Podrid’s Real-World ECGs

AMaster’s Approach to the Art and Practice of Clinical ECG Interpretation

Volume 1  The Basics

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy and daughter Sapna, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

Peter A. Noseworthy
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Foreword

The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven’s landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiodiagnostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today’s cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled “Real-World ECGs,” by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient’s clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.
These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important dividend for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, realworldECGs.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

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Foreword

The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable “workhorse” in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician’s office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of Podrid’s Real-World ECGs aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in Podrid’s Real-World ECGs teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

Volume 1 provides an essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the
interpretation of all ECGs. The subsequent volumes focus on particular disease entities for which the ECG is useful:

- Myocardial abnormalities, including infarction, hypertrophy, and inflammation
- Atrioventricular (AV) and intraventricular conduction disturbances and enhanced AV conduction
- Sinus, atrial, junctional, and ventricular arrhythmias
- Narrow and wide complex tachycardias and forms of aberration
- Recording methods and miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs and clinical scenarios to further enhance the student’s skills at ECG analysis. Importantly, each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: realworldECGs.com

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers easy access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q & A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

Philip Podrid, MD
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**Introduction**

**The Basics**

This workbook presents the essentials of electrocardiogram (ECG) analysis: the heart’s conduction system, the normal activation sequence, the lead system, a systematic approach for analysis of the ECG, the normal waveforms and intervals, and axis determination. The ECGs that follow the introduction demonstrate various abnormalities of atrioventricular (AV) and intraventricular conduction, features of myocardial abnormalities, and the common supraventricular and ventricular arrhythmias.

**Components of the ECG**

The ECG is a recording of the electrical activity of the heart. The waveforms that make up the ECG (P, QRS, and T) reflect depolarization and repolarization of the atrial and ventricular myocardium. The activity of the heart’s electrical, or conduction, system (sinus node, AV node, and His-Purkinje system) is not transmitted to the surface of the body and hence is not recorded or manifested on the ECG. However, abnormalities of the conduction system can be established by careful analysis of the waveforms and intervals that are routinely measured on the ECG (PR interval, QRS duration, and QT interval).

The electrical system of the heart (Figure 1), which is responsible for generating an action potential and transmitting this action potential to all parts of the atrial and ventricular myocardium in a uniform...
and simultaneous fashion, includes the sinus or sinoatrial node (the dominant pacemaker of the heart as it generates an impulse with higher frequency than any other tissue), conduction pathways through the right and left atria, the AV node or junction, the bundle of His, the right bundle (which innervates the right ventricle), the left bundle (which innervates the left ventricle), and the Purkinje fibers (which bring the impulse to the individual myocardial cells). Since the muscle mass of the left ventricle is large, the left bundle splits into two major fascicles and one minor fascicle that result in simultaneous activation of the entire left ventricular myocardium. These fascicles include a minor septal branch or median fascicle (innervating the interventricular septum) and two major fascicles (the left anterior fascicle and left posterior fascicle).

Normal Activation of the Atria and Ventricles

The normal activation sequence of the heart can be seen in figures 2 and 3. The initial impulse activating the heart is normally generated by the sinus node, which is located in the proximal portion of the right atrium. This impulse then spreads to depolarize the right and left atria. The activation sequence is from right to left and proximal to distal (up to down). Atrial depolarization generates a P wave on the surface ECG.

**Figure 2:** Normal activation of the atria and ventricles and the ECG waveforms generated. The dominant pacemaker is the sinus node. The impulse that originates in this structure conducts to the right and left atria, and depolarization or activation of these structures generates a P wave on the surface ECG. The impulse then conducts through the atrioventricular (AV) node and then the bundle of His and right and left bundles. These structures are too small to generate electrical activity that can be recorded. Hence on the ECG this is the PR segment, which is the time for conduction from the atrium to the ventricles via the AV node and His Purkinje system. The first part of the ventricles to be depolarized or activated is the left side of the septum. As the impulse goes from left to right, there is a small negative deflection in most leads, termed a Q wave. The rest of the left and right ventricular myocardium becomes activated or depolarized in a right to left direction. This generates the entire QRS complex on the surface ECG. After the ventricles complete their depolarization, there is a period of electrical quiescence, which is the ST segment on the surface ECG. Repolarization of the ventricles follows this and generates a T wave on the ECG.
Based on the direction of activation, the P wave will be positive in the left-sided and inferior leads (ie, leads I, II, aVF, and V4-V6) and negative in the right-sided lead (ie, lead aVR). The impulse then reaches the AV node, which is the structure within the electrical system with the slowest conduction velocity. Hence there is a delay in impulse conduction through this structure. After passing through the AV node, the impulse reaches the His-Purkinje system and then enters the right and left bundles. Since these structures are small and generate very little electrical activity, no manifestation of their activation is seen at the surface of the body. The ECG does not record any electrical activity as the impulse travels through the AV node and His-Purkinje system. This accounts for the PR segment, which is at baseline or zero potential. Therefore, the PR segment represents AV conduction time and includes conduction through the AV node and His-Purkinje system.

The first part of the ventricle to be depolarized is the intraventricular septum, and the impulse arises from a septal (median) branch of the left bundle. Hence the direction of septal depolarization is from left to right, accounting for the small initial negative waveform (septal Q wave) of the QRS complex seen in left-sided leads and the small positive deflection (R wave) seen in right-sided leads. Thereafter, the rest of the right and left ventricles are depolarized simultaneously. Since the left ventricular muscle mass is much greater than that of the right ventricle, the QRS complex on the surface ECG primarily represents left ventricular depolarization; this occurs in a right-to-left direction, accounting for a tall positive deflection (R wave) in the left-sided leads (ie, leads I and V4-V6) and a negative deflection (S wave) in the right-sided leads (ie, leads aVR and V1). The last part of the left ventricle to be depolarized is the lateral and posterior walls; this occurs in a left-to-right direction, accounting for the terminal negative deflection (S wave) in the left-sided leads. Once depolarization is completed, there is a brief period during which no electrical activity occurs (ie, the ST segment is at baseline or zero potential). This is followed by repolarization, which generates a T wave (see figure 3).

**Figure 3**: Normal activation of the atria and ventricles and the waveforms generated.
Frequently seen following the T wave is a U wave, which is believed to represent late repolarization of the His-Purkinje system. Some believe that the U wave may represent late repolarization of the papillary muscles. This is a low-amplitude positive waveform after the T wave, best seen in the right precordial leads (i.e., leads V1-V3).

**Lead System**

The standard ECG includes 12 leads: six limb leads (recording the electrical current in the frontal plane) and six precordial or chest leads (recording the electrical current in the horizontal plane).

The six limb leads include the following (figures 4 and 5):

- **Lead I** is a bipolar lead that records the impulse as it travels from right arm to left arm. Impulses going toward the left produce a positive waveform in this lead; impulses going toward the right produce a negative waveform. Therefore, in normal situations the P wave (due to atrial activation that goes from right to left) is positive in this lead. The QRS complex, which is due to impulse conduction in a right-to-left direction, is also positive, and there may be a small septal Q wave (representing septal depolarization that occurs in a left-to-right direction) before the tall positive waveform, or R wave, representing left ventricular depolarization.

- **Lead II** is a bipolar lead that records the impulse as it travels from the right arm to the left foot. Impulses going toward the foot generate a positive waveform in this lead; a negative waveform is generated if the impulse is directed away from the foot toward the right arm. Therefore, in normal situations the

**Figure 4**: Leads I, II, and III are bipolar leads. In lead I an impulse that travels toward the left arm generates a positive waveform, and an impulse that travels toward the right arm generates a negative waveform. In leads II and III, an impulse that travels toward the foot generates a positive waveform and an impulse that goes away from the foot toward the arms generates a negative waveform.
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P wave (representing atrial activation that occurs in a proximal-to-distal direction) is positive in this lead. The QRS complex (representing ventricular depolarization that occurs from proximal to distal) is also positive, and there may be a small septal Q wave before the tall positive waveform, or R wave, representing left ventricular depolarization.

- **Lead III** is a bipolar lead that records the impulse as it travels from the left arm to the left foot. Impulses going toward the foot generate a positive waveform in this lead; a negative waveform is generated if the impulse is directed away from the foot toward the left arm. Based on the angle of lead III in relation to the heart, the waveforms may be positive or negative depending on small changes in the direction of electrical activation. Hence lead III is an indeterminate lead and should not be evaluated by itself.

- **Lead aVR** is an augmented unipolar right arm lead, meaning that the impulse is recorded as if it originates from the center of the heart. An impulse that is directed toward the right arm produces a positive waveform in this lead; a negative waveform is generated if the impulse is directed away from the right arm. Hence lead aVR is the only limb lead that is right sided. Since the impulses in the heart are primarily directed from right to left (away from the right arm), the waveforms are normally negative in this lead (*i.e.*, they are mirror images of waveforms in other leads). Therefore, the P wave is normally negative and there is an initial septal R wave followed by a large negative waveform, or S wave, representing left ventricular depolarization.

**Figure 5:** Leads aVR, aVL, and aVF are unipolar leads. The impulse is imagined as originating from the middle of the heart. An impulse that goes toward any of these leads generates a positive waveform, and an impulse that goes away from these leads generates a negative waveform. Since the cardiac impulse goes primarily from right to left and up to down (superior to inferior), the waveforms are positive in leads aVL and aVF but negative in lead aVR.
• **Lead aVL** is an augmented unipolar left arm lead, meaning that the impulse is recorded as if it originates from the center of the heart. An impulse that is directed toward the left arm produces a positive waveform; the waveform is negative if the impulse is directed away from the left arm. Therefore, in normal situations the P wave (generated by an impulse that is directed from right to left) is positive in this lead. The QRS complex (due to impulse generation in a right-to-left direction) is also positive, and there may be a small septal Q wave before the tall positive waveform, or R wave, representing left ventricular depolarization.

• **Lead aVF** is an augmented unipolar left foot lead, meaning that the impulse is recorded as if it originates from the center of the heart. An impulse that is directed toward the left foot generates a positive waveform; an impulse directed away from the left foot produces a negative waveform. Therefore, in normal situations the P wave (due to impulse generation that travels proximally to distally) is positive in this lead. The QRS complex (resulting from an impulse that travels proximally to distally) is also positive, and there may be a small septal Q wave before the tall positive waveform, or R wave, representing left ventricular depolarization.

Precordial leads V1-V6 are placed on the chest wall in the following locations (**Figure 6**):

• **Lead V1** is placed at the second intercostal space below the clavicle to the right of the sternum.

• **Lead V2** is placed at the second intercostal space below the clavicle to the left of the sternum.

• **Lead V3** is placed midway between leads V2 and V4.

• **Lead V4** is placed at the fifth intercostal space below the clavicle at the midclavicular line.

• **Lead V5** is placed at the fifth intercostal space below the clavicle at the anterior axillary line.

• **Lead V6** is placed at the fifth intercostal space below the clavicle at the midaxillary line.

As with the augmented limb leads, leads V1-V6 are unipolar leads and the impulse is recorded as if it originates from the center of the heart. An impulse that is directed toward a precordial lead produces a positive deflection, and the deflection is negative if the impulse is directed away from a precordial lead (**Figure 7**). Lead V1 is mostly
The Basics:

Introduction

Over the right ventricle and right atrium, while leads V2-V3 are primarily over the intraventricular septum toward the left ventricle and leads V4-V6 are over the left ventricle and left atrium. Therefore, in lead V1 (and occasionally in lead V2) the P wave is biphasic, as the initial depolarization is from the right atrium (going toward lead V1) and the second part of the P wave is from the left atrium, going away from lead V1. The P waves in leads V3-V6 are normally positive as these leads are primarily over the left atrium and reflect left atrial activation, which goes toward these leads.

Based on the location of the V leads, there is an initial small positive waveform (R wave) in lead V1, representing septal depolarization going from left to right and hence toward this lead. This is followed by a deep S wave representing left ventricular depolarization, which goes in a right-to-left direction (away from this lead). In contrast, lead V6 has an initial negative waveform (Q wave) representing initial septal activation going from left to right or away from this lead. This is followed by a tall positive deflection (R wave) representing left ventricular depolarization, which travels in a right-to-left direction and hence toward this lead. As one inspects leads V1-V6 (which go in a right-to-left direction across the chest), there is a gradual increase in the amplitude of the R wave (reflecting more left ventricular forces directed toward the lead) and a decrease in the depth of the S wave (which reflects left ventricular forces going away from the lead). Hence the gradual increase in the amplitude of the R wave (going from V1-V6) is called R-wave progression across the precordium (figure 8).

The transition (R/S ≥ 1) usually occurs between leads V3 and V4.
Approach to ECG Analysis

ECGs should be analyzed thoroughly and systematically while considering the patient’s clinical history. By following a standardized sequence of steps, subtle abnormalities in the ECG will become evident.

1. Establish the heart rate. The normal heart rate ranges from 60 to 100 bpm; bradycardia is defined as a heart rate of 60 bpm or lower and tachycardia as a heart rate of 100 bpm or higher. Heart rate can be established in two ways:
   • Count the number of QRS complexes and multiply by 6 (as an ECG recording takes 10 seconds). This is the preferred method when the rhythm is irregular.
   • Use the grid on the ECG tracing. Heart rate = 300 ÷ the number of large boxes within one RR interval. If the RR interval duration is between two large boxes, the number of small boxes (five per large box) is then used to approximate the heart rate. For example, if the RR interval is between one and two large boxes (ie, the heart rate is between 300 and 150), each small box is 150 ÷ 5 or 30 bpm. If the RR interval is between two and three large boxes (ie, heart rate between 150 and 100 bpm), each small box is 50 ÷ 5 or 10 bpm. If the RR interval is between three and four large boxes (ie, heart rate between 100 and 75 bpm), each small box is 25 ÷ 5 or 5 bpm. If the RR interval is between four and five large boxes (ie, heart rate between 60 and 75 bpm), each small box is 15 ÷ 5 or 3 bpm.

2. Establish where the rhythm originates, that is, in which structure is the pacemaker that initiates the impulse. This is based on the presence or absence of a P wave, location of the P wave (before or after the QRS complex), and P-wave morphology. A sinus origin for the atrial activity is associated with a positive P wave in leads I, II, aVF, and V4-V6. The normal atrial impulse direction generates a positive waveform in these leads. If the P wave is negative in any of these leads, the impulse direction is not normal and hence the origin is not the sinus node but rather somewhere else in the atrium; therefore, this is an atrial rhythm. If there is no P wave before any QRS complex, the origin of the impulse is not sinus or atrial but is either the AV node or AV junction (if the QRS complex is narrow and normal) or the ventricular myocardium (if the QRS complex is wide with an unusual morphology).

3. Establish the regularity of the rhythm. Note whether the RR intervals are regular, regularly irregular (irregular but with a pattern often based on abnormalities in AV conduction or AV block), or irregularly irregular (no pattern to the RR intervals).

4. Determine the electrical axis in the frontal plane (ie, normal, leftward, rightward, or indeterminate).

5. Measure the PR interval, QRS complex duration, and QT interval.

6. Evaluate the R-wave progression across the precordium. This will also establish the electrical axis in the horizontal plane (ie, normal, clockwise, or counterclockwise rotation).

7. Evaluate the P-wave amplitude, duration, and morphology.

8. Establish the QRS complex duration, amplitude, and morphology.

9. Indicate the presence of pathologic Q waves (ie, longer than 0.04 sec in duration).
10. Evaluate the ST-segment (morphology and elevation or depression) and J-point changes.

11. Note T-wave abnormalities.

12. Indicate the presence of other waveforms (e.g., U waves, pacemaker spikes, artifact).

Normal Waveforms and Intervals
Components of the normal waveform include the P wave, PR interval, QRS complex, QT interval, T wave, and U wave (Figure 9).

P Wave
The normal P wave, which should be positive in leads I, II, aVF, and V4-V6, represents right atrial followed by left atrial depolarization. The normal P-wave duration is 0.12 second or less, and the usual amplitude is less than 2.5 mV (2.5 small boxes).

PR Interval
The PR interval, which includes the P wave and PR segment, is a measure of AV conduction, or the time required for the impulse to go from atrium to ventricle (including conduction through the atrium, which is the P wave, and conduction through the AV node and His-Purkinje system, which is the PR segment). The PR interval is determined by measuring the duration from the beginning of P wave to the first wave of QRS complex (either a Q or R wave). The normal PR interval is between 0.14 and 0.20 second. The PR segment should be at baseline or zero potential as there is no electrical activity measured on the surface.

Figure 9: Components of a typical waveform and the usual intervals that are measured. The PR interval (time for AV conduction) is measured from the beginning of the P wave to the beginning of the QRS complex (either a Q or R wave). The PR interval includes the P wave and PR segment. The QRS complex duration or interval, which is the time for ventricular depolarization, is measured from the beginning of the QRS complex (either a Q or R wave) to the end of the QRS complex, which is the J point. The QT interval, which is the time for ventricular depolarization and repolarization, is measured from the beginning of the QRS complex (either a Q wave or R wave) to the end of the T wave. This interval includes the QRS complex, ST segment, and T wave.
ECG during this time, even though there is electrical activity occurring within the AV node and His-Purkinje system.

The PR interval changes with heart rate, primarily reflecting changes in AV nodal conduction time (which is the major determinant of the PR segment). Conduction through the His-Purkinje system is constant as there is no alteration of conduction velocity related to heart rate changes (i.e., conduction through this part of the conduction system is all or none). At slower sinus rates (higher vagal tone and less sympathetic stimulation), conduction through the AV node slows and hence the PR interval (segment) lengthens. With fast sinus rates, reflecting less vagal tone and increased sympathetic stimulation, AV nodal conduction velocity increases and the PR interval (segment) shortens. There is, however, no method available to correct the PR interval for rate.

At a given heart rate, the PR interval should be constant. Any variability reflects an AV conduction abnormality. When there is no pattern to the variability, AV dissociation is present. The presence of progressive lengthening of the PR interval is seen with type 1 second-degree AV block (Mobitz I or Wenckebach).

QRS Complex

The QRS complex duration (or interval) represents time for ventricular depolarization. The duration is measured from the beginning of the QRS complex (either a Q or R wave) to the end of the QRS complex (which is defined as the J point and is located at the end of the QRS complex and the beginning of the ST segment). Right and left ventricular depolarization is simultaneous. However, since the left ventricular myocardial mass is much greater than the right ventricular mass, the QRS complex primarily reflects left ventricular depolarization. The normal QRS duration is between 0.06 and 0.10 second and does not change with heart rate (i.e., His-Purkinje impulse conduction is all or none). A QRS complex duration that is 0.10 second or longer is called an intraventricular conduction delay (IVCD). If the QRS complex duration is 0.12 second or longer with a typical pattern, the IVCD may represent a bundle branch block.

QT Interval

The QT interval is a measure of the time for ventricular repolarization. It is measured from the beginning of QRS complex (either a Q or R wave) to the end of T wave. It must be remembered that since the QT interval measurement includes the QRS complex, it is not just a measure of left ventricular repolarization but also includes the time of left ventricular depolarization. It is also important to remember that a prolonged QRS duration (due to a bundle branch block or a nonspecific IVCD) may result in lengthening of the measured QT interval. In this situation, the prolonged QT interval is not due to prolongation of repolarization. As the normal QT interval measurements are based on a normal QRS duration (i.e., 0.06 to 0.10 sec), any prolongation of the QRS duration above this value needs to be considered and the increased duration (in milliseconds) subtracted from the QT measurement. The QT interval changes with heart rate, that is, it is prolonged at slower rates and shortens with faster rates. Therefore, the QT interval must be corrected for rate (i.e., QTc), using Bazett’s formula:

\[ QTc = QT \div \sqrt{RR \text{ interval}} \]

The normal QTc is less than 0.44 to 0.48 second.
T Wave
The T wave actually represents ventricular repolarization. The T-wave axis is usually the same as that of the QRS complex. That is, the T-wave direction (positive or negative) is the same direction as the major deflection of the QRS complex; if the QRS complex is positive the T wave is positive, and if the QRS complex is negative the T wave is negative. The normal T wave is asymmetric regardless of amplitude (i.e., it has a slower upstroke than downstroke). Also, the normal T wave is smooth in both its upstroke and downstroke. Any notches, bumps, or other irregularities on the T wave may represent superimposed P waves.

U Wave
The U wave is a low-amplitude positive waveform that follows the T wave. The U wave is believed to represent delayed repolarization of the His-Purkinje system, although it has been suggested that it may represent delayed repolarization of the papillary muscles. It is most often seen in the right precordial leads (i.e., leads V1-V3).

Normal ECG Recording
In most cases, 12 leads are recorded (i.e., six limb leads and six precordial [chest] leads). The standard layout for the ECG is four columns with three leads in each column (I, II, III; aVR, aVL, aVF; V1, V2, V3; V4, V5, V6). Each column is simultaneously recorded while each line is continuously recorded. One or several rhythm strips (one lead continuously recorded over time) may be present at the bottom of ECG.

The paper speed for recording is usually 25 mm/sec (10 sec for a full 12-lead recording). Occasionally a paper speed of 50 mm/sec is used. In this situation, there are only six leads per sheet of paper; the PR, QRS, and QT intervals are very long (twice normal), and the heart rate is very slow (half of normal) (Figure 10).

Most often, normal standardization is used. Standardization indicates the amplitude covered by 1 mV of electric current. Normal standardization means that 1 mV = 10 mm (10 small boxes) in height. When QRS amplitude is high, half-standard may be used, or 1 mV = 5 mm (five small boxes). When QRS amplitude is small, double standard may be used, or 1 mV = 20 mm (20 small boxes) (see Figure 10).

Figure 10: Paper speeds and standardizations used for recording an ECG. In a typical ECG, the normal paper speed is 25 mm/sec and normal standardization is used.
P Wave and PR Interval
Since the sinus node is located in the proximal portion of the right atrium and depolarization occurs in a right-to-left and up-to-down (proximal to distal) direction, the normal P wave is positive (upright) in leads I, II, aVF, and V4-V6. Atrial repolarization occurs during the time of the QRS complex and hence is not seen.

There is often a slight notching of the P wave, reflecting right followed by left atrial depolarization. A broad, notched P wave is seen with left atrial hypertrophy (or abnormality) and is called P mitrale (Figure 11). A narrow, peaked P wave is seen with right atrial hypertrophy (or abnormality) and is called P pulmonale (Figure 12). The P wave is negative in lead aVR.

The P wave is often biphasic (positive-negative) in lead V1, reflecting right atrial depolarization (impulse toward lead V1) followed by left atrial depolarization (impulse away from lead V1) (Figure 13). The P-wave duration is 0.12 second or less, and the amplitude is usually 0.25 mV or less.

P waves that are inverted or biphasic (negative-positive) in leads I, II, aVF, and V4-V6 are abnormal, reflecting an ectopic atrial focus. Negative P waves that follow a QRS complex are retrograde, due to retrograde (ventriculoatrial) conduction.

Every P wave should have an associated QRS complex, and every QRS complex should be preceded by a P wave. The PR interval should be stable. The normal PR interval is 0.14 to 0.20 second measured from the beginning of the P wave to the beginning of the QRS complex (either a Q or R wave). Inconsistent variability of PR interval is AV dissociation; gradual prolongation of the PR interval is seen with Wenckebach or second-degree AV block (Mobitz type I).

Figure 11: The P wave in left atrial hypertrophy (or abnormality) is broad and notched. This is termed P mitrale.

Figure 12: The P wave in right atrial hypertrophy (or abnormality) is tall, narrow, and peaked. This is termed P pulmonale.
QRS Complex
The direction of the waveforms or deflections of the QRS complex determine the letter applied to the components of the QRS complex (figure 14). If the first deflection is negative it is called a Q wave. Any first positive deflection is called an R wave (there may or may not be a Q wave). Any negative deflection after the R wave is called an S wave. If there is a second positive deflection, it is termed an R’ (R prime).

The first depolarization of the left ventricle occurs in the left septum (via the septal or median branch of the left bundle) in a left-to-right direction. Hence there is normally a small septal Q wave in leads I and V5-V6 and a small septal R wave in lead V1. The direction of the rest of ventricular activation is right to left and proximal to distal. Therefore, the normal QRS complex is positive in leads I, II, aVF, and V1-V6 and negative in lead aVR. There may be a small R’ in lead V1, which is a normal variant reflecting a slight conduction delay in the right ventricle. The QRS complex may have notching, which is a normal variant.

Since the precordial leads reflect activation from the right to the left ventricle, the initial septal forces are directed toward lead V1 (small R wave) and away from lead V6 (small Q wave). The rest of the left ventricular forces are directed away from lead V1 (hence an S wave) and toward lead V6 (hence an R wave). Therefore, going from lead V1 to V6, the R-wave amplitude progressively increases and the S-wave depth decreases; that is, R/S becomes greater.

<table>
<thead>
<tr>
<th></th>
<th>Lead II</th>
<th>Lead VI</th>
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<tbody>
<tr>
<td><strong>Normal</strong></td>
<td>RA</td>
<td>LA</td>
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<td><strong>Combined</strong></td>
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<tr>
<td><strong>Right atrial hypertrophy or abnormality</strong></td>
<td>RA</td>
<td>LA</td>
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<tr>
<td><strong>Left atrial hypertrophy or abnormality</strong></td>
<td>RA</td>
<td>LA</td>
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**Figure 13:** Appearance of the P wave in leads II and V1 in right and left atrial hypertrophy. The normal P wave in lead VI is biphasic as the initial force (depolarization) is from the right atrium, going toward lead V1 (and hence a positive deflection), while left atrial depolarization is slightly later, going away from lead V1 and hence generating a negative waveform. With right atrial hypertrophy or abnormality, the depolarization is primarily toward lead V1, thereby generating a positive waveform. With left atrial hypertrophy or abnormality, the depolarization is primarily away from V1, thereby generating a negative waveform.

**Figure 14:** The direction of the waveforms or deflections of the QRS complex determine the letter applied. If the first deflection is negative it is termed a Q wave. Any first positive waveform (with or without a Q wave) is termed an R wave. A negative deflection after the R wave is termed an S wave. If there is a second positive deflection after the S wave it is called R’.
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than 1 (ie, normal R-wave progression). The transition point at which R/S is 1 or greater is leads V3-V4. This is termed R-wave progression (see figure 8).

An increased QRS amplitude in precordial and/or limb leads, which reflects increased voltage recorded at the body surface, is seen in young subjects, in those with thin chests and no lung disease, and in individuals with myocardial hypertrophy. Low QRS amplitude is defined as a QRS amplitude of 5 mm or less in each limb lead and/or less than 10 mm in each precordial lead. This may reflect a reduction in impulse conduction to the surface of the body, which may be due to lung disease, obesity, thick pericardium, pericardial effusion, or a reduction in myocardial muscle mass.

Small septal Q waves are often seen in limb leads and lateral precordial leads. Significant (pathologic) Q waves—that is, any Q wave in leads V1-V3 (although QS may normally be seen in leads V1-V2) or a Q wave more than 0.04 second in duration and more than 1 mm in depth in leads I, II, aVL, or aVF or in two consecutive leads V4-V6—are indicative of old myocardial infarction (MI). An isolated Q wave in lead III is of no significance as it may be normal. An infarction is diagnosed if there is also a significant Q wave in lead II and/or lead aVF.

The normal QRS complex duration is 0.06 to 0.10 second. A QRS complex duration longer than 0.10 second is considered an IVCD. When the QRS complex duration is 0.10 to 0.12 second, the IVCD is often called incomplete right bundle branch block (RBBB) or incomplete left bundle branch block (LBBB) if there is a morphology resembling either RBBB or LBBB. However, since His-Purkinje conduction is all or none and not incomplete, an IVCD (to either the right or left ventricle) is a more appropriate term. A QRS complex duration 0.12 second or

Figure 15: The QRS axis in the frontal plane is determined by analyzing the direction of the QRS complex in the limb leads. The heart is divided into four equal quadrants of 90° each (0° to +90°, +90° to +180°, 0° to –90° and –90° to +180°). The two leads that are perpendicular to each other and divide the heart in this fashion are leads I and aVF. Hence these two leads are looked at first. A normal axis is between 0° and +90°. A rightward axis, which is never normal, is between +90° and +180°. If the axis is leftward (ie, between 0° and –90°), it may be physiologic (and hence a normal leftward axis) if between 0° and –30° or pathologic (and hence abnormal) if between –30° and –90°. This is established by looking at lead II, which is perpendicular to –30°. If the QRS complex is positive in lead II, the axis is physiologically leftward; if the QRS complex is negative in lead II, the axis is pathologically leftward.
longer occurs with RBBB or LBBB; bundle branch blocks are associated with a specific QRS complex morphology. When the QRS complex morphology is without a specific pattern of a bundle branch block, it is an IVCD. A QRS complex duration of 0.16 to 0.22 second is often seen with severe cardiomyopathy, drug effect, or hyperkalemia. When the QRS complex duration is 0.24 second or longer, the cause is hyperkalemia.

QRS Axis

Frontal Plane

The QRS axis in the frontal plane is determined by analysis of the QRS complex direction in the limb leads (figure 15). The axis may be normal, leftward, rightward, or indeterminate, as established by an initial analysis of leads I and aVF, which are perpendicular to each other and divide the heart into four equal quadrants (0° to +90°, +90° to ±180°, 0° to –90° and –90° to +/-180°). An impulse going toward the left (toward 0°) is positive in lead I, and an impulse going toward the right (toward +180°) is negative in lead I. An impulse directed toward the foot (toward +90°) is positive in lead aVF, and an impulse directed away from the foot (toward –90°) is negative in lead aVF.

A normal axis is 0° to +90°. In this situation the QRS complex is positive in leads I and aVF. A leftward axis is 0° to –90° (the QRS complex is positive in lead I and negative in lead aVF). However, a leftward axis may be physiologic (normal) when it is between 0° and –30° or pathologic (abnormal) when it is between –30° and –90°. In the presence of a left axis, therefore, lead II is evaluated, as it is perpendicular to –30°. An impulse that is directed toward the foot (less negative than –30°) is positive in lead II, and an impulse that is directed away from the foot (more negative than –30°) is negative in lead II.

A physiologic left axis is 0° to –30° (the QRS complex is positive in leads I and II and negative in lead aVF). An extreme or pathologic left axis is –30° to –90° (the QRS complex is positive in lead I and negative in leads II and aVF, ie, rS complex). This is referred to as a left anterior fascicular block (LAFB). However, it is important to exclude inferior wall MI as a cause for left axis (ie, Qr complex in leads II and aVF). An LAFB cannot be established in the presence of an inferior wall MI. Limb lead switch must also be excluded.

A right axis is between +90° and +180° (the QRS complex is negative in lead I, ie, rS complex, and positive in lead aVF). This is referred to as a left posterior fascicular block (LPFB). However, other causes for a right axis must be excluded, including a lateral MI (Qr complex in leads I and aVL), right ventricular hypertrophy (RVH), Wolff-Parkinson-White pattern, dextrocardia, or right-to-left arm lead switch. An indeterminate axis, between –90° and +/-180° (the QRS complex is negative in leads I and aVF), is either an extreme leftward axis or an extreme rightward axis. There is no conduction sequence through the normal His-Purkinje system that will be associated with an indeterminate axis. Hence an indeterminate axis is seen whenever two abnormalities co-exist. For example, RVH, which is associated with a right axis, may co-exist with an LAFB, which is associated with a marked left axis, resulting in an indeterminate axis. Other situations include a lateral wall MI (with deep Q waves in leads I and aVL), which will present as a right axis, associated with an LAFB; a lateral wall MI
Podrid’s Real-World ECGs

Chest leads V1 to V6

Clockwise rotation (late transition)

Counter-clockwise rotation (early transition)

Beat-to-beat changes in the QRS axis and/or amplitude are called electrical (or QRS) alternans. There may also be beat-to-beat changes in T-wave amplitude/morphology, i.e., T-wave alternans.

ST Segment

The ST segment begins at the J point (the point of transition at the end of the QRS complex and the beginning of the ST segment) and ends at the onset of T wave (figure 17). It represents the period of time between the end of depolarization and the beginning of repolarization. The normal ST segment is slightly concave. The J point and ST segment are usually isoelectric or at zero potential, which is established by the TP segment. If the TP segment cannot be identified, as with a tachycardia (when the T and P waves are on top of each other), the PR segment is used to establish the isoelectric baseline.

Figure 16: The QRS axis in the horizontal plane is determined by analyzing the direction of the QRS complex in the precordial leads. This is established by imagining the heart as viewed from under the diaphragm, i.e., the right ventricle is anterior and the left ventricle is to the left. Here, the normal QRS transition point (R/S > 1) occurs at leads V3-V4. Clockwise rotation is present when the left ventricular electrical forces are shifted to the back and seen late in the precordial leads. Therefore, there is poor R-wave progression with late transition. R-wave amplitude increases slowly across the precordium, and the transition (R/S > 1) is later, between leads V4-V6. Counterclockwise rotation is present when the left ventricular electrical forces are shifted anteriorly and seen early in the precordial leads. There is early transition (R/S > 1 in lead V2) or a tall R wave in lead V2.
ST-segment flattening is a nonspecific change. J-point and ST-segment elevation are seen with various situations, including early repolarization (which may be seen when the QRS amplitude is increased as with young subjects or patients with left ventricular hypertrophy [LVH]), transmural ischemia (as may occur with coronary artery vasospasm), ST-segment elevation MI (STEMI, or transmural MI), or pericarditis.

J-point and ST-segment depression (upsloping, horizontal, or downsloping) are seen with subendocardial ischemia (LVH or coronary disease) and non-ST-segment elevation MI (NSTEMI, or subendocardial MI). ST-segment depression is considered significant if it is more than 1 mm below the baseline (ie, the TP segment). J-point depression and upsloping ST-segment depression may be seen as a normal finding in sinus tachycardia. This is due to alteration or depression of the J point by the T wave of the P wave (ie, atrial repolarization), which results from the shortening of the PR interval (due to sympathetic enhancement of AV nodal conduction) and movement of the T wave of the P wave from out of the QRS complex and onto the J point. As J-point depression and upsloping ST-segment depression may be a normal variant, the ST segment should be evaluated 80 msec past the J point, accounting for the effect of the T wave of the P wave. If at this point the ST segment is back to baseline, the ST-segment depression is a normal variant. If the ST segment is still more than 1.5 mm below baseline, then myocardial ischemia can be diagnosed.

A normal J point with ST-segment depression (sagging, scoped out, hammock-like) is seen as an effect of digoxin (not digoxin toxicity). J-point elevation and a normal ST segment may be an Osborne (J) wave, as seen with hypothermia.

**QT Interval**

The QT interval, indicating the time for repolarization, is measured from the onset of the QRS complex (either a Q or R wave) to the end of the T wave (see figure 9). The lead that has the best or sharpest T wave is used. As the QT interval includes the QRS complex, the presence of an increased QRS complex duration needs to be considered when measuring the QT interval, and any increase in QRS
Podrid’s Real-World ECGs

The Basics: Introduction

Complex duration (msec) above normal (i.e., 0.06 to 0.10 sec) needs to be subtracted from the QT-interval measurement. Thereafter, the QT interval needs to be corrected for heart rate. The normal QTC is 0.44 to 0.48 second or less.

A long QT interval may result from delayed or prolonged repolarization. With delayed repolarization, the ST-segment duration is long while the T-wave duration is normal. This is seen with metabolic abnormalities, particularly low calcium or low magnesium levels. In prolonged repolarization, the ST segment is normal in duration but the T wave is broad or prolonged. This is due to drugs (acquired QT prolongation) or a genetic abnormality producing a channelopathy (congenital long QT syndrome). Congenital QT prolongation may have a prominent U wave interrupting the T wave (QT-U wave).

A short QT interval is due to a metabolic abnormality (high calcium or high magnesium levels) or a congenital short QT syndrome.

T Waves / U Waves

Normal T waves are asymmetric, regardless of amplitude, with a slower upstroke than downstroke (Figure 18). The hyperacute T wave is tall, peaked, and symmetric, as can be seen with hyperkalemia (systemic or localized as in acute MI). The T-wave upstroke and downstroke are smooth. Any notches, bumps, or irregularities of the T wave suggest a superimposed P wave. T-wave abnormalities can be flat, biphasic, or inverted. They are very common and may be seen in many situations, including ischemia (inverted symmetric T waves usually associated with ST-segment changes), LVH, pericarditis/myocarditis, metabolic abnormalities, anemia, fever, lung disease, enhanced catecholamine state, pH changes, use of certain drugs, central nervous system abnormalities, or even as a normal physiologic change. T-wave abnormalities may also be nonspecific when there is no clinical history to suggest a cause.

The U wave is an upright waveform following the T wave. It is normally seen in the right precordial leads. Increased U-wave amplitude and U waves that are present more diffusely in all precordial leads, and often in the limb leads as well, are seen with hypokalemia. A U wave may also be seen in congenital long QT syndrome, in which case the U wave interrupts the T wave (QT-U wave). Negative U waves, particularly during exercise testing, are suggestive of ischemia as a result of a stenosis of the left anterior descending artery.
Core ECGs
A 32-year-old man presents to an outpatient clinic with complaints of a productive cough, headache, shortness of breath, and fever. A routine ECG is obtained.

Are there any abnormalities of concern on the ECG?
Podrid’s Real-World ECGs

ECG 1 Analysis: Normal sinus rhythm, normal axis and interval
There is a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6; it has a normal morphology and duration (0.12 sec). Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and it has a normal morphology. There is normal R-wave progression across the precordium, and the transition (R/S > 1) is at lead V3. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The ST segment, which begins at the J point (▲) and ends at the beginning of the T wave, is at baseline (which is defined by the TP segment), and it has a normal concave morphology. The QT interval is 400 msec (QTc = 400 msec). The T wave (↓) has a normal morphology; it is asymmetric with a slower upstroke and more rapid downstroke. A low-amplitude waveform can be seen following the T wave in leads V2-V3; this is a U wave (↑). Hence this is a normal 12-lead ECG.
A 22-year-old woman with scleroderma presents to your office with progressive dyspnea on exertion. There is a loud P2 with a holosystolic murmur that exhibits respirophasic variation, heard best at the lower left sternal border. A routine ECG is obtained.

What is the most likely cause of the patient’s symptoms?
What ECG findings support this diagnosis?
ECG Analysis: Sinus tachycardia, right atrial hypertrophy, right ventricular hypertrophy (RVH), right axis deviation, counterclockwise rotation
The ECG shows a regular rhythm at a rate of 110 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6, and it has a normal duration (0.10 sec). Hence this is a sinus tachycardia. The P waves are abnormal, and they are tall, narrow, and peaked in leads II, aVF, and V1-V2. The P-wave morphology is characteristic of a P pulmonale, a result of right atrial hypertrophy. This may also be termed a right atrial abnormality.

The QRS complex duration is normal (0.08 sec). The QT/QTc intervals are normal (300/410 msec). However, the QRS complex has an abnormal morphology. The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). The major finding is a tall R wave in lead V1 (←), defined as an R wave taller than 7 mm (seven small boxes) or an R/S > 1. The tall R wave in lead V1 along with the rightward axis and a P pulmonale (right atrial hypertrophy) are characteristic of right ventricular hypertrophy (RVH). In addition, there is a tall R wave in lead V2 (↓). Although this can be the result of RVH, it may also represent counterclockwise rotation of the electrical axis in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm; the right ventricle is in front and the left ventricle is to the left side. With counterclockwise rotation there is early transition, that is, left ventricular forces are shifted anteriorly and occur earlier in the precordial leads.

The diagnosis of RVH is often difficult to establish as the QRS complex primarily represents depolarization of the left ventricle, which has a far greater myocardial mass compared with the right ventricle. Hence the finding of RVH on the ECG implies a substantially thickened right ventricular myocardium.

The criteria for the diagnosis of RVH include:

- R-wave amplitude (in mm) in lead V1 > 7 mm
- R/S ratio in lead V1 > 1
- R/S ratio in lead V6 (or V5) < 1, indicating increased forces directed from the left to the right

Supporting criteria for RVH include:

- Right axis deviation (between +90° and +180°), which is diagnosed by a QRS complex that is negative in lead I and positive in lead aVF
- Right atrial hypertrophy (P pulmonale); the P wave is tall (> 0.25 mV), narrow (< 0.12 sec), and peaked in the limb leads and positive in lead V1
- Associated ST-segment depression and T-wave abnormalities in leads V1-V3

continues
The combination of RVH and right atrial hypertrophy on the ECG and a loud P2 on exam suggests the presence of elevated pulmonary pressures. Pulmonary arterial hypertension is clinically associated with scleroderma and is the most likely diagnosis.

It is important to note that there are other causes for a tall R wave in lead V1 that need to be excluded, although the presence of the other ECG features will generally establish RVH as the etiology. The other causes include a posterior wall myocardial infarction (MI) (which is usually associated with an inferior wall MI), Wolff-Parkinson-White pattern (with a short PR interval and a QRS complex that is widened as a result of a delta wave), hypertrophic cardiomyopathy with septal hypertrophy (often with prominent septal Q waves in the lateral leads), early transition (counterclockwise rotation), Duchenne muscular dystrophy (associated with a posterolateral MI pattern), dextrocardia (associated with reverse R-wave progression, right axis, and negative P wave in lead I), and lead switch (V1, V2, V3); it may also be a normal variant. n
A 72-year-old man who has not seen a physician in more than 20 years comes to your clinic with no specific complaints. Physical examination reveals a blood pressure of 185/100 mm Hg, equal in both arms, as well as a left ventricular heave and an S4. An ECG is obtained.

What are the pertinent findings on the ECG?
What is the clinical diagnosis?
What is the most likely etiology?
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, left atrial hypertrophy, left ventricular hypertrophy (LVH)
The ECG shows a regular rhythm at a rate of 88 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The P wave is broad (> 0.12 sec), notched (∗) in leads II and V4 (termed P mitrale), and negative (↑) in leads V1 and V2. This is characteristic of left atrial hypertrophy, also termed left atrial abnormality.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (the QRS complex is positive in leads I and aVF). The QT/QTc intervals are normal (360/440 msec). The major finding is the marked increase in QRS voltage (R-wave amplitude or S-wave depth) seen in the precordial leads (S-wave depth in lead V2 = 39 mm [ ] and R-wave amplitude in lead V5 = 40 mm [ ] for a total of 79 mm), which is diagnostic for left ventricular hypertrophy (LVH) (i.e., S-wave depth in lead V2 + R-wave amplitude in lead V5 ≥ 35 mm).

Associated with LVH are ST-T wave changes or repolarization abnormalities seen in leads V4-V6 (↑). These ST-T wave changes, often referred to as a “strain pattern,” actually reflect subendocardial ischemia. The last portion of the myocardium to receive blood supply is the subendocardium, and when LVH is present the oxygen supply to this territory is limited.

A number of criteria have been proposed for diagnosing LVH on the surface ECG. They are primarily related to QRS complex amplitude or voltage. However, an important limitation to the use of QRS complex voltage is related to factors that may influence the transmission of the electrical impulse to the surface of the body, including body habitus (especially obesity), pulmonary disease, and pericardial thickening or effusion. In these settings, the ECG may not reflect the presence of LVH. The proposed criteria for diagnosing LVH include:

- S-wave depth (in mm) in lead V1 or V2 + R-wave amplitude (in mm) in lead V5 or V6 ≥ 35 mm if over age 45 or ≥ 45 mm if under age 45 (Sokolow-Lyon criteria)
- Deepest S wave (in mm) + tallest R wave (in mm) in any two precordial leads ≥ 35 mm (or ≥ 45 mm if under age 45)
- S-wave depth (in mm) or R-wave amplitude (in mm) in any one precordial lead ≥ 25 mm
- R-wave amplitude (in mm) in lead aVL ≥ 11 mm (≥ 18 mm in presence of left axis) (Sokolow-Lyon criteria)
- R-wave amplitude (in mm) in any one limb lead ≥ 20 mm
- R-wave amplitude (in mm) in lead aVL + S-wave depth (in mm) in lead V3 ≥ 28 mm for men or ≥ 20 mm for women (Cornell criteria)

The voltage criteria are based on the ECG recorded at normal standard, where 1 mV = 10 mm or 10 small boxes in height.
LVH may be associated with other changes on the ECG, including:

- Intraventricular conduction delay due to slow activation of the thickened myocardium.
- Often the upstroke of the QRS complex is prolonged (> 0.05 sec); this is termed a delayed intrinsicoid deflection (the intrinsicoid deflection is measured from the beginning of the QRS complex to the peak of the R wave).
- Physiologic left axis deviation, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVf).
- Presence of left atrial hypertrophy (or abnormality), called a P mitrale, defined as a P wave that is broad (> 0.12 sec in duration) and notched (with a tall second component). Left atrial hypertrophy may also be present when the P wave in lead V1 (and V2) is primarily negative (rather than biphasic positive-negative, which is normal).
- Ischemic-type ST-T wave abnormalities (ie, due to subendocardial ischemia), most often seen in leads I, aVL, and V4-V6.
- J-point and ST-segment elevation (early repolarization), most often seen in leads V4-V6.

As indicated, QRS amplitude, as measured at the surface of the body, is affected by a number of conditions; therefore, LVH may be present even if QRS amplitude criteria are not. This is the basis for the Romhilt-Estes scoring system, which assigns a point score to various ECG abnormalities seen with LVH.

**Romhilt-Estes Scoring System**

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<tr>
<th>Romhilt-Estes Criterion</th>
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<td>R-wave height or S-wave depth in any limb lead ≥ 20 mm</td>
<td>3</td>
</tr>
<tr>
<td>OR S-wave depth in lead V1 or V2 ≥ 30 mm</td>
<td>3</td>
</tr>
<tr>
<td>OR R-wave height in lead V5 or V6 ≥ 30 mm</td>
<td>3</td>
</tr>
<tr>
<td>ST-T wave changes typical of LVH</td>
<td>1</td>
</tr>
</tbody>
</table>

Taking digoxin | 1 |

Not taking digoxin | 3 |

Left atrial hypertrophy

- (terminal force in lead V1 ≥ 1 mm in depth and > 0.04 sec in duration) | 3 |
- Left axis deviation (< –30°) | 2 |
- QRS duration ≥ 90 ms (ie, intraventricular conduction delay) | 1 |
- Intrinsicsoid deflection in lead V5 or V6 > 0.05 sec | 1 |

*An score of 5 or more indicates definite LVH; a score of 4 indicates probable LVH.*

The patient has physical exam findings and ECG features all consistent with LVH, most likely from chronic untreated hypertension. Other etiologies of LVH include aortic stenosis, coarctation of the aorta, extreme athleticism, and hypertrophic cardiomyopathy from genetic mutations. The mainstay of treatment is adequate blood pressure control for hypertension, particularly with β-blockers, calcium-channel blockers, angiotensin-converting enzyme inhibitors, or angiotensin receptor blocking drugs; surgery for coarctation or aortic stenosis; and assessment of hemodynamic abnormalities and ventricular arrhythmia risk in patients with hypertrophic cardiomyopathy.
A 61-year-old woman with hypertension as her only known risk factor for coronary artery disease now presents with 3 days of persistent chest pressure. Sublingual nitroglycerin does not relieve her symptoms. An ECG is obtained, and an initial set of cardiac biomarkers reveals normal troponin, creatine kinase (CK), and CK-MB levels.

Based on the ECG, is this patient having an acute coronary syndrome?
Podrid’s Real-World ECGs

ECG 4 Analysis: Normal sinus rhythm, left ventricular hypertrophy (LVH) with ST-T wave changes
The ECG shows a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec), and the axis is leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (360/410 msec). The major finding is an increase in QRS voltage in lead I (R-wave amplitude = 20 mm) and lead aVL (R-wave amplitude = 24 mm), which is diagnostic for left ventricular hypertrophy (LVH) (ie, voltage in any limb lead > 20 mm or R-wave amplitude in lead aVL > 11 mm or > 18 mm in the presence of a left axis). Also noted is a delayed intrinsicoid deflection in leads V4-V6 (↑), an intraventricular conduction delay (ie, QRS width ≥ 0.10 sec), leftward axis, and the ST-T wave changes (↑) associated with LVH as noted in leads I, aVL, and V4-V6.

Typically, the ST-T wave abnormalities associated with LVH consist of ST-segment depressions and deep, asymmetric T-wave inversions, as illustrated in this case. Therefore, in the presence of LVH with the associated ST-T wave abnormalities, which are due to chronic subendocardial ischemia, the ECG cannot definitively diagnose nor can it rule out acute ischemia or a non–ST-segment elevation myocardial infarction (STEMI) due to coronary artery disease. Although the clinical story is potentially concerning for an acute coronary syndrome, the ECG in this setting does not help to make the diagnosis. In contrast, the presence of ST-segment elevations is indicative of an acute ST-segment elevation myocardial infarction (STEMI) despite the presence of LVH. In this case, the LVH could be explained by the patient’s chronic hypertension.
A 54-year-old woman presents to the emergency department after a syncopal episode. She has had three similar episodes in the past and has noted worsening chest pressure with exertion over the past many months. On physical examination, you hear a grade III/VI systolic murmur, loudest at the upper sternal border, that radiates to the carotids and does not increase with Valsalva. The murmur is mid to late peaking. S2 is heard but does not split. An ECG is obtained.

What is the most likely diagnosis?
ECG Analysis: Normal sinus rhythm, left ventricular hypertrophy (LVH) with ST-T wave changes
The ECG shows a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/410 msec). The striking feature is the very markedly increased QRS voltage in leads V4-V6 (R-wave amplitude = 40 to 50 mm), which meets one of the criteria for left ventricular hypertrophy (LVH) (S-wave depth or R-wave amplitude in any precordial lead > 25 mm). In addition, there are marked ST-T wave changes (↑) associated with the LVH (asymmetric and deeply inverted) in leads I, II, aVR (the positive T wave is actually inverted in this lead), and V4-V6.

Given the clinical history, physical exam, and ECG findings, the most likely diagnosis is valvular aortic stenosis. The classic triad of symptoms associated with severe aortic stenosis is angina, syncope, and heart failure. The murmur associated with aortic stenosis is a crescendo–decrescendo systolic (ejection type) murmur best heard at the right upper sternal border. The timing of the murmur (early, mid, or late) correlates with the severity of aortic stenosis. The quality of the carotid pulse also is an indicator of the severity of aortic stenosis (ie, parvus [low amplitude] and tardus [slow upstroke]). Physiologic splitting of S2 can also be lost due to delayed opening and closure of the aortic valve. When valvular stenosis is very severe, paradoxical splitting of S2 may be present. Aortic stenosis is associated with findings of LVH and occasionally a left bundle branch block on the ECG. The major etiologies of valvular aortic stenosis include calcific or senile changes (predominant in the over-70 age group), rheumatic heart disease, and congenital bicuspid aortic valve.
A 74-year-old man presents to his physician for a routine physical examination. He states that his only medical history is that of hypertension. A routine ECG is obtained.

Does this patient have left ventricular hypertrophy?
Podrid's Real-World ECGs

ECG Analysis: Normal sinus rhythm, left ventricular hypertrophy, half-standard
The ECG shows a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex has a normal morphology. The QT/QTc intervals are normal (360/390 msec). Although the voltage in all leads is normal as recorded, it is important to note the standardization (^) seen at the end of the ECG tracing, which indicates that the limb leads were recorded at normal standard (1 mV = 10 mm or 10 small boxes in height) while the precordial leads were recorded at half-standard (1 mV = 5 mm or five small boxes in height). Hence the QRS amplitude as measured in the precordial leads needs to be doubled. Therefore, the R-wave amplitude in lead V5 is 24 mm and the S-wave depth in leads V1 and V2 is 26 and 14 mm, respectively, for a total of 50 mm using S-wave depth in lead V1 + R-wave amplitude in lead V5 and 38 mm using S-wave depth in lead V2 + R-wave amplitude in lead in V5. This meets one of the criteria for left ventricular hypertrophy (S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). Also present are the typical ST-T wave changes associated with left ventricular hypertrophy (↑) seen in leads I, II, aVR (the positive T wave is actually inverted in this lead), and V4-V6.
A 45-year-old man with no past medical history presents with symptoms of “indigestion,” with chest burning and throat tightness over the past 2 weeks occurring with activity. Over the past 2 days he experienced chest burning at rest. He has a 25 pack-year history of smoking, and his father died of a myocardial infarction at the age of 52. An ECG is obtained. Cardiac troponins are negative.

What is the diagnosis on ECG?

What is the next step in the management of this patient?
ECG 7 Analysis: Normal sinus rhythm, upsloping
ST-segment depression due to subendocardial ischemia
The ECG shows a regular rhythm at a rate of 92 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/420 msec). The major finding is J-point depression (↓) with upsloping ST-segment depression (^) seen in leads II, III, aVF, and V2-V6 and ST-segment elevation (▼) in lead aVR (which is actually ST-segment depression). J-point and ST-segment depression is an indicator of subendocardial myocardial ischemia; the subendocardium of the myocardium is the last region to receive blood flow and hence is prone to ischemia. Ischemia usually occurs first in the subendocardial region before extending to the entire thickness of the myocardium.

The J point and ST segment should be at baseline, which is determined by the TP segment; the PR segment can be used to establish the baseline if there is no obvious TP segment. Three types of ST-segment depression (J point and ST segment below baseline) are indicative of myocardial ischemia: upsloping, horizontal, and downsloping. Upsloping ST-segment depression is the least specific for myocardial ischemia as it may occur with sinus tachycardia as a normal finding. In this situation, the J-point depression results from the atrial repolarization (the T wave of the P wave). Normally, the T wave of the P wave occurs during the QRS interval. During sinus tachycardia (which is the result of an augmented sympathetic state) and the shortening of the PR interval (due to enhanced AV nodal conduction), the T wave of the P wave moves out from the QRS complex and falls on the J point, depressing it. The ST segment therefore slopes back up toward baseline. As a result, when upsloping ST-segment depression is present, the amount of depression is determined by evaluating the degree of ST-segment depression at 80 msec past the J point. An ST segment that is more than 1.5 mm below baseline at this time is diagnostic for ischemia. The baseline is the TP segment, although with tachycardia the TP segment may be difficult to establish; hence the PR segment may be used. In this ECG, sinus tachycardia is not present and the ST segment is still depressed 2 mm below baseline at 80 msec beyond the J point, confirming that these are true ischemic ECG changes.

continues
The patient’s symptoms and ECG findings are concerning for an acute coronary syndrome. The spectrum of acute coronary syndrome includes unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Since cardiac biomarkers are negative, the patient would fall into the unstable angina category. His symptoms first began as chronic stable angina of effort but became worse and unstable as they also occurred at rest. The first step in the management of a patient with unstable angina includes administration of aspirin, oxygen, nitrates, and β-blockers. Anticoagulation with some form of heparin should also be initiated, and antiplatelet therapy with clopidogrel along with aspirin has been shown to have a mortality benefit. A high-dose statin is also recommended, regardless of the lipid levels, as these agents have been shown to stabilize unstable plaques, which are the cause of unstable angina. Once the patient’s symptoms stabilize and the patient is free of angina, a noninvasive evaluation (exercise test) should be performed to determine whether there is any ongoing ischemia. If so, cardiac catheterization may be performed.
A 74-year-old veteran presents with symptoms of angina. The following ECG is obtained.

Which coronary arteries are likely to have significant stenoses?
Podrid's Real-World ECGs

ECG Analysis: Sinus tachycardia, horizontal
ST-segment depression due to subendocardial ischemia, low voltage
The ECG shows a regular rhythm at a rate of 110 bpm. There are P waves (+) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus tachycardia.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/380 msec). There is low QRS voltage (< 5 mm in each limb lead and/or < 10 mm in each precordial lead). Diffuse ST-segment depressions (↑) can be seen in all leads (the ST-segment elevation in lead aVR [▼] is actually ST-segment depression). The ST-segment depression is horizontal in leads I and V3-V6, reaching a maximal depth in lead V3 of 5 mm below baseline, which is the TP segment (↓). The ST segments are downsloping in leads II, III, aVF, and V2. Although the leads in which ST-segment depression are seen do not generally indicate the location of subendocardial ischemia, the diffuse nature of the ST-segment depression in this case indicates widespread ischemia due to multivessel, likely three-vessel, coronary artery disease.
The patient is a 60-year-old man with hypertension and known coronary artery disease who underwent percutaneous coronary intervention (balloon angioplasty) of the right coronary artery and left anterior descending artery 3 years ago as therapy for angina. He presents with 2 months of recurrent angina. The baseline ECG following an exercise test showed left ventricular hypertrophy with 1-mm ST-segment depression in leads I, aVL, and V4-V6. This ECG was obtained 3 minutes into the recovery period.

Does this patient have ischemia?
ECG Analysis: Normal sinus rhythm, downsloping
ST-segment depression due to subendocardial ischemia
The ECG demonstrates a regular rhythm at a rate of 80 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec) and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/420 msec). The major finding is significant ST-segment depression (↑), which is downsloping. Although there is voltage criteria for left ventricular hypertrophy (LVH) (ie, R-wave amplitude of 25 mm in lead II [ ]), the ST segments are more depressed than is generally seen with LVH and are more depressed compared with baseline (ie, > 1 mm), reaching a maximum level of 5 mm below the baseline TP segment. Downsloping ST-segment changes have the strongest correlation with ischemia and generally indicate more severe coronary artery disease, particularly when the changes are widespread.

However, ST-segment depression can be nondiagnostic for ischemia due to coronary artery disease if the patient has LVH (which is associated with subendocardial ischemia not due to coronary artery disease) or routinely takes digitalis. The ST-segment changes seen with digitalis drugs are different as the J point is usually at baseline while there is sagging depression of the ST segment (hammock-like or scooped). Nevertheless, significant ST-segment depression beyond that seen at baseline is indicative of ischemia despite the present of LVH or digitalis use. n
A 56-year-old diabetic man presents with acute-onset crushing substernal chest pressure over the past 2 hours. The following ECG is obtained.

What is the diagnosis?
What is the next step in management?
Podrid's Real-World ECGs

ECG 10 Analysis: Normal sinus rhythm, ST-segment elevation due to acute anterior wall myocardial infarction (MI), left anterior fascicular block (LAFB)
The ECG shows a regular rhythm at a rate of 90 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I but a negative QRS complex in leads II and aVF with an rS morphology). An extremely leftward axis (in the absence of other etiologies for a left axis, specifically an inferior wall myocardial infarction [MI]) is termed left anterior fascicular block (LAFB). The criteria for LAFB include left axis deviation with an rS pattern in leads II and aVF. In contrast, a Qr complex in leads II and aVF is a pattern associated with a chronic inferior wall MI, not a conduction abnormality or fascicular block. Indeed, LAFB cannot be diagnosed in the presence of an inferior wall MI. The QT/QTc intervals are normal (320/390 msec).

The major abnormality is significant J-point (→) and ST-segment (↓) elevation (when compared with the TP segment, which is baseline) in leads V2-V5 and less marked ST-segment elevation in leads I and aVL (▲). The ST segments are no longer concave but are now convex in morphology and merged with the T waves. In addition there is loss of the R wave in leads V2-V3 (▼). These ECG changes are diagnostic for an acute MI involving the apex and anteroapical regions of the left ventricle. There is also involvement of the lateral wall, indicated by the ST-segment elevation in leads I and aVL as well as ST-segment depression (▲) in leads III and aVF. In the setting of an acute MI, these ST-segment depressions do not represent ischemia in the inferior wall, but rather they are reciprocal changes due to the fact that these leads are seeing the infarcted region from an opposite direction. When an ST-segment elevation MI (STEMI) is present, immediate revascularization is indicated with either cardiac catheterization and percutaneous coronary intervention or thrombolytic therapy.

In the setting of an acute STEMI, the ECG demonstrates a typical progression of changes. During the first several minutes after the onset of chest discomfort, the earliest ECG changes indicating an acute MI are hyperacute (tall, peaked, and symmetric) T waves. This T-wave abnormality is the result of local hyperkalemia, which occurs early after the infarction as a result of loss of membrane integrity from the ischemia with a leaking of potassium; the potassium stays in the infarcted tissue as there is no blood flow into or out of the infarction zone. This initial ECG finding is often not seen because it is present soon after the onset of symptoms, while patients usually present several hours after symptoms begin. The T-wave changes are followed by ST-segment elevation. The ST segments initially maintain their normal concave morphology, but they become convex as the MI evolves, merging with the T waves. Over the ensuing hours to days, the ST-segment elevation persists, the R wave loses amplitude, and Q waves begin to continue
develop. As the infarct evolves, the ST-segment elevation decreases, the Q waves become deeper, and T-wave inversions develop. In the absence of revascularization, the ST-segment elevations normalize over several days. The Q waves and T-wave inversions persist, and this is the pattern of a chronic MI.

Therefore, an acute MI is identified by the presence of localized ST-segment elevation; hyperacute T waves, which are tall, peaked, and symmetric; and reciprocal ST-segment depressions, which are the same ST-segment changes viewed from another direction. The leads that demonstrate these changes identify the location of the acute MI:

- **Inferior wall MI:** ST-segment elevation in leads II, III, and aVF (or any two of these three leads). An inferior wall MI may be associated with involvement of the posterior left ventricular wall or the free wall of the right ventricle. The presence of ST-segment elevation in lead V1 and ST-segment depression in lead aVR (which is actually elevation) suggests involvement of the right ventricle, which can be confirmed by obtaining right-sided precordial leads. The presence of ST-segment elevation in RV3-RV4 (right-sided leads V3-V4) confirms involvement of the free wall of the RV.

- **Anterior wall MI:** ST-segment elevation in any two contiguous precordial leads (V1-V6):
  - Anteroseptal MI: ST-segment elevation in leads V1-V2
  - Anteroapical MI: ST-segment elevation leads V3-V4
  - Anterolateral MI: ST-segment elevation leads V5-V6

- **Lateral wall MI:** ST-segment elevation leads I and aVL

- **Posterior wall MI:** ST-segment depression in leads V1-V2, particularly when there is an inferior wall MI, suggests posterior wall involvement, which is often associated with inferior wall MI. Leads placed on the back (V7-V8), below the left scapula and over the posterior wall, may show ST-segment elevation, confirming a posterior wall infarction.
A 67-year-old man presents with chest pain, and the following ECG is obtained.

What is the underlying mechanism for the findings noted in leads V4-V6, I, and aVL?
 Podrid’s Real-World ECGs

ECG 11 Analysis: Normal sinus rhythm, ST-segment elevation due to acute anterolateral and lateral myocardial infarction, left atrial hypertrophy, low voltage
The ECG shows a regular rhythm at a rate of 96 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/400 msec).

The major finding is localized ST-segment elevation (↓) in leads V4-V6, I, and aVL, diagnostic for an acute lateral and anterolateral myocardial infarction. The ST segments have become convex and are merged with the T waves, resulting in a QRS complex that looks like a monophasic action potential or a current of injury; this has also been referred to as “tombstoning.” Reciprocal ST-segment depressions (▲) are seen in leads III and aVF. In addition, there is low voltage in the limb leads (QRS complexes of < 5 mm in each lead) and the precordial leads (QRS complexes of < 10 mm in each lead). Lastly, the deep and broad negative P waves in leads V1-V2 (▲) are indicative of left atrial hypertrophy.

According to the diastolic current theory, injured myocardial cells release ions that result in partial depolarization of the territory at baseline. Thus, the original baseline voltage (TP segment) in the affected leads on the ECG is actually shifted downward. During electrical activation of the myocardium, the baseline is reset to true zero as there is no electrical activity in the infarcted tissue; thus on the ECG recording, the ST segments appear to be elevated from the original baseline.
A 47-year-old man with hypertension and dyslipidemia presents with acute-onset crushing substernal chest pressure. Initial vital signs are within the normal range, with a blood pressure of 135/85 and a pulse rate in the 90s. After an aspirin is administered, the patient receives sublingual nitroglycerin. He subsequently becomes pre-syncopal, with a blood pressure of 70/palp and a pulse rate in the 100s. He must be intubated for airway protection in the setting of altered mental status.

What is the diagnosis?
What is a likely explanation for the patient’s hypotension?
Podrid’s Real-World ECGs

ECG 12 Analysis: Normal sinus rhythm, elevation due to acute inferior wall myocardial infarction (MI), acute right ventricular infarction
The ECG shows a regular rhythm at a rate of 96 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/400 msec).

There is significant ST-segment elevation (↓) in leads II, III, and aVF, which is diagnostic for an acute inferior wall myocardial infarction (MI). The ST-segment elevation seen in lead V1 (▼) is strongly suggestive of right ventricular free wall infarction, which should be confirmed using right-sided chest leads (ie, the six chest leads placed on the right side of the chest in the same positions as used on the left side of the chest); the presence of ST-segment elevation in RV3-RV5 (right-sided leads V3-V5) would establish a right ventricular free wall infarction. Also noted are ST-segment depressions in leads I and aVL (▲), which represent reciprocal changes (ie, the same changes of the acute inferior infarction seen from another direction).

Hypotension in the setting of an acute coronary syndrome can be a manifestation of multiple etiologies. First, significant left ventricular dysfunction can lead to poor stroke volume and a resultant drop in blood pressure. Second, vagal reflexes (eg, the Bezold-Jarisch response), which often occur in the setting of inferior MI, can predominate, and the patient becomes bradycardic and hypotensive. However, this patient did not become bradycardic. Third, right ventricular infarction can result in a reduction in both right ventricular stroke volume and left ventricular filling and hence stroke volume. Therefore, patients with a right ventricular infarction are preload dependent, requiring adequate volume to maintain cardiac output. Given the ST-segment elevation in lead V1, this patient likely has right ventricular involvement. In patients with right ventricular involvement, care should be taken in giving agents that reduce preload, such as nitrates, diuretics, and morphine. Indeed, these patients often require intravenous fluid to maintain blood pressure.

Because the acute right ventricular marginal branches of the right coronary artery supply the right ventricle, inferior infarctions are the type of MI most commonly associated with right ventricular infarcts. Anterior MIs can also result in right ventricular infarcts due to blood supply from septal branches.
A 74-year-old woman presents with progressive symptoms of dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. She has noted a 15-lb weight gain over the past 2 months. She denies any current or past chest pain. On physical examination, jugular venous pressure is 12 cm, crackles are audible, and there is bilateral lower extremity edema. An ECG is obtained.

What is the most likely etiology for the patient’s symptoms?
What is the initial step in management?
**Podrid’s Real-World ECGs**

**ECG 13 Analysis:** Normal sinus rhythm, intraventricular conduction delay, chronic (old) anterior wall myocardial infarction (MI)
The ECG shows a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is prolonged (0.12 sec), and there is an intraventricular conduction delay. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec). The major abnormality is the absence of R waves in leads V3-V5, resulting in complexes that have a QS pattern (↓) and also a pathologic Q wave (defined as being > 0.04 sec in duration) in lead V6 (∧). The presence of significant Q waves indicates that the initial electrical forces are going away from the lead involved, which means that the tissue under the lead is electrically silent or infarcted. Hence, the QS complexes and Q wave on this ECG are indicative of an old or chronic myocardial infarction (MI) involving the anteroapical and anterolateral walls of the left ventricle. The T waves are biphasic in leads V2-V3 (∗), although often they are inverted in the leads that demonstrate an infarction pattern. There are also small Q waves (∧) in leads II, III, and aVF; however, they are narrow and represent normal septal depolarization.

The patient is exhibiting symptoms of biventricular heart failure, likely resulting from an anterior MI she suffered sometime in the past. Since the patient denies any previous symptoms of chest discomfort, the infarction is determined to have been silent. The age of the Q wave or transmural infarction cannot be ascertained from the ECG. Initial steps in management include diuresis for fluid management and hydrazaline and nitrates or an angiotensin-converting enzyme (ACE) inhibitor for preload and afterload reduction. β-blockers have also been shown to have a significant mortality benefit in patients with heart failure with reduced ejection fraction. Revascularization of the coronary artery stenosis that resulted in the Q-wave MI (likely a lesion of the left anterior descending artery) has not been shown to be associated with a mortality benefit once the infarct is more than 3 days old (ie, a chronically occluded coronary artery). However, an ischemic evaluation with stress testing would be reasonable once the patient has been effectively treated for the heart failure to assess the level of any residual ischemic burden as well as establish functional capacity and status.
Notes
A 68-year-old man presents to his physician because of the recent onset of a productive cough associated with slight fever and shortness of breath. He has a history of a previous myocardial infarction (MI) but denies any recent cardiac symptoms. Physical examination demonstrates bilateral rhonchous sounds. An ECG is obtained and a chest X-ray is negative. The patient is diagnosed with bronchitis, and antibiotics are prescribed.

Does this patient have a posterior wall MI?

What is this patient’s axis of ventricular depolarization in both the anterior (limb leads) and horizontal (precordial leads) planes?

What are the causes for these abnormal findings?
ECG 14 Analysis: Normal sinus rhythm, first-degree AV block, chronic (old) inferior wall MI, counterclockwise rotation
The ECG shows a regular rhythm at a rate of 64 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm with a first-degree AV block.

The QRS complex duration is normal (0.08 sec), and the axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, the QRS complex in leads II, III, and aVF has a QS pattern (↓), indicating that the initial electrical forces are going away from the inferior wall. In addition, the T waves are inverted (↑) in these leads. This pattern represents an old or chronic inferior wall myocardial infarction (MI). The QT/QTc intervals are normal (360/370 msec).

Although the QRS complex is positive in lead I and negative in leads II and aVF, this does not represent a conduction abnormality (ie, left anterior fascicular block [LAFB], in which the QRS morphology in leads II and aVF would have an rS configuration). In this case the QRS complex has a QS pattern representing an inferior wall MI, which accounts for the left axis in the frontal plane. The diagnosis of an LAFB cannot be made in the presence of an inferior wall MI.

Although there is a tall R wave in lead V2 (→), the R wave in lead V1 is not increased in amplitude. Hence this is not indicative of posterior wall involvement. Rather, the tall R wave in lead V2 (R/S > 1) is a result of counterclockwise rotation of the electrical axis in the horizontal plane. This is also called early transition. The axis in the horizontal plane can be determined by imagining the heart as viewed from under the diaphragm. The right ventricle is in front, while the left ventricle is to the left side. When there is counterclockwise rotation, the left ventricular forces are shifted anteriorly and are seen earlier in the precordial leads (eg, in lead V2), presenting with a tall R wave in this lead.

Also noted are T-wave inversions in leads V4-V6 (↑). T-wave inversions are nonspecific, although in the setting of known coronary artery disease (ie, prior inferior wall MI) T-wave inversions are often interpreted as representing ischemia. However, T-wave inversions must be interpreted in association with the clinical situation.
A patient comes to your clinic for the first time and reports a history of prior myocardial infarction (MI). You obtain an ECG.

Is an infarction present?
If so, which myocardial territory or territories have been infarcted?
ECG 15 Analysis: Normal sinus rhythm, chronic (old) inferior wall MI, chronic (old) posterior wall MI, counterclockwise rotation
The ECG shows a regular rhythm at a rate of 90 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6, indicating a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a leftward axis. However, the left axis is the result of significant Q waves (↑) (width of Q waves > 0.04 sec) in leads II, III, and aVF (Qr pattern) that indicate an old inferior wall myocardial infarction (MI). If this leftward axis were the result of a conduction abnormality, the QRS morphology in leads II, III, and aVF would have an rS pattern. In addition, there is a prominent R wave (←) in lead V1 (R/S > 1). When associated with an inferior wall MI, this represents posterior wall involvement. Although the R wave in lead V2 is also tall (→), this is in part the result of counterclockwise electrical rotation in the horizontal plane or early transition, which represents left ventricular forces occurring earlier in the precordial leads. Therefore, the patient has had an inferoposterior MI. The QT/QTc intervals are normal (320/390 msec).
A 72-year-old woman presents with worsening dyspnea over the past 2 days. She reports having an episode of nausea, vomiting, shortness of breath, and diaphoresis 2 weeks ago that lasted for 12 hours and then gradually resolved on its own. She also recalls feeling a strange sensation in her chest, described as mild substernal discomfort. Her past medical history is significant for diabetes, and she has no documented heart murmurs. However, on physical examination, she now has a blowing systolic murmur that is loudest at the lower sternal border. An ECG is obtained.

What is the diagnosis on ECG?
What is the likely cause for her dyspnea, and how would you confirm the diagnosis?
Podrid’s Real-World ECGs

ECG 10 Analysis: Normal sinus rhythm, chronic (old) lateral wall myocardial infarction (MI), chronic (old) anterior wall MI
The ECG shows a regular rhythm at a rate of 82 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6, indicating a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the axis is rightward, between +90° and +180° (QRS complex is negative in lead I and positive in lead aVF). A number of conditions and situations can cause a rightward axis, including right ventricular hypertrophy (RVH) (associated with a tall R wave in lead V1 and P pulmonale), a lateral wall myocardial infarction (MI) (with Q waves [ie, a QS or Qr morphology] in leads I and aVL), Wolff-Parkinson-White pattern (with a short PR interval and widened QRS complex resulting from a delta wave), right-to-left arm lead switch (associated with a negative P wave in leads I and aVL and a positive P wave and QRS complex in lead aVR), dextrocardia (which resembles right-to-left arm lead switch and is also associated with reverse R-wave progression across the precordium [ie, a tall R wave in lead V1 that becomes progressively shorter going to lead V6]), or a left posterior fascicular block (LPFB) (a diagnosis of exclusion that is established when other causes for a rightward axis have been ruled out).

In this case, there is a negative QRS complex in lead I that is the result of a QS morphology (↓) and not an rS morphology; this is diagnostic for an old or chronic lateral wall MI and not a rightward axis due to a conduction abnormality (eg, LPFB). There is also a QS complex in lead aVL (▼) that further confirms the lateral MI. In addition, the lack of significant R waves in leads V1-V6 is suggestive of an old or chronic anterior wall MI. The QT/QTc intervals are normal (340/400 msec).

The patient has had a large lateral and anterior wall MI, likely 2 weeks ago based on the timing of her described symptoms. Patients with diabetes can often have masking of anginal chest discomfort, and hence have “silent” (without pain or discomfort) ischemia. However, they will still often experience the other symptoms associated with angina, including nausea, vomiting, shortness of breath, and diaphoresis. Therefore, this is best termed discomfortless (or painless) ischemia.

Mechanical complications of an MI most commonly occur in the 5 to 10 days after infarction when granulation tissue develops. These complications include papillary muscle rupture of the mitral valve leading to severe mitral regurgitation, free wall rupture of the ventricular wall, and ventricular septal defects (VSDs). This patient’s murmur is most consistent with a VSD. The diagnosis can be confirmed either with an echocardiogram (using Doppler color flow) or right heart catheterization and measurement of oxygen saturation within the cardiac chambers. A VSD with left-to-right shunting will have an oxygen step-up between the right atrial and right ventricular chambers. Most patients with acute VSD require surgical closure.
Notes
A patient with shortness of breath describes an episode of chest discomfort 1 week earlier that resolved after 12 hours. Since then, he has had progressive dyspnea on exertion and weight gain. Physical examination demonstrates a loud holosystolic murmur at the lower left sternal border. An ECG is obtained. Cardiac catheterization confirms single-vessel coronary disease.

What is the abnormality on the ECG? What is the most likely mechanical complication associated with what the patient has experienced?
ECG 17 Analysis: Normal sinus rhythm, chronic (old) anteroseptal myocardial infarction
The ECG shows a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (the QRS complex is positive in leads I and aVF). The QT/QTc intervals are normal (440/440 msec).

The major finding is the presence of QS complexes in leads V1-V3 (↓), which are diagnostic for a myocardial infarction of the anteroseptal wall of the left ventricle. The QS complex indicates that all of the ventricular forces are directed away from leads V1-V3, meaning that the myocardium beneath these leads is electrically silent (ie, infarcted). This is consistent with an infarction in the territory of the left anterior descending artery. The most likely diagnosis is a mechanical complication (ie, a ventricular septal defect [VSD] of the apical septum). Other mechanical complications include rupture of the posteromедial papillary muscle, which is supplied by the posterior descending artery. This is usually associated with an inferior wall infarction. Rupture of the anterolateral papillary muscle is less common as this structure typically receives dual supply from the left anterior descending and circumflex arteries. It is, therefore, less likely to rupture in the setting of single-vessel disease. Conversely, VSDs are more likely to occur in patients with one-vessel disease since they have not developed an extensive degree of collateral circulation. Other risk factors for post–myocardial infarction VSD include large infarct sizes and ventricular aneurysm formation. An infarct in the territory of the left anterior descending artery will cause a VSD in the apical septum. Inferior infarcts will result in a VSD of the basal septum.
A 42-year-old man with no cardiac risk factors is brought to the emergency department by his wife, who states that he had acute-onset substernal chest pressure. She says that over the past 2 weeks he has had a viral syndrome with upper respiratory symptoms, fever, and myalgia. On physical examination, his blood pressure is noted to be 70/palp, his neck veins are distended, and he is confused and disoriented. An arterial line is placed showing a systolic blood pressure ranging from 65 to 85 mm Hg, which varies with respiration. An ECG is obtained.

What is the most likely diagnosis?

Does the patient need to go to the cardiac catheterization lab?
Podrid's Real-World ECGs

ECG 18 Analysis: Normal sinus rhythm, ST-segment elevation due to acute pericarditis
The ECG shows a regular rhythm at a rate of 88 bpm. There is P wave before each QRS complex (+), and the PR interval (0.16 sec) is stable. The P wave is positive in leads I, II, aVF, and V4-V6.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (280/340 msec). There is widespread J-point ↑ and ST-segment elevation ▼ involving all leads except aVL; the ST-segment depression in lead aVR ▲ is actually ST-segment elevation. There are no reciprocal ST-segment depressions. The J point is elevated, up to 6 mm in lead V4, and the ST segment maintains its normal concave morphology. Also noted is the fact that the T waves retain their normal asymmetric pattern (ie, slower in upstroke and more rapid in downstroke). This ECG pattern is diagnostic for pericarditis, which also involves the epicardial pericardium and the surface of the myocardium (ie, there is an associated myocarditis). Although not always seen in pericarditis, there is evidence of PR-segment depression in leads II and aVF ▼. There is also evidence of left ventricular hypertrophy (LVH), with an R wave in lead V5 that is 28 mm in height ( ▲).

The ECG findings associated with pericarditis include:

- Diffuse ST-segment elevation. The ST segments maintain a normal concave morphology regardless of the height of ST-segment elevation or the duration of symptoms. There is no reciprocal ST-segment depression.
- The T waves are normal (ie, asymmetric).

- PR interval depression may be seen.
- T-wave inversion may occur after ST segments return to isoelectric baseline.

The changes with pericarditis should be distinguished from those seen with an acute myocardial infarction, in which the ST-segment elevation is localized; the ST-segment morphology becomes concave, merging with the T wave; T waves early on are often symmetric in configuration; and reciprocal ST-segment depressions are usually seen in other leads.

There is no indication for a coronary angiogram in the setting of acute pericarditis. However, hemodynamic instability and hypotension may occur, often due to a large pericardial effusion resulting in tamponade. Physical examination findings of tamponade include jugular venous distention, muffled heart sounds, and hypotension (Beck’s triad) as well as pulslus paradoxus, which is a drop in systolic blood pressure of more than 10 mm Hg during inspiration. This patient does have pulslus paradoxus and most likely has tamponade. This could be confirmed on echocardiography, which would show a large pericardial effusion, right atrial and right ventricular diastolic collapse, and right and left ventricular interdependence. With inspiration, the right ventricular dimensions increase and there is increased flow across the tricuspid valve while the left ventricular dimensions decrease and there is decreased flow across the mitral valve. With expiration, the findings are reversed. If a large effusion and tamponade are confirmed, the patient will need to go to the cardiac catheterization lab urgently for drainage of his pericardial effusion.
A 40-year-old man presents to the emergency department with left-sided chest and shoulder pain. He reports that he was lifting heavy furniture over the past few days and may have “overdone it.” His only medical history is hypertension, for which he takes a thiazide diuretic. Physical examination is normal except for a blood pressure of 170/90 mm Hg. An ECG is obtained.

What is the most likely diagnosis?
ECG 19 Analysis: Sinus bradycardia, left ventricular hypertrophy (LVH), early repolarization
The ECG shows regular rhythm at a rate of 54 bpm. There is a P wave (+) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Therefore, this is a sinus bradycardia.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/380 msec). There is markedly increased R-wave voltage seen in leads V3-V5 (↑) (up to 40 mm in lead V4), which is characteristic of left ventricular hypertrophy (LVH). However, body habitus needs to be considered, and this might be a normal QRS amplitude in a young subject who is thin and has no lung disease. Also noted is J-point and ST-segment elevation (↑), particularly in leads V2-V5. In association with the prominent QRS complex amplitude, this pattern is termed early repolarization. It is often seen in the precordial leads in the presence of LVH, although it may also be present in younger patients who do not have LVH. It is a normal variant, although has occasionally been diagnosed as an early acute myocardial infarction when the ST-segment changes are very marked and the patient presents with chest pain. However, the presence of asymmetric T waves and the absence of reciprocal ST-segment depressions make the diagnosis of an acute myocardial infarction unlikely.
A routine ECG is obtained in an asymptomatic 30-year-old woman.

Is the ECG characteristic of ischemia?
What is the QT interval?
ECG 20 Analysis: Sinus bradycardia, left axis, nonspecific T-wave abnormalities
The ECG shows a regular rhythm at a rate of 54 bpm. There are P waves (+) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Therefore, this is a sinus bradycardia.

The QRS axis is between 0° and –30° (positive QRS complex in leads I and II, negative QRS complex in lead aVF), which represents a physiologic left axis. Although the QRS voltage in the limb leads is low, the criterion for low voltage is not met (ie, QRS complex < 5 mm in amplitude in each lead). Biphasic T waves (↑) are noted in leads V2-V6 (positive-negative); these are nonspecific T-wave abnormalities. Although of no particular importance, such abnormalities do affect the ability to accurately establish the QT interval, which should be measured in the lead in which a normal-looking T wave is seen and a distinct end of the T wave can be identified. In this case the best lead to use would be II, III, or aVF. The QT interval (↔) is 440 msec, or 420 msec when corrected for heart rate. Of note, in asymptomatic individuals, T-wave inversions or biphasic T waves can often represent normal variants. However, one must also consider pathologic etiologies, including coronary artery disease, hypertrophic cardiomyopathy, evolving pericarditis, and electrolyte abnormalities. T-wave abnormalities should be interpreted in association with the clinical picture of the patient. In the absence of a clinical story to suggest ischemia, the T-wave changes are “nonspecific.”

\[99\]
A 52-year-old woman comes for a clinic visit complaining of occasional palpitations. An ECG is obtained. What is the most likely etiology for her symptoms?
Podrid’s Real-World ECGs

ECG Analysis: Sinus bradycardia, premature atrial complexes, nonspecific T-wave changes, clockwise rotation
The ECG shows a regularly irregular rhythm at a rate of 50 bpm. The irregularity results from several early or premature QRS complexes (fourth, sixth, ninth, and 10th). The QRS complex duration is normal (0.08 sec), and the axis as normal, between 0° and +90°. The QT/QTc intervals are normal (480/440 msec). There is low voltage in the limb leads (< 5 mm in each lead). There are P waves (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus bradycardia.

However, as indicated, the rhythm is not regular as there are occasional early or premature P waves (*) and QRS complexes associated with a shorter RR interval (complexes 4, 6, 9, and 10). Although irregular, there is a pattern to the irregularity in that all of the shorter RR intervals are the same (└┘) and all of the longer intervals (┌┐) are the same. Hence this is said to be regularly irregular. The early QRS complexes have a P wave (*) before them, and there are very subtle differences in the P-wave morphology when compared with the sinus P waves; this is seen best in leads aVF and V1-V6. Hence the early P waves and QRS complexes are called premature atrial complexes, and since the P-wave morphology is very similar to the sinus P waves they are originating very close to the sinus node.

Premature atrial premature complexes can often be symptomatic and cause the feeling of palpitations. The sensation of palpitations is not a result of the premature complex, however, but rather is due to the fact that after the premature complex there is a variable pause (longer RR interval) and a longer period of diastole during which there is continued left ventricular filling. Hence with the next sinus complex and ventricular contraction there is increased contractility and stroke volume (via the Starling effect) that accounts for the sensation of palpitations (postextrasystolic potentiation). In this patient, it would be reasonable to obtain an event monitor in order to correlate her symptoms with any rhythm disturbance, such as frequent premature atrial complexes, or with other associated arrhythmias that may be initiated by these premature complexes, such as atrial tachycardia, atrial flutter, or atrial fibrillation.

In addition, asymmetric T-wave inversions can be seen in leads V1-V4 (♦) along with flat T waves in leads aVF and V5 (▲). In the absence of any clinical history or other ECG changes, these T-wave inversions are nonspecific.

Lastly, there is poor R-wave progression from leads V1-V3 with late transition (R/S ratio becomes > 1) seen in lead V5. This is indicative of clockwise electrical rotation in the horizontal plane, determined by imagining the heart as viewed from under the diaphragm. With clockwise rotation the left ventricular forces are shifted posteriorly and are seen in the more lateral precordial leads. Poor R-wave progression may also be seen in women as a result of attenuation of forces, possibly as a result of breast tissue.
Which of the following is this patient most likely not to have?

A. Untreated hypertension  
B. Critical aortic stenosis  
C. Severe mitral regurgitation  
D. Hypertrophic cardiomyopathy
Podrid’s Real-World ECGs

ECG 22 Analysis: Normal sinus rhythm, left anterior fascicular block, counterclockwise rotation, ST-segment depression due to subendocardial ischemia
The ECG shows a regular rhythm at a rate of 68 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the axis is extremely leftward, between −30° and −90° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). Because the complexes in leads II and aVF have an rS morphology, the leftward axis is not the result of an inferior wall myocardial infarction (in which there would be a QS or Qr morphology) but rather is the result of left anterior fascicular block, a conduction abnormality. In addition, there is a tall R wave in lead V2 (<−), a result of early transition or counterclockwise rotation in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are shifted anteriorly and are apparent earlier in the precordial leads (ie, the tall R wave in lead V2 (<−)). However, the amplitude of the QRS complex in lead V2 is increased (R wave = 30 mm), which likely represents left ventricular hypertrophy (LVH). The QT/QTc intervals (↔) are prolonged (520/550 msec).

The major finding, however, is the presence of diffuse, symmetric, deeply inverted T waves in leads II, III, aVF, and V3-V6 (^). The positive T wave in lead aVR (•) is actually T-wave inversion. In addition, there is J-point depression (▼) and downsloping ST-segment depression (↑). In this situation the symmetric T-wave inversions as well as the prolongation of the QT interval are reflective of diffuse subendocardial ischemia, which is likely the result of LVH. The presence of subendocardial ischemia may be the cause of the QT prolongation. The most common causes of LVH include uncontrolled hypertension, aortic stenosis, and hypertrophic cardiomyopathy. Mitral regurgitation results in a dilated left ventricle, which can often result in similar ST-segment and T-wave abnormalities but normally without the prominent voltages of LVH. n
A 46-year-old man with polycystic kidney disease presents with new-onset left arm weakness and headache. The following ECG is obtained.

What is the most likely etiology of the abnormal findings?
Podrid’s Real-World ECGs

ECG 23 Analysis: Normal sinus rhythm, left axis, cerebral T waves, QT prolongation
The ECG shows a regular rhythm at a rate of 62 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The axis is leftward and the QRS complex is positive in lead I, negative in lead aVF, and isoelectric in lead II, indicating that the axis is –30°. The major finding is prominent deep T-wave inversions in leads V1-V3 (†). The T waves are asymmetric, with a more rapid upstroke and slower downstroke (the reverse of the normal T-wave morphology). Also present is significant prolongation of the QT/QTc intervals (↔) (620/630 msec). This is a pattern often seen with an intracerebral process, such as a subarachnoid or intracerebral hemorrhage, and the T waves are termed cerebral T waves. The QT interval prolongation is the result of autonomic imbalance. About 5% to 10% of patients with polycystic kidney disease have cerebral aneurysms.
A 57-year-old woman with a history of intermittent lightheadedness presents to the emergency department after having a syncopal episode. She denies any prodromal symptoms. An ECG is obtained while the patient is feeling dizzy.

What is the most likely diagnosis?
How should you treat this patient?
Podrid’s Real-World ECGs

ECG 24 Analysis: Normal sinus rhythm, intraventricular conduction delay (IVCD), left anterior fascicular block, sinus node exit block
The ECG shows a regularly irregular rhythm at a rate of 84 bpm. There is a P wave (+) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex is widened (0.14 sec), but there is no specific pattern (there is a tall, broad R wave in lead I [→], suggesting left bundle branch block, but also terminal S waves in leads V5-V6 [←], suggesting right bundle branch block). Hence this is considered an intraventricular conduction delay (IVCD).

There is an important difference between a bundle branch block and an IVCD. With a bundle branch block, activation of the ventricle served by the affected bundle is no longer via the normal conduction system but rather by impulse conduction through an abnormal pathway and direct myocardial activation. Therefore, abnormalities affecting that ventricle cannot be interpreted. In contrast, an IVCD is diffuse slowing of conduction via the normal His-Purkinje system. As ventricular activation is still via the normal conduction pathway, abnormalities of the involved ventricle can be established. In this case, the axis is very leftward (between −30° and −90°), with a positive QRS complex in lead I and negative QRS complexes in leads II and aVF (rS morphology). This is a left anterior fascicular block, which can be diagnosed with an IVCD but could not be diagnosed if there was a true left bundle branch block. The QT/QTc intervals are prolonged (400/470 msec) but are normal after accounting for the prolonged QRS interval (340/400 msec).

The rhythm is regularly irregular as there are two long (and similar) RR intervals between the 10th, 11th, and 12th QRS complexes (┌┐). The long RR intervals are the result of an abrupt slowing of the heart rate to 42 bpm. There is no evidence for a nonconducted P wave during the pause; this is termed a sinus pause. This type of sinus node abnormality can be a manifestation of either abnormal impulse formation (ie, sinus node arrest) or abnormal impulse propagation (ie, sinus node exit block). In this case, the PP interval around the pause is equal to two sinus intervals (└┘) and hence is termed sinus node exit block. The sinus node has discharged on time, but the impulse fails to propagate out of the sinus node area and does not activate the atrium, hence the absence of a P wave. Since the sinus node rate is unaffected, the next sinus impulse is on time and does activate the atrium, maintaining the regularity of the PP intervals.

continues
Another cause for a sinus node pause is termed a sinus node arrest. In this situation the sinus node fails to generate an impulse. Therefore, there is no relationship between the PP interval around the pause and the underlying sinus interval. The pause may be shorter than or longer than two sinus intervals. A pause that is longer than two sinus intervals may be indicative of underlying sinus node dysfunction, termed sick sinus syndrome.

A sinus node pause generally is transient, so there is usually no reason for any therapy. However, if there are continuous sinus node pauses, resulting in symptoms, therapy would involve atropine, isoproterenol, or a temporary pacemaker. In the absence of a reversible etiology, such as a medication effect, symptomatic sinus node disease that is persistently manifested (ie, a continuous symptomatic sinus bradycardia) is a class I indication for a permanent pacemaker. If this were an incidental finding in an asymptomatic patient, then no pacemaker would be indicated. However, sinus node dysfunction in an asymptomatic individual with a heart rate less than 40 bpm while awake is a class IIa indication for placement of a permanent pacemaker.
A 74-year-old man with known coronary artery disease is admitted to the hospital with atrial fibrillation and a rapid ventricular response. In addition to his known regimen of aspirin, β-blocker, and statin, he is prescribed verapamil for rate control. The next day, he begins to complain of intermittent dizziness. An ECG is obtained while the patient is symptomatic.

What is the most likely cause of the patient’s dizziness?
Podrid’s Real-World ECGs

**ECG Analysis:** Normal sinus rhythm, intraventricular conduction delay (IVCD), left anterior fascicular block (LAFB), sinus node arrest
The ECG shows an irregular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, VF, and V4-V6. Hence this is a sinus rhythm.

The axis is extremely leftward (between –30° and –90°), and the QRS complex is positive in lead I but negative in leads II and aVF. The QRS complex is wide (0.16 sec), with a tall, broad R wave in leads I and V5-V6 (←), a wide QS complex in lead V1 (→), and no septal forces. This is characteristic of a left bundle branch block (LBBB). However, there are terminal S waves (left-to-right forces) in leads V5-V6 (↑), which suggests a right bundle branch block. Left-to-right forces are not seen in an LBBB. Hence this is an intraventricular conduction delay (IVCD). In this situation there is only diffuse slowing of conduction through the normal His-Purkinje system and no block in the left bundle. With an IVCD conduction is through the normal conducting system; it is just slower. Therefore, abnormalities of the left ventricle can be diagnosed. With an LBBB, left ventricular activation is not via the normal conduction system but directly through the myocardium, and hence left ventricular abnormalities cannot reliably be diagnosed. As an LBBB is not present, the extreme left axis is the result of a left anterior fascicular block (LAFB); if there were an LBBB, the diagnosis of LAFB would not be appropriate as both fascicles would be affected. The QT/QTc intervals are normal (360/410 sec).

The irregularity of the rhythm is due to two pauses (⊔⊔) in the rhythm with absence of atrial activity or a P wave. These are termed sinus pauses. The PP interval of the pause is unrelated to the underlying sinus interval (⊤⊤) (ie, it is shorter than two PP intervals). The PP intervals are otherwise fixed. This is termed sinus node arrest as the sinus node has failed to discharge but resumes its activity after a variable time. This may be an early manifestation of underlying sinus node dysfunction (ie, sick sinus syndrome).

The most likely cause of the patient’s rhythm disturbance is polypharmacy. The combination of β-blocker and calcium-channel blocker can lead to significant bradycardia involving either the sinus or AV node. At this point, nodal agents should be withheld. However, if these agents are needed to control tachyarrhythmia, then this is a class I indication for a permanent pacemaker if the sinus node pauses are continuous and associated with symptoms or if the pauses become longer, indicating an underlying sick sinus syndrome.

It should be noted that ischemia is not the cause of sinus node abnormalities. The sinus and AV nodes generate an action potential that is based on calcium ion fluxes, which are energy independent and do not require an energy-dependent ATPase pump. Hence sinus and AV nodal activity is unaffected by ischemia.
A 62-year-old man with no cardiac history presents for a routine physical examination. An ECG is obtained as part of his evaluation. Has this patient had an anterior myocardial infarction?
Podrid’s Real-World ECGs

**ECG 26 Analysis:** Sinus bradycardia, intraventricular conduction delay (IVCD), left anterior fascicular block (LAFB)
The ECG shows a regular rhythm at a rate of 56 bpm. There is a P wave (+) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus bradycardia.

The QRS complex duration is prolonged (0.12 sec). There is no pattern typical for a left or right bundle branch block; hence this is an intraventricular conduction delay (IVCD). The QT/QTc intervals are normal (400/350 msec). The axis is extremely leftward, between –30° and –90° (QRS complex is positive in lead I and negative in leads II and aVF with an rS morphology). This is called a left anterior fascicular block (LAFB), or hemiblock. The left bundle, which activates the left ventricle, divides into a minor fascicle (a median or septal branch that innervates the intraventricular septum) and two major fascicles, the left anterior and left posterior fascicle. The left anterior fascicle innervates the base of the left ventricle, while the left posterior fascicle travels along the inferior portion of the left ventricle. In the presence of an LAFB, activation of the left ventricle is via the left posterior fascicle and the direction of activation is upward and toward the left, producing the extreme leftward axis. There is no major delay in left ventricular activation and hence a fascicular block does not cause any IVCD (ie, the QRS duration is normal). When a wide QRS complex is present there is also an IVCD.

It is important to distinguish between an extreme left axis due to LAFB, in which the QRS complex has an rS morphology in leads II and aVF, and an inferior wall myocardial infarction, in which the QRS complex has a Qr morphology.

Small Q waves are noted in leads V2-V3 (†). Although it has been suggested that any anterior Q waves represent a myocardial infarction, such small anterior Q waves may instead represent block of the septal branch of the left bundle. This may portend the development of a complete LBBB, which may be more likely in a patient who also has an LAFB.

Fascicular blocks are often due to idiopathic conduction system disease (called Lev’s or Lenègre’s disease), which results in fibrosis or a calcificofibrosis of the bundles. They may be due to ischemic heart disease with prior infarction and fibrosis of the conduction system, idiopathic cardiomyopathy with diffuse fibrosis of the myocardium, hypertension, or drugs that may alter conduction through the His-Purkinje system. Fascicular blocks may be permanent, intermittent, or rate related.
Notes
A 64-year-old asymptomatic patient is seen for a routine physical examination. What is the most likely etiology for this patient’s abnormal QRS axis?
Podrid’s Real-World ECGs

ECG 27 Analysis: Normal sinus rhythm, right axis due to a left posterior fascicular block (LPFB)
The ECG shows a regular rhythm at a rate of 82 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), as are the QT/QTc intervals (380/440 msec). The axis is rightward, between +90° and +180° (the QRS complex is negative in lead I and positive in lead aVF). The QRS complex in lead I has an rS morphology.

A rightward axis is always abnormal and may be due to:

- Right ventricular hypertrophy (RVH), in which case there is a tall R wave in lead V1 and often evidence of right atrial hypertrophy with P pulmonale, which is indicated by a tall, narrow, and peaked P wave.
- A lateral wall myocardial infarction, in which case the initial waveform of the QRS complex in leads I and aVL is a Q wave (a Qr complex).
- Right-to-left arm lead switch, in which case negative P waves are also seen in leads I and aVL.
- Dextrocardia, as evidenced by negative P waves in leads I and aVL as well as reverse R-wave progression across the precordium (ie, there is a tall R wave in lead V1 that becomes progressively shorter from leads V1 to V6).

- Wolff-Parkinson-White pattern, in which the PR interval is short and the QRS complex is wide as a result of a delta wave. The rightward axis results from an initial Q wave in leads I and aVL (QS complex) and not an rS complex. This is a pseudo lateral infarction pattern.
- Left posterior fascicular block (LPFB), in which the initial QRS waveform in lead I is an R wave (an rS complex). The diagnosis of an LPFB is established when the other causes of a rightward axis have been excluded.

The rightward axis in this ECG is a result of an LPFB (with an rS complex in leads I and aVL) as there are no features to suggest any other causes for a rightward axis (ie, evidence of RVH, dextrocardia, right-to-left arm lead switch, lateral wall myocardial infarction, or Wolff-Parkinson-White pattern). Although less common than a left anterior fascicular block (LAFB), the causes for an LPFB are the same as for an LAFB, including idiopathic conduction system disease (Lev’s or Lenègre’s disease), hypertension, ischemic heart disease, or a cardiomyopathy.
A healthy 26-year-old presents with an ankle fracture that resulted from a sports injury. A routine ECG is obtained, and an abnormality is noted. Would further cardiac workup be necessary?
Podrid’s Real-World ECGs

ECG28 Analysis: Normal sinus rhythm, right bundle branch block (RBBB)
The ECG shows a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is increased (0.14 sec). There is an RSR’ complex in lead V1, and the R’ (←) is broad and tall. A broad terminal S wave (↑) is seen in lead I; although not well seen on this ECG, there are usually broad S waves in leads V5-V6 (↑). This is a typical pattern of right bundle branch block (RBBB) in which there is delayed activation of the right ventricle. The ST-T wave abnormalities noted in leads V1-V3 (↑) are associated with an RBBB. The QRS complex has a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal when the prolonged QRS duration is considered (420/430 and 360/370 msec).

The diagnosis of an RBBB is based on the following:

- The QRS duration is ≥ 0.12 second due to delayed activation of the right ventricle.
- Right ventricular activation is from left bundle and left ventricle, directly through the myocardium. Therefore, the terminal forces of the QRS complex are directed from left to right.

- As a result of the terminal forces going from left to right, the QRS complex has a secondary R wave in lead V1 (ie, an RSR’ morphology) with a broad terminal R wave (R’) in leads V1-V2 and a broad terminal S wave in leads I and V5-V6. The broad terminal waveforms are due to prolonged time for right ventricular activation as the impulse is conducted directly through the ventricular myocardium and not the normal Purkinje system.

- Right ventricular repolarization is abnormal, and secondary ST-T wave changes can be seen in leads V1-V3.
- As right ventricular activation is abnormal, right ventricular hypertrophy cannot be reliably diagnosed.
- Left ventricular activation is normal. Therefore, the initial portion of the QRS complex is normal and abnormalities of the left ventricle (eg, left ventricular hypertrophy, infarction, ischemia, pericarditis) can be recognized.

RBBB can be seen in healthy individuals as well as in several pathologic conditions, including idiopathic conduction system disease (Lev’s or Lenègre’s), ischemic heart disease with previous myocardial infarction, myocarditis, hypertension, cardiomyopathy, acute elevation of right ventricular pressure (as with a pulmonary embolism), and chronically elevated right ventricular pressure (such as cor pulmonale) or a left-to-right shunt (such as with an atrial septal defect).
A 75-year-old patient is seen in the emergency department for an acute abdomen that is believed to be due to acute cholecystitis. Just before going to the operating room, an ECG is obtained and an abnormality is noted.

Is any further workup or therapy indicated prior to surgery?
Podrid’s Real-World ECGs

ECG 29 Analysis: Normal sinus rhythm, right bundle branch block (RBBB), left anterior fascicular block (LAFB), bifascicular block
The ECG shows a regular rhythm at a rate of 74 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is increased (0.16 sec), and the morphology shows a typical right bundle branch block (RBBB) pattern, with an RSR' in leads V1-V2 (←) and broad terminal S waves (↑) in leads I, aVL, and V5-V6. The T-wave abnormalities seen in leads V1-V3 (↑) are associated with an RBBB. In addition, the axis is very leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complexes in leads II and aVF with an rS morphology). The extreme left axis is not the result of an inferior wall MI, in which the QRS complexes would have a QS or Qr morphology. Here the QRS complex has an rS morphology, indicating that the extreme leftward axis is the result of a left anterior fascicular block (LAFB). Therefore, there is conduction block in two of the three major fascicles (right bundle and left anterior fascicle); this is termed bifascicular block. The QT/QTc intervals are normal when accounting for the prolonged QRS complex duration (400/440 msec).

The presence of bifascicular block has been a concern in patients who are to undergo surgery because of the potential development of complete heart block as a result of further conduction problems. However, there are no data to indicate that this will definitely occur, and the presence of bifascicular block is not an indication for temporary pacing prior to surgery or permanent pacing after surgery. Pacing would be indicated for a symptomatic patient who had evidence of disease also affecting the left posterior fascicle (ie, trifascicular disease), such as alternating left bundle branch block and RBBB, RBBB with alternating LAFB and left posterior fascicular block, evidence of Mobitz type II block, or intermittent complete heart block with a ventricular escape rhythm.
The following ECG was obtained from a patient who sustained a myocardial infarction (MI). The patient’s baseline ECG 6 months earlier was completely normal.

What is the most likely territory of this patient’s MI?
What is the patient’s overall prognosis given the presence of new conduction system disease?
Podrid’s Real-World ECGs

**ECG Analysis:** Normal sinus rhythm, right bundle branch block (RBBB), right axis due to left posterior fascicular block (LPFB), bifascicular block, old anteroseptal myocardial infarction (MI)
The ECG shows a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is prolonged (0.16 sec) and has a right bundle branch block (RBBB) morphology, with a broad R wave or qR complex in lead V1 (←) and a broad S wave (→) in leads I, aVL, and V5-V6. The axis is rightward, between +90° and +180° (positive QRS complex in lead aVF and negative QRS complex in lead I). The broad terminal S wave in lead I is a result of the RBBB, so it is important that this waveform not be included in determination of the axis because this represents right ventricular depolarization. For axis determination, only the first 0.08 second of the QRS complex is considered. However, even ignoring the terminal S wave, the QRS complex in lead I is still negative and hence the axis is indeed rightward. In the absence of any other ECG features accounting for the rightward axis (ie, right ventricular hypertrophy, right-to-left arm lead switch, lateral infarction, dextrocardia, Wolff-Parkinson-White pattern), it is the result of a left posterior fascicular block (LPFB). The presence of an RBBB and LPFB is termed bifascicular block, similar to what was discussed for Case 29. There are also Q waves in leads V1-V2 (▼), consistent with an old anteroseptal MI.

Isolated LPFB (or hemiblock) is uncommon and is the least common of the fascicular conduction diseases because of its anatomic location; the left posterior fascicle often spreads out within the posterior and infero-posterior walls and is less likely to be affected by disease processes. In addition, it has dual blood supply from the septal branches of the left anterior descending artery and the AV nodal branch from the posterior descending artery. It may have an idiopathic etiology (ie, Lev’s or Lenègre’s disease) or may occur in association with a cardiomyopathy, hypertension, myocarditis, or extensive coronary artery disease and myocardial infarction (MI). When isolated LPFB does occur in the setting of coronary artery disease and previous MI, it generally indicates more extensive disease. The presence of LPFB and RBBB is associated with a 21% to 75% increased risk for complete heart block compared with RBBB and left anterior fascicular block. Mortality is increased when an LPFB occurs in the setting of acute MI, primarily as a result of the presence of more extensive coronary disease.
A 24-year-old man presents for a routine physical exam before entering graduate school. He has no known heart disease and no cardiac symptoms.

