going so far as to deny the possibility of such influence, it is demonstrably inadequate to produce the result; for it has been conclusively proved that if, after section of the trigeminus on one side, the eye be carefully nursed, *i.e.*, protected from what may be called rough accidental injury during the process, no change whatever ensues; and, further, that if both eyes be subjected simultaneously to irritation of the same kind and degree, the paralysed eye is not one whit more affected than the sound one;* so that there is no ground, so far as the cornea is concerned, for supposing that loss of innervation acts either as an exciting or as a predisposing cause of inflammation.

This being admitted, it is evident that the real cause of damage is traumatic, *i.e.*, it is dependent on the fact that the unprotected and feelingless eye is brought by the animal repeatedly into collision with surrounding objects in such a way as to spoil the surface of the cornea; and we are justified in this conclusion by the observation that a spoiling of exactly the same kind can be produced experimentally by mechanical means, provided these are of such a nature as to leave the epithelium intact. But the question which now interests us lies behind this. What we have to ask is, not how the surface was damaged, but how it happens that this damage leads to inflammation. A dozen years ago, we should without hesitation have said, the disordered nutrition of the non-vascular cornea extends by virtue of continuity of structure to the vascular limbus, causing the vessels to exude liquor sanguinis, and the connective elements in their neighbourhood to proliferate. Such an explanation is no longer possible, for we know that the primary damage has nothing to do with proliferation; its condition is simply (to borrow Mr. Lister's expressive language) "approximated to the condition of ordinary matter." The question is, how does this purely negative change acquire the power of so affecting, or as we may, without assuming too much, call it, infecting, the surrounding vascular tissues? The answer which modern pathologists make to this question is simply—micrococci; and this, indeed, is the only one that can be given; for neuroparalytic keratitis is a type of what may be called a mycotic inflammation, one of which the spread from tissue to tissue is accompanied by the growth and multiplication of those organisms.

Twenty, or even ten, years ago, we should, for want of the means of observation, have failed to recognise them. Now, their existence can be determined (thanks to recent methods of preparation) with as much certainty as pus-corpuscles, and almost as easily. Admitting all this, we have still not got to the root of the matter. In the case of the cornea, it is scarcely necessary to show that, however probable it may be that micrococci are the agents by which the primary lesion extends its influence to the surrounding vascular structures, their appearance on the scene is subsequent to the damage of which they are, in fact, among the pathological consequences. In other words, while their presence is the proximate cause of the secondary iritis and conjunctivitis, it does not follow that they are but originators of the whole process. The question of the mode of origin of acute abscesses is a similar one. Dr. Ogston has, I think, pretty conclusively shown that micrococci invariably take part in this process, and act as if they were the sparks from which every such inflammatory focus is kindled.† All this we may admit, and at the same time demur to the inference that he seems to draw from it, namely, that acute inflammation in general depends on the morbid action of septic organisms. As regards this particular instance, we have sufficient reason for demurring, in the consideration that an acute abscess is no more a primary inflammatory lesion than the conjunctivitis and iritis with which we have compared it. An abscess does not come into existence in a healthy organ *d'emblée*; it must always have been preceded by an inflammatory process. The proof, therefore, that micrococci were concerned in its origin, even if it could be carried so far as to show that around them the first pus-corpuscles grouped themselves, leaves the question of the origin of the preceding inflammation still open.

In a paper published in the Royal Medical and Chirurgical Society's *Transactions* in 1873‡, I endeavoured to show, on the ground of experiment, that the only inflammations to which minute organisms stand in relation are those which, from their proved dependence on previously existing inflammation, may be properly termed secondary or infective; and, consequently, that the organisms in question were, in all probability, not so much mischief-makers as mischief-spreaders. That is to say, although an inflammation may come into existence without their aid, their presence communicates to it, after it has come into existence, the power of reproducing itself in previously healthy tissues, whether by extension or by dissemination. During the ten years which have

