This book is dedicated to my parents, Anne and George Stouffer, who encouraged me in all of my endeavors.
Practical ECG Interpretation
Clues to Heart Disease in Young Adults

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Cardiac death in an apparently healthy young adult, whether sudden or due to irreversible damage from unrecognized congenital heart disease, is a catastrophic event. It is rare but not as rare as we think – the most common cause of sudden death in young adults in general and in competitive athletes of all ages is unsuspected cardiovascular disease. Furthermore, there are occasionally symptomatic young adults who have congenital or valvular heart disease that has not been diagnosed. In some of these cases, the correct diagnosis is not made until irreversible cardiac damage occurs. In any young adult, the occurrence of cardiac morbidity and mortality is tragic, but even more so if it could have been prevented.

The 12-lead ECG can provide clues to the presence of cardiovascular disease in young adults and thus it is essential that the clinician be trained to recognize the ECG patterns that warrant further evaluation. The ECG is neither sensitive nor specific for the diagnosis of cardiovascular pathology, but this disadvantage is outweighed by its low cost and ready availability. For educational purposes, it is useful to divide ECG findings that may provide important clues to unsuspected disease into two broad categories – those associated with the hemodynamic changes of congenital or valvular heart disease and those associated with cardiovascular disease that increases the risk of sudden cardiac death. These groups are discussed in more detail in chapters 8 and 9.

While many forms of congenital or valvular heart disease will become clinically apparent early in life, there are other forms which may not cause symptoms until adulthood (e.g. an atrial septal defect or mitral stenosis). These patients may manifest ECG changes prior to the development of symptoms or in the presence of non-specific complaints. The “classic” ECG changes of congenital heart disease (e.g. right axis deviation, a large R-wave in V1 and evidence of right atrial abnormality) are rare; much more common are incomplete clues to the presence of pathology. These might include a change in axis (either QRS or more rarely atrial), hypertrophy of the right ventricle or left ventricle, incomplete right bundle branch block or an abnormality in the P-wave pointing towards left atrial or right atrial enlargement.

To further complicate matters, many of these ECG findings are common in children and/or highly trained athletes and thus not always specific for underlying pathology. It is important to differentiate pathologic ECG changes from the normal aging process as an individual passes from childhood through adolescence and into adulthood (for example, as we age, “normal” changes in the ECG include a decrease in heart rate, increase in PR interval, a leftward shift in the QRS axis and an increase in QRS duration). With proper training and experience, the astute clinician can recognize those ECG changes which are pathologic and initiate further cardiac evaluation (usually an echocardiogram and possibly other studies) which can lead to the diagnosis of previously unsuspected heart disease.

A second group of patients are at a high risk of ventricular arrhythmias and sudden cardiac death. These disorders include certain forms of “electrical” heart disease (e.g. Brugada Syndrome, long QT syndrome) as well as hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia and Wolff–Parkinson–White syndrome. ECG findings that should prompt consideration of these disorders include a prolonged QT interval, a shortened PR interval, increased voltage and/or changes in the...

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Practical ECG Interpretation: Clues to Heart Disease in Young Adults
ST/T segment in leads V1 through V3. An accurate diagnosis in these patients can truly be a matter of life and death as the first clinical manifestation of heart disease may be sudden cardiac death.

It is important to recognize that young adults with unsuspected cardiovascular disease may not come to medical attention in the traditional way. More commonly, they will have an ECG performed for unrelated symptoms or as part of a screening program (e.g. for life insurance, pre-employment or prior to participation in athletic events). It is important that these clues are not missed as an accurate diagnosis may enable corrective treatment prior to the development of irreversible hemodynamic changes (e.g. closure of an ASD), may preclude participation in certain athletic endeavors (e.g. hypertrophic obstructive cardiomyopathy) and/or may lead to treatment (e.g. with an implantable cardioverter defibrillator) that reduces the risk of sudden death.

The purpose of this book is to present real-life clinical scenarios in which the recognition of unsuspected cardiovascular disease from the ECG was important to the proper diagnosis and management of the patient. This book is not intended as a primer on congenital heart disease nor is it an ECG book. Rather, its purpose is to emphasize important findings on a 12-lead ECG that should prompt further evaluation for cardiovascular pathology in young adults.
PART I

The Basics of Electrocardiography – A Brief Introduction to the Essentials
One of the most fearsome memories from medical school for many physicians is the lecture dealing with action potentials. Unfortunately, the over-emphasis on esoteric details in these lectures has prevented most of us from remembering the basic details of the action potential which we need to know to properly interpret ECGs.

Action potential refers to changes in membrane potential over time. The membrane potential, in turn, depends upon the maintenance of ionic concentration gradients across the membrane. The maintenance of these concentration gradients is an active process requiring ionic pumps. The various phases of the cardiac action potential are associated with changes in the permeability of the cell membrane, mainly to sodium, potassium and calcium ions and/or inhibition of ionic pump activity. Changes in permeability are accomplished by the opening and closing of ion channels that are specific for the individual ions.

Two main types of action potentials are observed in the heart. The “slow response” or pacemaker cell response is found in the sinoatrial (SA) node and the atroventricular (AV) node (Fig. 1.1). “Fast responses” or non-pacemaker cell responses are observed in normal myocardial fibers in the atria and ventricles. Important differences between the two types include resting membrane potential, slopes of Phase 1 and Phase 4, and amplitude of the action potential.

**Pacemaker cells**

Pacemaker cells generate regular, spontaneous action potentials. In these cells, the depolarizing current is carried primarily by relatively slow, inward calcium currents instead of by fast sodium currents as observed in most other depolarizing cells types (e.g. nerve and muscle cell). Spontaneous depolarization (Phase 4) is due to a small increase in intracellular calcium, a decrease in intracellular potassium and a slow inward Na⁺ current. Once this spontaneous depolarization reaches a threshold, a new action potential is triggered. The rate of Phase 0 depolarization is much slower than that found in other cardiac cells (see Fig. 1.1).

An understanding of action potentials helps explain the effects of various stimuli on heart rate. Sympathetic activation releases norepinephrine which increases the slope of Phase 4 and thereby increases heart rate. Parasympathetic activation releases acetylcholine which decreases the slope of Phase 4 and hyperpolarizes the cell which in turn increases the time to reach threshold voltage. The effect of profound elevation in serum potassium concentrations can be predicted based on the action potential: severe hyperkalemia causes sinus arrest.

Non-pacemaker cells can change into pacemaker cells under certain conditions. For example, if a cell...
becomes hypoxic, the membrane depolarizes which closes fast Na⁺ channels. When this occurs, action potentials can still be elicited but the inward current will be carried by Ca²⁺ (slow inward channels). These action potentials are similar to those found in pacemaker cells and display spontaneous depolarization and automaticity.

**Non-pacemaker cells**

The concentration of potassium ions inside a cardiac muscle cell greatly exceeds the concentration outside the cell. The reverse situation exists for sodium ions. The relative concentration of ions (and negatively charged intracellular proteins) determines membrane potential. The resting membrane potential (Phase 4; Fig. 1.2) in non-pacemaker cells remains near the equilibrium potential. When these cells are rapidly depolarized by a conducted action potential from another cell, this causes a transient increase in fast sodium channel conductance and a decrease in potassium influx (Phase 0). Subsequent repolarization is a three-phase process. Phase 1 is caused by the opening of potassium channels. Because of a (relatively) slow increase in calcium, repolarization reaches a plateau phase (Phase 2). Phase 3 occurs when potassium influx increases and calcium influx decreases.

The effective refractory period (ERP) refers to the time during Phases 0, 1, 2 and part of Phase 3, when the cell will not respond to action potentials transmitted from other cells. The ERP acts as a protective mechanism by preventing chaotic, irregular cellular depolarization. The length of the refractory period limits the frequency of action potentials (and therefore contractions) that can be generated by the heart.
The sinoatrial (SA) node is in direct contact with surrounding atrial muscle (Fig. 2.1). The action potential generated in the nodal tissue spreads throughout both atria at a rate of approximately 0.3 meters per second. Activation of the normal human atria takes approximately 90–100 msec. Controversy exists about whether SA to atrioventricular (AV) nodal conduction occurs over specialized fiber bundles within the atria (which conduct the action potential to the AV node with a greater velocity than seen in ordinary atrial muscle) or via ordinary atrial myocardium. At the AV node there is a short delay (approximately 0.1 second) in transmission of the impulse to the ventricles. This enables the atria to complete their contraction before the ventricles contract. Once the action potential leaves the AV node, it enters specialized muscle fibers called Purkinje fibers. These are grouped into a mass termed the bundle of His. The Purkinje fibers are very large and conduct the action potential at about six times the velocity of ordinary cardiac muscle (i.e. 1.5–4.0 meters per second).

In the absence of bundle branch block, ventricular activation is quite rapid and usually takes less than 100 msec. The impulse proceeds through the ventricular muscle at about 0.3–0.5 meters per second. This results in a contraction of the ventricles that proceeds upward from the apex of the heart toward its base.

**Sinoatrial node**

The SA node is a crescent-shaped structure 9–15 mm in length. It is located in the posterior part of the right atrium at the junction of the superior vena cava. Because it is epicardial in location, SA nodal function may be affected by pericardial diseases such as pericarditis or metastatic disease.

The SA node surrounds the nodal artery from which it receives a rich blood supply (Figs 2.1, 2.2). It originates from the right circumflex artery (RCA) in the majority of individuals (approximately 75%) but can also arise from the left circumflex or there can be a dual supply from both arteries. The origination of the SA nodal artery is unaffected by coronary dominance (i.e. which artery supplies the posterior descending coronary artery), in contrast to the AV nodal artery which always originates from the dominant circulation.
Atrioventricular node

The AV node is an oval or elliptical structure located in the right atrium near the lower part of the interatrial septum. It is anterior and superior to the ostium of the coronary sinus and directly above the insertion of the septal leaflet of the tricuspid valve (see Fig. 2.1). This area is located at the apex of the triangle of Koch, which is formed by the tricuspid annulus, the tendon of Todaro, and the ostium of the coronary sinus. The bundle of His originates from the anteroinferior pole of the AV node and travels through the central fibrous body to reach the dorsal edge of the membranous septum. It then divides into right and left bundle branches. The right bundle continues first intramyocardially, then subendocardially, toward the right ventricular apex. The left bundle continues distally along the membranous septum and then divides into anterior and posterior fascicles.

Blood supply to the AV node is provided by the nodal artery which arises from the proximal portion of the posterior descending artery (PDA) (see Fig. 2.2). The PDA is a branch of the right coronary artery in approximately 85% of individuals and of the left circumflex in approximately 15% of individuals.

Bundle of His

The His bundle originates at the apex of the triangle of Koch and then penetrates the central fibrous body at the attachment of the tendon of Todaro, runs between the membranous septum and the muscular septum, and bifurcates at the crest of the muscular ventricular septum. The His bundle has a dual blood supply from branches of the anterior and posterior descending coronary arteries.

The His bundle descends in the interventricular septum for a short distance and then divides into two large branches, the right and left bundle branches. Each of these descends along its respective side of the interventricular septum immediately beneath the endocardium and divides into smaller and smaller branches. Terminal Purkinje fibers extend beneath the endocardium and penetrate approximately one-third of the distance into the myocardium. Their endings terminate upon ordinary cardiac muscle within the ventricles. The bulk of the arterial blood supply to the right and left bundle branches comes from the left anterior descending artery (LAD) but the bundle branches are supplied by both left and right coronary arteries.
**Bundle branches**

The bundle branches and Purkinje network comprise the ventricular conduction system. The right bundle branch is a direct continuation of the penetrating bundle, originates distal to the attachment of the septal leaflet of the tricuspid valve, lies on the right side of the interventricular septum and does not branch until it gets to the right ventricular apex.

The left bundle branch lies on the left side of the septum and almost immediately divides into two main divisions: the anterior (superior) and the posterior (inferior) division. Each subdivides into a number of fascicles. The anterior division supplies the anterior and superior wall, and the posterior division supplies the posterior inferior wall of the left ventricle. The anterior division of the left bundle branch is fragile, easily exposed to damage, and has a single blood supply (LAD). The posterior division is much thicker and has a double blood supply (left and right coronary arteries).

**Autonomic nervous system**

The rate and rhythm of the heart are influenced by both the sympathetic and parasympathetic branches of the autonomic nervous system. The sympathetic nervous system supply to the heart leaves the spinal cord at the first four thoracic vertebrae. Within the heart, sympathetic efferent nerves are present in the conduction system (including the SA and AV nodes), throughout the atria, and in the ventricles. The parasympathetic supply to the heart comes via the left and right vagus nerves, branches of the 10th cranial nerve. In general the right vagus nerve innervates the SA node and the left vagus nerve innervates the AV node but the anatomical distribution of the two vagus nerves can have significant overlap. The vagus nerve also innervates atrial muscle but in contrast to the sympathetic system, provides minimal innervation to the ventricles.

A rich supply of sympathetic and parasympathetic nerves innervates the SA node. Combined inhibition of both sympathetic and parasympathetic tone results in a faster resting heart rate, demonstrating that vagal tone predominates at the SA node in the normal heart. The AV node, in contrast, appears to have a relatively balanced input from the sympathetic and parasympathetic systems. There is minimal effect of the autonomic nervous system on His–Purkinje conduction.
A typical ECG waveform of an individual in normal sinus rhythm consists of a P wave, PR interval, QRS complex, ST segment, T wave and U wave (Figs 3.1, 3.2). The voltage of sinoatrial (SA) node depolarization is too small to reflect on the surface ECG and thus the first deflection is the P wave which reflects depolarization of the atria. This is followed by the QRS complex which reflects depolarization of the ventricles and then the ST segment and T wave which reflect repolarization of the ventricles. The cause of the U wave remains unclear.

**P wave**

The sinoatrial node sits within the right atrium and thus the right atrium depolarizes before the left atrium. The beginning, midportion and end of the P wave reflect depolarization of the right atrium, both atria and left atrium, respectively. Atrial depolarization occurs from right to left, from posterior to anterior, and from superior to inferior (from the right shoulder to the left leg). A “normal” P wave is thus positive in leads I, II, V5, and V6 and inverted in lead aVR. In V1, the P wave may be upright, biphasic or inverted. P wave morphology and/or axis can change depending on the site of origin of the impulses initiating atrial activation (e.g. in wandering atrial pacemaker), atrial enlargement and conditions which affect the position of the heart within the chest (e.g. lung disease). In general, the further the ectopic focus is from the sinus node, the more abnormal will be the P wave configuration. Factors that prolong impulse propagation in the atria, such as fibrosis or hypertrophy, will lengthen the duration of the P wave.

**Clinical correlate**

“Lewis leads” can be used to get a more detailed picture of atrial activity. This involves placing a negative electrode in the second intercostal space on the right side, a positive electrode in the fourth intercostal space on the right side and a ground in the second intercostal space on the left side with all electrodes placed close to the sternum. In practice this can be accomplished by placing the right arm lead in the second intercostal space on the right, the left arm lead in the 5th intercostal space on the right, the leg leads in their normal position and then recording lead I.

**PR interval**

The route and the speed of conduction from the atria to the ventricles, which usually occurs via the AV node and specialized conducting system,
influence the PR interval. The PR interval is prolonged by factors that slow conduction through the AV node, such as fibrosis of the node, increased vagal tone or medications (e.g. beta-blockers, digoxin, calcium channel blockers). The PR interval is shortened when impulses reach the ventricles via a bypass tract to cause ventricular pre-excitation. In Lown-Ganong-Levine syndrome, an AV nodal bypass track terminates into the His bundle thus reducing the PR interval but maintaining normal ventricular activation (i.e. there will be no delta wave).

LV depolarization will generally obscure right ventricular (RV) depolarization because of its larger size. The spatial vector of the QRS complex reflects LV dominance and is directed to the left and posteriorly. The QRS complex is usually positive in the left-sided and more posterior leads (leads I, V5, and V6), and negative or inverted in the most right-sided and more anterior leads (leads aVR and V1). Only in situations where RV depolarization is delayed (e.g. right bundle branch block) or enhanced (e.g. RV hypertrophy) is the electrical activity associated with RV depolarization identifiable.

Conditions that alter the QRS complex include abnormalities in the sequence of ventricular activation (e.g. right and left bundle branch blocks), pre-excitation (Wolff–Parkinson–White syndrome), myocardial infarction where the loss of ventricular muscle results in the formation of Q waves, medications that interfere with the sodium inward current, and electrolyte abnormalities (e.g. hyperkalemia). The amplitude of the QRS complex can be increased by ventricular hypertrophy and decreased by the presence of pericardial or pleural fluid, infiltrative myopathic diseases and/or amount of tissue between the heart and the chest wall.

Clinical correlate
Spontaneous fluctuation in PR interval is most often seen in individuals with normal hearts.

QRS complex

Normally, depolarization of both ventricles occurs simultaneously, spreading from endocardium to epicardium and from apex to base. Left ventricular
PART I  The Basics of Electrocardiography

ST segment and T wave
The ST segment and T wave reflect repolarization of the ventricles. Repolarization occurs in the opposite sequence of depolarization – the cells on the epicardium are the first to repolarize, though they are the last to depolarize. This explains why the normal T wave is generally upright (positive) in leads with an upright or positive QRS complex (leads I, V5, and V6) and inverted (negative) in leads with an inverted QRS complex (aVR and V1). The voltage change during depolarization is opposite that seen during repolarization and the direction of depolarization is opposite that of repolarization; together these result in the vector of depolarization and repolarization tending in the same direction in the normal heart.

Certain conditions will affect both the ST segment and the T wave (e.g. LV hypertrophy or anti-arrhythmic drugs) whereas others will affect primarily the ST segment (e.g. hypocalcemia, Brugada Syndrome) or the T wave (e.g. hyperkalemia, respiration). The ST segment and T wave can both be affected by certain conditions but at different times; for example in acute ST elevation myocardial infarction the ST segment changes occur prior to T wave changes. Similarly, T wave changes tend to occur later than ST segment changes in pericarditis. Changes in repolarization can also result from changes in the depolarization, such as in bundle branch blocks or ventricular pre-excitation.

The ST segment will be at the same level on the ECG as the PR and TP segments in the normal heart (ventricular action potentials are at their plateau voltage during these segments). Disorders which affect the action potential plateau voltage can result in ST segment elevation or depression. The duration of the QT interval is affected by the duration of the ST segment and T wave. Please see chapter 16 for a more complete discussion of long QT syndromes.

U wave
The precise etiology of the U wave is not clearly understood. U waves are more prominent at slow heart rates and usually best seen in the right precordial leads. U waves can be compared to T waves in the same lead: the U wave is usually oriented in the same direction as the T wave and amplitude is usually <1/3 T wave amplitude.

U waves are usually best seen in the right precordial leads especially V2 and V3. An increase in U wave amplitude can be seen with sinus bradycardia, hypokalemia, central nervous system disease, congenital long QT syndrome, and cardiac medications.

Clinical correlate
Low amplitude on the surface ECG can be seen in any condition where there is a significant amount of poorly conducting tissue or fluid between the heart and chest wall. For example, this occurs in pericardial effusion, emphysema and obesity.

Clinical correlate
Negative U waves in the precordial leads have been reported to correlate with significant coronary artery disease.
The first ECG signals from humans were recorded by the Dutch physiologist Willem Einthoven in 1902. He attached a galvanometer to both arms and the left leg of patients and recorded what are today labeled leads I, II and III (Fig. 4.1). Six chest leads were added in 1932 by Charles Wolferth and Francis Wood. Then in 1934, Frank Wilson joined the wires from the right arm, left arm and left foot with 5000 Ohm resistors. The combined lead acts as a ground and is attached to the negative terminal of the ECG. An electrode attached to the positive terminal then becomes “unipolar” and can be placed anywhere on the body. Wilson and colleagues defined the unipolar limb leads as VR, VL and VF where “V” stands for voltage. Finally, in 1942, Emanuel Goldberger increased the voltage of Wilson’s unipolar leads by 50% and created the augmented limb leads aVR, aVL and aVF (Fig. 4.2). The augmented leads were added to Einthoven’s three limb leads and the six chest leads, arriving at the 12-lead electrocardiogram that we use today. In 1924, Willem Einthoven won the Nobel Prize for inventing the electrocardiograph.

Traditionally, the ECG consists of 12 leads (Table 4.1) but in special circumstances other leads can also be useful. Right-sided chest leads are often used in adults with inferior myocardial infarction to diagnose right ventricular infarction, and one or more leads positioned on the back are sometimes useful in diagnosing posterior wall infarction.
Figure 4.3 How voltage changes in the heart lead to deflections on the ECG.
The chest leads are much closer to the heart than are the limb leads and are influenced by the electrical activity directly under the recording lead. Changes in the relation of the individual chest lead to the heart may cause significant changes in the ECG waveform. Thus care should be taken when

Electrophysiology of the ECG

Simply put, the ECG records the results of 12 voltimeters placed at different locations on the body. As myocardial cells depolarize and repolarize, tiny changes in voltage take place which are recorded and displayed on the ECG (Fig. 4.3). The ECG simply reflects difference in potential between two locations. A positive deflection on the ECG indicates that voltage is greater at the positive terminal whereas a negative deflection indicates the opposite (Fig. 4.4).

In the bipolar limb leads, the negative pole for each of the leads is different, whereas the negative pole for all of the chest leads (an average of three limb leads) is the same. In theory, the information available from all six limb leads can be deduced from any two.

<table>
<thead>
<tr>
<th>Lead</th>
<th>Negative pole</th>
<th>Positive pole</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Right arm</td>
<td>Left arm</td>
</tr>
<tr>
<td>II</td>
<td>Right arm</td>
<td>Left leg</td>
</tr>
<tr>
<td>III</td>
<td>Left arm</td>
<td>Left leg</td>
</tr>
<tr>
<td>aVR</td>
<td>Left arm + left leg</td>
<td>Right arm</td>
</tr>
<tr>
<td>aVL</td>
<td>Right arm + left leg</td>
<td>Left arm</td>
</tr>
<tr>
<td>aVF</td>
<td>Right arm + left arm</td>
<td>Left leg</td>
</tr>
<tr>
<td>V1</td>
<td>Average of 3 limb leads</td>
<td>Right of sternum in 4th intercostal space</td>
</tr>
<tr>
<td>V2</td>
<td>Average of 3 limb leads</td>
<td>Left of sternum in 4th intercostal space</td>
</tr>
<tr>
<td>V3</td>
<td>Average of 3 limb leads</td>
<td>Halfway between V2 and V4</td>
</tr>
<tr>
<td>V4</td>
<td>Average of 3 limb leads</td>
<td>Midclavicular line in 5th intercostal space</td>
</tr>
<tr>
<td>V5</td>
<td>Average of 3 limb leads</td>
<td>Horizontal with V4 at anterior axillary line</td>
</tr>
<tr>
<td>V6</td>
<td>Average of 3 limb leads</td>
<td>Horizontal with V4 at anterior midaxillary line</td>
</tr>
</tbody>
</table>

**Clinical correlate**

In the United States, typically the right arm lead is white, the left arm lead is black, the right leg lead is green and the left leg lead is red. In Europe, the leads are red, yellow, black and green, respectively.

Leads

Twelve leads are routinely recorded:
- three bipolar limb leads (I, II, and III)
- three augmented limb leads (aVR, aVL, and aVF)
- six unipolar chest leads (V1 through V6).

Since the ECG is a voltmeter, it is essential to know what constitutes the positive and negative pole for each lead. Knowing this information, then a positive deflection on the ECG indicates that voltage is greater at the positive pole whereas a negative deflection indicates the opposite (Fig. 4.4).

In the bipolar limb leads, the negative pole for each of the leads is different, whereas the negative pole for all of the chest leads (an average of three limb leads) is the same. In theory, the information available from all six limb leads can be deduced from any two.

Lead III is the most affected by respiration and the morphology of this lead can change depending on the respiratory cycle. Thus, the appearance of an isolated Q wave in this lead (without corresponding changes in AVF or II), carries no prognostic significance.

The chest leads are much closer to the heart than are the limb leads and are influenced by the electrical activity directly under the recording lead. Changes in the relation of the individual chest lead to the heart may cause significant changes in the ECG waveform. Thus care should be taken when
positioning the chest wall leads. For example, if a lead is misplaced an interspace too high or too low, R wave voltage can change dramatically, leading to the misdiagnosis of poor R wave progression or old anterior myocardial infarction. Similarly, if the patient is in a sitting rather than a supine position, the relation of the leads to the heart and therefore the ECG waveform will change.

