empty pelt, the young mosquito floats on the surface of the water while its wings are drying and acquiring rigidity. When this is complete it flies away.

The young mosquito larvæ, to satisfy their prodigious appetites, devour everything eatable they come across; and one of the first things they eat, if they get the chance, is the dead body of their parent, now soft and sodden from decomposition and long immersion. They even devour their own cast-off skins. In examining mosquito larvæ one often comes across specimens whose alimentary canals are stuffed with the scales, fragments of limbs, and other remains of the parental insect and larval pelts.

THE HISTORY OF A MOSQUITO GREGARINE.

I am indebted to Surgeon-Major Ross for many valuable facts in support of my views as to the life-history of the plasmodium; amongst other facts, for the instructive story of a gregarine which inhabits the stomach and appendages of the Secunderabad mosquitos and their larvæ. He tracked the germs of this gregarine into the stomach of the mosquito larva, where, after an intracellular stage of short duration and which was not quite satisfactorily made out, it became a large, free, actively moving gregarine. At maturity the adult gregarines crawled out of the stomach and into the Malpighian tubes, along which they crept towards the cæcal end. There, with or without conjugation, they encapsulated themselves and generated in the interior of their capsules multitudes of pseudo-navicellæ. At maturity, which was attained at the nymph stage of the insect, or a little later in the perfect insect, the capsule ruptured liberating the pseudo-navicellæ in such numbers that they sometimes seemed completely to fill, and even to distend, the lumen of the Malpighian tubes. Many of the pseudo-navicellæ escaped in the fæces of the nymph; others were carried away by the mosquito when it emerged, and were emitted in the fæces of the insect, sometimes even on to the human skin while she was in the act of sucking blood. As mentioned, the development of the gregarine was sometimes effected so rapidly that numerous pseudo-navicellæ were emitted before the nymph stage of the mosquito was concluded, so that it was a common thing to find the empty nymph pelts thickly sprinkled over with multitudes of tiny gregarine germs. As we have seen that the mosquito larva devours its own and its neighbour's exuvæ, we can readily understand how, once gregarines have been introduced into a pool of water, the larval mosquitos in that particular pool become infected by the parasite. But as the mature mosquito when she quits her nymph husk also contains numerous gregarines, we can also understand how she, too, carries the infection with her, scattering it about the country in her fæces or conveying it to any other pool she may elect to lay her eggs and afterwards to die in. Her body is then devoured by her progeny or by any other mosquito larvæ that already chance to be in the pool. Along with the parental body the larvæ will swallow any gregarine germs it may contain, and very probably, when feeding on the mud at the bottom of the pool, may pick up other free pseudo-navicellæ.

Does not this little story of the gregarine indicate the way, or a way, in which that other mosquito sporozoon—the plasmodium malarie—multiplies? Does it not indicate how this parasite, in which man is so much interested, passes from mosquito to larva, from larva to mosquito in never-ending series? Does it not indicate how the plasmodium disease of mosquitos spreads from pool to pool and is scattered broadcast about the country; and does it not indicate how it may get back to man again?

DIFFUSION OF THE PLASMODIUM BY THE MOSQUITO.

We can readily understand how the mosquito-bred plasmodium may be swallowed by man in water, as so many disease germs are, and we can readily understand how it may be inhaled in dust. Mosquito-haunted pools dry up. The plasmodia in the larvæ, and those that have been scattered about in the water, finding themselves stranded by the drought and so placed in a condition unfavourable for development, pass into a resting stage, just as they do when by quinine or other means man is rendered temporarily unsuited for their active life. They may, probably do, become encysted as so many of the protozoa do in similar circumstances. The dried

sediment of the pool, blown about by winds and currents of air, is inhaled by man, and so the plasmodium may find its way to the human host, from whom its ancestors, perhaps, had started generations back. I would conjecture that on entering man and on entering the larval mosquito it develops into a flagellated spore similar to the flagellated spore it develops into in the mosquito's stomach. In this way it would be enabled to penetrate the mucous surfaces and get to the human blood cell.

Many mosquitos die without getting to water: all male mosquitos die without seeking water. They may die far from water, blown away, as we know mosquitos are, by high winds. The bodies of such mosquitos fall in time on the soil and decompose. The parasites they contained pass into the resting stage; and in this form they may be carried into the air by currents, or be blown about as dust, or be shaken out by man when he disturbs the soil. In this way, too, the plasmodium may find a route to man. In this way, too, we may explain the occurrence of those cases of malaria which apparently, though not really, are unconnected with swamp or stagnant water.

