

Electrocardiography: The ECG

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The electrocardiogram (ECG) is a standard American Society of Anesthesiologists monitor and should be continuously displayed during an operative procedure. In addition to providing a wealth of physiological information, including information on the electrical activity of the heart, the ECG assists in monitoring and detecting a variety of changes, such as cardiac arrhythmias, electrolyte changes, and ischemia.

Myocardial cells are dynamic, undergoing depolarization and repolarization. The result of this activity is the contraction and relaxation of the heart and the maintenance of cardiovascular function. The depolarization and repolarization of the myocardial cell generates electrical dipoles, which can be pictorially represented as vectors. Vectors have mathematical properties that allow summation. Einthoven was the first to visualize the electrical activity of the heart as a summation of vectors with the resultant vector representing a single dipole within an equilateral triangle. The six standard leads (I, II, III, aVR, aVL, aVF) give a frontal-plane view of the heart. The combination of these leads gives a biplanar or two-dimensional view of a dynamic three-dimensional structure. The ECG represents the surface recording of the instantaneous electrical activity of the heart.

Components of ECG analysis

The normal ECG consists of P waves, PR interval, QRS complex, ST segment, T wave, QT interval, and, at times, a U wave (Fig. 1). Atrial activity is represented in the P wave and PR interval while the QRS wave, ST segment, T wave, and QT segment provide information on ventricular activity. ECG analysis provides information on atrial size, atrial activity, ventricular size,

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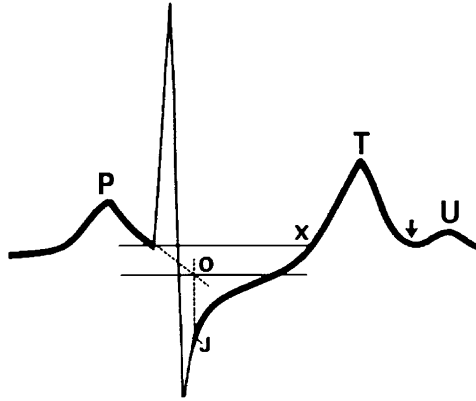


Fig. 1. Basic ECG. Arrow indicates. J, J wave; P, P wave; T, T wave; U, U wave.

ventricular activity, rate of cardiac activity, the synchronization of atrial and ventricular conduction, and the presence of damaged or ischemic myocardium. Through a detailed and systematic approach to analysis, the clinician can extract precise information provided in the ECG. However, the clinician should always bear in mind potential sources of error in the analysis.

Normal electrical activity begins in the sinoatrial node, spreads to the right atrium, then to the interatrial septum and left atrium on the way toward the atrioventricular node. The P wave represents this atrial electrical activity as the electrical impulse moves through the right atrium anteriorly and inferiorly, the interatrial Bachman bundle, and the left atrium posteriorly and inferiorly to the atrioventricular node via anterior, middle, and posterior Purkinje pathways with the left atrial appendage being the last atrial component to contract. A biphasic P wave in lead V1 and V2 often represents the transition of right atrial contraction anteriorly and inferiorly and the left atrial contraction posteriorly and inferiorly, but this is not seen in the other leads because of the sequential spread of electrical activity from the right to the left atrium. A negative deflection of the P wave is normal in aVR and may also be seen in lead III [1]. The width of the impulse represents the time duration for the impulse to spread, while the height of the impulse or amplitude represents the strength of the signal received by the sensing electrode. The normal duration of a P wave is <0.12 seconds and presents with an amplitude of <0.2 mV [2].

The PR interval measures the time for an impulse through atrioventricular conduction to travel from the sinoatrial node, through the atria and the atrioventricular node, down the bundle branches, and through the Purkinje network until the onset of ventricular contraction. The PR interval includes the time for atrial depolarization, as revealed in the P wave, and the time for atrial repolarization, as measured by the PR segment—the time from the P wave to the onset of the QRS complex—and thus the PR interval assesses

the period of atrial activity. To accurately measure this period, the lead with the widest P wave, the longest QRS wave, and shortest PR interval should be chosen for measurement. As revealed in the PR segment, the period of atrial repolarization, which is usually in an opposite direction to the P wave, is usually flat or horizontal, but during periods of increased heart rate may be sloping downward with sloping steeper in leads with P waves of larger amplitude. The normal PR interval varies and increases with age. Children tend to have shorter PR intervals and older individuals longer PR intervals, but the normal PR interval should remain <0.20 second [1].

The QRS complex encompasses the period of ventricular activation, depolarization, and contraction. This is the time when most of the heart's musculature engages in a smooth, synchronized, concerted effort to propel the blood to both the pulmonic and systemic circulations to maintain life processes. The morphology, duration, axis, and amplitude of the QRS complex represents the summation of this electrical activity, which results in the twisting, wringing contractions of the heart and forward propulsion of the blood. Electrical activation, which begins in the high left portion of the middle septal region, spreads downward toward the apex. Out of this initial wave, two waves emerge. One travels from the lower left septum spreading toward the right. The other travels from the right lower septum spreading to the left. Both waves follow anterior and posterior pathways. The apical anterior aspects of the heart are initially activated with electrical activity spreading finally to the posterior basilar portion of the heart [1].