Is the QRS complex abnormal in this ECG?
ECG 31 Analysis: Sinus bradycardia, left axis, intraventricular conduction delay (IVCD) to the right ventricle (incomplete RBBB)
The ECG shows a regular rhythm at a rate of 54 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6.

The QRS complex duration is normal (0.10 sec), and there is a physiologic left axis, between 0° and –30° (negative QRS complex in lead aVF and positive QRS complex in leads I and II). The QT/QTc intervals are normal (400/380 msec). Although the QRS duration is normal, there is an RSR' complex in leads V1-V2 (→). As a result, the QRS complex morphology resembles a right bundle branch block (RBBB). However, the QRS complex duration is not prolonged, there is no broad S wave in lead I (↑), and the S wave in leads V5-V6 (↑) has a normal duration. When the QRS width is greater than 0.10 but less than 0.12 second, the pattern is often referred to as an incomplete RBBB, although it is best termed an intraventricular conduction delay (IVCD) to the right ventricle. As conduction through the bundles (ie, right bundle) is all or none, incomplete conduction is actually an IVCD. When the QRS width is normal (ie, ≤ 0.10 sec), the RSR’ pattern is a normal variant, representing a minor right ventricular conduction delay; this is referred to as a crista pattern, indicating a delay in the activation of the crista supraventricularis of the right ventricle. n
The following ECG is obtained from a 66-year-old patient who came to your clinic for a routine physical. The patient is doing well and complains of no symptoms. However, blood pressure is found to be elevated at 170/100 mm Hg.

What further evaluation needs to be performed?
Podrid’s Real-World ECGs

ECG 32 Analysis: Sinus tachycardia, left bundle branch block (LBBB)
The ECG shows a regular rhythm at a rate of 100 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus tachycardia.

The QRS complex duration is prolonged at 0.16 second. The QT/QTc intervals are prolonged (400/520 msec) but normal when accounting for the prolonged QRS complex duration (320/410 msec). There is a broad R wave in leads I and V5-V6 (←), with a wide and deep QS complex in lead V1 (→). This is a typical pattern of left bundle branch block (LBBB). The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, since LBBB involves both the left posterior and left anterior fascicles, it would not be appropriate for this extreme left axis to be called a left anterior fascicular block. There are ST-T wave changes in leads I, aVL, and V5-V6 (↑) that are secondary to the left bundle branch block.

Characteristics of an LBBB include the following:

- The QRS duration is 0.12 second or longer due to delayed activation of the left ventricle.
- Left ventricular activation is from the right bundle and right ventricle and is directly through the myocardium. Therefore, all ventricular forces are directed from right to left and there is a slow velocity of activation as the impulse is via the ventricular muscle and not the Purkinje system. This results in a broad, tall R wave in leads I, aVL, and V5-V6 and a wide and deep QS complex in leads V1-V2. Not uncommonly, a QS pattern may be seen across the entire precordium (ie, from leads V1-V6).
  - Since the septum is innervated by a small septal branch from the left bundle, there are no septal forces seen and hence no Q waves in lead I, aVL, or V5-V6 and no R wave in lead V1.
  - All forces are directed from right to left. No forces are directed from left to right, and hence terminal S waves in leads I and V6 are not present.
  - Since depolarization is abnormal, there is also abnormal repolarization (ie, diffuse ST-T wave abnormalities).
  - The axis may be normal or leftward. Since both the left posterior and left anterior fascicles are involved, axis shift is not related to fascicular block but to the abnormal activation sequence of the left ventricle that occurs directly through the left ventricular myocardium and not the Purkinje system. A rightward axis is not seen.
  - Since left ventricular activation is abnormal and does not occur via the normal His-Purkinje system, left ventricular abnormalities (eg, left ventricular hypertrophy [LVH], infarction, ischemia, pericarditis, myocarditis) cannot be recognized.

continues
LBBB is an uncommon finding in young patients, but when present it is not associated with heart disease. It is more common in older patients, as it is a frequent finding associated with heart disease. However, an LBBB does not suggest the presence of active ischemia but is seen with ischemic heart disease when there has been a previous infarction, primarily of the septum. However, ischemia (if present) cannot be reliably detected on the surface ECG in the setting of an LBBB as there are ST-T wave changes that are due to the LBBB. As indicated, an old infarction cannot be reliably diagnosed in the setting of an LBBB. An LBBB is often the result of idiopathic conduction system disease (Lev’s or Lenègre’s) and may also occur in the setting of LVH, left ventricular scar or fibrosis, cardiomyopathy, infiltrative processes of the myocardium, aortic valve endocarditis, rheumatic fever, and after cardiac surgery. Even though the patient is asymptomatic, obtaining an echocardiogram is indicated in light of the elevated blood pressure to assess for any structural heart disease, particularly LVH.
A 47-year-old man with a known history of an idiopathic dilated cardiomyopathy, severe mitral regurgitation, and low ejection fraction now presents with congestive heart failure.

How would the conduction abnormalities be classified in this ECG?
Podrid’s Real-World ECGs

ECG 33 Analysis: Normal sinus rhythm, first-degree AV block, left anterior fascicular block, intraventricular conduction delay (IVCD)
The ECG shows a regular rhythm at a rate of 68 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.24 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with a first-degree AV block. The P waves are broad, there is a prominent negative component in leads V1 and V2 (↑), and some notching is noted in leads V3-V4 (▼), features that are consistent with left atrial hypertrophy.

The QRS complex is widened (0.16 sec), and the axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). Although there is a broad R wave in lead I (→) and a deep S wave in lead V1 (←), a pattern that resembles left bundle branch block (LBBB), there are septal Q waves (↑) seen in leads I and aVL as well as a prominent septal R wave (↓) in lead V1. As the septal branch activating the septum arises from the left bundle, septal forces cannot be present with an LBBB. In addition, there is a terminal S wave in lead V6 (▲), indicating terminal forces that are directed in a left-to-right direction. In an LBBB all forces are directed right to left and there should not be any left-to-right forces, which are indicative of a conduction delay to the right ventricle. Therefore, this is not an LBBB but rather an intraventricular conduction delay (IVCD).

This QRS pattern, with a very wide QRS complex duration resembling an LBBB, is commonly seen in patients with severe dilated cardiomyopathy. There is a correlation between QRS width and left ventricular ejection fraction: The wider the QRS complex, the lower the ejection fraction is. The QT/QTc intervals are prolonged (480/510 msec) but are normal after correcting for the prolonged QRS complex duration (400/430 msec).

With an LBBB, left ventricular activation is abnormal as impulse conduction does not travel through the normal His-Purkinje system but rather travels directly through the ventricular myocardium. Therefore, abnormalities of the left ventricle cannot be reliably diagnosed. Since the widened QRS complex is not the result of an LBBB, left ventricular activation is via the normal His-Purkinje system, but it is prolonged or slowed, usually as a result of diffuse and severe fibrosis affecting the terminal Purkinje system. Hence abnormalities of the left ventricular myocardium, such as infarction, ischemia, inflammation (as with pericarditis or myocarditis), and hypertrophy can be diagnosed. In addition, axis shifts resulting from block of the left anterior or posterior fascicle can be identified. On this ECG the axis is very leftward (more negative than –30°); hence there is a left anterior fascicular block present.
An 80-year-old woman presents with an acute fracture of her left hip. She denies any previous cardiac history. Prior to surgery, an ECG is obtained and a cardiology consult requested for further advice.

Has this patient suffered from a prior myocardial infarction (MI)?
Podrid’s Real-World ECGs

ECG 34 Analysis: Normal sinus rhythm, intraventricular conduction delay (IVCD), left anterior fascicular block, old anteroseptal MI, nonspecific ST-T wave changes
The ECG shows a regular rhythm at a rate of 74 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm.

The QRS complex duration is slightly less than 0.12 second, and the axis is extremely leftward, between $-30^\circ$ and $-90^\circ$ (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). The QT/QTc intervals are slightly prolonged (440/490 msec), even when adjusted for the prolonged QRS complex duration (410/450 msec). Although the QRS complex has a pattern that resembles a left bundle branch block (LBBB) (monophasic R wave in leads I and V5-V6 [-], deep S wave in lead V1 [->]), the QRS duration is not quite 0.12 second and hence is slightly less than that which defines an LBBB (ie, $\geq 0.12$ sec). In addition, there is a small septal Q wave (▲) in lead aVL. This pattern has often been termed an incomplete LBBB. However, impulse conduction through the bundles is “all or none”; therefore, incomplete conduction is actually an intraventricular conduction delay (IVCD). This distinction is important because left ventricular abnormalities, such as infarction, ischemia, and hypertrophy, can be identified on the ECG in the setting of IVCD but not LBBB. An IVCD is due to diffuse slowing of conduction through the left ventricle as a result of diffuse involvement of the terminal Purkinje fibers. Therefore, impulse conduction is still along the normal His-Purkinje system, although it is slower. In an LBBB, left ventricular activation is not via the normal His-Purkinje system but rather is directly through the left ventricular myocardium (ie, an alternative pathway).

Since this is not an LBBB, the extreme leftward axis indicates that a left anterior fascicular block is present. In addition, the initial QRS force in leads V1-V3 is a Q wave (▼), which is diagnostic for an anteroseptal myocardial infarction (MI). Also present are nonspecific ST-T wave changes (↑) in leads V4-V6. As the patient is without any symptoms, no further cardiac evaluation is necessary prior to orthopedic surgery. However, an echocardiogram would be useful to confirm the old anteroseptal MI as well as to evaluate left ventricular function.
A 56-year-old man with bicuspid aortic valve undergoes surgical valve replacement for aortic stenosis. His postoperative course is unremarkable, and his ECG is shown below.

What is the principal abnormality?
How would you manage this?
Podrid’s Real-World ECGs

ECG 35 Analysis: Sinus bradycardia, first-degree AV block, intraventricular conduction delay, left axis, clockwise rotation
The ECG shows a regular rhythm at a rate of 56 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.58 sec) (↔). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus bradycardia with first-degree AV block, defined as a PR interval longer than 0.20 second.

The QRS complex duration is 0.12 second. However, there is no specific pattern suggesting either a right or left bundle branch block. While there is a QS complex in lead V1 (←) and a broad R wave in lead I (→), there is also a broad S wave in leads V5-V6 (↑) and a septal Q wave (↑) in lead aVL. As the septal branch innervating the septum comes from the left bundle, septal forces are not present with a left bundle branch block. In addition, all forces are directed from right to left and there are no left-to-right forces; terminal S waves in leads V5-V6 are not present. Hence this is a nonspecific intraventricular conduction delay. The QT/QTc intervals are normal (400/390 msec and 360/350 msec when corrected for prolonged QRS complex duration). The axis is leftward, between 0° and –30° (the QRS complex is positive in leads I and II and negative in lead aVF). There is poor R-wave progression in leads V1-V3 and late transition (ie, R/S > 1 in lead V6), which are the result of clockwise rotation, as determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are more posteriorly directed and seen in the more lateral (left-sided) precordial leads.

The major finding is a very long PR interval (primarily PR segment) that is 0.58 second and is termed a first-degree AV block. It may result from a slowing of conduction through the AV node or the His-Purkinje system. As there is no actual block of impulse conduction, a more appropriate term is prolonged AV conduction.

The PR interval includes the P wave (intraatrial conduction time) and the PR interval (conduction through the AV node and His-Purkinje system). Therefore, it represents the time for AV conduction between the atrium and ventricle. It is measured from the beginning of the P wave to the beginning of the QRS complex (Q or R wave). The normal PR interval is between 0.14 and 0.20 second. The PR interval changes with heart rate; that is, it lengthens with sinus bradycardia as a result of slower conduction through the AV node due to enhanced vagal tone or decreased sympathetic inputs, and it shortens with sinus tachycardia as a result of enhanced AV nodal conduction due to increased sympathetic tone. The PR interval, regardless of its length, is constant for each QRS complex.

A first-degree AV block is a common complication after aortic valve surgery as the aortic valve is close to the AV annulus and the AV node. No pacemaker is needed unless there is evidence of high-grade AV block, primarily complete heart block. Further PR interval prolongation or development of higher-grade AV block during follow-up may be a sign of aortic valve endocarditis in the appropriate clinical setting.
A 22-year-old college student comes to the emergency department with episodic lightheadedness and dizziness. A few days after returning from a hiking trip in Connecticut, she developed an erythematous, circular rash on her left leg with central clearing. Over the past several weeks the rash spread to other areas of her legs. She has had fatigue and myalgia but denies any fevers. An ECG is obtained while the patient is asymptomatic.

What is the major abnormality?
What is the overall clinical diagnosis?
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, second-degree AV block (Mobitz type I or Wenckebach), left anterior fascicular block, nonspecific T-wave abnormalities
The ECG shows a rhythm that is irregular, but there is a pattern of group beating with all the short RR intervals being the same and three longer RR intervals that are all the same. Hence the rhythm is regularly irregular. There is an underlying sinus rhythm of 70 bpm, established by finding sequential P waves (+). The PP intervals are regular throughout ( ). The P waves are upright in leads I, II, aVF, and V4-6; hence they are originating from the sinus node. The PR interval is not constant. The baseline PR interval is established as the PR interval that is present after the pause in the RR interval. Hence the baseline PR interval ( ) is 0.20 second. The second PR interval is lengthened to 0.28 second, and the third is 0.32 second. The fourth P wave ( ^ ) is nonconducted ( ie, it is not followed by a QRS complex). The pattern of PR interval lengthening then repeats itself. In addition, there is a slight shortening of the RR interval, from 0.92 to 0.84 second. This represents a second-degree AV block, Mobitz type I or Wenckebach pattern. There is a pattern of 4:3 Wenckebach.

A second-degree AV block as a result of Wenckebach is identified by a progressive prolongation of the PR interval prior to a single non-conducted P wave. After the nonconducted P wave (^), conduction resumes with a resetting of the PR interval back to its baseline. Thus there is always one more P wave than QRS complexes and Wenckebach is termed 3:2, 4:3, 5:4, etc. The pattern of block may be stable ( ie, always 3:2, 4:3, etc) or may have variable pattern ( ie, alternating periods of 4:3 with 3:2, etc). Often the increment of PR lengthening may progressively decrease with each complex, with the greatest degree of lengthening in the first interval and then a progressive decrease in the increment of lengthening, as is seen in this case (the PR interval increased from 0.20 to 0.28 and then to 0.32 sec). As a result there is a shortening of the RR interval, as can be seen between the first and second RR intervals ( ie, 0.92 and 0.84 sec). However, shortening of the RR interval may not be observed and is not necessary to establish Wenckebach.

A second-degree AV block is identified by a pause in the RR interval due to occasional nonconducted P waves. The sinus rate or PP intervals are constant. First-degree AV block may also be present.
Podrid's Real-World ECGs

Mobitz type I second-degree AV block results from a conduction abnormality involving the AV node. As the action potential generated by the AV node is mediated by calcium currents, the conduction velocity through this structure may not be constant (i.e., it is not all or none) but can change depending on the presence of AV nodal disease, changes in autonomic tone, or drugs that affect the AV node such as digoxin, calcium-channel blockers (specifically verapamil or diltiazem), or β-blockers. Therefore, the AV node manifests decremental conduction (i.e., conduction through it slows as the heart rate increases), which likely accounts for the Wenckebach pattern of progressive slowing of impulse conduction through the AV node.

Mobitz type I AV block is generally not serious. If the patient is asymptomatic, no further therapy is warranted. However, if there are symptoms from a bradycardia and the patient is not receiving AV nodal blocking agents, then a pacemaker would be indicated for symptomatic bradycardia. Should complete heart block develop, the escape rhythm will originate in the AV node (i.e., a nodal or junctional rhythm), which is typically stable. Unless the escape rhythm is slow and/or associated with symptoms, there is no indication for the acute insertion of a temporary pacemaker.

Additional findings on this ECG include a normal QRS complex duration (0.08 sec) and the presence of an extreme leftward axis, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). An extreme leftward axis may be seen with an inferior wall myocardial infarction, which is associated with a deep initial Q wave in leads II and aVF. When the QRS complexes in leads II and aVF do not show evidence of an inferior wall myocardial infarction, but rather have an rS morphology, the cause of the left axis is a left anterior fascicular block. There are also diffuse T-wave inversions (†), which are nonspecific. The QT/QTc intervals are slightly prolonged (420/450 msec).

The patient presents with fatigue, myalgias, and AV block after a camping trip in Connecticut. This, along with the rash she has developed, which is a typical pattern for erythema migrans, supports a diagnosis of Lyme disease. The fact that she has AV nodal Wenckebach that is asymptomatic is concerning for the occurrence of intermittent episodes of higher-grade AV block as an etiology of her dizziness. Observation with continuous monitoring would be useful to establish the presence of more advanced conduction problems, which, if present, are an indication for temporary pacing. The AV nodal conduction abnormalities associated with Lyme disease are transient and reversible, although the time course for recovery is variable.
A 92-year-old woman with chronic hypertension treated with atenolol presents with significant pre-syncopal symptoms of dizziness over the past day. She reports significant watery diarrhea over the past 3 days with nausea, resulting in poor oral intake. She denies fevers, sweats, or blood in her stools. Her physical exam reveals a blood pressure of 90/45 mm Hg, jugular venous pressure of 5 cm, dry mucous membranes, a regularly irregular heartbeat, and a soft abdomen with prominent bowel sounds. An ECG is obtained.

What is the primary ECG abnormality?
What is the most likely cause of her dizziness?
ECG 37 Analysis: Sinus bradycardia, second-degree AV block (Mobitz type II), left ventricular hypertrophy, nonspecific ST-T wave changes
The ECG shows P waves (+) occurring at regular intervals (┘) and a heart rate of 48 bpm. The P waves are upright in leads I, II, aVF, and V4-V6. Hence this is a sinus bradycardia. However, the ventricular rate is not regular as a result of an occasional long RR interval (┌┐) that occurs because of a single nonconducted P wave (▼). Hence the rhythm is regularly irregular. There is a stable PR interval at 0.20 second (↔). Every fourth P wave (▼) lacks a conducted QRS. Hence this represents a second-degree AV block (ie, a pause in the RR interval due to an occasional nonconducted P wave). The fact that the PR intervals of each of the conducted complexes is constant defines this as Mobitz type II AV block. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (460/410 msec).

Mobitz type II second-degree AV block results from conduction failure within the His-Purkinje system. The His-Purkinje system generates a fast action potential that is mediated by sodium currents. It is all or none (ie, it either conducts the impulse or does not conduct the impulse). It does not manifest decremental conduction. With a Mobitz II block, therefore, the His-Purkinje system intermittently fails to conduct an impulse, but when an impulse is conducted the rate of conduction is stable, accounting for the constant PR interval.

Mobitz type II second-degree AV block is a more serious condition than Mobitz type I, which involves the AV node. This is because, should complete heart block develop, the escape rhythm will be ventricular and possibly unstable and unpredictable. In contrast, complete heart block with Mobitz type I is associated with an escape junctional rhythm that is stable. Hence a Mobitz type II second-degree AV block is an indication for a pacemaker if there are symptoms suggesting bradycardia or if there is transient complete heart block.

continues
The ECG also shows a borderline criterion for left ventricular hypertrophy (S-wave depth in lead V2 $R V 2$ + R-wave amplitude in lead V4 $RV 4$ = 36 mm), and there are diffuse T-wave abnormalities ($T \uparrow$).

The patient’s clinical history and physical examination are significant for profound dehydration, likely resulting in a pre-renal state of acute renal insufficiency. Although blood urea nitrogen and creatinine are not reported, low blood pressure in a normally hypertensive patient generates concern for the potential of renal insufficiency due to reduced renal perfusion. One would expect a reflex tachycardia in the setting of hypotension. However, the patient has been taking atenolol, which can blunt this effect. Since atenolol is primarily cleared renally, it is likely that the patient’s bradycardia is a result of high atenolol levels in the setting of a pre-renal state.

However, the β-blocker will not affect conduction through the His-Purkinje system. Therefore, another etiology for Mobitz II should be sought. It may be the result of other metabolic abnormalities associated with renal insufficiency that will alter His-Purkinje conduction. Another possible cause is underlying disease of the His-Purkinje system that was exposed by the metabolic abnormalities. Although the symptoms of pre-syncope and dizziness can be explained by dehydration and hypotension, Mobitz II with subsequent complete heart block could also account for the symptoms. Complete heart block in this situation would be associated with a ventricular escape rhythm. Therefore, continued monitoring after correction of dehydration, metabolic abnormalities, and renal insufficiency would be indicated to evaluate for the presence of continued conduction abnormalities.
A 49-year-old man with diabetes and hypertension presents acutely to the emergency department following 6 hours of substernal chest pressure. When an ECG on presentation shows inferior ST-segment elevations, the patient is taken urgently for cardiac catheterization; a stent is placed in the distal right coronary artery. His ST segments normalize and his chest pain resolves. The next morning, the patient continues to be asymptomatic. A routine ECG is obtained.

How would you classify the rhythm disturbance?
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, second-degree AV block (2:1 AV conduction or block), clockwise rotation, old inferior wall myocardial infarction (MI)
The ECG shows an underlying regular ventricular rate of 45 bpm. There is a P wave (+) before each QRS complex. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a sinus rhythm. A second on-time but nonconducted P wave (^) can be seen between each QRS complex. Therefore, there is a stable normal sinus rhythm (□) at a rate of 90 bpm. The P waves that are conducted have a stable PR interval of 0.20 second (↔).

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (420/360 msec). Noted is poor R-wave progression, which is the result of clockwise rotation of the electrical axis in the horizontal plane, established by imagining the heart as viewed from under the diaphragm. With clockwise rotation, left ventricular forces occur later (more leftward) in the precordial leads. Also noted are Q waves (↑) in leads III and aVF, which are characteristic of an inferior wall myocardial infarction (MI). There are also U waves (▲) in leads V2-V4; these are normal.

Therefore, this ECG demonstrates second-degree AV block with 2:1 AV conduction, although it cannot be established if this is a Mobitz I with 2:1 AV conduction or AV block or Mobitz II with 2:1 conduction. In the acute setting of an MI, the indications for temporary pacing include Mobitz type II AV block, given the high risk for progressing to complete heart block with an escape ventricular rhythm that could be unstable. Therefore, making the distinction between Mobitz type I and II is clinically important. Since this patient had an inferior wall MI, which is often associated with transient AV nodal conduction abnormalities resulting from either increased vagal tone or inflammation and/or edema around the AV junction, it may be assumed that this is a Mobitz type I and will resolve. In the presence of an anterior wall MI, there is often permanent damage to the septum and His-Purkinje system, which would be associated with a Mobitz type II block and, given the permanently damaged conduction system, with a risk for complete heart block and an escape ventricular rhythm.

However, the only way to establish the etiology with certainty is to see another pattern develop during monitoring. Therefore, if a pattern typical for Mobitz type I or complete heart block with an escape junctional rhythm developed, then the 2:1 AV block would be due to Mobitz type I. A subsequent pattern of Mobitz II or the development of complete heart block with a ventricular escape rhythm would confirm that the 2:1 AV block was the result of Mobitz II.
An 18-year-old Olympic track and field competitor needs a complete medical evaluation prior to running in her upcoming competitions. She has no medical complaints, her medical history reveals no major problems, and her physical examination is completely normal. An ECG is obtained.

How would you classify the rhythm disturbance?
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, left axis, first-degree AV block, second-degree AV block (Mobitz type I)
The heart rate (36 bpm) is stable initially, with a consistent RR interval. There are P waves (+) before each QRS complex, with a stable PR interval (↔) (0.28 sec). A second on-time but nonconducted P wave is seen after each QRS complex (▼). The PP intervals are constant (⊥), at a rate of 72 bpm. The P waves are upright in leads I, II, aVF, and V4-V6. Hence there is an underlying sinus rhythm. The QRS complex duration is normal (0.08 sec), and the axis is leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (400/310 msec).

Therefore, the initial portion of the ECG has a pattern of second-degree AV block with 2:1 AV conduction or 2:1 AV block (ie, every other P wave is nonconducted [▼]). The baseline PR interval is stable but prolonged at 0.28 second, indicating that a first-degree AV block (prolonged AV conduction) is also present. At the end of the ECG recording (fifth and sixth QRS complexes), there is a change in the QRS pattern. The fifth QRS complex (●) is conducted with a PR interval of 0.28 second, which is the baseline PR interval. However, the sixth QRS complex (▼), which is preceded by an on-time P wave (●), is early and the PR interval preceding it is longer (0.52 sec). After the sixth QRS complex there is a nonconducted P wave (▼), which can be timed out by marching out the PP intervals; this occurs on top of the T wave of the sixth QRS complex (▼), altering its morphology. Hence there is a brief period of a second-degree heart block with a pattern of a Mobitz type I and 3:2 conduction. Therefore, the 2:1 AV block in this situation represents a Wenckebach pattern (Mobitz type I) and not Mobitz type II.

The presence of Mobitz type I, an AV nodal conduction problem, in a young athlete is the result of high vagal tone, which can reduce the rate of conduction through the AV node and result in decremental conduction at a slower rate, and hence the Mobitz type I. High vagal tone also occurs at night, while sleeping, so Mobitz type I is not uncommon at this time. Other causes for Mobitz type I could be AV nodal blocking agents or intrinsic AV nodal disease. This conduction abnormality does not require any additional therapy and often resolves when there is an increase in sympathetic tone and a reduction in vagal tone, as with exercise.
A 17-year-old male high school senior comes to your clinic with worsening exercise tolerance. He has noticed that over the past 6 months he has been unable to keep up during his track and field practices. He remembers being told that he was born with a slow heart rate, but no treatment was needed at the time. The following ECG is obtained. An echocardiogram confirms that there is no structural heart disease.

What is the rhythm disturbance?
What is the most likely underlying etiology for this ECG finding?
What is the next step in management?
Podrid’s Real-World ECGs

ECG 40 Analysis: Normal sinus rhythm, complete heart block (third-degree AV block), escape junctional rhythm
The ECG shows an underlying regular atrial rate (regular PP interval) of 72 bpm. The P waves (+) are positive in leads I, II, aVF, and V4-V6. Some of the P waves are not obvious as they are at the end of the QRS complex (*), in the ST segment (^), or on T waves (▼). Hence there is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF).

However, the ventricular rate is 40 bpm and the RR intervals are constant (┐). The QT/QTe intervals are normal (460/380 msec). Additionally, the PR intervals (↔) are variable without any notable pattern (ie, there is no association between the P waves and the QRS complexes). This is termed AV dissociation as the atrial rate is independent of the ventricular rate. The atrial rate is faster than the ventricular rate, which is characteristic of third-degree or complete AV block. The QRS complexes are narrow and normal in morphology; therefore, the escape rhythm is nodal or junctional, meaning that the location of the conduction block is the AV node. The location of the escape rhythm is based not on its rate but rather on the morphology of the QRS complexes, which is normal in this case. Junctional escape rhythms tend to be predictable, better tolerated, and more stable than ventricular escape rhythms.

In view of the history of a slow heart rate since birth and the finding of complete heart block at a young age, the most likely etiology for the patient’s complete heart block is neonatal lupus, an immunologic process involving transplacental migration of maternal antibodies (anti-Ro and anti-La antibodies) that bind to fetal cardiac tissue and destroy the AV node. Neonatal lupus is responsible for the majority of cases of congenital complete heart block. The diagnosis is often missed due to a lack of symptoms. Because some affected individuals do not develop complete heart block until later in life, they often present for the first time in childhood. The major indications for pacemaker placement in this setting include symptomatic bradycardia, significant exercise intolerance, heart failure related to bradycardia, left ventricular systolic dysfunction, or a wide QRS escape rhythm.
Notes
A 56-year-old man with known coronary artery disease and ischemic cardiomyopathy with an ejection fraction of 25% presents to the emergency department with syncope. He is currently experiencing symptoms of lightheadedness. You perform a physical examination and are able to diagnose a rhythm disturbance. An ECG confirms your suspicions.

What is the rhythm abnormality?
What findings on the physical exam support this diagnosis?
ECG 41 Analysis: Normal sinus rhythm, complete heart block (third-degree AV block), escape ventricular rhythm
The ECG shows P waves (+) at a rate of 80 bpm. Some of the P waves are within the ST segments or T waves (▼) and not obvious. The PP intervals are constant (□). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. The ventricular rate (RR intervals) is also regular (), but at a rate of 30 bpm. There is no stable relationship between the P waves and the QRS complexes (PR intervals are variable [↔]), and hence there is AV dissociation. The atrial rate is faster than the ventricular rate; therefore, this is third-degree or complete AV block.

The QRS complexes are wide (0.16 sec) and abnormal in morphology, without a typical pattern for a right or left bundle branch block. Therefore, the escape rhythm is ventricular in origin and the location of the AV block is within the His-Purkinje system. The QRS complexes are ventricular, so abnormalities of the ventricular myocardium cannot be diagnosed because neither left nor right ventricular activation occurs via the normal His-Purkinje system. Among the most common etiologies for this rhythm is ischemic heart disease (especially with a previous myocardial infarction), severe cardiomyopathy, or idiopathic conduction system disease (Lev’s or Lenègre’s disease).

The major physical exam findings of complete heart block besides bradycardia include cannon A waves in the neck, a variable S1, and fluctuations in blood pressure reflecting the independent contraction of the atria and ventricles. Cannon A waves, which are irregular A waves with increased volume and amplitude, are visible in the jugular venous pulsation when the right atrium intermittently contracts against a closed tricuspid valve. This occurs when a P wave (atrial activation) occurs simultaneously with a QRS complex (ventricular activation). In complete heart block, the P waves are more frequent than the QRS waves, so the cannon A waves are interspersed with A waves that have a normal amplitude. The intensity of S1 is determined by the extent to which the AV valve leaflets (mitral and tricuspid) are open just before they close with ventricular contraction. When the AV valve leaflets are farther apart (ie, with a short PR interval), S1 intensity is increased. When the AV valve leaflets are closer together (ie, with a long PR interval in which the leaflets float back together), the S1 intensity is reduced. In complete heart block, since the P waves are at variable times in relation to the QRS complexes, the mitral and tricuspid valve leaflets are open to varying degrees before each ventricular contraction. This results in a variable S1 sound. The variability of the blood pressure results from changes in beat-to-beat stroke volume caused by variability in ventricular filling from left atrial contraction.
A 75-year-old woman presents with intermittent lightheadedness and dyspnea. An ECG is obtained. She is otherwise healthy and is taking no medications. An echocardiogram reveals significant calcification of the mitral annulus and ventricular septum.

What is the rhythm disturbance?
What is the anatomic location of the abnormality?
**Podrid’s Real-World ECGs**

**ECG 42 Analysis:** Sinus tachycardia, complete heart block (third-degree AV block), ventricular escape rhythm
The ECG shows regular P waves (+) with a stable PP interval (□) at a rate of 120 bpm. It is important to determine the sinus rate by identifying two sequential P waves and then making certain that the P waves, when seen, are on time (ie, they “march out,” or occur at a regular interval). They should be at a regular interval even if they are not apparent everywhere (^); that is, they may occur simultaneously with the QRS complex and hence not be obvious. This is best seen in the lead II rhythm strip. The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The ventricular rate is stable at 75 bpm (constant RR intervals [□]). There is no relationship between the P waves and QRS complexes as the PR intervals are variable (↔); hence there is AV dissociation. As the atrial rate is faster than the ventricular rate, this represents third-degree or complete heart block.

The QRS complexes are wide (0.14 sec) and abnormal in morphology, resembling neither a right or left bundle branch block. This represents a ventricular escape rhythm, even though the ventricular rate is 75 bpm, faster than what is expected from a ventricular escape focus. The etiology of the escape rhythm in complete heart block is based on the QRS morphology and not the ventricular rate. The ventricular rate is faster than expected in this case because of enhanced sympathetic stimulation, indicated by the fact that the sinus rate is 120 bpm. As the escape rhythm originates in the ventricle, the site of the AV conduction block is within the His-Purkinje system. This patient has no history of underlying heart disease and hence the most likely etiology is Lev’s or Lenègre’s disease, conditions involving fibrosis and calcification of left-sided cardiac structures, including the mitral annulus and ventricular septum, that often result in AV conduction system disease.
The following ECG is from a 72-year-old man who is in the recovery room after undergoing an elective cholecystectomy. His vital signs are normal.

What is the diagnosis?
How would you manage this patient?
ECG 43 Analysis: Normal sinus rhythm, isorhythmic dissociation, junctional rhythm, left ventricular hypertrophy (LVH), ST-T wave abnormalities
The ECG shows a stable ventricular rate of 54 bpm. The QRS complex duration is normal (0.09 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/380 msec). P waves (+) can be seen in front of each QRS complex at a rate of 54 bpm. The P waves are upright in leads I, II, aVF, and V4-V6; hence they are sinus in origin. However, the PR intervals are short and not stable. Therefore, AV dissociation is present. Nevertheless, the atrial (PP intervals [ ]) and ventricular (RR intervals [ ]) rates are identical. When the atrial rate is faster than the ventricular rate, third-degree or complete heart block is present. In contrast, when AV dissociation is present but the ventricular rate is faster than the atrial rate, the etiology is an accelerated lower ectopic focus (either junctional or ventricular). However, when the atrial and ventricular rates are identical, the etiology for the AV dissociation cannot be established and this is termed isorhythmic dissociation.

If this were an accelerated junctional rhythm, any maneuver to increase the sinus rate (eg, administration of atropine) would result in restoration of normal conduction as the increased sinus rate would overdrive the lower focus and result in sinus captures and a sinus rhythm with a stable PR interval. In contrast, with complete heart block, an increase in the sinus rate would not result in capture and AV dissociation would persist, with now an atrial rate that is faster than the ventricular rate. Isorhythmic dissociation can be seen in the post-anesthesia setting and often resolves on its own. As long as the patient is stable, there is no specific treatment other than avoidance of any nodal blocking agents.

Also noted on this ECG is a deep S wave in lead V2 ( [ ] ) and a tall R wave in lead V5 ( [ ] ). Together they have an amplitude of 55 mm, which meets one of the criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth in leads V1-V2 + R-wave amplitude in leads V5-V6 ≥ 35 mm). Also, ST-T wave (↑) abnormalities are noted in leads I, II, aVL, and V4-V6; these are associated with LVH and probably represent chronic subendocardial ischemia.
Notes
A 21-year-old college student is seen at his student health service for intermittent palpitations. He otherwise has no complaints, and his physical examination is normal. An ECG is obtained.

What is the abnormality seen on this ECG?
What is the most likely etiology for the patient’s palpitations?
Podrid’s Real-World ECGs

ECG 44 Analysis: Sinus bradycardia, Lown-Canong-Levine (LCL) pattern
(preexcitation, accelerated AV conduction)
There is a regular rhythm at a rate of 54 bpm. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/380 msec). There is a P wave (+) before each QRS complex, and the interval is stable but very short (‖) at 0.10 second (normal, 0.14–0.20 sec). The P waves are upright in leads I, II, aVF, and V4-V6, indicating that this is a sinus bradycardia.

The short PR interval indicates enhanced AV conduction, which has two etiologies. One is accelerated AV nodal conduction and the other is a preexcitation condition known Lown-Ganong-Levine (LGL) pattern. The LGL pattern on the ECG is due to an accessory pathway, known as the bundle of James, which originates in the atrial myocardium and bypasses the AV node, connecting directly to the bundle of His. As the AV node is bypassed (the AV node being the site of slowest conduction in the heart), AV conduction time is fast. Since the activation of the ventricles is via the normal His-Purkinje system, the QRS complexes are narrow and normal in morphology. As there are two pathways that link the atrium to the ventricles (ie, the normal AV node and the bundle of James), LGL is associated with a specific form of a reentrant arrhythmia known as AV reentrant tachycardia, which results from antegrade conduction to the ventricles via one of the pathways and retrograde conduction back to the atria via the other pathway. Other atrial arrhythmias, such as atrial tachycardia, atrial flutter, and atrial fibrillation, may also occur in association with LGL. However, these arrhythmias are not the result of the two pathways; instead, they occur independently, generated within the atrial myocardium, but use the accessory pathway to transmit the impulses to the ventricles. In this situation the ventricular response rate may be very rapid, as the AV node (which controls the ventricular response rate with atrial arrhythmias) is bypassed. When arrhythmias occur in association with the LGL pattern, this is known as the LGL syndrome.
A 32-year-old man with diabetes comes to your clinic with complaints of intermittent lightheadedness. He denies any episodes of chest pain, chest pressure, or shortness of breath. His exercise tolerance has been stable, but he does admit to living a sedentary lifestyle. Another healthcare provider was concerned about his having a myocardial infarction and recommended cardiac catheterization. He is in your clinic for a second opinion. You obtain an ECG.

What is the most likely cause of this patient’s symptoms?
Does he need a cardiac catheterization?
**ECG 45 Analysis:** Normal sinus rhythm, Wolff-Parkinson-White (preexcitation) pattern
The ECG shows a regular rate of 60 bpm, and a P wave (+) of normal morphology (upright in leads I, II, aVF, and V4-V6) can be seen in front of each QRS complex. Hence this is a normal sinus rhythm. Although the PR interval is stable, it is very short (\( \| \)) (0.12 sec). In addition, the QRS complex is widened to 0.14 second and there is a slow or slurred upstroke to the QRS complex; this is called a delta wave (↑, ↓). As a result of the delta wave the base of the QRS complex is widened while the peak is narrow.

The QT/QTc intervals are normal (400/400 msec and 340/340 msec when adjusting for the prolonged QRS duration). The presence of a short PR interval and a wide QRS complex (due to a delta wave) is termed Wolff-Parkinson-White (WPW) pattern. It is the result of an accessory pathway, called the bundle of Kent, that links the atrium directly to the ventricle and bypasses the AV node, accounting for the short PR interval. Since the initial activation of the ventricle is via the fast-conducting accessory pathway and not the normal His-Purkinje system, the initial portion of the QRS complex is slow in upstroke and the complex is widened due to the abnormal left ventricular activation that is directly through the ventricular myocardium; hence the widened base of the QRS complex. This initial slowed ventricular activation produces the slurred QRS upstroke, which is termed a delta wave.

However, WPW is due to a fusion complex resulting from left ventricular activation via two different pathways. The initial part of the QRS complex is due to early activation (preexcitation) via the accessory pathway, and later activation (terminal portion of the QRS complex) is via the normal AV node–His-Purkinje system. The impulse via these two pathways “fuses,” resulting in a fusion complex. Therefore, the terminal portion of the QRS complex is the result of activation via the normal AV node–His-Purkinje pathway; the peak of the QRS complex is narrow.

The PR interval, the duration of the delta wave, and hence the QRS width are dependent on the relative balance of conduction via the two pathways. This is related to AV nodal conduction as the accessory pathway is similar to Purkinje fibers and conduction through this pathway is “all or none” and hence not variable. If conduction via the normal nodal His-Purkinje pathway is relatively slow compared with conduction via the accessory pathway, there is more myocardial activation via the accessory pathway and the PR interval is shorter, the delta wave continues.
and QRS complexes are wider, and the QRS complex is more abnormal. In contrast, if conduction via the normal AV node–His-Purkinje system is more rapid, less myocardium is activated via the accessory pathway and the PR interval is longer, the delta wave is less prominent, and the QRS complex is narrower and more normal in morphology.

Since left ventricular activation (to a greater or lesser extent) is initiated by conduction via an accessory pathway directly through the ventricular myocardium and not via the normal His-Purkinje pathway, abnormalities of the left ventricular myocardium (i.e., infarction, ischemia, left ventricular hypertrophy, inflammation such as pericarditis or myocarditis) cannot be diagnosed reliably on ECG. The ECG has inferior Q waves and diffuse nonspecific ST-segment and T-wave abnormalities. In the presence of a WPW pattern this is a pseudo inferior infarction pattern and is the result of a posteroseptal bypass tract. It is not an inferior wall M1 but is actually a negative delta wave. In the absence of any other clinical evidence suggesting ischemia, there is no indication for cardiac catheterization.

The short PR interval, delta wave, and widened QRS complex are diagnostic for a WPW pattern. Since there are two pathways linking the atria to the ventricles (the bundle of Kent and the normal AV node–His-Purkinje system), a large (macro) circuit is formed, linked proximally by the atrial myocardium and distally via the ventricular myocardium. The presence of this macro-circuit predisposes to a reentrant form of supraventricular tachycardia called AV reentrant tachycardia. Other supraventricular tachyarrhythmias, such as atrial tachycardia, atrial flutter, and atrial fibrillation, may also occur; however, they are not dependent on the presence of this circuit but rather use the accessory pathway as an alternative pathway for activation of the ventricles. In this situation the ventricular response rate may be very rapid, as the AV node (which controls the ventricular response rate with atrial arrhythmias) is bypassed. The presence of arrhythmias in a patient with a WPW pattern on the ECG defines the WPW syndrome.
A 44-year-old woman with schizophrenia and a history of narcotic dependence presents to your clinic with dysuria. A urinalysis is positive for white blood cells, and a urine culture reveals a gram-negative bacterium that is sensitive to ceftriaxone and levofloxacin. The patient has a known allergy to cephalosporins. Her medications include quetiapine and methadone. She is otherwise healthy and has a normal physical examination.

Why do you need an ECG before you start this patient on antibiotics?
Podrid's Real-World ECGs

ECG 46 Analysis: Normal sinus rhythm, normal ECG
The ECG shows a regular rhythm at a rate of 64 bpm. The P wave (+), which has a normal duration of 0.12 second, is upright in leads I, II, aVF, and V4-V6; the P wave is biphasic in lead V1 (+), which is normal. This is, therefore, a normal sinus rhythm.

Each QRS complex is preceded by a P wave, and each P wave is followed by a QRS complex. The axis in the frontal plane is normal as the QRS complex is positive in leads I and aVF, thereby indicating that the axis is between 0° and +90°. The PR interval (↔) is normal at 0.20 second and it is constant, the QRS width is normal at 0.08 second, and the QT/QTc intervals (┌┐) are normal at 360/370 msec. There is normal R-wave progression across the precordium (ie, the R waves become progressively higher in amplitude and the S waves have less depth going from leads V1 to V6). The transition point (R/S > 1) occurs between leads V3 and V4. The ST segments, which have a normal concave morphology (▲), are at baseline (determined by the TP segment [↑]). The T waves have a normal morphology (▼); they are upright in leads with a positive QRS complex and are asymmetric, with a slower upstroke and a more rapid downstroke. Lead aVR, being the mirror image of all the other limb leads, shows a negative P wave, QRS complex, and T wave, all of which are normal in this lead. Hence this is a normal ECG.

This patient needs a baseline ECG prior to starting her on a quinolone in order to document her QT/QTc intervals. She is already chronically taking two agents known to prolong the QT/QTc intervals. The antipsychotics, including haloperidol and quetiapine, are a family of compounds known to cause QT prolongation. Methadone is the one narcotic that also can cause significant QT prolongation. Among the antibiotics, quinolones and macrolides are known to prolong the QT interval. Prolongation of the QT interval is more likely to occur when multiple drugs that can cause this change are prescribed. Therefore, it is essential to check baseline and follow-up QT intervals. Prolongation of the QT interval puts a patient at risk for a serious ventricular tachyarrhythmia called torsade de pointes.
Notes
A 56-year-old man with hypertension and hyperlipidemia underwent cardiac catheterization earlier in the day in the setting of progressive anginal symptoms and was found to have a 95% distal left anterior descending artery lesion. The stenosis was successfully stented. No other coronary lesions were identified. Later in the day, he becomes tachycardic and the following ECG is obtained. His vital signs are otherwise stable and he is asymptomatic.

Which of the following is the most likely cause of this patient’s tachycardia?

A. In-stent restenosis  
B. Acute stent thrombosis  
C. Acute bleed  
D. Infected stent with sepsis  
E. New stenosis of the right coronary artery
Podrid’s Real-World ECGs

ECG 47 Analysis: Sinus tachycardia, upsloping ST-segment depression
The ECG shows a regular rhythm at a rate of 200 bpm. Any heart rate over 100 bpm is defined as a tachycardia. The P wave (+) is upright in leads I, II, aVF, and V4-V6; it appears to be biphasic in lead V1. Hence this is a sinus tachycardia. Each QRS complex is preceded by a P wave, and each P wave is followed by a QRS complex. It should be pointed out that the P wave may not be seen clearly in every lead; for example, it is not well seen in leads V2-V3, but it can be seen clearly in lead V1 (▼). Because every column is simultaneous, the P wave is present in leads V2-V3 but is just not obvious because it is superimposed at the end of the T wave in these leads (^). The axis in the frontal plane is normal, between 0° and +90° (the QRS complex is upright in leads I and aVF).

The PR interval is 0.12 second (‖), which is short as a result of the rapid sinus rate. This is due to an increase in conduction velocity through the AV node as a result of sympathetic stimulation, which is the usual cause of a sinus tachycardia. The QRS width is 0.08 second (normal), and the QT/QTc intervals are normal (220/400 msec). There is normal R-wave progression across the precordium (R wave becomes progressively higher in amplitude and S wave less deep going from lead V1 to lead V6), with the transition point (R/S > 1) between V3 and V4. The ST segments have a normal concave morphology (↑), and while they are at baseline in most leads (in the presence of a sinus tachycardia during which the TP segment cannot be seen, the PR segment [▲] is used to define baseline) there is some J-point and upsloping ST-segment depression in leads V4 and V6 (↓). In the presence of a tachycardia, J-point and ST-segment depression may be a normal finding as it is due to atrial repolarization (T wave of the P wave).

continues
With sinus tachycardia and the shortening of the PR interval, the T wave of the P wave, which normally occurs during ventricular depolarization and is within the QRS complex, moves out from the QRS complex and is superimposed on the J point, which now becomes depressed. As a result, the ST segment slopes upward toward baseline. Therefore, in the presence of upsloping ST segments, the presence of actual ST-segment depression, which is the marker of ischemia, is established by evaluating the ST segment at 0.08 second (two small boxes) beyond the J point. If at this time the ST segment is more than 1.5 mm below the baseline, it is considered to be abnormal ST-segment depression, suggestive of ischemia. An ST segment that is at baseline at this time is considered to be normal. Such is the case on this ECG. Although the T waves are tall and peaked (a frequent finding during a tachycardia), they are still asymmetric with a slower upstroke and more rapid downstroke. Noted is that lead aVR, which is the mirror image of the other limb leads, has a negative P wave, QRS complex, and T wave.

Tachycardia in the setting of a cardiac catheterization should always be evaluated urgently for potential procedural complications. The most worrisome complications include acute stent thrombosis, pericardial tamponade, aortic dissection, a ventricular arrhythmia, or a serious bleed such as a retroperitoneal bleed, all of which can be associated with tachycardia and hemodynamic instability. In this case, the patient’s ECG does not suggest acute stent thrombosis, which is total occlusion of the stent and results in signs and symptoms of an acute ST-segment elevation myocardial infarction. In-stent restenosis, which results from smooth muscle cell migration, proliferation, and hyperplasia within stents, is unlikely given that it is a chronic process, occurring several months after stent insertion and not within 1 to 2 days. Similarly, coronary artery disease is a chronic process, so a new stenosis in the right coronary artery developing within a day is unlikely. Infection of stents is an extremely rare phenomenon. However, bleeding often occurs due to the antiplatelet therapy and anticoagulation administered in the setting of a percutaneous coronary intervention. This can either be from a retroperitoneal bleed, gastrointestinal bleed, or an expanding groin hematoma.
The following ECG is obtained from a sleeping patient with complaints of daytime sleepiness and morning headaches.

What are the most likely diagnosis and treatment?
Podrid's Real-World ECGs

ECG 48 Analysis: Sinus bradycardia
The rhythm is regular at a rate of 44 bpm, defined as a bradycardia. The P wave (+) is upright in leads I, II, aVF, and V4-V6; it is biphasic in lead V1 (^), which is normal. Hence this is a sinus bradycardia. Each QRS complex is preceded by a P wave, and each P wave is followed by a QRS complex. The P-wave duration is normal at 0.12 second, and the PR interval is constant at 0.20 second.

The axis in the frontal plane is normal, between 0° and +90° (the QRS complex is positive in leads I and aVF). There is a deep but narrow Q wave in lead III (↑). However, lead III is an indeterminate or ambiguous lead and hence abnormalities that are present in only this lead are nondiagnostic. The QRS complex duration is 0.08 second, the QT interval is 440 msec, and the QT interval corrected for heart rate (ie, QTc) is 380 msec, all of which are normal. The ST segments have a normal concave morphology and are at baseline (defined as the TP segment [↔]) in most leads. However, a slight ST-segment elevation (↓) can be seen in leads V2-V3, which represents early repolarization and is a normal variant seen in young individuals who have tall QRS complexes as well as in those with left ventricular hypertrophy. There is flattening of the ST segment in leads II, aVF, and V5-V6 (▲), which is a nonspecific change. The R-wave progression from leads V1 to V6 is normal (R-wave amplitude progressively increases while S-wave depth progressively decreases). The T waves have a normal morphology (ie, they are upright in all leads and are asymmetric with a slower upstroke and faster downstroke). Noted is that lead aVR, which is the mirror image of the other limb leads, has a negative P wave, QRS complex, and T wave.

Sinus bradycardia is often seen in situations with enhanced vagal tone, such as during sleep or in young well-conditioned individuals, and does not represent a pathologic process. However, bradyarrhythmias during sleep can also be observed with sleep apnea, particularly if they correlate with episodes of oxygen desaturation. Daytime sleepiness and morning headaches are common symptoms of sleep apnea, and the treatment is nocturnal continuous positive airway pressure.
This ECG is obtained from a 20-year-old marathon runner.

What is the diagnosis?
Podrid’s Real-World ECGs

ECG 49 Analysis: Sinus arrhythmia
The ECG shows a sinus rhythm with P waves (+) of uniform morphology before each QRS complex. The P waves are upright in leads I, II, aVF, and V4-V6. The PR interval is constant (↔) at 0.16 second. However, the rhythm is not regular (⊔) and the heart rate (RR intervals) ranges from 36 to 62 bpm. There is no pattern to the irregular heart rate (ie, it is irregularly irregular). The QRS complex duration is normal (0.08 sec), as is the morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF), and the QT/QTc intervals are normal (400/370 msec). Also noted are prominent but normal U waves (^) in leads V2-V3.

Only three supraventricular rhythms are irregularly irregular: 1) sinus arrhythmia in which there is one P-wave morphology and a stable PR interval, 2) multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) in which there are three or more different P-wave morphologies and PR intervals without one dominant P wave, and 3) atrial fibrillation in which there is no organized atrial activity or P waves. Atrial flutter and atrial tachycardia may be irregular, but there will be a pattern based on the degree of AV block; hence these rhythms will be regularly irregular.

The etiology for this arrhythmia is sinus arrhythmia and it is a respirophasic rhythm (ie, the changes in the sinus rate are the result of respiration). Heart rate increases with inspiration and decreases with expiration. These fluctuations in heart rate are mediated by changes in vagal tone and are a normal finding. It may be more apparent in patients with high vagal tone, such as athletes or other well-conditioned people. Respiratory sinus arrhythmia serves to increase pulmonary blood flow during lung inflation. As it is a normal physiologic change in heart rate related to respiration, no further evaluation or therapy is warranted.
A 44-year-old woman with anxiety presents with the frequent sensation of “skipped heartbeats.” She otherwise denies chest pain, dyspnea, syncope, or lightheadedness. She smokes two packs of cigarettes per day and drinks three glasses of wine per day to calm her nerves. She takes clonazepam and was recently started on a β-blocker for the palpitations. Her physical examination is normal. An ECG is obtained.

What is the etiology of her symptoms?
How would you treat her?
Podrid’s Real-World ECGs

**ECG Analysis:**
- Sinus bradycardia, first-degree AV block,
- Low-voltage limb leads, premature atrial complexes
The ECG shows an irregular rhythm at a rate of 50 bpm. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVf). There is low voltage in the limb leads (< 5 mm in each limb lead). The QT/QTc intervals are normal (400/370 msec).

The rhythm is irregular as a result of several complexes that are early (↓) (second, sixth, and eighth). There is a P wave (+) before each QRS complex, but there are different PR intervals. The PR interval before the first, third, fourth, fifth, and seventh QRS complexes is constant at 0.24 second (↔) (ie, a first-degree AV block or prolonged AV conduction). The P wave is positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes with a first-degree AV block. The three QRS complexes that occur early (↓) (complexes 2, 6, and 8) are also preceded by an early P wave (▼), but the P-wave morphology and PR interval (┘) (0.26 second) are different from those of the sinus QRS complexes. The QRS complexes have the same duration and morphology as the sinus complexes. These are premature atrial complexes. As a result the rhythm is irregular, but the premature atrial complexes have a fixed coupling interval (┏) with the sinus beats and hence there is an underlying pattern to the QRS complexes; thus the rhythm is regularly irregular.

Premature atrial complexes are identified by the following:

- An early (premature) P wave preceding a premature QRS complex. The P-wave morphology and/or the PR interval differ from that of sinus rhythm.
- Premature atrial complexes may be unifocal (each premature atrial complex has the same P-wave morphology) or multifocal (the premature atrial complexes have different P-wave morphologies).
- Following the premature atrial complex there is a pause of variable duration; the PP interval surrounding the premature atrial complex may be less than, the same as, or greater than two PP intervals. The variability reflects the effect that the premature atrial complex has on the sinus node; that is, it may not alter the sinus rate, it may reset the sinus node, or it may depress sinus node activity.

The pause associated with the first premature atrial complex (┗) (ie, second QRS complex) is equal to two PP intervals; hence the premature atrial complex did not affect sinus node automaticity. However, the

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pause after the second premature atrial complex (ie, sixth QRS complex) is much greater than two PP intervals; this is the result of sinus node depression by the premature atrial complex with a longer time before sinus node impulse generation recurred. This is probably due to depression of sinus node automaticity as a result of the β-blocker. However, it may also be a manifestation of underlying sinus node dysfunction.

Premature atrial complexes are often asymptomatic but can be associated with symptoms of palpitations or the sensation of skipped heartbeats. The sensation of a skipped beat is due to the fact that the stroke volume of the premature beat is small because there is less time for ventricular filling. However, the beat after the premature beat has an increased stroke volume due to the pause that results in increased ventricular filling. This increased stroke volume results in an increase in left ventricular contractility, due to the Starling effect. The increased volume and force of left ventricular ejection cause a sensation of palpitations.

Reassurance should be given to this patient that the rhythm is benign and can often go untreated as long as there are no major symptoms. Indeed, premature atrial complexes occur in up to 70% of normal healthy people, although they are infrequent in most. However, given the patient’s symptoms, a reasonable initial approach would be to remove potential triggers of the premature beats by reducing alcohol or caffeine intake or quitting smoking. These factors have been associated with increased frequency of premature atrial complexes. If this is unsuccessful, β-blockers can be used to reduce the associated symptoms (ie, the sensation of palpitations due to reduction in left ventricular inotropy). Although β-blockers are not likely to reduce the frequency of premature atrial complexes because they do not affect the atrial myocardium directly, they may reduce the frequency if there is an associated sympathetic influence to the premature complexes. Antiarrhythmic agents that do have a direct effect on the atrial myocardium include the class IA (quinidine, disopyramide), class IC (propafenone, flecainide), and class III (amiodarone, sotalol, and dofetilide) agents.
A 24-year-old pregnant woman presents with palpitations. She has no past medical history and is only taking prenatal vitamins. She denies any caffeine or alcohol intake and does not smoke. The following ECG is obtained.

What are your next steps in management?
ECG 51 Analysis: Normal sinus rhythm, premature atrial complexes in a bigeminal pattern (atrial bigeminy)
The ECG shows an irregular rhythm at a rate of 84 bpm. Although it is irregular, there is a repeating pattern of long (□) and short (■) RR intervals; hence the rhythm is regularly irregular.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/440 msec). Every other QRS complex (ie, third, fifth, seventh, ninth, 11th, and 13th) is early (↓) or premature. A P wave (+) precedes each of these QRS complexes, and it is positive in leads I, II, aVF, and V4-V6. The PR interval is stable (0.16 sec) (╔╗). However, the QRS complexes that follow the longer RR interval have a different P-wave morphology (∗) and PR interval (0.20 sec) (╚╝). Hence the complexes after the pause are sinus complexes and they are followed by premature atrial complexes that have a constant relationship to the preceding P wave (hence a fixed coupling interval [↔]).

When every other QRS complex is a premature atrial complex in a repeating pattern, the rhythm is called atrial bigeminy. When every third QRS complex is a premature atrial complex the term used is atrial trigeminy. Bigeminy and trigeminy indicate a repeating pattern; otherwise there are no important clinical implications. It should be noted that on occasion the premature P wave is superimposed on the preceding T wave, altering its morphology.

Reassurance should be given to the patient that this is a benign rhythm of pregnancy. Premature atrial complexes may occur as a result of the increased plasma volume during pregnancy, which results in stretch of the atria and the development of enhanced automaticity. It is likely that the symptoms are also due to the increased plasma and stroke volume associated with pregnancy. In the absence of any history of heart disease or cardiac symptoms, it is unnecessary to pursue any further workup. No specific medical therapy is necessary as long as the patient can tolerate her symptoms. Indeed, drugs should be avoided during pregnancy; in this case they should be used only if there is a symptomatic or potentially serious sustained arrhythmia.
The following resting ECG is obtained as part of a routine evaluation of a healthy 28-year-old medical student.

What is the most likely mechanism for the abnormal finding?
Podrid’s Real-World ECGs

ECG 52 Analysis: Atrial rhythm
The ECG shows a regular rhythm at a rate of 64 bpm. The QRS complexes are normal in duration (0.08 sec) and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/410 msec). Although there is a P wave (+) in front of each QRS complex with a stable PR interval (0.10 sec), the P wave is abnormal as it is inverted or negative in leads II, aVF, and V4-V6. Therefore, this is not a sinus rhythm in which the P wave is positive in leads I, II, aVF, and V4-V6. Rather, the P wave is the result of an impulse generated by some other focus within the atrial myocardium, but not the sinus node. This is an ectopic atrial rhythm that is identified by the presence of a distinct P wave of uniform morphology before each QRS complex, but it is negative or biphasic in leads in which it should be positive (ie, leads I, II, aVF, and V4-V6). As atrial activation is no longer via the normal conduction pathway, abnormalities of the right or left atrium cannot be established reliably based on P-wave morphology. The PR interval is constant and may be the same or different from that of sinus rhythm. The QRS intervals are regular.

The presence of negative P waves in the inferior leads means that the ectopic focus is in the low part of the atrium; this has on occasion been termed a coronary sinus rhythm. The short PR interval indicates that the ectopic atrial focus is close to the AV node. The most likely mechanism for this rhythm is enhanced automaticity, in which the ectopic focus has accelerated activity and takes over the pacemaker function from the sinus node. It is also possible that the atrial rhythm is an escape rhythm, a result of a very slow sinus rate. Often, but not always, exercise will increase the sinus node rate and the sinus node can resume dominant pacemaker function because it is more susceptible to sympathetic regulation than the ectopic atrial focus. This is more likely to occur if the atrial rhythm is an escape rhythm. Atropine, which will increase the sinus node rate but not the rate of the ectopic atrial focus, may also be useful; however, this is only of short-term benefit.
A 56-year-old man with hypertension and paroxysmal atrial fibrillation who takes hydrochlorothiazide, digitalis, and Coumadin now presents with acute-onset intermittent palpitations. He has also been taking a significant amount of NSAIDs over the past 2 weeks after injuring his knee. His initial laboratory analysis reveals acutely elevated blood urea nitrogen and creatinine levels. An ECG is obtained.

What potential mechanisms are underlying this rhythm disturbance?

Which of these mechanisms is most likely in this patient?
Podrid’s Real-World ECGs

ECG 53 Analysis: Atrial tachycardia
The ECG shows a regular rhythm at a rate of 120 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QcT intervals are normal (310/440 msec). Although there are P waves (+) in front of each QRS complex, they are negative in leads II and aVF. Therefore, they are not originating from the sinus node but from a low atrial focus within the right atrium, as the P waves are still upright in leads I and V5-V6. As atrial activation is no longer via the normal conduction pathway, abnormalities of the right or left atrium cannot be established reliably based on P-wave morphology. All of the P waves have the same morphology, and there is a short PR interval (\( \| \) ) (0.12 sec). This is classified as a long RP tachycardia since the RP interval (\( \leftrightarrow \) ) is longer than half the RR interval (ie, the P wave is closer to the QRS complex that follows it than to the QRS complex that precedes it).

Etiologies for a long RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atypical AV nodal reentrant tachycardia (AVNRT) (ie, fast-slow) or, uncommonly, AV reentrant tachycardia (AVRT) associated with a preexcitation syndrome. The negative P waves exclude sinus tachycardia, and there is no second atrial waveform seen, excluding atrial flutter. The most common etiology for this would be an atrial tachycardia with 1:1 AV conduction. This could be confirmed by blocking the AV node (ie, via vagal maneuver or adenosine). The occurrence of AV block with a stable PP interval or atrial rate would confirm atrial tachycardia. If the arrhythmia abruptly terminated, a reentrant mechanism (AVNRT or AVRT) would be the etiology.

Tachycardias, including atrial tachycardia, have three possible mechanisms: enhanced automaticity, triggered activity, and reentry:

- With enhanced automaticity, the tachycardia is produced by an ectopic focus within the myocardium that has enhanced pacemaker function, which may be a result of catecholamines. The tachycardia often has a warm-up period in which the atrial rate gradually speeds up. There may also be a cool-down phase, or a gradual slowing of the rate, before the tachycardia terminates.

- The mechanism of triggered activity is delayed after-depolarizations of the myocardium that occur at the very end of the action potential (ie, these low-amplitude depolarizations are triggered by the preceding action potential). These delayed after-depolarizations are the result of calcium currents. If the magnitude of these calcium currents increases, such as with catecholamine stimulation, and the depolarizations generated are of sufficient amplitude, they may provoke another spontaneous action potential. This mechanism is most often seen in the setting of excessive digitalis levels enhanced by sympathetic stimulation and catecholamines. It may also be seen in dilated cardiomyopathy. This patient has acute renal insufficiency, likely the result of excessive NSAID use. Digoxin levels usually increase in the presence of renal insufficiency and hence the atrial tachycardia may be a digoxin-toxic arrhythmia.
• The third mechanism is reentry, which results from an electrical circuit (micro or macro) within the myocardium. The circuit consists of two distinct pathways with different electrophysiologic properties. The pathways, which are capable of antegrade and retrograde conduction, are linked proximally and distally through the normal myocardial tissue. Often these circuits are the result of fibrosis or scar in the myocardium (structural block); they may also be caused by changes in refractoriness of the tissue (functional block), such as with autonomic inputs. The reentrant tachycardia results from antegrade (unidirectional) block in one pathway (pathway 1) while the impulse conducts through the other pathway (pathway 2). If the impulse reaches the terminal portion of pathway 2 and is able to enter pathway 1 in a retrograde fashion, it is conducted back to the proximal portion of the circuit and then may reenter pathway 2 in an antegrade direction. If this process continues, a reentrant rhythm is established. These tachycardias typically have an acute onset without a warm-up period and acute termination without a cool-down period. Hence the ventricular rate abruptly increases and decreases.

The treatment for atrial tachycardia when there is a rapid ventricular rate associated with symptoms is AV nodal blocking agents (β-blockers, calcium-channel blockers, or digoxin). These agents will reduce the ventricular response rate by slowing or blocking conduction through the AV node. In addition, a β-blocker might actually suppress the ectopic atrial focus in the setting of digoxin toxicity, although often Digibind is used as therapy. Long-term therapy for atrial tachycardia generally requires the use of a class IA (quinidine, disopyramide), class IC (propafenone, flecainide), or class III (amiodarone, dronedarone, sotalol, dofetilide) antiarrhythmic agent. In some cases, ablation of the atrial focus may be used.
A long-term-care facility sends one of its residents urgently to the emergency department after receiving the following ECG report: “Patient complains of intermittent palpitations. No lightheadedness or dizziness. Stable vital signs. However, ECG with evidence of heart block. Send to EW for a pacemaker.” On physical examination, you note intermittent cannon A waves and are concerned about heart block.

Does this patient need a pacemaker?
Podrid’s Real-World ECGs

ECG 54 Analysis: Atrial tachycardia with AV block
The QRS complexes are occurring at a rate of 70 bpm, and they are slightly irregular. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I, II, aVF, and V4-V6). The QT/QTc intervals are normal (320/350 msec). Atrial activity (+) is easily seen in leads II, III, and aVF. The atrial rate is regular (└┘) at 210 bpm. Atrial activity can also be seen in leads V1-V3, although some P waves are within the ST segment (^) and not obvious. The P waves are inverted in leads II, aVF, and V1-V4. They are originating not from the sinus node but from an ectopic atrial focus. Hence there is an underlying atrial tachycardia. However, there is one QRS complex for every three P waves, and hence there is 3:1 AV block. An isoelectric baseline can be seen between each P wave (↑).

An atrial tachycardia is identified by the following:

- Distinct P waves of uniform morphology before each QRS complex. The mechanism is most often an ectopic focus that fires and stops, resulting in a distinct P wave. When two sequential P waves are seen, there is an isoelectric baseline between the P waves. The P wave is distinct even when the mechanism is a small reentrant circuit (micro-reentry) since only a small area is involved with the reentrant circuit that then stimulates the rest of the atrium.

- The atrial rate is typically between 100 and 220 bpm.

- P waves differ from those of sinus rhythm (ie, they are inverted or biphasic in any or all of leads I, II, aVF, and V4-V6).

- Although the PR intervals are usually constant, some variability of the PR intervals (‖) (and hence the QRS intervals) may also be seen as a result of antegrade concealed AV nodal conduction. At the rapid atrial rate, some of the atrial impulses completely penetrate the AV node, resulting in a QRS complex; some impulses do not penetrate the AV node (ie, they are blocked). Some impulses may penetrate the AV node, but they are blocked within this structure and so do not conduct through the entire node. However, such impulses, which are called concealed, partially depolarize the AV node and alter (and slow) the conduction of the subsequent atrial impulse through the node. This results in variability of the PR interval and hence the QRS intervals, as is seen in this patient.

- QRS intervals are regular (or may have a slight irregularity as discussed above) when there is a fixed degree of AV block (eg, 2:1, 3:1, 4:1) or regularly irregular if there is a variable degree of AV block present; Mobitz type I AV block (Wenckebach) may also be present.

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The Basics: Case 54

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This patient does not need a pacemaker for the atrial tachycardia with AV block. Rapid atrial rates (i.e., atrial flutter or atrial tachycardia) are often accompanied by varying degrees of AV block that are stable with adequate ventricular rates. This block is due to the intrinsic refractory period of the AV node. A ventricular pacemaker would only be indicated if the patient were having extremely slow ventricular rates or symptomatic bradycardia, due to a high degree of AV block or even complete heart block. The first line of therapy for this rhythm when associated with 1:1 conduction is often a β-blocker or a calcium-channel blocker (verapamil or diltiazem), which slows conduction through the AV node, creating AV block and slowing the ventricular response rate without affecting the atrial rate.

The cannon A waves are the result of the variable relationship between the P waves and QRS complexes, very similar to the situation seen with complete heart block. In addition, there is 3:1 AV block and hence several P waves do not result in a ventricular complex but instead result in an atrial contraction. Hence there is occasional right atrial contraction against a closed tricuspid valve, resulting in an increased amplitude of the A wave of the jugular pulsation.

Treatment for the atrial tachycardia requires the use of an antiarrhythmic drug that affects the atrial myocardium; these include the class IA (disopyramide or quinidine), class IC (flecainide or propafenone), and class III (amiodarone, dronedarone, sotalol, dofetilide) antiarrhythmic agents. Alternatively, electrophysiologic mapping of the atria can be performed to localize the ectopic focus (or reentrant circuit). If identified, radiofrequency ablation may be effective as therapy.
You are called in the middle of the night to see a hospitalized patient who has acutely developed an irregular rhythm. The patient is asymptomatic and sleeping. He is a 63-year-old man without a known cardiac history who was admitted a few days ago with hypoxemia as a result of exacerbation of severe chronic obstructive lung disease. His clinical status has been improving with antibiotics.

What is the rhythm on ECG?
What are your next steps in management?
Podrid’s Real-World ECGs

**ECG Analysis:** Wandering atrial pacemaker (multifocal atrial rhythm), left ventricular hypertrophy (LVH) with associated ST-T wave changes
The QRS intervals are irregular (\[\]), and there is no pattern present; hence the rhythm is irregularly irregular. The average heart rate is 84 bpm. The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/430 msec). The QRS (R-wave) amplitude is at least 30 mm in leads V4-V5 (\[ \]). This meets one of the criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The typical ST-T wave changes associated with LVH (\(^\uparrow\)) are also present.

Only three supraventricular rhythms are irregularly irregular: 1) sinus tachycardia in which there is one P-wave morphology and a stable PR interval, 2) multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) in which there are three or more different P-wave morphologies and PR intervals without a dominant P wave, and 3) atrial fibrillation in which there is no organized atrial activity or P wave. There is a P wave (\(+\)) before each QRS complex, but there is variability of the P-wave morphology as well as the PR intervals (\(\leftrightarrow\)); there are three or more different P-wave morphologies (and possibly as many as six). This is most apparent on the lead II rhythm strip. This is known as a wandering atrial pacemaker or multifocal atrial rhythm, which is identified by the following:

- Average heart rate < 100 bpm; when the rate is > 100 bpm, the rhythm is called a multifocal atrial tachycardia
- Distinct P wave before each QRS complex
- Presence of three or more different P-wave morphologies; inability to identify a stable or dominant P wave
- Variable PR intervals
- PP and QRS intervals that are irregularly irregular

Wandering atrial pacemaker represents multiple foci of atrial activity that compete with the sinus node for pacemaker function. As there are multiple foci in the right and left atrium, the P-wave morphology and PR intervals are variable. 

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Wandering atrial pacemaker can be a benign rhythm that occurs in normal individuals, often during sleep. It is a very common arrhythmia seen in association with pulmonary disease. It is, however, also associated with other conditions, including electrolyte disturbances, digitalis intoxication, or organic heart disease. Not uncommonly, wandering atrial pacemaker (multifocal atrial rhythm) or multifocal atrial tachycardia can degenerate into atrial fibrillation.

In this patient, the exacerbation of chronic obstructive lung disease is likely the underlying process. At this point it would be reasonable to confirm that the patient has normal electrolyte levels. Treatment of the underlying process is the mainstay of therapy. An echocardiogram to assess for structural heart disease is only necessary if an abnormality is found on cardiac examination or if the rhythm persists despite adequate treatment of the chronic obstructive lung disease.
A 72-year-old man with known chronic obstructive pulmonary disease and a 100-pack-year history of smoking presents to the emergency department with significant respiratory distress. His past medical history is only significant for hypertension. On physical examination, he has pronounced wheezes and profound use of accessory muscles for respiration. His cardiac examination reveals tachycardia with an irregular rhythm. An ECG is obtained.

What is the diagnosis?
How would you manage this ECG abnormality?
Podrid’s Real-World ECGs

ECG 56 Analysis: Multifocal atrial tachycardia, left anterior fascicular block (LAFB), old lateral wall myocardial infarction, clockwise rotation
The QRS complexes (RR intervals) are irregular (⏟), and there is no pattern present; hence the rhythm is irregularly irregular. The average heart rate is 126 bpm. The QRS complex duration is normal (0.08 sec), and the axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). This is a left anterior fascicular block (LAFB).

The QT/QTc intervals are slightly prolonged (320/460 msec). Only three supraventricular rhythms are irregularly irregular: 1) sinus tachycardia in which there is one P-wave morphology and a stable PR interval, 2) multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) in which there are three or more different P-wave morphologies and PR intervals without a dominant P wave, or 3) atrial fibrillation in which there is no organized atrial activity or P wave. There is variability of the P-wave (+) morphology (at least seven different P-wave morphologies) and PR intervals, most apparent in the lead II rhythm strip. This is known as a multifocal atrial tachycardia, which is identified by the following:

- Average heart rate > 100 bpm
- Distinct P wave seen before each QRS complex
- Presence of three or more different P-wave morphologies; inability to identify a stable and dominant P wave.
- Variability of the PR intervals
- Irregularly irregular PP and QRS intervals

continues
Q waves are noted in leads I and aVL (^), diagnostic for an old lateral wall myocardial infarction. Also noted is poor R-wave progression across the precordium (ie, the amplitude of the R waves does not progressively increase from leads V1 to V6). This is often the result of clockwise electrical rotation of the heart in the horizontal plane, which is determined by imagining the heart as viewed from under the diaphragm. In this situation the right ventricle is in front and the left ventricle is to the left. With clockwise rotation, the left ventricular forces are more laterally and posteriorly directed and occur later in the precordial leads (ie, poor R-wave progression and late transition). With counterclockwise electrical rotation, the left ventricular forces are more anteriorly directed and appear earlier in the precordial lead (ie, early transition with a tall R wave in lead V2). LAFB is sometimes associated with poor precordial R-wave progression. Another cause for the poor R-wave progression is right ventricular hypertrophy, suggested by the deep S waves in leads V5-V6. Poor R-wave progression may also be seen with severe lung disease.

Multifocal atrial tachycardia is often associated with pulmonary disease, particularly chronic obstructive pulmonary disease or acute pulmonary embolism, as well as cardiac conditions including heart failure, valvular heart disease, and coronary artery disease. Management focuses on treating the underlying precipitant, in this case the exacerbation of chronic obstructive lung disease. Caution should be used in administering β-agonists for chronic obstructive lung disease, as this can worsen the tachycardia. Treatment of multifocal atrial tachycardia initially involves slowing the ventricular rate; calcium-channel blockers (verapamil or diltiazem) or β-blockers are often used as well (although β-blockers may worsen the underlying lung disease if there is a significant asthmatic component). Therapy of the arrhythmia itself involves administration of antiarrhythmics, primarily the class IC (propafenone or flecaïnine) or class III (amiodarone, dronedarone, or sotalol) agents. There is some evidence that magnesium may be of some benefit. Not uncommonly, multifocal atrial tachycardia degenerates into atrial fibrillation.
A 56-year-old woman with known mitral regurgitation from mitral valve prolapse presents with acute-onset palpitations and lightheadedness.

What is the diagnosis?
What would be the best treatment?
Podrid’s Real-World ECGs

ECG 57 Analysis: Atrial flutter, left axis, clockwise rotation
The ECG shows a regular rhythm at a rate of 150 bpm. The QRS complex duration is normal (0.08 sec), and the axis is physiologically leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (240/380 msec). There is also poor R-wave progression from leads V1 to V4, with transition (R/S > 1) occurring at lead V5, characteristic of clockwise rotation of the heart in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. In this situation the right ventricle is in front and the left ventricle is to the left. With clockwise rotation, the left ventricular forces are more posteriorly directed and occur later in the precordial leads (ie, poor R-wave progression and late transition).

Although no distinct P waves can be seen, there is a continuous undulation of the baseline between each RR interval, particularly evident in leads II, III, and aVF. Two distinct atrial waveforms can be seen for each QRS complex in lead V1. These atrial waveforms (+) are occurring at a regular rate of 300 bpm. This identifies the underlying rhythm as atrial flutter with 2:1 AV block or 2:1 AV conduction. The only regular atrial arrhythmia that occurs at a rate of 260 bpm or higher is atrial flutter. Importantly, close examination of leads II, III, and aVF demonstrates that there is one atrial waveform before the QRS complex (↑) and a second flutter wave that can be seen at the end of the QRS complex (↑), giving the appearance of an S wave. The waveforms are negative-positive in the inferior leads, consistent with a typical atrial flutter.

Typical atrial flutter is identified by the following characteristics:

- Flutter waves (with negative-positive morphology in leads II, III, and aVF from a counterclockwise reentrant pathway in the right atrium) are uniform in morphology, amplitude, and interval.
- There is no isoelectric baseline between flutter waves (ie, the baseline between the waveforms is continuously undulating, giving the appearance of a saw-tooth pattern). This is due to the fact that atrial flutter results from a reentrant circuit located within the right atrium. There is then subsequent left atrial depolarization; therefore, there is continuous electrical activity. By contrast, atrial tachycardia is due to an ectopic focus (or micro-reentrant circuit) that fires and stops, in which there is an isoelectric baseline between each atrial waveform.
- The atrial rate is between 260 and 320 bpm. Flutter rate may be slower as a result of antiarrhythmic drugs or disease of the atrial myocardium; however, waveforms maintain typical flutter morphology.

*continues*
• The QRS intervals are regular when there is a stable pattern of AV conduction (eg, 2:1, 3:1, 4:1) or may be regularly irregular if there is a variable pattern of AV conduction, including second-degree Mobitz type I AV block (Wenckebach).

• There may be a variable relationship between the flutter wave and QRS complex due to antegrade concealed AV nodal conduction, similar to what may be seen with atrial tachycardia. As a result of the rapid atrial rate some of the atrial impulses completely penetrate the AV node, resulting in a QRS complex. Some impulses do not penetrate the AV node (they are blocked). Other impulses may penetrate the AV node, but instead of conducting through the entire AV node they are blocked within this structure. However, such impulses, which are called concealed, partially depolarize the AV node and alter (and slow) conduction of the subsequent atrial impulse through the node. This results in variability of the PR interval and hence the QRS intervals.

Atrial flutter is associated with multiple conditions, including mitral valve prolapse, rheumatic heart disease, sick sinus syndrome, left ventricular dysfunction (diastolic or systolic), pulmonary embolism, and pulmonary disease; it is also associated with cardiac surgery. The initial treatment for atrial flutter is rate control, which involves AV nodal blockade with a ß-blocker, calcium-channel blocker (verapamil or diltiazem), or digoxin. Subsequent reversion of the arrhythmia may be achieved with electric cardioversion or with an antiarrhythmic drug that affects atrial myocardium (ie, class IA, IC, or III antiarrhythmic agents). Long-term therapy to prevent recurrence is with antiarrhythmic agents or radiofrequency ablation.
An 85-year-old man presents with new-onset symptoms of heart failure. An echocardiogram demonstrates an ejection fraction of 30% with diffuse hypokinesis and no regional wall motion abnormalities; there is mild left ventricular hypertrophy (LVH). No valvular disease is seen. Coronary angiography reveals only mild, non-obstructive coronary artery disease. Evaluation for non-ischemic causes of his systolic dysfunction, including thyroid dysfunction, infiltrative diseases, autoimmune disorders, and hemochromatosis, has been unrevealing. He does report feelings of palpitations over the past 5 or 6 months. His past medical history is significant for hypertension, for which he takes atenolol; he has otherwise been healthy. The following ECG is obtained.

What is the diagnosis?
How would you manage this patient?
Podrid’s Real-World ECGs

ECG Analysis: Atrial flutter, LVH, nonspecific ST-T wave changes
The ECG shows a rhythm that is irregular, but with a pattern to the irregularity: The short RR intervals (└┘) are the same, and the long RR intervals (┌┐) are the same. Hence the rhythm is regularly irregular. The average heart rate is 130 bpm. During the longer RR intervals, typical atrial flutter waves (+) (that are negative-positive in the inferior leads) can be seen at an atrial rate of 280 bpm. The irregularity of the rhythm is due to various degrees of AV conduction block: 2:1 (└┘) and 4:1 (┌┐) (which accounts for the shorter and longer RR intervals). There is a variable relationship between the flutter wave and QRS complex (║) due to antegrade concealed AV nodal conduction, similar to what may be seen with atrial tachycardia.

With the rapid atrial rate some of the atrial impulses completely penetrate the AV node, resulting in a QRS complex. Some impulses do not penetrate the AV node (ie, they are blocked). Other impulses may penetrate the AV node, but they do not conduct through the entire AV node because they are blocked within this structure. However, such impulses, which are called concealed, partially depolarize the AV node and alter (and slow) the conduction of the subsequent atrial impulse through the node. This results in variability of the “PR” interval and hence the QRS interval. This can be observed between the third and fourth QRS complexes. This RR interval (──) is slightly longer than the other intervals, and there is a longer interval between the flutter wave and the QRS complex. Variability in the interval between the flutter wave and QRS complex is also seen between the fourth and fifth QRS complexes.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal, but left ventricular hypertrophy (LVH) is present, with an S-wave depth in lead V2 ( [ ) of 25 mm and an R-wave amplitude in lead V5 ( ] ) of 15 mm. This meets one of the criteria for LVH (ie, S-wave amplitude in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). There are nonspecific ST-T wave changes in leads I, aVL, and V4-V6 (^) that are secondary to LVH and represent chronic repolarization abnormalities, likely due to subendocardial ischemia. The QT/QTc intervals are normal (280/410 msec).
The initial treatment for heart failure includes diuresis, angiotensin-converting enzyme inhibitors, β-blockers, and maintenance of AV synchrony. This patient has a cardiomyopathy of unclear etiology, since coronary artery disease, valvular disease, and other non-ischemic causes have been ruled out. However, the presence of palpitations for many months, likely the result of the atrial flutter, is concerning for a tachycardia-mediated cardiomyopathy. Persistent atrial tachyarrhythmias have been associated with systolic dysfunction. This is commonly seen with atrial flutter, which is often at a persistent ventricular rate of 130 to 160 bpm (due to 2:1 AV block). With physical activity, 1:1 flutter may occur with ventricular rates of 260 to 320 bpm. It is often difficult to achieve adequate rate control with atrial flutter as rates will often increase dramatically with any sympathetic activation; the ventricular rates are related to the underlying atrial rate of 300 bpm (ie, 60, 75, 100, 150, and 300 bpm) depending on the degree of AV block.

In addition to the acute management of heart failure, management of atrial flutter in this patient includes rate control with an AV nodal blocking agent followed by restoration of sinus rhythm with direct current or chemical cardioversion. Since the atrial flutter was of several months duration, 3 to 4 weeks of adequate anticoagulation would be required prior to reversion to reduce the potential for an embolic event after reversion. Although this is less common with atrial flutter than atrial fibrillation, any small risk should be reduced. An alternative if earlier reversion is important, particularly if management of heart failure is difficult or if heart rate control is not adequately achieved, would be a transesophageal echocardiogram prior to reversion in order to assess for left atrial appendage thrombus, which is usually the site of thrombus formation and is associated with an increased risk for arterial embolism. Anticoagulation for at least a month thereafter would be indicated as there is a period of time during which atrial contraction remains depressed as a result of atrial stunning from the arrhythmia and only slowly returns to normal. The addition of an antiarrhythmic to prevent recurrence of atrial flutter would be reasonable. If this were unsuccessful, then radiofrequency catheter ablation of the reentrant circuit is an option.
This patient presented to the emergency department with a narrow complex rhythm at a rate of 150 bpm. After a diagnosis of supraventricular tachycardia was made, 6 mg of intravenous adenosine was administered. This ECG was obtained immediately thereafter.

What is the diagnosis?
ECG 59 Analysis: Atrial flutter, third-degree AV block, right bundle branch block
The ECG shows a fairly regular rhythm ( ■ ) at a rate of 32 bpm; however, the last RR interval is slightly shorter ( ▼ ). The atrial rate (+) is regular at 280 bpm. The slow ventricular rate is a result of a high degree of AV block (8:1). Given the high degree of AV block, it is possible that complete or third-degree AV block is present with an underlying escape rhythm. As a result, the atrial flutter waves (+) are well seen. The flutter waves are negative-positive inferiorly and uniform in morphology, amplitude, and interval; there is no isoelectric baseline between them ( ie , the baseline between the waveforms is continuously undulating, giving the appearance of a saw-tooth pattern). This is due to the fact that atrial flutter results from a reentrant circuit located within the right atrium; therefore, there is continuous electrical activity activating the right and then the left atrium. The interval between the flutter wave and QRS complex is variable ( □ ), which is a result of antegrade concealed AV nodal conduction. With the rapid atrial rate some of the atrial impulses completely penetrate the AV node, resulting in a QRS complex. Some impulses do not penetrate the AV node ( ie , they are blocked). Other impulses may penetrate the AV node, but they do not conduct through the entire AV node because they blocked within this structure. However, such impulses, which are called concealed, partially depolarize the AV node and alter (and slow) the conduction of the subsequent atrial impulse through the node. This probably also accounts for the slight difference in the last RR interval.

In addition, the QRS complexes are wide (0.16 sec) and there is an RSR’ morphology in lead V1 ( ← ) and a broad S wave ( ↑ ) in lead V6. This is a pattern of a typical right bundle branch block, which results in late forces to the right ventricle directed in a left-to-right direction, accounting for the R’ in lead V1 and the small but broad terminal S wave in lead V6. The axis is physiologically leftward (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are prolonged (680/500 msec) but are normal when accounting for the prolonged QRS complex (600/440 msec).

Adenosine can be administered safely in the setting of any narrow complex tachycardia to terminate the arrhythmia or help identify the underlying rhythm by blocking the AV node and exposing atrial waveforms. When the complex is narrow, the impulse is conducted normally through the AV node and His-Purkinje system to activate the ventricle. Adenosine, which blocks AV nodal conduction, is safe in this situation since the AV node is involved with the arrhythmia. Reentrant rhythms in which the AV node is a necessary part of the circuit, such as AV nodal reentrant tachycardia and AV reentrant tachycardia, will be converted to a normal rhythm or will not be altered. There will be transient slowing of the ventricular rate of other supraventricular tachycardias originating in the atrial myocardium and using the AV node for conduction to the ventricles, such as atrial flutter, atrial tachycardia, and atrial fibrillation. This is a result of transient AV nodal block. Although this does not affect the underlying arrhythmia, the transient slowing may be useful for exposing the P waves (or atrial waveforms), determining the atrial rate, and hence establishing the etiology of the atrial arrhythmia.
This ECG was obtained from a 52-year-old man with diabetes, hypertension, and ischemic cardiomyopathy with an ejection fraction of 40%. He has no history of stroke or thromboembolic disease. He has normal renal function. A transthoracic echocardiogram reveals trace mitral regurgitation without evidence of mitral stenosis.

What is the diagnosis?

How would you assess his future risk for thromboembolic stroke?
Podrid’s Real-World ECGs

ECG Analysis: Atrial fibrillation, nonspecific ST-T wave changes
The rhythm is irregularly irregular (†), with an average rate of 78 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/410 msec). Diffuse nonspecific ST-T wave changes are present, especially in leads V4-V6 (↑). There are no organized P waves. However, prominent atrial waveforms (↑) are noted in leads V1-V2. Although these waveforms look similar to atrial flutter, they are not uniform and vary in morphology, amplitude, and interval. As a reentrant arrhythmia within the right atrium, flutter waves are completely uniform in morphology, amplitude, and interval. Thus this is coarse atrial fibrillation, which generally implies a more recent onset. In contrast to atrial flutter (in which the rhythm is either regular or regularly irregular), the ventricular rate in atrial fibrillation is irregularly irregular.

The features of atrial fibrillation include the following:

• The atrial rate ranges from 320 to 450 bpm or even faster.
• There is no organized atrial activity or distinct P wave; fibrillatory waves are present.
• Fibrillatory waves are irregular in morphology, amplitude, and interval. They may be coarse (resembling atrial flutter waveforms) when atrial fibrillation is recent in onset or fine when atrial fibrillation is of longer duration. When atrial fibrillation is present longer, there is electrical and structural remodeling with dilation of the left atrium and the development of fibrosis, which results in finer fibrillatory waveforms.
• QRS intervals are irregularly irregular due to the irregular atrial rate and hence the irregularity of impulse conduction through the AV node. The ventricular rate depends entirely on AV nodal conduction. It is generally up to 170 bpm when the AV nodal conduction properties are normal, as this is the maximum rate that the normal AV node will conduct in the absence of a sympathetic stimulus. When ventricular rates exceed 200 bpm, sympathetic activation is likely enhancing AV nodal conduction. Hence conditions that are associated with elevated sympathetic tone should be considered as the cause for the atrial fibrillation. When ventricular rates in atrial fibrillation are less than 100 bpm, increased vagal tone, intrinsic AV nodal disease or the presence of AV nodal blocking agents (digoxin, β-blockers, or calcium-channel blockers such as verapamil or diltiazem) should be considered.

continues
Patients with atrial fibrillation are at risk for thromboembolic stroke due to clot formation in the noncontractile or hypocontractile left atrial appendage. The atrial fibrillation in this patient is not the result of a valvular abnormality, particularly mitral stenosis or significant mitral regurgitation. Nonvalvular atrial fibrillation carries a 4.5% risk per year for thromboembolic stroke, whereas the risk is much higher with valvular atrial fibrillation. The CHADS2 scoring system can be used to quantify stroke risk in nonvalvular atrial fibrillation, with 1 point each for heart failure, hypertension, age over 75, and diabetes and 2 points for prior stroke or other embolic event. A score of 2 or more requires warfarin for anticoagulation. Treatment with aspirin is sufficient for a score of 0. A score of 1 is intermediary and can be treated with either aspirin or warfarin. This patient’s CHADS2 score is 3, placing him at high risk for thromboembolic stroke.
A 46-year-old man with a history of coronary artery disease and a small inferior wall myocardial infarction who had a stent placed in the right coronary artery 2 years ago has the following ECG in the setting of palpitations. The patient has no history of hypertension, diabetes, stroke, or thromboembolic disease and has normal renal function.

What is the diagnosis?
If this arrhythmia were paroxysmal, which of the following would be the most appropriate suppressive antiarrhythmic therapy for this patient?
A. Amiodarone  B. Flecaïnide  C. Propafenone  D. Sotalol
Would anticoagulation with Coumadin be necessary if the rhythm were chronic?
Podrid’s Real-World ECGs

ECG 61 Analysis: Atrial fibrillation
The ECG shows a rhythm that is irregularly irregular (чин), with an average heart rate of 114 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal QRS morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are 320/420 msec. Although there is no evidence of organized atrial activity, irregular atrial waveforms (^) can be seen in leads II, III, aVF, and V1; these waveforms are not uniform in morphology, amplitude, or interval, and they are not present in front of each QRS complex. There is no obvious atrial activity in other leads. Hence this is atrial fibrillation with fine fibrillatory waves, implying that the atrial fibrillation has been present for a long period of time.

The features of atrial fibrillation include the following:

- The atrial rate ranges from 320 to 450 bpm or even faster.
- There is no organized atrial activity or distinct P wave; fibrillatory waves are present.
- Fibrillatory waves are irregular in morphology, amplitude, and interval. They may be coarse (resembling atrial flutter waveforms) when atrial fibrillation is recent in onset or fine when atrial fibrillation is of longer duration. When atrial fibrillation is present longer, there is electrical and structural remodeling with dilation of the left atrium and the development of fibrosis, which results in finer fibrillatory waveforms.
- The QRS complex intervals are irregularly irregular due to the irregular atrial rate and hence the irregularity of impulse conduction through the AV node. The ventricular rate depends entirely on AV nodal conduction. It is generally up to 170 bpm when the AV nodal conduction properties are normal, as this is the maximum rate that the normal AV node will conduct in the absence of a sympathetic stimulus. When ventricular rates exceed 200 bpm, sympathetic activation is likely enhancing AV nodal conduction. Hence conditions that are associated with elevated sympathetic tone should be considered as the cause for the atrial fibrillation. When ventricular rates in atrial fibrillation are less than 100 bpm, increased vagal tone, intrinsic AV nodal disease, or the presence of AV nodal blocking agents (digoxin, β-blockers, or calcium-channel blockers such as verapamil or diltiazem) should be considered.

continues
Long-term antiarrhythmic therapy for prevention of atrial fibrillation typically consists of class IA agents such as quinidine and disopyramide, class IC agents such as propafenone or flecainide, and class III agents such as amiodarone, sotalol, or dofetilide. The class III agent ibutilide is only available for intravenous administration and hence it is used for acute termination of the arrhythmia. However, the class I agents in general and the class IC agents specifically are felt to be contraindicated in patients with structural heart disease, which includes a previous myocardial infarction and myocardial scar, cardiomyopathy, or left ventricular hypertrophy. The class IA agents are infrequently used because of the side effects associated with these drugs. Amiodarone is the best agent available to maintain sinus rhythm; however, long-term therapy is associated with side effects, some of which are serious (eg, pulmonary fibrosis, thyroid and hepatic abnormalities). It would not be a preferred first-line agent in this relatively young patient.

Sotalol would be the most ideal antiarrhythmic for this patient because it tends to be well tolerated and is only contraindicated in patients with significant renal insufficiency (the drug is excreted renally) or a prolonged QT interval.

As discussed previously, the need for long-term anticoagulation therapy for chronic or permanent atrial fibrillation when there is no attempt made to restore sinus rhythm is based on the CHADS2 scoring system. This patient, who is young and does not have heart failure, diabetes, hypertension, or a prior embolic event, is at low risk for an embolism (ie, CHADS2 score = 0). Hence anticoagulation with Coumadin would not be necessary. However, aspirin is often prescribed for such patients. This patient, however, is likely already taking aspirin as a result of coronary disease and a previous stent placement.
A 57-year-old woman with a history of paroxysmal atrial fibrillation currently on digoxin presents to the urgent care clinic with feelings of extra heartbeats.

What is the diagnosis?
Is any therapy needed?
Podrid’s Real-World ECGs

ECG 62 Analysis: Normal sinus rhythm, premature junctional complexes in a bigeminal pattern
The ECG shows a regularly irregular rhythm, with intermittent long (\(\uparrow\)) and short (\(\downarrow\)) RR intervals. The average heart rate is 72 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is physiologically leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (400/440 msec). There is a P wave (+) before the QRS complex after the long RR interval, and the P wave is positive in leads I, II, aVF, and V4-V6; hence this is a sinus P wave. After each sinus complex there is a premature QRS complex (^), accounting for the short RR interval. Thus the rhythm has a bigeminal pattern.

The early or premature QRS complexes (^) are not preceded by a P wave (i.e., atrial activation); hence they are not originating from the sinus node or atrial myocardium. However, they are narrow (and hence supraventricular because they are being conducted via the normal His-Purkinje system) and have a morphology that is similar to the sinus complexes, although in some leads their amplitude (\(\downarrow\)) is different than that of the sinus complexes. These are premature junctional complexes occurring in a bigeminal pattern (i.e., junctional bigeminy) and can often be seen in the setting of digoxin therapy, although they can also be seen in normal subjects. The premature junctional complexes have a slight negative notching at the very end of the QRS complex, seen best in the limb leads. This is a retrograde P wave (↑), especially obvious in lead II. However, not all premature junctional complexes will have visible retrograde P waves.

The difference in amplitude reflects the fact that impulses originating in the AV junction, a result of an ectopic focus in this structure, enter the bundle of His (which is composed of a series of conduction pathways or tracks) at a different location compared with impulses that originate in the atrium and are conducted through the AV node. Hence junctional or AV nodal beats are conducted through different tracts within the His-Purkinje system compared with impulses from the atrium conducted through the AV node. Thus they often have an amplitude and/or axis that is different than that seen with the sinus (or atrial) complexes. 

continues
Premature junctional complexes have the same clinical implications as premature atrial complexes. Hence they do not require therapy unless they are very symptomatic or are associated with other sustained arrhythmia. The first approach in this patient would be to discontinue the digoxin, as it is not indicated for therapy of paroxysmal atrial fibrillation. Digoxin has antiarrhythmic activity only by increasing vagal tone and does not revert or prevent atrial fibrillation, with the possible exception of atrial fibrillation associated with overt heart failure. In this patient, improvement in left ventricular function and improvement in heart failure may result in atrial fibrillation reversion. However, the major indication for digoxin is to slow conduction through the AV node and hence slow the ventricular response rate during sustained atrial fibrillation. If the premature complexes should continue and are associated with symptoms, a β-blocker might reduce the sensation associated with this arrhythmia. The mechanism for the symptom of palpitations is generally the increased inotropy and stroke volume associated with the post-extrasystolic beat (via the Starling mechanism). By reducing the inotropy of this beat, β-blockers often alleviate the symptoms, although they do not usually suppress the arrhythmia.
A 74-year-old woman with no significant past medical history develops dyspnea and lightheadedness only with exertion. She is on no medications. Echocardiography reveals a structurally normal heart, and cardiac computed tomography shows no evidence of coronary artery disease. During exercise, the following ECG is obtained while the patient is having symptoms.

What is the diagnosis?
How would you manage this patient?
Podrid’s Real-World ECGs

ECG 03 Analysis: Junctional rhythm, retrograde atrial activity
The ECG shows a stable rhythm at a rate of 72 bpm. The QRS complexes are narrow (0.08 sec) and have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/350 msec). The QRS complexes are supraventricular, conducted through the normal His-Purkinje system. There are no P waves before any of the QRS complexes; therefore, the QRS complexes are not the result of initial sinus or atrial activation. The complexes originate in the AV node or junction, and hence this is a junctional (or AV nodal) rhythm. There is in fact retrograde atrial activity (ie, an inverted P wave [+] after the QRS complexes), best seen in leads II, III, and aVF. Although these P waves might be confused for T waves, T waves are generally broader and have a longer duration. Moreover, in leads V5-V6 the retrograde P wave can be seen following the T wave (^). It is common for ectopic junctional rhythms to have retrograde P waves as a result of ventriculoatrial (VA) conduction from the junction back to the atrium. This atrial activation occurs from the bottom of the atrium toward the top; hence the P waves are inverted in the inferior leads, especially lead aVF, which is at 90° and hence is a vertical lead. The RP interval is usually constant, reflecting stable VA conduction time. In this case, the RP interval (^→) is fairly prolonged (0.28 sec), reflecting slow VA conduction.

A junctional rhythm may be an escape rhythm, as a result of complete heart block, or it may be accelerated, with a rate faster than the sinus rate. In this case the junctional rhythm is not an escape rhythm. It is due to an “accelerated” ectopic junctional focus that is faster than the sinus rhythm. As a result the retrograde P wave (retrograde atrial activation) suppresses sinus node activity so that it is not seen on the ECG. Junctional rhythms are often well tolerated and associated with sick sinus syndrome, digoxin toxicity, administration of nodal blocking agents, and cardiac surgery. In this case, the patient is asymptomatic at rest. With exercise, her junctional rhythm occurs at a heart rate of 72 bpm, suggesting that the junction has responded to the increase in catecholamine levels with exercise, but the sinus node has not responded appropriately, as its rate must be less than 72 bpm so that the junctional focus assumes the dominant pacemaker function. This is a marker for sinus node dysfunction, manifest as chronotropic incompetence of sinus node activity. Without any reversible causes of her sinus node dysfunction and given her exertional symptoms, which are likely from an inappropriately slow heart rate with exercise, a rate-responsive permanent pacemaker would be useful.
A 45-year-old man has a medical history significant for asthma, for which he uses inhalers, and supraventricular tachycardia, for which he was prescribed digoxin. He now presents with palpitations, which he feels occurred after excessive use of his inhaler for an exacerbation of asthma. His physical examination is normal, without cardiac murmur or evidence of volume overload. The following ECG is obtained.

What is the diagnosis?
Does this patient need cardiac catheterization urgently?
Podrid’s Real-World ECGs

ECG 64 Analysis: Ectopic junctional tachycardia
The ECG shows a regular rhythm at a rate of 120 bpm. The QRS complexes are narrow (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF); therefore, the complexes are supraventricular (i.e., conduction to the ventricle is via the normal His-Purkinje system). The QT/QTC intervals are normal (300/420 msec). There are no P waves before any of the QRS complexes. Therefore, the impulse is not originating in the atrium or sinus node but rather is being generated by the AV node or junction. This is a junctional rhythm. Because the rate exceeds 100 bpm, it is termed an ectopic junctional tachycardia. Retrograde (negative) P waves (+) are seen after the QRS complexes in most leads. These retrograde P waves should not be confused with abnormal ST segments. Indeed, the ST segments are normal. The RP interval (||) is short and constant, reflecting stable ventriculoatrial conduction. This rhythm is termed a short RP tachycardia. Therefore, there is no evidence of coronary artery disease or ischemia and hence catheterization is not indicated.

As the patient has a history of supraventricular tachycardia treated with digoxin, it is likely that the cause of his tachycardia is an ectopic junctional rhythm. This is supported by the fact that the rate is 120 bpm and there is a distinct retrograde P wave with a short RP interval (short RP tachycardia), features typical for an ectopic junctional tachycardia. However, an ectopic junctional tachycardia is the result of an ectopic focus that does not respond to digoxin (digoxin works electrophysiologically by increasing vagal tone). Hence digoxin is effective for reentrant arrhythmias involving the AV node and not for arrhythmias that are due to an ectopic focus. As the mechanism is enhanced automaticity, cardioversion is not effective and indeed should not be performed if the etiology is digoxin toxicity. Initial therapy involves discontinuing agents that may be responsible, such as sympathomimetic drugs. If the arrhythmia persists, therapy with a β-blocker or a calcium-channel blocker might be effective.
A 22-year-old college student presents to the university health services with an episode of sustained palpitations and lightheadedness. This episode occurred when he was playing basketball, but he reports several previous episodes that occurred both during exercise and at rest. Earlier
episodes were self-limiting, lasting about 1 hour. However, this episode had already lasted 2 hours when he decided to seek medical attention. The following two are ECGs obtained, one during the symptomatic episode (65A) and one after the symptoms resolved (65B).

What is the diagnosis?
What immediate treatment would be useful?
Podrid’s Real-World ECGs

ECG 65A Analysis: AV nodal reentrant tachycardia (AVNRT)
ECG 65A shows a regular rhythm at a rate of 160 bpm; hence this is a tachycardia. No obvious P waves are seen either before or after the QRS complexes. There is, however, a small waveform at the end of the QRS complex, especially seen in leads V1-V2 (↓). Although not an obvious P wave, it is suggestive of atrial activity.

The QRS complexes are narrow (0.08 sec) and have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). Hence the QRS complexes are supraventricular in origin. The QT/QTc intervals are normal (240/390 msec). Given the absence of any obvious P waves, this rhythm may be referred to as a no-RP tachycardia. The rhythm is a junctional tachycardia, but in contrast to an ectopic junctional tachycardia in which there are usually retrograde P waves, the absence of atrial activity before or after the QRS complex is characteristic of an AV nodal reentrant tachycardia (AVNRT), which is a type of junctional tachycardia in which the mechanism for the arrhythmia is reentry within the AV node.

By comparison, ECG 65B (see next page) shows normal sinus rhythm. There is a regular rhythm at a rate of 78 bpm. P waves (+) can now be seen in front of each QRS complex, with a stable PR interval (0.16 sec). The QRS complexes are identical in duration and axis to those during the AVNRT (ECG 65A). The QT/QTc intervals are normal (320/360 msec). However, there is a subtle difference in the QRS morphology in leads V1-V2. During the AVNRT (ECG 65A), a small R’ (↓) is noted at the very end of the QRS complex. This R’ is not seen during sinus rhythm (▼) on ECG 65B. This waveform is the retrograde P wave, which occurs almost simultaneously with ventricular activation. Hence AVNRT presents without any distinct P wave seen before or after the QRS complex, or the P wave is superimposed at the end of the QRS complex, as can be seen in this case. Only by comparison with an ECG of sinus rhythm is it obvious that this represents a change in the QRS morphology and hence is likely the retrograde P wave.

continues
Podrid’s Real-World ECGs

ECG 65B Analysis: Normal sinus rhythm, normal ECG
AVNRT generally results from dual pathways within the AV node. There is a fast pathway that conducts the impulse rapidly but has a long refractory period (i.e., recovers slowly) and a slow pathway that conducts the impulse slowly but has a short refractory period (i.e., recovers more quickly). These two pathways are proximally linked in the upper portion of the AV junction (within the atrial tissue) and distally linked at the lower portion of the AV junction (within the bundle of His), forming a circuit. During sinus rhythm the fast pathway predominates and the impulse reaches the His-Purkinje system and hence ventricular myocardium via this pathway. However, if a premature atrial complex reaches the AV node when the fast pathway has not fully recovered, it may be blocked in the fast pathway (unidirectional block) and conducted to the ventricle via the slow pathway, which recovers more quickly. Therefore, the premature atrial complex has a long PR interval. Since conduction through the slow pathway is slow, the impulse may reach the distal portion of the circuit at a time when the fast pathway has recovered, and hence the impulse will enter the fast pathway retrogradely to activate the atrium at the same time there is antegrade conduction to the ventricles. Hence the P wave and QRS complex occur simultaneously (or nearly simultaneously). If the retrograde impulse conducted via the fast pathway reaches the proximal portion of the circuit at a time when the slow pathway has recovered, the impulse also reenters the slow pathway, setting up a reentrant arrhythmia. This is the typical or common form of AVNRT and is called slow-fast (slow to the ventricle and fast back to the atrium). An uncommon (atypical) form of AVNRT in which the circuit is fast-slow (i.e., fast to the ventricle and slow to the atrium) presents as a long RP tachycardia.

