

ECG Practice Strips Discussion part 1:

The first 20 strips are for teaching various abnormalities of the morphology of the waves of the ECG. Strips 21 and following are for teaching some abnormalities of the cardiac rhythm. Please note that you are not required to know the QRS axis in degrees, only whether the QRS axis is normal, left axis deviation, right axis deviation, or northwest axis, or indeterminate axis.

1.

Rate: atrial 65 ventricular 65

Axis: QRS +60 **Axis: P** +60 **Axis: T** +60 **QRS AXIS IS NORMAL**

PR interval: 0.16 **QRS interval:** 0.10 **QT interval** 0.40

Rhythm: sinus rhythm (There is a P wave in front of every QRS, and the P waves come at regular intervals, and the P waves are uniform in shape and normal in direction, and the rate is between 60 and 100. All these must be true for normal sinus rhythm. Also the distance from the P to the QRS is normal, eliminating first degree AV block, and the P wave is normal in shape and direction, eliminating left or right atrial abnormality.)

Hypertrophy: The voltage is not high enough in any of the leads to be LVH, and there is no LAE or secondary repolarization abnormality, and there are no criteria for RVH or RAE present

Infarction: No pathologic Q waves along with normal precordial R wave progression indicates no old MI, and the ST segment being normal indicates no injury, and the T waves being normal indicate no ischemia. Recall, the T waves should be upright in the same leads that the QRS is upright in the limb leads (frontal plane), and the T waves should be upright in all the precordial leads (transverse plane, V1-V6), except V1 which can be any direction (upright, inverted, or isoelectric)

Conduction abnormality: none. There are three large categories of conduction delay. One is conduction delay in the atria (intra-atrial conduction delay), a second is in the AV node (AV conduction delay), and the third is in the ventricles (intraventricular conduction delay). The QRS interval (QRS duration) is normal, excluding bundle branch block or WPW or non-sinus beat or IVCD. The PR interval is normal and constant, excluding AV conduction abnormality, and the P wave duration is normal, excluding intra-atrial conduction delay.

Diagnoses:

1. Normal sinus rhythm
2. Normal ECG

Comment: This is a normal ECG. I would be happy to have this ECG as my own. It is nice to start out with an "easy" one, but actually to make the diagnosis of a normal ECG, it is necessary to exclude all abnormalities.

2.

Rate: atrial 90 ventricular 90

Axis QRS +0 degrees **Axis P** +45 to 60 degrees **Axis T** +135 to 150 degrees **QRS AXIS IS NORMAL**

PR interval 0.18 sec **QRS interval** 0.08 sec **QT interval** 0.32 sec

Rhythm: sinus rhythm

Hypertrophy: There are no criteria for LVH or RVH, but the P wave shows atrial abnormality with the P wave being abnormally deep and wide in its terminal force (actually the entire P wave is inverted) in lead V1. This is the best criterion for left atrial enlargement, and so that is present. Another criterion for LAE is a notch in the P wave with 0.04 seconds between the peaks surrounding the notch, often seen in lead II and called "P mitrale" because of its association with mitral stenosis.

Infarction There is a small Q wave in lead aVF and a deep Q in III, with the QRS axis being fairly horizontal, so the Q wave is over 25% of the height of the R wave in aVF, and this is diagnostic for prior anterior wall MI. The ST segment is not elevated, and the T wave is upright, so this infarction is old. There are no other pathologic Q waves, and the precordial R wave progression is normal, so there are no other old infarctions present. Repolarization is abnormal. This requires explanation, since it is not secondary to a problem of hypertrophy or conduction

abnormality. In this case, there is ST segment depression in leads I and aVL and V5 and V6 and just a little in V4 too. There is also T wave inversion in I and aVL and V5-V6, and these abnormalities (lateral ST depression and T inversion) are seen in LVH and LBBB, but in this case are more likely due to ischemia. Recall that ischemic T waves are classically symmetric, and these look fairly symmetric.

Conduction abnormality: None.

Diagnoses:

1. Normal sinus rhythm
2. Left atrial enlargement
3. Old inferior MI
4. Lateral ischemia

Comment: Integrating these findings into a clinical context is important. The rate of 90, although normal, is somewhat concerning. The left atrial enlargement raises the possibility of elevated left atrial pressure, and a rate of 90 could indicate mild CHF. A prior inferior wall MI is not usually a cause for CHF, but there are also some ischemic changes (which aren't all that specific by themselves). All in all, this tracing is concerning.

3.

Rate: atrial 115 ventricular 115

Axis QRS +60 **Axis P??** **Axis T??** **QRS AXIS IS NORMAL**

PR interval 0.12 sec **QRS interval** 0.06 sec **QT interval??** sec

Rhythm: sinus tachycardia. Although the P wave is hard to see, it appears upright in II, and there seems to be a P wave preceding each QRS on the three lower rhythm strip leads, so the diagnosis of sinus tachycardia is confident. Notice that a rate of greater than 100 in the vital signs mandated a diagnosis.

Hypertrophy: Here is the section where the subject of voltage is addressed. By background, remember that before interpreting any ECG any time, it is necessary to notice the technical factor of the selection of voltage display by examining the standardization marking, usually found on the left side of the page before the tracing display begins. The standardization height is normally 10 mm and indicates 10 mm deflection is equivalent to 1 mV. This tracing is normal standard, and it is necessary to determine that before evaluating the voltage further. The voltage in this case is not high at all. To the contrary it is very low. The QRS complexes are less than 5 mm high in all 6 limb leads (R + S), and the QRS complexes are also less than 10 mm high (R + S) in all 6 chest leads. This meets the strict criteria for low voltage. Definitely there is no RVH or LVH or atrial abnormality noted here.

Infarction: Despite the smallness of the QRS complexes, there appear to be no pathologic Q waves, and the R wave progression is normal. The repolarization is not normal, because the T waves are all very flat. The T waves in the inferior leads are inverted, and that is abnormal, but they are very flat, and they are not specific for ischemia.

Conduction abnormality: none

Diagnoses:

1. Sinus tachycardia
2. Low voltage
3. Nonspecific T wave abnormality

Comment: Low voltage is a relatively uncommon diagnosis, and has some important clinical implications. This means that something has insulated the heart from the skin, like obesity, pulmonary disease, pericardial or pleural effusions, or that the ventricles have reduced electrical output from problems like amyloidosis, scleroderma, myxedema, or coronary artery disease (infarction reduces depolarizing cells). The nonspecific T wave abnormality does not add much to this interpretation. This ECG is concerning for a significant problem, and integration of these findings into the clinical situation is important. The presence of sinus tachycardia is also an important finding, indicating possible increased sympathetic tone, and this can indicate poorly compensated CHF or pulmonary disease, among other diagnoses.

4.

Rate: atrial - 65 ventricular - 65
Axis QRS: +15 **Axis P:** +30 **Axis T:** +150 **QRS AXIS IS NORMAL**
PR interval: 0.15 sec **QRS interval:** 0.09 sec **QT interval:** 0.42 sec
Rhythm: sinus rhythm

Hypertrophy: There is obvious large voltage, so LVH comes immediately to mind. The voltage criteria for LVH are clearly met in lead I. There is definite repolarization abnormality that is classic for LVH with the downsloping ST segment and inverted T waves in the lateral leads (I and aVL and V5-V6). The left atrium also is abnormal, with a P wave that has a terminal force that is over 0.04 sec in duration and 0.1 mV deep in V1. There is a notch in the P wave as well, seen in lead II and even better in V3. When there is LVH, infarctions should not generally be sought, because false positive and negative findings are common. This is a good time to review the point score system for LVH. This patient receives 3 points for voltage, 3 points for LAE, and 3 points for secondary repolarization abnormality not on digitalis preparation. All that is needed is 5 points, and there are at least 9 points here.

Infarction: none

Conduction abnormality: none

Diagnoses:

1. Normal sinus rhythm
2. Left atrial abnormality/enlargement
3. LVH with secondary repolarization abnormality

Comment: This is a very obvious LVH, and LVH is often an obvious finding. It is important to distinguish from LBBB, which often has similar axis and high voltage and has similar repolarization abnormality. The LBBB is different in that the QRS duration is prolonged to 0.12 sec or more, and there is usually a notch near the peak of the QRS, especially in lead I.

5.