The QRS complex in its entirety represents this electrical activity with the component elements of the QRS complex including the Q wave, the R wave, the S wave, and, at times, a QS wave and an R^1 wave. The Q wave is the first negative or downward deflection after the P wave and represents ventricular vectors moving away from the sensing electrode. A small Q wave may be present in normal adults in the inferior leads (II, III, and aVF); the left lateral precordial leads ($V_6, V_5 > V_4 > V_3$), and occasionally in leads I and VL. The greater the amplitude and the longer the duration, the greater the likelihood that the Q wave is indicative of a myocardial infarction, especially when it was not present on a prior ECG. A significant Q wave is taken to be >40 ms with an amplitude greater than one quarter to one third the height of the QRS complex. The R wave is the first positive deflection after the P wave and increases in amplitude from the right to the left precordium, indicating a greater proximity of the heart to the electrode. The S wave is a negative or downward deflection following the R wave. The S wave tends to be most prominent in the right precordial leads, especially V2 and also somewhat prominent in aVR. The QS wave represents a single downward or negative deflection after the P wave, which is not followed by an R wave. This downward deflection may be seen in obese patients with a horizontal heart position in lead III, whereas in thin patients with a vertical heart problem, it may be seen in lead aVL. In rare cases, the QS wave may represent a normal variant if present in leads III, aVL, and aVF [1]. The R^1 wave is another

positive deflection after the R wave and may represent the late depolarization of the right ventricular outflow tract or the late depolarization of the basilar aspect of the septum. When seen in lead V1, the R¹ wave may be indicative of a right bundle branch block [1].

The morphology of the QRS complex must sum a variety of three-dimensional vectorial representations of a dynamic process occurring in a large mass of cardiac tissue, accounting for the vectorial representation of the initial septal and paraseptal depolarization, followed by the ventricular free-wall activation, and finally the vectorial representation of basilar ventricular activity. The morphology of the QRS complex with its constituent elements helps to define ventricular electrical activity and also reveals the presence of abnormal conduction, ischemia, and hypertrophy, as well as the presence of pacing devices. The amplitude of the QRS complex reflects not only the proximity and size of the heart, but also the presence of disease processes, such as emphysema, pneumothorax, cardiac tamponade, constrictive pericarditis, and scleroderma, which presents with low voltage. The electrical axis represents the direction of the mean QRS complex vector in the frontal plane. This axis may be determined in one of two ways:

- Find the lead with the most isoelectric QRS complex. The axis will be perpendicular to this lead in the direction of the limb lead with the most positive deflection; or
- Determine the vectorial sum of the QRS complex in leads I and AVF.

These two methods do not always produce the same result. The normal QRS axis lies between -30° and $+90^\circ$, usually $+60^\circ$ in the direction of lead II. Left axis deviation begins to occur as the axis moves toward 0° and -30° and becomes markedly pronounced when it extends beyond -30° toward -90° with the QRS positive in lead I and negative in lead II. With aging, the electrical axis gradually moves leftward and, with obesity, the heart shifts to a more horizontal plane, also resulting in leftward movement. In addition, left axis deviation is also associated with atrioventricular canal defects; ostium primum atrial septal defects; left anterior hemiblock; right bundle branch block; a combination of right bundle branch block and left anterior hemiblock; intraventricular conduction disturbance, such as hyperkalemia; and a periinfarction block [1]. Right axis deviation occurs as the electrical axis moves toward $+90^\circ$ and $+180^\circ$ with a negative QRS in lead I and, in cases of extreme right axis deviation, the QRS is negative in both leads I and aVF. A rightward axis is normal in infants and children, and may also be present in thin adults whose hearts lie in a more vertical plane. Medical conditions associated with right axis deviation include right ventricular hypertrophy; a left posterior fascicular block, which may also be associated with other signs of inferior or posterior ischemia; chronic obstructive pulmonary disease, especially when presenting with an S1, S2, or S3; and lateral myocardial infarction [1]. The QRS duration is indicative of the time it takes for ventricular activation and should be measured in

the lead with the clearest and widest QRS complex in the frontal plane with normal being defined as <0.12 second [2].

The J point in an ECG represents the transition from ventricular activation and depolarization to the period preceding ventricular repolarization. The ST segment encompasses this period between ventricular depolarization and ventricular repolarization. The analysis of the ST segment of this specific aspect and time period of electrical activity plays a preeminent role in the analysis, definition, and detection of myocardial ischemia.

ST segment changes may reveal depression, which indicates endocardial ischemia and subendocardial injury, or they may reveal elevation, which indicates transmural ischemia, transmural injury, and infarction (Box 1). ST segment changes are reflective of local currents of injury, which may result in cells having a lower resting membrane potential, causing partial depolarization. Early repolarization may also occur from these local currents of injury and result in ST segment changes [3]. The significance of ST segment changes is defined by a change from baseline of >0.1 mV, where baseline is defined by a line connecting the beginning of the QRS complex of one beat with the beginning QRS complex of the subsequent beat for three consecutive beats on a line that is isoelectric and free of tachycardia [1]. Other causes of ST-segment changes are discussed throughout this article.

The T wave represents the period of ventricular repolarization with ventricular recovery as repolarization proceeds in the same direction as depolarization, represented by the QRS complex. Certain areas of the ventricle take longer to repolarize than to depolarize and this time differential encompasses the concept of ventricular gradient. A normal T wave has a slower ascent and a more rapid descent and, in the right precordium, a biphasic T wave that is initially positive and then negative may be seen. However, a negative initial deflection followed by a positive deflection is usually abnormal. Since the normal T-wave vector is directed leftward, anteriorly and inferiorly the T wave is usually negative in aVR and may be inverted in the right precordial leads as part of a juvenile pattern, but should be upright in the lateral precordium with upright T waves in leads I, II, V5, and V6 [1]. Since repolarization is a highly energy-dependent process, T-wave

Box 1. Causes of ST segment changes

- Myocardial ischemia
- Myocardial infarction
- Conduction changes
- Drug effects (eg. digitalis)
- Autonomic effects
- Left ventricular hypertrophy
- Measurement artifacts

changes or abnormalities reflect changes in ventricular repolarization and, as such, may be indicative of myocardial ischemia. There are other causes of T-wave changes. Cerebrovascular accidents, especially subarachnoid hemorrhage, may present with diffuse T-wave inversion putatively from a hypothalamic-mediated vagal activation. A neurogenic T-wave pattern may also be seen after vagotomy, bilateral carotid endarterectomy, or radial neck dissection. Altered ventricular activation, such as bundle branch block, Wolff–Parkinson–White, strain patterns, apical hypertrophic cardiomyopathy, preexcitation, post-tachycardia, pacing, or, in certain patients, an idiopathic global T-wave inversion, may result in T-wave changes [3].

