THE GULSTONIAN LECTURES

PULMONARY CAVITIES: THEIR ORIGIN, GROWTH, AND REPAIR.

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LECTURE I.—PART I.

MR. PRESIDENT, FELLOWS, AND GENTLEMEN,—The study of disease with the help of anatomy was the special object contemplated by the founder of these lectures, of whom we read that he was yet greater as a theologian than as a man of science. The well known words of his bequest I need not repeat to you; they contain evidence of his scientific insight, and of his belief in the great future reserved for pathological anatomy; they prove also that his was not a theology opposed to the progress of science, but capable of rising above the prejudice of the hour in the practical search for truth. After two centuries, his objects in science are still our objects; and his enlightened theology may be set as an example singularly fitted for our own times.

This brief allusion to Dr. Goulston's original purpose will be my apology for inviting you to a study mainly anatomical and pathological. The pathology of pulmonary cavities has not hitherto been carried beyond an elementary stage; in the large works on phthisis it is treated in general terms, and almost exclusively from a clinical standpoint; and I have sought in vain for any important monograph on this subject. I shall have occasion, however, to refer to some valuable Lectures on Pulmonary Excavation delivered by my colleagues Dr. C. T. Williams (in 1873), Dr. Douglas Powell (in 1877), and Dr. Mitchell Bruce (in 1881). Some attempt at a systematic treatment of this subject is also made by Professor Rindfleisch, in his article on Tuberculosis in von Ziemssen's *Cyclopadia*; and to the views of this observer I shall specially revert in the course of my lectures. This gap in a somewhat important branch of pathology, which it is my humble ambition to point out rather than to fill, has an explanation in the method usually adopted in the study of phthisis.

The word phthisis has too often been understood to apply to a single affection, rather than to a collection of varying types of disease. I doubt whether the attempt to bind together all forms of pulmonary consumption by the thread of tubercle, so readily traceable in most, if not in all, has assisted the advance of our knowledge. Our notions of tubercle have not gained in definiteness, and we are compelled to admit that the word phthisis has ceased to be free from ambiguity. Since the late Debate on Consumption at Glasgow, we have become conscious of widely divergent meanings attached to this term. "Phthisis", it cannot be too clearly understood, is but a name covering many conditions, in which destruction of lung-tissue is a common feature. Used in this local sense, the term is convenient. But it possesses no place in a strictly pathological nomenclature of disease; and every attempt to study phthisis without due regard to its morphological varieties must end in failure.

The analytical method, which I believe to be indispensable in any investigation on phthisis, is equally required for a fruitful study of the destructive changes in the lung. Too little attention has hitherto been paid to the topography and to the anatomical relations of cavities. The lung has been regarded as an unit, and its excavation as a more or less constant quantity. I will not deny that the most persevering analysis has been carried into the depths of the pulmonary tissue; but too often the tube of the microscope appears to have obstructed the view of the coarser anatomy. My endeavour in these lectures will be to study the pulmonary lesions rather in the light which strikes the naked eye, and to attempt as it were their geography, appealing to the microscope in all matters of detail, but relying mainly on the practical data furnished by the unaided senses.

The tendency to a formation of permanent air-containing cavities is peculiar to the lung, and is obviously connected with the special structure and the ventilating function of the organ. The photograph which I place before you illustrates the production of a cavity from a gunshot-wound. The patient was shot seven years ago through the middle of the left clavicle, and bled profusely from some large vessel. The lung, which was also wounded, became the seat of an aerial fistula and of apex-

excavation. Although of healthy descent, and originally strong, the patient is following the usual declivity of phthisis, showing how much depends upon the position of the cavity, and within given limits how little upon its exact causation. In this case, at least, tubercle does not bear the original blame.

More usually, excavation has its cause in disease. Bronchiectasis and emphysema, anatomically considered, may be said to occupy the border-land between physiological and pathological conditions—both being essentially the outcome of the dilatation of normal structures. The size of the air-spaces in emphysema is often so considerable, and they imply so much destruction of lung-tissue, that they virtually constitute cavities, although we prefer to term them bullæ, having regard to the thinness and to the elasticity of their walls. The latter features, together with their position along the fringes and their lung-like aspect, sufficiently warn us against any confusion with cavities of a different origin. However, if the bulla should become surrounded by a thickened pleura, and if obliteration of its bronchus should take place, and the gaseous contents be exchanged for fluid, a cyst-like cavity may result, which differs from the more habitual vomicae, mainly in the limpidity of its contents and in the polish of its wall. Lebert, who describes in the lung the occurrence of serous cysts, refers them exclusively to ancient deposits of blood or of pus. Hydatids, which are not unfrequently met with, and the very rare dermoid cysts, of which isolated examples have been recorded by Lebert, Wunderlich, Büchner, Collenberg, etc., also belong to the category of cystic cavities.

