DISEASES OF THE HEART AND ARTERIES

BY

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DISEASES OF THE HEART AND ARTERIES

TO

SIR JOHN PARKINSON

IN RECOGNITION OF HIS MANY CONTRIBUTIONS TO CARDIOLOGY,
HIS LEADERSHIP AND OUTSTANDING TEACHING IN THE SUBJECT,
AND IN GRATITUDE FOR HIS FRIENDSHIP

PREFACE

THE older textbooks on heart disease, and which were of substantial size, dealt with symptoms and clinical signs in the light of morbid anatomy. In that these had been assembled at the bedside and unaided by methods of examination which have since so often confirmed the rightness of their interpretation, it causes no surprise that their pronouncements should sometimes have been tinged with conjecture which led to wrong conclusions. At this distance of time, however, we continue to marvel at the accuracy of their predictions made without the accoutrements which we now instinctively summon to help us.

During the past three decades especially, our knowledge of cardiovascular disease has grown apace. This progress, remarkable both in regard to its speed and its expanse, inevitably requires many more pages than hitherto for its recording.

At the turn of the century the size of the heart was estimated haphazardly by percussion. Now, recourse to radiological examination determines the precise shape of each of the heart's chambers, while angiocardiography, if needed, will delineate them even more accurately. The significance of added heart sounds, and the meaning of certain murmurs, were then interpreted without the authority of the phonocardiograph. Now, this test has allotted to each auscultatory sign its special character, and made it possible to recognize many clinical states through attentive listening alone. The many conditions whose diagnosis yields only to electrocardiography, had once to be reckoned with in the absence of this device. Now, the addition of this test can tell hypertrophy of individual atria and ventricles, and can decipher the significance of chest pain, a function of inestimable worth. Cardiac catheterization and angiocardiography have brought precision to the diagnosis of congenital cardiac defects, while the application of surgery in their treatment has tested the accuracy of the instrumental findings. In the realm of treatment too, immense progress has taken place, medicinally as in the management of heart failure with our greater understanding of the blood electrolytes and agents capable of adjusting them, and surgically in the relief of obstruction by stenosed valves and the closure of congenital cardiac and arterio-venous shunts. The part played by autoimmunity reactions in the etiology and treatment of certain forms of heart disease has yet to be fully described, but already it is apparent that in the pursuit of its study we are entering exciting fields.

At this moment of time in the specialty of cardiology, when current views are in need of modification, some to be expanded in the light of recent research work, and others to be discarded altogether, while theories as yet unproven have to be appraised, there is laid upon the author of a textbook a trust which he must take care to discharge faithfully and responsibly. The first obligation imposed on him is to do justice to the specialty, and his next is to the reader, whether a student or practitioner of medicine, who looks for sound counsel for the readier diagnosis of heart disease and its treatment.

It is the author's intention always to emphasize the clinical approach to the subject, and to advocate the use of ancillary aids to diagnosis only when this is

imperative, when a close regard is taken of the simplicity of any laboratory test and the discomfort it might cause an already ailing patient. Extravagant and painful tests which only confirm the diagnosis made by a careful clinical examination, are spurned, but they are described for most conditions in case their use becomes obligatory in unusual circumstances.

It has been inevitable that many years spent in the practice of cardiology should have compelled the author to hold distinctive views on some problems embraced by the specialty, and these have been laid before the reader alongside the evidence upon which they have been fashioned. This applies specially to systemic and pulmonary hypertension, and to the electrocardiographic diagnosis of coronary arterial disease, subjects which have specially attracted personal research work.

In that electrocardiography is the most helpful ancillary test in the diagnosis of heart disease, particular advice and guidance have been offered on the nature and number of leads to use. Seven only are recommended and the superiority of bipolar leads over the so-called unipolar leads in the readier recognition of the lesser cardiographic signs of coronary arterial disease, which is the supreme function of the test, is emphasized.

