OVERVIEW

For purposes of this study, a breakdown of cardiac diseases will be divided into three categories: vascular, structural, and electrical conductivity.

VASCULAR
Vascular will include diseases of the coronary arteries. Those vessels which supply blood to the heart muscle itself. These vessels are the site of myocardial infarctions (heart attacks) when they become blocked. This is due to an imbalance between oxygen supply and demand in the heart muscle itself. The types of diseases we see with these vessels are coronary artery disease (CAD), angina pectoris (stable, effort angina), acute coronary syndrome (unstable angina), atherosclerosis and arteriosclerotic coronary vascular disease (ASCVD). Surgical treatments for these blocked arteries are coronary artery bypass graft (CABG) or angioplasty (balloon insertion and expansion), or stent placement.

The blockage could occur through a progressive narrowing of the inner walls of the artery through plaque accumulation which is commonly associated with high cholesterol. Nitrates (Transderm Nitro, Isordil, lmDur (long acting), IsMo (short acting), Sombritrate, nitropaste, NTG tablets/spray, NitroBid, etc.) are commonly prescribed and work by causing vasodilation which then allows more blood flow through the vessels. Ranexa (ranolazine) is a new agent with a novel mechanism of action that appears to block "late sodium channels" in heart muscle cells, which appears to decrease the damage the heart cell may receive from low oxygen states (ischemia).

These vessels may also have episodes of where they contract (spasm) and thus decrease blood flow to the heart muscle. This is known as vasospastic angina, variant angina, Prinzmetal's angina or angina inversa and may occur in cycles without any trigger such as exercise. Calcium channel blockers are the primary treatment course. They try to keep the vessel from contracting by keeping calcium from entering the cell (block the calcium channel) and thus leave the vessel in a resting state vs. contracted state. Beta-blockers are utilized as a way to try to keep the heart from exercising too much and putting an oxygen load (demand) and subsequently getting angina symptoms.

When the vessels are about 90% blocked or clogged, the symptoms of angina may occur. Pain (arm, jaw, neck, back, shoulder) may or may not (silent angina) be present with angina. Atypical symptoms including breathlessness, pallor, weakness, nausea, or epigastric discomfort or burping may be the only ones manifested.
STRUCTURAL
The structural elements of the heart include the vessels going into the heart: inferior and superior vena cavae, pulmonary vein and out of the heart: pulmonary artery and aorta. Also, are the four chambers: left and right atriums and left and right ventricles. The tricuspid controls blood flow from the right atrium into the right ventricle, the pulmonary valve from the right ventricle to the pulmonary artery. The return blood from the lungs enters the left atrium and the mitral valves controls the flow to the left ventricle. Finally, the aortic valve allows blood flow from the left ventricle into the aorta.

Diseases occur with the valves: stenosis (stiffness) which can lead to partial open or closed states and irregular blood flow which opens the door for infections in the heart, primarily valves (endocarditis) or clot formation (thrombosis). A prolapsed valve, primarily occurring in the mitral valve, means one or more of the valve leaflets is weak and floppy which then doesn't allow the valve to completely close. The end result could allow blood to flow backwards during the contraction which is then called regurgitation. Any one of these disorders will show symptoms in the patient of weakness, tiredness, shortness of breath, exercise intolerance and possibly edema. Most can be treated symptomatically with vasodilators (hydralazine, nitrates), diuretics, digoxin, antiarrythmics, and perhaps anticoagulants to try to prevent clot formation. If symptoms proceed to a significant state, valve replacement (artificial or pig valve), open heart surgery, may be necessary.

