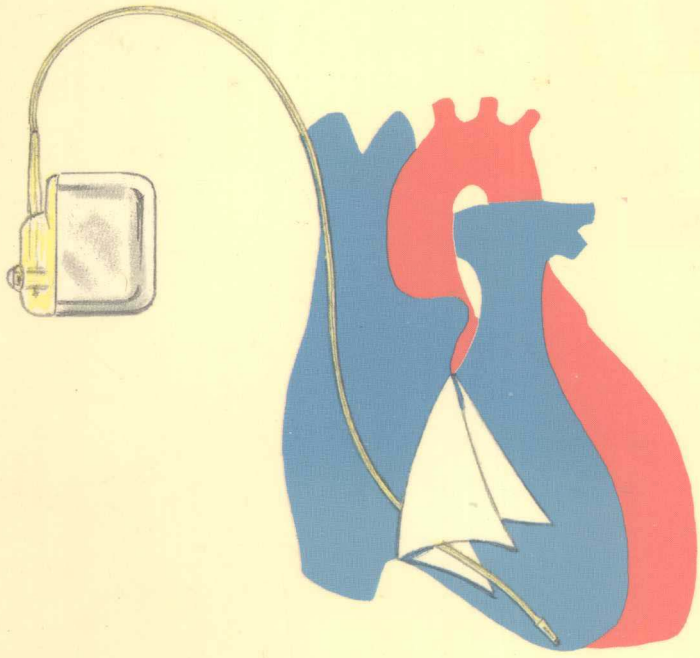
THE TARDIEU SERIES

the essentials in cardiac pacing

an illustrated guide



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FOREWORD

The classic medical textbook tends to be overly technical, excessively detailed, profusely referenced, and the antithesis of enjoyable reading. With the expectation of many hours of hard work, I was unprepared for the pleasure that lay ahead.

This book is what we in the United States call a « sleeper ». Without pomp or solemnity it captures you with a light-hearted style that subtly belies its sophistication. The authors have indeed mastered the art of simplicity, combining profound knowledge with an airy format, to a degree that is hard to emulate. All the salient features of pacing are presented here, from history and pathology to complications and long-range follow-up. Perhaps it is a mark of excellence, rather than a confession of my personal ignorance, to say that there is scarcely a section that did not provide me with a new bit of information or a new insight.

Permanent pacing of the heart is so common nowadays, at least in the more affluent sections of the world, that almost every person must know of someone with an implanted pacemaker. In the United States, where there are more than 100,000 new implants each year, almost every sizeable hospital has a pacemaker implantation service and almost every physician in a related field is interested in doing this surgery. All that should really be required is that the surgeon make himself an expert. This book, if thoroughly understood, will provide the expertise, and reading it will be a labor of love. The illustrations are laced with humor and charm, and the text will appeal to almost any reader.

After reviewing the first edition I had urged the authors to prepare an American version because I believed the book to be extremely valuable not only to the beginner in pacing, but to the more experienced physician as well. This second edition has added new illustrations and information on the most recent technical advances, and there is also an expanded reference list. The authors present solid reasoning for all of their concepts and methods of practice, and give food for thought even to those whose opinions may differ slightly. It is my hope to see still later editions of this delightful book in years to come.

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PREFACE

A pacemaker is an implanted electronic device which takes over command of the cardiac rhythm when the natural mechanism fails. In practical terms, it consists of a battery, and an electronic circuit which transforms the continuous electric current into short electrical impulses. The rhythm of the myocardium will be that imposed by these impulses which are therefore regulated at 70-80/min (lasting about 1/1000 second each with an amplitude in the order of several volts). The whole device is enclosed in a sealed case made out of metal or epoxy-resin. The electrical impulses are conducted to the heart muscle by isolated electrodes and depolarisation of the heart follows the minute electrical discharge which takes place at the tip of the electrodes. **Depolarisation** leads to contraction of the ventricular muscle.

The main use of the pacemaker has been in the treatment of Stokes-Adams' syndrome, a permanent slow pulse or atrio-ventricular block, the prognosis of which has been completely changed. Formerly, half the patients with this disease died within a year of diagnosis. Nowadays, with a pacemaker, the expectation of life of these patients is practically the same as for people of the same age without the disease.

Pacemakers have been used in the treatment of other cardiac irregularities giving rise to syncope and even for symptomatic simple bradycardias. Recent uses include the treatment of tachycardia-bradycardia syndromes and prevention of certain tachycardias.