Electrical systole is represented by the QT interval, which should be measured in the lead with most clearly defined T waves from the beginning of the QRS complex to the end of the T wave (Fig. 2). The QT interval varies with heart rate as well as the location of the heart with respect to the recording electrode, which results in the phenomenon of QT dispersal. To correct for heart rate variation, a corrected QT interval (QTc) is most commonly defined by the QT interval in seconds divided by the square root of the

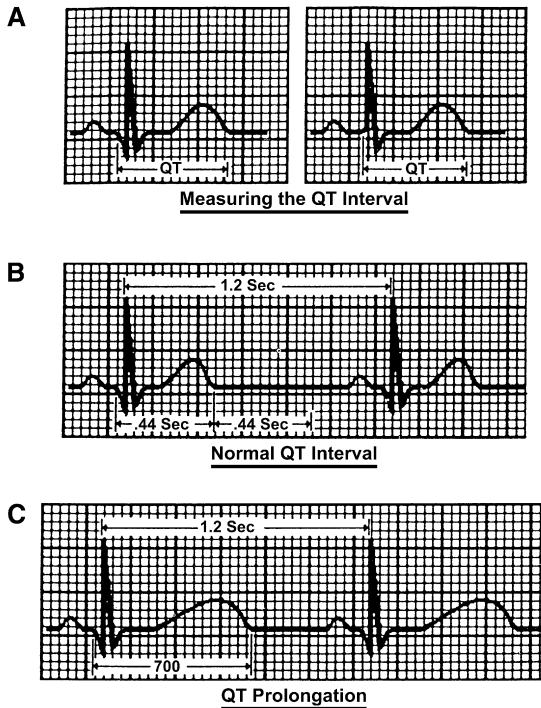


Fig. 2. Measuring the QT interval. (A) Two ECGs with QT interval measurements. (B) ECG with normal QT interval. (C) ECG showing prolonged QT interval (From Grauer K, Curry RW Jr. Clinical electrocardiography. 2nd edition. Cambridge (MA): Blackwell Scientific Publications; 1992; with permission.)

R–R interval in seconds with normal considered below 0.44 to 0.46 second [1]. The QT interval may be shortened in the presence of certain conditions, such as acidosis, hyperkalemia, and hypercalcemia, and with the use of medication, such as digoxin. A prolonged QT interval is often taken as a precursor to the onset of malignant ventricular arrhythmias and the presence of ischemia. Other causes include congenital prolonged QT syndrome, such as Jervell–Lange–Nielsen, which is associated with deafness, and Romano–Ward, which has no association with deafness. Organophosphate poisoning can produce a prolonged QT syndrome similar to congenital prolonged QT syndrome. Other causes of QT prolongation include hypokalemia, hypocalcemia, hypomagnesemia, class-IA antiarrhythmics, class-III antiarrhythmics, psychotropic medications, central nervous disorders (eg, subarachnoid hemorrhage, ruptured berry aneurysms, and cryptococcal meningitis), hypothermia, and even fad diets [1,4].

The U wave occurs in diastole in the same direction as the T wave, but with a fraction of its amplitude and is best seen in leads V2 and V3. The cause of the U wave is not certain and at times may even represent a second notching of the T wave. A large positive U wave may be due to hypokalemia or antiarrhythmic medication, whereas a negative U wave may represent ischemia, hypertension, or right ventricular hypertrophy [1,4].

Once the components of the normal ECG with the information that they convey are understood, an analysis of the ECG can be undertaken. The ECG should be analyzed in a consistent and systematic manner. The first step is to assess if all the component elements of the ECG as described above are present and, if not, to determine which components are missing. Second, the rate of ventricular conduction should be determined with the adult normal being defined as a heart rate between 60 to 100 beats per minute. Tachycardia represents a heart rate >100 beats per minute and bradycardia represents a heart rate <60 beats per minute. After the rate has been determined, the rhythm should be determined. Does a P wave precede every QRS complex? Is the patient in sinus rhythm? If not, what is the rhythm? Clinical correlation with assessment of cardiovascular perfusion should be immediately undertaken. Is there a pulse? What is the blood pressure? What is the oxygen saturation? Hemodynamically unstable rhythms should be treated emergently according to the protocols of advanced cardiac life support and with cardiopulmonary resuscitation. If the patient is hemodynamically stable, then further analysis should determine electrical axis, conduction delays and blocks, atrial and ventricular size, electrolyte abnormalities, and the presence or absence of ischemia. The most common sources of error continue to be incorrect analysis, misplaced leads, loss of leads, motion artifacts, and shivering, all of which can contribute to the misinterpretation of data and suboptimal care. Lead placement and filters have a significant impact on intraoperative echocardiography. Although 12-lead systems are available for continuous intraoperative monitoring, most systems are restricted to 5 leads (with only 1 precordial lead). The surgical incision may

also influence lead placement, which affects sensitivity and specificity required for detecting and interpreting the aforementioned abnormalities. Finally, filtering systems may be used to decrease the effects of electrocautery and movement, but may lead to distortion and incorrect interpretation of the ECG.

