Basic Cardiology

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BASIC CARDIOLOGY

BY

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PREFACE TO THE SECOND EDITION

THIS Edition is longer than the first by the addition of three new chapters, two by younger colleagues here in Sheffield and one by myself on Electrocardiography. Dr. David Verel contributed the chapter on 'Cardiac Catheterization', and Dr. Ronald Grainger the chapter on 'Angiocardiography'. I am greatly indebted to them both and feel that the book is now more worthy of its title.

The revision has been painstaking and, I hope, thorough. Much has been deleted, large sections have been rewritten, especially those bearing on treatment, and there are many new illustrations. Nevertheless, what I have written remains essentially a collection of lectures given in the course of my teaching duties at the Sheffield Royal Hospital, and as a consequence often has a didactic and reminiscent quality which must remain my excuse for the frequent intrusion of the first personal pronoun. I would like to think that I have brought the book reasonably up to date, but this is, of course, a vain hope in this day and age of rapid progress, experimentation, and research.

I must confess, moreover, to having shirked bibliographic references, partly through laziness, but largely because the book is addressed to students, house-physicians, junior registrars, and general practitioners interested in the subject. They would probably not have the time nor the inclination to consult the original references had they been listed at the end of each chapter. I am particularly indebted in this connexion to Louis Wolff for his stimulating book on Electrocardiography, to Wallace Brigden for his articles on Myocarditis, to our own Professors Stuart-Harris and Graham Wilson for their writings on Pulmonary Heart Disease and Diuretics respectively, to the late J. W. Brown for his book on Congenital Heart Disease, to Aubrey Leatham for his Classification of Cardiac Murmurs, to H. R. S. Harley for his *Modern Trends in Cardiac Surgery*, and to B. B. Milstein for his *Cardiac Arrest and Resuscitation.*

Once again I have had the kindest of assistance from my publishers, often on matters I would otherwise have overlooked. It is a pleasure to record my thanks to them.

T. E. G.

Sheffield
1964

FROM THE PREFACE TO THE FIRST EDITION

THERE has been a tendency of late to disparage the lecture as a means of conveying information to the medical student. The reason is not obscure. Until recently the curriculum was overloaded with compulsory lectures of all sorts, many of them delivered at the end of the afternoon to students who were physically and mentally jaded and no longer at their most receptive,

and often by a lecturer who for similar reasons was no longer at his best. Moreover, the average lecture, though full of good things, was ill prepared and often badly delivered.

Those of us at Sheffield who were privileged to sit at the feet of the late Professor Leathes in the Department of Physiology will remember to this day the virtuosity of his lecturing and the skill with which he made his points. In their own way they were just as inspiring as the speeches of Sir Winston Churchill during the darkest days of the last war. I have no doubt they were just as carefully prepared.

The argument that lectures are no longer necessary because textbooks have improved so much during the last twenty-five years is not a valid one. The average student has not the ability, nor indeed the time, to sift the gold from the dross, the essential from the inessential, or the important from the unimportant in the course of his hurried reading. As the examination approaches, he turns his attention more and more to various aids and synopses, where he will find a condensed account of the facts without the reasoning and the argument that are so essential for their comprehension. If he has a good memory and good fortune he will defeat the examiners, but in a short space of time he will be in the doldrums again, never quite knowing how to tackle the difficult problems of clinical practice.

Now the lecturer, if he enjoys teaching, is in a position by virtue of his knowledge and experience to supply not only the essential facts but also the arguments on which they are based; he can sense by the attention of the class whether he is getting his points across, and he learns by the questions he is asked where he has failed to give a clear account.

It is for these reasons, therefore, that I have ventured to write this book. The approach is that of the general physician with an abiding interest in cardiology. Some of the lectures were given at Sheffield University, some at the Royal Hospital, and some elsewhere: some to medical students, often in the form of lecture demonstrations, and others to postgraduates. They are not intended to take the place of a standard textbook on cardiology. My aim is not so ambitious. But I hope they may fulfil a useful purpose in bridging the gap which exists between the detailed exposition of the specialist textbooks on the subject and the more concentrated and often rather dull account which is to be found within the covers of one of the standard textbooks embracing the whole of medicine. Perhaps they may stimulate the earnest student, and even the postgraduate, to a fuller understanding and a more rational practice of one of the most satisfying branches of clinical medicine.