Heart muscle wall disorders occur primarily as a result of ischemic episodes from myocardial infarctions in the coronary arteries. This lack of oxygenated blood to the heart muscle will cause these downstream cells to die. The result of is this affected muscle to turn into scar tissue which is stiff and no longer pliable. The heart then contracts with "sort of a limp", in terms of an analogy to walking. It is not as effective in its contraction. If it occurs in either of the right atrium or ventricle symptoms will more likely show up as pedal edema due to back up of blood coming from the body via the vena cavae. If the damage is on the left side, then symptoms will initially show as congestive (lung, congestion, sob, exercise intolerance) symptoms due again to the back up of blood in the lungs. Since the left side of the heart, primarily the left ventricle, is the main pump of the heart, both lung and pedal edema may be seen. The degree and extent of damage to the heart muscle will determine the degree and type of symptoms seen clinically. A second cause of heart failure occurs from the heart muscle becoming too large, primarily due to long-standing hypertension. The heart muscle is a unique muscle in that once it reaches a certain size, any increase (cardiomegally) above that size will cause it to then become weaker. Symptoms will mimic CHF due to an ineffective pumping of blood through the heart. The term cardiomyopathy ("bad heart") may be seen as either a catch-all term or when the heart disease crosses over with multiple disorders in the heart.

Medication treatment usually starts with an ACE or ARB (try to decrease the pressure against which the heart has to push blood out) and loop diuretics (decrease total fluid load in the body) . Beta-blockers (low dose) which were once considered contraindicated in treating CHF are now almost considered necessary in most cases. They work as seen above in CAD, to keep the heart from exercising too much which could lead to more damage (cardiomegally) and congestive symptoms. Digoxin used to be the mainstay of CHF treatment, but has been waning through the years. Amiodarone use has shown a large increase in use for in some cases and areas of the country, depending on the prescribing habits of the local physician cardiology group in the local area. Both agents are thought to
try to increase the strength of the contraction. When any of the above treatments are not effective or tolerated, the combination of hydralazine with a nitrate is often seen. Spironolactone may or may not be seen in combination with the loop diuretics. Zaroxylyn is often added in end stage situations for added diuresis, usually only a few times per week, at the risk of a large potassium loss which almost always requires a large potassium supplementation.

