Cardiac Arrhythmias

Practical Notes on Interpretation and Treatment

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Preface to the Second Edition

The purpose of this second edition remains the same: to provide a practical guide to the diagnosis, investigation and management of the main cardiac arrhythmias with particular emphasis on the problems commonly encountered in practice.

The author is grateful for the generous reviews of the first edition and has made a point of responding to suggestions reviewers have made. Since the first edition, new anti-arrhythmic drugs have been introduced, a substantial amount of new information about the older drugs has been acquired, approaches to the management of ventricular arrhythmias (particularly in acute infarction) have been modified and there have been important improvements in cardiac pacing.

Accordingly, the sections relating to treatment have been extensively updated and expanded. Elsewhere, changes to the text have been made to highlight areas where there are common pitfalls.

A summary has been added at the end of each chapter to draw attention to the main points and to highlight those facts of practical importance that are often forgotten or neglected.

A number of new ECGs and illustrations have been added and the quiz section at the end of the book has been increased in size.

D.H.B.

Preface to the First Edition

The purpose of this book is to describe the main cardiac arrhythmias, with particular emphasis on the problems commonly encountered in their interpretation, and to discuss the practical aspects of current methods of investigation and treatment. Information of purely academic value has not been included.

This book is intended to fill the gap between those textbooks that cover only the basics of arrhythmias and those that are written for the cardiac electrophysiologist. It 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 and to physicians who want a brief review of the practical aspects of cardiac arrhythmias.

I would like to thank the cardiac technicians, coronary care nurses and medical staff at Wythenshawe Hospital for their help. I am particularly grateful to my colleagues, Dr Colin Bray and Dr Christopher Ward.

Thanks are also due to Mrs Mary Rooney for typing the manuscript and to the Wythenshawe Hospital Medical Illustration Department.

Finally, I would like to acknowledge the distractions provided by my family, Irene, Samantha and Sally, to whom this book is dedicated.

D.H.B.

Note

Unless otherwise indicated, the electrocardiograms in this book have been recorded at a paper speed of 25 mm/s. At this speed, each large square represents 0·2 s and each small square represents 0·04 s.

Heart rate (beats/minute) can 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.

In order to include all the necessary features in the electrocardiograms, some records have had to be reduced in size.

Contents

Chapter 1

Sinus Rhythm

The sinus node is the primary pacemaker of the heart, initiating the electrical activity that leads to the orderly activation of atrial and then ventricular myocardium during each heart beat. Sinus node activity does not register on the electrocardiogram (ECG).

Atrial activity, the P wave, can be seen in most ECG leads (*Fig.* 1.1). Sometimes the P wave is of low amplitude and it may be necessary to inspect all leads of the ECG to establish that the patient is in sinus rhythm (*Fig.* 1.2).

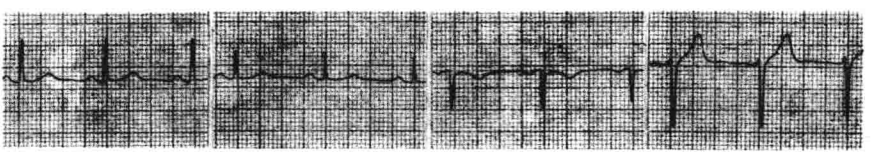

Fig. 1.1. Sinus rhythm (leads I, AVF, AVR and V2). Atrial activity is clearly seen in the limb leads but is only just discernible in V2.

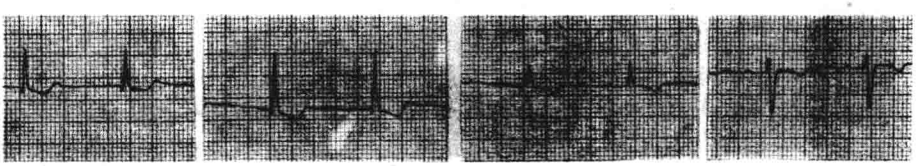

Fig. 1.2. Sinus rhythm with low amplitude P waves (leads I, II, III and V1). Atrial activity is only clearly seen in V1.

Atrial activation spreads from the sinus node, which lies at the junction of the superior vena cava and right atrium, in an inferior direction. The P wave, therefore, is upright in leads II, III and AVF, which are orientated towards the inferior surface of the heart, and is inverted in AVR, which is orientated towards the superior heart surface (*Fig.* 1.1). If the P wave does not have these characteristics then, even though each ventricular complex is preceeded by a P wave, the rhythm is abnormal (*Fig.* 1.3).