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BASIC CARDIOLOGY

CHAPTER I

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HEART DISEASE

OUR conception, understanding, and appraisal of heart disease have changed very materially over the last fifty or sixty years, as a result of a widespread adoption of science and the application of the experimental method. This approach has been directed to the study of the haemodynamics of the circulation and the accompanying acoustic phenomena, no less than to the biochemical and electrical changes which occur in the heart.

During these years more progress has been made, more new facts have been learnt about the circulation, and more theories elaborated to account for them than in any comparable period of time. Moreover, this knowledge is based on a securer scientific foundation than was available in former times. Much of it, as a consequence, is likely to be of lasting value and permanent validity.

It is of more than academic interest to compare the various agencies at work today with those in operation at the beginning of the present century. One has only to dip into the medical writings of those days to appreciate that they consist almost entirely of descriptive case reports and post-mortem analyses. In fact, the study of the dead was almost as important as clinical observation, and constituted perhaps the most scientific aspect of medicine. The disorders of cardiac rhythm were at that time only just beginning to be studied by means of arterial and venous pulse tracings. Auricular fibrillation was known as perpetual arrhythmia or even delirium cordis—without a clue as to its nature. Coronary thrombosis, though accepted at autopsy as a cause of sudden death, had not achieved clinical recognition in the living. The electrocardiograph had not appeared on the scene, nor had the recently discovered Röntgen ray (1895) been applied to cardiac investigation. The systolic murmur reigned supreme. Weak hearts abounded, and the significance of dropsy was such that its mere mention in grown-up conversation made my youthful flesh creep in pre-World-War-I days. Syphilitic aortitis played an important role in cardiac pathology; the blood-pressure was only just beginning to be measured with Riva-Rocci's mercurial sphygmomanometer; cor pulmonale was unknown; and the only endocrine disorder of which the association with heart disease was recognized was exophthalmic goitre.

But the turn of the century witnessed mechanical and technological advances unparalleled in the history of man. The principles of pure science were being applied with a will to everyday problems in industry and in the home, and men's minds turned naturally to the machine. The automobile had been born, and electricity was being harnessed as a means of lighting and more rapid communication. Wireless telegraphy and the aeroplane were in the throes of development. Small wonder then that medicine, perhaps the oldest of the applied sciences, should have shared in this renaissance. Experiment began to supplement experience, new instruments were fashioned to study the living circulation, and the validity of theory was tested as never before.

In 1902, Mackenzie had perfected his polygraph, and it became a matter of routine to record simultaneously the radial arterial and jugular venous pulses in cases of cardiac arrhythmia. In 1903, Einthoven introduced the electrocardiograph, which shed further light on the mechanisms of cardiac contraction and also initiated the study of changes of electrical potential in the heart. Radiology gradually and increasingly came to be applied to the determination of cardiac size and shape from 1902 onwards, though routine screening of the heart is surprisingly recent. In the late twenties the circulation rate began to be studied, and in the thirties the introduction of the unipolar limb and chest leads gave an additional and welcome fillip to electrocardiography. Phonocardiography, or the graphic recording of the heart-sounds and murmurs, was introduced as early as 1904, but suffered strange neglect until quite recently. Kymography (1931) enabled the radiologist to record the pulsations of the lateral cardiac and aortic borders, and ballistocardiography (1947), though essentially still a research tool, affords useful information about the propulsive performances of the heart. Last but not least, the advent and practice of cardiac catheterization (1929) and angiocardiography (1938) have added much to our knowledge of the haemodynamics of the human circulation, and have also played no small part in the revolutionary development of cardiac surgery.

The conclusion is inescapable that cardiology owes its spectacular advance during the last fifty or sixty years to the application of the experimental scientific method to the understanding of the circulation, in both animals and man, and to the application of the instrumental method to the living subject.

CLASSIFICATION OF HEART DISEASE

Some sort of classification of heart disease is essential, and none is more useful than one based on aetiology. Thus, cases are grouped together according to whether the essential defect is congenital, inflammatory, or degenerative, toxic, metabolic, or endocrine.

Congenital Morbus Cordis.—Congenital morbus cordis is the result of maldevelopment occurring either during the crucial 2nd month of intrauterine life or immediately after birth, when the lungs take over their function of oxygenating the blood.