Whenever medicinal treatment of a disease proves successful it can be indicated in an area occupied by a postage stamp. Thus, in heart failure digitalis leaps to one's mind, or penicillin in the case of bacterial endocarditis, and aspirin in rheumatic fever. In the text, therefore, only drugs which have a real claim in the treatment of a condition are described in any detail, and only when such claim has been justified following a controlled clinical trial where a particular medicine has proved beneficial and without inducing toxic effects. To afford space for the description of remedies which have failed to fulfil these requirements has been deemed wrong.

It has become customary to add a bibliography at the conclusion of each chapter, and the desire to do this is natural in order to acknowledge the contribution made by so many research workers in this section of medicine, and whose endeavours have made possible the writing of a book like this. The compilation of such a list of publications, however, is bound to be incomplete and inadequate for the purpose of a reader interested in a particular research project. Nonetheless, it has been considered helpful if a few papers should be named at the end of each chapter to guide the reader in the pursuit of this purpose.

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THE writing of a textbook can never be the product of the author's lone effort. He draws help from outside sources.

The inspiration for this work was kindled many years ago by a request from postgraduate students to place on record a series of lectures on cardiology given at the London Hospital; their generous appreciation has earned my lasting gratitude and given me continued encouragement.

The instruction I received from my teachers at the London Hospital, and the ideas born of discussions with my colleagues there and at the National Heart Hospital, or when attending congresses both in this country and abroad, have naturally influenced the thoughts which are set out in this book. Contemporary writings on the application of technology to clinical cardiology have enabled me to see more clearly the function which laboratory tests should exercise in the specialty in the future.

To acknowledge the help freely given in the preparation of this book by the publishers, Messrs. E. & S. Livingstone Ltd., and especially by Mr. Charles Macmillan and Dr. W. A. R. Thomson, affords me real pleasure.

My greatest debt I owe to my wife, who through many years has deprived herself of participation in many pleasing pursuances in order to allow me the time needed to prepare this book and other writings in cardiology.

CONTENTS

CHAPTER 1

INCIDENCE OF HEART DISEASE. INTERROGATION AND EXAMINATION OF THE PATIENT

DISEASE of the cardiovascular system accounts for a large proportion of the illnesses to which man is heir, in this country as in any other, and it claims a similar proportion of deaths among its population. The truth of this is apparent to every practising physician whether engaged in general medical practice or in hospital work. Accurate figures that tell of the number of people inflicted by heart disease are not available, because even those prepared in the office of the Registrar General can only represent approximate values, in that they are propounded from declarations on diagnosis made by the country's doctors, verified infrequently by necropsies, and therefore inevitably reflecting some inaccuracies. Nonetheless, since the figures convey a general impression of the situation they are given in Table I which outlines such a review for one year.

Table I.—EXTRACTS FROM THE REGISTRAR'S STATISTICAL REVIEW OF ENGLAND AND WALES FOR 1959.

Cause of death	*Number of deaths*	*Death rate per million living*
Cardiovascular Syphilis	734	48
Rheumatic Heart Disease	7,197	473
Coronary Arterial Disease	84,922	5,649
Hypertension and Hypertensive Heart Disease	18,199	1,200
Miscellaneous Heart Disease	12,764	842
Miscellaneous Arterial Disease . . .	13,576	897

HISTORY TAKING

Surveyance of the electrocardiogram and of the products of other instrumental or chemical tests may enable the physician to name the disease that affects the cardiovascular system, but his casual attention and cursory glances at the bed-side or couch-side, will fail to gain the confidence and trust of the patient whom he should recruit as a strong ally to overcome a temporary or permanent handicap imposed on him by illness. As devotees to laboratory methods of investigation increase in number, there is a danger that clinical medicine is becoming less fashionable. There is, however, no danger that it will ever go out of fashion, because the patient and his ailment will ever remain inseparable. In the case of heart disease especially, the heart and the mind are close companions, and often treatment of the mind takes precedence over the care of the heart. In no other specialty is time so well expended in keeping

company with the patient, for such unhurried attendance encourages him to combat a temporary or enduring illness, and ultimately wins for the physician the gratitude of one who has recaptured health.

