its own definite and well marked pathological processes, but in simulating others, from the stupor of typhus, the collapse of cholera, the high temperature of insolation, the sickness of an irritant poison, to the convulsions of epilepsy or of dentition, which may occur in the pernicious forms. It induces anæmia and general cachexia, with structural changes in the liver, spleen, or other viscera. Neuralgia, asthma, and various other symptoms of disturbed innervation and sanguification; and, as I have before said, appears to be in close etiological relation with dysentery, cholera, diarrhoea, beri-beri, hydrocele, elephantiasis, bronchocele, and hepatic disease.

Whatever its nature may be, the action of malaria on the human economy is very striking; it affects the central nervous system, causing disturbance of vaso-motor action, paroxysms of fever, and congestion of the abdominal viscera, which become periodic in recurrence, or pass on to structural changes in the liver and spleen, or intestinal mucous membrane. No one can have resided long in a malarious climate, like Assam, without observing the cachectic, deteriorated aspect of the people, who, although they may never have had a single attack of fever, and do not feel ill, and would resent being told so, are yet victims to the insidious action of the poison, and present evidences of anæmia, degenerate tissues, and chronic visceral disease. In my next lecture, I shall describe intermittent fever as it occurs in India.

THE GULSTONIAN LECTURES

ON

PULMONARY CAVITIES: THEIR ORIGIN, GROWTH, AND REPAIR.

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LECTURE II. PART II.

THE occasional occurrence of decomposition in the secretion of cavities leads us to inquire into the influences which may be exerted by this cause upon the walls of vomice, and particularly into the existence of any absorptive power special to the latter.

I have already pointed out that fluids may regurgitate from the cavity into the open mouths of the bronchioles, and, gaining access into the alveoli, may diffuse into the blood. This circumstance introduces into the problem a serious complication, which will be discussed hereafter. But for the present, I am specially considering the cavity-wall, independently of the bronchi opening into it.

The extent to which simple percolation may take place is determined by the condition of the internal surface of cavities. During the period of formation of the latter, the fluids act locally upon the decaying structures. Dr. Reginald E. Thompson, in his book on *Pulmonary Hæmorrhage*, alludes to this mode of excavation by maceration, leading to the appearance of a "nibbling". If we exclude the more virulent cases of acute phthisis, where the lung-substance literally dissolves away in its own juices, and the cedematous forms of softening, the soakage to which I have alluded does not extend into the depths of the tissues where the work of repair is progressing, but the dead tissue acts as a temporary shield for the living. On the other hand, in the matured cavities, any extensive diffusion of the fluid contents is opposed by the fibrous layer and by the outer zone of fibroid cell-growth, the special function of which is to add thickness to the cavity-wall. The possibility of any direct absorption in these cavities mainly depends upon the tightness or the looseness of the fibrous tissue around them; all fibrous structures are by nature fitted for some degree of absorption, and even the densest are capable of opening up hidden paths under the agency of pressure. Much pressure does not usually exist in vomice; neither do they contain many of those cells which have a tendency to force their way through lymph-passages. The current of secretion is altogether from the tissue towards the cavity; and circumstances rarely arise in ordinary phthisis which would occasion a reversal of the stream. The case, however, is different in the bronchiectatic sacculations: these are from time to time filled—nay, distended—with their own secretion, and the pressure within them is periodically increased by ineffectual efforts at expectoration. Although the existence of a limiting membrane has been microscopically demonstrated by Grancher and by Dr. D. J. Hamilton, it remains an open question whether, in its stretched and thinned condition, this membrane opposes

an effectual barrier to the more soluble products of decomposition. The clinical evidence appears to justify this doubt; symptoms of putrid infection, such as nausea and diarrhoea, frequently coincide with a period of fulness of the tubes, and disappear with the evacuation of their contents. No such continuous layer exists in the common phthisical cavities. If placed under similar pressure, the meshes of their fibrous coat would probably allow of free filtration. Some filtration actually occurs from simple stagnation in the putrid cavities to which I have referred; and it is significant that, in these cases, the peculiar factor is present after death in the other organs, showing that the whole blood is contaminated. From this dangerous degree of filtration of secretion, the vomice in phthisis are usually protected by the absence of pressure within them, and perhaps in a slight degree by the false membrane due to necrosis, which their inner surface exhibits.