Inflammatory Lesions.—The inflammatory lesions are the result of acute or subacute *rheumatism*, in the great majority of cases. The common end-result is a valvulitis, and the most vulnerable valves are the mitral and the aortic, but the myocardium is also affected, and in some cases, especially in the acute stage, the pericardium and blood-vessels are also involved. *Subacute bacterial and acute ulcerative endocarditis* may occur *de novo* or be grafted on to existing rheumatic or congenital lesions. *Syphilis* has a predilection for the aorta and leads to aortitis. If the mouths of the coronary arteries are narrowed in the fibrotic process which occurs, the clinical result is angina pectoris. If the root of the aorta is diffusely involved the aortic valve is rendered incompetent. If a localized portion of the aorta yields, an aortic aneurysm is produced and behaves rather like a mediastinal tumour. Or a combination of these effects may occur. *Tuberculosis* attacks the pericardium and produces constrictive pericarditis, characterized in the early stages by effusion and later by calcification in the pericardial sac. It is constrictive in the sense that diastolic relaxation of the heart is hindered. The net effect is inadequate venous filling of the heart, which leads to chronic venous engorgement and ascites in the absence of myocardial failure.

Degenerative Lesions.—The degenerative lesions are usually part and parcel of a generalized arteriosclerosis. Sometimes the coronary arteries appear to bear the brunt of this process, with resultant angina pectoris and coronary thrombosis. More often the peripheral arteries are affected at the same time and betray their inadequacy in the legs as intermittent claudication. Sometimes angina and claudication occur in the same subject, who is thus doubly incapacitated. Not infrequently the aorta is the seat of advanced atheroma with resulting aneurysm formation, leading to aortic incompetence (if the root of the aorta is affected), diffuse dilatation if the degenerative process is widespread, and, rarely, to dissecting aneurysm where the media yields and splits at a weak point and in extreme instances forms a double-barrelled aorta. Often arteriosclerosis is preceded by hypertension, but this is not always so, though there is good reason to suppose that a high blood-pressure accelerates the degenerative process.

Toxic Cardiomyopathy is due to infection, hypersensitivity, or metabolic change. Thus, while infection gives rise to *myocarditis*, hypersensitivity phenomena and metabolic diseases cause *myocardosis*. Examples of the former occur in many of the virus diseases—in pneumonia, meningococcal septicaemia, and typhus—while hypersensitivity myocardosis is seen in the collagen diseases, notably in polyarteritis, systemic lupus erythematosus, and scleroderma, and metabolic myocardosis occurs in beriberi and alcoholism.

Metabolic Disorders.—The common metabolic disorders which have an effect upon the heart are obesity, diabetes, and gout, which are frequently connected with hypertension and atherosclerosis. The uncommon ones are glycogen disease, in which the youthful myocardium (and usually the liver too) is infiltrated with glycogen; haemochromatosis, in which there is abnormal deposition of haemosiderin in the heart muscle (as well as in the skin, liver, and pancreas); and wet beriberi due to vitamin B_1 deficiency.

Endocrine Disorders.—The endocrine disorders which affect the heart are numerous. Thyrotoxicosis, if unchecked, is likely to lead to high output failure; myxoedema is characterized by a sluggish heart-action combined with cardiomegaly—sometimes as a result of oedema of the muscle-fibres and sometimes as a result of hydropericardium; in acromegaly the cardiac enlargement which occurs is greater in degree than the generalized splanchnomegaly which inevitably occurs in this condition—often it is associated with a diffuse arteriosclerosis; in pituitary cachexia, or Simmonds's disease, the heart undergoes brown atrophy and degeneration of the muscle-fibres; hyperfunction of the adrenal cortex, or Cushing's syndrome, proclaims itself by the moon-face appearance of the patient, and is constantly associated with hypertension and cardiac hypertrophy; phaeochromocytoma (or tumour formation of the chromophil cells of adrenal or sympathetic ganglion origin) characteristically gives rise to paroxysmal attacks of hypertension and hyperidrosis, but the hypertension may be persistent; in either case the heart tends to suffer secondary atheromatous and degenerative change. In hypofunction of the adrenal cortex, Addison's disease, the heart is small and hypodynamic and the blood-pressure low.

