## Section 1 - Physical Exam

Preview | Review

- True or false? Pulsus paradoxus can be seen in cardiac tamponade.
  - o True
  - Pulsus paradoxus is present with:
    - Cardiac tamponade (especially)
    - Constrictive pericarditis
    - Asthma
    - Tension pneumothorax
- What is pulsus bisferiens? What does it indicate?
  - o 2 systolic peaks of the aortic pulse per cardiac cycle
  - Seen w/ aortic regurgitation (with or without stenosis) & hypertrophic cardiomyopathy
- What does pulsus alternans indicate?
  - Varying pulse pressure w/ a regular pulse rate
  - Seen w/ severely depressed systolic function of any cause that leads to decreased stroke volume
- True or false? Sustained handgrip increases the murmur of mitral valve prolapse but decreases the murmur of HCM.
  - o False, it delays the murmur of MVP by increasing left ventricular volume
- When is a persistently split S2 heard?
  - o A persistently split S2 varies w/ respiration but does not disappear on expiration.
  - o Pulmonic stenosis
  - Acute pulmonary embolism
  - o Ectopic or pacemaker beats originating in the left ventricle
  - Pulmonary hypertension
  - Right bundle branch block (RBBB)
  - Mitral regurgitation
- What causes a paradoxically split S2?

0

In which patient population is an S3 usually pathologic?

0

• When are large v waves seen on the right side (jugular vein)? Left side (PA catheter tracing)?

0

• When is rapid x and y descent seen?

0

• When is the y descent absent?

0

• When are large, right-sided a waves seen?

0

• When are cannon a waves seen?

0

When does a slow y descent occur?

0

• When are large, left-sided a waves seen?

0

# Section 2 - Chest X-Rays

Preview | Review

- On a lateral view CXR, extension of the heart border posteriorly and inferiorly indicated enlargement of which ventricle?
- On lateral view CXR, extension of the cardiac shadow of the lower part of the anterior clear space behind the sternum indicates enlargement of which ventricle?

## Section 3 - Procedures and Labs

Preview | Review

- Name the anatomic structures and conditions that are evaluated with a TEE.
- What are the absolute indications for terminating an ETT?
- When are stress imaging studies done instead of an ETT?
- Which stress imaging tests are used in patients with LBBB? With paced ventricular rhythm?
- When are exercise stress echo and MPI indicated instead of ETT?
- Which patients may benefit from CPX?
- When is a PCWP increased?
- At which PCWP do you expect to see frank pulmonary edema?
- In which condition is diastolic pressure equal in all 4 chambers?
- Name 1 indication for endomyocardial biopsy.

# Section 4 - Hypertension

Preview | Review

- In which patients should you suspect secondary hypertension?
- True or false? A systolic abdominal bruit without a diastolic bruit suggests renal vascular hypertension.
- What disorder should you think of if a patient presents with hypertension, hypokalemia, and low renin?

## Section 5 - Cardiac Medications

Preview | Review

- True or false? Digoxin prolongs survival.
- Which medication has been shown to prolong survival both post-MI and for patients with heart failure?
- With which medication should a nitrate be paired to improve survival in heat failure patients?

#### Section 6 - Angina

- What is the most common cause of acute coronary syndrome?
- What is the cause of Prinzmetal angina?
- What does transient ST-segment elevation suggest on an exercise ECG stress test?
- What are causes of resting ST-segment elevation?
- Explain the similarities and differences between hibernating myocardium, reperfusion injury, and stunned myocardium.

- What are the main drugs used to treat angina?
- Which patients might benefit from Ranolazine?
- Which antianginal drugs decrease myocardial oxygen demand?
- Which antianginal drugs decreased afterload?
- Which antianginal drugs decreased preload?
- Which antianginal drug do you avoid in patients with RV infarct? Why?
- Why should you determine the probability of CAD in a person with intermittent chest pain?
- What is the most important test for risk stratification in patients with stable ischemic heart disease?
- For which patients with chronic stable angina do you do an echocardiogram? Why?
- A patient undergoing a workup for chronic stable angina is determined to be at high risk for death. What is the next step?
- What is the goal for blood pressure management in a patient with stable ischemic heart disease?
- Would you recommend aspirin in a healthy woman < 65 years of age for primary prevention of MI?

