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 pneumonoconiosis.

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

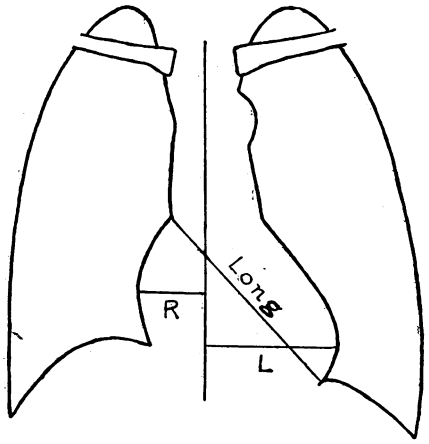

FIG. 1.—Orthodiagram of diagonally placed heart in normal subject of average build. R and L = transverse diameter of heart to right and left of middle line. Long = longitudinal diameter.

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.

Here we are faced with a difficulty. The transverse diameter of the heart shadow varies with the lie of the heart in the chest. When the diaphragm is high (Fig. 2) the heart lies more transversely, when it is low (Fig. 3) it lies more vertically. The hearts of people of "stocky" build, who are too heavy for their height, therefore tend to be wide, and those of people of "lanky" build, who are too tall for their weight, tend to be narrow in proportion to their size. These facts were perfectly

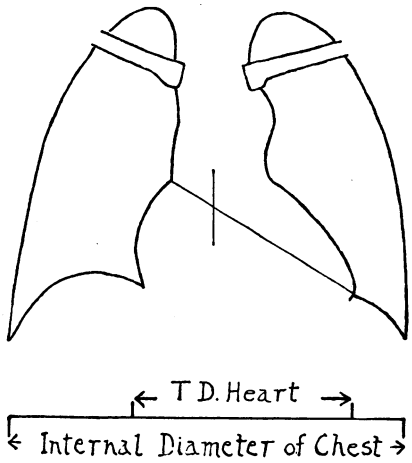

FIG. 2.—Transversely disposed heart of "stocky" normal subject.

familiar to us before the days of radiology. It was well known that, in normal healthy subjects, an apex beat situated in the sixth intercostal space tended to lie nearer to the middle line, and one situated in the fourth space farther from the middle line than one situated in the fifth space. In other words, in assessing cardiac enlargement, we must take into account not only the width of the heart but also the lie of the heart, which varies with the shape of the chest and the build of the subject. The

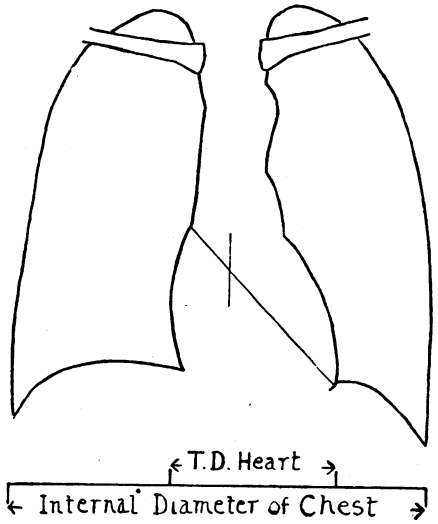

FIG. 3.—Vertically disposed heart of "lanky" normal subject.

lie of the heart is indicated radiographically by the inclination of the longitudinal diameter of the heart shadow to the vertical, the longitudinal diameter (Long, Fig. 1) being the line joining the apex of the heart to the angle formed by the junction of the shadow of the right auricle with that of the great vessels.

To render comparable measurements obtained by different workers, it is important that teleradiograms (and orthodiagrams) should be taken under standard conditions. The examination should be made in the erect posture, with the breath held at the end of a normal expiration; and, since the heart is larger in diastole than in systole, the exposure should be not less than one second's duration, in order to include both phases of the cardiac cycle.