Rate: atrial 70 ventricular 70
Axis QRS: -15 **Axis P:** +45 **Axis T:** +135 **QRS AXIS IS NORMAL**
PR interval: 0.18 sec **QRS interval:** 0.10 sec **QT interval:** 0.42 sec
Rhythm: sinus rhythm

Hypertrophy: Again, the very high QRS voltage, this time in the precordial leads, raises the immediate consideration of LVH, and all the criteria are here - the voltage criteria, the secondary repolarization abnormality, and the LAE best seen in V1. In this case, the QRS voltage is a little long, maybe really longer than 0.10, but the borderline degree of prolongation and the very impressive voltage mitigate for LVH. Increase in LV size or wall thickness gives an increase in LV mass, and the larger mass takes longer to depolarize. Notice that in the leads with prominent upright QRS complexes, the repolarization is opposite (=discordant), with ST depression and T inversion, and conversely in the leads with very negative QRS complexes (V1 and III), the repolarization is again discordant, with ST elevation and upright T wave.

Infarction: none

Conduction abnormality: none

Diagnoses:

1. Normal sinus rhythm
2. Left atrial abnormality/enlargement
3. LVH with secondary repolarization abnormality

Comment: This is a second example of clear-cut LVH for practice. The clinical correlation is most frequently hypertension or aortic valve stenosis, but other situations of congenital hypertrophy (hypertrophic cardiomyopathy) or volume or pressure load on the LV are considerations as well.

6.

Rate: atrial 80 ventricular 80
Axis QRS: +150 to 165 **Axis P:** +15 **Axis T:** 0 **QRS AXIS IS RIGHT AXIS DEVIATION**
PR interval: 0.17 sec **QRS interval:** 0.08 sec **QT interval:** 0.40 sec

Rhythm: sinus rhythm

Hypertrophy: There is no huge voltage for LVH as seen in the 2 prior examples. But the QRS axis is abnormal and shows right axis deviation, so RVH should be considered. This ECG is a classic example of RVH. The axis is rightward, and there are dominant positive forces in the right precordial leads V1 and V2, with the additional supportive finding of persistent S wave in V5 and V6, and these criteria are clearly diagnostic. The possible confusing differential diagnosis includes right bundle branch block, but this ECG has normal QRS duration so has no conduction abnormality. Additionally, the terminal rightward forces (S wave in I and V5-V6 and R wave in aVR) are not delayed, but are brief, and that portion of the QRS is not slurred or wide. The repolarization abnormality in RVH can show T wave inversion in the anterior precordial leads V1-V3 as in this example. The T waves in V5-V6 are abnormally flat. The P wave in V1 is suspicious for LAE, but there are no other criteria for LAE and the diagnosis of LAE should not be made here.

Infarction: none. The Q waves in III and aVF are narrow and not 25% of the height of the R wave. In this patient, they represent normal septal depolarization occurring directed superiorly. The poor precordial R wave progression is a result of the RVH and needs no further attention. The T wave abnormality does not represent ischemia from coronary obstruction but is a result of the RVH.

Conduction abnormality: none.

Diagnoses:

1. Normal sinus rhythm
2. Right ventricular hypertrophy

Comment: Generally if the ECG shows RVH, the RVH is severe, and so is usually a very significant clinical concern. The ECG is not sensitive for mild or moderate RVH because of the overriding dominance of the LV in forming the QRS. Not everything that gives right axis deviation is RVH. Some other causes include lateral wall MI, limb lead reversal (the treacherous technician!), and dextrocardia.

7.

Rate: atrial 70

ventricular 70

Axis QRS +165

Axis P +60

Axis T +60

QRS AXIS IS RIGHT AXIS DEVIATION

PR interval 0.18 sec

QRS interval 0.08 sec

QT interval 0.40 sec

Rhythm: sinus rhythm

Hypertrophy: Again, this is RVH. In this case the same considerations apply as in example 6 above. The axis is more rightward than the usual RVH in both these examples, often being only about +120. The very prominent anterior forces and the right axis deviation are the main diagnostic clues. In this example, there is again persistent S wave in V5-V6. No atrial abnormality is seen here. The repolarization abnormality again is seen with T inversion in V1-V2.

Infarction: none.

Conduction abnormality: none.

Diagnoses:

1. Normal sinus rhythm
2. Right ventricular hypertrophy

8.

Rate: atrial 95

ventricular 95

Axis QRS +75 to 90

Axis P +60 to 75

Axis T +75

QRS AXIS IS NORMAL

PR interval 0.14 sec

QRS interval 0.09 sec

QT interval 0.38 sec

Rhythm: sinus rhythm

Hypertrophy: The P wave is slightly positive in I and is flat or slightly negative in aVL, and is upright in aVF, so is +60 to 75 degrees, a little rightward. It doesn't meet other criteria for RAE, so a diagnosis of RAE should not be made. The QRS axis is rightward, but within the normal range. There is abnormal R wave dominance in V1 and V2, with almost no S wave in V1 and R greater than S in V2. The combination of dominant anterior forces and rightward axis and, additionally, relatively small R wave in V6 raise the suspicion of RVH and I think the diagnosis should be made. Another important cause of prominent anterior forces is true posterior myocardial infarction, and that diagnosis should be entertained here, but there is no other evidence of infarction, such as an accompanying inferior MI. The ST depression in this patient is very significant, even striking, especially in V3 and V4, and that also raises the consideration of subendocardial infarction, but the changes may also represent repolarization abnormality from the RVH. I favor RVH in this case, and would mention the possibility of subendocardial injury as "possible." There is also mild ST depression in leads I, II, III, and aVF, less dramatic than the anterior changes, but definitely abnormal.

Infarction: none, see the section on RVH above.

Conduction abnormality: none

Diagnoses:

1. Normal sinus rhythm
2. Right ventricular hypertrophy
3. Nonspecific ST abnormality, possible subendocardial injury

Comment: This ECG represents a somewhat more difficult level of analysis using more judgment, and is a good example of how the considerations should be weighed.

9.

Rate: atrial 70 ventricular 70

Axis QRS +30 **Axis P** +60 **Axis T** -165 (down in I and F, isoelectric in aVL)

QRS AXIS IS NORMAL

PR interval 0.14 sec **QRS interval** 0.09 sec **QT interval** 0.64 sec

Rhythm: sinus rhythm

Hypertrophy This ECG doesn't meet the criteria for low voltage, even though the voltage is very low in the limb leads, because the chest lead voltage is not low. There are no criteria for hypertrophy of atria or ventricles.

Infarction There are no pathologic Q waves and the R wave progression across the precordium is not wonderful but is within normal range. The really dramatic thing about this tracing is the prolongation of the QT interval. These T waves are strikingly abnormal with deep negativity in V2-V5 and very wide. This is a very concerning finding and mandates clinical correlation. Notice the T waves are abnormal in shape, in that the downstroke to the nadir is steep, and the return to baseline is gradual. This is the reverse of the usual T wave shape (the downstroke being more gradual and return to baseline being more steep).

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Prolonged QT interval

Comment: This dramatic QT prolongation can be associated with lethal ventricular arrhythmias, so should be evaluated promptly. A common cause is medication, especially some antiarrhythmic agents, but other medications, acute ischemic processes, CNS disorders, and congenital ion channel abnormalities can also cause QT prolongation.

10.

Rate: atrial 65 ventricular 65

Axis QRS +30 **Axis P** +45 **Axis T** +75 **QRS AXIS IS NORMAL**

PR interval 0.18 sec **QRS interval** 0.09 sec **QT interval** 0.40 sec

Rhythm: sinus rhythm with PVC's. Looking at the bottom 3 leads, the rhythm strip portion of the recording, notice the 6th QRS is early and in leads V1 and V5 has a different configuration from the regular QRS. This beat has no P wave preceding it. Likewise the 9th beat is a very similar

beat, seen without a preceding P wave. These beats are PVC's. They are a little unusual for PVC's in that they are not very wide, and the ST segment and T waves are not very bizarre. They are PVC's nonetheless. Since these 2 beats are identical to each other, the diagnosis would be unifocal PVC's. The other beats all have uniform P waves preceding them and are sinus.

Hypertrophy no atrial or ventricular hypertrophy

Infarction There is clear ST segment elevation seen in leads II, III, and aVF. This pattern of ST segment elevation indicates acute transmural injury of the inferior wall. This pattern indicates the need for immediate attempts at revascularization of the involved coronary artery, and immediate means "within minutes," because it is highly likely that muscle cells are dying before your eyes. In the setting of inferior transmural injury, the small Q in aVF and deeper Q in III indicate that infarction should be diagnosed. This patient is suffering an acute inferior wall MI and needs immediate attention, either emergency percutaneous coronary revascularization or intravenous thrombolysis, unless there are compelling contraindications.