# Section 7 - Acute Coronary Syndrome

#### Preview | Review

- What are the 2 major categories of ACS?
- Name 1 group of patients that is more likely to present with MI without chest pain.
- How are troponin I and T used? How long do they stay elevated after an MI?
- What are the prehospital guidelines for chest pain?
- What are the major things you should do in early risk stratification of a patient who presents with ACS in the emergency department?
- Which anti-ishemic measures are done initially for all patients with ACS?
- Which patients should receive a platelet GP IIb/IIIa inhibitor?
- Of those with ACS, which group gets considered for fibrinolytic therapy and which group definitely does not?
- Which NSTE-ACS patients should be considered for an early invasive strategy?
- Which reperfusion therapies are appropriate in patients with STEMI or new LBBB? Who gets what?
- What are the absolute and relative contraindications to fibrinolytic therapy?
- How does management of RV infarction differ from LV infarction?
- Which patients with tachyarrhythmias after an MI get DC cardioversion?
- What are the medical options for hemodynamically stable MI patients with VT?
- What time frame after MI do the major mechanical complications tend to occur? How do they present? What is the best test to diagnose such a complication?
- When should a patient with STEMI be referred for an ICD?

## Section 8 - Coronary Artery Disease

- What are the primary risk factors for CAD?
- Which lab tests should general lipid screening include?
- In which situations are LDL receptors down-regulated? Up-regulated?

- Which familial dyslipidemia is the most common? Which lipoproteins are elevated?
- Which lipid test result suggests the need to work up familial hypoalphalipoproteinemia?
- What is the primary endpoint of lipid screening done for primary prevention of ASCVD?
- Per ACC/AHA ASCVD guidelines, name the 4 statin benefit groups.
- How is a high-intensity statin defined? A moderate-intensity statin?
- Which fats are the "good" farts? The "bad" fats?
- Which class of drugs is recommended 1st line to reduce LDL?
- What are the major side effects of statins?
- What are the side effects of bile resins?
- What is the main action of the fibrate drugs?
- What are relative contraindications to niacin?
- Which lifestyle activities increase HDL? Lower HDL?
- What happens to the lipid panel in ACS?
- Which patient groups definitely should get CABG?
- Which patient groups could get PCI or CABG?
- In 3-vessel disease, what is the benefit of CABG -- survival, symptoms, or both?
- Why is dual antiplatelet therapy so important after stent placement?

## Section 9 - Peripheral Arterial Disease

Preview | Review

- What are the causes of arteriosclerotic PAD?
- What is Buerger disease?
- What is the difference between claudication and pseudoclaudication?
- What is the first test to establish the diagnosis of lower extremity PAD? What result is considered abnormal?
- Which antiplatelet therapy is recommended for patients with PAD?

# Section 10 - Carotid Artery Disease

Preview | Review

- Which of these adverse cardiovascular events is most likely to occur in a patient with atherosclerotic disease of the carotid artery: MI, stroke, or TIA?
- When is carotid endarterectomy indicated?
- How might spontaneous dissection of the internal carotid artery present clinically? What is its prognosis?

## Section 11 - Cerebral Embolic Disease

Preview | Review

• What is the most common cause of cerebral embolic events?

#### Section 12 - Aortic Disease

Preview | Review

- What are the procedures of choice for diagnosing a dissecting aortic aneurysm?
- At what size is surgery indicated for a thoracic aortic aneurysm?
- At what size is surgery indicated for an abdominal aortic aneurysm?