The interrogation of a patient and the analysis of the symptoms which he recounts are practices that will survive in an electronic age, for no push-button contrivance can prove itself to be better, or its equal, in sifting the evidence which eventually uncovers the nature of the disease. The help given by diverse machines and tests is never to be rebutted outright, but such means must not be allowed to circumvent the presiding authority of an unhurried personal catechism and the subsequent assay of the collected information.

The interrogation of a patient suspected of cardiovascular disease does not differ materially from that adopted for any other anatomical territory. The plan is the same, but obviously the nature of the questions will vary. In the first instance the patient should be allowed time to recount his symptoms spontaneously, and this is followed by questioning which will test the significance of the complaints outlined by the patient.

A patient with heart disease may only tell of his chief symptom which often masks the lesser. Thus, if dyspnœa is severe it will overshadow most other symptoms, even pain. After a record has been made of the presenting symptom, therefore, it is necessary to ascertain by direct interrogation, the presence of other subsidiary complaints, or those the outcome of heart failure. Although chest pain and dyspnœa are the most common symptoms in patients with heart disease, those associated with hypertension, renal disease, affections of the nervous system, and certain vascular disorders are not uncommon, and should be kept in mind.

It is appropriate to postpone a discussion of the several symptoms that arise from heart disease until the clinical states in which they appear, are described, Thus, **cardiac pain** will be discussed in the section dealing with coronary arterial disease, **cyanosis** will be dealt with under the heading of congenital heart disease, **palpitation** under arrhythmia, **dyspnœa, œdema, ascites** and **jaundice** under heart failure, and **syncope** under Stokes-Adams disease.

The desirability of interrogating a patient in the presence of a relative or friend is determined by the doctor's custom, bought by experience and governed by the special circumstances of the interview. Naturally, the patient's wishes in the matter, and these are seldom explicit, have to be respected, but if no objection is advanced, as is usual, a more accurate description of the illness, in the case of husband and wife for instance, is obtained in the presence of each other. Such practice not only ensures a fuller account of symptoms, it also accumulates information about the patient's environment in the home, and when a history of alcohol consumption is sought a truer estimate of the quantity is more likely to be obtained. If the conversation tends to be monopolized by the witnessing partner, the doctor can easily correct this.

When the history is obtained in the presence of the patient's family doctor it should be a rule to take a fresh account of the illness, and one uninfluenced by a preliminary version of it at the hand of another. This apart, the physician will elect to depend on his own ingenuity to marshal help from any who can give it, including the patient, relative, friend, or another medical practitioner.

In order to ascertain how rapidly the condition is progressing it is necessary to know the duration of an illness, and neglect to inquire earnestly about past symptoms

has often meant a failure to discover a symptom-free period, and has led to a false estimate of the benefit gained from previous treatment, and of the future outlook.

Apart from knowing a patient's occupation, a record of his habits and daily activities should be known because it helps to pass judgment on the capacity for work in the future. Thus, ability to follow a strenuous occupation in the past helps to assess the degree of cardiac injury and the likely response to a period of rest. An acquaintance with the patient's domestic responsibilities and financial obligations provides the measure of his opportunity to take such rest during a period of treatment, and even during actual employment. Information regarding customary recreation should also be available when advice on future activities is outlined. The influence of injurious agents which contrive to aggravate the symptoms of cardiovascular disorders must come under review, so that excesses, especially of spirit-drinking, or dietary indiscretion, may be corrected.

