Chapter 1

The Normal Electrocardiogram
A Brief Review

Chapter 1 reviews the genesis and inherent logic of the normal 12-lead electrocardiogram (ECG). This chapter explains, electrophysiologically and anatomically, "normal sinus rhythm," junctional rhythms, normal and abnormal q-waves and cardiac axis. This chapter also reviews several common (albeit non-life-threatening) abnormalities, such as poor R-wave progression, atrial enlargement, old anterior, septal and inferior myocardial infarctions and common ECG artifacts (for example, limb lead reversal and misplacement of the chest leads).

The Basics

The electrophysiologic principles that underlie the normal 12-lead ECG are more than a century old. Here are the basics:

- The ECG recording represents an electrical current (a depolarization wave) flowing between myocardial cells. The depolarization current is possible because the myocardial cells are coupled to one another through electrical gap junctions ("electrical synapses"). The ECG records the summed electrical currents of millions of myocytes, depolarizing in a synchronous fashion (Surawicz and Knilans, 2008; Wagner and Strauss, 2014).
- The standard ECG includes 12 different leads located in different positions; these permit us to record depolarization currents flowing toward or away from specific monitoring leads. The leads are labeled in Figure 1.1 *according to their positive poles.* We can refer to them as "monitoring" or "exploring" leads because they record electrical activity in the myocardial segment right beneath them.
- "Depolarization" represents electrical activation of the myocardium. Depolarization is followed by contraction of the chamber (the process of excitation-contraction coupling). "Repolarization" represents restoration of the original electrical potential of the myocardial cells.
- If there is greater myocardial mass (more electrically active myocytes), the depolarization wave (R-wave or P-wave) is taller. That is, there is greater voltage in the leads facing that portion of the heart. Taller R-waves in the left-facing leads may be a sign of left ventricular enlargement, while loss of R-wave voltage often indicates old myocardial infarction (electrical silence). In later chapters, we examine other life-threatening conditions, such as pericardial tamponade and myocarditis, that may present as "low-voltage" QRS complexes.

The Cardiac Depolarization Current Is *Directional*

This is the most important concept of all. Understanding the direction of the depolarization current, and also the position of the ECG leads, will help explain not only normal sinus rhythm but also junctional rhythms, the "regional changes" of ST-elevation myocardial infarctions (STEMIs), most "STEMI equivalents," old myocardial infarctions, atrial enlargement and numerous other conditions.

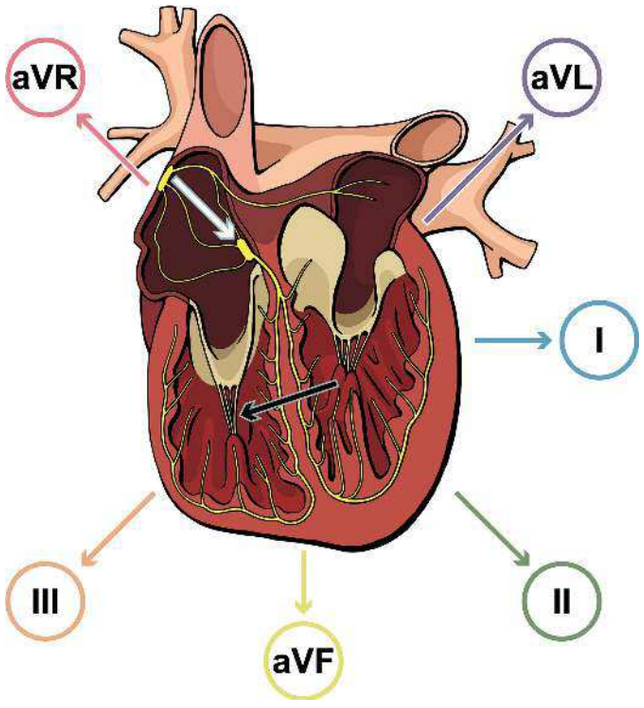


Figure 1.1 The normal electrical conduction pathways through the heart.

- The depolarization current originates in the sinus (SA, or sino-atrial) node, which is a collection of spontaneously firing pacemaker cells located in the upper reaches ("ceiling") of the right atrium (Surawicz and Knilans, 2008; University of Minnesota, 2014).
- The impulse then travels through the atria on its way to the AV node. Although this is controversial, the depolarization wave appears to proceed through the right and left atria via semi-specialized (or "preferential") pathways, known as internodal tracts, leading to activation of the right and left atria and inscription of the P-wave (Surawicz and Knilans, 2008; University of Minnesota, 2014).
- Not surprisingly, given the location of the SA node in the right atrium, the P-waves are often slightly notched in the limb leads (and they may be biphasic in lead V1), as the right atrium is depolarized slightly before the left atrium.

Even when the P-wave is smooth and rounded, we know that the first one-third of the P-wave represents right atrial depolarization, while the terminal third represents left atrial depolarization. Both atria contribute to the middle third of the P-wave (Wagner and Strauss, 2014). The duration, "notching" and biphasic shape of the P-wave are more pronounced if the left atrium is enlarged. (See the discussion and the self-study ECG tracings later in this chapter for examples of left and right atrial enlargement.)

