3 Electrocardiography

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This chapter covers basic information about the different aspects of the standard electrocardiogram (ECG) in adult clinical cardiology. Arrhythmias, including monogenetic forms, conduction disturbances, and ECG findings in congenital cardiology are discussed elsewhere in this book.

Electrocardiography, more than 100 years after its invention by Einthoven, remains one of the most frequently used bedside tools for the evaluation of the cardiac patient. Its old age does not imply that electrocardiography has become a rusty static science. On the contrary, along with the development of new pathophysiologic concepts, electrocardiography was reevaluated, resulting in new insights as to the use of the ECG in current daily practice. Examples of such developments are new insights into cellular electrophysiology and cardiogenetics, leading to the definition of new ECG syndromes, such as the Brugada syndrome and better understanding of existing syndromes such as the long QT syndrome. Availability of new imaging techniques has facilitated revisiting electrocardiographic–anatomic correlations. The advent of possibilities to reopen a coronary artery, either by thrombolytic therapy or by percutaneous coronary intervention, has led to new information in the ECG regarding ischemia and infarction, as to the site of occlusion within the coronary system and the area at risk, and to noninvasively diagnose reperfusion of the ischemic tissue. Other features of electrocardiography are its easy and repeated applicability. This enables, better than any other technique, the study of the dynamic behavior and natural history of cardiac diseases.

This chapter discusses new findings in standard electrocardiography and the use of the ECG to describe the dynamicity of cardiac disease.

Electrical Activation of the Heart

The pump function of the heart is accomplished by electrical activation of the myocardium. This process occurs through depolarization of cells, aimed at (1) driving the heart action (automaticity), (2) conducting the electrical impulse, and (3) initiating contraction.

Depolarization and Repolarization

The basic electrophysiologic action is depolarization of cells that are in a state of polarization. The amount of polarization differs between automatic cells and conducting and contracting cells. This process is driven by different ion currents, with sodium, calcium, and potassium being the most important ones. Sodium is the ion used for depolarization, calcium follows thereafter to initiate and maintain contraction, and potassium is needed to repolarize the cell. During the state of polarization sodium concentration is high outside and potassium concentration is high inside the cell. This is phase 4 of the action potential. During depolarization, sodium enters the cell quickly (phase 0); after a slight repolarization (phase 1), the plateau phase is reached, during which calcium influx occurs (phase 2). Finally repolarization occurs through potassium currents (phase 3). The equilibrium of ion concentrations is restored by energy-dependent ion pumps, such as the sodium/calcium exchanger and the sodium/potassium pump.

Automaticity

Automatic cells are less polarized than the other cardiac cells and have the ability of spontaneous depolarization during the resting phase 4. This leads to spontaneous de- and repolarization, allowing automatic activity. These cells are located within the sinus node, the atrioventricular (AV) node, and the specific distal conduction system. Differences in speed of phase 4 depolarization lead to a hierarchical organization, allowing the sinus node to dominate the heart rhythm. The other potential pacemakers are depressed by the faster activation rate, a phenomenon, known as overdrive suppression. In cases of failure of the dominant pacemaker or conduction block, the secondary pacemakers become active, frequently after a pause, and prevent in this way the heart from asystole. The ensuing rhythm is called an escape rhythm.

Conduction

Depolarization of a cardiac cell is propagated to a neighboring cell. In this way propagation of the electrical impulse occurs, resulting in activation of the whole heart.

Excitation of the cells spreads along myocardial cells or specifically conducting fibers, such as the Bachmann bundle in the atria and the His–bundle–branch–Purkinje system in the ventricles. After atrial activation the impulse passes through the AV node. Here the impulse is slowed down, allowing time for the blood to pass to the ventricles. Also the AV node has the property of decremental conduction and block at higher rates, preventing impulses from passing to the ventricles in cases of atrial tachycardias, such as atrial fibrillation. In this way the ventricles are protected in cases of high-imposed rates. Once the impulse has passed the AV node, conduction through the distal conduction system, that is, the His bundle, the right (RBB) and left bundle branch (LBB), and the Purkinje network, is fast and in an all-or-none fashion. These properties secure prompt and synchronous activation of both ventricles.

The P-QRS-T-U Complex

Electrocardiography is the graphic representation of cardiac electrical activity, registered at the body surface. Cardiac activation starts with discharge of the sinus node; the electrical activity spreads over the atria, and passes to the ventricles through the AV node, the His bundle, and the bundle branches. Sinus node activity and conduction through the specific conduction system are electrically silent. Therefore, the basic electrocardiogram consists of a P wave as the result of atrial activation, followed by an isoelectric PQ interval during conduction through the specific conduction system, the QRS complex because of ventricular myocardial depolarization, the isoelectric ST segment during homogeneous and simultaneous myocardial activation, and the T wave as the consequence of repolarization of ventricles (Fig. 3.1). The T wave has the same polarity as the QRS complex because the sequence of myocardial repolarization is reversed. The T wave is followed by a small final deflection with a similar polarity known as the U wave.

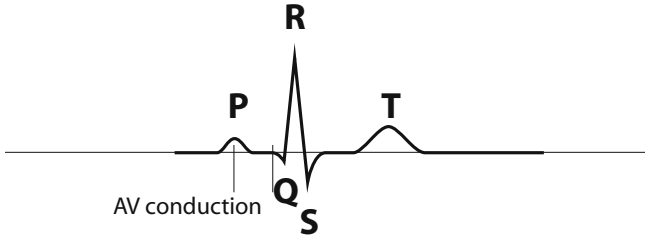

FIGURE 3.1. The basic electrocardiogram (see text).

Recording the Electric Activity

Electrode Leads

The electric activity of the heart is recorded from the body surface using electrode leads. These consist of a positive pole, connected to either a negative pole (bipolar leads) or a reference (zero) pole (unipolar leads). An electrical activation front with the main direction toward the positive pole, records an upward deflection (Fig. 3.2A). An activation front going in a direction opposite to the positive pole, is recorded as a downward deflection (Fig. 3.2B), and activation perpendicular to this pole is electrically silent (Fig. 3.2C).

Factors Influencing the Amplitude of Electrocardiogram Deflections

An activation front directly toward the electrode results in a larger amplitude than an activation front with an angle to the + pole (Fig. 3.3). Larger amplitudes will also be recorded in cases of increased wall thickness, such as in myocardial hypertrophy, because of the larger activation front (Fig. 3.4A,B) or with the electrode located closer to the myocardium (Fig. 3.4C).

Similarly, smaller voltages than usual can be observed and may include the total P-QRS-T complex (Table 3.1). This is seen (1) in cases of an electrode more distant to the myocardium (Fig. 3.5B)); (2) in conditions affecting both atrial and

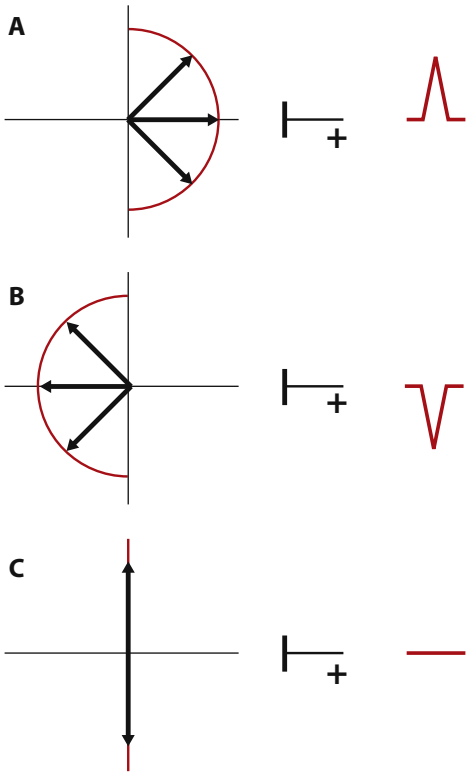

FIGURE 3.2. (A) Any electrical activity in the hemi-segment directed toward the positive pole is recorded as an upward deflection. (B) Any electrical activity in the hemi-segment directed away from the positive pole is recorded as a downward deflection. (C) Wave fronts perpendicular to the + pole are electrically silent.

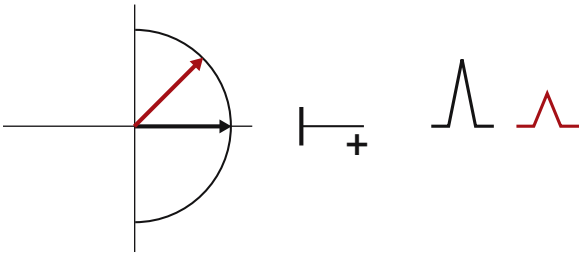

FIGURE 3.3. The electrode depicts a larger amplitude when the activation front is directed right toward the + pole, than in case of an angle with this electrode.

TABLE 3.1. Low-voltage ECG

Low voltage	*Extremity leads*	*Precordial leads*
Global cardiac muscle loss	+	+
Insulation myofibrils		
Pericardial fluid		
Pleural fluid		
Emphysema		
Obesity		
RV dilatation	+	–

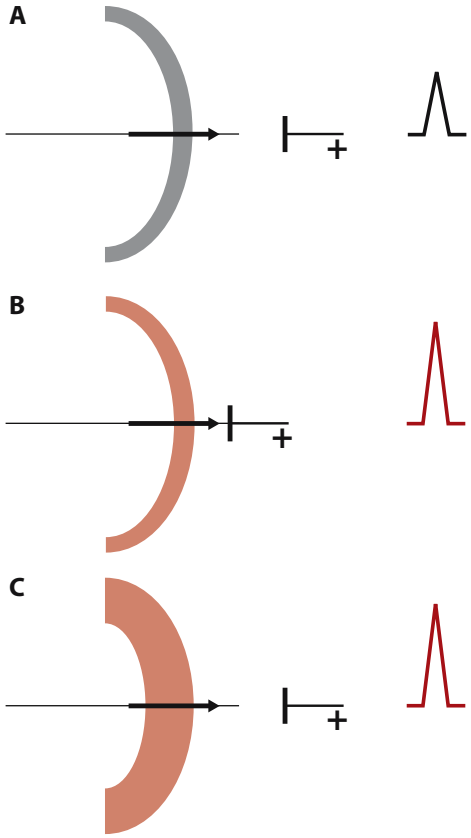

FIGURE 3.4. The voltage (A) will be higher when the electrode is closer to the myocardium (B) or when the myocardial wall thickness is increased (C).

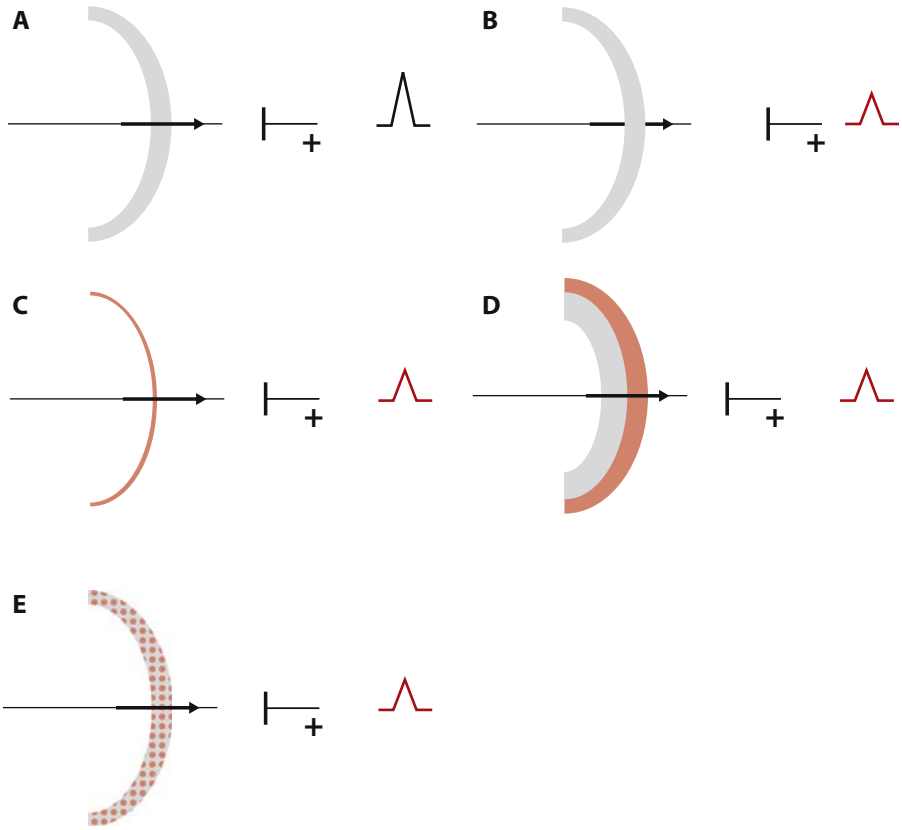

FIGURE 3.5. Mechanisms to induce low voltage ECG. (A) Normal situation. (B) Electrode more distant to myocardium. (C) Decrease in myocardial wall thickness. (D) Interposed tissue or substance between myocardium and electrode. (E) Storaged substance within the myocardium causing electrical insulation.

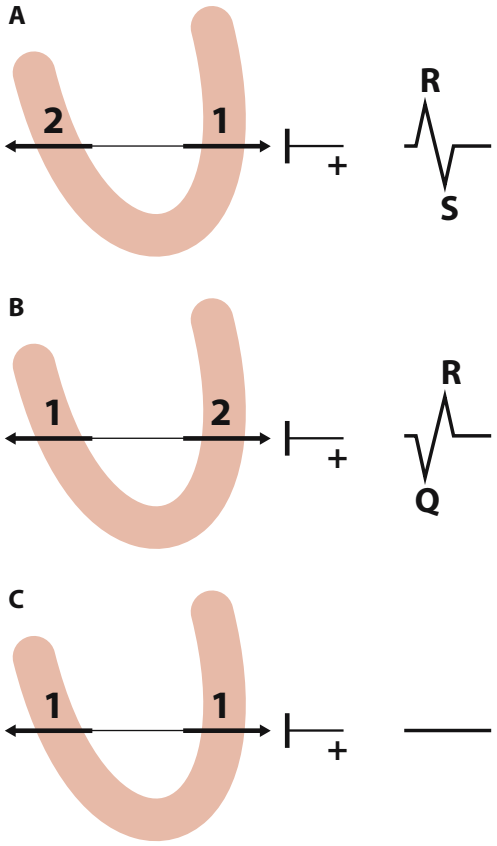

FIGURE 3.6. Activation sequence and resulting ECG configuration. See text.

ventricular myocardium, such as in myocarditis, and dilated cardiomyopathy, where loss of myocardial wall thickness plays a role (Fig. 3.5C); (3) in the presence of extracardiac interposing tissue, such as in obesity, pleural or pericardial effusion, increased air content, for instance, in emphysema, or pneumothorax (Fig. 3.5D); and (4) in cardiac amyloidosis or storage diseases, where myocardial cells are insulated by nonconductive material (Fig. 3.5E). Low voltage restricted to the QRS complex in the extremity leads, and thus with normal voltage in the precordial leads, is seen in cardiac dilatation, especially of the right ventricle (RV). This is the result of increased intracardiac volumes (Brody effect[1]) and/or rotation of the dominant vector perpendicular to Einthoven's triangle.

Changes in Electrocardiogram Deflections Due to the Activation Sequence

Electrical activity recorded from global structures such as cardiac compartments are the result of the direction, force, and timing of activation of the myocardial walls (Fig. 3.6). Consecutive activation of the proximal vs. the distal wall will lead to a positive/negative deflection, in cases of ventricular activation in an RS complex (Fig. 3.6A). In contrast, activation of the distal wall first and subsequently the proximal wall will lead to a QR complex (Fig. 3.6B). Activation of both walls simultaneously results in cancellation of both forces, resulting in no deflection (Fig. 3.6C).

Lead Systems

The Standard 12 Electrocardiogram

To enable the heart to be viewed in a standardized way and from different sites, lead systems were developed. The first three were the three bipolar leads I, II, and III, based on Einthoven's triangle. Lead I measures the potential differences between the left arm and the right arm, lead II between the right arm and the left foot, and lead III between the left arm and the left foot. A simple mathematic relation exists among these three leads: At every instant during the cardiac cycle the potential in lead II equals the sum of the voltages in leads I and III: I + III = II. This basic relation is Einthoven's law. These bipolar limb leads were followed by the augmented unipolar limb leads, aVR, aVL, and aVF, aimed at deriving more local information from different parts of the heart. These leads were constructed by using one of the limb electrodes as the positive pole and the combined other two as the reference electrode. Lead aVR equals the potential difference between the right arm and the reference potential, which is the mean of the potentials of the left arm and the left leg. Lead aVF equals the potential of the left foot minus the mean of the potentials of the left arm and right arm, and lead aVL records the potential difference between the left arm and the mean potential of the left leg and the right arm. These three bipolar and three unipolar limb leads form the hexaxial lead system in the frontal plane (Fig. 3.7A,B).

Subsequently, precordial leads V_1, V_2, V_3, V_4, V_5, and V_6 were developed to obtain information in the transversal

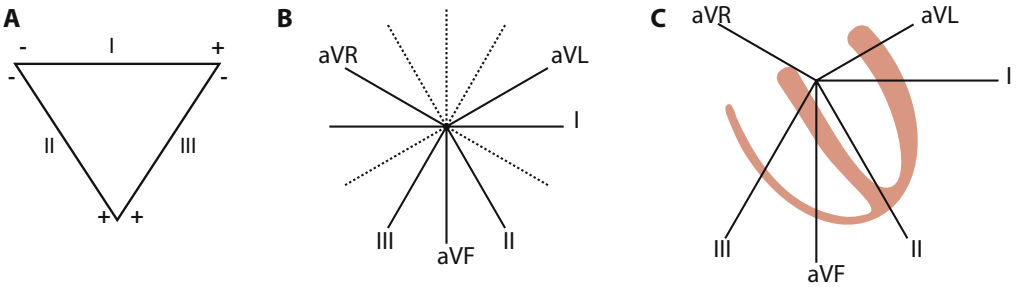

FIGURE 3.7. Electrode lead system in the frontal plane. (A) Einthoven's triangle. (B) Hexaxial lead system positioned within the ventricles with the electrical center at the base of the left ventricle (C).

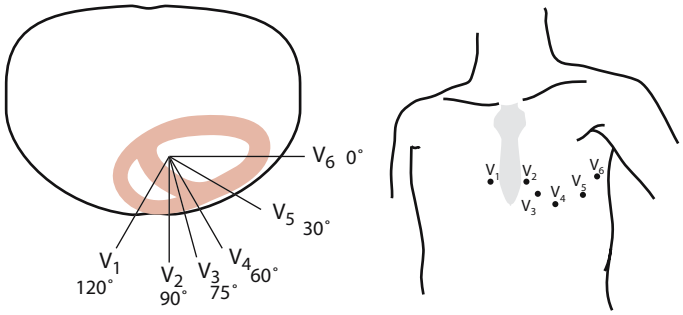

FIGURE 3.8. Precordial leads. Placement on the chest and position to the ventricles in the transverse plane.

plane. These leads have their positive pole at a specific precordial site and the combined limb electrodes as a reference electrode. The most common system is the Wilson central terminal, which consists of inputs from three limbs (right arm, left arm, and left leg) connected through 5000-Ohm resistors. In this way six precordial leads are constructed positioned from the right (lead V_1) to the left lateral side (Fig. 3.8). Leads V_1 and V_2 are located more superiorly on the chest in the four intercostal space, lead V3 is midway between leads V_2 and V_4, and leads V_4 to V_6 are in the same transversal plane with V_4 being in the fifth intercostal space. It should be realized that the plane formed by the six precordial leads is not in an exactly transverse direction.

The frontal leads are more at a distance from the heart, whereas the precordial leads are located closer to the myocardium. This allows the potential in the frontal plane to be analyzed preferably according to a vectorial approach (single dipole model). The potentials recorded in the precordial leads are determined not only by the global cardiac activation but also by local events close to their exploring electrode. Therefore, the analysis fits also with the multiple dipole array model.[2]

Additional Leads

Apart from the standard 12 leads, additional leads are frequently used. Right precordial leads V_3R to V_6R are placed opposite to the regular precordial leads (Fig. 3.9). The most frequently used lead is V_4R, which is particularly useful to record right-sided processes such as right ventricular infarction. Less frequently, leads V_7 at the left posterior axillary line, V_8 at the left midscapular line, and V_9 left paravertebrally, all at the V_6 level, are used, usually to diagnose posterior wall infarction.

The Format of the 12-Lead Electrocardiogram

The 12-lead ECG is mostly recorded in an order according to their historical sequence of development, that is, first leads I, II, and III, thereafter the augmented limb leads aVR, aVL, and aVF, and finally the precordial leads. Especially regarding the frontal plane, a more logical order would be to use the sequence aVL, I, –aVR, II, aVF, and III. Because this way of representing the leads also has disadvantages, in the following discussion the traditional representation is used. All recordings are taken at a paper speed of 25 mm/s and at a calibration of 10 mV/cm.

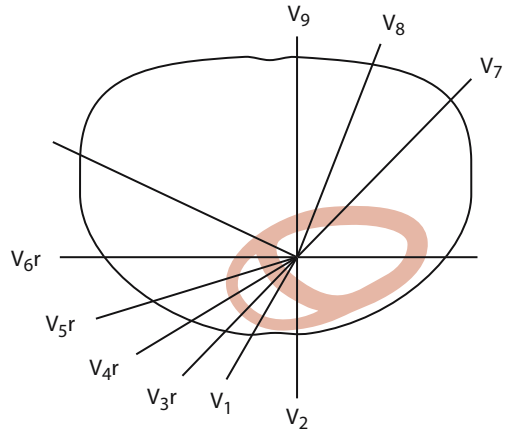

FIGURE 3.9. Additional leads. Right precordial and left posterior leads.