ELECTRICAL CONDUCTIVITY
Each cardiac cell has its own ability to beat on its own, sort of like a single instrument in an orchestra. When joined together as a heart they still will beat at their own individual rate. These cells need something to tell them to beat in a synchronized rhythm, as in a conductor for a band or symphony. The part of the heart that does this control, or conductor, service are first in the SA (sino-atrial) node, which, as being the natural pacemaker, tells all the cells in both atriums to beat together. Then through the internodal pathways the impulse goes through to the AV (atrioventricular) node and down the bundle branches which conduct the cells in the ventricles to beat together. Hence, we then hear the "lub-dub" of the heart beat.
Abnormalities in this system will have the possibility of the cardiac cells or groups of them, which were under control to beat in rhythm to now beat at their own rate. This may lead to rate changes (tachycardia or bradycardia) or irregular beats (arrhythmias).
- ATRIAL ARRHYTHMIAS
When this occurs in SA node/atrium we see atrial fibrillation (AF, uncoordinated quivering of the atriums), premature atrial contractions (PAC, benign, palpitations), wandering pacemaker (natural, not mechanical, SA node pacemaker shifts to the AV node back and forth, often caused by increased vagal tone), sinus arrhythmia (rate changes during inspiration-expiration), tachycardia (multifocal tachycardia, supraventricular tachycardia), and atrial flutter (sensation of regular palpitations, often converts to AF). Treat with lifestyle changes - exercise, decrease alcohol and caffeine intake, diet. Multaq (dronedarone) has been approved specifically to treat AF, though usage outside this indication may be seen. Wolff–Parkinson–White syndrome (WPW) is a syndrome of pre-excitation of ventricles of the heart due to an accessory pathway known as the bundle of Kent. This accessory pathway is an abnormal electrical communication from the atria to the ventricles and is a type of atrioventricular reentrant tachycardia.
- JUNCTIONAL ARRHYTHMIAS
Arrhythmias which occur in between the SA and AV nodes are called junctional arrhythmias and they include supraventricular tachycardia (SVT, which may appear as fast as it disappears, but could last for days with a pulse rate of 150-250 bpm) and paroxysmal supraventricular tachycardia/AV node reentrant tachycardia (PSVT, AVNRT, 75% occur in women, palpitations are the symptoms sometimes due to stress, alcohol, or caffeine and may resolve with specific exercises or medications - if simple - amiodarone or sotalol, if AV node involvement then diltiazem, verapamil or metoprolol). Ventricular arrhythmias can be as premature ventricular contractions-ventricular extra beats (PVC/VEB, may be felt as a skipped beat or palpitations, found more commonly in men).
Treatment options are the antiarrhythmics (Norpace, Enkaid, Tambocor, Mexetil, Procan, Rhythmol, quinidine, Tonocard), beta-blockers, or calcium channel blockers.
- VENTRICULAR ARRHYTHMIAS
Continuous PVCs can become a form of ventricular tachycardia (VT, VTach) causes an extra ventricular contraction and can be life threatening leading to
ventricular fibrillation or asystole. Amiodarone or procainamide in addition to defibrillation are treatment options. Ventricular fibrillation (VF, VFib) is when the ventricles do not beat, they only quiver. And will show a barely observable or no pulse and often leads to cardiac arrest or sudden cardiac death. Defibrillation is required. Amiodarone may be used as subsequent treatment. Torsades de pointes, or simply torsades is a French term that literally means "twisting of the points". It refers to a specific, rare variety of ventricular tachycardia that exhibits distinct characteristics on the electrocardiogram (twisting of the points). It is also associated with a fall in arterial blood pressure, which can produce syncope. Torsades de pointes can degenerate into ventricular fibrillation, which will lead to sudden death in the absence of medical intervention. Treatment is directed at withdrawal of the offending agent, infusion of magnesium sulfate, antiarrhythmic drugs, and electrical therapy as needed. The patient may require an unsynchronized shock (or defibrillation).
- BRANCH BLOCKS
First degree heart blocks are the most common cause of bradycardia and occur from the AV node. Causes come from increased vagal tone, myocarditis, myocardial infarction, electrolyte disturbances and medications including digoxin, calcium channel blockers, beta-blockers, and acetycholinesterase inhibitors (Aricept, Exelon, Razadyne). Bundle branch block (BBB) occurs in the branches which stretch over the ventricles from the AV node. Treatment may require a pacemaker and removal of offending medication if one -> one of the reasons we check a conditional pulse. Second degree heart block (Type I or Mobitz or Wenckebach) is a conduction block of the AV nodes. Type II (Mobitz II or Hay or infranodal) is a conduction block of the fibers distal to the AV node (bundle of HIS or Purkinje fibers) and this can progress to a life threatening situation. Symptoms are light-headedness, syncope, or fainting. Treatment is a pacemaker. Third degree AV block (complete heart block) occurs between the SA and AV nodes. There is a natural accessory pacemaker which will kick in and activate the ventricles to some degree. This is also known as an escape rhythm. Two independent rhythms may be seen on an EKG. Bradycardia, hypotension, and exercise intolerance may be seen. Dual chamber artificial pacemakers are utilized for treatment. AF or A flutter are often seen in association with this type of block.
- SADS
Sudden arrhythmic death syndrome (SADS) often results in sudden death brought on by an arrhythmia and upon autopsy there are no anatomical defects noted. The most likely cause is postulated to be coronary artery disease and there were no prior symptoms seen.

MEDICATION USAGE SUMMARY

CHF
- ACE, ARB, diuretics (loops, spironolactone, metolazone), beta-blocker (BB - low dose), digoxin, amiodarone, hydralazine/isosorbide (nitrate) combination.

CAD, ANGINA, s/p MI
- Beta-blocker, nitrates, Ranexa, calcium channel blocker (CCB)

VALVULAR (often none, varies depending on degree of symptoms)
- Diuretic, ACE, BB, CCB, vasodilators, digoxin

ARRHYTHMIAS
- Antiarrhythmics, BB, CCB, amiodarone-drnedarone, digoxin

ANTICOAGULANTS
- Used in an attempt to decrease the risk of a thrombotic event anytime there might be irregular blood flow through the heart.

