07 - 247 Electrocardiography

247 Electrocardiography

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An evidence-based approach, in Braunwald’s Heart Disease. A Textbook of Cardiovascular Medicine, 12th ed, RO Bonow et al (eds). Philadelphia, Elsevier/Saunders, 2022, p. 123. Jani V et al: The discerning ear: Cardiac auscultation in the era of artificial intelligence and telemedicine. Eur Heart J Digit Health 2:456, 2021. PART 6 Disorders of the Cardiovascular System Ary L. Goldberger

Electrocardiography An electrocardiogram (ECG or EKG) is a graphical representation of electrical activity generated by the heart. The signals, detected by means of metallic electrodes attached to the extremities and chest wall, are amplified and recorded by the electrocardiograph device. ECG leads are configured to display the instantaneous differences in electrical potentials between specific sets of electrodes. The utility of the ECG derives from its immediate availability as a noninvasive, inexpensive, and highly versatile test. In addition to its use in detecting arrhythmias, conduction disturbances, and myocardial ischemia/infarction, the ECG may reveal findings related to life-threatening metabolic distur bances, drug toxicities, and increased susceptibility to sudden cardiac arrest (see also Chaps. 317 and 420). The importance of electrocar diologic abnormalities in the diagnosis, prognosis, and management of muscular dystrophies and other hereditary neuromuscular diseases is discussed separately (see Chap. 360). ■ ■ELECTROPHYSIOLOGIC BACKGROUND Depolarization of the heart is the initiating event for cardiac contrac tion. The electrical currents that spread through the heart are produced by three components: cardiac pacemaker cells, specialized conduction tissue, and the heart muscle itself. The ECG records only the depolar ization (stimulation) and repolarization (recovery) electrical activity generated by the “working” atrial and ventricular myocardium (see also Chaps. 251 and 253). The stimulus initiating the normal heartbeat originates in the sinoatrial (SA) node (Fig. 247-1), which possesses spontaneous auto maticity. Spread of the depolarization wave through the right and left atria induces contraction of these chambers. Next, the impulse LA Sinoatrial (SA) node Ventricular myocardium AV junction AV node RA His bundle LV Purkinje fibers RV Left bundle branch Right bundle branch Ventricular septum FIGURE 247-1 Schematic of the cardiac conduction system. AV, atrioventricular; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

stimulates specialized conduction tissues in the atrioventricular (AV) nodal and His-bundle areas; together, these two regions constitute the AV junction. The bundle of His bifurcates into two main divisions, the right and left bundle branches, which rapidly transmit depolariza tion wavefronts in a synchronous way to the right and left ventricu lar myocardium via the Purkinje fibers. The main left bundle fans out into left anterior and left posterior fascicular subdivisions. The depolarization wavefronts then spread through the ventricular wall, from endocardium to epicardium, triggering

coordinated ventricu lar contraction. Since the cardiac depolarization and repolarization wavefronts have direction and magnitude, they can be represented by vectors. ■ ■BASIC ECG WAVEFORMS AND INTERVALS The ECG waveforms are labeled alphabetically, beginning with the P wave, which represents atrial depolarization (Fig. 247-2). The QRS complex represents ventricular depolarization; the ST-T-U complex (ST segment, T wave, and U wave) represents ventricular repolariza tion. The J point is the junction between the end of the QRS complex and the beginning of the ST segment. Atrial repolarization waveforms (ST-Ta) are usually too low in amplitude to be detected, but they may become apparent in acute pericarditis, atrial infarction, and AV heart block syndromes. The QRS-T waveforms of the surface ECG correspond to sequential phases of simultaneously obtained ventricular action potentials, the intracellular recordings from single myocardial fibers (Chap. 251). The rapid upstroke (phase 0) of the action potential corresponds to the onset of QRS. The plateau (phase 2) corresponds to the isoelectric ST segment, and active repolarization (phase 3) corresponds to the inscription of the T wave. Factors that decrease the slope of phase 0 by impairing the influx of Na+ (e.g., hyperkalemia and drugs such as flecainide) tend to increase QRS duration. Factors that prolong phase 2 or 3 (e.g., amiodarone, hypocalcemia) increase the QT interval. In contrast, factors (e.g., hypercalcemia, digoxin) associated with short ening of ventricular repolarization duration abbreviate the QT. The hereditary short QT syndrome and its relationship to sudden cardiac arrest are discussed in Chap. 262. The ECG is usually recorded on graph paper divided into a grid of 1-mm2 boxes. When the recording (sweep) speed is 25 mm/s, the smallest (1 mm) horizontal divisions correspond to 40 ms (0.04 s), with heavier lines at intervals of 200 ms (0.20 s). Vertically, the ECG graph measures the amplitude of a specific wave or deflection (1 mV = 10 mm with standard calibration; the voltage criteria for hypertrophy mentioned below are given in millimeters). There are four major sets of ECG intervals: RR, PR, QRS, and QT/QTc (Fig. 247-2). The instan taneous heart rate (beats per minute) can be computed from the inter beat (RR) interval by dividing the number of large (0.20 s) time units QRS T P ST U J PR interval QRS interval QT interval FIGURE 247-2 Basic ECG waveforms and intervals. Not shown is the RR interval, the time between consecutive QRS complexes.

