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16.3.1 Electrocardiography 3294 Andrew R. Houghton and David Gray

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16.3.1 Electrocardiography Andrew R. Houghton and David Gray ESSENTIALS The resting 12-lead ECG The ECG has been recognized as a valuable diagnostic tool since the end of the 19th century. The normal ECG waveform consists of P, QRS, and T waves (and sometimes U waves)—P waves result from atrial depolarization, QRS complexes from ventricular depolariza- tion, and T waves from ventricular repolarization. The standard 12- lead ECG utilizes four limb electrodes and six precordial electrodes to generate 12 leads or ‘views’ of the heart’s electrical activity. There are six limb leads (termed I, II, III, aVR, aVL, and aVF) and six precor- dial leads (termed V1, V2, V3, V4, V5, and V6). Supplementary ‘views’ can be obtained by using additional leads, such as V7, V8, and V9 to assess the posterior aspect of the heart and right-sided chest leads to look for a right ventricular myocardial infarction. Assessment of the 12-lead ECG—this should be done in a method- ical manner, working through each aspect in turn. Conventionally, the heart rate, rhythm, and axis are assessed before inspection of each component of the waveform—the P wave, PR

interval, QRS complex, ST segment, T wave, QT interval, and U wave, with each component having its own range of normal attributes. Myocardial hypertrophy—the ECG can be a specific but generally insensitive tool for detecting myocardial hypertrophy: (1) left ven- tricular hypertrophy can be assessed using certain diagnostic cri- teria, including the Cornell criteria and the Romhilt–Estes scoring system; (2) right ventricular hypertrophy is indicated by a dominant R wave in lead V1 with right axis deviation; (3) left atrial hypertrophy is indicated by broad, bifid P waves; and (4) right atrial hypertrophy by tall P waves. Conduction blocks—(1) left anterior hemiblock results from a block of conduction in the anterosuperior fascicle and is a cause of left axis deviation; (2) left posterior hemiblock results from a block of con- duction in the posteroinferior fascicle and is a cause of right axis devi- ation; (3) left and right bundle branch blocks both cause broadening of the QRS complexes by prolonging ventricular depolarization, and both exhibit characteristic diagnostic features. Ventricular pre-excitation—causes shortening of the PR interval and can result from Wolff–Parkinson–White-type pre-excitation, short PR-type pre-excitation, or Mahaim-type pre-excitation. Acute coronary syndromes The ECG is the most useful bedside triage tool in acute coronary syn- dromes, with utility in diagnosis, in location of the site of ischaemia/ infarction, and as a prognostic indicator. ST elevation myocardial infarction—the first indication of infarc- tion on the ECG is usually ST segment elevation, which occurs within a few hours. The J point (the origin of the ST segment at its junction with the QRS complex) is elevated by 1 mm or more in two or more limb leads, or by 2 mm in two or more precordial leads. The ST seg- ment returns to the baseline over the next 48–72 h, during which Q waves and symmetrically inverted T waves appear. Some patients
develop left bundle branch block, either transiently or perman- ently. The ECG of a completed infarct shows new Q waves greater than 2 mm, R waves reduced in size or absent, and inverted T waves. Non-ST-elevation myocardial infarction—ECG changes are more variable than in ST-elevation myocardial infarction. The ECG may be normal on first presentation and remain unchanged throughout the acute admission; there may be transient ST segment depression in- dicative of myocardial ischaemia; in 20–30% the only change will be T-wave inversion. Difficulties in interpretation of the ECG in acute coronary syndromes— the ECG diagnosis of acute myocardial infarction can pose challenges in the setting of right ventricular infarction, atrial infarction, coronary

 artery spasm, reciprocal changes, ‘stuttering’ infarction, noninfarct
ST segment elevation, late presentation, left bundle branch block, prior infarction, pre-excitation, and T-wave inversion. Clinical decision-making—incorrect interpretation of an ECG can lead to inappropriate patient triage, either missing the opportunity to provide appropriate reperfusion therapy, or leading to inappro- priate treatment with attendant risk. Up to 12% of those with a high- risk ECG are missed on admission to the emergency department, yet pressure to provide treatment promptly to fulfil audit ‘targets’, for example, door-to-balloon time for primary percutaneous coronary intervention, should not replace accuracy in diagnosis. It is some- times better to repeat the ECG than to make an incorrect diagnosis. It is easy to place too much reliance on minor changes on the ECG; it is gross changes of ST elevation or depression within the parameters just outlined that should determine treatment. Exercise ECG testing Exercise ECG testing is better as an indicator of prognosis than as a diagnostic tool. The sensitivity of exercise ECG testing, the proportion with coronary disease correctly identified by the test, is 68% (range 23–100) and specificity, the proportion free of disease correctly iden- tified by the test, is 77%

(range 17–100). In multivessel disease, these figures are 81% (range 40–100) and 66% (range 17–100), respectively. This means that exercise testing frequently yields both false-positive results—incorrectly diagnosing disease when coronary arteries are normal or minimally diseased—and false-negative results—missing coronary disease when a flow-limiting, even critical left main stem, coronary stenosis is present. Appearance of symptoms or ECG changes early in an exercise test is generally associated with more severe and extensive coronary dis- ease and a poor prognosis. Changes within the first 3 min usually indicate severe coronary disease affecting the left main stem or the proximal segments of at least one major coronary artery. Multivessel coronary disease is more likely with ST segment down-sloping, de- layed ST normalization after exercise, increased number of leads af- fected, and lower workload at which ECG changes appear. The resting 12-lead ECG History The first electrocardiogram (ECG), of an exposed frog's heart, was performed by Marey in 1876 using the mercury capillary electrom- eter that had recently been invented by Gabriel Lippmann. Two years later the British physiologists John Burdon Sanderson and Fredrick Page demonstrated that recordings of the frog heart's elec- trical activity consisted of two phases (which were subsequently to become known as the QRS complex and T wave). The first human ECG was published in 1887 by Augustus D Waller, who had worked under Sanderson in the Department of Physiology at the University College of London. While working at St Mary's Hospital, London, Waller used a capillary electrometer to record the ECG of a labora- tory technician, Thomas Goswell. Electrocardiography was developed further by the Dutch physi- ologist Willem Einthoven, who witnessed a demonstration by Waller at the First International Congress of Physiology in Basle, Switzerland, in 1889. Although Einthoven made considerable im- provements to the technique of recording ECGs with the capillary electrometer, it was only with his invention of the string galvan- ometer at the turn of the century that high-quality ECG recording became possible. Within a decade of Einthoven's publication of the first string galvanometer ECG recordings in 1902, a commercial ECG machine became available. Manufactured by the Cambridge Scientific Instrument Company, the first machine was delivered to Sir Thomas Lewis, who would play a major role in developing the clinical application of electrocardiography. Einthoven's invention led to him being awarded the Nobel Prize in 1924. Einthoven was also the first to use the PQRST notation to de- scribe the ECG waveforms. In the early ECG recordings, the wave- forms were named ABCD (four deflections were recognized). Mathematical correction, using differential equations, was used to correct and improve ECG recordings, and it was traditional that mathematical notation used letters from the latter half of the al- phabet. The letters N and O were already used elsewhere, so it was decided to begin the notation at P. Over the following years further refinements were undertaken, most notably in the 1930s when the use of the chest leads was first described. At around the same time Frank Wilson invented the 'in- different electrode' (also known as the 'Wilson central terminal'). This led to the development of the 'unipolar' limb leads VR, VL, and VF ('V' stands for 'voltage'). In 1942 the American cardiologist Emanuel Goldberger increased the voltage of these leads by 50%, leading to the term 'augmented' leads (aVR, aVL, and aVF), and the 12-lead ECG which remains familiar today finally took shape. Although the format of the 12-lead ECG has remained essen- tially unchanged since that time, there have nevertheless been other significant developments in electrocardiography over more recent years. Ambulatory ECG recorders and implantable cardiac moni- tors have gained a central role in the investigation of patients with suspected arrhythmias, and the use of intracardiac ECG recording has enabled the rapid development and widespread use of electro- physiological studies. Normal ECG appearances The ECG waveform The three fundamental deflections on the normal ECG are termed the P wave, the QRS complex, and the T wave (Fig. 16.3.1.1). The origins of each deflection are as follows. P wave

