Bennett’s Cardiac Arrhythmias
Practical Notes on Interpretation and Treatment, 8th Edition

David H. Bennett MD FRCP, Senior Consultant Cardiologist, University Hospital of South Manchester, Manchester, UK

A trusted source for junior doctors, students, nurses and cardiac technicians for over 30 years, the new edition of this classic reference continues the winning formula of previous editions while at the same time incorporating essential new content on today’s most important clinical topics, including:

- Atrial fibrillation: ablation, drugs, rate control versus rhythm control, risk of systemic embolism, prognosis
- Indications for and management of implantable defibrillators including complications such as arrhythmia storms
- Indications for pacemaker implantation
- Anticoagulant therapy (for atrial fibrillation)
- Long QT syndromes and other channelopathies
- Recently-approved anti-arrhythmia drugs

The 8th edition also features the latest guidelines on ECG screening of athletes and clear guidance for anaesthetists and surgeons dealing with patients with arrhythmias and/or implantable devices.

Rich with example ECGs and designed for ease of access to information, Bennett’s Cardiac Arrhythmias is the reference you can trust to help you master arrhythmia diagnosis and provide optimal treatment of any patient under your care.

Reviews of previous editions:

‘...a well conceived practical guide to the interpretation and treatment of the main cardiac rhythm disturbances’ Lancet

‘This book presents a concise and simplified approach to the diagnosis and management of abnormalities in cardiac rhythm...One of the book’s strengths is the number and quality of electrocardiographic tracings’ New England Journal of Medicine

‘...this book provides an excellent foundation for all those involved in the care of arrhythmia patients’ British Journal of Hospital Medicine

‘...would recommend it unreservedly to anaesthetists who wish to improve their knowledge of cardiac arrhythmias’ British Journal of Anaesthesia

‘This book about cardiac arrhythmias is of much educational value’ European Heart Journal

Also of Interest:
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Practical Notes on Interpretation and Treatment

8th Edition

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University Hospital of South Manchester
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There are several large textbooks which comprehensively cover the field of cardiac arrhythmias with thorough referencing of scientific papers. This book does not attempt to replicate these texts. The purpose of this eighth edition (the first edition was published in 1981, and there have been translations into five other languages) remains the same as that of its predecessors: to provide a concise, up-to-date, practical guide to the diagnosis, investigation and management of the main cardiac arrhythmias, with particular emphasis on the problems commonly faced in practice.

In order to be proficient in the interpretation of arrhythmias it is necessary to study a range of examples of each rhythm disturbance. For this reason, it has always been a purpose of this book to present a large number of electrocardiograms so that the reader can gain experience in ECG interpretation and can test him- or herself out, and thereby gain confidence, during the reading of the book. In this edition there are many new electrocardiograms, and the quiz section has been revised and enlarged to provide a challenge to those who may be familiar with previous editions.

The book has been written with junior hospital doctors in mind. They receive little formal training in the management of cardiac arrhythmias and yet, because prompt action is often required, the onus of diagnosis and treatment usually falls on them. It should also be of interest to medical students, who themselves will soon be responsible for dealing with arrhythmias, to nurses working in coronary and intensive care units, to cardiac technicians/physiologists, whose responsibilities nowadays include a major input into arrhythmia management, and to physicians who want a brief review of the practical aspects of cardiac arrhythmias. In recent years there has been a trend to sub-specialisation in cardiology. Whatever the sub-speciality, cardiac arrhythmias will frequently be encountered. An appreciation of the significance and management of cardiac arrhythmias is required of all who treat patients with cardiac disease.

I am most grateful to my technical, medical and nursing colleagues for their help, and to the staff of my new publisher at Wiley-Blackwell for their expertise.

The title of this edition, Bennett’s Cardiac Arrhythmias, was chosen by the publisher to indicate that the author has described the major disturbances of heart rhythm, not, at least to date, that the author has experienced all of them!

The book is dedicated to my family, Irene, Samantha and Sally.