Immediate treatment for an AVNRT involves altering the electrophysiologic characteristics of the AV nodal pathways. The quickest way to do this is through a maneuver that enhances vagal tone (and hence slows conduction and prolongs refractoriness within the AV node), such as a carotid sinus pressure or Valsalva maneuver. Another effective therapy is adenosine, which transiently slows or blocks conduction through the AV node. Alternative therapies that can be used are other AV nodal blocking agents, such as intravenous verapamil or diltiazem, a β-blocker, or even digoxin. If these therapies are ineffective, electric cardioversion is usually effective.

Long-term therapy involves oral drugs that affect AV nodal conduction, such as digoxin, verapamil, diltiazem, or a β-blocker. If the AV nodal blocking agents are ineffective, class IA, IC, and III antiarrhythmic drugs can be useful. However, AV nodal ablation is preferred, especially in younger patients, to avoid the toxicity associated with these agents. n
A 50-year-old lawyer with a history of hypertension who is on a thiazide diuretic presents to the emergency department with acute-onset palpitations and dyspnea. He admits to drinking much more alcohol and caffeine recently due to long business meetings and other stressors at work. He also mentions that he has had several
similar episodes that always require a visit to the emergency department, where he received some unknown intravenous medication. ECG 66A is obtained while the patient is experiencing palpitations. ECG 66B is the patient’s baseline ECG, obtained when he is not symptomatic.

What is the diagnosis?
How would you manage the patient long-term?
Podrid’s Real-World ECGs

ECG Analysis: AV nodal reentrant tachycardia
(AVNRT), left axis, nonspecific ST-T wave abnormalities
ECG 66A shows a regular narrow QRS complex (duration = 0.08 sec) tachycardia at a rate of 160 bpm. The QRS morphology is normal, and the axis is physiologically leftward, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (260/420 msec). There are nonspecific ST-T wave abnormalities in leads I, aVL, and V3-V6 (^). This is a supraventricular tachycardia. There are no obvious P waves before or after any of the QRS complexes; no R’ is seen in lead V1. Therefore, the origin of the tachycardia is the AV node or junction. The absence of P waves (a no-RP tachycardia) is characteristic of an AV nodal reentrant tachycardia (AVNRT), which is the most common regular narrow complex (supraventricular tachycardia) to present without any obvious P waves.

continues
ECG 66B Analysis: Normal sinus rhythm, nonspecific ST-T wave abnormalities
In ECG 66B, the patient’s baseline ECG, the QRS complex duration, morphology, and axis and the QT/QTc intervals are identical to those seen during the AVNRT in ECG 66A. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). Hence another typical way for AVNRT to present is with QRS complexes that are identical to those seen in sinus rhythm without a P wave superimposed on the terminal portion of the QRS complex.

Common precipitants of AVNRT include alcohol, stimulants, exercise, and nicotine. Acute termination of the reentrant rhythm can be achieved with vagal maneuvers such as carotid sinus pressure or Valsalva. Increased vagal tone slows conduction through the AV node and by altering the electrophysiologic properties can terminate the reentrant arrhythmia. Another effective approach is the administration of intravenous adenosine, which also slows and blocks conduction through the AV node. β-blockers and calcium-channel blockers such as verapamil or diltiazem can be used for immediate termination. If the patient is unstable or if pharmacologic therapy is ineffective, direct current cardioversion is recommended. If this is unsuccessful, anti-arrhythmic therapy with class IA, IC, or III agents is an option. For episodic arrhythmia, a single large dose of an antiarrhythmic drug previously established as being effective for arrhythmia termination may be used for the episode. This approach, termed the “pill in the pocket approach” or “cocktail drug therapy” is used to treat an arrhythmia event when it occurs and avoids the use of long-term drug therapy. Long-term therapy to prevent AVNRT often begins with an AV nodal blocking agent (verapamil, diltiazem, β-blocker, or digoxin given alone or in combination). If these therapies are ineffective, a class IA, IC, or III antiarrhythmic agent can be used. However, given the potential for side effects with antiarrhythmic therapy, catheter ablation of the slow pathway is often used to manage AVNRT, with a success rate of approximately 90%.
An asymptomatic 52-year-old woman whom you see in clinic has the following baseline ECG.

How would you characterize the abnormality?
What is the most likely mechanism for this finding?
How would you manage this patient?
ECG 67 Analysis: Sinus bradycardia, premature ventricular complexes (PVCs) (unifocal)
There is a regularly irregular rhythm at a rate of 56 bpm. The irregularity is due to two wide (duration = 0.16 sec) premature QRS complexes (^) (second and eighth complexes) that are followed by a pause. The PP interval (▼) around the premature complex is equal to two PP intervals (▲) and hence is termed a compensatory pause. All other QRS complexes are narrow (0.08 sec) and occur at regular intervals. There is a P wave (+) before each narrow QRS complex, with a stable PR interval (0.30 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence there is a sinus bradycardia with a first-degree AV block.

The narrow QRS complexes have a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/370 msec). These complexes are the result of normal conduction through the AV node and His-Purkinje system. The premature complexes are wide with an abnormal morphology and do not have a P wave before them. They are called premature ventricular complexes (PVCs), premature ventricular beats, or ventricular extrasystoles. They may also be called ventricular premature beats or ventricular premature complexes. Since all the PVCs have the same morphology, they are termed unifocal PVCs.

Characteristics of PVCs include the following:

- An early (premature) and wide QRS complex (≥ 0.12 sec) that is without a preceding P wave. A P wave after the QRS complex may be seen. This P wave may be retrograde due to ventriculoatrial conduction or may be an on-time sinus P wave. This can be determined by comparing the interval between the P waves of the sinus complexes prior to the PVC and the P wave following the PVC with the underlying PP interval. If the P wave after the PVC has the same timing as the sinus P waves, it is the on-time sinus P wave. If the P wave after the PVC is early, it is related to the PVC and hence is retrograde.

- Usually the PVC is associated with a full compensatory pause (ie, the PP interval surrounding the PVC is twice the baseline PP interval). This is due to retrograde (ventriculoatrial) conduction through the AV node. The PVC causes retrograde activation of the His-Purkinje network and penetrates the AV node in a retrograde fashion. The next on-time sinus impulse finds the AV node refractory and hence there is no AV conduction and the sinus P wave (which is usually not apparent on the ECG as it usually occurs during the PVC) is blocked or nonconducted. The subsequent sinus P wave then conducts through the AV node in normal fashion.

continues
• On occasion the PVC is interpolated (ie, there is no pause after the PVC and the PP interval surrounding the PVC is the same as the baseline PP interval). In this situation, the PR interval of the complex following the PVC is often longer than the baseline PR interval. This is the result of concealed retrograde conduction, which is due to the fact that the retrograde conduction through the AV node is only partial. Thus, the AV node is only partially refractory; the subsequent atrial impulse can penetrate the AV node, but it is conducted through the node at a slower rate (longer PR interval) due to the fact that the partially refractory AV node will conduct more slowly.

The PP interval surrounding the PVC (\( \text{\textcircled{\textbullet}} \)) is twice the underlying PP interval (\( \text{\textcircled{\textuparrow}} \)) or sinus rate; hence there is a full compensatory pause.

The most common mechanism for PVCs is reentry. Therefore, unifocal PVCs usually have a fixed coupling relationship (\( \leftrightarrow \)) with the preceding sinus beat (ie, with each premature beat the interval between the sinus QRS complex and PVC is the same). PVCs occur frequently both in healthy individuals and in those with heart disease. When they are present in a patient with heart disease they have been associated with an increased risk for morbidity and mortality (due to sudden cardiac death), particularly if they occur frequently or are repetitive (ie, two or more sequential PVCs). For this patient, it would be reasonable to obtain an echocardiogram to rule out structural heart disease. Even in the absence of any abnormality, a Holter monitor might also be used to assess the frequency of PVCs and the presence of repetitive forms.

Treatment of unifocal PVCs is usually not needed if there are no symptoms. Therapy is not warranted even for patients with heart disease who may be at an increase risk as there are no data that suppression of PVCs will prevent a sustained ventricular arrhythmia or sudden cardiac death. However, symptoms associated with PVCs, including palpitations, shortness of breath, or even dizziness, may prompt therapy for their suppression. Symptoms of palpitations are often the result of the post-PVC sinus beat, which has increased left ventricular diastolic filling due to the post-PVC pause and an increase in inotropy via the Starling effect resulting in an increased stroke volume. β-blockers may help to alleviate the symptom of palpitations. For more serious symptoms, suppressive therapy with a class IA, IC, or III agent may be needed.

Therapy to suppress asymptomatic PVCs may be necessary in patients with significant left ventricular systolic dysfunction if the PVCs are very frequent or repetitive and impact left ventricular function, causing a worsening of heart failure. The PVCs may result in a dysynchronous and ineffective contraction, and there may be failure to augment stroke volume with the post-extrasystolic beat. In a patient with underlying sinus bradycardia, frequent PVCs result in a further slowing of the effective heart rate. As PVCs have a suboptimal stroke volume, the effective cardiac output is reduced and this may be associated with development of symptoms related to a bradycardia.
A 71-year-old man with known coronary disease, hypertension, and diabetes presents with frequent episodes of an irregular heartbeat. He reports transient sensations of a racing heartbeat followed very quickly by normal heartbeats. An ECG is obtained.

What is the diagnosis?
Anatomically, where would you localize the two abnormalities?
**ECG 68 Analysis:** Sinus bradycardia, intraventricular conduction delay (IVCD), chronic inferior wall myocardial infarction (MI), premature ventricular complexes (PVCs) (multifocal, interpolated)
The ECG shows a regularly irregular rhythm at a rate of 48 bpm. The irregularity is the result of two premature complexes (^) (third and eighth QRS complexes) that are wide (0.16 sec) and have an abnormal morphology. Each of the narrower QRS complexes (duration = 0.12 sec) have a P wave (+) before them with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a sinus bradycardia.

There is no specific pattern to the widening of the sinus QRS complexes (ie, it is not a right or left bundle branch block), and hence this is an intraventricular conduction delay (IVCD). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There are Q waves (↑) in leads II, III, and aVF associated with T-wave inversion (*); this is characteristic of a chronic inferior wall myocardial infarction (MI). The QT/QTc intervals are normal (440/390 msec and 400/360 msec when corrected for the prolonged QRS interval).

The two premature and wide QRS complexes do not have a preceding P wave, and they have an abnormal morphology. These are premature ventricular complexes (PVCs) with different morphologies; hence they are multifocal PVCs. The coupling intervals (↔) between the sinus beat and the PVCs are different (0.44 and 0.70 sec), meaning that there are two distinct locations for the reentrant circuits that are responsible for the two different PVC morphologies. Neither of the PVCs is associated with a pause, and indeed the PP interval surrounding the PVC (└┘) is the same as the sinus PP interval (╔╗). Hence these PVCs are interpolated. However, the PR interval of the on-time sinus beat following the PVC (┌┐) is slightly longer (0.24 sec) than the baseline PR interval (╠╣) (0.20 sec). This is the result of concealed retrograde (ventriculoatrial) conduction; the premature ventricular impulse partially penetrates the AV node in a retrograde direction but does not completely depolarize this structure. Hence the impulse is concealed within the AV node. However, the AV node becomes partially depolarized and is partially refractory. Therefore, the next on-time sinus impulse conducts through the AV node but with a slower conduction velocity (due to slower AV nodal conductivity), accounting for the longer PR interval.

continues
The site of origin of a PVC within the myocardium can be established based on the ECG. This patient has a known inferior infarction that could very easily be the source of a scar-related PVC. In general, the leads in which the PVC is negative identify the site of origin of the PVC (i.e., the direction of impulse activation is away from the site of origin and hence the impulse is directed away from the lead that is located over the site of origin, thereby producing a Q wave in this lead). The first PVC on this ECG is negative in leads II and aVF, suggesting that it is originating from the inferior wall, activating the ventricle in a direction away from the inferior wall. Since the complex has a right bundle branch–like morphology in lead V1, it is originating in the left ventricle. Thus, this PVC originates from the inferior wall of the left ventricle, correlating with the site of the old infarction. The second PVC, however, has a left bundle branch–like pattern in lead V1 (QS morphology), suggesting that it is originating in the right ventricle. The PVC is positive in lead II and therefore appears to be inferiorly directed. Thus, the second PVC may be coming from the right ventricular outflow tract, a common source of non–infarct-related PVCs. It is possible that there is also a right ventricular infarction associated with the inferior wall MI, and hence the origin of this PVC is the right ventricular free wall.
A 52-year-old man with newly diagnosed idiopathic dilated cardiomyopathy (based on the absence of coronary artery disease on previous coronary angiograms) and an ejection fraction of 40% presents with palpitations. The following ECG is obtained.

What is the rhythm diagnosis?
Does this patient need an implantable cardioverter–defibrillator placed?
ECG Analysis: Normal sinus rhythm, physiologic left axis, premature ventricular complexes (PVCs) (unifocal) in a bigeminal pattern (ventricular bigeminy)
The ECG shows a regularly irregular rhythm with alternating short (↓) and long (↑) RR intervals. There are narrow QRS complexes (▲) (duration = 0.08 sec) associated with P waves followed by premature complexes (●) that are not preceded by a P wave. The premature QRS complexes are wide (0.14 sec) and have an abnormal morphology that resembles neither a right nor a left bundle branch block. These are premature ventricular complexes (PVCs). Each of the narrow QRS complexes has a preceding P wave (+) with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes. The axis is physiologically leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (380/420 msec).

As there is a PVC after each sinus complex; this is called ventricular bigeminy, which is a term used to identify a repeating pattern (ie, every other QRS complex is a PVC). The morphology of the PVCs is uniform, so these are unifocal PVCs. The coupling interval (↔) between the sinus beat and PVC is fixed, meaning that the mechanism is reentry and there is a single reentrant circuit accounting for the one morphology of the PVCs and a fixed relationship (fixed coupling interval) between the sinus complex and the PVC. Noted with each PVC is an alteration of the T wave (↓), and in lead V1 (▲) this is a P wave, which is positive in leads II, aVF, and V5-V6. In addition, the PP interval (╚╝) between the P wave of the sinus complex and the P wave after the PVC is the same as the PP interval between the P wave after the PVC and the next sinus P wave. Hence this is the on-time sinus P wave, since the P waves can be marched out (ie, the PP intervals are constant). This P wave is blocked and not conducted through the AV node as a result of retrograde activation and depolarization of the AV node by the PVC, causing this structure to be refractory to the on-time sinus node activation. Hence the patient has an underlying sinus rhythm at a rate of 76 bpm.

The term ventricular bigeminy is of significance only because it means a repeating pattern (ie, every other QRS complex is a PVC). There is no clinical importance to the presence of a bigeminal pattern, except that the PVCs are frequent. No antiarrhythmic therapy (antiarrhythmic drug or implantable cardioverter–defibrillator) is indicated simply based on the presence of ventricular bigeminy. Typically, a defibrillator will be placed for non-ischemic cardiomyopathy only if one of the following criteria is met:

- The ejection fraction is less than 35% and the cardiomyopathy has been present for more than 9 months regardless of any rhythm disturbances. This is based on the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT).
- The patient has an ejection fraction less than 40%, nonsustained ventricular tachycardia, and inducible ventricular tachycardia on electrophysiology study.
- The patient experiences an episode of sustained ventricular tachycardia or ventricular fibrillation, regardless of ejection fraction.

It is important to remember that such a high frequency of PVCs over a period of months may uncommonly result in a cardiomyopathy. If this occurs, the PVCs should be treated aggressively (ie, with a class III antiarrhythmic drug or even radiofrequency ablation) if no other etiology is identified.
A 46-year-old woman with no past medical history presents to your clinic with feelings of extra heartbeats. She denies chest pain, dyspnea, lightheadedness, or syncope. Her physical examination is normal except for a regularly irregular rhythm. The following ECG is obtained.

**What is the diagnosis?**

**What is the underlying mechanism for this rhythm abnormality?**
ECG 70 Analysis: Sinus bradycardia, premature ventricular complexes (PVCs) (unifocal, interpolated) in a trigeminal pattern (ventricular trigeminy), retrograde concealed conduction
The ECG shows a regularly irregular rhythm, and there appears to be group beating. All the long intervals (∪) are the same, the short intervals (∩) are the same, and the intermediate intervals (¶) are the same. Noted are two narrow QRS complexes (*), after which there is a wide QRS complex (^) that is early or premature. The narrow QRS complex duration is 0.08 second, and there is a normal morphology and normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). There is low voltage (ie, < 5 mm) in each limb lead. The QT/QTc intervals are normal (400/390 msec). There is a P wave before each narrow QRS complex (+), and the P wave is positive in leads I, II, aVF, and V4-V6. Hence there is an underlying stable sinus mechanism, at a rate of 56 bpm (sinus bradycardia), as a P wave is seen before each of the narrow QRS complexes.

There are no P waves before the early, wide QRS complexes, which have an abnormal morphology that resembles a left bundle branch block (broad QS complex in lead V1 [←] and broad R wave in leads I and V5-V6 [→]). These are premature ventricular complexes (PVCs) and they are occurring after every second sinus complex; hence this is ventricular trigeminy. As with a bigeminy, trigeminy indicates a repeating pattern. There is no clinical importance to the presence of trigeminy except that the PVCs are frequent. Each PVC has the same morphology, so they are unifocal. The interval between the sinus complex and the PVC (¶) is the same each time (ie, there is a fixed coupling interval). This indicates that the etiology for the PVC is a reentrant circuit that is activated and generates only a single premature impulse.

The PVCs are not followed by a pause and they do not affect the underlying PP interval (↔), which is stable. Hence the PP interval around the PVC is identical to the PP interval without the PVC. Therefore, these PVCs are called interpolated. However, the PR intervals of the sinus complexes before and after the PVC are different. The sinus complex following the PVC has a PR interval that is longer (0.20 sec) than the PR interval of the sinus complex before the PVC (0.16 sec). This is the result of concealed retrograde (ventriculoatrial) conduction. The PVC partially penetrates the AV node in a retrograde direction but does not completely depolarize this structure. Hence it is concealed within the AV node. However, the AV node becomes partially depolarized and hence the next on-time sinus impulse conducts through the AV node but with a slower conduction velocity, accounting for the longer PR interval. n
A 32-year-old black man presents with palpitations and dyspnea. He has noted the dyspnea for many months now, ever since he lost 30 pounds. His only past medical history is that of uveitis treated with steroid drops 6 months ago. On physical examination, his jugular venous pressure is elevated, lower extremity edema and hepatomegaly are present, and his heart is dilated on palpation with an audible S3. There is also cervical lymphadenopathy. Chest imaging reveals bilateral hilar lymphadenopathy with pulmonary reticular opacities. An ECG is obtained because of the palpitations.

What is the diagnosis on ECG?

What is the most likely overall clinical diagnosis?
ECG 71 Analysis: Sinus rhythm, right bundle branch block (RBBB), left anterior fascicular block (LAFB), bifascicular disease, ventricular couplet (unifocal), ventricular triplet (nonsustained ventricular tachycardia [NSVT], monomorphic)
The ECG shows a regularly irregular rhythm. There are two different QRS complex morphologies. Complexes 1, 4, 5, 8, 9, 13, and 14 (up) are wide (0.14 sec) and have a right bundle branch block (RBBB) morphology (RSR' in lead V1 [→] and broad S waves in leads I and V4-V6 [←]). The broad R' in lead V1 and terminal broad S waves in leads I and V4-V6 result from terminal forces going from left to right. There is a P wave (+) before each of these QRS complexes with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence these are sinus complexes and the sinus rate is 80 bpm. The axis is extremely leftward, between –30° and –90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). This is a left anterior fascicular block (LAFB), which, along with the RBBB, indicates bifascicular disease (ie, disease of two of the three major fascicles innervating the ventricles). The QT/QTc intervals are normal (380/440 msec and 320/370 msec when corrected for the prolonged QRS complex duration).

After the first and fifth complexes (both of which are sinus), there are two sequential wide (0.18 sec) and early QRS complexes (↓) that are not preceded by a P wave. These QRS complexes have a left bundle branch block morphology (wide and deep QS complex in lead V1 [▼] and broad R waves in leads I and V5-V6 [↑]). These are premature ventricular complexes (PVCs). Two sequential PVCs is termed a ventricular couplet (⌜). After the ninth QRS complex (which is sinus) there is one episode of three sequential PVCs (▲); this is called a ventricular triplet (▏). By definition, three or more sequential PVCs lasting up to 30 seconds has also been called nonsustained ventricular tachycardia (NSVT). The term ventricular triplet or NSVT can be used in this situation. As all of the ventricular complexes have the same morphology, the couplets are unifocal and the NSVT is termed monomorphic.

This patient’s overall clinical presentation includes evidence of biventricular heart failure, conduction abnormalities, uveitis, lymphadenopathy, and pulmonary reticular opacities. These are the characteristic findings of sarcoidosis, which typically presents between the ages of 10 and 40 years and is much more common among blacks than other races. Cardiac sarcoidosis typically presents with heart failure or rhythm disturbances such as AV nodal block, intraventricular conduction delay, or ventricular arrhythmias, which are due to direct granulomatous inflammation and infiltration of the myocardium.
Notes
A 73-year-old man with an idiopathic dilated cardiomyopathy and a known ejection fraction of 30% presents with palpitations and dizziness. An implantable cardioverter–defibrillator was placed 2 years ago for primary prevention of sudden cardiac death. The following ECG is obtained while the patient is symptomatic.

What is the diagnosis?
Would any additional treatment be necessary?
ECG I2 Analysis: Sinus tachycardia, nonsustained ventricular tachycardia (NSVT) (monomorphic), fusion complexes
The ECG shows a regularly irregular rhythm with wide (▲) and narrow (●) QRS complexes. There are three narrow QRS complexes (●) (duration 0.08 sec), which are preceded by a P wave (+) with a stable PR interval (0.14 sec). The P wave is positive in leads II, aVF, and V4-V6; hence these are sinus complexes. These QRS complexes have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (360/460 msec). Following each of the sinus complexes is a four- to five-beat run (■) of a wide complex rhythm (QRS complex duration = 0.16 sec) at a rate of 134 bpm. The first QRS complex of these runs is premature. These QRS complexes do not have any apparent P wave before them. Therefore, these are ventricular complexes, or nonsustained ventricular tachycardia (NSVT), which is defined as a ventricular rhythm of three or more sequential premature ventricular complexes lasting up to 30 seconds at a rate higher than 100 bpm. The morphologies of the ventricular complexes are similar, with very subtle differences; hence this is monomorphic NSVT.

There does appear to be a P wave (●) before the first QRS complex of the NSVT run (▲). It should be noted that the PR interval before this complex is much shorter (‖) (0.10 sec) than the PR interval of the sinus complexes. In addition, the width of the first QRS complex of the run of NSVT (▲) is slightly wider (0.10 sec) than the sinus complexes but narrower than the subsequent ventricular complexes. Hence this is termed a fusion complex (ie, the QRS complex of this first complex of the run is the result of ventricular activation via both the normal AV node–His-Purkinje system and the ventricular pathway that is responsible for the NSVT). Hence, the complex represents fusion of activation by these two pathways. The presence of fusion is a feature of AV dissociation, which is commonly seen during a ventricular tachyarrhythmia (either NSVT or sustained ventricular tachycardia). Hence there is an independent atrial and ventricular rhythm; the ventricular rate is faster than the atrial rate. If the PP interval observed on the ECG is measured (ie, between the sinus P wave and the P wave just prior to the first QRS complex of the NSVT), it can be seen that the P waves thereafter occur on time and “march through” the NSVT; that is, there is a stable PP interval or atrial rate. Thus, the interval between the P wave of the sinus complex and the P wave before the first complex of the run is at a rate 100 bpm. By using this atrial rate, it can be seen that although the next P wave is within the second QRS complex of the run, the following on-time P wave is seen as a waveform at the very end of the third QRS complex of the run. By continuing to march out the P waves, it can be seen that the sinus P wave is on time.

continues
Ventricular rhythms can be identified by the following characteristics:

- QRS complexes are wide (> 0.12 sec) and have an abnormal morphology that resembles neither a typical right nor left bundle branch block.
- P waves (if seen) are usually unrelated to the QRS complexes (ie, AV dissociation is identified by a variable PR interval), and the ventricular rate is higher than the atrial rate. Often no P waves can be identified, especially if the ventricular rate is rapid.
- A negative P wave may be seen after the QRS complex if ventriculoatrial (VA) conduction is present. This usually occurs when the ventricular rate is slower and hence allows for VA conduction.
- Often the QRS complexes and ST-T waves demonstrate non-rate-related variability in morphology. This may be the result of subtle changes in ventricular depolarization and repolarization due to the fact that direct myocardial activation originates from a ventricular circuit that bypasses the normal His-Purkinje system. Hence there may be changes in the direction of the ventricular activation sequence. The changes in ST-T waves may also represent P waves that are superimposed on these waveforms.

- Fusion beats or completely captured (Dressler) beats may be seen. Fusion beats result from activation via two different pathways (the normal His-Purkinje system and the ventricular reentrant circuit). There is a P wave before the QRS complex, and the PR interval is shorter than that of the sinus complex. The QRS complex does not resemble either the sinus or the ventricular QRS complex but has features of both. Complete capture (Dressler complex) is more commonly seen when the rate of the ventricular rhythm is slower. At a slower rate there is less retrograde conduction into the AV node, thereby allowing more time for a sinus beat to completely penetrate the AV node antegradely and capture the ventricular myocardium. In this situation there is a definable PR interval followed by a QRS complex that has the same morphology as the sinus complex.

NSVT is commonly seen in patients with an idiopathic dilated cardiomyopathy. Although the patient does have an implantable cardioverter-defibrillator (ICD) placed for primary prevention of sudden death as a result of a low ejection fraction, the ICD only terminates sustained arrhythmia; it does not prevent arrhythmia. Hence it will not prevent the episodes of NSVT. If the symptoms do correlate with NSVT, this arrhythmia might need to be suppressed (for symptom relief and not for mortality benefit). In addition, if the runs of NSVT are long, they could impact ICD function and result in multiple ICD therapies and possible shocks. Suppression of the NSVT would require a standard antiarrhythmic drug. In a patient with a dilated cardiomyopathy, the safest agent would be amiodarone, dofetilide, or sotalol.
A 40-year-old man with diabetes and a family history of early coronary artery disease presents to the emergency department with acute-onset chest burning, jaw pain, and diaphoresis. When an initial ECG reveals inferior ST-segment elevations, the patient is taken emergently for cardiac catheterization. Angiography reveals a proximal right coronary artery thrombotic occlusion, which is opened via angioplasty and then stented. He subsequently is admitted to the hospital’s coronary care unit. The following ECG is obtained a few hours later when the patient’s vital signs are stable.

What is the diagnosis?
How should this be managed?
Podrid’s Real-World ECGs

ECG 73 Analysis: Normal sinus rhythm, accelerated idioventricular rhythm (AIVR)
The ECG begins with two narrow QRS complexes (▲) (duration = 0.08 sec) that are preceded by P waves (+) (PR interval = 0.16 sec) and are at a rate of 64 bpm. These are sinus complexes. The QT/QTc intervals are normal (400/410 msec). The third QRS complex (*) is wide (0.14 sec) with an abnormal morphology. This QRS complex is early, with a PR interval (‖) that is shorter (0.10 sec) than the baseline PR interval. The fourth QRS complex (▲) is also premature and wide with an abnormal morphology; the PR interval (●) is even shorter (0.08 sec). There are no P waves before the remaining QRS complexes. These are ventricular complexes and there is AV dissociation, as established by the initial variable PR intervals. The QRS complexes have a QS morphology in leads II and aVF, suggesting that they are originating from the inferior wall, likely from the area of the recent myocardial infarction (MI). The wide QRS complexes occur at a rate that is slightly faster (ie, 68 bpm) than the sinus rate. Hence this is an accelerated idioventricular rhythm (AIVR), which has also been termed slow ventricular tachycardia. The QRS complexes of the AIVR have a right bundle branch block pattern, with positive concordance (ie, there are tall R waves across the precordium). In addition, P waves can be seen following the fifth ventricular complex (▼) and the QRS complexes thereafter (note notching [▼] of the ST segment), with a fixed RP interval (↔). These are retrograde P waves as a result of intact ventriculoatrial conduction.

An AIVR arises below the AV node and has, by definition, a rate between 60 and 100 bpm. Although it may be the result of pacemaker failure, and therefore an escape rhythm, AIVR most often represents an abnormal ectopic focus in the ventricle that is accelerated by sympathetic stimulation and circulating catecholamines. It may be seen in up to 50% of patients with an acute MI. AIVR is often observed in the setting of coronary artery reperfusion, especially with thrombolytics. It is believed to be a reperfusion arrhythmia, although its mechanism is not clear. It may be related to rapid reperfusion with the washout of various substances that are released by the damaged myocardium, including potassium and other electrolytes. AIVR is usually transient and asymptomatic.

No specific treatment is necessary unless the rate is rapid and it is associated with symptoms or evidence of hemodynamic impairment. In this situation, a standard antiarrhythmic drug may be effective for its suppression. However, before therapy is initiated it is important to make certain that the ventricular rhythm is not an escape rhythm as a result of complete heart block. In this situation a pacemaker may be required before suppression of the ventricular rhythm as the ventricular arrhythmia is escape and its suppression would result in asystole. If the onset of the arrhythmia can be seen, a ventricular rate that is faster than the sinus rate is characteristic of AIVR. Observing AV dissociation during the ventricular rhythm with an atrial rate slower than the ventricular rate is also characteristic of an accelerated ventricular focus. In contrast, a long pause and often a nonconducted P wave after which the ventricular rhythm occurs is characteristic of complete heart block and an escape ventricular rhythm. If AV dissociation is seen in this situation, the atrial rate is faster than the ventricular rate.
A 59-year-old man who had a myocardial infarction 5 months previously presents to the emergency department with a history of palpitations, presyncope, and one syncopal episode. While in the
emergency department, his symptoms return. ECG 74A is obtained while the patient is symptomatic; ECG 74B is the baseline ECG obtained when he first presented and before the onset of symptoms.

What is the diagnosis?
What therapy would be indicated for this patient?
ECG 74A Analysis: Sustained monomorphic ventricular tachycardia
ECG 74A shows a wide complex tachycardia (QRS width = 0.18 sec) at a rate of 130 bpm. No obvious P waves are seen before or after the QRS complexes. However, there appears to be a P wave (*) in front of the second, seventh, and 12th QRS complexes in lead V1. In addition, the T waves after the third, eighth, 13th, and 18th QRS complexes in lead V1 have a different morphology (+) compared with the other T waves; they have a positive deflection at the very end of the waveform (+), suggesting a superimposed P wave. Lead I shows a notching of the T wave (↓) after the third QRS complex that is not seen after the other QRS complexes in this lead. Thus, there is evidence of AV dissociation. Although the P waves cannot be marched out through the ECG tracing, the fact that there are P waves associated with some but not all of the QRS complexes is indicative of AV dissociation. Moreover, the association between the P wave and QRS complex is variable. The presence of AV dissociation is also indicated by the presence of subtle changes in the morphology of the ST-T waves. Any wide complex arrhythmia with evidence of AV dissociation is ventricular tachycardia.

It should be noted that the positive waveform after the QRS complex in leads V1-V2 is not a P wave but is the terminal part of the QRS complex, which is determined by comparing the QRS width in another lead, such as lead aVF, II, or V3 (‖). The QRS complexes have a uniform morphology that resembles a right bundle branch block (RBBB), but it is not a typical RBBB and there is a marked leftward axis. This is sustained monomorphic ventricular tachycardia. Sustained ventricular tachycardia is defined as a ventricular rhythm at a rate higher than 100 bpm that lasts for more than 30 seconds or is terminated within 30 seconds because of hemodynamic instability. Sustained monomorphic ventricular tachycardia is not an ischemically mediated arrhythmia; it results most often from reentry in myocardium that has been previously damaged by infarction (ischemic heart disease) or inflammation (cardiomyopathy) and has areas of fibrosis adjacent to areas of normal myocardial tissue, allowing for the existence of reentrant circuits. Therefore, in patients with heart disease it is scar related.

An important concern is establishing the etiology for a wide complex tachycardia (ie, ventricular tachycardia vs. supraventricular tachyarrhythmia associated with aberrancy). Ventricular tachycardia has the following features:

- QRS complexes are wide (> 0.12 sec) and abnormal in morphology. QRS complexes wider than 0.16 second are usually ventricular.
- P waves (if seen) are dissociated from QRS complexes (AV dissociation; ie, the PR interval is variable), and the ventricular rate is faster than the atrial rate.
- On occasion a negative P wave may be seen after the QRS complex, indicating the presence of ventriculoatrial conduction. This is most often seen when the ventricular tachycardia rate is slower.

continues
• Often, QRS complexes and ST-T waves show non-rate-related variability in morphology. This may be the result of subtle changes in ventricular depolarization and repolarization due to the fact that there is direct myocardial activation originating from a ventricular circuit that bypasses the normal His-Purkinje system. Hence there may be changes in the direction of the ventricular activation sequence. The changes in ST-T waves may also represent P waves that are superimposed on these waveforms.

• Fusion or captured (Dressler) beats may be seen. Fusion beats result from myocardial activation via two different pathways that “fuses” (ie, the normal His-Purkinje system and the ventricular reentrant circuit). There is a P wave before the QRS complex, and the PR interval of the QRS complex is shorter than that of the sinus complex. The QRS complex does not resemble either the sinus or the ventricular QRS complex but has features of both. Complete capture is more commonly seen when the rate of the ventricular rhythm is slower, allowing more time for a sinus beat to completely penetrate the AV node and capture the ventricular myocardium in an antegrade direction. In this situation there is a definable PR interval (similar to or longer than that of the sinus PR interval) followed by a QRS complex that is the same as the sinus complex.

Other important features that also suggest ventricular tachycardia as the etiology for a wide complex tachycardia include:

• Indeterminate axis (ie, a QRS complex that is negative in leads I and aVF). An indeterminate axis may also be seen when there is direct myocardial activation, such as with arrhythmias associated with a Wolff-Parkinson-White (WPW) pattern or in paced rhythms. A significant shift in axis compared with sinus rhythm is also seen with ventricular tachycardia.

• Positive concordance (tall R waves) across the precordium. This may also be seen in situations in which there is direct myocardial activation, such as WPW or a paced rhythm. Negative concordance is less useful as this pattern may be seen with a left bundle branch block.

• QRS complex width longer than 0.16 second

• If R/S morphology is seen in any precordial lead, an R wave that is wider than the S wave (R/S >1) or an R wave longer than 100 msec is strongly correlated with a ventricular complex. With a ventricular complex the initial ventricular activation is directly through the ventricular myocardium and hence it is slow, resulting in a QRS complex that has an initial waveform (ie, the R wave) that is widened. Indeed, the entire QRS complex is abnormal. In contrast, when there is a supraventricular complex that is aberrated, the widened QRS complex is a result of terminal delay in activation (ie, the terminal portion of the QRS
complex is widened as a result of conduction block in the right or left bundle branch). In this situation, the R wave is narrower than the S wave and less than 100 msec because the initial forces and waveform of the QRS complex are normal as a result of normal ventricular conduction through the normal functional bundle, while the terminal portion of the QRS complex is due to abnormal and slow conduction through the ventricle served by the bundle that is not functional.

- Other morphologic features of the QRS complex that may be useful include the following:
  
  - A monophasic R or biphasic qR complex in lead V1 favors ventricular tachycardia; this represents the lack of an RSR’ pattern.
  
  - A triphasic RSR’ or RsR’ complex (the so-called “rabbit-ear” sign) in lead V1 usually favors a supraventricular tachyarrhythmia. As an exception, if the R wave (initial positive waveform) of the RsR’ complex is taller than the R’ (terminal positive deflection), ventricular tachycardia is suggested.

  - An rS complex (R wave smaller than S wave) in lead V6 favors ventricular tachycardia. In contrast, an Rs complex (R wave larger than S wave) in lead V6 favors a supraventricular tachyarrhythmia.

  - A broad initial R wave 40 msec in duration or longer in lead V1 or V2 favors ventricular tachycardia. In contrast, the absence of an initial R wave or a small initial R wave of less than 40 msec in lead V1 or V2 favors supraventricular tachyarrhythmia.

  - A slurred or notched downstroke of the S wave in lead V1 or V2 and a duration from the onset of the QRS complex to the nadir of the QS or S wave of 60 msec or longer in lead V1 or V2 favors ventricular tachycardia. In contrast, a swift, smooth downstroke of the S wave in lead V1 or V2 with a duration of less than 60 msec favors a supraventricular complex.

  - The presence of any significant Q wave or a QS complex in lead V6 is suggestive of ventricular tachycardia. In contrast, the absence of a Q wave in lead V6 favors a supraventricular complex.

continues
Podrid’s Real-World ECGs

ECG 743 Analysis: Normal sinus rhythm, intraventricular conduction delay, first-degree AV block
**ECG 74B**, from the same patient, is the baseline ECG. There is a regular rhythm at a rate of 64 bpm. There is a P wave (+) before each QRS complex with a stable PR interval of 0.24 second (first-degree AV block). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is increased (0.14 sec) without any specific pattern of a bundle branch block. Therefore, this is an intraventricular conduction delay. The axis is normal, between $0^\circ$ and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/390 msec and 320/330 msec when corrected for the prolonged QRS complex duration). When compared with the QRS complexes seen in **ECG 74A**, it can be seen that the morphology and axis of the QRS complexes during the tachycardia are unlike those during sinus rhythm. Along with the presence of AV dissociation, it is clear that the rhythm in **ECG 74A** is ventricular tachycardia.
The following ECG was obtained during an exercise tolerance test in a 32-year-old man who collapsed after 3 minutes of exercise. He had initially presented with progressive dyspnea on exertion. On physical examination prior to the test, a grade III/VI systolic murmur was audible, best heard at the left lower sternal border and increased with Valsalva. The patient reported having an uncle who died suddenly in his 30s. Of note, his resting blood pressure was 140/90 mm Hg, but within 2 minutes of exercise it had dropped to 90/50 mm Hg.

What is the rhythm diagnosis?
What is the overall likely clinical diagnosis?
Podrid’s Real-World ECGs

ECG 75 Analysis: Sustained monomorphic ventricular tachycardia (ventricular flutter)
The ECG shows a wide QRS complex (0.20 sec) tachycardia at a rate of 270 bpm. There are only two rhythms that present with rates higher than 260 bpm: atrial flutter with 1:1 AV conduction and ventricular tachycardia. Changes can be seen in the QRS morphology (↑) as well as in the ST-T waves (↓), most apparent in the precordial leads. These changes would not be seen with any supraventricular tachyarrhythmia because impulse conduction to the ventricles always follows the same conduction pathway and hence there is uniformity of the QRS complexes and ST-T waves. Therefore, this is sustained monomorphic ventricular tachycardia. When ventricular tachycardia occurs at a rate exceeding 260 bpm, it is often termed ventricular flutter.

Typically, 12-lead ECGs are not obtained in the setting of ventricular tachycardia arrest as this would delay time to appropriate therapy (ie, defibrillation). However, as the arrest occurred during an exercise test, the patient was already wearing the leads. Based on the history and results from the exercise test, the overall clinical diagnosis is likely hypertrophic (obstructive) cardiomyopathy, a genetic disorder that classically presents with a systolic murmur audible at the left lower sternal border that increases with Valsalva. This aortic stenosis–type murmur represents turbulent flow through an obstructed left ventricular outflow tract (ie, subvalvular stenosis). The obstruction is due to a hypertrophic but hypokinetic septum. As a result the pressure is less along the septum compared with the rest of the posterior ventricular wall, resulting in a Venturi effect that “sucks” the anterior leaflet of the mitral valve to the septum (ie, systolic anterior movement of the mitral valve), which results in a midsystolic gradient within the left ventricular chamber.

The clinical finding of a systolic murmur that gets louder with Valsalva is due to the decrease in venous return, the decreased filling of the left ventricle, and hence a reduction in the dimension of the outflow tract, causing an increase in the obstruction and in the intensity of the murmur. Patients with hypertrophic cardiomyopathy are at increased risk for sudden cardiac death, particularly those who experience blood pressure drops with exercise, have marked left ventricular hypertrophy (> 30 mm), have a family history of sudden cardiac death, experience syncope or a documented sustained ventricular tachyarrhythmia (ventricular tachycardia or ventricular fibrillation), or have episodes of nonsustained ventricular tachycardia. Therapy for these patient is generally an implantable cardioverter–defibrillator.
A 70-year-old man with no known prior cardiac history presents with 3 days of intermittent chest pressure along with episodes of dizziness occurring at rest. The following lead II ECGs are obtained during an episode of chest discomfort.

What is the diagnosis?
ECG 76 Analysis: Nonsustained polymorphic ventricular tachycardia
This series of rhythm strips shows narrow QRS complexes (\(^\wedge\)), preceded by P waves (\(\dagger\)) with a stable PR interval (0.16 sec); this is sinus rhythm at a rate of 75 bpm. Each strip shows a nonsustained episode of a rapid wide complex tachycardia that is ventricular in origin. Each episode terminates spontaneously after a few seconds; therefore, this is nonsustained ventricular tachycardia. However, there are marked changes in QRS morphology and QRS axis (\(\uparrow\)); therefore, this is polymorphic ventricular tachycardia. The rate of the ventricular tachycardia is about 300 bpm. It should be noted that the QT interval (\(\leftrightarrow\)) of the sinus beats is normal at 360 msec. In the presence of a normal QT interval this is termed polymorphic ventricular tachycardia, and the most common etiology is active ischemia. The only three arrhythmias provoked by active ischemia are polymorphic ventricular tachycardia, ventricular flutter (ventricular tachycardia with a rate > 260 bpm), and ventricular fibrillation.

Another less common cause for polymorphic ventricular tachycardia is familial catecholaminergic polymorphic ventricular tachycardia, which is the result of a genetic abnormality affecting the ryanodine or calsequestrin 2 gene.
A 54-year-old woman with hypertension, treated with hydrochlorothiazide, and chronic back pain, for which she takes methadone, presents to the emergency department with a severe cough. She is diagnosed with bronchitis, and levofloxacin is prescribed. Three days later she again presents to the emergency department with complaints of severe nausea and vomiting as well as diarrhea. She has attempted to stay hydrated and to eat but has been unable to due to severe emesis with intake. She was prescribed ondansetron for her nausea and ciprofloxacin for her diarrhea at an outpatient clinic. However, her symptoms did not improve. While in the emergency department, the patient has a syncopal episode that is captured on the telemetry strip below.

What is the diagnosis?
What is contributing to her rhythm disturbance?
Podrid’s Real-World ECGs

ECG 77 Analysis: Nonsustained polymorphic ventricular tachycardia, torsade de pointes
This continuous lead II rhythm strip shows three narrow QRS complexes (▼) that are preceded by P waves (+). These are likely sinus beats. The third and fifth QRS complexes (★) are premature ventricular complexes (PVCs) that occur slightly after the apex of the T wave (↓) (R on T). After the sixth QRS complex there is an episode of a rapid wide complex tachycardia with QRS complexes that are changing in morphology and axis. The rate of the episode approaches 300 bpm. This is polymorphic ventricular tachycardia.

Although the T waves are interrupted by the PVC, it can be noted that the sinus complexes have a prolonged QT interval (↔), as what can be measured shows a QT interval of at least 600 msec, which is very prolonged. The episode of polymorphic ventricular tachycardia in this case is called “torsade de pointes” or twisting of points, which is defined as polymorphic ventricular tachycardia associated with QT prolongation.

QT prolongation may be congenital or acquired as the result of a medication. In this case, the QT prolongation is most likely acquired. Given her severe diarrhea and emesis with poor oral intake, she is likely hypokalemic and hypomagnesemic, which are two major risk factors for acquired QT prolongation and torsade de pointes. In addition, she has been taking two known QT prolonging agents: a quinolone and methadone. Other commonly known QT prolonging agents include class IA and III antiarrhythmics, psychotropic agents such as haloperidol, macrolides, and antifungal agents such as voriconazole.
An 81-year-old man with diabetes but no known cardiac disease and plans for below-the-knee amputation of his right leg for severe osteomyelitis and unhealed skin ulcers develops profound hypotension and altered mental status requiring vasopressor therapy. He is intubated and admitted to the intensive care unit.

Initial laboratory assessment reveals acute renal insufficiency, hyperkalemia, lactic acidosis, and a blood pH of 7.10. Blood cultures grow methicillin-resistant Staphylococcus aureus. Despite antibiotic therapy, his clinical situation worsens. Overnight, he acutely loses blood pressure and the following ECG is captured on telemetry.

**What is the diagnosis?**
Podrid’s Real-World ECGs

ECG 78 Analysis: Ventricular fibrillation
No organized QRS complexes can be seen on this ECG. Rather, there are rapid and irregular waveforms that are completely disorganized and without any distinct morphology. Therefore, this is ventricular fibrillation. At times the waveforms look more organized (↓), as in lead V3 for example, and resemble polymorphic ventricular tachycardia. However, they fragment thereafter (^).

An arrest due to ventricular fibrillation, which is the most frequent cause of sudden cardiac death, is most commonly associated with active ischemia or an acute coronary syndrome (unstable angina, non-ST-segment elevation myocardial infarction, or ST-segment elevation myocardial infarction) or with significant structural heart disease (ischemic cardiomyopathy, non-ischemic cardiomyopathy, aortic stenosis, aortic dissection, myocarditis, or pericardial tamponade). However, it can also occur in the setting of profound metabolic disturbances such as acidosis and septic shock, as illustrated in this case. Respiratory failure due to aspiration, bronchospasm, sleep apnea, or pulmonary embolism can also result in ventricular fibrillation arrest.

The only effective therapy for ventricular fibrillation is prompt electrical defibrillation. Ventricular fibrillation does not revert spontaneously, nor are antiarrhythmic drugs effective for reversion.
An 86-year-old woman with known calcific aortic stenosis and a valve area of 1.0 cm² presents with worsening dyspnea on exertion. The following is her ECG.

What are the abnormal findings?
ECG 79 Analysis: Normal sinus rhythm, first-degree AV block, intraventricular conduction delay (IVCD), left anterior fascicular block, left ventricular hypertrophy (LVH)
The ECG shows a regular rhythm at a rate of 74 bpm. There is a P wave (+) before each QRS complex, with a fixed PR interval (0.24 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with a first-degree AV block.

The QRS duration is increased (0.12 sec). The QRS pattern is not typical for a right bundle branch block and it is not a left bundle branch block, as there is a septal Q wave in lead aVL (▲) and a septal R wave in lead V1 (▼), which cannot be seen with a left bundle branch block as the septal branch innervating the septum is from the left bundle. Hence this is an intraventricular conduction delay (IVCD). The axis is very leftward, between –30° and –90° (the QRS complex is positive in lead I and negative in leads II and aVF) as a result of a left anterior fascicular block. The QRS complexes have a normal morphology, although there is increased amplitude, with an S wave in lead V3 that is 25 mm deep (▼). This meets a criterion for borderline left ventricular hypertrophy (LVH) (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). Along with the IVCD and left axis, LVH is likely present.

The QT/QTc intervals are normal (400/440 msec), but there are very prominent T waves (▼) that are tall and peaked (especially in leads V1-V4). However, the T waves are asymmetric, with an upstroke that is slower than the downstroke. Hence these T waves are normal and are not hyperacute, the result of hyperkalemia. It is likely that they are very prominent as a result of LVH. There are also T-wave inversions in leads I and aVL (▲), which are probably repolarization abnormalities due to LVH.
A 52-year-old man is started on spironolactone along with an angiotensin-converting enzyme inhibitor for what is thought to be salt-sensitive hypertension. The following ECG is obtained routinely at a clinic visit 4 weeks later.

What is your next step in management?
ECG Analysis: Atrial rhythm, left ventricular hypertrophy, hyperacute T waves (hyperkalemia)
The ECG shows a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.14 sec). However, the P waves are negative in leads II and aVF. Hence this is not a sinus rhythm; it is an atrial rhythm.

The QRS complex duration is normal (0.08 sec), and the QRS complexes have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal, but there is increased voltage with an R wave in lead V4 of 30 mm (1), which is diagnostic for left ventricular hypertrophy (i.e., S-wave depth or R-wave amplitude in any one precordial lead ≥ 25 mm). The QT/QTc intervals are normal (400/400 msec).

The T waves are tall and peaked and, most importantly, symmetric, or tented (↓). These are termed hyperacute T waves and are seen with hyperkalemia (systemic or local as in an acute myocardial infarction). The patient’s potassium level should be checked urgently, and the patient should be maintained on telemetry in a monitored setting with routine vital sign assessment.

Hyperkalemia can cause serious ventricular arrhythmias and conduction abnormalities, particularly if hyperacute T waves and QRS widening are seen on the ECG. QRS complex widening associated with elevated serum potassium levels should be treated in the short-term with intravenous administration of regular insulin with dextrose (for an acute decrease in potassium levels by shifting potassium into cells) and sodium bicarbonate. This treatment should be preceded by calcium infusion to stabilize the cardiac membrane potential in all settings except when digitalis toxicity is also present. Kayexalate (sodium polystyrene sulfonate) is also given for elimination of excess potassium from the body via the gastrointestinal tract.

Spironolactone is a potassium-sparing diuretic that is often used in hypertension and in patients with class IV heart failure. It can cause life-threatening hyperkalemia, particularly in patients with renal dysfunction or in patients also receiving an angiotensin-converting enzyme inhibitor. For this reason potassium levels should be monitored routinely in anyone taking this medication.
Notes
A 68-year-old woman with end-stage renal disease due to diabetic and hypertensive nephropathy develops an infection as a result of cellulitis at a newly placed arteriovenous fistula. Her blood cultures are positive for staphylococcal bacteremia, and intravenous antibiotics are prescribed. Two days later her blood urea nitrogen and creatinine levels increase, and she develops nausea. An ECG is obtained.

What is the diagnosis?
What is the next step in management?
ECG 81 Analysis: Hyperkalemia
The ECG shows a regular wide QRS complex rhythm at a rate of 48 bpm. There is no evidence of atrial activity; therefore, this may be a junctional or ventricular rhythm. The QRS width (→) is 0.28 second. The only etiology for a QRS that is this wide is hyperkalemia, which causes the resting membrane potential of the ventricular myocardium to become less negative. The normal resting membrane potential is –90 mV, which is maintained by an intracellular potassium level that is higher than the extracellular potassium level. When extracellular hyperkalemia is present, the balance is abnormal and the resting membrane potential becomes less negative. As the resting membrane potential approaches the threshold membrane potential (–60 mV), there is a decrease in the rate of the upstroke velocity of phase 0, which determines the impulse conduction velocity. This decrease in the velocity of impulse conduction through the ventricular myocardium results in a widening of the QRS complex. Hyperkalemia is the only condition that will result in a QRS width of 0.24 second or longer.

Also seen on this ECG are T waves that are very symmetric (+). Although no P waves are seen, this could still be a sinus rhythm, as the atrial myocardium is more sensitive to hyperkalemia than the ventricular myocardium. Atrial asystole can develop before there is QRS widening. This results in a continued sinus rhythm but no obvious P waves since the atrial myocardium is nonresponsive to electrical activity. This has been termed a sinoventricular rhythm.

In addition to the treatment described for Case 80, this patient may need urgent dialysis to treat the hyperkalemia.
A 56-year-old man is diagnosed with essential hypertension and is started on hydrochlorothiazide. He has no other medical problems and is otherwise healthy. This ECG is obtained on routine follow-up.

What is the abnormality?
**ECG Analysis:** Normal sinus rhythm, physiologic left axis, prominent U wave (hypokalemia)
The ECG shows a regular rhythm at a rate of 64 bpm. There are P waves (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration (0.08 sec) and morphology are normal. The axis is physiologically leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are slightly prolonged (440/460 sec).

There is a prominent positive waveform after the T wave in leads V3-V5. This is a U wave (↓), which is believed to represent delayed repolarization of the His-Purkinje system (or possibly the papillary muscles). The His-Purkinje system is the first part of the myocardium to depolarize and the last to repolarize. Small U waves are frequently seen in the right precordial leads (V1-V3). However, U waves that become very prominent and extend to the left precordial leads suggest the presence of hypokalemia. Loop diuretics and thiazides can often cause hypokalemia; therefore, serum potassium levels should be checked routinely while patients are taking these medications. Severe hypokalemia (usually < 2.0 mEq/L) can result in serious ventricular tachyarrhythmias, including cardiac arrest due to ventricular fibrillation. Potassium supplementation is necessary for short-term treatment, and either discontinuation of the offending agent or daily potassium supplementation is a reasonable long-term management strategy.
A 37-year-old man with an underlying manic disorder has been taking chlorpromazine for mood stabilization. He presents to a walk-in clinic with complaints of lightheadedness and pre-syncope. He states that he mistakenly took several extra chlorpromazine pills. An ECG is obtained.

What are the abnormal findings?
What is the appropriate treatment?
Podrid’s Real-World ECGs

ECG 83 Analysis: Ectopic atrial rhythm, long QT interval, long QT syndrome
The ECG shows a regular rhythm at a rate of 48 bpm. There are P waves (+) before each QRS complex. They are inverted in leads II, aVF, and V3-V5, indicating an ectopic atrial (not sinus) rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0 and +90° (positive QRS complex in leads I and aVF). Noted is marked prolongation of the QT interval (↔), which measures 680 msec (QTc = 640 msec). The long QT interval is the result of a very prolonged T-wave duration, which represents prolonged repolarization. A long QT interval may be congenital or acquired. QT prolongation (either congenital or acquired) is associated with polymorphic ventricular tachycardia, called torsade de pointes. In this case, the long QT syndrome is possibly related to the use of the antipsychotic agent chlorpromazine, which is known to prolong the QT interval.

The treatment for an acquired QT prolongation is withdrawal of the implicated medication and observation. The occurrence of torsade de pointes is generally bradycardic or pause dependent in acquired QT prolongation. It can be suppressed by increasing the heart rate, as with overdrive pacing or the use of isoproterenol. Magnesium may also be beneficial. In contrast, torsade de pointes in association with congenital QT prolongation is provoked during tachycardia. Initial treatment is β-blockade to interfere with sympathetic stimulation of the heart, which can provoke torsade de pointes in the congenital QT syndrome.
Notes
A 52-year-old woman develops diffuse muscle cramps 2 days after undergoing thyroidectomy for papillary thyroid cancer. Physical examination is notable for facial muscle twitching upon tapping of the ipsilateral peri-auricular area. The following ECG is obtained.

What is the abnormality?
What is the overall clinical diagnosis?
What treatment is indicated?
ECG 84 Analysis: Normal sinus rhythm, long QT interval (hypocalcemia)
The ECG shows a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6, indicating a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), with a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). Notable is a prolonged QT interval (↔) of 560 msec (QTc = 590 msec). The QT prolongation is the result of a long ST segment (↑); the T wave itself is normal in duration. This is termed delayed repolarization and is seen with metabolic abnormalities, primarily hypocalcemia or hypomagnesemia. QT prolongation due to delayed repolarization (a long ST segment) is not associated with arrhythmia, particularly torsade de pointes.

This patient has hypocalcemia due to thyroidectomy, which has resulted in hypoparathyroidism. Hypocalcemia is typically transient in this setting but can be permanent in roughly 1% of patients undergoing thyroidectomy. Chvostek’s sign, in which tapping of the facial nerve results in contraction of the facial muscles, is a sensitive sign for hypocalcemia. When QT prolongation is seen, treatment consists of urgent intravenous calcium supplementation.
A 56-year-old man with 2 days of epigastric pain and nausea is found to have evidence of cholecystitis on abdominal ultrasound. He undergoes laparoscopic cholecystectomy without complication.
On postoperative day 1, a routine ECG (85A) is obtained. The patient is otherwise asymptomatic and stable. The preoperative ECG (85B) is also shown.

Has this patient had a perioperative myocardial infarction?
If so, where would you localize the infarct?
ECG 85A Analysis: Sinus bradycardia, right-to-left arm lead switch, left ventricular hypertrophy (LVH), early repolarization
ECG 85A shows a regular rhythm at a rate of 56 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are upright in leads II, aVF, aVR, and V4-V6; they are inverted in leads I and aVL (^). In addition, the QRS complex is negative in these leads, giving the appearance of a rightward axis, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF), from either a lateral myocardial infarction (MI) or a left posterior fascicular block.

The QRS complex duration is normal (0.08 sec), and the QT/QTc intervals are normal (380/370 msec). The QRS complex has a QS morphology in leads I and aVL (↓), which suggests a lateral wall MI. There is a tall R wave in lead aVR (▼), which is abnormal. The T waves are also inverted in leads I and aVL (*) and upright in lead aVR. Although a lateral wall MI is strongly suggested by the Q waves and T-wave inversions, the negative P wave (^), QRS complex (↓), and T wave (*) in leads I and aVL and the positive P wave and QRS complex in lead aVR are characteristic of a right-to-left arm lead switch, which is a very common error. Although the activation sequence of the atria and ventricles is normal, going from right to left, the impulse is going toward the right arm lead (rather than away from the right arm lead) and away from the left arm lead (rather than toward the left arm lead), accounting for the negative waveforms. Thus, the patient has not suffered from a lateral MI.

Also noted is an R wave in leads V4-V5 ( [] ) that is increased in amplitude (27 to 33 mm), which is diagnostic for left ventricular hypertrophy (LVH) (ie, S-wave depth or R-wave amplitude in any one precordial lead ≥ 25 mm). A minimal amount of early repolarization (↑) (slight J-point and ST-segment elevation) can also be seen in leads V2-V4. **continues**
Podrid’s Real-World ECGs

ECG Analysis: Sinus bradycardia, LHV
In ECG 85B, the leads have been placed on the correct arms. The rate is 50 bpm, and the PR interval is normal (0.16 sec). This is a normal sinus rhythm. The P waves (+), QRS complex, and T waves (▲) are now upright and normal in appearance in leads I and aVL. The P wave (*), QRS complex (↑), and T wave (▲) are negative in lead aVR, which is normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). LVH is present as previously noted.

Deciphering whether an ECG has limb lead reversal is easiest done by first looking at the unipolar leads (aVR, aVL, and aVF). In a right-to-left arm lead switch, the QRS complex in lead aVR (which should be negative) and lead aVL (which should be positive) will be inverted (positive and negative, respectively) on the ECG and lead aVF will be unchanged. Additionally, in a right-to-left arm lead reversal, lead I—which is a bipolar lead that looks at the impulse as it goes from right to left and should always have positive P, QRS, and T waveforms—will have negative P, QRS, and T waveforms.
A 67-year-old man with a history of obesity, obstructive sleep apnea, hypertension, and diastolic dysfunction presents to your office with complaints of shortness of breath. He tells you that he has been smoking two packs of cigarettes per day for about 50 years.
On physical examination, he has rhonchous sounds in both lung fields and 2+ peripheral edema in both legs. An ECG is obtained (86A) and is compared with the baseline ECG (86B).

Does this patient have right ventricular hypertrophy?
ECG 86A Analysis: Normal sinus rhythm, V1-V3 lead switch, left ventricular hypertrophy (LVH)
ECG 86A shows a regular rhythm at a rate of 66 bpm. The P waves (+) are upright in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/420 msec). However, lead V1 has a very tall R wave (←), which is characteristic of right ventricular hypertrophy or a posterior wall myocardial infarction. The R-wave amplitude in lead V2 is smaller (→); it is even smaller in lead V3 (↓). The R-wave amplitudes in leads V4-V6 are normal. Hence leads V1-V3 show reverse R-wave progression. This is the result of a V1-V3 lead switch, which is a very common error.

In addition, there are voltage criteria for left ventricular hypertrophy (LVH), with R waves in leads V4-V5 (↑) of 30 to 37 mm (S-wave depth or R-wave amplitude in any one precordial lead ≥ 25 mm is one of the criteria for LVH).

continues
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, LVH
In ECG 86B, the precordial leads have been placed in the correct position. The R-wave progression in leads V1-V3 is now normal. By comparison, lead V1 in ECG 86A is actually lead V3, V2 is actually V1, and V3 is actually V2. The rate is 72 bpm, and the P waves; PR interval; QRS complex morphology, axis, and duration; and QT/QTc intervals are the same as in ECG 86B.
A 47-year-old man with HIV and a new diagnosis of B-cell lymphoma presents with progressive lightheadedness and dizziness as well as the sensation of a racing heartbeat. On physical examination, his blood pressure is 72/palp, his jugular venous pressure is elevated, and his lung fields are clear. An arterial line is placed for precise blood pressure monitoring, and there is significant respiratory variation in the tracing with a decrease occurring on inspiration. The following ECG is obtained.

What is the ECG abnormality? What is the overall clinical diagnosis?
Podrid’s Real-World ECGs

ECG 87 Analysis: Sinus tachycardia, low voltage
The ECG shows a regular rhythm at a rate of 140 bpm. P waves (+) are noted before each QRS complex, with a fixed PR interval (0.10 sec), and the P waves are upright in leads I, II, aVF, and V4-V6. Hence this is a sinus tachycardia. The PR interval (‖) is short as the result of increased sympathetic stimulation, which accounts for the sinus tachycardia as well as increased AV nodal conduction velocity and hence the shorter PR interval.

The QRS complex duration is normal (0.06 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (260/400 msec). However, the R-wave voltage in all the leads is very reduced, although it should be noted that the ECG is recorded at normal standardization (♦) (ie, 1 mV = 10 mm or 10 small boxes in height). Therefore, this demonstrates low voltage in all leads.

The definition of low voltage is less than 5 mm of amplitude in each of the limb leads and less than 10 mm of amplitude in each of the precordial leads (when the ECG is recorded at normal standard). Low voltage may be seen in the limb leads, precordial leads, or all leads. The presence of low voltage indicates that there is less electrical activity from the heart reaching the surface of the body to be recorded. This may be the result of body habitus (ie, obesity), significant lung disease (particularly chronic obstructive pulmonary disease), a pericardial effusion or thickened pericardium, or loss of myocardial muscle mass (eg, as in amyloidosis).

In this case, based on the history and physical examination, the patient has a pericardial effusion with tamponade physiology. Beck’s classic triad for a pericardial effusion consists of elevated jugular venous pressure, muffled heart sounds, and hypotension. A drop of more than 10 points in systolic arterial blood pressure during inspiration is a classic finding in tamponade known as pulsus paradoxus and is related to the interventricular dependence that occurs with a substantial pericardial effusion. With inspiration and an increase in venous return to the right ventricle, there is reduced filling of the left ventricle and hence a reduction in stroke volume and blood pressure. With expiration, there is a reduction of venous return and right ventricular filling during inspiration, and hence left ventricular filling, stroke volume, and blood pressure increase. Treatment consists of intravenous fluids and pericardiocentesis. The most common ECG findings are sinus tachycardia and low voltage. Diagnostic for tamponade, but not always seen, is electrical (QRS) alternans (beat-to-beat variation in the QRS amplitude). If pericarditis is also present, then one might also see diffuse concave ST-segment elevations with PR depression. In this patient with B-cell lymphoma, a malignant pericardial effusion is high on the differential diagnosis.
A 65-year-old man with known coronary artery disease and chronic hypertension has the following ECG.

Does this ECG meet criteria for left ventricular hypertrophy?
Podrid’s Real-World ECGs

ECG Analysis: Normal sinus rhythm, chronic inferior wall myocardial infarction, low-voltage limb leads, recorded at double standard.
The ECG shows a regular rhythm at a rate of 86 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are upright in leads I, II, aVF, and V4-V6, indicating a normal sinus rhythm. There are Q waves (↑) in leads II, III, and aVF, defining the presence of a chronic inferior wall myocardial infarction (MI). As a result of the inferior wall MI, with negative QRS complexes in leads II and aVF and a positive QRS complex in lead I, the axis is extremely leftward, between –30° and –90°. Because there is an infarction pattern (QS morphology), however, this is not a left anterior fascicular block, which cannot be diagnosed in the presence of an inferior wall MI.

The R-wave voltage in the limb leads is small and is considered low (< 5 mm of amplitude in each limb lead). However, the QRS voltage in the precordial leads is increased, and the S-wave depth in lead V2 (↓) is 23 mm and the R-wave height in lead V4 (↑) is 32 mm. This meets one of the criteria for left ventricular hypertrophy (LVH) (S-wave depth + R-wave amplitude in any precordial lead ≥ 35 mm). In addition, there is a very tall R wave in lead V1 (←) (9 mm), which meets a criterion for right ventricular hypertrophy (RVH). There is also a tall P wave (^) in lead V1, which suggests right atrial hypertrophy, and T-wave abnormalities (*) in leads V2-V6, which are often seen in association with ventricular hypertrophy.

However, it should be noted that the ECG was recorded at double standard (↓) (ie, 1 mV = 20 mm or 20 small boxes in height). Hence the QRS voltage as measured needs to be reduced by half in all leads. Therefore, the QRS voltage in the precordial leads is normal (S-wave depth in lead V2 = 12 mm; R-wave amplitude in lead V4 = 16 mm; and R-wave amplitude in lead V1 = 4 mm), and LVH and RVH are not present. Despite the fact that the ECG was recorded at double standard, the QRS amplitude in the limb leads is low. If recorded at normal standard the QRS complexes would be barely visible. The limb leads reflect voltages in the frontal plane of the heart, whereas the precordial leads reflect voltages in the horizontal plane. Hence, there can be a marked difference in voltage amplitude between the limb leads and precordial leads.
The following routine ECGs are obtained from a 32-year-old athletic man with no cardiac history who is undergoing surgery for a torn...
What is the diagnosis?

anterior cruciate ligament. He reports being anxious and feeling his heart racing a bit.
Podrid’s Real-World ECGs

ECG 89A Analysis: ECG recorded at double speed
**ECG 89A** shows a regular rhythm at a rate of 50 bpm. There is a P wave (+) before each QRS complex, with a stable PR interval (↔) (0.32 sec). The P waves are positive in leads I and aVF, suggesting sinus bradycardia with a first-degree AV block.

The QRS complex duration is prolonged (ربع) (0.18 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals (🩹) are 600/550 msec, which are very long. Hence there is a bradycardia and the PR, QRS, and QT intervals are prolonged. Also noted is the fact that there are only six leads present. This ECG was recorded at double speed, or 50 mm/sec, rather than at the normal speed of 25 mm/sec. Hence the actual heart rate is 100 bpm, the PR interval is 0.16 second, the QRS complex duration is 0.09 second, and the QT interval is 0.34 second, or 0.43 second when corrected for heart rate (QTc). Thus the ECG is normal.

*continues*
Podrid’s Real-World ECGs

ECG 89B Analysis: ECG recorded at double speed
**ECG 89B** represents the precordial leads of **ECG 89A**, again recorded at 50 mm/sec. As before, the heart rate is twice as fast as measured and the intervals are half of what is measured. Thus the ECG is normal.
You are on call overnight in a hospital’s intensive care unit when you are STAT paged for a patient in the telemetry unit who was admitted for chest pain. As you run to the patient’s room, a nurse hands you the following ECG printed from the patient’s remote telemetry unit. When you enter the room, you are surprised to see the patient standing next to the bathroom sink, brushing her teeth. She is completely asymptomatic and feeling well. What is the diagnosis?
Podrid’s Real-World ECGs

ECG 90 Analysis: Sinus tachycardia, artifact
The ECG appears to show a rapid heart rate of 210 bpm and QRS complexes that are wide, bizarre, and show some variability in morphology, suggesting a sustained ventricular tachycardia. However, lead II shows regular and normal QRS complexes (†). There is a P wave (^) before each QRS complex, with a stable PR interval. The rate is 110 bpm, indicating a sinus tachycardia. Since the leads in each column are simultaneous, the presence of a normal rhythm and QRS complexes in lead II means that the same is present in leads I and III. With closer inspection of most of the leads, organized and narrow QRS complexes (↑) can be seen occurring at a regular interval (⊔) at a rate of 110 bpm. Therefore, this ECG shows sinus tachycardia with artifact being present in all leads except lead II. It is important to recognize the fact that normal narrow QRS complexes can be seen occurring at a regular interval throughout all of the leads. This artifact is likely due to patient motion, often repetitive motion such as tooth brushing.
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Podrid’s Real-World ECGs combines traditional case-based workbooks with a versatile Web-based program to offer students and physicians an indispensable resource for developing the technical skills and systematic approach needed to interpret ECGs with confidence.

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Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can.

—from the Foreword by Hein J. Wellens, MD
Podrid’s Real-World ECGs
A Master’s Approach to the Art and Practice of Clinical ECG Interpretation

Volume 2 Myocardial Abnormalities
Podrid’s Real-World ECGs
A Master’s Approach to the Art and Practice of Clinical ECG Interpretation

Volume 2  Myocardial Abnormalities

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy and daughter Sapna, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

Peter A. Noseworthy
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The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven’s landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiodiagnostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today’s cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled “Real-World ECGs,” by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient’s clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.

continues
These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important dividend for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, realworldECGs.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

Roman W. DeSanctis, MD
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Foreword

The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable “workhorse” in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

Hein J. Wellens, MD
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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician’s office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of Podrid’s Real-World ECGs aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in Podrid’s Real-World ECGs teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

Volume 1 provides an essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the
interpretation of all ECGs. Volume 2 discusses the diagnosis of hypertrophy of the atria and ventricles, acute myocardial ischemia, acute and chronic myocardial infarction, and pericarditis. The subsequent volumes focus on other disease entities for which the ECG is useful:

- Atioventricular (AV) and intraventricular conduction disturbances and enhanced AV conduction
- Sinus, atrial, junctional, and ventricular arrhythmias
- Narrow and wide complex tachycardias and forms of aberration
- Recording methods and miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs and clinical scenarios to further enhance the student's skills at ECG analysis. Importantly, each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: realworldECGs.com

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers easy access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q&A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

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Introduction
Myocardial Abnormalities

The surface electrocardiogram (ECG) is often used to diagnose abnormalities of the atrial and ventricular myocardium, including hypertrophy, ischemia, infarction, and inflammation (myocarditis/pericarditis).

Hypertrophy

Left Ventricular Hypertrophy

A number of criteria have been proposed for diagnosing left ventricular hypertrophy (LVH) on the surface ECG (FIGURE 1). An important limitation is related to factors that may influence the transmission of the electrical impulse to the surface of the body, including body habitus (especially obesity), pulmonary disease, and pericardial thickening or effusion. Therefore, the ECG may not accurately reflect the presence of LVH.

The following criteria have been proposed for diagnosing LVH:

- S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm (small boxes) if over age 45 or ≥ 45 mm if under age 45 (Sokolow-Lyon criterion)
- Deepest S wave + tallest R wave (in mm) in any two precordial leads ≥ 35 mm (or ≥ 45 mm if age under 45)
- S-wave depth or R-wave amplitude (in mm) in any one precordial lead ≥ 25 mm

Figure 1. Left ventricular hypertrophy.
Note the deep S wave in leads V1-V2 and the tall R wave in leads I and V5-V6 (which are due to left ventricular depolarization in these leads). The R wave may also be tall in the limb leads, especially lead aVL. The axis is often leftward. The arrows indicate the direction of ventricular activation. Left ventricular activation begins in the interventricular septum, going in a left-to-right direction. This is followed by left ventricular depolarization (apex, anterolateral, and high lateral wall) going in a right-to-left direction.
• **R-wave amplitude in lead aVL ≥ 11 mm (≥ 18 mm in presence of left axis deviation)** (Sokolow-Lyon criterion)
• **R-wave amplitude in any one limb lead ≥ 20 mm**
• **R-wave amplitude in lead aVL + S-wave depth in lead V3 ≥ 28 mm for men or ≥ 20 mm for women** (Cornell criteria)

The voltage criteria are based on the ECG recorded at normal standardization (*i.e.*, 1 mV = 10 mm or 10 small boxes in height). If the ECG is recorded at half-standard (*i.e.*, 1 mV = 5 mm or five small boxes), the measured QRS amplitude is half of the actual value and hence the number is doubled. If the ECG is recorded at double standard (*i.e.*, 1 mV = 20 mm or 20 small boxes), the measured QRS amplitude is twice normal and the number must be reduced by half.

LVH may be associated with other changes on the ECG as well, including:

• Intraventricular conduction delay (IVCD) due to slow activation of the thickened myocardium
• Delay or a slurring in the upstroke of the QRS complex. This is called delayed intrinsicoid deflection, which is measured from the beginning of the QRS complex to the peak of the R wave. An intrinsicoid deflection longer than 0.05 second is considered delayed.
• Physiologic left axis deviation, between 0° and –30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF)

• **Left atrial hypertrophy** (or abnormality), called a P mitrale, defined as a P wave that is broad (> 0.12 sec in duration) and notched (with a tall second component). In lead V1, and often in lead V2, the P wave is entirely negative (rather than biphasic).
• **Associated ST-T wave abnormalities** (*i.e.*, J-point and ST-segment depression and/or T-wave inversions), which represent repolarization abnormalities, most likely due to subendocardial ischemia. These abnormalities are most often seen leads I, aVL, and V4-V6. The ST-T wave changes often seen with LVH have been called a “strain pattern.” However, they actually reflect repolarization abnormalities as a result of subendocardial ischemia. The last part of the myocardium to receive blood supply (or oxygen supply) is the subendocardium. When LVH is present, the blood supply to the subendocardium may be limited and reduced; hence it is chronically ischemic.
• **J-point and ST-segment elevation**, termed early repolarization, which is most often seen in leads V4-V6. This may be seen when the QRS complex voltage is increased, even if LVH is not present.

As indicated, QRS amplitude is affected by a number of conditions; therefore, LVH may be present even if QRS amplitude criteria are not met but other associated changes are seen. This is the basis for the Romhilt-Estes scoring system, which assigns a point score to various ECG abnormalities seen with LVH.
Romhilt-Estes Scoring System

Romhilt-Estes Criterion Points
R-wave height or S-wave depth in any limb lead ≥ 20 mm 3
OR
S-wave depth in lead V1 or V2 ≥ 30 mm
OR
R-wave height in lead V5 or V6 ≥ 30 mm

ST-T wave changes typical of LVH
Taking digoxin 1
Not taking digoxin 3

Left atrial hypertrophy
(terminal force in lead V1 ≥ 1 mm in depth and > 0.04 sec in duration) 3

Left axis deviation (< -30°) 2
QRS duration ≥ 90 ms (ie, IVCD) 1
Intrinsicoid deflection in lead V5 or V6 > 0.05 sec 1

A point score of ≥ 5 indicates definite LVH; a point score of 4 is probable LVH.

Right Ventricular Hypertrophy

The mass of the left ventricular myocardium is far greater than that of the right ventricle. Hence the QRS complex primarily reflects depolarization of the left ventricular myocardium. Since depolarization occurs simultaneously in the left and right ventricles, manifestations of right ventricular depolarization are not usually seen. However, evidence of right ventricular depolarization may be seen when there is a significant increase in right ventricular myocardial mass (ie, right ventricular hypertrophy, RVH). The diagnosis of RVH can be established on the surface ECG, although as with LVH, the absence of evidence of RVH on the surface ECG does not exclude its presence (FIGURE 2).

The criteria for RVH include:
- R-wave amplitude (in mm) in lead V1 > 7 mm
- R/S ratio in lead V1 > 1
- S/R ratio in lead V6 (or V5) > 1

Figure 2. Right ventricular hypertrophy.
Note the tall R wave in leads V1-V2 (this is due to right ventricular depolarization, which is augmented by the RVH) and the deep S wave in leads I and V5-V6 (this is due to the last part of ventricular activation, which is directed from left to right as a result of RVH). The axis is often rightward. The arrows indicate the direction of ventricular activation. Left ventricular activation begins in the interventricular septum, which goes in a left-to-right direction. This is followed by early activation of the left ventricle going from right to left. However, as a result of RVH, the terminal portion of ventricular activation goes from left to right.
Other ECG findings may help confirm the diagnosis:

- Right axis deviation (≥ +90°)
- Right atrial hypertrophy (or abnormality), termed P pulmonale. This is identified by a P wave that is narrow, tall, and peaked in the limb leads (> 2.5 mm of height in lead II) and primarily positive in lead V1 (and often V2).
- Associated ST-T wave abnormalities in leads V1-V3. As with LVH, ST-T wave abnormalities represent chronic ischemia of the subendocardial layer of the right ventricle.

However, other causes for a tall R wave in lead V1 need to be considered and excluded before diagnosing RVH. These include a posterior wall myocardial infarction (MI, usually associated with an inferior wall MI), Wolff-Parkinson-White pattern (short PR interval and widened QRS-delta wave), hypertrophic cardiomyopathy (septal hypertrophy with prominent septal Q waves in other leads), early transition (counterclockwise rotation), Duchenne muscular dystrophy (posterolateral infarction pattern), dextrocardia (reverse R-wave progression in leads V1-V6, right axis deviation, negative P wave in lead I), lead switch (leads V1, V2, V3), recording of right-sided leads (reverse R-wave progression in leads V1-V6), and a normal variant.

**Normal P Wave**

The P wave reflects depolarization of the right followed by the left atrium (FIGURES 3, 4, AND 5). When the impulse is generated by the sinus node, the direction of transmission is from right to left and from the arms to the legs (downward direction), and the normal P wave is positive (upright) in leads I, II, aVF, and V5-V6 and negative in lead aVR. There is often a slight notch, reflecting right followed by slightly delayed left atrial depolarization. The P-wave duration is 0.12 second or less, and the amplitude is usually 0.25 mV (2.5 small boxes) or less. The P wave is often biphasic (positive–negative) in lead V1 (FIGURE 4). Since lead V1 sits over the right ventricle and right atrium, the initial portion is positive, reflecting right atrial depolarization going toward lead V1 (positive) and then left atrial depolarization going away from lead V1 (negative).

**Left Atrial Hypertrophy/Abnormality**

When left atrial hypertrophy (or a left atrial conduction abnormality) is present, the P wave is broad (≥ 0.12 sec) with prominent notching, reflecting depolarization of the right atrium followed by delayed depolarization of the hypertrophied left atrium. The left atrial waveform (second component of the P wave) has an increased amplitude,
Right Atrial Hypertrophy/Abnormality

With right atrial hypertrophy, depolarization of the hypertrophied right atrium is delayed, occurring simultaneously with left atrial depolarization. The superimposed atrial waveforms result in a P wave that is narrow, tall, and peaked as the right atrial waveform has an increased amplitude, reflecting the hypertrophy (FIGURES 4 AND 5). This has been termed P pulmonale. The etiology is either right-sided valvular disease or pulmonary artery hypertension (primary or secondary from lung disease or left ventricular abnormalities).

Also seen with right atrial hypertrophy is a P wave in lead V1 that is predominantly positive (due to a right atrial impulse that is directed toward lead V1) (FIGURE 4); this P-wave morphology may also be seen in lead V2.

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reflecting the hypertrophy (FIGURES 3 AND 4). This has been termed P mitrale. It is seen in conditions that cause left atrial hypertrophy, such as systemic hypertension, aortic stenosis, or mitral valve disease (especially mitral stenosis).

Also seen with left atrial hypertrophy is a P wave in lead V1 that is predominantly negative (ie, left atrial forces going away from lead V1) (FIGURE 4); a prominent negative component of the P wave may be seen also in leads V2-V3.

<table>
<thead>
<tr>
<th></th>
<th>Lead II</th>
<th>Lead V1</th>
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<tbody>
<tr>
<td><strong>Normal</strong></td>
<td>![Diagram]( normally P wave )</td>
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<tr>
<td><strong>Right atrial hypertrophy or abnormality</strong></td>
<td>![Diagram]( right atrial P wave )</td>
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<td><strong>Left atrial hypertrophy or abnormality</strong></td>
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**Figure 4.** Appearance of the P wave in leads II and V1 in right and left atrial hypertrophy. The normal P wave in lead V1 is biphasic as the initial force (depolarization) is from the right atrium, going toward lead V1 (and hence a positive deflection), while left atrial depolarization is slightly later, going away from lead V1 and hence generating a negative waveform. With right atrial hypertrophy or abnormality, the depolarization is primarily toward lead V1, thereby generating a positive waveform. With left atrial hypertrophy or abnormality, the depolarization is primarily away from V1, thereby generating a negative waveform.

**Figure 5.** The P wave in right atrial hypertrophy (or abnormality) is tall, narrow, and peaked. This is due to delayed activation of the right atrium and activation is superimposed on left atrial activation. As there is increased muscle mass, the amplitude of the right atrial waveform is also increased. This is termed P pulmonale.
Low QRS Complex Voltage

Low QRS complex voltage is defined as a QRS amplitude ≤ 5 mm in each limb lead and/or ≤ 10 mm in each precordial lead. The low voltage reflects reduced electrical activity measured at the surface of the body and may be due to loss of myocardial muscle mass, pericardial effusion, thickened pericardium, lung disease, or obesity.

ST-segment Changes

The normal ST segment is slightly concave and is at baseline, which is always the TP segment. ST-segment flattening is a nonspecific change. ST-segment depression or ST-segment elevation is of importance.

J-point and ST-segment depression (up sloping, horizontal, downsloping) are due to ischemia of the subendocardium, which is the first part of the myocardium to become ischemic. There are a number of causes of J-point and ST-segment elevation:

- **Transmural ischemia:** ST-segment elevation in the absence of Q waves; in the presence of Q waves (indicating a prior transmural MI), ST-segment elevation most likely indicates a wall motion abnormality
- **Acute MI:** Localized ST-segment elevation; reciprocal ST-segment depressions may be seen in other leads (ie, the ST-segment change viewed from another direction); hyperacute T waves (tall, peaked, and symmetric) are often present
- **Aneurysmal segment:** Persistent ST-segment elevation after an MI
- **Pericarditis:** Diffuse concave ST-segment elevation in all or most leads; there are no reciprocal ST-segment changes and the T waves are normal (asymmetric)
- **Early repolarization:** Most common in left precordial leads V4-V6; early repolarization is seen in healthy individuals as a normal variant, with LVH, or when there is high QRS voltage; ST segments are concave and T waves have normal morphology.

Mechanism for ST-segment Changes

The ST-segment depression associated with subendocardial ischemia is actually caused by elevation of the baseline TP segment, which is the baseline or isoelectric plane. Under normal situations, this segment is at zero potential as it represents a period of time between QRS complexes when there is no movement of electrical current. With subendocardial ischemia, there is a loss of membrane integrity and leakage of potassium from the cell to the extracellular region, moving down its concentration gradient. As a result, the resting membrane potential becomes less negative, or more positive. The remaining portion of the myocardium is normal, with a resting membrane potential of ~90 mV. Since current travels from positive to negative voltage, the ischemic region generates electrical currents that travel away from this region and toward the overlying normal epicardial myocardium. The net effect is that there are positive currents directed toward the overlying electrode, which will result in elevation of the baseline or TP segment. With depolarization at the time of the QRS complex generation, the entire myocardium becomes depolarized, and hence the ST segment is at zero potential or voltage. When compared with the elevated TP segment, it appears that there is ST-segment depression.

When transmural ischemia is present, as occurs with a Q-wave MI, there is a similar loss of membrane integrity and leakage of potassium ions from the cell to the extracellular region, moving down its
concentration gradient. As a result, the resting membrane potential becomes less negative, or more positive, in the injured tissue. The adjacent normal myocardium has a normal resting membrane potential of \(-90\) mV. Since current travels from positive to negative voltage, the ischemic region generates positive currents that travel away from the entire ischemic region toward the normal myocardium. Hence the electrical current travels away from the overlying electrode. In this situation the baseline voltage, and hence the TP segment, will be more negative (\textit{ie}, there will be depression of the TP segment) in the leads overlying the injured myocardium. With depolarization at the time of the QRS complex, the entire myocardium becomes depolarized, and hence the ST segment is at zero potential or voltage. When compared with the depressed TP segment, it appears that there is ST-segment elevation.

**Myocardial Ischemia**

Myocardial ischemia (regardless of etiology) is diagnosed by the presence of J-point and ST-segment depression, which reflects subendocardial ischemia. Since myocardial blood flow is from epicardium to endocardium, the subendocardium is the last layer to receive blood. As a result, it is the first part of the myocardium to become ischemic when there is an increased oxygen demand or reduced oxygen supply. The ST segment is compared with the baseline or 0 potential on the ECG (\textit{ie}, TP segment). If the TP segment is not obvious (as may happen with a sinus tachycardia where the T wave and P wave merge), the PR segment can be used. Three types of ST-segment depression may be seen (FIGURE 6):

- **Upsloping ST-segment depression**. Upsloping ST-segment depression is the least specific for ischemia as it may be a normal finding during sinus tachycardia. It should be remembered that atrial repolarization (\textit{ie}, the T wave of the P wave) occurs during the QRS complex and hence is normally not seen. However, when the sinus rate increases the PR segment shortens (due to sympathetic enhancement of conduction through the atrioventricular [AV] node) and this causes the T wave of the P wave to move out from the QRS complex and become superimposed on the J point, causing both the J point and ST segment to be depressed. The ST segment, therefore, slopes upward to return to baseline. Hence when upsloping ST-segment depression is present, it is standard practice to evaluate the degree of ST-segment depression present.

![Figure 6. Types of ST segment shifts.](image-url)

- **Normal ST segment**.
- **J-point depression**.
- **Upsloping ST-segment depression (≥1.5 mm at 80 msec)**.
- **Horizontal ST-segment depression (≥1 mm)**.
- **Downsloping ST-segment depression (≥1 mm)**.
- **ST-segment elevation (≥1 mm)**.

**Figure 6. Types of ST segment shifts.** (A) Normal ST segment. The normal J point and ST segment are at baseline, which is established by the TP segment (\textit{ie}, from the T wave of the preceding complex to the P wave). (B) A depressed J point. (C) Upsloping ST-segment depression; the J point is depressed and the ST segment slopes upward toward baseline. (D) Horizontal ST-segment depression; the J point is depressed and the ST segment is flat or horizontal. (E) Downsloping ST-segment depression; the J point is depressed and the ST segment slopes downward. (F) ST-segment elevation; the J point and ST segment are above the baseline TP segment.
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at 80 msec (two small boxes) past the J point, which eliminates any effect from the atrial T wave on the J point and ST segment. If the ST segment at 80 msec past the J point is still depressed 1.5 mm or more below baseline (the TP segment or, if not obvious, the PR segment), then subendocardial ischemia is likely present.

- **Horizontal ST-segment depression.** The J point and ST segment are depressed (≥ 1 mm below baseline) and are flat thereafter.
- **Downsloping ST-segment depression.** The J point is depressed below baseline (≥ 1 mm), and the ST segment slopes downward.

This is the most specific pattern for ischemia.

Although the usual criterion for ischemia is ST-segment depression more than 1 mm below baseline, specificity for ischemia increases as the degree of ST-segment depression increases. However, sensitivity for the diagnosis of ischemia decreases. In addition, specificity increases with an increased number of leads showing ST-segment depression. The location of ischemia based on the leads that show ST-segment depression does not always identify the region of subendocardium involved.

Myocardial Infarction

**Acute Infarction**

Acute infarction is the result of a total cessation of blood flow and oxygen supply to the myocardium. This causes loss of membrane integrity and loss of the normal sodium–potassium–ATPase pump, which is energy dependent and requires an oxygen supply. Potassium leaks out of the cell, based on its electrochemical gradient (intracellular potassium levels are normally higher than extracellular levels as maintained by this ATPase pump). Since there is no blood flow into or out of the region of infarction, potassium remains in this area, producing local hyperkalemia.

Therefore, the earliest change with a transmural or ST-segment elevation MI (STEMI) is a hyperacute (tall, peaked, and symmetric) T wave seen in the area of the involved myocardium (FIGURE 7). The hyperacute T wave occurs even before there are any ST-segment changes (if the ECG is obtained shortly after the onset of symptoms). Thereafter, the ST segment begins to elevate, maintaining its normal concave morphology; hyperacute T waves are still present. The ST segment elevates farther and is still concave; hyperacute T waves are also still present. ST segments continue to elevate and become convex, merging with the T wave. The amplitude of the R wave decreases. When the ST segment and T waves merge and the R wave is no longer obvious, the complex has the morphology of a “current of injury.” It resembles the fast, sodium ion–mediated action potential. This ECG pattern of an acute infarction has been referred to as “tombstoning,” as the QRS complex looks like a tombstone. The ST segment begins to return to baseline, Q waves develop, and T waves begin to invert, resulting in a chronic infarct pattern (ie, Q-wave and T-wave inversion). In addition to ST-segment elevation, there are reciprocal changes (ie, ST-segment depressions in other leads). This actually represents the ST-segment elevation observed from another angle. Persistent ST-segment elevation, present weeks to years after an acute event, indicates the presence of an aneurysm in the area of the previous infarction.

The location of these changes identifies the region of the myocardium involved; it may not identify which coronary artery is the “culprit”:
Inferior wall MI: ST-segment elevation in leads II, III, and aVF. The right ventricle is often involved in an inferior wall MI; this is suggested by the presence of ST-segment elevation in lead V1 and ST-segment depression in lead aVR (which actually represents ST-segment elevation) and is confirmed by obtaining right-sided leads and seeing ST-segment elevation in right-sided leads V3R-V4R. There may also be involvement of the posterior wall, which is suggested by the presence of ST-segment depression in leads V1-V2. Posterior leads placed on the back below the left scapula (V7-V8) showing ST-segment elevation help support the diagnosis of a posterior wall MI.

Anteroseptal MI: ST-segment elevation in leads V1-V2

Anteroapical MI: ST-segment elevation in leads V3-V4

Anterolateral MI: ST-segment elevation in leads V5-V6

Anterior wall MI: ST-segment elevation in two or more contiguous leads across the precordium (ie, leads V1-V6)

Lateral MI: ST-segment elevation in leads I and aVL

Chronic Infarction

A chronic or old MI is identified by the presence of abnormal Q waves, defined as any Q wave in leads V1-V3 or a Q wave in leads I, II, aVF, or V4-V6 (in two or more contiguous leads) that is 0.04 second or longer.
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in duration (or, as per new guidelines ≥ 0.03 sec, which is difficult to measure) and at least 1 mm in depth. However, Q waves may be normal and are ignored in lead III (unless they are also in leads II and aVF), in lead V1 (unless also in lead V2), and in lead aVL (unless Q-wave depth ≥ 50% R-wave height). A QS complex in leads V1-V2 may also be normal variant, especially in women.

T-wave inversions are usually present in the leads showing Q waves. ST-segment elevation that persists for more than a few weeks after the acute infarction suggests an aneurysm of the involved wall. The location of Q waves identifies the region of the left ventricle that is involved, but not necessarily the vessel that is occluded:

- **Inferior wall MI**: Q waves in leads II, III, and aVF
- **Anteroseptal MI**: Q waves in leads V1-V2
- **Anterolateral MI**: Q waves in leads V3-V4
- **Anterolateral MI**: Q waves leads V5-V6
- **Anterior wall MI**: Q waves in two or more contiguous precordial leads
- **Lateral MI**: Q waves in leads I and aVL
- **Posterior MI**: Tall R wave in lead V1 (R-wave amplitude > 7 mm or R/S > 1) with a duration of 0.04 second or longer (or ≥ 0.03 sec). This is typically seen in association with inferior wall MI, although there may be a true posterior wall MI in which there is no inferior wall involvement. However, other abnormalities associated with a tall R wave in lead V1 need to be considered, including evidence of RVH (usually associated with a right axis deviation and right atrial hypertrophy), Wolff-Parkinson-White pattern (short PR interval and a wide and abnormal QRS complex with a delta wave), dextrocardia (reverse R-wave progression across the precordium with a right axis deviation and negative P waves in leads I and aVL), lead misplacement (V1, V2, V3), recording of right-sided leads (reverse R-wave progression in leads V1-V6), Duchenne muscular dystrophy (a posterolateral infarction pattern), or hypertrophic cardiomyopathy (prominent septal Q waves). On occasion it may be a normal variant or due to counterclockwise rotation (ie, early transition).

**Pericarditis/Myocarditis**

Pericarditis, which is often associated with myocardial inflammation or myocarditis of the epicardium, causes diffuse inflammation and diffuse ECG changes reflecting involvement of all or most of the heart. The characteristic ECG findings with pericarditis include:

- Diffuse ST-segment elevation is seen in all or almost all leads. ST segments have normal concave morphology regardless of the height of ST-segment elevation. There is no change in the ST-segment morphology over time (ie, there are no evolutionary changes, as are seen with an acute MI).
- There is no reciprocal ST-segment depression.
- T waves are normal (ie, asymmetric).
- PR depression may be seen. However, its absence does not exclude pericarditis.
- T-wave inversion may occur after ST segments return to isoelectric baseline.
Core ECGs
A 29-year-old pregnant woman is admitted to the hospital in early labor. She reports fleeting palpitations during the weeks prior to presentation. She is resting comfortably between contractions.

What does her ECG show?
What is the likely diagnosis?
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ECG 1 Analysis: Normal sinus rhythm, normal ECG
There is a regular rhythm at a rate of 66 bpm. There is a P wave (\(\ast\)) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. They have a normal morphology. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/420 msec). The QRS complex morphology is normal, and there is normal R-wave progression across the precordium. The ST segments are normal in morphology (concave) and are at baseline (the TP segment). The T waves have a normal morphology (\(\text{ie,}\) they are asymmetric and the upstroke is slower than the downstroke). A U wave (^) can be seen after the T wave in leads V1-V3. The U wave is believed to represent delayed repolarization of the His-Purkinje system and hence is usually more prominent in the right precordial leads. This is a normal ECG.

It should be noted that very prominent U waves seen across the precordium can be a sign of hypokalemia. In the context of her palpitations, it would be reasonable to check this woman’s electrolytes because hypokalemia causes increased myocardial excitability and could precipitate arrhythmia.
A 55-year-old man presents for an insurance physical examination before starting a new job. He feels well and has no complaints. On exam, his blood pressure is 165/90 mm Hg in both arms. His cardiac exam is notable for an S4 and a sustained but nondisplaced apical impulse.

What does his ECG show?
What is the likely diagnosis?
ECG 2 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), left ventricular hypertrophy (LVH)
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant (0.20 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

However, the P wave is very broad (0.16 sec), with an increased amplitude (0.3 mV) and prominent notching (+), seen best in leads II, III, aVF, and V3-V5. The second component of the P wave has a greater amplitude than the first component. The P-wave morphology has been termed P mitrale. The P wave is completely negative in lead V1 (^). These patterns are characteristic of left atrial hypertrophy (or left atrial abnormality). Although this is often termed left atrial enlargement, it should be remembered that the waveforms on the ECG represent myocardial muscle depolarization and repolarization and hence reflect muscle mass or intraatrial conduction and not chamber size. Left atrial hypertrophy is the result of systemic hypertension, other causes for restrictive left ventricular cardiomyopathy, aortic stenosis, or mitral valve disease.

The QRS complexes are of normal duration (0.08 sec) and have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). However, the QRS amplitude (voltage) is markedly increased, especially in leads V4-V6, with an R wave 30 mm in height (\( \uparrow \)). The S-wave depth (voltage) in lead V3 is almost 25 mm (\( \downarrow \)). The sum of these two QRS voltages (S-wave depth in lead V3 + R-wave amplitude in lead V4 = 55 mm) meets one of the criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth in any precordial lead + R-wave amplitude in any precordial lead ≥ 35 mm). Associated with the LVH are ST-segment changes, particularly seen in leads II, III, aVF, and V6 (\( \uparrow \)). LVH is also present based on the Romhilt-Estes criteria (QRS amplitude, left atrial hypertrophy, and ST-T wave changes [score = 9]). The QT/QTc intervals are normal (420/420 msec).

It is likely that this patient has longstanding hypertension. The increased arterial afterload resulting from the systemic hypertension causes an increase in systolic pressure that is generated by the left ventricle to overcome this increased resistance. This results in LVH. When LVH develops, there is also left ventricular diastolic dysfunction and the left atrium generates a higher pressure to fill the “stiff or rigid” left ventricle. This results in left atrial hypertrophy.

Treatment of hypertension often results in regression of LVH as well as improvement in diastolic dysfunction and left atrial hypertrophy (abnormality).
A 65-year-old woman presents to her primary care physician with exertional chest discomfort and shortness of breath while climbing the stairs to her second-floor apartment. The discomfort is becoming progressively worse and occurring with less effort. She never experiences the discomfort at rest. On exam her blood pressure is 126/72 mm Hg. She has a grade III/VI systolic crescendo–decrescendo murmur at the upper sternum (ie, base of the heart) that radiates to the clavicles, and a second murmur with the same timing is audible at the apex.

What does her ECG show?
What is the likely diagnosis?
What further evaluation should be performed?
What therapy would you recommend?
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ECG 3 Analysis: Sinus bradycardia, left atrial hypertrophy, left ventricular hypertrophy (LVH), early repolarization
There is a regular rhythm at a rate of 48 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is upright in leads II, III, aVF, and V4-V6 and negative in lead aVL. This indicates a sinus bradycardia.

The P waves are broad (0.14 sec) and have an increased amplitude (0.35 mV). There is significant notching of the P wave (+), especially prominent in leads II and V4-V6. The second component of the P wave has a greater amplitude than the first. In addition, the P wave in lead V1 (▲) is predominantly negative and there is a significant negative component seen in leads V2-V3 (^). This is characteristic of left atrial hypertrophy (abnormality). The prominently notched P waves are termed P mitrale.

The QRS complex has a normal duration (0.08 sec) and a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (480/430 msec). The amplitude (voltage) of the QRS complex is markedly increased, especially in leads V4-V5 (48 mm) ( ], and the S-wave depth (voltage) in lead V3 is very increased (27 mm) ( ] ); hence the S-wave depth in lead V3 and the R-wave amplitude in lead V5 is 75 mm, which meets one of the voltage criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth + R-wave amplitude in any two precordial leads ≥ 35 mm). Also noted is ST-segment elevation in leads V2-V4, which is often seen with LVH and is termed early repolarization (↓). In addition, there are significant ST-T wave abnormalities (↑) seen primarily in leads I, aVL, and V4-V6. The Romhilt-Estes criteria for LVH are also met (QRS voltage, ST-T wave abnormalities, and left atrial hypertrophy; score = 9).

This patient has a murmur that suggests significant aortic stenosis (ie, crescendo–decrescendo systolic) at the base (upper sternum) that radiates to the clavicles. The murmur often radiates to the apex, known as the Gallavardin effect. This abnormality was confirmed on echocardiography, which showed severe calcific aortic stenosis (aortic valve area 0.9 cm²) and mild aortic regurgitation. Mild to moderate aortic regurgitation is often associated with aortic stenosis as a result of inadequate closure of the stenotic leaflets as well as post-stenotic dilation of the aortic root, which results in the high-pressure jet of blood. The presence of concentric LVH was confirmed on echocardiography (left ventricular wall thickness = 14 mm).

continues
As a result of the stenotic aortic valve, there is increased resistance to blood flow as the left ventricle contracts. Hence more pressure is generated by the left ventricle to force blood past the stenotic valve. In order to generate greater pressure and maintain normal wall stress, the left ventricular muscle becomes hypertrophic. As the severity of aortic stenosis increases, the degree of LVH increases. The degree of hypertrophy may increase to a point that the blood and oxygen supply to the myocardium is inadequate, resulting in chronic subendocardial ischemia, which manifests as ST-T wave changes. This may be associated with anginal symptoms. Thus, angina may be seen with LVH and subendocardial ischemia even in the absence of epicardial coronary artery disease and represents a reduction in myocardial reserve from a relative reduction in blood and oxygen supply to the hypertrophied myocardium.

As a result of severe aortic stenosis associated with symptoms, this patient underwent cardiac catheterization, which is usually performed in those with known severe aortic stenosis to evaluate the coronary arteries. The valve area was 0.7 cm², confirming the results of the echocardiogram, and there was no coronary artery disease. She underwent aortic valve replacement, which is the only definitive therapy for aortic stenosis. There is often regression of LVH and diastolic dysfunction after aortic valve replacement.
A 78-year-old black man is referred to a cardiologist for progressive dyspnea on exertion, orthostatic hypotension, and fatigue. On physical examination, his blood pressure is 88/52 mm Hg, heart sounds are regular, and there is a soft S3 but no murmurs. Chest auscultation reveals reduced breath sounds. There is significant neck vein distension and 3+ peripheral edema to the mid-calf.

What does his ECG show?
What is the likely diagnosis?
Is any further evaluation necessary?
ECG 4 Analysis: Normal sinus rhythm, first-degree AV block, left atrial hypertrophy (abnormality), left anterior fascicular block, low voltage in limb leads, clockwise rotation (late transition)
There is a regular rhythm at a rate of 98 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.22 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm with a first-degree AV block. The P wave is broad (0.16 sec) with some notching seen in leads II and aVF (*). The P wave is significantly negative in leads V1-V2 (▲). These features are characteristic of left atrial hypertrophy or a left atrial abnormality.

The QRS complexes have a normal duration (0.10 sec). However, low amplitude is present in the limb leads (defined as a QRS complex amplitude [voltage] ≤ 5 mm in each limb lead). The axis is extremely leftward in the frontal axis, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). As there is no evidence of an inferior wall myocardial infarction (Q waves in leads II, III, and aVF), which can result in an extreme left axis deviation, the left axis deviation in this case is the result of a left anterior fascicular block. The QT/QTc intervals are prolonged (360/460 msec) but are actually normal when corrected for the slightly widened QRS complex duration (340/430 msec). There is poor R-wave progression in leads V1-V5 with late transition (ie, R/S > 1 in lead V6). This is termed clockwise rotation, which reflects the horizontal axis of the heart. This is determined by imagining the heart as viewed from under the diaphragm. With clockwise rotation the left ventricular electrical forces are shifted backward and hence develop late in the lateral precordial leads, accounting for the poor R-wave progression and late transition. With counterclockwise rotation, the electrical forces from the left ventricle are shifted forward and hence they develop earlier in the precordial leads (ie, a tall R wave in lead V2).

The presence of low voltage in the limb leads and significant left atrial hypertrophy in a patient who presents with signs of a low cardiac output and evidence of right-sided volume overload should prompt an evaluation for constriction or a restrictive cardiomyopathy, which may be due to an infiltrative disease. In this age group and in a black man, amyloid cardiomyopathy is of particular concern.
A 38-year-old woman with a history of Raynaud’s disease presents to her primary care physician with progressive decline in exercise tolerance. Over the past 6 months, she has gone from being able to participate in aerobics twice per week to having dyspnea with walking a single flight of stairs. Her examination is notable for a loud P2, subtle right precordial heave, and trace pedal edema.

What does her ECG show?
What is the likely diagnosis?
What further evaluation would be useful?
Podrid’s Real-World ECGs

ECG 5 Analysis: Sinus tachycardia, right atrial hypertrophy (abnormality), right axis deviation, right ventricular hypertrophy (RVH)
There is a regular rhythm at a rate of 140 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a sinus tachycardia. The P waves are tall (0.4 mV), peaked, and narrow (0.10 sec), particularly obvious in leads II and aVF (*). The P wave is also primarily positive and tall and peaked in lead V1 (^). This is characteristic of right atrial hypertrophy (or abnormality) and has been termed P pulmonale.

The QRS complex duration is normal (0.08 sec), and the axis is rightward, between +90° and +180° (QRS complex is negative in lead I and positive in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include right ventricular hypertrophy (RVH; associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (a deep Q wave in leads I and aVL), right–left arm lead switch (associated with negative P waves and T waves in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation).

There is a tall R wave (7 mm) and no S wave in lead V1 (→) and an S/R > 1 in leads V5-V6. This is characteristic of RVH, which is the cause for the right axis deviation. Associated with RVH are ST-T wave changes seen primarily in leads V1-V2 (↑), which reflect subendocardial ischemia of the thick right ventricular myocardium. The QT/QTc intervals are normal (280/430 msec).

continues
The patient’s physical exam and ECG findings are consistent with RVH and right atrial hypertrophy, likely resulting from severe pulmonary hypertension (PH). Progressive exercise intolerance and dyspnea on exertion are typical of PH. In this case, the condition may be idiopathic (primary) or related to an unrecognized collagen vascular disease such as systemic lupus erythematosus (which may also explain the Raynaud’s phenomenon), although the differential diagnosis is broad. Other potential causes of PH include left ventricular dysfunction (causing pulmonary venous hypertension); mitral stenosis; diseases associated with chronic hypoxia (which cause PH by hypoxic vasoconstriction), such as interstitial lung disease, sleep apnea, or chronic obstructive pulmonary disease; chronic thromboembolic disease; or mechanical obstruction of the pulmonary vasculature (by infiltrative or scarring lung diseases). Based on the physician’s relative suspicion for each of these underlying causes, testing includes a chest X-ray, echocardiogram, pulmonary function tests, overnight sleep oximetry, ventilation/perfusion (V/Q) scan, or computed tomography (CT) pulmonary angiogram.

Right heart catheterization is necessary for the diagnosis of PH. A mean pulmonary arterial pressure higher than 25 mm Hg is diagnostic, although pressures may be much higher in this symptomatic patient. At the time of right heart catheterization, a pulmonary capillary wedge pressure (PCWP) can be measured to exclude left-sided heart failure as a cause (ie, a normal PCWP). Furthermore, inhaled nitric oxide (or other vasodilator such as epoprostenol) may be administered at the time of catheterization to assess potential responsiveness to vasodilating agents.
A 54-year-old black man presents for evaluation of multiple episodes of syncope and palpitations. A 24-hour Holter monitor shows evidence of both supraventricular tachycardia and brief, nonsustained ventricular tachycardia. A chest X-ray shows a normal cardiac silhouette with prominent hilar lymphadenopathy.