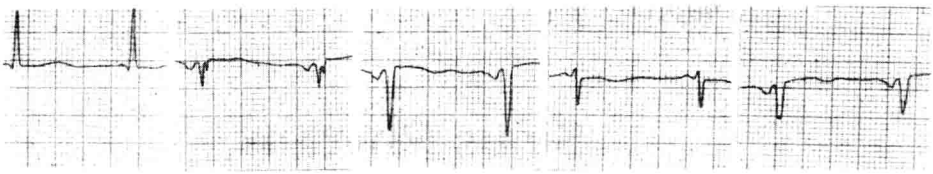

Fig. 1.3. Junctional rhythm (leads I, II, III, AVR, AVF): a P wave precedes each QRS complex but is superiorly directed.

The atrioventricular (AV) node delays the transmission of the activating impulse from atria to ventricles. This is reflected by the PR interval, which is measured from the onset of the P wave to the onset of the ventricular complex. The normal PR interval ranges from 0·12 to 0·21 s. It shortens with increasing heart rate.

After traversing the AV node, the electrical impulse is conducted very rapidly by the bundle of His and right and left bundle branches to the ventricular myocardium. Ventricular activation is represented by the QRS complex which, in the absence of bundle branch block, should be less than 0·08 s in duration.

SINUS BRADYCARDIA

This is defined as sinus rhythm at a rate less then 60/min (*Fig.* 1.4). It may be physiological, as in athletes, or secondary to acute myocardial infarction, sick sinus syndrome or beta-adrenoceptor blocking drugs. Non-cardiac disorders such as myxoedema, jaundice and raised intracranial pressure can also cause sinus bradycardia.

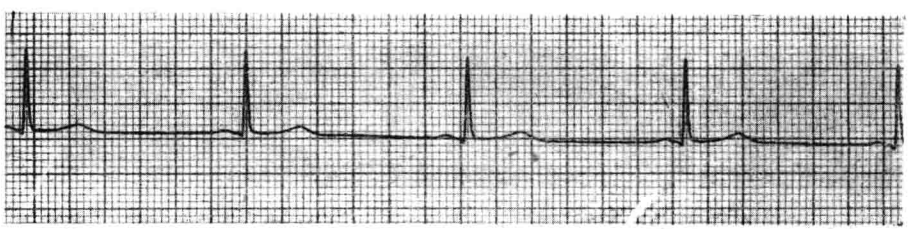

Fig. 1.4. Sinus bradycardia. Rate is 48/min.

SINUS TACHYCARDIA

This is defined as sinus rhythm at a rate greater than 100/min (*Fig.* 1.5). Sinus tachycardia is caused by exercise, anxiety or any disorder that increases sympathetic nervous system activity. Occasionally it may be due to a primary disorder of the sinus node (sinus node re-entry).

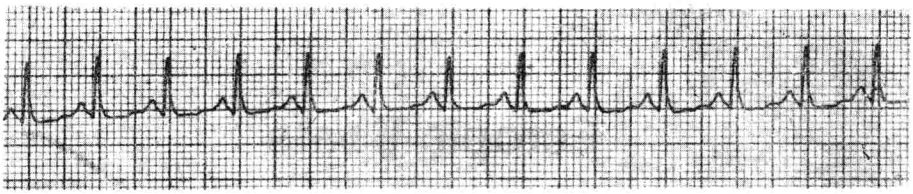

Fig. 1.5. Sinus tachycardia during exercise (lead II). The rate is 150/min.

At rest the sinus node rate is seldom above 120/min unless the patient is very ill. In contrast, atrial flutter with 2:1 AV block often leads to a heart rate of 140–160/min and can easily be mistaken for sinus tachycardia (*see* Chapter 6).

SINUS ARRHYTHMIA

Normally there are only minor changes in rate during sinus rhythm. In sinus arrhythmia there are alternating periods of slowing and increasing sinus node rate. Usually the rate increases during inspiration (*Fig.* 1.6). Sinus arrhythmia is most commonly seen in the young.