between consecutive R waves into 300 or the number of small (40 ms) segments into 1500. The PR interval measures the time (normally 120–200 ms) between atrial and ventricular depolarization, which includes the physiologic delay imposed by stimulation of cells in the AV junction area. The QRS interval (normally 100–110 ms or less) reflects the duration of ventricular depo larization. The QT interval subtends both ventricular depolarization and (primarily) repolarization times and varies inversely with the heart rate. A variety of formulas has been proposed for computing a rate-corrected QT interval, termed QTc, but without formal consensus. The classic “square root” formula ($QTc = QT/\sqrt{RR}$, computed in second units) has been criticized for systematic errors at both lower and higher heart rates. One alternative is the Framingham formula (given here for units of mil liseconds): $QTc = QT + 0.154 (1000 – RR)$. The following upper normal limits (based visually on the longest QT) have been proposed: QTc of 460 ms in women and 450 ms in men. Lower limits are less well defined. Visual or electronic QT/QTc mea surements should be assessed in light of inherent limitations in their precise determination from standard ECGs waveforms. Right Left A FIGURE 247-3 The six frontal plane (A) and six horizontal plane (B) leads provide a threedimensional representation of cardiac electrical activity. ■ ■ECG LEADS The 12 conventional ECG leads are divided into two groups: six limb (extremity) leads and six chest (precordial) leads. The limb leads record potentials transmitted onto the frontal plane (Fig. 247-3A); the chest leads record potentials transmitted onto the horizontal plane (Fig. 247-3B). The orientation and polarity

of the frontal plane leads are repre sented on a hexaxial diagram (Fig. 247-4). The six chest leads are obtained by exploring electrodes as shown in Fig. 247-5. Each lead is analogous to a different video camera angle “look ing” at the same events—atrial and ventricular depolarization and repolarization—from different spatial orientations. The 12-lead ECG can be supplemented with additional leads in special circumstances. For example, right precordial leads V3R to V6R are useful in detect ing evidence of acute right ventricular ischemia/infarction. Bedside monitors and ambulatory ECGs (e.g., Holter monitors, event recorders, patch electrode and other medical wearable devices) usually employ

Le ft on ax ti is ia d ev ev d ia is –90° –aVF –60° –III ti ax on –120° –II e m e tr Ex –150° +aVR –30° +aVL

0° +I 180° –I

+30° –aVR +150° –aVL

Ri gh t ax s +60° +II +90° +aVF xi is +120° +III a d al ev m ia or ti N on

FIGURE 247-4 The frontal plane (limb or extremity) leads are represented on a hexaxial diagram. Each ECG lead has a specific spatial orientation and polarity. The positive pole of each lead axis (solid line) and the negative pole (hatched line) are designated by their angular position relative to the positive pole of lead I (0°). The mean electrical axis of the QRS complex is measured with respect to this display.

Posterior Superior CHAPTER 247 – – – – – Right Left + aVR aVL – ––– + + V6 – I + – – + V5 Electrocardiography + + + + + + + V4 V3 V2 V1 II III aVF B Anterior Inferior only one or two modified leads. The standard ECG leads are config ured such that a positive (upright) deflection is recorded in a lead if a wave of depolarization spreads toward the positive pole of that lead, and a negative deflection is recorded if the wave spreads toward the negative pole. If the mean orientation of the depolarization vector is at right angles to a particular lead axis, a biphasic (equally positive and negative) deflection will be inscribed. GENESIS OF THE NORMAL ECG ■ ■P WAVE The normal atrial depolarization vector is oriented downward and toward the subject’s left, reflecting the spread of depolarization from the sinus node to the right and then the left atrial myocardium. Since this vector points toward the positive pole of lead II and toward the negative pole of lead aVR, the sinus-generated P wave will be positive in lead II and negative in aVR. By contrast, activation of the atria from an ectopic pacemaker in the lower part of either atrium or in the AV junc tion region may produce retrograde P waves (negative in II, positive in aVR). The normal P wave in lead V1 may be biphasic with a positive component reflecting right atrial depolarization, followed by a small (<1 mm2) negative component reflecting left atrial depolarization. ■ ■QRS COMPLEX Normal ventricular depolarization proceeds as a rapid, continuous spread of activation wave fronts. This complex process can be divided V1 V2 V3R V3 V4 V5 V6 V4R FIGURE 247-5 The horizontal plane (chest or precordial) leads are obtained with electrodes in the locations shown. Additional posterior leads are sometimes placed on the same horizontal plane as V4 to facilitate detection of acute posterolateral infarction (V7, midaxillary line; V8, posterior axillary line; and V9, posterior scapular line). Right chest leads (V3R–V6R) may enhance detection of right ventricular involvement in the context of inferior infarction.