The Six Limb Leads

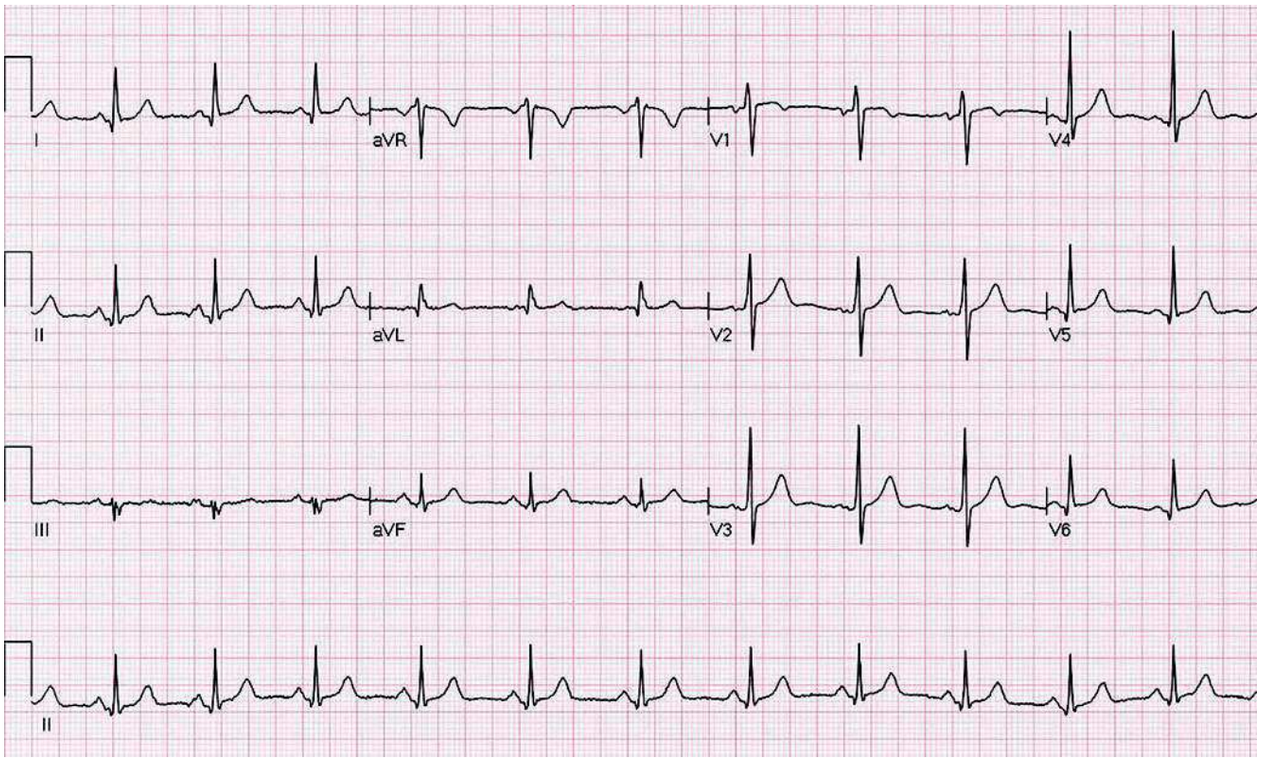
Figure 1.1 shows the "locations" of the six limb leads.

- The six limb leads are labeled according to their *positive poles.*[1] When we say that lead III "points to the right and inferiorly" (toward the right leg), we mean that this is the location of the positive pole of lead III.
- The ECG circuitry is configured so that a positive (upright) deflection – a P-wave or R-wave – is inscribed if the depolarization wave is traveling toward the positive pole of that lead.
- A negative deflection – a negative P-wave or (for the QRS complexes) a Q-wave if it is the first deflection or an S-wave – is inscribed if the depolarization wave is moving away from the positive pole.

Leads II, III and aVF are the inferior leads; leads I and aVL, which point toward the upper left side and the left shoulder, are the "high lateral" leads. Lead aVL is electrically "reciprocal" to lead III, since their positive poles point in nearly opposite directions. It is no surprise that an acute inferior STEMI is usually characterized not only by ST-segment elevations in leads II, III and aVF but also by *ST-segment depressions* in the "reciprocal leads (I and, especially, aVL). For additional discussion of the importance of ST-segment depressions in lead aVL, see Chapter 2, Inferior Wall Myocardial Infarction.

Normal Sinus Rhythm: The Complete Definition

Most introductory textbooks and lectures insist that a rhythm is "normal sinus" if there is a P-wave before every QRS and a QRS after every P-wave. However, this definition is unsatisfactory and incomplete. Because the sinus node, which initiates the depolarization wave, resides in the upper portion of the right atrium, the atrial depolarization wave begins at the "right shoulder" (beneath lead aVR); then it moves away from aVR toward lead II. Therefore, in normal sinus rhythm not only must there be a P-wave before every QRS, but *the P-wave must be negative in lead AVR, and it must be upright in lead II.* This is the complete definition of "normal sinus rhythm." Refer again to Figure 1.1 and ECG 1.1 (which demonstrates normal sinus rhythm).



ECG 1.1 The normal electrocardiogram.

[1] Each ECG lead monitors a specific region of the heart (for example, the inferior or high lateral wall of the left ventricle). Although limb leads I, II and III are "bipolar" while the "augmented" limb leads aVR, aVL and aVF are said to be unipolar, this distinction just introduces confusion. In fact, all the ECG leads are bipolar. Even the three "unipolar" leads have a "reference electrode that is constructed electrically from the other limb electrodes" (Gorgels et al., 2001; Wagner and Strauss, 2014; Kligfield et al., 2007). What is critical is that each lead is labeled according to its positive pole.

As an aside, in normal sinus rhythm the P-wave is often flat or indistinct in lead aVL. This is not surprising, as the atrial depolarization vector proceeds in a direction that is approximately 90 degrees perpendicular to the positive pole of aVL. Refer again to Figure 1.1 and ECG 1.1.

Conduction through the AV Node

- As noted previously, after leaving the SA node, the depolarization wave travels through the atria, along semi-specialized pathways (internodal tracks), until it arrives at the AV node (Surawicz and Knilans, 2008; University of Minnesota, 2014). It takes approximately 30 msec for the impulse to travel from the SA node through the internodal bundles to the AV node. It takes an additional 130–150 msec to travel through the AV node and His bundle. Thus, the normal PR interval (which includes the P-wave and the PR segment) ranges from 120–200 msec.
- The PR interval is not static; rather, AV nodal conduction is highly sensitive to the balance of sympathetic and parasympathetic tone. The PR interval is often shorter during tachycardias and when there is heightened sympathetic tone and longer when parasympathetic tone predominates. Interestingly, some young, healthy individuals may develop first-degree AV block or even Mobitz Type 1 second-degree heart block during sleep, when parasympathetic tone is high.
- After emerging from the AV node, the depolarization wave travels through the short His bundle, which pierces the interventricular septum. The AV node and the His bundle, together, constitute the "AV junction."

