THE LUMLEIAN LECTURES ON HEART INFLAMMATION IN CHILDREN.

Delivered before the Royal College of Physicians.

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II.

MR. PRESIDENT, FELLOWS, AND GENTLEMEN,—In my first lecture I attempted some classification of rheumatic heart inflammation in childhood, dwelling especially on that acute form of carditis which, while it is almost peculiar to that time of life, is at once so insidious and so dangerous as to demand special attention. Reference was made to the great diversity in its earlier symptoms, the trivial character of its arthritis, and the absence sometimes of any distinct evidence of rheumatic origin. Further, the prominence of nervous phenomena was alluded to, and the suddenness of cardiac failure was contrasted with the very slight anatomical changes found in some instances after death.

I would now shortly consider some points of physical diagnosis, already incidentally mentioned, in reference to pericarditis; and proceed thence to the more controverted question of the earliest recognisable signs of endocarditis in children.

In most instances of pericarditis, if not in all, there is slight displacement outwards of the heart's apex. The sign, however, is of some delicacy even when the heart is of normal size, and the patient seen early, and it needs for its appreciation accurate notions of the natural position of the apex at various ages.

Much labour has been expended on the determination of this point. In his observations upon the locality of the apex beat in 300 healthy children between a month and 14 years of age, Stärcck¹ found that in many cases the position was difficult to determine. Up to the fourth year it is, in his estimate, just outside the nipple line; from 5 to 6 it is *in* the nipple line; and after 6 inside that line. As to the cardiac area of superficial (not deep) dulness he found it diminish with the years both as to its upper and left boundary, falling by the width of a rib (that is, the fourth left rib) between the sixth and the twelfth year. Stärcck recognises three types of superficial cardiac dulness corresponding with 1 year old, 6 years, and 12 years. It is sufficient for our present purpose to notice the second and third types. The deep dulness at both 6 and 12 years is practically the same (dotted line). The superficial dulness is, as here represented, lower at 12 years by the width of a rib (darker area), and having its left border on the parasternal line.

Fig. 1.

My friend Dr. Garrod has been good enough to investigate the subject of the superficial cardiac area dulness, and the lifesize tracings here produced are the result. They accord generally with Stärcck's conclusions. "The observed peculiarities are explained," Dr. Garrod believes, "by a more

¹ *Archiv für Kinderkunde*, 1888.

horizontal or recumbent position of the heart in children than in adults, the heart gradually becoming more erect as the child grows."

Fig. 2.—Influence of position on cardiac dulness; — outlines of dulness in erect position; ---- outlines of dulness when lying on the left side; outline of dulness when lying on the right side. E. L., 11 years. Physiological albuminuria; heart natural.

Fig. 3.—W. P., 5 yrs. 10 mos. Cough; no physical signs; heart natural.

Another point included in Dr. Garrod's tracings concerns the amount of displacement of the child's heart in different positions. It was observed of a patient of my own (L. H., aged 8) that the displacement to the left, following the child being laid upon that side, was maintained for some minutes after the dorsal position had been resumed. Drs. Ringer and Phear record a similar observation in a recently published paper.² They noted further a similar delay in movement lasting a minute or two on the first disturbance of the patient from the back to the left side.

Dr. Walsham, at the same hospital, has made observations not only as to the movements of the heart right and left, but also in the inverted position, upside down. In the case of four boys, aged 5, 7, 8, and 9, he found that no change whatever occurred in the cardiac area when they were placed head downwards. One of these boys, aged 8, as I myself witnessed

² *Lancet*, February 10th, 1891.

at Dr. Walsham's invitation, remained in that position five minutes. At the end of that time the cardiac area, that had been carefully marked out before, had in no way altered.

Fig. 4.—E. M., 7 years. Debility; no evidence of heart lesion.

Fig. 5.—E. E., 10 years. Headache, vomiting; no sign of cardiac lesion.