r RV LV

q V6 V1 PART 6 Disorders of the Cardiovascular System A V1 R r RV LV V1 V6

q B S – – – – – –

- V6 + V5 + + + + V1 V2 V3 V4 C FIGURE 247-6 Ventricular depolarization can be divided into two major phases, each represented by a vector. A. The first phase (arrow 1) denotes depolarization of the ventricular septum, beginning on the left side and spreading to the right. This process is represented by a small “septal” r wave in lead V1 and a small septal q wave in lead V6. B. Simultaneous depolarization of the left and right ventricles (LV and RV) constitutes the second phase. Vector 2 is oriented to the left and posteriorly, reflecting the electrical predominance of the LV. C. Vectors (arrows) representing these two phases are shown in reference to the horizontal plane leads. (Reproduced with permission from AL Goldberger et al: Goldberger’s Clinical Electrocardiography: A Simplified Approach, 10th ed. Philadelphia, Elsevier, 2024.) into two major sequential phases, and each can be represented by a mean vector (Fig. 247-6). The first and shortest phase is depolarization of the interventricular septum, proceeding from the left to the right and anteriorly (vector 1). The second and major phase results from the simultaneous depolarization of the right and left ventricles (vector 2). This phase is normally dominated by the more massive left ventricle, so that vec tor 2 points leftward and posteriorly. Therefore, a right precordial lead (V1) will record this biphasic depolarization process with a small positive deflection (septal r wave) followed by a larger negative deflection (S wave). A left precordial lead, for example, V6, will record the same sequence with a small negative deflection (septal q wave) fol lowed by a relatively tall positive deflec tion (R wave). Intermediate leads show a relative increase in R-wave amplitude (normal R-wave progression) and a decrease in S-wave amplitude progress ing across the chest from right to left. The lead where the R and S waves are of about equal amplitude is referred to as the transition zone (usually V3 or V4) (Fig. 247-7). aVR I II III FIGURE 247-7 Normal electrocardiogram from a healthy male subject. Sinus rhythm is present with a heart rate of 75 beats per minute. PR interval is 160 ms; QRS interval (duration) is 80 ms; QT interval is 360 s; QTc (Framingham formula) is about 390 ms; the mean QRS axis is about +70°. The precordial leads show normal R-wave progression with the transition zone (R wave ≈ S wave) in lead V3.

The QRS pattern in the extremity leads may vary considerably from one normal subject to another depending on the electrical axis of the QRS, which describes the mean orientation of the QRS vector with reference to the six frontal plane leads. Normally, the QRS axis ranges from –30° to +100° (Fig. 247-4). An axis more negative than –30° is referred to as left axis deviation, and an axis more positive than +90 to +100° is referred to as right axis deviation. Left axis deviation may occur as a normal variant but is more commonly associated with left ventricular hypertrophy, a block in the anterior fascicle of the left bundle system (left anterior fascicular block or hemiblock), or inferior myocardial infarction. Right axis deviation also may occur as a normal variant (particularly in children and young adults), as a spurious find ing due to reversal of the left and right arm electrodes, or in conditions such as right ventricular overload (acute or chronic), lateral infarction, dextrocardia, left pneumothorax, and left posterior fascicular block. ■ ■T WAVE AND U WAVE Normally, the mean T-wave vector is oriented roughly concordant with the mean QRS vector (within about 45° in the frontal plane). Since depolarization and repolarization are electrically

opposite processes, this normal QRS–T-wave vector concordance indicates that repolarization normally must proceed in the reverse direction from depolarization (i.e., from ventricular epicardium to endocardium). The normal U wave is a small, rounded deflection (≤1 mm) that follows the T wave and usually has the same polarity as the T wave. An abnormal increase in U-wave amplitude is most commonly due to hypokalemia or drugs (e.g., dofetilide, amiodarone, sotalol, quinidine). Very prominent U waves, as part of prolonged ventricular repolarization syndromes, are a marker of increased susceptibility to torsades de pointes (Chap. 253). MAJOR ECG ABNORMALITIES ■ ■CARDIAC ENLARGEMENT AND HYPERTROPHY Right atrial overload (acute or chronic) may lead to an increase in P-wave amplitude (≥2.5 mm) (Fig. 247-8), previously referred to as “P-pulmonale.” Left atrial overload typically produces a biphasic P wave in V1 with a broad negative component or a broad (≥120 ms), often with a notched P wave in one or more limb leads (Fig. 247-8). This pattern, historically referred to as “P-mitrale,” may occur with interatrial con duction delays in the absence of actual atrial enlargement, leading to the more general designation of left atrial abnormality. Right ventricular hypertrophy due to a sustained, severe pressure load (e.g., with pulmonic valve stenosis or certain pulmonary artery hypertension syndromes) is characterized by a relatively tall R wave in lead V1 (R ≥ S wave), usually with right axis deviation (Fig. 247-9); alternatively, there may be a qR pattern in V1 or V3R. ST depression and T-wave inversion in the right to mid-precordial leads are also V1 V4 V2 V5 aVL V3 V6 aVF

