Chapter 26 – Disorders of Cardiac Function

Pericardial Disease.
Pericardium surrounds the heart, has 2 layers, which he says we don’t need to know. Pericardium can become inflamed without heart affect. If short term, no big deal, decent recovery. If long term, inflammation will scar the Pericardium. As tissue heals it tends to tighten and lose flexibility. In the pericardium, this becomes “restrictive pericarditis” by diagnosis. (And yes –itis means “inflammation” which is now resolved by the time the scarring forms, but too bad for us, that’s still what it’s called. ) Clinically, looks like heart attack – SOB, fluid accumulation, diaphoresis (excessive sweating prior to an MI or in ppl about to go into shock), extreme sharp pain (where heart attack is a dull deep ache)

Pericarditis manifests with sharp chest pains with a “friction rub” which in Biomed Terminology was called rales, I think. Sounds like leaves crackling. The pain is precordial, radiating to the neck, back, abdomen. Also, a strong sign of this is it is relieved by sitting up and leaning forward. To lean back or lay back really intensifies the pain. The pain is also worse with breath in, coughing, swallowing.

Chronic pericarditis with effusion is quite rare if patient doesn’t have autoimmune diseases like lupus or scleroderma. Often found incidentally on an Xray. This is different from the previous paragraphs – people seek out medical help for that thinking they have heart attack. In chronic pericarditis the exudate will eventually restrict the filling of the cardiac chambers.

Constrictive pericarditis – fibrous scar between visceral and parietal pleura. This is a scar with retraction of scar which compromises diastolic filling of the heart. Can lead to ascites. Might see “Kussmaul’s Sign” which is a really enlarged vein in the neck when the patient takes a deep breath.

Coronary Heart Disease
This can be CAD, coronary artery disease, which is a restriction of blood flow to the heart organ itself. Can also be arteriosclerotic cardiovascular disease. Coronary arteries are unable to supply the heart with a needed amount of blood. Can be due to coronary artery spasm, blockage, wall thickening/hardening. Arteriosclerotic Cardiovascular Disease, which is this thickening and hardening, is very common.

Vasospastic Coronary Artery Disease is a major spasm of the coronary artery cutting off blood flow—no blockage, no hardening or thickening, but still this is a heart attack. No pain associated or if pain, in left shoulder, middle of back, left side of the jaw. This is an atypical heart attack.

Know: there are a variety of major arteries circulating to the heart. See slide 14 of Ch 26 - Disorders of Cardiac Function.pdf. By the way, LAD = left artery descending.
If the heart chronically cannot get blood somewhere due to occlusion, collateral arteries will form. This will function pretty well until the main artery that was blocked by about 75% at which time the collaterals can’t keep up anymore.

Atherosclerotic Heart Disease: occlusion created by a thrombus. Gotta worry about 1) will the plaque formed here block the artery? And 2) what happens if the plaque ruptures and sends an embolus downstream, thus blocking an artery rather thoroughly? That’s usually a fatal heart attack.

Aspirin makes the platelets less sticky, keeps the plaques from forming. A plaque is a blockage with a lipid-rich core with a fibrous cap on it. The size of the core and the thickness/stability of the fibrous cap determines whether or not the plaque will rupture. Another determining factor of plaque rupture is the presence of inflammation with plaque degeneration. A lack of smooth muscle cells with impaired healing and plaque stabilization are the final factor. Ruptures change spontaneously, with a change in hemodynamics (like a blood pressure spike). Also, there is an increased risk in the 1st hour after waking. This is what is referred to in slide 18

Ischemic Heart Disease
Slide 20
Ischemia is basically a deficient or compromised blood flow. Ischemia isn’t dead tissue, but compromised tissue, and almost always causes pain. In the case of heart related ischemic pain, this is called “Angina.” There are 3 kinds:

- Stable angina – has pain, doesn’t lead to heart attack when they get it. Treat it by giving nitroglycerine which opens the arteries and lets the blood flow through. If nitro won’t work for whatever reason, patient breathes pure oxygen and pain goes away.
- Unstable angina – pain, but is heading to a full MI (myocardial infarction which = dead heart tissue).
- MI

Stable Angina is a fixed coronary obstruction of 75% or more, leading to a disconnect between demand for oxygen + and the ability of the body to deliver those needs. This might be due to increased metabolic demands on the heart – exertion, emotional stress, cold. Manifestations of stable angina = pain which is substernal and in front of the heart (about 3 inches to the left of center). This pain is steady, squeezing, crushing, or suffocating. It may radiate to the left shoulder, arm or jaw. It can often be confused by arthritic or musculoskeletal pain, or heartburn/indigestion. Stable angina can be relieved by 1) NTG or nitroglycerine or by 2) rest. Usually better within 5-10 minutes. If not, it’s probably not ischemic…or is severe ischemia.

