Systematic Interpretation of ECG and Review

Although some new concepts are introduced, this chapter largely reviews what has already been introduced and provides a framework for systematic evaluation of ECGs. A variety of parameters (PR interval, QRS duration, relationship of P waves to QRS complexes, etc.) must be evaluated for proper ECG interpretation. The task can seem overwhelming and mistakes will be made if a systematic strategy is not used. The particular system used is probably not as important as is the use of an approach that is organized and inclusive. This chapter will describe a logical system that, after determination of rate, begins with examination of the P waves and proceeds to the right. Table 13.1 summarizes the steps. The capitalized words highlight which areas of interpretation are covered during each step. We will return to this system following a brief review of T wave patterns.

T Waves

In previous chapters a variety of T wave patterns were encountered, and they are summarized in Figure 13.1. Very small (positive or negative) or virtually absent T waves fall into the “nonspecific T wave abnormality” category. Deep, symmetrically inverted T waves represent either ischemia or the repolarization abnormalities (“strain”) associated with hypertrophy. In order to be considered indicative of strain they must appear in leads showing R waves meeting voltage criteria for hypertrophy. As ischemia commonly occurs in hypertrophied hearts it is still not possible to rule out ischemia in such cases.

Deep or shallow T wave inversions can also occur with an evolving ST segment elevation myocardial infarction (STEMI). The ST segments may still be elevated (although not as much as during the acute phase) or may have returned to baseline.

Tall, peaked T waves occur with early hyperkalemia or very early in the course of a STEMI.

Table 13.1 Systematic Examination of ECG

<table>
<thead>
<tr>
<th>Rate</th>
<th>What is the rate? For many purposes it is sufficient, at least initially, to simply establish if it is slow, fast or normal. RHYTHM Is the rate (rhythm) regular?</th>
</tr>
</thead>
<tbody>
<tr>
<td>P waves</td>
<td>Are they positive in lead II? RHYTHM Are they unusually tall or wide in lead II? HYPERTROPHY Is the negative part of the P in lead V1 1 mm wide and deep? HYPERTROPHY Is the PR interval normal and consistent? RHYTHM, av blocks Is one P present for each QRS complex? RHYTHM, av blocks</td>
</tr>
<tr>
<td>QRS complexes</td>
<td>Is the QT interval normal? QT prolongation, drug EFFECTS, electrolyte disturbances Are abnormal Q waves present? INFARCT, conduction DEFECTS Is the QRS wide in any of the limb leads? RHYTHM, conduction defects What is the axis (for many purposes it is not necessary to determine degrees)? AXIS, conduction defects Are any of the criteria for LVH met? HYPERTROPHY Is the height of the R waves in V1 ≥ the depth of the S waves? HYPERTROPHY, infarct low voltage?</td>
</tr>
<tr>
<td>ST segments and T waves</td>
<td>Are the ST segments elevated or depressed? ISCHEMIA/infarction, conduction defects, HYPERTROPHY Are the T waves normal in appearance and orientation? ISCHEMIA/infarction, CONDUCTION defects, hypertrophy, electrolyte disturbances</td>
</tr>
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</table>

Systematic Interpretation of ECGs: the P QRS-T Method

It is important to perform all of the steps shown in Table 13.1, but temporary deviation from the pattern is often useful for exploring particular issues. For example, the discovery of negative P waves in lead II should lead to a search for the cause (technical problem, rhythm disturbance, etc.). Once an explanation is found return to the P wave step and continue from there. An advantage of this system is that a list of questions to answer need not be memorized. As one moves systematically forward from P waves to T waves all of the pertinent issues (rhythm, hypertrophy, ischemia/infarct, etc.) will be addressed.

Use the method described above (or any other comprehensive and systematic method) to interpret the cardiograms that follow. Most of these ECGs are complex and would challenge the average physician. They are intentionally complicated in order to review as much as possible and to help instill an appreciation of the value of a systematic approach to ECG
analysis. Most ECGs are not as difficult as these examples; in many areas of clinical practice, it is common to encounter cardiograms devoid of abnormalities that may simply be described as normal sinus rhythm, normal QRS complex configuration, normal ST segments and T waves.