#### Section 13 - Valvular Heart Disease

- With which common clinical symptoms do patients with aortic valve stenosis present?
  - o Heart failure, angina, and syncope with exercise.
- When should valve replacement occur for aortic valve stenosis?
  - Do aortic valve replacement (AVR) for all symptomatic (Stage D) patients and all with asymptomatic severe AS (Stage C) i.e., with a gradient > 40 mmHg, velocity > 4.0 m/s, or valve area < or = 1 cm<sup>2</sup>. It is also indicated in patients with severe asymptomatic AS who have an EF < 50% or those who need CABG.</li>
  - Transcatheter aortic valve replacement (TAVR) is recommended for patients with prohibitive risk for surgical AVR and. Predicted post-TAVR survival > 12 months.
- Name 2 conditions that cause chronic aortic regurgitation.
  - Valve deformity (e.g., bicuspid valve, rheumatic fever, endocarditis, or degenerative valve disease)
  - Abnormal aortic root (e.g., dilation seen in Marfan syndrome, senile aortic disease, giant cell arteritis, relapsing polychondritis, or syphilis)
- What is the usual treatment for acute aortic regurgitation with associated heart failure?
  - Immediate AVR
- When the mitral stenosis is more severe, is the S2-OS interval smaller or larger?
  - Smaller
  - The time interval between the 2nd heart sound (S2) and the OS (the S2-OS interval) is inversely related to the severity of the MS: The more severe the MS, the higher the left atrial (LA) pressure, and thus the earlier the mitral valve is forced open in diastole, the smaller the S2-OS interval.
- Which type of murmur occurs in mitral stenosis?
  - S1 sometimes accentuated/enhanced/snapping
  - Opening snap
  - o Diastolic rumble
- Hemoptysis is seen with which mitral lesion?
  - Mitral stenosis
- Describe the murmur sometimes heart with MVP. Does that murmur's intensity decrease or increase with standing? With Valsalva maneuver?
- Carcinoid usually results in which type of tricuspid murmur?
- On physical exam, in patients with tricuspid regurgitation, what large waves are noted on the jugular waveform?
- True or false? Pulmonic stenosis is virtually always acquired.
- Ebstein anomaly is occasionally seen with which structural and electrical abnormalities?
- Are blood cultures more frequently positive with right-sided or left-sided endocarditis?
- Which type of ASD requires antibiotic prophylaxis before a dental procedure?
- Which of these require antibiotic prophylaxis: previous CABG? VSD? Mitral valve prolapse without murmur? Mitral valve prolapse with murmur? Prosthetic valve? Are your answers based on the ACC/AHA 2014 guidelines update?
- Following acute rheumatic fever, how many years on average does it take for valvular dysfunction to occur, if it does?
- What are the major prognostic factors after valve surgery?
- Describe the abnormal heart sounds found in AS, chronic AR, and MS.

#### Section 14 - Arrhythmias

Tachyarrhythmias Preview/Review

- What is the treatment sequence for atrial flutter?
  - Determine whether patient is asymptomatic vs. symptomatic first
  - o Asymptomatic/hemodynamically stable
    - Nonemergent cardioversion w/ antiarrhythmic drugs
      - 1st line IV ibutilide
        - Can cause QT prolongation (8%) & torsades de pointes
        - Make sure K+ and Mg2+ levels are normal prior to administration
      - Procainamide
      - Flecainide
      - Propafenone
  - o Symptomatic/hemodynamically unstable
    - Synchronized direct current (DC) cardioversion
    - Always shock if the patient is hemodynamically compromised
    - Do not continue DC cardioversion if the patient repeatedly reverts back to atrial flutter
  - o In patient's w/ atrial flutter + preexcitation syndrome (Wolff-Parkinson-White syndrome [WPW]), avoid: digoxin, calcium channel blockers, and beta-blockers
- What procedure can cure the most common types of atrial flutter with 85-95% success rate?
  - Radiofrequency ablation
- In which circumstance is immediate DC cardioversion indicated for A-fib?
  - Hemodynamic instability (angina pectoris, MI, shock, pulmonary edema)
  - Ongoing myocardial ischemia
  - Symptomatic hypotension
  - o Angina
  - Heart failure
  - o WPW w/ RVR
- What can happen after DC cardioversion to the patient who has A-fib with a slow rate? What intervention prevents this complication?
  - o Asystole can occur after DC cardioversion
  - Inserting temporary pacemaker before DC cardioversion can prevent this complication
- According to the 2014 update to the ACC/AHA Practice Guidelines: Management of Patients with Atrial Fibrillation, what HR is an acceptable target for patients with A-fib and stable ventricular function? For others, how is strict control of heart rate defined?
  - The 2014 ACC/AHA Guidelines for the Management of Patients with Atrial Fibrillation state that a strict rate-control strategy (resting heart rate < 80 bpm) is reasonable for management of symptomatic A-fib.
  - However, a lenient rate-control strategy (resting heart rate < 110 bpm) may also be okay if symptoms are controlled and LV function is normal.
- Which medication should you use to prevent postoperative A-fib in patients undergoing cardiac surgery?