In a proportion of cases of acute pericarditis the variations in the area of cardiac dulness due to fluid are but slight. There is a little widening and a little heightening of this area, neither of these changes being of long duration. The period occupied by the three stages of effusion—its advance, acme, and decline—is included within a fortnight. The time of decline is apt to be longest and the acme or period of greatest effusion the shortest. Sibson (p. 11, vol. iv), who made minute study of this point, found that in as many as 39 of 44 cases, the period of acme (or greatest area) lasted but a day. In chronic pericarditis these periods may be long exceeded; but even in the most prolonged the change in area of cardiac dulness will be found to vary from day to day, now increasing, now decreasing, now increasing again.

And where effusion is considerable there is, besides that which percussion makes out, what the eye best measures, namely, widening of the left intercostal spaces from the second rib downwards, with accompanying elevation of these spaces. An appearance of raised flatness is thus produced,

contrasting strongly with the natural contour of ribs and spaces of the right chest.

Fig. 6.—E. C., 11 yrs. 8 mos. Eczema; cardiac lesion.

It may seem at first thought a tedious task to map out from day to day by percussion the several fluctuations of pericardial effusion. But it is not so, for two reasons: one, that the patient is a child, small, thin, and easily handled; the other that, with certain precautions to be presently mentioned, the upper boundary of cardiac dulness taken

L. H., aged 8. Changes with position.

Fig. 7.—On back.

Fig. 8.—On left side.

alone indicates sufficiently by its rise and fall the daily course of effusion.

Fig. 9.—Immediately after being turned on back from left side.

It need not be said that the limits of the heart's superficial dullness—upper, right, and left—are not equally defined. The right border is the most abrupt in case of slight enlargement, owing to the interference of the sternum. The left and the upper borders of superficial dullness are bounded by narrow regions of lessened dullness due to the interposition of thin edges of lung, overlying the left ventricle in the one case and the upper portion of the heart and great vessels in the other. In extreme heart enlargement the left border is carried so far out as to be lost, so to speak, in the lateral region which is itself dull owing to the contiguous left ventricle. The upper margin of cardiac dullness is best estimated by approaching it gradually in percussion from the clavicle downwards; the right and the left borders by first seeking the apex place or "centre" of dullness, and thence percussing outwards in both directions.

With the percussion signs and signs that the eye notes there concur others not less important, which the palms and fingers perceive. But these latter will be found to vary with the amount of effusion and consequent displacement of the heart.

The manner of this displacement has been much discussed. Sibson, in his later work, lays much stress on the fact that the heart in effusion is displaced "upwards into the higher and narrower portion of the chest." He thus explains the elevation of apex—which he was the first to point out—and the close apposition of the heart to the anterior chest wall as well as the vibrations and grating friction. "When the effusion lessens," he adds, "the thrill disappears, the creak vanishes, friction sound softens." In his first article on pericarditis, however, in 1849 (vol. iii, p. 20), Sibson clearly described the effect of distension of the sac as causing the fluid to rise so as to cover the heart in front, separating successively the ventricles, right auricle, and vessels from the pericardium. This earlier view of Sibson is that which is now generally accepted. Thus Dr. Bristowe (p. 496) asserts that "the heart in effusion is necessarily carried backwards, and consequently away from the anterior thoracic wall"; while Balfour and others believe that the rise of the apex is only apparent. "What we feel," says Balfour (p. 325) "is not the apex, but the part of the ventricle lying above it, and appearing to rise as more and more of the heart is separated from the anterior wall, with which the base of the heart always remains in contact."

Latham and Walshe are in substantial agreement with Sibson's later views, believing that with extensive effusion the heart is pushed upwards to the fourth, third, and second interspaces, the rub, thrill, and vibration being all correspondingly raised.