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The electrocardiograms in this book have been recorded at the conventional paper speed of 25 mm/s, unless otherwise indicated. At this speed, each large square represents 0.2 s and each small square represents 0.04 s. Heart rate (beats/minute) can therefore be calculated by dividing the number of large squares between two consecutive complexes into 300, or by dividing the number of small squares between two complexes into 1500.

A single ECG ‘rhythm strip’ may be inadequate for diagnosis. Scrutiny of several ECG leads, preferably recorded simultaneously, may be necessary. For example, atrial activity is often the key to diagnosis but may not be clearly shown in all ECG leads: it is often best seen in leads II and V1. Frequently, a ‘12-lead ECG’ will provide much more information than a rhythm strip.

An ECG recorded during an arrhythmia that is of diagnostic importance should always be safely stored in the patient’s notes. This guideline, which may be very important to the long-term management of a patient, is often ignored, particularly on intensive and coronary care units!
1 Sinus Rhythm

The sinus node lies at the junction of the superior vena cava and right atrium. Atrial activation travels inferiorly from the sinus node to the atrioventricular (AV) node, resulting in a positive P wave in the inferior ECG leads, II, III and aVF. If the QRS complex is preceded by a P wave that is not positive in the inferior leads then the rhythm is other than sinus rhythm. The sinus node impulse is conducted relatively slowly via the AV node to reach the His–Purkinje system, which then conducts very rapidly to activate the ventricular myocardium.

Normal sinus rhythm is characterised by a rate of 60–100 beats/min; PR interval 0.12–0.21 s; QRS duration ≤0.10 s; QTc ≤0.44 s.

**ECG characteristics**

The sinus node initiates the electrical impulse that activates atrial and then ventricular myocardium during each normal heartbeat. Sinus node activity itself does not register on the electrocardiogram (ECG).

**P wave**

Atrial activity, the P wave, is usually apparent in most ECG leads (Figure 1.1). However, occasionally the P wave in some leads is not visible or is of low amplitude, and it may be necessary to inspect all leads of the ECG to establish that there is sinus rhythm (Figure 1.2).

The sinus node lies at the junction of the superior vena cava and right atrium. *Atrial activation therefore spreads from the sinus node in an inferior direction* (i.e. towards the feet) to the atrioventricular (AV) junction. The P wave, therefore, is upright in those leads that are directed to the inferior surface of the heart (i.e. II, III and aVF), and is inverted in aVR, which faces the superior heart surface (Figure 1.1). If a P wave does not have these characteristics then, even though a P wave precedes each ventricular complex, the sinus node has not activated the atria and the rhythm is abnormal (Figure 1.3).

**PR interval**

*The AV node is the only electrical connection between atria and ventricles*: the mitral-tricuspid valve ring that separates the atria from the ventricles is fibrous and cannot
conduct electrical impulses. The AV node conducts relatively slowly, thereby delaying conduction of the atrial impulse to the ventricles. Conduction through the AV node does not register on the ECG. The PR interval, which is measured from the onset of the P wave to the onset of the ventricular complex, indicates the time taken for an atrial impulse to reach the ventricles. The normal PR interval ranges from 0.12 to 0.21 s. It should shorten during sinus tachycardia.

**QRS complex**

After traversing the AV node, the activating impulse reaches the bundle of His, which divides into the right and left bundle branches. The bundle of His, the bundle branches and their ramifications, the Purkinje fibres, constitute the ‘specialised intraventricular conducting system’ which facilitates very rapid conduction of the impulse through the ventricular myocardium. Ventricular activation (i.e. depolarisation) is represented by the QRS complex, which is normally no greater than 0.10 s in
Chapter 1: Sinus Rhythm

The amplitude of the QRS complex is larger than that of the P wave because the mass of the ventricles is much greater than that of the atria.

**T wave**

The T wave is the result of the electrical recovery of ventricular myocardium prior to the next heartbeat, i.e. repolarisation. Sometimes, a low-amplitude wave can be seen following the T wave, termed a U wave. It is thought to result from repolarisation of the Purkinje fibres and is usually seen in leads V2–4.