The Normal Electrocardiogram

The P Wave

Atrial activation starts from the sinus node, which is located at the right superior side in the right atrium. Therefore, the right atrium is activated first and from right superior to inferior. Thereafter the left atrium is activated, which is a left posteriorly located structure. The normal P wave is therefore in the frontal plane, and is usually positive in I and II and negative in lead aVR, with an axis in the frontal plane between 0 and 90 degrees (Fig. 3.10). In the transversal plane lead V_1 records initial positivity during right atrial activation and thereafter negativity during left atrial activation. Lead V_6 records positive deflections throughout right and left atrial activation.

The height of the P wave normally does not exceed 2.5 mm and its duration of 110 ms. Above that latter value conditions are present such as left atrial enlargement, intraatrial fibrosis, or the use of medication, slowing intraatrial conduction.

The PQ Interval

The PQ or PR interval consists of the time of atrial activation, for conduction through the AV node, and the distal

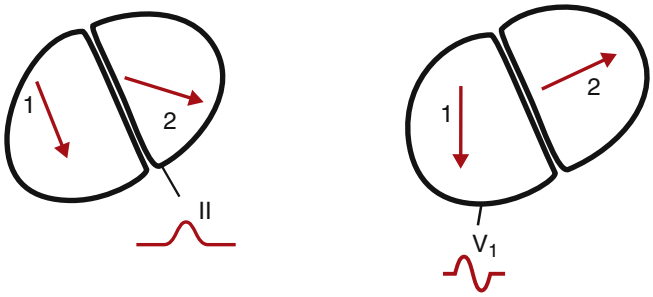

FIGURE 3.10. Left and right atrial activation and resulting P wave configuration in the frontal and transverse plane.

conduction system, the latter comprising the His bundle, bundle branches, and Purkinje network, until the ventricular myocardium becomes activated. The AV node starts to be activated about midway through the P wave. Atrioventricular nodal conduction is characterized by its slowness (allowing time for the blood to pass from the atria to the ventricles) and long refractory period. The latter is useful to prevent fast atrial activity (such as atrial fibrillation or flutter) to reach the ventricles. Conduction slowing is decremental, implying that the higher the imposing rate the more the conduction will be impaired. Conduction time through the AV node is at normal rates between 100 and 130 ms.

In contrast, conduction through the distal conduction system is fast and because of the absence of decremental conduction properties, the impulse traverses in an all-or-none fashion, securing prompt and synchronous activation of both ventricles. Conduction time through the distal conduction system is between 35 and 55 ms.

The total PR interval is usually between 120 and 200 ms. A PR interval above this value is called prolonged conduction and is due to a number of conditions such as ischemia, fibrosis, myocarditis, the use of medication, etc. A prolonged PR interval should not be called first-degree block because the impulse is not blocked but only delayed in conduction.

The QRS Complex

The QRS configuration is determined by the activation sequence and contribution of both ventricles. Under normal circumstances the thin-walled right ventricle contributes little to the dominant activation fronts and therefore to the QRS configuration. Activation mapping of the human heart has revealed that the first structure to be activated is the interventricular septum, immediately followed by the dominant activation front, directed laterally, and finally the left and right posterobasal areas are depolarized.[3] Initial septal activation leads to a negative deflection in leads I and aVL (septal q), after which the activation spreads to the lateral and posterior wall. The dominant force in the direction of these leads results therefore in an R wave (Fig. 3.11). In the transverse plane this activation sequence leads to an rS, an RS, and a qR complex in V_1, V_3 or V_4, and V_6, respectively (Fig. 3.11).

The normal QRS width does not exceed 90 ms. The height of the R wave is less than 25 mm in V_5 and V_6, and 20 mm in leads I and aVL. The Q wave is not wider than 40 ms. Widening of the QRS may be due to sequential activation of both ventricles in the bundle branch block; intramyocardial conduction delay, as seen in ischemia or fibrosis; or prolonged conduction times due to increased muscle mass, as seen in hypertrophy or ventricular dilatation.

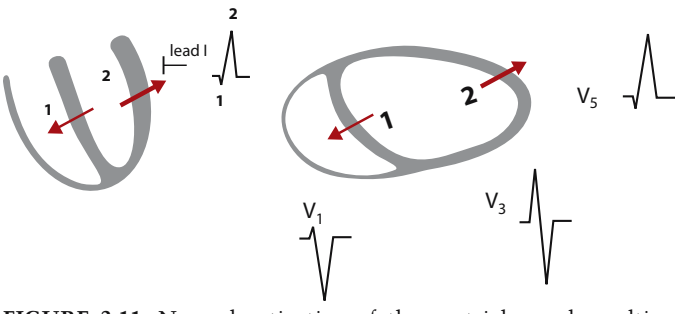

FIGURE 3.11. Normal activation of the ventricles and resulting QRS configuration in the frontal and transverse plane.

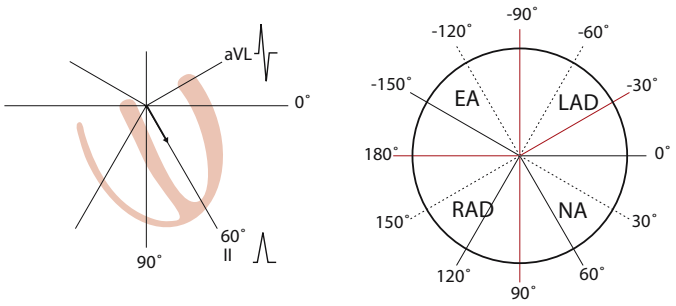

FIGURE 3.12. Assessment of the QRS axis in the frontal plane. Left: In this example in lead aVL R height is equal to S depth. The overall direction is therefore 0 and thus the axis is perpendicular to aVL. The axis could either be +60 or –120 degrees. Lead II shows a positive QRS complex, therefore the axis is +60 degrees. Right: Definition of normal and abnormal QRS axes in the frontal plane. NA, normal axis; LAD, left axis deviation; RAD, right axis deviation; EA, extreme axis deviation.

The height of the QRS complex is dependent on many factors such as age, thickness of the thoracic wall, and individual differences in stature.

The Electrical Axis

In clinical practice the electrical axis is frequently used to indicate the main electrical activation front. The axis can be assessed for any part of the P-QRS-T complex. The axis is defined as the global direction of electrical activation. A convenient way to determine the axis is to go from a lead where the global direction is (most) perpendicular to, for example, an isoelectric segment or a lead with an equal upward and downward deflection. The next step is to assess in another lead whether this direction is toward or opposite from its positive pole (Fig. 3.12).

The QRS axis is normally between –30 and 90 degrees. Between –30 and –90 degrees left axis deviation is present, and between 90 and 180 degrees right axis deviation. The term *extreme axis* is used in case of an axis between –90 and 180 degrees.

Clinical Significance of the QRS Axis

Axis deviation may occur due to factors such as (1) the anatomic position of the heart; (2) gain of forces in a specific direction, for instance, right axis deviation in right ventricular hypertrophy; (3) loss of forces in a localized area, such as in myocardial infarction; and (4) changes in the activation sequence such as in conduction delay or block in the bundle branches or in the left anterior or posterior fascicle.

The ST Segment

The ST segment occurs during calcium influx into the myocardium allowing contraction to occur. This process is simultaneous and homogeneous throughout the ventricles and therefore the ST segment is usually isoelectric. In the normal ECG lead V_2 may show some ST elevation, especially in men.

ST segment deviation may occur as the consequence of a disease process, such as hypertrophy, ischemia, pericardi-

tis, or infarction, and is then called a primary ST segment change. When ST-T changes occur as the result of a concomitant ECG abnormality, such as in bundle branch block, preexcitation, or a paced rhythm, secondary ST-T segment changes are present.

The T Wave and the QT Interval

The T wave reflects the repolarization of the ventricles and although it has a similar polarity as the QRS complex, slight differences in axis may be present, but usually not more than 45 degrees. Changes in polarity and T wave configuration may be due to many different causes and are outlined in more detail below.

The duration of the QT interval is generally used to indicate the duration of the repolarization time. This interval is rate dependent and normalization is done using different methods, Bazett's formula [corrected QT interval (QTc) = QT (ms)/$\sqrt{R - R(s)}$] being the most frequently used. Normal values are below 440 ms for men and below 450 ms for women. The QT interval is prolonged in the setting of the long QT syndrome, comprising different congenital and acquired forms.

The U Wave

The normal U waves are likely to be produced by the repolarization of the His-Purkinje system, but there is some doubt about whether large or inverted U waves are produced in this manner. It is suggested that abnormal U waves are actually due to split T waves created by two voltage gradients across the ventricular myocardium. The first voltage gradient is responsible for the first part of the T wave (the usual T), and the second voltage gradient is responsible for the second wave that is currently called an abnormal U wave.[4]

The Abnormal Electrocardiogram

ECG abnormalities can be divided grossly into (1) configurational changes and (2) time and sequence related changes of the P-QRS-T complex. The latter changes are disturbances of the heart rhythm and are discussed elsewhere.

Configurational changes, considered here, are P wave abnormalities (such as atrial enlargement and hypertrophy, and atrial infarction), changes in QRS configuration (increase in QRS voltage, for instance, in hypertrophy, widening of QRS duration as in bundle branch block, decrease in QRS voltage, localized changes such as in healed infarction), ST segment changes (acute coronary syndromes, acute pericarditis), and primary T-wave abnormalities (acute ischemic or postischemic T-wave changes, electrolyte abnormalities).

In the examples given below one can observe that many disease states will simultaneously change different parts of the P-QRS-T complex.

Changes in the P-Wave Configuration

Left Atrial Enlargement

Changes in the P wave, indicating overload of the left atrium, are due to several factors, such as hypertrophy of the atrial muscle, enlargement of the atrial compartment, slowing of conduction, and prolonged conduction time due to the larger muscular mass.[5] This change leads to an increase in voltage and, in cases of left atrial pathology, to an increase in duration of the latter part of the P wave. Leads II, aVL, and V1 usually depict these abnormalities best (Figs. 3.13 and 3.14A). Lead II shows a wide P wave with a prominent second part, frequently after a notch due to a transition from right to left atrial activation. In lead aVL typically a late positive deflection is observed. In lead V_1 small initial positivity, due to right atrial activation, is followed by deep and broad late negativity. Criteria for left atrial enlargement are P wave duration in lead II ≥ 110 ms,[6] and late negativity in lead V_1 ≥ 1 mm^2 (Table 3.2).[7]

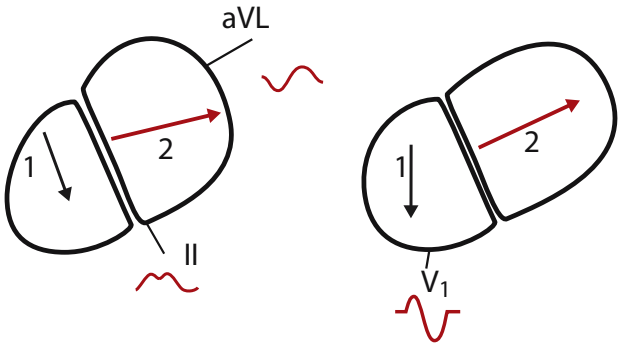

FIGURE 3.13. Left atrial hypertrophy. Electrical forces in left atrial hypertrophy in the frontal and transverse plane and the resulting P wave configuration.

Another important P wave abnormality in this setting is interatrial conduction block and retrograde activation of the left atrium (Fig. 3.14B).[8]

Diagnosing left atrial enlargement and intraatrial conduction block has important clinical implications as it frequently indicates left ventricle (LV) disease, for instance mitral valve disease, LV hypertrophy, or end diastolic pressure elevation, but it also indicates a substrate for atrial arrhythmias, such as atrial fibrillation.

Right Atrial Enlargement

In right atrial enlargement mostly the initial part of the P wave is distorted, because the right atrium is activated before the left atrium. The increase in voltage and duration will lead to superposition on left atrial activation in lead II and thus to a tall P wave (Figs. 3.15 and 3.16). In lead V_1 prominent initial positivity is seen. Criteria for right atrial enlargement are a P wave in lead II ≥ 0.25 mV (2.5 mm) and lead V_1 ≥ 0.15 mV (1.5 mm) (Table 3.2). Also criteria in the QRS complex, and related to RV hypertrophy, have been defined to diagnose right atrial enlargement. A qR pattern in V_1, a QRS voltage of 4 mm or more, has been found to be very specific.[9] Right atrium (RA) enlargement is present in congenital and valvular heart disease and in pulmonary hypertension.

Increased sympathetic tone also sometimes results in a more cranial origin of the sinus, and faster atrial activation with superposition of right and left atrial activation results in taller P waves in the inferior leads, mimicking right atrial enlargement.

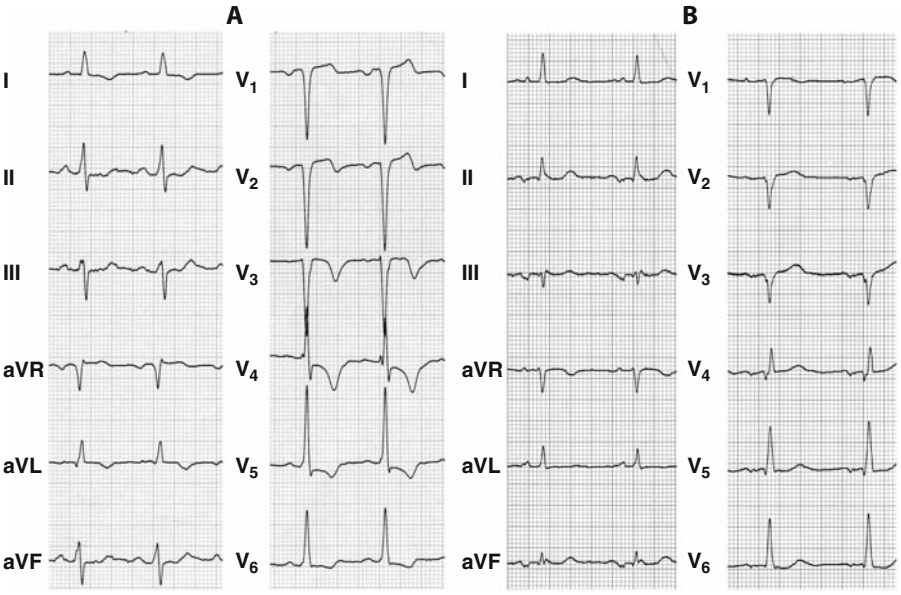

FIGURE 3.14. ECG with left atrial hypertrophy (A) and interatrial conduction block (B). (A) Sinus rhythm, 70 beats/min, wide P wave, 120 ms in lead II, with prominent terminal part, >1 mm^2, in V_1, indicating left atrial hypertrophy, in this case in anterior wall infarction. (B) Sinus rhythm, P waves with markedly prolonged terminal part, negative in leads II, III, and aVF, due to interatrial conduction block with retrograde left atrial activation.

TABLE 3.2. Criteria for left and right atrial enlargement

	P wave		*QRS*	*Sens*	*Spec*	*PV*
Left atrial enlargement	II, III, aVF	≥120 ms		65%	70%	
	V_1	≥1 mm^2		51%	87%	
Right atrial enlargement	II	>2.5 mm		Low	Low	20%
	V_1	≥1.5 mm				
			qR	Low	Low	
			QRS ≤ 4 mm		100%	
			$QRSV_2/V_1 \geq 5$	46%	90%	80%
Biatrial enlargement	V_1 pos	1.5 mm				
	peak neg					
	peak	≥1 mm^2				

PV, predictive value; Sens, sensitivity; Spec, specificity.

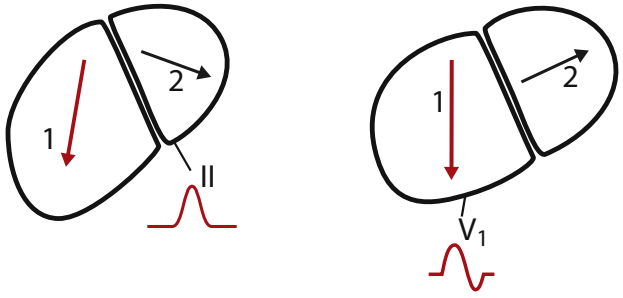

FIGURE 3.15. Right atrial hypertrophy. Electrical forces in right atrial hypertrophy in the frontal and transverse plane and the resulting P wave configuration.

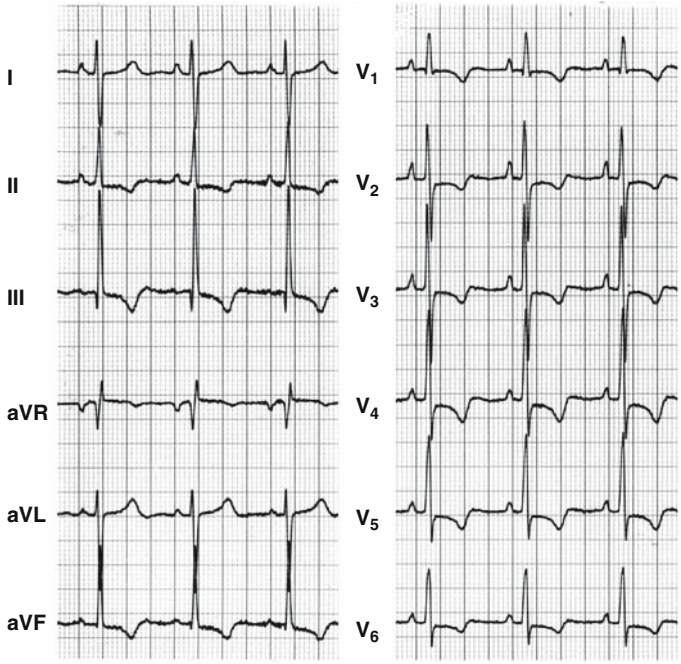

FIGURE 3.16. Right atrial and ventricular hypertrophy. Sinus rhythm, rate 75 beats/min electrical axis shifted to the right, +120 degrees, prominent positive P waves in V_1 and V_2, indicating right atrial hypertrophy, tall R waves in III and V_1, persistent s in V_6, secondary T wave abnormalities indicating severe right ventricular hypertrophy. Case of severe primary pulmonary hypertension.

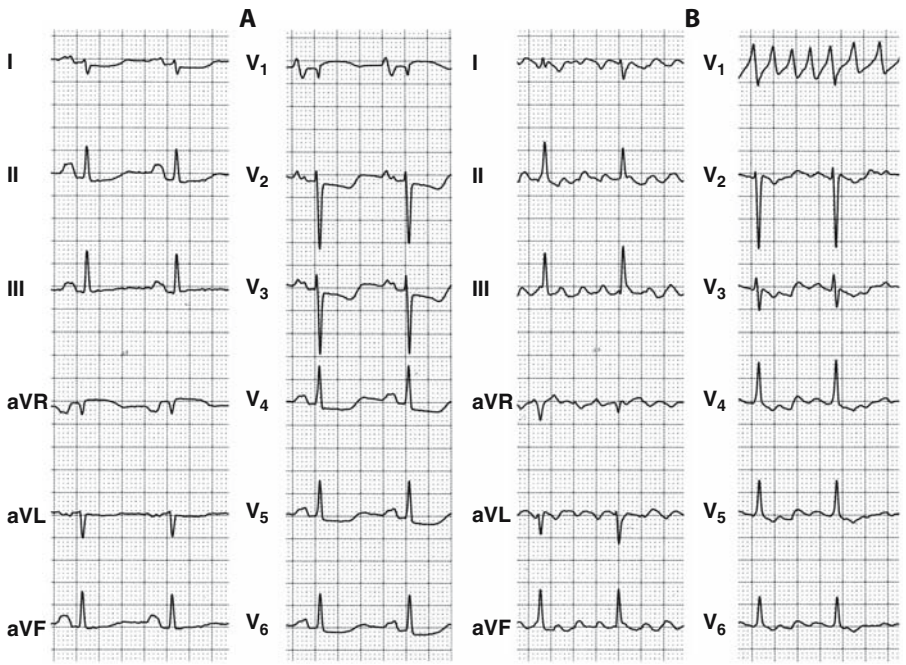

FIGURE 3.17. Biatrial hypertrophy. (A) Sinus rhythm, rate 75 beats/min, wide and tall P wave in lead II, prominent biphasic P wave in lead V_1, indicating biatrial enlargement, right axis deviation, s in V_1, abnormal ST-T segments, indicating right ventricular overload. (B) Same patient in atrial fibrillation. Note huge fibrillation waves in V_1. Case of restrictive cardiomyopathy.

Biatrial Enlargement

In biatrial hypertrophy features of both right and left atrial hypertrophy are present (Table 3.2 and Fig. 3.17): (1) P wave in lead II is taller and wider than normal, 2.5 mm and 0.12 s, respectively; and (2) left atrial enlargement is combined with QRS criteria for right atrial enlargement.

Changes in QRS Configuration

Conditions with Voltage Increase

Left Ventricular Hypertrophy

Left ventricular hypertrophy is an important ECG diagnosis. The ECG features are based on the pathophysiologic and structural changes in the left heart. Increased load to the left ventricle such as in hypertension or aortic valve disease lead to increased wall thickness and therefore an increase of the QRS voltage. The hypertrophic process is accompanied by fibrosis, and impaired cell-to-cell coupling that may cause impaired intramyocardial conduction and also impaired bundle branch conduction. This leads to widening of the QRS complex and altered initial activation. The result will be disappearance of the septal q and a delayed intrinsicoid deflection. The latter is assessed as an increase of the qR time in V_5 beyond 50 ms. Increase of dominant forces of the basal left ventricle due to the larger muscle mass causes the electrical axis to shift leftward. Increased atrial contribution to fill the less compliant left ventricle leads to left atrial hypertrophy, and the presence of subendocardial demand ischemia leads to secondary ST T abnormalities, apparent as downsloping ST depression in the anterolateral leads I, aVL, V_5, and V_6. The latter is more frequently present in pressure than in volume overloaded left ventricles.