Do not be discouraged if the exact cause of all of the abnormalities is not readily apparent. Particularly when first starting out one of the main skills is to recognize that something is abnormal and seek appropriate guidance. For example, if while working in a stress lab you encounter a resting ECG that you recognize as abnormal but are not sure what the problem is it is usually appropriate to seek guidance before proceeding with the test. As you encounter various abnormalities in the field and discuss cardiograms with colleagues your ECG knowledge will grow.

Figure 13.2 shows a normal sinus rhythm with complete heart block and an idioventricular escape rhythm, left axis deviation.

Rate: The atrial rate is 91 and regular, the ventricular rate is 26 beats per minute (bpm) and regular.

P: The P waves are upright and of normal appearance. Many of the P waves are not followed by QRS complexes, those that are have differing PR intervals (the P-P intervals are consistent). The atrial rates and ventricular rates are regular and seem to have no relationship to each other; the atrial rate is faster than the ventricular rate. The rhythm is complete (third degree) atrioventricular block.

QRS-T: The QRS complexes are wide and bizarre in appearance and have a left bundle branch block morphology (QS in V1, wide R in V6). Given that the rhythm is complete heart block, this likely represents an idioventricular escape rhythm. If one P wave preceded each QRS complex in a normal fashion, then the same QRS morphology would represent left bundle branch block. As the appearance of the QRS is explained by the idioventricular rhythm, this is not a conduction defect, although some clinicians would add the term “with a left bundle branch block morphology” to the interpretation to infer where in the ventricles the idioventricular rhythm is originating. The QS complexes or small r waves and ST segment elevations in the rightward precordial leads are explained by the abnormal conduction of the ventricular beats (analogous to what occurs with left bundle branch block); the QS complexes in leads III and aVF are not due to an old inferior wall infarct (although it would be reasonable for the beginning/intermediate student of ECG to assume that they are) but are associated with the abnormal conduction of the idioventricular rhythm. The axis is left axis deviation (approximately -60); since the altered mean QRS vector is explained
by the ventricular origin of the impulses, this does not imply a left anterior hemiblock.

Figure 13.2

Figure 13.3 shows normal sinus rhythm with frequent premature atrial complexes (PACs) and a ventricular couplet, old inferior wall infarct, and persistent S waves in the lateral precordial leads.

Figure 13.3

Rate: The rhythm is very irregular with an average rate of about 90 bpm.

P: The P waves are upright in lead II, more than one P wave morphology is seen, and some QRS complexes do not appear to be preceded by P waves. This would be a good point to determine why these things are so. It might help to first establish what the normal P wave morphology is. Inspection of the lead II rhythm strip is helpful in establishing the rhythm. Starting with the fifth complex a series of QRS complexes that are regularly occurring and associated with P waves with consistent and normal appearance and consistent PR intervals is seen. This is the underlying rhythm. Having established the normal R-R interval and P wave morphology, it becomes evident that several of the QRS complexes in the rhythm strip appear early. Some of them have narrow QRS complexes that look like the normal QRSs in this lead and are preceded by P waves that do not look like the normal P waves; these are PACs. The second QRS complex in this strip has a narrow QRS, but does not appear to be preceded by a P wave. Careful inspection of the T wave after the first QRS complex shows that the “missing” P wave is sitting on top of this T wave, so this is also a PAC. Toward the middle of the rhythm strip two consecutive, early, wide, and bizarre appearing QRS complexes not preceded by P waves appear; these are premature
ventricular complexes (PVCs), and since two with similar appearance occur consecutively this is a "uniform couplet." Leads I, III, and aVL show an irregular baseline that appears similar to that seen with atrial fibrillation (A-Fib). Combined with the irregularity of this strip, it might seem reasonable to suspect A-Fib; however, given the regular appearance of P waves and the explanation of the rhythm irregularities due to PACs and PVCs it becomes apparent that this is not the rhythm. The baseline irregularities seen in these leads are from artifact produced by poor skin preparation prior to placement of the electrodes or perhaps from patient tremor. The three leads that show this artifact all share the left arm electrode, so it might help to replace this electrode or establish if a left upper extremity tremor is present.

QRS-T: With the exception of the two PVCs, the QRS complexes are of a normal duration. The axis is approximately -30 and the r waves are taller than the s waves are deep in lead II, so the axis may be considered normal. Significant Q waves are seen in the inferior leads. The lateral precordial leads (V5 and V6) show S waves.