Such is my view of the life-history of the malaria parasite and the rôle of the mosquito with regard to it, and of the process by which man becomes infected. In my concluding lecture I shall bring forward further evidence in support of this theory, and endeavour to answer certain objections which have been, or which may be, brought against it.

PRACTICAL AIDS IN THE DIAGNOSIS OF PERICARDIAL EFFUSION, IN CONNECTION WITH THE QUESTION AS TO SURGICAL TREATMENT.¹

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SOME years ago, when honoured by a junior colleague with a request to examine one of his patients as to the existence of a pericardial effusion or of a cardiac dilatation, I happened to diagnose the case correctly; but on how insecure a ground my opinion was based I have since then realised. I can also bring to mind a painful instance—probably not unparalleled in the experience of others—in which I failed to perceive during the patient's life, though not from neglect of examination, the presence of a large effusion which should have been aspirated. This early mistake led me to work out gradually a more complete method of physical examination, which I trust others may find of as much advantage as it has been to myself.

The elements of the method are (1) accurate percussion and palpation, (2) careful auscultation, and (3) observation of the pulse. All the signs to be enumerated should be understood to apply to effusions sufficiently large to raise the question as to surgical interference. We cannot attempt today the more delicate diagnosis of slight and early effusion. Lastly, as our time is short, and our object a practical one, I must reserve all theoretical considerations, and put before you the facts with as little comment as possible.

I. PERCUSSION² AND PALPATION.

Pericardial distension taking place usually in all directions, vertically as well as transversely, the first requisite is a competent knowledge of the normal levels and lateral extensions of the præcordial dulness, a subject to which I have formerly given some attention.³

(a) *The Normal Boundaries of the Total Cardiac Dulness, and its Angles.*—The lower level of the healthy dulness is that of the junction of the xiphoid cartilage with the sternum: in other words the level of the infrasternal notch. It is identical with the "hepatic line" of dulness, or upper line of the absolute dulness of the liver (above which partial or modified hepatic dulness ranges as far as the "suprahepatic"

¹ Read before the North London District of the Metropolitan Branch of the British Medical Association.

² In working out these facts I have found Sansom's pleximeter of invaluable service.

³ Cf. *Lancet*, 1891, vol. ii, p. 473.

line, dotted in Fig. 1). At the lower half of the præcordium

Fig. 1.—Illustrating "Rotch's sign" (dullness in the right 5th space—s to H); also contrasting the angles (on either side of H) of the dullness as due respectively to effusion and to dilatation. The heart's outline is normal in size and position. The outer lines are those of the dullness in moderate effusions. The "suprahepatic line" (dotted) and the "hepatic line" limit the normal "modified" dullness of the liver; and H' is placed on the absolute dullness.

the lateral boundaries of the anterior projection of the entire heart are situated respectively $1\frac{1}{2}$ or $1\frac{3}{4}$ inch to the right of the sternal margin, on the right side; and on the left, just inside the nipple line. This interval comprises both the absolute cardiac dullness, which is usually small, and which also has definite normal boundaries and the total cardiac dullness. The normal *right lower angle*: Very careful percussion shows that the right boundary of the total dullness does not drop vertically on to the hepatic line. Its lower end is gently curved, and inclines inwards so as to terminate not far from the infrasternal notch; this is due in great measure to the convex shape of the right auricle, but it is also due to the resonance which arises from the overlapping lower angle of the right lung. The normal *left lower angle*: This, again, is usually rounded off, although corresponding to the angular projection of the heart's apex. As I have elsewhere pointed out, thanks to its contact with much pulmonary and with much gastric resonance, the apex invariably becomes resonant also. We do not, therefore, find the normal apex beat within, but immediately outside, the outline of dullness, which is thus blunted or rounded off in a convexity towards the left. The upper level of the retrosternal cardiac dullness does not usually receive much attention. The line joining the lower edges of the third costal cartilages separates the præcordial dullness below from the prævascular dullness above; the latter, being narrow and slight, needs care for its recognition.

