ECG Diagnosis in Clinical Practice

Second Edition

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This book is dedicated to my teachers who
have stimulated me to understand.

Romeo Vecht

Borné dans sa nature, infini dans ses voeux
L'homme est un Dieu tombé, qui se souvient des cieux

Lamartine, 19th Century

Augustus Desire Waller (1856–1922) Professor of Physiology St Mary's Hospital London.

The picture from the 1880s shows his dog Jimmy and the Lippman Capillary Electrometer. Waller recognised an electrical signal preceding the heart contraction of his dog and thereby initiated the science of electrocardiography.

Foreword to the First Edition

Electrical signals associated with the contraction of muscles were identified in the middle of the nineteenth century. It was probably Muirhead of St. Bartholemew's Hospital, London, who was the first to record the electrical signals from the human heart. Waller from St. Mary's Hospital and later the National Heart Hospital in London published records in 1888 of the electrical potentials recorded from humans. But it was the development of the string galvanometer by Willem Einthoven that is widely regarded as the beginnings of the clinical application of the electrocardiogram (ECG). The key publication was in 1904 and Einthoven received the Nobel Prize in 1924. He probably did not imagine that his discovery was to remain critical to clinical cardiology for an entire century and probably many more years. Acute myocardial infarction was first described only in 1910. Over the last century, the ECG has been used by clinicians to make major clinical decisions with regard to electric pacing, the use of thrombolytic drugs in acute myocardial infarction and the timing of surgery. In conjunction with a chest X-ray and the echocardiogram it is a fundamental part of the initial investigation of a patient with suspected heart disease.

These electrical squiggles have always been difficult for students to understand. In part the problem has been that the formatting of the ECG has become standard only in the last two decades . Some important books have not provided the full twelve-lead ECG. On occasion the interpretation of the ECG has been related to complex explanations of the shapes of the electrical signals. For the practising physician much of the interpretation is a matter of pattern recognition.

This CD and the book it accompanies have two great advantages. Firstly they are written by a single author who has had wide experience in cardiology over many years. Secondly they contain an outstanding collection of traces which are easy to inspect in detail. *ECG Diagnosis Made Easy* should be extremely helpful to students, particularly those who wish to pursue a career in cardiovascular medicine.

London
2004

Philip A. Poole-Wilson

Foreword to the Second Edition

Medicine has made great advances in the last few decades and no branch has remained untouched by science, especially biology. In cardiovascular medicine, one investigation that has been tested over time and not found wanting is the electrocardiogram. It remains an essential tool in clinical practice for the diagnosis of cardiovascular disease.

This book was first published in 2004. The second edition brings with it several changes and quite rightly so. One is the change of the title of the book, to emphasize the importance of the electrocardiogram in clinical practice rather than the inspection of the electrocardiogram and an abstract analysis of wiggly lines on a strip of paper. The second change is more important in that more electrocardiograms have been added and these are more difficult to interpret correctly. The electrocardiogram in paediatric cardiology is a new section and there is a chapter on electrophysiology. These latter additions reflect the advances in the last few years. Electrical and mechanical engineering have allowed physicians to devise totally new treatments for common conditions such as atrial fibrillation and heart failure in the form of pathway ablation and cardiac resynchronisation therapy (CRT). The increasing availability of internal cardioverter defibrillators (ICDs) means that the correct interpretation of the electrocardiogram to diagnose the different forms of tachycardia becomes of greater clinical relevance.

The book has been written and put together by a clinician with a long standing interest in, and indeed love of, interpreting the electrocardiogram. The book retains its simplicity and high quality images which will be helpful to students but now will also be a valuable source of information for the more experienced cardiovascular physician.

London
2008

Philip A. Poole-Wilson

Preface to the First Edition

This book is intended primarily for those who want to acquire an understanding of electrocardiography. Therefore I have attempted to keep it simple, and explain the basic concept of the electrocardiogram; only a few references have been included to tempt further reading.

The illustrations were chosen to cover a wide spectrum of ECG pathologies, with an emphasis on changes observed in real tracings. I have added short historical notes where appropriate.

Electrocardiography is the ability to recognise electric patterns based on sound scientific principles. It has vast applications in the fields of cardiology, cardiac surgery and general medicine. The electrocardiogram must be seen as a support to clinical diagnostic skill and not as a primary decision-making instrument.

I have enjoyed producing this book, based on the belief that the art of teaching should be as pleasurable as the practice of medicine. I hope that my efforts will prove beneficial and stimulating, and as enjoyable to read as they were to compile.

London R.J. Vecht
2004

Preface to the Second Edition

It is a privilege to have been afforded the opportunity to extend this book to a second edition. The original text has been corrected and upgraded to conform with ongoing progress in the field of electrocardiography, while additional selected references have been introduced to encourage further interest. Also increased is the number of original ECG traces, the last 50 being introduced for the benefit of the more discerning readers.

There are two new chapters, one on paediatric electrocardiography and the other on electrophysiology (EPS), to achieve a more comprehensive overall understanding of the subject.

I am grateful to J. O'Mahony for her exemplary efforts at typing the manuscript and particularly her ability to decipher my handwritten notes. I express my gratitude to Grant Weston of Springer for support and advice, without which this book would not have come to fruition.

London R.J. Vecht
2008

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Chapter 1
Basic Principles

ELECTRICAL IMPULSES IN THE HEART

Electrical impulses are required to synchronize the four pumping chambers. The atria are electrically isolated from the ventricles by a fibrous atrioventricular separation. The impulse originates in the sino-atrial (SA) node, travels across the atrial musculature towards the atrioventricular (AV) node, thence down through the bundle of His to the ventricles via the right and left bundles and into the Purkinje system (Fig. 1.1). The SA node has an inherent rate of approximately 70 bpm and is under autonomic and chemical hormonal influence. The inherent rate of the AV node is lower, at 60 bpm, and the ventricles beat in isolation at approximately 40 or less beats per minute. The electrical impulse, having reached the ventricular musculature, then travels outwards from the endocardial to the epicardial surface. The electrical current is produced by a change of ionic forces from positive at rest to negative when activated (Fig. 1.2).

POSITIONING ELECTRODES FOR ELECTROCARDIOGRAPHY

The electrocardiogram consists of 12 electrodes placed around the heart. For mathematical purposes, the heart is at the center of a triangle (Fig. 1.3).

The electrode placements are designated as follows:

- *The three limb leads:* lead I joins the right and left arms, lead II connects the right arm and left leg and lead III joins the left arm and left leg.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
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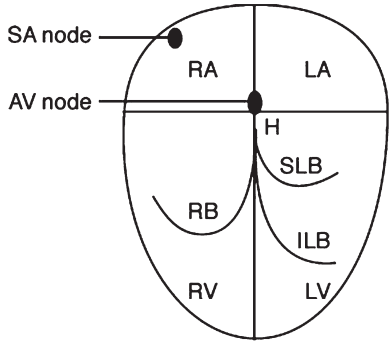

FIG. 1.1. Electrical conduction through the heart: From the sinoatrial node across the atrial musculature, the impulse reaches the atrioventricular node. It proceeds downwards through the bundle of His, then simultaneously to the right ventricle, through the right bundle and the left ventricle through the two left bundles (termed anterior and posterior; or superior and inferior). Finally from endocardium to epicardium, the Pukinje system conducts the tail-end impulses, *SA* sinoatrial node; *AV* atrioventrical node; *H* bundle of His; *RV* right ventricle; *LV* left ventricle; *RB* right bundle; *RA* right atrium; *LA* left atrium; *SLB* superior left bundle; *ILB* inferior left bundle.

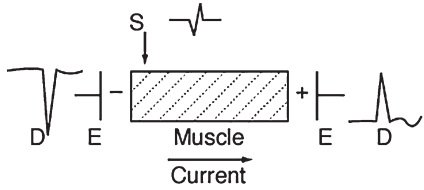

FIG. 1.2. Muscle depolarization. When stimulated, the muscle develops a negative charge. An electrode facing the oncoming current will record an upright (positive) deflection. The current moving away inscribes a downward (negative) signal. Halfway between the two, the deflection is biphasic. *D* deflection; *E* electrode; - negative; + positive; *S* stimulus.

- *The three augmented leads:* aVL is positioned facing the heart from the right arm VL from the left arm and aVF from the left foot. These electrodes are placed in a frontal plane.
- *The precordial leads (V1–V6):* these are placed on the front of the thorax and record horizontal impulses.

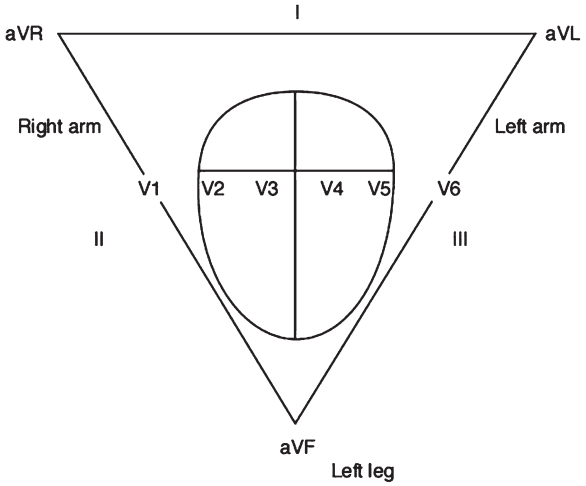

FIG. 1.3. Schematic representation of 12 leads (electrodes) placed around the heart.

TOPOGRAPHY OF IMPULSES

The conventional nomenclature is illustrated in Fig. 1.4.

P can be positive or negative, Q is always negative, R is always positive, S is always negative and T can be either. U wave is upright; when inverted it implicates ischemia.

Physiological Measurements

- PR interval = 0.12–0.2 s (120–200 ms)
- QRS duration = 0.06–0.1 s (60–100 ms)
- QT interval = 0.30–0.46 s (300–460 ms)

For heart rates varying between 45 and 115 bpm.

The QT interval lengthens with bradycardia and shortens with tachycardia.

The Atria

The SA node discharges from right to left. This is recorded as the P wave, which represents atrial depolarization leading to atrial contraction (Fig. 1.5). The repolarization of the atria is lost in the QRS complex. The P wave is inscribed as positive in leads facing the incoming signal (aVF, III and V6) and as negative in leads from which the current is moving away (aVR and V1).

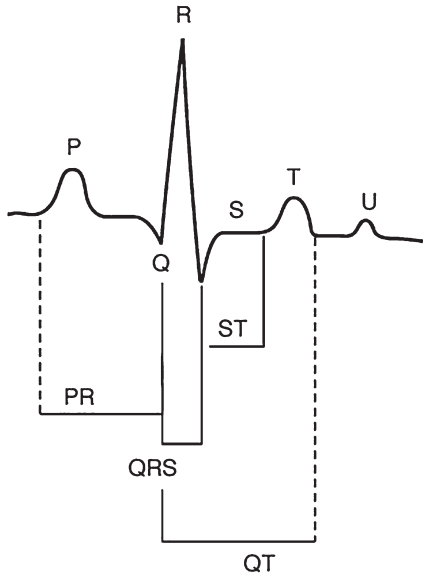

FIG. 1.4. Normal ECG recording. This denotation was introduced by Einthoven. The U wave is often not discernible; *P* atrial depolarization. *QRS* ventricular depolarization; *T* ventricular repolarization; *U* either represents after potentials of the ventricular myocardium or repolarization of the Purkinje fibers. The PR interval represents the time taken from atrial to ventricular depolarization. The ST segment should be isoelectric. The QT interval is the time taken from ventricular depolarization to repolarization.

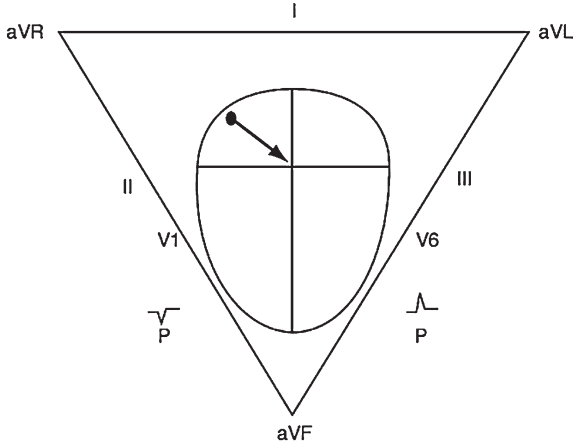

FIG. 1.5. The P wave vector: The P wave impulse travels from right to left. Leads facing the incoming signal (III, avf and V6) will record a positive trace. Negative deflections appear in avr and V1, the impulse being carried away from these sites.

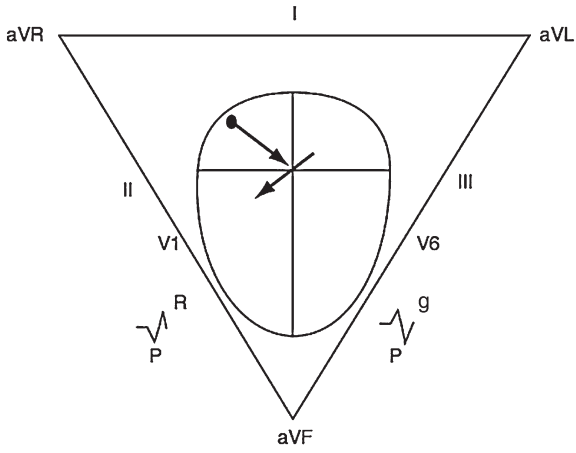

FIG. 1.6. Septal activation. The initial QRS activation results from the electrical impulse stimulating the interventricular septum through the bundle of His. The initial deflection will be positive in lead V1 and negative in lead V6 (i.e., R and Q).

The Ventricles

The QRS complex represents ventricular contraction, i.e., depolarization. The\complex consists of an *initial* septal activation followed by the major ventricular signal. Septal activation is from left to right, so that the leads facing the oncoming signal (II, aVF and V1) will record a positive wave R; lead V6 will record a negative trace Q (Fig. 1.6).

The *main* ventricular activation affecting the left ventricle moves from right to left (Fig. 1.7). This comprises all the signals activating the left ventricle (the right ventricular currents are dwarfed by the left). Again, electrodes facing the oncoming current (e.g., leads III and V6) will record a positive wave, and those carrying the impulse away (e.g., V1) will record a negative wave.

Repolarization of the ventricle gives rise to the T wave, which usually follows the QRS complex.

THE ELECTRICAL AXIS

Each ECG lead has a positive and a negative terminal. The three standard limb leads, I, II and III, are illustrated in Fig. 1.8.

The augmented leads are aVR, aVL and aVF. Here each limb lead has a positive terminal, the negative pole being connected to all three limb electrodes. The sum of the three limb leads equals zero potential, so the augmented leads have a positive terminal and a negative terminal at zero potential.

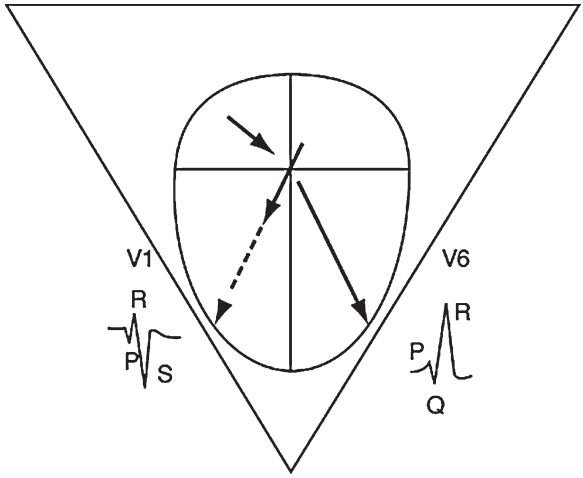

FIG. 1.7. Ventricular activation. The main vector (an electrical force that has both magnitude and direction) travels from right to left (right ventricular currents are dwarfed by the thicker left ventricular musculature). Positive tracing are observed in leads III and V6 (R) and a negative tracing is recorded from lead VI.

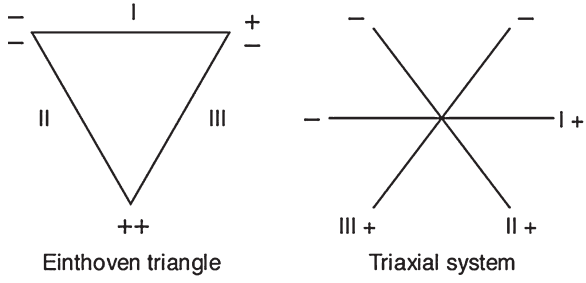

FIG. 1.8. The three standard (limb) leads. The Einthoven triangle translated into a triaxial system shows the positive and negative terminals of each lead.

Using the hexaxial system (Fig. 1.9), one is able to calculate the mean P, QRS or T wave axis.

The mean frontal QRS electrical axis is the one to concentrate on. The depolarization of the ventricles (QRS) can be represented by a mean vector running from right to left (the RV vector is masked). The maximum deflection in an ECG lead represents a force running parallel to this lead. Thus, considering an example

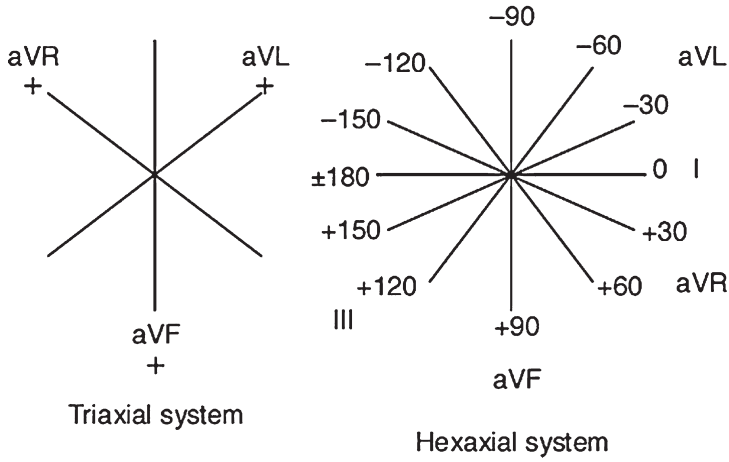

FIG. 1.9. Standard and augmented leads. Combination of all 12 leads showing polarity and degrees within a circle.

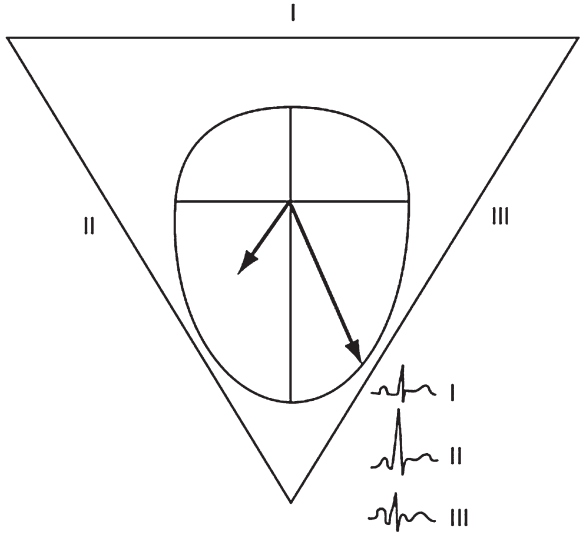

FIG. 1.10. The mean vector of left ventricular depolarization. Lead II is the closest parallel to the mean vector of depolarization. It shows the greatest deflection and an axis of +60°.

in which lead II shows a maximum positive deflection, the axis will be +60° (Figs. 1.10 and 1.11).

To keep things even simpler: if the vectors in leads I and III move away from one another, this represents left axis deviation

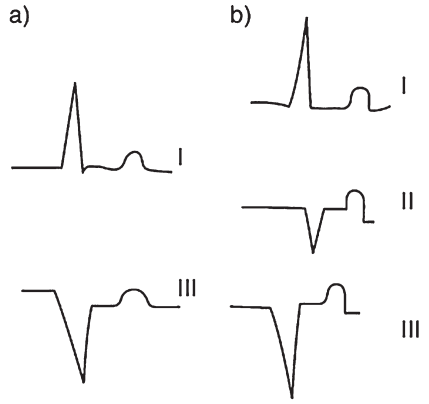

FIG. 1.11. Left (superior) electrical axis deviation. (**a**) Left (superior) axis deviation is present when the main vectors in leads I and III move away from each other. (**b**) When lead II is also negative (–30°), the trace is described as showing a *pathological* left axis deviation and represents left ventricular problems.

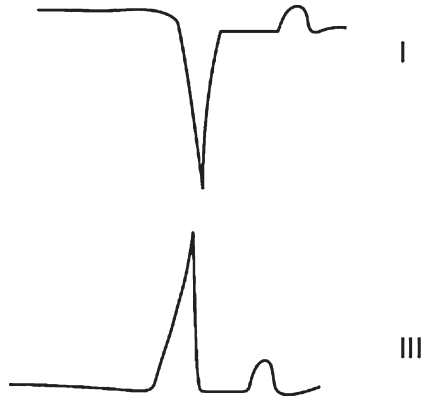

FIG. 1.12. Right (inferior) electrical axis deviation. The main deflections in leads I and III point towards one another. This indicates right-sided problems.

(LAD). If, in addition, lead II is negative and the axis points to –30° or more, the trace is described as showing pathological left axis deviation. Left axis deviation indicates an abnormality of the left ventricle caused by numerous pathological entities.

If the vectors in leads I and III point towards one another, it is known as right axis deviation (RAD) and denotes right-sided problems (Fig. 1.12).

LAD is present when the axis moves from 0 to –120° (i.e., it is superior) and RAD is present when the range is from +90 to +180 (i.e., it is inferior). NB: LAD is also known as left anterior hemiblock, and RAD as left posterior hemiblock)

HISTORICAL NOTES

A Von Koellitzer (1817–1905) Swiss physiologist. First demonstrated muscular contraction associated with an electrical current.

AD Waller (1856–1922) Physiologist, St Mary's Hospital, London, UK. Demonstrated electrical activity preceding cardiac contraction.

W. Einthoven (1860–1927) Physiologist, Leiden, Netherlands.

Introduced P, QRS, T nomenclature and string galvanometer.

W His (1863–1934) Professor of Medicine, Basel. Switzerland.

Demonstrated the bundle named after him

JE Purkinje (1787–1869) Professor of Physiology, Prague, Czechoslovakia.

Discovered fiber formation beneath mucous membrane of the heart without recognizing physiological significance.

KEY MESSAGES

- – electrical impulses traveling towards an electrode are inscribed upwards (positive) and away from an electrode downwards (negative).

Chapter 2
Ischaemic (Coronary) Heart Disease

Atheromatous narrowing of the coronary arteries is a frequent pathological finding in the developed world. Stenosis and/or occlusion of a coronary artery leads to ischemia and/or infarction of myocardial tissue with characteristic ECG changes.

Non ST elevation myocardial infarction (NSTEMI) refers to ECG findings due to a partially occluded epicardial vessel.

ST elevation myocardial infarction (STEMI) indicates total occlusion of an epicardial coronary artery. Q-wave infarction signifies transmural or full thickness damage.

NON Q WAVE INFARCTION (SUBENDOCARDIAL)

When the infarct spares some muscle, a non Q wave infarction results. As blood flows from the epicardium to the endocardium, the latter is more vulnerable to ischemia, being subjected to greater contractile forces. Ischaemic muscle produces a current of injury; healthy muscles have a positive charge which turns negative when stimulated. The baseline of the electrocardiogram thereby becomes depressed. During depolarization, when the healthy muscle becomes negatively charged, no current flows. As the electrical signal returns to baseline, this leads to elevation of the ST segment in the electrodes facing the injured muscle. Thus a non Q wave infarct (also known as a subendocaridal infarct) is characterized by ST elevation in the leads facing the damage.

Conversely, ST depression is seen in leads facing the uninjured surface (Fig. 2.1). Repolarization is abnormal and gives rise to inverted T waves.

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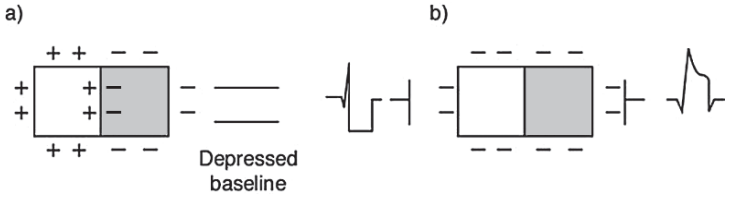


FIG. 2.1. Current of injury. (a) The shaded area is ischaemic and becomes negatively charged. The ECG shows a depression of the baseline. (b) During depolarization the residual healthy muscle becomes negatively charged. No current flows. The baseline returns to normal and the ST segment appears elevated. The lead facing the injured muscle shows ST segment elevation. The lead facing the uninjured portion inscribes ST segment depression.

Q WAVE INFARCTION (TRANSMURAL, FULL THICKNESS)

Q waves are seen when the entire wall of the ventricle is infarcted (a full thickness or transmural infarction). Three surfaces of the heart can be damaged: anterior, inferior (diaphragmatic) or true posterior (Fig. 2.2). Infarcted tissue carries no electrical charge. The ECG electrode picks up electrical impulses traveling towards it through a "dead window."

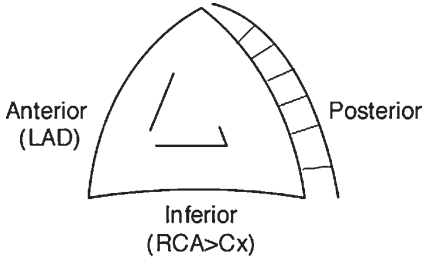


FIG. 2.2. The three areas of myocardial infarction. The anterior surface of the myocardium is supplied by the left anterior descending (LAD) artery. The inferior surface obtains its supply essentially from the right coronary artery (RCA) and occasionally from the circumflex (Cx) artery.

Anterior Myocardial Infarction

Leads facing the infarct record negative vectors (Q waves). As the main left ventricular vector that moves from right to left has been obliterated, negative vectors are seen, inscribing Q waves on the ECG. These negative vectors result from "unopposed" forces, that is septal and right ventricular depolarization, seen through the "dead window" (Fig. 2.2). Q waves are seen in leads I and aVL and V1–V6, depending on the extent of the damage. Q waves in leads V1–V3 indicate an anteroseptal infarct; in leads V3–V6 they indicate an anterolateral infarct, and in leads V1–V6 they signal extensive myocardial infarction (Figs. 2.2–2.33). ST elevation follows the same distribution and localization.

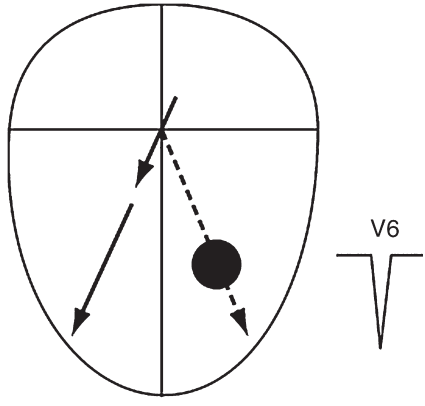


FIG. 2.3. Anterior infarction. Leads facing the infarcted territory pick up unopposed forces moving away through the nonconducting "window," e.g., Q waves in V6.

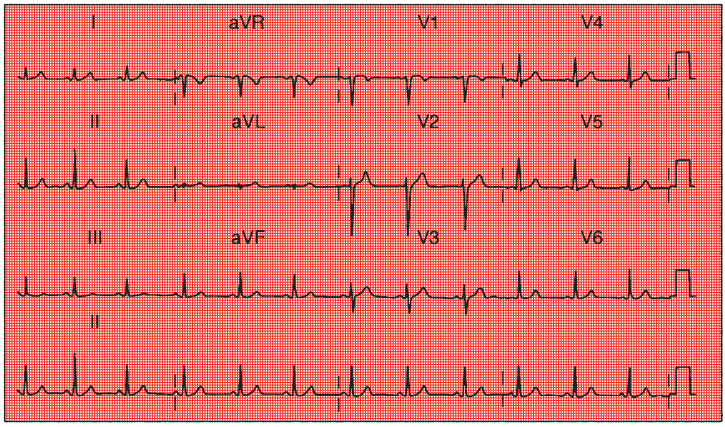


FIG. 2.4. Normal trace (LG; 24/4/98).

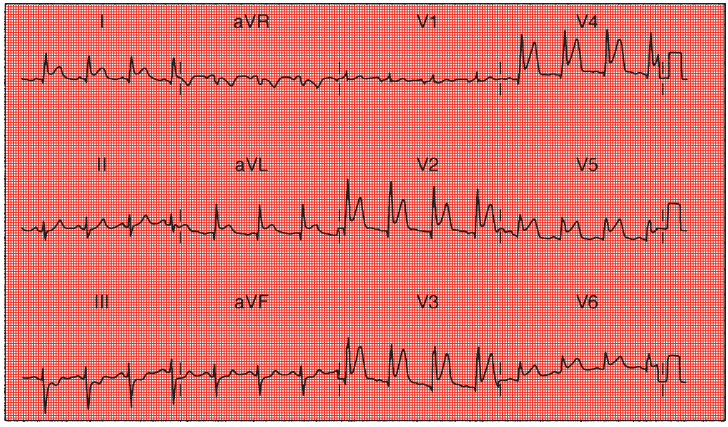


FIG. 2.5. Acute anterior infarction. ST elevation in leads I, aVL and V2–V6 (AN; 15/9/85).

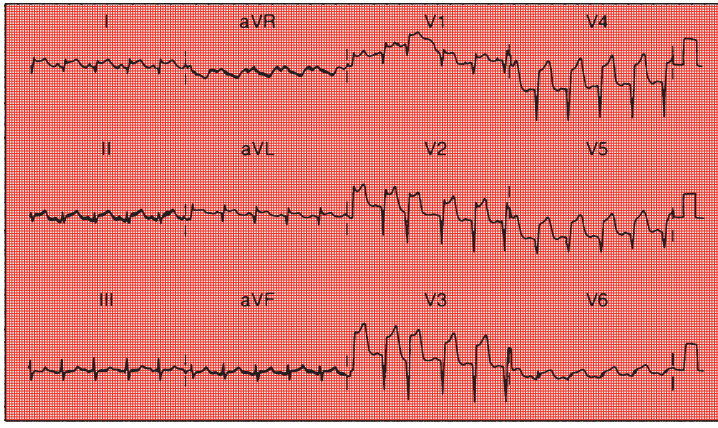


FIG. 2.6. The same patient 3 days later. Q waves are apparent in leads I, aVL and V1–V5. ST elevation is still present (AN; 18/9/85).

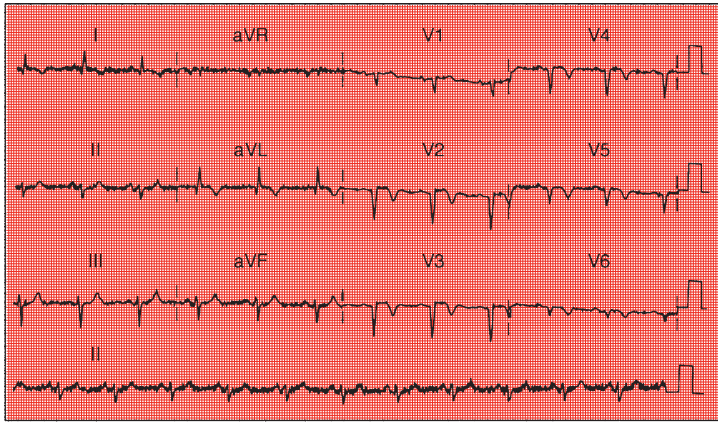


FIG. 2.7. Two years later the ST segments are back to baseline but Q waves are seen in leads I, aVL and V1–V6 with T wave inversions (AN; 6/2/87).

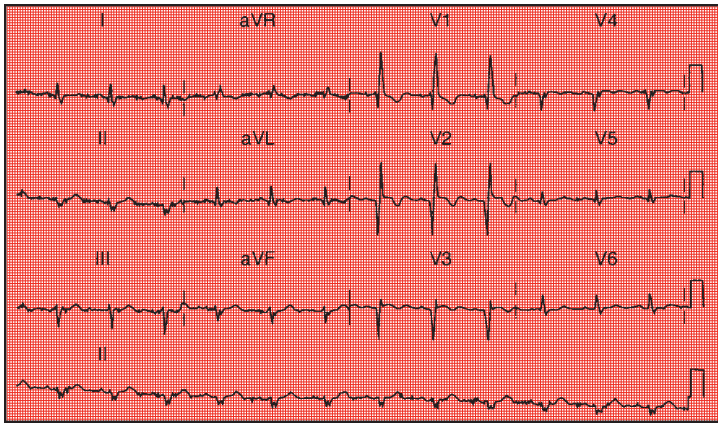


FIG. 2.8. Eleven years later the patient has developed right bundle branch block. The anteroseptal Q waves remain. Residual ST elevation in leads V2 and V3 are indicative of left ventricular aneurysm (AN; 25/11/98).

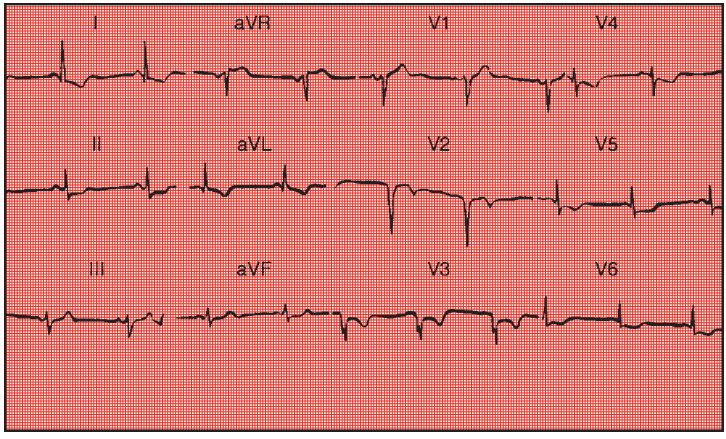


FIG. 2.9. Q wave anteroseptal infarct with widespread ST depression. The latter indicates widespread ischaemic territory (MB; 3/10/79).

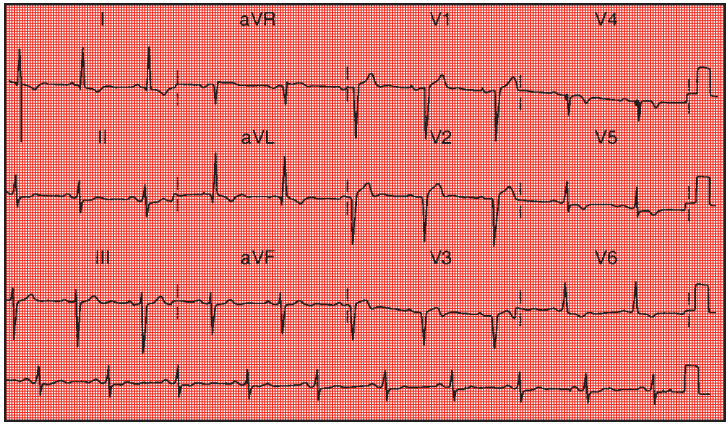


FIG. 2.10. The same patient 19 years later. The ECG has a very similar pattern, with fewer ST segment depressions. The patient was treated conservatively (MB; 29/6/98).

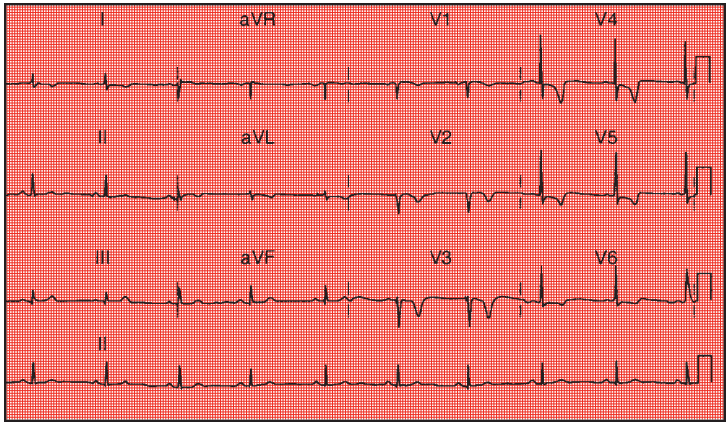


FIG. 2.11. Young male patient with acute anteroseptal infarction (non ST elevation infarction) (CA; 4/6/98).

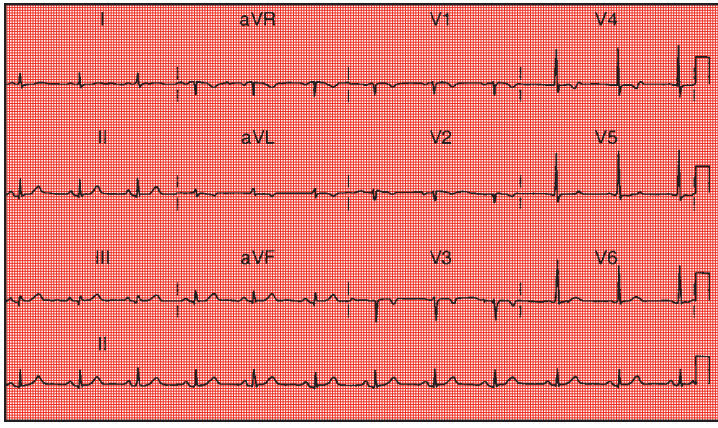


FIG. 2.12. This trace was recorded soon after successful angioplasty and stent insertion in LAD (CA; 18/6/98).

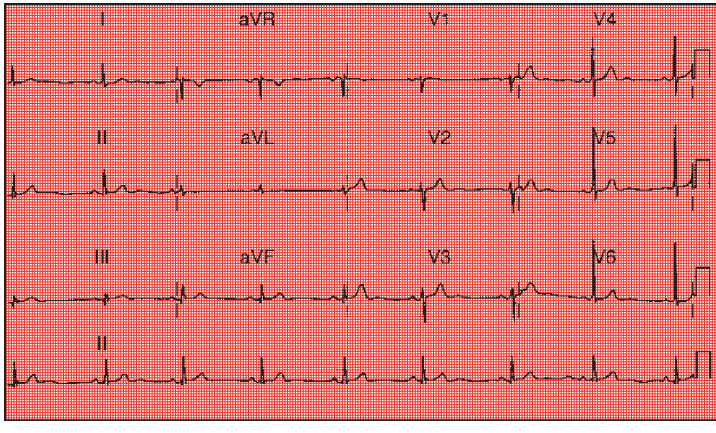


FIG. 2.13. After 3 months, the patient had made a full recovery (CA; 28/9/98).

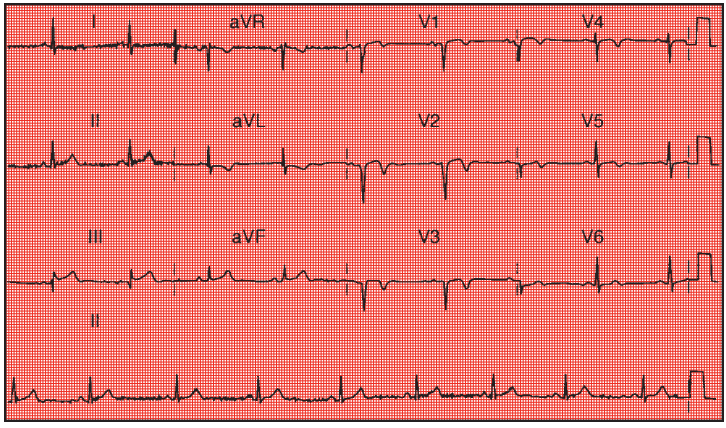


FIG. 2.14. Young male patient with anteroseptal Q wave infarction. He underwent a successful bypass operation (MG; 15/10/93).

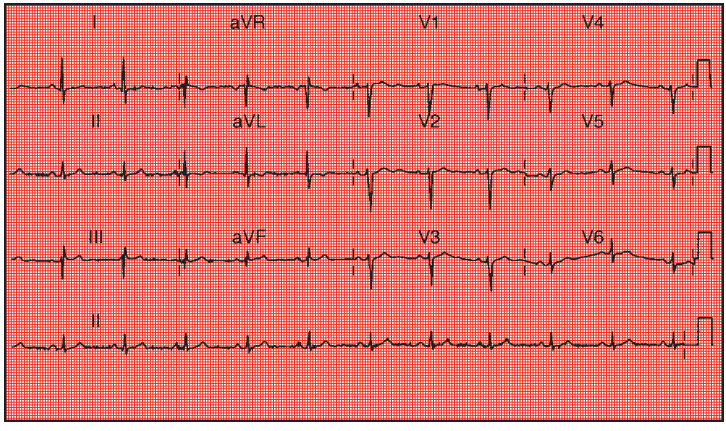


FIG. 2.15. After 6 years the only remaining abnormality is mild T-wave inversion in lead aVL. R wave progression over the praecordium has not fully recovered (MG; 22/9/99).

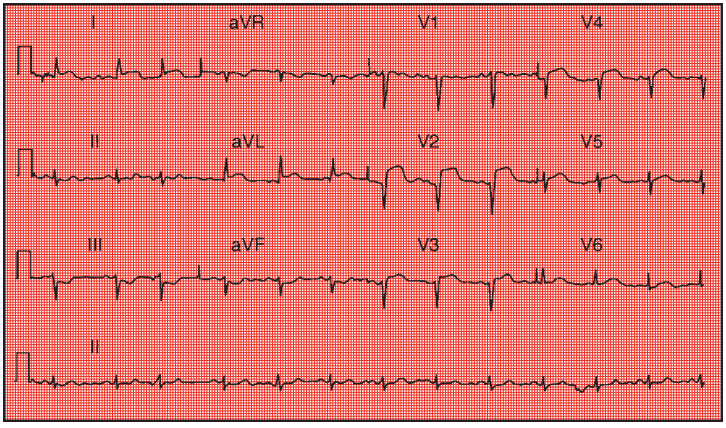


FIG. 2.16. Widespread Q wave acute anterior infarction. ST segment elevation is present in leads I, aVL and V2–V5 with Q waves in lead V2 and reciprocal changes (ST segment depression) in leads III and aVF (ST elevation infarction) (TB; 30/7/99).

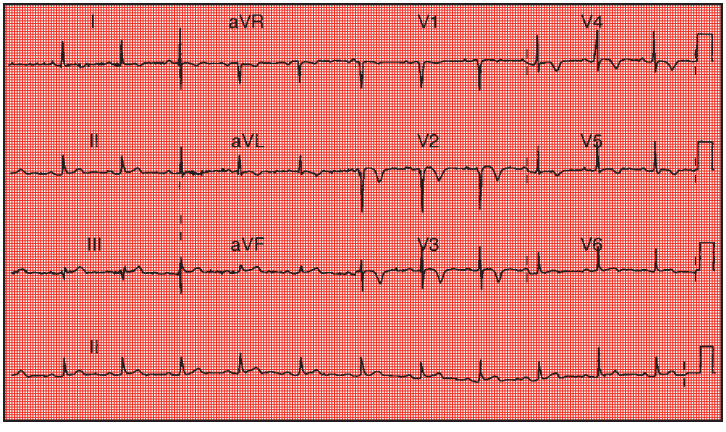


FIG. 2.17. This 43-year-old male suffered severe chest pain after cycling. He had an anteroseptal infarct. The LAD artery was successfully ballooned and stented (AF; 12/4/99).

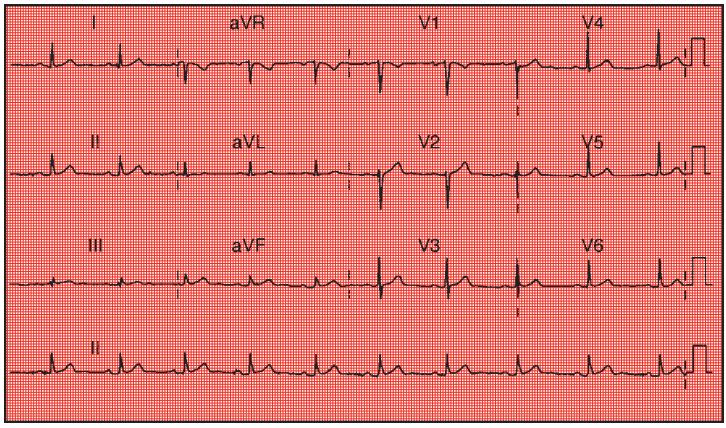


FIG. 2.18. The same patient was asymptomatic 5 months later. The patient made a full recovery (AF; 21/9/99).

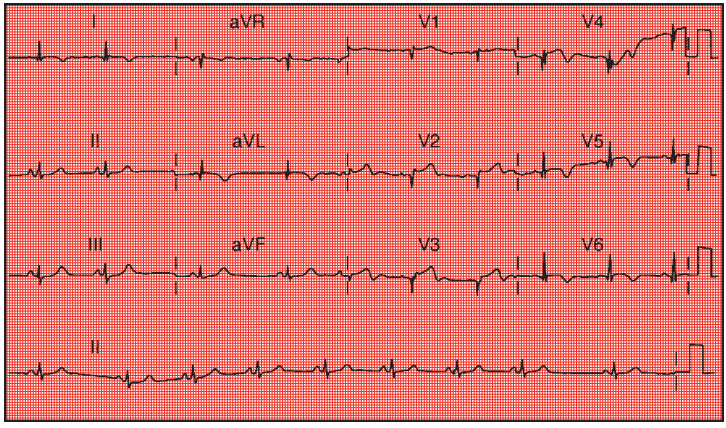


FIG. 2.19. Acute anteroseptal infarction (AR; 2/10/95).

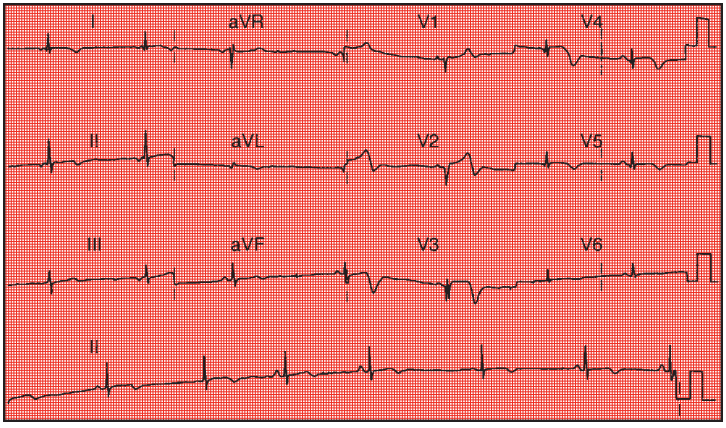


Fig. 2.20. More profound ischaemic changes are visible in leads V2 and V3, T wave inversions. These changes occurred 24 h later, suggesting extension of the infarct (AR; 3/10/95).

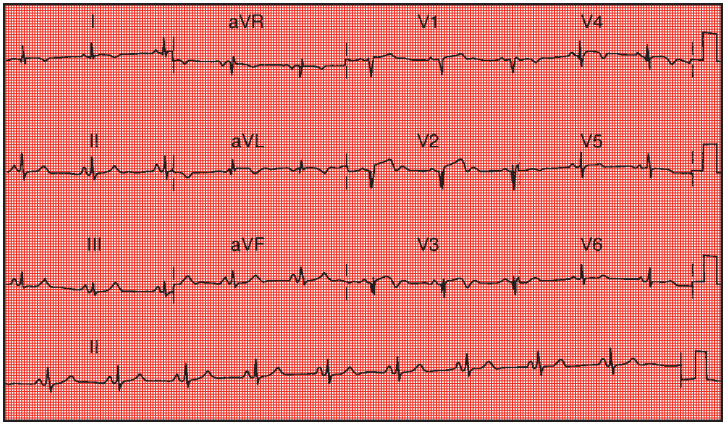


Fig. 2.21. There are fewer T wave inversions – a sign of recovery – 2 days later (AR; 5/10/95).

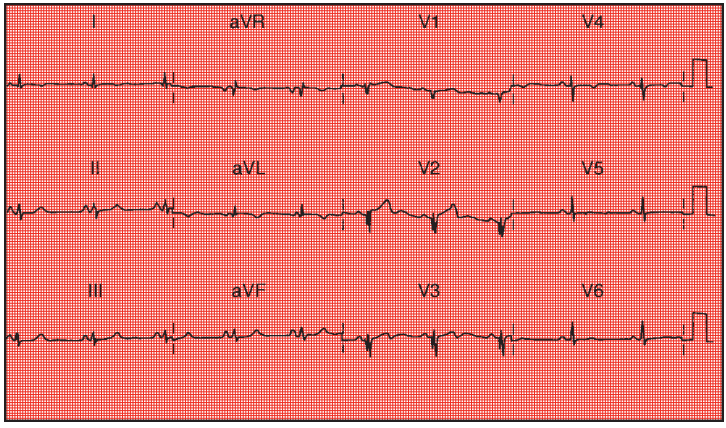


FIG. 2.22. Further improvement is evident 6 days after infarction (AR; 8/10/95).

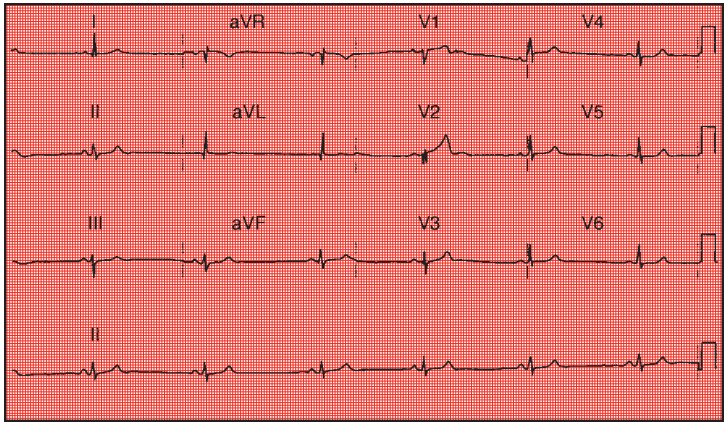


FIG. 2.23. Three years later, residual Q waves are visible in lead V2. ST segment elevation is present, possibly indicating a localized LV aneurysm. The patient underwent angioplasty to the left anterior descending artery soon after admission (AR; 7/10/98).

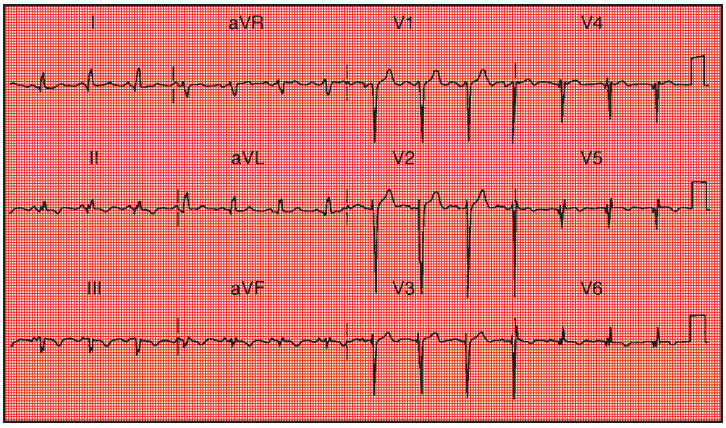


FIG. 2.24. Pre-intervention ECG. There is reduced R wave progression in leads V4–V5 (Mr. P; 22/5/98).

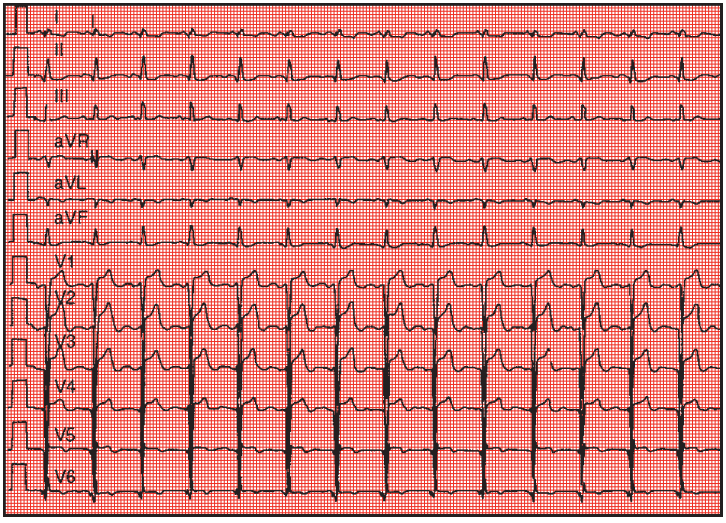


FIG. 2.25. During stent implantation. Marked ST segment elevation in leads V1–V5 during occlusion of the left anterior descending artery. The patient made a full recovery (Mr. P; 22/5/98).

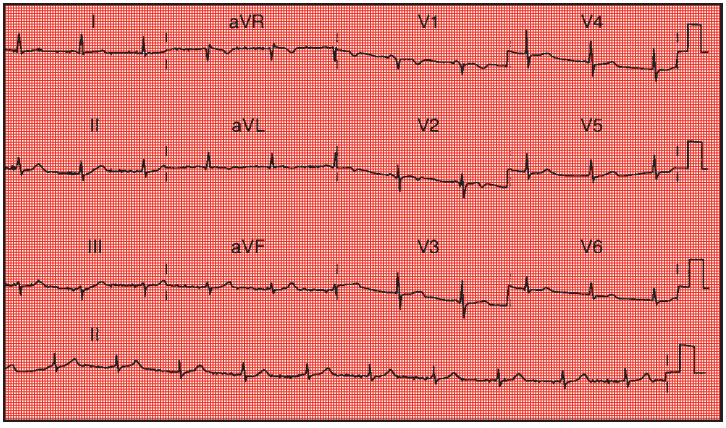


FIG. 2.26. Patient with previous bypass surgery. Minor T wave changes are present in leads aVL and V2 (LG; 5/5/98).

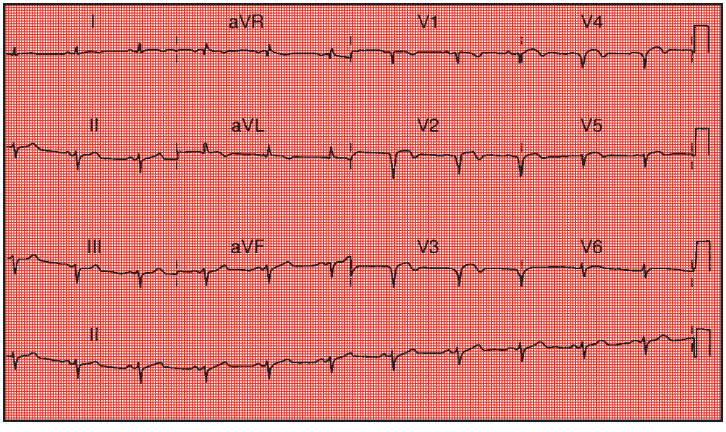


FIG. 2.27. Acute anterior infarction caused by an occluded LAD vein graft was demonstrated at cardiac catheterization 4 months later (LG; 22/9/98).

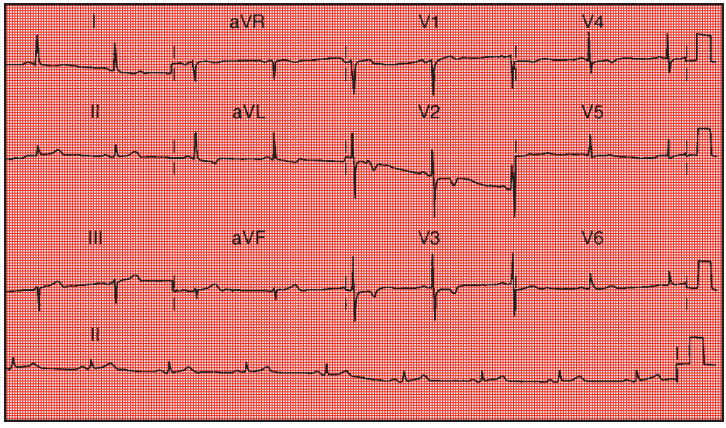


FIG. 2.28. This 57-year-old patient presented with unstable angina, ischaemic changes (T wave inversions) are noted in the anteroseptal leads (aVL, V2, V3, V4) (SM; 13/3/93).

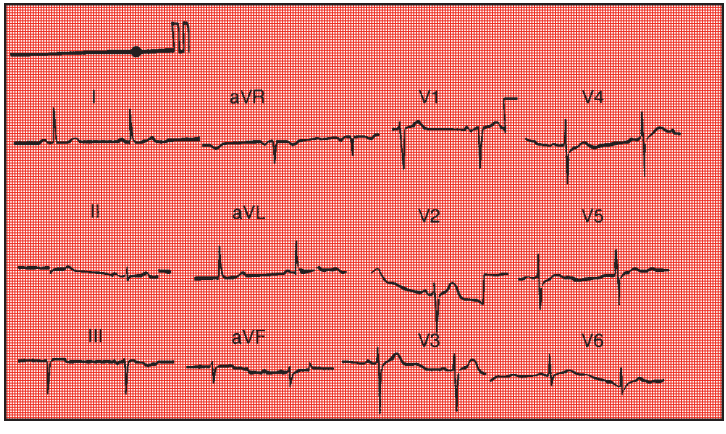


FIG. 2.29. After successful angioplasty, all changes returned to normal (SM; 24/3/93).

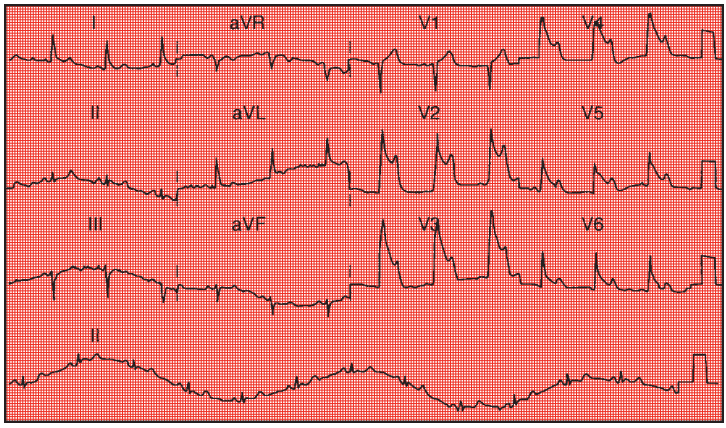


FIG. 2.30. The same patient experienced further angina 3 months later. Angioplasty resulted in total occlusion of the left anterior descending artery, with marked ST segment elevation in leads V2–V6. Emergency bypass surgery was performed the same day (SM; 15/6/93).

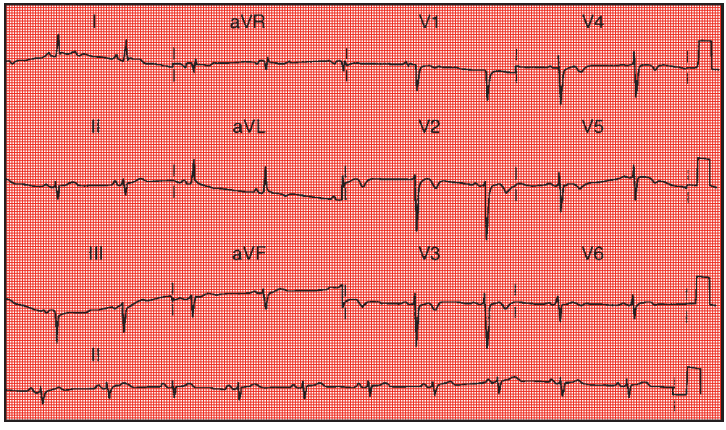


FIG. 2.31. Three days later, residual ischaemic changes are visible in leads V2–V5 (SM; 18/6/93).

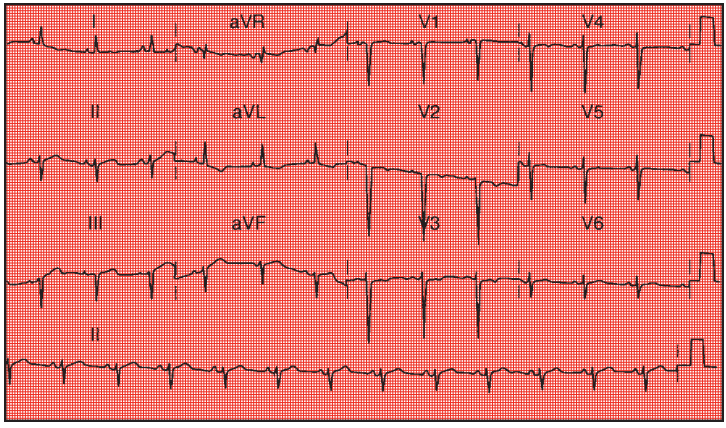


FIG. 2.32. Further improvement but Q waves are now visible in leads V1 and V2 (SM; 22/6/93).

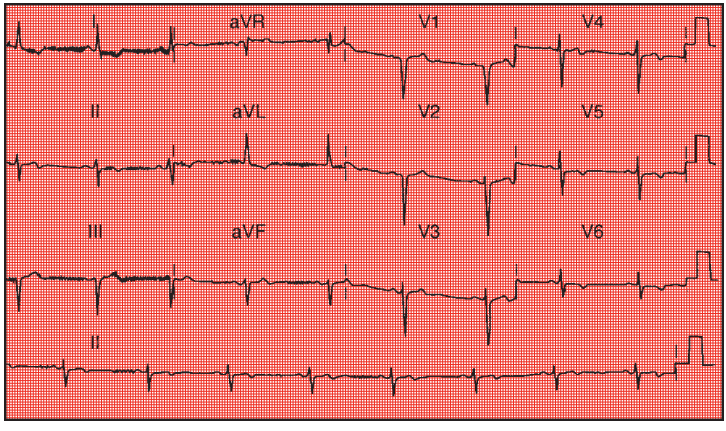


FIG. 2.33. After 5 years, the patient is well, but permanent "ischaemic" changes are visible anterolaterally. Q waves are visible only in lead V1, indicating regenerating healthy muscle (SM; 18/5/98).

Inferior Myocardial Infarction

The unopposed right-sided vectors (inferior window) show up as Q waves in the inferior leads – that is leads II, III and aVF (Figs. 2.34–2.43). ST elevation demonstrates similar distribution.

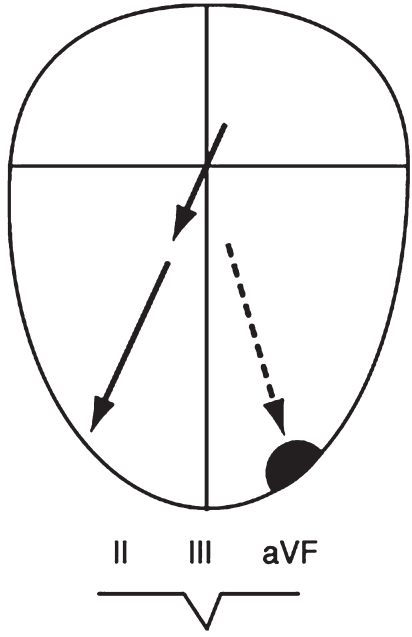


FIG. 2.34. Inferior infarction. Here the Q waves are seen in the inferior leads (II, III, aVF).

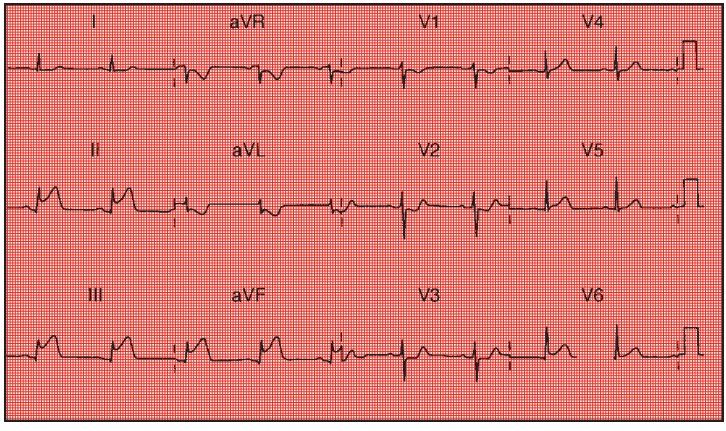


FIG. 2.35. Acute inferior infarction. ST segment elevations are present in leads II, III and aVF. Reciprocal changes are present in leads aVR, AVL, V1 and V2 (JB; 12/10/98).

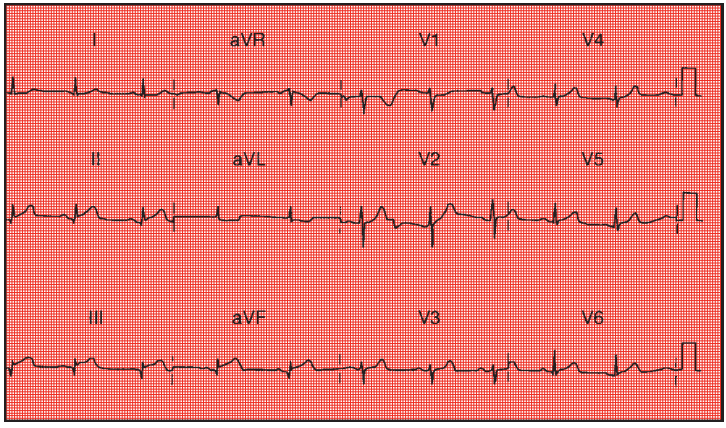


FIG. 2.36. There is rapid resolution after administration of intravenous thrombolysin (JB; 12/10/98).

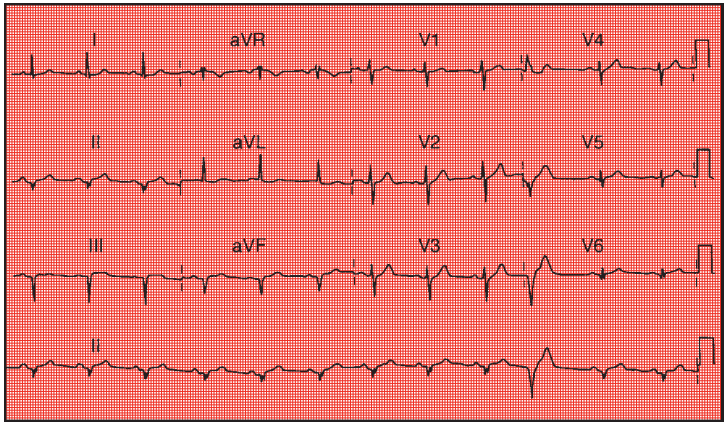


FIG. 2.37. Several hours later, Q waves are seen in leads II, III and aVF (JB; 12/10/98).

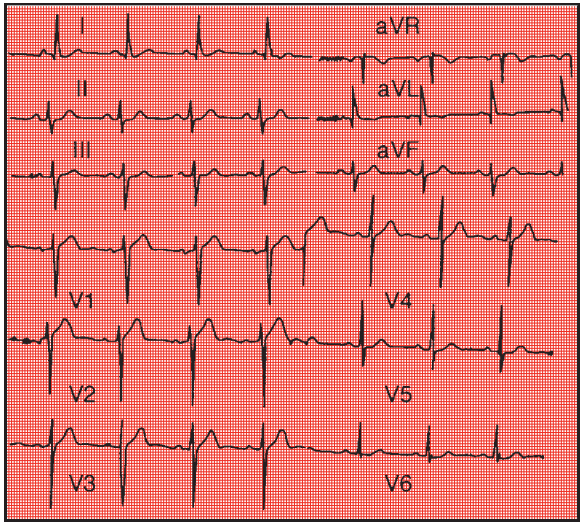


FIG. 2.38. Normal trace (Al; 14/9/70).

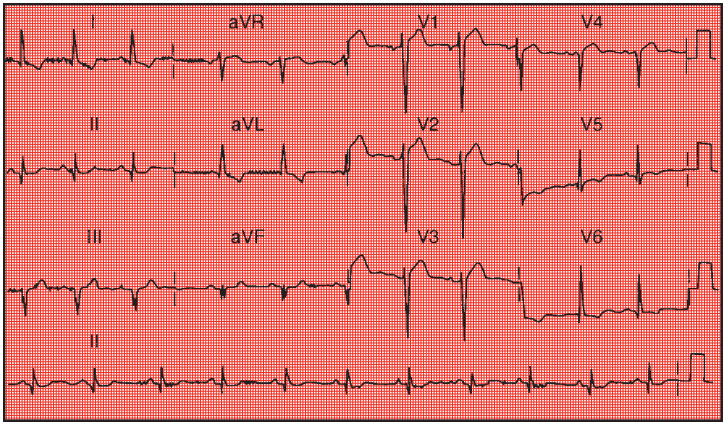


FIG. 2.39. The same patient suffered an inferior infarction 16 years later. There are Q waves in leads II, III and aVF, and T inversions in the anterior lateral leads. The patient underwent bypass surgery (AL; 3/12/86).

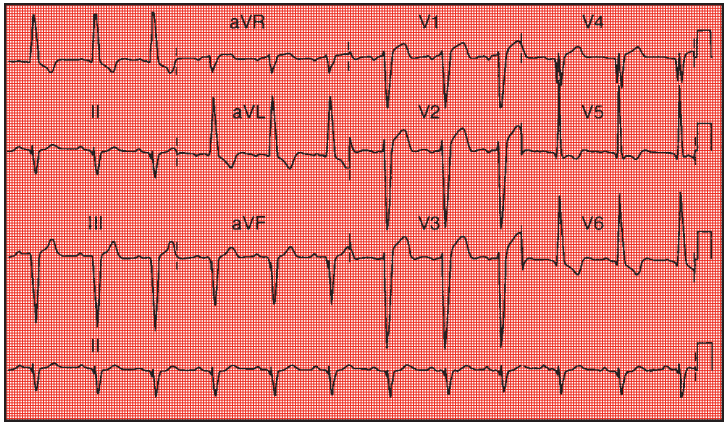


FIG. 2.40. After another 12 years, the patient has developed left bundle branch block. Q waves are still visible in leads II, III and aVF (AL; 2/2/98).

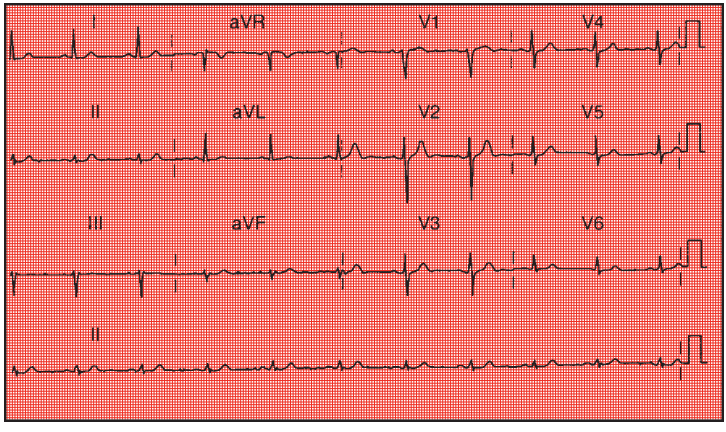


FIG. 2.41. Normal trace (ED; 28/1/98).

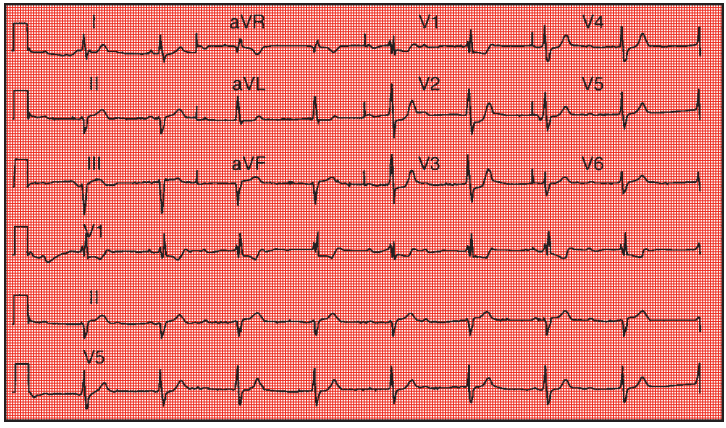


FIG. 2.42. Seven months later, the ECG indicates recent inferior infarction. There are Q waves in leads II, III and aVF. Reciprocal changes (ST segment depressions) are visible in leads V2 and V3. Lead V1 shows AV dissociation, suggesting complete heart block. P waves and QRS complexes are not connected, indicating conduction defect commonly seen with acute inferior infarction (ED; 3/8/98).

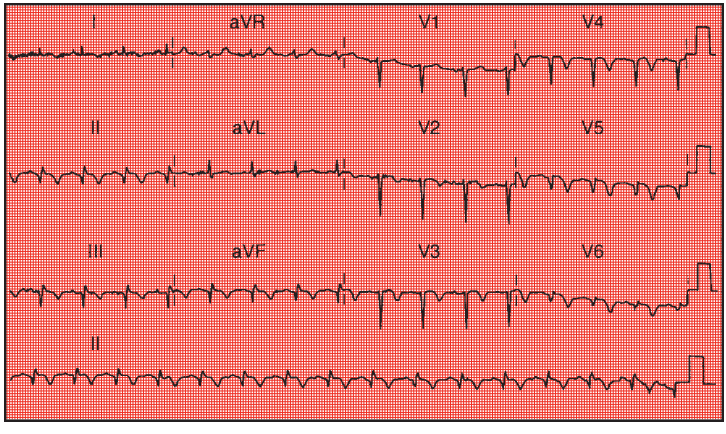


FIG. 2.43. Acute inferior and anterolateral infarction. Q waves and T wave inversions are seen in leads II, III, aVF, V4, V5 and V6 (Mr. R; 26/1/98).

True Posterior Infarction

True posterior infarction is rare. The dead window is situated posteriorly, therefore electrodes facing healthy tissues record unopposed positive forces manifested by dominant R waves in leads V1 and V2 (Figs. 2.44–2.46).

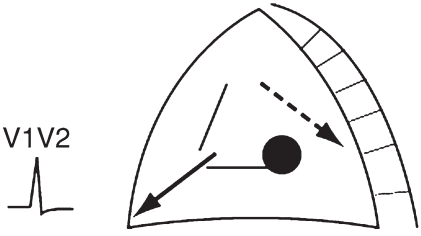


FIG. 2.44. True posterior infarction. Unopposed positive vectors are inscribed as dominant R waves in leads V1 and V2.

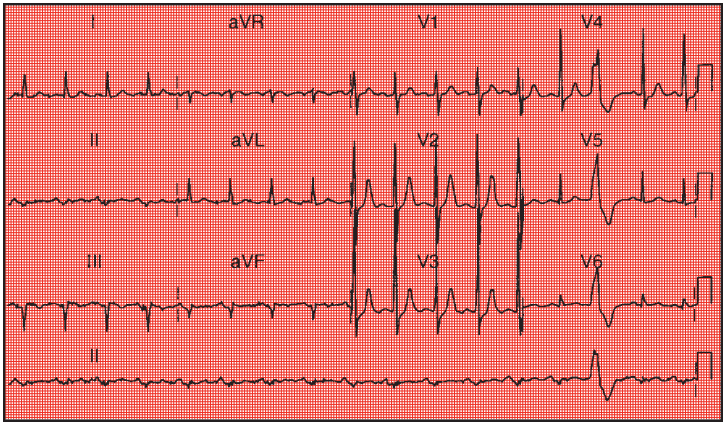


FIG. 2.45. Inferior and true posterior infarction. Q waves are seen in leads II, III and aVF. Dominant R waves are present in leads V1 and V2 (JF; 6/7/98).

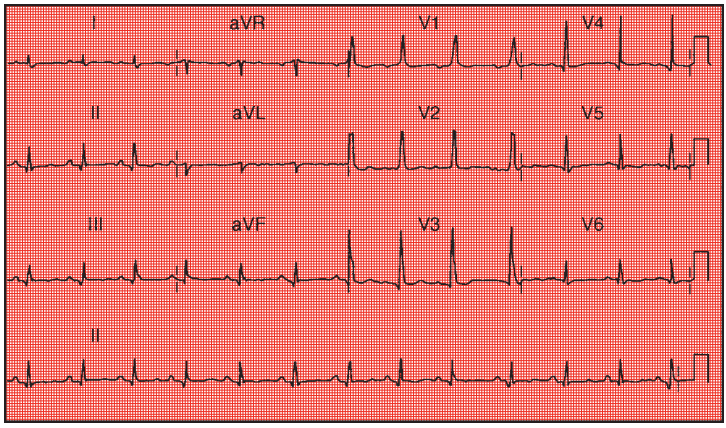


FIG. 2.46. True posterior infarction. Dominant R waves are present in leads V1 and V2. Probably significant, small Q waves are seen in the inferior leads (Mr. R; 26/6/97).

PROGRESSION OF CHANGES AFTER ST ELEVATION INFARCTION

ST elevation, which is the first sign of infarction, resolves within a few hours; the T waves on the other hand revert to normal after several days or weeks but the Q wave is nearly always permanent, although it can become less prominent over a period of time (Figs. 2.47–2.90).

Further signs of ischaemic changes in ECG

- New, tall and peaked T waves may appear as a result of narrowing or obstruction of an epicardial artery; "hyperpolarization" occurs in the epicardial layer.
- Depression of the ST segment with T wave inversion in the lateral leads can be caused by acute elevation of the left ventricular end diastolic pressure (related to subendocardial ischemia).
- Distortion of the terminal QRS complex with reduced S waves can appear as a result of late depolarization of the Purkinje system.
- Reduction of R wave progression over precordial leads indicates loss of LV musculature (also seen in obesity and emphysema).

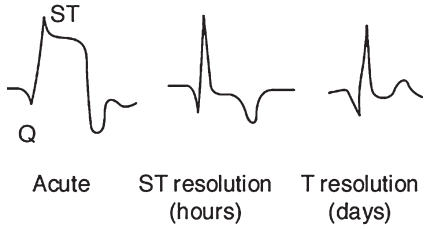


FIG. 2.47. Progressive ECG changes after Q wave infarction.

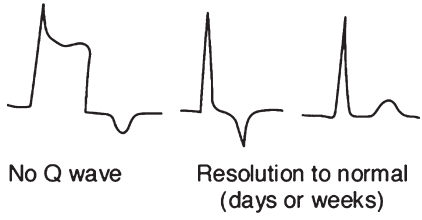


FIG. 2.48. Progression of ST elevation myocardial infarction.

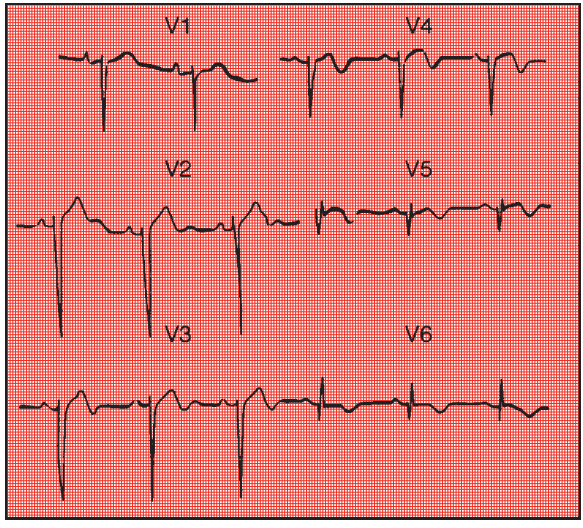


FIG. 2.49. Apical infarction: Q waves are visible in leads V5 and V6.

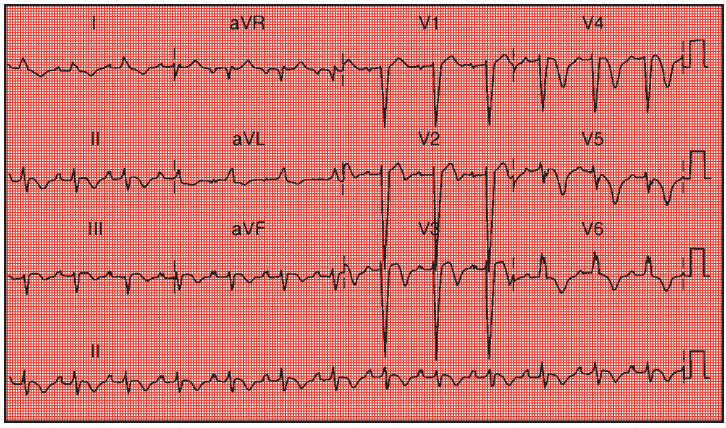


FIG. 2.50. This 87-year-old man had a subendocardial infarction in September 1996 (VS; 10/9/96).

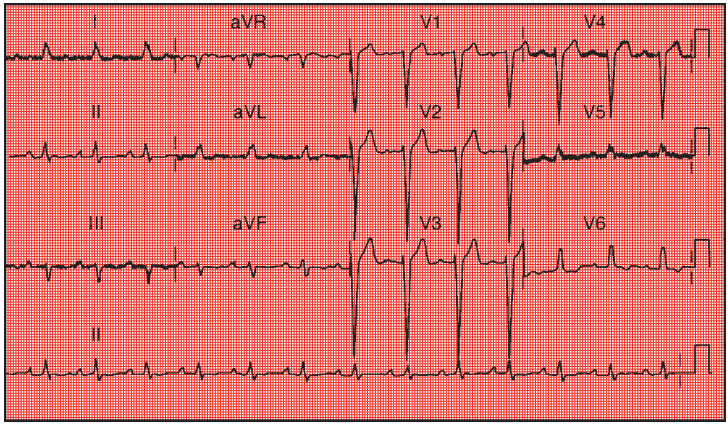


FIG. 2.51. He made a good recovery with medical treatment, as seen in this trace taken 7 months later (VS; 14/4/97).

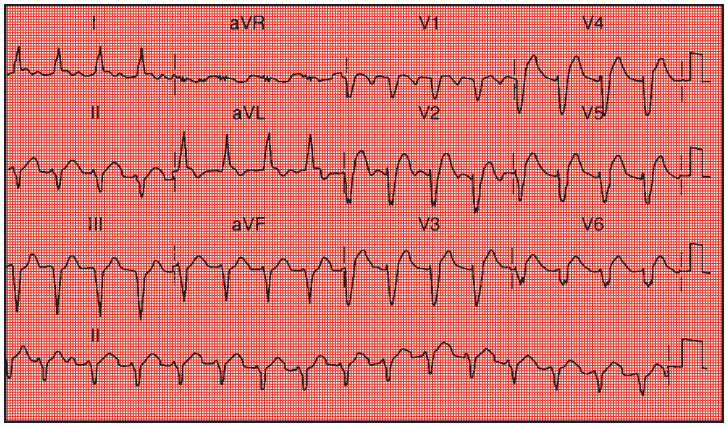


FIG. 2.52. After a further 7 months he suffered anterior infarction with left bundle branch block. ST segment elevation is visible in the precordial leads with reduced R wave progression anterolaterally. The patient died. Subendocardial infarctions carry a bad prognosis (VS; 4/11/97).

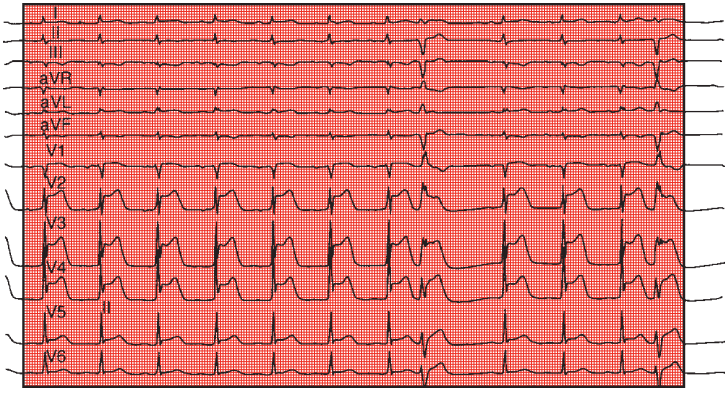


FIG. 2.53. Trace obtained during catheterization of the left internal mammary artery in an elderly patient who had undergone bypass surgery several years previously. Marked anterior ischaemic changes are noted (STEMI). The patient died soon after the procedure (CC; 23/4/98).

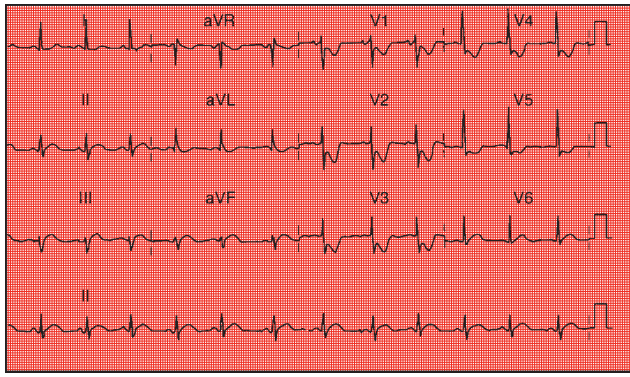


FIG. 2.54. Elderly female patient with subendocardial anterior infarction (NSTEMI). Reciprocal ST segment elevation is visible in leads III and aVF. The patient died on the way to catheter laboratory 5 days after this event (HP; 11/7/99).

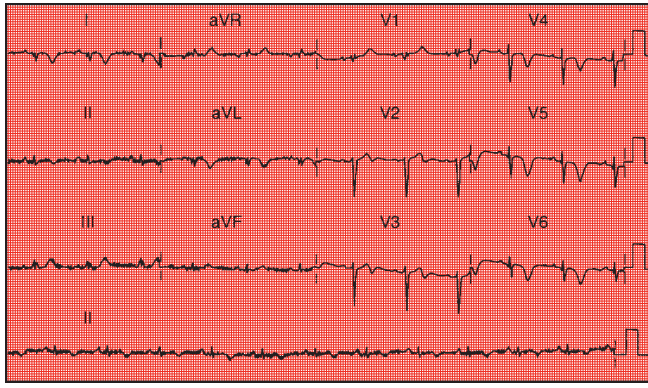


FIG. 2.55. Anterolateral infarction. Q waves are present in leads I and aVL. Reduced R waves are seen in leads V4, V5 and V6 and there are marked lateral T wave inversions (Mr. E; 28/2/96).

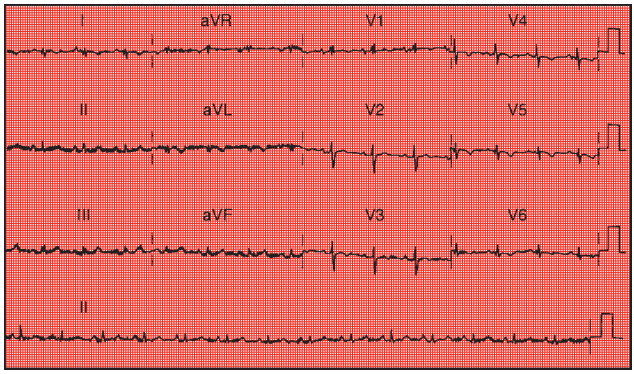


FIG. 2.56 The same patient 1 month after bypass surgery, showing marked improvement in ECG indices (Mr. E; 10/5/96).

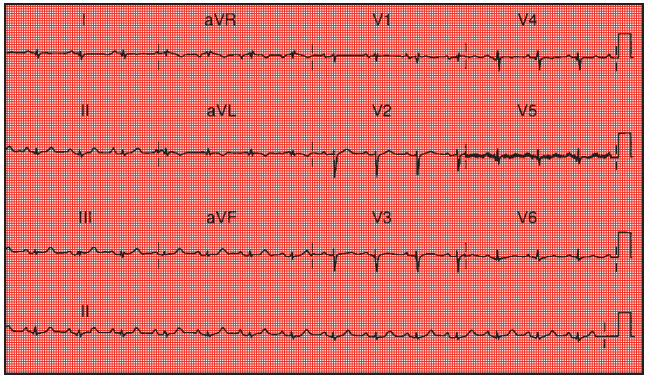


FIG. 2.57. A further 3 months later, there are minor residual Q waves in leads I and aVL with T wave inversion. Reduced R wave progression in leads V4–V6 is maintained (Mr. E; 11/8/96).

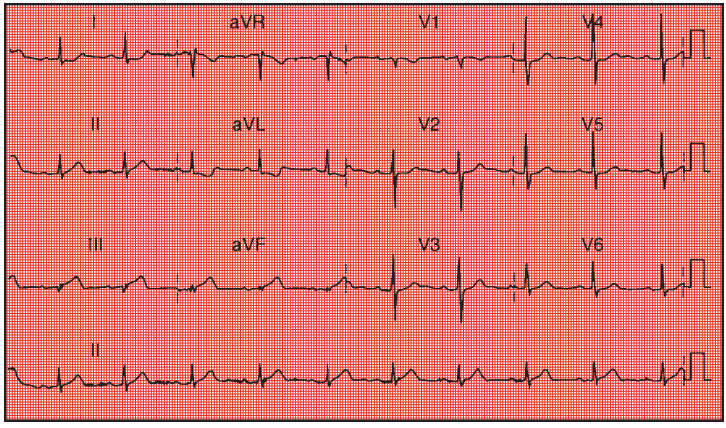


FIG. 2.58. Early changes of acute inferior infarction. ST segment elevation is present in leads III and aVF. Reciprocal changes are seen in leads I and aVL (Fl; 15:10-28/4/96).

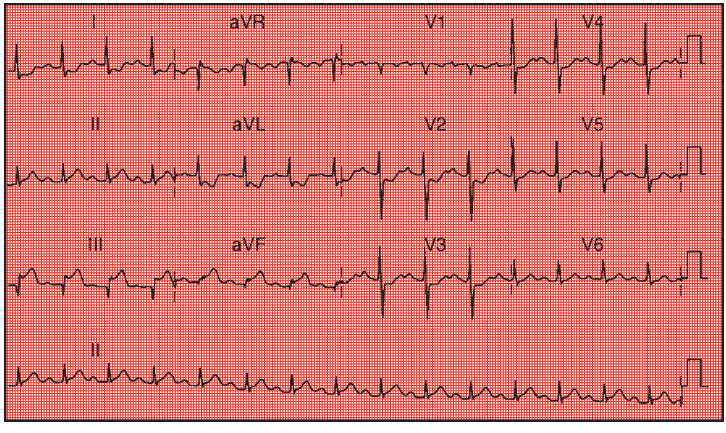


FIG. 2.59. Some four-and-a-half hours later Q waves appear in leads III and aVF with more pronounced ST elevation and reciprocal changes in I, aVL and V2–V5 (FL; 19:35-28/4/96).

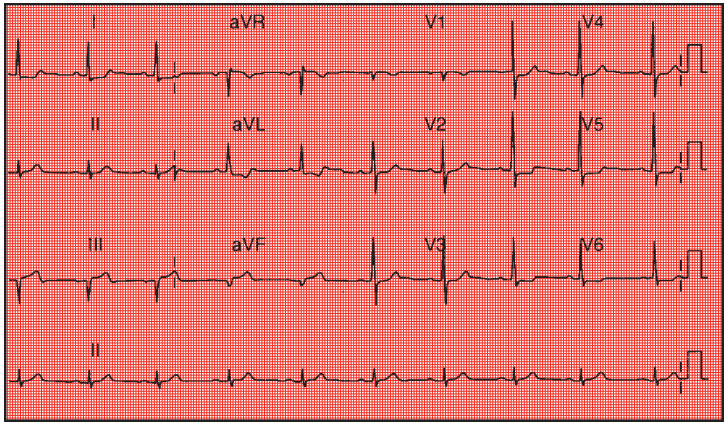


FIG. 2.60. A further 24 h later, inferior infarction is established. Q waves are visible in leads III and aVF, with minor ST segment elevation. These are resolving reciprocal changes (FL; 29/4/96).

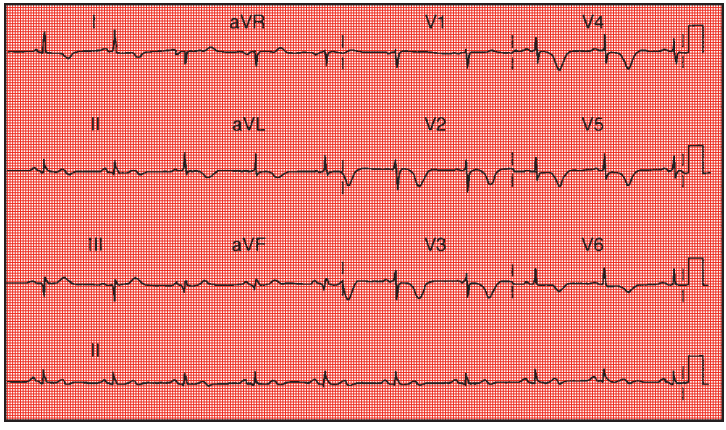


FIG. 2.61. This 83-year-old female presented with subendocardial anterior infarction (NSTEMI) (BE; 5/5/96).

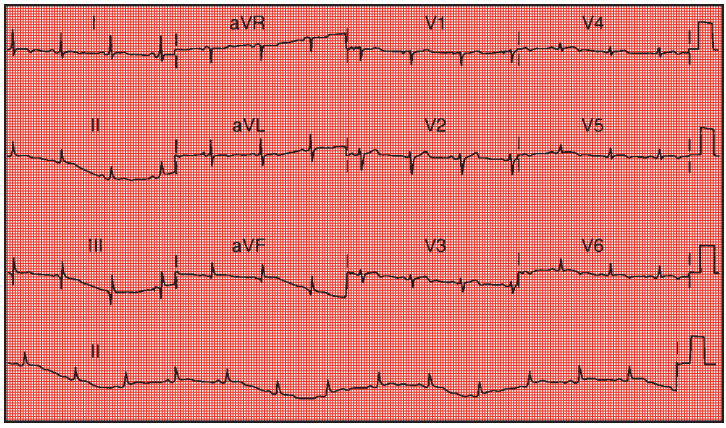


FIG. 2.62. Marked improvement in ECG indices 10 days later, following bypass surgery (BE; 15/5/96).

