ACUTE DILATATION OF THE HEART IN RHEUMATIC FEVER

BY

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Received 5th April—Read 28th June, 1898

It is not, I think, sufficiently recognised that acute dilatation of the heart is a frequent, almost a constant, occurrence in a rheumatic attack. Attention has been too exclusively directed to the auscultatory phenomena as indicative of the presence of pericarditis or endocarditis, and it seems to have escaped notice that whether either or both of these inflammatory conditions be present, or whether both be absent, there is almost always in rheumatic fever more or less dilatation of the heart. As a part of the chronic cardiac disease caused by rheumatism dilatation is generally acknowledged to be of great importance, but it is too much regarded as merely one of the results of a valve-lesion or possibly of an adherent pericardium.

In studying the curative influence of the ice-bag in pericarditis I found it necessary to determine carefully day by day the limits of the præcordial dulness, and I was struck with the evidence of a rapid and often permanent dilatation of the heart accompanying the signs of pericarditis. In a paper on the "Treatment of Peri-

carditis," published in the 'Lancet,' July 22nd, 1893, I attributed this early dilatation to the weakening effect of the pericardial inflammation on the cardiac muscular wall.

But observation of the fact that cardiac dilatation is much less marked in pericarditis of renal origin, or of tubercular or suppurative nature, suggested a doubt whether this explanation of the dilatation accompanying rheumatic pericarditis was altogether adequate, and the subsequent discovery of cases of rheumatism in which marked dilatation of the heart was present without any evidence of either pericarditis or endocarditis proved that there must be some other influence at work.

In December, 1894, I had under my care in St. Mary's Hospital a young man of seventeen suffering from his first attack of rheumatism, in whom the præcordial dulness was greatly enlarged without any definite murmur and without any friction or other indication of pericarditis. I am aware that an audible rub is not invariably present in pericarditis, but the cases in which I have observed this have been of suppurative nature, and I doubt whether this complete absence of rub throughout the case ever occurs in rheumatic pericarditis. That the increase of the præcordial dulness was not due to effusion into the pericardium seemed to be proved by the palpable cardiac impulse, the distinctly audible heart-sounds, and the absence of dyspnœa and distress. Under treatment this increase in the dulness gradually became less, and returned to the normal. In 1896 I had three similar cases; in these I took tracings of the enlarged area of dulness, and again found that it diminished under treatment and gradually returned to the normal. One of these patients had been in the hospital under my care four months before (1895) with a previous attack of rheumatism, attended with great increase of the præcordial dulness without either rub or murmur, and on this occasion also the excess in the dulness had disappeared on his recovery.

Before showing these tracings as evidence of alteration in the outline of the heart one must decide whether per-

cussion is trustworthy as a means of determining with accuracy the size of the heart. If an opinion on this matter had to be formed from the statements in text-books, this would seem extremely doubtful; but the valuable papers of Dr. Ewart in the 'Lancet' of August 29th, 1891, and the 'British Medical Journal' of March 21st, 1896, show that the true outline of the heart can be much more precisely ascertained than most physicians have supposed, while the paper read by Dr. Herringham before the British Medical Association at Carlisle in July, 1896, proves that the results thus obtained are almost exactly verified by post-mortem examination.

Confusion is introduced into this question by the terms "superficial" and "deep." What is called "superficial cardiac dulness" is of no *cardiac* value whatever. It is of importance as indicating the condition of the left lung, whether on the one hand it is emphysematous or on the other shrunken and fibrosed. But it would be much better to speak of the "uncovered cardiac area." The term "cardiac dulness" would then be understood to indicate the real size of the heart, a fact of the greatest importance.

In this way we should get rid also of the term "deep," which is apt to suggest that the percussion required to determine the true cardiac outline must be of a forcible kind. This is the very reverse of the truth, for such percussion brings out the pulmonary and gastric resonances and defeats its own object. Light percussion is absolutely necessary, especially over the sternum, which very readily conducts resonance from the lungs. If this be constantly kept in mind, there is usually little difficulty in defining with very considerable accuracy the outline of the heart on both left and right sides, for the cardiac margin on both sides is comparatively thick, and the alteration of the note on light percussion at the margin is generally quite definite.

