Diagnosis and Treatment of Cardiac Arrhythmias

Second Edition

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Dedication

This book is dedicated to Dr Adolf Schott, M.D., F.R.C.P.(Ed.), who first stimulated my interest in this subject. I hope he will forgive any errors or omissions, for although I have frequently sought his advice, I have not imposed on his kindness by asking him to read the manuscript.

INTRODUCTION TO THE SECOND EDITION

Knowledge of the mechanisms responsible for disorders of cardiac rhythm continues to grow rapidly and at the same time new drugs and improved electrical techniques are increasing our ability to treat them successfully. Inevitably the second edition is enlarged and much has been re-written. A new chapter has been added on the Wolff–Parkinson–White syndrome and its associated arrhythmias. Apart from this, the general plan of the book has remained unchanged.

In Chapter 1, the internodal tracts whose existence has been fully confirmed are described. In the introduction to the first edition, it was remarked that the subject of arrhythmias had unfortunately become beset with semantic inconsistencies. Changes in long-established terminology, therefore, require full justification. Recent electrophysiological studies have failed to confirm that the A.V. node is the most important subsidiary pacemaker in the heart. The term A.V. junctional or junctional must therefore be used instead of nodal for that type of extrasystole, escape rhythm or tachycardia believed to originate in the A.V. junction. This change in traditional terminology is fully justified on physiological grounds and has been adopted in this edition. I agree with Papp, however, that to substitute dysrhythmia for arrhythmia is false linguistic purism. The term arrhythmia has become hallowed by tradition and there is no sound physiological reasons for changing it, although the occasional use of the term dysrhythmia as an alternative is acceptable.

It is a pleasure to acknowledge my very sincere thanks to Dr A. Schott for his very helpful criticisms of the first edition and for his constant help and advice with the second.

I am grateful to Dr R. Gold for allowing me to use *Figure 10.7* and also to Dr Hudson for *Figure 10.3.*

Once again I must acknowledge my indebtedness to my secretary, Mrs O. Adams, for all her help in the preparation of the manuscript and index for the second edition.

INTRODUCTION TO THE FIRST EDITION

Disorders of cardiac rhythm have long been a source of great interest to physicians, but any appreciation of their underlying mechanism or significance had to await some understanding of cardiac physiology. Towards the end of the nineteenth century, the development of graphic methods for recording the activity of the different heart chambers, together with a recognition of the fundamental physiological properties of heart muscle,

began to shed light on a subject which had been wrapped in mysticism and dogma.

At the beginning of this century, Sir James Mackenzie, when working as a general practitioner in Burnley, Lancashire, made perhaps the first major clinical contributions to our understanding of different forms of irregular heart action. He developed his 'polygraph' which recorded simultaneously the jugular venous pulse and an arterial pulse. By painstaking and inspired analysis of his polygraphic records, he was able to differentiate between several different forms of irregular pulse, such as sinus arrhythmia, extrasystoles and atrial fibrillation, and to recognize their widely varying clinical significance.

The development of the electrocardiograph by Einthoven provided a new instrument for the analysis of arrhythmias, for it enabled the electrical activity of the different heart chambers to be recorded on a single tracing. Today, the electrocardiograph remains largely unchallenged as the most informative method of studying clinical disorders of cardiac rhythm. More recently, in animal studies, the electrophysiologist has begun to shed new and fundamental light on the initiation and propagation of the cardiac impulse. There seems little doubt that disturbances of rhythm encountered clinically will eventually be explained in terms of changes in the flow of electrically charged ions across the cardiac cell membrane.

A relatively brief acquaintance with the subject of clinical arrhythmology soon reveals that it has two distinct sides. On the one hand, there is the essentially practical aspect which entails the rapid recognition, from the electrocardiogram, of the type of arrhythmia present and the institution of the appropriate therapy. On the other hand, there is the more academic aspect, in which the record of an unusual arrhythmia is carefully studied and analysed in the hope of understanding its underlying mechanism in terms of cardiac physiology.

