of GI Bleeds**
- To combat dehydration, electrolyte imbalance and blood loss, ensure you have two large-bore IVs in place
- Draw labs: type & screen (or type & crossmatch if imminently transfusing), CBC, and BMP
- Prep patient for endoscopy or interventional radiology if needed (used to stop active bleeding)
- Patient will most likely be NPO initially, then clear liquids, then BRAT diet (bananas, rice, applesauce, toast)
- Drugs to decrease acid (protonix is common; pt may be on continuous protonix infusion)
- Replace fluids and electrolytes; transfuse PRBCs as needed

**Peptic Ulcer Disease**
Peptic ulcers can occur in the duodenum or within the stomach itself. Risk factors are: smoking, steroids, use of aspirin or NSAIDS, caffeine, alcohol and stress.

**Gastric Ulcers:** Ulcers in the stomach cause breaks in the mucosal barrier.
- Often caused by H. Pylori infection.
- The patient will experience bouts of gastritis (inflammation of the stomach lining).
- Pain with eating; antacids do not relieve pain; patients are likely to be malnourished.
- Can cause hematemesis (blood in emesis); vomiting can relieve pain.

**Duodenal Ulcers:** Most ulcers are duodenal.
- Occurs in patients with rapid gastric emptying which results in increased hydrochloric acid secretion.
- Things that cause higher acid secretion: protein-rich meals, alcohol and vagal stimulation.
- Pain when stomach is empty; eating or taking antacids help relieve pain.

**Stress Ulcers:** Occur after an acute illness, trauma, head injury or severe burn (especially if it results in shock or sepsis). Also caused by medications such as aspirin, NSAIDs and steroids.

**Signs and Symptoms of Ulcers**
- Acute pain: burning, gnawing, cramping, aching
- Nausea/vomiting: more likely with gastric ulcer as a result of stasis
- Bleeding: substantial blood loss can occur quickly and can be fatal
Acute Gastritis
Gastritis is the inflammation of the mucosal lining of the stomach. It can be caused by a viral or bacterial infection, or ingestion of a corrosive substance (aspirin, NSAIDs, oral chemotherapy, food poisoning, alcohol, acidic beverages such as coffee, and even steroids). Symptoms include:
- nausea/vomiting
- bleeding
- lethargy
- anorexia
- epigastric pain or discomfort
- tender abdomen, not localized
- hyperactive bowel sounds

Treatment of Acute Gastritis
Treatment revolves around decreasing the inflammation and removing the cause. The patient will receive a PPI such as protonix and antiemetics such as Zofran if needed. They will also need to let the bowel rest. This is done by ingesting only clear fluids and slowly advancing diet (clear liquids to full liquids to soft/bland foods). Once the patient is symptom-free, they can return to a regular diet with some recommendations:
- Eat a fiber-rich diet
- Avoid high-fat foods
- Avoid coffee, alcohol, carbonated beverages and acidic foods/beverages
- Foods high in flavonoids can help fight *H. pylori* infection (apples, cranberries, tea, onions, garlic)

Inflammatory Bowel Disease
Inflammatory bowel disease (often referred to as IBD) is a blanket term used to refer to Crohn’s Disease and Ulcerative Colitis. Both conditions cause severe abdominal pain, diarrhea, malnourishment, weight loss, anemia and fatigue.

Crohn’s Disease
Crohn’s is inflammation of the GI tract and can occur anywhere from top to bottom (mouth to anus) and symptoms will vary based on where the inflammation occurs.
- Abdominal pain with diarrhea
- Rectal bleeding
- Reduced appetite and weight loss (can be severe)
- Fever
- Fatigue
- Persistent vomiting in severe cases

Crohn’s Complications
- Intestinal blockages and abscesses
- Fistula formation (a fistula is when two structures of the body connect via the formation of an abnormal pathway)
- Scar tissue in the intestines
- Anal fissures
- Severe anemia
- Increased risk of colon cancer
- Malnourishment and deficiencies in vitamins and minerals (especially Vitamin D, B-12 and iron)
- Kidney stones if inflammation is in the small intestine
- It’s important to note that Crohn’s symptoms, which are due to inflammation, can show up in the eyes, skin and joints as well...it’s not JUST the GI tract that’s affected.
Crohn’s Medications
There is no cure for Crohn’s but medications can help patients manage symptoms and decrease the inflammation.

<table>
<thead>
<tr>
<th>Medication Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corticosteroids:</td>
<td>Suppress the immune response (Prednisone)</td>
</tr>
<tr>
<td>Immunomodulators:</td>
<td>Suppress the immune response</td>
</tr>
<tr>
<td>Biologics:</td>
<td>Usually reserved for patients who have not responded to other therapies; also suppresses the immune response</td>
</tr>
<tr>
<td>Aminosalicylates:</td>
<td>Reduce inflammation in intestines</td>
</tr>
<tr>
<td>Antibiotics:</td>
<td>Treats infections which are common in Crohn’s patients</td>
</tr>
</tbody>
</table>

Crohn’s Surgery
When the disease is severe, surgery is often the only reliable option. It can be conducted to remove a diseased portion of the GI tract (resection or colectomy) or address a complication such as a blockage or abscess. In some cases, an ostomy will need to be created for the passage of waste (more on ostomies further down).

Ulcerative Colitis
Ulcerative Colitis is an inflammatory disease of the colon and rectum (large intestine). Just like Crohn’s, the inflammation can pop up in the eyes, skin and joints as well...though it’s mainly seen in the large intestine. Symptoms include:

- Abdominal pain
- Persistent feeling that one has to move the bowels (tenesmus)
- Multiple loose bowel movements per day (can be severe)
- Blood or pus in the stool
- Weight loss, reduced appetite, malnourishment
- Fever
- Fatigue
- Anemia (can be severe enough to require blood transfusions)

Complications of Ulcerative Colitis
- Bowel rupture requiring emergent surgery
- Persistent bleeding requiring surgical intervention
- Increased colon cancer risk
- Malnourishment due to vitamin and mineral deficiencies

Ulcerative Colitis Medications
The medications used for Crohn’s are the same ones used for UC since the whole goal is to reduce inflammation.

Ulcerative Colitis Surgery
Unlike Crohn’s disease, surgical interventions are considered a “cure” for UC...remove the diseased colon and the patient typically bounces right back! Procedures include:

End ileostomy with proctocolectomy: The colon is removed along with the rectum and anus. This patient will have to wear a pouch to collect waste.

Proctocolectomy with ileal pouch-anal anastomosis (IPAA): In this surgery, the colon and rectum are removed and the ileum is made into a “pouch” that essentially acts as a rectum. Stool is passed through the anus, eliminating the need for the patient to wear an external bag. Note that this surgery is typically done in two parts. In the first surgery, the colon and rectum are removed, the ileal pouch is formed and a temporary ostomy is created. The patient will have this temporary ileostomy for about three months while the new pouch “rests” and heals. In the second surgery, the ostomy is taken down and the pouch is ready to start functioning. This is commonly referred to as a “J-Pouch.”
Irritable Bowel Syndrome
While IBD is an inflammatory disease of the bowel, IBS is a disorder affecting the muscle contractions of the bowel. Typical symptoms are abdominal pain, bloating, diarrhea and/or constipation. The abdominal pain is usually relieved after a bowel movement, and the patient can suffer from diarrhea, constipation or a mixture of both.

The causes of IBS are not well-understood. It may be genetic or related to infection, stress or trauma.

Treatments for IBS
Lifestyle changes: avoid foods that trigger IBS flare-ups, reduce stress, smaller meals
Medications: antispasmodics, laxatives, antidiarrheals, antibiotics and probiotics

Pancreatitis
Pancreatitis is acute inflammation of the pancreas, most commonly caused by alcoholism but can also be due to gallstones. It is extremely painful and can be so severe that the pancreas is essentially digesting itself (ouch!).

Common Assessment Findings in Pancreatitis
- Severe, unrelenting epigastric pain that may radiate to the back, chest or lower abdomen
- If pancreatitis is related to alcohol ingestion, usually occurs 12-48 hours after drinking
- If pancreatitis is related to a gallstone, pain typically occurs after a large meal
- Nausea and vomiting (can be severe and does not provide relief of pain)
- Abdominal distention
- Abdominal tenderness
- Fever
- Maybe jaundice if gall bladder disease present
- If severe: Turner’s sign (bluish left flank), Cullen’s sign (bluish periumbilical area), hypotension, pallor, cool/clammy skin, hypovolemia, hypoperfusion, obtundation and shock

Lab Values
- Elevated amylase (normal is 20-110) and lipase (normal is <160). THESE ARE THE MAIN ONES!
- Elevated WBC (normal is 4.5-11)
- Elevated LFTs (liver function tests)
- Elevated blood glucose due to the pancreas not secreting insulin correctly (normal is 70-110)
- Elevated CRP (elevated when >150 mg/dL)

Management of Pancreatitis
There are three MAIN things you will do for pancreatitis:
1) rest the pancreas by keeping the patient NPO
2) manage vomiting and nausea by inserting an NGT
3) treat the severe pain

In addition, you will need to:
- Maintain fluid volume with IV fluids
- Correct electrolyte imbalances
- Remove obstructing gallstone if that is the cause
- Treat complications (sepsis, hypotension, pleural effusions, respiratory distress)
Because the pancreas is not an encapsulated organ, the inflammation can affect nearby organs...namely the lungs. This can lead to pleural effusions and, in severe cases, ARDS (acute respiratory distress syndrome). Small pleural effusions can often just be monitored, but larger ones may require surgical drainage (thoracentesis) or a VATS procedure. ARDS is a critical problem that we’ll talk about in detail in Med/Surg 2. In general, pleural effusions are prognostic for poor outcomes when they occur in pancreatitis.

Cholecystitis
Cholecystitis is acute inflammation of the gallbladder, typically caused by an obstruction of the cystic duct that leads to a buildup of bile. It tends to occur with more prevalence in certain ethnic groups (Chinese, Jewish, Italian) and in those who are obese and sedentary. Other risk factors include: gallstones, cystic duct obstruction, age 40s-50s and high fat diet.

**Cholecystitis Symptoms** (typically occur after a high-fat or large meal)
- Severe pain in epigastric or RUQ that may or may not radiate to right shoulder; the pain usually starts suddenly and peaks in about 30 minutes; pain will increase with movement (including respirations)
- Murphy’s sign when taking a deep breath
- 60-70% will have biliary colic episodes as the stone moves from gallbladder into the ducts
- Abdominal tenderness
- Nausea and vomiting
- Low-grade fever
- Mild jaundice

**Murphy’s Sign**
Pain on deep inspiration when the inflamed gallbladder is palpated by pressing fingers under the rib cage

**Complications and Treatment**
Left untreated, the built-up bile can become infected and lead to sepsis, the gallbladder wall could tear or portions of the gallbladder could die (leading to rupture).

The first line treatment is to simply let the gallbladder rest by keeping the patient NPO. If infection is present, antibiotics will be given, and the patient will most likely need pain medication initially. Sometimes we’ll put in a “chole drain” if the ducts are blocked so that the organ doesn’t fill up with fluid; this procedure is called a cholecystostomy.

If cholecystitis continues to occur, the only thing left to do is remove the stones or remove the entire gallbladder surgically in a procedure called a cholecystectomy (more on these in a bit).

Cholelithiasis
Cholelithiasis is the presence of one or more gallstones in the cystic duct or the common bile duct. It results in rapid distention of the gallbladder causing a whole host of symptoms:
- Midline epigastric pain that may radiate to intrascapular region
- Nausea and vomiting
- Bloating, flatulence, belching

This patient will need fluid and pain meds while you draw labs and get an ultrasound to confirm the diagnosis. The main treatment for gallstones is removal of the stones or removal of the gallbladder itself (cholecystectomy).
ERCP

ERCP (endoscopic retrograde cholangiopancreatogram) is a procedure done to remove stones, widen a narrowed duct, insert a drain or take a tissue sample. It involves the use of a flexible scope (endoscope) to visualize the tubes that drain the gallbladder, liver and pancreas.

The procedure is done under conscious sedation and the scope is inserted through the mouth and gently moved down the throat into the esophagus, stomach and so on until it reaches the point where the ducts drain into the duodenum.

ERCP Prep

- NPO for 4 hours prior to the procedure
- Check for allergies to contrast dye
- Assess renal function
- CBC, coags
- IV in right arm (pt will partially lie on left arm during procedure)
- Administer topical anesthetic to reduce gag reflex (gargle and swallow)

Paralytic Ileus

Paralytic ileus can happen for a variety of reasons, but it’s important to note that it is an EXPECTED FINDING in the first 6-72 hours after abdominal surgery. If the ileus extends longer than expected, then there is likely some sort of complication going on and will require additional assessment.

Bowel Obstruction

Bowel obstructions typically occur in the small intestine (especially the ileum) because of the narrow passageway here. If your patient has a large bowel obstruction, be highly suspicious that it’s related to cancer and tumor growth. Signs of a bowel obstruction are abdominal pain, nausea, vomiting and dehydration. Intestinal blockages are a surgical emergency as they can quickly lead to strangulation of the bowel, tears and leakage of waste into the peritoneal cavity. The result? Sepsis.

Peritonitis

Peritonitis is inflammation of the peritoneum and it occurs due to infection, perforation, liver disease, trauma or problems with a PEG tube or peritoneal dialysis catheter. It is a medical emergency and must be treated ASAP! In bowel obstruction, peritonitis occurs via a three-part process: 1) the increased pressure caused by the obstruction leads to “reverse peristalsis” and vomiting; 2) bacteria proliferate; 3) the bowel perforates causing systemic effects of toxemia and peritonitis.

Signs and Symptoms of Peritonitis

- Severe abdominal pain
- Rigid, board-like abdomen
- Guarding
- Decreased urine output due to increased intra-abdominal pressure and/or sepsis
- Fever

Cholecystectomy Prep

Keep pt NPO
- Draw labs: H/H, coags, type & screen
- CHG bath the morning of procedure
- Possibly enema to reduce colon mass
- Antibiotic hung right before procedure

Laparoscopic Cholecystectomy

Contraindicated if stones are located in the common bile duct, otherwise it’s the preferred procedure (less invasive)

Small incisions and patient can usually go home within 24 hours.

Open Cholecystectomy

Used when laparascopic is contraindicated, pt is obese or adhesions present.

Larger incision, longer recovery time.

Post-Op Teaching

- Low fat diet
- Avoid spicy foods
- No alcohol
- Smaller meals
- Add high-fiber foods slowly
- Vitamin supplements

The bile ducts will eventually dilate to accommodate the volume of bile once held by the gallbladder to aid in the digestion of fats.
• Nausea and vomiting
• Diarrhea or constipation

Other Causes for Paralytic Ileus
• Inflammation causing the already narrow ileum to narrow even further
• Strangulated hernia (any time part of the bowel is “strangulated” this means its oxygen supply has been cut off).
  The affected piece of bowel will die and having any dead bowel in the body is very very very bad.
• Volvulus (twisted bowel) or intussusception (telescop ed bowel)
• Cancerous masses (neoplasms)

General Signs and Symptoms of a Paralytic Ileus
• Cramping abdominal pain, patient may say that it “comes in waves”
• High-pitched bowel sounds
• Abdominal distention
• Nausea and vomiting. If the patient vomits feces, this is very bad for everyone involved. Yes it happens.

Nursing Management of Paralytic Ileus
• Assume your patient is going to be going to surgery if you suspect an obstruction is present
• Decompress the bowel (NGT set to intermittent low wall suction)
• Monitor NGT drainage for amount, color, consistency...watch for blood!
• Administer meds to promote gastric motility (metoclopramide/Reglan)
• Keep pt NPO; give TPN if needed for nutrition
• Monitor CBC and electrolytes
• If post-surgical, encourage mobility...this will help get the gut moving!

Weight Loss Surgery
Weight loss surgery reduces the size of the stomach in order to force the patient to eat substantially smaller volumes of food.