LA RA V1 Normal Right Left RA LA RA RA LA LA II RA RA RA V1 LA LA LA FIGURE 247-8 Right atrial (RA) overload may cause tall, peaked P waves in the limb or precordial leads. Left atrial (LA) abnormality may cause broad, often notched P waves in the limb leads and a biphasic P wave in lead V1 with a prominent negative component representing delayed depolarization of the LA. (Reproduced with permission from MK Park, WG Guntheroth: How to Read Pediatric ECGs, 4th ed. St. Louis, Mosby/Elsevier, 2006.) often present. This pattern, formerly called right ventricular “strain,” is attributable to repolarization abnormalities in acutely or chroni cally overloaded muscle. Prominent S waves may occur in the left lateral precordial leads. Right ventricular hypertrophy due to ostium secundum atrial septal defects, with the accompanying right ventricu lar volume overload, is commonly associated with an incomplete or complete right bundle branch block pattern in concert with a right ward QRS axis. Main QRS vector QRS in hypertrophy V1 V6 V1 Normal LVH RVH or or FIGURE 247-9 Left ventricular hypertrophy (LVH) increases the amplitude of electrical forces directed to the left and posteriorly. In addition, repolarization abnormalities may cause ST-segment depression and T-wave inversion in leads with a prominent R wave. Right ventricular hypertrophy (RVH) may shift the QRS vector to the right; this effect usually is associated with an R, RS, or qR complex in lead V1. T-wave inversions may be present in right precordial leads.

Acute cor pulmonale due to pulmonary thromboembolism (Chap. 290) or acute respiratory distress syndromes (e.g., COVID-19) may be associated with a normal ECG or a variety of abnormalities. Sinus tachycardia is the most common arrhythmia, although other tachyarrhythmias, such as atrial fibrillation or flutter, may occur. The QRS axis may shift to the right, sometimes in concert with the so-called S1Q3T3 pattern (prominence of the S wave in lead I and the Q wave in lead III, with T-wave inversion in lead III). Acute right ventricular dilation also may be associated with slow R-wave progression and ST-T abnormalities in V1 to V4 simulating acute anterior infarction. A right ventricular conduction disturbance may appear.

CHAPTER 247 Electrocardiography Chronic cor pulmonale due to obstructive lung disease (Chap. 307) usually does not produce the classic ECG patterns of right ventricular hypertrophy noted above. Instead of tall right precordial R waves, emphysema is more typically associated with diminished r waves in right to mid-precordial leads (slow R-wave progression) due in part to downward displacement of the diaphragm and the heart. Low-voltage complexes are commonly present, owing to hyperaeration. Multiple voltage criteria for left ventricular hypertrophy (Fig. 247-9) have been proposed based on the presence of tall left precordial R waves and deep right precordial S waves (e.g., SV1 + [RV5 or RV6] >35 mm). Repolar ization abnormalities (ST depression with T-wave inversions, formerly called the left ventricular “strain” pattern) may appear in leads with prominent R waves. However, prominent precordial voltages occur as a common normal variant, especially in athletic or young individu als. Left ventricular hypertrophy may increase limb lead voltage with or without increased precordial voltage (e.g., RaVL + SV3 >20 mm in women and >28 mm in men). The presence of left atrial abnormality increases the likelihood of underlying left ventricular hypertrophy in cases with borderline voltage criteria. Left ventricular hypertrophy often progresses to incomplete or complete left bundle branch block. The sensitivities of conventional voltage criteria for left ventricular hypertrophy are low in middle age to older adults and may be decreased further in obese persons and smokers, as well as with right bundle branch block. ECG evidence for left ventricular hypertrophy is a major noninvasive marker of increased risk of cardiovascular morbidity and mortality rates, including sudden cardiac death. However, because of false-positive and false-negative diagnoses, the ECG is of limited util ity in diagnosing atrial or ventricular enlargement. More definitive anatomic and functional information is provided by echocardiographic and cardiac magnetic resonance imaging studies (Chaps. 248 and A9). V6 ■ ■BUNDLE BRANCH BLOCKS AND RELATED PATTERNS Intrinsic impairment of conduction in either the right or the left bundle system (intraventricular conduction disturbances) leads to prolonga tion of the QRS interval. With complete bundle branch blocks, the widest QRS interval is ≥120 ms in duration; with incomplete blocks, the QRS interval is between about 110 and 120 ms. The QRS vector usually is oriented in the direction of the myocardial region where depolariza tion is delayed (Fig. 247-10). Thus, with right bundle branch block, the terminal QRS vector is oriented to the right and anteriorly (rSR′ in V1 and qRS in V6, typically). Left bundle branch block alters both early and later phases of ventricular depolarization. The major QRS vector is directed to the left and posteriorly. In addition, the normal early left-toright pattern of septal activation is disrupted such that septal depolar ization proceeds from right to left as well. As a result, left bundle branch block generates wide, predominantly negative (QS) complexes in lead V1 and entirely positive (R) complexes in V6. Waveform patterns identi cal to those of left bundle branch block, preceded by a sharp (sometimes very low amplitude) spike, are seen in most cases of electronic right ventricular pacing due to the relative delay in left ventricular activation. Bundle branch block may occur in a variety of conditions. In sub jects without structural heart disease, right bundle branch block is seen more commonly than left bundle branch block. Right bundle branch block also occurs with heart disease, both congenital (e.g., atrial septal defect) and acquired (e.g., valvular, ischemic). Left bundle branch block is often a marker of one of four underlying conditions associated with increased risk of cardiovascular morbidity and mortality rates: coronary

