ECG A Methodical Approach

Nowadays almost every practice has its own ECG machine. Most modern machines have a computerised system for interpretation but the clinician (doctor or nurse) should have at least a basic understanding of interpretation, as machines are not infallible. The best results are obtained when machine interpretation is used to enhance the physician's diagnosis, even for cardiologists.¹

Most texts to aid interpretation of ECGs go back to basics in that they deal with the effects of depolarisation in various directions and the sum of vectors. Many non-specialists find this daunting and confusing. This is a simple, methodical, dogmatic and pragmatic approach to interpreting an ECG. A systematic approach, looking for certain features in a certain order, should substantially reduce the risk of major abnormality being overlooked. Minor abnormalities of the ECG are very common and become exponentially more common with advancing years.² Even young, highly-trained athletes may have some minor changes.³ An ECG alone as a screening test for CHD in the asymptomatic population is not a valid means of assessment.⁴

Interpretation requires some clinical details. How old is the patient? Is there a history of chest pain or previous myocardial infarction? Is there complaint of palpitations? Is the patient tall and thin or short and wide? Is there COPD, or is there hypertension?

Identification

The tracing should include the name of the patient, date and possibly the time of recording.

Quality and calibration

- There must be a full 12 leads recorded and labelled plus a rhythm strip, usually from lead II.
- The baseline must be stable and not wandering. Leads must be well attached, even if this means shaving a hairy chest.
- There should be little interference from skeletal muscle. The patient must be relaxed and comfortable. Sometimes dyspnoea makes this difficult.
- There should be a square wave calibration to show that 1 mV is equivalent to 1 cm in height.
- Speed should be 25 mm/sec. Hence 1 large square is 200 msec and 1 small square is 40 msec.

Rhythm and rate

Start with the rhythm strip. This is a more prolonged recording of lead II.

- Are there P waves?
- Are they regular?
- Does every one precede a QRS?
- Is the PR interval constant?
- What is the PR interval?

The PR interval should be between 120 and 240 msec (3 to 6 small squares).

What is the ventricular rate?

**Count the number of R waves over 15 large squares (3 seconds) and multiply by 20.** To be slightly more accurate count the number of R waves over 30 large squares (6 seconds) and multiply by 10. The latter is preferable if the rate is slow or irregular.
QRS complex

Turn next to the precordial leads.

- In V1, R should be much smaller than S
- In V6, Q should be much smaller than R
- R and S should be of approximately equal size around V3 or V4.
- How long is the QRS complex?

It should be between 80 and 120 msec or 2 to 3 small squares.

- Look at the V leads and measure the height of the tallest R and the deepest S. The sum should not exceed 40 mm (some say 35 mm). If the sum of the deflections exceeds 40 mm there is electrical left ventricular hypertrophy. Assessment of left ventricular mass by ECG criteria is unreliable but electrical LVH carries a poor prognosis.
- Where there are Q waves, the depth should be no more than the height of the R in that lead.
- The ST segments should not be more than 1 mm above or below the base line. A little more deviation may be allowed in V1 and V2.
- In V1, T may be upright, inverted biphasic or flat.
- In V3 to V6, the T wave must be upright.

Now turn to the limb leads:

- If there is a Q wave in I, II, aVL or aVF it should not be more than a quarter of the size of the R wave.
- Larger Q waves may be found in III and aVR.
- Abnormal Q waves suggest myocardial infarction, old or recent.

The ST segments should not be more than 1 mm above or below the baseline.

aVR often has inverted P and T waves and a predominantly negative QRS complex. It should be thought of as an upside down lead.

Electrical axis

A tall, thin person will have a fairly vertical heart showing a right axis shift.

A short, broad person will have a fairly horizontal heart showing a left axis shift.

Electrical axis can be made unnecessarily complex but the following is a very simple guide:

- Look at lead I. Measure the height of the positive deflection and subtract from it the depth of the negative deflection.
- Look at aVF and do the same.
- If the mean vectors in both I and aVF are both positive the axis is between 0 and 90° and this is normal.
- If I is positive and aVF is negative this is left axis deviation (LAD).
- If I is negative and aVF is positive this is right axis deviation (RAD).
- If both I and aVF are negative this is extreme axis deviation, sometimes called "north-west territory".
- LAD up to -30° can be normal. If there is LAD, look at lead aVL. If aVL is predominantly negative, the axis is < 30° and normal. If it is predominantly positive the axis is > 30° and there is significant LAD.

