4. The difficulties surrounding the radiology of heart disease largely disappear if, instead of looking for general enlargement, attention is centred upon the particular chambers or great vessels which may be modified in size, shape, or position.

5. If electrocardiography has reached its present sound position by the support of physiology, radiology of the heart will establish best its rightful position by enlisting the support of anatomy and morbid anatomy.

In conclusion, special care should be taken not to distress a patient by confronting him with x -ray findings as if they implied an additional diagnosis or a complication which magnified his disease. For instance, the cardiac enlargement of hypertension is an associated sign which should not be regarded as a disease in itself, but as a completion of the diagnosis.

RADIOLOGY IN HEART DISEASE*

BY

PETER KERLEY, M.D., D.M.R.E.

ASSISTANT RADIOLOGIST TO WESTMINSTER HOSPITAL; RADIOLOGIST
TO THE ROYAL CHEST HOSPITAL

(With Special Plate)

Radiology of the heart is still in its infancy. While the radiologist can justly claim to have advanced the diagnosis and pathology of pulmonary and gastro-intestinal diseases, he has so far added comparatively little to the study of heart disease. He has certainly greatly improved the diagnosis of enlargement of the heart and aorta, but in the realm of pure pathology he has contributed practically nothing. The modern conceptions of the causation and course of heart diseases have not been materially influenced by x -ray investigations. The reasons for this deficiency are largely technical. First, the rapid movement of the heart does not lend itself to accurate radiography, and although the necessary apparatus for cardiac cinematography has been perfected the cost is prohibitive. Secondly, the radiograph shows only the borders of the heart. We have not yet found a drug which, when injected into the blood stream, renders the blood more opaque than the muscular wall of the heart, and there is not much hope of the discovery of such a drug. Until the production of a cheap photo-electric cell, cardiac cinematography will not be a practical proposition.

THE VALUE OF SCREENING

The most important method of x -ray examination of the heart is simple screening. On the fluorescent screen we can at once form an accurate estimate of the size of the heart, and the experienced observer can tell at a glance whether the heart is enlarged or not. The screen also shows the position of the heart relative to the chest wall and to the other intrathoracic organs. Variations in the position of the heart due to deformities of the bony thorax or to fibrotic or emphysematous changes in the lungs can be most misleading and, indeed, impossible to diagnose by the usual clinical methods. Such variations are perfectly clear on the fluoroscopic screen.

The shape of the heart, which varies considerably both in health and in disease, is also noted on the screen, and by rotating the patient into different angles the observer is able to tell whether the alteration in shape is due to enlargement of any of the chambers of the heart. Certain diseases of the heart cause a characteristic alteration in shape, such as the boot shape in aortic incompetence or the flask shape in pericarditis with effusion. The pulsa-

tion of the heart as observed on the screen does not indicate as much as one would expect. The rate of pulsation of the heart is too fast for fine variations to be seen by the naked eye. Gross variations such as occur with heart-block and auricular fibrillation are easily identified, but these are conditions in which clinical diagnosis is easy.

ORTHODIAGNAPHY: TELERADIOGRAPHY: KYMOGRAPHY

There are three other methods of radiographic investigation. The oldest of these still in common use is known as orthodiagnaphy. On leaving their source x rays diverge widely, and, as Figure I shows, the image of the

(a) Showing distortion due to tube R being too close to object. (b) Showing absence of distortion with parallel rays coming from a distance.

heart, if too near the source, is grossly distorted. To overcome this difficulty an instrument known as the orthodiagraph has been devised. With this instrument only the central ray, which does not diverge, is employed. This central ray appears on the screen as a tiny point of light, and is slowly manipulated by the observer round the shadow of the heart. At suitable points the observer marks the edge of the heart on the screen with a pencil, and the final result is a drawing of the type shown in Figure II. As only the central undeviating ray has been

FIG. II.—Orthodiagram after Groedel.

used, accurate measurements of the heart can be made on the drawing.

The second method, known as teleradiography, has many advantages over orthodiagnaphy, but is more expensive. Powerful apparatus now enables radiographs to be taken at a distance of two metres, when all the rays are more or less parallel and distortion is negligible. The heart measurements can therefore be made on the actual radiograph.

The third method, known as kymography, has only recently been devised. This method makes use of a fine

* Read in opening a discussion at a joint meeting of the Sections of Radiology and Medicine at the Annual Meeting of the British Medical Association, Dublin, 1933.

slit or diaphragm, which is fixed over the border of the heart. The x rays are projected through this slit, and a film is made on which is recorded a zigzag image representative of the pulsations of the heart. The interpretation of a kymograph is by no means easy, and the claim of German workers that this method yields more information than the electrocardiograph has yet to be substantiated.

