

Pathophysiology of Artherosclerosis

Look up the pathophys..what causes it. The consequences of artherosclerosis are decreased blood flow and decreased elasticity. The difference between chronic and acute artherosclerosis relates to the time it takes to develop collateral vessels and the amount of ischemia, pain and necrosis.

Congestive Heart Failure

The definition of CHF is “The situation when the heart is incapable of maintaining a cardiac output adequate to accommodate metabolic requirements and the venous return.” (E. Braunwald) It affects 4.6 million Americans, with 400,000 new cases each year. 260,000 people die each year from this disease, and many of these pts died without ACEi.

Right vs. Left

The two things that basically occur with CHF is that 1) the heart is not pumping enough blood, so you get decreased BP, decreased cardiac output and fatigue; and 2) blood backs up!

Most common symptoms of CHF are dyspnea and fatigue.

In left heart failure, 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. If the right heart fails, you get peripheral edema and a tender/enlarged liver.

CHF Etiology and Risk Factors

Heart failure may result from a primary abnormality of the heart muscle (like an infarction) that impairs ventricular fxn 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 a-fibb.
- 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.

Factors Favorable to Failure

arrhythmias
bradycardia
pregnancy
thyrotoxicosis
pulmonary embolism
infectins
anemia
increased physical activity
increased salt or water intake
emotional stress
failure to comply with heart disease Tx

To sum this up:

- Heart attack
- High BP...the heart is pumping against increased pressure, so the left heart is affected.
- Lung disease decreases O₂, the body compensates with vasoconstriction in the lungs causing a lot of resistance and right heart has trouble pumping.

Determinants of Ventricular Function

- Contractility: How well does the heart contract? This affects stroke volume, which affects cardiac output.
- 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)
- Afterload: Is the heart pumping against a lot of pressure? If so, stroke volume will go down.
- Heart rate: less time for ventricular filling, so reduced cardiac output.
- LV wall integrity, synergistic LV contraction and valvular competence all related to cardiac output.

Evolution of clinical stages

CHF can range from asymptomatic LV dysfunction all the way to refractory CHF. If we treat it early before symptoms appear, we have a better chance of treating the disease.

- Normal heart has no symptoms, normal exercise and normal LV fxn
- Asymptomatic LV dysfunction has no symptoms, normal exercise and abnormal LV fxn (EF may be down, but you'd only know this if you did an ECHO)
- Compensated CF has no symptoms unless exercising and abnormal LV fxn
- Decompensated CHF has symptoms when not exercising and abnormal LV fxn
- Refractory CHF has symptoms that are not controlled with treatment. This guy needs a heart transplant.

Treatment Objectives

The goals with treating CHF are to increase survival, decrease morbidity (well, duh), 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 BP is low, the neurohormonal response activates the SNS...this stresses the heart (think about what the SNS does to the heart). So, the heart remodels which means it gets fibrous tissue. Unfortunately, fibrous tissue does not work as well so we will turn off the neurohormonal response with medications to prevent fibrosis in the heart.

Treatments for CHF

The main goal with CHF is to correct aggravating factors:

- Pregnancy
- Arrhythmias (AF)
- Infections
- Hyperthyroidism
- Thromboembolism
- Endocarditis
- Obesity
- Hypertension
- Physical activity
- Dietary excess
- MEDICATIONS (note that digoxin causes arrhythmias, which are probably never good)

Key Points

Know that CHF used to be managed with the goal to relieve the symptoms...now the progression of the disease can be altered. Yay!
The key to this is early interpretation with the appropriate drugs. B-Blockers and ACEi can slow the disease by turning off the neurohormonal pathway.

The disease is classified by the New York Heart Association as Class I-IV, based on the ability of the pt to exercise without symptoms.

The disease is staged as A-D, based on the evolution of the disease.

- A = High Risk. 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 angiotensin II RB 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 Features

Fatigue and dyspnea
Cheyne Stokes
Orthostatic Hypotension
Liver tenderness
Peripheral edema
Pulmonary crackles
Weak pulses

Q: A 56 year old tests negative for a heart attack. His BP is 145/84 and he has an ejection fraction of 55%. He denies dyspnea and walks several miles a day. On discharge from the hospital, which meds would you anticipate he should have?

A: A thiazide diuretic...his BP is just too high to ignore. He won't get the other therapies because his EF is ok, and he has never had a heart attack.