Common abnormalities
The following are just a few of the commonest abnormalities to be encountered. The list is far from exhaustive.

**Atrial flutter**

P waves are well demonstrated in II, III and aVF but are best examined in V1.

*Atrial flutter* shows clear P waves like the teeth of a saw. There is usually a rapid ventricular rate and a 2:1 A-V block. The atrial rate is usually around 300 a minute with a ventricular rate around 150.

**Atrial fibrillation**

In *atrial fibrillation* the pattern is far less obvious. There may be rapid, small undulations, no apparent pattern or a pattern like a worn-out saw, compared to the new saw pattern of flutter. This means that the amplitude is much lower than in flutter.

**Abnormal PR interval**

- The PR interval is short in *Wolff-Parkinson-White* (WPW) syndrome. It is also short in the *Lown-Ganong-Levine syndrome* that is also a pre-excitation syndrome. It is less common, more controversial and is also described in its own article. There is a small deflection of the upstroke of the R wave in WPW. This is called a delta wave and is due to conduction through the aberrant Bundle of Kent. It is absent in LGL syndrome.
- In 1st degree heart block there is a prolonged but constant PR interval. It is over 200 msec and all P waves are followed by a QRS.
- 2nd degree heart block is called Mobitz I or Mobitz II:
  - In Mobitz I (Wenckebach's phenomenon) the PR interval becomes progressively longer after each beat until a QRS is dropped and the pattern starts again.
  - In Mobitz II the PR interval is prolonged but, at regular intervals such as every 3rd or 4th P wave, there is not a QRS to follow.
- 3rd degree heart block is complete A-V dissociation and there is no relationship between the P waves and the QRS complexes. They are fired from different pacemakers. Bradycardia is usual.

![ECG](image)

Intermittent Wolff-Parkinson-White syndrome (first and fourth beats). By comparison with the normal beats, it can be seen how the delta wave both broadens the ventricular complex and shortens the PR interval.

**Ventricular tachycardias**

In *supraventricular tachycardia* the rate is very fast, without a preceding P wave. There may be retrograde conduction to excite the atria in reverse order. SVT may look rather similar to a fast AF but the rhythm is likely to be more regular. With a fast rate there may be little time between QRS complexes so that atrial forms are difficult to find. The shape of the QRS complex is basically
normal, as the pacemaker is at or near the A-V node and conduction spreads down the Purkinje fibres in the usual way.

In **ventricular tachycardia** the rate is also very fast but the complex is abnormal, as the pacemaker is not high up in the conducting system. A broader complex and inverted T waves are usual.

![ECG Torsade de pointes ventricular tachycardia](image)

**Myocardial infarction**

The picture with myocardial infarction will vary according to the site of the infarction and whether it is partial or full thickness. Leads I and aVR are anterior leads. II, III, and aVF are inferior leads. The V leads will show if it is anterior-septal with early V leads affected or anterior-lateral with late V leads involved.

- In the acute phase (within minutes) there is ST elevation over the affected area.
- With a full thickness infarct there may be ST depression over the reciprocal leads (inferior leads in an anterior infarct and anterior leads in an inferior infarct).
- T waves are higher and wider. This is called hyperacute T wave changes.
- Pathological Q waves are defined as duration beyond 40 msec or > of R wave amplitude
- Over the next few hours the ST segments return to normal and, shortly after, the T waves become inverted but Q waves remain.
- If occlusion of the coronary artery is incomplete, infarction can occur without Q waves.
- If there is ST depression in V1 and 2 but no other signs add V7, 8, 9 as it could be a true posterior infarct. True **posterior myocardial infarction** is discussed in its own article.

V7 is along the same line as V5 and V6 but in the posterior axillary line and V8 and V9 are the same distance along again.

The use of ECGs to diagnose myocardial infarction and to start **thrombolysis** is validated.² The diagnostic ECG criteria for thrombolysis have varied slightly over the last few years but for both anterior and inferior infarction the criteria are:

- 1 mm of ST change in at least 2 contiguous limb leads (II, III, AVF, I,AVL).
- 2 mm of ST change in at least 2 contiguous chest leads (V1-V6).
- New left bundle branch block.