Conduction abnormality no intra-atrial conduction abnormality, atrioventricular conduction abnormality or intraventricular conduction abnormality. In the setting of an inferior wall myocardial infarction, the most likely conduction abnormalities are atrioventricular conduction abnormalities, such as first degree AV block, or second degree AV block of the Wenckebach type, with progressive prolongation of the PR interval to the dropped beat, and usually a narrow QRS.

Diagnoses:

1. Normal sinus rhythm
2. Occasional unifocal PVC's
3. Acute inferior wall MI
4. Clinical correlation suggested
5. Suggest serial tracings

11.

Rate: atrial 75

ventricular 75

Axis QRS +60

Axis P +60

Axis T +75

QRS AXIS IS NORMAL

PR interval 0.16 sec **QRS interval** 0.09 sec **QT interval** 0.38 sec

Rhythm: sinus rhythm with one PAC. Notice that the 8th beat on the bottom 3 leads is early and has a subsequent pause. This QRS looks essentially identical to the other normal QRS's. Also notice that it is preceded by a P wave, and the P wave is early and is different from the normal P waves. This is a PAC.

Hypertrophy none

Infarction This is the same patient on the same day (actually about 30 minutes earlier) as the patient for example number 10 above. There is inferior wall dramatic ST elevation. The additional significant information added by this ECG is the very strange R wave progression. Notice that the R wave gets smaller from V1 to V6. The S wave also gets smaller. The reason for this is that the leads are not placed in the usual location, but on the opposite side of the chest, called right chest leads. These leads are indicated by the letter R at the end of the lead designation. So V1R is the left 4th interspace (the normal location for V2), V2R is the right 4th interspace (normal location for V1), V3R is further to the right, V4R is at the V4 location but on the right side, and similarly for V5 and V6. The most important of these right chest leads is V4R. This is the best lead for evaluation of right ventricular infarction. In the setting of an acute inferior wall MI, an ECG of the right chest leads should always be done looking for an accompanying right ventricular wall MI. The criterion for RV MI is ST elevation of at least 1 mm in V4R. In this example there is mild ST elevation in V4R but not a full 1 mm, so the criterion for RV MI is not met. This ST elevation in V4R is not a very sensitive test for RV MI, and the ST elevation is frequently brief in this lead, so the right chest leads should be taken quickly in the setting of an inferior wall MI.

Conduction abnormality: none

Diagnoses:

1. Normal sinus rhythm

2. One PAC
3. Acute inferior wall MI
4. Does not meet criterion for RV MI.

Comment: This tracing affords the opportunity to notice other details about the right chest leads. Essentially the only time that right chest leads are obtained is in evaluation of possible RV MI in the setting of an inferior MI. However, there are occasional patients who have dextrocardia. If a patient with dextrocardia has an ECG performed with leads in their normal location, the limb leads will show inversion of the P wave as well as usually the QRS and the T wave in lead I, and they also show the precordial pattern seen in this ECG with reversal of the normal R wave progression and with progressive smaller R and S waves.

12.

Rate: atrial 80 ventricular 80
Axis QRS +90 **Axis P** +75 to 90 **Axis T** -60 **QRS AXIS IS NORMAL**
PR interval 0. sec **QRS interval** 0. sec **QT interval** 0. sec

Rhythm: sinus rhythm with sinus arrhythmia. Notice that the initial beats in the rhythm leads are regular at a rate of about 80, but there is a gradual slowing of the sinus rate without any change in the shape or axis of the P wave. This is sinus arrhythmia. It is probably abnormal, because it is occurring in a pattern that is not typical for respiratory phasic sinus arrhythmia, the normal type of sinus arrhythmia seen in healthy people and athletic individuals with dominant parasympathetic tone.

Hypertrophy: none

Infarction: there is ST segment elevation seen in II, III, and aVF, with Q waves and T wave inversion. This indicates recent inferior wall myocardial infarction. Since Q waves have already appeared and the T waves have already inverted, it is likely too late to intervene, but the final determination for that is made on clinical grounds (when the symptoms began, the current clinical situation, etc.). The R wave in V1 and V2 is somewhat prominent but does not meet criteria for posterior wall MI. Whenever an inferior wall MI is seen on ECG, the anterior precordial leads should be examined for the presence of a posterior wall MI (prominent wide initial R wave in V1 and V2) and for the presence of a lateral MI (in leads V5-V6). If an inferior wall MI is associated with a posterior MI or a lateral MI, it is more likely to be the result of an occlusion of the left circumflex coronary artery. If there is no association with posterior or lateral MI, then it is more likely to be from an occlusion of the right coronary artery, but this rule is not totally reliable, just good for an initial guess. Notice in this tracing the similarity in appearance between the inferior leads and V6, with a slightly prominent Q in V6, very subtle ST elevation and T inversion, likely indicating mild lateral involvement, although not diagnostic. A good guess would be left circumflex as the culprit vessel.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Sinus arrhythmia
3. Recent inferior wall MI

13.

Rate: atrial 95 ventricular 95
Axis QRS -45 **Axis P** +60 **Axis T** -15 **QRS AXIS IS LEFT AXIS DEVIATION**
PR interval 0.16 sec **QRS interval** 0.09 sec **QT interval** 0.34 sec
Rhythm: sinus rhythm

Hypertrophy none

Infarction There are definite deep Q waves in II, III, and aVF indicating another example of inferior wall MI. The ST segment is normal, and the T waves are flat, not upright, so the infarction age is not determined, but is probably at least several weeks old. There is again prominent R wave in V1-V2 and a small Q wave in V6, raising the suspicion of posterior and lateral involvement, but again these are not diagnostic changes. Again a good guess would be left circumflex.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Inferior wall MI, age indeterminate.

Comment: The relatively rapid rate is concerning. Most patients who have suffered a prior MI should be taking beta-blocking agents, and will therefore have relatively slow heart rates. The rapid rate raises the suspicion of CHF (although there is no left atrial abnormality seen) or the administration of sympathomimetic agents, such as inhalants for bronchoconstriction.

14.

Rate: atrial 75 ventricular 75
Axis QRS 0 **Axis P** 0 **Axis T** 60 **QRS AXIS IS NORMAL**
PR interval 0.14 sec **QRS interval** 0.07 sec **QT interval** 0.36 sec
Rhythm: sinus rhythm

Hypertrophy the P wave is definitely abnormal. There is a prominent terminal negativity to the P in V1 indicating LAE, and this is confirmed by 2 other findings: a notch in the P in lead II with 0.04 seconds between the peaks, and a P wave in V3 that retains a biphasic shape. These are also criteria for LAE. In a tracing with some baseline artifact, as this one, looking at the rhythm strip form of lead II in the bottom half of the page gives a better idea of the shape of the P wave. The variability in the P wave shape from beat to beat is not a true change, but just baseline artifacts distorting the P wave. There is no ventricular hypertrophy.

Infarction The precordial R wave progression is very abnormal with Q waves in V2-V5, and with almost no R waves in any precordial lead. This absence of anterior forces indicates an anterior wall MI, and is a large MI. There is very mild ST elevation, and the T waves are largely upright in these leads, so the infarction is probably but not certainly old. There are minor ST and T wave abnormalities that do not bear mentioning as separate diagnostic statements – the big MI is bad enough. The association of the large MI and the LAE is not unusual, and indicates that the MI has likely provoked CHF. The normal heart rate in this setting most likely means either that the patient is taking a heart rate-lowering medication (beta-blocker or non-dihydropyridine calcium blocker), or that the CHF is well-compensated, or both.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Anterior wall MI, age indeterminate.
3. Left atrial abnormality

15.

Rate: atrial 107 ventricular 107
Axis QRS +90 **Axis P** +60 **Axis T** ?? **QRS AXIS IS NORMAL**
PR interval 0.18 sec **QRS interval** 0.09 sec **QT interval** ?? sec
Rhythm: sinus tachycardia

Hypertrophy The QRS axis is somewhat rightward, but not distinctly abnormal, and there are not prominent anterior forces, so the diagnosis of RVH cannot be made. There are no criteria for LVH. There is dramatic LAE seen in V1. The P wave in II is mildly peaked but not tall enough for the diagnosis of RAE.

Infarction The low voltage in the limb leads, especially I and aVL, makes the consideration of a lateral MI difficult, and the criteria are not made. This is supported by the absence of any Q wave in V6, although a prior lateral MI cannot be entirely ruled out. There is poor R wave progression across the precordial leads, with a Q in V2, and with an R wave in V3 less than 2 mm tall, and also a very small R in V4. The presence of Q in V1 and V2 meets criteria for septal wall MI. There is diffuse ST and T wave abnormality but these changes are nonspecific.