In a search for the cause of cardiovascular disease every record of past ill-health should receive attention. When valvar disease is present, inquiry might be directed to finding a past history of any of the manifestations of rheumatic fever like joint pain, chorea, or tonsillitis, but there is need to emphasize that the presence or absence of such a history must on no account sway an opinion based on the findings of clinical examination; to ensure this freedom from bias, an inquiry about a previous attack of rheumatic fever should be postponed until decision has been taken from a consideration of the physical signs which by themselves must determine the presence or otherwise of valvar heart disease. In the case of heart pain or aortic regurgitation a previous history of syphilis gains significance. In atrial fibrillation without obvious cause, or when obscure cardiac enlargement and failure defy an explanation, relentless questioning might draw an admission of heavy spirit-drinking. When a congenital cardiovascular disorder is suspected, evidence that a heart murmur was present in infancy or early childhood, is of value.

PHYSICAL EXAMINATION

The cardiovascular system is not the only one to claim attention when disease of it is either present or suspected, for the nervous, pulmonary, digestive, renal and endocrine systems, should be examined as well. Neglect of them has often meant a failure to discover some associated or complicating lesion, and this in turn has resulted in wrong diagnosis, prognosis, and treatment.

A scheme for the clinical examination of the heart may be outlined under four heads.

GENERAL EXAMINATION

The patient's physical development and nutrition should be noted. Obesity will concern treatment and leanness may be important in diagnosis. The appearance of the face may disclose cyanosis, pallor or frank anæmia, icterus, or the features of an endocrine disturbance. Examination of the thyroid gland must never be omitted, for its enlargement will give rise to a search for the signs of thyroid toxæmia. Clubbing of the fingers and toes, and developmental anomalies, have great significance when congenital heart disease is suspected, and evidence of atavism instanced by the presence of polythelia (accessory nipples) will support the genetic etiology as in systemic and pulmonary hypertension, and in certain forms of myocardiopathy.

To notice the breathing, and the posture adopted to provide for the greatest respiratory ease, is of first importance. The height of the head-rest, told from the number of pillows necessary to promote sleep, gives an indication of the severity of cardiac dyspnœa. Other objective signs of heart failure should be looked for and which include distension of neck veins, enlargement and tenderness of the liver, ascites, œdema of the ankles, and crepitations or evidence of fluid at the lung bases. Examination of the urine and of the optic discs and retinae, should never be neglected in any patient with heart failure, and especially in hypertensive heart disease. Changes in the central nervous system may sometimes explain certain cardiovascular signs, as in syphilis, Friedreich disease, or myotonia atrophica.

EXAMINATION OF THE PERIPHERAL PULSE

The **radial and brachial** pulses should be examined unhurriedly for they reflect particularly the behaviour of the left ventricle. In turn, information is sought on their rate, rhythm, and character, the state of the arterial wall, and the blood pressure.

The *rate* should be counted at the wrist and compared with the speed of the heart counted by auscultation whenever the rhythm is irregular; any deficiency of the wrist-pulse is thereby noted. It should not be sufficient to record that the *rhythm* is irregular, but an attempt should be made to tell the kind in terms like extra-systoles, atrial tachycardia, atrial fibrillation, or heart block, an exercise aided by keeping the eyes on the venous pulse and the fingers on the radial pulse. The *character* of the pulse, both radial and brachial, should be described as small, natural, or large and collapsing. If large it will promote a search for aortic regurgitation whose murmur might otherwise evade casual auscultation. The radial pulse may be absent in the so-called 'pulseless disease', or misplaced should the artery pursue an abnormal course. The right pulse is small or absent in the case of an innominate aneurysm, and the left when the site of aortic coarctation is proximal to the left subclavian artery. The condition of the *brachial arterial wall* has great importance whenever the blood pressure is raised, and the diagnosis between the innocent state of hypertonia and the diseased state of hypertension has to be made. In the latter condition it is usually firm and narrow (whipcord-like) from contracture, while in the former it is natural as a rule.

A record of the *blood pressure* is necessary in every patient suspected of cardio-vascular disease, and its registration should be governed by the following instructions:

The Instrument.—The equipment used for measuring arterial blood pressure, whether of the mercurial or aneroid type, must be in good condition and in the case of the aneroid type, should be frequently calibrated against a standard mercurial manometer. The mercurial type is the most dependable.