Prinzmetal’s Angina
This is the vasospastic type. Can occur at rest, can occur without atherosclerosis. Often happens at night. Some sources say increase your intake of magnesium to prevent the vasospasm – inhibits overreactivity of smooth muscle (governed by the autonomic nervous system) all over the body – uterus, lungs, bowels, heart, etc. Well accepted in western medicine, but underemphasized.

Cardiac Enzymes
Slide 25. These enzymes are produced by the death of cardiac cells – normally they are in the cells. When the cells die, the enzymes are released and you see them freefloating in the blood. Very useful for evaluating whether an MI has occurred. If levels are normal/immeasurable, no MI. The problem is that it sometimes takes up to 12 hours before you can see them in the blood! Bummer.
Unstable angina
Basically, patient is heading for an MI here. This is part of the way between stable angina and MI. Can be a primary disorder, or a progression from stable angina, can be a secondary disorder like the result of cocaine, anemia, or other non-coronary related things. Pain is often more intense and persistent. Serum markers and enzymes determine if it’s an actual heart attack or just unstable angina.

Cardiomyopathies
These are dysfunctions of the heart organ independent of the artery diseases.

Cardiomyopathy is disorder of the heart organ for reasons independent of circulation. Cardiomyopathy can evolve from damage to the muscle, swelling, inflammation, etc. Can be primary or secondary

Primary Cardiomyopathy can be classified in the following ways:
1. Dilatation
2. Hypertrophy of the cardiac muscle.
3. Restriction (opposite of hypertrophy), often due to childhood diseases, many of which we’ve vaccinated away for now.

Hypertrophic Cardiomyopathies
Most common type – sudden cardiac death in young ppl or athletes for instance. IHSS is the heart growing beyond the capacity of the stenotic aortic valve. Another problem is asymmetrical septal hypertrophy - the septum gets very large and dysfunctional. The result is a communication problem between the parts of the heart and a non-coordinated contraction. The heart begins to quiver and fibrilate – fatal. Very vigorous activity can lead to this type of heart failure. This is an inherited autosomal dominant thang. Most easily diagnosed with and echocardiogram. Remember when this happened to a kid in AISD? Now they require ECG’s before they let kids play vigorous sports.

Peripartum Cardiomyopathy
Rare condition. This is a left ventricle dysfunction, occurs from 1 month before delivery of a child up to 5 months post-partum. Risk factors: advanced maternal age, multi-fetal pregnancies, pre-eclampsia, gestational hypertension. It has a mortality factor of 18-56%. The most likely cause is myocarditis (inflammation of the heart wall).

Valvular Heart Disease
Much more rare now due to antibiotics. A while back, respiratory infection migrated to the heart more frequently and did damage to the heart valve. Rheumatic fever often caused mitral valve disease – streptococcal bacteria infection. Another reason not so much any longer is that valve replacement is possible and frequently done. Mitral and Aortic valves are the most commonly affected when it happens.

If the valve diseased is the one on the right, this is congenital.
**Mitral Stenosis** is the most common type of valvular heart disease. The stenotic (dysfunctional) valve resists the flow of blood, failing to open fully and close fully. It is no longer pliable, but rigid and firm and fibrous (i.e., stenotic). Symptoms are like pulmonary congestion – shortness of breath, orthopnea (hard to breathe if lying down), paroxysmal nocturnal dyspnea (PND – wakes up at night short of breath). Palpitations, chest pain, weakness, fatigue, PAC’s (premature atrial contractions which leads to…→), atrial fibrilation.

When a patient has this you can hear the blood rushing thru the stenotic valve – this is called a “murmur” which means you can hear the flow because it is turbulent. Sounds like a fingernail scraping against something ridged.

[Click here for a website that plays the sounds for you.](#) Click on the Systolic and Diastolic tabs to hear the different ones mentioned in this section.

**Mitral Regurgitation**

**Mitral Valve Prolapse**
Most are asymptomatic, but some get anxiety, palpitations, and a vague sense of forboding (vague chest pain). Murmur won’t always be audible.

**Aortic Stenosis**
Can be from rheumatic fever, congenital malformation, trauma. Left ventricle is pushing hard and the valve doesn’t want to let the blood thru.

**Aortic Regurgitation**
Review cardiac function and rhythm functions from A and P.

**Premature Ventricular Contractions**
Also called PVC’s. Contraction occurs just before it’s actually supposed to. Ventricle isn’t filling completely, insufficient ejection of blood into the arterial system. The pulse will be absent or greatly diminished. **This is clinically insignificant in absence of heart disease!** In a patient with cardiac disease, however, this predisposes the patient to serious arrhythmias.