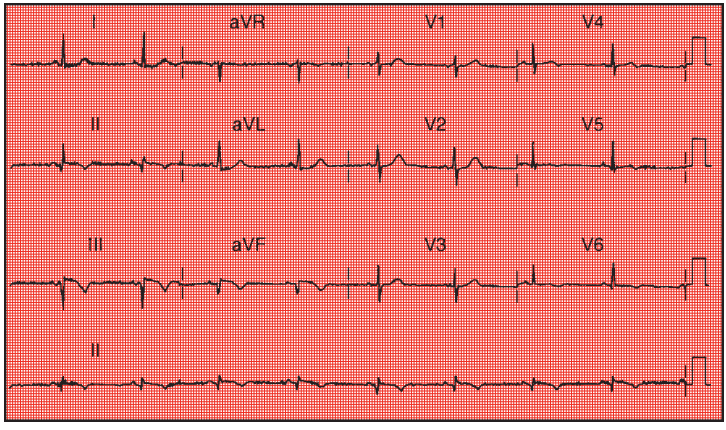


FIG. 2.63. Acute inferior infarction (STEMI) (HT; 28/11/96).

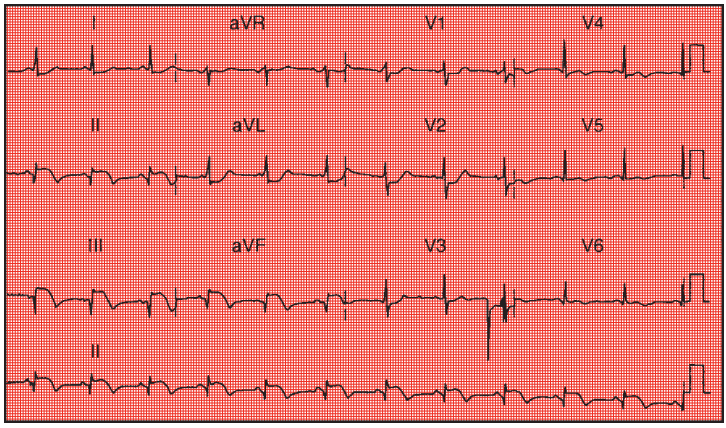


FIG. 2.64. ECG changes worsen 24 h later. Reciprocal ST segment depressions are visible in leads V1 and V3 (HT; 29/11/96).

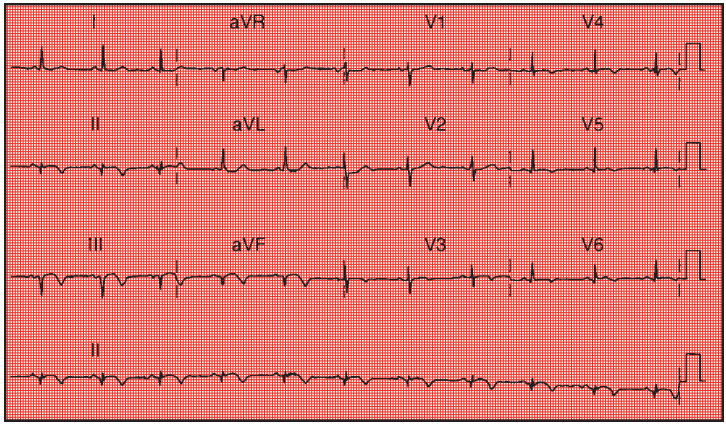


FIG. 2.65. One week later, after successful angioplasty of the right and left anterior descending coronary arteries. There is improvement in the inferior territory with minor T wave ischaemic changes in the LAD territory (HT; 6/12/96).

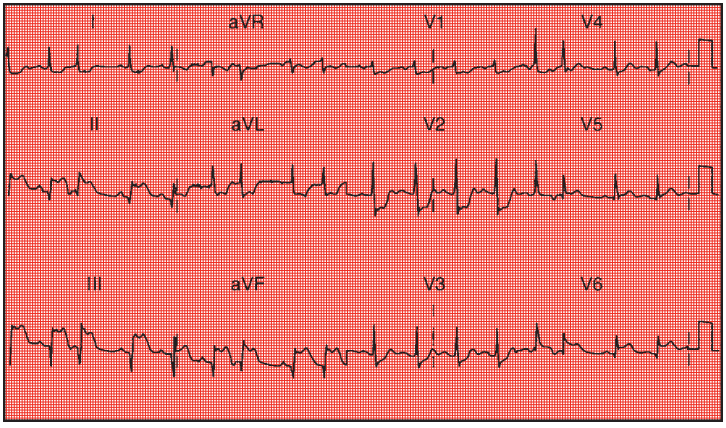


FIG. 2.66. The ECG of this 70-year-old male with acute inferior infarction showed widespread reciprocal ST depressions. The patient was given thrombolysins (RW; 6/10/95).

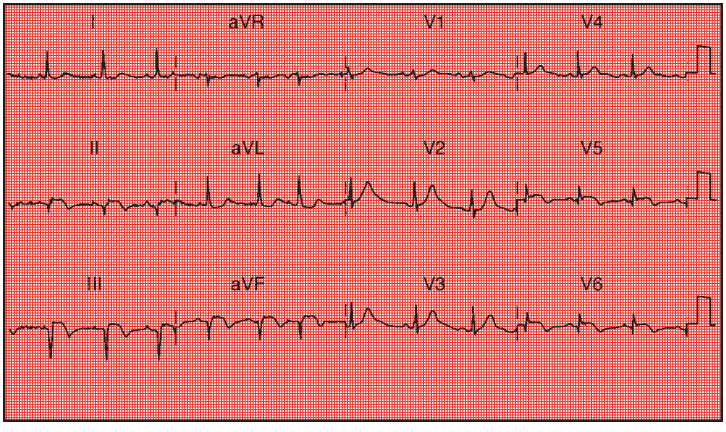


FIG. 2.67. After 2 days the reciprocal changes have largely disappeared. The inferior ST changes are resolving but Q waves are evident (RW; 8/10/95).

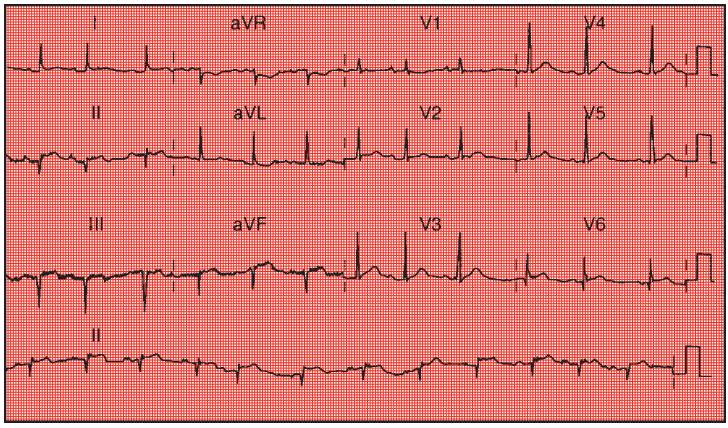


FIG. 2.68. After a further 3 days only the inferior Q waves remain in leads II, III and aVF (RW; 11/10/95).

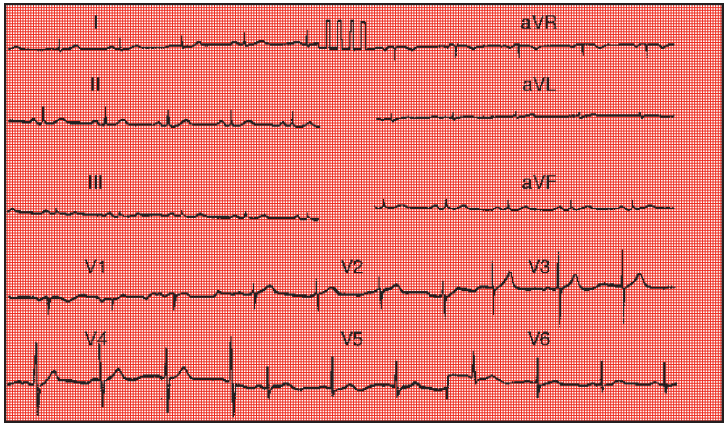


FIG. 2.69. This 52-year-old patient has a normal ECG (AS; 15/4/85).

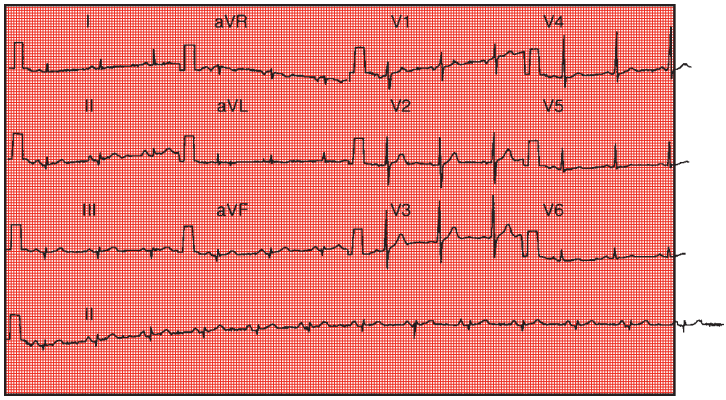


FIG. 2.70. Ten years later, there are Q waves indicating inferior infarction. The patient was unaware of this (silent myocardial infarction) (AS; 4/9/95).

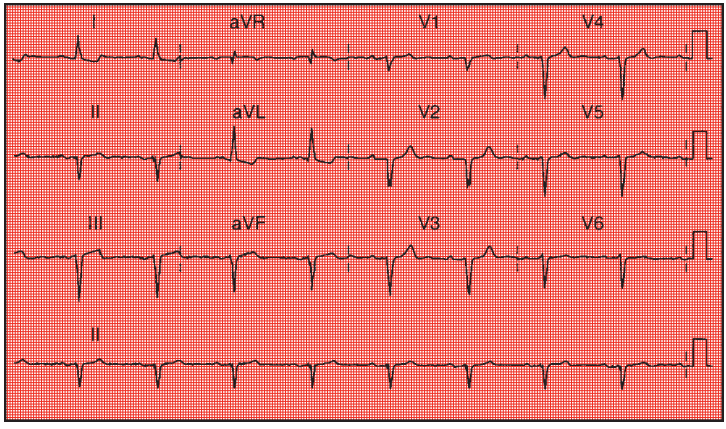


FIG. 2.71. ECG showing inferior and anterolateral Q waves with no R waves in leads V4–V6 indicating widespread infarcted territories (SK; 10/9/99).

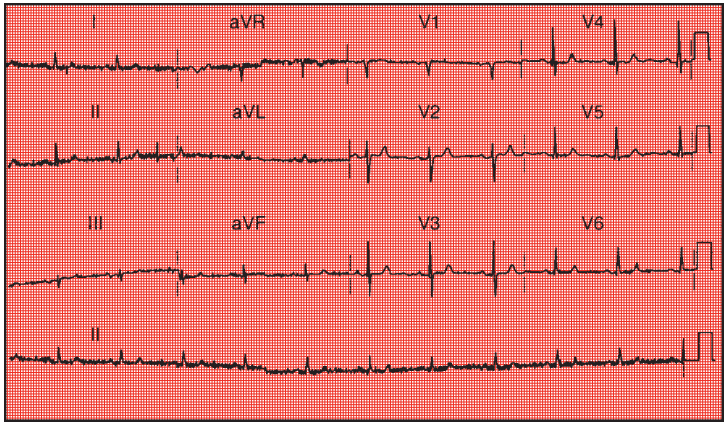


FIG. 2.72. Normal ECG in an elderly female patient in her mid-80s (HI; 1/3/99).

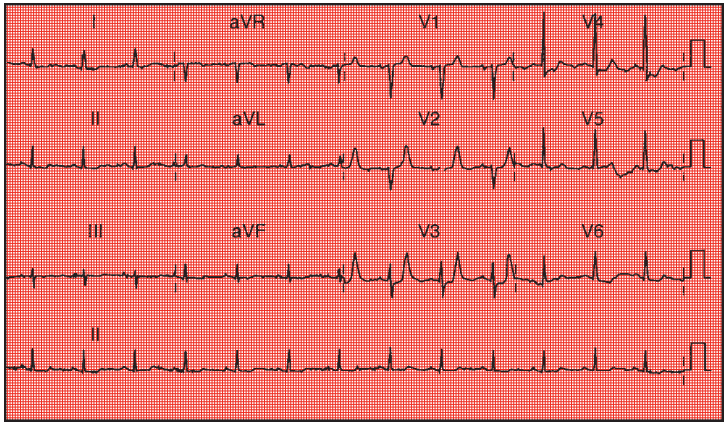


FIG. 2.73. The same patient presented with chest pain 4 months later. There are peaked T waves in leads V2 and V3 with some ST segment depressions in leads V3 and V4; cardiac enzyme levels were elevated (HI; 27/7/99).

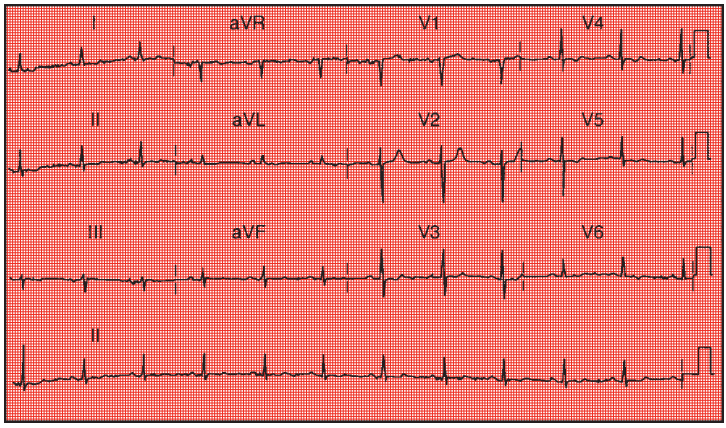


FIG. 2.74. ECG changes resolving with only minor lateral T wave inversions remaining. At catheterization, the patient was found to have three vessel disease which was treated medically (HI; 6/9/99).

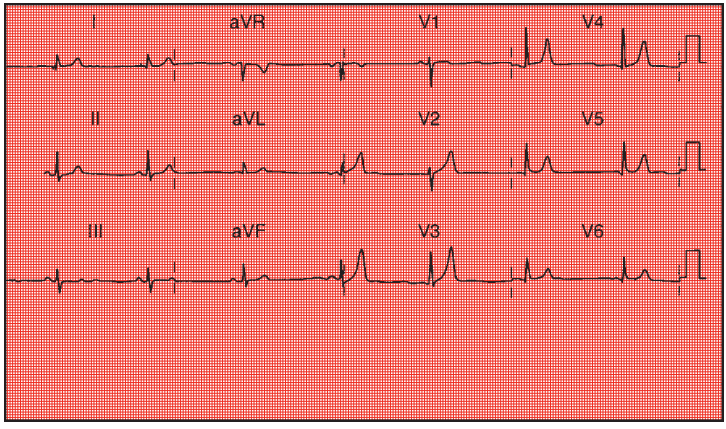


FIG. 2.75. This patient was due to have a mitral valve replacement and had a normal coronary arteriogram (PM; 31/5/99).

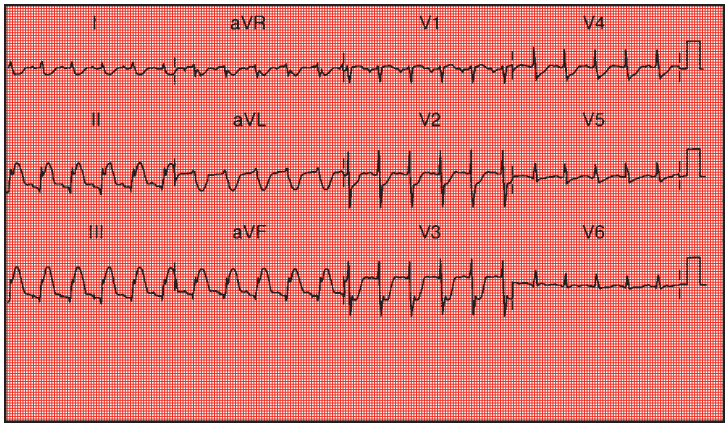


FIG. 2.76. The patient suffered a cardiac arrest in ITU after mitral valve replacement; there is acute inferior ST segment elevation with marked reciprocal changes in leads V2–V4. The patient was resuscitated successfully. The acute changes are due to coronary emboli, ? calcium, ? air (PM; 3/6/99).

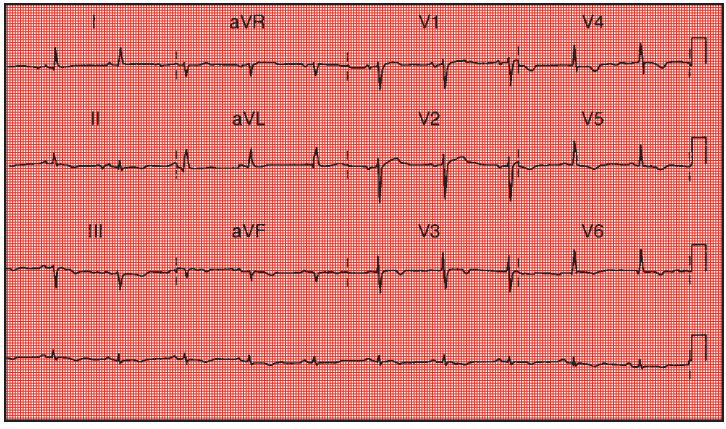


FIG. 2.77. Inferior Q waves with anterolateral T wave inversions are seen 1 week later (PM; 11/6/99).

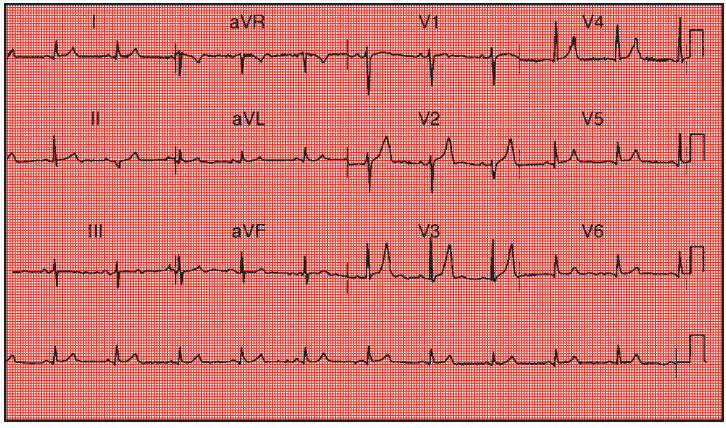


FIG. 2.78. All ECG indices returned to normal 4 months later (PM; October 1999).

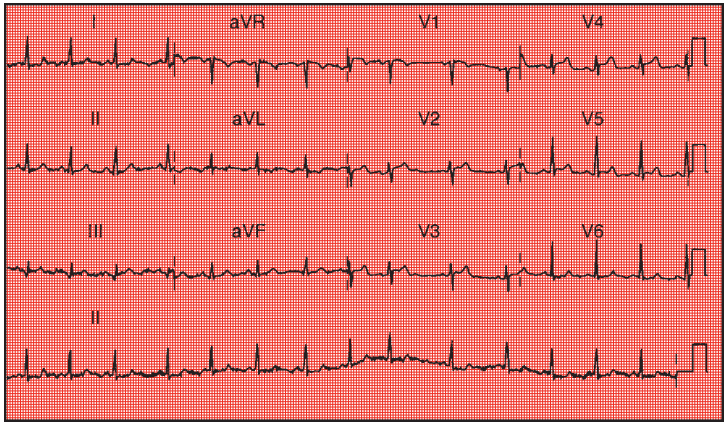


FIG. 2.79. This patient had angina at rest due to coronary arterial spasm. There are ST segment elevations in leads V2–V4 (Mr. K; 1/1/89).

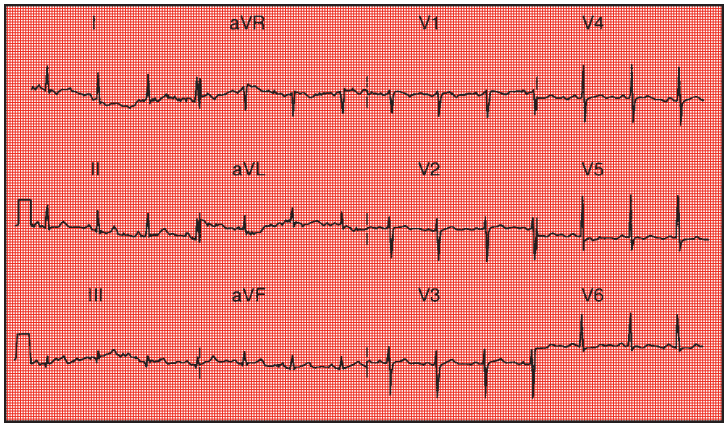


FIG. 2.80. Full resolution (Mr. K; 26/4/99).

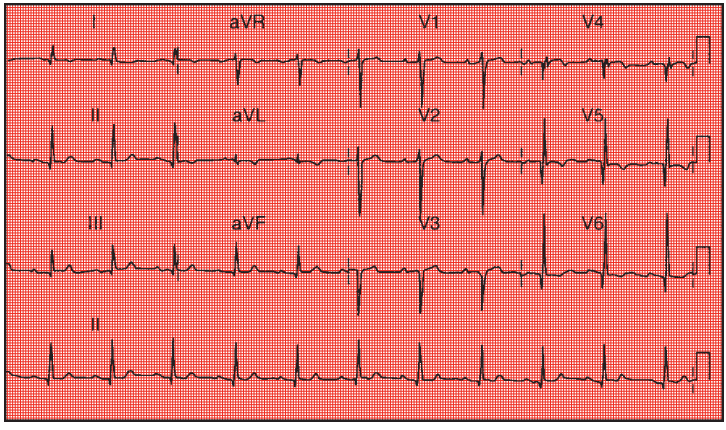


FIG. 2.81. Anteroapical infarction with deep Q waves in leads V3–V6 (RM; 30/11/98).

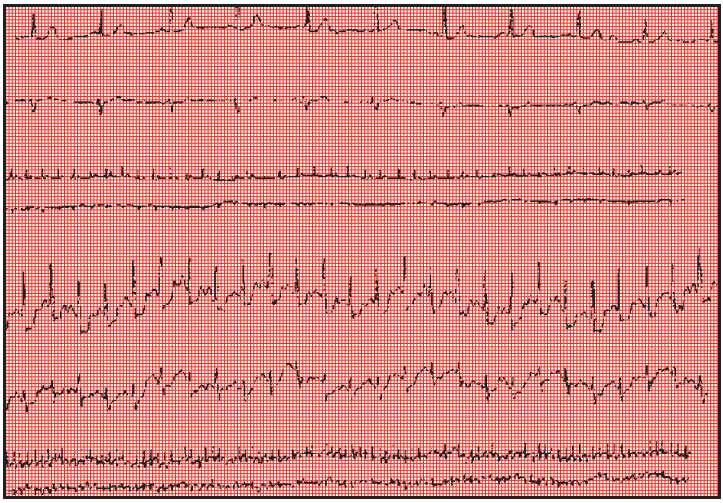


FIG. 2.82. Twenty-four hour Holter monitor showing acute ST segment depressions. The patient was unaware of her symptoms, she suffered from silent ischemia (KB; 18/12/98).

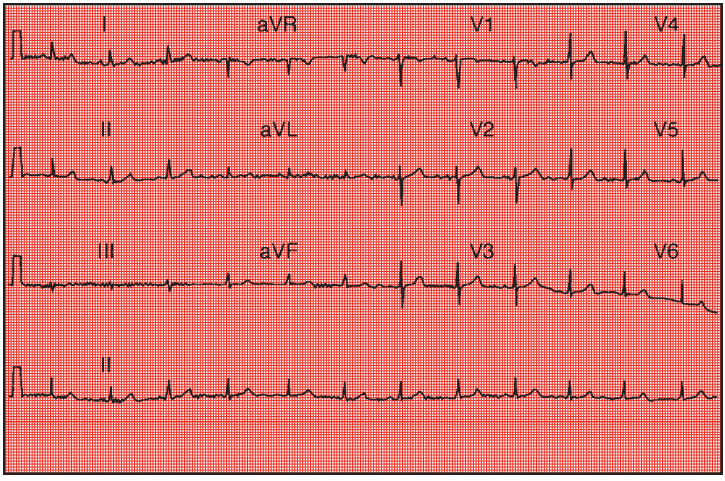


FIG. 2.83. This young Japanese patient has a normal electrocardiogram (CH; 24/12/94).

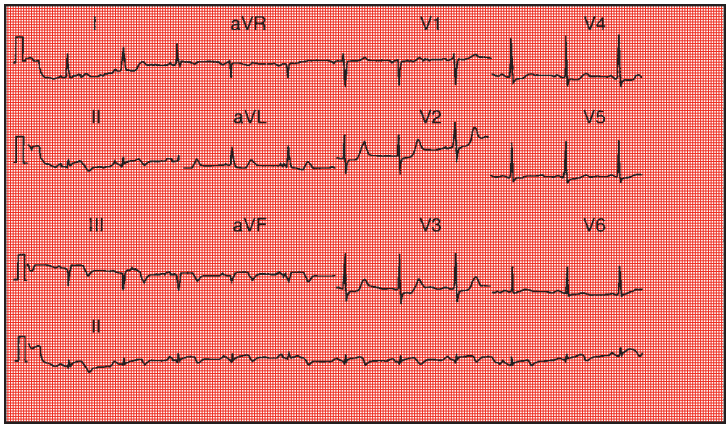


FIG. 2.84. Five months later, she presented with acute inferior infarction (CH; 13/5/95).

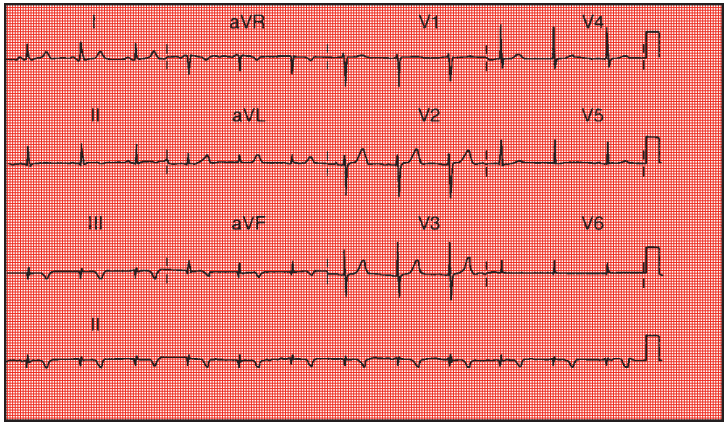


FIG. 2.85. Good resolution is evident 4 months later. Cardiac catheterization revealed spontaneous dissection of a coronary artery (see Fig. 2.86). (CH; 5/9/95).

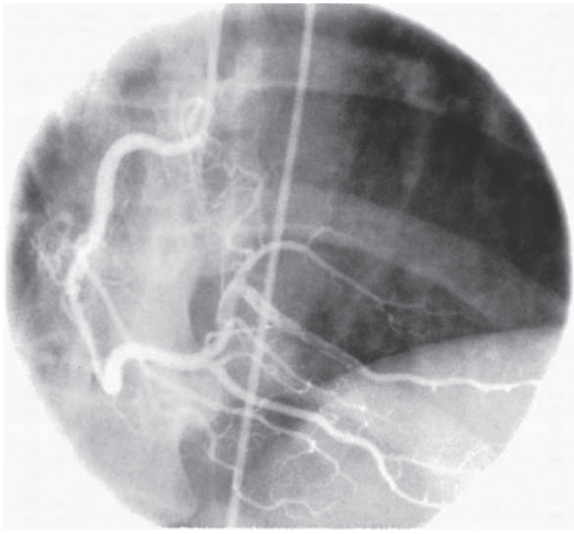


FIG. 2.86. Right coronary arteriogram from the same patient (Fig. 2.84) showing dissection of the right coronary artery, which is well seen distally. The patient made a spontaneous recovery.

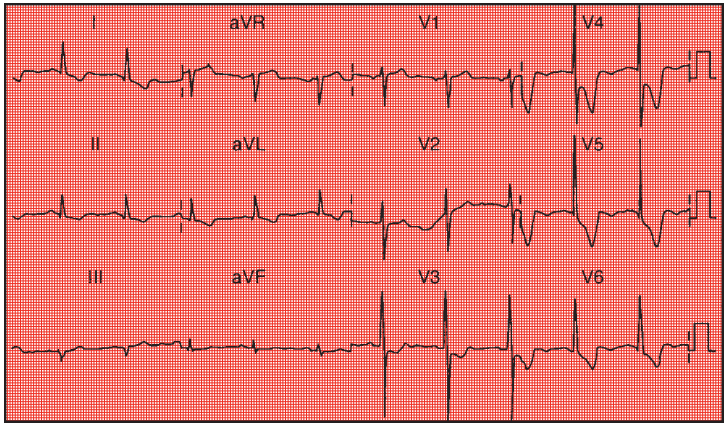


Fig. 2.87. Unstable angina with marked anterolateral ischaemic changes. Cardiac catheterization showed critical mainstem stenosis. The patient underwent surgery on the same day (MK; 5/12/95).

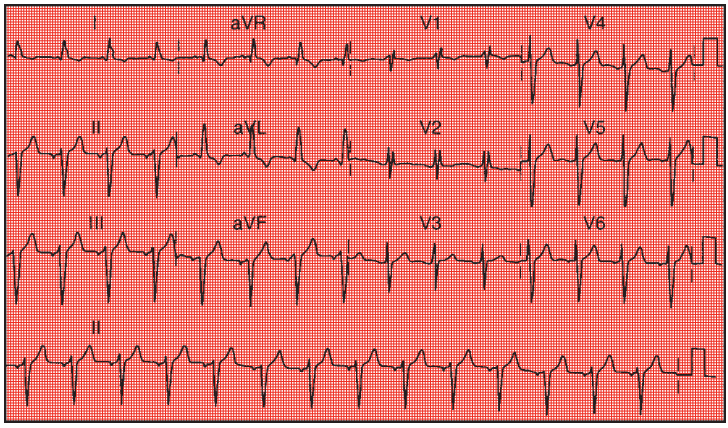


FIG. 2.88. The patient made a good recovery; 6 days after surgery, right bundle branch block and LAD are now evident (MK; 11/12/95).

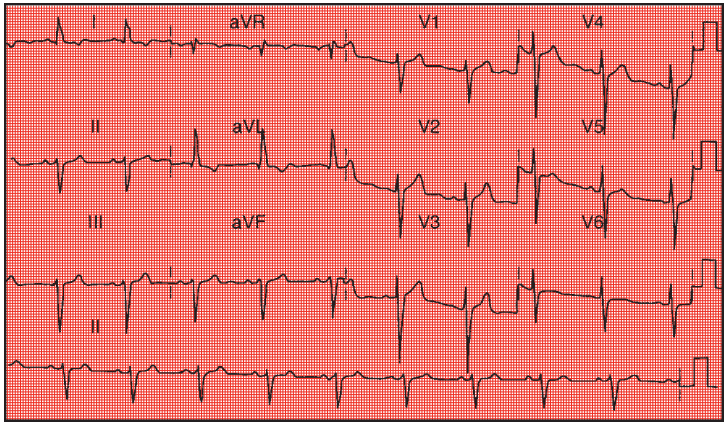


FIG. 2.89. Three years later the patient is well with some reduction of R wave progression (MK; 6/11/98).

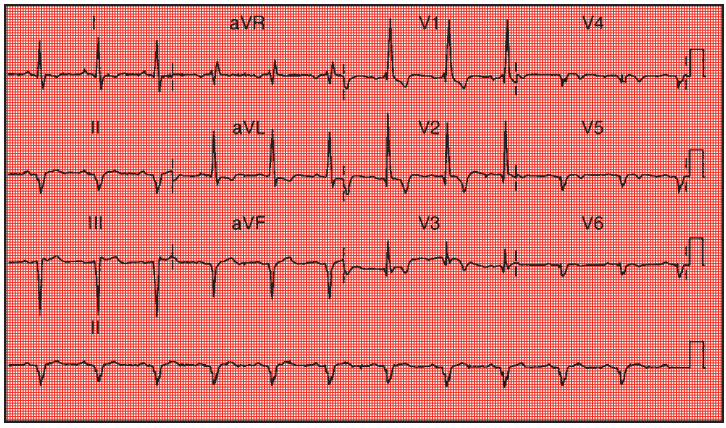


FIG. 2.90. Documented inferior and anterolateral infarctions with right bundle branch block. Q waves are seen in leads V2, V3, II, III and aVF (Mr. J; 12/6/99).

Notes on Q Wave

A Q wave is considered abnormal when it is

- 0.03 s (30 ms) in duration, or greater than 25% of the following R wave
- Seen in leads normally showing an initial R wave

EXERCISE ECG/STRESS TEST

Angina pectoris classically occurs during physical exertion. The oxygen demand on the myocardium exceeds the supply because of the narrowing of the coronary arteries. This process eventually results in chest pain which is fairly characteristic. The pain radiates from the praecordium to the throat, and down the arms, and is relieved by interruption of the physical exertion or by inhaling, sucking or chewing nitroglycerine. Ischemia can also manifest as breathlessness, caused by elevation of the left ventricular end diastolic pressure. Silent ischemia refers to evidence of myocardial ischemia (ST changes) in the absence of pain. This is particularly evident in diabetics who suffer from autonomic nervous dysfunction.

Stress testing is performed by means of a standardized treadmill or bicycle (ergometry). The test should never be undertaken in a patient who has not been fully examined; it would be dangerous in the presence of congestive cardiac failure, rhythm abnor-

mality or hypertension, or immediately after an infarct. It should be noted that certain drugs, such as digoxin, cause ST or T wave changes. Beta-blockers slow down the heart, preventing the heart rate response that is necessary to provoke an attack of angina. A supine ECG is required to ascertain that there are no acute changes before the stress test is undertaken. On standing, there may be certain ECG changes that are not necessarily abnormal, hence the need for a supine trace. Modern equipment provides heart rate, blood pressure and oximetry measurements with computer analysis of the ECG changes.

Both the treadmill and bicycle are standardized (according to the Bruce protocol) so that after 3 min of exercise the load is automatically increased until the patient is unable to proceed, or ECG abnormalities develop (for example ischemia or arrhythmia), or there is a drop or rise in blood pressure beyond normal levels. The abnormality on the ECG that indicates an ischaemic response consists of a horizontal ST depression of at least 1 mm of 80 ms duration (Figs. 2.91–2.94). The stress test should be abandoned at 4–5 mm depression of the ST wave.

Other Ecg Abnormalities On Stress Testing

ST elevation, rhythm or conduction disturbances can develop. A drop in blood pressure indicates severe coronary artery disease

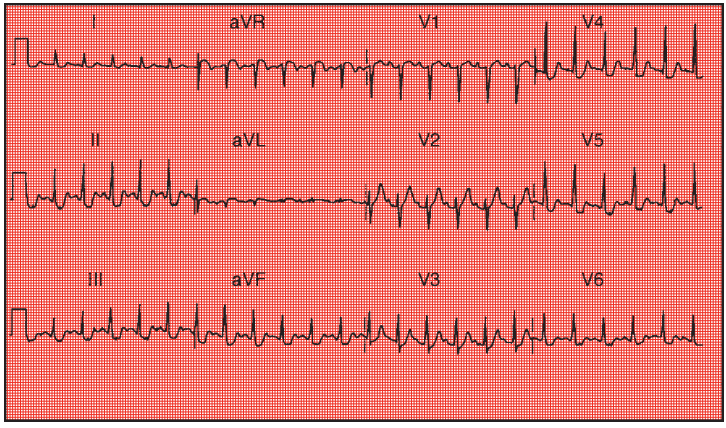


Fig. 2.91. Positive stress test in a patient with coronary artery disease. At a heart rate of 139 bpm, with symptoms of angina, the patient develops marked ST segment depressions in the inferior and lateral leads (II, III, aVF, V4, V5 and V6) (LL; 11/11/96).

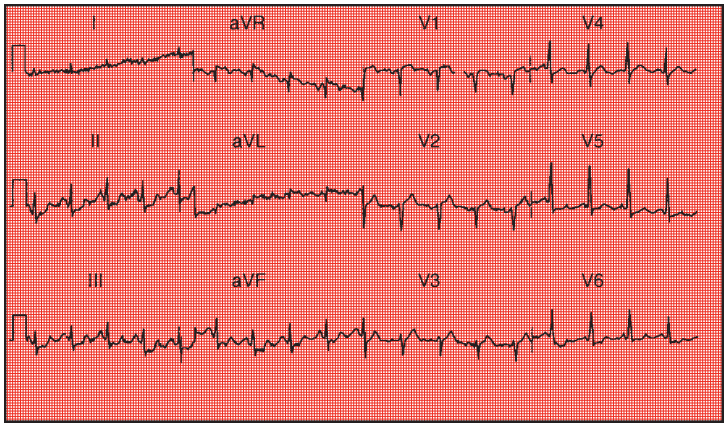


FIG. 2.92. Positive stress test with angina at a heart rate of only 107 bpm. ST segment depressions are seen in the inferior leads and in lead V6 (CW; 29/11/99).

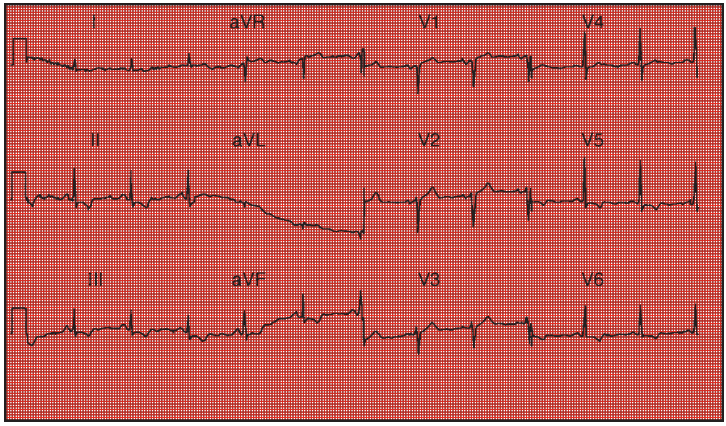


FIG. 2.93. During recovery at a heart rate of 71 bpm, classical T wave inversions occur in the same leads. These T wave inversions during recovery are pathognomonic for coronary artery disease. The patient had three vessel disease at coronary angiography (CW; 29/11/99).

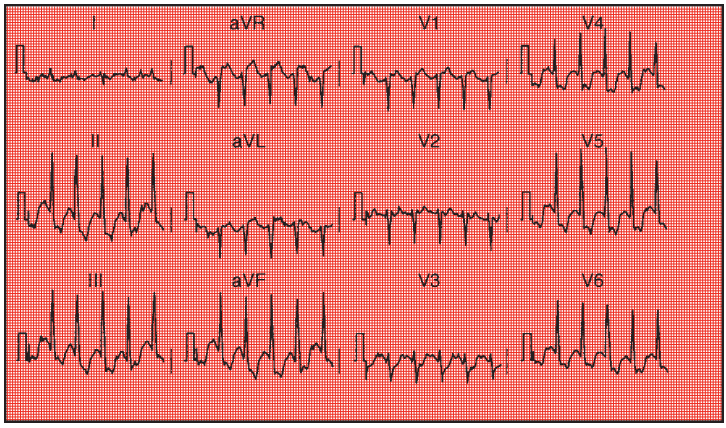


FIG. 2.94. Middle-aged female patient with severe aortic stenosis. Marked ST segment depressions during exercise at 158 bpm are noted in the inferior lateral leads. The coronary arteriogram was normal (AS; 3/7/91).

or a vasovagal response caused by hyperventilation with peripheral vasodilatation. In such instances, the patient is monitored in a horizontal position until recovery. Full resuscitative facilities must be available (defibrillator, cardiac drugs, oxygen and suction apparatus). The test is usually performed by technicians with a doctor in attendance. Complications are rare when the test is carried out properly and the personnel are well trained. However, occasional infarction can occur and fatalities are on record. The patient is observed for 5–10 min after the stress test, or longer if complications arise, with continuous ECG and blood pressure evaluation. The severity of a positive stress test – that is, the degree of ischemia –is judged by the duration of the exercise, the appearance time of either symptoms or ECG changes, heart rate, blood pressure and oxygen saturation response. The last reflects left ventricular and pulmonary function. Abnormal levels will occur with chronic pulmonary disease, right to left shunting or pulmonary edema.

The occurrence of supraventricular and particularly ventricular arrhythmias (other than occasional extrasystoles), the appearance of bundle branch block or conduction defects, or a delayed decrease in heart rate during the first minute after the exercise are all parameters indicating a negative prognosis.

It has to be made quite clear that a positive stress test does not necessarily indicate underlying coronary pathology. ST and T wave changes during exercise can occur for other reasons:

- A false positive test occurs in up to 5% of the population, more frequently in female patients (Fig. 2.95)
- Related to hyperventilation
- Associated with aortic valve disease (mainly severe aortic stenosis) or hypertrophic cardiomyopathy (Fig. 2.94)
- Hypertension (Fig. 5.10)

Nuclear (Isotope) Stress Exercise Testing (Scintigraphy)

Intravenous radioactive substances (thallium, technetium, sestamibi) supplemented by coronary vasodilators (dipyridamole or adenosine) are injected during exercise. Gamma camera pictures are obtained during exercise and subsequently at rest. Pictures of the isotope within the myocardium underline areas of underperfusion during exercise with improved flow after resting (indicating reversible ischaemic changes).

These findings relate to occlusive or stenotic lesions affecting the coronary arteries (Figs. 2.96–2.98).

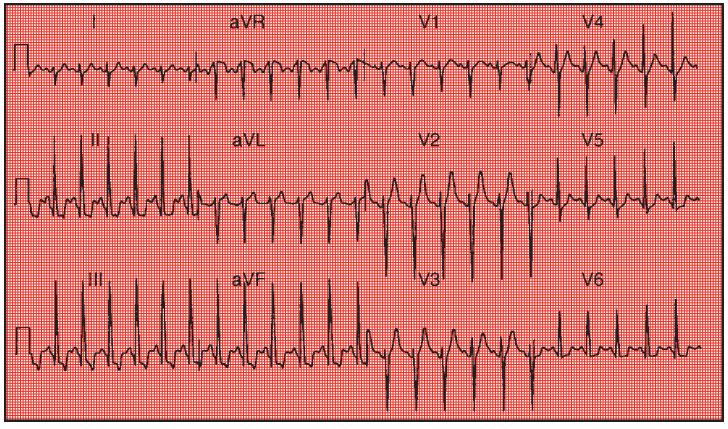


FIG. 2.95. This young patient, a professional diver, had a routine stress test that indicates coronary artery disease based on ST depressions with T wave inversions in the inferolateral leads (II, III, VF and V6). The patient had no symptoms and coronary arteriography is entirely normal. This is an example of a false positive stress test (JB; 16/11/99).

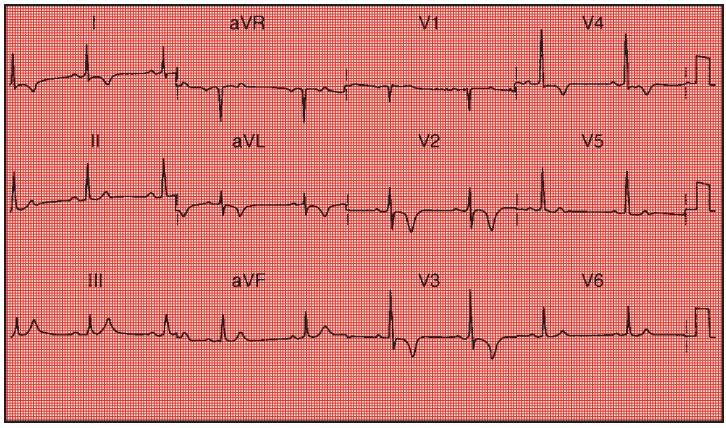


FIG. 2.96. This 86-year-old man presented with severe unstable angina. His cardiac enzyme levels were not elevated. He was treated medically (PR; 16/4/95).

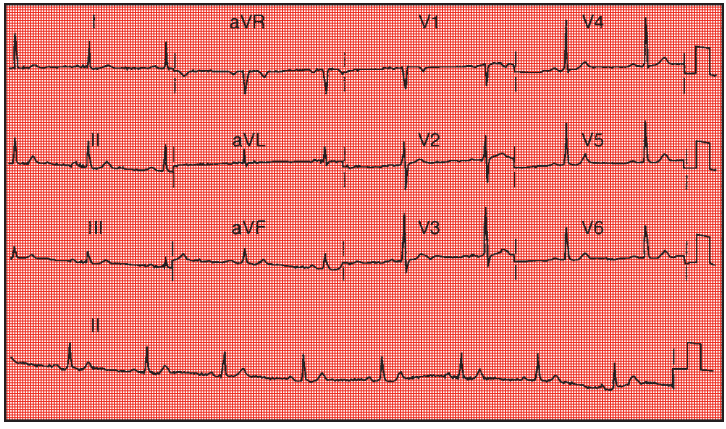


FIG. 2.97. After a strongly positive myocardial perfusion study (see Fig. 2.98), the patient underwent stenting to a tight LAD lesion 3 days after admission. This produced a good result and a total resolution of ECG changes (PR; 19/4/95).

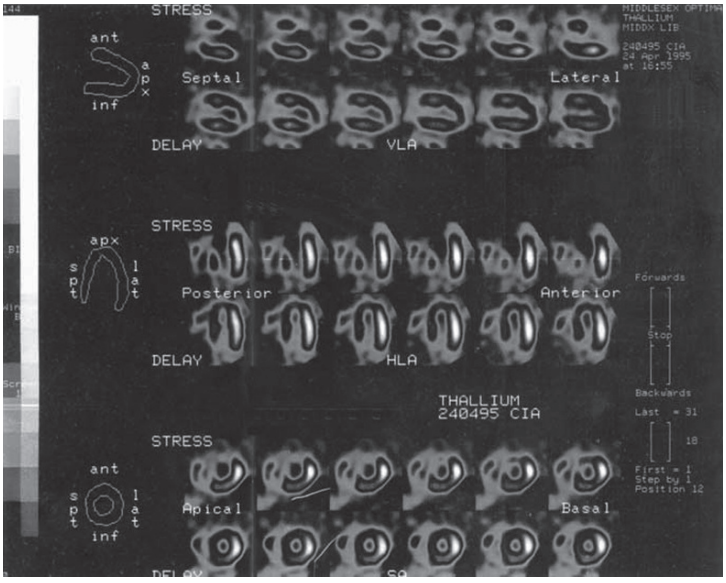


FIG. 2.98. Myocardial perfusion stress test in three oblique views. The *upper pictures* were taken during stress, and the *bottom pictures* after a period of recovery. In all views there is some resumption of flow in the LAD territory during recovery, indicating reversible ischaemic changes in the apex, anterior wall and septum. A tight LAD stenosis was confirmed at angiography (PR; April 1995).

Stress Echocardiography

This investigation involves echocardiographic imaging of the left ventricle at rest and during infusion of dobutamine and atropine. The purpose is to detect areas of undercontractile LV musculature related to regional coronary pathology (regional wall motion abnormality).

ANGINA AT REST (VARIANT, PRINZMETAL, OR VASOSPASTIC ANGINA)

Angina pain occurs at rest, rather than related to physical exertion. It can be brought on by mental stress or nightmares. The symptoms are similar to effort angina, and usually occur in advanced three vessel disease (LAD, circumflex and right coronary arteries) or mainstem disease. Less frequently, localized coronary arterial spasm can be demonstrated (ST elevation) or provoked (ergonovine test) during coronary angiography.

Management consists of calcium antagonists, potassium channel openers and nitrates (beta-blockers are contraindicated).

UNSTABLE ANGINA

In unstable angina, the classical symptoms in a patient who was previously stable change so that the pain becomes more intense, occurs at rest, is provoked by less physical exertion and/or becomes less responsive to the usual medication. Unstable angina is now considered a medical emergency requiring hospitalization with aggressive management (medical and/or interventional). This consists of intravenous nitrates, heparin and/or clopidogrel, and aspirin, usually followed by angiography. This will indicate the need for percutaneous transluminal coronary angioplasty (PTCA) with or without stenting, or even coronary artery bypass surgery (CABG).

OTHER ISCHAEMIC HEART CONDITIONS

Syndrome X

Syndrome X is characterized by exertional angina. Classical ST changes are observed during exercise stress testing, but coronary arteriography shows no obvious disease in the epicardial arteries. The pathology is believed to reside within the myocardial vessels, caused by either abnormal resistance to flow or some metabolic disorder.

Patients usually respond to beta-blockers, calcium antagonists, potassium channel openers, nitrates or a combination of any of these. Oestrogens appear to have a beneficial effect in some women.

Hibernating Myocardium

Hibernating myocardium is a chronic state of muscle underperfusion. This results in reduced LV function, which usually responds to revascularization – in other words, viable muscle is not functioning but is able to return to normal contractility after reperfusion.

Stunned Myocardium

Stunned myocardium follows an acute event resulting from myocardial ischemia that persists after coronary reperfusion. The situation usually remits and responds to inotropes.

Cardiogenic Shock

It is caused by a large infarct with hypotension, profuse sweating, vasoconstriction and pre-renal failure. The mortality can approach 70%. Early aggressive intervention is associated with a better 6-month prognosis.

OTHER ECG INDICES

Other ECG indices can be used to assess prognosis after myocardial infarction. These are listed below.

- Ventricular late potentials: low amplitude, high frequency electrical signals which are seen at the end of QRS complexes (using specialized equipment). These have been used to predict the development of arrhythmias after myocardial infarction. However, the predictive accuracy of the technique is low.
- Heart rate variability: used to obtain prognostic information after myocardial infarction by analysis of beat to beat variations of RR intervals. Vagal influence after myocardial infarction has a significant prognostic value.
- QT dispersion: measures differences between maximal and minimal QT intervals on a 12 lead ECG. Lengthening of the QT interval after myocardial infarction predicts the development of ventricular arrhythmias.
- Heart rate turbulence found in low risk patients after MI by analysis of acceleration or deceleration after a single ventricular premature beat (VPB). A small risk of mortality has been found in patients who do not show this response.

Notes on ST changes

ST ELEVATION

- Seen in normal subjects (in precordial leads)
- Usual in black people (known as high uptake)
- ST elevation is otherwise a sign of:
- Acute myocardial infarction
- Acute coronary spasm (variant angina pectoris)
- Pericardial effusion
- LV aneurysm

ST DEPRESSION

- Always abnormal
- Usually due to ischemia
- Seen in patients on digoxin
- Associated with left ventricular hypertrophy (strain pattern)
- Very rarely seen in the presence of intracranial hemorrhage

HISTORICAL NOTES

RA Bruce (1916–2004) Professor of Medicine, Seattle, USA. Originator of "Bruce protocol," laying down stress testing guidelines.

M Prinzmetal (1908–1987) Professor of Cardiology, UCLA, California, USA. Described "variant" angina due to coronary arterial spasm (1955).

KEY MESSAGES

- STEMI:ST elevated infarction due to occluded vessel
- NSTEMI: non-occlusive infarction with elevated enzymes
- Q wave infarction: full thickness muscle infarction
- Stress testing: treadmill or bicycle (ergometry)
- Stable and unstable angina
- Variant angina
- Acute coronary syndrome (ACS) two presentations NSTEMI and STEMI (ACC – AHA guidelines 1996)

Chapter 3
Conduction Impairment

SINUS ARRHYTHMIA

Sinus arrhythmia is normal in children. It is abnormal in adults but not indicative of specific pathology. The heart accelerates during inspiration and slows during expiration (Fig. 3.1).

Wandering Pacemaker

Wandering pacemaker is usually seen with sinus arrhythmia, with the same clinical significance. The sinus pacemaker may move location within the SA node from beat to beat. This shows up as a change in P wave size and PR interval.

SINUS BRADYCARDIA

Sinus bradycardia is defined as a sinus rate below 50 bpm with otherwise normal conduction (Fig. 3.2). It occurs mainly in patients taking beta-blockers but is seen also in hypothyroidism, obstructive jaundice and raised intracranial pressure. It is physiological in athletes and is a feature of chronotropic incompetence.

SINUS TACHYCARDIA

Sinus tachycardia is defined as a sinus rate over 100 bpm. It is normal in childhood. In adults it is a physiological response to exercise or a reaction to numerous insults such as fever, anemia, hyperthyroidism, anxiety or heart failure. It can occur in response to certain drugs such as amlodipine, nifedipine, sympathomimetic agents, atropine, adrenaline or isoprenaline.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_3,

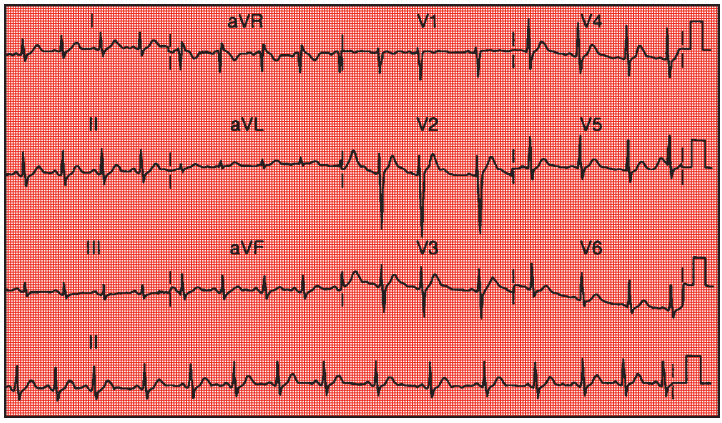


FIG. 3.1. Young medical student with sinus arrhythmia, seen at the bottom rhythm strip in lead II. Acceleration and deceleration of the heart are related to respiration. This is a normal finding in young adults; it is said to have pathological connotations later in life (MS; 28/5/99).

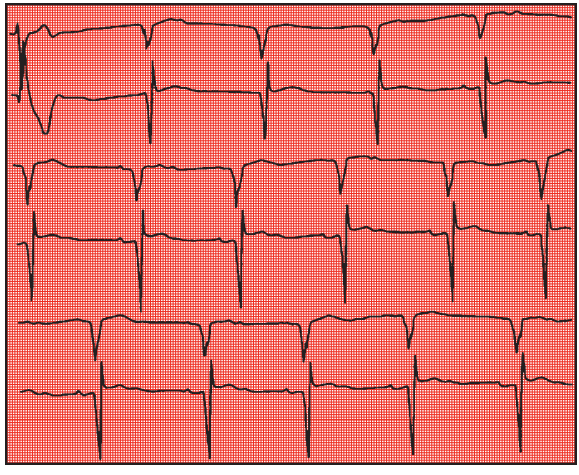


FIG. 3.2. A 90-year-old patient with marked sinus bradycardia and dizzy spells. Twenty-four hour Holter ambulatory recording was performed on two channels, the lower one showing the P waves more clearly. The upper strip shows a ventricular rate of 36 bpm, suggesting complete heart block. The last two complexes in the second strip confirm independent P waves (LC; 7/12/99).

SINOATRIAL DISEASE

Sinoatrial slowing can be physiological (for example in athletes) or acquired in response to old age (fibrosis), atheromatous disease or certain drugs (such as digoxin, beta-blockers, verapamil and amiodarone).

Definitions of sinoatrial disease are listed below.

- Sinoatrial block: a non-conducted P wave (not to be confused with an atrial premature beat arising in a refractory period).
- Sinoatrial arrest: a complete PQRST cycle is missing.
- Sinoatrial tachycardia (within the SA node): this can be caused by a localized re-entry circuit (rare).
- Chronotropic incompetence: represents the inability to increase the heart rate in response to exercise, caused by slowing down of the SA node. Seen in elderly individuals.
- Sick sinus syndrome: also known as tachy/brady syndrome.

Sinoatrial disease characterized by episodes of intermittent bradycardia, atrial fibrillation, atrial flutter and supraventricular tachycardia.

Changes characteristic of sinoatrial disease are illustrated in Figs. 3.3–3.9.

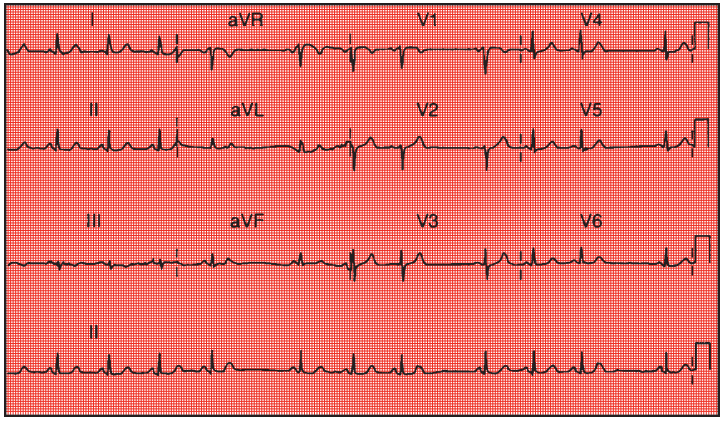


Fig. 3.3. A 48-year-old female patient with sinoatrial arrest. Rhythm strip lead II at the *bottom* shows complete absence of an entire PQRST complex (CM; 7/8/98).

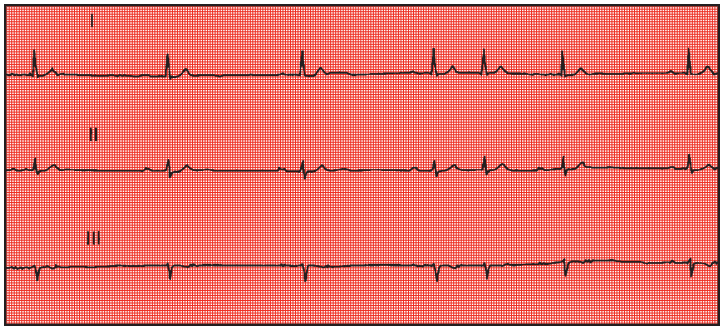


FIG. 3.4. A 57-year-old man with profound sinus bradycardia due to sinoatrial disease. The fifth complex is a junctional escape beat (Mr. R; 3/4/97).

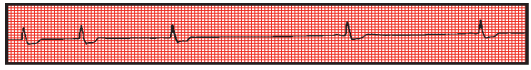


FIG. 3.5. This elderly woman developed profound sinus bradycardia after brainstem infarction (21/12/96).

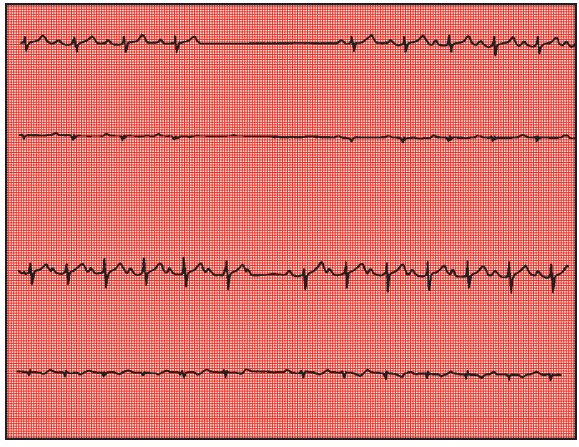


FIG. 3.6. A 33-year-old woman with sinoatrial disease. The upper strip shows sinus arrest. The bottom strip (in the *middle*) shows sinoatrial block. (DL; 6/11/96).

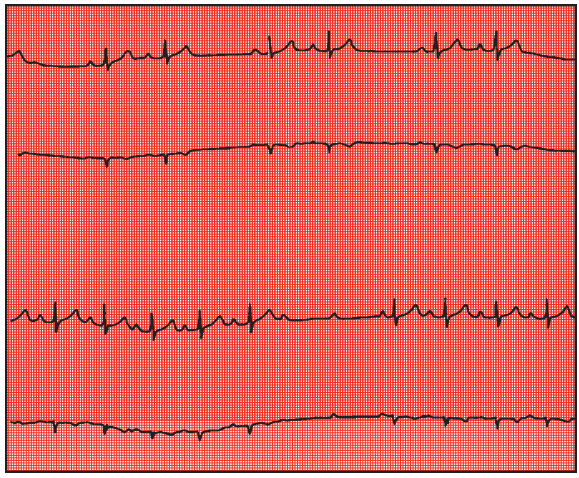


FIG. 3.7. The same patient. The upper strip shows sinoatrial arrest. The bottom strips shows a short spell of complete heart block. This is an unstable situation requiring permanent pacing (DL; 6/11/96).

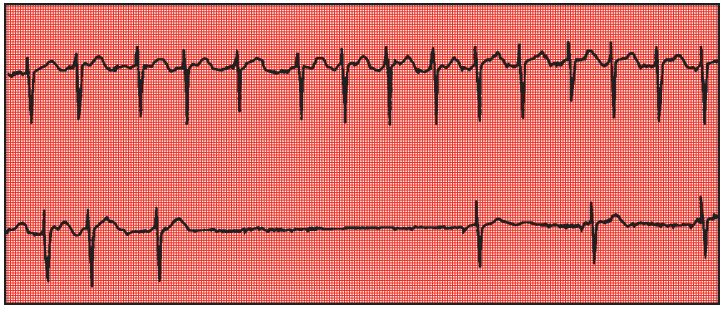


FIG. 3.8. Sinoatrial disease. Elderly patient with ischaemic heart disease. There is sinoatrial arrest with a long pause. This patient required a pacemaker. As a rule, pacing is indicated when pauses are longer than 3 s in duration.

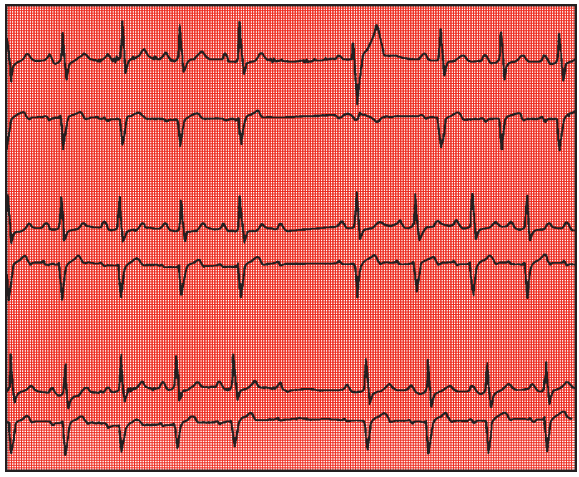


FIG. 3.9. Sinoatrial block. P waves are not conducted. The trace at the top shows sinoatrial arrest. In the *middle* there is sinoatrial block (BB; 3/8/95).

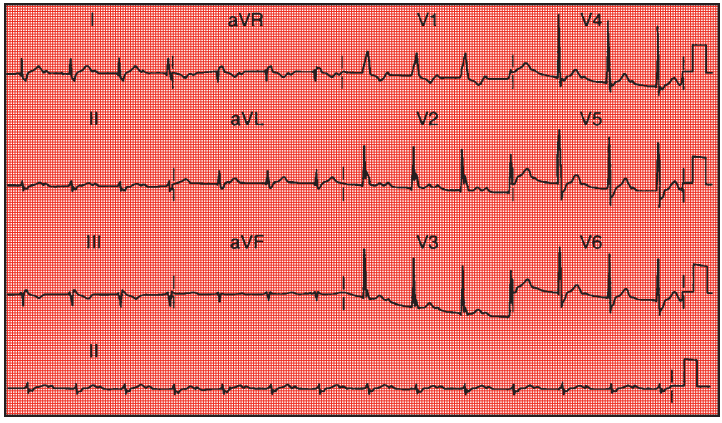


FIG. 3.10. This 80-year-old patient presented with dizzy spells. He had first degree heart block. There is a prolonged PR interval, which can be seen clearly in leads V1 and V2. There is in addition right bundle branch block, left axis deviation, hence trifasicular block (MC; 19.2.95).

ATRIOVENTRICULAR BLOCK

First Degree AV Block

Prolonged PR interval over 0.2 s (200 ms) is usually seen in the elderly, associated with coronary artery disease or acute rheumatic carditis or caused by certain drugs such as digoxin (Figs. 3.10–3.15).

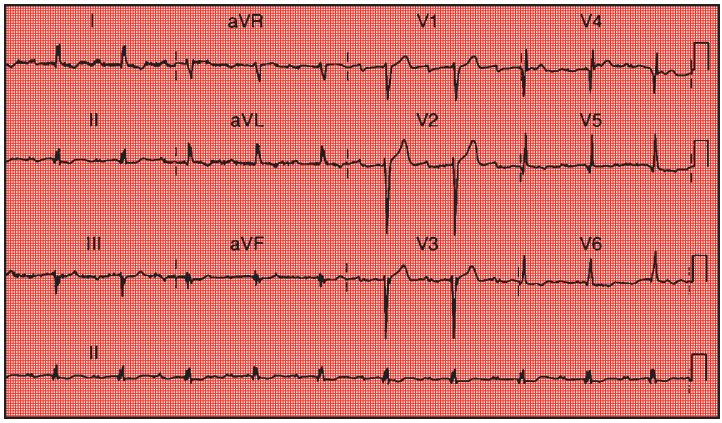


FIG. 3.11. Ischaemic heart disease. First degree heart block is indicated by the prolonged PR interval (WN; 17/2/99).

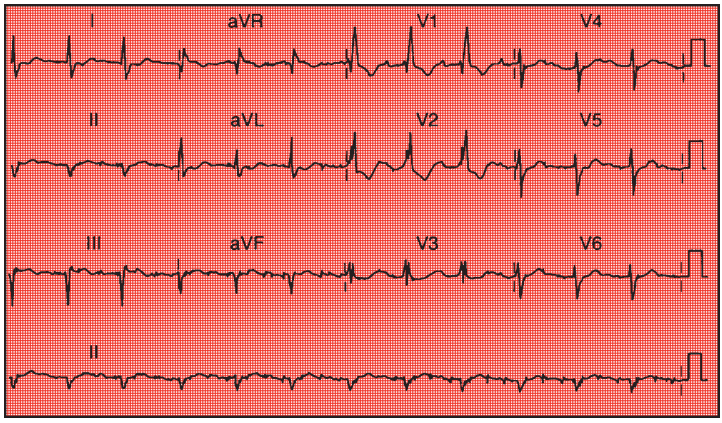


FIG. 3.12. This patient in his 80s has ischaemic heart disease. The ECG indicates first degree heart block. There is a right bundle branch block and left axis deviation (i.e., trifascicular block) (JK; 21/9/98).

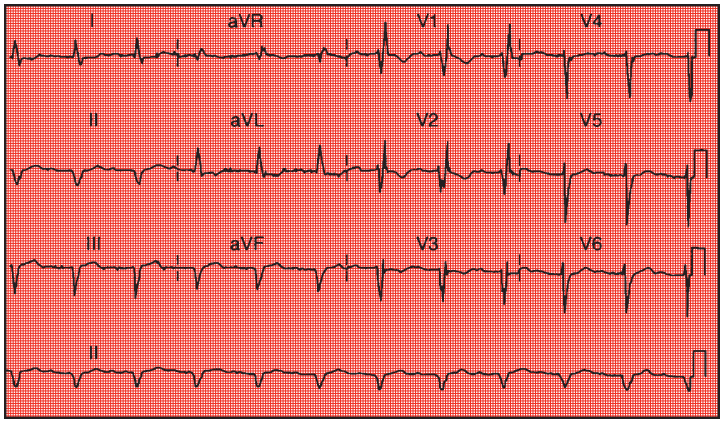


FIG. 3.13. Some months later, the first degree heart block is worsening (JK; 25/5/99).

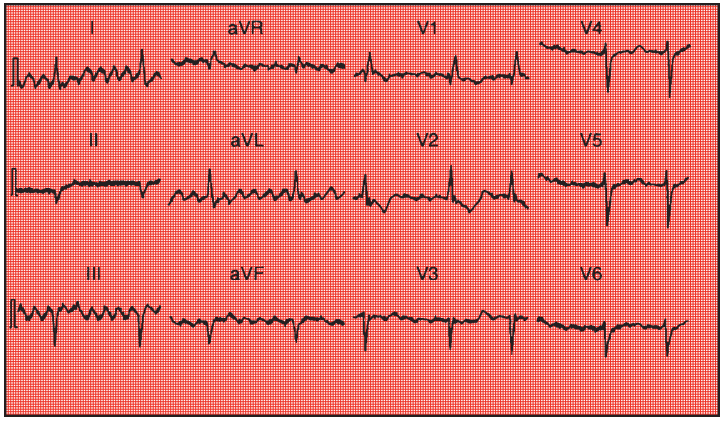


FIG. 3.14. After a further 6 months, the patient went into atrial fibrillation (JK; 23/11/99).

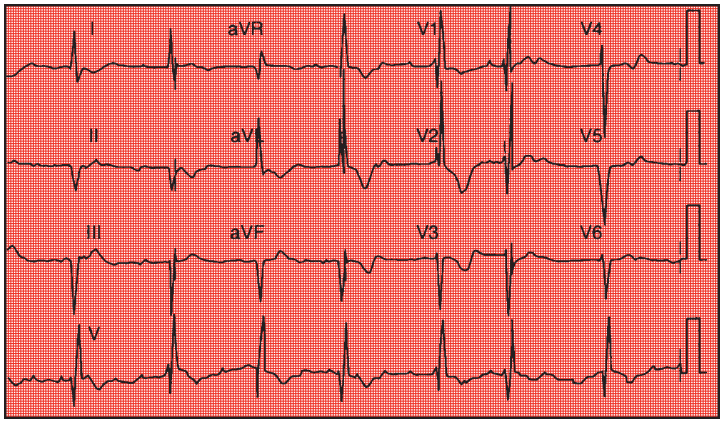


FIG. 3.15. Five days later, there is complete heart block. The P waves are independent from the QRS complexes (JK; 28/11/99).

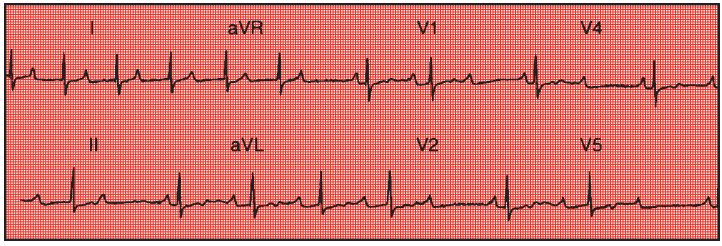


FIG. 3.16. Continuous strip showing episodic Wenckebach (Mobitz I) second degree heart block. The PR interval becomes prolonged in both strips and a ventricular complex is then dropped. The P wave is not conducted and the cycle restarts (Mr. P; 29/3/97).

Second Degree AV Block

There are two types:

- *Mobitz I (Wenckebach):* the PR interval is prolonged with each beat until an entire cycle is dropped (Figs. 3.16 and 3.17). It can occur in athletes, the elderly and people with coronary artery disease. When associated with acute myocardial infarction, it may require temporary pacing.
- *Mobitz II:* regularly occurring non-conducted P waves (Figs. 3.18 and 3.19). The aetiology is the same as that of Mobitz I heart block.

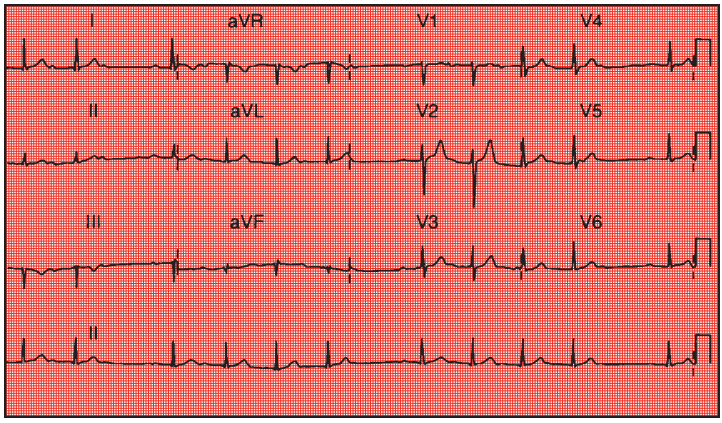


FIG. 3.17. A further example of Wenckebach phenomenon. The bottom strip (II) shows the PR interval to be prolonged. The complex is dropped due to a blocked P wave (DL; 3/2/97).

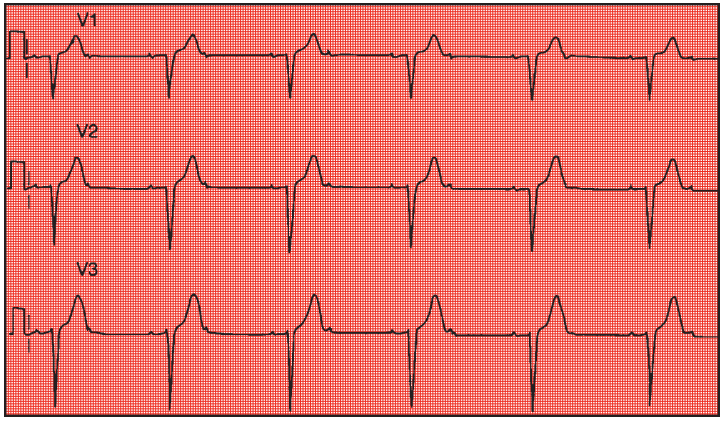


FIG. 3.18. An 81-year-old lady with second degree heart block (Mobitz II). Each second P wave is not conducted (EL; 12/3/96).

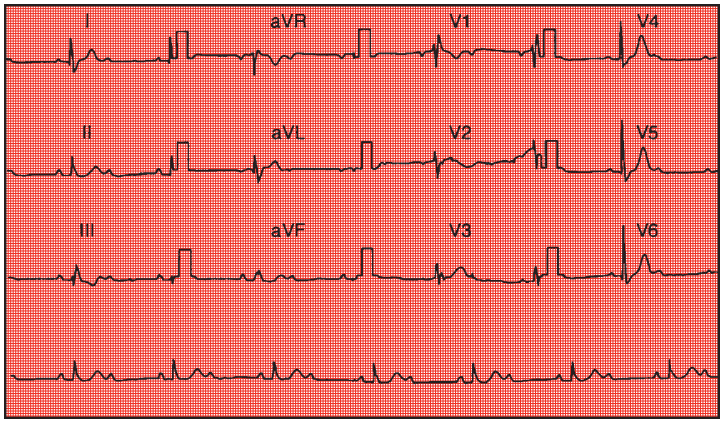


FIG. 3.19. Another example of second degree heart block (Mobitz II) (HG; 4/5/95).

Third Degree AV Block

P waves discharge at their inherent rate, but cannot be conducted to the ventricles (an example of AV dissociation). If the QRS complexes are narrow (high grade AV block) the heart block is usually at the level of the bundle of His. The ventricular rate will be of the order of 50 bpm. If the QRS complexes are wide, the block is lower down and the rate is between 30 and 40 bpm (Figs. 3.20 and 3.21). Adams–Stokes attacks refer to syncope caused by impaired cerebral circulation related to extreme bradycardia or ventricular arrest (Figs. 3.22–3.29).

Management

- Atropine is of no use.
- Give isoprenaline by mouth or intravenous infusion.
- A temporary pacemaker can be useful, particularly after acute myocardial infarction.
- A permanent pacemaker may be necessary (Chap. 8).
- Use an external pacemaker when other methods are unavailable.

Predisposing Conditions

Conditions that predispose to third degree AV block include acute MI, calcified aortic valve disease, bifascicular and trifascicular block, cardiac surgery and occasionally drugs (beta-blockers and cocaine); also fibrotic changes relating to aging.

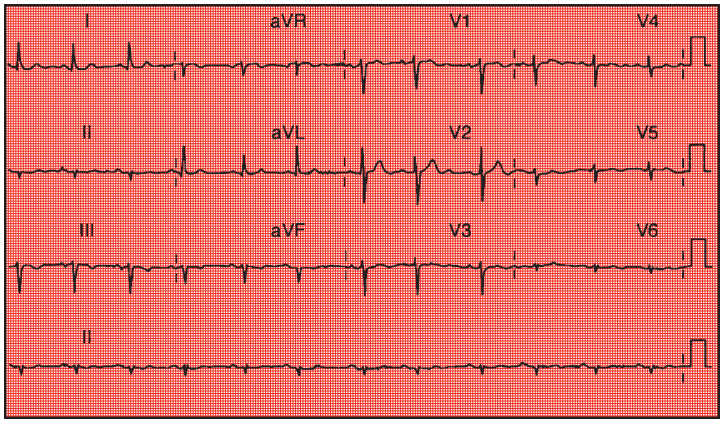


FIG. 3.20. This 70-year-old woman with ischaemic heart disease had normal sinus rhythm in July 1988 (QH; 28/7/98).

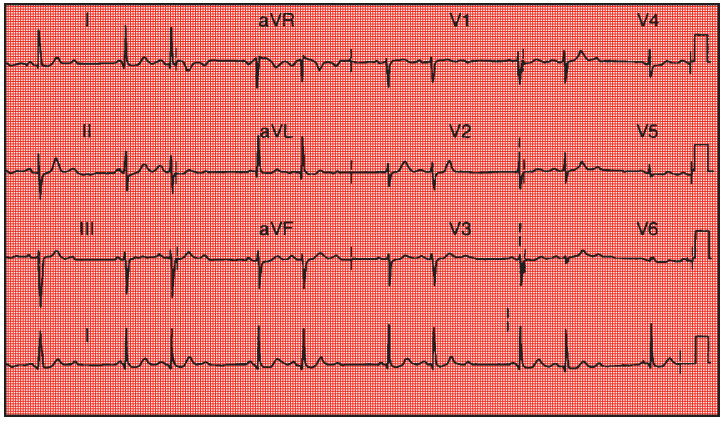


FIG. 3.21. Sinoatrial block was evident 1 month later (QH; 5/8/98).

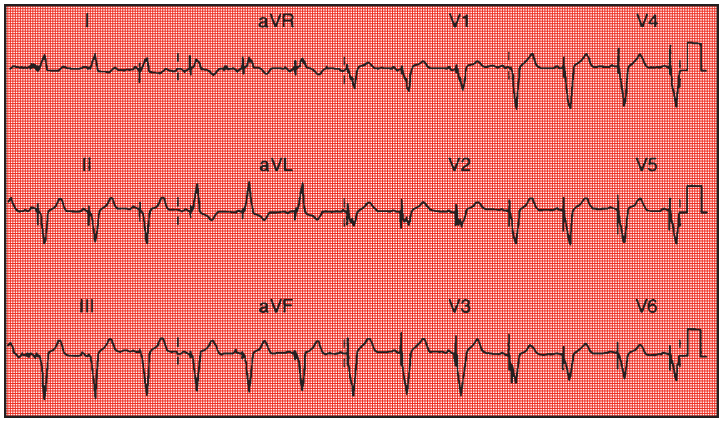


FIG. 3.22. Two days later the heart is pacing adequately (VDD). The AV sequential contraction is maintained through a single path lead. The electrode "floating" in the right atrium senses the atrial P wave and sends a signal to the right ventricle, which is then paced (QH; 7/8/98).

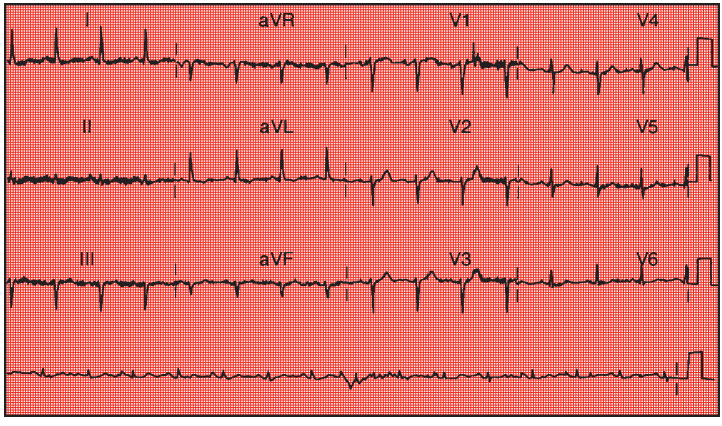


FIG. 3.23. Normal sinus rhythm (WP; 23/7/93).

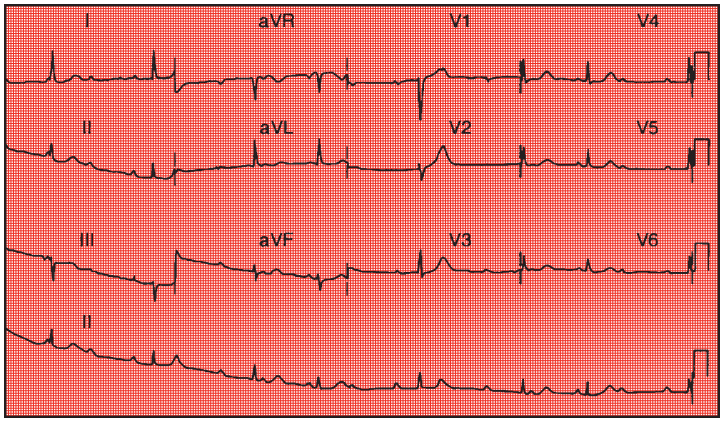


FIG. 3.24. The same patient, 5 years later, has complete heart block. The bottom strip (lead II) shows P waves appearing at a normal rate. These are not conducted to the ventricles, which are beating independently at a rate of 40 bpm (WP; 26/11/98).

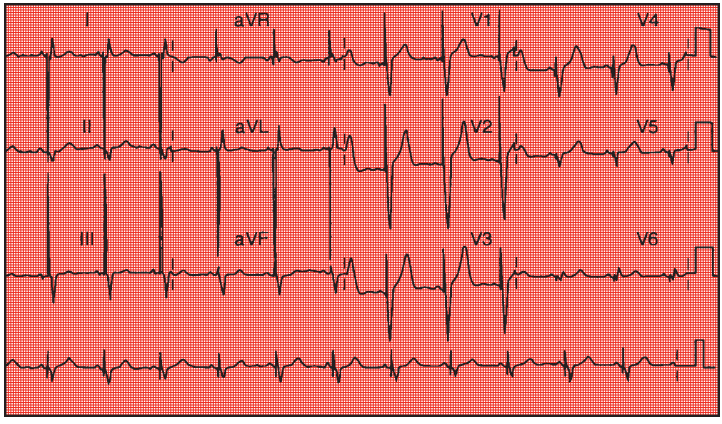


FIG. 3.25. The same patient with AV sequential pacing. Each P wave is followed by a paced signal (WP; 27/11/98).

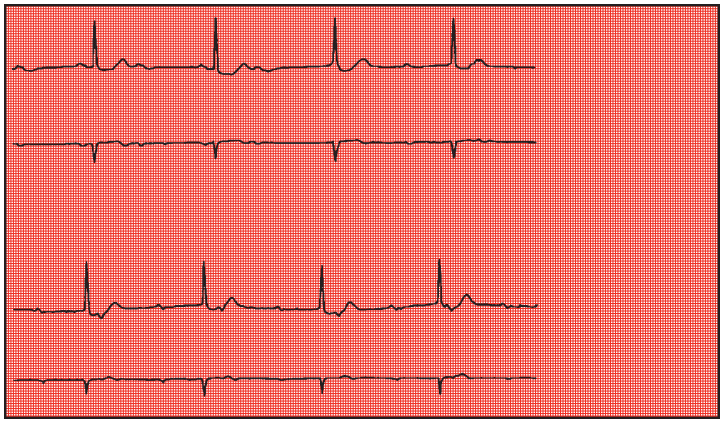


FIG. 3.26. Complete heart block on 24-h Holter monitor. The ventricular rate is 34 bpm. The atria are beating independently at a faster rate (SW; 11/7/96).

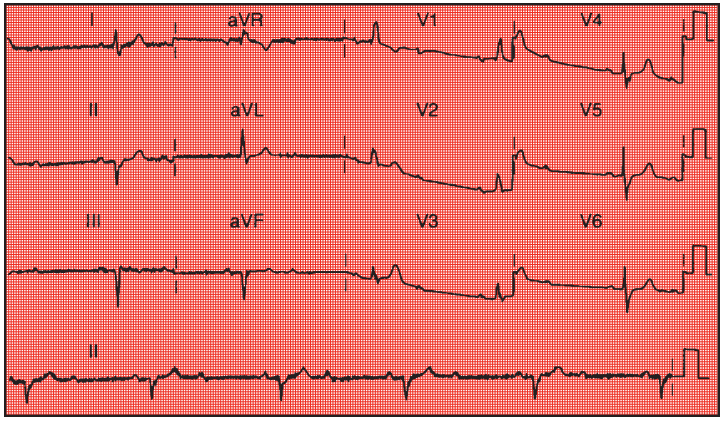


FIG. 3.27. This elderly man presented with second degree (Mobitz II) heart block (SJ; 8/5/95).

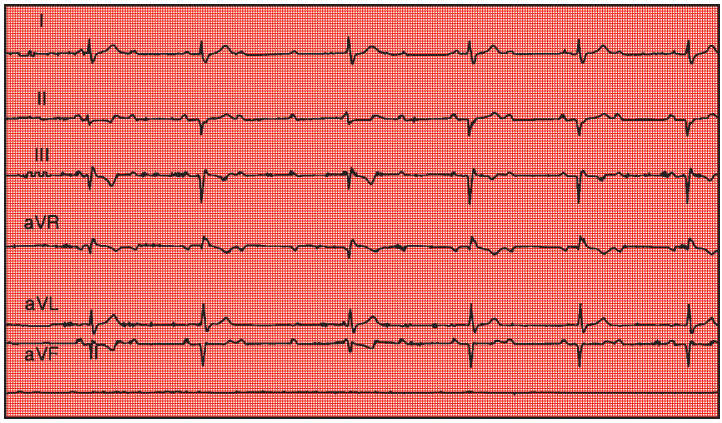


FIG. 3.28. The condition rapidly proceeded to complete heart block (SJ; 8/5/95).

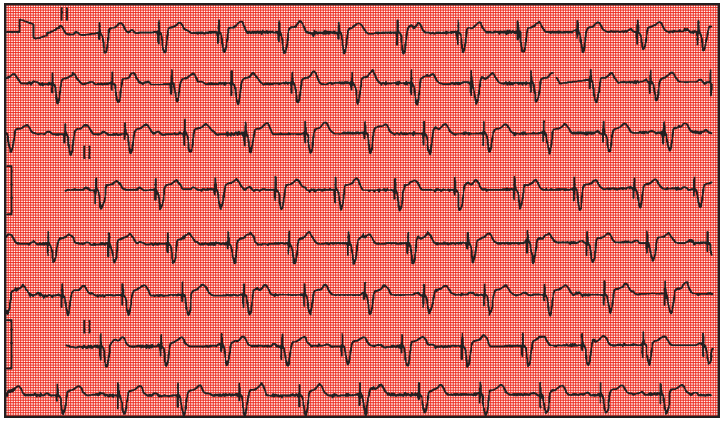


FIG. 3.29. VV1 packing in the same patient. The right ventricle is paced independently of P wave activity, which is seen to be unrelated to the pacing signals (SJ; 15/5/95).

CONGENTIAL COMPLETE HEART BLOCK

Congenital complete heart block is usually seen in children. The ventricular rate is around 60 bpm. Syncopal attacks are rare, but they do occur and permanent pacing may be required.

BUNDLE BRANCH BLOCK

Normal Conduction

Having reached the bundle of His, situated in the upper portion of the interventricular septum (IVS), the electrical impulse is transmitted to the bundles, supplying the right and left ventricles (right and left bundles). The right bundle is single and the left divides into two: the superior or anterior branch, and the inferior or posterior branch (Fig. 3.30).

Right Bundle Branch Block

Right bundle branch block (RBBB) occurs when there is an interruption of the right bundle below the bundle of His. The right ventricle is now electrically stimulated from the left ventricle (Fig. 3.31).

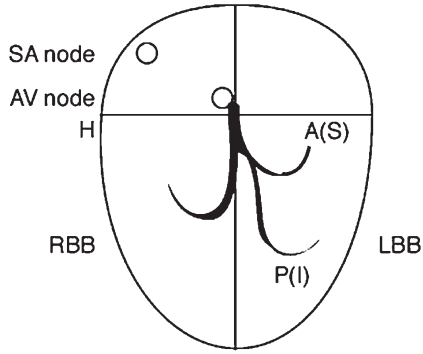


FIG. 3.30. Conduction system: The sinoatrial node discharges at the inherent rate of around 70 bpm in adults (faster in children). It is influenced by neurogenic and hormonal input. The impulse is filtered down the AV node, enters the bundle of His and then travels to both right and left bundle branches. The left divides into an anterior (superior) and inferior (posterior) branch and thence ramifies through the Purkinje system to all parts of the ventricles (from endocardium to epicardium). *SA node* sinoatrial node; *AV node* atrioventricular node; *H* His; *RBB* right bundle branch; *LBB* left bundle branch; *A* anterior; (*S* superior); *P* posterior (*I* inferior).

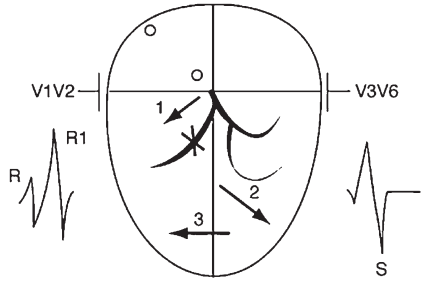


FIG. 3.31. Right bundle branch block. Vector 1 represents septal activation from left to right giving rise to a positive deflection in leads facing the right ventricle (R wave). Vector 2 represents left ventricular activation, that is, a negative wave (S wave) and Vector 3 represents late right ventricular activation (R1). These occur in electrodes facing the right ventricle. By contrast, left ventricular facing leads (1, aVL, V5, V6) will demonstrate a late negative S wave reflecting the delayed right ventricular depolarization. Complete right bundle branch block refers to a QRS complex wider than 0.10 s (100 ms). Incomplete right bundle branch block occurs as above, but the QRS complex is less than 0.10 s (100 ms) in duration.

In complete RBBB the QRS complex measures more than 0.10 s (100 ms). In partial or incomplete RBBB the QRS complex duration is less than 0.10 s (100 ms). "M" shaped complexes are seen in leads V1–V2.

Right bundle branch block can be congenital and of no clinical importance. If acquired it bears a different connotation, indicating right ventricular pathology (ischemia, pulmonary hypertension, pulmonary embolism, or congenital conditions such as ASD).

Left Bundle Branch Block

Left bundle branch block (LBBB) occurs when there is interruption of the left bundle below the bundle of His, affecting both anterior and inferior divisions.

In left bundle branch block the QRS complex duration is usually over 1.2 s (120 ms). Left bundle branch block generally indicates left ventricular disease

(such as hypertension, ischemia, or valvar disease). Rarely, it can be congenital. "M" shaped complexes are seen in leads V5–V6 (Fig. 3.32). When the QRS complex is under 120 ms it is called incomplete LBBB.

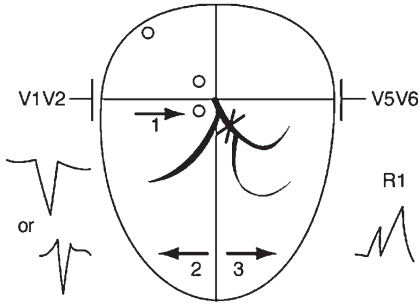


FIG. 3.32. Left bundle branch block. Septal activation originates from the right bundle (1) showing an initial positive deflection in left ventricular facing leads. Right ventricular activation (2) is directed away, hence a negative S wave is produced. Vector 3 is caused by delayed LV depolarization and is positive (RSR^1). In the right ventricular facing leads it is usual to see a deep negative (Q) wave reflecting septal and late LV activity. Occasionally, if the right ventricle is activated earlier than the septum, a small positive R wave is seen.

In both right and left bundle branch block the ST/T wave pattern is inscribed in the opposite direction from the main QRS vectors. They do not indicate an underlying pathology.

Stress testing can be undertaken in patients with right bundle branch block. With left bundle branch block the ECG is not readily interpretable. Right bundle branch block with myocardial infarction will show an initial negative (Q) wave. Left bundle branch block with myocardial infarction is generally undiagnosable, although expertise may detect minor relevant abnormalities.

Hemiblocks

Anterior Hemiblock

In anterior hemiblock there is interruption of the superior left bundle characterized by *left axis deviation (LAD)*, the electrical impulse stimulating the left ventricle from below (Fig. 3.33).

Inferior Hemiblock

Inferior hemiblock shows up as *right axis deviation (RAD)*, the impulse traveling downwards from the unaffected superior left bundle (Fig. 3.34).

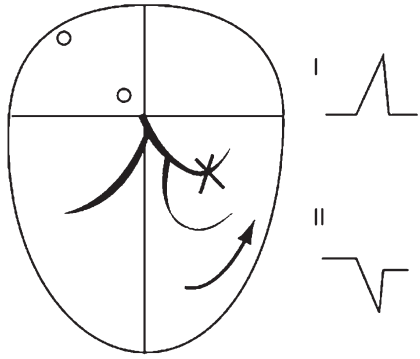


FIG. 3.33. Left anterior hemiblock. The upper portion of the left ventricle is stimulated from below, that is from the intact inferior bundle (LAD) (RAD).

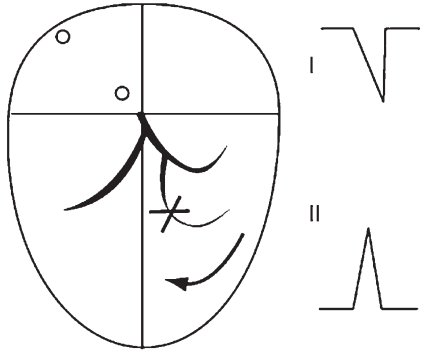


FIG. 3.34. Inferior hemiblock. The inferior bundle does not conduct. The signal travels downwards from the intact anterior bundle (RAD).

Bifascicular Block

Bifascicular block is right bundle branch block with either left or right axis deviation, or complete left bundle branch block. The former reflects interruptions through the right bundle on the one hand and the superior or inferior left bundle on the other. Complete left bundle branch block indicates interruption of both superior and inferior bundles. Progression to complete heart block is feasible, particularly in an acute situation such as myocardial infarction, necessitating pacing.