The QT interval, which is measured from the onset of the QRS complex to the end of the T wave, represents the duration of ventricular activation plus recovery. The QT interval normally shortens with increasing heart rate, partly due to the increase in rate itself and partly due to the increase in sympathetic nervous system activity related to sinus tachycardia. When measuring the QT interval it is necessary to correct the measured interval for heart rate. The corrected QT interval (QTc) is calculated by selecting the ECG lead showing the longest QT interval, and then dividing the square root of the cycle length into the measured QT interval. For example, a patient with a

**ECG characteristics of normal sinus rhythm**

<table>
<thead>
<tr>
<th>Component</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td><strong>P wave</strong></td>
<td>Precedes each QRS complex</td>
</tr>
<tr>
<td></td>
<td>Upright in leads III, aVF</td>
</tr>
<tr>
<td></td>
<td>Inverted in lead aVR</td>
</tr>
<tr>
<td><strong>PR interval</strong></td>
<td>Duration 0.12–0.21 s</td>
</tr>
<tr>
<td><strong>QRS complex</strong></td>
<td>Duration ≤ 0.10 s</td>
</tr>
<tr>
<td><strong>QTc interval</strong></td>
<td>Duration ≤ 0.42 s (men), ≤ 0.44 s (women)</td>
</tr>
</tbody>
</table>
measured QT interval of 0.40 s at a heart rate of 60 beats/min has a cycle length of 1.0 s and therefore also has a QTc of 0.40 s. QT prolongation and a prominent U wave are seen in certain hereditary and acquired conditions.

**Relative speeds of impulse conduction**

Appreciation of the relative speeds of impulse conduction through the heart – slowest through the AV node, fastest through the specialised intraventricular conducting system and at an intermediate rate through ordinary working myocardium – is important in understanding the mechanisms of a number of arrhythmias as well as generation of the normal P-QRS complex.

<table>
<thead>
<tr>
<th>Speed of impulse conduction</th>
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<td>His–Purkinje system &gt; myocardium &gt; AV node</td>
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**Sinus bradycardia**

Sinus bradycardia is sinus rhythm at a rate less than 60 beats/min (Figure 1.4). It may be physiological, as in athletes or during sleep, or it may result from acute myocardial infarction, sick sinus syndrome or from drugs such as beta-adrenoceptor blocking drugs (beta-blockers). Non-cardiac disorders such as hypothyroidism, jaundice and raised intracranial pressure can also cause sinus bradycardia.

Atropine, isoprenaline or pacing can be used to increase the rate but are only necessary when sinus bradycardia causes symptoms or marked hypotension, or leads to tachyarrhythmia.

**Sinus tachycardia**

Sinus tachycardia is defined as sinus rhythm at a rate greater than 100 beats/min (Figure 1.5). Exercise, anxiety or any disorder that increases sympathetic nervous system activity may cause sinus tachycardia.

Occasionally, sinus tachycardia can be inappropriate. Hyperthyroidism is a possible cause. However, often no cause is found. Young females are most commonly affected. Fast rates are usually persistent and there is an exaggerated response to exercise with rates increasing rapidly almost immediately exertion begins. Rarely, inappropriate sinus tachycardia is due to a primary disorder of the sinus node (sinus node re-entry).

Since sinus tachycardia is usually a physiological response, there is rarely a need for specific treatment. However, if sinus tachycardia is inappropriate, the rate may be slowed by a beta-blocker, or by ivabradine, which is a selective inhibitor of sinus node function.

At rest, the sinus node rate is seldom above 100 beats/min unless the patient is very ill. If there is apparent sinus tachycardia at rest alternative rhythms such as atrial tachycardia or atrial flutter should be considered.

