The apical murmurs of an aortic insufficiency and a mitral stenosis may resemble one another, but there is this difference, that the Flint murmur is associated with a thumping, thudding first sound, whereas the true presystolic murmur of a mitral stenosis is followed by the snappy first sound.

The Flint murmur was present in our series twice in 11 cases (18.1 per cent.), and the Graham-Steell has been found twelve times in 36 cases (33.3 per cent.). A differential diagnosis of considerable interest if not of great importance from the practical side (as in either event the recruit should be rejected) must be made in quite a large number of cases.

CONCLUSIONS. The differential diagnosis rests on the findings revealed by inspection, palpation and percussion, with the auscultatory findings, excepting the quality of the first sound at the apex, holding a subordinate place. The most important features in favor of the diagnosis of aortic insufficiency (not arranged in the order of importance) are: (1) Displacement of the apex-beat. (2) Heaving feel of the apex impulse to the palpating hand. (3) Hypertrophy of the left ventricle. (4) Vascular signs, *i. e.*, marked pulsation of vessels, Corrigan pulse, capillary pulse, systolic tone in brachial, with arm above the head. (5) Blood-pressure increase of pulse-pressure, marked discrepancy between the arm and the leg pressures.

In favor of a mitral stenosis are—

1. Loud snappy first sound at the apex unless marked by an insufficiency of the mitral valves.
2. Absence of apical displacement and of cardiac hypertrophy.
3. Systolic tap or shock to the palpating hand.
4. Absence of vascular signs.
5. Absence of any characteristic blood-pressure phenomena.

THE CARDIOTHORACIC RATIO: AN INDEX OF CARDIAC ENLARGEMENT.

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THE question of heart size, especially in its relationship to hypertrophy and dilatation, is important to the civilian as well as to the military physician. Its knowledge is necessary in the diagnosis and interpretation of certain systolic murmurs as well as in the prognosis of cardiac, nephritic and vasosclerotic conditions.

For example, a systolic murmur at the apex, with the definite knowledge of cardiac enlargement, will certainly differentiate an actual valvular insufficiency (endocarditic or myocarditic) from a functional murmur. Without such knowledge an accurate diagnosis is often impossible.

Let us see how important this knowledge of the size of the heart would be in prognosis. To illustrate this we need not go very far. For instance, a case of chronic nephritis with cardiac enlargement is surely of graver significance than without it. The same might be said of cases of hyperthyroidism in which cardiac enlargement would indicate permanent damage to the heart by the thyrogenic poison. Thus one might go on indefinitely with such examples. Suffice it to say, however, that enough instances might be found to warrant investigation into problems that might yield us a method of exact, yet simple determination of the cardiac size.

Cardiac hypertrophy and dilatation are terms frequently used though seldom verified except in conditions of extreme enlargement. Errors in this direction are common enough to justify us in the opinion that many a diagnosis is confused because the method of estimating the heart's size is faulty. This fact was brought out quite strikingly when the writer was engaged in heart work in the Army.

The physician in private practice relies upon percussion for the determination of cardiac size. But to percuss out a heart accurately for diagnostic purposes requires fine technic and is, at best, difficult, especially when slight or moderate enlargement exists. In fact, the London School of Cardiologists denies its possibility altogether even by the most expert. Personally, I cannot accept this extreme viewpoint. To me it seems that much information may be obtained by careful percussion of the heart. I must admit, however, the difficulty occasionally experienced and the great possibility of error.

The location of the position of the apex-beat for the same purpose has recently been reclaimed by Thomas Lewis and his disciples. Our experience on this point has shown us that the reliability of this sign as an index of cardiac enlargement is only moderate. It is of no help in early cases in which the apex-beat is little if at all displaced. This much may be said: (1) that cases of marked enlargement will cause displacement of the apical impulse to the left of the midclavicular line and sometimes downward; (2) that in spite of cardiac enlargement, such as must exist in definite forms of aortic regurgitation, the apex-beat is often within the midclavicular line; (3) that widely diffused apex-beats, as often occur in thin, nervous individuals, frequently seem outside of the midclavicular line even though the heart be unusually small; (4) that occasionally the point of maximum intensity cannot be located because of the diffusion of the apex-beat.

The other methods of investigating the heart's size are the instrumental ones. They consist of polygraphy, electrocardiography and orthodiography.

The polygram is occasionally useful in the determination of right ventricular hypertrophy. In this condition the outstanding feature is the retraction or negative type of apical pulsation. (See Fig. 1.)

But this can usually be visualized on the chest, so can the retraction of the third, fourth and fifth interspaces on the left side and the negative type of epigastric pulsation. By physical examination as much information is elicited as by the polygraphic studies. Thus the usefulness of the latter is minimized.