Trifascicular Block

Trifascicular block is the same as bifascicular block with additional prolongation of the PR interval (first degree AV block). Again, this can progress to complete heart block. These patients need careful supervision and are requested to report back in the event of dizzy spells or syncope. Examples from patients with bundle branch blocks are illustrated in Figs. 3.35–3.64.

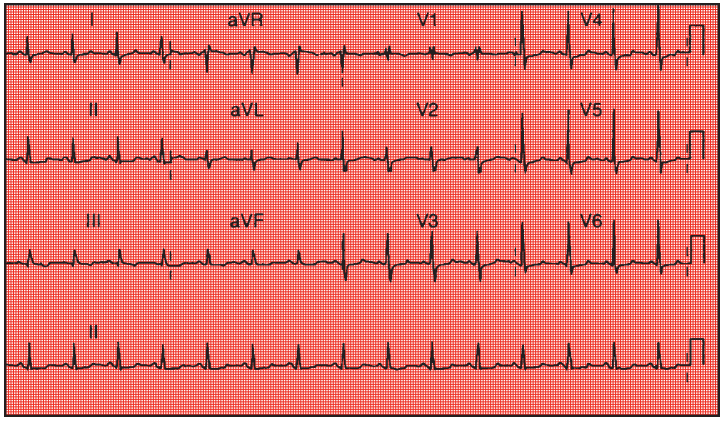


FIG. 3.35. Incomplete right bundle branch block (Mr. A; 20/6/97).

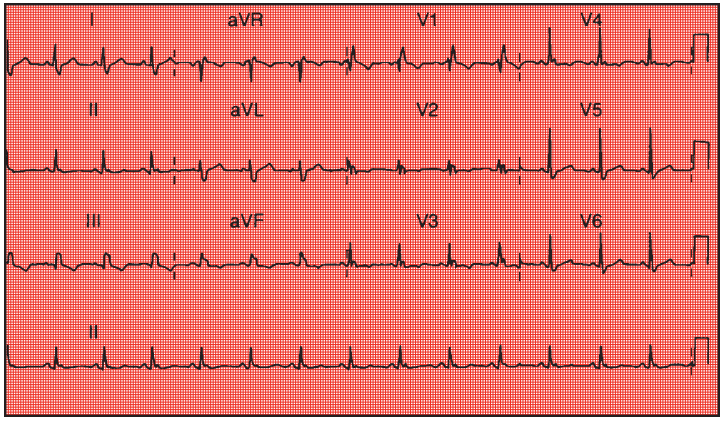


FIG. 3.36. Complete right bundle branch block develops 12 months later. The QRS complex is now more prolonged. There is a deeper, wider S wave in leads I, V5 and V6 (Mr. A; 8/6/98).

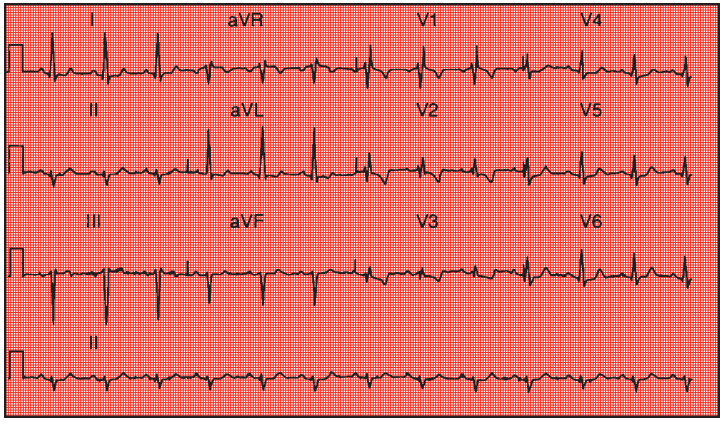


FIG. 3.37. Right bundle branch block with left axis deviation, constituting bifascicular block (ML; 19/3/99).

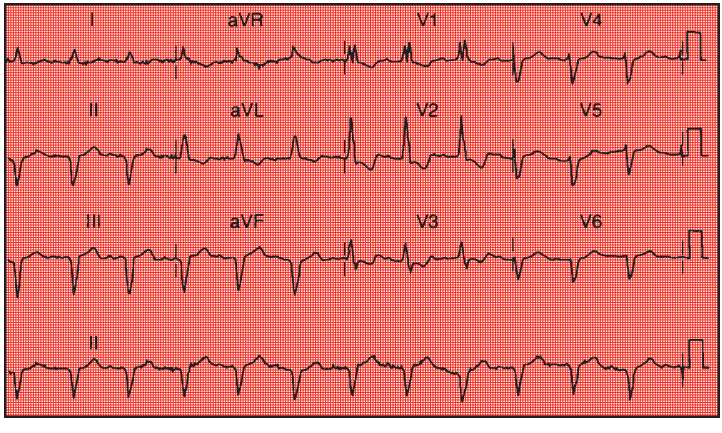


FIG. 3.38. Right bundle branch block with left axis deviation. There is a prolonged PR interval, constituting trifascicular block. The patient developed subsequent complete heart block requiring permanent pacing (LC; 23/11/99).

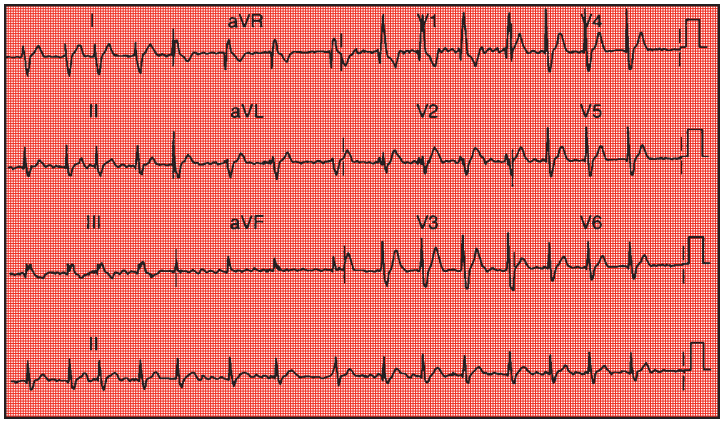


FIG. 3.39. Atrial fibrillation, right bundle branch block and right axis deviation, constituting a bifascicular block (SR; 12/11/99).

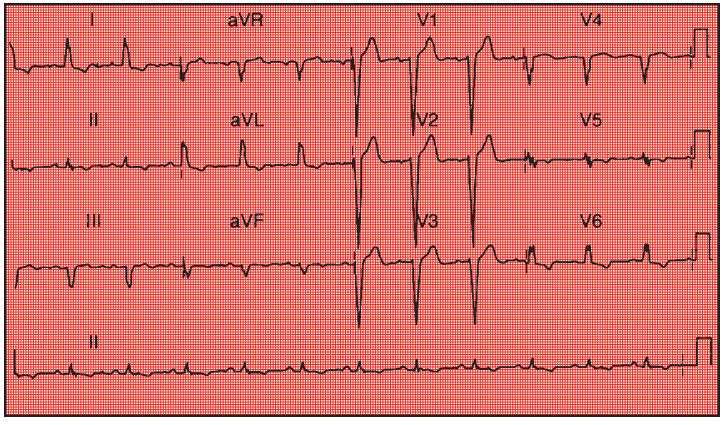


FIG. 3.40. Complete left bundle branch block. Normal axis with normal PR interval. There is bifascicular block since both left bundles are blocked.

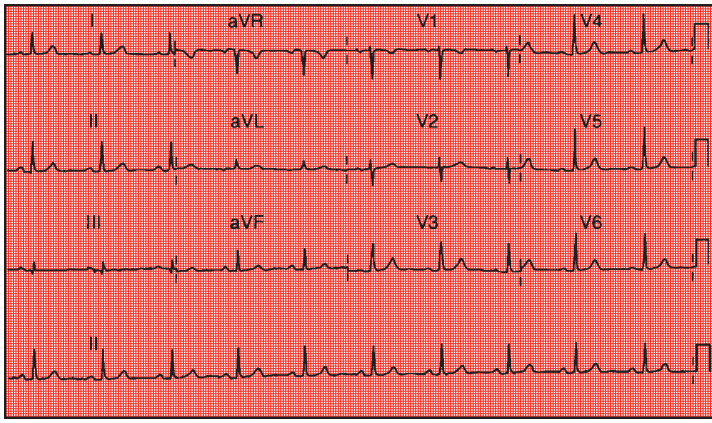


FIG. 3.41. Normal conduction in a middle-aged woman with mild aortic stenosis (ST; 27/8/97).

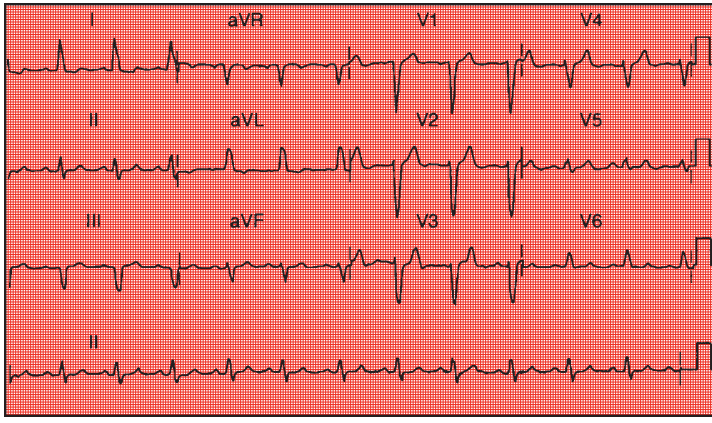


FIG. 3.42. Left bundle branch block is seen in the same patient 2 years and 3 months later. There is a normal axis and normal PR interval with no worsening of the aortic valve gradient. There is no evidence of ischaemic heart disease, indicating progressive AV conduction dysfunction (ST; 30/11/99).

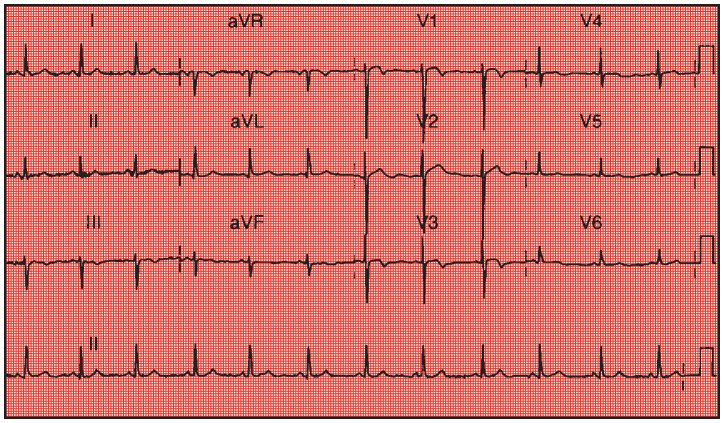


FIG. 3.43. This elderly woman with marked aortic stenosis has a fairly normal electrocardiogram with T wave inversions in lead V3 (JS; 3/6/96).

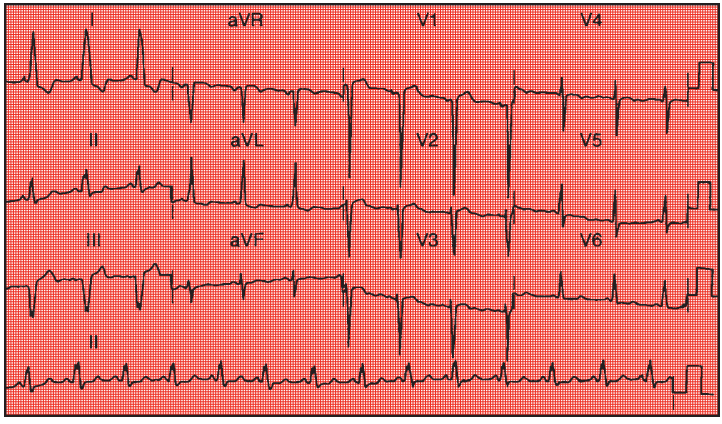


FIG. 3.44. Just over 2 years later there is now complete left bundle branch block. The patient underwent successful aortic valve replacement (JS; 10/7/98).

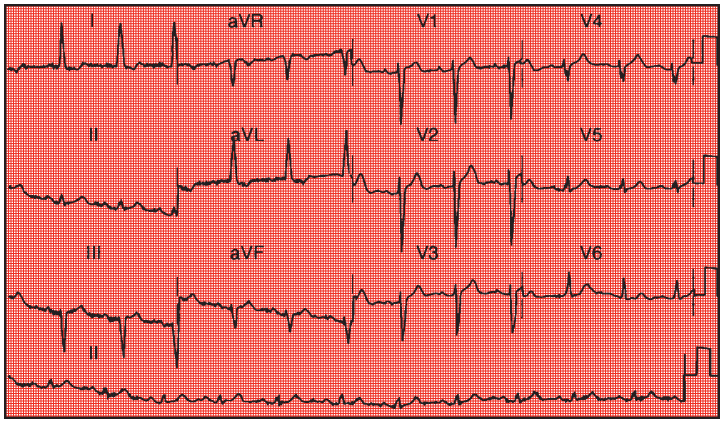


FIG. 3.45. ECG from an elderly woman with incomplete left bundle branch block (SR; 2/6/92).

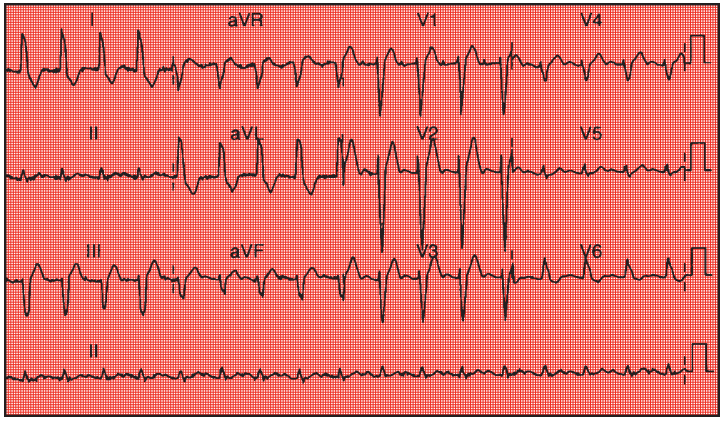


FIG. 3.46. After 7 years, the patient has progressed to complete left bundle branch block with first degree heart block. Same electric axis maintained. This is a trifascicular block (SR; 12/1/99).

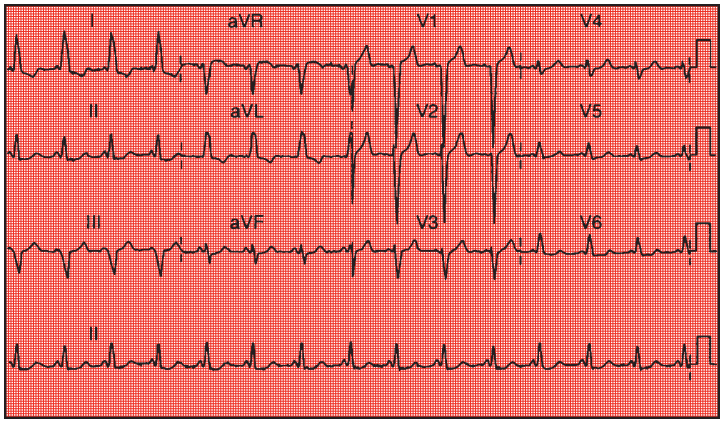


FIG. 3.47. The patient is an elderly woman, diagnosed with acute viral myocarditis. There is left ventricular failure and left bundle branch block (FO; 8/4/97).

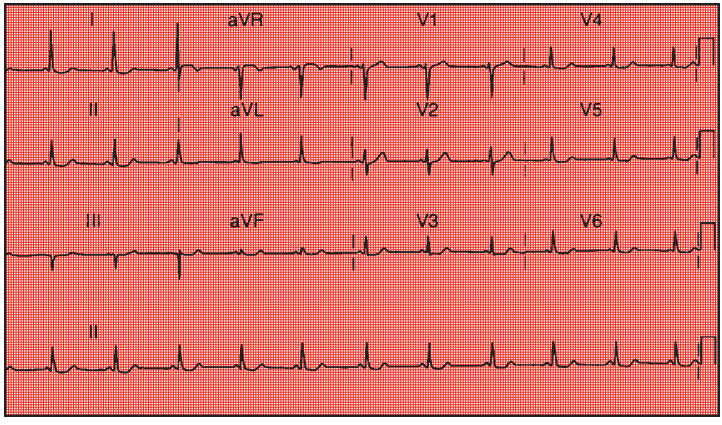


FIG. 3.48. The patient made a full recovery several months later. The ECG shows normal conduction, maintaining a short PR interval (FO; 9/7/97).

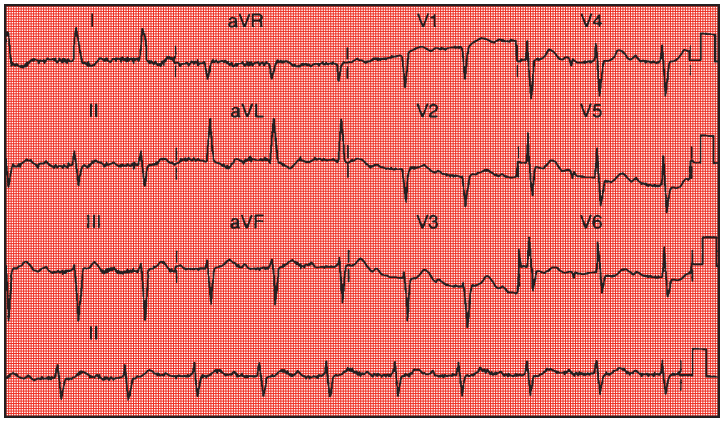


FIG. 3.49. ECG from a 77-year-old patient with ischaemic heart disease. There is an intraventricular conduction defect (widened QRS complexes) and a left bundle branch block pattern. Left axis deviation and prolonged PR interval are best seen in the rhythm strip at the bottom (MB; 3/6/98).

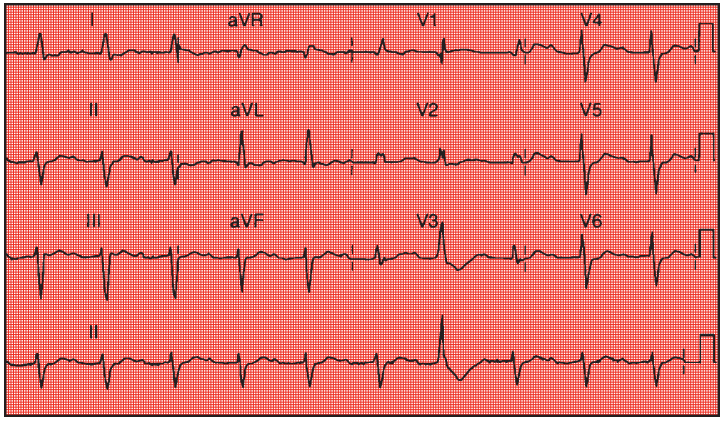


FIG. 3.50. Fifteen months later there is a prolonged PR interval and left axis deviation. The patient has developed right bundle branch block with clearly visible R1 waves in lead V2 and an S wave in lead I. This is an unusual situation and there is obviously still some conduction from the sinus node to the ventricles. The patient complained of dizzy spells (MB; 30/9/99).

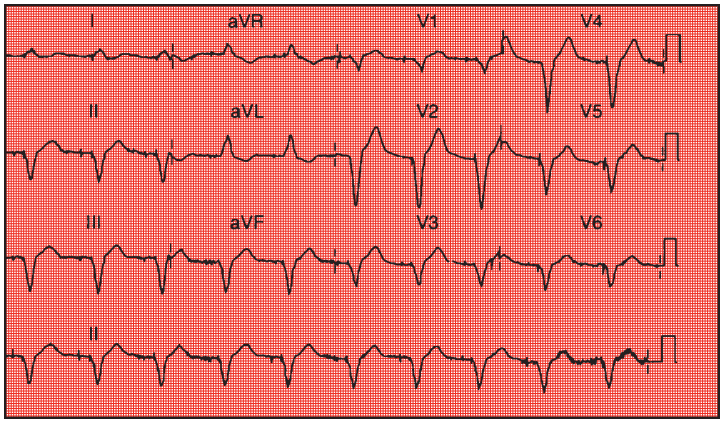


FIG. 3.51. The same patient following pacemaker implantation (DDD) (MB; 1/10/99).

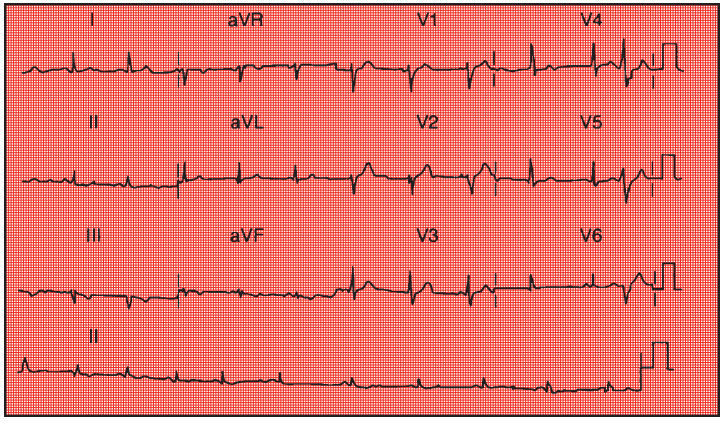


FIG. 3.52. Pre-bypass surgery ECG (SB; 4/2/87).

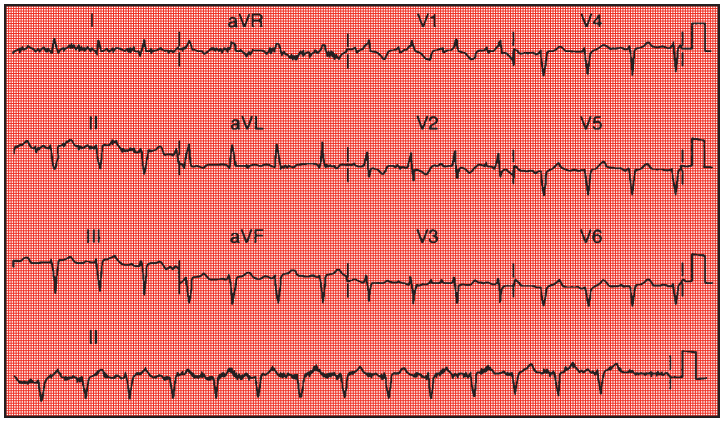


FIG. 3.53. Postoperative changes showing prolongation of PR interval, right bundle branch block and left axis deviation. This is an example of surgical trauma to the conducting system (SB; 20/2/87).

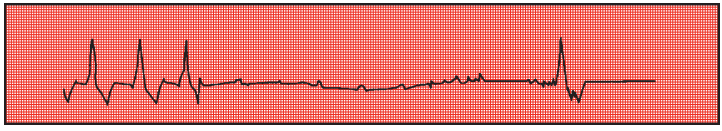


FIG. 3.54. The same patient developed ventricular asystole with only P waves visible. A pacemaker was implanted successfully (SB).

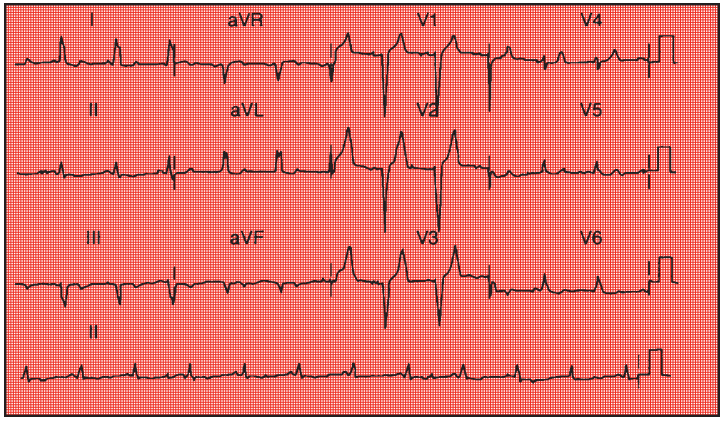


FIG. 3.55. Pre-bypass surgery ECG (AR; 26/3/86).

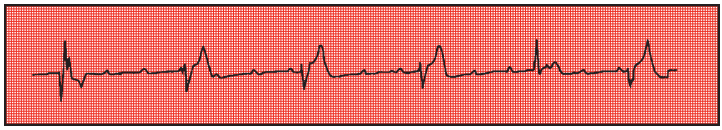


FIG. 3.56. The same patient developed complete heart block as a result of surgical trauma to the conducting system (AR; 29/3/86).

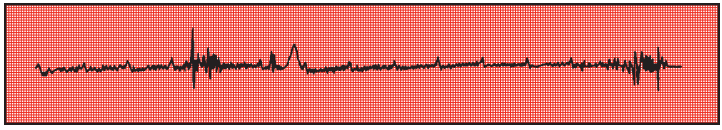


FIG. 3.57. This deteriorated to ventricular asystole. Only P waves are visible. There is only one QRS complex (escape beat). This patient survived pacemaker implantation (AR; 4/4/86).

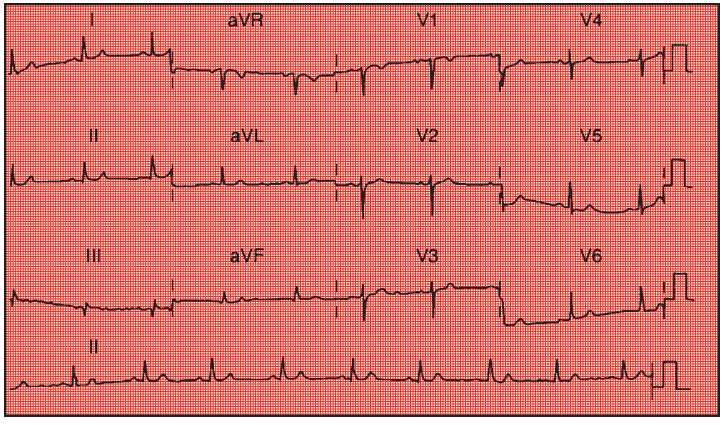


FIG. 3.58. Pre-operative (CABG) ECG, which is quite normal (FC; 20/11/87).

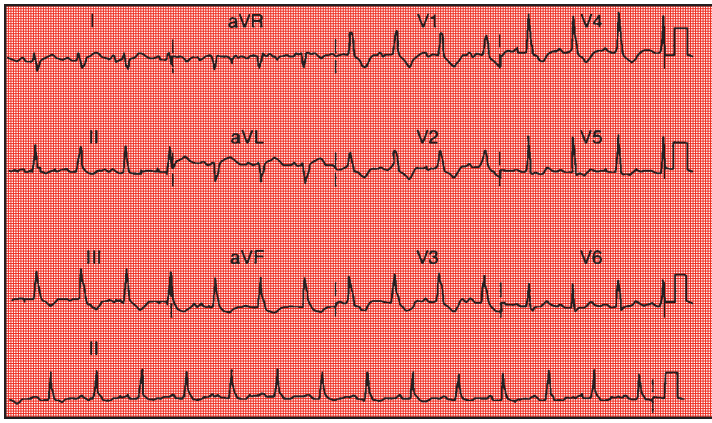


FIG. 3.59. Postoperative ECG showing right bundle branch block. There is a prolonged PR interval and right axis deviation, caused by surgical trauma to the conducting system (FC; 26/11/87).

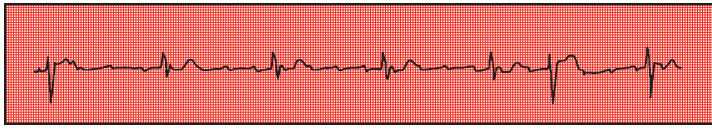


FIG. 3.60. The patient's condition progressed to complete heart block, which was treated by pacemaker insertion (FC; 30/11/87).

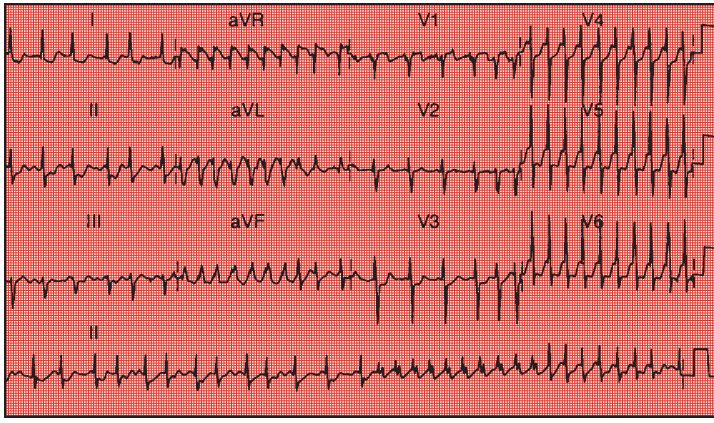


FIG. 3.61. ECG from an elderly woman with supraventricular arrhythmia showing marked ST depressions in the lateral leads with rate related bundle branch block developing towards the end of the rhythm strip (lead II) at bottom of the ECG. She was treated medically (Mrs. L; 6/5/93).

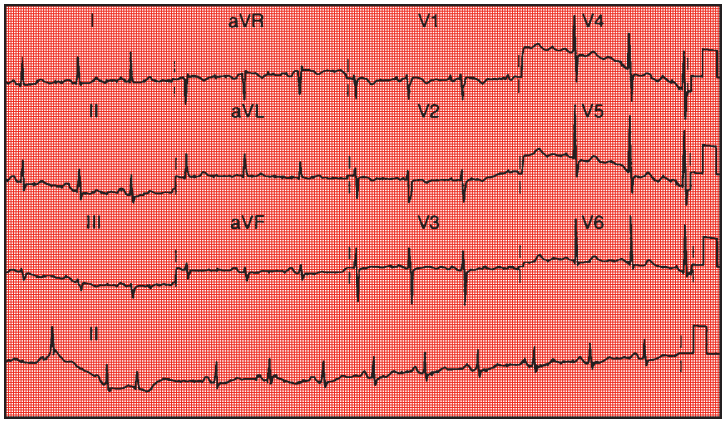


FIG. 3.62. The following day, the same patient had fully recovered. The bundle branch block is rate dependent due to "fatiguing" of a bundle. Subsequent coronary arteriography was quite normal. The ischaemic changes reflect underperfusion caused by an increased heart rate with otherwise normal coronary arteries (Mrs. L; 7/5/93).

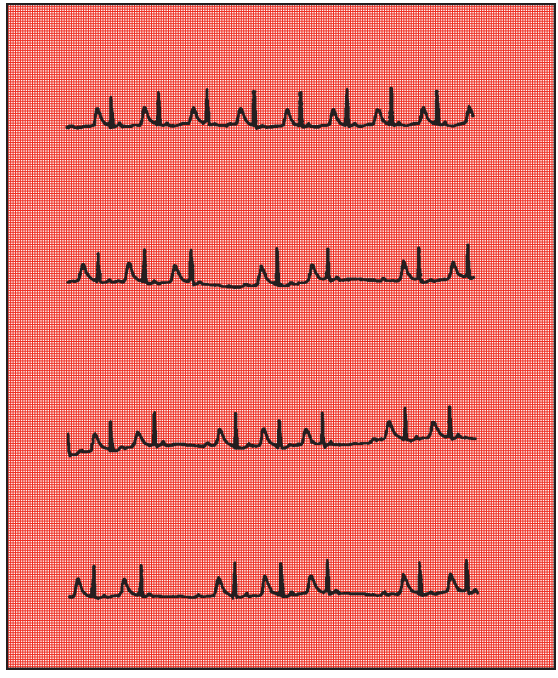


FIG. 3.63. Holter monitoring indicating sinoatrial block in a young, active "sporty" female who complained of palpitation. This ECG appearance is often seen in healthy young adults. No medication is indicated and the prognosis is favorable (Mrs. G; 23/12/97).

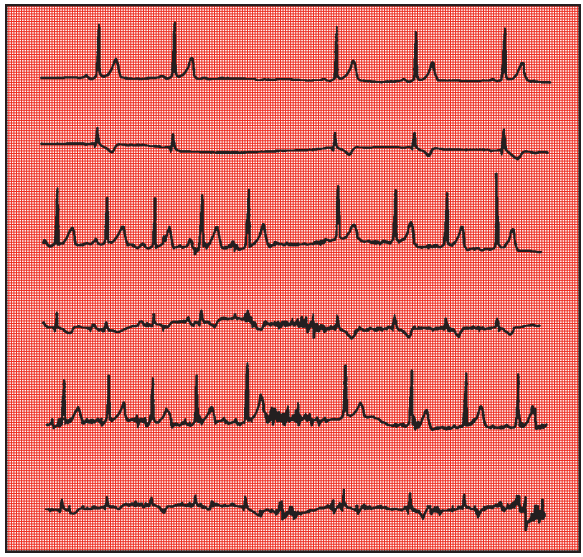


FIG. 3.64. Prolonged pause (2.53 s) caused by sinoatrial arrest in a young, athletic solicitor. As with the previous case, no medication is indicated and the prognosis is good (AB; 3/11/99).

CAROTID SINUS SYNDROME

Following the application of pressure, a hypersensitive carotid body can cause profound vagal activation, resulting in extreme cases in ventricular asystole. Normal carotid massage causes sinus bradycardia. The manoeuver is useful, occasionally, to terminate supraventricular arrhythmias, to bring in pacing activity when an increased heart rate inhibits the pacemaker setting and to slow down an atrial flutter allowing the flutter waves to be recognized more easily (Figs. 3.65–3.68).

PRE-EXCITATION CONDUCTION: ACCESSORY PATHWAYS

Wolff–Parkinson–White Syndrome

This is an important ECG pattern to recognize as it mimics a number of different conditions. An accessory (congential) bundle of Kent allows conduction from the atria to the ventricle resulting in early excitation (no AV nodal delay) of parts of the right or left ventricles (pre-excitation). The impulse travels down both the normal and abnormal pathways but moves at a faster rate down the latter. This pre-excitation results in a short PR interval.

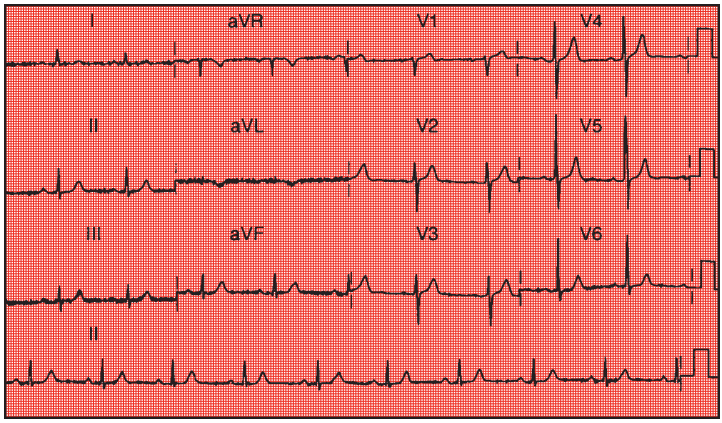


FIG. 3.65. Normal ECG from an elderly man with syncopal attacks attributed to cervical spondylosis (BH; 21/6/93).

FIG. 3.66. Right carotid massage caused ventricular asystole. The patient had no further symptoms after pacemaker implantation ↓ indicates right carotid massage (BH).

Having reached the ventricle through this abnormal pathway, the impulse has to travel through the myocardium as opposed to the normal conducting system. This slowed down impulse gives rise to a slurred delta wave on ECG. Wolff–Parkinson–White (WPW) syndrome can occur intermittently. It can be associated with rapid supraventricular arrhythmias (re-entry), atrial fibrillation or atrial flutter (no protection from AV nodal delay). The mechanism

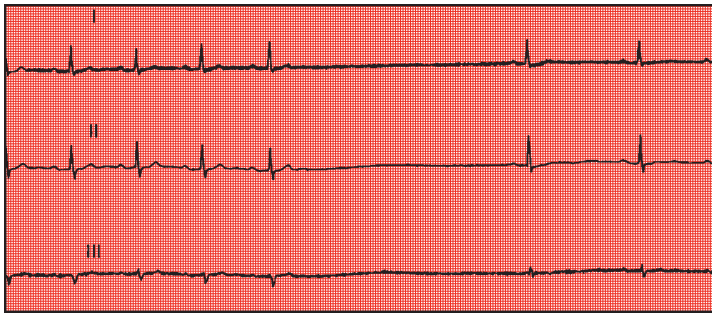


FIG. 3.67. ECG from a patient who presented with a CVA, with previous myocardial infarction. Right carotid massage caused profound bradycardia. The patient refused a pacemaker and eventually died (2/3/99).

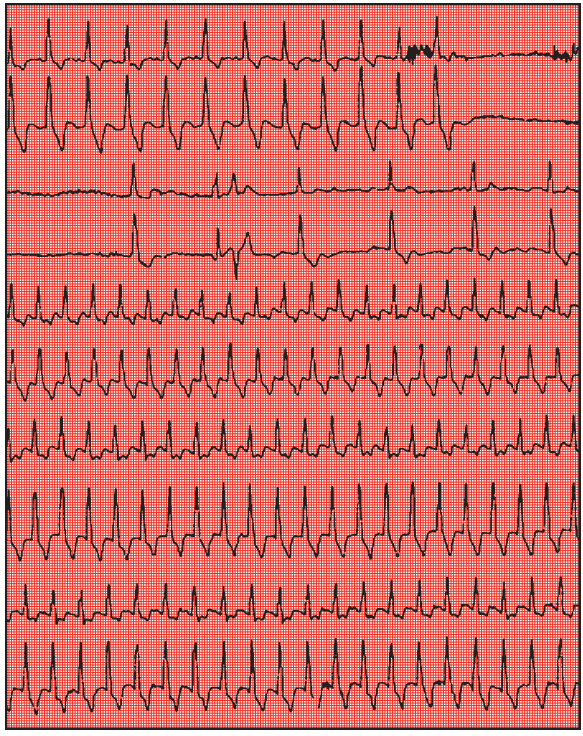


FIG. 3.68. This patient with ischaemic heart disease and bypass surgery reported numerous syncopal attacks. Repeated Holter monitoring was negative. On this occasion the patient was carrying a Holter when he had an argument with a taxi driver and collapsed. The top of the strip, which is continuous, shows the sinus tachycardia associated with aggravation, prolonged pause (cardiac arrest), slow recovery and subsequent accelerated rhythm (Mr. L; July 1994).

consists of a re-entry circuit, with the impulse moving down the normal pathway and re-entering the atria via the abnormal accessory pathway. Very fast supraventricular rates can ensue and rarely can progress to ventricular tachycardia and even ventricular fibrillation (Figs. 3.69–3.81).

Lown–Ganong–Levine Syndrome

The typical ECG has a short PR interval (under 0.10 s (100 ms)) without a delta wave. The presence of an accessory pathway can promote rapid re-entry supraventricular tachycardias. The accessory pathway ends distally within the AV node or the bundle of His. The impulse does not stimulate the myocardium (unlike WPW syndrome) and hence no delta waves are seen (Fig. 3.82).

LONG QT INTERVAL

This ECG sign is associated with syncope caused by ventricular tachycardia. It can cause sudden death. Two separate syndromes are recognized. Jervel–Lange–Nielsen syndrome, which is associated with deafness and Romano–Ward syndrome, which is familial and affects children.

Certain drugs prolong the QT interval. This can lead to ventricular arrhythmias (torsade de pointes), e.g., quinidine, disopyramide, procainamide, sotalol, amiodarone and proprafenone. Certain psychotropic drugs (phenothiazine, tricyclic antidepressants, lithium, and droperidol) and antihistamines (terfenadine) are known to prolong the QT interval and may cause sudden death.

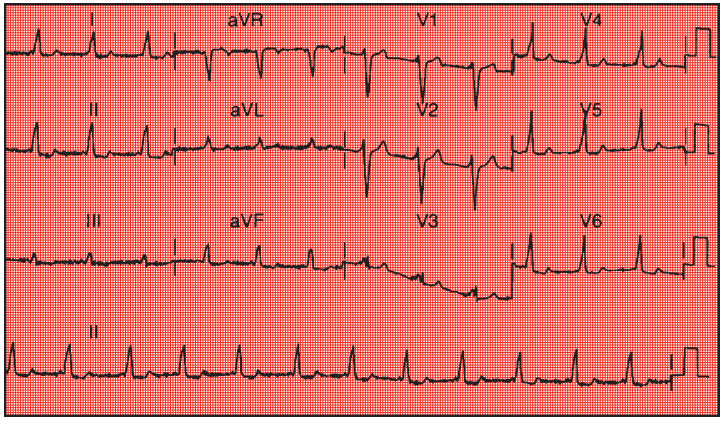


FIG. 3.69. Wolff–Parkinson–White syndrome, with a characteristic very short PR interval. Delta waves are best seen in leads V4–V6 (with slurring on upstoke of the R wave) (SR; 26/10/95).

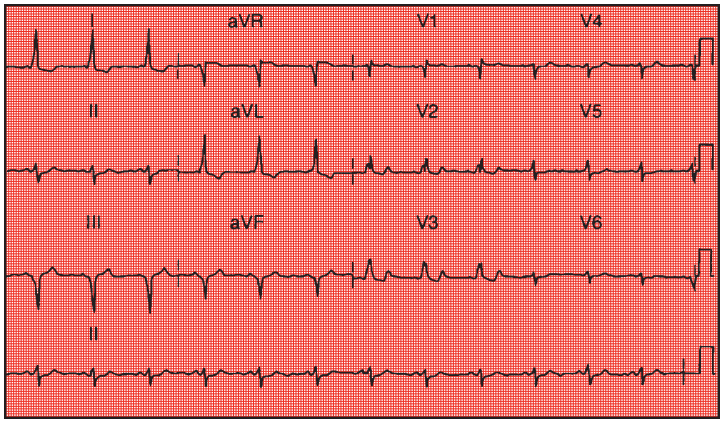


FIG. 3.70. WPW syndrome. There is a short PR interval, with delta waves in leads I and AVL. Note inferior Q waves. These do not indicate underlying myocardial infarction (PN; 15/2/96).

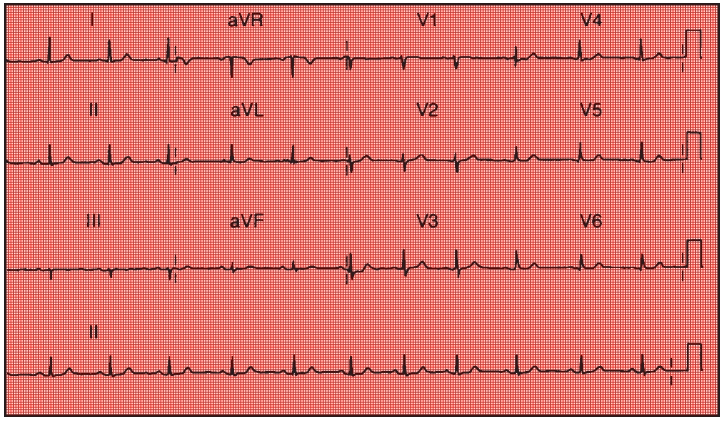


FIG. 3.71. The same patient, 2 years later after successful ablation. There is a normal PR interval with no delta or inferior Q waves. WPW can mimic infarction patterns (PN; 17/12/98).

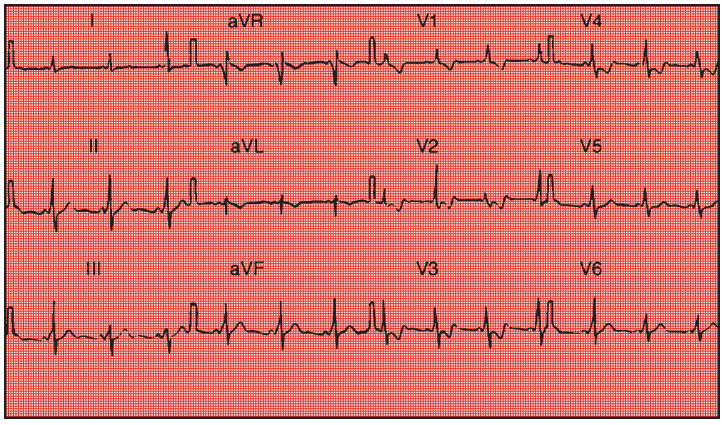


FIG. 3.72. ECG from a young man, showing classical WPW syndrome changes. There is a short PR interval and delta waves are well seen. In addition, there are abnormal waves, which could be misconstrued as a sign of ischaemic process (MS; 22/4/91).

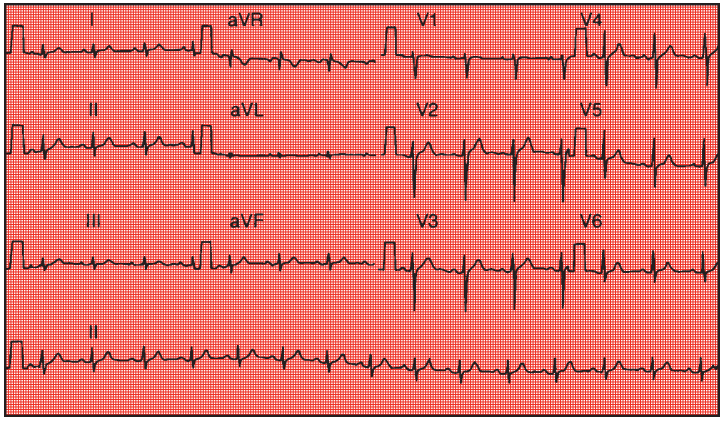


FIG. 3.73. After ablation of the accessory pathway, normality is restored (MS; 19/2/96).

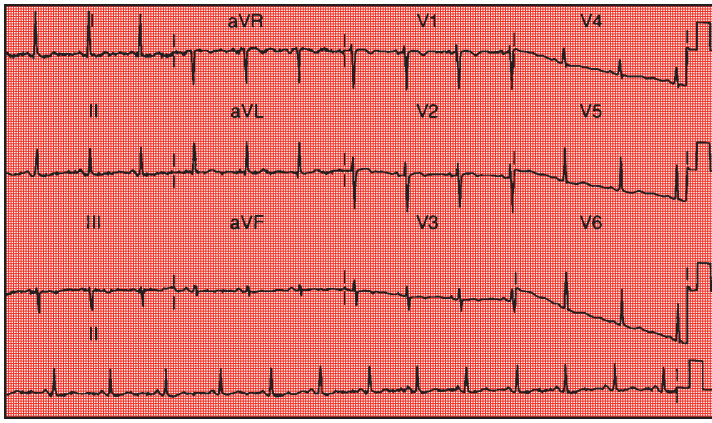


FIG. 3.74. Fairly normal ECG from a middle-aged woman. The precordial T wave inversions are of no significance (FJ; 16/2/95).

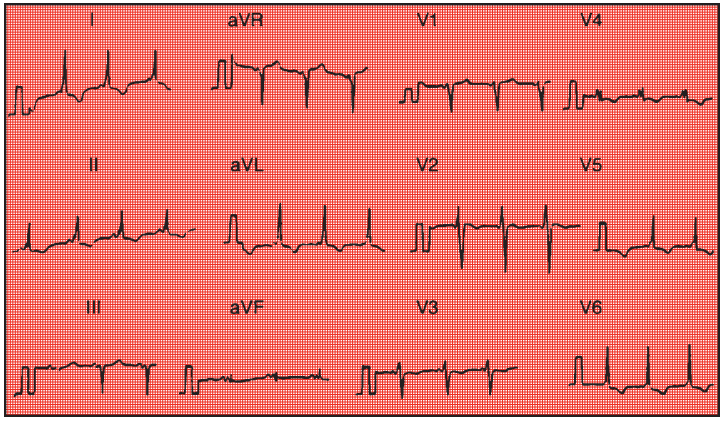


FIG. 3.75. One month later, the ECG indicates classical WPW syndrome (FJ; 8/3/95).

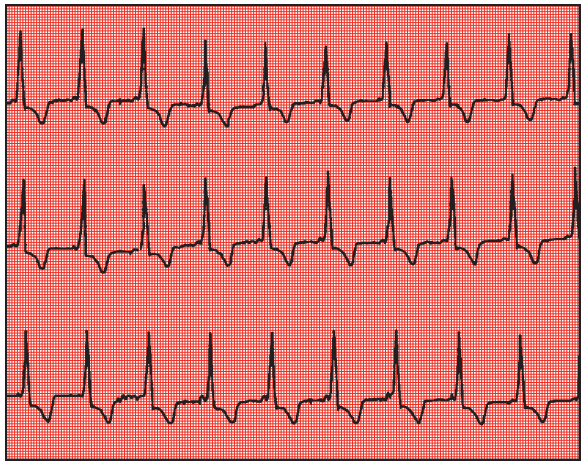


FIG. 3.76. The diagnosis is confirmed on Holter monitor. There is a short PR interval, with delta waves (FJ; 15/3/95).

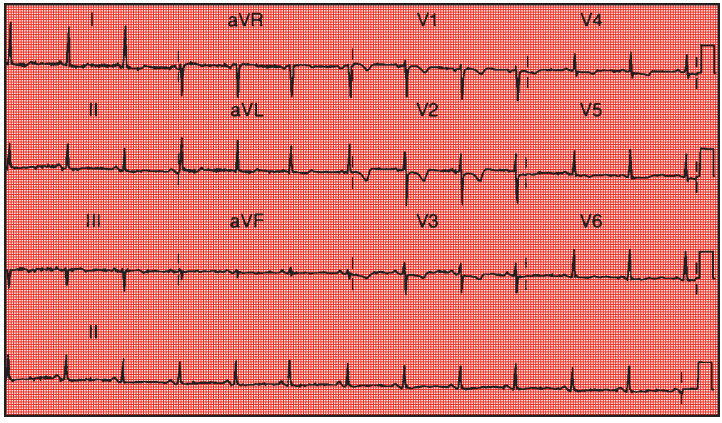


FIG. 3.77. After a further 4 years, there is normal conduction with marked anteroseptal T wave inversions. This an example of intermittent WPW syndrome (FJ; 5/7/99).

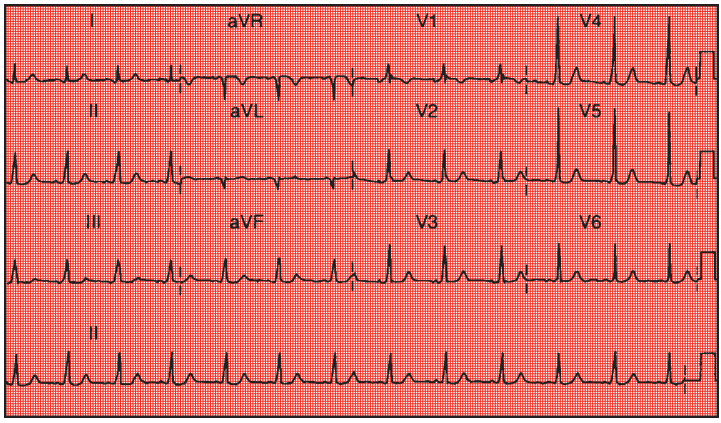


FIG. 3.78. WPW syndrome. Delta waves are apparent in leads II, III, aVF and V1. The PR interval is not particularly short (LK; 13/10/95).

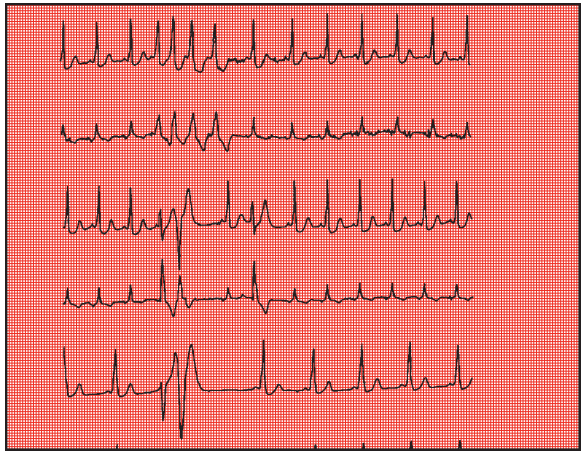


FIG. 3.79. Twenty-four hour Holter monitoring showing runs of supraventricular tachycardia in the same patient. This appearance could suggest a ventricular tachycardia (LK; 13/10/95).

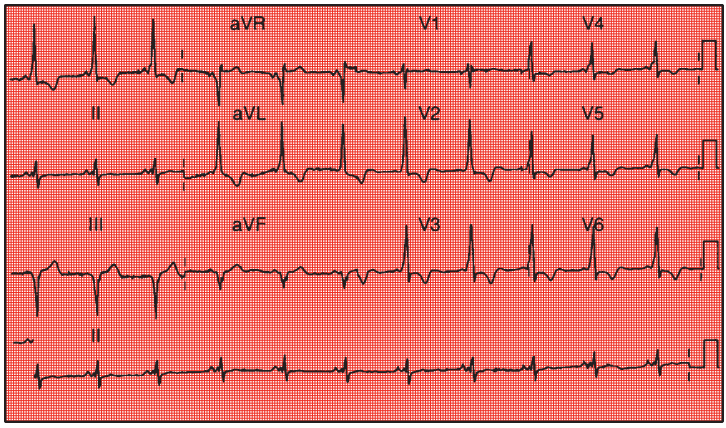


FIG. 3.80. Classical WPW change in a 26-year-old woman. Note inferior Q waves and widespread T wave inversions (JD; 11/7/93).

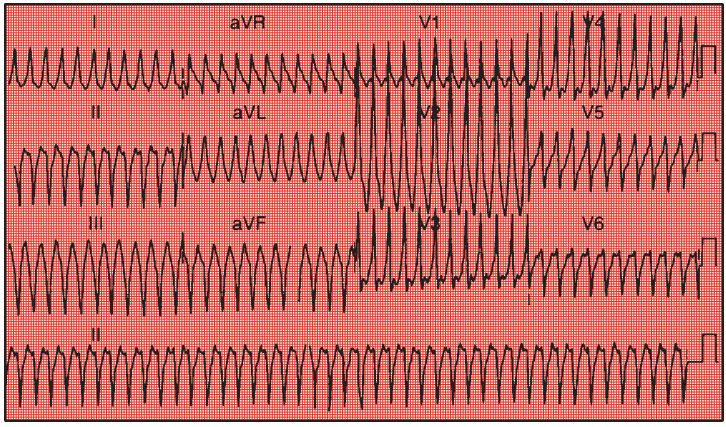


FIG. 3.81. ECG from the same patient showing a very fast supraventricular re-entry tachycardia. This could be confused with a ventricular tachycardia. Small retrograde P waves are discernible in lead I (JD; 25/7/93).

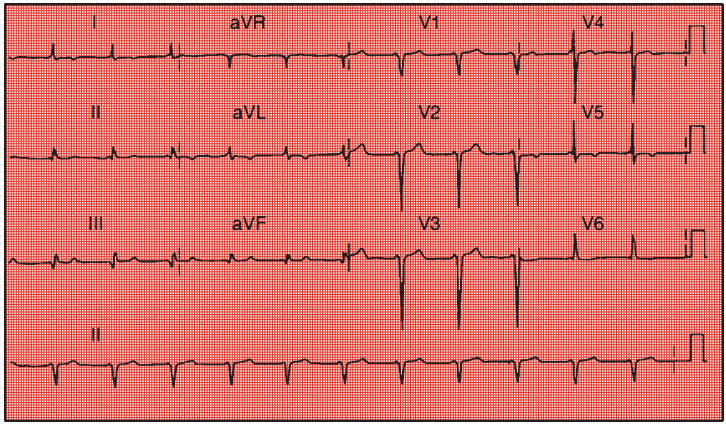


FIG. 3.82. Lown–Ganong–Levine syndrome. The short PR interval is clearly seen in lead V1. there are no delta waves (KB-G; 23/11/95).

SHORT QT SYNDROME

This is a rare cause of deaths in infants. It is a genetic ion channel disease.

HISTORICAL NOTES

R Adams (1791–1875) Irish Physician.

AFS Kent (1863–1958) Professor of Physiology, Bristol University, UK. Demonstrated muscular bridges between atria and ventricles.

L Wolff (1898–1972) American cardiologist, Massachusetts General Hospital, USA.

J Parkinson (1885–1976) English cardiologist, London Hospital UK.

PD White (1886–1973) American cardiologist, Massachusetts General Hospital, USA. The Wolff–Parkinson–White syndrome was first published in 1930.

B Lown (born 1921) American cardiologist, Peter Brent Brigham Hospital, Boston, USA. Nobel Peace Prize 1985.

WF Ganong (born 1924) American.

SA Levine (1891–1966) American cardiologist.

Lown–Ganong–Levine syndrome: short PR interval associated with supraventricular arrhythmias.

A Jervel (1901–1987) Professor of Medicine, Allerval Hospital, Oslo.

F Lange Nielsen. Contemporary of A Jervel. Jervel–Lange–Nielsen syndrome; Prolonged QT interval associated with sudden death.

C Romano (born 1924) Professor of Paediatrics, Genoa, Italy.

W Stokes (1804–1878) Irish Physician.

OC Ward (born 1923) Professor of Clinical Paediatrics, Dublin, Ireland. Romano–Ward syndrome: Prolonged QT interval.

KF Wenckebach (1864–1940) Dutch cardiologist, Groningen and Vienna, Austria. Described periodically dropped beats (1899).

W Mobitz Germany (1889–1951) Published work on partial conductor block.

KEY MESSAGES

- Sinus arrhythmia
- Sinus bradycardia
- Sinus tachycardia
- SA disease
- SA block
- SA arrest
- AV block – first, second and third degrees
- Bundle branch block
- Right and left hemiblocks
- Carotid sinus syndrome
- Pre-excitation i.e., accessory pathways
- QT interval

Chapter 4
Rhythm Disturbances

Arrhythmias are rhythms of the heart arising outside the normal conductive pathways or within them, but at an abnormal rate and with an abnormal ECG pattern. They are conventionally divided into two categories:

1. Supraventricular (SVT) arrhythmias, which originate from the atria or AV node complex (junctional).
2. Ventricular arrhythmias, which arise below the AV junction.

Rhythm disturbances can be intermittent or established, benign or lethal and, more often than not, disturbing to the patient.

SUPRAVENTRICULAR ARRHYTHMIAS

The impulse travels down the established pathway and characteristically produces a narrow complex. There are three exceptions, which give rise to wide QRS complexes:

- Concomitant bundle branch block
- Aberration (fatigue of a bundle)
- Accessory conduction (for example WPW syndrome)

Atrial Ectopic Beats

Atrial ectopic beats are also known as atrial premature beats, or APBs. Unlike sinus P waves, they are characterized by premature P waves, with an altered PR interval. The latter can be prolonged if the atrial ectopic beat encounters a refractory AV node or bundle. In atrial bigeminy, every second beat is an atrial ectopic beat. In atrial trigeminy every third beat is an atrial ectopic beat.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_4,

APBs can occur in normal individuals, caused by anxiety, or excessive caffeine or alcohol consumption, or they may reflect cardiac pathologies (Figs. 4.1–4.7). Treatment, when indicated, includes beta-blockers, verapamil, disopyramide, and sotalol.

Atrial parasystole is rare. Independent P waves are seen and the parasystolic P wave intervals have a mathematical relationship.

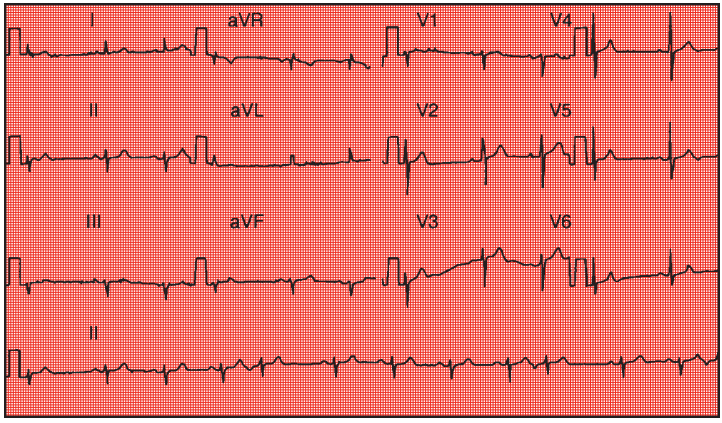


FIG. 4.1. This 62-year-old woman presented complaining of palpitation. The rhythm strip at the bottom of the ECG shows atrial ectopic activity in lead II. Atrial ectopic beats are characterized by inverted or altered P waves followed by a compensatory pause. This patient consumed 12 cups of strong tea per day (NB; 4/3/96).

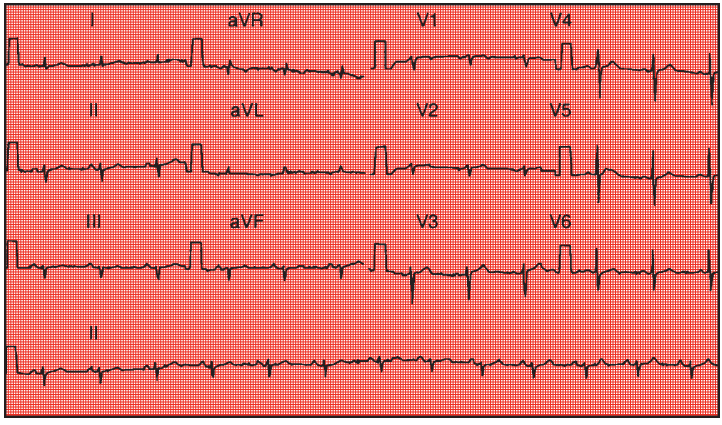


FIG. 4.2. After discontinuing tea, there is no further palpitation and only sinus rhythm is evident (NB; 15/4/96).

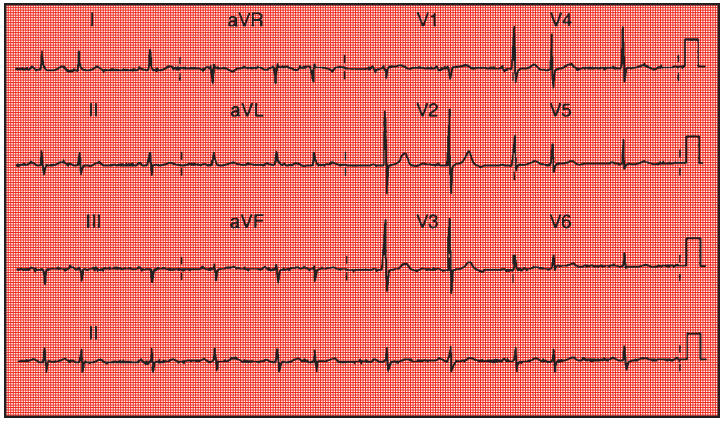


FIG. 4.3. An elderly man with ischaemic heart disease suffered chronic atrial extrasystoles. There was no change over the years and no attempt at medication. The atrial ectopic beats are best seen in lead II. The P wave is different from that of the sinus beats. There is a compensatory pause (RW; 29/10/99).

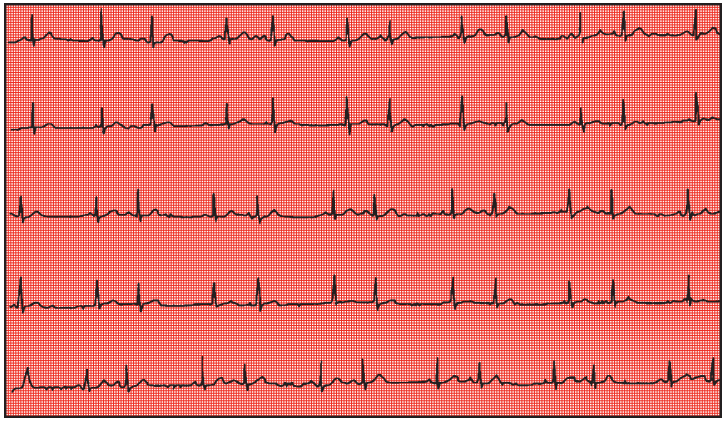


FIG 4.4. Twenty-four-hour Holter monitoring showing atrial ectopic beats. Every second beat is an atrial extrasystole, hence the rhythm is described as atrial bigeminy.

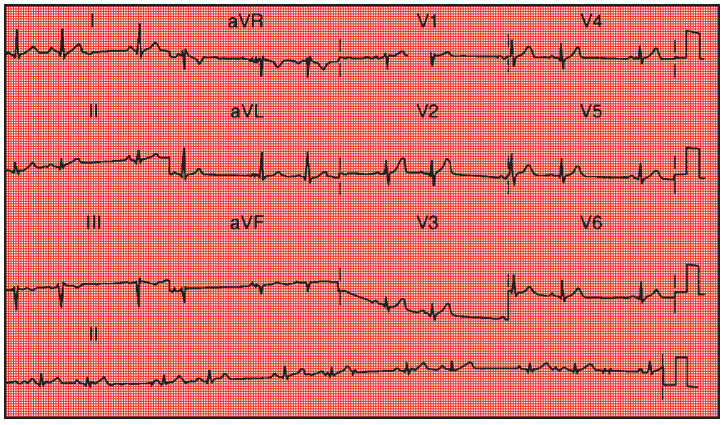


FIG. 4.5. A further example of atrial bigeminy (NT; 24/10/85).

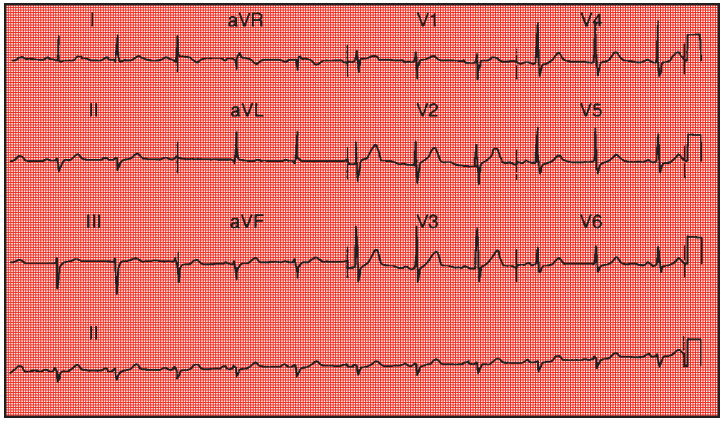


FIG. 4.6. Preoperative ECG from an elderly man undergoing knee replacement (RB; 1/6/99).

Supraventricular arrhythmias can be caused by:

- A re-entry mechanism
- Abnormal atrial activity

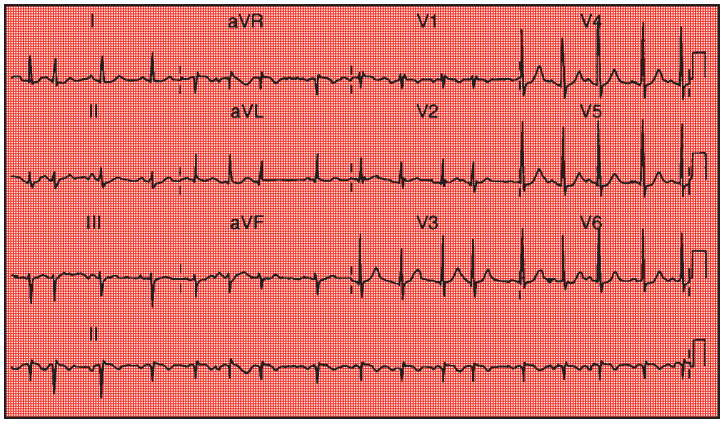


FIG. 4.7. Immediate postoperative ECG showing atrial ectopic activity. The patient responded to metoprolol (RB; 6/6/99).

Re-entry Mechanism

Atrioventricular Nodal Re-entrant (AVNR) Tachycardia

This accounts for more than 50% of all SVT arrhythmias. Two functional conducting pathways, fast and slow, are demonstrable electrophysiologically *within the AV node.*. During sinus conduction the impulse travels down the fast pathway. Simultaneous conduction occurs down the slow pathway, but is blocked (concealed) by retrograde activation of the slow pathway, caused by the anterograde impulse traveling through the AV node. The two impulses collide and only the fast pathway is able to conduct, giving rise to a tachycardia.

AV nodal re-entrant tachycardia is defined as *common* when the re-entry circuit conducts down the slow pathway and returns via the fast pathway. P waves are not usually visible as they coincide with the QRS complex and the arrhythmia is commonly triggered by an atrial premature beat (APB). Common AVNR tachycardia is illustrated in Figs. 4.8–4.11.

Uncommon AVNR tachycardia occurs in only 10% of cases. The anterograde conduction travels down the fast pathway and retrogradely up the slow pathway. Usually retrograde late P waves are seen in the inferior leads (Figs. 4.12 and 4.13). This form of SVT arrhythmia is characteristically triggered by a ventricular premature beat (VPB).

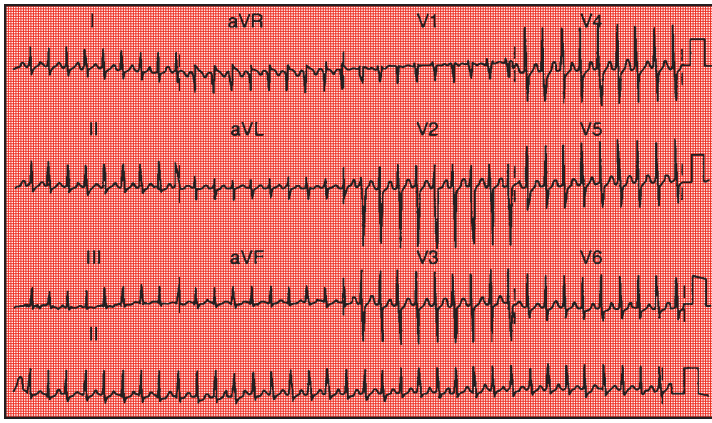


FIG. 4.8. This young female patient has AV nodal re-entrant tachycardia (common). The heart rate is just over 180 bpm, the complexes are narrow, the RR interval is regular and there are no visible P waves (AB; 12/1/93).

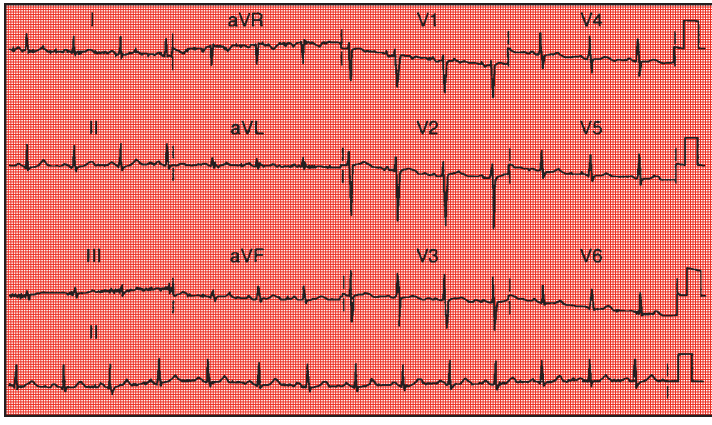


FIG. 4.9. The patient underwent radiofrequency ablation. She had no further symptoms. This ECG, taken 2½ years later, shows normal sinus conduction. The axis remains unchanged, confirming the supraventricular origin of the initial tachycardia (AB; 21/7/95).

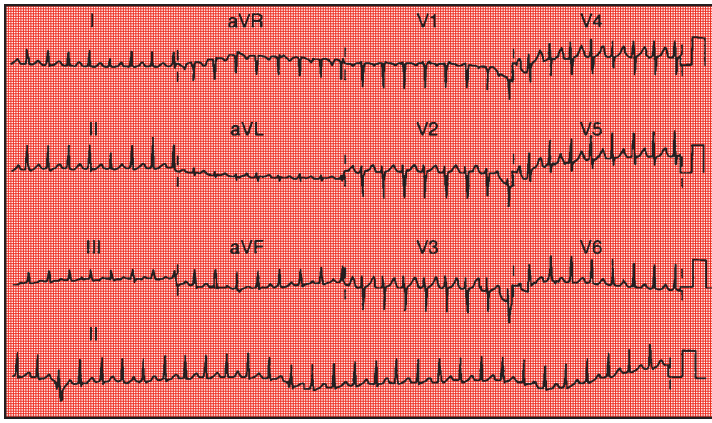


FIG. 4.10. This young female patient had a frequent history of palpitation. AV nodal re-entrant tachycardia is present; the heart rate is 170 bpm. P waves are not visible (SA: 23/6/99).

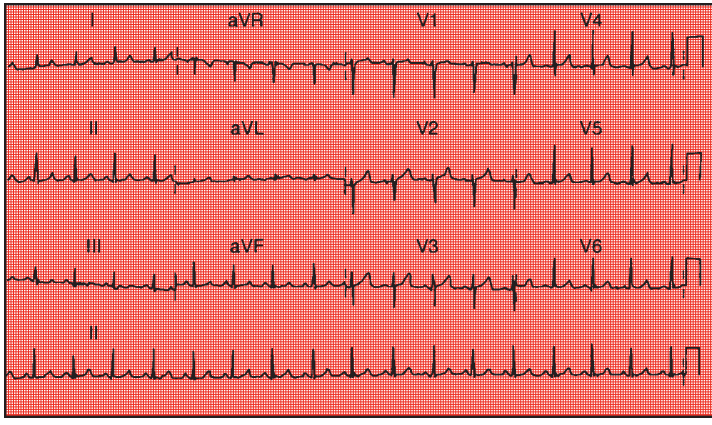


FIG. 4.11. ECG from the same patient, taken half an hour after administration of adenosine. There is normal sinus conduction. The patient subsequently underwent ablation (SA; 23/6/99).

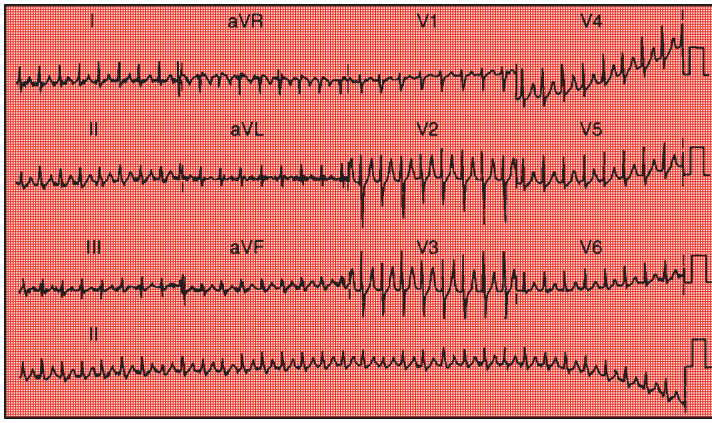


FIG. 4.12. This 77-year-old woman has supraventricular trachycardia (AV nodal re-entry tachycardia). This is the uncommon variety, with retrograde P waves in lead V1. The heart rate if 180 bpm (23/10/97).

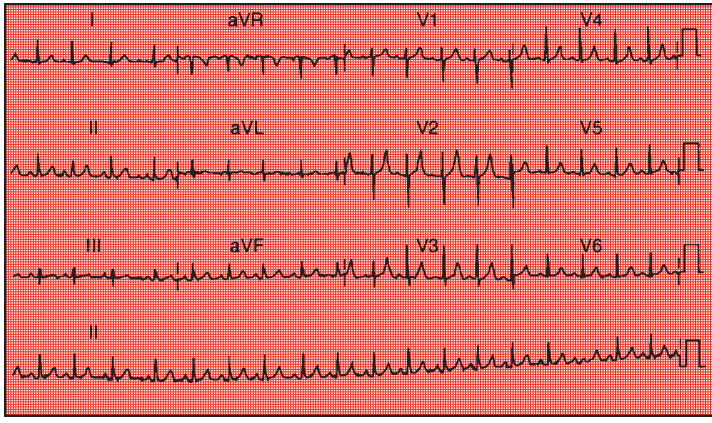


FIG. 4.13. ECG from the same patient 20 min later, after administration of intravenous adenosine; the pattern is reversed to normal sinus conduction (23/10/97).

Atrioventricular Re-entrant Tachycardia

Atrioventricular re-entrant tachycardia occurs at a heart rate of 130–250 bpm. It is the most common form of SVT arrhythmia after AVNR tachycardia. One or more accessory pathways consisting of *extra bands of conducting tissue*, connect the atria and ventricles, for example in WPW syndrome. An APB or VPB can set off a re-entry SVT arrhythmia. The impulse travels anterogradely down the normal pathway (through the nAV ode) and retrogradely to the atrium along the accessory pathway. No delta waves appear as conduction occurs normally. This is known as *orthodromic* atrio-ventricular re-entrant tachycardia.

In 10% of patients with WPW syndrome and AV re-entrant tachycardia the impulse travels down the accessory pathway and retrogradely through the bundle branches, the bundle of His and the AV nodal system. This is referred to as *antidromic* atrioventricular re-entrant tachycardia. It is characterized by wide QRS complexes (with exaggerated delta waves). Examples of atrioventricular re-entrant tachycardia are shown in Figs. 4.14–4.17.

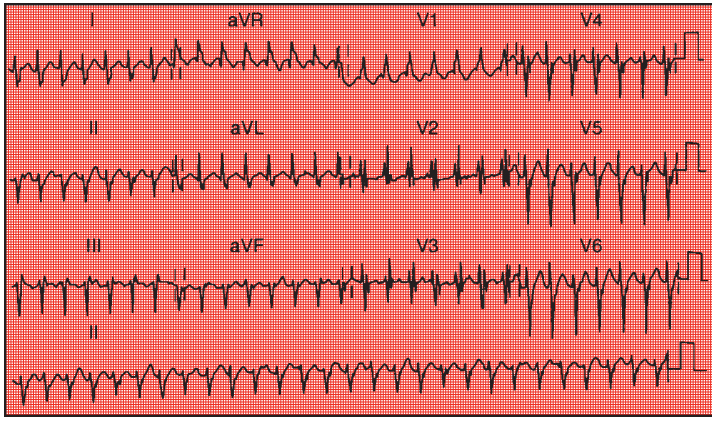


FIG. 4.14. ECG from a young man with regular tachycardia (150 bpm) with right bundle branch block. The bottom rhythm strip shows retrograde P waves on the upslope of the S wave in lead II. This is probably AV re-entrant tachycardia. The complexes are wide because of the right bundle branch block (RBBB) (FC; 23/7/88).

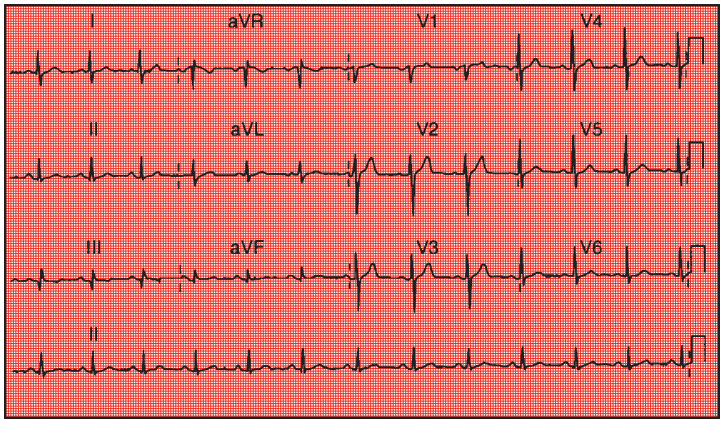


FIG. 4.15. ECG from the same patient 11 years later, by which time he was receiving medication. Note the change of axis and bundle branch block (aberration) during tachycardia in Fig 4.14 (FC; 20/1/99).

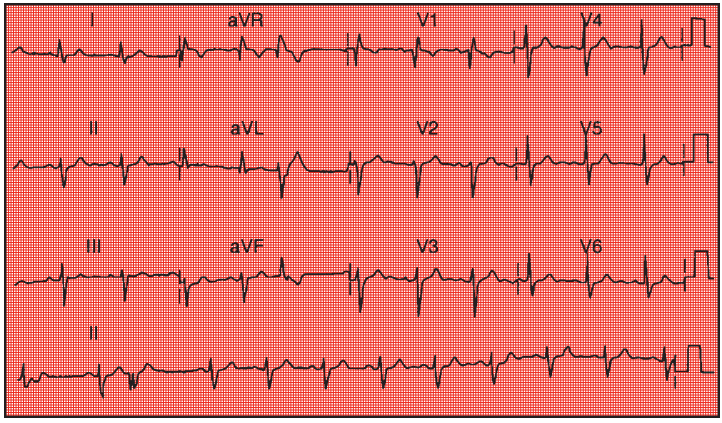


FIG. 4.16. Preoperative ECG showing sinus rhythm, right bundle branch block and left axis deviation (HB; 4/3/96).

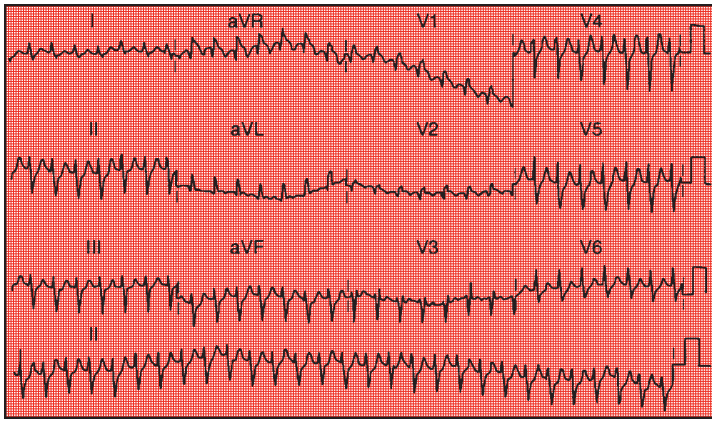


FIG. 4.17. Perioperative ECG showing rapid supraventricular arrhythmia. The complexes are wide because of the underlying right bundle branch block; the heart rate is 150 bpm. The patient made a spontaneous recovery. The axis remains unchanged (HB; 8/3/96).

Abnormal Atrial Activity

Junctional Tachycardia

In junctional tachycardia there is enhanced automaticity or triggered activity within the AV junction (not re-entry), a rare cause of SVT in adults. This condition occurs in the presence of trauma to the AV junction (for example surgery, digoxin toxicity, acute myocardial infarction). Characteristically, the SVT produces a narrow complex showing AV dissociation or 1:1 ventricular atrial activation (Figs. 4.18–4.22).

Atrial Tachycardia (Rate 120–250 bpm)

Unifocal

The P wave is of single morphological pattern. The atrial rate is usually below 250 bpm. The arrhythmia originates in atrial musculature; the mechanism can involve a re-entry circuit, enhanced automaticity, or triggered activity such as in digoxin toxicity (Figs. 4.23 and 4.24).

This is a rare cause of SVT arrhythmia. It can occur in the presence of heart disease, but not necessarily so.

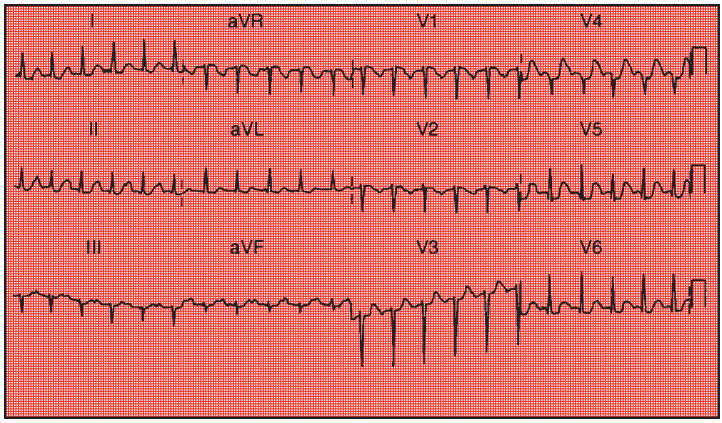


FIG. 4.18. Probable junctional tachycardia. Retrogarde P waves are best seen in lead III and aVF. Rate 128 bpm (SV; 20/6/99).

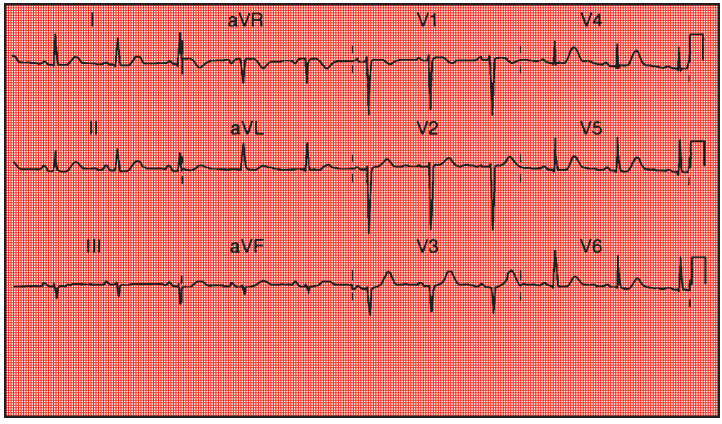


FIG. 4.19. ECG from the same patient taken the next day, after medication (SV; 21/6/99).

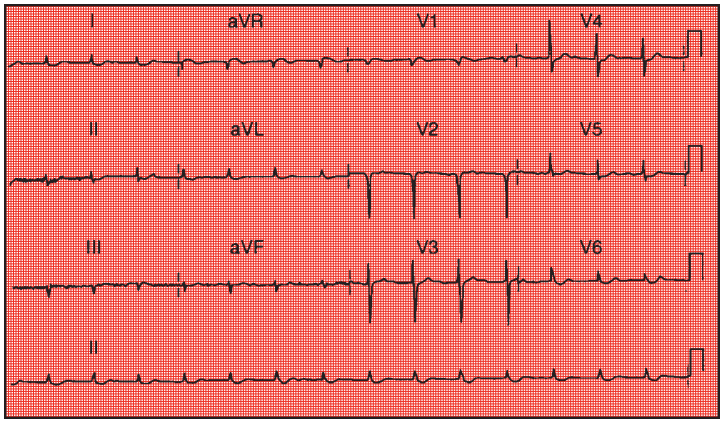


FIG. 4.20. Junctional rhythm. The complexes are narrow and the rhythm is regular. There are no visible P waves. The patient is elderly, postoperative and died soon after this trace was taken. "U" waves are seen in a number of leads (V2, V3). (RG; 2/11/99).

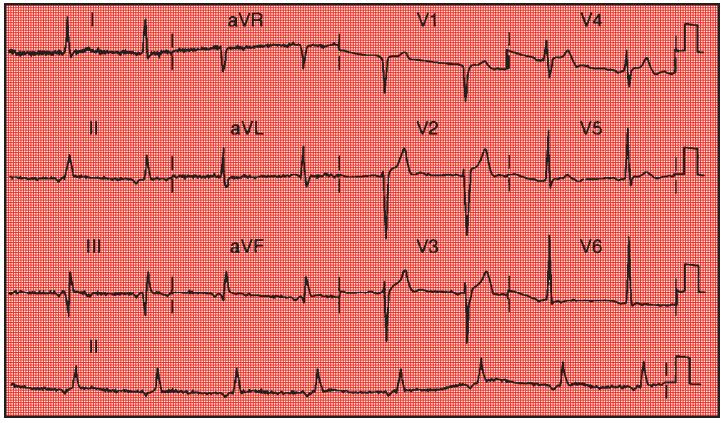


FIG. 4.21. AV junctional rhythm (coronary sinus rhythm). There are inverted P waves in leads II, III and aVF. This patient had previously undergone bypass surgery (AB; 21/7/95).

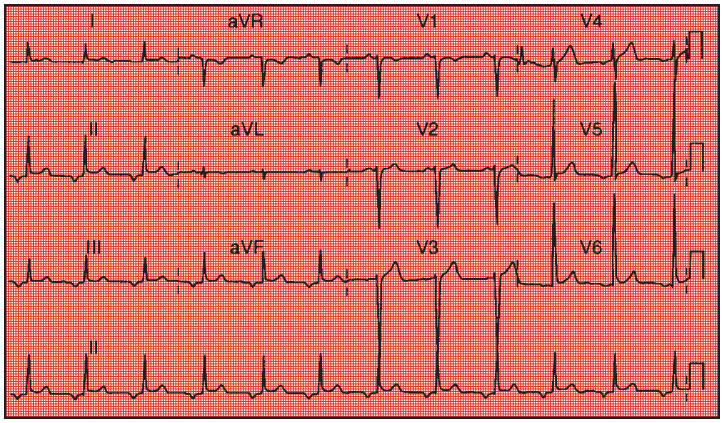


FIG. 4.22. A further example of atrioventricular junctional rhythm. The diagnosis is mitral valve prolapse (PJ; 6/10/97).

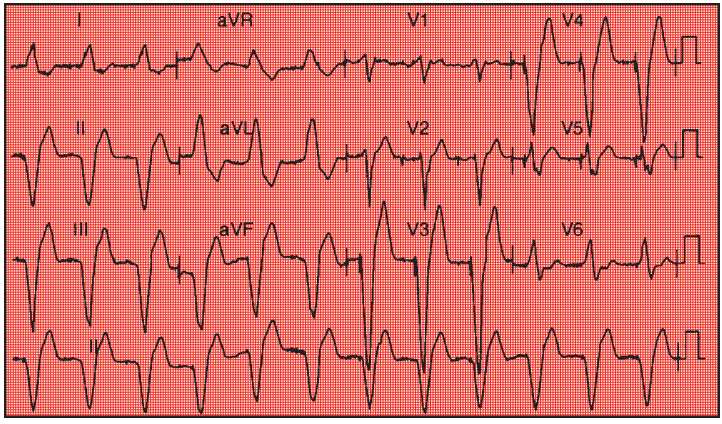


FIG. 4.23. Dual chamber pacing. Atrial and ventricular pacing signals are seen clearly in lead V2 and V3 (SB; 20/4/98).

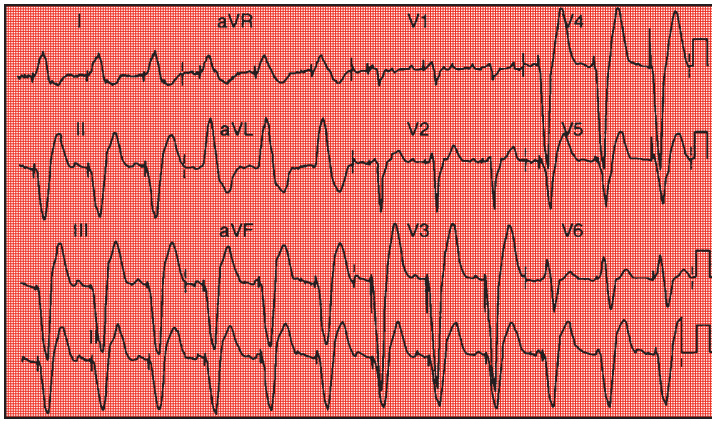


FIG. 4.24. The same patient developed an atrial tachycardia. P waves are seen clearly in lead V1. P rate is 180 bpm (atrial tachycardia P rate is usually between 120 and 250 bpm) (SB; 11/8/99).

Multifocal

P waves of varying morphology are recognizable. This arrhythmia is rare; it is seen in acutely ill patients, often with underlying pulmonary disease.

Atrial Flutter

Typical (Type I)

Typical atrial flutter is caused by a re-entrant counterclockwise macro-circuit in the right atrium. Negative saw-toothed F waves are seen in inferior leads. The atrial rate averages 300 bpm. If the F waves are positive in the inferior leads, the microcircuit conducts clockwise.

Atypical (Type II)

The atrial rate is faster than in typical atrial flutter, at up to 400 bpm, with positive deflections in the inferior leads. Atypical flutter can conduct with 2:1 or 4:1 heart block or variable block, depending on the refractory properties of the conducting system. If the block is variable, the pulse is irregular; hence the necessity of ECG documentation.

Causes of atrial flutter include hyperthyroidism, ischaemic heart disease, hypertension, rheumatic heart disease, cardiomyopathies, pericarditis, congenital heart conditions, pulmonary disease, sick sinus syndrome and drug abuse, particularly alcohol.

Examples of ECG appearance in patients with atrial flutter are shown in Figs. 4.25–4.49.

Atrial Fibrillation

Atrial fibrillation is the commonest of all arrhythmias. It has the same aetiology as atrial flutter, increasing in frequency with ageing. Here the atria do not contract: they "fibrillate" at a rate of 300–600 bpm. The AV node cannot cope with this number of impulses and only some will get through to the ventricles. Atrial fibrillation is caused by multiple microwavelet re-entry circuits. Therefore the ventricular response is totally irregular. The ventricular rate is usually fast, but occasionally it is extremely slow and pacing may be required (for example in sick sinus syndrome). Rarely, atrial fibrillation can coexist with complete heart block. The ventricular rate then becomes regular and slow, the complexes are wide (originating in the ventricles), but usually the F waves are still visible. Atrial flutter or AV nodal re-entrant tachycardia can degenerate to atrial fibrillation. Lone atrial fibrillation refers to this arrhythmia for which no cause can be detected. Figures 4.50–4.77 illustrate the different appearances of atrial fibrillation.

Left untreated, these supraventricular arrhythmias can bring on congestive cardiac failure with dilatation of the heart, which is more likely in patients with underlying cardiac pathologies. Atrial fibrillation poses a significant risk; emboli can occur and can pass

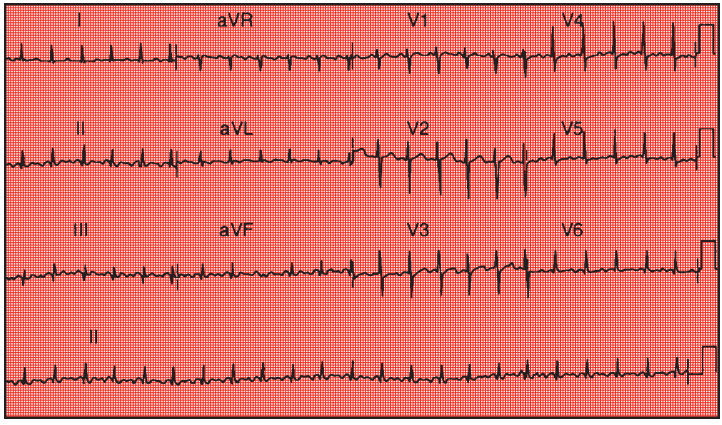


FIG. 4.25. Perioperative ECG (after bypass surgery) showing rapid tachycardia at 140 bpm. Note the narrow complexes. Flutter waves are seen in rhythm strip II, occurring at 300 bpm. The diagnosis is atrial flutter (JB; 20/6/99).