Voltage criteria were developed to diagnose left ventricular hypertrophy (LVH); all have a similar drawback of a high specificity at the expense of a low sensitivity (Table 3.3).[7] The Cornell voltage criteria for LVH include sex specificity.[10]

TABLE 3.3. Voltage criteria for left ventricular hypertrophy

	Sensitivity (%)	*Specificity (%)*	*Accuracy (%)*
RI+SIII > 25 mm	10.6	100	55
RaVL > 7.5 mm	22.5	96.5	59.5
RaVL > 11 mm	10.6	100	55
RaVF > 20 mm	1.3	99.5	50
$SV_1 + RV_{5-6} \geq 35$ mm	42.5	95	74
$SV_1 + RV_{5-6} > 33$ mm	55.6	89.5	73
In $V_1 - V_5$ any S + R > 45 mm	45	93	69
RV5–6 > 26 mm	25	98	62
$RaVL + SV_3 > 2.8$ mV in men	42	96	68
$RaVL + SV_3 > 2.0$ mV in women			
Romhilt-Estes score	60	97	78

TABLE 3.4. Romhilt-Estes score to diagnose left ventricular hypertrophy (LVH)

QRS changes			*Points*	*Total*
	Voltage criteria		3	
		R or S in frontal plane ≥20 mm $SV_1 - V_2 \geq 20$ mm $RV_5 - V_6 \geq 30$ mm		
	Frontal axis ≥–30		2	
	ID in $V_5 - V_6 \geq 0.05$ s		1	
	QRS duration ≥0.09 s		1	
ST-T changes	ST depression without dig		3	
	ST depression with dig		1	
P wave	Terminal part V1 ≥1 mm^2		3	
LVH probable				4
LVH present				5

Dig, digitalis.

Apart from gender, other factors influence the accuracy of the ECG, such as obesity decreasing sensitivity and black race decreasing specificity.[11] Combined QRS voltage and duration criteria, expressed as their product, have been found to increase sensitivity to 51%.[12] To improve the accuracy of the ECG diagnosis of LVH, scoring systems were developed (Table 3.4),[7] including not only changes in the QRS complex but also in the P wave and the ST segment. More recently, continuous rather than dichotomous scoring systems were developed, increasing the sensitivity without sacrificing specificity.[13] Although echocardiography is more sensitive than the ECG to diagnose LVH, both techniques contain independent prognostic information and, at least in hypertensive patients, both should be performed to fully assess the increased risk.[14]

Unloading the left ventricle can lead to regression of hypertrophy and to normalization of the ST segment, and decrease in the voltage and width of the QRS complex (Fig. 3.18).[15]

Right Ventricular Hypertrophy

Right ventricular pressure and/or volume overload leads to right ventricular hypertrophy (RVH) and dilatation of the right ventricle.[16] Owing to the thinner wall of the right ventricle, this compartment has less opportunity to be exposed in the ECG, even when pathologic changes are present. This holds especially during the regular synchronous activation of both ventricles. When sequential activation is present, such as in left- or right-sided aberrant conduction, abnormalities will be apparent more easily. In RVH rightward forces counteract the left ventricular forces and could even become the dominant direction of electrical activation (Table 3.5).[7] In the frontal plane this leads to rightward shift of the electrical QRS axis. In the precordial leads, the QRS configuration in the V_1 lead shows the most pronounced consequence of the rightward force; this may vary from a diminished depth of the S wave to the occurrence of a tall R wave and a qR complex. The latter indicates severe RVH, frequently in the setting of high pulmonary artery pressures (Fig. 3.19). Another feature of hypertrophy is conduction delay in the right bundle or the RV myocardium. This will lead to a secondary R and/or delayed intrinsicoid deflection of RV, best seen in V_1 (Fig. 3.20).

Dilatation of the RV. Dilatation of the RV frequently accompanies hypertrophy, due to the thin-walled RV not prepared to generate high pressures. RV dilatation is seen in

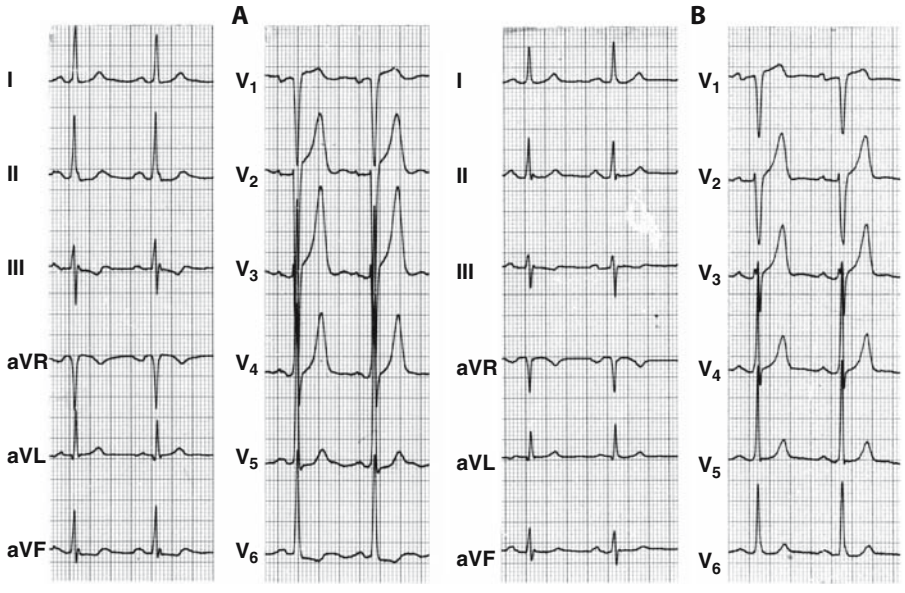

FIGURE 3.18. Left ventricular hypertrophy (LVH) (A) and regression of LVH (B). Sinus rhythm, 75/min, electrical axis +30 degrees, absence of septal q waves, increased QRS voltage in the precordial leads, ST-T segment changes in V_6, indicating severe LVH. Clinically severe aortic valve stenosis. (B) Generalized decrease in voltage indicating regression of LVH 2 months after aortic valve replacement.

TABLE 3.5. Criteria for RVH

	Criterion	*Sens*	*Spec*
V_1	R/S $V_1 \geq 1$	6	98
	R ≥ 7 mm	2	99
	QR	5	99
	S < 2 mm	6	98
	IDT ≥ 0.35 s	8	98
$V_5 - V_6$	R/S ≤ 1	16	93
	R < 5 mm	13	87
	S ≥ 7 mm	26	90
$V_1 + V_6$	$RV_1 + SV_6 > 10.5$	18	94
QRS axis	≥110 degrees	15	96
	$S_IS_{II}S_{III}$	24	87

IDT, intrinsicoid deflection (time from onset to R wave peak).

the extremity leads as a generalized decrease in QRS voltage, and an undetermined electrical axis. In the precordial leads RV dilatation is observed as slow R progression and a persistent S until V_6. This is due to extension of the RV anterolaterally. ECG signs of RV dilatation may normalize when pressure or volume overload in the pulmonary circulation improves (Fig. 3.21).

Acute RV Pressure Overload (Pulmonary Embolism). Acute RV pressure overload may occur in several clinical circumstances, the most important being acute pulmonary embolism. The ECG is important to support the correct diagnostic workup in a suspicious clinical condition. The sudden increase of the resistance in the pulmonary circulation leads to abrupt changes in the right ventricle, such as a sudden decrease in stroke volume, dilatation, conduction delay, and ischemia. The typical ECG features in the acute phase (Fig. 3.22 and Table 3.6)[17] are sinus tachycardia, right atrial and ventricular premature beats, or atrial fibrillation/flutter. Bradycardia is rare and an agonal sign. The P wave is usually normal and only rarely tall P waves are present. The QRS complex shows a rightward shift and signs of right ventricular conduction delay, apparent as an S in lead I, a Q in lead III (McGinn and White's sign), and a late R in leads aVR and V_1. The amount of right ventricular conduction delay correlates with the extent of obstruction within the pulmonary circulation. Right ventricle dilatation may cause a generalized voltage decrease in the extremity leads and slow R progression in the precordial leads. ST elevation in leads aVR and V_1 is part of the picture, and is probably due to demand ischemia. In the subacute phase these signs gradually normalize within a few days, but negative T waves develop especially in leads V_1 to V_4, which remain for days to weeks.

RV Hypertrophy in Chronic Obstructive Pulmonary Disease. In chronic obstructive pulmonary disease (COPD) the ECG is determined by the amount of RVH and RV dilatation due to pulmonary hypertension, the increased pulmonary volume due to conditions such as emphysema and backward tilt of the apex of the heart as the low diaphragm pulls down on the pericardium. The latter leads to generalized decrease in voltage and an upward shift of the QRS axis in the frontal plane. Sinus tachycardia and not infrequently atrial fibrillation is present, a QRS axis in the frontal plane pointing in an extreme direction leading to the typical $S_IS_{II}S_{III}$ pattern, and in the precordial leads a shallow s wave in V_1 as the consequence of RVH, slow R progression, and a persistent s wave in V_6 due to RV dilatation (Fig. 3.23).

Biventricular Hypertrophy

RV Involvement in LV Disease. Left ventricle disease, such as hypertensive heart disease, myocardial infarction, and mitral and aortic valve abnormalities, are usually

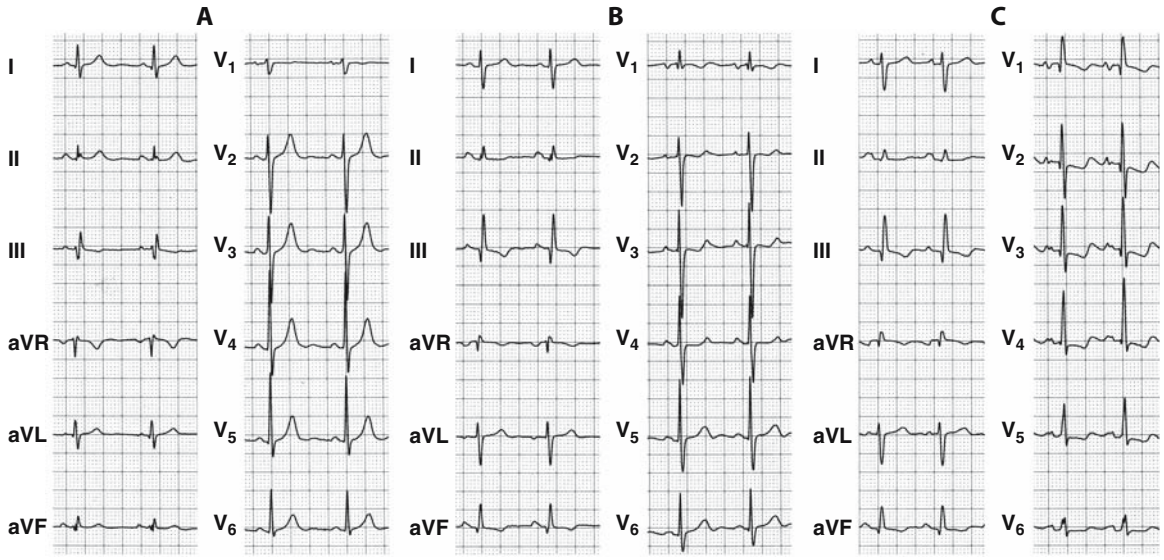

FIGURE 3.19. Development of right ventricular hypertrophy (RVH). Three panels illustrating the development of RVH. All panels show sinus rhythm. (A) QRS axis is 35 degrees. A s is present in lead I. Also a small s is present in V_1, as the result of right ventricle (RV) forces counteracting left ventricle (LV) activation (cancellation), the shallow s sign, which is consistent with RVH. (B) Recorded 6 years later, it shows right axis deviation (+110 degrees), a qR pattern, an s in V_6. Changes are even more pronounced in panel C.

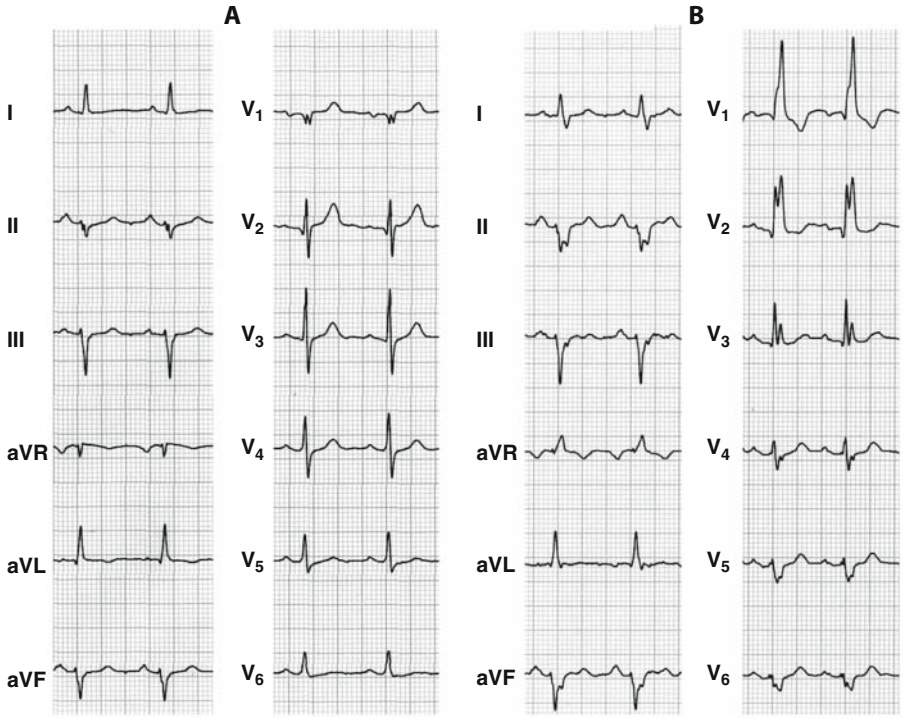

FIGURE 3.20. Right ventricular hypertrophy in right bundle branch block (RBBB). (A,B) RBBB in the same patient with ischemic heart disease and mitral regurgitation. Sinus rhythm, left atrial hypertrophy, left axis deviation, q wave in V_2. (A) Note the shallow S during synchronous activation of both ventricles. (B) During RBBB, signs of RVH are more evident because of the sequential activation of both ventricles. RBBB has a qR pattern, delayed intrinsicoid deflection and tall R.

reflected in the ECG. With increasing severity of these conditions the pulmonary circulation may become pressure or volume overloaded and in this way the right ventricle becomes involved. Diagnosing RV involvement in LV disease is therefore a marker of the severity of the disease process. The ECG can give clues as to the presence of this situation. Biventricular hypertrophy (BVH) may occur in hypertensive heart disease (Fig. 3.24); in valvular heart disease, such as mitral regurgitation; and in hypertrophic cardiomyopathy (HCM) (Fig. 3.25; Table 3.7). Next to signs of LVH, frequently left atrial hypertrophy is present, and signs of RV involvement are present, such as increased voltage signs of hypertrophy, the typical feature being a tall R and a deep S in V_3 (Katz-Wachtel complex), rightward shift in the frontal plane,

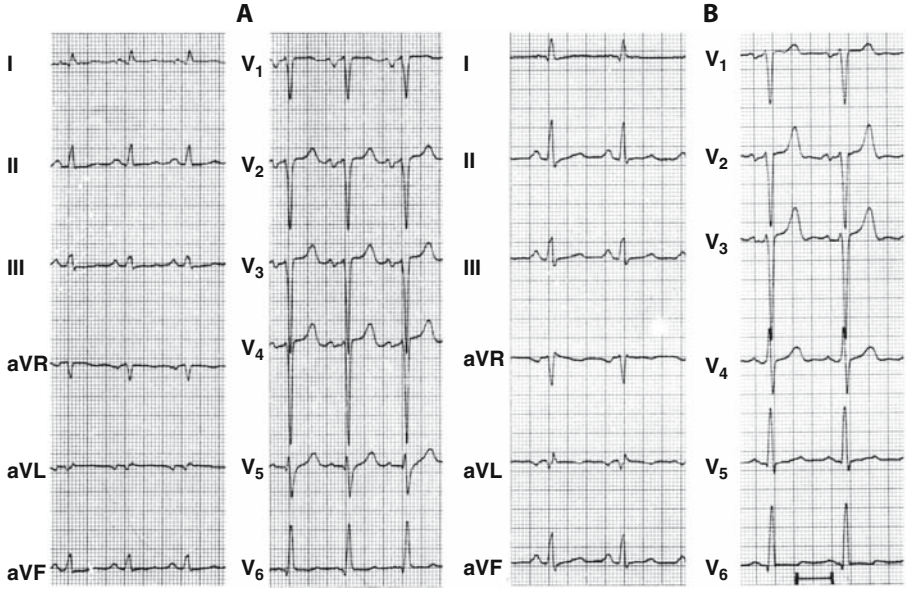

FIGURE 3.21. RV dilatation and regression after heart failure treatment. (A) Recorded during severe congestive heart failure. Sinus tachycardia, 95 beats/min. Left atrial enlargement, low voltage in the extremity in contrast to the precordial leads, slow R progression in the precordial leads due to RV dilatation. (B) Recorded after treatment. Slowing of the sinus rate, 75 beats/min, no left atrial hypertrophy, normalization of voltage in the extremity leads, R progression in the precordial leads. The dynamic QRS behavior is explained by RV dilatation and subsequent normalization.

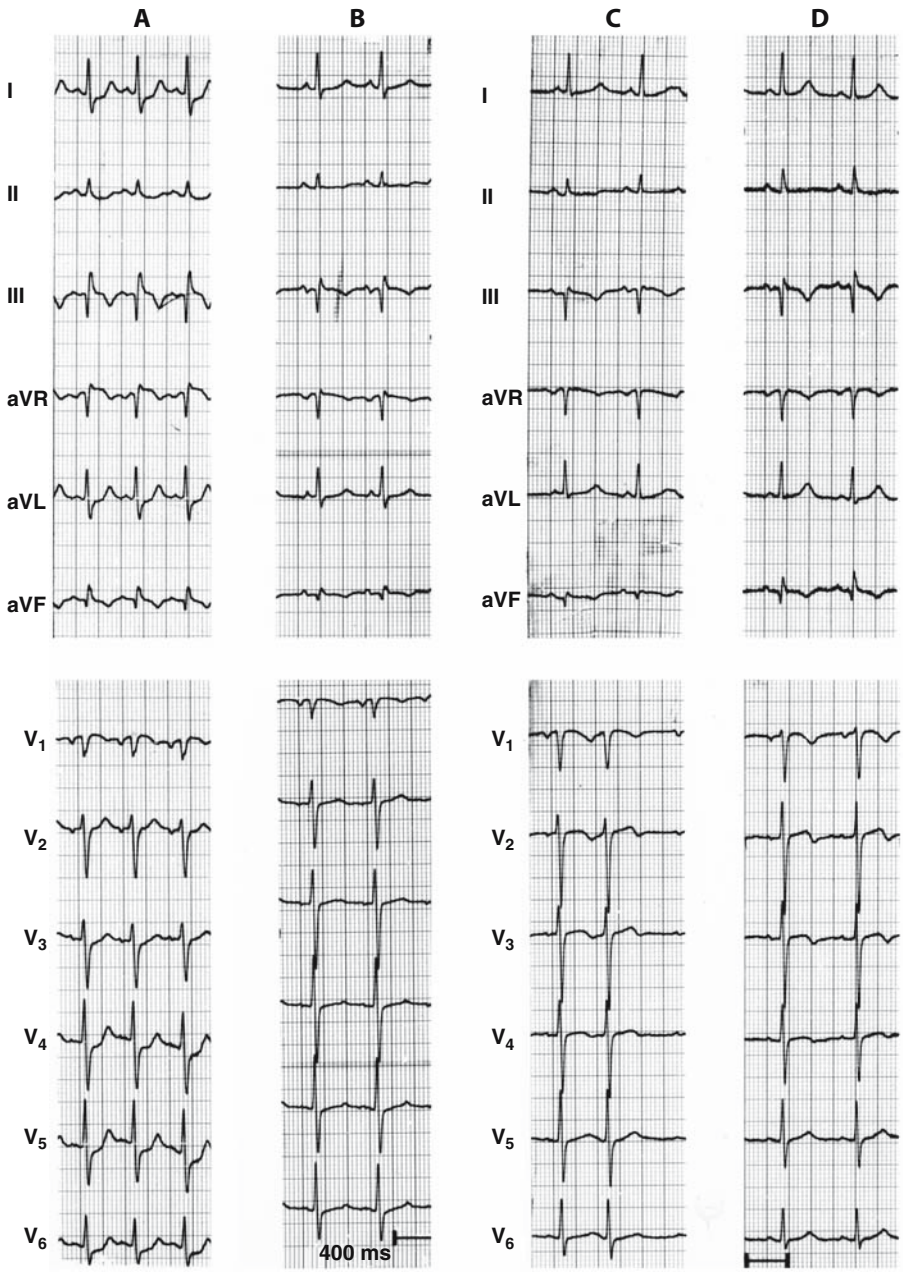

FIGURE 3.22. Acute RV pressure overload. Four panels with ECGs recorded during the acute, subacute, and later stages of acute pulmonary embolism. Left ECG shows sinus tachycardia rightward shift of QRS axis, S1, Q3, T3 pattern, right bundle conduction delay, S in V_6 and ST elevation in aVR and V_1. These changes normalize over time, but T wave negativity in V_1 to V_4 remains for weeks to months.

TABLE 3.6. Pulmonary embolism

P wave	P pulmonale	8%
	Atrial arrhythmias	22%
QRS complex	Frontal axis > 90 degrees	33%
	Axis undetermined	31%
	Voltage <5 mm in extremity leads	20%
	S lead I and/or aVL	73%
	Q in lead III and/or aVF	49%
	RBBB incomplete	53%
	RBBB complete	14%
	Slow R progression precordial leads	51%

RBBB, right bundle branch block.

increase of the intrinsicoid deflection of the RV (as measured in lead V_1), and dilatation such as decrease of QRS voltage in the extremity leads and slow R progression in the precordial leads.