V6 V1 Normal PART 6 Disorders of the Cardiovascular System R′ R r RBBB T q S S LBBB T FIGURE 247-10 Comparison of typical QRS-T patterns in right bundle branch block (RBBB) and left bundle branch block (LBBB) with the normal pattern in leads V1 and V6. Note the secondary T-wave inversions (arrows) in leads with an rSR′ complex with RBBB and in leads with a wide R wave with

LBBB. heart disease (frequently with impaired left ventricular function), hyper tensive heart disease, aortic valve disease (including after transcatheter aortic valve replacement), and cardiomyopathy. Bundle branch blocks may be chronic or intermittent. A bundle branch block may be raterelated, most commonly observed when the heart rate exceeds some critical value. Bundle branch blocks and depolarization abnormalities second ary to artificial pacemakers not only affect ventricular depolariza tion (QRS) but also are characteristically associated with secondary repolarization (ST-T) abnormalities. With bundle branch blocks, the

T wave is typically opposite in polarity to the last deflection of the QRS (Fig. 247-10). This discordance of the QRS–T-wave vectors is caused by the altered sequence of repolarization that occurs as a consequence of altered depolarization. In contrast, primary repolarization abnor malities are independent of QRS changes and are related instead to actual alterations in the electrical properties of the myocardial fibers themselves (e.g., in the resting membrane potential or action potential duration), not just to changes in the sequence of repolarization. Isch emia, electrolyte imbalance, and drugs such as digoxin all cause such primary ST–T-wave changes. Primary and secondary T-wave changes may coexist. For example, T-wave inversions in the right precordial leads with left bundle branch block or in the left precordial leads with right bundle branch block may be important markers of underlying ischemia or other abnormalities. A distinctive abnormality simulating right bundle branch block with ST-segment elevations in the right chest leads is seen with the Brugada pattern (Chap. 262). Partial blocks in the left bundle system (left anterior or posterior fascicular blocks; formerly called hemiblocks) generally do not prolong ST V5 ST A B FIGURE 247-11 Acute ischemia causes a current of injury. A. With predominant subendocardial ischemia, the resultant ST vector will be directed toward the inner layer of the affected ventricle and the ventricular cavity. Overlying leads therefore will record ST depression. B. With ischemia involving the outer ventricular layer (transmural or epicardial injury), the ST vector will be directed outward. Overlying leads will record ST elevation.

the QRS duration substantially. Instead, they are associated with shifts in the frontal plane QRS axis (leftward or rightward, respectively). Left anterior fascicular block (QRS axis more negative than –45°) is prob ably the most common cause of marked left axis deviation in adults. In contrast, left posterior fascicular block (QRS axis more rightward than +110–120°) is extremely rare as an isolated finding and requires exclusion of other factors causing right axis deviation. Intraventricular conduction delays also can be caused by factors extrinsic (toxic) to the conduction system that slow ventricular conduction, particularly hyperkalemia or drugs (e.g., class 1 antiarrhythmic agents, tricyclic antidepressants, phenothiazines). Prolongation of QRS duration does not necessarily indicate a conduction delay but may be due to preexci tation of the ventricles via a bypass tract, as in Wolff-Parkinson-White (WPW) patterns (Chap. 256) and related variants. ■ ■MYOCARDIAL ISCHEMIA AND INFARCTION (See also Chap. 286) The ECG is central to the diagnosis of acute and chronic ischemic heart disease. Ischemia exerts complex timedependent effects on the electrical properties of myocardial cells. Severe, acute ischemia lowers the resting membrane potential and shortens the duration of the action potential. Such changes cause a voltage gradient between normal and ischemic zones. As a con sequence, current flows between those regions. These currents of injury are represented on the surface ECG by deviation of the ST segment (Fig. 247-11). When the acute ischemia is transmural, the ST vector usually is shifted in the direction of the outer (epicardial) layers, producing ST elevations and sometimes, in the earliest stages of ischemia, tall, positive so-called hyperacute T waves over the ischemic zone. With ischemia confined primarily to