The pragmatist may object that the leisurely study of an arrhythmia, treating its analysis as a proposition in logic, with no sense of urgency in reaching a solution, is a task better suited to the philosopher, for, he may argue, a belated solution can offer little practical help to the patient. Such a view is very short term. In the first place, its acceptance would close the door, to the physician, on a fascinating and intellectually rewarding study. In the second place, to acquire virtuosity in the essentially practical side of arrhythmias demands an apprenticeship on the academic side. In the past 20 years in Great Britain, interest in this side of arrhythmias has tended to lapse. This book has been written, firstly, in the hope that it may prove useful to those engaged in hospital practice who are called upon to deal with patients suffering from arrhythmias and, secondly, that it may re-awaken interest in the purely academic side of the subject.

It is now the established practice in recording an electrocardiogram to employ approximately twelve different leads from each patient; consequently, the length of the individual leads has tended to become shorter. When the patient is in normal sinus rhythm, short strips of individual leads are quite adequate, but many arrhythmias demand much longer records of at least one lead before an adequate analysis is possible. It is impossible to give any dogmatic guide as to the best lead to use or the optimum length to

record. As a general rule, the lead for recording an arrhythmia, for analysis, should be that in which the P waves are most clearly discernible. This is most commonly lead II or VI, but the best lead may vary from patient to patient. The optimum length of the record depends on the arrhythmia; one lasting half a minute is usually adequate, but intermittent arrhythmias may demand much longer recordings.

TERMINOLOGY

It is unfortunate that during the past 50 years the subject of arrhythmias has become beset with semantic inconsistencies. There is still, for example, no general agreement about the terminology for the commonest disturbance of rhythm, the extrasystole. The terminology used in this book is, as far as possible, the one most generally accepted by writers on the subject.

No attempt has been made to provide a complete bibliography to the subject. Nevertheless it is hoped that the limited number of references given may prove useful to those seeking further information.

Most of the records given were recorded at the conventional paper speed of 25 mm per second, so that the large squares on the records represent one-fifth of a second. Some were recorded at a paper speed of 50 mm per second and this is indicated in the legend as 'time markings one-tenth of a second'.

I am deeply grateful to many colleagues and friends who have encouraged me in writing this book and who have helped by allowing me to use their illustrations. I am particularly grateful to Dr D. Scherf and Dr A. Schott for allowing me to use *Figure 5.20b* and to Dr Schott for *Figure 9.7*. I am very grateful to Professor R. Hudson for *Figure 1.1*, and to Dr L. Schamroth for *Figure 5.20a* and *Figure 7.4*. My thanks are also due to Dr Evan Fletcher, Dr Clifford Parsons, Dr A. Hudson and Dr R. Gibson for allowing me to reproduce some of their records. The photograph illustrated in *Figure 1.4* was taken by Mr E. Bailey of Imperial Chemical Industries.

My particular thanks are due to my secretary, Mrs O. Adams, not only for typing the manuscript, but also for her unfailing help in many onerous tasks associated with the preparation of this book.

J. P. P. STOCK

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CHAPTER 1

ANATOMY AND PHYSIOLOGY IN RELATION TO ARRHYTHMIAS

ANATOMY OF THE 'SPECIALIZED' TISSUES OF THE HEART

In addition to the purely contractile muscle fibres composing the atria and ventricles, the heart possesses certain 'specialized' tissues, the primary functions of which are the genesis and conduction of impulses. These specialized tissues consist of the sino-atrial (S.A.) and atrioventricular (A.V.) nodes, the bundle of His, with its left and right main branches, and the Purkinje network of fibres in each ventricle. Although each of these structures is macroscopic in size, they are none of them readily identifiable by the naked eye, and histological or chemical techniques are necessary to demonstrate them. It is curious that historically these structures were discovered in the reverse order in which they are normally activated during the cardiac cycle. The terminal elements of the system, the conduction networks in the ventricles, were first described by Purkinje in 1845, although he did not appreciate their function. The A.V. bundle was identified and described by His, Jnr., in 1893. The A.V. node was first described by Tawara in 1906, although perhaps his most important contribution was the demonstration that the A.V. node, bundle of His and the Purkinje networks formed one continuous structure linking the atria to all parts of the ventricles. The sino-atrial node, the primary pacemaker of the heart, was the last of the specialized tissues to be identified by Keith and Flack in 1907. It is now well established that these specialized tissues are essentially muscle fibres, although they differ in histological detail from ordinary atrial and ventricular muscle cells.