A pacemaker was first implanted in man in 1958. V. Parsonnet during the Vth International Symposium in Tokyo (1976) stated that there are now about 156,000 patients with permanent pacemakers in the United States alone and we estimate the world population of pacemaker patients to be about 300,000. The therapeutic results achieved by these devices are among the most spectacular of modern medicine. Pacemakers are in certain cases, the only means of maintaining human life from second to second throughout many years.

PHYSIOLOGY OF THE INTRACARDIAC CONDUCTION SYSTEM

The structure and function of cardiac muscle is unique. It is striated muscle, not directly under control of the central nervous system and quite independant of voluntary control. It does not tetanise. It has an inherent automatism and contracts in a constant predetermined manner.

These singular properties are better understood by study of the individual myocardial cells. Recent advances have increased our knowledge of the physiology and pathology of the intracardiac conduction system, the essentials of which are outlined below.

A) CELLULAR ELECTROPHYSIOLOGY

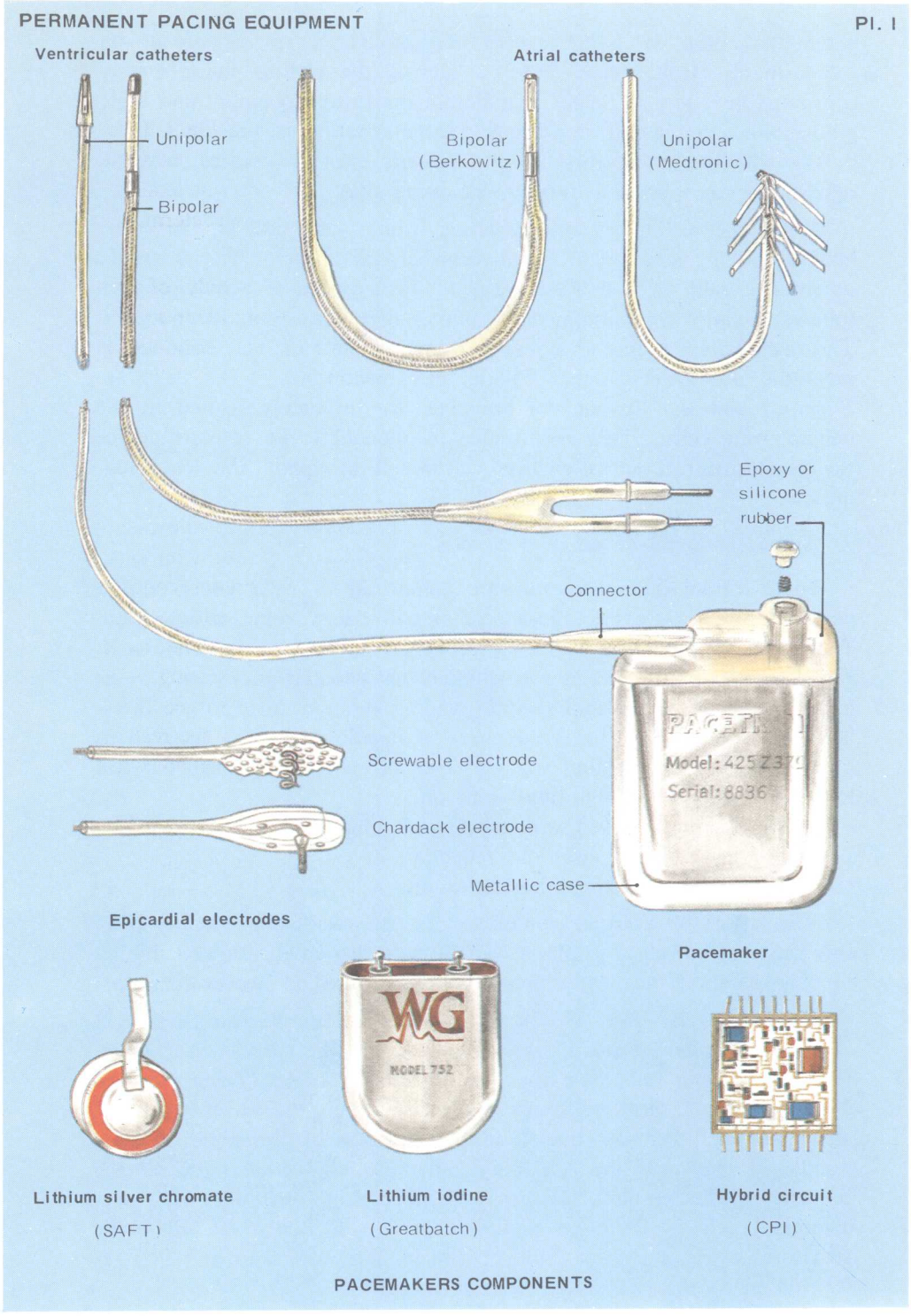
1. Resting potential

Each myocardial cell is enclosed in a lipo-protein cytoplasmic membrane (Plate II, p 12). The ionic concentrations are different on each side of this membrane. In the resting state, there is a higher concentration of potassium ions inside the cell than in the interstitial fluid. Sodium ions are more abundant in the interstitial fluid. The **ionic gradients** across the cell membrane are responsible for the resting electrical potential of the cell, the exterior being positively charged with respect to the interior. If a micro-electrode is introduced into the resting myocardial cell, a potential of — 90 millivolts will be recorded with reference to the interstitial fluid.

The myocardial cell is **excitable,** that is to say that if a mechanical, chemical or electrical stimulus is applied at a site on the cell membrane, the polarisation will be disturbed and this change of polarisation will be propagated all along the cell membrane. The external charge becomes negative and the internal positive. This change in potential may be recorded with a micro-electrode producing a characteristic trace known as the **action potential.**

2. Action potential

Activation of the resting myocardial cell suddenly changes the internal potential from — 90 millivolts to + 20 millivolts. This is the phase of rapid depolarisation (Phase 0). At the same time, ionic transfers occur with a sudden influx of sodium ions into the cell and an efflux of potassium ions from inside the cell. This is followed by a plateau phase (Phases 1 and 2), attributed to a slow flow of sodium and calcium ions into the cell. A recovery phase with a return to the resting potential of — 90 millivolts which remains stable until the next depolarisation then occurs (Phase 4). Repolarisation to the resting