**Figure 1.4** Sinus bradycardia (lead II): rate 34 beats/min.
In sinus arrhythmia, which is of no pathological significance, there are alternating periods of slowing and increasing sinus node rate. Usually the rate increases during inspiration (Figure 1.6). Sinus arrhythmia is most commonly seen in the young.
2 Ectopic Beats

The terms ectopic beat, extrasystole and premature contraction are, for practical purposes, synonymous. They refer to an impulse originating from the atria, atrioventricular (AV) junction or ventricles that arises prematurely in the cardiac cycle.

Usually the AV junction and bundle branches will conduct an atrial ectopic to the ventricles normally, resulting in a narrow QRS complex. The prematurity of an atrial ectopic beat is such that the P wave may be superimposed on the preceding T wave.

The impulse of a ventricular ectopic beat is not conducted through the ventricles via the rapidly conducting His–Purkinje system. The resultant complexes are therefore broad (> 0.12 s) and bizarre in shape, and will not be preceded by a premature P wave. Ventricular ectopic beats are often idiopathic but when caused by cardiac disease are associated with an increased cardiovascular mortality that will not be reduced by antiarrhythmic drugs.

Prematurity

The terms ectopic beat, extrasystole and premature contraction are, for practical purposes, synonymous. They refer to an impulse originating from the atria, AV junction (i.e. the AV node together with the bundle of His) or ventricles that arises prematurely in the cardiac cycle (Figures 2.1–2.3).

By definition, an ectopic beat must arise earlier in the cardiac cycle than the next normally timed beat would be expected. Thus the interval between the ectopic beat and the preceding beat, i.e. the coupling interval, is shorter than the cycle length of the main rhythm. If this fact is ignored, other beats with abnormal configurations such as escape beats (Chapter 3) and intermittent bundle branch block (Chapter 4) may be misinterpreted as ectopic beats.

The site of origin of an ectopic beat can be determined by careful examination of the ECG. A single rhythm strip may be inadequate. Scrutiny of simultaneous recordings of several ECG leads is often necessary to detect the diagnostic clues (Figures 2.4, 2.5).
Figure 2.1 The second, fourth, sixth and eighth complexes are atrial ectopic beats. The ectopic P waves are premature and differ in shape from those of sinus origin (the PR intervals of the atrial ectopic beats are prolonged).

Figure 2.2 The fourth beat is a junctional ectopic beat (lead III). The junctional focus has activated the atria as well as the ventricles, resulting in an inverted P wave which precedes the QRS complex.

Figure 2.3 The fifth beat is a ventricular ectopic beat.

Figure 2.4 Simultaneous recording of leads V1 and V2. The third and sixth beats are unifocal ventricular ectopic beats. Their ventricular origin is not apparent in lead V1 but is obvious in V2.
Atrial ectopic beats

P wave

An atrial ectopic beat results in a P wave that is premature. The site of origin and therefore direction of atrial activation will differ from that during sinus rhythm, so a premature P wave will usually differ in shape to a P wave of sinus node origin (Figure 2.1).

Because atrial ectopic beats are premature, they may be superimposed on and thus deform the T wave of the preceding beat. Careful examination of the ECG is essential to detect ectopic P waves; often, lead V1 is the best lead (Figures 2.5, 2.6).

Atrioventricular and intraventricular conduction

Usually the AV junction and bundle branches will conduct an atrial ectopic beat to the ventricles in the same manner as if the sinus node had activated the atria. Thus the PR interval and QRS complex of the ectopic beat will be identical to those during sinus rhythm (Figure 2.1). If the QRS complex during sinus rhythm is abnormal due to bundle branch block, then so will be the QRS complex of the ectopic beat.

Sometimes, however, atrial ectopic beats, especially those that arise very early in the cardiac cycle, may encounter either an AV junction or a bundle branch which has not yet recovered from conduction of the last atrial impulse and is, therefore, partially or completely refractory to excitation. Partial and complete refractoriness of the AV junction will result in prolongation of the PR interval and blocked atrial ectopic beats, respectively (Figures 2.1, 2.6–2.8). Atrial ectopics that are not conducted to the ventricles have been wrongly taken as an indication for cardiac pacing!

