ECG tutorial: Basic principles of ECG analysis

AUTHOR: Jordan M Prutkin, MD, MHA, MHS
SECTION EDITOR: Ary L Goldberger, MD
DEPUTY EDITOR: Naomi F Botkin, MD

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INTRODUCTION

Even though there continues to be new technologies developed for the diagnostic evaluation of patients with cardiovascular disease, the electrocardiogram (ECG) retains its central role. The ECG is the most important test for interpretation of the cardiac rhythm, conduction system abnormalities, and the detection of myocardial ischemia. The ECG is also of great value in the evaluation of other types of cardiac abnormalities including valvular heart disease, cardiomyopathy, pericarditis, and hypertensive disease. Finally, the ECG can be used to monitor drug treatment (specifically antiarrhythmic therapy) and to detect metabolic disturbances.

A systematic approach systematic approach to interpretation of the ECG is imperative. Pattern recognition is helpful, but it is important to review all aspects of the ECG to avoid overlooking abnormalities. This topic review provides the framework for a systematic analysis of the ECG.

ECG GRID

The ECG is a plot of voltage on the vertical axis against time on the horizontal axis. The electrodes are connected to a galvanometer that records a potential difference. The needle (or pen) of the ECG is deflected a given distance depending upon the voltage measured.

The ECG waves are recorded on special graph paper that is divided into 1 mm^2 grid-like boxes (figure 1). The ECG paper speed is ordinarily 25 mm/s. As a result, each 1 mm (small) horizontal box corresponds to 0.04 s (40 ms), with heavier lines forming larger boxes that include five small boxes and hence represent 0.20 s (200 ms) intervals. On occasion, the paper speed is increased to 50 mm/s to better define waveforms. In this situation, there are typically only six leads per sheet of paper. Each large box is therefore only 0.10 s and each small box is only 0.02 s. In addition, the heart rate appears

to be one-half of what is recorded at 25 mm/s paper speed, and all the ECG intervals are twice as long as normal. Other paper speeds are occasionally used.

Vertically, the ECG graph measures the height (amplitude) of a given wave or deflection. The standard calibration is 10 mm (10 small boxes), equal to 1 mV. On occasion, particularly when the waveforms are small, double standard is used (20 mm equals 1 mv). When the wave forms are very large, half standard may be used (5 mm equals 1 mv). Paper speed and voltage are usually printed on the bottom of the ECG for reference.

COMPLEXES AND INTERVALS

The normal ECG is composed of several different waveforms that represent electrical events during each cardiac cycle in various parts of the heart (figure 2). ECG waves are labeled alphabetically starting with the P wave, followed by the QRS complex and the ST-T-U complex (ST segment, T wave, and U wave). The J point is the junction between the end of the QRS and the beginning of the ST segment (waveform 1).

P wave — The P wave represents atrial depolarization. The normal sinus P wave demonstrates depolarization from the right to left atrium and is an initial low amplitude deflection preceding the QRS complex that is positive in most leads. The duration is generally <0.12 s (three small boxes) and the amplitude <0.25 mv (2.5 small boxes). Since right atrial depolarization precedes that of the left atrium (as the sinus node is in the high right atrium), the P wave is often notched in the limb leads and usually biphasic in lead V1. The initial positive deflection in V1 is due to right atrial depolarization that is directed anteriorly, while the second negative deflection represents left atrial depolarization that is directed posteriorly.

The atrial repolarization sequence (atrial ST and T wave phases) occurs just before, simultaneously, and just after depolarization of the ventricular myocardium. The atrial "T wave" itself is usually hidden by the QRS complex and not observed on the routine ECG. In addition, the amplitude of the atrial T wave is usually too small to be observed at standard gain. When the heart rate is increased (eg, with sinus tachycardia) and there is enhanced sympathetic tone, the PR interval is shortened; atrial repolarization (the atrial T wave) may sometimes then be observed at the very end of the QRS complex, altering the J point, and resulting in J point depression with rapidly upsloping ST segments, particularly during the first 80 ms after the QRS complex. This finding is physiologic but may be confused with true ST depression, generating a false positive reading. Clinically, atrial repolarization (the atrial ST phase) is most evident during acute pericarditis, in which one often sees PR segment elevation in lead aVR and PR segment depression in the infero-lateral leads, reflecting an atrial current of injury. The low amplitude atrial T wave may also be unmasked in certain cases of high degree AV block, especially when the atria are enlarged. Finally, alterations in the atrial ST segment and T wave may occur with other pathologies, such as atrial infarction or atrial tumor invasion.

PR interval — The PR interval includes the P wave as well as the PR segment. It is measured from the beginning of the P wave to the first part of the QRS complex (which may be a Q wave or R wave). It includes time for atrial depolarization (the P wave) and conduction through the AV node and the His-Purkinje system (which constitute the PR segment). The length of the PR interval changes with heart rate but is normally 0.12 to 0.20 s (three to five small boxes). The PR interval is shorter at faster heart rates due to sympathetically mediated enhancement of atrioventricular (AV) nodal conduction. It is longer when the rate is slowed as a consequence of slower AV nodal conduction resulting from withdrawal of sympathetic tone or an increase in vagal input.

QRS complex — The QRS complex represents the time for ventricular depolarization.

- If the initial deflection is negative, it is termed a Q wave. Small Q waves are often seen in leads I, aVL, and V4-V6 as a result of initial septal depolarization and are considered normal.
- The first positive deflection of the QRS complex is called the R wave. It represents depolarization of the left ventricular myocardium. Right ventricular depolarization is obscured because the left ventricular myocardial mass is much greater than that of the right ventricle. The small R wave in lead V1 represents initial septal depolarization.
- The negative deflection following the R wave is the S wave, which represents terminal depolarization of the high lateral wall.
- If there is a second positive deflection, it is known as an R'.
- Lower case letters (q, r, or s) are used for relatively small amplitude waves of less than 0.5 mV (less than 5 mm with standard calibration).
- An entirely negative QRS complex is called a QS wave.