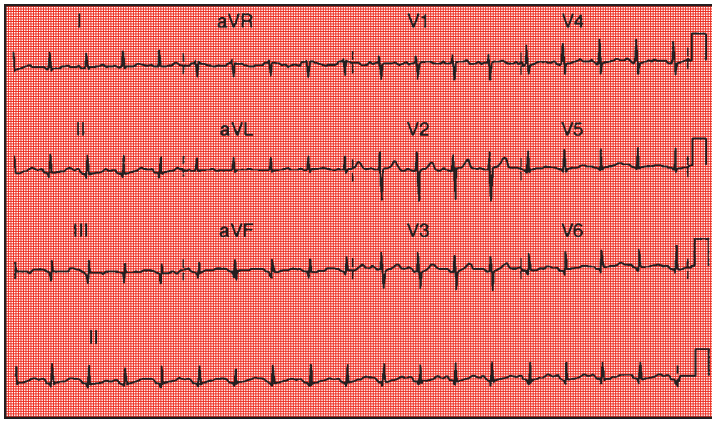


FIG. 4.26. ECG from the same patient after medication, 2 h later (JB; 20/6/99).

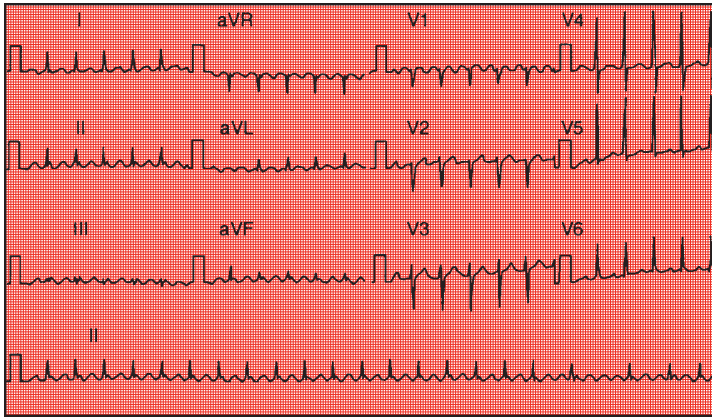


FIG. 4.27. Classical atrial flutter. The saw tooth appearance is present in lead II. Flutter waves are occurring at ±300 bpm with a ventricular rate of 142 bpm, in other words a 2:1 response. At the end of the rhythm strip (bottom line) there is more profound block. The ventricular rate slows down, but the flutter waves are unaltered (ET; 9/1/95).

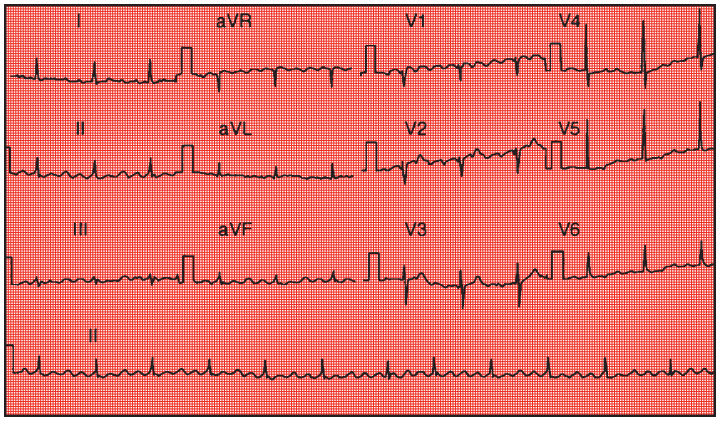


FIG. 4.28. The same patient 2 months later, on digoxin, has converted to atrial fibrillation. The QRS complexes are now occurring irregularly (ET; 6/3/95).

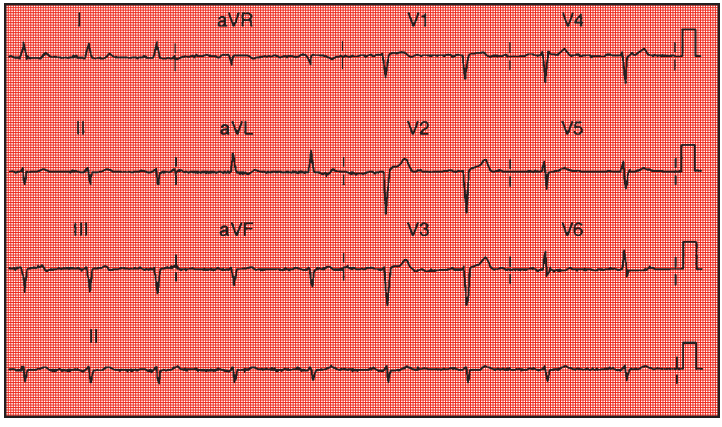


FIG. 4.29. An elderly patient who had undergone bypass surgery shows sinus rhythm, anteroseptal Q waves and left axis deviation (HI; 10/11/98).

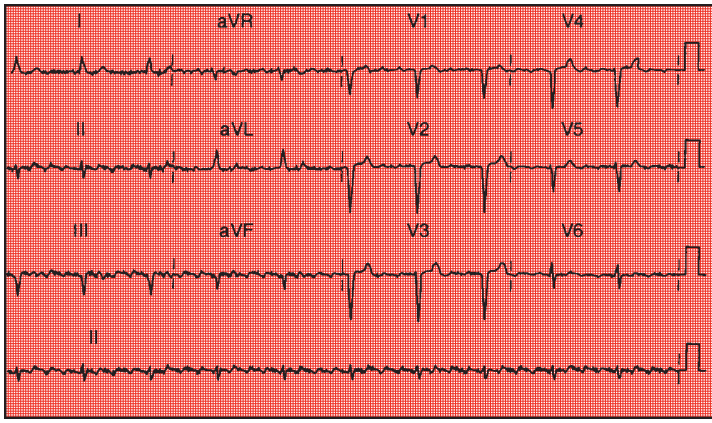


FIG. 4.30. The same patient presented with an abnormal rhythm several months later. There is no change in the QRS complexes or axis. The ventricular rate is 60 bpm. There are P waves (best seen in lead V1), occurring at a rate of 190 bpm. Rhythm strip II at the bottom of the ECG would suggest an atrial flutter, but the P rate is too slow. The diagnosis is more likely an atrial tachycardia (HI; 5/1/99).

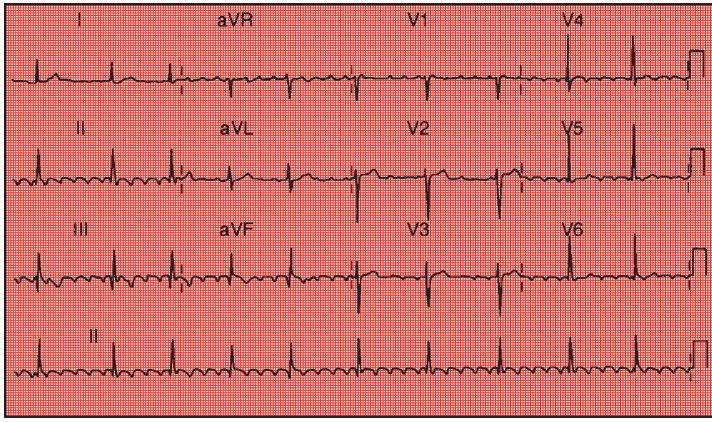


FIG. 4.31. Atrial flutter. The P rate is 300 bpm, and the ventricular rate is 56 bpm. This is atrial flutter with 4:1 AV block (AC; 7/4/97).

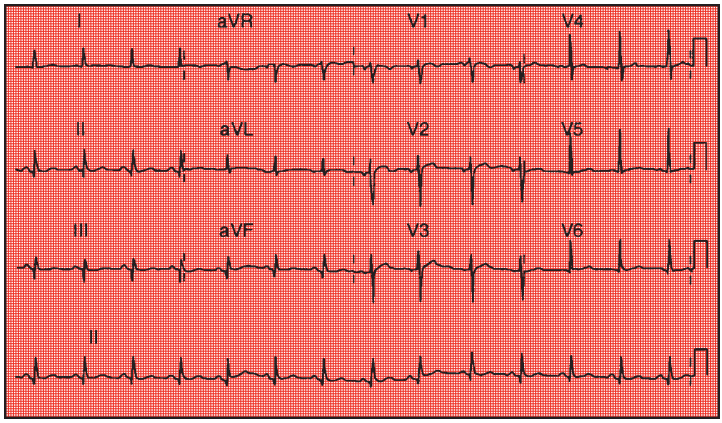


FIG. 4.32. The same patient was in sinus rhythm several months later. Inferior Q waves are more clearly visible. They can also be appreciated during the flutter episode (AC; 23/6/97).

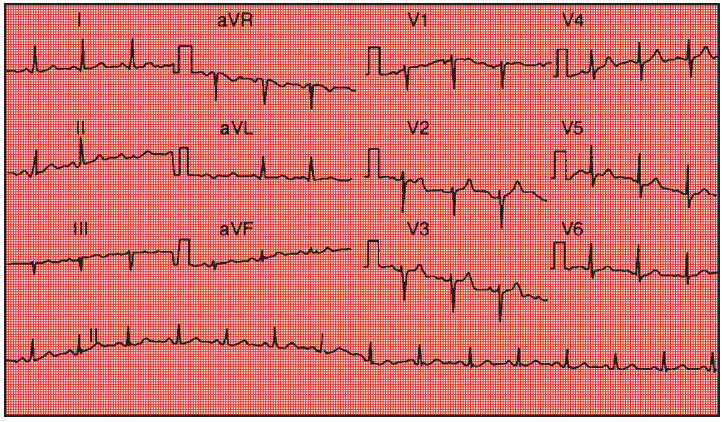


FIG. 4.33. Normal sinus conduction (R McL; 22/1/96).

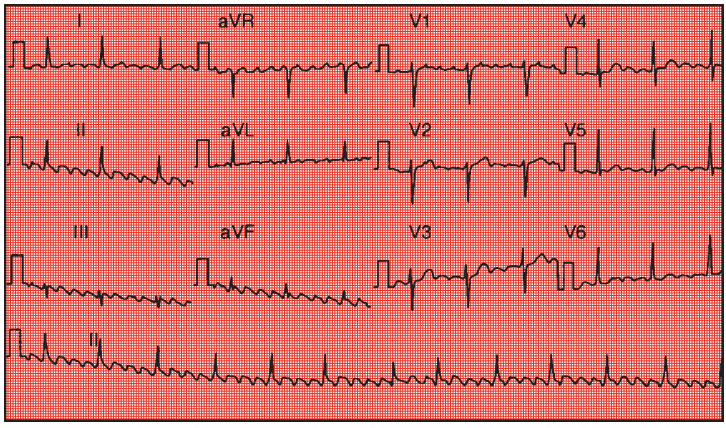


FIG. 4.34. Nine months later there is atrial flutter, with a ventricular rate of 70 bpm. The flutter rate if 300 bpm. Rhythm strip lead II at the bottom of the ECG shows variable AV block, with a characteristic saw tooth appearance (R McL; 21/10/96).

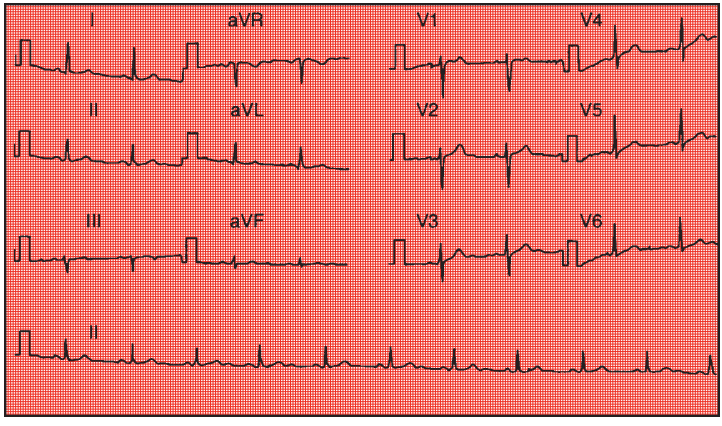


FIG. 4.35. Normal sinus conduction is evident 2 months later, after successful cardioversion (R McL; 9/12/96).

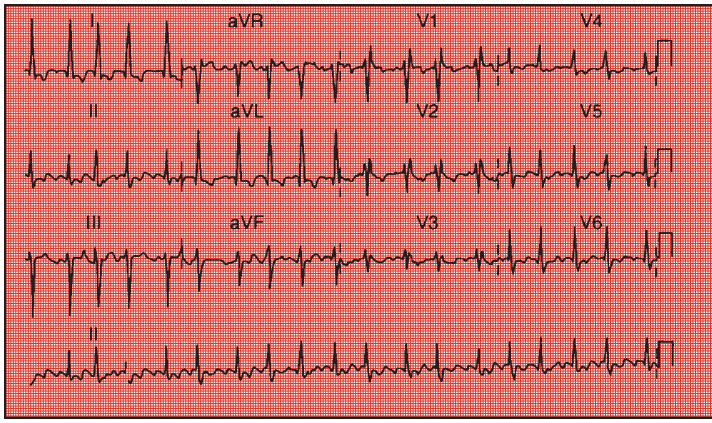


FIG. 4.36. ECG from a 40-year-old patient after aortic valve replacement. There is atrial flutter, P wave activity at 300 bpm and variable AV block (PL; 15/6/99).

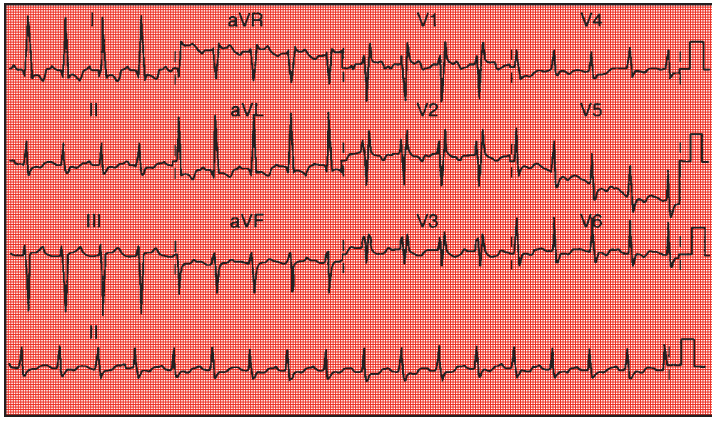


FIG. 4.37. The same patient immediately after cardioversion. Sinus rhythm is re-established. There are upright P waves in lead I, and negative P waves in lead V1 (PL; 15/6/99).

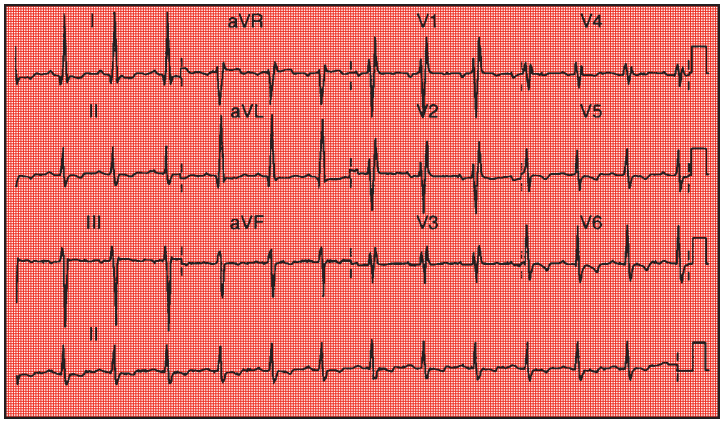


FIG. 4.38. Two months later sinus rhythm is maintained. Note right bundle branch block and left ventricular voltages (PL; 8/9/99).

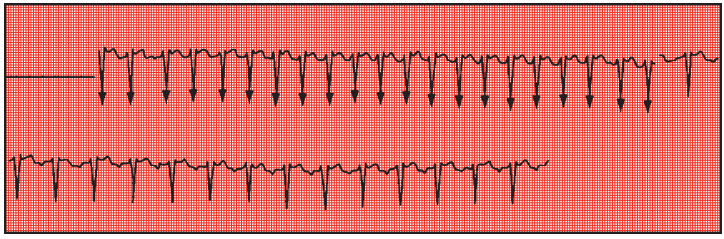


FIG. 4.39. The same patient showing a flutter rhythm at cardioversion. The initial rate is 158 bpm. The thick arrows indicate a synchronized signal from the cardioversion apparatus. Immediately after cardioversion, the rate is just over 90 bpm and inverted P waves are now clearly visible (PL; 15/6/99).

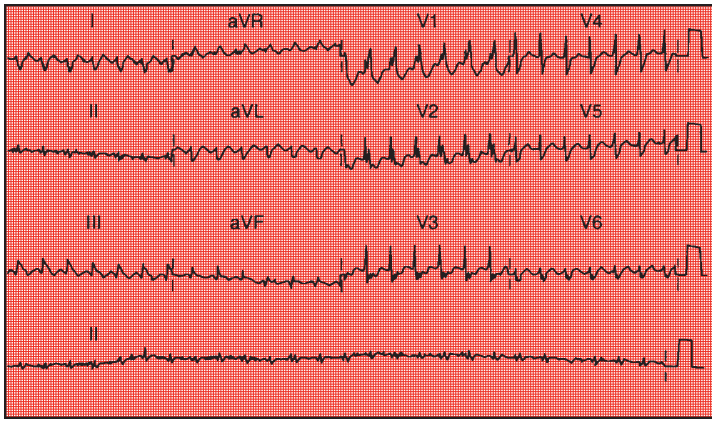


FIG. 4.40. Atrial flutter with right bundle branch block. The ventricular rate is 140 bpm; flutter waves are occurring at 300 bpm. The saw tooth appearance is evident in lead III (HB; 2/5/96).

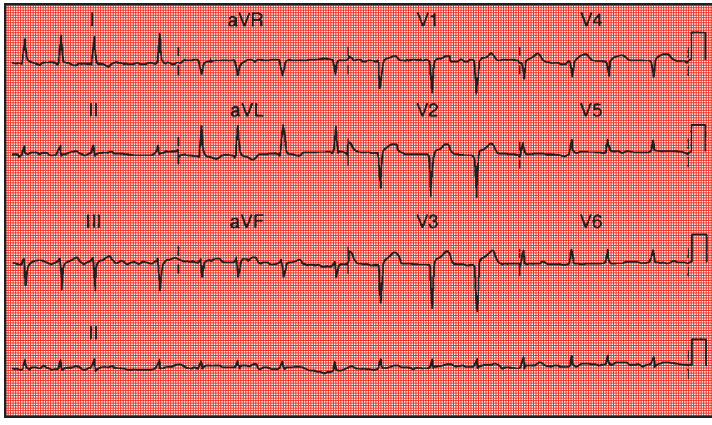


FIG. 4.41. Middle-aged patient after bypass surgery showing atrial fibrillation (AL; 16/6/98).

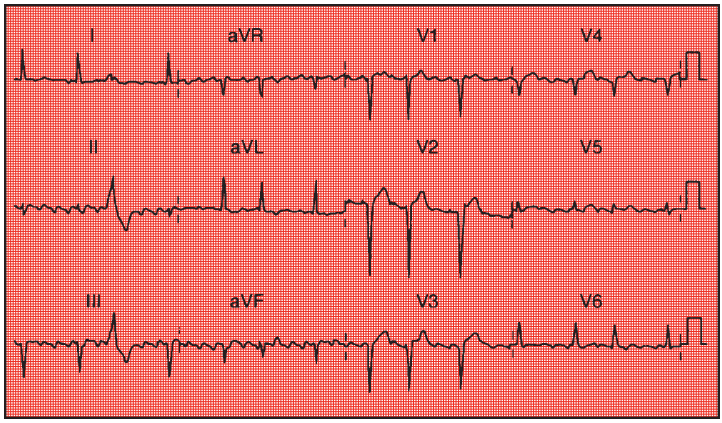


FIG. 4.42. On medication, atrial flutter waves develop (at 300 bpm); these are best seen in leads II and aVR (AL; 20/6/98).

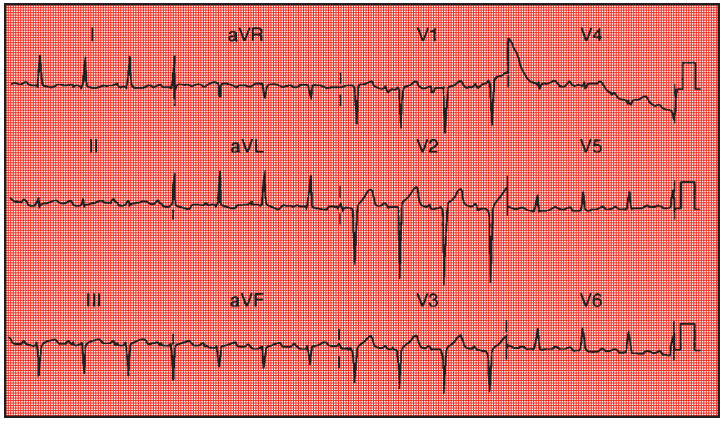


FIG. 4.43. The patient was restored to sinus rhythm after successful cardioversion. P waves in leads II, III and aVF are notched, indicating high left atrial pressure (AL; 20/6/98).

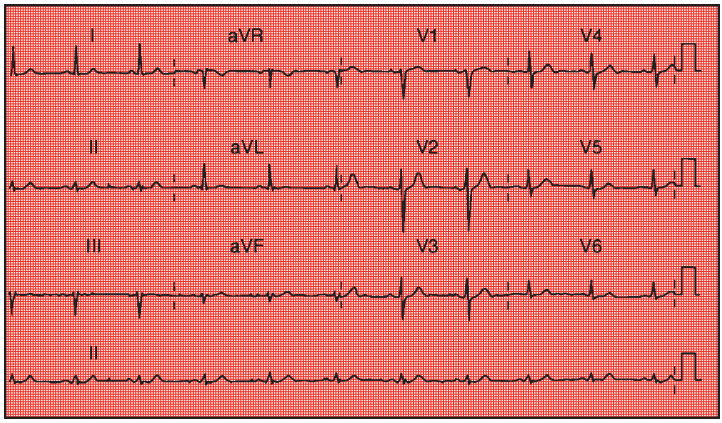


FIG. 4.44. ECG from a middle-aged patient, years after bypass surgery (ED; 28/7/98).

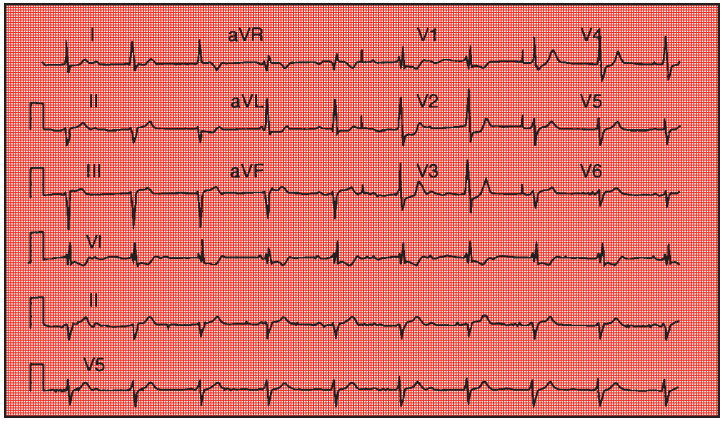


FIG. 4.45. During bladder surgery, the patient suffered a perioperative inferior infarction. Ventricular rate is 57 bpm. Dissociated P waves are best seen in lead I and II; there is nodal rhythm with AV dissociation (ED; 3/8/98).

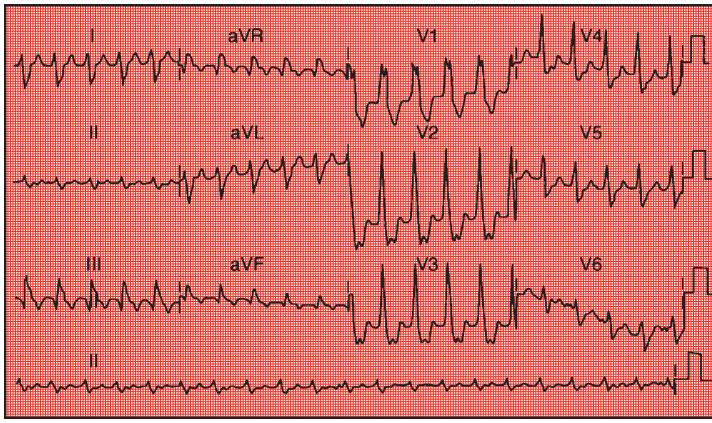


FIG. 4.46. The same patient, 2 months later. The ECG shows tachycardia and a ventricular rate of 120 bpm causing right bundle branch block (aberration). Leads III suggests atrial flutter; atrial activity is 180 bpm (ED; 2/10/98).

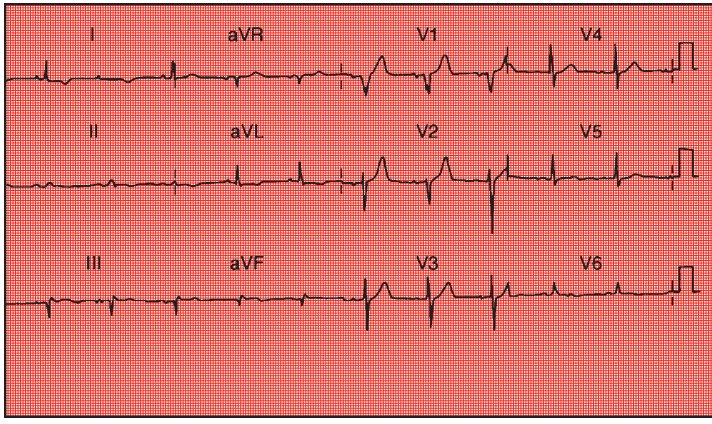


FIG. 4.47. Two days later the patient is back to sinus conduction (with medication) with normal QRS complexes. Residual Q waves are visible in leads III and aVF (ED; 4/10/98).

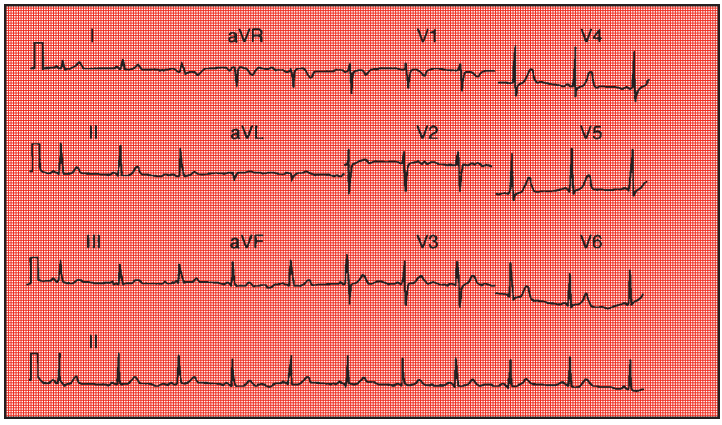


FIG. 4.48. Normal electrocardiogram in a young Japanese patient (MS; 13/2/93).

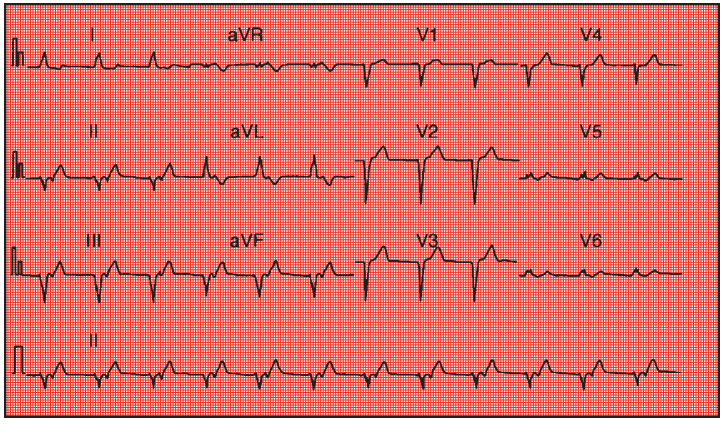


FIG. 4.49. ECG from the same patient showing nodal (junctional) rhythm, a ventricular rate of 72 bpm and regular, widened QRS complexes (LBBB). Retrograde P waves are best seen in the inferior leads (MS).

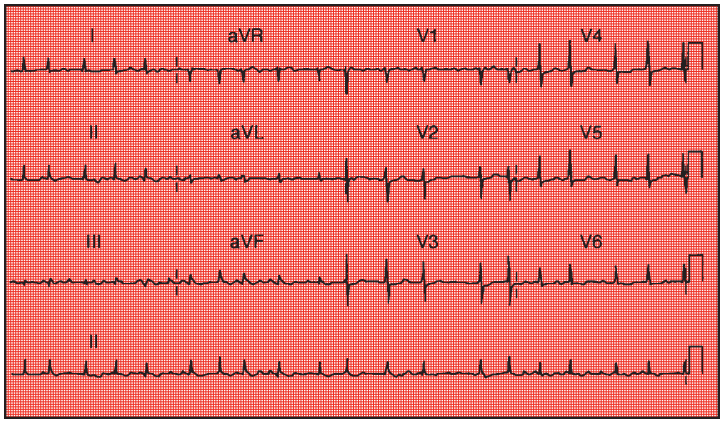


FIG. 4.50. From a middle-aged man with ischaemic heart disease. There is paroxysmal atrial fibrillation and the ventricular rate is irregular with classical "fibrillatory waves" in lead V1 (AS: 4/6/98).

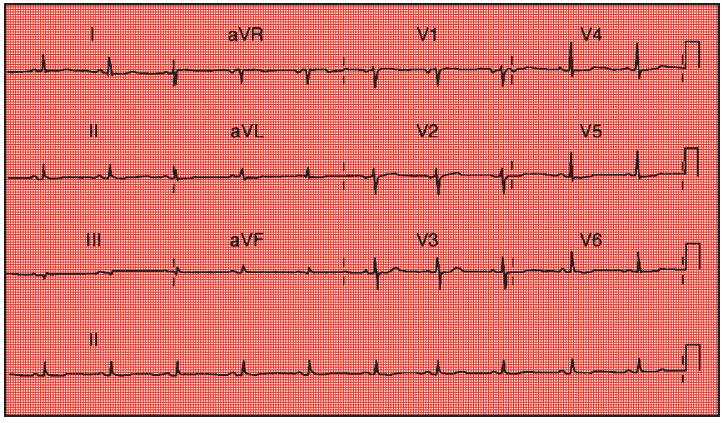


FIG. 4.51. The same patient after electrical cardioversion. There is sinus rhythm with similar complexes and axis (AS; 15/6/98).

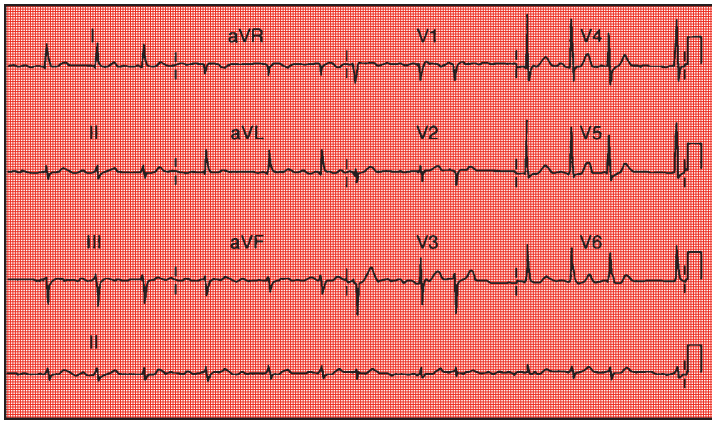


FIG. 4.52. Atrial fibrillation of recent onset in a middle-aged man who had consumed an excessive amount of alcohol (SG; 25/3/98).

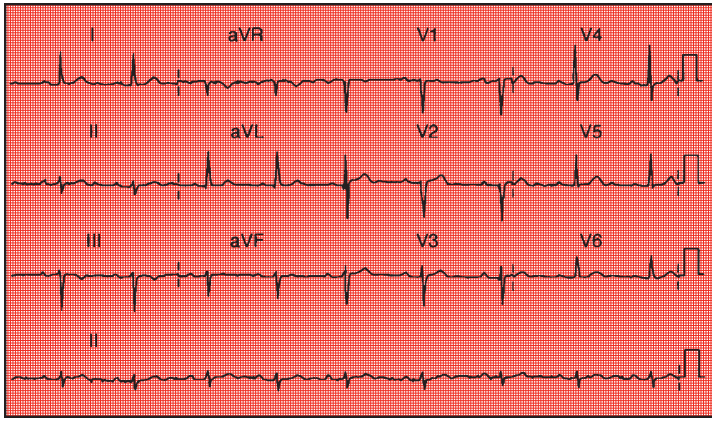


FIG. 4.53. After failed medical conversion with amiodarone and digoxin the patient is successfully restored to sinus rhythm by electrical cardioversion. Note similarity of complexes and electric axis (SG; 19/5/98).

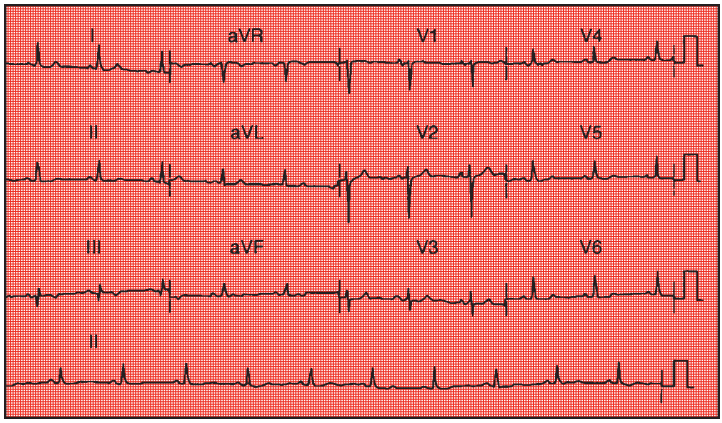


FIG. 4.54. ECG from an elderly woman with ischaemic heart disease (KF; 11/3/93).

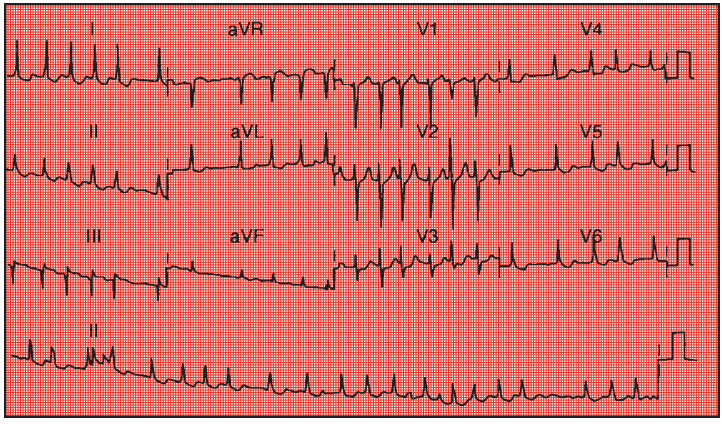


FIG. 4.55. The patient was admitted in rapid atrial fibrillation, with a ventricular rate of 150–170 bpm. The tachycardia gives rise to angina, and digoxin induced ST changes are also visible. The third "complex" in the rhythm strip is an artefact (KF; 9/1/94).

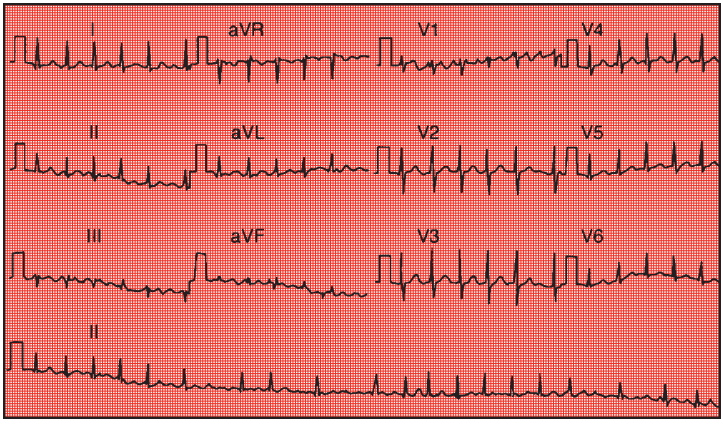


FIG. 4.56. Rapid atrial fibrillation in a middle-aged man. The patient had no symptoms and no cause for atrial fibrillation was found (Mr H; 21/2/97).

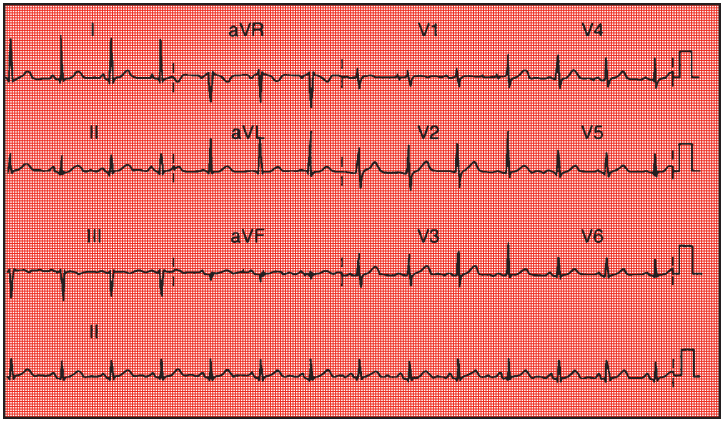


FIG. 4.57. The same patient 18 months later, in sinus rhythm (Mr H; 19/8/98).

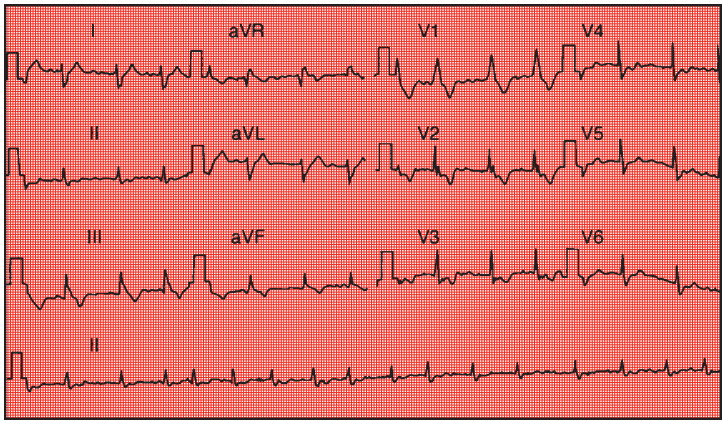


FIG. 4.58. This elderly man had established atrial fibrillation. There is right bundle branch block, with right axis deviation constituting bifascicular block, The wide complexes are due to the RBBB (AS; 6/12/96).

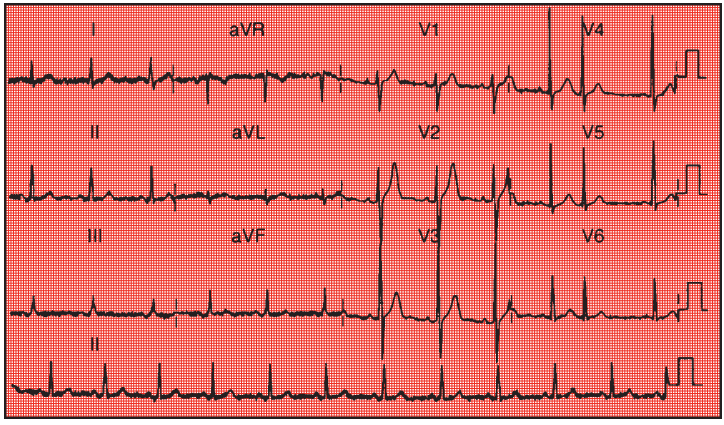


FIG. 4.59. ECG from a middle-aged man with previous rheumatic mitral disease and mitral valvotomy. Sinus rhythm is evident (SC; 23/6/93).

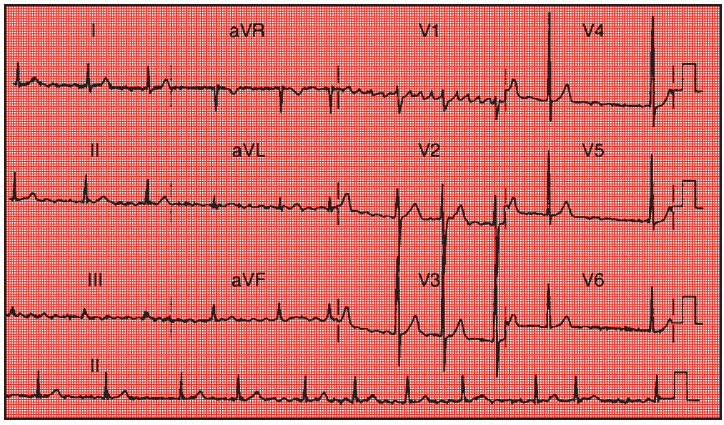


FIG. 4.60. Coarse atrial fibrillation is evident in V1, 2 years later (SC; 8/4/95).

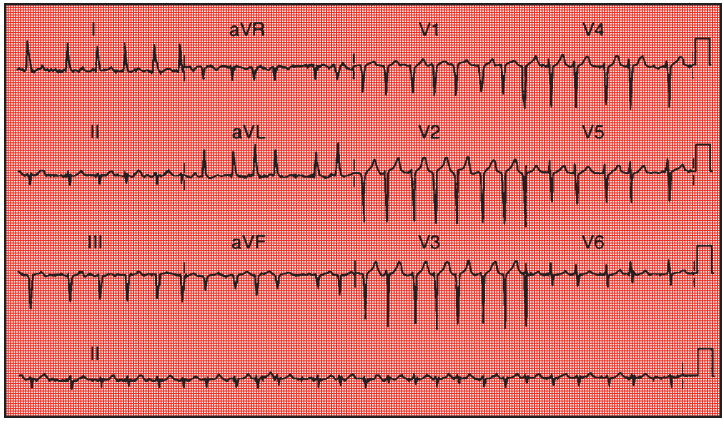


FIG. 4.61. Rapid atrial fibrillation. There are narrow complexes, an irregular ventricular response and a ventricular rate of 156 bpm. There are no P waves (FJ; 12/6/99).

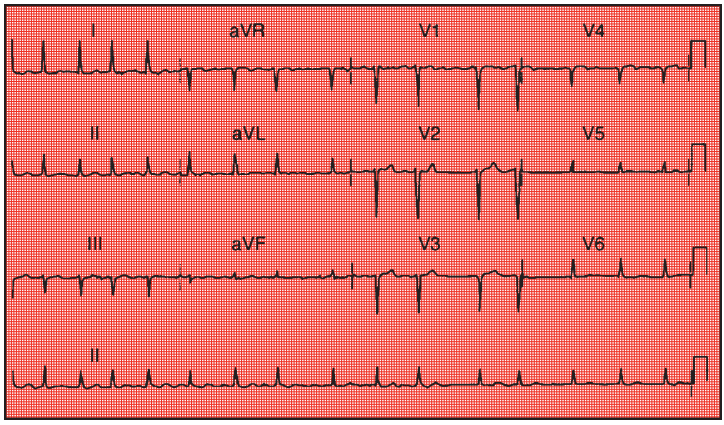


FIG. 4.62. This elderly woman with stable angina presented with atrial fibrillation that was controlled medically (Mrs H; 4/6/98).

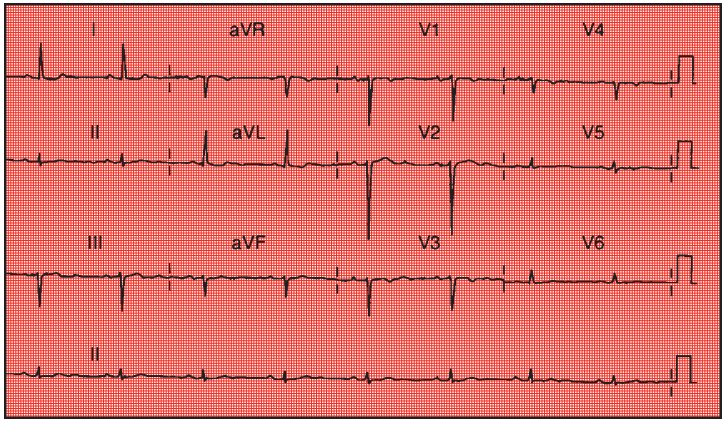


FIG. 4.63. Three weeks later after medical cardioversion to sinus rhythm. Note similarity of complexes and electric axis (Mrs H; 30/6/98).

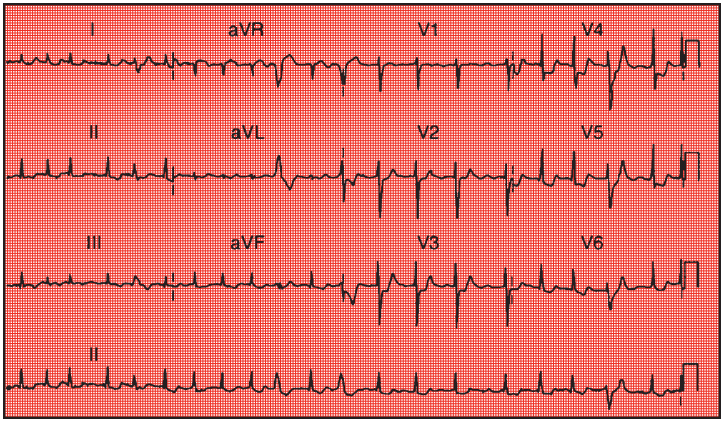


FIG. 4.64. Episodic atrial fibrillation in an elderly patient with ischaemic heart disease and a dual chamber pacemaker. Atrial fibrillation causes marked precordial ischaemic changes (Rate related). Pacing is inhibited by the ventricular rate (SB; 15/6/98).

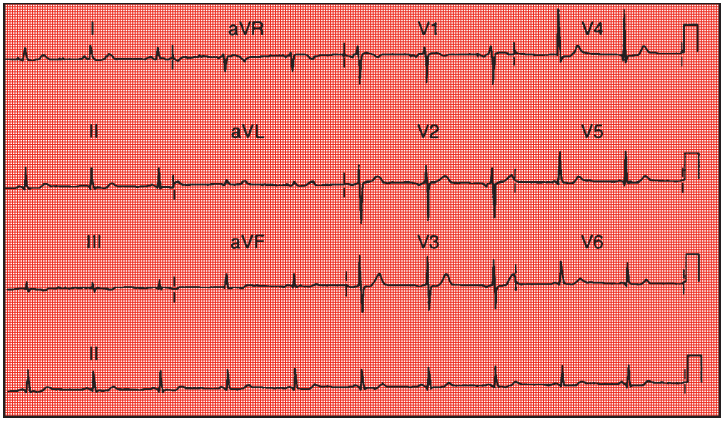


FIG. 4.65. Two weeks later after medical cardioversion. A pacing signal is seen in lead V1. There are no residual ischaemic changes (SB; 1/7/98).

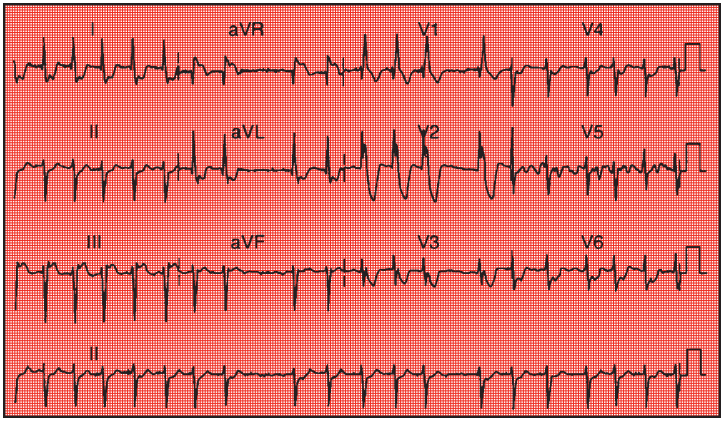


FIG. 4.66. ECG from an elderly lady 10 years after bypass surgery. There is sudden onset of atrial fibrillation, with marked ischaemic changes in leads I, aVL and V6, and right bundle branch block. The trace shows left axis deviation (bifascicular block) (SB; 25/9/99).

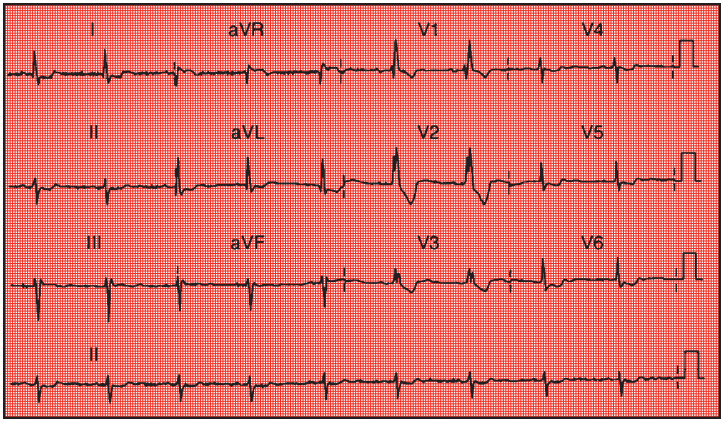


FIG. 4.67. Reversal with oral amiodarone 24 h later. The patient is restored to sinus rhythm; there is right bundle branch block with minimal antero-lateral ST ischaemic changes (SB; 26/9/99).

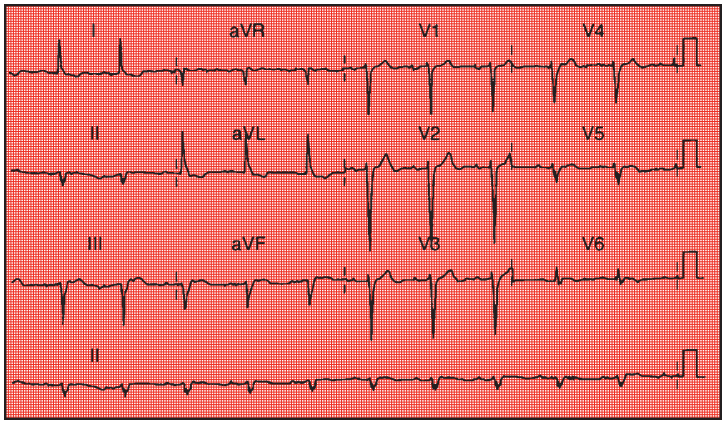


FIG. 4.68. ECG from a middle-aged woman with silent inferior infarction. There is normal sinus conduction with widened QRS complexes (intraventricular conduction defect) (Mrs J; 11/11/97).

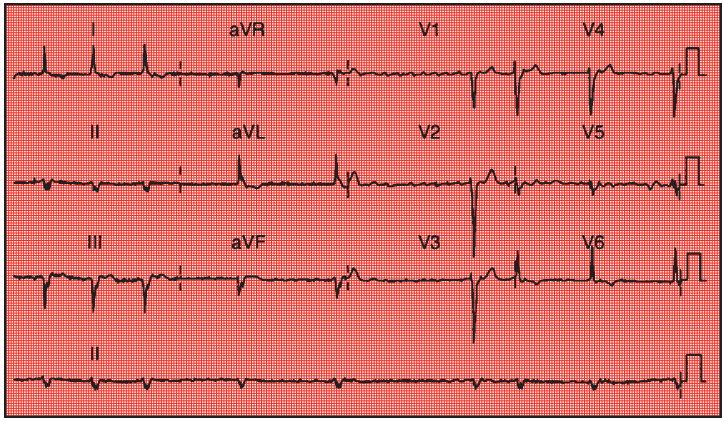


FIG. 4.69. Six months later there is onset of very slow atrial fibrillation. The remaining ECG pattern is unchanged. The patient was successfully restored to sinus rhythm with electrical cardioversion (Mrs J; 26/5/98).

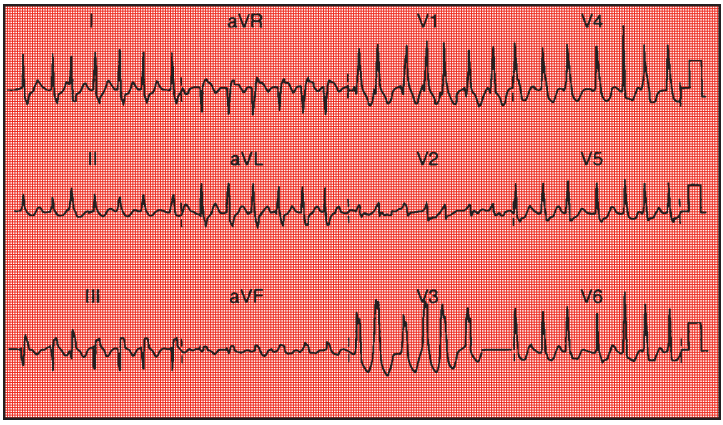


FIG. 4.70. Broad complex tachycardia. This could be confused with ventricular tachycardia (Mr K; 14/7/98).

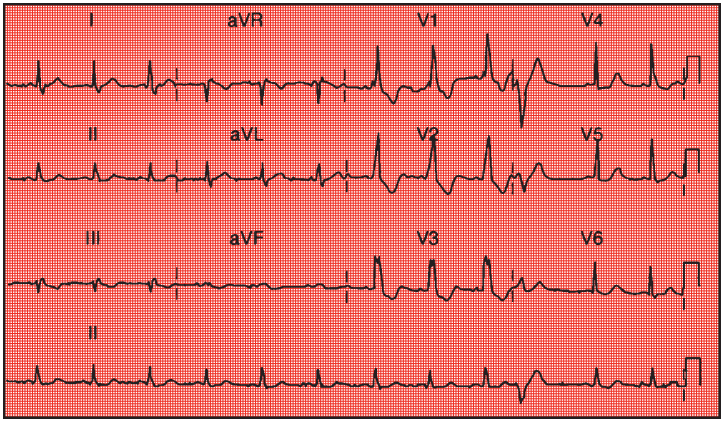


FIG. 4.71. ECG from the same patient showing sinus rhythm with right bundle branch block. The diagnosis was atrial fibrillation (Mr K).

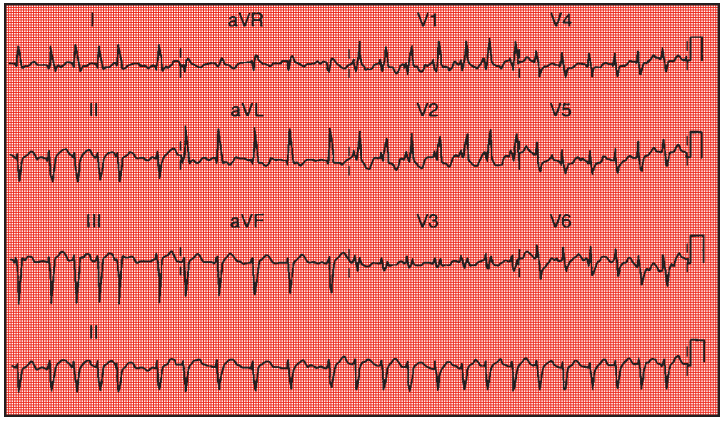


FIG. 4.72. This 82-year-old patient was admitted in rapid atrial fibrillation with right bundle branch block and left axis deviation. The ventricular rate is clearly irregular. Medication was instituted with amiodarone. The patient was already on atenolol, digoxin and verapamil (SF; 14/10/98).

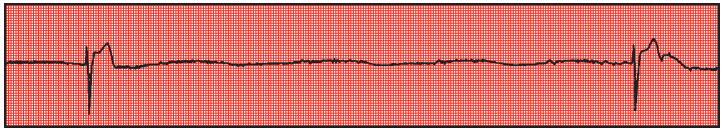


FIG. 4.73. The effect of overmedication. There is ventricular asystole and P waves are just discernible. (SF; 20/10/98).

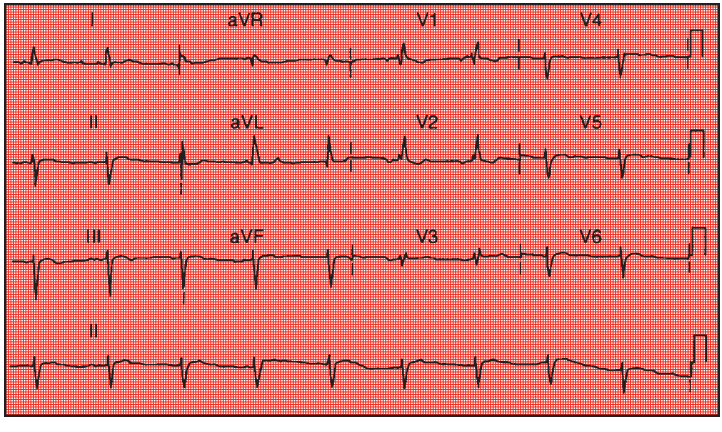


FIG. 4.74. The patient reverted to sinus rhythm 4 days later, without ill effects. This series shows that accumulative antiarrhythmics can have profound negative chronotropic effects (SF; 24/10/98).

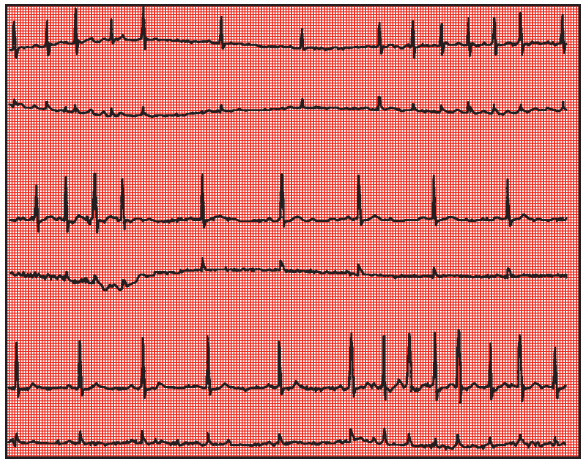


FIG. 4.75. Twenty-four-hour ambulatory Holter monitoring showing classical so-called sick sinus syndrome (tachycardia/bradycardia).

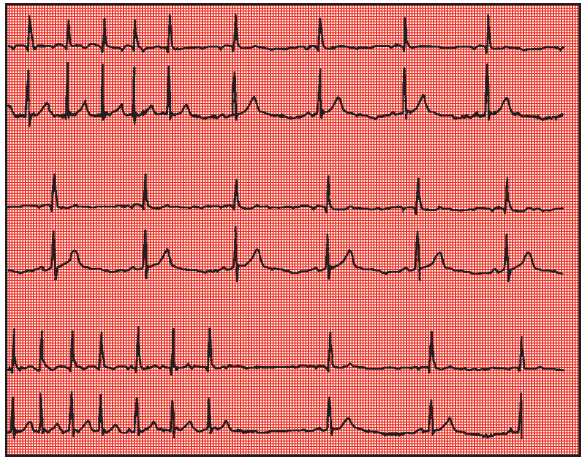


FIG. 4.76. These continuous strips show the patient going from fairly rapid atrial fibrillation into sudden bradycardia.

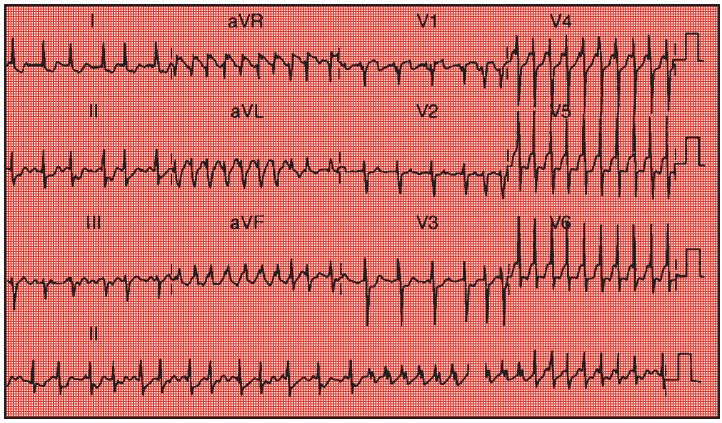


FIG. 4.77. Rapid atrial fibrillation with marked lateral ischaemic changes. The rhythm strip at the bottom (lead II) shows a short run of bundle branch block. This is a rate dependent aberration (Mrs L; 6/5/93).

to the lungs from the right atrium, or to the systemic circulation from the left atrium or rarely, across a patent foramen (paradoxical emboli). The prevalence of atrial fibrillation is 0.5% in people aged 50–59 years, rising to 8.8% in people aged 80–90 years. There is a sixfold increase in thromboembolic phenomena (5% per annum). It is known that 36% of strokes in the elderly are the result of atrial fibrillation. Cardiac output is reduced from between 25 and 50% (depending on the function of the heart) giving rise to symptoms of fatigue and breathlessness.

Proper anticoagulation with warfarin reduces the risk of stroke by up to 70%. The target is an international ratio (INR; a measure of prothrombin time) of 2.0–2.5 and so at present warfarin is the drug of choice, even in the elderly, although aspirin or clopidogrel are an alternative in low risk patients (those with normal left atrial dimension, no congestive cardiac failure or mitral disease).

The aim of treatment is to restore sinus rhythm if possible. This can be done medically or by electrical cardioversion. Ideally the patient should be given warfarin for at least 3 weeks and started on oral amiodarone with a loading dose (200 mg tid) for several days, continuing with 200 mg daily for up to 4 weeks. The success

rate is variable. The best results are obtained with recently developed atrial fibrillation in the presence of normal left ventricular function and normal left atrial dimension (4 cm) on echocardiography. IV flecainide is also useful, but is to be avoided after acute myocardial infarction.

Electric cardioversion is the next step. If there is any doubt of a left atrial thrombus (i.e., large left atrium, poor LV function) a transesophageal echocardiogram examination should be performed prior to electric cardioversion. The patient's INR should be between 2.0 and 2.5. The usual starting output is 200 J, moving up to 360 J. Several shocks may have to be administered under general anesthesia. Warfarin should be continued for at least 3 weeks. There is some evidence that atrial "stunning" persists, so late emboli can occur. In dire emergencies, when anticoagulation with warfarin is not possible, an intravenous bolus of 5,000 units of heparin should be administered half an hour before electric cardioversion.

Pharmacological Management of SVT Arrhythmias

Acute

Supraventricular Re-entry Tachycardia

Suitable pharmacological agents include intravenous adenosine, verapamil, disopyramide, flecainide, beta-blockers (esmolol), or amiodarone through a central vein. Digoxin can be administered intravenously (slowly) or intramuscularly. A new drug, dofetilide, has a promising profile.

Atrial Flutter, Fibrillation, Atrial Tachycardia

Treatment is the same as that for supraventricular re-entry tachycardia, with the exception of adenosine, which is not suitable.

WPW Syndrome with SVT

Suitable pharmacological agents include IV flecainide, disopyramide, beta-blockers (sotalol) and amiodarone. It is important to note that verapamil and digoxin are contraindicated; they slow down AV conduction and can therefore accelerate the rhythm.

Chronic

Suitable oral preparations include digoxin, verapamil, sotalol, disopyramide, amiodarone, flecainide and propafenone.

Special Precautions

Note that verapamil and beta-blockers slow the heart rate and verapamil increases digoxin toxicity. Amiodarone can cause severe bradycardia alone or in combination with other medication (Fig. 4.73). Long-term treatment with amiodarone is best avoided because of thyroid, liver and lung toxicity. Warfarin will react with a number of preparations, and INR monitoring is essential.

Prevention

After successful medical or electrical cardioversion the following drugs can prevent recurrences: flecainide, sotalol, proprafenone, amiodarone and quinidine.

Quinidine was previously used regularly. It is not favored now as it can generate a form of VT (torsade de pointes) giving rise to so-called quinidine syncope. However, it is still used in the USA and is worth trying if all else fails. New drugs that are presently being evaluated include azimilide and dofetilide.

Mechanical Management of SVT Arrhythmias

DC Conversion

DC conversion is covered in Chap. 8.

Implantable Atrial Defibrillators

Implantable atrial defibrillators will sense the onset of SVT arrhythmia and deliver a DC shock to the atrium. These devices are presently being evaluated.

Ablation

Electrophysiological study (EPS) is required to map the abnormal circuits. Once established, radiofrequency energy is applied to one of the abnormal pathways, "burning it off," and disabling the re-entry circuit. This technique is particularly useful in re-entry supraventricular tachycardia, in particular WPW syndrome, offering a very high percentage of cure at a very low risk.

Surgery

The "Maze" operation involves creating "corridors" within the atria to interfere with the propagation of the abnormal impulses. This procedure is more favored in the United States. This can be performed at surgery or by catheterization technique.

AV Nodal Ablation

AV nodal ablation is useful in intractable symptomatic atrial fibrillation. It requires permanent pacemaker implantation as patients become entirely dependent on pacing.

Pulmonary Vein Ablation

In some patients atrial arrhythmias originate in the pulmonary vein, where ablation can be beneficial. However this technique is not without risks, although it is now increasingly used in patients with intractable atrial fibrillation.

VENTRICULAR ARRHYTHMIAS

Ventricular Extrasystoles

Ventricular extrasystoles are also known as ventricular premature beats (VPBs). These are *broad complexes* (QRS over 120 ms) that are premature by definition (hence the compensatory pause), therefore the coupling interval (interval between ectopic and previous beat) is short. VPBs can occur in isolation, arising from the right ventricle (giving rise to a left bundle branch block pattern) or the left ventricle (producing a right bundle branch block pattern). They can be benign or associated with myocardial damage (for example ischemia, infarction, cardiomyopathies and myocarditis). They can be caused by drugs such as digoxin, antiarrhythmics, cocaine, excessive caffeine, alcohol, catecholamines (anxiety) and bronchodilators. VPBs are described as *unifocal* when they arise from a single source, all being similar in configuration, *or multifocal*, when they originate from different foci and have a varying appearance.

Ventricular Bigeminy

In ventricular bigeminy, every sinus beat is followed by a ventricular extrasystole (Fig. 4.78).

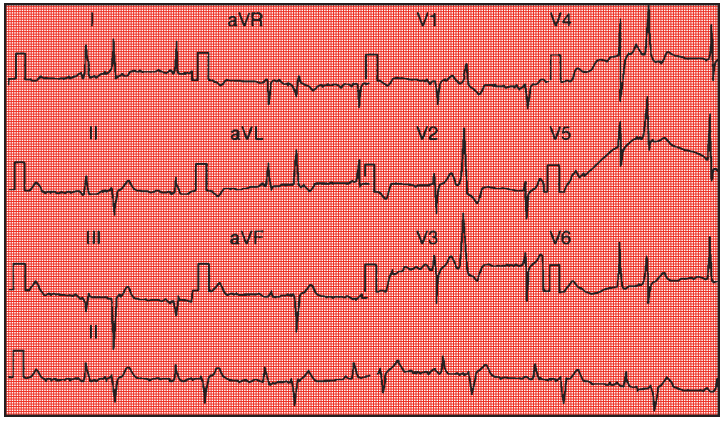


FIG. 4.78. Ventricular extrasystoles (or premature beats). These are clearly different to sinus beats as they originate in the ventricles as opposed to atrial extrasystoles (premature atrial beats) which would use the same conducting pathway as the sinus conducted beats. In leads V1 and V6 these extrasystoles have a right bundle branch block configuration and originate from the left ventricle. The rhythm strip at the bottom of the page shows that every second beat is a ventricular extrasystole and this would therefore be called ventricular bigeminy (HH; 27/3/95).

Ventricular Trigeminy

In ventricular trigeminy every second sinus beat is followed by a ventricular extrasystole.

Ventricular Couplets

Ventricular couplets are defined as two consecutive VPBs (Fig. 4.79).

Ventricular Salvo

A ventricular salvo constitutes more than two consecutive VPBs.

Ventricular Parasystole

Ventricular parasystole is uncommon. VPBs occur regularly and are not related to the dominant rhythm. This syndrome is characterized by variable coupling intervals, mathematically related RR intervals and the presence of fusion beats. No treatment is indicated.

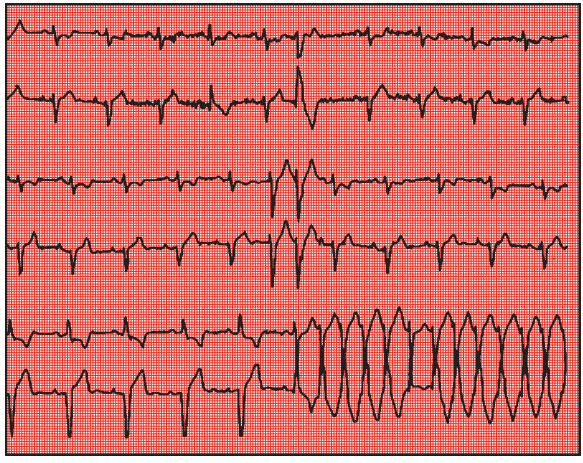


FIG. 4.79. Twenty-four-hour ambulatory Holter monitor traces from a patient who had undergone bypass surgery. The two simultaneous channels in the upper strip show one ventricular extrasystole in the middle followed by a compensatory pause. The middle strip shows two consecutive ventricular extrasystoles. This a ventricular couplet. The bottom strip shows a run of normal sinus rhythm with a ventricular extrasystole falling just at the end of the T wave of the sinus beat, setting off a run of ventricular tachycardia (MS).

Management of Ventricular Premature Beats

If there is no underlying cardiac pathology VPBs are best ignored. The patient should be advised to reduce consumption of caffeine, bronchodilators, alcohol and tobacco. In the presence of unpleasant symptoms a course of mexilitene, disopyramide, flecainide or propafenone is recommended. Beta-blockers are also useful. All can prove beneficial. If innocent, VPBs usually disappear during a stress test. In the presence of underlying cardiac pathology (for example metabolic disorders or drug overdose or acidosis) they should be treated accordingly. If the VPBs are related to ischaemic heart disease (indicated by worsening during a stress test), they may respond to anti-anginal medication. Particular care is required after myocardial infarction as ventricular extrasystoles may herald ventricular tachycardia (VT); this responds to intravenous lignocaine. If VPBs are related to dilated cardiomyopathy or congestive cardiac failure, amiodarone becomes the drug of choice (it has no negative inotropic effect).

Ventricular Tachycardia

These are broad complex tachycardias arising from the right or left ventricles with QRS complex duration usually over 120 ms. Most are caused by re-entry mechanisms, some from enhanced automaticity. They can be sustained (lasting over 30 s) or non-sustained. They are described as monomorphic when each complex has the same configuration or polymorphic when the QRS pattern twists across the baseline (torsade de pointes). VT can degenerate into VF, particularly in the context of acute myocardial infarction or dilated cardiomyopathy with lethal consequences. However, there are also benign ventricular tachycardias.

In ventricular tachycardia, atrial activity remains unaffected, hence it is imperative to obtain a 12-lead ECG during an attack. The P wave is generally of a slower rate compared to the ventricular arrhythmia. They appear independently (AV dissociation), at regular intervals, and are distinctly recognizable, deforming QRS or T waves when they coincide. Retrograde P waves are seen when the ventricle activates the atria. As this can occur in up to 45% of cases of VT, AV dissociation is clearly not invariable. Fusion beats (simultaneous activation when atrial and ventricular impulses coincide) or captured beats (when the atrial stimulus captures the AV node and stimulates the ventricles during a VT) also point to a diagnosis of ventricular tachycardia. Examples of ventricular tachycardia are shown in Figs. 4.80–4.88.

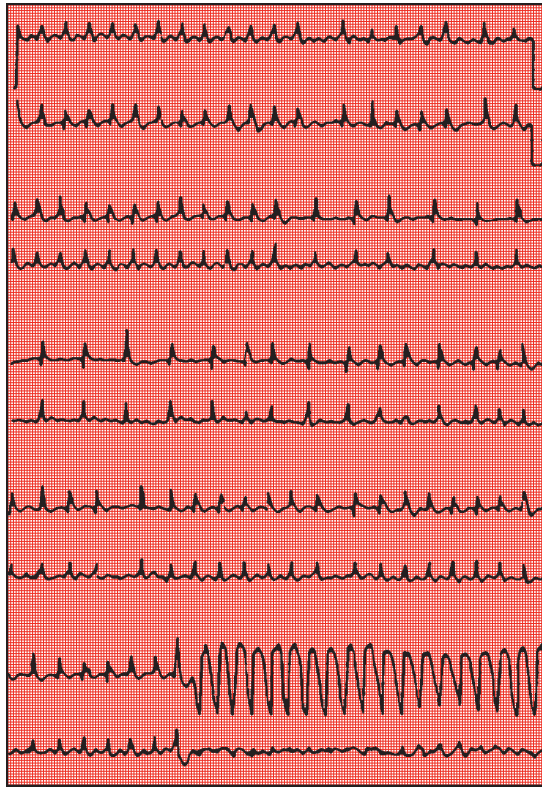


FIG. 4.80. Twenty-four-hour Holter monitor trace. At the bottom of the continuous strip, a ventricular extrasystole occurs at the end of a T wave, setting off a run of ventricular tachycardia. The patient is in AF.

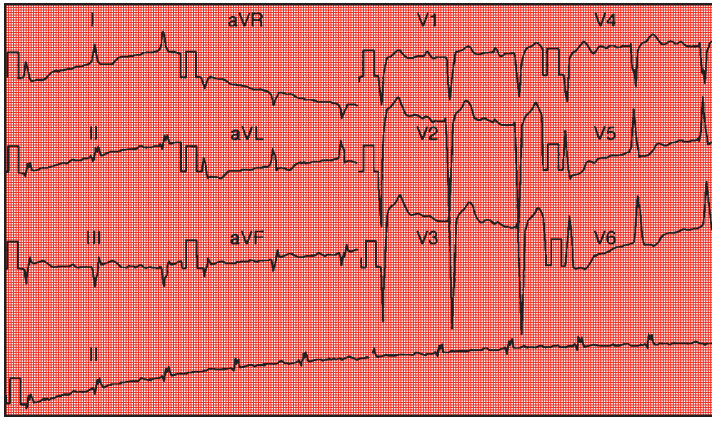


FIG. 4.81. ECG trace showing sinus rhythm, left bundle branch block, a prolonged PR interval and inferior Q waves (WL; 1980s).

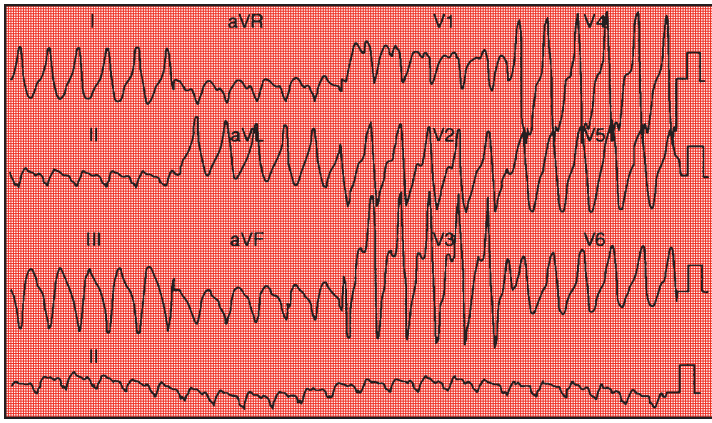


FIG. 4.82. The same patient developed ventricular tachycardia. The patient had bypass surgery and developed an ischaemic cardiomyopathy and eventually died as a result of VT/VF. Here the VT occurred after treatment with flecainide (pro-arrhythmic effect) (WL; April 1990).

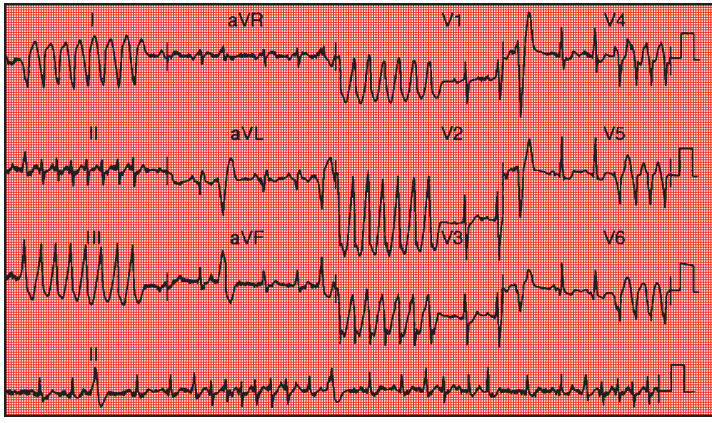


FIG. 4.83. This patient presented with syncopal attacks caused by runs of self-remitting (non-sustained) ventricular tachycardia. He had a myocardial infarction many years previously. After electrophysiological study he was treated medically with amiodarone and metoprolol (JG; 30/12/92).

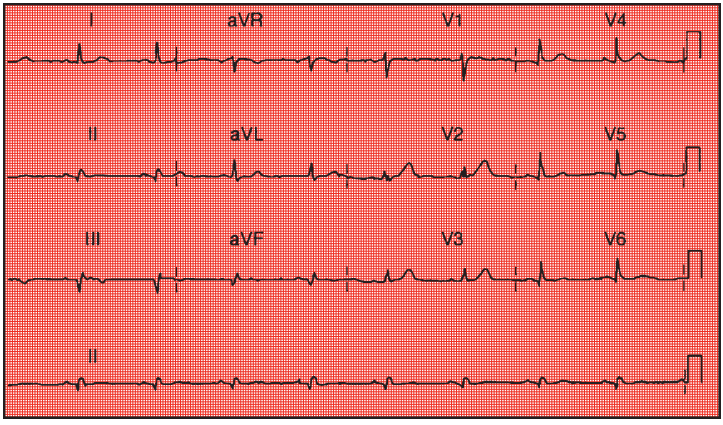


FIG. 4.84. The same patient 6 years later is in sinus rhythm with an old inferior Q wave infarction. He had no further symptoms on this regimen. Note the prolonged QT interval resulting from medication (JG; 7/10/98).

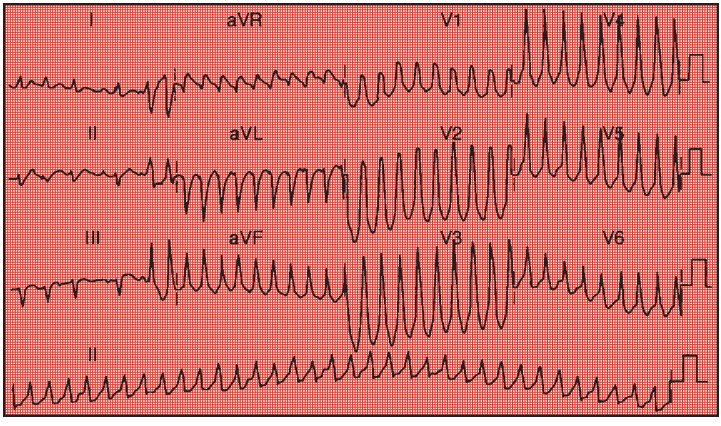


FIG. 4.85. ECG from an 89-year-old woman with ischaemic heart disease and chronic congestive cardiac failure showing ventricular tachycardia treated medically. Note that the patient is in sinus rhythm at the beginning of the trace. P waves are seen clearly in lead II. Ventricular tachycardia develops during the recording. Note the different rhythm strip from lead II at the bottom of the page. The run of VT shows so-called concordance. All the precordial leads show a similar axis. This is pathognomonic of ventricular tachycardia (EL; 10/10/89).

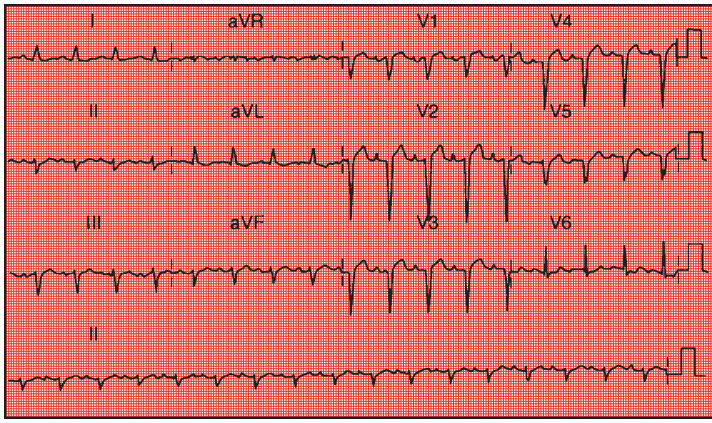


FIG. 4.86. Four days later a routine ECG shows sinus conduction with widespread anterolateral Q waves (EL; 14/10/89).

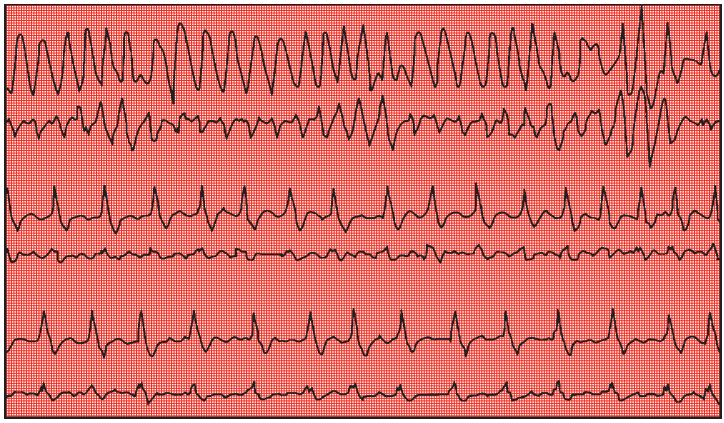


FIG. 4.87. Non-sustained ventricular tachycardia recorded on a 24-h tape. The patient spontaneously returns to sinus conduction.

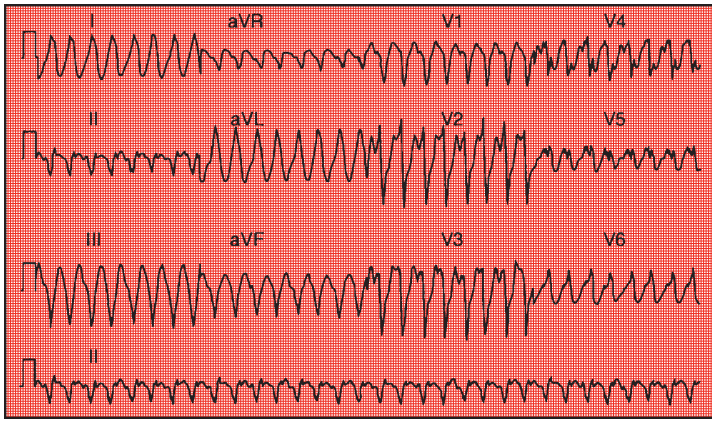


FIG 4.88. ECG taken during ventricular tachycardia. There are wide QRS complexes. Retrograde P waves are seen in lead V1 independent of ventricular activity (CG; 12/4/99).

Other useful pointers

1. Left rabbit ear: the R wave in V1 is taller than the R′ wave.
2. Concordance: can be positive or negative when all the complexes in the precordial leads point either up or down.
3. As a general rule, 90% of broad complex tachycardias (QRS complexes over 120 ms) will be ventricular tachycardias.
4. Wide QRS complexes can also occur with:

- Bundle branch block or aberration
- An accessory pathway (e.g., WPW syndrome)
- Slow conduction through the bundle of His and Purkinje system (toxic, metabolic, myocardial damage)

Causes of Ventricular Tachycardia

Ventricular tachycardia is usually caused by organic heart disease, most commonly after acute myocardial infarction. In addition, it can occur in chronic ischaemic heart disease, left ventricular aneurysm, cardiomyopathies (dilated and hypertrophic), myocarditis related to drugs or as a result of antiarrhythmic medication (proarrhythmia). It can be related to hypokalemia, low serum magnesium, hypoxia, or catecholamines (bronchodilators). Frequent ventricular premature beats often herald the development of ventricular tachycardia, particularly in acute ischaemic situations.

Specific Ventricular Tachycardias

Slow VT or accelerated idioventricular rhythm (in red)

Slow VT is seen after acute myocardial infarction. The ventricular rate is usually below 120 bpm, and is monomorphic. AV dissociation is evident. The condition is benign and self-remitting; no treatment is indicated.

Right Ventricular Outflow Tract Tachycardia

The condition is not generally dangerous, is exercise-induced and usually not sustained. It is a re-entrant tachycardia that originates in the right ventricular outflow tract without obvious underlying pathology. Right ventricular outflow tract tachycardia is characterized by left bundle branch block and right axis deviation. Treatment is with beta-blockers or ablation.

Fascicular Ventricular Tachycardia

Fascicular ventricular tachycardia is uncommon, usually arising from the posterior fascicle of the left bundle, but occasionally from the anterior fascicle. There is no evidence of underlying heart disease. It affects young people, is exercise-induced and re-entrant. Posterior fascicle ventricular tachycardia is characterized by right bundle branch block with left axis deviation, and that which arises from the anterior fascicle produces a right bundle branch block with right axis deviation. Treatment is with verapamil or ablation.

Arrhythmogenic Right Ventricular Dysplasia

The condition occurs in the presence of a dilated right ventricle caused by lack of muscle fibers (for example Uhl's syndrome). There is a left bundle branch block pattern. It affects young people and is exercise-related.

Torsade de pointes

Torsade de pointes produces an ECG that is polymorphic, undulating, and associated with a prolonged QT interval (Fig. 4.89).

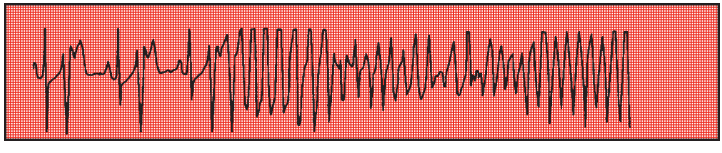


FIG. 4.89. Torsade de pointes. Note the "undulating" effect.

Prolonged QT Interval

Acquired

An acquired prolonged QT interval is associated with certain drugs, for example quinidine, disopyramide, amiodarone and sotalol. It can be associated with bradycardia (for example in sick sinus syndrome or AV block). In addition, it can be seen with hypokalemia, hypomagnesemia and rarely with erythromycin, or tricyclic antidepressants. Treatment is by withdrawal of the culprit drug, and avoiding bradycardia. Magnesium can be given as 2 g (20 ml of 10% solution) intravenously over 2 min or by a continuous infusion of 3–10 mg/min. DC defibrillation is indicated if ventricular fibrillation develops.

Congenital

Congenital prolonged QT intervals occur in Romano–Ward and Jervell–Lange–Nielson syndromes. Treatment is with beta-blockers, magnesium and pacing.

Symptoms of Ventricular Tachycardia

These are cerebral, consisting of dizziness, lightheadedness, and syncope.

Ventricular Fibrillation

Ventricular fibrillation is usually fatal unless quickly rectified. Cerebral ischemia occurs within 20 s, and irreversible brain damage at 3 min. Occasionally it is self-limiting. Treatment consists of a blow to the praecordium, DC shock (200 J), intravenous lignocaine, repeat DC shock if necessary, and intravenous bretylium tosylate for recurring VF.

Sudden Death

Sudden death (SD) is usually caused by ventricular fibrillation associated with coronary artery disease or, less frequently, cardiomyopathies. In 5–10% of survivors no cause is found.

Brugada Syndrome

Brugada syndrome is associated with a family history of sudden death and is most prevalent in South-East Asia and Japan. The characteristic ECG abnormality consists of a right bundle branch block pattern with ST elevation in leads V1–V3, occasionally with left axis deviation. Drug treatment is not beneficial. An implantable defibrillator is at present the best option.

Treatment of Ventricular Tachycardia

Acute

An intravenous lignocaine bolus (50–100 mg) should be followed by an intravenous infusion (procainamide is a valid option). DC shock should be administered if the patient is hemodynamically compromised (BP below 80 mmHg systolic). Intravenous amiodarone is given into a central vein, but it may take several hours to be effective.

Chronic

Beta-blockers should be given for non-sustained VT. Sotalol, mexiletine, disopyramide, amiodarone and implantable cardiac defibrillators are other options.

ADDITIONAL NOTES

Electrophysiological Study

This is an invasive procedure undertaken under local anesthesia. Several electrode catheters are introduced from a femoral vein and artery into the heart using complex ECG apparatus.

"Mapping" of abnormal conduction pathways is obtained. Stimulation of the heart is achieved through pacing wires to reproduce arrhythmias.

This can give clues as to which medication might be appropriate in the treatment of a particular arrhythmia.

EPS is a prerequisite prior to ablation treatment.

Sinus Node Recovery Time (Overdrive Suppression Test)

This is used to assess the normality of the sinus-atrial node. The sensitivity of the test is not very satisfactory.

HISTORICAL NOTES

P Brugda, Contemporary Spanish Cardiologist, Heart Institute, Aalst, Belgium.

NJ Holter (1914–1983) USA. Provided technology for continuous ECG monitoring known as Holter monitor.

KEY MESSAGES

- SVT – Supraventricular tachycardia
- VT – Ventricular tachycardia

- VF – Ventricular fibrillation
- Atrial flutter
- Fibrillation
- Tachycardia
- Re-entry
- Junctional tachycardia
- Atrial and ventricular premature beats
- Specific ventricular tachycardias
- Narrow and broad complex tachycardias
- Torsade de pointes
- Prolonged QT interval
- Ventricular parasystole
- Bigeminy
- Trigeminy
- Couplets
- Salvo

Chapter 5
Hypertrophy

LEFT VENTRICULAR HYPERTROPHY

Increased voltages are detected over left ventricular leads.

A. Limb leads. When the R waves in lead I plus the S wave in lead III measure over 25 mm. When the R wave in aVL is over 11 mm or over 20 mm in aVF. Also when the S wave in aVR is greater than 14 mm.
B. Precordial leads. When the R wave in leads V5 or V6 plus the S wave in V1 measure over 35 mm. When the tallest R wave plus the tallest S wave measure over 45 mm, or when the R wave in V5 or V6 is greater than 26 mm.

Q waves in leads V1–V3 can indicate septal hypertrophy. LVH with strain has the same ECG appearance as LVH, with additional ST depression and T wave inversions in leads V4–V6, caused by underlying subendocardial ischemia (Figs. 5.1–5.5).

Note that lean people with normal hearts have increased voltages.

Causes of LVH

Hypertension, aortic valve disease, mitral incompetence, hypertrophic cardiomyopathy, and coarctation.

RIGHT VENTRICULAR HYPERTROPHY

Dominant R waves are seen in leads V1–V3 with T wave inversion (constituting RV strain). There is right axis deviation over 110° provided the QRS complex is under 120 ms.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_5,

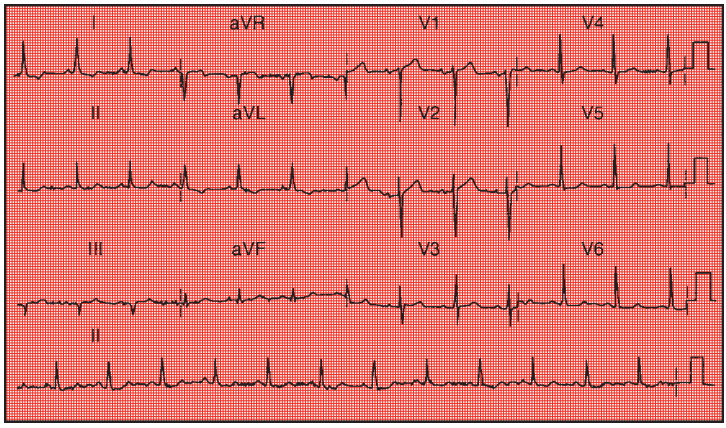


FIG. 5.1. ECG from an elderly woman with moderate aortic stenosis (GT; 28/8/90).

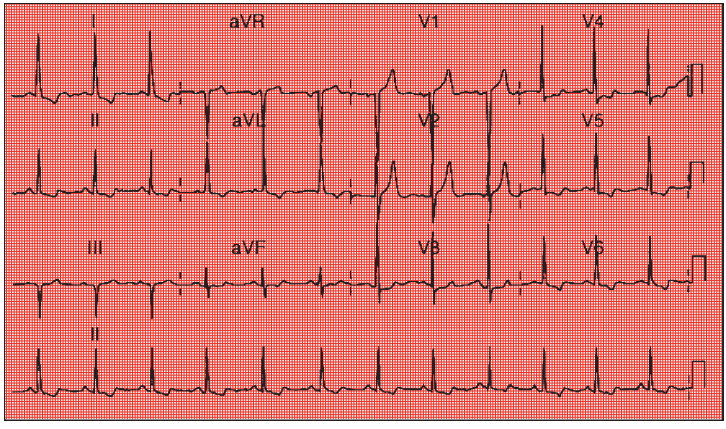


FIG. 5.2. Seven years later there is severe aortic stenosis with left ventricular voltages and strain pattern. The patient had her aortic valve replaced (GT; 18/6/97).

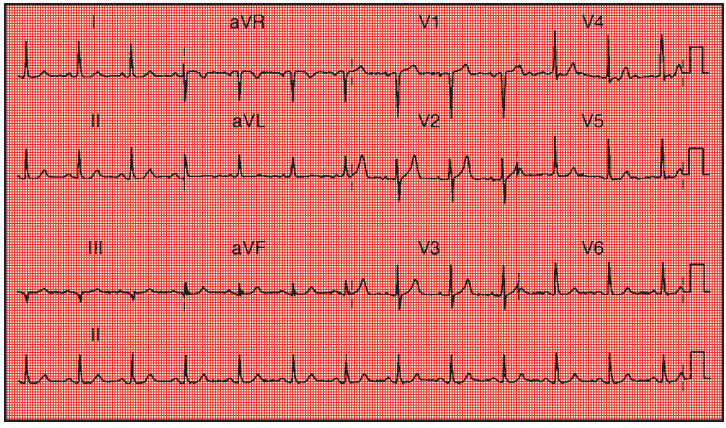


FIG. 5.3. Almost 2 years later the ECG appears remarkably normal. Biphasic P waves in lead V1 indicate residual left atrial hypertrophy (GT; 4/5/99).