the subendocardium, the ST vector typically shifts toward the subendocardium and ventricular cav ity, so that overlying (e.g., anterior precordial) leads show ST-segment depression (with ST elevation in lead aVR). Multiple factors affect the amplitude of acute ischemic ST deviations. Profound ST elevation or depression in multiple leads usually indicates very severe ischemia. The division of acute myocardial infarction due to obstructive coronary artery disease into ST-segment elevation and non-ST elevation types is useful since the consistent efficacy of emergency (minutes to hours) reperfusion therapy is limited to the former group. Indications for acute reperfusion therapy in non-ST elevation myocardial infarction are a focus of ongoing investigation (Chap. 285). Takotsubo syndrome, as well as other causes of myocardial infarction without atherosclerotic coronary disease, can simulate the patterns of acute or evolving ST-seg ment elevation or non-ST-segment elevation infarction (Chap. 285). The ECG leads are usually more helpful in localizing regions of ST elevation than non-ST elevation ischemia. For example, acute transmu ral anterior (including apical and lateral) wall ischemia is reflected by ST elevations or increased T-wave positivity in one or more of the precor dial leads (V1–V6) and leads I and aVL. Inferior wall ischemia produces changes in leads II, III, and aVF. “Posterior” wall ischemia (almost always associated with lateral or inferior involvement) may be indirectly recognized by reciprocal ST depressions in leads V1 to V3 (thus constitut ing an ST elevation “equivalent” acute coronary syndrome). Acute right ventricular ischemia usually produces ST elevations in right-sided chest leads (Fig. 247-5). When ischemic ST elevations occur as the earliest sign of acute infarction, they typically are followed within a period ranging from hours to days by evolving T-wave inversions and often by Q waves occurring in the same lead distribution. Reversible transmural ST ST V5

V1 V2 V4 V5 V6 V3 FIGURE 247-12 Severe anterior wall ischemia (with or without infarction) may cause prominent T-wave inversions in the precordial leads and in leads I and aVL. This pattern (sometimes referred to as the Wellens T wave sign) is usually associated with a high-grade stenosis of the left anterior descending coronary artery. ischemia, for example, due to coronary vasospasm (Prinzmetal’s angina) may cause transient ST-segment elevations without development of Q waves. Depending on the severity and duration of ischemia, ischemic ST elevations may resolve completely in minutes or be followed by T-wave inversions that persist for hours or even days. Patients with ischemic chest pain who present with deep T-wave inversions in mul tiple precordial leads (e.g., V1–V4, and sometimes I and aVL) with or without cardiac enzyme elevations typically have severe obstruction in the left anterior descending coronary artery (Fig. 247-12). With infarction, depolarization (QRS) changes often accompany repolarization (ST-T) abnormalities. Necrosis of sufficient myocardial tissue may lead to decreased R-wave amplitude or abnormal Q waves (even in the absence of transmural ischemia) in the anterior or inferior leads (Fig. 247-13). Abnormal Q waves were once considered markers of transmural myocardial infarction, whereas subendocardial infarcts were thought not to produce Q waves. However, transmural infarcts may occur without Q waves, and subendocardial (nontransmural) infarcts may be associated with Q waves. Therefore, evolving or chronic infarcts are more appropriately classified as “Q-wave” or “non-Q-wave” (Chap. A7). Loss of depolarization forces due to posterior or lateral infarction may cause reciprocal increases in R-wave amplitude in leads V1 and V2 without diagnostic Q waves in any of the conventional leads. (Additional leads V7–V9 may show acute changes.) In the weeks and months after infarction, these ECG changes may persist or begin to resolve. Complete normalization of the ECG after Q-wave infarction is uncommon but may occur, particularly with smaller infarcts. In con trast, ST-segment elevations that persist for several weeks or more after a Q-wave infarct usually correlate with a severe underlying wall motion disorder, although not

necessarily a frank ventricular aneurysm. A ECG sequence with anterior ST-elevation/Q-wave infarction I II III Acute Evolving ECG sequence with inferior ST-elevation/Q-wave infarction B I II III Acute Evolving FIGURE 247-13 Sequence of depolarization and repolarization changes with acute and evolving anterior (A) and (B) inferior ST-elevation/Q-wave infarctions. With anterior infarcts, ST elevation in leads I and aVL and the precordial leads may be accompanied by reciprocal ST depressions in leads II, III, and aVF. Conversely, acute inferior (or posterolateral) infarcts may be associated with reciprocal ST depressions in leads V1 to V3. (Reproduced with permission from AL Goldberger et al: Goldberger's Clinical Electrocardiography: A Simplified Approach, 10th ed. Philadelphia, Elsevier, 2024.)