THE NORMAL HEART

The x -ray appearance of the normal heart varies considerably with the age, sex, and habitus of the individual. In children up to the age of 4 years the heart relative to the width of the chest is very large, and both heart and great vessels appear as an opaque, pear-shaped opacity, with an even, regular border on each side. From 4 years of age until puberty the heart shadow gradually becomes smaller, and a well-defined notch separates it from the vascular shadow. Irrespective of sex or habitus, the heart shadow at puberty is generally of the long, narrow type. From puberty until the age of 21 the heart gradually changes in form, until it finally assumes one of the three normal adult shapes. In the average adult the heart shadow is of the so-called oblique type, its transverse diameter bearing a relation to the chest width of 1 to 2. This ratio is known as the heart-lung coefficient. In wide-chested individuals with a high diaphragm the heart lies transversely rather than obliquely in the chest, and the heart-lung coefficient may be as low as 1 to 1.9. Roughly speaking, the transverse heart is seen more often in women than in men. In long, narrow-chested individuals with low diaphragms the heart shadow appears very narrow, and the heart-lung coefficient may be as high as 1 to 4.

The right border of the normal heart shadow is formed by the right auricle, which is separated from the shadow of the ascending aorta by a well-defined notch. The left border is formed partly by the edge of the left ventricle and partly by the edge of the appendage of the left auricle. The latter is separated by a notch from the shadow of the pulmonary artery. The popular idea that a notch separates the ventricular shadow from the auricular one is erroneous. The two shadows merge together imperceptibly, and it is only when the heart is diseased that they appear as separate entities. The heart-lung coefficient is a reasonably accurate measurement for practical purposes, and provided the ratio is not lower than 1 to 1.9 it can be taken that the heart is not enlarged. This measurement can easily be made on the screen, and is more reliable than clinical estimation of size. Every doctor is aware of the unreliability of percussion of the heart in obese and emphysematous subjects. It is not generally known how many normal subjects are suspected of cardiac enlargement. I have seen perfectly healthy men rejected for life insurance or jobs involving physical strain because percussion and palpation suggested that the apex beat was outside the nipple line. Screen examination, with a mark on the nipple, showed the heart to be well inside the nipple line.

In addition to the heart-lung coefficient a complicated system of radiographic heart measurements has been worked out. This system is too detailed to go into in this paper, but a few observations on its value are worth noting. Provided the technique is constant, the measurements are extremely accurate, and reveal minute alterations in the size of the heart. In clinical practice the system is seldom required, but it has proved of the greatest value in many physiological investigations, and particularly in the investigation of the athletic heart. During a great epidemic of diphtheria in Leipzig in 1915 Dorner carried out a large number of measurements on infected children, and found that radiographic evidence of cardiac enlargement was present long before symptoms

or clinical investigation pointed to cardiac involvement. These measurements have also been helpful in establishing enlargement of the heart from hyperthyroidism.

THE "SMALL" HEART

The small or narrow heart was unknown before the radiological era. There is no radiographic method of estimating either the volume or weight of the heart, and the radiological definition of a small heart simply means that it appears small relative to the width of the chest. The situation of the diaphragm rather than the shape of the bony thorax appears to be the chief factor in the production of the so-called small heart. If the diaphragm is in the fifth interspace the heart appears wide; if it is lower than the sixth interspace the heart appears small. When for any reason the diaphragm is depressed the heart rotates slightly to the right and forward, and consequently appears smaller on a radiograph. Wenckebach suggests that, if the diaphragm is situated unusually low, the effect of its respiratory contractions on the circulation is diminished, so that the amount of blood passing into the heart is lessened and the heart is actually smaller. This is very doubtful, as there are many common diseases which lower the diaphragm and are not accompanied by a lowering of the blood pressure. That the small heart is apparent, not real, is proved by the alteration in the radiological size during pregnancy. I have made some observations on changes in the shape and size of the heart during pregnancy, and have found that a woman, who normally has a small heart, has a transverse and apparently wide heart during the last two months of pregnancy. This alteration occurs with the elevation of the diaphragm, and about six weeks after delivery the diaphragm is back in its normal position and the heart again appears to be small. But for the frequency with which a small heart is seen in cases of tuberculosis it is doubtful if the term would ever have been coined. Many observers have suggested that the toxæmia associated with chronic pulmonary tuberculosis causes atrophy of the heart muscle and a real decrease in the size of the heart. Necropsy findings certainly do not support this view, and I would suggest that the compensatory emphysema which always accompanies pulmonary tuberculosis depresses the diaphragm, with consequent rotation of the heart to the right and an apparent diminution in size.