**Further reading**


PART II

Helpful Hints in Interpreting ECGs in Young Adults
Genesis of the heart beat
The specialized conduction system of the heart is composed of cells that conduct electrical impulses faster than the surrounding myocardium. The conduction system can be divided into distinct anatomic segments including the sinoatrial (SA) and atrioventricular (AV) nodes, bundle of His and right and left bundles.

The heart rate is determined by spontaneous firing (depolarizing) of specialized cells. In the normal heart, the SA node controls the heart rate because it fires more rapidly than other parts of the heart (approximately 60–80 beats per minute (bpm)). If the SA node is diseased, control of the
heart rate may fall to atrial cells, the AV node or ventricular cells (Table 5.1).

Sinus node depolarization occurs before the onset of the P wave, but the voltage gradients associated with sinus node depolarization are too small to be recorded by an ECG machine. Therefore, this event is electrocardiographically silent. Similarly, the electrical activity of the AV junction, which occurs during the PR interval, is also electrocardiographically silent.

<table>
<thead>
<tr>
<th>Origin of rhythm</th>
<th>Intrinsic rate (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node</td>
<td>60–80</td>
</tr>
<tr>
<td>AV node</td>
<td>40–50</td>
</tr>
<tr>
<td>Ventricular escape</td>
<td>30–40</td>
</tr>
</tbody>
</table>

Table 5.1 Intrinsic rate of cells within different sections of the heart that are capable of spontaneous depolarization

Figure 5.2 Determining rate from the ECG when the rhythm is regular. To get the rate in beats per minutes, count the number of large boxes between beats and divide by 300 (since each large box is 0.2 seconds there are 300 large boxes/minute).

Figure 5.3 Determining rate from the ECG when the rhythm is irregular. To get the rate in beats per minutes, count the number of beats over a period of 6–30 seconds (5 large boxes represent one second) and then multiply by the appropriate factor to get the number of beats per minute.
Determine the rate

The standard paper speed is 25 mm/sec for recording an ECG in most countries (although it is 50 mm/sec in some countries). If the rhythm is regular, the heart rate can be rapidly estimated by counting the number of large boxes between QRS complexes and then dividing this number into 300 or by counting the number of small boxes and dividing into 1500. If the rhythm is irregular, the heart rate can be rapidly estimated by counting the number of large boxes between QRS complexes and then dividing this number into 300 or by counting the number of small boxes and dividing into 1500 (Fig 5.2). If the rhythm is irregular, the rate can be estimated by counting the number of QRS complexes in 10 seconds (50 large boxes) and multiplying by 10 (Fig. 5.3).

By convention, tachycardia is defined as rates greater than 99 bpm and bradycardia as rates less than 60 bpm (some sources use <50 bpm).

Normal sinus rhythm

In the normal heart, the rhythm originates in the sinus node and propagates through the heart in an orderly fashion and within defined intervals. Characteristics of sinus rhythm include an upright P wave in leads I and II, QRS complexes follow each and every P wave and the heart rate is between 60 and 99 bpm. Sinus tachycardia is a normal response to exercise, excitement and other conditions which stimulate the sympathetic nervous system. Similarly, sinus bradycardia can be a normal finding (described below).

Normal findings in healthy (generally young) individuals

ECG findings that indicate pathology in older individuals or patients with cardiac disease can be normal findings in young adults. Sinus bradycardia during sleep is common while sinus bradycardia while awake is common in athletically fit individuals. Sinus arrhythmia, wandering atrial pacemaker, Wenckebach phenomenon, junctional rhythm and first-degree AV block are also common, especially during sleep.

Important features of a standard ECG

Paper speed is 25 mm/sec which means that each large box is 0.2 sec and each small box is 0.04 sec. An amplitude of 1 mm generally corresponds to 0.1 mV (the calibration of voltage in mV to deflection in mm should be deflected somewhere on each ECG).
The 12-lead ECG provides information about the heart’s electrical activity in three dimensions – right to left, superior to inferior and anterior to posterior. Each of the 12 leads is oriented in a specific direction (Table 6.1).

### Table 6.1 Derivation of voltage represented on the standard 12-lead ECG

<table>
<thead>
<tr>
<th>Lead</th>
<th>Negative terminal</th>
<th>Positive terminal</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Right arm</td>
<td>Left arm</td>
</tr>
<tr>
<td>II</td>
<td>Right arm</td>
<td>Left foot</td>
</tr>
<tr>
<td>III</td>
<td>Left arm</td>
<td>Left foot</td>
</tr>
<tr>
<td>aVR</td>
<td>Left arm + left foot</td>
<td>Right arm</td>
</tr>
<tr>
<td>aVL</td>
<td>Right arm + left foot</td>
<td>Left arm</td>
</tr>
<tr>
<td>aVF</td>
<td>Right arm + left arm</td>
<td>Left foot</td>
</tr>
<tr>
<td>V1–V3</td>
<td>Average of 3 limb leads (center of chest)</td>
<td>Anterior chest wall</td>
</tr>
<tr>
<td>V4–V6</td>
<td>Average of 3 limb leads (center of chest)</td>
<td>Lateral chest wall</td>
</tr>
</tbody>
</table>

### Axis

Axis refers to the mean direction of the wave of depolarization (P wave axis or QRS axis) or repolarization (T wave axis). The most important axis to be determined is the QRS axis. This axis can be measured in two planes – a vertical plane (also referred to as the frontal plane and calculated using the limb leads; Fig. 6.1) and a horizontal plane (also referred to as precordial plane and calculated using the chest leads; Fig. 6.2). By definition, horizontal and vertical refers to the planes if an individual was standing but the ECG is always obtained while the individual is supine.

The vertical plane is most commonly used to determine axis. By convention, the zero line is the same as lead I (i.e. negative pole on the right arm and positive pole on the left arm; Table 6.2, Fig. 6.3). The normal range for axis in individuals older than 40 years is between –30 and 90 degrees. In young adults, an axis of 90–105 degrees is occasionally seen in normal hearts. The axis tends to shift leftwards as we age and this is accentuated in obese individuals.
When determining axis, the net deflection is considered positive if the R wave is greater than the S wave (Fig. 6.4).

**Clinical correlate**

There are two relatively easy ways to calculate QRS axis. One is to find an isoelectric lead (i.e. R wave = S wave) – the axis is perpendicular to this lead. The other way is to use two perpendicular leads (usually I and AVF) and plot out (or estimate) the axis. For example, if the net deflection is positive in I and AVF, then the axis is between 0 and 90 degrees and within the normal range. If the net deflection is negative in I and positive in AVF then the axis is greater than 90 degrees, consistent with right axis deviation.

Left axis deviation is defined as an axis between –30 and –90 degrees. Lead I will be positive, lead AVF negative and lead II mostly negative. Note that axis between 0 and –30 degrees is considered normal. Common causes of left axis deviation include:

- left anterior fascicular block (LAFB)
- left bundle branch block
- some cases of inferior myocardial infarction
- ostium primum atrial septal defect and other endocardial cushion defects
• some cases of Wolff–Parkinson–White syndrome (large negative delta wave in lead II).

Right axis deviation (RAD) is defined as an axis between 90 and 180 degrees. Lead I will be negative and lead AVF positive. Common causes include:

• left posterior fascicular block (LPFB)
• right ventricular hypertrophy
• high lateral wall myocardial infarction (MI) with QR or QS complex in leads I and aVL
• some cases of WPW syndrome.

RAD can be a normal finding in children, teenagers, and some young adults.

In rare individuals, the QRS axis will be between 180 and −90 degrees (leads I and II are both negative). Axes in this range are variously labeled extreme LAD, extreme RAD or “bizarre.” In these patients, consider limb lead reversal (usually right and left arm), dextrocardia or other types of congenital heart disease (e.g. transposition).

The frontal axis also provides important information. In particular, determination of the transitional zone (the lead having positive and negative deflections of equal size) is useful. In normal adults, the transition usually occurs in leads V2, V3 or V4. Transition occurring in V1 is consistent with counterclockwise rotation of the heart and is seen in right ventricular hypertrophy and posterior myocardial infarction. Transition in V5 or V6 is clockwise rotation of the heart (see Fig. 6.2).

**Intervals**

Intervals provide important information about conduction through the heart (Table 6.3). Measurement of important ECG intervals is illustrated in Figure 6.5. In general, intervals should be measured in multiple leads and the largest value should be used. The PR interval is an indicator of conduction from the SA node to the ventricles and reflects conduction through the atria, the AV node, and the His–Purkinje system (Fig. 6.6). Lead II is generally used to measure the PR interval because the initial deflections of the P wave and QRS complex are well defined although occasionally other leads are more useful. The QRS duration is a function of activation of the ventricles and should be measured in the lead with the widest QRS. The QT interval provides important information about ventricular repolarization.

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**Clinical correlate**

Einthoven’s Law states that the complex in lead II is equal to the sum of the corresponding complexes in leads I and III. This law is useful in detecting instances where the limb leads have been reversed by the technician.
### Table 6.3 Transmission of electrical impulses through the heart: conduction system intervals that can be measured on the ECG

<table>
<thead>
<tr>
<th>Interval</th>
<th>Measured</th>
<th>Normal value</th>
<th>Conditions which decrease</th>
<th>Conditions which increase</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PR</strong></td>
<td>Beginning of P wave to beginning of QRS</td>
<td>0.12–0.20 sec</td>
<td>Pre-excitation</td>
<td>Intrinsic heart disease, Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>Varies with heart rate but to a lesser extent than QT</td>
<td></td>
<td>Low atrial rhythms, Glycogen storage disease, Fabry disease, Pheochromocytoma</td>
<td>Drugs (e.g. verapamil), Vagal stimulation, Hyperkalemia</td>
</tr>
<tr>
<td><strong>QRS</strong></td>
<td>QRS duration should be measured in the lead with the widest QRS complex</td>
<td>0.06–0.11 sec</td>
<td></td>
<td>Bundle branch block, Intraventricular conduction delay, Drugs (e.g. Class 1c anti-arrhythmics), Pre-excitation, Hyperkalemia</td>
</tr>
<tr>
<td><strong>QT</strong></td>
<td>Beginning of Q wave to end of T (or U) wave</td>
<td>Varies with heart rate. QT corrected can be calculated by dividing the measured QT interval by the square root of the RR interval. The upper limit of normal for QTc is 0.39 in men and 0.41 in women</td>
<td>Digoxin, Hypercalcemia</td>
<td>Drugs (including antiarrhythmics and antipsychotic medications), Myocardial ischemia, Jervell–Lange–Neilson syndrome, Romano–Ward syndrome, CNS disease, Autonomic nervous system disease, Hypocalcemia, Hypothermia, Severe bradycardia</td>
</tr>
</tbody>
</table>

**Figure 6.5** Measuring intervals on the ECG.
Clinical correlate

A useful rule of thumb is that the QT interval should be less than half the preceding RR interval.

Figure 6.6 Anatomic features contributing to the PR interval. H, His bundle; BB, bundle branches; P, Purkinje fibers.
## CHAPTER 7

Effects of Electrolyte Abnormalities on ECG

### Table 7.1  ECG changes associated with electrolyte abnormalities.

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Increased</th>
<th>Decreased</th>
</tr>
</thead>
<tbody>
<tr>
<td>K⁺</td>
<td>Tall, narrow, peaked T waves</td>
<td>ST segment depression</td>
</tr>
<tr>
<td></td>
<td>QRS prolongation</td>
<td>Decreased T wave amplitude or T wave inversion</td>
</tr>
<tr>
<td></td>
<td>Decreased amplitude of P waves</td>
<td>Prominent U waves (mid precordial leads)</td>
</tr>
<tr>
<td></td>
<td>Absent P waves</td>
<td>QRS widening (children)</td>
</tr>
<tr>
<td></td>
<td>ST segment elevation in V1 and V2</td>
<td>Arrhythmias and AV block</td>
</tr>
<tr>
<td></td>
<td>Sine wave appearance</td>
<td>Prolongation of QTc</td>
</tr>
<tr>
<td>Ca⁺</td>
<td>Decrease in QTc (a short QT interval with minimal ST segment is characteristic of hypercalcemia)</td>
<td>Prolongation of QTc</td>
</tr>
<tr>
<td></td>
<td>No change in morphology of P or T waves</td>
<td>P and T waves usually not affected</td>
</tr>
<tr>
<td></td>
<td>PR interval and QRS duration may be prolonged</td>
<td>QRS and PR duration usually not affected</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias are uncommon although AV block has been reported</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Can worsen digitalis toxicity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Many patients with hypercalcemia develop hypokalemia</td>
<td></td>
</tr>
<tr>
<td>Na⁺</td>
<td>No effect</td>
<td>No effect</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>Minimal effects; extremely high serum magnesium levels may produce bradycardia or cardiac arrhythmias</td>
<td>Usually minimal effects but can co-exist with hypokalemia. Torsades de pointes (polymorphous ventricular tachycardia) may be precipitated.</td>
</tr>
</tbody>
</table>

The magnitude of the electrolyte gradients across cell membranes has an important influence of depolarization and repolarization of myocardial cells. Thus it is no surprise that electrolyte abnormalities can have an effect, occasionally profound, on the ECG (Table 7.1, Fig. 7.1). Recognition of these changes can provide clues to diagnosing and treating potentially life-threatening conditions.

### Hyperkalemia

The most common ECG change with hyperkalemia is tall T waves, best seen in leads II, III, and V2–V4. Tall T waves are usually seen when the potassium concentration rises above 5.5–6.5 mmol/L. However, only about one in five hyperkalemic patients will have the classic tall, symmetrically narrow and peaked T waves (“pinched” T waves); the rest will merely have large-amplitude T waves (Fig. 7.2). Hyperkalemia should be suspected when the amplitude of the T wave is greater than or equal to that of the R wave in more than one lead.
Hypokalemia  
Hyperkalemia  
Advanced hyperkalemia  
Hypocalcemia  
Hypercalcemia

Figure 7.1 Common ECG changes in various electrolyte abnormalities.

Figure 7.2 (a) ECG in a 39-year-old patient with hyperkalemia (K⁺ 6.8 mmol/L) and (b) six hours later (K⁺ 4.7 mmol/L).
At higher potassium concentrations, the P wave widens and flattens and the PR segment lengthens (Table 7.2). As the concentration rises further, the P waves may disappear.

The QRS complex will begin to widen with a potassium concentration of 7.0–8.0 mmol/L. Unlike right or left bundle branch blocks, the QRS widening in hyperkalemia affects all portions of the QRS complex and not just the terminal forces. Severe untreated hyperkalemia can cause the QRS complex to merge with the T wave (a sine wave pattern), idioventricular rhythms and asystolic cardiac arrest.

ST segment elevation in leads V1 and V2 has been reported in severe hyperkalemia. Since dialysis corrects hyperkalemia and results in normalization of the ST segment elevation, this has been referred to in older literature as the “dialyzable current of injury.”

### Hypokalemia

ECG changes are rare with mild hypokalemia but may be present with more severe hypokalemia, especially if the individual has hypomagnesemia or coronary artery disease or is taking a digitalis derivative. These changes are primarily due to delayed ventricular repolarization and include decreased T wave amplitude, ST segment depression, prolongation of the QT interval and accentuation of the U wave (Fig. 7.3). A prominent U wave in association with a small T wave in the setting of a prolonged QT interval are findings that should raise the suspicion of hypokalemia.

Ventricular extrasystoles and malignant ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation can occur with hypokalemia with the risk increased by myocardial ischemia and/or digitalis derivatives. Hypokalemia is an important cause of acquired long QT syndrome (LQTS) and can thus predispose to torsades de pointes. Hypokalemia can also cause arrhythmias due to enhanced automaticity.

### Hyper- and hypocalcemia

Alterations in calcium levels predominantly alters Phase 2 of the action potential, resulting in shortening (hypercalcemia) or prolongation (hypocalcemia) of the QT interval. The influence on the QT interval is primarily due to a modification of the duration of the ST segment although both conditions can affect T wave morphology. Changes in calcium levels generally do not cause T wave changes because they do not affect Phase 3 of the action potential, although there are case reports of altered T waves in hypocalcemia.

Hypercalcemia is associated with shortening of the QT interval (again, primarily due to shortening of the ST segment). At high calcium concentrations the duration of the T wave increases and the QT interval may then become normal. Prolongation of the PR and QRS intervals may also occur at high calcium concentrations. More rarely, second-degree or third-degree AV block and the appearance of Osborn waves (generally seen in hypothermia) have been reported.

QT prolongation can be seen in hypocalcemia and is primarily due to ST segment prolongation. The combination of hypocalcemia and hypokalemia, which is seen most frequently in patients with renal insufficiency, produces a characteristic ECG pattern of tall, narrow T waves (from hyperkalemia) and ST segment prolongation (from hypocalcemia).

### Magnesium and sodium

Magnesium and sodium concentrations within the range generally encountered in clinical practice do not produce specific ECG patterns. Hypomagnesemia can occur simultaneously with hypokalemia which can cause characteristic ECG changes.
Further reading


• right ventricular hypertrophy
• right atrial abnormality
• left ventricular hypertrophy (LVH)
• left atrial abnormality.

When interpreting an ECG in a young adult, two important caveats should be kept in mind. One is that "normal" ECG patterns change as we age. Many of the findings above are common in children but rare in adults. The same ECG findings that are normal in an 8 year old may suggest congenital heart disease if present in a 28 year old. Since ECG changes with aging vary in different individuals, there will be some individuals in whom specific ECG findings reflect cardiovascular disease whereas the same pattern may reflect a non-pathologic, persistent juvenile ECG pattern in others.

Second, ECG patterns suggestive of cardiovascular disease may be present in well-conditioned athletes but do not necessarily reflect underlying pathology. ECG findings of LVH are common in athletes and can be a normal response to exercise and reversible with deconditioning. Also common are incomplete RBBB (14–31% of athletes), early repolarization and bradycardia. These changes are more common in endurance sports (e.g. rowing or long-distance running) and in sports with high peak level of activity (e.g. basketball or football). Note that the diagnosis of "athlete's heart" is one of exclusion and all athletes, most especially those with symptoms, should have a complete work-up before attributing an abnormal ECG to cardiac conditioning.2

This chapter will focus on findings on a 12-lead ECG with the patient in sinus rhythm that are significant cardiovascular disease in young adults (in the next chapter ECG patterns that are indicative of an increased risk of ventricular arrhythmias and/or sudden cardiac death will be discussed). The ECG has several limitations – it is neither sensitive nor specific and is rarely diagnostic of a specific condition. It is, however, readily available and when properly interpreted can prompt further evaluation (e.g. echocardiography) that will provide specific information as to the presence of congenital heart disease or valvular heart disease.

ECG patterns suggestive of hemodynamically important congenital (Table 8.1) or valvular heart disease reflect abnormalities of one of the cardiac chambers.1 Most commonly, these cardiovascular conditions will alter right heart pressures and/or volumes and thus lead to ECG changes reflective of right atrial abnormalities (RAA) or right ventricular hypertrophy (RVH). Less commonly, abnormal hemodynamics will affect the left atrium and/or left ventricle (LV), leading to ECG changes pointing to these chambers. In this chapter several ECG findings that may be clues to the presence of underlying cardiovascular disease in young adults will be discussed. These include:

- tall R wave in lead V1
- right bundle branch block (RBBB)
- right axis deviation (RAD)
suggestive of underlying cardiovascular pathology. Arrhythmias will not be discussed although they can be a marker of underlying cardiac or pulmonary disease. Also, ECG findings in patients acutely ill (e.g., with an acute myocardial infarction (MI) or pulmonary embolus) will not be mentioned as these are covered in detail in specific chapters.

Table 8.1 Common ECG findings in adult patients with congenital heart disease. Note that the ECG findings are dependent upon the hemodynamic effects of the congenital heart defect. PDA, patent ductus arteriosus; ASD, atrial septal defect; VSD, ventricular septal defect; MS, mitral stenosis; PS, pulmonic stenosis; PHTN, pulmonary hypertension; TOF, tetralogy of Fallot.

<table>
<thead>
<tr>
<th>QRS axis</th>
<th>QRS</th>
<th>RAA</th>
<th>RVH</th>
<th>LAA</th>
<th>LVH</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secundum ASD</td>
<td>Normal or RAD</td>
<td>rsR' in V1, IRBBB or RBBB</td>
<td>+</td>
<td></td>
<td></td>
<td>1AVB is common</td>
</tr>
<tr>
<td>Primum ASD</td>
<td>LAD</td>
<td>rsR' in V1</td>
<td>+</td>
<td></td>
<td></td>
<td>1AVB is common</td>
</tr>
<tr>
<td>Sinus venosus ASD</td>
<td>Normal</td>
<td>Deep S wave in V1/-&gt; tail R waves in V5 and V6</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td>Ectopic atrial rhythm is common</td>
</tr>
<tr>
<td>PDA</td>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PS or PHTN</td>
<td>RAD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VSD</td>
<td>RAD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dextrocardia</td>
<td>RAD</td>
<td>Small R waves in left precordial leads</td>
<td></td>
<td></td>
<td></td>
<td>ECG findings are dependent upon hemodynamic effects</td>
</tr>
<tr>
<td>Unrepaired TOF</td>
<td>RAD</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>ECG is abnormal in 95%</td>
</tr>
</tbody>
</table>

Findings suggestive of abnormal right heart hemodynamics

A pronounced R wave in lead V1

V1 is the lead closest to the RV and thus is one of the most sensitive indicators of right ventricular pressure or volume overload. A pronounced R wave in V1, in the presence of a normal QRS duration, is a common finding in children, especially those less than 8 years. The prevalence is less in older children, less still in young adults and rare in older adults. An R wave in V1 is generally considered abnormal when >6 mm (Fig. 8.1). Also abnormal are small S waves (<2 mm) and/or large R prime (R') waves (>10 mm). Another useful indicator is an R/S ratio of >1; this occurs in less than 1% of adults. Any of these findings should prompt consideration of RVH, especially in the presence of RAD.

A pronounced R wave in V1 can be a normal variant, especially in younger adults. Other causes of a large R wave in V1 include lead misplacement, posterior myocardial infarction (MI), Duchenne muscular dystrophy, type A pre-excitation pattern (Wolff–Parkinson–White) and displacement of the heart due to pulmonary disease (Fig. 8.2).

Right bundle branch block

Right bundle branch block can be a marker of dilation of the RV and thus may reflect cardiovascular disease leading to RV volume or pressure overload. The prevalence of RBBB increases with age and it is unusual in young adults. In a study of 237,000 airmen under the age of 30 years, the incidence of RBBB was only 0.2%. In the presence of RBBB, the T wave will be discordant to the terminal forces of the QRS complex (i.e., the T wave will be deflected opposite the terminal deflection of the QRS complex). Note that the discordance is between the T wave and the terminal deflection of the QRS, not with predominant deflection of the QRS.

The right bundle branch originates from the bundle of His, courses through the ventricular septum to the apex and then proceeds to the RV free wall. RBBB reflects a delay in RV depolarization (via the right bundle branch), usually resulting in a
large terminal R’ wave in V1 (rSR’) and a broad terminal S wave in leads I, aVL, and V6. The World Health Organization (WHO)/International Society and Federation for Cardiology Task Force criteria for the diagnosis of RBBB include: QRS duration >120 msec; an rsr’ pattern in leads V1 and V2 (an rsr’ pattern is present when there are two positive deflections in the QRS complex; if there is a marked difference in the size of the r and r’ voltage, the predominant deflection is indicated by which letter is capitalized); S wave longer than 40 msec in V6 and I; normal R peak time in leads V5 and V6 but ≥50 msec in V1.4 The term incomplete right bundle branch block is used to describe the ECG when changes such as those described are present but the QRS interval is between 80 and 110 msec. Note that the QRS axis is unaffected by RBBB and thus axis deviation (either right or left) should prompt consideration of conditions that result in axis deviation.