It is well to begin in the region of the apex, and to remember that the left limit of the cardiac dulness extends

beyond, in dilatation often much beyond, the position of the impulse. The precise position of this limit should be found in the fourth and in the fifth spaces, sometimes in the sixth also.

Next, the extension towards the right of the right auricle should be determined by very careful light percussion to the right of the sternum in the fourth space, just below the line joining nipple to nipple. In the fifth space it is somewhat more difficult, because the partial hepatic dulness is here present also, but it can usually be defined satisfactorily. In health there is always about one fingerbreadth of dulness to the right of the sternum in the fourth space, in accordance with the anatomical facts.¹ When the auricle is dilated, as in mitral stenosis, the limit may be two or even three fingerbreadths to the right of the sternum. This is a most valuable indication of the condition of the right auricle, and one too much neglected; it is often of great assistance in determining whether removal of blood, by venesection or by leeches, is desirable in mitral disease or in pneumonia.

I measure by fingerbreadths rather than by inches or centimetres, for the use of these measures would imply that the limit can be determined with mathematical precision, which is not the case; no more than a close approximation can be claimed. The measure by fingerbreadths is made during the routine examination by percussion, it is made without any marking of the patient's skin, and without exciting his attention; it is a measure which the physician always carries with him, and it is invariable for the same observer.

Having determined the extreme cardiac limit both to left and to right, the position of the lateral margins of the heart may easily be defined if it be remembered that they both slope upwards and inwards, and the percussed finger be held in a position parallel to this slope in each case. The right margin above the nipple-level rapidly

¹ See also the "radiographs" of the cardiac outline by Dr. Williams of Boston, 'Brit. Med. Journ.,' 1898, vol. i, 1006.

approaches the sternum. The left margin normally rises to the inner side of the nipple, but in a moderately dilated heart the limit of dulness will be found to pass through the nipple, and where the dilatation is great it may cross the vertical nipple-line at one, two, in extreme cases even three fingerbreadths above the nipple. A little practice gives facility in determining the opposite borders of the heart, so that even without any marking of the skin one seems almost to *see* the outline of the heart. But a permanent record can easily be obtained by marking the outline with a blue pencil and taking a copy of this on tracing-paper, care being taken to indicate in all cases the median line, the infra-costal angle, and the position of the nipple, as points of reference. The extension of the cardiac dulness to left and to right of the median line may then be measured in inches or centimetres.

The upper and lower limits of the cardiac outline are less easy to determine satisfactorily. Dr. Ewart's observations about them, in the papers already referred to, will repay perusal. I have usually refrained from attempting to include the lower limit in the tracings, and I lay no stress on the accuracy or the precise meaning of the upper limit of the dulness, for the sternum and the great vessels here cause difficulty.

It may be well to add that the precise determination of the limit of the heart to right or to left may be impossible when there is fluid in the pleura, and it may be difficult when the breasts are large, when there is consolidation of parts of the lungs adjacent to the heart, or when emphysema exists. Fortunately, emphysema is rarely present in the subjects of acute rheumatism, who are usually in the earlier half of life. But if emphysema, pleurisy, and consolidation of lung are absent the lateral outline of the heart obtained by careful light percussion is almost absolutely correct, as I have found by post-mortem investigation. If the outline determined by percussion be marked on the cadaver, and long needles be passed through various points in the bounding line on either

side, it is found that they correspond with surprising accuracy to the margin of the heart.

It seems to me very important that a determination of its exact size by careful light percussion should invariably form a part of the routine examination of the heart. For it is dilatation that is the enemy, and an exact knowledge of the size and strength of the heart is far more important than the most elaborate and minute study of murmurs. It is very necessary in typhoid fever, where the cardiac condition is of great importance; it is indispensable in influenza and in diphtheritic paralysis, in both of which sudden death may occur from syncope; it may save life by indicating the need for bloodletting when the right heart is becoming paralysed from over-distension in pneumonia, capillary bronchitis, or mitral disease. And surely in rheumatic fever, a disease known specially to injure the heart, it ought never to be neglected. Yet are we not all too apt to use our stethoscope and neglect percussion, forgetting Sir George Humphry's rule, "*Eyes first, fingers next, ears last*"? We are prone to be satisfied if no murmur is audible. Yet in the absence of murmur there may be great dilatation; in proof of this I submit the following tracings.