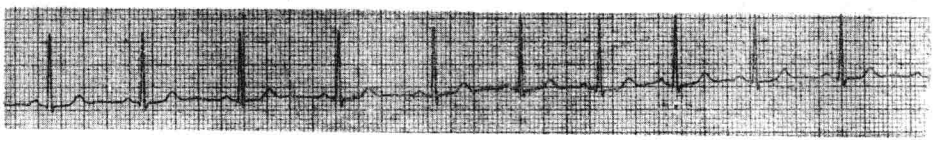

Fig. 1.6. Sinus arrhythmia.

Main Points

1. For sinus rhythm, an inferiorly directed P wave (i.e. upright in leads III and AVF) must precede each QRS complex.
2. If AV conduction is normal, the duration of the PR interval will be between 0·12 and 0·21 s.
3. Normal intraventricular conduction results in a QRS complex whose duration will be less than 0·08 s.
4. In cases of apparent sinus tachycardia at rest, atrial flutter should be excluded.

Chapter 2

Ectopic Beats

Ectopic beats may arise from the atria, AV junction (i.e. AV node plus bundle of His) or ventricles (*Figs.* 2.1, 2.2, 2.3). For practical purposes, the terms ectopic beat, extrasystole and premature contraction are interchangeable.

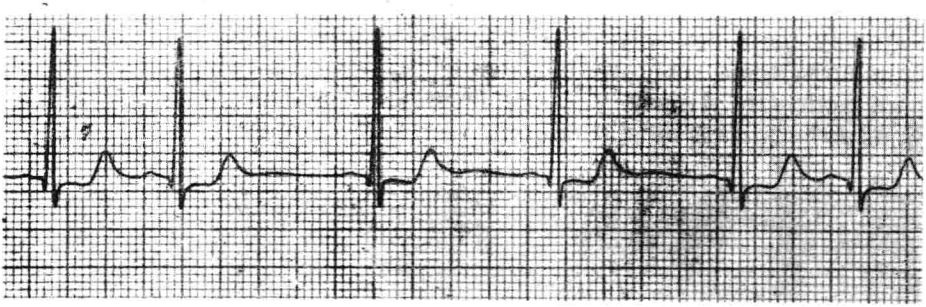

Fig. 2.1. Atrial ectopic beats (second and sixth beats). The ectopic P waves differ slightly in shape from those of sinus origin.

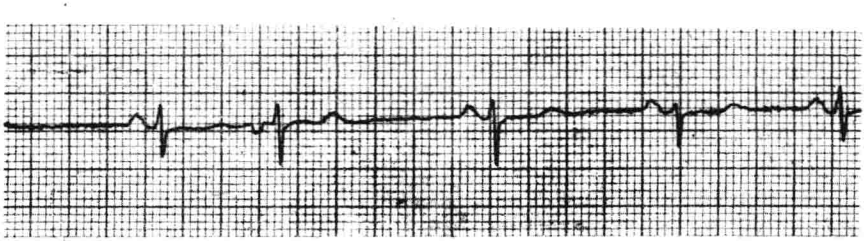

Fig. 2.2. The second beat is a junctional ectopic beat (lead III). The junctional focus has activated the atria as well as the ventricles, resulting in an inverted P wave which precedes the QRS complex.

Ectopic beats are premature. Thus, the interval between the ectopic beat and the preceding beat (i.e. the coupling interval) is always shorter than the cycle length of the dominant rhythm. This fact is often forgotten, with the result that other beats with abnormal configurations, i.e. escape beats (*see* Chapter 3) and intermittent bundle branch block (*see* Chapter 4)

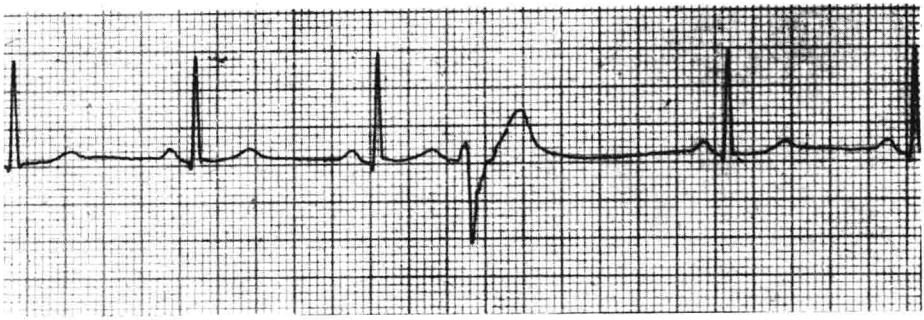

Fig. 2.3. The fourth beat is a ventricular ectopic beat.