elapsed since 1872, the question of the intervention of micrococci, which was then strange even to professed pathologists, has become familiar to every one, and now the tendency exists to believe that germs explain everything; so that, whereas formerly one had to vindicate the very existence of such things as parts of pathological processes, it has now become one's business to protest, with all needful vehemence, against the attribution to them of functions which they do not possess. It will perhaps tend to clearness (which, in relation to so complicated an inquiry, is a matter of moment), as well as to the saving of time, if I submit to you at once the propositions which seem to represent the actual state of our knowledge on the subject. They are (1) that the exudation of a normal inflammation is not infective; (2) that no organisms endowed with inflammation-producing (for which term the word phlogogenic may be conveniently substituted) functions exist in the atmosphere, or in the ordinary aqueous liquids with which our bodies come into contact; (3) that whenever an inflammation becomes infective, it owes that property to chemical change in the exudation liquid, of which the presence of microzymes is a necessary condition; (4) that the introduction into the lymphatics, and eventually into the blood-stream, of microzymes which have been grown in septic exudations, is one of the mechanisms by which the seeds from which secondary or infective inflammations spring, are disseminated. Thus we attribute to our organisms two distinct functions, that of developing what may be called the phlogogenic infection, and that of conveying it to all parts of the body. What we deny is, that there exists at present any evidence that the infection in question actually originates in any other way than by the development of a septic process inside the organism. These several points I will endeavour to establish on the basis of experiment.

[To be continued.]

THE GULSTONIAN LECTURES

ON

PULMONARY CAVITIES: THEIR ORIGIN, GROWTH, AND REPAIR.

By WILLIAM EWART, M.D. Cantab., F.R.C.P.,

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LECTURE III.—PART I.

MR. PRESIDENT, FELLOWS, AND GENTLEMEN,—I turn with a feeling of relief, which you doubtless share, from the consideration of the destructive activities of phthisis to the methods adopted by nature for the healing of the pulmonary lesions. The doctrine of the curability of pulmonary excavation is comparatively young. Although Hippocrates had noticed the beneficial effects of the rupture of pulmonary abscesses into the bronchi, and founded upon this discovery a method of treatment by succussion, the recognition of cavities during life remained matter of inference rather than of observation; and speculations as to the possibility of their healing were controlled by no scientific test, until, with the progress of medicine, it became possible to follow the history of a cavity from its origin to its ultimate stages. To Laënnec belongs the credit of having pointed out the reparative action frequently observed in phthisis (see Cases XVII to XXIII, *Traité de l'Auscultation Médiate*). By him, two degrees are recognised in the healing of cavities: (1) the transformation of an ulcerating excavation into what he terms a fibro-cartilaginous fistula (see Cases XVII to XIX, *loc. cit.*); (2) absolute closure by means of cicatrisation.

With Laënnec, we are justified in considering the dwindling of cavities and the fibrous metamorphosis of their walls to be a step towards cure; and I have no hesitation in stating that a fair proportion of pulmonary cavities not resulting from acute phthisis undergo more or less of this reparative action. Of the complete cicatrisation of cavities, I am unable to speak with the same confidence. Roger and Boudet's observations on the frequency of concretions and scars in the lungs (present, according to them, in four-fifths of the necropsies performed on subjects beyond seventy years of age) give us a high idea both of the prevalence of cavities and of their complete obsolescence. More recently, Heitler ("Ueber die Spontan Heilung der Lungen-Schwindsucht", *Anzeiger der k. k. Gesellschaft der Aerzte in Wien*, 3 Juin, 1880), from a study of the *post mortem* registers of the Vienna a

* Senfleben, "Nachträgliche Bemerkungen", Virchow's *Archiv*, vol. 72.

† "Micro-Organisms in Surgical Diseases", *BRITISH MEDICAL JOURNAL*, March 12th, 1881.

‡ On the Infective Product of Acute Inflammation", *Med.-Chir. Trans.*, vol. vi, p. 345.

Pathologisch-Anatomische Anstalt for a period of ten years, has collected seven hundred and eighty instances of healing or of obsolescence of tubercular lesions, among which he mentions several cases of more or less complete cicatrization of cavities; and he admits the curability even of large caverns, provided the latter be strictly confined to the upper lobe.