The P wave results from depolarization of the atrial myocardium. Depolarization of the sinoatrial node itself, which triggers normal atrial depolarization, cannot be seen on the surface ECG (although it can be identified in intracardiac recordings). However, the presence of a P wave with normal morphology and orientation is generally taken to infer normal sinoatrial node depolarization. Repolarization of the atrial myocardium is represented on the ECG by the Ta wave (the atrial equivalent of the ventricular T wave). The Ta wave is seen as a small asymmetrical deflection after the P wave, with an opposite polarity to the preceding P wave. The Ta wave is often hidden within the QRS complex and is therefore not easily

section 16 Cardiovascular disorders 3296 seen—in fact, it is unusual to be able to appreciate the Ta wave at all. However, it can extend right through to the following ST segment, where it can be mistaken for the ST segment depression of myocar- dial ischaemia (particularly because the Ta wave is most likely to be seen extending into the ST segment during exercise-induced sinus tachycardia). There is one case report of a positive Ta wave (after an inverted P wave) giving the erroneous impression of an acute ST seg- ment elevation myocardial infarction. QRS complex The QRS complex represents depolarization of the ventricular myocardium. Of all the deflections, the QRS complex can exhibit the greatest variability in appearance. As a result, the individual compo- nents of the QRS complex can be labelled in upper case (Q, R, or S) or lower case (q, r, or s) to represent the relative size of the compo- nent. For example, QRS complexes with a small Q wave deflection can be termed qRS complexes, and those QRS complexes with no Q wave component and a small R wave component can be termed rS complexes. T wave The T wave (together with the preceding ST segment) represents repolarization of the ventricular myocardium. The 12 conventional ECG leads Lead nomenclature It is important to emphasize that the term ‘lead’ does not refer to the electrode connecting the ECG machine to the patient. For a standard 12-lead ECG recording, 10 electrodes are used to generate the 12 conventional ECG leads. The 12 leads can be categorized as limb (or frontal plane) leads (I, II, III, aVR, aVL, aVF) and chest (or precordial) leads (V1, V2, V3, V4, V5, V6). The 12 leads can also be categorized as bipolar (I, II, III) or unipolar (aVR, aVL, aVF, V1, V2, V3, V4, V5, V6). The leads aVR, aVL, and aVF can be further de- scribed as ‘augmented’ leads, as they are modified versions of the ori- ginal VR, VL, and VF leads, having a voltage amplification of 50%. The bipolar leads are generated by measuring the potential (voltage) between two electrodes. One electrode acts as a positive terminal and the other as a negative terminal. For instance, lead I measures the potential between the left arm electrode (positive) and right arm electrode (negative). Lead I is obtained by subtracting the right arm vector from the left arm vector. Similarly, lead II meas- ures the potential between the left leg electrode and the right arm electrode, and lead III measures the potential between the left leg electrode and the left arm electrode. The augmented unipolar leads measure the voltage between a single positive electrode and a ‘central’ point of reference generated from the other limb electrodes. Thus, aVR uses the right arm elec- trode as the positive terminal, aVL uses the left arm electrode, and aVF uses the left leg electrode. The three bipolar leads and the three augmented unipolar leads together comprise the six limb leads that view the heart in the frontal plane. The unipolar chest leads measure the voltage between six elec- trodes placed across the surface of the chest and a central point of reference, providing a view of the heart that is perpendicular to the frontal plane leads. For all 12 ECG leads, it is conventional that a wave of depolarization moving towards a lead generates a positive (upward) deflection on the ECG recording and vice versa. The six limb leads (frontal plane leads) Because the limbs act as linear conductors, it does not matter where- abouts the limb electrodes are attached on each limb. The six limb leads provide general spatial information (being

less localized than the six chest leads). Fig. 16.3.1.2 shows the orientation of the six Fig. 16.3.1.1 Basic ECG waveform. aVR aVL aVF Left Right I II III Fig. 16.3.1.2 The six limb leads and their ‘view’ of the heart. Note that leads II, III, and aVF are inferior to the heart, I and aVL are anterolateral to the heart, and aVR looks into the cavity of the heart.

16.3.1 Electrocardiography 3297 limb leads in relation to the heart. In simple terms, one can visualize lead aVR as ‘looking’ at the heart from the right shoulder, lead aVL from the left shoulder, and lead aVF from the feet. Lead I ‘looks’ at the heart from the left horizontal position. Similarly, the ‘views’ of leads II and III are shown in Fig. 16.3.1.2. The six chest leads (precordial leads) For the chest (precordial) leads, each of the six electrodes is at- tached to a particular site on the chest wall. The chest electrodes act as positive terminals, and the indifferent terminal is formed from a combination of leads R, L, and F. The location of each electrode is important, in contrast to the limb leads. The surface positions of the chest electrodes are shown in Fig. 16.3.1.3, and the relation be- tween the chest leads and the heart in Fig. 16.3.1.4. The electrodes are placed as follows: • The V1 electrode is placed at the right sternal edge in the fourth intercostal space • The V2 electrode is placed at the left sternal edge in the fourth intercostal space • The V3 electrode is placed midway between the V2 and V4 electrodes • The V4 electrode is placed at the left midclavicular line in the fifth intercostal space • The V5 electrode is placed at the left anterior axillary line in a hori- zontal line with V4 • The V6 electrode is placed at the left midaxillary line in a hori- zontal line with V4 and V5 Reading a normal 12-lead ECG Fig. 16.3.1.5 shows a normal 12-lead ECG. As is conventional, this shows the leads arranged in four columns, each column containing three leads. In addition, a rhythm strip runs along the bottom of the ECG across its whole width. This is conventionally lead II, but any one of the 12 leads can be used for the rhythm strip as required. The ECG is recorded at a paper speed of 25 mm/s, and at a sensitivity of 10 mm/mV. The speed and sensitivity settings can also be adjusted on most ECG machines, if required, and so it is important that the actual recording speed and sensitivity are always noted on the ECG for future reference. In the following paragraphs we will describe the appearances of the normal ECG, looking at each wave, interval, and segment in turn. We will assume that the patient is in normal sinus rhythm, and that a standard paper speed (25 mm/s) and calibration (10 mm/mV) have been used—this should always be checked be- fore reading any ECG. Identification details Before reading the ECG, check the patient’s details (the patient’s name and at least one other form of identification, such as date of birth or identification number, should be recorded on the ECG) and the date and time on which the ECG was recorded. It is good practice to note on the ECG any relevant clinical features. For instance, a note that the patient was experiencing chest pain or palpitations at the time the ECG was recorded can prove invaluable later on. Indeed, ECG interpretation should always take into account the appropriate clinical context. For instance, the ECG shown in Fig. 16.3.1.5 can be interpreted as showing normal sinus rhythm in a patient who is well. However, in a patient who is unconscious and pulseless, the same ECG would be interpreted as showing pulseless electrical activity, a cardiac arrest rhythm. Before interpreting any ECG, it is therefore appropriate (and important) to ask, ‘How is the patient?’ Rate A normal heart rate is between 60 and 100 beats/min. A rate below 60 beats/min is termed bradycardia; a rate greater than 100 beats/ min is termed tachycardia. Heart rate normally applies to the ven- tricular rate, as shown on the ECG by the rate of QRS complexes. However, the atria have their own rate, as shown by the P wave rate. The atrial and ventricular rates are usually the same, and there is a 1:1 ratio between P waves and QRS complexes. However, the rates can differ; for instance, in complete heart block (Fig. 16.3.1.6), the atrial rate is usually greater than the ventricular rate, and both rates should therefore be quoted.

Ventricular rate can be calculated in two different ways. One method necessitates counting the number of large (5 mm) squares between two adjacent QRS complexes. This figure is then divided into 300 to give the ventricular rate per minute. For instance, if there are five large squares between QRS complexes, the ventricular rate is 300/5 = 60 V1 V2 V3 V4 V5 V6 Fig. 16.3.1.3 Surface positions of the chest electrodes. V1 V2 V3 V4 V5 V6 Fig. 16.3.1.4 The chest leads and their anatomical relationship to the heart. Return to the top.