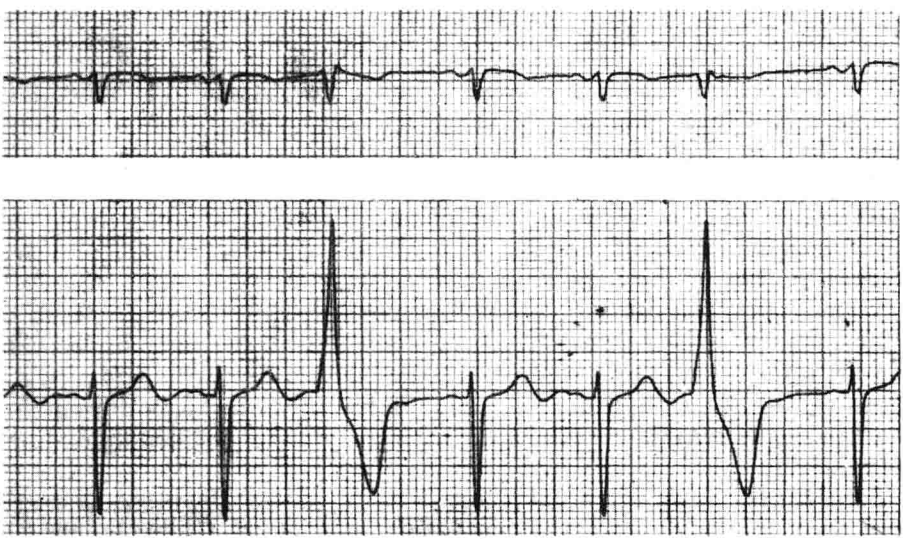

Fig. 2.4. Simultaneous recording of leads V1 and V2. The third and sixth beats are unifocal ventricular ectopic beats. Their ventricular origin is not apparent in lead V1 but is obvious in V2.

are misinterpreted as ectopic beats. Whereas suppression of ectopic beats may be desirable, attempts to suppress escape beats and beats with bundle branch block can be dangerous.

Usually, ectopic beats arising from the same focus have the same coupling interval and configuration (*Fig.* 2.4).

The site of origin of an ectopic beat can be ascertained from careful examination of the ECG. It cannot be stressed too strongly that a single rhythm strip does not always reveal the diagnostic clues and that scrutiny of simultaneous recordings of several ECG leads is often necessary (*Figs.* 2.4 and 2.5).

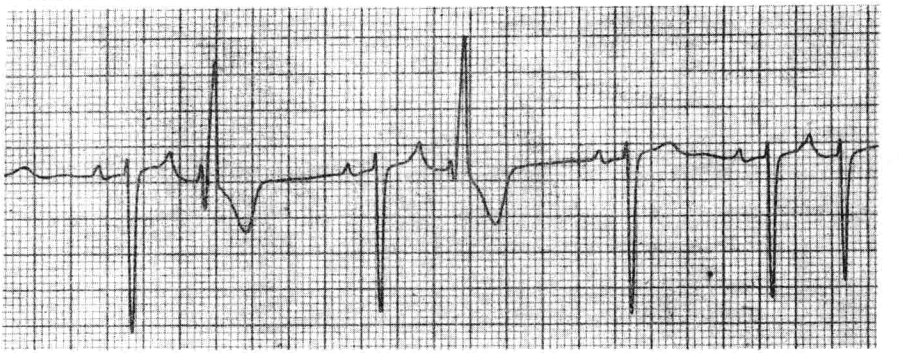

Fig. 2.5. Atrial ectopic beats are superimposed on the T waves of the first, third and sixth ventricular complexes (lead V1). It can be seen how the T waves of these beats are modified by comparing them with the T wave of the fifth ventricular complex which is not followed by an atrial ectopic. The first two atrial ectopic beats are aberrantly conducted, resulting in right bundle branch block.

ATRIAL ECTOPIC BEATS

These are recognized by a P wave which is premature and, because the source and hence direction of atrial activation differ from that during sinus rhythm, these P waves will often be of abnormal shape (*Fig.* 2.1). Ectopic P waves may be smaller than normal, and because they are premature they may be superimposed on the T wave of the preceding beat. Careful examination of the ECG is essential to detect ectopic P waves; frequently they are best shown in lead V1 (*Figs.* 2.5, 2.6).