Conduction defects and blocks

Conduction defects or blocks may occur anywhere within the cardiac conducting pathways from the sinoatrial node to the atrioventricular node, the bundle of His, down the bundle branches, fascicle, and Purkinje system to the ventricular myocardium. The blocks commonly encountered include right and left bundle branch blocks; left anterior and posterior fascicular block; first, second, and third heart block; and atrioventricular dissociation. Conduction defects may be due to various causes with varying degrees of significance. They may also require treatment and intervention.

In first-degree (1°) atrioventricular block, there is sequential conduction from the sinoatrial node to the atrioventricular node to the ventricles, but the time required for conduction is prolonged with the PR interval being >0.20 second. First-degree atrioventricular block may be caused by an increased vagal tone; medications, such as β -blockers, calcium channel blockers, propafenone, flecainide, and amiodarone; differing diseases, including myocardial ischemia, myocardial infarction, congenital heart disease (eg, atrial septal defect), Ebstein's anomaly, infectious diseases (eg, streptococcal infection and rheumatic fever), myocarditis, infiltrative myocardial disease; and adrenal insufficiency [5]. Patients with 1° atrioventricular block usually do not require any treatment for their conduction defect. Possible exceptions are patients with heart failure and a markedly prolonged 1° block >0.30 second who may benefit from atrioventricular sequential pacing [4].

In second-degree (2°) atrioventricular block, certain atrial impulses are not conducted to the ventricles. This is designated by a ratio of P wave to QRS complexes. If there are four P waves but only three QRS complexes, this is designated as 4:3 conduction and also 4:1 block, where conduction refers to the P waves followed by QRS complexes and block refers to the number of QRS complexes that fail to follow a P wave [1]. There are two types of 2° atrioventricular block: Mobitz type I (Wenckebach) and Mobitz type II. In Mobitz type-I 2° block, there is usually a progressive lengthening of the PR interval until there is a nonconducted P wave. The QRS morphology is not widened, but remains normal and this block most commonly occurs at the level of the atrioventricular node. Mobitz type-I block may be seen with intense vagal stimulation in normal individuals during sleep and in well-conditioned athletes. It may also be seen in hyperkalemia, acute rheumatic fever, degenerative conduction disease, and calcium deposition

in the conducting system, as well as in myocardial disease. Digitalis, lithium, clonidine, methyldopa, flecainide, and propafenone, as well as calcium channel and β -blockers are associated with Mobitz type-I conduction block and should be discontinued if this block occurs. Inferior ischemia or infarction may result in type-I 2° block, which tends to resolve within hours or may last up to a week. If there is associated bradycardia and hypoperfusion, a right ventricular infarction should be excluded and appropriate antivagal therapy and resuscitation, including, on occasion, temporary pacing, may be required [4,5]. In Mobitz type-II 2° block, the PR interval tends to be constant and accompanied by a nonconducted impulse with a widened QRS complex. This is a more ominous block because the site of the conduction defect tends to be infranodal with an increased tendency to progress to complete heart block. When there is a 2:1 block, it may be hard to differentiate a Mobitz type-I block from a Mobitz type-II block. Mobitz type-I block in this situation may have an increased PR interval and narrow QRS complex and tends to decrease with increased heart rate or atropine. On the other hand, a Mobitz type-II block would be favored by a widened QRS complex and a tendency to decrease with carotid sinus massage. A Mobitz type-II block indicates significant disease of the conduction system and is associated with infiltrative diseases, such as amyloidosis, hemochromatosis; connective tissue diseases, such as lupus and scleroderma; cardiac disease; cardiomyopathy; and Chagas' disease. Also, Mobitz type-II block, when associated with an anterior myocardial infarction, signifies a large amount of myocardial damage. A Mobitz type-II block requires prompt recognition and treatment with pacemaker placement [1,3–5].

Third-degree (3°) atrioventricular block or complete heart block represents the failure of atrial impulses to activate the ventricles. The location of this block may be at the atrioventricular node or infranodal. The P–P interval and R–R interval may be regular, but atrial impulses are not conducted to the ventricle. If the conduction defect occurs at the atrioventricular node, the QRS complex tends to be narrow and the ventricular escape rhythm occurs at a rate of 40 to 60 beats per minute. If the escape rhythm is <40 beats and the QRS complex is widened, then the block tends to be infranodal. On physical examination, an S1 may vary in intensity and occasional canon *a* waves may be seen on central venous pressure examination. Medications, such as digitalis, lithium, propafenone, flecainide, methyldopa, clonidine, calcium channel blockers, and β -blockers, may cause complete heart block. Third-degree atrioventricular block may be caused by infiltrative diseases, such as amyloidosis and hemochromatosis; calcium and fibrous deposition; granulomatous diseases, such as sarcoidosis; collagen vascular disease, such as lupus and rheumatoid arthritis; infectious diseases, such as Lyme disease, Chagas' disease, endocarditis, diphtheria and syphilis; hyperkalemia; hypo- and hyperthyroidism; and valvular diseases, such as calcific aortic stenosis and mitral annular calcification. Third-degree atrioventricular block may also follow cardiac surgery. Complete 3° heart block

associated with an inferior myocardial infarction is more likely to be transient, but may take 1 or 2 weeks to resolve. By comparison, complete 3° atrioventricular block associated with an anterior myocardial infarction tends to be permanent with a worse prognosis, and requires permanent pacing. Complete heart block is often unresponsive to treatment with medications atropine and isoproterenol, and requires pacing, especially if hemodynamically unstable [1,3–5].