PERMANENT PACING EQUIPMENT
Pl. I
Ventricular catheters
Atrial catheters
Unipolar
Bipolar
Bipolar
(Berkowitz)
Unipolar
(Medtronic)
Epoxy or
silicone
rubber
Connector
Screwable electrode
Chardack electrode
Metallic case
Model: 425 Z 37
Serial: 8836
Epicardial electrodes
Pacemaker
WG
MODEL 752
Lithium silver chromate
(SAFT)
Lithium iodine
(Greatbatch)
Hybrid circuit
(CPI)
PACEMAKERS COMPONENTS

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potential (Phase 3) is an energy consuming process dependant on Adenosine 5'-Triphosphate (ATP). During the resting phase, energy consumption is just sufficient to maintain the resting potential and ionic gradients across the cell membranes. Depolarisation on the other hand, during which the transmembrane potentials even out seems to be a passive phenomenon and does not consume energy.

When the resting potential changes from — 90 millivolts to — 60 millivolts, depolarisation of the myocardial cell occurs. The **threshold potential** is said to have been attained. Propagation of activation then follows a pathway determined by the geometric position of the cells. The myocardium seems to act as a syncytium but, in fact, each cell is separated one from another by the cell membranes. The cells are Y-shaped and the tips of the branches are intimately related to the neighbouring cells. This arrangement is thought to be responsible for the easy transfer of activation from cell to cell throughout the ventricular muscle.

3. Action potential of pacemaker cells

Some intracardiac structures are comprised of cells whose action potentials are different to those observed in the working myocardium. Their rate of depolarisation is slower, there is no plateau phase and, above all, the time between two spontaneous depolarisations appears as a long phase of **slow depolarisation** leading spontaneously to the threshold potential. Then the rapid phase of depolarisation is observed as in the working myocardium. In this type of cell, "automatism" is said to exist. This property is dependant on:

—The slope of the slow depolarisation phase;
—The level of the threshold potential;
—The level of maximal repolarisation.

The whole intracardiac conduction system varies only slightly from this pattern from place to place. These cells also have a slowed rhythm of depolarisation the nearer they are to the working myocardium.