The entire QRS duration normally lasts for 0.06 to 0.10 s (1.5 to 2.5 small boxes) and is not influenced by heart rate.

The R wave should progress in size across the precordial leads V1-V6. Normally there is a small R wave in lead V1 with a deep S wave. The R wave amplitude should increase in size until V4-V6 while the S wave becomes less deep. This is termed R wave progression across the precordium.

ST segment — The ST segment occurs after ventricular depolarization has ended and before repolarization has begun. It is a time of electrocardiographic silence. The intersection of the end of the QRS complex and the initial part of the ST segment is termed the J point (waveform 1).

The ST segment is usually isoelectric (ie, zero potential as identified by the T-P segment) with a slight upward concavity. However, it may have other configurations depending upon associated disease states (eg, ischemia, acute myocardial infarction, left ventricular hypertrophy, or pericarditis). In these situations, the ST segment may be flattened, depressed (below the isoelectric line) with an upsloping,

horizontal, or downsloping morphology, or elevated in a concave or convex direction (above the isoelectric line). (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "ECG tutorial: ST- and T-wave changes" and "Acute pericarditis: Clinical presentation and diagnosis", section on 'Electrocardiogram'.)

In some normal cases (as with sinus tachycardia) the J point is depressed and the ST segment is rapidly upsloping, becoming isoelectric within 0.08 s after the end of the QRS complex.

T wave — The T wave represents the period of ventricular repolarization. Since the rate of repolarization is slower than depolarization, the T wave is broad, has a slow upstroke, and a more rapid downslope to the isoelectric line following its peak. Thus, the T wave is asymmetric and the amplitude is variable. In addition, the T wave is usually smooth up and down. If there is any irregularity on the T wave (bump, notch, rippled, etc) a superimposed P wave should be considered.

Since depolarization begins at the endocardial surface and spreads to the epicardium, while repolarization begins at the epicardial surface and spreads to the endocardium, the direction of ventricular depolarization is opposite to that of ventricular repolarization. Thus, the T wave vector on the ECG normally is in the same direction as the major deflection of the QRS. Another way of saying this is that the QRS and T wave axes are generally concordant. Various disease states can lead to T wave discordance. (See "ECG tutorial: ST- and T-wave changes".)

QT interval — The QT interval consists of the QRS complex, the ST segment, and T wave. Thus, the QT interval is primarily a measure of ventricular repolarization. The JT interval, which does not include the QRS complex, is a more accurate measure of ventricular repolarization since it does not include ventricular depolarization, but in most clinical situations, the QT interval is used. If the QRS complex duration is increased, this will lead to an increase in QT interval but does not reflect a change in ventricular repolarization. A widened QRS, therefore, must be considered if a prolonged QT interval is being evaluated.

The QT (or JT) interval is dependent upon the heart rate. It is shorter at faster heart rates and longer when the rate is slower. Thus, a QT interval that is corrected for heart rate (QTc) has been classically calculated based on Bazett's widely used formula (calculator 1):

QTc = QT interval / square root of the RR interval (both measured in seconds)

Although this approach is simple, it is inaccurate at heart rate extremes and results in overcorrecting at high rates and under correcting at low ones [1].

Another method (Fridericia) corrects the QT interval by the cubed root of the RR interval [1,2]. Linear (eg, Hodges and Framingham algorithms) as well as logarithmic regression formulas have also been proposed to predict ("correct") the effect of heart rate on QT interval [3,4]. However, because of substantial variability of the QT-RR relationship among individuals, no single formula for QT correction

based on heart rate has been universally adapted [5,6]. The QT calculator allows comparison of the results of QT correction using the Bazett's square root formula and those due to Fridericia, Framingham, and Hodges (using heart rate in beats per minute [bpm] or based on RR intervals in milliseconds) (calculator 1). The corresponding formulas for these corrections are given with the calculator.

The upper normal value for the QTc in men is usually given as about ≤440 ms and in females as about ≤450 to 460 ms. However, the topic is further complicated because of intrasubject variability in QTc intervals during the course of the day and intraobserver variability in visually or electronically identifying the end of the T wave. Furthermore, even subjects with congenital long QT syndromes may have intermittently less prolonged or even intermittently normal QTc values. The lower limits of the QTc are less well defined, including normal variants and rare individuals with the congenital short QT syndrome. (See "Congenital long QT syndrome: Diagnosis".)

Clinicians also need to be mindful of the fact that since the QRS widens in the setting of a bundle branch block, the QT interval will also increase. The larger QT interval does not reflect an abnormality of ventricular repolarization, since the increase is due to an abnormality of depolarization. There have not been many descriptions on how to measure QT interval in the setting of QRS widening. One study showed that the QT increased 48.5 percent of the width of the QRS due to a left bundle branch block, and proposed a rough formula of $QT_{modified} = QT_{measured} - 0.5 \times QRS_{measured}$) to calculate the QT interval [7]. This must be still be corrected for heart rate. Another option is to measure the JT interval, corrected for rate: QTc – QRS = JTc [8]. This equation has some limitations, as it is dependent on heart rate and as normal values have not been derived. Other, more complicated models have been created for correcting QTc with ventricular pacing [9].

U wave — A U wave may be seen in some leads, especially the precordial leads V2 to V4. The exact cause of this wave is uncertain, though data suggest it may be from late repolarization of the mid-myocardial M cells, due to a longer action potential duration compared with the endocardium or epicardium, especially at slow heart rates [10].