Atrioventricular dissociation is not synonymous with complete heart block. Rather, it is a manifestation of a primary rhythm disturbance whereby the atria are activated by one pacemaker and the ventricles are activated by a separate pacemaker. Default represents the failure or slowing of the primary pacemaker and the replacement or control of the ventricular rate by a secondary or latent pacemaker. Usurpation represents the acceleration of a secondary or latent pacemaker to take control over ventricular rate and may manifest as an accelerated idioventricular rhythm, accelerated junctional rhythm, or ventricular tachycardia. Usurpation may occur in addition to or as well as atrioventricular block. Treatment is dependent on identification of the primary rhythm disturbance and assessment of hemodynamic stability [1,3,4].

Fascicular and bundle branch blocks

The conduction system to the left ventricle consists of the left anterior fascicle, which lies anteriorly and superiorly and is supplied by the left anterior descending artery; and the left posterior fascicle, which lies posteriorly and inferiorly and is supplied by branches of both the left and right coronary arteries. A left anterior fascicular block (LAFB) is characterized by (1) a left axis deviation of the QRS complex from -30° to -90° , (2) a narrow QRS complex ≤ 0.12 second, (3) small Q waves in I, aVL or the left precordium, and (4) RS complexes in II, III, and aVF (* 1, 3, 5). It is important to exclude other causes of left axis deviation, such as Wolff–Parkinson–White syndrome, hyperkalemia, emphysema, and inferior myocardial infarction, before making the diagnosis of LAFB. The most common causes of LAFB are coronary artery disease, hypertensive heart disease, cardiomyopathy, and valvular heart disease, especially aortic valvular disease. Other causes include congenital heart disease, such as primum atrial septal defects, atrioventricular canal defects, and ventricular septal defects. Prognosis of LAFB depends on the severity of the underlying heart disease. A left posterior fascicular block (LPFB) is a rarer finding and is characterized by (1) right axis deviation of the QRS complex to $+90^\circ$ to $+140^\circ$, (2) a narrow QRS complex ≤ 0.12 second, (3) Q waves or QR pattern in inferior leads II, III, and aVF, and (4) RS pattern in the lateral leads I and aVL. Other causes of right axis deviation, such as a lateral myocardial infarction, chronic obstructive pulmonary disease,

and right ventricular hypertrophy, must first be excluded. The most common cause of LPFB is coronary artery disease with hypertensive heart disease. Cardiomyopathy and aortic valvular disease are other common causes of LPFB. As with LAFB, the prognosis of LPFB depends on the underlying heart condition and no specific treatment is required for either LAFB or LPFB [1,3–5].

Right bundle branch block (RBBB) occurs when the right bundle is blocked and the left ventricle is depolarized normally with the electrical impulses to depolarize the right ventricle spreading from the left ventricle. RBBB is characterized by (1) a wide QRS complex ≥ 0.12 second, (2) an RSR' pattern in the right precordium leads V1 and V2, (3) a wide S wave in the left lateral precordium V5, V6, I, and aVL [1,3–5]. RBBB sometimes occurs in patients without heart disease and, for these patients, there is no adverse prognosis. Most patients with RBBB, however, have heart disease, including coronary artery disease, hypertensive heart disease, cor pulmonale, myocarditis, Chagas' disease, cardiomyopathies, degenerative disease of the conduction system, and sclerosis of the cardiac skeleton. For those patients with RBBB presenting with an acute myocardial infarction, there is a markedly increased mortality. Patients with the Brugada syndrome—RBBB with ST segment elevation in the right precordium—have an increased risk of ventricular tachycardia and sudden death [3]. RBBB usually requires no specific therapy.

Left bundle branch block (LBBB) is usually indicative of significant cardiac disease and, in the setting of an acute myocardial infarction, is associated with a significantly increased risk of mortality. LBBB is characterized by (1) widened QRS complex ≥ 0.12 second, (2) broad, notched and slurred R waves in the lateral precordium V5 and V6 as well as I and aVL, (3) small or absent R waves in the right precordium V1 and V2 with deep S waves, (4) absent septal Q waves in the left-sided leads, and (5) delayed intrinsicoid deflection in V5 and V6 [1,3–5]. The presence of LBBB makes the diagnosis of an acute myocardial infarction, especially lateral and inferior myocardial infarction, more difficult. LBBB is usually the result of structural heart disease due to ischemia, cardiomyopathy, advanced valvular disease, inflammation, or degeneration. In the presence of significant structural disease or bilateral bundle branch block, pacing is warranted [1,3–5]. Dorman and colleagues [6] evaluated the prognostic significance of bundle branch block and found that it is associated with co-existing cardiovascular disease, such as hypertension, coronary artery disease, and heart failure. The investigators also found that LBBB may be associated with a decreased response to stress, but that bundle branch block alone is not an independent risk factor for cardiac complication. Rather, bundle branch block should alert to the possibility of co-existing cardiovascular conditions.