Conduction abnormality none

Diagnoses:

1. Sinus tachycardia
2. Left atrial abnormality
3. Prior septal MI, age indeterminate

Comment: the poor R progression can sometimes lead to the false positive diagnosis of septal MI, and the rightward axis and the P wave peaking in II raise the suspicion of pulmonary disease as the cause of these abnormalities, especially the poor R wave progression. These can't really be distinguished without further evaluation of the patient, beginning of course with a thoughtful history and physical examination. Either CHF or pulmonary problems could give the sinus tachycardia.

16.

Rate: atrial 95 ventricular 95

Axis QRS +120 **Axis P** +60 **Axis T** ?? **QRS AXIS IS RIGHT AXIS DEVIATION**

PR interval 0.16 sec **QRS interval** 0.09. sec **QT interval** ?? sec

Rhythm: sinus rhythm

Hypertrophy With the right axis deviation, RVH becomes a consideration, but the pattern in I is not right for RVH, because RVH has a RS pattern and this is a QR pattern. In this case, the diagnosis is not RVH. There are no prominent anterior forces. There is no LVH but again this example shows the V1 criterion for LAE.

Infarction There are pathologic Q waves in I and aVL, and in V2-V3, indicating prior localized anterior wall MI and lateral wall MI. There is mild ST elevation in the infarction leads that could indicate either that the MI was fairly recent or that there is an associated LV aneurysm (LV aneurysm is a cause of chronic ST elevation in some patients). A lateral wall MI is a cause for right axis deviation and explains the QRS axis of +120.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Left atrial enlargement
3. Lateral wall MI, age indeterminate
4. Anterior wall MI, age indeterminate

Comment: In this example, the combination of prominent MI criteria and LAE and relatively high sinus rate raise the concern for CHF.

17.

Rate: atrial 55 ventricular 55

Axis QRS +15 **Axis P** +0 **Axis T** -150 **QRS AXIS IS NORMAL**

PR interval 0.16 sec **QRS interval** 0.16 sec **QT interval** 0.48 sec

Rhythm: sinus bradycardia

Hypertrophy Although there is very significant voltage of the S wave in V2 raising the suspicion for LVH, the QRS is definitely too wide for LVH, and so a conduction abnormality must be present. No LVH or RVH can be diagnosed in the presence of a QRS >0.12 sec. The atrium shows no hypertrophy/enlargement.

Infarction Although there are pathologic Q waves in V1 and V2, the diagnosis of MI cannot be made in the presence of LBBB. Likewise, the presence of diffuse ST and T wave abnormalities is a part of the LBBB and does not indicate ischemia or injury.

Conduction abnormality There is a classic LBBB. Notice the notch near the peak of the R wave in I and also aVL and also V5 and V6. The entire QRS, initial forces, mid-QRS forces and terminal forces, is distorted by the LBBB. By contrast, in a RBBB only the terminal forces are distorted. A QRS duration of at least 0.12 sec is needed to satisfy duration criteria for either left or right BBB.

Diagnoses:

1. Sinus bradycardia
2. Left bundle branch block

18.

Rate: atrial 110 ventricular 110

Axis QRS -15 **Axis P** +45 **Axis T** +165 **QRS AXIS IS NORMAL**

PR interval 0.14 sec **QRS interval** 0.16 sec **QT interval** 0.40 sec

Rhythm: sinus tachycardia

Hypertrophy The voltage is prominent and would meet criteria for LVH but the diagnosis is excluded by the wide QRS. Atrial abnormalities could still be diagnosed, but none is present here. The negative P wave in V1 is not deep and wide enough to meet criteria for LAE

Infarction In the presence of LBBB, MI cannot be diagnosed, and the discordant repolarization is not indicative of ischemia or injury, but rather is a result of the conduction abnormality.

Conduction abnormality There is a classic LBBB, with the same characteristics of example 17. The ST segment and T wave are discordant very similar to the discordant repolarization of LVH, with ST depression and T inversion in the leads with a tall positive QRS (I, aVL, V5, V6), and conversely ST elevation and upright T wave in the leads with the deepest negative QRS (III, V1 to V3). Notice that the appearance is somewhat different in a patient with a faster heart rate. This tracing has very little isoelectric time, with a P wave following almost immediately behind the prior T wave. The situation of sinus tachycardia makes interpreting the ECG a little more of a challenge. Notice how significant the ST deviation is, especially in V5. Even ST segment deviation this great is still simply due to the LBBB. Remember, that if there is a LBBB, you should not look for hypertrophy or infarction – they cannot be diagnosed in the presence of LBBB.

Diagnoses:

1. Sinus tachycardia
2. Left bundle branch block.

19.

Rate: atrial 85 ventricular 85

Axis QRS +30 **Axis P** +30 **Axis T** +30 **QRS AXIS IS NORMAL**

PR interval 0.22 sec **QRS interval** 0.13 sec **QT interval** 0.38 sec

Rhythm: sinus rhythm

Hypertrophy There are prominent anterior forces raising the consideration of RVH but the wide QRS excludes RVH. There are no criteria for LVH or LAE or RAE.

Infarction There are no pathologic Q waves. In the presence of a RBBB, since the initial forces are not disturbed, pathologic Q waves can be diagnosed.

Conduction abnormality This example shows all three types of conduction abnormality. First, intra-atrial conduction abnormality: the P wave duration is too long. This is more of a fine point, but the P wave, especially in leads III and V1 and V2 lasts 0.13 seconds, and normally it should not last more than 0.10 seconds. Second, atrioventricular conduction abnormality: the PR interval is too long, indicating first degree AV block. And third, and most important in this example, an intraventricular conduction abnormality, a RBBB. Notice that the initial 0.06 seconds of the QRS is normal, with a sharp peak. Then there is an abrupt transition to a slurred terminal portion of the QRS, a wide S wave in I and aVL and V5 and V6, and a wide R-prime in V1 and aVR. These characteristics are classic for a RBBB, a "rightward anterior terminal conduction delay." The repolarization abnormality expected for a RBBB is that the ST segment is not affected, except slightly depressed in V1-V3, and the T wave is directed opposite the terminal delay, being upright in I and aVL and V5-V6, and inverted in V1-V2 and aVR. The pattern is variable in the inferior leads.

Diagnoses:

1. Normal sinus rhythm
2. First degree AV block
3. Right bundle branch block

20.

Rate: atrial 85 ventricular 85
Axis QRS +90 **Axis P** +60 **Axis T** +30 **QRS AXIS IS NORMAL**
PR interval 0.13 sec **QRS interval** 0.14 sec **QT interval** 0.38 sec
Rhythm: sinus rhythm

Hypertrophy No LVH, RVH, LAE, or RAE is present

Infarction There is significant ST deviation. The ST segment is elevated in V2-V6 and I and II. There are pathologic Q waves in V1-V3. This is an ongoing, or acute, anteroseptal MI, and needs immediate attention.

Conduction abnormality There is a RBBB present. There is a rightward anterior terminal conduction delay, and again a distinct transition between the normal conduction and the terminal delay, best seen in lead I. It is clear that the ST elevation is not a part of the RBBB. The RBBB generally does not cause ST segment deviation, and if it affects the ST segment at all, it results in ST depression in the anterior leads. This is an example of the possibility of reading ischemia and infarction in the presence of a RBBB (in contrast to the LBBB). The combination of an acute anteroseptal MI and a RBBB have a special significance. If the RBBB is known to be new with the acute MI, then the risk of complete heart block is significant and a temporary transvenous pacemaker is sometimes recommended.

Diagnoses:

1. Normal sinus rhythm
2. Acute anteroseptal MI
3. Right bundle branch block

ECG Practice Strips Discussion, Part 2:

Strips 21 and following are for teaching the abnormalities of the cardiac rhythm.

21.

Rate: atrial 65 ventricular 65
Axis QRS +15 **Axis P** +60 **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval 0.16 sec **QRS interval** 0.08 sec **QT interval** 0.40 sec

Rhythm: sinus rhythm. Notice that on the three lower leads, the rhythm leads, that all the P waves are similar to each other, they are uniform. The P wave axis is normal, indicating that the P originates in the sinus node. The rate is between 60 and 100, and the PR interval is normal, between 0.12 and 0.20 second (3 to 5 mm).

Hypertrophy none

Infarction none, but repolarization is not normal. The T waves are flat and even slightly inverted in I and V6, and should be clearly upright in these leads.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Nonspecific T wave abnormality

22.