Partial or complete refractoriness of one or other bundle branch (it is usually the right bundle) will correspondingly lead to partial or complete bundle branch block (Figures 2.6, 2.7). This phenomenon of functional bundle branch block is referred to by some as ‘phasic aberrant intraventricular conduction’. The resultant QRS
complexes are broad and can therefore be confused with ventricular ectopic beats if the premature P wave preceding the ventricular complex is not detected.

**ECG characteristics of atrial ectopic beats**

<table>
<thead>
<tr>
<th>The P wave of an atrial ectopic beat:</th>
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<tbody>
<tr>
<td>Is premature</td>
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<tr>
<td>May be superimposed on and distort the preceding T wave</td>
</tr>
<tr>
<td>Is usually followed by a normal QRS complex</td>
</tr>
<tr>
<td>Is sometimes not conducted to the ventricles, or is conducted with a bundle branch block pattern</td>
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</table>

**Significance**

Atrial ectopic beats occur in many cardiac disorders but are also commonly found in individuals with normal hearts, particularly the elderly. They are usually benign. However, if they are frequent they may herald atrial fibrillation or atrial tachycardia.

**Atrioventricular junctional ectopic beats**

AV junctional beats used to be called ‘nodal’ beats. It is now recognised that at least part of the AV node is not capable of pacemaker activity and that it is not possible to distinguish between beats originating from the AV node and those from the bundle of His. Hence the more general term ‘AV junctional’ is used. AV junctional ectopic beats are not as common as atrial or ventricular ectopics. Treatment is rarely necessary.

**ECG appearance**

AV junctional ectopic beats are recognised by a premature QRS complex that is similar in appearance to that occurring in sinus rhythm. The junctional focus may activate the atria as well as the ventricles, leading to a retrograde P wave (i.e. negative in leads II, III and aVF). The retrograde P wave may precede, follow or be buried within the
QRS complex, depending on the relative speeds of conduction of the premature junctional impulse to the ventricles and to the atria (Figure 2.2).

### Ventricular ectopic beats

The impulse of a ventricular ectopic beat is not conducted through the ventricles via the His–Purkinje system but through relatively slowly conducting myocardium. The abnormal course and consequent slowing of ventricular activation result in ventricular complexes that are both bizarre in shape and of prolonged duration.

### ECG appearance

The complexes are premature, broad (≥ 0.12 s), bizarre in shape and, in contrast to atrial ectopic beats, are obviously not preceded by a premature P wave (Figures 2.3, 2.4).

#### ECG characteristics of ventricular ectopic beats

<table>
<thead>
<tr>
<th>Characteristic</th>
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<tbody>
<tr>
<td>The QRS complex of a ventricular ectopic beat is:</td>
</tr>
<tr>
<td>Premature</td>
</tr>
<tr>
<td>Broad (≥ 0.12 s)</td>
</tr>
<tr>
<td>Abnormal in shape</td>
</tr>
<tr>
<td>Not preceded by a premature P wave</td>
</tr>
</tbody>
</table>

Several terms are used to describe the origin, timing and quantity of ventricular ectopic beats:

#### Focus

Ectopic beats with the same shape and coupling intervals are assumed to arise from the same focus and are termed ‘unifocal’ (Figure 2.4), whereas differing shapes and coupling intervals suggest more than one focus. These are called ‘multifocal’ or ‘multiform’ (Figure 2.9).

#### Timing

Beats that occur very early in the cardiac cycle will be superimposed on the T wave of the preceding beat and are described as ‘R on T’ (Figure 2.10). Most episodes of ventricular fibrillation and many episodes of ventricular tachycardia are initiated by ‘R on T’ ectopics; though by no means do all ‘R on T’ ectopic beats precipitate these arrhythmias.

A ventricular ectopic beat that occurs only slightly prematurely in the cardiac cycle may fall, by chance, immediately after a P wave initiated by normal sinus node activity: the P wave will not, therefore, in contrast to an atrial ectopic beat, be premature. Such a ventricular ectopic beat is described as ‘end-diastolic’ (Figures 2.11, 2.12).