Hypertensive Heart Disease.—Hypertensive heart disease alone does not readily fit into this aetiological classification, for the simple reason that we are still ignorant of the cause of hypertension. Hypertension may occur independently of recognizable disease elsewhere. It is then spoken of as essential, idiopathic, or benign hypertension, which is usually an extremely chronic affection and sometimes entirely symptomless, until some arteriosclerotic complication asserts itself. There is also a rapidly developing hypertension in which there are profound renal changes—this is known as malignant hypertension. It is much less common than the benign variety. The other kinds of hypertension are secondary to other conditions, such as chronic nephritis, the Kimmelstiel–Wilson syndrome, and the numerous endocrine conditions to which reference has already been made. It would seem that hypertension is an important contributory factor in the genesis of arteriosclerosis, though the latter can and does occur sometimes without any elevation of the blood-pressure. In the present state of our knowledge, it would appear that there are three important factors in the production of hypertension—neurogenic influences, renal ischaemia, and complex endocrine mechanisms, probably centring around the adrenal gland. The net effect of a sustained elevation of the blood-pressure is to increase the work of the left ventricle and to impose a heavy load on the arterial system generally. Left ventricular hypertrophy and a generalized arteriosclerosis are the result. The common complications are left ventricular failure, coronary arterial disease, cerebral vascular and ocular manifestations, peripheral arterial disease, and chronic interstitial nephritis.

THE CLINICAL METHOD

History.—Our first task in the cardiac case, as in the general medical case, is the extraction of the history. To be able to take a good history

requires, in a high proportion of cases, tact, patience, and, above all, practice, and that is why we devote so much time to medical clerking. I am a great believer in asking leading questions when I cannot get the information I want in any other way. If leading questions are deliberately avoided much valuable information may never be gleaned; with experience and the application of common sense it is usually easy to assess the value of the reply. The common symptoms will be breathlessness in some form, pain, awareness of the heart's beating (palpitations), weakness, and syncope. Each symptom is carefully analysed by sympathetic cross-questioning. At the end of the history one is often well on the way to a diagnosis, or at least to a differential diagnosis. Angina pectoris, coronary thrombosis, paroxysmal nocturnal dyspnoea, paroxysmal tachycardia, and Stokes-Adams attacks are often confidently diagnosed from the history alone.

Examination.—During the time you are taking the history you will observe many things, including the patient's demeanour, his colouring, the breathing, and the appearance of the hands, though you may wish to examine the hands more closely later on; but if something about the hands or face excites your interest, get up, interrupt your questioning, and settle the point at once or you may forget to do so later on under the stimulus of some other line of thought. I like to begin the examination in the upright position whenever possible. One gets a better overall view of the chest. Scoliosis, pigeon chest, depression of the sternum, and epigastric pulsation are all better seen in this position, and premature beats, which may be present, often disappear most obligingly with the effort of climbing on to the couch. Sometimes a systolic murmur which is heard at the beginning of the examination disappears when the patient lies down, and vice versa: if so, it is likely to be innocent.

But the more important and prolonged part of the examination will be carried out on the couch with the patient propped up, lowering the head of the couch only, unless there is pronounced orthopnoea, during the examination of the abdomen. The precordium and the neck will be inspected for abnormal pulsations, and the external jugular veins examined with a view to determining whether or not there is an increased venous pressure. I believe in getting my stethoscope on to the precordium early in the proceedings before the heart has had time to settle down—it makes for easier recognition of murmurs; and the same applies to palpation with regard to thrills. No one, nowadays, would omit listening to the heart in the left semilateral position in the phase of full expiration, as this manœuvre accentuates a mitral diastolic murmur and also the third heart-sound. The most difficult murmur to spot, however, is the faint aortic diastolic; it is best heard, using the diaphragm type of chest piece, with the patient sitting up and leaning forwards, again at the end of expiration. The same position is helpful in the detection of the aortic systolic thrill, which is so useful a confirmatory sign in aortic stenosis.

Percussion.—Percussion can be left to the end; it is less esteemed than it used to be, but still has its uses, especially if you are not in a position to

do your own X-ray screening. Percussion of the right cardiac border is notoriously unreliable in the absence of gross enlargement of the heart, but the left border can usually be percussed out satisfactorily unless there is emphysema, or a left-sided pleural effusion. Percussion is of great value if there is a sizeable pericardial effusion.

Murmurs will be discussed in subsequent pages.