What does his ECG show?
What is the likely diagnosis?
**Podrid’s Real-World ECGs**

**ECG 6 Analysis:** Sinus tachycardia, right atrial hypertrophy (abnormality), left atrial hypertrophy (abnormality), biatrial hypertrophy (abnormality)
The rhythm is regular at a rate of 100 bpm. There is a P wave in front of each QRS complex (*), and the PR interval is stable (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a sinus tachycardia. The P waves are narrow (0.10 sec), tall (0.5 mV), and peaked in leads II, III, and aVF (+). This is characteristic of right atrial hypertrophy (abnormality) or P pulmonale. In addition, the P wave is very negative in leads V1-V2 (▲), which is characteristic of left atrial hypertrophy (abnormality). Hence biatrial hypertrophy (abnormality) is present.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal, and the QT/QTc intervals are normal (320/410 msec).

Hilar lymphadenopathy and ventricular tachycardia in this patient are possibly due to sarcoid granulomas involving the lungs and myocardium. After pulmonary disease, cardiac involvement is the second most common presentation of sarcoidosis and ventricular tachycardia is a common cause of death. Ventricular tachycardia can arise either from increased automaticity of the affected myocardium or reentry within the sarcoid granuloma. Because the clinical symptoms of cardiac sarcoid are nonspecific, the diagnosis is often difficult to make. A tissue diagnosis can only be made by endomyocardial biopsy, but involvement is often patchy, making the yield of biopsy small.

Treatment of cardiac sarcoidosis, if present, is challenging. The response to systemic glucocorticoids is variable. Endocardial radio-frequency ablation of sarcoid granulomas can reduce the burden of ventricular tachycardia, although the disease often progresses to other regions of myocardium and the efficacy of ablation tends to be short lived. Patients with complete (third-degree) heart block may require permanent placement of a pacemaker. Patients deemed to be at high risk for a serious life-threatening ventricular tachyarrhythmia often receive an implantable cardioverter–debrillator, which can be useful for the prevention of sudden death.
A 36-year-old man presents to the emergency department with complaints of chest pain after cocaine use. Upon arrival, his pain has resolved and he is hemodynamically stable. On examination, his blood pressure is 180/96 mm Hg, his heartbeat is regular, and there is an audible S4.

What does his ECG show?
What is the likely diagnosis?
Is any additional evaluation or therapy necessary?
**Podrid’s Real-World ECGs**

**ECG 7 Analysis:** Normal sinus rhythm, first-degree AV block, left atrial hypertrophy (abnormality), left ventricular hypertrophy (LVH) with ST-T wave abnormalities
There is a regular rhythm at a rate of 68 bpm. There is a P wave (*) in front of each QRS complex, with a stable PR interval (0.24 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm with a first-degree AV block. The P wave is inverted in lead V1 (+) and biphasic in lead V2 (^); this is suggestive of left atrial hypertrophy (abnormality).

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/400 msec). The amplitude (voltage) of the QRS complex is increased, with an S-wave depth in lead V2 of 32 mm (\[\]) and an R-wave amplitude in lead V5 of 16 mm (\[\]). The S-wave depth in lead V2 + R-wave amplitude in lead V5 = 47 mm, which meets one of the criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm in patients ≥ 45 years of age or ≥ 45 mm in patients < 45 years of age). Although this voltage may be seen in patients who are younger than 45 years of age, there are also ST-T wave changes noted in leads I, aVL, and V4-V6 (\[\]), which are associated with LVH and represent repolarization abnormalities resulting from subendocardial ischemia. The presence of a tall QRS amplitude, ST-T wave abnormalities, and left atrial hypertrophy meet the Romhilt-Estes criteria for LVH (score = 9). There is slight J-point and ST-segment elevation in leads V1-V2, which is early repolarization, commonly seen with LVH or a tall QRS complex amplitude.

Since the patient’s pain has resolved and he does not have any acute ischemic changes on ECG, an acute coronary syndrome seems unlikely. Nonetheless, cocaine use is known to cause vasospasm, accelerated atherosclerosis, hypertension, aortic dissection, and cardiomyopathy, all of which must be considered by the treating physician.

Most striking on this ECG is the LVH in this young patient. It is likely that he has longstanding hypertension, perhaps caused or exacerbated by his cocaine use. If this patient stops using cocaine, his LVH may resolve. If hypertension and LVH persist despite abstinence from cocaine, the clinician should investigate for other causes of hypertension.
A 59-year-old woman presents to the emergency department with complaints of indigestion. She has longstanding hypertension and is noncompliant with her medical regimen of lisinopril and amlodipine. Blood pressure on presentation is 190/100 mm Hg.

What does her ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 8 Analysis: Normal sinus rhythm, left anterior fascicular block, left ventricular hypertrophy
There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm. The P wave has a normal morphology, amplitude, and duration. It is biphasic in lead V1.

The QRS complex duration is normal (0.10 sec), although in leads V4-V5 there is some delay in the upstroke of the R wave (→) (ie, a delayed intrinsicoid deflection). The axis is leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complexes in leads II and aVF). An extreme left axis deviation may be due to an inferior wall myocardial infarction, in which case Q waves would be present in leads II, III, and aVF, accounting for the left axis deviation. In this case, the QRS complexes have an rS morphology, which is characteristic of a left anterior fascicular block. The QT/QTc intervals are normal (360/420 msec). There are small Q waves in leads I and aVL (*); these represent normal septal depolarization. The amplitude of the QRS complexes in leads V1-V6 is normal; however, a markedly increased R-wave amplitude in leads I (22 mm) and aVL (23 mm) (↑) meets criteria for left ventricular hypertrophy (ie, an R-wave amplitude in any limb lead ≥ 20 mm or an R-wave amplitude in lead aVL ≥ 11 mm or ≥ 18 mm when a left axis deviation is present). In addition, there are associated ST-T wave abnormalities (↑), best seen in leads I, aVL, and V4-V6. These are repolarization abnormalities that represent chronic subendocardial ischemia, which occurs as a result of reduced blood supply to the endocardium due to the hypertrophy.

The ECG is diagnostic for left ventricular hypertrophy (tall R wave in leads I and aVL, ST-T wave changes, left axis deviation, and delayed intrinsicoid deflection; Romhilt-Estes score = 9), which is likely attributable to poorly controlled hypertension. The treating clinician in the emergency department must be mindful of potential myocardial ischemia, which can often present as indigestion. Myocardial ischemia could be related to the increased myocardial oxygen demands resulting from the markedly elevated blood pressure. In the emergency department, blood tests for myocardial ischemia (creatine kinase [CK], CK-MB, and troponin) should be drawn, and serial ECGs should be ordered periodically or with any change in symptoms. Markedly elevated blood pressure requires acute therapy, and the clinician should take this as an opportunity to stress compliance with antihypertensive medications given the long-term risk for end-organ dysfunction with uncontrolled hypertension.
A 28-year-old man is referred by his primary care physician to a cardiologist for refractory hypertension. His blood pressure is 190/76 mm Hg bilaterally in his arms but only 128/72 mm Hg in his legs. Cardiac exam reveals a prominent S4 and a sustained apical impulse. A chest X-ray shows prominent rib notching.

What does his ECG show?
What is the likely diagnosis?
ECG 9 Analysis: Normal sinus rhythm, left ventricular hypertrophy (LVH), recording at half-standard
Although the leads are not labeled, the pattern is always the same (column 1 is leads I, II, and III; column 2 is leads aVR, aVL, and aVF; column 3 is leads V1-V3; and column 4 is leads V4-V6). There is a regular rhythm at a rate of 70 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is 0.08 second, and there is a normal QRS complex morphology. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/410 msec). As recorded, the QRS voltage is not increased in any lead. However, noted is the standardization used (→) in recording this ECG: The limb leads were recorded at normal standardization (1 mV = 10 mm), while the precordial or chest leads were recorded at half-standard (1 mV = 5 mm). Hence the amplitude of the QRS complexes as recorded in the precordial leads is half of their actual amplitude. The R-wave amplitude in lead V5 is 26 mm ( ), while the S-wave depth in lead V1 is 26 mm ( ) (S-wave depth in lead V1 + R-wave amplitude in lead V5 = 52 mm); therefore, this meets one of the criteria for left ventricular hypertrophy (LVH) (ie, S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm in subjects > 45 years of age and ≥ 45 mm in those < 45 years of age). In addition, ST-T wave changes (↑) are noted in leads I, II, aVR (T wave should be negative), aVL, aVF, and V4-V6; these changes are characteristic of LVH and represent chronic subendocardial ischemia due to reduced perfusion of the endocardial layer of the hypertrophied left ventricular myocardium.

This young man appears to have hypertension on the basis of coarctation of the aorta. A blood pressure differential between the arms and lower extremities is caused by resistance to flow into the descending aorta. The diastolic pressures are often similar, as is observed in this patient. In most cases, the coarctation occurs just distal to the left subclavian artery so the blood pressures are similar in the two arms. Notching of the posterior one-third of the third to eighth ribs is due to erosion by the large collateral arteries. Computed tomography or magnetic resonance angiography can delineate the anatomic characteristics of the coarctation. There is no effective medical therapy for coarctation of the aorta discovered in adulthood. The treatment of choice is either surgical correction or balloon angioplasty with stenting.
A 72-year-old man is brought to the emergency department after suffering an eye injury at work. An intake ECG is abnormal and raises the suspicion for cardiac ischemia among the treating medical team. He denies chest pain. Exam reveals a blood pressure of 162/88 mm Hg, but the patient is otherwise normal. Initial screening creatine kinase (CK)-MB and troponin I are normal.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 10 Analysis: Sinus bradycardia, left atrial hypertrophy, left ventricular hypertrophy (LVH) with ST-T wave changes (ST-segment depression and T-wave inversions)
There is a regular rhythm at a rate of 58 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P waves are negative in leads V1-V2 (∗) and notched in leads V4-V6 (+), diagnostic for left atrial hypertrophy (P mitrale) or a left atrial abnormality.

The QRS complex duration is normal (0.10 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/430 msec). The morphology of the QRS complex is normal, but there is a marked increase in R-wave amplitude; it is 50 mm in lead V5 (+), which meets one of the criteria for left ventricular hypertrophy (LVH; S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). Also noted are marked ST-T wave changes (asymmetrically inverted or biphasic T waves) in most leads with ST-segment depression (†) in leads V4-V6. These are repolarization abnormalities associated with LVH and are due to chronic ischemia of the subendocardial layer caused by a reduction in subendocardial perfusion and oxygen supply resulting from LVH.

An ECG such as this can often raise suspicion for silent ischemia or an acute coronary syndrome. In the absence of clinical evidence for cardiac ischemia (no chest discomfort, dyspnea, pre-syncope, or nausea), this ECG is unlikely to indicate acute coronary ischemia. The pattern is much more typical of severe LVH with secondary repolarization changes attributable to increased wall stress. The primary goal of therapy should be to treat the hypertension and reduce wall stress. Since the patient’s blood pressure is not dangerously high, it is reasonable to start an oral antihypertensive medication and have the patient return to the outpatient office for further titration of the regimen. With aggressive treatment of the hypertension, there may be some resolution of the LVH and ECG changes.
You are asked to interpret an ECG performed at a preoperative anesthesia clinic. The patient plans to undergo elective rotator cuff surgery the following day.
The treating physicians are concerned about left ventricular hypertrophy. The patient has a normal physical examination and feels well.

**What do the ECGs show?**

**What is the likely diagnosis?**
Podrid's Real-World ECGs

ECG 11A Analysis: Normal sinus rhythm, ECG recorded at double standard, U waves
In ECG 11A, there is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.14 sec). The P waves are upright in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the morphology is normal. The QT/QTc intervals are normal (400/400 msec). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The R waves in leads V4-V6 ( ) have an increased amplitude (30 to 35 mm or small boxes), and the S wave in lead V2 is deep (27 mm or small boxes) ( ) (S-wave depth in lead V2 + R-wave amplitude in lead V6 = 62 mm). This meets one of the criteria for left ventricular hypertrophy (LVH; S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). Also noted are prominent U waves (↑) in leads V1-V4. However, it should be noted that the ECG was recorded at double standard (↔), that is, 1 mV equals 20 mm (20 small boxes) rather than at normal standard (1 mV = 10 mm or 10 small boxes). Hence the normal amplitude of the R wave or S wave is half of what is measured. Thus, LVH is not present.

Myocardial Abnormalities: Core Case 11 continues
Podrid’s Real-World ECGs

ECG 11B Analysis: Normal ECG recorded at normal standard
ECG 11B, obtained from the same patient, is recorded at normal standardization (←). The P-wave morphology, PR interval, QRS complex duration and axis, and QT/QTc intervals are the same as noted in ECG 11A. However, it should be noted that the amplitude (voltage) of the QRS complexes is normal and there is no evidence of LVH. U waves are still seen in leads V2-V3, but they are less prominent (↑).
A 33-year-old man with a history of surgically corrected tetralogy of Fallot returns to his cardiologist for routine follow-up. He notes mild pedal edema, which has worsened since his last visit 6 months earlier. Examination is notable for a harsh systolic murmur along the left upper sternal border and a right-sided S4.

What does his ECG show?

What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 12 Analysis: Normal sinus rhythm, right atrial hypertrophy (abnormality), right ventricular hypertrophy with associated repolarization abnormalities (ST-T wave changes), right axis deviation.
There is a regular rhythm at a rate of 94 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm. The P waves are positive in lead V1 (↑) and tall and peaked in lead II (↑) (P pulmonale), suggestive of right atrial hypertrophy (abnormality).

The QRS complex duration is normal (0.08 sec). There is a tall R wave in lead V1 (←) that is 9 mm in amplitude, and there is no S wave, so the R/S ratio is greater than 1; these meet criteria for right ventricular hypertrophy (ie, R-wave amplitude in lead V1 ≥ 7 mm or R/S > 1). The QRS axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include right ventricular hypertrophy (associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (a deep Q wave in leads I and aVL), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation). The QT/QTc intervals are normal (320/400 msec).

The tall R wave in lead V1 (←) along with the right axis deviation and right atrial hypertrophy are characteristic of right ventricular hypertrophy. Also noted are ST-T wave changes in leads V1-V4 (↑). These abnormalities are associated with right ventricular hypertrophy and, similar to the situation with left ventricular hypertrophy, they represent subendocardial ischemia of the right ventricular myocardium due to reduced endocardial perfusion of the hypertrophied right ventricular myocardium.

Tetralogy of Fallot (TOF) is a congenital abnormality that results from anterior deviation of a portion of the infundibular septum into the right ventricular outflow tract (RVOT). TOF presents with four characteristic abnormalities: RVOT obstruction, ventricular septal defect, overriding aorta (rightward deviation of the origin of the aorta), and concentric right ventricular hypertrophy. Most patients with TOF undergo intracardiac repair as the first operation. This procedure is aimed at relieving the RVOT obstruction by enlarging the RVOT; the ventricular septal defect is also closed with a patch. This patient has clinical and ECG evidence of persistent RVOT obstruction. Although the obstruction is reduced at the time of surgery, persistent RVOT obstruction or pulmonic insufficiency can be a long-term complication, depending on changes in the RVOT geometry as the heart grows. In cases of persistent RVOT obstruction the patient may require re-operation or catheter-based intervention to relieve the residual obstruction.
A 62-year-old man with known bicuspid aortic valve presents for evaluation of pedal edema and weight gain. He has been followed for several years for aortic stenosis but has been reluctant to undergo surgery. He has had no syncope and has remained free of chest discomfort by limiting his physical activity.

In recent months, he has gained weight and developed pedal edema. Exam shows an elevated jugular venous pressure with prominent V waves, harsh crescendo–decrescendo murmur at the right upper sternal border that radiates to the carotids, and a holosystolic murmur at the apex that radiates to the axilla.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 13 Analysis: Normal sinus rhythm, right atrial hypertrophy (abnormality), left atrial hypertrophy (abnormality), biatrial hypertrophy (abnormality), right axis deviation, right ventricular hypertrophy (RVH), left ventricular hypertrophy (LVH; biventricular hypertrophy), intraventricular conduction delay, QT prolongation
There is a regular rhythm at a rate of 98 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P waves are tall and peaked in leads II and aVF as well as in lead V2 (P pulmonale) (+). This is characteristic of right atrial hypertrophy (abnormality). There is also evidence of left atrial hypertrophy (abnormality), with broad P waves in leads II, III, and aVF (↓) and a marked negative component to the P wave in lead V1 (χ). This represents biatrial hypertrophy (abnormality).

The QRS complex duration is prolonged (0.12 sec) with a nonspecific morphology (intraventricular conduction delay), and the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include right ventricular hypertrophy (RVH; associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (a deep Q wave in leads I and aVL), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation). The QT/QTc intervals are prolonged (400/510 msec), even when the prolonged QRS complex duration is considered (360/460 msec).

There is a tall R wave in lead V1 (←), which, along with the right axis deviation and the right atrial hypertrophy, is characteristic of RVH. In addition, the R-wave amplitude in lead V5 (33 mm) (↑) and the S-wave depth in lead V2 (27 mm) (↓) (S-wave depth in lead V2 + R-wave amplitude in lead V5 = 60 mm) meet one of the criteria for left ventricular hypertrophy (LVH; S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). Hence biventricular hypertrophy is present.

This patient has demonstrated the typical progression of symptomatic aortic stenosis. He has delayed symptoms by limiting his physical activity and has thereby delayed surgical repair. However, he has now developed evidence of biventricular and biatrial hypertrophy. Left ventricular dysfunction arises from decreased left ventricular compliance resulting from LVH. Left ventricular dilation occurs to reduce wall tension once the left ventricle has maximally hypertrophied. In the context of left ventricular dysfunction, pulmonary hypertension can arise from chronic elevation of the left ventricular end-diastolic pressure resulting from decreased compliance of the hypertrophied left ventricle. Pulmonary hypertension is a marker for poor survival both with and without valve replacement, but this patient’s aortic stenosis has progressed to the point that surgical repair is imperative. The presence of coexisting mitral valve disease (ie, mitral stenosis or mitral regurgitation) should be considered as these conditions will also produce pulmonary artery hypertension resulting in RVH and right atrial hypertrophy. The presence of both aortic and mitral valvular disease is a common presentation for patients with rheumatic heart disease.
Notes
A 65 year-old man with severe chronic obstructive pulmonary disease (COPD) presents with weight gain and lower extremity edema. He was recently hospitalized for exacerbation of COPD and was discharged on a course of corticosteroids.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 14 Analysis: Normal sinus rhythm, right atrial hypertrophy, right ventricular hypertrophy, right axis deviation, recorded at half-standard
Although the leads are not labeled, the pattern is always the same (column 1 is leads I, II, and III; column 2 is leads aVR, aVL, and aVF; column 3 is leads V1-V3; and column 4 is leads V4-V6). There is a regular rhythm at a rate of 86 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P waves (+) are tall (0.3 mV), narrow (0.08 sec), and peaked in leads II and aVF and positive and tall in lead V1 (^). This is characteristic of P pulmonale or right atrial abnormality.

The QRS complex duration is narrow (0.08 sec), and the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include right ventricular hypertrophy (associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (a deep Q wave in leads I and aVL), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation).

There is a tall R wave in lead V1 (11 mm) (←), and there is an S/R greater than 1 in lead V6; these meet criteria for right ventricular hypertrophy (ie, R-wave amplitude in lead V1 ≥ 7 mm and S/R in lead V6 > 1). There are also ST-T wave changes in leads V1-V3 (↑). However, while the limb leads were recorded at normal standard (1 mV = 10 mm), the precordial or chest leads were recorded at half-standard (↓) (1 mV = 5 mm). Hence the actual amplitude of the R wave in lead V1 is 22 mm. The tall R-wave voltage in lead V1 along with the right axis deviation, ST-T wave changes in leads V1-V3, and right atrial hypertrophy are characteristic of right ventricular hypertrophy.

This patient has right ventricular and right atrial hypertrophy arising in the context of COPD; this is termed cor pulmonale. This entity describes any cause of right heart dysfunction caused by the pulmonary vasculature. In COPD, pulmonary hypertension can arise from multiple causes, including hypoxic vasoconstriction, destruction of the pulmonary vascular bed by scarring emphysema, and increased pulmonary blood flow. The presence of cor pulmonale is a marker for advanced COPD and portends a poor prognosis. Treatment of cor pulmonale is focused on improving oxygenation (supplemental oxygen) and diuretic therapy to minimize peripheral edema. Pulmonary vasodilators can be used, but success is often limited in this setting.
A 28-year-old man presents for a pre-employment physical examination. He has no complaints. Physical examination is normal.

What does his ECG show?
What is the likely diagnosis?
ECG 15 Analysis: Sinus bradycardia, early repolarization, prominent U waves
The rhythm is regular at a rate of 46 bpm. There is a P wave (*) in front of each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a sinus bradycardia. The P-wave morphology is normal.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal, and there is normal R-wave progression across the precordium. The QT/QTc intervals are also normal (400/350 msec). U waves (^) can be seen after the T waves in leads V3-V6. The ST segments have a normal configuration (ie, they are slightly concave). The J point and ST segments in leads V5-V6 are slightly above baseline (↑), which is defined by the TP segment. This is a normal ECG with minimal early repolarization.

Early repolarization is seen in about 5% of normal ECGs, most often in the lateral precordial leads (V4-V6). It does not have any known pathologic correlates and does not portend adverse events. It is most commonly observed in young men as well as in the black population but can be seen at any age in either sex and in any ethnic group. It is often seen with tall QRS voltage (with or without left ventricular hypertrophy) as an exaggeration due to the increased QRS complex amplitude. The abnormalities can sometimes be dynamic, which may raise concern for myocardial ischemia or pericarditis. In these cases, the ECG findings must be considered in the clinical context. Moreover, with pericarditis the ST-segment elevations are diffuse, involving most or all leads. An acute myocardial infarction usually has associated hyperacute T waves (tall, peaked, and symmetric) as well as reciprocal ST-segment depressions. Sinus bradycardia, defined as a heart rate less than 60 bpm, is a common finding in young, healthy patients.
A 28-year-old woman presents for evaluation of palpitations. She often notes that she feels “extra beats” when lying still in bed. She denies any syncope or pre-syncope. She feels well otherwise. Her internist obtains an ECG and refers her for further evaluation of possible coronary artery disease.

What does her ECG show?
What is the likely diagnosis?
ECG 16 Analysis: Normal sinus rhythm, premature atrial complexes (PACs), nonspecific ST-segment flattening
The rhythm is basically regular at a rate of 60 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. There are, however, two QRS complexes that are early or premature (second and fourth) (+). They have a QRS morphology that is identical to all of the other QRS complexes. Both are preceded by a P wave (↑), but the P-wave morphology is slightly different from those of the sinus complexes (ie, they are negative in lead V1 compared with the sinus P wave, which is biphasic in lead V1 and flat in lead II compared with the sinus P wave, which is positive in lead II). These are, therefore, premature atrial complexes (PACs).

The QRS complex duration is normal (0.08 sec), and there is a normal QRS axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal, with normal R-wave progression across the precordium. The QT/QTc intervals are normal (420/420 msec), and the T waves have a normal asymmetric morphology. The J point and ST segments are at baseline (ie, the same level as the TP segments). However, in some of the leads (ie, leads II, III, aVF, and V5-V6) the ST segments (↑) no longer have their normal concave morphology but are flattened. This is a nonspecific ST-segment abnormality, which does not imply any cardiac abnormality if flattened without depression.

This patient has symptomatic PACs. PACs are a common finding that, if infrequent, are not considered pathologic. Although they are common in the general population, the frequency of PACs increases with age and they are more common in the presence of structural heart disease, exogenous stimulant administration, or other medical illnesses. No therapy is indicated for asymptomatic PACs. However, if a patient is bothered by palpitations, the symptoms can be suppressed with β-blockers (although β-blockers do not have any direct effect on the myocardium and do not generally suppress PACs). Not uncommonly, PACs trigger other atrial arrhythmias, such as atrial tachycardia, atrial flutter, and atrial fibrillation. Hence PACs are occasionally treated to prevent the occurrence of these sustained atrial tachyarrhythmias. Treatment to suppress PACs requires the use of antiarrhythmic agents that work on atrial myocardium and include the class IA, IC and III antiarrhythmic drugs.
A 48-year-old woman is evaluated for atypical chest pain. Her father suffered a myocardial infarction in his early 50s, but she has no other risk factors. She is evaluated with an office exercise stress test.
The technologist notes that there is a subtle change in the P-wave morphology, the PR interval duration, and the ST segment. ECG 17A is the patient's ECG before the stress test (baseline); ECG 17B was obtained after the stress test.

How do you explain these findings?
What is the diagnosis?
Is any further evaluation necessary?

ECG 17B
Podrid’s Real-World ECGs

ECG 17A Analysis: Normal
ECG 17A shows a regular rhythm at a rate 76 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.14 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There is normal R-wave progression from leads V1 to V6. The ST segments, T waves, and QT/QTc intervals (360/400 msec) are also normal.

continues
ECG 17B Analysis: Sinus tachycardia, upsloping ST-segment depression
ECG 17B was obtained immediately upon termination of the patient’s exercise test on a treadmill. There is a regular rhythm at a rate of 150 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P wave has the same axis and morphology as seen in ECG 17A; this is sinus tachycardia. Of note is the fact that the P waves have a greater amplitude in leads II, III, and aVF (+) compared with baseline. This is commonly seen with sinus tachycardia and is the result of a change in the direction of atrial activation originating from the sinus node (ie, atrial activation is more inferiorly directed).

Anatomically, the sinus node is a cylindric crescent-shaped structure located at the proximal portion of the right atrium. Within the sinus node are pacemaker cells that have different rates of action potential generation. The more proximal pacemaker cells generate an action potential at a faster rate compared with those located at the more distal end. The sinus node is also innervated by the parasympathetic and sympathetic nervous systems. There are more vagal fibers located at the proximal portion of the node compared with the distal portion. When vagal tone is increased (and sympathetic tone decreased), the pacemaker cells at the proximal portion of the node are suppressed and the dominant pacemaker location is shifted to the distal portion of the node where pacemaker activity is slower; so the heart rate slows. In addition, the activation of the atrial tissue occurs in a direction that is more horizontal and therefore parallel to lead I and perpendicular to leads II and aVF. In this situation the P wave is taller in lead I and of a lesser amplitude in leads II and aVF. When there is an increase in sinus rate, which is due to withdrawal of vagal tone and increased sympathetic tone, the location of dominant pacemaker activity is shifted to the more proximal portion of the node, where action potential generation is faster. Therefore, activation of the atrium is in a more vertical direction, parallel to leads II and aVF, so the amplitude of the P wave increases in these leads while it decreases in lead I.

continues
The QRS complex duration and morphology in ECG 17B are normal and are identical to those in ECG 17A. The QT/QTc intervals are normal (240/380 msec). However, there is pronounced J-point and ST-segment depression, particularly seen in leads II, III, aVF, and V4-V6 (^); note that ST-segment elevation in lead aVR is actually ST-segment depression. Although a TP segment is not obvious due to the sinus tachycardia, the PR segment can be used as baseline. The ST-segment depression is described as upsloping. Upsloping ST-segment depression may be a normal finding during sinus tachycardia or can be a manifestation of ischemia, although it is the least predictive. It should be remembered that atrial repolarization (ie, the T wave of the P wave) occurs during the QRS complex and hence is not seen. When the sinus rate increases, however, there is a shortening of the PR segment (due to sympathetic enhancement of conduction through the AV node), which causes the T wave of the P wave to move out from the QRS complex and fall on the J point, causing both the J point and the initial portion of the ST segment to be depressed. Therefore, the ST segment slopes upward to return to baseline. When there is upsloping ST-segment depression, it is standard practice to evaluate the degree of depression present at 80 msec (two small boxes) past the J point, which eliminates any effect from the atrial T wave on the J point and ST segment. If the ST segment at 80 msec past the J point is still depressed more than 1.5 mm below baseline (the TP segment or, if not obvious, the PR segment), then subendocardial ischemia is present.

In this case the ST segment is back to baseline at 80 msec past the J point. Hence, this is a normal rate-related change.
A 58-year-old man with hypertension, dyslipidemia, and a family history of cardiac disease presents for evaluation of cardiovascular risk prior to undergoing elective spine surgery. He undergoes an exercise stress test that provokes chest discomfort after 6 minutes; as a result the test is terminated. The ECG at 2 minutes of recovery is shown.

What does his ECG show?
What is the likely diagnosis?
ECG 18 Analysis: Normal sinus rhythm, upsloping ST-segment depression
There is a regular rhythm at a rate of 92 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and it has a normal morphology and axis, between 0° and 90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/420 msec). J-point and ST-segment depression can be seen in leads II, III, aVF, and V2-V6 (↑); ST-segment elevation (▼) in lead aVR is actually ST-segment depression. The ST-segment depression is upsloping. However, at 0.08 sec past the J point (→) the ST segment is still depressed 2 mm below the baseline (TP segment). This is highly suggestive of an ischemic response. The location of ST-segment depression does not correlate with the location of the ischemia or with the coronary artery that is involved.

There are three types of ST-segment depression that reflect ischemia: upsloping, horizontal, and downsloping. Upsloping ST-segment depression is the least specific change of ischemia as it may be seen in sinus tachycardia as a rate-related change. However, when the ST segment remains more than 1.5 mm below baseline at 80 msec past the J point, it is diagnostic for ischemia. In addition, the upsloping ST-segment changes were seen at a rate of 92 bpm; therefore, they are not a normal rate-related change.
A 64-year-old woman with longstanding diabetes is evaluated for complaints of atypical chest discomfort (left-sided ache). She has been taking a β-blocker for hypertension. She undergoes an exercise stress test, which provokes her symptoms. The peak exercise ECG is shown below.

What does the ECG show?

What is the likely diagnosis?

What further diagnostic evaluation could be undertaken?
ECG 19 Analysis: Sinus tachycardia, low voltage, horizontal ST-segment depression
The ECG shows a regular rhythm at a rate of 120 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is upright in leads I, II, aVF, and V4-V6, and the P-wave morphology is normal. This is sinus tachycardia.

The QRS complex duration is normal (0.08 sec), and the axis is slightly leftward, between 0° and −30° (positive QRS complex in leads I and II and slightly negative QRS complex in lead aVF). The QRS morphology is normal, and there is normal R-wave progression from lead V1 to lead V6. However, the QRS amplitude in the limb leads is low (defined as a QRS complex amplitude ≤ 5 mm in each limb lead). The QT/QTc intervals are normal (260/370 msec). Diffuse J-point and ST-segment depression (↑) can be seen in almost all leads. The ST segments are 3 to 4 mm below the baseline (TP segment) (↓) and are horizontal, except in leads I and V6, where the ST-segment depression is downsloping. Horizontal ST-segment depression is more specific for ischemia than upsloping ST-segment depression. Although the leads in which ST-segment depression is seen do not necessarily correlate with the location of ischemia, the finding of widespread ST-segment depression does correlate with significant, probably multivessel coronary artery disease. The degree of ST-segment depression (ie, number of millimeters depressed) also correlates with the severity of the coronary disease.

This patient likely has multivessel coronary disease. Although her chest discomfort is atypical for ischemia, she is both diabetic and female. In both of these situations ischemic chest discomfort may present with atypical features. Therefore, additional anti-ischemic therapy should be initiated (ie, a long-acting nitrate and increased dose of β-blocker to further reduce her heart rate with activity). If symptoms persist, further delineation of the coronary anatomy is warranted. This is usually accomplished with diagnostic coronary angiography. If multivessel disease is confirmed, this patient will likely require revascularization, either with a percutaneous intervention or coronary artery bypass graft surgery. In most cases, symptomatic ischemia in the presence of diabetes mellitus and multivessel disease, particularly involving the left main or left anterior descending artery, is an indication for surgical revascularization.
An 81-year-old woman is admitted with fever, hypotension, and bacteremia. She is found to have *Escherichia coli* sepsis with a likely urinary...
tract source and is admitted to the intensive care unit for antibiotics and fluids. An ECG (20A) is obtained and compared with her baseline ECG (20B).

**What does ECG 20A show?**

**What is the likely diagnosis?**

**What therapy is appropriate?**

**ECG 20B**
ECG 20A Analysis: Normal sinus rhythm, downsloping ST-segment depression
In ECG 20A, there is a regular rhythm at a rate of 92 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the morphology is normal with normal R-wave progression in leads V1 to V6. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/420 msec). There are small Q waves in leads II, III, and aVF. They are not deep or wide (0.04 sec). These are septal Q waves.

There is prominent J-point and ST-segment depression (^) in leads I, II, aVF, and V3-V6. The ST-segment depression is downsloping, especially in leads V3-V6, and is 2 to 4 mm below the baseline (↕) (TP segment). Downsloping ST-segment depression is the type of ST-segment change that is most predictive of significant coronary artery disease. The number of leads showing ST-segment depression and the depth of the depression are predictive of multivessel disease.
Podrid's Real-World ECGs

ECG 20B Analysis: Normal
ECG 20B is the patient’s baseline ECG. The P waves, PR interval, QRS complex, T waves, and QT interval are normal and are the same as in ECG 20A. However, there is no J-point or ST-segment depression present (↑).

These ECGs demonstrate evidence of myocardial ischemia in the setting of the hemodynamic stress of bacteremia and sepsis. Though rate-related ST-segment changes are common and can be nonspecific, these downsloping ST-segment depressions are likely to indicate subendocardial ischemia. It is not uncommon for patients to suffer myocardial ischemia or small elevations of cardiac biomarkers in the setting of this kind of hemodynamic perturbation. This is termed demand ischemia (also called a type II myocardial infarction) and has a different pathophysiology than a non-ST-segment elevation myocardial infarction (NSTEMI) (also called a type I myocardial infarction), which is due to acute plaque rupture and intracoronary thrombosis. In most cases of NSTEMI, patients have symptoms of myocardial ischemia associated with the ECG changes and elevated biomarkers. Therapy for demand ischemia should be focused on restoring normal hemodynamics, including volume resuscitation, treatment of the underlying infection, and support of the blood pressure with vasoactive agents. Antithrombotic medications are not indicated in this clinical scenario as this is not a type I NSTEMI.
A 55-year-old woman presents to the emergency department for evaluation of chest discomfort. She notes typical angina occurring with activity two to three times per week, most recently about 30 minutes prior to presentation. She has risk factors including hypertension and a strong family history of coronary artery disease.
Her presenting ECG is shown (21A). She undergoes an exercise stress test, during which she exercises for 7 minutes 24 seconds with no chest pain. One minute into recovery, she develops severe retrosternal chest discomfort associated with nausea and diaphoresis. A second ECG is obtained (21B).

What do these ECGs show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 21A Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), nonspecific ST-T wave abnormalities
The baseline ECG (21A) shows a regular rhythm at a rate of 66 bpm. There is a P wave (\(\ast\)) before each QRS complex, with a stable PR interval (0.14 sec). The P waves are upright in leads II, III, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P waves are prominently notched in leads II and V5-V6 (\(^\wedge\)) and are primarily negative in leads V1-V2 (\(\wedge\)). This is consistent with left atrial hypertrophy or a left atrial abnormality.

The QRS complex duration is normal (0.08 sec), with a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT interval is normal (400/420 msec), and the ST segments are normal and at baseline. The T waves are biphasic (\(\ast\)) in leads V2-V3 and negative (\(\wedge\)) in leads V4-V5. Although T-wave inversions are nonspecific, they are meaningful when associated with a clinical story that is characteristic for discomfort due to myocardial ischemia.

Although not completely typical (as the T waves are biphasic in leads V2-V3), the T-wave changes seen in this ECG are suggestive of Wellens’ syndrome. Wellens’ T waves are deep and symmetrically inverted T waves in leads V2-V4 or V5. This pattern should alert the clinician to a potential high left anterior descending coronary artery (LAD) stenosis. This pattern is frequently overlooked by treating physicians because it is not a typical pattern of ischemia. Nonetheless, this could represent a potentially unstable LAD plaque. If the frequency of angina is increasing or is occurring at rest, indicating an unstable pattern, the patient should be evaluated using coronary angiography rather than exercise stress testing. However, if the angina has a stable pattern, exercise testing is reasonable.

continues
ECG 21B Analysis: Normal sinus rhythm, T-wave inversions suggesting ischemia, exercise-induced ST-segment elevation due to ischemia
ECG 21B was obtained within the first minute of recovery after the stress test. The rhythm is regular at a rate of 94 bpm. The P waves (*), PR interval, QRS complexes, and QT/QTc intervals are the same as in ECG 21A. However, there is significant ST-segment elevation (↓) in leads V2-V4. ST-segment elevation occurring in leads that do not have Q waves indicates transmural ischemia. This was provoked by exercise and is associated with a critical, subtotal occlusion of a coronary artery. In this case the ST-segment elevations are indicative of transmural anteroapical ischemia, likely from a proximal to mid-LAD lesion. The provocation of ST-segment elevation during an exercise test is an indication for urgent cardiac catheterization and intervention as appropriate.

In contrast, ST-segment elevation during an exercise test that is seen in leads where there has been a previous infarction, indicated by a Q wave, is associated with the development of a wall motion abnormality.
A 58-year-old man with a prior inferior and anterior wall myocardial infarction presents with sharp chest pain while playing tennis. The pain is reliably reproduced with vigorous activity but has also occurred at rest. The chest pain is sharp and located at the left sternal border. Episodes last about 30 seconds. A baseline ECG (22A) is obtained.
in his primary care physician’s office. His physician refers him for an exercise stress test. He exercises for 8 min 14 seconds on a Bruce protocol and stops due to shortness of breath but does not develop chest pain. Within 1 minute of stopping the test, his ECG changes (ECG 22B) and he develops chest pain similar to his presenting pain.

What do these ECGs show?
What is the likely diagnosis?


**Podrid's Real-World ECGs**

**ECG 22A Analysis:** Normal sinus rhythm, intraventricular conduction delay, old inferior wall myocardial infarction (MI), old anteroapical and anterolateral wall MI
ECG 22A shows a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves have a normal morphology and are upright in leads II, III, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is increased (0.12 sec). There is no specific morphology indicative of a bundle branch block; hence this is an intraventricular conduction delay. There are significant Q waves (+) in leads II, III, and aVF, indicative of a previous inferior myocardial infarction (MI). There is also a small but significant Q wave (↑) in lead V2 as well as Q waves (↑) in leads V3-V6, indicative of an anteropical and anterolateral MI. The QRS axis appears to be about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). However, the biphasic QRS complex in lead aVF is the result of a deep Q wave that is the result of an MI. If this is not considered, the axis is normal, between 0° and +90°. The QT/QTc intervals are normal (360/380 msec and 320/340 msec when corrected for the prolonged QRS complex duration).

This baseline ECG is consistent with an old inferior and anteropical and anterolateral MI. There is no evidence of active ischemia by ECG, but the patient’s recent exertional chest pain is suspicious for residual ischemia (although it is not typical for myocardial ischemia). Since the symptoms occur primarily with exertion, a stress test is indicated.

continues
ECG 22B Analysis: Exercise-induced ST-segment elevation due to wall motion abnormality
ECG 22B was recorded immediately upon the termination of exercise, within the first minute of recovery. The rate is 150 bpm. The P wave (*); PR interval; QT/QTc intervals; and QRS complex duration, axis, and morphology are the same as in ECG 22A. However, ST-segment elevation (↓) is noted in leads II, III, aVF, and V3-V6. These ST-segment changes are seen in the same leads in which there are Q waves (^), which are indicative of a previous MI. Hence these ST-segment changes are the result of the development of an exercise-induced wall motion abnormality in the area of a previous MI. In this situation they are not the result of transmural ischemia as would be the case if the ST-segment elevation occurred in leads without Q waves. However, there is 1 mm horizontal ST-segment depression in leads I and aVL (↑), which may be indicative of subendocardial ischemia.
A 84-year-old woman is admitted to the hospital following an elective cholecystectomy. She has a history of hypertension and chronic renal insufficiency. Six hours after her surgery, she developed chest tightness and dyspnea. An ECG at the time of her symptoms is shown.

What does her ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 23 Analysis: Normal sinus rhythm, acute anterior wall myocardial infarction (MI), hyperacute T waves, left atrial hypertrophy
Although the leads are not labeled, the pattern is always the same (column 1 is leads I, II, and III; column 2 is leads aVR, aVL, and aVF; column 3 is leads V1-V3; and column 4 is leads V4-V6). The ECG shows a regular rhythm at a rate of 92 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is negative in leads V1-V2 (^), indicating left atrial hypertrophy or left atrial abnormality.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/400 msec), although there is a broad T wave, and the ST segment seems to be short. The T waves (↓) in leads V3-V5 are tall and symmetric (the T-wave upstroke and downstroke are equal), in contrast to the normal T wave, which is asymmetric (ie, the upstroke of the T wave is slower than the downstroke). T waves that are tall, peaked and, most importantly, symmetric are seen with hyperkalemia and they are called hyperacute. Hyperkalemia may be systemic and hence associated with diffusely symmetric T-wave changes, or it may be localized, as occurs with an acute myocardial infarction (MI). Therefore, the hyperacute T waves, primarily in leads V3-V5, indicate an acute anteroapical MI. The shortened ST segment is also indicative of an acute MI. There are minimal ST-segment elevations in leads V1 and V2 and ST-segment depressions in leads II, III, and aVF (^), which are reciprocal changes. Reciprocal changes are the same ST-segment changes seen from another angle.

An acute infarction is the result of total cessation of blood flow and oxygen supply. This causes loss of the normal energy (oxygen)-dependent sodium–potassium–ATPase pump and therefore loss of membrane integrity. As a result, potassium leaks out of the cell, based on its electrochemical gradient (normally intracellular potassium levels are higher than extracellular levels because of this ATPase pump). As there is no blood flow into or out of the infarcted tissue, the potassium remains in this area, producing local hyperkalemia. There is also an inward “leak” of calcium and the development of intracellular hypercalcemia, probably accounting for the shortening of the QT interval.

The earliest change with a transmural infarction is hyperacute (symmetric) T waves seen in the area of the involved myocardium. This is due to the localized hyperkalemia that develops and is seen even before there are any ST-segment changes. Thereafter, the ST segment begins to elevate, maintaining its normal concave morphology; hyperacute T waves are still present. ST segments continue to elevate and then become convex, merging with T waves. The amplitude of the R wave decreases. When the ST segment and T waves merge and the R wave is no longer obvious, the complex has the morphology that is called a “current of injury.” It resembles the fast action potential. The ST segment begins to return to baseline, Q waves develop, and T waves begin to invert, resulting in a chronic infarct pattern (ie, Q-wave and T-wave inversion). Persistent ST-segment elevation, present weeks to years after an acute event, indicates the presence of an aneurysm in the area of the previous infarction.

continues
The location of these changes identifies the region of myocardium involved:

- **Inferior wall MI**: ST-segment elevation in leads II, III, and aVF. The right ventricle is often involved in an inferior wall MI; this is suggested by the presence of ST-segment elevation in lead V1 and ST-segment depression in lead aVR (which actually represents ST-segment elevation) and is confirmed by obtaining right-sided leads, which will show ST-segment elevation in leads V3R-V4R. There may also be involvement of the posterior wall, which is suggested by the presence of ST-segment depression in leads V1-V2. Posterior leads placed on the back below the left scapula (V7-V8) showing ST-segment elevation help support the diagnosis of a posterior wall MI.
- **Anteroseptal MI**: ST-segment elevation in leads V1-V2
- **Anteroapical MI**: ST-segment elevation in leads V3-V4
- **Anterolateral MI**: ST-segment elevation in leads V5-V6
- **Anterior wall MI**: ST-segment elevation in two or more contiguous leads across the precordium (ie, leads V1-V6)
- **Lateral MI**: ST-segment elevation in leads I and aVL

The clinical story associated with the ECG changes that indicate an acute transmural or ST-segment elevation myocardial infarction (STEMI) should result in emergent activation of the catheterization laboratory because an acute percutaneous coronary intervention (ie, stenting) is indicated. In acute MI, the size of the infarct can be reduced and survival improved with prompt revascularization (ie, within 90 minutes of symptom onset). This should be easily achievable for a hospitalized patient, assuming there is recognition that the hyperacute T wave indicates an acute infarction.
A 72-year-old man with hypertension and dyslipidemia is admitted with an inferior wall myocardial infarction (MI). He undergoes urgent percutaneous coronary intervention and placement of a bare metal stent in a dominant mid–right coronary artery. The initial changes on the ECG resolve. Twelve hours after his intervention, he develops recurrent chest discomfort and diaphoresis, resembling the symptoms associated with the MI. An ECG is obtained immediately.

What does his ECG show? What is the likely diagnosis?
ECG 24 Analysis: Normal sinus rhythm, acute inferior wall MI, hyperacute T waves
There is a regular rhythm at a rate of 84 bpm. There is a P wave (*) in front of each QRS complex, with a stable PR interval (0.18 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a small R′ in lead V1 (↓), indicating delayed activation of the right ventricle. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are short (280/330 msec) as a result of a short ST segment.

The T waves (+) in leads II, III, and aVF are tall and hyperacute (T-wave upstroke and downstroke are equal) in contrast to the normal T wave, which is asymmetric (ie, the upstroke of the T wave is slower than the downstroke). T waves that are tall, peaked and, most importantly, symmetric (hyperacute) are seen with hyperkalemia. Hyperkalemia may be systemic or localized, as is seen in acute transmural myocardial infarction (MI). Hyperacute T waves are the first abnormality noted in an acute transmural MI as a result of localized hyperkalemia. They occur in the area of myocardium involved and are seen even before there are any ST-segment changes. Hence this is a very early acute inferior wall MI. Also noted are ST-segment depressions in leads V2-V4 (↑), which possibly represent involvement of the posterior wall. This can be confirmed by recording from posterior leads placed on the back below the left scapula (leads V7-V8). These ST-segment depressions may also represent reciprocal ST-segment changes (ie, the same ST-segment changes [elevations] viewed from another angle or direction). However, the ST segments in leads II, III, and aVF are not yet elevated, so these are probably not reciprocal ST-segment changes.

An acute MI occurring shortly after percutaneous coronary intervention, as seen in this case, is likely related to acute stent thrombosis. Acute stent thrombosis is a rare but life-threatening complication of the use of coronary stents to treat obstructive coronary disease. The risk factors for stent thrombosis include procedural factors (eg, stent underdeployment or stent fracture), complicated target lesions (eg, bifurcation lesions), local factors (eg, poor blood flow, residual thrombus), and hematologic factors (eg, hypercoagulable state or incomplete platelet inhibition). Patients require immediate intervention to restore adequate blood flow through the stent. Since acute stent thrombosis is very often caused by a mechanical problem with the stent (such as underdeployment or fracture), intravascular ultrasound can be used to assess the apposition of the stent to the vessel wall and the integrity of the stent struts. Patients should be treated with intensified dual antiplatelet therapy (usually aspirin and double-dose clopidogrel). Although data on long-term outcomes are still being collected, most practitioners will continue dual antiplatelet therapy indefinitely in the absence of contraindications.
A 38-year-old man presents to the emergency department within 30 minutes of developing chest discomfort. The discomfort occurred after using intranasal cocaine and has been unrelenting since its onset.

What does his ECG show?
What is the likely diagnosis?
**Podrid’s Real-World ECGs**

**ECG 25 Analysis:** Sinus bradycardia, acute anterior wall ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 56 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus bradycardia.

The QRS complex duration is normal (0.08 sec); there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF), and normal morphology. The QT/QTc intervals are normal (400/390 msec). There are tall and symmetric T waves (+) in leads V2-V6. The normal T wave is asymmetric (i.e., the upstroke is slower than the downstroke). T waves that are tall, peaked and, most importantly, symmetric (hyperacute) are seen with hyperkalemia. Hyperkalemia may be systemic or localized, as is seen in acute transmural myocardial infarction (MI). Hyperacute T waves are the first abnormality noted in an acute transmural MI and are due to localized hyperkalemia. They occur in the area of myocardium involved and are seen even before there are any ST-segment changes. Also noted is ST-segment elevation (↑) in leads V2-V4, and the ST segments are convex. Hence this is an early acute anteroapical ST-segment elevation MI (STEMI). In addition, ST-segment depressions (↓) are noted in leads III and aVF; these are reciprocal ST-segment changes that are due to the same ST-segment changes viewed from another angle or direction.

This patient is having acute transmural myocardial ischemia and possibly an acute anterior wall MI, likely resulting from his recent cocaine use. If the T-wave and ST-segment abnormalities resolve with nitrate therapy, the cause is most likely spasm. Persistent changes would suggest an acute transmural MI. Non-ischemic chest pain is common in patients who use intranasal cocaine, so differentiating myocardial ischemia from noncardiac chest pain can be challenging. Cocaine can cause ischemic chest discomfort by inducing vasospasm and increasing myocardial oxygen demand by increasing heart rate, blood pressure, and inotropy. Although uncommon, cocaine can provoke an acute MI by spasm associated with thrombus formation due to increasing thrombogenicity as a result of enhanced platelet activation and aggregation. Chronic use is associated with hypertension, left ventricular hypertrophy, cardiomyopathy, myocarditis, and occasionally aortic dissection and stroke. Chronic cocaine use can also result in accelerated atherosclerosis as a result of endothelial dysfunction. Therefore, it is also possible that the infarction is the result of an acute plaque rupture, induced by cocaine and vasospasm. This would occur if the spasm occurs in the area of an atherosclerotic plaque, with resultant plaque disruption, setting up the usual mechanism for thrombus formation and a typical MI.

There are several considerations that the practitioner should take into account when treating cocaine-related myocardial ischemia and/or infarction. β-blockers are relatively contraindicated because of a theoretical risk for severe hypertension and arterial vasoconstriction caused by unopposed α-adrenergic stimulation with ongoing cocaine use. Thrombolysis is relatively contraindicated because of the risk for intracranial hemorrhage and because many patients with ST-segment elevation may also have vasospasm that will not respond to thrombolysis. Also, the treating physician must consider the likelihood of compliance with clopidogrel before placing an intracoronary stent. Balloon angioplasty is a reasonable alternative to restore adequate coronary blood flow with minimal long-term risks.
A 49-year-old man with chronic obstructive pulmonary disease (COPD) develops severe retrosternal chest tightness, diaphoresis, and lightheadedness while eating lunch. This continues without relief for several minutes, so he has a friend drive him to the emergency department. An ECG is obtained immediately upon arrival.

What does his ECG show?
What is the likely diagnosis?
What further treatment is indicated?
ECG 26 Analysis: Sinus tachycardia, right bundle branch block (RBBB), acute anterior wall ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 110 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a sinus tachycardia.

The QRS complex duration is increased (0.12 sec), and there is a pattern of a right bundle branch block with an RSR’ complex (←) in lead V1 and broad S waves (^) in leads I and V5-V6. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/430 msec and 280/380 msec when corrected for the prolonged QRS complex duration). There is significant ST-segment elevation (↓) in leads V1-V5. The ST-segment morphology is no longer concave but is now convex and merges with the T wave. Also noted are minimal ST-segment depressions in leads III and aVF (↑); these are reciprocal changes. Hence this is an acute anterior wall ST-segment elevation myocardial infarction (STEMI) involving the septum and apex of the left ventricle.

Interpretation of ischemic changes on ECG in the setting of a right bundle branch block (RBBB) requires an understanding of the effects of an RBBB on the phases of the QRS complex. An RBBB does not alter left ventricular depolarization, which is normal. It does result in a delay in activation of the right ventricle and primarily affects the terminal portion of the QRS complex. Since it does not affect the initial depolarization involving the left ventricle, abnormalities affecting the left ventricle can be interpreted on the ECG, including left ventricular hypertrophy, acute and chronic MI, and pericarditis. Thus Q waves that arise from left ventricular infarction are not affected. Therefore, the diagnoses of old anterior, inferior, apical, or lateral wall MIs are not affected by the presence of an RBBB.

The diagnosis of an acute MI is slightly more challenging. Disruption of the terminal phase of the QRS complex by an RBBB often results in ST-segment depression and T-wave inversion in the right precordial leads. Therefore, the more subtle findings of myocardial ischemia (unstable angina or a non–ST-segment elevation MI [NSTEMI]) of the anterior septum or apex can be obscured by the presence of an RBBB. However, a STEMI, as seen in this case, results in ST-segment elevations that will be obvious on the 12-lead ECG despite the presence of an RBBB.

This patient likely has a chronic RBBB on the basis of his known COPD. However, an RBBB can also develop acutely in the context of an MI that involves the intraventricular septum. A large anterior MI can disrupt the path of the right bundle, although this is uncommon. More often, acute anteroseptal MI results in a left bundle branch block. When an RBBB occurs as a result of an MI, the degree of myonecrosis is usually extensive and the prognosis is poorer. Occasionally, an RBBB reflects acute right ventricular dilation and pressure overload in the context of heart failure with elevated left ventricular filling pressures and pulmonary hypertension.

An acute STEMI is treated with prompt revascularization, with either a thrombolytic agent or percutaneous coronary intervention (ie, stenting), which should optimally be performed within 90 minutes of symptom onset. This depends largely on the time it takes for the patient to present to the emergency room.
A 34-year-old woman presents with crushing retrosternal chest pain occurring at rest. She has no prior medical history, although she had an uneventful term pregnancy and delivery 3 weeks prior. The pain is unrelenting and associated with mild dyspnea. Upon arrival to the emergency department, an ECG is obtained.

What does her ECG show?
What is the likely diagnosis?
What further treatment is indicated?
ECG 27 Analysis: Normal sinus rhythm, first-degree AV block, acute anterior wall ST-segment elevation myocardial infarction (STEMI), acute lateral wall MI, left atrial hypertrophy (left atrial abnormality)
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.22 sec). The P wave is negative in leads V1-V2, suggesting left atrial hypertrophy. This is a normal sinus rhythm with a first-degree AV block.

The QRS complex duration is normal (0.08 sec), and there is a normal QRS morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/390 msec).

There is significant ST-segment (↓) elevation in leads I, aVL, and V4-V6, representing an acute ST-segment elevation myocardial infarction (STEMI) of the anterolateral and lateral walls. The ST segments are convex and are merged with the T waves; there is loss of R-wave amplitude. This type of QRS complex has been termed “tombstone,” but actually the QRS complex morphology has a current of injury and resembles the fast action potential that is generated by the ventricular myocardium as a result of the rapid influx of sodium ion. This is, therefore, a monophasic action potential. Also noted are significant ST-segment depressions (↑) in leads III and aVF, which are reciprocal changes.

An acute STEMI is identified by the presence of localized ST-segment elevation, hyperacute T waves (tall, peaked, and symmetric), and reciprocal ST-segment depressions (due to the same ST-segment changes viewed from another angle or direction). The location of these changes identifies the region of the myocardium involved.

This young woman may have a coronary artery dissection, which might be due to an acute dissection of the aortic root or an isolated coronary artery dissection. Although coronary artery dissection is rare, it is an important cause of MI in young, otherwise healthy individuals. There is a particular increase in the risk for coronary dissection in the peripartum period. It has been suggested that arterial dissection during or soon after pregnancy is related to structural changes in the intima and media of the arterial wall that are produced by hormonal and hemodynamic changes. Although dissection can occur in the absence of risk factors, it may occur more frequently in conditions associated with hypertension that independently damage the arterial wall. The finding of spontaneous coronary artery dissection should trigger the search for a previously undiagnosed connective tissue disease, such as Marfan or Ehler-Danlos syndrome.

Unfortunately, the treatment for coronary artery dissection is difficult and complications of MI, including malignant ventricular arrhythmias and sudden cardiac death, can occur. Patients are often treated with systemic anticoagulation in order to minimize thrombosis at the site of the dissection. There is a potential role for coronary stenting, although this is controversial. In theory, placing an intracoronary stent may stabilize the leading edge of the dissection flap, although there is a risk for stenting into the false lumen and thus propagating the dissection and jeopardizing flow through distal vessel branches.
A 72-year-old woman presents for her first clinic visit with a new primary care provider. She has a history of hypertension and diabetes and suffered a myocardial infarction 18 months earlier. She is currently feeling well but does note heartburn on a nearly daily basis when she lies down to go to sleep at night. Concerned about her heartburn symptoms possibly being related to cardiac ischemia, her primary care physician obtains an ECG.

What does her ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 28 Analysis: Normal sinus rhythm, left anterior fascicular block, old anterior wall myocardial infarction (MI), ST-segment elevation due to aneurysm
There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm. The P wave is broad in leads II and aVF and notched in leads V2-V4, suggesting left atrial hypertrophy.

The QRS complex duration is normal (0.08 sec). There is low voltage in the limb leads (defined as a QRS complex amplitude ≤ 5 mm in each lead). The QRS axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). As there are no Q waves in leads II, III, or aVF, indicating an old inferior wall myocardial infarction (MI) accounting for the left axis deviation, this is a left anterior fascicular block. The QT/QTc intervals are prolonged (420/480 msec).

There is a QS complex (↑) in leads V1-V3 and an initial Q wave in lead V4 (^), diagnostic for an MI involving the left ventricular septum and apex. The ST segments are still elevated (↓), and the T waves (▲) are inverted. Based on the patient’s history of an MI 18 months earlier, the ST-segment elevation, which is persistent, represents an old MI and development of an apical left ventricular aneurysm. However, without this history it would be unclear whether the ST-segment elevation was persistent and the result of an aneurysm or had recently occurred in the setting of an acute MI and hence represents evolution of the ECG changes. A left ventricular aneurysm is a complication of ST-segment elevation (transmural) MI (STEMI), which results from a thin and dyskinetic region of myocardium in the distribution of a prior MI. Left ventricular aneurysms are associated with mural thrombus formation and systemic cardio-embolization (when they develop early in the setting of an acute MI), re-entrant ventricular arrhythmia, and heart failure. Patients are treated with anticoagulation with warfarin for 3 months after an acute MI if thrombus is seen. Some physicians will anticoagulate patients with a left ventricular aneurysm even in the absence of thrombus to minimize the risk for thrombus formation that occurs as a result of the acute inflammation of the myocardium. Occasionally, surgery (aneurysmectomy) is necessary when symptoms of recurrent embolization, heart failure, or intractable arrhythmias develop.

A true left ventricular aneurysm should be distinguished from a pseudo-aneurysm, both of which can arise as sequelae of MI. A pseudo-aneurysm is a contained myocardial rupture in which the outer layer of the outpouching is comprised of adherent pericardium rather than myocardium. Pseudo-aneurysms are at risk for rupture and often require surgical resection.
A 54-year-old man develops epigastric discomfort and nausea while driving home from his job as a night watchman. The sensation does not abate and when he arrives home he begins to feel diaphoretic and the chest and epigastric pain intensify. He calls emergency medical services, who obtain an ECG immediately upon arrival.

What does his ECG show?
What is the likely diagnosis?
What coronary artery is likely involved?
Podrid's Real-World ECGs

ECG 29 Analysis: Normal sinus rhythm, acute inferior wall
ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), with a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec). The T waves (+) are tall and symmetric in leads II, III, and aVF, and the ST segments (↑) are slightly elevated in these leads and have a normal concave morphology. This is an early acute inferior wall ST-segment elevation myocardial infarction (STEMI). The ST segments (▲) in leads I, aVL, and V2-V4 are depressed and there is T-wave inversion, representing reciprocal changes. However, it is possible that the ST-segment changes in leads V2-V4 are the result of posterior wall involvement. A posterior wall MI can be confirmed by obtaining posterior leads V7-V8, placed under the left scapula.

This patient should undergo urgent reperfusion therapy. If he can be transferred within 90 minutes to a tertiary care center that has cardiac catheterization capability, he should undergo primary percutaneous coronary intervention. Otherwise, he should receive thrombolytic therapy. Coronary angiography is likely to show occlusion of the right coronary artery (RCA), although an inferior MI can also be caused by occlusion of a dominant left circumflex coronary artery that supplies the posterior descending artery. A clue to the culprit vessel is the relative degree of ST-segment elevation in leads II and III. If the degree of ST-segment elevation in lead III is greater than that in lead II, the artery most likely to be involved is the RCA. If the degree of ST-segment elevation in lead II is greater than that in lead III, the occlusion is more likely to be in a dominant circumflex artery. The left circumflex artery would most likely be the culprit vessel if there were a posterior wall MI.
A 48-year-old man with dyslipidemia and hypertension develops nausea and chest pressure while mowing the lawn. He stops to rest and the pain slowly abates. A few hours later the pain recurs and he takes an antacid with no effect. Over the course of the next 2 hours the pain waxes and wanes.
until he decides to seek medical attention. By the time he arrives in the emergency department, the man is nauseated and diaphoretic and he subsequently vomits. An ECG (30A) is obtained. Based on the results, a second ECG (30B) is recorded shortly thereafter.

**What do the ECGs show?**

**What part of the myocardium is likely involved?**

**What simple noninvasive test can be performed at the bedside to clarify the diagnosis?**
Podrid’s Real-World ECGs

ECG 30A Analysis: Normal sinus rhythm, left atrial hypertrophy, counterclockwise rotation (early transition), acute inferior wall ST-segment elevation myocardial infarction (STEMI)
ECG 30A shows a regular rhythm at a rate of 74 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is negative in leads V1-V2 (^), suggesting left atrial hypertrophy.

The QRS complex duration is normal (0.08 sec). There is early transition, with a tall R wave (←) in lead V2. This is termed counterclockwise rotation, which is determined by imagining the heart as viewed from under the diaphragm. When there is counterclockwise electrical rotation, left ventricular forces are seen earlier in the precordial or chest leads, accounting for early transition with a tall R wave in lead V2. The QT/QTc intervals are normal (380/420 msec).

There is ST-segment elevation (↓) in leads II, III, and aVF, and the ST segments are convex and merge with the T waves. The T waves are biphasic in these leads. Therefore, this is an acute inferior wall ST-segment elevation myocardial infarction (STEMI) in evolution. There are also Q waves (▲) in these leads, giving the appearance of an extreme left axis deviation, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, this is the result of an inferior wall infarction and not an axis shift due to a conduction abnormality (ie, this is not a left anterior fascicular block).

There are also ST-segment depressions (↑) in leads I and aVL as well as lead V2. These are reciprocal ST-segment changes, although the ST-segment depression in lead V2 might suggest posterior wall involvement. There are also nonspecific ST-T wave changes in leads V4-V6 (+).

continues
Podrid’s Real-World ECGs

ECG 30B Analysis: Right-sided leads, right ventricular infarction
In ECG 30B, the limb leads are identical to those in ECG 30A and they show an acute inferior wall STEMI. However, the precordial leads are different and there is loss of R waves (↓) in leads V3-V6 with QS complexes noted. These are right-sided leads, which are often obtained in patients presenting with an acute inferior wall STEMI to evaluate for the presence of an infarction of the free wall of the right ventricle. Although the inferior wall of the right ventricle, which is contiguous with the inferior wall of the left ventricle, is often involved with an inferior wall MI, involvement of the free wall of the right ventricle, which is a true right ventricular wall infarction, is less common. The ST-segment elevation (+) seen in right-sided leads RV3-RV5 indicates that a right ventricular infarction is also present along with the inferior wall infarction. The presence of a right ventricular infarction means that the right coronary artery is the culprit vessel.

Establishing the presence of a right ventricular infarction is of clinical importance as the patient is volume dependent. Any change in venous return or vascular volume has major effects on right ventricular filling and stroke volume from the right ventricle, which then impacts left ventricular filling and left ventricular stroke volume. For example, the administration of nitroglycerin will reduce venous return to the right ventricle and in the presence of significant right ventricular dysfunction left ventricular preload and hence left ventricular stroke volume will be reduced, resulting in hypotension. A similar situation may occur if the patient receives a diuretic, which is often administered when there is evidence of pulmonary vascular congestion. The occurrence of hypotension might suggest the development of cardiogenic shock, rather than an issue of significant reduction in left ventricular preload.

In this patient, posterior leads placed under the left scapula (ie, leads V7-V8) should also be obtained given the ST-segment depression in lead V2, which may represent posterior wall involvement. Although the tall R wave in lead V2 is most likely due to early transition (counterclockwise rotation), there is a possibility that it may be a posterior wall MI. However, this is not likely given the absence of a tall R wave in lead V1.
A 64-year-old woman with diabetes and a long smoking history presents with left-sided chest discomfort that awakened her from her sleep. The discomfort was unrelenting, and she sought immediate medical attention. An ECG (31A) is obtained upon arrival to the emergency department, and a second ECG (31B) is obtained before the patient is given nitroglycerin.
What do the ECGs show?
What is the rationale for obtaining ECG 31B?
What is the likely diagnosis?
What coronary artery is likely to be involved?
Podrid’s Real-World ECGs

ECG 31A Analysis: Normal sinus rhythm, counterclockwise rotation (early transition), premature atrial complex, acute inferior wall ST-segment elevation myocardial infarction (STEMI)
The rhythm in ECG 31A is regular at a rate of 72 bpm. There are P waves (*) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm. The eighth QRS complex is early (+) and the P wave before it (^) has a different morphology (ie, it is negative in leads V1-V3) compared with the other P waves. This is a premature atrial complex.

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/390 msec). There is early transition with a tall R wave (←) in lead V2. This is termed counterclockwise rotation, determined by imagining the heart as viewed from under the diaphragm. When there is counterclockwise electrical rotation, left ventricular forces are seen earlier in the precordial or chest leads, accounting for early transition with a tall R wave in lead V2.

There is ST-segment elevation (↓) in leads II, III, and aVF, and the ST segments have a normal convex morphology. This is an acute inferior wall ST-segment elevation myocardial infarction (STEMI). ST-segment depression (▲) can be seen in lead aVL, which represents a reciprocal change.

continues
Podrid’s Real-World ECGs

ECG 31B Analysis: Acute inferior wall ST-segment elevation myocardial infarction (STEMI), right-sided leads, no right ventricular infarction
In ECG 31B, the P waves (↑), QRS complexes, and inferior ST-segment elevation (↓) are the same as in ECG 31A. Therefore, there is an acute inferior wall STEMI present. The precordial or chest leads are different from those in ECG 31A, and there is loss of R-wave amplitude (+) in leads V4-V6. These are right-sided leads and the ECG was recorded with the precordial leads on the right side of the chest. No ST-segment elevation (▼) is seen in leads RV3-RV6. Hence there has not been any acute involvement of the right ventricular free wall (ie, there is no right ventricular infarction present). This is typical for occlusion of either a dominant circumflex artery or the right coronary artery after the takeoff of the principal right ventricular branch.

Right-sided leads should be obtained routinely in patients presenting with an inferior wall STEMI as right ventricular involvement is not uncommon. When there is a right ventricular infarction, the patient is volume dependent (ie, there needs to be adequate volume in the setting of reduced right ventricular function for a normal stroke volume to fill the left ventricle). Situations that reduce right ventricular filling, such as administration of nitrates (reducing venous return and preload) and diuretics (reducing vascular volume), may result in underfilling of the right ventricle and hence underfilling of the left ventricle with resultant hypotension.
A 57-year-old woman is awakened from sleep in the early morning with sudden-onset chest pressure. She calls emergency medical services, who obtain an ECG at her bedside. She is then given sublingual nitroglycerin, and the pain resolves within 5 minutes. A second ECG is then obtained.
What does the initial ECG (32A) show?
What does the second ECG (32B) show?
What is the likely diagnosis?

ECG 32B
ECG 32A Analysis: Sinus tachycardia, acute inferior wall transmural ischemia
due to vasospasm (variant or Prinzmetal’s angina), ST-segment alternans
ECG 32A was obtained at 3:38 AM because of the acute-onset substernal chest pressure awakening the patient from sleep. There is a regular rhythm at a rate of 100 bpm, with a P wave (*) before each QRS complex and a stable PR interval (0.16 sec). The P wave is upright in leads II, aVF, and V4-V6 and negative in lead aVR. This is a sinus tachycardia.

The QRS complex duration (0.08 sec) and morphology are normal. The QRS axis is not able to be determined accurately because of very significant ST-segment elevation (↓) in leads II, III, and aVF. ST-segment elevation (↓) can also be seen in lead V6. The ST segments are convex and are merged with the T waves. There is also ST-segment depression (^) in leads I, aVL, and V2-V4. The ECG is typical for an acute inferior wall ST-segment elevation myocardial infarction (STEMI), and the ST-segment depressions are reciprocal changes, although in leads V2-V3 the depressions could represent involvement of the posterior wall of the left ventricle.

Interestingly, there is evidence of ST-segment alternans (ie, beat-to-beat changes in the height of the ST segment), best seen in leads aVL and aVF. This has primarily been described with coronary artery vasospasm and transient occlusion of a coronary artery during angioplasty. It is a marker of more severe transmural ischemia.
Podrid's Real-World ECGs

ECG 32B Analysis: Junctional tachycardia
ECG 32B was obtained at 4:00 AM, about 10 minutes after the administration of sublingual nitroglycerin and the relief of the chest discomfort. The rhythm is regular at a rate of 120 bpm. There are no obvious P waves seen before or after any of the QRS complexes, which have a normal duration (0.08 sec) and morphology. This is a junctional tachycardia. The QT/QTc intervals are slightly prolonged (320/450 msec).

The ST-segment elevations and depressions have resolved, and the ST segments are now normal and at baseline (as defined by the T-QRS segment). There are persistent T-wave inversions in leads I, aVL, and V2-V4 (^). Although ECG 32A showed evidence of acute transmural ischemia and what appeared to be an acute ST-segment elevation myocardial infarction (STEMI), the rapid resolution of the ECG changes (i.e., the transmural ischemia) and relief of chest discomfort are characteristic of coronary artery vasospasm, which is termed variant, vasospastic, or Prinzmetal’s angina. It results from acute spasm of a coronary artery, which most commonly occurs at night. The right coronary artery is most often involved. The diagnosis of vasospastic or Prinzmetal’s angina is based on the presence of ST-segment elevation that is quickly reversed with nitroglycerin. The mechanism for the vasospasm is believed to be based on reflex activation of the sympathetic nervous system and increased α tone due to elevated vagal tone that occurs at night. The stimulation of α receptors in the coronary arteries results in vasospasm of susceptible coronary artery segments.

Although there is transmural ischemia, an acute myocardial infarction is uncommon as local autoregulatory mechanisms often occur, resulting in vasodilation. This may be due to the release of adenosine during ischemia. The initial treatment for vasospastic angina is a calcium-channel blocker and/or nitrates. The vasospasm and transmural ischemia are the same as that which is seen with cocaine-induced vasospasm.
A 22-year-old man with a strong family history of premature coronary artery disease presents to the emergency department with sudden-onset chest heaviness. He has had no prior episodes. He is noted in the emergency department to have several xanthomata on extensor surfaces.

What does his ECG show? What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 33 Analysis: Normal sinus rhythm, first-degree AV block, premature ventricular complexes, premature atrial complex, acute lateral wall ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.22 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm with a first-degree AV block. There are four wide QRS complexes (third, fifth, eighth, and ninth) (+) that are not preceded by a P wave and have an abnormal morphology. These are premature ventricular complexes. In addition, the 15th QRS complex (^) is early. There is a P wave of a different morphology (▼) preceding it, and the QRS complex morphology is the same as the sinus complexes. This is a premature atrial complex.

The sinus QRS complex duration is normal (0.10 sec). There is ST-segment elevation (↓) in leads I, aVL, and V6, and the ST segments have a convex morphology, merging with the T waves. This is an acute lateral wall ST-segment elevation myocardial infarction (STEMI). There is also significant ST-segment depression (↑) in leads II, III, aVF, and V1-V4; these are reciprocal changes. The premature ventricular complex in leads I and aVL also has ST-segment elevation (▲), indicating that evidence of an acute STEMI can be identified in QRS complexes that are generated by direct activation of the ventricular myocardium, bypassing the normal His-Purkinje pathway (ie, ventricular complexes, paced complexes, or supraventricular complexes with a left bundle branch block). This is based on the Sgarbossa criteria.

### Sgarbossa Criteria for Acute Myocardial Infarction in Left Bundle Branch Block (and Paced Rhythm and Ventricular Complex)

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST-segment elevation ≥ 1 mm that is in the same direction (concordant) as the QRS complex in any lead</td>
<td>5</td>
</tr>
<tr>
<td>ST-segment depression ≥ 1 mm in any lead from V1 to V3</td>
<td>3</td>
</tr>
<tr>
<td>ST-segment elevation ≥ 5 mm that is discordant with the QRS complex (ie, associated with a QS or rS complex)</td>
<td>2</td>
</tr>
</tbody>
</table>

A minimum score of 3 is required for a specificity of 90%.

MI is quite rare in this age group, raising the possibility of an unusual etiology. MI in the young can be related to vasculitis (such as Kawasaki’s disease), chronic inflammatory states, coronary dissection, coronary artery embolism from endocarditis or atrial fibrillation, or accelerated atherosclerosis from an inherited dyslipidemia. This young man with a strong family history of coronary disease and physical exam notable for xanthomata likely has an inherited dyslipidemia. The most common of these, familial hypercholesterolemia, is a monogenic, autosomal disorder caused by a defect in the low-density lipoprotein (LDL) receptor. Patients who harbor the homozygous form of the disease often present with MI during the second or third decade of life. Patients require very aggressive cholesterol-lowering therapy with high-dose statins, inhibitors of cholesterol absorption, and often apheresis.
A 55-year-old man with diabetes presents to a walk-in clinic for evaluation of 1 week of increased dyspnea on exertion. He does not recall any episodes of prolonged chest pain but decided to seek medical attention when the dyspnea persisted for more than a week. Prior to this episode he was in good physical condition and able to complete his work as a mail carrier without difficulty, but recently he has had trouble walking his route.

What does his ECG show?
What is the likely diagnosis?
ECG 34 Analysis: Normal sinus rhythm, old inferior wall myocardial infarction (MI), nonspecific ST-segment changes, left axis deviation due to an old inferior wall MI
There is a regular rhythm at a rate of 58 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS duration is normal (0.08 sec), as is the morphology. Although the axis appears to be extremely leftward due to a negative QRS complex in leads II and aVF and a positive QRS complex in lead I (ie, axis between −30° and −90°), this is a result of deep Q waves (+) in leads II and aVF. This, along with the Q wave in lead III (+), is diagnostic for an inferior wall myocardial infarction (MI). Hence the left axis deviation is not the result of a conduction abnormality (ie, it is not a left anterior fascicular block in which the QRS complex has an rS morphology) but of an inferior wall MI (ie, it has a Qr morphology). The QT/QTc intervals are normal (400/390 msec).

There is minor ST-segment elevation (↓) and T-wave inversion (▲) in leads II, III, and aVF, suggesting that the inferior wall MI may have been relatively recent (within the past few days or weeks). ST-segment flattening (↑) is also noted in leads I, aVL, and V4, which is a non-specific abnormality.

A chronic or old MI is identified by the presence of abnormal Q waves, defined as any Q wave in leads V1-V3 or a Q wave 0.04 second or longer in leads I, II, aVF, or V4-V6 (in two or more contiguous leads) and at least 1 mm in depth. However, Q waves may be normal and are ignored in lead III (unless they are also in leads II and aVF), in lead V1 (unless also in lead V2), and in lead aVL (unless Q-wave height ≥ 50% R-wave height). The QS interval in leads V1-V2 may also be normal variant.

T-wave inversions are usually present in the leads showing Q waves. Persistent ST-segment elevation weeks, months, or years after an infarction suggests the presence of an aneurysm. The location of Q waves identifies the region of the left ventricle that is involved:

- Inferior wall MI: Q waves in leads II, III, and aVF
- Anteroseptal MI: Q waves in leads V1-V2
- Anteroapical MI: Q waves in leads V3-V4
- Anterolateral MI: Q waves leads V5-V6
- Anterior wall MI: Q waves in two or more contiguous precordial leads
- Lateral MI: Q waves in leads I and aVL

continues
• Posterior MI: Tall R wave in lead V1 (R-wave amplitude > 7 mm or R/S > 1) with a duration 0.04 second or longer. This is typically seen in association with inferior wall MI and in the absence of other conditions associated with a tall R wave in lead V1, including evidence of right ventricular hypertrophy, Wolff-Parkinson-White pattern, dextrocardia, lead misplacement, recording of right-sided leads, Duchenne muscular dystrophy, or hypertrophic cardiomyopathy. On occasion it may be a normal variant or due to counterclockwise rotation.

Although the patient does not give a history of a prior MI, silent infarctions are not uncommon and up to 40% may go unrecognized, especially those of the inferior wall. This may be seen even more frequently in patients with diabetes, who often do not have typical symptoms of an MI. They may have other symptoms, such as fatigue, shortness of breath, nausea, and diaphoresis, which may often be overlooked or not considered to be of any importance. Given the presenting symptoms, it is likely that the patient has developed signs and symptoms of heart failure as a result of significant left ventricular myocardial dysfunction due to the previous infarction. It is also possible that the patient may have mitral regurgitation as a result of papillary muscle dysfunction from a previous MI.
An 88-year-old man with insulin-dependent diabetes, hypertension, and a 60 pack-year smoking history presents to his primary care physician with dyspnea on exertion and a 10-pound weight gain. On exam, he has evidence of jugular venous distention, bilateral rales, and pedal edema. Echocardiography documents multiple wall motion abnormalities with areas of akinesia and a left ventricular ejection fraction of 25%.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

**ECG 35 Analysis:** Sinus bradycardia; first-degree AV block; intraventricular conduction delay; old lateral, anteropapical, and anterolateral myocardial infarction (MI); old inferior wall MI; old posterior wall MI; counterclockwise rotation (early transition)
There is a regular rhythm at a rate of 46 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.22 sec). The P-wave morphology is normal. This is a normal sinus rhythm with a first-degree AV block.

The QRS complex duration is slightly prolonged (0.11 sec), and there appears to be a delay in right ventricular conduction as there is an R′ (←) in lead V1 and prominent S waves (↑) in leads I and V5-V6. This is an intraventricular conduction delay. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (460/400 msec and 430/380 when corrected for the prolonged QRS complex duration).

There are significant Q waves (↑) in leads I, aVL, and V3-V6, diagnostic for an old lateral, anteropical, and anterolateral myocardial infarction (MI). There are also significant Q waves (▲) in leads II, III, and aVF, diagnostic for an old inferior wall MI. Lastly, there is a tall R wave (→) in lead V1; along with the inferior wall MI this indicates an old posterior wall MI. The tall R wave in lead V2 (↓), along with the tall R wave in lead V1, may also be due to the posterior wall MI. However, it may also be the result of counterclockwise rotation or early transition. This is due to a shift of the electrical axis in the horizontal plane and is established by imagining the heart as viewed from under the diaphragm. When the electrical axis is shifted in a counterclockwise direction, left ventricular forces occur early and are prominent in the right precordial leads (ie, a tall R wave in lead V2). There is ST-segment elevation in leads V1-V3. The etiology for this is not certain. It may actually represent ST-segment depression seen in posterior leads V7-V8 or early repolarization.

This patient has suffered multiple prior MIs involving several territories. As a result, there is a significant decline in systolic function resulting in heart failure. There is no evidence of active myocardial ischemia on the ECG, so clinical care should focus on medical therapy for heart failure, including diuresis to achieve euvolemia, afterload reduction with an angiotensin-converting enzyme inhibitor, and long-acting β-blockade. It has been proposed that establishing the presence of a viable hibernating myocardium in patients with an ischemic cardiomyopathy is important and that revascularization would help improve left ventricular function if there is substantial viable myocardium. However, a recent study (STICH) has reported that this approach does not lead to any improvement in outcome when compared with medical therapy and that establishing hibernation with imaging does not help. In this patient, there are multiple areas of infarction with fibrosis and akinesis that will not recover with revascularization.
A 56-year-old woman presents to a cardiologist for evaluation of exertional chest pressure. Over the course of the previous 3 weeks, the intensity and frequency of the chest discomfort have increased. She reports that she had a myocardial infarction about 3 years earlier. Her cardiologist diagnoses unstable angina on a clinical basis and arranges for her to undergo urgent coronary angiography. He performs an ECG in his office. On angiography she is found to have a chronic total occlusion of her right coronary artery with left-to-right collaterals to the posterior descending artery and an 80% mid-circumflex stenosis.

What does the ECG show? Which lesion is likely to be responsible for her angina?
Podrid’s Real-World ECGs

ECG 36 Analysis: Normal sinus rhythm, old inferior wall myocardial infarction (MI), old posterior wall MI, counterclockwise rotation (early transition)
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.15 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS duration is normal (0.08 sec). The QT/QTc intervals are normal (320/390 msec). Although the axis is leftward (between 0° and -30°), this is the result of significant Q waves (+) in leads II, III, and aVF, diagnostic for an inferior wall myocardial infarction (MI). In addition, there is a tall R wave in lead V1 (←), which, along with an inferior wall MI, is characteristic of posterior wall involvement with the infarction; hence this is an inferoposterior wall MI. The tall R wave in lead V2 (→) is probably the result of counterclockwise rotation or early transition. This is due to a shift of the electrical axis in the horizontal plane and is established by imagining the heart as viewed from under the diaphragm. When the electrical axis is shifted in a counterclockwise direction, left ventricular forces occur early and are prominent in the right precordial leads (ie, a tall R wave in lead V2).

The old inferoposterior MI on the ECG corresponds with the finding of an occluded right coronary artery on angiography. It is likely that her unstable angina is attributable to the stenosis of the mid-circumflex artery. Not uncommonly, lesions of the left circumflex artery are electrically silent and there are no acute ECG changes seen with either ischemia or acute MI. Percutaneous coronary intervention of the circumflex artery is indicated. It has been shown that there is no benefit to opening a chronically occluded artery. Hence there would be no reason to perform a percutaneous coronary intervention of the right coronary artery, especially since there are left-to-right collaterals to the distal right coronary artery and posterior descending artery.
An 81-year-old woman is transferred from the coronary care unit to a cardiac step-down unit 1 week after suffering a large myocardial infarction (MI) complicated by hypotension and vascular congestion. She is currently feeling well and is pain free. She denies any lightheadedness or pre-syncope.

What does her ECG show?
What is the likely diagnosis?
ECG 37 Analysis: Normal sinus rhythm, first-degree AV block, right bundle branch block, left anterior fascicular block (bifascicular block), old anteroseptal and anteroapical MI
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.24 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm with a first-degree AV block.

The QRS complex duration is prolonged (0.14 sec), and the morphology has a right bundle branch block pattern with a broad S wave (→) in leads I and V5-V6 and a tall, broad R wave in lead V1 (→). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I after eliminating the terminal S wave, which is the result of a right bundle branch block and delayed right ventricular activation, and negative QRS complex in leads II and aVF). This is termed a left anterior fascicular block. It should be noted that the broad S wave in lead I caused by the right bundle branch block gives the appearance that the QRS complex is negative in this lead; if this actually were a negative QRS complex, this would be an indeterminate axis. However, the S wave should not be considered as part of axis determination and, when eliminated, the QRS complex is indeed positive. The presence of a right bundle branch block and left anterior fascicular block is termed bifascicular block. There is also a first-degree AV block, and this has often been thought to represent trifascicular disease or block. However, this is not correct unless the etiology for the first-degree AV block is known to be the result of conduction slowing in the remaining fascicle (ie, left posterior fascicle). However, the first-degree AV block may also be due to slow conduction through the AV node, in which case this would be bifascicular block associated with AV nodal conduction slowing. The QT/QTc intervals are normal (440/440 msec and 380/380 when corrected for the prolonged QRS complex duration).

There are significant Q waves in leads V1-V4 (^), diagnostic for an anteroseptal and anteroapical myocardial infarction (MI). The left anterior fascicle and most of the fibers of the right bundle branch are supplied by the septal perforators of the left anterior descending artery. Therefore, an anteroseptal MI resulting in myocardial fibrosis can lead to a bifascicular block. This degree of conduction disease following MI is an indicator of an extensive amount of infarcted tissue and is a poor prognostic factor. In the absence of symptoms, this degree of conduction disease is not an indication for permanent cardiac pacing, although the clinician must remain vigilant for signs of high-grade heart block.
A 48-year-old man is transferred from a rural health clinic to an emergency department at a tertiary care center for evaluation of chest pain. The patient had been on a week-long hunting trip with his friends when he developed chest discomfort. The discomfort persisted for about an hour and eventually resolved partially with rest. He had continued intermittent chest discomfort for the next 48 hours and eventually decided to leave his hunting troop and seek medical attention. Upon arrival at a local medical clinic, he has continued to complain of 1/10 chest discomfort. An ECG is obtained.

What does his ECG show?

What is the likely diagnosis?
ECG 38 Analysis: Normal sinus rhythm, anteroapical myocardial infarction (MI) (age indeterminate), anteroapical aneurysm versus MI in evolution, nonspecific T-wave abnormalities
There is a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/430 msec). There are Q waves (▼) (QS complexes) in leads V2-V3 and an initial Q wave in lead V4 (^), diagnostic for an anterioapical myocardial infarction (MI). In addition, there are T-wave inversions (↑) in these leads as well as in leads I, aVL, and V5-V6 that are nonspecific. There is minimal ST-segment elevation (↓) in leads V2-V3. This might be indicative of a recent MI with ongoing evolution of the ECG changes or an old infarction with the presence of an aneurysm of the infarcted area.

In this clinical setting (ie, the acute onset of symptoms about 1 week before being seen), the ECG findings likely represent an evolving MI. Since the patient presented late into his course, his ECG shows evidence of transmural myocardial necrosis. His ongoing chest discomfort may be evidence of some persistent ischemia in the remaining viable tissue. He is at high risk for mechanical complications of MI, including free wall rupture, ventricular septal defect, left ventricular aneurysm or pseudoaneurysm, and pericarditis. Cardiac catheterization is indicated due to the continued chest discomfort. Although the finding of a totally occluded vessel this long after the acute event would not require percutaneous coronary intervention if the patient were asymptomatic, the ongoing symptoms suggest areas of ischemia and hence percutaneous coronary intervention for a totally occluded vessel might be useful for symptom relief.
A 78-year-old man with longstanding hypertension and a prior myocardial infarction (MI) presents to his cardiologist’s office with progressive chest discomfort on exertion. Over the course of the previous 6 weeks he has noticed increasing chest discomfort at progressively lower workloads. He is now having chest discomfort with walking only about half a flight of stairs. On his cardiologist’s recommendation, he undergoes urgent cardiac catheterization and is found to have a right dominant circulation with a totally occluded proximal left anterior descending artery (LAD). The distal LAD fills via right-to-left collaterals. There is an 80% mid–right coronary artery stenosis.

What does his ECG show?
What is the likely diagnosis?
Which vessel is likely to be causing his symptoms?
Podrid's Real-World ECGs

ECG 39 Analysis: Normal sinus rhythm, first-degree AV block, left atrial hypertrophy (abnormality), old lateral wall MI, old anteroapical MI, nonspecific T-wave abnormalities, right axis deviation due to an old lateral wall MI
There is a regular rhythm at a rate of 94 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.22 sec). The P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm with a first-degree AV block. The P waves (+) in leads II and aVF are broad and notched, characteristic of left atrial hypertrophy (abnormality).

The QRS complex duration is normal (0.08 sec). The QT/QTc intervals are normal (340/430 msec). The axis is rightward, between +90° and +180°, but this is the result of Q waves (QS complex) (^) in leads I and aVL, diagnostic for an old lateral wall myocardial infarction (MI). Hence the right axis deviation (negative QRS complex in lead I and positive QRS complex in lead aVF) is the result of a lateral wall infarction (QS morphology) and not a conduction abnormality resulting from a left posterior fascicular block, which would show a QRS complex with an rS morphology in leads I and aVL. Other causes of a right axis deviation include right ventricular hypertrophy (with a tall R wave in lead V1), right-to-left arm lead switch (which would also result in negative P waves in leads I and aVL), Wolff-Parkinson-White pattern (with a short PR interval and wide QRS complex due to a delta wave), or dextrocardia (with negative P waves in leads I and aVL). When there are no other causes for a right axis deviation, a left posterior fascicular block can be diagnosed. There are also Q waves (QS complexes) (↓) in leads V2-V4, diagnostic for an old anteroapical MI. Also noted are nonspecific T-wave abnormalities (▲) in the limb leads and leads V5-V6.

This patient has evidence of a completed infarction in the lateral and anteroapical territories. These have likely been caused by prior occlusion of his left anterior descending artery. It is most likely that the stenotic right coronary artery is causing incomplete collateral flow to the remaining anterior wall territory as well as the inferior wall. Revascularization of the right coronary artery would likely result in an improvement in his symptoms.
A 58-year-old woman is admitted to the cardiac care unit for management of decompensated heart failure. The treating physicians notice a marked left axis deviation on her ECG.

What does her ECG show?

What is the likely diagnosis and cause of her left axis deviation?
Podrid's Real-World ECGs

**ECG 40 Analysis:** Sinus bradycardia, old inferior wall myocardial infarction (MI), old anterior wall MI, left axis deviation due to an old inferior wall MI
There is a regular rhythm at a rate of 54 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a sinus bradycardia. The P-wave morphology is normal.

The QRS duration is normal (0.08 sec). The QT/QTc intervals are normal (440/420 msec). There are significant Q waves (^) in leads II, III, and aVF, diagnostic for an old inferior wall myocardial infarction (MI). As a result of the deep Q waves the axis is extremely leftward (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, this is not a conduction abnormality resulting from a left anterior fascicular block (in which the QRS complex would have an rS morphology) but rather is due to the inferior wall infarction (the QRS complex has initial Q waves and a Qs morphology). There are also Q waves (QS complex) (↓) in leads V1-V5 and a Q wave in lead V6 (▲), diagnostic for an old extensive anterior wall MI. The T waves (↑) are inverted in leads V2-V6 as well as leads I and aVL.
A 58-year-old man is admitted to the hospital with a prolonged episode of chest discomfort. His initial laboratory studies are normal, and an ECG (41A) is obtained.
The following morning, his cardiac biomarkers become positive, with elevated creatine kinase (CK), CK-MB, and troponin I levels. A second ECG (41B) is obtained.

What do the ECGs show?
What is the likely diagnosis?
ECG 41A Analysis: Normal sinus rhythm, counterclockwise rotation (early transition), prolonged QT interval, nonspecific T-wave abnormalities
ECG 41A shows a regular rhythm at a rate of 68 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P-wave morphology is normal.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (430/460 msec). The tall R wave in lead V2 (←) is the result of early transition or counterclockwise rotation (ie, an axis shift in the horizontal plane). This is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation the left ventricular forces develop early in the chest leads and are seen in the right precordial leads, accounting for the tall R wave in lead V2. There are no acute changes, but diffuse nonspecific T-wave changes (flattening) are seen.
ECG 41B Analysis: Normal sinus rhythm, acute posterior wall myocardial infarction (MI)
ECG 41B was obtained on the day following admission. As noted, cardiac biomarkers were now positive with elevated creatine kinase (CK), CK-MB, and troponin levels. ECG 41B is identical to ECG 41A except for the presence of a tall R wave in lead V1 (→) and ST-segment depressions below the baseline (TP segment) in leads V1-V4 (↑). This is characteristic of an acute true posterior wall myocardial infarction (MI; without inferior wall involvement). At the time of catheterization there was an acute thrombotic occlusion of the first obtuse marginal branch of the left circumflex artery.

In light of the clinical presentation, a tall R wave in lead V1 (R-wave amplitude > 7 mm or R/S > 1) with a duration of 0.04 second or longer, when associated with ST-segment depression in leads V1-V4, is indicative of a posterior wall myocardial MI. The posterior wall MI can be confirmed by obtaining posterior leads, placed below the left scapula (leads V7-V8). The presence of ST-segment elevation in these leads would be seen with a posterior wall MI. As the patient has ST-segment depressions in leads V1-V4, which are reciprocal changes, ST-segment elevation would likely be seen. In addition, the QRS complex would show a Q-wave or QS pattern, indicating the MI. Although a posterior wall MI is typically seen in association with inferior wall MI, there may be a true isolated posterior wall infarction often associated with a lesion of the obtuse marginal branch. Although there are other causes of a tall R wave in lead V1, the presence of ST-segment depression in leads V1-V4 supports the diagnosis of a posterior wall MI. Other causes for a tall R wave in lead V1 that need to be considered include right ventricular hypertrophy, Wolff-Parkinson-White pattern, dextrocardia, lead misplacement, Duchenne muscular dystrophy, and hypertrophic cardiomyopathy. On occasion it may be a normal variant or due to counterclockwise rotation.
Notes
A 64-year-old man presents with complaints of epigastric and mid-chest discomfort. The epigastric pain began about 2 weeks earlier and was associated with belching and nausea. He initially attributed the pain to indigestion and did not seek medical attention. The pain eventually subsided but was replaced with a sharp chest pain, made worse by inspiration or supine positioning. His presenting ECG is shown.

What does his ECG show?
What is the likely diagnosis?
**Podrid's Real-World ECGs**

**ECG 42 Analysis:** Normal sinus rhythm, old inferior wall myocardial infarction (MI), pericarditis
There is a regular rhythm at a rate of 98 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/410 msec). There are Q waves (^) in leads II, III, and aVF that are diagnostic of an old inferior wall myocardial infarction (MI). The Q waves in leads V4-V6 (↑) are small and narrow and represent septal depolarization.

Diffuse J-point and ST-segment elevation (↓) above the baseline (TP segment) can be seen in leads I, II, aVR (ST-segment depression in this lead is actually ST-segment elevation), aVL, aVF, and V2-V6. Diffuse ST-segment elevation is characteristic of pericarditis. Noted is that the concave morphology of the ST segment is maintained and the T-wave morphology is normal (asymmetric). Also noted is slight PR-segment depression (PR segment compared with the baseline [TP segment]), which is most obvious in leads I and II.

The characteristic ECG findings in patients with pericarditis are as follows:

- There is no reciprocal ST-segment depression.
- T waves are normal (ie, asymmetric).
- PR interval depression may be seen. However, its absence does not exclude pericarditis.
- T-wave inversion may occur after ST segments return to isoelectric baseline.

Based on his history, this patient likely suffered an inferior MI at the time of his initial symptom onset about 2 weeks before presentation. The description of his chest discomfort is consistent with angina. This is manifest by the inferior Q waves. His subsequent chest pain is different and is consistent with pericarditis, suggesting that he has now developed a post-infarction pericarditis. MI can be associated with pericarditis at various stages of the evolution and healing process. Infarction pericarditis occurs simultaneously with the onset of MI and is caused by acute injury to the myocardium and subsequent myocardial and pericardial inflammation (sympathetic effusion). Pericarditis that occurs after the initial infarction period has resolved (3 days up to several months) is termed postcardiac injury syndrome (also called Dressler’s syndrome) and is believed to be caused by an immune mechanism. Myocardial antigens are released with the infarction, resulting in the production of anti-myocardial antibodies. The development of antigen–antibody immunocomplexes results in a serum sickness episode that can produce fever, serositis (pleural and pericardial), acute renal insufficiency, and arthritis and arthralgias. Pericarditis of the same etiology can also be seen after cardiac surgery and is termed a postcardiotomy syndrome.
Notes
A 19-year-old man with no prior medical history presents with 2 days of intermittent chest discomfort and fever. He had been seen in the medical walk-in clinic 1 week earlier for an upper respiratory tract infection and was treated with a short course of antibiotics.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 43 Analysis: Atrial fibrillation, pericarditis
There is an irregularly irregular rhythm at an average rate of 102 bpm. There are no P waves before or after any QRS complexes. This is atrial fibrillation.

The QRS complex duration is normal (0.08 sec), and there is a physiologic left axis deviation, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (320/420 msec). The ST segments are elevated (^) above the baseline (TP segment) in leads I, II, aVR (ST-segment depression in this lead is actually ST-segment elevation), aVL, and V3-V6. Therefore, this is pericarditis. The concave morphology of the ST-segment is maintained, and the T-wave morphology is normal (asymmetric).

This patient likely has a viral pericarditis. The most common viruses implicated in viral pericarditis are the echoviruses, coxsackieviruses A and B, adenoviruses, and HIV. Atrial fibrillation is commonly associated with pericarditis and often resolves after resolution of the pericarditis. This patient was treated with a 2-week course of ibuprofen and achieved full resolution of his symptoms. Initial therapy for symptomatic acute pericarditis involves nonsteroidal anti-inflammatory drugs (NSAIDs, such as ibuprofen or aspirin) or colchicine. Persistent severe symptoms despite NSAIDs and colchicine can be treated with a brief course of steroid therapy. An echocardiogram should be obtained to evaluate for the presence of a pericardial effusion. Although uncommon, a large effusion with evidence of tamponade would require pericardiocentesis.
A 26-year-old athlete attends a pre-participation medical examination. No abnormalities are found on physical examination, and he has no current symptoms of cardiovascular disease. An ECG is obtained.

What does his ECG show?
What is the likely diagnosis?
ECG 44 Analysis: Sinus bradycardia, tall QRS voltage, early repolarization
There is a regular rhythm at a rate of 56 bpm. There are P waves (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6, and they have a normal morphology. This is sinus bradycardia.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/390 msec). The QRS amplitude (voltage) is increased in lead V4 (42 mm) and in lead II (23 mm). Although the voltage present may meet criteria for left ventricular hypertrophy (ie, R-wave amplitude in any limb lead ≥ 20 mm and S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm), the tall QRS amplitude is most likely a normal variant, consistent with a young age and a very fit and athletic individual. Although the T waves are prominent in leads V3-V4, they are asymmetric and hence normal. They are likely prominent as the result of the tall QRS voltage. There is also J-point and ST-segment elevation (↑) above the baseline (TP segment), noted primarily in leads V3-V5. This is termed early repolarization and it is a normal finding, often seen with left ventricular hypertrophy or in young subjects with or without prominent R-wave amplitude.
A 48-year-old body builder presents for a routine examination with his primary care physician. His blood pressure is noted to be 146/88 mm Hg and is symmetric in both arms. An ECG is obtained.

What does his ECG show?

What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 45 Analysis: Normal sinus rhythm, possible left ventricular hypertrophy (LVH), early repolarization, prominent but normal T waves
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. They have a normal morphology. This is normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec). The QRS complex morphology is normal, although there is an R' in lead V2 (+), which is a normal variant, suggesting a minimal right ventricular conduction delay. The R-wave amplitude (voltage) in lead V5 is increased (30 mm) (+), and the S-wave depth (voltage) in lead V2 is 15 mm (+) (S-wave depth in lead V2 + R-wave amplitude in lead V5 ≥ 45 mm), which meets a voltage criterion for left ventricular hypertrophy (LVH) (S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). However, this patient is young and healthy and hence the prominent QRS complex amplitude may be a normal finding. J-point and ST-segment elevation (+) are noted in leads V2-V4, which is termed early repolarization. This is often seen with tall QRS complex amplitude or voltage.

The T waves are tall and peaked, especially in leads V2-V5 (+). However, they are asymmetric and hence normal. They likely appear tall and peaked as a result of the prominent QRS complex amplitude.

Normal T waves are asymmetric regardless of amplitude. They have a slower upstroke and faster downstroke. T waves that are tall, peaked and, most importantly, symmetric are hyperacute and are seen with hyperkalemia (systemic) or acute myocardial infarction (localized hyperkalemia).

Physiologic LVH, if indeed present, can develop in well-conditioned athletes, especially in those performing isometric exercise, such as weightlifting. LVH often resolves during a period of inactivity. Although LVH is a physiologic response to a high level of exercise, it can be associated with rhythm disturbances. Unlike pathologic LVH seen with hypertension, fibrosis of the myocardium, which occurs with LVH due to hypertension, generally does not develop with physiologic LVH. Hence diastolic dysfunction is less likely to occur.
A 56-year-old man with hypertension presents to a walk-in clinic with complaints of diarrhea and headaches. He reports that he developed diarrhea about 10 days earlier and has had malaise and headaches on a daily basis; oral intake has been poor. He has been taking high doses of ibuprofen to ease the pain but with little effect. He takes lisinopril for hypertension and is on a low-dose aspirin regimen. His primary care physician draws blood for laboratory testing in his office and notes elevated blood urea nitrogen (BUN) and creatinine levels.

What does his ECG show? What is the likely diagnosis?
ECG 46 Analysis: Sinus bradycardia, left ventricular hypertrophy, hyperacute T waves (hyperkalemia), prolonged QT interval
There is a regular rhythm at a rate of 58 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. They have a normal morphology. This is sinus bradycardia.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. The QT/QTc intervals are prolonged (480/470 msec). The R-wave amplitude (voltage) in leads V3-V4 is increased (33 mm) ( ], which meets a criterion for left ventricular hypertrophy (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm), although the tall voltage could be a normal finding in a patient who is thin and does not have any lung disease. There are narrow and small Q waves (\( \wedge \)) (particularly in relation to the amplitude of the R wave) in leads V3-V6. These are septal Q waves.

The T waves (\( \downarrow \)) are tall, peaked, and symmetric (ie, upstroke and downstroke are similar, and the T wave appears to be tented). Even though there is tall QRS complex amplitude, which is a possible etiology for the tall T waves, the symmetry of the T waves is unusual and hence these T waves are hyperacute, associated with hyperkalemia.

Normal T waves are asymmetric regardless of amplitude. They have a slower upstroke and faster downstroke. T waves that are tall, peaked and, most importantly, symmetric are hyperacute and seen with hyperkalemia (systemic or localized as in acute myocardial infarction).

This patient likely has hyperkalemia on the basis of acute renal failure. He is volume depleted from days of diarrhea and poor oral intake. Additionally, he is taking an angiotensin-converting enzyme inhibitor and a nonsteroidal anti-inflammatory medication. When taken in combination, these medications cause a marked reduction in the glomerular filtration rate and can exacerbate an acute renal insult. The hyperacute T waves do not correlate with the level of hyperkalemia. Although conduction abnormalities (which indicate more serious hyperkalemia) are not present, therapy is indicated. Generally sodium polystyrene sulfonate is all that is necessary, but if the potassium level is very high, glucose and insulin, calcium gluconate or chloride, and sodium bicarbonate (especially if the patient is acidotic) would be indicated. If anuric renal failure worsens, acute dialysis may be necessary.
A 48-year-old executive presents for his company-sponsored annual physical examination. He is in good health and has no current complaints. He has mild dyslipidemia, which has been managed with dietary and lifestyle changes.

What does his ECG show?
What is the likely diagnosis?
ECG 47 Analysis: Normal sinus rhythm, nonspecific T-wave abnormalities
There is a regular rhythm at a rate of 66 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are of low amplitude but appear to be positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. The QT/QTc intervals are normal (400/420 msec).

The T waves (^) in most of the leads are flat and barely obvious. A normal T wave (+) can be seen in leads V1-V2. The flattened T waves represent a nonspecific T-wave abnormality.

Nonspecific T-wave abnormalities include flat, biphasic, or inverted T waves. T-wave abnormalities may be seen in various situations, including ischemia (inverted T waves that are often symmetric and usually associated with ST-segment changes), left ventricular hypertrophy (due to repolarization abnormalities), pericarditis/myocarditis, metabolic and pH abnormalities, old myocardial infarction, or central nervous system abnormalities; or they may be nonspecific. T-wave abnormalities may be seen in healthy subjects as a normal variant. In the absence of any clinical history, such T-wave abnormalities are considered nonspecific.

In addition, a U wave (↑) (the positive deflection following the T wave) can be seen in leads V1-V2. A U wave may be normally seen in right precordial leads. These are considered to result from repolarization of the His-Purkinje system.
A 38-year-old woman presents to the emergency department with fatigue and dyspnea on exertion. About 3 weeks prior to developing these symptoms, she recalls having a febrile illness. She now notes swelling of her ankles and is only able to walk about 50 m on level ground before becoming short of breath.

What does her ECG show?
What is the likely diagnosis?
ECG 48 Analysis: Normal sinus rhythm, biphasic (nonspecific) T waves
There is a regular rhythm at a rate of 76 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. The QT/QTc intervals are normal (380/430 msec).

The T waves (^) are abnormal and have a biphasic morphology in most of the leads; the T wave in lead aVR is positive, although the normal T wave in this lead is negative. These T-wave abnormalities are nonspecific and may be seen in various conditions, including ischemia, myocarditis/pericarditis, metabolic or pH abnormalities, old myocardial infarction, dilated or infiltrative cardiomyopathy, or left ventricular hypertrophy. Such T-wave abnormalities may also be seen in healthy subjects as a normal variant. T-wave abnormalities must be considered along with a clinical history.