The electrocardiogram is very reliable. Enlargement of the auricle is evidenced by an increase in the size of the *P* wave. Hypertrophy of the ventricles is indicated by the downward displacement of the *R* wave in one lead and the lessening of its amplitude in another direction. Thus in right ventricular hypertrophy, such as in a case of mitral stenosis, one would get such a tracing. (See Figs. 2, 3, 4, 5, 6, 7.)

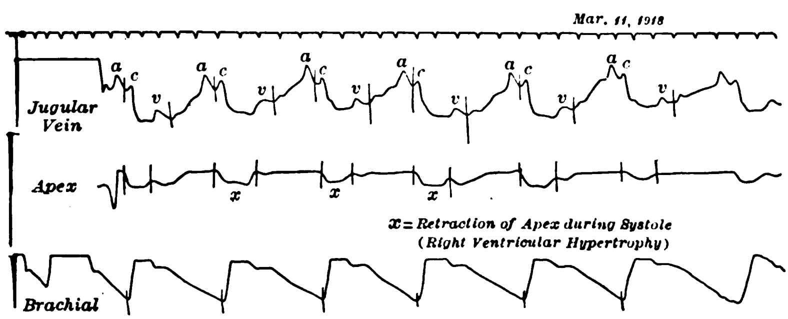

FIG. 1

The disadvantage, however, is that electrocardiography, which requires special training and technic, is not within the reach of the average practising physician. It must be repeated here that we started out to find a simple and easily applied method that should be sufficiently accurate for scientific diagnosis.

Hence we come to the orthodiagram or the roentgen-ray plate taken with the tube at six feet or more from the patient. This distance is chosen because of the divergent or enlarging effect of roentgen rays which occurs when the tube is placed nearer the patient than six feet. At this distance the rays become parallel, thus casting a true image of the heart on the roentgen-ray plate.

This method is simpler and quicker than the original orthodiagraph of Levy-Dorn and Groedel.

The roentgen-ray image of the heart is then perpendicularly bisected and the distance to the right and to the left of the median line is recorded. Thus we say the left border is 8 cm. and the right 3.5 cm. from the midline.

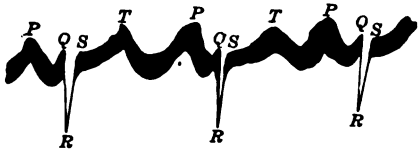

Fig. 2.—*R*, downward in Lead I.

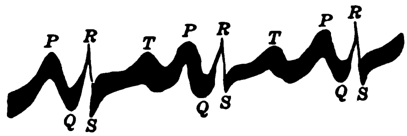

Fig. 3.—*R*, small in Lead II.

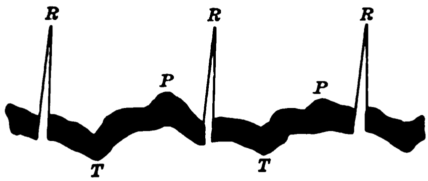

Fig. 4.—*R*, Upright in Lead III.

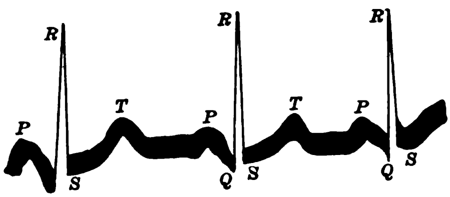

Fig. 5.—Left ventricular hypertrophy. *R*, upright and large in Lead I.

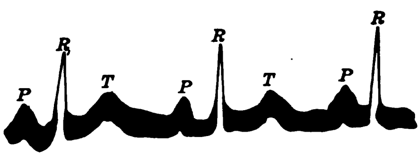

Fig. 6.—*R*, smaller in Lead II.

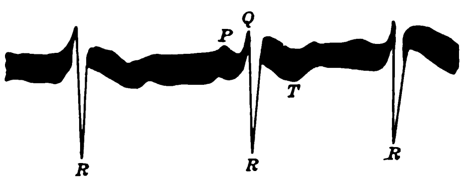

Fig. 7.—*R*, inverted in Lead III.

This method, though apparently reliable, is objectionable because there are no absolute standards for normal individuals with which to compare the resultant figures. Some text-books, however, give 4.4 cm. to the right and 9.1 cm. to the left as the upper marginal limits. I cannot agree with them, because I have seen absolutely normal hearts in which the left borders were as much as 13 cm. from the midline, while in cases of chronic valvular disease with undoubted hypertrophy corresponding diameters from 7.5 to 9 cm. were not rarities. Although paradoxical on the surface, its truth was many times emphasized on the writer.