The transverse diameter of the heart shadow in normal healthy subjects varies directly with the body weight; but, from what I have said about the lie of the heart, it will be clear that it varies inversely with the height of the subject. Hodges and Eyster[1] have worked out, statistically, a formula for predicting the transverse diameter of the heart from weight and height: $TD = 95.9 + (W \times 0.8179) - (H \times 0.1941)$. In this equation the transverse diameter of the heart shadow (TD) is expressed in millimetres, the weight (W) in kilograms, and the height (H) in centimetres. Eyster also introduces a correction for age, but this makes comparatively little difference to the result, and I do not propose to consider it here.

Let me now refer briefly to certain limitations of this method of heart measurement. The frontal silhouette is a two-dimensional picture, whereas the heart is a three-dimensional organ. Hence our estimate of the cubic volume of the heart can only be approximate when based on this measurement alone. Nevertheless, we must remember that in the pre-radiological era the diagnosis of cardiac enlargement rested entirely on the results of percussion and palpation. Even under the most favourable circumstances the measurement of the heart by these means is much less accurate than when it is taken from the radiographic frontal silhouette, while in emphysematous and obese subjects it is rarely possible to obtain any idea of the size of the heart without x-ray examination.

Further, under pathological conditions, enlargement of the heart rarely involves all chambers to an equal extent. Some are more affected than others, as, for example, the left auricle and the right ventricle in mitral stenosis, or the left ventricle in hyperpiesis. These lesions are clearly shown by changes in the shape of the heart outline, as seen in the postero-anterior and in the two oblique positions. A change in shape is therefore of even greater importance than a change in size of the heart shadow, since it gives us a clue to the cause of the enlargement. Such regional enlargement is easily recognized in the radiogram, but cannot be satisfactorily expressed in simple numerical terms, like the transverse diameter of the frontal silhouette. While admitting, therefore, that we have not yet reached our final objective, it must be recognized that it is to radiology that we owe the great advance which has been made in accuracy of measurement. The size of the heart varies considerably in different healthy individuals, and it is not until we pass beyond the range of normal variation that we can speak of cardiac enlargement as pathological. What is the extent of that range? Bedford and Treadgold[2] found that in 86 per cent. of a series of 116 healthy adult males the observed transverse diameter of the heart shadow fell within $\pm$ 10 per cent. of the predicted normal value as calculated from Eyster's formula. Of the remainder, in 9 per cent. the hearts were smaller, and in 4 per cent. larger, than those of the intermediate "normal" group. This leads on to the further consideration of the pathological significance of cardiac enlargement.

At the Olympic Games in Amsterdam in 1928 Ellis and I[3] examined a group of over 200 athletes, including long-distance runners, sprinters, cyclists, weight lifters, and competitors in various other kinds of sport. We found that the average transverse diameter of the heart shadow in each group of athletes was approximately proportional to the body weight. There was one exception,

however—namely, the group of marathon runners; in this group the heart was very much larger than the body weight would have led one to predict. It would be impossible to find any group of men whose circulatory mechanism is functionally more efficient than that of Olympic marathon runners, and it would be absurd to regard the cardiac enlargement in this case as pathological. Similar findings have been reported by other workers[4 5] in men who participated in other types of sport entailing prolonged and severe exertion, notably ski-running and cycling. The same is true throughout the animal kingdom, the hearts of athletic animals being heavier in proportion to their body weight than those of animals which lead a more sedentary life. One is bound, therefore, to conclude that slight cardiac enlargement is, by itself, insufficient evidence on which to base a diagnosis of heart disease; although, when considered in conjunction with the other clinical findings and the habits of the patient, it may be of great value in diagnosis.

I have confined my attention to cardiac enlargement because I do not believe that one is ever justified in regarding a heart which is too small as pathological, unless there be additional evidence to support the diagnosis. Bedford and Treadgold[2] found a heart with a very small transverse diameter in a member of the Schneider Cup team, and Ellis and I[3] found a similar heart in the Olympic champion for the 100 and 200 metres!

In conclusion, let me summarize my remarks by endeavouring to answer the question, "What can *x*-ray examination contribute to the diagnosis of pathological cardiac enlargement?" I would submit that it can make at least three important contributions.

1. It can tell us all that we can learn from percussion and palpation regarding cardiac enlargement, and it can do so with a much higher degree of accuracy than either of these methods.