![Figure 2.9 Multifocal ventricular ectopic beats. The second ventricular ectopic beat has a different shape and coupling interval from the first and third ectopic beats.](image-url)
Usually there is a pause after a ventricular ectopic beat. When there is no such pause and the ectopic beat is thus ‘sandwiched’ between two normal beats, the ectopic beat is said to be ‘interpolated’ (Figure 2.13).

Figure 2.10 An “R on T” ventricular ectopic beat, which in this case initiates ventricular fibrillation.

Figure 2.11 The third beat is an end-diastolic ventricular ectopic beat. It is preceded by a normally timed P wave.

Figure 2.12 Simultaneous recording of leads V1 and V2. Two end-diastolic ventricular ectopic beats. The second mimicking the Wolff–Parkinson–White syndrome.
Frequency
When an ectopic beat follows each sinus beat the term 'bigeminy' is applied (Figure 2.14). If an ectopic follows a pair of normal beats there is 'trigeminy' (Figure 2.15). When two ectopics occur in succession (Figure 2.16) they are referred to as a 'couplet'. A 'salvo' refers to more than two ectopic beats in succession.

Atrial activity
The pattern of atrial activity following a ventricular ectopic beat depends on whether the AV junction transmits the ventricular impulse to the atria. If this occurs, the result is an inverted P wave which is often superimposed on and may therefore be concealed
by the ventricular ectopic beat (Figure 2.17). When the AV junction does not transmit
the ventricular impulse to the atria, atrial activity continues independently of ven-
tricular activity; it is only in these cases that a ventricular impulse is followed by a full
compensatory pause (i.e. the lengths of the cycles before and after the ectopic beat
will equal twice the sinus cycle length) (Figures 2.3, 2.4).

Sometimes a ventricular impulse only partially penetrates the AV junction. The
next impulse arising from the sinus node may therefore encounter an AV junction that
is partially refractory and be conducted with a prolonged PR interval (Figure 2.13).
This phenomenon of ‘retrograde concealed conduction’ often occurs following inter-
polated ventricular extrasystoles.

Causes and significance of ventricular ectopic beats
Ventricular ectopic beats are very common, and their frequency in the general adult
population increases with age. Causes of ventricular ectopic beats include acute myo-
cardial infarction; myocardial ischaemia; hypertension; myocardial damage caused
by previous infarction, myocarditis or cardiomyopathy; mitral valve prolapse; valvu-
lar heart disease and digoxin toxicity; but frequently, there will be no evidence of heart
disease.

In patients presenting with symptomatic and/or frequent ventricular ectopic beats
a cause should be sought by use of non-invasive tests including scrutiny of the 12-lead
ECG, echocardiography and, where appropriate, exercise testing.

Occasional ventricular ectopic beats during routine electrocardiography, and even
complex ectopic beats (i.e. frequent, multifocal, ‘R on T’ or those that occur in salvos)
during ambulatory electrocardiography, can be found in subjects with otherwise nor-
mal hearts and are not necessarily pathological or of prognostic significance. On the
other hand, in several surveys of adult, predominantly male, subjects referred for
exercise testing, frequent ventricular ectopic beats during and particularly immedi-
ately after exercise have been shown to be associated with an increased mortality
(approximately x 3) in follow-up periods of 5–15 years.

In patients who have sustained myocardial damage from coronary heart disease,
there is a correlation between severity of damage and frequency of ventricular ectopic
beats. Recent evidence, however, points to the presence of ectopic beats as an added
and independent risk factor, but there is no evidence to show that suppression of
ectopic beats by antiarrhythmic therapy improves prognosis. Indeed, several antiar-
rhythmic drugs have been shown to increase mortality in patients with ventricular
ectopic beats after myocardial infarction.