Diagnosis of an acute MI can be very difficult in the presence of an old MI or left bundle branch block (LBBB).

The diagnostic criteria for a true posterior infarct are not so well agreed, as discussed in the article, and this makes the early initiation of thrombolysis more difficult.
Acute coronary syndrome

This term encompasses a range of coronary artery diseases, including unstable angina and both ST segment elevation and non-ST segment elevation myocardial infarction. Diagnosis requires an ECG and a careful clinical review. In acute coronary syndrome, common ECG abnormalities include T-wave tenting or inversion, ST segment elevation or depression including J-point elevation in multiple leads and pathological Q waves.\(^8\)

Bundle branch block

Right bundle branch block (RBBB) may occur as a congenital anomaly or in association with volume overload in the right ventricle. LBBB is almost always pathological, reflecting disease of the left ventricle. There are left and right bundles but the left has an anterior and posterior fascicle.

- RBBB produces a prolonged QRS, usually about 160 msecs or 4 small squares and a RSR pattern seen best in V1. There may well be T wave changes in the septal leads.
- Left anterior hemiblock is associated with LAD (with an initial R in II, III and aVF, Q in I and S in III).
- Left posterior hemiblock is rarer. There is RAD, S in I and Q in III.
- Complete LBBB produces a longer QRS, usually around 200 msec with a more square
pattern than RBBB. The changes are best seen in the lateral V leads. There may also be some ST elevation or depression.

**Axis deviation**

**Causes of LAD:**
- Left anterior hemiblock
- Inferior myocardial infarction
- Pacemaker
- Emphysema
- Hyperkalaemia
- Wolff-Parkinson-White syndrome
- Ostium primum atrial septal defect
- Some authorities say that left ventricular hypertrophy causes LAD whilst others are adamant that it does not

**Causes of RAD:**
- Normal finding in children and tall thin adults
- Right ventricular hypertrophy
- Chronic lung disease even without pulmonary hypertension
- Anterolateral myocardial infarction
- Left posterior hemiblock
- Pulmonary embolism
- Wolff-Parkinson-White syndrome
- Ostium secundum atrial septal defect
- Ventricular septal defect

**Causes of extreme axis deviation:**
- Emphysema
- Hyperkalaemia
- Pacemaker
- Ventricular tachycardia

**Causes of ST segment changes**
- **Digoxin** produces a down-sloping ST depression.
- **Pericarditis** causes ST elevation that tends to be generalised and not located to the area supplied by one coronary artery.
- Ventricular aneurysm causes ST elevation. This usually follows a large anterior infarct and, if the ST segment fails to return to the baseline, an echocardiogram is required to exclude aneurysm.
- ST depression suggests ischaemia and/or unstable angina. Extensive ST depressions with a clinical picture of MI can indicate subendocardial damage.

**Right ventricular hypertrophy**

The R wave is bigger than the S in V1.

**T waves**
- Inverted T waves in a predominantly positive lead suggest ischaemia or old MI. T waves should be upright in V3 to V6.
- Tall T waves occur in the acute phase of MI and in hyperkalaemia.

**Misplaced leads**
Wrongly attached leads, such as left and right arm reversed, will produce abnormalities in the tracing. This is a standard "trick question" in the MRCP(UK). It is a justified question because in real life, it happens.

- If the 2 ankle leads are reversed, it will make little difference.
- If the F lead is reversed with one of the arm leads, the ECG will seem very strange except in the V leads. There may be extreme axis deviation.
- If the left and right arm leads are reversed, lead I will show inverted P and T with a predominantly negative QRS whilst V leads are normal. Dextrocardia will show a similar pattern in lead I but leads V1 to V6 will show remarkably little change across their course. Misplacement of the arm leads is much more common than dextrocardia.

**Pulmonary embolism**

Traditional teaching is that S in 1 and Q and inverted T in III are features of pulmonary embolism. It is taught as $S1, Q3, T3$. However, where the diagnosis has not already been made, this is of no value.\(^9\)

**Learning to read ECGs**

A written text like this is of limited value in helping to acquire the expertise to read ECGs. Practice looking at tracings is essential. For that reason several websites have been given where samples of ECGs can be seen and interpreted. They all have text to accompany them.