In connection with the subject of absorption, it is interesting to compare with the cavities, relatively well drained by means of patent bronchi, the deposits which remain permanently isolated from contact with the air, and gradually undergo shrinking within the lung-tissue. Microscopical sections usually show around a caseous nodule a clear space or vallum separating the mass from a zone of fibro-nuclear growth. This space is traversed by wandering cells, which may be noticed on their return laden with pigment and granular matter, and which can be followed into the plasmatic spaces. They are sometimes detected at the periphery of the caseous mass, lodged as it were in recesses, which almost seem to be of their own formation. I have never discovered any evidence at the circumference of cavities of a cell-traffic comparable to that which I have just described. Cell-migration, if it occurs, takes place in the opposite direction from the tissue into the cavity.

But whether active or slow, continuous or intermittent, the slight absorption occurring through the walls of cavities has, for its consequence, a progressive thickening of the surrounding lung-tissue, proportionate to the irritation. I readily admit that mechanical factors have a share in promoting this fibrosis; so contractile an organ as the lung cannot, with impunity, be retarded in its movements by the presence within it of patches of consolidation, or by the uneven traction from a thickened or adherent pleura. These mechanical agencies must, however, be considered as secondary in importance to the more subtle influences of chemical irritation.

Formation of simple fibrous tissue is but one result of irritation by absorption. I propose to devote a few remarks to another more important consequence arising from the same cause: I refer to tubercle, and first to that form of tubercle which is observed with great frequency in the immediate vicinity of the cavity-wall. Tubercle in this situation is clearly local and secondary. We are familiar with its exact analogon in the trachea. When, in a case of excavation at the right apex, we find the trachea deeply ulcerated and tubercular at a point of its left wall, situated about one inch above the bifurcation, in a line with the axis of the right bronchus, as shown on the diagram (see fig. 4), we possess a proof of the formation of secondary tubercle under the direct influence of a primary excavation. A wholesale ulceration of the trachea, and even of the larynx, occurring under similar circumstances, is capable of the same explanation. These observations are of extreme importance as establishing the causation of tubercle from the direct application of the contents of a cavity to the healthy mucous surface. If the comparatively resistant structures of the trachea suffer such marked lesions from this form of irritation, how much more decisive must be the effect produced on the tender wall of the alveolus, or on the bronchiole. Thus we are prepared for the recognition of a form of secondary pulmonary tubercle purely irritative in its origin, not necessarily arising in visible contact with the primary disease, but capable of being generated at considerable distances from it, by the impact of air contaminated with the products of disintegration; and it may be incidentally stated that such is the usual origin of the bunchy or racemose tubercle. Another link in this chain of evidence is supplied by the local tuberculosis observed in the walls of bronchiectatic cavities: from cases of this nature, the suspicion of pre-existing tubercle, independent of the cavity, is absent conveniently to our purpose. These local tubercles do not widely differ in aspect from those belonging to a more generalised tuberculosis, which often affects the pulmonary tissue at a later date; and there is, I believe, no escape from the conclusion, that their presence is indicative of the direct causation which I seek to establish.

But let us return to a consideration of the ordinary phthisical cavities. Bordering upon the latter, I have very commonly found large dense masses of tubercle, resembling, by their pigmentation, by their firmness, and by their tendency to fibrosis, the usual racemose deposits. (Fig. 2.) From these, however, they differ both in their size and in their shape; they present a wider expansion, and a cuboidal rather than a spherical outline. The position in which they are found is remarkably constant in

relation to cavities. Their seat of election is the upper axillary border, external to the cavity very usually found in the outer part of the upper lobe; and they extend from the external wall of the cavity to the visceral layer of the pleura. Next in frequency of occurrence is a tubercular mass in the middle third of the axillary region, commonly filling the wedge-shaped summit of the lower lobe. This mass borders upon the cavities which I have shown to be very constant in the dorso-axillary region. Lastly, I have frequently found a smaller mass at the outer apex, as a dependency of a vomica which did not reach the summit of the lung. The reason for the uniformity of these situations will be readily perceived, if we refer to the diagram of the bronchial distribution.