Hypertrophic Cardiomyopathy. The 12-lead ECG is abnormal in 75% to 95% of HCM patients. The abnormalities are related to (1) the involvement of the respective compartments, (2) the hemodynamic consequences, and (3) the stage and severity of the disease. Involvement of the ventricles and/or atria leads to LV and/or RV and LA and/or RA hypertrophy (Fig. 3.25). Abnormal initial activation (pseudo–δ-wave), Q waves (pseudo-infarction), and QRS widening (pseudo–bundle branch block) may also be present. ST-T abnormalities and prolonged QT interval are not unusual.

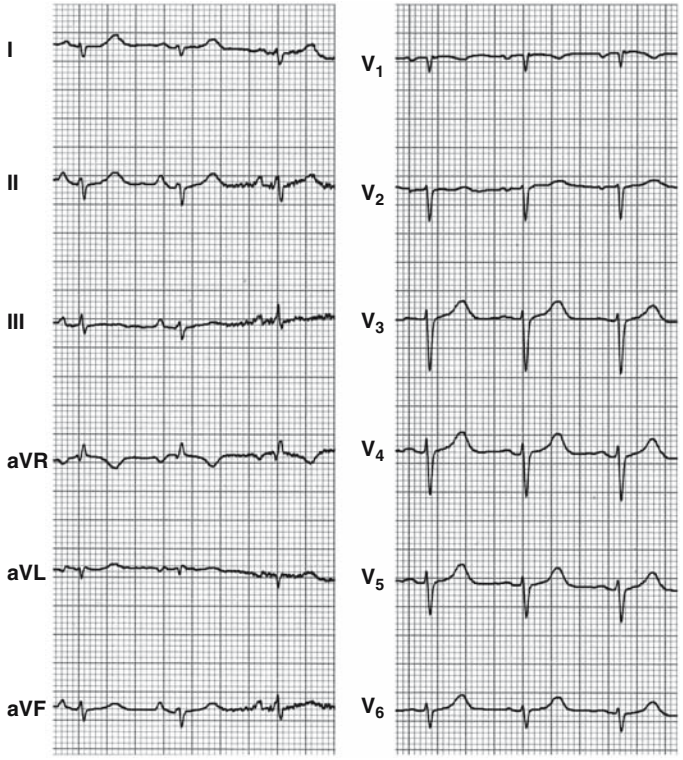

FIGURE 3.23. RV hypertrophy and dilatation in emphysema. Sinus rhythm, generalized decrease in voltage, extreme axis deviation, small s in V_1, slow R progression in the precordial leads, persistent S in V_6.

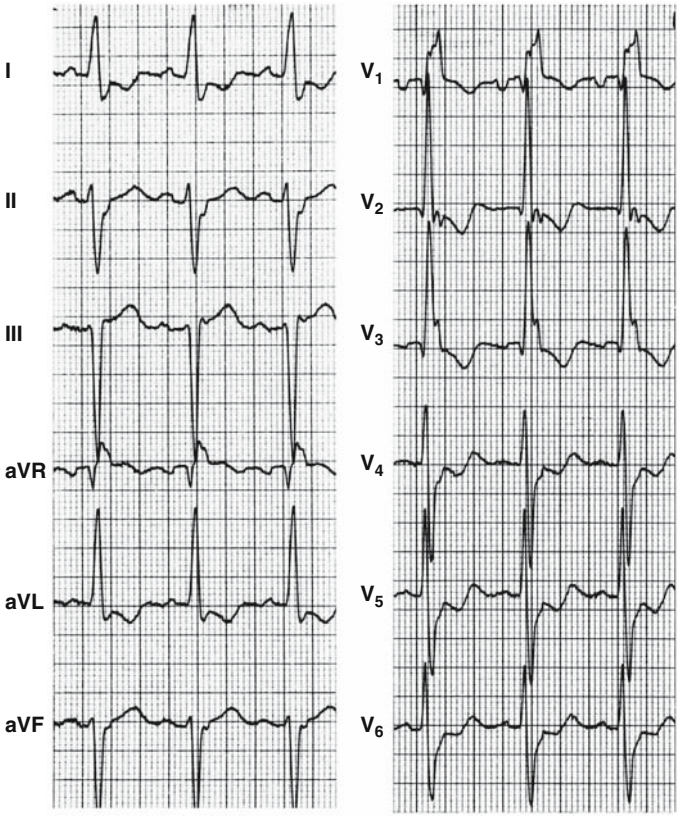

FIGURE 3.24. Biventricular hypertrophy. Atrial fibrillation, left axis deviation due to left anterior hemiblock, positive criteria for LVH, qR in V_1 and delayed intrinsicoid deflection, persistent S in V_6. Hypertensive heart disease.

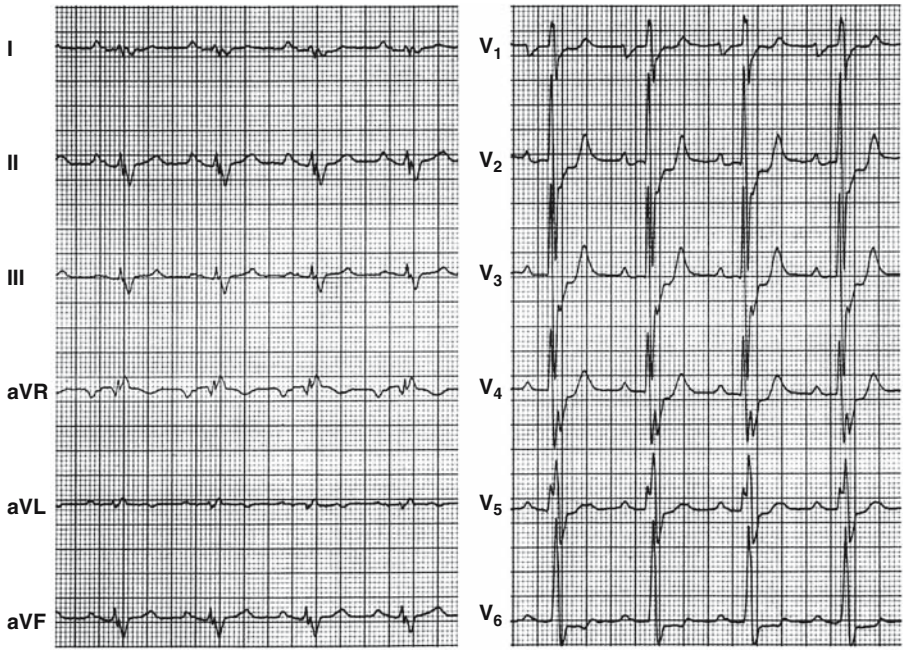

FIGURE 3.25. Sinus rhythm, 75 beats/min, wide P waves, 120 ms in lead II, >1 mm^2 in V_1, indicating left atrial hypertrophy, electrical axis in frontal plane perpendicular to all leads (indeterminate axis), discrepancy of low voltage in the extremity leads with high voltage in precordial leads, consistent with right ventricular dilatation, tall R in V_1 to V_3, suggesting RVH, R in $V_6 > V_5$ indicating LVH. Widened QRS complexes. Case of hypertrophic cardiomyopathy.

TABLE 3.7. Right ventricle (RV) involvement in left ventricle (LV) disease

LV disease	*RV involvement*
LVH	Rightward shift electrical axis
Old infarction	Slow R progression precordial leads
LBBB	Low voltage extremity leads In RBBB tall R´ In RBBB persistent R´ over precordial leads In LBBB increased R V_1–V_2

Marked T-wave inversion in the precordial leads should alert the clinician to the diagnosis of the apical form of HCM. Arrhythmias such as atrial fibrillation and ventricular ectopic activity, and also conduction disturbances such as AV junctional delay or block and bundle branch block, are seen.

Widening of the QRS Complex

Left Bundle Branch Block

In left bundle branch block (LBBB) both ventricles are activated through the right bundle branch. Slowing of conduction or complete block may be structural or functional. Therefore, LBBB may be intermittent, through mechanisms such as fast (phase 3 block) or slow rate (phase 4 block), retrograde invasion into one of the bundles by premature ventricular beats, or a mechanism called acceleration dependent block.

The configuration of LBBB in the ECG is explained by the activation sequence of the ventricles through the right bundle branch solely. This structure inserts into the right ventricle anteriorly in the apex. Therefore, the first part to be activated is the right ventricular anterior wall, which may result in a tiny r wave in lead V_1 (Figs. 3.26 and 3.27). Thereafter the interventricular septum is activated from right to left, resulting in initial positivity in the lateral leads I, aVL, and V_6. The LV apex is the structure next to be activated, frequently leading to slightly less voltage due to the smaller amount of tissue. This is typically seen as a notch at the nadir of the QRS complex. Finally the lateral wall is activated, producing positivity in the lateral leads. The serial activation of the LV and the conduction through the myocardium results in a widened QRS complex, but not exceeding 140 ms (Table 3.8). Exceeding this duration suggests additional reasons for slow or prolonged conduction within the myocardium, such as hypertrophy, dilatation, ischemia, or use of medication, and is termed overcomplete LBBB. A typical QRS configuration but without widening of the QRS is named incomplete LBBB.

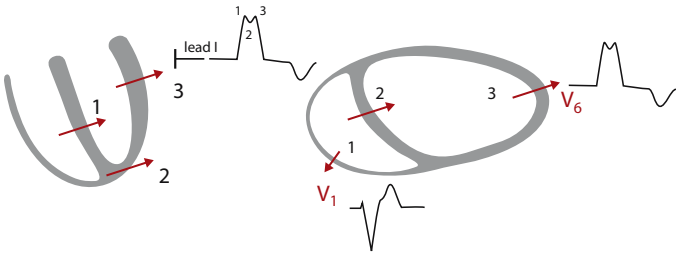

FIGURE 3.26. Left bundle branch block. Directional changes in left bundle branch block in the frontal and transverse plane and the resulting QRS configuration.

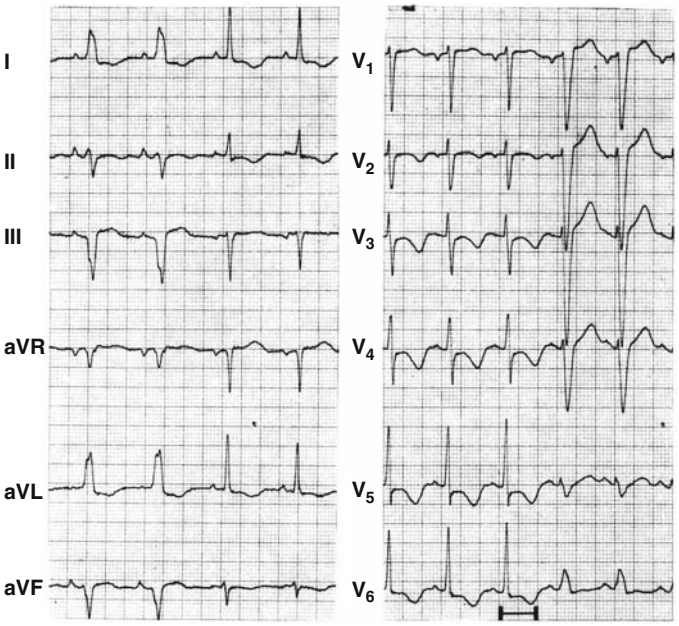

FIGURE 3.27. Intermittent left bundle branch block. Sinus rhythm, the first and last two beats show wide QRS, left axis deviation, absence of the septal q, widened QRS, 140 ms, notch in mid-QRS, secondary ST-T segment changes, indicating left bundle branch block. The middle five beats show normal conduction due to slight slowing in rate. The T wave abnormalities during normal conduction are caused by the preexisting LBBB, a phenomenon known as the cardiac memory sign.

Additional Heart Disease and LBBB. Additional features in the LBBB may unmask concomitant heart disease (Table 3.9). In LVH the Sokolow index ($S_{V1or2} + R_{V5or6} \geq 35$ mm) is valid in LBBB. In old myocardial infarction (MI) and LBBB the QRS is frequently distorted; slurring in the initial upstroke in leads I, aVL, and V_6 (Chapman's sign), slurring in the terminal upstroke in V_4 and V_5 (Cabrera's sign), Q waves in the leads I and aVL, and notches in the leads II, III, and aVF all indicate scar due to previous MI.

Right ventricular hypertrophy (RVH) is apparent in the ECG as a rightward shift of the QRS axis and gain of initial voltage (R wave) in the leads V_1 to V_3, and RV dilatation as low voltage in the extremity leads (Fig. 3.28). Acute ischemia is diagnosed by recording additional ST segment changes

TABLE 3.8. Criteria for LBBB and RBBB

	LBBB	*RBBB*
Axis	–30° to +60°	0°–120°
QRS width	120–140 ms	120–130 ms
Septal q wave	Absent	Present
Late R lateral leads	Present	Absent
V_1	rS complex	RsR´ complex
V_6	RR´ complex	Rs complex

TABLE 3.9. LBBB and additional heart disease

LVH		Sokolow index positive
Acute MI		Additional ST segment changes
Old MI		Initial slurring I, aVL, V_6 (Chapman's sign) Slurring S wave V_4 – V_5 (Cabrera's sign)
	Septal MI	Q wave I, aVL
	Inferior MI	Notches II, III, aVF
RVH/dilatation		Vertical/right axis Low voltage extremity leads Tall R $V_{1–3}$

MI, myocardial infarction.

next to the secondary repolarization abnormalities due to the LBBB. Serial comparison of subsequent ECG's is very helpful for this purpose. Very specific is ST positivity in leads with a positive QRS complex.

Right Bundle Branch Block

In right bundle branch block (RBBB), sequential activation of the left ventricle and right ventricle occurs due to conduction delay or block in the right bundle (Fig. 3.29). First the left ventricle is activated in the usual way, that is, firstly septal activation from to left to right, leading to a septal q wave in leads I and aVL, followed by activation of the other parts of the LV. The dominant direction in the lateral direction results in a large R wave in these same leads. After this, due to the right bundle branch block (RBBB) the right ventricle is activated, leading to a late S, in the leads I and aVL, and a tall secondary R in lead V_1. The s wave in the lateral leads typically has a rounded shape due to slow activation of the RV through the myocardium rather than through the Purkinje network (Table 3.8; Fig. 3.30A).

The RBBB is caused by similar pathologic mechanisms as in LBBB, such as hypertrophy, dilatation, ischemia, and the use of medication impairing conduction, such as class IA and IC drugs and tricyclic antidepressant drugs.

Also concomitant heart disease can influence the typical RBBB configuration. Examples are the initial r wave in V_1 increasing in height and width in old posterior wall infarction, but disappearing in septal infarction (Fig. 3.30B,C); in concomitant RVH the secondary R′ wave increases in height (Fig. 3.20). The RBBB masks left ventricular hypertrophy by decreasing the Sokolow index (Fig. 3.31).

Distortion of the QRS Complex

The QRS complex will be distorted due to local changes within the myocardium, such as scar formation, fibrosis, infiltration by proteins, granulomas, tumor metastases, etc. This distortion leads to changes of the QRS complex due to absent, diminished, or delayed local activation. Pseudoinfarction is diagnosed when one or more of the above-mentioned QRS distortions are present in the absence of ischemic heart disease. Examples are infiltrative heart disease such as cardiac sarcoidosis, hypertrophic cardiomyopathy, and preexcitation syndromes.

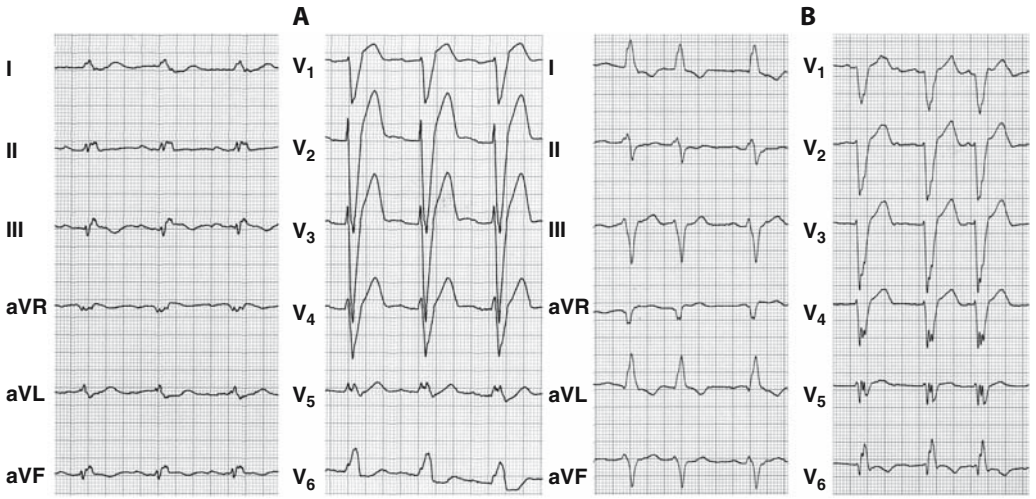

FIGURE 3.28. Changes in LBBB due to additional heart disease. (A) LBBB and right ventricular overload. Sinus rhythm, low voltage in the extremity leads, indicating right ventricular dilatation, tall R in V_2 to V_3, consistent with RVH. (B) LBBB in healed myocardial infarction. Sinus rhythm, notches in V_4 and V_5 (Cabrera's sign) and Q in V_6.

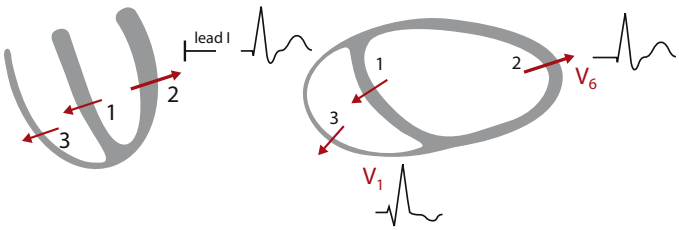

FIGURE 3.29. Right bundle branch block. Directional changes in the frontal and the transverse plane and the resulting changes in QRS configuration.

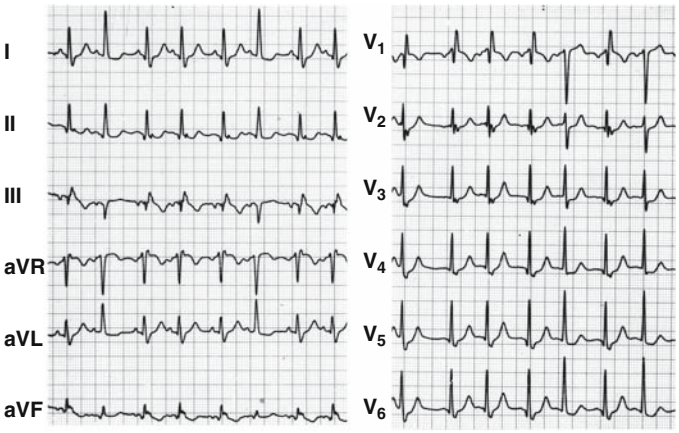

FIGURE 3.31. Right bundle branch block masks left ventricular hypertrophy. Atrial arrhythmia leading to changing RR intervals, leading to intermittent RBBB. Note the decrease in voltage in lead I and aVL during RBBB and the positive Sokolow index in the synchronous QRS, which is masked during RBBB.

Healed Infarction

After acute myocardial infarction the ischemic myocardium becomes necrotic and heals within weeks with scar formation. This leads in the QRS to the following possible changes: (1) decrease of R voltage due to less myocardium to be activated, (2) a Qr complex due to incomplete loss of myocardium and slow local conduction, (3) a QS complex in case of loss of local myocardium (Figs. 3.30B and 3.32). Dependent on the leads showing these changes, the infarction can be classified as anterior, inferior, or lateral. The loss of myocardium in cases of posterior wall infarction leads to a gain of R voltage in the precordial leads.

Based on the changes in the QRS, complex scoring systems were developed to estimate the infarct size.[18]

Pseudoinfarction

The QRS complex will also be distorted due to other local changes within the myocardium, such as fibrosis, infiltration by foreign materials, granulomas, and tumor metastases. This will lead to changes of the QRS complex due to absent, diminished, or delayed local activation. Pseudoinfarction is diagnosed when one or more of the above-mentioned QRS distortions are present in the absence of ischemic heart disease. Examples are infiltrative heart disease such as cardiac sarcoidosis (Fig. 3.33), hypertrophic cardiomyopathy, and preexcitation syndromes.

Decrease in QRS Voltage

A number of mechanisms and causes of a decrease in voltage have been mentioned above. Another not infrequent cause is starvation, a typical example being anorexia nervosa. Probably due to protein loss of the myocardium, a generalized decrease of voltage occurs (Fig. 3.34).[19] In addition, the ECG is characterized by sinus bradycardia and long QT time. The latter may lead to torsades de pointes and sudden cardiac death. After refeeding the ECG picture is reversible.