**Right axis deviation**

Right axis deviation is normal in infants and children but unusual in adults. In one study, an axis >105 degrees was found in only 2% of “normals”
between the ages of 20 and 30 years. RAD can reflect right heart pathology as a hypertrophied RV can generate forces that balance and/or exceed the electrical forces generated by the usually much larger LV. The combination of a large R wave in V1 and RAD is highly suggestive of RVH. Other conditions associated with RAD include arm lead reversal, left posterior hemiblock, lateral wall MI or lung disease. RAD can also be a normal variant, especially in individuals who are tall and slender.

**Right ventricular hypertrophy**

In the normal heart, RV voltage is almost completely masked by LV voltage and thus prominent RV forces are unusual and suggestive of RVH. As the RV develops increased muscle mass (i.e. hypertrophies), electrical voltage generated by the RV increases. This produces progressive anterior and rightward displacement of the QRS vector. Increases in RV muscle mass can cause prolongation of RV depolarization, resulting in an increase in the duration of the QRS complex. These changes become more pronounced as the degree of hypertrophy increases.

There are different types of ECG patterns which have, with various success, been correlated to different causes of RVH. The first type is associated with lesions causing pressure overload of the RV and is characterized by a tall R wave in V1. This pattern has been associated with congenital pulmonic stenosis, tetralogy of Fallot, primary pulmonary hypertension and other conditions associated with RV outflow obstruction. A second type, characterized by an rsr’ pattern, is seen in conditions with RV volume overload such as atrial septal defect. Lastly, ECG evidence of RVH in the presence of lung disease is affected by the spatial orientation of the heart and is characterized by low R wave amplitude and a posteriorly and superiorly oriented QRS vector (Fig. 8.3).

The sensitivity and specificity of the ECG diagnosis of RVH are limited and this has led to a proliferation of criteria for the diagnosis of RVH. The sensitivity and specificity of the different criteria vary depending on the particular study and the patient population but in general for all of these formulas, specificity is greater than sensitivity. There are numerous criteria for the diagnosis of RVH but it is probably necessary only to keep the most common criteria in mind.

- Tall R waves in RV leads; deep S waves in LV leads
- R/S ratio in V1 > 1

![Figure 8.3](image-url) Right ventricular hypertrophy in a patient with lung disease. Note the right axis deviation, relatively large R wave in V1, low limb lead voltage, relatively large R wave in aVR and deep S waves in the lateral precordial leads. This ECG is from an 18-year-old male with cystic fibrosis.
CHAPTER 8 ECG Clues to the Presence of Congenital or Valvular Heart Disease

- R wave in V1 > 7 mm
- S wave in V1 of less than 2 mm
- rsr’ in V1 with R’ > 10 mm
- S1S2S3 pattern
- R/S ratio in V5 or V6 < 1
- R wave in aVR of more than 5 mm

The ECG diagnosis of RVH is enhanced if there are other findings of right heart pathology in the presence of increased RV voltage. These include RAA, RAD, an increase in QRS duration and/or ST and T wave abnormalities in the right precordial leads directed opposite to the predominant QRS direction (i.e. wide QRS–T angle). These findings are analogous to those which increase the likelihood of LVH in the presence of increased LV voltage.

**Clinical correlate**

A simple rule that is important to remember is: RAD + tall R wave in V1 = RVH.

**Right atrial abnormality**

Normally the P wave is formed by overlapping depolarization of the right and left atria, leading to a smooth rounded wave less than 0.12 sec in duration and less than 1 mm in height. The forces generated by right atrial depolarization are directed anteriorly and inferiorly and produce the early part of the P wave. In the presence of right atrial enlargement, P wave amplitude increases although the overall duration of the P wave is usually not prolonged. As P-pulmonale progresses, the voltage in the P wave increases both from delayed activation of the right atrium, causing simultaneous activation of the right and left atria, and the increase in right atrial tissue that is depolarizing.

P-pulmonale is defined as tall (≥ 2.5 mV), peaked P waves in any of the inferior leads (II, III and aVF) with normal P wave duration (Fig. 8.4). Less consistently, it can also include a positive deflection in the P wave in V1 or V2 of ≥ 1.5 mm. Remember that since the right atria depolarizes first, the first part of the P wave is indicative of the right atrium in V1. P-pulmonale is a marker of right atrial dilation or hypertrophy and can be seen in chronic obstructive pulmonary disease (COPD), pneumonia, congenital heart disease, congestive heart failure, pulmonary emboli, asthma or tricuspid valve disease.

The presence of an ectopic atrial rhythm can be a clue to congenital heart disease (e.g. sinus venosus atrial septal defect). The SA node is the normal pacemaker of the heart and is located in the posterior part of the right atrium at the junction of the superior vena cava. When the pacemaker function is controlled by the sinus node, P waves are positive in leads I, II and aVF and negative in aVR (the normal P wave axis is 15–75 degrees). An ectopic atrial rhythm means that the pacemaker function is now controlled by a focus within the atria (usually due to increased triggered automaticity). Normally the PR interval will remain within normal limits and the atrial rate will be below 100 beats per minute but the P wave morphology and axis will change. When the focus is lower in the atrium, the P waves in the inferior leads are inverted and the rhythm can be mistaken for an AV junctional rhythm.

**Findings suggestive of abnormal left heart hemodynamics**

**Left ventricular hypertrophy**

Electrical forces generated during LV activation produce the normal QRS complex. With an increase in the amount of LV myocardium (LVH), electrical preponderance of LV over RV is further accentuated. The mean vector of the LV becomes more posterior and leftward, increasing QRS complex voltage and ventricular activation time (see Fig. 8.5). Additional ECG clues to the diagnosis of LVH are left axis deviation, increased QRS duration, ST-T abnormalities (a “strain pattern”) and the presence of left atrial abnormality (LAA) (Fig. 8.5) (see Chapter 26 for a more detailed discussion).
There are dozens of published criteria that can be used to diagnose LVH on an ECG, although none is universally accepted. Some of these criteria are based solely on voltage present on the ECG (e.g. the Sokolow–Lyon Voltage Criteria and the Cornell Voltage Criteria) whereas others also take into account related findings such as left axis deviation, LAA and ST-T wave abnormalities (e.g. the Estes Criteria). For all of these formulas, the specificity tends to be high while the sensitivity is much lower. To further complicate the situation, the sensitivity and specificity of the various ECG criteria vary with age, gender and obesity. Modified formulas that “correct” for these variables have been developed for some of the specific criteria (e.g. Cornell Voltage Criteria). Despite all of these limitations, the ECG remains a useful, although far from perfect tool to identify ventricular hypertrophy.

On voltage criteria alone, there is general agreement that LVH may be diagnosed based on increased voltage in the limb leads (R wave in lead I plus S wave in lead III >25 mm or R wave in lead aVL >11 mm or R wave in lead aVF >20 mm) or increased voltage in the precordial leads (S wave in V1 exceeding 24 mm or R wave in leads V4, V5 or V6 >26 mm or R wave in leads V5 or V6 plus S wave in lead V1 >35 mm).

**Left atrial abnormality**

Left atrial abnormality is suggested by notching or prominence of the terminal portion of the P wave in the limb leads, prominent negativity of the terminal portion of the P wave in V1 or a P wave duration of >120 msec (see Fig. 8.4). Normal P waves may be bifid in the limb leads with a minor notch probably resulting from slight asynchrony between right and left atrial depolarization. However, a pronounced notch with a peak-to-peak interval of >0.04 sec suggests LAA. In V1, the P wave is often biphasic. This results because early right atrial forces are directed anteriorly, giving rise to an initial positive deflection whereas left atrial forces travel posteriorly, producing a later negative deflection. A large negative deflection (>1 small square in area) suggests LAA.
P-mitrale is defined as a widened P wave (≥0.12 sec) with normal or slightly increased voltage that is notched, bifid or flat-topped. It is a sign of long-standing mitral valve disease.

References
Sudden cardiac death is a catastrophic occurrence, especially when it occurs in an apparently healthy young adult. It is thus essential to recognize ECG patterns of congenital cardiovascular diseases that put a patient at an increased risk of ventricular arrhythmias and sudden cardiac death. In some cases there are specific treatments, either pharmacologic, mechanical (e.g. ablation) or implantable cardioverter defibrillators, which reduce the risk of sudden death.

In this chapter, some of the conditions that predispose an individual to an increased risk of sudden cardiac death and which have a characteristic ECG pattern will be reviewed. Patients with these disorders may be symptomatic or asymptomatic, and in many cases the disorder is diagnosed by an astute clinician on an ECG obtained for an unrelated reason. These diseases can be classified as purely electrical heart diseases (e.g. long QT syndrome, Wolff–Parkinson–White or Brugada syndrome) or disorders that have an effect on cardiac structure and function (e.g. hypertrophic cardiomyopathy or arrhythmogenic right ventricular dysplasia). This chapter is not exhaustive and there are other congenital heart defects, especially those with hemodynamic effects on the heart, that can increase the risk of sudden cardiac death (the increased risk can persist despite surgical repair). Some of these disorders were reviewed in the previous chapter. Lastly, there are several causes of sudden cardiac death in young adults that may be electrically silent.

Examples of these disorders include coronary artery disease, coronary artery anomalies and cardiomyopathies.

Brugada syndrome

The Brugada syndrome is a heterogeneous genetic disease characterized by abnormal electrophysiologic activity in the right ventricular epicardium. Approximately one-fourth of cases are caused by loss of function mutations in the cardiac sodium channel SCN5A (abnormalities in this gene have also been linked to long QT syndrome 3). The characteristic ECG pattern of the Brugada syndrome is ST elevation (≥2 mm at the J point) in the right precordial leads (Fig. 9.1). In some patients, complete or incomplete right bundle branch block is present. In others, high take-off ST segment elevation (accentuated J point elevation) in the right precordial leads mimics the pattern of right bundle branch block, but wide S waves in leads I, aVL, V5 and V6 typically seen in right bundle branch block are absent.4,5

There are three subtypes of Brugada syndrome. In type 1 the ST segment is continuously down-sloping from the top of the R’ wave, is not elevated above baseline at the terminal portion and ends with an inverted T wave. Types 2 and 3 have a “saddleback” ST-T wave configuration in which the ST segment descends towards the baseline with upward concavity and then rises again to an upright or biphasic T wave (the difference between types 2 and 3 is related to the elevation of the terminal portion of the ST segment). The Brugada pattern on ECG can be either persistent or inducible and
the ECG changes can be dynamic, with the same patient manifesting all three types at various points in time.

Diagnosis of Brugada syndrome depends on both characteristic ECG findings (either spontaneous or inducible) and appropriate clinical findings (unexplained syncope, self-terminating polymorphic ventricular tachycardia, documented ventricular fibrillation, family history of sudden cardiac death at less than 45 years of age, Brugada ECG pattern in a family member, and/or inducibility of ventricular tachycardia by electrophysiologic study). A patient who presents with the Brugada ECG criteria but without the clinical characteristics is said to have the Brugada pattern but not the syndrome. The Brugada ECG pattern is seen much more frequently in men than in women, and there may be an increased frequency in Asians. The criteria that establish a definitive diagnosis of Brugada syndrome remain under debate.

Arrhythmias and sudden death in the Brugada syndrome generally occur during sleep or at rest.
and are commonly associated with bradycardia. Specific factors that have been reported to trigger fatal arrhythmias include fever, antiarrhythmic drugs, beta-blockers, tricyclic antidepressants, alcohol, cocaine and electrolyte imbalances such as hypokalemia, hyperkalemia, and hypercalcemia. Exercise has not been linked to sudden cardiac death. Patients with Brugada syndrome tend to present later in life than some other forms of inherited arrhythmias (mean age at death = 45 years).

**Long QT syndrome**

The QT interval measures the time needed for ventricular depolarization and repolarization but in the presence of a normal QRS duration, long QT intervals occur when there is a prolongation of repolarization (see Fig. 9.1). A prolonged QT interval can be either acquired or genetic. Causes of acquired prolonged QT intervals include severe hypothermia, hypokalemia, severe hypocalcemia, hypothyroidism, antiarrhythmic drugs in class IA and III, other medications (e.g. haloperidol, methadone, etc.), severe bradycardia, atrioventricular block, myocardial ischemia, and neurogenic causes (including organophosphorus). Previously unrecognized long QT syndrome (LQTS) is present in 5–20% of patients with drug-induced torsades de pointes.

LQTS is a genetic disorder that is associated with polymorphous ventricular tachycardia (torsades de pointes) causing syncope and sudden cardiac death. LQTS is a disease of cardiac ion channels with clinical manifestations being linked to over 300 mutations in 10 different genes with mutations in three genes comprising the vast majority of cases. LQTS1 and LQTS2 are caused by mutations in genes that cause a decrease in repolarizing K potassium ion channels whereas LQTS3 is caused by mutations of the SCN5A sodium channel gene (Table 9.1). Increased sympathetic stimulation can provoke arrhythmias in LQTS1 and LQTS2 patients. In LQTS1, exercise (especially swimming) and emotional stress can precipitate syncope and sudden cardiac death. Arrhythmic events in LQTS2 can occur at stress or rest; triggering by unexpected loud noises (e.g. alarm clock) is very suggestive of LQTS2. LQTS1 and LQTS2 account for the majority of all LQTS cases. LQTS3 accounts for <10% of all LQTS cases and individuals with LQTS3 tend to experience cardiac events while sleeping. LQTS3 can also be associated with bradycardia and syncopal events can be precipitated by slow heart rates in addition to rapid ventricular arrhythmias.

**Short QT syndrome and catecholaminergic polymorphic ventricular tachycardia**

Brugada syndrome and LQTS are examples of channelopathies – disorders of ion channels. There are two other channelopathies associated with an increased risk of sudden cardiac death which deserve mention. The short QT syndrome is a recently described entity that is characterized on the 12-lead ECG by a QTc of less than 300 msec (ranging from 220 to 300 msec) and the lack of a clear ST segment with the T wave originating immediately after the S wave. In approximately one-half of patients, tall peaked symmetric T waves are apparent in the right precordial leads. The disorder is familial and appears to be due to gain-of-function mutations in genes encoding cardiac potassium channels. There are limited data regarding triggers for sudden cardiac death.

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is characterized by bidirectional or polymorphic ventricular tachycardia occurring during sympathetic stimulation. These patients usually present with syncope or sudden cardiac death during exercise or emotional stress and do not have any evidence of structural heart disease. The 12-lead ECG in the absence of ventricular tachycardia is usually normal although lower than normal resting heart rate and a high incidence of prominent U waves have been reported by some authors. Mutations in the gene encoding the sarcoplasmic reticulum ryanodine receptor (RYR2) cause roughly half of cases, with mutations in CASQ2 being found in a few individuals.

**Arrhythmogenic right ventricular dysplasia**

Arrhythmogenic right ventricular dysplasia (ARVD) is a disorder in which there is patchy replacement of the normal myocardium by fatty and/or fibrofatty tissue. The disease is genetically heterogeneous...
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Characteristic ECG finding</th>
<th>Gene(s) linked to the disease</th>
<th>Common triggers of syncope or SCD</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brugada</td>
<td>ST elevation (≥2 mm at the J point) in the right precordial leads with complete or incomplete complete or incomplete right bundle branch block</td>
<td>SCN5A in 25%</td>
<td>Rest or sleep, commonly with bradycardia. Other reported triggers include fever, antiarrhythmic drugs, beta-blockers, tricyclic antidepressants, alcohol, cocaine and electrolyte imbalances</td>
<td>Men &gt; women</td>
</tr>
<tr>
<td>LQTS1</td>
<td>Long QT interval</td>
<td>KCNQ1</td>
<td>Exercise (e.g. swimming) or emotional stress</td>
<td>Most common form of LQTS</td>
</tr>
<tr>
<td>LQTS2</td>
<td>Long QT interval</td>
<td>KCNH2</td>
<td>Can occur at stress or rest. Triggering by loud noises is very suggestive of LQTS2</td>
<td>Risk increases during post-partum period</td>
</tr>
<tr>
<td>LQTS3</td>
<td>Long QT interval</td>
<td>SCN5A</td>
<td>Rest or sleep</td>
<td>Disorder is also associated with bradycardia</td>
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<tr>
<td>Short QT syndrome</td>
<td>Short QT interval</td>
<td>KCNH2, KCNQ1, and KCNJ2</td>
<td>Most common form of LQTS</td>
<td>Recently identified and thus data are limited; associated with atrial fibrillation</td>
</tr>
<tr>
<td>Catcholaminergic polymorphic ventricular tachycardia</td>
<td>Usually normal; lower than normal resting heart rate and a high incidence of prominent U waves have been reported by some authors</td>
<td>RYR2 and CASQ2</td>
<td>Exercise or emotional stress</td>
<td>Generally manifests early in life and thus is more common in children and adolescents than in adults</td>
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<td>Arrhythmogenic RV dysplasia</td>
<td>a) Epsilon waves; b) T wave inversion in leads V1–V3 in the absence of RBBB; and c) PVCs that originate from the right ventricle</td>
<td>Desmoplakin, plakoglobin and ryanodine receptor genes</td>
<td>Exercise or emotional stress</td>
<td>Epsilon waves are indicative of localized QRS prolongation (&gt;110 msec) in V1–V3 and are often present in the terminal potential in leads V1–V3 causes QRS duration to exceed the QRS duration in lead V6 by more than 25 msec</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Left ventricular hypertrophy, repolarization abnormalities (e.g. “strain pattern” in V4–V6), left atrial abnormality, and Q waves in inferior and lateral leads</td>
<td>Cardiac sarcomere genes (e.g. beta-myosin heavy chain, cardiac troponin T, and myosin-binding protein C)</td>
<td>Exercise</td>
<td></td>
</tr>
<tr>
<td>Wolff–Parkinson-White syndrome</td>
<td>Short PR interval, widening of the QRS complex and delta waves (slurred, slowly rising onset of QRS)</td>
<td>PRKAG2</td>
<td>Exercise</td>
<td>Atrial fibrillation can precipitate sudden cardiac death</td>
</tr>
</tbody>
</table>

Table 9.1 Characteristic ECG findings in various cardiovascular disorders associated with an increased risk of sudden cardiac death.
with mutations having been identified in the genes for desmoplakin, plakoglobin and the ryanodine receptor. ARVD is characterized clinically by ventricular arrhythmias (originating from the RV) and more rarely by RV pump failure. The disease is progressive and as the myocardium is replaced by fatty or fibrofatty tissue, the RV becomes dilated and dysfunctional. Arrhythmias and sudden cardiac death are more common during exercise.

Common ECG findings include T wave inversion in leads V1–V3 and premature ventricular contractions (PVCs) that originate from the right ventricle (i.e. with a left bundle branch block pattern; see Fig. 9.1). Epsilon waves, small deflections at the terminal end of the QRS complex that are best seen in V1–V3, are a specific but not a sensitive marker of the disease. Epsilon waves are indicative of localized QRS prolongation (>110 msec) in V1–V3 and are present if the terminal potential in leads V1–V3 causes QRS duration to exceed the QRS duration in lead V6 by more than 25 msec.

Electrocardiogram abnormalities are detected in more than 90% of patients with ARVD. The juvenile pattern of T wave inversion in leads V1–V3 is a normal variant in children less than 12 years of age, rare in adults >19 years (found in 1–3% of healthy individuals) but present in 87% of patients with ARVD. The differential diagnosis of these ECG findings includes myocarditis, Naxos disease, sarcoid heart disease, dilated cardiomyopathy, and right ventricular outflow tract tachycardia.

**Hypertrophic cardiomyopathy**

Hypertrophic cardiomyopathy (HCM) is caused by a mutation in one of the genes encoding proteins of the cardiac sarcomere. At least 10 different genes have been linked to HCM, but three predominate: beta-myosin heavy chain, cardiac troponin T, and myosin-binding protein C. Familial HCM is an autosomal dominant disease and present in 50% of patients with HCM; spontaneous mutations are suspected for sporadic forms of the disease.16,17

HCM can present at any age. Many patients are asymptomatic throughout life but others will develop dyspnea, angina, palpitations and syncope. Symptoms primarily result from hemodynamic alterations which include dynamic left ventricular outflow tract obstruction, mitral regurgitation, diastolic dysfunction and myocardial ischemia. The most feared complication of the disease is sudden cardiac death, primarily due to ventricular arrhythmias.

There is no characteristic ECG pattern that is diagnostic of HCM but the 12-lead ECG is abnormal in 75–95% of HCM patients. The most common ECG findings in HCM are due to left ventricular hypertrophy (LVH) and include increased voltage and repolarization abnormalities (Fig. 9.1).18 T wave inversion and ST segment depression with upward convexity are commonly seen in V4–V6 (commonly although incorrectly known as a “strain” pattern), while tall T waves and ST segment elevation are present in the right precordial leads. Because many HCM patients have septal hypertrophy, abnormal Q waves are common. These Q waves commonly mimic those of a myocardial infarction. The increased amplitude of electrical forces from depolarization of the hypertrophied septum can cause deep Q waves in the lateral and inferior leads and taller R waves in the right precordial leads. T wave abnormalities are present in most symptomatic HCM patients. Septal hypertrophy causes the T wave to be directed opposite to the prominent septal depolarization force, so the T wave will be positive in leads with a deep Q waves. Left atrial abnormality and prolonged QTc interval can also be seen in patients with obstructive HCM. Atrial fibrillation is the most common sustained arrhythmia in HCM but atrial flutter, ventricular ectopy, ventricular tachycardia, and ventricular fibrillation can also occur.

In summary, there is a wide variety of patterns of ECG changes in patients with HCM including LVH, repolarization abnormalities (e.g. T wave inversion and ST segment depression with upward convexity in V4–V6), left atrial abnormality, and Q waves in inferior and lateral leads. The ECG is not diagnostic but rather serves to raise suspicion of HCM.

**Wolff–Parkinson–White syndrome**

Wolff–Parkinson–White syndrome (WPW) is characterized by “pre-excitation” of the ventricles. This process occurs via an accessory pathway (also known as a bypass tract), a thin filamentous structure that has conductive properties, thus enabling electrical impulses from the atrium to reach the
ventricles without passing through the atrioventricular (AV) node. Accessory pathways, in general, do not have decremental conduction (defined as rate-dependent prolongation of conduction time) and thus do not have the normal safety mechanisms of the AV node in controlling ventricular rates. The most common bypass tract is a direct connection between the atria and ventricles and is known as a Kent bundle although numerous other locations of accessory pathways have been reported (e.g. atriofascicular, fasciculoventricular, intranodal or nodoventricular).

ECG findings of pre-excitation include a short PR interval, widening of the QRS complex (>0.12 sec) and delta waves (slurred, slowly rising onset of QRS) (see Fig. 9.1). Less commonly, repolarization abnormalities (e.g. T wave inversions) will also be present. The PR interval, the time required for the atria to depolarize and the wave of depolarization to arrive at the ventricles, is shortened in pre-excitation syndromes because the accessory pathway permits the impulse to bypass the AV node. A portion of the ventricles (depending on where the accessory pathway inserts) activates first leading to an initial slurring or “delta wave” at the start of the QRS complex. Conduction also occurs via the AV node and thus simultaneous activation of both ventricles can occur via the His–Purkinje system. Thus the size of the delta wave and the QRS duration is a function of where in the ventricles the accessory pathway inserts and the amount of relative conduction between the accessory pathway and the AV node.19,20

The most frequently encountered arrhythmias in WPW (in order of occurrence) are: orthodromic atrioventricular re-entrant tachycardia (AVRT); atrial fibrillation; and antidromic AVRT. The AVRT arrhythmias are due to a re-entry circuit that involves the AV node and the accessory pathway(s) and are generally triggered by an atrial ectopic beat. Orthodromic AVRT involves antegrade conduction down the AV node (with retrograde conduction via the accessory pathway) and results in a narrow complex tachycardia since the ventricles are activated via the normal conduction system. The most common complaint is palpitations that are triggered by caffeine intake, stress, exercise (during or in recovery) or hormonal changes (in women). In more than 80% of cases, the tachycardia originates from the RVOT leading to a LBBB configuration with an inferior axis. Right ventricle outflow tract tachycardia and other causes of ventricular tachycardia in the absence of structural heart disease and with a normal resting 12-lead ECG

Right ventricular outflow tract (RVOT) tachycardia is due to cAMP-mediated triggered activity, usually presents between the ages of 20 and 40 years old and generally is precipitated by a specific trigger. Two typical presentations have been described: non-sustained, repetitive, monomorphic ventricular tachycardia and paroxysmal, exercise-induced, sustained ventricular tachycardia. The most common complaint is palpitations that are triggered by caffeine intake, stress, exercise (during or in recovery) or hormonal changes (in women). In more than 80% of cases, the tachycardia originates from the RVOT leading to a LBBB configuration with an inferior axis.
In most of these cases, the tachycardia originates from the muscular tissue just below the pulmonic valve. A tachycardia can also originate from other sites such as within the pulmonary artery or the left ventricular outflow tract (LVOT). The diagnosis of RVOT tachycardia is one of exclusion and other causes of tachycardia must be excluded. Note particularly that ventricular tachycardia in ARVD may have LBBB with an inferior axis, similar to RVOT tachycardia. Prognosis of patients with RVOT tachycardia is excellent and sudden death is rare.