There is yet another view of the conduct of the heart in effusion, adopted by those who question the fact of the organ being pushed either upwards or backwards. It is that which maintains that the heart in such cases remains where it was. Dr. Rotch seeks to establish this conclusion partly by experiment, and more cogently by the argument in which Balfour

preceded him—namely, that the visible impulse "need not be that of the apex, but of the tumultuous right ventricle above it."³

Professor Henoch, in his well-known lectures on children, does not allude to this particular subject, deeming it, perhaps, unsuited to his dogmatic method.

In my own belief, the opinions just quoted, though apparently conflicting, are in fact reconcilable. They all express the truth in various circumstances. The heart may be moved either forwards, upwards, or backwards in effusion; or it may remain where it was; and of the factors that determine its conduct, pericardial adhesion—here or there, temporary or permanent—is the chief.

I have repeatedly in fatal cases of pericardial effusion inserted needles, just before the *post-mortem* examination, into the proper apex place, and above the fifth right costal cartilage, close to the sternum, without being able to detect upon opening the chest any dislocation of the heart. The validity of such experiments may be questioned; but there are clinical facts to show that the early pushing forward of the heart, with consequent pericardial friction, increasing in area and intensity, although it may be the rule, is not without exception. The fluid may cover the heart from the first, and no friction be audible until it has been absorbed.

This was so in the case of a child, aged 9 (E. R.), who during the time of her acute symptoms showed the physical signs of pericardial effusion merely. The first sound of friction concurred with an abatement of the worst symptoms, and lasted on almost to convalescence.

The diagrams before you and on the table are illustrations of the several ways in which the normal area of cardiac dullness is deformed and the apex displaced in effusion. It is by design that I omit minutiae such as the exact changes of shape in the cardiac area from roughly triangular to roughly square. What I would impress is the sort of heart percussion required in the daily routine and practice no more and no less. In most cases the utmost degree of effusion is soon reached. It continues in that stage but for a short time, often less than a day, receding in many cases only to return, so that, without corresponding change in the general symptoms, the alternate ebb and flow of pericardial effusion may be watched over a considerable period; the upper margin of cardiac dullness higher or lower sufficing, as I have said, as the daily gauge. In the more lengthened attacks the pericardial sac is observed to widen disproportionately to its heightening, and it is in such cases, when newly observed, that much difficulty may arise in distinguishing between a dilated heart and a fluid-laden pericardium. It is, in fact, by the daily changes and not by the particular shape or extent of cardiac dullness at a given time, nor yet by any necessary distinction in the character of the heart's sounds in the two cases, that this differential diagnosis between effusion and dilatation is safely made.

It has been shown by Rotch and others that cardiac dullness transgressing the right margin of the sternum, and apparent in the fifth right interspace, makes fluid effusion probable, and paracentesis has been successfully made in that region, as, for example, by Dr. Dickinson in a case of purulent effusion;⁴ but the sign is not infallible even when this extension to the right is both rapid and recent.

Thus, in a case of carditis to be presently mentioned (Babbage), the area of cardiac dullness remaining unaltered, it was conjectured that the pericardium might be adherent. Presently pericardial rubbing became audible, and with this the right border of cardiac dullness extended well into the fifth interspace. The child quickly died. Yet was there no fluid whatever to account for the sudden widening of cardiac area. That change was due to rapid dilatation of the right side of the heart at the time of its sudden failure. The pericardial surfaces were adherent everywhere, with no interposing fluid whatever.

In this instance the fact that the area of cardiac dullness, though widened to the right, was not heightened, might suggest dilatation rather than effusion, but there are cases (like that of Thomas) where rapid pericardial adhesion seems to arrest the ascent of the upper margin of dullness.

In another case of markedly dilated heart of such width as

³ Keetly, *Cyclopædia*, p. 865.

⁴ *Clin. Soc. Trans.*, 1889, p. 50.

to be clearly perceptible in the fifth right space, extensive friction rub arose some days after admission; yet was there no accompanying increase of cardiac area. *Post mortem* a notable amount of fluid was found surrounding the heart; it lay round it, so to speak, in such manner as to make discovery during life impossible.