But the opposite side of the question is supported by weighty authorities. By Andral, the alleged remnants of an ancient phthisis were viewed with some scepticism. They failed to convince Louis, who says that he never found at the apices those masses of membrane with dilated bronchial ramifications terminating in them, which Laënnec records as cicatrices of tuberculous cavities. Dr. Walshe alludes to the subject in very guarded terms as follows: "If on a small scale, cavities may probably cohere by their opposite walls, of the cicatrization of a large excavation I have in vain looked for an example; and, without meaning to assert the sheer impossibility of the event, I must maintain it to rank among the *mirabilia* of morbid anatomy." (*Practical Treatise on Diseases of the Lung*, 4th ed., p. 470.) A limit is undoubtedly given to the cicatrization of cavities by their size. It would be rash, however, to state that the smaller cavities alone are susceptible of healing. I am informed by physicians of experience that vomicae of considerable dimensions, which had come under their observation during life, have been recognised after death to have perfectly healed. Instances of this nature are given in the late Dr. H. W. Fuller's *Treatise on the Diseases of the Chest*, p. 405-10. Mine has not been the good fortune to witness hitherto this happy result, nor do I possess any specimens illustrating complete healing of large cavities. If for no other reasons, such specimens are probably rare, owing to the long interval of life which may succeed the disease. But partial cicatrization I have occasionally witnessed in cavities of good size, and an example of this kind will be placed before you.

On the other hand, the array of clinical evidence in favour of the healing of vomicae is imposing. The number of cases in which cavities cease to give signs of their presence is very large. As an instance of this class, let me briefly allude to the following case.

Henry S., aged 22, a tall thin man, of pale and rather muddy complexion, first applied for treatment at the end of January 1881. He had suffered from slight cough for three years; but this symptom had only during the last three months become distressing; and during the same comparatively short period his general health had failed noticeably, although not to an alarming extent, for he had followed his daily occupation as a tailor up to five days previous to his first visit. On examination, gurgling cavernous sounds and pectoriloquy were found to extend anteriorly over the upper two-thirds of the left chest; the right lung yielded normal breathing sounds. He made rapid progress under tonic treatment. On March 8th, the cavity was found to have materially diminished; and when he was again examined, on May 24th, a remarkable change had taken place. The cavity was reduced to small proportions, whilst the resonant note of the healthy right lung could be obtained for a considerable distance beyond the middle line of the sternum. The heart was in a great measure uncovered. Measurements of the chest taken the same day gave $14\frac{1}{2}$ inches for the right side, $14\frac{1}{4}$ inches for the left. He was subsequently admitted into the hospital under Dr. R. E. Thompson's care. On June 29th, no cavernous breathing could be detected; but the signs of fibrosis were well developed at the upper and outer part of the left lung, and large creaking sounds and an almost gurgling "suction-sound" were audible in the upper half of the axillary region. He left the hospital much improved in strength on July 17th, a striking instance of the rapid and practically complete disappearance of the signs of excavation. Unfortunately, two days later he was seized with severe hæmoptysis, an accident very liable to supervene in cases of excavation undergoing fibrosis; and he died on July 27th, after several recurrences of the bleeding. No *post mortem* examination was performed.

Instances of a similar nature are far from uncommon, in which life is prolonged for considerable periods. But an absence of the physical signs of a cavity does not of necessity imply complete cicatrization. It is usual to find after death that the cavity is rather collapsed than obliterated; and that, in a strict sense, healing has not been accomplished.

I freely confess that, after careful consideration, my conclusions do not materially differ from those of Dr. Walshe. Absolute closure occurs only in cavities of moderate size, but is less common than imperfect cicatrization with implication of cheesy or calcareous residues. Large cavities, although in rare instances they may completely heal, as a rule go through the reparative process too slowly to reach obliteration before life is brought to a close.

Too often the local efforts at repair, observable in cavities, are not evidence that a favourable course is taken by the disease itself. Whilst

cavities are approaching closure, consumption may be fatally progressing. Conversely, the activity of phthisis may have become extinguished where some excavation still persists as an abiding danger; I would insist upon this last combination, which practically represents the full measure of the hopes we entertain in the majority of cases. What I have said not only provides an excuse, but points to a necessity for an independent study of the local processes of healing.

Arrested phthisis is a correct expression as applied to the lung-disease, but it inadequately describes the condition of cavities which have ceased to extend. The latter are not only arrested, but in retrocession. Cavities are never at a standstill, but must either grow or decrease. The course, degree, and the mode of the reparative changes within them are mainly governed by the following factors: 1. their size and number; 2. their situation; 3. the facilities for contraction; 4. the facilities for compensatory hypertrophy.