Rate: atrial 45 ventricular 45

Axis QRS +30 **Axis P** +45 **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval 0.16 sec **QRS interval** 0.08 sec **QT interval** 0.48 sec

Rhythm: sinus bradycardia. The rhythm looks normal except the rate is just too slow. There is a P wave before each QRS and the P waves look uniform, and the P wave axis is normal, but the rate is less than 60. There are many possible causes, including aerobic conditioning, medications, and sick sinus syndrome to name a few. There may be a mild irregularity to the P-P interval in sinus bradycardia, a sinus arrhythmia, as seen here.

Hypertrophy none

Infarction none. The repolarization is abnormal, and nonspecific, so the diagnosis is nonspecific T wave abnormality.

Conduction abnormality none

Diagnoses:

1. Sinus bradycardia and sinus arrhythmia
2. Nonspecific T wave abnormality

23.

Rate: atrial 140 ventricular 140

Axis QRS 0 **Axis P** 0 **Axis T** 0 **QRS AXIS IS NORMAL**

PR interval 0.12 sec **QRS interval** 0.08 sec **QT interval** 0.28 sec

Rhythm: sinus tachycardia. This looks just like sinus rhythm, except the rate is greater than 100. Sinus arrhythmia is not present here, and is usually not present in the setting of sinus tachycardia.

Hypertrophy none

Infarction none. In this tracing, the T waves are too flat in the limb leads and in V6.

Conduction abnormality none

Diagnoses:

1. Sinus tachycardia
2. Nonspecific T wave abnormality

24.

Rate: atrial 80 ventricular 80

Axis QRS +15 **Axis P** +0 **Axis T** +60 **QRS AXIS IS NORMAL**

PR interval 0.16 sec **QRS interval** 0.08 sec **QT interval** 0.38 sec

Rhythm: This rhythm is not regular. There is an irregularity and there is a pattern of 2 close beats, then a pause. Looking at the pair of 2 close beats, the first is the sinus beat, and the second is an earlier beat, and the P wave of the second is different from the first, an early different P wave. The second is a PAC. So there is a pattern in which each sinus beat is followed by a PAC. This is called atrial bigeminy. In this case, the PAC is not dramatically

different in P wave shape or axis from the sinus beat, making the diagnosis more challenging, but still it is clear.

Hypertrophy none

Infarction none, the QRS and the repolarization are normal.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Frequent PAC's with atrial bigeminy

25.

Rate: atrial 84 ventricular 84

Axis QRS +45 **Axis P** +60 **Axis T** -120 (isoelectric in aVL) **QRS AXIS IS NORMAL**

PR interval 0.16 sec **QRS interval** 0.09 sec **QT interval** 0.36 sec

Rhythm: This rhythm is not regular. Remember that calculating the rate when the rhythm is not regular requires use of either the six second rule or the 10 second rule. For the 10 second rule, count all the QRS's on the rhythm strip at the bottom of the page (10 seconds of rhythm) and multiply by 6: $14 \times 6 = 84$. Analyzing the rhythm: There are 2 different P waves noted in the rhythm leads. The second QRS is preceded by a pause, then a P wave that is upright in II and looks like a sinus P wave in V1 as well. The next QRS is early and is preceded by an early P wave that is different from the sinus P wave. The next P is sinus, and the next, then another PAC, then 4 more sinus beats, then another PAC, then another sinus beat, then another PAC. These PAC's are much more easily recognized as different from the sinus beats because they are different in direction, very different in shape. So this is sinus rhythm with frequent PAC's. The PAC's look identical to each other, so they could be called unifocal PAC's, but this is more detail than we need.

Hypertrophy there is very prominent voltage and repolarization abnormality and LAE, so there is LVH. Notice that on the sinus beats, LAE can still be diagnosed.

Infarction no diagnostic criteria for infarction, and the repolarization abnormality is secondary to the LVH.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Frequent PAC's
3. LVH with secondary repolarization abnormality
4. LAE

26.

Rate: atrial 78 ventricular 78

Axis QRS +30 **Axis P** +60 **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval 0.14 sec **QRS interval** 0.09 sec **QT interval** 0.38 sec

Rhythm: The rhythm is mainly regular and sinus with basically uniform P waves and QRS's with just a few irregularities. The irregularities are the interesting part. There are 3 early beats, the 6th, the 11th, and the 15th. The 11th has a clearly distorted T wave of the preceding QRS, and that represents the P wave of a PAC. The 15th beat also has a preceding (early and different) P wave and is clearly a PAC. The QRS of the 15th beat is normal. The QRS of the 11th beat is not normal. But, since there is an early different P wave before it, it must be a PAC, and the explanation for the abnormal QRS is that there is aberrant ventricular conduction. Now since the abnormal QRS of the 11th beat is identical to the abnormal QRS of the 6th beat, that beat may also be a PAC with aberrant ventricular conduction. And looking closely at the T wave of the preceding beat, especially in lead aVL above, another early different P wave can be seen. Therefore there are 3 PAC's, two conducted aberrantly and one conducted normally. Remember that the essential element to a PAC is the presence of an early and different P wave. The QRS

following the PAC can be normal, aberrant, or even missing (a nonconducted PAC, and that would result in a pause).

Hypertrophy none

Infarction none, but repolarization is not quite normal, with the T waves too flat in I, and with minor ST depression in V5.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. PAC's, some aberrantly conducted
3. Nonspecific ST and T wave abnormality

27.

Rate: atrial ?? ventricular 150

Axis QRS +60 **Axis P** ?? **Axis T** +60 **QRS AXIS IS NORMAL**

PR interval ?? **QRS interval** 0.07 sec **QT interval** 0.26 sec

Rhythm: The QRS is narrow and normal looking, but there are no P waves to be seen anywhere. Nothing in front of the QRS, and nothing in the ST segment, and nothing in the T wave can be seen that looks like a P wave. That means that the P wave is either totally absent, or, more likely, it is buried right in the QRS complex. So this is a tachycardia, and it is very regular. This meets criteria for the nonspecific diagnosis of "supraventricular tachycardia", since it is a regular tachycardia and no P waves can be found. Statistically, the most likely diagnosis specifically is AV nodal reentrant tachycardia, of the typical variety. In this case there is a reentrant circuit that runs antegrade down one of the 2 AV nodal pathways, and returns retrograde up the other, and then back down the first, and back up the second again, and so forth. The 2 pathways are called the fast pathway, and the slow pathway, and in the typical type, the impulse goes down the slow pathway and up the fast pathway, and then the P wave happens during the QRS. In the atypical type, the impulse goes down the fast pathway, and up the slow pathway, resulting in a retrograde P wave (slightly upright in I, and inverted in II, III, and aVF) occurring long after the QRS.

Hypertrophy none

Infarction none

Conduction abnormality none

Diagnoses:

1. Supraventricular tachycardia

28.

Rate: atrial 156 ventricular 156

Axis QRS +45 **Axis P** ?? **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval variable **QRS interval** 0.07 sec **QT interval** ??

Rhythm: Calculating the rate must be done by the 6 or 10 second rule, since the rhythm is not regular. In analyzing a rhythm, a very important first step is to identify all the P waves and assess their origin. In this case, there are many P waves, and they are definitely not uniform. The first P wave in the rhythm strip is very flat, and is near the peak of the preceding T wave. The second P is deep and inverted near the beginning of the T wave of the second QRS. The third P wave looks normal, and has a normal PR interval. The fourth P wave looks similar in V1, but in lead II it is clearly different. So all four of the first P waves are different, and the rate is tachycardic. This meets criteria for multifocal atrial tachycardia ("MAT"), an irregular tachycardia with 3 or more different P wave morphologies. Notice that it is not possible to really find a sinus P wave with confidence. The P wave shape is unpredictable from one beat to the next. One important differential diagnostic possibility is atrial fibrillation. In atrial fibrillation, as in this tracing, the QRS complexes are irregularly irregular. The crucial difference between MAT and atrial fibrillation is that in atrial fibrillation there are not well-formed P waves, just an undulating baseline of varying size and shape, and with no instant of atrial silence. By contrast, in MAT, there are distinct P waves and there are periods of atrial silence (atrial diastole). So the judgment is whether there

are well-formed P waves or undulating baseline. There is really no assistance found in analyzing the QRS irregularity or pattern when trying to distinguish between these.

Hypertrophy none

Infarction none. The repolarization is abnormal with flat T waves and mild ST depression, nonspecific abnormalities

Conduction abnormality none

Diagnoses:

1. Multifocal atrial tachycardia
2. Nonspecific ST-T wave abnormality

29.

Rate: atrial 250 ventricular 60

Axis QRS +75 **Axis P** ?? **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval ?? **QRS interval** 0.14 sec **QT interval** ??