Various Drugs for Treating CHF (need to look over the slides, and maybe look these up)

- 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 ang I. Ang I is then converted into ang II via the angiotensin converting enzymes (ACE) in the lungs (also in the endothelium of blood vessels in many parts of the body). Ang 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 it shows that pts who received quinapril did not require any additional treatment, as compared to the group who took quinapril for a while and then took a placebo.
 - The advantages of ACEi are:
 - Inhibit left ventricular remodeling post myocardial infarction
 - Modify the progression of chronic CHF (increased survival and decreased hospitalizations, also improved quality of life)
 - In contrast to other vasodilators, does not produce neurohormonal activation or reflex tachycardia
 - Tolerance to its effects does not develop!

- Studies show that the probability of death decreases when taking Enalapril vs a placebo.
 - ACEi indications are:
 - Clinical cardiac insufficiency (all patients)
 - Asymptomatic ventricular dysfunction (LVEF < 35%)
 - High risk for CHF pts (DM, HTN, ASVD, s/p MI)
- Angiotensin II Inhibitors (AKA AT1 receptor blocker, or ARB)
 - These drugs block vasoconstrictor and aldosterone-secreting effects of ang II at various receptor sites including vascular smooth muscle and the adrenal glands. It leads to vasodilation, an antiproliferative action and lowers blood pressure!
 - Some common ARBs are Losartan, Valsartan, Irbersartan and Candersartan.
- Diuretics
 - Thiazides act on the cortex of the kidney. They inhibit active exchange of Cl and Na in the cortical diluting segment of the ascending loop of Henle. They increase the excretion of sodium and water by inhibiting sodium reabsorption.
 - K-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 not too high! They also save H ions...will this affect pH?
 - Loop diuretics act on the medulla and inhibit the exchange of Cl, Na, K in the thick segment of the ascending loop of Henle.
- Digoxin
 - Digoxin binds to the Na/K/ATPase pump in the membranes of heart cells and decrease its function. This causes an increase in the level of sodium ions in the myocytes which then leads to a rise in the level of calcium ions. This causes an increase in the length of Phase 4 and Phase 0 of the cardiac action potential, which when combined with the effects of Dig on the NS, leads to a decrease in heart rate. Increased amounts of Ca are then stored in the sarcoplasmic reticulum and released by each action potential, which is unchanged by dig. This leads to increased contractility of the heart. Dig also increases vagal activity via its action on the CNS, decreasing the conduction of electrical impulses through the AV node.
 - 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.
 - Studies show that when pts are given digoxin (along with a diuretic and ACEi), the CHF does not experience dramatically increased worsening (as compared to pts who received a placebo instead of digoxin).
 - The long term effects of dig are:
 - 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. Anginal pts 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. This also decreases proximal capillary hydrostatic pressure, which reduces capillary fluid filtration and edema formation (which is a result of heart failure).

- Nitrates

- Have several different affects:
 - Venous Vasodilation (reduce preload leading to reduced pulmonary congestion, reduced ventricular size, reduced ventricular wall stress and reduced MVO₂)
 - Coronary Vasodilation increases myocardial perfusion
 - Arterial Vasodilation decreases afterload leading to decreased cardiac output and decreased BP
- Tolerance
 - Develops with all nitrates
 - Is dose-dependent
 - Disappears in 24 hours after stopping the drug
 - Can be avoided
 - Use the last effective dose
 - Create discontinuous plasma levels
 - Intermittent administration (nitrate-free period)
 - Allow peaks and valleys in plasma levels
- Hold Nitrates when BP is low...it will drop BP even more!

- Aldosterone Inhibitors

- The one discussed in class is called Spironolactone...it is a diuretic, but not as potent as Lasix. It is the only diuretic that actually makes people live longer. It is a competitive agonist of the aldosterone receptor in the myocardium, arterial walls and kidney. It BLOCKS aldosterone which decreases the fibrous tissue formation. Note that this drug conserve potassium, so you will hold the drug if the pt becomes hyperkalemic.

- Beta-Blockers

- The Beta Blocker discussed in class is Carvedilol. It blocks Beta-1 (myocardial) and Beta-2 (pulmonary, vascular, uerine) adrenergic receptor sites. It also has alpha-1 blocking activity which means it may result in orthostatic hypotension. Be careful when getting this pt out of bed!
- Therapeutic effects are decreased heart rate and BP, improved cardiac output, slowing of the progression of CHF and decreased risk of death. Yay!
- These drugs have other awesome possible benefits:
 - Increased density of B-1 receptors (not sure what this means)
 - Inhibit cardiotoxicity of catecholamines

Remember This!
Beta Blockers end in "olol"
ACEi end in "pril"
ARB end in "sartan"

