ECG Diagnosis in Clinical Practice

Second Edition

ECG Diagnosis in Clinical Pratice

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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.

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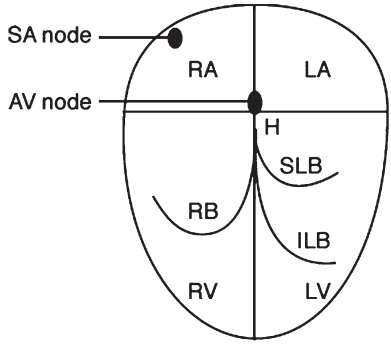

FIG. 1.1. Electrical conduction through the heart: From the sinoatrial node across the atrial musculature, the impulse reaches the atrioventricular node. It proceeds downwards through the bundle of His, then simultaneously to the right ventricle, through the right bundle and the left ventricle through 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.

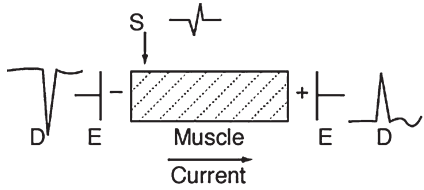

FIG. 1.2. Muscle depolarization. When stimulated, the muscle develops a negative charge. An electrode facing the oncoming current will record an upright (positive) deflection. The current moving away inscribes a downward (negative) signal. Halfway between the two, the deflection is biphasic. *D* deflection;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

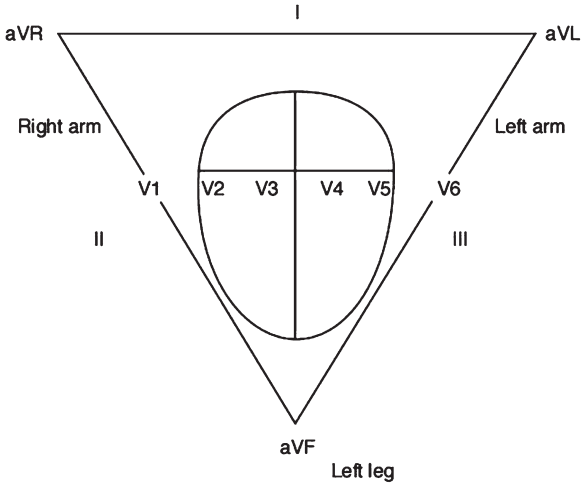

FIG. 1.3. Schematic representation of 12 leads (electrodes) placed around the heart.

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).

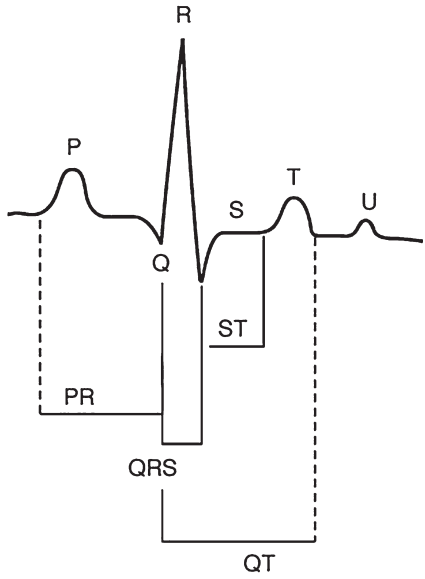

FIG. 1.4. Normal ECG recording. This denotation was introduced by Einthoven. The U wave is often not discernible; *P* atrial depolarization. *QRS* ventricular depolarization; *T* ventricular repolarization; *U* either represents after potentials of the ventricular myocardium or repolarization of the 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.