(b) *The Normal Sternal Resonance*.—Although there is no lung tissue, but only the heart behind the lower half of the sternum, the latter is not dull, but more or less resonant. This is due to a strong conduction of resonance from the upper part of the bone, where this is in contact with the upper lobes of both lungs, and also from the right costal cartilages. Whenever this conducted resonance is lost we conclude that the sternum has ceased to be in contact above with the lungs. In health the absolute dullness of the heart is strictly limited towards the right by the left edge of the sternum, entirely owing to this bony resonance by conduction.

The order in which the following signs are given has not any reference to priority in time:

FIRST SIGN.

Considerable Extension of the Lateral Boundaries of the Total Area of Dullness.—In the diagram (Fig. 2), which shows the

total pericardial dullness, the border of the lungs, depicted as they are often found, does not coincide with the lateral boundaries of the distended sac, but overlaps the latter. It is the superficial resonance of their fringes and the puerile

Fig. 2.—Outline of a large effusion, which the pulmonary fringes overlap, and of its total area of dullness. The liver is depressed from its normal level H (infrasternal notch) to the tip of the xiphoid. F shows the position of the finger for the "first rib sign."

vesicular murmur arising from them which are so apt to mislead us at times into rejecting the idea of effusion. A careful percussion will guard us against this danger, and will enable us to delineate a complete outline of the sac.

SECOND SIGN.

Great Extension of the Absolute Dullness; the Sternum Absolutely Dull.—The same diagram sufficiently illustrates this point also. It is seen that the two upper lobes are widely separated, and removed from all contact with the sternum. It should be noted that any retrosternal accumulation, whether of the solid or of the fluid kind, which will bring this about would likewise completely deprive the sternum of its normal resonance. A much enlarged heart (and particularly that form of hypertrophy and dilatation arising from pericardial adhesions), aneurysms, abscesses, mediastinal growths, etc., may bring about this change, which therefore, taken apart from all others, is not absolutely diagnostic, but is of great importance when taken in conjunction with them.

THIRD SIGN.

The Depression of the Liver.—Any cardiac enlargement, and any considerable pulmonary distension will produce more or less displacement downwards of the hepatic line of absolute dullness, and enable us to feel more or less directly the epigastric pulsation of the heart; but in no other condition, except in pneumothorax and in intrathoracic sarcoma, is the hepatic depression so marked, at least in the middle line, as in large effusions. Instead of being found at the infrasternal notch, the hepatic percussion note begins at the level of the tip of the xiphoid, or even lower. As a result of this depression, the finger applied immediately below or at the side of the xiphoid can be made, by being pushed upwards and backwards, to ride over the upper surface of the liver, which normally is quite out of reach.

In another set of cases where pericardial dullness is much increased, namely, in obesity, the diaphragm is apt to rise instead of falling. If therefore any distinction were likely to be needed between the two clinical conditions, this point would be, practically speaking, diagnostic.

FOURTH SIGN.

Dr. Rotch's Sign: Dullness in the Right Fifth Intercartilaginous

Space.—Some years ago Dr. Rotch¹ published this valuable suggestion for the early diagnosis of effusion into the pericardium. The diagram (Fig. 1) will explain to you the meaning and the value of this sign. As a result of accumulation of fluid within the right corner of the sac, the usually resonant area in question becomes dull on percussion. Is this absolutely diagnostic? In other words, are there other possible sources for a dullness in this situation? In the *Glasgow Medical Journal* for April, 1894, Dr. George S. Middleton reports a remarkable case of tricuspid stenosis, with enormous dilatation of the right auricle. The diagram which illustrates the case shows considerable width of dullness at the base of the thorax; the dullness extending 2 inches outside the right, and $2\frac{1}{2}$ inches outside the left nipple line, and rising to the second rib. This outline being almost identical with that of a large pericardial dullness, the question as to the possible existence of pericardial effusion was considered, but decided in the negative. In connection with the shape of the dullness depicted, especially that of its right lower corner, it is to be regretted that the illustration does not appear to be an actual tracing from the chest, but a diagram only. We are left in doubt whether the sternum was absolutely dull. But as regarded the right fifth intercostal space near the sternum, it must be concluded from the diagram and from the *post-mortem* observations that that space was absolutely dull, owing to the retirement of the lung, just as it would have been dull as the result of effusion. Here, then, is a case in which Rotch's sign in itself could not have supplied the diagnostic verdict. We need something additional, and this need may I think be supplied.