This patient may have viral myocarditis with development of an acute cardiomyopathy as she has clinical evidence of heart failure preceded by a likely viral prodrome. This would account for the T-wave abnormalities. An echocardiogram should be performed to assess left ventricular and right ventricular function. In the setting of a left ventricular ejection fraction less than 0.40, the patient should be initiated on angiotensin-converting enzyme inhibitor and β-blocker therapy. Therapy is directed at relief of symptoms and is also supportive for any hemodynamic abnormalities. In most cases the cardiomyopathy resolves over time.
A 59-year-old man presents with several episodes of retrosternal chest pain occurring with minimal activity. Upon arrival in the emergency department, his pain has resolved. An ECG (49A) is obtained. Initial markers of cardiac
ischemia (creatine kinase [CK], CK-MB, and troponin I) are within normal limits. He is observed overnight, during which he has another episode of chest pain. A second ECG (49B) is obtained about 30 minutes after his chest pain resolved.

**What do these ECGs show?**
**What is the likely diagnosis?**
**What further testing is indicated?**
ECG 49A Analysis: Normal sinus rhythm, premature ventricular complex, left atrial hypertrophy, intraventricular conduction delay, nonspecific ST-segment abnormalities
ECG 49A shows a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is negative in lead V1 and biphasic in lead V2 (^). The P waves in leads II, III, aVF, and V4-V6 are broad with slight notching (*). These P-wave abnormalities are characteristic of left atrial hypertrophy.

The third QRS complex (+) is early, wide, and abnormal and does not have any preceding P waves. This is a premature ventricular complex and is followed by a full compensatory pause; that is, the PP interval around the premature complex (↔) is equal to two sinus PP intervals (⊥). This is due to the fact that there has been an on-time sinus impulse (the P wave is simultaneous with the premature ventricular complex and hence not seen) that is blocked within the AV node as a result of retrograde conduction from the premature ventricular contraction, which causes the AV node to be refractory and hence there is no antegrade conduction. The next on-time sinus impulse does conduct through the node.

The QRS complex duration is prolonged (0.12 sec), and there is no pattern characteristic of a bundle branch block. Hence this is an intraventricular conduction delay. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/420 msec and 320/370 msec when corrected for prolonged QRS complex duration). Noted are nonspecific ST-segment abnormalities (↑), with flattening of the ST segment in leads V5-V6.

continues
Podrid's Real-World ECGs

ECG 49B Analysis: Ischemic T-wave abnormalities (Wellens’ T waves), prolonged QT interval
**ECG 49B** was taken shortly after the second episode of prolonged substernal chest discomfort. There were no elevations of the cardiac enzymes. The rhythm is regular at a rate of 60 bpm. The P waves (*)& PR interval, and QRS complexes are identical to those in ECG 49A. The QT/QTc intervals are prolonged (560/560 msec), even when the prolonged QRS complex duration is considered (520/520 msec). In addition, there are new T-wave inversions (^) in leads I, aVL, and V2-V5. The T waves are deeply and symmetrically inverted, and the QT/QTc interval is prolonged. These T-wave changes have been referred to as Wellens’ T waves (pattern) and have been associated with a significant lesion in the proximal left anterior descending artery (LAD).

This patient should undergo urgent coronary angiography rather than noninvasive stress testing. The pretest probability of significant LAD disease is high, and there is a risk for precipitating a myocardial infarction with stress testing as it is likely that there is a very tight stenosis and the vessel is subtotally occluded. Of note, the QT prolongation is a further clue that there may be ongoing myocardial ischemia.
A 28-year-old intravenous drug user is admitted with fever and altered mental status. On admission he is tachycardic at 110 bpm and has a blood pressure of 148/30 mm Hg. Physical exam reveals a soft diastolic murmur at the upper sternal border (base of the heart).

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

**ECG 50 Analysis:** Junctional rhythm, premature ventricular complexes, cerebral T waves, prolonged QT interval
There is a regular rhythm at a rate of 50 bpm. There are no obvious P waves before or after the QRS complexes, although baseline artifact makes this uncertain. However, it is likely that this is a junctional rhythm. The second and eighth QRS complexes (+) are early, wide, and abnormal; they are premature ventricular complexes.

The QRS complex duration is normal (0.08 sec), and the axis is physiologically leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). There are diffuse, deep T-wave inversions (↑) (the positive T wave in lead aVR is actually inverted) that are asymmetric, with an initial portion of the T wave (descent) that is faster than the terminal portion (ascent). This is in contrast to the normal T wave, which has a slower initial portion or upstroke (ascent) and a faster terminal portion or downstroke (descent). In addition, there is significant prolongation of the QT/QTc intervals (↔) (720/660 sec). There is no ST-segment depression. The finding of deeply inverted and asymmetric T waves along with marked prolongation of the QT interval suggests that these are cerebral T waves, a result of some central nervous system process such as a subarachnoid hemorrhage, cerebral hemorrhage, tumor, head injury, or infection.

This patient has evidence of aortic insufficiency (wide pulse pressure, tachycardia, diastolic murmur) attributable to acute bacterial endocarditis. The clinical presentation of altered mental status suggests that he possibly has developed a brain abscess secondary to embolism of a bacterial vegetation from the aortic valve to the brain. The ECG findings are typical of an acute central nervous system insult, which is most likely cardioembolic in this context. Therapy for endocarditis should be emergently instituted and acute drainage of a brain abscess, if present, performed. Replacement of the aortic valve usually is performed once blood cultures are negative. If the patient remained stable, an aortic valve replacement would generally be performed after several weeks of antibiotic therapy. However, if there were worsening of the aortic regurgitation, development of AV conduction abnormalities (suggesting an abscess of the septum), or recurrent cerebral emboli, valve surgery would be performed more urgently.
Practice ECGs
A 54-year-old man collapses while working out at a gym. He was observed riding a stationary bicycle when he slowed down and collapsed, falling off the bike. A bystander could not feel a pulse and initiated CPR immediately. An employee connected an automated external defibrillator (AED), which advised a shock. After a single shock the patient became responsive and regained a pulse. Emergency medical services arrived shortly thereafter and obtained a 12-lead ECG.

What does his ECG show?
What is the likely diagnosis?
What likely caused his collapse?
ECG 51 Analysis: Normal sinus rhythm, left atrial hypertrophy, acute anterior wall ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P wave in lead V1 is negative, suggesting left atrial hypertrophy. This is a normal sinus rhythm.

The QRS complex duration (0.08 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/380 msec). The T waves (↓) are peaked and symmetric in leads V3-V5, and there is significant ST-segment elevation (↑) of up to 9 mm (↕) in leads V2-V5. The ST segments still have a concave morphology. This is an acute anterior wall ST-segment elevation myocardial infarction (STEMI) involving primarily the left ventricular apex.

An acute MI is identified by the presence of localized ST-segment elevation, hyperacute T waves (tall, peaked, and symmetric), and reciprocal ST-segment depressions (due to the same ST-segment changes viewed from another angle or direction). The location of the ST-segment elevations identifies the region of the myocardium involved.

This patient suffered a cardiac arrest, likely attributable to an acute anterior STEMI complicated by ventricular tachycardia or ventricular fibrillation. Normal sinus rhythm was restored with a single shock delivered by an automated external defibrillator (AED). AEDs are designed to be operated by first responders at a cardiac arrest. They contain sophisticated software that analyzes the rhythm and advises a shock if ventricular fibrillation or rapid ventricular tachycardia is detected. These devices are useful in reducing the time between the onset of an unstable tachyarrhythmia and restoration of a perfusing rhythm, particularly if there is a delay in access to emergency medical services.

Once brought to the hospital, the patient is a candidate for urgent catheterization and primary angioplasty and stenting. The use of a thrombolytic is relatively contraindicated in a patient who has undergone CPR, particularly when CPR has been traumatic or lasted longer than 10 minutes.
A 48-year-old man presents to the emergency department with 24 hours of intermittent but severe retrosternal chest discomfort. He was slow to seek medical attention because he believed that the discomfort would abate. Upon arrival at the hospital, he is taken urgently for coronary angiography. He states that he had a previous myocardial infarction (MI) but is unable to provide details.

What does his ECG show?
What do you expect the angiogram to show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 52 Analysis: Normal sinus rhythm, left atrial hypertrophy, old lateral wall MI, acute anterior wall ST-segment elevation myocardial infarction (STEMI), right axis deviation due to an old lateral wall MI
There is a regular rhythm at a rate of 82 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. Hence this is a normal sinus rhythm. The P wave is negative (+) in leads V1-V2, suggesting left atrial hypertrophy.

The QRS complex duration is normal (0.08 sec). The QT/QTc intervals are normal (320/370 msec). There is a Q wave in lead I (↑), diagnostic for a lateral wall myocardial infarction (MI). As a result of the Q wave in lead I (negative QRS complex) and the positive QRS complex in lead aVF, the axis is rightward. However, the right axis deviation is not caused by a conduction abnormality resulting from a left posterior fascicular block (QRS morphology in lead I is rS) but rather is due to the lateral infarction.

In addition, there are Q waves (QS complex) in leads V3-V6 (↓), diagnostic for an extensive anterior wall MI. ST-segment elevations are noted above the baseline (TP segment) in leads V3-V6 (↑). If the patient had a recent infarction, the ST-segment changes would be due to the acute ST-segment elevation myocardial infarction (STEMI) that is evolving. If the clinical history were that of a previous MI occurring weeks or years prior to obtaining the ECG, these persistent ST-segment changes would represent an aneurysm in the area of the previous infarction.

Given the history, this patient is likely to have had an acute thrombotic occlusion of his proximal left anterior descending artery (LAD) approximately 24 hours prior to presentation, when his symptoms first began. Since he is so late in presenting to the hospital, he has developed deep Q waves across the precordium and hence an intervention would not likely be helpful in reducing the extent of the infarction. However, given the presence of ongoing discomfort indicating ongoing coronary ischemia (post-MI angina), the ST-segment elevations would indicate that the ECG changes of an acute MI were evolving. Since the acute MI is of the anterior wall and there are ongoing symptoms (suggesting some residual viable myocardium), a percutaneous coronary intervention of the LAD would be indicated primarily for symptom relief. There would not likely be any survival benefit at this point in time.
A 35-year-old woman from India is in the United States visiting her family. She develops dyspnea and pedal edema after eating a large salty meal at a family celebration. She is taken to the emergency department where she is found on exam to have a blood pressure of 106/68 mm Hg and heart sounds that are regular with an early diastolic opening snap followed by a low-pitch grade II/IV diastolic rumble at the apex. She has diffuse rales and trace pedal edema.

What does her ECG show? What is the likely diagnosis? What therapy is indicated?
ECG 53 Analysis: Normal sinus rhythm, first-degree AV block, left atrial hypertrophy (abnormality), left anterior fascicular block, possible left ventricular hypertrophy, nonspecific ST-T wave changes
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.22). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. Hence this is a normal sinus rhythm. The P wave has a markedly increased amplitude (0.4 mV) and is very broad (0.16 sec). There is striking notching of the P wave seen in leads I, II, aVR, aVF, and V4-V6 (*), and there is a marked negative component seen in leads V1-V2 (+). Hence this is P mitrale, which is indicative of left atrial hypertrophy or a left atrial abnormality.

The QRS complex duration is normal (0.08 sec), and there is an extreme left axis deviation, between −30° and −90° (QRS complex is positive in lead I and negative in leads II and aVF), indicating a left anterior fascicular block. The QT/QTc intervals are normal (400/400 msec). The QRS complex has a normal morphology, although there is an RSR′ morphology in lead V1 (→), indicative of conduction delay to the right ventricle; this is often a normal variant. However, it is possible that this R′ represents right ventricular hypertrophy. The S wave in lead V4 is 34 mm (↑), suggesting possible left ventricular hypertrophy. However, there are no other features of hypertrophy present and, given the young age of the patient, this could be a normal finding. There are nonspecific ST-T wave changes in leads I, aVL, and V4-V6 (↑).

The physical examination reveals an opening snap occurring in diastole after S2; this is due to the opening of a nonpliable mitral valve leaflet. Along with a diastolic rumble, this is pathognomonic for mitral stenosis. The most likely etiology for the patient’s mitral stenosis is rheumatic heart disease, which is common in India. The clinical history suggests that with volume overload, there is a further increase in left atrial and hence pulmonary venous pressure, resulting in evidence of pulmonary congestion.

As a result of the stenotic mitral valve, there is development of left atrial hypertrophy so as to increase the pressure generated by the left atrium in an attempt to force blood through the smaller mitral valve orifice and fill the left ventricle in diastole. The presence of significant left atrial hypertrophy increases the risk for atrial fibrillation and, in association with mitral stenosis, is a major risk factor for stroke.

In general, the left ventricle is not affected in mitral stenosis, so the presence of prominent voltage may be a normal finding in a young patient, although it might also reflect left ventricular hypertrophy due to another condition, such as hypertension, mitral regurgitation, or aortic valve involvement.

When mitral stenosis progresses and becomes more severe, there is an increase in pulmonary artery pressure and right-sided pressure. The ECG can progress in parallel with hemodynamic changes and develop right ventricular hypertrophy (tall R wave in lead V1) and a right axis deviation; on occasion there is also right atrial hypertrophy (bifascicular hypertrophy). Therapy for mitral stenosis depends on the anatomy of the mitral valve. In some cases surgical or balloon valvulotomy is effective. If the valve is very calcified or if there is significant mitral regurgitation, mitral valve replacement is necessary.
Notes
A 38-year-old man who recently immigrated to the United States from Pakistan was found to have a positive tuberculin skin test. He was started on isoniazid (INH) by his primary care provider and took it for 6 weeks. He then developed mild chest discomfort and returned to his primary care provider’s office, where an ECG was obtained.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 54 Analysis: Junctional tachycardia, pericarditis
There is a regular rhythm at a rate of 100 bpm. There are no P waves seen before or after any QRS complex. The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). This is a junctional tachycardia. The QT/QTc intervals are normal (320/410 msec).

The J points and ST segments (^) are elevated above the baseline (TP segment) in leads I, II, aVR (ST-segment depression in this lead is actually ST-segment elevation), aVL, aVF, and V2-V6. This is, therefore, pericarditis. The concave morphology of the ST segment is maintained, and the T-wave morphology is normal (asymmetric).

This patient may have drug-induced pericarditis caused by isoniazid (INH; which can cause a lupus-like syndrome). This should be treated with withdrawal of the INH therapy and initiation of NSAID therapy. If the patient’s symptoms do not resolve with these interventions, the clinician should consider the possibility of active tuberculous pericarditis. An echocardiogram should be ordered to evaluate for a pericardial effusion. If an effusion is present, pericardiocentesis should be performed as a diagnostic technique; the presence of tamponade requires pericardiocentesis as a therapeutic intervention.
A 68-year-old man is seen in a preoperative clinic for risk optimization before undergoing elective abdominal aortic aneurysm repair. He has hypertension and a 40 pack-year smoking history. He denies any chest pain but lives a sedentary lifestyle. He has trouble climbing a single flight of stairs due to shortness of breath. Prior to being found to have an abdominal aortic aneurysm on his first visit with a primary care provider, he had not sought any medical care. Until recently, he took no medications but has been started on a daily aspirin and lisinopril.

What does his ECG show?
What is the likely diagnosis?
What further workup is indicated?
Podrid’s Real-World ECGs

ECG 55 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), nonspecific ST-T wave changes, old anteroseptal myocardial infarction (MI), old inferior wall MI
There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave (+) is broad and slightly notched in leads II, III, and aVF, suggesting that left atrial hypertrophy may be present.

The QRS complex duration is normal (0.08 sec). The axis is normal, approximately 0°, with a positive QRS complex in lead I and a biphasic QRS complex in lead aVF. The QT/QTc intervals are normal (360/420 msec). There are Q waves (↑) in leads II, III, and aVF, which are diagnostic for an old inferior wall myocardial infarction (MI). In addition, there are Q waves (↓) in leads V1-V3, which are characteristic of an anteroseptal MI. Lastly, there are diffuse nonspecific ST-T wave changes (^).

A chronic or old MI is identified by the presence of abnormal Q waves, defined as any Q wave in leads V1-V3 or a Q wave 0.03 to 0.04 second or longer in lead I, II, aVF, or V4-V6 (in two or more contiguous leads) and at least 1 mm in depth. However, Q waves may be normal and are ignored in lead III (unless they are also in leads II and aVF), in lead V1 (unless also in lead V2), and in lead aVL (unless Q-wave height ≥ 50% R-wave height). The QS complexes in leads V1-V2 may also be a normal variant.

Although a Q wave in leads V1-V2 can be a normal variant, in this case there are large Q waves in leads V1-V3; therefore, this is indicative of an old anteroseptal MI. This patient has poor functional capacity and likely significant coronary artery disease. However, the presence of ischemia may not be reliably established given his poor functional status. He should undergo further risk stratification before his elective high-risk vascular surgery. This generally involves some form of exercise testing. It is unlikely that he can perform any substantial exercise on a treadmill given his poor functional status. In this situation a dobutamine stress echocardiogram or a pharmacologic stress test would be preferred. The presence of substantial areas of ischemia might prompt a cardiac catheterization, which should be performed for clinical indications prior to vascular surgery. It has been shown (CARP trial) that in such patients the outcome from medical therapy (especially β-blockers), percutaneous coronary intervention, or bypass surgery is the same. This finding calls into question the need for preoperative risk stratification rather than just beginning an intensive anti-ischemic medical program.
An 88-year-old woman is admitted to the intensive care unit with hematemesis and dizziness. On endoscopy, she is found to have a bleeding gastric ulcer that is clipped. Her hematocrit is 19 and she is transfused with 4 units of red blood cells.

What does her ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 56 Analysis: Normal sinus rhythm, upsloping and horizontal ST-segment depression
There is a regular rhythm at a rate of 86 bpm. There is a P wave (*) prior to each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (360/420 msec). There is normal R-wave progression from leads V1 to V6. J-point and ST-segment depression (*) are seen in the inferior leads (II, III, and aVF) and the anterolateral leads (V3-V6). The ST-segment depression (about 3 mm [↑]) is upsloping in leads V3-V4, while it is horizontal in leads II, III, aVF, and V5-V6 and is up to 7 mm (↑). The ST-segment elevation in lead aVR (↓) is actually ST-segment depression.

This patient has diffuse ST-segment depression, likely indicating global ischemia. Since she has not had any cardiac symptoms before or during this event, the ischemia is undoubtedly exacerbated by marked anemia (demand ischemia). It is likely that this patient has significant fixed coronary disease that has become clinically evident under the hemodynamic stress of her acute blood loss. The primary treatment for ischemia in this setting is blood transfusion and volume resuscitation followed by definitive endoscopic or surgical therapy for the bleeding ulcer.
A 48-year-old woman with Hodgkin’s disease undergoes chest and mediastinal radiation. After her fourth radiotherapy treatment she develops chest pain and a sharp sensation while inhaling.

What does her ECG show?
What is the likely diagnosis?
ECG 57 Analysis: Normal sinus rhythm, pericarditis
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P-wave morphology is normal.

The QRS complex duration is normal (0.10 sec), and there is a normal axis, between $0^\circ$ and $+90^\circ$ (positive QRS complex in leads I and aVF). The QRS morphology is normal. The QT/QTc intervals are normal (320/390 msec).

There is widespread J-point and ST-segment elevation (^) above the baseline (TP segment), apparent in leads I, II, III, aVR (ST-segment depression in this leads is actually ST-segment elevation), aVF, and V2-V6. The ST segment has a normal concave morphology. Also noted is PR-segment depression (†), most obvious in leads I, II, aVF, and V4-V5. In lead aVR the PR-segment elevation is actually PR depression.

The baseline is the TP segment, and the PR segment can be seen below this plane. Although the T waves are prominent, especially in leads V2-V4, they are asymmetric (slow upstroke, faster downstroke) and hence normal. The ECG, therefore, shows pericarditis.

This patient probably has radiation-induced pericarditis. Pericardial involvement from Hodgkin’s disease is possible but less likely. Radiation-induced pericarditis is less common now than in the past as a result of better shielding techniques. It tends to occur during ongoing radiotherapy. It may develop years after the radiation therapy and can result in chronic and/or constrictive pericarditis. On occasion the pericardium may become calcified. Other long-term complications may also occur from chest radiation. These include coronary atherosclerosis, valvular heart disease (most commonly aortic regurgitation, aortic stenosis, or mitral regurgitation), conduction system disease and, rarely, radiation-induced restrictive cardiomyopathy.
A 74-year-old woman who suffered a myocardial infarction (MI) 2 months ago suddenly experiences word-finding difficulties and transient right-sided weakness. She waits about 3 hours before presenting to the hospital. Shortly after arriving in the emergency department, her symptoms begin to resolve. The treating neurologist suspects she has suffered a transient ischemic attack in the left middle cerebral artery distribution. There is no evidence of ischemic stroke on brain computed tomography. An ECG is performed on admission.

What does it show?
What is the likely diagnosis?
**Podrid’s Real-World ECGs**

**ECG 58 Analysis:** Normal sinus rhythm, intraventricular conduction delay, old anterior wall myocardial infarction, ST-segment elevation (left ventricular aneurysm)
There is a regular rhythm at a rate of 66 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.16 sec). The QRS duration is prolonged (0.12 sec) without any specific pattern; hence this is an intraventricular conduction delay. The axis is leftward, at –30° (positive QRS complex in lead I, negative QRS complex in lead aVF, isoelectric QRS complex in lead II). The QT/QTc intervals are normal (360/380 msec and 320/360 msec when corrected for the prolonged QRS complex duration).

In leads V1-V3, there is a QS complex (↓), the ST segments (↑) are elevated, and the T waves are inverted (+). In the clinical context of a prior myocardial infarction and no current chest pain, the ST-segment elevation likely represents a chronic anterior wall myocardial infarction associated with a left ventricular aneurysm. She may have a mural thrombus associated with the aneurysm that has caused a cardioembolic stroke. This diagnosis can be confirmed by echocardiography, which will show an area of myocardial thinning and akinesis. A thrombus may be visualized along the luminal surface of the ventricle. Therapy with systemic anticoagulation should be initiated to prevent further thrombus formation in the aneurysm and to reduce the risk for further cardioembolic phenomena.
A 62-year-old man with diabetes presents for a visit with his new primary care physician. Given his longstanding diabetes, the man is concerned about his risk for cardiovascular disease. He reports no recent illness and has good functional capacity. He does not recall any episodes of prolonged chest discomfort or dyspnea.

What does his ECG show?
What is the likely diagnosis?
What further testing is warranted?
ECG 59 Analysis: Normal sinus rhythm, left atrial hypertrophy, old inferior wall myocardial infarction (MI), nonspecific T-wave abnormalities
There is a regular rhythm at a rate of 64 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.20 sec). There is a P wave prior to each QRS complex, and the P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is negative (+) in lead V1 and biphasic in leads V2-V3 (^), suggesting left atrial hypertrophy.

The QRS complex duration is normal (0.10 sec). There are significant Q waves (↑) in leads II, III, and aVF as well as T-wave inversions (▲), diagnostic for an old inferior wall myocardial infarction (MI). As a result of the inferior Q waves, the axis appears to be leftward (positive QRS complex in lead I and negative QRS complex in lead aVF); however, this is the result of the inferior wall MI. There are also nonspecific T-wave changes (flattening) (↓) in leads V4-V6. The QT/QTc intervals are normal (360/370 msec).

Silent MIs are common and are seen in up to one-third of those who have an MI pattern on the ECG. They are especially common in diabetic patients, who often have discomfortless ischemia and infarctions. Since he suffered an MI without developing chest discomfort, this man is at risk for further episodes of “silent” or discomfortless myocardial ischemia. He should undergo further risk stratification with an exercise test with imaging (either echocardiography or nuclear imaging) to assess for further territory at risk. Should he have evidence of significant myocardial ischemia, he may elect to undergo coronary revascularization.
A medical student is learning to perform electrocardiography and obtains an ECG on her 23-year-old classmate.

What does the ECG show?
What is the likely diagnosis?
What further treatment is warranted?
ECG 60 Analysis: Sinus bradycardia, counterclockwise rotation (early transition), early repolarization
The rhythm is regular at a rate of 50 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6, and they have a normal morphology. This is sinus bradycardia.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/370 msec). Although the R wave in leads V4-V5 is prominent, the amplitude does not meet any of the criteria for left ventricular hypertrophy. There is a tall R wave in lead V2 (←), a result of early transition or counterclockwise rotation of the electrical axis in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. When there is counterclockwise rotation, electrical forces from the left ventricle occur early in the precordial leads, accounting for the tall R wave in lead V2. Although the T waves are prominent in leads V3-V4, they are asymmetric and have a normal morphology.

J-point and ST-segment elevation (↑) are noted in leads V2-V5. This is termed early repolarization, which is a normal finding often seen in association with left ventricular hypertrophy; it is also seen in young patients with or without prominent R-wave amplitude.

Sinus bradycardia is a normal finding in young, physically fit individuals. Although the appearance of early repolarization can be alarming (suggesting an early myocardial infarction), it is a normal variant and is present in about 5% of the population. The pattern most often occurs in the precordial leads (as is seen in this case) but can affect any lead. The normal T waves also exclude an acute myocardial infarction. No further diagnostic studies or therapy is warranted.
A 64-year-old man with a previous myocardial infarction (MI) but no current symptoms is in the operating room undergoing an abdominal aortic aneurysm repair. The lengthy surgery appears to progress without incident; however, after returning to the recovery room he becomes progressively hypoxic despite increasing fraction of inspired oxygen (FiO₂) and has short runs of a wide-complex tachycardia determined to be ventricular tachycardia. A 12-lead ECG is obtained.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 61 Analysis: Normal sinus rhythm, left atrial hypertrophy, old anterior wall MI, persistent ST-segment elevation (left ventricular aneurysm)
Although the leads are not labeled, the pattern is always the same (column 1 is leads I, II, and III; column 2 is leads aVR, aVL, and aVF; column 3 is leads V1-V3; and column 4 is leads V4-V6). There is a regular rhythm at a rate of 88 bpm. There is a P wave (\( * \)) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is negative in lead V1 (\(^\wedge\)), suggesting left atrial hypertrophy (abnormality).

The QRS complex duration is normal (0.10 sec), and there is a physiologic left axis deviation, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (360/440 msec and 340/410 msec when corrected for the prolonged QRS complex duration). There are Q waves (\( \nabla \)) in leads V1-V5 (the complex has a QS morphology in leads V1-V2), diagnostic for an extensive anterior wall myocardial infarction (MI). In addition, ST-segment elevation (\( \downarrow \)) can be seen above the baseline (TP segment) in leads V2-V5; slight elevation is noted in lead aVL. ST-segment elevations are seen in the setting of an acute MI, and they return to baseline during the normal evolution of the ECG after the infarction. ST-segment elevation that persists for weeks to years after an MI indicates the presence of an aneurysm.

Although this patient was initially thought to have suffered a large anterior MI during his surgery, a pre-surgery ECG was identical to this one. Hence there has been a previous MI (as per his history) and the ST-segment changes are indicative of an anterior wall aneurysm. The episodes of tachycardia, likely ventricular tachycardia, as well as the increasing hypoxemia could perhaps be the result of heart failure due to increased vascular volume from excessive fluid infusion and possibly the effect of hypoxia. It is most likely that the ventricular tachycardia originates in the area of the aneurysm that is associated with scar.
A 61-year-old man with factor V Leiden presents for evaluation of a swollen and painful right calf, dyspnea, and pleuritic chest pain that began shortly after returning home from a long transatlantic flight. He has had two prior deep vein thromboses (DVTs) and had been maintained on warfarin for several years. However, warfarin was discontinued 1 year ago. His examination is notable for a loud P2, subtle right precordial heave, and a warm, edematous right calf.

What does his ECG show?
What is the likely diagnosis?
What further evaluation or therapy is indicated?
ECG 62 Analysis: Normal sinus rhythm, right atrial hypertrophy (abnormality), right ventricular hypertrophy (RVH), right axis deviation, prolonged QT interval
There is a regular rhythm at a rate of 94 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The P waves are very tall (0.4 mV), peaked, and narrow (0.10 sec), especially in leads II, aVF (▲), and V1-V3, where the P wave is primarily positive and narrow (▼). The P-wave morphology is characteristic of right atrial hypertrophy or P pulmonale. The etiology for right atrial hypertrophy (abnormality) is often right ventricular hypertrophy (RVH) resulting from right-sided valvular disease or pulmonary artery hypertension (primary or secondary from lung disease), left ventricular disease (systolic or diastolic), or left-sided valvular abnormalities.

The QRS complex duration is normal (0.10 sec), and the axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include RVH (associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (a deep Q wave in leads I and aVL), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation). The QT/QTc intervals are prolonged (400/500 msec).

Noted is a tall R wave in lead V1 (←). This is characteristic of RVH, particularly when associated with a right axis deviation. Other causes for a tall R wave in lead V1 include a posterior wall myocardial infarction, lead misplacement, recording of right-sided leads, dextrocardia, Wolff-Parkinson-White pattern, hypertrophic cardiomyopathy, or Duchenne muscular dystrophy. However, no other ECG findings support any of these diagnoses. Associated with RVH are ST-T wave changes seen primarily in leads V1-V2 (↑), which possibly reflect subendocardial ischemia of the thick right ventricular myocardium.

The physical exam and ECG are consistent with deep vein thrombosis (DVT), the result of a long airplane trip, and likely a pulmonary embolism. The RVH suggests that the right ventricular pressure overload is chronic, perhaps due to prior pulmonary emboli. The diagnosis of DVT can be made by venous Doppler study of the lower extremity. Either a ventilation/perfusion (V/Q) scan or computed tomography pulmonary angiogram should be done to assess for pulmonary emboli. The patient should also be evaluated for pulmonary hypertension, initially with an echocardiogram. This patient should be treated with long-term anticoagulation. As he has had recurrent episodes of DVT and has a condition predisposing to thrombus formation, anticoagulation should be continued indefinitely.
A 72-year-old woman is admitted to the emergency department with unheralded syncope. She reports that she was walking in the grocery store when she collapsed and struck her head. She does not recall the event, but bystanders report that she was unconscious for about 90 seconds. When emergency medical services arrived, she was awake but groggy. An ECG is obtained.

What does her ECG show?
How does this finding affect the further workup she will require for her syncope?
Podrid’s Real-World ECGs

ECG 63 Analysis: Normal sinus rhythm, old anteroseptal myocardial infarction (MI), nonspecific ST-T wave changes
There is a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There are Q waves or QS complexes in leads V1-V2 (↓), which are diagnostic for an old anteroseptal myocardial infarction (MI). In addition, nonspecific ST-T wave changes (↑) can be seen in leads I, aVL, and V4-V5.

This patient had un heralded syncope and suffered a head injury during the event. The clinical history strongly suggests that she may have had a cardiac etiology for the syncope. This would include a malignant ventricular arrhythmia or a profound bradycardia, particularly transient complete heart block. The finding of an old anteroseptal MI on ECG increases the possibility that she may have had either a ventricular tachycardia arising from the site of her myocardial scar or complete heart block with a slow ventricular escape rhythm as a result of significant septal damage that involves the His-Purkinje system. In this situation the syncopal episodes are termed Stokes-Adams attacks.

MI with scarring is the most common substrate abnormality associated with ventricular tachycardia in this age group and is also a common etiology for complete heart block. She should be monitored for evidence of a tachyarrhythmia or bradyarrhythmia. If complete heart block is noted, the treatment is a pacemaker. The occurrence of a spontaneous ventricular arrhythmia would be an indication for an implantable cardioverter-defibrillator to reduce her risk for a recurrent syncopal episode as well as the potential for sudden death. If no abnormality is noted spontaneously with monitoring, an electrophysiologic study may be in order to evaluate the conduction system and attempt to induce a ventricular tachyarrhythmia, indicating a site within the myocardium that is electrically unstable. Additionally, she should be evaluated for evidence of myocardial ischemia by an exercise test.
A 72-year-old woman was mugged while walking through the park. She alerted a nearby police officer, who found her distraught and clutching her chest. The woman reported marked chest discomfort and shortness of breath. The police officer took her to a nearby emergency department, where an ECG was obtained.

What does her ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

**ECG 64 Analysis:** Normal sinus rhythm, T-wave inversions and ST-segment depression (ischemia), prolonged QT interval
There is a regular rhythm at a rate of 70 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is physiologically leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QRS complex morphology is normal. There are inverted T waves (^) in leads I, II, aVR (normal T wave is inverted in this lead), aVL, and V2-V6. The inverted T waves are symmetric, and the QT/QTc intervals (↔) are prolonged (460/500 msec). In addition, there are ST-segment depressions (↓) in leads V3-V4. These T-wave inversions are strongly suggestive of ischemia (especially when associated with ST-segment depression and QT-interval prolongation), although this can only be established definitively with a clinical history.

This patient was taken to the catheterization laboratory given the high suspicion of acute myocardial ischemia. However, angiography revealed normal coronary arteries. Left ventriculography was then performed and showed apical and basal hypokinesis. Based on these findings, the patient was diagnosed with stress-induced cardiomyopathy (also called takotsubo cardiomyopathy). This syndrome was initially described by Japanese clinicians who noted that the shape of the left ventricular cavity resembled an octopus trap (called a takotsubo) in some patients experiencing significant emotional or physical stress. Stress cardiomyopathy is believed to be mediated by high levels of circulating catecholamines. Typically, the cardiomyopathy resolves over days to weeks and has no long-term consequences. The syndrome can mimic a myocardial infarction in almost every way. However, coronary artery disease is absent.
A 72-year-old man is awakened from sleep with severe retrosternal chest pressure and dyspnea. He calls emergency medical services. The responding medical technician finds him diaphoretic and in mild respiratory distress. An ECG is obtained in the field and transmitted to you in the emergency department.

What does his ECG show?
What is the likely diagnosis?
What is the next step?
Podrid’s Real-World ECGs

ECG 65 Analysis: Normal sinus rhythm, acute anterior wall ST-segment elevation myocardial infarction (STEMI), hyperacute T waves
There is a regular rhythm at a rate of 60 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, III, aVF, and V4-V6 and negative in lead aVR; they have a normal morphology. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the morphology is normal. There is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/360 msec). Noted are tall, peaked, and symmetric T waves (↑) in leads V3-V4 and symmetric T waves in leads V2-V5. In addition, there is ST-segment elevation (↑↑) in leads V2-V5; the ST-segment elevation is up to 6 mm (↑↑). The ST segments are still concave. This is an acute anterior wall ST-segment elevation myocardial infarction (STEMI; apex and possibly anterolateral).

An acute MI is identified by the presence of localized ST-segment elevation, hyperacute T waves (tall, peaked, and symmetric), and reciprocal ST-segment depressions (due to the same ST-segment elevations viewed from another angle or direction). The location of these ST-segment elevations identifies the region of the myocardium involved.

The goal of therapy for a patient with an acute anterior wall MI should be to restore adequate coronary blood flow in a timely manner. This can be accomplished either with primary angioplasty and stenting or thrombolysis. If the transit time to a primary angioplasty center is longer than 90 minutes, the patient should undergo thrombolysis; otherwise, primary angioplasty and stenting is preferred.

Because the ECG is available before the patient arrives in the emergency department, the treating physicians can expedite care by preparing for the patient’s arrival. A commonly used metric for the efficiency of this process is the “door-to-balloon time” (DTBT), which measures the time from presentation to the emergency department to the inflation of the angioplasty balloon. Many hospitals set a goal DTBT of less than 90 minutes and door-to-needle time for thrombolysis of less than 30 minutes.
Notes
An 81-year-old man presents for his first visit with a new primary care physician. Over the past 6 months he has noted progressive dyspnea on exertion and pedal edema. He recalls an episode of severe chest discomfort and dyspnea about 8 months ago, but he did not seek medical attention at that time.

What does his ECG show?
What is the likely diagnosis?
What therapy is indicated?
ECG 66 Analysis: Normal sinus rhythm, right atrial hypertrophy (abnormality), left atrial hypertrophy (abnormality), biatrial hypertrophy (abnormality), old inferior wall myocardial infarction (MI), old anterior wall MI, left axis deviation due to an old inferior wall MI.
The rhythm is regular at a rate of 90 bpm. There is a P wave (*) in front of each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is narrow (0.10 sec) and of increased amplitude (0.3 mV), especially in leads I, II, aVF, and aVR (*). The P waves in these leads have a morphology characteristic of right atrial hypertrophy (abnormality) or P pulmonale. The P waves in leads V1-V2 are deeply inverted or negative (▲), which is a feature of left atrial hypertrophy (abnormality). This represents biatrial hypertrophy (abnormality).

The QRS complex duration is normal (0.08 sec), and the axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, the QRS complex has a QS morphology in these leads (↓), consistent with a diagnosis of a chronic (old) inferior wall myocardial infarction (MI). The left axis deviation is not the result of a conduction problem (ie, it is not a left anterior fascicular block). There is also a QS complex in leads V1-V5 (▼), diagnostic for an extensive chronic (old) anterior wall MI. The QT/QTc intervals are normal (320/390 msec).

This patient has suffered a prior inferior and anterior wall MI. At this point the focus of care should be on treating modifiable risk factors and reducing the likelihood of future coronary events. This would include controlling blood pressure, screening for diabetes, quitting smoking, and treating hyperlipidemia. Most patients like this would be treated with a daily aspirin, an HMG-CoA reductase inhibitor (statin), and an anti-hypertensive regimen.

When both an inferior wall MI and a large anterior wall MI are present, there can be a significant reduction in left ventricular systolic function, although this patient does not complain of symptoms of low stroke volume and low cardiac output. The finding of biatrial hypertrophy does suggest elevated ventricular filling pressures and perhaps some degree of occult heart failure. An echocardiogram is warranted to assess chamber size and function. Stress testing, once symptoms are treated and the patient is stable, is useful for establishing functional status and predicting outcome as well as risk stratification if he again develops exertional chest discomfort.
A 62-year-old man with multiple cardiovascular risk factors, including insulin-dependent diabetes, dyslipidemia, and hypertension, undergoes a stress test for evaluation of dyspnea on exertion. He exercises for 6 minutes, 38 seconds without
developing dynamic ECG changes or chest pain. He stops the test due to fatigue. Blood pressure at the time was 220/110 mm Hg. Two minutes into recovery, his ECG changes (67A). His baseline pre-exercise ECG is also shown (67B).

What does his posttest ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 67A Analysis: Normal sinus rhythm, downsloping ST-segment depression
ECG 67A was obtained during recovery after an exercise test. There is a regular rhythm at a rate of 88 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, III, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is a normal QRS morphology with normal R-wave progression from leads V1 to V6.

The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (360/440 msec). There is significant J-point and ST-segment depression (↑) (up to 2 mm below baseline, ie, TP segment) in leads II, III, aVF, and V3-V6. The ST-segment depression is downsloping. Downsloping ST-segment depression is the type of ST-segment change that is most predictive of myocardial ischemia, most often the result of occlusive coronary artery disease.
Podrid’s Real-World ECGs

ECG 67B Analysis: Normal
ECG 67B is the baseline ECG obtained prior to the exercise test. The P waves (•), PR interval, QRS complexes, QT interval, and T waves are normal and are the same as those seen in ECG 67A. However, there is no J-point or ST-segment depression present. The ST segments are at baseline (↑) and have a normal concave morphology.

Although the stress test is positive for myocardial ischemia based on the ST-segment depression, the depressions occurred in the absence of symptoms and were associated with a significant hypertensive response. Not uncommonly, ST-segment changes occur after exercise, during recovery. This is likely due to the fact that during recovery there is still an increase in heart rate, blood pressure, and inotropy. Additionally, after exercise patients lie on a bed or stretcher, which results in an increase in venous return or preload—another factor that increases myocardial oxygen demand.

Stress tests should always be interpreted in the context of the patient’s pretest probability of coronary artery disease. This patient with diabetes, dyslipidemia, hypertension, and a history of dyspnea on exertion has a high pretest probability of coronary artery disease. In this setting, the probability of a false-positive test is very low. Further medical therapy is warranted, even though the patient does not give a history of angina. However, diabetics often have discomfortless ischemia (ie, they do not experience angina but do have other symptoms that in patients with angina often occur along with the chest discomfort, including fatigue, diaphoresis, nausea, and shortness of breath or dyspnea on exertion).

Therapy should be directed at controlling exercise-induced hypertension as well as modifying risk factors. Therapy includes aspirin, an angiotensin-converting enzyme inhibitor, a statin, and a β-blocker as well as a long-acting nitrate as the patient does have evidence of ischemia associated with the symptom of dyspnea on exertion. However, there might still be uncertainty about the presence of significant coronary artery disease as the patient had a significant increase in blood pressure, which would also result in ST-segment depression due to impaired subendocardial blood and oxygen supply. In addition, exercise did not provoke his symptom of shortness of breath. In this situation, exercise testing could be repeated with echocardiography to look for the development of a wall motion abnormality correlating with the ST-segment changes. In some situations diagnostic coronary angiography would be indicated to define the coronary anatomy. If severe disease is present, percutaneous or surgical revascularization might be necessary.
A 52-year-old man presents to the emergency department with chest discomfort occurring at rest. He has had several episodes similar to this over the past few years but has not sought medical attention. On exam, he is noted to have jugular venous distension, trace pedal edema, and bilateral rales. The first set of cardiac markers is negative.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 68 Analysis: Normal sinus rhythm, old anteroseptal and anteroapical myocardial infarction, ST-segment depression (ischemia)
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and the axis is normal, about +90° (biphasic QRS complex in lead I and positive QRS complex in lead aVF). The QT/QTc intervals are normal (360/360 msec). There are significant Q waves (↑) in leads V1-V4, diagnostic for an old anteroseptal and anteroapical myocardial infarction. There are diffuse ST-T wave abnormalities with slight horizontal ST-segment depression (+) in leads I, II, aVF, and V5-V6, suggestive of ischemia.

This patient has significant coronary disease on the basis of his ECG and an acute coronary syndrome based on his clinical presentation. He has evidence of an old anterior wall myocardial infarction and possibly superimposed myocardial ischemia. Additionally, he has some evidence of congestive heart failure on examination. Given his evidence of prior myocardial infarction and heart failure as well as chest discomfort at rest, he is considered to be at high risk for adverse events. He should undergo coronary angiography within the next 48 hours. ■
A 55-year-old man presents to the emergency department with 2 days of sharp retrosternal chest pain. He tried taking an over-the-counter pain reliever (acetaminophen) with little effect. His ECG prompts activation of the coronary angiography suite. Before starting the procedure, the treating cardiologist reviews the presenting ECG.

What is the ECG diagnosis?

What further diagnostic testing would be useful?
**Podrid’s Real-World ECGs**

**ECG 69 Analysis:** Normal sinus rhythm, left ventricular hypertrophy, counterclockwise rotation (early transition), pericarditis
There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm. The P-wave morphology is normal.

The QRS complex duration is normal (0.08 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/370 msec). The amplitude (voltage) of the R wave in leads V4 and V5 is increased (48 mm) ( ), meeting one of the criteria for left ventricular hypertrophy (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The T wave (+) has an increased amplitude in lead V2 but is asymmetric and hence normal. Prominent T waves are often associated with left ventricular hypertrophy. Also noted is early transition with a tall R wave (→) in lead V2. This is due to counterclockwise rotation of the axis in the horizontal plane, determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are seen early in the right precordial leads (ie, the tall R wave in lead V2).

There is widespread J-point and ST-segment elevation (↑) of 6 to 7 mm (↑), apparent in leads I, III, aVR (ST-segment depression in this lead is actually elevation), aVF, and V2-V6. The ST segment has a normal concave morphology. Also noted is slight PR-segment depression, most obvious in leads V2-V3. The ECG shows pericarditis. Although J-point and ST-segment elevation can be seen with left ventricular hypertrophy and is termed early repolarization, this is usually present only in the lateral precordial leads and the J point and ST segments are not generally this elevated.

Although the marked ST-segment elevation raises concern for an acute myocardial infarction, this pattern is more typical of acute pericarditis. The ST segments have a normal concave morphology, the T waves are normal, the ST-segment elevations are diffuse, and there are no reciprocal changes. Additionally, this patient has had chest pain for 2 days and has not developed the pathologic Q waves that are typical of an evolving myocardial infarction. An echocardiogram to evaluate for wall motion abnormalities would be useful in determining if there is active myocardial ischemia.
A 48-year-old man with a 20 pack-year smoking history develops retrosternal chest pain while playing squash. He stops playing and sits to rest. When the pain does not remit, his partners bring him to a nearby emergency department, where an ECG is obtained.

What does his ECG show?
What is the likely diagnosis?
What coronary artery is likely to be involved?
ECG 70 Analysis: Normal sinus rhythm, acute inferior wall
ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 84 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS duration is normal (0.08 sec), and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/400 msec). The T waves (+) in leads II, III, and aVF are tall and symmetric. In addition, there is ST-segment elevation (↑) in these leads and the ST segment has a normal concave morphology. This is an early acute inferior wall ST-segment elevation myocardial infarction (STEMI). Noted are ST-segment depression (▲) in leads I, aVL, and V1-V3. Reciprocal changes associated with an inferior wall myocardial infarction are usually seen in leads I and aVL. While the ST-segment depression in leads V1-V3 could represent reciprocal changes, in the setting of an inferior wall infarction they may be indicative of posterior wall involvement. In this situation it would be useful to place posterior leads under the left scapula (leads V7-V8, over the posterior wall of the left ventricle) to look for ST-segment elevation and possible posterior wall involvement.

In the case of a right dominant circulation, the posterior wall is supplied by the posterior left ventricular branch of the right coronary artery. In the case of a left dominant circulation, the posterior wall is supplied by branches of the circumflex artery.
A 54-year-old Taiwanese woman is referred to a cardiologist because of an abnormal ECG. She feels generally well but has had two episodes of syncope that came on without warning over the past 2 years. On exam, she has a palpable S4 but no other abnormalities.

What does her ECG show?
What is the likely diagnosis?
What further testing is indicated?
Podrid’s Real-World ECGs

ECG 71 Analysis: Normal sinus rhythm, T-wave inversions
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and the axis is normal at about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). The QRS morphology is normal. The QT/QTc intervals are normal (440/440 msec). There are deeply inverted T waves (†) in leads I, aVL, and V2-V6. However, the T waves are asymmetric with a faster upstroke and a slower downstroke.

Diffuse T-wave inversions are nonspecific and not definitely the result of ischemia, especially since they are asymmetric. However, as this woman is from southeast Asia, the possibility of an apical variant of hypertrophic cardiomyopathy should be considered. This diagnosis is supported by the presence of prominent and inverted T waves across the left precordial leads (V3-V5); these T-wave changes are a frequent finding in this disease. An echocardiogram should be obtained. If there were an apical hypertrophic cardiomyopathy, the echocardiogram would show a thickened apex that obliterates the ventricular cavity at end systole. Unlike hypertrophic cardiomyopathy affecting the base of the heart, the apical variant does not cause left ventricular outflow tract obstruction. If she were to have this entity, strong consideration would be given to a ventricular tachyarrhythmia as the cause of her syncope. In this variant of hypertrophic cardiomyopathy, mechanical obstruction and decreased cardiac output do not occur.
A 68-year-old woman presents with marked dyspnea on exertion. She has been reluctant to seek medical care for much of her life, but her dyspnea is now preventing her from carrying out her daily activities. In recent weeks, she has gained about 15 pounds and notes marked swelling of her ankles. She feels a fullness in her abdomen and notes that her pants are no longer fitting.

What does her ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

**ECG 72 Analysis:** Normal sinus rhythm, right bundle branch block (RBBB) associated with ST-T waves, old lateral wall myocardial infarction (MI), old anterolateral wall MI, right axis deviation due to an old lateral wall MI
There is a regular rhythm at a rate of 68 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The QRS complex duration is prolonged (0.16 sec), and there is a typical right bundle branch block morphology, that is, an RSR’ morphology in lead V1 (→) and a broad terminal S wave (←) in leads V4-V6. There are T-wave inversions (^) in leads V1-V3 that are associated with the right bundle branch block (RBBB). The QT/QTc intervals are normal (400/430 msec and 320/340 msec when corrected for the prolonged QRS complex duration).

There is a broad QS complex in lead I (↑) and two distinct negative deflections in lead aVL (↑↑), reflecting not only the RBBB but also a lateral wall myocardial infarction (MI) (ie, Q waves in leads I and aVL).

The right axis deviation (negative QRS complex in lead I and positive QRS complex in lead aVF) is the result of the lateral wall infarction and not a conduction abnormality (ie, a left posterior fascicular block, in which the QRS complex has an rS morphology in leads I and aVL). There are also significant Q waves in leads V5-V6 (▲), diagnostic for an old anterolateral MI.

This patient has clinical evidence of both right- and left-sided heart failure. Her ECG shows evidence of an old MI, which likely resulted in left ventricular systolic dysfunction. Although there are many etiologies for an RBBB, this conduction abnormality associated with clinical evidence of right-sided heart failure may be a consequence of longstanding left-sided heart failure with secondary pulmonary hypertension and now marked right ventricular dysfunction.
A 79-year-old woman with known mild aortic stenosis presents to her cardiologist after a hiatus of 5 years for evaluation of chest discomfort. She reports that the discomfort occurs with minimal activity such as doing light housework. She denies any pain at rest.

What does her ECG show?  
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 73 Analysis: Normal sinus rhythm, intraventricular conduction delay, left ventricular hypertrophy (LVH) with associated ST-T wave abnormalities
There is a regular rhythm at a rate of 76 bpm. There are P waves (*) before each QRS complex, and the P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The PR interval is stable (0.18 sec). The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is prolonged (0.12 sec), but the morphology is normal. This is, therefore, an intraventricular conduction delay. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (400/450 msec) but are actually normal when the increased QRS complex duration is considered (360/410 msec). There is marked increased amplitude of the QRS complex: 22 mm in lead aVL ( ), 28 mm in lead I ( ), and 31 mm in lead V6 ( ). In addition, the S wave depth is 32 mm in lead V2 ( ). These meet various criteria for left ventricular hypertrophy (LVH; R-wave amplitude in lead aVL ≥ 11 mm, R-wave amplitude in any limb lead ≥ 20 mm, and S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The presence of LVH is the reason for the intraventricular conduction delay as it takes longer to depolarize the thickened left ventricular myocardium. Associated ST-T wave changes ( ) are also seen in leads I, II, aVR (T wave should be negative in this lead), aVL, and V5-V6. These repolarization changes are the result of chronic subendocardial ischemia due to reduced sub-endocardial blood flow resulting from the hypertrophy.

In this case, LVH is likely attributable to aortic stenosis. Over time, the increased force generated by the left ventricle to overcome the stenotic aortic valve results in compensatory LVH. Exertional angina is a typical presenting symptom of progressive aortic stenosis (along with shortness of breath and syncope). Angina may be the result of concomitant coronary artery disease or due to relative ischemia and poor reserve of the hypertrophied myocardium, with a reduction in the supply of blood and oxygen to the subendocardium. Asymptomatic aortic stenosis can exist for many years before producing symptoms, but once a patient becomes symptomatic, there is often rapid progression of the disease. Aortic valve replacement is the only effective therapy for aortic stenosis and is indicated once a patient develops angina, syncope, or symptoms of heart failure.

The degree of aortic stenosis (as well as LVH, left atrial hypertrophy, left ventricular dysfunction, and other valvular abnormalities) should be evaluated by echocardiography. At this patient’s advanced age, the etiology of the aortic stenosis is most certainly degenerative calcific disease, but rheumatic and bicuspid aortic valves can also produce symptomatic aortic stenosis.
A 63-year-old man with a known bicuspid aortic valve and a history of hypertension presents to the emergency department with severe chest and mid-back discomfort. He was unloading groceries from his car when he developed 10/10 chest discomfort that caused him to fall to the ground.
His wife called an ambulance immediately. Upon arrival to the emergency department he has a blood pressure of 70/44 mm Hg and is intermittently alert. He vomits several times. An ECG (74A) is obtained. Based on the results, a second ECG (74B) is obtained.

**What do his ECGs show?**

**What is the likely diagnosis?**
Podrid's Real-World ECGs

ECG 74A Analysis: Normal sinus rhythm, premature atrial complex, counterclockwise rotation (early transition), acute inferior wall ST-segment elevation myocardial infarction (STEMI), prolonged QT interval
ECG 74A shows a regular rhythm at a rate of 88 bpm. There is a P wave (*) before each QRS complex, and the P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. The PR interval is constant (0.18 sec). This is a normal sinus rhythm. However, the ninth QRS complex (+) is early and is preceded by a P wave (▲) that has a different morphology than the other P waves. It is followed by a longer RR interval (↔). This is a premature atrial complex.

The QRS complex duration is normal (0.08 sec), and there is a normal morphology except for early transition, with a tall R wave (←) in lead V2. This is termed counterclockwise rotation, determined by imagining the heart as viewed from under the diaphragm. When there is counterclockwise electrical rotation, left ventricular forces are seen earlier in the precordial or chest leads, accounting for early transition with a tall R wave in lead V2. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (400/480 msec).

The T waves (↓) in leads II, III, and aVF are symmetric, and there is ST-segment elevation (↑) in these leads. The ST segments still have a concave morphology. This is an acute inferior wall ST-segment elevation myocardial infarction (STEMI). There is reciprocal ST-segment depression (^) in leads I, aVL, and V5-V6. The tall R wave in lead V2 could reflect posterior involvement of the myocardial infarction; however, this is less likely since lead V1 does not have a tall R wave.

Although there is ECG evidence of an acute inferior wall MI, the history of sudden-onset chest and mid-back discomfort is not common for a primary MI but is more typical of an aortic dissection, which should be considered. Of note, the presence of a bicuspid aortic valve puts a patient at increased risk for aortic dissection due to an association with aortic root and ascending aortic aneurysms. Another risk factor is the history of hypertension. Occasionally, a dissection flap will extend into the ostium of the right coronary artery and occlude flow, resulting in an acute inferior MI. An urgent transthoracic echocardiogram would be useful to evaluate the proximal aortic root for the presence of a dissection. Other useful imaging modalities include urgent computed tomography or transesophageal echocardiography. An aortic dissection involving the ascending aorta is a type A dissection, and the treatment involves prompt surgical repair.

continues
Podrid’s Real-World ECGs

ECG 74B Analysis: Right-sided leads, right ventricular infarction
In ECG 74B, the P waves (*), PR interval, QRS complexes, QT/QTc intervals, and ST-segment elevation (↑) in the limb leads are identical to those seen in ECG 74A. However, the precordial or chest leads are different and there is loss of R-wave amplitude in leads V4-V6. These are right-sided leads, which means the ECG was recorded with the precordial leads placed on the right side of the chest. Right-sided leads are commonly obtained when an inferior wall infarction is present to evaluate for an associated right ventricular infarction. ST-segment elevation (↓) is noted in leads V4R-V6R. This represents acute infarction of the free wall of the right ventricle (i.e., a right ventricular infarction). Right ventricular infarction may be present when the right coronary artery is occluded proximally. Diagnosing the presence of a right ventricular infarction is of clinical importance as patients are preload dependent, requiring an adequate volume to maintain normal right ventricular stroke volume to adequately fill the left ventricle. When right ventricular preload is reduced, as with nitrates or diuretics, there is a reduction in right ventricular stroke volume and left ventricular filling and hence a reduction in stroke volume. This causes a drop in blood pressure, as was seen in this patient, and raises concern for cardiogenic shock. Treatment in this situation involves giving an increased amount of fluids.
A 48-year-old man is evaluated for episodic headaches, sweating, and palpitations. Physical exam shows a prominent S4 and a sustained apical impulse. Blood pressure is 188/92 mm Hg supine and 155/80 mm Hg standing. Screening fractionated plasma free metanephrines are elevated.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 75 Analysis: Sinus bradycardia, left ventricular hypertrophy (LVH), U waves, early repolarization
There is a regular rhythm at a rate of 50 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a sinus bradycardia.

The QRS complex duration is at the upper limit of normal (0.10 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/400 msec). The R-wave amplitude (voltage) is significantly increased, ie, 50 mm in lead V5 (↑), which meets one of the criteria for left ventricular hypertrophy (LVH) (S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The T waves are tall and peaked-looking in leads V3-V4 (▲). However, the T-wave morphology is normal (ie, the T waves are asymmetric). The T waves are tall and more prominent as a result of LVH. Also noted is slight ST-segment elevation in leads V2-V3 (↑), which is early repolarization and is also commonly seen with LVH since with the increased voltage all components of the ventricular waveform are augmented. There are small Q waves in leads V4-V6 (▲) that are due to septal depolarization (septal Q wave).

Although the Q waves are prominent, they are in proportion to the amplitude of the R wave. Lastly, there are prominent U waves seen after the T waves in leads V2-V4 (▲). They are likely prominent because of LVH, which may make some of the ECG waveforms more prominent, including J-point elevation, T-wave amplitude, and U waves.

Given the clinical history and laboratory findings, this patient may have hypertension (and resultant LVH) attributable to a pheochromocytoma. Pheochromocytoma is a rare catecholamine-secreting tumor arising from the adrenal medulla. This patient demonstrates the classic triad of episodic headache, sweating, and tachycardia. Although these symptoms are typical, they are not unique to pheochromocytoma and can be seen in other cases of severe hypertension. This patient also demonstrates an orthostatic drop in blood pressure, which likely arises from intravascular volume depletion. If a pheochromocytoma is diagnosed, the patient should be treated with α-adrenergic receptor blocking agents and intravascular volume expansion before undergoing surgical resection of the tumor.
A 50-year-old man presents for a routine physical exam by a new provider at a primary care clinic. The physician notes a marked difference between the ECG in his chart from 1 year earlier (ECG 76A) and the one obtained in the clinic today (ECG 76B).
What do his ECGs show?
What accounts for this difference?
Podrid’s Real-World ECGs

ECG 76A Analysis: Normal sinus rhythm, ECG recorded at half-standard, left ventricular hypertrophy, left atrial hypertrophy or abnormality
ECG 76A shows a regular rhythm at a rate of 70 bpm. There is a P wave (•) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are upright in leads I, II, aVF, and V4-V6. This is a normal sinus rhythm. The P waves are notched in leads II and aVF and are primarily negative in lead V1; this is characteristic of left atrial hypertrophy or abnormality.

The QRS complex duration (0.10 sec), morphology, and amplitude are normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/370 msec). This is a normal ECG. However, it should be noted that the ECG was recorded at half-standard (←), in which 1 mV equals 5 mm (five small boxes), rather than the normal standard, in which 1 mV equals 10 mm (10 small boxes). Hence the amplitude (voltage) of the QRS complex (R-wave amplitude in lead V4 = 17 mm and S-wave depth in lead V3 = 13 mm) is actually twice the actual measurement. Thus the R-wave amplitude (voltage) in lead V4 is actually 34 mm and the S-wave depth in lead V3 is 26 mm (S-wave depth in lead V3 + R-wave amplitude in lead V4 = 60 mm); these values meet criteria for left ventricular hypertrophy (ie, S-wave depth in any precordial lead + R-wave amplitude in any precordial lead ≥ 35 mm).

Although many ECG machines indicate whether the ECG is recorded at half-standard or double standard, it is important to look at the standardization as recorded on the ECG (at the beginning or end of the tracing) to confirm what standardization was used. Failing to do this can result in an incorrect diagnosis of hypertrophy or low voltage.

continues
Podrid's Real-World ECGs

ECG 76B Analysis: ECG recorded at normal standard, left ventricular hypertrophy, left atrial hypertrophy, early repolarization
ECG 76B is recorded at normal standardization (→). The R-wave amplitude in leads V4-V5 (|) is in fact increased (35 mm), and the S-wave depth in leads V2-V3 (|) is increased (25 mm) (S-wave depth in lead V2 + R-wave amplitude in lead V5 = 60 mm). Left ventricular hypertrophy is indeed present (ie, S-wave depth in lead V2 + R-wave amplitude in lead V5 ≥ 35 mm). While the T waves are tall and prominent, they are asymmetric and prominent as a result of left ventricular hypertrophy. There is also evidence of early repolarization (↓) in leads V2-V3. In addition, the P waves (*) in leads II and aVF are notched and they are notched in leads V3-V4 (^), suggesting the presence of left atrial hypertrophy.
You are asked to review an ECG obtained in a 55-year-old woman who was evaluated at a medical walk-in unit. The patient has known longstanding hypertension. The treating physician asks you to comment on the ECG, which he thinks is abnormal.

What does the ECG show?

What is the likely explanation?
ECG 77 Analysis: Normal sinus rhythm, intraventricular conduction delay, counterclockwise rotation, left ventricular hypertrophy, ECG recorded at half-standard, ST-T wave changes
There is a regular rhythm at a rate of 70 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P waves are upright in leads I, II, aVF, and V4; they are not obvious in leads V5-V6. The P-wave morphology is normal. This is most likely a normal sinus rhythm.

The QRS complex duration is prolonged (0.12 sec), but the morphology is normal; hence this is an intraventricular conduction delay. In addition, there is a slight slowing in the upstroke of the R wave, which is termed a delayed intrinsicoid deflection (→). The R-wave upstroke is measured from the beginning of the QRS complex to the peak of the R wave, and when the duration is longer than 0.05 second the intrinsicoid deflection is delayed. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/430 msec and 360/390 msec when corrected for the prolonged QRS complex duration). Also noted is a tall R wave in lead V2 (←), which is termed early transition or counterclockwise rotation and is related to the electrical axis in the horizontal plane as determined by imagining the heart as viewed from under the diaphragm. When there is counterclockwise electrical rotation, left ventricular forces develop early and are seen in the right precordial leads; this causes the tall R wave in lead V2, or early transition.

The amplitude of the QRS complex is increased in leads I and II (30 mm) (↑), which meets criteria for left ventricular hypertrophy (ie, R-wave amplitude in any limb lead ≥ 20 mm). However, the measured amplitude of the R waves in the precordial or chest leads is not increased. However, the standardization (↓) noted at the beginning of the ECG tracing indicates that the limb leads were recorded at normal standard (1 mV = 10 mm or 10 small boxes) while the precordial or chest leads were recorded at half-standard (1 mV = 5 mm or five small boxes). Hence the measured R-wave amplitude in the precordial leads must be doubled to obtain their actual height. Therefore, the actual R-wave amplitude (voltage) in lead V5 is 50 mm, which meets one of the criteria for left ventricular hypertrophy (S-wave depth or R-wave amplitude in any one precordial lead ≥ 25 mm). Also noted are diffuse ST-T wave abnormalities (↑) in each lead. These are repolarization abnormalities that reflect chronic subendocardial ischemia resulting from reduced perfusion and oxygen supply to the endocardial layer of the hypertrophied myocardium. LVH would also be present based on the Romhilt-Estes criteria (ie, 7 points).
Notes
A 56-year-old woman is awakened from sleep with severe, crushing retrosternal chest discomfort. The discomfort is unrelenting, and after 5 minutes she calls emergency medical services. The ECG obtained on arrival is shown. She is given aspirin and sublingual nitroglycerin, and her discomfort and ECG changes resolve almost immediately.

What does her initial ECG show?
What is the likely diagnosis?
What are the likely findings on coronary angiography?
How should this condition be treated?
ECG 78 Analysis: Normal sinus rhythm, acute anterior wall ST-segment elevation myocardial infarction (STEMI) or transmural ischemia, acute lateral wall STEMI or transmural ischemia, left atrial hypertrophy
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4-V6. The P wave is negative in leads V1-V2, suggesting left atrial hypertrophy. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec). It is difficult to establish the QRS morphology and axis because of the significant ST-segment elevations (↓) in leads I, aVL, and V2-V6. This is an extensive acute anterior wall ST-segment elevation myocardial infarction (STEMI) involving the apex and anterolateral and lateral walls. The ST-segment morphology is convex, and it merges with the T wave. There is loss of R-wave amplitude, especially obvious in leads I, aVL, and V5-V6. The complex is said to show a current of injury, and it resembles the fast action potential that is generated by the ventricular myocardium as a result of the rapid influx of sodium ion. This is, therefore, a monophasic action potential. Also noted is significant ST-segment depression (↑) in leads II, III, and aVF; these are reciprocal changes.

An acute MI is identified by the presence of localized ST-segment elevation, hyperacute T waves (tall, peaked, and symmetric), and reciprocal ST-segment depressions (due to the same ST-segment elevations viewed from another angle). The location of these ST-segment elevations identifies the region of the myocardium involved.

This ECG is typical for an acute anterior STEMI, but the rapid resolution of the chest pain and ECG abnormalities is not typical. This patient most likely has variant angina (also called Prinzmetal’s angina), which is believed to be caused by coronary artery vasospasm and transient transmural myocardial ischemia. Most often, these patients have minimal or no demonstrable coronary artery disease on angiography. Provocative testing with intracoronary acetylcholine or ergonovine may induce vasospasm (and the resulting ECG changes) and can be used to establish the diagnosis. These patients should be treated with vasodilators such as nitroglycerin with each episode. There is a role for calcium-channel blockers and long-acting nitrates in the long-term control of symptoms when episodes are frequent.
A 64-year-old man is admitted to the emergency department with chest pressure that occurred 1 hour earlier while doing yard work. Although the pain decreased initially with rest, it continues to wax and wane. He is treated with aspirin and nitroglycerin with moderate relief. His presenting ECG is shown.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 79 Analysis: Sinus tachycardia, left ventricular hypertrophy, downsloping ST-segment depression
There is a regular rhythm at a rate of 100 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is upright in leads I, II, III, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a sinus tachycardia.

The QRS complex duration is normal (0.08 sec), and there is a normal QRS morphology with normal R-wave progression from leads V1 to V6. The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/440 msec). The QRS amplitude (voltage) in lead V4 is 38 mm ( ), meeting one of the criteria for left ventricular hypertrophy (S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm).

There is significant 2- to 3-mm (†) downsloping ST-segment depression (↑) in leads II, III, aVF, and V4-V6. The ST-segment elevation in lead aVR is actually ST-segment depression. In lead V3 there are upsloping ST-segment depressions (^). The ST-segment depression indicates myocardial ischemia, which may be the result of left ventricular hypertrophy or coronary artery disease.

This patient is experiencing an acute coronary syndrome. Although similar ECG changes can be seen with hypertension and left ventricular hypertrophy, in the context of the clinical scenario this patient requires immediate therapy for an acute coronary syndrome and an unstable coronary plaque. He should be treated with antiplatelet agents, nitroglycerin, anticoagulation, a statin, and β-adrenergic blocking agents depending on blood pressure and heart rate. Biomarkers such as creatine kinase (CK and CK-MB) and troponin are useful in determining whether there has been any myocardial necrosis. If there is evidence of infarction (ie, non-ST-segment elevation myocardial infarction or NSTEMI), patients most often undergo early invasive coronary intervention to restore adequate blood flow. If cardiac biomarkers are negative (ie, unstable angina) and the patient responds to medical therapy with resolution of the ST-segment changes, medical therapy can be continued with assessment of anginal control using exercise testing. ■
A 66-year-old woman presents with 2 hours of intermittent retrosternal chest pain. She was working in her garden when she became faint and diaphoretic and developed substernal chest discomfort. The pain lasted about 10 minutes, abated when she rested, and recurred shortly after she began working again. Eventually, she had a family member drive her to the emergency department, where an ECG was obtained.

What does her ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 80 Analysis: Normal sinus rhythm, short PR interval (enhanced AV nodal conduction or Lown-Ganong-Levine pattern), right atrial hypertrophy (abnormality), left atrial hypertrophy (abnormality), biatrial hypertrophy, ST-segment depression (ischemia), T-wave inversions, prolonged QT interval
There is a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex, with a stable but very short PR interval (0.12 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm.

The short PR interval (with a normal QRS complex) may be the result of enhanced AV nodal conduction or a preexcitation syndrome, known as Lown-Ganong-Levine, in which there is a bypass of the AV node due to an accessory pathway (known as a bundle of James). This accessory pathway links the atrium with the bundle of His, producing a short PR interval with a normal QRS complex morphology. The P waves are narrow and peaked in leads II, III, and aVF (+) (consistent with right atrial hypertrophy or abnormality), and they are negative in leads V1-V2 (−) (consistent with left atrial hypertrophy or abnormality). Therefore, there is evidence of biatrial hypertrophy.

The QRS complex duration is normal (0.08 sec), and the morphology is normal. There is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (460/530 msec). Deeply inverted T waves (↑) can be seen in most leads. The inverted T waves are symmetric. More importantly, there is ST-segment depression (↓) in leads II, aVF, and V3-V5. The T-wave changes, along with the ST-segment depression and QT interval prolongation, is strongly suggestive of ischemia.

Although this patient does not have the typical ST-segment elevations associated with myocardial infarction or the isolated downsloping ST-segment depression of myocardial ischemia, her ECG (ie, symmetrically inverted T waves) and the clinical history are consistent with myocardial ischemia. The diagnosis is an acute coronary syndrome, the result of an acute plaque rupture with subtotal occlusion of the vessel by active thrombus. If cardiac biomarkers are negative, consistent with a diagnosis of unstable angina, aggressive treatment of her symptoms (with anticoagulation, antiplatelet therapy, β-blocker, nitrates, and statins) would be the initial step. Depending on the patient’s response to medical therapy, the next step might be either a noninvasive ischemic evaluation (if she has stabilized) or proceeding directly to coronary angiography (if she remains unstable or has refractory symptoms). If cardiac biomarkers are positive, consistent with a diagnosis of a non-ST-segment myocardial infarction, aggressive therapy as above is often followed by cardiac catheterization within 48 to 72 hours along with percutaneous coronary intervention if indicated.
A 36-year-old woman presents to a cardiologist for the first time as an adult. She was followed for a heart murmur as a child but was lost to follow-up during her late teens. She feels well and has no complaints. Examination is notable for a jugular venous pressure of 12 cm H₂O, a systolic murmur at the left upper sternal border, a right-sided S4, and a widely split S2.

What does her ECG show?  
What is the likely diagnosis?  
What treatment is indicated?
ECG 81 Analysis: Normal sinus rhythm, right ventricular hypertrophy (RVH) with associated ST-T wave abnormalities, right axis deviation
There is a regular rhythm at a rate of 78 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P-wave morphology is normal.

The QRS complex duration is normal (0.08 sec), and there is a right axis deviation, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). There are a number of causes for a right axis deviation that should be considered. These include right ventricular hypertrophy (RVH; associated with a tall R wave in lead V1 and right atrial hypertrophy or abnormality), a lateral wall myocardial infarction (MI; a deep Q wave in leads I and aVL), right–left arm lead switch (associated with a negative P wave and T wave in leads I and aVL), dextrocardia (which resembles right–left arm lead switch and also has reverse R-wave progression across the precordium), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and a short PR interval), and a left posterior fascicular block (a diagnosis of exclusion when there are no other causes for the right axis deviation). The QT/QTc intervals are normal (400/450 msec).

There is a tall R wave in leads V1 (17 mm) and V2 (—). There are a number of causes for a tall R wave in lead V1, including RVH, posterior wall MI (usually with evidence of an inferior wall MI), Wolff-Parkinson-White pattern (with a prolonged QRS complex duration due to a delta wave and short PR interval), Duchenne muscular dystrophy (with a posterolateral MI pattern), hypertrophic cardiomyopathy (also with prominent septal Q waves in other leads), dextrocardia (reverse R-wave progression, right axis deviation, negative P and T waves in leads I and aVL), V1-V3 lead switch, recording of right-sided leads (reverse R-wave progression), and counterclockwise rotation. In this case, the tall R wave in lead V1 in conjunction with the right axis deviation is characteristic of RVH. The marked amplitude of the R wave in lead V1 is suggestive of more severe RVH and is often seen with congenital heart disease. In addition, there are ST-T wave changes (↑) in leads V1-V3; these are repolarization abnormalities associated with RVH and represent chronic subendocardial ischemia resulting from a reduction in perfusion and blood supply to the endocardial layer of the hypertrophied right ventricular myocardium.

This patient has evidence of marked RVH. This degree of RVH is usually associated with congenital heart disease. In this case it is due to congenital pulmonic stenosis. People with mild pulmonic stenosis can remain asymptomatic for many years. These patients are generally believed to have a benign disease with little risk for progression. However, if the obstruction is moderate it often leads to an inability to augment pulmonary blood flow during exertion, causing exercise intolerance, fatigue, or syncope. In such cases, balloon valvotomy is recommended to reduce the degree of obstruction. Pulmonic stenosis responds well to balloon valvotomy and rarely requires surgical correction.
A 54-year-old man presents for follow-up 6 weeks after suffering a myocardial infarction (MI). The treatment for his heart attack was delayed because the infarction occurred when he was traveling in a rural area. Since his MI he has been free of further symptoms.

What does his ECG show?
Which region of myocardium was affected?
Involvement of which vessel likely explains this pattern?
ECG 82 Analysis: Normal sinus rhythm, old inferior wall MI, old posterior wall MI, counterclockwise rotation (early transition), nonspecific T-wave abnormalities, left axis deviation due to an old inferior wall MI, left atrial hypertrophy or abnormality
There is a regular rhythm at a rate of 94 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is very negative in leads V1-V2, consistent with left atrial hypertrophy (or abnormality).

The QRS complex duration is normal (0.10 sec). The QT/QTc intervals are normal (320/400 msec). There are Q waves (↑) in leads II, III, and aVF, diagnostic for an old inferior wall myocardial infarction (MI). As a result of the inferior Q waves, the axis appears to be extremely leftward (between −30° and −90°). An extreme left axis deviation is also characteristic of a left anterior fascicular block (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). However, the left axis deviation in this case is a result of the inferior wall MI (QRS complexes have a Qr morphology) and is not a morphology of a fascicular block.

There is also a tall R wave in lead V1 (←), which, along with the inferior wall MI, is characteristic of posterior wall infarction; hence there is an inferoposterior wall MI. Simultaneous infarction of the posterior and inferior walls is most often caused by infarction of a large dominant right coronary artery (with posterolateral ventricular extension), although it can also occur from circumflex occlusion in the setting of a left dominant circulation.

The tall R wave in lead V2 (→) may be from the posterior wall infarction, but it is also likely the result of counterclockwise rotation or early transition. This is due to a shift of the electrical axis in the horizontal plane and is established by imagining the heart as viewed from under the diaphragm. When the electrical axis is shifted in a counterclockwise direction, left ventricular forces occur early and are prominent in the right precordial leads (ie, a tall R wave in lead V2).

Lastly, there are T-wave inversions (↑) present in leads I, aVL, and V2-V6. In the absence of any clinical history or data, these are considered nonspecific.
A 48-year-old African-American man presents to his primary care physician with epigastric discomfort for 3 weeks. The pain often occurs when he lies down to go to bed. It has not occurred with eating. His physician obtains an ECG.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 83 Analysis: Sinus bradycardia, left ventricular hypertrophy, early repolarization
There is a regular rhythm at a rate of 58 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6, and they have a normal morphology. This is sinus bradycardia.

The QRS complex duration is normal (0.08 sec), and the complexes have a normal morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/390 msec). The amplitude of the R wave in lead V4 is increased (38 mm) (↑), meeting one of the criteria for left ventricular hypertrophy (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). However, this voltage might be normal if this were a thin-chested, healthy man free of lung disease. There are small Q waves (↑) in leads I, aVL, and V4-V6, representing septal activation (septal Q waves). Although the T waves are prominent, especially in leads V2-V4, they are asymmetric and prominent as a result of left ventricular hypertrophy. J-point and ST-segment elevation (↑) are noted above the baseline (TP segment) in leads V2-V5. This is early repolarization, which is a normal finding often seen in association with left ventricular hypertrophy as well as in young patients with or without prominent R-wave amplitude. This pattern is more common among African Americans but is seen in people of all races. The T waves are asymmetric and are normal in morphology.

No changes on the ECG are suggestive of a cardiac cause for his abdominal discomfort. Indeed, the symptoms are not typical for myocardial ischemia or angina. The symptoms are more likely gastrointestinal and hence a gastrointestinal workup is indicated.
A 54-year-old man presents to the emergency department with fever, cough, and pleuritic chest pain. On exam he is noted to be tachycardic and has bronchial breath sounds in the left lower lung zone. An ECG is obtained.

What does his ECG show?
What is the likely diagnosis?
Podrid's Real-World ECGs

ECG 84 Analysis: Sinus tachycardia, nonspecific ST-segment changes
There is a regular rhythm at a rate of 110 bpm. There is a P wave (*) before each QRS complex, and the P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a sinus tachycardia.

The QRS complex duration is normal (0.08 sec), and it has a normal morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There is normal R-wave progression from leads V1 to V6. The QT/QTc intervals are normal (320/430 msec). ST-segment flattening (↑) is noted in leads II and V4-V6. This is a nonspecific ST-segment change. The ST segments in leads III and aVF (↑) are downsloping but are less than 1 mm below baseline (the TP segment). These are also nonspecific ST-segment changes.

There is no definitive evidence of myocardial ischemia on this ECG, suggesting that the patient’s pleuritic chest pain is noncardiac in nature. There is no clinical or ECG evidence for an acute coronary syndrome. The presence of fever, tachycardia, and bronchial breath sounds makes a pulmonary infection a more likely diagnosis. Nonspecific ST-segment changes are common in patients with concomitant medical illnesses.
A 76-year-old woman with hypertension and diabetes develops lightheadedness and diaphoresis while sitting at the dinner table with her family. She begins to feel nauseated and vomits. Her family calls emergency medical services, who find her appearing fatigued and diaphoretic. She complains of mild chest discomfort and is given one tablet of sublingual nitroglycerin, after which she transiently loses consciousness.

What does her ECG show?
What is the likely diagnosis?
What coronary artery is likely to be involved?
ECG 85 Analysis: Normal sinus rhythm, intraventricular conduction delay, acute inferior wall ST-segment elevation myocardial infarction (STEMI)
There is a regular rhythm at a rate of 86 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), as is the axis, which is between 0° and +90° (positive QRS complex in leads I and aVF). There is an RSR' morphology (--) in lead V1, which represents a conduction delay to the right ventricle (intraventricular conduction delay). The QT/QTc intervals are normal (340/410 msec).

ST-segment elevation (↓) with symmetric T waves is noted in leads II, III, and aVF. The ST segments still have a concave morphology. This is, therefore, an acute inferior wall ST-segment elevation myocardial infarction (STEMI). Q waves (^) are present in leads III and aVF. Also noted are ST-segment depressions (↑) in leads I and aVL; these are reciprocal changes.

This patient demonstrates marked sensitivity to nitroglycerin. The action of nitroglycerin is venodilation and a reduction in venous return. Hypotension with this medication can occur in the setting of volume depletion. Another situation in which hypotension can occur with nitroglycerin is infarction of the free wall of the right ventricle, which may be associated with an inferior wall STEMI. With a right ventricular infarction the left ventricle may be underfilled due to poor right ventricular output and a situation that resembles venous pooling of blood. Administration of venodilators, such as sublingual nitroglycerin, can augment this effect by reducing right ventricular filling and hence right ventricular stroke volume and left ventricular filling. This may produce profound hypotension as was observed in this case. The treatment is fluids to maintain right ventricular pressure and hence left ventricular filling. Another cause for hypotension is enhanced vagal tone, which often occurs with an inferior wall STEMI. The hypotension may be enhanced with the use of nitroglycerin. Right ventricular infarction can occur in the setting of a very proximal right coronary artery occlusion.
A 55-year-old man is seen in clinic for a preoperative evaluation before undergoing elective surgery for an abdominal aortic aneurysm. He has hypertension, dyslipidemia, and a 40 pack-year smoking history. As he walks into the clinic, he complains of substernal chest discomfort and an ECG is quickly obtained. Blood pressure at the time is 190/90 mm Hg.

What does his ECG show?
What is the likely diagnosis?
ECG 86 Analysis: Normal sinus rhythm, left ventricular hypertrophy (LVH), horizontal ST-segment depression, left atrial hypertrophy or abnormality
There is a regular rhythm at a rate of 78 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. This is a normal sinus rhythm. The P wave is primarily negative in lead V1, suggesting left atrial hypertrophy or abnormality.

The QRS complex duration (0.10 sec) and morphology are normal. The axis is normal, between 0° and +90° (positive QRS complex in lead I and aVF). The QT/QTc intervals are normal. There is normal R-wave wave progression from leads V1 to V6. However, the R-wave amplitude (voltage) in lead V5 is increased (30 mm) as is the S-wave depth (voltage) in lead V3 (28 mm) (S-wave depth in lead V3 + R-wave amplitude in lead V5 = 58 mm). Criteria for left ventricular hypertrophy (LVH) are present (ie, S-wave depth in any precordial lead + R-wave amplitude in any precordial lead ≥ 35 mm). There is J-point and 1- to 2-mm horizontal ST-segment depression (↑) noted in leads I, aVL, and V3-V6. This represents subendocardial ischemia, which may be the result of coronary artery disease or underlying LVH.

This patient has LVH on the basis of longstanding hypertension. The ECG changes could be related to subendocardial ischemia caused by increased wall stress occurring as a result of markedly elevated blood pressure. However, the chest pain and ECG changes may also represent epicardial coronary artery disease and exertional angina as it is common for angina due to coronary artery disease to be associated with a hypertensive response. This patient should undergo further evaluation. The chest discomfort and markedly elevated blood pressure should be treated immediately, and a repeat ECG should be obtained. Once blood pressure is controlled, the patient should undergo further coronary risk stratification, likely with some form of noninvasive evaluation, such as a treadmill exercise test or exercise echocardiogram. The exercise test would also be important to document the blood pressure response and evaluate the adequacy of antihypertensive therapy. If there is evidence of provoked ischemia, further therapy with anti-ischemic medications would be important.

The diagnosis of coronary artery disease before a major surgery poses a difficult problem. If the patient presents with an acute coronary syndrome, he or she should undergo revascularization before surgery not only to get through the surgery but also for clinical reasons. Elective revascularization prior to noncardiac surgery (especially high-risk vascular surgery) in the absence of an acute coronary syndrome or a clinical indication is not necessary; the outcome with medical therapy is the same as with revascularization (CARP trial).
A 78-year-old man develops chest discomfort while on an international flight. The discomfort started shortly after takeoff and persisted despite taking sublingual nitroglycerin. Three hours into the flight, he alerts the flight staff, who divert the plane to land urgently. The plane lands about 1 hour later, and the patient is rushed to the emergency department. Upon questioning, he recalls that he had a myocardial infarction (MI) 10 years earlier. The heart attack was treated with an angioplasty and medical therapy.

What does his ECG show? What is the likely diagnosis?
ECG 87 Analysis: Normal sinus rhythm, first-degree AV block, anteroapical MI, low-voltage limb leads
There is a regular rhythm at a rate of 60 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.24 sec). The P wave is upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm with a first-degree AV block.

The QRS complex duration is borderline normal (0.10 sec), and the axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex amplitude (voltage) is low in the limb leads (defined as a QRS amplitude ≤ 5 mm in each limb lead), and hence there is low voltage. The QRS complex morphology is abnormal, and there are QS complexes (↓) in leads V2-V4, diagnostic for an antero-apical myocardial infarction (MI). The QT/QTc intervals are normal (440/440 msec and 420/420 msec when corrected for the prolonged QRS complex duration).

The ST segments (^) are still elevated, and the T waves (+) have become inverted. Therefore, this might be a later stage of an acute ST-segment elevation MI (STEMI) that is evolving. It could, however, represent an old MI with the presence of an aneurysm. To establish whether these findings represent evolutionary changes or an aneurysm, it would be important to know the patient’s history, prior ECGs, and cardiac biomarkers. If the event were recent, cardiac biomarkers would be elevated and prior ECGs would not show any evidence of a previous MI in the same leads. The ECG abnormalities would therefore represent an acute MI in evolution. In contrast, if a prior ECG showed evidence of an infarction in the same location and if cardiac biomarkers were not elevated, then the ECG would represent an aneurysm.
A 72-year-old man presents to the emergency department with chest pain. The attending physician asks the resident if there is any evidence of ischemia or infarction on his ECG. The resident replies that there is a left bundle branch block and hence acute changes cannot be established.

Can one assess for ischemia on this ECG?
What does his ECG show?
What is the likely diagnosis?
**Podrid's Real-World ECGs**

**ECG 88 Analysis:** Sinus bradycardia, intraventricular conduction delay, old lateral wall myocardial infarction (MI), old anterior wall MI, left anterior fascicular block, first-degree AV block
There is a regular rhythm at a rate of 58 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.26 sec). The P waves are upright in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm with first-degree AV block or prolonged AV conduction.

The QRS complex duration is prolonged (0.14 sec). There is a pattern resembling a left bundle branch block (LBBB), with a broad R wave in lead I and a deep, broad S wave in lead V1. However, there is an initial R wave in lead V1 (↓), indicating an initial septal force, as well as Q waves (↑) in leads I and aVL. In addition, there are broad QS complexes in leads V4-V6. Although this can be seen with an LBBB, the QS complexes in leads V4-V6 generally mean that the electrical impulse is going in a left-to-right direction, which does not occur with an LBBB (in which all the ventricular forces travel in a right-to-left direction only). The rightward-directed forces, when associated with the presence of a septal R wave in lead V1 and lateral Q waves, means that this is not an LBBB but rather an intraventricular conduction delay. In this situation the Q waves in leads I and aVL are diagnostic for an old lateral wall myocardial infarction (MI). There are also QS complexes (↑) in leads V2-V6, diagnostic for an extensive anterior wall MI. The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology), a result of a left anterior fascicular block. The QT/QTc intervals are normal (440/430 msec and 380/370 msec when corrected for the prolonged QRS complex duration).

Although it is difficult to make the diagnosis of myocardial ischemia in the context of an LBBB, physicians must be careful not to confuse an anterior MI with an LBBB as the QRS complex morphology can be similar (ie, a QS complex in the precordial leads). As the ECG shows intact septal forces, resulting from septal activation that occurs via a septal branch arising from the left bundle, the left bundle cannot be blocked. Therefore, this patient does not have an LBBB but has an intraventricular conduction delay associated with a prior lateral and anterior MI. Hence in this situation the ECG can be evaluated with regard to ischemic changes or other abnormalities of the left ventricular myocardium. Axis can also be determined. With an LBBB, left ventricular activation is not via the normal His-Purkinje system but rather by direct myocardial activation. Hence, abnormalities of the left ventricle cannot be reliably diagnosed. In contrast, an intraventricular conduction delay is slowed conduction through the normal His-Purkinje system. Hence left ventricular activation is via the normal conduction system and left ventricular abnormalities can be diagnosed.
A 72-year-old woman with chronic kidney disease on hemodialysis presents to her primary care physician for evaluation of chest discomfort. She has missed two successive hemodialysis sessions due to a family emergency. She reported worsening of the discomfort with inspiration and supine positioning.

What does her ECG show?
What is the likely diagnosis?
ECG 89 Analysis: Normal sinus rhythm, premature junctional complex, pericarditis
There is a regular rhythm at a rate of 94 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.14 sec). The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.10 sec), and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/400 msec). The QRS complex morphology is normal, although there are small and narrow Q waves (*) in leads II and aVF and a wider and deeper Q wave in lead III (▲). Lead III is an indeterminate lead, and the Q waves in leads II and aVF do not meet criteria for an old myocardial infarction (ie, > 0.04 sec and >1 mm deep) and therefore represent septal forces.

The eighth QRS complex is early or premature (+). It has the same QRS morphology as the other complexes. There is no apparent P wave before this early complex. Therefore, this is a premature supraventricular complex and is likely a premature junctional complex.

There is diffuse J-point and ST-segment elevation (†) above the baseline (TP segment) in leads I, II, aVR (ST-segment depression in this lead is actually elevation), aVL, aVF, and V3-V6. The ST segments have a concave morphology. The T waves are normal and asymmetric. The ECG shows pericarditis.

This patient likely has acute uremic pericarditis as a consequence of having missed two successive hemodialysis sessions. Uremic pericarditis responds to initiation of hemodialysis and tends to respond poorly to anti-inflammatory medications.
A 36-year-old man with type 1 diabetes mellitus is found collapsed on the floor of his apartment. He has been drinking heavily and has not been seen by any of his family members for 48 hours. He is intubated in the field and brought to the nearest emergency department. There he is found to have a blood glucose level of 780 mg/dL, potassium level of 6.7 mmol/L, creatinine level of 4.8 mg/dL, and an anion gap of 28.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 90 Analysis: Sinus bradycardia, old anteroseptal myocardial infarction (MI), nonspecific T-wave abnormalities
There is a regular rhythm at a rate of 54 bpm. The “waveform” seen between the first and second QRS complexes is artifact (^). There is a P wave (*) before each QRS complex, with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is sinus bradycardia.

The QRS complex duration is normal (0.10 sec), and the axis is physiologically leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (470/440 msec). There are no R waves (↓) in leads V1-V3 (hence QS complexes), diagnostic for an old antero-septal myocardial infarction (MI).

The T waves are abnormal and are biphasic in leads V1-V3 and V6 (+), while they are inverted in leads V4-V5 (↑). The T waves are asymmetric in morphology. These are, therefore, nonspecific T-wave abnormalities and they may be seen in various conditions, including ischemia, myocarditis/pericarditis, metabolic or pH abnormalities, old MI, or left ventricular hypertrophy; they may also be seen in healthy subjects as a normal variant. T-wave abnormalities must be considered along with a clinical history. In this case of diabetic ketoacidosis, these T-wave abnormalities are likely due to profound metabolic disarray and acidosis. With several days of supportive care and resolution of the metabolic disturbance, these ECG changes will likely resolve.
A 38-year-old man is involved in a high-speed motor vehicle accident. He suffered a chest contusion against the steering wheel and blunt head trauma. Upon arrival to the emergency department he is somnolent and hypotensive.

What does his ECG show?
What is the likely diagnosis?
Podrid’s Real-World ECGs

ECG 91 Analysis: Normal sinus rhythm, left anterior fascicular block, prolonged QT interval, cerebral T waves
There is a regular rhythm at a rate of 66 bpm. There are P waves (*) before each QRS complex, and the P waves are positive in leads I, II, aVF, and V4-V6 and negative in lead aVR. The P-wave morphology is normal. This is a normal sinus rhythm.

The QRS complex duration is normal (0.08 sec), and there is an extremely left axis deviation, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology). This is a left anterior fascicular block. An inferior wall myocardial infarction may also be associated with an extreme left axis deviation; however, the QRS complexes would have a deep initial Q wave. The QRS morphology is normal. Deeply inverted and asymmetric T waves (↑) are seen in most leads (the T wave in lead aVR should be negative). Unlike the normal T wave, which has a slower initial portion or upstroke (ascent) and a faster terminal portion or downstroke (descent), the initial portion (descent) of the T wave is more rapid than the terminal portion (ascent) of the T wave. The ST segment is not depressed, and the QT/QTc intervals are prolonged (620/650 msec). These T-wave abnormalities, associated with the QT interval prolongation, are suggestive of cerebral T waves, a result of a central nervous system process such as a subarachnoid hemorrhage, cerebral hemorrhage, tumor, head injury, or infection.

This patient likely has these ECG changes on the basis of his head injury and possible intracranial bleed. He will require therapy to lower his intracranial pressure, including elevation of the head of the bed, mannitol infusion, hyperventilation, and possible surgical evacuation. The clinician should also be mindful of the possibility of traumatic injury to the heart or coronary arteries. Aortic dissection, coronary artery dissection, or cardiac contusion could be caused by blunt chest trauma and could produce a similar ECG pattern, although if this were the result of myocardial ischemia, the ST segments would be depressed and the T waves would be symmetric. ■
A 56-year-old man with hypertension but no cardiac history has been treated with lisinopril and hydrochlorothiazide for the past 4 years. He states that his blood pressure has been well controlled whenever measured. He presents with a complaint of headache.
associated with sinus congestion and a productive cough associated with chest pain. His physical examination is unremarkable, with a blood pressure of 160/80 mm Hg. The ECG (92A) that is obtained during this visit is found to be different from that obtained 1 year earlier (ECG 92B).

What abnormality on the current ECG is a cause for concern?

What is the change between the two ECGs?

Is any additional evaluation necessary?
Podrid’s Real-World ECGs

ECG 92A Analysis: Normal sinus rhythm, tall QRS voltage, ECG recorded at double standard
ECG 92A shows a regular rhythm at a rate of 66 bpm. There are P waves (*) before each QRS complex, with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. The P-wave morphology is normal. This is a normal sinus rhythm. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/400 msec). U waves (^) are seen in leads V1-V3.

The QRS complex duration (0.08 sec) and morphology are normal. However, the QRS complex amplitude is increased; the R-wave amplitude is 28 mm in lead V5 ( ), and the S-wave depth is 22 mm in lead V2 ( ). This meets a criterion for left ventricular hypertrophy (ie, S-wave depth in lead V2 + R-wave amplitude in lead V5 ≥ 35 mm). In addition, the R-wave amplitude (voltage) in lead aVL ( ) is 13 mm, also a criterion for left ventricular hypertrophy (ie, R-wave amplitude ≥ 11 mm).
ECG 92B Analysis: Normal
When compared with ECG 92B, which was obtained 1 year earlier, the P waves, PR interval, QRS complex duration, QRS complex morphology, and axis are the same. However, there is a marked difference in the QRS complex amplitude, which was a cause for concern as it was believed that the patient’s blood pressure was not well controlled and that he had developed left ventricular hypertrophy. However, closer examination of ECG 92A reveals that it was recorded at double standard (→). Therefore, the QRS amplitude is only half of what is measured. When this is taken into account, it can be seen that the QRS amplitude is normal and is identical to that noted in ECG 92B, which was recorded using normal standardization (←).

ECGs are generally recorded using normal standardization, that is, 1 mV of electrical current produces a signal that measures 10 mm in amplitude (or 10 small boxes). On occasion, however, the ECG will be recorded using double standard. This may be the case when the waveforms are very small and difficult to interpret or if waveforms are not clearly seen and may become more apparent with an increased amplitude. Not uncommonly, the ECG is recorded using double standard by mistake, as appears to be the situation with this patient. Once recognized, it is clear that there has not been any change in the ECG compared with 1 year earlier. Hence there is no left ventricular hypertrophy and no additional evaluation or therapy is necessary. An ECG recorded at normal standardization could be obtained in order avoid confusion in the future.
acute anterior wall myocardial infarction, 112–113
acute anteroapical ST-segment elevation myocardial infarction, 121, 386–387
acute coronary syndrome, 39, 51, 307, 355, 359
acute inferior wall transmural ischemia caused by vasospasm, 152–153
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Podrid’s Real-World ECGs combines traditional case-based workbooks with a versatile Web-based program to offer students and physicians an indispensable resource for developing the technical skills and systematic approach needed to interpret ECGs with confidence.

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Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can.

— From the Foreword by Hein J. Wellens, MD

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A Master’s Approach to the Art and Practice of Clinical ECG Interpretation

Volume 3  Conduction Abnormalities

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy and daughter Sapna, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

Peter A. Noseworthy
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Foreword

The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven’s landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiodiagnostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today’s cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled “Real-World ECGs,” by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient’s clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.

continues
These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important dividend for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, realworldECGs.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

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The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable “workhorse” in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician's office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of Podrid's Real-World ECGs aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in Podrid's Real-World ECGs teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

Volume 1 provides an essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the
Podrid’s Real-World ECGs

The mechanism, diagnosis, and treatment of atrioventricular (AV) and intraventricular conduction disturbances and enhanced AV conduction. The remaining volumes focus on other disease entities for which the ECG is useful:

- Atrial and ventricular hypertrophy, acute myocardial ischemia, acute and chronic myocardial infarction, and pericarditis
- Sinus, atrial, junctional, and ventricular arrhythmias
- Narrow and wide complex tachycardias and forms of aberration
- Recording methods and miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs and clinical scenarios to further enhance the student's skills at ECG analysis. Importantly, each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: realworldECGs.com

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers easy access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q&A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

Philip Podrid, MD
Rajeev Malhotra, MD, MS
Rahul Kakkar, MD
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Introduction
Conduction Abnormalities

There are two general types of conduction abnormalities in the heart. Atrioventricular (AV) conduction abnormalities affect impulse transmission from the atria to the ventricles, while intraventricular conduction abnormalities affect impulse conduction through the ventricular myocardium.

Atrioventricular Conduction Abnormalities

AV conduction abnormalities may result from problems with impulse transmission through either the AV node or the His-Purkinje system. There are three types of AV conduction abnormalities: first-degree, second-degree, and third-degree block.

First-degree Atrioventricular Block

First-degree AV block is defined simply by a prolonged PR interval (ie, > 0.20 sec). However, the PR interval includes the P wave, representing atrial depolarization, and the PR segment, which represents conduction through the AV node and His-Purkinje system. With first-degree AV block, it is the PR segment that is prolonged as a result of slow conduction through either the AV node or the His-Purkinje system. This is usually the result of slowing of conduction through the AV node. Because the impulse does conduct from the atria to the ventricles, it is not accurate to refer to this as AV block; it is more appropriately termed prolonged or slow AV conduction.

Because the P wave is part of the PR interval, a very broad P wave, as seen in left atrial hypertrophy or abnormality, may cause the PR interval to be slightly prolonged while the PR segment is normal. It should be noted that AV nodal conduction velocity is variable and can change with heart rate as a result of change in autonomic inputs affecting AV nodal conduction velocity. Hence with sinus tachycardia, there is enhancement of sympathetic tone and an increase in conduction velocity through the AV node; this results in a shortening of the PR interval. With enhanced parasympathetic tone, resulting in sinus bradycardia, there is prolongation of the PR interval due to a slowing of conduction through the AV node. However, there is no correction equation available to establish a rate-corrected PR interval. For example, a PR interval of 0.22 second is prolonged and by definition would be first-degree heart block. However, if the heart rate were 30 bpm, this PR interval would be appropriate for bradycardia. Likewise, a PR interval of 0.20 second, defined as normal, would probably be prolonged for a heart rate of 150 bpm.

Second-degree Atrioventricular Block

Second-degree AV block is identified by a pause in the RR interval due to an occasional nonconducted P wave. The PP intervals (ie, sinus or atrial impulses) are constant. The PR intervals may be normal or prolonged as first-degree AV block may be present along with
second-degree AV block. There are two types of second-degree AV block as the block may be in either the AV node (Mobitz type I) or the His-Purkinje system (Mobitz type II).

*Mobitz Type I Atioventricular Block (Wenckebach)*

Mobitz type I AV block (Wenckebach) results from a conduction abnormality within the AV node. In Wenckebach, progressive PR interval prolongation results in a single nonconducted P wave and pause in the RR interval. The duration of the pause (the PP interval around the pause) is equal to two sinus intervals. After the pause, the PR interval shortens to the baseline PR interval. Hence the baseline interval is the PR interval of the complex immediately following the pause, and the PR interval after each pause should be the same. It may be normal or prolonged. There is only one nonconducted P wave each time, and hence there will be one more P wave than QRS complex. Therefore, Wenckebach is termed 3:2, 4:3, 5:4, etc. This results in a pattern of grouped beating. The pattern of block may be fixed (only 3:2, only 4:3, etc) or variable (periods of both 3:2 and 4:3, etc).

The basis of Mobitz type I AV block is that impulse generation within the AV node results from calcium currents (ie, a slow action potential). The slow action potential demonstrates changes in refractoriness and conduction velocity. Therefore, the rate of impulse conduction through the AV node is variable. AV nodal refractoriness and conduction velocity change as a result of various factors, including autonomic impulses, drugs, and electrolyte abnormalities. Because of changes in refractoriness, impulse velocity can change; that is, the ability of the AV node to conduct impulses is not all or none, as is seen in the His-Purkinje system where impulse generation is the result of fast sodium currents (ie, the fast action potential). Therefore, the AV node demonstrates a property known as decremental conduc­tion, (ie, conduction velocity through the AV node changes based on how rapidly the node is stimulated). In the absence of sympathetic stimulation, when the rate of impulse stimulation of the AV node increases, the impulse conduction velocity through it slows. This results in lengthening of the PR interval. Each successive atrial impulse reaches the AV node earlier, when it may be even more refractory. As a result, there is a progressive slowing of each subsequent impulse due to decremental AV nodal conduction, until the node finally fails to conduct the impulse because it has arrived at a time when it is absolutely refractory. As a result of blockade of impulse conduction within the AV node, there is no activation of the ventricle and no QRS impulse on the ECG. The increment of PR lengthening is greatest at the beginning of a Wenckebach cycle and may decrease progressively from one beat to the next. For example, the PR interval may lengthen from the baseline of 0.16 second, to 0.24 second, then to 0.28 second, and finally to 0.30 second. Since the PP interval is constant, this results in shortening of the RR interval. However, shortening of the RR interval may not be seen, especially if there is a long Wenckebach cycle length or if the underlying heart rate is fast. Hence this finding is not necessary to identify Wenckebach.

Although Wenckebach is seen most often in sinus rhythm, it may occur with an atrial rhythm, atrial tachycardia, or atrial flutter. Regardless of the underlying rhythm, there is a regular PP interval.

Wenckebach is most often benign and does not require any therapy. Importantly, if a failure of impulse conduction from atria to ventricles
Introduction: Conduction Abnormalities

would develop (ie, complete heart block), the escape rhythm would be junctional. Junctional rhythms are generally reliable, predictable, and stable with regard to rate. In general, urgent pacemaker insertion is not required unless there are symptoms resulting from bradycardia.

Mobitz Type II Atrioventricular Block

With Mobitz type II AV block, there is an occasional nonconducted P wave, but when there is AV conduction the PR interval is constant (ie, the PR intervals before and after the pause are constant). There may be one or more nonconducted P waves; the occurrence of two or more nonconducted P waves is often called high-degree AV block. Mobitz type II block is failure of conduction within the His-Purkinje system. Since impulse generation within the His-Purkinje system results from the fast action potential mediated by a rapid influx of sodium currents, conduction through the His-Purkinje system is all or none; that is, it either conducts (always at the same rate) or does not conduct. Therefore, when there is AV conduction, the PR interval is always the same. Mobitz type II block has more serious implications because if there is a failure of AV conduction (ie, complete heart block), the escape rhythm will be ventricular. Ventricular escape rhythms are unreliable, unpredictable, and unstable with regard to rate. A temporary pacemaker should be inserted, even if there are no symptoms.

2:1 Atrioventricular Block

In 2:1 AV block, which is also a type of second-degree AV block, every other P wave is nonconducted. When the P wave is conducted, the PR interval is constant. 2:1 AV block may be Mobitz type I or II. The etiology can only be established if another pattern of AV conduction is seen as a result of several sequentially conducted P waves. If there is progressive lengthening of the PR interval, then the 2:1 AV block is Mobitz type I. In contrast, if all the PR intervals are the same, then the 2:1 AV block is Mobitz type II. Alternatively, if complete heart block develops, the presence of an escape junctional rhythm indicates that the problem is within the AV node and hence the 2:1 AV block is Wenckebach. If there is an escape ventricular rhythm, the 2:1 AV block is within the His-Purkinje system and hence is Mobitz type II.

Third-degree Atrioventricular Block

Third-degree AV block (complete heart block) is manifest by the presence of AV dissociation; that is, there is no association between P waves and QRS complexes. Hence the PR intervals are variable and the variability has no pattern (unlike Mobitz type I, in which there is a pattern to the variability of the PR intervals, ie, progressive lengthening). In addition, the atrial rate is faster than the rate of the QRS complexes as the QRS complexes are the result of an escape rhythm. Complete heart block may be due to disease of the AV node or the His-Purkinje system (ie, infranodal); therefore, the escape rhythm may be junctional or ventricular. The origin of the escape rhythm is based on QRS morphology and not the rate of the escape rhythm.

Atrioventricular Dissociation

AV dissociation is identified by the absence of a relationship between the P wave and the QRS complex. Hence there are variable PR intervals that are random without any pattern to the variability. In contrast, there is variability of the PR intervals with Wenckebach, but there is a
pattern with progressive PR interval lengthening. There are two causes for AV dissociation:

- Third-degree AV block, or complete heart block, in which there is no impulse conduction between the atria and ventricles. In this situation, the atrial rate is faster than the rate of the QRS complexes, which are the result of an escape rhythm. The location of the escape rhythm may be the junction (junctional escape) or the ventricle (ventricular escape). The location of the escape rhythm is not based on the rate of the escape rhythm but on the morphology of the QRS complexes. Although the teaching is that junctional escape rhythms have a rate of 50 to 60 bpm and ventricular escape rhythms have a rate of 20 to 30 bpm, the rate is influenced by autonomic tone and circulating catecholamines. Hence there may be an escape junctional rhythm at a rate of 30 bpm and an escape ventricular rhythm at a rate of 70 bpm.

- An accelerated junctional or ventricular rhythm may also present with AV dissociation. In this situation, the atrial rate is slower than the rate of the QRS complexes. The focus responsible for the accelerated rhythm (junction or ventricle) is based on the QRS complex morphology and not on the rate.

**Isorhythmic Dissociation**

Isorhythmic dissociation is a variation of AV dissociation. As noted, with complete heart block the atrial rate is faster than the rate of the QRS complexes, while with an accelerated rhythm the atrial rate is slower than the rate of the QRS complexes. However, if AV dissociation is present (ie, variable PR intervals), but the rate of the P waves and QRS complexes is the same, the etiology for AV dissociation (ie, complete AV block or an accelerated lower focus) cannot be established. This is termed isorhythmic dissociation.