The amplitude of the U wave is typically less than 0.2 mV and is clearly separate from the T wave. It is more evident in some circumstances such as hypokalemia and bradycardia. The U wave may merge with the T wave when the QT interval is prolonged (a QT-U wave) or may become obvious when the QT or JT interval is shortened (eg, with digoxin or hypercalcemia).

HEART RATE

If the cardiac rhythm is regular, the interval between successive QRS complexes determined from the ECG grid can be used to determine heart rate.

- The division of 300 by the number of large boxes calculates the heart rate. If the interval between two successive complexes is one large box, then the rate is 300 bpm (300 / 1 = 300 bpm). If the

interval is two large boxes, the rate is 150 (300 / 2 = 150 bpm). This calculation may be carried on down the line for each additional large box, to 100 bpm, 75 bpm, 60 bpm, 50 bpm, etc.

- Alternatively, the time between QRS complexes can be measured in seconds. This number can be divided into 60 to derive the heart rate. For instance, if the time between two QRS complexes is 0.75 s, the heart rate is 80 bpm (60 s/min / 0.75 s/beat = 80 bpm).

If the rhythm is irregular, the simplest way to determine the rate is by counting the number of complexes on the ECG and multiplying by six, since the standard ECG displays 10 s of time.

A rate of 60 to 100 bpm is considered normal. A rate less than 60 bpm is bradycardia (though some use 50 bpm), while a rate over 100 bpm is tachycardia (algorithm 1A-B).

AXIS

The electrical signal recorded on the ECG contains information relative to direction and magnitude of the various complexes. The average direction of any of the complexes can be determined.

The normal QRS electrical axis, as established in the frontal plane, is between -30 and 90° (directed downward or inferior and to the left) in adults [11]. An axis between -30° and -90° (directed superior and to the left) is termed left axis deviation. If the axis is between 90° and 180° (directed inferior and to the right), then right axis deviation is present. An axis between -90° and -180° (directed superior and to the right) is referred to as extreme right or extreme left axis. If the QRS is equiphasic in all leads with no dominant QRS deflection, it is indeterminate axis. The QRS axis moves leftward throughout childhood and adolescence, from a normal value of 30 to 190° at birth to 0 to 120° during ages 8 to 16 years. There is some disagreement among authors on the definitions (in degrees) of a normal, right, and left axis. (See "Left anterior fascicular block" and "Left posterior fascicular block".)

The QRS axis can be determined by examining all of the limb leads, but the easiest method involves looking at leads I, II, and aVF only (figure 3).

- If the QRS complex is positive (upright) in both leads I and II, then the axis falls between -30 and 90°, and the axis is normal.
- If the QRS complex is positive in lead I but negative in lead II, then the axis is leftward (-30 to -90°).
- If the complexes are negative in lead I and positive in aVF, then the axis is rightward (90 to 180°).
- If the complexes are negative in both I and aVF, then the axis is extreme (180 to -90°).

Another method of axis determination is to find the lead in which the complex is most isoelectric. The axis is directed perpendicular to this lead. As an example, if the QRS is isoelectric in lead III which is directed at 120°, then the electrical axis is either 30° or -150°.

A third method is to determine the frontal lead in which the QRS is of the greatest positive amplitude. The axis is parallel to this lead.

By combining the quadrant determined by analysis of leads I, II, and aVF with the isoelectric lead information, one can accurately and rapidly determine the electrical axis.

The causes of right axis deviation include:

- Normal variation (vertical heart with an axis of 90°)
- Mechanical shifts, such as inspiration and emphysema
- Right ventricular hypertrophy
- Right bundle branch block
- Left posterior fascicular block
- Dextrocardia
- Ventricular ectopic rhythms
- Preexcitation syndrome (Wolff-Parkinson-White)
- Lateral wall myocardial infarction
- Secundum atrial septal defect

Causes for left axis deviation include:

- Normal variation (physiologic, often with age)
- Mechanical shifts, such as expiration, high diaphragm (pregnancy, ascites, abdominal tumor)
- Left ventricular hypertrophy
- Left bundle branch block
- Left anterior fascicular block
- Congenital heart disease (primum atrial septal defect, endocardial cushion defect)
- Emphysema
- Hyperkalemia
- Ventricular ectopic rhythms
- Preexcitation syndromes (Wolff-Parkinson-White)
- Inferior wall myocardial infarction.

The heart also has an axis in the horizontal plane, which is determined by imagining the heart as viewed from under the diaphragm. If the axis is rotated in a clockwise direction, left ventricular forces are directed more posteriorly and occur later in the precordial leads. This is termed poor R wave progression or late transition. If there is counterclockwise rotation, left ventricular forces occur earlier in the right precordial leads and this is termed early transition in which there is a tall R wave in lead V2.

There is no agreement on how to estimate the QRS axis in patients with bundle branch block (BBB). As the prolonged terminal part of the QRS in right bundle branch block reflects delays in right ventricular activation, and axis determination is of importance in diagnosing fascicular blocks, one reasonable approach is to estimate the frontal plane QRS axis based on just the first 80 to 100 ms of the QRS

deflection (primarily reflecting activation of the left ventricle). For left bundle branch block and other intraventricular conduction delays, the entire QRS can be used or just the initial 80 to 100 ms.

APPROACH TO ECG INTERPRETATION

A systematic approach to interpreting an ECG is essential.

Step 1: Rate — Is the rate between 60 and 100 bpm? Rates less than 60 bpm are bradycardic (some use 50 bpm) and greater than 100 bpm are tachycardic.

Step 2: Rhythm — Are P waves present? Is there a P wave before every QRS complex and a QRS complex after every P wave? Are the P waves and QRS complexes regular? Is the PR interval constant? (See 'Rhythm analysis' below.)