The sino-atrial node (Keith and Flack) has the fastest rhythm at 70/min and under normal circumstances acts as the cardiac pacemaker. If the S-A node fails then the atrio-ventricular node (Tawara) takes over command (these cells are situated between the atrium and the atrial roots of the A-V node and the junction of the node and the bundle of His). If the A-V node fails, the conductive tissue of the branches of the bundle of His or even the Purkinje cells may take over command. It is therefore apparant that a **hierarchy** of automatic structures exists, the most rapid of which takes command and imposes its rhythm on the rest of the heart muscle.

4. Excitability of the myocardial cell

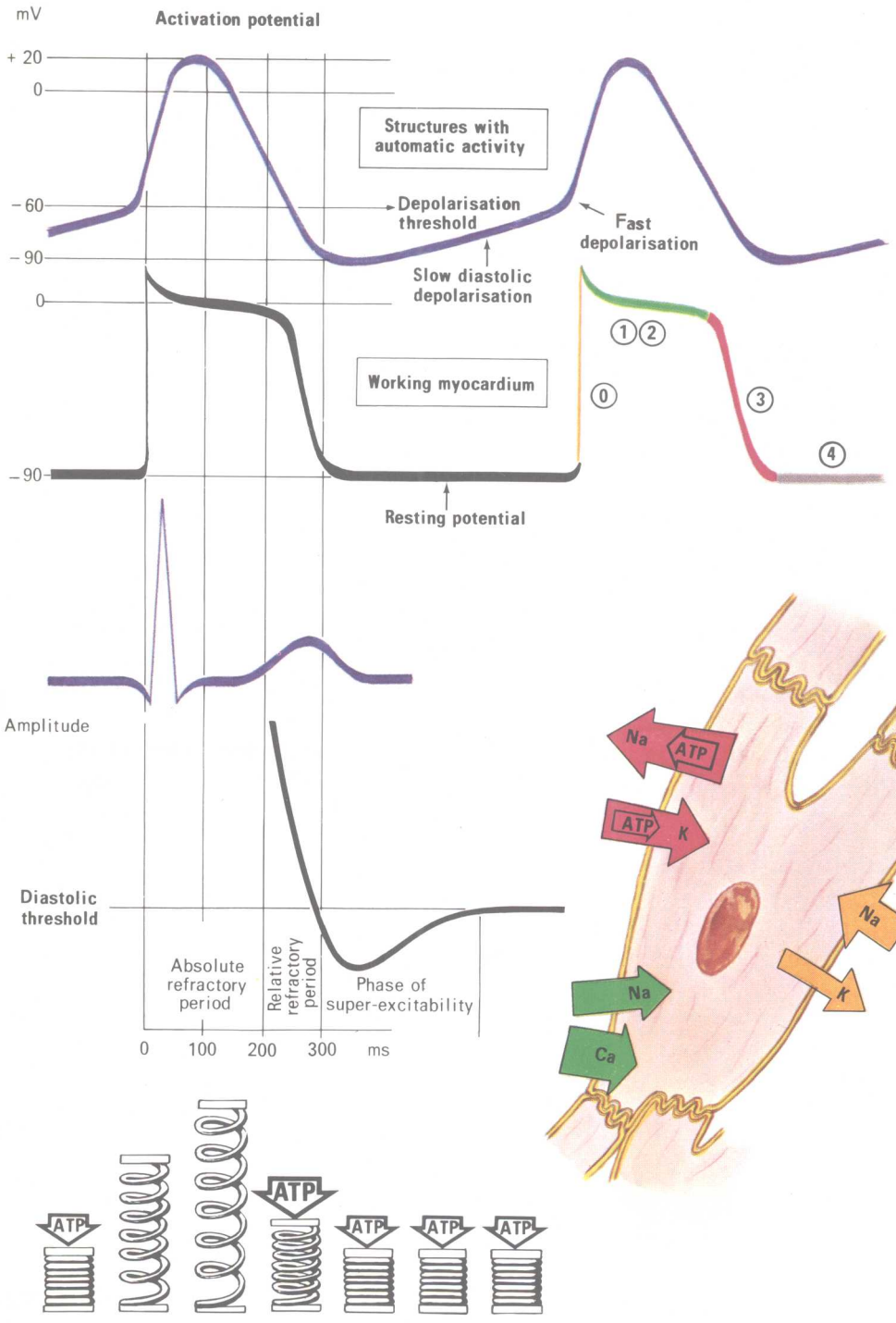
If the excitability of a myocardial cell is studied using an electrical stimulus just strong enough to depolarise the cell (threshold stimulus) the amplitude of the stimulus needed to obtain activation remains constant during the interval between two depolarisations of the same cell. This is called the **diastolic threshold.** If the stimulus is applied earlier in the cycle, near the end of the action potential, a depolarisation may be obtained with a weaker stimulus. This is known as the **super-normal phase.** It is situated near the end of the T-wave of the surface ECG. If the stimulus is advanced even earlier, that is to say during the repolarisation phase, it is still possible to obtain depolarisation but with a stronger stimulus. This is the **relative refractory period** which corresponds to the summit of the T-wave. If a strong stimulus is applied during this period, the myocardial cells, at different stages of repolarisation may be activated in an asynchronous fashion leading to ventricular fibrillation.