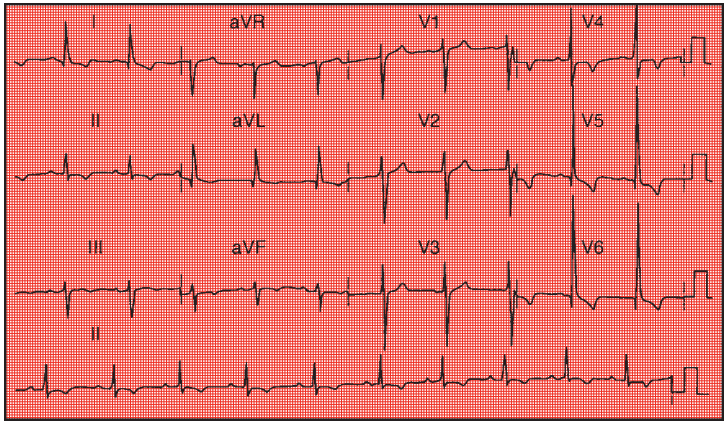


FIG. 5.4. ECG from a patient with severe aortic regurgitation showing LVH/strain pattern (HR; 10/2/88).

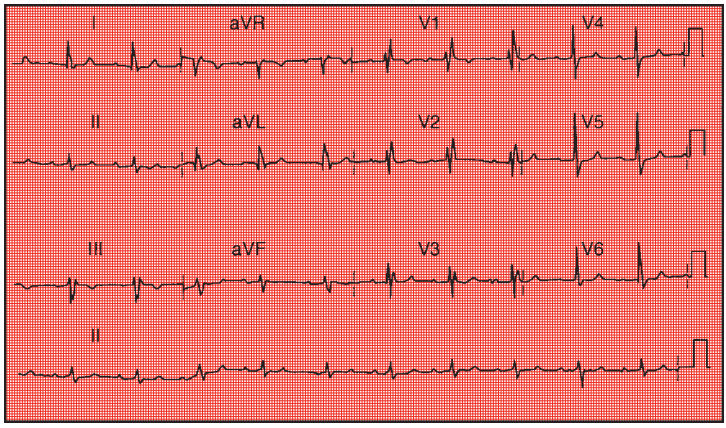


FIG. 5.5. There is marked improvement of the previous ECG changes 11 years later, after a successful aortic valve replacement. The patient has now developed right bundle branch with a prolonged PR interval (HR; 13/7/99).

Causes of Right Ventricular Hypertrophy

Pulmonary hypertension, pulmonary stenosis, Eisenmenger syndrome, acute pulmonary embolism and cor pulmonale.

LEFT ATRIAL HYPERTROPHY

Wide notched P waves are seen in lead II. When these reach more than 120 ms, the ECG is said to show "P mitrale" (Fig. 5.6). Biphasic P waves in lead V1 also indicate LAH (Figs. 5.7–5.13).

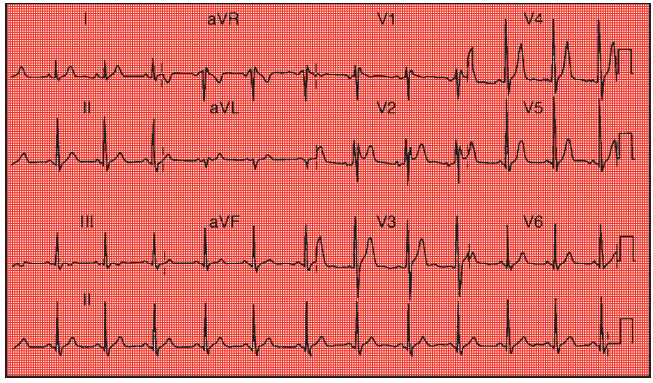


FIG. 5.6. ECG from a young patient with mild to moderate mitral stenosis with biphasic P waves in lead V1 and V2 (MK; 17/3/99).

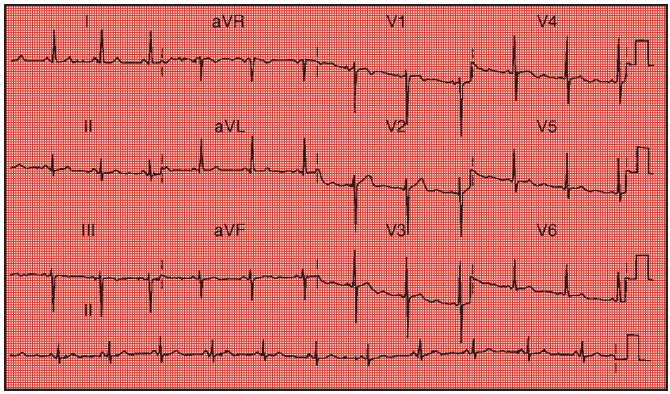


FIG. 5.7. Fairly normal electrocardiogram in an elderly woman with moderate aortic stenosis (JS; 21/11/95).

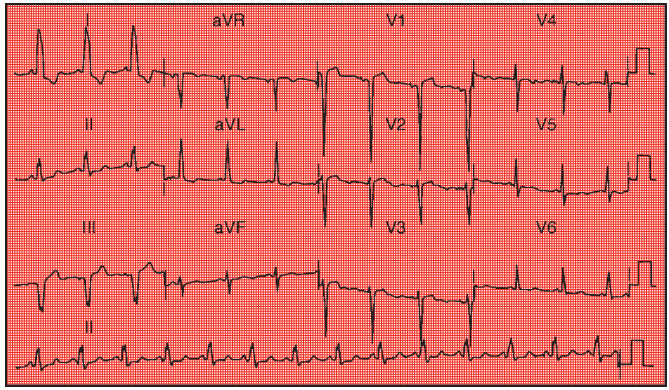


FIG. 5.8. Three years later there is left bundle branch block with left ventricular voltages and left atrial hypertrophy (biphasic P waves in lead V1). The patient now has severe aortic stenosis (JS; 10/7/98).

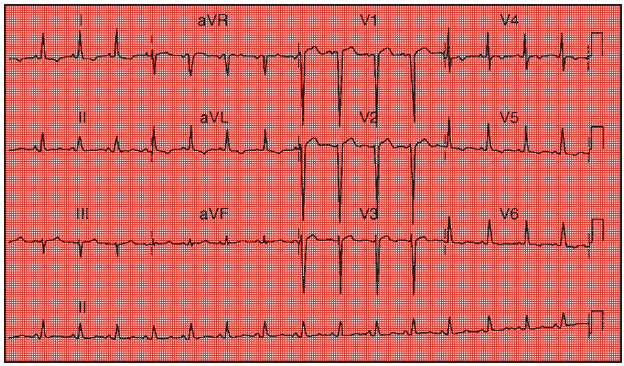


FIG. 5.9. Several weeks after aortic valve replacement, only left ventricular voltages and postoperative T wave inversions are visible (JS; 11/9/98).

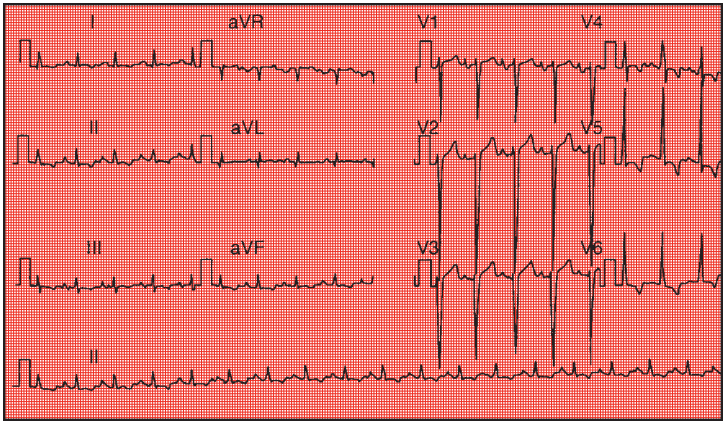


FIG. 5.10. Marked left atrial hypertrophy (biphasic P waves in lead V1) with LVH/strain pattern (HD; 27/3/95).

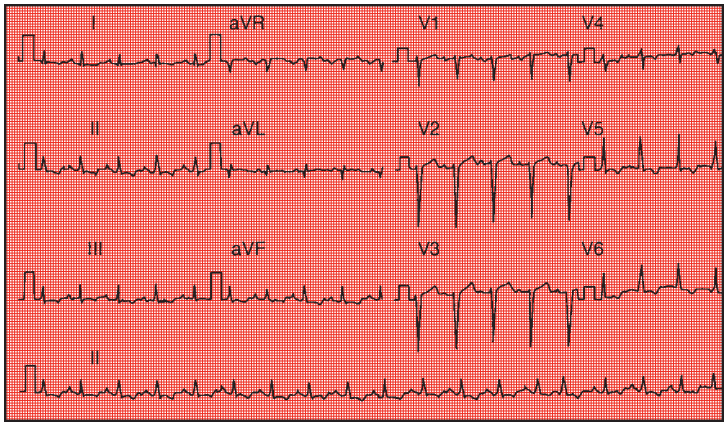


FIG. 5.11. Half-calibrated ECG from the same patient (precordial leads). The changes are much less evident. Always check calibration (HD; 27/3/95).

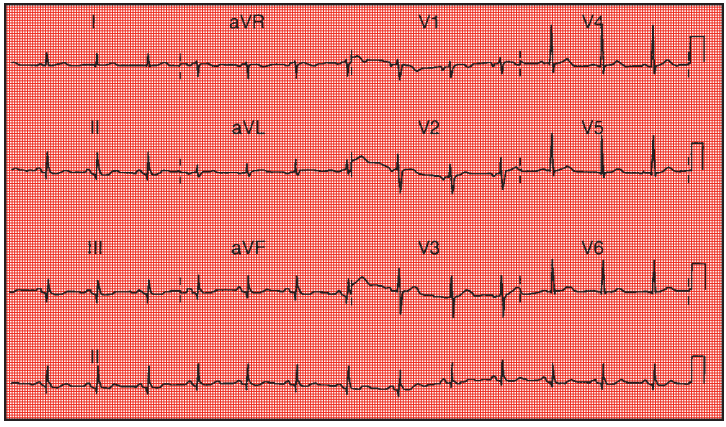


FIG. 5.12. Preoperative ECG prior to bypass surgery. The patient has been complaining of breathlessness. There are negative P waves in leads V1 and V2 indicating left atrial hypertrophy (HC; 10/10/97).

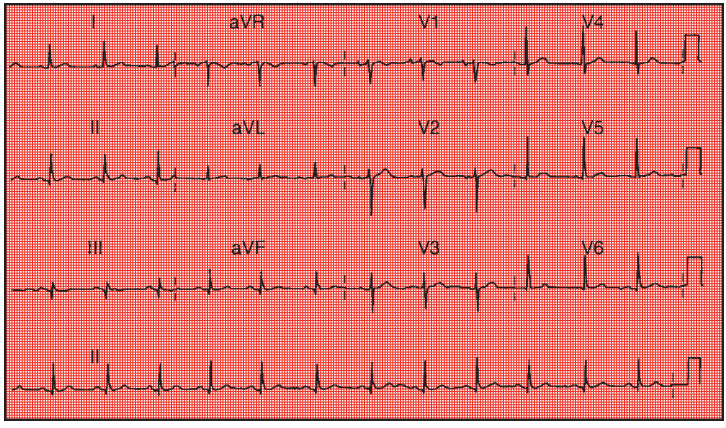


FIG. 5.13. Almost 2 years later the patient is much improved after bypass surgery. He no longer suffers from breathlessness and the P waves in leads V1 and V2 have returned to normal (HC; 22/11/99).

Causes of Left Atrial Hypertrophy

Causes include increased left ventricular end diastolic pressure, ischaemic heart disease, hypertension, aortic and mitral valve disease and congenital shunting.

RIGHT ATRIAL HYPERTROPHY

There are tall P waves present in leads II and VI. When these reach more than 2.5 mm (in lead II), the ECG is said to show "P pulmonale."

Causes of Right Atrial Hypertrophy

Tricuspid and pulmonary valve disease, right heart failure, pulmonary embolism, right ventricular infarction and cor pulmonale.

KEY MESSAGES

- Left ventricular hypertrophy (LVH)
- Right ventricular hypertrophy (RVH)
- Left atrial hypertrophy (LAH)
- Right atrial hypertrophy (RAH)

Chapter 6
Cardiomyopathies and Autoimmune Disorders

Cardiomyopathy is defined as a disease of the myocardium without other associated recognizable pathologies.

PRIMARY CARDIOMYOPATHIES

There are three groups of primary cardiomyopathies:

- Hypertrophic.
- Dilated.
- Restricted.
- NB: Noncompaction of LV is now classified as a cardiomyopathy. It consists of *trabeculations* seen on echo, MRI, or angiography. The condition is usually benign although arrhythmias, progression to heart failure, and sudden death (SD) have been described (ECG 323).

Hypertrophic (Obstructive) Cardiomyopathy

This inherited disorder is characterized by septal or apical hypertrophy and muscle cell disarray. The term obstructive refers to a left ventricular outflow tract gradient, which is not invariably present.

The prevalence of the condition is of the order of 1:500 population in the UK.

ECG findings are as follows:

- LAD in 10–30% of cases
- LVH in 50–75% of cases
- Precordial Q waves in leads V1–V3 are common
- Giant T wave inversions are common

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_6,

- SVT arrhythmia/AF are not infrequent
- VT and AV block can occur, all badly tolerated owing to *stiffness* of the left ventricle (diastolic dysfunction)

These are illustrated in Figs. 6.1–6.7.

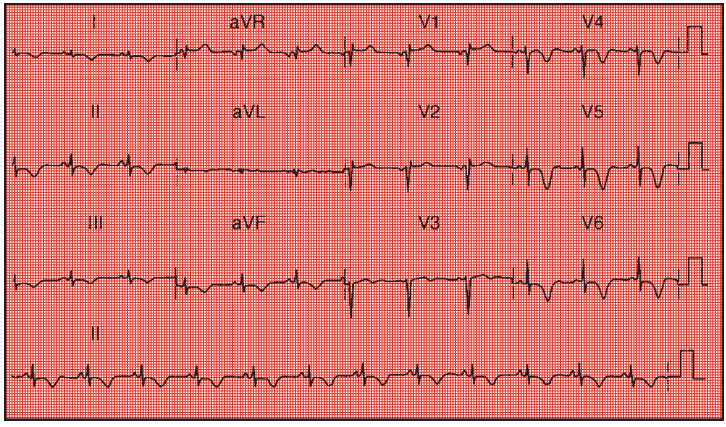


FIG. 6.1. ECG from a middle-aged patient with classical hypertrophic cardiomyopathy. Note signs of left atrial hypertrophy in lead V1 and pronounced lateral T-wave inversions (SS; 14/5/93).

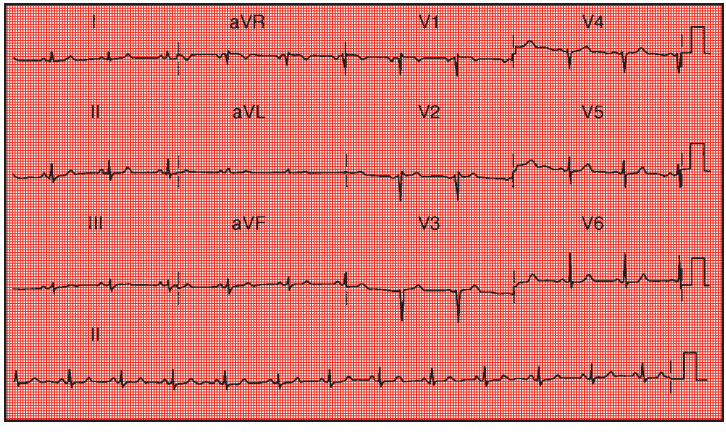


FIG. 6.2. One year later the patient is on medication and remarkable improvement is seen in ECG indices. Left atrial hypertrophy remains. These pronounced T-wave changes in hypertrophic cardiomyopathy can come and go, can be very confusing, and can lead to erroneous diagnosing of coronary artery disease (SS; 4/3/94).

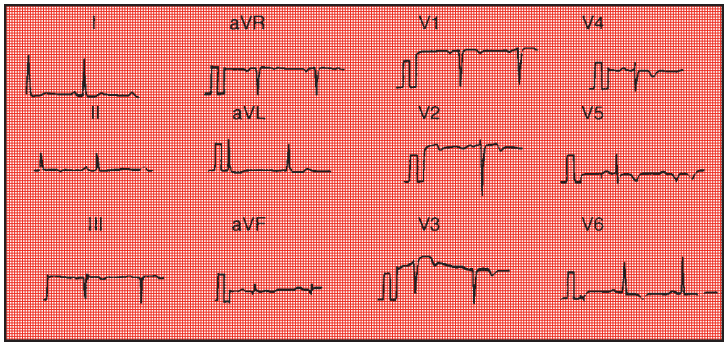


FIG. 6.3. Precordial T-wave changes are present in this ECG from a patient with documented hypertrophic cardiomyopathy (SS; 4/1/95).

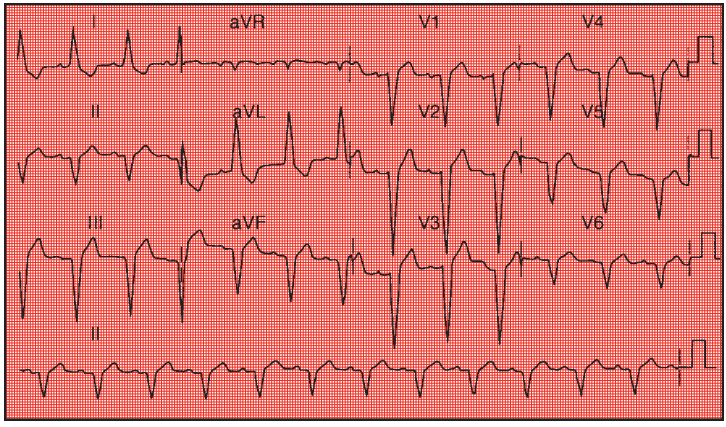


FIG. 6.4. Four years later, the patient has developed left bundle branch block in lead I and LAD. Note Q-waves V4–V6 (SS; 9/7/99).

Sudden death can occur, often in young athletes. Right ventricular HOCM is rare.

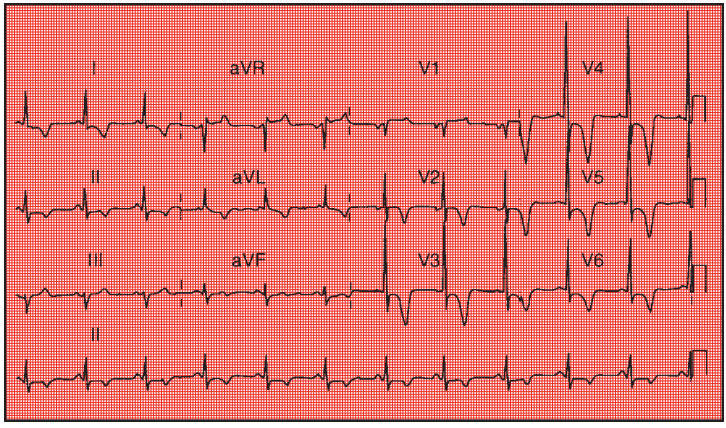


FIG. 6.5. Hypertrophic cardiomyopathy. Very abnormal downward-pointing T waves with increased left ventricular voltages. Note also LAH (MR; 19/8/98).

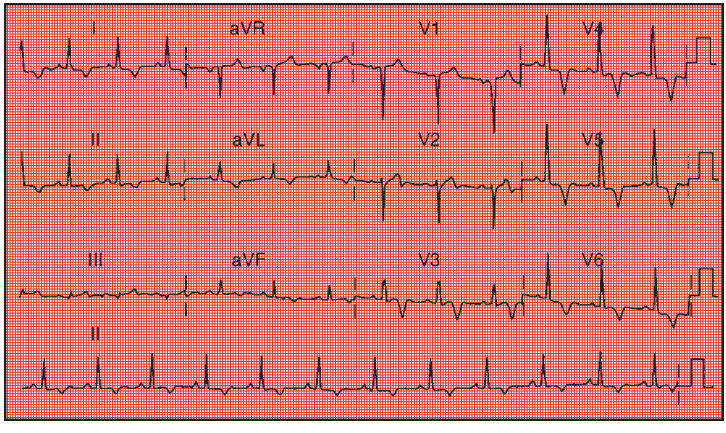


FIG. 6.6. Another example of a patient with hypertrophic cardiomyopathy. The T-wave inversions are often wrongly attributed to coronary artery disease. In fact the coronary arteries in hypertrophic cardiomyopathy are usually of large caliber and free from disease (PP; 20/7/98).

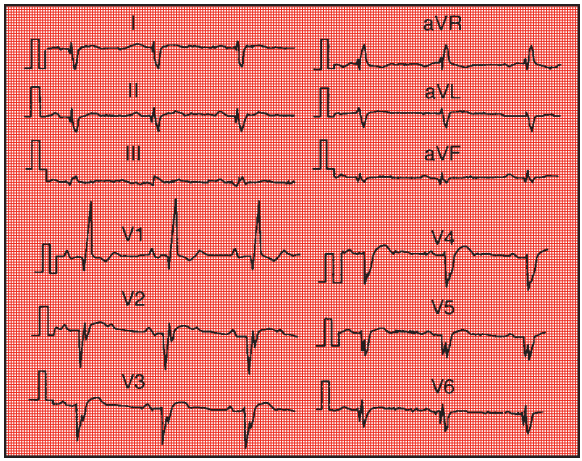


FIG. 6.7. Hypertrophic cardiomyopathy with normal coronary arteries. Septal Q waves are indicative of left ventricular hypertrophy. Right ventricular involvement is indicated by development of right axis deviation and right bundle branch block (WH; 7/8/97).

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy is characterized by an enlarged ventricle. ECG findings are listed below:

- LAH (notched P waves) is frequent.
- LVH in 30%.
- Small QRS complexes if marked fibrotic changes affect the ventricles.
- LBBB is common.
- ST/T wave changes are common.
- AF and VPBs are common.
- VT–VF–SD is seen at the end stage of the disease.

These are illustrated in Figs. 6.8–6.11.

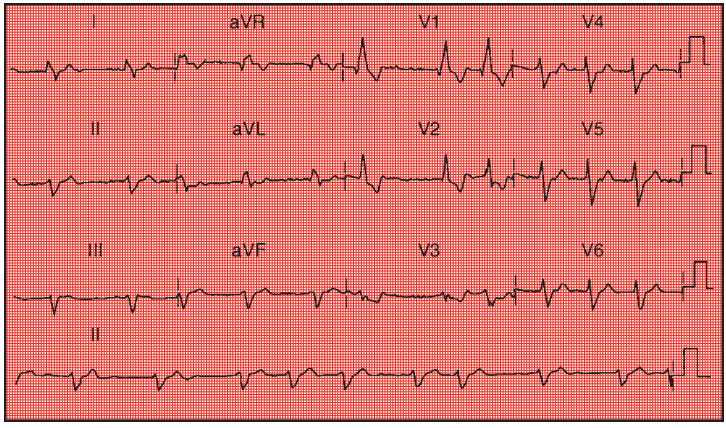


FIG. 6.8. Dilated cardiomyopathy in a renal failure patient receiving dialysis. There is atrial fibrillation, right bundle branch block, and left axis deviation (GU; 27/11/98).

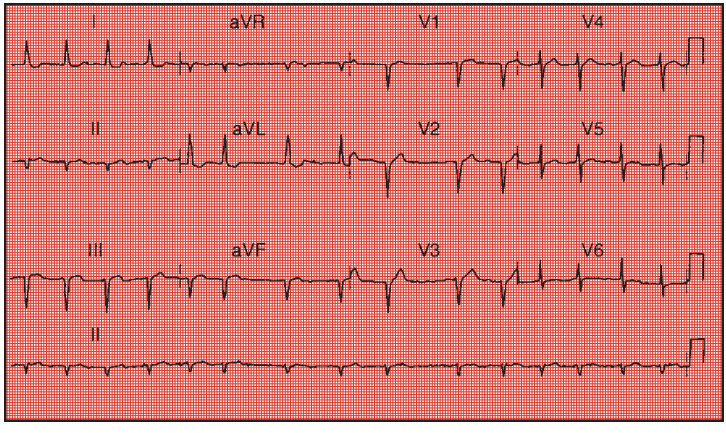


FIG. 6.9. This patient had a dilated cardiomyopathy and a normal coronary ateriogram. Note the inferior and anteroseptal Q waves. Atrial fibrillation is present (ES; 18/2/98).

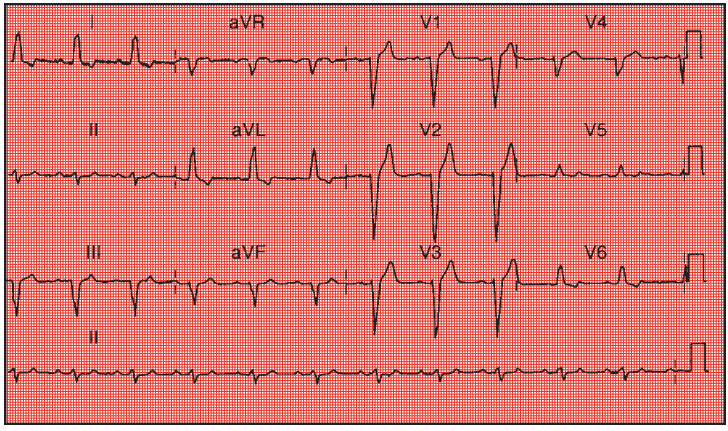


FIG. 6.10. Alcoholic dilated cardiomyopathy. There is left bundle branch block, left axis deviation, and inferior Q waves (JB; 27/7/95).

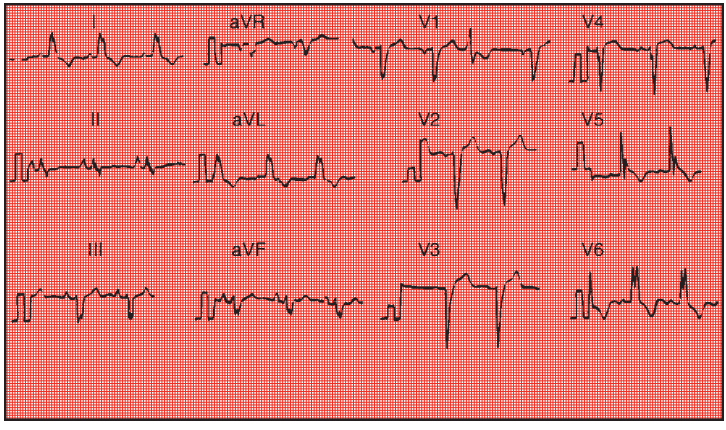


FIG. 6.11. Ischaemic cardiomyopathy caused by advanced coronary artery disease. There is left bundle branch block and marked left and right (V1–V2) atrial hypertrophy. P pulmonale in lead II denotes right atrial hypertrophy (AH; 13/9/95).

Restrictive Cardiomyopathy

Restrictive cardiomyopathy is the result of infiltration of the myocardium (for example, in amyloid, hemochromatosis, or fibrosis). There are no specific ECG changes although small voltages can occur.

SECONDARY CARDIOMYOPATHIES

Duchenne Muscular Dystrophy

Tall R waves are seen in lead V1 and there is a right bundle branch block and deep narrow Q waves.

Friedreich's Ataxia

ST/T wave changes are common. Right axis deviation is more frequent than left axis deviation. Tall R waves are seen in leads V1–V2. A short PR interval occurs in 25% of cases.

Myotonic Dystrophy

Conduction defects are common.

Myxoedema

Characteristic ECG changes include small voltages, sinus bradycardia, and T-wave changes.

Sarcoidosis

ECG changes include conduction defects (AV block), ectopic beats, and Q waves. There are no electrical signals from infiltrated areas (Fig. 6.12).

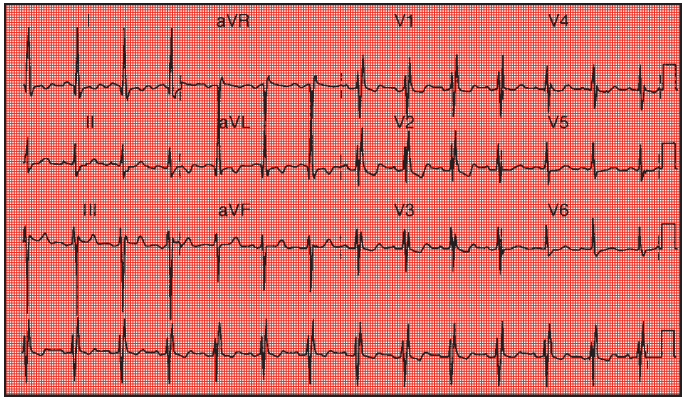


FIG. 6.12. Sarcoidosis with normal coronary arteries. Q waves are noted in leads I and aVL with T-wave inversions and right bundle branch block (TD; 5/10/95).

Amyloid

There are low voltages, left axis deviation, conduction and rhythm defects. The disease can involve the coronary arteries, giving rise to infarction.

AUTOIMMUNE DISORDERS

Scleroderma

Right ventricular hypertrophy is common (caused by pulmonary hypertension from lung involvement). Left ventricular hypertrophy can occur if the renal arteries are involved.

Systemic Lupus Erythematosis (SLE) and Polyarteritis Nodosa (PAN)

T-wave changes caused by pericarditis are seen. Vasculitis can give rise to myocardial infarction.

KEY MESSAGES

- Hypertrophic obstructive cardiomyopathy (HOCM)
- ECG often misrepresented with changes suggestive of ischaemic heart disease (IHD)
- Dilated cardiomyopathy (DCM)
- ECG can also mimic IHD

Chapter 7
Pericarditis, Myocarditis, and Metabolic Disorders

PERICARDITIS

Acute
Acute pericarditis is characterized by ST elevation (concave upward) in leads facing the effusion (Figs. 7.1–7.3). Usually there are widespread T-wave inversions. In addition, the ECG can exhibit P-wave changes, low-voltage QRS complexes, arrhythmias, and electrical alternans [alternating size of P, QRS, and T waves (Fig. 7.4)]. Lead aVR shows ST segment depression (reflecting heart cavity signals).

Chronic
Chronic pericarditis produces small voltages, T-wave inversions, arrhythmias, abnormal P waves, and right axis deviation (Fig. 7.5). Similar changes can be seen in hypothyroidism, hypopituitism, obesity, and emphysema.

MYOCARDITIS

Viral
ST/T wave changes and AV block are not uncommon (Figs. 7.6 and 7.7). Occasionally there are Q waves, mimicking acute myocardial infarction.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_7,

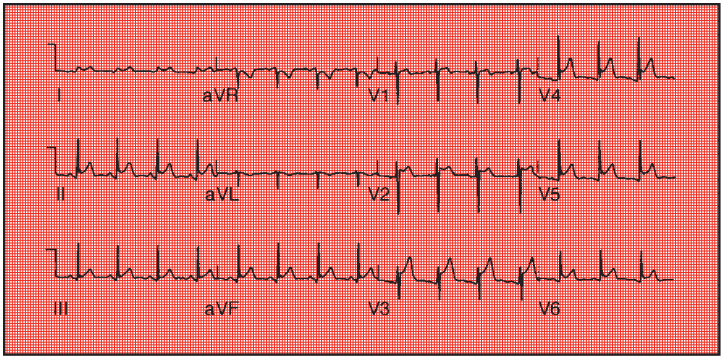


FIG. 7.1. ECG showing classical changes of pericardial effusion (concave ST elevation). This 23-year-old man had attempted suicide with a nail gun to his anterior chest wall. Courtesy of Dr. DH Spodick. Previously published in Clin Cardiol 1999; 22: 544.

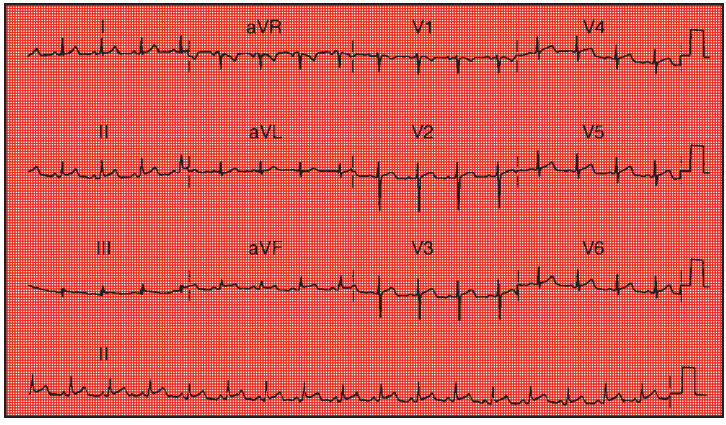


FIG. 7.2. This black patient presented with documented tuberculous pericarditis during pregnancy. Note the ST elevation particularly in leads II, III, and aVF and less so in leads V4–V6 (Mrs. M; 9/10/92).

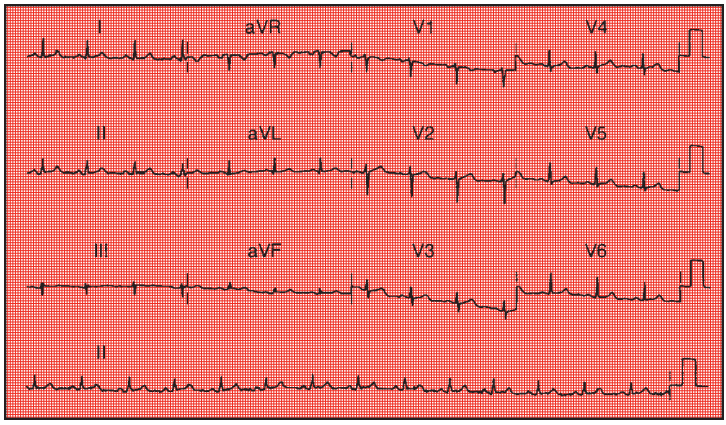


FIG. 7.3. Three years later after tapping and treating the ST elevations have disappeared (Mrs. M; 3/4/95).

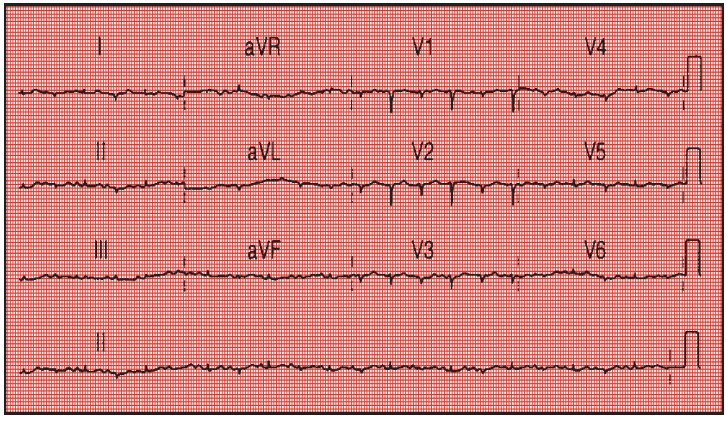


FIG. 7.4. This is a good example of electrical alternans, which is particularly well seen in leads V1 and V2 (FO; April 1997).

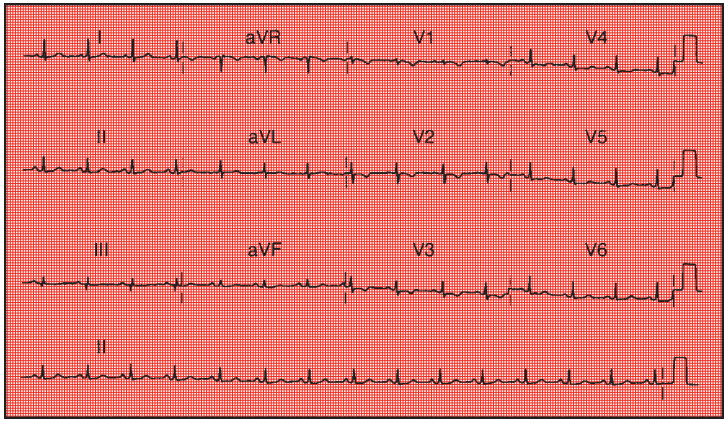


FIG. 7.5. Chronic nonspecific benign pericarditis. The patient had a normal coronary arteriogram. The ECG shows T wave and ST changes in the precordial leads (DS; 10/7/98).

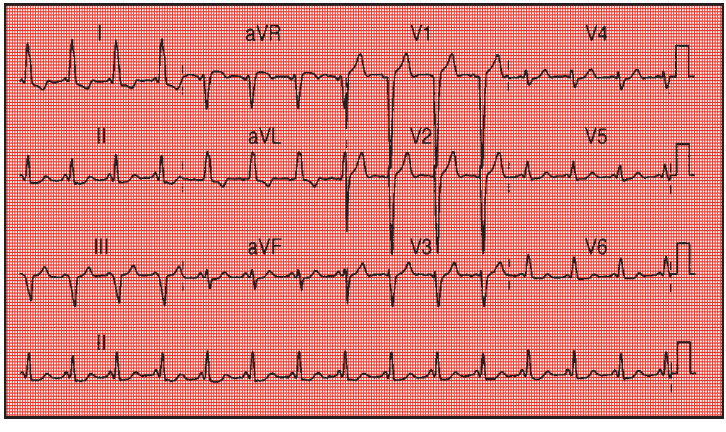


FIG. 7.6. This middle-aged woman was diagnosed with acute viral myocarditis. She presented with sudden onset left ventricular failure. The ECG shows left bundle branch block (FO; 8/4/97).

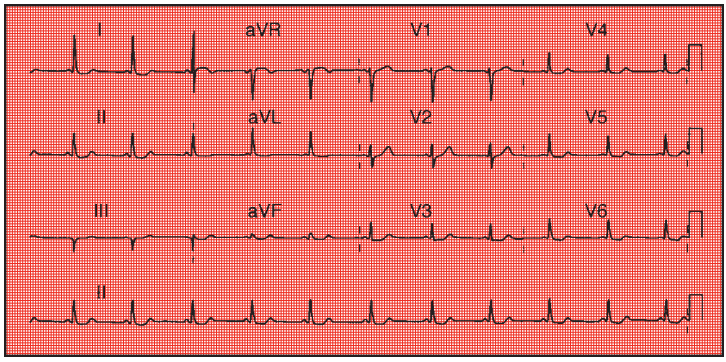


FIG. 7.7. This patient greatly improved with medication and the ECG changes returned to normal (FO; 9/7/97).

Rheumatic Fever

First-degree heart block is common with a prolonged PR interval. ST/T wave changes are often present. Junctional rhythm can develop.

Aids

AIDS-related myocarditis produces ST/T wave changes and bundle branch block on the ECG.

Chagas' Disease

T-wave changes are seen with right bundle branch block and ventricular premature beats (VPBs).

DRUGS, ELECTROLYTES, AND METABOLIC DISTURBANCES

Drugs

Digoxin

Digoxin causes ST depression with small T waves that are biphasic and/or negative. There is a short QT interval with an increase in size of U waves. In addition, digoxin can cause arrhythmias, ventricular premature beats, and junctional rhythms. There may be AV dissociation, AV block, sinoatrial dysfunction, or supraventricular arrhythmias (atrial trachycardia). An example of digoxin toxicity is illustrated in Figs. 7.8–7.10.

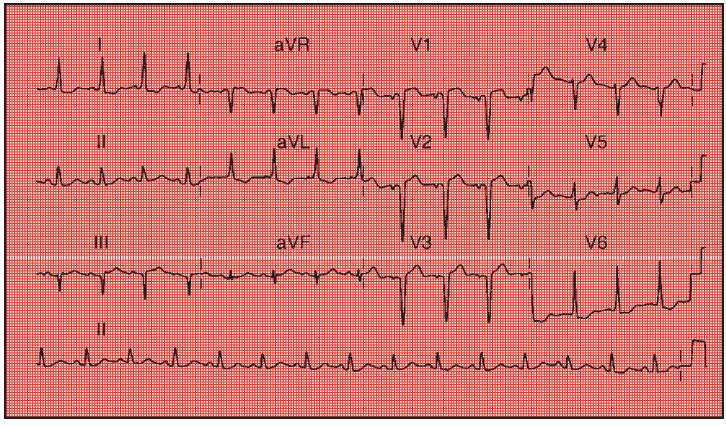


FIG. 7.8. This elderly woman with renal and heart failure and moderate aortic stenosis has incomplete left bundle branch block. There are ST changes in leads I, aVL, and V6 and left atrial hypertrophy (LB; 10/4/95).

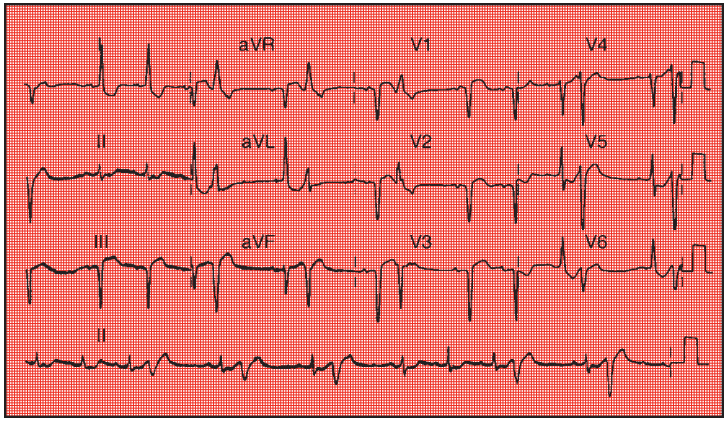


FIG. 7.9. Same patient with digoxin toxicity (toxic level 3.4). There are marked ST segment changes in leads I and aVL along with ventricular extrasystoles (LB; 22/4/95).

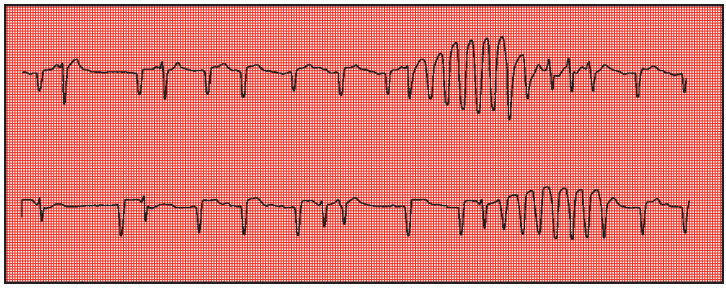


FIG. 7.10. The patient's condition worsened as she developed ventricular tachycardia and ventricular fibrillation. She died despite medication (LB; 23/4/95).

Quinidine

Quinidine produces small inverted T waves, ST depression, increased U waves, and prolonged QT interval. Similar changes are seen with disopyramide and procainamide.

Phenothiazine and Tricyclic Antidepressants

These drugs produce ECG changes similar to those caused by quinidine.

Lithium

Lithium causes T-wave changes and sinoatrial disorders (for example, sinus bradycardia and SA block).

Cocaine

Cocaine causes coronary spasm (marked by transient ST elevation) and myocardial infarction. Ventricular tachycardia and ventricular fibrillation can occur.

Electrolytes and Metabolic Disturbances

Hypokalemia

ECG changes include arrhythmias and AV block, ST depression, large R waves, wide QRS complexes, and P-wave changes.

Hyperkalemia

Arrhythmias develop: brady/tachycardia, or AV block, peaked T waves, small or absent P waves, wide and bizarre QRS complexes, and ST elevation.

Hypercalcemia
Hypercalcemia produces a shortened QT interval.

Hypocalcemia
Hypocalcemia produces a prolonged QT interval.

Hypermagnesemia
In hypermagnesemia there are prolonged PR intervals and QRS complexes. AV block can occur.

Hypomagnesemia
There are narrow QRS complexes, peaked T waves, and prominent U waves.

Altered Sodium Concentration
Changes in sodium concentration do not produce specific ECG changes.

Hypothermia
In hypothermia, the ECG shows sinus bradycardia. There are prolonged PR and QT intervals. J waves (a signal between QRS and ST segments) are present (Fig. B50).

KEY MESSAGES

- ECG changes related to *digoxin* medication can be very confusing
- Patients undergoing exercise tolerance tests should be off *digoxin* for at least 1 week

Chapter 8
Pacemakers, Implantable Cardiac Defibrillators, and Cardioversion

PACEMAKERS

Pacemakers were first introduced in 1958 by Senning as a treatment for complete heart block. They now have much wider applications. The system consists of a battery-operated generator (pacemaker) and electrodes (leads), which stimulate the heart. Pacemakers are inserted under local anesthesia. The electrode is placed into the right ventricle and/or right atrium, either by cutting down on a cephalic vein or by subclavian Seldinger technique. The position of the electrode is checked radiologically. Electronic parameters are obtained and need to be entirely satisfactory. A subcutaneous pocket is then created to position the generator. Very rarely, when venous systems prove inadequate, an epicardial approach is required, using a corkscrew electrode, through a small thoracotomy. The tip of the electrode (which makes contact with the myocardium) can be unipolar or bipolar.

Pacemakers are affected by high powered electromagnetic environments such as radar installations, microwave ovens, certain cordless telephones, and security screening at airports. Patients have to be warned about these hazards. Detailed international identify cards are provided and should be carried at all times.

PERMANENT PACEMAKERS

Unipolar leads are prone to extrinsic electrical signals, which can inhibit the ventricular output (myopotential interference). Bipolar leads are now preferred.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_8,

Nomenclature
Four letters conventionally describe the pacing mode.

First letter: Chamber paced
V Ventricle
A Atrium
D Dual chamber (atrium and ventricle)

Second letter: Chamber sensed
V Ventricle
A Atrium
D Dual chamber

Third letter: Mode of response
I Inhibited output
T Triggered output
D Both inhibited and triggered
O None

Fourth letter: Progammability
P Simple programmable
M Multiprogammable
R Rate variation in response to a sensed variable

The latter permits an increase in pacing rate. Different types of sensors are stimulated by physical exertion according to the patient's demands, and the generator is activated accordingly.

Indications for Permanent Pacing

Symptomatic Bradycardias

Permanent pacing is indicated for sinoatrial disease, slow atrial fibrillation (prolonged pauses lasting over 3 s), AV block, second degree or complete heart block (Adams–Stokes attacks), symptomatic fascicular block (bi- and trifascicular), carotid sinus syndrome with pauses of 3 s or more on carotid massage, and chronotropic incompetence.

Asymptomatic Bradycardias

A heart rate below 40 bpm, periods of asystole and atrial fibrillation/flutter with pauses over 3 s will benefit from pacemaker implantation.

TILT Testing Causing Profound Bradycardia

The test is undertaken to ascertain a patient's response to tilting at 60° for 45 min. Special attention is given to detect a drop in heart rate and/or in blood pressure. It is a test of autonomic nervous system integrity.

Other Indications

Some patients with hypertropic obstructive cardiomyopathy, dilated cardiomyopathy, and intractable congestive cardiac failure will benefit from dual chamber pacing. Hemodynamic improvement can result from careful programming of the AV sequential interval. Biventricular pacing or cardiac resynchronization therapy (CRT), i.e., pacing both ventricles is now a recognized mode of treatment for severe heart failure.

Pacemaker Syndrome

This refers to symptoms attributed to a fall in systolic pressure resulting from loss of AV synchrony, atrial contraction against a closed tricuspid valve, and retrograde P activation. This is seen with VV1 systems. Symptoms of syncope, dizziness, and fatigue can be remedied by upgrading the patient to a dual chamber system.

Types of Pacemakers in Use

VVI	One lead in the right ventricle
DDD	One lead to the right atrium, and one to the right ventricle
VDD	One lead to the right ventricle with a sensor in the right atrium (all on one lead)
AAI	Atrial pacing mainly for sick sinus syndrome
AV sequential	Pacing of the right atrium and right ventricle, thus adding atrial contribution to the overall cardiac output (DDD–VDD). This is physiologically superior, but cannot be used in atrial fibrillation.
Biventricular	One lead to the right ventricle, one lead to the coronary sinus. Presently used for improving left ventricular function in severely compromised patients. The lead to the coronary sinus stimulates the LV (CRT).

Figures 8.1–8.22 here were recorded from patients with various types of pacemakers.

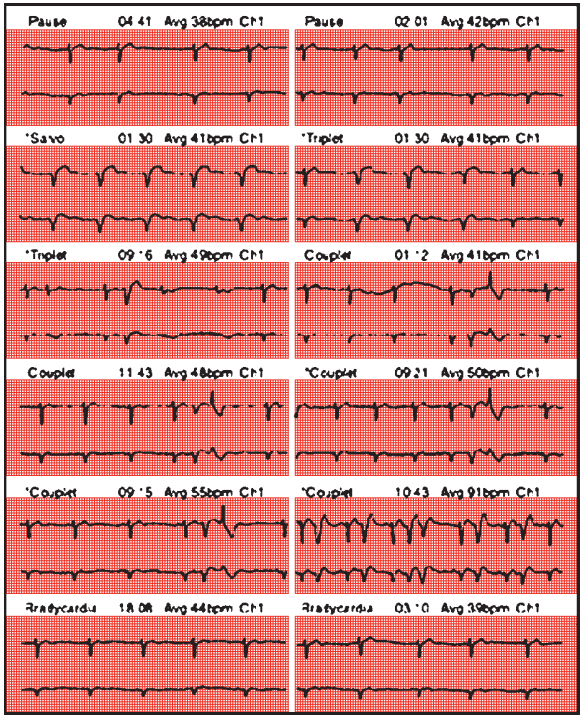


FIG. 8.1. This middle-aged patient with numerous TIAs was diagnosed suffering from sick sinus syndrome. The upper trace shows evidence of sinoatrial disease. There are episodes of atrial fibrillation lower down the recording (Mr. M; 1995).

Myopotential Inhibition

The generator senses pectoral muscle activity and ceases to function temporarily (seen with unipolar leads).

Hysteresis

This consists of programming the *escape interval* to be longer than the pacing interval. The patient's sinus rhythm can function at a lower rate than the pacing rate (sinus rhythm is more physiological than VV1 pacing).

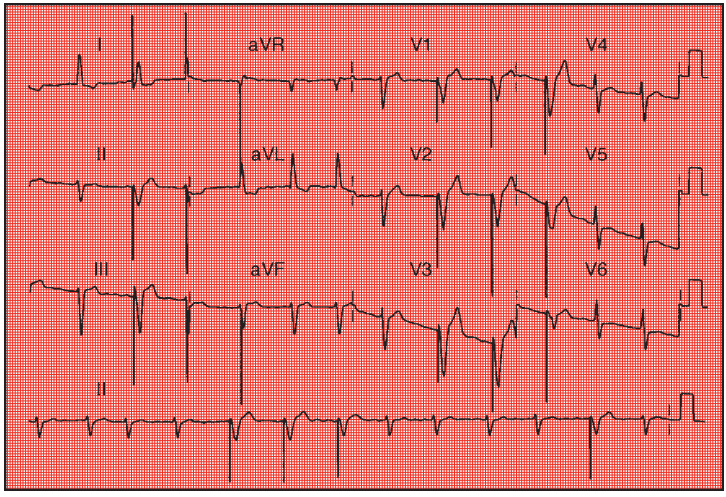


FIG. 8.2. The patient had a VV1 pacemaker. This trace shows variable demand pacing activity with underlying atrial fibrillation. No further TIAs were reported (Mr. M; 27/3/95).

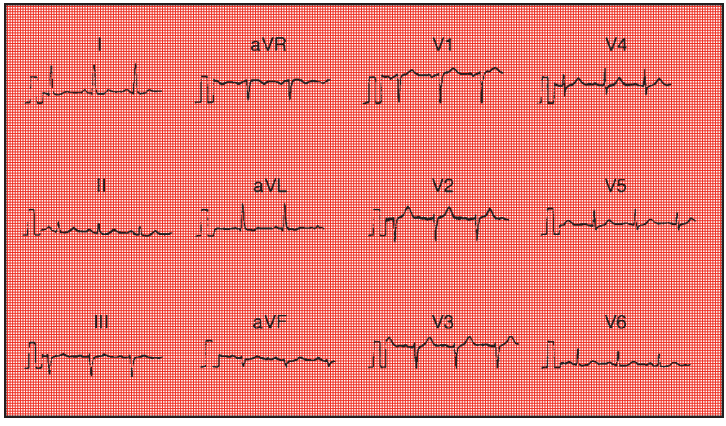


FIG. 8.3. Normal electrocardiogram from an elderly woman (WP; 28/9/93).

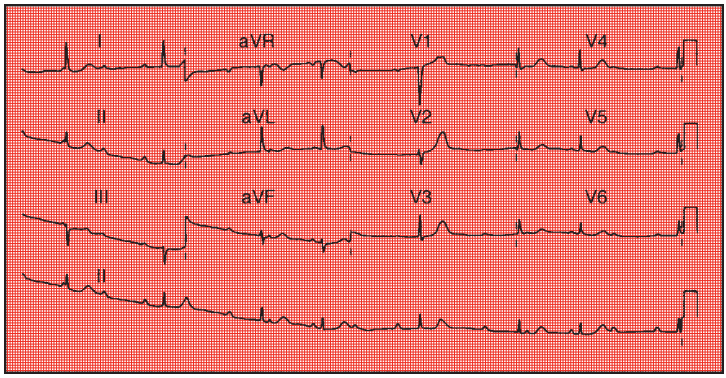


FIG. 8.4. The same patient 5 years later has complete heart block. The ventricular rate is 40 bpm and there are narrow QRS complexes. The *P* rate is about 90 bpm. There is AV dissociation (WP; 26/11/98).

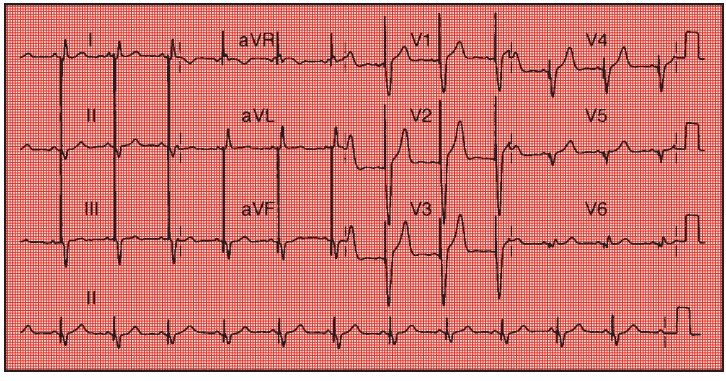


FIG. 8.5. After insertion of a VDD pacemaker, each P wave is followed by a pacing signal. The single electrode has a sensing device *floating* in the right atrium, which picks up atrial activity and then transmits a signal to the ventricles so that AV sequential activation is maintained (WP; 27/11/98).

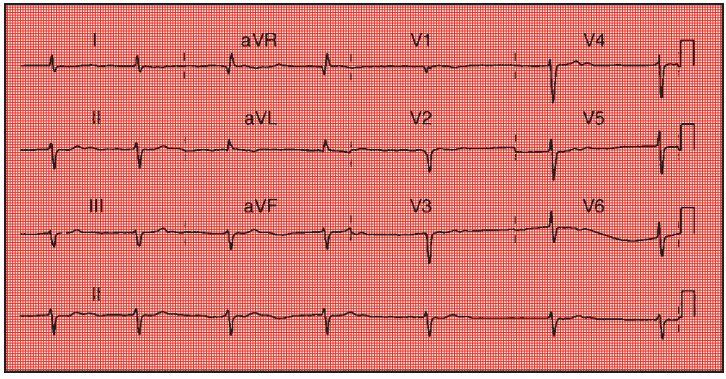


FIG. 8.6. Complete heart block with underlying atrial fibrillation. The ventricular rate is 39 bpm (AJ; 19/1/99).

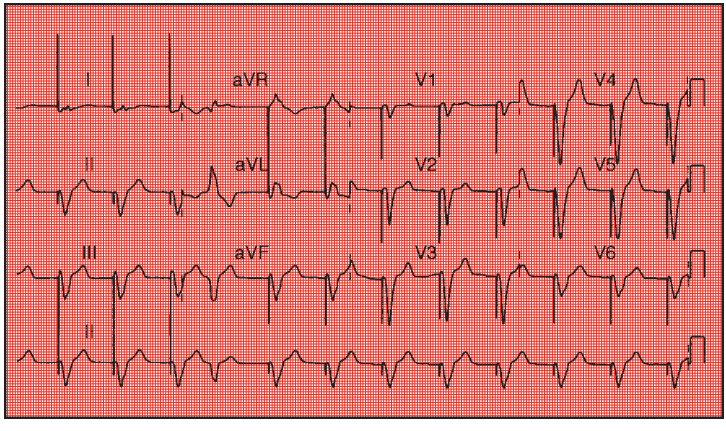


FIG. 8.7. After insertion of a VVI pacemaker, the ventricular rate is 67 bpm. There is no AV sequential pacing in this case. Retrograde P waves are seen in lead I (AJ; 22/1/99).

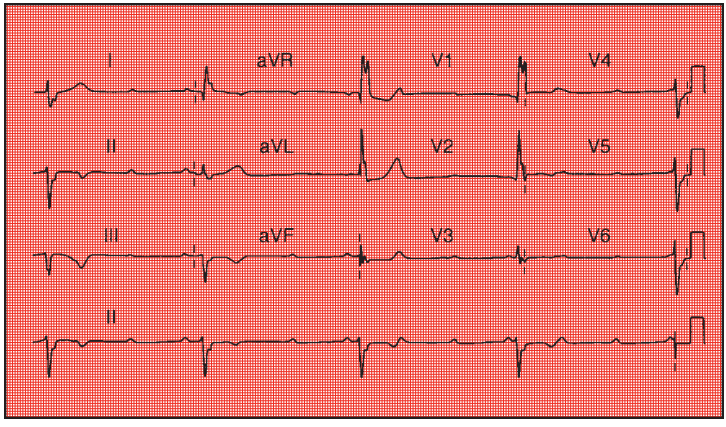


FIG. 8.8. Complete heart block. There is AV dissociation, wide QRS complexes, and a ventricular rate of 25 bpm (GM; 7/3/99).

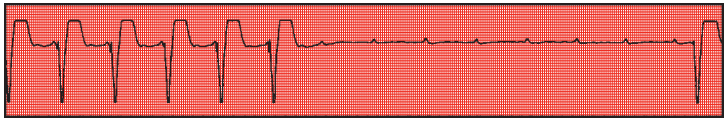


FIG. 8.9. Rhythm strip from the same patient showing good pacing activity to the left, P waves are only seen to the right of the strip. The patient is completely dependent on pacing as demonstrated during pacemaker check (GM; March 1999).

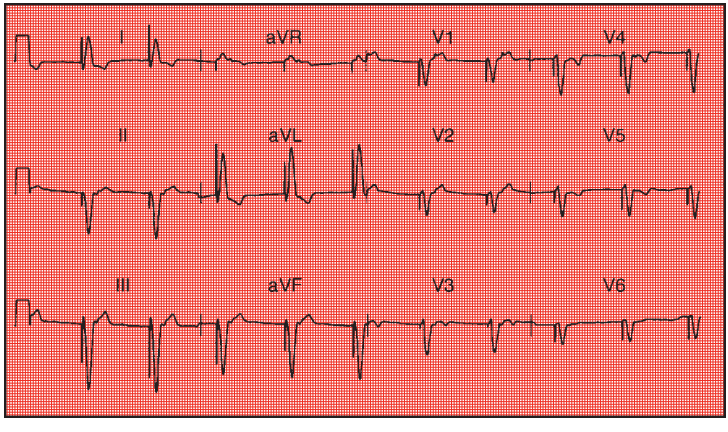


FIG. 8.10. VVI pacing showing retrograde P waves. The patient is well with no symptoms (PD; 16/8/99).

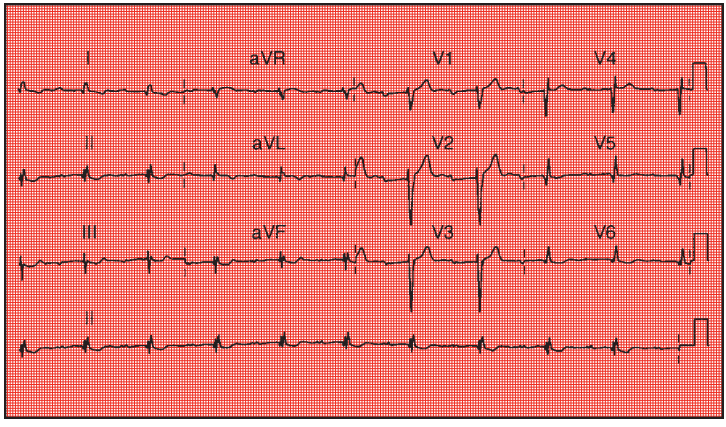


FIG. 8.11. A 72-year-old patient who had undergone bypass surgery years before was diagnosed with first-degree heart block. The PR interval is prolonged (WN; 10/5/99).

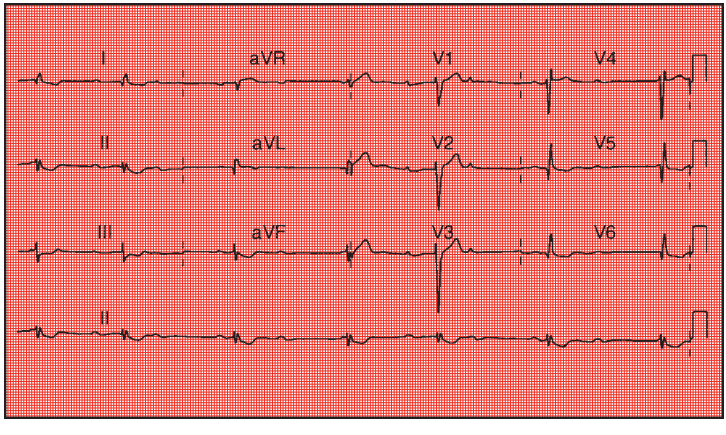


FIG. 8.12. The same patient, several days later, progressed to complete heart block, which is best seen in rhythm strip (lead II) (WN; 28/5/99).

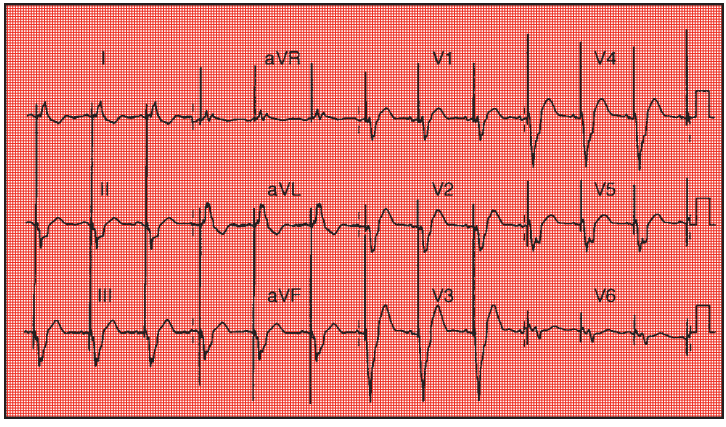


FIG. 8.13. After insertion of a VDD pacemaker there was a good AV sequential response (WN; 12/6/99).

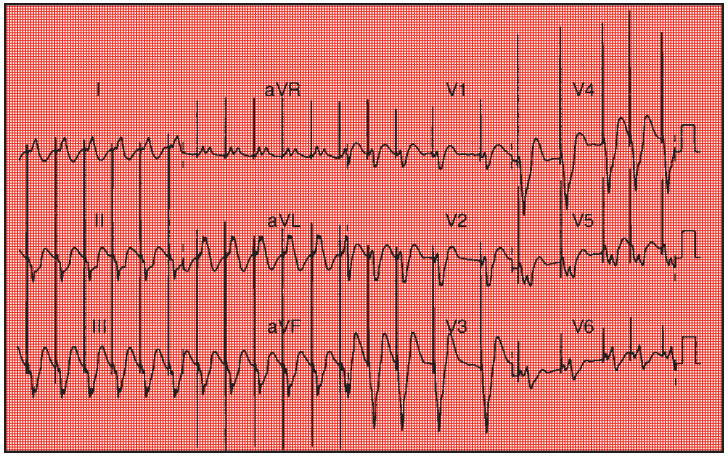


FIG. 8.14. The patient suddenly felt unwell; his blood pressure dropped to 90/47 mmHg with signs of tachycardia. This was attributed to improper setting of the pacemaker, which is sensing inappropriately. The pacemaker was reset and all was well (WN; 12/6/99).

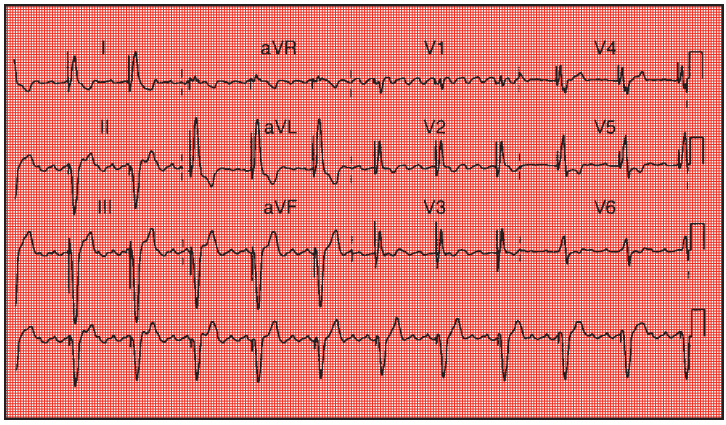


FIG. 8.15. Pacing activity with underlying atrial flutter. The *P* rate is 300 bpm (GB; 16/12/98).

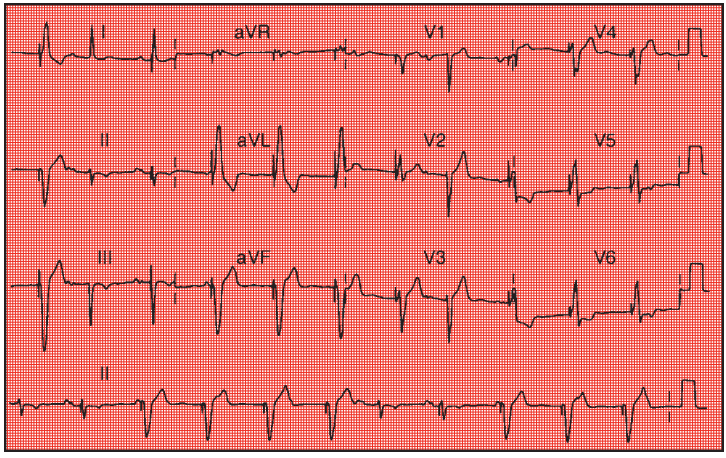


FIG. 8.16. The same patient now has underlying complete heart block. Dissociated P waves are clearly seen in lead V1 (GB; 1999).

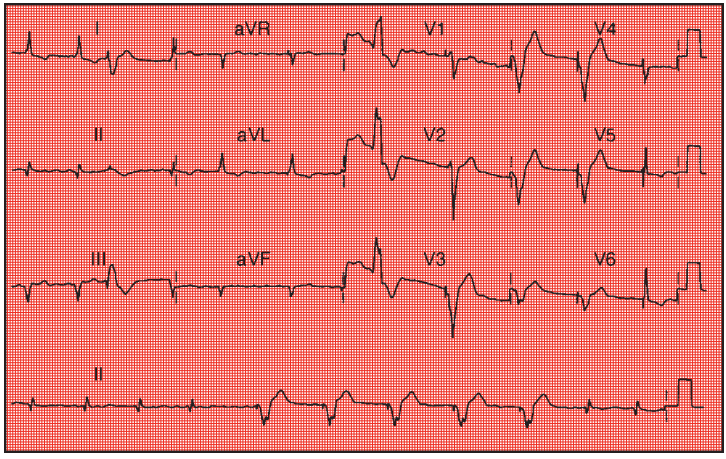


FIG. 8.17. ECG from a patient who had undergone two bypass operations and had poor LV function. A VVI pacemaker was inserted for episodic slow atrial fibrillation. The rhythm strip clearly shows atrial fibrillation and pacing activity (PP; 2/10/98).

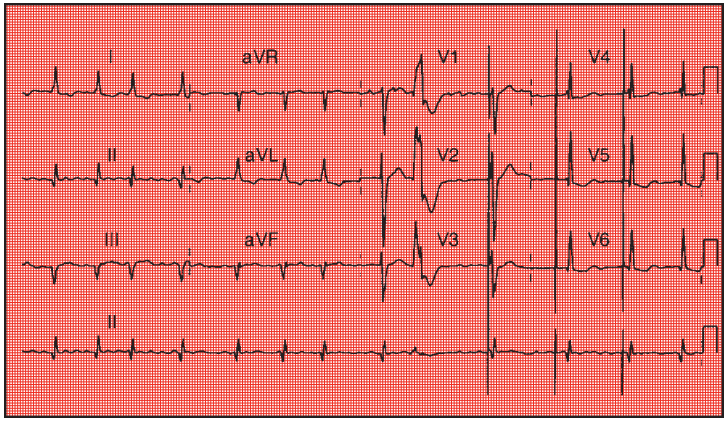


FIG. 8.18. Several weeks later the rhythm strip at the bottom shows that pacing is not capturing well. The middle of the three paced beats shows a prolonged pacing to the QRS interval, which is also seen in leads V4, V5, and V6. This resulted from dislodgement of the pace wire (PP; 23/11/98).

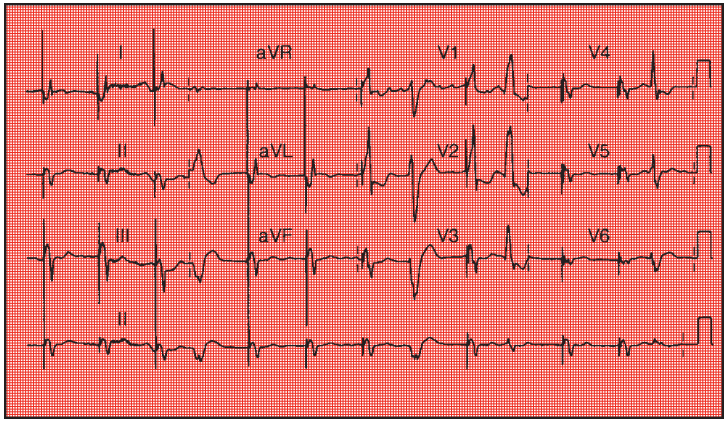


FIG. 8.19. The patient then had a biventricular system (for reduced LV function). One electrode was placed in the right ventricle and the other in the coronary sinus. Two clear pacing signals are seen in leads V4, V5, and V6. Note the short interval between the two pacing signals (PP; 12/5/99).

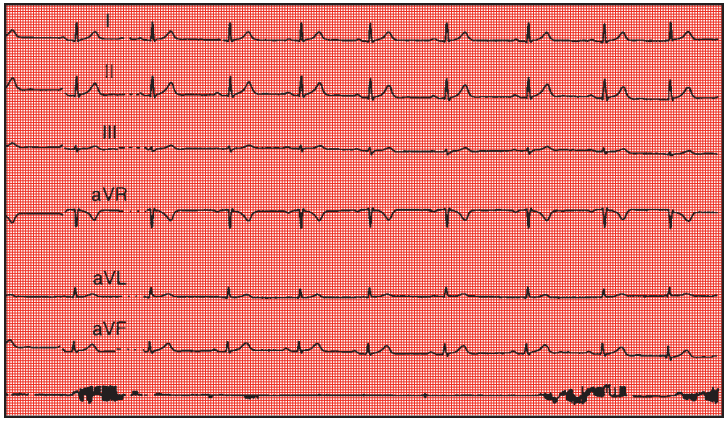


FIG. 8.20. Beginning of a tilt test in a 40-year-old librarian who was suffering from syncopal attacks (KD; September 1999).

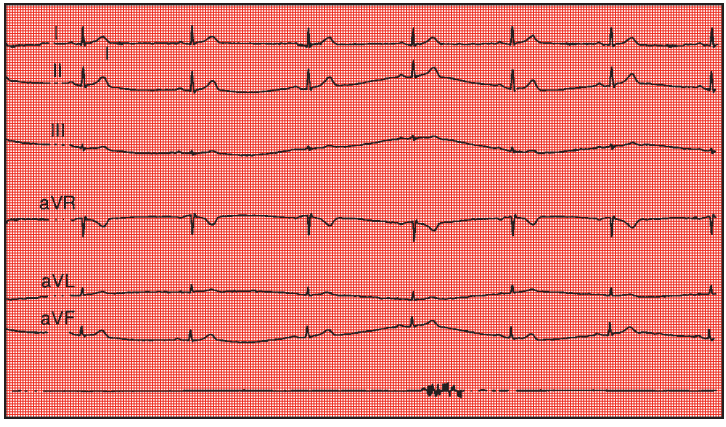


FIG. 8.21. The patient's heart rate dropped down to 20 bpm as he fainted during tilt test (KD; September 1999).

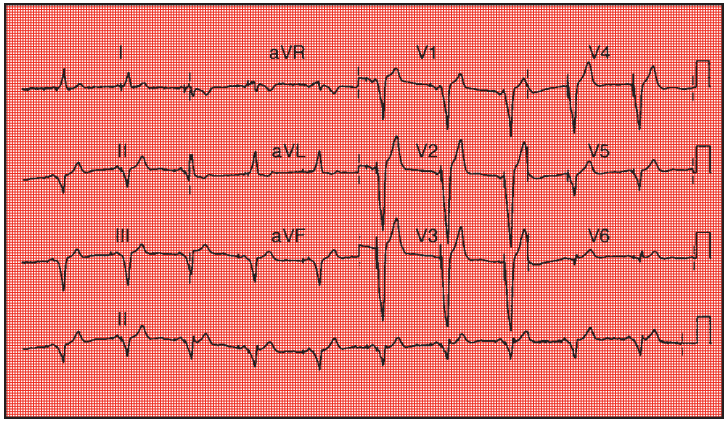


FIG. 8.22. A dual chamber pacemaker was inserted. The two pacing signals are seen in the rhythm strip at the bottom and the precordial leads, stimulating first the atria and then the ventricles in sequence. No further symptoms were reported (KD; 15/9/99).

Follow-Up

Follow-up is undertaken in specialized pacing clinics. Modern generators are interrogated and programmed noninvasively. Battery depletion can now be predicted fairly accurately.

Failure to Pace

This is uncommon nowadays but can occur as a result of any of the following:

- Migration of electrodes (particularly in the first few days after implantation)
- High threshold (exit block), for example, thrombus formation at the tip of the electrode, or fibrotic changes in a damaged right ventricle
- Damaged electrodes (occasional perforation of myocardium)
- Faulty generator (very rare)

COMPLICATIONS OF PACEMAKERS

Complications are rare but can include the following:

- Infection
- Thrombosis of the vein in which the pace wire is inserted
- Perforation of the myocardium
- Tamponade

TEMPORARY PACING

An electrode is inserted into the right ventricle (through the femoral, external jugular, subclavian, or brachial vein) and is connected to a nonimplanted generator.

Indications

These include second- or third-degree heart block particularly after myocardial infarction. Preoperatively, it can be used in the presence of important bradycardia and during angioplasty in selected cases. Permanent and temporary pacing are covered with antibiotic medication. It is customary to give a course for several days after implantation. Antibiotic powder can also be inserted into the subcutaneous pouch at the time of generator implantation.

Occasionally, pacing can be useful to correct a supraventricular or ventricular arrhythmia by a method known as *overpacing*. The technique consists of pacing *temporarily* at a rate above the arrhythmia and then bringing down the pace rate to reestablish sinus conduction.

External Pacemakers

These fairly crude devices can be life-saving. They are unpleasant for the patient, consisting of large plate electrodes that are applied to the thorax in extreme situations, while awaiting proper pacemaker implantation. They are painful to the patient, who will need profound sedation.

General Precautions

DC cardioversion is usually possible in patients with pacemakers. If in doubt, obtain the manufacturer's advice. The electrodes should be kept as far as possible from the pacemaker site. Mobile telephones can interfere with pacing activity and should be tested individually.

IMPLANTABLE CARDIOVERTER/DEFIBRILLATORS

These devices sense the onset of a ventricular tachycardia or fibrillation and respond by sending out an electrical discharge to restore sinus rhythm. Low energy is used for cardioversion and high energy for defibrillation, which can be fairly unpleasant to the patient. The system also acts as a pacemaker for bradycardias.

Initially external electrodes were applied around the heart, necessitating thoracotomy. The present models use transvenous electrodes and the generators are only slightly larger than an ordinary pacemaker.

Clinical trials have recently demonstrated that ICDs are superior to antiarrhythmic medication for VT/VF. Atrial defibrillators are presently being evaluated.

Driving

Patients fitted with permanent pacemakers and ICDs have to report to the national driving authority. Ordinary pacing allows continued driving. ICD regulations are more complex.

CARDIOVERSION

Direct current (DC) cardioversion is the delivery of an electrical shock to the heart to depolarize the myocardium. This allows the sinus node to regain control. The procedure require a short-acting general anesthesia.

Internal Debrillators

Internal defibrillators are used during open heart surgery. The electrodes are placed directly over the myocardium.

External Defibrillators

Two electrodes are applied to the chest, and each electrode is marked sternum or apex. The skin is covered with a gel pad (to prevent burning). The patient is heavily sedated or given a short anesthetic for elective procedures. The energy delivered is measured in Joules. Atrial flutter requires 50 J, whilst atrial fibrillation and VT/VF usually require 200 J, building up to 360 J if necessary. Synchronization is used to avoid discharging on the patient's T wave, which could

induce ventricular arrhythmias (Fig. 4.79). As the shock is delivered, assisting personnel are not to touch the patient.

Risks

Cardioversion can cause occasional profound bradycardia requiring intravenous atropine or even temporary pacing.

Systemic emboli at the time of cardioversion or in weeks following the event are known to occur. It is therefore essential that the patient is given adequate anticoagulation.

In established cases of AF warfarin is the drug of choice; the INR should be between 2.0 and 2.5. In acute cases, a bolus of intravenous heparin (5,000 units half an hour before cardioversion) is indicated.

Cardioversion should only be undertaken after 3 weeks of adequate anticoagulation with warfarin. Anticoagulation has to be continued for 4 weeks after successful cardioversion. TOE should be undertaken in high-risk patients to exclude LA appendage thrombus.

In patients with AF it has been shown that amiodarone given for a few weeks prior to, or atropine at the time of DC conversion improve the success rate. After successful DC conversion, recurrence of the arrhythmia can be prevented by the use of flecainide, propafenone, sotalol, or amiodarone. Quinidine is still in use in the USA.

Emergency Cardioversion

For VT/VF, immediate DC shock is indicated without synchronization. The procedure is usually supplemented by intravenous lignocaine or bretylium tosylate, particularly in resistant ventricular arrhythmias. Cardiopulmonary resuscitation (CPR) may be required.

For uncontrolled supraventricular tachycardia with cardiogenic shock (BP below 80 mmHg systolic), synchronization is applied and intravenous heparin and sedation are indicated.

HISTORICAL NOTES

A Senning (1915–2000) Swedish Professor of Surgery, Zurich, Switzerland.

SI Seldinger (1921–1998) Swedish Radiologist

KEY MESSAGES

- Dual chamber pacing (RA, RV) is the preferred mode of pacing.
- RV pacing (VVI) is used in atrial fibrillation.
- Biventricular pacing (RV, LV) is beneficial in patients with severe heart failure (CRT).

Chapter 9
ECG in Congenital Heart Disease

GENERAL PRINCIPLES

Normal ECG: Transition from Neonate to Adult

Heart Rate

Heart rate varies with age (Table 9.1), status at the time of ECG recording and other physical factors.

P wave

P wave represents atrial depolarization. Mean P wave amplitude in lead II is 1.5 mm (0.15 mV) with a maximum 3.0 mm (0.3 mV). Normal P wave duration is 0.06 ± 0.02 s in children. Maximum P wave duration is 0.10 s in normal children and 0.08 s in infants less than 12 months.

PR interval

It represents the time interval for atrial depolarization and physiological delay in the atrioventricular node. The normal PR interval varies with age and heart rate (Table 9.2). The PR interval is longer in older persons and in persons with a slower heart rate. The lower limit of PR interval is important in determining if pre-excitation exists (Table 9.3).

QRS complex

QRS complex represents the integration of different instantaneous vectors from ventricular depolarization.

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
DOI: 10.1007/978-1-84800-312-5_9,

TABLE 9.1. Range of heart rate in different age groups

Age	Mean heart rate (bpm)	Range (bpm)
Newborn	145	90–180
6 months	145	105–185
1 year	132	105–170
2 years	120	90–150
4 years	108	72–135
6 years	100	65–135
10 years	90	65–130
14 years	85	60–120

Park MK, Guntheroth WG. How to read paediatric ECGs. 4th Ed

Q wave

Q wave represents the initial phase of ventricular depolarization with mean vector directed superiorly and rightward. Normally, Q wave is absent in right precordial leads (V1, V4R) except for rare instances in the newborn infant. Therefore, the amplitude and the duration of the Q wave are important in distinguishing the normal from the abnormal Q wave. Q wave duration is usually 0.01–0.02 s and normally does not exceed 0.03 s.

R and S waves

One of the most characteristic changes of ECG from children to adults is the transitional change of R and S waves due to the change from right ventricular to left ventricular dominance. The horizontal plane is preferred in separating right ventricular from left ventricular force because the ventricles overlie each other to a great extent in the frontal plane (Table 9.4). R/S progression refers to the pattern of change of the QRS complexes in the precordial leads. In children older than 3 years and adults, there is a smooth progression of the QRS complexes in the precordial leads from rS pattern in right precordial leads (V1, V4R) through RS pattern in V2 and V3, to qRs pattern in V4–V6 position (adult R/S progression). In the first month of life, a dominant R in right precordial leads (V1–V2, V4R) resulting in Rs pattern, progresses to a dominant S in left precordial leads (V5–V6) (complete reversal of R/S progression). In children between 1 month and 3 years old, a dominant R is present in V1 and also in V5–V6 (partial reversal). R/S ratio is the ratio of voltage in R wave to that in S wave. R/S ratio in right precordial leads and that in left precordial leads are also age-dependent. The ratio is greater in right precordial leads and smaller in left precordial leads in children, due to right ventricular

TABLE 9.2. Normal PR intervals among different age groups and heart rate

	Heart rate (bpm)							
Age	<60	60–80	80–100	100–120	120–140	140–160	160–180	>180
0–1 month	–	–	0.10 (0.12)	0.10 (0.12)	0.10 (0.11)	0.09 (0.11)	0.10 (0.11)	0.09
1–6 months	–	–	–	–	0.11 (0.14)	0.10 (0.13)	0.10 (0.12)	0.09 (0.11)
6–12 months	–	–	–	–	0.11 (0.14)	0.11 (0.13)	0.10 (0.12)	0.10 (0.11)
1–3 years	–	–	–	–	0.12 (0.14)	0.11 (0.14)	0.10 (0.12)	–
3–8 years	–	0.15 (0.17)	0.14 (0.16)	0.13 (0.16)	0.13 (0.15)	0.12 (0.15)	–	–
8–12 years	0.16 (0.18)	0.15 (0.17)	0.15 (0.16)	0.14 (0.15)	0.14 (0.15)	–	–	–
12–16 years	0.16 (0.19)	0.15 (0.18)	0.15 (0.17)	0.l5 (0.16)	–	–	–	–
Adults	0.17 (0.21)	0.16 (0.21)	0.15 (0.20)	0.15 (0.19)	0.15 (0.18)	–	–	–

PR interval in second: mean (upper limit of normal) Park MK, Guntheroth WG. How to read paediatric ECGs. 4th Ed

TABLE 9.3. Lower limit of normal PR interval by different age groups

Age	LLN (s)
<12 months	0.07
1–3 years	0.08
3–16 years	0.09
Adults	0.10

dominance. In contrast, this is small in right precordial leads, but large in left precordial leads, in adults.

QRS axis

QRS axis represents the mean vector of ventricular depolarization. In general, there is a transition of mean frontal vector from infant to adult (Table 9.5). Abnormal axis deviation should therefore take the age-related change into consideration.

QRS duration

With increase in the mass of ventricular muscle with age, the QRS duration tends to increase with age (Table 9.6).

ST segment

Normal ST segment is horizontal and isoelectric. There is no significant age-dependent change.

T wave

T wave amplitude

T wave represents ventricular repolarization and its amplitude is best appreciated in the left precordial leads. There is also age-related limit to the amplitude (Table 9.7).

T wave vector

T axis represents the mean vector of the repolarization process. This could be appreciated at the frontal plane (Table 9.8) as well as the horizontal plane (Table 9.9).

QT interval

The QT interval represents the time required for both ventricular depolarization and ventricular repolarization. The measurement is important as abnormal QT interval is associated with serious ventricular arrhythmias and sudden death. As the QT interval varies

TABLE 9.4. Normal amplitudes of R and S waves in different age groups

	R					S				
Age	V4R	V1	V2	V5	V6	V4R	V1	V2	V5	V6
0–1 month	6 (12)	13 (24)	18 (30)	12 (23)	5 (15)	4 (9)	7 (18)	18 (33)	9 (17)	3 (10)
1–6 months	5 (10)	10 (19)	20 (31)	20 (33)	13 (22)	4 (12)	5 (15)	15 (26)	7 (16)	3 (9)
6–12 months	4 (8)	10 (20)	22 (32)	20 (31)	13 (23)	5 (12)	7 (18)	16 (29)	6 (15)	2 (7)
1–3 years	4 (8)	9 (18)	19 (28)	20 (32)	13 (23)	5 (12)	8 (21)	18 (30)	5 (12)	2 (7)
3–8 years	3 (8)	8 (6)	15 (25)	23 (38)	15 (26)	5 (14)	11 (23)	20 (33)	4 (10)	2 (5)
8–12 years	3 (7)	5 (12)	12 (20)	26 (39)	17 (26)	6 (20)	12 (25)	21 (36)	3 (8)	1 (4)
12–16 years	3 (7)	4 (10)	10 (19)	21 (35)	14 (23)	6 (20)	11 (22)	18 (33)	3 (8)	1 (4)
Adult	–	3 (14)	6 (21)	12 (33)	10 (21)	–	10 (23)	14 (36)		1 (3)

Voltages measured in millimeters (1 mV = 10 mm): mean (upper limit of normal)Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60

TABLE 9.5. Normal range of QRS vector in different age groups

	Mean QRS vector (°)	Range (°)
Newborn	+134	+64 to +161
1–3 months	+74	+31 to +125
3–12 months	+60	+5 to +110
1–3 years	+60	+1 to +101
3–15 years	+60	+1 to +120
Adults	+50	−30 to +105

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60
Garson AJ. Diagnostic electrocardiography. In: Anderson RH, Baker EJ, MaCartney FJ, Rigby ML, Shinebourne EA, Tynan M l (eds) Paediatric cardiology. Churchill Livingstone, London, pp. 295–378

TABLE 9.6. Normal age-related range of QRS duration

	Mean QRS duration (s)	Range (s)
0–1 month	0.050	0.031–0.07
1–6 months	0.055	0.032–0.076
6–12 months	0.055	0.034–0.076
1–3 years	0.056	0.038–0.076
3–8 years	0.060	0.041–0.079
8–12 years	0.062	0.041–0.085
12–16 years	0.065	0.044–0.087
Adults	0.08	0.10[a]

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60
Garson AJ. Diagnostic electrocardiography. In: Anderson RH, Baker EJ, MaCartney FJ, Rigby ML, Shinebourne EA, Tynan M l (eds) Paediatric cardiology. Churchill Livingstone, London, pp. 295–378
[a]Upper limit of normal

TABLE 9.7. T Wave amplitude in different age groups

Age	V5 (mm)	V6 (mm)
<1 year	7	5
>1 year	11	7
After adolescence		

TABLE 9.8. T wave vector at frontal plane in different age groups

	Mean T wave vector at frontal plane (°)	Range (°)
<1 month	+25	–40 to +100
>1 month	+45	0 to +90

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60
Garson AJ. Diagnostic electrocardiography. In: Anderson RH, Baker EJ, MaCartney FJ, Rigby ML, Shinebourne EA, Tynan M l (eds) Paediatric cardiology. Churchill Livingstone, London, pp. 295–378

TABLE 9.9. T axis at horizontal plane in different age groups

Age	Direction of T vector	V1	V4–V6
Newborn	Anterior and leftward	Upright	Upright
First week of life	Posterior and leftward	Negative	Upright
First 4–5 years	Posterior and leftward	Negative	Upright
5–10 years	Either anterior or posterior	Negative or upright	Upright
>10 years	Progressive anterior	Upright	Upright

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60

with heart rate (Table 9.10), it is interpreted in relation to the heart rate, with correction using Bazett's formula (Table 9.11).

JT interval

In patients with a congenital heart problem, lengthening of QRS duration secondary to congenital heart disease or previous surgery may affect QT interval. JT interval, measuring from the J point (the junction of S wave and the ST segment) to the end of the T wave, may be a more sensitive predictor of repolarization abnormalities. This interval is corrected with Bazett's formula. Normal JTc is 0.32 ± 0.02 s with a normal upper limit of 0.34 s in children and adolescents (Berul CI, Sweeten TL, Dubin AM, et al. Am J Cardiol 1994;74:1254–1257).

TABLE 9.10. Normal values of QT interval in different heart rate

Heart rate (bpm)	Mean QT interval (ms)	Upper limit of normal (ms)
40	450	490
50	410	450
60	390	420
70	360	380
80	359	395
90	345	380
95	328	370
100	325	360
105	318	365
110	306	355
115	300	365
120	293	350
125	288	335
130	279	330
135	273	325
140	272	325
145	264	305
150	255	290

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60

TABLE 9.11. Normal range of corrected QT interval by age-groups

Age	QTc interval (ms)	Upper limit of normal (ms)
Newborn	–	470
Early infancy	–	450
Children	400	440

Park MK, Guntherorth WG (eds) How to read paediatric ECGs. Mosby, pp. 45–60

Cardiac Morphology and Chamber Localization

Cardiac morphology is fundamental in studying congenital heart disease. With advances in different imaging modalities, various cardiac morphologies could be elucidated. However, ECG could provide important clues to different morphologies.

Atrial location

The sinoatrial node, where atrial depolarization is initiated, is located in the morphological right atrium. The P vector is directed toward the left lower quadrant (0 to +90°) in patients with a normal atrial position. If the morphological right atrium is on the left side, the P vector is directed toward the right lower quadrant (+90 to +180°). In atrial isomerism, both the atria are of the same morphology, i.e., both are morphological right or left atria. The atrial depolarization may then be abnormal and can vary significantly.

Ventricular position

In the normal heart, where the left ventricle is on the left and posterior to the right ventricle, septal depolarization results in the Q wave being present in leads I, V5 and V6. When the morphological left ventricle is to the right of the morphological right ventricle, the Q wave is present in right-sided precordial leads (V1, V4R) but is absent in leads I, V5 and V6.

Heart in the right side of the chest (Dextrocardia)

In general, the QRS amplitude shows progressive decrease toward V6 when the heart is in the right side of the chest. This encompasses several possible cardiac morphologies.

Mirror-image-dextrocardia

Mirror-image-dextrocardia refers to the complete reversal of the right and left relationship, thus the right atrium and right ventricle are on the left side and vice versa. As a result, the mean P wave axis directs into the right lower quadrant (+90 to +180°) with a negative P wave in leads I and aVL. Septal depolarization occurs from right to left with Q waves being present in right-side precordial leads and absent in left-sided precordial leads. The left-sided precordial leads may show a small r wave producing rsR¢ pattern.

Dextroposition

Dextroposition refers to the situation where the cardiac silhouette has shifted toward the right side of the chest with normal right-to-left relationship. This can occur secondary to hypoplasia of the right lung. The P axis is in normal left lower quadrant (0 to +90°). The Q waves are present in leads I, V5 and V6 as in the normal situation.

Discordant atrioventricular connection and dextrocardia

Discordant atrioventricular connection and dextrocardia is characterized by the connection of the right atrium to the left ventricle

and the left atrium to the right ventricle. Usually there is also discordant ventriculoarterial connection (transposition of great arteries) leading to congenitally corrected transposition. If the right atrium is still on the right side, the P axis is directed normally (0 to +90°) but the septal depolarization is in the reverse direction, producing Q waves in the right precordial leads. If the right atrium is on the left side, then the P axis is directed toward the right lower quadrant (+90 to +180°). As septal depolarization occurs from left to right, the Q waves will follow normal distribution. This is further discussed in the section on congenitally corrected transposition of the great arteries.

Complex cardiac anomalies

Complex cardiac anomalies such as atrial isomerism or "univentricular" hearts may be associated with dextrocardia.

Single ventricle

In "univentricular" hearts, the septum is usually malformed (absent or rudimentary), and the ventricular septal depolarization, thus, is affected, producing abnormal Q waves and abnormal QRS morphologies.

Abnormal Q wave

The Q wave may be absent in all precordial leads (60% of cases) such that the initial QRS vector directs anteriorly and slightly to the left.

The Q waves are present in all the precordial leads (15% of cases). The initial QRS vector directs posteriorly and slightly to the right.

Q waves are present in right precordial leads but absent in left precordial leads (25% of cases).

QRS morphology may be similar throughout all the precordial leads, resulting in abnormal RS progression.