Step 3: Axis — Is there left or right axis deviation? (See 'Axis' above.)

Step 4: Intervals — What is the PR interval? Short PR intervals may suggest of Wolff-Parkinson-White syndrome. Long PR intervals are usually seen in first degree AV block, but there may be other causes. What is the QRS interval? Long QRS intervals represent a bundle branch block, ventricular pre-excitation, ventricular pacing, or ventricular tachycardia. What is the QT interval? Short and long QT intervals may be present.

Step 5: P wave — What is the shape and axis of the P wave? The P wave morphology should be examined to determine if the rhythm is sinus or from another atrial location. (See 'P wave' above.) Amplitude and duration should also be analyzed to determine left and right atrial enlargement. (See "Normal sinus rhythm and sinus arrhythmia".)

Step 6: QRS complex — Is the QRS wide? If so, examination of the morphology can determine if there is left or right bundle branch block or pre-excitation present. In addition, increased voltage may indicate left or right ventricular hypertrophy. Are Q waves present, suggestive of infarction? Is there QRS notching that could represent myocardial disease?

Step 7: ST segment-T wave — Is there ST elevation or depression compared with the TP segment? The TP segment, between the T wave of one beat and the P wave of the next beat, should be used as the baseline. Are the T waves inverted (see "ECG tutorial: ST- and T-wave changes")? Abnormalities of the ST segment or T wave may represent myocardial ischemia or infarction, among other causes.

Step 8: Overall interpretation — Only after the prior steps have been completed should an overall description be given, followed by an interpretation and possible diagnoses. For instance, the description may state that the rate is rapid and irregular with no P waves and ST elevation in leads II, III, and aVF with ST depression in leads I, aVL, and V4-6. The interpretation would be that there is rapid atrial fibrillation and an inferior ST-elevation myocardial infarction with reciprocal ST depressions. This ensures assimilation of all information in the ECG and that no detail will be overlooked.

RHYTHM ANALYSIS

Interpreting the rhythm of the ECG similarly should proceed with a systematic approach. Calipers are extremely helpful for rhythm analysis.

Step 1: Locate the P wave — The most important and first step in rhythm interpretation is the identification of P waves and an analysis of their morphology. There are several questions that should be addressed:

- Are P waves visible? Each lead needs to be examined for P waves, as they may not be obvious in some leads. On occasion, P waves may be located on or at the end of T waves and not clearly seen. They will therefore cause the T wave upslope or downstroke to no longer be smooth. It is also important to look for P waves during any pause in the rhythm, such as after a premature ventricular complex/contraction (PVC; also referred to as premature ventricular beats or premature ventricular depolarizations). Absence of P waves may occur secondary to atrial fibrillation. Alternatively, P waves may be present but not visible if they are simultaneous with and buried within the QRS complex as in a junctional rhythm or atrioventricular (AV) nodal re-entrant tachycardia. In addition, they may be located within the ST segment as with an AV reciprocating tachycardia or ventricular tachycardia. If a P wave is halfway between two QRS complexes, a second P wave may be buried within the QRS complex.

- What is the rate of the P waves (ie, the PP interval)? If the rate is less than 60, then a bradycardia is present, though some suggest 50. If the atrial or P wave rate is over 100, then a tachycardia is present. In general, sinus tachycardia occurs at rates of 100 to 180. Atrial tachycardia, AV nodal re-entrant tachycardia, or AV reciprocating tachycardia usually occur at rates of 130 to 220. Atrial flutter has a wide possible range of atrial rates of 150 to 320, and the corresponding ventricular rates are typically the atrial rate divided by an integer between 1 and 8.

- What is the morphology and axis of the P waves? The normal sinus P wave is generally upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. It may be negative or biphasic in leads III and V1. A negative P wave in the inferior leads or lead I suggests an ectopic rhythm (low atrial or left atrial, respectively). Similarly, a completely positive P wave in V1 suggests a left atrial location.

Step 2: Establish the relationship between P waves and the QRS complex — The next step is to determine the relationship between the P waves and the QRS complexes, addressing the following questions:

- Are the P waves associated with QRS complexes in a 1:1 fashion? If not, are there more or fewer P waves than QRS complexes and what are the atrial and ventricular rates? If there are more P waves than QRS complexes, then some form of AV block is present, which may be physiologic if there is a concomitant atrial tachycardia or flutter. If there are more QRS complexes than P waves, then the rhythm originates in the AV node, His-Purkinje system, or ventricles.

- Do the P waves precede each QRS complex as is the case with most normal rhythms? What is the PR interval, and is this interval fixed?

- Do P waves occur after each QRS complex (ie, retrograde P waves) as occurs in junctional or ventricular rhythms, AV nodal reentrant tachycardia, and AV reciprocating tachycardias? The RP interval should be noted and it should be established if it is fixed or variable.

Often, establishing the relationship between the P wave and the QRS complex is the most important diagnostic step in rhythm interpretation. (See 'Overall approach to rhythm analysis' below and "Wide QRS complex tachycardias: Approach to the diagnosis".)

Step 3: Analyze the QRS morphology — If the QRS complexes are of normal duration (<0.12 s) and morphology, then the rhythm is supraventricular. It is essential to analyze the QRS in all 12 leads to be sure that it is normal.

If the QRS is wide (ie, >0.12 s), then the rhythm is supraventricular with aberrant conduction, has pre-excitation, has ventricular pacing, or is of ventricular origin. It may be possible to differentiate them by careful inspection of the QRS morphology, especially if the QRS morphology appears similar to the baseline QRS. (See "Wide QRS complex tachycardias: Approach to the diagnosis" and "Basic approach to delayed intraventricular conduction".)