The Three Functions of the AV Node

The AV node serves three electrophysiologic functions:

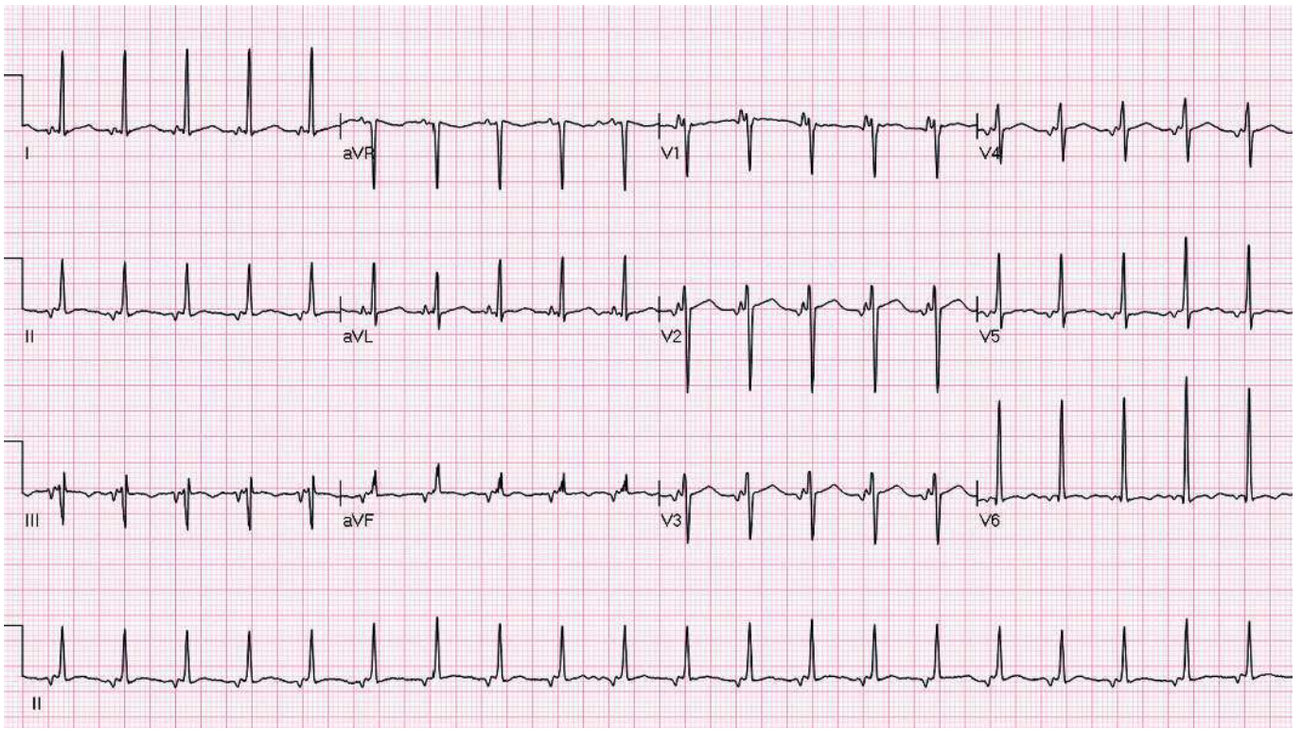
- The principal function of the AV node is to generate a pause; this enables the atria to contract and optimizes filling of the ventricles prior to ventricular systole.
- A second function of the AV node is to block rapid or ultra-rapid atrial impulses from reaching the ventricles (for example, during atrial fibrillation or flutter). The ability of the AV node to slow rapid impulses from the atria – and, thus, the "ventricular response" during atrial flutter or fibrillation – is exquisitely sensitive to the balance of sympathetic and parasympathetic tone. Of course, AV nodal conduction is also slowed in the presence of AV nodal blocking drugs and in the presence of sclerodegenerative conduction system disease, a condition primarily of elderly patients.
- Third, the AV junction can serve as a pacemaker, either when excited or when serving as an escape pacemaker. Acceleration of junctional pacemaker activity (accelerated junctional rhythm or nonparoxysmal junctional tachycardia) is commonly seen in patients with digitalis toxicity or acute inferior wall myocardial infarction, in the setting of cardiac surgery, during therapy with calcium channel blocking agents, and (years ago) in patients with acute rheumatic carditis (Surawicz and Knilans, 2008; Wagner and Strauss, 2014).

Junctional Rhythms

Junctional rhythms arise from a discrete pacemaker within the AV node or His bundle. They are characterized by inverted P-waves in the inferior leads and an upright P-wave in lead aVR, reflecting retrograde atrial depolarization (toward aVR and away from lead II). The inverted P-waves in the inferior leads may appear before or after the QRS complex; or, commonly, if atrial and ventricular depolarization occur concurrently, the inverted P-waves are hidden in the QRS complex.

Negative P-waves in the inferior leads may also represent an ectopic pacemaker originating in the low right or left atrium. It is more likely that the inverted P-wave represents a junctional pacemaker if the PR interval is short (< 120 msec). Conversely, if the PR interval is normal (≥ 120 msec), the origin of the inverted P-wave is more likely to be within the atria (ectopic or low atrial rhythm) (Surawicz and Knilans, 2008; Wagner and Strauss, 2014; Mirowski, 1966). The important point is that negative P-waves in II, III and aVF and upright P-waves in aVR signify that the atria are being activated from the junctional or low atrial tissue, with the atrial activation wave moving upward and to the patient's right shoulder (aVR).