Usually an atrial ectopic beat will be conducted to the ventricles in the same manner as if the atria had been activated by the sinus node. Thus, the PR interval and QRS complex of the ectopic beat will be identical to those during sinus rhythm (*Fig.* 2.1). If the QRS complex during sinus rhythm shows bundle branch block, then so will the QRS complex in 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 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 (*Figs.* 2.6–2.8). Blocked atrial ectopics have on occasion been erroneously taken as an indication for cardiac pacing! Partial or complete refractoriness of one or other bundle branch (it is usually the right bundle) will lead to correspondingly partial or complete bundle branch block (*Fig.* 2.7). This phenomenon of functional bundle branch block is referred to as 'phasic aberrant intraventricular conduction'. The practical significance of this phenomenon is that the resultant QRS complexes are broad and can therefore mimic ventricular ectopic beats.

Atrial ectopic beats are often benign. If they are frequent, however, they may clinically mimic atrial fibrillation, and may herald its onset. When

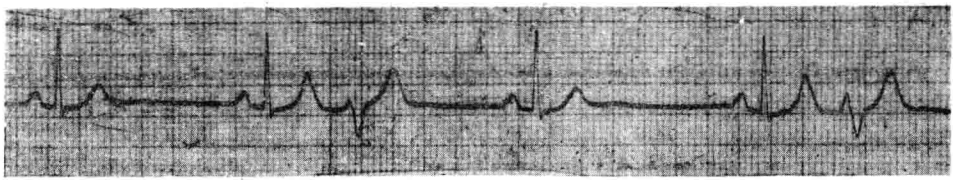

Fig. 2.6. The third and sixth beats are atrial ectopic beats. The premature P waves are superimposed on the preceding T wave, as can be seen by comparing the T waves of sinus beats preceding and not preceding ectopic beats. The ectopic beats show first degree AV block and phasic aberrant conduction.

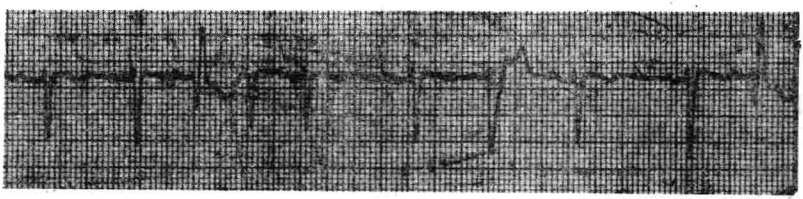

Fig. 2.7. Frequent atrial ectopic beats (lead V1). The seventh beat is an atrial ectopic beat conducted with left bundle branch block and marked prolongation of the PR interval. The third and tenth beats are atrial ectopic beats conducted with right bundle branch block and slight prolongation of the PR interval.

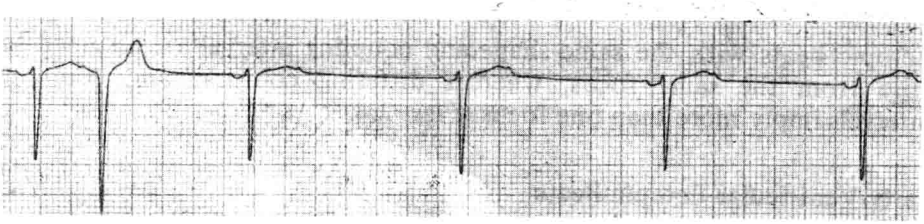

Fig. 2.8. Atrial ectopic beats are superimposed on the T wave of each ventricular complex. The first atrial ectopic is conducted with partial left bundle branch block. The other atrial ectopic beats are not conducted to the ventricles.

frequent atrial ectopic beats occur in patients with heart disease, especially valve disorders, myocardial infarction, cardiomyopathy or following cardiac surgery, treatment with digoxin should be considered so that the ventricular rate will be controlled should atrial fibrillation occur.

AV JUNCTIONAL ECTOPIC BEATS

AV junctional beats used to be referred to as 'nodal' beats. It is now appreciated that at least part of the AV node is not capable of pacemaker

activity and that it is not possible to distinguish between beats of AV nodal and His bundle origin. Hence the more general term 'AV junction'.