- Decreased neurohormonal activation
 - Decreased HR
 - Antihypertensive and antianginal
 - Antiarrhythmic
 - Antioxidant
 - Antiproliferative (I've got to look this up and see what it means!)
- Survival of pts on Beta-Blockers is pretty darn good. See the slides to get the details, but basically, if people are on B-Blockers and ACEi, the mortality rate goes down to 13.3% (down from 27.7%)
- Anticoagulants are given for lots of really interesting reasons (interesting if you're a NS!)
 - Previous embolic episode
 - Atrial fibrillation
 - Identified thrombus
 - Left ventricular aneurysm (3-6 months post MI)
 - Class III-IV CHF in the presence of an Ejection Fraction less than 30%, and/or an aneurism or a very dilated left ventricle.
 - Phlebitis (inflammation of a vein)
 - Prolonged bed rest (ok, that one's not interesting)

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 (ACEi and B-Blockers, plus others discussed above)

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. Once again, Dr. Van warned us about getting the arterial diseases all combobulated with the venous ones.

Probably the best thing to do would be to look at the table in the book. In general, here's what to look for when determining arterial vs. venous:

- Location
 - Arterial: Can be in aorta, legs? What about arms?
 - Venous: Is this always in extremities?
- Characteristics
 - Arterial: Dependent rubor, pallor with elevation, hypertrophied toenails, cool skin, hairless extremity, tissue atrophy.
 - Venous: Red color to skin, induration, warmth
- Pain
 - Arterial: Typically brought on by exercise and relieved by rest (intermittent claudication); sometimes unremitting pain in foot even at rest.
 - Venous: Tenderness along vein, discomfort may be relieved by applying heat
- Pulses
 - Arterial: Weak or absent
 - Venous: not sure?
- Surrounding tissue
 - Arterial: Gangrene, delayed wound healing

- Venous: Edematous (did I just make up a word?), itchy and scaly skin, thick/coarse/brownish skin around the ankles

Assessment for PVD is the check for CSM. To manage it, we're going to do a few really fun things:

- Reduce risks
- Clot prevention/dissolution
- Surgery

Aneurysm

- Classification
 - Saccular = a unilateral outpouching
 - Fusiform = a bilateral outpouching
 - Dissecting = a bilateral outpouching in which layers of the vessel wall separate, creating a cavity
 - False = the wall ruptures and a blood clot is retained in an outpouching of tissue... or there is a connexion btwn a vein and an artery that does not close
- Pathophysiology.
 - 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.
 - True aneurysms contain all three layers of the arterial wall; saccular have a neck and mouth; fusiform involve the entire circumference; a dissecting is not a true aneurysm, but is a hematoma in the arterial wall that separates the layers of the wall.
 - Other causes of aneurysms include infection, mycotic infections and even trauma (though that last one is rare)
- Clinical Manifestations
 - AAA
 - Most abdominal ones are asymptomatic, can be palpated once it's at 5 cm (unless obese...see, another reason to go for a run!).
 - 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 dx tools
 - Ruptured AAA
 - Pulsating sensation in abdomen
 - Abrupt excruciating pain (ripping or knife-like that radiates)
 - Hypertension (but elsewhere it says hypotension?)
 - Manifestations of shock (pallor, tachycardia, hypotension, dry skin, excessive thirst)

- Diminished peripheral pulses or unequal pulses
- Abdominal rigidity
- Differing BP in arms
- Paraplegia, hemiplegia
- Decreased urine output or hematuria

Raynaud's Syndrome

An arterial disease in which the small arteries and arterioles constrict in response to various stimuli. This can be caused by cold, nicotine, caffeine and stress. Obstructive Raynaud's often seen with autoimmune diseases.

Buerger's Disease

This 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. Pts may also have cold sensitivity with color changes and pain. Pulsations 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 pt don't stop smoking they are very likely to lose fingers (or whatever part is affected). This disease is hard to treat...drugs don't work very well.

DVT

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

- Endothelial injury due to trauma or surgery
- Circulatory stasis due to immobility
- Hypercoagulable state due to birth control pills (may be other things, not sure)

Prevention and Treatment of DVTs

- Heparin
- Warfarin (COUMADIN)
- TEDs/SCDs (prevention only)
- Elevation
- Early ambulation (prevention only)

Mechanism of Action of Antithrombotic Agents (sounds so fancy!)

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

Pulmonary Embolism

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

What to look for:

- First sign is usually dyspnea, may be accompanied by angina or pleuritic chest pain
- Tachycardia
- Air hunger
- Feeling of impending doom
- 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
- Give heparin
- If can't tolerate heparin, then surgery

Edema

Lymphedema occurs when lymphatic flow is blocked, but can also occur with low serum protein and high venous pressure. It is treated by elevating the affected body part, compressing the affected body part, providing skin care and infection treatment and 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/sclerosis causing lower cardiac output, arrhythmias and valve incompetence. The vasculature changes as well, with decreased elastin and increased arteriosclerosis.

Nursing Dx (related to???)