ECG 1.2 is an example of a junctional rhythm.



ECG 1.2 A 21-year-old female with end-stage renal disease, systemic lupus and severe vasculitis presented because of lethargy and general weakness.

The Electrocardiogram

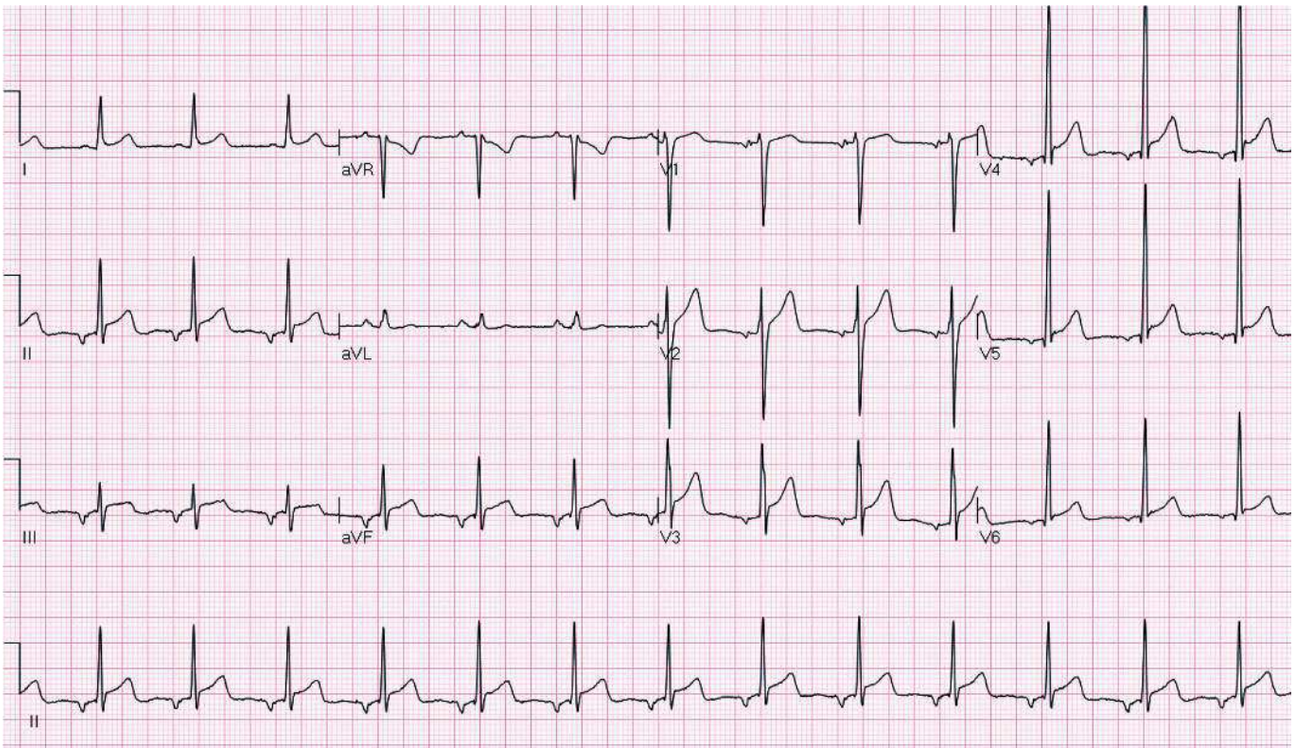
The ECG demonstrates a junctional tachycardia (accelerated junctional rhythm) with a heart rate of 122. The P-waves are inverted in the inferior leads and upright in lead aVR. This unusual, superiorly directed P-wave axis is indicative of a junctional pacemaker. Also, since the PR interval is short (90 msec), we are reasonably confident that this is a junctional, rather than an ectopic atrial, tachycardia. The ECG also demonstrates nonspecific ST- and T-wave flattening. Accelerated junctional rhythms (also referred to as "nonparoxysmal junctional tachycardias) typically have a heart rate of 60–100 beats per minute.[2]

Clinical Course

No cause for her junctional tachycardia was identified, and it resolved spontaneously after treatment with antibiotics, intravenous fluids, stress-dose corticosteroids and other supportive care.

[2] The usual rate for a junctional escape rhythm is approximately 40–60 beats per minute. If the rate is < 40, it is a junctional bradycardia. When the rate is 60–100, the rhythm is usually referred to as an "accelerated junctional rhythm." Common etiologies of accelerated junctional rhythms include digitalis excess, inferior myocardial infarction, cardiac surgery and (in years past) acute rheumatic carditis. Junctional tachycardias that exceed 100–120 beats per minute are called "accelerated junctional tachycardias" (Surawicz and Knilans, 2008).

ECG 1.3, from a young man with chest pain, is an example of an ectopic atrial rhythm.



ECG 1.3 A 29-year-old man presented with sharp left-sided chest pain that was "better when he rode his bicycle."

The Electrocardiogram

The P-waves are inverted in the inferior leads and are upright in lead aVR. Although there is a "P-wave before every QRS and a QRS after every P-wave," this cannot be normal sinus rhythm. The P-wave (atrial depolarization) vector is directed superiorly and to the patient's right. The PR interval is normal (164 msec). Therefore, this is an ectopic atrial rhythm. There are borderline voltage criteria for left ventricular hypertrophy (LVH), although this is likely a nonspecific finding in a patient under age 35. There are diffuse ST-segment elevations involving all the precordial and limb leads (notably, except aVR).

Clinical Course

His eventual diagnosis was acute pericarditis. He recovered uneventfully.

Ventricular Depolarization (the QRS Complexes)

Once the cardiac action potential has traversed the His bundle, it moves antegrade through the left and right bundle branches and spreads to the contractile myocytes via the ultra-rapidly conducting purkinje fibers.