AV junctional ectopic beats are recognized by a premature QRS complex similar to that occurring in sinus rhythm. The atria as well as the ventricles may be activated by the junctional focus, leading to an inverted P wave (i.e. negative in leads II, III and AVF) which may precede, follow or be buried within the QRS complex, depending on the relative speeds of conduction from AV junction to ventricles and from AV junction to atria (*Fig.* 2.2).

AV junctional ectopic beats are not as common as atrial or ventricular ectopics. Treatment is rarely required.

VENTRICULAR ECTOPIC BEATS

These are recognized by a premature ventricular complex which is broad (usually >0·12 s), bizarre in shape and, in contrast to atrial ectopic beats, will clearly not be preceded by an ectopic P wave (*Figs.* 2.3, 2.4). The abnormal shape and prolonged duration of the ventricular complex reflect the abnormal course and consequent slowing of ventricular activation.

There are a number of different terms – important because they are of either therapeutic or diagnostic significance – used to describe ventricular ectopic beats.

Unifocal or Multifocal

Ectopic beats with the same shape and coupling interval are assumed to arise from the same focus, whereas differing contours and coupling intervals imply more than one focus (*Figs.* 2.4, 2.9).

Early Ventricular Ectopic Beats

Ectopic beats which 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' (*Fig.* 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).

Late Ventricular Ectopic Beats

A ventricular ectopic beat which occurs late in the cardiac cycle may fall, by chance, immediately after a P wave initiated by sinus node activity (the P wave will therefore be normal in timing and configuration). This is referred to as an 'end-diastolic' ventricular ectopic beat (*Fig.* 2.11). This situation, in which the atrial impulse will clearly not be conducted to the ventricles, must not be confused with an ectopic atrial beat with aberrant conduction, in which case, of course, the P wave will be premature.

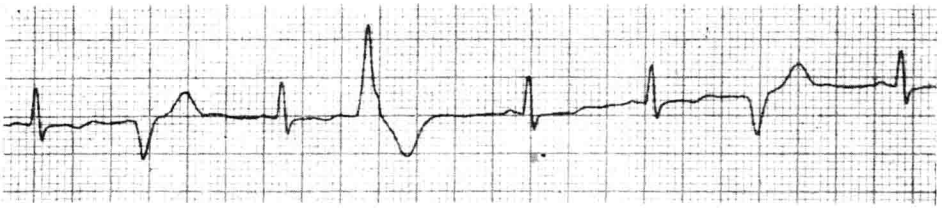

Fig. 2.9. Multifocal ventricular ectopic beats. The second ventricular ectopic beat has a different shape and coupling interval from the first and third ectopic beats.

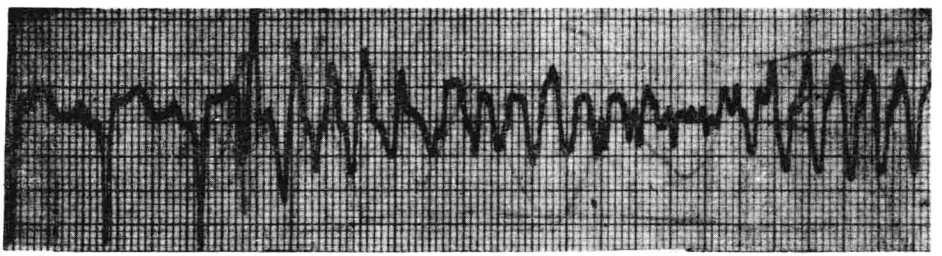

Fig. 2.10. An 'R on T' ventricular ectopic beat initiates ventricular fibrillation.

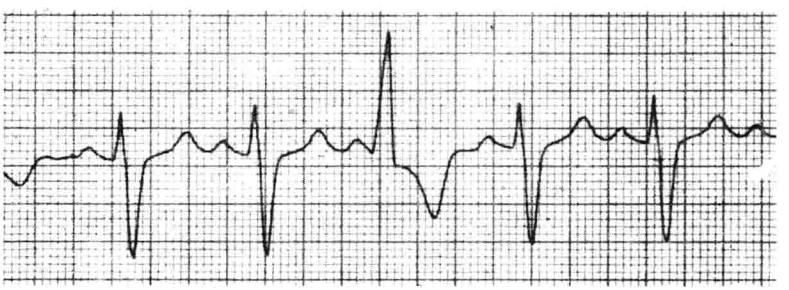

Fig. 2.11 The third beat is an end-diastolic ventricular ectopic beat. It is preceded by a *normally* timed P wave.