Heart-sounds vary in quality and number and are of considerable importance. They were rather neglected until recently, when technological advances rekindled our interest in phonocardiography. The first thing to decide is whether you hear two or three. And if there are three sounds instead of two, are you hearing a *physiological third heart-sound* or *gallop rhythm* ? The third heart-sound is a normal finding at the apex in youthful subjects, especially in the left lateral decubitus. In the elderly patient the presence of a third heart-sound is likely to be due to gallop rhythm and warrants a full cardiac investigation. There is no excuse for missing gallop rhythm : there are three distinct sounds instead of two, they tumble over each other, and are also quite different from the triple rhythm due to a first heart-sound followed closely by a split second sound. The gallop is described as presystolic (or auricular) or protodiastolic (i.e., early diastolic), depending on whether the added sound occurs just before the first sound or just after the second. The presence of gallop rhythm denotes ventricular stress, either left or right, and is common after a coronary occlusion.

A *slapping first sound* at the apex, which persists after the patient has settled down, will make you hunt all the more diligently for the mitral diastolic murmur and the opening snap which may or may not accompany it. Unfortunately a persistent slapping first sound, and even a suggestion of a presystolic murmur at the apex, occurs in thyrotoxicosis as well as in mitral stenosis.

The *opening snap* is easy to recognize. It has the staccato quality one would expect from a snap : it is sufficiently distant in time from the second sound to be distinguishable from a split second sound, and it is best heard between the left sternal edge and the apex.

The *pulmonary second sound* is often *split*, especially in young people, because the pulmonary valve closes a fraction of a second later than the aortic. The splitting is more obvious at the height of inspiration. Later on in life the second sound is usually not split, but in right bundle-branch block wide splitting occurs. Accentuation of the pulmonary second sound occurs when there is an enhanced pressure in the pulmonary circulation, notably in mitral stenosis. A weak or absent pulmonary second sound may be noted in pulmonary stenosis. Similarly, accentuation of the aortic second sound is heard in systemic hypertension and a weak or absent aortic second sound may be noted in aortic stenosis.

The Lungs.—Examination of the lungs may yield important clues. There may be basal crepitations in left ventricular failure, rhonchi in the chronic bronchitic, and both in paroxysmal nocturnal dyspnoea and acute pulmonary oedema. Pleural effusion may be present on one or other side

and sometimes on both. It is not uncommon in congestive failure and sometimes complicates pulmonary embolism. Emphysema is as common a cause of dyspnoea as heart disease; it is usually associated with chronic bronchitis and is usually easily recognized. It is the commonest cause of chronic cor pulmonale. Occasionally the signs of basal bronchiectasis and pulmonary fibrosis will be found instead.

The Abdomen.—The abdomen is next examined with special reference to the liver and the presence of ascites. In congestive failure the liver is often tender as well as enlarged. The lower edge of the liver is by no means always easy to feel, especially in the obese subject. Percussion is fortunately a reliable alternative method of mapping out the lower border of the liver. Systolic pulsation of the liver occurs in tricuspid incompetence, but it is not an easy sign to be sure of. Ascites, on the other hand, gives rise to easily recognized shifting dullness. Ascites is commonly seen in gross congestive failure; in the absence of marked gravitational oedema its presence suggests the possibility of constrictive pericarditis, tricuspid incompetence, or cirrhosis of the liver. Although the spleen is engorged in congestive failure, it is not usually sufficiently enlarged to be palpable. The presence of splenomegaly in valvular disease would make one think of infective endocarditis. Rarely, one comes across a palpable abdominal aortic aneurysm of luetic or arteriosclerotic origin.

The Pulse.—I usually leave the pulse to the end. By this time one will have gleaned a lot of information about the case, and examination of the pulse may have a considerable confirmatory value.

Irregularity is more easily noted at the wrist than by listening to the heart—you will analyse it more carefully. Usually you will have little difficulty in deciding whether the irregularity is due to auricular fibrillation, premature beats, or sinus arrhythmia. The only difficult pulses to count are those which are very rapid (160 or more) and those which are grossly irregular in both time and force (auricular fibrillation with a rapid ventricular rate). You will note whether the radial artery is harder and more tortuous than normal. Sometimes it feels rather like a quill, or a wavy piece of blind-cord, under the skin. And you will also observe whether it is palpable for three or four inches towards the elbow instead of the usual one or two. These findings are common in arteriosclerosis and are usually accompanied by visible locomotor brachialis with a spring-like action in the neighbourhood of the elbow, and visible and palpable tortuosity of the temporal arteries.