DILATATION SIMULATING EFFUSION.

D. B., aged 8. October 21st (on admission) slight increase of A. C. D. November 28th (two days before death) sudden extension of A. C. D. to right (dotted line).

Fig. 10.

Post-mortem, pericardium universally adherent. No fluid in sac. Visceral and parietal pericardium covered with lymph. Heart's weight $9\frac{1}{2}$ ounces.

Such is often the manner of cardiac empyema where pus surrounding the heart makes no perceptible change in its apparent size or shape. How rarely is this condition recognised until *post-mortem* examination discloses it!

Of the difficulty of founding a prognosis upon the observation of the fluctuations in the area of cardiac dulness, a good illustration is afforded by two children, one of whom (Bishop) died, and the other (Drinkwater) eventually recovered. In the fatal case, the worst symptoms concurred with increasing area of dulness. In the non-fatal, but very severe and prolonged case, the boy was at his worst, unconscious, and passing motions into the bed, with rapid decrease of the same area.

Other diagrams on the table illustrate the same point; as, for example, Thomas, the case related in the last lecture, where the area of dulness decreased for some time before death.

It is very rarely that the pericardial effusion of rheumatic children becomes so extreme as to need paracentesis. In the whole of the cases I am now reviewing, there was no such occurrence.

While the signs of rheumatic heart inflammation are in full progress as regards the pericardium, the endocardium, as we know, is suffering also. But just as pericarditis does not show at the first either by increased cardiac dulness or audible rubbing, so endocarditis, at its commencement, does not impair the efficiency of the valves so as to permit reflux. In other words, the obvious and familiar physical signs we are in the habit of associating with endocarditis refer less to it than to its consequences.

The early observers were not ignorant of the fact that mitral reflux might occur late. Latham especially, though regarding the advent of such murmur as of itself and without qualification indicating endocarditis in cases of rheumatism, was fully persuaded that the first onset of mitral valve inflammation might precede this particular sign. His were the days of enthusiasm for physical signs when a single one was held sufficient to indicate a particular disease state. Mitral murmur in the course of rheumatism was for Latham the signal for bringing into play the whole array of anti-phlogistic remedies, just as the subcrepitant *râle* was warrant enough for a like treatment in lung disease. But no one was

more alive than he to the significance of certain cardiac signs antecedent to murmur, and he often saw reasons for commencing his remedies before this sound appeared.⁵

"There may as yet," said he, in 1845, nearly fifty years ago, "be no endocardial murmur, but the systolic sound may be unnaturally prolonged and unnaturally rough. I hardly ever knew a case in which unnatural length and roughness of sound such as a practical ear could well discriminate has not become an unequivocal murmur in twenty-four hours. How often have I anticipated that the murmur would be formed to-morrow. It is almost as certain a sign as murmur itself. I no sooner perceive it than, without waiting for the murmur, I begin the treatment of endocarditis at once."

A period prior to any audible murmur may, in Latham's opinion (pp. 72, 73) cover all the essential morbid processes of the disease and even outlast the time when it can be successfully treated. And accordingly, with a precision and accuracy not exceeded in the present day, Latham goes on to describe three distinct relations of mitral murmur in respect of endocarditis. Thus:

1. In some murmur is coincident in time with the very commencement of the inflammation, the natural sound is changed into the genuine murmur without notice or preface.

2. In some (and most frequently) murmur does not arise until inflammation has somewhat advanced.

3. In some (and least frequently) murmur does not arise until inflammation is on the decline or has ceased.

Since the days of Latham and Watson the earliest physical indications of endocarditis have been much discussed, and the general conclusion seems to be that no single sign—perhaps no combination of signs—indicates with certainty the very beginning of endocarditis. And if this be said of a first attack, how much greater is the difficulty with subsequent attacks?

Time only serves for one or two quotations in testimony of this obscurity.