Other causes of ventricular tachycardia in young adults in the absence of structural heart disease and with a normal resting 12-lead ECG include Brugada syndrome (the characteristic ECG findings may be dynamic and not present when the ECG is obtained), idiopathic left ventricular tachycardia (ILVT), idiopathic propranolol-sensitive VT (IPVT), and catecholaminergic polymorphic VT. Most cases of ILVT are verapamil-sensitive infratricuscular tachycardias with a right bundle branch block (RBBB) and left axis configuration.

Catecholaminergic polymorphic ventricular tachycardia often manifests at a young age and thus is more common in children and adolescents than in adults. It is a genetic disorder caused by mutations in the ryanodine receptor 2 gene (RYR2) and the cardiac calsequestrin 2 gene (CASQ2) (i.e. genes involved in the calcium homeostasis of cardiac cells). The disease is characterized by ventricular arrhythmias (usually polymorphic ventricular tachycardia or ventricular fibrillation), that tend to occur more often with physical exercise and emotional stress.

References

PART III

Specific ECG Tracings and Diagnoses
SECTION A
Conduction System Disease
You are the emergency room physician on duty when a 31-year-old male is brought in by the rescue squad with complaints of palpitations and dizziness. Past medical history is remarkable for an atrioventricular (AV) canal defect that was surgically repaired in infancy.
**Atrial flutter with 1:1 conduction**

The ECG shows a regular, wide complex tachycardia at a rate of approximately 250–270 bpm. The differential diagnosis is limited and includes atrial flutter with 1:1 conduction, AV reciprocating tachycardia and ventricular flutter. If you are evaluating a patient with a heart rate near 300 bpm, make sure that you are confident about the width of the QRS. Occasionally you will see a narrow complex rhythm at these rates, but ventricular rhythms are more common than supraventricular rhythms and occasionally a patient will be misdiagnosed because of a cursory review of an ECG.

If the patient is hemodynamically stable, you can attempt to make the diagnosis using carotid sinus massage or adenosine. Obviously if the patient is not tolerating the rate, immediate cardioversion is necessary.

This patient was given adenosine with the following result:

Adenosine elicits transient AV block and enables the visualization of flutter waves (arrows). This patient had atrial flutter with 1:1 conduction. He underwent electrical cardioversion and the ECG after cardioversion is shown below. Note the underlying right bundle branch block (RBBB) and left anterior fascicular block (LAFB) which explains the wide QRS complex during a supraventricular arrhythmia. Long term therapy would consist of an EP study with ablation of the flutter circuit +/- pharmacological treatment if required.
You are on duty when a 31-year-old, previously healthy female presents to the emergency room with complaints of the sudden onset of dyspnea.
Atrioventricular nodal re-entry tachycardia

This ECG shows a regular, narrow complex tachycardia at a rate of 140 bpm. The differential diagnosis includes: atrioventricular nodal re-entry tachycardia (AVNRT), atrial flutter with 2:1 AV block, sinus tachycardia, atrial tachycardia and AV reciprocating tachycardia (AVRT; a manifestation of Wolff–Parkinson–White syndrome or pre-excitation). This patient most likely has AVNRT with retrograde activation of the atria. This is indicated by inverted P waves superimposed on the end of the QRS complex or within the ST segment (arrows in lead II above). In this patient, P waves can be seen at the end of the QRS in leads II, III, and aVF because they distort terminal vector forces in these leads leading to pseudo-S waves. In some patients, retrograde P waves will cause a pseudo r' in V1 that mimics the RSr' or incomplete RBBB.

AVNRT is the most common type of re-entrant supraventricular tachycardia (SVT). The AV node can be functionally divided into 2 pathways based upon the speed of conduction through these two pathways (cleverly labelled the “fast” and the “slow” pathways). In 20% of individuals, however, the speed of conduction varies enough between the two pathways that a re-entrant circuit can form. The tachycardia is initiated when a premature beat is blocked in one pathway but conducts in the other pathway. Usually, AVNRT is initiated when an atrial premature complex is blocked in the fast pathway and conducts in the slow pathway but AVNRT can also be precipitated by premature ventricular complexes. In the majority of patients, during AVNRT, antegrade conduction occurs to the ventricle over the slow (alpha) pathway and retrograde conduction occurs over the fast (beta) pathway. AVNRT is labeled as “atypical” when antegrade conduction occurs in the fast pathway and retrograde conduction in the slow pathway.

Clinically, AVNRT is characterized by sudden onset and abrupt termination. It is more common in females with an increase in incidence between 20 and 40 years old. Episodes may be brief or last for hours and, in the absence of coronary artery disease or structural heart disease, are usually well tolerated. Common symptoms include palpitations, anxiety, light-headedness, and dyspnea.
In a patient with a regular, narrow complex tachycardia, AVNRT needs to be differentiated from atrial flutter with 2:1 AV block, sinus tachycardia, atrial tachycardia and AVRT. A 12-lead ECG is essential as a rhythm strip provides limited information in regards to P wave morphology. If the P wave is upright (in leads II, III and aVF) and precedes the QRS complex, the impulse is most likely originating in the sinus node. In addition to sinus tachycardia, other possibilities include sinus node reentry or sinoatrial reentry. Two P waves preceding each QRS, especially if they are in a ‘sawtooth’ pattern, should suggest the diagnosis of atrial flutter (Fig. 11.1). If the P wave is inverted and precedes the QRS complex, consider atrial tachycardia. If the P wave follows the QRS complex, this implies retrograde activation of the atria and thus AVNRT, AVRT and ventricular tachycardia should be considered. The P wave can be absent (generally buried in the QRS complex) and in junctional tachycardia.

If a patient has a narrow complex tachycardia and is stable in terms of symptoms and blood pressure, adenosine is often given during continuous ECG monitoring. This can serve as a diagnostic aide (flutter waves may become apparent during AV block) and also as a treatment. Because adenosine interrupts the re-entry pathway, AVNRT will often convert to sinus rhythm. Patients with recurrent episodes can be educated to use vagal maneuvers (e.g. Valsalva, drinking cold water, etc.) to terminate episodes. For patients with recurrent episodes who desire definitive treatment, radiofrequency ablation can be curative in 95% of individuals.

Figure 11.1 Rhythm strip in lead II showing characteristic pattern of atrial flutter.
A 26-year-old male without any significant past medical history presents to the emergency department complaining of sudden onset of a rapid heart beat. He was “celebrating” (it is not clear what) earlier this evening and has consumed a large quantity of alcohol. What does the ECG show?
Atrial fibrillation

The ECG shows an irregular, narrow complex tachycardia at a rate of approximately 150 bpm. The lack of P waves and presence of oscillatory f waves (small, irregular waves seen as a rapid-cycle baseline fluctuation) point to atrial fibrillation. The various amplitudes of QRS complexes and variable RR intervals are also consistent with atrial fibrillation. Atrial fibrillation is a disorganized and nonsynchronous series of fibrillatory waves at a rate of 400–700 bpm that, in the majority of cases, originate within the pulmonary veins (recognition of this fact has led to the development of pulmonary vein isolation as a form of atrial fibrillation ablation). Atrioventricular (AV) node conduction is variable leading to the irregularly irregular rhythm which is typical of atrial fibrillation. The ventricular response to atrial fibrillation depends on many factors, but is determined by the number of impulses that overcome the AV node’s refractory period and the His–Purkinje system’s refractory period to reach the ventricles.

There are two arrhythmias which cause the vast majority of irregular, narrow complex tachycardias, atrial fibrillation and multifocal atrial tachycardia (MAT), with atrial fibrillation being the most common arrhythmia in clinical practice while MAT is rare. MAT occurs when there are various sites within the atria which have increased automaticity and thus compete to serve as controlling pacemakers. This rhythm is distinguished by the presence of P waves with at least three distinct morphologies (Fig 12.1). MAT is generally seen in older adults and usually occurs in the presence of significant co-morbidity such as advanced lung disease or congestive heart failure. Wandering atrial pacemaker is the name of the rhythm when there are three or more P wave morphologies and the rate is less than 100 bpm.

Figure 12.1 Rhythm strip showing different P wave morphologies found in wandering atrial pacemaker (rate <100 bpm) and multifocal atrial tachycardia (rate ≥100 bpm).

Atrial fibrillation can be classified as: (a) recent onset (<48 hours); (b) paroxysmal; (c) persistent (>1 week but potentially subject to cardioversion); or (d) permanent (resistant to cardioversion). Recurrent atrial fibrillation occurs when a patient develops two or more episodes of the disorder, which could be paroxysmal or persistent in nature. Paroxysmal atrial fibrillation is diagnosed if the episodes stop spontaneously within 7 days, but is regarded as persistent if electrical or pharmacologic cardioversion is needed to stop the arrhythmia. Permanent atrial fibrillation occurs when the patient remains in the arrhythmia as cardioversion is either not successful or deemed inappropriate. Sustained atrial fibrillation causes changes in the electrophysiology and structural properties of the atrium (a process known as atrial remodeling) that makes the atrium more susceptible to the initiation and maintenance of atrial fibrillation (atrial fibrillation begets atrial fibrillation).

Atrial fibrillation is generally a disease of aging. The prevalence of atrial fibrillation is less than 0.5% in young adults but increases to approximately 10% in individuals older than 80 years. Generally accepted causes of atrial fibrillation in young adults include hyperthyroidism, valvular heart disease, cardiomyopathy, electrocution, acute myocardial infarction, acute pericarditis, acute myocarditis, acute pulmonary embolus, cardiac trauma, amyloidosis, excessive alcohol consumption, and pheochromocytoma. Atrial fibrillation caused by vagal stimulation (e.g. vomiting) and familial atrial fibrillation have been described. Lone atrial fibrillation is defined by the presence of atrial fibrillation with a structurally normal heart and no evidence of other precipitating cause. Lastly, other potential causes such as sleep apnea, tall stature and obesity have also been proposed. In this individual, excessive alcohol consumption was thought to trigger the arrhythmia.

Holiday heart syndrome is defined as an acute cardiac rhythm and/or conduction disturbance, most commonly supraventricular tachyarrhythmia, associated with heavy alcohol consumption in a person without other clinical evidence of heart disease. Typically, this resolves rapidly with spontaneous recovery during subsequent abstinence from alcohol use. Atrial fibrillation is the most common arrhythmia observed with holiday heart syndrome but atrial flutter, junctional tachycardia, increased
ectopics and other arrhythmias have been reported. The etiology of the holiday heart syndrome is controversial but alcohol has been shown to possess direct proarrhythmic properties, especially in susceptible individuals. The frequency of cardiac arrhythmias that can be attributed to alcohol use is unclear but one estimate is that approximately 5% of all new episodes of atrial fibrillation are explainable by alcohol use.

Atrial fibrillation in young individuals due to excessive alcohol consumption is generally well tolerated and self-limiting. There are, however, case reports of sudden death. It is worth noting that in the presence of pre-excitation (an accessory path-

Figure 12.2 Atrial fibrillation in an individual with an accessory bypass tract. ECG after cardioversion shows pre-excitation (Wolff–Parkinson–White Syndrome) with a short PR interval and delta waves.
way), atrial fibrillation can be a fatal arrhythmia (see Chapter 18 for more details). The lack of the normal rate control mechanism provided by the AV node can result in 1:1 transmission from the atria to the ventricles with resulting ventricular rates greater than 300 bpm (Fig. 12.2).

One special situation that can occur on the ECG of a patient with atrial fibrillation deserves mention. Ashman’s phenomenon is an aberrantly conducted supraventricular beat that occurs in atrial fibrillation (marked with an arrow in Fig. 12.3). This phenomenon is due to the relationship of cycle length and cardiac repolarization and can be recognized on a rhythm strip by noting a wide complex beat occurring after a short RR interval that follows immediately after a much longer RR interval. This phenomenon arises because the rate of repolarization of the cardiac conduction system is dependent upon heart rate. Repolarization is relatively slow when the heart rate is slow but increases as the heart rate increases. Right bundle branch block morphology is common in Ashman’s phenomenon because the right bundle branch is slower to repolarize than the left bundle branch. Ashman’s phenomenon generally occurs in patients with atrial fibrillation when the heart rate is relatively constant and well controlled.

Further reading
A 34-year-old female who is 26 weeks pregnant is referred to you by her obstetrician for evaluation of “palpitations.” What clues does the ECG provide in establishing a differential diagnosis?
Atrial tachycardia

This ECG shows a regular, narrow complex tachycardia at a rate of approximately 150 bpm. The differential diagnosis includes atrioventricular (AV) nodal re-entry tachycardia, atrial tachycardia, sinus tachycardia, atrial flutter with 2:1 block and AV reciprocating tachycardia with retrograde conduction through an accessory pathway. Induction of transient AV block via vagal stimulation (e.g. carotid massage) or adenosine administration is generally helpful in the diagnosis. AV nodal re-entry tachycardia and AV reciprocating tachycardia will generally be terminated by AV block. In atrial tachycardia or atrial flutter, the tachycardia will not be interrupted since the ectopic focus is confined to the atria and does not involve the AV node but regular non-conducted P waves will appear during transient high-degree AV block. Similarly, P wave morphology and rate will not change in sinus tachycardia.

In this patient, further testing established that the diagnosis was atrial tachycardia. Atrial tachycardia is due to increased automaticity of an ectopic focus in the atrium. The rhythm is regular, the atrial rate is generally between 100 and 180 bpm, and the P wave morphology is generally abnormal (note that the P wave is inverted in lead I). There can be a gradual increase in the rate in the beginning of the episode, representing a warm-up period. The PR interval may be short, normal or prolonged but the QRS complex is similar to that seen in sinus rhythm unless there is a rate-dependent conduction delay.

Atrial tachycardia can be either transient or persistent and accounts for approximately 10% of supraventricular tachycardias. It is a clinically important rhythm because it can produce symptoms (e.g. dyspnea on exertion) and also because incessant tachycardia can produce tachycardia-induced cardiomyopathy. This patient has increased intravascular volume secondary to pregnancy, which probably contributed to her atrial tachycardia. She also had atrial tachycardia with her last pregnancy. She was treated with an antiarrhythmic drug and converted to sinus rhythm.
CHAPTER 14

Brugada Syndrome

A 45-year-old male was in a motor vehicle collision this evening. He had been slowing to a stop, was hit by the car behind him who in turn was hit by an 18-wheeler from behind. He was wearing a seatbelt and the air bag did not deploy. He had no loss of consciousness or head trauma. He denies any steering wheel impact with his chest. After he got out of his car, he experienced some substernal chest pain, deep and dull, 5/10, without radiation. After he sat down in the ambulance, the pain alleviated. He has had no previous episodes of chest pain per his report and had a stress test as part of an executive physical 3 years ago that was unremarkable. He denies nausea, vomiting or diaphoresis with this episode. The automated interpretation is reading the ECG as an acute myocardial infarction. What is the differential diagnosis in this patient based on this ECG?
Brugada syndrome

This ECG shows “coved” ST segment elevation and negative T wave in leads V1–V3. These are typical ECG features of the Brugada syndrome. This is an “electrical” disorder that predisposes to life-threatening ventricular tachyarrhythmias and sudden cardiac death. It has a characteristic ECG pattern that can be either persistent or inducible. In this case, Brugada syndrome was unrelated to the motor vehicle accident (the patient had no palpitations or loss of consciousness and the accident was not attributable to his error). Recognition of this pattern on ECG is very important, however, for at least two reasons: (a) interpretation of the ST elevation as an acute injury pattern would result in the faulty diagnosis of acute MI or cardiac contusion and (b) individuals with Brugada syndrome are at risk of sudden cardiac death and this patient should be further evaluated to see if he would benefit from primary prevention.

The Brugada syndrome was initially described in 1992 by Brugada and Brugada who reported eight patients with a history of cardiac arrest and ECG findings of right bundle branch block (RBBB) and ST segment elevation in the right precordial leads and no evidence of any structural heart disease. Prior to that, the Centers for Disease Control and Prevention had reported cases of sudden death in young immigrants from Southeast Asia, described as sudden unexplained death syndrome (SUDS), which has subsequently been shown to be closely related to, if not the same as, the Brugada syndrome.

The Brugada syndrome is a heterogeneous genetic disease characterized by abnormal electrophysiologic activity in the right ventricular epicardium. Approximately one-fourth of cases are caused by loss of function mutations in the cardiac sodium channel SCN5A (abnormalities in this gene have also been linked to long QT syndrome 3). This gene encodes the alpha-subunit of the sodium ion channel and, when abnormal, results in increased inactivation of the sodium channel with a prolonged recovery time. Brugada syndrome is transmitted within families in an autosomal dominant fashion with variable penetrance.

Three ECG patterns have been described in the Brugada syndrome, with all three types having in common ST elevation (≥2 mm at the J point) in the right precordial leads. In the “classic” Brugada ECG (type 1) the ST segment is continuously downsloping from the top of the R’ wave, is not elevated above baseline at the terminal portion and ends with an inverted T wave (such as in this patient). Types 2 and 3 have a “saddleback” ST-T wave configuration (Fig. 14.1). The ST segment descends towards the baseline with upward concavity and then rises again to an upright or biphasic T wave. Type 2 is defined by the terminal portion of the ST segment being elevated ≤1 mm whereas in type 3 the terminal portion of the ST segment is elevated <1 mm. The ECG changes can be dynamic, with the same patient manifesting all three types at various points in time.

In some patients, complete or incomplete RBBB is present. In others, high take-off ST segment elevation (accentuated J point elevation) in the right precordial leads mimics the pattern of RBBB but wide S waves in leads I, aVL, V5 and V6 typically seen in RBBB are absent (as in this case). In these cases, the R’ in V1 is thought to be due to early repolarization of the right ventricular epicardium rather than a RBBB.

The Brugada pattern on ECG can be either persistent or inducible. Medications (including sodium channel blockers, cocaine, antidepressants and antihistamines), electrolyte abnormalities and acute illnesses may elicit ST segment elevation in leads V1–V3 in susceptible patients. To aid in the diagnosis, provocation testing with selected class IC antiarrhythmic drugs (e.g. flecainide or procainamide) has been used as infusion of these drugs.
can unmask the Brugada ECG pattern in affected subjects.

Diagnosis of Brugada syndrome depends on both characteristic ECG findings (either spontaneous or inducible) and appropriate clinical findings (unexplained syncope, self-terminating polymorphic ventricular tachycardia, documented ventricular fibrillation, family history of sudden cardiac death at less than 45 years of age, Brugada ECG pattern in a family member, and/or inducibility of ventricular tachycardia by electrophysiologic study). A patient who presents with the Brugada ECG criteria but without the clinical characteristics is said to have the Brugada pattern but not the syndrome. The Brugada ECG pattern is seen much more frequently in men than in women, and there may be an increased frequency in Asians. It is important to note that criteria to establish a definitive diagnosis of Brugada syndrome remain under debate.

The most feared complication of Brugada syndrome is sudden cardiac death. Arrhythmias and sudden death in the Brugada syndrome generally occur during sleep or at rest and are commonly associated with bradycardia. Specific factors that have been reported to trigger fatal arrhythmias include fever, antiarrhythmic drugs, beta-blockers, tricyclic antidepressants, alcohol, cocaine and electrolyte imbalances such as hypokalemia, hyperkalemia, and hypercalcemia. Exercise has not been linked to sudden cardiac death. Patients with Brugada syndrome tend to present later in life than some other forms of inherited arrhythmias (mean age at death = 45 years).

ECG changes resembling the Brugada pattern have been reported in various conditions including isolated right ventricular infarction, pectus excavatum, arrhythmogenic right ventricular cardiomyopathy, pulmonary embolism and hyperkalemia.

There is currently no pharmacologic treatment that is completely effective in preventing sudden cardiac death in individuals with Brugada syndrome. In contrast, implantable cardioverter defibrillators (ICD) do appear to provide protection against sudden cardiac death in this population. This patient underwent electrophysiology studies which showed that ventricular fibrillation could be induced and an ICD was implanted.

**Further reading**


This ECG is from a 19-year-old female, 5 months post partum, with a prior history of myocarditis who presented to the emergency department with chest pain and shortness of breath. These symptoms were preceded by a “few days” of fever and cough. She also describes an episode where she felt dizzy and like she was going to pass out. What is the diagnosis?
**Complete heart block**

This ECG shows complete heart block (CHB) which is also known as third-degree heart block. The ECG is notable for complete dissociation of the P waves and QRS complexes as the atria and ventricles are electrically independent of each other. The atrial rate is approximately 130 bpm while the ventricular rate is 61 bpm. The atrial rate is calculated by measuring the PP interval (note that the P wave can be hidden within the QRS or T waves). A clue to CHB is the presence of variable and random PR intervals. In this case, the PR intervals are different in nearly every beat, thus highlighting the independence of the atria and ventricles.

The atrial rate is generally determined by the sinus node although CHB can also occur with atrial arrhythmias. The ventricular rate is determined by an escape rhythm which can vary from 20 to 70 bpm. In general, the lower in the conduction system that the escape pacemaker originates from, the slower the rate and the wider the QRS complex.

Criteria for diagnosing CHB include: (a) regular P waves at a rate faster than the ventricular rate; (b) regular QRS complexes at a rate slower than the P waves; and (c) no relationship between the P waves and QRS complexes with variable and random PR intervals. Be careful not to confuse CHB with AV dissociation from other causes. For example, in accelerated idioventricular rhythm, a pacemaker focus originates from within the ventricles at a rate that is greater than the atrial rate. This results in AV dissociation but does not imply impairment in conduction through the AV node; rather, the atria and ventricles beat independently because the ventricles are going faster than the atria.

CHB in this age group can be congenital or acquired. Causes of acquired CHB include medications (e.g. digitalis preparations), Lyme disease, endocarditis, acute rheumatic fever, infiltrative diseases or acute myocardial infarction.

Echocardiography in this patient showed severe left ventricular dysfunction. A temporary pacemaker was placed and myocardial biopsies taken that showed inflammation and necrosis consistent with acute myocarditis. Following treatment, left ventricular function improved and the CHB resolved. ECG following resolution of CHB is shown in Figure 15.1.

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![Figure 15.1 ECG after resolution of CHB.](image-url)
A 31-year-old healthy female has the following ECG as part of an “executive physical.” What is the diagnosis?
Long QT syndrome

This patient has long QT syndrome (LQTS), a genetic disorder associated with polymorphous ventricular tachycardia (torsades de pointes) causing syncope and sudden cardiac death.\(^1\,2\)

The QT interval represents the time from the onset of ventricular activation to the end of ventricular repolarization and is thus measured from the beginning of the QRS complex to the end of the T wave. The QT interval should be measured over 3–5 beats in leads II, V5 and V6, with the longest value being used. The length of QT varies inversely with heart rate and thus the QT interval needs to be “corrected” (QTc) using the Bazett formula (\(QTc = \frac{QT}{RR^{0.5}};\) with all intervals in seconds). The QT interval also varies with gender and the upper limit of normal for QTc is 0.44–0.45 seconds for males and 0.46–0.47 seconds for females. A rough rule of thumb is that the QT interval should not be greater than half the RR distance when the rhythm is regular.

The QT interval measures the time needed for ventricular depolarization and repolarization but in the presence of a normal QRS duration, long QT intervals occur when there is a prolongation of repolarization. Long QT can be either acquired or genetic. Causes of acquired prolonged QT intervals include severe hypothermia, hypokalemia, severe hypocalcemia, hypothyroidism, antiarrhythmic drug classes IA and III, other medications (e.g. haloperidol, methadone, etc.), severe bradycardia, atrioventricular block, myocardial ischemia, and neurogenic causes (including organophosphorus). Previously unrecognized LTQS is present in 5–20% of patients with drug-induced torsades de pointes.