Let me begin the discussion of these various influences with a brief allusion to the subject of *compensatory hypertrophy*. A vicarious development of the spongy tissue is not only seen in cases of recovery, but among the fatal cases in all those where the disease runs a chronic course. Upon cavities the beneficial influence of this compensation is twofold: it not only acts indirectly by improving the general condition of the system, but it will be shown by its gentle and sustained pressure mechanically to assist the contraction of cavities. A question spontaneously arises as to the nature of this hypertrophy. According to Dr. Douglas Powell, the practical results will decide in each case whether the hypertrophy is of a true or of a false kind. In true hypertrophy, recovery may be practically complete, the breathing is easy, the chest of fair size, the fitness for exertion good. Conditions are different in the other instance: the chest may be large, but the patient is constantly breathless on the least exertion; the lung is in the condition so well described by you, Mr. President, as "hypertrophous emphysema", in which the bulk of the organ is not a measure of its power.

I am not convinced that we have to deal in either of these cases with a genuine hypertrophy, in the sense of new formation of parenchyma. The bronchial tree, with its appended air-sacs, must be considered as originally perfect and not admitting of reconstruction; and we are assured by Dr. Waters that air-cells are incapable of being regenerated. I believe that the differences between so-called true and false hypertrophy are susceptible of a simple explanation by the more or less of development of the vascular system of the alveolus. As long as the alveolus is submitted to a tension, for which neither its walls nor its capillaries are fitted, impaired respiratory function and impairment of circulation must follow. But supposing the energy of growth be sufficiently great to accommodate the air-cells to new conditions, the alveolus may be enlarged, and yet not suffer from over-stretching. This view equally explains the recovery of younger subjects, even after extensive loss of breathing-surface; and the opposite result observed in the aged, where the formative powers are too feeble to adapt the dilated air-cells to their increasing duties.

Thanks to this secondary expansion, the damaged organ, even after extreme excavation, may reconquer almost the whole of the thoracic space allotted to it; but, in the majority of cases, the work of compensation devolves upon the healthy lung. Of this unilateral hypertrophy, the specimen which I have placed on the table is a striking illustration. When opened after death, the chest in this case presented for inspection anteriorly as far as the left axillary border nothing beyond right lung and heart, the left lung being entirely hidden from view. Had the comparatively healthy right lung been tied down by adhesions, such a remarkable degree of vicarious development would have been obviously impossible. We are reminded by the specimen, not only of the value of compensatory hypertrophy, but also of the importance which attaches to the presence or absence of pleural adhesions.

From practical surgery we learn that the healing of large tracts of tissue is only possible at the cost of considerable contraction. In the skin this is well exemplified after destructive diseases or lesions, and a similar contraction is the means of recovery in visceral affections (e.g., syphilis, infarcts, scrofula, etc.). Thus, it may be stated generally that the facilities for healing are commensurate with the opportunities afforded for this essential preliminary. As applied to pulmonary excavations, the term "contraction" is clearly a figurative expression. Strictly speaking, cavities cannot originate, but only suffer, contraction. They are entirely dependent for any alteration in their shape upon external agencies, and, above all, upon the condition of the tissue by which they are surrounded. The boundary zone may be in one of five conditions:—(1) in recent consolidation; (2) in chronic pneumonia with fibroid tendency; (3) in simple fibrosis, due to collapse of lung,

tissue; (4) in simple fibrosis connected with thickening of the pleura; (5) in a normal spongy state.

The first or pneumonic condition is absolutely unfitted for contraction. The acutely pneumonic lung is too much stiffened by inflammatory deposits, or sodden from cedema, to be the subject of any noticeable shrinking. But if chronic, and sufficiently moderate, the pneumonic action in phthisis is usually combined, as I have elsewhere insisted, with more or less of fibrosis, which may ultimately arrest all further progress of the disease, whilst it lays slowly the foundation for further contraction.