Walshe (2nd Edition, 615-617) would base the diagnosis of endocarditis on "febrile action, cardiac uneasiness, and endocardial murmur, occurring in a person presumably free from prior cardiac disease." At the same time, and along with this reservation, he alludes to the possible occurrence of dynamic murmurs, and admits that "endocarditis may exist without murmur."

Rosenstein believes that the diagnosis of endocarditis is purely arbitrary.

Bristowe (p. 528) frankly admits that the symptoms of endocarditis are not striking, and that it is by the development of the valve lesion that we mainly assume its presence and trace its progress.

Professor Potain,⁶ of La Charité Hospital, is more confident. Admitting the great diversity of opinion as to early endocardial signs, he considers all those hitherto put forth as absolutely unreliable. What is reliable, according to this authority, is the change which takes place in the quality (*timbre*) or tone of the normal sounds. This change, expressed in a word, is progressive obscurity of sound. Sometimes the first sound is obscured, sometimes the second, sometimes both. Its gradations may be thus expressed: "Slightly muffled," "muffled," "obscure," "very obscure," "inaudible." In a certain proportion he admits the changes were very slight, and considerable experience and a practised ear were needed for their detection, nor "was it possible to form an opinion, except by comparison of the day before." The professor goes on to describe the several morbid changes productive of these altered sounds, but, inasmuch as death very rarely occurs at this early stage, we may safely assume that the description is no more than conjecture.

My own experience, as I shall presently relate, does not coincide with that of M. Potain, except as regards the changeful character of the heart sounds and rhythm, and the value of successive observations from day to day. Inaudible first sound is familiar to all of us in connection with enteric fever, and was first pointed out by Stokes. It is met with, no doubt, in some cases of rheumatic endocarditis, but, according to my observation, occurs late rather than early.

In a recent instance, however, partial and very temporary effacement of the first sound preceded by some days the

⁵ Latham, p. 65, vol. ii, Sydenham Soc.

⁶ *La Semaine Médicale*, December 8th, 1881.

signs of pericarditis. The patient was a girl of 13, admitted with a third attack of acute rheumatism, with old mitral murmur. Fourteen days after admission, when the temperature had been normal for ten days, pulse was steady at 68, and the joints free of pain, the first sound became very indistinct. A week later temperature rose, without any return of joint pain, and four days later, when the first sound had regained all its distinctness, but there was increased fever. Some indistinct friction could be heard for two days, the patient making rapid recovery. In this case Professor Potain's sign was not distantly imitated, but an interval occurred between the obscuring of the first sound and the earliest obvious signs of carditis. It may be added that Dr. Barr, of Liverpool, considers "a dull first sound with delayed radial pulse amongst the earliest signs of endocarditis."

Midway between such frank expressions of imperfect knowledge as I have just quoted, and the more confident statements of the French professor, we may place the elaborate researches of Sibson in the same field. "His are tentative conclusions," as his pious editor, Dr. Ord, remarks (I, xii): "conclusions such as a man would arrange before him to aid his final judgments; a multitude of facts in which the reader finds himself lost, and is conscious that in many points he is not put in possession of clearly-stated inferences."

For such reasons it is difficult to estimate the precise value of Dr. Sibson's work, and not always easy even to follow him. Yet without doubt we owe much to this indefatigable worker—the vivid description of the aspect and demeanour of commencing endocarditis; the minute and accurate account of the several declensions from the normal sounds, from such as are to be regarded as "threatening" endocarditis to those which, in their further development, make the actual presence of that inflammation "probable," thus precisely defining the degrees and the limits of knowledge in this respect. And we may notice by the way, and with gratitude, Sibson's very careful and guarded—it may be too guarded—use of the word "presystolic," a term which I believe, except in one place, he hardly allows himself without qualification.