Roux-en-Y gastric bypass: The size of the stomach is reduced to about the size of an egg, the duodenum is bypassed and food goes straight from the stomach to the jejunum.
Sleeve gastrectomy: A portion of the stomach is removed, leaving behind a small tube.
Gastric banding: A band is placed around the stomach, greatly reducing the amount of food that can be held.
Biliopancreatic diversion with duodenal switch: A large part of the stomach is removed but the duodenum is left in place. The duodenum is then connected further down on the intestines, bypassing a significant portion. The bypassed section of intestine is attached to the distal portion of the intestine, which allows digestive juices and bile to flow in and aid digestion.

Something to watch out for (besides the usual post-op things) is dumping syndrome (also called “rapid gastric emptying.”) This occurs when food is essentially dumped too quickly into the duodenum before it has been digested by the stomach. Early dumping happens within 30 minutes of ingesting food and late dumping occurs about 1-3 hours after eating. Symptoms include feeling faint, diarrhea, tachycardia and abdominal pain.

When the food is dumped straight into the small intestine, the body senses this and the gut releases hormones. The body responds by shifting vascular fluid into the intestine which makes them bloat. Next comes the diarrhea. Good times.

With “late dumping syndrome” the symptoms happen because the blood sugar drops about 1-3 hours after eating (which is also 1-3 hours after the pancreas excretes a large dose of insulin). Patients who eat a meal heavy in simple carbohydrates are at risk for experiencing symptoms of late dumping syndrome, so watch closely for hypoglycemia!
Treating Dumping Syndrome
• No liquids with meals; have patient wait to drink at least 30 minutes after eating
• Smaller, more frequent meals
• Complex carbs vs. simple carbs
• Center meals around protein and fat
• Instruct patient to lie down for 30 minutes after eating

Ostomies
Ostomies are used as a surgical remedy for serious bowel problems such as colon cancer, ulcerative colitis, Crohn’s disease, bowel obstruction and trauma. A patient with an ostomy wears an external pouch to collect stool, and there are a few different types:

Transverse Colostomies
These colostomies are in the upper abdomen, allowing stool to leave the body before it reaches the colon.

Loop colostomy: Looks like one large stoma, but it has two openings side by side: one for stool and one for mucus.

Double barrel colostomy: Two separate stomas, one produces stool and one produces mucus.

Sigmod and Distal Colostomies
Colostomies in this area pass formed stool. Sigmoid colostomies are the most common type and are typically done when the patient is being treated for colon cancer. Typically these are single-stoma colostomies, but could be double-barrel.

Ileostomy
Usually located on the right side of the body, these types of ostomies put out liquid content and contain a lot of digestive enzymes (which can be quite harmful to the skin). Your patient with ulcerative colitis (and had the colon removed) will have an ileostomy.

TPN
Total parenteral nutrition (TPN) is used to provide nutrition to patients who are unable to take food in via the gastrointestinal system. It is not ideal, so it is not considered until all other options have been exhausted. Here are the takeaways you need to know about TPN:
• TPN has to be administered through a central line (PICC lines count!)
• It is mixed daily based off that morning’s lab results
• It is typically hung each night with the 2100 meds and runs continually for 24 hours
• It carries a high risk of infection (lots of sugar and other goodies in that bag!)
• It is not compatible with anything else, so you’ll need a dedicated line
• Monitor for signs of fluid overload as each bag is about 2 liters
• The bags will usually contain all the electrolytes and vitamins the patient needs, though additional electrolyte supplementation may be needed
• If your patient has had high blood sugars, the TPN bag may contain some insulin, so you’ll need to watch for signs of hypoglycemia and monitor blood sugar levels at least every four hours

Gastrointestinal Tubes
Gastrointestinal tubes are used to either decompress the bowel or provide nutrition to patients who can’t swallow safely. The
name of the tube will tell you where it’s inserted and where it ends:

**NGT** = nasogastric tube; inserted through the nares/nose and ends in the stomach; used for feeding and decompression. Disadvantage is that it can cause skin breakdown at nares...watch closely! The large-bore tubes are called Salem Sumps and can be used for feeding or decompression (never both at the same time!)

**OGT** = orogastric tube; inserted through the mouth and ends in the stomach; only used on intubated patients and the OGT is typically taped to the ETT for securement; advantage is that it does not cause skin breakdown at the nares like NGTs can, though its pressing against the lip can cause breakdown there. Can be used for decompression or feeding.

**NJT** = nasojejunal tube; inserted through the nares and ends in the jejunum. These are considered “post-pyloric” meaning it’s past the sphincter that empties the stomach contents into the small intestine, so less risk for aspiration. These tubes are not used for decompression...feeding only. You will often see these called “Dobhoff” tubes.

**PEG** = percutaneous endoscopic gastrostomy tube; inserted directly into the abdominal wall and used for more long-term enteral feeding. Not used for decompression, just feeding.

**Specific Tubes for Specific Purposes**

**Salem Sump** is used for decompression, but can also be used for feeding and medication administration. If using for decompression AND meds, clamp the tube and turn off suction for about 30-60 minutes after administration. Can be an NGT or OGT.

**Dobhoff Tubes** are used for feeding only. They have a weighted end that helps them “migrate” past the pyloric sphincter into the jejunum, and are sometimes placed via endoscopy. Because of the end location, these are considered NJTs.

**Sengstaken-Blakemore** tubes have a balloon that exerts gentle pressure against the esophagus. These tubes are used to prevent bleeding of esophageal varices.

**Caring for GI Tubes**

- Watch for signs of skin breakdown
- Flush Q4 hours with sterile water (and prn)
- Flush before and after giving meds (and also between each med)
- Provide good oral care
- Monitor PEG tube insertion site for signs of leakage and infection

**EGD**

Esophagogastroduodenoscopy (EGD) is a procedure used to evaluate the GI tract and perform therapeutic interventions. EGD is used for a variety of reasons:

- Evaluate patient who is experiencing upper GI problems: GERD, dyspepsia, chest pain (non-cardiac), recurrent emesis, dysphagia, esophageal varices
- Biopsy to evaluate things like cancer and infections
- Therapeutic intervention such as stopping GI bleeds, stenting/dilating a narrowed esophagus or placing a gastrostomy tube
- Evaluation of patients at risk of upper GI cancer (such as those with Barrett esophagus)

**How EGD Works**

To perform an EGD, the patient will be sedated so the physician can pass a lighted, flexible scope down through the esophagus into the GI tract. The scope
camera communicates with a monitor so the GI doc can visualize the anatomic structures, look for bleeding or disease and possibly even stop active bleeding. One of the most common things you’ll see an EGD used for is to stop upper GI bleeds.

### What You Need to Do
- NPO 4-8 hrs before procedure
- Monitor patient for respiratory depression related to sedation medications
- Monitor VS during and after procedure

### Colonoscopy
Colonoscopies are done to evaluate problems of the lower GI tract (colon and rectum), including ulcers, tumors, polyps, bleeding and inflammation (such as you get with ulcerative colitis). Colonoscopies are routinely done on individuals at age 50 to screen for cancer.

#### How Colonoscopy Works
Like an EGD, the patient will be sedated for a colonoscopy. The physician inserts a long scope with a video camera on the end into the anus and through the rectum to the colon. At this time, she can take tissue samples or therapeutically treat lower GI bleeds, but only if the patient is actively bleeding (otherwise it’s too difficult to see where the bleeding is coming from).

#### What You Need to Do
- Pt will be on clear liquid diet 24-hrs before procedure
- The night before, the patient will start their “bowel prep” which includes drinking a pretty awful concoction called “Go Lytely.” It’s horrible taste is legendary (mixing with Crystal Light seems to help quite a bit) and it makes the patient have many bowel movements until they’re simply passing clear fluid. Sometimes you’ll need to give enemas “until clear” if the Go Lytely hasn’t done the trick. Many patients will balk at the taste of the Go Lytely, and it is very important that they understand that the GI physician will not perform the colonoscopy the next morning if they have not completed their bowel prep.
- Monitor patient for respiratory depression related to sedation meds (end-tidal carbon dioxide monitoring should be standard protocol)
- Monitor VS during and after procedure
NEUROLOGY BASICS

brought to you by

STRAIGHT A NURSING
Degenerative Neurological Disorders - Overview
By their definition, degenerative neurologic disorders get worse over time. Some progress quickly while others progress over decades. A major goal of intervention is to help the client achieve an optimal level of functioning in light of their neurological deficits.

Dementia
Dementia refers to the loss of memory, reasoning, judgment and language to such an extent that it interferes with daily life. How the changes come about is a key factor in determining if the dementia is chronic or temporary. Patients with dementia experience impairment of cognitive activities and may undergo behavioral/personality changes as well (depending on the area of the brain affected). The causes and types of dementia are numerous. The most common form in people over 65 is Alzheimer’s Disease (AD).

Alzheimer’s Disease
AD is a progressive degenerative disease of the cerebral cortex and hippocampus that presents as progressive memory loss. Recall that the cerebral cortex is the part of the brain that plays a key role in memory, attention, perceptual awareness, thought, language and consciousness. Researchers recognize two forms of AD: familial and sporadic. In the familial form genetics cause the disease (rare). In sporadic AD (most common type) genes don’t cause the disease but may influence the risk of developing the disease. Not much is known about the cause of the disease, but four factors are thought to play a role: neurochemical transmitters (such as deficiencies in acetylcholine), somatostatin, substance P, and norepinephrine.

The characteristic microscopic findings of AD include senile plaques, which are collections of degenerative presynaptic endings along with astrocytes and microglia. These plaques are more numerous in the cerebral cortex and hippocampus.

The brain tissue of Alzheimer’s patients has three distinguishing features:
1) neurofibrillary tangles formed of cytoskeletal intermediate filaments
2) beta-amyloid plaques (deposits of protein-like substances)
3) granulovascular degeneration of neurons

The brain mass of an Alzheimer’s patient atrophies and the brain has narrowed gyri and widened sulci mainly in the frontal and parietal regions.

<table>
<thead>
<tr>
<th>Clinical Manifestations of Early-Stage AD</th>
<th>Clinical Manifestations of Mid-Stage AD</th>
<th>Clinical Manifestations of Late-Stage AD</th>
</tr>
</thead>
<tbody>
<tr>
<td>memory loss, especially recent</td>
<td>speech problems</td>
<td>bedridden</td>
</tr>
<tr>
<td>neglect of responsibilities</td>
<td>nonsense talkativeness</td>
<td>spastic paralysis/contractures</td>
</tr>
<tr>
<td>carelessness</td>
<td>complete disorientation</td>
<td></td>
</tr>
<tr>
<td>job performance suffers</td>
<td>restless/wandering</td>
<td></td>
</tr>
<tr>
<td></td>
<td>failure to recognize</td>
<td></td>
</tr>
<tr>
<td></td>
<td>movement problems</td>
<td></td>
</tr>
</tbody>
</table>

Communicating with an Alzheimer’s Patient
Successful communication with an Alzheimer’s patient revolves around being a patient listener. A few key guidelines include:
- Reduce distractions.
- Be patient.
• Offer alternatives if the individual is having trouble getting the words out and appears to want help.
• Pay attention to body language. Sometimes you can learn more by watching facial expressions, listening to tone and noticing gestures.
• Speak slowly and clearly.
• Keep it simple, one thought at a time.
• Ask a question and wait patiently for a response; resist the urge to ask follow-up questions.
• Avoid use of language that may be difficult to interpret such as sarcasm, irony or idioms such as “it's raining cats and dogs out there.”

Drug Treatment for Alzheimer’s
Acetylcholinesterase inhibitors lead to increases in acetylcholine levels, which can help AD patients with things like memory and judgment, confusion, depression, anxiety and psychoses. These drugs can improve symptoms, but they do not slow or halt the disease:
• Rivastigmine (EXELON): for mild to moderate dementia; SE = dizziness, weight loss, flu-like symptoms
• Donepezil (ARICEPT): for mild to moderate dementia; SE = N/V, diarrhea, muscle cramps, weight loss
• Galanthamine (RAZADYNE, REMINYL): for mild to moderate dementia; SE = dizziness, N/V, weight loss, and orthostatic hypotension
• Tacrine (COGNEX): for mild to moderate dementia; SE = N/V and liver toxicity

Some other drugs/therapies:
• Glutamine blocker is a newer type of drug that is useful in later stages and allows combo therapy (ex: Memantine )
• Ginkgo biloba extract has been shown to be better than placebo (patients on anticoagulants should not take)
• Vitamin E slows progress by about 6 months
• NSAID naproxen being studied and looks promising
• New therapies are continually being researched

Diagnosing AD
Sadly, AD can not be confirmed until the patient has died and an autopsy can be performed. However, tests are conducted to rule out other disorders:
• PET scan measures the metabolic activity of the cerebral cortex
• CT scan can show more brain atrophy than occurs in normal aging
• MRI evaluates the condition of the brain and rules out intracranial lesions
• EEG evaluates the brain’s electrical activity and may show brain wave slowing late in the disease. It also helps to differentiate tumors, abscesses, and other intracranial lesions
• CSF analysis helps determine whether signs & symptoms stem from a chronic neurological infection
• Cerebral blood flow studies may detect abnormalities in blood flow to the brain

The physician will also take a detailed history, and based on what the findings are, can typically diagnose AD with about 90% accuracy. But again, definite confirmation can only be done via autopsy. These warning signs published by the Alzheimer’s Association can help physicians (and family members) spot the early signs.

Management of Patient’s with AD
Management of AD involves protecting the patient from injury, assisting with memory and providing respite for caregivers.

Parkinson’s Disease
Parkinson’s Disease (PD) is a movement disorder characterized by rigid muscles that make it difficult to move normally. Individuals with Parkinson’s may progress to memory loss and dementia in later stages.
PD affects 1 to 1.5 million people in the U.S. It most commonly strikes people over the age of 50, but can affect people even before age 40. Approximately 10 percent of PD cases are estimated to be young-onset (like Michael J. Fox).

The etiology of this horrible disease is unknown, but one source says that some cases result from exposure to toxins that destroy cells in the substantia nigra of the brain, so stay away from manganese dust and carbon monoxide. The disease is mainly caused by a deficiency of dopamine. Recall that dopamine is an important neurotransmitter in the brain and is essential for governing movement, balance and walking. By the time symptoms have developed, 80-90% of the dopamine producing cells have been lost.

**Symptoms of Parkinson’s Disease**
- Muscle rigidity
- Tremor
- Slow movement (bradykinesia) or loss of movement (akinesia)
- Difficulty with balance or walking

**Associated Problems of Parkinson’s Disease**
60-90% of people with PD will develop some difficulty speaking (dysarthria). This is often characterized by weak, slow or uncoordinated speaking. Often, speech problems worsen over time. Speech therapists may help with this but there is limited data about which techniques are most successful.

50% develop swallowing problems (dysphagia). Food or fluids may spill from the mouth and the patient is more prone to choking on food or drink (and even on their own saliva), essentially aspirating food or fluids into the lungs. It is usually easier for this patient to swallow thickened fluids. A speech therapist performs a swallow evaluation to determine what type of food and liquid consistency is best for the patient. A good strategy when feeding the Parkinson’s patient is to have them sit up and swallow twice with each bite. They should concentrate on eating and take small bites. Food should be pureed or chopped small (usually called “diced” in the hospital).

More than 50% of Parkinson’s patients have mild intellectual changes, and about 20% have more substantial cognitive impairment. The memory problems with PD are typically milder than with AD. The patient with PD may have difficulty concentrating, acquiring new information and recalling names.

**Prognosis**
There is no cure for Parkinson’s, but symptoms can be managed with medications (levodopa & carbidopa are most common) or surgical procedures where part of the brain is destroyed or stimulated.

**Treatments**
- Selegiline (ELDEPRYL) is an MAO inhibitor that may be started at the time of diagnosis. It blocks the breakdown of dopamine.
- As the disease progresses beyond minor symptoms, additional drug treatment may be indicated.
- Dopamine cannot cross the blood-brain barrier, so we give the precursor L-DOPA (levodopa). Levodopa is rapidly metabolized so only a small fraction of the drug is available to the CNS, so we give an inhibitor to the metabolism (carbidopa).
- Sinemet, a combination of levodopa and carbidopa, is usually the drug most doctors use to treat Parkinson’s disease patients. While side effects including large uncontrollable movements called “dyskiniesias” and confusion may develop after about 8 years, drug therapy for Parkinson’s typically provides relief for about 10-15 years or more. Keep the drug dose low and timing regular to minimize the
“on-off phenomenon” where the patient responds to the meds ok and then at some point before the next dose the dopamine level seems to drop below a threshold and the patient develops sudden bradykinesia.