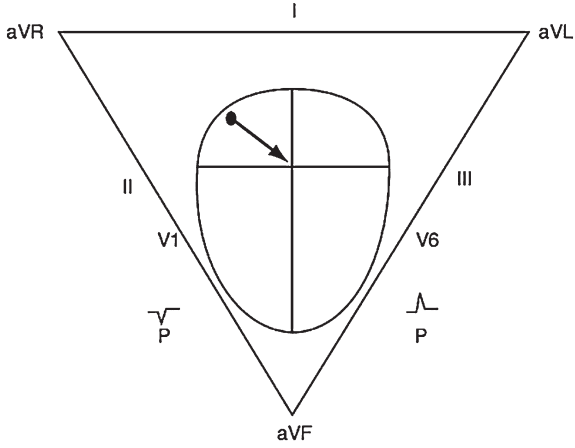

FIG. 1.5. The P wave vector: The P wave impulse travels from right to left. Leads facing the incoming signal (III, avf and V6) will record a positive trace. Negative deflections appear in avr and V1, the impulse being carried away from these sites.

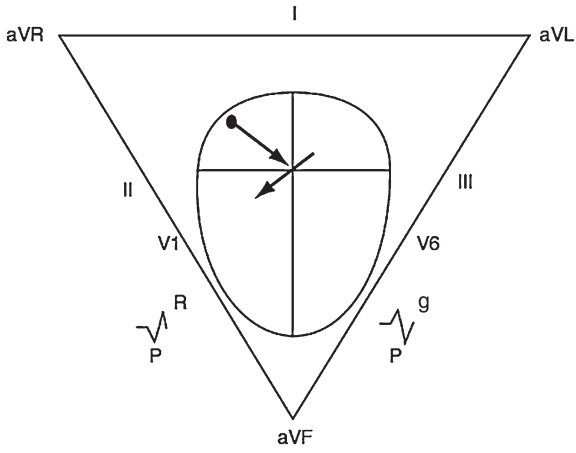

FIG. 1.6. Septal activation. The initial QRS activation results from the electrical impulse stimulating the interventricular septum through the bundle of His. The initial deflection will be positive in lead V1 and negative in lead V6 (i.e., R and Q).

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.

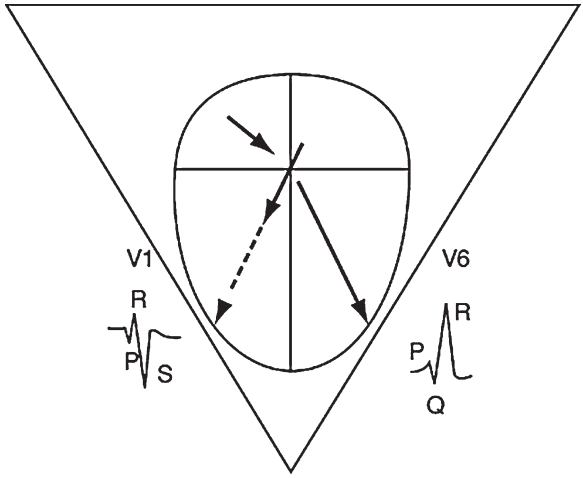

FIG. 1.7. Ventricular activation. The main vector (an electrical force that has both magnitude and direction) travels from right to left (right ventricular currents are dwarfed by the thicker left ventricular musculature). Positive tracing are observed in leads III and V6 (R) and a negative tracing is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

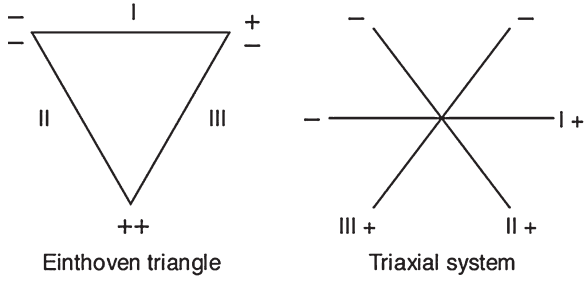

FIG. 1.8. The three standard (limb) leads. The Einthoven triangle translated into a triaxial system shows the positive and negative terminals of each lead.