Until comparatively recently, it was generally thought that no special pathways existed in the atrium, linking the sino-atrial and atrio-ventricular nodes. It was considered that the sinus impulse spread radially over the atrial myocardium to reach the atrio-ventricular node and the left atrium in a non-selective manner. However, in the past few years increasing evidence has accumulated that there are in fact three specialized pathways in the atria for rapid conduction of a sinus impulse, both to the left atrium and to the atrio-ventricular node. James (1963) has called these the internodal tracts.

The old controversy between the myogenic and the neurogenic views of the conduction of the cardiac impulse is now of only historical interest. Recent studies, both of morphology by the electron microscope and of electrophysiology by modern techniques, have established beyond doubt that the genesis and transmission of impulses in the heart are solely effected by muscular structures.

Although the specialized tissues are macroscopic in size, they are difficult to differentiate from the surrounding myocardium with the unaided eye. The most certain and consistent method of showing them is by taking

histological sections of appropriate blocks of the myocardium. The histological appearance of both the sinus and A.V. nodes is closely similar. The muscle fibres are smaller than those of the neighbouring atrial musculature. They are striated, but the striations are difficult to see unless stained with silver preparations. The most characteristic feature of nodal tissue is that the fibres are branched and interwoven to form a complex three-dimensional network (*Figure 1.1*). In section, therefore, some of them are cut transversely and others at varying angles. At the periphery the fibres tend to run more vertically forming an incomplete border to the node. The sinus node is fairly clearly demarcated from the surrounding tissues by being embedded

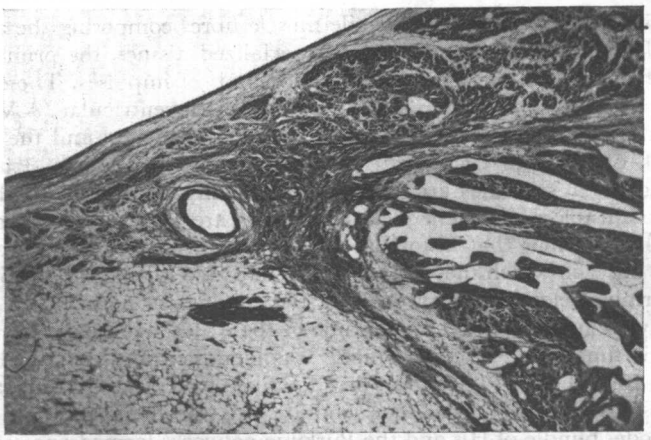

Figure 1.1—Low-power view of human sinus node, stained by Holme's silver method × 22. The sinus node is seen disposed round its nutrient artery. Reproduced from The Human Pacemaker and its Pathology by R. E. B. Hudson by courtesy of Edward Arnold

in a matrix of fibro-elastic tissue. The A.V. node contains rather less collagenous tissue than the sino-atrial node. Both nodes are richly supplied with autonomic nerve fibres and blood vessels.

The muscle fibres of the bundle of His, its left and right branches, and the Purkinje network in the ventricles all show the same basic structure, although differing in minor details. The cells tend to be arranged in parallel with only occasional interconnecting branches. They are larger and paler staining than the cells of the common myocardium and are arranged in staggered fashion. The individual cells are approximately 100–200 μm in length. They tend to be arranged in groups to form fibres. Each fibre contains from two to seven cells, the membranes of which are in direct contact, the group being surrounded by a basement membrane which does not penetrate between the individual cells.

Until comparatively recently it had always been believed that there was protoplasmic continuity between myocardial cells, so that the whole heart could be regarded as a syncytium. Electron microscopic studies, however, have established that the intercalated discs are primarily formed by the cell membranes of two fibres in end-to-end contact, which separates the protoplasms of the two cells completely.