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**Introduction to ECG Interpretation**

Electrocardiogram interpretation is an invaluable clinical skill that is taught in many different ways at medical schools across the country. It is often informal and clinicians are expected to "pick it up" as they see patients on the wards and in clinics. There are many "courses" which can be purchased off the shelves at the bookstore -some of them too simplistic and others hopelessly detailed. In an effort to better meet the needs of ecg interpretation this course started out at UW Madison as a self study manual with some workshops where clinicians reviewed tracings with preceptors. The popularity of this course was immediate - we soon had clinicians asking for it on other rotations and the workshops were often visited by an assortment of clinicians from the health care fields. Reasons identified for the course's success was that it was directly applicable to patient care, a skill that was enjoyable to master, of value as a lifelong career tool (ecg interpretation content has been stable for years),and "adult learning" principles were respected.

This computerized version of the course has some significant improvements. At the end of each content area is a quiz. This not only reinforces the material but the advanced clinician can try the quiz first, if they already feel comfortable with the content material, and go on to another section, to optimize their time. The tool bars at the bottom of the page allow this flexibility to go directly to any section. The "guidelines" which summarize the ENTIRE interpretive process can be printed out on a single page and carried in the lab coat pocket when seeing patients. The clinical cases at the end are an assortment of real life cases to demonstrate that ecgs are always interpreted in the context of patient care and not in a "vacuum". Differential diagnosis is emphasized to encourage thinking about the "art" of interpretation, not just a cookbook mechanistic approach. In addition, this course sets up the basic skills needed for interpretation in a self directed learning format, allowing clinician-preceptor interaction to focus on more advanced patient specific ecg correlation.

**ECG Videos**

If your site does not provide ECG lectures, watch these videos:

- [ECG Course 1](#)
- [ECG Course 2](#)
As always take every opportunity to read your patient's ECG first before reviewing it with staff. It is our sincere hope that you find this course worthwhile for all your inpatient and outpatient rotations and a skill you will continue to use throughout your careers in medicine.

A note on using this site: Navigation through the ecg site is provided by two menu bars. One appearing at the bottom of the page, in red, and one appearing at the top of the page, in white. The red menu bar at the bottom of every page, provides links to the main sections of the site in white letters. The top menu bar, found in some sections, provides links to pages within each section in blue letters. Click on a word or phrase in either menu bar to go to that section.

**Primer on Basic Concepts**

1. P wave = depolarization of the atria.
   QRS = depolarization of the ventricle.
   T wave = repolarization of the ventricle.

2. Cardiac muscle cells depolarize with a positive wave of depolarization, then repolarize to a negative charge intracellularly.

3. Skin "leads" or electrodes have a positive and negative end.

4. A positive wave form (QRS mainly above the baseline) results from the wave of depolarization moving towards the positive end of the lead. A negative waveform (QRS mainly below the baseline) is when a wave of depolarization is moving away from the positive electrode (towards the negative end of the lead).

5. ECG paper has 1 millimeter small squares - so height and depth of wave is measured in millimeters.

   10 mm = 1.0 mVolt

6. Horizontal axis is time.
   .04 seconds for 1 mm (1 small box).
   .2 seconds for 1 large box = 5 small boxes = 5 x .04 seconds.
Figure 2:
Positive QRS in Lead I.
Negative QRS in Lead aVR.
R wave = 7-8 mm high in Lead I.
QRS wave = .06 seconds long in Lead I.

7. Lead nomenclature.

<table>
<thead>
<tr>
<th>Limb Leads</th>
<th>Chest Leads</th>
<th>Rhythm Strip</th>
</tr>
</thead>
<tbody>
<tr>
<td>I, II, III aVR, aVF, aVL</td>
<td>V1 - V6</td>
<td>Located on the bottom of the ECG printout. Selected to give the best relationship of the P wave to the QRS.</td>
</tr>
</tbody>
</table>

Figure 3: A normal ECG and rhythm strip.

8. ECG interpretation: look at five areas, in order, on each ECG.

<table>
<thead>
<tr>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm (Intervals)</td>
</tr>
<tr>
<td>Axis</td>
</tr>
<tr>
<td>Hypertrophy</td>
</tr>
<tr>
<td>Infarct</td>
</tr>
</tbody>
</table>

**Rate**

Rate is cycles or beats per minute.