CHAPTER 247 Electrocardiography The ECG has important limitations in both sensitivity and speci ficity in the diagnosis of acute and chronic ischemic heart disease. Although a single normal ECG does not exclude ischemia or even acute infarction, a normal ECG throughout the course of an acute infarct is distinctly uncommon. Prolonged chest pain without diagnostic ECG changes therefore should always prompt a careful search for other non coronary causes of chest pain (Chap. 15). Furthermore, the diagnostic changes of acute or evolving ischemia are often masked by the presence of left bundle branch block, electronic ventricular pacemaker patterns, and WPW preexcitation. However, clinicians may also overdiagnose ischemia or infarction based on the presence of ST-segment elevations or depressions; T-wave inversions; tall, positive T waves; or Q waves not related to ischemic heart disease (pseudoinfarct patterns). For example, ST-segment elevations simulating acute ischemia/infarction may occur with acute pericarditis or myocarditis, including COVID-19 infections, as a normal variant (including the typical "early repolarization" pat tern), or in a variety of other conditions (Table 247-1). Similarly, tall T waves do not invariably represent hyperacute ischemic changes but may also be caused by normal variants, hyperkalemia, or cerebrovas cular injury, among other causes. ST-segment elevations and tall, positive T waves are common find ings in leads V1 and V2 in left bundle branch block or left ventricular hypertrophy in the absence of ischemia. The differential diagnosis of Q waves includes physiologic or positional variants, ventricular hyper trophy, acute or chronic noncoronary myocardial injury, hypertrophic cardiomyopathy, and ventricular conduction disorders. Ventricular hypertrophy, hypokalemia, drugs such as digoxin, and a variety of other factors may cause ST-segment depression mimicking suben docardial ischemia. Prominent T-wave inversion may occur with aVR aVL aVF V2 V4 V6 aVR aVL aVF V2 V4 V6

TABLE 247-1 Differential Diagnosis of ST-Segment Elevations Myocardial ischemia/infarction Noninfarction transmural ischemia (e.g., Prinzmetal's syndrome) Acute myocardial infarction Due to atherosclerotic coronary occlusion Due to nonatherosclerotic causes (e.g., takotsubo syndrome, coronary PART 6 Disorders of the Cardiovascular System dissection) Post-myocardial infarction (left ventricular motion abnormality/aneurysm) Acute pericarditis Normal variants (including benign "early repolarization" patterns) Left ventricular hypertrophy/left bundle branch blocka Other (rarer) Acute pulmonary embolisma Brugada patterns (right bundle branch block–like morphology with ST elevations in right precordial leads) Class 1C antiarrhythmic drugsa DC cardioversion (transient) Hypercalcemiaa Hyperkalemiaa Hypothermia (J [Osborn] waves) Nonischemic myocardial injury Myocarditis syndromes (infectious and noninfectious) Tumor invading left ventricle Trauma to ventricles aUsually localized to V1–V2 or V3. Source: Modified from AL Goldberger et al: Goldberger's Clinical Electrocardiography: A Simplified Approach, 10th ed. Elsevier, 2024. ventricular hypertrophy, cardiomyopathies, myocarditis, and "stress cardiomyopathies" associated

with takotsubo syndrome and cerebro vascular injury (particularly intracranial bleeds), among others causes. Diagnostic confusion may also occur when nonischemic T-wave inver sions (“cardiac memory” effect) appear in normally conducted beats in patients with intermittent wide QRS complexes, most commonly due to ventricular pacing or to left bundle branch block. ■ ■METABOLIC FACTORS AND DRUG EFFECTS A variety of metabolic abnormalities and pharmacologic agents alter the ECG and, in particular, cause changes in repolarization (ST-T-U) and sometimes QRS prolongation. Certain life-threatening electrolyte disturbances may be diagnosed initially and monitored from the ECG. Hyperkalemia produces a sequence of changes (Fig. 247-14), usually Hyperkalemia Mild-Moderate Moderate-Severe Very Severe T V1 V1 P T V2 V2 P FIGURE 247-14 The earliest ECG change with hyperkalemia is usually peaking (“tenting”) of the T waves. With further increases in the serum potassium concentration, the QRS complexes widen, the P waves decrease in amplitude and may disappear, and finally a sine-wave pattern leads to asystole unless emergency therapy is given. (Reproduced with permission from AL Goldberger et al: Goldberger’s Clinical Electrocardiography: A Simplified Approach, 10th ed. Philadelphia, Elsevier, 2024.)