**Ventriculophasic Arrhythmia**

Ventriculophasic arrhythmia (ie, phasic changes in the sinus rate related to ventricular contraction) may be seen with 2:1 AV block or AV dissociation, particularly when there is complete heart block. Ventriculophasic arrhythmia is diagnosed when the sinus PP intervals are irregular with a repeating pattern (ie, the PP interval surrounding a QRS complex is slightly shorter than the PP interval without a QRS complex). This is termed ventriculophasic arrhythmia and is due to one of several causes:

- With ventricular contraction there is an increase in pulsatile sinus nodal artery blood flow that results in an increase in sinus node automaticity and hence the shorter PP interval.
- With ventricular contraction the stretch on the right atrium enhances sinus node automaticity.
- With ventricular contraction (and hence stroke volume) there are effects on the baroreceptors and, as a result, there are changes in vagal outputs that alter the sinus rate.

**Intraventricular Conduction Abnormalities**

Intraventricular conduction abnormalities may be due to one of three causes:

- Diffuse slowing of impulse conduction through the normal His-Purkinje system that innervates the ventricles (known as intraventricular conduction delay [IVCD])
Introduction: Conduction Abnormalities

• Block of conduction through either fascicle of the left bundle (left anterior or left posterior fascicle)

• Block of conduction within one of the bundle branches (right or left)

Impulse conduction of the ventricles for their activation or depolarization is via the His-Purkinje system, which originates at the distal portion of the AV node as a cylindric structure that contains many tracts; this is the bundle of His. This structure divides into two major bundles: a right bundle that innervates the right ventricle and a left bundle that innervates the left ventricle. As left ventricular muscle mass is far greater than right ventricular muscle mass, the left bundle divides into two major and one minor fascicles. The minor fascicle is the septal (intermediate or median) branch, which innervates the interventricular septum (the first part of the ventricle to depolarize in a left-to-right direction). Hence leads that are located laterally or in a right-to-left direction (ie, leads I, aVL, and V4-V6) often show a small septal Q wave (septal depolarization goes away from these leads), while lead Vl, which is on the right side of the sternum, has a small septal R wave as the septal impulse goes toward this lead. The two major fascicles are the left anterior fascicle and the left posterior fascicle:

• The left anterior fascicle crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle.

• The left posterior fascicle appears as an extension of the main bundle and fans out extensively posteriorly toward the papillary muscle and inferoposteriorly toward the free wall of the left ventricle.

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**Figure 1.** The QRS axis in the frontal plane is determined by analyzing the direction of the QRS complex in the limb leads. The heart is divided into four equal quadrants of 90° each (0° to +90°, +90° to +/-180°, 0° to –90° and –90° to +/-180°). The two leads that are perpendicular to each other and divide the heart in this fashion are leads I and aVF. Hence these two leads are looked at first. A normal axis is between 0° and +90° (positive QRS complex in leads I and aVF). A right axis, which is never normal, is between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). A left axis (ie, between 0° and –90° with a positive QRS complex in lead I and a negative QRS complex in lead aVF) may be physiologic (and hence normal) if it is between 0° and –30° or pathologic (and hence abnormal) if it is between –30° and –90°. This is established by looking at lead II, which is perpendicular to –30°. If the QRS complex is positive in lead II, the axis is physiologically leftward; if the QRS complex is negative in lead II, the axis is pathologically leftward.
Since the QRS complex is generated by the activation of the ventricular myocardium, abnormalities of intraventricular conduction affect the QRS complex (either axis or width). There are several types of intraventricular conduction abnormalities.

**Fascicular Block**

Fascicular block, which is due to impulse conduction block through one of the major fascicles of the left bundle (ie, left anterior or left posterior fascicle), causes an axis shift in the frontal plane (from normal to either the extreme left or right) (FIGURE 1, previous page). The QRS complex duration remains normal as intraventricular conduction is still via the His-Purkinje system. The normal activation sequence through the left ventricle without a fascicular block is associated with a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). An axis between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF) is a physiologic left axis and is not a conduction abnormality; that is, it is not a left anterior fascicular block. A physiologic left axis may be seen with left ventricular hypertrophy or in normals.

**Left Anterior Fascicular Block**

With left anterior fascicular block, left ventricular activation is via the left posterior fascicle (FIGURE 2). Ventricular innervation originating from this fascicle results in an impulse that is directed superiorly and to the left. Hence a left anterior fascicular block is associated with an extreme or pathologic left axis that is between −30° and −90°. This produces a positive QRS complex in lead I and a negative QRS complex in leads II and aVF. The QRS complexes in leads II and aVF have an rs morphology as ventricular activation remains normal. In contrast, an inferior wall myocardial infarction (MI), which may also result in a negative QRS complex in leads II and aVF, has a Qr morphology (FIGURE 3). The Q wave indicates that the initial electrical forces are directed away from the lead that is over this part of the myocardium, meaning that the tissue under the lead is infarcted. Hence it is important to distinguish between a left anterior fascicular block, which represents a conduction abnormality, and an

**Figure 2. Left anterior fascicular block.**
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Infarction

Qr morphology indicates an infarction pattern
—When present in leads II, III, and aVF, represents inferior wall myocardial infarction
—When present in leads I and aVL, represents lateral wall myocardial infarction

Conduction Abnormality

rS morphology indicates a conduction abnormality
—When present in leads II, III, and aVF, represents left anterior fascicular block
—When present in lead I (and aVL), represents left posterior fascicular block

Figure 3. Infarction versus conduction abnormality.

In contrast, a lateral wall MI, which is not a conduction problem. Indeed, a left anterior fascicular block cannot be diagnosed in the presence of an inferior wall infarction pattern.

Left Posterior Fascicular Block

With a left posterior fascicular block, left ventricular activation is via the left anterior fascicle and the direction of impulse transmission is inferiorty and to the right (FIGURE 4). Hence a left posterior fascicular block results in a right axis (between +90° and +180°). There is a negative QRS complex in lead I and a positive QRS complex in leads II and aVF. The QRS complex has an rS morphology in lead I. In contrast, a lateral wall MI, which is not a conduction abnormality, will also be associated with a right axis (FIGURE 3). However, in this situation the QRS complex has a Qr morphology as the infarction results in the initial activation going away from the lateral wall and hence from left to right. There are several other causes for a right axis in addition to a lateral wall MI, and these must be considered before establishing the right axis as a left posterior fascicular block, which is a diagnosis of exclusion. Other causes for a right axis include:

• Right ventricular hypertrophy, which is diagnosed by the presence of a tall R wave in lead V1 and P pulmonale (right atrial hypertrophy or abnormality)
• Right-left arm lead switch, which is associated with negative P and T waves in leads I and aVL and positive P and T waves and a positive QRS complex in lead aVR

Figure 4. Left posterior fascicular block.
- **Dextrocardia**, in which there is a pattern that resembles right–left arm lead switch *(ie, an inverted P wave in leads I and aVL, a positive QRS complex, and positive P and T waves in lead aVR)*, as well as reverse R-wave progression across the precordium
- **Wolff-Parkinson-White syndrome**, with negative delta waves in leads I and aVL that are indicative of a left lateral pathway
- A biventricular pacemaker, in which the initial waveform of the QRS complex in lead I is a Q wave or QS pattern. However, most often a biventricular pacemaker is associated with an indeterminate axis.

A broad and deep terminal S wave in a right bundle branch block (RBBB) may give the appearance of a right axis. However, in this case the terminal S wave represents delayed right ventricular activation and not a left ventricular force. In this situation the terminal S wave should be ignored and is not considered for axis determination.

**Indeterminate Axis**

An indeterminate axis with a supraventricular complex is defined as an axis that is between −90° and +180°. The QRS complex is negative in leads I and aVF. This may represent either a very extreme left axis or a very extreme right axis. In a human adult heart, it is unusual to see an indeterminate axis with a supraventricular QRS complex. There is no type of conduction pattern through the normal His-Purkinje system that will result in an indeterminate axis. Thus an indeterminate axis is the result of the simultaneous presence of two different abnormalities. For example, an indeterminate axis may be seen when there is right ventricular hypertrophy (which shifts the axis rightward) associated with a left anterior fascicular block or an inferior wall MI (which shifts the axis leftward). An indeterminate axis may appear to be present if an old lateral and inferior wall MI is evident. However, in this case there are Q waves in leads I, II, III, and aVF and hence the axis shift is the result of an infarction. The presence of a lateral wall MI (which has a right axis) with a left anterior fascicular block (which has a left axis) or an inferior wall MI (which has a left axis) with a left posterior fascicular block (which has a right axis) will also have an indeterminate axis. However, in this case the negative QRS complex in either lead I (with a lateral wall MI) or lead aVF (inferior wall MI) is due to an infarction and not an axis shift. A right–left arm lead switch (which has a right axis) associated with either an inferior wall MI or a left anterior fascicular block (which has a left axis) will have an indeterminate axis. Lastly, the presence of a deep S wave due to an RBBB may give the appearance of a negative QRS complex in lead I, and the presence of a left anterior fascicular block will give the appearance of an indeterminate axis. In the presence of an RBBB the deep S wave in lead I is the result of terminal delay in right ventricular activation and is not considered as part of axis determination (which is the direction of impulse conduction within the left ventricle).

An indeterminate axis associated with a wide QRS complex may be seen with any situation in which there is direct ventricular myocardial activation, including a ventricular complex, a paced complex (especially biventricular pacing), or a preexcitation pattern, specifically Wolff-Parkinson-White.

**Intraventricular Conduction Delay**

An IVCD is a nonspecific QRS widening (QRS duration > 0.10 sec) without any specific bundle branch block pattern. Conduction is
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still via the entire His-Purkinje system, but it is slower than normal, accounting for the QRS widening. When the QRS complex is longer than 0.10 second but shorter than 0.12 second and has an RBBB pattern (ie, an RSR' complex in lead V1), it is often called an incomplete RBBB. When the QRS complex is longer than 0.10 second but shorter than 0.12 second with a left bundle branch block (LBBB) pattern, it is often called incomplete LBBB. However, the term incomplete bundle branch block is not accurate because conduction through the His-Purkinje system and bundle is all or none and will not be incomplete. Hence a more appropriate term is an IVCD to either the right or left ventricle. As the activation of the ventricles is via the normal His-Purkinje system, abnormalities of the right or left ventricular myocardium can be diagnosed. This is in contrast to an LBBB or RBBB in which ventricular activation is not via the normal conduction system, but rather is via an abnormal pathway with direct myocardial activation. Hence abnormalities of the ventricular myocardium served by the particular bundle cannot be diagnosed.

A QRS complex duration exceeding 0.12 second is also termed an IVCD if there is no specific bundle branch block pattern present. IVCDs with QRS complexes that are very wide (> 0.18 sec) without a bundle branch block pattern may be seen with severe dilated cardiomyopathy or hyperkalemia. The only etiology for a QRS complex duration exceeding 0.24 second is hyperkalemia, which is a medical emergency that requires immediate therapy. In the absence of hyperkalemia, a bundle branch block, a ventricular complex, or a severe cardiomyopathy is not associated with a QRS complex that is this wide.

Bundle Branch Blocks

There may be failure of impulse conduction (complete block) through either the right or left bundle branch, resulting in delayed activation of the ventricular chamber served by that bundle.

Right Bundle Branch Block

An RBBB has the following characteristic morphology (Figure 5):

- The QRS complex duration is 0.12 second or longer due to delayed and slow activation of the right ventricle.

Figure 5. Complete right bundle branch block.
• Right ventricular activation is from the left bundle and left ventricle, directly through the myocardium.

• The terminal forces of the QRS complex, representing delayed right ventricular activation and accounting for the widened QRS complex duration, are directed from left to right. Therefore, there is a second positive deflection in leads V1-V2 (i.e., an RSR', RR', or only a broad R complex) and a broad terminal negative deflection or S wave in leads I and V5-V6.

• Right ventricular repolarization is abnormal and hence there may be secondary ST-T wave changes seen in leads V1-V3.

Since right ventricular activation is abnormal, right ventricular hypertrophy cannot be reliably recognized. However, left ventricular activation is normal and so the initial portion of the QRS complex or R wave, which represents left ventricular activation, is normal. Therefore, left ventricular abnormalities (e.g., left ventricular hypertrophy, myocardial ischemia or infarction, pericarditis) can be recognized in the presence of an RBBB.

A left anterior fascicular block (extreme left axis) or left posterior fascicular block (right axis) may also be present. This is termed bifascicular block. However, it must be remembered that with an RBBB there is a broad terminal S wave in lead I that may give the appearance of a negative QRS complex in lead I and hence a right axis. The terminal S wave, which represents delayed right ventricular activation, should not be considered when determining the axis in the frontal plane as this is based on left ventricular activation.

**Figure 6. Complete left bundle branch block.**

An RBBB may be intermittent or rate-related, termed a functional bundle branch block. A rate-related RBBB may occur at any heart rate. Not uncommonly it resolves at a heart rate that is slower than the rate at which it develops. With a rate-related RBBB, the widening of the QRS complex occurs abruptly and resolves abruptly; it is not gradual. A QRS complex that has an RBBB morphology but a duration that is between 0.10 and 0.12 second has been called an incomplete RBBB but is actually an IVCD with conduction delay to the right ventricle.
Left Bundle Branch Block

An LBBB has the following characteristic morphology (FIGURE 6):

- The QRS complex duration is 0.12 second or longer due to delayed and slow activation of the left ventricle.
- Left ventricular activation is entirely from the right bundle and right ventricle and travels directly through the myocardium (hence prolonging the QRS complex duration).
- All ventricular forces are directed from right to left. Hence there is a broad, tall R wave in leads I, aVL, and V5-V6 (although a deep QS complex may be seen in leads V5-V6) and deep QS complexes in lead V1 and occasionally in lead V2. There should not be any ventricular forces directed from left to right. If there is an LBBB pattern present, but there are terminal S waves in leads I and V6, the etiology for the widened QRS complex is an IVCD.
- As an LBBB also involves the septal branch of the left bundle, there are no septal forces present; that is, small septal Q waves in leads I, aVL, and V5-V6 are not seen and there is no septal R wave in lead V1. If there is an LBBB pattern present in addition to septal forces, the etiology is an IVCD.
- Abnormal ventricular repolarization is associated with an LBBB, and hence there are typically diffuse secondary ST-T wave abnormalities.
- The QRS axis in the frontal plane may be normal or leftward. A left axis may represent abnormalities in the direction of impulse conduction through the left ventricular myocardium (eg, due to the presence of fibrosis) and is not a left anterior fascicular block as both fascicles are blocked. A right axis should not be seen with an LBBB as all forces are directed from right to left.

Since left ventricular activation is abnormal (ie, bypassing the normal His-Purkinje system), abnormalities affecting the left ventricular myocardium (eg, left ventricular hypertrophy, myocardial ischemia and chronic infarction, pericarditis) cannot be recognized. However, an acute MI can be established with an LBBB (or any other situation in which there is direct myocardial activation, such as ventricular pacing, a ventricular complex, and likely a preexcited complex in Wolff-Parkinson-White pattern) using the Sgarbossa criteria:

- ST-segment elevation of 1 or 2 mm or more that is in the same direction (concordant) as the QRS complex in any lead
- ST-segment depression of 1 mm or more in any lead from V1 to V3
- ST-segment elevation of 5 mm or more that is discordant with the QRS complex (ie, associated with a QS or rS complex)

An LBBB may be intermittent or rate-related (functional bundle branch block). As with an intermittent RBBB, a rate-related LBBB may occur at any heart rate. Not uncommonly, it resolves at a heart rate that is slower than the rate at which it develops. With a
rate-related LBBB, the widening of the QRS complex occurs abruptly and resolves abruptly; the widening is not gradual. When the QRS complex has an LBBB morphology but a duration of 0.10 to 0.12 second, it is often referred to as an incomplete LBBB. However, it is actually an IVCD. In this situation, left ventricular abnormalities and a left axis (left anterior fascicular block) or right axis (left posterior fascicular block) can be diagnosed because conduction is still via the normal His-Purkinje system, albeit slowed. As indicated, with an LBBB the normal His-Purkinje system is circumvented as left ventricular activation occurs via direct myocardial activation.

**Trifascicular Block**

Trifascicular block, or trifascicular disease, indicates conduction disease affecting both the right and left bundles. Because all three fascicles are affected, diagnosis of trifascicular disease requires evidence of conduction abnormalities in the right bundle, left anterior fascicle, and left posterior fascicle. This includes the following scenarios:

- Alternating RBBB and LBBB, which is also termed bi-bundle branch block
- RBBB with alternating left anterior and left posterior fascicular block
- Bifascicular disease (LBBB or RBBB with either a left anterior or left posterior fascicular block) with Mobitz type II second-degree AV block (with or without an associated first-degree AV block or prolonged AV conduction), which indicates disease of the His-Purkinje system

- Bifascicular disease with the development of complete heart block with an escape ventricular rhythm, which indicates disease of the His-Purkinje system

Trifascicular disease is not established by the finding of bifascicular disease (LBBB or RBBB with either a left anterior or left posterior fascicular block) and first-degree AV block (prolonged AV conduction) or the presence of second-degree AV block with 2:1 AV conduction. In

![Diagram of QRS axis in horizontal plane](image)

**Figure 7.** The QRS axis in the horizontal plane is determined by analyzing the direction of the QRS complex in the precordial leads. This is established by imagining the heart as viewed from under the diaphragm. With clockwise rotation, the left ventricular forces are seen later in the precordial leads. This presents with poor R-wave progression and late transition. With counterclockwise rotation, left ventricular forces develop earlier in the precordial leads. This presents with a tall R wave in lead V2, which is termed early transition.
these situations the AV conduction abnormality may be within either the AV node or the His-Purkinje system (in which case trifascicular disease cannot be definitively diagnosed). Trifascicular disease can only be established if there is evidence of a conduction abnormality within the remaining fascicle.

**Axis in the Horizontal Plane**

In addition to the axis in the frontal plane (ie, normal, right, left, indeterminate), there is also an axis in the horizontal plane of the heart. However, abnormalities in the axis of the horizontal plane are not the result of a conduction abnormality. The axis in the horizontal plane is established by imagining the heart as viewed from under the diaphragm (FIGURE 7). In this situation, the right ventricle is in front and the left ventricle is to the left. When there is an electrical rotation in a clockwise direction, right ventricular forces are still present in the lateral precordial leads, while the development of left ventricular forces is delayed. In this situation there is poor R-wave progression from leads V1-V3 or V4 and late transition (ie, the R wave does not develop an amplitude greater than the S wave until lead V5-V6) compared with the normal transition between leads V3-V4. The poor R-wave progression should not be confused with an anterior wall MI. When there is counterclockwise rotation, the left ventricular forces develop earlier and there is a tall R wave in lead V2 (ie, early transition). The tall R wave in lead V2 should not be confused with a posterior wall MI or right ventricular hypertrophy, in which there is a tall R wave in lead V1.

**Enhanced Atrioventricular Conduction**

In addition to slowing or block of AV conduction, AV conduction may be fast, due either to enhanced (accelerated) conduction through the AV node or to an accessory pathway (bypass tract) that bypasses the AV node (which is the site of slowest impulse conduction in the heart), serving as a second pathway for AV conduction from the atria to the ventricles. With enhanced AV conduction, the ECG shows a short (< 0.14 sec) PR interval at a normal rate (ie, < 100 bpm). It should be remembered that a short PR interval may also be seen with sinus tachycardia due to enhanced conduction through the AV node resulting from sympathetic stimulation.

There are two patterns for enhanced AV conduction due to a bypass tract. These patterns are also called preexcitation.

**Wolff-Parkinson-White Pattern**

Wolff-Parkinson-White pattern is due to an AV nodal bypass tract called the bundle of Kent, which is a direct connection between the atrial and ventricular myocardium, resulting in early ventricular myocardial activation that precedes activation via the AV node (ie, preexcitation). The QRS complex is widened as a result of the early, direct, and slow myocardial activation via the accessory pathway, which bypasses the normal AV node–His-Purkinje system. Thereafter there is normal ventricular activation via the AV node–His-Purkinje system. The QRS complex is, therefore, a fusion beat representing early but slow ventricular activation initiated via the accessory pathway and normal ventricular activation via the AV node–His-Purkinje.
system. As a result, the initial upstroke of the QRS complex is slowed due to direct myocardial activation. This is termed a delta wave, which produces a widening of the initial portion of the QRS complex, while the remainder of the QRS complex, due to activation via the His-Purkinje system, is narrow. The degree of aberration (delta wave width) is related to the balance between conduction through the AV node and accessory pathway, which is determined by AV nodal conduction because conduction through the accessory pathway is fixed (ie, it is all or none as it is Purkinje-like tissue). Hence if AV nodal conduction is slow, more of the initiation of ventricular activation is via the accessory pathway, resulting in a shorter PR interval and wider delta wave. If AV nodal conduction is fast, then less ventricular myocardial activation is via the accessory pathway and the PR interval is longer and the delta wave narrower. As the QRS complex in Wolff-Parkinson-White pattern is a fusion complex, the degree of fusion may vary from one period of time to another, as changes in AV nodal conduction may occur in a variable and unpredictable fashion, in part related to changes in autonomic inputs into the AV node. Hence there may be variability in the PR interval and width of the QRS complex (ie, prominence of the delta wave). This is termed the concertina effect.

Lown-Ganong-Levine Pattern

Lown-Ganong-Levine pattern results from conduction via a bypass tract known as the bundle of James, which is a connection between the atrium and the bundle of His. Since the AV node is bypassed, the PR interval is short. However, ventricular activation is via the normal His-Purkinje system and, therefore, the QRS complex (morphology and duration) is normal.
Core ECGs
A 76-year-old man with a history of hypertension presents for routine evaluation. On review, he admits to fatigue and a mild reduction in exercise capacity over the past year, but he is not functionally limited. He denies other symptomatology. On exam, he has a bradycardic but regular radial pulse. His carotid pulses are brisk and he has a soft S1, but S2 is normal. There is no S3 or S4. A soft, nonradiating early systolic murmur is heard best at the right upper sternal border. An ECG is obtained.

What does the ECG show?
ECG 1 Analysis: Sinus bradycardia, first-degree AV block (prolonged AV conduction or AV conduction delay), intraventricular conduction delay
There is regular rhythm at a rate of 50 bpm. There is a P wave (*) before each QRS complex, with a stable but prolonged PR interval (0.60 sec) (+). The P wave is positive in leads I, II, aVF, and V4-V6. Although the PR interval is long, it is constant and hence AV conduction is intact. Thus this is sinus bradycardia with first-degree AV block (or prolonged AV conduction).

The QRS complex duration is increased (0.12 sec). Although the morphology resembles that of a left bundle branch block (LBBB), there is a septal Q wave (†) in lead aVL (due to left-to-right conduction across the septal myocardium) that cannot be present with an LBBB because the septal branch, which activates the septum, originates from the left bundle. In addition, there is a terminal S wave in leads V5-V6 (—), indicating left-to-right forces, which are also not seen with an LBBB (as all of the forces are directed right to left). Therefore, this is an intraventricular conduction delay (IVCD). Importantly, with an IVCD the impulse is conducted through the normal His-Purkinje system but conduction is slower than normal. Hence there is a normal activation sequence of the left ventricular myocardium and abnormalities of the left ventricle can be diagnosed. With an LBBB there is no conduction through the left bundle and hence activation of the left ventricular myocardium bypasses the normal conduction system and is via an alternative pathway (ie, directly through the ventricular myocardium). Since the LV activation sequence is abnormal, abnormalities of the left ventricular myocardium cannot be reliably diagnosed. On this patient's ECG, the QRS axis is physiologically leftward, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (480/440 msec and 440/400 msec when the prolonged QRS complex duration is considered).

The normal PR interval, measured from the onset of the P wave to the onset of QRS complex (either a Q wave or an R wave), ranges from 0.14 to 0.20 second and represents AV conduction (ie, the time for the impulse to be conducted from the atrium to the ventricle). The

*continues*
PR interval can be further divided into the P wave and the PR segment. The P wave includes conduction through the right and left atria. Conduction through these structures is via three separate bundles: Bachman’s bundle courses along the posterosuperior aspect of the atria and conducts the impulse from the right to the left atrium, and two additional bundles (bundle of Thorel and bundle of Wenckebach) conduct the impulse from the sinus node to the AV node. The isoelectric PR segment represents conduction through the AV node and His-Purkinje system. As these are small structures, they do not generate enough electrical activity to be measured on the surface of the body; hence the PR segment is at baseline (i.e., it is isoelectric).

A PR interval longer than 0.20 second defines first-degree AV block (or prolonged AV conduction), which may represent slowing of conduction anywhere along the conduction pathway from the AV node to the terminal portion of the Purkinje fibers. In the healthy heart, the most frequent site of conduction delay is the AV node, which is the part of the conduction system that manifests the slowest rate of conduction. As AV nodal conduction is affected by autonomic balance, the PR interval changes with heart rate. Sinus tachycardia, which is the result of enhanced sympathetic activity that increases AV nodal conduction velocity, is associated with a shortening of the PR interval while sinus bradycardia, which is the result of sympathetic withdrawal and an increase in vagal tone, is associated with a slowing of AV nodal conduction velocity and hence an increase in the PR interval.
This ECG, coupled with the description of a septuagenarian with physical exam findings consistent with aortic valve disease (sclerosis), may suggest Lev’s disease (senile conduction system degeneration) as the etiology of a prolonged PR interval and IVCD. Idiopathic slowing of AV conduction in the elderly has been termed Lev’s disease, attributed to the extension of mitral or aortic valve calcification into fibers of the conduction system. Idiopathic slowing of AV conduction in younger individuals has been termed Lenègre’s disease, attributed to progressive sclerofibrotic degeneration of the conduction system that may be hereditary. Some cases that may be hereditary are autosomal dominant and associated with mutations in the cardiac sodium channel SCNSA.
A 58-year-old man is admitted to the hospital with an anterior wall myocardial infarction (MI), and an ECG is obtained (2A). He is treated...
with thrombolysis without complication. Several days later, he complains of palpitations and feels diaphoretic. Another ECG (2B) is obtained.

**What do the ECGs show?**

ECG 2B
Podrid's Real-World ECGs

ECG 2A Analysis: Sinus tachycardia, first-degree AV block, intraventricular conduction delay (IVCD) to the right ventricle, left anterior fascicular block, anterior wall MI
In ECG 2A there is a regular rhythm at a rate of 100 bpm; hence this is tachycardia. The QRS interval is prolonged (0.16 sec). The morphology is not typical for either a left or a right bundle branch block. There is an RSR' morphology in lead V1 (+-) but no broad terminal S wave in lead I. In addition, there is a QS morphology from leads V2 to V6, which suggests a left bundle branch block. Hence this is an intraventricular conduction delay (IVCD). The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). An extreme left axis may be seen with an inferior wall myocardial infarction (MI) in which there are deep initial Q waves in leads II and aVF. In contrast, the QRS complexes in leads II and aVF have an rS morphology. This is characteristic of a left anterior fascicular block.

There are Q waves (↑) in leads V2-V6, diagnostic for an anterior wall MI. The QT/QTc intervals are prolonged (400/520 msec) but are normal when corrected for the prolonged QRS complex duration (320/410 msec). Although the P waves are not obvious, a suggestion of P waves can be seen after the QRS complexes, particularly in leads V1-V2 (*). The RP interval is short (0.20 sec, ⌂) and shorter than the PR interval (0.46 sec, ⌂). Therefore, this is termed short RP tachycardia. There are a number of etiologies for short RP tachycardia, including sinus tachycardia with first-degree AV block (prolonged AV conduction), atrial tachycardia, junctional tachycardia (with a retrograde P wave), atrial flutter with 2:1 AV block, AV reentrant tachycardia (associated with a preexcitation syndrome) or typical AV nodal reentrant tachycardia (AVNRT). Typical AVNRT does not usually manifest any P wave before or after the QRS complex. This is because the mechanism is a slow pathway to the ventricles and a fast pathway back to the atria, with simultaneous activation of the atria and ventricles. A variant of this is termed slow-slow (ie, the fast pathway conducting retrogradely back to the atria conducts relatively slowly).
ECG 2B Analysis: Normal sinus rhythm, first-degree AV block, LVCD to the right ventricle, left anterior fascicular block, anterior wall MI, premature ventricular complex
In ECG 2B, the rhythm is regular at a rate of 86 bpm. The QRS complex morphology, duration, and axis are the same as those in ECG 2A. The QT/QTc intervals are also the same. Although the P waves are not obvious, there are waveforms (*) seen at the end of the QRS complex (particularly in leads V1-V2), within the ST segment, that suggest a superimposed P wave. If these were superimposed P waves, the PR interval would be prolonged (0.54 sec) (+). One premature complex can also be seen (*). It is wider than the other QRS complexes and has a different morphology. Hence it is a premature ventricular complex (PVC), and it aids in our assessment of the PR interval. After the PVC there is a pause (LJ). The P wave can be seen during the pause (+), and the measured PR interval is indeed 0.54 second (+). Hence there is first-degree AV block (prolonged AV conduction). Using this PR interval, it can be seen that the ST-segment abnormality in both ECGs is in fact the P wave. In ECG 2A, the PR interval is slightly shorter (0.46 sec), perhaps due to the faster sinus rate. Thus the short RP tachycardia in ECG 2A is actually sinus tachycardia as the P waves are positive in leads I, II, aVF, and V4-V6.

The His bundle and the proximal bundle branches are generally resistant to ischemia given dual blood supply from both the AV nodal artery and the proximal septal perforator branches of the left anterior descending artery in most patients. However, in some patients, blood supply to the proximal bundle branches is not collateralized. In addition, infarction of the intraventricular septum can damage parts of the bundles, resulting in conduction abnormalities. In such cases, proximal left anterior descending artery occlusion results in ischemic injury and infarction of the septum and the conduction system, likely explaining the presence of a left anterior fascicular block associated with an anterior wall MI. The etiology of the prolonged PR interval is not clear; it may be due to slow conduction through the AV node or diffuse slowing of conduction through the His-Purkinje system, suggested by the presence of the IVCD as well as the anterior wall MI.
A 24-year-old man presents to the emergency department with a complaint of palpitations that had occurred spontaneously while he was watching TV. The episode lasted for about 2 hours but terminated abruptly as he arrived at the hospital. He stated that he had
experienced occasional palpitations in the past, but they usually lasted less than 30 minutes and seemed to resolve with coughing. ECG 3A is the initial ECG. About 2 minutes, later, without any intervention, a second ECG (ECG 3B) was obtained.

What is the difference between the two ECGs?
What abnormality is suggested when both ECGs are considered?
What is the likely cause of the palpitations?
ECG 3A Analysis: Sinus tachycardia, left atrial hypertrophy (abnormality), early transition
In ECG 3A, there is a regular rhythm at a rate of 110 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is broad and notched, particularly in leads II, aVF, and V3-V6, consistent with left atrial hypertrophy (abnormality). The QRS complex duration is normal (0.08 sec), and there is a normal morphology, except for a tall R wave in lead V2, which is termed early transition or counterclockwise rotation. This is an axis shift in the horizontal plane. The axis is determined by imagining the heart as viewed from under the diaphragm; the right ventricle is in front and the left ventricle is lateral. With counterclockwise rotation the left ventricle is electrically shifted anteriorly and hence left ventricular forces occur early in the right precordial leads, producing a tall R wave in lead V2. The axis in the frontal plane is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (300/410 msec).
ECG 3B Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), dual AV nodal pathways, left atrial hypertrophy (abnormality), early transition
ECG 3B was obtained 2 minutes after ECG 3A without any intervention. There is a regular rhythm at a rate of 122 bpm. There is a P wave (+) before each QRS complex. The P wave might be mistaken for the T wave, especially in the limb leads, but the P wave is clearly distinct in leads aVL and V1. Also, this waveform is much narrower than a normal T wave and has a sharp upstroke and downstroke. The P wave in ECG 3B has the same axis and morphology as the P wave in ECG 3A. It is positive in leads I, II, aVF, and V4-V6 and there is a left atrial abnormality (hypertrophy). Although there is a stable PR interval, it is longer (0.34 sec) (+) than what was seen in ECG 3A. Hence this is sinus tachycardia with first-degree AV block (prolonged AV conduction). The QRS complex duration, morphology, and axis are the same as in ECG 3A, as are the QT/QTc intervals.

Although ECG 3A shows a normal PR interval, the PR interval in ECG 3B is much longer, even though the sinus rate is also faster. The PR interval usually shortens when the sinus rate increases as a result of faster AV conduction due to sympathetic stimulation. However, in this case the PR interval is longer at a faster rate and shorter at a slower rate. The PR interval lengthening occurred abruptly in the absence of any intervention. The only situation during sinus rhythm in which this phenomenon can occur is with the presence of dual AV nodal pathways.

The presence of dual AV nodal pathways is the anatomic basis for atrioventricular nodal reentrant tachycardia (AVNRT). In order for AVNRT to occur, the following is required: One of the pathways conducts rapidly but has a long refractory period (ie, slow recovery). The second pathway conducts slowly but has a short refractory period (ie, fast recovery). These two pathways are linked proximally in the atrial myocardium and distally at the distal part of the AV junction. During sinus rhythm, AV conduction is via the fast pathway. However, if a premature atrial complex occurs before the fast pathway has recovered, then the impulse is blocked in the fast pathway (unidirectional block) but is conducted to the ventricles via the slow pathway, which recovers more quickly. However, the PR interval will be much longer than the PR interval of the sinus complex. If the fast pathway has recovered by the time the impulse reaches the distal end of the circuit, the impulse can be conducted retrogradely by this fast pathway to activate the atrium in a retrograde direction at the same time the impulse travels antegradely to activate the ventricles. If the slow pathway has recovered by the time the impulse reaches the proximal part of the circuit, the impulse can reenter this pathway antegradely and then again conduct retrogradely through the fast pathway. This establishes a reentrant arrhythmia, known as AVNRT. This is termed slow-fast AVNRT, and in this situation no P wave is seen as there is simultaneous atrial and ventricular activation.

As the reentrant circuit is within the AV node, AVNRT can often be terminated by any intervention that alters the conduction characteristics of the AV nodal pathways. This includes enhancement of vagal tone, as with Valsalva, carotid sinus pressure, or coughing, which slows conduction primarily through the slow AV nodal pathway. The history from this patient is consistent with this arrhythmia, especially since there is evidence of dual AV nodal pathways. Adenosine, a calcium-channel blocker, or a β-blocker also alter AV nodal conduction and can be used to terminate this arrhythmia.
A 60-year-old woman with a prior inferior wall myocardial infarction (MI) is noted on routine exam to have an irregular pulse. She is otherwise asymptomatic. Her medications include a β-blocker, aspirin, and an angiotensin-converting enzyme inhibitor. An ECG is obtained.

**What abnormalities are shown?**

**What is the cause of her irregular pulse?**
ECG 4 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type I second-degree AV block (Wenckebach), right bundle branch block, counterclockwise rotation, inferior wall MI
The rhythm is irregular, although there is a repeating pattern of long (U) and short (n) RR intervals. Therefore, the rhythm is regularly irregular at an average rate of 66 bpm. There are P waves (*) seen with a constant PP interval and a sinus rate of 84 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complexes are wide (0.14 sec) with an RSR' morphology in lead V1 (+) and a broad terminal S wave in lead I (+). This is the pattern of a right bundle branch block. There is a tall R wave (R > S) in lead V2 (+), which is early transition, the result of counterclockwise rotation. This is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, left ventricular forces are seen early in the precordial leads. There are Q waves in leads III and aVF (+), consistent with a prior inferior wall myocardial infarction (MI). The QT/QTc intervals are normal (440/420 msec).

The ventricular rate is irregular, but there is a pattern to the irregularity. All of the short intervals are the same (n) and the long intervals (U), or pauses, are also the same. Therefore, the rhythm is regularly irregular and there is a pattern of grouped beating with three QRS complexes followed by a pause. The pause is the result of a nonconducted P wave (+). Hence there is second-degree AV block. The PR interval after each pause (+) is the same (0.40 sec), and hence there is first-degree AV block (prolonged AV conduction). This represents the baseline PR interval. The PR intervals that follow (U) become progressively longer (0.48 and 0.56 sec). The third QRS complex of the group is followed by a nonconducted P wave (+), after which the PR interval shortens to its baseline (0.40 sec). This recurrent abnormality in AV conduction causes the appearance of “grouping” of the QRS complexes (so-called “grouped beating”); there is a pattern of 4:3 conduction (ie, four atrial beats for every three ventricular beats). This pattern is classified as second-degree AV block, Mobitz type I (Wenckebach). Second-degree AV block is identified by a pause in the RR interval due to a nonconducted P wave. With Wenckebach there is only one nonconducted P wave. Any pattern of block (eg, 2:1, 3:2, 4:3, etc) may occur depending on the AV nodal electrophysiologic properties and the degree of vagal tone. As a result the rhythm will be regularly irregular.

Second-degree AV block is subdivided into two types: Mobitz type I (or Wenckebach) and Mobitz type II. Mobitz type I is characterized by a progressive lengthening of the PR interval until a normally timed P wave is not followed by a QRS complex (ie, it is nonconducted). With PR interval prolongation, each successive atrial impulse reaches the AV node earlier, while it is still partially refractory. There is a progressive

continues
slowing of AV conduction of each subsequent impulse ("decremental" AV nodal conduction). Finally, the AV node fails to conduct completely if a sinus impulse arrives when it is absolutely refractory; the result is a lack of ventricular activation and the absence of a QRS complex.

Wenckebach is a conduction abnormality within the AV node. Since the electrophysiologic properties of the AV node are mediated by calcium currents (slow action potential), conduction through this structure is not all or none but can vary depending on changes in nodal electrophysiologic properties. Non-pathologic changes in the speed of AV conduction are usually due to changes in the node's refractory period. Entities that affect the refractory period include increases in parasympathetic vagal tone such as that seen in the pediatric population, during sleep, with gastrointestinal distress such as vomiting, and in well-conditioned people. An increase in the refractory period can be seen with an acute inferior wall MI, which is frequently accompanied by increased vagal discharge. Some pathologic conditions, such as sclerodegenerative disease and myocarditis, can result in Mobitz type I block. Many medications can alter AV node conduction as well, including digoxin, lithium, calcium-channel blockers, and β-blockers. It is possible that a β-blocker is the cause of Wenckebach in this patient. Wenckebach is predominantly a benign conduction abnormality that, if asymptomatic, does not require any therapy.
A 55-year-old woman presents with several hours of nausea culminating in an episode of vomiting and chest pain that prompts activation of emergency medical services. On arrival to the emergency department, she appears pale, diaphoretic, and in moderate distress. Her vital signs are notable for tachycardia. Her physical exam reveals faint pulmonary rales, normal S1 with a widely but physiologically split S2, and an S3. No murmurs are noted. An ECG is performed.

What abnormalities are noted on the ECG, and what is the diagnosis?

What is the etiology of the conduction abnormalities?
ECG 5 Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), Mobitz type I second-degree AV block (Wenckebach), right bundle branch block, acute inferior wall myocardial infarction
The rhythm is regularly irregular as a result of two pauses that have the same duration (LI). P waves (**) can be seen during each pause, with a PR interval of 0.28 second after each pause (**-**). Although other P waves are not immediately obvious, closer inspection of leads I, II, V2, and V3 reveals deflections consistent with P waves (**). Importantly, two sequential P waves (**,**) can be seen in either lead I or II (surrounding the first QRS complex of the ECG), establishing the PP interval and an atrial rate of 122 bpm. The P wave is positive in leads I and II; hence this is likely sinus tachycardia.

Having established the PP interval, P waves with a constant PP interval can be seen in lead II beginning with the P wave after the pause at the beginning of the ECG. Over the course of the lead II rhythm strip, P waves (**), which may be initially confused for T waves, are moving into the QRS complex, ultimately not being obvious because they are buried within the QRS complexes just prior to the second pause. Therefore, the change in the morphology of the terminal portion of the QRS complex (or actually within the ST segment) over the course of the lead II rhythm strip is the result of fusion between the P wave and the QRS complex; this waveform is not the T wave. This observation suggests that the PR interval is lengthening (rather than the QT interval shortening). Hence this is second-degree AV block, Mobitz type I (Wenckebach), with a long cycle. The long cycle length is the result of underlying sinus tachycardia, which results from enhanced sympathetic tone.

The QRS complex duration is increased (0.12 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). There is a right bundle branch block morphology (RSR' in lead V1 [-]) and broad terminal S waves in leads I and V5-V6 [+--]). The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (320/460 msec) but are normal when corrected for the prolonged QRS complex duration (280/400 msec). There is ST-segment elevation (↑) in leads III and aVF, consistent with an acute inferior wall myocardial infarction (MI), and ST-T wave changes (!) in leads V3-V6 that may be reciprocal (associated with the inferior wall MI).
This patient is suffering from an acute inferior wall MI. The lack of a tall R wave in lead V1 or ST-T wave changes in leads V1-V3 argues against posterior left ventricular wall involvement. In terms of coronary anatomy, 80% to 85% of patients are “right dominant”; that is, the right coronary artery gives rise to the posterior descending and posterior left ventricular coronary arteries, which supply the inferior wall of the left ventricle as well as the inferior septum. About 10% of patients are “left dominant,” with the left circumflex artery giving rise to these arteries, and 5% to 10% are co-dominant (with the posterior descending artery arising from either the right coronary artery or left circumflex artery, and the posterior left ventricular artery arising from the other). As the AV nodal artery is usually a proximal branch from the right coronary artery, AV conduction delay (manifesting as first- and second-degree AV block) is not likely to represent AV nodal ischemia. Moreover, the AV node generates a slow action potential, mediated by a calcium current, which is not energy dependent and hence does not depend on oxygen supply. The bundle branches usually receive blood supply from the septal perforators of the left anterior descending artery. Therefore, right bundle branch block in this patient is not likely to be due to the acute MI. Transient AV nodal conduction delay may be seen with an inferior wall MI due to resultant elevation in vagal tone as well as edema that may occur around the AV node. Therefore, AV nodal abnormalities are likely to be transient.

Note that despite elevated vagal tone and the presence of Wenckebach, this patient is tachycardic. This may be due to the presence of heart failure, which is evidenced on the physical exam.
A 73-year-old woman presents for her annual evaluation. She does not have a significant cardiac history. Her exam is normal. A routine ECG is performed.

What abnormality is noted?
What further management (if anything) is needed?
Podrid's Real-World ECGs

ECG 6 Analysis: Normal sinus rhythm, Mobitz type II second-degree AV block
There is a regular rhythm at a rate of 70 bpm. There is a P wave (\(^*)\) before each QRS complex. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The PR interval (\(\rightarrow\)) is stable at 0.16 second. Noted is a pause that results from a single on-time but nonconducted sinus P wave (\(\star\)). An occasional nonconducted P wave is the hallmark of second-degree AV block. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/430 msec).

The PR intervals are stable and hence the etiology for the pause or second-degree AV block is Mobitz type II. Mobitz type II block is characterized by an episodic and unpredictable failure of AV conduction. Mobitz type II block is due to blocked electrical conduction below the level of the AV node, within the bundle of His or bundle branches. Since the His-Purkinje system conducts in an “all or none” fashion (it either conducts with the same conduction velocity each time or fails to conduct any impulse), there is no change in the PR interval before or after the nonconducted P wave, in contrast to what is seen in Mobitz type I block. Therefore, failure of conduction represents underlying structural disease of the infranodal His-Purkinje system. In contrast, Mobitz type I AV block is a conduction abnormality within the AV node and is generally an exaggeration of AV nodal decremental conduction; it typically does not represent underlying disease of the cardiac conduction system. In Mobitz type II block, there may be more than one successive nonconducted P wave, resulting in several P waves in a row without QRS complexes. This has often been termed “high-degree” AV block.

Both Mobitz type I and Mobitz type II block may progress to complete heart block. However, when Mobitz type II progresses to complete heart block, the escape rhythm is infra-Hisian (i.e., ventricular), since the His-Purkinje system is diseased. Not uncommonly, the heart rate with a ventricular escape rhythm is slow and may be inadequate for systemic perfusion. The result may be “Stokes-Adams attacks” (unheralded syncope) and possibly sudden death. Therefore, even asymptomatic Mobitz type II block is a class IIA American Heart Association and American College of Cardiology recommendation for permanent pacemaker implantation. Therefore, pacemaker implantation should be considered in this patient.

In contrast, asymptomatic Mobitz type I block is not an indication for a pacemaker. In this situation the development of complete heart block is most often associated with an escape junctional rhythm, which is generally stable. 

\(\Box\)
A 28-year-old man presents with a chief complaint of fatigue. He has no medical diagnoses and does not take medications. His family history is notable for his mother who has a pacemaker and his maternal uncles and grandfather who had heart failure of unclear etiology. He has two siblings in their 20s and 30s who have no medical issues. This patient’s ECG is presented.

What abnormality is depicted?
What is the cause of this abnormality?
What further workup or therapy is indicated?
ECG 7 Analysis: Normal sinus rhythm, Mobitz type II second-degree AV block, early repolarization
There are regularly occurring P waves (•,+\(\square\)) at a constant rate of 74 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is an underlying sinus rhythm. Although each QRS complex is preceded by a P wave (\(\square\)), there are a number of P waves without QRS complexes (+), resulting in frequent pauses in the ventricular rate (long RR intervals) as a result of on-time but nonconducted P waves (+). The PR interval of all the conducted beats is stable (\(\square\)) (0.18 sec). The occurrence of an occasional nonconducted P wave defines second-degree AV block. Since all of the PR intervals are constant, this is Mobitz type II with a variable conduction pattern of 3:2 and 2:1 block. The QRS complex duration is normal. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). Slight J-point and ST-segment elevation (+\(\square\)) is noted in leads V4-V6; this is consistent with early repolarization, which is frequently seen in young individuals. The QT/QTc intervals are normal (380/340 msec).

This patient’s fatigue is likely the result of his marked bradycardia from the frequent pauses. The rate is possibly slower at other times. His age and family history suggest a genetic cause for his conduction system disease. Some familial cardiomyopathies present initially with conduction system disease, including AV block. Permanent pacemaker implantation is warranted. As a familial cardiomyopathy is a very possible diagnosis, the patient will require close clinical surveillance as well as routine echocardiograms. In addition, ECG and echocardiographic surveillance should be considered for his siblings. Referral to a specialized center that performs genetic testing should be considered. ■
A 51-year-old man is admitted to the coronary care unit after suffering an acute non-ST-segment elevation myocardial infarction (NSTEMI). Coronary angiography documented 95% occlusion of the right coronary artery. He was treated with angioplasty and stenting, which was complicated by transient complete heart block requiring temporary transvenous pacing. On post-infarction day 2, the following ECG was obtained.

What abnormalities are depicted?
What is the appropriate management?
ECG 8 Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block, right bundle branch block, left anterior fascicular block, poor R-wave progression and late transition (clockwise rotation)
There is a regular rhythm with a ventricular rate of 50 bpm. A P wave (*) can be seen before each QRS complex, and the PR interval (++) is constant at 0.26 second (first-degree AV block or prolonged AV conduction). The P waves are positive in leads I, II, aVF, and V4-V6. The QRS complex duration is increased (0.16 sec), and there is a right bundle branch block pattern (RSR' morphology in lead V1 [→] and a broad S wave in leads I and V5-V6 [→]). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). As there are no Q waves in leads II and aVF, but rather an rS morphology, the extreme left axis is the result of a left anterior fascicular block. The QT/QTc intervals are prolonged (520/470 msec), but they are normal when the prolonged QRS complex duration is considered (440/400 msec).

A second P wave (+) is present in the T wave (best seen in leads V2-V3), although there are also irregularities seen on the T waves in leads II and V4-V6 (▼), which are the result of superimposed P waves. The PP intervals (▼▼) are regular. Hence the atrial rate is regular at 100 bpm. Therefore, there is sinus tachycardia with both first- and second-degree AV block with 2:1 AV conduction. The etiology (ie, Mobitz type I or II) cannot be established, even though other conduction system disease is present. While there is evidence of bifascicular disease (right bundle branch block and left anterior fascicular block), this does not necessarily establish Mobitz type II as the etiology. Moreover, the presence of first-degree AV block does not help establish the etiology; the prolonged PR interval may be due to slowing of conduction through the AV node or the remaining functioning fascicle (ie, left posterior fascicle). Importantly, this ECG does not show evidence of trifascicular block as the site of the first-degree AV block cannot be established even though there is evidence for bifascicular disease.

The QRS complexes in the precordium show poor R-wave progression and late transition. This is the result of a clockwise rotation of the electrical axis in the horizontal plane, determined by imagining the heart as viewed from under the diaphragm. With clockwise rotation, the left ventricular forces are seen late in the precordial leads.

As there is a right coronary artery lesion, the non-ST-segment elevation myocardial infarction involves the inferior wall. It is likely that the 2:1 AV block as well as the first-degree AV block are due to increased vagal tone or possibly edema of the AV node. Therefore, the AV conduction abnormalities are likely to be transient and will not require implantation of a permanent pacemaker. However, if this conduction abnormality persists and is symptomatic, permanent pacemaker

*continues*
insertion may be necessary. If no symptoms are present, it would be useful to establish the location of the block (i.e., AV nodal or infranodal) with an electrophysiologic study.

It is uncertain whether the bifascicular disease is preexistent or the result of the acute infarction. However, as the infarction involves the inferior wall, it is most likely that these conduction abnormalities were present before the acute event, since the right bundle receives its blood supply primarily from septal branches of the left anterior descending artery. Permanent pacemaker implantation in patients with evidence of bifascicular conduction block is not necessary.

Class I indications for pacemaker insertion after a myocardial infarction include:

- Third-degree AV block within or below the His-Purkinje system
- Persistent second-degree AV block in the His-Purkinje system with alternating bundle branch block
- Transient advanced second-degree infranodal AV block and associated bundle branch block (if the site of block is uncertain, an electrophysiologic study may be necessary.)
- Persistent and symptomatic second- or third-degree AV block
A 79-year-old woman presents with syncope. A neighbor answered her calls for help and found her lying on the floor of her apartment. Her medical history and current medications are unknown. She is bradycardic and hypotensive in the emergency department. On exam, she is quite somnolent but does move her extremities to vocal commands. Her lungs are clear and her cardiac exam is notable only for marked, regular bradycardia. An emergent ECG is obtained and you are asked to interpret it.

What is the diagnosis?
What treatment is indicated?
Podrid's Real-World ECGs

ECG 9 Analysis: Normal sinus rhythm, 2:1 second-degree AV block, ventriculophasic sinus arrhythmia
There is a regular rhythm at a rate of 34 bpm. The QRS complex duration is increased (0.12 sec), and it has a right bundle branch morphology with an RSR' morphology in lead V1 (−→) and a broad terminal S wave in leads I and V5-V6 (+→). The QRS axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (500/380 msec). There is a P wave (*) before each QRS complex with a stable PR interval (+-) of 0.16 second. A second, nonconducted P wave can be seen after each T wave (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm at a rate of 66 bpm. However, the PP interval is not completely regular and it can be seen that the PP interval surrounding the QRS complex (LJ) is slightly shorter (0.76 sec) than the PP interval when there is no intervening QRS complex (0.88 sec) (Π). This is termed ventriculophasic sinus arrhythmia and it can be seen whenever 2:1 or complete AV block is present.

The etiology for ventriculophasic arrhythmia with a shortening of the PP interval surrounding the QRS complex is not certain. It has been suggested that ventricular contraction causes an increase in pulsatile blood flow through the sinus nodal artery that enhances the automaticity of the sinus node. Ventricular contraction also causes a stretch of the right atrium and of the sinus node that enhances nodal automaticity. Lastly, the occurrence of ventricular contraction and stroke volume results in activation of the baroreceptors in the carotid artery that affects sinus node automaticity.

Although the etiology of the 2:1 AV block (ie, Mobitz type I or II) cannot be established, this patient is hemodynamically unstable as a result of marked bradycardia and hence temporary transvenous pacing should be performed. If the 2:1 AV block is the result of Mobitz type II second-degree heart block (as established by some change in the conduction pattern or with electrophysiologic study demonstrating a prolonged HV interval with the conducted complexes in addition to A and H waves but no V wave associated with the nonconducted P wave, indicating block below the AV node within the His-Purkinje system) or if the 2:1 AV block is permanent, a permanent pacemaker would be indicated. A permanent pacemaker would also be indicated for Mobitz type I if the 2:1 block was frequent and associated with symptoms. With electrophysiologic study, Mobitz type I block would be associated with an A wave not followed by an H or V wave, indicating block in the AV node. ■
A 72-year-old woman with a history of myocardial infarction (MI) is seen by her cardiologist for a routine follow-up. When a slight irregularity is noted during carotid artery palpation, an ECG is obtained.

What is the cause for the physical exam finding noted by the physician?
ECG 10 Analysis: Sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type I second-degree AV block (Wenckebach) with 2:1 and 3:2 AV conduction, old inferior wall MI
The rhythm is regularly irregular as a result of one shorter RR interval (between the first and second QRS complexes). P waves (*) can be seen before each QRS complex. The PR interval (++) is stable (0.26 sec); hence there is first-degree AV block (prolonged AV conduction). In addition, nonconducted P waves (+) can be seen after each QRS complex. The P waves are positive in leads I, II, aVF, and V4-V6. The PP interval is constant (L), and thus there is a normal sinus rhythm at a rate of 80 bpm. There is a pattern of 2:1 AV block with a stable ventricular rate of 40 bpm. However, it can be seen that the second QRS complex is early (▼) and is preceded by a P wave (▲). However, the PR interval (□) is longer (0.40 sec) than the baseline PR interval (0.26 sec) (++).

The on-time sinus P wave after the second QRS complex is nonconducted (†). This is a pattern of Mobitz type I second-degree AV block (Wenckebach) with 3:2 AV conduction. Thereafter, there is a pattern of second-degree AV block with 2:1 conduction. Since there initially was Mobitz type I, the 2:1 AV conduction is also Mobitz type I.

The QRS complex duration is normal (0.08 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). There are Q waves (▲) in the inferior leads II, III, and aVF, indicating an old inferior wall myocardial infarction (MI). The QT/QTc intervals are normal (520/424 msec).

As this patient is without symptoms, there is no reason for any therapy. The Wenckebach is the result of abnormalities in AV nodal conduction, possibly resulting from the old inferior wall MI.
An 18-year-old man with a history of an unspecified cardiomyopathy affecting only the men in his family presents to the emergency department with lightheadedness. He states that he has had on-and-off lightheadedness without frank syncope over the past month. Today, he noted its abrupt onset while having breakfast. The symptoms persisted long enough to cause concern. His review of systems is otherwise notable for an inability to keep up with his teammates on the track and field team over the past several months.
On exam, the man’s vital signs include a radial pulse of 40 bpm and a blood pressure of 122/84 mm Hg. His overall appearance is normal. His head, ears, nose, and throat exam and neck exam are normal. His lung fields are clear. Jugular venous pressure is 5 cm H2O. His cardiac exam displays a normal point of maximal impulse and normal cardiac sounds without a gallop or murmur. His abdominal, extremity, and neurologic exams are normal. An ECG is obtained (11A), and the man is admitted to the hospital. On the following day, a second ECG is obtained (11B).

What is the cause of the patient’s symptoms?
What further workup is warranted?
What therapy is indicated?
ECG 11A Analysis: Normal sinus rhythm, second-degree AV block with 2:1 AV conduction, Mobitz type II second-degree AV block
In ECG II A, the ventricular rate is regular at 38 bpm; however, the last two QRS complexes (↓) are at a rate of 76 bpm. The QRS complex duration is normal (0.08 sec) with a normal morphology and axis, between 0° and ±90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/350 msec). There are P waves (+/++) occurring at a regular interval at a rate of 76 bpm. The P waves are positive in leads I, II, aVF, and V4-V6; hence this is a normal sinus rhythm. There is a P wave (+) before each QRS complex. However, every other P wave is nonconducted (+) and there is a pattern of second-degree AV block with 2:1 AV conduction. The PR interval is stable at 0.20 second (+L). Hence this can be either Mobitz type I or II. The last two QRS complexes (↓), at a rate of 76 bpm, have P waves (▼) before them and show intact AV conduction with a stable PR interval of 0.20 second (++). Hence the 2:1 AV block is Mobitz type II as the two sequential complexes that are conducted have the same PR interval.

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ECG 118 Analysis: Normal sinus rhythm, Mobitz type II second-degree AV block
ECG 11B is obtained the following day. The QRS duration, morphology, and axis are the same as in ECG 11A. Noted are a stable rhythm at a rate of 70 bpm and a P wave (*) before each QRS complex. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The PR intervals are stable at 0.20 second (└┘). There is one on-time sinus P wave (+) that is nonconducted. Hence this is second-degree AV block. As the PR intervals are constant, this is Mobitz type II block and confirms the presence of Mobitz type II block on ECG 11A.

Conduction Abnormalities: Core Case 11

It is likely that this patient is manifesting pathology attributable to a familial, X-linked conduction system disease. Although the patient does not have signs of global left ventricular dysfunction, an echocardiogram is warranted to confirm the physical exam findings. Pacemaker implantation is warranted based on Mobitz type II block that is symptomatic.
A 48-year-old man with a recent anterior wall myocardial infarction treated with a stent presents for routine follow-up. He was placed on standard post-infarction medical therapy, and his recovery has been unremarkable. Echocardiography shows that left ventricular function has been preserved. He states that other than some mild fatigue, he feels well, can exercise without limitation, and is free of dyspnea and angina.
On review of systems, he notes symptoms consistent with erectile dysfunction since the event. His medical therapy includes dual antiplatelet therapy with aspirin and clopidogrel, a high-dose β-blocker, and a statin.

A routine ECG is obtained (12A). When the patient presents the following day to an urgent care clinic with complaints of fatigue and some lightheadedness, a second ECG is obtained (ECG 12B).

What abnormalities are notable, if any, on ECG 12A?

What abnormalities are notable on ECG 12B?

What is the likely cause?
ECG 12A Analysis: Normal sinus rhythm, first-degree AV block, 2:1 second-degree AV block
ECG 12A shows a regular rhythm at a rate of 48 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.32 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence there is a sinus rhythm with first-degree AV block (prolonged AV conduction). It is noted in lead V1 that there is a second P wave (+) that is nonconducted. The PP interval is constant (0.4 sec), with an atrial rate of 96 bpm, and there is a pattern of 2:1 AV conduction. Hence there is a normal sinus rhythm with second-degree AV block and a pattern of 2:1 AV conduction in addition to first-degree AV block. This may be Mobitz type I or II. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis, between 0° and 90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/435 msec).
ECG 12B Analysis: Normal sinus rhythm, complete heart block with a junctional escape rhythm
ECG 12B shows regular QRS complexes at a rate of 40 bpm. The QRS complexes have a morphology, axis, and QT/QTc intervals that resemble those in ECG 12A. The atrial rate is regular (L) at 86 bpm, and the P waves (•) are positive in leads I, II, aVF, and V4-V6. Hence there is an underlying normal sinus rhythm. The PR interval (+-) is very variable, indicating AV dissociation. The presence of AV dissociation with an atrial rate that is faster than the ventricular rate indicates complete heart block. In contrast, an atrial rate that is slower than the ventricular rate would indicate an accelerated lower focus (ie, accelerated junctional or ventricular rhythm). The escape rhythm has a narrow QRS complex that is similar to the QRS complex in ECG 12A. Hence the escape rhythm is junctional. The escape rhythm is determined by the QRS morphology and not the heart rate. The fact that with the development of complete heart block the escape rhythm is junctional means that the 2:1 AV conduction seen in ECG 12A is Mobitz type I.

Complete heart block is manifest by AV dissociation (ie, there is no association between P waves and QRS complexes and the PR intervals are variable). The atrial rate is faster than the rate of the QRS complexes. The escape rhythm may be junctional or ventricular; the location of the escape rhythm is based on QRS morphology and not rate.

The patient is manifesting multiple symptoms of an excess β-blocker effect (fatigue, erectile dysfunction, and AV block). High doses of agents that prolong the normal, physiologic AV delay, such as digoxin, β-blockers, and calcium-channel blockers, may precipitate symptomatic AV block (second or third degree). The patient’s infra-Hisian conduction system appears intact as he manifests a junctional escape rhythm when in complete heart block. Hospital admission for β-blocker wash-out and controlled re-institution of a low-dose β-blocker is a reasonable course of action at this time.
A 72-year-old homeless Korean War veteran is admitted to the Veterans Administration hospital with an episode of syncope. It is clear from his electronic record that he has obtained no medical care since his honorable discharge from the service. He is awake, alert, oriented, and conversant on admission. He denies any prodrome to the episode and is...
asymptomatic at present. His vital signs are notable for a heart rate of 42 bpm and a blood pressure of 170/90 mm Hg. An ECG is obtained (13A). The next morning, he has a witnessed episode of syncope. Again, no prodrome was evident, and he recovers consciousness spontaneously after a few seconds. The nurse quickly obtains an ECG (13B).

What abnormalities are depicted?
How does the ECG immediately after his syncopal event inform you of the mechanism and point to the necessary therapy?
Podrid's Real-World ECGs

ECG 13A Analysis: Sinus bradycardia, first-degree AV block (prolonged AV conduction), second-degree AV block with 2:1 conduction, right bundle branch block, left ventricular hypertrophy
In ECG 13A, there is a regular rhythm at a ventricular rate of 42 bpm. There is a P wave (*) before each QRS complex with a stable PR interval of 0.32 second (±) (ie, first-degree AV block or prolonged AV conduction). There is a second nonconducted, on-time sinus P wave (+), best seen in leads I (at the end of the T wave), aVL, and V1. T waves should be smooth in upstroke and downstroke. Any notching or bump on the T wave is suspicious for a superimposed P wave. This can be seen in leads V3 and V6 (±), for example. The PP intervals are regular (∓) at an atrial rate of 84 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a sinus rhythm with first-degree AV block as well as second-degree AV block with 2:1 AV conduction. The QRS complexes are wide (0.14 sec) with a right bundle branch block pattern (RSR' morphology in lead V1 [→] and broad S wave in leads I and V5-V6 [→]). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The tall QRS amplitude in leads V3-V4 (27 mm) (|) meets the criteria for left ventricular hypertrophy (R-wave amplitude or S-wave depth in any precordial lead ≥ 25 mm). The QT/QTc intervals are normal (520/435 msec and 480/400 msec when the prolonged QRS complex duration is considered).
**Podrid’s Real-World ECGs**

**ECG 13B Analysis:** Complete heart block with ventricular escape rhythm with a left bundle branch block morphology and intermittent capture (AV conduction)
In ECG 13B, there is a regular rhythm at a rate of 42 bpm. There are regular P waves (*) at an atrial rate of 98 bpm. Some of the P waves are not obvious as they are within the QRS complex. However, when seen, the P waves are regular with a stable rate. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is an underlying sinus rhythm. The PR intervals are variable (U) and therefore dissociated from the QRS intervals (AV dissociation). An atrial rate that is faster than the ventricular rate with AV dissociation is diagnostic for complete or third-degree AV block. The QRS complexes are wide (0.16 sec) with a left bundle branch block morphology (ie, QS complex in lead V1 [-] and a tall R wave in lead I [+]). Moreover, the QRS complexes in ECG 13B are very different from those in ECG 13A, which have a right bundle branch block pattern. Therefore, the QRS complexes in this ECG are ventricular (ie, there is a ventricular escape rhythm). The last QRS complex (▲) is different and has a PR interval of 0.32 second (<> ) and right bundle branch pattern, similar to the QRS complexes and PR intervals seen in ECG 13A; this complex is conducted. Therefore, this is complete heart block with an escape ventricular rhythm and intermittent capture. The presence of a ventricular escape rhythm means that the 2:1 AV conduction seen in ECG 13A is Mobitz type II and that the conduction abnormality is within the His-Purkinje system. Mobitz type II second-degree heart block as well as complete heart block with a clear relationship to symptoms of syncope is a definitive indication for permanent pacemaker implantation. ■
A 52-year-old man is in the cardiac catheterization laboratory undergoing percutaneous coronary intervention for acute myocardial infarction. His presentation was typical, with marked substernal chest pressure and diaphoresis prompting activation of emergency medical services. Diagnosis and immediate catheterization laboratory referral were based on his history and the appearance of a left bundle branch block pattern on his index ECG; it was uncertain whether this pattern was new or predated his acute presentation. During the initial angioplasty of an occluded left anterior descending artery, the patient complains of severe lightheadedness. The ECG shown is representative of his rhythm at the time. His vital signs are notable for a heart rate of 46 bpm and a blood pressure of 88/46 mm Hg.

What abnormalities are depicted on the ECG?
What is the precise nature of the abnormality?
ECG Analysis: Normal sinus rhythm, left bundle branch block, complete heart block with an escape ventricular rhythm, second-degree heart block with 2:1 conduction (2:1 AV block)
The first two QRS complexes have a rate of 64 bpm. Thereafter, the rhythm is regular at a ventricular rate of 46 bpm. There is a P wave before each QRS complex with a stable PR interval (0.16 sec). In addition, there is a second on-time P wave after each QRS complex; these P waves are nonconducted. Occasionally this nonconducted P wave is seen at the end of the T wave, causing the downstroke of the T wave to have an irregular bump on it. The PP intervals are regular, and the atrial rate is 92 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. This is, therefore, a normal sinus rhythm with second-degree AV block and 2:1 AV conduction. The QRS complexes are wide (0.16 sec) and have a left bundle branch block pattern (deep S wave in lead V1 and broad R wave in lead V6). The QT/QTc intervals are prolonged (520/460 msec) but are normal when the prolonged QRS complex duration is considered (440/390 msec).

The first two QRS complexes have a different morphology and rate (64 bpm). There are P waves noted at the same rate as the other P waves seen during 2:1 AV block (ie, 92 bpm). It can be seen that the morphology of the first QRS complex is slightly different than that of the second, as a result of a superimposed P wave at the end of the complex. However, there is no association between the P waves and the QRS complexes; hence AV dissociation is present. As the atrial rate is faster than the ventricular rate, this is a brief period of complete (third-degree) AV block with an escape ventricular rhythm. Following this there is second-degree heart block with 2:1 AV conduction that is the result of Mobitz type II due to block within the His-Purkinje system. This is established by the fact that with the development of complete AV block the escape rhythm is ventricular.

During an anterior wall myocardial infarction involving the septum, there may be significant injury to the infra-Hisian conduction system that can result in Mobitz type II second-degree AV block or complete AV block, the latter of which will be associated with a ventricular escape rhythm. When there is complete AV block, a temporary pacing electrode is often inserted for ventricular pacing. Although there may be some recovery of His-Purkinje conduction during the period after reperfusion, the fact that the patient has a left bundle branch block (which may be new or preexistent) that appears to be permanent would suggest that the septal damage is not reversible and the conduction abnormality within the His-Purkinje system will be permanent, requiring a permanent pacemaker.
A 72-year-old man presents to an outpatient clinic for a routine physical exam. He has no complaints and does not admit to any previous cardiac problem. Except for an elevated blood pressure (170/90 mm Hg), his physical exam is normal. He is started on therapy with a β-blocker and hydrochlorothiazide. An ECG (15A) is obtained but not read.
Two days later he presents to an emergency department with complaints of lightheadedness and a feeling that he might pass out, although he denies syncope. His physical exam is remarkable for a blood pressure of 140/80 mm Hg, although it appears to vary. His pulse rate is about 40 bpm. There are intermittent cannon A waves noted in his neck. An ECG (15B) is obtained.

What abnormalities can be seen on the ECGs? Are the abnormalities related to each other? Is there a relationship between the rhythm and the drug therapy that was prescribed?
Podrid's Real-World ECGs

ECG 15A Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type II second-degree AV block, high-grade AV block, old anterior wall myocardial infarction, low voltage
In ECG 15A, there is a regular rhythm with one long RR interval seen (••). The ventricular rate is 42 bpm. There are P waves (*) before each QRS complex with a stable PR interval (0.48 sec), indicating first-degree AV block. A second P wave can be seen after each QRS complex (+); the P wave is within the T wave, best seen in leads V1-V2 (*). Normal T waves should be smooth in upstroke and downstroke. It can be seen that these T waves have a notching on the upstroke; this is apparent in leads I, II, aVL, and V6 (*). The PP intervals are constant (••), and the atrial rate is 84 bpm. Hence this is a normal sinus rhythm with first-degree AV block (prolonged AV conduction) and second-degree AV block with 2:1 AV conduction (or 2:1 AV block). There are two on-time but nonconducted P waves (⊥) during the long RR interval (••). With Mobitz type I there is only one nonconducted P wave at a time, while with Mobitz type II there may be more than one sequential nonconducted P wave. Hence this block is Mobitz type II, although it is often called high-grade AV block when more than one nonconducted P wave is seen.

The QRS complex duration is normal (0.08 sec), but there is low voltage (< 5 mm in each limb lead and < 10 mm in each precordial lead). The axis is probably normal, between 0° and +90°, although this is not certain as the QRS complex amplitude in lead aVF is very small. The QT/QTc intervals are normal (520/435 msec). There are QS complexes in leads V1-V3 (⊥), consistent with an old anterior wall (anteroseptal) myocardial infarction (MI). The T waves are inverted in leads V1-V4 (⊥); this is the result of the old MI.

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Podrid's Real-World ECGs

ECG 15B Analysis: Normal sinus rhythm, complete heart block, escape ventricular rhythm
ECG 15B shows a regular rhythm at a rate of 46 bpm. P waves can be seen (+), although some of them are obscured by the QRS complex or are within T waves or ST segments (+). However, the PP interval (LA) is constant and the atrial rate is 98 bpm. There is no relationship between the P waves and the QRS complex (ie, the PR intervals are variable). Hence this is a sinus rhythm with AV dissociation. As the atrial rate is faster than the ventricular rate, this is complete (third-degree) AV block. The QRS complex duration is increased (0.14 sec) and, although the QRS complexes have a morphology that resembles a left bundle branch block (LBBB; ie, QS complex in lead V1 [+]+ and a tall R wave in leads I and V6 [-I]), the morphology is not typical of an LBBB as there is a Q wave in lead aVL (+) and a strange QRS morphology in leads II, III, and aVF with a prominent terminal S wave. Importantly, the QRS complex in ECG 15B is wider and has a different morphology than the conducted QRS complexes in ECG 15A. Hence this is a ventricular escape rhythm, confirming the fact that ECG 15A showed a Mobitz type II block with infranodal disease. Cannon A waves are seen with AV dissociation of any cause and are due to intermittent right atrial contraction against a closed tricuspid valve (ie, when the P wave resulting in atrial contraction is simultaneous to or slightly after the QRS complex, resulting in ventricular contraction).

Since Mobitz type II is due to a conduction abnormality that is infranodal (below the AV node and within the Purkinje system), the occurrence of complete heart block will be associated with an escape ventricular rhythm, as the ventricular myocardium is the only remaining distal site from which an impulse can activate the ventricles. The development of complete heart block in this patient is the result of degenerative disease within the His-Purkinje system, and it may be the result of a previous anterior wall MI with the development of fibrosis of the interventricular septum. This conduction abnormality is not related to the medications that were prescribed; specifically, it is not due to the β-blocker. A β-blocker can cause complete heart block, but this is due to block of the AV node and hence would be associated with an escape junctional rhythm. The His-Purkinje system is not affected by β-blockade. The appropriate therapy for this patient is implantation of a permanent pacemaker.
A 68-year-old woman is brought to the hospital by emergency medical services after being found unresponsive at home. The time from loss of consciousness to presentation is unknown, but she began to regain consciousness en route to the hospital. She is not responding to commands but is moving her left side spontaneously. Her vital signs are notable for a pulse of 42 bpm and a blood pressure of 210/105 mm Hg. She is breathing spontaneously but has been intubated for airway protection. She is afebrile. An ECG is obtained.

What abnormalities are depicted?
What etiologic entity do these abnormalities suggest in this clinical scenario?
ECG 16 Analysis: Normal sinus rhythm, AV dissociation from third-degree AV block, junctional escape rhythm, diffuse T-wave inversions
The heart rate is stable at 42 bpm. The P waves (*) are positive in leads I, II, aVF, and V4-V6. There is a stable PP interval (LJ), and the atrial rate is 76 bpm; hence this is a normal sinus rhythm. However, the PR intervals (+) are not constant and there is no association between the P waves and the QRS complexes. Some of the P waves are superimposed on the T waves or on the beginning or end of the QRS complex and, therefore, are not obvious (+). However, when P waves are seen (*), they are on time. Hence this represents AV dissociation. As the atrial rate is faster than the rate of the QRS complexes, this is complete or third-degree AV block. The QRS complex has a normal duration (0.08 sec) and morphology. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/360 msec). The QRS complexes are, therefore, supraventricular and the escape rhythm is junctional.

AV dissociation may be subclassified based on etiology. AV dissociation may be due to an accelerated idioventricular rhythm or a junctional rhythm, in which a ventricular or junctional pacemaker is faster than the atrial rate. A ventricular or junctional etiology is based on the morphology of the QRS complex and not the rate. AV dissociation may also be due to complete (third-degree) AV block in which the atrial activity is not conducted to the ventricles and the atrial rate will be faster than the ventricular rate. In these cases, it is important to identify the origin of the “escape” rhythm (ie, junctional/AV nodal or ventricular). The origin of the escape rhythm is based on QRS morphology and not rate. Junctional escape rhythms may be at rates of less than 50 bpm, while a ventricular escape rhythm may have a rate of that exceeds 70 bpm. 

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The QRS complex of an escape junctional rhythm will resemble the native QRS complex; the complex will be narrow if no baseline conduction disease is present, whereas it will be wide with a conduction delay if such a conduction abnormality was present prior to the development of complete AV block. The QRS complex of an escape ventricular rhythm will be wide and unusual in morphology (not resembling either a right or left bundle branch block), and it will not resemble the native QRS complex during intact AV conduction. In the ECG shown, the QRS complexes are narrow and, therefore, junctional in origin. A temporary pacemaker is not necessary unless there are symptoms from the bradycardia.

There are also T-wave abnormalities (inversions) in leads II, III, aVR (positive T waves are actually inverted in this lead), aVF, and V3-V6. T-wave inversions have a broad differential diagnosis, including subendocardial ischemia, myocardial infarction, cerebrovascular accidents (particularly hemorrhagic), left ventricular hypertrophy and hypertrophic cardiomyopathies, “memory” T waves (post-ventricular pacing), and idiopathic. Without a history or other ECG changes, they are considered to be nonspecific. Importantly, the T waves are asymmetric, which also suggests that they are nonspecific.
The clinical scenario in this case suggests that the T-wave inversions may be the result of a cerebrovascular accident. However, these are not cerebral T waves, which are usually markedly deeply inverted in all leads and associated with a long QT interval. Common ECG changes during acute stroke include a long QT interval, diffuse deep T-wave inversions, supraventricular and ventricular ectopy, atrial arrhythmias, and various forms of AV block, often the result of autonomic imbalance. ST-segment changes (both elevation and depression) and elevation of cardiac biomarkers may be seen as well. Predictors of increased mortality include atrial fibrillation, AV block, ST- and T-wave changes, and elevation of cardiac biomarkers.
A 59-year-old woman with dilated cardiomyopathy of unknown etiology is brought to the hospital emergency department by her sister after she spontaneously lost consciousness at home. On arrival, the woman is semi-conscious and moaning. A history is unobtainable. Telemetric monitoring is initiated and an ECG is obtained (17A) as vital.
signs are gathered, a primary survey is completed, and preparations are made for intubation. Before any intervention can be performed, the woman’s level of consciousness increases and she asks where she is and how she got there. You notice that her heart rate appears faster than on the ECG tracing you were provided. You request a second tracing (17B).

What do the tracings suggest regarding the location of the defect in her conduction system?
ECG 17A Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), complete heart block, escape ventricular rhythm, diffuse T-wave inversions
In ECG 17A, there is a regular rhythm at a rate of 40 bpm. There are P waves (*) with a regular PP interval at a rate of 62 bpm (>). The P waves are positive in leads I, II, aVF, and V4-V6. The negative deflection of the P wave in lead V1 is prominent (> 1 mm wide and 1 mm deep), indicating left atrial hypertrophy. Hence this is a normal sinus rhythm. The PR intervals (±) are not constant; hence there is AV dissociation. The atrial rate is faster than the ventricular rate, diagnostic for complete heart block. The QRS complexes are wide (0.14 sec) and have a left bundle branch block (LBBB)-like pattern, although it is not typical LBBB as there is a terminal S wave (--) in lead I. There are no left-to-right forces seen with an LBBB. The axis is normal, between 0° and 90° (positive QRS complex in leads I and aVF). It is not certain whether the complexes are ventricular or junctional with an intraventricular conduction delay. Therefore, the location of the escape rhythm is not certain on this ECG. T-wave inversions can be seen in leads I, II, III, aVR (the positive T wave in this lead is actually inverted), aVF, and V3-V6 (*). The QT/QTc intervals are normal (500/410 msec).

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ECG 17B Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), intraventricular conduction delay
ECG 17B shows a regular rhythm at a rate of 68 bpm. There is a P wave (*) before each QRS complex, and the PR interval (Li) is stable at 0.20 second. The P wave is positive in leads I, II, aVF, and V4-V6. Therefore, this is a sinus rhythm. The QRS complexes are wide (0.16 sec) and have an LBBB-like pattern (broad R wave in leads I and V6 [-] and deep S wave in lead V1 [→]). However, there is a small initial R wave in lead V1 (▼), representing an initial septal force, which is not present in an LBBB. In addition there is a terminal S wave in lead V6 (▲), representing terminal left-to-right forces that are not seen with an LBBB. Therefore, this is an intraventricular conduction delay. The QT/QTc intervals are prolonged (440/470 msec) but are normal when the prolonged QRS complex duration is considered (360/385 msec). The QRS complexes seen in ECG 17A have a different morphology from these complexes, which are supraventricular in origin as this is a sinus rhythm. This confirms the fact that the escape rhythm in ECG 17A is ventricular in origin and that the location of the conduction block is infranodal. As this is complete heart block with an escape ventricular arrhythmia and it is associated with significant symptoms, a permanent pacemaker is indicated. If there is a delay in placing a permanent device, a temporary pacemaker would be indicated as an escape ventricular arrhythmia is not stable and is unpredictable with regard to rate.
A 42-year-old man with a long history of drug abuse and known cocaine-associated dilated cardiomyopathy presents for routine follow-up. His review of systems is unremarkable, and his exam suggests that he is euolemic. However, the physician notices that the patient’s heart rate is
44 bpm and obtains an ECG (18A). The physician promptly admits the patient to the hospital and calls for an electrophysiology consult for pacemaker implantation. The following day, the physician obtains a follow-up ECG (18B) and is gratified to learn that his initial suspicion was correct.

What on the initial ECG was so concerning to the physician? What on the follow-up tracing solidified the diagnosis?
Podrid's Real-World ECGs

ECG 18A Analysis: Sinus rhythm, AV dissociation/complete heart block, ventricular escape rhythm
ECG 18A shows a regular wide QRS complex rhythm at a rate of 44 bpm. The QRS complex duration is 0.20 second, and the morphology does not resemble either a left or right bundle branch block. It is likely a ventricular complex. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (440/380 msec). There are P waves (*) that are positive in leads I, II, aVF, and V4-V6; hence they are sinus P waves. The P waves are not always visible as some are within or at the beginning of the QRS complex (▼). Two sequential P waves can be seen between the last two QRS complexes (+), and when this PP interval is used it can be seen that there is a regular PP interval (□) and the sinus rate is 90 bpm. Although some P waves are not seen, those that are obvious occur on time with a regular interval (□). There is variability of the PR interval (+–), indicating AV dissociation. Since the atrial rate exceeds the QRS complex rate, this is complete heart block with an escape ventricular rhythm.
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ECG 18B Analysis: Sinus rhythm, first-degree AV block (prolonged AV conduction), intraventricular conduction delay to the right ventricle
ECG 18B shows a regular wide complex rhythm at a rate of 76 bpm. The QRS complex duration is very prolonged (0.20 sec). Although the morphology is that of a right bundle branch block (RSR’ morphology in lead V1 [-++] with a very broad R’ wave and very broad S waves in leads I and V6 [---]), the QRS complex is much wider than what is seen with a bundle branch block. In general, the QRS complex width in a bundle branch block is 0.18 second or less. This suggests that this is an intraventricular conduction delay to the right ventricle (supported by the very broad R’ and S waves) caused by dilated cardiomyopathy with myocardial fibrosis. There is a P wave (*) before each QRS complex with a stable PR interval (++), which is prolonged at 0.28 second (ie, this is sinus rhythm with first-degree AV block or prolonged AV conduction). The QRS complex is supraventricular and has a right bundle branch block pattern that is different from the QRS complex seen in ECG 18A, establishing the fact that the escape rhythm in ECG 18B is ventricular and hence the complete heart block is a result of His-Purkinje system disease. ■
A 78-year-old woman with end-stage ischemic cardiomyopathy is admitted to the coronary care unit with decompensated heart failure. She is intubated on full inotropic support. The patient's heart rate has been stable. However, in watching the telemetry monitor, an astute medical student expresses concern about the patient's rhythm and obtains an ECG.

What conduction system abnormality is depicted on the ECG?
ECG 19 Analysis: Sinus tachycardia, AV dissociation/complete heart block, escape ventricular rhythm
There is a regular rhythm at a rate of 72 bpm. P waves can be seen (*), although occasionally the P wave is at the beginning or end of the QRS complex (+) and is not obvious. When seen, however, the P waves are occurring at a regular interval (L) at a rate of 110 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is sinus tachycardia. The PR intervals (+) are not stable; therefore, the P waves are independent of the QRS complexes, thus defining AV dissociation. AV dissociation in this case is due to complete heart block since the atrial rate exceeds the rate of the QRS complex. The QRS complexes are widened, with a duration of 0.16 second and a right axis, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). The QT/QTc intervals are prolonged (440/480 msec) but are normal when the prolonged QRS complex duration is considered (360/395 msec).

The QRS complexes do not have a pattern of either a right or left bundle branch block, but rather are wide with an unusual morphology. Hence there is an escape ventricular rhythm, even though the ventricular rate is 72 bpm. The fast ventricular rate is likely a result of an enhanced sympathetic state as the sinus rate is 110 bpm. As indicated, the location of the escape rhythm (junctional or ventricular) is based on the QRS morphology and not the rate. In this case the escape ventricular rhythm is 72 bpm, faster than is usually seen with an escape ventricular focus. However, this is in association with a sinus rate of 110 bpm, meaning that there is an increase in catecholamines or sympathetic tone that is driving both the sinus node and ventricular focus.

Noted in lead V2 are waveforms (W) just before the QRS complex. These are not P waves, but rather are part of the QRS complex, established by measuring the maximum QRS complex width from simultaneously occurring complexes (in either lead V3 or the lead II rhythm strip) (II). It can be seen that the waveform just before the QRS complex is part of the QRS complex and not a P wave.
A 74-year-old woman with a history of hypertension that has not been well controlled despite therapy with a β-blocker and an angiotensin-converting enzyme inhibitor presents to her physician with a complaint of fatigue that has been present for the past week. She denies chest discomfort, shortness of breath, and lightheadedness. The physical exam reveals a blood pressure of 170/90 mm Hg and a heart rate of 40 bpm, but is otherwise unremarkable. An ECG is obtained.

What abnormality is seen on the ECG?
Is this abnormality the result of the patient’s medication?
What therapy is indicated?
Podrid's Real-World ECGs

ECG 20 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), third-degree or complete heart block, escape ventricular rhythm, intermittent AV capture, premature atrial complex
There is a regular rhythm at a rate of 42 bpm. There is evidence of atrial activity (+), and the PP interval (~) is generally regular at a rate of 64 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. The P waves are broad and notched, particularly in leads II, III, and aVF, consistent with left atrial hypertrophy (or abnormality). There is no association between the P waves and the QRS complexes as the PR intervals are variable (~). Hence there is AV dissociation. As the atrial rate is faster than the ventricular rate, this is complete or third-degree AV block. There is a P wave that is premature (~), and it has a different morphology than the sinus P waves. This is a premature atrial complex, which resets the sinus P waves, accounting for the slight irregularity of the PP interval.

The QRS complexes have two different durations and morphologies. QRS complexes 1, 2, 4, 5, and 7 (*) have a wide duration (0.16 sec) and are associated with a variable PR interval (~). They have a left bundle branch block morphology with a broad R wave in leads I and V6 (~) and a QS complex in lead V1 (~). The narrow QRS complexes (complexes 3 and 6) (~) have a normal duration (0.08 sec) and morphology. Although the narrow complex is not seen in lead I, the axis is likely very leftward, between −30° and −90°, as the QRS complex is negative in leads II and aVF. These are thus supraventricular complexes. The QRS amplitude is markedly increased in lead V5 (22 mm) (~). Although it does not meet a criterion for left ventricular hypertrophy, a narrow QRS complex is not seen in leads V1-V3 and hence it is possible that left ventricular hypertrophy is present. The narrow QRS complexes are slightly early and both are preceded by a P wave with the same PR interval (0.18 sec) (~). Therefore, these two complexes are responding to the P wave that precedes them; hence they result from AV conduction. As the conducted QRS complexes have a normal duration and morphology that is different from the wide dissociated QRS complexes, the escape rhythm is ventricular and there is intermittent capture or AV conduction.

Although the patient does have a history of hypertension, her elevated blood pressure on presentation may be the result of the complete heart block. Elevated systolic pressure is often observed with complete heart block as a result of AV dyssynchrony and increased left ventricular stroke volume. With AV dissociation and the more rapid atrial rate there is an increase in ventricular filling with an increased left ventricular end-diastolic volume. Hence as a result of a Starling effect, left ventricular contractility and stroke volume are increased, resulting in systolic hypertension. Any decision about blood pressure and further medical therapy should wait until after a permanent pacemaker has been placed.
A 63-year-old man with end-stage renal disease is being evaluated by his nephrologist during a hemodialysis session. He complains of intermittent palpitations but is otherwise feeling well. On exam, the patient's heart rate is slow. On observation of the jugular venous pulse, intermittent brisk deflections to the angle of the jaw are noted (i.e., cannon A waves). The cardiopulmonary exam is otherwise normal. These observations prompt recording of a surface 12-lead ECG.

What on the ECG explains the physical exam findings and the patient's symptoms?
ECG 21 Analysis: Normal sinus rhythm, premature atrial complex, AV dissociation, third-degree or complete heart block, intermittent retrograde (ventriculoatrial) conduction
The RR intervals are regular at a rate of 44 bpm. The QRS complexes are narrow (0.08 sec), with a normal morphology and normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complexes are, therefore, supraventricular. The QT/QTc intervals are normal (480/410 msec). There are P waves (*), which are positive in leads I, II, aVF, and V4-V6. These are, therefore, sinus P waves. The PR intervals are variable (+), and hence there is AV dissociation. The sinus rate is not constant. The first four P waves (+) occur with a regular interval (LI) at a rate of 74 bpm. The P waves (▼) before and after the fifth and seventh QRS complexes (▼) have the same PP interval (LI) (rate of 74 bpm). It is noted that the P waves after the third, fourth, and sixth QRS complexes (●e, the fifth, sixth, and ninth P waves) are late (●); that is, there is a longer PP interval (LIJ). The third, fourth, and sixth QRS complexes (▼) are different as there is a negative waveform (▼) at the end of each complex. This is a retrograde P wave, with a fixed RP interval, indicating intermittent ventriculoatrial (VA) conduction from the junctional beat back to the atrium. As a result the sinus node is reset and hence the PP interval is altered and the P waves are late (LIJ).

Therefore, this is complete heart block with an escape junctional rhythm and occasional VA conduction. The VA conduction occurs whenever the atrium is able to depolarize (●e, when the atrial activity is prior to the QRS complex with a long enough PR interval such that the AV node and atrium have had a chance to recover and are responsive to retrograde activation). It is important to note that even though there is complete antegrade AV block, there is still retrograde VA conduction, which is seen in about 20% to 30% of cases of complete AV block. As the antegrade and retrograde nodal properties are different, there may be intact retrograde or VA conduction even though there is no antegrade conduction. Alternatively, retrograde or VA conduction may be due to a concealed bypass tract, which usually conducts only retrogradely.

Also noted are additional P waves that are early (▲) (●e, after the fifth QRS complex and before the last [eighth] QRS complex). These are atrial premature beats that are not conducted.

The physical exam is consistent with complete heart block, particularly the presence of cannon A waves, which are due to atrial contraction against a closed tricuspid valve. This occurs whenever P waves superimpose on the QRS complexes, as in complete (third-degree) AV block.

Although there is complete AV block, it is not certain whether this is transient or permanent. Moreover, it is not clear whether this is related to electrolyte abnormalities associated with renal failure. While complete AV block is an indication for a permanent pacemaker, even though the escape rhythm is junctional, the risk for this must be weighed against the fact that the patient is on long-term dialysis, a factor that is associated with an increased risk for infection.
A 59-year-old college professor presents to the health clinic with complaints of intermittent palpitations but no other symptoms. Although he has no cardiac history, he states that he was told years ago that he had hypertension but has not been on any therapy. He is fully active and jogs about 6 miles per day. He has no symptoms while running. His physical exam and blood pressure are normal, and his heart rate is 90 bpm. An ECG is obtained.

What abnormality is noted? Is a pacemaker indicated?
Podrid's Real-World ECGs

ECG 22 Analysis: Atrial rhythm, AV dissociation, accelerated junctional rhythm, occasional captured complex, left ventricular hypertrophy with associated ST-T wave changes
There is a regular rhythm at a rate of 94 bpm, although QRS complexes 5 and 14 (+) are early. The QRS complexes have a normal duration (0.08 sec) and a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/425 msec). The QRS complex has a tall R wave in lead V5 (32 mm) (▲) and a deep S wave in lead V2 (22 mm) (S-wave depth in lead V2 + R-wave amplitude in lead V5 = 54 mm). This meets one of the criteria for left ventricular hypertrophy (ie, S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm [or ≥ 45 mm if age under 45]). There are significant ST-T wave abnormalities seen in leads V4-V6 (▲), which are secondary to left ventricular hypertrophy and represent repolarization changes due to chronic subendocardial ischemia. Coronary blood flow goes from epicardium to endocardium and hence the last part of the myocardium to receive blood and oxygen supply is the subendocardial layer. When the myocardium is hypertrophied, the amount of blood or oxygen reaching the endocardial layer is limited, resulting in chronic subendocardial ischemia.

There are P waves seen (.*,▲), and they are negative in leads II, III, and aVF. The P waves appear to be occurring after the QRS complex, suggesting that they are retrograde resulting from the junctional complexes. In addition, it appears that there is a progressively prolonging RP interval (▲), suggesting retrograde or ventriculoatrial (VA) Wenckebach. If this were the case, the fifth and 14th QRS complexes, which are early and preceded by a negative P wave, would probably represent echo beats, resulting from retrograde or VA conduction stimulating the atria and then conducting antegradely through the AV node to reactivate the ventricles. However, the same negative P wave can be seen before the sixth to eighth QRS complexes (▲), meaning that the P waves are in fact not retrograde but are antegrade. Indeed, there is a stable PP interval (▲) at an atrial rate of 86 bpm. The PR interval is not constant and hence there is AV dissociation. Since the atrial rate is slower than the ventricular rate, the AV dissociation is not the result of complete heart block (in which the atrial rate is faster than the ventricular rate). The AV dissociation is, therefore, the result of an accelerated junctional rhythm that is faster than the atrial rate. As the P waves are negative in leads II, III, and aVF, this is not a sinus rhythm but an atrial rhythm. The two early QRS complexes (fifth and 14th) (+), which have the same PR interval (0.24 sec), are thus captured complexes. The ability of the atrial impulse to conduct through the AV node and capture the ventricle depends on appropriate timing of the atrial impulse relative to the previous QRS complex. The AV dissociation is due to the fact that the junctional impulse retrogradely depolarizes the AV node, preventing antegrade impulse conduction from the atrium. If the AV node has recovered before the atrial impulse reaches it antegradely, the atrial impulse can be conducted through the node to capture the ventricle.

Since the AV dissociation is the result of an accelerated junctional rhythm and not complete heart block, there is no indication for the insertion of a pacemaker. An accelerated junctional rhythm can occur in many clinical scenarios but often is seen as a result of sinus or atrial bradycardia and an ectopic junctional focus that generates an impulse at a rate that is faster than the rate of the sinus node or atrial focus. ▲
Notes
A 92-year-old woman is found unconscious by her family and brought to the local emergency department. Her daughter is not fully aware of her medical history, but states that her mother has been treated for hypertension, atrial fibrillation, and heart failure and is on medications for this. For the past several days, the patient had been complaining of abdominal pain and diarrhea but seemed to be doing fine otherwise. She has no other active medical conditions. On presentation, the patient's vital signs are notable for a heart rate of 36 bpm and a blood pressure of 84/58 mm Hg. She is quite somnolent and responds minimally to voice commands. She is anicteric, her lungs are clear on auscultation, and her heart has a nondisplaced point of maximal impulse and is regular without murmur, gallop, rub, or tap. Her extremities are cool, and skin turgor is decreased. No edema is evident. Her laboratory studies are notable for a serum sodium level of 156 mEq/L, potassium 3.2 mEq/L, blood urea nitrogen 92 mg/dL, and serum creatinine 4.6 mg/dL. Her cardiac biomarkers and complete blood count are normal. Intravenous fluid resuscitation is initiated and an ECG is obtained.

What abnormalities are depicted?

What medications are likely to cause these abnormalities?
ECG 23 Analysis: Atrial fibrillation with complete heart block and escape junctional rhythm, clockwise rotation, nonspecific ST-segment abnormality
There are regular RR intervals (LJ) at a rate of 36 bpm. There are no organized P waves seen; however, there are rapid undulations (W) of the baseline between each QRS complex; these undulations are irregular in morphology, amplitude, and interval. Hence the underlying rhythm is atrial fibrillation. The regularization of the QRS complex intervals indicates that there is complete heart block with an escape rhythm. The QRS complexes have a normal duration (0.08 sec) and morphology. Hence the escape rhythm is junctional. There is a left axis, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). There is poor R-wave progression from lead V1 to lead V3 with late transition (ie, R/S > 1 in lead V5). This is consistent with a diagnosis of clockwise rotation in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. With clockwise rotation, the left ventricular forces are directed more posteriorly and occur later in the precordial leads. In addition, there is flattening of the ST segments (‘), as noted in leads I, II, aVF, and V4-V6. This is a nonspecific abnormality. The QT/QTc intervals are normal (520/400 msec).

The patient presents with signs, symptoms, and laboratory data suggesting significant dehydration, as a result of diarrhea, likely poor intake, and possibly the continued use of diuretics, although what drugs she is taking is unclear. She carries a diagnosis of heart failure, suggesting that she may be taking diuretics, and also atrial fibrillation for which digoxin, a β-blocker, or a calcium-channel blocker may be taken for rate control. Digoxin is cleared by the kidney, but in acute renal failure or prerenal azotemia, in this case due to dehydration in the context of an acute diarrheal illness, digoxin levels can increase acutely, leading to signs of digoxin toxicity that are manifest as significant conduction slowing through the AV node and the possibility of complete AV block, as seen in this ECG. Establishing digoxin toxicity in the presence of atrial fibrillation may be difficult. The earliest sign of excessive digoxin effect due to elevated digoxin levels (mediated by increased vagal tone) is the development of bradycardia, although the RR intervals remain irregularly irregular. With further elevation of digoxin levels there is the development of complete AV block, which is initially intermittent and presents with long RR intervals all of which have the same

continues
duration intermittent regularization), indicating intermittent heart block with an escape focus. With further elevation of digoxin levels, the heart block becomes persistent, presenting as regularization of the RR intervals and the presence of an escape junctional rhythm, as is seen on this ECG. With further elevation of digoxin levels, the rate of the escape junctional rhythm increases (due to an increase in sympathetic output from the central nervous system), and nonparoxysmal junctional tachycardia occurs. Further elevation of digoxin levels results in His-Purkinje system abnormalities, with slowing of conduction through the bundles. This results in junctional tachycardia with alternating bundle branch block, called bidirectional tachycardia. Ultimately, there may be complete block within the bundles, resulting in an escape ventricular rhythm. The rate of the escape ventricular rhythm may be variable.

Another possible drug that she may be taking is a β-blocker (for hypertension and rate control of atrial fibrillation), which can also produce complete AV block. The two long-acting β-blockers, atenolol and nadolol, are cleared primarily by the kidneys. With reduced renal function, there may be an increase in the blood level and hence in the therapeutic effect of the β-blocker, leading to AV nodal blockade and the development of complete heart block.

In addition to rehydration, treatment is to withhold any agents that can affect AV nodal impulse transmission until there is resolution of the renal failure and the complete heart block. If the complete AV block is reversible, there is no indication for a pacemaker. ■
A 24-year-old man is admitted to the hospital with traumatic osteomyelitis. Because of marked bradycardia noted on exam, an ECG is performed. The patient's physical exam is notable for a soft, early diastolic murmur at the left upper sternal border. The ECG technician approaches you with the ECG, stating that he thinks the patient has Wolff-Parkinson-White syndrome.

Do you agree with the technician's diagnosis? 
What further evaluation is warranted? 
What therapy would help establish the diagnosis?
Podrid's Real-World ECGs

ECG 24 Analysis: Sinus bradycardia, isorhythmic AV dissociation, junctional rhythm
There is a regular narrow complex rhythm with a regular RR interval of 1.6 second and a rate of 38 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (520/414 msec). Hence these are supraventricular complexes. P waves (*) are seen before each QRS complex, with a stable PP interval (1.6 sec) (+ +) and an atrial rate of 38 bpm. The P waves are positive in leads II, aVF, and V4-V6. Hence this is a normal sinus rhythm. However, the PR intervals are not constant (\(\downarrow\)), becoming progressively longer. The P wave is actually superimposed on the first QRS complex (\(\uparrow\)). As a result it looks like there is a delta wave; however, the apparent slurred upstroke is the P wave. Hence AV dissociation is present. As measured on the ECG when recorded at 25 mm/sec, the atrial and ventricular rates are identical. In this situation it cannot be established if there is complete AV block with an escape junctional rhythm (in which the atrial rate is faster than the junctional rate) or an accelerated junctional rhythm (in which case the atrial rate is slower than the junctional rate). Hence this is termed isorhythmic dissociation with a junctional rhythm.

The combination of possible heart block as well as a diastolic murmur suggestive of aortic regurgitation in a patient with osteomyelitis is concerning for aortic or mitral valve endocarditis. If endocarditis is present, complete (third-degree) AV block would be a more likely diagnosis. At this point no therapy is warranted as the junctional rhythm is stable. Even if the diagnosis were complete AV block, a temporary pacemaker would not be indicated, not only because of the absence of symptoms but also because of osteomyelitis and the possibility of endocarditis. Electrode catheters should not be inserted if there is an active infection because of the possibility of an infection occurring on the electrode catheter, which might result in a worsening of an underlying infection.

If clinically important, the etiology for the AV dissociation can be established with the use of atropine, which will increase the sinus rate but not affect the junctional rate. If there is capture with the increase in sinus rate, with consistent PR intervals, the mechanism for the isorhythmic dissociation would be established as an accelerated junctional rhythm. In contrast, if AV dissociation persisted with the increased sinus rate, the diagnosis would be complete heart block, as now the atrial rate would be faster than the junctional rate.
A 69-year-old man presents to the emergency department with complaints of substernal chest discomfort of 6 hours’ duration. An ECG (25A) is obtained. His serum troponin level is elevated, and a diagnosis of non-ST-segment elevation myocardial infarction is made. The patient is
brought to the catheterization laboratory, where coronary angiography shows a thrombus in the first obtuse marginal artery. A drug-eluting stent is placed and the patient is brought to the coronary care unit. He is asymptomatic. A second ECG (25B) is obtained routinely.

What is noted on the ECGs?
What is the etiology of the rhythm on ECG 25B?
Is any therapy necessary?
ECG 25A Analysis: Normal sinus rhythm, left anterior fascicular block, unifocal premature ventricular complexes, quadrigeminy
In ECG 25A, there is a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). As the QRS complexes have an rS morphology in leads II and aVF, this is a left anterior fascicular block. The QT/QTc intervals are normal (400/440 msec).

There are three premature QRS complexes (complexes 4, 8, and 12) (▲), occurring every fourth beat; this is often termed quadrigeminy. They have an increased duration (0.16 sec) and an abnormal morphology, with positive concordance across the precordium (tall R waves in leads V1-V6 [▲]). There is no distinct P wave before any of these QRS complexes, but there appears to be a P wave superimposed on the beginning of the eighth and 12th QRS complexes (▲). The P wave is not conducted as there is really no PR interval. Hence the wide and premature QRS complexes are premature ventricular complexes. Although there are no acute changes associated with a myocardial infarction, this is often the case with a lesion of the left circumflex artery or the obtuse marginal branch of the left circumflex artery. Infarction involving this vessel is not uncommonly electrically silent.

continues
Podrid's Real-World ECGs

ECG 258 Analysis: Normal sinus rhythm, isorhythmic AV dissociation, ventricular rhythm
ECG 25B was obtained shortly after reperfusion of the first obtuse marginal artery. There is a regular rhythm at a rate of 75 bpm. The QRS complexes have the same duration and morphology as the premature ventricular complexes in ECG 25A. There is positive concordance across the precordium (tall R waves in leads V1-V6) (→), similar to the morphology of the premature ventricular complexes in ECG 25A. Also noted is a slight difference in the morphology of the QRS complexes in lead V1 (▲). Lastly, there are P waves noted before some of the QRS complexes (●) but the PR interval is not constant. Some of the P waves are also superimposed at the beginning of the QRS complex (▲), which is evident by the slight widening of the initial portion of the QRS complex. Hence the P waves and QRS complexes are independent and there is AV dissociation. The presence of AV dissociation, the positive concordance across the precordium, the slight variability of the QRS complex morphology noted in lead V1, and the fact that the QRS complex is identical to the premature ventricular complexes seen in ECG 25A establish the etiology as a ventricular rhythm. The PP intervals are regular (●) and the atrial rate is 75 bpm, which is identical to the ventricular rate. Hence this is isorhythmic AV dissociation and since the atrial and ventricular rates are identical it is not clear whether this is complete AV block with an escape ventricular rhythm or an accelerated idioventricular arrhythmia. However, based on the clinical scenario (i.e., a non-ST-segment elevation myocardial infarction with reperfusion [stenting]), the most likely diagnosis is an accelerated idioventricular rhythm (AIVR), which is a reperfusion arrhythmia that often occurs after a percutaneous intervention or thrombolytic therapy for an acute myocardial infarction. An AIVR is usually benign and no therapy is typically required. However, if the ventricular rate were to accelerate and be associated with symptoms, then the ventricular arrhythmia should be suppressed with an antiarrhythmic, such as intravenous lidocaine, procainamide, or amiodarone. Therapy would likely be for a limited time as this arrhythmia is usually self-limited. ■
An asymptomatic 58-year-old man presents for a routine physical. His review of symptoms, physical exam, and general laboratory evaluation are all normal. A routine ECG is obtained.

What abnormality is depicted?
ECG 26 Analysis: Normal sinus rhythm, left anterior fascicular block (LAFB)
There is a regular rhythm at a rate of 78 bpm. There is a P wave (*) before each QRS complex with a stable PR interval of 0.18 second. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complexes are of a normal width (0.08 sec) and morphology. The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). There are two reasons for an extreme left axis. The first is an inferior wall myocardial infarction (MI) (or a pseudo inferior wall MI, as seen in Wolff-Parkinson-White pattern), in which there are Q waves and a QS or Qr morphology in leads II and aVF. The second is a left anterior fascicular block (LAFB), also called a left anterior hemiblock, in which there are rS complexes in leads II and aVF. In this case, the left axis is due to an LAFB. The QT/QTc intervals are normal (380/430 msec).

The left bundle consists of two major fascicles (left anterior and left posterior) as well as a small septal branch. The concept of the hemi, or fascicular, block was put forth by Rosenbaum and colleagues in 1970. The definition of a fascicular block was based on the electrical axis of the heart in the frontal plane. An LAFB results in all of ventricular activation coming from the left posterior fascicle. The direction of the impulse is up and to the left. Hence the axis is extremely leftward (>−30°). Some authors consider an extreme left axis as >−45° or >−60°. A fascicular block results in an axis shift but does not cause any widening of the QRS complex. A widened QRS complex indicates the presence of an intraventricular conduction delay in addition to the fascicular block or might be an associated right bundle branch block.

Other causes for a left axis need to be excluded, primarily an inferior wall MI (which is also associated with a positive QRS complex in lead I and a negative QRS complex in leads II and aVF). The difference is related to the initial force. With an inferior wall MI the initial wave of the QRS complex is a Q wave (Qr or QS complex), while with an LAFB the initial wave is an R wave (rS complex). Another cause would be lead misplacement. However, in this situation the P wave would also be negative in leads II and aVF.