- Oral beta-blocker
- How is the CHA2DS2-VASc score calculated? At what score should you treat with warfarin/DOACs (unless contraindicated)?
  - CHA<sub>2</sub>DS<sub>2</sub>-VASc scoring system:
    - Congestive) HF during last year or EF < 35% (any history): 1 point
    - HTN (prior history): 1 point
    - Age ≥ 75 years: 2 points
    - DM:1 point
    - Prior stroke, TIA, or embolic event: 2 points
    - Vascular disease, known (e.g., CAD, PAD, aortic aneurysm): 1 point
    - Age 65–74: 1 point
    - Sex category = female: 1 point
  - Medications based on CHA<sub>2</sub>DS<sub>2</sub>-VASc:
    - 0 points = ASA alone
    - 1 point = oral anticoagulation or ASA
    - $\geq$  2 points = oral anticoagulation
- In which patient group is MAT found?
  - o Patients with pulmonary disease, theophylline use, very low K+ or Mg2+ levels
- What is the treatment for acute A-fib in WPW?
  - o Instead, treat acute A-fib in WPW with IV procainamide, ibutilide, or amiodarone.
  - Shock if there are any signs of hemodynamic deterioration in any WPW tachyarrhythmia; especially watch those with ventricular rate > 285 bpm because they are at greatest risk of V-fib.

## Ventricular Arrhythmias Preview/Review

- On an ECG, PVCs are often followed by what type of pause?
  - Compensatory pause
- VT is defined as  $\geq 3$  sequential PVCs occurring at what bpm?
  - o ≥ 100 bpm
- List the ECG criteria consistent with VT.
  - AV dissociation
  - Fusion and capture beats
  - Northwest axis (between -90° and +/- 180°)
  - Positive or negative concordance in precordial leads (Recall concordance is when QRS complexes in all 6 precordial leads are monophasic with the same polarity)
  - Absence of rS complex in all precordial leads
  - o If rS is present, r to S time > 100 msec
  - o QRS width of > 140 msec with a RBBB
  - o QRS width of > 160 msec with a LBBB
- With which type of tachycardia should you never use verapamil?
  - Any wide-complex tachycardias in the emergency setting
- ICDs are recommended for primary prevention in which situations with ischemic and nonischemic cardiomyopathy?
  - Patients with LVEF ≤ 35% due to prior MI who are at least 40 days post-MI and are in the NYHA functional Class II or III; also, LVEF < 30% and in the NYHA functional Class I

- Patients with nonischemic dilated cardiomyopathy (DCM) who have an LVEF ≤ 35% and who are in the NYHA functional Class II or III
- Which antiarrhythmic drugs can cause TdP by prolonging the QT interval?
- What is the treatment for torsades de pointes?

## Section 15 - Syncope

Preview | Review

- What is the most common cause of syncope?
- Explain how you approach the diagnostic workup in a patient with probable neurocardiogenic (vasovagal) syncope.
- What are the tests used to work up high-risk patients with syncope?