The first three sets of tracings are taken from the cases of three young men suffering from their first or second attack of rheumatism, in none of whom any rub or definite murmur was audible, but in all of whom the cardiac dulness was for a time considerably enlarged. In all three the cardiac dulness extended one and a half or two fingerbreadths to the left of the nipple line, and one and a half or two fingerbreadths to the right of the sternum, on their admission into hospital; in all three it shrank to the normal dimensions as the rheumatic symptoms disappeared under treatment. I now show three tracings in each case, the first taken on commencing treatment, the third on recovery, and the second at an intermediate stage. Other tracings were taken, and these manifest a gradual reduction, but for the sake of

clearness I show only three in each case. *Every tracing was taken without reference to previous diagrams of the same case*, so as to eliminate the element of bias as much as possible. They have been carefully reduced in size on the same scale by Dr. Poynton, in order to make it possible to throw them on a screen by a lantern. The first tracing in each case has been coloured black, the second red, the third blue, so that the reduction in size is at once obvious.

Of course in all diagrams of this kind allowance must be made for some unavoidable exaggeration due to the fact that the tracing is taken on the curved surface of the thorax and is then flattened out on a plane surface. So that the transverse diameter of the diagram is necessarily somewhat longer than the actual transverse diameter of the heart.

CASE 1.—G. G—, aged 34, admitted into St. Mary's Hospital March 19th, 1896, suffering from slight arthritis of the left wrist and both shoulders. Temperature, on admission $100\cdot5^{\circ}$; on the next day 99° ; on the third day 98° . Treatment, sodium salicylate 20 grains every three hours.

G. G—, aged 34. Rheumatic fever.

The first tracing shows the area of cardiac dulness on his admission.

The second tracing was taken twelve days after the first; the left border of the dulness has now retreated to the nipple-line.

The third tracing was taken sixteen days after the second, a month after the first; the left border is now well within the nipple-line. The right border shows also a return to the normal.

CASE 2.—Alfred H—, aged 16 years, admitted February 3rd, 1896. Slight affection of ankles, knees, and hip-joints. Temperature normal. After four 20-grain doses of salicylate, the treatment was by 40-grain doses of sodium carbonate every two hours; this was continued for a fortnight.

Alfred H—, aged 16. Rheumatic fever.

The first tracing shows the cardiac area on admission.

The second tracing was taken ten days after the first; it shows a diminution of two fingerbreadths in the left margin of the heart, the border now passing through the nipple.

The third tracing was taken seven days after the second; the heart has now nearly returned to its normal dimensions.

CASE 3.—Albert R—, aged 21 years, admitted March 6th, 1896. Slight arthritis of left knee, ankle, and shoulder. Temperature 103° on first evening, 99° next morning; some irregular pyrexia continued for fifteen days. After about six 20-grain doses of salicylate the treatment was by 40-grain doses of sodium carbonate hourly during the daytime.

The first tracing shows the cardiac area on admission.
The second tracing was taken four days after the first,

Albert R—, aged 21. Rheumatic fever.

and shows a marked shifting inwards of the left edge of dulness; but the area of dulness towards the right has somewhat increased. This was apparently due to an attack of pleurisy, pleural friction being plainly heard. Leeches were applied, and in the third tracing, taken on the following day, the right border of the heart as well as the left has returned to the normal position. I have already said that I lay no stress on the upper limit of these tracings, and I think it possible that the upper limit of this third tracing may be erroneous.

This patient had been under my care four months before, during a previous attack of rheumatism. On this occasion also he had acute dilatation without pericarditis or endocarditis. At first the cardiac dulness extended to one and a half fingerbreadths, or one inch, outside the nipple line. On his recovery it had become normal, and was found half an inch internal to the nipple.

The fourth set of tracings shows the supervention of acute dilatation in a heart already damaged by previous rheumatism.

CASE 4.—Harriet W—, aged 14 years, admitted into St. Mary's Hospital, March 10th, 1896, suffering from her third attack of rheumatism. The first attack occurred four years before, the second three years before; in both chorea accompanied the rheumatic symptoms. This third

attack commenced three weeks ago ; in it there had been dyspnœa, pain referred to the heart, also pain in the elbows and feet.