Also noted is a small Q wave in lead V2 (†). Although this might be considered evidence of an anteroseptal MI, this pattern may be seen with conduction disease of the septal (median) branch that originates from the left bundle and is responsible for activation of the septum, which is the first part of the left ventricle to depolarize. The direction is from left to right, accounting for small initial R waves in leads V1-V2. The presence of Q waves suggest that in these leads the initial septal activation is from right to left, which is abnormal. This abnormality, along with the presence of an LAFB, may be predictive of the occurrence of a full left bundle branch block in the future.

The most common reason for an LAFB in the absence of heart disease is Lenègre's disease, which is a fibrotic sclerodenerative disease. LAFB in the absence of apparent organic heart disease and not associated with block in the other fascicles is usually benign and infrequently progresses to bifascicular disease or complete heart block. There is no reason for any additional workup or therapy.
A 72-year-old man is brought to the emergency department after he suddenly lost consciousness while getting out of bed. The syncopal episode was observed by his wife, who called 911. The man states that the episode was preceded by a feeling of lightheadedness and weakness. He also reports that he has had abdominal cramps associated with diarrhea, nausea, vomiting, and anorexia for the past 2 days, during which time he has been unable to take in anything orally. When emergency medical services arrived, he was awake but diaphoretic and nauseated. In the emergency department he relates a history of a previous myocardial infarction (MI) but no other cardiac or medical problems. His current medications are aspirin, simvastatin, low-dose lisinopril, and low-dose metoprolol. The physical exam is unremarkable; his blood pressure is 110/60 mm Hg. Laboratory data are unremarkable, except for a blood urea nitrogen level of 45 mg/dL and a creatinine level of 1.3 mg/dL. An ECG is obtained.

What abnormality is seen?
Does the ECG suggest an etiology for the syncopal episode?
Podrid's Real-World ECGs

ECG 27 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), right bundle branch block, left axis, old inferior wall MI
There is a regular rhythm at a rate of 84 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.28 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with first-degree AV block (prolonged AV conduction). The QRS complex duration is prolonged (0.14 sec), and it has a right bundle branch block (RBBB) morphology with an RSR' complex in lead V1 (→) and a broad terminal S wave in leads I and V5-V6 (←). The QT/QTc intervals are prolonged (420/500 msec) but are normal when the prolonged QRS complex duration is considered (360/430 msec).

The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). While the axis is consistent with a left anterior fascicular block (LAFB), the QRS complex has a QS morphology in leads II and aVF (↓), which is indicative of an old inferior wall myocardial infarction (MI). In contrast, a left anterior fascicular block has an rS morphology in leads II and aVF. Hence the left axis is not the result of a conduction abnormality but rather is due to an infarction.

The ECG suggests trifascicular conduction disease (ie, RBBB, left axis, and first-degree block). However, the left axis is due to an inferior wall MI and not a conduction abnormality; hence this is not trifascicular disease. However, if trifascicular disease were present, the occurrence of syncope would suggest a more advanced conduction abnormality (ie, complete heart block with an escape ventricular rhythm). In this situation, further evaluation of the conduction system would be indicated. However, the presence of first-degree AV block in association with bifascicular disease (ie, RBBB and LAFB, if present) does not necessarily imply trifascicular disease as the prolonged PR interval might reflect an AV nodal conduction abnormality or slowing of conduction in the remaining fascicle (ie, the left posterior fascicle). In this case, since the left axis is not the result of an LAFB, the only definite conduction abnormality of the His-Purkinje system is an RBBB, while the first-degree AV block might be AV nodal or involve the remaining fascicles. This makes syncope from complete heart block far less likely. Indeed, the history strongly suggests that the patient was dehydrated as a result of 2 days of diarrhea, vomiting, and decreased oral intake. Further support for a diagnosis of volume depletion is the elevated blood urea nitrogen level, which is out of proportion to the serum creatinine level. The syncopal episode following a change in position is consistent with hypotension or perhaps a vasovagal episode as the etiology for the syncope.
An asymptomatic 58-year-old man presents for a routine checkup. His vital signs, physical exam, and general laboratory evaluation are all normal. An ECG is obtained.

What abnormality is depicted?
Podrid's Real-World ECGs

ECG 28 Analysis: Normal sinus rhythm, left posterior fascicular block
The rhythm is regular at a rate of 62 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex is of normal duration (0.08 sec) and morphology. The QT/QTc intervals are normal (360/370 msec). The QRS complex is negative in lead I and positive in lead aVF. Hence the axis is rightward (between +90° and +180°). There are several etiologies for a right axis, including:

- Lateral wall infarct pattern, that is, an initial Q wave (Qr or QS) in leads I and aVL
- Right ventricular hypertrophy, which will be associated with a tall R wave in lead V1 and evidence of right atrial hypertrophy or abnormality (P pulmonale)
- Right-left arm lead switch, which is associated with negative P and T waves in leads I and aVL and positive P and T waves and positive QRS complexes in lead aVR
- Dextrocardia, in which there are inverted P and T waves in leads I and aVL and reverse R-wave progression across the precordium
- Right bundle branch block with a deep and broad S wave in lead I may be diagnosed as a right axis. In this situation the terminal S wave, which reflects delayed right ventricular activation, should be ignored as it is not reflective of left ventricular activation and hence not considered as part of axis determination.

- Wolff-Parkinson-White (WPW) pattern, which will be associated with a short PR interval and widened QRS complex resulting from a delta wave
- Biventricular pacemaker, which will have a QS or deep Q wave in lead I. Most often an indeterminate axis will be seen. With a pacemaker, pacemaker stimuli will be seen.
- Left posterior fascicular block (LPFB), with all left ventricular forces originating from the left anterior fascicle and directed down and to the right. This diagnosis is made when all other causes for a right axis have been excluded.

In this tracing, the QRS complexes have an initial R wave in leads I and aVL (rS complex), and there is no evidence for right ventricular hypertrophy, arm lead switch, dextrocardia, lateral infarction, WPW pattern, right bundle branch block, or pacemaker. Hence the right axis is due to an LPFB.

The left bundle divides into two major fascicles that innervate the left ventricle. The left anterior fascicle crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle. The left posterior fascicle appears as an extension of the main bundle and fans out extensively posteriorly toward the papillary muscle and inferoposteriorly toward the free wall of the left ventricle. There is often a third, smaller fascicle that is a septal branch (median fascicle) that innervates the interventricular septum, which
is the first part of the left ventricle to be activated (in a left-to-right direction). The left posterior fascicle is exposed to lower pressures and less turbulence than the left anterior fascicle; it also has a dual blood supply. These characteristics probably explain why isolated LPFB is an uncommon finding. Isolated LPFB can, however, be seen in the setting of extensive arteriosclerotic cardiovascular disease, as an association with inferior wall myocardial infarction and extensive coronary disease with diffuse myocardial fibrosis. LPFB can also occur with cardiomyopathies, hypertension with hypertrophy, myocarditis, hyperkalemia, acute cor pulmonale and, perhaps most commonly, chronic degenerative and fibrotic processes of the conducting system. An LPFB does not cause widening of the QRS complex, only a shift in axis. Hence any QRS widening is due to an associated intraventricular conduction delay or a right bundle branch block.

Isolated LPFB does not require any additional evaluation or therapy. However, additional evaluation may be required if there is evidence of trifascicular disease.
A 54-year-old man presents to his primary doctor as a new patient. He has never had any meaningful medical care in the past. He comes today with complaints of shortness of breath with everyday activities that has progressed to the point that he cannot ascend a flight of stairs or even a slight grade incline without taking frequent rests to catch his breath. His review of systems is notable for a 40-pound unintentional weight loss over the past year. He has no known past medical history and does not take any medications. His family and social histories are remarkable for 35-year employment as a coal miner. He smoked cigarettes regularly for 20 years but quit 10 years ago. On exam, he appears emaciated. His heart rate is 90 bpm, his blood pressure is 92/60 mm Hg, and his lips are faintly blue. His head, ears, eyes, nose, and throat exam is notable for temporal wasting. His lungs are hyperinflated with poor air movement. His jugular venous pressure is 10 cm H₂O with taller CV waves. His precordium demonstrates a right ventricular lift. His point of maximal impulse is nondisplaced. His rhythm is regular, with a normal S1 and prominent P2. A respirophasic holosystolic murmur is noted at the right lower sternal border.

His abdominal, extremity, and neurologic exams are normal. Arterial blood gases confirm hypoxemia and hypercapnia. An ECG is obtained as part of his evaluation.

What abnormalities are noted?
To what cardiopulmonary process do these abnormalities point, as suggested by his exam and history?
ECG 29 Analysis: Normal sinus rhythm, intraventricular conduction delay, right axis, right atrial hypertrophy (abnormality), P pulmonale, right ventricular hypertrophy (RVH) with ST-T wave changes, premature atrial complex, left ventricular hypertrophy (LVH) with ST-T wave changes, biventricular hypertrophy
The rhythm is regular at a rate of 90 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The P waves are very tall in leads II and aVF and also positive and tall in leads V1-V2; this is characteristic of right atrial hypertrophy or abnormality (P pulmonale). Given the height of the P waves in leads V1-V2, the term “Himalayan P waves” has been used. The QRS duration is slightly increased (0.12 sec), and there is an intraventricular conduction delay. The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). The QT/QTc intervals are prolonged (440/540 msec) even when the slightly prolonged QRS complex duration is considered (420/490 msec). There is a tall R wave (+) in lead V1. When associated with a right axis and P pulmonale, the tall R wave in lead V1 is indicative of right ventricular hypertrophy (RVH). Hence the right axis is the result of RVH and is not due to a left posterior fascicular block.

In addition, the S-wave depth in lead V2 is 26 mm [ ] , and the R-wave amplitude in lead V4 is 22 mm [ ] S-wave depth in lead V2 + R-wave amplitude in lead V4 = 48 mm), meeting the voltage criteria for left ventricular hypertrophy (LVH; S-wave depth + R-wave amplitude in any two precordial leads ≥ 35 mm). Therefore, there is evidence of biventricular hypertrophy. There are also ST-T wave abnormalities in leads V1-V3 (*), likely the result of RVH, while the ST-T wave abnormalities in leads V4-V6 (▲) are likely the result of LVH. The 10th QRS complex (¶) is early, is preceded by a premature P wave (+), and has the same QRS complex morphology as the sinus complexes. Hence it is a premature atrial complex.

This patient has signs, symptoms, and ECG evidence of RVH, the result of chronic obstructive lung disease from coal dust exposure as well as cigarette smoking. There is also evidence on the physical exam of tricuspid regurgitation (respirophasic holosystolic murmur and tall CV waves of the jugular pulsation). The patient should have an echocardiogram to evaluate pulmonary artery and right-sided pressures as well as left ventricular function. Initial therapy for pulmonary arterial hypertension as well as chronic obstructive lung disease consists of oxygen therapy. As there is no clinical evidence for fluid accumulation, diuretics are not needed. More advanced therapy includes treatment with prostanoids such as epoprostenol, endothelin receptor antagonists, phosphodiesterase 5 inhibitors such as sildenafil, or certain calcium-channel blockers (eg, a dihydropyridine or diltiazem). Patients with pulmonary hypertension who are selected for advanced therapy should undergo an invasive hemodynamic assessment prior to the initiation of advanced therapy. A vasoreactivity test with intravenous adenosine, intravenous epoprostenol, or inhaled nitric oxide is often performed at the time of hemodynamic assessment. The LVH may be the result of previous hypertension, although there is no clear history for this.
A 68-year-old diabetic woman presents to her primary care physician as a new patient. She states that “she had some heart trouble in the past” but is unaware of any specific diagnosis. She has stopped taking all medications that were previously prescribed. She declares, “No, I’m doing fine!” to all questions on a review of systems. Her vital signs and exam are unremarkable. As part of her evaluation, an ECG is performed.

What abnormalities are noted on the ECG, and to what pathology do they point?
Podrid's Real-World ECGs

ECG 30 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), right axis, old lateral wall myocardial infarction (MI), old anterior wall MI, left atrial hypertrophy (abnormality)
There is a regular rhythm at a rate of 86 bpm. There is a P wave (*) before each QRS complex with a stable PR interval of 0.22 second. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with first-degree AV block (prolonged AV conduction). However, the P waves are broad in leads II and aVF (*), suggesting left atrial hypertrophy or abnormality. The QRS complex duration is normal (0.10 sec). The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). However, the right axis is a result of a Q wave (—) in leads I and aVL (Qr morphology). This is, therefore, a lateral myocardial infarction (MI) and not a left posterior fascicular block. In addition, there are Q waves (J) in leads V2-V5, indicating an anterior wall MI.

Although the patient does not provide any history to suggest a prior MI or its timing, the presence of Q waves indicates that the infarction is chronic or old. Although she claims a history of some heart problem in the past, she provides no specifics about this. Silent infarctions are not uncommon, especially in the diabetic patient. Her therapy should consist of aggressive risk factor modification, including aspirin; LDL-lowering therapy with statins; and good control of her diabetes. She is not having any symptoms, suggesting that there is no actual role for anti-ischemic medication, including β-blocker or nitrates. However, diabetic patients do have silent (discomfortless) ischemia, so exercise testing would be useful to document any ECG changes or symptoms that suggest underlying ischemia.
A 42-year-old man without prior medical diagnoses but with a strong family history of myocardial infarction is seen by his nurse practitioner for a routine evaluation. He has no complaints on review of systems. His exam
is normal. An ECG is obtained as part of his evaluation (31A). Upon viewing the tracing, the ECG technician becomes very concerned. The nurse practitioner then proceeds to repeat the ECG herself (31B).

**What abnormality was noted on the initial ECG (31A)?**

**What is revealed by the subsequent ECG (31B)?**
ECG 31A Analysis: Normal sinus rhythm, right axis, right-left arm lead switch, U waves
ECG 31A shows a regular rhythm at a rate of 62 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.14 sec). The QRS duration (0.08 sec) and morphology are normal. The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). However, the right axis is the result of a Q wave (+) in lead I and an rS complex in lead aVL. In addition, the P waves (+) and T waves (−) are inverted in these leads. The P wave and QRS complex are positive in lead aVR, which is abnormal. Although the ECG suggests that the right axis is a result of a lateral wall myocardial infarction (and not left posterior fascicular block), the presence of negative P waves in leads I and aVL means that neither of these conditions is present but that the right axis is a result of right-left arm lead switch. This is suggested by the fact that all of the waveforms in leads I and aVL are negative and the waveforms in lead aVR are positive; this is abnormal. The QT/QTc intervals are normal (420/426 msec). Also noted are low-amplitude U waves in leads V2-V5 (−).
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ECG 318 Analysis: Normal sinus rhythm, normal axis, normal PR interval, normal QRS complex, normal ECG
In ECG 31B, the leads are placed correctly and it can be seen that the axis is normal (positive QRS complex in leads I and aVF). The QRS complex has a normal morphology, and the P (+) and T (+) waves are normal in axis. The ECG is, therefore, normal.

Right-left arm lead switch is a common mistake made when recording the ECG. As the QRS complexes will be abnormal in leads I and aVL, a left posterior fascicular block or lateral wall myocardial infarction is often diagnosed in error. Hallmarks of lead switch, along with the abnormal QRS complex morphology, are negative P and T waves in leads I and aVF and a positive P and T waves and QRS complex in lead aVR, which are also abnormal. It should be remembered that dextrocardia will also present with the same P-wave, QRS complex, and T-wave abnormalities in leads I and aVL. However, there will also be abnormal QRS complexes in leads V1-V6, with reverse R-wave progression (R-wave amplitude progressively decreases).
A 70-year-old woman presents to the emergency department with complaints of fever, chills, burning with urination, and left-sided costovertebral tenderness. Her temperature is 103°F. A urinalysis shows

ECG 32A
white blood cells and many bacteria. She is diagnosed with pyelonephritis, appropriate antibiotics are initiated, and an ECG is obtained (ECG 32A). She is afebrile on the following day, and another ECG is obtained (ECG 32B).

What is the difference between the two ECGs? What is the etiology of the abnormalities?
ECG 32A Analysis: Sinus tachycardia, premature ventricular complex, rate-related intraventricular conduction delay, rate-related left anterior fascicular block, long QT interval, diffuse T-wave abnormalities.
In ECG 32A, there is a regular rhythm at a rate of 100 bpm. There are P waves (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia. The QRS complex duration is prolonged (0.12 sec). There is an RSR' complex in lead V1 (+−), but there are no terminal S waves in leads I or V5-V6. Thus while the morphology is suggestive of a right bundle branch block, it does not have a typical morphology. Therefore, it is an intraventricular conduction delay to the right ventricle. The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). As the QRS complex does not have a QS or Qr morphology in leads II and aVF, the left axis is not due to an inferior wall myocardial infarction. This is a left anterior fascicular block. The QT/QTc intervals are prolonged (400/520 msec), even when the prolonged QRS complex duration is considered (380/490 msec). There are diffuse T-wave abnormalities (↑) seen in leads I, aVL, and V1-V6.

The 10th QRS complex is premature (↓). It is not preceded by a P wave, and it has a wider duration and an abnormal morphology. Hence this is a premature ventricular complex. Following this premature complex there is a full compensatory pause (+−) (ie, the PP interval around the premature complex is equal to two PP intervals). Following the premature ventricular complex is a narrow complex (↑) that is preceded by an on-time P wave (+). Unlike the other QRS complexes, it has a normal duration (0.10 sec) and does not have an intraventricular conduction delay. The axis is probably normal (ie, positive QRS complex in lead II). Hence it appears that there is a rate-related intraventricular conduction delay and a rate-related left anterior fascicular block, since after the pause and with a slower rate (longer RR interval) the conduction abnormalities are no longer present.

Continues...
Podrid's Real-World ECGs

ECG 32B Analysis: Normal sinus rhythm, diffuse T-wave abnormalities
In ECG 32B, obtained on the day after admission when the patient was afebrile, there is a regular rhythm at a rate of 60 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.18 sec), the same as the PR interval seen in ECG 32A. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec). However, there are deep S waves seen in leads II and aVF (–), suggesting a slight conduction delay. Despite these S waves, the QRS axis is normal at about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF). The QRS complex in ECG 32B has the same morphology as the QRS complex that follows the premature ventricular complex in ECG 32A. The absence of an intraventricular conduction delay and left anterior fascicular block at the slower heart rate confirms the fact that they were rate related.

Although a rate-related right or left bundle branch block is more commonly seen, a rate-related intraventricular conduction delay and a rate-related left anterior fascicular block do occur, although the latter are not common. As with rate-related bundles, the presence of a rate-related fascicular block likely indicates underlying disease of this fascicle and the potential for a permanent left anterior fascicular block in the future.
A 76-year-old man presents to his cardiologist with complaints of intermittent fatigue, during which time he notes that his pulse rate is slow. He denies any previous cardiac history except for hypertension, which is being treated with an angiotensin-converting enzyme inhibitor. His physical exam is unremarkable, but his pulse is noted to be slow and slightly irregular. His blood pressure is 180/100 mm Hg. An ECG is obtained.

What abnormality is noted?
Is any therapy needed?
ECG 33 Analysis: Sinus bradycardia, accelerated AV conduction, premature atrial complexes, premature junctional complexes, escape junctional complexes, rate-related left posterior fascicular block, left ventricular hypertrophy
The rhythm is irregularly irregular. There are two different QRS complexes, but they both have a normal duration (0.10 sec). The first three QRS complexes (*) have P waves before them (*), and the PR interval is the same in each, but short (0.12 sec). These QRS complexes have a rate of 50 bpm. The P waves are positive in leads I, II, and aVF. This is, therefore, sinus bradycardia with accelerated AV conduction. The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). As the QRS complexes in leads I and aVL have an rS morphology, this is not a lateral infarction. The positive P waves in leads I and aVL eliminate lead switch as the cause. There is no evidence for right ventricular hypertrophy, pacemaker activity, or Wolff-Parkinson-White pattern. Hence, this is a left posterior fascicular block. The fourth QRS complex (+) is premature, and it is preceded by a P wave (●). Although the P wave looks similar to the sinus P waves, the PR interval is slightly longer (0.14 sec) and this is a premature atrial complex. The QRS complex that follows (complex 5) (▲) has the same morphology but is not preceded by a P wave. The RR interval of this complex is shorter than that of the sinus complexes; this is, therefore, a premature junctional complex. The next two complexes (complexes 6 and 7) (†) occur after a pause of 1.32 seconds at a rate of 46 bpm. There is a P wave before both of these QRS complexes (▼), but the PR interval is very short, and shorter than the sinus complexes. In addition, the two PR intervals are different from each other. Hence these two complexes are not conducted. The QRS complex has the same duration as the sinus complexes; therefore, these are escape junctional complexes. The axis is different from what was seen with the conducted sinus complexes. The axis cannot be established as the junctional complexes are not seen in leads II and aVF. However, the axis is certainly not rightward, as the QRS complex is positive in lead I. It is likely to be a normal axis. The 8th QRS complex (★) is premature and is preceded by a P wave. Hence this is a premature atrial complex with a QRS duration, morphology, and axis that are the same as the first sinus complexes. The last QRS complex (++) is identical in duration, morphology, and axis to the first three sinus complexes. There is no P wave before it and hence this is a premature junctional complex. The QRS complexes have an increased voltage, with an S-wave amplitude in lead V2 of 34 mm (●); this meets a criterion for left ventricular hypertrophy (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The QT/QTc intervals are normal (500/440 msec).
It can be seen that the QRS complexes with a left posterior fascicular block always have a shorter RR interval (faster rate) compared with the two QRS complexes without a left posterior fascicular block (i.e., complexes 6 and 7). Hence there is a rate-related left posterior fascicular block.

Although rate-related left and right bundle branch blocks are more common, a rate-related fascicular block may also occur as a result of conduction slowing present in only one of the fascicles from the left bundle. As with other rate-related conduction abnormalities of the bundles, the presence of a rate-related fascicular block may predict the development of a permanent fascicular block in the future.

In this patient it is not clear if the symptom of fatigue is related to the slow heart rate or the conduction abnormalities. One concern is the presence of significant hypertension, which may be associated with symptoms, including fatigue. Better blood pressure management is important. Although there is sinus bradycardia as well as evidence of junctional complexes, there is no definite indication for a pacemaker.
The patient is a 14-year-old girl with a history of congenital heart disease, which she states required surgery when she was 8 years old. Review of her records confirms that she has an ostium primum atrial septal defect associated with an endocardial cushion defect and a cleft mitral valve.

What is the QRS complex axis on the ECG?
ECG 34 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), right atrial hypertrophy (P pulmonale), right ventricular hypertrophy (RVH) with associated ST-T wave changes, intraventricular conduction delay, indeterminate axis (due to left anterior fascicular block associated with a right axis due to RVH)
There is a regular rhythm at a rate of 94 bpm. (Note that the rhythm strip is not simultaneous with the 12-lead recording.) There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The P wave is tall and peaked in lead II and primarily positive in lead V1; this is consistent with right atrial hypertrophy (or abnormality), termed P pulmonale. The QRS complex has a widened duration (0.12 sec), without any pattern of a right or left bundle branch block. Hence this is an intraventricular conduction delay. The R wave is tall in leads V1 (23 mm) and V2 (33 mm). In addition, the R/S ratio is less than 1 in leads V5-V6 (ie, the S-wave depth is greater than the R-wave amplitude). These features are consistent with a diagnosis of right ventricular hypertrophy (RVH, ie, R-wave amplitude in lead V1 > 7 mm or R/S ratio in lead V1 > 1 and R/S ratio in lead V6 < 1). Associated with the hypertrophy are ST-T wave abnormalities in leads V1-V3 (▲). The RVH likely is the cause of the intraventricular conduction delay. The QT/QTc intervals are normal (320/410 msec). The axis is indeterminate, between -90° and +/-180° (negative QRS complex in leads I and aVF). This may be either an extreme right or extreme left axis.

An indeterminate axis is not uncommonly seen with a wide QRS complex, and in this situation typically reflects direct activation of the ventricular myocardium, as with a ventricular complex, a paced complex (particularly biventricular pacing), or Wolff-Parkinson-White pattern. However, an indeterminate axis is uncommon when there is a supraventricular and narrow QRS complex and typically results from an underlying conduction abnormality (ie, left anterior or left posterior fascicular block associated with another myocardial abnormality that causes an axis shift). For example, if there is an underlying left anterior fascicular block, the presence of RVH (which shifts the axis rightward) or a lateral wall myocardial infarction (with deep Q waves in leads I and aVL) can result in an indeterminate axis. The presence of a left posterior fascicular block associated with an old inferior wall myocardial infarction (with deep Q waves in leads II and aVF) is also associated with an indeterminate axis. Other combinations include an old inferior wall myocardial infarction with RVH and a right axis or both an inferior and lateral wall myocardial infarction.

Patients with an ostium primum atrial septal defect and endocardial cushion defect often have a congenital left anterior fascicular block and hence an extreme left axis. With left-to-right shunting and the development of RVH, the axis shifts rightward, resulting in an indeterminate axis. Therefore, the indeterminate axis in this patient is the result of a left anterior fascicular block with a right axis due to RVH.
A 76-year-old man with a long history of chronic obstructive pulmonary disease presents for a routine physical exam. He states that he had a previous myocardial infarction (MI) but is unable to provide any additional details.

His physical exam demonstrates decreased breath sounds and significant wheezes. His cardiac exam is normal. Arterial blood gases include an $O_2$ saturation of 86% on room air. He denies using supplemental oxygen at home. An ECG is obtained.

What does the ECG show? What is the QRS complex axis?
ECG 35 Analysis: Normal sinus rhythm, old inferoposterior wall MI, left posterior fascicular block, indeterminate axis, counterclockwise rotation (early transition)
There is a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec). There are significant Q waves in leads II, III, and aVF (+-), which are diagnostic for an old inferior wall myocardial infarction (MI). There is a tall R wave in lead V1 (-+), which, in association with an inferior wall MI, is consistent with posterior wall involvement (or a posterior wall MI). There is also a tall R wave in lead V2, likely due to early transition or counterclockwise rotation of the electrical axis in the horizontal plane. This is established by imagining the heart as viewed from under the diaphragm. The right ventricle is anterior and the left ventricle is lateral. With counterclockwise rotation the left ventricular forces are shifted anteriorly and hence develop earlier in the right precordial leads, producing the tall R wave in lead V2. The QT/QTc intervals are normal (360/395 msec).

The axis in the frontal plane is indeterminate, between –90° and +/-180° (negative QRS complex in leads I and aVF). The QRS complex morphology in lead I is due to an rS complex, and this morphology is the result of a left posterior fascicular block. However, the negative QRS complex in leads II and aVF is the result of an old inferior wall MI (Qr complex). Therefore, the indeterminate axis is due to a left posterior fascicular block associated with a myocardial abnormality (a chronic inferior wall MI).
A 61-year-old man with longstanding multidrug-refractory essential hypertension is seen by a cardiologist. He states that he feels well and has been compliant with his medications, although on further questioning admits to some fatigue over the past few months. He denies dyspnea and angina. His exam is notable for a blood pressure of 152/88 mm Hg. As part of his evaluation, an ECG is obtained.

What abnormalities are noted?
ECG 36 Analysis: Atrial fibrillation, counterclockwise rotation (early transition), left ventricular hypertrophy
The rhythm is irregularly irregular (average rate of 78 bpm), and no organized atrial activity is seen. Rapid and irregular low-amplitude undulations of the baseline can be seen in leads II, III, aVF, and V1 (*). These are fibrillatory waves; therefore, the underlying rhythm is atrial fibrillation. The QRS complex duration is normal (0.08 sec). The axis is leftward, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). Thus this is a physiologic left axis, which is a normal variant or may be seen with increased age or left ventricular hypertrophy (LVH). Borderline LVH is present based on an R-wave amplitude in lead V4 () of 26 mm (S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm is a criterion for LVH). Also noted is a tall R wave in lead V2 (+), which is termed early transition or counterclockwise rotation in the horizontal plane. The QT/QTc intervals are normal (340/390 msec).

In addition to the electrical axis of the heart in the frontal plane (normal, left, right, indeterminate), there is also an electrical axis in the horizontal plane, which is established by imagining the heart as viewed from under the diaphragm. From this direction, the right ventricle is in front and the left ventricle is to the left. When the heart is electrically rotated in a counterclockwise direction, the left ventricular forces appear earlier in the precordial leads, and hence there is early transition with the R wave being tall in lead V2. With clockwise rotation, the left ventricular forces are generated late, resulting in poor R-wave progression and late transition. It is important not to confuse the tall R wave in lead V2 as indicating right ventricular hypertrophy or a posterior wall myocardial infarction. In these situations, a tall R wave is also seen in lead V1 and not just in lead V2. The presence of counterclockwise rotation, which is only an electrical axis shift, has no clinical implications except for not confusing it with any other abnormality.
A 22-year-old woman presents to her primary care physician for a routine physical exam prior to joining a college basketball team. An ECG is obtained, raising questions about an abnormality that suggests there may have been a previous anterior wall myocardial infarction.

What is the abnormality?
Podrid's Real-World ECGs

ECG 37 Analysis: Sinus tachycardia, clockwise rotation (late transition), poor R-wave progression
There is a regular rhythm at a rate of 130 bpm. There is a P wave (*) before each QRS complex with a stable PR interval of 0.16 second. The P wave is upright in leads I, II, aVF, and V4-V6. Therefore, this is sinus tachycardia. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). There is low voltage in the limb leads (QRS complex amplitude < 5 mm in each lead). The QT/QTc intervals are normal (300/440 msec). Poor R-wave progression is noted in leads V1-V3 (the R-wave amplitude does not become progressively taller), and there is also late transition (R/S > 1) in lead V6 rather than in leads V3-V4. This is also known as clockwise rotation in the horizontal plane.

In addition to the electrical axis of the heart in the frontal plane, there is an electrical axis in the horizontal plane, which is established by imagining the heart as viewed from under the diaphragm. From this direction the right ventricle is in front and the left ventricle is to the left. When the heart is electrically rotated in a clockwise direction in the horizontal plane, the left ventricular forces appear late and are seen in the lateral precordial leads. This results in poor R-wave progression and late transition. Poor R-wave progression is a normal variant and should not be confused with an anterior wall myocardial infarction, in which there are Q waves or QS complexes in the precordial leads and no initial R waves. With clockwise rotation there are normal anterior forces (ie, R waves), but their progression is delayed or slow. Clockwise rotation can be seen with a left anterior fascicular block or with significant lung disease. Women may have poor R-wave progression as a result of breast tissue attenuation of anterior forces.

Conduction Abnormalities: Core Case 37
A 38-year-old woman with metastatic breast cancer associated with a BRCA mutation is admitted for progressive shortness of breath and is found to have multiple, bilateral pulmonary emboli. She is hemodynamically stable, and her cardiopulmonary exam is normal. Her laboratory values show a mild elevation in brain natriuretic peptide of 400 pg/mL but undetectable troponin. An ECG is obtained.

What is the abnormality? How is the abnormality related to the woman’s current diagnosis? What further evaluation is needed?
Podrid's Real-World ECGs

ECG 38 Analysis: Normal sinus rhythm, right bundle branch block with associated T-wave abnormalities
There is a regular rhythm at a rate of 64 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (0.15 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS duration is increased (0.14 sec). The QT/QTc intervals are normal (410/420 msec and 350/360 msec when the increased QRS complex duration is considered). The QRS complex in lead V1 has an RSR' morphology (→). The initial 0.08 second of the QRS complex is normal and the increased QRS duration is a result of the R', which represents a delay in right ventricular activation, with the terminal forces being directed from left to right (toward lead V1). Leads I and V5-V6 have broad S waves (←) that also represent the terminal forces being directed from left to right (away from these leads), a result of delayed right ventricular activation. This is a pattern that indicates the presence of a right bundle branch block (RBBB). There are T-wave changes (*) in leads V1-V2 that are due to the RBBB. Although the R' in lead V1 is very tall in amplitude, a diagnosis of right ventricular hypertrophy cannot be made on the ECG in the presence of an RBBB as right ventricular activation is abnormal, not occurring through the normal His-Purkinje system but rather directly through the right ventricular myocardium.

An RBBB is identified by the following characteristics:

- The QRS complex duration is 0.12 second or longer due to delayed activation of the right ventricle.
- Delayed right ventricular activation occurs directly through the right ventricular myocardium originating from the left bundle and left ventricle. Right ventricular activation, therefore, bypasses the normal His-Purkinje system. The terminal forces of the QRS complex are directed from left to right. Since conduction velocity is slow (due to direct myocardial conduction) the terminal forces are delayed, accounting for the widened QRS complex. Hence there is an RSR' complex in leads V1-V2 (due to delayed right ventricular forces directed toward the right-sided leads V1-V2) and broad S waves in leads I and V5-V6 (due to delayed right ventricular forces directed away from the left).
- Right ventricular repolarization is also abnormal, and secondary ST-T wave changes are often seen in leads V1-V3.
- Since right ventricular activation is abnormal, right ventricular hypertrophy cannot be recognized.
- Since left ventricular activation is normal, the initial portion of the QRS complex (first 0.08 sec) is normal. Abnormalities affecting the left ventricle can be recognized (eg, left ventricular hypertrophy, infarction, ischemia, pericarditis).

continues
• An RBBB may also be associated with conduction abnormalities of the fascicles of the left bundle (i.e., a left anterior or left posterior fascicular block). This is established by the presence of an extreme left or right axis in addition to the RBBB. The presence of an RBBB and fascicular block is termed bifascicular disease.

• An RBBB may be intermittent or related to increased heart rate (rate-related RBBB).

• A QRS complex duration of 0.10 to 0.12 second in conjunction with an RBBB-like morphology has been termed an incomplete RBBB. However, as conduction through the RBBB is all or none, it is more appropriate to term this an intraventricular conduction delay to the right ventricle.

There are multiple etiologies of isolated RBBB. Any entity that raises pulmonary pressures and induces geometric alteration of the right bundle may impair right bundle conduction. This is often in association with a rightward shift in the axis in the frontal plane. Cardiomyopathies, myocardial fibrosis from infarction, and myocarditis may also cause RBBB, generally with other ECG findings consistent with the underlying diagnosis. Idiopathic conduction system disease (Lev’s or Lenègre’s disease) may be isolated to the right bundle. In this case, chronic pulmonary emboli have likely raised pulmonary pressures to the point of right ventricular pressure overload (as evidenced by an elevated brain natriuretic peptide) but without physical findings of right ventricular hypertrophy or dilation (no parasternal lift on palpation, no murmur of tricuspid regurgitation). An echocardiogram would assist in evaluation of right ventricular morphology and pulmonary pressures.
A 48-year-old man with known obstructive sleep apnea presents to your clinic for routine evaluation. On review, the man admits that he is noncompliant with his continuous positive airway pressure (CPAP) machine as he is unable to sleep when wearing the mask. Due to severe dyspnea, his exercise capacity is severely diminished and he is limited to ambulating only within his home. He denies orthopnea or anginal symptoms. His heart rate is 100 bpm, and his blood pressure 162/110 mm Hg. His respiratory rate is 18 breaths/min, and his resting O₂ saturation is 94% on ambient air. His body mass index is 42. Cyanosis is absent. His lungs are clear. His jugular venous pressure is 14 cm H₂O with CV waves. His cardiovascular exam is notable for a precordial (right ventricular) lift, pulmonary artery tap, and a prominent P₂ component of S₂. A soft holosystolic murmur is noted at the left lower sternal border. Carvallo’s sign (respiratory variation in the intensity and duration of the murmur) is noted. His abdomen is protuberant without gross hepatomegaly. His lower extremities display mild pitting edema bilaterally. An ECG is obtained.

What abnormalities are shown? What is the underlying cause of these abnormalities? What further diagnostic testing may be helpful?
Podrid's Real-World ECGs

ECG 39 Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), right bundle branch block, left anterior fascicular block, bifascicular block
There is a regular rhythm at a rate of 100 bpm. There is a P wave (*) before each QRS complex with a stable PR interval of 0.24 second. The P wave is positive in leads I, II, aVF, and V4-V6 (although it is of very low amplitude in these leads). Hence this is sinus tachycardia with first-degree AV block (prolonged AV conduction). The complex duration is prolonged at 0.16 second, and the morphology is that of a right bundle branch block (RBBB; tall, broad R wave in lead V1 [+-] and broad S waves in leads I and V5-V6 [+-]). The QT/QTc intervals are prolonged (380/490 msec) but are normal when the prolonged QRS complex duration is considered (300/388 msec). There are associated ST-T wave changes (*) in leads V1-V3. The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). The QRS complex has an rS morphology; hence this is a left anterior fascicular block and not an inferior wall myocardial infarction, in which the QRS complex would have a QS or Qr morphology. The presence of an RBBB and left anterior fascicular block is termed bifascicular block. In addition, there is first-degree AV block, which is often called “trifascicular block,” implicating disease of all three fascicles. However, first-degree AV block may be the result of slow conduction through the AV node or the His-Purkinje system (ie, the remaining fascicle). Therefore, this should not be termed trifascicular disease. Trifascicular block can only be diagnosed on the ECG with either alternating RBBB and left bundle branch block (LBBB) or RBBB with alternating left anterior or left posterior fascicular block. Other situations in which trifascicular block can be diagnosed include (1) bifascicular block (ie, LBBB or RBBB with either left anterior or left posterior fascicular block) associated with Mobitz type II block or (2) bifascicular block with development of complete heart block with an escape ventricular rhythm.

Obstructive sleep apnea has been associated with advanced conduction system disease, and 70% of patients display a left anterior fascicular block. Other findings are persistent deep S waves in the lateral precordial leads and, less commonly, an RBBB (~5%). An RBBB may suggest pulmonary hypertension as a secondary phenomenon. In this patient, there is evidence on physical exam for significant pulmonary hypertension: a right ventricular lift (suggesting right ventricular enlargement), a pulmonary artery tap (suggesting enlargement of the pulmonary artery), a prominent P2, and a holosystolic murmur that demonstrates respirophasic changes in intensity (suggesting the presence of tricuspid regurgitation). Conduction system disease tends to progress in parallel with the severity of obstructive sleep apnea. An echocardiogram would be helpful in better delineating the extent of pulmonary hypertension and evaluating for right ventricular dysfunction suggested by the tricuspid regurgitation and peripheral edema. His tachycardia is quite concerning for incipient right ventricular failure.
An ECG of a 62-year-old man with known progressive, idiopathic conduction system disease is shown. What portions of the conduction system are affected based on this tracing?
ECG 40 Analysis: Sinus tachycardia, left atrial hypertrophy (abnormality), left posterior fascicular block, right bundle branch block with associated ST-T wave changes, bifascicular block
There is a regular rhythm at a rate of 100 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6, consistent with a sinus rhythm. However, the P waves are broad in leads II and aVF and notched in leads V4-V6. There is also a prominent negative component to the P wave in leads V1-V2. Hence this indicates left atrial hypertrophy or abnormality. The QRS duration is prolonged (0.16 sec), and there is a pattern of a right bundle branch block (broad R wave in lead V1 [→] and broad S wave in leads I and V5-V6 [←]) with associated ST-T wave changes in leads V1-V3 (*). The QT/QTc intervals are prolonged (380/490 msec) but are normal when the prolonged QRS complex duration is considered (300/388 msec). The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). The QRS complex in lead I is negative even when the S wave in lead I (due to the right bundle branch block) is ignored. When there is a right bundle branch block, there is a broad terminal S wave in lead I that should not be considered as part of axis determination as it reflects a terminal delay in right ventricular activation. The axis in the frontal plane is based on the impulse direction in the left ventricle. Hence there is also a left posterior fascicular block associated with the right bundle branch block. This is termed bifascicular disease or block.

The presence of bifascicular block indicates more advanced conduction system disease. Bifascicular block due to a left posterior fascicular block is less commonly seen than a left anterior fascicular block, but it has the same implications for the future development of complete heart block. While such patients are at increased risk for complete heart block, the incidence is low. In several studies, the incidence of progression to complete AV block was about 1% to 1.5% per year. Indeed, mortality in these patients, often due to arrhythmia, was related to the underlying heart disease and not to the development of complete heart block. There is no specific therapy for bifascicular block except for discontinuation or avoidance of drugs that may further impair cardiac conduction. Pacemakers are not indicated for asymptomatic patients. However, insertion of a pacemaker should be considered (class II indication) for patients with a bifascicular block associated with syncope that can be attributed to transient complete heart block, based on the exclusion of other plausible causes of syncope.
A 48-year-old man with no previous cardiac history presents to the emergency department after 2 hours of severe substernal chest discomfort that radiates to his jaw and left arm and is associated with diaphoresis and nausea. An ECG shows evidence of an acute myocardial infarction (MI). He is taken urgently to the cardiac catheterization laboratory where angiography demonstrates a thrombotic occlusion of one of the coronary arteries. The vessel is stented in an uncomplicated procedure, and the patient is brought to the critical care unit. He remains stable, and an ECG is obtained the following day.

What was the location of the acute MI?
What other abnormalities are shown?
Is additional therapy necessary?
ECG 41 Analysis: Normal sinus rhythm, acute anterior wall (septum and apex) MI, 2:1 right bundle branch block
There is a regular rhythm at a rate of 76 bpm. There are P waves (+) before each QRS complex with a stable PR interval (0.16 sec) (LJ). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complexes have two different morphologies that alternate in a repeating fashion. There are narrow complexes (duration, 0.08 sec) (*) that have a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). These QRS complexes have a QS morphology (ie, no initial R wave) in leads V1-V4 (!), which indicates an anterior wall myocardial infarction (MI) (septum and apex). There is also ST-segment elevation in these leads (^), indicating that the anterior wall MI is recent.

The alternate QRS complexes have a wide duration (0.16 sec) (†) with a qR morphology in lead V1 (<--) and a broad terminal S wave in leads I and V5-V6 (-->), typical for a right bundle branch block (RBBB). These QRS complexes also have a QS complex (▼) and ST-segment elevation (▲) in leads V1-V4, similar to what is seen with the narrow QRS complexes. Hence the alternating QRS complex (ie, normal conduction and RBBB) is termed 2:1 RBBB. Importantly, left ventricular abnormalities can be recognized in the presence of an RBBB since the initial depolarization via the left bundle remains intact and normal, while the QRS complex widening is the result of a terminal delay in right ventricular activation. Hence both QRS complexes demonstrate the same changes that are consistent with a recent (acute) anterior wall MI. The QT/QTc intervals are normal (360/405 msec).

The presence of a new bundle branch block associated with an anterior wall MI indicates significant damage to the septum, which is where the bundle of His and proximal portion of the bundles are located. As the right bundle is located on the right side of the septum, the occurrence of an RBBB generally indicates more extensive infarction. It is possible that the 2:1 RBBB is in fact rate related and would become persistent at a faster heart rate. It is also possible that the RBBB will become permanent in the future. The occurrence of an RBBB, whether intermittent or permanent, is not an indication for permanent pacemaker implantation. No additional therapy is necessary.
A 56-year-old woman is admitted to the hospital for upper gastrointestinal bleeding. While on telemetry, the nurse notices an abrupt change in the patient’s surface ECG tracing. He obtains a 12-lead ECG.

What abnormalities are noted?
ECG 42 Analysis: Normal sinus rhythm, left anterior fascicular block, intermittent right bundle branch block, clockwise rotation (late transition)
The rhythm is regular at a rate of 86 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.18 sec) (\(\text{\( n\)}\)). The P wave is positive in leads I, II, aVF, and V4-V6. Hence there is a stable normal sinus rhythm. There are two different QRS morphologies, both of which are preceded by a P wave that has the same morphology; the PR interval of both QRS complexes is the same. The narrow QRS complex (+) has a normal duration (0.08 sec) and morphology. The axis is extremely leftward, between \(-30^\circ\) and \(-90^\circ\) (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS complex). This is diagnostic for left anterior fascicular block.

Also noted are QRS complexes that are wide (\(\text{\( t\)}\)) (0.14 sec) with a typical right bundle branch block (RBBB) morphology (RSR' morphology in lead V1 [\(\text{\( \rightarrow\)}\]) and broad S wave in leads I and V5-V6 [\(\text{\( \rightarrow\)}\]). This is an intermittent RBBB that does not appear to be rate related. However, subtle differences in rate may be present but not apparent when the ECG is recorded at slow speed (ie, 25 mm/sec). The P wave and PR interval before both the wide and narrow QRS complexes are the same. Of note is the fact that the intermittent RBBB, associated with a persistent left anterior fascicular block, indicates bifascicular disease. Both QRS complexes show poor R-wave progression across the precordium and late transition as a result of clockwise rotation in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. In this situation, the right ventricle is in front and the left ventricle is to the left. With clockwise rotation the left ventricular forces are delayed and develop later, occurring in the lateral precordial leads. Clockwise rotation can be seen with a left anterior fascicular block. The QT/QTc intervals for the narrow QRS complexes are normal (320/380 msec). The QT/QTc intervals for the QRS complexes with an RBBB are prolonged (380/455 msec) but are normal (320/380 msec) when correcting for the prolonged QRS complex duration.

Intermittent or rate-related RBBB is indicative of underlying conduction disease of the right bundle that becomes manifest most often when the heart rate is faster. Occasionally there is no change in heart rate. The onset of a widened QRS complex due rate-related bundle branch block is abrupt (ie, the widening of the QRS complex is not gradual). When an intermittent RBBB is present, the diseased bundle is unable to conduct at rapid rates and hence block develops within the bundle. This is often the precursor of a permanent RBBB. It has the same implications and prognosis as a persistent RBBB and a persistent bifascicular block.
A medical student is concerned about his patient's admission ECG. He notices an abnormality that prompts review by the patient's physician.

What abnormality is noted on the ECG?
What does it represent?
ECG 43 Analysis: Normal sinus rhythm, intraventricular conduction delay to right ventricle (crista pattern)
The rhythm is regular at a rate of 68 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The axis is normal, between 0° and +90°, and the QRS duration (0.10 sec) and morphology are normal. The QT/QTc intervals are normal (380/405 msec). However, the QRS complex in leads V1-V2 has an RSR' morphology (+ -). This pattern represents a conduction delay to the right ventricle and has been called a "crista" pattern as the delay is in the last portion of the right ventricle to depolarize (ie, crista supraventricularis). It is a normal variant.

The RSR' morphology seen in the early precordial leads (V1-V2) represents a spectrum of physiologic changes ranging from normal delayed depolarization of the crista supraventricularis of the right ventricle, to right intraventricular conduction delay, to pathologic right bundle branch block. The QRS complex duration defines the diagnosis. If the QRS complex duration is normal (< 0.10 sec), the morphology is deemed a normal variant and termed a crista pattern. If the QRS complex duration is slightly prolonged (between 0.10 and 0.12 sec), an intraventricular conduction delay (often referred to as an incomplete right bundle branch block) is present. The term incomplete right bundle branch block is not accurate, however, as the His-Purkinje system and bundles manifest all-or-none conduction characteristics; that is, the bundles either conduct (always at the same rate) or do not conduct. If the QRS complex duration is longer than 0.12 second, the diagnosis is a right bundle branch block. In this ECG there is a crista pattern present, which is a normal variant that does not have any clinical implications.
An 88-year-old woman who was diagnosed with hypertension in her 20s but has refrained from medical treatment is seen in your clinic. She has complaints of progressive dyspnea on exertion that has started to interfere with her daily 5-mile walks. She is otherwise asymptomatic, and a general review of systems is unremarkable. Her heart rate is 70 bpm, with a blood pressure of 154/90 mm Hg. Her cardiopulmonary exam is notable for normal jugular venous pressure, a slight lateral displacement of a prominent point of maximal impulse, an S4 gallop, and a soft holosystolic murmur at the apex. An ECG is obtained.

What abnormality is evident on the ECG?
What is the cause of this abnormality?
What further evaluation is warranted?
Podrid's Real-World ECGs

ECG 44 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), first-degree AV block (prolonged AV conduction), left bundle branch block
The rhythm is regular at a rate of 70 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant (0.24 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm with first-degree AV block (prolonged AV conduction). The QRS duration is increased (0.16 sec). In leads I and V5-V6 there is a broad R wave (±), and in lead V1 there is a deep QS complex (−). This is the pattern of a left bundle branch block (LBBB). The QT/QTc intervals are prolonged (480/520 msec) but are normal when considering the prolonged QRS complex duration (400/430 msec). Noted are T waves that are tall and peaked in leads V3-V4 (▲); however, they are asymmetric and hence normal. The P wave in lead V1 is predominantly negative (•), suggesting left atrial hypertrophy.

An LBBB is identified by the following characteristics:

- The QRS complex duration is 0.12 second or longer due to delayed activation of the left ventricle. Left ventricular activation occurs after right ventricular activation.
- Left ventricular activation occurs from the right bundle and right ventricle directly through myocardium, bypassing the normal His-Purkinje system. Therefore, conduction is slow (causing the QRS complex to be widened) and all forces are directed from right to left (broad, tall R wave in leads I, aVL, and V5-V6; deep QS complex in leads V1 and possibly V2). No left-to-right forces should be seen (ie, there is no terminal S wave in lead I or V6).
- Since the interventricular septum is activated by a small septal branch (median fascicle) that comes from the left bundle and activates the septum in a left-to-right direction, no septal forces are seen (ie, no initial Q waves in lead I, aVL, or V3-V6 and no initial R wave in lead V1).
- Associated with the abnormal depolarization is abnormal repolarization. Hence diffuse ST-T wave abnormalities are seen.
- The axis may be normal or leftward. The left axis is not due to an isolated left anterior fascicular block as both fascicles are blocked as a result of the LBBB. Rather, the left axis is due to an abnormal direction of left ventricular activation from the right. A right axis is not seen with an LBBB as there should not be any left-to-right forces.
- Since left ventricular activation is abnormal and does not occur through the normal His-Purkinje system but rather through direct myocardial activation, left ventricular abnormalities (eg, left ventricular hypertrophy, infarction, ischemia, pericarditis) cannot be recognized.

Continues
Longstanding hypertension may result in pathologic left ventricular hypertrophy, which is associated with diffuse fibrotic changes. This often produces chronic elevation of left ventricular filling pressure and diastolic dysfunction that result in secondary left atrial hypertrophy. These morphologic alterations of the left ventricle can result in disruption of the architecture of the conduction system and may lead to an LBBB as noted in this case. An echocardiogram may be helpful in corroborating the physical exam findings consistent with left ventricular hypertrophy (prominent point of maximal impulse, S4 gallop). Although left ventricular hypertrophy cannot be diagnosed on the surface ECG in the presence of an LBBB, left atrial hypertrophy is clearly seen and supports this diagnosis.
A 59-year-old man with premature coronary artery disease, a history of multiple myocardial infarctions, and ischemic cardiomyopathy is admitted with heart failure. His ECG is shown.

What abnormalities are evident?

What mechanism for the conduction abnormality does the ECG suggest?
Podrid's Real-World ECGs

ECG 45 Analysis: Sinus tachycardia, intermittent (2:1)
left bundle branch block (possibly rate related), premature atrial complex
There is a regular rhythm at a rate of 100 bpm. There is a P wave (*) before each QRS complex with a constant PR interval (U) (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia. There are alternating changes in QRS complex width from beat to beat. The narrow QRS complexes (+) have a normal duration (0.10 sec) and morphology, while the wider QRS complexes (▼) (0.16 sec) have a left bundle branch block (LBBB) morphology (broad R wave in leads I and V5-V6 [▼] and a deep QS complex in lead V1 [▼]). The changes in QRS duration do not appear to be rate related. This is termed 2:1 LBBB. However, the 15th QRS complex (▼) is premature. A P wave with a different morphology than the sinus P waves (*) can be seen before this QRS complex; hence this is a premature atrial complex. This premature complex also has an LBBB morphology, suggesting that there is some relation to rate. The QT/QTc intervals of the wide QRS complexes are prolonged (400/516 msec) but are normal when corrected for the prolonged QRS complex duration (320/410 msec). The QT/QTc intervals of the narrow QRS complex are normal (320/410 msec).

The presence of 2:1 LBBB suggests that there is an underlying conduction abnormality in the left bundle such that it is not capable of conducting every QRS complex. Every other complex is blocked in the left bundle and hence not conducted through it. This is likely rate related, although it is difficult to establish this, except for the presence of a premature atrial complex that appears to have a rate-related LBBB. It is likely that in the future the patient will develop a permanent or persistent LBBB. There are no clinical implications to this finding, although it is important to recognize that the development of a persistent LBBB at a higher heart rate is not related to the presence of ischemia or another cause.
An ECG from a 67-year-old diabetic woman is shown.

What abnormalities are noted?
What pathology does the ECG suggest?
Podrid's Real-World ECGs

ECG 46 Analysis: Normal sinus rhythm, premature ventricular complex, rate-related left bundle branch block
There is a regular rhythm at a rate of 90 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec) (\(\text{PR}\)). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. All of the QRS complexes, except for the third complex (\(\text{\\&}\)), are wide (0.16 sec), and they have the morphology of a left bundle branch block (LBBB; broad R wave lead I [−+−] and QS complex in lead V1 [−+]). Although there is an rS complex in leads V4-V6, this is still consistent with an LBBB.

An occasional premature complex (\(\text{+}\)) can be seen (complexes 2 and 13). There are no P waves before these complexes, and they are wider with a different morphology compared with the other QRS complexes. Hence these are premature ventricular complexes. Both of them have the same morphology, so they are unifocal. There is a compensatory pause (\(\text{U}\)) following the second QRS complex (i.e., the PP intervals surrounding the pause are equal to two sinus PP intervals). A full compensatory pause is due to the fact that the on-time P wave that follows the premature ventricular complex does not conduct through the AV node, which is blocked as a result of retrograde conduction from the premature ventricular complex. As a result of the pause and a longer RR interval, the QRS complex following the ventricular premature complex (\(\text{A}\)) is narrow, with the same PR interval (\(\text{PR}\)) as the other complexes. This QRS complex is normal in both duration and morphology, as it does not have an LBBB. Therefore, the LBBB is rate related as it is not present when the rate is slower (i.e., after the pause).

A diseased conduction system may manifest obvious abnormalities at faster rates. A rate-related conduction abnormality per se does not suggest a specific disease entity. Although this patient has no symptoms, underlying coronary artery disease with resultant myocardial fibrosis may be present. It is also possible that the underlying conduction system disease is due to idiopathic changes of the conduction system, as are seen with Lev's or Lenègre's disease. Lastly, the patient may have an underlying cardiomyopathy of any cause. Although the rate-related LBBB has no important clinical implications, a persistent or permanent LBBB may develop in the future. Importantly, an LBBB is not related to acute ischemia, although it may often be due to underlying ischemic heart disease and the presence of myocardial fibrosis or prior infarction.

No therapy is indicated. Generally, no further evaluation is necessary, although not uncommonly an echocardiogram will be obtained to assess for cardiac structural abnormalities. If an exercise test was indicated for diagnosing underlying coronary artery disease, nuclear or echocardiographic imaging would be essential as ST-segment changes could not be interpreted in the presence of a rate-related LBBB.
A 64-year-old man presents to the emergency room with complaints of substernal chest pressure and diaphoresis that began 1 hour earlier. He states that he has a history of hypertension and hyperlipidemia. He sees his primary care physician on a regular basis and remembers being told that he has some type of block on his ECG. His baseline ECG (47A) is
available for review. His initial vital signs are stable, with a blood pressure of 130/90 mm Hg and a pulse of 72 bpm. Physical examination is unremarkable, although he is noted to be very diaphoretic. Nitroglycerin is given for the chest discomfort, providing incomplete relief. Morphine is administered. A 12-lead ECG (47B) is obtained.

What type of “block” is shown on the baseline ECG (47A)?
What diagnosis can be made based on ECG 47B?
What therapy is indicated?
ECG 47A Analysis: Normal sinus rhythm, left atrial abnormality (hypertrophy), left bundle branch block with associated ST-T wave abnormalities.
ECG 47A shows a regular rhythm at a rate of 75 bpm. There is a P wave before each QRS complex (+), with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6; therefore, this is a normal sinus rhythm. However, the P wave is broad and prominently notched in leads II and V3-V6 and is negative in leads V1-V2. This is consistent with left atrial hypertrophy (or abnormality). The QRS complex duration is increased (0.14 sec) and it has a typical left bundle branch block (LBBB) morphology, with a broad R wave in leads I and V5-V6 (++) and a deep QS complex in lead V1 (→). The axis is extremely leftward, between −30° and −90°. Although this axis is consistent with a left anterior fascicular block, this cannot be diagnosed in the presence of an LBBB as both fascicles (left anterior and left posterior) are blocked and not conducting an impulse to the ventricular myocardium. There are ST-T wave changes in leads I, aVL, and V3-V6 (−) that are secondary to the LBBB. The QT/QTc intervals are prolonged (420/470 msec) but are normal when the prolonged QRS complex duration is considered (380/425 msec).

continues
Podrid's Real-World ECGs

ECG 478 Analysis: Normal sinus rhythm, left bundle branch block, acute inferior wall and anterolateral wall myocardial infarction
In ECG 47B the rhythm is regular at a rate of 72 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6; hence this is a normal sinus rhythm. The QRS complex duration is increased (0.14 sec), and it has an LBBB morphology, with a broad R wave in leads I (→) and V6 and a deep QS complex in lead V1 (→). However, there is now significant ST-segment elevation (↑) in leads II, III, aVF, and V5-V6 that ranges from 2 to 5 mm (↑) as well as ST-segment depression in leads I and aVL (↓). These ST-segment depressions are more pronounced than those in the baseline ECG (ECG 47A) and are reciprocal changes seen with an inferior wall myocardial infarction (MI). In addition, the T waves in leads II, III, aVF, and V5-V6 are tall, domed, and symmetric. These changes are diagnostic for an acute inferior and anterolateral wall ST-segment elevation MI.

In the presence of an LBBB, left ventricular abnormalities (eg, left ventricular hypertrophy, pericarditis, ischemia, chronic MI, and axis) cannot be established reliably since left ventricular activation is no longer through the normal His-Purkinje system but instead is by direct myocardial activation from the right bundle and right ventricular myocardium. However, an acute ST-segment MI can be established based on the Sgarbossa criteria:

1. ST-segment elevation of ≥ 1 to 2 mm that is in the same direction (concordant) as the QRS complex in any lead.
2. ST-segment depression of ≥ 1 mm in any lead from V1 to V3
3. ST-segment elevation of ≥ 5 mm that is discordant with the QRS complex (ie, associated with a QS or rS complex)

Although the Sgarbossa criteria have been reported in patients with an LBBB, the same criteria have been found to be useful with a paced QRS complex and are likely also useful whenever there is direct myocardial activation, which would also include a ventricular complex and a Wolff-Parkinson-White pattern.

Even though this patient has an underlying LBBB, there are ECG changes that are diagnostic for an acute inferior wall ST-segment elevation MI, including the hyperacute T waves and the significant ST-segment elevation with reciprocal ST-segment depression. Important therapy would be prompt reperfusion either in the catheterization laboratory with percutaneous coronary intervention (stenting) or with a thrombolytic agent.
A 34-year-old man with a history of mild hypertension, well controlled on hydrochlorothiazide, presents to the emergency department with signs and symptoms of acute bronchitis. He has a productive cough and low-grade temperature and also notes pleuritic chest pain. A chest X-ray and white blood cell count are normal. An ECG is obtained, and the emergency department physician has some concerns about what it shows.

What is the abnormality? Is it of any concern?
ECG 48 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), nonspecific ST-T wave changes, intraventricular conduction delay (incomplete left bundle branch block)
There is a regular rhythm at a rate of 72 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The P waves are broad (0.16 sec) in leads II and aVF (+) and negative in lead V1. These changes are consistent with left atrial hypertrophy (abnormality). The QRS complex duration is increased (0.11 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex morphology is normal. There are nonspecific ST-T wave changes noted in leads I, II, aVL, and V5-V6 (*). The QT/QTc intervals are slightly prolonged (430/470 msec) but are normal when the prolonged QRS complex duration is considered (400/440 msec).

The major finding is the slightly prolonged QRS complex duration. Although it has a morphology resembling a left bundle branch block (LBBB; R wave in lead I and QS complex in lead V1), it is not wide enough for a true or complete LBBB. Therefore, this has been termed an incomplete LBBB. However, this is a misnomer since conduction through the bundles is all or none and not incomplete. The slight widening of the QRS complex is actually an intraventricular conduction delay to the left ventricle. Although this seems like only semantics, it is clinically important. An LBBB (complete or incomplete) means that the activation of the left ventricle bypasses (or partially bypasses) the normal His-Purkinje system. Thus activation of the left ventricle is not via the normal Purkinje system but directly through the ventricular myocardium. Therefore, abnormalities that affect the left ventricle (e.g., ischemia, myocardial infarction, pericarditis, left ventricular hypertrophy) cannot be diagnosed. The presence of an intraventricular conduction delay means that impulse conduction still occurs through the normal conduction system, but there is diffuse slowing of conduction through the Purkinje network and ventricular myocardium. As the normal conduction system is used, abnormalities that affect the left ventricle can still be diagnosed.

As there is no major abnormality on the ECG, there is no reason for any concern and no need for any additional evaluation aside from making certain that blood pressure is well controlled.
A 40-year-old woman who recently emigrated from Brazil presents to a primary care physician as a new patient. On review, she states that she has become progressively limited in her activity over the past several months because of shortness of breath. She denies chest discomfort, orthopnea, or changes in weight. On exam, her heart rate is 62 bpm and blood pressure is 110/70 mm Hg. Her head, ears, eyes, nose, and throat exam is normal. Her jugular venous pressure is 12 cm H₂O with Kussmaul's sign. Her lungs are clear. Her precordium is notable for a diffuse point of maximal impulse. Heart sounds are regular, and an S3 gallop is evident. Her abdomen manifests a fluid wave. The remainder of her exam is normal. An ECG is obtained.

What abnormalities are evident?
Podrid's Real-World ECGs

ECG 49 Analysis: Normal sinus rhythm, intraventricular conduction delay
There is a regular rhythm at a rate of 62 bpm. There is a P wave (*) before each QRS complex with a stable, short PR interval (0.12 sec). The QRS complex is wide (0.18 sec). Although the morphology looks like that of a left bundle branch block (broad R wave in leads I and V5-V6 [−] and deep S wave in lead V1 [−]), there are small septal Q waves (*) in leads I, aVL, and V5-V6 and a prominent septal R wave in lead V1 (↑). Septal Q waves cannot be present in a left bundle branch block, as septal activation is via the septal branch (median fascicle) from the left bundle. Hence the wide QRS complex is actually due to an intraventricular conduction delay. The QT/QTc intervals are prolonged (460/470 msec) but are normal when corrected for the wide QRS complex duration (360/370 msec).

With a left bundle branch block, activation of the left ventricle is not via the normal His-Purkinje system but rather is by direct myocardial activation. In contrast, the presence of an intraventricular conduction delay indicates that left ventricular activation is occurring via the normal conduction system but is diffusely slowed. Since the impulse travels along the normal His-Purkinje system, abnormalities that affect the left ventricle (eg, axis shift, acute and chronic myocardial infarction, ischemia, left ventricular hypertrophy, pericarditis) can be identified. Commonly a QRS complex that is this wide is the result of an underlying cardiomyopathy and is due to diffuse myocardial fibrosis and therefore marked slowing of impulse conduction. There is in fact a correlation between the left ventricular ejection fraction and the QRS complex duration with an intraventricular conduction delay. ■
A 68-year-old woman presents to her physician with complaints of progressive shortness of breath. She is known to have nonischemic dilated cardiomyopathy with a left ventricular ejection fraction of 40%, but she has never had heart failure. Physical exam demonstrates an S3, a murmur of mitral regurgitation, and bibasilar rales. Her heart rate is 120 bpm, and an ECG demonstrates sinus
tachycardia. She is admitted to the hospital and treated with intravenous diuretics as well as an angiotensin-converting enzyme inhibitor. She responds well, and by the third hospital day she feels back to her baseline. A routine ECG is obtained (ECG 50A) prior to the institution of a β-blocker. Her physician is concerned about the ECG and later in the day orders another (ECG 50B).

Why is there a concern about the initial ECG?
What is the nature of the abnormality?
Could therapy with a β-blocker be safely instituted?
ECG 50A Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), second-degree AV block with 2:1 AV conduction (2:1 AV block), right bundle branch block, left anterior fascicular block, bifascicular block, ventriculophasic arrhythmia
ECG 50A shows a regular rhythm at a rate of 46 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.32 sec). A second P wave (−) can be seen after each QRS complex. In some leads (V2-V3) the P wave is superimposed on the upstroke of the T wave (+), causing a notching of this waveform. The PP interval is slightly irregular (−), and the average atrial rate is 92 bpm. It can be seen that the PP interval surrounding the QRS complex is slightly shorter (0.60 sec) than the PP interval without a QRS complex (0.66 sec). This is termed ventriculoblastic arrhythmia and is due to acute acceleration of sinus node activity with ventricular contraction. This may be seen with either 2:1 AV block or complete (third-degree) AV block. Proposed mechanisms include acceleration of sinus node automaticity due to augmentation of pulsatile blood flow through the sinus node artery, stretch of the right atrium resulting from ventricular contraction, or changes in autonomic tone due to changes in baroreceptor activity resulting from a stroke volume.

The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm with first-degree AV block (prolonged AV conduction) and second-degree AV block with 2:1 conduction (2:1 AV block). The QRS complex duration is increased (0.16 sec), and it has a morphology of a typical right bundle branch block (RSR' complex in lead V1 [−]) and a broad terminal S wave in leads I and V5-V6 [−]). The axis is very leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology). As the QRS complex has an rS morphology, this is a left anterior fascicular block. The QT/QTc intervals are slightly prolonged (520/455 msec) but are normal when the prolonged QRS complex duration is considered (440/385 msec).

Therefore, there is bifascicular disease (right bundle branch block and left anterior fascicular block) as well as first-degree AV block (prolonged AV conduction). Although this might be considered to represent trifascicular disease, this diagnosis cannot be established by this ECG as the first-degree AV block may be the result of either AV nodal disease (which would not be trifascicular disease) or disease of the remaining fascicle (which would represent trifascicular disease). Even though there is also second-degree AV block present with a pattern of 2:1 conduction, this may be either Mobitz type I or Mobitz type II. If the 2:1 AV block is Mobitz type II, then trifascicular disease can be diagnosed. If the 2:1 AV block is Mobitz type I, which is an AV nodal abnormality, then this would be bifascicular disease as well as AV nodal disease. The etiology of the 2:1 AV block can only be established if there was a change in the pattern of AV nodal conduction. Thus, if there were several sequential P waves that were conducted with a stable PR interval, then the diagnosis would be Mobitz type II. If the P waves were conducted with a progressive increase in the PR interval, the diagnosis would be Mobitz type I. If complete heart block were to develop, the etiology of the escape rhythm would also establish the etiology of the 2:1 AV block. That is, if there was a junctional escape rhythm the etiology would be Mobitz type I, while an escape ventricular rhythm would establish Mobitz type II as the etiology.

continues
ECG 50B Analysis: Normal sinus rhythm, complete heart block, escape junctional rhythm, right bundle branch block, left anterior fascicular block, bifascicular block, ventriculophasic arrhythmia
In ECG 50B, there is a regular rhythm at a rate of 42 bpm. The QRS complex duration, morphology, and axis are identical to what was seen in ECG 50A (ie, right bundle branch block and left anterior fascicular block). The QT/QTc intervals are the same. P waves are seen (+), and the PP interval is fairly constant (\( \pm \)) with an average atrial rate of 84 bpm. As with ECG 50A, ventriculophasic arrhythmia is present and the PP interval surrounding the QRS complex (0.70 sec) is slightly shorter than the PP interval without an intervening QRS complex (0.78 sec) (\( \pm \)). However, the PR intervals are variable (\( \pm \)), particularly evident at the end of the ECG, indicating AV dissociation. As the atrial rate is faster than the ventricular rate, this is complete or third-degree AV block. The QRS complexes are identical to those seen in ECG 50A, so the escape rhythm is junctional. Therefore, the 2:1 AV block seen on ECG 50A is a result of Mobitz type I. The conduction abnormalities are not the result of trifascicular disease but rather are due to bifascicular disease with associated AV nodal abnormality. As the complete heart block is a result of failure of AV nodal conduction, a \( \beta \)-blocker should not be administered as this could exacerbate the presence of complete heart block and might depress an escape junctional focus, resulting in a slowing of the junctional rate.
A 36-year-old man with a family history of sudden death and dilated cardiomyopathy presents to the emergency department following 2 days of extreme fatigue and lightheadedness. On initial ECG...
triage, his heart rate is 30 bpm with a blood pressure of 90/60 mm Hg. An ECG (51A) is obtained. The patient is admitted to the critical care unit where a second ECG (51B) is obtained.

What abnormalities are shown?

What is the management?
Podrid's Real-World ECGs

ECG 51A Analysis: Normal sinus rhythm, complete heart block, escape junctional rhythm with right bundle branch block (RBBB) and left anterior fascicular block (LAFB), bifascicular block
ECG 51A shows regular RR intervals at a rate of 30 bpm. There are P waves (*) seen, and they have a stable PP interval (LU) at a rate of 90 bpm. Some of the P waves are not apparent (+) as they are either on T waves or superimposed on the QRS complexes. However, the PP intervals of the obvious P waves are regular (LJ). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The PR intervals (+-) are variable, indicating AV dissociation. The atrial rate is faster than the ventricular rate, and hence this is complete heart block. The QRS complexes are wide (0.16 sec) with a pattern of right bundle branch block (RBBB; RSR' morphology in lead V1 [←] and S wave in leads I and V5-V6 [→]). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology), indicating a left anterior fascicular block (LAFB). Hence the escape rhythm is junctional with an RBBB and LAFB. The QT/QTc intervals are prolonged (560/400 msec) but are normal (480/340 msec) when adjusted for the increased QRS complex duration. 

continues
ECG 518 Analysis: Normal sinus rhythm, complete heart block, escape junctional rhythm with RBBB and left posterior fascicular block (LPFB), trifascicular block
In ECG 51B, there is a regular rhythm at a ventricular rate of 32 bpm. P waves (*) are seen, and they have a stable PP interval (LJ) at an atrial rate of 78 bpm. The PR interval (+-0) is not constant, and hence there is AV dissociation as a result of complete heart block (the atrial rate is faster than the ventricular rate). The QRS complexes are wide (0.16 sec), similar to those in ECG 51A, with an RBBB pattern (RR’ morphology in lead V1 [+] and an S wave in leads I and V5-V6 [-]). The axis is rightward, between +90° and +180°. The QRS complex is negative in lead I (with an rS morphology), even when accounting for the terminal S wave caused by the RBBB, and positive in lead aVF. This is indicative of a left posterior fascicular block (LPFB). Hence the escape rhythm is junctional with an RBBB and LPFB. The QT/QTc intervals are the same as those seen in ECG 51A.

When compared with ECG 51A, the RBBB is still present in ECG 51B, with the same QRS complex duration and same QT/QTc intervals. However, there is also evidence of conduction disease involving both the left anterior and left posterior fascicles, as indicated by the axis change of the QRS complexes in the two ECGs (extreme left to right). Although the complete heart block is the result of block within the AV node (as the escape rhythm is junctional), there is also trifascicular disease present, as indicated by the RBBB associated with alternating LAFB and LPFB.

The diagnosis of trifascicular disease is made when there is evidence of conduction abnormalities affecting all three fascicles. This might be alternating RBBB and left bundle branch block (LBBB), also known as bi-bundle branch block or, as in this patient, RBBB with alternating LAFB and LPFB.

In this case, the escape junctional rhythm indicates a conduction abnormality of the AV node. However, there is also evidence of severe infra-Hisian disease of the His-Purkinje conduction system (ie, trifascicular disease). Insertion of a permanent pacemaker is indicated. In this patient with familial cardiomyopathy, the decision to place an implantable cardioverter-defibrillator in conjunction with the pacemaker will depend on the left ventricular ejection fraction (LVEF) and New York Heart Association (NYHA) functional class (LVEF < 35% and class II–III heart failure for non-ischemic cardiomyopathy based on current guidelines). In general, a biventricular pacemaker is not indicated when the widened QRS complex is the result of an RBBB. It has been shown that a biventricular pacemaker is effective primarily for patients with an LBBB and not those with an RBBB or an intraventricular conduction delay. However, this patient has complete heart block and will likely be pacemaker dependent. It has been suggested that biventricular pacing should be used in such patients who will, therefore, have continuous right ventricular pacing with an LBBB pattern.
A 55-year-old woman with advanced HIV and related cardiomyopathy is admitted to the hospital with *Pneumocystis pneumonia*. An ECG is obtained (52A) as...
part of her initial evaluation. The following day, the attending physician notes a change in the appearance of her telemetry tracing and obtains a follow-up ECG (52B).

What abnormalities on the ECGs explain the physician’s observations?
Podrid's Real-World ECGs

**ECG 52A Analysis:** Normal sinus rhythm, right bundle branch block, nonspecific ST-T wave abnormalities
ECG 52A shows a regular rhythm at a rate of 86 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex has a prolonged duration (0.16 sec) with a pattern that is typical for a right bundle branch block (RBBB; RSR' morphology in lead V1 [−−] and broad S wave in leads I and V5-V6 [→]). There is a physiologic left axis, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). Nonspecific ST-T wave changes noted in leads V1-V3 (*) are associated with the RBBB. The ST-T wave changes seen in leads V4-V6 (†) are, however, primary and not associated with the RBBB. The QT/QTc intervals are prolonged (420/500 msec) but are normal when the increased QRS complex duration is considered (340/410 msec).
Podrid's Real-World ECGs

ECG 52B Analysis: Normal sinus rhythm, left bundle branch block, bi-bundle branch block (trifascicular disease)
ECG 52B, obtained the following day, shows a regular rhythm at a rate of 64 bpm. There is a P wave (*) before each QRS complex with a PR interval that is constant (0.20 sec). The P-wave morphology and PR interval are the same as in ECG 52A. The QRS complex duration is increased (0.16 sec), and the morphology is that of a left bundle branch block (LBBB; broad R wave in leads I and V6 [-] and QS complex in lead V1 [+] ). The axis is now more leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). However, this is not a left anterior fascicular block as there is an LBBB present and hence both fascicles are not functional. The axis is due to the direction of left ventricular activation via direct myocardial activation. The QT/QTc intervals are the same as in ECG 52A. Hence the patient has evidence of both RBBB and LBBB, indicative of bi-bundle branch block or trifascicular disease.

The presence of both RBBB and LBBB represents high-grade conduction system disease of the His-Purkinje system, which is likely the result of the underlying cardiomyopathy. Although there is a high risk for complete heart block, which would be associated with an escape ventricular rhythm, a pacemaker is not indicated at this time. In the presence of bi-bundle branch block or trifascicular disease, a pacemaker is indicated for documented complete AV block, high-degree AV block (Mobitz type II) associated with symptoms, or a history of syncope that has no other definable etiology.
An ECG from a 24-year-old man with a history of palpitations associated with lightheadedness is presented.

What is your interpretation of this ECG?
ECG 53 Analysis: Sinus bradycardia, short PR interval (accelerated or enhanced AV conduction)
There is a regular rhythm at a rate of 56 bpm. There is a P wave (*) before each QRS complex. The P wave is upright in leads I, II, and V4-V6 and is thus originating from the sinus node. The PR interval is constant but short (0.12 sec) (IIJ). Indeed, there is no PR segment seen. The QRS complex is narrow (0.08 sec) and has a normal morphology and normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (420/406 msec). The short PR interval represents accelerated AV conduction, which is due to either enhanced AV nodal conduction or an accessory pathway that bypasses the AV node. Since the QRS complex is narrow, conduction to the ventricle is via the normal His-Purkinje system. When resulting from an accessory bypass tract, this pattern is known as Lown-Ganong-Levine (LGL) and is due to a bypass tract known as the bundle of James that links the atrium and the bundle of His. LGL pattern accounts for the short PR interval and normal QRS complex.

Since this patient has symptoms, it is important to perform ambulatory monitoring either with a Holter monitor if his symptoms are daily, or with an event recorder (or even an implantable loop recorder) if his symptoms are less frequent. LGL pattern is associated with a preexcitation syndrome, and the palpitations might be indicative of a reentrant arrhythmia involving a circuit that includes the accessory pathway and AV node. This arrhythmia is termed atrioventricular reentrant tachycardia. It is also possible that he could be experiencing another type of supraventricular arrhythmia in which the accessory pathway is being used as the conduction pathway to the ventricles. The documentation of an arrhythmia associated with an LGL pattern of the ECG is termed LGL syndrome.
A 24-year-old woman presents to your clinic with a history of episodic palpitations that occur several times per week. She has never felt lightheaded or experienced loss of consciousness. She is active, running 2 to 3 miles several times per week. She has no other medical problems and denies any family history of cardiac disease. Her vital signs and physical exam are normal. An ECG is obtained.

What abnormality is depicted?
What is the suggested cause of her symptoms?
What is the next step in her evaluation?
Podrid's Real-World ECGs

ECG 54 Analysis: Sinus bradycardia, Wolff-Parkinson-White pattern
There is a regular rhythm at a rate of 56 bpm. There is a P wave (•) before each QRS complex with a stable PR interval that is short (0.10 sec) (‖). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus bradycardia. The QRS complex is widened (0.12 sec) as a result of a slurred upstroke (†) of the initial portion of the QRS complex (producing a widening of the base of the QRS complex but not the peak). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (420/406 msec). This slurred upstroke, which can be seen in most but not all leads, is called a delta wave and indicates initial direct and slow myocardial activation via an accessory pathway. The presence of a short PR interval associated with the widened QRS complex and delta wave is known as Wolff-Parkinson-White (WPW) pattern. This is a form of preexcitation syndrome and may be associated with a specific reentrant arrhythmia that occurs as a result of the accessory pathway called atrioventricular reentrant tachycardia (AVRT). Other atrial arrhythmias may occur and use the accessory pathway to activate the ventricle; however, these arrhythmias do not require the accessory pathway for their occurrence. These arrhythmias may present with a wide QRS complex reflecting accessory pathway conduction.

WPW pattern is due to an AV nodal bypass tract called the bundle of Kent that serves as a direct connection between atrial and ventricular myocardium, bypassing the AV node and resulting in direct and early ventricular myocardial activation that precedes activation via the AV node (ie, preexcitation). The manifestation of this preexcitation is a short PR interval and an early and slurred upstroke of the QRS complex (reflecting initial direct myocardial activation) termed a delta wave. The QRS complex is widened as a result of this delta wave. The QRS complex is a fusion beat representing early ventricular activation initiated via the accessory pathway and slightly later activation via the normal AV node–His-Purkinje system. The QRS complex is thus widened and abnormal. The widening is more apparent at the base of the QRS complex, as a result of the delta wave, while the QRS complex is narrower at its peak. The width of the delta wave and the degree of aberration are related to balance between conduction through the AV node and accessory pathway. It is the AV nodal conduction properties that determine the extent of the delta wave and the QRS complex duration, as conduction through the accessory pathway is "all or none," similar to what is seen in His-Purkinje fibers. When AV nodal conduction is slow, more myocardium is activated via the accessory pathway; hence the PR interval is shorter, the delta wave is more prominent, and the QRS complex is wider. When AV nodal conduction is faster, less myocardium is activated via the accessory pathway; hence the PR interval is longer, the delta wave is less prominent, and the QRS complex is narrower. 

continues
Palpitations have a broad differential diagnosis and may be related to an increase in sinus rate, isolated premature complexes, or a supraventricular tachyarrhythmia. Supraventricular arrhythmia (i.e., atrial tachycardia, atrial flutter, atrial fibrillation) may conduct to the ventricles via the normal His-Purkinje system, by the accessory pathway, or via both conduction systems. AVRT is supraventricular tachycardia that is specific for a preexcitation syndrome and involves a macroreentrant circuit (which includes the accessory pathway, AV node, normal His-Purkinje system, and the atrial and ventricular myocardium) that links these two pathways proximally and distally. The circuit must be complete for AVRT to exist.

The next step in this patient’s evaluation is to correlate her symptoms with an arrhythmia via ambulatory monitoring, especially using an event or loop recorder that can record a patient’s rhythm for up to 1 month. Documentation of an arrhythmia, particularly an AVRT, would be diagnostic for WPW syndrome and should prompt an electrophysiologic study with consideration of radiofrequency ablation of the bypass tract. Other supraventricular arrhythmias may also occur and may use the bypass tract to conduct to the ventricle. The treatment would depend on the type of supraventricular tachyarrhythmia documented and also whether the QRS complex during the arrhythmia was widened or preexcited.
A 18-year-old man has an ECG performed given his involvement in competitive athletics at the collegiate level and a family history of sudden death.

What abnormality is noted on the ECG?
ECG 55 Analysis: Sinus bradycardia, Wolff-Parkinson-White pattern
There is a regular rhythm at a rate of 48 bpm. There is a P wave (*) before each QRS complex, and the PR interval is 0.10 second ([ ]). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus bradycardia. The QRS complex is widened (0.18 sec) as a result of a slurred upstroke due to a very broad delta wave (*). This is Wolff-Parkinson-White (WPW) pattern. The PR interval is very short, and the delta wave is wide. The delta wave presents as a negative waveform (Q wave) in leads II, III, and aVF (*). This is a pseudo inferior wall myocardial infarction pattern and is associated with a bypass tract located in the posterior septal wall. With WPW pattern initial ventricular activation is by direct myocardial activation, bypassing the His-Purkinje system. Therefore, a chronic infarction cannot be recognized or diagnosed; hence it is termed a pseudo infarction pattern. For the same reason, an extreme left axis due to a conduction abnormality of the His-Purkinje system (ie, a left anterior fascicular block) cannot be diagnosed in WPW pattern. Furthermore, other left ventricular abnormalities (eg, left ventricular hypertrophy, myocardial ischemia or infarction, pericarditis) cannot be diagnosed. As WPW pattern represents a fusion between conduction via two different pathways that connect the atria with the ventricles (accessory pathway and normal AV node–His-Purkinje system), the markedly short PR interval and wide delta wave indicate that the majority of ventricular activation is via the accessory pathway and less ventricular myocardium is being activated via the AV node–His-Purkinje system. This is due to the fact that conduction via the AV node is relatively slow, resulting in more ventricular myocardial activation through the rapidly conducting accessory pathway. The QT/QTc intervals are prolonged (560/500) but are normal (460/410 msec) when the increased QRS complex duration is considered.

Patients with WPW pattern are susceptible to a reentrant supraventricular arrhythmia known as atrioventricular reentrant tachycardia (AVRT). The reentrant circuit for this arrhythmia involves the accessory pathway and the normal AV node–His-Purkinje system, which are the two pathways (or limbs) that connect the atria and ventricles. These two limbs are connected via the atrial and ventricular myocardium, forming a macro-reentrant circuit. This entire circuit needs to be intact for an AVRT to exist. Other atrial arrhythmias may also occur, such as atrial fibrillation, atrial flutter, or atrial tachycardia. 

continues
These arrhythmias originate in the atrium, and they use the normal His-Purkinje and accessory pathways to conduct impulses to the ventricles. Unlike AVRT, they do not depend on the conduction system or accessory pathway for their existence.

Any supraventricular arrhythmia may be associated with symptoms. However, the major concern is a risk for sudden cardiac death in patients with WPW syndrome. Sudden death occurs primarily in those patients who experience atrial fibrillation with an atrial rate in excess of 350 to 450 bpm. If the refractoriness of the accessory pathway is very short, it is capable of rapid conduction and may transmit the atrial fibrillatory impulses to the ventricle at very rapid rates. In this situation the ventricle, even if normal, may develop ventricular fibrillation, which is the mechanism for sudden death. Although the actual incidence of sudden death in patients with WPW pattern on their ECG is low, this patient has a family history of sudden death. In addition, he is involved in competitive sports. Radiofrequency ablation is often performed in this type of patient, even in the absence of symptoms.
A 52-year-old woman is seen by her physician because of complaints of palpitations. However, the etiology for these symptoms has never been documented. Her physician recommends an exercise test. When she presents for this study, a resting ECG is obtained. The exercise technician thinks the ECG is abnormal, and he cancels the test.

What abnormality is noted?
What is the significance of this finding?
ECG 56 Analysis: Normal sinus rhythm, intermittent Wolff-Parkinson-White pattern, low limb lead voltage, old anterior wall myocardial infarction
There is a slightly irregular rhythm at a rate of 96 bpm. There is a P wave (+) before each QRS complex, and the atrial rate (PP intervals) is regular. The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. However, the PR intervals are not constant, alternating between 0.16 and 0.12 second (\(\frac{1}{10}\)). As a result, the RR intervals are slightly irregular (ie, 0.56 and 0.64 sec, respectively) (\(\frac{1}{10}\)). Therefore, the rhythm is regularly irregular.

The longer PR interval is associated with a QRS complex that has a normal duration (0.08 sec), a normal axis of about 0° (positive QRS complex in lead I and biphasic QRS complex in lead aVF), and low limb lead voltage (defined as a QRS complex amplitude < 5 mm in each limb lead and < 10 mm in each precordial lead). There are QS complexes in leads V1-V3 and a significant Q wave in lead V4 (\(\frac{1}{10}\)), consistent with an old anterior wall myocardial infarction (MI). The shorter PR interval is associated with a QRS complex with a wide duration (0.16 sec). In addition, this QRS complex has a slurred upstroke of the R wave (\(\frac{1}{10}\)), accounting for the excess width, which is particularly evident at the base of the QRS complex. This slurred upstroke is called a delta wave and is consistent with the preexcitation pattern of Wolff-Parkinson-White (WPW). Interestingly, the QRS complexes that are wide and preexcited do not have evidence of the old anterior wall MI. This is because there is direct myocardial activation via the accessory pathway and, similar to other situations in which there is direct myocardial activation (ie, left bundle branch block, paced QRS complexes, ventricular complexes), myocardial abnormalities, including a chronic MI, cannot be reliably diagnosed. Therefore, this patient’s stress test must be performed with imaging (either nuclear imaging or echocardiography) as the ECG is unreliable at detecting ischemia in the presence of WPW pattern.

There is an alternating pattern of normal QRS complexes and preexcited QRS complexes, or an intermittent WPW pattern. The QT/QTc intervals are normal (360/450 msec). Although the QRS complexes have a small amplitude in lead I, it appears that there is an initial Q wave in this lead as well as in lead aVL (\(\frac{1}{10}\), a pseudo lateral wall infarction pattern), with a positive delta wave in lead V1, consistent with a bypass tract located in the left lateral wall.

The presence of intermittent preexcitation generally means that the refractory period of the accessory pathway is relatively long and is not able to conduct at very rapid rates. This implies that if the patient should develop atrial fibrillation, the ventricular response rate would not be very rapid and hence there would be a reduced risk for provoking ventricular fibrillation.
Practice ECGs
A 52-year-old man is admitted for an anterior wall ST-segment elevation myocardial infarction (MI), which is treated with angioplasty and stenting of a proximal left anterior descending artery occlusion. After the procedure he is treated with aspirin, clopidogrel, and a β-blocker. On hospital day four, a nurse notices irregular QRS complexes on the patient’s telemetry monitor. She checks on the patient, who is sound asleep. The patient has had an uneventful post-intervention course without recurrent chest pain or arrhythmia. The nurse obtains an ECG from the telemetry monitor and asks you to review it.

What is the explanation for the irregular complexes noted on telemetric monitoring?
Is any therapy warranted?
ECG 57 Analysis: Normal sinus rhythm, Mobitz type I second-degree AV block (Wenckebach), left anterior fascicular block, anteroseptal MI, no specific T-wave abnormalities
The PP intervals are stable (+-+), and the atrial rate is regular at 98 bpm. The P wave (*) is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The average ventricular rate is 78 bpm. The QRS complex duration is normal (0.08 sec) and the axis is extremely leftward, between $-30^\circ$ and $-90^\circ$, consistent with a left anterior fascicular block (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). There is also evidence of an old anteroseptal myocardial infarction (MI; QS complex in leads V1-V2 (%)). Also noted is nonspecific T-wave flattening in the limb leads and in leads V4-V6 (+). The QT/QTc intervals are normal (380/430 msec).

Grouped beating is seen with occasional pauses or long RR intervals. The PR interval (□) after each pause is normal at 0.20 second. This is the baseline PR interval. Following this pause there is progressive PR interval lengthening (0.28, 0.32 sec) (□I) followed by nonconducted P wave (+), which accounts for the pause. This P wave is superimposed on the T wave and not readily apparent. However, by using the PP intervals, it can be seen that the P wave is occurring on time. This is the pattern of Mobitz type I second-degree AV block or Wenckebach. The first group of QRS complexes shows 3:2 Wenckebach, followed by a pattern of 4:3 Wenckebach. The Wenckebach cycle length is even longer at the end of the ECG. Mobitz type I second-degree AV block (Wenckebach) can be seen during periods of increased vagal tone, such as during sleep. It is indicative of an increased AV nodal refractory period with delayed conduction through the node and is not considered suggestive of AV node injury. Mobitz type I AV block in this case is unlikely related to the patient's recent anterior wall MI. It is, however, commonly seen with an inferior wall MI, generally the result of increased vagal tone as well as edema within the AV junction, around the AV node.

Wenckebach is a conduction abnormality within the AV node. Since the electrophysiologic properties of the AV node are mediated by calcium currents, conduction through this structure is not all or none but can vary depending on changes in nodal properties. The AV node exhibits decremental conduction (ie, conduction through the node prolongs as the heart rate increases). These non-pathologic changes in the speed of AV conduction are usually due to changes in the node's refractory period. It is common for Wenckebach to be noted at night while patients are sleeping. This is likely the result of increased vagal tone in addition to therapy with a β-blocker. In the absence of symptoms, which are unlikely to be present while the patient is sleeping, no therapy is necessary. β-blocker therapy does not need to be withdrawn. Likewise, asymptomatic Wenckebach that occurs during the day does not require therapy.
A 54-year-old woman is admitted to the hospital after she presented to her primary physician with complaints of fatigue. The admission was prompted by an ECG obtained in the clinic. On review, the patient states that 1 week prior to the onset of fatigue she noted an unheralded episode of jaw and chest discomfort that lasted several hours and then resolved. She did not seek any medical care.

**What rhythm abnormality is shown?**

**Based on the ECG, what is the cause of the patient's symptoms over the past week?**

**What therapy is indicated?**

**What further testing may be helpful?**
ECG 58 Analysis: Normal sinus rhythm with complete (third-degree) AV block, junctional escape rhythm, acute inferior wall myocardial infarction
There is a regular narrow complex rhythm at a rate of 62 bpm. The QRS complex duration is normal with a normal morphology. There is a physiologic left axis, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (380/386 msec). Noted are P waves (*) at a rate of 86 bpm. Occasionally the P waves are superimposed on the T waves or the QRS complexes (▼), presenting with bumps on the T waves, which should have a smooth upstroke and downstroke. On occasion a P wave is not seen, being hidden within the QRS complex. The P waves that are seen, however, occur at regular PP intervals (LJ) and are dissociated from the QRS complexes as the PR intervals are variable (+/−). Hence AV dissociation is present. Since the atrial rate is faster than the ventricular rate, this is complete heart block with an escape junctional rhythm as the QRS complexes are normal in width and morphology.

Also seen are ST-segment elevations (↑) in leads II, III, and aVF with T-wave inversions (*). These abnormalities are consistent with an acute inferior wall myocardial infarction (MI). The presence of Q waves (●) in these leads indicates that the ECG changes are resolving, confirming that the MI most likely occurred several days before this ECG was obtained, likely a week ago when she experienced chest and jaw pain.

As a result of the inferior wall MI the patient likely had ischemic damage or edema in the AV junctional area. The AV node itself is relatively impervious to ischemia as its electrophysiologic properties are mediated by calcium currents and it is energy and oxygen independent. Thus, AV nodal conduction abnormalities, including complete heart block (with an escape junctional rhythm) are not generally the result of structural damage to the AV node but usually are due to high vagal tone as well as edema in the AV junction around the AV node. Therefore, the conduction abnormalities with an inferior wall MI are usually transient and resolve spontaneously.

The patient will require routine post-MI therapy with aspirin and a statin. β-blocker therapy is important but should not be started until the complete heart block has resolved. Infrequently, the AV block persists, indicating more significant damage to the AV junction. In these cases a pacemaker may be indicated, especially since β-blocker therapy is needed long-term. Evaluation of left ventricular function with an echocardiogram is also warranted. Exercise testing is often performed to establish functional capacity as well as to evaluate other areas of potential ischemia. If symptoms of ischemia recur or if there is evidence of ischemia with exercise testing, anti-ischemic medications (primarily nitrates) would be indicated. Coronary angiography would be indicated if there were recurrent anginal symptoms despite medical therapy or if there were other signs of ongoing ischemia such as hemodynamic instability, heart failure, or ventricular tachycardia.
A 77-year-old woman is admitted to the emergency department after having a pre-syncopal episode. At home, she had become unsteady on her feet and fallen to the ground without loss of consciousness. On arrival, she is conscious but disoriented. Her heart rate is 35 bpm, and her blood pressure 92/60 mm Hg. While she is being stabilized, an ECG is obtained.

What diagnosis is revealed by the ECG? What is the next step in her management?
ECG 59 Analysis: Sinus bradycardia, isorhythmic AV dissociation with a captured complex, junctional rhythm, left posterior fascicular block, intraventricular conduction delay to the right ventricle, U waves
The RR intervals are normal at a regular rate of 35 bpm. P waves are seen before each QRS complex (*), with regular PP intervals at a rate of 35 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a sinus rhythm. The PR intervals are not constant (\( \uparrow \)) as they range from 0.10 to 0.12 second. Therefore, AV dissociation is present. The P wave is less obvious before the second QRS complex as it is superimposed on the initial portion of the QRS complex (\( \downarrow \)). The atrial and ventricular rates are identical, indicating isorhythmic AV dissociation. AV dissociation may be due to complete heart block, in which the atrial rate is faster than the rate of the QRS complexes, which may be either junctional or ventricular in origin. The location of the escape rhythm is based not on the rate of the QRS complexes but on their morphology. AV dissociation may also be due to an accelerated lower pacemaker (either junctional or ventricular), in which case the atrial rate is slower than the rate of the QRS complexes. When the rate of the P waves and QRS complexes is identical, the term isorhythmic dissociation is applied.

There is a change in the QRS morphology. The first through fifth QRS complexes (+) have a slightly increased duration (0.11 sec) and an intraventricular conduction delay to the right ventricle with a terminal S wave in lead I (\( \rightarrow \)) and an RSR' morphology in lead V1 (\( \rightarrow \)). The axis is rightward, between +90° and +180° (negative QRS complex in lead I even when the terminal S wave is considered and positive QRS complex in lead aVF). In the absence of any other reason for a right axis (ie, right ventricular hypertrophy, right–left arm lead switch, lateral wall myocardial infarction, dextrocardia, Wolff-Parkinson-White pattern), the diagnosis is a left posterior fascicular block. The last complex (†) has a narrow duration (0.08 sec), and there is no right ventricular conduction delay; the axis is no longer rightward as the QRS complex is positive in lead I. There is an on-time P wave before this QRS complex with a PR interval of 0.16 second, which is slightly longer than the previous PR intervals. This suggests that there is intact AV conduction and that this is a captured beat. QRS complexes 1 to 5 are slightly wider and have a right axis, but the initial forces are the same (ie, there is a narrow initial R wave in lead I). Hence these are junctional complexes. Junctional complexes, which originate from an ectopic focus within the AV junction, commonly enter the bundle of His at a slightly different location than the sinus complexes, in which the impulse travels through the AV node. Hence junctional complexes are conducted through the His-Purkinje system slightly differently, resulting in mild differences in axis, amplitude, and occasionally width, with the development of a slight conduction delay. The QT/QTc intervals are normal (500/380 msec). Low-amplitude and normal U waves are also noted in leads V2 and V4 (\( \uparrow \)).

The presence of a captured complex does not help establish the etiology of the AV dissociation as there could be complete AV block with a captured complex or capture as a result of a slight increase in the sinus rate (not apparent on the ECG), resulting in capture and hence suppression of the junctional focus. Regardless of the etiology of the AV dissociation, the patient appears to have a symptomatic bradycardia. Unless a reversible etiology is discovered, with resolution of the isorhythmic dissociation and bradycardia (as can occur in vasovagal or neurocardiogenic syncope or other conditions associated with an increased vagal tone), a permanent pacemaker should be considered.
An 81-year-old woman presents to her physician with palpitations, which she has been experiencing intermittently for 1 month. She has not experienced any syncopal or pre-syncopal symptoms and denies exposure to caffeine-containing foods or over-the-counter medications containing a stimulant. A recent thyroid-stimulating hormone (TSH) test was normal. Her physical exam, including vital signs, is unremarkable. As part of her office-based evaluation, an ECG is obtained.

What is your interpretation of the tracing? Does it suggest a cause for her symptoms? If so, what therapy might you pursue?
ECG 60 Analysis: Normal sinus rhythm, Mobitz type II second-degree AV block, left bundle branch block
There is a regular atrial rhythm at a rate of 94 bpm. There are P waves (*) before each QRS complex, and they are positive in leads I, II, aVF, and V4-V6. Hence this is normal sinus rhythm. The QRS complex intervals are regularly irregular and grouped beating is present. There are three pauses (L), each of which has the same duration (ie, twice the underlying sinus rate). The pause is due to a nonconducted, on-time sinus P wave (+); this defines a second-degree AV block. The PR interval (0.18 sec) (**) is stable when AV conduction is present. Therefore, this is second-degree AV block, Mobitz type II, which is due to intermittent failure of impulse conduction through the His-Purkinje system. In addition, the QRS complexes are wide (0.16 sec) and have a left bundle branch block pattern (tall, broad R wave in leads I and V5-V6 [—] and a broad QS complex in lead V1 [→]). As both the left anterior and left posterior fascicles are involved, this can be termed bifascicular disease. The QT/QTc intervals are prolonged (440/550 msec) but are normal (360/450 msec) when the increased QRS complex duration is considered.

Mobitz type II second-degree block, when symptomatic or when associated with a widened QRS complex or evidence of bifascicular disease, is an indication for permanent pacemaker implantation as it indicates infra-Hisian disease of the conduction system. When it progresses to complete heart block, Mobitz type II disease is associated with an escape ventricular rhythm that may be slow and associated with symptoms, especially syncope. Even if the rate of the ventricular rhythm is normal, the ventricular focus may be unstable and unpredictable as there is no autonomic control. Hence the ventricular rate may vary widely. The Mobitz type II block, however, is not the cause of this patient’s symptoms of palpitations, unless what she means is “skipped beats.” The etiology for palpitations needs to be further evaluated.
A 79-year-old woman presents to your urgent care clinic with complaints of 1 day of lightheadedness that came on suddenly and has not resolved. She states that standing makes the lightheadedness worse but denies syncope. On review, she states that preceding this symptom, she had several days of gastroenteritis (with vomiting and diarrhea). These symptoms have largely resolved, but she admits to continued dehydration. Her medical history is notable only for hypertension, for which she takes atenolol.

On exam, her vital signs are notable for bradycardia at 44 bpm with an orthostatic fall in her blood pressure. Her cardiopulmonary exam is otherwise normal. Laboratory data are normal except for a blood urea nitrogen level of 46 mg/dL (baseline, 24 mg/dL) and a creatinine level of 1.8 mg/dL (baseline, 1.0 mg/dL). You obtain an ECG.

What abnormalities are shown?
What is the likely etiology?
**ECG 61 Analysis:** Normal sinus rhythm, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block
The RR intervals are regular at a rate of 44 bpm. A P wave (*) is seen before each QRS complex and the PR interval (+-) is stable at 0.44 second, representing first-degree AV block (prolonged AV conduction). However, a second P wave (+) can be seen at the beginning of the T wave; it is not followed by a QRS complex (ie, it is nonconducted). The presence of an occasional nonconducted P wave defines second-degree AV block. The PP intervals are regular (square), and hence the atrial rate is stable at 88 bpm. The P wave is positive in leads I, II, aVF, and V4-V6. There is, therefore, a normal sinus rhythm with first- and second-degree AV block present. The second-degree AV block has a pattern of 2:1 conduction. This can be either Mobitz type I or Mobitz type II. The etiology can only be established if there is a change in the pattern of AV conduction (ie, there are two or more sequentially conducted P waves). If the PR intervals lengthen progressively, then the diagnosis is a Mobitz type I block; if the PR intervals are constant, it is Mobitz type II.

The QRS complex duration is normal and it has a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (500/430 msec).

The patient appears to be somewhat dehydrated, with elevated blood urea nitrogen and creatinine levels. This is likely due to the gastroenteritis. The conduction system abnormalities seen on this tracing could be due to an excess effect of β-blocker, as atenolol is a long-acting hydrophilic β-blocker that is cleared from the systemic circulation by the kidneys. This patient's gastroenteritis and dehydration, resulting in pre-renal azotemia, have likely caused an increase in serum atenolol levels and an enhanced β-blocking effect. If this is the case, then this would be Mobitz type I block, which is an AV nodal conduction abnormality, since the AV node is the structure affected by β-blockade. In contrast, Mobitz type II block is a problem of conduction through the His-Purkinje system, and conduction through this pathway is not affected by β-blockade. Her lightheadedness may be due to continued dehydration from her recent illness or could be the result of her bradycardia. Intravenous fluid infusion to relieve her orthostatic blood pressure response and correct her azotemia is appropriate. If bradycardia worsens or symptoms progress despite intravenous fluid administration, options for immediate treatment of β-blocker toxicity include a catecholamine (eg, epinephrine), glucagon, calcium, and possibly insulin plus glucose. As atenolol is a long-acting β-blocker with a half-life of approximately 8 hours and a duration of effect of up to 12 hours with normal renal function and up to 35 hours with marked reduction in renal function, these maneuvers will be short lived. Therefore, if symptoms continue, a catecholamine infusion or placement of a transvenous temporary pacing wire may be required to stabilize the patient until her renal function improves and serum levels of atenolol decrease.
A 42-year-old man with known pulmonary sarcoidosis is admitted with an episode of unheralded syncope while at home alone. He recovered from the event spontaneously.
without sequelae and called 911. His presentation ECG in the emergency department is shown (62A). A second ECG is repeated after admission to the hospital (62B).

What abnormalities are depicted?
What do these abnormalities suggest about the cause of his syncope?
ECG 62A Analysis: Normal sinus rhythm, right bundle branch block (RBBB), left anterior fascicular block (LAFB), 2:1 AV block progressing to complete heart block with an escape ventricular rhythm, trifascicular disease.
In ECG 62A, the first two QRS complexes (▼), which are at a rate of 34 bpm, have a P wave (*) in front of them with a stable PR interval (▲) of 0.20 second. The P wave is positive in leads I, II, aVF, and V4-V6, consistent with a normal sinus rhythm. However, an on-time but nonconducted P wave (+) (ie, not associated with a QRS complex) is seen after each of these two QRS complexes. Hence the atrial rate is 68 bpm. As every other P wave (+) is nonconducted, this is a second-degree AV block with 2:1 AV conduction.

The first two QRS complexes, which are conducted, have a right bundle branch block (RBBB) pattern (RSR’ morphology in lead V1 [▼]) and broad S wave in lead I [▲] and a left anterior fascicular block (LAFB), denoted by an extreme left axis, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology). The last four QRS complexes (▲) are at a slightly faster rate (38 bpm) and have a left bundle branch block (LBBB) pattern (deep QS complex in lead V1 [▼] and broad R wave in lead V6 [▲]). Noted is atrial activity (●) at a rate of 68 bpm (the same atrial rate as was seen at the beginning of the ECG) with variable PR intervals (+); this is consistent with AV dissociation. The atrial rate is faster than the ventricular rate and hence this is complete heart block. The QRS complex of the escape rhythm (LBBB morphology) differs from that of the conducted beats (RBBB morphology), and hence this is an escape ventricular rhythm. This indicates that the 2:1 AV block is Mobitz type II and that the block is within the His-Purkinje system. The presence of RBBB and LAFB is diagnostic for bifascicular disease; however, in this case, it can be seen that the complete heart block is associated with an escape ventricular rhythm, indicating disease in the His-Purkinje system. This means that there is conduction disease in the remaining fascicle (ie, left posterior fascicle). Therefore, this is trifascicular block.

continues
ECG Analysis: Normal sinus rhythm, complete heart block with an escape ventricular rhythm and intermittent capture with complexes having an RBBB and LAFB, trifascicular disease
ECG 62B shows the same pattern as ECG 62A. The first and last QRS complexes (+) have an RBBB pattern and there is a P wave (↑) before each; the PR intervals preceding them are the same (0.20 sec) (↑). These complexes are identical to the conducted complexes seen in ECG 62A. The second, third, fourth, and fifth QRS complexes (↓) have an LBBB pattern at a rate of 36 bpm, similar to the complexes during complete heart block on ECG 62A. Noted are P waves (↑), which have a regular PP interval (↓) at an atrial rate of 68 bpm. Some of the P waves are not seen because they are superimposed on the QRS complex (▲). However, the P waves that are seen occur at a regular interval (↓). There is variability of the PR intervals (↔); therefore, AV dissociation is present. As the atrial rate (68 bpm) is faster than the ventricular rate (36 bpm), this is complete AV block with an escape ventricular rhythm. The block is within the His-Purkinje system. However, the first and last QRS complexes (+) are supraventricular in origin and have the same PR interval. Hence they are captured complexes and, therefore, there is complete heart block with intermittent capture. It is not uncommon for there to be intermittent AV conduction (and hence capture) during complete heart block. Indeed, the complete heart block may be transient, with subsequent restoration of intact AV conduction. With the complete heart block and a slow ventricular rate the patient might become symptomatic and even experience syncope. If there is resolution of the complete AV block with resumption of AV conduction, the patient spontaneously recovers; this has been termed Stokes-Adams attacks and is seen with transient complete heart block. □
Notes
A 71-year-old woman is admitted to the hospital with a new diagnosis of atrial fibrillation and rapid ventricular response. She was started on verapamil and achieved rate control quickly, eventually spontaneously converting to sinus rhythm. On the day of discharge, an ECG reviewed by the medical resident causes alarm. The resident presents the ECG to you.