Gross Anatomy of Specialized Tissues

Sino-atrial Node

The sino-atrial node is the primary pacemaker of the heart. It is situated in the upper part of the right atrium, close to its junction with the superior vena cava and close to the sulcus terminalis. It is usually described as being about 25 mm in length, having a head, body and tail, and with its long axis running backwards, downwards and to the left. However, Hudson (1960) carefully examined the sino-atrial node histologically in 65 human hearts and gave a very clear description of its normal anatomy. On the antero-lateral aspect of the junction of the superior vena cava with the right atrium,

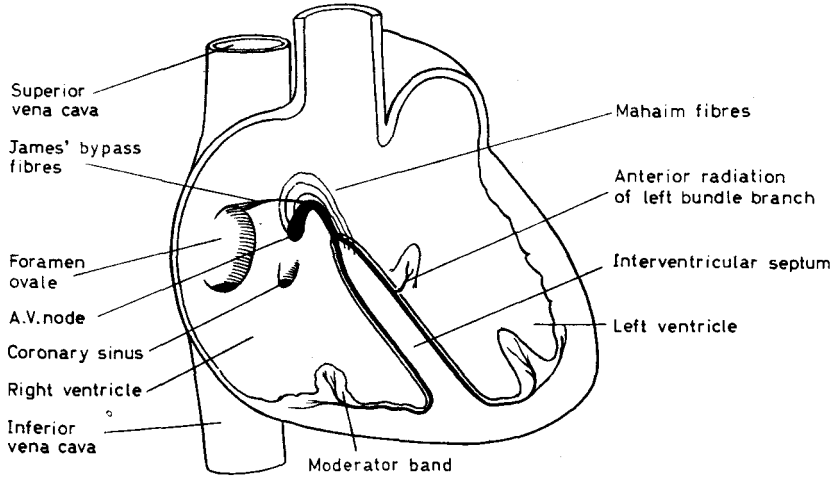

Figure 1.2—Diagram of A.V. conduction system of human heart

the latter rises to a well defined summit which is constantly present. This summit is the landmark for the node, the main part of which lies just below it, immediately beneath the epicardium. He describes the normal average human node as a crescentic structure, measuring 3–4 mm at its widest part and tapering medially and laterally to a point, the long axis lying virtually transversely. The nodal tissue is usually disposed round its central artery, and there is no overlying atrial muscle. Although it lies immediately beneath the epicardium, it is rarely discernible to the naked eye but readily demonstrated in histological sections.

The Internodal Tracts

The anatomy of the three internodal tracts in the atria was clearly described by James (1963) and his findings have been largely accepted by other workers. He called them the anterior, middle and posterior internodal tracts and pointed out that each had previously been described; the anterior tract was partially described by Bachmann (1916), the middle tract by Wenckebach (1907) and the posterior tract by Thorel (1910). The delay

in accepting the existence of these specific fast-conducting internodal pathways was probably largely due to the fact that although they contain numerous Purkinje fibres, they are not exclusively composed of such specialized cells. Nevertheless, a direct fibre-to-fibre connection has been established in all three pathways, between the sinus and A.V. nodes. The anterior internodal tract arises from the anterior part of the sinus node and passes to the left round the superior vena cava to enter the anterior interatrial

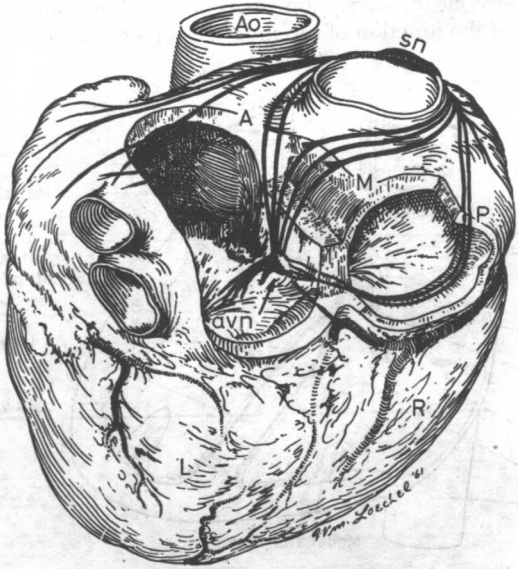

Figure 1.3—The internodal tracts. The anterior tract (marked A) is seen to divide into two. The upper fibres pass in the interatrial myocardial band to reach the left atrium. They are often referred to as Bachmann's bundle. The posterior internodal tract (marked P) is the longest of the three tracts and may bypass the major portion of the A.V. node (marked A.V.N.) The terminal portion of these fibres is often referred to as James' bypass tract (after James. Reproduced by Courtesy of Dr. T. James and 'Diseases of the Chest')