Figure 13.4 shows normal sinus rhythm with a second-degree atrioventricular (AV) block with 2:1 conduction, normal QRS configuration, and s waves present in V5 and V6.

Rate: The ventricular rate is slow and regular with a rate of about 35 bpm. The atrial rate is about 70 bpm.

P: Two P waves with consistent morphologies are associated with each QRS complex and the PR intervals of the P waves directly preceding each QRS are consistent.

QRS-T: The QRS complexes are narrow and have normal appearance with the exception of persistent s waves in the lateral precordial leads. The axis is approximately 0 degrees.

Comment: With 2:1 second-degree AV block it is not always possible to discern if it is a Mobitz type I or a Mobitz type II and therefore may simply be called a second-degree 2:1 block. Longer observation may reveal occasional 3:2 block with lengthening PR intervals establishing a diagnosis of Mobitz type I.

Figure 13.5 shows an atrioventricular nodal re-entrant tachycardia, left anterior fascicular block, poor R wave progression, and possible inferolateral wall ischemia.

Rate: The rate is fast and very regular at about 188 bpm.

P: P waves may not be present, or negative P waves may be appearing after the QRS (e.g., lead II). In either case AV nodal re-entrant tachycardia (AVNRT) would be the presumed rhythm. Atrial abnormalities and PR intervals cannot be determined.

QRS-T: No abnormal Q waves are present, the QRS complexes are narrow with septal q waves in leads I and aVL, and the axis is approximately -60 degrees, indicating left anterior hemiblock. Left axis deviation is implied in the diagnosis of the hemiblock. The r waves do not increase in height in a normal fashion from the right to the left chest leads. The ST segments are significantly depressed in leads II, aVF, V5, and V6, and nonsignificant depressions occur in other leads. These depressions may indicate ischemia caused by the increased oxygen demands of the heart associated with the fast rate or may just indicate repolarization abnormalities associated with the fast rate. Some leads (e.g., lead II) appear to have down-sloping ST depression, but this appearance may be due to a negative P wave occurring in the ST segment.
Figure 13.6 shows a normal sinus rhythm and Wolff-Parkinson-White (WPW).

**Rate:** Regular at about 70 bpm.

**P:** The P waves are of modest size, but positive in leads II, III, and aVF. The P waves in lead II may appear to be biphasic, but the negative deflection is actually the beginning of the QRS complex. This can be established using the rhythm strips by comparing where the QRS complex begins in lead V\textsubscript{5} (where it is obvious) with the same time point in the lead II rhythm strip (bottom three lines). Having established where the QRS begins in lead II it becomes apparent that the PR interval is short. A short PR interval should be a signal to look for causes such as WPW.

**QRS-T:** The QRS duration is prolonged and several leads (e.g., I, V\textsubscript{2}) show the distinctive “delta wave” slurring at the beginning of the QRS. This establishes the triad (short PR, wide QRS, delta wave) of WPW. Left axis deviation (-60 degrees) is present, so it might be reasonable to assume a left anterior hemiblock; however, due to the abnormal ventricular activation associated with WPW the usual criteria for hemiblocks cannot be used. Leads III and aVF may appear to have Q waves, but in fact tiny R waves are present. The ST segments are elevated in several leads and the T waves are inverted in leads I and aVL. This is a “pseudoinfarction” associated with WPW, not an actual infarct.

Figure 13.7 shows a normal sinus rhythm, right atrial abnormality, old septal wall infarct, prolonged QT interval, and diffuse nonspecific ST-T abnormalities.

**Rate:** Regular at about 91 bpm.
P: The P waves are taller than 2.5 mm in lead II, indicating right atrial abnormality.

QRS-T: The axis is normal and about +60 degrees. The QT interval appears to be more than half of the R-R interval, indicating possible prolongation of the QT. The rate-corrected QT interval (QTc) is >440 ms (approximately 475 ms), confirming a prolonged QT interval. Significant Q waves are present in V₁ and V₂, indicating an old infarct of the septum. The modest ST segment elevations in the septal leads (V₁ and V₂) are consistent with the diagnosis of old septal wall infarct. Mild T wave inversions are present in numerous leads, but the shape and depth of these inversions is not specific for any particular abnormality within the context of this ECG.

Figure 13.8 shows a normal sinus rhythm, complete right bundle branch block, and old inferior wall infarct.