What finding on the ECG concerned the house officer?
What would be an appropriate course of action?
Podrid's Real-World ECGs

ECG 63 Analysis: Normal sinus rhythm, complete heart block with escape junctional rhythm, intermittent capture, first-degree AV block (prolonged AV conduction)
The rhythm is regularly irregular with long and short RR intervals. The ventricular rate is 42 bpm, although there are two QRS complexes (third and sixth) that are early (+), at a rate of 50 bpm. There are P waves seen (+), and they have a stable PP interval (⊥). The atrial rate is 72 bpm. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The PR intervals are variable (L); hence AV dissociation is present. As the atrial rate is faster than the ventricular rate, this is complete (third-degree) AV block. However, the PR intervals associated with the two early QRS complexes (+) are the same (0.38 sec) (+). Since these two complexes are early, they are in response to the P waves before them. In addition, the presence of the same PR interval in both cases indicates intact AV conduction. Therefore, these two complexes represent AV conduction with first-degree AV block (prolonged AV conduction). Since the conducted and nonconducted QRS complexes have the same morphology, there is complete heart block with an escape junctional rhythm. Therefore, the AV block is within the AV node. The QRS complexes have a normal duration (0.08 sec) and morphology. There is a left axis, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (480/400 msec).

It is possible that the complete heart block, which is due to an abnormality of AV nodal conduction, is the result of verapamil. Since verapamil was used for ventricular rate control during atrial fibrillation, this drug is no longer necessary now that sinus rhythm has been restored. Verapamil, a calcium-channel blocker, works only on the AV and sinus nodes, which have their electrophysiologic properties mediated by calcium currents. It has no direct effects on atrial or ventricular myocardium, which have electrophysiologic properties mediated by a fast action potential that is a result of rapid influxes of sodium ion. It is unlikely that the reversion to normal sinus rhythm was related to the verapamil. ■
A 53-year-old woman with systemic hypertension and resultant mild renal dysfunction is seen by her primary physician. She has not had any new symptoms, and a review of systems is unremarkable. An ECG is obtained as part of her routine evaluation.

What abnormalities are seen?
Podrid's Real-World ECGs

ECG 64 Analysis: Normal sinus rhythm, biatrial hypertrophy (abnormality), left bundle branch block
There is a regular rhythm at a rate of 74 bpm. A P wave (*) is seen before each QRS complex, and the PR interval is constant (0.20 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The P waves are tall and peaked (+) in leads II and aVF, suggesting right atrial hypertrophy. The presence of a negative P wave (−) in lead V1 suggests left atrial hypertrophy. These P-wave abnormalities are consistent with biatrial hypertrophy (abnormality). The QRS complex duration is lengthened (0.16 sec), and there is a pattern of a left bundle branch block (broad R wave in leads I and V5-V6 [−] and deep QS complex in lead V1 [−]). Also noted are ST-T wave abnormalities (+) associated with the left bundle branch block. The QT/QTc intervals are prolonged (440/490 msec) but are normal when corrected for the prolonged QRS duration (360/400 msec).

A very common ECG finding in hypertension is left ventricular hypertrophy (LVH). However, in this case, LVH cannot be diagnosed because of the presence of the LBBB. With an LBBB there is direct activation of the left ventricular myocardium, bypassing the normal His-Purkinje system. Hence the diagnosis of left ventricular abnormalities such as LVH cannot be reliably established. An LBBB is often seen with pre-existing LVH due to fibrosis of the septum, which can often affect the His-Purkinje system (especially the left bundle). The presence of LVH can, however, be established with an echocardiogram. In addition, left atrial hypertrophy may develop as a result of the LVH, which is associated with diastolic dysfunction and elevated left ventricular filling pressures. Although the presence of right atrial hypertrophy is uncommon with systemic hypertension, it may occur if there is significant diastolic dysfunction, as may be seen with LVH resulting in pulmonary hypertension and hence elevated right-sided pressures.
A 34-year-old man is seen for a physical exam before getting his pilot’s license. He denies any current symptoms or any history of symptoms. An ECG is performed as part of his evaluation.

What is your interpretation of his ECG?

What additional evaluation is indicated?
**ECG 65 Analysis:** Normal sinus rhythm, short PR interval (enhanced or accelerated AV conduction)
The rhythm is regular at a rate of 86 bpm. There is a P wave (*) before each QRS complex, and it is positive in leads I, II, aVF, and V4-V6; hence it is a normal sinus rhythm. The PR interval (II) is constant but short (0.10 sec). The QRS complex is normal in width and morphology. The QT/QTc intervals are normal (380/430 msec).

The normal PR interval ranges from 0.14 to 0.20 second. The PR interval includes the P wave (which reflects atrial depolarization) and the PR segment, which reflects conduction through the AV node and His-Purkinje system. In this case, there is a short PR segment. The major component of the PR segment is AV nodal conduction as the AV node is the site of slowest conduction velocity within the conduction system. A PR interval less than 0.14 second is considered short and indicates either enhanced conduction through the AV node or bypass of the AV node, a result of an accessory pathway. In the case of an accessory pathway, an ECG with a short PR interval and normal QRS complex is due to a pathway called the bundle of James that bypasses the AV node but connects into the bundle of His. This is termed a Lown-Ganong-Levine (LGL) pattern. It is a form of preexcitation syndrome and may be associated with a reentrant arrhythmia called AV reentrant tachycardia. This patient does not have any history of arrhythmia and has no symptoms suggestive of arrhythmia. He has an LGL pattern, but not LGL syndrome. Hence no additional evaluation or therapy is warranted.
A 55-year-old man with a strong family history of cardiac disease presents to the clinic for a routine physical. He states that most of the male members of his family have had pacemakers.
implanted in their fifth to sixth decade of life. His review of systems is negative. Given his family history of cardiac disease, a routine ECG is performed (66A) and is compared with a previous ECG (66B).

What abnormality is present?
Is there any change in the ECGs?
Is any therapy warranted?
ECG 66A Analysis: Sinus tachycardia, short RP tachycardia, nonspecific T-wave abnormalities, left ventricular hypertrophy
ECG 66A shows a regular rhythm at a rate of 100 bpm; hence this is a tachycardia. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The depth of the S wave in lead V2 is 30 mm (\( ] \)), and the R-wave amplitude in lead V5 is 17 mm (\( ] \)), for a total of 47 mm. This meets one of the criteria for left ventricular hypertrophy (LVH; S-wave depth in lead V1 or V2 + R-wave amplitude in lead V5 or V6 ≥ 35 mm). Apparent T-wave abnormalities (+) are seen in leads V4-V6; these are likely secondary to LVH. The QT/QTc intervals are normal (320/410 msec).

P waves are not obvious, but abnormal waveforms are seen in the ST segments in leads I and V1-V3 that are suggestive of superimposed P waves (*). This is, therefore, short RP tachycardia with a long PR interval (0.42 sec) (\(+\)). The etiology for short RP tachycardia includes sinus tachycardia with first-degree AV block (prolonged AV conduction), atrial tachycardia, ectopic junctional tachycardia (with a retrograde P wave), atrial flutter with 2:1 AV block, typical AV nodal reentrant tachycardia (ie, slow-fast), or AV reentrant tachycardia (associated with preexcitation or an accessory pathway). It appears that the P waves are positive in lead aVF, eliminating any arrhythmia with retrograde P waves (ie, ectopic junctional tachycardia, AV nodal reentrant tachycardia, or AV reentrant tachycardia). A second atrial waveform is not seen, and hence this is not atrial flutter with 2:1 AV block. It is not certain whether the P waves are positive or negative in leads I and V4-V6 as the negative T-wave abnormalities might be due to P waves. Thus this is either sinus or atrial tachycardia. 

\*continues
**ECG 66B Analysis:** Normal sinus rhythm, first-degree AV block (prolonged AV conduction), nonspecific T-wave abnormalities
ECG 66B shows a regular rhythm at a rate of 72 bpm. The QRS complex duration, morphology, and axis are identical to those seen in ECG 66A. The QT/QTc intervals are also the same. Positive or upright P waves (*) are now obviously seen before some of the QRS complexes in leads I, II, aVF, and V3-V6. The PR interval is constant at 0.38 second (+). Therefore, this is a normal sinus rhythm with first-degree AV block (prolonged AV conduction). The P waves are obvious on this ECG because the sinus rate is slower than in ECG 66A. The PR interval in ECG 66B is very similar to the interval seen in ECG 66A (+) between the abnormal waveform and the subsequent QRS complex. Therefore, this abnormal waveform is a superimposed P wave, as a result of the more rapid heart rate, and it is not apparent as a distinct waveform. The fact that the PR interval as measured on ECG 66B is very similar to the interval on ECG 66A confirms that the short RP tachycardia is sinus tachycardia with first-degree AV block.

When the sinus rate increases in the setting of first-degree AV block, the P wave may become superimposed on the preceding ST segment or T wave; hence the P wave may not be obvious. However, the ST segment and T-wave upstroke and downstroke should be smooth. Any notching or bumps in the ST segment or on the T waves should raise suspicion for superimposed P waves. In this situation, it may be useful to slow the sinus rhythm, which can be accomplished by enhancing vagal tone, as with carotid sinus pressure or Valsalva maneuver. In addition, P waves should be sought if there is any pause in the RR intervals, as may occur with a premature complex.
A 34-year-old man presents to his primary physician for a routine exam. He is about to embark on his third triathlon, for which he has been actively training for over a year. A thorough review of systems is negative.
The patient has no known medical diagnoses. As part of the evaluation, the physician obtains an ECG (67A). The physician requests that the patient return the next day for follow-up, and a second ECG is obtained (67B).

**What conduction abnormalities are noted on ECG 67A?**

**What is the likely cause?**

**What further diagnostic information does the second ECG (67B) provide?**
ECG 67A Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block, low limb lead voltage
ECG 67A shows P waves (*) that have a regular PP interval (−−) at a rate of 84 bpm. There is a second P wave after each QRS complex (+). This second P wave is nonconducted. There are P waves (*) before each QRS complex, and they are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The presence of occasional nonconducted P waves defines second-degree AV block. When the P wave is conducted the PR interval (U) is constant, although prolonged at 0.28 second; hence there is also a first-degree AV block (prolonged AV conduction). As every other P wave is nonconducted there is a second-degree AV block with 2:1 conduction; this may be either Mobitz type I or Mobitz type II. The ventricular rate is 42 bpm.

The QRS complex duration is normal (0.08 sec), and there is low voltage in the limb leads (<5 mm in each limb lead). There is a physiologic left axis, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (460/384 msec).
ECG 67B Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type I second-degree AV block (Wenckebach), premature atrial complex
In ECG 67B the rhythm is irregularly irregular. There are P waves (*) occurring at a regular rate of 96 bpm (L). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. However, the fourth P wave (+) is early and has a different morphology. Hence this is a premature atrial complex. Complexes 1 through 4 (1) are preceded by a P wave, but there is progressive lengthening of the PR interval from 0.28 second (which is the baseline PR interval and is identical to that in ECG 67A) to 0.36 second (→). The P wave following the second and fourth QRS complexes (2) is nonconducted. Hence this is a pattern of 3:2 Wenckebach. The QRS complexes that follow (complexes 5–8) are regular at a rate of 46 bpm. Each QRS complex is preceded by a P wave (†) with a PR interval of 0.28 second, similar to the PR intervals of the first complex of the Wenckebach cycles and identical to those in ECG 67A. However, after each QRS complex there is another P wave (▼) that is not conducted. Hence this is second-degree heart block with 2:1 AV conduction, similar to the pattern seen in ECG 67A.

As there is 3:2 Wenckebach along with 2:1 AV block, the 2:1 AV block is also Wenckebach or Mobitz type I. Hence the conduction abnormality on both ECGs is second-degree AV block, type I or Wenckebach.

Highly conditioned athletes and well-conditioned people in general demonstrate ECG markers of high vagal tone, including sinus bradycardia, first-degree AV block (prolonged AV conduction), and Mobitz type I second-degree AV block. These are not pathologic entities in these subjects. The physiologic effect of conditioning is enhanced vagal tone. Indeed the athletic heart functions more efficiently at a slower heart rate and with a greater stroke volume.
A 56-year-old woman with known familial dilated cardiomyopathy presents to the emergency department having suffered an episode of syncope while dining with friends in a restaurant. She regained consciousness spontaneously and relates that she felt lightheaded before the episode. She has never had an event like this in the past but states that her primary doctor has told her that her ECG is “not normal.” Her vital
signs are notable for an apical heart rate of 30 bpm. Her physical exam is notable for clear lung fields, a jugular venous pressure of 7 cm H₂O, a diffuse and displaced point of maximal impulse, and an S3 gallop with a grade I/VI apical holosystolic blowing murmur. Her abdominal, extremity, and neurologic exams are normal. An ECG is obtained (68A). She is admitted to the hospital for further evaluation, and a second ECG (68B) is obtained the following day.

**What abnormality is depicted on ECG 68A?**

**What further information does ECG 68B provide?**

**What therapy is indicated?**
ECG 68A Analysis: Sinus bradycardia, left atrial hypertrophy, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block, left anterior fascicular block (LAFB), right bundle branch block (RBBB), nonspecific ST-T wave abnormalities
In ECG 68A, there is a regular rhythm at a rate of 28 bpm. The QRS complexes are wide (0.16 sec) and have a right bundle branch block (RBBB) pattern (tall R wave in lead V1 [→] and broad terminal S wave in leads V5-V6 [←]). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology). This is, therefore, left anterior fascicular block (LAFB). Hence there is bifascicular block.

The QT/QTc intervals are normal (580/400 msec and 500/340 msec when accounting for the widened QRS complex). There are also ST-T wave changes (↑) noted in leads I and V3-V6. There are P waves (↑) before each QRS complex, and they are positive in leads I, II, aVF, and V4-V6. The PR interval (↑) is prolonged at 0.40 second and is stable. Hence there is first-degree AV block (prolonged AV conduction).

This has often been called trifascicular disease; however, the location of the conduction slowing accounting for the prolonged PR interval (ie, AV nodal or His-Purkinje) cannot be established from this ECG. Hence trifascicular disease cannot be established with certainty. There is a second on-time but nonconducted P wave (↑) (ie, unassociated with a QRS complex) after each QRS complex. The PP intervals are constant (↓) at an atrial rate of 56 bpm; hence this is sinus bradycardia. There is also second-degree AV block with a 2:1 conduction pattern. This may be Mobitz type I or Mobitz type II. The presence of 2:1 AV block does not help establish trifascicular disease. The presence of other conduction abnormalities (RBBB, LAFB, and first-degree AV block) does not help establish the etiology of the second-degree AV block. In addition, the P waves in leads V1-V3 are primarily negative (↓), consistent with left atrial hypertrophy.
ECG 68B Analysis: Sinus bradycardia, left atrial hypertrophy, first-degree AV block (prolonged AV conduction), Mobitz type II second-degree AV block, LAFB, RBBB, trifascicular block disease
In ECG 68B, the QRS morphology, duration, and axis are the same as in ECG 68A. The QT/QTc intervals and ST-T wave abnormalities are also the same. There is a regular atrial rhythm at a rate of 54 bpm. The P wave is positive in leads I, II, aVF, and V4-V6; hence this is sinus bradycardia. There is a P wave (*) before each QRS complex and the PR interval (+) is stable at 0.40 second, similar to the PR interval of the conducted complexes in ECG 68A. There is a pause seen (n) with an on-time but nonconducted P wave (+). The pause is equal to two RR intervals. All the PR intervals are constant, including those before and after the pause. This is Mobitz type II second-degree AV block. Therefore, the 2:1 AV block seen in ECG 68A, obtained the previous day, is also Mobitz type II. As Mobitz type II block is due to disease within the His-Purkinje system, it is now established that the patient has evidence of trifascicular disease; and the pause (ie, Mobitz type II) is the result of intermittent block in the left posterior fascicle, which is the only part of the conduction system that is responsible for AV conduction to the ventricles.

The patient displays a degree of AV block that warrants pacemaker implantation. Depending on her degree of heart failure and left ventricular dysfunction, more advanced therapies such as cardiac resynchronization as well as implantation of a cardiac defibrillator may be considered.
A 48-year-old diabetic man with atrial fibrillation on digoxin has a history of viral gastroenteritis and polyuria. As his oral intake was poor due to gastroenteritis, he stopped taking his insulin for several days. He is brought to the hospital by his wife, who noted that he was confused and at times obtunded. On exam, his mucous membrane are very dry. His blood pressure is 90/60 mm Hg, and his heart rate is 40 to 50 bpm. His respiratory rate is rapid with deep inspirations. His consciousness is waxing and waning, and he is barely rousable. Laboratory values include the following: blood urea nitrogen 65 mg/dL, creatinine 2.3 mg/dL, serum sodium 158 mmol/L, serum potassium 3.0 mEq/L, and serum bicarbonate 16 mmol/L. The serum glucose level is 760 mg/dL. Blood gases show a pH of 7.24, normal \( P_O_2 \), and \( P_C_O_2 \) of 25 mm Hg. A diagnosis of ketoacidosis is made, and the patient is treated appropriately. An ECG is obtained.

What is the abnormality on the ECG?
What is a possible cause?
ECG 69 Analysis: Atrial fibrillation with intermittent complete AV block and an escape ventricular rhythm, intraventricular conduction delay, premature ventricular complex, U waves
The underlying rhythm is atrial fibrillation; there is no organized atrial activity and fibrillatory waves are seen, particularly in lead V1 (†). The fibrillatory waves are irregular in morphology, amplitude, and interval. The RR intervals of the first three complexes (*) are irregular (1.0 and 1.14 sec, with rates of 48–60 bpm), and hence they are the result of intact AV nodal conduction that occurs irregularly due to the atrial fibrillation. The QRS complex is wide (0.14 sec) and has a morphology resembling a left bundle branch block, with a broad R wave in lead I (―) and a QS complex in lead V1 (―). However, there is also a normal and narrow R wave and a prominent S wave in lead V5 (††), reflecting terminal forces being directed from left to right. This is not seen with a left bundle branch block but rather is more consistent with a right bundle branch block. Hence the QRS complexes have an intraventricular conduction delay. The QT/QTc intervals are slightly prolonged (480/450 msec), although they are normal when the prolonged QRS complex duration is considered (420/400 msec).

Noted is that the fourth QRS complex (+) occurs after a longer RR interval (++) (1.6 sec, rate 38 bpm). Following this complex is a very wide and abnormal QRS complex (complex 5), which is a premature ventricular complex (/>. The RR interval from the premature ventricular complex to the next QRS complex (complex 6) (▲) as well as the RR intervals of the last two QRS complexes (7 and 8) (▼) are the same (RR interval 1.6 sec, rate 38 bpm) and are identical to the RR interval between complexes 3 (*) and 4 (+). Hence there is regulation of these RR intervals during atrial fibrillation, which indicates the presence of complete AV block. The fourth, sixth, seventh, and eighth QRS complexes have a wider duration (0.18 sec) and a morphology (in leads II and V5) that is different from the first three QRS complexes. These are, therefore, escape ventricular complexes. Also noted after the ventricular complexes in leads V1-V3 are prominent U waves (●), suggesting hypokalemia, although prominent U waves may also be seen with significant bradycardia.

This patient has ketoacidosis, precipitated by a viral infection as well as the discontinuation of insulin. He is very dehydrated due to the ketoacidosis (associated with polyuria) as well as poor oral intake. Renal insufficiency (due to prerenal azotemia) has likely led to an increase in serum digoxin levels, resulting in signs of digoxin toxicity. Although this can result in complete AV block, it would be associated with an escape junctional rhythm. In this case, the escape rhythm is ventricular, meaning that the complete heart block results from a conduction abnormality within the His-Purkinje system. Although digoxin toxicity is usually associated with conduction block within the AV node and hence a junctional escape rhythm, very high levels may also suppress conduction within the His-Purkinje system, resulting in an escape ventricular rhythm. It is also likely that the His-Purkinje conduction abnormality is the result of the acidosis and other metabolic derangements. Appropriate therapy includes correcting the ketoacidosis and electrolyte abnormalities, as well as withholding digoxin until renal function recovers. If the complete AV block resolves, permanent pacing is not indicated. Persistence of complete heart block means that there is permanent His-Purkinje conduction disease and in this situation permanent pacing would be indicated. ■
An ECG from an asymptomatic 42-year-old man is shown.

What abnormalities are noted?
Podrid’s Real-World ECGs

ECG 70 Analysis: Sinus tachycardia, right bundle branch block
There is a regular rhythm at a rate of 100 bpm. There is a P wave (*) before each QRS complex and the PR interval is stable (0.16 sec). The QRS complex duration is prolonged (0.14 sec), and there is an RSR' morphology (−−−) in lead V1 and a broad S wave (→→) in leads I and V5-V6. This is a pattern of a right bundle branch block. The QT/QTc intervals are prolonged (360/465 msec) but are normal when the prolonged QRS complex duration is considered (300/388 msec). Although the axis appears to be rightward, this is a result of the broad S wave in lead I.

Since axis determination is based on left ventricular forces, the S wave (representing right ventricular activation) should not be considered.

An isolated right bundle branch block is not uncommon in the normal population and generally is not indicative of any underlying cardiac abnormality. In the absence of any symptoms that point to a cardiac problem, no further evaluation or therapy is necessary.
An 89-year-old man with a history of paroxysmal atrial fibrillation, for which he takes warfarin, is admitted to the neurosurgical intensive care unit after presenting with a traumatic fall and large intracerebral hemorrhage. His admission ECG (71A) is interpreted as...
sinus bradycardia. The following day during rounds, an astute medical student notices that the patient's PR interval is different on the lead II telemetry than the previous day. An ECG is obtained (71B) and the primary neurology team requests your interpretation.

What are the findings on the initial ECG (71A)? How does the follow-up ECG (71B) assist in the diagnosis?
**ECG 71A Analysis:** Sinus rhythm with ventriculophasic arrhythmia, first-degree AV block (prolonged AV conduction), second-degree AV block with 2:1 conduction, right bundle branch block, left anterior fascicular block
ECG 71A shows a regular rhythm at a rate of 32 bpm. There are P waves (*) before each QRS complex and the PR interval is stable (**) but prolonged (0.44 sec), defining a first-degree AV block. Noted in leads V1-V2 is a second, nonconducted P wave (+) that has the same morphology as the conducted P wave. The atrial rate is 64 bpm. Hence every other P wave is conducted, indicating second-degree AV block with 2:1 AV conduction. The PP intervals (atrial rate) are not regular; the PP interval around the QRS complex is shorter (0.8 sec) than the PP interval without a QRS complex (1.0 sec). This represents ventriculophasic arrhythmia. Ventriculophasic arrhythmia can be seen whenever there is 2:1 AV block or complete heart block. Although the exact mechanism is not clear, it is hypothesized that the shortened PP interval around the QRS complex can be a result of enhancement of sinus node rate with ventricular contraction due to pulsatile blood flow through the sinus node artery, which increases sinus node automaticity; increased stretch on the right atrium, which increases sinus node automaticity; or alteration of carotid sinus outputs (baroreceptor changes) related to the stroke volume with ventricular contraction. It is a normal physiologic occurrence.

The QRS complexes are widened (0.14 sec) and there is a pattern of right bundle branch block (RBBB; RSR' morphology in lead V1 [-] and broad S wave in leads I and V5-V6 [+]). The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology), defining a left anterior fascicular block (LAFB).

Even though there is a first-degree AV block (prolonged AV conduction) and bifascicular disease (RBBB and LAFB), this cannot be called trifascicular disease as it is not certain whether the first-degree AV block is the result of AV nodal disease or His-Purkinje disease (ie, whether the 2:1 AV block is Mobitz type I or Mobitz type II).
ECG 718 Analysis: Sinus tachycardia, complete heart block, junctional escape rhythm
ECG 71B shows a regular rhythm at a rate of 34 bpm. There are P waves (*) at a regular rate of 100 bpm (L1), although some of the P waves are not obvious as they are superimposed on the QRS complex or T wave (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia. The PR interval (+ +) is inconsistent and always different than the baseline PR interval (0.44 sec as seen on ECG 71A), indicating AV dissociation. The atrial rate is faster than the ventricular rate, which is diagnostic for complete heart block. In contrast, an atrial rate that is slower than the ventricular rate would indicate an accelerated lower focus (ie, junctional or ventricular). The QRS complex has an RBBB and LAFB, identical in morphology and axis to the QRS complexes seen in ECG 71A; hence the escape rhythm is junctional and the 2:1 AV block is a Mobitz type I. This confirms the fact that trifascicular disease was not present but that the first-degree AV block with 2:1 AV conduction is the result of AV nodal disease (Mobitz type I).

AV block is seen in approximately 10% of patients with intracerebral hemorrhage as well as in patients with acute ischemic stroke. Although AV block in the context of cerebral infarction portends a worse prognosis, it does not seem to be an independent predictor of mortality in cerebral hemorrhage. Despite the baseline conduction abnormalities, the development of complete heart block is associated with a stable junctional escape, so pacemaker implantation may be avoided as AV block is likely a transient complication of the acute neurologic process rather than a progression of the underlying conduction system disease.
A 65-year-old man with known hereditary hemochromatosis presents with palpitations. He is otherwise well. A review of systems is unremarkable, and his vital signs are normal. However, the following are noted on physical exam: a normal cardiac point of maximal impulse with an irregular rhythm and an S4 gallop, bronzing of the skin, and small testes. Laboratory values include a normal thyroid-stimulating hormone level, a transferrin saturation of 80%, and mild transaminitis. An ECG is obtained.

What abnormalities are noted?
What therapy is indicated?
ECG 72 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type II second-degree AV block (high-degree AV block)
The rhythm is irregular, although a pattern can be seen (all of the short intervals are the same [LI]); hence the rhythm is regularly irregular. The QRS complexes are normal in duration (0.08 sec) and morphology. There is a left axis, between 0° and −30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are slightly prolonged (380/455 msec). P waves (+) are seen before each QRS complex, with a stable PR interval (0.22 sec). There are additional P waves seen (+) with a stable PP interval and an atrial rate of 86 bpm (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm with first-degree AV block (prolonged AV conduction). The RR intervals between the third and fourth QRS complexes and between the last four QRS complexes are similar (LI); the ventricular rate is 86 bpm. There are three pauses or long RR intervals seen (LI). During these pauses there are on-time, nonconducted P waves (+) (ie, three nonconducted P waves during the first pause, one during the second pause, and two sequential nonconducted P waves during the third pause). The presence of two or more sequential nonconducted P waves is seen with Mobitz type II second-degree AV block; with Mobitz type I block only one nonconducted P wave is seen. The presence of two or more nonconducted P waves is often called high-degree AV block.

Patients with hemochromatosis may develop conduction defects due to the underlying genetic mutation or the infiltrative cardiomyopathy. The iron deposition is associated with features of restrictive cardiomyopathy or dilated cardiomyopathy. This patient does not manifest physical exam findings of depressed left ventricular function or heart failure. However, the S4 gallop may point to a disorder of left ventricular relaxation in the context of cardiac iron deposition. An echocardiogram is warranted. Given Mobitz type II heart block, particularly with multiple nonconducted P waves and a slow ventricular rate, pacemaker implantation is indicated. Phlebotomy and/or chelation therapy is indicated as well, which in some cases can reverse left ventricular dysfunction. The effect of these therapies on conduction system disease is unknown.
A 68-year-old woman with ischemic cardiomyopathy requests an urgent appointment with her cardiologist for lightheadedness, dyspnea, and fatigue over the past 24 hours. Upon evaluation, her heart rate is noted to be 32 bpm, and her blood pressure is 88/60 mm Hg. Her exam shows clear lung fields, a jugular venous pressure of 8 cm H₂O, a 2-cm laterally displaced cardiac point of maximal impulse, normal cardiac sounds without gallops, and a soft blowing systolic murmur at the apex. Her abdominal and extremity exams are normal.

An ECG is obtained.

What ECG abnormality explains her symptoms?

What therapy is indicated?
Podrid's Real-World ECGs

ECG 73 Analysis: Normal sinus rhythm, left bundle branch block, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block progressing to complete heart block, escape ventricular rhythm
The initial portion of the ECG (first three QRS complexes [\(\square\)]) shows a regular rhythm at a rate of 32 bpm. P waves are seen before each of the first three QRS complexes (\(\bullet\)) and the PR interval (\(\Rightarrow\)) is constant (0.24 sec), indicating first-degree AV block (prolonged AV conduction). There are also on-time but nonconducted P waves after each of these QRS complexes (+). Therefore, there is a stable atrial rate (\(\square\)) of 64 bpm. The P wave is positive in leads I, II, aVF, and V4-V6; hence the rhythm is sinus. As every other P wave (\(\bullet\)) is nonconducted, this is second-degree AV block with 2:1 AV conduction; the second-degree block may be either Mobitz type I or Mobitz type II. Hence there is first- and second-degree AV block.

The QRS complex is wide (0.16 sec) and has a left bundle branch block (LBBB) pattern (broad R wave in lead I [\(\Rightarrow\)]). The QT/QTc intervals are normal (560/410 msec and 480/350 msec when the prolonged QRS complex duration is considered). The presence of an LBBB is considered bifascicular disease. Along with the first-degree AV block and second-degree AV block with 2:1 conduction, a diagnosis of trifascicular disease may be considered. However, it is not clear whether the first- and second-degree AV block is due to an AV nodal or His-Purkinje conduction abnormality. A diagnosis of trifascicular disease can only be made if the abnormality is in the His-Purkinje system. A change in the pattern of conduction would be necessary to establish the etiology for the 2:1 AV block.

In the second portion of the ECG (\(\text{ie}\), the last three QRS complexes [\(\square\)]), there is a change in the pattern. P waves are seen (\(\bullet\)) and the atrial rate is the same (\(\square\)) (64 bpm), but the ventricular rate is 36 bpm and there is now AV dissociation with a variable PR interval (\(\Rightarrow\)). In addition, the QRS complex morphology has changed, although the complexes still have an LBBB pattern. This is now complete heart block with an escape ventricular rhythm. Hence, the 2:1 AV block was Mobitz type II, related to conduction problems within the His-Purkinje system. This indicates that indeed trifascicular disease is present.

At this time, the patient should be stabilized and temporary pacemaker insertion considered as this is a ventricular escape rhythm that is potentially unstable and unreliable, resulting in wide variability in rate. Permanent pacemaker implantation is warranted thereafter. Provided there are no signs of active ischemia, more advanced device therapy for heart failure may be considered. Depending on the patient's left ventricular ejection fraction, an implantable cardioverter-defibrillator may be considered. As the patient's QRS complex is wide with an LBBB, cardiac resynchronization therapy may also be considered if the patient has systolic dysfunction and heart failure symptoms persist despite medical therapy.
A 77-year-old man is admitted to the hospital with syncope heralded by lightheadedness. On admission, an ECG is obtained (74A). During his first night in the hospital, the patient’s heart
rate slows and the medical resident notes a change in the appearance of his tracing on the telemetry monitor. She obtains a second ECG (74B).

ECG 74B

What abnormalities are evident?
What accounts for the change noticed by the resident?
What therapy is warranted?
ECG 74A Analysis: Normal sinus rhythm, left bundle branch block
In ECG 74A, there is a regular rhythm at a rate of 76 bpm. A P wave (+) precedes each QRS complex, and the PR interval is stable (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is prolonged (0.16 sec), and the morphology is that of a left bundle branch block (LBBB; broad R wave in leads I and V5-V6 [→] with a deep QS complex in lead V1 [+-]). The QT/QTc intervals are prolonged (440/495 msec) but are normal when the increased QRS complex duration is considered (360/405 msec).
ECG 748 Analysis: Normal sinus rhythm, AV dissociation/complete heart block, escape junctional rhythm with right bundle branch block and left posterior fascicular block
ECG 74B shows regular RR intervals at a rate of 42 bpm. P waves (*) can be seen, occurring at regular PP intervals (LI) and an atrial rate of 90 bpm. Some of the P waves are not obvious as they are superimposed on the QRS complex or located in ST segments or T waves (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The PR intervals are not constant (T) and are variable, indicating AV dissociation. Since the atrial rate is faster than the ventricular rate, this is complete (third-degree) AV block. The QRS complexes are wide (0.16 sec), and they have a typical right bundle branch block (RBBB) morphology (RSR' morphology in lead V1 [-] and broad S wave in leads I and V5-V6 [−]). The axis is rightward, between +90° and +180°. The QRS complex is negative in lead I (even when the terminal S wave is ignored) and positive in lead aVF. As there is an rS morphology in lead I, this is a left posterior fascicular block (LPFB). Since the QRS complexes are supraventricular in morphology (typical RBBB with normal initial forces), this is an escape junctional rhythm with an RBBB and LPFB. The complete AV block is the result of block within the AV node.

However, there has been a marked change in the QRS morphology in ECG 74B compared with ECG 74A: During sinus rhythm there is an LBBB, and with complete heart block the escape junctional rhythm has an RBBB and an LPFB. This indicates the presence of bi-bundle branch block or trifascicular disease in addition to disease within the AV node.

As this patient has significant conduction system disease, this represents a potentially unstable condition. Although the presence of complete heart block with an escape junctional rhythm may not require permanent pacing in the absence of symptoms, he does present with a history of syncope and lightheadedness, which warrants permanent pacemaker implantation. He also has evidence of trifascicular disease with symptoms, another indication for permanent pacemaker insertion.
A 31-year-old man presents to the emergency department with a complaint of palpitations associated with lightheadedness.

What abnormality is depicted?
**ECG 75 Analysis:** Normal sinus rhythm, Wolff-Parkinson-White pattern
Conduction Abnormalities: Practice Case 75

There is a regular rhythm at a rate of 86 bpm. There is a P wave (*) before each QRS complex, and the PR interval is constant but short (0.12 sec) (\(\square\)). The QRS complex duration appears to be normal in the limb leads (0.08 sec) but prolonged in the precordial leads (0.14 sec). Noted in precordial leads V2-V6 is a widening of the base of the QRS complex, resulting from a slurred upstroke or a delta wave (\(\triangle\)), that accounts for the QRS widening. A delta wave with short PR interval defines Wolff-Parkinson-White pattern.

The apparent absence of a delta wave, short PR interval, and widened QRS complex in the limb leads are due to the fact that the delta wave is isoelectric in these leads and hence not obvious. In lead II the waveform seen immediately after the P wave (\(\Delta\)) is indeed the delta wave. The Q waves in leads III and aVF (\(\ast\)) are also delta waves, although the Q waves could be confused with an inferior wall myocardial infarction. This is a pseudo-infarction pattern and indicates that the accessory pathway is in the posteroseptal wall. In Wolff-Parkinson-White pattern there is direct myocardial activation via the accessory pathway and hence ventricular abnormalities cannot be reliably diagnosed. It is important to note that the delta wave and short PR interval may, therefore, not be obvious in every lead.

The presence of symptoms of palpitations and lightheadedness is strongly suggestive of an arrhythmia. In patients with Wolff-Parkinson-White pattern on the ECG, the presence of symptoms suggesting an arrhythmia requires an evaluation. This should start with ambulatory or more extended monitoring. Many such patients undergo electrophysiologic testing to establish whether any supraventricular arrhythmia can be provoked. If so, radiofrequency ablation of the accessory pathway is often performed. Many such patients undergo radiofrequency ablation even if no arrhythmia is documented.
A 69-year-old man with a history of coronary artery disease presents to his cardiologist for a routine visit. He has no complaints except for brief episodes of lightheadedness that occur sporadically throughout the day. He has continued to take his medications, which include a β-blocker, aspirin, an angiotensin-converting enzyme inhibitor, and a long-acting nitrate. An ECG is obtained.

What does the ECG show?
ECG 76 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), Mobitz type II second-degree AV block, third-degree (complete) AV block, escape ventricular complexes, fusion complex, retrograde concealed conduction
There is an irregular rhythm at an average rate of 54 bpm; however, there are P waves (+) with a regular PP interval (U) at an atrial rate of 96 bpm, although some of the P waves are within the T waves and ST segments (▼). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is an underlying normal sinus rhythm. The QRS complexes have several different morphologies. QRS complexes 1, 2, 6, and 7 (+) have a prolonged duration (0.16 sec), while complexes 3, 4, 8, and 9 (▼) have a normal duration (0.10 sec). The fifth QRS complex (●) has a morphology that is different than both the wide and narrow QRS complexes, although it also resembles both of them; the duration of this QRS complex is 0.12 sec. The wide QRS complexes are associated with PR intervals that are variable (▼), indicating AV dissociation. As the atrial rate is faster than the ventricular rate, complete or third-degree AV block is present. The QT/QTc intervals of these QRS complexes are normal (400/380 msec).

The PR intervals before the third and eighth QRS complexes, which are narrow and occur earlier with a shorter RR interval, are the same (0.48 sec) (▼). Hence these are conducted and the QRS complex is thus the native complex. The PR intervals before the fourth, fifth, and ninth QRS complexes (0.36 sec) (▼) are also the same, meaning that these narrow complexes are also captured. Thus, there is complete heart block with intermittent AV conduction or capture. However, the PR interval of the first captured QRS complexes (complexes 3 and 8) is slightly longer (0.48 sec) than that of the subsequent captured QRS complexes (4 and 9) (0.36 sec). This is due to retrograde concealed conduction. That is, the preceding QRS complexes (2 and 7), which are ventricular in origin, have produced retrograde (ventriculoatrial) conduction into the AV node but do not completely conduct through the node; rather they partially penetrate the node (ie, they are concealed within the node), rendering it partially refractory. As a result, the next P wave is conducted at a slower rate and hence a slightly longer PR interval. Therefore, the PR interval (0.36 sec) before the fourth and ninth QRS complexes represents the baseline PR interval.

The fifth QRS complex has a morphology that resembles both the narrow and wide QRS complexes. The PR interval before this complex is the same as the PR interval before the fourth and ninth QRS complexes (0.36 sec; ie, the baseline PR interval). Therefore, this is a fusion complex, resulting from an impulse conducted through the AV node–His-Purkinje system that fused with the impulse coming from the ventricle. Fusion complexes reflect the presence of AV dissociation.

Finally, the narrow QRS complexes are associated with a pattern of 2:1 AV conduction (2:1 AV block); that is, every other P wave is nonconducted. Therefore, this is second-degree AV block with 2:1 AV conduction. As the escape rhythm is ventricular, the 2:1 AV block is the result of Mobitz type II second-degree AV block.
A 72-year-old woman presents to her primary care physician with complaints of palpitations. These episodes, which have become more frequent over the past several months, are unheralded and sometimes wake her from sleep. She has noted lightheadedness.

ECG 77A
on one occasion but has never lost consciousness. Her physician obtains an ECG during the initial portion of the visit (77A). She was asymptomatic at the time. During the interview, she notes the abrupt onset of palpitations. The physician obtains another ECG (77B).

**What rhythm does ECG 77A show?**

**Is a conduction abnormality present?**

**Is therapy indicated based on the finding in ECG 77B?**
ECG 77A Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), nonspecific T-wave abnormalities
In ECG 77A, there is a regular rhythm at a rate of 78 bpm. The QRS complex duration is normal (0.10 sec) and there is a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (380/430 msec). There are diffuse T-wave abnormalities (+) that are nonspecific. P waves are not obviously seen in most of the leads; however, there is a small waveform noted after the T wave in leads V1-V6 (*). On closer inspection this waveform can also be seen in the limb leads (^), altering the end of the T wave. This is the P wave and the PR interval is 0.36 second (\texttt{+++}), indicating a first-degree AV block (prolonged AV conduction).

continues
ECG 778 Analysis: Normal sinus rhythm, premature atrial complexes, first-degree AV block (prolonged AV conduction), nonspecific T-wave abnormalities
The rate in ECG 77B is 80 bpm, but the rhythm is regularly irregular as a result of two premature QRS complexes (second and sixth) that are followed by pauses. The QRS complex duration, morphology, and axis are the same as in ECG 77A, as are the QT/QTc intervals. Diffuse T-wave abnormalities are also noted and are the same as in ECG 77A. As with ECG 77A, small waveforms noted at the end of the T wave in leads V1-V3 are suggestive of P waves (*). The second and sixth QRS complexes are premature (!), and in leads II, III, and aVF small P waves (+) can be seen in the T waves preceding these two QRS complexes; the PR interval is 0.30 second (U). These are, therefore, premature atrial complexes. After the premature beat there is a pause that allows the sinus P wave to be seen clearly (^), confirming the presence of a normal sinus rhythm with a first-degree AV block and a PR interval of 0.36 second (\( \text{PR} \)). This is the same PR interval as that measured on ECG 77A. Importantly, P waves that are not obvious (as a result of first-degree AV block and tachycardia) may be seen superimposed on the T wave or after a pause or long RR interval, as was seen after the premature atrial complex.

It appears that the palpitations are the result of premature atrial complexes. These premature complexes are common, benign, and generally do not require therapy. However, in the presence of associated symptoms, medical therapy with a \( \beta \)-blocker, calcium-channel blocker, or anti-arrhythmic drug can be attempted.
A 76-year-old man with an underlying cardiomyopathy, believed to be the result of excessive alcohol use, presents to the emergency department with complaints of intermittent lightheadedness associated with shortness of breath. An ECG is obtained (78A), but no specific diagnosis is provided. He is scheduled for a
Holter monitor and referred to a cardiologist for further evaluation. The Holter monitor does not show any abnormality, but he had no symptoms while wearing it. When the man sees a cardiologist, he states that he has been experiencing lightheadedness that began the previous day. An ECG is obtained (ECG 78B).

Are there any findings of concern on ECG 78A?
What does ECG 78B show?
What therapy is indicated?
ECG 78A Analysis: Normal sinus rhythm, bi-bundle branch block (alternating right and left bundle branch block), trifascicular disease
ECG 78A shows a regular rhythm at a rate of 64 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. The QRS complex duration is increased (0.14 sec), and all but two QRS complexes (5 and 10) (+) have a right bundle branch block morphology with an RSR' morphology in lead V1 (−) and broad terminal S waves in leads I and V5-V6 (−). The axis is normal, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/410 msec and 340/350 msec when the prolonged QRS complex duration is considered). However, the fifth and 10th QRS complexes (+) have the same duration (0.14 sec) but a different morphology; they have a left bundle branch block morphology with a QS complex in lead V1 and a broad R wave in lead V5. The P wave and PR intervals before these complexes are the same as before all the other QRS complexes. Hence there is evidence of block in both the right and left bundles. This is termed bi-bundle branch block; it can also be termed trifascicular disease.

The presence of trifascicular disease or bi-bundle branch block, which indicates the presence of diffuse conduction disease involving the entire ventricular conduction system, increases the risk for complete heart block; this will be associated with an escape ventricular rhythm because an escape rhythm can only originate from below the His-Purkinje system (ie, the ventricular myocardium). The patient's symptoms of lightheadedness and shortness of breath are likely due to intermittent complete heart block with an escape rhythm that is slow.
**ECG 788 Analysis:** Normal sinus rhythm, complete heart block, escape ventricular rhythm, nonconducted (blocked) premature atrial complex
In ECG 78B, obtained while the patient was symptomatic, there is a regular rhythm at a rate of 40 bpm. P waves are seen (+), although some of the P waves are not obvious as they are within the QRS complexes or on the T waves (\(\downarrow\)). When the P waves are seen, however, the PP interval is constant (\(\uparrow\)) at an atrial rate of 98 bpm. The last P wave (\(\downarrow\)) is early and has a different morphology: it is a premature atrial complex that is nonconducted. Another premature P wave can be seen immediately before the fourth QRS complex (\(\downarrow\)). There is no relationship between the P waves and the QRS complexes (\(\text{i.e., the PR intervals are variable; \(\downarrow\}\)). Therefore, AV dissociation is present. As the atrial rate is faster than the ventricular rate, this is complete heart block (or third-degree AV block).

The QRS complexes are wide (0.16 sec), and their morphology is unlike that of the QRS complexes in ECG 78A (\(\text{i.e., they have neither a typical right or left bundle branch block morphology}\). Hence these are ventricular complexes.

As would be expected in the presence of bi-bundle branch block, the development of complete heart block is associated with an escape ventricular rhythm. The slow escape ventricular rhythm is very likely the cause of the patient’s symptoms. Hence the insertion of a permanent pacemaker is indicated.
A 75-year-old woman presents to the emergency department with dizziness and headache. Her blood pressure is noted to be 220/100 mm Hg. The exam is otherwise unremarkable except for a slow heart rate. An ECG is obtained (79A). She is admitted to the hospital for severe
hypertension and initiated on therapy with an angiotensin-converting enzyme (ACE) inhibitor, calcium-channel blocker, and β-blocker. After therapy her blood pressure is 140/80 mm Hg, and her pulse remains slow. However, telemetry shows a change and another ECG is obtained (79B).

**What accounts for the slow heart rate?**

**What therapy is necessary?**
ECG 79A Analysis: Normal sinus rhythm with ventriculophasic arrhythmia, first-degree AV block (prolonged AV conduction), 2:1 second-degree AV block, intraventricular conduction delay
ECG 79A shows regular RR intervals at a rate of 36 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (+) of 0.24 second (first-degree AV block or prolonged AV conduction). A second P wave (++) can be seen after each QRS complex. This P wave is nonconducted. The presence of an occasional nonconducted P wave defines second-degree AV block, and there is a pattern of 2:1 AV conduction. The atrial rate is not constant, and the PP interval surrounding the QRS complex (\( \Box \)) is shorter (0.70 sec) than the PP interval without a QRS complex (\( \mathbf{L} \)) (0.84 sec). This represents ventriculophasic arrhythmia. This finding can be seen whenever there is 2:1 AV block or complete heart block. Ventriculophasic arrhythmia is due to enhanced sinus node impulse generation resulting from ventricular contraction. This may be the result of increased sinus nodal artery pulsatile blood flow with ventricular contraction; stretching of the right atrium with ventricular contraction, resulting in an increase in sinus node automaticity; or baroreceptor changes resulting from the stroke volume occurring with ventricular contraction.

The QRS complexes are wide (0.11 sec) but are not wide enough to be a full left bundle branch block (LBBB; i.e., they are not \( \geq 0.12 \) sec). They do not demonstrate a pattern of a right bundle branch block nor do they have a typical LBBB pattern as there is a terminal S wave in lead V6 (\( \mathbf{A} \)) representing terminal forces in a left-to-right direction, which cannot be seen with an LBBB. Hence this is an intraventricular conduction delay (IVCD). There are nonspecific ST-T wave changes (\( \Box \)). The axis is leftward, between 0° and -30° (positive QRS complex in lead I, biphasic QRS complex in lead II, and negative QRS complex in lead aVF). The QT/QTc intervals are 500/390 msec (460/360 msec when corrected for the prolonged QRS complex duration).
ECG 798 Analysis: Normal sinus rhythm, complete AV block, escape junctional rhythm
In ECG 79B, there are regular RR intervals at a rate of 34 bpm. The atrial rate is constant at 72 bpm, and the P waves are positive in leads I, II, aVF, and V4-V5. The PR interval is not constant, and AV dissociation is present. AV dissociation with an atrial rate faster than the ventricular rate indicates complete heart block. The escape rhythm has a widened QRS complex (0.11–0.12 sec) that is almost identical in morphology to that seen in ECG 79A; hence this is an escape junctional rhythm. The slight difference in the QRS morphology of these junctional complexes when compared with the sinus complexes is due to the fact that the junctional complex originates from an ectopic focus within the AV junction and the impulse it generates enters the bundle of His at a different location when compared with the sinus impulse, which is conducted through the AV node. The difference in conduction pathway through the His-Purkinje system will result in a slightly different morphology, axis, or amplitude.

The etiology of the conduction problem with 2:1 AV conduction can be established when complete heart block develops; the origin of the escape rhythm indicates whether the block is within the AV node or is infra-Hisian (ie, within the His-Purkinje system). If the escape QRS complex is narrow or similar to the conducted QRS complexes, the 2:1 AV block is Mobitz type I due to conduction abnormality within the AV node. An escape QRS complex that is wide and abnormal is consistent with a ventricular focus and hence the 2:1 AV block is Mobitz type II with infra-Hisian disease. If ECG 79B were the only tracing available, it would be uncertain whether the escape rhythm was junctional with an underlying IVCD or a ventricular rhythm. In this situation an invasive electrophysiologic study would be required to determine the locus of the block. However, as ECG 79A is available and shows that the conducted complexes have the same morphology as the dissociated complexes in ECG 79B, it is clear that this is an escape junctional rhythm.

The 2:1 AV block is a result of underlying AV nodal disease. Severe hypertension can result in a decrease in sympathetic tone and an increase in vagal tone mediated through the baroreceptors and, therefore, can contribute to AV nodal conduction abnormalities. The repeat ECG shows complete heart block with an escape junctional rhythm that is possibly a result of β-blocker therapy in the presence of underlying AV nodal disease. The first step would be to discontinue the β-blocker. If conduction improves, then no further therapy is indicated. However, if complete heart block persists, indicating structural disease of the AV node, then a permanent pacemaker is indicated.
A 25-year-old woman with persistent cardiomyopathy following a pregnancy 6 months ago presents to her primary care physician with complaints of intermittent palpitations. Episodes are unheralded and without any other associated symptoms. She denies dyspnea. Of note, symptoms started 1 week ago, approximately 2 weeks after she self-discontinued her β-blocker. She states that she “doesn’t need those pills anymore.” An ECG is obtained during one of her episodes of palpitations.

What is the cause of the patient’s symptoms?
ECG 80 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), left bundle branch block, premature ventricular complex, complete heart block with ventricular escape, trifascicular disease
There is a regular rhythm at a rate of 62 bpm. There is a P wave (*) before the first four QRS complexes (†) with a constant PR interval of 0.24 second (↔) (first-degree AV block or prolonged AV conduction). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. The QRS complex is widened at 0.18 second, and it has a left bundle branch block pattern (broad R wave in lead I [−]). A single premature ventricular complex (‡) is followed by an on-time, nonconducted sinus P wave (+); thereafter, the QRS complexes are wider (0.20 sec), have a different morphology, and occur at a regular rate of 50 bpm. There are still sinus P waves at a regular interval (Π) and a rate of 62 bpm (∗, †), although on occasion they are not obvious because they are superimposed on QRS complexes, ST segments, or T waves (κ). There is no relationship between the P waves and QRS complexes, although the PP interval remains constant when the P waves are seen (Π). Therefore, there is AV dissociation and, as the atrial rate is faster (62 bpm) than the ventricular rate (50 bpm), there is complete heart block with an escape ventricular rhythm. Thus the patient’s symptoms are the result of the slow ventricular rhythm and not related to the discontinuation of β-blocker. A permanent pacemaker is warranted.

The patient has bifascicular disease given the presence of a left bundle branch block (ie, disease of the left anterior and left posterior fascicles). Diagnosing trifascicular block requires evidence of disease in the remaining fascicle. The presence of first-degree AV block (with the LBBB) is not sufficient to diagnose trifascicular block since the first-degree AV block could be due to disease of either the His-Purkinje system (ie, of the remaining fascicle) or the AV node. However, the ECG also demonstrates complete heart block with a ventricular escape rhythm. Since the escape rhythm comes from the ventricle (and is not junctional), this implicates disease of the His-Purkinje system. Therefore, trifascicular disease can be established in this patient. ■
A 48-year-old man with idiopathic dilated cardiomyopathy is undergoing routine evaluation by his cardiologist. An ECG is obtained. What abnormalities are noted?
Podrid's Real-World ECGs

ECG 81 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), left anterior fascicular block, intraventricular conduction delay, long QT interval
There is a regular rhythm at a rate of 60 bpm. There is a P wave (\(^{\uparrow}\)) before each QRS complex that can be seen superimposed on the end of the T wave (resulting in a notching of the downslope of the T wave); this is seen primarily in leads I, II, III, aVL, and V3-V6. The P wave is positive in leads I, II, and V4-V6. Hence this is a normal sinus rhythm. There is a stable but prolonged PR interval (\(\pm\)) (0.56 sec), indicating first-degree AV block or prolonged AV conduction. The QRS complex duration is prolonged (0.12 sec). Although the morphology of the QRS complex looks like a left bundle branch block (LBBB), there is a septal Q wave in leads I and aVL (\(\downarrow\)) and an initial septal R wave in lead V1 (\(\downarrow\)). Septal Q waves cannot be present with an LBBB as the septal branch (which innervates the septum in a left-to-right direction, accounting for the septal waveforms) comes from the left bundle. Hence this is an intraventricular conduction delay (IVCD).

The axis is extremely leftward, between \(-30^\circ\) and \(-90^\circ\) (positive QRS complex in lead I and negative QRS complex in leads II and aVF). The QRS complexes have an rS morphology in leads II and aVF. No Q wave is noted; therefore, the left axis is not the result of an inferior wall myocardial infarction. Hence this represents a left anterior fascicular block (LAFB). As this is not an LBBB, diagnosing an LAFB is possible. In the presence of an LBBB, an LAFB is not an appropriate diagnosis, since there is no conduction through either of the fascicles coming from the left bundle (ie, left anterior and left posterior fascicles). An LAFB does not prolong the QRS complex duration; therefore, the IVCD is an independent finding. The QT/QTc intervals are prolonged (500/500 msec) but are only slightly prolonged when the QRS complex duration is considered (460/460 msec).

Idiopathic dilated cardiomyopathy is often associated with conduction system disease due to the diffuse myocardial fibrosis that can occur. Although an LBBB may be seen, it is also common to see a nonspecific IVCD as a result of diffuse fibrosis of the left ventricle associated with slowing of His-Purkinje conduction. An LAFB is also commonly seen. The first-degree AV block may be due to AV nodal disease or possibly underlying disease of the right bundle branch as well as the left posterior fascicle (ie, trifascicular disease). There is no way on this surface ECG to establish the etiology for the first-degree AV block. ■
A 42-year-old man is admitted to the hospital with a non-ST-segment elevation myocardial infarction. He is rendered pain free with institution of medical therapy and is admitted to the step-down unit. During his initial hours of hospitalization, he remains stable, but a change in his QRS complexes is noted on telemetry. An ECG is obtained.

**What abnormality is noted?**

**What further action is warranted based on this ECG?**
ECG 82 Analysis: Normal sinus rhythm, rate-related or intermittent left bundle branch block
The rhythm is regular at a rate of 86 bpm. There is a P wave (*) before each QRS complex and the PR interval is constant at 0.16 second (LJ). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The first seven QRS complexes are wide (+) (0.14 sec) and have a left bundle branch block (LBBB) morphology (broad R wave in lead I [+]), while the last QRS complexes (−) are narrow and have a normal duration and morphology. This is an intermittent LBBB. Although the RR intervals of the wide and narrow QRS complexes appear to be the same, there are likely subtle differences, with the RR interval of the complexes with LBBB being slightly shorter. Hence this may represent a rate-related LBBB. The QT/QTc intervals of the narrow QRS complexes are normal (360/430 msec), and the QT/QTc intervals of the complexes with an LBBB are prolonged (400/480 msec) but are normal when the widened QRS complex duration is considered (340/410 msec).

The emergence of a rate-related LBBB in a patient with a myocardial infarction (ST-segment elevation or non-ST-segment elevation) suggests that there has been injury to the interventricular septum. Damage to the conduction system in this area can lead to fascicular block or even a bundle branch block. The rate-related LBBB is not a manifestation of ongoing ischemia, as the patient was at this point asymptomatic and there are no acute ECG changes after the resolution of the LBBB. This conduction abnormality is the result of damage due to the myocardial infarction. While the LBBB is intermittent or rate related at this point, it is possible that a permanent or persistent LBBB will develop in the future.
A 71-year-old woman is admitted with unheralded syncope. She states that she is not taking any cardiac medications. An ECG is obtained on admission.

What abnormalities are noted?
What pathology is suggested?
What therapy is warranted?
ECG 83 Analysis: Atrial fibrillation with slow ventricular response, intermittent left and right bundle branch block, bi-bundle branch block
The rhythm is slow and irregularly irregular at an average rate of 56 bpm. There are no P waves seen, but there is evidence of fibrillatory waves, seen best in lead V1 (*). The first three QRS complexes (*) and the fifth complex (*) are wide (0.12 sec) and have a right bundle branch block morphology (broad R wave in lead V1 [-] and broad S wave in lead I [+-]). The axis is rightward, between +90° and +180°. There is a negative QRS complex in lead I even when considering the broad terminal S wave, which is due to the right bundle branch block. This is consistent with a left posterior fascicular block (negative QRS complex in lead I and positive QRS complex in lead aVF). In contrast, the fourth and sixth through ninth QRS complexes (+), which are also wide (0.12 sec), have a left bundle branch block morphology (broad QS complex in lead V1 [\~] and broad R wave in lead V6 [\~\~]). The QT/QTc intervals are prolonged (480/464 msec) but are normal when the increased QRS complex duration is considered (440/425 msec).

The alternating right and left bundle branch block pattern indicates conduction block in both bundles and is termed bi-bundle branch block, which may also be called trifascicular disease. In addition, the patient has atrial fibrillation with a slow ventricular response rate in the absence of taking an AV nodal blocking agent. This represents high-grade conduction system disease (of the AV node and His-Purkinje system) and warrants permanent pacemaker implantation as the syncope was very likely due to transient complete heart block with an escape ventricular rhythm.
An ECG from a 32-year-old man with familial cardiomyopathy is shown.

What abnormalities are depicted?
Podrid's Real-World ECGs

ECG 84 Analysis: Normal sinus rhythm, intraventricular conduction delay
There is a regular rhythm at a rate of 62 bpm. There is a P wave (*) before each QRS complex with a consistent PR interval (0.20 sec). The QRS complex duration is prolonged (0.18 sec). Although the morphology resembles that of a left bundle branch block (LBBB; broad R wave in leads I [-] and V5-V6 and deep S wave in lead V1 [→]), there are septal Q waves (↑) in leads I and aVL and a septal R wave (↑) in lead V1. Septal forces cannot be seen with an LBBB as the septal (median) fascicle, which innervates the interventricular septum in a left-to-right direction (accounting for the initial septal Q waves in leads I, aVL, and V5-V6 and the septal R wave in lead V1), comes from the left bundle. Thus with an LBBB there is no initial septal activation. Hence this is not an LBBB but is an intraventricular conduction delay (IVCD). The QT/QTc intervals are prolonged (520/530 msec) but are normal when corrected for the increased QRS complex duration (420/430 msec).

With an LBBB, activation of the left ventricle is not via the normal His-Purkinje system but rather occurs directly through the ventricular myocardium, and hence the activation sequence is abnormal. Abnormalities of the left ventricular myocardium cannot be diagnosed reliably with an LBBB. In contrast, the presence of an IVCD indicates that left ventricular activation is occurring via the normal conduction system but is diffusely slowed. Since the impulse travels along the normal His-Purkinje system, abnormalities that affect the left ventricle can be identified (eg, axis shift, acute and chronic myocardial infarction, ischemia, left ventricular hypertrophy, pericarditis). Commonly a wide QRS complex is the result of an underlying cardiomyopathy and is due to diffuse myocardial fibrosis and therefore marked slowing of impulse conduction. There is in fact a correlation between the left ventricular ejection fraction and the QRS complex duration with an IVCD.
A 30-year-old woman with known congenital heart disease and significant left-to-right shunting that has now progressed to Eisenmenger's syndrome presents for a routine physical exam. Her only complaint is that of intermittent palpitations. Her ECG is shown.

What is the abnormality?
Podrid's Real-World ECGs

ECG 85 Analysis: Atrial tachycardia, right ventricular hypertrophy, right axis
There is a regular rhythm at a rate of 110 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is upright in leads I and V4-V6. However, it is negative in lead aVF (−) and biphasic in lead II (+), indicating that the rhythm is not sinus but rather originates from the low atrium (ie, negative P wave in lead aVF). Hence this is atrial tachycardia. The QRS complex duration is normal (0.10 sec). However, there is a tall R wave in lead V1 (12 mm) (−) and an R/S ratio in leads V5-V6 < 1 (ie, the S wave is deeper than the R-wave height). These are criteria for right ventricular hypertrophy. The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). In this case, the right axis is due to right ventricular hypertrophy and not a left posterior fascicular block.

Patients with congenital heart disease who have significant left-to-right shunting can develop severe pulmonary arterial hypertension and subsequent right ventricular hypertrophy (RVH). RVH is often associated with a shift in the electrical axis to the right, as is seen in this ECG. With the development of significant pulmonary arterial hypertension and RVH, the shunting becomes right to left, and this is Eisenmenger's syndrome. Atrial tachycardia is often seen in this condition, reflecting abnormalities of the right atrium. As the atrial tachycardia results from an ectopic focus, the rate of the tachycardia may vary and fast rates may account for the patient's palpitations.
A 72-year-old man with known multi-vessel coronary disease presents for routine follow-up. He has stable New York Heart Association class II heart failure. He has no new complaints. His vital signs and physical exam are normal. As part of his evaluation, an ECG is obtained.

What abnormalities are noted?
Is any additional evaluation or therapy necessary?
ECG Analysis: Normal sinus rhythm, left posterior fascicular block, long QT interval (delayed repolarization), nonspecific T-wave abnormality
The rhythm is regular at a rate of 74 bpm. There is a P wave (*) before each QRS complex, and the PR interval is stable at 0.20 second. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration (0.08 sec) and morphology are normal. The QRS complex is negative in lead I (rS morphology) and positive in lead aVF. Hence the axis is rightward, between +90° and +180°. In the absence of any other cause for a right axis (ie, right ventricular hypertrophy, lateral wall myocardial infarction, dextrocardia, right–left arm lead switch, Wolff-Parkinson-White pattern), the etiology for this is a left posterior fascicular block (LPFB). The left posterior fascicle is a broad, fan-shaped extension of the main left bundle that spreads along the inferoposterior aspect of the left ventricle. Since it is broad and diffuse, an LPFB is not common and is seen far less often than a left anterior fascicular block. No additional evaluation or therapy for the LPFB is necessary. However, the long QT interval suggests either low calcium or magnesium levels. These levels should be checked and, if low, repleted.

The concept of the hemiblock, or fascicular block, was put forth by Rosenbaum and colleagues in 1970. The definition of a fascicular block was based only on the electrical axis of the heart in the frontal plane. An LPFB was defined as a right QRS axis, between +90° and +180°. The left posterior fascicle is a broad, fan-shaped extension of the main left bundle that spreads along the inferoposterior aspect of the left ventricle. Since it is broad and diffuse, an LPFB is not common and is seen far less often than a left anterior fascicular block. No additional evaluation or therapy for the LPFB is necessary. However, the long QT interval suggests either low calcium or magnesium levels. These levels should be checked and, if low, repleted.
A 17-year-old who is trying out for his high school basketball team presents for a routine physical exam. He denies any symptoms or past medical conditions. As part of the screening, an ECG is obtained and he is told that he is not eligible to try out for the team.

What is the abnormality of concern?
Is additional testing warranted?
Should he be permitted to try out for the team?
Podrid's Real-World ECGs

ECG 87 Analysis: Normal sinus rhythm, intermittent preexcitation (Wolff-Parkinson-White pattern)
Conduction Abnormalities: Practice Case 87

There is a regular rhythm at a rate of 68 bpm. There are P waves (+) before each QRS complex, and they are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. There are two different PR intervals (0.12 sec [\(\square\)] and 0.20 sec [\(\blacksquare\)]). There are also two different QRS complex morphologies. Complexes 1 to 4 and 7 to 10 are wide with a duration of 0.16 second; these complexes are associated with the short PR interval (0.12 msec). They are wide as a result of a slowed upstroke (+). A wide QRS complex with a sinus P wave and a short PR interval is the hallmark of preexcitation (i.e., a Wolff-Parkinson-White [WPW] pattern). The slowed upstroke is the result of a delta wave, which is quite prominent in this patient. The delta wave is due to initial myocardial activation via the accessory pathway that occurs before ventricular activation via the normal AV node-His-Purkinje system (hence preexcitation). As a result of preexcitation, abnormalities of the left ventricle cannot be diagnosed and hence no further analysis of the QRS complex is possible. The fifth and sixth QRS complexes have a normal duration (0.08 sec) and morphology. They have a normal PR interval (0.20) msec. They likely have a normal axis, between 0° and +90° (positive QRS complex in leads II and aVF).

These are, therefore, normal complexes due to ventricular activation via the normal conduction system. Therefore, there is an intermittent WPW pattern. The QT/QTc intervals of the preexcited complexes are prolonged (460/490 msec) but are normal when the prolonged QRS complex duration is considered (400/425 msec). The QT/QTc intervals of the non-preexcited QRS complexes are normal (400/425 msec).

The presence of an intermittent WPW pattern generally means that the accessory pathway has a relatively long refractory period. In this situation the accessory pathway is not likely to conduct impulses rapidly. Importantly, should atrial fibrillation develop, it is not likely that it would be associated with very rapid impulse conduction to the ventricles. The rapid ventricular activation during atrial fibrillation is the mechanism for sudden death in these patients. As he has never had any symptoms to suggest an arrhythmia, there is no reason for any additional electrophysiologic evaluation in this asymptomatic young patient and there is no reason to prevent him from partaking in sports. Indeed, only about 10% of patients with a WPW pattern on the ECG have documented arrhythmia. ■
A 71-year-old man is undergoing treadmill testing. His complaint is that of decreasing exercise tolerance and fatigue. He has no known medical diagnoses and does not take any medications. His physical exam is notable only for a resting heart rate of 58 bpm. His baseline ECG is notable only for a first-degree AV block. Presented is his ECG during peak exercise.

What abnormality is noted?

What therapy is indicated?
Podrid's Real-World ECGs

ECG 88 Analysis: Sinus tachycardia, complete heart block, escape junctional rhythm, low limb lead voltage
There are regular RR intervals at a rate of 46 bpm. There are P waves (*) with a regular PP interval (\( \square \)) at a rate of 124 bpm. Although there are occasional P waves that are not seen because they are simultaneous with the QRS complex or T wave (+) (the measured PP interval when the P wave is not seen is thus equivalent to two PP intervals), the PP interval remains regular. The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is sinus tachycardia. The PR intervals are variable (\( \square \)); thus AV dissociation is present. The QRS complex duration is normal (0.08 sec) and it has a normal morphology and axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (520/440 msec).

There is low voltage in the limb leads (< 5 mm in each lead). The atrial rate is faster than the ventricular rate. Hence this is complete heart block with an escape junctional rhythm. The site of the block is the AV node.

In patients with early disease of the AV node, the node may show signs of disease prior to the onset of complete heart block only in the context of increased stimulation. During exertion, as the sinus node rate rises, the normal AV node will conduct at a commensurate increased rate with a shortening of the PR interval (on average 5 msec for each 10-beat increase in sinus rate). The diseased AV node may not be able to conduct impulses at an elevated rate, leading to progressive prolongation of the PR segment and even complete heart block, as in this patient. With the complete AV block the escape junctional rhythm may have an inappropriately slow rate if there is intrinsic junctional disease. This is one form of "chronotropic incompetence." Although his sinus rate increases appropriately, he develops complete AV block and an escape junctional rhythm with a rate that does not increase appropriately in response to exercise and increased catecholamines. This is likely the cause of this patient’s exertional intolerance. Implantation of a dual-chamber pacemaker that is programmed to function as a P-wave–synchronous or P-wave–activated ventricular pacemaker should alleviate the symptoms.
A 72-year-old woman with longstanding hypertension, treated only with a β-blocker, undergoes a routine physical exam. During the exam, an irregular radial pulse is noted and an ECG is obtained.

What abnormality on the ECG explains the physical exam findings?

What consequences of the patient's hypertension are evident on the tracing?
**ECG 89 Analysis:** Normal sinus rhythm, P mitrale (left atrial hypertrophy or abnormality), first-degree AV block (prolonged AV conduction), Mobitz type I second-degree AV block (Wenckebach), left ventricular hypertrophy, normal early repolarization
There are P waves (*), and the PP interval is fairly regular (L-) at a rate of 88 bpm. Occasionally, the P wave is superimposed on the T wave or is immediately after the T wave (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence there is a normal sinus rhythm. In addition, the P waves are abnormal; they are broad and notched in leads II, aVF, and V3-V5 (ie, P mitrale due to left atrial hypertrophy). The QRS complexes (at an average rate of 54 bpm) are regularly irregularly with a variable pattern. There is grouped beating with a pause that is the result of on-time but nonconducted P waves (+), which as stated above are occasionally superimposed on the T wave, altering the morphology. There are P waves before each QRS complex, but the PR interval (L-) is prolonging from a baseline of 0.28 second to 0.30 second. The PR interval (L-) after the pause represents the baseline PR interval (ie, 0.28 sec). Therefore, there is first-degree AV block (prolonged AV conduction). Second-degree heart block is also present (occasional nonconducted P wave), and the pattern of progressive PR interval prolongation is diagnostic for Mobitz type I or Wenckebach second-degree AV block. The pattern of AV block is variable, however, and there is 2:1, 3:2, and 4:3 conduction present.

The QRS complex duration is normal (0.08 sec) and there is a normal axis, between 0° and +90° (positive QRS complex in leads I and aVF). The QRS complex amplitude is increased, especially in lead V4 (38 mm) ( ), and this meets one of the criteria for left ventricular hypertrophy (ie, S-wave depth or R-wave amplitude in any precordial lead ≥ 25 mm). The J-point and ST-segment elevation (j) seen in leads V3-V4 represents early repolarization, frequently seen with left ventricular hypertrophy. The QT/QTc intervals are normal (440/420 msec).

Longstanding elevation in ventricular and, by extension, atrial pressures results in left ventricular and left atrial hypertrophy, which are evident on the surface ECG in this case. These structural alterations may result in conduction abnormalities as the specialized cardiomyocytes and their intracellular connections that comprise the conduction system are injured and fibrose over time. In addition, the β-blocker alters AV nodal conduction and may be a contributing factor to the occurrence of Wenckebach. Although Wenckebach usually occurs with a fixed pattern of AV block, there may be variable AV block present. ■
An 85-year-old man presents to the emergency department because of chest discomfort that occurred about 1 hour before presentation and lasted for 35 minutes. He states that the chest discomfort was similar to what he experienced before his heart attack 5 years ago. His physical exam is unremarkable. Vital signs are stable.

Cardiac biomarkers are obtained and they are negative. He is admitted to the hospital to “rule out” a myocardial infarction (MI) and for observation. Laboratory tests and cardiac biomarkers obtained 4 and then 8 hours later are unremarkable. His admission ECG is shown.

Did he have a previous MI?
If so, what is the location of the infarction?
What other abnormalities are seen?
Podrid's Real-World ECGs

ECG 90 Analysis: Atrial fibrillation, indeterminate axis due to an old lateral wall MI and left anterior fascicular block, intraventricular conduction delay, nonspecific ST-T wave changes
The rhythm is irregularly irregular, and there is no organized atrial activity or P waves. The average ventricular rate is 60 bpm. Occasional low-amplitude undulations can be seen (*). Hence this is atrial fibrillation. The QRS complex duration is almost 0.12 second, but there is no pattern of a left or right bundle branch block. Hence this is an intraventricular conduction delay. The QT/QTc intervals are normal (420/420 msec). There are nonspecific ST-T wave changes in leads V4-V6 (*). The axis appears to be indeterminate, between −90° and +180° (negative QRS complex in leads I and aVF). However, the initial waveform in leads I and aVL is a Q wave (QS morphology) (†), and hence this is an old lateral wall myocardial infarction (MI). The presence of an rS complex in lead I would suggest an axis shift (to the right) due to a conduction abnormality. In contrast, a QS or Qr complex in lead I is the result of a lateral wall MI and does not represent a conduction problem accounting for the axis shift. If this QRS complex is not considered, the axis is actually extremely leftward, between −30° and −90° (negative QRS complex in leads II and aVF; both have an rS morphology), this is termed a left anterior fascicular block. Hence this patient has a left anterior fascicular block and an old lateral wall MI, which together give the appearance of an indeterminate axis.

When there is a supraventricular rhythm, the presence of an indeterminate axis will only occur when there are two coexisting abnormalities, as there is no conduction pattern through the normal His-Purkinje system that will produce this axis. Conditions associated with an indeterminate axis, in addition to what is present in this patient, include right ventricular hypertrophy with an inferior wall MI or a left anterior fascicular block, a left posterior fascicular block associated with an inferior wall MI, or both an inferior and lateral wall MI. Another situation in which there is an indeterminate axis is a right–left arm lead switch associated with a left anterior fascicular block or an old inferior wall myocardial infarction.
A 78-year-old man presents to his physician with dyspnea on exertion and general fatigue for the past several weeks. He states that his symptoms have been variable, with days of profound fatigue with minimal exertion and days of full energy. A review of systems is otherwise unremarkable, and the patient specifically denies angina as well as orthopnea. An ECG is obtained as part of his evaluation.

What abnormalities on the ECG might explain his symptoms?
ECG 91 Analysis: Normal sinus rhythm, third-degree (complete) heart block, escape ventricular rhythm, intermittent capture
There are regular RR intervals at a rate of 34 bpm. Regular P waves (*) are seen with a stable PP interval (LJ) at a rate of 64 bpm. Some of the P waves are not obvious because they are superimposed on the QRS complex, in ST segments, or on T waves (+). However, P waves that are seen are at a regular interval (LJ). The P waves are positive in leads I, II, aVF, and V4-V6; hence there is an underlying normal sinus rhythm at a rate of 64 bpm. The PR intervals are variable (n), indicating AV dissociation. The atrial rate is faster than the ventricular rate; hence there is complete (third-degree) AV block. The QRS complexes are wide (0.14 sec) and have an unusual right bundle branch block pattern (RSR' morphology in lead V1 [+-] but no broad S wave in lead I). The second complex (*) has a normal duration (0.08 sec) and morphology. There is an on-time sinus P wave (▼) preceding this complex with a PR interval of 0.20 second (▼▼). This is a conducted complex and it has a normal morphology that is different than that of the dissociated QRS complexes. This confirms the fact that the dissociated QRS complexes are ventricular; hence the escape rhythm is ventricular. Not uncommonly, complete heart block may be associated with intermittent capture, or indeed the complete heart block itself may be intermittent. The first QRS complex (▼) is also wide but has a morphology that is different from the dissociated QRS complexes that are ventricular. This is also a ventricular complex that is from a different location within the left ventricle.

Ventricular escape rhythms are inherently unstable as they are an unprotected focus within the ventricular myocardium. Hence there is no control over the rate at which this ventricular focus can stimulate the ventricle; the rate may vary widely, from very slow to fast, depending on sympathetic tone and circulating catecholamines. This is in contrast to supraventricular rhythms, in which the ventricular response rate is controlled by the rate of conduction through the AV node. Escape ventricular rhythms also signify disease of the His-Purkinje conduction system. Hence complete heart block with a ventricular escape rhythm is an indication for pacemaker implantation, even if the patient is asymptomatic. In this patient, the escape ventricular rhythm, although quite bradycardic, did not result in pre-syncope or syncope. However, during times of even minimal exertion, this escape ventricular rhythm may not mount an appropriate heart rate response or increase in stroke volume and may not be able to supply the cardiac output required to meet demand, resulting in dyspnea and fatigue. This phenomenon of a limitation in cardiac output by bradycardia has been referred to as chronotropic incompetence.
A 50-year-old woman with known idiopathic first-degree AV block is seen by her cardiologist for a routine appointment. Her condition has been stable for years. The patient states that she has been feeling well overall and only notes some mild dysmenorrhea for which she recently began taking an herbal supplement. It seems to have helped. On physical exam, the heart rate is 46 bpm, consistent with prior assessments. Her exam is otherwise normal. An ECG is obtained.

What abnormalities are evident?
ECG 92 Analysis: Sinus bradycardia, complete heart block with junctional escape rhythm, intermittent AV conduction (capture) with first-degree AV block (prolonged AV conduction), U waves
There are regular RR intervals at a rate of 46 bpm. The QRS complexes have a normal duration (0.08 sec) and morphology. They have a left axis, between 0° and -30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (420/370 msec). There are low-amplitude U waves in leads V3-V6 (+). U waves are seen most commonly in leads V1-V3 and are unusual in the lateral precordial leads; their presence may be due to the bradycardia, although they may also suggest hypokalemia.

There are P waves (+,+) with a fairly constant PP interval (LJ) at a rate of 50 bpm. Some of the P waves are less obvious, being superimposed on the QRS complex or within the ST segment (+). The P waves are positive in leads I, II, aVF, and V4-V6. Hence they are sinus P waves and the underlying rhythm is sinus bradycardia. There is no consistent relationship between the P waves and the QRS complexes (ie, variable PR intervals [\(\square\)]), and hence AV dissociation is present. The atrial rate is faster than the ventricular rate; therefore, there is complete (third-degree) AV block. As the QRS complex duration and morphology are normal, these are supraventricular complexes and, therefore, there is an escape junctional rhythm. The last two QRS complexes (7 and 8) (+,), which have the same morphology as the others, are early and they are occurring at a rate of 50 bpm. There is an on-time P wave before each of these complexes (+) with a stable PR interval (+,+) (0.28 sec). This indicates that they are captured and hence sinus complexes with a first-degree AV block (prolonged AV conduction). Overall, this ECG demonstrates complete (third-degree) AV block with an escape junctional rhythm and intermittent capture. Since the escape rhythm is junctional, the cause for the complete heart block is disease within the AV node.

Certain herbal preparations contain compounds that can inhibit cardiac conduction by altering autonomic tone. Passion flower and chamomile may have \(\beta\)-adrenergic-blocking effects. Cohosh, used for dysmenorrhea, exhibits enhanced vagal effects. Interactions between unregulated herbal and “natural” supplements and medications as well as underlying organic disease must be considered. In this case, the patient’s conduction disease, evidenced by marked first-degree AV block when AV conduction is intact, may have been exacerbated by the vagal effects of her new herbal medication, resulting in a complete heart block at the level of the AV junction. Fortunately, she has no infra-Hisian disease and the AV junctional escape rhythm is intact.
A 72-year-old man who has no previous cardiac history presents to the emergency department with severe pleuritic chest pain. His lung exam is unremarkable. Laboratory values are all within normal limits. An ECG is obtained. As there is a concern about a pulmonary embolism, he undergoes angiography, which is unremarkable. He is admitted to the hospital for further workup.

What does the ECG show?

Based on the ECG, is any further evaluation warranted?
ECG 93 Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), right bundle branch block, old inferior wall myocardial infarction, right–left arm lead switch
There is a regular rhythm at a rate of 96 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.28 sec). The P waves are positive in leads II, aVF, and V4-V6. However, they are negative in leads I and aVL (-), while they are positive in lead aVR. This is an unusual P-wave axis and suggests that there may be an ectopic atrial focus or that there is a right-left (R-L) arm lead switch. The QRS complexes have a widened duration (0.16 sec) and a morphology of a right bundle branch block (RSR' morphology in lead V1 [-] and terminal broad S waves in leads V5-V6 [-]). However, the morphology in lead I is strange, as there is a deep QS complex (!) as well as a terminal broad R wave (i). Because there is a right bundle branch block morphology in the precordial leads, the QRS complex is the result of an R-L arm lead switch. If the leads were properly placed, lead I would have a normal R wave with a broad terminal S wave and lead aVR would have a normal Q wave. The lead switch also accounts for the negative P wave in lead I and the positive P wave in lead aVR. The axis appears to be indeterminate, between −90° and +180° (negative QRS complex in leads I and aVF). However, since there is a switch present, the QRS complex is not actually negative in lead I. Moreover, the QRS complexes in leads II and aVF have a QS morphology, which is characteristic for an old inferior wall myocardial infarction. This accounts for what looks like a left axis or left anterior fascicular block (negative QRS complex in leads II and aVF, not considering lead I). Hence the indeterminate axis in this patient is not indicative of a conduction problem; rather it is the result of R-L arm lead switch and an old inferior wall myocardial infarction. No fascicular or other conduction abnormalities are present. The R-L arm lead switch should be confirmed by repeating the ECG with the leads on the correct limbs. Otherwise, no other workup is necessary. The QT/QTc intervals are prolonged (400/510 msec) but are normal when the prolonged QRS complex duration is considered (320/405 msec).

R-L arm lead switch generally affects leads I and aVL (negative P wave, QRS complex, and T wave) and aVR (positive P wave, QRS complex, and T wave). Leads II, III, and aVF are usually not affected.
This ECG is from a 48-year-old man with advanced multivessel coronary disease and ischemic cardiomyopathy.

What portions of the cardiac conduction system have been affected by his disease?

What other abnormalities are depicted?
Podrid's Real-World ECGs

ECG 94 Analysis: Normal sinus rhythm, left posterior fascicular block, right bundle branch block, premature ventricular complexes
The ECG shows a regular rhythm at a rate of 80 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence, there is a normal sinus rhythm. The QRS complex duration is prolonged (0.16 sec), and there is a pattern of a right bundle branch block (RBBB; broad R wave in lead V1 [–+] and broad terminal S wave in leads I and V5-V6 [–]). The QT/QTc intervals are prolonged (400/460 msec) but are normal when the prolonged QRS complex duration is considered (320/370 msec). The axis is rightward, between +90° and +180°. The QRS complex is negative in lead I (even when the S wave [+] due to the RBBB is not considered) and positive in lead aVF. Hence there is a left posterior fascicular block. It should be remembered that with an RBBB there is a terminal broad S wave in lead I that may give the appearance of a negative QRS complex but actually represents delayed right ventricular activity and is not considered part of axis determination. The RBBB and left posterior fascicular block are indicative of bifascicular block.

In addition there are three complexes that are premature, are wider, and have a different morphology (†). An on-time sinus P wave can be seen before the first and third of these premature complexes (¶); however, the PR interval is very short and hence there is not likely to be AV conduction. These premature complexes are followed by a full compensatory pause (ie, the PP interval around the premature complex [LI] is twice the underlying sinus [PP] interval). These are premature ventricular complexes and since the morphology of all three is the same they are unifocal.

The presence of bifascicular block indicates more advanced conduction system disease. In this case, it is related to the presence of an ischemic cardiomyopathy. Bifascicular block due to left posterior fascicular block is less commonly seen than bifascicular block due to left anterior fascicular block; however, it has the same implications with regard to the development of complete heart block. While such patients are at increased risk for developing complete heart block, the incidence of this is low. In several studies, the incidence of progression to complete AV block was about 1% to 1.5% per year. Indeed, mortality in these patients, often due to arrhythmia, was related to the underlying heart disease and not to the development of complete heart block. There is no specific therapy for bifascicular block except for discontinuation or avoidance of drugs that may further impair cardiac conduction. Pacemakers are not indicated for asymptomatic patients. However, the insertion of a pacemaker is considered (class II indication) for patients with a bifascicular block associated with syncope that can be attributed to transient complete heart block, based on the history and the exclusion of other plausible causes of syncope (specifically ventricular tachycardia in a patient with coronary disease, an old myocardial infarction, and ischemic cardiomyopathy).
A 56-year-old man admitted for abdominal pain is diagnosed with acute cholecystitis. He is treated with intravenous fluids and antibiotics. An ECG is obtained and the resident calls the cardiology fellow for advice about what it shows.

What is the abnormality?
What diagnostic medication could be given to further evaluate this abnormality?
Is any therapy warranted?
ECG 95 Analysis: Normal sinus rhythm, isorhythmic AV dissociation, junctional rhythm
There is a regular rhythm at a rate of 94 bpm. There is a P wave (+) before each QRS complex, and the P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The PR interval is very short, but it is not constant (LJ); the PR intervals are becoming progressively shorter (from 0.12 to 0.08 sec). Hence AV dissociation is present. The ventricular rate is also 94 bpm, and the QRS complex has a normal duration (0.08 sec) and morphology. The axis is normal, between 0° and +90°. Hence these are junctional complexes and this is a junctional rhythm.

With AV dissociation there is no relationship between the atrial and ventricular activity (ie, the P waves are dissociated from the QRS complexes). Hence there are variable PR intervals. There are two etiologies for AV dissociation. The first is complete heart block (third-degree AV block), in which the atrial rate is faster than the ventricular rate because there is an escape rhythm (junctional or ventricular). The second is an accelerated lower pacemaker focus (junctional or ventricular), in which case the atrial rate is slower than the ventricular rate. When the two rates are identical, the etiology for AV dissociation is unclear; this is termed isorhythmic dissociation.

In general, isorhythmic dissociation is a benign condition and no specific therapy is needed because in this case there is a stable junctional rhythm. The etiology for the isorhythmic dissociation can be established if there is a change in either the atrial or ventricular rate and observing a difference between the two. This might happen spontaneously. If not, atropine might be effective as it will increase the sinus rate but will have no effect on the junctional rate. Hence, if there is capture and a stable PR interval with the faster atrial rate, the etiology is an accelerated junctional rhythm. If AV dissociation persists at the faster sinus rate, the diagnosis is complete heart block.
A 32-year-old man with HIV presents to his infectious disease physician with complaints of recurrent pre-syncope. The episodes are unheralded and worsen during exercise or exertion. He further notes some decrement in his functional capacity due to an indolent onset of exertional dyspnea. On exam, his vital signs are notable for a radial pulse of 80 bpm and his blood pressure is normal. The physical exam is notable for temporal wasting. His lung fields are clear. Jugular venous pressure is 5 cm H₂O. His cardiac exam reveals a diffuse point of maximal impulse that is displaced 2 cm laterally and a soft, blowing systolic apical murmur. Gallop sounds are not heard. His abdominal exam is unremarkable. His extremities show some wasting and fat redistribution. The physician obtains a surface 12-lead ECG.
ECG 96 Analysis: Normal sinus rhythm, left atrial hypertrophy (abnormality), first-degree AV block (prolonged AV conduction), 2:1 and 3:2 Mobitz type I second-degree AV block.
The initial portion of the ECG shows a regular rhythm at a rate of 38 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (+) of 0.30 second, defining first-degree AV block (prolonged AV conduction). A left atrial abnormality is present based on the negative P wave (**) in leads V1-V2. A second P wave (+) can be seen after the T wave. The PP interval is regular (|=), and the atrial rate is 80 bpm. The P waves are positive in leads I, II, aVF, and V4-V6, defining a normal sinus rhythm. Hence initially there is a second-degree AV block with a pattern of 2:1 AV conduction. However, another pattern is seen at the end of the tracing. There is an early QRS complex (\(|\) with a PR interval (\(\Delta\)) that is now prolonged to 0.48 second. The occurrence of an early QRS complex means that it is in response to the preceding P wave (\(\triangledown\)). There is an on-time but nonconducted P wave (\(\uparrow\)) following this early QRS complex (by measuring the PP interval [\(\square\)] it can be seen that the P wave is on the T wave, altering its morphology).

This represents second-degree AV block with a Wenckebach pattern and 3:2 conduction. Therefore, in this case the 2:1 AV conduction is the result of Wenckebach or Mobitz type I. With 2:1 AV block, the etiology (Mobitz type I or II) cannot be established unless there is a change in the conduction pattern (with two or more sequentially conducted P waves), as occurred in this case. The QRS duration is normal (0.08 sec) and there is a physiologic left axis, between 0° and ~30° (positive QRS complex in leads I and II and negative QRS complex in lead aVF). The QT/QTc intervals are normal (420/334 msec).

HIV-associated cardiomyopathy is a well-described entity in patients with AIDS, particular in those with prolonged duration after the time of diagnosis. The prevalence of cardiomyopathy is approximately 10% in industrialized nations. Etiologies include direct effects of the HIV virus with associated myocarditis, opportunistic pathogen-associated myocarditis, and highly active antiretroviral therapy (HAART)-associated cardiomyopathy. This patient manifests signs on physical exam of a dilated left ventricle and systolic dysfunction. In that context, relatively benign AV block such as first-degree and second-degree Mobitz type I (Wenckebach) as seen in this patient can be symptomatic. Pacemaker implantation may be considered in these patients (whereas it is generally not indicated in patients who have normal left ventricular function). Initial evaluation and therapy should include echocardiographic assessment of left ventricular function, inpatient institution of medical therapy for systolic heart failure, and consideration of pacemaker implantation if his conduction abnormality persists and the pre-syncopal symptoms are not relieved by initiation of heart failure therapy. His antiretroviral medications should be reviewed for those known to cause left ventricular dysfunction, such as nucleoside reverse-transcriptase inhibitors. A complete review of his HIV status, viral load, and compliance/response to antiretroviral therapy is warranted.
A 56-year-old man is admitted to the hospital for pneumonia. While on telemetry, the nurse notices variation in the patient’s surface ECG tracing. She obtains a 12-lead ECG.

What abnormalities are noted?
ECG 97 Analysis: Atrial fibrillation, left anterior fascicular block, rate-related right bundle branch block
The rhythm is irregularly irregular at an average rate of 78 bpm. There are no organized P waves, but there are occasional low-amplitude unorganized undulations of the baseline (■), best seen in the lead V1 rhythm strip. The rhythm is, therefore, atrial fibrillation. Two QRS complex morphologies are seen. The narrower complexes (△), with a duration of 0.10 second and an axis between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology), is a left anterior fascicular block. These complexes have a narrow R’ morphology in lead V1 (→) and an S wave in leads I and V5-V6 (→), suggesting a right ventricular conduction delay (although this morphology is often referred to as an incomplete right bundle branch block [RBBB]). The wider QRS complexes (+) have a duration of 0.16 second with a typical RBBB pattern (broad R wave in lead V1 [▼] and broad S wave in leads I and V5-V6 [■]). Hence this is evidence for bifascicular disease (ie, RBBB and left anterior fascicular block). The RBBB occurs whenever the RR interval is shorter (+), that is, with a faster ventricular rate. The RBBB is not present when the RR intervals are longer (■). Therefore, this is a rate-related RBBB. The QT/QTc intervals of the narrower QRS complex are normal (400/456 msec and 380/430 when corrected for the slightly prolonged QRS complex duration). The QT/QTc intervals for the complexes with an RBBB are the same (460/525 msec and 380/430 when corrected for the prolonged QRS complex duration).

Intermittent or rate-related RBBB is indicative of underlying conduction disease of the right bundle that becomes manifest only when the heart rate is faster. In this situation, the diseased bundle is unable to conduct impulses that are at rapid rates and hence block within the bundle develops. It is often the precursor of a permanent RBBB and has the same implications and prognosis of a persistent RBBB and a persistent bifascicular block. ■
A 55-year-old woman is admitted with transient syncope. The episode was unheralded and lasted a few seconds. She has otherwise been well, improving gradually after presenting 2 months ago with fatigue, weight loss, and tremor leading to the diagnosis of Graves’ disease. She has been on anti-thyroid medication since her diagnosis, and her symptoms have largely resolved. On exam,
exophthalmos is noted. Her heart rate is 58 bpm. Her cardiac exam is notable for a fixed split S2. The remainder of her exam is normal. As part of her evaluation, an ECG is obtained (ECG 98A). The patient is placed on telemetry and on the following day the resident notices a change in her QRS complexes. On jugular venous pressure inspection, cannon A waves are now seen. A second ECG is obtained (ECG 98B).

What abnormality is evident on the initial ECG (98A)?
What is the likely cause of this abnormality?
What does ECG 98B show?
What therapy is indicated?
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ECG 98A Analysis: Normal sinus rhythm, Mobitz type II second-degree AV block, left anterior fascicular block

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In ECG 98A, the rhythm is irregular as a result of two long pauses (→→) with the same duration (2.08 sec). The rhythm, therefore, regularly irregular. The remaining RR intervals are all the same, at a rate of 58 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF with an rS morphology). This is a left anterior fascicular block. The QT/QTc intervals are normal (400/395 msec). There are P waves (+) before each QRS complex with a stable PR interval (0.28 sec) (Γ), except for a slightly shorter PR interval (0.24 sec) before the third and fifth QRS complexes (Γ). It is likely that the slightly shorter PR interval is the result of slight enhancement of AV conduction due to the longer RR interval (slower rate), which allows for more rapid AV conduction as the AV node has had more time for recovery. During each pause there is an on-time P wave (PP intervals are constant) (邙) that does not have a QRS complex following it. Hence this is a second-degree AV block (ie, an occasional nonconducted P wave). As all of the PR intervals are constant (except for the PR interval after the long pause), this is a Mobitz type II second-degree AV block. The block is, therefore, within the His-Purkinje system.

continues
Podrid's Real-World ECGs

ECG 98B Analysis: Normal sinus rhythm, complete heart block, escape ventricular rhythm, intermittent capture (intermittent AV conduction)
ECG 98B shows regular RR intervals at a rate of 44 bpm. However, the second and seventh QRS complexes (▼) are early. All the QRS complexes (except for the second and seventh) are wide (0.18 sec), and their morphology resembles that of a right bundle branch block (tall, broad R wave in lead V1 [→] and broad S wave in leads I and V5-V6 [←]). It is, however, not a typical right bundle branch block as there is a tall monophasic R wave in lead V1-V3. There are P waves (*) that have a regular PP interval (▼) at a rate of 60 bpm. Some of the P waves are located within the ST segment (●) and are, therefore, less obvious. However, the P waves that are seen occur at a regular PP interval (▼). The P waves are positive in leads I, II, aVF, and V4-V6 and hence there is an underlying normal sinus rhythm. The PR intervals are very variable (+→), and the P waves are dissociated from the QRS complexes. As the atrial rate is faster than the ventricular rate, there is complete (third-degree) AV block.

As indicated, the second and seventh QRS complexes (▼) have a normal duration (0.08 sec) and morphology. There is a P wave (*) before each, with the same PR interval (▼) (0.28 sec). In addition, these two QRS complexes are early, indicating that they are the result of the P wave preceding them. Importantly, the two narrow QRS complexes have the same duration, morphology, and axis as the QRS complexes seen in ECG 98A. The PR interval associated with these QRS complexes is also identical to the PR intervals seen in ECG 98A. This is, therefore, complete AV block with an escape ventricular rhythm and intermittent capture. The captured complexes are identical to the conducted complexes seen in ECG 98A. The fact that the dissociated QRS complexes are wider and have a different morphology when compared with the captured complexes, which are narrow and have a normal duration and morphology, indicates that the dissociated complexes are ventricular in origin.

In this patient with Graves' disease, it would be important to check thyroid status as continued hyperthyroidism may be associated with complete AV block, although this usually occurs in patients with underlying conduction system disease. When the heart is "stressed" by the hyperthyroid state, complete AV block may become manifest. However, in this situation the sinus rate is likely to be more rapid than seen on these ECGs due to sympathomimetic effects seen with hyperthyroidism. Nevertheless, if hyperthyroidism was still present, with further therapy there would likely be resolution of the complete AV block. However, the presence of persistent complete AV block (in the absence of hyperthyroidism) associated with an escape ventricular rhythm is an indication for implantation of a permanent pacemaker.
An ECG from an asymptomatic 24-year-old woman is presented.

What notable findings are evident?
ECG 99 Analysis: Sinus bradycardia, intraventricular conduction delay to the right ventricle
The rhythm is regular at a rate of 46 bpm. There is a P wave (*) before each QRS complex with a stable PR interval (0.20 sec). The QRS duration is slightly prolonged (>0.10 but < 0.12 sec), and there is an RSR' complex in lead V1 (→) and a prominent S wave (!) in leads I and V5-V6. Although the QRS complex morphology is that of a right bundle branch block (RBBB), the QRS complex duration is not wide enough for a bundle branch block. This has often been termed an incomplete RBBB, although it is actually a conduction delay to the right ventricle and is best termed an intraventricular conduction delay. The QT/QTc intervals are normal (440/385 msec).

The RSR' morphology seen in the early (right) precordial leads (ie, V1-V2) represents a spectrum of physiology ranging from normal delayed depolarization of the crista supraventricularis of the right ventricle to right intraventricular conduction delay to pathologic RBBB. The QRS complex width defines the diagnosis. If the QRS complex width is normal (ie, < 0.10 sec), the morphology is deemed a normal variant and is termed a crista pattern. If the QRS complex duration is slightly prolonged (0.10 to 0.12 sec), an intraventricular conduction delay to the right ventricle is present (this is often called an incomplete RBBB). If the QRS complex duration is longer than 0.12 second, the diagnosis is complete RBBB.

In this case, there is an intraventricular conduction delay to the right ventricle. The term incomplete RBBB, often applied to this pattern, is not accurate as the bundle manifests “all or none” conduction characteristics; that is, it either conducts (always at the same rate) or does not conduct. This is not of any clinical importance, although it may be associated with the development of a complete RBBB in the future.
A 76-year-old man with a history of coronary artery disease and a previous myocardial infarction (MI) is seen in the emergency department for complaints of intermittent fatigue and lightheadedness that have been occurring for the past week. He states that the symptoms occur episodically, unrelated to activity. Each episode lasts for about 30 minutes and then resolves spontaneously. Between the episodes he feels well. He came to the...
emergency department because of an episode that was of longer duration and associated with more severe lightheadedness. His physical exam is unremarkable, except for a blood pressure of 170/90 mm Hg. An ECG is obtained (ECG 100A). Because he lives alone, it was decided to admit him to a telemetry unit for observation. The following day he complained of lightheadedness, and on telemetry there was a change in his heart rate. An ECG was obtained (ECG 100B).

What abnormality is noted on the ECGs?
What is the etiology of the abnormality?
What treatment is indicated?
ECG 100A Analysis: Normal sinus rhythm, right bundle branch block (RBBB), left anterior fascicular block, old anterior wall MI
In ECG 100A, there is a regular rhythm at a rate of 76 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The QRS complex duration is increased (0.16 sec), and it has a morphology of a typical right bundle branch block (RBBB), with an RSR' complex in lead V1 (++) and a broad terminal S wave in leads I and V5-V6 (+-). The axis is extremely leftward, between -30° and -90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF, with an rS morphology).

There are no Q waves in leads II and aVF; hence the extreme left axis is due to a left anterior fascicular block. There is a deep Q wave in lead V3 and probably in leads V4-V6 (||), consistent with an old anterior wall myocardial infarction (MI). The QT/QTc intervals are slightly prolonged (400/450 msec) but are normal when the prolonged QRS complex duration is considered (320/360 msec). The presence of an RBBB and left anterior fascicular block indicates bifascicular disease.

continue
Podrid's Real-World ECGs

ECG 100B Analysis: Complete heart block, escape junctional rhythm with RBBB and left posterior fascicular block, intermittent capture with Mobitz type I 2:1 AV block, trifascicular disease, old anterior wall MI
In ECG 100B, the rhythm is regular except for the second QRS complex, which is early (associated with a shorter RR interval). The rate is 32 bpm. P waves (+) are seen, and they occur with a regular interval (LJ) at a rate of 64 bpm. The PR intervals are variable, except for those associated with the second and third QRS complexes (*), both of which have the same PR interval (0.18 sec). This is the same as the PR interval seen in ECG 100A. Therefore, there is AV dissociation and, as the atrial rate is faster than the ventricular rate, complete heart block (third-degree AV block) is present. Complexes 2 and 3 (**) represent intermittent capture. All of the QRS complexes have a prolonged duration (0.16 sec) with an RBBB morphology, as noted in the lead V1 rhythm strip. The RBBB morphology is identical to the QRS morphology seen in ECG 100A. Similar to the QRS complexes in ECG 100A, there are also Q waves in leads V2-V5 due to an old anterior wall MI. The QT/QTc intervals are also the same.

However, there are changes in the QRS complex as seen in the lead II rhythm strip. This is primarily a result of a change in axis. Complexes 1, 4, and 5 (**), which are dissociated, have an RBBB morphology and a QRS complex that is negative in lead I and positive in lead II. Although this QRS complex is not seen in lead aVF, the fact that it is negative in lead I and positive in lead II suggests that it is likely also positive in lead aVF and hence it has a right axis, a result of a left posterior fascicular block. Complexes 2 and 3, which are conducted from the sinus P wave (ie, they are captured), also have an RBBB morphology, but they have a positive QRS complex in lead I and negative QRS complex in leads II and aVF, which is a left anterior fascicular block. These QRS complexes are identical in morphology and axis to those seen in ECG 100A. The rhythm is, therefore, sinus with complete heart block and intermittent capture. The escape rhythm is junctional as all of the QRS complexes have a typical RBBB morphology that is identical to what is seen in ECG 100A. However, there is alternating fascicular conduction, with the conducted complexes having a left anterior fascicular block that is identical to the QRS complexes in ECG 100A and the dissociated QRS complexes having a left posterior fascicular block. Therefore, while the escape rhythm is junctional, there is also evidence for trifascicular disease (ie, an RBBB with alternating left anterior and left posterior fascicular block).

It can be seen that there is a nonconducted P wave (*) between complexes 2 and 3, representing a brief period of second-degree AV block with a pattern of 2:1 AV conduction (2:1 AV block). Since the escape rhythm is junctional, the 2:1 AV block is a Mobitz type 1. As this patient is symptomatic and has evidence of AV nodal as well as His-Purkinje disease, placement of a permanent pacemaker is indicated.
A 72-year-old diabetic woman presents to her primary care physician 4 days after "a transient illness." When asked to elaborate, she states that several days ago she suffered a prolonged bout of indigestion with substernal burning resembling gastroesophageal reflux, intermittent sweats, fatigue, and mild shortness of breath. She assumed she had "caught a 24-hour stomach bug." For 2 days thereafter, she felt exhausted. Her symptoms have largely abated except for some lingering fatigue, which has brought her to her physician.

On exam, her heart rate is 72 bpm, and her blood pressure 148/88 mm Hg. There is no lymphadenopathy, and her head, ears, eyes, nose, and throat exam is normal. Her lungs are clear. Her jugular venous pressure is 7 cm H₂O with normal X and Y descents. Her cardiac exam is notable for an S4 gallop and a faint holosystolic murmur at the apex. Her abdominal, extremity, and neurologic exams are normal. An ECG is obtained.

What abnormality is noted?
Does it shed any light on her preceding illness?
ECG 101 Analysis: Normal sinus rhythm, left axis, recent inferior wall myocardial infarction
There is a regular rhythm at a rate of 72 bpm. There is a P wave (*) before each QRS complex, with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4-V6. Hence this is a normal sinus rhythm. The axis is extremely leftward, between −30° and −90° (positive QRS complex in lead I and negative QRS complex in leads II and aVF). Although these are the criteria for a left anterior fascicular block, it can be seen that the initial waveform in leads II, III, and aVF is a Q wave (†) and not an R wave (which is seen with a left anterior fascicular block). Hence the left axis in this case is the result of an inferior wall myocardial infarction (MI). There is slight ST-segment elevation (‡) in leads II, III, and aVF, and the T waves are inverted (§). In addition, there is slight ST-segment depression in leads I and aVL (+); these are reciprocal changes. These abnormalities indicate that the infarction is likely recent and reflect the ECG changes of an acute infarction that is still evolving. The QT/QTc intervals are normal (400/440 msec).

Her symptoms 4 days ago likely represented the onset of the ST-segment elevation MI. Although she had symptoms, they were misinterpreted as being gastrointestinal rather than cardiac in origin. This is not infrequent as an inferior wall MI often presents with substernal burning and fullness, symptoms that are often confused with a hiatal hernia or gastroesophageal reflux. In addition it has been reported that almost 50% of inferior wall MIs may be silent, without any recognizable symptoms. This may be especially frequent in a patient who has diabetes in whom there may be autonomic dysfunction. The anginal discomfort is a result of impulse transmission through unmyelinated fibers that travel along with the sympathetic nerves that innervate the heart and enter the spinal cord at the C7–T4 level. Hence neural transmission of anginal discomfort is interrupted in the presence of a diabetic autonomic neuropathy. These patients are said to have silent ischemia. However, it is actually painless or discomfortless ischemia; while they may not experience anginal discomfort, they will often have the other associated symptoms of ischemia, including shortness of breath, nausea, and diaphoresis.

Her physical exam also demonstrates a holosystolic murmur at the apex. Although it is not certain whether this murmur predated the MI, such murmurs, which are mitral in origin, are not uncommon with an inferior wall MI and are due to associated posteromedial papillary muscle dysfunction. An echocardiogram would be essential to further evaluate the murmur as well as left ventricular function and wall motion abnormalities. Routine post-MI therapy is indicated in this patient, including β-blocker, aspirin, statin, and possibly an angiotensin-converting enzyme inhibitor, depending on left ventricular function and the severity of the wall motion abnormality of the inferior segment of the left ventricle.