section 16 Cardiovascular disorders 3298 beats/min. The same method can be used to calculate atrial rate, counting the large squares between two consecutive P waves. If the heart rhythm is irregular, the square-counting method is not so useful. An alternative method is to count the number of QRS complexes in a certain time period, and then multiply the number up to obtain a rate per minute. Traditionally one counts the number of QRS complexes in a period of 30 large squares, which equates to 6 s of recording (a paper speed of 25 mm/s covers five large squares per second, or 300 large squares per minute). One then multiplies the result by 10 to obtain the rate per minute. Thus, if there are 8 QRS complexes within 30 large squares, then the ventricular rate is 8 × 10 = 80 beats/min. Once again, the same method can be used to calculate atrial rate. Rhythm A detailed description of arrhythmias can be found in Chapter 16.4. In general terms, the assessment of rhythm on the ECG requires careful attention to the following: • Whether there is ventricular activity (QRS complexes) and what is the ventricular rate; • Whether there is atrial activity (P waves) and what is the atrial rate; • Whether the heart rhythm is regular or irregular; • Whether the QRS complexes are normal or broad (broad com- plexes indicating either a ventricular origin to the rhythm or aber- rant conduction of a supraventricular rhythm); • Whether there is a relationship between P waves and QRS complexes. Assessing the ECG along these lines will provide a basis upon which to describe the rhythm and begin to identify the nature of the arrhythmia. Axis The concept of axis is often regarded as one of the hardest principles to grasp when learning ECG interpretation. The concept is, none- theless, straightforward: axis refers to the overall direction in which the wave of depolarization travels. There is a QRS (ventricular) axis, which is what most people refer to when discussing cardiac axis, but the P wave has its own axis too, representing the overall direction of depolarization in the atria. The T wave also has an axis, in this case referring to the overall direction of the wave of repolarization. In this section the discussion is confined to the QRS (ventricular) axis, but the same principles apply to P wave and T wave axes too. As the ventricles depolarize, the wave of depolarization travels through the atrioventricular node, into the bundle of His, and then to the ventricular myocardium via the Purkinje fibres. The overall direction of this depolarization wavefront is usually towards the apex of the heart. If, by convention, we regard the ‘view’ that lead I has of the heart (a horizontal line to the left of the heart) as 0°, and any angle clockwise from that line is positive (and any angle anti- clockwise from that line is negative), then the normal ventricular depolarization wavefront travels through the ventricles at an angle of approximately +60° (Fig. 16.3.1.7). As Fig. 16.3.1.7 illustrates, the six limb leads ‘view’ the heart from different angles. Lead I is taken as the horizontal reference point, 0°. Moving in a clockwise (positive) direction, lead II views the heart from an angle of +60°, lead aVF from an angle of +90°, and lead III from an angle of +120°. Moving anticlockwise from lead I, lead aVL views the heart from an angle of −30°, and lead aVR from an angle of −150°. This system of looking at axes, using the six limb leads, is known as the hexaxial reference system. The shaded area in Fig. 16.3.1.7 shows the normal range for the QRS axis, which lies between −30° and +90°. This does vary with Fig. 16.3.1.5 A normal 12-lead ECG. Fig. 16.3.1.6 Complete heart block: complete dissociation of atrial (P waves) and ventricular (QRS complexes) rate.

16.3.1 Electrocardiography 3299 body morphology—tall, slim individuals tend to have axes towards the rightward (+ 90°) end of the normal range; short, overweight individuals have axes towards the leftward (−30°) end of the normal range. An axis more negative (anticlockwise) than −30° is abnormal and termed left axis deviation. Similarly, an axis more positive (clockwise) than + 90° is abnormal and termed right axis deviation. Left axis deviation is seen in left anterior hemiblock (see next), in- ferior myocardial infarction, and also in ostium primum atrial septal defect. Right axis deviation is seen in left posterior hemiblock, right ventricular hypertrophy, lateral myocardial infarction, ostium secundum atrial septal defect, and Wolff–Parkinson–White (WPW) syndrome. There are several ways to calculate the QRS axis. One method is to look for which of the six limb leads has a QRS complex in which the R wave and S wave are closest to being equal (i.e. in which the positive and negative deflections cancel each other out). The QRS axis will be at right angles to this 'equipolar' lead, but could be pointing in either direction. For instance, if the equipolar lead is lead III (which looks at the heart from + 120°), then the QRS axis will be at right angles to this, namely either + 30° or −150° (refer back to Fig. 16.3.1.7). Next, find which lead is at right angles to the equipolar lead—in this example, the answer would be lead aVR. Now, if the QRS axis is −150°, then you would expect a positive QRS complex in lead aVR (because the wave of depolarization would be travelling directly towards it). If, how- ever, the QRS complex in lead aVR is negative, the depolarization must be moving away from it and the QRS axis must be therefore be + 30°. This method works whichever limb lead is equipolar, as every limb lead has another lead at right angles to it. An alternative and quick method of checking whether the QRS axis is within the normal range is simply to look at leads I and II. If the QRS complex in lead I is positive (or at least equipolar), then the QRS axis must lie somewhere in the range of −90° to + 90°. Similarly, if the QRS complex in lead II is positive, then the QRS axis must lie somewhere in the range −30° to + 150°. Therefore, we can say that if the QRS complexes in leads I and II are both positive then the QRS axis must lie somewhere in the range −30° to + 90°. Thus, a posi- tive QRS complex in leads I and II means the QRS axis is within the normal range; a positive QRS complex in lead I and a negative QRS complex in lead II indicate left axis deviation; a negative QRS com- plex in lead I and a positive QRS complex in lead II indicate right axis deviation. More precise calculations of the QRS axis can be made by measuring the individual R and S waves in each of the limb leads and using vector analysis to plot out the overall direction of depolar- ization, but this degree of precision is usually unnecessary. P wave The P wave represents atrial depolarization. P waves are usually up- right except in leads aVR and V1 (and sometimes V2), where they can be inverted (or biphasic). P waves are seen most clearly in lead II and this is usually the lead of choice for the rhythm strip so that atrial activity can be assessed clearly. P waves can be inverted in other leads, indicating that atrial de- polarization has been initiated somewhere other than the sinoatrial node. For instance, an ectopic focus of depolarization near the atrioventricular node will give rise to inverted P waves in the in- ferior leads (II, III, and aVF) as the wave of atrial depolarization will spread upwards rather than downwards. P waves are normally no broader than three small squares (0.12 s) and no taller than 2.5 mm. The features of atrial hypertrophy are discussed later. PR interval The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. A normal PR interval is between 0.12 s and 0.20 s in adults. A long, fixed PR interval is termed first-degree atrioven- tricular block and results from a delay in conduction between the atria and ventricles (Fig. 16.3.1.8). In second-degree atrio- ventricular block the PR interval may gradually increase with each beat before a P wave is not conducted (Mobitz type I or Wenckebach phenomenon) or may be fixed and long (or normal) with intermittent nonconduction of P waves (Mobitz type II). In third-degree atrioventricular block and also in atrioventricular dissociation, the PR interval will vary

because of the absence of any association between atrial and ventricular activity. See Chapter 16.4 for further discussion. 150 III II I aVF aVL 120 90 60 30 0 −30 −60 −90 −120 −150 180 aVR Fig. 16.3.1.7 The standard convention for describing the orientation of cardiac axis, and the corresponding ‘views’ of each of the six limb leads. The shaded area represents the normal range for the QRS axis. Fig. 16.3.1.8 First-degree atrioventricular block (long PR interval).

section 16 Cardiovascular disorders 3300 A short PR interval is seen in ventricular pre-excitation (see next) or when the focus of atrial depolarization arises not from the sino- atrial node but from the vicinity of the atrioventricular node. QRS complex The QRS complex represents ventricular depolarization. The first nega- tive deflection of the complex is termed the Q wave and the first positive deflection the R wave (whether or not it follows a Q wave). A negative deflection after an R wave is termed an S wave. If the deflections are small, lower-case letters (q, r, and s) are used. Thus, it is possible to have QRS complexes, qRS complexes, rS complexes, and so on. Normal ‘physiological’ q waves are usually narrow (no more than 0.04 s in duration) and small (less than 25% the amplitude of the following R wave) and result from the left to right depolarization of the interventricular septum (‘septal q waves’). Larger Q waves may be pathological, although can be normal in leads III and aVR, and may also been seen in lead aVL if the QRS axis is greater than + 60°. The normal QRS complex duration is less than 0.12 s. The amp- litude of the QRS complex varies normally from lead to lead and, in the precordial leads, normally increases progressively from lead V1 to V6. At least one R wave in the precordial leads must be at least 8 mm in height, and the tallest R wave should be no more than 27 mm (and the deepest S wave no more than 30 mm), and the sum of the tallest R wave and the deepest S wave should be no more than 40 mm. In the limb leads, the R wave height should be no more than 13 mm in lead aVL and 20 mm in lead aVF. ST segment The ST segment should be horizontal and should not normally de- viate by more than 1 mm above or next the isoelectric line (which is the line between end of the T wave and the start of the subsequent P wave). T wave T waves in the limb leads are normally concordant—if the QRS com- plex is positive, the subsequent T wave is upright, and vice versa. The T wave is normally inverted in lead aVR and upright in leads I and II. With regard to the precordial leads, normal T waves are always upright in leads V4 to V6. A flat or inverted T wave is found in lead V1 in 20% of adults, and in lead V2 in 5% of adults (in which case, the T wave should be inverted in lead V1 as well). An inverted T wave in lead V3 can, rarely, be found in normal young adults. T waves should not change their orientation—an inverted T wave is not normal if previous ECGs show that it was previously upright. There are no strict criteria for normal T wave size, so ‘tall’ and ‘small’ T waves are not well defined and deciding on their presence tends to be a subjective judgement. ‘Tall’ T waves can occur in early acute myocardial infarction (‘hyperacute’ T waves) and in hyperkalaemia (‘tented’ T waves). Small T waves can be seen in hypokalaemia. QT interval The QT interval is measured between the start of the QRS complex and the end of the T wave. The normal range for the QT interval varies according to heart rate. It is therefore convenient to correct the measured QT interval to what it would be if the heart rate were 60 beats/min. This is done most commonly using Bazett’s formula, in which the measured QT interval (in seconds) is divided by the square root of the RR interval (in seconds), to give the corrected QT interval (QTc). The normal range for the QT interval at a heart rate of 60 beats/min, and thus for the QTc, is between 0.35 s and 0.45 s (men) or 0.46 s (women) (Fig. 16.3.1.9). U wave The T wave is occasionally followed by a U wave, most clearly seen in the right precordial leads, which has the same orientation as the T wave and is usually no more than one-third of its size. The physio- logical origin of the U wave is still debated but is often said to relate to after depolarizations in the