myocardial band which was first described by Bachmann. Bachmann was only interested in interatrial conduction and he did not note that his tract divided into two parts, the first going on to the left atrium and known as Bachmann's bundle, and the second group of fibres descending anteriorly in the interatrial septum to reach the A.V. node (*Figure* 1.3). The middle internodal tract originally described by Wenckebach also descends in the interatrial septum to reach the A.V. node. The posterior internodal tract originally described by Thorel terminates with most of its fibres bypassing the bulk of the A.V. node and joining the lower part of the node. The lower part of this tract is often referred to as James' bypass tract.

In the experimental animal hyperkalaemia may produce atrial paralysis yet sinus rhythm is maintained via the internodal tracts although no P waves appear in the electrocardiogram.

The remaining 'specialized' tissues of the heart may be viewed as a single continuous structure linking the atria to all parts of the ventricles and containing the subsidiary pacemakers of the heart (*Figure 1.2*). It consists of the A.V. node which is continuous with the main bundle of His. This divides into left and right main branches, which ramify in their respective ventricles to form the peripheral conduction pathways, the ultimate twigs of which probably penetrate from endocardium to epicardium, terminating in the fibres of the common ventricular myocardium. In the normal heart, the atria and ventricles are otherwise completely separated from each other by the two fibrous A.V. rings and this specialized pathway forms the only link between them over which an impulse can pass from one to the other.

Atrioventricular Node

The A.V. node lies on the right side of the lower part of the interatrial septum, just in front of the opening of the coronary sinus and above the attachment of the septal cusp of the tricuspid valve to the right A.V. ring. Unlike the sinus node, it is possible, with practice, to dissect it macroscopically from the surrounding musculature, and its continuity with the main bundle of His can be demonstrated. The macroscopic appearance of the A.V. node has been compared to a flask, measuring approximately 6 × 3 × 2 mm. The 'neck' of the flask is continuous with the bundle of His, which is narrow in comparison, having a diameter of only 2–4 mm.

In addition to these bypass fibres of James, Mahaim (1947) described fibres linking the A.V. node directly to the interventricular septum. He termed these 'preferential' or 'paraspecific' fibres. Their existence, at least in some hearts, has been confirmed by Lev and Lerner (1955). The precise function of these fibres is also uncertain, and they may be responsible for the delta wave in some cases of the Wolff–Parkinson–White syndrome (page 139).

Theory of Kent

In 1893 Kent first described direct muscular connections between the atria and ventricles in some newborn animals. In a later series of papers in 1913 and 1914 he suggested that conduction between the atria and ventricles was via a series of muscular bridges linking the lateral walls of the atria to the ventricles. Since that time many workers have searched for the muscular bridges described by Kent without success. It is possible that such connections occasionally exist and may explain some examples of ventricular preexcitation (Wolff–Parkinson–White syndrome), but it is now certain that in the vast majority of mammalian hearts, the A.V. node and bundle of His form the only link between the atria and ventricles.

Bundle of His

The main bundle of His is approximately 20 mm in length and penetrates the central fibrous body reaching the top of the muscular interventricular septum, where it divides into its left and right main branches.

In the terminology of arrhythmias, it is convenient to refer to the A.V. node and the main bundle of His as the 'A.V. junction'.

Right Main Branch

The right main bundle passes down the right ventricular side of the muscular septum towards the apex. It gives off few branches at first and lies rather more deeply beneath the epicardium than does the left main bundle. It then runs in the free edge of the moderator band to reach the base of the anterior papillary muscle where it breaks up into a network to supply all parts of the right ventricular musculature.