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

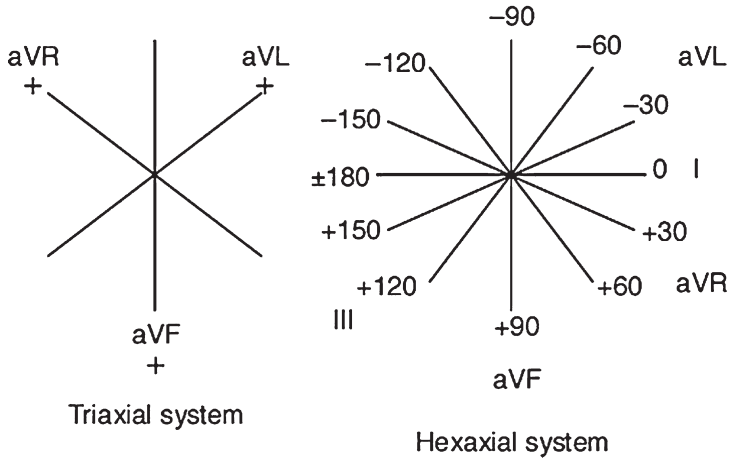

FIG. 1.9. Standard and augmented leads. Combination of all 12 leads showing polarity and degrees within a circle.

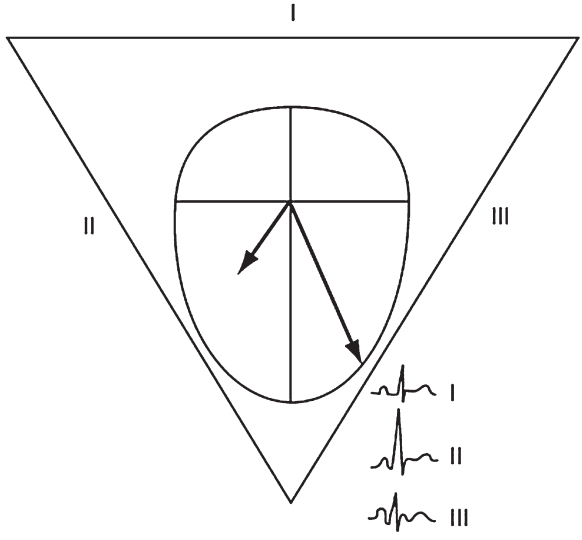

FIG. 1.10. The mean vector of left ventricular depolarization. Lead II is the closest parallel to the mean vector of depolarization. It shows the greatest deflection and an axis of +60°.

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

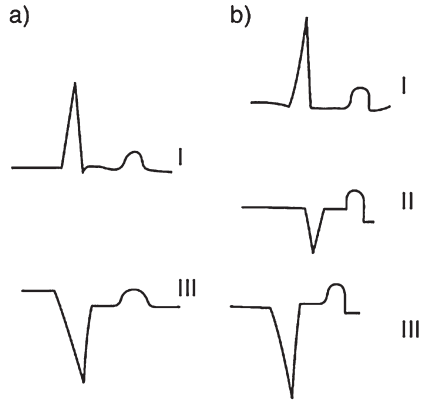

FIG. 1.11. Left (superior) electrical axis deviation. (**a**) Left (superior) axis deviation is present when the main vectors in leads I and III move away from each other. (**b**) When lead II is also negative (–30°), the trace is described as showing a *pathological* left axis deviation and represents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

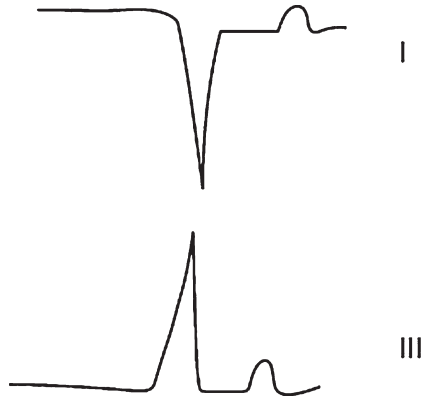

FIG. 1.12. Right (inferior) electrical axis deviation. The main deflections in leads I and III point towards one another. This indicates right-sided problems.