Step 4: Search for other clues — Often the diagnosis of a rhythm disturbance can be made by clues provided by breaks in the rhythm or other irregularities in an otherwise regular rhythm. As an example, an increase in the degree of AV block as occurs with carotid sinus massage may unmask atrial flutter waves.

Capture beats and fusion beats may be the clues that help establish the diagnosis of ventricular tachycardia.

The regularity of the QRS complexes should be established by asking the following questions:

- Do the QRS complexes occur at regular intervals or are they irregular?

- If the complexes are irregular, is there a pattern to the irregularity? Is the rhythm regularly irregular (ie, there is a repeating pattern of irregularity) or is the rhythm irregularly irregular? At least five supraventricular rhythms are irregularly irregular: sinus arrhythmia (in which there is only one P wave morphology and a stable PR interval); sinus rhythm with premature atrial complex (PAC; also referred to a premature atrial beat, premature supraventricular complex, or premature supraventricular beat); sinus or other rhythm with variable AV block; multifocal atrial rhythm (wandering atrial pacemaker) when the rate is <100 or multifocal atrial tachycardia with a rate >100 (in which there are ≥3 different P wave morphologies and PR intervals); or atrial fibrillation (in which there is disorganized electrical conduction to the ventricles).

Step 5: Interpret the rhythm in the clinical setting — Often, the clinical history, including drugs being taken, can be helpful in establishing a diagnosis. As an example, a regular wide complex rhythm in an older patient with a history of ischemic cardiomyopathy is most likely ventricular tachycardia (see "Wide QRS complex tachycardias: Approach to the diagnosis"). Similarly, a narrow complex tachycardia of sudden onset in a young person with no medical history is likely AV nodal re-entrant or AV reciprocating tachycardia. (See "Narrow QRS complex tachycardias: Clinical manifestations and evaluation of the electrocardiogram".)

However, the clinical presentation and associated hemodynamic findings are not perfectly correlated with the etiology of an abnormal rhythm. The presence of hemodynamic stability during a tachycardia, for example, does not imply a supraventricular etiology, nor does instability mean that the diagnosis is ventricular tachycardia. Hemodynamic changes are related to the rate of the arrhythmia and the presence and extent of underlying heart disease.

OVERALL APPROACH TO RHYTHM ANALYSIS

Rhythm analysis with a standard method, as shown in the prior algorithms, permits the correct diagnosis to be established in most circumstances. An approach to the diagnosis of tachycardia and bradycardia is shown (algorithm 2A-B and algorithm 1A-B). This issue is discussed in other ECG tutorials. (See "ECG tutorial: Ventricular arrhythmias" and "ECG tutorial: Atrial and atrioventricular nodal (supraventricular) arrhythmias" and "ECG tutorial: Rhythms and arrhythmias of the sinus node".)

SUMMARY

- **Complexes and intervals** – The electrical activity of each normal cardiac cycle is represented in sequence by the P wave, the PR interval, the QRS complex, the ST segment, the T wave, and (sometimes) the U wave. The following pieces of information should be evaluated for each of these.
- **Systemic approach to interpretation** – A systematic approach to interpretation of the ECG is critically important. We sequentially evaluate each of the following findings before making a final interpretation of the ECG (see 'Approach to ECG interpretation' above):
  - **Rate** – Is the rate between 60 and 100? (See 'Step 1: Rate' above.)
  - **Rhythm** – Is it normal sinus or other? (See 'Step 2: Rhythm' above and 'Rhythm analysis' above.)
  - **Axis** – Is there axis deviation? (See 'Step 3: Axis' above.)
  - **Intervals** – Are all intervals normal? (See 'Step 4: Intervals' above.)
  - **P wave** – What is its height, width, and axis? (See 'Step 5: P wave' above.)

- **QRS complex** – Are there pathologic Q waves, bundle branch block, or chamber hypertrophy? (See 'Step 6: QRS complex' above.)
- **ST-T waves** – Is it isoelectric, elevated, or depressed relative to the TP segment? (See 'Step 7: ST segment-T wave' above.)
- **Overall interpretation** – What is the diagnosis? (See 'Step 8: Overall interpretation' above.)

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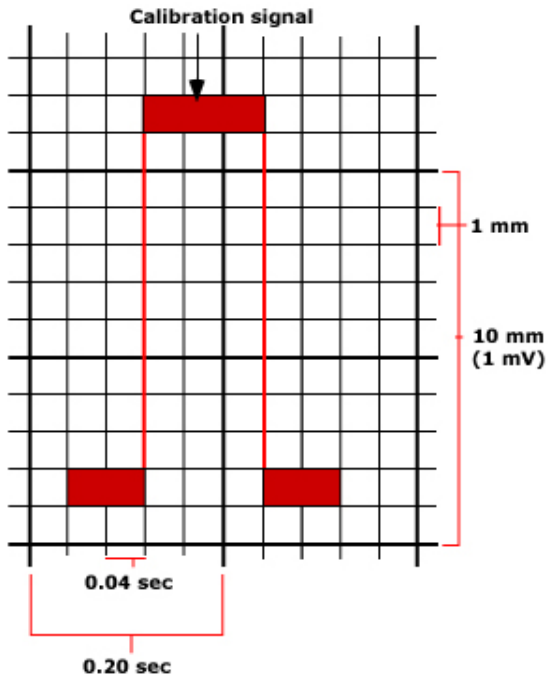
Topic 2115 Version 36.0

GRAPHICS

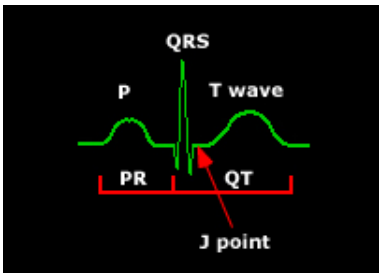
Figure 1: **Grid lines and standardization of the ECG**



The electrocardiogam is recorded on paper that has large boxes (heavy lines) of 0.5 cm sides. On the horizontal axis, each large box, which represents 0.2 seconds at a typical paper speed of 25mm/sec, is divided into five smaller boxes, each one representing 0.04 seconds. On the vertical axis, the large box also has five subdivisions, each 1 mm in height; 10 mm equals 1 mV with standard calibration.