Rhythm: The atrial rate is much higher than the ventricular rate. Again, begin with the atrial activity. In this case, the atrial rate is 250 and the atrium is organized and regular in its action. From the beginning of the strip to the end, in the II rhythm strip, the atrial activity is predictable. The appearance in lead II is of a "sawtooth", a classic appearance for atrial flutter. Flutter waves are often best recognized in leads II, III, or aVF, and less commonly in V1. The slow ventricular rate indicates that the conduction through the AV node is slow, often this is by medication. The natural ventricular rate for a healthy AV node to conduct atrial flutter is at a conduction ratio of 2:1, or a ventricular rate in this case of 125. In atrial flutter, the key to the diagnosis is the shape and rate and regularity of the atrial activity. The ventricular response is secondary. The ventricle may respond with a regular 2:1 response, or a regular 4:1 response, or an alternating 2:1 and 4:1 response, and rarely even a 3:1 response. The ventricular response may also be variable and not predictable, so can produce an irregularly irregular pulse. In this example, the last half of the strip shows a regular ventricular response at 4:1, but in the beginning of the strip, the response is not quite regular. The 4th QRS is early and wide, probably a PVC.

Hypertrophy none

Infarction none. The repolarization is abnormal, but the significance of the abnormal repolarization is unclear in the presence of an intraventricular conduction delay. Another important aspect to interpreting ECG examples of atrial flutter is the frequent difficulty in analyzing ST and T information, especially in the inferior leads where the flutter waves are large enough to hopelessly distort the repolarization. In those cases, the proper response is to not try to overinterpret that information.

Conduction abnormality Intraventricular conduction delay is present. The QRS is wide but does not meet criteria for LBBB or RBBB. The fact that the atrial rate is faster than the ventricular rate indicates that some of the atrial impulses are blocked in the AV node before reaching the ventricle. If the atrium were firing at a normal rate, this would be second degree AV block, but in this case, with the atrium going so fast, the AV block is to some extent physiologic, and so AV block is not an important part of the diagnosis. However if the ventricular response is slow and not associated with the atrial activity, complete heart block would be present. In this case, the last 6 QRS complexes are clearly associated with the atrial activity, because the QRS's occur with the same relationship to the position on the flutter wave, easily noted in leads II and V1.

Diagnoses:

1. Atrial flutter with variable ventricular response
2. Occasional PVC
3. Intraventricular conduction delay
4. Nonspecific ST and T wave abnormality.

30.

Rate: atrial 270 ventricular 90

Axis QRS -60 **Axis P** ?? **Axis T** ?? **QRS AXIS IS LEFT AXIS DEVIATION**
PR interval ?? **QRS interval** 0.16 sec **QT interval** 0.40 sec

Rhythm: atrial flutter with variable conduction. This conduction alternates between 2:1 and 4:1, so there is a bigeminal appearance (long-short-long-short-long-short-etc). This is a common pattern of ventricular conduction. When trying to accurately determine a rapid regular rate such as this flutter rate, it is helpful to determine the distance between not 2 successive waves, but a multiple of waves. In this example, 9 flutter waves in lead II occupy almost exactly 2 seconds (10 large 5-mm blocks), so 60 seconds has 9 times 30 or 270 flutter waves per second.

Hypertrophy none. Note that it is not possible to assess for atrial abnormality if definite sinus P waves are not present. There are prominent anterior forces, but the QRS is abnormally wide, from the RBBB, so RVH cannot be diagnosed

Infarction No definite pathologic Q waves are present in the limb leads, but there are Q waves in V1-V3, indicating a prior anteroseptal MI. Recall that it is possible to diagnose a MI by ECG in the presence of a RBBB, since the initial forces are undisturbed in the presence of a RBBB (a rightward anterior terminal conduction delay).

Conduction abnormality The QRS is wide, and is typical for a RBBB. There is also the conduction deficit at the AV node, the alternating 2:1 and 4:1 block.

Diagnoses:

1. Atrial flutter with alternating 2:1 and 4:1 conduction
2. Right bundle branch block
3. Anteroseptal MI, age indeterminate

31.

Rate: atrial ?? **ventricular** 72
Axis QRS +30 **Axis P** ?? **Axis T** +75 **QRS AXIS IS NORMAL**
PR interval ?? **QRS interval** 0,09 sec **QT interval** ??

Rhythm: Atrial fibrillation. This is an easy example. There is a relatively slow ventricular response, leaving plenty of opportunity to examine atrial activity in the generous line between the end of the T wave and the beginning of the next QRS. Notice that there is really no place where the baseline is straight and quiet, but atrial activity is continuous. It is not possible to determine an atrial rate, and since there are really no P waves, only fibrillation waves, it is not possible to determine a P wave axis or duration. The fibrillatory waves are not regular. They vary in size and shape, the chief difference between fibrillation and flutter. In flutter, it is also not possible to determine P wave axis or duration, but the flutter waves are regular and are constant in size and shape, because flutter is an organized reentrant rhythm as opposed to fibrillation, which is a disorganized rhythm. There is some discussion about the terminology of ventricular response in atrial fibrillation, but the best terminology is that a ventricular rate of less than 70 is a slow ventricular response, and a ventricular rate of 70-110 is a moderate response, and that >110 is a rapid ventricular response. The examples with the slow ventricular response are the easiest to interpret. The fibrillatory waves are small in this example, and so the appellation of fine atrial fibrillation could be applied. If the waves were large, then coarse atrial fibrillation would be appropriate.

Hypertrophy none

Infarction none

Conduction abnormality none. The considerations for the ventricular response in atrial fibrillation are similar to flutter, except that there can never be a regular response to atrial fibrillation through the AV node. Recall that in atrial flutter, there can be 2:1 or 4:1 conduction and these are regular ventricular rhythms, generally at a rate of about 150 for 2:1 and a rate of about 75 for 4:1. But if the atrium is fibrillating and the ventricular response is regular, then the ventricle is beating through some mechanism other than conduction through the AV node, such as junctional escape rhythm or junctional tachycardia, or ventricular escape rhythm or ventricular tachycardia.

Diagnoses:

1. Atrial fibrillation with a moderate ventricular response.
2. Otherwise unremarkable ECG.

32.

Rate: atrial ?? ventricular 78
Axis QRS +60 **Axis P** ?? **Axis T** +15 **QRS AXIS IS NORMAL**
PR interval ?? **QRS interval** 0.08 sec **QT interval** ??

Rhythm: Atrial fibrillation. This example is similar to the previous. Notice again the irregularly irregular ventricular response and notice that in this patient, the fibrillatory waves are larger and seem at times to have a shape similar to the sawtooth of atrial flutter. But these waves are not regular, and the pattern breaks down from time to time. Atrial flutter does not break down, so this cannot be flutter. Also, this might at first glance be MAT, but there are no distinct P waves here and no distinct intervals without atrial activity. This is coarse atrial fibrillation.

Hypertrophy none

Infarction none

Conduction abnormality none, other than the moderate ventricular response. The atrial rate in fibrillation is generally considered to be greater than 350/min, up to 500 or 600/min

Diagnoses:

1. Atrial fibrillation with a moderate ventricular response
2. Otherwise unremarkable ECG.

33.

Rate: atrial ?? ventricular 138
Axis QRS -75 **Axis P** ?? **Axis T** +75 **QRS AXIS IS LEFT AXIS DEVIATION**
PR interval ?? **QRS interval** 0.12 sec **QT interval** 0.30 sec

Rhythm: Atrial fibrillation with a rapid ventricular response. This is a more difficult tracing with a rapid rate, so very little time is spent in diastole, but there are times when the R-R interval is long enough to get a fairly good look at the fibrillatory waves, especially after the 7th and 13th and 20th QRS complexes. Also, in front of the other QRS complexes, there is an almost infinite variety of atrial waveforms, and there are no times of atrial diastole or silence. This is classic for atrial fibrillation. In terms of the QRS pattern, there is the natural irregularly irregular rhythm of the QRS, and this is an important factor to notice in these patients (other causes of an irregularly irregular rhythm include atrial flutter with a variable ventricular response and MAT, and less commonly, just very frequent PAC's or even possibly very frequent PVC's). When the ventricular response is more rapid, the irregularity in the R-R interval becomes more subtle, and the complexes begin to look a little more regular, as in this case. So, in a patient with atrial fibrillation and a rapid ventricular response more careful attention must be paid to 2 things: the atrial fibrillatory waves, and the irregularity of the R-R interval.

Hypertrophy LVH might cross your mind, with the large deflections, but the QRS is too wide, so the diagnosis can't be made.

Infarction Inferior MI and anterior MI might cross your mind with Q waves in the Inferior leads and with such poor Q wave progression, but the intraventricular conduction delay and the severe left axis deviation make these diagnoses uncertain.