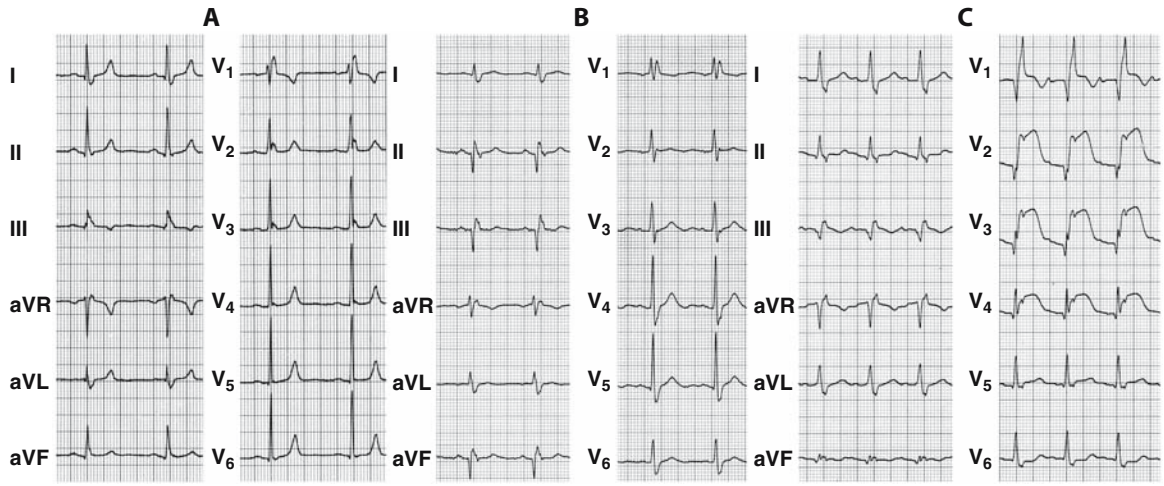

FIGURE 3.30. Right bundle branch block and changes in configuration due to location of myocardial infarction. (A) Sinus rhythm, QRS axis +60 degrees, normal initial QRS activation but late right ventricular activation best seen as a late wide S in lead I and aVL (and typically also in V_6) and a late R′ in V_1 (rSR′ complex). (B) RBBB in healed inferoposterior wall myocardial infarction. Sinus rhythm, left axis deviation due to myocardial loss in the inferior wall, resulting in Q waves in leads II, III, and aVF. Complete RBBB with a high initial R in V_1 as the result of posterior wall infarction. (C) Right bundle branch block in anteroseptal myocardial infarction. Sinus tachycardia, slightly prolonged PR interval, likely due to distal conduction delay. Due to muscle loss in the septum, the initial r in lead V_1 is absent, leading to the typical qR pattern in this situation.

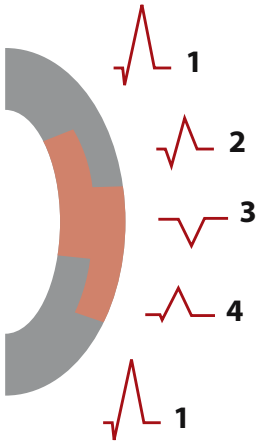

FIGURE 3.32. Different QRS configurations in healed myocardial infarction. (1) Normal tissue, qR complex. (2) Subendocardial infarction, Qr complex as the result of slow conduction and muscle loss. (3) Transmural infarction, QS complex due to total loss of local activation. (4) Subepicardial infarction, qr or r complex, as the result of normal subendocardial activation and epicardial muscle loss.

The ST Segment

The most important cause of ST segment changes, either ST elevation or depression, is ischemia of the myocardium. This can either be demand ischemia, occurring during situations such as exercise, anemia, or tachycardia, but also supply ischemia in the setting of a critical stenosis frequently due to plaque instability. This situation is covered by the term *acute coronary syndrome*, being further classified as ST elevation acute myocardial infarction (STEMI)[20] and non-ST elevation acute myocardial infarction (non-STEMI).[21]

Acute Coronary Syndromes

Management of STEMI and non-STEMI strongly depends on the assessment of the risk of extensive damage to the myocardium and its possible complications such as heart failure, ventricular arrhythmias, and sudden or nonsudden death. The ECG has proven to be a very useful tool for that purpose. STEMI usually leads to more immediate damage than non-STEMI and therefore treatment strategies are more aggressive, including thrombolytic therapy and primary and rescue PCI (percutaneous coronary intervention). Non-STEMI, however, also comprises high-risk situations, such as proximal left anterior descending branch (LAD) disease and left main and proximal three-vessel disease, and ECG characteristics have been described for their identification.[22] The ECG also diagnoses anterior versus non-anterior (inferior, posterior, lateral, and combinations) and basal versus apical STEMI locations. In both instances the former conditions involve larger areas at risk.[23] Right ventricular and atrial infarction and involvement of the specific conduction system can also be identified, all being situations indicating higher risk. The ECG of STEMI has been correlated with the culprit coronary artery and a proximal or distal site of occlusion in that vessel. These findings are helpful not only for assessment of the area at risk but also to guide the interventional cardiologist to the culprit lesion in cases of multivessel disease. The ECG also gives information about the acuteness[24] and the severity of STEMI, facilitating in both situations the choice of the most appropriate treatment strategy.

The Ischemia Vector

The term *ischemia vector* implies the direction and magnitude of the ST segment deviation during acute ischemia. Similarly to the QRS vector, the most convenient way to determine its direction is to go from a lead with an isoelectric ST segment. The ST vector is perpendicular to this lead and points to the direction of leads with ST elevation. Assessment of the direction of the ischemia vector facilitates assessing the site of (most) ischemia, and the amount of ST elevation provides information about its severity. Especially in the frontal plane, this vector has been shown to be helpful for this purpose.

STEMI

Occlusion of an epicardial coronary artery or of a side branch leads to acute transmural myocardial ischemia (also termed

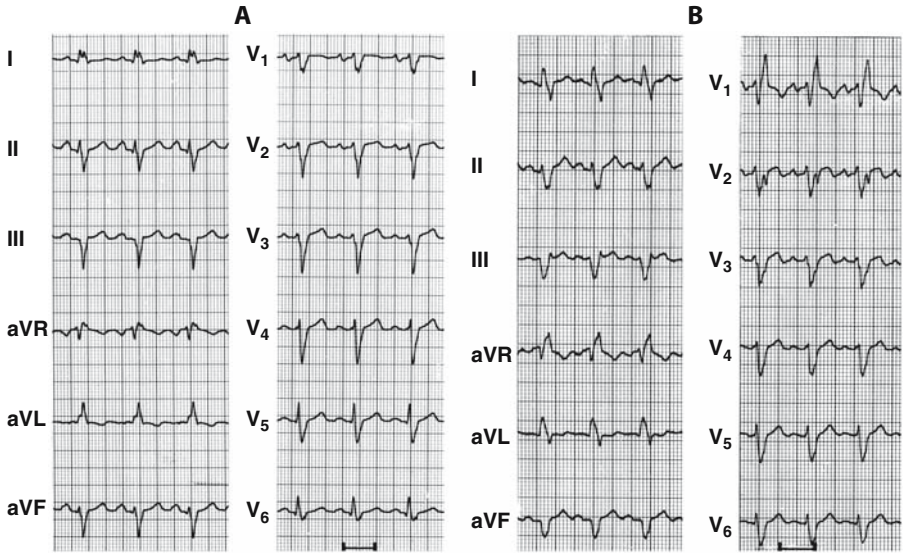

FIGURE 3.33. Distortion of the QRS complex. Cardiac sarcoidosis. (A) Sinus rhythm, left atrial hypertrophy, atypical widening of the QRS complex with pseudoinfarction pattern in leads II and III. (B) Same patient with RBBB. Involvement of the conduction system is frequent in cardiac sarcoidosis.

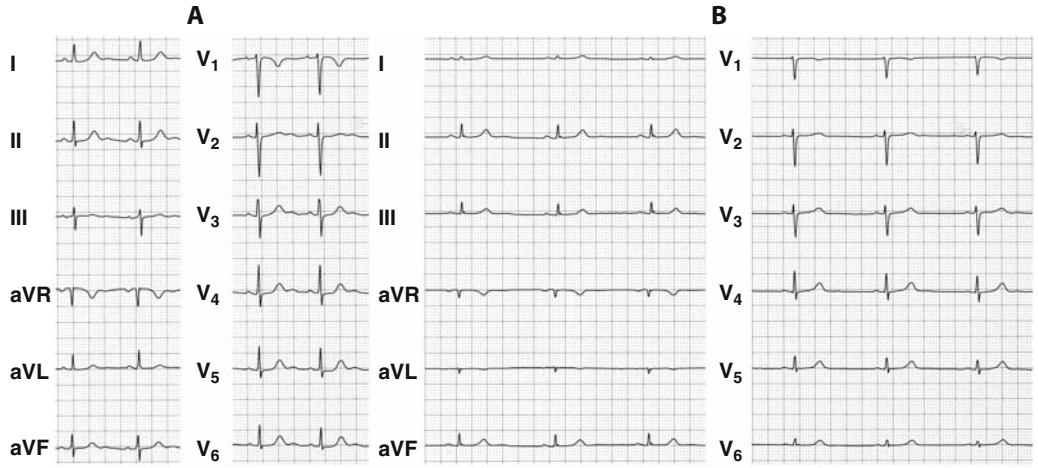

FIGURE 3.34. Decrease in P-QRS-T voltage. Anorexia nervosa. Before (A) and during (B) an episode of anorexia nervosa. Note sinus bradycardia, axis deviation to vertical, generalized loss of voltage, deflections in lead I almost being absent.

acute myocardial infarction). The site and extent of the ischemic area depends on (1) the artery being occluded, (2) the perfusion area of the occluded coronary, and (3) the site of occlusion within the vessel. The system coronary artery is shown in Figure 3.35.

Anatomy of the Coronary Artery System. The LAD runs along the anterior part of the interventricular septum and perfuses the interventricular septum and the anterior and anterolateral part of the left ventricle. In 40% to 50% of cases this vessel wraps around the apex and also perfuses the inferoapical area. The LAD is usually the most dominant coronary branch, and occlusion leads to the largest infarctions with possible complications such as heart failure, ventricular arrhythmias, and death. By way of the first septal perforator, it also perfuses the His bundle and the right and left bundle branch, the latter also being supplied by the right coronary artery (RCA). As a consequence, RBBB and intra-Hissian block can occur in LAD occlusion and indicate a proximal obstruction and therefore a large area at risk.

The circumflex branch (CX) runs along the mitral annulus to the lateral and posterior parts of the LV. Side branches perfuse the posterolateral, posterobasal, and not infrequently, also the inferolateral part of the left ventricle. Also atrial branches can branch off. This may lead to atrial infarction in case of occlusion or to sinus node dysfunction, because in 40% of cases, this structure is perfused by this branch. Only in a minority (10%), does the CX perfuse the interventricular and interatrial septum, including the AV node. The rare occasion of AV conduction delay or block in the setting of CX occlusion therefore identifies dominance of this vessel.

The RCA runs opposite to the CX in the groove between the right ventricle and atrium and perfuses the conus pulmonalis; the right atrium, including the sinus node (in 60% of cases); the right ventricle; the interventricular septum, including the posteromedial papillary muscle; the interatrial septum, including the AV node; the posterior wall; and sometimes also the posterolateral wall of the left ventricle. Occlusion of the RCA, therefore, may lead to distinct clinical features, such as bradycardia because of sinus node and AV node dysfunction (both also frequently aggravated by strong vagal discharges occurring in this setting), atrial infarction, right ventricular infarction, inferoposterior wall infarction of the left ventricle, and mitral regurgitation. Recognizing proximal RCA occlusion is therefore important so as to be prepared for these possible complications.

ST Elevation in Acute Myocardial Infarction. Electrodes facing the ischemic area record ST elevation in acute transmural myocardial ischemia. This is considered to be the consequence of differences in shape and height of the plateau phase of endocardial and epicardial action potentials. In anterior wall infarction ST elevation is observed in the precordial electrodes, at least in V_2 and V_3. In inferior wall infarction ST elevation occurs in at least leads II, III, and aVF. Lateral infarction is apparent in leads I, aVL, V_5, and V_6. The situation is more complex in posterior wall infarction because no standard leads face this part of the LV. The precordial leads record the reverse of ST elevation of the posterior wall, that is, ST segment depression. In clinical trials and in clinical practice, posterior wall infarction, mostly in the setting of CX disease, is underdiagnosed and therefore undertreated, resulting in increased morbidity and mortality.

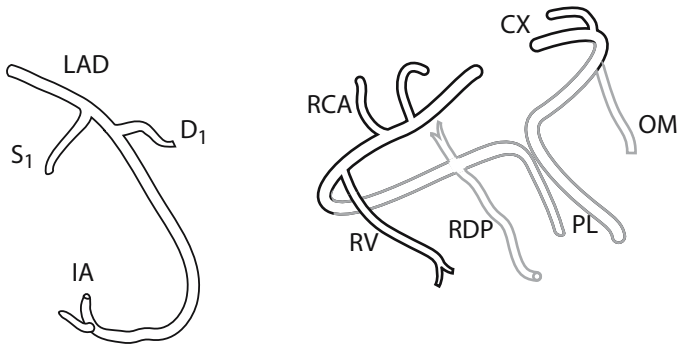

FIGURE 3.35. Scheme of the coronary artery system. See text.

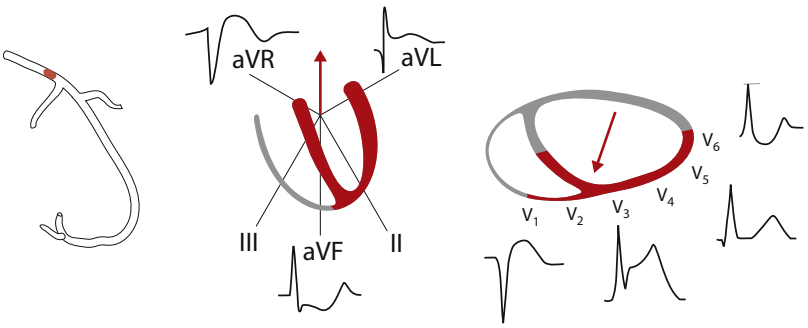

FIGURE 3.36. Proximal LAD occlusion in anterior wall myocardial infarction. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

In the following subsection the ischemia vector is used to explain the ECG changes in different forms of acute STEMI and the resulting assessment of the area at risk.

Acute Anterior Wall Myocardial Infarction. The LAD perfuses anterior, basal, apical, lateral, and frequently inferior parts of the LV (Fig. 3.35). The resulting ECG during obstruction depends on the involvement and size of these respective segments. The more proximal the occlusion, the more segments will be ischemic. By definition the anterior wall will always take part, resulting in an anterior direction of the ST vector in the transversal plane and thus in ST segment elevation in leads V_2 and V_3. The ST segment behavior in the other leads depends on the competing forces in the basal versus the apical area and the medial versus the lateral area. The apical part is smaller than the more basally located segments. Involvement of the septum and the lateral areas depends on the location of the obstructing lesion before a dominant septal or diagonal branch, most frequently being the proximal branches, perfusing the basal areas (Fig. 3.35).

Proximal Occlusion. An occlusion before the first septal and diagonal branch will lead to dominance of the basal part balanced between the septal and lateral segments. In the frontal plane the result will be an ST vector in the superior direction (Fig. 3.36). The ECG will show ST elevation in leads aVL and aVR and ST depression in the inferior leads. It should be noted that ST negativity in leads II, III, and aVF does not indicate absence of ischemia or subendocardial ischemia, but is the result of more dominant forces at the opposite site. In the transverse plane the vector will point anteriorly and sometimes even anteromedially leading to marked ST elevation in V_1 and ST depression in V_6 (Figs. 3.36 and 3.37A).

Distal Occlusion. In cases of a distal occlusion, below the dominant diagonal and septal side branches, the apical segments can be exposed leading to an inferior direction of the ST vector in the frontal plane (Figs. 3.37B and 3.38). The ECG now shows ST elevation or isoelectric ST segments in the leads II, III, and aVF, and ST depression in lead aVR and sometimes in aVL. The ST vector in the transversal plane is oriented in a lateral direction, which leads to ST elevation in lead V_6 and sometimes to ST depression in lead V_1 (Fig. 3.38).

Occlusion Behind the First Septal Branch. In cases in which the septal tree is spared, ischemia in the lateral

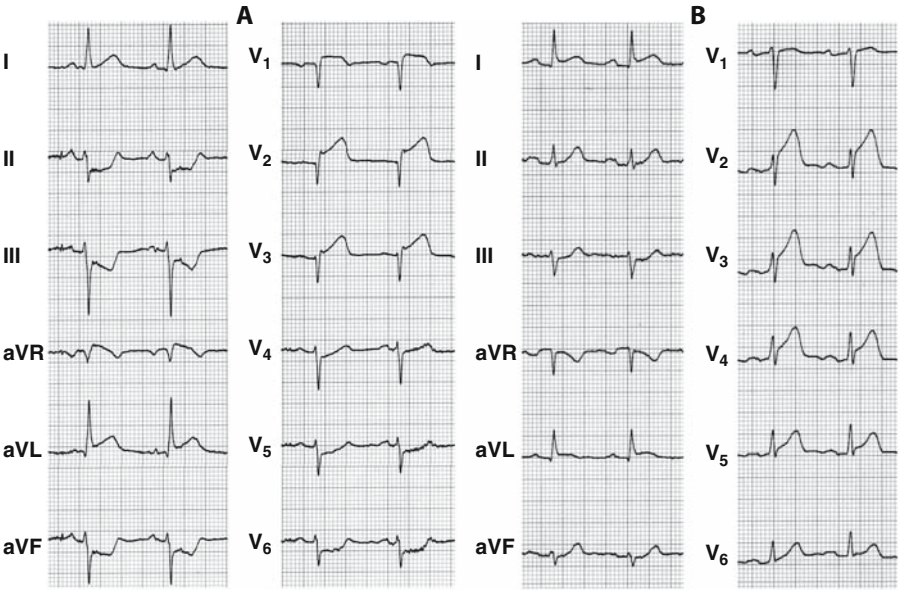

FIGURE 3.37. (A) Anterior wall myocardial infarction due to proximal LAD occlusion. Sinus rhythm, the ST segment vector is perpendicular to lead I, resulting in the frontal plane in ST elevation in aVL and aVR, and ST depression in the inferior leads. In the transverse plane typically there is ST depression in V_6. (B) Anterior wall myocardial infarction due to distal LAD occlusion. ST elevation is present in the precordial leads indicating anterior wall infarction. In the frontal plane the ST segments in the inferior leads are isoelectric, indicating distal occlusion behind the major septal and diagonal branches.

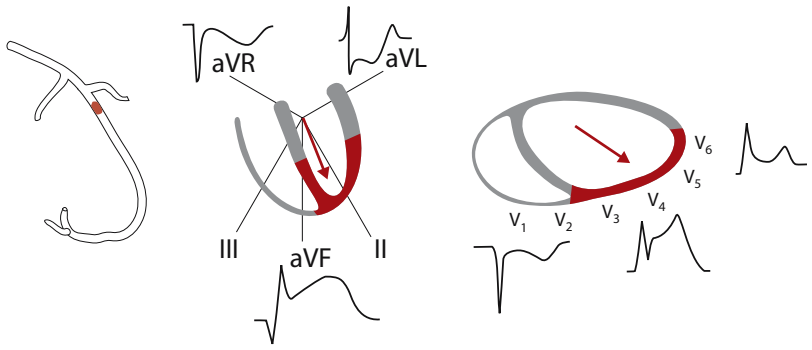

FIGURE 3.38. Distal LAD occlusion in anterior wall myocardial infarction. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

wall will dominate and the frontal ST vector will point laterally (Figs. 3.39 and 3.40A), frequently perpendicular to lead II. This leads to ST depression in leads III and aVR, ST elevation in lead aVL, and an isoelectric ST segment in lead II. In the transversal plane the ST behavior is similar to that in distal occlusion.

Occlusion Behind the First Diagonal Branch. Occasionally the first diagonal branch originates before the first septal branch or there is an anterolateral (intermediate) branch taking off between the LAD and CX. In these circumstances the occlusion can be located behind the first diagonal and before the first septal perforator (Figs. 3.40B and 3.41). The ST vector points medially in the frontal plane, leading to ST elevation in leads III and aVR, and sometimes to ST segment negativity in lead aVL. In the transverse plane similar behavior will be observed as in a proximal occlusion (see above).

The ECG findings as described have a high specificity, but a limited sensitivity to predict the correlation with the coronary anatomy. Most sensitive are ST depression in the inferior leads and ST elevation in aVR to predict a proximal occlusion, and absence of ST depression in the inferior leads to predict a distal occlusion (Table 3.10).[25]

Occlusion of a Diagonal Branch. Occlusion of a dominant mostly first diagonal branch or of an anterolateral branch results in ST elevation restricted to the leads V_2, V_3, I, and aVL. This picture should be differentiated from other STEMIs with ST elevation in I and aVL, such as a dominant marginal branch from the CX. In that case V_2 and V_3 will depict ST depression. When this same picture is present but also V_1 and aVR are elevated, left main or three-vessel disease have to be considered (see below).

Acute Non-Anterior Wall Myocardial Infarction. Non-anterior wall infarction comprises involvement of the posterior, inferior, and lateral parts and combinations. The culprit vessel could be either the RCA or the CX or one of its side branches.

The ECG gives information about which of both vessels is occluded and whether the right ventricle is involved. The latter points to a proximal RCA obstruction and identifies a high-risk situation with early and late hemodynamic and arrhythmic complications such as cardiogenic shock, sinus and AV node conduction impairment (sinus arrest or bradycardia, AV block of different degrees), and ventricular arrhythmias.

RCA Occlusion. In cases of RCA occlusion, the ischemia vector in the frontal plane will point in an inferomedial direction, because the RCA perfuses the right ventricle and the inferior part of the septum and of the left ventricle (Figs. 3.42 to 3.44). This leads to ST segment elevation in leads II, III, and aVF, ST being higher in lead III than in lead II. Consequently lead I will show ST depression. Usually also aVR and aVL show ST segment depression. Rarely ST elevation in aVR is observed. This identifies also more basally located ischemia, due to either a dominant posterior descending branch or to multivessel disease.