- The purkinje fibers reside in the bundle branches and fascicles, and their principal function is to conduct impulses rapidly to all the cardiac myocytes, allowing for orderly and synchronous ventricular excitation.
- The overall direction (electrical vector) of the ventricular depolarization wave is downward and to the patient's left. Thus, the QRS axis points downward and to the left (and also posteriorly). See Figure 1.1.
- The initial deflection of the QRS complex (approximately 0.03 seconds) represents depolarization of the interventricular septum, in a left-to-right direction (Figure 1.1; also discussed later).
- The normal ECG produces predominantly upright, tall-amplitude QRS complexes in the left-facing leads (leads I, aVL and V5–V6). The ECG records mostly negative deflections (S-waves) in leads that are right-sided and anterior (for example, lead aVR, lead III and precordial leads V1 and V2).

Cardiac Axis

"Cardiac axis" refers to the overall electrical direction of the QRS complexes. As highlighted earlier, the normal direction of ventricular depolarization is downward and to the patient's left, producing upright QRS complexes in limb leads I and aVF. This represents a normal cardiac axis. If the QRS complex is upright in lead I but negative in aVF, the axis has shifted leftward. Conversely, if the QRS complex is negative in lead I (a larger than normal S-wave) but upright in aVF, there is right axis deviation. Infants, children, adolescents and young adults often have a right axis, but the axis generally shifts leftward with age. Significant S-waves in lead I (right axis deviation) are rarely normal in middle-aged or older adults, and their sudden appearance may signify pulmonary embolism or another cause of acute right heart strain.

Left axis deviation is a common ECG abnormality, which often reflects left anterior fascicular block, left ventricular hypertrophy, left bundle branch block or prior inferior myocardial infarction. As noted previously, modest degrees of left axis deviation also occur commonly with advancing age. Right axis deviation is common in children and young adults; after middle age, it usually suggests right ventricular hypertrophy, acute right heart strain or left posterior fascicular block. In older patients with chest pain, dizziness or shortness of breath, the simple finding of an S-wave in lead I should raise the suspicion of acute pulmonary embolism.[3]

The Six Precordial (Chest) Leads

Figure 1.2 depicts the six precordial (chest) leads.

- Lead V1, which is placed in the fourth intercostal space just to the right of the sternum, monitors the septum and is referred to as the "septal" lead. ST-segment elevation in V1 usually signifies an acute septal infarction.
- Lead V1 also monitors the right ventricle. Therefore, when an acute inferior wall STEMI is present, ST-segment elevation in V1 usually indicates a concomitant right ventricular infarction. (See Chapter 2.)
- Leads V2–V4 are the anterior (or "anteroapical") precordial leads, whereas leads V5 and V6 are the lateral precordial leads. A STEMI that involves V2–V4 is referred to as an anterior wall infarction; if ST-segment elevations are present in leads V1–V4, an anteroseptal myocardial infarction is present.
- ST-elevations in V5 and V6 usually represent a lateral wall infarction. Limb leads I and aVL are also lateral-facing electrodes. (In this atlas, they are called the "high lateral" leads.)

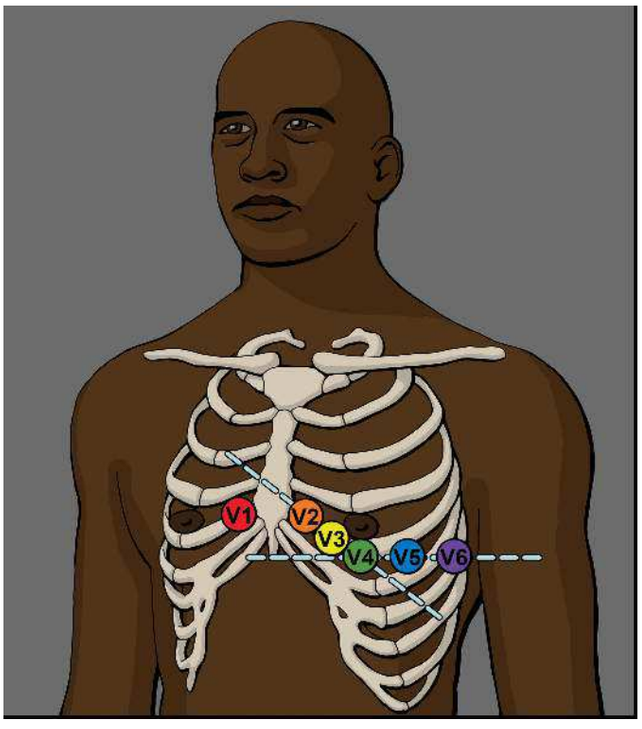


Figure 1.2 The precordial leads. Note that lead V1 is placed just to the right of the sternum (in the fourth intercostal space); it "monitors" the interventricular septum and is referred to as the "septal lead." V1 also monitors the right ventricle; in the setting of an acute inferior wall STEMI (caused by a right coronary artery occlusion), concomitant ST-segment elevation in precordial lead V1 usually signifies a right ventricular infarction. As highlighted in chapter 2, lead V1 is also a "right ventricular lead."

R-Wave Progression

Refer to Figure 1.3 and the normal ECG (ECG 1.1). In the normal heart, the R-wave amplitude should increase steadily (while the depth of the S-wave decreases) across the precordium from V1 to V5. The *precordial transition* zone – where the R-wave and S-wave voltages are equal – should occur no later than lead V4 (Surawicz and Knilans, 2008). If the precordial transition zone is delayed until V5 or V6 or if it never occurs (the R-wave height never exceeds the depth of the S-wave), the pattern is termed

[3] The other causes of an abnormal right axis deviation include: left posterior fascicular block; chronic hypoxic pulmonary disease; prior, extensive lateral wall myocardial infarction; and other causes of right ventricular hypertrophy (Surawicz and Knilans, 2008).