On admission temperature 99° , pulse 122, respirations 24. Fluid in both knees, ankles also swollen. Faint systolic murmur at apex, and over pulmonary artery ; suspicion of pre-systolic murmur at apex. Patient emotional, but not distinctly choreic. Urine faintly acid. No salicylate was employed in this case, the treatment being by 30-grain doses of sodium carbonate given hourly during the daytime.

The first tracing was taken the day after her admission ; it shows the left border of the heart extending three fingerbreadths outside the nipple-line, and two and a half above the nipple, while the right border is fairly normal.

Harriet W—, aged 14. Rheumatic fever.

No. 1.

March 11, 1896.

No. 2.

March 12, 1896.

No. 3.

March 16, 1896.

The second tracing, taken the next day, is identical with the first on the right side, but shows a marked reduction on the left side. The pre-systolic murmur was now distinct.

The third tracing, taken four days after the second, is similar to the others on the right side, but shows a further large reduction on the left side. The urine was now strongly alkaline. A fourth tracing, taken nine days after the third, is practically identical with it ; she was now taking the alkaline medicine every three hours, and the urine had become again faintly acid.

The third and fourth tracings represent doubtless the

permanent increase in size of her heart caused by the previous disease, which had left behind it distinct slight stenosis and regurgitation at the mitral. The first tracing shows the additional temporary acute dilatation of the present attack of rheumatism.

The fifth set of tracings shows the effect of the combination of acute dilatation with acute pericarditis.

CASE 5.—Daisy D—, aged 9 years, was admitted into St. Mary's Hospital February 7th, 1896, suffering from chorea, pericarditis, and slight arthritis of hands and feet. This was her second rheumatic attack. Twelve months previously she had been an in-patient for rheumatism, followed by chorea. On her admission a loud pericardial rub and a systolic apex murmur were heard by the house physician, who at once applied an ice-bag over the heart, and ordered 10-grain doses of sodium salicylate with fifteen grains of sodium carbonate every four hours. I saw her the next day and took the first diagram, which

Daisy D—, aged 9. Rheumatic fever.

No. 1.

Feb. 8, 1896.

No. 2.

Feb. 21, 1896.

shows an enormous increase of the transverse cardiac dullness, the left border extending to more than three finger-breadths outside the nipple-line, and about two above the nipple; the right border also is much too far to the right. It is not unlikely that part of this increased dullness was due to the presence of some pericardial effusion. The medicine was repeated every two hours. Next day the frequency was altered to every three hours, and on the following day (10th) to every four hours. On the

11th the urine was found to be still acid, and again on the 13th it continued acid, so that the medicine was again given every three hours. On the 14th the pericardial rub had disappeared and there was no longer any chorea, but a loud systolic murmur was heard at the apex, and pleuritic friction was detected in the right axilla. The medicine was therefore given every two hours, and a second ice-bag was placed over the anterior base of the right lung. Two days later, the urine being still acid, the dose of sodium carbonate was doubled, and as the pleurisy had disappeared the second ice-bag was removed from the right lung. On the 19th the urine had at last become alkaline. On the 22nd the ice-bag over the heart was removed, having been applied continually for fifteen days.

The second tracing was taken on the 21st, thirteen days after the first. It shows an extraordinary diminution in the cardiac dulness, and the improvement in the general condition corresponded to the improvement in the tracing. The pericarditis had subsided, but evidence of endocarditis remained. The second tracing probably indicates the permanent cardiac enlargement produced by the first rheumatic attack. The great increase in the præcordial dulness shown in the first tracing may have been due in part to effusion into the pericardium, but was probably mainly due to acute dilatation.

It is worth while to point out incidentally that in this case, in spite of the pericarditis and the dilatation, the free and prolonged administration of salicylate of sodium produced no "depressing" effect.

I do not claim that these tracings are absolutely accurate; no doubt they are open to criticism in points of detail, and I would again refer to Dr. Ewart's valuable papers. But they were taken with care, in all cases by myself, usually verified by another observer, and *always without reference to the preceding tracing.*

I think they are sufficient to prove that an acute dilata-

tion of the heart does occur in acute rheumatism ; that it may be marked even when there is only slight pyrexia and but little arthritis, with no modification of the cardiac auscultatory phenomena beyond slight alteration in character of the heart-sounds, and that it may complicate the evidence of former heart-disease or of fresh endocarditis or pericarditis.