Normal rate for the SA node 60-100.

<60 bradycardia | >100 tachycardia

SA node is the usual pacemaker, other potential pacemakers (if SA node fails) are atrial pacemakers with inherent rates of 60-80, AV node (rate 40-60), or ventricular pacer (rate 20-40). In certain
pathologic conditions ectopic (out of place) pacemakers can go much faster at rates 150-250 cycles/minute. There are three methods of calculating rate:

1. **Most Common Method:**
   (Most rates can be calculated this way). Find an R wave on a heavy line (large box) count off "300, 150, 100, 75, 60, 50" for each large box you land on until you reach the next R wave. Estimate the rate if the second R wave doesn't fall on a heavy black line.

<table>
<thead>
<tr>
<th>Rate calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memorize the number sequence:</td>
</tr>
<tr>
<td>300, 150, 100, 75, 60, 50</td>
</tr>
</tbody>
</table>

   **Figure 4:** Common Method.

2. **Mathematical method:**
   Use this method if there is a regular bradycardia, i.e. - rate < 50. If the distance between the two R waves is too long to use the common method, use the approach: \(300/\# \text{ large boxes between two R waves}\).

   **Figure 5:** Count number of large boxes between first and second R waves = 7.5. \(300/7.5\) large boxes = rate 40.

3. **Six-second method:**
   Count off 30 large boxes = 6 seconds (remember 1 large box = 0.2 seconds, so 30 large boxes = 6 seconds). Then, count the number of R-R intervals in six seconds and multiply by 10. This is the number of beats per minute. This is most useful if you have an irregular rhythm (like atrial fibrillation) when you want to know an average rate.

   **Figure 6:** Count 30 large boxes, starting from the first R wave. There are 8 R-R intervals within 30 boxes. Multiply 8 \times 10 = Rate 80.
Rhythm (to include intervals)

We will focus on the basic "core" of rhythms and measured "intervals" (PR, QRS, QT). Rhythms are often the most challenging aspect of ECG's. You will see most rhythms several times over the next few years of your training, and you will eventually recognize them at a glance.

Now for some basics - "arrhythmia" means abnormal rhythm.

The normal conduction pathway is: SA node --> AV node --> Bundle of HIS --> Bundle Branches.

Arrhythmia can be understood by realizing the existence of ectopic (out of place) foci (pacemakers) and understanding the normal conduction pathway of the heart. Very simply put, if the beat originates in the atria or AV node (supraventricular) the QRS is usually narrow (normal), because it comes from above along the normal pathway.

![Figure 6a: QRS is narrow (normal).](image)

If the beat is ventricular in origin, the QRS is wide and bizarre because it doesn't come down the normal pathway.

![Figure 6b: QRS is wide.](image)

Aberrancy is an exception to this rule - here it does actually follow the normal pathway (atria - AV node - ventricle) but for some reason the pathway is refractory to the beat and you get a wide QRS.

A reasonable way to group arrhythmias is in four general groups. Let us briefly review these four groups, then we will develop some common sense principles for evaluating rhythm (to include intervals).

Axis

Direction of depolarization (vector) of the QRS complex.

1. The left ventricle is thicker so the mean QRS vector is down and to the left. (The origin of the vector is the AV node with the left ventricle being down and to the left of this).
2. The vector will point toward hypertrophy (thickened wall) and away from the infarct (electrically dead area).
Normal axis -30 to +90 degrees
Left axis deviation -30 to -90 degrees
Right axis deviation +90 to +/-180 degrees
Indeterminate (extreme) axis deviation -90 to +/-180 degrees

Since lead I and aVF are perpendicular to each other, you can use those two leads to quickly determine axis.

Lead I runs from right to left across a patient's body, positive at the left hand: (See figure 28).

If the QRS in lead I is positive (mainly above the baseline), the direction of depolarization will be in the positive half (right half) of the circle above. You can make a diagram and shade in the positive half of the circle.

Lead aVF runs from top to bottom across a patient's body, positive at the feet: (See figure 28).