- **Other meds**: entacapone, anticholinergics, amantadine

- **Thalamotomy** is a surgical procedure that surgically destroys cells in the thalamus to correct a disabling tremor in the hands or arms. It is used for patients with few other symptoms. Risks are considered low, and immediate improvement is seen in 80-90 percent of patients after the operation. Full recovery takes about 6 weeks.

- **Pallidotomy** surgically destroys certain cells in the globus pallidus (part of the brain that controls movement). The operation may help correct problems in slow movement, tremor and imbalance. It is performed with the patient conscious. Generally, the most dramatic result is the decrease in dyskinesia. Post-surgical recuperation is similar to that of thalamotomy.

- **Stimulator implantation** involves the implantation of an electrode with the tip in the target site. It is connected to a wire running beneath the skin that leads to a stimulator placed in the chest wall. When electrical current is activated it modifies the function of the target site. The stimulator can be adjusted externally using a programmer.

- **Fetal tissue implantation** is an experimental technique used to restore the brain's ability to produce dopamine. It is rife with controversy and we may see genetically engineered cells for this type of surgery in the future. To date, long-term studies are not available on the efficacy of this procedure. Preliminary findings indicate that the surgery can dramatically decrease the need for meds, although the improvement may not begin until 6 months after the surgery and may not peak for 12 to 24 months.

- Physical therapists can help develop and monitor a home exercise program. A good exercise routine should include strengthening and flexing all limbs, stretching legs and feet, walking, facial and breathing exercises, and specific exercises to gain better control in swallowing.

- Occupational therapists can help the person accomplish everyday activities.

- Speech therapists can help the person with PD improve voice volume, quality and articulation.

- Therapeutic exercises including verbalizations and tongue movements.

- Where speech is severely impaired, a machine or computer-generated voice could be used.

---

**Multiple Sclerosis**

Multiple Sclerosis (MS) is an autoimmune process in which immune cells think myelin is foreign and mount an immune response against it. This results in the progressive demyelination of the white matter of the brain and spinal cord, leading to widespread neurologic dysfunction. The structures usually involved are the optic and oculomotor nerves and the spinal cord tracts. Depending on where nerve damage occurs, MS can affect vision, sensation, coordination, movement, and bladder & bowel control.

Approximately 350,000 people in the US have MS, and 50% of them will need a walking aid within 15 years of diagnosis. The prognosis of the disease varies. It may progress rapidly causing death or disability by early adulthood, but about 70% of people with MS lead active, productive lives with prolonged remissions.

**Who gets MS?**

MS is more common in women and generally first occurs in people in their 20s and 30s. Evidence shows that people of northern European descent (especially Scandinavian) may be genetically predisposed to MS, and many of these people have settled in the northern U.S. The disease does appear to run in families, with as many as 20% of people with MS having at least one affected relative. Compared to the general population, first-degree relatives (children, siblings) of people with MS have a 20 to 40-fold increase in risk of eventually having MS. However, it’s not just one gene that passes down the disease…multiple genes likely contribute.

**Causes of MS**

The exact cause of the disease is unknown, but there are several theories. It may be caused by a slow-acting viral infection,
an autoimmune response of the nervous system or an allergic response. Exposure to common bacteria or viruses (such as the human herpes virus 6) may trigger the disease in some people with a genetic tendency. This may explain why in identical twins, only one twin develops MS 70% of the time. Because MS is twice as common in women as in men, it’s been suggested that hormonal factors predispose women to the disease. This theory has not been confirmed, but one study showed that symptoms of MS were reduced during the second and third trimesters when estrogen levels are high.

Other possible causes include trauma, anoxia, toxins, nutritional deficiencies, vascular lesions and anorexia nervosa, all of which may help destroy axons and the myelin sheath.

**Types of MS**

**Benign:** In 10 to 15 percent of patients, symptoms are mild to moderate, don’t worsen and don’t lead to permanent disability.

**Relapsing-remitting:** About 85% of people with MS begin with this form, and more than half have this form of the disease at any one time. It is characterized by one or two flare-ups every 1 to 3 years, followed by periods of remission. Such flare-ups, which may include any of the symptoms of MS alone or in combination, typically appear suddenly, last a few weeks or months, and then gradually disappear. Symptoms may worsen with each recurrence.

**Primary progressive:** From the onset of symptoms, neurological function deteriorates without periods of remission. About 10% of patients begin with this disease pattern.

**Secondary progressive:** Usually after years of having relapsing-remitting MS, at least half will enter a stage of continuous deterioration. Relapses may still occur.

**Progressive relapsing:** This is primary progressive MS with the addition of sudden episodes of new symptoms or worsened existing ones. This form is quite rare.

**Treatments for MS**

- Steroids (prednisone)
- Immunomodulating agents (Aubagio, Gilenya)
- Betaseron, Avonex, Rebif (interferons) and Copaxone (synthetic)
- Immunosuppressive drugs such as Mitoxantrone
- Ocrevus (a monoclonal antibody) has recently received FDA approval (as of 2017)
- Caution patient that immune boosting herbs may worsen the disease

Treatments for MS aim at shortening exacerbations and relieving neurologic deficits. Corticotropin, prednisone or dexamethasone are used to reduce edema of the myelin sheath during exacerbations, relieving symptoms and hastening remission. These drugs do not prevent future exacerbations.

Currently (as of 2009) the preferred treatment during an acute attack is a short course of methylprednisolone (with or without a short prednisone taper). Interferons, immunomodulating agents and other medications (such as Ocrevus) may also be given to decrease the frequency of relapses.

During acute exacerbations, supportive measures include bedrest, massage, prevention of fatigue and pressure ulcers, bowel and bladder training, treatment of bladder infections with antibiotics, physical therapy and counseling.

**Myasthenia Gravis**

Myasthenia gravis (MG) is a rare autoimmune disease that produces sporadic, progressive weakness and abnormal fatigue of voluntary skeletal muscle. It usually affects muscles in the face, lips, tongue, neck and throat, but can affect any muscle group including bulbar muscles, ocular muscles, and respiratory muscles. Muscle fibers may eventually degenerate and weakness becomes permanent. When this happens to the respiratory muscles it can be life-threatening as the patient will no longer be able to breathe on his own.
The cause of MG is unknown, but it often occurs along with autoimmune disorders and thymus disorders. For some reason that no one has yet figured out, the patient's blood cells and thymus gland produce antibodies that block, destroy or weaken the neuromuscular junction. During normal neuromuscular transmission, acetylcholine is released to diffuse across the synapse, and receptor sites in the motor endplate react and depolarize the muscle fiber. The depolarization spreads throughout the muscle fiber, causing contraction. In MG, antibodies attach to the acetylcholine receptor sites making them insensitive to acetylcholine, thereby blocking neuromuscular transmission.

**Treatment of Myasthenia Gravis**
- **Cholinesterase Inhibitors**: cause considerable improvement in some patients, but little to none in others. Strength rarely returns to normal. Most common ChE inhibitors are Pyridostigmine bromide (MESTINON) and neostigmine bromide (PROSTIGMIN).
  - **SE of Mestinon**: muscarinic reaction, including abdominal cramping and skeletal muscle fasciculations (small, involuntary contractions), weakness from excessive depolarization
- **Thymectomy** is recommended for most patients with MG. The maximal favorable response generally occurs 2-5 years after surgery. Response is unpredictable.
- **Corticosteroids**: Marked improvement or complete relief of symptoms occur in more than 75% of patients treated with prednisone with the other 25% showing some improvement. About one-third of the patients become weaker within 7-10 days of starting prednisone, but this only lasts for up to 6 days. The major disadvantage of this therapy are the side effects.
- **Immunosuppressant Drugs (decrease antibodies to AcH receptor)**
  - **Azathioprine** reverses symptoms in most patients, but the effect is delayed 4-8 months
  - **Cyclosporine** inhibits predominantly T-lymphocyte-dependent immune responses and is sometimes beneficial in treating MG. Most patients with MG improve 1-2 months after starting the drug, and improvement is maintained as long as the therapeutic doses are given. SE include renal toxicity and HTN.
  - **Cyclophosphamide** has been used IV and PO. More than half of the patients become asymptomatic after one year. SE are common.
- **Plasma exchange** is used as a short-term intervention for patients with sudden worsening of MG symptoms, to rapidly improve strength before surgery, and as a chronic intermittent treatment for patients who are refractory to all other treatments. Improvement of symptoms typically lasts for 1-2 months.
- **Intravenous Immune Globulin (IVIG)**. Several groups have reported favorable responses to high-dose (2 grams/kg infused over 2-5 days) IVIG. Improvement occurs in 50-100% of patients, usually beginning within 1 week and lasting for several weeks or months. Very expensive $$$.

**The Big Picture**
These are only a few of the many neurologic diseases, but there are common themes:
- **Safety**
  - Are pulmonary muscles weak? (think atelectasis, respiratory distress and risk for pneumonia)
  - Are the esophageal muscles weak? (think aspiration due to inability to safely manage secretions or swallow)
  - Is the brain dysfunction affecting judgment or control of movement (think injury, falls)
  - Is innervation to bowel/bladder compromised? (think constipation, urinary retention)
  - Are skeletal muscles affected? (think immobility, skin breakdown, constipation and pneumonia)
Brain Attack, CVA, Stroke

Most strokes are caused by thromboembolisms, meaning they are embolic strokes related to clots. As of 2017, stroke is the 5th leading cause of death in the United States. If that isn’t enough, here are some other devastating truths about stroke:

- Stroke kills about 140,000 Americans each year.
- About 87% of all strokes are ischemic strokes, in which blood flow to the brain is blocked.
- Someone in the United States has a stroke every 40 seconds. Every 4 minutes, someone dies of stroke.
- Risk of having a first stroke is nearly twice as high for blacks as for whites, and blacks have the highest rate of death due to stroke.
- Hispanics have seen an increase in death rates since 2013.
- Stroke costs the United States an estimated $34 billion each year. This total includes the cost of health care services, medicines to treat stroke, and missed days of work.
- Stroke is a leading cause of serious long-term disability.

The concepts of perfusion/stasis are the same as for CV disease, as are the risk factors. As a leading cause of death in the US, stroke and is very expensive for both the government and the individual patient. The good news is, the death rate due to stroke has declined due to better risk prevention and management, quicker diagnoses and more timely interventions.

Causes & Pathophysiology of Stroke

High blood pressure, high cholesterol, smoking, obesity, and diabetes are leading causes of stroke. 1 in 3 US adults has at least one of these conditions or habits.

There are three causes of stroke. They can be due to a thrombus, hemorrhage or an aneurysm. The brain has no oxygen reserve, so when an artery is occluded (ischemia) this causes the Na/K pump to no longer work. The Na stays in the cell and since water follows salt, the cell swells. This swelling limits the amount of perfusion that can get to the brain and causes cells to die.

Sadly, people don’t often recognize the signs of stroke, but if you can get the patient to the hospital quickly the chances of survival increase due to the ability to intervene appropriately and, hopefully, before permanent damage sets in.

Stages of Stroke

- TIA (transient ischemic attack): These symptoms resolve on their own in minutes or hours. They are caused by microemboli from plaque. They are likely to go on to have a true CVA at some point.
- RIND (reversible ischemic neurological disease): Symptoms persist for 24-48 hours. There is ischemia, but no necrosis and patients can expect a complete recovery.
- Stroke in evolution: 20-35% of patients have symptoms that get worse over the course of the week after the CVA.
- Completed stroke causes permanent neurological damage.

Clinical Manifestations of Stroke

The clinical manifestations have to do with which blood vessel is involved. The most common are the middle cerebral artery and the internal carotid artery.

- Middle Cerebral Artery
  - Contralateral paralysis, paresis, sensory loss
  - Dysphasia, aphasia
  - Spatial perception problems, judgment/behavior
  - Contralateral homonymous hemianopsia

**Signs of Stroke**

ACT “FAST”

F: facial droop
A: arms exhibit drift
S: slurred speech
T: time..call 9-1-1 immediately!
• Internal Carotid Artery
  • Same as above, plus...
  • Ipsilateral visual impairment
  • Ipsilateral Horner’s syndrome (ptosis, miosis, no sweating on same side of face)

With both of these you have decreased LOC, could have seizures and vital signs changes. Visual problems vary with the site of the stroke. Also, the motor and sensory tracts cross over, so a stroke on the left side of the brain is going to cause right-sided weakness.

Prevention of Brain Attack/CVA/Stroke
- Risk factor modification (ex: stop smoking, lose weight, control HTN)
- Anticoagulants
- If the patient has atrial fibrillation, will need to be on anticoagulants
- Aspirin
- Aspirin/extended release dipyridamole
- Heparin not really used that much for prevention, maybe if patient has DVT or a-fib
- Surgical revascularization

Treatment: Evolving Stroke
- Call 911 and get the stroke team activated.
- Stabilize airway, give O2, check BP, get the patient to CT scan within 25 minutes (some even aim for 15 minutes!)
- Give or not to give thrombolytics? Door-to-needle time is 60 minutes.
  - Thrombolytics are only given in ischemic stroke. If the CT is “negative” then this means it is negative for a bleed. That’s because the blood from a hemorrhagic stroke will show on a CT scan and is a quick way to know if you can give the patient thrombolytics. You obviously would NOT give thrombolytics if the patient were actively bleeding into his brain, right? So, if your patient comes in with stroke symptoms, and the CT is negative for a bleed, then this patient will be evaluated for thrombolytics.
- The contraindications for giving TPA are considered when deciding whether or not to treat:
  - Intracranial hemorrhage on CT
  - Suspicion of other active bleeding
  - Prior stroke in past 3 months
  - Neurosurgery or serious head trauma in past 3 months
  - Uncontrolled hypertension (BP > 185 systolic or > 110 diastolic)
  - History of intracranial hemorrhage
  - Seizure at time of stroke
  - Known AVM (arteriovenous malformation), neoplasm or aneurysm
  - Suspected or confirmed endocarditis
  - Increased bleeding time: platelets < 100,000, heparin within 48 hours with elevated aPTT, current use of oral anticoagulants, current use of direct thrombin inhibitors or direct factor Xa inhibitors
  - Blood glucose < 50 or > 400
  - Note that there are several other relative contraindications (one of which is being over 80 years old), but these listed above are the absolute contraindications and are the most important.
- Maintain cerebral perfusion. You achieve this by doing a few things simultaneously:
  - Reduce swelling
    - BP: < 200/120 (<185/110 with TPA). You might think “WOAH”, that BP is high! Well, we want it to be high so the blood can perfuse the brain. Recall your cerebral perfusion pressure equation is CPP = MAP - ICP. You’re aiming for a CPP above 70-80 mmHg.
    - Good ventilation: Note that elevated CO2 is going to cause cerebral edema. When CO2 levels
(reduce swelling cont’d)

rise, blood vessels in the brain dilate, pressures in the brain increase and perfusion decreases. Patients who are really bad off are probably going to be intubated, at least in the acute stage. Once they regain conscious ness (if they do), you’ll wean the ventilator asap.

• Mannitol: This is an osmolar agent used in first 72-96 hours. It pulls fluid out of the cell in order to decrease edema. It is a HIGH-ALERT medication that is typically only given in the ED and ICU. You have to keep a close eye on labs (Na and serum osmolality) and monitor for neuro changes.
• Steroids: May be used with SAH (subarachnoid hemorrhage)
• HOB: 30-degrees to help CSF drain effectively
• Glyburide: Studies show it reduces swelling
• Prevent vasospasm: nimodipine (a calcium channel blocker) is used for bleeds but not ischemic strokes. Nimodipine is a bit labor intensive because you usually give it every two hours (sometimes q 4-hr) and the pills are HUGE. Note that one of the things that nimodipine does (besides prevent vasospasm) is drop blood pressure. So, it’s often a balancing act of giving the nimodipine and then having to titrtrate vasopressors up and down.
• Protect brain cell: Ensure no fever, use neuroprotectants (experimental drugs), monitor for vasospasm or worsening stroke. For each 1-degree Celsius increase in temp, the negative effects from the stroke increase. Note that many times acetaminophen does not work very well for controlling “neuro temps.” You can likely anticipate having to use external cooling measures in order to decrease temperatures.
• Surgery: External decompressions, remove clot with hemorrhagic stroke.
• Seizure prevention with anti-seizure medication (Keppra is a common one).