Conduction abnormality there is a wide QRS. In this case, the delay is not typical for a LBBB or a RBBB, and also not paced beats and not WPW, so the remaining possibility is merely an IVCD, a nonspecific intraventricular conduction delay. In any ECG that has marked left axis deviation (no matter what the cause), as in this patient, the R wave progression is likely to be poor, and the more severe the left axis deviation, the poorer the R wave progression.

Diagnoses:

1. Atrial fibrillation with a rapid ventricular response
2. Nonspecific intraventricular conduction delay

34.

Rate: atrial 84 ventricular 84
Axis QRS +90 **Axis P** +60 **Axis T** +30 **QRS AXIS IS NORMAL**

PR interval 0.12 sec **QRS interval** 0.10 sec **QT interval** 0.38 sec

Rhythm: sinus rhythm with frequent PVC's. In this case, the 2nd, 5th, 8th, 11th, and 15th beats are early and different from the normal sinus beats. Importantly, the T wave of the beat preceding the PVC has nothing unusual about it. Those T waves look just like the T waves on the beats not preceding the PVC's. This is important because such a distortion would indicate the likelihood of an early P wave distorting the T wave, and causing the early wide QRS, and then the diagnosis would be PAC with aberrant ventricular conduction instead of PVC. Since every third beat is a PVC, this is ventricular trigeminy. Since the PVC's in one lead all look like each other, these are unifocal PVC's. Another thing to notice about the PVC's is that after the wide bizarre QRS, there is a little wiggle in the ST segment, best seen in the V1 rhythm strip. And this is a P wave hiding. This P wave is actually earlier than would be expected of the next sinus beat. It is also different in shape from the normal sinus beat. So, it is early and it is different from a sinus beat. You might want to say that the PVC is then followed by a PAC, and that would be an attractive thought, given that PAC's are early different P waves, but that is not what is happening here. Here, the P wave is being caused by retrograde conduction of the PVC impulse up the AV node and into the atrium. So the diagnosis is PVC with retrograde conduction, and these are "retrograde P waves". The presence of the retrograde P wave gives certainty to the atrial rate of 84.

Hypertrophy none noted. The rightward QRS axis raises the suspicion of RVH, but none is seen in V1. The P wave is peaked in II, but not tall enough to make criteria of RAE.

Infarction none. No pathologic Q waves and normal precordial R wave progression. There is mild ST segment depression, but this is nonspecific, and the T waves look OK.

Conduction abnormality none.

Diagnoses:

1. Normal sinus rhythm
2. Frequent unifocal PVC's with trigeminy and retrograde conduction
3. Minor nonspecific ST segment abnormality

35.

Rate: atrial 72 ventricular 72

Axis QRS -45 **Axis P** +30 **Axis T** -30

QRS AXIS IS LEFT AXIS DEVIATION

PR interval 0.16 sec **QRS interval** 0.10 sec **QT interval** 0.46 sec

Rhythm: sinus rhythm with frequent PVC's. These PVC's are unifocal (uniform), and they occur every other beat, called ventricular bigeminy. In this case, with PVC's being so frequent, determining the actual sinus rate is not certain. There is a P wave that is at the end of the PVC complex, at the beginning of the ST segment, and in lead II, that P wave is deeply inverted. After the first PVC on the tracing however, there is no P wave at the end of the QRS. So, this is intermittent retrograde conduction. The P wave is clearly retrograde, being deeply negative in II.

Hypertrophy none

Infarction There are deep Q waves in II, III, and also in aVF from an inferior wall MI. The T wave inversion means that the age of the MI is not determinable. There is also a prolonged QT interval, and nonspecific T wave flattening in the lateral leads.

Conduction abnormality none

Diagnoses:

1. Normal sinus rhythm
2. Frequent unifocal PVC's with bigeminy and intermittent retrograde conduction.
3. Prior inferior wall MI, age indeterminate
4. Prolonged QT interval.

36.

Rate: atrial 70 ventricular 70

Axis QRS +45 **Axis P** 0 **Axis T** +120

QRS AXIS IS NORMAL

PR interval 0.14 sec **QRS interval** 0.08 sec **QT interval** 0.38 sec

Rhythm: sinus rhythm with frequent unifocal PVC's. There is again an early different QRS without a preceding distortion of the T wave. Notice that in this tracing that the PVC's come at

various intervals from the preceding normal sinus QRS. This is called variable coupling, and is not the usual occurrence. Usually PVCs are from a reentrant mechanism, and so the coupling to the preceding normal beat is constant, or "fixed". The coupling intervals (called R-R-prime) are 0.43 sec, 0.53, 0.69, and 0.51 sec. In this case, the difference is not very important. This is not of great prognostic importance. In this case, there are retrograde P waves seen best in leads II. The first, second, and fourth PVC's have retrograde conduction. The third PVC, with the longest coupling interval, has no retrograde conduction because the sinus node had already captured the atrium first, the PVC being too late.

Hypertrophy none

Infarction none. However the repolarization again is abnormal with nonspecific ST and T abnormality.

Conduction abnormality none.

Diagnoses:

1. Normal sinus rhythm
2. Frequent unifocal PVC's, with variable coupling interval.
3. Nonspecific ST and T wave abnormality

37.

Rate: atrial 60 ventricular 60

Axis QRS -30 **Axis P** +60 **Axis T** +90 **QRS AXIS IS NORMAL**

PR interval 0.19 sec **QRS interval** 0.10 sec **QT interval** 0.44 sec

Rhythm: sinus rhythm with the P wave preceding the 5th QRS being early, and also being a little different from the sinus P wave, slightly different in shape from the others, especially in II and V5. Then there are 2 PVC's, and they are different from each other, so the diagnosis is multifocal PVC's. The first PVC is followed by a retrograde P wave best seen in II and V1, and the second PVC does not have a visible P wave associated with it.

Hypertrophy The P wave in lead III has a notch with 0.04 sec between the peaks and the terminal negativity in V1 is more than 1 mm deep and wide, so LAE is present

Infarction none

Conduction abnormality none. The PAC has a slightly longer PR interval, but that is physiologic.

Diagnoses:

1. Normal sinus rhythm
2. Occasional multifocal PVC
3. Left atrial enlargement
4. PAC

38.

Rate: atrial 95 ventricular 120

Axis QRS +105 **Axis P** ?? **Axis T** --- **QRS AXIS IS RIGHT AXIS DEVIATION**

PR interval ?? **QRS interval** 0.19 sec **QT interval** ??

Rhythm: sinus rhythm at a rate of 95, with ventricular tachycardia (VT) at a rate of 120. There is an obvious wide complex tachycardia. The presence of a wide complex tachycardia makes the search for P waves quite difficult. In this case, the QRS is quite wide, and that makes finding P waves more difficult. But also in this case, the rate is on the slow side for wide complex tachycardia, making the search a little easier. In this example, the P waves are easily seen in V1, after the 3rd and 4th complexes as deep negative deflections. Using calipers and marching towards the beginning of the strip, P waves can be found in many other areas as well, and the atrial rhythm is mercifully regular, making the task easier. Wide complex tachycardia is usually ventricular tachycardia, especially if there is a history of coronary disease, and especially if there is AV dissociation with the ventricular rate faster than the atrial rate, as in this case. In this case, the AV dissociation is complete without either complex interfering with the rhythm of the other. In this case, the AV dissociation is due to the mechanism, "usurpation", since the VT usurps the normally dominant role of the atrium.

Hypertrophy can't say, no normally conducted QRS complexes. There is left atrial enlargement in V1.

Infarction can't say, no normally conducted QRS complexes.

Conduction abnormality can't say, no normally conducted QRS complexes.

Diagnoses:

1. Sinus rhythm
2. Left atrial enlargement
3. Ventricular tachycardia

39.

Rate: atrial 55 ventricular 55

Axis QRS -30 **Axis P** +30 **Axis T** +120 **QRS AXIS IS NORMAL**

PR interval 0.27 sec **QRS interval** 0.09 sec **QT interval** 0.42 sec

Rhythm: sinus bradycardia

Hypertrophy none

Infarction There are significant Q waves in III and aVF, indicating prior inferior wall MI. There are Q waves in V1-V3 indicating prior anterior wall MI

Conduction abnormality The PR interval is abnormal, and it is prolonged and constant. Every P wave is associated with a QRS, so this is first degree AV block. This is very possibly a result of medication, and does not really represent a threat to this patient. No special treatment need be undertaken.

Diagnoses:

1. Sinus bradycardia
2. First degree AV block
3. Prior anteroseptal MI
4. Prior inferior wall MI

40.