The gold standard for the determination of atrial and ventricular size is the echocardiogram. Nevertheless, the ECG still provides useful information on atrial and ventricular size. Right atrial enlargement or abnormality

is characterized by (1) a peaked P wave in lead II >0.25 mV, (2) P wave in leads V1 or V2 >0.15 mV, and (3) a rightward shift of the P-wave vector to more than $+75^\circ$. The right atrium contributes to the early component of the P wave, which manifests as an increase in amplitude. Signs of right atrial enlargement are often found in association with signs of right ventricular enlargement. P pulmonale is usually described as a rightward shift of the P-wave vector and peaked P waves in leads II, III, and aVF. P pulmonale is often seen in congenital heart lesions, such as tetralogy of Fallot, Eisenmenger syndrome, tricuspid atresia and pulmonic stenosis, and has a stronger correlation with decreased arterial saturation than right atrial enlargement. P pulmonale may also be found in association with left atrial enlargement, tachycardia, and angina [1,3]. Left atrial enlargement is characterized by (1) a broad notched P wave with a duration of >0.12 second in lead II (resembling an M), (2) a leftward shift of the P-wave vector to $+15^\circ$ to -30° , (3) an increased terminal negative portion of the P wave in lead V1, resulting in a biphasic P wave in lead V1 that is negative for >1 mm in depth, with a duration of >0.04 second. An enlarged left atrium manifests with an increase in the duration of the P wave, which correlates with an increase in left atrial volume. Valvular heart disease, especially mitral stenosis, is associated with this pattern of left atrial enlargement, and patients with increased left atrial size have a higher likelihood of developing paroxysmal atrial tachycardia [1,3].

The right ventricle has much less mass than the left ventricle and even with right ventricular hypertrophy (RVH) the electrical manifestations on the ECG may not be as apparent. RVH may present with (1) significant right axis deviation of $>90^\circ$, as long as other causes, such as RBBB, lateral myocardial infarction, and left posterior fascicular block, can be excluded, (2) tall R wave, small S wave, and increased R/S ratio in lead V1 with $R >0.7$ mV in V1, and R/S in V1 >1 , as long as Wolff–Parkinson–White, posterior infarction, normal young-adult variation, and isolated left ventricular hypertrophy can be excluded, (3) QR pattern in V1, which may be present only in severe RVH, (4) deep S wave, small R wave, R/S ratio <1 in leads V5 and V6 (However, deep S wave, small R wave, R/S ratio <1 in leads V5 and V6 may also be present in patients with LAFB, anterior myocardial infarction, or chronic obstructive pulmonary disease), (5) ST segment depression and T-wave inversion in the right precordium and also II, III, and aVF (A negative–positive biphasic T wave may also be seen with RVH.), and (6) S1, S2, and S3, which also may be seen in emphysema and occasionally in normal individuals [1,3].

Even though an echocardiogram allows better visualization of the myocardium, the presence of left ventricular hypertrophy on the ECG identifies a subset of the general population at a significantly increased risk for myocardial morbidity and mortality, especially if there are any ST-T-wave abnormalities and particularly for women [3]. Although many criteria are available for determining left ventricular hypertrophy, including the

Sokolow–Lyon index, the Romhilt–Estes point system, and the Cornell voltage criteria, currently cited voltage criteria include the following:

- Largest R wave + largest S wave in the precordial leads >4.5 mV
- S in V1 + R in V6 >3.5 mV
- S in V2 + R in V6 >4.3 mV
- S in V1 >2.5 mV
- R in aVL >1.3 mV [1,3].

Noordzij and colleagues [7] hypothesized that, in the presence of multiple cardiac-risk indices, the routine use of electrocardiography may not be of value in providing prognostic information. Rather, electrocardiography may be redundant. In a retrospective examination of a 10-year period (1991–2000) encompassing over 23,000 patients and 28,000 surgical procedures where patients had been classified by type of surgery, cardiovascular risk factors, and preoperative electrocardiography, the prognostic value of electrocardiography was assessed. Abnormal ECG changes were defined as atrial fibrillation, bundle branch block (left or right), left ventricular hypertrophy, premature ventricular complexes, paced rhythms, Q waves, or ST changes. The investigators determined that those patients with an abnormal ECG, compared with those patients with a normal ECG, had an increase risk of cardiovascular death (1.8% versus 0.3%), and the addition of ECG findings to cardiovascular risk indices improved the predictive value of the index. The prognostic value of the ECG was more beneficial for patients undergoing intermediate and high-risk procedures than for those undergoing low-risk procedures [7].

Electrolyte, thyroid, temperature effects

Although sodium plays an important role in electrical activity, it does not produce consistent or significant effects on the ECG. Hypernatremia does prolong the cardiac action potential. In the setting of hyperkalemic conduction disturbances, hypernatremia shortens the duration of the QRS complex, while hyponatremia prolongs the duration of the QRS complex [1,3,4].

Potassium is the electrolyte most often associated with distinctive ECG changes. Hyperkalemia may be mild, moderate, or severe with varying or progressive ECG changes. Mild to moderate hyperkalemia is characterized by a narrowing and peaking of the T wave, while the QRS complex remains normal. Moderate to severe hyperkalemia is characterized by a flattening of the P wave or even loss of the P wave; the widening of the QRS complex, which becomes less distinct; the possible elevation of the ST segment, especially in the right precordium V1 and V2; an increasing PR interval; and the possible development of 1° and 2° blocks. Severe hyperkalemia may present with a nondiagnostic ECG, or may show the progressive widening of the QRS complex, which may resemble wide complex ventricular tachycardia

or even a sine wave before the onset of ventricular fibrillation or asystole. Failure to appreciate the significance of hyperkalemia may lead to inadequate treatment, inappropriate treatment, and loss of life [1,3,4]. Hypokalemia promotes the development of both ventricular and supraventricular ectopy, which may lead to ventricular tachycardia, torsades de pointes, or ventricular fibrillation. Rapid onset of hypokalemia may occur with intense sympathetic stimulation from epinephrine and may also occur with the administration of non-potassium-sparing diuretics or sodium bicarbonate. ECG manifestations of hypokalemia are due to abnormal and delayed repolarization and are manifested by (1) ST segment depression ≥ 0.5 mm, (2) an increasing U-wave amplitude, (3) a decreasing T-wave amplitude with the U-wave amplitude being greater than the T wave, and (4) an unchanging QRS duration [1,3,4].