Inherited LQTS was first described by Jervell and Lange-Nielsen in 1957 when they reported a family with deafness, long QT and recurrent sudden cardiac death. That family has subsequently been shown to have a homozygous mutation of KCNQ1 (a heterozygote mutation in this gene causes LQTS1; more details below). A few years later, Romano et al and then Ward described long QT, syncope and sudden cardiac death in individuals with normal hearing. More recent work has shown that LQTS is a disease of cardiac ion channels with clinical manifestations being linked to over 300 mutations in 10 different genes. In general, LQTS is caused by a heterozygous mutation and thus inheritance is autosomal dominant with variable penetrance. Mutations in three genes comprise the vast majority of cases and are described below. Homozygous mutations such as described by Jervell and Lange-Nielsen are rare (less than 1% of all LQTS cases) and associated with autosomal recessive inheritance and congenital deafness.

LQTS1 and LQTS2 are caused by mutations in genes that cause a decrease in repolarizing K potassium ion channels (Table 16.1). LQTS1 is caused by a mutation in the gene KCNQ1, encoding the ion channel for the slow delayed rectifier potassium current. LQTS2 is caused by a mutation in the gene KCNH2 (also known as HERG),

<table>
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<th>Table 16.1 Congenital long QT syndromes</th>
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<tr>
<td><strong>Gene affected</strong></td>
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<td>Ion channel</td>
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<td>Triggers of syncope and sudden death</td>
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<td>Response of QT interval to exercise</td>
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encoding the ion channel for the rapid delayed rectifier potassium current. The mutations can cause the channels to be blocked completely, open after a delay or close prematurely, thus causing decreased potassium outward current and a longer duration of repolarization. Increased sympathetic stimulation can provoke arrhythmias in LQTS1 and LQTS2 patients. In LQTS1, exercise (especially swimming), and emotional stress can precipitate syncope and sudden cardiac death. Arrhythmic events in LQTS2 can occur at stress or rest; triggering by unexpected loud noises (e.g. alarm clock) is very suggestive of LQTS2. LQTS1 and LQTS2 account for the majority of all LQTS cases.

LQTS3 is caused by mutations of the SCN5A sodium channel gene, leading to delayed closing of the channel, a constant inward sodium current, and prolonged repolarization (abnormalities in this gene have also been linked to Brugada syndrome). LQTS3 accounts for <10% of all LQTS cases and individuals with LQTS3 tend to experience cardiac events while sleeping. LQTS3 can also be associated with bradycardia and syncopal events can be precipitated by slow heart rates in addition to rapid ventricular arrhythmias.

In patients with LQTS, the most powerful predictor of risk of arrhythmia is the length of the QTc. In an analysis of more than 600 patients with LQTS1, 2 or 3, the risk of syncope or sudden cardiac death was >70% if QTc was greater than 498 msec. Other risk factors include age (syncope and sudden death are unusual >40 years old), gender, previous syncope or sudden death and type of mutation. Genetic testing can be helpful but does not exclude the diagnosis (the individual may have an unrecognized variant).

In this ECG, the QTc is 497 msec. This suggests LQTS and should prompt an evaluation for causes of long QT. Also a family history of death at an early age should be sought (in both first-degree and distant relatives) with particular attention paid to death from drowning or death due to trauma which could have been caused by syncope (e.g. while driving). In patients with long QT interval and a clinical diagnosis of syncope or sudden cardiac death, the diagnosis of LQTS can be made. In other patients, a clinical scoring system may be useful. Genetic testing will confirm the diagnosis if the patient has a known mutation, which is the case for approximately 75% of LQTS patients at this time. Echocardiography can be used to eliminate structural cardiac disease. Family screening is indicated if LQTS is diagnosed.

References
A 28-year-old female who is a local running coach presents to the emergency department with complaints of palpitations. She was running on the treadmill earlier this morning and noted that just before she finished she felt “tingly and like my heart rate was really high.” The elevated heart rate persisted. At around noon she “got tingly and cold and started vomiting.” She reports two similar episodes in the past. Approximately 3 months ago similar symptoms occurred after she was running and lasted about 3 hours before spontaneously going away. The second episode was 1 month ago. She was running and noticed the symptoms after stopping. The episode lasted 45 minutes and resolved spontaneously. She denies use of any caffeine, herbal medications, illicit drugs or decongestants. What does the ECG show and what clues does it provide in establishing a differential diagnosis?
Ventricular tachycardia

The ECG shows a wide complex tachycardia. The patient was given adenosine, procainamide and beta-blockers. Her rate accelerated to 220 bpm and she became hypotensive and lost consciousness. She was successfully defibrillated which resulted in the following ECG.

Right ventricular outflow tract tachycardia

The ECG during the tachycardia shows a wide complex rhythm with a left bundle branch pattern (LBBB) and an inferior axis (QRS is positive in II, III and aVF). The ECG following cardioversion is normal except for a rightward axis which can be a normal variant at this age. These ECG findings are consistent with a right ventricular outflow tract (RVOT) tachycardia. Also suggestive of this diagnosis is the fact that all episodes of palpitations occurred during or immediately after exercise.

RVOT tachycardia usually presents between the ages of 20 and 40 years old and is the most common type of ventricular tachycardia in patients under 40 years of age. There are various degrees of symptoms associated with RVOT tachycardia but in general two typical presentations have been described: non-sustained, repetitive, monomorphic ventricular tachycardia and paroxysmal, exercise-induced, sustained ventricular tachycardia. The most common complaint is palpitations that are triggered by caffeine intake, stress, exercise (during or in recovery) or hormonal changes (in women).

In more than 80% of cases, the tachycardia originates from the RVOT, leading to a LBBB configuration with an inferior axis. In most of these cases, the tachycardia originates from the muscular tissue just below the pulmonic valve. Recently, there have been several reports of the tachycardia originating from other sites such as within the pulmonary artery or the left ventricular outflow tract (LVOT). LVOT tachycardia is rare (<10% of outflow tract tachycardias) and may originate from either the supravalvular or infravalvular regions of the coronary cusps. LVOT tachycardia is suggested if the ECG during VT has an earlier precordial transition, more rightward axis, taller R waves in the inferior leads and small R waves in lead V1.

Tachycardia in RVOT tachycardia is due to cAMP-mediated triggered activity (triggered activity, rather than re-entry or enhanced automaticity, is the cause of RVOT tachycardia). The tachycardia will generally terminate with adenosine administration but adenosine is not 100% effective, as
demonstrated by this case. In the electrophysiology laboratory, the tachycardia may be induced by programmed extra stimuli, by burst pacing the ventricle or atrium or by infusion of isoproterenol. RVOT tachycardia can be treated with radiofrequency (RF) ablation with an overall success rate of >80%. In about 10% of cases, a second tachycardia with a different morphology will be present and may be a cause for recurrence despite successful ablation of the clinical ventricular tachycardia. A specific genetic defect has not been identified and the disease does not appear to be familial. Prognosis of patients with RVOT tachycardia is excellent and sudden death is rare.

The diagnosis of RVOT tachycardia is one of exclusion and other causes of ventricular tachycardia must be excluded. A complete evaluation for the presence of structural heart disease must be undertaken, including an evaluation for coronary ischemia, ventricular function, valvular heart disease and congenital abnormalities. In particular, arrhythmogenic right ventricular dysplasia (ARVD) and sarcoidosis can mimic RVOT tachycardia. The ventricular tachycardia in ARVD may have LBBB with an inferior axis, similar to RVOT tachycardia (please see Chapter 31 for a more detailed discussion of ARVD).

There are various causes of ventricular tachycardia in young adults in the absence of structural heart disease (generally defined as normal coronary angiography, right heart pressures, echocardiogram and cardiac MRI). Brugada syndrome and long QT Syndrome can generally be identified by their characteristic ECG appearance when in normal sinus rhythm (please see Chapters 14 and 16 for a more detailed discussion of these disorders). In the absence of structural heart disease and with a normal ECG during sinus rhythm, idiopathic left ventricular tachycardia (ILVT), idiopathic propranolol-sensitive VT (IPVT), catecholaminergic polymorphic VT (CPVT), and outflow tract tachycardias are in the differential diagnosis.

A starting point for the diagnosis of the cause of ventricular tachycardia in the absence of structural heart disease is to examine the morphology of the tachycardia precipitating the clinical event. If the inciting clinical event is precipitated by polymorphic ventricular tachycardia, torsades de pointes or ventricular fibrillation, the differential diagnosis includes long QT syndrome, Brugada syndrome and CPVT. RVOT tachycardia, LVOT tachycardia and ILVT cause monomorphic ventricular tachycardias. The tachycardia associated with IPVT may be monomorphic or polymorphic. Most cases of ILVT are verapamil-sensitive intrafascicular tachycardias with a right bundle branch block (RBBB) and left axis configuration in 90–95% of cases (the rest have RBBB with a right axis pattern). CPVT is characterized by a uniform pattern of bidirectional polymorphic VT that can be easily and reproducibly induced during exercise, emotional stress or catecholamine infusion.

This patient had no evidence of structural heart disease after an extensive evaluation. She underwent successful catheter ablation of a focus in the RVOT and resumed her running career.
An 18-year-old male presents to the emergency department with complaints of palpitations. Upon questioning, he reports using cocaine at the onset of symptoms. What does the ECG show?
A diagnosis of an irregular wide complex tachycardia is made. Because of worries about hemodynamic deterioration with pharmacologic treatment, the patient is electrically cardioverted. The ECG after cardioversion is shown below.
Atrial fibrillation with Wolff–Parkinson–White syndrome

The initial ECG shows an irregular wide complex tachycardia. The degree of irregularity is consistent with atrial fibrillation and the wide complex nature suggests aberrant atrioventricular (AV) conduction. The ECG following cardioversion shows a pattern of pre-excitation which means that electrical impulses from the atrium to the ventricle are being conducted via one or more accessory pathways rather than via the AV node. An accessory pathway (also known as a bypass tract) is a thin filamentous structure that has conductive properties and thus enables the electrical impulse to go from the atria to the ventricles without passing through the AV node. While numerous locations of accessory pathways have been reported including atriofascicular, fasciculoventricular, intranodal, and nodoventricular, the most common bypass tract is a direct connection between the atria and ventricles and is known as a Kent bundle. Accessory pathways, in general, do not have decremental conduction (defined as rate-dependent prolongation of conduction time) and thus do not have the normal safety mechanisms of the AV node in controlling ventricular rates.

ECG findings of pre-excitation include a short PR interval (≤0.12 sec; because conduction down the accessory pathway is faster than through the AV node; Fig. 18.1), widening of the QRS complex (>0.12 s) and delta waves (slurred, slow rising onset of QRS seen best in the inferior leads and in leads V1–V4 in this case). Less commonly, repolarization abnormalities (e.g. T wave inversions) will also be present. The PR interval, the time required for the atria to depolarize and the wave of depolarization to arrive at the ventricles, is shortened in pre-excitation syndromes because the accessory pathway permits the impulse to bypass the AV node. A portion of the ventricles (depending on where the accessory pathway inserts) activates first, leading to an initial slurring or “delta wave” at the start of the QRS complex. Conduction also occurs via the AV node and thus simultaneous activation of both ventricles can occur via the His–Purkinje system. Thus the size of the delta wave and the QRS duration is a function of where in the ventricles the accessory pathway inserts and the amount of relative conduction between the accessory pathway and the AV node.

Accessory pathways are congenital in origin and result from a failure of complete separation of the atria and ventricles. Approximately 0.1–0.3% of the general population have ECG findings of pre-excitation. The incidence of associated congenital abnormalities (e.g. Ebstein’s anomaly, atrial septal defect, ventricular septal defect, tricuspid atresia, aortic coarctation) ranges from 7% to 20%. In around 10% of cases multiple accessory pathways exist.

Subjects with ventricular pre-excitation on the ECG but who are asymptomatic and have no clinical arrhythmias are usually described as having “ventricular pre-excitation” or “a Wolff–Parkinson–White (WPW) ECG pattern.” When arrhythmias are present, the disorder is called the WPW syndrome. A concealed pathway is said to be present if the accessory pathway only conducts retrograde. In this case, the resting ECG will be normal in sinus rhythm with no evidence of pre-excitation.

The most frequently encountered arrhythmias in WPW (in order of occurrence) are: (a) orthodromic atrioventricular reciprocating tachycardia (AVRT); (b) atrial fibrillation; and (c) antidromic AVRT. The AVRT arrhythmias are due to a re-entry circuit that involves the AV node and the accessory pathway(s) and are generally triggered by an atrial ectopic beat. Orthodromic AVRT involves antegrade conduction down the AV node (with retrograde conduction via the accessory pathway) and results in a narrow complex tachycardia since the ventricles are activated via the normal conduction system (the delta wave will disappear during this arrhythmia). It can be difficult to definitively diagnose orthodromic AVRT during an episode of narrow complex tachycardia but clues include a rate that is usually between 140 and 250 bpm and inverted P waves within the ST segment indicating that atrial depolarization is occurring after ventricular
depolarization. Orthodromic AVRTs account for most tachycardias in WPW syndrome. Antidromic tachycardias occur when the ventricles are activated via the accessory pathway (i.e. antegrade conduction in the accessory pathway and retrograde conduction in the AV node). This results in a wide QRS reflecting abnormal ventricular activation (Fig. 18.2).

For reasons that are not clear, atrial fibrillation is more common in WPW syndrome than in the normal population. In atrial fibrillation, activation of the ventricles is predominantly via the accessory pathway which causes an irregular wide complex tachycardia, as observed in this case. Atrial fibrillation can be life-threatening if the accessory pathway has a short anterograde refractory period,

Figure 18.2 ECG in a 38-year-old male with WPW during an antidromic tachycardia and after conversion to sinus rhythm.
allowing rapid conduction of the atrial impulses to
the ventricle. This will result in very high ventricu-
lar rates with possible deterioration into ventricular
fibrillation and sudden death.

The location of the accessory pathway can be
approximated based on the 12-lead ECG. In type A,
the QRS complex is positive in V1 and V2 and
this correlates with a left-sided accessory pathway.
(a posteroseptal location characterized by a leftward axis and a lateral pathway characterized by an inferior axis). In the more common type B, the QRS complex is negative in V1 and V2. This correlates with a right-sided pathway (posteroseptal and lateral pathways have a leftward axis while an anteroseptal pathway is negative in both V1 and V2 with a normal axis). Type A is occasionally confused with a right bundle branch block (RBBB), right ventricular hypertrophy (RVH) or an inferior myocardial infarction (MI) while type B can be confused with left bundle branch block (LBBB) or anterior MI. This patient had a type A pathway.

If a patient is symptomatic, treatment can be either pharmacologic or by catheter ablation. Elimination of an accessory pathway by catheter ablation can be curative and result in normalization of the ECG (Fig. 18.3). Treatment of asymptomatic individuals to prevent sudden cardiac death is controversial and the reader is directed to the extensive literature on this subject for a more complete discussion.

There is a rare syndrome, Lown–Ganong–Levine (LGL), which is characterized by a short PR but a normal QRS. In this syndrome, there is an accessory pathway (James fibers) that connects directly with the His bundle. A note of caution is that not all patients with short PR intervals and normal QRS complexes have LGL as there are other causes of this combination.

**Further reading**


SECTION B

Congenital Heart Disease, Pulmonary Hypertension and Valvular Heart Disease
You are seeing a 24-year-old male for a routine pre-employment physical. His only complaint is mild dyspnea on exertion. The following ECG is obtained. What is the diagnosis?
Ostium secundum atrial septal defect

This ECG is from a patient with a secundum atrial septal defect (ASD), a congenital defect characterized by an opening in the septum between the two atria at the fossa ovalis and ostium secundum. This defect usually arises because the septum secundum cannot cover a large ostium secundum due to increased resorption; it can also be caused by inadequate formation of the septum secundum.

ASDs occur primarily in three locations: ostium secundum, ostium primum, and sinus venosus. The ostium secundum is the most common form of ASD and is a true defect of the atrial septum involving the region of the fossa ovalis. The most common cardiac anomaly associated with a secundum ASD is a persistent left superior vena cava (SVC) draining into the coronary sinus. Ostium primum defects are more extensive (also known as AV canal defects or endocardial cushion defects) and usually involve the tricuspid and mitral valves. The complete form of this defect includes a ventricular septal defect and a common atrioventricular valve. Ostium primum defects are present in approximately 15% of patients with Down syndrome. Sinus venosus defects are usually located at the junction of the right atrium and superior vena cava and are commonly associated with partial anomalous pulmonary venous return. Rarely, sinus venosus defects can involve the inferior vena cava. Another rare type of ASD involves a connection between the coronary sinus and the left atrium (Fig. 19.1).

The most characteristic ECG finding in patients with an ostium secundum ASD is an rSR’ or rSr’ pattern in lead V1 with a QRS duration of less than 110 msec. This finding is observed in approximately 60% of patients with an ASD. Complete RBBB is found in 5–19% of ASD cases, but less prevalently in younger patients such as this one. The mean QRS axis is usually greater than 100°, and almost always between 0° and 180° in secundum type ASD. Voltage evidence of right ventricular hypertrophy may be seen in ASDs and becomes more pronounced as pulmonary hypertension progresses. In most patients, P wave morphology is normal although right atrial enlargement can be present.

Frontal plane QRS axis is the most important indicator for differentiation between primum and secundum type ASD. Most patients with primum type ASD have left axis deviation beyond −30 degrees. Right axis deviation between −90 and −180 degrees is so rare in ostium primum ASD as to call the diagnosis into question. First-degree heart block suggests a primum ASD but may be seen in older patients with a secundum ASD.

The rhythm in patients with ASD is usually sinus although atrial arrhythmias can also occur. An ectopic atrial rhythm with inverted P waves in the inferior leads suggests sinus node deficiency, as may be seen in a sinus venousus defect.

Common physical exam findings in patients with ostium secundum ASD include fixed split S2 and a systolic murmur heard best at the left upper sternal border. This murmur reflects increased flow across the pulmonary valve. There is no murmur associated with flow across an ostium secundum ASD. A loud P2 is occasionally present and suggests pulmonary hypertension.

This ECG shows a pronounced R wave in lead V1, marked right axis deviation, and evidence of RVH. These findings suggest right heart pathology and warrant further investigation. Echocardiography would be helpful in establishing a diagnosis.

Further reading


CHAPTER 20

Atrial Septal Defect – Primum

A 34-year-old female with a history of “congenital heart defect” of unclear nature comes to your office on self-referral to request an evaluation for disability. She complains of often feeling fatigued and has fluttering in her chest approximately 1–2 times per day. What clues does the ECG provide in establishing a differential diagnosis?
Ostium primum atrial septal defect

Ostium primum ASD defect occurs within the spectrum of the atrioventricular (AV) septal defects (also known as endocardial cushion defects). Patients with ostium primum ASDs commonly also have mitral valve involvement and in the most severe form of AV septal defects can have a common AV valve and a ventricular septal defect. Approximately 10–15% of ASDs are ostium primum and it can be associated with a secundum ASD and/or a persistent left superior vena cava that empties into the coronary sinus. It is commonly associated with Down syndrome (trisomy 21) although it can occur independently of this genetic disorder.

ASDs account for approximately one-third of the cases of congenital heart disease detected in adults and occur two to three times as often in women as in men. The formation of the atrial septum is a complex process, consisting of the growth and partial reabsorption of the septum primum and septum secundum followed by the fusion of these membranes to form the endocardial cushion and then reabsorption of the fetal sinus venosus to form the right atrium. The different ASDs result from abnormalities in this process: ostium primum defects result from a deficiency in endocardial cushion formation, ostium secundum defects result from inadequate formation of the septum secundum or excessive resorption of the septum and sinus venosus defects result from problems in the incorporation of the sinus venosus chamber into the right atrium.

ECG findings associated with an ostium primum ASD are similar to those seen with an ostium secundum ASD with the major exception being left axis deviation. An rSr’ or rsR’ pattern in lead V1 is common. In addition, there may be first-degree AV block. Since most ostium primum ASDs are relatively large (causing significant left-to-right shunts) and associated with mitral regurgitation because of abnormalities in the mitral valve, right ventricular hypertrophy and/or right atrial abnormalities are common. The incidence of atrial arrhythmias increases with age and becomes common after 30 years.

This patient underwent successful surgery with a pericardial patch repair of the ostium primum ASD, repair of a cleft anterior leaflet of the mitral valve and closure of an ostium secundum ASD.

Please see the description accompanying the patient with an ostium secundum ASD, in Chapter 19, for further details on ASDs.

Further reading

A vascular surgeon asks your opinion on an abnormal ECG in a 39-year-old patient with end-stage renal disease. The patient has recently moved to the area and was referred to the surgeon for revision of dialysis access. The ECG was done as a routine preoperative assessment. The patient has no cardiac complaints.
Dextrocardia

Dextrocardia (from Latin dexter-, on the right-hand side, and Greek kardia-, heart) is a rare condition in which the heart is situated on the right side of the chest. There are various clinical scenarios that can lead to dextrocardia including: (a) the heart is shifted to the right side of the chest by factors extrinsic to the heart (e.g. the scimitar syndrome with hypoplasia of the right lung); (b) rotational abnormalities of the cardiac loop during development (dextroversion, e.g. single ventricle or Cantrell syndrome); and (c) dextrocardia associated with situs inversus in which the abdominal organs are also transposed. About one-third of patients with situs inversus will have heart malformations such as ventricular septal defect, atrial septal defect, tetralogy of Fallot, tricuspid atresia, pulmonary stenosis, single ventricle or complete or corrected transposition of the great arteries. Kartagener syndrome consists of situs inversus, sinusitis and bronchiectasis. Approximately two-thirds of people with dextrocardia and situs inversus are well, have structurally normal hearts and live a normal life span.

The two most prominent features in an ECG of dextrocardia are the P wave axis and the morphology of the QRS complexes of the precordial leads. In the normal heart with normal sinus rhythm, the P wave axis (determined from the limb leads) varies from 0 to +75 degrees (i.e. directed inferiorly and leftward). The P wave is always upright in leads I and II. An ECG showing marked right axis deviation of the P wave (negative in aVL and lead I) and low voltage in leads V4–V6 should prompt consideration of dextrocardia. Atrial depolarization, and thus P wave axis, will be rightward in dextrocardia. In the precordial leads, there will be reverse R wave progression with the R wave being tallest in V1 and progressively decreasing in amplitude in leads V2–V6. Other ECG findings which should prompt the thought of dextrocardia include: (a) a net positive QRS vector in AVR; (b) an extreme rightward QRS axis (between −90 and −180 degrees) and (c) a tall R wave in V1.

Be aware of “pseudo-dextrocardia” in which the right arm and left arm leads are reversed (Fig. 21.1). This will lead to “rightward” P wave and QRS axes. In general, P wave and QRS that are both predominantly negative in lead I generally only occur in two conditions: reversed arm leads or dextrocardia. A clue to the diagnosis of limb lead reversal in this setting is that the precordial wave voltage will generally appear normal.

![Figure 21.1](image)
A 32-year-old female who lived in Mexico until several months ago presents to your emergency department with profound dyspnea. According to the patient, she has been treated for heart failure for the past 5 years. She has been told since she was a child that she has heart problems, but has not been given a specific diagnosis. She was admitted to another hospital several weeks ago for “pneumonia” and was treated with bilevel positive airway pressure (Bipap) and antibiotics. What clues does the ECG provide in establishing a differential diagnosis?
Patent ductus arteriosus

The ECG shows right axis deviation, right bundle branch block and right atrial enlargement. Right axis deviation in the frontal plane, large R waves in the right precordial leads, deep S waves in the left precordial leads and a mild increase in QRS duration are consistent with right ventricular hypertrophy (RVH). These findings are consistent with a lesion causing right ventricular volume or pressure overload.

In this individual, the cause was a patent ductus arteriosus (PDA). Other types of heart disease that would be in the differential diagnosis based on this ECG include congenital heart defects that result in left-to-right shunting, such as atrial septal defect or ventricular septal defect, and acquired or congenital disorders that result in right ventricular pressure overload, such as mitral stenosis, cor pulmonale, primary pulmonary hypertension, pulmonic stenosis, tetralogy of Fallot and other rare types of congenital heart disease.