ventricles. Myocardial hypertrophy Left ventricular hypertrophy Evidence of left ventricular hypertrophy on the ECG is a significant risk factor for cardiovascular morbidity and mortality. Several diag- nostic ECG criteria for left ventricular hypertrophy have been de- veloped which, in general, are relatively specific (>90%) but not very sensitive (20–60%). The diagnostic criteria shown in Box 16.3.1.1 are commonly used. The Cornell criteria involve measuring the S wave in lead V3 and the R wave in lead aVL. Left ventricular hypertrophy is indicated by a sum of more than 28 mm in men and more than 20 mm in women. The Romhilt–Estes scoring system allocates points for the pres- ence of certain criteria, with a score of five indicating left ventricular hypertrophy and a score of 4 indicating probable left ventricular hypertrophy. Points are allocated as follows: • 3 points for (a) R or S wave in limb leads of 20 mm or more; (b) S wave in right precordial leads of 25 mm or more; or (c) R wave in left precordial leads of 25 mm or more; • 3 points for ST segment and T wave changes (‘typical strain’) in a patient not taking digitalis (1 point with digitalis); • 3 points for P-terminal force in V1 greater than 1 mm deep with a duration greater than 0.04 s; • 2 points for left axis deviation (beyond −15°); • 1 point for QRS complex duration greater than 0.09 s; • 1 point for intrinsicoid deflection (the interval from the start of the QRS complex to the peak of the R wave) in V5 or V6 greater than 0.05 s. Fig. 16.3.1.9 A prolonged QT interval. Measurement can be difficult since the precise beginning and end of the interval may not be easy to determine, particularly if the end of the T wave is obscured by a superimposed U wave or the following P wave.

16.3.1 Electrocardiography 3301 A left ventricular ‘strain’ pattern (ST-T wave abnormalities) is as- sociated with around double the risk of myocardial infarction and stroke as left ventricular hypertrophy in the absence of strain. Left ventricular hypertrophy cannot be assessed reliably using the ECG in patients with bundle branch block, previous myocardial in- farction, or WPW syndrome; visualization via echocardiography or cardiac MRI is required. An example of left ventricular hypertrophy is shown in Fig. 16.3.1.10. Right ventricular hypertrophy As with left ventricular hypertrophy, the ECG criteria for right ven- tricular hypertrophy tend to be relatively specific but not very sen- sitive. Right ventricular hypertrophy shifts the QRS complex axis rightwards as well as producing higher-voltage QRS complexes in the right precordial leads. ECG criteria include: • a dominant R wave (R wave ≥ S wave) in lead V1, in the presence of a normal QRS duration; • a QRS complex axis of greater than + 90°. These criteria are supported by: • ST segment depression and T-wave inversion in the right pre- cordial leads; • deep S waves in the lateral precordial and limb leads. It is not essential for all these criteria to be present, but the greater the number of features present, the greater the likelihood of right ventricular hypertrophy. It is prudent to remember that a dominant R wave in lead V1 can also be seen in right bundle branch block, WPW syndrome, and a posterior wall myocardial infarction. Atrial hypertrophy Left atrial hypertrophy Left atrial depolarization is responsible for the terminal portion of the normal P wave. Left atrial hypertrophy increases the voltage and duration of this depolarization, and thus usually evidences itself by abnormalities of the terminal portion of the P wave. The P wave dur- ation is prolonged, and it becomes bifid in lead II and biphasic, with a predominant negative component, in lead V1. So-called ‘P mitrale’ can be seen in the left atrial enlargement that results from mitral valve stenosis (hence the term) and also in association with condi- tions that cause left ventricular hypertrophy, such as hypertension (most commonly) and aortic stenosis (Fig. 16.3.1.11). Right atrial hypertrophy Right atrial hypertrophy increases the voltage, but not the duration, of the P wave, and this is usually best seen in the inferior and right precordial leads. A P wave height greater than 2.5 mm is regarded as abnormal. So-called ‘P pulmonale’ can result from

right ventricular hypertrophy or from tricuspid valve stenosis (Fig. 16.3.1.12). The hemiblocks The left bundle branch divides into anterosuperior and postero inferior fascicles. A block of either fascicle (hemiblock) causes a deviation of the QRS axis. Left anterior hemiblock A block of the anterosuperior fascicle leads to a left anterior hemiblock. This causes a leftward shift in the QRS axis, as the Box 16.3.1.1 Diagnostic criteria for left ventricular hypertrophy Limb leads • R wave >11 mm in lead aVL • R wave >20 mm in lead aVF • S wave >14 mm in lead aVR • Sum of R wave in lead I and S wave in lead III >25 mm Precordial leads • R wave of ≥25 mm in the left precordial leads • S wave of ≥25 mm in the right precordial leads • Sum of S wave in lead V1 and R wave in lead V5 or V6 >35 mm (Sokolow–Lyon criteria) • Sum of tallest R wave and deepest S wave in the precordial leads

> “ 45 mm Fig. 16.3.1.10 Left ventricular hypertrophy.

section 16 Cardiovascular disorders 3302 right/inferior region of the left ventricle depolarizes first (via the posteroinferior fascicle) and then the wave of depolarization spreads to the left/superior region. Although this hemiblock introduces a minor delay in ventricular depolarization, the QRS duration re- mains within the normal range (up to 120 ms). The QRS axis shifts to the left (beyond −30°). As a similar axis shift can result from an inferior myocardial infarction, the diagnosis of an left anterior hemiblock requires the presence of left axis deviation in the absence of an abnormal q wave in lead aVF. Left posterior hemiblock Block of the posteroinferior fascicle leads to left posterior hemiblock. This causes a rightward shift in the QRS axis, as the left/superior region of the left ventricle depolarizes first (via the anterosuperior fascicle) and then the wave of depolarization spreads to the right/in- ferior region. As with a left anterior hemiblock, the QRS duration re- mains within the normal range (up to 120 ms). The QRS axis shifts to the right (beyond + 90°). However, right axis deviation can occur in several conditions (most commonly right ventricular hypertrophy, but also in lateral myocardial infarction and WPW syndrome). It is therefore not possible to diagnose left posterior hemiblock with cer- tainty from the 12-lead ECG alone. Bundle branch block Left bundle branch block A left bundle branch block (LBBB) leads to a delay in left ventricular depolarization, as the left ventricle is depolarized via the right-sided Purkinje system. In addition, the interventricular septum depolar- izes from right to left instead of the usual left to right. Thus, in LBBB: • The QRS duration is prolonged (≥120 ms); • The normal ‘septal’ q waves usually seen in the lateral leads are absent; • A secondary r wave is not seen in lead V1 (this distinguishes LBBB from right bundle branch block (RBBB) with clockwise cardiac rotation). These findings may be accompanied by ST segment depression and T-wave inversion in the lateral precordial and limb leads, broad QS waves in the right precordial leads and broad R waves in the lat- eral leads, and R wave notching (‘M-shaped’ QRS complexes). An example of LBBB is shown in Fig. 16.3.1.13. The extensive na- ture of the ECG changes means that further interpretation of the QRS complexes, ST segments, or T waves cannot be made. The dif- ficulties of diagnosing myocardial infarction in the setting of LBBB are discussed later. Right bundle branch block RBBB leads to a delay in right ventricular depolarization, as the right ventricle is depolarized via the left-sided Purkinje system. However, the normal left to right activation of the interventricular septum is preserved. The ECG changes seen in RBBB are therefore not as ex- tensive as in LBBB. The QRS duration is prolonged (≥120 ms) and the right ventricular leads contain a second positive wave (and, con- versely, the left ventricular leads contain a second negative wave). Thus, in RBBB: • the QRS duration is

prolonged (≥120 ms); • lead V1 contains a second positive wave (rsR); • lead V6 contains a second negative wave (qRs). These findings may be accompanied by deep slurred S waves in the lateral precordial and limb leads, and abnormal ST-T wave changes in the right precordial leads. An example of RBBB is shown in Fig. 16.3.1.14. Ventricular pre-excitation The normal progression of a wave of depolarization is from the sino- atrial node through the atria to the atrioventricular node, and then through the bundle of His and the Purkinje fibres to the ventricular myocardium. However, approximately 1 in 1000 of the population has an accessory pathway—an alternative pathway from atria to ventricles that bypasses part of this normal route. Such a pathway initiates depolarization of the ventricles at a slightly earlier stage in the cardiac cycle than would otherwise be the case, hence the term 'ventricular pre-excitation'. This is because the accessory pathway lacks the inherent delay to conduction that is normally found in the atrioventricular node, thus allowing faster conduction of the wave of depolarization from atria to ventricles. There are several types of pathway that can give rise to ventricular pre-excitation. WPW-type pre-excitation WPW-type pre-excitation is exemplified by WPW syndrome. In WPW syndrome an accessory pathway, the bundle of Kent, connects Fig. 16.3.1.11 Left atrial hypertrophy ('P mitrale'). Fig. 16.3.1.12 Right atrial hypertrophy ('P pulmonale').