Stimulation earlier still in the cycle is unable to elicit a response as it falls in the **absolute refractory period.**

B) INTRACARDIAC CONDUCTION

The cardiac rhythm is normally determined by the **sino-atrial node**, that is to say the conductive tissue with the fastest automatic activity. The node is situated in the upper part of the right atrium near the point of entry of the superior vena cava (Plate III, p 16).

After depolarising the atrium, the activation arrives at the **node of Tawara** or atrio-ventricular (A-V) node on the opposite side of the atrium. This is the first part of the atrio-ventricular conduction system. Its structure is unique, the histology resembling an intermediate between cardiac muscle and nervous tissue. It is separated from the neighbouring myocardium by a band of collagen tissue. The A-V node is situated on the right side of the inter-ventricular septum more or less parallel to the site of insertion of the septal leaflet of the tricuspid valve. The time delay for the activation to arrive at the A-V node from the S-A node is in the order of 70 ms. At this point, the activation is suddenly slowed, taking about 60 ms to cross the 1 cm length of the A-V node. It is then conducted to the **bundle of His** which crosses the septal leaflet of the tricuspid valve and follows the crest of the inter-ventricular septum for 1·5 to 2 cm.


mV
Activation potential
+ 20
0
– 60
– 90
0
– 90
Structures with automatic activity
Depolarisation threshold
Slow diastolic depolarisation
Fast depolarisation
Working myocardium
Resting potential
① ②
0
③
④
Amplitude
Diastolic threshold
Absolute refractory period
Relative refractory period
Phase of super-excitability
0
100
200
300
ms
Na
ATP
ATP
K
Na
Na
K
Ca
ATP
ATP
ATP
ATP
ATP

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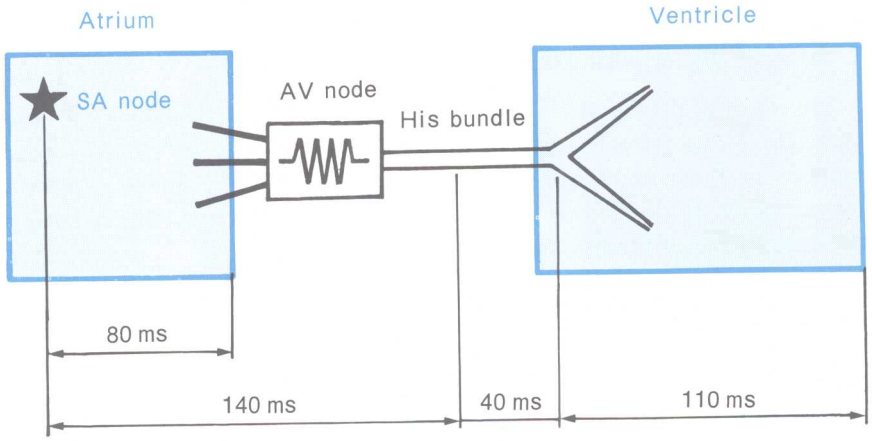
NORMAL A-V CONDUCTION TIMES

P-R:	0·12 - 0·20	s=	120 - 200 ms
A-H:	0·08 - 0·16	s=	80 - 160 ms
H-V:	0·04 - 0·055	s=	40 - 55 ms

The bundle of His divides into a long narrow right branch which activates the endocardium of the right ventricle. On the left side, the bifurcation gives rise to a wide thick left bundle which itself branches to activate the endocardium of the left ventricle. These branches are schematically represented as the **antero-superior** and **postero-inferior** fascicles. They terminate in the same manner as the right bundle, in the numerous branches of the **Purkinje system** which therefore forms the intermediate between the branches of the bundle of His and the ventricular muscle. The conduction interval between the trunk of the bundle of His and the start of ventricular activation is about 40 ms.