In an adult with a PDA, the ECG reflects the hemodynamic effects. With a small PDA, a normal ECG is common. This ECG shows left atrial abnormality which is not uncommon in patients with a PDA with a moderate or large left-to-right shunt. Left ventricular hypertrophy (with or without RVH) can also be seen in a PDA with a moderate shunt. RVH on ECG generally indicates a large shunt with the development of pulmonary hypertension.

The ductus arteriosus usually originates just distal to the left subclavian artery and connects the descending aorta to the left pulmonary artery. In the fetus, blood flow via the ductus arteriosus is from the pulmonary artery to the aorta, thus enabling blood to bypass the unexpanded lungs and enter the descending aorta for oxygenation in the placenta. At birth there is a decline of prostaglandin with the increase in PaO₂ and the ductus arteriosus usually closes within a few days. If closure does not occur, blood can flow from the aorta to the pulmonary artery, leading to increased pressure and volume in the pulmonary circulation. Symptoms depend on the size of the shunt, with large shunts having the possibility of leading to Eisenmenger syndrome.

In this patient, cardiac catheterization demonstrated a PDA with a calculated shunt fraction (Qp/Qs) of 2.5. Cardiac output (Qs) as measured using the assumed Fick method was 9.1 L/min and pulmonary vascular resistance was 6.3 Wood units. An attempt was made to close the PDA percutaneously but it was too large for any existing closure device and the patient subsequently underwent successful surgical closure.

Further reading


A 26-year-old female is arrested and incarcerated. While in custody, she complains of shortness of breath. An ECG is done which prompts a referral to you for evaluation. On physical examination, cyanosis is apparent. She has not seen a physician in over 10 years. What clues does the ECG provide in establishing a differential diagnosis?
Tetralogy of Fallot

This ECG is distinctly abnormal for a 26-year-old female. It shows right axis deviation, right ventricular hypertrophy (RVH) and right atrial (RA) enlargement. The diffuse ST and T wave abnormalities (especially those in the right precordial leads) are most likely due to right ventricular “strain” (i.e. RVH with strain). These findings are all highly suggestive of increased RV and RA pressures. The differential diagnosis in a patient of this age would include disorders causing pulmonary hypertension (e.g. mitral stenosis, primary pulmonary hypertension, an atrial septal defect with pulmonary hypertension), and disorders causing RV pressure or volume overload (e.g. pulmonic stenosis, ventricular septal defect). The next step in evaluation of this patient would be a careful physical exam and an echocardiogram.

This patient had uncorrected tetralogy of Fallot. She had been diagnosed at birth and her family was told that she would need surgery but she had not sought medical care until she was incarcerated. The natural history of tetralogy of Fallot, if uncorrected, is progressive right heart failure and hypoxemia and it is unusual for a patient to survive for 26 years. This patient’s O₂ saturation while breathing room air was in the 60s. She died during an attempt at corrective surgery.

Tetralogy of Fallot is characterized by a large ventricular septal defect, an “over-riding” aorta, RV outflow tract obstruction (that is generally sub-valvular but may also be valvular, supravalvular or in the pulmonary arterial branches), and RVH. Most adults with uncorrected tetralogy of Fallot have substantial right-to-left shunting and it is the most common cyanotic congenital heart defect after infancy. The rate of survival without surgery is dismal, with only 3% alive at 40 years. ECGs in adults with uncorrected tetralogy of Fallot are almost always abnormal, with the most common abnormalities being RA enlargement, right axis deviation and RVH.

Further reading


A 38-year-old female is referred to you for evaluation of complaints of exertional dyspnea and fatigue. She has a history of a congenitally corrected transposition of great vessels with a ventricular septal defect that was diagnosed as an infant. She never had reparative cardiac surgery and has not seen a physician in many years. She was in her usual state of health until several months ago when she began to have dyspnea on exertion. Several weeks ago she had sudden worsening in her symptoms to the point where she was unable to work or care for her two children. At the same time she began to experience palpitations and an irregular heart beat. What does the ECG show and what clues does it provide in establishing a differential diagnosis?
Complete heart block +
hemodynamic changes associated
with a ventricular septal defect

The ECG is abnormal because of complete heart block (CHB) with a junctional escape rhythm, left atrial enlargement, voltage criteria for left ventricular hypertrophy (≥20 mm in II and III and aVF) and right axis deviation.

CHB (also known as third-degree heart block) is indicated by the complete dissociation of the P waves and QRS complexes. The atrial rate is approximately 70 bpm while the ventricular rate is 40 bpm. The atrial rate is calculated by measuring the PP interval; note that the P waves can be hidden within the QRS or T waves but in this ECG are well seen in lead II. A clue to CHB is the presence of variable and random PR intervals. Criteria for diagnosing CHB include: (a) regular P waves at a rate faster than the ventricular rate; (b) regular QRS complexes at a rate slower than the P waves; and (c) no relationship between the P waves and QRS complexes with variable and random PR intervals.

In an individual with CHB, the ventricular rate is determined by an escape rhythm which can vary from 20 to 70 bpm. In general, the lower in the conduction system the escape pacemaker originates from, the wider the QRS complex and the slower the rate will be. The escape rhythm in this case is probably within the AV node or His bundle as the QRS is not excessively wide (please see Chapter 15 for a more detailed discussion of CHB).

Ventricular septal defects (VSD) are a common congenital heart defect and account for approximately 10% of congenital heart disease diagnosed in adults. They vary greatly in location, clinical presentation, associated lesions, and natural history. They can be associated with other congenital anomalies including atrial septal defect, patent ductus arteriosus, right aortic arch, and pulmonary stenosis. Multiple VSDs occur but usually in association with complex congenital heart disease. In young adults, concomitant aortic insufficiency may be present in patients with VSDs and indicates a worse prognosis.

Ventricular septal defects are surrounded by fibrous tissue. Defects that involve the membranous septum and extend into the muscular portion of the septum are called perimembranous. The trabecular septum is the largest part of the interventricular septum. Muscular VSDs within the trabecular septum can undergo spontaneous closure as a result of muscular occlusion. The inlet portion of the septum begins at the level of the atrioventricular (AV) valves and defects in the inlet septum can include abnormalities of the tricuspid and mitral valves (e.g. AV canal defects). The infundibular septum separates the right and left ventricular outflow tracts. The majority of VSDs in adults are perimembranous.

ECG changes in a patient with a VSD are a result of the hemodynamic effect of the defect which in turn is influenced by the size of the VSD, the pressures in the right and left ventricles, and the pulmonary resistance. The shunt is initially left to right with volume being determined largely by the size of the defect and the pulmonary vascular resistance. In general, small VSDs have no discernible effect on the ECG. In a study of 221 adults with a VSD diagnosed in childhood that did not require closure, 215 had a normal QRS axis (six had left axis deviation). Incomplete right bundle branch block was present in 23 patients, complete right bundle branch block was present in two patients, and complete left bundle branch block in one patient. No first-degree or higher-degree heart block was found in any patient.

In patients with larger defects, left atrial abnormalities and left ventricular hypertrophy can develop. Pulmonary resistance will increase over time, leading to increased right ventricular pressures. This can lead to the development of right ventricular hypertrophy, right axis deviation and right atrial abnormalities on ECG. If pulmonary artery pressures increase to the level of systemic pressures, Eisenmenger syndrome will develop with right-to-left shunting. The ECG diagnosis of biventricular hypertrophy can be difficult but right axis deviation in the presence of LVH is suggestive of RVH + LVH. Combined LV + RVH should raise the possibility of a large VSD or patent ductus arteriosus.

Patients with VSD have a high incidence of ventricular arrhythmias including ventricular couplets,
multiform ventricular ectopies, and ventricular tachycardia. The incidence of ventricular arrhythmias increases with increasing pulmonary pressures. CHB is uncommon in patients with VSD although it can be a complication of surgical repair.

This patient had a large VSD with predominant left-to-right shunting \((Qp/Qs = 3.6)\) but also with some right-to-left shunting. Left and right ventricular pressures were the same. There was also a right ventricular outflow tract obstruction with a pressure gradient of approximately 60 mmHg. Left and right ventricular systolic function were normal. The patient underwent surgical repair of the ventricular septal defect with resection of the subpulmonic stenosis, and placement of permanent epicardial pacemaker leads.

**Further reading**


A 55-year-old male is referred to you for follow-up care after being recently hospitalized. He had no significant past medical history before presenting to the emergency department 2 weeks ago with a 3-day history of cough productive of rusty-colored sputum and shortness of breath. Chest x-ray showed bilateral infiltrates with small bilateral pleural effusions. The patient was treated with ceftriaxone and azithromycin with some symptomatic improvement. His main complaint at this point is persistent dyspnea on exertion. What clues does the ECG provide in establishing a differential diagnosis?
Mitral stenosis

This ECG shows evidence of left atrial (LA) enlargement, right axis deviation, right ventricular hypertrophy (RVH) and P-mitrale (lead II). Mitral stenosis (MS) can be suspected based on ECG findings and is strongly suggested, in the appropriate patient population, by the presence of LA enlargement and RVH in the absence of left ventricular dysfunction.

The cross-sectional area of the mitral valve is 4–6 cm² in healthy adults. MS occurs when this area decreases, resulting in increased resistance to flow from the LA to the left ventricle and increased LA pressure. The increased LA pressure is transmitted to the pulmonary circulation, resulting in elevated right heart pressures. MS is a progressive disorder with symptoms worsening as mitral valve area decreases. Symptoms do not usually appear until the mitral valve area <2 cm². Initially symptoms occur only with exertion, emotional stress or pregnancy and consist primarily of dyspnea. Symptoms can progress over time as the valve opening declines further and in severe cases will eventually include dyspnea at rest, orthopnea, and paroxysymal nocturnal dyspnea. More rarely, there may be angina, palpitations, recumbent cough and/or hemoptysis.

Almost all cases of MS are rheumatic in origin. Isolated MS occurs in 40% of all patients presenting with rheumatic heart disease and women with MS outnumber men by approximately 2:1. Rheumatic mitral valve changes including thickening of the mitral valve leaflets, calcification and commissural fusion occur over the course of decades. Other rare causes of MS include systemic lupus erythematous, rheumatoid arthritis, carcinoid, mucopolysaccharidosis, mitral annular calcification and congenital valve deformity. Conditions that cause increased LA pressure can mimic MS. Examples include LA myxoma, pulmonary vein obstruction, and cor triatriatum (a thin membrane across the left atrium which obstructs pulmonary venous inflow).

ECG findings suggestive of MS include LA abnormalities and evidence of right ventricular pressure overload. P-mitrale is defined as a widened P wave (≥0.12 seconds) with normal or slightly increased voltage that is notched, bifid or flat-topped (seen in leads II, III and aVF in this patient). It is a sign of long-standing mitral valve disease. Atrial fibrillation is common in patients with severe MS. While ECG findings can be suggestive of MS, it is important to note that the ECG is neither a sensitive nor specific technique for diagnosing MS.

This patient had severe mitral stenosis with a mean transmirtal pressure gradient of 25 mmHg. The calculated mitral valve area was 0.7 cm². He had pulmonary hypertension with pulmonary artery pressures of 68/30 mmHg.

Further reading


A 31-year-old male with a history of ankylosing spondylitis is brought to the emergency department by a friend. He reports 1 week of low-grade fevers (100.5 degrees), cough, progressive shortness of breath, nausea, diarrhea, and occasional vomiting. He has noticed a rapid decline in his exercise tolerance in the past week, and he states he can’t walk more than 10–20 feet without getting very short of breath. He also complains of a frothy cough sometimes laced with blood, paroxysmal nocturnal dyspnea and orthopnea. He denies any chest pain. What clues does the ECG provide in establishing a differential diagnosis?
Aortic insufficiency

This ECG is distinctly abnormal for a 31-year-old and provides a strong clue that there is underlying, chronic cardiovascular pathology. The major findings are left atrial enlargement and left ventricular hypertrophy (LVH) with a "strain" pattern.

Electrical forces generated during left ventricular activation produce the normal QRS complex. With an increase in the amount of left ventricular myocardium (left ventricular hypertrophy), electrical preponderance of left ventricle over right ventricle is further accentuated. The mean vector of the left ventricle becomes more posterior and leftward, increasing QRS complex voltage and ventricular activation time. Additional ECG clues to the diagnosis of left ventricular hypertrophy are left axis deviation, increased QRS duration and the presence of left atrial abnormality (LAA).

There are dozens of published criteria that can be used to diagnose LVH on an ECG, although none is universally accepted. Some of these criteria are based solely on voltage present on the ECG (e.g. the Sokolow–Lyon Voltage Criteria and the Cornell Voltage Criteria) whereas others also take into account related findings such as left axis deviation, LAA and ST-T wave abnormalities (e.g. the Estes Criteria). A detailed discussion of the specific formulas is beyond the scope of this case but a few generalities are useful.

- The ECG remains a useful, although far from perfect tool to identify ventricular hypertrophy.
- The specificity of the formulas tends to be high whereas the sensitivity (when compared to echocardiography) is much lower.
- The sensitivity and specificity of the ECG criteria vary with age, gender and obesity. Modified formulas that "correct" for these variables have been developed for some of the specific criteria (e.g. Cornell Criteria).
- ECG diagnosis of LVH in individuals less than 40 years of age is further limited because young people often have high-amplitude QRS complexes in the absence of left ventricular disease.

On voltage criteria alone, there is general agreement that LVH may be diagnosed if any of the following criteria are present in the precordial leads:

- R wave in V1 exceeding 24 mm
- R wave in leads V4, V5 or V6 >26 mm
- R wave in leads V5 or V6 plus S wave in lead V1 >35 mm.

The diagnostic accuracy in determining LVH by ECG is improved if various non-voltage criteria are present in patients with increased voltage. These include the presence of ST depression and T wave inversion (ventricular strain), left axis deviation, increased QRS duration and/or left atrial abnormality. Ventricular strain is characterized by a downsloping convex ST segment with an inverted asymmetric T wave that is opposite in direction to the predominant QRS vector axis in V5 and V6. In general, the proximal descending limb of the inverted T wave has a slow descent, and the ascending limb rises steeply. The ST segment and T wave deviation in the direction opposite to the predominant QRS vector leads to a widening of the QRS/T angle (i.e. the angle between the directions of ventricular depolarization and repolarization).

LAA is suggested by notching or prominence of the terminal portion of the P wave in the limb leads, prominent negativity of the terminal portion of the P wave in V1 or a P wave duration of >120 msec. Normal P waves may be bifid in the limb leads with a minor notch probably resulting from slight asynchrony between right and left atrial depolarization. However, prolongation of the P wave and/or a pronounced notch with a peak-to-peak interval of >0.04 s suggests LAA. In V1, the P wave is often biphasic. This is because early right atrial forces are directed anteriorly, giving rise to an initial positive deflection whereas left atrial forces travel posteriorly, producing a later negative deflection. A large negative deflection (>1 small square in area) suggests LAA (please see Chapter 8 for a more detailed discussion of LAA).

Conditions that can cause LVH in this age group include hypertrophic cardiomyopathy and diseases that increase ventricular afterload. Causes of increased afterload include aortic stenosis, aortic insufficiency (AI), severe hypertension and aortic coarctation. The athlete’s heart can also be associated with increased voltage.

AI is a condition of increased afterload with the associated hemodynamic changes varying depend-
ing on the time course of the valve dysfunction. If AI develops rapidly (i.e. acute or subacute AI), the left ventricle is unable to handle the pressure and volume overload, causing a rapid increase in left ventricular pressures during diastole, markedly elevated pressures at end diastole, and premature closure of the mitral valve. Aortic diastolic pressures may be low but generally there is a minimal increase in pulse pressure in acute AI; in very severe cases of acute AI, cardiac output may fall leading to hypotension. This leads to dilation of the left ventricle, leading in some patients to the development of a massively dilated left ventricle termed cor bovinum.

In this patient, an echocardiogram showed severely decreased left ventricular contraction with left atrial dilation. The aortic valve was trileaflet with inadequate coaptation and severe aortic regurgitation. Right ventricular contractile performance was decreased. Hemodynamic changes of severe aortic insufficiency were found at cardiac catheterization, including elevated left ventricular end-diastolic pressure (38 mmHg), equalization of aortic and left ventricular pressures during diastole and premature closure of the mitral valve during diastole. There was moderate pulmonary hypertension (50/33 mmHg) and reduced cardiac output (2.3 L/min by assumed Fick method). The patient underwent successful aortic valve replacement with a 25 mm St Jude prosthesis.

Ankylosing spondylitis is an autoimmune spondylarthropathy that is characterized by chronic, painful, degenerative inflammatory arthritis primarily affecting the lower spine and sacroiliac joints. The cause has not been identified although there is a strong association with the class I major histocompatibility complex antigen HLA-B27. In patients with ankylosing spondylitis the incidence of AI is approximately 10%.

Further reading
A 49-year-old female who works in the hospital as a Certified Nursing Assistant is brought to the emergency department by co-workers because of an episode of near-syncope at work. She describes the sudden onset of feeling cold and clammy while standing. Her blood pressure at the time was reported to be 70/40 mmHg. Her pulse rate was not taken by her co-workers but they felt that it was not overly fast. The presyncopal symptoms resolved slowly once she was laid in a supine position. She denies any chest pain, shortness of breath, palpitations or any prior similar episodes. She has no significant past medical history but on questioning reports that she has had a cardiac murmur all of her life. What clues does the ECG provide in establishing a differential diagnosis?
Pulmonic stenosis

The major findings on this ECG are right axis deviation, an rSr' pattern in V1 and an incomplete right bundle branch block. These findings are highly suggestive of right ventricular hypertrophy (RVH). R wave voltage greater than S wave voltage in V1 is common in children but found in less than 1% of a normal adult population. The likelihood of RVH with R > S in V1 is further increased in the presence of right axis deviation. Note also that the P waves in leads III and aVF are biphasic (the P wave axis is normal). This is not reported as being associated with (much less diagnostic of) right atrial abnormality but is abnormal and suggestive of atrial pathology. The ECG findings are suggestive of right heart pressure overload (remember that ECG findings are rarely diagnostic of congenital or valvular heart disease but rather reflect the hemodynamic changes). The emergency physician requested an echocardiogram that showed pulmonic valvular stenosis.

Congenital pulmonic stenosis can be divided into three types based on the morphology of the valve. The most common type is a “dome-shaped” pulmonic valve characterized by impaired opening of a preserved valve. Less common are the dysplastic pulmonary valve (myxomatous, poorly mobile valve without commissural fusion) and the unicuspid or bicuspid pulmonic valve (generally seen with tetralogy of Fallot).

The natural history of pulmonic stenosis is variable but in general many patients remain asymptomatic for years and survival into adulthood is the norm. The First Natural History Study of Congenital Heart Defects (NHS-1) enrolled 592 patients between 1958 and 1969, mostly children, with pulmonic stenosis. NHS-2 studied these patients >15 years later and found that the probability of 25-year survival was 95.7%, with 97% of these patients being New York Heart Association class I. In patients with symptoms, complaints generally include decreased exercise tolerance, dyspnea on exertion, fatigue, chest pain, and/or palpitations.

The severity of pulmonic stenosis is generally graded based on the pressure difference between right ventricular and pulmonary artery systolic pressures. In NHS-2, there was no progression in patients with initial peak gradients <25 mmHg whereas the vast majority of patients with gradients >50 mmHg required invasive treatment (either surgery or balloon valvuloplasty). The course of the disease was less predictable in patients with initial gradients between 25 and 49 mmHg as 20% of these individuals required an intervention at some point.

The ECG findings in severe pulmonic stenosis can be predicted by the hemodynamic effects and include RVH, right axis deviation and right atrial abnormalities. RV pressures increase in order to maintain pulmonary artery pressures in face of a stenotic valve. When RV pressures reach a threshold (postulated to be around 60 mmHg in systolic...
pressure but with significant interpatient variability), ECG changes become apparent. In patients with pulmonic valve stenosis and an incomplete RBBB, the R' voltage has been reported to correlate with the degree of stenosis.

In this patient, invasive evaluation showed a mean pressure gradient of 16 mmHg and a peak-to-peak gradient of 25 mmHg between the right ventricle and pulmonary artery. Right ventricular systolic pressure was 55 mmHg and end-diastolic pressure was 16 mmHg. An angiogram of the right ventricle revealed normal right ventricular function, pulmonic stenosis and significant post-stenotic dilation of the pulmonary artery (Fig. 27.1).

Further reading


You are asked to evaluate a 43-year-old female because of increasing dyspnea. During the last 2 years she has noted progressive dyspnea on exertion, progressing to the point where she can no longer walk up a flight of stairs because of significant shortness of breath. She was admitted to an outside hospital several months ago and treated for what was termed "respiratory failure." She was subsequently admitted 2 months later for similar complaints and discharged with a diagnosis of "heart failure." A month ago, she was found to be markedly hypoxemic on room air and was started on home oxygen therapy. Past medical history is notable for sarcoidosis that was diagnosed on an open lung biopsy 11 years ago. What clues does the ECG provide in establishing a differential diagnosis of causes of dyspnea?
Pulmonary hypertension

This ECG shows right atrial abnormality, right axis deviation and right ventricular hypertrophy (RVH), strongly suggesting the presence of right ventricular pressure overload. ECG evidence of right atrial abnormality includes P wave voltage in leads II, III and aVF of greater than 2.5 mm and positive voltage of the P wave in lead V1 and V2 of greater than 1.5 mm. R wave voltage greater than 5 mm in aVR is consistent with RVH. Further evidence for RVH includes R = S in V1, S wave >7 mm in V5 and V6 and right axis deviation. These ECG findings are suggestive of right heart pathology but are not specific for the cause. Right heart catheterization showed pulmonary hypertension with PA pressures 68/26 mmHg. Right atrial pressure was elevated at 13 mmHg. The cause of the pulmonary hypertension was progressive sarcoid pulmonary disease.

The normal pulmonary circulation is characterized by high blood flow, low pressure, and low resistance. The normal adult pulmonary vascular bed is highly distensible and capable of accommodating large increases in blood flow with minimal elevations of pressure. Normal peak systolic pressures in the pulmonary arteries range from 18 to 25 mmHg with a normal mean pressure of 10–16 mmHg. Most commonly, pulmonary hypertension is defined as a mean pulmonary artery pressure (MPAP) of >25 mmHg although other definitions have been proposed.

Pulmonary hypertension may occur in response to a multitude of mechanisms, divided into primary and secondary hypertensive states based on disease etiology. Primary pulmonary hypertension is a rare disease characterized by elevated pulmonary artery pressure with no apparent cause. It has been reported in patients of nearly all demographics, although two-thirds of cases are in women with typical age of onset of 36 years. The vast majority of patients with elevated pulmonary pressures have secondary pulmonary hypertension. The most common causes of secondary pulmonary hypertension include left ventricular systolic or diastolic dysfunction, valvular disease, cor pulmonale, and thromboembolism. Less common etiologies include systemic sclerosis, sarcoidosis, congenital heart defects with left-to-right shunts (e.g. atrial septal defect, ventricular septal defect or patent ductus arteriosus), hepatopulmonary hypertension, HIV infection, and pulmonary veno-occlusive disease.
SECTION C
Coronary Artery Disease, Pericarditis and Cardiomyopathies
What does the ECG show and what is the differential diagnosis?

A 36-year-old male presents to the emergency department with worsening dyspnea. He denies chest pain but reports weight gain and orthopnea.
Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is characterized by a dilated left ventricle with reduced systolic function. Symptoms of DCM indicate the severity of the disease and include shortness of breath, fatigue, dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, and/or edema. Causes of DCM in younger adults include infections (e.g. as a sequelae of myocarditis), ischemia, hypertension, toxin exposure (e.g. alcoholism, heavy metals, doxorubicin, cocaine, metamphetamine), valvular disease, human immunodeficiency virus (HIV), Chagas disease, peri-partum, collagen vascular disease, glycogen storage disease, hemochromatosis, thiamine deficiency, some forms of chemotherapy, amyloidosis, and neuromuscular disorders.1 Recently, genetic dilated cardiomyopathies have been increasingly recognized and it is now estimated that approximately 30% of idiopathic DCM cases are genetic in origin.