STATINS
- Proven to be able to decrease the risk of a future cardiovascular event, independent of cholesterol levels.

ELECTROLYTES
- Magnesium and potassium deficiency may lead to poor cardiac performance, increased risk of medication toxicity, and arrhythmia.

HEART SOUNDS

<table>
<thead>
<tr>
<th>Mitral Stenosis</th>
<th>Aortic Stenosis</th>
<th>Aortic Regurgitation</th>
<th>Mitral Regurgitation</th>
<th>Tricuspid Regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening snap followed by a low-pitched diastolic rumble with presystolic accentuation.</td>
<td>Systolic murmur of a harsh crescendo-decrescendo type, heard in 2nd right intercostal space, radiating to the carotid arteries</td>
<td>Increased pulse pressure by increased systolic and decreased diastolic blood pressure, but may not be significant if acute</td>
<td>Holosystolic murmur at the apex, radiating to the back or clavicular area</td>
<td>Pulsus parvus et tardus, that is, diminished and delayed carotid pulse</td>
</tr>
<tr>
<td>The opening snap follows closer to the S2 heart tone with worsening stenosis.</td>
<td>Pulsus parvus et tardus, that is, diminished and delayed carotid pulse</td>
<td>Diastolic decrescendo murmur best heard at left sternal border</td>
<td>Commonly atrial fibrillation</td>
<td>Fourth heart sound</td>
</tr>
<tr>
<td>The murmur is heard best with the bell of the stethoscope lying on the left side and its duration increases with worsening disease.</td>
<td>Water hammer pulse</td>
<td>Water hammer pulse</td>
<td>Third heart sound</td>
<td>Third heart sound</td>
</tr>
<tr>
<td>Loud S1 - may be the most prominent sign</td>
<td>Austin Flint murmur</td>
<td>Austin Flint murmur</td>
<td>Laterally displaced apex beat, often with heave</td>
<td>Laterally displaced apex beat, often with heave</td>
</tr>
<tr>
<td>Advanced disease may present with signs of right-sided heart failure such as parasternal heave, jugular venous distension, hepatomegaly, ascites and/or pulmonary hypertension (presenting with a loud P2. Signs increase with exercise.</td>
<td>Decreased A2 sound</td>
<td>Apex beat displaced down and to the left</td>
<td>In acute cases, the murmur and tachycardia may be only distinctive signs</td>
<td>Atrial f usually</td>
</tr>
<tr>
<td></td>
<td>Sustained apex beat</td>
<td>Third heart sound may be present</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Antiarrhythmic Types

<table>
<thead>
<tr>
<th>CLASS</th>
<th>KNOWN AS</th>
<th>EXAMPLES</th>
<th>MECHANISM</th>
<th>CLINICAL USES</th>
</tr>
</thead>
</table>
| 1a    | fast channel blockers | quinidine, procainamide, Norpace | - Sodium channel block (intermediate association - disassociation) | - Ventricular arrhythmia  
  - prevention of paroxysmal recurrent atrial fibrillation (triggered by vagal overactivity)  
  - procainamide in Wolff-Parkinson-White syndrome |
| 1b    |     | lidocaine, Dilantin, Mexitil | - Sodium channel block (fast association - disassociation) | - treatment and prevention during a immediately after myocardial infarction  
  though this practice is now discouraged given the increased risk of asystole  
  - ventricular tachycardia  
  - atrial fibrillation |
| 1c    |     | Tambocor, Rhythmol, Ethmozine | - Sodium channel block (slow association - dissociation) | - prevents paroxysmal atrial fibrillation  
  - treats recurrent tachyarrhythmias in abnormal conduction system.  
  - contraindicated immediately post-myocardial infarction. |
| II    | Beta-blockers | Inderal, Blocadren, Metoprolol, Atenolol, Zebeta | - beta blocking  
  - Inderal also shows some class I action | - decrease myocardial infarction mortality  
  - prevent recurrence of tachyarrhythmias |
| III   |     | amiodarone, Betapace, Multaq | - Potassium channel blocker (Sotalol is also a beta blocker) | - In Wolff-Parkinson-White syndrome  
  - (sotalol) ventricular tachycardias and atrial fibrillation |
| IV    | slow-channel blockers | - Verapamil, Diltiazem | Ca2+ channel blocker | - prevent recurrence of paroxysmal supraventricular tachycardia  
  - reduce ventricular rate in patients atrial fibrillation |
| V     |     | - digoxin, magnesium | - Work by other or unknown mechanisms (Direct nodal inhibition). | - Used in supraventricular arrhythmia especially in Heart Failure with Atrial Fibrillation, contraindicated in ventricular arrhythmias. Or in the case of Magnesium Sulfate, used in Torsad Pointe. |
* 1. Of the following categories of cardiac disease: electrical conductivity, connective tissue, vascular, and structural, as described in the above summary, the one which doesn't belong is:

* 2. The category of the following cardiac diseases: ASHD, CAD, angina, ASCVD, would fall into:

* 3. Congestive heart failure (CHF) falls under which of the following categories: vascular, structural, or electrical conductivity?

* 4. Which of the following diagnoses is not a cardiac conductivity disorder: supraventricular tachycardia (SVT), atrial fibrillation (AF), coronary artery disease (CAD), bradycardia?

* 5. The monitoring symptom for coronary heart disease least likely to occur is: edema, chest pain, exercise intolerance, faintness?

* 6. Monitoring parameters for CHF include the following except: edema, weight, difficulty in breathing while in a supine position, irregular heart rate?
* 7. Symptoms of atrial fibrillation (AF) include all of the following except: weakness, palpitations, fainting, and arm pain?

* 8. Structural cardiac conditions include all of the following except: mitral valve prolapse, bundle branch block, aortic valve stenosis, congestive heart failure?

* 9. Lisinopril, carvedilol, HCTZ, digoxin or amiodarone are all common treatments for CHF except which one:

* 10. CHF and AF are commonly treated with any of the following except: Coreg, ImDur, digoxin, or amiodarone?

* 11. Medications which can affect cardiac rate and should have a conditional pulse include the following except: Coreg, isosorbide, amiodarone, Multaq, or diltiazem.

* 12. The following combinations of medications - amiodarone, Coumadin, diltiazem, metoprolol, or verapamil - are indicative of someone with which of the following diagnosis? HTN, CAD, AF, ASCVD, or mitral valve prolapse.
* 13. Nitroglycerin containing products, which treat angina, include the following except: Isordil, Transderm-Scopolamine, Imdur, Sorbitrate or IsMo?

* 14. Medications used to treat status post myocardial infarction (MI) commonly include any of the following except: beta-blockers, ACE/ARBs, Norpace, statins, antiplatelet agents (Aggranox, Effient, Plavix or ASA)?

* 15. Ranexa (extended release) 500mg or 1000mg BID dosing is used to treat which of the following diagnosis: CHF, AF, angina, SVT, mitral valve prolapse?

* 16. Pradaxa (dabigatran) 150mg BID (if CrCl < 30, then 75mg BID) is used to treat AF and is an alternative for: amiodarone, Coreg, warfarin, or digoxin.

* 17. The "statin" drugs (ex: Mevacor, Lipitor, Pravachol, Zocor, etc) are prescribed to treat coronary artery disease, angina, and status post myocardial infarction and are effective irregardless of the cholesterol levels. (True / False)
* 18. Heart blocks are symptomatic because the heart beats so slowly or irregular that cardiac output is decreased. The symptoms may include lightheadedness or passing out (syncope), weakness, shortness of breath, and chest pain. (True / False)

* 19. Which if the following is NOT a loop diuretic, but still treats edema from CHF: furosemide, Edecrin, metolazone, Bumex, or Demadex. (May be dosed only a few times per week and also tends to require a much higher counter dose of potassium.)

* 20. The combination of isosorbide and hydralazine is an alternative treatment for: CAD, ASCVD, CABG, CHF, or AF?