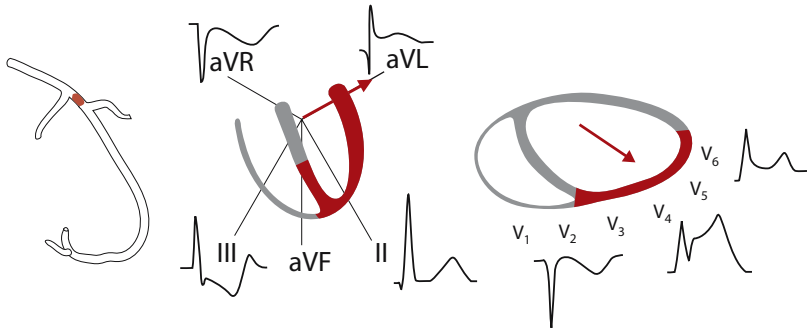

FIGURE 3.39. Anterior wall myocardial infarction with involvement of the first diagonal, but not the first septal branch. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

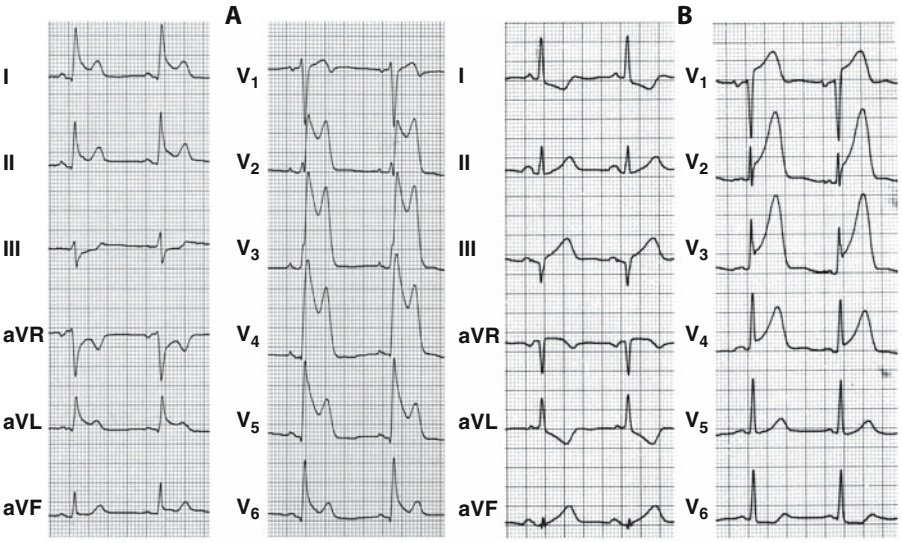

FIGURE 3.40. (A) Anterior wall myocardial infarction with involvement of the first diagonal, but not the first septal branch. Because of dominance ischemia of the lateral wall, the ST segment vector points perpendicular to aVF in lateral direction. Therefore, III, aVR, and aVF show ST depression and in the transverse plane also, VI has ST depression. (B) Anterior wall myocardial infarction with involvement of the first septal, but not the first diagonal branch. Because of dominance ischemia of the septum, the ST segment vector points perpendicular to aVF in medial direction. Therefore, III, aVR, and aVF show ST elevation and the ST segment in aVL may be depressed. In the transverse plane, V_1 has ST elevation and V_6 ST depression.

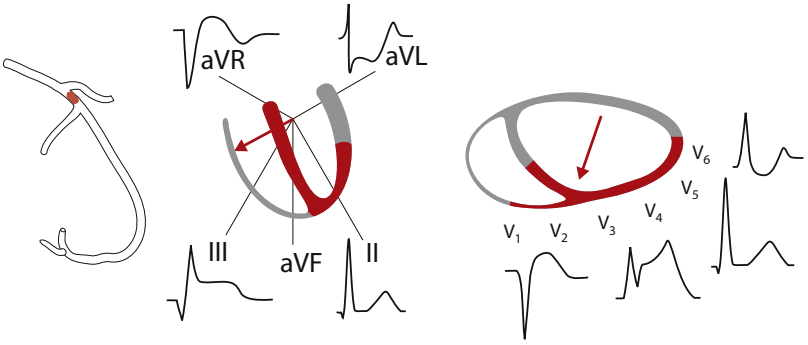

FIGURE 3.41. Anterior wall myocardial infarction with involvement of the first septal, but not the first diagonal branch. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

TABLE 3.10. Criteria to identify the occlusion site in the LAD

Criterion	*Occlusion site*	*Sens*	*Spec*	*PPA*	*NPA*
CRBBB	Proximal to S_1	14	100	100	62
ST↑V1 ≥2.5 mm	Proximal to S_1	12	100	100	61
ST↑aVR	Proximal to S_1	43	95	86	70
ST↓V_5	Proximal to S_1	17	98	88	62
Q aVL	Proximal to D_1	44	85	67	69
ST↓ II ≥1.0 mm	Proximal to S_1/D_1	34	98	93	68
Q V_5	Distal to S_1	24	93	71	53
ST↓ aVL	Distal to D_1	22	95	87	46
No ST↓ III	Distal to S_1/D_1	41	95	92	53

NPA, negative predictive accuracy; PPA, positive predictive accuracy; RBBB, right bundle branch block.

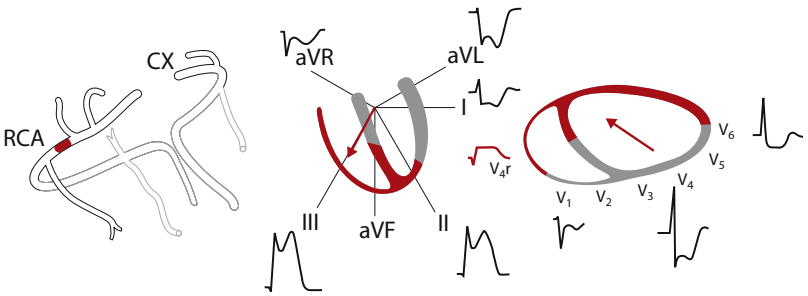

FIGURE 3.42. Inferior wall infarction due to proximal RCA occlusion. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

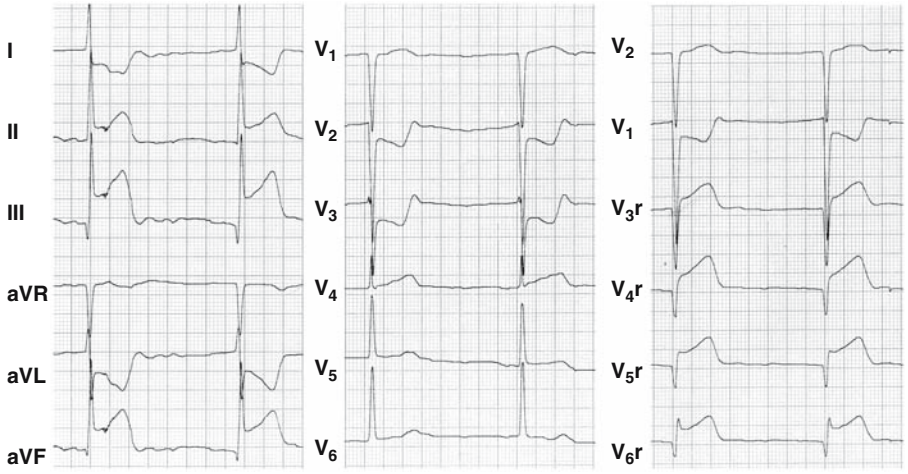

FIGURE 3.43. Inferior wall infarction due to proximal RCA occlusion. Atrial fibrillation, complete atrioventricular (AV) block, junctional escape rhythm with QRS configuration similar to conducted beats, ST elevation in leads II, III, and aVF, ST in III higher than in II, ST depression in lead I. In V_2 to V_3 ST depression due to posterior wall infarction and in the right precordial leads ST elevation as a sign of right ventricular involvement.

CX Occlusion. In CX occlusion the ischemia vector in the frontal plane points in an inferolateral direction, leading also to ST elevation in leads II, III, and aVF, but now lead II is equal to or higher than III. Consequently, in lead I the ST segment will be isoelectric or elevated (Figs. 3.45 and 3.46).

CX Side Branch. Circumflex branch occlusion or one of its side branches may also lead to posterolateral and even pure posterior wall infarctions (Figs. 3.47 and 3.48). The ECG will predominantly show ST depressions and may fail to have two contiguous leads with ST elevation. The latter is a guideline requirement to diagnose STEMI. This has led to underrecruitment of CX infarctions in clinical trials and, as stated previously, to underdiagnosis and undertreatment of patients with CX infarctions.

The Value of V_4R. The described ECG findings in the frontal plane correlate well with the culprit vessel but depend on the dominance of the vessel within the coronary system. More specific, therefore, is the assessment of involvement of the right ventricle, because this compartment is always perfused by the RCA. For this purpose, assessment of a rightward shift of the ischemia vector in the transverse plane is very useful. To be able to record this feature the use of right precordial leads V_3R to V_6R is needed. Lead V_4R has been found to be the most sensitive and specific lead. The direction of the ST vector in the transverse plane is determined by the involvement of the posterior wall, the posterior septum, and the right ventricular posterior and anterior wall. In cases of CX occlusion, only the posterior wall is involved, leading to an ischemia vector pointing in a posterior direction, apparent in the precordial leads V_1 to V_4, but also in lead V_4R as ST depression. When the distal RCA is the culprit, the posterior wall of the left ventricle and the posterior septum, but not the right ventricle, is involved. This leads to a slight rightward shift of the ST vector now coming more perpendicular to V_4R, resulting in an isoelectric ST segment. When the RCA is occluded in the proximal part, the ischemic right ventricle will shift the ST vector more to the right, and now the right precordial leads become positive.

Conduction Disturbances in STEMI. The conduction system is perfused by different coronary arteries (Fig. 3.49). All parts of the conduction system can be involved in STEMI. Sinus node and AV node dysfunction is frequently a feature of RCA or CX disease. Sinus bradycardia, sinoauricular block of different degrees, and sinus arrest may occur in this setting. First-degree AV nodal conduction delay, or Mobitz I (or Wenckebach block) or complete AV block may occur. Reperfusion usually leads to fast recovery. The LAD perfuses the His bundle and ischemia may induce prolonged PR interval, Mobitz II block, and complete AV block. The proximal part of the right bundle is perfused by the first septal perforator of the LAD, and the RBBB in this setting identifies a proximal LAD occlusion and a large area at risk. The RBBB has the typical QR configuration in lead V_1, due to loss of the initial septal r wave. Additional fascicular blocks may occur, anterior fascicular block more frequently than posterior hemiblock (Fig. 3.50). Both situations indicate increased risk of large infarctions, posterior more than anterior block. This is likely due to the double blood supply of

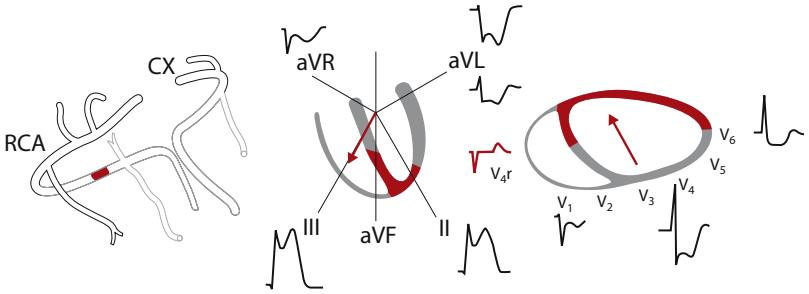

FIGURE 3.44. Inferior wall infarction due to distal RCA occlusion. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

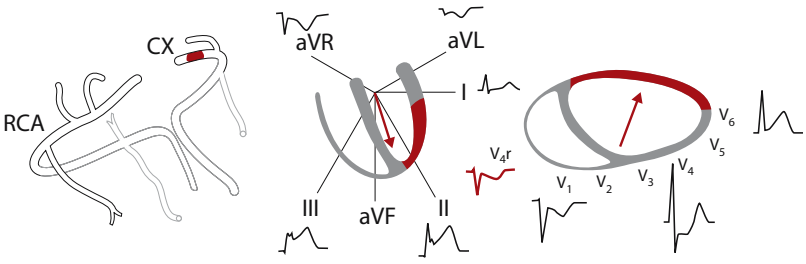

FIGURE 3.45. Inferior wall infarction due to CX occlusion. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

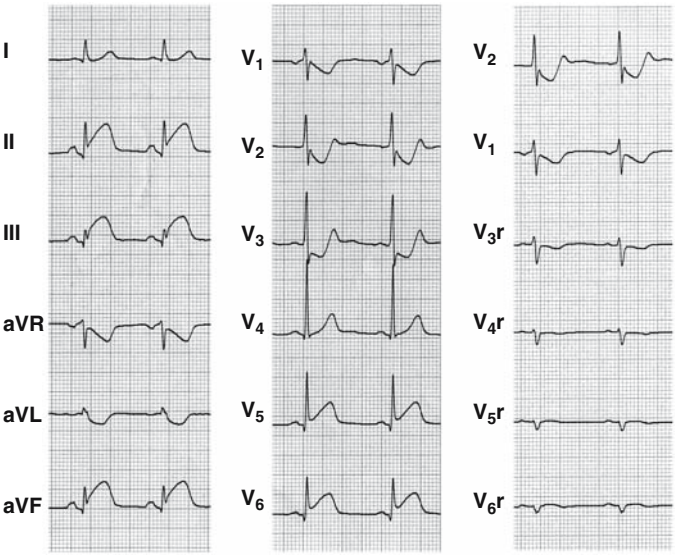

FIGURE 3.46. Inferior wall infarction due to CX occlusion. Sinus rhythm, ST elevation in the inferior leads, lead II equal to III, isoelectric ST segment in I, ST depression in aVR and aVL; ST depression V_2 and V_3 due to posterior wall infarction and ST elevation in V_5 to V_6 due to lateral infarction. The right precordial leads show ST depression indicative of CX occlusion.

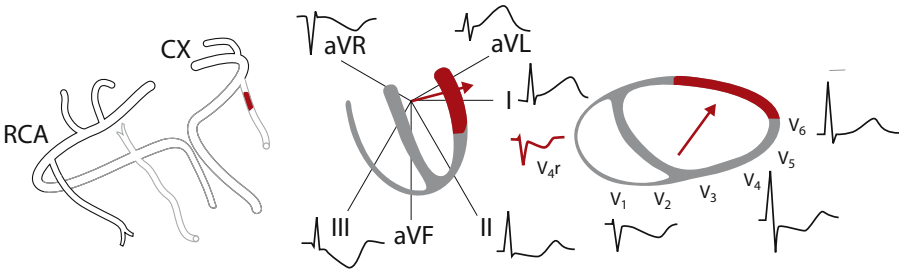

FIGURE 3.47. Occlusion of an obtuse posterolateral branch of the CX. Directional changes in the frontal and the transverse plane and the resulting changes in the ST segment.

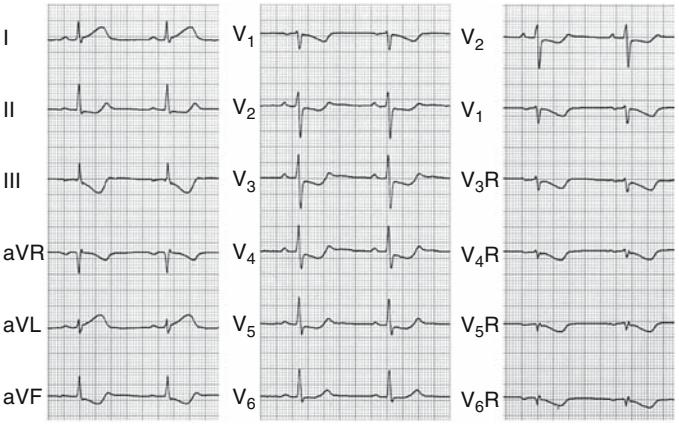

FIGURE 3.48. Posterolateral infarction with predominantly ST depression.

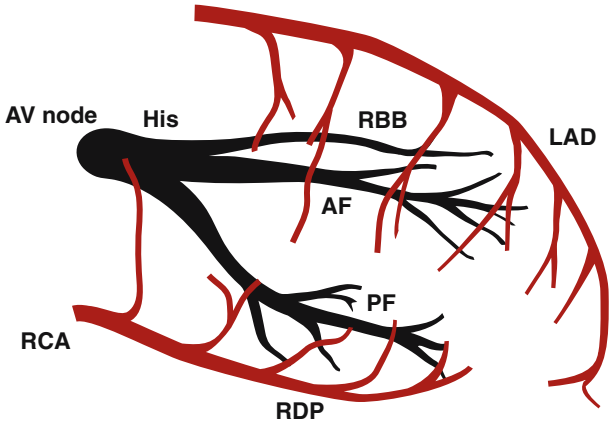

FIGURE 3.49. The AV conduction system and its blood supply. See text. AF, anterior fascicle; LAD, left anterior descending branch; His, bundle of His; PF, posterior fascicle; RBB, right bundle branch; RCA, right coronary artery; RDP, right descending posterior.

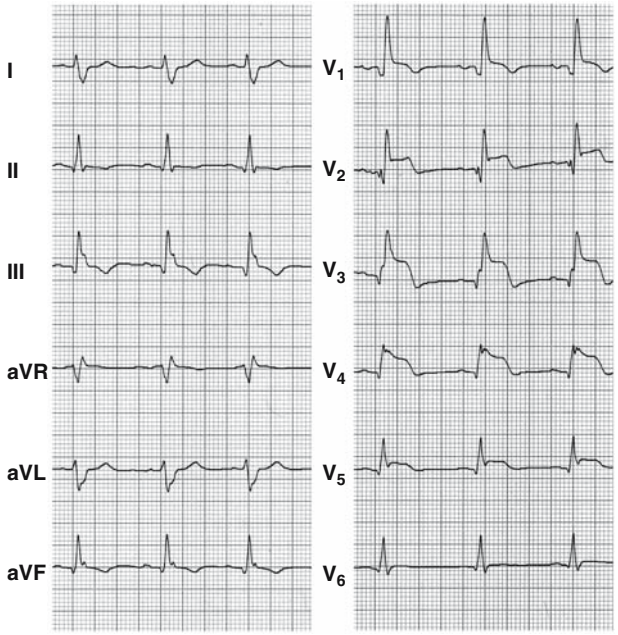

FIGURE 3.50. RBBB and posterior fascicular block in anteroseptal wall infarction. Sinus rhythm, right axis deviation, q wave in lead III, absence of septal q in RBBB.

the left bundle, that is, both from the LAD and from the RCA. Complete LBBB is a rare complication in the setting of STEMI, likely because of this double blood supply. This rare occasion, therefore, implies multivessel disease and an extensive area at risk (Fig. 3.51).

Acute Ischemia in Cases of a Widened QRS Complex. Widened QRS complexes occur in conditions such as BBB, ventricularly paced rhythms,[26] and preexcitation syndromes. Diagnosing acute infarction is complicated by the primary repolarization abnormalities occurring in these situations. Serial comparison of ECGs recorded within and without the ischemic episodes is helpful in identifying additional ischemic changes (Figs. 3.51 and 3.52). Criteria were developed in LBBB to help in diagnosing acute infarction.[27] These criteria are highly specific but their sensitivity is low (Table 3.11).

Isolated Right Ventricular Infarction. When only the right ventricle is ischemic, no counteraction of the posterior forces is present and this will lead to ST elevation in the standard precordial leads. Because the right ventricle is an inferior structure, some ST elevation in the inferior leads II, III, and aVF also may be present (Fig. 3.53). Recognizing RV infarction is important, because this condition may be confused with an LAD infarction. Usually, however, no grade 3, but rather grade 2 ischemia is present. Isolated right ventricular infarction is seen in three possible situations: (1) a nondominant RCA, (2) a collaterally perfused RCA, or (3) an isolated occlusion of a right ventricular branch.

Atrial Infarction. A frequently unrecognized condition is an atrial infarction. The ECG signs of atrial infarction are elevation of the Ta segment, the repolarization phase of the atria, in leads I, II, III, V_5, or V_6, or a depression in the precordial leads that may exceed 0.15 mV and 0.12 mV in leads I, II, and III (Fig. 3.54). Recognizing infarction of the atria is important because of its clinical implications. It can occur both in RCA or CX occlusion and indicates a proximal location of the lesion. Atrial infarction is often complicated by atrial fibrillation and also by more serious conditions, such as atrial thrombosis and even rupture of the atrial wall.

ST Recovery Following Reperfusion

Persistent ST deviation is the hallmark of transmural ischemia. In the preintervention era the ST segment gradually returned to baseline within the first 24 hours, frequently being accompanied by T-wave inversion and Q-wave formation. Resolution of ST segment elevation and T-wave

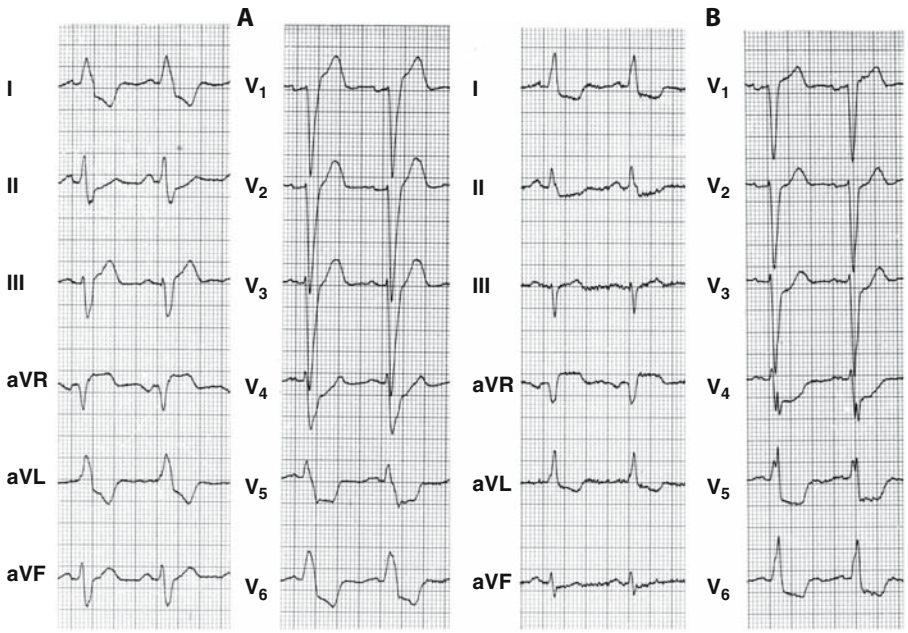

FIGURE 3.51. A. Ischemia-induced LBBB. During chest pain LBBB is present with additional ischemic ST segment changes. (B) On relief of chest pain, QRS widening diminishes indicating the ischemic nature of the conduction impairment. Rate-related LBBB as a mechanism is excluded because of the equal heart rates in both situations. Case of left main stenosis and multivessel disease.