(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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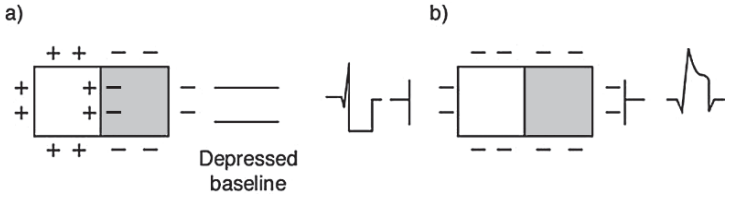

FIG. 2.1. Current of injury. (a) The shaded area is ischaemic and becomes negatively charged. The ECG shows a depression of the baseline. (b) During depolarization the residual healthy muscle becomes negatively charged. No current flows. The baseline returns to normal and the ST segment appears elevated.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

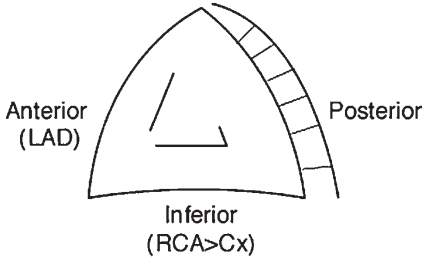

FIG. 2.2. The three areas of myocardial infarction. The anterior surface of the myocardium is supplied by the left anterior descending (LAD) artery. The inferior surface obtains its supply essentially from the right coronary artery (RCA) and occasionally from the circumflex (Cx) artery.

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.

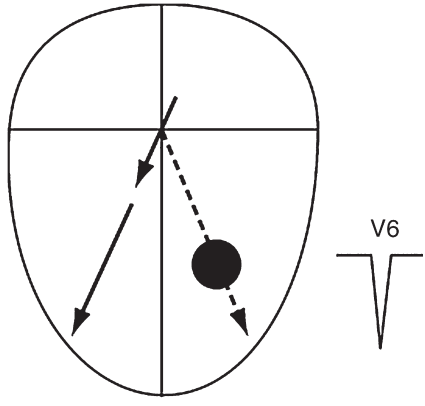

FIG. 2.3. Anterior infarction. Leads facing the infarcted territory pick up unopposed forces moving away through the nonconducting "window," e.g., Q waves in V6.

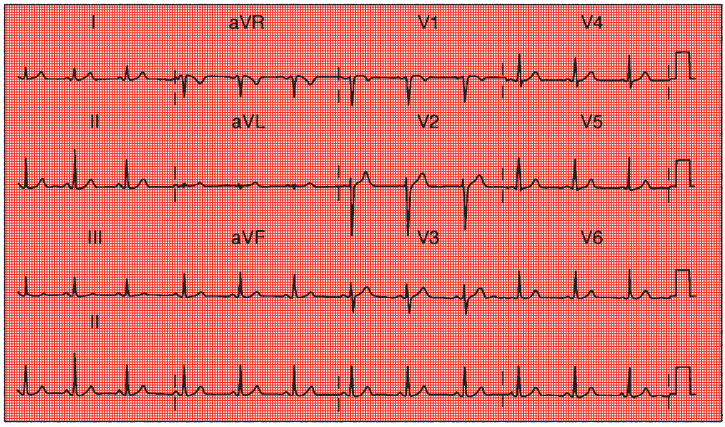

FIG. 2.4. Normal trace (LG; 24/4/98).