Position of the Patient.—The patient should be allowed time to recover from any recent exercise or excitement, and must be comfortable either in the sitting or reclining posture. There should be no constriction of the arm or interference with the armlet by any clothing.

Application of the Cuff.—The cuff must be of standard size and with its rubber bag at least 12 cm. wide. The cuff, completely deflated, should be applied with the middle of the rubber bag over the inner side of the arm, and its lower edge 1 inch (2·5 cm.) above the bend of the elbow. It should fit closely and evenly around the arm to ensure against bulging at the sides when inflated, particularly in obese subjects.

Determination of the Systolic Pressure.—With the stethoscope applied lightly and evenly over the brachial artery just below the cuff, the bag is inflated quickly to a pressure about 30 mm. of Hg above the point at which the sound of the pulse disappears in order to avoid the 'silent gap' which may be present, and deflated slowly. The systolic pressure is the level at which successive sounds are first heard.

Determination of the Diastolic Pressure.—With the pressure in the cuff continuing to fall slowly and uniformly, the sound increases to its maximal intensity and then decreases, at first gradually and later suddenly. The point where the loud clear sounds change abruptly to the dull and muffled sounds should be taken as the diastolic pressure.

Special Conditions.—In atrial fibrillation only approximate blood pressure readings can be obtained; the systolic value should be taken at the point at which the majority of the beats appear, and the diastolic (if possible to obtain) at the point where they become muffled. Alternation in the strength of the beats (pulsus alternans) should be carefully looked for; this must be distinguished from the alternating values given by alternating extrasystoles (pulsus bigeminus). In aortic regurgitation the diastolic pressure is marked by a less obvious change in the quality of the sounds. If the pulse feels different on the two sides, the blood pressure should be taken in both arms. When a raised blood pressure in the arm is unexplained it should also be taken in the leg, when coarctation of the aorta may be discovered. For recording blood pressure in the leg the cuff and its covering should be wider and longer; the patient should be in the prone position with the leg extended and the sounds auscultated over the popliteal artery below the lower edge of the carefully applied cuff.

Excessive pulsation of the **retinal** arterioles is seen in aortic regurgitation and in coarctation, while the blood stream may be 'beaded' in 'pulseless disease'.

In the presence of intense headache, especially when one-sided, a thickened **temporal** artery should be sought.

When a stroke has taken place, especially in the absence of hypertension, a diminished **carotid** arterial pulse will suggest atherosclerotic narrowing with intra-arterial thrombosis as the cause of cerebral embolism.

Pulsation in hypertrophied **scapular** and internal **mammary** arteries confirms the presence of a collateral circulation established in coarctation of the aorta.

Palpation of the **femoral** arterial pulse should never be neglected in that its absence, even by itself, usually indicates coarctation of the aorta.

To feel for a **dorsalis pedis** arterial pulse is also important. As a rule its presence will exclude ischæmia of the lower limbs as a cause of pain, but its absence on both sides does not necessarily confirm such ischæmia in an individual without pain, although a failure to feel it on one side only, has that meaning.

EXAMINATION OF THE NECK PULSES

Critical inspection of the neck in a patient suspected of heart disease will often disclose signs which by themselves may supply an unerring diagnosis.

Half a century ago James Mackenzie emphasized that while the radial pulse or the apex beat told us of the doings of the left ventricle, a study of the venous pulse supplied information regarding systole and diastole of both the right atrium and right ventricle. For this reason, variations in the venous pulse supplies information about heart disease which a study of the arterial pulse cannot provide.

The venous pulse springs from the internal jugular and innominate veins which are too deeply situated to be seen as anatomical structures, and only pulsation tells

of their presence beneath. Should pulsation appear in the easily visible external jugular vein, as sometimes happens, its collapse coincides with the carotid pulse.