Left Main Bundle

The left main bundle emerges on the left ventricular side of the septum just below the posterior cusp of the aortic valve. As it passes down the septum, branches penetrate and ramify in the substance of the septum. At the junction of the upper and middle third of the septum, it divides into an anterior and a posterior branch, which pass to the bases of the corresponding papillary muscles. Each branch breaks up into a complex network, the fibres of which run in the trabeculae lining the ventricle, particularly the 'false tendons', and penetrating branches reach the sub-epicardial muscle fibres.

The A.V. node, the bundle of His and the Purkinje networks in the ventricles are often referred to collectively as the conduction system.

Blood Supply of Specialized Tissues

The blood supply to the S.A. node is by a special artery, which in 60 per cent of hearts is derived from the right coronary artery, and from the left main coronary in the remaining 40 per cent. There is a rich anastomosis with neighbouring vessels.

The blood supply to the A.V. node is generally from a specific artery, the ramus septi fibrosi, which is a branch of the right main coronary artery in 92 per cent of hearts, and from the left circumflex in the remaining 8 per cent.

Nerve Supply

Both the S.A. and the A.V. nodes are richly supplied with sympathetic and parasympathetic nerve fibres. The parasympathetic fibres to the sinus node are derived from the right vagus and those to the A.V. node from the left vagus.

Embryology

The S.A. node is derived from the right horn of the sinus venosus and the A.V. node from the left horn. Morphologically, therefore, the sinus node is a right-sided and the A.V. node a left-sided structure, which accounts for their different vagal supply.

Direct Visualization of the Peripheral Conduction System

Under certain circumstances, it is possible to visualize the peripheral conduction system in the left ventricle where the system lies immediately beneath the endocardium. In the sheep's heart, the conduction fibres are enclosed in a continuous sheath, and this arrangement enables them to be

displayed by injecting indian ink into the sheath. No such sheath is present in human, canine or ox hearts. A characteristic feature of the conducting tissues is their high content of glycogen. This enables fresh specimens to be selectively stained by various techniques. The simplest method of demonstrating the Purkinje system in the left ventricle is to flood it with Lugol's iodine. *Figure 1.4* shows the left main bundle and its branches in the left ventricle of a dog's heart displayed in this way. Unfortunately, application of this technique is limited to fresh specimens, for glycogen rapidly undergoes post-mortem autolysis and ceases to stain after about two hours. Nevertheless, the human left main bundle and its branches has been demonstrated in this way when an autopsy has been possible $1\frac{1}{2}$ hours after death (Uhley and

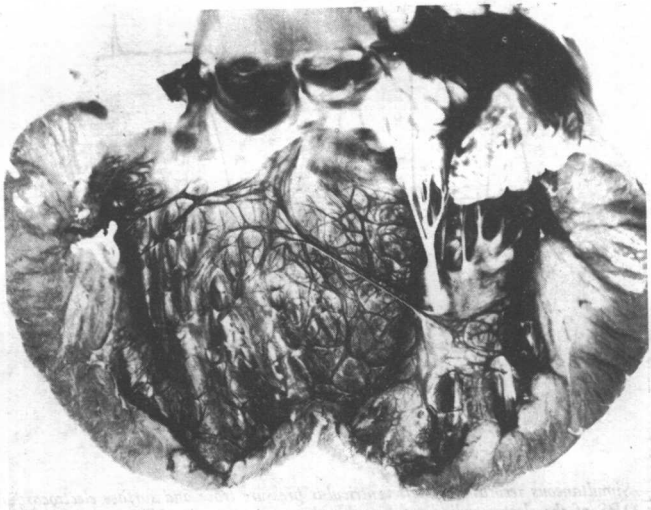

Figure 1.4—Distribution of left bundle branch in the dog, stained by Lugol's iodine. The division of the left main bundle into its two chief branches is clearly seen and takes place at a rather higher level on the septum than it does in the human heart

Rivkin, 1959, Spach and colleagues, 1963). The chance of success is much greater if death occurred suddenly; when death was more gradual, antemortem autolysis of glycogen may occur. It is possible that this accounts for the bizarre conduction anomalies seen in the electrocardiogram of the 'dying' heart. Although at first sight this technique looks promising, it clearly has very limited applications and would probably prove valueless for demonstrating pathological lesions.