If the QRS in lead aVF is positive (mainly above the baseline), the direction of depolarization will be in the positive half (lower half) of the circle above. You can make a diagram and shade in the positive half of the circle:
To find the axis overlap the two circles. The common shaded area is the quadrant in which the axis lies. In this example, the axis lies in the normal quadrant, which on a patient, points down and to the left.

You can repeat this process for any two leads, but I and aVF are the classic places to look. If you realize that there are two leads to consider and a positive (+) or (-) orientation for each lead, there would be four possible combinations. Memorize the following axis guidelines.

<table>
<thead>
<tr>
<th>Lead I</th>
<th>Lead aVF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

1. Normal axis (0 to +90 degrees)
2. Left axis deviation (-30 to -90) Also check lead II. To be true left axis deviation, it should also be down in lead II. If the QRS is upright in II, the axis is still normal (0 to -30).
3. Right axis deviation (+90 to +180)
4. Indeterminate axis (-90 to -180)

Figure 29: Normal axis.
Figure 30: Left axis deviation.

Figure 31: Right axis deviation.

The bottom line is, *if the axis is shifted out of the normal quadrant*, evaluate the reasons for this.

**Differential Diagnosis**

Left axis deviation  LVH, left anterior fascicular block, inferior wall MI
Right axis deviation RVH, left posterior fascicular block, lateral wall MI
Infarct

Accurate ECG interpretation in a patient with chest pain is critical. Basically, there can be three types of problems - ischemia is a relative lack of blood supply (not yet an infarct), injury is acute damage occurring right now, and finally, infarct is an area of dead myocardium. It is important to realize that certain leads represent certain areas of the left ventricle; by noting which leads are involved, you can localize the process. The prognosis often varies depending on which area of the left ventricle is involved (i.e. anterior wall myocardial infarct generally has a worse prognosis than an inferior wall infarct).

<table>
<thead>
<tr>
<th>Leads</th>
<th>Area of the Left Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1-V2</td>
<td>anteroseptal wall</td>
</tr>
<tr>
<td>V3-V4</td>
<td>anterior wall</td>
</tr>
<tr>
<td>V5-V6</td>
<td>anterolateral wall</td>
</tr>
<tr>
<td>II, III, aVF</td>
<td>inferior wall</td>
</tr>
<tr>
<td>I, aVL</td>
<td>lateral wall</td>
</tr>
<tr>
<td>V1-V2</td>
<td>posterior wall (reciprocal)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infarct</th>
<th>Represented by symmetrical T wave inversion (upside down). The definitive leads for ischemia are: I, II, V2 - V6.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Ischemia</td>
<td>Acute damage - look for elevated ST segments. (Pericarditis and cardiac aneurysm can also cause ST elevation; remember to correlate it with the patient.</td>
</tr>
<tr>
<td>2. Injury</td>
<td>Look for significant &quot;pathologic&quot; Q waves. To be significant, a Q wave must be at least one small box wide or one-third the entire QRS height. Remember, to be a Q wave, the initial deflection must be down; even a tiny initial upward deflection makes the apparent Q wave an R wave.</td>
</tr>
</tbody>
</table>
Figure 34: Ischemia: Note symmetric T wave inversions in leads I, V2-V5.

Figure 35: Injury: Note ST segment elevation in leads V2-V3 (anteroseptal/anterior wall).

Figure 36: Infarct: Note Q waves in leads II, III, and aVF (inferior wall).

For the posterior wall, remember that vectors representing depolarization of the anterior and posterior portion of the left ventricle are in opposite directions. So, a posterior process shows up as opposite of an anterior process in V1. Instead of a Q wave and ST elevation, you get an R wave and ST depression in V1.
Figure 37: Posterior wall infarct. Notice tall R wave in V1. Posterior wall infarcts are often associated with inferior wall infarcts (Q waves in II, III and aVF).

Two other caveats: One is that normally the R wave gets larger as you go to V1 to V6. If there is no R wave "progression" from V1 to V6 this can also mean infarct. The second caveat is that, with a left bundle branch block, you cannot evaluate "infarct" on that ECG. In a patient with chest pain and left bundle branch block, you must rely on cardiac enzymes (blood tests) and the history.

fascicular Blocks

Fascicular blocks are blocks of part of the left bundle, either the posterior or anterior division:

Figure 38: Divisions of the bundles.