Rehab/Treatment: Evolving and Completed Stroke

In this stage you will protect your patient from complications of immobility and decreased neuromuscular status. This is a multidisciplinary approach with a lot of people involved.

• No aspiration (HOB up, NPO/PEG or feeding tube, speech therapy, swallow evaluation)
• No pressure sores (turn pt, OOB to chair as soon as possible, get PT in there asap)
• No contractures (position of maximal function, ROM and PROM)
• Bowel and bladder function support
• Communication support
• Interaction with environment when visual field impaired
• Monitor for depression

\[ \text{CPP} = \text{MAP} - \text{ICP} \]
\[ \text{GOAL} > 70-80 \text{ mmHg} \]
**Overview of Diabetes & Insulin**

According to 2014 data from the CDC, diabetes mellitus affects 29.1 million Americans, with 8.1 million of them undiagnosed. It is the 7th leading cause of death in the U.S., but we suspect this is a low estimate as DM is considered to be under-reported on death certificates as a contributing cause.

Diabetes mellitus is an autoimmune disease of either insulin resistance or deficiency, and it is characterized by changes in carbohydrate, fat and protein metabolism. When you eat, the pancreas secretes insulin in response to your blood sugar rising. Insulin helps “unlock” the cell so the sugar can travel into the cell and be of use to the body. Remember, insulin is a hormone and hormones make metabolic processes happen in the body. Hormones get stuff done!

Why is insulin important? Most tissues need insulin in order for “glucose uptake” to occur. Glucose uptake refers to the ability of the cell to use glucose...note that the brain and liver don’t need insulin for glucose uptake to occur (these cells use a different type of transporter that is not related to insulin, but we don’t need to get that fancy here). Please do not take this to mean that the brain does not need glucose, it most certainly does!

**The Types of Diabetes**

In Type 1 Diabetes Mellitus, the pancreas does not secret enough insulin, and the ability to do so decreases as the pancreatic beta cells are destroyed. The onset is typically childhood and by the time the diagnosis occurs, no beta cells remain, so these folks are considered “insulin-dependent.”

In Type 2 Diabetes Mellitus, the body can still produce insulin. Most cases of DM are Type 2 (about 80-95%). In Type 2, some insulin is still produced by the body but it is either not enough or the cells are resistant. Onset for Type 2 is typically adulthood, but note that as the child obesity problem continues, more and more children are being diagnosed.

And, there is even a Type 1.5 Diabetes Mellitus...it’s just now being understood as a subset of DM that shares traits of both type 1 and 2. It’s called LADA (latent autoimmune diabetes in adults). It typically shows up after age 30 and shares more commonalities with Type 1 than Type 2 in that the patient will test positive for GAD antibodies, which destroy the body’s GAD (glutamic acid decarboxylase) cells. LADA will usually require insulin therapy and it is important to note that in the early stages it is often misdiagnosed as Type 2 DM. Initially the conservative treatment for Type 2 DM will help lower blood glucose levels (diet modification and oral meds). However, the beta cells will continue to be destroyed and ultimately the patient will need insulin. As we learn more about LADA we hope to diagnose people accurately from the beginning so that beta cell function can be preserved as long as possible (early insulin therapy seems to be the key to this.)

And here’s an interesting side note for you to ponder. Some research is indicating that Alzheimer’s is actually Type 3 DM in that it appears that it is the result of the brain’s inability to utilize glucose. [Check it out here](#) if you’re interested!

There’s also gestational diabetes, drug-induced hyperglycemia secondary to steroids...and maybe others. We are learning more and more about diabetes so keep an eye out for interesting new research as it comes available.

**Diagnosing Diabetes**

To diagnose diabetes or prediabetes, the patient will have their blood sugar checked in three different states:

1) Fasting blood sugar on two different occasions
   - Fasting blood sugar > 125 mg/dL = diabetes
   - Fasting blood sugar 100-125 mg/dL = prediabetes

Approximately one third of children in the United States are classified as overweight or obese. [Source National Health and Nutrition Examination Survey](#)
2) Random blood sugar test  
- BS > 200 with symptoms (polydipsia, polyuria, polyphagia, weight loss) = diabetes

3) Glucose tolerance test (patient takes in 75 grams glucose and their BS is tested two hours later)  
- BS two hours after taking the glucose: ≥ 200 mg/dL = diabetes  
- BS two hours after taking the glucose: 140-199 = prediabetes

**What is Metabolic Syndrome?**

*Metabolic syndrome is a group of risk factors that increase the patient’s risk for diabetes, heart disease and stroke. These risk factors are:*

- elevated blood glucose levels  
  - a large waistline  
  - high triglycerides  
    - low HDLs  
    - hypertension

**Diagnostic Screening**

It is important to screen individuals for Type 2 diabetes so that it can be treated before it starts causing problems (more on this later on). It is currently not recommended to routinely screen for Type 1 diabetes, because it is the presenting signs/symptoms that prevail. Below are the general diagnostic screening guidelines currently considered best practice by the esteemed endocrinologists at the American Diabetes Association

- Annual screening for asymptomatic overweight adults (BMI ≥ 25) with one or more additional risk factors:  
  - Elevated A1C (above 5.7%) or elevated blood sugar with prior testing  
  - Acanthosis nigricans (dark patches of skin that are velvety in texture)  
  - Cardiovascular disease  
  - Close family member with DM Type 2  
  - Low HDL (< 35 mg/dL) and/or high triglycerides (>250 mg/dL)  
  - High-risk ethnicity: African American, Native American/Alaska Native, Hispanic/Latino, Asian American, and Pacific Islander  
  - Hypertension  
  - Sedentary lifestyle  
  - Women with polycystic ovary syndrome  
  - History of gestational diabetes or delivery of a baby weighing > 9 lb  
- Screen individuals with no risk factors every three years beginning at age 45

**Signs and Symptoms of Diabetes**

Type 1 DM: weight loss, polyuria, polydipsia, polyphagia  
Type 2 DM: obesity or weight loss, fatigue, nausea, vomiting, nocturia, pruritus, vaginitis

**Associated Labs**

**Elevated glucose:** This one is a no-brainer. As the body’s inability to utilize or produce insulin continues, the glucose will not be able to enter the cell and will, instead, hang out in the vascular space where it does the body NO good and LOTS of harm.

**Dyslipidemia:** Patients with diabetes have lipid abnormalities...namely low HDL and high triglycerides. The causes are
complex and are likely to be related to the effects insulin has on liver apoprotein production, regulation of lipoprotein lipase and other actions of insulin on adipose tissue and muscle.

**Abnormalities in Na level:** Diabetes can cause both hypo and hypernatremia via numerous mechanisms. The most simple and most common is that when blood sugar is high, serum osmolality is increased, which causes water to move out of the cells leading to hyponatremia by dilution (we expect it to drop by 2 points for every 100 increase in blood glucose). Recall that high blood sugar has a diuretic effect on the body (one of the symptoms is polyuria, remember?)....so these hypotonic renal losses (when the loss of water exceeds the loss of sodium) lead to hypernatremia, which is a late sign (and very serious!)

**Low potassium:** Hypokalemia results for a variety of reasons in the presence of diabetes. When insulin is injected, there’s a shift of potassium from the extracellular to the intracellular space (this is because potassium “hitches a ride” on sugar molecules). People with diabetes can also have malabsorption syndromes, which leads to GI losses of potassium. In addition, osmotic diuresis will cause potassium levels to be decreased as well. And, lastly, hypomagnasemia causes hypokalemia due to the low magnesium level signaling the kidneys to secrete more K.

**Low magnesium:** Magnesium levels can be low due to osmotic diuresis.

**Elevated HbA1c:** Sugar is very “sticky” so it actually adheres to hemoglobin over the whole life span of the RBC (which is 2-3 months). We can look at how much sugar is “stuck” to the hemoglobin to determine the patient’s average blood sugar level over the past few months. We refer to this as the “hemoglobin A1c” or usually just the “A1c.”

<table>
<thead>
<tr>
<th>HbA1c</th>
<th>Avg BS mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>6%</td>
<td>135</td>
</tr>
<tr>
<td>8%</td>
<td>205</td>
</tr>
<tr>
<td>10%</td>
<td>275</td>
</tr>
<tr>
<td>12%</td>
<td>345</td>
</tr>
</tbody>
</table>

**Low pH:** When blood sugar levels are very high (as in diabetic ketoacidosis) the patient is in a metabolic acidosis. We’ll talk more about DKA and HHNS later on.

**Low HCO3:** Low bicarbonate (HCO3) results as it is depleted in an effort to correct the acidosis.

**Elevated BUN & Cr:** Over time, high blood sugar levels damage the kidneys leading to renal failure.

**Diabetes Management**

**Diet**
The very first method used to manage diabetes is diet. Both Type 1 and Type 2 diabetics will need to be on a carb-controlled diet when hospitalized. We call it a consistent carb-controlled diet, meaning each meal has roughly the same number of carbs (often 60 grams).

However, for general lifestyle modification, the American Diabetes Association lists the following goals for nutrition therapy:

1) Promote and support healthful eating that emphasizes a variety of nutrient-dense foods in proper portion sizes. Mediterranean and plant-based diets are highlighted by the ADA as “healthful eating patterns.”
2) Maintain a healthy weight

**Diabetes Goals**

Fasting blood sugar 90-130
HbA1c < 7%

Note that in the hospital setting, we let blood glucose ride a little higher due to the MUCH higher danger of hypoglycemia. The general rule of thumb is to only give insulin for a blood glucose > 150 mg/dL.
3) Meet glycemic, blood pressure and lipid goals (individualized for each patient)
4) Minimize complications of diabetes
5) Maintain the pleasure of eating and cultural preferences

Let's take a quick look at some nutrition basics as they relate to diabetes:
- Carbohydrate sources: vegetables, low-glycemic fruits and whole grains.
- Protein: Choose low-saturated fat sources; protein does not raise blood sugar, but it does stimulate insulin release.
- Fat: choose naturally-occurring fats like those from nuts, seeds and avocado. Don’t overdo it!
- Sodium: If you have high blood pressure or are obese, you’ll want to decrease sodium intake.
- Sugar: Avoid “added sugar” and high-glycemic fruits.

**Weight Management**
Research shows that weight loss has a direct reduction on blood sugar levels. A fasting blood sugar of 108-144 means the patient needs to lose about 10 kg or 16% of bodyweight; a fasting blood sugar of > 250 means that the patient needs to lose a lot more...about 26 kg or 40% of bodyweight. In fact, a weight loss of up to 30 pounds can reduce mortality significantly...about 25% over 12 years, according to the journal Diabetes Care (2000; 23:1499). And, a recent study finds that even a weight loss of just 5-10% can reduce A1c. It’s clear that achieving and maintaining a healthy weight are key components to diabetes management.

**Exercise**
Another key component to blood sugar control is exercise. Not only does it help with weight management, exercise brings those glucose transport channels to the surface so that the glucose can move inside the cell. Educate your patient that if they are going to be working out a particular muscle group to avoid injecting into that area. The increase in blood flow can cause the insulin to be absorbed too quickly (and cause a dangerous drop in blood sugar). Also, avoid injecting insulin into the arm with a BP cuff for the same reason.

**Pharmacological Management**
Blood sugar levels can be controlled by using insulin or oral medications. Let’s start with a quick overview of insulin types:

<table>
<thead>
<tr>
<th>insulin</th>
<th>type</th>
<th>onset</th>
<th>peak</th>
<th>duration</th>
<th>misc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humalog &amp; Novalog</td>
<td>rapid-acting</td>
<td>10-15 m</td>
<td>1-2 hr</td>
<td>3-4 hr</td>
<td>give 15 mins before meal</td>
</tr>
<tr>
<td>Humalin R. &amp; Novalin R.</td>
<td>short-acting</td>
<td>30-60 m</td>
<td>2-4 hr</td>
<td>5-7 hr</td>
<td>the only kind given IV</td>
</tr>
<tr>
<td>Humalin N. &amp; Novalin N.</td>
<td>intermediate</td>
<td>1-4 hr</td>
<td>6-12 hr</td>
<td>24-48 hr</td>
<td>also called “NPH”; cloudy solution</td>
</tr>
<tr>
<td>Lantus &amp; Levemir</td>
<td>long-acting</td>
<td>1 hr</td>
<td>none</td>
<td>24 hr</td>
<td>do not mix with other insulins</td>
</tr>
<tr>
<td>Humulin 70/30 (+ others)</td>
<td>combo</td>
<td>varies</td>
<td>varies</td>
<td>varies</td>
<td>a combo of rapid and intermediate</td>
</tr>
</tbody>
</table>

**Oral Medications**
- Sulfonylureas: stimulate the pancreas to secrete insulin while also increasing sensitivity to insulin at the receptor sites. Common ones are Glyburide, Glibizide and Repaglinide.
  - requires some pancreatic function
  - effectiveness of warfarin is reduced if taken together
  - can cause photosensitivity
  - bad side effects with alcohol (abdominal cramps, headache, hypoglycemia, flushing)
  - dosing varies; some are once per day, others are before each meal
• Biguanides: decrease glucose production in the liver while increasing glucose utilization by the cells. You’ll see this used a lot in early stages of DM2 and the main one here is Metformin. One of the big issues with this drug is that you can’t take it AND get IV contrast. The patient will have to be OFF metformin the day of the scan and for 48-hrs post IV contrast administration, otherwise it’s bye-bye kidney function.
  • requires some pancreatic function
  • common undesirable side effect is diarrhea (give with meals to help alleviate this)
  • common desirable side effect is weight loss
  • hold for IV contrast
  • may cause metallic taste in mouth (transient)
  • requires good kidney function, otherwise it builds up causing lactic acidosis

• Meglitinides: stimulate the release of insulin, but the mechanism of action has to do with potassium and calcium channels. It is often used in conjunction with metformin or a thiazolidinedione. Common ones are nateglinide (Starlix) or repaglinide (Gluconor).
  • requires some pancreatic function
  • effects increased by a lot of other drugs (warfarin, NSAIDs, simvastatin, etc…)
  • effects decrease by a lot of drugs (corticosteroids, thyroid drugs, CCBs)

• Thiazolidinediones: easily recognized because they end in the suffix “-zone.” A common one is Actos/pioglitazone (the other is Avandia/rosiglitazone). These drugs improve sensitivity to insulin, so insulin must be present. You’ll often see these drugs used in combo with other antidiabetics, namely Metformin (a sulfonylurea) and/or insulin.
  • requires insulin in order to work
  • do not give in liver disease
  • Avandia causes an increase in LDL and HDL; Actos increases HDL and lowers triglycerides
  • can cause life-threatening CHF
  • once-per-day dosing
  • side effects include weight gain, edema, headache and respiratory infections

• Alpha-glucosidase inhibitors: used for Type 1 diabetes to inhibit the release of the alpha-glucosidase enzyme, which reduces the absorption of dietary glucose. A common one is Precose/acarbose.
  • used for Type 1 DM along with dietary therapy
  • can cause abdominal pain, diarrhea and flatulence
  • avoid using with amylase and pancreatin; may decrease the absorption of digoxin
  • sign of overdose is an increase in flatulence, diarrhea and GI discomfort
  • take once per day with a meal

• Incretin-inhibitors: used along with diet and exercise to control Type 2 DM; often used with Metformin, Actos or a sulfonylurea. Januvia works by slowing the inactivation of incretin hormones, which play a role in glucose homeostasis.
  • can increase digoxin levels
  • excreted in urine, so use cautiously in renal impairment
  • once-per-day dosing, take with or without food