ENLARGEMENT OF THE HEART

The x -ray diagnosis of enlargement of the heart may be very simple or very difficult. In cases of heart failure associated with arterial disease or chronic nephritis the enlargement is obvious, and when the increase in size is associated with valvular or congenital disease the shape of the heart simplifies the diagnosis. In borderline cases, however, in which there is no heart failure and no gross alteration in shape, there may be great difficulty in arriving at the correct diagnosis. Adults with tachycardia or precordial pain form the bulk of the borderline cases. A screen examination of a rapidly pulsating heart often gives the impression of enlargement; therefore reliance should be placed on orthodiagrammatic measurements and telerradiographs, which must be carefully correlated with the clinical findings. In tachycardia associated with myocardial disease the measurements invariably reveal a real enlargement. I do not believe that x -ray examination is of any value in the diagnosis of early myocardial disease. Symptoms may point to the presence of this condition, but I have never yet seen a case in which x -ray evidence of enlargement preceded electrocardiographic alterations. I would go so far as to state that in doubtful cases an x -ray diagnosis of enlargement of the heart should never be made unless there is definite clinical evidence to support it. In many of these cases

of tachycardia and precordial pain the x -ray examination shows elevation of the left side of the diaphragm due to gaseous distension of the stomach and splenic flexure. Measurements show the heart to be within the normal limits, and dietetic treatment brings relief.

A differential x -ray diagnosis of the cause of cardiac enlargement is not possible unless the enlargement is associated with some definite alteration in the shape of the heart or great vessels. For example, an enlarged pulmonary artery points to congenital disease and an enlarged aorta to acquired disease. The shape and position and density of the aorta offer valuable evidence as to the nature of enlargement of the left ventricle. Although we are not primarily dealing with aortic disease at this meeting, I would like to emphasize the following points. An aorta which shows up denser than the left ventricle is atheromatous. An aorta which on a teleradiograph is not dilated but is as high as the level of the first costal cartilage is atheromatous unless there is some fibrotic lesion in the lungs to account for its high position. Visible atheromatous plaques in the aorta are not necessarily of serious import. I have had under observation for seven years a man with dense plaques in his aorta. He has never had any aortic pain nor any signs of cardiac disease, and he leads a normal life.

CHANGES IN THE SHAPE OF THE HEART

The most characteristic alterations in the shape of the heart are produced by congenital and valvular diseases. Nearly all congenital lesions with an admixture of the venous and arterial streams show enlargement of the pulmonary artery, which appears as an additional bulge on the left border of the heart just below the aortic knob. An exception to this is the tetralogy of Fallot, in which there is a defect at the level of the pulmonary artery and the heart has what is known as the "*cœur en sabot*" shape. Coarctation of the aorta reveals itself radiographically, first, by an alteration in the shape of the heart; the left ventricle is enlarged and the aortic knob is invisible. Secondly, there is visible erosion of the lower borders of the ribs due to the dilatation of the intercostal arteries forming a collateral circulation. Thirdly, in many cases of coarctation a left oblique view reveals the actual stenosis, and the bifurcation of the trachea stands out as if it were drawn in on the radiograph.

Aortic regurgitation and mitral stenosis cause typical alteration in the shape of the heart shadow. The well-known boot shape of aortic regurgitation needs no detailed description, but there are so many variations in the shape produced by mitral stenosis that they deserve special mention. In a well-established case of mitral stenosis the heart shadow has what is described as the loaf shape. The right border is unaltered. The left border shows a small aorta, a large pulmonary artery, and an extra bulge caused by enlargement of the appendage of the left auricle. In many cases this extra bulge is not visible, and all the enlargement of the left auricle is posteriorly and to the right. An enlarged left auricle is often visible through the heart shadow, and in the first oblique position fills up the normally clear posterior mediastinal space. In many cases the left auricle enlarges so much to the right that it overlaps the shadow of the right auricle and actually forms the right border of the heart shadow. Occasionally aneurysmal dilatation of the left auricle takes place, and the radiograph shows the auricle to be reaching almost to the right axillary line.