Graphic 62799 Version 1.0

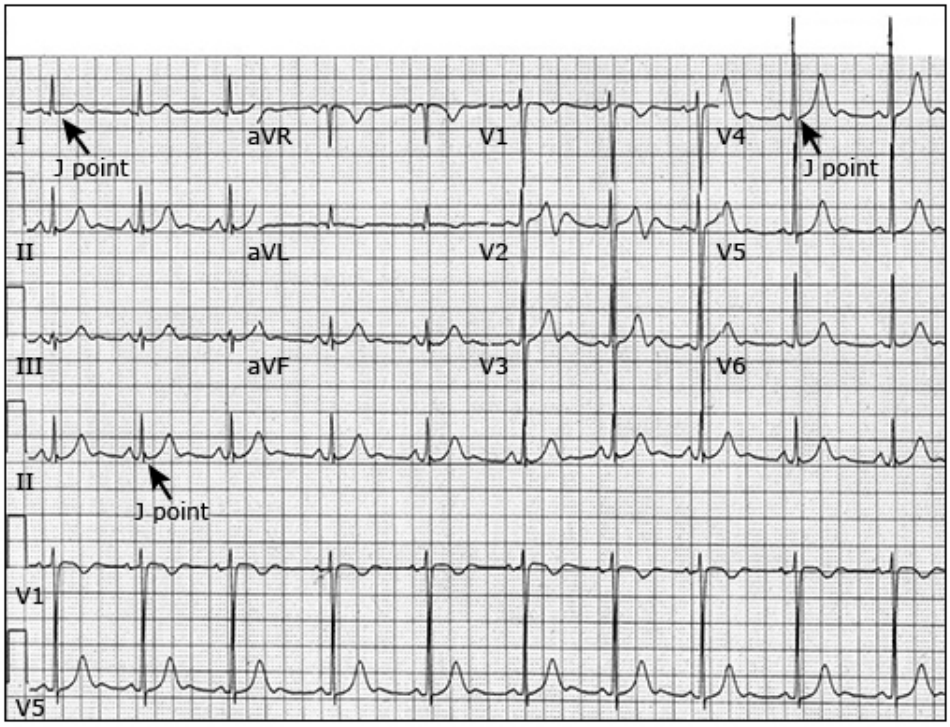
Figure 2: **ECG complexes and intervals**



ECG waves are labeled alphabetically starting with the P wave, followed by the QRS complex, and the ST-T complex (ST segment and T wave). The J point is the junction between the end of the QRS and the beginning of the ST segment. The PR interval is measured from the beginning of the P wave to the first part of the QRS complex. The QT interval consists of the QRS complex which represents only a brief part of the interval, and the ST segment and T wave which are of longer duration.

Graphic 67069 Version 1.0

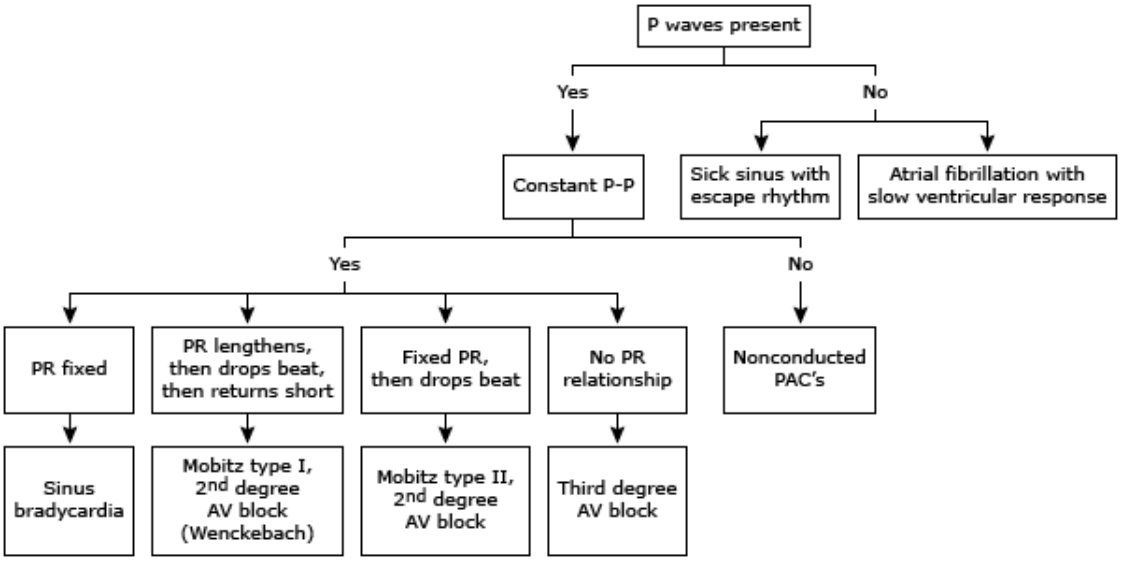
Waveform 1: **J point**



The J point is the junction between the end of the QRS and the beginning of the ST segment.