The most refined and accurate technique for studying the specialized tissues of the heart is that evolved by Lev, Widron and Erickson (1951). The method is painstaking, laborious and time consuming, for it involves the cutting and histological examination of over 3,000 serial sections from a single heart. The authors rightly point out that this is essential to determine the site and nature of any pathological lesions in the conduction system which may be correlated with the electrocardiographic findings in life. However, at present, this technique is necessarily restricted to the dedicated

expert. Even so, it has already made significant contributions to our knowledge of the anatomy and functions of the specialized tissues.

CARDIAC PHYSIOLOGY IN RELATION TO ARRHYTHMIAS

The clinical electrocardiogram, which is recorded from the body surface, registers the electrical changes produced by the spread of the excitation wave over the heart. The genesis of these electrical changes will be considered later under the heading Electrophysiology. The excitation wave may be regarded as the trigger which releases the contractile forces of the heart, and therefore the onset of the electrocardiogram precedes the onset of contraction. *Figure 1.5* shows a simultaneous recording of the electrocardio-

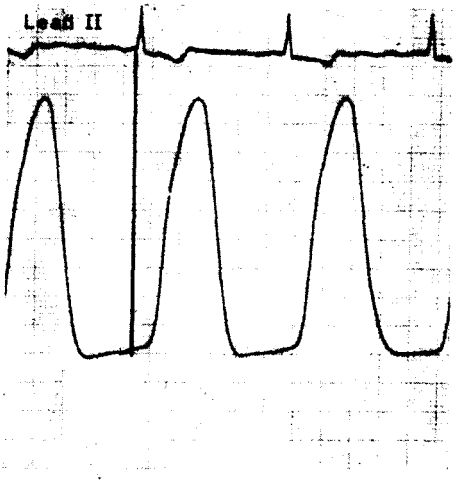

Figure 1.5—Simultaneous recording of left ventricular pressure trace and surface electrocardiogram. The onset of the QRS of the electrocardiogram precedes the commencement of left ventricular systole by 0·12 second. Part of this delay is an artefact

gram and the left ventricular pressure pulse. The onset of the QRS complex precedes the rise in left ventricular pressure by 0·12 second. While part of this time interval is an artefact due to delay in transmission of the pressure wave down the catheter, accurate simultaneous recordings show that the electrical changes in the heart precede the mechanical changes. In fact, the electrocardiogram tells us nothing about the contractile events which follow. Occasionally, during angiography, the heart has been seen to stop while the electrocardiogram continued unchanged. On the other hand, careful analysis of the electrocardiogram almost always enables the site of origin of the excitation wave to be recognized and its pathway of conduction to be deduced. All disorders of cardiac rhythm are primarily due to disturbances either of the origin or conduction of the excitation wave, and for this reason the electrocardiogram reigns supreme in the analysis and interpretation of arrhythmias.

An appreciation of some of the fundamental physiological properties of heart muscle is essential for an understanding of the mechanisms underlying

many disorders of cardiac rhythm. The description of cardiac physiology which follows is in no sense intended to be comprehensive but will be mainly restricted to those properties of heart muscle concerned with the formation and propagation of impulses. It is conventional to describe five fundamental physiological properties of heart muscle, namely excitability, conductivity, refractoriness, rhythmicity and contractility. In addition, the intact heart possesses remarkable powers of adaptation to changing conditions. These powers, which are independent of nervous control, have been termed, by Sarnoff and his colleagues (1960), homeometric responses.

Excitability

Heart muscle is excitable in that it responds to a variety of natural and artificial stimuli by contracting. The response of the heart to artificial stimuli obeys the 'all or none' law. This was first observed in 1884 by Bowditch in the frog ventricle. Using induction shocks as stimuli, he found that once the strength of a stimulus reached threshold level, it immediately evoked from the ventricle the maximum response of which it was capable at that moment. Further increase in the strength of stimulus did not increase the strength of the resulting contraction. His findings have been fully confirmed in mammalian heart muscle. The degree of excitability of heart muscle may be assessed by the strength of stimulus necessary to produce a response. As will be seen when the refractory period is discussed, this varies throughout the cardiac cycle. Excitability is at its peak during the non-refractory period, but the level of this peak may vary under different conditions. It is increased by such factors as CO_2 retention, or by circulating catecholamines and by numerous drugs. The 'irritability' of the heart is also profoundly influenced by electrolyte disturbances, particularly of potassium.