Calcium affects the duration of the plateau phase of the action potential (ie, phase two of the action potential) and manifests as changes in the duration of the ST segment and the QT interval. Hypercalcemia shortens the duration of the action potential. The ST segment becomes shorter or may even disappear. The QTc also shortens and occasionally the QRS duration may increase. The morphology of the P wave, T wave, and U waves remains the same, but occasionally the U-wave amplitude may slightly increase. Hypercalcemia may cause 2° and 3° atrioventricular block and severe hypercalcemia may cause sudden death from ventricular fibrillation. However, for the most part, arrhythmias tend to be uncommon with hypercalcemia [1,3,4]. Hypocalcemia manifests when ST segments and the QTc also become prolonged. With severe hypocalcemia, the T wave may become flat or inverted [1,3,4].

Magnesium blocks the calcium channel and modulates the effects of potassium currents. Severe hypermagnesemia depresses the atrioventricular node and causes intraventricular conduction delays that may lead to complete heart block and cardiac arrest. Hypomagnesemia is associated with hypokalemia and hypocalcemia and may be associated with ventricular arrhythmias, reduced T-wave amplitude, and ST depression. Whether these associations stem from magnesium or a deficiency of other electrolytes remains uncertain [1,3,4].

Thyroid disorders present with ECG changes. Hyperthyroidism often manifests with sinus tachycardia or atrial fibrillation before other clinical signs and diagnoses. The P wave and QRS complex often have increased amplitude and may simulate left ventricular hypertrophy. Nonspecific ST-T-wave changes, which are fluctuating, may be seen. The fleeting nature and day-to-day variations of the hyperthyroid state cause these fluctuations, which are more extreme than those seen in pericarditis or myocarditis. The PR interval tends to be prolonged and 1° atrioventricular block may be present, but 2° and 3° atrioventricular block are much less common [1,3,4]. Successful treatment of the hyperthyroid state tends to resolve these ECG changes. Hypothyroidism, especially myxedema, may present with

sinus brachycardia; low P-, QRS-, T-wave voltage; QT prolongation; flat or inverted T waves, ST-T-wave changes similar to those associated with pericarditis; and a high incidence of atrioventricular and intraventricular conduction defects, leading to torsades de pointes. These changes respond to treatment of the hypothyroid state [1,3,4].

Temperature changes are also associated ECG changes. Hyperthermia produces a hypermetabolic state with increased catecholamine stimulation, tachycardia, and, possibly, an increased risk for ventricular fibrillation and ventricular tachycardia [1]. As hypothermia develops, the duration of the action potential lengthens with profound hypothermia affecting both depolarization and repolarization. The J-Osborn wave represents an elevation of the J point and early ST segment. The lower the temperature, the more prominent the J-Osborn wave. As the temperature drops and hypothermia becomes more pronounced, atrial fibrillation develops, J-Osborn waves may be seen, with ventricular fibrillation and cardiac standstill occurring with profound hypothermia [1,3,4].

Alternans represents the regular alternating of the electrical pattern as noted on the ECG. Sinus tachycardia and total electrical alternans is often seen with pericardial effusion and cardiac tamponade. Sinus rhythm with heart rate alternans (R-R alternans) may be seen in congestive heart failure. ST segment alternans is associated with vasospastic angina, ischemia, and subarachnoid hemorrhage. P-wave alternans, though rare, may be noted with pulmonary embolism. QRS alternans with tachycardia usually represent reentrant atrioventricular nodal rhythms. T-wave alternans with QT prolongation may precede torsades de pointes [1,3].

Ischemia

The ECG is a key component in the diagnosis of ischemia. Changes in the ST segment caused by ischemia may result from currents of injury, which can lower resting-membrane potential, decrease the velocity and amplitude of the action potential, and cause early repolarization by decreasing the duration of the action potential. ECG changes that are considered significant are ST segment elevations or depression > 1 mm from baseline. Other ECG abnormalities, such as LBBB, left ventricular hypertrophy with strain, and conduction abnormalities may obscure ST segment changes. ST segment changes may present with reciprocal changes. That is, ST segment elevation with reciprocal ST depression and ST segment depression with reciprocal ST elevation. Ischemic changes to specific areas may present ECG changes in specific leads: extensive anterior I, aVL, V1 through V6; anterior septal V1 through V3; anterior apical V3 and V4; anterior lateral V4 through V6; high lateral I, aVL, V5 and V6; posterior reciprocal changes in V1; and right ventricular V4R, V1, V2, and V3 [1,3,4].