Fig. 2.—Masses of Secondary "Local" Tubercle at Distal Side of Cavities.

A line drawn through the bronchus supplying the upper axillary region will be seen to traverse the cavity situated in the upper lobe. Similarly, the bronchus belonging to the lower axillary region is interrupted by the dorso-axillary excavation, and the same relation subsists at the apex. With the help of these observations, we can readily appreciate the cause of the tubercular masses. The tubercle is a local consequence of the chronic cavity. At its distal aspect, the cavity-wall intercepts a set of nearly parallel bronchioles, through which irritation is conveyed to the terminal air-sacs; and the cuboidal extended shape of the resulting tubercular deposit is a simple consequence of the mode of its origin. The great constancy of the axillary deposit is obviously connected with the active respiratory function of this part of the lung, owing to which, some irritating matter is inevitably drawn to the periphery along the divided bronchioles.

Much more difficult to explain is the sporadic miliary tubercle, very often affecting the lung towards the termination of cases of pulmonary excavation. Two theories have been advanced in connection with this secondary miliary tuberculosis: the one based upon lymphatic conduction, the other resting upon the assumption of a virus circulating within the vessels. It is not my intention to deny the existence of a lymphatic irritation, such as described by Dr. D. J. Hamilton, in his lectures on Catarrhal Pneumonia and Tubercle, in connection with secondary pulmonary tuberculosis. But, however marked an influence conduction along the lymphatics may possess within restricted areas, a mechanical difficulty opposes the view, that the lymphatic stream from a small cavity in the upper lobe could affect the lower lobe; just as, in scattered tubercle, it would be difficult to account for the large intervals of healthy tissue which in reality occur. Against the view of lymphatic conduction, should also be mentioned the frequent immunity enjoyed by the pleura. Dr. Hamilton describes the pleural surfaces as universally adherent in secondary pulmonary tuberculosis. Such is not my experience. In not a few instances, I have found the lungs free from adhesions, although pervaded with tubercle.

The doctrine of the propagation of tubercle through the blood-vessels, has long acquired a firm footing in science. It is supported by the results of experimental inoculations, of intravenous injections, and by the frequent recognition of a tubercular activity in the walls of the vessels themselves. This theory lends itself, better perhaps than any other, to an explanation of generalised tuberculosis. In my opinion, tubercle may originate, and does originate, in vessels; but the germs of the disease need not be contained within their lumen. The vessels may inflame from causes external to them, as well as from those which act internally. In the rare cases, in which a distinct tubercular embolus occludes a vessel, we perceive a sufficient reason for the localisation of the lesion; but, in the great bulk of remaining instances, we are left to seek an explanation for the development of tubercle rather at one spot than at another.

In the lung, the localising agent appears to be contained within the bronchi. Dr. Reginald Thompson, in a paper on the Auto-infection of the Lung as a Cause of Tubercle (*Lancet*, 1878, vol. ii, page 38), dwells upon the influence of inhalation and of gravitation on the production of the disease; and Rindfleisch, in support of his theory of an infection of the lung by its own secretions, describes the mechanism of this infection, and places the seat of the earliest deposit at the spur separating two ultimate bronchioles. Tappeiner's experiments on the artificial inhalation of tubercular matter, in which the dogs submitted to the poisoned atmosphere developed tubercle, afford direct support to the view which has been mentioned.

I confess that the theory of inhalation is inadequate to explain the sporadic nature of the miliary eruption, and its presence in regions which are not distinguished by any great respiratory activity; but yet more improbable would appear the hypothesis, that the tubercular infection, originating in a cavity, should perform the great circuit of the circulation without generating tubercle, finally to settle once more upon the lung. Whichever way we turn, we are driven back to an assumption of predispositions. There exists a general predisposition to tuberculosis peculiar to the race, to the family, and to the individual; and there exists in the economy an organic predisposition, probably varying with age, but which, in the adult, usually has its seat in the lung. The nature of the determining cause still baffles our search in the case of acute miliary tuberculosis; but, in the other varieties, the localisation of the tubercle usually depends upon irritation of a coarser nature, lending to the deposit its characteristic features, situation and shape.

I have attempted briefly to describe the anatomical peculiarities of cavities, and some of their results. I must now pass to a study of their subsequent growth and multiplication.