Ectopic beats are usually asymptomatic. Some patients, however, do experience
distressing symptoms. They may be upset by the irregularity resulting from the
premature beats or by the compensatory pause or ‘thump’ caused by increased
myocardial contractility associated with the post-ectopic beat. They may be anxious
that their irregular heart rhythm is a sign of impending heart attack or other major
cardiac problem.
There is a group of patients with structurally normal hearts with distressing symptoms caused by ventricular ectopic beats in whom reassurance is inadequate. In these patients, therapy may be necessary for symptomatic purposes. Beta-blockers may help, particularly in patients whose symptoms are related to exertion. Flecainide is useful, provided the patient has a structurally normal heart and there is no evidence of coronary disease. Caffeine avoidance is frequently advised but rarely effective.

The significance of ventricular ectopic beats in acute myocardial infarction is discussed in Chapter 18.
Escape beats may arise from the AV junction or ventricles when there is sinus bradycardia or sinus arrest. In contrast to ectopic beats, the coupling interval of escape beats is greater than the cycle length of the main rhythm. The configuration of junctional escape beats is the same as that of normally conducted beats, whereas ventricular escape beats are of similar appearance to ventricular ectopic complexes. Escape beats themselves require no treatment. If treatment is necessary, it is to accelerate the basic rhythm.

**Timing**

When there is sinus bradycardia or the sinus node fails to discharge, escape beats may arise from secondary sites in the specialised conducting system. *In contrast to ectopic beats, escape beats are always late*, i.e. the coupling interval is greater than the cycle length of the dominant rhythm (Figure 3.1). Distinction between escape and ectopic
beats is important, because the former indicate impaired sinus node function. *Escape beats themselves require no treatment.* If treatment is necessary, it is to accelerate the basic rhythm.

**Origins**

Escape beats usually arise from the AV junction (Figures 3.1, 3.2); less commonly, they originate from the ventricles (Figure 3.3). The ventricular complexes of junctional escape beats are similar to those during normal rhythm because the impulse will be conducted normally via the His bundle and bundle branches. As with junctional ectopic beats, the junctional focus may activate the atria as well as the ventricles, leading to a retrograde P wave, i.e. inverted in leads II, III and aVF. The retrograde P wave may precede, follow or be buried within the QRS complex, depending on the relative speeds of conduction of the premature junctional impulse to the ventricles and to the atria.

Ventricular escape beats have a configuration similar to that of ventricular ectopic beats (Figure 3.3).
4 Bundle Branch and Fascicular Blocks

Right and left bundle branch block, and block in the left anterior and posterior fascicles of the left bundle branch, are commonly encountered.

Complete bundle branch block prolongs QRS duration to 0.12 s or greater. With right bundle branch block, there will be a secondary R wave in lead V1, resulting in an M-shaped complex. With left bundle branch block, there is no M-shaped complex in V1; there will be a notched complex in left ventricular leads.

Diagnosis of fascicular block requires an understanding of the hexaxial reference system. With left axis deviation, lead I is predominantly positive and both leads II and III are predominantly negative. The criteria for left anterior fascicular block are left axis deviation together with a small initial r wave in leads II and aVF: inferior infarction also leads to left axis deviation but there will be a Q rather than r wave in these leads.

The bundle of His divides into left and right bundle branches. These facilitate very rapid activation of the left and right ventricles. Block in conduction through one or other bundle branch results in delayed and disordered activation of ventricular myocardium, as evidenced on the ECG by a ventricular complex which is prolonged in duration and has an abnormal configuration.

**Right bundle branch block**

**ECG appearance**
In right bundle branch block there is delay in activation of the right ventricle, while activation of the interventricular septum and free wall of the left ventricle and hence the initial part of the QRS complex is normal (Figure 4.1). Delayed right ventricular activation results in:
1. an increase in duration of the QRS complex (≥ 0.12 s);
2. a secondary R wave in leads facing the right ventricle (V1 and V2) and hence an M-shaped complex in these leads; and
3. a broad S wave in left ventricular leads and lead I.
Partial right bundle branch block results in a similar ECG appearance but the QRS duration is 0.10 or 0.11 s.