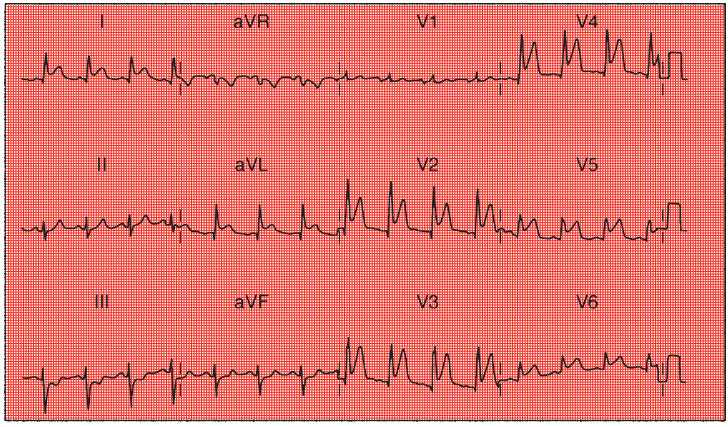

FIG. 2.5. Acute anterior infarction. ST elevation in leads I, aVL and V2–V6 (AN; 15/9/85).

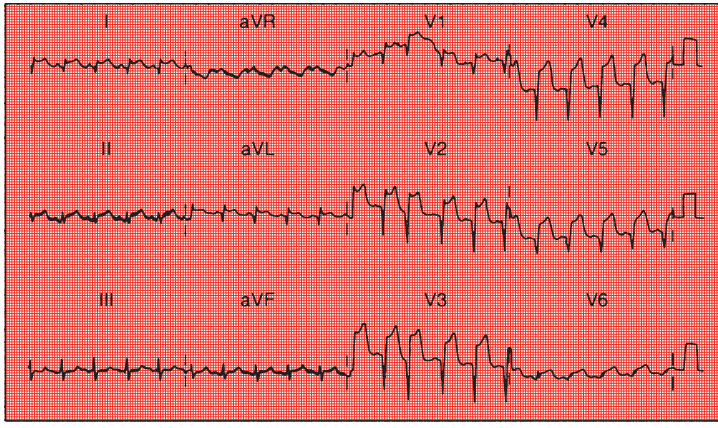

FIG. 2.6. The same patient 3 days later. Q waves are apparent in leads I, aVL and V1–V5. ST elevation is still present (AN; 18/9/85).

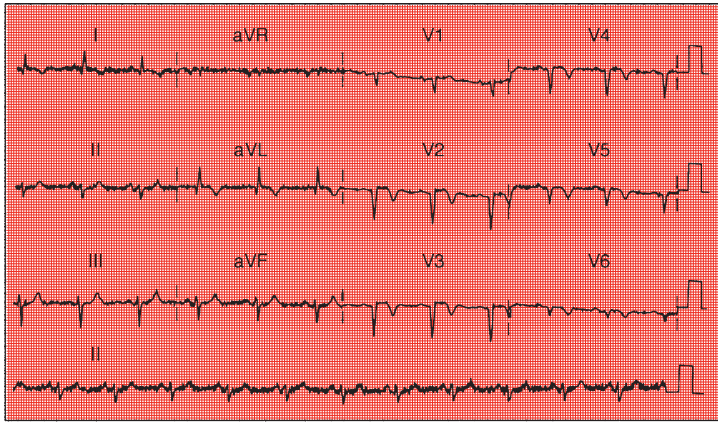

FIG. 2.7. Two years later the ST segments are back to baseline but Q waves are seen in leads I, aVL and V1–V6 with T wave inversions (AN; 6/2/87).