The venous pulse is most advantageously examined when the patient reclines with the head resting on one pillow and inclining to the left. It is more obvious during inspiration and is accentuated by pressure on the abdomen. Should the veins be greatly distended, the pulse is best examined when the patient sits up. The vascular

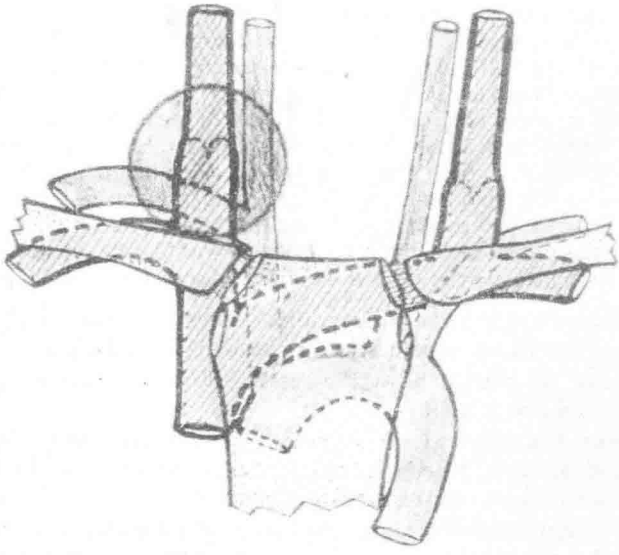

FIG. 1

Anatomy of the vessels in the neck whose pulsation may be visible. The circle over the jugular bulb on the right depicts the place where the tambour of the polygraph should be applied.

movements are watched at the jugular bulb above the inner end of the right clavicle, in the suprasternal notch where pulsation in the left innominate vein is seen, and again over the jugular bulb on the left side. When recording the pulse in the reclining position it is necessary to turn the head slightly to the right so as to relax the right sterno-mastoid muscle; otherwise its taut edge prevents a good contact between the tambour of the polygraph and the jugular bulb (Fig. 1).

Events in the Venous Pulse

The *a* wave

The first positive wave (the *a* wave in the polygram) begins just before the start of atrial systole, and reaches its peak as atrial systole proceeds. Its first phase is

caused by passive filling of the right atrium, and its remaining ascent is the outcome of atrial contraction. The wave coincides with a rise in atrial pressure taking place during atrial systole.

Prominence of the *a* wave. An excessive sudden rise of the atrial wave followed by an abrupt recession produces a hasty movement which is best described as a **flick**. It is caused by a rising pressure within the right atrium which produces a backward recoil into the jugular vein during its systole when its exit flow is impeded (Fig. 2). Naturally the *a* wave is not found in the presence of atrial fibrillation.

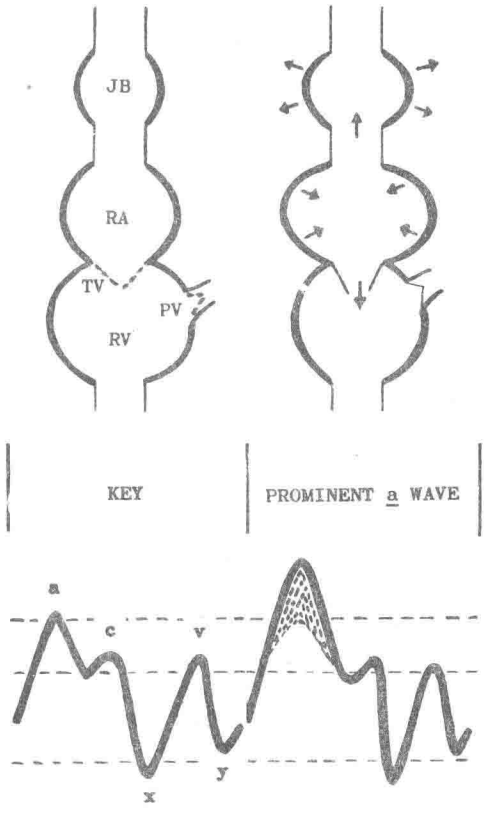

FIG. 2

Diagrammatic representation of events in the right heart which create a prominent *a* wave in the *jugular pulse*. JB: jugular bulb. RA: right atrium. TV: tricuspid valve. RV: right ventricle. PV: pulmonary valve.