In the early stages of excavation, the most frequent mode of extension is by simple coalescence of neighbouring vomicae. The intervening tissue, thinned by ulceration, finally gives way at the weakest point, and the resulting communication rapidly widens, until little remains of the original separation. A similar perforating process may affect the interlobar septum, and throw the cavities which it separates into a huge vomica; but commonly the ulceration of the septum is due to the rapid extension of a single cavity, situated above it, into the lower lobe, as yet free from disease.

Two vomicae, situated on the divisions of the same bronchial stem, sometimes coalesce by an ascending ulceration of their respective bronchi. The latter are destroyed as far as the bifurcation of their parent trunk, which then opens directly into the vomica.

In a third variety, a cavity, in the course of its extension, encroaches upon a neighbouring bronchial territory, laying open some of the bronchioles. Respiration, which is comparatively more active in the invaded district, hitherto healthy, will lead to an inhalation into the latter of the contents of the cavity, proportionate to the strength of the inspiratory current. Excavation, usually of a moist and rapid kind, will necessarily ensue, and involve the whole bronchial district in question. I have illustrated, by means of a separate diagram, this important mode of the extension of the disease.

The three varieties to which I have alluded are all peculiar to young cavities still in process of formation. The increase in size of the fully formed vomicae is much more gradual, and generally due to the slow necrosis incessantly at work within them. Occasionally, however, in consequence of the necrosis, outlying caseous masses may be exposed to the action of the air, and a somewhat sudden extension of the main cavity may take place at their expense.

Rindfleisch ascribes the growth of cavities to the influence of atmospheric pressure. His theory cannot be better stated than in his own words: "The cavities therefore continue to enlarge until they reach the pleura, which then usually becomes thickened, and forms their definite limit. The larger they become, so much more exclusively will the inspiratory force destined for that portion of the lung be expended in making them still larger. Like the torn edge of a veil, the remaining parenchyma contracts and is drawn in, partly to the periphery, and partly to the root of the lobe. It serves then to fill up all the corners which are left between the convex surfaces of the cavities, and to restore somewhat the contour of the lung." ("Chronic and Acute Tuberculosis", Von Ziemssen's *Cyclopaedia*, vol. v, page 680, American translation.) I confess my inability to reconcile this account with my own observations. In excavation, we have become acquainted with a gradual process, ill comparable to a laceration. Again, far from retracting towards the root of the lung, the spongy tissue rather tends to crowd around the vomica. It is the cavity itself which retracts, not the alveolar tissue. My criticisms are more especially directed against the supposed distension of the cavities under atmospheric pressure. I disagree with Rindfleisch on the fundamental point of the assumed yielding of the cavity

wall. Whosoever will inspect the limiting membrane of a tuberculo-pneumonic softening, will be readily convinced of the comparative toughness of this membrane, which is capable of resisting a greater force than the slight variations in pressure induced by inspiratory efforts. If with these tough structures the delicate alveolar wall be compared, it will become apparent that the atmospheric pressure would dilate the latter rather than the cavity. Exclusively preoccupied with the elasticity of the spongy tissue, Rindfleisch appears to have lost sight of its remarkable expansibility. As long as alveolar substance remains to fill up the space created by inspiration, the atmospheric pressure cannot influence the cavity. I shall have occasion to revert to this point, and to show that the great disproportion between the resistance of the cavity-wall and that of the alveolus, favours the contraction rather than the dilatation of vomicae.

Cavities in the progress of phthisis tend to increase in number as well as in size. The position of the younger cavities is not entirely the result of chance. The prevalence of cavities in the dorso-axillary region, to which I alluded in my last lecture, points to an element of regularity even in the later development of phthisis, and to the operation of definite causes which we may study with profit. A recognition of the extension of the disease from the apex downwards was among the earliest results of improved methods of investigation. In one sense, this is but a corollary of the proposition prominently asserted by Louis, as to the frequency of apex-phthisis. The position of the secondary mischief, not only beneath, but in the vicinity of, the primary lesion, could not fail to be construed in the sense of extension by continuity; and this impression, if I mistake not, is still uppermost in the minds of many. The existence of a progressive tendency in phthisis it is not in my power to deny; but I venture to oppose the view that inflammation necessarily extends by continuity of structure. Among the achievements of modern medicine, we can point with just pride to the recognition and the furtherance of the curability of phthisis. It is now very generally admitted that pulmonary consumption may sometimes be arrested. We have all witnessed the progress and the recession of pulmonary vomicae. This curability, special to some cases, rests entirely upon the local character of the lesions; in these cases, the disease is obviously not propagated by a creeping process.