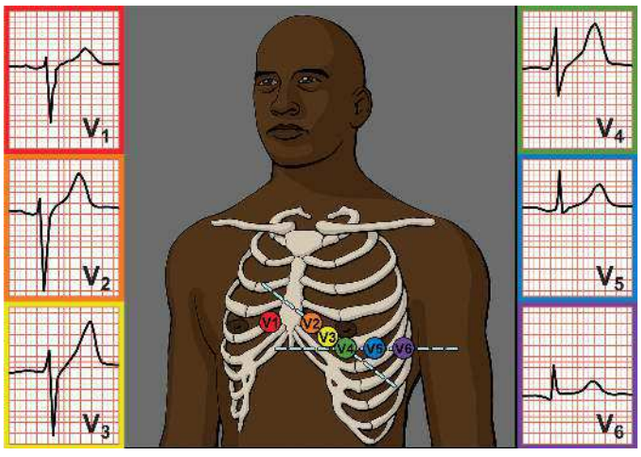


Figure 1.3 R-wave progression.

"poor R-wave progression" (Surawicz and Knilans, 2008; Wagner and Strauss, 2014).

The most common causes of poor R-wave progression are old anterior wall myocardial infarction, left ventricular hypertrophy, left bundle branch block, emphysema, dextrocardia or misplacement of the precordial electrodes (typically, 1–2 rib interspaces too high) (Rosen et al., 2014). Reverse R-wave progression (any decrement in the amplitude of the R-wave across the precordial leads moving from V1 to V5) may also occur, indicating a prior anterior wall myocardial infarction. These conditions are illustrated in the self-study ECGs.

V5 frequently has the highest amplitude because it is roughly situated at the apex of the left ventricle. If V6 is taller than V5, it may indicate left ventricular hypertrophy, as the enlarged LV pulls the depolarization vector in a more leftward and posterior direction. This corresponds to the bedside physical examination finding of a "laterally displaced point-of-maximal impulse" (PMI).

The Two Phases of the Normal QRS Complex – and the Concept of "Septal Q- and R-Waves"

As illustrated in Figures 1.1 and 1.4, depolarization of the ventricles – the QRS complex – actually consists of two distinct phases, which are readily detected on the ECG. The first phase (or "vector"), lasting 0.03 seconds or less, represents depolarization of the interventricular septum from left to right. The interventricular septum is depolarized from left to right simply because the depolarization wave, after exiting the His bundle, travels slightly faster down the left bundle branch (and more slowly down the right). This simple electrical fact explains the appearance of the normal, septal Q-waves that typically appear in the left-sided leads of the ECG (limb leads I and aVL and precordial leads V5 and V6). These R-waves are narrow (<.03 seconds) because the septum itself is so thin (Thygesen et al., 2012).

The small, leading R-wave in the septal lead (V1) is also easily explained: V1 is placed in the fourth intercostal space, just to the right of the sternum, an ideal position to record the left-to-right depolarization of the septum as a positive deflection. In ECG 1.1, and in almost every other normal tracing, the QRS complex in lead V1 begins with a small, narrow R-wave, representing normal left-to-right septal depolarization. If the initial R-wave is absent in V1, the patient has probably sustained a septal infarction (although faulty placement of the chest leads is an alternate explanation). Limb lead aVR also monitors the right side of the heart, and therefore aVR also begins with a small, initial septal R-wave. Because septal Q-waves (in the left-sided leads) and septal R-waves (in lead V1) represent depolarization of the septum from left to right, they are often absent if the patient has a left bundle branch block.

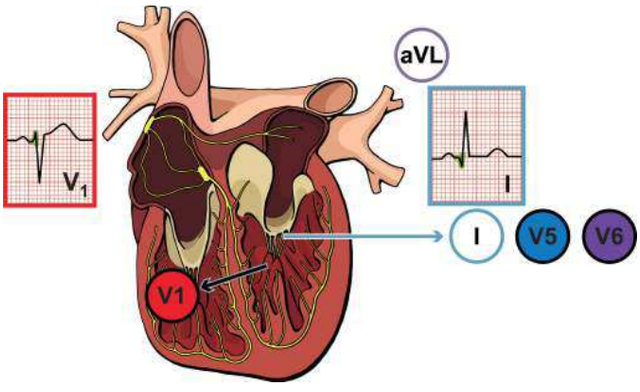


Figure 1.4 Septal depolarization: The initial phase of the QRS complex.