• SGLT2 inhibitors: increases renal glucose excretion, increases insulin sensitivity and uptake in muscle cells, decreases gluconeogenesis and improves the release of insulin. Invokana/canagliflozin is a common one.
  • take daily before first meal of the day
  • not to be used in renal impairment
  • may cause orthostatic hypotension
  • can cause genital infections and frequent urination
• DPP-4 enzyme inhibitors: inhibit the enzyme that breaks down the hormones responsible for insulin release and glucagon inhibition, resulting in more insulin release and less glucagon release (Januvia/sitagliptin)
  • side effects include GI upset, headache, upper respiratory tract infection
  • can be taken with or without food

Other Injectable Medications
• Incretin mimetics/GLP-1 receptor agonists: mimic the action of incretin, leading to insulin secretion and better blood glucose control. The two most common are Byetta and Trulicity. They are used to treat Type 2 DM.
  • Byetta/exenatide
    • given SubQ but is NOT insulin
    • dosing is twice per day, within 60 mins of morning and evening meal
    • can cause diarrhea, nausea, vomiting
    • may decrease absorption of PO meds, especially anti-infectives and oral contraceptives
  • Trulicity/dulaglutide
    • given SubQ but is NOT insulin
    • dosing is once per week
    • prescribed when diet/exercise aren’t controlling blood sugar levels
    • can cause stomach pain, diarrhea, loss of appetite
    • can aid in weight loss

• Non-insulin hormones: Symlin/pramlintide works by slowing gastric emptying, suppressing glucagon and regulating food intake. It is used in diabetics whose blood sugar can’t be well-controlled with insulin, and may also be used in coordination with sulfonylureas and Metformin.
  • given SubQ but is NOT insulin
  • can cause nausea and anorexia
  • avoid using with other meds that decrease GI motility (mainly anticholinergics)
  • do not use along with Acarbose
  • take immediately before a meal

• Latest generation long-acting insulin: Tresiba/insulin degludec is a new type of basal insulin with a couple of benefits over Lantus/insulin-glargine. The biggest advantage is that there is less incidence of night-time hypoglycemia. The other nice advantage is that the dose lasts up to 42 hours, so dosing can be more flexible than with Lantus (which must be taken every 24-hours at the same time every day).
  • missed doses can be taken within 8 hours of next dose
  • more expensive than Lantus

• Inhaled insulin: a rapid-acting insulin used for both Type 1 and Type 2 DM. Because it is an inhaled form of insulin, Afrezza is not to be used by people with lung issues and patients may need periodic pulmonary function tests.
  • less flexibility with dosages; cartridges come in 4 units, 8 units and 12 units (can be used in combination)
  • must be taken right before eating
  • watch closely for bronchospasm

Acute Diabetes Complications
Diabetic Ketoacidosis
DKA is a life-threatening condition that affects Type 1 diabetics. It involves a blood glucose > 250, a pH < 7.3, a serum bicarb of <15 and moderate to severe ketones in the blood or urine. Typical DKA Diagnostic Criteria
BS > 250
pH < 7.3
serum bicarb < 15
moderate to severe ketones
blood glucose ranges in DKA are high, usually about 300-800 mg/dL The pathophysiology of DKA is really pretty interesting!

Pathophysiology of DKA

Inadequate insulin in the body leads to cells not getting the energy they need and glucose building up in the blood.

The liver thinks the body needs more energy, so it converts glycogen to glucose while fats and proteins are also converted into glucose.

The use of fatty acids for energy leads to the buildup of ketones while serum glucose levels increase even more.

With all this serum glucose floating around in the bloodstream, the serum osmolarity goes up and fluids are pulled from cells as the body tries to normalize the concentration gradient. This leads to intracellular dehydration.

The intracellular dehydration causes a catecholamine response which further stimulates glycogenolysis, lipolysis and gluconeogenesis, so even more glucose is released into the bloodstream.

The kidneys can’t handle all the glucose and start spilling it into the urine along with some ketones and diuresis ensues.

This diuresis causes enormous fluid losses, electrolyte imbalances and dehydration.

Hyperosmolarity increases while acidic ketones continue to build up in the blood, causing dehydration and acidosis to worsen.

This thick, sticky blood clogs up the kidneys and GFR decreases, making the kidneys less effective at excreting glucose.

Serum glucose levels increase as the cycle continues.

Acidosis worsens leading to shock, coma and even death.

<table>
<thead>
<tr>
<th>Signs and Symptoms of DKA</th>
<th></th>
<th>Later Signs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Signs:</td>
<td>Polyphagia</td>
<td>Nausea and vomiting (very common sign!)</td>
</tr>
<tr>
<td></td>
<td>Polydipsia</td>
<td>Extreme fatigue</td>
</tr>
<tr>
<td></td>
<td>Polyurea</td>
<td>Kussmaul respirations (check it out!)</td>
</tr>
<tr>
<td></td>
<td>Headache</td>
<td>Weight loss</td>
</tr>
<tr>
<td></td>
<td>Fatigue</td>
<td>Dehydration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CNS depression</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Decreased LOC</td>
</tr>
</tbody>
</table>

Treating DKA

You would think the very first thing you would do to treat DKA is to give insulin. Actually, the very first thing you need to do is fluid resuscitate this patient. Their fluid losses and dehydration are enormous, and they sometimes need as much as six liters!
DKA treatment cont’d

• Fluids fluids fluids!
• Insulin via IV infusion with the goal of decreasing the blood glucose level 50-70 mg/dL per hour. If you decrease the blood glucose more quickly than that, you risk changing the concentration gradient too drastically and causing too much water to enter the cell. The result is cerebral edema.
• Once blood glucose is around 200-250, you’ll switch the IV fluids to something with a little bit of sugar to prevent hypoglycemia. This is typically D5W with 0.45% NaCl (you may hear it called “D5-Half”).
• You will be checking blood sugar levels hourly until they are within goal parameters and the patient is ready to switch to SubQ insulin.
• Labs to monitor
  • Anion gap
    • (Na + K) - (Cl + HCO3) = anion gap
    • the gap must be “closed” before the patient can switch to SubQ insulin
    • the gap is “closed” when it is < 11 (this may vary by institution)
  • Serum osmolality
  • Sodium
  • Potassium
  • BUN and Cr
• Switch to SubQ insulin when the following goals are met:
  • Blood glucose is within desired parameters (varies by institution)
  • pH is normal
  • Anion gap is normal
• Administer SubQ insulin. Typically this starts with giving them Lantus, waiting a couple of hours and then turning off the IV insulin infusion. If they are eating, they’ll get their blood sugar checked and sliding-scale insulin as needed.

Piece of cake!

HHS/HHNK
Hyperglycemic Hyperosmolar State (aka Hyperglycemic Hyperosmolar Nonketotic Syndrome) occurs in Type 2 diabetes and can also be fatal if not treated. The key difference between HHS and DKA is that the fluid losses are often higher in HHS, the blood glucose is often higher, and ketosis is absent or mild. In addition, there is no utilization of fats and proteins for energy in HHS because there is some insulin present. The presenting symptoms for HHS are similar to DKA with the exception of Kussmaul respirations as the patient is in an alkalotic vs acidic state.

Pathophysiology of HHS
Inadequate insulin in the body means cells don’t get the energy they need and glucose just builds up in the blood. The liver thinks the body needs more energy, so it converts glycogen to glucose, causing serum glucose levels to increase even more. This causes serum osmolality to go up, so fluid is pulled from inside the cells into the vasculature. The result is intracellular dehydration. The kidneys can’t handle all the glucose in the bloodstream, so it starts spilling over into the urine.

Diuresis ensues with enormous fluid losses and drastic electrolyte imbalances. Hyperosmolarity increases and ADH (antidiuretic hormone) is released in an effort to stop the diuresis, but it is too late. Dehydration worsens, leading to hypovolemia. Hypovolemia reduces renal perfusion, GFR decreases and oligura results. The malfunctioning kidneys are not able to excrete glucose effectively because they’re not making enough urine.

Meanwhile the SNS releases epinephrine in response to stress. Serum glucose levels rise even higher. The cycle continues,
leading to hemoconcentration of blood which puts your patient at high risk for clot formation and infarcts (brain, heart, lung) while CNS dysfunction leads to shock, coma and death.

**HHS Treatment**
The treatment for HHS is essentially the same for DKA, though some institutions may use slightly different “order sets” for each. However, the basics are the same:

- Rapid fluid resuscitation, can typically be 7 to 10 liters! Sometimes this is enough to correct the overwhelming hyperglycemia in HHS, but not always.
- Monitor serum sodium as you rehydrate with 0.9% NaCl, the fluids may need to be changed to a hypotonic solution if serum sodium rises. Generally, when the blood glucose reaches 200 to 250, the fluids are changed to D5W with 0.45% NaCl anyway.
- Insulin gtt with goal of dropping blood glucose by 50-70 every hour
- Hourly monitoring of blood glucose levels
- Electrolyte monitoring and replacement (especially K, Phos, Na)
- Monitor urine output, serum osmol, BUN, creatinine
- CVP monitoring if patient in hypovolemic shock
- Watch for signs of fluid overload (listen to those lungs!)

**Diabetes and the Hospitalized Patient**
Patients with diabetes will face some acute risks while hospitalized:

- Higher-than-usual blood glucose levels secondary to stress, glucocorticoid secretion and SNS activation
- Higher risk for infection (especially with BS > 220)
- Increased mortality secondary to infection
- Poor wound healing
- Studies show an increase in myocardial ischemia if the patient has diabetes
- Associated renal disease puts the patient at risk for anemia, fluid overload, electrolyte imbalances and build-up of medications cleared by the kidneys
- Neuropathy makes mobilization challenging, which can lead to further skin breakdown, constipation, falls and pneumonia

**Chronic Diabetes Complications**
Diabetes causes both microvascular and macrovascular damage throughout the body. The microvascular damage occurs because the elevated glucose causes a thickening of the capillary basement membrane causing fluid shifts and decreased function...the end result is that platelets adhere to the area which damages the tiny vessels of the eyes, kidneys and nerves. This translates to retinopathy, renal insufficiency (or failure) and diabetic neuropathy.

The macrovascular damage causes problems with larger vessels leading to hypertension, coronary artery disease, hyperlipidemia and atherosclerosis.

**Pathophysiology of Diabetes Complications**

*Free oxygen radicals:* Elevated blood glucose levels create alterations in the way proteins and lipids are metabolized in the microvasculature of the nerves and eyes. As a result, free oxygen radicals are produced which leads to “oxidative stress.” Recall from your chemistry classes that free radicals are not good. In this case, they deprive these delicate vessels of oxygen and certain growth factors. This pathological process leads to cell death (apoptosis) and can lead to retinopathy/blindness and neuropathy.

*Nitric Oxide:* NO levels are decreased in patients with diabetes, so it's important to recall that NO is a potent vasodilator.
Without this vasodilation, vessels constrict and platelets stick to the endothelium leading to hypertension, atherosclerosis, erectile dysfunction and even stroke.

**Glycosylation:** This is the process of sugar adhering to red blood cells as well as proteins (when it is adhered to the RBC, you get your HbA1c). When too much glucose is attached to proteins, it alters their cell function and leads to several diabetic complications such as impaired renal function and atherosclerosis.

**Diabetes Complication: Retinopathy**

Diabetic retinopathy occurs when high blood glucose levels cause damage to the delicate vessels of the retina. Non-proliferative diabetic retinopathy (NPDR) is the early stage of the disease. In NPDR, the vessels of the retina leak, leading to macular edema. If the vessels completely close off, then this leads to macular ischemia. Both will cause the patient’s vision to be blurry.

The more advanced stage of retinopathy is proliferative diabetic retinopathy (PDR). In this stage, the retina begins to grow new blood vessels in a process called neovascularization. While this may sound like a great idea, these new vessels are very delicate and can bleed into the vitreous. When this happens, the patient sees “floaters” across the field of vision. However, if it bleeds a lot then it can completely obscure all vision. In addition, these new blood vessels can create scar tissue which can lead to a detached retina (an ophthalmological emergency!)

Sadly, diabetic retinopathy is the leading cause of blindness in individuals age 20-74 in the United States.

**Treatments for Diabetic Retinopathy**

Blood sugar control: Keeping blood sugar within goal, following the recommended diet and taking all medications can help restore vision.

Medications: Intraocular injections can be used to reduce swelling. Yes, you read that correctly...injections IN THE EYE!!!

Laser Surgery: Laser surgery is used to seal off leaking blood vessels or shrink them in an effort to reduce the edema.

Vitrectomy: The removal of vitreous fluid and leaking blood can help restore vision in severe cases by allowing light to reflect properly off the retina.

**Preventing Diabetic Retinopathy**

- Control blood sugar
- Get regular eye exams that include dilation
- Manage high blood pressure

**Diabetes Complication: Peripheral Neuropathy**

Many diabetic patients will have neuropathic pain which they will typically describe as burning, tingling, electric shock or like walking on glass. So yes...it is very painful! It is a progressive disease that starts in the fingers/toes and extends to the hands, feet and so on. It is estimated that 60-70% of people with diabetes develop this complication.

Peripheral neuropathy is essentially nerve damage caused by chronically high blood glucose levels. In addition to feeling the burning sensations of the damaged nerves, those with neuropathy often cannot feel heat, cold or pain in the affected body part. Since pain is the body’s way of telling you something is wrong, this can be very dangerous! More on this in a bit.

**Treatments for Peripheral Neuropathy**

Medications: Gabapentin and Lyrica are very common (technically these are antiseizure meds, but are used a LOT for neuropathic pain), TCAs are sometimes used (amitriptyline) as are SNRIs such as duloxetine.
Preventing Peripheral Neuropathy
As with most things diabetes-related, keeping blood sugar under control is key to preventing neuropathy.

Diabetes Complications: Foot Ulcers
One of the most common complications you will see in your diabetic patients is the non-healing foot ulcer...also called “diabetic foot.” It is the unfortunate result of diabetic neuropathy + injury + poor wound healing. Because neuropathy decreases sensation to external pain, many diabetics get ulcers from simply having something press against their foot without their knowledge. For example, a small pebble in a shoe would do it, or an ingrown toenail. The wound develops and then, because of the diabetic’s poor wound healing, persists and persists. In many cases, it gets worse and worse and worse, often to the point of amputation.

Preventing Foot Ulcers
• Exquisite foot care!
• Regular cleansing and thoroughly dry, especially between toes.
• Lotion ok, just not between toes.
• Keep toenails short (be very careful not to cut skin when trimming); cut toenails right after bathing when they are soft and cut straight across.
• Wear thick socks and shoes that fit well (Always always always! No sandals and no bare feet!)
• Regular foot inspections to ensure nothing is causing pressure or any wounds have developed.

Diabetes Complications: Charcot Foot
Charcot foot (AKA Charcot arthropathy) is a complication characterized by disclocations and small fractures in the foot when no or little source of trauma is present. It occurs in individuals with neuropathy and is compounded by the presence of weak muscles and decreased blood flow. Over time, repeated small traumas to the foot occur before prior injuries can heal, which, along with alterations in blood flow, ultimately leads to the fractures and disclocations.

Treating Charcot Foot
Non-Surgical: Splinting or casting the foot. Immobilization is key!

Surgical: ORIF (open reduction internal fixation) and fusion are done in earlier stages while later-stage Charcot foot may require realignment or removal of bony prominences that could cause ulcers.

Preventing Charcot Foot
Guess what? Blood glucose control and excellent foot care are the name of the game! Also, well-fitting shoes!

Diabetes Complications: Neurogenic Bladder
Neurogenic bladder is a complication in which the nerves of the bladder are so damaged that the bladder loses the ability to sense when it is full. This leads to a whole host of problems such as incontinence, urgency, difficulty urinating or frequent urination. Over time the bladder will stretch out and lose its tone, leading to the inability to fully empty. This puts the diabetic patient at huge risk for UTI.

Treatments for Neurogenic Bladder
• Bladder training
  • Start with Foley catheter in place so the bladder can fully empty and start to regain its tone
  • Advance to self-catheterization on a set schedule
  • As the bladder recovers, the individual can practice “double-voiding” in which they void normally, wait 10-15 minutes and then self-catheterize or void again to remove any residual
  • Eventually the individual may be able to resume a normal voiding pattern
Diabetes Complication: Erectile Dysfunction
By now you understand that diabetes causes damage to blood vessels and nerves, especially those in the periphery. Well, guess what else is also at the periphery? Yep.