The accuracy as well as the sensitivity and specificity of the information obtained from intraoperative ECG monitors depends on a variety of factors,

including mode, filters, template and lead selection. The work of Mangano and colleagues [8,9] showed that visual detection of ST segment changes is poor, below 50%. Leung and colleagues [10] demonstrated that ST segment trend analysis available in intraoperative monitors has an average sensitivity of up to 78% and specificity of almost 90%. The narrowing of the bandwidth in the monitoring mode and the use of filters to minimize artifact from poor electrode contact and mechanical ventilation may adversely affect the accuracy of ST segment trend analysis [11,12]. Each monitor manufacturer has a proprietary algorithm for determining elevations and depression and these vary despite recommendations from the American Heart Association for standardization. The computer sets the isoelectric and J-points by default and if the computer-chosen set points are inaccurate, then the calculated ST segment analysis will be inaccurate. Fleisher [11], therefore, recommends that the computer-chosen template be displayed and reviewed before the start of each procedure to ensure the accuracy of ST segment analysis, and, if these are significant ST segment changes, to print the ECG strip for detailed analysis. The work of London and colleagues [13] verified that the combination of leads II and V5 detected 80% of significant ST segment changes and, with the addition of V4, sensitivity was increased to 96%. Recently, Landesberg and colleagues [14] reexamined optimum lead selection and found that lead V4 had 83% sensitivity versus 76% sensitivity for lead V5 in detecting significant prolonged ischemia, and, when two precordial leads were used, there was a 95% sensitivity in detecting significant perioperative ischemia (Fig. 3).

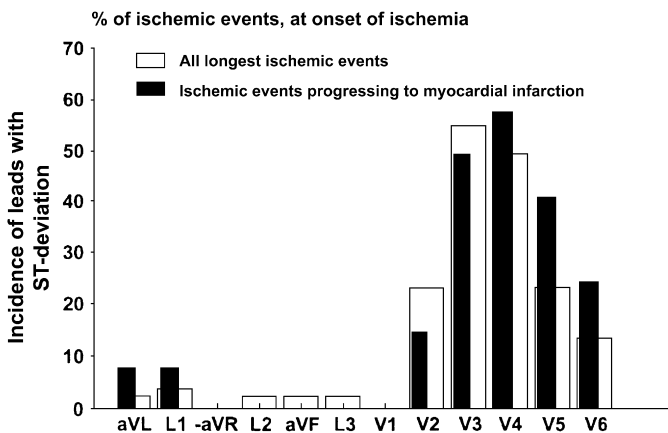


Fig. 3. The incidence of ST segment deviation by ECG lead in patients undergoing vascular surgery. Lead V4 is the strongest predictor of perioperative myocardial infarction. (From Landesberg G, Mosseri M, Wolf Y, et al. Perioperative myocardial ischemia and infarction: identification by continuous 12-lead electrocardiogram with online ST-segment monitoring. *Anesthesiology* 2002;96:264-70; with permission.)

With the advent and widespread use of echocardiography, many have questioned the need for ECG analysis. When using trained, skilled, and experienced cardiac anesthesiologists, Berquist and colleagues [15] found that real-time echocardiographic detection of myocardial ischemia had a sensitivity and specificity of approximately 76%. According to the American College of Cardiology/American Heart Association Task Force on Practice Guidelines, perioperative transesophageal echocardiography is still considered only a category-II indication for the detection of myocardial ischemia [16]. Even now echocardiography requires advanced training and expensive equipment not always routinely available. Also, because probe insertion occurs after the induction of anesthesia, echocardiography is often not available during the crucial period for ischemic detection. By comparison, the ECG is a routine monitor that provides a wealth of information and a high degree of sensitivity and specificity in ischemic detection.

Since the duration of ST segment changes correlates with the incidence of perioperative myocardial infarction in those at risk, prompt verification by obtaining a printed ECG recording and institution of therapy are warranted for optimal patient care.

The relationship between ST segment changes and perioperative myocardial ischemia developed from the work of Slogoff and Keats in 1985. This work noted the relationship between ST segment changes and perioperative myocardial infarction [17]. Using Bayes theorem, which states that the post-test probability is a function of pretest probability, Fleisher [11] notes that, in patients at risk for cardiac ischemia, the likelihood that significant ST segment changes correlate with ischemia is high. Cardiac-risk indexes continue to be validated as demonstrated by multiple studies, including the recent work by Boersma and colleagues [18]. The appropriate use of cardiac-risk indexes aids in the determination of patient risk in the perioperative setting, assessment of ECG information, and optimal patient care.

Implications

The ECG is a valuable tool in monitoring the patient. Information presented on an ECG should be analyzed systematically with an understanding of the constituent elements of an ECG, the rate, the rhythm, the morphology, the axis, the presence of conduction abnormalities, electrolyte changes, and ischemic changes. To accurately assess the information presented, hemodynamic information and cardiac-risk analysis should be integrated to have a complete picture. An RBBB is associated with a higher mortality and increased risk of cardiac events in those with a prior myocardial infarction or abnormal dobutamine stress echocardiogram [19]. Biagini and colleagues [19] found that a complete RBBB was as equally predictive as an LBBB for increased mortality. The presence of an LAFB is an independent predictor of mortality in those at risk for coronary artery disease, even if they have not had a prior myocardial infarction [20]. In patients with coronary artery disease

and ischemic cardiomyopathy, analysis of QT depression helps determine the amount of viable hibernating myocardium. That is, those with preserved QT depression have viable myocardium available, but those with high QT dispersion have nonviable scar tissue [21]. For patients with heart failure and an LBBB who have biventricular pacing, an analysis of the ECG can reveal, with a sensitivity and specificity of over 90%, if there is loss of resynchronization therapy. Lead V1 is analyzed and if the R/S ratio ≥ 1 , then there is left ventricular capture and biventricular pacing. If this is not the case, then lead I is assessed and if the R/S ratio is < 1 , then there is left ventricular capture. If the R/S ratio > 1 in lead I, then there is right ventricular capture and loss of biventricular pacing. The algorithm allows physicians to analyze an ECG and evaluate the presence of biventricular pacing with a high degree of sensitivity and specificity even without a programming device [22]. Thus the wealth of information available through a thorough and accurate ECG analysis is enormous.

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