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.

for the invading specific element of the disease. The obvious indication then is to aid the natural processes of repair by reducing the thoracic cavity, thereby relieving them of the work of strenuous, prolonged, and often inefficient cicatricial traction. In so far, then, as the inflammatory element in phthisis is concerned, rest and relaxation of the inflamed tissues seem indicated and essential to the healing process.

Comparative rest of the lung may be secured by keeping the patient at absolute rest day and night, but this system has obvious disadvantages, while it fails to fulfil the local indications of rest with relaxation. The English school aims at developing the latent resources of the lung so as to compensate for the loss of respiratory surface, while placing the patient under conditions inimical to the extension of the specific element of the disease and promoting general nutrition. In extreme cases where this object has been attained, one finds an immense development of the base of the lung with extensive cavitation towards the apex. The cavitation is a constant source of danger, while the hypertrophy does not necessarily secure proportionately increased aëration of blood, because the latter depends on range of movement as well as on effective respiratory area. With permanently increased circumference of the lower thorax there must necessarily be decreased range of movement. Such cases suggest how much better it would have been if the compensatory hypertrophy had extended inwards instead of outwards so as to have invaded and occupied the excavated region.

Two clinical considerations lend weight to the argument in favour of strapping the chest in phthisis, namely, (1) The fact that pleuritic effusion frequently leads to amelioration of the lung affection in phthisis; (2) The fact that during pregnancy the course of phthisis is often temporarily arrested. Both these phenomena produce similar effects on the lung as that produced by strapping.

The above considerations led me to the practice of strapping the chest wall as part of the treatment in phthisis, especially applicable in cases of unilateral disease of the apex. The method I adopt is to apply the anterior end of the strapping over the lower sternum and direct the patient to breathe out while I draw the strapping round the lower thorax and fix it over the vertebral column. I use either strips of strapping or a suitably sized (14 inches by 5 inches) piece of chamois leather spread with plaster, an appliance which adapts itself well to the chest wall without giving rise to discomfort. The appliance should be kept on till all signs of disease have disappeared and healing is complete. I direct the patient to take a moderate amount of regular exercise and to practise deep breathing. The exercise and deep breathing tend to bring into play all portions of lung not habitually active, while the strapping directs healthy lung to encroach on the diseased region.

The immediate effects of strapping, so far as my experience goes, are decrease of cough and expectoration, followed by cessation of night sweats, gradual fall of temperature, and improvement of general symptoms. The effect on the physical signs is to show a dislocation upwards of the seat of lesion, with gradually decreasing evidences of disease, an encroachment of abdominal organs towards the thoracic cavity, and an absence of falling in of the upper chest wall.

The advantages of strapping may be briefly summarised as follows:

1. In early phthisis (catarrhal stage) to give comparative rest and relaxation to affected lung tissue.
2. In the stage of consolidation, to secure the same results, thereby limiting the risk of extension and to promote elimination of the disease products by improving the circulation in and about the diseased area and to facilitate expectoration.
3. In the stage of cavitation, to promote closing of cavities by directing healthy lung to encroach on the diseased area instead of relying on natural processes of cicatrization.
4. Diminished tendency to hæmorrhage by reduced tension on vessels and cicatricial traction on vessel walls.
5. The ultimate object is to obtain a smaller thoracic cavity filled with healthy lung instead of an enlarged thoracic cavity partly filled with diseased lung.

CASE I.—Female, aged 21. History of tuberculous peritonitis. When she came under observation there was marked hectic, extensive breaking down in right infrascapular region, troublesome cough and copious expectoration. When strapped the cough was diminished at once and the

temperature gradually fell to subnormal and remained there. The physical signs moved upwards and decreased in area till they finally disappeared. The thorax was strapped for two winter seasons and patient sent home with strapping on. This spring—five years after original strapping—patient is reported to me as well.

CASE II.—Officer in artillery. Patient had repeated hæmoptysis, extensive breaking down in infrascapular region, cough and free expectoration. Was seen in consultation with leading London authority and bad prognosis expressed. Had been refused readmission to the service on one or two occasions by board of medical examiners. The last I heard of this patient was that he had rejoined his regiment.

CASE III.—Student, aged 19. Had been seen by family practitioner and by consulting physician. There was catarrh of left apex, and bacilli were found in abundance. Strapped for one season, after which his family doctor saw him in consultation with me, and could detect no disease. I advised colonial life instead of Cambridge; however, he was considered well enough to return to Cambridge, and broke down again.

CASE IV.—Female, aged 22. Infrascapular consolidation; cough, expectoration, and night sweats. Strapping was followed by relief of these symptoms. Two months later: Symptoms have all disappeared, and the signs indicate a greatly decreased area of disease.

I wish it to be understood that I do not propose this method as a cure for phthisis, but as an essential element in the treatment of that disease directed against the inflammatory lesion, an element which will greatly enhance the value of specific and climatic treatment, and without which those methods will be powerless to restore an extensively damaged lung to a condition of mechanical integrity. The latter essential is now habitually left to natural conservative processes which are inadequate to fulfil it when the lesion is extensive, and only does so with difficulty when it is limited.

PRELIMINARY NOTE ON THE ACTIVE SUBSTANCE IN THE THYROID.

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PAPERS have recently appeared by Baumann,¹ by Fraenkel,² and by Drechsel,³ each of whom claims to have discovered the active ingredient of the thyroid gland. Baumann's substance was obtained by boiling entire thyroids with sulphuric acid. He obtained as a residue an amorphous substance containing iodine, from which he was further able to extract a compound, to which he has given the name thyroiodin, and which he says he has found to be clinically active. The bodies isolated by Fraenkel and by Drechsel are very different in their nature. They are contained in the proteid-free watery extract of thyroids, and are stated to be crystalline substances. Their discoverers assert that they are active in animals, but have advanced no definite proof of clinical activity.

The time, therefore, seems ripe for the publication, in at least a preliminary form, of the results of certain observations which I have been carrying out during the last few months as to the effects of the clinical administration of the different ingredients of the thyroid. As idiosyncrasy plays a large part in these effects, I began always by estimating the reaction of the patient to a preparation of known activity—ordinary thyroid tabloids being the preparation employed. This having been ascertained, I compared with it the results of the administration of an isolated ingredient of the gland. The product of a mass of sheep's thyroids was first divided into proteids and proteid-free watery extract. The proteids were found to be active. These proteids are practically only two in number—a nucleo-albumin and the colloid matter. The former was inactive, the latter active.

The proteid-free watery extract was then concentrated. It would contain the bodies of Fraenkel and of Drechsel. It was found, even in very large doses, to produce no effect on a patient who had reacted distinctly to thyroid tabloids and to the isolated colloid matter.

The colloid matter is therefore the active ingredient of the thyroid. I have isolated it in a state of purity, but I do not propose to deal here with its chemistry. At present I shall only mention that, amongst other peculiarities, it contains a considerable quantity of iodine in organic combination, and I have succeeded in splitting off from it a body apparently identical with that obtained by Baumann from the entire

¹ Baumann, *Ztsch. f. phys. Chem.*, Bd. xxi, p. 319.

² Fraenkel, *Wiener med. Blätter*, No. 48, 1895.

³ Drechsel, *Centralbl. f. Physiologie*, February 22nd, 1896.