FIFTH SIGN.

The Diagnosis Between Pericardial Effusion and Cardiac Dilatation: The Lower Angle of the Pericardial Dullness projects towards the Right.—Instead of the normal convexity of the right auricular border, which retires downwards and inwards towards the xiphoid, the outline of effusion is that of a bag of fluid spreading out at the base. As shown in the diagram, the lowermost level is also that of the greatest width of dullness from side to side, and the *lowermost angle projects outwards*. This prominent angular outline cannot belong to uncomplicated dilatation of the right heart, however large a size it may attain, owing to the fixation of the lower part of the right auricle to the orifice of the vena cava near the middle line. From the surgical standpoint the practical importance of this physical sign, which seems to be the only one establishing a diagnosis between pericardial and cardiac dilatation lies in the fact that aspiration of a dilated right auricle, mistaken for effusion, has been repeatedly recorded, and probably has occurred more often than it has been published. For a skilled percussor the method presents no difficulties, but it entails a careful percussion of the entire length of the right border of dullness, and a faithful mapping out of its outline. Tracings of this can be taken directly from the chest, and these may prove to be of much value in connection with subsequent clinical events.

SIXTH SIGN.

The Left Lower Angle of Dullness. The Relation of the Apex Beat to this Angle.—Here again the pyramidal shape of the dullness gives towards the left, instead of the somewhat rounded-off outline which is normal, a prominent angle. This alone is very significant, but it is not an absolute guide, since any cause preventing the natural overlapping of the lung over the heart may give the same result. On the other hand, a determination of the relation of the heart's apex to the left angle of dullness is of great diagnostic value. In cases of cardiac enlargement or displacement to the left, howsoever brought about, the apex beats at the extreme left limit of the dullness and at its lowest level. This is not the case in pericardial effusion. The apex cannot be felt where there is much effusion; but it will be heard beating at a spot somewhat inside and above the boundaries of dullness. The small arrows in Fig. 3 draw attention to this striking and all-important relation.

Remarks on the Position of the Heart's Apex in Pericardial Effusion.—To the surgeon about to operate an accurate knowledge of the position of the heart and of its apex is of the

first importance. Yet it may be doubted whether correct information is often possessed at the time when it is most needed. For this reason I cannot avoid warning you against a remarkable misconception hitherto perpetuated by the textbooks as to the alleged elevation of the apex within the pericardial effusion, even as high as the third interspace.

Fig. 3.—Outlines of the total and of the absolute areas of pericardial dullness. A, position of the cardiac apex (fifth space) in the effusion. The dullness is shown by the arrows to extend far beyond and below A. The right auricle (not shown) descends with the diaphragm. T, the inframammary patch of tubular breathing.

That an impulse can usually be felt there is not surprising, since the antero-posterior diameter of the chest at that level (between sternum and spine) is not much greater than that of the heart itself, whilst the left lung no longer intervenes between the latter and the chest wall. This impulse is not, however, that of the apex of the heart, but rather of its base. Whether the extraordinary mistake arose with Sibson, or was only handed down by him, I know not; still his authority probably had something to do with its long survival. In Fig. 3 the letter A is placed in the usual situation of the healthy beat; and at that spot you will find the apex at any necropsy on a case of uncomplicated pericardial effusion. And you will then note that whilst the heart has preserved its normal situation, the floor and the sides of the pericardium have receded from it.

The impossibility of the apex being raised as alleged to the third interspace by the operation of gravitation or of ordinary mechanics is almost self-evident. The vessel which I show you contains serum, or rather a fluid which my able former clinical clerk, Mr. J. L. Kirk, who is giving me his valuable assistance, has prepared of specific gravity 1.018, which is, according to Professor Halliburton, the usual density of pericardial effusions. If we drop into it a heart, this will sink like lead. Were we to enclose some blood in a thin membranous bag and introduce the bag into the jar, this also would sink to the bottom. The heart, even in diastole, cannot therefore float in serum. Slight mechanical displacement might conceivably be brought about by one circumstance only—the lifting by the distended pericardial sac of the tracheal bifurcation and of the bronchi, and with them of the pulmonary veins and of the heart. Practically this rise is very inconceivable, and moreover it does not directly influence the ventricle. On the other hand, we must remember that the heart is tethered to the bottom of the pericardium by the attachment of the inferior vena cava to the foramen quadratum in the central tendon, and that the considerable descent of the diaphragm must depress the level of the right auricle and tend to depress the apex, far from allowing it to rise. I have in some cases

¹ *Boston Medical and Surgical Journal*, 1878, vol. xcix, p. 427.

detected a lowering of the heart's apex in pericardial effusion, and with it a more median position of the heart, which then tends to hang more vertically from the aortic arch, the latter becoming slightly straightened. This I have depicted diagrammatically in my book on *Cardiac Outlines*.