# Section 16 - Cardiomyopathies

Preview | Review

- What are the risk factors for sudden death in patients with HCM?
- WHat are the 3 main medications used in the treatment of HCM?
- WHich auscultatory finding is pathognomonic for constrictive pericarditis?
- What are some causes of restrictive cardiomyopathy?
- List some of the etiologies of DCM.

#### Section 17 - Heart Failure

Preview/Review

- In the 2013 ACC/AHA classification, what are the 2 major types of heart failure?
  - o Heart failure with reduced ejection fraction (HFrEF): EF ≤ 40%, systolic HF
  - Heart failure with preserved ejection fraction (HFpEF): EF > 50%, including diastolic HF
- Define Stage A through Stage D heart failure (ACC/AHA classification). What are the goals of therapy for each of these stages?
  - Stage A
    - Stage A HF patients are at high risk for heart failure but have no structural heart disease or symptoms of HF. This stage includes patients with any of the following: hypertension (HTN), atherosclerotic disease, diabetes, obesity, metabolic syndrome, use of cardiotoxins (such as anthracycline), family history of cardiomyopathy
    - Just having HTN means you have Stage A heart failure!
    - Goals for Stage A therapy
      - Treat the disorder (e.g., HTN, atherosclerotic disease)
      - Control/avoid other risk factors that can lead to or contribute to HF, such as hyperlipidemia, obesity, diabetes mellitus, tobacco use, and known cardiotoxic agents (e.g., excess alcohol/illicit drug use).

        Regular physical activity is recommended in all HF patients.
  - Stage B
    - Stage B HF patients have structural heart disease but without signs or symptoms of heart failure. This stage includes patients who have a history of a previous MI, those with LV remodeling from left ventricular hypertrophy (LVH) or low LVEF, and those with asymptomatic valvular heart disease.

- Goals of Stage B therapy
  - Prevent HF symptoms
  - Prevent further cardiac remodeling

#### Stage C

- Stage C HF patients have structural heart disease with prior or current symptoms of HF.
- These are patients with structural heart disease as described above in Stage B, and who additionally have signs and symptoms of HF (e.g., dyspnea, fatigue, and decreased exercise tolerance).
- Goals for Stage C therapy
  - Symptom control
  - Patient education
  - Improved health-related quality of life
  - Prevention of hospitalization and mortality

#### Stage D

- Stage D HF patients have marked symptoms at rest and frequent hospitalizations despite maximal medical therapy.
- Goals for Stage D therapy
  - Control symptoms
  - Improve health-related quality of life
  - Reduce hospital readmissions
  - Establish the patient's end-of-life goals
- What is the sequence of drugs used to treat HF based on ACC/AHA stages?
  - Stage A
    - Stage A drugs include ACEIs/ARBs/statins in appropriate patients.
  - Stage B
    - Stage B drugs are ACEIs/ARBs, beta-blockers, and statins if there is a history of MI/ACS.
    - Use ICD if indicated and revascularization or vascular surgery as appropriate.
  - Stage C
    - RAAS inhibitors (ACEI or ARB or combination ARB + neprilysin inhibitor) for all patients with chronic HFrEF to reduce morbidity and mortality
    - Combination ARB + neprilysin inhibitor to replace ACEI or ARB monotherapy in patients with chronic symptomatic HFrEF, to further reduce morbidity and mortality
    - Statins and beta-blockers as used in Stage B (i.e., if MI/ACS) and ACEIs/ARBs as used in Stage A
    - NYHA II-IV patients
      - Loop diuretics for all volume-overloaded NYHA II–IV patients
      - Aldosterone antagonist for NYHA II-IV patients (estimated GFR > 30 mL/min and K<sup>+</sup> < 5 mEq/L [5.00 mmol/L])</li>
    - NYHA III-IV
      - Hydralazine/isosorbide dinitrate for symptomatic African American NYHA III-IV patients after optimal treatment with ACEI/ARB and beta-blockers