The fibrous ring, to which the contraction of cavities is mainly referable, is not necessarily an inflammatory product. It often becomes developed, as it were, passively, from a layer of areolar tissue which has become compressed or collapsed. A zone of unexpanded lung-tissue is habitually observed at the periphery of pneumonic excavations, being the outcome partly of the pneumonia and partly of the softening. All pneumonia implies relative fulness of the inflamed air-cells, and imperfect expansion of the neighbouring alveoli. In fibrinous pneumonia, this fulness is rendered manifest by the rib-marks at the surface of the lung. Whilst admitting, with Dr. Douglas Powell, that this indentation is in a great measure due to the collapsed state of the thoracic wall, and to the fixation of the ribs in a position of expiration, it appears to me undeniable that the lung-tissue is, relatively to this expiratory condition, expanded. Similarly, in catarrhal pneumonia, the relative distension of the affected districts is evidenced by the sharp outline and often slightly tumefied surface of the deposits, by the side of which the surrounding tissue is imperfectly expanded. The zone of lung-substance thus compressed generally survives, whilst the pneumonic consolidation softens into a cavity; and it supplies the basis of the fibrous wall of the cavity.

But the process of excavation itself is the chief cause of the alveolar collapse to which I have alluded. Softening invariably leads to an interruption of the supply of air over a corresponding region, whether from ulceration of the bronchioles, from their inflammatory occlusion, or from tubercular deposit within them.

The zone of pulmonary collapse constitutes an important factor in the subsequent contraction, owing to the elastic elements which its tissue abundantly contains; but, sooner or later, fibrosis is set up within it, and a fibrous contraction is substituted for the elastic forces natural to the pulmonary structures.

The formation of fibrous tissue around cavities is greatly assisted, whenever the cavity is superficial, by the existence of pleural adhesions. The supply of fibrous tissue from this source is not subject to any limitation; indeed, exuberance of fibrous growth may in its turn become a danger, inasmuch as it opposes the constriction of cavities under expiratory efforts, and the voiding of their contents by the agency of cough. The best results due to the contractility of fibroid tissue are seen in the thin walls of cavities situated at a distance from the pleura, in the midst of healthy lung-tissue. Here the contraction of the fibrous element receives material assistance from the pressure of the adjoining spongy substance, provided the cavity-membrane, although free from rigidity, be sufficiently strong to resist expansion under the weight of the atmosphere. These conditions are often combined in the cavities, which result from the sudden discharge of the products of softening into the bronchus. Relieved from internal pressure, the elastic and fibrous constituents of the investing layer spontaneously contract.

The influence which atmospheric pressure may exert in aiding or in opposing this contraction deserves to be briefly discussed. If an ordinary cavity, which I will suppose to possess a patent bronchus, could be imagined to be connected on all sides with the parietes of the thorax by means of some unyielding substance, lateral traction would be exerted around it equal to the inspiratory expansion of the chest. Let the connection be established by means of spongy tissue, by nature remarkably yielding, then not only will the outward traction mentioned above be removed from the cavity, but some degree of pressure will be exerted upon its outer wall by the expanding alveoli. These views are illustrated in the accompanying diagram: *w.* represents the atmospheric pressure acting upon the inner surface of the cavity, where it is supported by a resistance *R.* equal to it. Similarly, the weight of the atmosphere acts upon the inner surface of the alveoli where it is opposed by a smaller force *E. R.*, which represents the elastic resistance of the spongy tissue. It appears from these considerations, that any inherent tendency (*C.*) to contract present in the fibrous wall would be assisted during inspiration by the quantity ($w - E. R.$). I am aware that the compressing force which I have described is very fractional, and that its importance must be measured by its continuous action. The bronchial orifice which I have supposed to be patent is, however, very frequently occupied with secretion more readily allowing the egress than the entrance of air. In such a case, the deep

inspiration which precedes muscular strain or the effort of coughing is directly utilised against the outer surface of the cavity.

We shall see elsewhere that the atmospheric pressure may be transmitted to cavities by the thoracic walls, but its influence is nowhere more striking than when, gaining access to the pleural cavity, it acts directly upon the external surface of the lung. The collapse of tissue and of cavities induced by internal perforation of the lung, is so striking as to have suggested to some the employment in excavating disease of artificial pneumo-thorax as a possible means of treatment; but of this aspect of the question it is not my province to speak.

Fig. 5.—Forces favouring and opposing Contraction of Cavities (Alveoli should have been depicted in a state of distension). *w.* Atmospheric Pressure. *R.* Resistance (equal to *w.*) offered by Cavity Wall. *E. R.* Elastic Resistance (not equal to *w.*) offered by Alveolar Wall. $w - E. R.$ Balance of Pressure acting from without upon Cavity Wall. *C.* Spontaneous Contractility of Fibrous Tissue of Cavity Wall.