This leads us to the question of the comparison of the cardiac diameters before and after the onset of valvular disease. If this were practicable, it would certainly tell us whether or not the heart in question was enlarged. For example, an adult develops rheumatic fever and the roentgenogram shows the left cardiac border to be 8.5 cm. and the right border 4 cm. from the midline. He develops in the course of his illness mitral valvular disease. Two years later the orthodiagram shows the l. b. to be 9.5 cm., and the r. b. 4.5 cm. from the midline. Thus the transverse diameter of the heart has increased 1.5 cm. It is apparent that a hypertrophy has taken place, principally of the left ventricle. Such information in the case of a systolic murmur heard at the apex would help a great deal in the diagnosis and the prognosis of the same.

Perhaps this question is being overemphasized in this article. If so, it is because of the numerous errors seen especially in military practice, where the diagnosis of mitral valvular disease was made altogether too frequently; certainly more so than is warranted by the lessons learned at the autopsy table. Also the diagnostic differentiation between the organic and the functional systolic murmur by the usual methods is inadequate.

In discussing comparative measurements of the cardiac diameters on the roentgen-ray plate before and after the onset of the heart affection I have chosen an adult for an example.

In a child whose heart is naturally growing, comparative measurements would be less valuable, because here an increase in the diameters could not be interpreted as indicative of hypertrophy.

Inasmuch as comparative mensuration such as was described would not be practicable, have we any other means of comparative measurement that could be of help? This calls for an affirmative reply.

It is based on the fact that the heart bears an almost constant relationship to its surrounding framework, the chest. This is seen in the narrow vertical type of heart found in association with the long status asthenicus chest. (See Fig. 8.)

The wide emphysematic chest has a heart of corresponding breadth and one which is more transverse and at a higher level than in the status asthenicus type.

These are the two extremes. Between them are numerous intermediary gradations. The outstanding feature in all of these is the parallelism between the form of the heart and that of the chest, as well as the constancy of the ratio between the transverse cardiac diameter and that of the thorax. (See Fig. 9.)

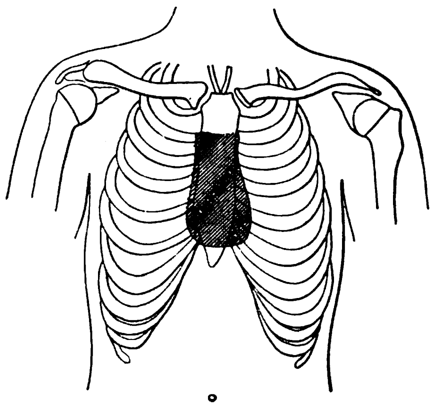

FIG. 8.—Heart, vertical, narrow "drop heart, found in status asthenicus.

It follows, then, that the newborn child, destined to be a person of asthenic proportions, will have a long tube-shaped or "drop" heart, which is the more primitive heart embryologically. The latter

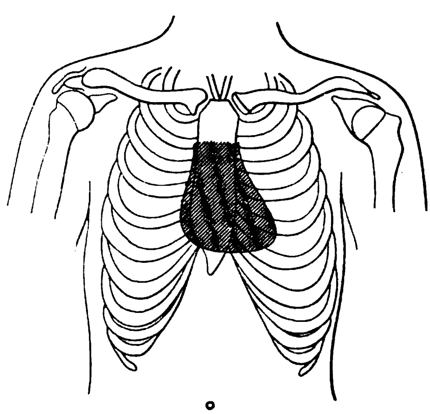

FIG. 9.—Heart, broad type, characteristic of habitus emphysematicus.

is more closely allied to the single tube from which the embryonic heart develops. Likewise the child with an emphysematic predisposition will have a heart that is in proportion to its wide chest.

This relationship is very constant, because of the parallelism in the growth of the heart and that of the chest wall. The only time there is any divergence at all is during childhood. Then the heart is a little wider proportionately, more round and lies more transversely than in the adult.

This, however, does not limit the scope of application of the relationship of the heart to the thorax for diagnostic purposes. The heart diameters in children have been studied by Sawyer[1] and Voith.[2] With their results as a basis, deviations from normal can be detected in children by comparison with the figures of the above authors.

The former found in his study of 500 young children that the apex was outside of the mammillary line in 63 per cent. of the cases. Voith, as the result of his studies, says that the hearts in children are apt to be rather right sided, the distance to the right of the median line being 50 per cent. of that to the left. In adults, of course, the right diameter is less than 50 per cent. of the left.