- Use ICD and/or cardiac resynchronization therapy (CRT) if indicated and revascularization or vascular surgery as appropriate.
- o Stage D
  - Options for Stage D patients include consideration of "extraordinary measures"—including heart transplant, chronic inotropes, temporary or permanent mechanical circulatory support (ventricular assist devices), experimental surgery or experimental drugs—and include consideration of palliative care, hospice, and ICD deactivation.
  - Until definitive therapy (coronary revascularization, mechanical circulatory support, or heart transplantation) is performed or the acute precipitating problem resolves, patients with cardiogenic shock should receive temporary IV inotropic support to maintain systemic perfusion and preserve end-organ performance.
- What are the most common causes of low-output HF?
  - Coronary artery disease (40–60%)
  - Dilated cardiomyopathy (30%)
  - Valvular disease (15%)
  - Hypertension (10%)
- Which factors result in a poor prognosis in HF?
  - Cardiac
    - Lower ejection fraction
    - Elevated troponin
    - High brain natriuretic peptide (BNP)
    - Increased width of QRS
    - Persistent sinus tachycardia
    - Poor functional capacity (NYHA III and IV)
  - Other
    - Hyponatremia
    - CKD
    - Anemia
    - High norepinephrine and catecholamine levels
- What is the sequence of events that worsens HF?
  - Low CO -> low renal perfusion -> high renin -> high angiotensin I -> high angiotensin II -> high aldosterone -> retention of Na+ and water -> high filling pressure (moving the Starling curve to the right) -> exacerbation of HF
- When are beta-blockers started in the treatment of HF?
  - Previously, it was taught that starting these drugs while patients are decompensated is contraindicated. The 2013 ACCF/AHA Guidelines for the Management of Heart Failure recommend initiation of beta-blockage at any stage of heart failure, after an ACEI has been initiated.
  - Carvedilol (nonselective beta blocker w/ alpha blocker effect, ~65% improvement in mortality), extended-release metoprolol succinate (beta-one selective beta blocker, ~66% improvement in mortality), and bisoprolol (beta-one selective beta blocker, ~35% improvement in mortality)
- With which type of HF do aldosterone antagonists prolong survival?
  - HFrEF

- Spironolactone
  - Decreased mortality by 30% at 24 months in patients with Class IV HF or Class III with a history of Class IV in the previous 6 months (1999 RALES trial)
- Eplerenone
  - Decreased mortality by 15% at 16 months in patients with recent MI, EF < 40%, and evidence of HF or diabetes mellitus (2003 EPHESUS trial)
- NYHA Class II patients benefit from aldosterone antagonists only if they have prior HF hospitalizations and elevated plasma natriuretic peptides
- True or false? Digoxin can be beneficial in HFrEF patients to decrease hospitalizations for HF.
  - True
- In which population is hydralazine + isosorbide dinitrate beneficial?
  - o African Americans with NYHA III-IV HFrEF
  - Patients with current or prior symptomatic HFrEF who cannot be given ACEIs/ARBs (e.g., drug intolerance, hypotension, renal insufficiency)
- True or false? Ivabradine can be beneficial in stable HF patients to decrease hospitalizations for HF.
  - True
- Which patients with chronic HF should receive anticoagulation (warfarin, dabigatran, apixaban, or rivaroxaban)?
  - Patients with chronic HF and permanent, persistent, or paroxysmal atrial fibrillation plus an additional risk factor for cardioembolic stroke (Hx HTN, DM, previous stroke/TIA, or ≥ 75 years of age
  - HF patients with a cardioembolic source (history of systemic or pulmonary embolism, or a mobile left ventricular thrombus)
- CRT is indicated for which HF patients?
  - Patients with EF ≤ 35%, sinus rhythm, LBBB with a QRS duration of ≥ 150 msec, and NYHA II, III, or ambulatory with NYHA IV symptoms despite optimal medical therapy
- Know all drugs used for emergency treatment of severe HF!
  - Dobutamine
  - Milrinone
  - Dopamine
- What does dopamine do at low doses (< 2 μg/kg/min)? At doses of 2–5 μg/kg/min?</li>
- True or false? MCS is beneficial in selected patients with Stage D HFrEF in whom definitive management (cardiac transplantation) or cardiac recovery is anticipated or planned.