The Thoracic Signs.—It is impossible to attempt a complete account of all the physical signs; but before dismissing the subject of anterior percussion and palpation, reference may be made to the great resonance of the upper part of the chest, where the lung (as happens also in pleuritic effusion) is partly retracted, and to the extreme activity of upper costal respiration, making up for the loss of diaphragmatic breathing. The mechanical results of this upper costal hyperpnoea are easily foretold; they imply fullness of the upper portion of the chest, which, however, is not symmetrical, as we shall presently note.

Among the thoracic signs there are two claiming our attention: (1) the bulging of the left chest; (2) the altered relation between the left clavicle and first rib. The bulging of the left chest is analogous to that observed in cardiac enlargement, only much greater. The left costo-cartilaginous arch is raised, and much restricted in its movements, and the fourth to sixth cartilages are more spaced than usual. The width of the fourth space is apt to be markedly increased, and the same is true of some of the upper spaces, partly in connection with the thoracic changes to be described.

SEVENTH SIGN.

The First Rib Sign.—In all cases of considerable pericardial effusion which I have examined for this sign, it was possible to feel with the finger the upper edge of the first rib as far as its sternal attachment (see *r* in Figs. 2 and 3). This points to a raising of the clavicle not only in its outer but also in its inner portion; and to a relaxation of the ligament between it and the first rib. In the absence of pericardial effusion I have rarely seen this sign, except in some cases of considerable cardiac enlargement. I regard it as specially connected with the immobility and with the elevation of the lower ribs, coupled with the great activity of the superior respiratory region of the chest. The left clavicle is apparently lifted to a higher level than it is possible for the first rib to reach.

The raising of the first rib is not of the same order as that witnessed in emphysema. In the latter its elevation is permanent; clavicle and rib move up together and remain lifted; here, on the contrary, the first rib contributes in its limited range to expiratory as well as to inspiratory movement. Moreover, the spinal mechanism is not the same in the two affections. There is here no rounding of the shoulders. On looking at the chest the general impression is rather that it is bowed out towards the left, the spine being probably bowed likewise by the increased internal pressure, and as a result of the attitude assumed for the relief of respiration. This sign has the advantage of being easily studied without disturbing the patient, and constitutes a definite and useful addition to our clinical evidence of pericardial effusion.

Signs Derived by Percussion in the Back: the Normal Dorsal Percussion.—Although little attention has been given to this subject, it is in the back that the crucial signs are to be found upon which for some time past I have most relied in the diagnosis of pericardial effusion, and in particular that dulness which I venture for convenience to term the posterior pericardial patch of dulness. Here again, as in front, familiarity with the normal percussion is requisite. As I cannot on this occasion enter into much detail, let me simply give you the assurance that the traditional statement that the lung extends posteriorly down to the tenth rib is a traditional error; the lung extends in health a great deal lower than this, and its resonance would be found by any of you on percussion to reach the upper border of the twelfth rib. This knowledge will enable you to appreciate the value of the following remarks.

EIGHTH SIGN.

The Posterior Pericardial Patch of Dulness.—Whenever fluid is effused into the pericardium the normal resonance is modified at the left posterior base in a most definite way. A patch of marked dulness (shown in Fig. 4) is found at the left inner base, extending from the spine for varying distances

outwards, usually not quite so far as the scapular (angle) line, and ceasing abruptly with a vertical outer boundary. Above, its extension is also variable, according to the size of the effusion; commonly it does not extend higher than the level of the ninth or tenth rib, and here again its horizontal boundary is abrupt. Its shape then is that of a square, and it is quite unlike that of any dulness arising from pleuritic effusion. You will not experience any difficulty in identifying the patch in question. Rather greater care in percussion is needed, however, to follow the dulness as it extends to the corresponding vertebræ, and for a short distance also to the right of them. For some time I had overlooked this extension, which, owing to the general resonance of the right base, is one of partial dulness only. When, however, the effusion is considerable, the extension of the patch in the right chest may become almost absolutely dull.