The mechanism of the reproduction of cavities from above downwards, especially in the axillary region, will be best understood from a glance at the physiological and anatomical conditions, which so often contain the solution of pathological problems. Just as the affections of the kidney are chiefly connected with their secreting duty, the affections of the liver with its functional activities, so with the lungs, most of their diseases are influenced by their function of aëration. The respiratory energies undoubtedly vary in different parts. This can be proved indirectly by stethometry. Dr. Ransome's tables conclusively show that different excursions are performed by the various points of the surface of the thorax. Collateral proof of the same fact is supplied by the atrophic changes which the lung undergoes in old age, when the requirements fall short of the available breathing space. The atrophy affects definite regions. Hourmann and Dechambre* have shown that the middle lobe is relatively more wasted than other parts. This observation points to a comparative loss of function in this region. In the healthy adult, differences can usually be detected in the regional activities of the lung. If the respiratory capabilities of the various parts of the chest be tested by the combined methods of auscultation and of surface-measurement, we arrive at the following results. Respiration is most active in the lower lobe, least active at the apex, and very active in the axilla; whilst it gradually diminishes towards the sternal border. The importance attaching to these relative respiratory values will become evident, when I have pointed out the anatomical peculiarities of the bronchial distribution (Fig. 3). You will observe, from an inspection of the diagram, that on each side the main bronchus divides essentially into two branches; the upper (A) being destined for the apex; the lower (B), which is, more strictly speaking, a continuation of the main bronchus, supplying its divisions to the base. In any careful dissection, horizontal branches will be seen to traverse the back of the lung from the root to the axilla. In each lung, two of these are present. I will call them, for convenience, the *upper and the lower posterior-horizontal bronchi*. The anatomical differences between the lungs occasion a corresponding difference in the origin and distribution of these tubes. The right upper lobe, possessing posteriorly a greater height than the left, includes the upper posterior-horizontal bronchus, which takes a direction parallel to the septum and immediately above it; whilst the lower posterior-horizontal branch occupies a similar position in the lower lobe. In the

left lung, these branches both belong to the lower lobe, which rises considerably higher at the back than the right lower lobe. The upper branch is by far the more important on this side; but the relative size of the two branches is liable to vary. The origin of the upper posterior-horizontal bronchus differs widely on the two sides. Whereas on the right this bronchus is a branch of the bronchial stem A, on the left it usually opens immediately beneath A into the main bronchus continued. I have witnessed its occasional derivation from A, but would consider this an abnormality. The lower posterior-horizontal bronchi present no differences; on both sides, they originate in the posterior wall of B, rather more than one inch below the primary division, being the first tube given off by B, with the exception of the bronchus (L. S.) destined for the lower sternal region, which has its orifice in the anterior wall.

Fig. 3.—Origin and Distribution of Upper and of Lower *Posterior-horizontal* Bronchi (other branches not accurately given). A. Bronchus to the Apex. B. Bronchus to the Base. U.P.H. Upper Posterior-Horizontal. L.P.H. Lower Posterior-Horizontal Bronchus. L.S. Lower Sternal Bronchus.

I would call your attention specially to the close relation existing between the upper branches and the bronchial supply to the upper lobe, and also to their rigidly patent circular orifice—a peculiarity which they share with the bronchial tubes of the apex. All branches originating below this point present a somewhat flattened oblique orifice, the patency of which is probably subject to variations. The posterior wall of the bronchus from which the lower branch arises is liable, owing to its position, to be clogged by viscous secretion during the hours of sleep, when sensitiveness of the membrane is lessened; and mucus would the more readily cling to its surface on account of its fine rugæ. The implication of the lower branches is, however, an event subsequent to the excavation in the distribution of the upper divisions, which should be considered first.