On the other hand, it may seem to some that he gives no very clear account of the physical signs of mitral narrowing; that he exaggerates (vol. iv, p. 242) the frequency of tricuspid murmur, mistaking it sometimes for the true presystolic, "grave and even vibrating," as he puts it, "beginning with an accent or shock," and that he is somewhat inconsistent in placing tricuspid murmur among the signs indicative of the very commencement of endocarditis, and also as coinciding "in almost all the cases with great general illness" (p. 244).

These may be shallow criticisms, and in any case we are to remember, as Dr. Ord remarks, that "the hand of the artist was stayed while he was still engaged in fashioning his work—that we have but the clay model for a work in marble."

On the threshold of this subject of the early signs of endocarditis so exhaustively discussed by Sibson, it is well to remember what he so well urged, namely, the fact that both from the structure and action of the mitral valve, as well as from the situation of the inflammatory product as seen after death, it is apparent that blood reflux is not an early but a late result of endocarditis (vol. iv, 234). "The border of contact in systolic closure is not a mere edge, but a surface or line of adaptation, made up of small bead-like cells that dovetail into each other along the margins of the flaps. Endocarditis affects not the very edge or rim of the flaps, but the line of contact just within the edge. When the mitral valve is inflamed it is this border of contact that shows a frill of small beadlike granulations, quite insufficient at first to prevent accurate and complete closure. Presently, however, each of these little prominences gets capped with fibrin, and it is by this addition, sooner or later, that adequate adaptation of the valve flaps becomes impossible, and reflux begins."

This early condition of endocarditis is seen best not in rheumatism but in chorea. For in rheumatism death seldom occurs without further morbid changes, which chorea, as such, does not share. In fatal cases of chorea, apart from rheumatism, we see endocarditis in its purity, so to speak, and it is well known that the bead-like granulations often found in

chorea, chiefly in connection with the mitral valve, have sometimes sufficed to produce mitral murmur and sometimes not.

But there yet remain some special circumstances of childhood which help to explain why in their case the earlier physical signs of endocarditis should so often escape notice. In the first place, the early joint pains seem so trivial that the child is seldom brought to hospital on account of them. In the second place, a large proportion of the patients have had previous attacks of rheumatism, which have so changed the heart as to confuse conclusions. It is rare to meet with a child at an early period of its first rheumatic attack. So it happens that the physical signs we get to associate with endocarditis belong properly not to its early and active stage, but to its results and consequences.

Having dealt thus far with the views of well-known authorities as to the earliest recognisable signs of endocarditis, and thus shown by their want of accord the difficulty of the subject, I shall venture to add some experience of my own as to the manner in which endocarditis comes about in the case of rheumatic children, and its several modes of development.

The earliest physical indications of endocarditis in children are only trustworthy when associated with rheumatism. Tumultuous, quickened, and uneven heart action sounds, that are changeful from day to day, especially the first; sounds reduplicated, especially the second at and above the apex (not at the base); a temporary tricuspid murmur; marked accent commencing the first sound, whether mitral or tricuspid; these are amongst the commonest of such signs.

But it is not in all cases that endocarditis signs are thus gradually developed. Sometimes mitral reflux arises very early, without previous notice or warning. Thus a child of 3 years and 8 months (R. E.) was admitted, October 4th, 1893, for some general œdema, but without albuminuria or cardiac murmur. There was no history of scarlatina, sore throat, or rheumatism. In two days the œdema disappeared, and the child seemed well. Ten days later, however (that is, on the 16th), some desquamation was found over the legs, and the child was thereupon removed, the heart sounds at the time being perfectly natural. On November 1st, that is, fourteen days after the examination of the heart, the child was again admitted with swollen and painful joints of five days' duration, the temperature being 102°. And now there was well-marked blowing murmur at the mitral which was conducted into the axilla, and accompanied at first by an occasional "squeak."