Fig. 4.—The "posterior pericardial patch of dulness" (shaded); and the "posterior pericardial patch of tubular breathing and ægophony."

I wish time permitted me to discuss with you the significance and the probable mechanism of production of this singular and most helpful sign. It is best I should confine myself on this occasion to practical points. The value of this sign is that, unlike many others, it is very sharply defined, and does not fit any other diagnosis. When, in a doubtful case, all the signs observed in front support the diagnosis of effusion, and this sign is also found, we have then in hand complete and crucial evidence of the existence of fluid; whilst when, as sometimes occurs, previous adhesion of the anterior surface of the heart to the chest wall renders diagnosis extremely difficult, this help is invaluable, and its place, so far as I am aware, cannot be supplied by any other available diagnostic method.

Signs derived by Auscultation in Front and Behind.—To the auscultatory signs we can only devote very cursory remarks. Anteriorly the first feature is the hyperpnoea, and the puerile breathing heard over the upper lobes; and, in severe or protracted cases, the catarrh set up within them, which tends to produce their further inflation, and to attenuate over a greater surface the dulness due to the distended sac.

NINTH SIGN.

Tubular Breathing Below the Right Mamma.—Although not constant, this sign, which does not appear to have been noticed, should be looked for in severe cases. At the anterior base (see *r* in Fig. 3), usually in the nipple line, and a little above the hepatic line, distinct tubular breathing is audible, which is sometimes restricted to expiration. Its localisation is due, I venture to think, to the active respiratory draught kept up by the movements of the upper lobe at the origin of

the bronchus for the middle lobe, coupled with the compression of the latter in the situation named, laterally by the base of the pyramidal collection of fluid, and above by the freely expanded upper lobe, often also by a superadded pleural effusion.

It is at the back that the most characteristic signs are to be heard. On auscultating the dull patch to the left of the spine respiratory sounds are found to be absent, and the voice sounds feeble.

TENTH SIGN.

The Posterior Pericardial Patch of Tubular Breathing and Egophony.—Immediately below or slightly to the left of the tip of the left scapula a patch about 2 inches in diameter presents well-marked tubular breathing and egophony (see r; in Fig. 4). This sign, although not so important as that of the patch of dullness, is very commonly, if not always, present in cases of considerable effusion, and gives valuable confirmation to other signs. It has been described by other observers. The mechanism of its production is analogous to that suggested above, and is doubtless connected with pressure on the bronchi descending to that district, and with partial collapse of the pulmonary tissues. It also occurs in pleural effusions.

ELEVENTH SIGN.

The Secondary Pleural Effusions.—Pleuritic effusion is among the most common complications of severe pericardial effusion, and is probably to be regarded as induced mechanically by pressure. If it should be limited to the right side, an opportunity would be given of contrasting the outline and situation of the pericardial patch of dullness with those of the dullness peculiar to pleural effusions. It frequently begins in the right pleura; but it is not uncommon for the effusion to occur ultimately on both sides. Its occurrence belongs to the later rather than the earlier stages, and therefore its diagnostic value is practically less.

The Pulse in Pericardial Effusion.—You are all familiar with the classical description of the *Pulsus cum inspiratione intermittens*. This is an important sign. I have occasionally observed it in pyopericardium; but it is characteristic of mediastinal rather than of pericardial disease, and it cannot be regarded as diagnostic of the latter.

TWELFTH SIGN.

The Large and Slapping Pulse of Pericardial Effusion.—I have frequently observed in cases of pericardial effusion an opposite condition of the pulse. Attention has not been pointedly directed to it; but, strange to say, it is incidentally mentioned in the light of a tactile hallucination in Marey's book on *La Circulation du Sang*, where a sphygmogram is given to show that the pulse is really small. Marey instances this as an example of the "illusions of touch," causing us to feel a larger wave than is actually produced. I cannot share in his view of the facts. The sphygmograph seems to me to be at fault, not the finger. The peculiarity of the pulse is its great size and velocity of impact, and the sudden collapse of the wave. In fact, it is Corrigan's pulse, almost of a typical kind, though never so extreme as in well-marked aortic regurgitation. Its occurrence under the circumstances of effusion may throw an interesting side-light on the mechanism of its production in aortic valvular disease. Time, however, forbids our discussing the suggestions which might be offered towards an explanation of its mode of origin in the affection we are studying.