Conductivity

Conductivity is that property of heart muscle whereby activation of any individual muscle fibre automatically initiates activity in neighbouring muscle cells. In consequence, if an adequate stimulus is applied at any point in cardiac tissue during its resting phase, an 'excitation wave', that is, a wave of electrical activity, will be propagated over the whole of the tissue without decrement. This property of conductivity is unique to cardiac muscle and visceral smooth muscle; it is not possessed by skeletal muscle. It used to be thought that the myocardium was a syncytium, so that the whole intact heart could be regarded as functioning as a single muscle cell. If this were true, it would of course explain conductivity; however, recent electron microscopic studies of the myocardium have established that the individual muscle fibres are separate anatomical units, each measuring approximately $100 \times 15 \times 15\ \mu m$. The precise mechanism by which the excitation wave is propagated from fibre to fibre (without innervation) has not yet been firmly established. There appear to be two possible mechanisms; one is that propagation is mediated by local electrical currents from cell to cell. This would imply that the electrical resistance of the myocardial cell membrane is low. The alternative mechanism is that propagation of the impulse is chemical via synaptic junctions between individual fibres.

Conductivity is a property possessed by all types of heart muscle. Under

'normal' conditions, if a stimulus arises (or is induced) in any part of the heart, an excitation wave will spread over the whole heart, provided all parts are in the non-refractory phase. In principle, this is true wherever the stimulus originates. In normal sinus rhythm, the excitation wave originates in the sino-atrial node and is conducted over the heart to activate the various parts of its four chambers in a definite orderly sequence which is precisely repeated from beat to beat. The sequence and timing of activation of the individual parts of the heart is so arranged as to ensure that the maximum mechanical efficiency results from their ensuing contraction. This is largely achieved by the special conduction system already described. Although conductivity is a property possessed by all myocardial cells, the speed of conduction varies appreciably in different types of heart muscle.

TABLE 1.1

Speed of Conduction in Mammalian Heart Muscle	*mm/second*
Atrial muscle	1,000
Ventricular muscle	400
Purkinje fibres	4,000
Atrioventricular junction	200

In the mammalian heart, the speed of conduction in atrial muscle is 1,000 mm/second. In ventricular muscle, it is substantially slower, measuring only 400 mm/second. Conduction is fastest in the Purkinje network where it is 4,000 mm/second. It is slowest of all in the A.V. junction where it averages only 200 mm/second. There is an important teleological reason for this; one of the main functions of the A.V. junction in sinus rhythm is to delay the transmission of the excitation wave to the ventricles in order to ensure that atrial systole is complete before ventricular systole begins. Most of the P–R interval is occupied by this slow transmission of the impulse through the A.V. junction. The importance of the length of the P–R interval has recently been elegantly demonstrated by Gillespie and colleagues (1967). They devised a method for measuring the right ventricular stroke output of successive beats. Very briefly, the technique consists of enclosing a subject in a whole body plethysmograph while breathing a weak mixture of nitrous oxide in air. The plethysmograph records the volume of gas absorbed from the lungs following each right ventricular systole and thus enables the volume of blood ejected at each systole to be calculated. They studied patients with congenital complete heart block, in whom the interval between atrial and ventricular systole was continuously varying. They found that when the P–R interval measured 0·2 second, the right ventricular stroke output was 30 per cent greater than when atrial and ventricular systole coincided, that is, when the P–R interval was zero. Similarly, during right heart catheterization, it is not uncommon for transient A.V. dissociation or nodal rhythm to be induced. When right atrial and right ventricular systole coincide, there is always an abrupt fall in right ventricular systolic pressure compared with when the time relationship of the contraction of the two chambers is normal. This pressure change is more marked when the right ventricle is under an increased load, that is, in pulmonary hypertension or pulmonary stenosis.