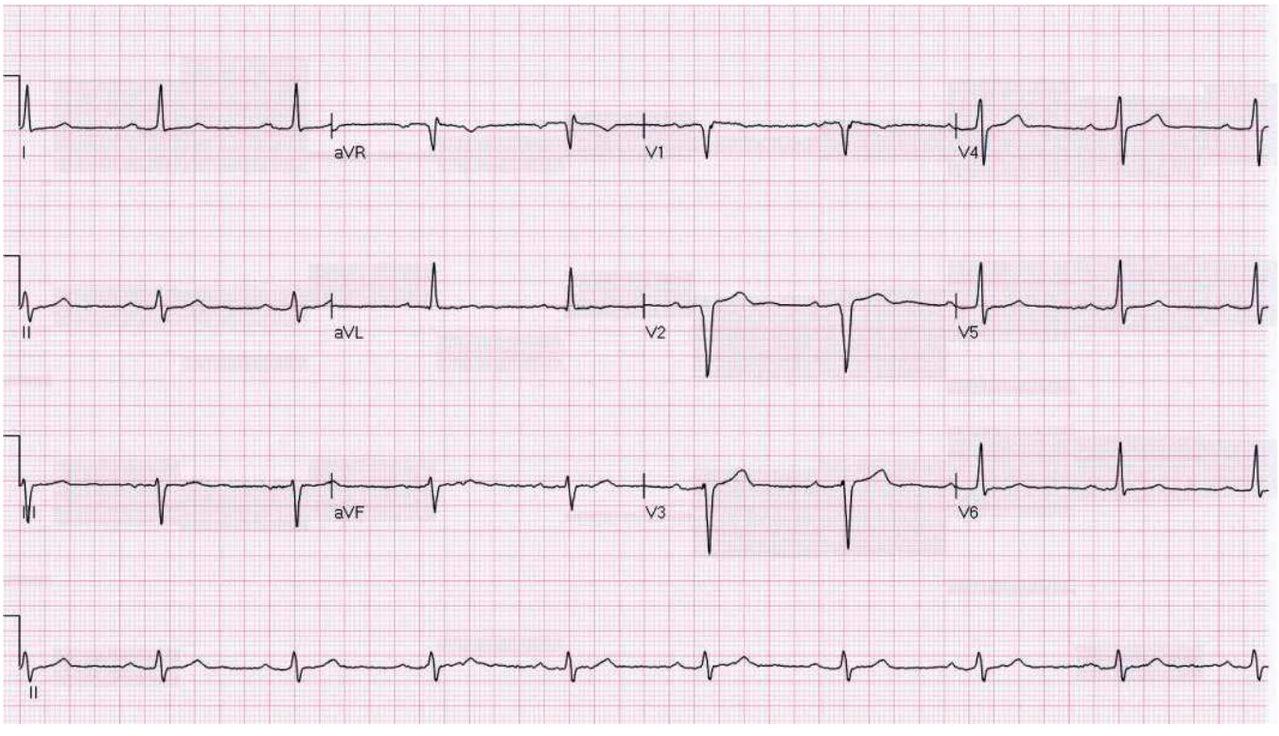
The second and longer phase of the QRS complex represents the simultaneous depolarization of the left and right ventricles, with the mass of the left ventricle predominating. Depolarization of the ventricles typically inscribes large R-waves in leads that monitor the left ventricle (V4, V5, V6 and leads I, II and aVF), since the impulse is traveling toward these leads; deep S-waves appear in right-sided leads (aVR, V1 and V2).

Abnormal Q-Waves Signifying Old Myocardial Infarction

As noted earlier, "septal" Q-waves are normal, thin, narrow Q-waves seen in the left-facing leads (limb leads I and aVL and precordial leads V5 and V6). Q-waves can, of course, also signal that the patient has sustained a prior myocardial infarction (variably labeled as "old," "remote" or "indeterminate age" myocardial infarction). These pathologic Q-waves reflect the absence of electrical activity (that is, absence of a normal transmural depolarization wave) in the zone of the infarction, beneath the exploring electrode. Stated differently, the upright QRS complex changes into a Q-wave or simply an S-wave (called a QS) beneath the electrode, reflecting electrical forces moving away from the lead. Old myocardial infarction can also be suspected if there is loss of R-wave voltage (a "Q-wave equivalent").

It is usually not difficult to distinguish normal from pathologic Q-waves. The distinction depends on duration, depth and location of the Q-waves. "Pathologic" Q-waves, signifying old infarction, typically include: (a) Q-waves involving contiguous leads in a defined region of the heart (for example, leads II, III and aVF); (b) any Q-wave in precordial leads V1–V3; (c) Q-waves in any leads that are >.04 seconds in duration (1 small box wide); or (d) Q-waves of a depth > 1 mm (1 small box) deep or deeper than 25 percent of the R-wave amplitude (Wagner and Strauss, 2014; Thygesen et al., 2012).

ECG 1.4 was obtained from a 70-year-old man.



ECG 1.4 A 70-year-old man reported a history of esophageal reflux disease, coronary artery disease, hypertension and hyperlipidemia. He underwent coronary artery bypass grafting 15 years earlier. He presented to the emergency department with chest pressure.

The Electrocardiogram

The ECG demonstrates sinus bradycardia and a first-degree AV block (PR interval = 212 msec). The ECG also demonstrates a left axis deviation along with absent R-waves in precordial leads V1–V3. The ECG is consistent with this patient's old anteroseptal myocardial infarction.

Clinical Course

In the hospital, serial troponins were all negative, and his ECG was stable. His antihypertensive medications were adjusted, and he had no further chest pain. A recent coronary angiogram showed patent saphenous vein grafts. He was discharged in stable condition.

Right and Left Atrial Enlargement

Conceptually, it is important to remember that the right atrium is activated first because atrial depolarization begins in the SA node, located in the upper portion of the right atrium. The left atrium is activated second. Thus, the normal P-wave may be slightly notched, although the duration of the P-wave should not exceed 0.12 seconds. The initial portion of the P-wave represents right atrial depolarization, while the terminal portion of the P-wave represents left atrial depolarization (Surawicz and Knilans, 2008; Wagner and Strauss, 2014; Hancock et al., 2009).

As illustrated in Figure 1.5, right atrial enlargement (RAE) does not prolong the duration of the P-wave; rather, RAE is characterized by an increase in the amplitude of the initial P-wave deflection – and loss of the normal, rounded contour of the P-wave. In RAE, the P-wave becomes taller and "peaked," "gothic" or "steeple-like" (Surawicz and Knilans, 2008). RAE is best seen in leads II and III. To meet strict criteria for RAE, the P-waves should be at least 2.5 mm (small boxes) tall in the inferior limb leads (Surawicz and Knilans, 2008; Wagner and Strauss, 2014; Hancock et al., 2009).

In the past, RAE was called "P-pulmonale," a logical term since RAE is most often caused by chronic hypoxic lung disease in association with pulmonary hypertension and right ventricular enlargement (cor pulmonale). Commonly, RAE on the ECG is associated with right ventricular enlargement, right axis deviation and other features of chronic lung disease. RAE is also commonly caused by congenital heart disease (for example, tetralogy of Fallot or pulmonic stenosis), primary pulmonary hypertension and other causes of chronic hypoxemia.