More common than the cavities which I have enumerated are those due to bronchiectasis, a disease at first not strictly speaking excavating, but fatally tending to the formation of vomicae not distinguishable from those of phthisis. Bearing in mind this tendency, I have preserved, in my tables of pulmonary cavities, a few instances of this affection. But bronchiectasis is too wide a subject to receive in these lectures the discursive treatment it deserves. Abscess followed by cavity-formation may result from a variety of causes, which I will not attempt to enumerate. Sarcomatous and cancerous deposits occasionally give rise to ulcerative excavation in the lung. In gangrene, the most rapidly destructive of our excavating diseases, the stage of excavation is commonly temporary; it is soon followed by a fatal termination, or by closure. When death occurs early, the cavity either presents the character of a putrid mortification, or possesses a secreting pyogenic membrane, which indicates a limitation of the necrotic process, as well as an effort at repair.

I have briefly enumerated the chief kinds of pulmonary excavation occurring from various causes; but the typical cavern which will claim the greater share of our attention is special to phthisis. It bears the impress of chronic inflammation, strikingly evidenced in the zone of condensed tissue by which it is surrounded, and in the pseudo-capsule which defines its outline. This is, however, but one variety of phthisical cavity—not, we must admit, the most common. It differs widely from the excavations of acute phthisis, and scarcely less does it differ from the ordinary pneumonic softening. We are thus made conscious from the onset of the necessity for a preliminary classification of varieties of phthisis if we would thoroughly appreciate the origin of excavation.

The great division between pneumonic and tubercular disease has not yet, to my belief, been superseded. In spite of the tendency of recent years to see in all phthisis the working of tubercle, a distinction is still upheld by some of the greatest authorities, who refuse to subscribe to the revived doctrine of Laënnec that caseation is invariably due to a tubercular origin.

1.—Of these two large groups, let us first take the pneumonic, which, clinically speaking, probably comprises the majority of phthisical cases. Its chief subclasses are: (1) alveolar catarrh and bronchopneumonia; (2) catarrhal pneumonia; (3) caseous pneumonia. A glance at the annexed diagrams will show a general agreement between this classification and those of Buhl and of Virchow, the differences being mainly in the terms.

Classification according to Buhl.

Superficial inflammation (in territory of pulmonary artery).	Fibrinous pneumonia. Catarrhal pneumonia following upon catarrhal bronchitis.
Deep inflammation in territory of bronchial artery.	Peribronchitis. Parenchymatous or Desquamative pneumonia.
	1. Consecutive desquamative pneumonia (induced by general diseases). 2. Genuine desquamative pneumonia. 3. Caseous pneumonia.

Classification according to Virchow.

1. Genuine fibrinous pneumonia.
2. Catarrhal pneumonia (subdivision, bronchopneumonia).
3. Caseous pneumonia (catarrhal and fibrinous pneumonia combined).

I have purposely reduced the pneumonic group of phthisical affect-

tions to its simplest factors—alveolar catarrh, catarrhal pneumonia, and caseous pneumonia; not alluding for the present to acute purulent phthisis, to oedematous softening, nor to the important group specially connected with pulmonary hæmorrhage.

1. The affections included in my first sub-class do not require any special comment. They are distinguished from all others by the moderate extent of the inflammation, which usually terminates in recovery; and by the small proportion which the epithelial elements bear to the other cells filling the alveolus.

2. A radical difference, more obvious under the microscope than to the naked eye, exists between catarrhal pneumonia and the former group. Structurally, this is scarcely less marked than the difference between a transient cuticular desquamation and a dermatitis; the former implicating only the epithelial surface; the latter striking its worst blow at the vascular layers, wherefrom the reproduction of healthy epithelium depends. This peculiarity renders catarrhal pneumonia, in a local sense, insusceptible of recovery, and implies softening as its probable termination.