2. It can give us reliable information regarding slight changes in heart size and the slighter degrees of cardiac enlargement, even under the most unfavourable circumstances. Although slight cardiac enlargement by itself is not sufficient ground on which to base a diagnosis of heart disease, it may sway the balance when considered in conjunction with evidence from other sources.

3. *X*-ray examination can reveal changes in the shape of the heart outline. These changes tell us which chambers are enlarged, and so indicate the probable cause of the enlargement. This, in my opinion, is its most important contribution. The frontal silhouette alone gives us a far more accurate idea than does percussion of the shape of the heart, whereas examination in the two oblique positions yields additional information of which percussion can tell us little or nothing.

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SOME OBSERVATIONS ON RICKETS,

WITH SPECIAL REFERENCE TO ITS OCCURRENCE IN PIGS

BY

JOHN POOL McGOWAN, M.A., M.D.

ABERDEEN

(*With Special Plate*)

In describing these observations on rickets in pigs, which were made during the period 1922–31, the question of the causation of the disease, whether due to deficiency of calcium, phosphorus, or vitamin D, comes under discussion.

CALCIUM DEFICIENCY

Since rickets was first observed the question of calcium deficiency as a cause has always been a major point for debate—for example, by Korenchevsky (1922).

Hutyra and Marek (1913) deal at great length with this aspect of the disease in pigs. Elliot, Crichton, and Orr (1922), also working with pigs, alleged that rickets in these animals could be prevented by adjusting the CaO percentage of their ration to that of the P_2O_5. As, however, 10 c.cm. per pig per day of the antirachitic substance cod-liver oil was administered in the basal ration, the condition resulting from the calcium deficiency could not have been rickets. Evidence will be adduced later to show that, in all likelihood, the lesions produced were those of osteoporosis or, very exceptionally in the circumstances, of osteoporosis complicated by a condition of rickets already present before the animals were submitted to the experiment.

During the years 1922–6 the opportunity occurred of observing, among large breeding stocks with an annual population of hundreds of susceptible pigs, the effect of "balancing" in the ration the CaO with the P_2O_5. Rickets as a natural disease in pigs occurs most frequently in January and February, and in the stocks just alluded to, fed on this "balanced" ration and during these months, hardly a pig which could be the subject of the disease escaped it. Zilva, Golding, and Drummond (1924) performed experiments on pigs with results which confirmed these observations, and showed that, while the administration of cod-liver oil prevented the occurrence of rickets, the "balancing" of the CaO with the P_2O_5 of the ration was quite ineffectual. Subsequent to the publication of their paper a "repeat" of their experiments confirmed these results. Later still, the specimens from an experiment, in which groups of "summer" pigs (free, therefore, from "latent" rickets) were fed, some on a calcium-deficient ration, others on this ration with the addition of a vitamin D preparation, and others again on this ration with cod-liver oil added, were examined by me. Advanced osteoporosis with fractures of the bones was present in all groups, and particularly in that receiving cod-liver oil. Rickety changes were entirely absent. In 1931 I was requested by Dr. R. D. Sinclair* (who has allowed me to refer to his results) to report on the specimens derived from a somewhat similar experiment. Groups of pigs were fed, some on a calcium-deficient ration, others on the same ration with exposure to sunlight, others on the same ration with exposure to ultra-violet light, and others again on the same ration with the addition of cod-liver oil.† Osteoporosis of an advanced type, often with fractures, was present in the bones of all the groups: rickety changes were absent (see Special Plate).

The following provisional interpretation of these collected observations seems justified—namely, that calcium deficiency produces osteoporosis which cod-liver oil is practically powerless to prevent; and that, while administration of calcium does not prevent "latent" rickets

* *Scientific Agriculture* (1933, xiii, 489) records the other details of this experiment.

† Observations were also made on a parallel series of groups where the P_2O_5 was "balanced" with $CaCO_3$. Osteoporosis, less in degree than in the calcium-deficiency series and worst in the basal group, was present except in the cod-liver oil group. The most feasible explanation of these phenomena would appear to be along the lines of a condition of alkalosis interfering with assimilation, remedied to some extent by a state of acidosis produced by vitamin D (*vide infra*).