In left atrial enlargement (LAE), the P-wave is classically broad and often notched ("double humped") in leads I and II (and sometimes aVL). The most important lead for diagnosing LAE is precordial lead V1, which is located on the right side of the chest, in an anterior position. LAE typically inscribes a biphasic P-wave in lead V1. The terminal portion of the P-wave represents the left atrium because the enlarged left atrium is depolarized later and for a longer time (Wagner and Strauss, 2014). The terminal portion of the P-wave is negative in the anterior-facing precordial lead V1. This is because the left atrium is normally located in a posterior position, almost abutting the esophagus.

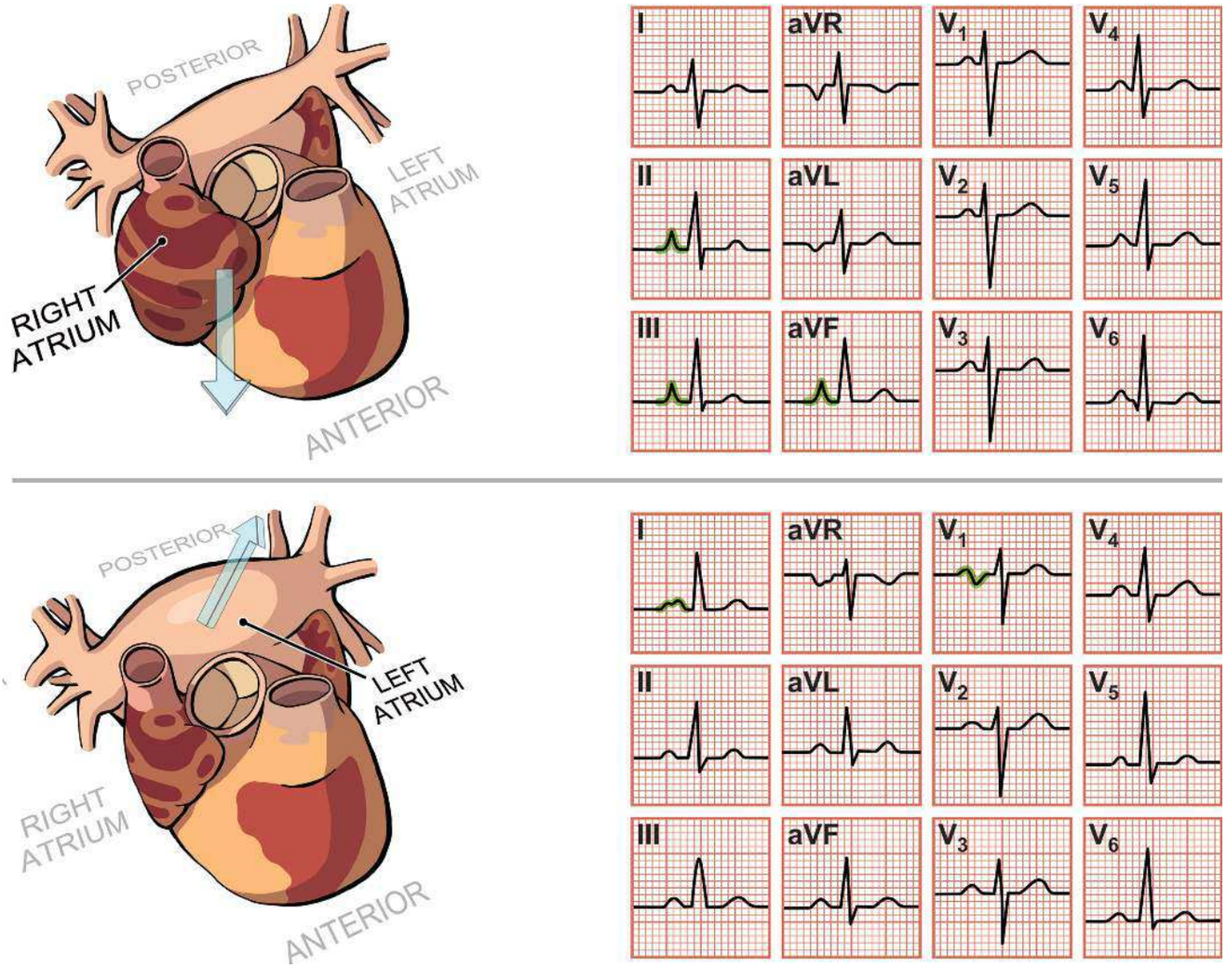


Figure 1.5 Right and left atrial enlargement.

Historically, the pattern of broad and notched P-waves was referred to as "P-mitrale" because mitral stenosis was the most common etiology of left atrial enlargement.

Because atrial chamber enlargement cannot always be distinguished from atrial fibrosis, distention, strain or conduction delay, the less specific terms "right atrial abnormality" and "left atrial abnormality" are frequently used (Hancock et al., 2009; Wagner and Strauss, 2014). Bi-atrial enlargement can often be recognized on the ECG as a hybrid of the two patterns described previously. Examples of atrial enlargement are included in the self-study electrocardiograms.

Left and Right Arm Lead Reversal

Reversal of the left and right arm leads is a relatively common technical error. It is easily detected by finding a negative P-QRS-T in lead I *in the presence of normal R-wave progression.* Thus, arm lead reversal represents a spurious cause of right axis deviation on the ECG. Not surprisingly, the P-QRS-T waves are all upright in aVR, a clear signal that the 12-lead is abnormal. That is, the patterns in lead I and lead aVR are reversed (Surawicz and Knilans, 2008; Wagner and Strauss, 2014; Rosen et al., 2014; Hancock et al., 2009; Kligfield et al., 2007; Harrigan et al., 2012).

Finding net negative P-waves and QRS complexes in lead I is sometimes referred to as the "lead 1 alerting sign" (ECGpedia.org, 2016). As a general rule, the most likely diagnosis is limb lead reversal. The "lead 1 alerting sign" (predominantly negative P-waves, QRS complexes and T-waves in lead I) is also seen with dextrocardia, but in dextrocardia there is loss of (actually, reverse) R-wave progression in the left chest leads. (See ECGs 1.5 and 1.6.)