CONCLUDING REMARKS.

In this imperfect sketch I have not dwelt upon various familiar but valuable signs, such as friction sounds and fremitus, nor on the symptoms of pain, alteration of respiration, position in bed, etc. My endeavour has been to add to your supply of available signs some which are novel and others which are not commonly described, such as might in conjunction with the old ones facilitate your diagnosis of any case. A full presentation of the clinical subject of pericardial effusion would be a much longer task than we could deal with at this short meeting.

Turning now to the practical application of the method, I believe that the confidence of the surgeon in operating upon any given case will be increased by a knowledge that

all the typical signs pointing to a normal effusion have been obtained.

The most difficult and anxious part of our subject is, however, one to which I have not yet alluded. In practice we must always be prepared to find a wide difference between the classical account of a disease and our patient's individual case. Pericarditis is no exception; and the irregular cases present special importance for the surgeon. The fact is that effusion may supervene, either after a previous attack which has left the heart adherent to some part of the sac, or after an early fibrinous stage more than usually agglutinative, leading to soft adhesions—perhaps likewise limited to one side—and to a partial filling of the sac with heavy gelatinous masses of fibrin. To such cases the ordinary rules as to the position of the heart do not apply, and it becomes our duty to work out for ourselves the special conditions of each case. This is often a very difficult task; still we must not despair of its accomplishment, but, on the contrary, regard it as our duty to spare no trouble till it is attained. At any rate, the surgeon whom we may call in to operate will expect us to satisfy him as to the position of the cardiac apex, and to give definite reasons for our view as to the relative positions of the heart and of the bulk of the fluid.

In dealing with difficult cases of this kind we shall appreciate the value of the systematic study which we may have devoted to the typical normal cases, as supplying a definite starting point for our investigation, and a standard with which we may gauge the extent of the irregularity peculiar to any given case. Bearing in mind the relative safety of the operation for opening the pleura, and even the pericardium, by incision, it behoves us to endeavour by every means within our reach to render equally safe as regards the heart, and as satisfactory as possible for the efficient relief of the effusion, the apparently more simple but decidedly more uncertain operation of aspiration.

ON MECHANICAL SUPPORT OF THE LUNG IN PHTHISIS.

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THE suggestion to strap the chest in phthisis is invariably met with the objection that one of the objects of treatment in that disease is to expand the chest and so call into play portions of lung which in conditions of sub-active health take little or no share in the respiratory process. This objection does not recognise the change in the mechanical conditions of respiration produced by disease of the lung substance. In health, the respiratory capacity of the lung is commensurate with the semi-circumference of the thorax. The diseased lung may be regarded as decreased in bulk, so far as function is concerned, by the amount of lung tissue involved, and it follows that the best mechanical conditions of respiration will be secured by reducing the thoracic cavity in proportion to the reduced bulk of lung. Natural processes of repair tend to secure these conditions, but only lead to complete cicatrization when the disease is of very limited extent. We then find, clinically, a flattening of the chest wall, dislocation of adjacent organs or hypertrophy of the opposite lung, and anatomically a cicatrix, bands of fibrous tissue, a puckered lung surface and thickened pleura—all evidences of a natural effort to fill up space originally occupied by healthy active lung tissue. Natural processes are unable to overcome the mechanical obstacles to complete cicatrization when the disease involves more than a small maximum of lung tissue. Bands of cicatricial tissue radiate from the focus of disease to surrounding parts and, contracting, draw inwards healthy lung to occupy the diseased area and the chest wall closes in and accommodates itself to the diminished bulk of lung. Auxiliary processes are pleuritic thickening and emphysematous dilatation of the alveoli about the diseased area.¹ All these processes are slow and uncertain. The cicatricial traction is itself a source of chronic irritation. The neighbourhood of the fibrous bands and the emphysematous zones are *loci resistentie minoris*, and form favourite lines of march

¹ Douglas Powell *Diseases of the Lungs*, p. 429.