I made a preliminary statement of these facts in a letter to the 'Lancet' of July 25th, 1896, and also mentioned therein that a similar dilatation occurs in most cases of chorea, even when no murmur exists,—a new evidence of the essentially rheumatic nature of chorea. Since that date I have made other observations on rheumatism in adults at St. Mary's Hospital, and have obtained results similar to those already exhibited. I have made observations also on the occurrence of acute dilatation of the heart in the rheumatism and chorea of childhood at the Hospital for Sick Children, mainly in conjunction with my friend Dr. Poynton, formerly house-physician at that hospital, and now medical registrar and pathologist at St. Mary's Hospital. We have embodied our results in a joint paper, illustrated with tracings taken very carefully by Dr. Poynton and confirmed in my own cases by myself. It seemed desirable that the results should be compared with observations on the hearts of children free from rheumatism and cardiac disease. Dr. Poynton therefore took tracings of the cardiac outline in forty-five children in the surgical wards at Great Ormond Street during his subsequent tenure of office as house-surgeon. He has also carefully examined the hearts of thirty-five healthy boys, aged twelve and thirteen, at Marlborough School, with the kind permission of Dr. Penny. Finally, he has analysed the post-mortem records of 150 cases of fatal rheumatic heart-disease in children under twelve.

I venture to think that the pathological and clinical results of his researches and observations afford additional and decisive proof of the frequency and the serious importance of rheumatic dilatation of the heart.

The occurrence of acute dilatation of the heart in rheumatism has not been entirely overlooked. Thus Dr. Sansom writes ('International Clinics,' 1894, p. 7): "I am convinced, however, that the rapid increases of dulness over the heart in rheumatism are not all due to pericardial inflammation and the effusion of fluid; the whole heart may become swollen and dilated—swollen with the products of inflammatory exudation, dilated because of the enfeeblement of the muscle of its right and left chambers. In some cases this condition of swollen heart disappears without any of the friction signs of pericarditis being manifested; in fact, the heart and its serous membranes may pass through changes like those occurring in a joint inflamed through rheumatism. These variations in the bulk of the heart may be observed in some cases to be considerable from day to day, and there may be repeated enlargement at intervals of a few days, just as there may be repeated swelling in the joints."

Dr. Samuel West reported to the Pathological Society in 1882 the case of an anæmic boy of sixteen, who died two months after an attack of rheumatic fever, and was found to have dilatation of all the cardiac cavities, especially of the ventricles. Evidence of endocarditis also existed. The myocardium was affected with fatty degeneration.

At a still earlier date, in 1879, Dr. Goodhart exhibited to the same society the heart of a boy of seventeen who died within a month after an attack which was certainly rheumatic, in whom the heart had rapidly enlarged. The left ventricle was "rather widely dilated." Weight of heart, 19 ounces; pericardium entirely adherent by soft lymph; recent valvulitis; muscle slightly fatty.

In the same year, in the 'Guy's Hospital Reports,' Dr. Goodhart described cases of acute dilatation of the heart following scarlet fever, and another was published in 1880 by Dr. Barlow in the 'Medical Times and Gazette' (1880, vol. i, p. 426). In none of these, however, was

there any evidence of rheumatism. But in a note appended to his paper Dr. Goodhart adds: "Sir William Gull was in the habit of giving special prominence to the fact that acute pericarditis in rheumatic fever is liable to give rise to rapid dilatation of the heart, which is often mistaken for pericardial effusion. Sir W. Gull also taught that dilatation is succeeded, not preceded, by hypertrophy in some of these cases." "I am very glad," Dr. Goodhart continues, "to be able to mention this, not only because it is a weighty confirmation of some of the remarks made in this paper from another point of view, but also because it has hitherto been unrecorded."