Treating Erectile Dysfunction:
• Medications
  • Oral Medications: tadalafil/Cialis and sildenafil/Viagra are common ED drugs.
  • Others: Medication can be injected into the base of the penis to improve blood flow; there’s even a tiny suppository that can be inserted into the tip of the penis before intercourse. Neither of these sound like they would inspire romance.
• Vacuum-device provides suction to improve blood flow and cause an erection.
• Surgical implants that are semi-rigid or inflatable.

Preventing Erectile Dysfunction in Diabetes
You’ll never guess! You got it....control that blood sugar, guys!

Cardiovascular Risk
Having diabetes puts patients at higher risk for cardiovascular disease, so it’s important that diabetics pay close attention to their CV health.

• Hypertension: Diabetic patients are advised to keep their blood pressure below 130 systolic and 80 diastolic.
• Hyperlipidemia: The goals for diabetic patients are LDL < 100 mg/dL, HDL > 40 mg/dL and triglycerides < 150 mg/dL; statins are considered the best option for decreasing LDL though some research is showing these meds are not without consequence. There is also some talk of making the LDL goal < 70 mg/dL. Something to watch for!
• Obesity: Being overweight puts diabetic patients at higher risk for CV disease. That’s a no-brainer, though! All patients with diabetes are encouraged to increase physical activity and lose weight.

CV Goals in Diabetes

| SBP | 130 |
| DBP | 80 |
| LDL | < 100 mg/dL |
| HDL | > 40 mg/dL |
| triglycerides | < 150 mg/dL |

Hypoglycemia
One of the most dangerous complications of diabetes is hypoglycemia. It can occur for a variety of reasons, including:

• Too much insulin (check those dosages very very carefully!) or too many oral hypoglycemics.
• Gastroparesis is essentially “stomach paralysis.” It is a sequela of nerve damage that has extended to the GI tract and the stomach doesn’t move things along with any type of regularity. The result is glucose (food) is absorbed irregularly, meaning that if your patient takes their insulin before dinner but doesn’t absorb as expected, they’ll become hypoglycemic. And then later, when the food IS absorbed, you get hyperglycemia. It’s a roller coaster.
• Increased physical activity.
• Alcohol consumption; alcohol blocks gluconeogenesis, so if your patient is going to drink, they need to order some nachos to go with that margarita!
• Lipohypertrophy caused by injecting insulin in the same site repeatedly, leading to scarring. The insulin will not be absorbed “on schedule” and its onset will be later than anticipated, causing hypoglycemia. Rotate those sites!
• Kidney disease can lead to the kidneys not clearing the insulin effectively, so levels build up.
Signs and Symptoms of Hypoglycemia

diaphoresis • confusion • shakiness • irritability • extreme hunger
anxiety • headache • dry mouth • nausea
palpitations • pallor • fatigue

Treating Hypoglycemia
If your patient is alert and able to swallow safely, the treatment for hypoglycemia is pretty simple: give ‘em some glucose! If they are unresponsive or unable to swallow safely, then you give dextrose IV or glucagon IM. The key to treating low blood sugar is to catch it before it becomes severe. Patients who have severe, frequent hypoglycemia may benefit from a service dog trained to sense hypoglycemia before the levels becomes dangerously low.

- Oral glucose: 15 grams of carbohydrate, wait 15 minutes and re-check blood sugar
  - 4 oz juice
  - 8 oz milk
  - glucose tablet
  - 1 tablespoon sugar or honey
  - 2 tablespoons raisins
- IM Glucagon: 1 mg
- IV Dextrose: 10 - 25 grams of D50 (often the dose is dependent on the blood sugar level)

Sick Day Protocol
When the body is under stress (like when you’re sick), it’s going to produce more glucose than usual. Combine this with the reduced intake that often comes with being sick and you’ve set yourself up for both hypo- and hyperglycemia (and most importantly, diabetic ketoacidosis). It is important that you educate your patients about how to manage their diabetes during illness with a “sick day protocol.”

- Monitor BS every 2 to 4 hours
- Check for urine ketones (if Type 1 DM)
- Drink 8 oz fluid every hour (if unable to keep fluids down, seek medical care)
- Take diabetes medications as scheduled
- If symptoms of DKA or HHS develop, seek immediate medical care
STRESS AND FATIGUE

brought to you by

STRAIGHT A NURSING
What Is A “Stressor?”
Stressors are environmental or internal events that tax adaptive resources. For example, encountering a vicious dog on your daily walk is a stressor, taking an exam in Med/Surg is a stressor, and so is any disease state. Obviously, these sorts of things happen all the time, so the body adapts in various ways.

How Do We Adapt to Stressors?
First, it is important to note that the body responds to physical stress and psychological stress the exact same way.

SNS: Enhancement of the sympathetic nervous system (SNS) causes the release of epinephrine and norepinephrine for increased vigilance and alertness. If you’re a survivor in the zombie apocalypse, you’re going to be super vigilant if you hear the sounds of a saber-toothed tiger nearby. That’s the SNS being activated!

Corticotropin releasing factor (CRF): CRF is released from the PVN (paraventricular nucleus) of the hypothalamus. This factor stimulates the pituitary to release ACTH (corticotropin) which, in turn, signals the adrenals to secrete glucocorticoid. This occurs within 30 minutes and is cleared hours after the stress is over, so it hangs around in the system for a bit.

The pancreas: The pancreas secretes glucagon (which converts glycogen to glucose for energy). You’ll need that extra energy to run from those zombies, right?

Growth hormone: An initial burst of growth hormone (GH) with a subsequent release of somatostatin (growth hormone inhibiting hormone). Why release the inhibiting hormone so quickly? In times of stress, the body only needs GH to do one part of it’s job, namely it just needs GH to convert fat in the cells to free fatty acids that are available for the muscles to use as energy. If GH were allowed to stick around and actually cause growth, then the body would be wasting energy on growing when it really just needs to focus on survival and running from that zombie.

Prolactin: The pituitary gland secretes prolactin (more on this in a bit!)

Antidiuretic hormone: The pituitary secretes antidiuretic hormone (ADH, aka Vasopressin). Remember that vasopressin vasoconstricts arteries to increase TPR (total peripheral resistance) which increases blood pressure so the blood can get where it needs to go. It also causes the tubule cells of the kidney to reabsorb more water from urine which also increases blood volume and increases blood pressure.

Endorphins and enkephalins: The pituitary and brain secrete morphine-like substances called endorphins and enkephalins. This comes in handy if you’ve got a broken leg but need to still get away from the undead.

Body System Adaptation to Stress
Various body systems adapt to the stress response, and this is typically a useful adaptation when the stress is infrequent and acute. However, when it is turned on a LOT, or just never turned off (as in the case of busy nursing students), then the risk of getting disease goes up. The body just can’t handle that prolonged and continuous stress response. Note that it doesn’t seem to matter if the body is dealing with a physiological stress or psychological stress (which is why it’s SO important that you take care of yourself while you’re in school!)

Metabolism: Adaptation to Stress
EPINEPHRINE antagonizes insulin: Remember that insulin “takes up” glucose in the blood stream. If insulin is antagonized, then it’s not going to do it’s job of “unlocking” the cell so glucose can enter. This leads to an elevated blood glucose level.

GLUCAGON levels increase: Remember that glucagon is responsible for the transformation of glycogen to glucose in the liver,
so this also increases blood sugar (and you need blood sugar for energy, so you can run for your life!)

GLUCOCORTICOID blocks insulin’s transport of nutrients into fat cells (gotta keep the nutrients available), decreases insulin secretion, breaks down triglycerides to free fatty acids (muscles can use these!) and stimulates gluconeogenesis in the liver. Whew! That’s a lot!

Inhibition of the parasympathetic nervous system (PNS) causes a decrease in energy storage. Why store energy when you need to use it NOW??? Remember, that the PNS stimulates the GI system to store nutrients.

Proteins are broken down for amino acids (only the proteins in non-exercising muscle), which are then converted to glucose in the liver...blood sugar goes up! Again.

**What happens if metabolism can’t adapt? The exhaustion phase!**
Fatigue occurs for several reasons. The biggest reason is that each time nutrients change between storage forms and useful forms there is a little bit of energy loss that occurs. Over time and with a lot of “stress on/stress off” kind of activity, you end up with a cumulative loss of energy and fatigue worsens. Also, too much SNS activity causes sleep disorders by interfering with the good ol’ Reticular Activating System (recall that the RAS detects new and novel stimuli and can help you “tune out” repetitive noises). Corticotropin releasing factor (CRF) causes appetite suppression which can lead to malnutrition and thus lead to fatigue.

**Cardiovascular System: Adaptation to Stress**
The SNS increases heart rate and blood pressure. Epinephrine makes platelets stickier in case you need to clot a wound. The SNS also vasodilates skeletal muscle so it gets all the blood and nutrients needed for “fight or flight.”

The pituitary increases the release of ADH, which increases water retention by the kidneys, keeping blood pressure and blood volume elevated.

The glucocorticoids increase platelets, increase blood sugar, and increase mobilization of fat for energy. Yay glucocorticoids! Sugar is used by the brain, and fat/sugar is used by muscle.

**What happens if the CV system can’t adapt and exhaustion sets in?**
Arteriosclerotic vascular disease (ASVD) is caused by chronic high blood pressure, chronic high glucose and chronic high levels of free fatty acids. When stress isn’t resolved and continues on to a chronic stage, the blood pressure, blood sugar and free fatty acids are elevated for long periods of time and go beyond what is therapeutic. When you have high blood pressure, there is a lot of turbulence at bifurcations in the blood vessels (bifurcations are where blood vessels branch off). This turbulence leads to the deposition of fatty acids, glucose and calcium. This causes damage to the vessel and voila, you have ASVD.

Also, an aging cardiovascular system does not respond well to epinephrine and norepinephrine, so older adults who are exercising are actually getting less than “maximal cardiac output.” Even in a resting state, the elderly secrete more epinephrine and glucocorticoid than do young people. It is thought that the epinephrine may be responsible for some of the increased instances of hypertension in the elderly.

**Gastrointestinal System: Adaptation to Stress**
The inhibition of the PNS causes digestion to be inhibited. Why digest when you may not even live long enough to need the nutrients? You also don’t need to carry around a bunch of stool, so note that the SNS increases motility in the tract. Maybe this explains why you get the urge to “go” before a big exam!
CRF (corticotropin releasing factor) suppresses appetite. Note that glucocorticoids stimulate appetite. This explains why sometimes you eat when stressed, and other times you don’t eat at all.

**What happens with the GI system can no longer adapt? Exhaustion!**

The loss of protective mucosal lining allows hydrochloric acid to damage the gut leading to GI bleeding and ulcers. You also get things like colitis and irritable bowel. Note that with repeated stress, recovery from decreased upper GI motility and increased lower GI motility may not occur simultaneously.

Some people also lose weight while others gain weight. How is this possible? Well, CRF clears rapidly after the stressor goes away, leaving a longer appetite stimulant effect of glucocorticoid. So…would constant stress cause weight loss while repeated short-term stressors cause weight gain? Something to ponder!

**Growth as an Adaptive Response to Stress**

One of the ways the body adapts to stress is to simply not grow. Why be a bigger target and why use up your nutrients for growth when you need them to run away from a blood thirsty zombie. So, what you’ll see first is an initial burst of growth hormone, which transforms fat cells into free fatty acids the muscles can use. BUT WAIT! We don’t actually want to grow now…so the body quickly releases somatostatin, which inhibits the growth effect of GH. Whew!

Also, glucocorticoid (there it is again!) decreases growth hormone (GH), and decreases the sensitivity of target cells for GH by 2-3x, so what little GH there is in the body isn’t being used. This is often seen in the arrested development of asthmatic children who used to be put on systemic glucocorticoids in the “olden days.” Glucocorticoid also inhibits bone growth by decreasing calcium uptake from the intestines and accelerating resorption of bone.

The stimulation of the SNS also causes decreased absorption of nutrients from the gut. Is this because it speeds up motility of the lower GI? Something to consider!

**What happens when growth can’t occur due to exhaustion from stress?**

Dwarfism, though rare, can occur. One would have to have some seriously severe levels of glucocorticoids to suppress GH that much. Also, osteoporosis and bone fractures result from decreased calcium uptake from the intestines and increased absorption from bone (though this is not likely to be significant). And finally, there is a likelihood that stress causes delays in wound healing, especially when you think about what elevated blood glucose does to wound healing and skin integrity.

**Reproductive System: Adaptation to Stress**

In general, the body does not care about reproducing when it’s about to be devoured by a reanimated corpse. When stress occurs, the SNS releases endorphins and enkephalines, which decrease LHRH (luteinizing hormone releasing hormone) in the brain. This leads to a decrease in LH and FSH being secreted from the pituitary. In males, this results in decreases in testosterone and sperm production. This also happens to people who have a lot of endorphins in their system such as “super jocks” or those who have a lot of opiates in their system (addicts).

The stress also causes an increased release of prolactin from the pituitary which decreases the sensitivity to LHRH, and you have the same situation as outlined above. Also, inhibition of the PNS causes less penile blood flow for erections. To sum up...in males, stress causes lowered sex drive, increased impotence and premature ejaculation...not a good combo!

For the ladies, adaptation and exhaustion are less clearly separated. For example, if a miscarriage is a stress response to save the mother’s life, is this adaptation or exhaustion? It’s a fuzzy area. More specifically, stress causes those endorphins and enkephalines to be released which reduces LH and FSH release from the pituitary. This leads to decreased estrogen, decreased ovulation and decreased progesterone. Decreased estrogen leads to decreased stimulation of estrogen receptors in the brain and genitals, which results in a lowered sex drive.
Women under stress have increased prolactin, which leads to decreases in LHRF. Glucocorticoid also blocks pituitary sensitivity to LHRH, and it inhibits ovarian sensitivity to LH, so there is less estrogen secreted from the ovaries. Prolactin also interferes with progesterone’s activity of maintaining the uterine lining for implantation. You can guess what happens to the lining when prolactin is on the job. No implantation, no pregnancy, no baby. In addition, the SNS vasoconstricts organs that are not vital for the mother’s survival, so it can cause increases in fetal death and miscarriages by decreasing blood flow to the uterus/fetus.

Athletes, anorexics and those starving may have delayed puberty due to lower estrogen levels. Enzymes in fat cells (which these people don’t have much of) convert androgen to estrogen. Since they have few fat cells, the androgens stay “male” and estrogen levels are relatively lower.

**Immune System: Adaptation to Stress**
Immunity is suppressed during stress. This process is not very well understood as of yet. We do know that the thymus atrophies when glucocorticoid levels are high. Lymphocytes decrease and there is decreased communication among WBCs.

However, some components of the immune system are *stimulated* by stress. Mild elevations of glucose may stimulate the immune system possibly due to suppression of immune suppressor cells. Also, WBCs that should be functioning (but are not) lead to a higher neutrophil count because they’re floating around in the blood stream where they can get drawn during a blood draw. If they were functioning, they would not be floating around, they would be getting out to the site of tissue infection or trauma in order to fight pathogens.

**What happens when the immune system can’t adapt? Exhaustion**
One of the biggest is cancer. Substantial evidence shows that stress accelerates cancer cell growth, but probably does not initiate it (at least that’s what we know now….who knows? New discoveries are made all the time!)
- The glucocorticoid levels usually seen in stress aid the development of blood vessels to the tumor, so the tumor now has a way to get nutrients. It essentially has a food supply and this is not good.
- Glucocorticoids suppress lymphocytes that normally detect and kill cancer cells.
- Glucocorticoids provide higher blood glucose, which is nutrition for the tumor.

Autoimmune disease can also occur. Note that stressful periods often precede the onset of immune-related diseases such as diabetes, multiple sclerosis and lupus. This is why it’s so important for you to take excellent care of yourself during school!