In this instance, it seems almost certain that a period of no more than five days at the utmost sufficed to develop a blowing and conducted mitral murmur having all the characters of regurgitation. In the more usual course, the progress is gradual, and it is nearly followed in the case of chorea. It is generally admitted nowadays that the heart signs of chorea, or at least the more pronounced of them, are due to endocarditis. Now these signs are in their order of occurrence, uneven rhythm, slight and inconstant mitral murmur, murmur that is constant, murmur both constant and conducted, though not always or even often permanent. Never thrill, rheumatism being absent; never accentuated beginning of the first sound; never any sound that its most passionate advocate could mistake for "presystolic." There can be little doubt that these signs concur with an endocarditis having a much more limited history than the rheumatic. They represent the utmost of choreic endocarditis, and but an early stage of the endocarditis, that is, rheumatic. But at this moment of time their physical indications are identical.

Endocarditis, there is reason to believe, occurs along with the joint pains, and not (as it seems to occur in the case of children) at some indefinite period afterwards. But the first heart signs are obscure and ambiguous, while the joint pains are early, slight, and brief. And so it habitually happens, as I believe, that the gradual development of endocarditis signs in the first rheumatic attack, the passage from the "threatened" to the "probable," and from the "probable" to the "certain" signs of that inflammation, concur with the time when the child, apparently recovered, has returned and is out of medical reach.

All this is well shown in the case of a girl (R. E.), aged

9 years, admitted to the Westminster Hospital, October 5th of last year, with her first attack of rheumatism, the joint pains being of two weeks' duration. The pain was trifling, and disappeared altogether in two days, as did the slight pyrexia. The patient, unlike most children with rheumatism, was of ruddy complexion and well nourished, but it was reported that she sweated much at night. With the pressure of more urgent cases I do not know that for this symptom alone the child would have long remained an in-patient; but for what I am about to relate and in view of these lectures she remained with us several months.

Now on October 14th, when the child was to all appearance well, the finger placed along the fourth space detects with ease and nicety the variable character of impulse and the unequal intervals between one revolution and the next. The ear confirms the finger, and adds these further signs—a lengthening of the first sound, or what some would call a just perceptible systolic murmur, and along with this reduplication of the second sound not at the base (where it is wished to be for the sake of explanation, but rarely is) but at a little above the apex.

Two days later the signs have altered. The uneven rhythm remains, the reduplication of the second sound has disappeared, and now there is an undoubted blowing murmur following the first sound. On the two next days the heart's action became almost regular, but it faltered occasionally; pulse was 96, perceptibly uneven, the mitral murmur less obvious.

During the succeeding three weeks (the patient continuing in bed, though bright and cheerful and without fever), the murmur varied, and the pulse was ill sustained; it was even as to rhythm, but unable to maintain an even force through the minutes, a feature which only becomes apparent when the observer will have the patience to keep his finger on the wrist for two or three minutes at a time.

This child, as has been said, was kept under notice from early in last October until late in January. Not to weary you with repetitions, let me call attention to two points: 1. That at the end of our watching a loud blowing murmur was established, together with signs of some heart enlargement; 2, that for a considerable time murmur, quite apparent in the recumbent position, ceased when the girl was erect. Thus mitral murmur was not fully established in this case until quite two months wholly free from disturbed health, and for some time after its establishment it was dependent on position, insomuch that were the child attending as an out-patient the discovery of valve lesion might have been long delayed.

This gradual development of mitral murmur applies, I believe, to a considerable number of rheumatic children, and is the source of much misconception. The patient is dismissed, as this child might have been, heart whole. He returns presently with a second attack of rheumatism and a mitral murmur. This latter has, in fact, developed very slowly and insidiously in the interval between the two attacks; it is the sequel of endocarditis not endocarditis itself; but the physician consulting the notes of the first admission takes it for new; he quotes it as proof that a first attack may fail to touch the heart, and that permanent reflux is not always the work of time. Both statements are, perhaps, true, but they have fewer illustrations than at first seems.