The influence of the compensatory dilatation of the neighbouring tissue, added to the natural contractility of the cavity-wall, may suffice to induce gradual collapse of the smaller cavities, leading to those solid scars, rather larger than a hemp-seed, which occasionally are found at the centre of the lung abruptly terminating an air-tube of good size. The relations of the common cavity situated near the surface are not so favourable to contraction. The pleural adhesions, which are seldom absent, restrict vicarious expansion around it; whilst encroachment from the opposite lung rarely exercises any lateral pressure upon the cavity. Contraction in these cases is rendered possible by two circumstances, which are usually associated: 1. falling-in of the thoracic parietes; 2. shrinking of lung within the thorax.

The alteration induced in the outline of the thorax may be general or local. From a consideration of the architecture of the chest, and of the interdependence of its component parts, it is, at first sight, easier to conceive a general than a local collapse of its walls.

Uniform flattening of one side of the thorax may occur, and is sometimes clearly traceable to excavation. But the local depression limited to the seat of excavation is undoubtedly a more common change. The mechanism of its production is somewhat complicate. If we bear in mind the remarkable power of recoil exhibited by any part of the thorax after the application of temporary pressure, it is evident that the permanent falling-in, to which I have alluded, must have been of very gradual origin. The first impression is made prior to the establishment of excavation. Ribs overlying the inflamed districts become dissociated from the concerted movements of the intercostal muscles by a species of local paralysis, and ultimately they remain motionless. Dr. Ransome, from careful examinations by means of his stethometer (an instrument which deserves to be better known and more extensively used), has proved that the minimum of respiratory excursion is afforded by those parts of the chest which conceal active pulmonary mischief. At this time, the other ribs are actively at work, and become more and more independent of the paralysed ribs, whilst the costo-sternal joint of the latter, having acquired considerable looseness, permits the sternum to follow without hindrance the movements of the healthy

ribs. The preliminary steps which I have described having paved the way for local retrocession, the thorax becomes gradually depressed with the progress of excavation. Exceptions to this rule are sometimes seen in which no marked depression is occasioned even by large cavities. In rare cases, as in the specimen which I place before you, the whole upper lobe may be completely excavated, and so adherent to the chest-wall, that for a long period the chest may present no striking collapse. In this special case, the lower part of the lung was completely adherent and hyperdistended, and the absence of fibrosis argued a more moderate degree of pleural irritation than usually accompanies excavation so superficial and so extensive.

Collapse of the thorax has its final limits in the perfect approximation of the ribs. When contact has occurred between the latter, any further retraction of cavities must entirely depend upon the fibrous shrinking of the lung itself. In fibroid phthisis, this shrinking attains a remarkable degree. At the base, owing to the yielding nature of the diaphragm, a retraction of the lung and a contraction of the cavities are specially favoured; and the most usual result of the contraction of large cavities in this situation is a shortening of the thoracic space, in consequence of which the position of the liver, the stomach, the spleen, and other viscera may suffer appreciable alterations.

Neither the shrinking to which I have alluded, nor the secondary hypertrophy, can occur without some disturbance of the normal relations between the visceral and the parietal surfaces of the pleura; and, conversely, rigid pleural adhesions are detrimental, not only as opposing contraction of the cavities, but as preventing the free expansion of the healthy tissue, and its gradual insinuation between the pleura and the wall of the cavity. In the diagrams and specimens before you, the extent to which the spongy tissue may expand is strikingly shown by the

Fig. 6.—Band-like Adhesions of Pleura, strictly limited to Surface corresponding to Excavation.

length of the band connecting the retracting cavity with the pleura. In these cases, adhesion was strictly limited to the point at which the cavity originally approached the surface. Extensive adhesions around this point would have rendered such a result impossible. It follows that the existence of adhesion over the healthy parts of the lung forms a most important element in our diagnosis. Especially important is the question as to adhesions of the inferior surface. I need not insist upon the value of an unfettered respiratory mobility of the diaphragm. Adhesions cannot fail more or less to lessen the latter; but the anatomical alterations which they introduce into the lung are not less important. It will presently be shown that adhesions to the diaphragm invariably restrict the area of the base, and consequently curtail the breathing surface of the lung.