There are no ECG findings diagnostic of DCM but those suggestive of the diagnosis include left atrial or biatrial enlargement, intraventricular conduction delays (e.g. left fascicular block, non-specific intraventricular conduction delay or left bundle branch block), poor R wave progression across the precordium, Q waves in multiple leads, diffuse T wave abnormalities or diffuse low QRS voltage. A combination of low QRS voltage in the limb leads, low voltage in V1–V4 and prominent voltage in V5 and V6 is suggestive of DCM. Atrial and ventricular ectopic beats, atrial arrhythmias and ventricular arrhythmias are common in patients with DCM.

This ECG shows sinus tachycardia with a non-specific intraventricular conduction delay.

Reference

Further reading
A 40-year-old male without past medical history or complaints has an ECG done as part of a routine pre-employment physical. What is your interpretation of the ECG and what do you recommend to the man?
Hypertrophic cardiomyopathy

The ECG shows left ventricular hypertrophy (LVH), QRS prolongation and repolarization abnormalities. These findings are markedly abnormal for a 40 year old, especially one without any significant past medical history who is not hypertensive. The ECG is consistent with an increased left ventricular mass and thus suggestive of hypertrophic cardiomyopathy (HCM).

HCM is caused by a mutation in one of the genes encoding proteins of the cardiac sarcomere. At least 10 different genes have been linked to HCM, but three genes predominate: beta-myosin heavy chain, cardiac troponin T, and myosin-binding protein C. Familial HCM is an autosomal dominant disease and present in 50% of patients with HCM; spontaneous mutations are suspected for sporadic forms of the disease. The phenotypic expression of hypertrophic cardiomyopathy occurs in 1 of every 500 adults in the general population and is characterized by ventricular hypertrophy. Different forms of HCM include asymmetric hypertrophy of the interventricular septum (with or without an outflow tract gradient), concentric hypertrophy and apical hypertrophy. Hypertrophy can be found in any region of the left ventricle, but it is most frequently found in the septum. Extensive fibrosis of the affected walls occurs as well. Dynamic left ventricular outflow tract obstruction (which differentiates hypertrophic obstructive cardiomyopathy (HOCM) from the larger group of hypertrophic cardiomyopathy) is present in 30–50% of patients with HCM. Septal hypertrophy causes the T wave to be directed opposite to the prominent septal depolarization force, so the T wave will be positive in leads with deep Q waves. Left atrial abnormality and prolonged QTc interval can also be seen in patients with HCM. Atrial fibrillation is the most common sustained arrhythmia in HCM but atrial flutter, ventricular ectopy, ventricular tachycardia, and ventricular fibrillation can also occur. In rare cases, there can be PR prolongation or ECG findings of preexcitation (Wolff–Parkinson–White pattern).

In a study of 134 patients with hypertrophic cardiomyopathy diagnosed using echocardiographic criteria, normal ECGs were uncommon (7%) and occurred primarily in individuals who were asymptomatic and had no left ventricular outflow obstruction. Repolarization abnormalities and LVH were the most common abnormalities, occurring in 81% and 62%, respectively, of the total population. A broad spectrum of other electrocardiographic abnormalities, non-specific for hypertrophic cardiomyopathy, was also found including left atrial abnormality, left axis deviation, abnormal Q waves, right atrial abnormality and atrial fibrillation.

In a study of genetically diagnosed familial hypertrophic cardiomyopathy, the sensitivity and specificity of “major” ECG criteria (LVH or Q waves >0.04 sec in duration and/or >1/3 of the ensuing R wave in depth and present in at least two leads) or repolarization alterations with marked T wave inversion in at least two leads) were 61% and 97% respectively. If the ECG criteria were expanded to include “minor” criteria such as left atrial abnormality, left axis deviation, abnormal Q waves, right atrial abnormality and atrial fibrillation.
the sensitivity and specificity were 77% and 86% respectively. In this study the sensitivity of "major" ECG criteria increased with age (46% in 18–29 year olds, 58% in 30–49 year olds and 89% in ≥50 year olds) with a modest loss in specificity (100%, 97% and 92%, respectively).

Voltages on a 12-lead ECG are not a reliable marker for the magnitude of LVH or degree of outflow tract obstruction. In a study of 448 consecutive patients with HCM, there was a relatively weak correlation between maximum LV wall thickness and ECG voltage. In 55 patients with massive LVH (LV wall thickness ≥30 mm), only 24 (44%) showed ECG voltage ≥30 mm in any lead. Similarly, the ECG had limited usefulness in diagnosing patients with hemodynamically significant outflow tract obstruction.

Highly trained athletes can manifest a variety of ECG changes, including a striking increase of R or S wave voltage, either flat or deeply inverted T waves, and deep Q waves, that can mimic hypertrophic cardiomyopathy. The distinction between hypertrophic cardiomyopathy and athlete’s heart can be difficult and will generally require consideration of echocardiographic and clinical features in addition to the ECG.

There is a rare form of HCM, seen mainly in Japan, called apical hypertrophic cardiomyopathy. Hypertrophy of the left ventricle is limited primarily to the left ventricular apex. The presence of "giant" negative T waves, primarily in leads V3–V5, has been reported as a typical ECG finding.

In summary, there is a wide variety of patterns of ECG changes in patients with HCM including LVH, repolarization abnormalities (e.g. T wave inversion and ST segment depression with upward convexity in V4–V6), left atrial abnormality, and Q waves in inferior and lateral leads. The ECG is not diagnostic but rather serves to raise suspicion of HCM. It is important to recognize ECG patterns suggestive of HCM in various populations including individuals who have symptoms compatible with HCM, in asymptomatic individuals at high risk of sudden death or in athletes as part of pre-participation screening.

In this patient, an echocardiogram showed systolic anterior motion of the mitral valve, mild mitral regurgitation, marked concentric LVH with hyperdynamic contraction, and small end-systolic left ventricular volume. The maximal instantaneous left ventricular outflow velocity was 4.4 m/sec with a late peak, consistent with a dynamic outflow gradient of approximately 80 mmHg.

**Further reading**


You are evaluating a 26-year-old male in the emergency department who reports that while standing, he suddenly felt light-headed. According to witnesses he collapsed but had no seizure-like activity. He denies any preceding chest pain, nausea/vomiting, shortness of breath or recent illness. He has had three prior episodes of syncope – the first episode was 4 years ago and then he had two episodes one year ago. On questioning, he also reports occasional palpitations associated with "not feeling well." What clues does the ECG provide in establishing a differential diagnosis?
Arrhythmogenic right ventricular dysplasia

This ECG should make you think of arrhythmogenic right ventricular dysplasia (ARVD) especially in a young individual who presents with syncope. Important findings include T wave inversion in leads V1–V3 and a PVC that may originate from the right ventricle (typically PVCs that originate from the right ventricle have a left bundle branch block pattern; in this patient the site of origin cannot be definitively stated to be the RV as the terminal deflection of the QRS complex is negative in both leads V1 and V5).

ARVD is a disorder in which there is patchy replacement of the normal myocardium by fatty and/or fibrofatty tissue. The fibrofatty form of the disease is characterized by thinning of the right ventricular wall and is often associated with focal myocarditis. The disease is genetically heterogeneous with mutations having been identified in the genes for desmoplakin, plakoglobin and the ryanodine receptor. Fatty or fibrofatty infiltration usually involves only the right ventricle (RV) but in 30–50% of cases will also involve the left ventricle.

The disease is characterized clinically by ventricular arrhythmias (originating from the RV) and more rarely by RV pump failure. A recent study of 100 patients found that the median age at presentation was 26 years with the most common symptoms being palpitations, syncope, and sudden cardiac death. The prevalence is thought to be approximately 5 per 10,000, although the actual number may be higher because of difficulty in accurate diagnosis. The disease is progressive and as the myocardium is replaced by fatty or fibrofatty tissue, the RV becomes dilated and dysfunctional. Arrhythmias and sudden cardiac death are more common during exercise. In some series, ARVD is the first or second most common cause of sudden cardiac death, after hypertrophic cardiomyopathy, in young individuals and in at least one large series it was the most common cause of death in young athletes.

The gold standard for the diagnosis of ARVD is biopsy evidence of fibrofatty or fatty infiltration in the RV. This is not available in all patients and thus major and minor criteria for the diagnosis of ARVD have been established by the Task Force of the Working Group of Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology. ECG changes listed under “major diagnostic criteria” include the presence of epsilon waves or localized QRS prolongation (>110 msec) in V1–V3 (Fig. 31.1). Epsilon waves are small deflections at the terminal end of the QRS complex that are best seen in V1–V3 (any terminal potential in leads V1–V3 that causes QRS duration to exceed the QRS duration in lead V6 by more than 25 msec should be considered an epsilon wave). Epsilon waves are seen in a minority of patients and their absence does not preclude the diagnosis. “Minor diagnostic criteria” include T wave inversion in the anterior precordial leads (V1–V3) in the absence of right bundle branch block, ventricular tachycardia with a left bundle branch block pattern (sustained or nonsustained; observed on ECG, Holter monitoring or stress testing) or frequent PVCs (>1000/24 hours). The amount of PVCs will often increase with exercise or emotional stress.

Electrocardiogram abnormalities are detected in more than 90% of patients with ARVD. The juvenile pattern of T wave inversion in leads V1–V3 is a normal variant in children less than 12 years of age, rare in adults >19 years (found in 1–3% of healthy individuals) but present in 87% of patients with ARVD. The differential diagnosis of these ECG findings includes myocarditis, Naxos disease, sarcoid heart disease, dilated cardiomyopathy, or right ventricular outflow tract tachycardia.

This patient was admitted to the hospital where telemetry demonstrated multiple runs of nonsustained ventricular tachycardia. A transthoracic echocardiogram demonstrated normal left ventricular function but a dilated right ventricle with decreased systolic function. A cardiac MRI demonstrated fat interdigitation in the basilar right ventricular free wall associated with diminished contractility at this site. During an electrophysio-
logy study, monomorphic ventricular tachycardia with a left bundle branch morphology was induced and voltage mapping was consistent with abnormal myocardium in the anterior free wall of the right ventricle. The patient was diagnosed with ARVD and treated with implantation of a cardioverter defibrillator.

**Further reading**


A 21-year-old male with a past medical history only significant for mild untreated hypertension presented to the emergency department via the emergency medical system with the complaint of acute onset of intense, substernal chest pain that began while sweeping a floor. The chest discomfort was associated with diaphoresis and dyspnea. Family history was significant for an uncle who suffered a myocardial infarction at the age of 36, and systemic lupus erythematosus in his mother. Physical examination revealed a blood pressure of 160/90 mmHg and a pulse of 110 bpm. Cardiovacular and pulmonary examinations were normal except for the presence of tachycardia. What clues does the ECG provide in establishing a differential diagnosis?
Anterior myocardial infarction

This ECG shows ST elevation with tall peaked T waves in leads V1–V3. This is worrisome for an anterior myocardial infarction (please see the discussion of inferior-posterior MI in Chapter 33 for a description of localizing myocardial damage based on ECG findings), especially in a patient with sudden onset of chest discomfort. If the cause is myocardial ischemia, ST segment elevation in leads V1, V2, and V3 without significant inferior ST segment depression suggests occlusion of the left anterior descending (LAD) artery after the origin of the first diagonal branch (ST elevation in V1–V6 together with ST elevation in leads I and aVL correlates with proximal occlusion of the LAD).

Because of the patient’s age, other causes of ST elevation were also considered including early repolarization, acute pericarditis, and left ventricular hypertrophy. But ST elevations in myocardial infarction are typically convex in shape and occur in a localized anatomic distribution such as in this patient. A bedside echocardiogram showed anterior hypokinesis consistent with the diagnosis of myocardial ischemia. Emergent angiography showed occlusion of the LAD just after the origin of the first diagonal (Fig. 32.1). Percutaneous coronary intervention restored flow in the LAD. The patient was subsequently diagnosed with antiphospholipid antibody syndrome. Unfortunately, he suffered a pulmonary embolism three months later despite chronic therapy with aspirin, clopidogrel and warfarin.

While not applicable in this patient, conduction defects and ventricular arrhythmias can occur in patients with anterior myocardial infarction thus necessitating constant telemetry monitoring during the peri-infarct phase. Ventricular tachycardia and/or fibrillation can occur unexpectedly, often precipitated by an “R on T” phenomenon (superimposition of an ectopic beat on the T wave of the preceding beat; Fig. 32.2). Accelerated idioventricular rhythm (AIVR) is a rhythm in which a ventricular pacemaker foci begins firing at an increased rate (“accelerates”) and takes over pacemaker function from the sinus node. AIVR has the appearance of ventricular tachycardia (a wide complex rhythm with AV dissociation) but can be differentiated by a rate which is generally <120 bpm. This rhythm is a marker of reperfusion in patients with ST elevation myocardial infarction and thus is commonly seen during primary angioplasty.

Conduction defects occurring in the setting of an anterior myocardial infarction carry an ominous prognosis. In contrast to inferior myocardial infarctions, where atrioventricular (AV) block is generally due to heightened vagal tone, advanced AV block or development of a new bundle branch...
block reflects extensive necrosis of the conduction system (most often in the presence of a proximal occlusion of the LAD). The right bundle is supplied by the septal branches of the proximal LAD and a new right bundle-branch block with a Q wave preceding the R wave in lead V1 is a specific but insensitive marker of proximal occlusion of the LAD.

**Further reading**


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Figure 32.2 ECG from a patient with an anterior wall myocardial infarction showing the initiation of ventricular fibrillation by an "R on T" phenomenon (best seen in the rhythm strip).
CHAPTER 33

Inferior-Posterior Myocardial Infarction

A 43-year-old male with a past medical history of treated hypertension arrives at the emergency department via a private vehicle with complaints of a fainting episode followed by the development of substernal chest pressure. Ninety minutes prior to arrival at the ED he was walking from the kitchen to the couch and passed out for a few seconds (per his wife), hitting his head on the way down. Upon waking he “felt fine” but his wife called the emergency medical services who evaluated him and determined that he was dehydrated and then left. Thirty minutes later he had severe substernal chest pressure which has persisted. What clues does the ECG provide in establishing a differential diagnosis?
Inferior-posterior myocardial infarction

Myocardial infarction (MI) can be readily diagnosed on this ECG. There is ST elevation in leads II, III and aVF, consistent with an injury pattern involving the inferior wall. There is ST depression in V2–V4 which probably represents posterior wall infarction (anterior ST depression is equivalent to posterior ST elevation). There is also ST depression in I and aVL. Taken together, these changes are consistent with a large myocardial infarction involving the inferior, posterior and lateral walls. Emergency angiography showed proximal occlusion of a large right coronary artery (Fig. 33.1).

Localizing coronary ischemia based on ECG findings is not an exact science but some generalities are useful. Based on typical anatomy, the left anterior descending coronary artery (LAD) and branches usually supply the anterior and anterolateral walls of the left ventricle and the anterior two-thirds of the septum. The left circumflex coronary artery and branches usually supply the posterolateral wall of the left ventricle. The right coronary artery supplies the right ventricle, the inferior and true posterior walls of the left ventricle, and the posterior third of the septum. In 15–30% of individuals, the posterior descending coronary artery (which supplies the inferior wall) will originate from the left circumflex. Rarely, the LAD will extend around the apex and onto the inferior wall.

Inferior wall infarcts are characterized by changes in the inferior leads – II, III and aVF (Table 33.1). An anterior wall infarct involves leads V3 and V4. If leads V1 and V2 are also involved it is sometimes labeled an anteroseptal infarct and if leads V5, V6 ± I and aVL are involved it is labeled an anterolateral infarct. ST elevation in leads V1–V6 is characteristic of an extensive anterior wall MI. Lateral wall infarcts are reflected in leads I and aVL ± V5 and V6. The “posterior wall” has no direct facing leads, but infarction of this territory can be reflected by a large R wave in V1 or ST depression in leads V1–V4. The use of the term “posterior wall” to identify the basal part of the left ventricle that lies on the diaphragm has fallen out of favor among some cardiologists who believe it is anatomically inaccurate and thus this terminology may change in the future.

In a recent AHA/ACC/HRS Scientific Statement,
however, the consensus was that there was insufficient evidence to change the existing terminology and they recommended using the following classifications: anterior, inferior, posterior, lateral, extensive anterior, and right ventricular MI and MI in the presence of bundle branch block.

The ECG is a tremendously useful tool in patients with chest pain and it is the most important variable in determining whether patients receive emergency reperfusion therapy. It is important, however, to emphasize that the specificity of the ECG in localizing myocardial infarction is limited by: (a) individual variations in coronary anatomy; (b) presence of coronary artery disease in the non-infarct related artery; (c) previous myocardial infarction; and (d) inadequate representation of the posterior, lateral, and apical walls of the left ventricle. The use of ECGs with more than 12 channels that better reflect the lateral and posterior walls is under investigation.

Right ventricular infarction occurs in a significant percentage of patients with inferior MI. To diagnose this condition based on ECG findings, the precordial leads should be placed on the right side of the chest in a mirror image of usual placement (Fig. 33.2). The most sensitive ECG sign of right ventricular infarction is ST segment elevation of more than 1 mm in lead V4R with an upright T wave in that lead. Other ECG findings suggestive of RV infarction include ST elevation in leads V3R, V5R, V6R or V1 in association with ST segment elevation in leads II, III, and aVF.

During ischemic events, ECG changes evolve over time. These changes are variable depending on reperfusion, the size and location of the myocardial infarction and other factors. An initial finding after the onset of ischemia may be “hyperacute” T wave changes – the appearance of tall T waves in leads corresponding to a vascular bed supplied by a coronary artery. This is followed by ST elevation. ST elevation will resolve over time with re-establishment of blood supply (via either reperfusion or development of collaterals), ST segment resolution is associated with T wave inversion and occasionally with Q wave formation (depending on the extent of the infarct). A pathologic Q wave is defined as an initial downward deflection that persists for 40 msec or more in any lead except III and aVR. An ECG on this patient, taken 36 hours after the event, shows resolution of ST elevation, development of Q waves and T wave inversion (Fig. 33.3). Over time, the T wave will return to an upright position in most individuals. Persistent ST elevation, especially in the setting of Q waves, is a marker of aneurysm formation.

Several studies have looked at ECG findings that help identify which artery is involved in an inferior wall MI. The major findings of these studies are that: (a) ST segment elevation in lead III > lead II; (b) ST segment depression of more than 1 mm in leads I and aVL; or (c) ST segment elevation in lead V1 suggest right coronary artery involvement. An ST segment vector directed toward the left (i.e. lead II > lead III) suggests circumflex involvement.

Arrhythmias, including atrioventricular (AV) block, accelerated idioventricular rhythm or ventricular arrhythmias, are common during inferior myocardial infarction. An ECG with second- and third-degree AV block in the setting of an inferior MI from a different patient is shown in Figure 33.4. AV block generally occurs early (within a few hours) in an inferior MI as a result of heightened vagal tone. Complete atrioventricular block is generally associated with a narrow complex escape rhythm of between 40 and 60 beats per minute.
Figure 33.3 ECG taken 36 hours after initial presentation.

Figure 33.4 Inferior myocardial infarction with second- and third-degree AV block.

Further reading

list, a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol 2007;49(10):1128–1135.


A 52-year-old male with diabetes and hypertension presented to the emergency department with complaints of a sudden worsening in chest pain. One week previously he was involved in a major motor vehicle accident with multiple injuries including rib fractures for which he was hospitalized. He was discharged to a rehabilitation facility where he had sudden worsening of chest pain while watching TV. What clues does the ECG provide in establishing a differential diagnosis?
Acute anterior myocardial infarction in the setting of right bundle branch block

In patients with an existing right bundle branch block (RBBB), ST-T abnormalities in leads V1–V3 are common due to abnormal right ventricular repolarization. The diagnostic specificity of ST elevation for myocardial infarction declines somewhat in RBBB compared to normal intraventricular conduction. However, the presence of ST elevation, especially in a patient with appropriate symptoms, should prompt consideration of myocardial infarction. In this patient, emergency angiography showed occlusion of the left anterior descending coronary artery just after the origin of the first diagonal. ECG after reperfusion showed resolution of the ST elevation (Fig. 34.1).

Patients who develop RBBB during an acute anterior ST elevation myocardial infarction have a high risk for mortality (e.g. 30-day mortality rate was fourfold higher compared to patients with normal intraventricular conduction in the Hirulog Early Reperfusion Occlusion (HERO-2) trial). The elevated risk in these patients results from extensive necrosis involving the interventricular septum as the right bundle branch is supplied by the septal branches of the proximal LAD. The presence of RBBB does not affect interpretation of Q waves since RBBB affects primarily the terminal phase of ventricular depolarization. Thus the criteria for the diagnosis of a Q wave MI in a patient with RBBB are the same as in patients with normal conduction.

Diagnosis of a myocardial infarction in the presence of an existing left bundle branch block (LBBB) can be difficult even in the presence of marked ST-T abnormalities or ST elevation. Although somewhat limited in sensitivity and specificity, features that suggest ischemia in the presence of a chronic LBBB include ST segment elevation in association with a positive QRS complex (in general in LBBB, ST elevation is only seen in leads with a predominantly negative QRS complex), ST segment depression in leads V1, V2 or V3 (which have predominantly negative QRS complexes) or extreme ST segment elevation (≥5 mm) in leads V1 and V2. The development of a new LBBB in a patient with an acute coronary syndrome is associated with a significant risk for adverse outcome and is consistent with proximal LAD occlusion.

Figure 34.1 ECG recorded 12 hours after reperfusion of the LAD.
Further reading


A 29-year-old previously healthy female presents with a 3-day history of fever, sore throat and chest pain. An ECG is obtained which is shown below. What is the diagnosis?
Pericarditis

This ECG is consistent with the diagnosis of diffuse atrial and ventricular injury. There is ST elevation in leads II, aVF and V2–V6. This elevation is widespread and not in a pattern generally seen with myocardial infarction (discussed in Chapter 33). The other ECG finding of interest is PR depression (seen especially in lead II) which reflects atrial injury (i.e. in the same way that ST elevation reflects ventricular injury).

The most likely clinical diagnosis given the patient’s complaints and the ECG is pericarditis. Acute pericarditis results from inflammation of the pericardium. It is more common in men than in women and in adults more than children. Causes of acute pericarditis include viral, idiopathic, uremia, pericardectomy associated with cardiac surgery, pulmonary embolism, collagen vascular diseases, Dressler syndrome, malignancy, tuberculosis, fungus (e.g. histoplasmosis), parasites (e.g. amoeba), myxedema, radiation, acute rheumatic fever and trauma. Remember that while the term "pericarditis" is commonly used, the inflammatory process also involves the myocardium in most patients.

Clinically pericarditis is characterized by chest pain which is generally sharp and pleuritic. The pain is positional, worsened by lying supine and improved by leaning forward. A pericardial friction rub, heard best along the lower left sternal border, is generally the most remarkable physical exam finding. It typically has three components (when the patient is in sinus rhythm) corresponding to atrial systole, ventricular systole and rapid ventricular filling during early diastole.

There are four stages of ECG changes associated with the evolution of acute pericarditis (Table 35.1). Stage I changes accompany the onset of chest pain and include the “classic” ECG changes associated with acute pericarditis: diffuse concave ST elevation with PR depression. The ST segment elevation (usually of 1–2 mm) is generally widespread and concave upward. PR depression is generally more evident in lead II, although it can be seen in other leads. Stage II occurs several days later and is represented by the return of ST segments to baseline and T wave flattening. In stage III, T wave inversion is seen in most leads, especially those in which ST elevation was present in stage 1. Stage IV represents the return of upright T waves. The approximate time frame for passage through all four stages of ECG changes in most cases of acute pericarditis is 2 weeks.

ECG abnormalities are present in approximately 90% of patients with acute pericarditis but only approximately 50% of patients will manifest all four stages. Other ECG presentations include isolated PR depression, absence of one or more stages or persistence of T wave inversion. Sinus tachycardia may be present and atrial arrhythmias are seen in 5–10% of cases.

A last important point: there are no ECG findings that are diagnostic of pericarditis. The ECG must be interpreted in light of the clinical scenario.