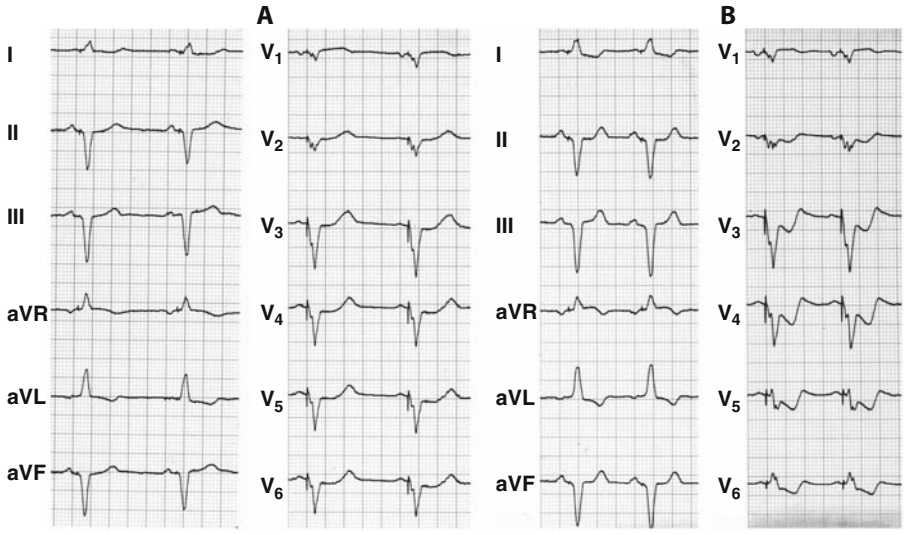

FIGURE 3.52. ST segment changes in ventricular paced rhythm. (A) Baseline. (B) During ischemia. Note the marked ST segment depression in the precordial leads during ischemia.

TABLE 3.11. Criteria to diagnose acute MI in LBBB

Criterion	*Odds ratio (95% conficence interval)*	*Score*
ST segment elevation ≥1 mm concordant with QRS complex	25.2 (11.6–54.7)	5
ST segment depression ≥1 mm in lead V_1, V_2, or V_3	6.0 (1.9–19.3)	3
ST segment elevation ≥5 mm discordant with QRS complex	4.3 (1.8–10.6)	2

To obtain a sensitivity of 78% and a specificity of 90%, the minimal total score should be 3 (modified from Sgarbossa et al.[26])

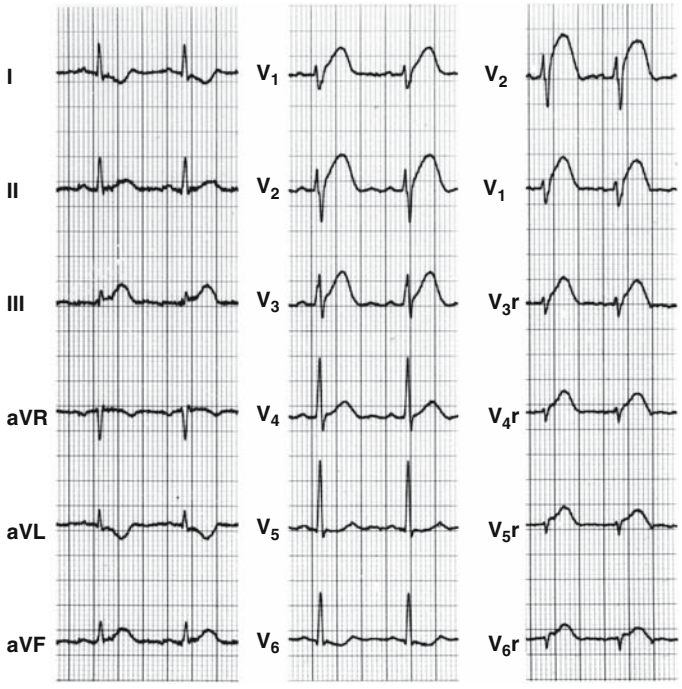

FIGURE 3.53. Isolated right ventricular infarction. ST elevation is present in leads II, III, and aVF, the ST vector is directed to the right +120 degrees (perpendicular to aVR). ST elevation is most pronounced in leads V_1 to V_4 and in the right precordial leads, due to the lack of counter forces from the posterior wall.

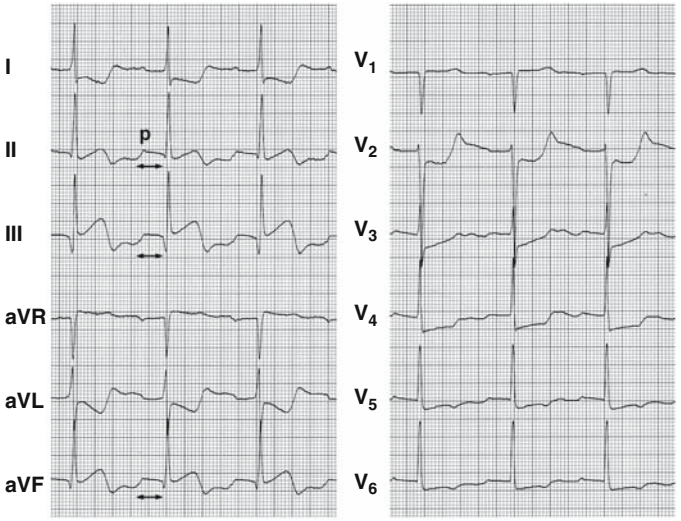

FIGURE 3.54. Atrial infarction. Inferoposterior wall infarction due to RCA occlusion. Ta segment elevation and prolonged atrial conduction or repolarization time, leading to merging of the P wave into the QRS complex.

inversion is accelerated when the occluded vessel is reopened, either by thrombolytic therapy or percutaneous coronary intervention. T-wave inversion is frequently one of the earliest signs of reperfusion, but is seen in only 60% of reperfused cases. The speed and completeness of ST segment normalization is a marker not only of reopening of the culprit vessel, but also of the quality of reperfusion at the tissue level. ST segment normalization of 30% or less is associated with poor reperfusion, 31% to 70% with moderate reperfusion, and 71% or more with good reperfusion. The amount of ST segment resolution correlates with clinical outcomes, such as heart failure and death, in the subacute and late phase after MI.

In about half of the patients receiving thrombolytic therapy and 10% of those with a PCI, the initial change is an increase of ST segment elevation at the time of onset of reperfusion, followed by ST segment normalization. Absence of ST segment resolution following thrombolytic therapy, especially in large infarctions, is an indication for PCI.

Continuous ST segment monitoring is the best way to document reflow in the infarct related coronary artery. It allows recognition of reopening, the quality of the reperfusion, and eventual reocclusion.

The onset of reperfusion is frequently accompanied by ventricular arrhythmias, the accelerated idioventricular rhythm (AIVR) being the most typical one (Fig. 3.55). These arrhythmias relate with worse quality of reperfusion at the tissue level.[28]

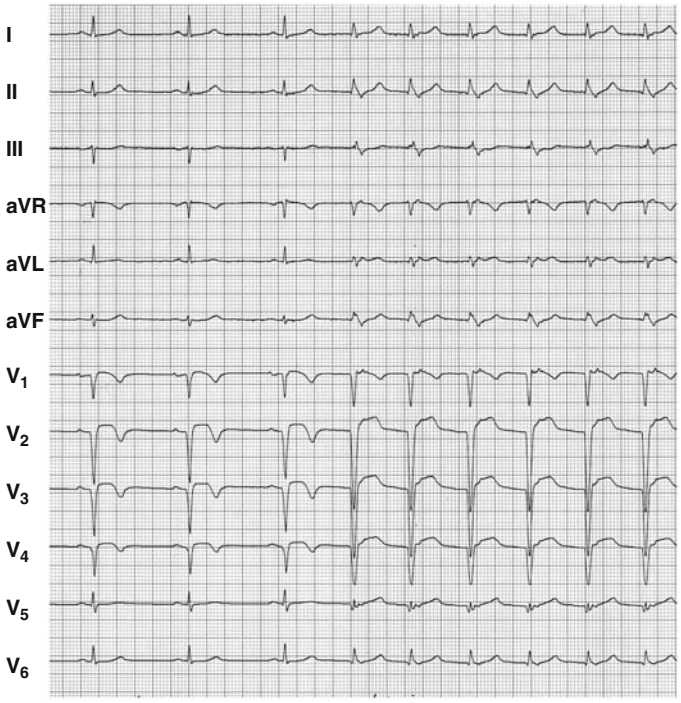

FIGURE 3.55. Accelerated idioventricular rhythm in reperfused acute anterior wall myocardial infarction.

Intraventricular Septal Rupture After STEMI

Acute infarctions are not infrequently complicated by rupture of the free wall, the interventricular septum, or a papillary muscle. Free wall rupture leads in most cases to electromechanical dissociation and sudden death. Rupture of a papillary muscle leads to severe acute pulmonary edema, and interventricular septal rupture leads to cardiogenic shock. In surviving patients the ECG is characterized by (1) sinus tachycardia, (2) subacute infarction evident as Q wave formation, and (3) persistent or recurrent ST elevation (Fig. 3.56).[29]

Left Ventricular Aneurysm

No or suboptimal reperfusion leads to persistent ischemia, hibernation, and finally necrosis or apoptosis of the myocardium with scar formation and not infrequently aneurysm formation. Electrocardiographically this leads to loss of QRS voltage or Q formation and persistence of ST segment elevation. Serious ventricular arrhythmias often develop in these situations.

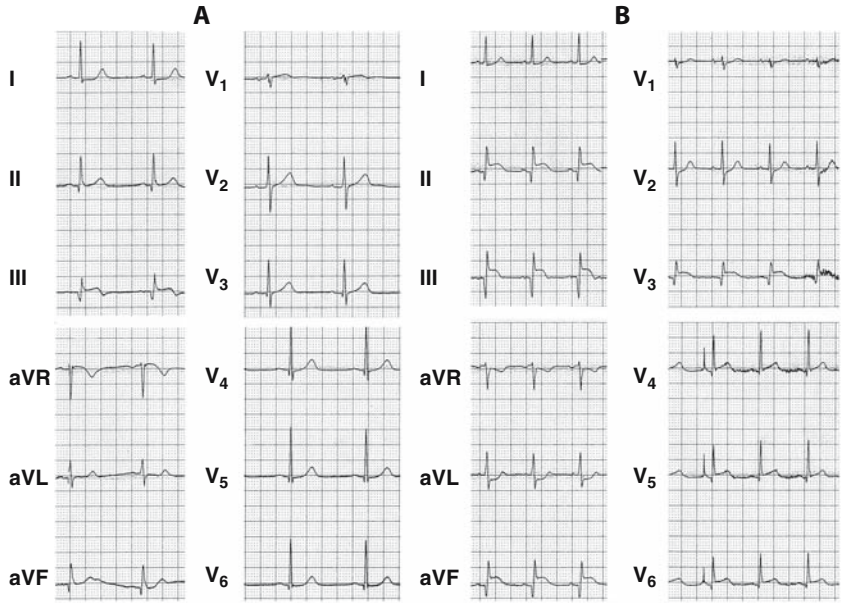

FIGURE 3.56. Interventricular septum rupture in inferior wall infarction. (A) Reperfused small inferior wall infarction with Q-wave formation and normalization of the ST segment, T-wave negativity in lead III. (B) Recurrent complaints with sinus tachycardia and new ST elevation.

Acute Pericarditis

Acute pericarditis leads to epicardial injury and in this way to generalized ST elevation, except for lead aVR (Fig. 3.57). Also part of the picture is Ta depression, best seen in lead II and V1. Pain, fever, or hemodynamic compromise leads to sinus tachycardia, and atrial fibrillation may occur. When pericardial effusion is present, it may lead to generalized loss of voltage of the P-QRS-T complex. Alternation of the QRS voltage is seen in hemodynamic compromising effusions, frequently in the setting of malignancies. In the subacute phase of pericarditis the ST segment normalizes and abnormal T waves are seen, such as flat or inverted T waves, which can remain for a prolonged period of time.

Non-STEMI

Non-STEMI comprises coronary syndromes with a seemingly less severe acute course, such as shorter lasting or spontaneously relieving chest pain, normalization of the ECG, and less increase of cardiac plasma enzymes. Therefore, the diagnostic and therapeutic strategies are frequently less aggressive and invasive than in STEMI patients. However, non-STEMI contains high-risk categories such as (1) left main stenosis, proximal three-vessel disease, and graft occlusion; (2) proximal LAD occlusion; and (3) as mentioned before, acute pure posterior wall infarctions. Left main and three-vessel disease frequently do not express as STEMI because the ECG changes are the consequence of a complex interplay due to subcritical stenoses at multiple sites, collateral circulation, and frequently already-old infarction(s). This leads to ST changes caused by endo- and epicardial ischemia in different sites in the heart.

Left Main and Three-Vessel Disease

One of the most critical conditions in clinical cardiology is ischemia due to left main stem stenosis. Frequently no complete obstruction is present, unless collateral circulation from the RCA has developed. In most instances in left main disease, multiple stenoses also are found in the other coronary arteries. Also not rarely an old MI is seen in the standard ECG. Recognizing left main disease is important in directing the patient without delay to the most optimal treatment strategy, most likely surgical coronary revascularization. The ECG in left main disease is similar to that of proximal three-vessel disease, and therefore they are discussed together.

The ischemia vector in left main disease in the frontal plane typically points in the superior direction, not infrequently even the right superior (extreme axis) direction, indicating involvement of the basal parts of the left ventricle. This leads to ST segment elevation in leads aVR and sometimes in lead III, whereas the other frontal leads show ST depression. In the precordial leads V_1 usually is elevated, whereas the other precordial leads show ST depression. Some specific features of the ECG pattern include (1) ST depression in eight leads or more in the 12-lead ECG[30]; (2) elevation in aVR and V_1; (3) being higher in aVR than in lead V_1[31]; (4) severe down-sloping ST configuration in the precordial leads, typically in lead V_4; and (5) ST elevation in lead III, not being present in lead II (Fig. 3.58).

ST vector in the Frontal Plane to Identify the High-Risk Patient

In the above discussion the usefulness of assessing the ischemia vector has been clarified. The ST vector can be used to identify patients with a large area at risk (Fig. 3.59). Vectors pointing inferiorly or laterally indicate lower risk than vectors in a superior or extreme direction. In cases of a posterior vector in the transversal plane inferomedial, the inferior and inferolateral or lateral direction indicates RCA or CX disease. When the vector is directed anteriorly, distal LAD disease is diagnosed. In case of a superior or extreme direction, the possibilities are high-risk situations such as proximal LAD disease and left main or three-vessel disease.

The T Wave

Ischemic T-Wave Behavior and Its Clinical Significance

During Ischemia

ST-segment elevation is a characteristic feature of acute transmural ischemia of ventricular myocardium. Also more discrete abnormalities due to ischemia may be observed

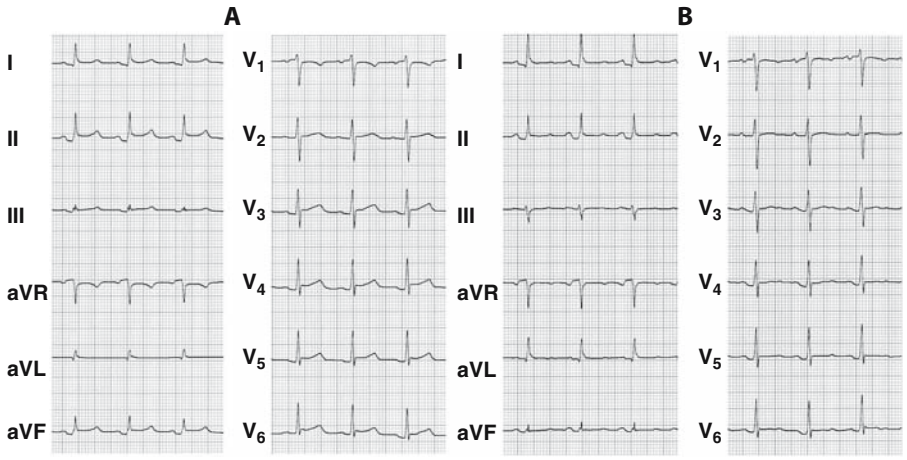

FIGURE 3.57. Acute pericarditis.

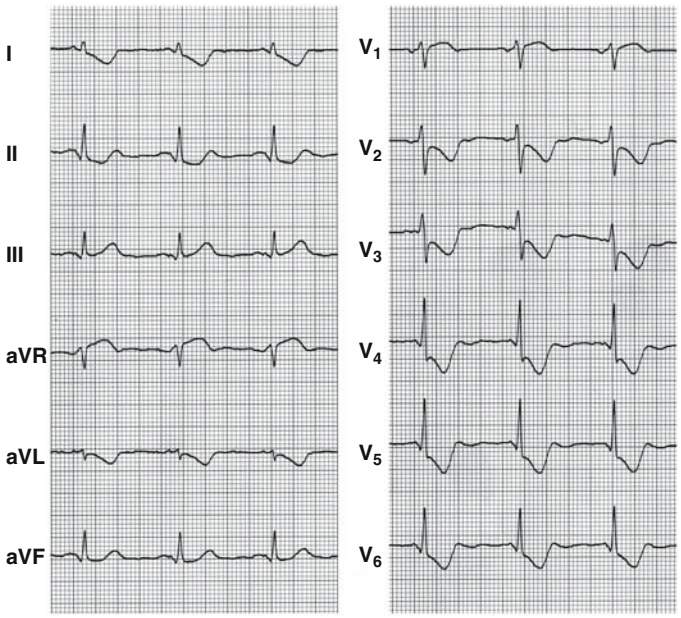

FIGURE 3.58. Left main and/or three-vessel disease. ST depression is present in most leads and is typically down sloping. Lead aVR shows more ST elevation than V_1.

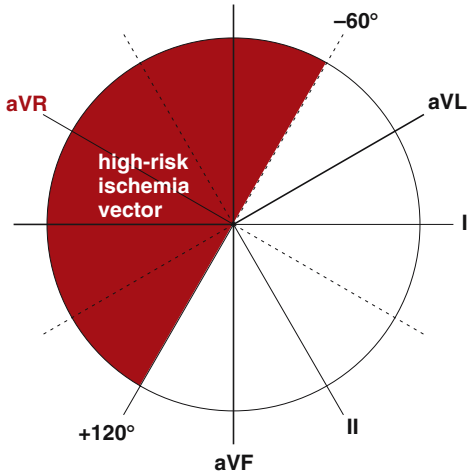

FIGURE 3.59. High-risk ST vector. ST segment vectors between –60 and 120 degrees, leading to ST elevation in aVR suggesting high-risk situations such as proximal LAD occlusion, left main and/or three-vessel disease.

during the repolarization phase of the ventricles: (1) Early, probably subendocardial, ischemia may present predominantly as shortening of the QT interval. It is observed in subtotal stenosis of the culprit artery or in complete obstruction in the presence of collateral circulation. (2) Peaked T waves are another early feature of acute ischemia and are classified as grade 1 ischemia[32] (Fig. 3.60). Pronounced ST-segment elevation leads to incorporation of the T wave in the ST segment. Severe ischemia with marked ST-segment elevation is sometimes accompanied by alternation of the ST segment and the T wave.[41–43]

After Ischemia

After an episode of chest pain, relieving spontaneously or by an intervention, negative T waves are commonly observed, both in unstable angina and in acute MI. Starting with negativity of only the terminal portion of the T wave, this is followed within hours or days by total negativity. In patients admitted with a normal ECG after an episode of chest pain, it is therefore helpful to repeat ECG recordings, because the possible rapid onset of T-wave negativity unmasks the ischemic nature of the chest complaints.

Site of T-Wave Abnormality in Relation to Coronary Anatomy

Generally, negative T waves are observed in the leads showing ST-segment elevation during chest pain. These changes may also be seen in other leads, suggesting a larger area of ischemia than suggested during the chest pain episode. Negative T waves in leads II, III, and AVF are related to inferior wall

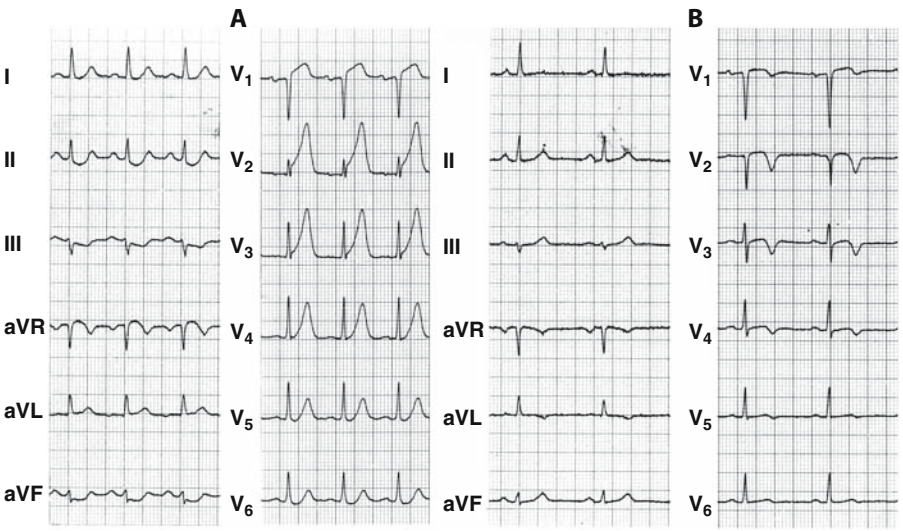

FIGURE 3.60. Peaked T waves during the acute phase of ischemia. The T-wave abnormalities are localized in the precordial leads, indicating a critical stenosis in the LAD. After relief of ischemia, terminal T-wave negativity is seen.

ischemia with either right coronary artery or circumflex branch occlusion and in the precordial leads to the anterior wall, being perfused by the left anterior descending branch. A diagnostic problem is presented by an ischemic event in the posterior wall. Here the postischemic changes are present as increased positivity of the T waves in the precordial leads.