MITRAL STENOSIS

It is not so much in the diagnosis of mitral stenosis as in the diagnosis of its complications that radiography is

of great help. One often hears of an enlarged left auricle displacing the oesophagus and causing dysphagia. Any enlargement of the left auricle displaces the oesophagus backwards, but in my experience neither the displacement of the oesophagus nor the pressure of the auricle on it is responsible for dysphagia. It is only when there is associated heart failure and chronic congestion of the liver that dysphagia occurs. In such cases the oesophageal veins are grossly dilated, and this is responsible for the difficulty in swallowing. The oesophagus is an elastic and very mobile organ, which can withstand extreme degrees of displacement without any disturbance of its function. A less frequent complication of mitral stenosis is paralysis of the left recurrent nerve. This is due to the enlarged left auricle pressing upwards into the bifurcation of the trachea and causing pressure on the nerve between the left main bronchus and the aorta. Splaying of the bifurcation of the trachea is very commonly associated with mitral stenosis and may be responsible for haemoptysis. I have seen several cases in which haemoptysis occurred and in which there was no radiographic or clinical evidence of hyperaemia of the lungs. Probably the pressure caused oedema of the tracheal mucosa, and slight coughing caused rupture of small vessels.

HYPERAEMIA, OEDEMA, AND COLLAPSE OF THE LUNG

Passive hyperaemia of the lungs caused by mitral stenosis or heart failure gives remarkable and very varied x -ray appearances. These variations throw much light on the pathology of the condition. While many of them are simply the result of the hyperaemia being observed at different stages, others are related to unsuspected pulmonary diseases, and some are certainly due to congenital abnormalities of the vessels themselves. The x -ray appearances of the early stages of passive hyperaemia are generalized loss of translucency and marked increase in the density of the vascular shadows. These shadows are also coarser than normal, and their ramifications are visible right out to the periphery of the lungs. The hilar shadows are enlarged, and the movements of the diaphragm are restricted. If the hilar shadows are observed closely on the screen pulsation can be seen on them. The loss of translucency is due to compression of the alveoli by the distended capillaries, and the enlarged hilar shadows and coarse vascular markings result from distension of the pulmonary arteries and veins. Assman states that in very early stages of mitral stenosis the enlarged pulmonary artery presses on the left upper lobe vein and causes hyperaemia localized to the left upper lobe. I have never seen a case showing such localized hyperaemia. If the hyperaemia becomes chronic the loss of translucency is more marked, and the vascular striae in the lungs become still coarser and wider because the perivascular lymphatics now show up. The hilar shadows are grossly enlarged on account of the additional shadows thrown by oedematous bronchial glands, and the interlobar fissure between the right upper and middle lobes is strikingly clear and thickened. This thickening of the interlobar fissure is due to transudation into the large lymphatic, which connects the superficial pulmonary plexus with the parietal pleura. On good lateral views it will be seen that the greater fissures are similarly affected. The shadow accompanying the second rib also shows thickening. It was previously thought that this shadow was pleural in origin, but Knutson has shown that it is produced by a sheath of connective tissue containing the superior thoracic artery and vein and a large branch of the first thoracic nerve. The clarity of this shadow in mitral stenosis is certainly due to swelling of the vessels in Knutson's sheath, and in many doubtful

cases of mitral disease I have found its thickened appearance to be of great diagnostic value.

Certain cases of chronic hyperaemia give rise to a miliary appearance in the lungs, and the inexperienced observer can easily confuse this with miliary tuberculosis. The miliary spots which are scattered all over both lungs in chronic hyperaemia are caused partly by dilated capillaries seen end-on and partly by exudation of heart-failure cells into the alveoli. There are two features which distinguish miliary hyperaemia from all other miliary diseases. First, the spots decrease in size towards the periphery, and secondly, the intercostal spaces are wider than normal. Chronic passive hyperaemia is the only intrinsic condition of the lungs in which the actual volume of the lungs is increased. Although the air content of the lungs appears to be reduced by half and little or no expansion can be observed on the screen, yet the intercostal spaces are widened. When the stasis is so severe that it is transmitted to the bronchial arteries all the previous signs are intensified and, in addition, the number of lung markings is increased. The oedematous bronchial mucosa gives off much secretion, and the affected bronchi become visible and add to the general loss of translucency. It is in this advanced stage that mistakes in diagnosis are frequently made. There is considerable exudation from the tense capillaries, and this process tends to be greatest near the hilum, where the veins are largest. At the same time there is collapse of lobules which are compressed by capillaries and have their air entry diminished by oedema of the bronchioles. These areas of collapse occur where there is most interstitial tissue—that is, near the bifurcation of the large vessels and bronchi. On a radiograph they produce an appearance similar to that of bronchopneumonia, with foci coalescing. Again, the chief point in the differential diagnosis is the widening of the intercostal spaces, which may be greatest in the immediate vicinity of the most opaque area. If by any chance pleural adhesion or fibrosis is present and causing loss of normal elasticity, the addition of chronic hyperaemia may cause a large area to collapse. Such an area may be situated in any part of the lungs, and may give an appearance similar to lobar pneumonia. It may even give appearances very like those of advanced pneumoconiosis.