#### Section 18 - Pericardial Diseases

- What are some causes of nonconstrictive pericarditis? What ECG changes can you see?
- What are the 2 clinical hallmarks of constrictive pericarditis?
- How is BNP used to differentiate constrictive pericarditis from restrictive pericarditis?
- In which conditions are the measured diastolic pressures of all 4 chambers equal?

- What treatments can be helpful in recurrent pericarditis?
- Name the 3 hallmarks of cardiac tamponade.

## Section 19 - Congenital Heart Disease

Preview | Review

- What is the most common congenital heart abnormality found initially in adults?
- True or false? Ostium secundum ASD often has right axis deviation and/or RBBB on ECG.
- When should surgery be performed for a secundum ASD?
- Which type of cyanosis would you expect to see in someone with a PDA?
- What is the most common congenital heart defect in children?
- What are the 2 most common causes of sudden cardiac death in an exercising young person? What 3rd cause do you consider in young women?

# Section 20 - Pulmonary Heart Disease

Preview | Review

• What is the only effective treatment for Eisenmenger syndrome?

## Section 21 - Pregnancy and the Heart

Preview | Review

• What are 2 cardiac-related absolute contraindications to pregnancy?

#### Section 22 - The Electrocardiogram

- Does a positive deflection on ECG tracing indicate the depolarization wave is moving toward or away from the lead?
  - A lead tracing is positive if the wave of depolarization spreads toward the positive pole of that lead, and it is negative if it spreads away from the positive pole.
  - The tracing is zero if the wave is maximal at a 90 degree angle to it.
  - Depolarization moving toward the lead causes a positive deflection (P wave and QRS), as does repolarization moving away from the lead (T wave).
  - The frontal leads (I, II, III, aVR, aVL, aVF) give inferior-superior-left-right information.
  - o The horizontal leads (v1-6) relay anterior-posterior-lateral information.
- Know how to very quickly determine the axis of an ECG. Brand Figure 13-22 into your brain!
  - A quick, fairly accurate method to determine axis is to look at I and aVF. If both are prominent, you can quickly tell in which quadrant the mean vector lies.
     Visualize the following:
    - Both (+) = normal
    - I (+) and aVF (-) suggest LAD
    - Both (-) = extreme right or left axis
    - I (-) and aVF (+) suggests RAD
- What are the causes of left axis deviation?
  - Left anterior hemiblock
    - Is a marker for CAD as are all fascicular blocks

- What are the causes of right axis deviation?
  - Normal finding in children and young adults
  - Left posterior hemiblock (LPHB)
  - o RVH
  - Acute or chronic RV overload syndromes (pulmonary hypertension/embolism and pulmonic stenosis)
- Does RAD always warrant additional workup in an adult?
  - Yes
- Describe one way to determine heart rate from a 12-lead ECG.
  - o Thin line/small squares 1 mm, 0.04 seconds or 40 msec
  - Thick line/large squares 5 mm, 0.2 seconds or 200 msec
  - A less accurate, but easier, method is to divide 300 by the number of big squares in the RR interval
  - A derivative of this is the method taught in Dale Dubin's book Rapid Interpretation of EKGs, in which you memorize 2 sets of triplicates: 300-150-100 and 75-60-50. These match to the heart rates corresponding to RR intervals of 1, 2, 3, 4, 5, and 6 big squares.
- Name the causes of prolonged QT intervals.
  - Congenital long QT syndromes
  - Medications:
    - Tricyclic overdose
    - Type Ia and III antiarrhythmics (e.g., quinidine, procainamide = Ia; e.g., amiodarone, sotalol = III)
    - Opioids, such as hydrocodone and methadone
    - Antipsychotics, including chlorpromazine, haloperidal, and thioridazine
  - Electrolyte abnormalities:
    - Hypocalcemia
    - Hypomagnesemia
    - Hypokalemia
  - Starvation
  - o CNS insult
  - Hypothermia
  - Liquid protein diet
- What are the P wave findings for right atrial hypertrophy? For left atrial hypertrophy?
  - Right atrial enlargement
    - With right atrial enlargement (aka right atrial hypertrophy), the right atrial (initial) portion of the P wave is widened and therefore overlaps onto the left atrial portion of the P wave
  - Left atrial enlargement
    - With left atrial enlargement (aka left atrial hypertrophy), the right side of the P wave is enlarged, resulting in a wide P wave with a shortened or absent PR interval (i.e., < 120 msec).
    - Other typical findings are a widened, notched P wave in II and an enlargement of the negative portion of the P wave in v1.
    - The most sensitive ECG finding for left atrial enlargement is a negative P wave in v1, with a duration of > 40 msec (1 small square). On the other hand,