The influence of posture both upon murmur and reduplication of the second sound may often be observed in these early cases. At first with the upright posture a more vigorous systole serves altogether to abolish these morbid sounds. After a while this effect is only accomplished for a short time, the murmur appearing so soon as the effort of sitting up causes fatigue, and at last the murmur becomes permanent, and position affects it but little. In noticing such posture changes, Dr. Walshe asks: "Is this changeableness of a murmur proof of its inorganic nature? My experience," he answers in his laconic way, "would support the negative."

I have presently to speak of varying murmur which at last almost disappeared; and if once more we make comparison between the endocarditis of chorea and that of rheumatism, allowing for their distinctive characters, it might seem that what is the common event in the one case is the less common event in the other. In chorea the murmur grows, it becomes

distinctly blowing, and is conducted into the axilla; it is then recognised by most of us as a sign of endocarditis. But presently it begins to lose these characters, and eventually the normal sounds return. Such is the common experience of chorea, rheumatism apart. We are not without evidence that similar recovery sometimes obtains in rheumatic endocarditis. But, for reasons to be presently mentioned, we are without accurate information upon this point.

ABSTRACT OF THE GOULSTONIAN LECTURES ON THE PHYSICS OF THE CIRCULATION.

Delivered before the Royal College of Physicians.

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(Continued from page 515.)

WHEN fluid is driven into elastic tubes by intermittent rhythmical action, as it is into the arteries, special considerations arise. The fluid driven into the tube has not to displace the whole mass of fluid beyond it at one time. The blood driven into the aorta from the heart meets, it is true, with the inertia of the mass of fluid against which it impinges, the friction in the many arterial branches, and the obstruction of the capillaries, but the root of the aorta is distended by the increase of blood volume. This distension exerts a pressure on the enclosed fluid, which continues when the heart has ceased to contract, and consequently the blood continues to stream onward until the root of the aorta has returned to its former tension. The force of the heart is, in fact, transmuted into terms of elastic tension, which acts during the period of the heart's rest. The expenditure of work is therefore much less than if the whole mass of blood had to be moved forward *en masse*, as would be the case if the tubes were rigid.

CLINICAL USE OF THE SPHYGMOMANOMETER.

That many difficulties lie in the way of measuring, with complete accuracy, the blood pressure in the sick, is not a reason why any approximate estimation of the pressure should be completely neglected, as is now almost universally done. A useful sphygmomanometer can be made for a few shillings, and its application would in most cases be accurate enough if we could always feel, at the wrist or elsewhere, an easily accessible artery lying on a flat and rigid bed. It is better to employ a slight extension of the wrist, as, owing to the increased tension put upon the fascia, the amount of yielding beneath will then be reduced to a minimum. I have been using a sphygmomanometer made by myself on a simple plan suggested to me by Dr. Waller. A U-shaped piece of glass tubing, one limb of which is bent at a right angle, is connected, by means of a perforated india-rubber cork, with an ordinary india-rubber finger stall. This is fixed on an upright frame scaled in millimetres. When the finger stall is applied over an artery pressure is exerted until the distal pulse beyond it is no longer perceptible. The ulnar artery should always be compressed to avoid the recurrent pulsation through the palmar arch, to which attention was first called by Dr. Waller, and which has since been described by Dr. Dickinson and by Dr. Douglas Powell. Since I have used the sphygmomanometer I have met with recurrent pulse so frequently that I have ceased to consider the phenomenon to be of much clinical significance, though Dr. Douglas Powell has described it specially in connection with the pulse of high tension.

By direct measurement in mammals we know that the mean blood pressure is remarkably independent of the kind or size of the animal. By analogy we may assume that the aortic pressure in man varies between 100 and 200 mm. Hg., the mean pressure being about 150 mm. Hg.

In using the sphygmomanometer which I have just described we must allow for certain errors. According to v. Basch,¹⁸ in order to occlude an artery through the over-

¹⁸ Berl. klin. Woch., 1887.