SPECIFIC CONGENITAL HEART DISEASE

Characteristic ECG findings – unrepaired ventricular septal defect (VSD) (Fig. 9.1)

Small VSD

- Usually normal
- RSR¢ pattern occasionally present in V1

Moderate VSD

- Broad notched left atrial P waves in leads I and II and broad deep P terminal force in V1 (left atrial hypertrophy)

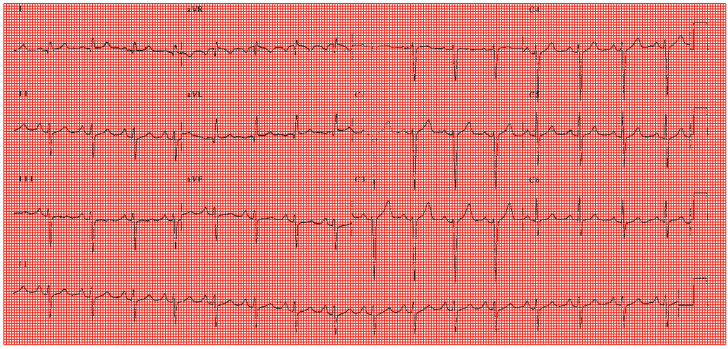


FIG. 9.1. Ventricular septal defect.

- QRS axis usually normal (left axis deviation found in 5% of restrictive and moderate VSD and in 40% of multiple VSDs)
- Tall R waves and tall peaked T waves in leads I, II and AVF and prominent Q waves, tall R waves and tall peaked T waves in V5–V6 (left ventricular volume overload)

Large nonrestrictive VSD

- Right atrial or combined atrial P wave abnormalities in leads II and V1–V2
- QRS axis shifts moderately to the right
- Combined right and left ventricular hypertrophy: increased R wave in V1 and deep Q waves, tall R waves and tall peaked T waves in V5–V6, and large equiphasic RS complex in mid-precordial leads

Eisenmenger VSD

- P waves often normal in younger and moderately peaked in older patients
- Moderate right axis deviation
- Tall monophasic R wave occasionally notched on its upstroke and followed by a small s wave (right ventricular hypertrophy)

Characteristic ECG findings – repaired VSD
Surgical repair

- RBBB found in 29–65%, more than 25 years after surgical repair
- Complete heart block in 0.7–3.5%

Transcatheter closure

- No long-term data available at present, only up to 1-year follow-up study
- Complete heart block found in 1.1–1.9%

Characteristic ECG findings
Secundum atrial septal defect (ASD) (Fig. 9.2a)

- Sinus rhythm is usual
- Right QRS axis deviation (+95 to +170°)
- Incomplete right bundle branch block pattern: rsR¢ or RSR¢ pattern in V1 (consistent with right ventricular overload)
- Tall and peaked P waves may be present (right atrial enlargement)

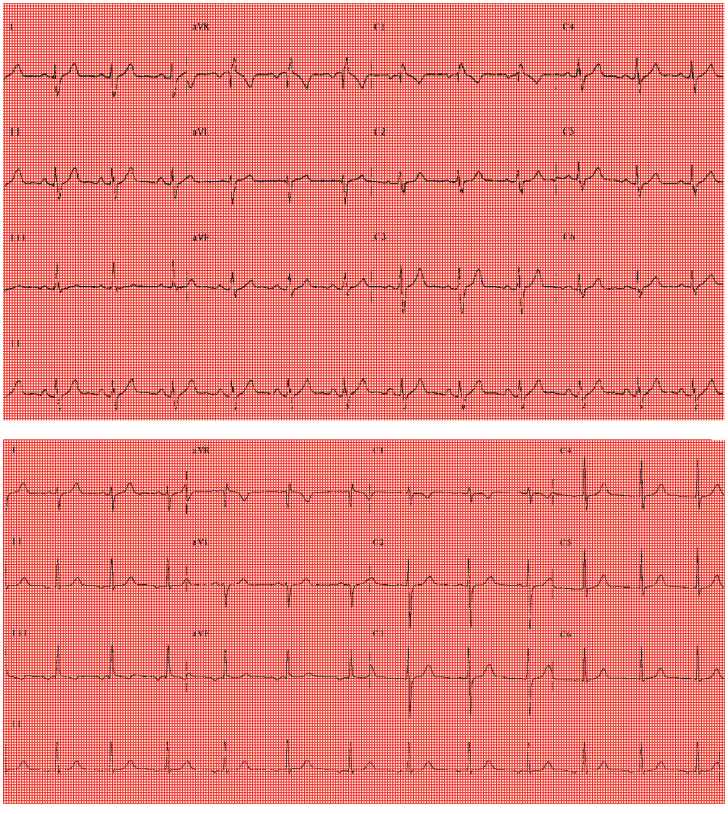


FIG. 9.2. Atrial septal defect. Top ECG: Secundum Atrial Septal Defect and the Bottom: Sinus venosus Atrial Septal Defect

Primum ASD

- Represented as part of the Atrioventricular Septal Defect (AVSD), of which the ECG findings are discussed in a separate chapter.

Sinus Venosus ASD (Fig. 9.2b)

- Right axis deviation and incomplete RBBB pattern present in 8% of patients
- Ectopic atrial rhythm, junctional rhythm or wandering atrial pacemaker reflecting sinus node dysfunction (as the defect occupies the vicinity of the area of the sinus node)

Additional ECG features
Supraventricular arrhythmias

- May occur especially in older subjects
- Includes supraventricular tachycardia (SVT), atrial flutter and atrial fibrillation

Pulmonary hypertension

- The ECG of this will be discussed in a separate chapter

Characteristic ECG findings
Atriventricular septal defect (AVSD) (Fig. 9.3)

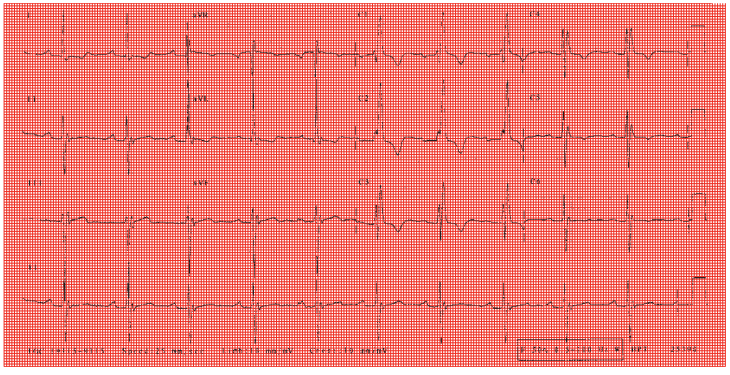


FIG. 9.3. Atrioventricular septal defect (AVSD): Sinus rhythm; heart rate 60 bpm; PR interval 260 ms i.e., first degree heart block; superior axis (left); right ventricular hypertrophy with RsRc pattern in precordial leads.

- Superior QRS axis (left axis or extreme right axis) with a counterclockwise depolarization pattern (95%) is the hallmark and is due to inferior displacement of the conduction system
- Prolonged PR interval may progress to complete heart block
- P wave amplitude or width may increase (right or left atrial enlargement)
- Right ventricular hypertrophy represented by rsR¢ pattern in right precordial leads
- Left or bi-ventricular hypertrophy may be present in case of severe left atrioventricualr valve regurgitation

Characteristic ECG findings

Left ventricular outflow tract obstruction (LVOTO) (Fig. 9.4)

Aortic valve stenosis (including bicuspid aortic valve), sub- and supra-valvar aortic stenos are discussed together as they share similar ECG features.

Mild stenosis

- ECG is usually normal.

Moderate to severe stenosis

- Left ventricular hypertrophy
- Left ventricular strain pattern may be present (ST depression and T inversion in left precordial leads)

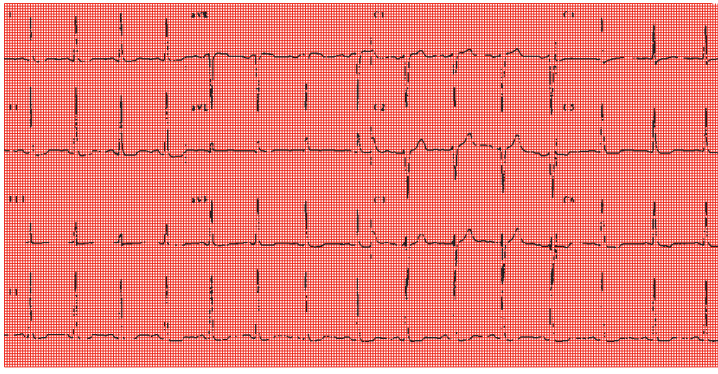


FIG. 9.4. Left ventricular outflow tract obstruction (LVOTO): Sinus rhythm; QRS axis: +45°; Left ventricular hypertrophy pattern; Left ventricular strain pattern: ST depression and flat T wave in left precordial leads.

Ventricular arrhythmias may be present

- Premature multiform ventricular contraction
- Ventricular tachycardia

Notes: Significant aortic regurgitation may develop concomitantly in these conditions, resulting in further ECG changes.

Characteristic ECG findings
Coarctation of the aorta (CoA) (Fig. 9.5)
The ECG abnormalities could be due to

- The pressure load imposed by the coarctation to the left ventricle
- The development of systemic artieral hypertension
- The frequent comorbidity of a stenotic or regurgitating bicuspid aortic valve

Mild coarctation

- ECG is usually normal.

Moderate or severe coarctation

- Left atrial enlargement and left ventricular hypertrophy, both of varying degrees, depend on the severity of the disease.

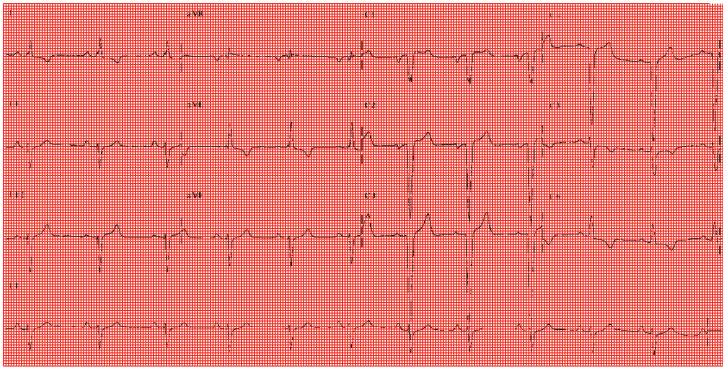


FIG. 9.5. Coarctation of aorta (CoA): Sinus rhythm; Left axis deviation (QRS axis: –60°); Left ventricular hypertrophy; T wave inversion at V5 and V6.

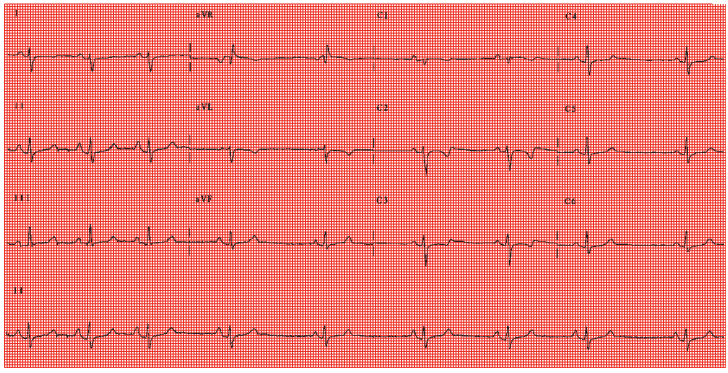


FIG. 9.6. Pulmonary stenosis (PS): Sinus rhythm; right axis deviation; tall P waves in lead II (right atrial enlargement); T wave inversion in V1 and V2 (right ventricular strain pattern).

Characteristic ECG findings
Pulmonary stenosis. (Fig. 9.6)

Mild stenosis

- ECG may be normal.

Moderate to severe stenosis

- Right axis deviation (mean QRS axis may range from +110 to +160°)
- Right atrial enlargement
- Right ventricular hypertrophy (dominant R wave is present in lead VR and right precordial leads; its amplitude may reflect the severity of the stenosis)
- Right bundle branch block pattern may be present

Repaired pulmonary stenosis

- ECG abnormalities can regress after the stenosis is relieved

Pulmonary regurgitation

- May develop with time after pulmonary valvotomy and head to QKS prolongation.

Characteristic ECG findings
Tetralogy of Fallot (TOF) (Repaired) (Fig. 9.7)
Repaired TOF

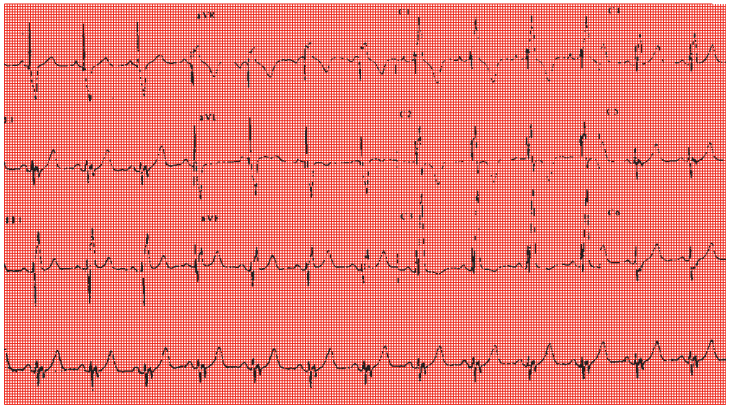


FIG. 9.7. Tetralogy of Fallot (TOF) (repaired): Sinus rhythm; right axis deviation; PR interval: 140 ms; QRS duration: 160 ms; Right bundle branch block pattern; Right ventricular hypertrophy.

- Sinus rhythm is usual
- Right axis deviation
- Right bundle branch block pattern with marked and progressive QRS width (reflecting RV dilatation due to significant pulmonary regurgitation)
- Right ventricular hypertrophy (reflecting RV overload caused by pulmonary regurgitation or residual pulmonary stenosis)
- Prolonged QRS interval is common: QRS duration >180 ms may represent a risk factor for ventricular arrhythmias and sudden cardiac death in these patients
- Supraventricular arrhythmias are common

Unrepaired TOF

- The ECG features may be similar to that of pulmonary stenosis.

Characteristic ECG findings
Pulmonary atresia with intact ventricular septum (PAIVS) (Fig. 9.8)

- QRS axis: normal or leftward
- Often right atrial enlargement (peaked P waves in limb leads and in right precordial leads).
- Decreased right ventricular voltage (reflecting a small right ventricle)
- Left ventricular hypertrophy common
- Atrial and ventricular arrhythmias may occur

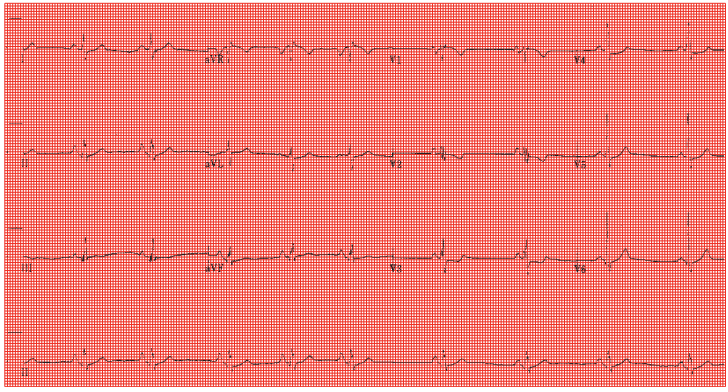


FIG. 9.8. Pulmonary atresia with intact ventricular septum (PAIVS): Sinus rhythm; Ventricular rate: 58 bpm; Normal QRS axis; PR interval: 146 ms; Right atrial enlargement (2.5 mm in II and 1.5 mm in V1); decreased right ventricular voltage

Characteristic ECG findings
Ebstein's anomaly of tricuspid valve (Fig. 9.9)

- Usually in sinus rhythm
- Prolonged PR interval (25–33%)
- Right atrial enlargement (Himalayan P wave) (25–75%)
- Right axis deviation (+90 to +150°)
- Right bundle branch block pattern (75–80%)
- Low-voltage QRS complexes in right precordial leads (small amplitude in V1–V3)
- Wolff–Parkinson–White accessory atrio-ventricular connection (4–30%)
- Arrhythmias are common
  - Paroxysmal SVT
  - Paroxysmal atrial fibrillation or flutter.
  - Frequent ventricular premature contraction.
  - Non-sustained ventricular tachycardia.
  - Atrioventricular block

Characteristic ECG findings
After Mustard and Senning operations (Fig. 9.10)

- Right axis deviation
- Right ventricular hypertrophy

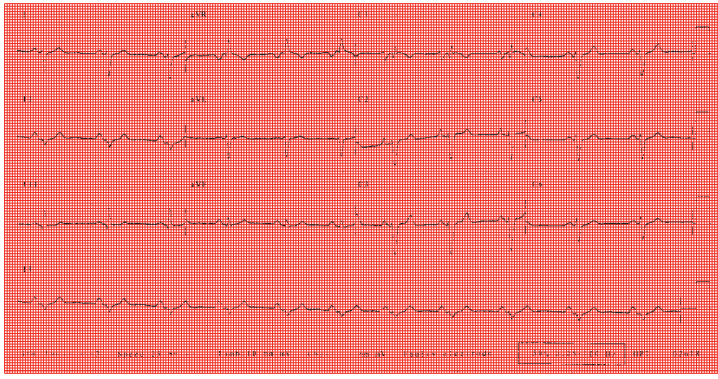


FIG.9.9. Ebstein's anomaly of tricuspid valve: Sinus rhythm; Heart rate: 70 bpm; Prominent peak P wave (right atrial enlargement); PR interval 160 ms; right axis deviation (frontal QRS axis: +168°); Low amplitude of QRS complexes V1; Right bundle branch block pattern

- Tachyarrhythmias (atrial reentry tachycardia)
- Bradyarrhythmias (sinus node dysfunction with junctional escape rhythm)

After Rastelli operation

- Right axis deviation and right ventricular hypertrophy
- Surgical right bundle branch pattern
- Complete heart block may occur

After arterial switch operation

- ECG may be normal
- Right ventricular hypertrophy if right ventricular outflow obstruction is present
- Signs of ischemia if coronary artery occlusion is present

Characteristic ECG findings

Congenitally Corrected Transposition of the Great Arteries (Fig. 9.11a)

- P wave morphology normal if usual atrial arrangement
- Left axis deviation
- Reversal of normal precordial Q waves (75% of cases): presence of Q waves in right precordial leads and absence of Q waves in left precordial leads
- Left-superior septal activation: prominent Q wave in leads III and aVF (50% of cases), QS complexes in leads III and aVF, absence of Q wave in leads I and aVL

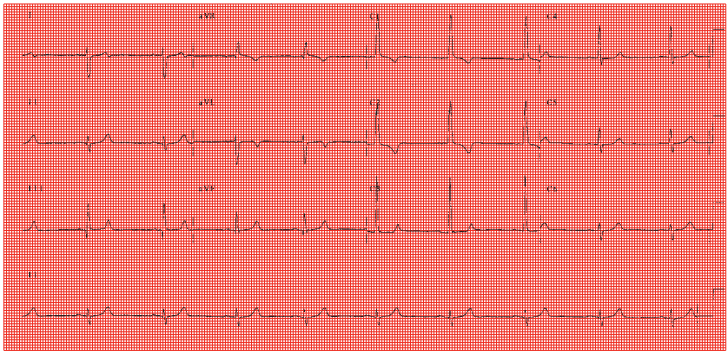


FIG. 9.10. Transposition of great arteries (TGA) (Post-Mustard repair): Sinus bradycardia; heart rate: 56 bpm; right axis deviation; PR interval: 156 msec; right ventricular hypertrophy; inverted T waves in leads V1 and V2.

Characteristic ECG findings

Variations in appearance may be affected by position of heart, atrial arrangement and hemodynamics

Mirror-imaged atrial arrangement

- Normal distribution of Q waves

Dextrocardia with usual atrial arrangement (Fig. 9.11b)

- Right axis deviation
- RS waves in precoridal leads

Morphological LV overload (Fig. 9.11c)

- Right axis deviation
- QR complexes over right praecordium
- Deep S waves over left praecordium

Characteristic ECG findings

Biventricular hypertrophy (Fig. 9.11d)

- Q waves may be absent in all precordial leads
- Large biphasic RS complexes in mid-precordial leads

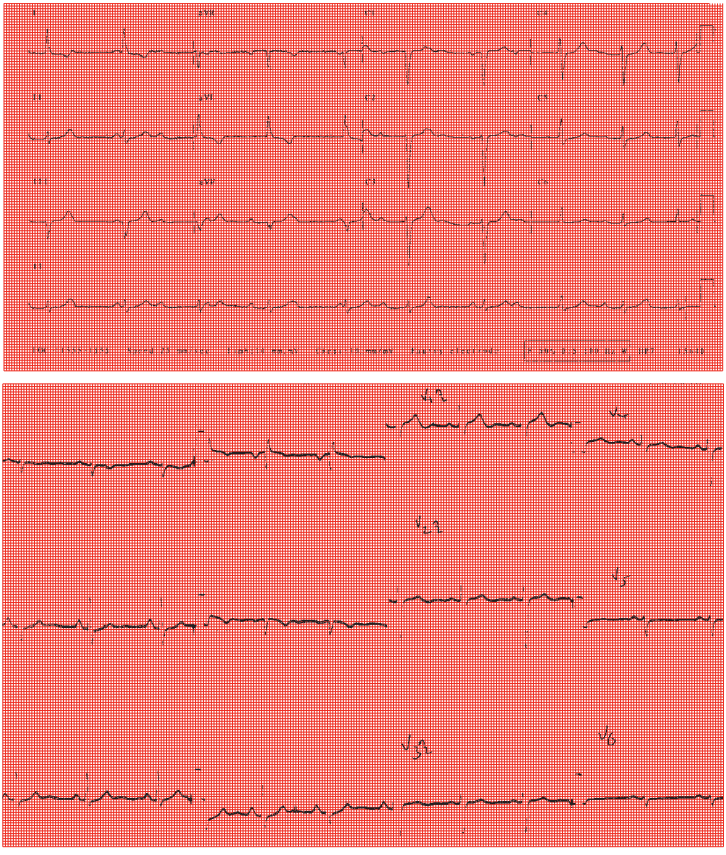
Complete heart block (Fig. 9.11e)

- May develop in these patients (reported incidence of 2% per year)

Characteristic ECG findings
Univentriccular physiology and Fontan circulation (*Tricuspid atresia with dominant left ventricle* (Fig. 9.12a, b)

- Left axis deviation
- Tall and broad P wave
- Small r waves and deep S waves over right precordial leads and tall r waves over left precordial leads

Dominant right ventricle
- Superior frontal QRS axis
- Right ventricular hypertrophy



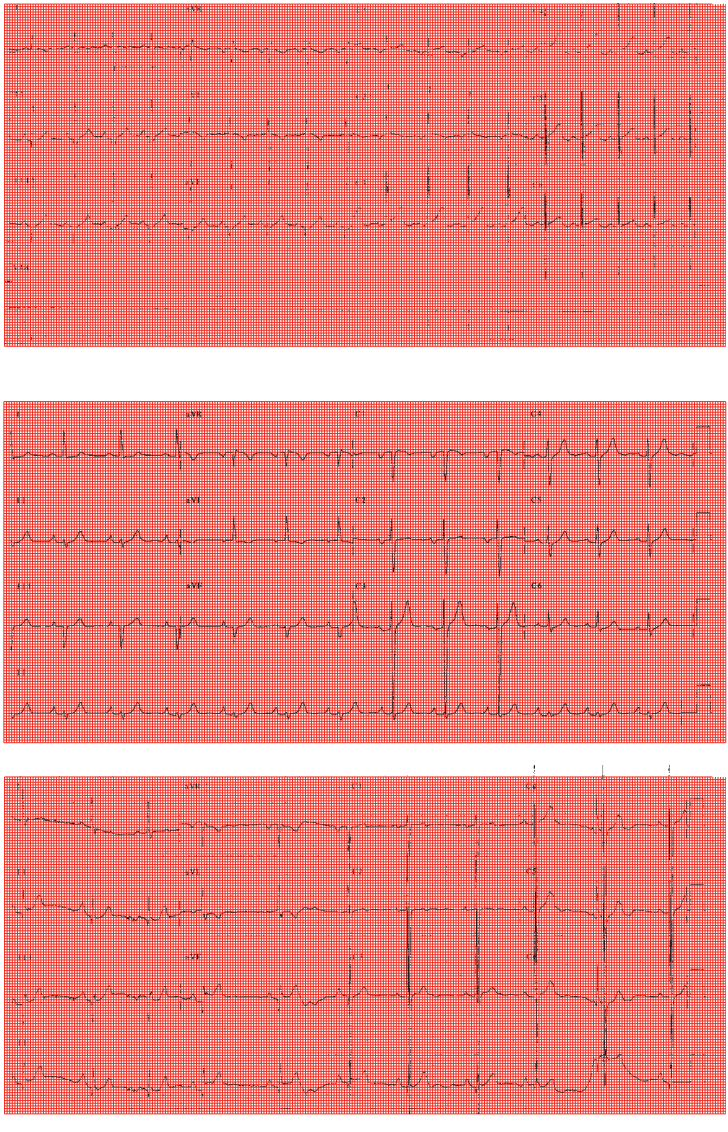


FIG. 9.11. **a** Congenitally Corrected Transposition of the Great Arteries (with levocardia, cardiac apex pointing to the left; usual heart position); Sinus rhythm; heart rate : 75 bpm; left axis deviation; PR interval 180ms; presence of Q wave in V1, absence of Q wave in V6; QS complexes in lead III and aVF. **b** Congenitally Corrected Transposition of the Great Arteries

Right atrial isomerism

- Often have two separate sinus nodes with a P-wave axis that fluctuates with the prevailing pacemaker.

Left atrial isomerism

- Most do not have a sinus node (slow atrial or junctional escape rhythm instead).

Characteristic ECG findings
After Fontan procedure

- Intra-atrial re-entry tachycardia or atypical atrial flutter in 57% of patients
- Atrial fibrillation common (Fig. 9.12b)
- Sinus node dysfunction occurs in 13–16% of patients with Fontan operation

Characteristic ECG findings
Eisenmenger syndrome (Fig. 9.13)

- ECG may be that of pulmonary hypertension
- Right ventricular hypertrophy: tall R wave and inverted T wave in V1
- Right atrial enlargement
- Right axis deviation is common

with dextrocardia (cardiac apex pointing to the right): Sinus rhythm (normal P vector indicating usual atrial arrangement); right axis deviation (QRS axis 110 degrees); RS waves in precordial leads small QRS amplitude in left precordial leads. **c** Congenitally Corrected Transposition of the Great Arteries (Left Ventricular Pressure Overload i.e. Pulmonary Stenosis): Sinus rhythm; right axis deviation (QRS axis 110 degrees); prominent Q wave in III and aVF; absence of Q wave in left precordial. **d** Congenitally Corrected Transposition of the Great Arteries (Biventricular Hypertrophy): Sinus rhythm; normal QRS axis (21 degrees); prominent Q wave in lead III and aVF; absence of Q wave in all precordial leads; large biphasic RS complexes in mid-precordial leads. **e** Congenitally Corrected Transposition of the Great Arteries (Complete Heart Block): Complete heart block; left superior axis; prominent Q wave in V1; absence of Q wave in V6; QS complexes in lead III and aVF.

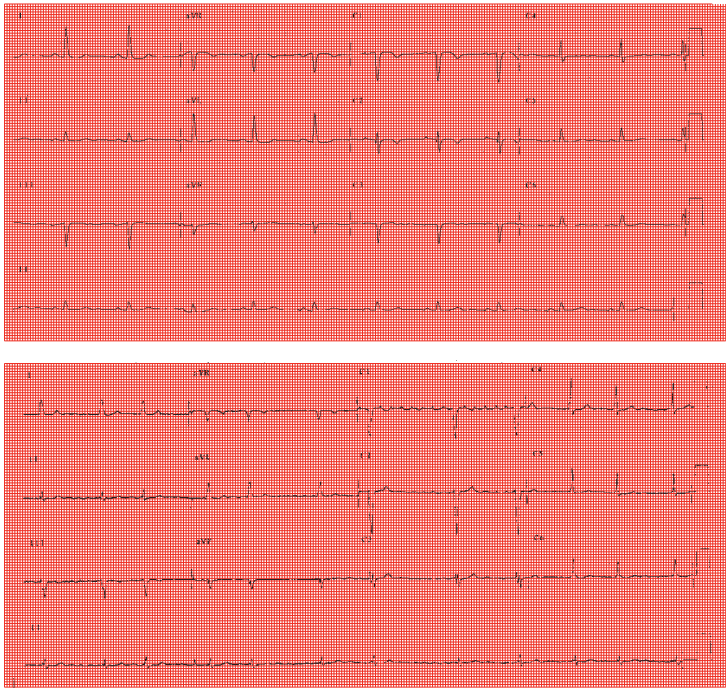


FIG. 9.12. **a** Univentricular physiology and Fontan circulation (Tricuspid atresia): Sinus rhythm; heart rate: 65 bpm; left axis deviation; PR interval: 187 ms; small R waves and deep S waves in V1 and V3; dominant R waves and no S waves in V5 and V6. **b** Atrial fibrillation; heart rate: 72 bpm; left axis deviation; small R waves and deep S waves in V1 and V2; dominant R waves and no S waves in V5 and V6.

ARRHYTHMIA MANAGEMENT

Arrhythmia management for patients with congenital heart disease has to be seen as part of their broader hemodynamic assessment and treatment. Such management includes hemodynamic intervention(s), empiric drug therapy, DC cardioversion, permanent pacing and/or AICDs. Please refer to specific textbooks or centers focused on ACHD for further advice.

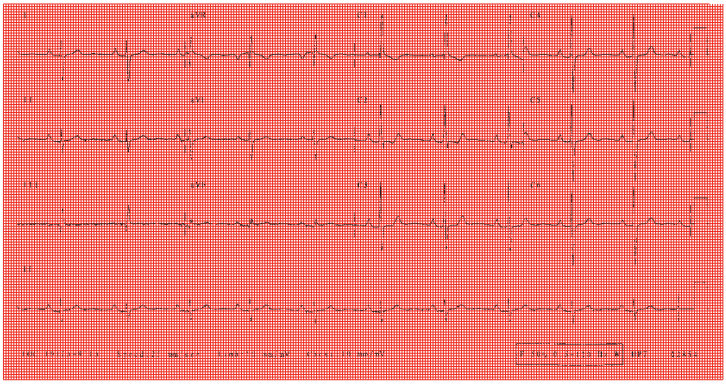


FIG. 9.13. Eisenmenger syndrome: Sinus rhythm; right axis deviation; right ventricular hypertrophy (tall R wave in V1 and S wave in V6); inverted T wave in V1

Chapter 10
Intracardiac Electrophysiology

Matthew Wright and Nicholas S. Peters

CARDIAC ELECTROPHYSIOLOGY

Cardiac electrophysiology is a subspecialty of cardiology, concerned with the diagnosis and treatment of cardiac rhythm disturbances. This includes the invasive treatment of tachyarrhythmias with catheter ablation and implantable cardiac defibrillators, the use of pacemakers for bradyarrhythmias and the use of biventricular pacemakers for cardiac resynchronization in heart failure. An understanding of the practice of cardiac electrophysiology is essential to all medical practioners to ensure that all patients receive the best available care.

CATHETER ABLATION

A number of tachyarrhythmias are best treated by catheter ablation as opposed to pharmacological treatment. The common arrhythmias of atrial flutter, atrio-ventricular nodal re-entry tachycardia (AVNRT) and atrio-ventricular re-entry tachycardia (AVRT) are successfully treated by catheter ablation, with a cure rate of greater than 95%, and a very low complication rate of less than 1%. Atrial fibrillation and ventricular tachycardia are also successfully treated by catheter ablation; however cure rates are not as high. Due to the expanding treatments available for patients with arrhythmias, it is essential that they are referred to specialist electrophysiologists to manage their condition, even though not all patients will be suitable for catheter ablation.

THE ELECTROPHYSIOLOGICAL STUDY

Patients undergoing an electrophysiological study have a number of electrode catheters placed in their heart for diagnosis of the arrhythmia mechanism and to guide ablation. These catheters are able to record electrical activation and also to deliver pacing impulses to the heart. The spread of electrical activation can be

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mapped through the placement of electrode catheters at different positions within the heart chambers.

In an electrophysiological study it is the timing of electrical activation that is important, so to aid this the electrical signals are filtered. Filtering the signals alters the morphology of the electrogram, which is of lesser importance for the study. The electrograms from all the catheters are displayed on a screen from which hard copies can be produced. The sweep speed is also different compared to a standard 12-lead ECG, normally being 4 times as fast at 100 mm/s compared to 25 mm/s. All these differences in the electrical signals can be intimidating; however, with a little patience, they can be easily understood.

STANDARD CATHETER POSITIONS

A 4-wire study is commonly undertaken in patients with suspected supraventricular tachycardia. Figure 10.1 shows catheters introduced via femoral veins and placed at the high right atrium, around the AV node/His region (known as the His catheter), the right ventricular apex, and within the coronary sinus (which has its orifice in the right atrium). The coronary sinus tracks around the mitral annulus, and the catheter records the activation of the left atrium and left ventricle. Additional specialized catheters are used for other arrhythmias, such as a HALO catheter for atrial flutter (Fig. 10.2) and circular mapping catheters for atrial fibrillation (Fig. 10.3).

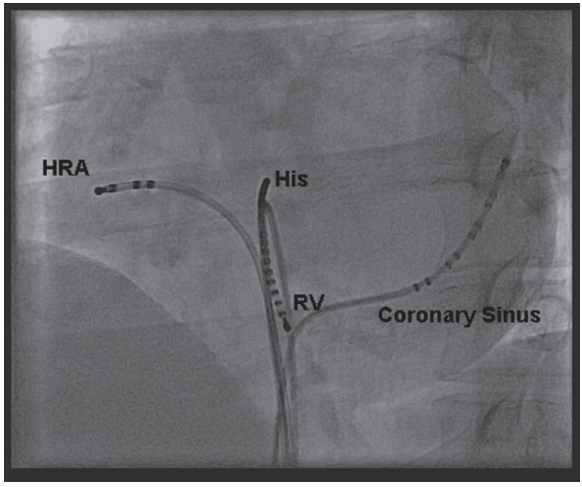


FIG. 10.1. This image is taken from the left anterior oblique projection. In the standard 4 wire procedure electrode catheters are placed at the high right atrium (HRA), His- to measure conduction at the AV node, right ventricular apex (RV), and in the coronary sinus to measure left atrial activation and left ventricular activation.

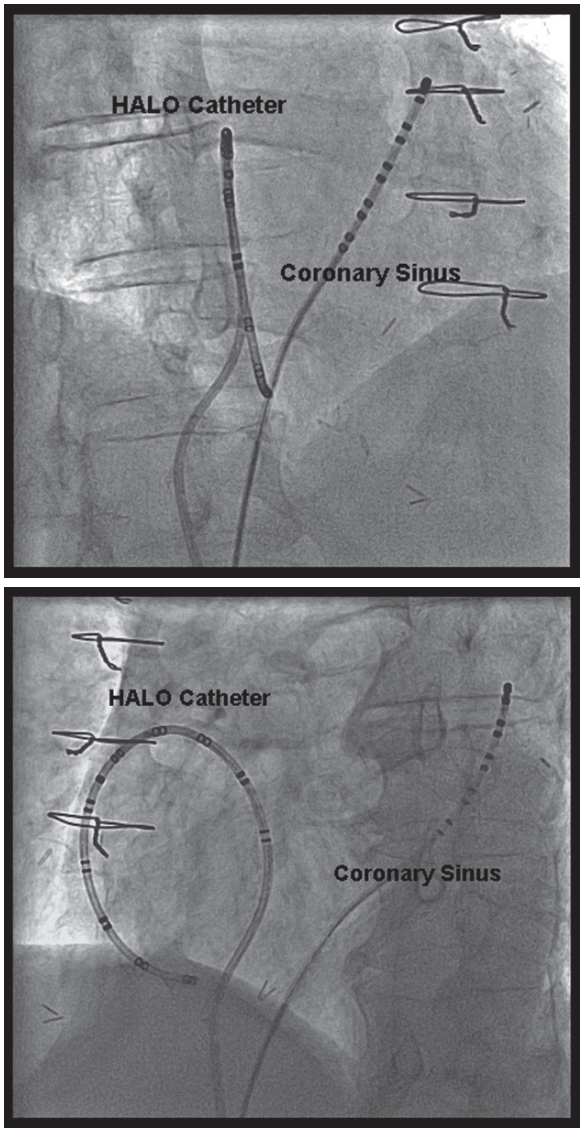


FIG. 10.2. (**a**) This image is taken from the right anterior oblique projection. The HALO catheter is a multipole electrode catheter, commonly used for mapping right atrial flutter and for right sided accessory pathways. (**b**) The electrode catheters have not moved, but this is in the left anterior oblique projection. Here the electrodes on the HALO catheter is seen face on, with a further electrode catheter (a decapole in this example) in the coronary sinus.

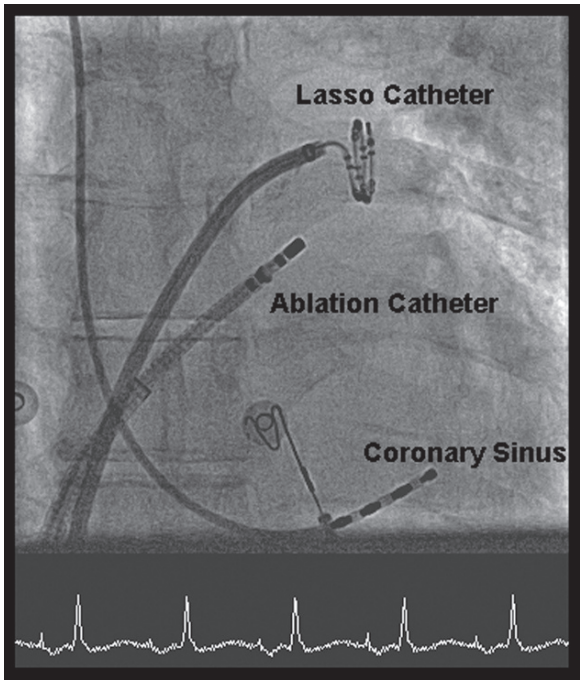


FIG. 10.3. This patient had paroxysmal atrial fibrillation. A Lasso catheter is at the ostium of the left upper pulmonary vein. A further electrode catheter is in the coronary sinus (accessed in this case from the right subclavian vein), and the ablation catheter is in the left atrium. From the ECG, a pacing spike is seen before each p wave, and the p wave has a negative deflection, this is because we are pacing from the coronary sinus to help distinguish signals that arise from the atrium and those that are from the pulmonary vein.

PROCEDURE

When the necessary catheters are in place, the timing of electrical activation is measured (Fig. 10.4). To gain additional information pacing pulses can be delivered from any of the electrodes on any of the catheters. These extra stimuli can trigger the clinical arrhythmia and help to verify the mechanism of the arrhythmia various pacing manoeuvers are used. Once a diagnosis has been made, the abnormal arrhythmia circuit or focus is ablated.

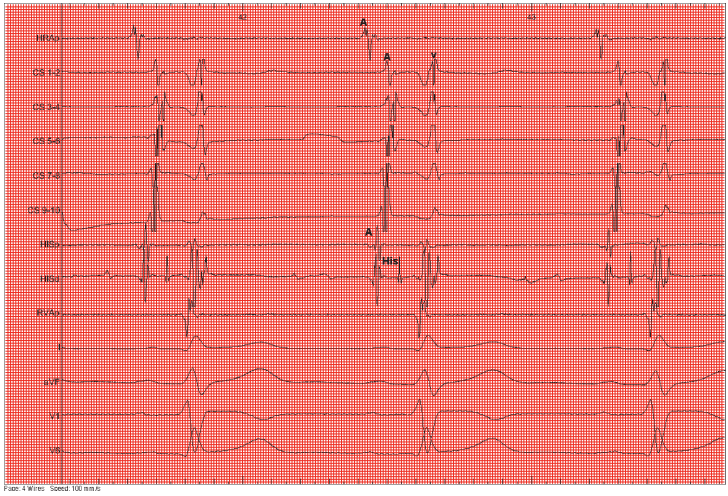


FIG. 10.4. Intracardiac electrograms recorded using a 4 wire setup. Catheters are in the high right atrium (HRAp), Coronary sinus (CS 1–10), His, and right ventricular apex (RVa). Proximal and distal electrodes are denoted by p and d respectively. The paper speed is 100 mm/s which is 4 times faster than a normal 12 lead ECG. In sinus rhythm initial activation is seen in the high right atrium. Activation of the left atrium occurs following this as can be seen by the signals recorded from the coronary sinus catheter. The His catheter records activity in the region of the AV node. The first deflection on the His catheter is local atrial signal (A) entering into the AV node. At the exit of the AV node there is another deflection, seen on the distal His electrode, known as His, the next signal that is seen on the His electrode is the local ventricular activation.

ATRIAL FLUTTER

Atrial flutter is a macroreentrant arrhythmia caused in which the activation wavefront travels around the circuit in perpuity. This is easily appreciated by observing the HALO sequence (Fig. 10.5). The circuit in typical flutter is around the tricuspid valve orifice. As discussed in Chap. X, there are two types of common right atrial flutter, typical counter-clockwise flutter and atypical clockwise flutter, This terminology deals with the tricuspid annulus as a clock face. Radiofrequency ablation of atrial flutter is a very successful procedure with long term success rates greater than 95%. A series of lesions are created from the tricuspid annululs tot eh inferior vena cava, the cavotricuspid isthmus line, creating a line of electrical block (Fig. 10.6). To test that, there is a line of block, a pacing

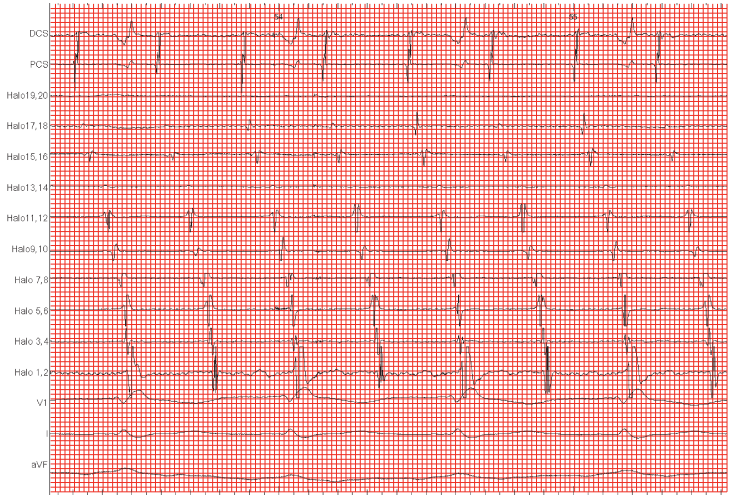


FIG. 10.5. Intracardiac electrograms from a patient with typical right atrial flutter. In this case a HALO catheter has been used (Fig. 10.2). The spread of activation can be seen to go from the proximal HALO electrodes (HALO 19,20, all the way round to the distal HALO electrodes (HALO 1,2). The rest of the circuit that is not seen is at the cavotricuspid isthmus, prior to the circuit continuing at HALO 19,20 again.

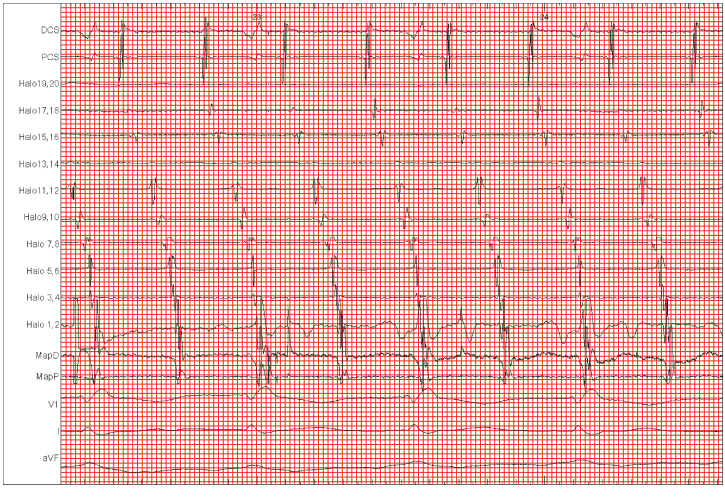
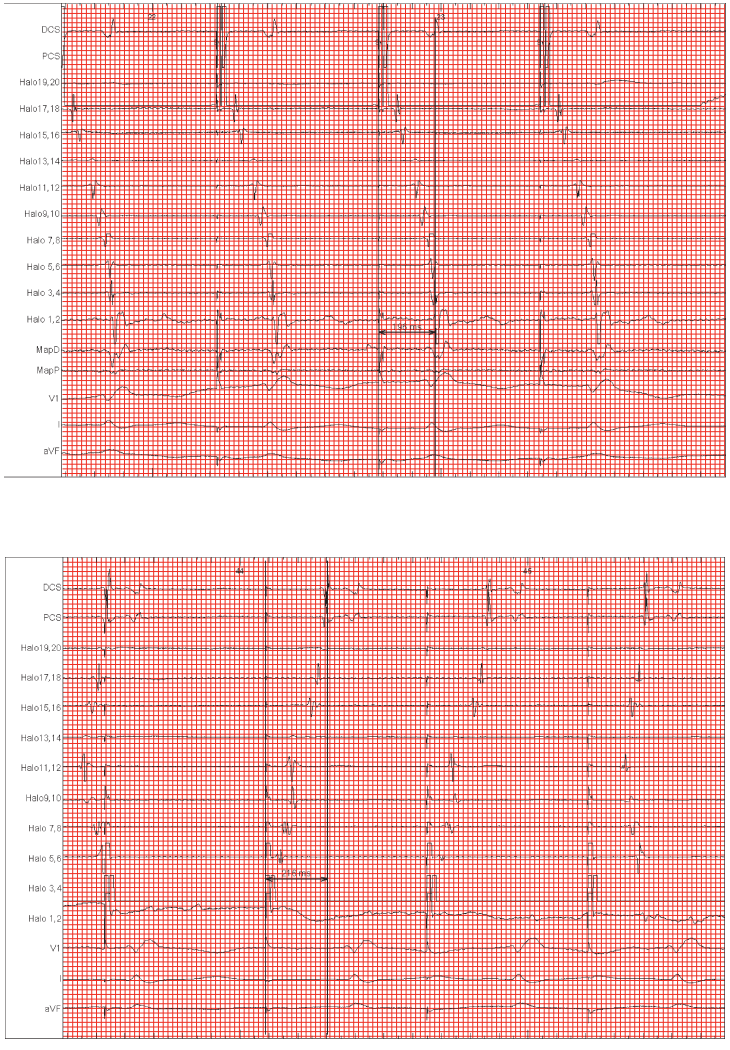


FIG. 10.6. An ablation catheter has been placed at the cavotricuspid isthmus (Map) and radiofrequency energy is being applied to create a line of block to terminate the circuit.

stimulus is applied either side of the line (Fig. 10.7), and the time the stimulus takes to reach another catheter is measured. When there is block, the time taken to reach a further catheter, only a short distance away, is long when on the side of the line furthest away from the stimulating catheter.



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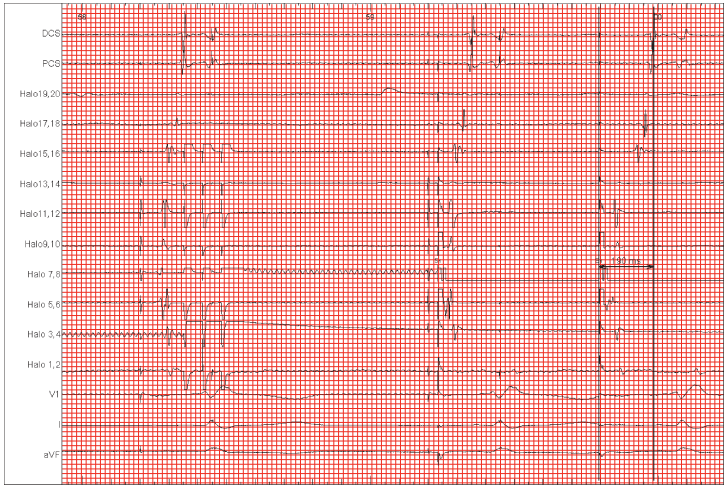
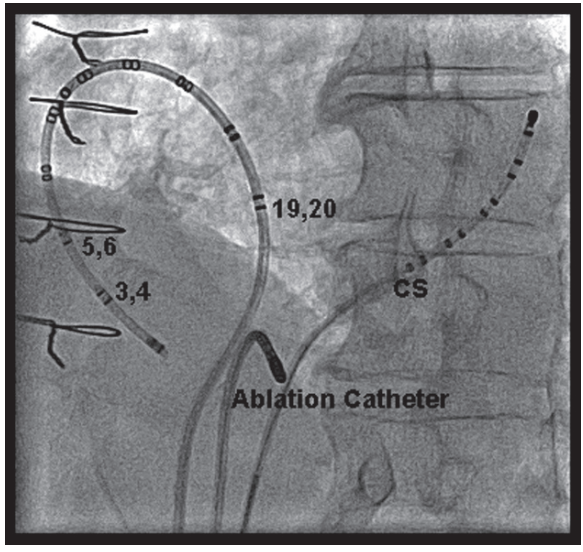

DCS
PCS
Halo19,20
Halo17,18
Halo15,16
Halo13,14
Halo11,12
Halo9,10
Halo 7,8
Halo 5,6
Halo 3,4
Halo 1,2
V1
II
aVF


19,20
5,6
3,4
CS
Ablation Catheter

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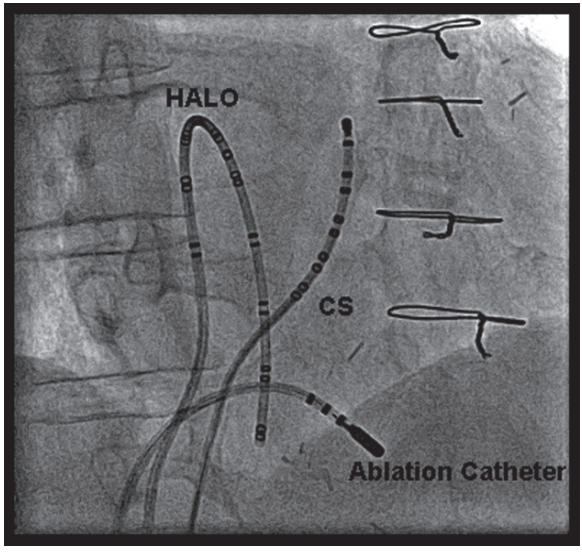


FIG. 10.7. Testing the line of block. The atrial flutter has been terminated with radiofrequency ablation. (**a**) Pacing stimulus is applied at the proximal pole of the coronary sinus catheter. This is close to the septal side of the line of block. The amount of time the stimulus takes to get from the CS catheter to the distal HALO poles (HALO 1,2) is measured at 196 ms. (**b**) Pacing stimulus is from poles 3,4 of the HALO catheter, electrical activation now spreads in an anticlockwise direction to HALO 19,20, rather than being able to go in a clockwise direction because of the line of block at the cavotricuspid isthmus. The time taken to reach the CS catheter is 216 ms, (**c**) Pacing stimulus has been applied from poles 7,8 of the HALO catheter. The time taken to reach the CS catheter is only 196 ms. This is because the electrical activation still reaches the CS catheter in an anticlockwise direction, and therefore takes a shorter time to reach this catheter. If the line of block was not complete and electrical activation spread in clockwise direction, it would take longer to reach the CS catheter than in (**b**) as it is further away from the catheter. (**d**) Fluoroscopic image in the left anterior oblique projection, demonstrating the different electrode pairs. (**e**) Fluoroscopic image in the right anterior oblique projection, the ablation catheter is at the ventricular end of the cavotricuspid isthmus line.

ATRIO-VENTRICULAR NODAL RE-ENTRY TACHYCARDIA

As discussed in Chap. X, AVNRT is another re-entrant rhythm. The circuit in AVNRT involves reentry along two pathways, a slow and fast pathway. These pathways are so called because of their conduction properties, with the fast pathway having a fast conduction

time, compared to the slow pathway, which has a slower conduction time. The other important difference is the respective refractory period of the two pathways, with the fast pathway having a relatively long refractory period and the slow pathway having a relatively short refractory time. In typical AVNRT, the electrical impulse travels down the slow pathway, entering the compact AV node, before going to the ventricle via the bundle of His, and turning around and entering the fast pathway and then the atrium, and perpetuating by entering the slow pathway again. In atypical AVNRT the circuit is reversed.

In an EP study, the refractory periods of the slow and fast pathways can be measured. This is done by programmed electrical stimulation, whereby electrical impulses are delivered to a specific electrode, for example in the right atrium, and then progressively earlier impulses are delivered (Figs. 10.8 and 10.9). When the fast pathway is refractory, a "jump" is seen as conduction to the ventricle goes via the slow pathway to the AV node. Although many people have slow and fast pathways, they do not all have AVNRT, so the electrophysiologist tries to initiate the tachycardia at the EP study. When tachycardia is initiated, extra stimuli are delivered at various timings to prove the diagnosis (Fig. 10.10).

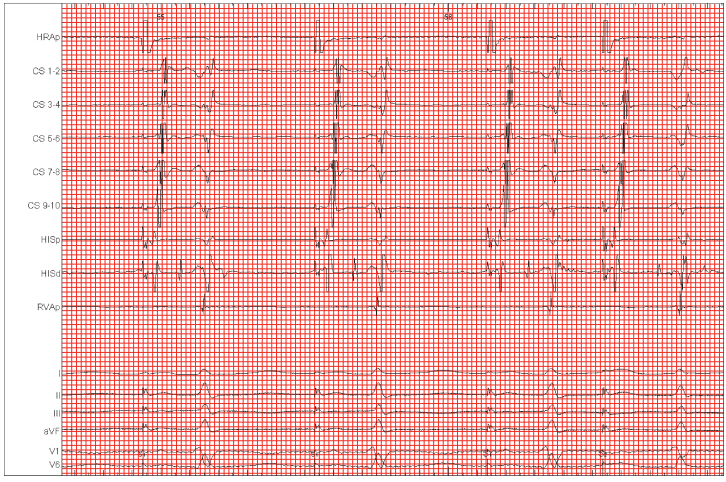


FIG. 10.8. A pacing stimulus is delivered via the HRA catheter at a given cycle length. Following eight paced beats, an earlier stimulus is delivered, an extra. In normal individuals the AV node will decrement, that is the stimulus will take longer to get to the ventricles. This can be seen by comparing the A–H intervals on the traces.

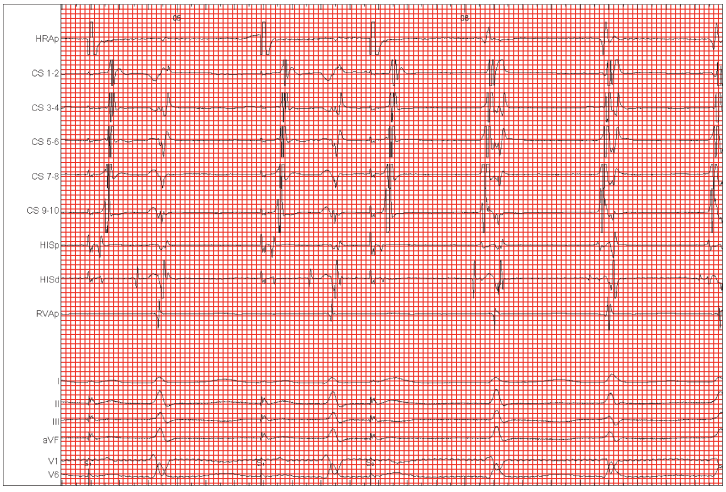


FIG. 10.9. A drive train with an earlier extra in this case causes a marked delay in the impulse getting to the ventricle, "a jump." This is due to the "fast" pathway being refractory, but the "slow" pathway being able to conduct the impulse to the ventricle. This His is far later than previously seen. This causes initiation of AVNRT. Once it gets to the AV node it propagates both to the ventricle and retrogradely up the fast pathway to the atria, before propagating down the slow pathway again.

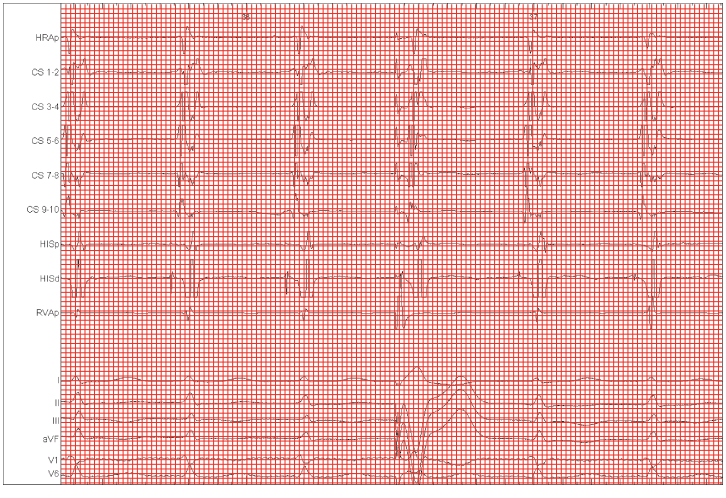


FIG. 10.10. An extra stimulus delivered from the RV catheter is delivered during AVNRT. It is delivered at a time when the bundle of His is refractory, "His synchronous." If there was an accessory pathway that mediated the tachycardia the stimulus would be able to reach the atrium and change the atrial activation sequence.

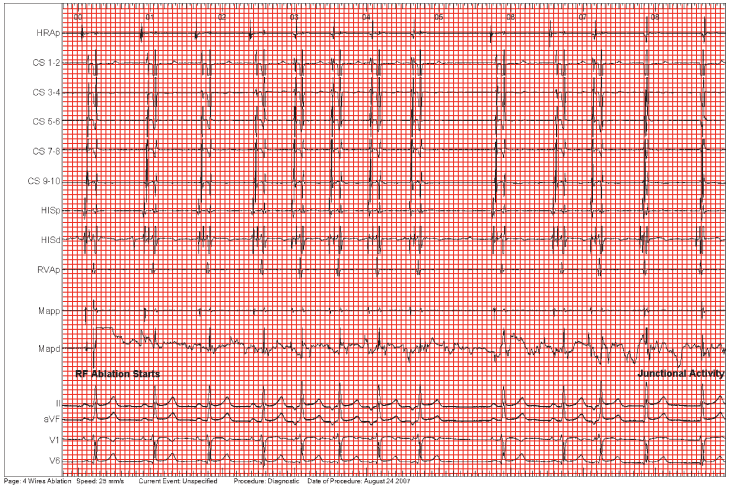


FIG. 10.11. Slow pathway ablation for a patient with AVNRT. The paper speed is at 25 mm/s, the same as for a 12 lead ECG. Here after starting ablation in sinus rhythm junctional activity is quickly seen. This is one of the markers of a good lesion for slow pathway ablation. A full 60 s delivery was applied. The patient has been symptom free since.

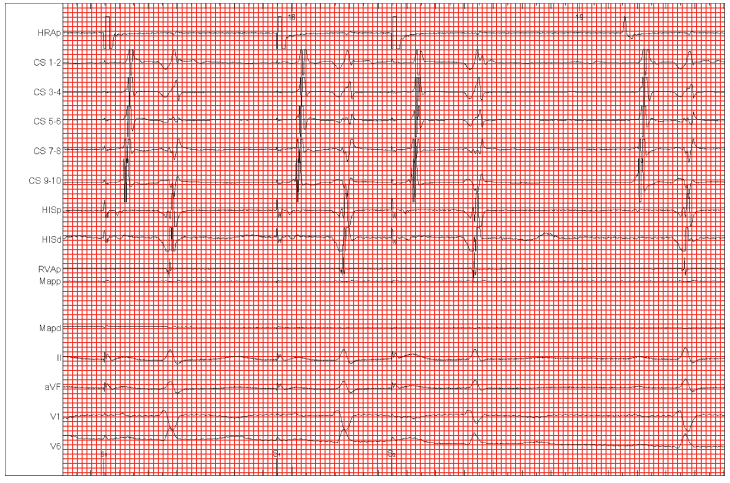


FIG. 10.12. Following ablation of the slow pathway an atrial extra does not produce a jump from the fast pathway to the slow pathway.

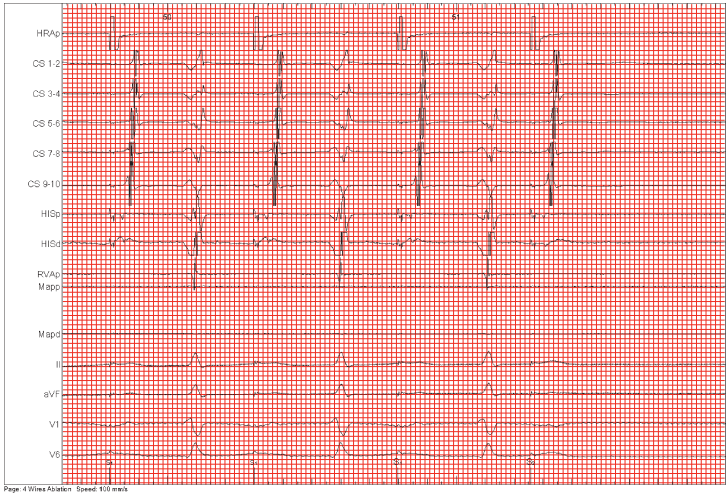
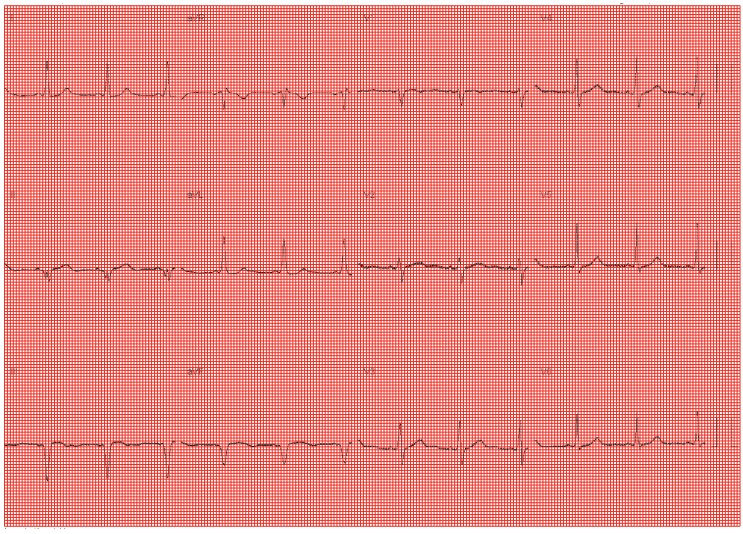


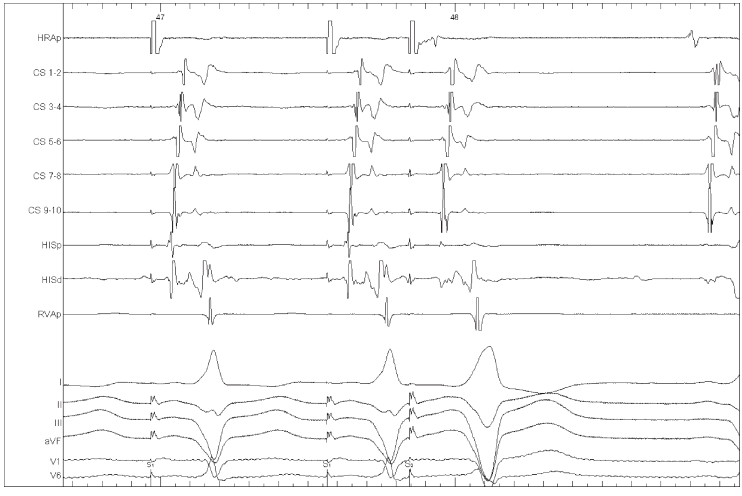
FIG. 10.13. Further testing confirms the slow pathway has been ablated and AVNRT cannot be re-induced.

Once the diagnosis is certain, the electrophysiologist ablates (Fig. 10.11) the slow pathway to stop the circuit and then retests it (Figs. 10.12, and 10.13). This is a very effective treatment for AVNRT, with a success rate of 95%. The complication rate is acceptably low at 1%, needing a permanent pacemaker due to AV damage result in heart block.

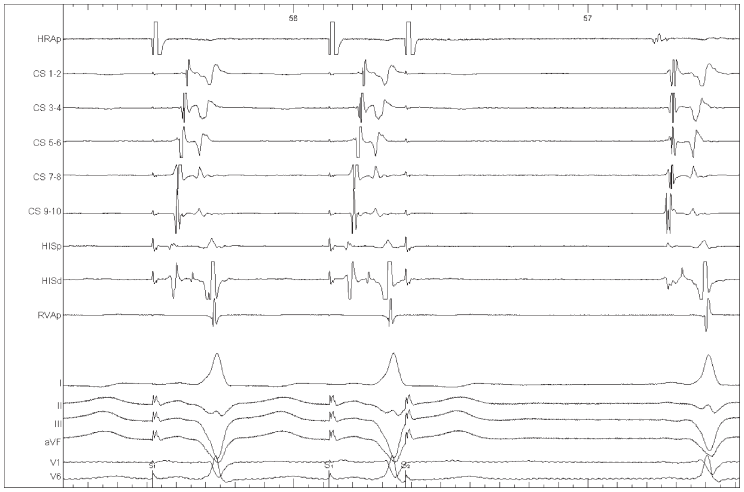
ATRIO-VENTRICULAR RE-ENTRY TACHYCARDIA

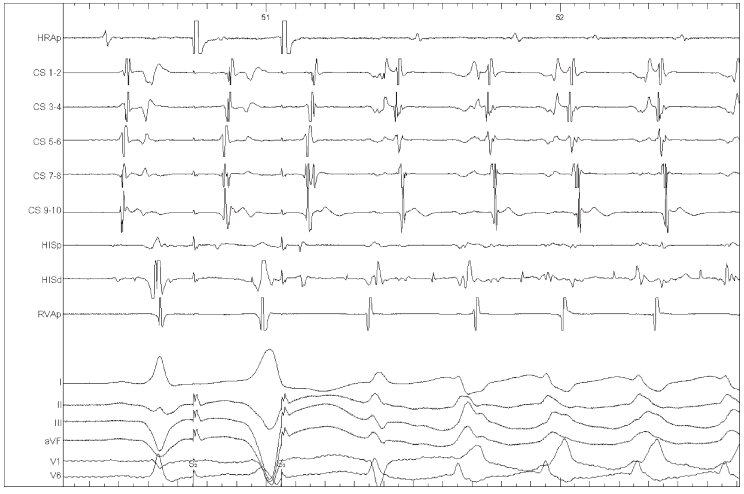
As discussed in Chap. X AVRT is also a macro re-entrant tachycardia, utilizing an accessory pathway between the atrium and the ventricle. With AVRT electrophysiologists use a number of terms to describe the circuit. The circuit in orthodromic AVRT is from the atrium, into the AV node, down the bundle of His to activate the ventricle and back to the atrium via an accessory pathway (Fig. 10.14). Antidromic AVRT refers to the circuit being in the opposite direction. An accessory pathway is manifest when it conducts from the atrium to the ventricle as well as from the ventricle to the atrium. A concealed accessory pathway is when the pathway conducts only from the ventricle to the atrium and not from the atrium to the ventricle (Figs. 10.15 and 10.16). Concentric activation refers to activation via the AV node, whereas eccentric activation refers to activation which is not solely via the AV node, but is instead via an accessory pathway (Figs. 10.17 and 10.18). The



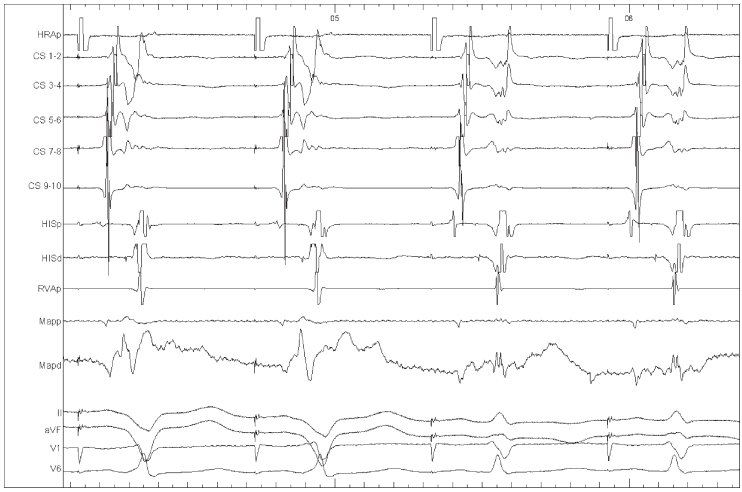

47
48
HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
I
II
III
aVF
V1
V6
St
St
St

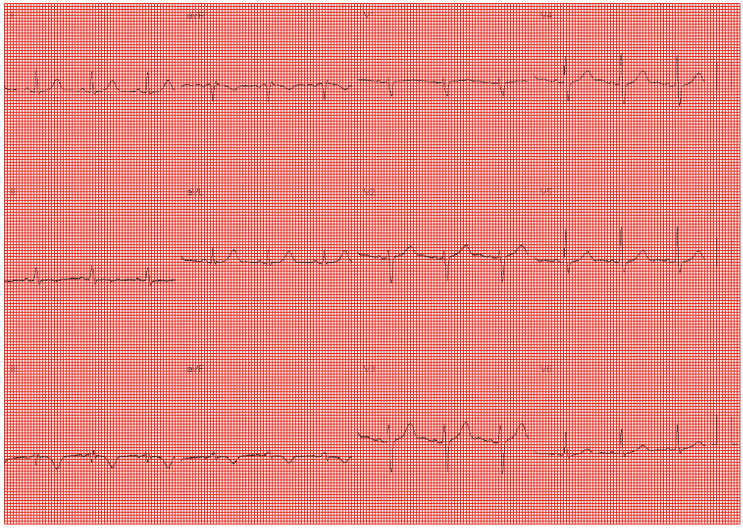
(continued)


58
57
HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
I
II
III
aVF
V1
V6
S1
S1
S2


51
52
HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
I
II
III
aVF
V1
V6
S2
S2

(continued)


HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
Mapp
Mapd
I
aVF
V1
V6



(continued)

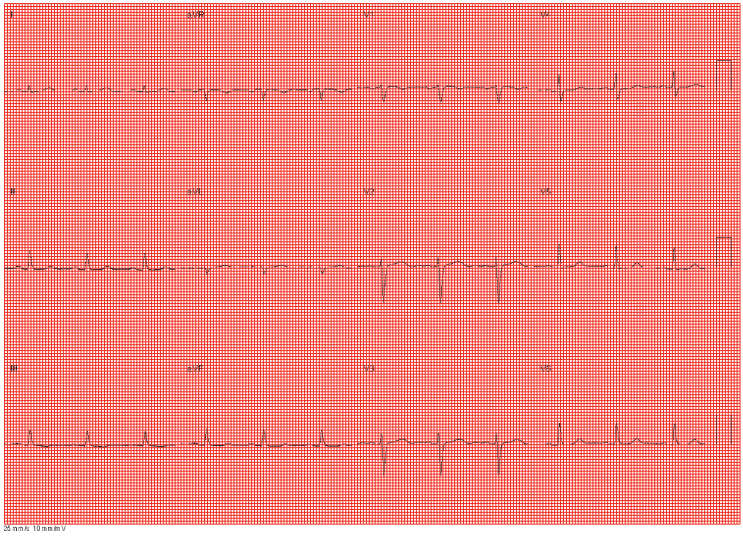
term Wolff–Parkinson–White syndrome is used when the pathway is manifest producing a short PR interval, a delta wave and tachycardia causing palpitations.

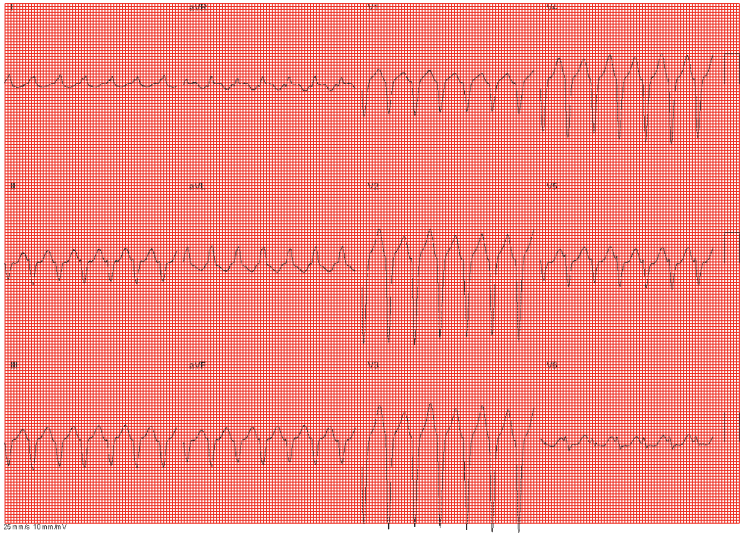
The treatment for AVRT is ablation of the accessory pathway. The pathway is located by observing the activation pattern in the different chambers of the heart. The ablation catheter is then manipulated to the accessory pathway and energy is delivered to ablate the pathway (Figs. 10.19–10.22).

ATRIAL FIBRILLATION

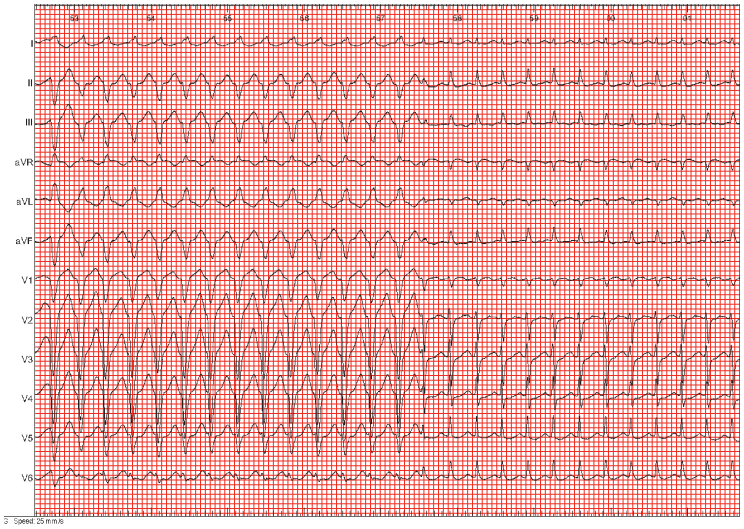
Atrial fibrillation is a far more difficult arrhythmia to treat with catheter ablation. A key finding in 1998 was that ectopic beats originating from the pulmonary veins trigger atrial fibrillation in a number of patients. Catheter ablation of paroxysmal atrial fibrillation can be achieved with a high success rate by electrically isolating the pulmonary veins. Access to the left atrium is via a trans-septal puncture (Fig. 10.16), which carries a risk of cardiac tamponade of 1%. Once in the left atrium, a circular mapping

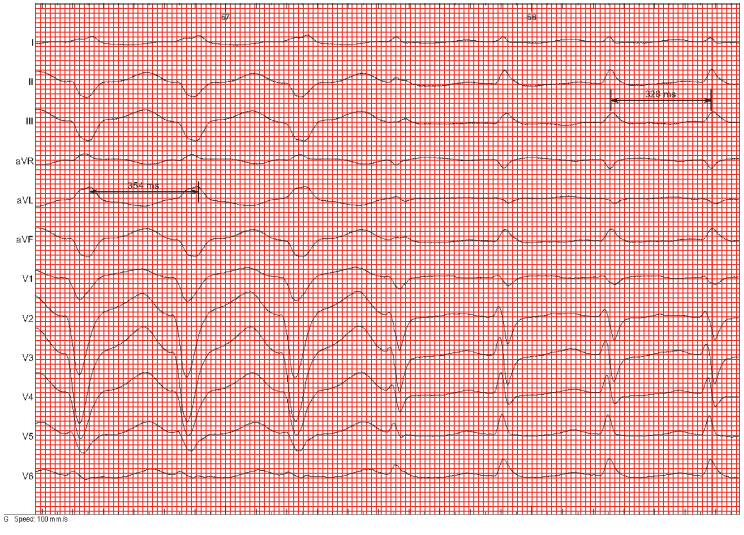
FIG. 10.14. This patient had been suffering from palpitation since the age of 16, but had never managed to have an ECG in time to capture the arrhythmia. (**a**) Surface ECG. From the surface ECG, there is clear pre-excitation with a positive delta wave in Lead I, aVL and V1 and V2, with negative delta waves in the inferior leads. This is suggestive of a left posteroseptal accessory pathway. (**b**) Antegrade pacing. Here stimuli are delivered from the HRA catheter. Delta waves can be seen on the surface ECG, which is more pronounced on the extra, due to the AV node being more refractory than the pathway. The earliest ventricular component is at CS 7,8. (**c**) Antegrade pacing with AV block. Here an earlier atrial extra stimulus is delivered and this does not conduct to the ventricles. This means that both the AV node and the pathway are refractory. (**d**) The coronary sinus catheter has been moved back to try and localize the pathway more accurately. An atrial extra stimulus is now able to initiate orthodromic AVRT. The spread of activation goes from the AV node down the bundle of His and via the ventricles back through the accessory pathway, to its atrial insertion, CS 3,4. This then perpetuates. (**e**) A mapping catheter is moved to the accessory pathway and energy delivered. In the first two beats the atrial and ventricular signals on the CS channels are closely spaced, due to the accessory pathway functioning, on the last two beats the ventricular component is later, as the pathway has been ablated and no longer functions. (**f**) Final surface ECG, with no pre-excitation.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25 mm/s 10 mm/mV

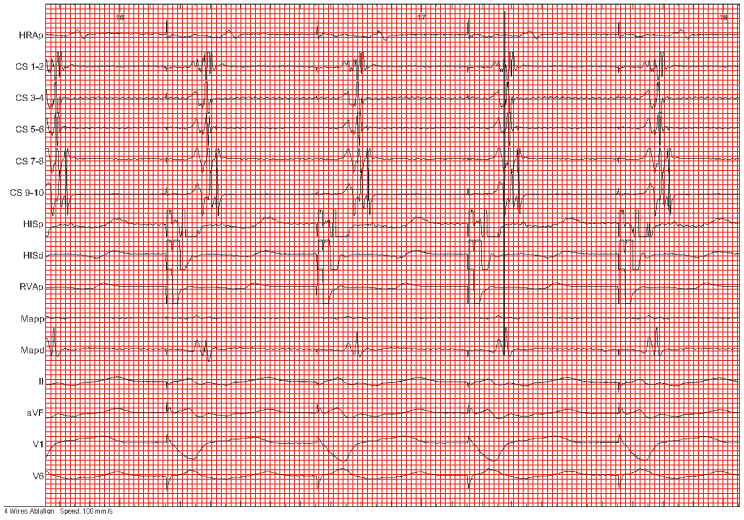

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
25 mm/s 10 mm/mV

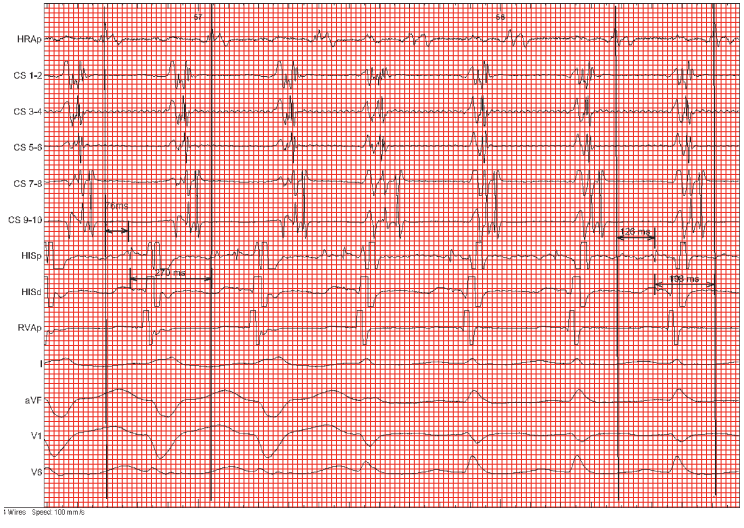
(continued)


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
Speed: 25 mm/s


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
354 ms
328 ms
Speed: 100 mm/s

(continued)


HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
Mapp
Mapd
II
aVF
V1
V5
4 Wires Ablation Speed: 100 mm/s


HRAp
CS 1-2
CS 3-4
CS 5-6
CS 7-8
CS 9-10
HISp
HISd
RVAp
I
aVF
V1
V5
1 Wires Speed: 100 mm/s

(continued)

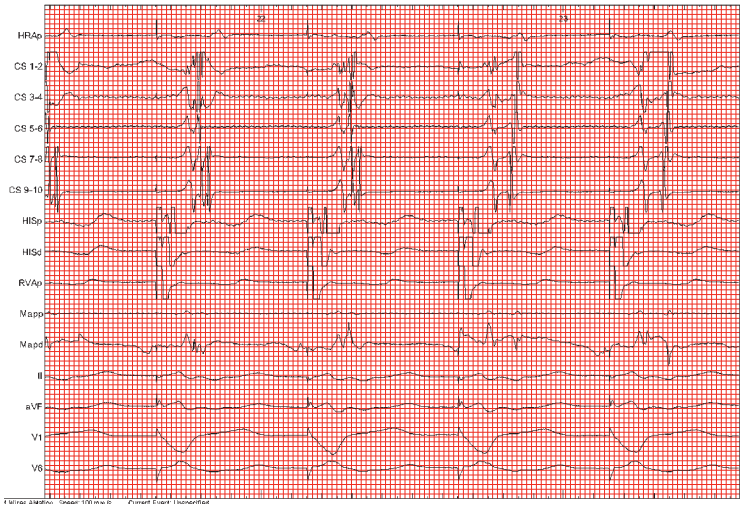


FIG. 10.15. This patient had recurrent regular rapid palpitations, and was intolerant of a number of anti-arrhythmics. (**a**) 12 lead ECG. There is no obvious pre-excitation seen on the 12 lead ECG. (**b**) 12 lead ECG during tachycardia. The patient has a regular tachycardia with a left bundle branch block (LBBB) morphology, without discernible p waves. (**c**) The LBBB resolves, however the tachycardia continues. (**d**) When the cycle length of the tachycardia is measured it is longer (i.e., slower) with the bundle branch block, than when the bundle branch resolves. (**e**) The time taken from the atrial activation until His activation is 76 ms with the LBBB morphology compared to 125 ms when the bundle branch block resolves. However the time from V to A (that is the return leg of the circuit) is longer with the LBBB compare to when the bundle branch has resolved. The explanation is that there has to be a left sided accessory pathway. The circuit is thus atrium–AV node–left ventricle–left sided accessory pathway and then back to the atrium. (**f**) The intracardiac signals confirm this deduction, as can be seen when pacing from the His catheter, the earliest A activation is on poles 3,4 of the CS catheter (Caliper to help). (**g**) When energy is applied to this area the pathway is ablated, and a change in the VA time can be observed, as well as a change in the activation sequence on the coronary sinus catheter.

catheter is placed in the pulmonary veins. This can detect if there are any muscular sleeves that are capable of conducting electricity from the veins to the left atrium. Another catheter is then manipulated to ablate these muscular sleeves to electrically isolate the veins (Figs. 10.17 and 10.18).

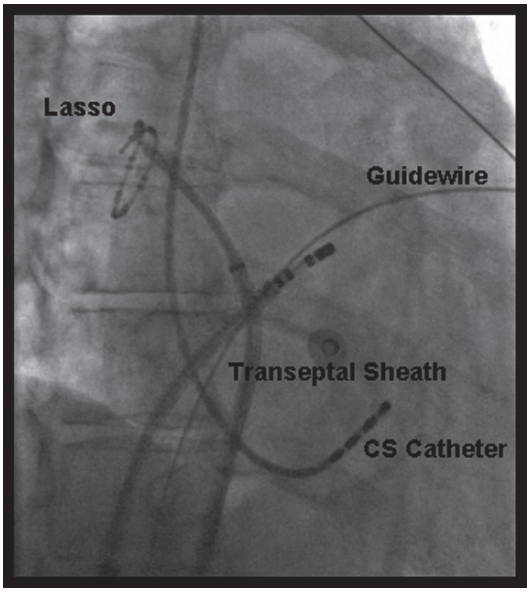


FIG. 10.16. Transeptal puncture. This patient underwent a robotic AF ablation. One transeptal puncture has already been made and a Lasso catheter has been placed in the right upper pulmonary vein. A guide wire is left in place across a second transeptal puncture, across which a robotic transeptal sheath will follow.

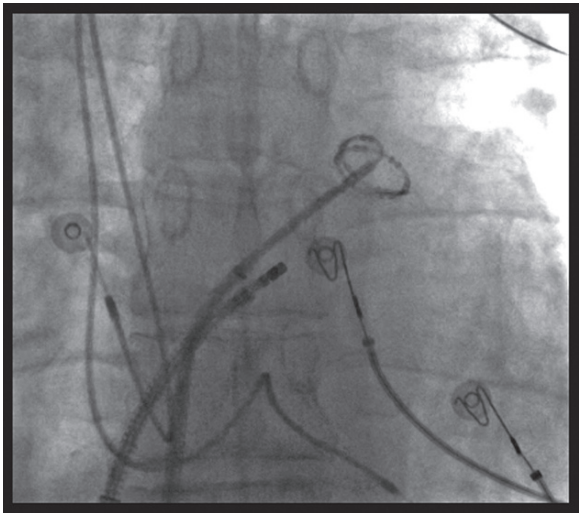


FIG. 10.17. This patient had persistent atrial fibrillation, as well as previous AV block. An AF ablation was performed to allow AV synchrony with his pacemaker. Here the Lasso catheter is in the left upper pulmonary vein, with the ablation catheter crossing the septum.

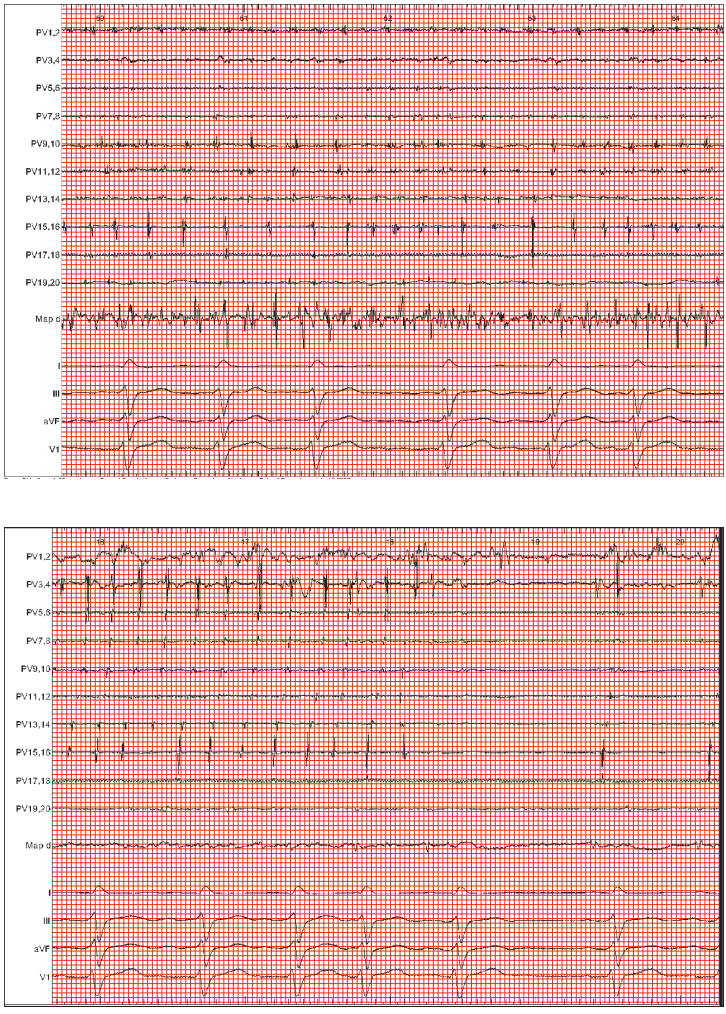


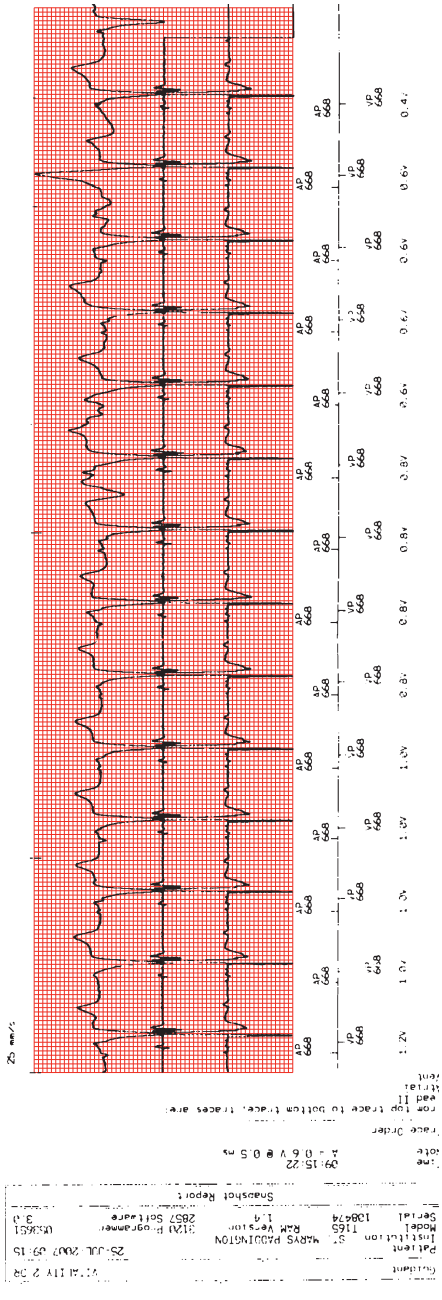
Fig. 10.18. This 68-year-old man had become very lethargic with the onset of his paroxysmal atrial fibrillation and underwent AF ablation. (**a**) The surface ECG leads I, III, aVF and V1; it is clear that the patient is in atrial fibrillation. The intracardiac signals from the circular pulmonary vein catheter (Lasso) show very fast activity and there is almost continuous activity on the mapping catheter at the left atrial – left upper pulmonary vein junction. (**b**) Radiofrequency ablation at the position seen in (**a**) results in termination of the pulmonary vein tachycardia which results in conversion from atrial fibrillation to sinus rhythm. The patient continues to feel better following the ablation. Paper speed 50 mm/s.

ATRIAL TACHYCARDIAS

Atrial tachycardias can be some of the most challenging arrhythmias to treat with catheter ablation. There are several types of atrial tachycardia, ranging from focal atrial tachycardia to macro-re-entrant tachycardia, of which typical atrial flutter is just one example. The tachycardia is mapped using the electrode catheters to define the circuit and point of earliest activation. In order to aid identification of the tachycardia, additional 3D mapping systems can also be used.