Anterior fascicular block - the most common.

You will see left axis deviation (-30 to -90) and a small Q wave in lead I and an S in lead III (Q1S3). The QRS will be slightly prolonged (0.1 - 0.12 sec).
Figure 39: Anterior fascicular block.

Posterior fascicular block - less common.
You will see right axis deviation, an S in lead I and an Q in lead III (S1Q3). The QRS will be slightly prolonged (0.1 - 0.12 sec).

Figure 40: Posterior fascicular block.

Bifascicular block.
This means two (2) of the three (3) fascicles (in diagram) are blocked. The most important example is a right bundle branch block and a left anterior fascicular block. Watch out for this. Only one fascicle is left for conduction, and if that fasicle is intermittently blocked, the dangerous Mobitz 2 is set up!
"fascicular Blocks" may seem a bit complicated - simply remember that axis deviation is the clue. In your differential, consider posterior fascicular blocks with right axis deviation and consider anterior fascicular blocks with left axis deviation. Fascicular blocks cause axis deviations, like infarcts and hypertrophy. If you see a left or right axis deviation, first look for infarct or hypertrophy. If neither are present, the remaining diagnosis of fascicular block is usually correct. Review differential diagnosis of right and left axis deviation.

SYSTEMATIC INTERPRETATION GUIDELINES for Electrocardiograms

RATE
Rate calculation
Common method: 300-150-100-75-60-50
Mathematical method: 300/# large boxes between R waves
Six-second method: # R-R intervals x10

RHYTHM
Rhythm Guidelines:
1. Check the bottom rhythm strip for regularity, i.e. - regular, regularly irregular, and irregularly irregular.
2. Check for a P wave before each QRS, QRS after each P.
3. Check PR interval (for AV blocks) and QRS (for bundle branch blocks). Check for prolonged QT.
4. Recognize "patterns" such as atrial fibrillation, PVC’s, PAC’s, escape beats, ventricular tachycardia, paroxysmal atrial tachycardia, AV blocks and bundle branch blocks.

AXIS

1. Normal axis (0 to +90 degrees) Lead I Lead aVF
   Lead I Positive
   Lead aVF Positive
2. Left axis deviation (-30 to -90) Also check lead II. To be true left axis deviation, it should also be down in lead II.
   Left axis deviation differential: LVH, left anterior fascicular block, inferior wall MI.
3. Right axis deviation (+90 to +180)
   Right axis deviation differential: RVH, left posterior fascicular block, lateral wall MI.
4. Indeterminate axis (-90 to -180)
   Negative Negative

HYPERTROPHY
1. LVH -- left ventricular hypertrophy = S wave in V1 or V2 + R wave in V5 or V6 > 35mm or aVL R wave > 12mm.
2. RVH -- right ventricular hypertrophy = R wave > S wave in V1 and gets progressively smaller to left V1-V6 (normally, R wave increases from V1-V6).
3. Atrial hypertrophy (leads II and V1)
   **Right atrial hypertrophy** -- Peaked P wave in lead II > 2.5 mm in amplitude. V1 has increase in the initial positive direction.
   **Left atrial hypertrophy** -- Notched wide (> 3mm) P wave in II. V1 has increase in the terminal negative direction.

### INFARCT

<table>
<thead>
<tr>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Injury</td>
<td>Acute damage -- look for elevated ST segments.</td>
</tr>
<tr>
<td>Infarct</td>
<td>&quot;Pathologic&quot; Q waves. To be significant, a Q wave must be at least one small square wide or one-third the entire QRS height.</td>
</tr>
</tbody>
</table>

**Certain leads represent certain areas of the left ventricle:**

<table>
<thead>
<tr>
<th>V1-V2</th>
<th>anteroseptal wall</th>
<th>II, III, aVF</th>
<th>inferior wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>V3-V4</td>
<td>anterior wall</td>
<td>I, aVL</td>
<td>lateral wall</td>
</tr>
<tr>
<td>V5-V6</td>
<td>anterolateral wall</td>
<td>V1-V2</td>
<td>posterior wall (reciprocal)</td>
</tr>
</tbody>
</table>

*Print this window.* (May not work in IE or old versions of either browser. If so, please use Print button at top of browser.)

Continue to the clinical cases.