This pathway (A-V node, bundle of His and branches) is the only way that atrial depolarisation may normally be conducted to the ventricles as the atrial and ventricular muscles form two isolated electrical entities. Slowing of conduction at the A-V node allows time for satisfactory filling of the ventricles before their contraction. Conduction in the branches of the bundle of His is 4-5 times more rapid than in myocardial muscle so ensuring an activation sequence of the ventricles from the endocardium outwards, giving the optimal haemodynamic result.



INTRACARDIAC CONDUCTION DEFECTS

A) ATRIO-VENTRICULAR CONDUCTION DEFEC

By nature of the activation of the heart from the sinus node to the contractile ventricle, any disease process involving the A-V node, bundle of His or its branches, if sufficiently diffuse, may interrupt the conduction to the ventricle. This is the situation in **atrio-ventricular block,** a more precise term for the **Stokes-Adams syndrome.**

1. Third degree atrio-ventricular block

Third degree A-V block is said to be present when there is **no transmission** of atrial activity to the ventricle (Plate V, p 21). In the absence of the pacing rhythm of the sinus node, command is taken over by more distal cells of the conduction system of a slower automatism. The slower rhythm arises distal to the zone blocked, either in the branches of the bundle of His or in the Purkinje system. In the latter case, which is the most frequently encountered, the ventricles beat at about 40/min. This is called **idio-ventricular** rhythm and is asynchronous with the beating of the atria which continue at the rhythm of the S-A node. The rate of the idio-ventricular rhythm is half that of sinus rhythm and it is not affected by effort. Its remarkable stability led to the term **permanent slow pulse** previously applied to the Stokes-Adams syndrome. Idio-ventricular rhythm may be considered as a safety device but is itself susceptible to failure. In this case, another more distal group of cells may take over command at an equal or even slower rate and with a different ECG morphology (the point of origin in the ventricle is different). If these secondary pacemakers all fail, syncopal episodes will occur. Even at a rate of 40/min, the cardiac output is reduced, causing symptoms such as shortness of breath on exertion and tiredness, and in the older persons, heart failure and diminished cerebral function.

2. Second degree atrio-ventricular block

In second degree A-V block, **some P-waves** are conducted to the ventricle. The term is qualified 2/1, 3/1, 4/1, etc., according to the number of P-waves required to obtain a ventricular response. Second degree block is divided into two sub-groups. Their importance lies in the significant difference in prognosis.

a) Type I (Wenckebach)

In this type of second degree A-V block, the P-R interval is frequently longer than normal. It is **not constant** from beat to beat showing a progressive lengthening until a blocked P-wave. The delay takes place in

most cases in the A-V node and it is thought that the P-wave which does not elicit a ventricular response is also blocked at this level. This is not the most dangerous type of block as the bundle of His may take over the pacing role should the process aggravate, so avoiding cardiac arrest.

b) **Type II** (Mobitz)

This type of second degree A-V block is characterised by the fact that the P-wave conducted is **constant** and followed by QRS complex after a frequently normal P-R interval. This type of block is usually situated in the bundle of His. It is associated with a poorer prognosis than nodal blocks as it may lead abruptly to complete heart block and sudden death may occur.

c) Luciani-Wenckebach period

This is a special type I second degree A-V block characterised by **periodic** lengthening of the P-R interval through several cardiac cycles until a P-wave is not followed by a ventricular response. This type of conduction defect is also generally due to a block in the A-V node.

3. First degree heart block

This is the least severe form of atrio-ventricular conduction defect in which there is a delay of conduction from the atrium to the ventricle. Each P-wave is followed by a ventricular response, the ECG showing a long fixed P-R interval.