In compensation for these great disadvantages, the only benefit accruing from pleural adhesions in respect of the contraction of cavities is the growth of fibrous tissue, to which I have incidentally referred. The remarkable thickness sometimes acquired by the pleural membrane, by filling up superfluous space, enables the lung to shrink to a smaller size; or it may ultimately exert a directly constricting pressure. My own views as to the mode of origin of this thickening do not materially differ from those expressed by Dr. Douglas Powell, in his work on *Consumption* (pages 17-19). Pleural adhesions having formed, the respiratory movements of the chest and the shrinking of the excavated lung lead to a stretching of the young fibrous tissue: hence the transverse striation plainly seen after death in the adhesions.

In harmony with this explanation, those parts are usually the seat of the greatest pleural thickening, where falling-in of the chest meets with the greatest difficulty. In the ordinary apex-excavations, the thickest pad of fibrous tissue occurs in the axillary region, coinciding with the greatest convexity of the ribs. Fibrous tissue is not the only substance employed by nature as a stop-gap. Very frequently fat is produced, in appreciable quantities, in the retro-pleural tissue. This I have often

observed over the site of contracting cavities. (See Fig. 7.) The favourite situation is the internal surface of the ribs, as originally pointed out to me by Dr. R. E. Thompson. A contracted lung, removed from the chest in connection with the adherent pleura, usually presents deep parallel grooves, which alternate with prominent ridges,

Fig. 7.—Transverse direction of the Fibres of thickened Pleura covering contracting Cavity. Deposition of Fat between the Ribs and the Pleura. C. Contracting Cavity. P. Thickened and adherent Pleura. F. Deposits of Fat beneath Ribs (R.).

suggesting an analogy with the costal indentations of a pneumonic lung, but in reality presenting the exact counterpart of this condition. In pneumonia, the groove corresponds to the bed of a rib; in the contracted lung, the position of the rib is marked in relief, and that of the intercostal space by a depression. The retraction of the fibrosed lung readily draws inwards the soft parts; but a similar in-bending of the rib is clearly impossible: in this situation the visceral layer of the pleura retreats from the periosteum, and the interval between pleura and bone is filled by fat.

[To be continued.]

REMARKS ON BALD TINEA TONSURANS AND VEGETABLE PARASITES.

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ON several occasions, I have called attention to the occurrence of smooth bald patches in tinea tonsurans, exactly resembling alopecia areata. I have also mentioned the fact that when, in these cases, one patch becomes quite bald, the others quickly follow suit. A remarkable example of this has lately come under my observation. Two brothers have been under my care for some time past with ordinary well marked tinea tonsurans, with abundance of the trichophyton. Their parents objected to vigorous treatment, and therefore mild remedies only have been used, which have not at any time produced the slightest soreness. The elder boy had six or seven separate spots of tinea tonsurans, and, during the last fortnight, every one of these spots, great and small, has become perfectly bald, smooth, and shining, with a well defined margin of healthy hair. What I wish especially to call attention to is, (1) the suddenness of the change, (2) that the bald spots are strictly confined to patches previously affected with tinea tonsurans, not a trace of baldness existing on any other part of the head. In this instance, there is not a stump nor a hair to be found, though, when I previously saw him, the patches were thickly covered with stumps and hairs. In other cases of this kind that I have met with, it has been generally possible to find one or two stumps left either on or at the margin of the patch, by the microscopic examination of which the nature of the case could be determined. When the baldness is as perfect as it is in this instance, we may probably regard it as a spontaneous cure. In the younger boy, the tinea tonsurans has run an ordinary course. It is somewhat difficult to say whether we should regard a case of this kind as one of true alopecia areata following on tinea tonsurans, or as a peculiar and exceptional feature in tinea tonsurans itself. Nor do I think the question of much importance, provided that we remember that infection is not always removed; or, in other words, that the denudation is not in every case so complete as to remove all the stumps affected with the *Trichophyton tonsurans*. A want of knowledge of this fact has, in past times, led to two mistakes: the one, that alopecia areata is a *parasitic* disease; and the other, that it is *occasionally contagious*.