In cases of excavation at the apex, overaction and ultimate distension of the axillary region, result in proportion to the loss of respiratory power at the summit; thus the velocity of the air-current destined for the axilla will be rather in excess of the velocity in the apex-bronchus. Dr. Reginald E. Thompson would even assume that the ventilation of the apex is mainly dependent upon the stronger current which blows from the lower air-tubes over the aperture of the bronchus distributed to the apex. This point, however, is not material to my argument.

Owing to the close bronchial relations subsisting between the apex and the axilla, the enfeebled current through the tubes of the apex, and the exaggerated traffic through the axillary branches, inevitably lead to axillary excavation. (See Fig. 4.) The secretions, imperfectly propelled by the deficient expiratory effort of the diseased apex through the main upper bronchial division, are in danger of being sucked, by the deep inspiration which succeeds cough, into the neighbouring bronchus, which supplies the axilla. That such a result really follows is rendered highly probable by the shape of the early consolidation, by its microscopical character, and by its subsequent history. The early deposits are quite distinct from ordinary catarrhal pneumonia. They do not involve a whole lobule forming a polygonal patch of tolerably even consolidation, but distinctly occupy a bronchiole and two or three of its terminal divisions; hence the trefoil appearance of the nodules, often described. In strict harmony with this view of their origin, nodules occur in groups in the district of a bronchial division of mode-

* "Recherches cliniques pour servir à l'histoire des maladies des vieillards; Maladies des organes de la respiration"—*Archives Générales de Médecine*, ii, t. 8, p. 417.

rate size, which is often thickened in the vicinity of the deposit for a short distance, but higher up is practically free from disease. The nodules present a remarkable combination of very perishable catarrhal cells, with a well defined fibroid cell-growth and with giant-cells; in fact, they unite the characters of a tubercular with those of a catarrhal inflammation, and are consequently liable to a moister and more rapid softening than is possible in pure tubercle.

Fig. 4.—Mode of Origin of Secondary Cavities in *Dorso-axillary* Region. Local Tubercle as in Fig. 2. Local Tubercular Ulceration of Trachea. P. Primary, S. Secondary Cavity. T. Tubercle. U. Tracheal Ulcers.

In the axillary districts thus infiltrated by deposits and deprived of expiratory vigour, excavation is brought about in the following manner: a small bronchus, under the irritation of the residues of imperfectly expelled secretion, becomes the centre of considerable congestion; ultimately ulcerates, and opens up the chief bronchus of the district at a point above the nodular masses. From this point, the softening rapidly extends to the mass itself, which is destroyed by maceration rather than by true caseation.

Such is the history of the common dorso-axillary cavity. The cavity is subject to variations as to its exact position; it often originates in the mid-dorsal region, or it may begin at the side and subsequently extend into the back. These differences are mainly determined by the course taken by the inhaled particles into one or into the other bronchiole. Cavities in this situation are commonly destitute of trabeculae, probably as a result of the early undermining of the chief bronchus of the district, which I have attempted to describe.

The extent of the irritation set up by insufflation of the secretion of cavities depends, as I have elsewhere stated, upon the regional activities of respiration. The axillary district, which is remarkable for its respiratory energies, is also distinguished by the frequency within it of secondary changes. Secondary tubercle in this region invariably occurs on the distal side of the cavity, that is to say, rather to the front than to the back of the axilla, and if a transverse section be carried through the lung parallel to the front of the chest, it will divide the tubercle in the angle of the lower lobe situated between the septum and the axillary border. The mass of tubercle encountered by the section is often but the zone of irritation of a cavity which a deeper section will bring into light. In proportion as the cavity extends and intercepts fresh bronchi, the tubercular masses progress towards the sternum, and, if time be allowed, the greater part of the outer portion of the lower lobe may become infiltrated by buncy tubercular deposits.

I have attempted to explain, on the data of physiology, the common mode of extension of the phthisical process to the mid region of the lung, and have clearly shown, I hope, that in the majority of cases phthisis is not due to gradual creeping of inflammation, but to the following sequence: 1. Nodular consolidations from the irritation of inhaled particles; 2. Loss of respiratory activity and diminished expiratory current in the corresponding bronchioles, whilst the original infecting influence continues unabated; 3. Acute irritation of the small tubes from stagnation, ending in their ulceration; 4. Deposition of secondary tubercle on the distal side of the cavities thus formed.