**Neurological System: Adaptation to Stress**
When you are faced with a stressor you go into high alert and get done whatever it is you need to get done (escape from a zombie, take a test or do CPR on a plane!) How does this happen?
- The SNS increases alertness
- Endogenous morphine (endorphins & enkephalins) blunt pain perception

**What happens when the neurological system can’t adapt? Exhaustion**
Stress depletes norepinephrine transmitters in the limbic system of the brain. Recall that the limbic system is involved with emotion, so a lot of people with chronic stress are also very sad. Depressed learning, coping and motivation are all associated with “learned helplessness.” People with learned helplessness have an external locus of control and are the most vulnerable for this. “There’s just nothing I can do.”

Aging also comes into play. Recall that the hippocampus is involved with memory. Research tells us that increased exposure to glucocorticoid causes neurons to lose their ability to re-branch effectively. This explains why stress can accelerate memory loss with aging.
Coping with Stress
Note that coping may or may not be functional. For example, if I go for a run or do breathing exercises when I am faced with stress, then I am probably coping pretty well. If I get drunk every night, then my coping is dysfunctional.

Coping Mechanisms
- Anxiety can be useful if it is mild and accompanied by increased alertness and vigilance. This is the type of anxiety that is helpful when you take an exam, but you wouldn’t want it to be so high that your brain freezes.
- Taking action to reduce the stressor or minimize its impact. This can be done by simply eliminating the stressor (change jobs if the workload stresses you out) or displacing major worry onto something more manageable. For example, maybe you yell at your spouse but you’re actually angry that you’re falling behind at work.
- Depression is definitely a dysfunctional way of coping. Glucocorticoid levels are elevated in many depressed individuals.
- Helplessness and hopelessness (not functional).
- Alcohol and drug use (not functional).
- Seek social support (great idea!).
- Denial (can be useful for short-term to help us get on with life).

Stress Reduction/Adaptation Therapies
One of the best things you can do for someone who is stressed is give them the perception of control or the ability to predict when stressors will occur. If you KNOW you’re going to get a shot every day for the rest of your life you know when to expect it and it’s no big deal. If the nurse tells you, “I’m going to be giving you a shot in your eyeball sometime in the next week,” then you’re going to stress about it the entire week…better to know and better to be predictable!

Examples of ways to accomplish this in a clinical setting:
- Call light…find out if the patient is worried we won’t respond right away. If so, he may be pressing the call light in anticipation of FUTURE needs. If we always respond (and let him know what time we’ll be back), he knows that he only needs to press the button when he actually needs something. He has control!
- PCA is associated with less medication need.
- Choice of room décor, meals, activities, etc…definitely key with nursing home residents.

Giving Control: Focus on giving control over future course of disease and not mulling over poor choices made in the past. Don’t berate the patient for smoking. Instead, talk to them about how they can cease smoking and improve their health.

When the outcome is good, give the patient all the credit. Say, “GOOD JOB! You walked all the way around the nursing station…way to go!” If the outcome is not good (patient has to sit down, can’t make it all the way), don’t blame the patient. Instead say something like, “We’ll try this again after we get your breathing treatment done.”

Sharing Knowledge: Basically, just be aware that you should give the right amount of information. Too much can be as stressful as too little information. You can also assist the patient in determining the significance of stressors and maximizing their ability to DO SOMETHING ABOUT IT!!!!

Touch: Touch can be very therapeutic, but you have to watch for the patient’s response in order to know if it is something they are comfortable with. One study showed that rats handled during the first few weeks of life secrete LESS glucocorticoid and aged more successfully as adults.

Moderate Exercise: Exercise lowers BP and resting HR while increasing lung capacity. Yay for exercise! It also releases those morphine-like substances that make us feel fantastic!
Hostility Management: If your patient is hostile or angry, LISTEN to him. Let him talk! If the situation escalates, report it to your charge nurse immediately. You should never feel like you are unsafe at work...if you work at a good place, they’ll have your back!

Social Support: Several studies have shown a link between fewer social relationships and shorter life expectancy. This factor appears to be at least as important as smoking, obesity and sedentary lifestyle. Make some friends!

Other therapeutic/adaptation mechanisms:
  • Provide outlet for frustration
  • Relaxation techniques (listen to music, breathing exercises)
  • Spiritual rationalization of stressor

Fatigue
Fatigue is a sensation of exhaustion that may or may not be related to activity. It can be acute or chronic. To be classified as chronic, it typically occurs for a duration of six months or more.

Chronic Fatigue Syndrome (CFS) is severe but has no known etiology and always occurs with other symptoms. About 2.4 million American adults suffer from CFS-like illness. Exercise can help decrease fatigue, but it may be difficult to get the patient to try...after all, they’re fatigued! In general, exercise works better when combined with education and antidepressants.

Fatigue Assessment
  • Ask patient, “Do you have more fatigue when you first wake up?” If yes, think about depression and psychologically related fatigue.
  • Ask patient, “Do you have more fatigue after activity?” If yes, think about metabolic related fatigue.
  • Evaluate meds for possible side effects of fatigue. This includes hypnotics, muscle relaxants, antidepressants, antipsychotics, anti-seizure meds, anti-hypertension meds and narcotics.
  • Duration of fatigue
  • Impact on quality of life: Can they work? Have fun?

Therapy for Fatigue
  • Assist patient to meet unmet needs, focus on the most important first! (ABCs, Maslow’s, etc..)
  • Identify most important things for patient to expend energy on
  • Provide specific treatments as indicated (i.e. may be anemic, may need sleep apnea mask)
  • Replace nutrients and restore healthy cellular environment (O2, fluids, iron/erythropoietin, electrolytes, energy source, BP, body temp, etc…)
  • Antidepressants, possibly stimulants
  • Cognitive behavioral therapy (help patient learn how to cope)
  • Exercise

Cancer and Fatigue
  • Some evidence shows that exercise benefits women with breast cancer (not adequately studied).
  • Bone marrow transplant patients who exercised for 30 mins/day x 6 weeks reported less fatigue.
  • Prevalence of fatigue in patients with advanced cancer is 50-70%.
  • Fatigue in the caregiver is also a factor...mainly relates to how much the care impacts their day-to-day life.

Anemia and Fatigue
  • Low Hgb associated with fatigue.
• Iron may be helpful in menstruating women.
• Patients with chronic kidney disease will have anemia due to the decrease in erythropoietin production. They will typically receive weekly erythropoietin injections to help with this.

**AIDS and Fatigue**
• AIDS meds cause many side effects. Exercise is most popular therapy.

**Depression and Fatigue**
• Depression and chronic fatigue are risk factors for each other.

**Sleep Deprivation and Fatigue**
• Psychological fatigue is related to prolonged and irregular work hours, not necessarily related to energy expenditure (a good example is long-haul truck drivers).
• Shift workers must be able to adapt to changes in schedule and control schedule...a challenge for night shifters!
• Napping during night shifts reduces fatigue (presumably on breaks). Some units will combine all the breaks together into one hour-long break for this purpose.

**Respiratory Disease & Obstructive Sleep Apnea and Fatigue**
• Nasal positive pressure ventilation improves daytime energy in patients with respiratory failure related to neuromuscular weakness.
• 5-15% of population has obstructive sleep apnea (OSA). Positive pressure airway mask prevents apnea!

**Renal Failure and Fatigue**
• Acupoint massage reduced fatigue and depression in patients on dialysis.

**Heat and Fatigue**
• High core temp can cause fatigue due to heat/stress.
• During hot weather, give enough fluids to permit loss of heat from increased metabolism in exercise (work). Also, don’t exercise during the hottest times of day!

**MS and Fatigue**
• Higher levels of helplessness correlate with more fatigue in MS patients. Cognitive behavioral therapy works well.
• Some find stimulants helpful, other studies do not support this.

**Brain Attack and Fatigue**
• Fatigue is common and debilitating after a stroke. More studies need to be done regarding intervention.
IV THERAPY BASICS

brought to you by STRAIGHT A NURSING
**What is IV Therapy?**
IV therapy is the practice of infusing fluids into the system via intravascular access. That's it! Sounds so simple, doesn't it?

**The Types of Vascular Access**
Before you can give the patient fluids intravascularly, you need vascular access. Lucky for you, there are a few different ways to obtain access and each has its own pros and cons. The type of access you choose depends on what your patient needs. Coming to the ED for nausea/vomiting and really dehydrated? A standard IV will do. Blood pressure 70/55? You’ll need to pull out the big guns. This chart summarizes the various types brilliantly.

<table>
<thead>
<tr>
<th>TYPE</th>
<th>LOCATIONS</th>
<th>USES</th>
<th>PROS</th>
<th>CONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral IV</td>
<td>In adults, usually the hands or lower arms. Upper arms used rarely.</td>
<td>Basic fluid and med administration not requiring a large vein.</td>
<td>Easy to obtain and maintain, inexpensive, can be done by any RN.</td>
<td>Cannot use for some types of medications, cannot use for lab draws, decreased freedom of movement if placed in antecubital space.. Smaller-sized catheters not suitable for power injectate or blood transfusions.</td>
</tr>
<tr>
<td></td>
<td>The external jugular is used somewhat often. When placed in the EJ, this line looks a lot like a central line, so be careful!</td>
<td>Ex: Normal saline, antibiotics, pain meds</td>
<td>Can be used for power injections (contrast dyes) if big enough and in the antecubital vein.</td>
<td></td>
</tr>
<tr>
<td>Midline Catheter IV (Ex: Powerglide®)</td>
<td>Mid forearm</td>
<td>Basic fluid and med administration not requiring a large vein.</td>
<td>Can stay in place up to 29 days, comfortable, lower infection risk than peripheral IV. Can use for lab draws and power injections.</td>
<td>Must be placed by specially trained RN.</td>
</tr>
<tr>
<td>Peripherally inserted central catheter (PICC line)</td>
<td>Inserted above elbow, the catheter extends to the SVC</td>
<td>Long-term fluid and med administration. Administration of powerful medications such as chemo, vasopressors and inotropes; can give TPN through a PICC.</td>
<td>Can stay in place for months if needed for long-term therapy, comfortable, multiple lumens. Can use for lab draws, power injections, ScVO2 monitoring and hemodynamic monitoring.</td>
<td>Must be placed by MD or specially trained RN. Placement must be confirmed via ultrasound or chest x-ray prior to use. Risk for infection and DVT.</td>
</tr>
<tr>
<td>Implanted port (Port-a-cath)</td>
<td>Chest wall, beneath the skin.</td>
<td>Long-term/frequent administration of meds or blood products. Commonly used for chemotherapy.</td>
<td>Decreased risk of extravasation, safer administration of chemo, may have one or two lumens (one is most common). Can use for blood draws and some can accept power injections.</td>
<td>Requires surgery for placement and removal. Accessing the port via a needle can be painful.</td>
</tr>
</tbody>
</table>
### Tunneled Central Venous Catheter

<table>
<thead>
<tr>
<th>TYPE</th>
<th>LOCATIONS</th>
<th>USES</th>
<th>PROS</th>
<th>CONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tunneled central venous catheter</td>
<td>Chest wall with catheter tip in the vena cava</td>
<td>Long-term/frequent administration of meds or blood products. Commonly used for chemotherapy.</td>
<td>Decreased risk of extravasation, safer administration of chemo, multiple lumens. Can use for blood draws and power injections. Access site is not under the skin, so not painful to access.</td>
<td>Requires surgery for placement and removal.</td>
</tr>
</tbody>
</table>

### Percutaneous Non-Tunneled Catheter (a standard “central line”)

<table>
<thead>
<tr>
<th>TYPE</th>
<th>LOCATIONS</th>
<th>USES</th>
<th>PROS</th>
<th>CONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percutaneous non-tunneled catheter</td>
<td>Internal jugular, subclavian vein, femoral vein</td>
<td>Administration of powerful medications such as vasopressors, chemotherapy, inotropes, TPN. Used for patients who are hemodynamically unstable and require fluid resuscitation and continuous hemodynamic monitoring (though CVP monitoring is not routinely used).</td>
<td>Can instill anything, multiple lumens. Subclavian location is comfortable, allowing for freedom of movement. Can be used for lab draws, ScVO2, power injections and CVP monitoring.</td>
<td>Must be placed by MD or specially-trained NP. Infection risk is higher, especially femoral placement. Subclavian placement not advised in patients with coagulopathy. Jugular placement is uncomfortable, difficult to keep dressing dry and intact (most common).</td>
</tr>
</tbody>
</table>

### What Does “Central Line” Mean?

You will hear the term “central line” a lot when you’re in the clinical setting. A central line is simply any vascular access where the tip of the catheter empties the fluid/medication into a central vein such as the superior vena cava. Many medications such as vasopressors (ex: levophed) should not be infused through a peripheral IV. These are highly powerful meds that should be given into a large vein very near the heart to reduce risk of extravasation AND ensure the medication is mixed into circulation more quickly. Some meds, like TPN, are so hypertonic they absolutely cannot ever be administered peripherally.

**UPDATE:** Studies show that short-term administration of low-dose vasopressors is acceptable if placed in a large vein (not a hand vein). The idea is that some patients only need vasopressors for a short period of time. The risk of infection related to a central line outweighs the risk of using a large peripheral vein for a short period of time. If the patient requires higher doses of vasopressors, more than one vasopressor or vasopressors longer than 24-hours, then a central line is indicated.

Most central lines are pretty obvious, but some aren’t so clear. For example, a peripheral line placed in the external jugular looks, at first glance, like a central line placed in the internal jugular. A mid-line catheter could possibly be mistaken for a PICC. A femoral line could actually be an arterial line, and not venous at all. **ALWAYS ask for clarification on your lines when you are receiving bedside report.**

### Vascular Access Management

You’ve got your lines placed, now what? The main responsibilities of the nurse are to 1) maintain patency and 2) reduce the
risk for infection. The facility where you do your clinicals/work will have different policies in this regard, but the basics are that you are going to flush the line before and after all meds, and you are going to flush the line at regular intervals. For example, a PICC line will get flushed with 10ml normal saline three times a day. Regular flushing of the line ensures that it remains patent and that if it becomes “sluggish” (or difficult to flush) you know right away and can do something about it. With peripheral lines, a sluggish IV is a sign that it’s time to remove the IV and start a new one. If your PICC or central line is sluggish, you can often “un-clog” it using tPA. The protocols on this will vary by facility, so for now just know that it’s possible if you come across it.

What about infection risk? You’ve got to keep those IV dressings CDI (clean, dry and intact). Lines that are only in for a few days, such as a peripheral IV, won’t require dressing changes unless the dressing becomes wet or soiled. These lines don’t stay in long enough to require routine dressing changes. However, lines that are in place for a while (PICC and central lines) will typically have their dressings changed weekly (or when needed). If your central line dressing is wet, not adhering well, or soiled in any way, get in there and change it! Note that changing a central line dressing is a sterile procedure, so please do not do this without supervision! PRO-TIP: when changing central lines, always place a mask on the patient as well!

When accessing your lines, always “scrub the hub” for the recommended period of time (current studies say 15 seconds, so sing “Happy Birthday” twice) and then let it air dry. As the alcohol dries it kills the pathogens. You know what they say...the “dry time” is the “kill time.”

Whenever a new central line is placed, it is best practice to change out all the tubings on your medications before attaching it to the new line. This can be quite the task if your patient is heavily vasopressor dependent, so expediency is key!

**All Fluids Are Not Created Equal**
There are a few different types of fluids that your patient will receive, depending on their clinical condition and lab values. The most basic is normal saline (NS) which is 0.9% sodium chloride. However, this is not the only fluid you will see, so let’s chat about it, shall we?

**A Note About Osmolarity**
Osmolarity refers to how dense a particular fluid is. Or, in fancier terms, the measure of solute concentration per liter. If you remember your general chemistry and physiology, you’ll recall that the body utilizes concentration gradients to maintain homeostasis. Remember osmosis? That’s basically just the exchange of water molecules across a permeable membrane from an area of less solute concentration to an area of higher solute concentration. The water is moving across the membrane in an attempt to equalize the concentration of solutes. Keep this basic concept in mind when you are thinking about different solutions/fluids being used in different clinical situations.

**Isotonic vs Hypotonic vs Hypertonic**
One of the most common questions nursing students ask is about toxicity and osmolarity. It can definitely be confusing, but these two concepts play key roles in why a certain fluid is chosen and what the expected effect is in the body.