It may perhaps be doubted whether the value of these observations of Sir William Gull's has been adequately appreciated. At any rate, I think that the frequency and extent of acute cardiac dilatation in rheumatic fever is insufficiently recognised by most physicians. Yet it is a condition of great gravity, and its presence or absence ought to be carefully ascertained in every case of the disease. When uncomplicated with pericarditis or endocarditis, if the patient be kept absolutely at rest it may prove transitory, and the heart may entirely recover itself, especially under appropriate medicinal treatment. But when it complicates pericarditis, it adds enormously to the danger of the latter condition. In the more severe cases of rheumatic pericarditis, the accompanying acute dilatation is probably largely responsible for the dangerous symptoms of cardiac failure, for the dyspnoea, the tendency to cyanosis, the feeble pulse, and the delirium.

When the pericarditis is over, and has ended in pericardial adhesion, the heart becomes fixed in its dilated state, and it is never again able to return to the normal. Some amount of hypertrophy may follow, enough to maintain compensation, in the absence of much exertion, for a limited time; but such hearts are permanently crippled and soon break down. Cases of this kind are only too frequent in children and young adults.

Acute dilatation is probably a much more important

factor than endocarditis in the production of many cases of chronic heart disease. In a girl recently under my care at Great Ormond Street, enormous dilatation of the heart was produced in six months after the first illness. She died from pericarditis; it was found after death that there was no fluid in the pericardial sac, but recent adhesions everywhere. There was some mitral endocarditis, but it was very slight, and entirely inadequate to account for the remarkable dilatation of the ventricles. If this patient had survived, and come under observation at a later period with an enlarged heart and a systolic apex murmur, it is almost certain that the cardiac dilatation would have been considered the consequence of her mitral insufficiency. It is probable that very many cases of chronic heart-disease now attributed to "mitral regurgitation" or to "adherent pericardium" are essentially the permanent results of an acute dilatation, the pericardial adhesions and the valvular damage taking but a small share in the morbid process.

It is unlikely that many cases of the acute dilatation above described are due to definite myocarditis. No doubt this is a possible cause, and it was present in a case recently reported (with autopsy) to the Clinical Society by Dr. Herringham. But the transitory nature of the affection under appropriate treatment, as shown by the tracings now exhibited, seems to prove that in these there cannot have been any actual inflammation, though there may have been an acute congestion, of the muscle. It is possible that skilled microscopical examination may reveal some change in the nutritive condition of the cardiac muscular fibre generally, but whether this be so or not, it seems likely that the effect is a toxic one.

Sixteen years ago ('Journal of Physiology,' vol. iii) Dr. Gaskell proved that a dilute solution of sodium hydrate caused gradual progressive contraction of the frog's ventricle until it remained persistently fully contracted and failed to relax at all; it caused also a similar contraction of the arterioles. On the other hand, a dilute

solution of lactic acid caused relaxation and "extreme dilatation" of the ventricle, and finally diastolic standstill; it caused relaxation also of the vessels. He investigated the action of various drugs, and found that some of them acted like soda, others like lactic acid.

These experiments seem to suggest that the production of the remarkable cardiac dilatation which occurs in rheumatism may be due to the presence in the circulating blood of a poison acting on the heart like lactic acid. May not this be the toxine resulting from the development of a micro-organism? It can hardly be doubted that this is the true explanation of the acute and sometimes fatal dilatation of the heart which occurs in influenza. The more carefully rheumatism in childhood is studied, the more reason will be found for the belief that it cannot be due to any mere perversion of metabolism, but must be due to some microbic process.

The suggestive observation of Dr. Gaskell as to the influence of soda in causing cardiac contraction seemed to me to afford reason for hope that this alkali might be found of service in diminishing the dilating effect of the rheumatic toxine. I have therefore treated some cases with large and frequent doses of sodium carbonate, and I have thought that a more rapid shrinking of the enlarged area of cardiac dulness occurred in these cases than in those treated with salicylates alone. The plan seems to me worthy of further trial. At all events I found that the drug was well tolerated in spite of its unpleasant taste, that it never caused vomiting or lessened appetite in rheumatic subjects, and that large doses were required to render the urine alkaline. I selected the carbonate in order to secure as large an amount as possible of the sodium element, forgetting for the time that the water of crystallisation contained in it makes it really less powerful as an antacid than the bicarbonate. I am now making further observations upon the action of the bicarbonate.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 189.