16.3.1 Electrocardiography 3303 the atria to the ventricles and bypasses the atrioventricular node al- together. This shortens the time between the onset of atrial depolar- ization and the onset of ventricular depolarization, and hence one of the ECG features of WPW syndrome is a short PR interval (<0.12 s). Because the accessory pathway leads directly to the ventricular myocardium, and not into the His–Purkinje system, the subsequent initial ventricular depolarization progresses slowly, as conduction of the wave of depolarization cannot take advantage of the rapidly-conducting Purkinje fibres. This gives rise to a δ-wave—a slurred initial upstroke of the QRS complex. These features can be seen in the ECG from a patient with WPW syndrome in Fig. 16.3.1.15. Conduction between atria and ventricles can be via the accessory pathway, or via the atrioventricular node, or via both routes, or can vary from one to another. If conduction does not occur via the acces- sory pathway, the ECG will appear normal and the pathway is said to be 'concealed'. The appearances of the δ-wave can vary from patient to patient. Indeed, the ECG appearances of the QRS complex can be used, to a limited extent, to predict the likely location of the accessory pathway. However, the exact location can only be found with elec- trophysiological studies. The QRS complex morphology in WPW syndrome can mimic LBBB, RBBB, or acute myocardial infarction. WPW syndrome can also lead to repolarization (and therefore T wave) abnormalities. Great care must be thus taken in diagnosing these conditions in the presence of WPW syndrome. The existence of two atrioventricular pathways (the normal atrioventricular node and the abnormal bundle of Kent) provides a substrate for atrioventricular re-entry tachycardia, which is dis- cussed in more detail in Chapter 16.4. Short PR-type pre-excitation A short PR interval in the absence of a δ-wave/abnormal QRS complex is often referred to as Lown–Ganong–Levine syndrome, in which an atrio-His accessory pathway bypasses the slow- conducting part of the atrioventricular node. Identical appearances can also be seen with a fast-conducting atrioventricular node. The Fig. 16.3.1.13 Left bundle branch block. Fig. 16.3.1.14 Right bundle branch block.

section 16 Cardiovascular disorders 3304 presence of an atrio-His accessory pathway is a substrate for atrio- ventricular nodal re-entry tachycardia. For further discussion, see Chapter 16.4. Mahaim-type pre-excitation Mahaim fibres, first described in the 1930s, are atriofascicular or

atrioventricular accessory pathways that connect the atrioven- tricular node to the right bundle or the right ventricle. Patients with Mahaim-type pre-excitation have a normal PR interval with a LBBB QRS complex morphology and are prone to re-entry tachycardia. For further discussion, see Chapter 16.4. Acute coronary syndromes Sudden disruption of existing coronary plaque may partially or to- tally occlude a coronary artery, causing myocyte necrosis. Symptoms of severe, centrally located chest pain develop suddenly, usually ac- companied by breathlessness due to left ventricular dysfunction and tachycardia, pallor, sweating, nausea, and extreme anxiety due to sym- pathetic drive. The American College of Cardiology and the European Society of Cardiology classification of myocardial ischaemia and in- farction recognizes that acute changes in coronary atheroma produce a spectrum of disease, the acute coronary syndromes (ACS): • Unstable angina, where coronary plaque has ruptured but stabilizes without major change in the lumen of the coronary ar- tery: the ECG may be normal, or indicate a previous myocardial infarction, or dynamic ST depression and/or T-wave inversion may appear. Serum troponin level, a marker of myocyte necrosis, is within normal limits. • Non-ST elevation myocardial infarction (NSTEMI), where plaque is ruptured with partial occlusion of a major coronary artery: ECG signs are variable—the ECG may be normal, or indicate a previous myocardial infarction, or ST depression may appear transiently, or symmetrically inverted T waves may appear. Troponin levels are elevated. • ST elevation myocardial infarction (STEMI) where thrombosis from a ruptured plaque completely occludes a coronary artery: the ECG shows ST segment elevation initially, then resolves within a day or two, with new Q waves and inverted T waves appearing in the leads subtending the infarcted area. Troponin levels are elevated. For discussion of the clinical features and management of ACS, see Chapters 16.2.1, 16.13.4, and 16.13.5. Role of the ECG in acute coronary syndromes The ECG remains the most useful bedside triage tool in the emergency setting, whether in the community, en route to hos- pital, or in the emergency department. Accurate interpretation is essential—misinterpretation of the ECG can be as high as 12% and lead to inappropriate management. The ECG is used to diagnose ACS, to locate the site of ischaemia and infarction (see Table 16.3.1.1) and to identify areas of impaired perfusion (see ‘Reciprocal changes’). Of those who suffer a STEMI, the initial ECG is diagnostic in 50%, abnormal but not diagnostic in 40%, and normal in the re- mainder. Repeat ECGs may be necessary to confidently diagnose or exclude an acute coronary syndrome, as diagnostic changes may not appear for several hours. Serial recordings increase the sensi- tivity to 95%. The presenting ECG and prognosis in acute coronary syndrome About 22% of all patients with acute chest pain will present with T-wave inversion, 28% with ST segment elevation, 35% with ST segment depression, and 15% with a combination of ST segment elevation and depression. T-wave inversion is most likely to be as- sociated with angiographically normal coronary arteries. Those with ST segment depression are more likely to have three-vessel disease. Mortality at 1 month is 1.7% in those with T wave changes, 5.1% with ST segment elevation or depression, and 6.6% with both depression and elevation. Severe ST segment depression (>2 mm Fig. 16.3.1.15 WPW syndrome, showing the short PR interval and δ-wave.

 in two contiguous leads) is associated with an increased risk of death at 1 year. The presenting ECG and probability
of acute coronary syndrome New, or presumed new, ST segment deviation greater than 0.1 mV, however transiently, or T-wave inversion in multiple precordial leads, is highly indicative of ACS. Q waves, ST segment depression of 0.05– 0.1 mV, or T-wave inversion greater than 0.1 mV have an intermediate probability of ACS. T-wave flattening or inversion less than 0.1 mV (in leads with

dominant R waves) or a normal ECG has a low probability of ACS. The likelihood of NSTEMI is increased threefold in chest pain with ST segment depression in three leads or more than 0.2 mV.
The presenting ECG and triage The presenting ECG can be used to triage patients with acute cardiac-sounding chest pain: • ST elevation present—immediate reperfusion should be considered, by primary percutaneous coronary intervention (PCI) (or by intravenous thrombolysis if primary PCI unavailable). • ST elevation not evident—immediate treatment with antiplatelet drugs and anti-ischaemic drugs, with consideration of cor- onary angiography where appropriate. Risk stratification using tools such as GRACE or TIMI scoring, can help identify those most likely to benefit from early coronary angiography and revascularization. Table 16.3.1.1 Location of infarction and affected coronary artery ECG leads affected Site of infarction Most likely artery occluded (positive predictive value) V3 and V4 I, aVL and V1 to V6 (in extensive infarction) Anterior Left anterior descending (96%) V1 and V2 Septal V1 to V4 Anteroseptal I, aVL, and V3 to V6 Anterolateral II, III, and aVF Inferior Right coronary (80%) Right or circumflex (94%) I, aVL and V6 I and aVL (high lateral) Lateral Circumflex (75%) ST depression in V1 and V2 followed by development of prominent R waves in lead V1 or V2 Posterior Circumflex (75%) Lateral or posterior Right or circumflex (94%) II, III, and aVF with aVL, V5, and V6 Inferolateral Right coronary (93%)
Fig. 16.3.1.16 Evolution of STEMI over several days.