The disastrous agency of insufflation of morbid secretions from vomicae is not limited to the production of a dorso-axillary cavity. Some of the lesions belonging to the latest stages of phthisis are under the same influence. With the progress of disease, the increasing respiratory distress renders the unaffected regions more and more liable to inhalation of secretion. During the attacks of dyspnoea which mark the last days of the unfortunate patient, the expiratory efforts of the diaphragm produce an appreciable current towards the sternal and basic

fringes, and abundant secretion is drawn and impacted into the distended alveoli. The resulting pneumonic masses present well marked characters: as large as a pea or as an almond, they stand out boldly in the midst of the hyperinflated tissue, and they are remarkably deficient in colour, probably owing to the anæmia induced in the pulmonary alveoli from distension. In these respects, they present a striking contrast with the deposits due to more chronic irritation. The softening of these masses, if death should be sufficiently delayed, and if oedema should supervene, proceeds with great rapidity.

The lesions which I have last described occur so late in the disease, that their recognition is seldom possible, nor is this of any special importance. Of vital moment is, on the contrary, the discovery of a cavity in the axillary region.

The absence of this lesion in any case of phthisis adds greatly to the chances of the patient; its existence in cases where youth or a naturally strong constitution does not establish a redeeming influence, is almost equivalent to a warrant of death.

ABSTRACT OF LECTURES ON THE MORPHOLOGY OF THE MAMMALIAN SKULL.

Delivered at the Royal College of Surgeons of England.

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LECTURE VI.—THE SENSE-CAPSULES OF THE VERTEBRATA.

THERE are, as a rule, three pairs of sense-capsules in the vertebrata: the olfactory, the optic, and the auditory. Only the first and second of these need be treated of, as the eyeball, although it becomes cartilaginous in all types but marsipobranchs and mammals, yet only affects the form of the skull by pressing against the sides, and so gaining a lodgement in the orbit. But the other capsules form most intimate connections with the skull.

One of the most remarkable forms of nasal capsule is seen in the lowest types of fishes—viz., the myxinoids—in which, as well as in the lampreys, there is only *one*. This single capsule is a tubular structure very similar to the trachea of a mammal, being composed of cartilaginous rings, which are imperfect on their deep face for a small extent. These rings, of which, both in Myxine and Bdellostoma, there are about twelve, are only imperfectly separate from each other in some parts; the two hindermost are a considerable distance apart, the tract between them being enclosed partially by about nine narrow parallel bands of cartilage, the lowermost of which is the broadest. These form a nearly perfect capsule for the proper olfactory region, which bulges so as to be wider than any part of the membrano-cranium behind it. In Bdellostoma, the foremost rings are separated and united by three parallel bands; in Myxine, the foremost ring sends forwards a rostral process. There are in both types two closely approximated external openings at the anterior end of the head, and there is a partial septum separating the two nasal passages in front. In the region of the capsule proper, a fold of membrane passes downwards from each parallel cartilaginous bar; these nine Schneiderian folds are strictly comparable to the folds of the nasal sac in other fishes. The nasal cavity does not end here, but passes backwards under the skull, and opens into the pharynx beneath, in front of the velum.

In the lamprey, the single nasal sac at first appears under the head; but, by the rapid growth of the upper lip, it is carried to the dorsal surface, and its short tubular passage opens by a single aperture. In the ammocoete, the capsule is simply a narrowish transverse band, imperfect below, similar to one of the bands in Myxine. After transformation, however, this narrow band is developed into a bilobed pouch of soft cartilage, the two sides of which do not quite meet below; within these are about eighteen (in *Petromyzon fluviatilis*) Schneiderian folds, converging to a common centre. As in Myxine, a tube passes backwards from the capsule; but, instead of opening into the pharynx, it ends blindly below the junction of the cranial and spinal regions. The hinder part of the capsule grows downwards to form a partial septum between the olfactory bulbs.

In cartilaginous fishes the olfactory capsules are paired, and form dome-like cartilaginous structures. In adult sharks, they are united by their upper edges; but not in the young shark, or adult skate. The