beginning with narrowing and peaking (tenting) of the T waves. Fur ther elevation of extracellular K+ leads to AV conduction disturbances, diminution in P-wave amplitude, and widening of the QRS interval. Severe hyperkalemia eventually causes cardiac arrest with a slow sinu soidal type of mechanism (“sine-wave” pattern) followed by asystole. Hypokalemia (Fig. 247-15) prolongs ventricular repolarization, often with prominent U waves. Prolongation of the QT interval is also seen with drugs that increase the duration of the ventricular action poten tial: class 1A antiarrhythmic agents and related drugs (e.g., quinidine, procainamide, tricyclic antidepressants, phenothiazines) and class III agents (e.g., amiodarone [Fig. 247-15], dofetilide, sotalol, ibutilide). Systemic hypothermia (Fig. 247-15) also prolongs repolarization, usu ally with a distinctive convex elevation of the J point (Osborn wave) and bradycardia. Marked QT prolongation, sometimes with deep, wide T-wave inversions, may occur with intracranial bleeds, particularly subarachnoid hemorrhage (“CVA T-wave” pattern) (Fig. 247-15). Hypocalcemia typically prolongs the QT interval (ST portion), whereas hypercalcemia shortens it (Fig. 247-16). Digitalis glycosides also shorten the QT interval, often with a characteristic “scooping” of the ST–T-wave complex (digitalis effect). ■ ■NONSPECIFIC ST-T CHANGES AND LOW QRS VOLTAGE Many other factors are associated with ECG changes, particularly alter ations in ventricular repolarization. T-wave flattening, minimal T-wave inversions, or slight ST-segment depression (“nonspecific ST–T-wave changes”) may occur with a variety of electrolyte and acid-base distur bances, infectious or inflammatory processes, central nervous system disorders, endocrine abnormalities, many drugs, ischemia, hypoxia, and virtually any type of cardiopulmonary abnormality, in addition to physiologic changes (e.g., with posture or after meals). Low QRS volt age is arbitrarily defined as peak-to-trough QRS amplitudes of ≤5 mm in the six limb leads and/or ≤10 mm in the chest leads. Multiple factors may be responsible. Among the most serious include pericardial (Fig. 247-17) or pleural effusions, chronic obstructive pulmonary disease, cardiac amyloid, and anasarca. ■ ■ELECTRICAL ALTERNANS SYNDROMES Electrical alternans—a beat-to-beat alternation in one or more com ponents of the ECG signal—is a common type of nonlinear cardio vascular response to a variety of hemodynamic and electrophysiologic perturbations. Total electrical alternans (P-QRS-T) with sinus tachy cardia is a relatively specific sign of pericardial effusion, usually with cardiac tamponade (Fig. 247-17). In contrast, pure repolarization Lead I Lead II 1mV 1s

Hypokalemia Hypothermia Amiodarone V3 II U Tricyclic overdose III V3 V2 I FIGURE 247-15 A variety of metabolic derangements, drug effects, and other factors may prolong ventricular repolarization with QT prolongation or prominent U waves. Prominent repolarization prolongation, particularly if due to hypokalemia, inherited “channelopathies,” or certain pharmacologic agents, indicates increased susceptibility to torsades des pointes ventricular tachycardia (Chap. 261). Marked systemic hypothermia is associated with a distinctive convex “hump” at the J point (Osborn wave, arrow) attributed to altered transmural ventricular action potential characteristics. Note QRS and QT prolongation along with sinus tachycardia in the case of tricyclic antidepressant overdose. Hypocalcemia Normal Hypercalcemia I I I II II II QT=480 ms QTc=500 ms QT=360 ms QTc=400 ms QT=260 ms QTc=350 ms FIGURE 247-16 Prolongation of the Q-T interval (ST-segment portion) is typical of hypocalcemia. Hypercalcemia may cause abbreviation of the ST segment with relative or absolute shortening of the QT interval. FIGURE 247-17 Classic triad of findings for pericardial effusion with cardiac tamponade: (1) sinus tachycardia; (2) low QRS voltages and (3) electrical alternans (best seen in leads V3 and V4 in this case; arrows). This triad is highly suggestive of pericardial effusion, usually with tamponade physiology, but is of limited sensitivity. (Adapted from LA Nathanson et al: ECG Wave-Maven. ecg.bidmc.harvard.edu.)

V5 V4 T CHAPTER 247 Electrocardiography Subarachnoid hemorrhage

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