section 16 Cardiovascular disorders 3306 ST segment elevation myocardial infarction The ECG changes of myocardial infarction, first described in 1920, reflect myocardial ischaemia, injury, and myocyte necrosis. Within an hour or so of occlusion of a coronary artery, the T wave be- comes more prominent, exceeding one-half the height of the pre- ceding R wave in the ECG leads subtending the infarcted area (see Fig. 16.3.1.16). Many patients present later than this, so these changes may pass unnoticed. In up to 50%, the presenting ECG is normal. The first documented indication of infarction is usually ST segment elevation which occurs within a few hours. The J point (the origin of the ST segment at its junction with the QRS complex) is elevated by 1 mm or more in two or more limb leads, or by 2 mm in two or more precordial leads. The ST segment returns to the baseline over the next 48–72 h, during which Q waves and symmetrically inverted T waves ap- pear. Some patients develop LBBB, either transiently or perman- ently. The ECG of a completed infarct shows new Q waves greater than 2 mm, R waves reduced in size or absent, and inverted T waves. This classical evolution of STEMI is seen in about 50–66% of patients. Reperfusion therapy, by primary PCI (or thrombolysis where primary PCI is unavailable) may alter this natural sequence of changes in the ECG. If treatment is given with thrombolysis, then an ECG performed 90 min after initiation should show that ST ele- vation has been reduced by at least 50% from pretreatment levels (Fig. 16.3.1.17). If chest pain persists and the ST segments remain elevated, coronary angiography and rescue PCI should be con- sidered. Where available, primary PCI should be offered in pref- erence to thrombolysis. Resolution of ST segment elevation predicts 30-day mortality. With greater than 70% ST segment resolution, mortality is 2.1%; with 30–70% ST segment resolution 5.2%; with no ST segment reso- lution 5.5%; and with worsening ST segment elevation 8.1%. Non-ST elevation myocardial infarction ECG changes in NSTEMI are more variable than in STEMI. The ECG may be normal on first presentation and remain unchanged throughout the acute admission. There may be transient ST seg- ment depression indicative of myocardial ischaemia. In 20–30%, the only change will be T-wave inversion. Risk-scoring systems have (b) (a) Fig. 16.3.1.17 (a) Acute inferolateral ST segment elevation myocardial infarction. (b) Substantial (but not complete) resolution of ST segment elevation 90 min after the initiation of thrombolysis.

16.3.1 Electrocardiography 3307 been developed, for example, by the Trials In Myocardial Infarction group (http://www.timi.org), for use in patients with ACS. These are described in Chapter 16.13.4: with regard to NSTEMI, ST segment deviation greater than 0.5 mm is one of the recorded parameters. The extent of ST depression identifies those who are most likely to benefit from early revascularization (FRISC II trial). Mortality with early invasive therapy is 4% with ST segment depression, 2% with no ECG changes, and 0.2% with T-wave inversion. Difficult diagnoses in acute myocardial infarction Right ventricular infarction The ECG provides prognostic as well as diagnostic information. An inferior infarction generally carries a good prognosis unless it is associated with a right ventricular infarction, when there is a six- fold increased risk of a major in- hospital complication, including ventricular fibrillation, reinfarction, and death. The right ventricle is involved in about 50% of those with an inferior infarction, occurring with occlusion of the right coronary artery, causing a transmural in- farct of the inferoposterior wall and the posterior septum. To determine whether the right ventricle is involved in an in- ferior infarction, an ECG should be recorded with the anterior chest leads placed on the right side of the chest, in equivalent (but mirrored) positions to a standard 12-lead ECG. The right ven- tricle is involved if there is greater than 1 mm ST segment eleva- tion in chest lead ‘right V4’ (RV4); this has a sensitivity of 100%, specificity of 87%, and positive predictive value of 92% for occlu- sion of the right coronary artery proximal to the right ventricular branch. If these changes are absent, the right ventricle has been spared (Fig. 16.3.1.18). Atrial infarction This occurs in up to 10% of myocardial infarcts in conjunction with ventricular infarction. A clue to its presence is PR segment (b) (a) Fig. 16.3.1.18 (a) Inferior ST segment elevation myocardial infarction. (b) Inferior ST segment elevation myocardial infarction with right ventricular involvement (note the right ventricular chest leads, with ST segment elevation in lead RV4).

section 16 Cardiovascular disorders 3308 displacement but there may also be an abnormal P wave. It can cause rupture of the atrial wall and is frequently associated with atrial arrhythmias including atrial fibrillation, atrial flutter, and atrioventricular nodal rhythm. Coronary artery spasm The pain of Prinzmetal’s or variant angina is not usually triggered by exercise, emotion, cold, or a meal but tends to occur at rest, ac- companied by transient, marked ST segment elevation. This rapidly reverts to normal when the pain resolves spontaneously or with gly- ceryl trinitrate. Atrioventricular block or ventricular arrhythmia may accompany spasm-induced myocardial ischaemia. Spasm suf- ficient to cause myocardial ischaemia, myocardial infarction, and sudden death can follow cocaine use. Reciprocal changes—septal ischaemia
or posterior infarction? ST or ‘reciprocal’ depression may be seen in leads remote from the site of a STEMI. For example, ST depression may be seen in leads V1 to V4 in an inferior STEMI. There are two explanations. First, in a right-dominant system (70% of the population), the right cor- onary artery supplies the posterior interventricular septum, which becomes ischaemic with an inferior STEMI; the ischaemia resolves within a few days as septal perforating arteries from the left anterior descending artery dilate in response to ischaemic stress. Second, in a left-dominant system, the circumflex supplies the posterior interventricular septum; if this occludes, a ‘true posterior infarc- tion’ follows. Difficulties in diagnosing STEMI ‘Stuttering’ infarction Symptoms of myocardial infarction are usually severe and of sudden onset. Occasionally, the onset of symptoms is not so clear cut and chest pain may resolve but recur at intervals over several hours. The time of arterial occlusion is at best a guess, but for prac- tical purposes is taken as the time that symptoms increase or are at their worst. Noninfarct causes of ST segment elevation Pericarditis may mimic the pain of myocardial infarction but is usu- ally relieved by sitting forward and is accompanied by

a pericardial rub. The ST segments are elevated diffusely, do not fit the usual lead pattern for an inferior or anterior infarction, and, unlike the con- vexity of STEMI, are concave upwards (Fig. 16.3.1.19). Prinzmetal's angina, caused by coronary artery spasm, can also mimic myocar- dial infarction. This usually occurs at rest, with marked ST eleva- tion during pain and a brisk response to glyceryl trinitrate. The ST segment can be elevated chronically in left ventricular aneurysm, left ventricular hypertrophy, LBBB, hypertrophic cardiomyopathy, acute cor pulmonale, hypothermia, and cocaine abuse. A normal variant is so-called 'high take-off' where serial ECGs show con- sistent ST elevation across most ECG leads; patients should be given a copy of the ECG to show to medical personnel to avoid unneces- sary investigations and treatment. Late presentation Patients who present to hospital outside the 12-h time limit for reperfusion are sometimes diagnosed as 'missed infarction'. The ECG may show signs characteristically seen later in the infarction process, with ST segments only slightly elevated, with established Q waves and inverted T waves. Over the next few days, the ST seg- ment fully returns to baseline and Q waves and T waves deepen. LBBB Recognition of acute STEMI in pre-existing LBBB is challenging, but the Sgarbossa criteria help. Five points are scored for ST eleva- tion 1 mm or greater in at least one lead with a positive QRS com- plex, 3 points for ST depression 1 mm or greater in leads V1–V3, and 2 points for 5 mm or greater ST elevation in leads with a negative QRS complex. A score of 3 points or greater has a 90% specificity (but a poor sensitivity) for acute myocardial infarction. ECG changes of 'old' infarction Q waves, once formed, usually persist indefinitely and so are a reli- able indicator of a previous myocardial infarction (Fig. 16.3.1.20). Fig. 16.3.1.19 Widespread elevation of the ST segments (concave upwards) in a case of pericarditis.

16.3.1 Electrocardiography 3309 However, there are several other causes of a Q wave that may cause confusion, the most common being hypertrophic cardiomyopathy and idiopathic cardiomyopathy. Rarer causes include myocarditis, cardiac amyloid, neuromuscular disorders (e.g. muscular dystrophy, myotonic dystrophy, Friedreich's ataxia), scleroderma, sarcoidosis, and an anomalous coronary artery. Pre-excitation WPW syndrome makes interpretation of the ECG more compli- cated. It may mask a myocardial infarction if conduction via the bypass tract is towards the left ventricle, as a Q wave will not be apparent. WPW may also simulate an infarction due to a negative δ-wave in the inferior leads producing Q waves. Serial or previous ECGs will reveal the true diagnosis. Patients with WPW syndrome should be given a copy of their ECG to avoid confusion and un- necessary future investigations. T-wave inversion Atypical ECG features are seen in up to half of all infarctions in the early stages. Alone, these changes are not diagnostic. They can occur in ventricular aneurysm, electrolyte abnormalities, myocarditis, and subarachnoid haemorrhage, and with some drugs. Serial ECGs are necessary to establish a firm diagnosis. Deep, symmetrical 'arrowhead' T waves developing during an infarction are most often due to proximal occlusion of the left an- terior descending coronary artery (Fig. 16.3.1.21). Where errors occur Incorrect interpretation of an ECG leads to inappropriate patient triage and misses the opportunity to provide reperfusion therapy, whether by angioplasty or thrombolysis. In the worst-case scenario, inappropriate thrombolysis might lead to a haemorrhagic stroke or ruptured aneurysm. Up to 12% of those with a high-risk ECG (i.e. ST segment elevation of at least 0.1 mV, ST segment depression of at least 0.05 mV, or T-wave inversion of at least 0.2 mV in two or more contiguous leads) are missed on admission to the emergency department. The ECG provides a 'snapshot' of electrical events within the heart, when the clinician really needs a 'movie' to monitor the dynamic changes of an acute coronary syndrome. If a diagnosis cannot be made on the presenting ECG but the history suggests an acute coronary syndrome, the patient should be admitted to a monitored area,

a review by a specialist should be arranged, and the ECG should be repeated if symptoms get worse or if ST segment changes are seen on the monitor. This will ensure prompt and ap- propriate treatment. While it may be important to provide treatment promptly to fulfil audit targets (e.g. door-to-balloon time for primary PCI), speed should not replace accuracy in diagnosis. It is sometimes better to repeat the ECG than to make an incorrect diagnosis. It is easy to place too much reliance on minor changes on the ECG; it is clear changes of ST elevation or depression within the aforementioned parameters, that should determine treatment. Fig. 16.3.1.20 ‘Old’ inferior myocardial infarction: pathological Q waves in leads II, III, and aVF. Fig. 16.3.1.21 Recent anterior ST segment elevation myocardial infarction with ‘arrowhead’
T-wave inversion.