Fluids can be either isotonic, hypotonic or hypertonic. Each of these terms relates to the fluids osmolarity in comparison with the osmolarity of human serum, which is around 290 mOsm/kg. When we say a fluid is “isotonic” we mean that it pretty closely matches the serum osmolarity of the human body; when it is hypotonic, it is less than the osmolarity of the serum; and when it is hypertonic, it is greater than the osmolarity of the serum. A great example is 0.9% sodium chloride (normal saline). It has an osmolarity of about 308, which makes it an isotonic solution. On the next page, let’s look at some of the main IV fluids you will see in the hospital and what they are used for:
<table>
<thead>
<tr>
<th>FLUID</th>
<th>OSM</th>
<th>TONICITY</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
</table>
| 0.9% NaCl “Normal Saline” | 308 | isotonic | • Basic fluid maintenance or resuscitation  
• Replace fluid losses related to vomiting, diarrhea, hemorrhage, GI suctioning  
• Used with blood product administration  
• Watch for elevated chloride levels when large volumes are used for fluid resuscitation |
| 0.45% NaCl “Half NS”      | 154 | hypotonic| • Used for patients who are hypernatremic  
• Rapid infusion can cause RBCs to hemolyse  
• Monitor for fluid overload  
• Used cautiously in patients with renal disease and heart failure |
| 3% NaCl “3% saline”       | 1027| hypertonic| • Used for patients who are hyponatremic. **HIGH ALERT MED!**  
• Must be given through a central line due to high osmolarity  
• Give slowly to avoid central pontine myelinolysis (locked-in syndrome)  
• Always use a filter  
• Monitor serum sodium and serum Osm per protocol |
| 5% Dextrose in Water “D5W”| 278 | isotonic AND hypotonic| • Basic fluid maintenance w/ dextrose for people who are NPO  
• Provides approximately 200 cals/liter  
• Initially is isotonic, but becomes hypotonic when dextrose is quickly utilized by cells  
• Used to treat hypernatremia (be careful Na does not drop too quickly!)  
• Monitor for hyperglycemia  
• Contraindicated in patients with elevated ICP |
| 10% Dextrose in Water “D10W”| 505 | hypertonic| • Used when patient needs more sugar than D5W provides  
• Used as a “back-up” fluid if TPN administration is abruptly stopped  
• Contraindicated in patients with elevated ICP, delirium tremens, intracranial or intraspinal hemorrhage, anuria  
• Recommended to give through a central line; may cause phlebitis  
• Monitor for hypokalemia  
• Provides about 340 calories per liter |
| 5% Dextrose in NS “D5NS”  | 560 | hypertonic| • Used when the patient needs sugar, but also needs some NaCl  
• Indicated in hypotonic dehydration and Addisonian Crisis  
• Not for use in patients with renal failure or heart failure due to risk of pulmonary edema |
| 5% Dextrose in 0.45% NaCl “D5 Half” | 406 | hypertonic| • Used when the patient needs sugar, but also a little NaCl  
• Indicated for maintenance fluid and hypovolemia  
• Used in DKA after blood sugar reaches 200-250 while patient remains on insulin infusion (prevents hypoglycemia and cerebral edema) |
| Lactated Ringers “LR”      | 273 | isotonic| • Basic fluid maintenance or resuscitation, especially in burns and trauma  
• Used to treat fluid losses due to fluid shifts, blood loss and GI losses  
• Has various electrolytes (Na, K, Ca, Cl)  
• The calcium in LR typically makes it incompatible with a lot of medications  
• Use cautiously in renal failure due to presence of K  
• Not recommended in liver disease as patient will be unable to metabolize the lactate; general rule of thumb is to avoid in pH > 7.5 |
| 5% Dextrose in LR “D5LR”   | 575 | hypertonic| • Patient needs sugar AND other electrolytes besides just NaCl  
• Indicated in cases of metabolic acidosis (lactate should help) |
| Fluids with potassium added| varies | | • K will often be added to fluids to maintain optimal K levels  
• Read the labels carefully...they all look very similar  
• D5 Half NS with 20K is a common post-op fluid |
IV THERAPY - MED/SURG 1

Fluids will be chosen based on what is happening with the patient’s serum electrolyte values and fluid volume status.

**Isotonic solutions** are used for general fluid maintenance or to increase intravascular volume in cases of dehydration or hypotension.

**Hypotonic solutions** are given to expand the intracellular space. If you think about how osmosis works and you give a lower-solute concentration to an area of higher-solute concentration, the fluid is going to try to equalize, so the fluid will go into the cell. For this reason they are commonly used to treat intracellular dehydration, which occurs in diabetic ketoacidosis and hyperosmolar hyperglycemic states. You will also see them used to treat severe hyponatremia (elevated sodium levels). Be careful, you don't want to drop the Na levels too quickly!

**Hypertonic solutions** are given to pull water from the cells. You will probably most likely see 3% NaCl used in severe cases of hyponatremia (decreased sodium levels). Note that this is a HIGH ALERT medication and should never ever ever ever ever be given via a bolus except by very specific physician order (and this is done very rarely and only in extreme cases). Also note that hypertonic solutions are extremely irritating to the veins, and many of them (especially the HIGHLY hypertonic ones) should only be given via a central line.

If you’d like more help with this concept, these illustrations might do the trick. The arrows show which direction the fluid flows:

A: cell in isotonic solution
B: cell in hypotonic solution
C: cell in hypertonic solution

When we think about the “tonicity” of a fluid, we are taking into consideration how the osmolarity of the solution compares to the osmolarity of the patient’s system at that time.

- Isotonic means it’s essentially the same
- HYPOtonic means it’s LESS concentrated than the patient's system
- HYPERtonic means it’s MORE concentrated than the patient’s system

In **Figure A** above, the round cell is in an isotonic solution such as normal saline. Since there is no concentration gradient for the water to use for osmosis, everything basically stays status-quo, things flow equally into and out of the cell. In **Figure B**, we've got a cell in a hypotonic solution. In order to equalize the solute concentration, water will move from the area of least concentration (outside the cell) into the area of higher concentration (inside the cell). This will cause the cell to expand as you see in the illustration. In **Figure C**, we have a cell in a hypertonic solution...yes, it does looks like a star, but it’s actually a shriveled-up cell. Again, think about your concentration gradients. Water is going to flow from the area of lesser concentration (inside the cell) to the area of higher concentration (outside the cell), resulting in a

**Hypo or Hyper...I forget?!?!**

When you’re first starting out, it is easy to mix up hypo and hyper. When you think of hypo, think of the cell swelling up like a great big O. When you think of hyper, think of a hyperactive puppy who is doing too much...hyper means it’s high.

You got this!
dehydrated cell. This will come into play in diabetic ketoacidosis in a big way, and you'll also see it a fair amount in hypernatremia. More on those in other sections.

**Crystalloids vs Colloids**
Two more terms you'll hear frequently are “crystalloid” and “colloid.” What are they and how are they different? A crystalloid is basically your typical IV fluids that we listed in the table above. It contains water-soluble molecules and is usually the first line of fluids chosen when looking at increasing intravascular volume.

Colloids, on the other hand, contain insoluble molecules that are larger and don't pass through the vessel wall. Instead, colloids stay in the vasculature and can more significantly increase vascular volumes by pulling fluid into the vascular space. Common colloids include albumin, Hextend and Dextran. Note that colloids are more expensive than crystalloids and typically only used when absolutely necessary to maintain blood pressure.

**When Good IVs Go Bad**
Knowing what to watch out for with your IVs is just part of being a good nurse. So, what are some of the things that can go wrong and what are you going to do about it?

**Pain at IV Site**
This is probably the most common problem you'll have. IVs typically shouldn't hurt (though they're not exactly comfortable either), so all complaints of pain should be investigated.

1) Check the IV for patency. Will it flush? Does flushing the IV cause pain? If so, the vein has probably blown and you'll need to change the IV site.

2) Does the IV flush fine, but the medication that's infusing cause pain? If you are running potassium through a peripheral vein, it burns. A good trick to use is to avoid running the potassium in all by itself. Let's say your patient has orders for normal saline at 100 ml/hr and an order to replace 10 mEq potassium at 50 ml/hr. With most IV pumps, when you "piggy back" the potassium into the pump, the pump will stop infusing your maintenance fluid and switch over to the piggyback fluid (in this case, the potassium). So, what you end up with is the potassium running in all by itself at 50 ml/hr. If your pumps allow concurrent meds to run off the same pump, then you can set yours up to also infuse the NS concurrently so that the potassium is diluted as it enters the vein. You can also set up a second pump, and connect the potassium tubing to the Y-site connector on the maintenance line.

3) Check the dressing. A loose dressing can cause the catheter to wiggle around just enough to be highly irritating.

4) Check the location of the IV. An IV in the antecubital (AC) is generally not well tolerated because patients bend their arms A LOT. Over time, this is irritating to the vein and therefore, irritating to the patient. Check to see if your patient is keeping his arm straight or bending it repeatedly despite your constant reminders. Sometimes the only thing you can do is just switch it out to a forearm location, which is generally easier to tolerate.

**Pain at IV Site with Redness and Edema (infiltration and extravasation)**
Every now and then the vessel wall loses its integrity and the fluid you are instilling is actually just getting pumped into the surrounding tissues instead of the vascular space. With normal saline and other IV fluids this is usually not a big deal (it is called infiltration) as the fluids will be reabsorbed over time. However, if this happens with a medication (extravasation), this can be a bigger problem. What do you do if you suspect infiltration or extravasation?

1) Stop the IV infusion

2) Leave the catheter in place in case you can administer an antidote

3) Call the pharmacy. The pharmacist will let you know if the medication in question is considered a “vesicant” or something harmful to the surrounding tissues. S/he will let you know what the antidote/treatment is (if one exists). Sometimes the antidote is infused through the catheter, other times it is injected into the subcutaneous tissue or applied topically. If no antidote exists, then heat or cold may be applied depending on the medication in question. For example, the antidote to amiodarone extravasation is application of nitropaste.
4) Call the MD. Depending on what you learned from the pharmacist, you may need to ask for specific orders for the antidote in question. If nothing else, you want him/her to know of the potential for injury and that their patient is not currently receiving whatever med was ordered.

Note that infiltration and extravasation can happen in patients who are unable to report pain...so keep an eye on those lines!

**Misc Things You Need to Know**

*If you bring the serum Na up too quickly in cases of hyponatremia*, the Na will act as an osmotic agent to draw water out of the cell. This will DEHYDRATE your patient and quite possibly pickle his or her brain. The treatment for severe hyponatremia is 3% sodium chloride, which again is a HIGH ALERT medication for this very reason. Raising a sodium level too quickly can lead to a serious condition called locked-in syndrome, which would be devastating for both you and your patient. 3% sodium chloride is always given on a pump and you will never bolus anything through this line. It will run at a max rate of 30-50 ml/hr and you’ll be checking serum sodium levels regularly.

*If your pt is NPO*, look at how long they have been NPO and what type of IV fluid she’s been receiving. Check the morning labs for electrolyte imbalances and hypoglycemia or even just down-trending blood sugars. You may want to consider a solution with some dextrose in it, which will provide your patient with some calories and carbohydrates.

Glucose/18 (the glucose level divided by 18) will equal the serum tonicity telling you what the osmolarity of the serum is.

*Every 100 increase in glucose causes the Na to drop by 2 (roughly).* The reverse is also true, so if you start with a BS of 1000 and a sodium of 130, by the time the BS is at 200, the Na will be at 146, so you wouldn’t want to give this patient too much Na. Essentially, by treating the sugar problem you will correct the Na problem. When blood sugars are that high the VERY FIRST thing you do is give fluids.

*For hyponatremia*, the pt is typically symptomatic at around Na of 120, but this can vary widely. Some patients have chronic hyponatremia and function decently in the 120s. It’s when the drop is sudden that you’ll typically have issues with neurological function.

When you are considering your IV therapy think about three things…the salt, the sugar and the rate.

When glucose goes into the cell, K enters also. Again, we’ll go into much more on this when we talk about electrolyte imbalances, but it’s something to watch for when treating patients for elevated blood sugars. An easy way to remember this, is the potassium piggybacking onto the glucose molecule as it enters the cell, the potassium molecule says, “Thanks for the ride, sugar!”

*When dextrose is infused*, the sugar is metabolized and no longer contributes to the solute concentration. So, what might start out as an isotonic or hypertonic solution actually becomes less concentrated. For example, when D5W is infused it starts out isotonic, but as the sugar is used by the body it quickly becomes hypotonic.

Do not ever administer a hypertonic solution into a small peripheral vein. If you think about what hypertonic solutions do, you’ll realize that the solution will pull water away from the vessel wall in an attempt to achieve hemostasis. This can be very damaging to the vein.

*A patient with brain edema* or a head injury will never receive a hypotonic solution. Also, because dextrose is metabolized and solutions containing it become hypotonic, you typically will not give these patients solutions containing dextrose...it would be the same as just giving them straight hypotonic solution, which would increase brain edema. Not good.
Practice Questions

1) Pancreatitis and NPO: Your patient was admitted for pancreatitis two days ago and the doc has prescribed that the pt be NPO. The patient currently has LR running at 125ml/hr and she’s likely to be NPO for another couple of days. Her morning labs show: Na 142, K 3.4, Glucose 72. What would be the best IV solution for her to change to?

   A) D5 1/2 NS with 20 KCl
   B) D5W
   C) Lactated Ringers
   D) Half NS with 40 KCl

If you chose A, you are right! Since her sodium is normal, we don’t need to add a lot of NaCl to her system. But look at her potassium and glucose. The K is borderline low and the glucose is definitely on the low side. We want to add a little sugar, but doing so will cause K to ride into the cell, so we’ll need to supplement the K a little bit.

2) Vomiting for Days: Your patient has severe morning sickness and comes to the ED after vomiting for two days pretty much non-stop. She is very dehydrated, but somehow all electrolytes are within normal limits. Her blood pressure, however is on the low side and skin signs are very dry. What would be the best fluid for her?

   A) D5NS
   B) D10W with 40 mEq of Potassium
   C) Lactated Ringers
   D) NS with 20 mEq of Potassium

Lactated Ringers is a great all-around fluid to give for volume depletion. It contains a general mix of electrolytes in roughly the same concentrations as the human body.

3) NPO Patient Counts Calories: Your patient is NPO and he’d like to figure out how many calories he is receiving each day. Currently he has D5W running at 100ml/hr. How many calories is he getting in a 24-hr period? Recall from your nutrition class that each gram of dextrose gives 5 calories. 5% dextrose in water provides 5 grams of dextrose in 100 ml.

   A) 5000
   B) 500
   C) 1500
   D) 1000

If you answered 500 (or about 480), then you are correct! Way to go!

4) High Blood Sugar: Your patient has diabetes that is difficult to control. You check his blood sugar in the morning and it’s 308. You check his morning labs and see that his Na level is 132. Being the good nurse, you know to:

   A) Encourage the patient to drink water
   B) Order a low-sodium diet
   C) Anticipate that the Na level will return to normal as the blood glucose is corrected
   D) Obtain seizure pads from central supply

The correct answer is C. As the blood sugar normalizes, the Na will increase by 2 for every 100 the glucose drops.
Nursing school is tough!

WE’RE HERE TO HELP

- Nursing School Thrive Guide -
  Get the 5-star rated book that has helped thousands of nursing students succeed. Available in paperback, Kindle and audiobook. Explore at straightanursingstudent.com/thrive-guide/

- Nursing School Bootcamp -
  Jumpstart your nursing school career by enrolling in bootcamp. These online intensives accelerate learning and solidify key concepts to lock in your nursing school success.

- Straight A Nursing Podcast -
  Learn on-the-go with our 5-star rated podcast. Available on iTunes and anywhere you get your podcast fix.

- Straight A Nursing Website -
  Explore a wealth of resources at our website that includes regular blog posts on a wide range of nursing school topics. Dive in at straightanursingstudent.com

- Follow Us On Social Media -
  Facebook Page - facebook.com/straightanursingstudent
  Facebook Group - Thriving Nursing Students
  Instagram - @straightanurse
  Pinterest - Straight A Nursing