PACEMAKERS AND IMPLANTABLE DEFIBRILLATORS

The basic functions of permanent pacemakers, biventricular pacemakers and implantable cardiac defibrillators (ICD) have been dealt with in Chap. X. Patients are followed up in specialist pacemaker clinics to ensure the pacemaker or ICD continues to function properly. The pacemaker functions can be altered remotely using a pacing system analyzer (PSA), which is a special computer that communicates either via a wand placed over the pacemaker or wirelessly via radiowaves. Using the PSA, all functions of the device (pacemaker/ICD) can be accessed and altered. The device also stores information on the therapy that it has delivered since the last pacing check; for example the amount of time the patient is ventricularly paced, or the number of shocks delivered. This is helpful to the technician and physician as it allows more tailored individual therapy. Rarely, pacemakers fail; this can be due to problems with the lead, problems at the connection between the lead and the device, and also problems with the device itself (Figs. 10.19–10.21).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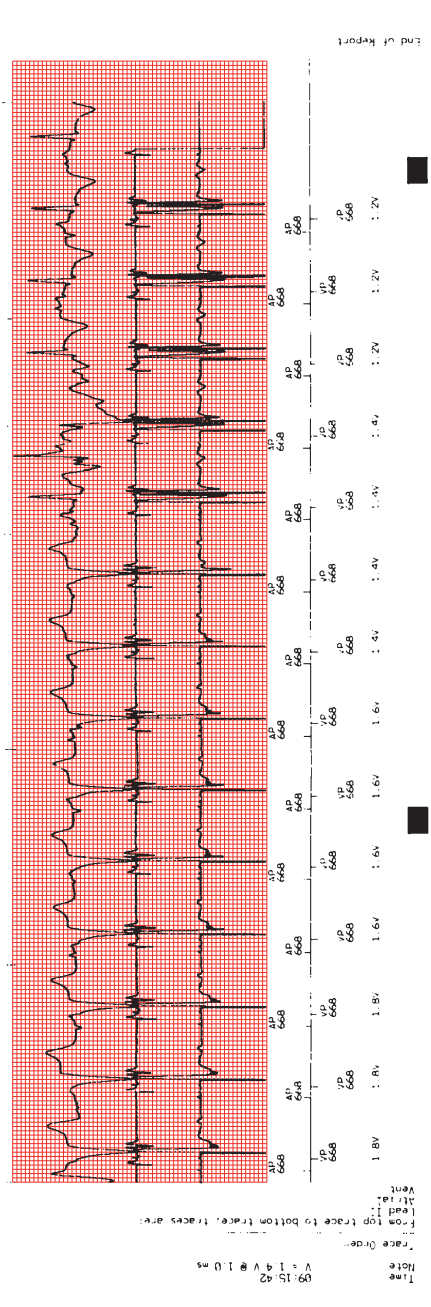
Vent
Atrial
25 mm/s

(continued)


Time
Date
09:15:42
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Trace Order:
From top trace to bottom trace, traces are:
lead I:
Atrial
Vent
End Of Report

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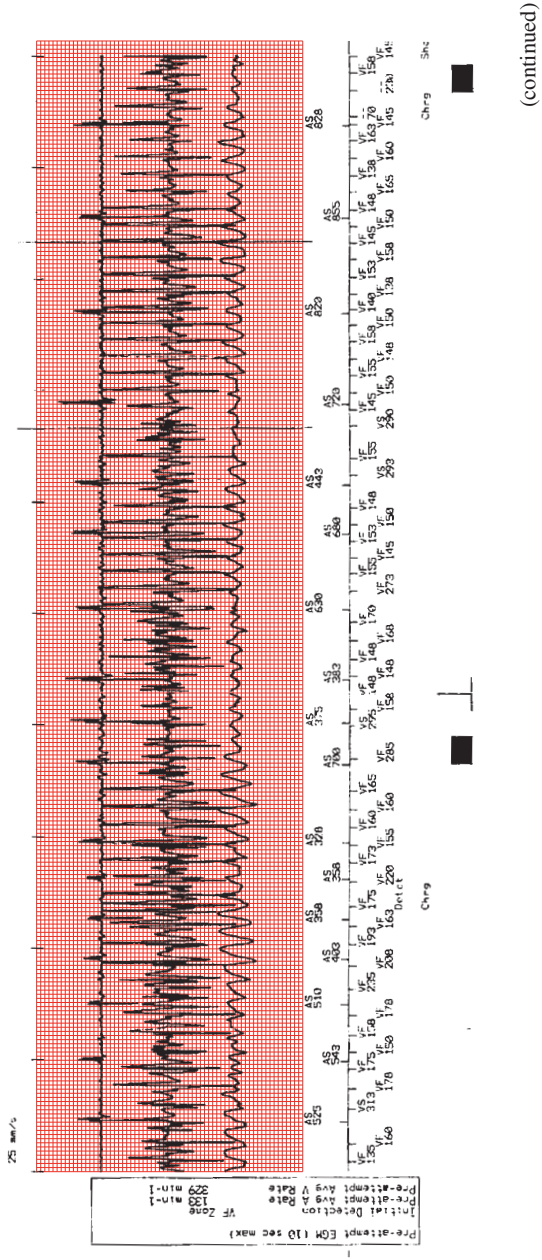
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Initial Detection
Pre-attempt Avg A Rate
Pre-attempt Avg V Rate
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139 min-1
VF Zone
25 mm/s
Delay
Chrg
Shc

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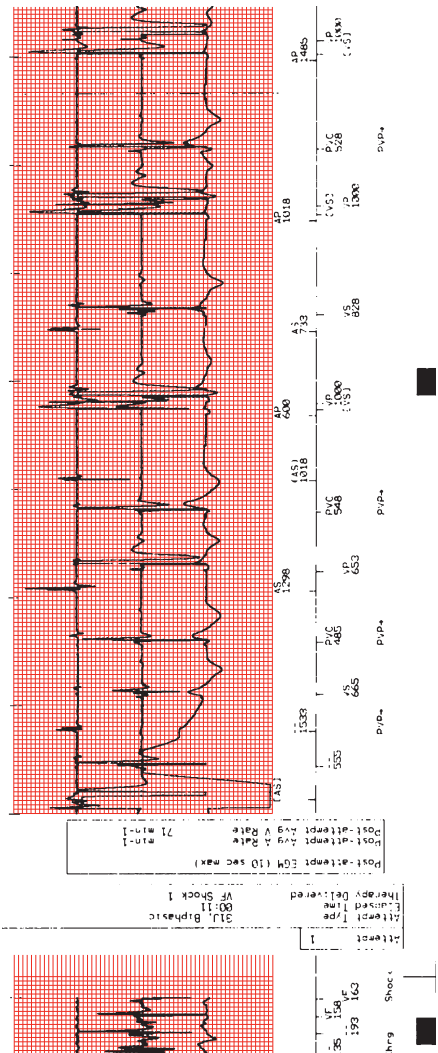
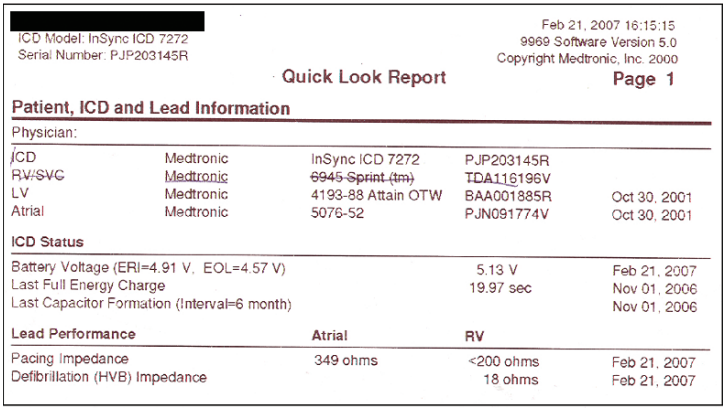


FIG. 10.19. A 37-year-old lady attends for a check following several discharges from her ICD. In the following images the sequence from top to bottom is, Surface ECG, Atrial lead, Ventricular lead, Marker channel. The Atrial and ventricular channels can detect either sensed (As or Vs) or paced (Ap or Vp) stimuli. The numbers on the marker channel refer to the duration from the previous stimuli in milliseconds. (**a**) Atrial threshold check. The pacing stimulus to the atrial channel is reduced from 1.2 to 0.4 V. A p wave can be seen preceding each paced QRS complex on the surface ECG. When the stimulus is between 0.6 and 0.4 V there is a loss of capture as the p wave can no longer be seen. The threshold is therefore 0.6 V. (**b**) Ventricular threshold check. The pacing stimulus to the ventricular channel is decreased from 1.8 to 1.4 V. The surface ECG changes between a paced, broad complex beat to narrow complex, non paced beats at 1.6 V. The threshold is therefore 1.8 V. (**c**) Stored electrogram from a shock. Here the patient is in torsade de pointes. The top channel (atrial) denotes dissociated atrial complexes, from the lower ventricular channel. The marker channel shows that the device has detected VF (Detct) and the device starts to charge (Chrg); when it is fully charged it delivers a shock. (**d**) Stored electrogram following the shock (contiguous to 30C). Following the shock, sinus rhythm is restored. *PVP* post ventricular pacing.



[redacted]
ICD Model: InSync ICD 7272
Serial Number: PJP203145R

Feb 21, 2007 16:15:15
9969 Software Version 5.0
Copyright Medtronic, Inc. 2000

Quick Look Report

Page 1

Patient, ICD and Lead Information

Physician:

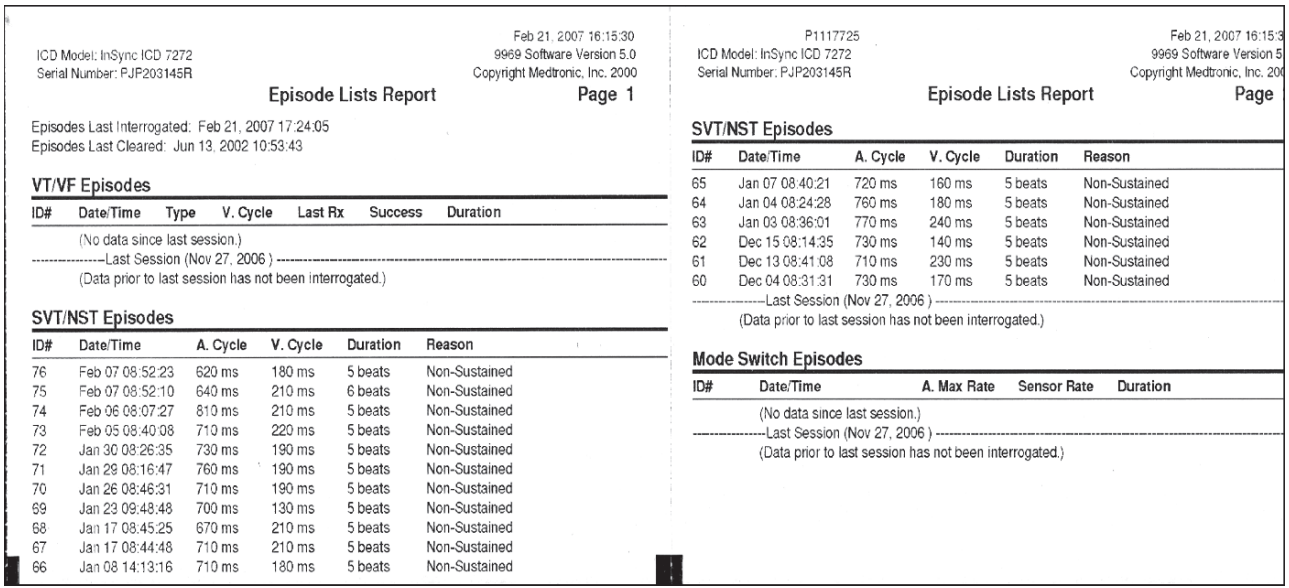
ICD	Medtronic	InSync ICD 7272	PJP203145R	
RV/SVC	Medtronic	6945 Sprint (tm)	TDA116196V	
LV	Medtronic	4193-88 Attain OTW	BAA001885R	Oct 30, 2001
Atrial	Medtronic	5076-52	PJN091774V	Oct 30, 2001

ICD Status

Battery Voltage (ERI=4.91 V, EOL=4.57 V)		5.13 V	Feb 21, 2007
Last Full Energy Charge		19.97 sec	Nov 01, 2006
Last Capacitor Formation (Interval=6 month)			Nov 01, 2006

Lead Performance	**Atrial**	**RV**	
Pacing Impedance	349 ohms	<200 ohms	Feb 21, 2007
Defibrillation (HVB) Impedance		18 ohms	Feb 21, 2007

FIG. 10.20. (**a**) At a routine ICD check the RV lead has a low impedance (<200 ohm), suggestive of insulation failure. (**b**) When the cycle length of the episodes of nonsustained VT are analyzed, the cycle length is found to be very short (140 ms), and cannot be physiological. Thankfully this patient had not received any shocks from the defibrillator. The RV lead was replaced.



ICD Model: InSync ICD 7272
Serial Number: PJP203145R

Feb 21, 2007 16:15:30
9969 Software Version 5.0
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Episode Lists Report Page 1

Episodes Last Interrogated: Feb 21, 2007 17:24:05
Episodes Last Cleared: Jun 13, 2002 10:53:43

VT/VF Episodes

ID#	Date/Time	Type	V. Cycle	Last Rx	Success	Duration

(No data since last session.)
----------Last Session (Nov 27, 2006) ----------
(Data prior to last session has not been interrogated.)

SVT/NST Episodes

ID#	Date/Time	A. Cycle	V. Cycle	Duration	Reason
76	Feb 07 08:52:23	620 ms	180 ms	5 beats	Non-Sustained
75	Feb 07 08:52:10	640 ms	210 ms	6 beats	Non-Sustained
74	Feb 06 08:07:27	810 ms	210 ms	5 beats	Non-Sustained
73	Feb 05 08:40:08	710 ms	220 ms	5 beats	Non-Sustained
72	Jan 30 08:26:35	730 ms	190 ms	5 beats	Non-Sustained
71	Jan 29 08:16:47	760 ms	190 ms	5 beats	Non-Sustained
70	Jan 26 08:46:31	710 ms	190 ms	5 beats	Non-Sustained
69	Jan 23 09:48:48	700 ms	130 ms	5 beats	Non-Sustained
68	Jan 17 08:45:25	670 ms	210 ms	5 beats	Non-Sustained
67	Jan 17 08:44:48	710 ms	210 ms	5 beats	Non-Sustained
66	Jan 08 14:13:16	710 ms	180 ms	5 beats	Non-Sustained

P1117725
ICD Model: InSync ICD 7272
Serial Number: PJP203145R

Feb 21, 2007 16:15:3
9969 Software Version 5
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Episode Lists Report Page

SVT/NST Episodes

ID#	Date/Time	A. Cycle	V. Cycle	Duration	Reason
65	Jan 07 08:40:21	720 ms	160 ms	5 beats	Non-Sustained
64	Jan 04 08:24:28	760 ms	180 ms	5 beats	Non-Sustained
63	Jan 03 08:36:01	770 ms	240 ms	5 beats	Non-Sustained
62	Dec 15 08:14:35	730 ms	140 ms	5 beats	Non-Sustained
61	Dec 13 08:41:08	710 ms	230 ms	5 beats	Non-Sustained
60	Dec 04 08:31:31	730 ms	170 ms	5 beats	Non-Sustained

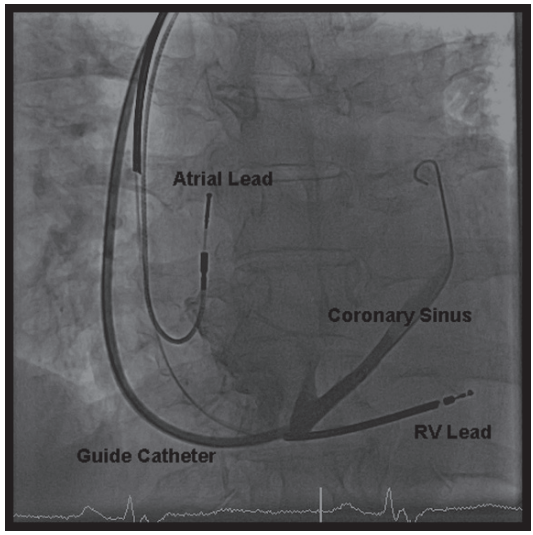
----------Last Session (Nov 27, 2006) ----------
(Data prior to last session has not been interrogated.)

Mode Switch Episodes

ID#	Date/Time	A. Max Rate	Sensor Rate	Duration

(No data since last session.)
----------Last Session (Nov 27, 2006) ----------
(Data prior to last session has not been interrogated.)

FIG. 10.20. (continued) (b)



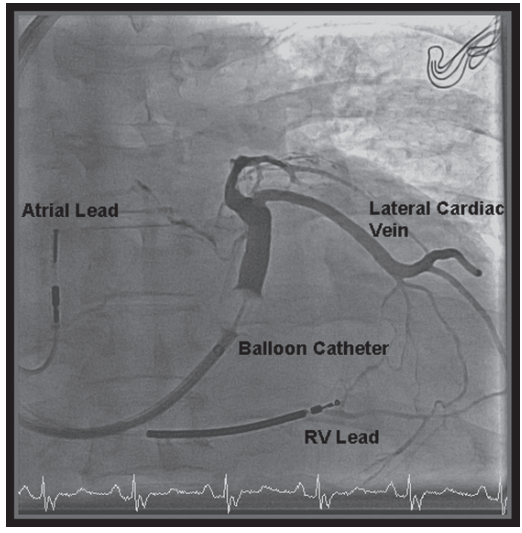
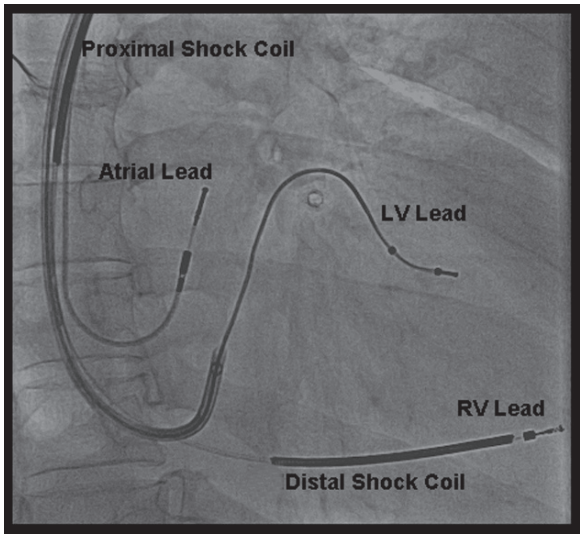


FIG. 10.21. Implantation of a Biventricular pacemaker and ICD. This patient with severe heart failure and non sustained VT on a Holter monitor, had an ejection fraction of less than 30%. A biventricular pacemaker with ICD was implanted. (**a**) The positions of the right atrial lead, in the right atrial appendage and the RV lead, with integrated shock coils have already been implanted. A guide catheter is in the coronary sinus ostium and contrast has been injected. (**b**) A balloon catheter is inflated and contrast injected, which



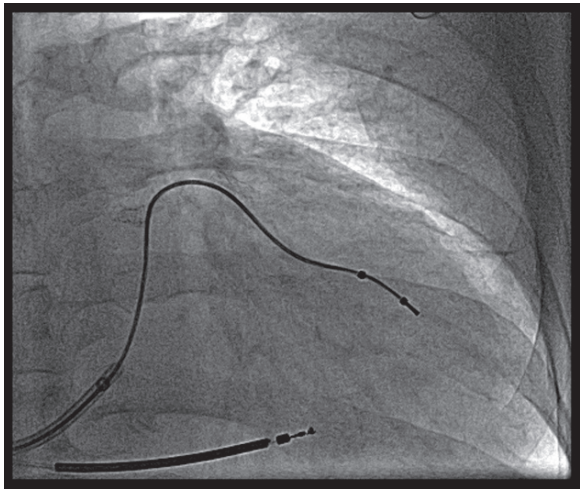


FIG. 10.21. (continued) more clearly demonstrates the lateral cardiac vein. (**c**) The left ventricular lead is inserted into the lateral cardiac vein. (**d**) The final position of the LV lead. This patient's exercise tolerance improved dramatically following the insertion of this device.

Appendix A

Appendix A

APPENDIX A MIXED ECG QUIZZES

If you have read the previous pages you should now be able to interpret Figs. A.1–A.38. The aim of the book is not to turn you into an expert but to enable you to read basic electrocardiogram. This should give you a certain amount of satisfaction and at least prove beneficial to the patients under your care.

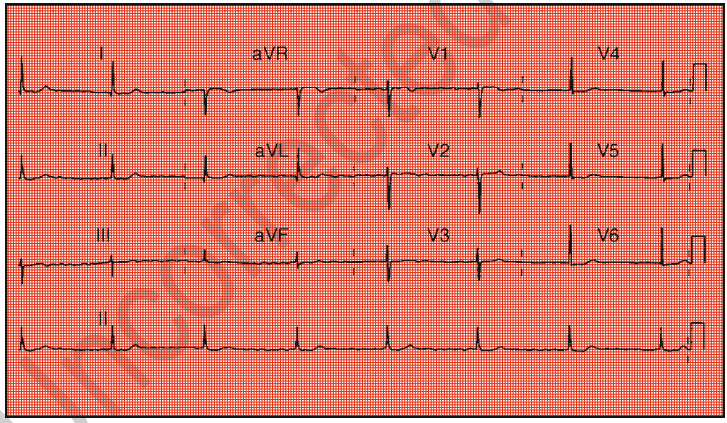


FIG. A.1. Atrial fibrillation and complete heart block, hence the regularity of the trace. The ventricular rate is 43 bpm. This exemplifies a nodal escape mechanism (Mrs. S; 30/12/99).

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DOI: 10.1007/978-1-84800-312-5_01, © Springer-Verlag London Limited 2009

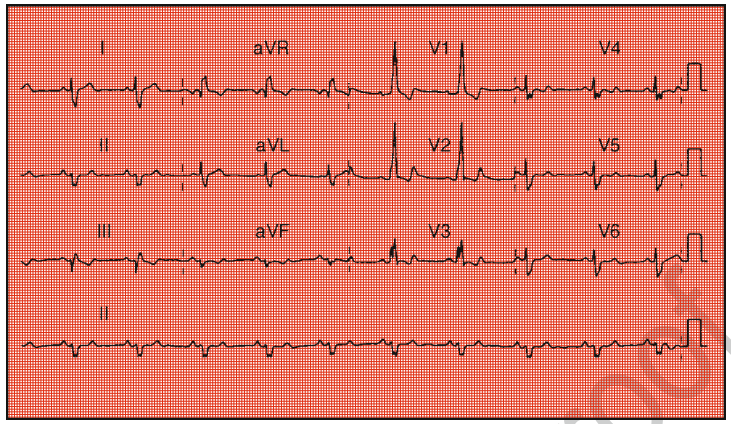

FIG. A.2. Right bundle branch block. Possible right ventricular hypertrophy (Mr. B; 8/9/98).

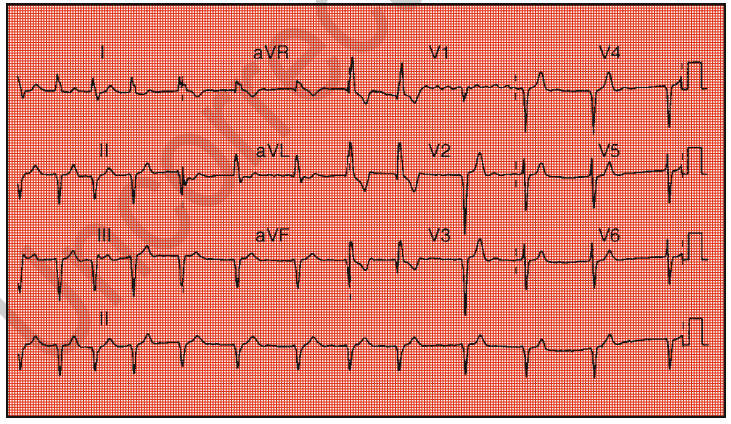

FIG. A.3. Atrial fibrillation, left axis deviation, right bundle branch block, anteroseptal and inferior Q waves. Probable inferior and anterior infarctions. (Bifascicular block) (WF; 11/8/98).

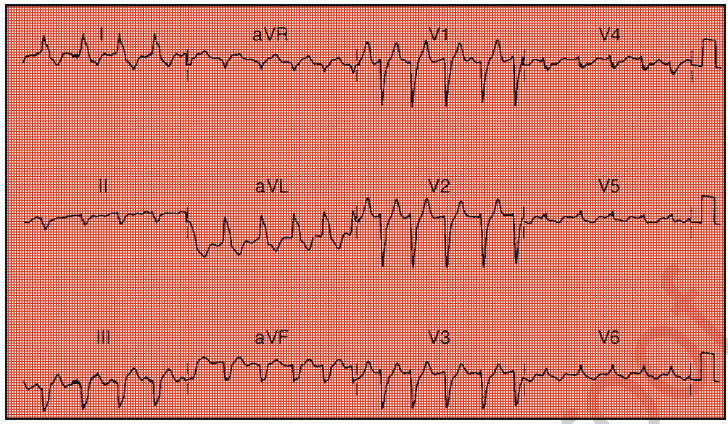

FIG. A.4 Atrial fibrillation, left bundle branch block and reduced R waves over the precordium suggestive of anterolateral damage (AR; 24/9/95).

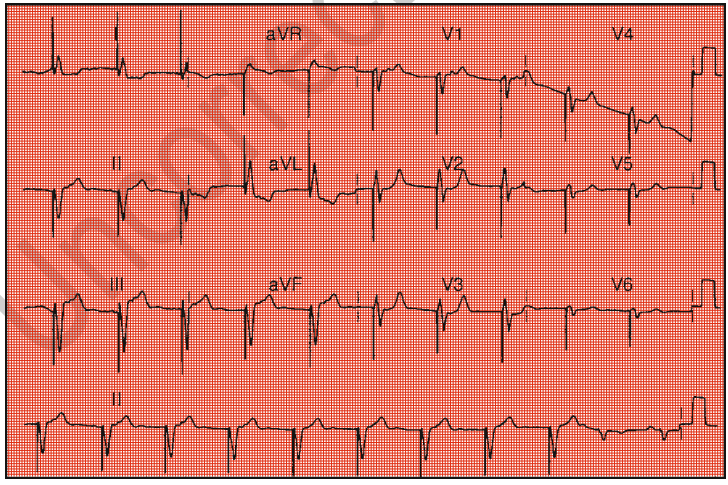

FIG. A.5. VV1 pacing. Retrograde P waves are seen in the bottom strip (AR; 26/9/95).

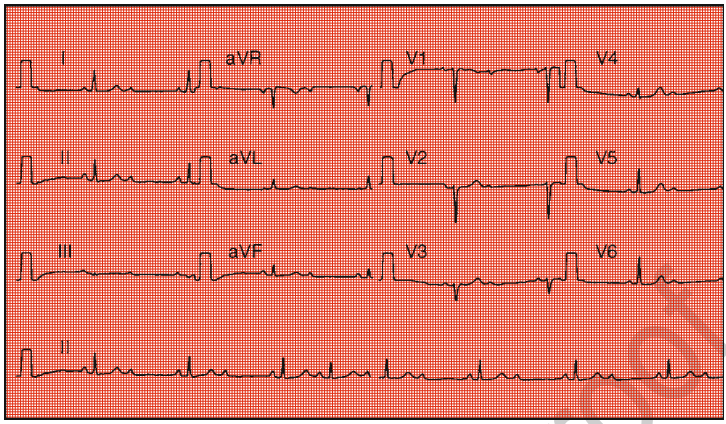

FIG. A.6. 2:1 AV block (Mobitz II). Second degree (BN; 7/2/97).

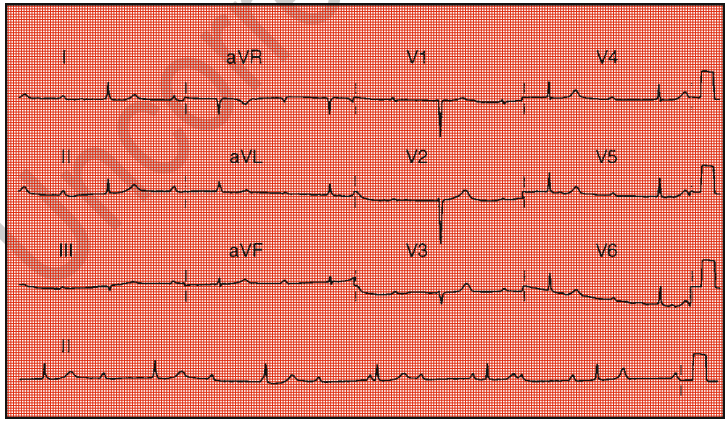

FIG. A.7. Complete heart block (third degree). AV dissociation. The P rate exceeds the QRS rate (BN; 18/2/97).

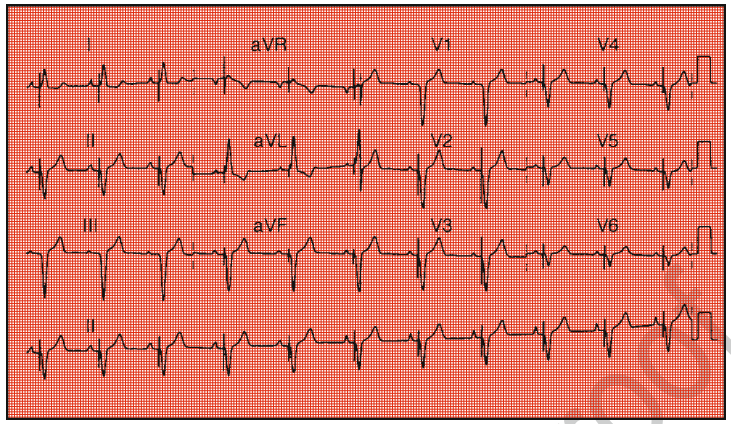

FIG. A.8. VDD pacing. Each P waves is followed by a paced signal (NB; 21/2/97).

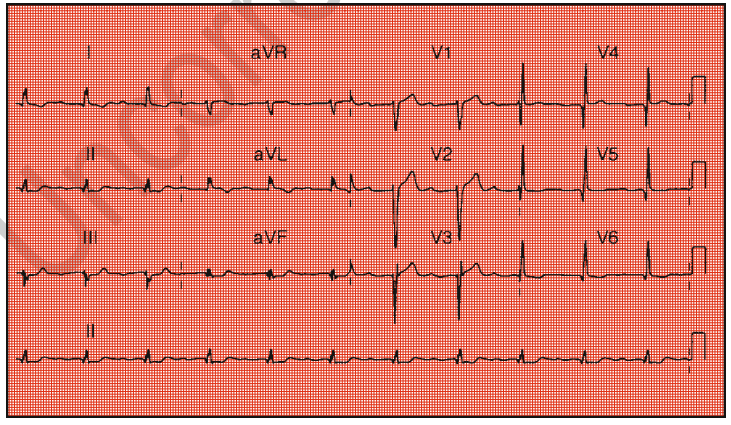

FIG. A.9. First degree AV block with anterolateral Q waves. Prolonged PR interval with anterolateral infarction (WN; 18/6/97).

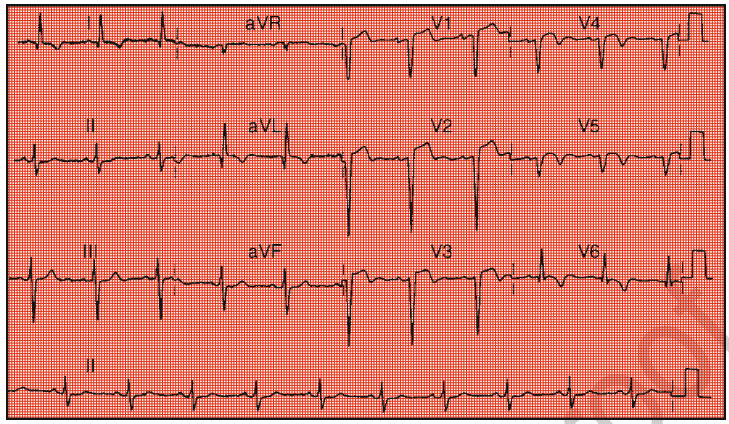

FIG. A.10. Anteroseptal Q waves. Left ventricular aneurysm is indicated by marked ST elevation in leads V1–V3 (HH; 18/11/91).

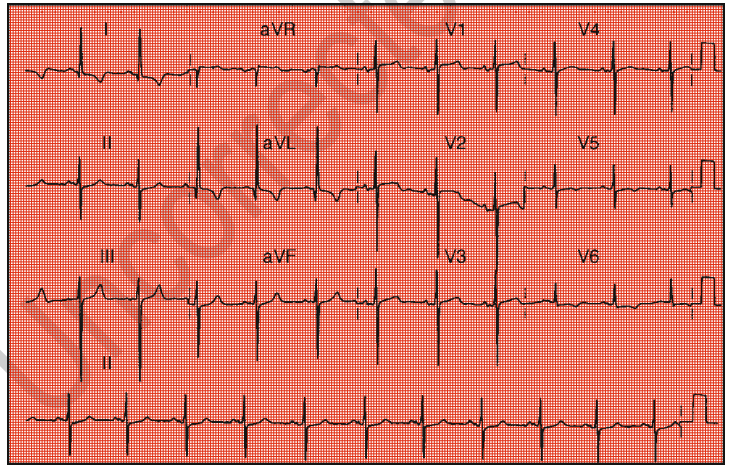

FIG. A.11. Abnormal ECG. This patient had a normal coronary arteriogram. No cause was found for the ECG abnormality which would otherwise indicate coronary artery disease or some cardiomyopathy. The patient in fact suffered from LV noncompaction. This is a rare form of congential cardiomyopathy (ME; 6/4/94).

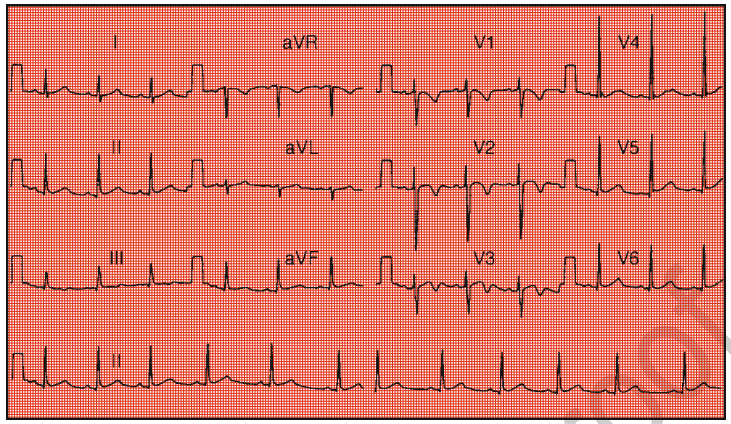

FIG. A.12. "Athlete's Heart." Well trained athletes develop ECG abnormalities that often lead to unnecessary investigations. Anteroseptal T wave inversion (PK; 26/2/96).

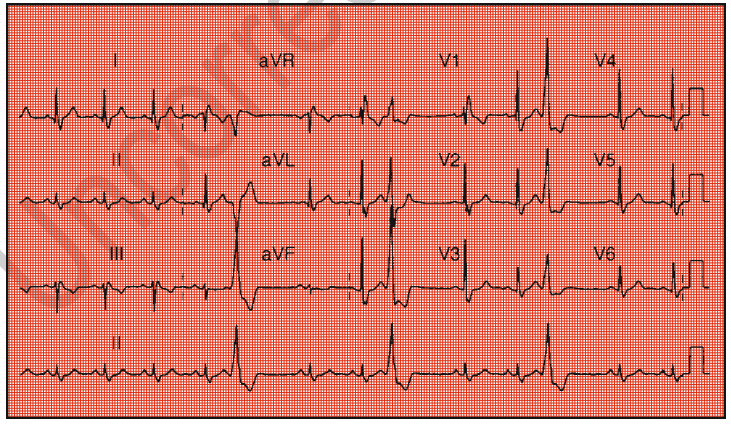

FIG. A.13. Right bundle branch block. Unifocal ventricular extrasystoles (right bundle branch pattern originating from the left ventricle) (RL; 5/1/99).

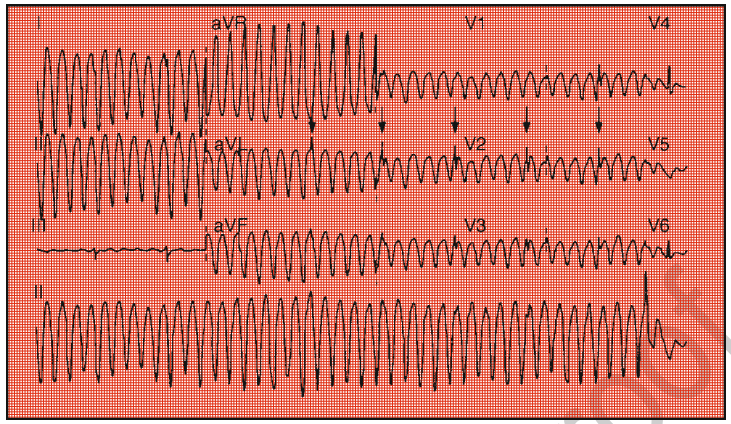

FIG. A.14. Tremor giving the appearance of ventricular tachycardia. Courtesy Dr. R Llinas and Dr. GV Henderson. Previously published in Images Clin Med 1999; 341: 1275.

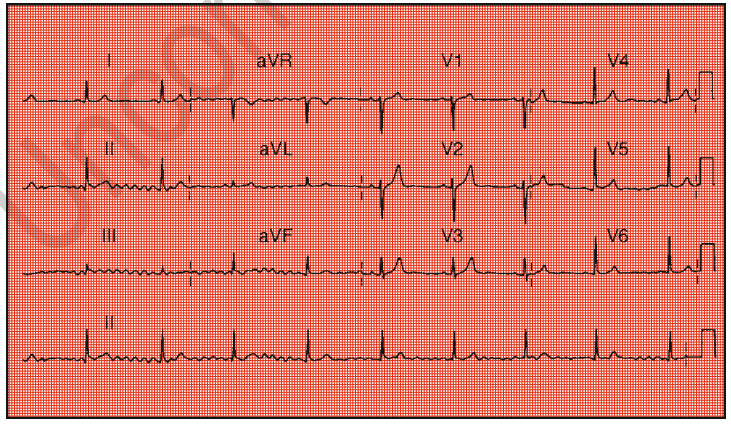

FIG. A.15. Artefact, again due to tremor (Mr. W; 20/10/99).

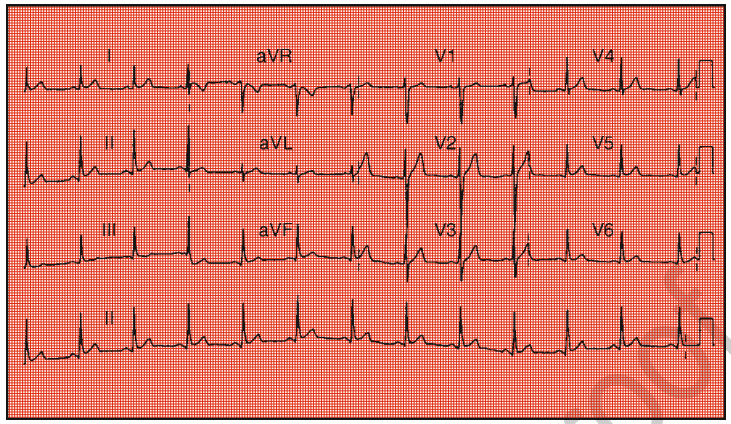

FIG. A.16. "High uptake" giving rise to ST elevation. This is a normal finding in black patients. Note LV voltages (JL; 18/10/99).

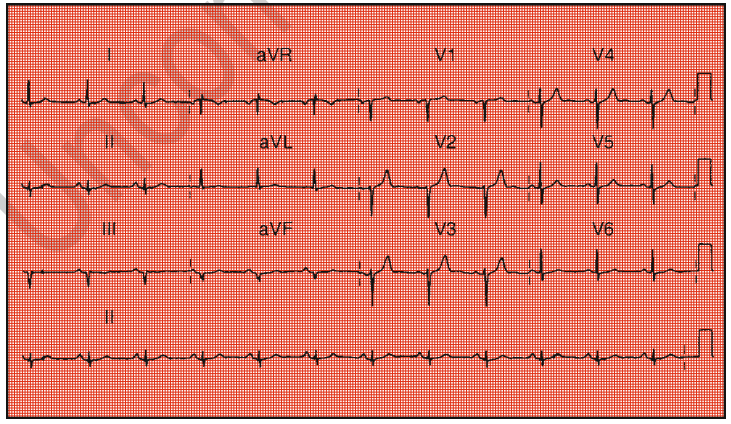

FIG. A.17. Inferior Q waves obtained in a supine patient (PC; 18/10/99).

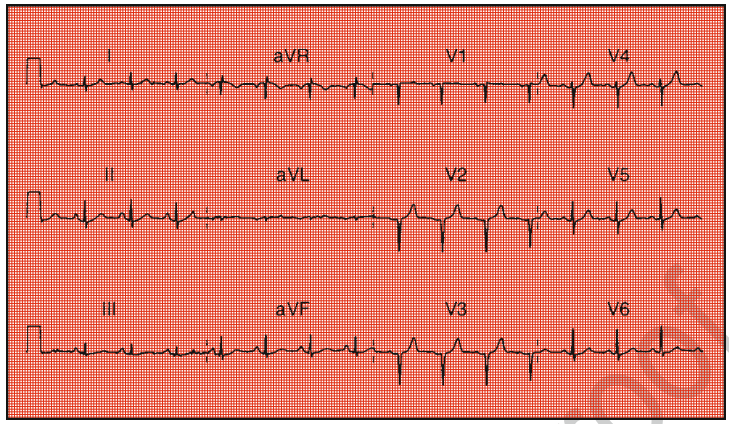

FIG. A.18. ECG from the same patient, now standing. The Q waves have disappeared. They are "positional" and not pathological (PC; 18/10/99).

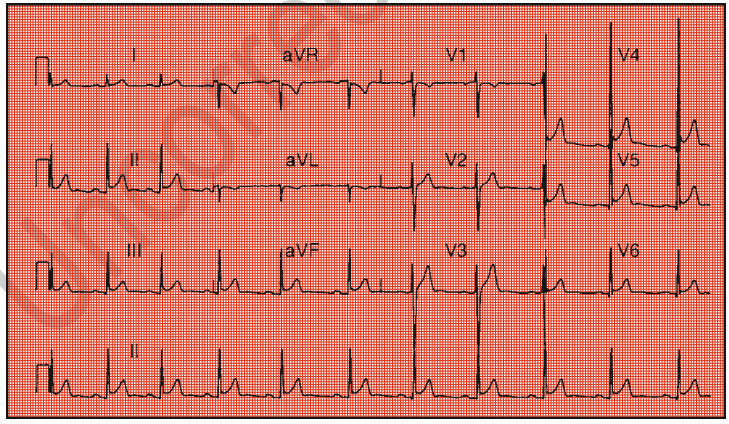

FIG. A.19. High ST uptake in a black patient. Normal ECG (GN; 18/3/99).

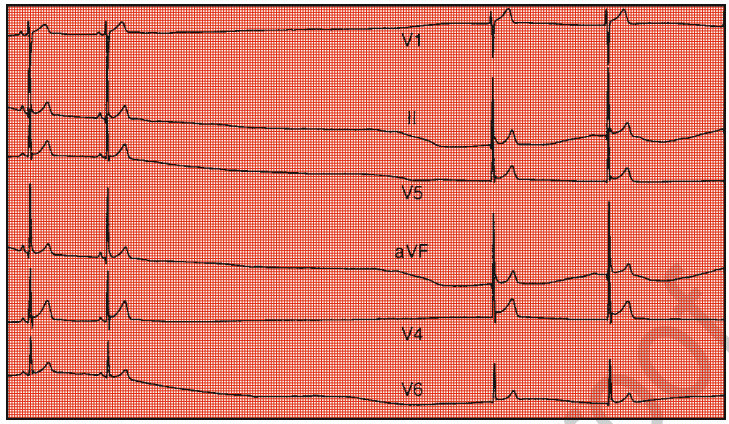

FIG. A.20. Cardiac standstill. This 14-year-old boy with syncopal attacks had a positive tilt test. He was subsequently paced (RS; 22/12/99).

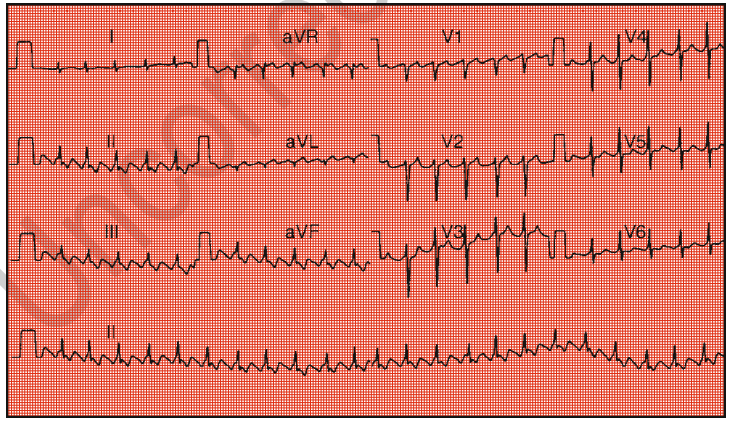

FIG. A.21. Atrial flutter (EB; 6/12/96).

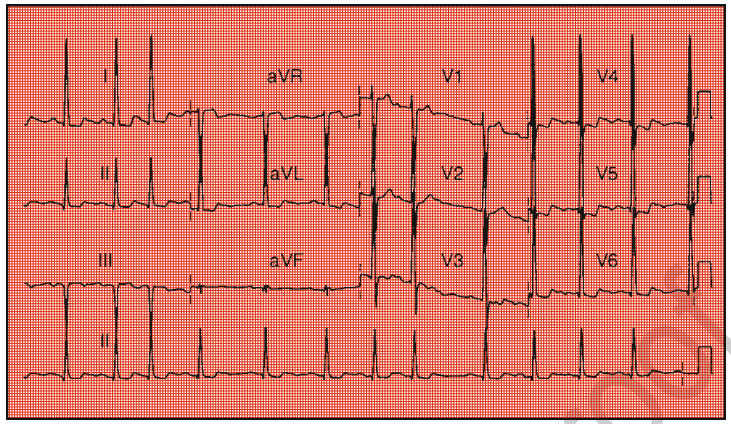

FIG. A.22. Left ventricular hypertrophy/strain. Atrial extrasystoles (severe aortic regurgitation) (FM; 18/8/99).

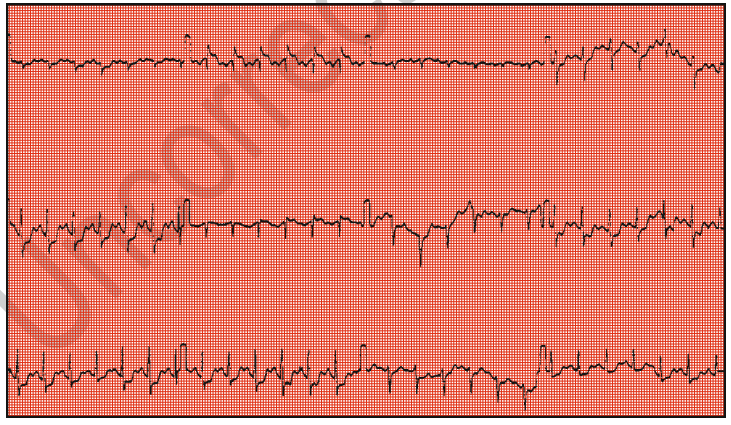

FIG. A.23. Positive stress test. Marked ST ischemic changes in inferolateral leads at heart rate of 150 bpm (EM; 19/11/93).

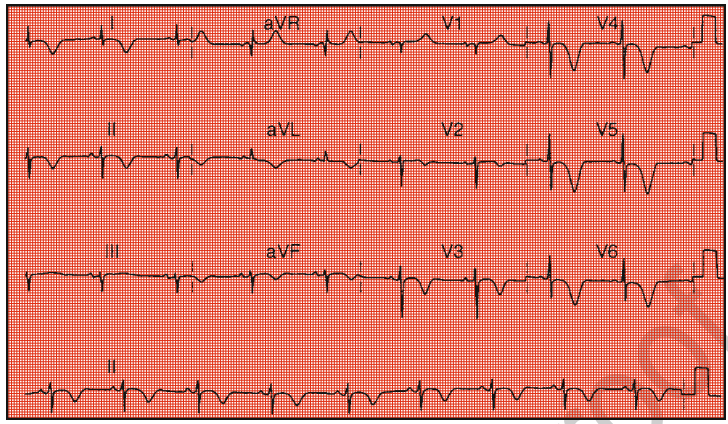

FIG. A.24. Young woman with hypertrophic cardiomyopathy and normal coronary arteries. These marked T wave inversions were not seen 1 year later on medication (EM; 19/11/93).

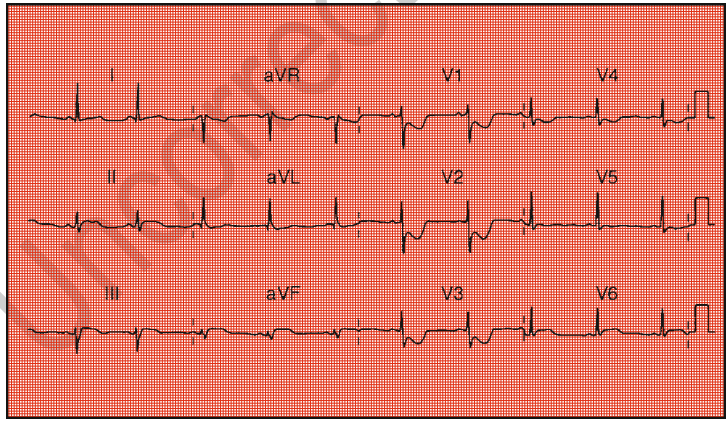

FIG. A.25. Acute subendocardial infarction. Elderly Asian woman. Non-ST elevation MI (Mrs. H; 11/7/99).

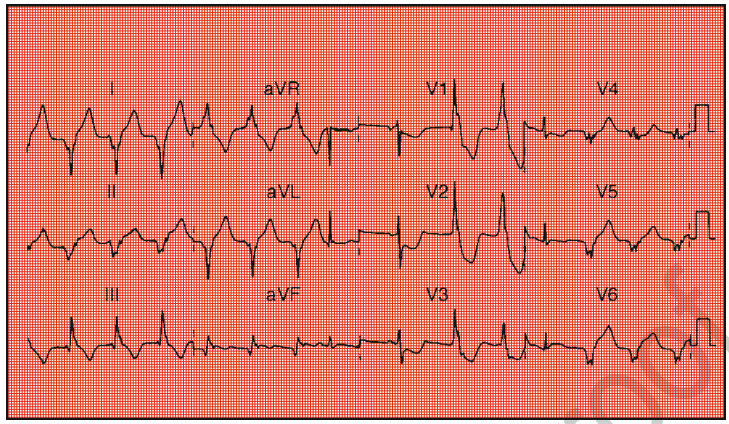

FIG. A.26. Same patient developing slow ventricular tachycardia several hours later. This does not require treatment (Mrs. H; 13/7/99).

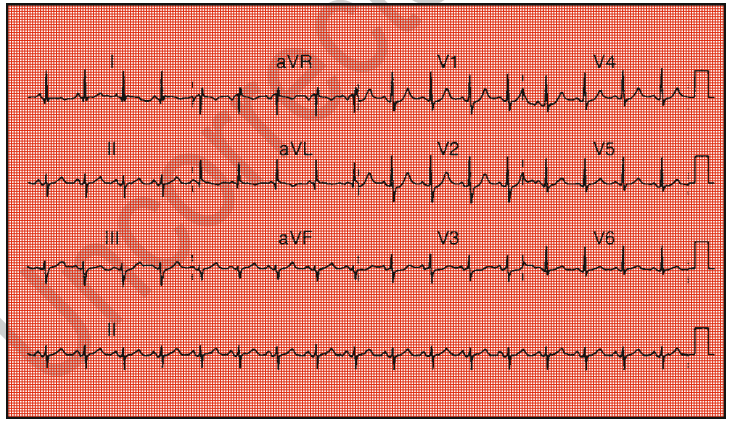

FIG. A.27. Two days later with medication, marked improvement. Dominant R wave in V1 and V2 due to true posterior infarction (Mrs. H; 13/7/99).

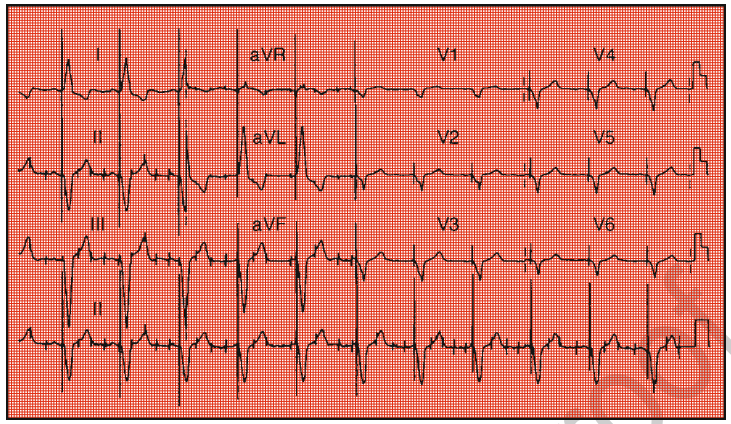

FIG. A.28. Patient with VDD pacemaker. Abnormal vertical spikes are visible in the inferior leads. These were due to inappropriate electrode contact with left leg (SP; 15/3/00).

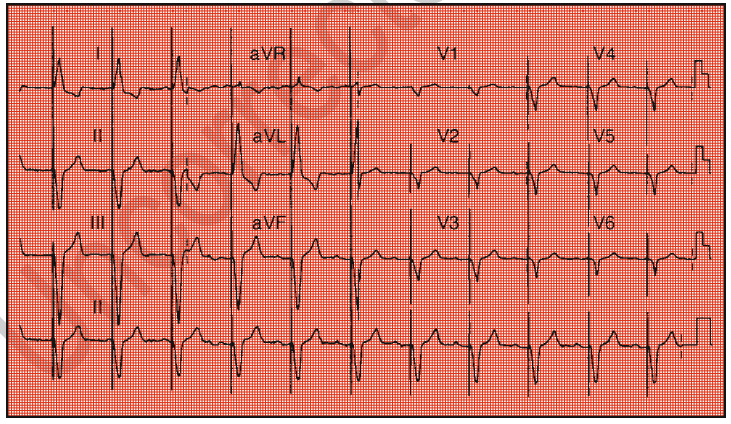

FIG. A.29. Situation rectified. Proper application of electrodes is important. Dried electrodes will cause artefacts (SP; 15/3/00).

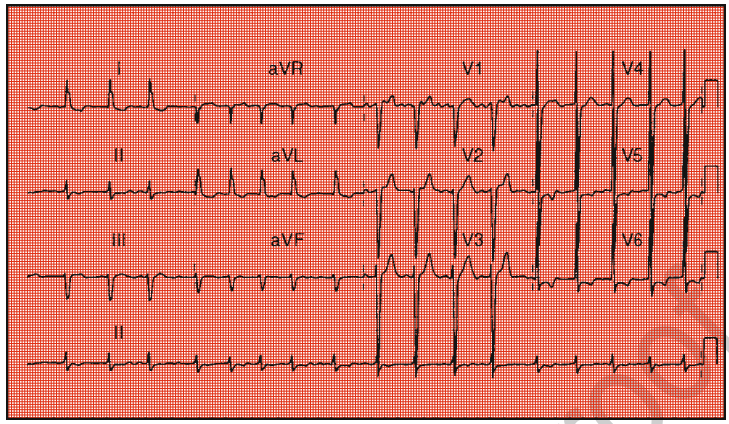

FIG. A.30. Atrial fibrillation. Left ventricular hypertrophy/strain pattern widened QRS all due to severe hypertension. (JW; 2/2/98).

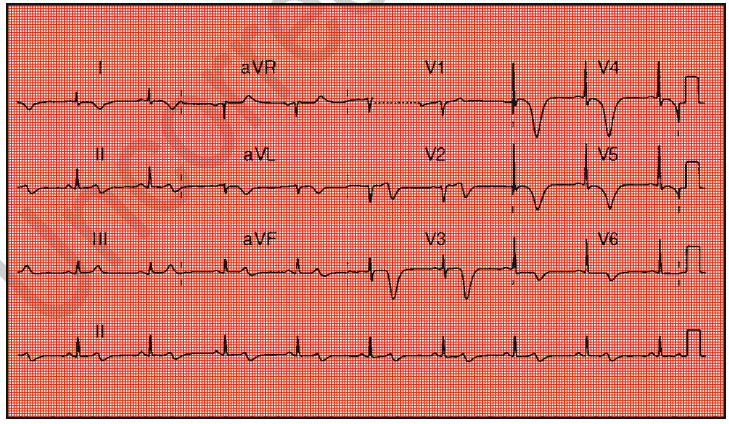

FIG. A.31. Subendocardial infarction (NSTEMI) (AS; 28/12/95).

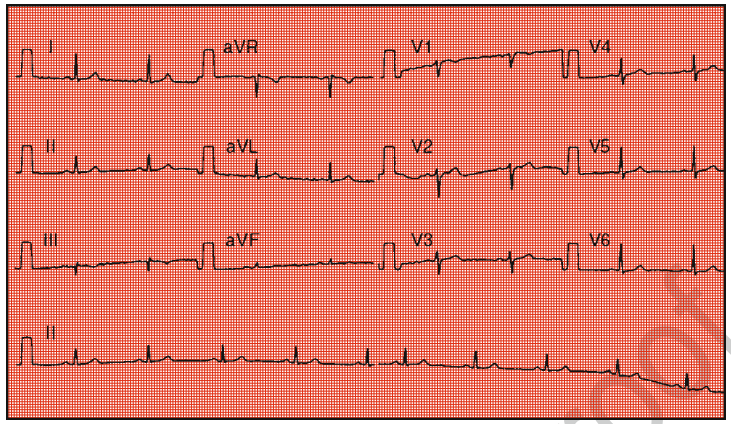

Fig A.32. Four months later good resolution is seen (AS; 22/4/96).

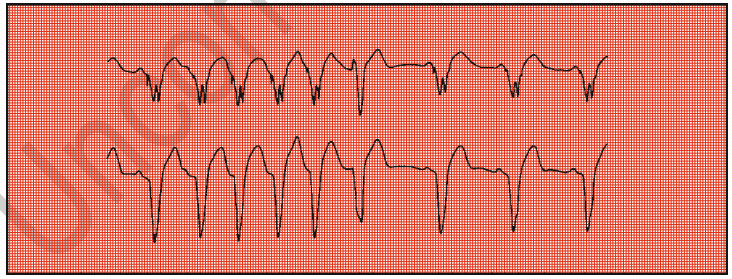

FIG. A.33. Pacemaker-induced tachycardia. The patient was aware of palpitation. There is inappropriate pacing due to incorrect sensing, giving rise to a reentry phenomenon. The situation is rectified by modifying the generator settings (LE; 1999).

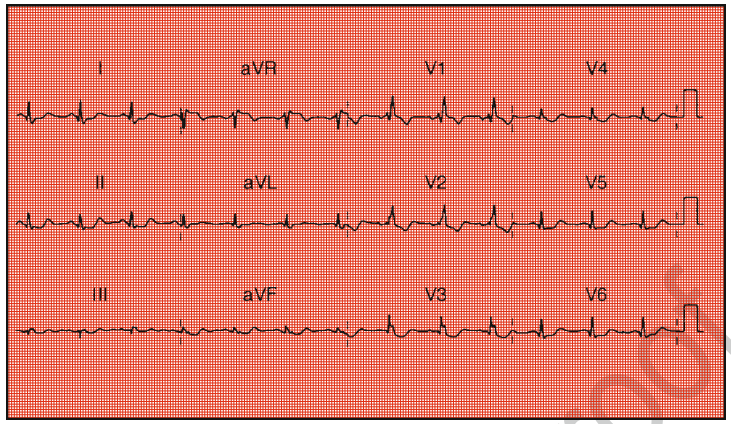

FIG. A.34. Elderly woman with previous CABG. There is normal electric axis, right bundle branch block and small anterior Q waves (MB; 8/3/00).

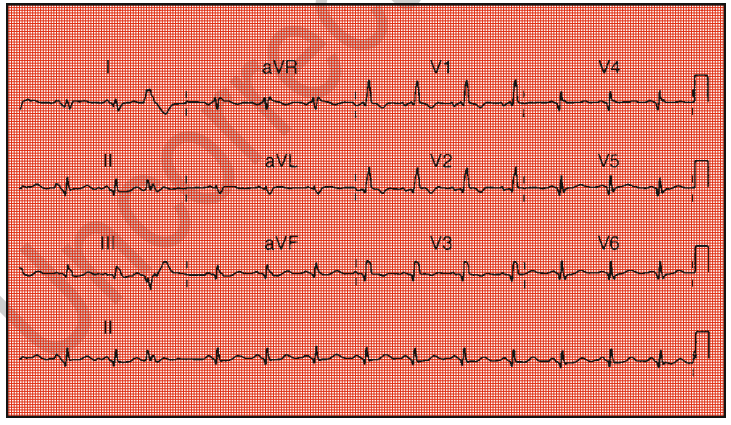

FIG. A.35. Same woman a few days later with "extension" of previous infarction. Q waves are now clearly seen in leads V2–V6 and the axis has changed indicating posterior hemi block, the new infarction having involved the conducting system (MB 13/3/00).

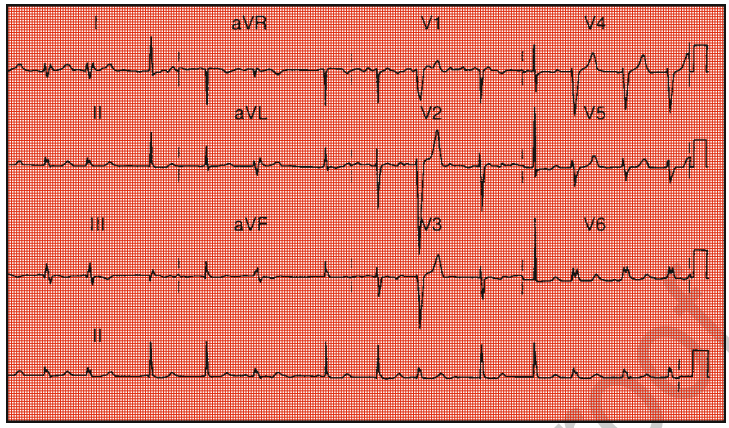

FIG. A.36. There is AF with intermittent LBBB. This is rate dependent (aberration). The RR intervals of the LBBB complexes are slightly shorter than the narrow complexes (RC; October 2000).

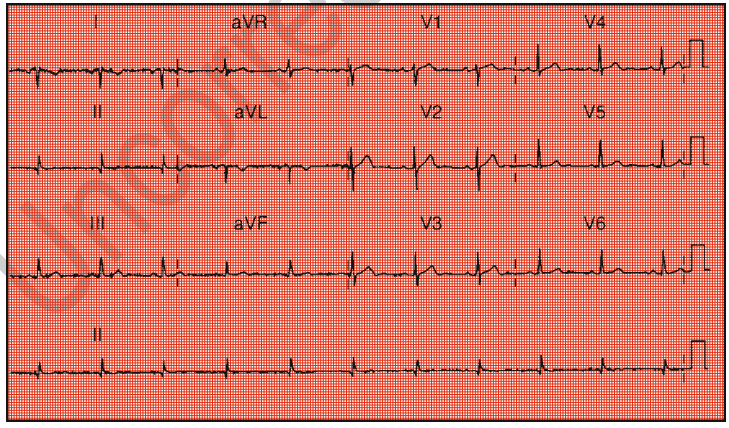

FIG. A.37. Difficult ECG to interpret. This is due to inversion of the limb leads. This can be very confusing (RV; October 2000).

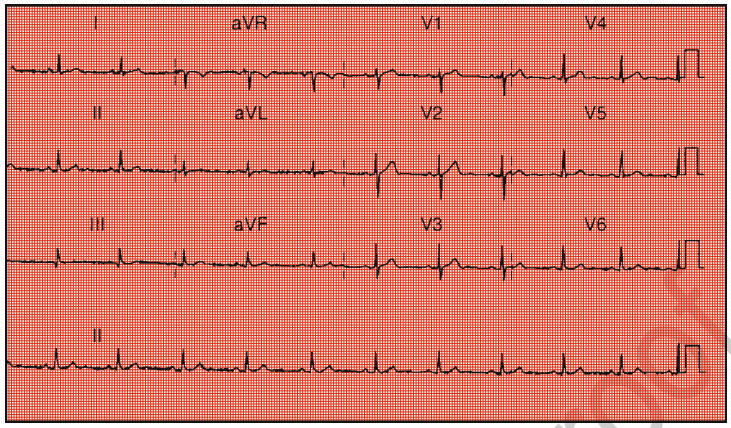

FIG. A.38. Same as ECG 349 with electrodes properly connected. This is the author's ECG which he regards as normal (RV, October 2000).

Appendix B

Appendix B

APPENDIX B ADVANCED ECG CASES

R. Vecht et al., *ECG Diagnosis in Clinical Practice*,
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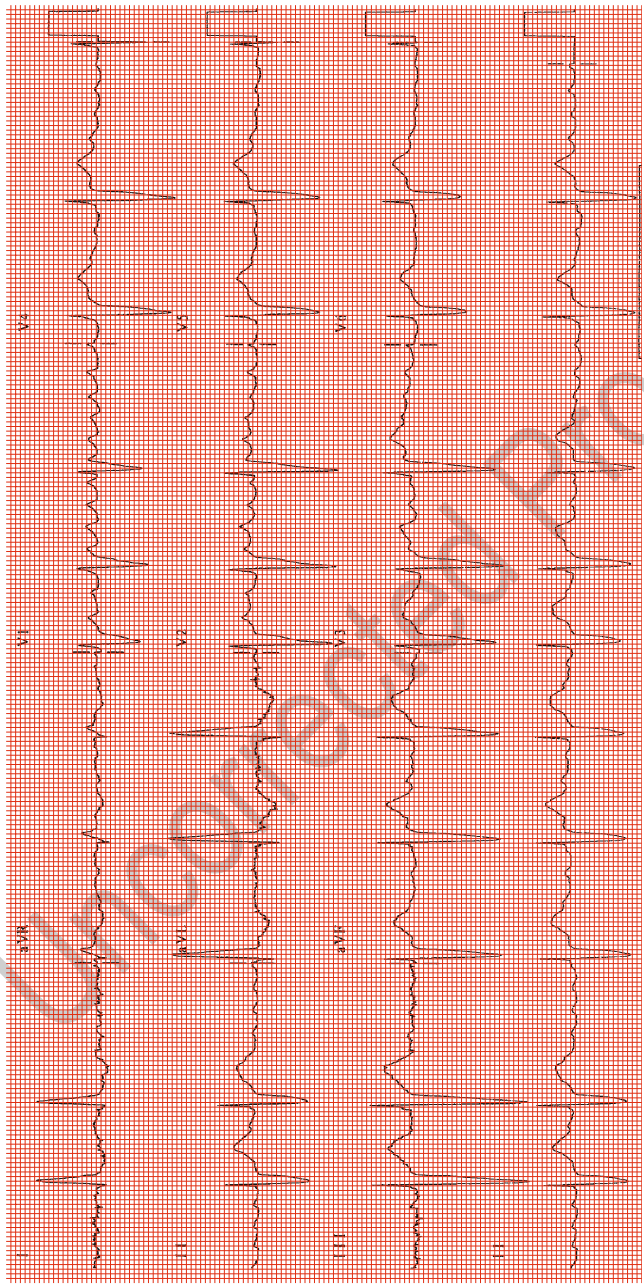


FIG. B.1. A 69-year-old male. ECG abnormal over 30 years. Recent AF. Wide QRS: LAD: Reduced R wave progression over precordial leads. Narrow Q wave in AVL. Diagnosis: LV non-compaction. This is a rare congenial cardiomyopathy consisting of "trabeculations" visible on LV imaging. The prognosis is variable but can lead to sudden death and arrhythmias (ME 2007).

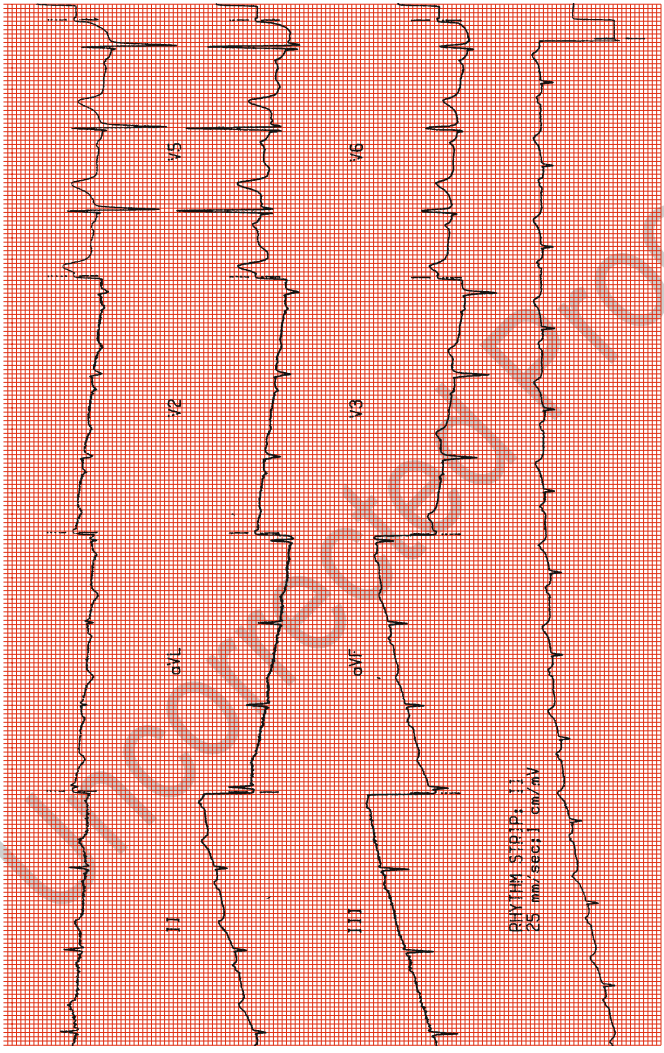


FIG. B.2. Elderly woman with COPD. "P" mitrale is well seen in lead II (MG 2007).

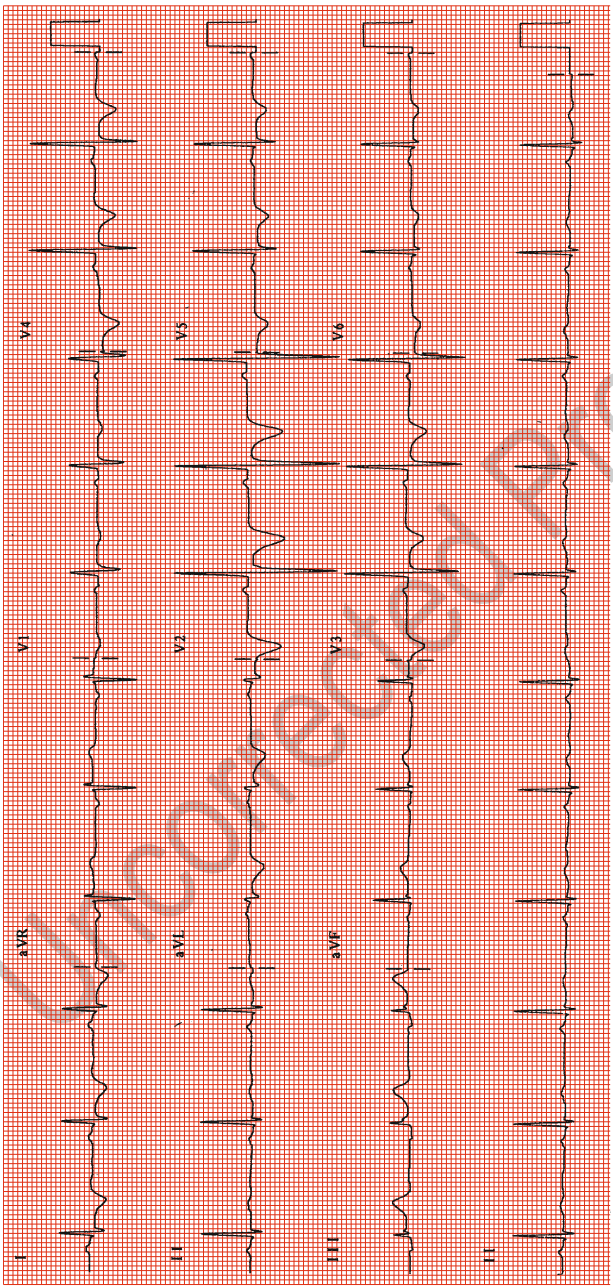


FIG. B.3. Abnormal ECG in 41-year-old male. Asymptomatic. Extensive investigations have excluded cardiac pathology. Diagnosis: Athletes heart (JP 2006).

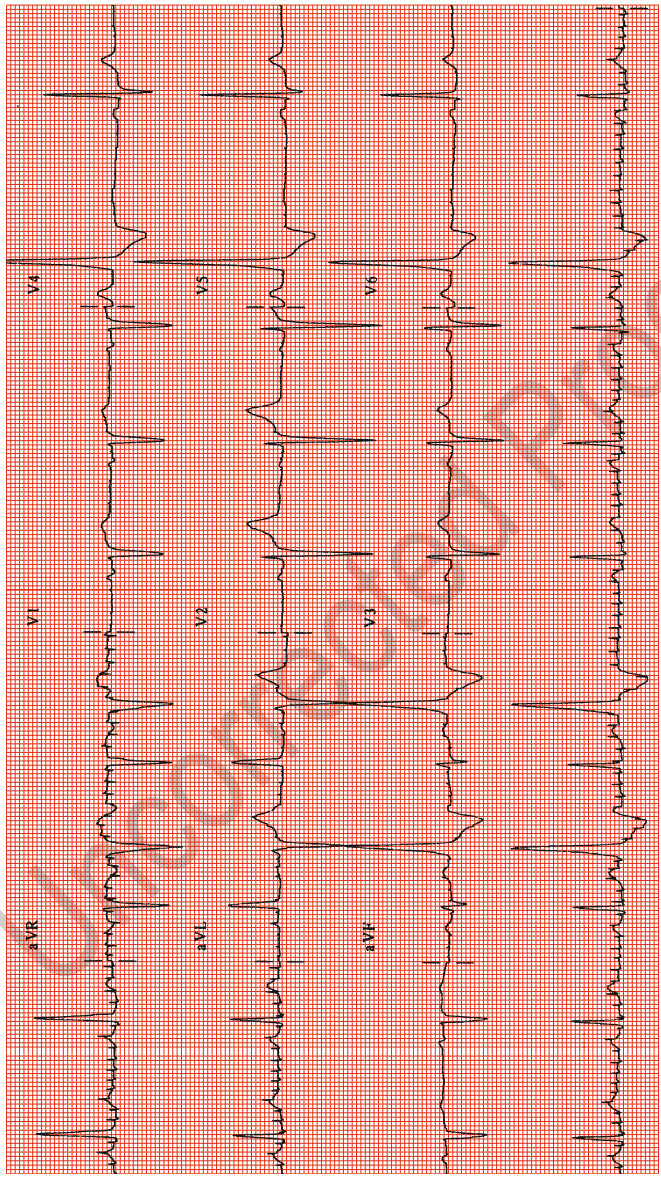


FIG. B.4. Elderly woman. Unifocal ventricular extrasystoles. Electrical interference (RG 2006).

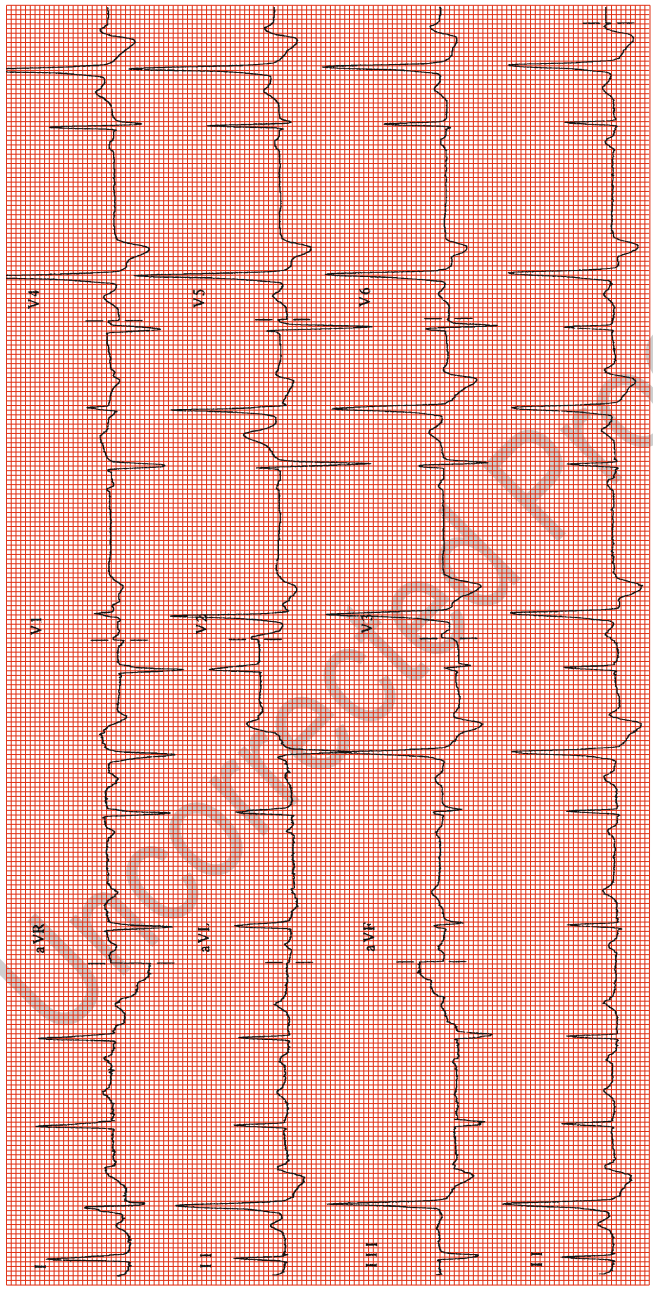


FIG. B.5 Same patient after disconnecting hearing aid. No further interference (RG 2006).

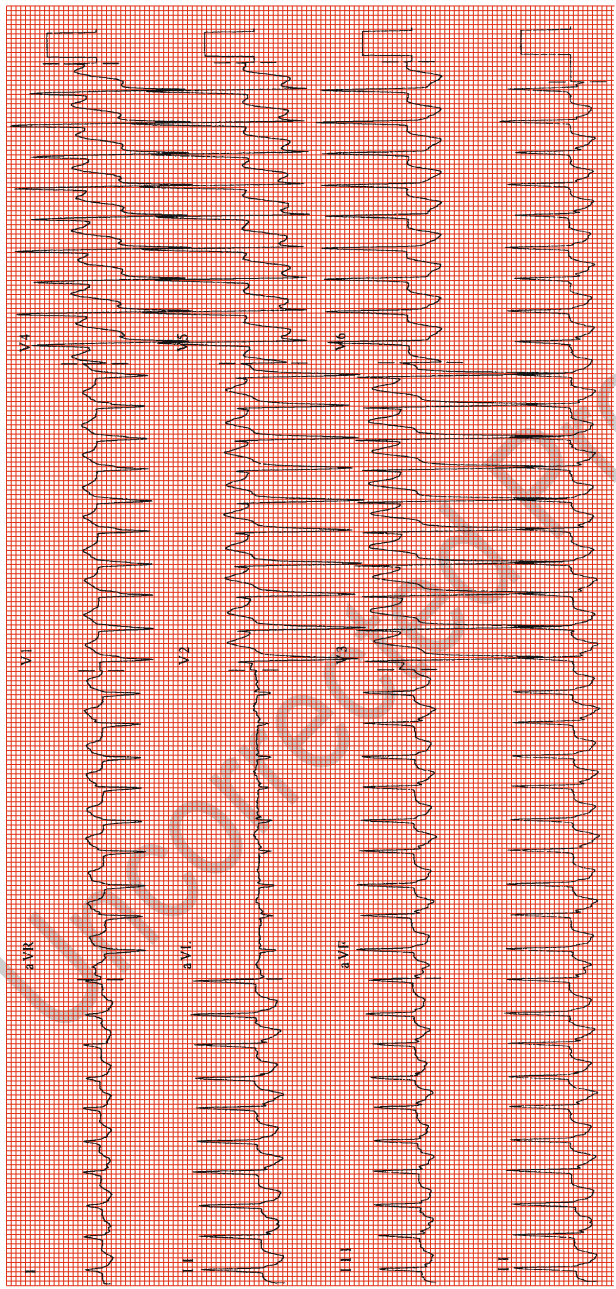


FIG. B.6. Middle aged male on digoxin. Long history of SVT. Note marked ST depressions in lateral leads. No evidence of IHD. Patient underwent curative ablation (JR 2006).

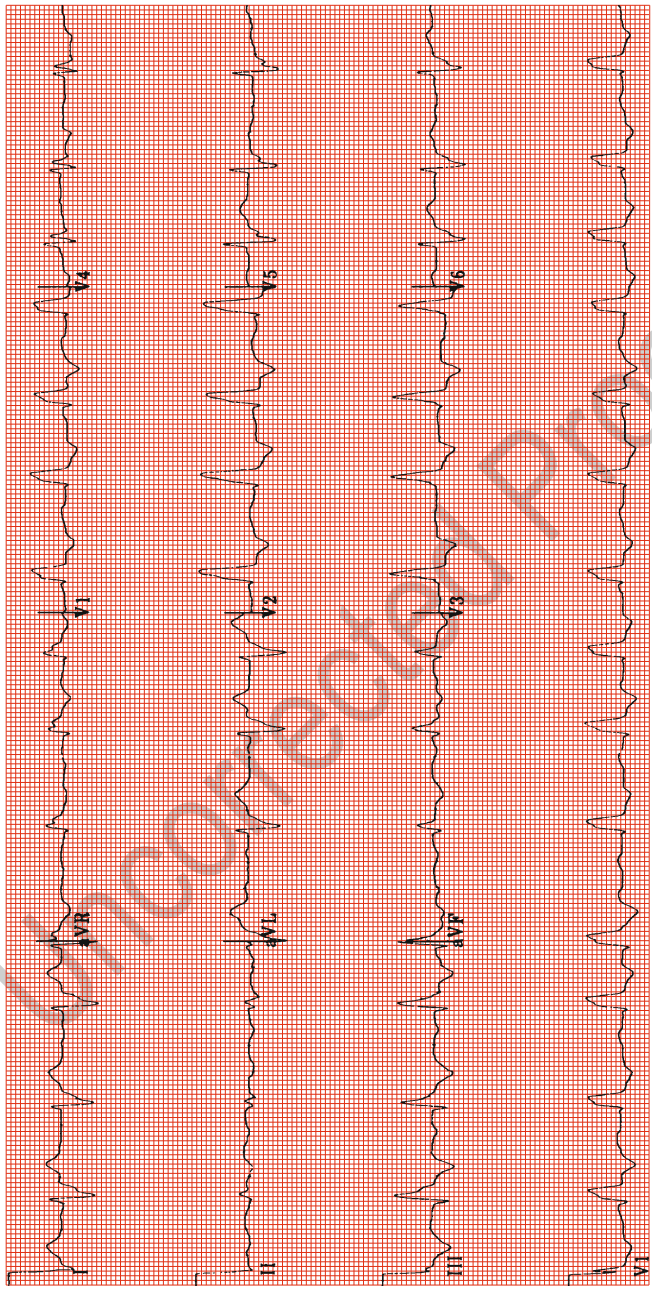


FIG. B.7. A 82-year-old male. Abnormal ECG. RAD and AF. Wide QRS and RVH. Diagnosis: Cardiac amyloidosis (HP 2003).

FIG. B.8. Echocardiogram of same patient (HP 2003).

Sinus rhythm with average heart rate of 56 bpm. There was an episode of brady/tachy (40/110 bpm) during echo. Patient however said he felt fine. Normal aortic root. The valve is tricuspid, the noncoronary cusp is thickened at the edges but opens well. Both a–v valves and pulmonary valve appear structurally normal. Dilated right atrium. Prominent RV with normal wall thickness and impaired function. LA dilated. LV dimension normal with markedly increased wall thickness and impaired function. Doppler study with CFM showed the presence of mild aortic and mitral regurgitation. Severity of MR may be underestimated in the presence of impaired LV function. Engorged IVC – 19 mm. Conclusion: Findings suggestive of cardiac amyloidosis.

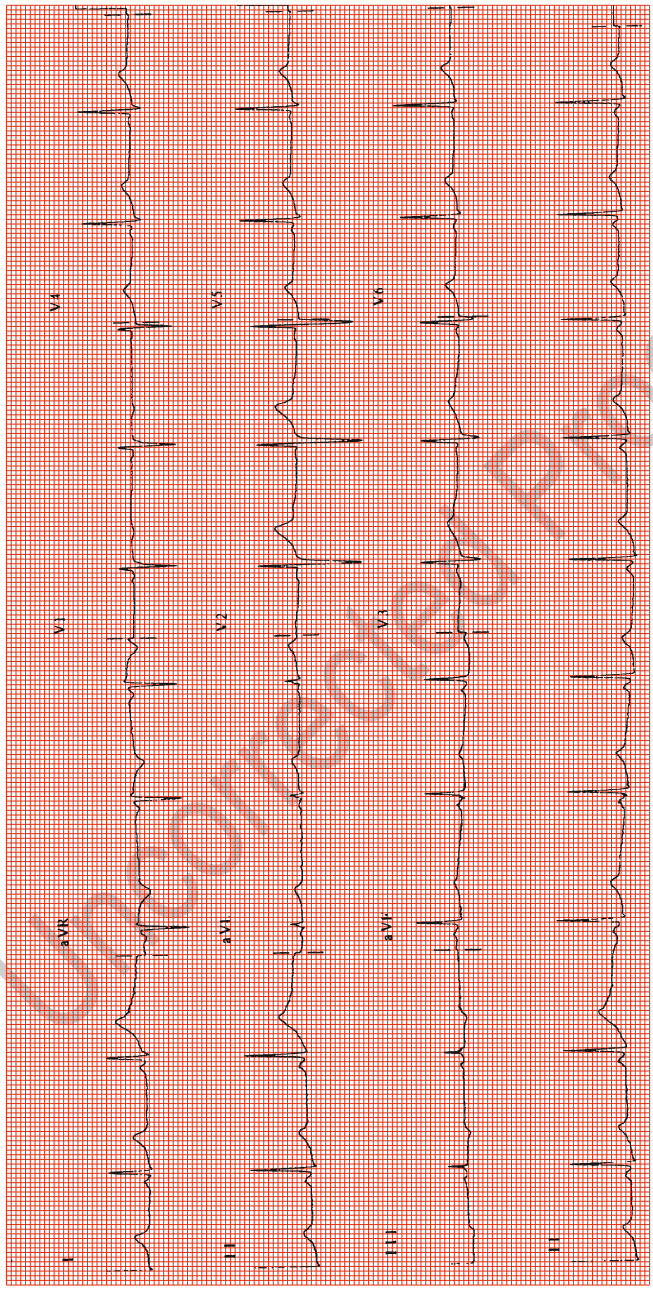


FIG. B.9. Young patient aged 20 with occasional palpitation. Short PR interval. Diagnosis: Lown Ganong Levine syndrome (AT 2000).

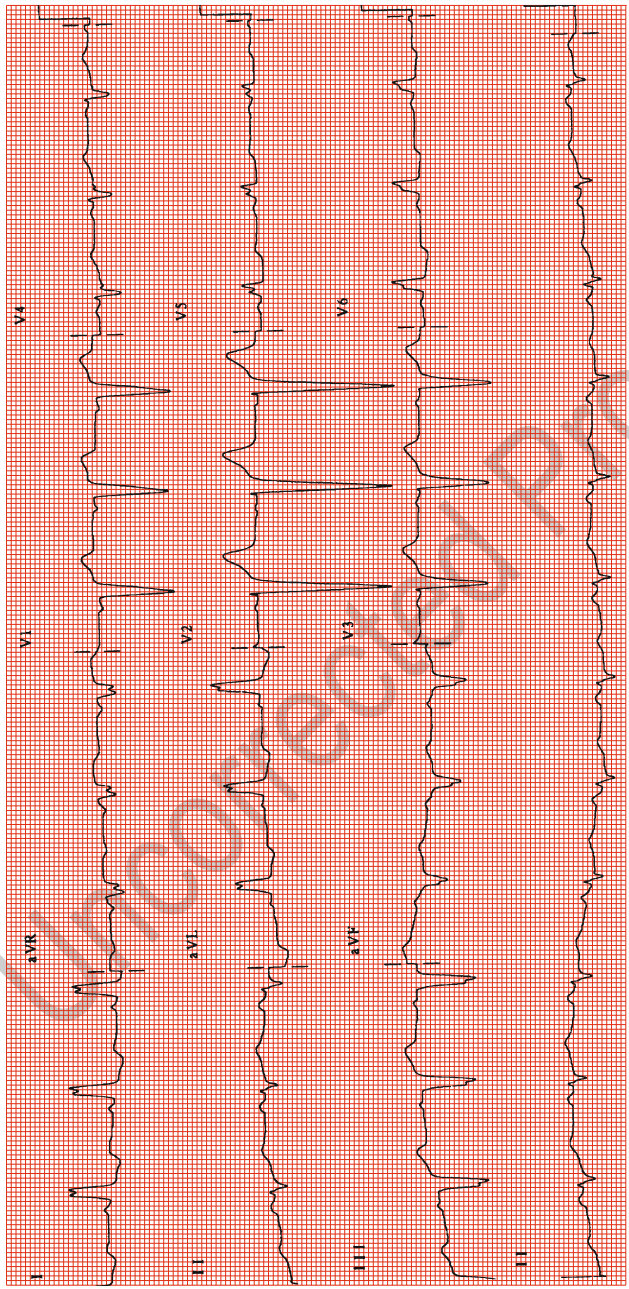


FIG. B.10. Middle aged woman. LBBB (JM 2003).

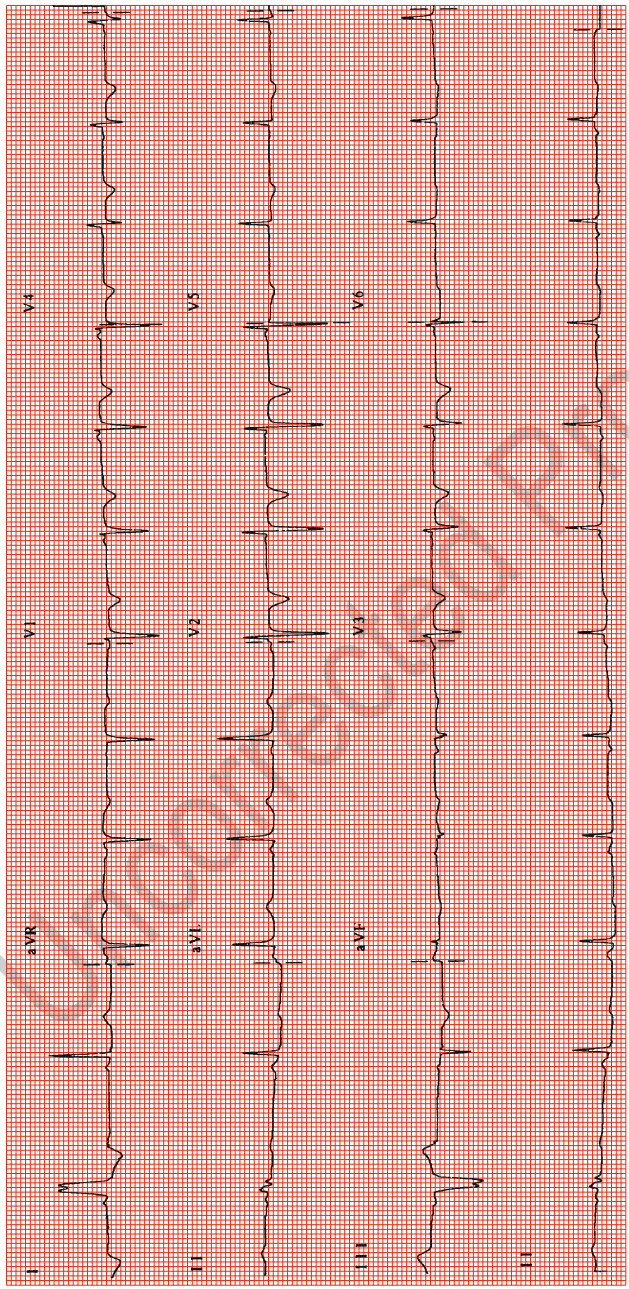


FIG. B.11. Same patient several months later: Normal conduction. Atrial extrasystoles. Anteroseptal T inversions. Diagnosis: Viral myocarditis. Recovered (JM 2003).

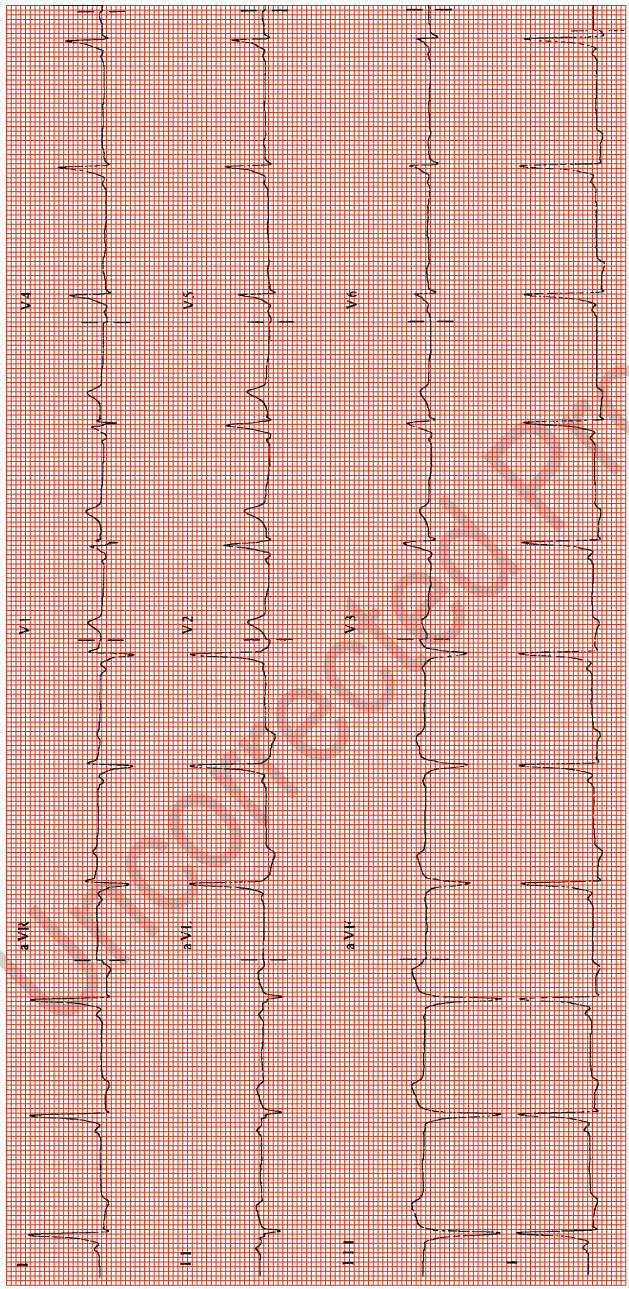


FIG. B.12. Classical WPW. Delta waves seen throughout (SJ 2004).