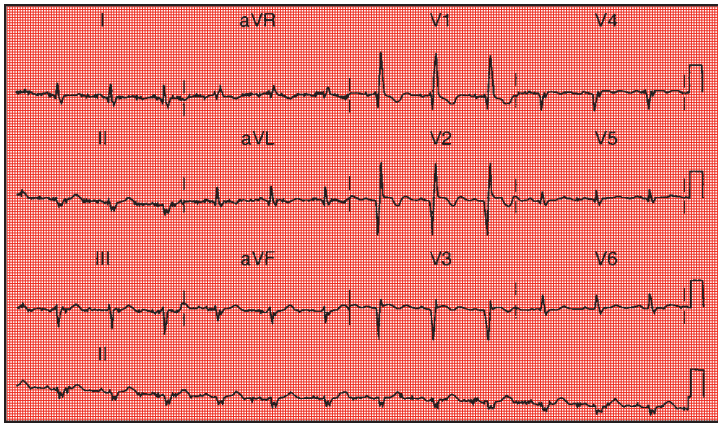

FIG. 2.8. Eleven years later the patient has developed right bundle branch block. The anteroseptal Q waves remain. Residual ST elevation in leads V2 and V3 are indicative of left ventricular aneurysm (AN; 25/11/98).

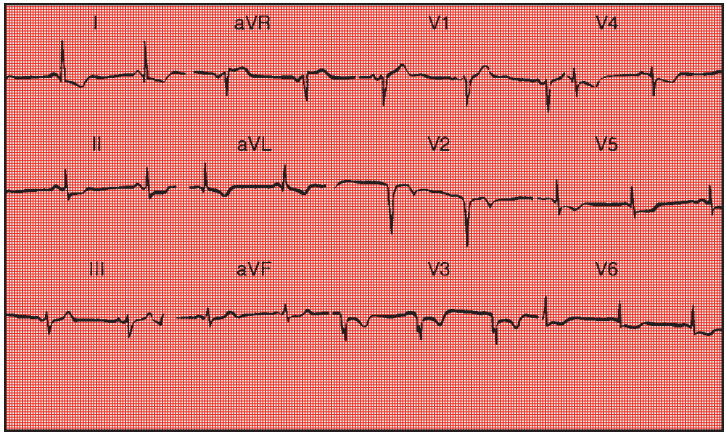

FIG. 2.9. Q wave anteroseptal infarct with widespread ST depression. The latter indicates widespread ischaemic territory (MB; 3/10/79).

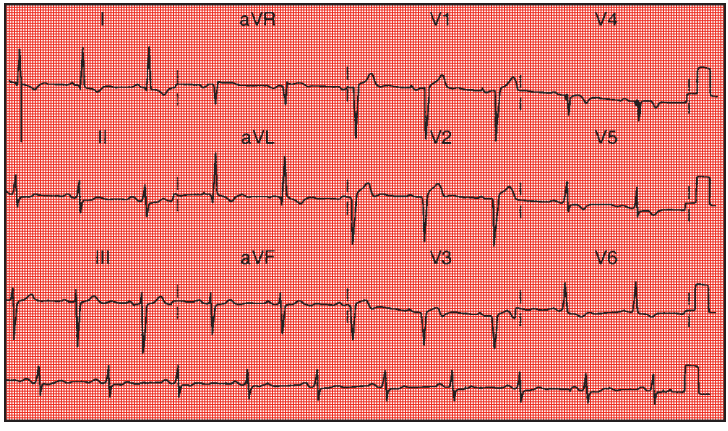

FIG. 2.10. The same patient 19 years later. The ECG has a very similar pattern, with fewer ST segment depressions. The patient was treated conservatively (MB; 29/6/98).

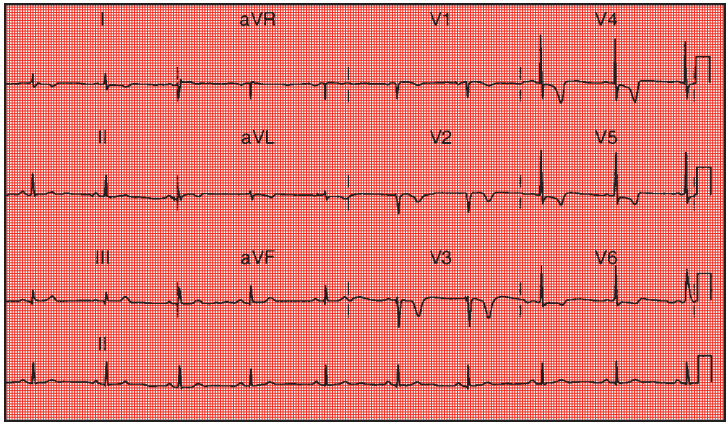

FIG. 2.11. Young male patient with acute anteroseptal infarction (non ST elevation infarction) (CA; 4/6/98).