The quality of the pulse is especially useful in a corroborative sense. The water-hammer pulse is characteristic of aortic regurgitation, but occurs also in the vasodilatation of thyrotoxicosis and pyrexial states. The hard pulse characterizes hypertension, and the small (weak) pulse mitral stenosis. A slow rising pulse with a sustained summit, known as the 'anacrotic pulse', is strong support for the diagnosis of aortic stenosis you have already made from your examination of the heart. And the rare, but very satisfying, recognition of pulsus bisferiens (which has a double quality) will enable

you to be pretty certain that the patient has both aortic stenosis and regurgitation. The dicrotic pulse also has a somewhat double quality. It is softer and less sustained than the pulsus bisferiens and occurs sometimes in febrile conditions, such as enteric fever. The paradoxical pulse occurs sometimes in pericarditis—during the inspiratory phase of respiration the pulse-beats feel weaker.

It is a good plan never to overlook careful palpation of the femoral arteries in the groins, especially if the brachial blood-pressure is high in a young or youngish patient. Absent or delayed pulsation is characteristic of coarctation of the aorta. On the other hand, a considerable thud over the same arteries is often encountered in aortic regurgitation. The arteries in the neighbourhood of the ankle will receive special attention, both if peripheral arterial disease is suspected and in the elderly patient with glycosuria.

The Retinal Arteries.—The state of the retinal arteries will be observed in all hypertensives; there may be narrowing of the arteries as compared with the veins, the veins may appear kinked where the arteries cross over them, or haemorrhages and exudates may be clearly seen. Occasionally in arteriosclerotic cases one sees small venous thromboses or arterial occlusions, the patient merely being under the impression that he requires new spectacles. Retinal haemorrhages and subconjunctival haemorrhages, especially in the lower eyelid, may be seen in infective endocarditis.

Blood-pressure.—You will save yourself time and trouble in the end if you localize the brachial artery in the antecubital fossa before listening to it. Never forget that the first (casual) blood-pressure reading is often too high, because the patient is nervous. You will never go wrong with the auscultatory gap, which occurs in some cases of hypertension, if you make it a practice to estimate the systolic pressure by palpation at the wrist as well as by auscultation over the brachial artery. Do not be worried if you cannot get a diastolic end point—quite often the sounds continue right to the bottom of the scale—and do not be surprised if you get a very low diastolic reading in aortic incompetence—readings below 60 mm. Hg are not uncommon. Sphygmomanometry is also useful in the detection of pulsus alternans. The pulse-rate suddenly doubles when both the weak and the strong beats succeed in getting through (at the upper systolic level). Often pulsus alternans can only be recognized in this way.

The Hands.—The hands call for more than a cursory glance. Digital tremor and unusual warmth of the hands, especially in women, are often found in thyrotoxicosis—and thyrotoxic heart disease is common. The hands are also unduly warm in coarctation of the aorta. Exceptionally long slender hands and fingers constitute arachnodactyly and an appreciable proportion of these cases have other congenital defects, including dislocation of the lens and congenital heart disease, or, later, develop a dissecting aneurysm of the aortic arch. About one in every four mongols has an atrial septal defect; and although one usually recognizes the mongolian idiot at a glance by the facial appearance, the stubby incurved little finger and single transverse palmar crease may be the initial clue pointing in the direction of a

congenital cardiac defect. Clubbing of the fingers is invariable in cyanotic cases of congenital heart disease. White clubbing of the fingers is a feature of subacute bacterial endocarditis, but is usually slight in degree; gross white clubbing occurs in pulmonary neoplasia, bronchiectasis, and in other suppurative conditions of the lungs, which may later cause chronic cor pulmonale. Osler's nodes on the fingers and flame-shaped or splinter haemorrhages beneath the nails occur in infective endocarditis.

The Urine.—Examination of the urine should never be omitted. Albuminuria occurs in cardiorenal disease, malignant hypertension, and congestive heart failure. Casts will also be found microscopically, but in congestive heart failure the urine is usually highly coloured and of high specific gravity (1020–1025). Albumin, casts, red and white blood-cells, and organisms occur in subacute bacterial and ulcerative endocarditis. Glycosuria is common in the elderly arteriosclerotic because of concomitant diabetes, and sometimes its presence will put you on the track of masked hyperthyroidism (where there is a reduced glucose-tolerance).