**Causes and significance**
Right bundle branch block may be an isolated congenital lesion. It often occurs in congenital heart disease, in other causes of right ventricular hypertrophy or strain such as obstructive airways disease, and where there is myocardial damage. Right bundle branch block is common when there is disease of the specialised conducting tissues.

Based on limited data, neither pre-existing nor acquired right bundle branch block are of prognostic significance. However, a recent long-term survey has demonstrated a four-fold increased risk of developing AV block.

Extrasystoles and tachycardias of supraventricular origin may encounter a right bundle branch that is refractory to excitation and be conducted to the ventricles with a right bundle branch block pattern.

**Left bundle branch block**

**ECG appearance**
In left bundle branch block, activation of the interventricular septum is in the opposite direction to normal (i.e. from right to left), being initiated by an impulse arising from the right bundle branch. Thus:

1. The initial small, negative q wave normally seen in left ventricular leads (V5, V6, I and aVL) is replaced by a larger, positive R wave.
2. Activation of the left ventricle will be delayed, resulting in a broad and usually notched R wave in left ventricular leads, and prolongation of the duration of the QRS complex (≥ 0.12 s) (Figure 4.2).
Partial left bundle branch block has a similar ECG appearance to complete left bundle branch block, but the QRS duration is 0.10 or 0.11 s.

There is a simple, pragmatic rule to help distinguish right from left bundle branch block. Assuming the absence of Wolff–Parkinson–White syndrome or gross ventricular hypertrophy, if during normal rhythm (or indeed a supraventricular tachycardia) the QRS duration is ≥ 0.12 s then there is bundle branch block. If the ventricular complex in lead V1 is M-shaped there is right bundle branch block, if it is not there is left bundle branch block.

Causes and significance
Causes of left bundle branch block include myocardial damage due to coronary artery disease or cardiomyopathy, and left ventricular hypertrophy. Left bundle branch block can also be caused by disease of the specialised conduction tissues.

Recently acquired left bundle branch block is associated with an increased risk of death: mainly sudden death from coronary disease. In addition, an 18-fold risk of developing AV block in the long term has recently been reported.

Supraventricular extrasystoles and tachycardias may encounter a left bundle branch that is refractory to excitation and be conducted to the ventricles with a left bundle branch block pattern.

Left bundle branch block can be intermittent.

Left anterior and posterior fascicular blocks
The left bundle branch has two main subdivisions, the anterior and posterior fascicles, which conduct impulses to the anterosuperior and posteroinferior regions of the
left ventricle, respectively. Block can occur in either anterior or posterior fascicle and is known as fascicular block or hemiblock. Left anterior and posterior fascicular block are common in conduction tissue disease, and one or other together with right bundle branch block, i.e. bifascicular block, can herald the onset of high-degree atrioventricular block (Chapter 15).

Diagnosis of the fascicular blocks is based on the hexaxial reference system.

**Hexaxial reference system**

This is a method of displaying the orientation of the six ECG limb leads to the heart in the frontal plane, i.e. the vertical plane that runs through the centre of the body, dividing it into anterior and posterior regions (Figure 4.3). For example, a superiorly directed impulse will move away from leads II, III and aVF, producing a negative wave in these leads, and towards aVL, producing a positive wave in this lead. The direction of an impulse can be expressed by the number of degrees clockwise (positive) or anticlockwise (negative) of lead I, which is the zero reference point. For example, an impulse towards lead aVL has an axis of –30 degrees and an impulse towards lead III has an axis of +120 degrees (Figure 4.3).

**Mean frontal QRS axis**

The mean frontal QRS axis describes the dominant or average direction of the various electrical forces that develop during ventricular activation. Normally, the mean frontal QRS axis lies between aVL (i.e. –30 degrees) and aVF (i.e. +90 degrees).

If the axis is to the left of aVL (i.e. less than –30 degrees), it is termed abnormal left axis deviation. If the axis is to the right of aVF (i.e. more than +90 degrees), there is right axis deviation.

Using the hexaxial reference system, the mean frontal QRS axis may be calculated to within a few degrees. However, this degree of precision is unnecessary and most