Rate: Regular at about 62 bpm.

P: The P wave morphology in lead II is normal, and one P wave is associated with each QRS in a normal fashion.

QRS-T: The axis is normal at about +60 degrees. The QRS duration is >120 ms in some of the limb leads. This should instigate a search for left bundle branch block (LBBB) or right bundle branch block (RBBB). An rSR' is present in V₁ and V₆ shows an S wave; therefore, an RBBB pattern is present. Significant Q waves appear in all of the inferior leads, suggesting an old infarct. ST segment depression and T wave inversions are present in the right chest leads secondary to the RBBB.
Figure 13.9 shows sinus tachycardia versus sinoatrial (SA) nodal re-entrant tachycardia versus ectopic atrial tachycardia, complete left bundle branch block.

Rate: Very regular at about 136 bpm.

P: The P wave morphology in lead II and the other inferior leads is difficult to discern as the P waves appear to be merging with the preceding T waves; P waves are clearly visible in V1 and have a normal relationship to the QRS complexes. The very regular rhythm and the rate would favor a diagnosis of SA nodal re-entrant tachycardias instead of sinus tachycardia (although it could be a sinus tachycardia). If the P waves are not positive in the inferior leads (difficult to tell in this ECG), ectopic atrial tachycardia is favored.

QRS-T: The axis shows left axis deviation (LAD) at about -60 degrees. The QRS duration is >120 ms in some of the limb leads. This should instigate a search for LBBB or RBBB. A wide R is present in V6 and a QS pattern is seen in V1; therefore, an LBBB pattern is present. ST segment elevations are present in the right chest leads secondary to the LBBB.

Figure 13.10 shows a junctional escape rhythm (ectopic atrial bradycardia) and normal QRS complex configuration.

Rate: Regular at about 52 bpm.

P: The P waves are negative in the inferior leads, implying a junctional rhythm.

QRS-T: The axis is normal at about +30 degrees. The QRS complexes are normal in amplitude, duration, and appearance. The ST segments and T waves are normal.

Figure 13.11 shows atrial fibrillation with moderate ventricular response and acute inferior and anterolateral wall infarct.

Rate: Very irregular at about 90 bpm.

P: Regularly occurring, organized atrial activity is not present, and the ventricular rhythm is grossly irregular.

QRS-T: The axis is normal at about +30 degrees. Significant Q waves are present in the inferior leads. The ST segments are significantly elevated in the inferior leads and V3 to V6, indicating an acute STEMI; the ST segment is significantly depressed in aVL likely due to remote ischemia.

Figure 13.12 shows a normal sinus rhythm with first-degree AV block and frequent premature atrial complexes sometimes occurring in a bigeminal pattern, biatrial abnormality, left anterior fascicular block, and evolving ST segment elevation infarct or ischemia.

Rate: The rate is normal (77 bpm); some beats occur early. What is causing the early beats can be explored here. Note that the P wave morphologies of the early beats are different from the norm (this is particularly evident in lead V1), these beats are PACs. From the middle to the end of the rhythm strip PACs are occurring every other beat, thus atrial bigeminy is present at that time.
The P waves are abnormally tall and wide in lead II (and the negative portion of the P waves in lead V₁ is a “box wide and box deep”), so criteria for left atrial abnormality (LAA) and right atrial abnormality (RAA) are both met (biatrial abnormality). The PR interval is >200 ms, indicating first-degree AV block. As previously noted, early beats are preceded by P waves with different morphologies from the other beats.

**QRS-T:** The axis is about -90 degrees, indicating left anterior fascicular block. An rs' pattern is present in V₁, and V₆ has s waves; this can be noted but as the QRS duration is not prolonged in the limb leads an RBBB is not present. Some mild ST segment elevations are present and the shape of the ST segments and the T wave inversions in leads V₂ to V₆ suggest an evolving STEMI; but usually Q waves would have developed by this point (and have not) so the T wave inversions could indicate ischemia/non-ST segment elevation myocardial infarction (NSTEMI). This may be confusing as the designation NSTEMI pretty clearly indicates that ST elevation should not be present; this case is one of the exceptions. For the physiologist the important point is that ischemia/infarction of some type may be occurring.

**Quiz 13**

Describe the rhythm shown in the following strips.

1.