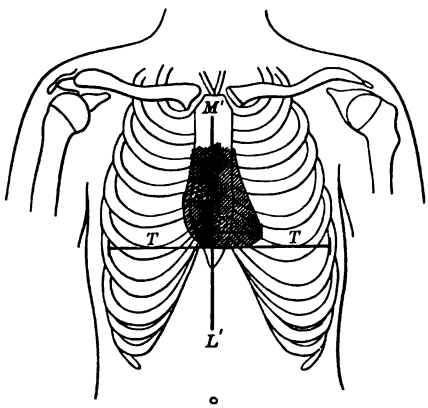

FIG. 10.—*M L'*, midline; *R*, from *M'L'* to right border (fourth space); *L*, from *M'L'* to left border (fifth space); *R* + *L*, *T. H.* (transverse diameter of heart); *T.T.* (transverse diameter of thorax); *C.T.* (cardiothoracic ratio) = $\frac{T.H.}{T.T.}$

Hence, in children we expect a high figure for the cardiothoracic ratio and the right side is slightly more prominent than in adults.

Then for practical purposes we may say that ratio between the size of the heart and chest is quite constant except when pathological processes set in. Then the relationship is disturbed, *e. g.*, the onset of valvular diseases will cause an enlargement of the heart without any accompanying change in the size of the chest. Thus it is seen that cardiac enlargement will show itself as an increase in the cardiothoracic ratio, if the latter is obtained by dividing the transverse diameter (right and left diameters) of the heart by that of the thorax taken at a given level.

Therefore, we studied a sufficiently large number of cases in order to establish the normal ratio. We also had to determine at what level we would take the chest measurement.

We finally decided on taking the thoracic measurement at its greatest diameter, which is usually at the level of the apex or one space lower and measuring to the inner borders of the ribs.

The cardiac measurements were also taken at their widest points. Usually it was the fifth space on the left side, the fourth space on the right. We used the metric scale. (See Fig. 10.)

A word ought to be said concerning the taking of the plate. The patient should be in the upright position and breathing should be shallow. The operator should wait until the initial excitement of the patient passes off. If this precaution is not heeded the cardiac excursions between systole and diastole will be great enough to cause an appreciable difference in the measurement of the heart therefore in the cardiothoracic ratio and in the interpretation of the same.

The patient should stop breathing in midinspiration, for the exposure. If the plate is taken at the height of inspiration the heart will be lengthened and narrowed; if at the height of expiration it will be widened and made more round. It will be seen that between these two extremes quite a margin exists.

The thoracic diameter will also vary with the phase of respiration. However, if the above precautions will be observed the margin of error or of variability will be reduced to a minimum.

We found as the result of these calculations that the normal heart is usually less than half the greatest diameter of the thorax:

$$\frac{\text{Heart (transverse diameter)}}{\text{Thorax (transverse diameter)}} \ 39 \text{ to } 50 \text{ per cent.}$$

The average was about 45 per cent.

Because of the possible variations previously described, a margin of safety of 2 per cent. above the upper limit was allowed.

We may summarize as follows: Anything over 50 per cent. was regarded as suspicious. If other evidence pointed against the existence of hypertrophy, values up to 52 per cent. were regarded as normal. However, 53 per cent. and over were considered definitely pathological.

In many of the long narrow-chested individuals with small, "drop" hearts the occurrence of valvular disease and coincident cardiac enlargement will often yield neither an evident increase in the actual nor the relative cardiac size above the standard diameters of normal hearts.

In fact, figures as low as 46 to 47 per cent. (cardiothoracic ratio) are quite common in spite of cardiac hypertrophy. This means that a heart may be absolutely small though hypertrophied. Although apparently paradoxical this fact rests on sufficient proof.

Hence in cardiac hypertrophy the relative size or the cardiothoracic ratio may or may not be increased. If it is, it indicates cardiac enlargement. If the ratio is not increased, cardiac hypertrophy cannot be ruled out. In such a case the diagnosis will have to rest on the study of the intrinsic diameters of the heart and the character of the murmur and the history.

It is evident, therefore, that the size of a hypertrophied heart will depend to a large extent on the type of heart and its size before the onset of the cardiac affection. Thus a small heart (Tropfen Herz) may, in spite of a severe aortic regurgation—which is so apt to produce cardiac enlargement—still remain smaller than the standard diameter for the average normal. The cardiothoracic ratio is very apt to be altered, however.

Incidentally I should like to call attention to the fact that the cardiothoracic ratio is of value in the recognition of a tubercular predisposition. This is based on the view of Brehmer,[3] who said that a small heart with too large lungs is an important element in the predisposition to phthisis.

Hence, when the cardiothoracic ratio is definitely under 45 per cent. it points in favor of tuberculosis in the presence of suspicious lung signs. The lower the percentage the greater the presumption.

SUMMARY. 1. The cardiothoracic ratio is based on the anatomical relationship that exists between the heart and its containing frame, the chest.

2. The method of obtaining this information is within the reach of the majority of practising physicians. In other words, wherever a roentgen-ray laboratory exists the facilities are sufficient for this work.

3. The method has been tried out on a sufficiently larger number of cases (some 500 or more) to warrant its practicability and usefulness in the estimation of cardiac size, particularly in cases of moderate or early enlargement.

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