INSTRUMENTAL AIDS TO DIAGNOSIS

Electrocardiography.—What of our much vaunted instrumental aids to diagnosis? I have already stressed, at too great a length perhaps, how dependent on instrumentation cardiological progress has been. But I should not like you to regard the electrocardiograph and the X-ray screen as instruments which give a prompt answer to every cardiological problem. They are not guinea-in-the-slot machines which reveal the immediate diagnosis, though that is rather what is expected of them—especially, perhaps, by the lay intelligentsia. The results they furnish require judgement and interpretation. For instance, although the electrocardiogram will tell you at once whether or not the auricles are fibrillating, it will not help you to decide whether the fibrillation is due to mitral stenosis, thyrotoxicosis, or pneumonia, and it will not give a hint as to whether digitalis or quinidine should be employed to combat it; and similarly a prominence of the pulmonary conus, as seen radiologically, will not of itself distinguish between atrial septal defect, patent ductus arteriosus, mitral stenosis, and cor pulmonale.

What, then, can the clinician expect of his electrocardiograph? I would say an immediate recognition of the mechanism responsible for any of the cardiac arrhythmias, tachycardias, or varieties of heart-block which may be present; valuable clues and sometimes strong evidence in favour of left or right ventricular hypertrophy (and even auricular hypertrophy); strong presumptive evidence and sometimes (if serial records are available) irrefutable evidence of cardiac infarction and pulmonary embolism. Useful and interesting, though not necessarily diagnostic, records can be obtained in cases of pericarditis, digitalis and quinidine overdosage, angina of effort, myxoedema, and disturbances of the blood-potassium level.

Radiology.—Radiology, though widely practised and of unique value in cardiology, is likewise no short cut to diagnosis. It enables one to assess the over-all size of the heart if due allowance is made for the position of the

diaphragm, the shape of the sternum, and any deformity of the thoracic spine which may be present. The position of the diaphragm is strongly influenced by body build; it is high in those who are short and stocky, and elevated in the obese and towards the end of pregnancy; it is situated low in the chest in tall and slender people. A high diaphragm gives rise to a relatively wide cardiac shadow, whereas the low diaphragm of the tall thin man is associated with a long and narrow cardiac shadow. Depression of the sternum may distort the heart and give rise to a spurious widening of the cardiac shadow. Similarly, as can well be imagined, pronounced kyphoscoliosis of the thoracic spine both distorts and dislocates the heart and gives rise to bizarre radiological appearances. Only when these anatomical factors have been

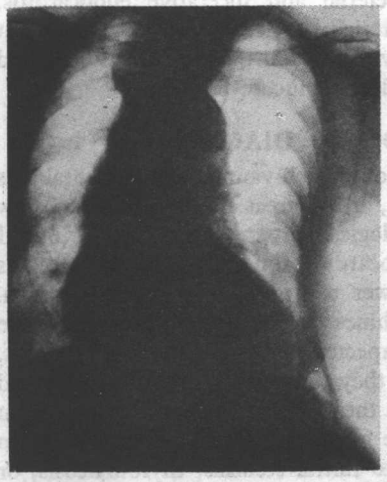

Fig. 1.—'Boot-shaped' heart showing left ventricular enlargement and unfolding of aortic arch.

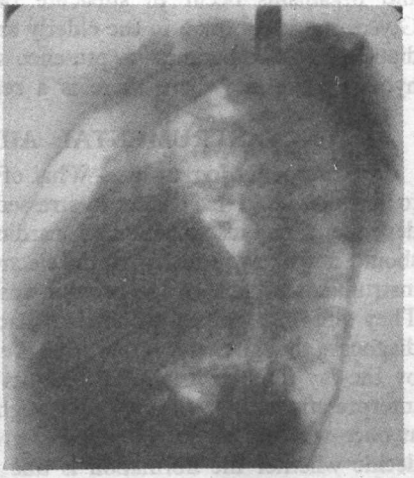

Fig. 2.—Left anterior oblique view showing left ventricular prominence towards and overlapping the spine.

considered and allowed for is it safe to come to any conclusions as to cardiac size and shape.