Because the initial upstroke of a ventricular ectopic beat may be slurred like a delta wave (*see* Chapter 7), end-diastolic ventricular ectopic beats can mimic the Wolff–Parkinson–White syndrome (*Fig.* 2.12).

Interpolated Ventricular Ectopic Beats

Usually there is a pause after a ventricular ectopic beat before the next 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' (*Fig.* 2.13).

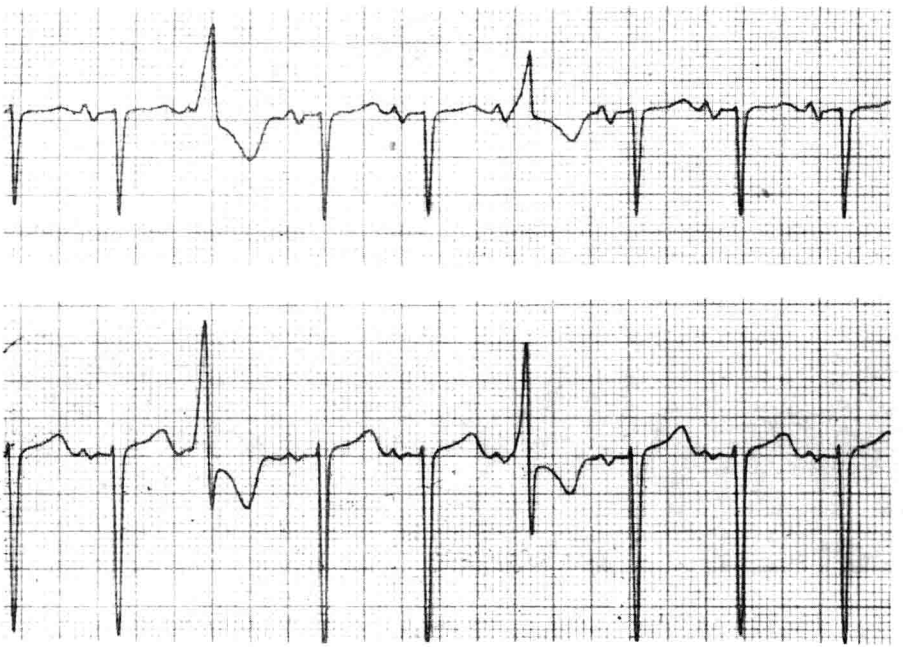

Fig. 2.12. Simultaneous recording of leads V1 and V2. Two end-diastolic ventricular ectopic beats. The second, mimicking the Wolff–Parkinson–White syndrome.

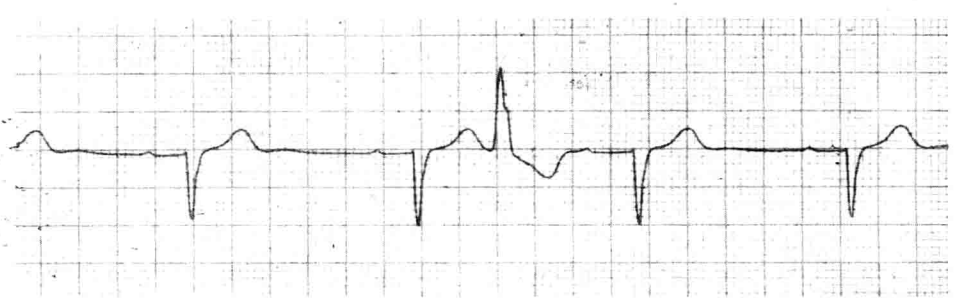

Fig. 2.13. Interpolated ventricular beat. The subsequent PR interval is prolonged due to retrograde concealed conduction.

Frequency

Ventricular ectopic beats are usually quantified in terms of the number occurring per minute.

When an ectopic beat follows each sinus beat the term bigeminy is applied (*Fig.* 2.14). If an ectopic follows a pair of normal beats, there is trigeminy. When two ectopics occur in succession (*Fig.* 2.15) they are referred to as a couplet, and when there are more than two ectopic beats in succession the group is termed a salvo or ventricular tachycardia (*see* Chapter 5).