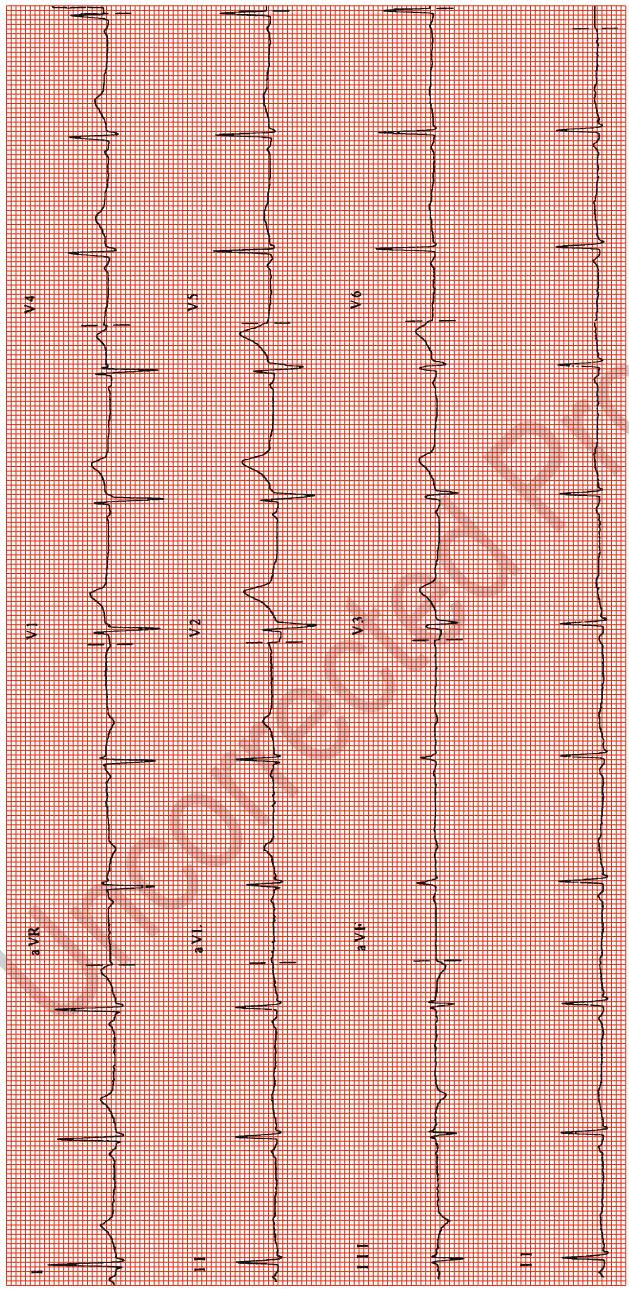


FIG. B.13. Same patient after radio-frequency ablation. Normal conduction (SJ 2004).

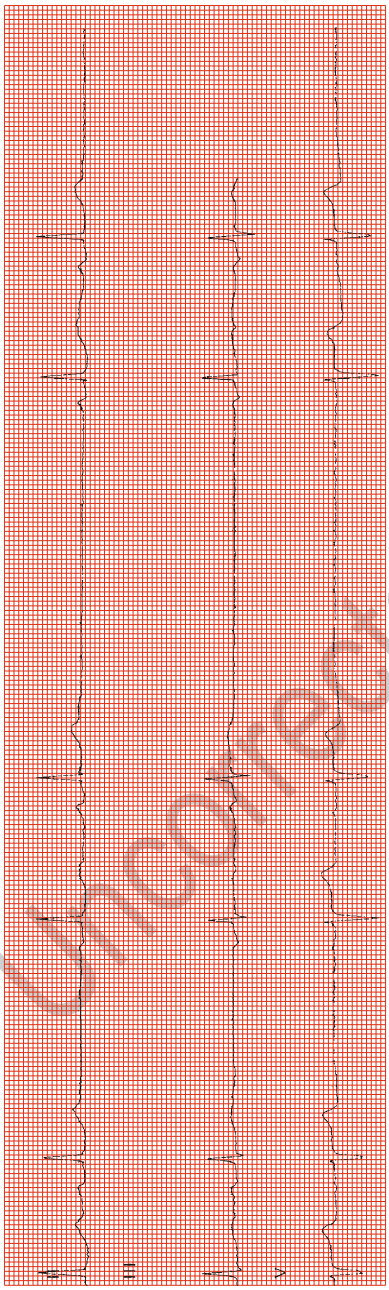


FIG. B.14. Elderly lady symptomatic. Significant SA disease. Pacemaker implanted (PD 2004).

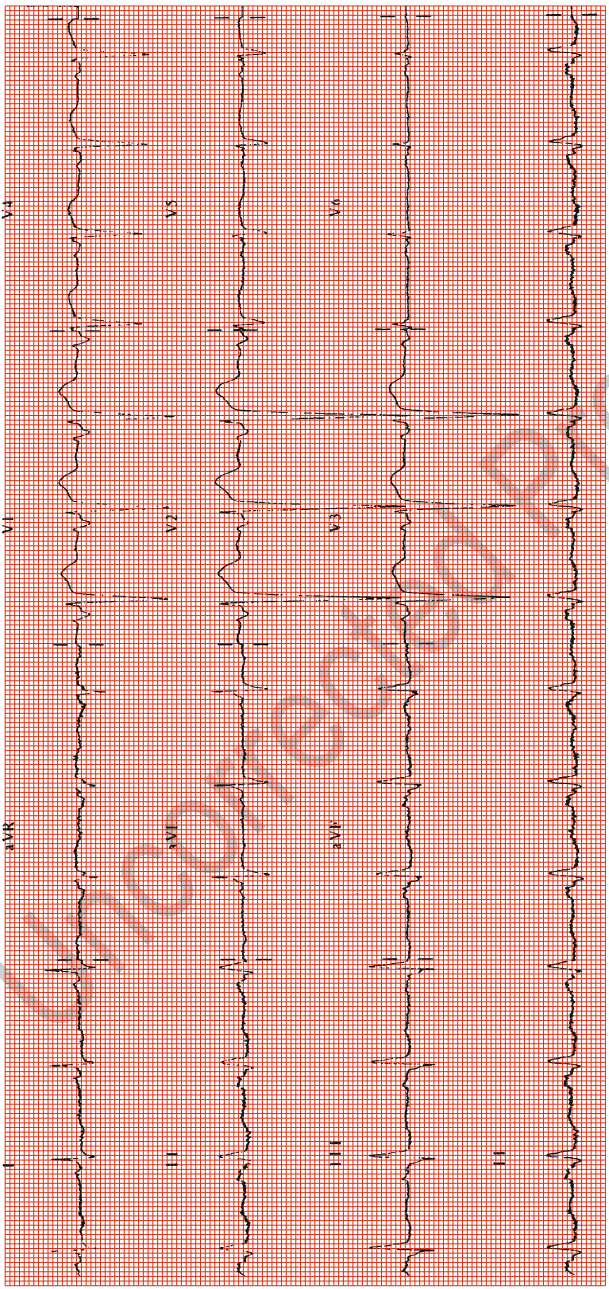


FIG. B.15. Elderly patient after CABG complaining of SOB. Marked biphasic P waves in V1. Note old inferior Q wave MI. Diagnosis: LAH.

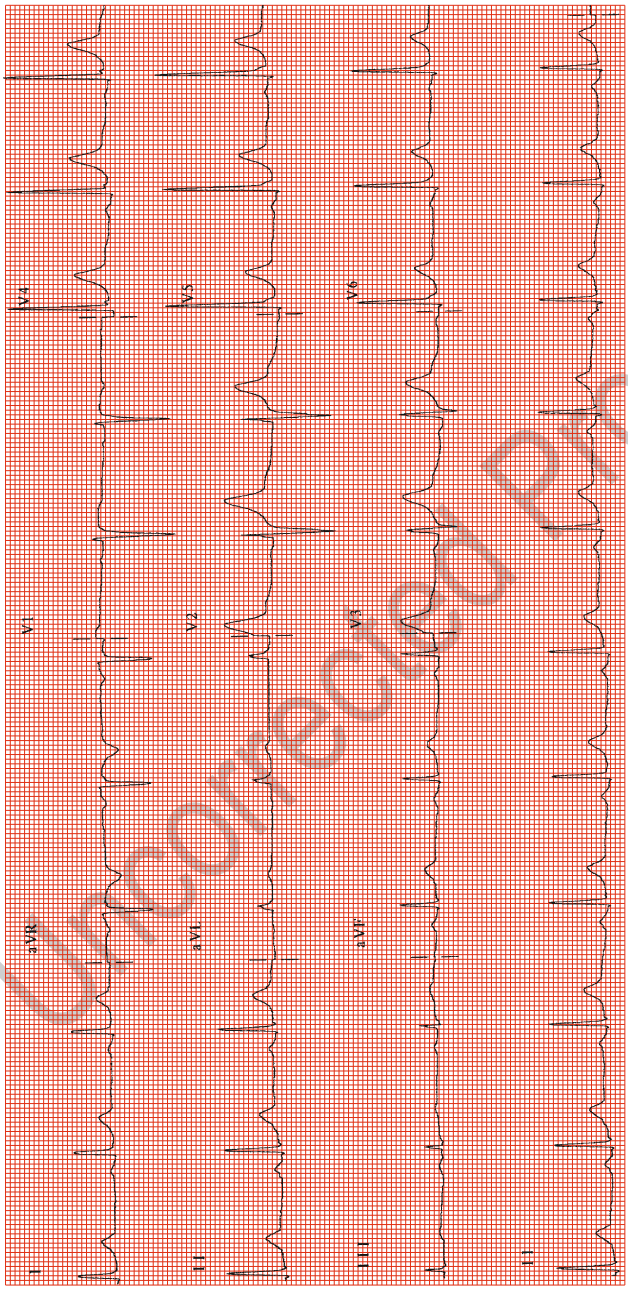


FIG. B.16. A 39-year-old male. No symptoms. The precordial leads (V4) show a small positive deflection at the bottom end of the QRS complexes. This is due to a small epicardial portion of the left ventricle being activated late (TA 2002).

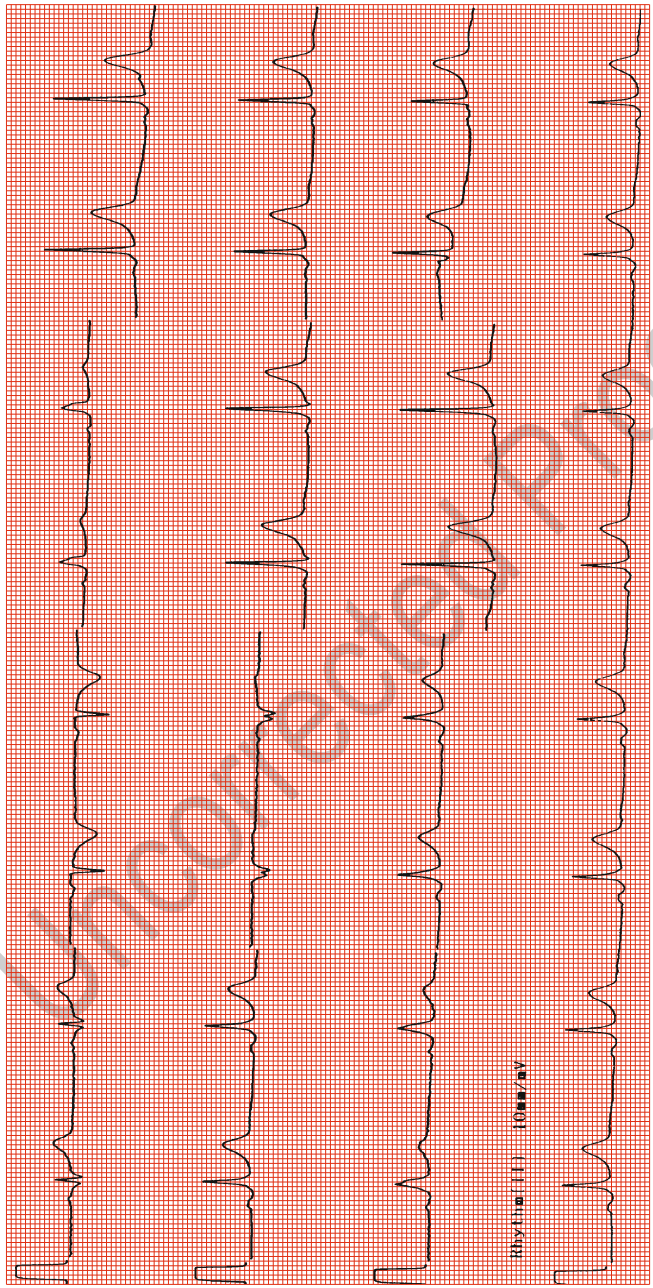


FIG. B.17. A 60-year-old asymptomatic Japanese male. ECG shows pre-excitation of left lateral accessory pathway. The degree of pre-excitation is small because the accessory pathway is at some distance from the sinus node (TM 2002).

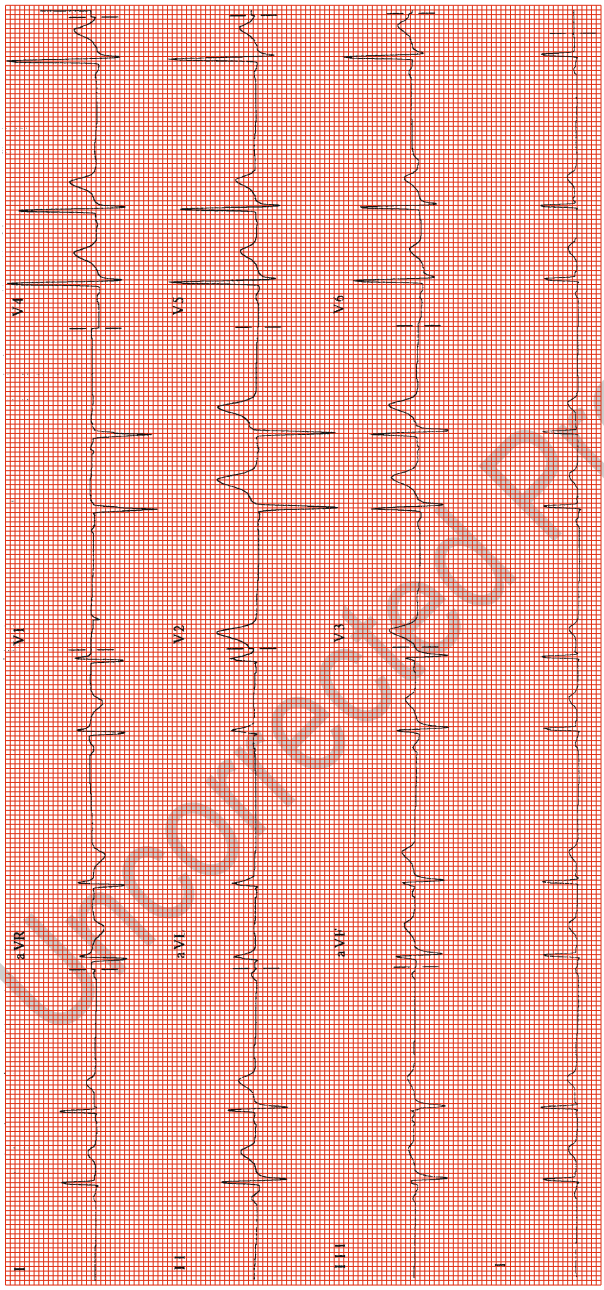


FIG. B.18. Diagnosis: Atrial bigeminy. Middle aged man with mild ischemic heart disease (AB 1989).

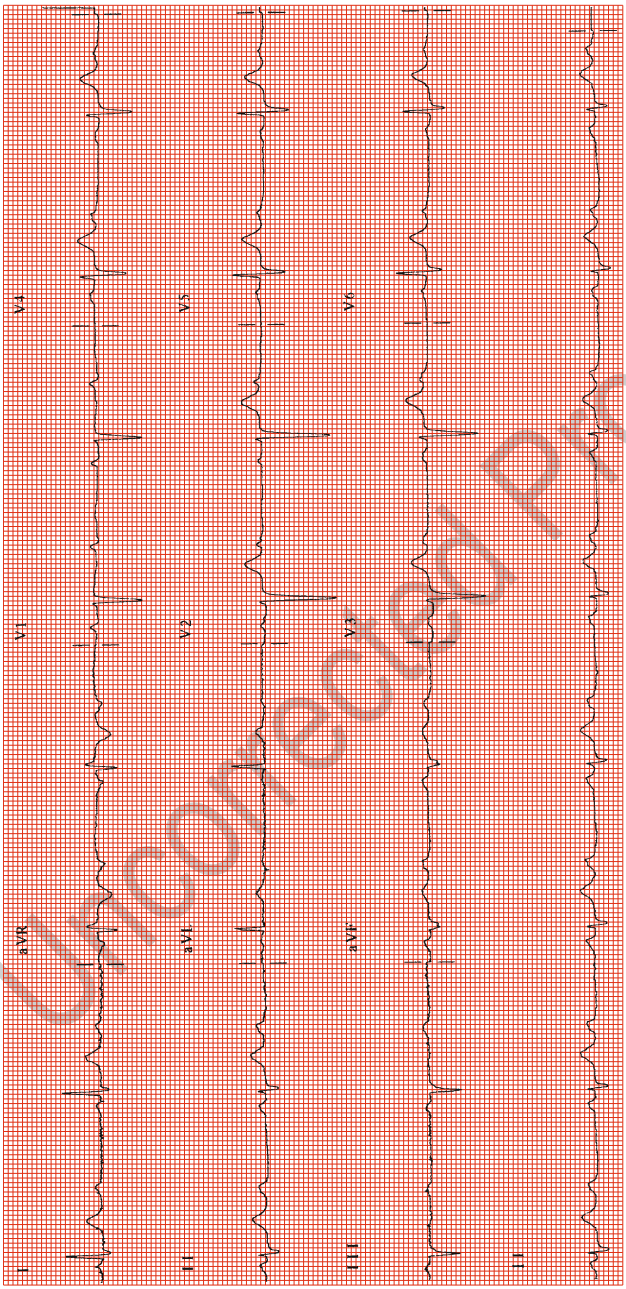


FIG. B.19. Young male showing second degree HB (Wenckebach 2). Diagnosis: Idiopathic dilatation RV (BC 2002).

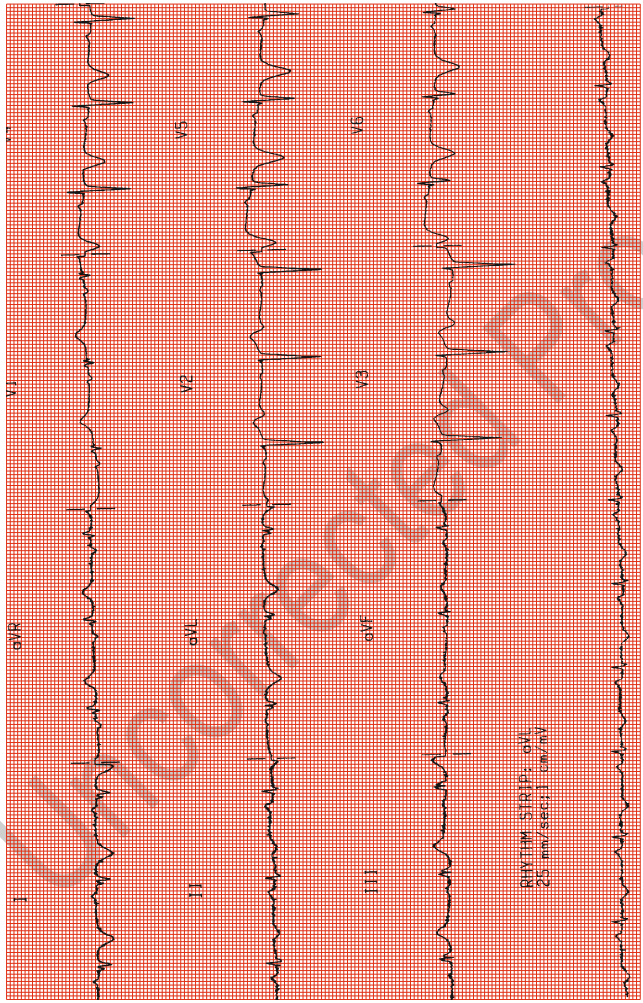


FIG. B.20. Elderly woman with non-ST elevation anterolateral MI. Early intervention (stent to LAD) (RE 1996).

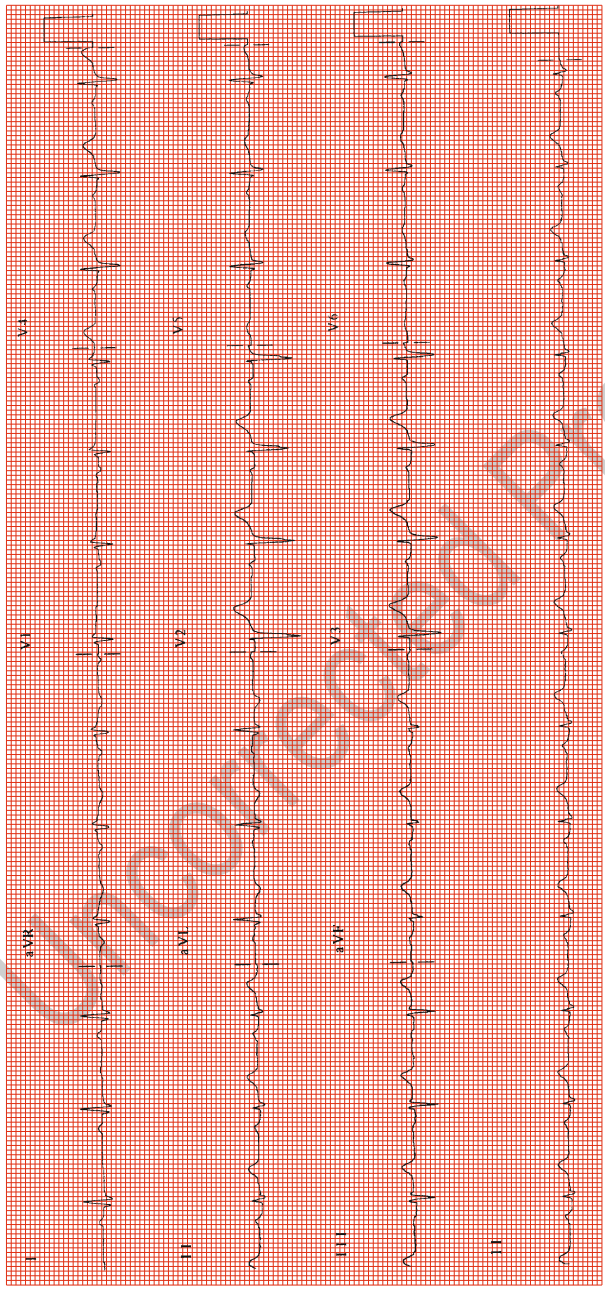


FIG. B.21. Same patient 5 years later – good recovery. Residual Q wave in I and AVL with T inversions (RE 2001).

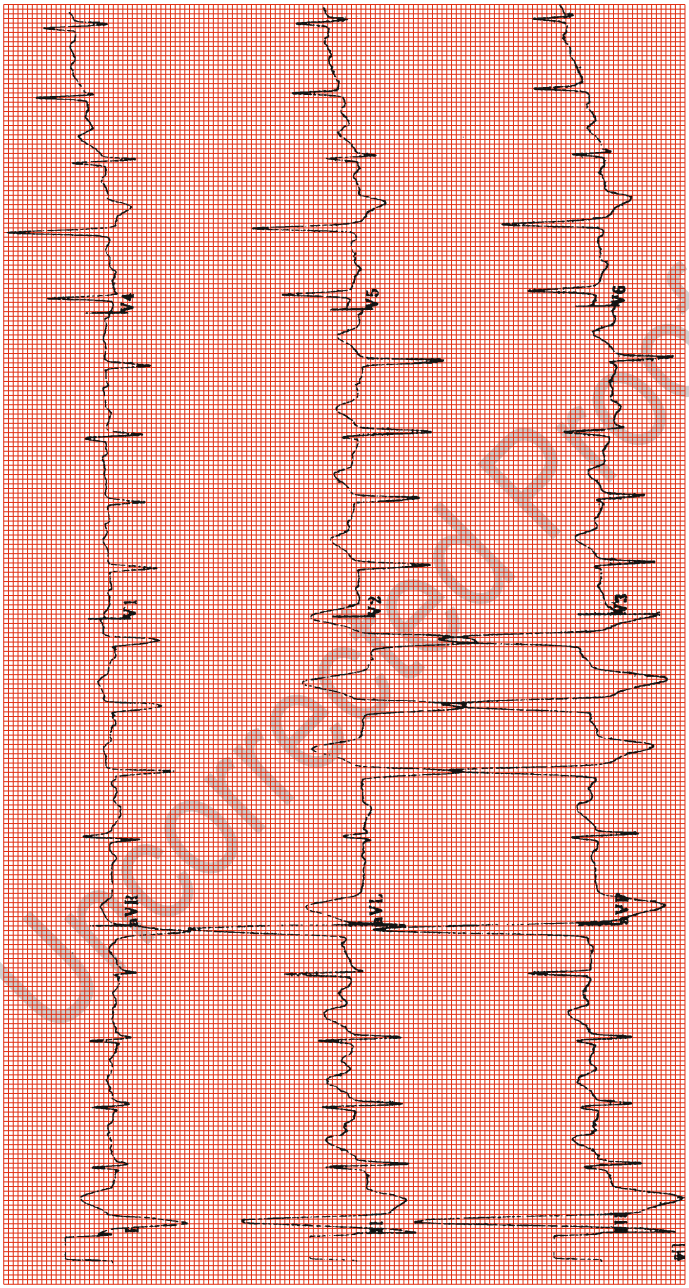


FIG. B.22. A 39-year-old man asympatomatic, HT. Positive FH. Routine ECG. Run of VT, AVR, AVL, AVF (SC 2001).

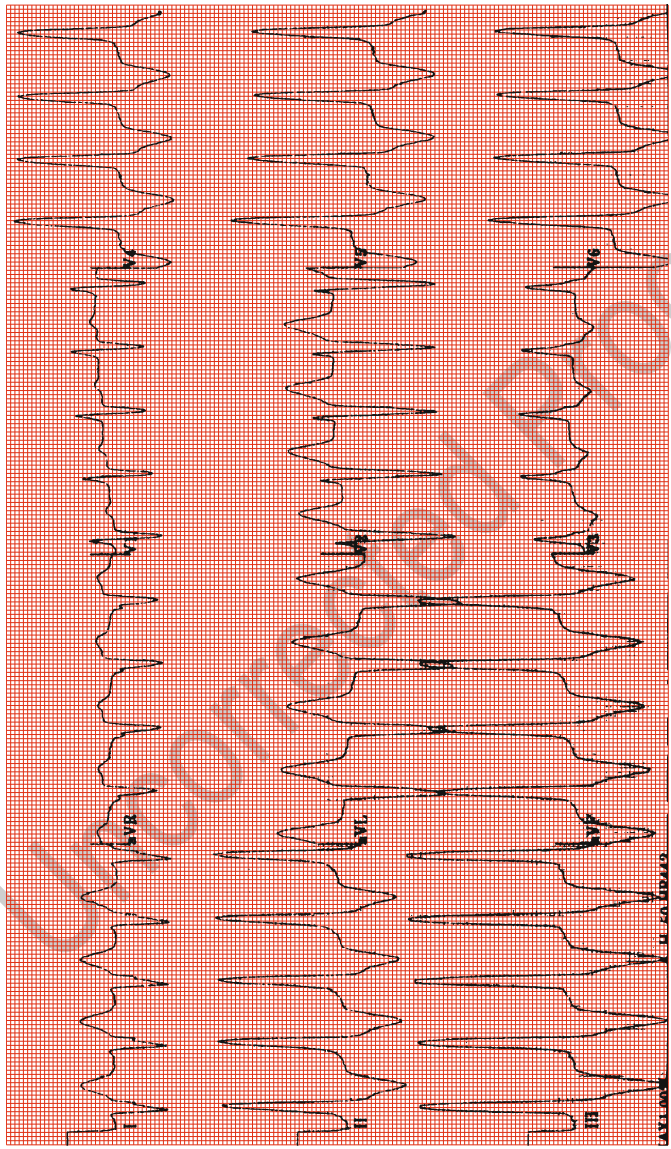


FIG. B.23. Same patient 1 min earlier during VT. Spontaneous recovery (SC 2001).

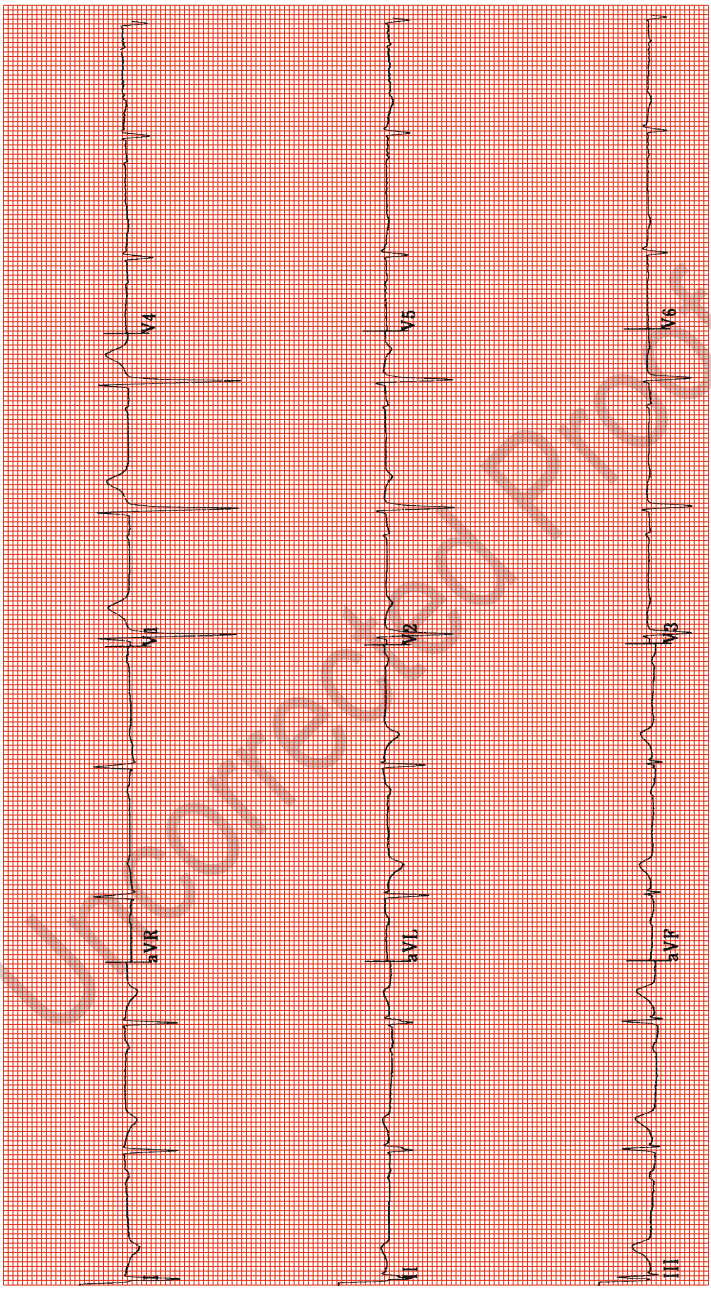


FIG. B.24. Very abnormal trace. This is a routine ECG (DA 2003).

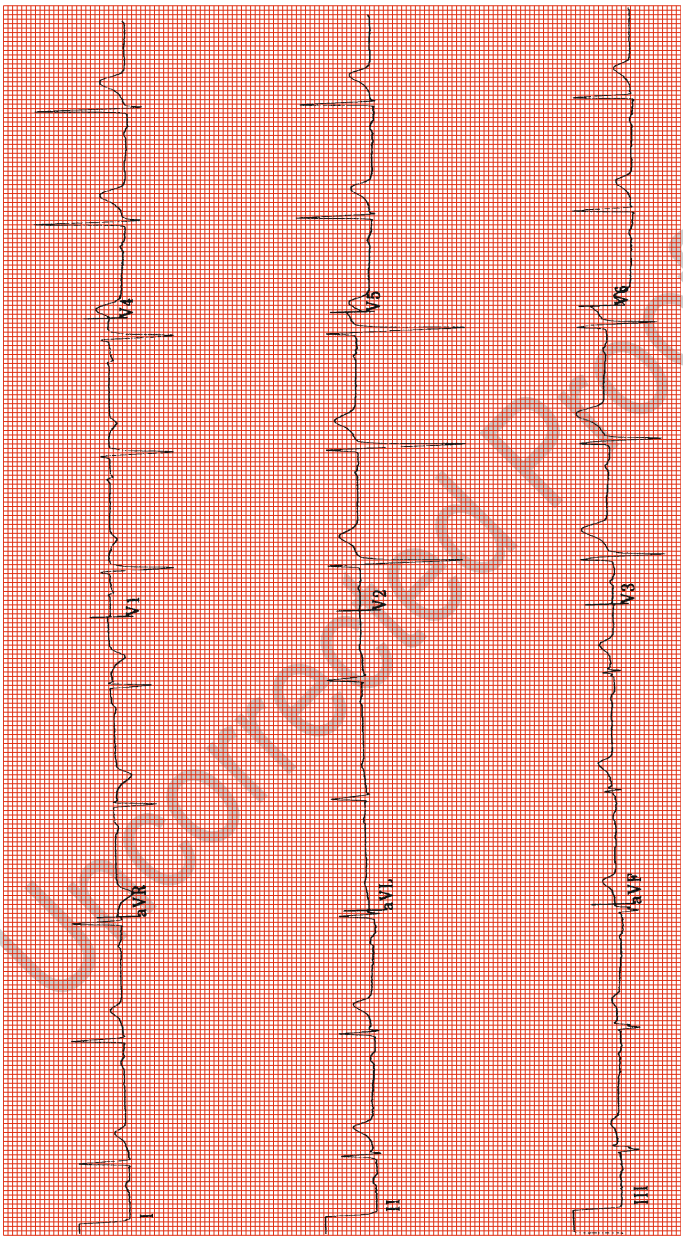


FIG. B.25. Same patient now normal trace. Diagnosis: Dextrocardia. This ECG was obtained from the right side of the chest (DA 2003).

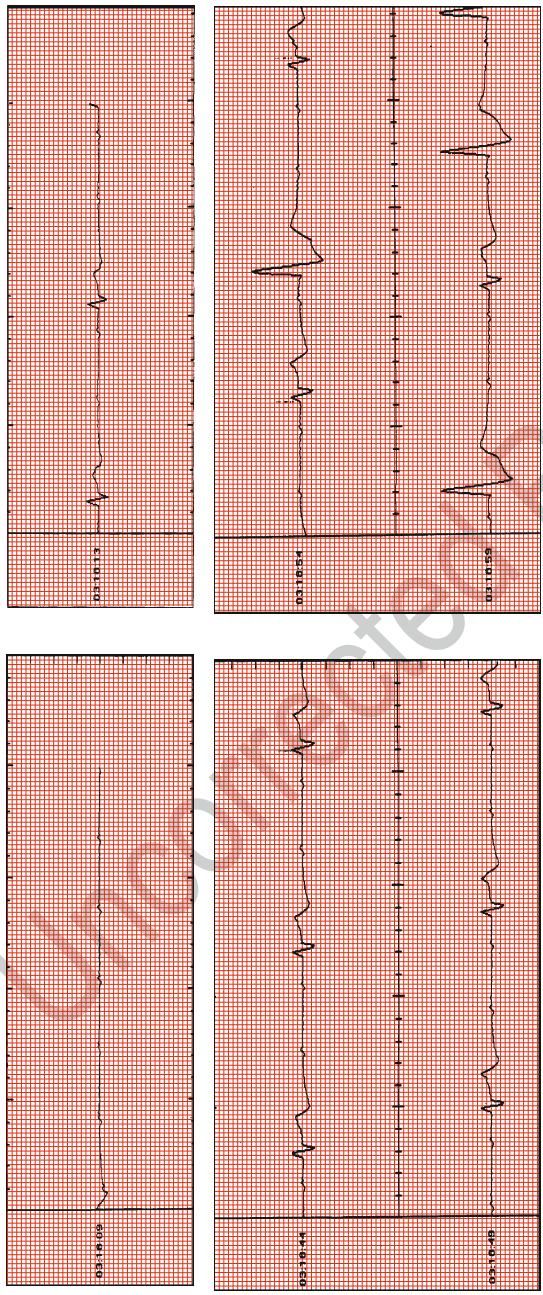


FIG. B.26. Elderly man complaining of dizzy spells. Routine ECG showed LBBB. Numerous Holter monitoring negative. During interrogation of a Reveal subcutaneous monitor CHB was detected. The patient received a pacemaker (HG 2004).

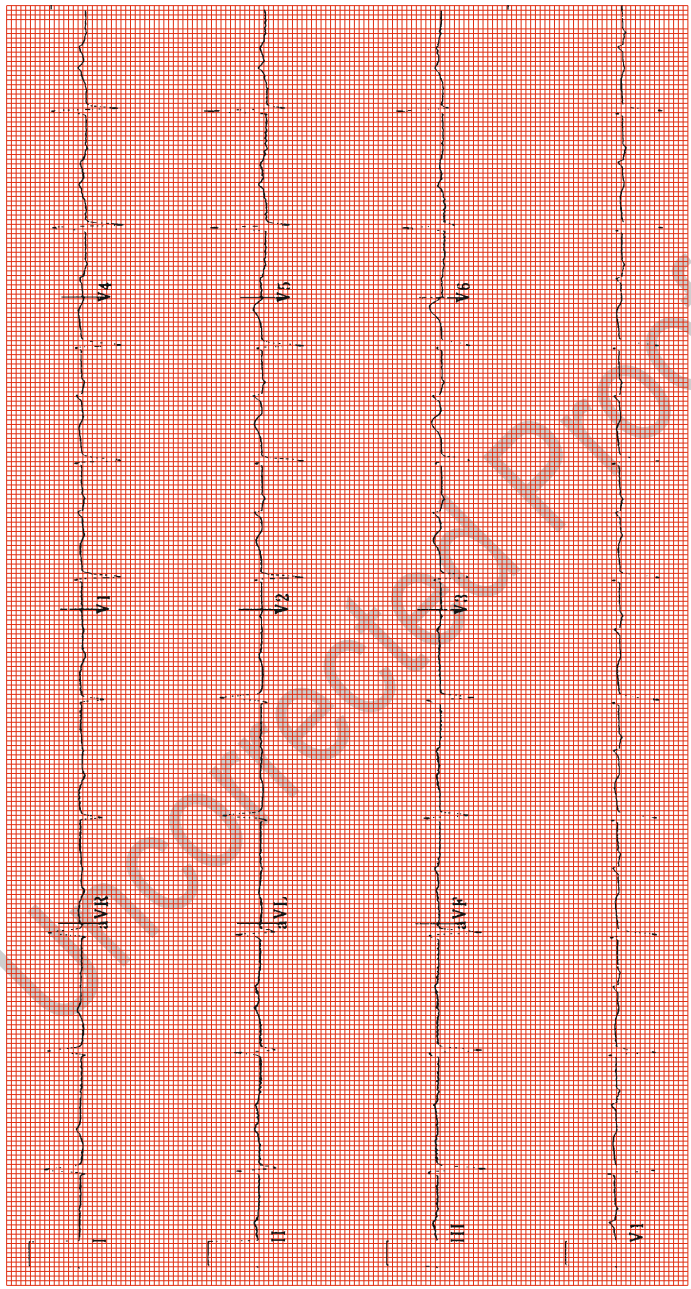


FIG. B.27. Prolonged PR interval in 70-year-old lady. Diagnosis: First degree heart block (GA 2003).

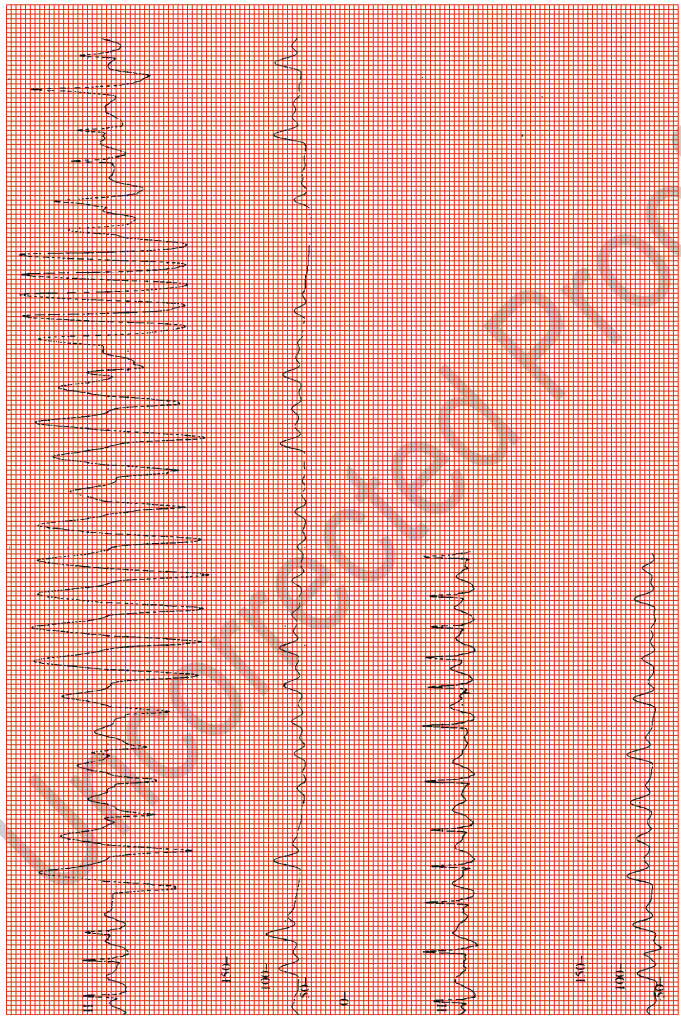


FIG. B.28. A 70-year-old male. Developed RV MI during spinal surgery. Rhythm strip VT (Tosade de Pointes). Patient succumbed despite immediate intervention in the catheter laboratory. RV infarctions are often fatal (ML 2004).

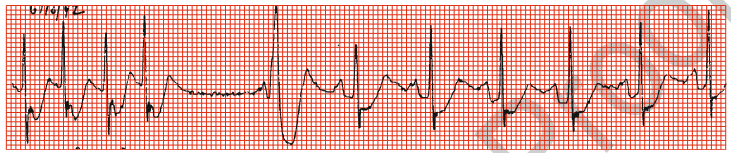


FIG. B.29. Patient with SVT. After a bolus of iv adenosine there is immediate reversal to SR (M 1992).

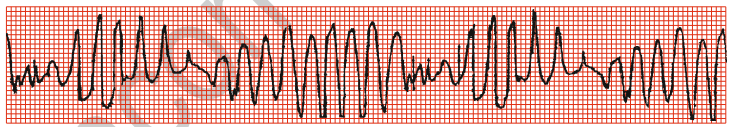


FIG. B.30. Torsade de Pointes.

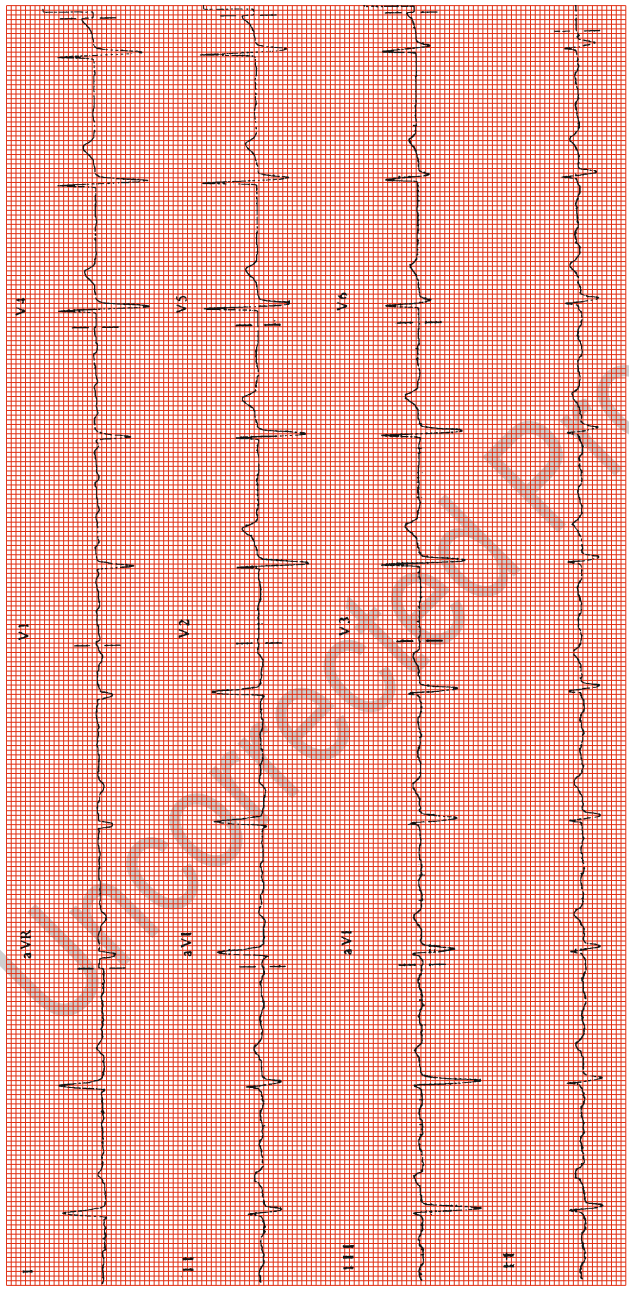


FIG. B.31. Atrial trachycardia (DS 04/01).

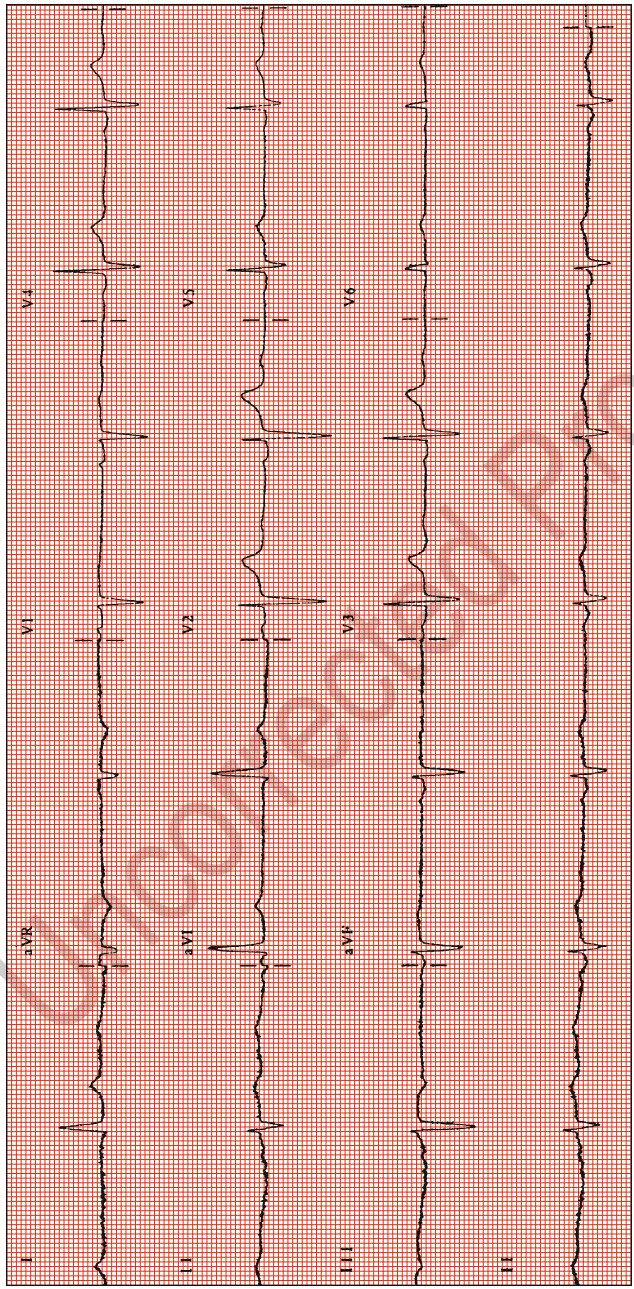


FIG. B.32. A 88-year-old woman with previous MI and CABG. SR after treatment with amiodarone (DS: 10/2001).

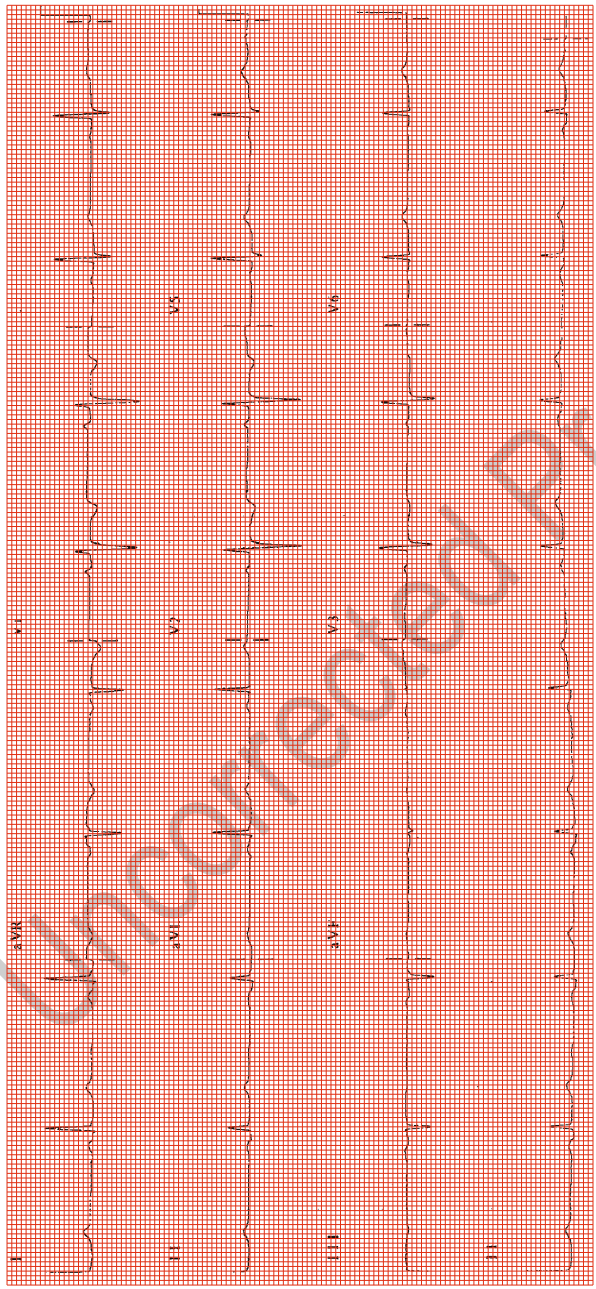


FIG. B.33. Elderly patient. ECG shows inverted T waves in V1, V3. Diagnosis: Pulmonary embolism (RR 6/02).

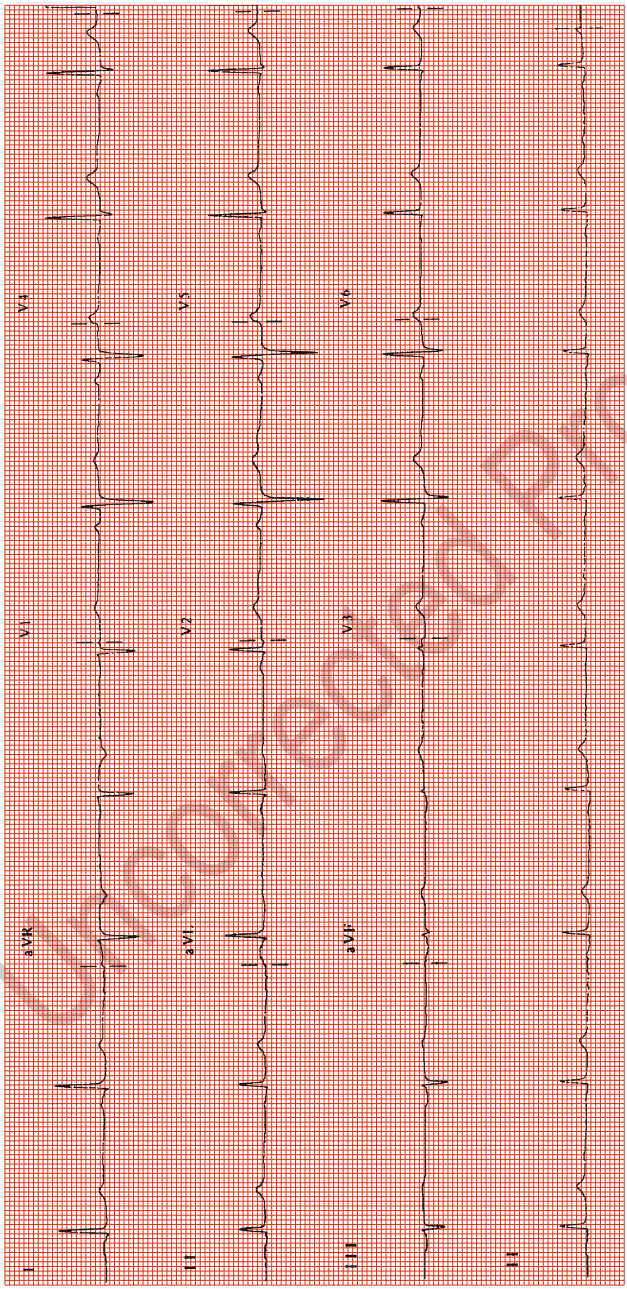


FIG. B.34. Same patient showing resolution of RV strain pattern 6 months later (RR 1/03).

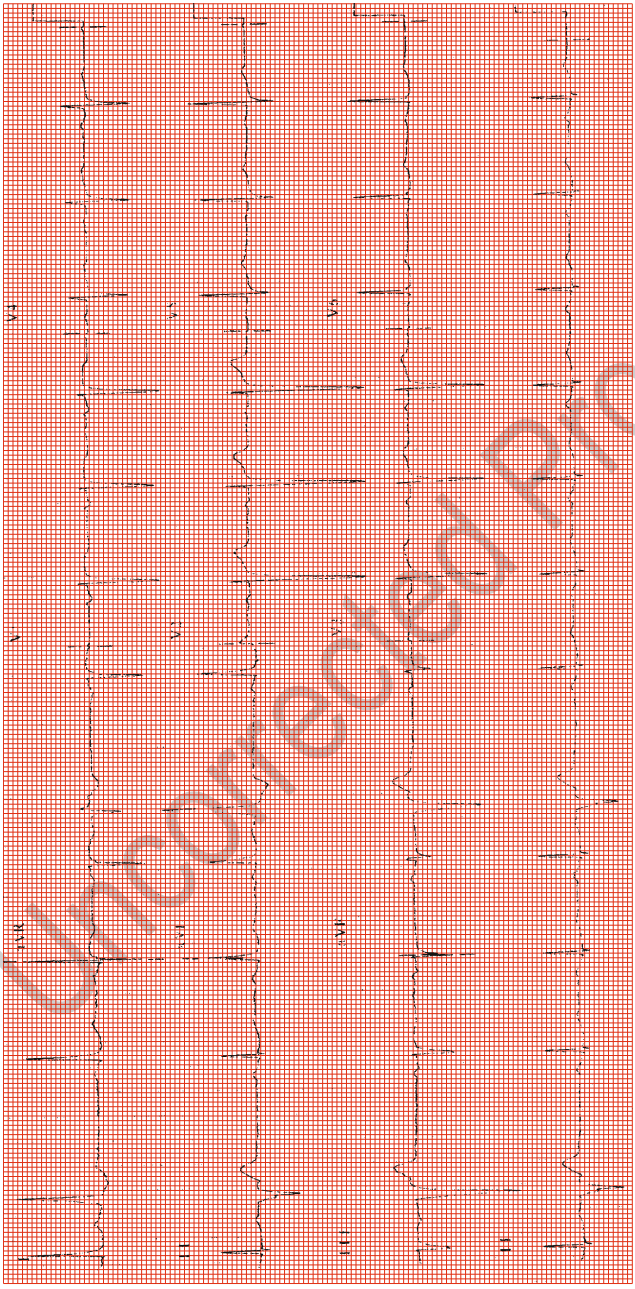


FIG. B.35. Elderly woman with angina pain. Normal ECG (VH 1999).

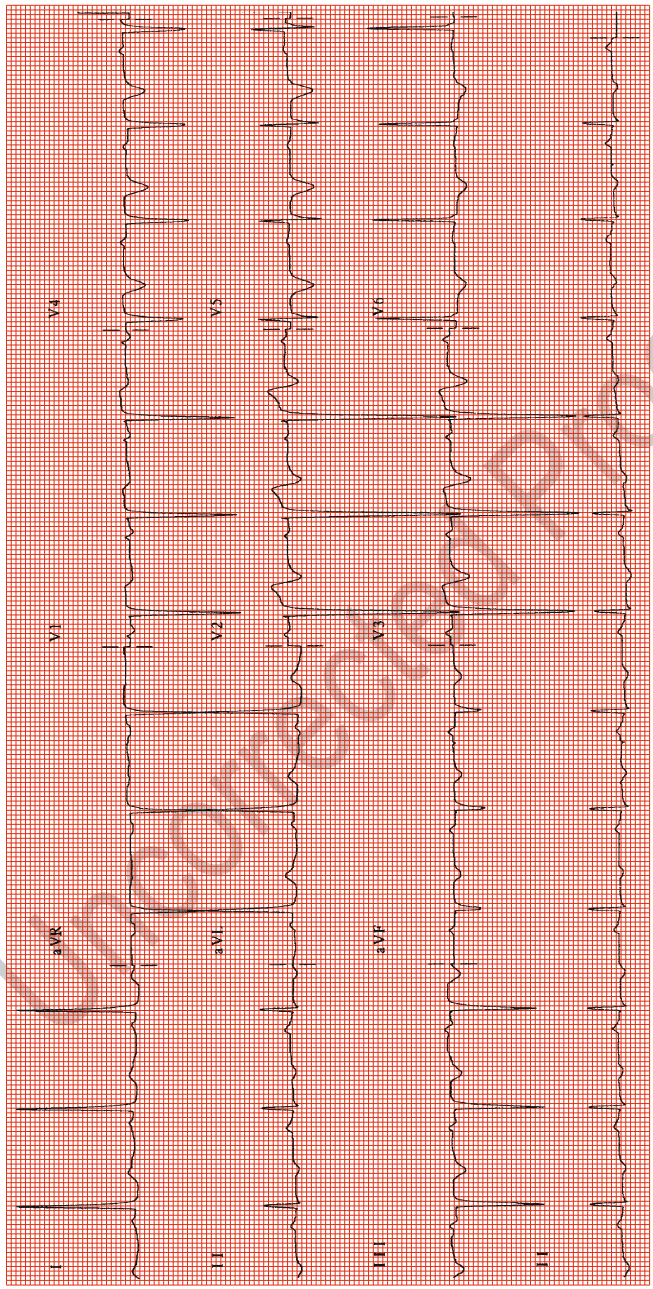


FIG. B.36. Same patient 3 years later. Acute anterior MI. Elevated troponin. Normal coronary arteriogram. Diagnosis: LV non-compaction (VH: 2002).

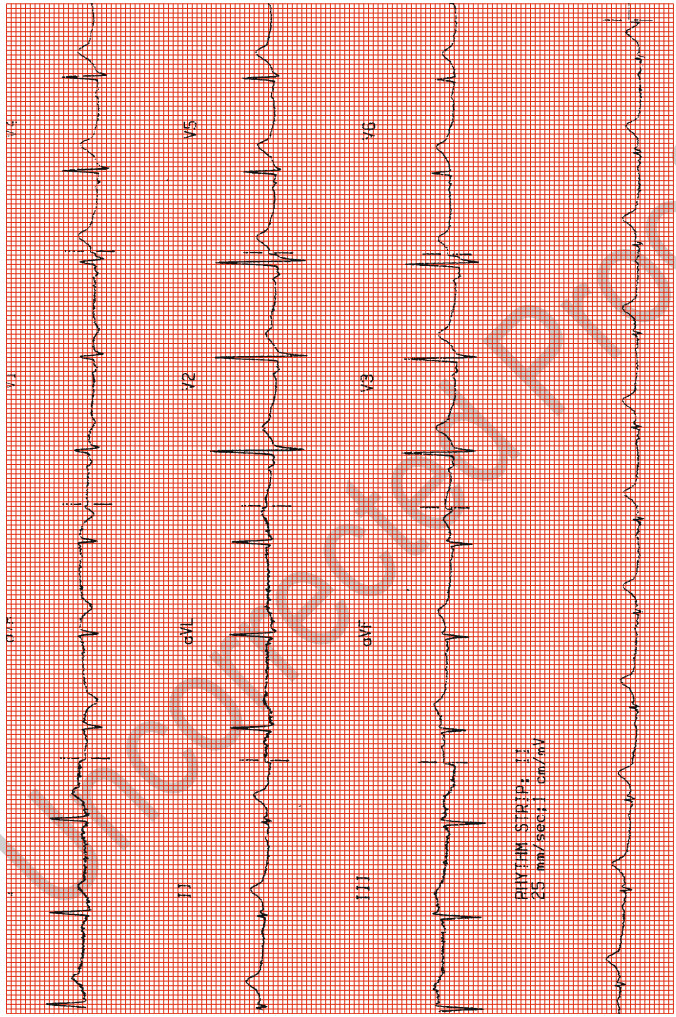


FIG. B.37. A 76-year-old male previous CABG. The ECG shows inferior Q infarct with ST elevation suggestive of localized LV inferior aneurysm. Dominant R wave in V1, V2 indicate true posterior infarction (CA 2007).

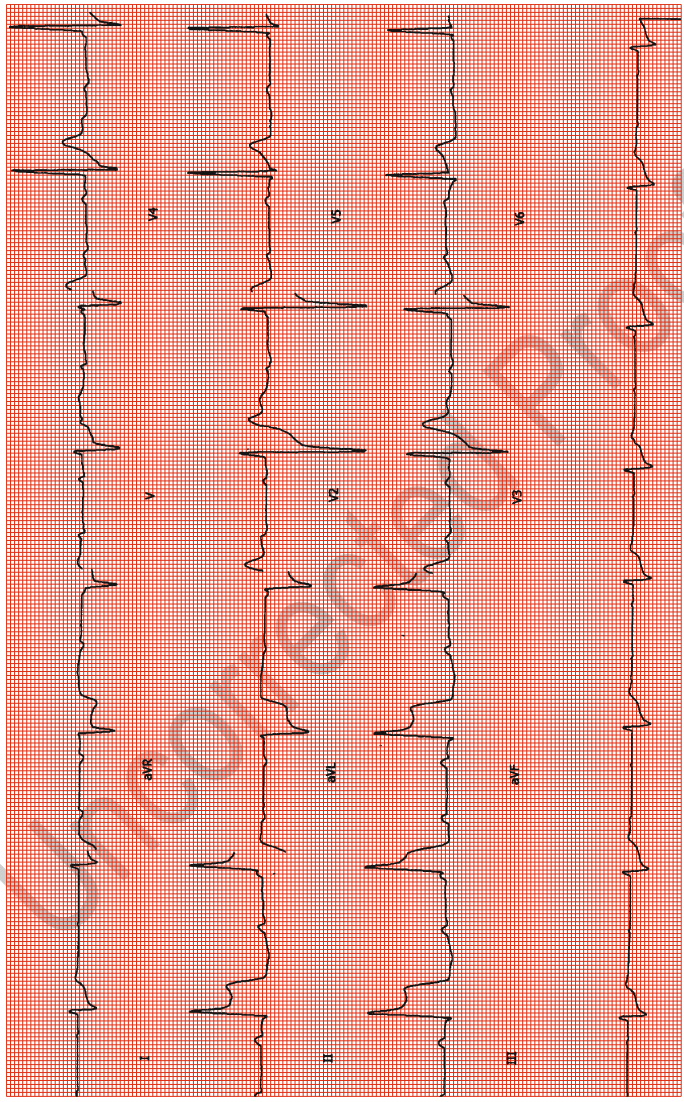


FIG. B.38. Acute occlusion RCA during PTCA (SD 9/6/05).

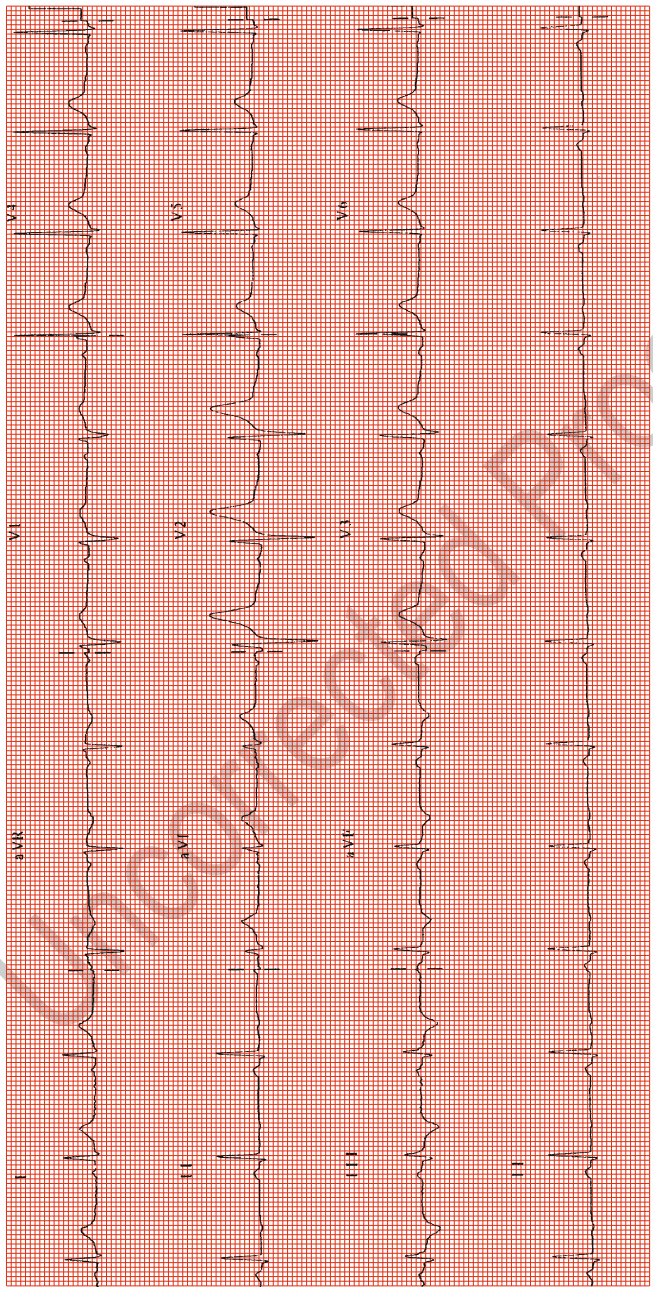


FIG. B.39. Successful resolution (stent and thrombolysis) the following day (SD 10/6/05).

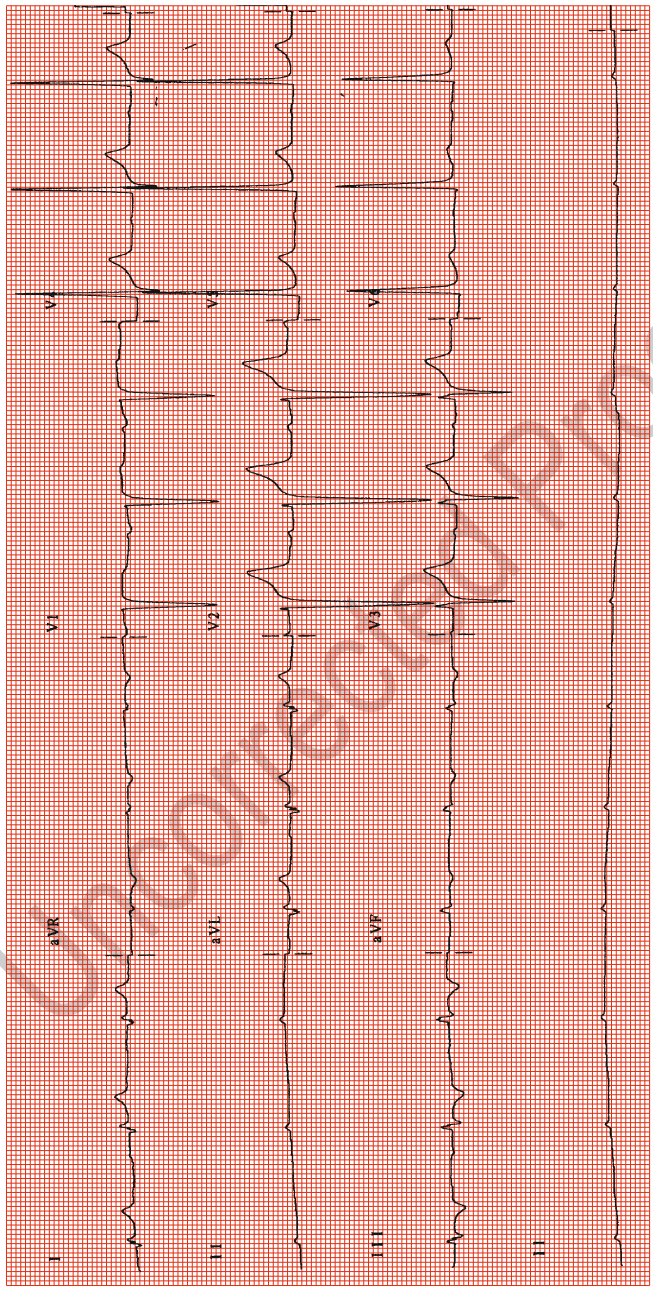


FIG. B.40. Limb leads crossed (TL 2004).

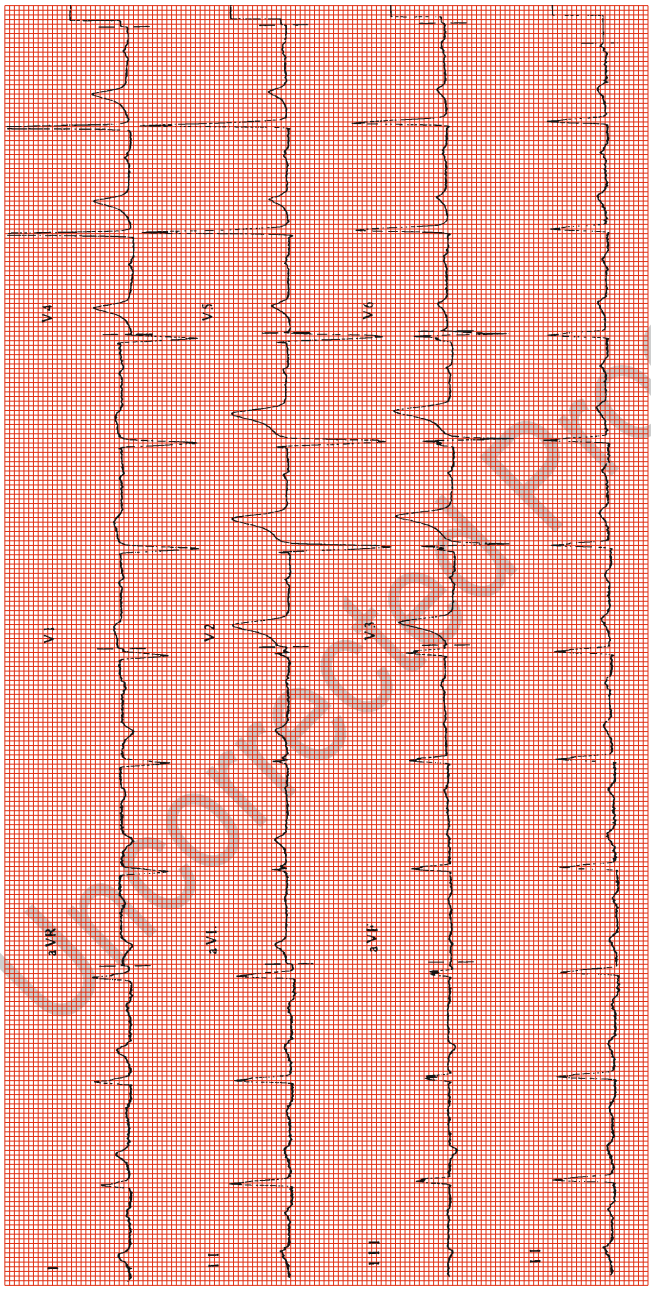


FIG. B.41. Same patient rectified (TL 2004).

FIG. B.42. A 82-year-old woman with documented PE. ECG shows inferior Q waves. Right coronary artery stented (AS 1998).

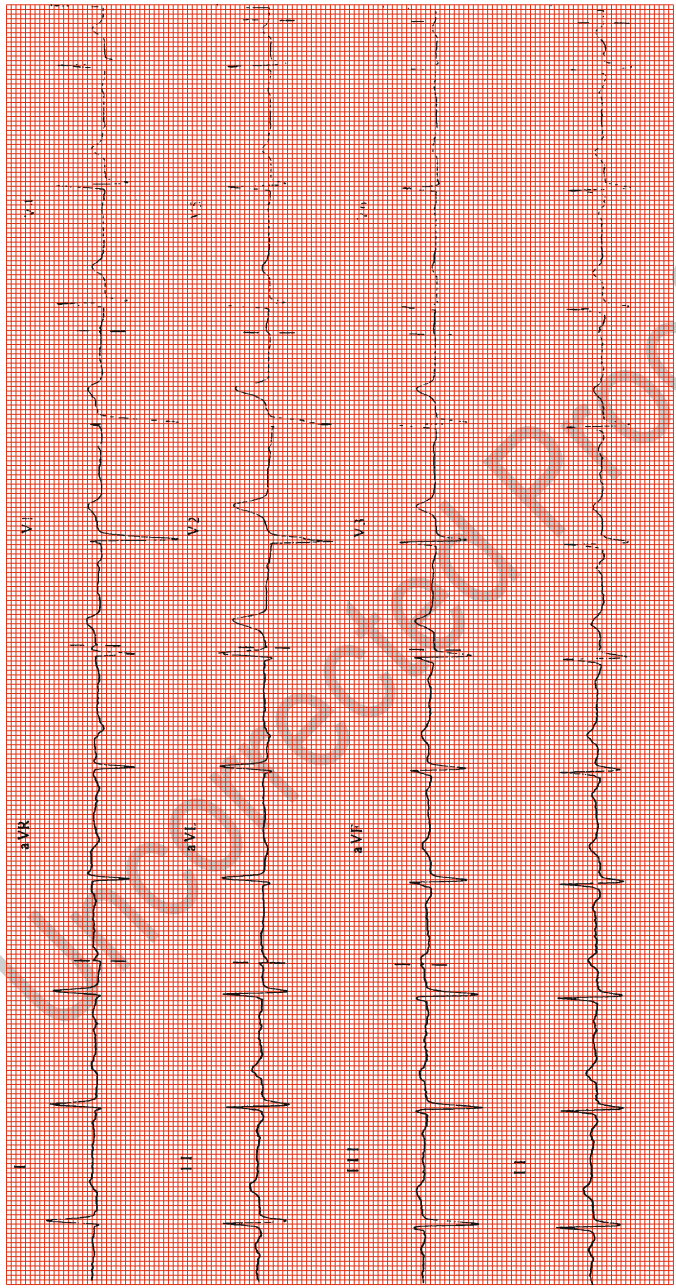


FIG. B.43. Same patient 7 years later. The inferior Q waves are no longer visible. This indicates excellent recovery with viable muscle (AS 2005).

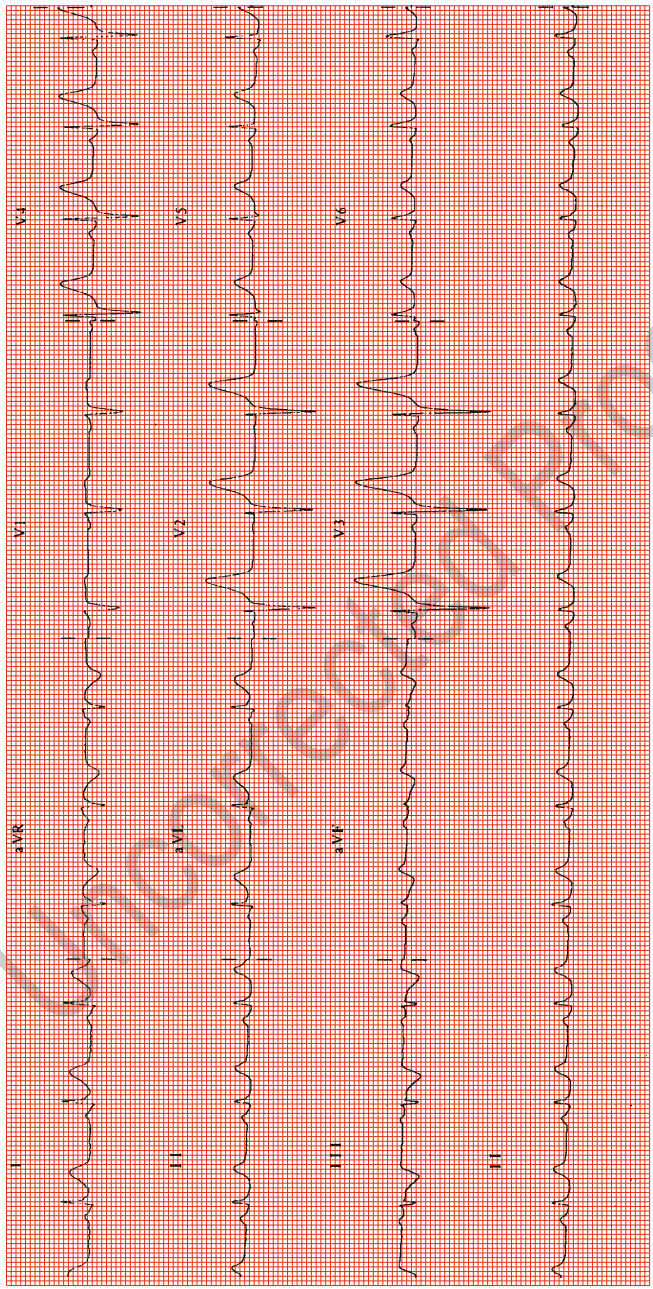


FIG. B.4. Middle aged man presenting with acute ST elevation MI (I, AVL) (A 12/1/98, 17:58 h).

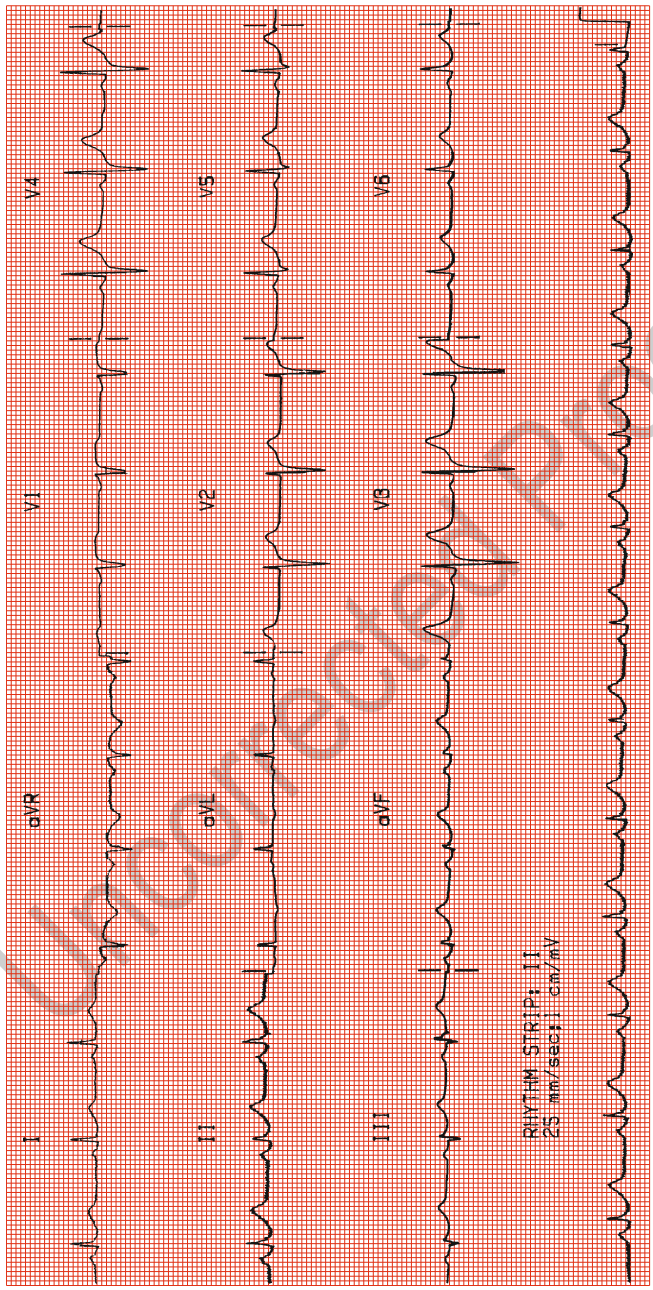


FIG. B.45. Same patient just over 1 h later after i.v. administration of RTPA. ST elevation in I and AVL have resolved. The peaked (ischemic) T waves in V2, V4 are no longer evident (A 12/1/98 19:08 h).

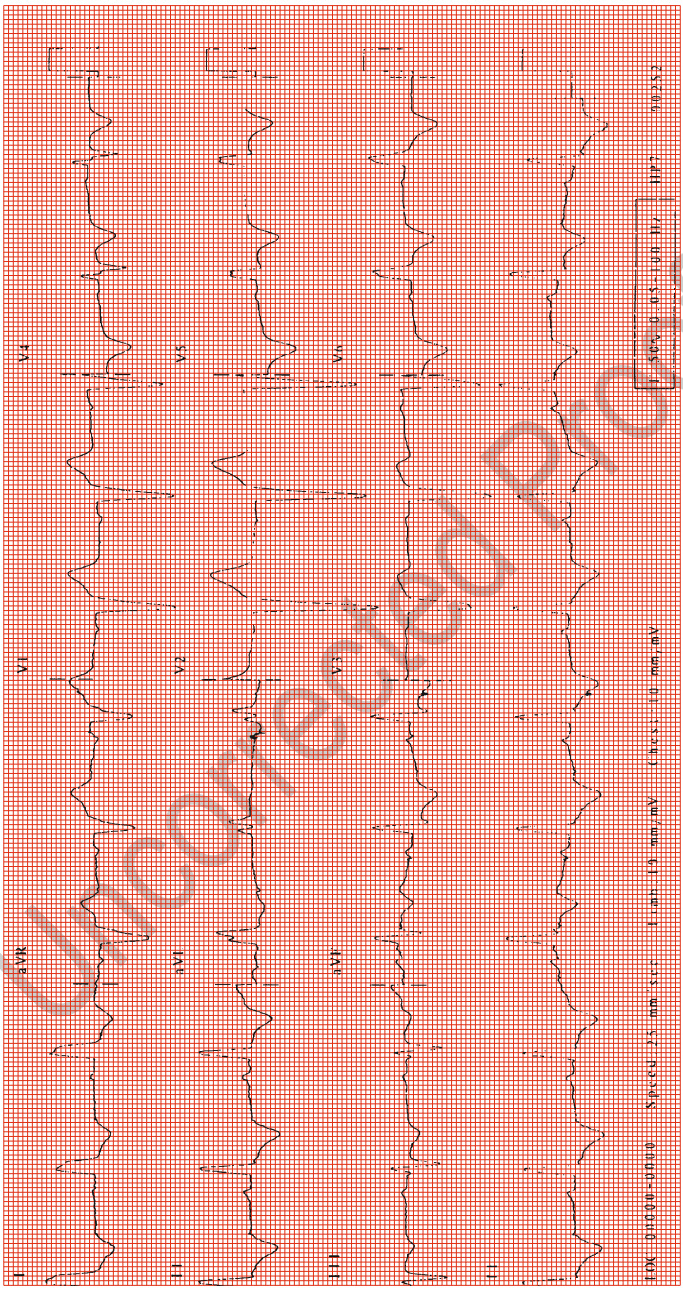


FIG. B.46. Patient with LBBB SR 66 bpm. Pacing activity inhibited (JM 8/06).

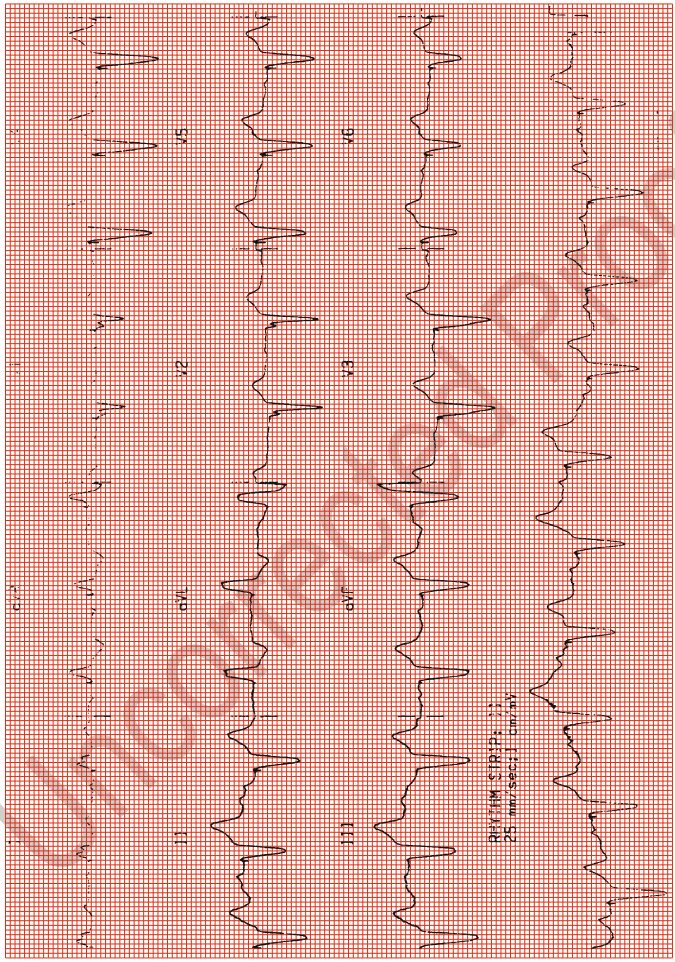


FIG. B.47. Pace rate 64 bpm. Same patient. No change of axis throughout due to RV pacing (JM 5/07).

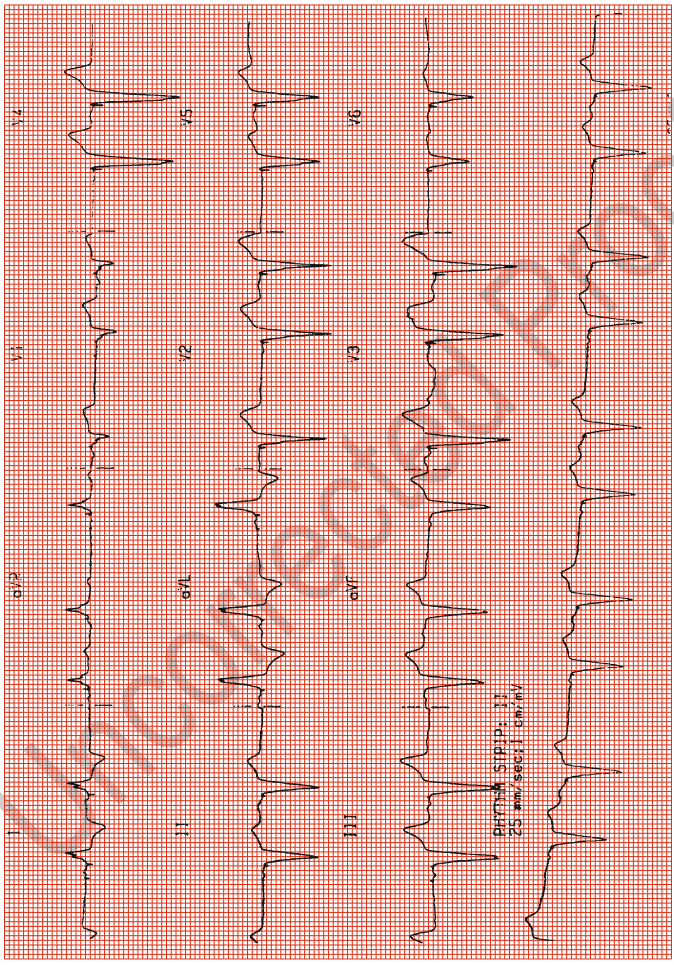


FIG. B.48. A 86-year-old patient. Pulse demonstrates bigeminy. ECG regularly occurring atrial premature beats followed by normal paced signal. In lead I the first beat shows dual chamber pacing. The second beat only shows atrial tracking (RH 6/07).

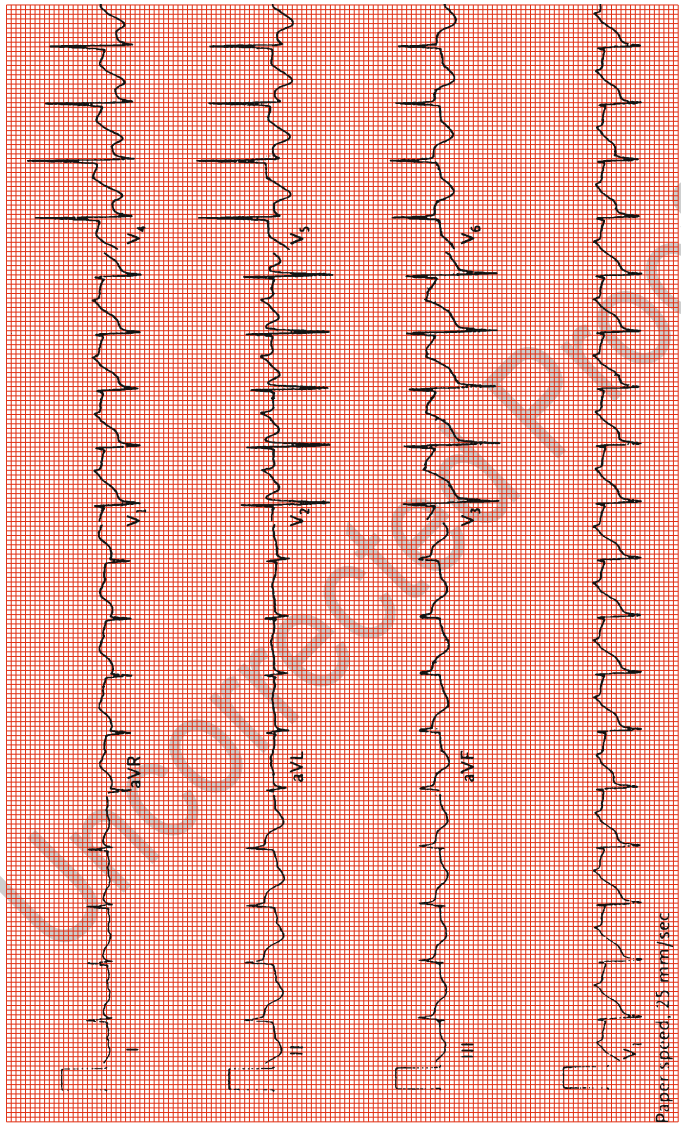
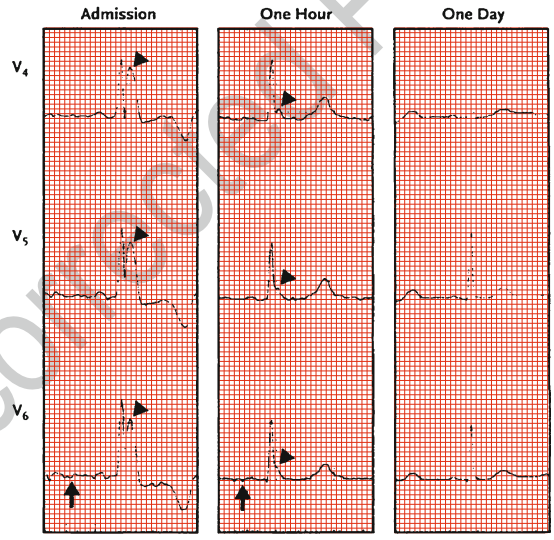


FIG. B.49. ECG changes due to intracranial hemorrhage. B Bailey, B Chaitman, NEJM; 2003: 349:6. Published by courtesy of New England Journal of Medicine.

A 19-year-old man was admitted to the trauma unit after being struck in the left temple by a brick. Surgery to evacuate an intracranial hemorrhage was performed, and an electrocardiogram (ECG) was recorded 48 h later, after a transient episode of hypoxemia. The tracing showed marked ST-segment elevation in leads II, III, aVF, and V_6 accompanied by ST-segment depression and inverted T waves in leads V_3–V_5, mimicking the changes seen in acute myocardial ischemia. The QT interval was markedly prolonged. Serial measurements of cardiac enzyme levels and an echocardiogram were normal. The next morning, the patients ECG showed less severe ST-segment deviation. This case illustrates the marked ECG changes that are occasionally seen with intracranial hemorrhage and that can lead to an erroneous diagnosis of acute myocardial ischemia.



	Admission	One Hour	One Day
Temperature (C°)	24.1	29.4	36.6
Heart rate (beats/min)	50	70	98
QRS interval (msec)	184	119	71
QTc interval (msec)	516	502	403

FIG. B.50. ECG changes in hypothermia. M Krantz and C M Lowery. NEJM; 2005: 352:2. Published by courtesy of New England Journal of Medicine.

A 47-year-old man with chronic schizophrenia was hospitalized after prolonged hypothermia. The initial electrocardiogram revealed Osborne waves (*arrowheads*) similar in amplitude to the R waves. Characteristic sinus bradycardia and prolongation of the QRS interval and the corrected QT interval (QTc) were also noted. During rewarming, the Osborn waves diminished in amplitude, and they disappeared after 24 h. The baseline tremor artifact caused a shivering (*arrows*) resolved on normalization of the patients core body temperature. In 1953 Dr. John Osborn described the J wave as an "injury current" resulting in ventricular fibrillation during experimental hypothermia. More recent findings suggest that hypothermia increases the epicardial potassium current relative to the current in the endocardium during ventricular repolarization. This transmural voltage gradient is reflected on the surface electrocardiogram as a prominent J, or Osborn, wave. The differential diagnosis of prominent Osborn waves includes early repolarization, hypercalcemia and the Brugade syndrome.