**Ventricular Tachycardia**
V-TACH is a regular, consistent rate of contraction, but is extremely rapid. Almost always converts to more serious arrhythmias that are irregular. It’s very difficult for the heart to maintain regularity when it’s beating that fast. 150bpm is common, but can go as high as 250bpm. Patient often loses consciousness when this happens.

Ventricular tachycardia often degrades into ventricular fibrillation…

**Ventricular Fibrillation**
VFIB. The ventricle “quivers” but can’t contract – there’s no cardiac output. This is fatal if not reversed within minutes by either cardioversion or defibrillation.

**Heart Block**

Is a disruption of conduction along the path of the AV node thru the Bundle of Hiss and the Perkinje fibers.

**Bundle of Hiss:**
The bundle of His is not easily visible grossly, because of its blending with adjoining tissues. It extends leftward along the top of the interventricular septum, from its beginning in the subendocardium of the right side of the atrial septum just above the annulus of the tricuspid valve. It branches after it reaches the membranous septum into the right and left bundles.

• A section of the IV septum perpendicular to the course of the bundle at the posterior edge of the membranous septum may reveal a 2 mm tan cross section of the bundle just above the septal myocardium surrounded by the gray tissue of the membranous cardiac skeleton.
Heart Block can be 1st degree – no symptoms, no treatment needed. 2nd degree and 3rd degree which are potentially fatal and require a pacemaker. In 1st degree part of the signal to beat is going through. In 2nd there is less – skips regularly in every 3rd or 4th beat. In 3rd degree it’s unpredictable and much of the signal to beat is not getting through. Symptoms are light headed, rate of beat is about 30-40, feel fatigued.

This is a slow arrhythmia. Often the 2nd and 3rd degree flavor is the result of an MI which has damaged the tissues that control beat.
Chapter 28 - Heart Failure and Circulatory Shock

Involves the decreased pumping ability of the heart and therefore cardiac output as well as the ability of the heart to increase the stroke volume. This is called a decrease in “cardiac reserve.”

Stroke volume is the amount of blood ejected per cardiac cycle. Cardiac output is the stroke volume multiplied by the heart rate. The heart rate is regulated by the autonomic nervous system. Stroke volume is regulated by:

1. Preload – how much blood the heart has at the end of the relaxation phase or how much goes into the heart before it can contract. Preload is the end-diastolic volume.
2. Afterload – resistance to the contraction of the heart. AKA, the force the heart has to use to eject the blood that’s in it. This is affected by systemic vascular resistance and ventricular wall tension. Afterload can impair the ejection of blood from the ventricle and increase the tension of the ventricular wall. Myocardial demand for oxygen is proportional to wall tension. If wall tension goes up, so does the need for oxygen.
3. Contractility – this can be enhanced by exercise. This is the ability of the contractile parts of the heart to interact with and shorten against a load of blood.

Congestive Heart Failure

Heart fails due to congestion of body tissues whether pulmonary or system venous. Basically, that’s a hydraulic problem! Often caused by acute MI, hypertension, cardiomyopathies. Excessive work demands can cause it too—not occupationally, but hypermetabolic states and fluid overload. Valvular stenosis and regurgitation can push this issue too.

Diastolic Failure is the impaired filling during diastole – a preload failure. 
Systolic Failure is impaired filling during systole.

CHF, or congestive heart failure has elements of both. CHF is fluid retention, legs or lungs or both. Edema can show in ascites which is congestion of the viscera. Pitting edema, weight gain, and more fun stuff. See slide 67.

Right versus Left Side Heart Failure

Left is more common. If right side heart failure, you’ll see the signs more in the lower extremities. Either way, patient has orthopnea – sob upon lying down, fatigue, pulmonary edema. Almost always on ACE inhibitors and diuretics. See slide 69-70.

These patients need lower blood pressure. Western Med tries to shift the fluid from the extra-vascular areas into the vascular areas – increase the urine output dramatically. Kidneys will then release hormones facilitating the migration of fluids to the vascular areas.

<<AND it’s 5:15pm and we should be on midterm break NOW!!! – TK, be glad you aren’t here.>>

Cardiogenic Shock

Pump failure – heart just completely fails. Usually caused by MI. Arrhythmias, disruption of valvular function (including a ruptured chordae tendoneae – tendons attached to the valves of the heart) and endstage coronary artery disease as well as cardiomyopathies can also cause this.
Cardiogenic shock results in sudden loss of cardiac output. All shock is defined by hypotension – including this type of shock.