section 16 Cardiovascular disorders 3310 Exercise ECG testing ECG changes on exercise were first reported in patients with chronic stable angina in the early 1900s. Exercise testing was adopted into routine clinical practice soon after a standardized exercise protocol was developed. Cardiovascular responses to exercise in normal subjects and in coronary disease Normally on treadmill exercise, heart rate increases as a result of diminished vagal and increased sympathetic outflow. Heart rate in- creases on commencing exercise and reaches a plateau during each stage of the exercise test. A rapid increase may be due to lack of fit- ness, prolonged bed rest, anaemia, or dehydration. Systolic blood pressure increases in line with increased cardiac output, while dia- stolic pressure is near constant or falls slightly due to vasodilatation. On stopping the test, heart rate slows within a few minutes to pretest levels and both systolic and diastolic blood pressure falls, often to below pretest levels, as a result of vasodilatation. With cardiac disease, the maximum cardiac rate may be attenu- ated (even in the absence of a β-blocker) due to sinus node dis- ease, coronary heart disease, or postinfarction (with or without β-blockade). Failure to achieve the maximum predicted heart rate, calculated as 220 minus age, is suggestive of cardiac disease. Brady-and tachyarrhythmias including atrial fibrillation may occur. Exercise-induced hypotension, even a transient fall in blood pres- sure at (near-)maximum heart rate, is indicative of severe heart dis- ease and increases the risk of ventricular fibrillation. On stopping exercise, systolic pressure falls to resting levels (or lower) within minutes, where it may remain for several hours. In some, venous pooling may cause a precipitous drop in systolic pressure. ECG changes with exercise
in normal subjects
and in coronary disease In normal subjects, exercise-induced tachycardia causes short- ened PR, QRS, and QT intervals, increased P wave amplitude, and down-sloping of the PR segment. R waves and T waves may di- minish, and S waves increase at maximum exercise. The J point (the isoelectric point where the S wave reaches the baseline) may become depressed in all leads and the ST segment may become up-sloping. The most helpful ECG marker of exercise-induced myocardial is- chaemia is the ST segment which becomes depressed with increasing heart rate. This is due to shortening of the action potential due to is- chaemia, setting up electrical gradients between endocardium and epicardium. Horizontal or down-sloping ST depression, measured 60–80 ms after the J point, of 1 mm (0.10 mV) or more for 80 ms in at least three complexes is considered significant (Fig. 16.3.1.22), but the leads in which ST depression appear do not reliably localize the site of myocardial ischaemia. Other indicators of myocardial ischaemia include: • ST segment elevation—this indicates severe ischaemia due to proximal disease or coronary spasm, or an aneurysmal or dyskinetic left ventricle. Unlike exercise-induced ST segment depression, the ECG site of ST segment elevation is relatively specific for the coronary artery involved. • T-wave inversion—this may occur with exercise-induced hyperventilation. • Normalization of an inverted T

wave—this alone is not indicative of coronary disease. • U wave inversion—this is relatively specific for coronary artery disease but is relatively insensitive; in precordial leads, it usually indicates left anterior descending coronary artery disease. Exercise protocols Various protocols have been developed but the most widely used are the following. Bruce protocol This is a multistage test with 3-min walking periods during which a steady state is reached before the workload is increased by increasing the speed and slope of the treadmill. It is clearly only suitable for Fig. 16.3.1.22 ECG recorded during an exercise treadmill test, showing anterolateral ST segment depression after 3 min of exercise using the Bruce protocol.

16.3.1 Electrocardiography 3311 those whose walking is not limited by other considerations (e.g. musculoskeletal or neurological). For older patients or those with limited exercise capacity, the test can be modified to include two stages with lower workload demands. Bicycle ergometry This is often combined with radionuclide imaging (see Chapter 16.3.3), which increases the sensitivity and specificity of the test. Cycling avoids motion artefact, and so ECG recordings are clearer. The patient pedals at a comfortable speed of between 60 and 80 revolutions/ min; the test is terminated if speed cannot be maintained above 40 revolutions/min. Exercise workload begins at 25 W and resistance is increased every 2 min in 25-W increments by applying either an electronic or mechanical brake. The workload achieved during exercise is measured in metabolic equivalents or METs. This allows comparison of different protocols. A MET is 3.5 ml/min per kg, the resting Vo2 for a 40-year-old 70 kg male. METs equivalent to normal daily activities have been esti- mated (Table 16.3.1.2). Conducting the exercise test Who should have an exercise test? Deciding who should and who should not undergo an exercise test requires clinical judgement and the test should not be organized as a routine. Exercise testing is used to: • assess functional capacity and estimate prognosis in the evalu- ation of chest pain; • assess patients with known coronary artery disease; • establish prognosis after myocardial infarction either predischarge (submaximal test) or 4–6 weeks post-discharge (symptom-limited); • assess the effectiveness of coronary revascularization; • assess patients with symptoms of exercise-induced cardiac arrhythmia; • risk-stratify before noncardiac surgery in patients with or at high risk of coronary disease; • determine the efficacy of rate-responsive pacemakers. Exercise testing may also be indicated in selected asymptomatic individuals: • in specific occupations for licensing purposes (e.g. airline pilots, bus, or heavy goods vehicle drivers); • with more than two cardiovascular risk factors for risk stratification; • wishing to commence a strenuous exercise programme; • to assess cardiovascular risk due to prior to major surgery. Who should not have an exercise test? In its 2016 guideline on assessing chest pain of recent onset, the National Institute for Health and Care Excellence (NICE) recom- mended that exercise testing should no longer be used to diagnose or exclude stable angina in those without known coronary artery disease. Some conditions are considered to be absolute contraindi- cations to exercise testing but even in these patients a submaximal test may be informative. Exercise testing is inappropriate: • in healthy individuals with a low-risk factor profile—the false- positive rate is increased (see next); • with unstable medical conditions such as unstable angina; se- vere congestive cardiac failure; uncontrolled ventricular or supraventricular arrhythmia; myocarditis; severe pulmonary hypertension; drug toxicity; haemodynamic instability; symp- tomatic aortic stenosis; active thromboembolic disease; hyper- tension with systolic blood pressure more than 200 mm Hg or diastolic blood pressure more than 110 mm Hg; • in extreme obesity; • when taking specific medication—digoxin depresses the ST seg- ment (Fig. 16.3.1.23); type 1 antiarrhythmics and tricyclic anti- depressants may be proarrhythmic; • in vasoregulatory disorders—pulse and blood pressure changes are unpredictable. Patients with aortic stenosis may fail to report

symptoms of an- gina, breathlessness, and syncope. Although severe symptomatic aortic stenosis is considered an absolute contraindication to exer- cise testing, a medically supervised symptom-limited test in those who appear to be asymptomatic during their everyday activities may identify those who warrant cardiac catheterization and valve replacement. Who should supervise an exercise test—cardiac technician, specialist nurse, or physician? Patients with new or recent-onset chest pain thought to be angina are often referred to a rapid-access chest pain clinic for assessment, where a specialist nurse carries out an initial assessment and then an exercise test. Experience shows that this approach is safe, provided a physician is available for consultation and advice. There are some high-risk situations where the test, if it must be carried out, should Table 16.3.1.2 Table of MET equivalents Occupation METs Activity METs Receptionist 1–2 Carrying a suitcase 7 Professional (active) 1.5–2.5 Cleaning floor 4 Homemaker 1.5–4 Washing clothes 5 Farm worker 3.5–7.5 Cooking 3 Construction worker 4–8.5 Gardening 4 Miner 4–9 Push mower 5 Postal carrier 2.5–5 Sex 5 Bed-making 5–6 Fig. 16.3.1.23 Depression of the ST segments caused by digoxin.