The High LAD Syndrome

The development of negative T waves in the precordial leads from at least V_2 to V_4 after an episode of ischemia has been found to be useful to identify a subgroup of patients with increased risk of subsequent anterior wall STEMI or sudden cardiac death[33,34] (Fig. 3.60). Coronary angiographic correlations revealed invariably severe stenosis in the left anterior descending branch or total occlusion in the presence of collateral circulation. As the dominant vessel of the coronary artery system, recurrence of the obstruction will lead to extensive MI of the anterior wall.

T-Wave Changes Following Reperfusion in STEMI

As pointed out above, the occurrence of terminal T-wave negativity is also very helpful in assessing reperfusion during thrombolytic therapy in acute MI. It has been shown to be one of the earliest noninvasive signs that the infarct vessel is reopening[28,35] (Figs. 3.60B and 3.61A). Sensitivity, specificity, and the likelihood ratio in this study of terminal T-wave negativity to predict reperfusion were 63%, 94%, and 10.6%, respectively. Close ECG monitoring of this finding and other noninvasive signs of reperfusion, such as disappearance of chest pain, decrease of ST-segment elevation, the occurrence of ventricular premature beats with long coupling interval, and accelerated idioventricular rhythms during this phase, are useful to diagnose reperfusion.

Giant T Waves and QT Prolongation

After an ischemic event the QT interval may increase considerably. QT prolongation is sometimes combined with giant T-wave negativity (Fig. 3.61B). This latter phenomenon has been described to predict a good prognosis, as evidenced by recovery of R waves and preservation of left ventricular function.[35]

The Tako-Tsubo Syndrome

This novel syndrome (transient left ventricular apical ballooning syndrome and stress-induced cardiomyopathy) is another cause of temporary (giant) T-wave inversions, QT prolongation, and Q waves, most of these features resolving within days to weeks.[36,37] This syndrome of chest pain or heart failure is caused by severe sudden stress, occurs most frequently in women, and induces severe temporary apical dyskinesia in the setting of a normal coronary angiogram.[38]

Recovery of T-Wave Abnormalities Following Ischemia

In patients with non-STEMI because of proximal LAD stenosis who have survived at least 6 months after the ischemic event, normalization occurs within 6 weeks in half of this population and in 80% within 6 months. Similar findings are seen after balloon angioplasty, revealing normalization of the T wave in 90% of patients after 28 weeks.[39,40] Persistence of T-wave inversion is reportedly related to worse outcome in comparison with those with recovery of their T wave.

Other Causes of ST-T Wave Changes

Cardiac Memory

Cardiac memory refers to persistent T-wave changes that follow resumption of sinus rhythm after a period of altered activation sequence.[39] Prolonged alteration of activation sequence has a variety of causes including ventricular pacing, intermittent LBBB, ventricular tachycardia, ventricular extrasystoles, and ventricular preexcitation. T-wave changes are more prominent and have slower regression dependent on the duration and extent of abnormal activation.[40]

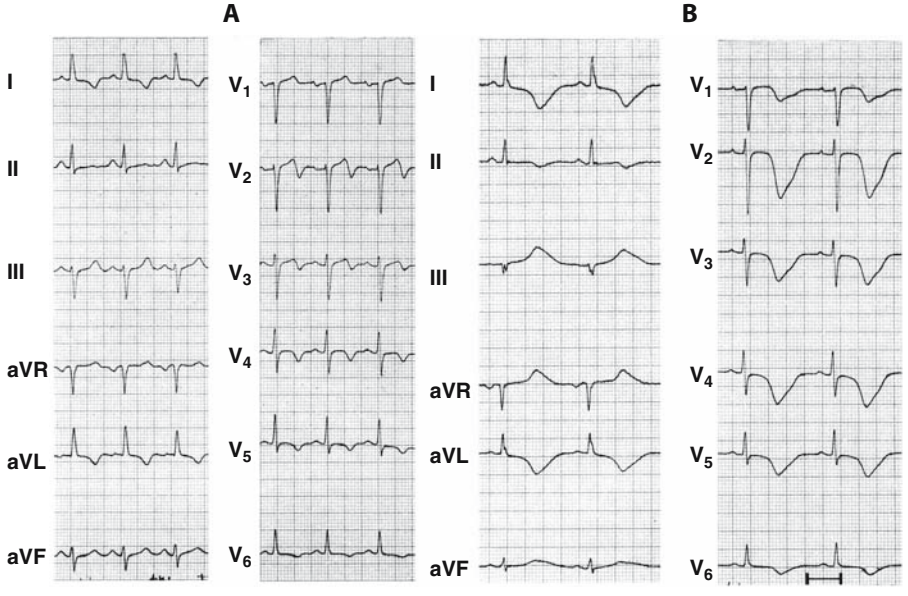

FIGURE 3.61. Postischemic T-wave changes and giant T waves. (A) ECG taken after relief of ischemic chest pain. Note terminal T-wave inversion. (B) One day later, giant negative T waves have developed.

The T-wave polarity during normalization of the QRS width is dependent on the QRS polarity during QRS widening: Negative QRS complexes during abnormal depolarization lead to negative T waves during normal activation and vice versa (Fig. 3.27).

Early Repolarization

Early repolarization has elevated, upward, concave ST segments, located commonly in precordial leads, with reciprocal depression in aVR; tall, peaked, and slightly asymmetrical T waves with notch; and slur on the R wave.[44] The other accompanying features in the ECG are vertical axis, shorter and depressed P-R interval, abrupt transition, counterclockwise rotation, presence of U waves, and sinus bradycardia. Males dominate and patients are often younger than 50 years of age. The incidence of 1% to 2% is found equally common in all races. Degree and incidence of ST elevation decrease as age advances. Exercise or isoproterenol administration may normalize the ST segment. Early repolarization is a benign condition. If the ECG conforms to a classic pattern of this syndrome on serial ECGs, it would exclude the unnecessary hazards of present-day revascularization therapy for MI such as primary angioplasty or thrombolytic therapy, or aggressive management of acute pericarditis.

Hypothermia

Hypothermia slows both conduction and repolarization, in this way prolonging all measured ECG intervals.[45–49] Also AV nodal block may be part of the picture. The presence of Osborn waves in hypothermic patients appears to be a function of temperature rather than the electrolyte or acid–base status (Fig. 3.62).[43] Below a temperature of 30°C, the J waves are detectable in 80% of patients. Ventricular fibrillation is a major risk at core body temperatures below 27°C.

The electrophysiologic mechanism of Osborn waves is suggested to be related to an epicardial–endocardial voltage gradient associated with the localized spike and dome morphology of I_{to}-mediated action potential in ventricular epicardium but not endocardium.[40]

Osborn waves are most commonly observed in hypothermia. Also other conditions reportedly may cause J waves, such as hypercalcemia, brain damage, cardiac arrest, Chagas' disease, ischemic heart disease, and the Brugada syndrome.

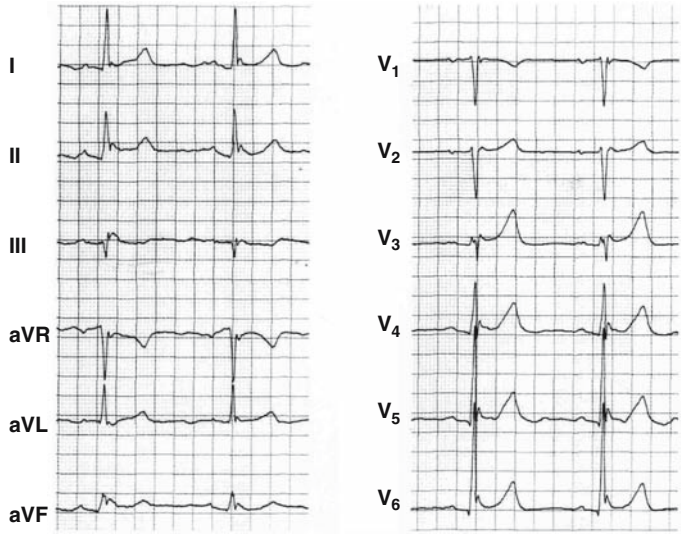

FIGURE 3.62. Hypothermia. Sinus bradycardia, late positive deflections in the QRS complex, known as Osborn waves, mild ST-segment elevation, most prominent in the anterolateral leads.

The Long QT Syndrome

The long QT syndrome is characterized by a prolonged QT interval and torsades de pointes ventricular arrhythmias, leading to collapse and sudden death. Hereditary and acquired forms exist, and recently the genetic aspects of the former and the pharmacologic aspects of the latter conditions have gained much attention. Characteristics of the ST-T interval have been described in relation to the genetic form of LQTS.[50,51] The ECG during sinus rhythm shows, besides the prolonged QT interval, a dynamic behavior of the T wave, such as T-wave alternation and bifid T waves, depending on the rate and regularity of the preceding rhythm and the state of the autonomic nervous system (Fig. 3.63).

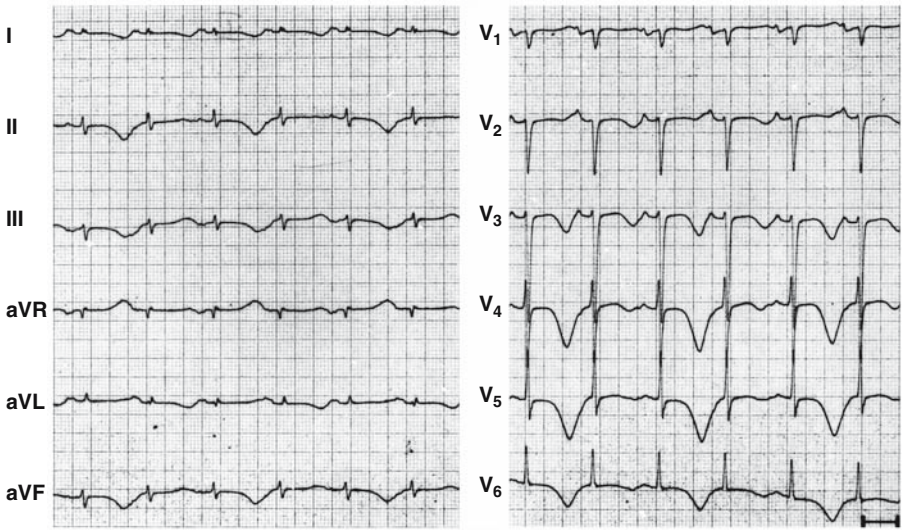

FIGURE 3.63. The long QT syndrome. Alternation of the T waves.

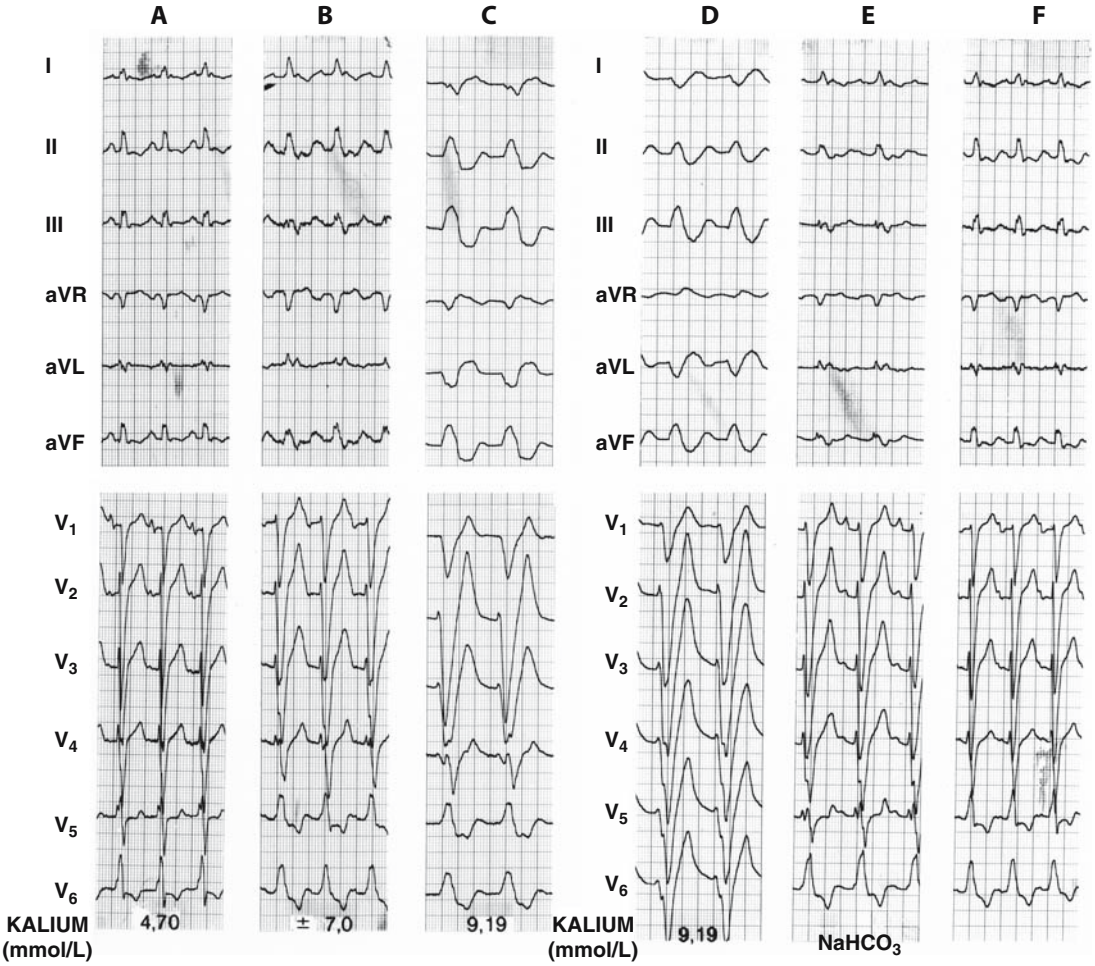

FIGURE 3.64. Hyperkalemia. Six panels showing increasing levels of serum potassium (A–D) and abrupt normalization on sodium bicarbonate infusion (E,F). Note the gradual flattening and disappearance of P wave, widening of the QRS complex, and occurrence of peaked and negative T wave. On treatment rapid normalization is seen.

Recently also a short QT syndrome was described as a familial cause of sudden cardiac death.[52]

Electrolyte Abnormalities[53]

Changes in Serum Potassium Concentration. The changes in the surface ECG are frequently correlated with the serum potassium level. However, no constant relationships between the potassium level and the ECG abnormalities exist. The more acute and severe the abnormalities, the better the correlation. Probably the ECG changes are the result of the extra-intracellular myocardial potassium gradient. This gradient is decreased in hyperkalemia and increased in hypokalemia.

Hyperkalemia. Hyperkalemia, frequently occurring in renal failure, extensive tissue damage, or adrenal dysfunction, may lead to profound changes in the ECG. Depending on the level of serum potassium, the cardiac action potential shows diminished diastolic polarization, slowing of phase 0, slowed conduction, and shortening of the action potential duration (Figs. 3.64 and 3.65). Above serum levels of 5.8 mmol/L the T wave become peaked and small, and with increasing levels ST segment depression and disappearance of the U

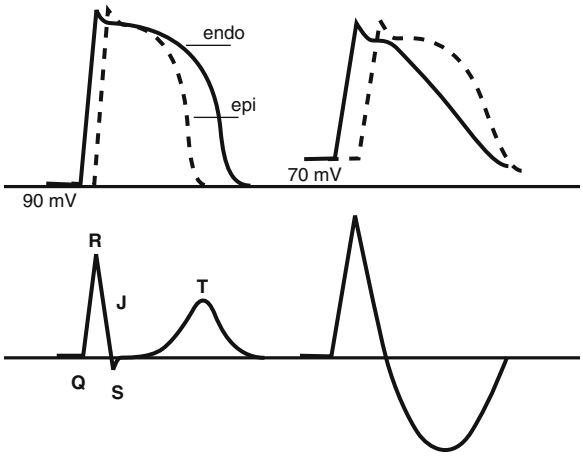

FIGURE 3.65. Hyperkalemia. Scheme of underlying mechanism. See text.

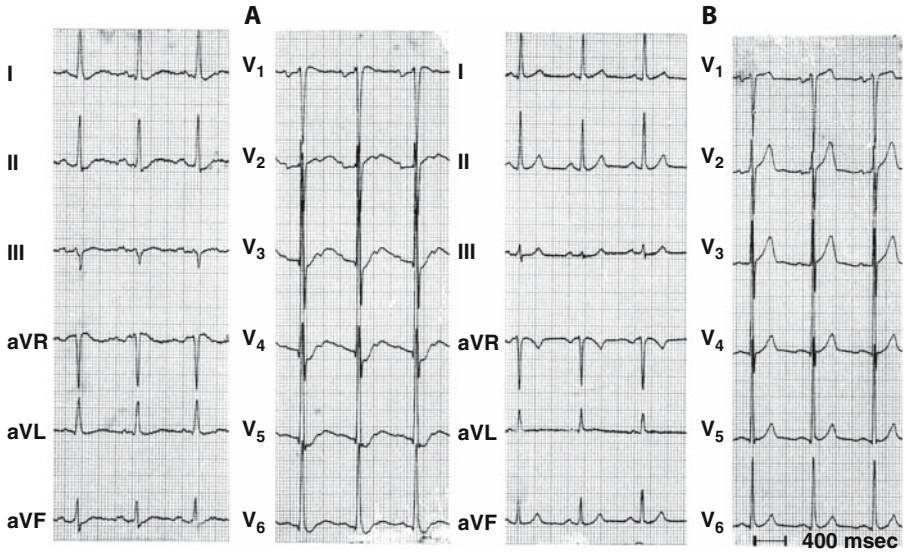

FIGURE 3.66. Hypokalemia. (A) Hypokalemia, as shown by the bifid T waves merging with U waves. (B) Normalization of repolarization waves after restoration of potassium concentration.

waves is seen. Above 6.k mmol/L atrial, atrioventricular and ventricular conduction impairment is seen. This leads to a decrease in and broadening of the P waves, a prolonged PR interval, and widening of the QRS complex and T-wave negativity. At higher levels no P waves are visible anymore and the widened QRS fuses with the T wave. This ECG picture may be confused with slow ventricular tachycardia. Indeed, lethal ventricular arrhythmias may occur in this setting. The ECG changes in hyperkalemia are more prominent in cases of concomitant hyponatremia, acidosis, or hypocalcemia.

Hypokalemia. Hypokalemia, a frequent complication of the use of diuretics, leads to decreasing potassium levels and to flattening of the T waves, ST depression, and increase of the U wave (Fig. 3.66). This further leads to increasing fusion of the T and U waves until both waves are not to be distinguished anymore. In this setting torsades de pointes arrhythmias may occur. Also digitalis-induced arrhythmias occur more frequently in hypokalemic states. The picture is enforced by concomitant hypercalcemia.

Changes in Serum Calcium Concentration. Calcium influences the duration of the plateau phase of the monophasic action potential. The ECG picture is dependent on the ionized calcium concentration rather than on the amount of protein bound calcium. The correlation between serum calcium levels and the ECG is better than in disturbances of the potassium metabolism. In hypocalcemia the repolarization phase is prolonged, especially at the expense of lengthening of the ST segment, whereas the QRS and T wave duration remain the same. In hypercalcemia QT shortening is seen, due to shortening of the ST segment, and in severe cases the T wave follows the QRS complex directly.

Summary

Electrocardiography is the graphic one-dimensional representation of the electrical activity of the heart as recorded from the body surface. The basic principle is the vectorial nature of the ECG, the deflections in the respective leads being the consequence of timing, direction, and strength of the electrical instantaneous forces. Standard recordings are derived from six leads in the frontal plane and six leads in the transversal plane. Additional (mostly right precordial) leads are used mainly for the purpose of diagnosing RV infarction. The ECG is especially helpful not only in diagnosing structural and functional aspects of cardiac disease but also in monitoring its natural history, in assessing its severity, in identifying the patient at risk, and in evaluating the effect of treatment. Structural changes of the four cardiac compartments are recognized, such as left and right atrial and ventricular hypertrophy, infarction, and cardiomyopathy. Also conduction disturbances in atria and ventricles can be diagnosed, leading to specific widening of the P wave and QRS complex. In the left and right bundle branch block, sequential activation of the ventricles is present, allowing more accurate assessment of each ventricle separately. Changes from the typical configuration of the right and left bundle branch block give information about additional disease affecting the heart.

The ECG has proven to be especially useful in diagnosing acute ischemia. ST elevation and non-ST elevation acute coronary syndromes are recognized, enabling the stratification of patients to different diagnostic and treatment strategies. Assessment of the acuteness and severity of the ischemia, the area at risk, and the coronary vessel involved can be derived from the surface ECG. For the latter purpose, the directional change of the ST segment, as depicted by the ST vector, was found to be very useful. ST vectors pointing in a rightward direction, leading to ST elevation in aVR, identify high-risk patients both in STEMI, such as in proximal LAD occlusion, and in non-STEMI, such as in main stem or three-vessel disease. In inferoposterior infarction, high-risk situations are recognized by diagnosing right ventricular involvement and atrial infarction. Recognizing conduction abnormalities in ischemic syndromes is also of help in identifying the high-risk patient.

The analysis of T-wave changes has been found to be important in acute, subacute, and chronic coronary syn-

dromes, and in a number of other important disorders, such as early repolarization, hypothermia, the long and short QT syndrome, and electrolyte abnormalities.

Acknowledgments

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