Otherwise the shape of the heart as revealed by radiography is dependent primarily on which chamber, or chambers, have suffered enlargement.

Left Ventricular Enlargement.—Left ventricular enlargement gives rise to a bold left border to the cardiac shadow, and in extreme cases this is described as the 'boot-shaped' heart (*Fig.* 1) (with the toe-cap pointing to the left axilla), especially if it is associated with radiographic unfolding of the aorta: the transverse diameter is markedly increased in the postero-anterior view. In the left anterior oblique view (*Fig.* 2) the back of the heart, which is the left ventricle, assumes prominence and may resemble in size and shape half a medium-sized grapefruit.

Right Ventricular Enlargement.—Right ventricular enlargement causes a slighter, generalized widening of the cardiac shadow and is therefore not

so easily recognized in the postero-anterior view, unless the interventricular septum is sufficiently rotated to give the apex of the left ventricle a higher situation than normally; this is known as the *cœur-en-sabot* appearance (*Fig.* 3), and is well seen in many cases of Fallot's tetralogy. In the left anterior oblique view, right ventricular enlargement can be made out by the increased prominence of the anterior border of the heart shadow.

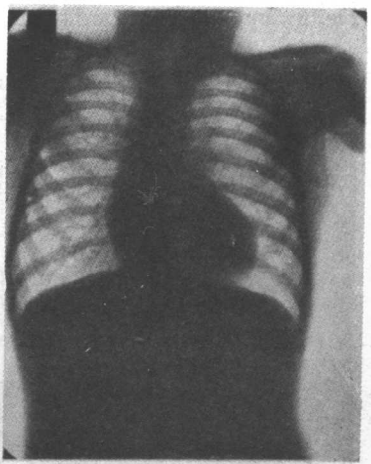

Fig. 3.—*Cœur-en-sabot* in Fallot's tetralogy; the apex of the left ventricle is higher in the chest than in the boot-shaped heart.

Left Auricular Enlargement.—Enlargement of the left auricle is characteristic of mitral disease. If it is considerable it may be seen from the front as a prominence along the left border of the cardiac shadow between the pulmonary artery and the left ventricle; and not infrequently it is possible to recognize a double shadow along the right border where the right auricle overlaps the left (*Fig.* 4). But left auricular enlargement is best made out in the right anterior oblique position, where the lower three or four inches of the barium-filled oesophagus are pushed backwards as though by a large convex lens (*Fig.* 5). Often in these circumstances the normal impressions

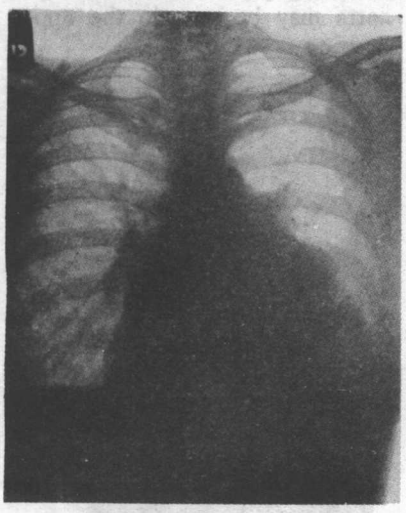

Fig. 4.—Enlargement of the left auricle in mitral disease, showing a prominence between the pulmonary artery shadow and the left ventricular shadow on the left border, and a double contour on the right border consisting of left auricle above and right auricle below.

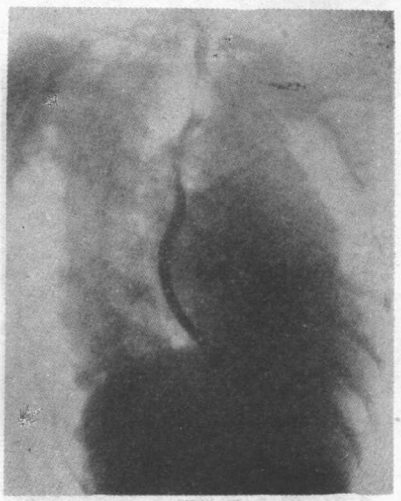

Fig. 5.—Right anterior oblique view showing a bowing of the lower part of the barium-filled oesophagus by the enlarged left auricle. Above this the aortic and left bronchial impressions can be seen to be fused.