A severe attack of passive hyperaemia always leaves permanent radiological evidence behind it. The interlobar pleura remains thickened, and the shadows of the perivascular lymphatics persist as fine, sharp lines, most marked at the bases and near the hila. This appearance simulates fibrosis and bronchiectasis. The differential diagnosis is made on the screen by noting the normal expansion in the apparently fibrotic area. Occasionally one sees on radiographs of mitral stenosis and hyperaemia true fibrosis at the base of a lung. This fibrosis may be due to infarction, but I am more inclined to the opinion that it follows thrombosis. I have observed radiographically eight cases of infarction following operations and injuries. In all of them there was complete resolution in the infarcted area without any subsequent fibrosis. In rare cases one finds a severe degree of hyperaemia and a marked increase in the density of the pulmonary arteries out of all proportion to the heart lesion. There is little doubt that these are cases of advanced atheroma of the pulmonary arteries, and, as the condition is found in young people, it probably has a congenital basis. I am not referring to true Ayerza's disease nor to chronic cases in which the pressure in the arteries has been consistently high. Abnormalities of this nature can present the most baffling clinical signs. Until mechanical and chemical advances are made it is in the solution of such problems that the greatest scope of cardiac radiology lies.

RADIOLOGICAL DIAGNOSIS OF CARDIAC ENLARGEMENT*

BY

CRIGHTON BRAMWELL, M.A., M.D., F.R.C.P.

ASSISTANT PHYSICIAN, MANCHESTER ROYAL INFIRMARY

I propose to devote the time at my disposal in this discussion to considering a single problem—namely, the measurement of the heart and the recognition of cardiac enlargement; this is a very important problem, because enlargement of the heart generally points to heart disease. Gross enlargement can easily be recognized in most cases by palpation and percussion; but the slighter degrees of enlargement cannot be detected with certainty by these methods, and when the patient is fat or emphysematous the task becomes quite impossible without the aid of *x*-ray examination. Let us therefore consider how radiology can help us in such cases, how it is applied, and what its limitations are.

An orthodiagram (Fig. 1) or a teleradiogram gives us a frontal silhouette of the heart, which enables us to

FIG. 1.—Orthodiagram of diagonally placed heart in normal subject of average build. R and L = transverse diameter of heart to right and left of middle line. Long = longitudinal diameter.

measure with accuracy what, previously, we had been able to map out only very roughly by percussion. As an index of heart size we can take either the area of this frontal silhouette or its transverse diameter. The latter is preferable, because the borders of the silhouette, where the shadow of the heart merges with that of the great vessels above and of the liver below, are not clearly defined. Accurate radiographic measurements are of considerable value in comparative studies on individual patients. They tell us definitely whether the heart is slightly larger or slightly smaller than it was on some previous occasion, whereas percussion and palpation can only reveal comparatively gross changes in the size of the organ.

Clinically, provided the heart is not displaced, we regard it as enlarged if palpation or percussion leads us to believe that its left border crosses the mid-clavicular line or that its right border extends more than a finger-breadth beyond the sternum. Radiographically we regard the heart as enlarged when the transverse diameter of the heart shadow, as calculated from the sum of its two components to the right and left of the middle line (R and L, Fig. 1), is greater than half the internal diameter of the thorax.

* Contribution to a joint meeting of the Sections of Medicine and Radiology at the Annual Meeting of the British Medical Association, Dublin, 1933.

PETER KERLEY: RADIOLOGY IN HEART DISEASE

FIG. 1.—The boot-shaped heart of aortic regurgitation.

FIG. 2.—Mitral stenosis with chronic passive hyperaemia of the lungs. Note the thickening of the interlobar pleura and Knutson's sheath.

FIG. 3.—Miliary type of chronic passive hyperaemia of the lungs.

FIG. 4.—Chronic passive hyperaemia of the lungs with thrombosis at the base of the right upper lobe. Note the oedematous bronchial glands.