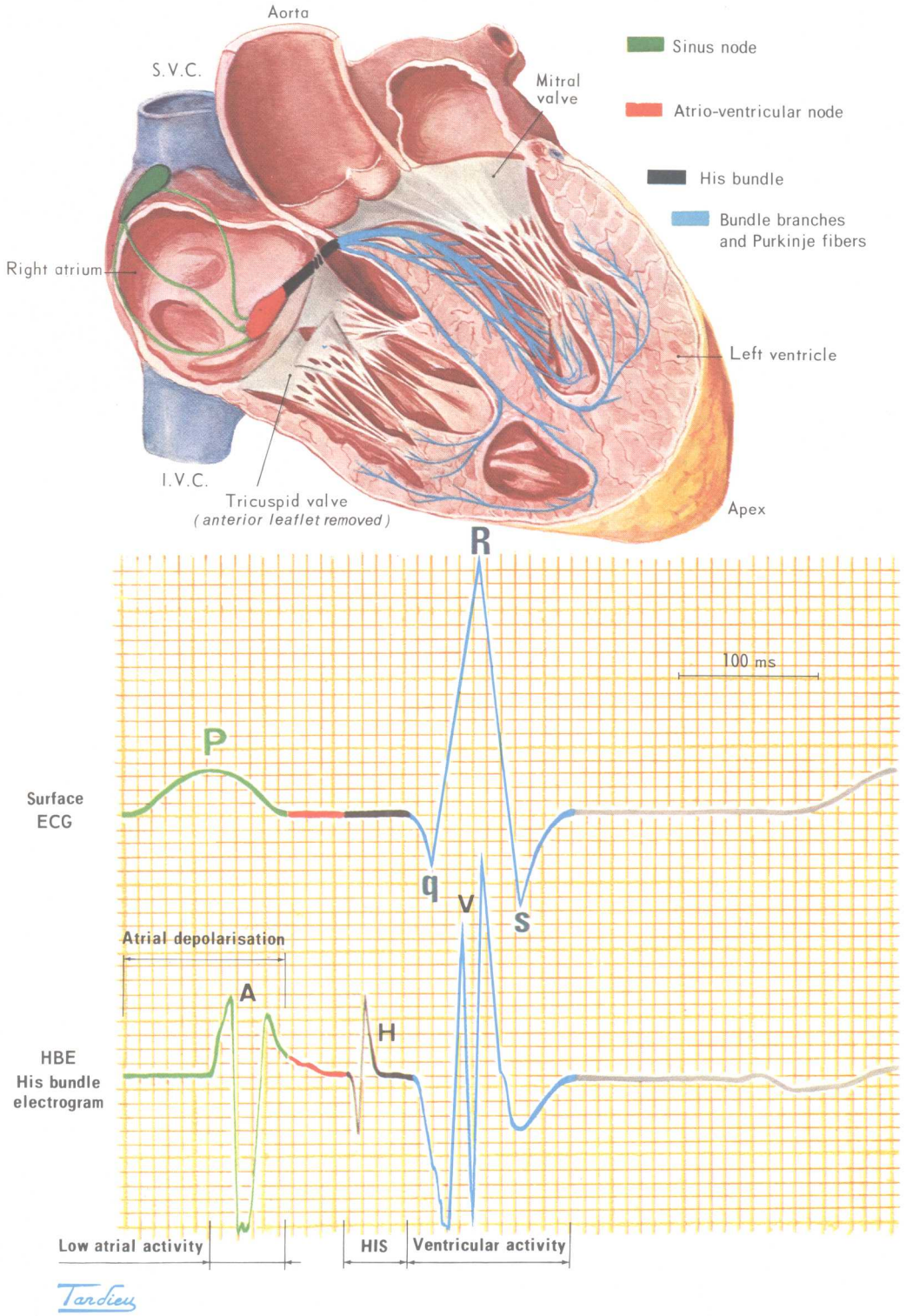
4. Clinical consequences

Atrio-ventricular blocks may be paroxistic, the **ECG being completely normal** between attacks. Great care is therefore required when faced with a patient of over 60 years of age presenting with syncopal episodes. Stokes-Adams attacks must be considered in the differential diagnosis before attributing the symptom to a neurological or functional illness. One must also be vigilant when dealing with patients presenting with symptoms varying from lightheadedness, vertigo, tinnitus and unsteadiness to "sudden turns" without loss of consciousness. In some cases, complete heart block does not present with syncope but with shortness of breath on exertion and tiredness.

Diagnosis is easily made when the pulse rate is slow and the ECG shows a high grade A-V block. However, it may be more difficult in the early stages of the illness when the ventricle is dependant or partially so on sinus rhythm. A practically normal A-V conduction may be present.

Usually, however, progression to complete block is preceeded by diffuse disease of the intra-ventricular system which gives rise to particular ECG abnormalities. It is important to be able to recognize these in order to make the correct diagnosis and consider the indications for the implantation of a pacemaker.

THE CONDUCTION SYSTEM OF THE HEART



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