- Decreased tissue perfusion
- Health maintenance (risk reduction, medication compliance)
- Knowledge deficit

Labs, labs, labs!

Na < 135 = fluid overload

K < 3.5 = loop diuretics

K > 5.0 = renal insufficiency

BUN > 20 = renal insufficiency

BUN < 7 = malnutrition

Creatinine > 1.2 = renal insufficiency

INR 1 = normal, 2-3 therapeutic warfarin level

aPTT 30 = normal, 60-90 therapeutic heparin

Cholesterol > 200mg/dl = risk; >240 mg/dl = high risk

LDL >130 mg/dl = risk

HDL <37 = male risk; <47 = female risk

Triglycerides >200 mg/dl = risk; > 400 mg/dl = high risk

C-reactive protein = women < 1.5 mg/l; men < 5mg/l

Outcomes

BP < 130/86, No MI, CVA or renal failure

Brain Attack, CVA, Stroke

Most strokes are caused by thromboemboli! The concepts of perfusion/stasis are the same as for CV disease, as are the risk factors. Brain attack is the 3rd leading cause of death in the US and is very expensive for both the government and the individual patient. The good news is, the death rate due to brain attack has declined due to better risk prevention and management, quicker diagnoses and more timely intervention technologies.

Causes & Pathophysiology Brain Attack

Thromboembolic, hemorrhage and 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 pt to the hospital within 3 hours (and it's a thromboembolic stroke), then that person can fare very well with the administration of TPAs (which dissolve clot).

Stages of Brain Attack

- TIA (transient ischemic something or other)...these symptoms resolve on their own in minutes or hours. They are caused by microemboli from plaque. However, all is not peaches and candy for this person...they are likely to go on to have a bona fide CVA.
- RIND (reversible ischemic neurological disease) symptoms persist for 24-48 hours. There is ischemia, but no necrosis...complete recovery!
- Stroke in evolution: 20-35% of pts have symptoms that get worse over the course of the week after the CVA.
- Completed stroke causes permanent neuro damage.

Clinical Manifestations of Brain Attack (CVA)

The clinical manifestations have to do with which blood vessel is involved (see book). 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
- Internal Carotid Artery
 - Same as above, plus...
 - Ipsilateral visual impairment
 - Ipsilateral Horner's syndrom (ptosis, miosis, no sweating on same side of face)

With both you have decreased LOC, could have seizures and VS changes. Visual problems vary with the site of the stroke...see book for diagram of how the ocular pathways criss-cross. 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
 - Warfarin for atrial fibrillation
 - Aspirin
 - Aspirin/extended release dipyridamole
 - Not heparin (except for DVT/afib*)
- Surgical revascularization

*Pts with past history of afib are taught to feel radial pulse to evaluate return of irregular heart beat and to self administer LMWH prior to seeking medical care. Several strokes have been prevented!

Treatment: Evolving Stroke

- 911...get the stroke team activated!
 - Stabilize airway, O2, BP, CT scan
 - Give or not to give thrombolytics
- Maintain cerebral perfusion
 - Reduce swelling
 - BP < 200/120 (<185/110 with TPA plans). You might think “WOAH”, that BP is high! Well, we want it to be high so the blood can perfuse the brain.
 - Good ventilation
 - Mannitol: osmolar agent used in first 72-96 hours (pulls fluid out of cell to decrease edema)
 - Steroids: Not useful except after SAH (subarachnoid hemorrhage)
 - HOB up slightly (though this may get sticky with MD...let them has it out)
 - Glyburide: experimental, reduces swelling
 - Prevent vasospasm: nimodipine (a calcium channel blocker)...used for bleeds but not ischemic strokes.
- Protect brain cells...make sure no fever, use neuroprotectants (experimental drugs)
- Surgery: external decompressions, remove clot with hemorrhagic stroke
- Seizure prevention (not sure how you prevent a seizure)...anti-seizure meds?

When CO2 goes up, blood vessels in the brain dilate, causing increased pressure in the brain, leading to decreased perfusion.

Rehab/Treatment: Evolving and Completed Stroke

In this stage you will protect your pt 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)
- No contractures (position of max function, ROM)
- Bowel and bladder function support
- Communication
- Interaction with environment when visual field impaired
- Depression

Q: A patient is in rehab until 2 weeks after a right middle cerebral artery infarct. Which is the best plan for activity?

A: the pt should learn how to get up from the right side of the bed (they have left-sided weakness).

Q: Which intervention will be most helpful to a pt with aphasia?

A: Encourage use of gestures when communicating

Q: W pt with a vertebrobasilar stroke is being managed for dysphagia. Which intervention will be most helpful?

A: Hmm... not sure on this one.