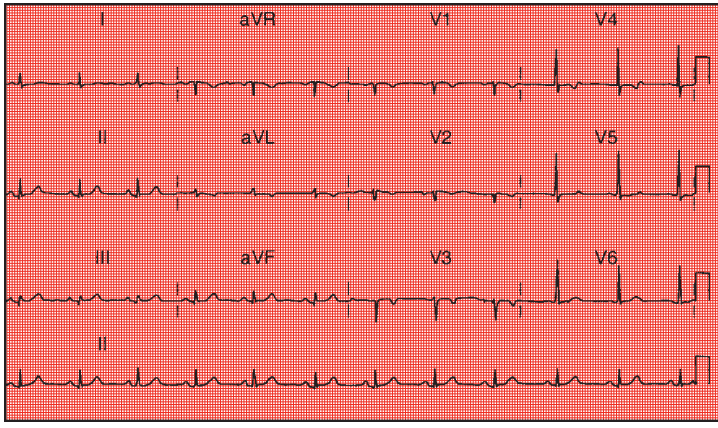

FIG. 2.12. This trace was recorded soon after successful angioplasty and stent insertion in LAD (CA; 18/6/98).

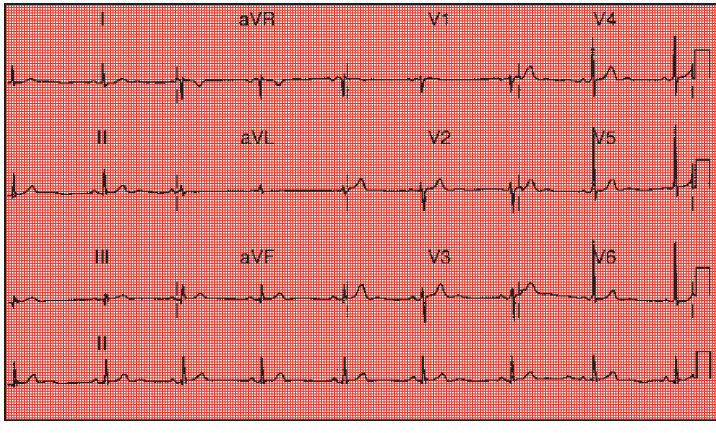

FIG. 2.13. After 3 months, the patient had made a full recovery (CA; 28/9/98).

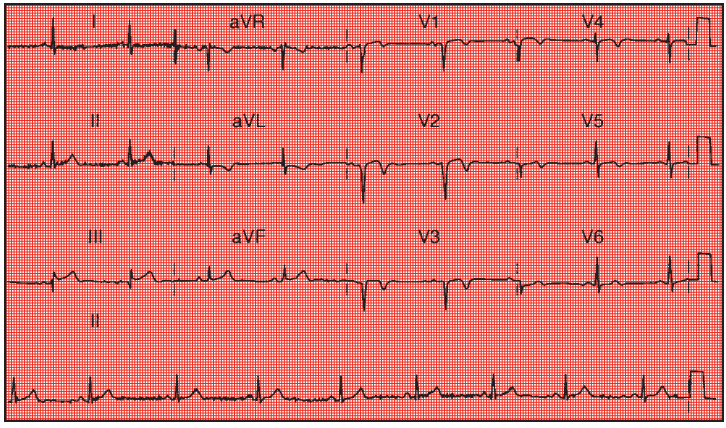

FIG. 2.14. Young male patient with anteroseptal Q wave infarction. He underwent a successful bypass operation (MG; 15/10/93).