section 16 Cardiovascular disorders 3312 be supervised by a physician. These include patients whose symp- toms are unstable, aortic stenosis, known severe coronary disease, severe or moderate systemic or pulmonary hypertension, severe left ventricular dysfunction, congestive or hypertrophic cardiomy- opathy, or a history of ventricular tachycardia, or second or third- degree atrioventricular block. Risks of exercise testing Exercise testing is generally considered a safe procedure but full resuscitation facilities, including defibrillator, emergency drug kit, airways management equipment, and oxygen are essential. Serious complications are rare. The risk of myocardial infarction and sudden death is less than 1 in 1000, more when testing pa- tients after myocardial infarction or with malignant ventricular arrhythmia. When to stop an exercise test Reasons for stopping a test include: • achieving 90% of the maximum predicted heart rate; • symptoms—establish if these are typical symptoms of chest pain or breathlessness; exercise may continue provided that symptoms are not distressing or severe; • systolic blood pressure—if systolic blood pressure falls below baseline levels or if systolic increases to greater 250 mm Hg or dia- stolic to greater than 115 mm Hg; • change in ECG—if more than 2 mm ST segment depression or more than 1 mm ST segment elevation; or if LBBB (this may look remarkably like ventricular tachycardia at fast heart rate) or ar- rhythmia develops; • clinical signs—if signs of poor peripheral perfusion such as cyan- osis appear; • symptoms of central nervous system dysfunction—dizziness, near syncope, or ataxia; • serious arrhythmia—ventricular tachycardia, multifocal ectopics, ventricular couplets; • technical difficulties—failure of blood pressure recording or poor ECG trace; • patient request—distressing symptoms of fatigue, breathlessness, wheeze, or claudication; maximal patient effort; or inability to maintain speed of treadmill. Recovery period It is important to observe the patient into the recovery period until the pretest heart rate and blood pressure have been restored. Minor ECG abnormalities early in recovery are common but late changes usually indicate myocardial ischaemia. Interpreting the results of an exercise test Like all medical tests, the exercise test is not a perfect indicator of the presence or absence of disease. Nevertheless, a test is often described as: • positive—chest pain develops with or without ST displacement; blood pressure falls; arrhythmia occurs; the patient fails to com- plete the first two stages of the Bruce protocol or reach 90% of predicted maximum heart rate; • negative—the patient completes uneventfully three stages of the Bruce protocol or reaches 90% of predicted maximum heart rate; • indeterminate—90% predicted heart rate is not reached; symp- toms occur which are not typical of cardiac pain with a normal ECG throughout. A positive test does not necessarily mean that the

patient has cor- onary disease, nor does a negative test mean the patient has some other, noncardiac, cause for chest pain. The exercise test has limited use as a diagnostic test for coronary disease. Limitations and strengths of the exercise test The exercise test as a diagnostic tool The sensitivity of the exercise test, the proportion with coronary dis- ease correctly identified by the test, is 68% (range 23–100) and spe- cificity, the proportion free of disease correctly identified by the test, is 77% (range 17–100). In multivessel disease, these figures are 81% (range 40–100) and 66% (range 17–100), respectively. This means that exercise testing frequently yields false-positive results, incorrectly diagnosing disease when coronary arteries are normal or minimally diseased; and false-negative results, missing coronary disease when a flow-limiting, even critical left main stem, coronary stenosis is present. Selection of patients for exercise testing is important as a false-positive result is more likely when an individual has few predisposing risk factors for coronary disease or the prevalence of coronary dis- ease prevalence in the population is low. Example 1 A positive test in a middle-aged man with multiple coronary risk factors (smoking, dyslipidaemia, hypertension, diabetes mellitus, and family history) and typical chest pain on exertion (who is highly likely to have coronary disease) is most likely to be correct. Example 2 A positive test in a young woman with atypical chest pain and few or no cardiovascular risk factors is likely to be incorrect and may lead to other, more invasive tests including coronary angiography. The prevalence of coronary disease is lower in women than men and the specificity of exercise testing is lower in women, which means that the test is more likely to be positive in the absence of coronary disease, possibly due to increased catecholamine secretion during exercise contributing to coronary vasoconstriction. The exercise test as an indicator of prognosis Although the exercise test is of limited value as an aid to diagnosis, it is more reliable as a marker of prognosis. Generally, appearance of symptoms or ECG changes early in the test is associated with more severe and extensive coronary dis- ease and a poor prognosis (Table 16.3.1.3). Changes within the first 3 min usually indicate severe coronary disease affecting the left main stem or the proximal segments of at least one major cor- onary artery. Multivessel coronary disease is more likely with ST segment down-sloping, delayed ST normalization after exercise, increased number of leads affected, and lower workload at which ECG changes appear.

 Difficulties with exercise testing Baseline ECGs that make interpretation of the exercise
test difficult ECG patterns that may make exercise-induced changes hard to recognize include: • ST depression or elevation at rest • Ventricular strain patterns—left and right ventricular hypertrophy; • T wave changes—inversion secondary to previous infarction or ‘strain’ • Conduction abnormalities—LBBB affects ST segment and T wave; RBBB affects ST segment and T wave changes in V1, V2, and V3 • Prolonged QT interval Alternative tests that do not rely on the ECG to identify myo- cardial ischaemia are dobutamine stress echocardiography, radio- nuclide thallium, or myocardial perfusion imaging (MIBI) stress test, or cardiac MRI (see Chapters 16.3.2 and 16.3.3). Medication and exercise testing β-Blockers and rate-modifying calcium antagonists may mask myocardial ischaemia by limiting exercise-induced tachycardia and so delay the appearance of ST depression. Blood pressure- lowering medication may blunt the normal exercise-induced rise in pressure. Digoxin may induce or accentuate ST depression on the resting ECG. Medication may be continued if the indication for exercise testing is to assess the efficacy of treatment but should be temporarily stopped in all other circumstances. Specific rules apply if assessing for driving licensing purposes—always check local rules, but gen- erally, antianginal drugs must be stopped at least 48 h prior to the assessment. ST segment depression in the absence of symptoms Asymptomatic, exercise-induced ST segment depression, or ‘silent ischaemia’, is seen in 60% of patients with

coronary disease but does not increase the risk of cardiac death compared with those who re- port angina. Technical issues Current ECG machines filter out motion and muscle artefact to facilitate measurement of the ST segment. Because leads placed on the limbs produce motion artefact, moving these to the torso exaggerates the degree of change and increases the amplitude of the R wave, potentiating exercise-induced ST segment changes. It can be difficult to identify ST segment depression during exercise. If there is any doubt about the extent of ST segment depression on the running ECG, most automated machines will provide a filtered 12-lead ECG for comparison with baseline. Exercise testing in special groups Peri-and post-myocardial infarction Exercise testing after myocardial infarction may be performed for risk stratification and selection for revascularization. A submaximal predischarge test to identify residual ischaemia appears to be safe, with 0.05% morbidity and 0.02% mortality. An abnormal blood pressure response or low exercise capacity predicts a poor outcome and is an indication for urgent revascularization. Evidence of myo- cardial ischaemia, especially at low workload, is an indication for referral for coronary angiography. Elderly patients Advanced age alone is not a contraindication to exercise testing, provided that the individual can walk at a reasonable speed. If mobility is limited, dobutamine stress echocardiography, radionuclide thallium, or MIBI stress test, or cardiac MRI are alternative means of identifying ischaemia (see Chapters 16.3.2 and 16.3.3). Asymptomatic individuals Testing may be undertaken in asymptomatic individuals, gener- ally a low-risk population, as part of health screening, for insur- ance purposes, or for risk stratification. Up to 12% of middle-aged men and up to 30% of women will have an abnormal exercise test in the absence of symptoms; the risk of a cardiac event is low unless the test result indicates a poor prognosis. The presence of cardiovascular risk factors increases the likelihood of coronary disease. Cardiac arrhythmia Exercise testing can be useful in evaluating cardiac arrhythmia, supplementary to ambulatory monitoring and electrophysiological studies. In about 10%, it may provoke an arrhythmia. Table 16.3.1.3 Prognostic indicators on treadmill testing Indicators of a good prognosis Indicators of a poor prognosis ST segment No displacement or up-sloping 2 mm or more depression in stage 1 Bruce-within 3 minutes Down-sloping or horizontal Duration of exercise 9 minutes (>9 METs) Unable to complete stage 2 Bruce or equivalent (<6.5 METs) Heart rate at onset of limiting symptoms Reaches maximum predicted heart rate (220 – age) Unable to attain >120/min off β-blocker Systolic BP response Maintained or increased Sustained decrease >10 mm Hg or failure to rise with exercise Changes on exercise No changes Ventricular tachycardia U wave inversion T wave normalization Recovery Recovers normal heart rate <10 min Delayed recovery >10 min Symptoms None or atypical Test terminated due to increasing angina on exercise

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