Rate: atrial 66 ventricular 66

Axis QRS 0 **Axis P** +60 **Axis T** +135 **QRS AXIS IS NORMAL**

PR interval 0.38 sec **QRS interval** 0.09 sec **QT interval** 0.38 sec

Rhythm: sinus rhythm

Hypertrophy none

Infarction there is poor R wave progression in V1 to V4. There is an inferior MI, with pathologic Q in aVF and III.

Conduction abnormality definite first degree AV block, somewhat more severe than the prior example. The only way to be sure about catching this diagnosis is to be consistent about measuring the PR interval, because the tracing otherwise doesn't look like a heart block, with normal rate and constant PR interval, and regular QRS complexes

Diagnoses:

1. Normal sinus rhythm
2. First degree AV block
3. Poor precordial R wave progression
4. Prior inferior wall MI, old

41.

Rate: atrial 125 ventricular 72

Axis QRS -30 **Axis P** +60 **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval variable **QRS interval** 0.10 sec **QT interval** ??

Rhythm: sinus tachycardia. The P waves are easily discernable and rapid. Recall that a rapid sinus rate is a clinically significant finding, indicating increased sympathetic tone and should raise the issues of possible CHF, pulmonary problems, thyrotoxicosis, infection, etc.

Hypertrophy the QRS voltage in V2 is high, but this patient is too young for the voltage criteria to apply.

Infarction none. The repolarization in the precordial leads, especially V4-6 appears definitely prolonged, and even though there is not a definite QT interval measurement, this late repolarization is concerning.

Conduction abnormality The most important first finding is that there are more P waves than QRS's, that is, the atrial rate is faster than the ventricular rate. The atrial rhythm is regular and the P waves are uniform, sinus tachycardia. The next step is to assess the regularity of the QRS complexes, and they are not regular. Occasionally they look a little regular, but overall are quite variable in their R-R intervals. Third, notice the relationship between the atria and the ventricles, the PR interval. Notice that the PR interval after a pause is relatively short, about 0.20 sec. Then on a subsequent conducted beat (not after a pause) the PR interval is longer, in this example about 0.28 sec. Then the third conducted beat may be even longer, about 0.32 sec, then there is a dropped beat. In the precordial leads the relationship is loosely a 2:1 relationship. When some atrial beats conduct and some don't, this is second degree AV block. In second degree AV block, if the PR interval is variable, then the diagnosis is Wenckebach, or Mobitz I AV block. This is not a very easy example of Wenckebach, because the atrial rate is fast.

Diagnoses:

1. Sinus tachycardia.
2. Second degree AV block, Wenckebach type.
3. Nonspecific ST and T wave abnormality.
4. Prolonged QT interval.

43.

Rate: atrial 92 ventricular 46

Axis QRS 0 **Axis P** +60 **Axis T** +90 **QRS AXIS IS NORMAL**

PR interval NA **QRS interval** 0.14 sec **QT interval** 0.54 sec

Rhythm: sinus rhythm.

Hypertrophy The P waves in II appear tall and over 2.5 mm in height in the setting of a normal standardization ECG. This is right atrial enlargement (RAE).

Infarction In the setting of a RBBB, infarction can still be diagnosed, and an inferior wall MI is present. The repolarization is also abnormal with a prolonged QT interval.

Conduction abnormality This is a tricky ECG, because many P waves are buried in the T waves of conducted beats. The PR interval is constant for the conducted beats, but half of the P waves are nonconducted. They are best seen in aVF, and also in the rhythm strip in lead II. The strange nature of the T wave is the main clue. This bump or distortion on the T wave is halfway between the obvious P waves, a strong factor in deciding that those are in fact nonconducted P waves. This is 2:1 AV block, and is presumptive Mobitz II AV block. In Mobitz II AV block, the PR interval is always constant for the conducted beats. There is also a RBBB. This RBBB in the setting of Mobitz II AV block raises the concern that the conduction system below the AV node is severely diseased, and complete heart block risk is significant. Mobitz II AV block usually occurs below the AV node, where conduction in the His-Purkinje system is "all-or-none", and Mobitz I AV block (Wenckebach) usually occurs in the AV node, where conduction is "decremental". In 2:1 AV block, the R-R intervals are regular. In Wenckebach, the R-R intervals are not regular, but often exhibit beats occurring in groups.

Diagnoses:

1. Normal sinus rhythm
2. Right bundle branch block
3. Inferior Wall MI
4. Prolonged QT interval
5. Second degree AV block, 2:1

43.

Rate: atrial 88 ventricular 54
Axis QRS +45 **Axis P** +45 **Axis T** +75 **QRS AXIS IS NORMAL**

PR interval variable **QRS interval** 0.10 sec **QT interval** 0.48 sec

Rhythm: sinus rhythm. The P waves are regular and uniform in appearance.

Hypertrophy none

Infarction none

Conduction abnormality no intra-atrial or intraventricular conduction abnormality, but a definite atrioventricular conduction abnormality. The P waves are regular. The QRS complexes are regular. The PR interval is totally random. These are the criteria for complete, or third degree AV block, or complete heart block. In complete heart block, an important consideration is what is driving the ventricle. In this case, the QRS is narrow, and so the AV junction is driving the ventricle, and this is called a junctional escape rhythm.

Diagnoses:

1. Normal sinus rhythm
2. Complete AV block
3. Junctional escape rhythm

44.

Rate: atrial 100 ventricular 35
Axis QRS +60 **Axis P** +30 **Axis T** +60 **QRS AXIS IS NORMAL**

PR interval variable **QRS interval** 0.09 sec **QT interval** 0.58 sec

Rhythm: sinus tachycardia. Recall that it is the P wave that is mainly responsive to autonomic stimuli, so this patient is probably not doing well.

Hypertrophy none

Infarction none

Conduction abnormality Again, no intra-atrial or intraventricular conduction abnormality, but a definite atrioventricular conduction abnormality. This is another example of complete heart block, this time with a very slow, and probably inadequate, ventricular junctional escape rate of 35. Again the PR interval is totally random. Occasionally a P wave will occur at a decent interval before the QRS, but that is just a coincidence.

Diagnoses:

1. Sinus tachycardia
2. Complete AV block
3. Slow junctional escape rhythm

45.

Rate: atrial 50 ventricular 50
Axis QRS 0 **Axis P** -90 **Axis T** ?? **QRS AXIS IS NORMAL**

PR interval ?? **QRS interval** 0.09 sec **QT interval** 0.46 sec

Rhythm: Junctional escape rhythm. The QRS is narrow and normal in appearance, but it is not preceded by a P wave, so it is firing on its own. This is a good thing, since it is only going at a rate of 50, and if it weren't firing, maybe nothing would, we don't know. Notice that after every QRS, especially in the rhythm strip lead II, that there is an inverted P wave. Notice that the P wave axis is very superior at -90, indicating that the P wave is retrograde, produced by the junctional escape rhythm.

Hypertrophy none

Infarction none. The repolarization again is abnormal.

Conduction abnormality none. Actually, the problem is not with conduction here, but with automaticity. The sinus node is failing to do its job as the pacemaker.

Diagnoses:

1. Junctional escape rhythm with 1:1 retrograde conduction

2. Nonspecific ST and T wave abnormality

46.

Rate: atrial 70 ventricular 70

Axis QRS -75 **Axis P** ?? **Axis T** +100 **QRS AXIS IS LEFT AXIS DEVIATION**

PR interval 0.16 sec **QRS interval** 0.26 sec **QT interval** 0.58 sec

Rhythm: The very spikey things that are too sharp to be normal physiologic heart complexes are discharges from an electronic cardiac pacemaker. In this case, there are 2 spikes for each QRS, separated by 0.16 seconds. The first of these is an atrial spike and the second is a ventricular spike. The atrial spike is followed by a small wave, best seen in rhythm strip V1. The second spike is followed by a very wide QRS complex. This is a dual chamber (A-V sequential) pacemaker, and it is constantly firing, never pausing. Every spike is followed by a physiologic cardiac wave, indicating that each spike successfully captures its chamber. The atrial lead is usually in the right atrial appendage, and the ventricular lead is usually in the RV apex. The QRS complex often looks a little like a LBBB pattern, but with a left axis deviation, as seen in this typical example.

Hypertrophy cannot say, no sinus beats

Infarction cannot say, no sinus beats

Conduction abnormality cannot say, no sinus beats. It is expected that there must have been a prior underlying conduction abnormality, to have provoked this patient's physician to implant the pacemaker in the first place.

Diagnoses:

1. AV sequential pacemaker with 100% capture.