The patient was treated with non-steroidal anti-inflammatory medications and her symptoms resolved.
SECTION D
Cardiovascular Manifestations of Systemic Diseases
You are asked to evaluate a 26-year-old male in your clinic because of an abnormal ECG. He has a long history of muscle weakness which runs in his family.
Duchenne muscular dystrophy

This ECG is from a patient with Duchenne type muscular dystrophy (DMD). DMD is a recessive sex-linked hereditary degenerative muscular disorder usually caused by large deletions in the dystrophin gene; spontaneous mutations are also common. This results in a damaged sarcolemmal membrane, excess calcium entry, and muscle fiber necrosis. DMD is characterized by progressive cardiac involvement, and abnormal ECG patterns are found in 95% of patients. ECG changes are often significantly distinct to strongly suggest the diagnosis of DMD although the diagnosis is usually made before an ECG is recorded.

A common finding is fibrous scarring of the posterior wall of the left ventricle, as seen in the irregularly shaped QRS complexes of slightly long duration in this ECG. The tall R waves in this leads V1–V3 and the deep Q waves in the lateral leads in this patient can be confused with those of lateral myocardial infarction (MI), however they are thinner than those of a MI. Posterior and posterolateral MI’s are also commonly simulated by DMD.

Arrhythmias often accompany DMD: persistent and labile sinus tachycardia, sinus arrhythmia, sinus pauses, atrial ectopic beats, atrial ectopic rhythm, junctional rhythm, atrial flutter, and ventricular premature beats. Conduction is often altered by DMD as well and may include: abnormal intraatrial or interatrial conduction, Mobitz type I AV block, nonconducted atrial premature beats, short PR interval, right ventricular conduction delay, and rightward axis deviation associated with left posterior fascicular block.

Further reading

A 39-year-old female is brought to the emergency room with abrupt onset of altered state of consciousness. She is a known drug abuser with her most recent drug of choice being cocaine. Past medical history includes end-stage renal disease on hemodialysis, long-standing severe hypertension, and a history of pulmonary embolism. What clues does the ECG provide in establishing a differential diagnosis?
Intracerebral bleed

This ECG shows sinus rhythm with prolonged QTc interval, biphasic T waves in V1 and V2 and diffuse T wave flattening in the other leads. ECG one month prior (Fig. 37.1) showed normal sinus rhythm at a rate of 84 beats per minute. P wave and QRS duration and axis are within normal limits. There is T wave inversion in leads I and aVL and the patient may have left ventricular hypertrophy (LVH) by the Cornell voltage criteria (S in V3 + R in aVL >20 mm). Comparison of the two ECGs shows that major changes include prolongation of the QTc interval and diffuse T wave changes. These changes are non-specific, however, the pattern of a marked increase in the QTc interval and diffuse ST/T wave changes, in a patient with altered mental status, should prompt consideration of the diagnosis of an intracerebral event. In this patient, a head CT showed basal ganglia hemorrhage with hydrocephalus.

Acute brain injury, via hemorrhage, trauma, meningitis, malignancy or other causes, can result in ECG changes and arrhythmias and, in rare cases, cardiac dysfunction and isoenzyme elevation. The majority of patients with intracerebral hemorrhage present with ECG abnormalities such as prolonged QT intervals, ST segment elevation or depression, inverted T waves, and/or ventricular ectopy. ST segment elevation mimicking acute infarction and acute pericarditis has been described. The ECG changes are dynamic and may evolve over several days. The mechanism has not been clearly established but is thought to relate to an abrupt elevation of serum catecholamine levels with or without decreased parasympathetic nervous activity. Intracerebral hemorrhage also has been shown to effect intracellular Ca²⁺ levels in cardiac myocytes.

Occasionally, the ECG can provide clues that are helpful in the diagnosis of rare causes of altered mental status. A short QTc interval is found in severe hypercalcemia. Sinus bradycardia and low voltage are seen in hypothyroidism. QRS widening, especially in the presence of QTc prolongation, is consistent with an overdose of tricyclic antidepressants. Ischemic changes can suggest carbon monoxide poisoning.

Further reading

A 66-year-old female with a history of treated hypertension presents to the emergency department via emergency medical system transport. She reports progressive dyspnea over the past few weeks, with sudden worsening tonight while walking to the bathroom. What clues does the ECG provide in establishing a differential diagnosis?
**Pulmonary embolus**

Electrocardiographic abnormalities, including unexplained tachycardia, are common in acute pulmonary embolism (PE) but non-specific. The ECG is abnormal in over 70% of patients with PE, with sinus tachycardia being the most common abnormality. Other ECG findings generally relate to the effect of the PE on right ventricular and right atrial pressure and function and may include right bundle branch block (RBBB), large R wave in lead V1, right axis deviation, T wave inversion in leads V1–V4, P-pulmonale, and/or atrial arrhythmias. The combination of an S wave in lead I and a Q wave and inverted T wave in lead III gives the SIQIII-TIII pattern suggestive of right ventricular strain. This ECG pattern has been reported to occur in 12% of patients with massive PE. Numerous other ECG findings have also been reported in PE.

All of these findings are non-specific and the ECG is neither sensitive nor specific for the diagnosis of pulmonary embolus. Rodger et al studied the diagnostic value of 30 different ECG changes in 246 patients with suspected PE. They found that tachycardia and incomplete right bundle branch block were the only ECG findings that were significantly more common in patients with proven PE. The positive predictive value of tachycardia and incomplete RBBB was 38% and 100% respectively. Acute changes of right ventricular overload can also be seen in other conditions such as pneumonia or acute exacerbation of chronic obstructive pulmonary disease.

In contrast to limited usefulness in diagnosing PE, some studies have shown that prognostic information can be derived from the ECG in patients with known PE. The presence of low-voltage atrial fibrillation and/or premature ventricular extrasystoles has been associated with increased 30-day mortality. A 21-point ECG scoring system has been developed that predicts RV dysfunction in patients with acute PE although the ability to predict in-hospital outcomes is limited. ECG abnormalities have been observed to return to normal after treatment for PE and resolution of anterior T wave changes has been linked to an improved prognosis.

**Further reading**


A psychiatrist asks you to evaluate a 22-year-old male who is being treated for agitation and attention deficit hyperactivity disorder. You obtain a history of weight loss, sweats, headache, diarrhea, and heat intolerance for the past nine months. An ECG is shown below. What is the diagnosis?
Hyperthyroidism

Thyroid hormones have direct effects on the heart and peripheral vasculature and many of the clinical manifestations of hyperthyroidism are due to altered cardiovascular hemodynamics. Hyperthyroidism is associated with increased resting heart rate, left ventricular contractility, cardiac output, pulse pressure and blood volume and decreased systemic vascular resistance. Systolic hypertension is common. Cardiovascular complaints include palpitations, tachycardia, exercise intolerance and dyspnea on exertion. There are no specific ECG findings that can be used to diagnose hyperthyroidism but rather tachycardia and atrial arrhythmias can be clues to the presence of thyroid dysfunction.

Sinus tachycardia is the most common rhythm disturbance in hyperthyroidism with an increase in resting heart rate observed in almost all patients with overt hyperthyroidism. Continuous ECG monitoring usually demonstrates that heart rate is consistently increased throughout the 24-hour period and exaggerated in response to exercise. Thyroid hormone has direct effects on pacemaker cells and affects the action potential duration and repolarization currents. Atrial fibrillation occurs in 5–20% of patients with hyperthyroidism. In contrast, hyperthyroidism is rare in patients with atrial fibrillation, occurring in <1%. Other supraventricular arrhythmias (e.g. atrial flutter or atrial tachycardia) are rare but can occur with hyperthyroidism. Ventricular arrhythmias are usually not seen.

In a study of 393 patients with overt hyperthyroidism and undetectable thyroid stimulating hormone (TSH), complaints of chest pain, palpitations, dyspnea and cough were more common than in age- and gender-matched controls. Pulse rate and systolic blood pressure at rest were higher in hyperthyroid patients but there was no difference in diastolic blood pressure. The resting pulse rate was 82 ± 1 bpm in patients with hyperthyroidism versus 73 ± 1 bpm in controls. In this study, 7.3% of patients with hyperthyroidism had atrial fibrillation compared to 1% of the control group.

The hemodynamic changes of hypothyroidism are opposite to those of hyperthyroidism and include bradycardia, narrowed pulse pressure, decreased cardiac contractility and increased systemic vascular resistance. Hypothyroidism prolongs the cardiac action potential and the QT interval and, in rare cases, can predispose to ventricular irritability and cause torsade de pointes. Atrial arrhythmias are uncommon, in contrast to hyperthyroidism. Thyroxine therapy reverses all the cardiovascular changes associated with hypothyroidism.

In this patient, total T4 was >24.9 µg/dL (normal 5.5–11.0), total T3 was >7.8 ng/mL (normal 1.0–1.7) and TSH was undetectable. After additional testing, the patient was diagnosed with Graves disease.

References

SECTION E

Miscellaneous
chapter 40

hyperkalemia

you are asked to evaluate a 25-year-old graduate student without any past medical history who comes to the emergency room complaining of progressive dyspnea for one week.
Hyperkalemia

The tall pointed T waves of this ECG together with the wide initial and terminal parts of the QRS complex allow diagnosis of hyperkalemia. Hyperkalemia begins to alter the ECG once potassium concentrations in the plasma exceed 5.5 mM, as the T waves become taller and more peaked. Once 6.5 mM is reached, uniform QRS widening tends to occur.

P wave amplitude decreases and duration increases at concentrations above 7.0 mM. The PR interval also increases at this concentration, but this is mostly due to the increased P wave duration. At concentrations above 8.0 mM, the P wave often disappears altogether. Once the plasma concentration reaches 10 mM, several escape pacemakers in the depressed myocardium can cause irregular ventricular rhythm; in the absence of P waves, this may simulate atrial fibrillation. However, in cases of pre-existing atrial fibrillation, hyperkalemia will usually cause a slow ventricular rate. Ventricular asystole or ventricular fibrillation will occur once the potassium concentration reaches 12–14 mM. Advanced hyperkalemia can mimic an ECG from a dying heart: the ST segment can simulate the “acute injury” pattern, but this will quickly subside with treatment.

The characteristic tall, steep T waves are helpful in the diagnosis of hyperkalemia but the lack of this feature cannot be used to exclude elevated potassium levels. Only 22% of hyperkalemic patients have T wave changes that are sufficient to diagnose hyperkalemia.1

This patient had a potassium concentration of 7.3 mM in the setting of new-onset renal failure. An ECG showing resolution of the ECG changes associated with hyperkalemia is shown in Figure 40.1.

Further reading

A 32-year-old male without prior cardiac disease is being evaluated in the emergency room with complaints of chest pain that began 2 hours ago. He admits to using cocaine last night. What is the diagnosis?
Early repolarization

This ECG demonstrates the findings of early repolarization with J point elevation and ST segment elevation in leads I, II, aVF and V2–V6. Early repolarization is generally defined as J point elevation (elevation of the QRS–ST junction), slurring or notching of the J point and ST segment elevation of at least 0.1 mV from baseline. The location of the maximal ST elevation in early repolarization is variable, with the most common site being chest leads V3 and V4. Maximal ST elevation can, however, occur laterally (leads I, aVL, V5, and V6), inferiorly (leads II, III, and aVF) or anteriorly (leads V1 and V4).

Early repolarization is a common ECG variant, being found in 1–7% of individuals. It is more common in young individuals, males, athletes and African-Americans. Dynamic changes may occur in the width and height of the ST segment. For example, interventions that increase heart rate, such as exercise testing or isoproterenol infusion, generally reduce or eliminate early repolarization whereas agents that slow heart rate, such as beta-blockers, will accent ST elevation in these patients.

Early repolarization has historically been considered benign although recent studies have linked early repolarization changes in the inferolateral leads to sudden cardiac death. In a recent study involving 22 tertiary care arrhythmia centers, Haïssaguerre et al found early repolarization in the inferolateral leads in 64 of 206 patients (31%) who were resuscitated after idiopathic ventricular fibrillation. While the evidence is not conclusive, “early repolarization” changes in the inferolateral leads, especially in men, maybe useful in identifying individuals at an increased risk of syncope and sudden death.

Early repolarization can be confused with acute myocardial infarction or pericarditis. Several criteria can be used to identify patients with early repolarization although none is 100% accurate. These include:

- absence of chest pain
- young age
- widespread ST elevation
- marked J point elevation
- concavity of initial upsloping portion of ST segment
- notching or irregular contour of J point
- prominent, concordant T waves
- current ECG is unchanged from prior ECGs.

Distinguishing pericarditis from early repolarization may be difficult although serial ECGs over several days may be helpful. In early repolarization the ST segments remain constant whereas, in general, the ECG in patients with acute pericarditis evolves over time.

Reference

CHAPTER 42

P-Pulmonale

This is an ECG of a 34-year-old female with systemic lupus erythematosus and hyperthyroidism who is referred to you for evaluation of complaints of increasing fatigue.
P-pulmonale

The rhythm is sinus tachycardia with a rate of 108 beats per minute. The P waves are tall and peaked, especially in lead II, with an amplitude of 2.5 mV. These ECG findings are consistent with a diagnosis of P-pulmonale.

P-pulmonale is defined as tall (>2.5 mV), peaked P waves in any of the inferior leads (II, III and aVF) with normal P wave duration. Less consistently, it can also include a positive deflection in the P wave in V1 or V2 of ≥1.5 mm. Remember that since the right atrium depolarizes first, the first part of the P wave is indicative of the right atrium in V1. P-pulmonale is a marker of right atrial dilation or hypertrophy and is frequently seen in patients with lung disease. Clinically these patients are often those with chronic obstructive pulmonary disease (COPD) but P-pulmonale can also be present in pneumonia, congenital heart disease, congestive heart failure, pulmonary emboli, asthma or tricuspid valve disease. P-pulmonale can be either a constant finding or transient, especially with pulmonary emboli or an exacerbation of COPD.

Normally the P wave is formed by overlapping depolarization of the right and left atria leading to a smooth rounded wave less than 0.12 sec and usually less than 1 mm in height. The forces generated by right atrial depolarization are directed anteriorly and inferiorly and produce the early part of the P wave. In the presence of right atrial enlargement, P wave amplitude increases although the overall duration of the P wave is usually not prolonged. As P-pulmonale progresses, the voltage in the P wave increases both from delayed activation of the right atrium, causing simultaneous activation of the right and left atria, and the increase in right atrial tissue that is depolarizing.

Lastly, while a diagnosis of P-pulmonale on the ECG provides a clue to the presence of cardiac or pulmonary disease, remember that electrocardiography is neither a sensitive nor specific tool for diagnosing conditions such as right atrial enlargement, right ventricular hypertrophy or pulmonary hypertension. This was demonstrated in a study of 100 consecutive patients with P-pulmonale on ECG, 46 of whom did not have any clinical condition that would cause right atrial abnormalities. A small echocardiographic study found that right atrial enlargement was found in only two of 11 patients with P-pulmonale and one of five with prominent positive P wave forces in lead V1.

In this patient, echocardiography revealed normal right atrial and right ventricular size and function.

References

A 46-year-old male with congestive heart failure and atrial fibrillation presents to the emergency department with complaints of palpitations for approximately 12 hours. He reports nausea and vomiting for several days which has recently worsened. What clues does the ECG provide in establishing a differential diagnosis?
**Digoxin toxicity**

This ECG shows a regular, narrow complex tachycardia at a rate of approximately 140 bpm. There are P waves prior to each QRS complex with a prolonged PR interval. The P wave axis is abnormal (negative in II, III and aVF) and thus the rhythm is an atrial tachycardia with first-degree atrioventricular (AV) block. The QT interval is short and there are pronounced diffuse ST abnormalities. The combination of an atrial tachycardia with AV block, shortened QT interval and diffuse ST changes is consistent with a diagnosis of digoxin toxicity. This patient’s digoxin level was 8 nmol/L (therapeutic concentration is generally 1–2 nmol/L). He was given Digibind, a specific antidote for digoxin toxicity which utilizes antigen-binding fragments (Fab) derived from specific antidigoxin antibodies raised in sheep to neutralize circulating digoxin. After treatment, ECG showed sinus rhythm (note that the P wave axis has normalized) with a rate of approximately 84 bpm and resolution of ST segment depression (Fig. 43.1).

Digitalis derivatives are found in several plants, including oleander and foxglove (*Digitalis lanata*), and are used therapeutically as digoxin or digitoxin. Cardiac glycosides have both inotropic and AV blocking effects and were once widely used in heart failure and atrial fibrillation. They are now less popular but occasionally still utilized in selected patients.

The effects of digitalis result from the inhibition of the sodium/potassium/adenosine triphosphatase (NA\(^+\)/K\(^+\) ATPase) pump. It binds in a reversible process to a specific site on the extracytoplasmic surface of the alpha subunit of the sodium- and potassium-activated adenosine triphosphatase pump. This results in an increase in intracellular sodium and a decrease in intracellular potassium. The increase in intracellular Na\(^+\) levels reduces the extrusion of calcium (Ca\(^{2+}\)) by the Na\(^+\)/Ca\(^{2+}\) exchange pump, resulting in increased intracellular Ca\(^{2+}\) which increases the force of myocardial contraction. These effects cause a decreased resting potential which slows the rate of Phase 0 depolarization, a decrease in action potential duration, and enhanced automaticity which results in an increase in the rate of Phase 4 depolarization. Digitalis also has a negative chronotropic action, which is due to increased vagal tone and direct effects on the sinoatrial (SA) and AV nodes.

The effect of digitalis on cardiac cells and thus the potential for toxicity is a function of various factors including amount ingested, renal excretion (which is regulated by renal function, dehydration, drug interactions, etc.) and electrolyte levels.

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**Figure 43.1** ECG after administration of Digibind.
Hypokalemia, hypernatremia or hypomagnesemia increases the toxic cardiovascular effects of digitals because of their depressive effects on the Na⁺/K⁺ ATPase pump. Acidosis and myocardial ischemia will also affect the response to digitals. Various medications can affect digitals levels including anti-arrhythmics (e.g. amiodarone, quinidine), diuretics, antibiotics and cyclosporine.

Digitalis has effects on the ECG even at therapeutic concentrations. These can include a shortened QT, downward sagging or “scooping” of the ST segment with concomitant ST depression (this is usually most pronounced in the lateral leads), an increase in U wave amplitude and T wave changes. Direct effect of digitalis on repolarization is often reflected on the ECG by ST segment and T-wave forces which are opposite in direction to the predominant QRS force.

In contrast, PR or QRS prolongation, varying degrees of AV block, and arrhythmias may signify digitalis toxicity. Cardiac dysrhythmias associated with digitalis generally arise from either an increase in automaticity or a decrease in conduction. Premature ventricular contractions are the most common arrhythmia associated with digitalis toxicity but other arrhythmias that can be seen include atrial tachycardia, junctional tachycardia, ventricular tachycardia, atrial flutter, alternating ventricular pacemakers, SA arrest, and varying degrees of AV block. Almost any dysrhythmia maybe precipitated by digitalis except sinus tachycardia, AV nodal reentry tachycardia, atrioventricular reciprocating tachycardia and rapid atrial fibrillation. Atrial tachycardia with AV block and bidirectional ventricular tachycardia (alternating bundle branch blocks) are particularly characteristic of severe digitalis toxicity. Although there is no single arrhythmia that is diagnostic of digitalis toxicity, the combination of enhanced automaticity and impaired conduction (e.g. atrial tachycardia with 1st degree AV block as in this patient) should raise suspicion of toxicity in a patient treated with digitalis.

Further reading
A 46-year-old female presented to the emergency department with 2 weeks of accelerating dyspnea on exertion and daily emesis. She reported some global fatigue and being “short-winded” over the last 2–3 months. She initially attributed these symptoms to her hectic schedule but over the last 2 weeks her dyspnea progressed, becoming so severe that walking several steps in her house is completely exhausting. She also reports episodes where she feels as though she may pass out, but denies frank syncope. What clues does the ECG provide in establishing a differential diagnosis?
Ectopic atrial rhythm with diffuse T wave inversion

The sinoatrial (SA) node, the normal pacemaker of the heart, is located in the posterior part of the right atrium at the junction of the superior vena cava. In normal sinus rhythm, the P wave axis (determined from the limb leads) varies from 0 to +75 degrees (i.e. directed inferiorly and leftward). The P wave is always upright in leads I and II. In an ectopic atrial rhythm, the P wave axis is generally abnormal and P waves are negative in either lead I or lead II. The morphology of the P wave and the direction of the frontal plane P wave axis depends on the location of the automatic focus and the pathway of atrial activation. For example, when the focus is lower in the atrium, the P waves in the inferior leads are inverted. In general, in ectopic atrial rhythm the rate is <100 bpm and the PR interval is normal. If the P wave morphology varies and there are multiple P wave morphologies, the rhythm is likely to be wandering atrial pacemaker (see Chapter 12 for more information on this arrhythmia).

Ectopic atrial rhythm is generally a disorder of increased automaticity although re-entry mechanisms can also be a cause. Increased automaticity can be observed in tissue within the atria, the AV junction, the vena cava or pulmonary veins. Enhanced automaticity results from increased diastolic Phase 4 depolarization causing an increase in rate of activation. If the rate of the ectopic focus exceeds that of the sinus node, then the ectopic focus will become the predominant pacemaker of the heart.

This patient was found on echocardiography to have an atrial myxoma (Fig. 44.1). Myxomas are the most common primary cardiac tumor, with 75% being found in the left atrium. There are no ECG findings diagnostic of a myxoma. Rather, the ECG findings associated with a myxoma are indirect and depend on the location of the tumor. If the tumor is only in the atria, there can be P wave abnormalities or atrial arrhythmias (including ectopic atrial rhythm as in this patient) on ECG. If the tumor extends into the mitral annulus and affects the function of the valve, findings associated with mitral valve disease, such as atrial enlargement, can also be found on the ECG. Right bundle branch block and ST abnormalities have been reported with myxomas. Lastly, diffuse T wave inversion and/or ventricular ectopy (note the fifth and sixth beats), as seen on this ECG, can also be present.

Figure 44.1 Echocardiogram showing left atrial mass.
A 25-year-old homeless man is brought to the emergency room after being found unconscious. What clues does the ECG provide in establishing a differential diagnosis?
Hypothermia

This ECG is from a patient suffering from hypothermia or low core body temperature. The main diagnostic clue is the late positive deflections after the terminal portion of the QRS complex, known as Osborn or J waves (present in the inferior and lateral leads but most prominent in leads V3–V6). Other manifestations of hypothermia on this ECG include prolongation of the PR interval, T wave inversion, increased QT interval duration and QRS prolongation.

The rhythm is atrial fibrillation which occurs in approximately 50% of patients with hypothermia. Atrial flutter, AV junctional rhythm, ventricular ectopic beats and ventricular fibrillation can also occur with hypothermia. Note the artifact due to rapid, intermittent oscillations in this ECG. This is often present in hypothermic patients because of shivering.

A distinct inflection in the J point was described by John Osborn in 1953 based on his experiments on cardiac and respiratory function in hypothermic dogs. Over the years different names have been applied to this phenomenon including camel hump sign, late delta wave, hypothermic wave, J point wave, K wave, H wave and current of injury, with most current commentators using the term Osborn waves. Osborn thought acidemia induced by hypothermia was the primary cause of the inflection in the J point but subsequent work has shown that a difference in the electrophysiology of the ventricular epicardium and endocardium is the basis for Osborn waves. Specifically, Osborn waves are thought to arise from differences in the transient outward current of the endo- and epicardium during early repolarization.

Osborn waves (Fig. 45.1) are commonly seen in the inferior and lateral leads (II, III, aVF, V5 and V6), become more prominent as the body temperature drops, and regress with rewarming. At temperatures below 86 degrees, 80% of patients have Osborn waves. Osborn waves are traditionally associated with hypothermia but have also been reported in patients with extreme hypercalcemia, acute brain injury, subarachnoid hemorrhage, cardiopulmonary arrest, vasospastic angina, and idiopathic ventricular fibrillation.

ECG changes become more prominent with progressive decreases in temperature. Sinus tachycardia may be the only ECG manifestation of mild hypothermia. If the core body temperature continues to drop, sinus bradycardia supervenes and is associated with progressive prolongation of the PR interval, QRS complex, and QT interval. Further decreases in temperature are often associated with premature atrial contractions and/or atrial fibrillation. With temperature <86 degrees Fahrenheit, a progressive widening of the QRS complex increases the risk of ventricular fibrillation. When the temperature drops to ≈60 degrees Fahrenheit, asystole supervenes.

The Osborn waves in this patient resolved and sinus rhythm returned as his body temperature increased (Fig. 45.2).
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