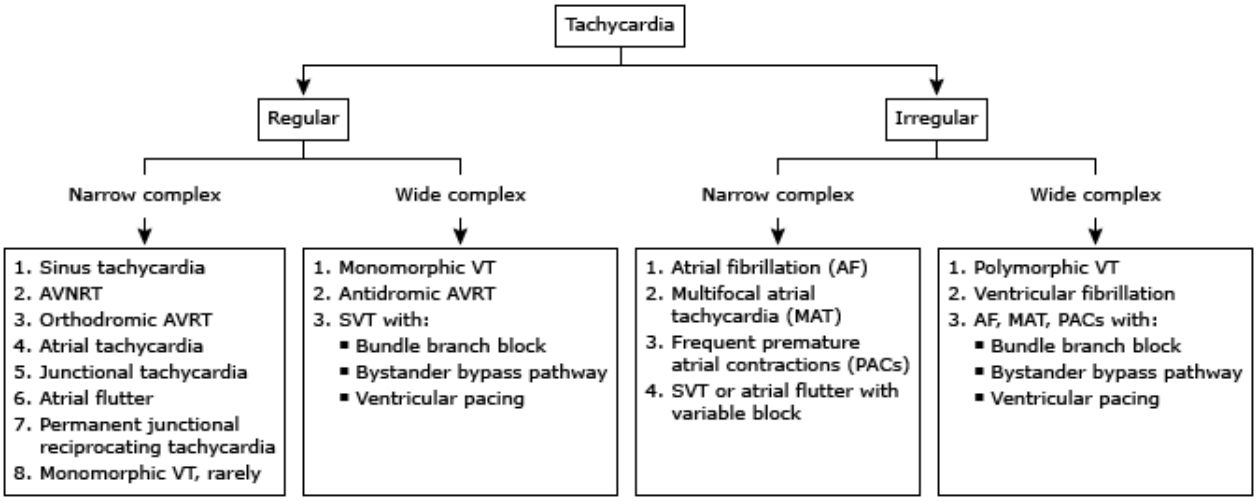
Graphic 82922 Version 2.0

Algorithm 1A: **Approach to bradycardia**



Graphic 85685 Version 3.0

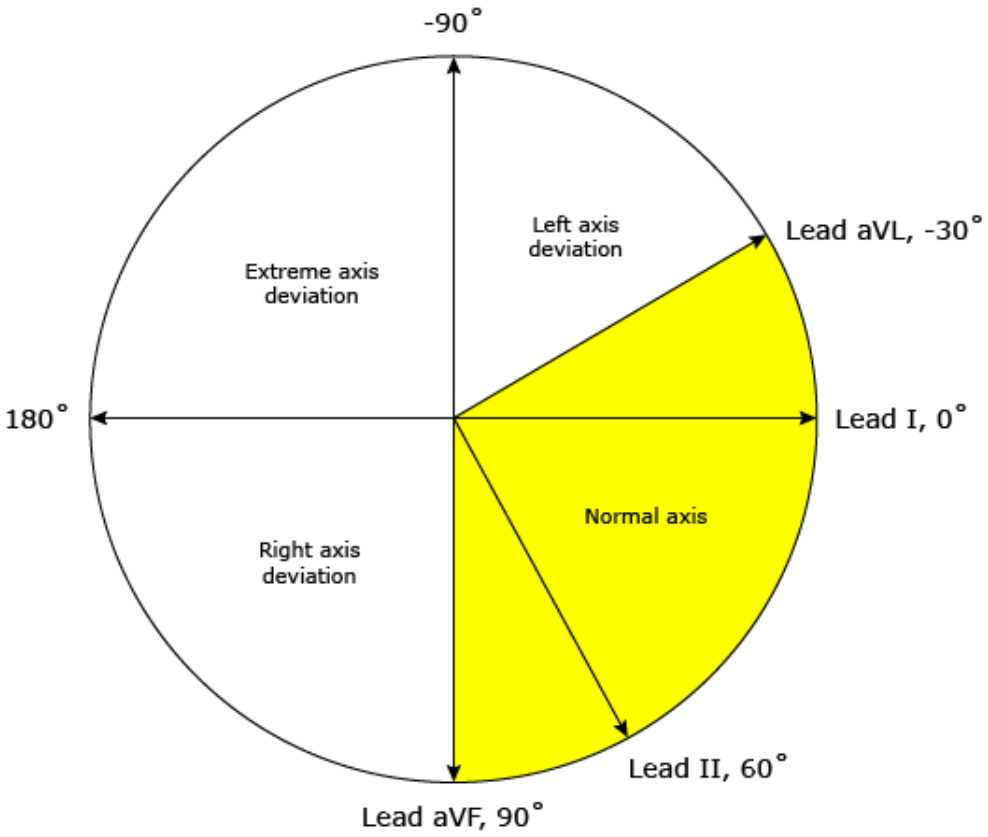
Algorithm 1B: **Approach to tachycardia**



AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reentrant tachycardia; VT: ventricular tachycardia; SVT: supraventricular tachycardia.