Tracing the causes of a prominent *a* wave in relation to the course of the blood flow, they include **myxoma** of the right atrium, a greatly distended atrium in **atrial septal defect, tricuspid stenosis,** enlargement of the right ventricle in **myocardiopathy,** rigidity of the hypertrophied left ventricle as in aortic stenosis or hypertension **(Bernheim effect), pulmonary stenosis, pulmonary hypertension,** and the exhibition of **cannon waves** in nodal rhythm, or as occasional events in partial heart block; such waves result from coincidence of atrial and ventricular systole, so that the right atrium contracts in the face of a closed tricuspid valve (Fig. 3).

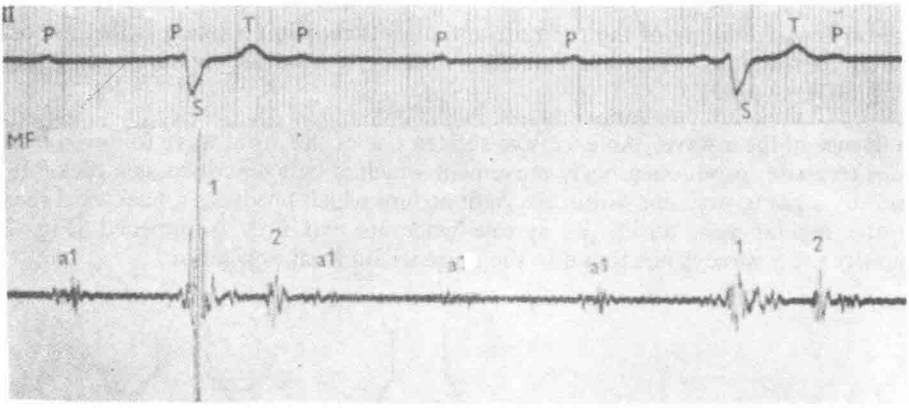

FIG. 3

Bruit de canon. In this patient with complete heart block, only two ventricular beats are seen; the first of these produced a very loud first heart sound owing to coincidence of the atrial and ventricular contractions.

The *x* trough

This negative wave is caused by relaxation of the right atrium during atrial diastole. The descent *a*-*x* coincides with a fall in atrial pressure, and is interrupted by the *c* wave caused by the carotid pulse.

Change in the *x* trough. The trough becomes *deep* as in enlargement of the right atrium in atrial septal defect (Fig. 4), *shallow* in atrial fibrillation, and *absent* in gross tricuspid regurgitation when it is replaced by a positive *s* wave at the commencement of the *v* wave.

The *v* wave

This is caused by passive filling of the right atrium and it coincides with ventricular distension and contraction. The beginning of the *v* wave varies in its timing because its formation is dependent on the quantity of blood stored in the right atrium during ventricular systole; such blood, arriving from the peripheral veins, overfills the right atrium and wells up into the superior vena cava and jugular veins, creating the *v* wave. While the initiation of the wave is variable, its peak is one of the most certain landmarks in the venous tracing, indicating as it does the time of opening of the tricuspid valve.

Prominence of the *v* wave. A prominent *v* wave in the venous pulse appears as a **surge**, and is indicative of tricuspid regurgitation. This results from *rheumatic* affection of the valve in company with mitral stenosis, or rarely from *congenital* malformation (Ebstein disease). The most common fault, however, is a *relative tricuspid regurgitation* which follows great enlargement of the right ventricle as in atrial septal defect, severe pulmonary stenosis, severe pulmonary hypertension, and myocardiopathy. The effect of tricuspid regurgitation is to increase the amount of blood accumulating in the atrium during ventricular systole, causing a premature wave (*s*) on the tall *v* wave (Fig. 5).