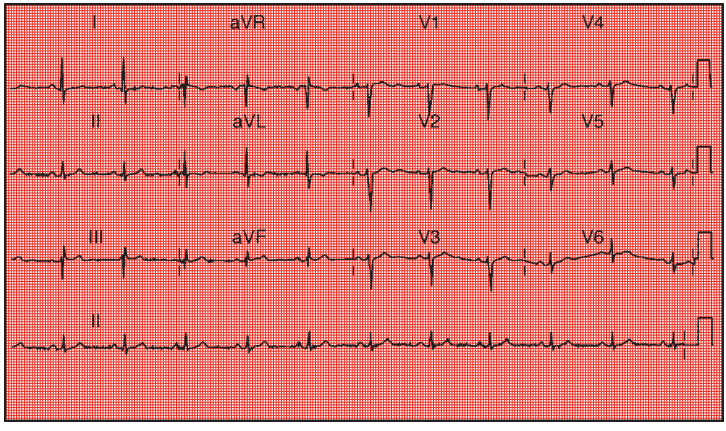

FIG. 2.15. After 6 years the only remaining abnormality is mild T-wave inversion in lead aVL. R wave progression over the praecordium has not fully recovered (MG; 22/9/99).

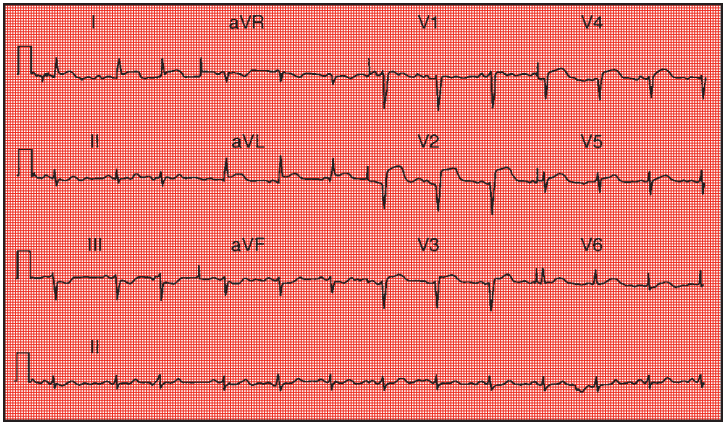

FIG. 2.16. Widespread Q wave acute anterior infarction. ST segment elevation is present in leads I, aVL and V2–V5 with Q waves in lead V2 and reciprocal changes (ST segment depression) in leads III and aVF (ST elevation infarction) (TB; 30/7/99).

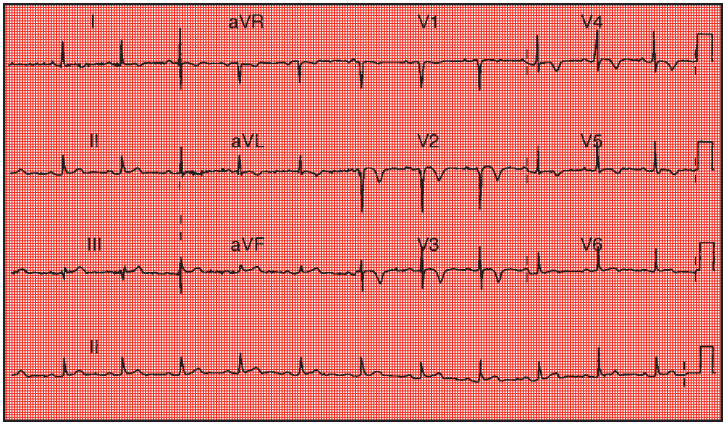

FIG. 2.17. This 43-year-old male suffered severe chest pain after cycling. He had an anteroseptal infarct. The LAD artery was successfully ballooned and stented (AF; 12/4/99).