Graphic 85684 Version 3.0

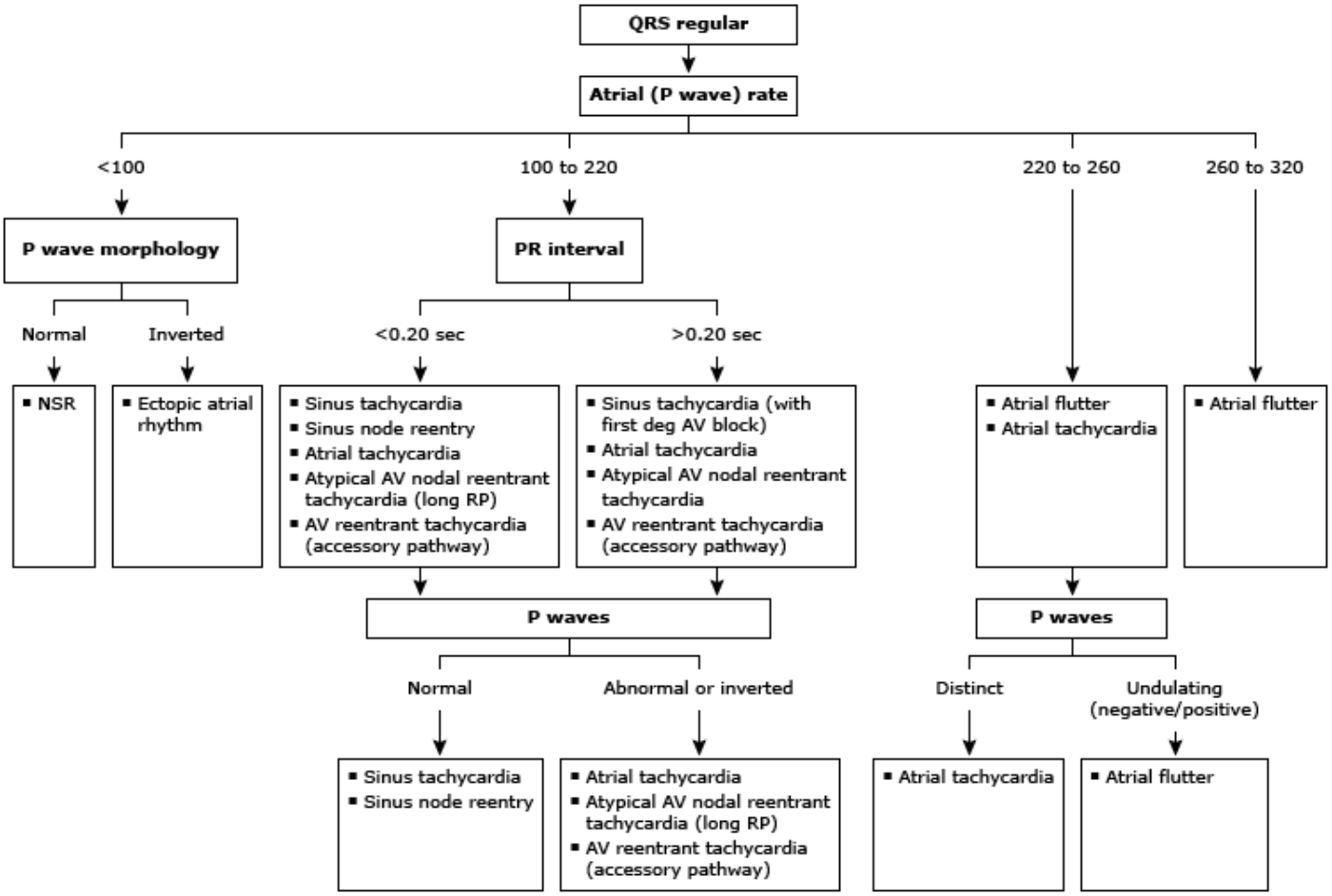
Figure 3: **Calculation of frontal plane axis**



If the QRS complex is positive in leads I and II, it falls between -30 and 90° and is normal, as indicated by the yellow area. If the QRS complex is negative in I and positive in aVF, there is right axis deviation. If the QRS complex is positive in I and negative in II, there is left axis deviation. If the QRS complex is negative in I and aVF, there is extreme axis deviation.

Graphic 85682 Version 1.0

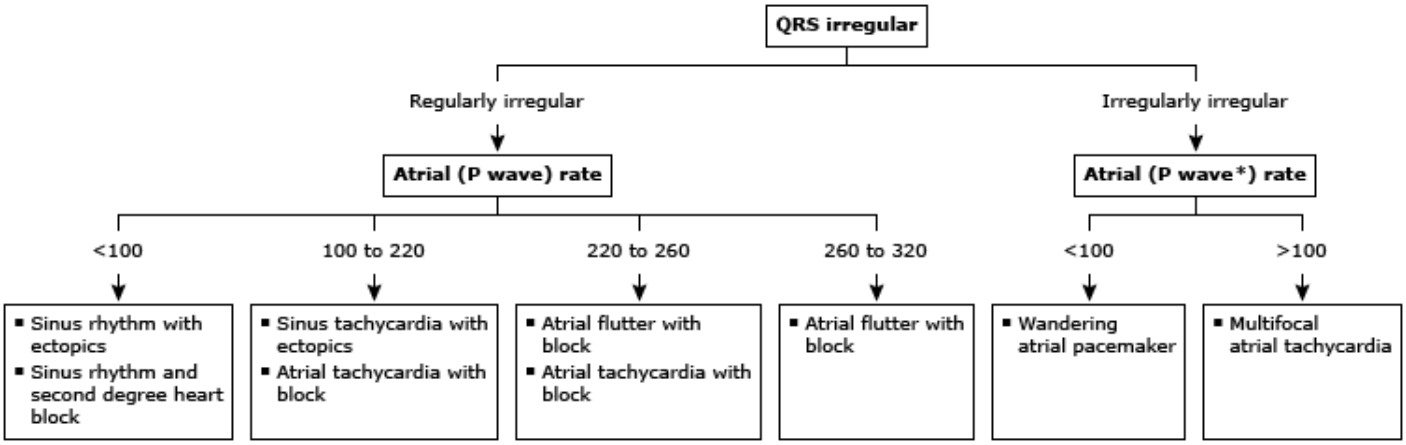
Algorithm 2A: **P wave before each QRS complex with constant PR relationship**



NSR: normal sinus rhythm; AV: atrioventricular.

Graphic 77276 Version 3.0

Algorithm 2B: **P wave in front of each QRS complex: P wave and QRS related**



* P wave morphology and PR interval variable.

Graphic 52259 Version 3.0

Contributor Disclosures

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