the most specific ECG finding is a notched P wave (usually in II) with an interpeak distance of > 40 msec.

- What are the 5 causes of an R wave in lead V1?
  - o RBBB
  - o RVH
  - WPW pattern
  - Prior RV infarct
  - Duchenne muscular dystrophy (rare in adults)
- What does failure of R waves to progress across the precordial leads indicate?
  - Prior anterior MI
- When are peaked T waves seen?
  - Hyperkalemia
  - Acute MI (early stage)
  - o Intracerebral hemorrhage
  - Evolving post-MI in septal leads (v1-2)
- What do focal, flipped T waves signify?
  - May accompany:
    - Ischemia
    - RBBB, RVH, and elevated RV pressure (in v1-2)
    - LVH (in v1-2)
    - LBBB (in lateral leads I, aVL, v6)
    - LVH with strain (in precordial leads)
- U waves indicate a predisposition to which serious condition?
  - U wave occurs just after the T wave, commonly small and is best seen in v2-3, usually < 1 mm, rounded deflection in the same direction as the T wave</li>
  - o If the U wave is prominent, there is an increased tendency for torsades de pointes
- What are the common causes of U waves?
  - Hypokalemia
  - Bradycardia
  - Digoxin
  - Amiodarone
- What is the significance of an inverted U wave?
  - Ischemia
    - 60% of patients with an anterior MI
    - 30% of patients with an inferior MI
    - 30% of angina patients
  - HTN
  - AV valve disease
  - o RVH
- Name 3 main causes of ST-segment elevation.
  - Acute MI
  - o Vasospastic (i.e., Prinzmetal) angina
  - Pericarditis
  - Early repolarization variant
  - o Intracerebral hemorrhage
  - Stress (i.e., takotsubo) cardiomyopathy

- Hypertrophic cardiomyopathy
- o LVH
- LBBB
- o Cocaine abuse
- Myocarditis
- o Hypothermia (J, aka, Osborn waves)
- What are the causes of ST-segment depression?

0

What are the ECG criteria for LVH? RVH?

0

• What ECG changes would you expect to see in the presence of a large pulmonary embolism causing acute cor pulmonale?

C

What do you see on an ECG in LBBB? RBBB?

0

- What is so serious about a recent MI and the development of bifascicular block?
- What is the difference between an ectopic beat and an escape beat?
- What are the ECG findings with A-fib?

0

What are the ECG findings with MAT?

0

What are the ECG findings with PVC?

0

• Describe the sequence of ECG changes with the different phases of an Ml.

0

What ECG changes occur with septal MI? Anterior? Lateral? Know all these!

• Which conditions can cause diffuse, inverted T waves?

0

Which conditions can cause a prolonged QT?

0

AV node dysfunction is seen in which type of MI? What about bifascicular block?

Which conditions cause resting ST elevation?

0