3. The separation of caseous pneumonia into a definite class appears to be thoroughly warranted, not only by its anatomy, but by its clinical bearings; and I am fortified in this view by the example of Virchow and of Buhl. The difference between catarrhal and caseous pneumonia is not alone one of intensity, but of kind. Whereas the morbid deposit in catarrhal pneumonia nearly exclusively consists of alveolar cells and of leucocytes, in caseous pneumonia it is largely made up of fibrin. In addition to this partly fibrinous character of the alveolar contents, it is common for fibrinous pneumonia to supervene independently upon the chronic inflammation; and the extent of the ultimate disorganisation will greatly depend upon the behaviour of this recent fibrin, which is sometimes reabsorbed, but more commonly is involved in the caseous destruction.

II.—Perhaps the most strictly defined of all varieties of phthisis is the tubercular. I am not alluding to acute primary miliary tuberculosis; from an anatomical point of view, this is not phthisis, and clinically it also occupies an independent position. I would rather refer to a special class, in which tubercle of tolerably pure type, occurring in groups, is present from an early date, and leads to the common symptoms and termination of phthisis. The arrangement of this grouped tubercle resembles roughly that of grapes hanging from a branch, and explains the term “racemose” and “bunchy”, under which it is variously described. Whereas in catarrhal and caseous pneumonia the alveolar structures are primarily involved, in this variety the alveolus is rather encroached upon than originally affected. The bulk of the lung is usually free from disease, and its tissue of good spongy quality. The chest preserves a large size, seldom revealing to auscultation the extent of the mischief; the cavities for a long time are capable of slow development only, and often escape detection. With these anatomical features, the etiology of the affection is in full harmony. The disease is very apt to attack those habitually exposed to irritating atmospheres, and the subjects of chronic or recurrent bronchial disorders.

III.—By far the largest number of fatal cases are attributable to a *mixed* type of disease, including the pneumonic element by the side of the tubercular. I cannot enter here on the arguments which render it probable that catarrhal pneumonia is the prime factor in the majority of these instances, and that tubercle is superadded as an outcome of the phthisis. According to the proportion and to the distribution of the two elements, varieties ensue, both in respect of the clinical phenomena and of the anatomical appearances, too numerous for description.

Holding fast by this rough nomenclature, we shall be better able to discuss the mode of origin of cavities. To whatever form phthisis may be due, excavation is invariably preceded by *softening*. The softening in phthisis is purely a chemical action, over which the living tissue has no control; but, like other chemical processes, it is mainly influenced by the “conditions of the experiment”. Our endeavour should be to become more familiar with these circumstantial factors, whereby the course and duration of the process are so variously determined.

The chemical nature of the decay is well shown by the selective action which it exercises upon the tissues. In ordinary catarrhal pneumonic softening, the last survivors are the elastic fibres. Experimentally, they are also found to be the most indifferent to a contact with acids or with alkalis. Here the ordinary methods of oxidation and the methods of softening are in perfect accord. From this ordinary type, the putrefactive disorganisation of gangrene or of bronchiectasis is entirely distinct. In the latter diseases, cavities have been shown by Filehne to contain in solution a substance which dissolves elastic tissue and coagulated albumen, but is inert with regard to the glutin-forming substance of the connective tissues.

Among the circumstances which most modify the march of phthisical

destruction, should be mentioned the varying density of the deposit and the degree of moisture within and around it. The term *caseation*, which has been rather loosely used in connection with a variety of changes, should be restricted to the dry and dense products from which the moistening influence of the atmosphere and blood-irrigation are in a great measure excluded. In this state of dryness, caseous accumulations may long continue unaltered; but with the admission of air the caseum ripens, and the degenerative reactions ensue which within recent years have been chemically studied by Duclaux (*Comptes Rendus*, vol. lxxxv, page 1171, 1877).

We should not, however, imagine that this complete absence of moisture is an early feature of caseous infiltration. The only morphological peculiarity of caseation, which consists in its extremely fine subdivision, is susceptible of being explained on a totally opposite assumption. Alveoli, in the stage of fully developed catarrhal pneumonia, are almost exclusively occupied by spherical cells, which we will admit to be the outcome of alveolar proliferation. I am aware that the epithelial nature of these cells has been denied by many; and that Senftleben professes to have settled all doubts on this point by the artificial production of the same appearance in pieces of lung, hardened in alcohol prior to their insertion into the peritoneal cavity of rabbits; but, with Buhl and with Rindfleisch, I still hold by the alveolar origin of catarrhal products; and the diagram which I have copied from a drawing by Dr. D. J. Hamilton will show that the same view is taken by that distinguished observer. The differences in shape and general aspect between the swollen epithelial elements and the normal epithelium are very striking; most striking, however, are the differences in their relative size. The enormous development which these cells undergo is plainly due to imbibition, and presupposes abundant moisture, probably arising from collateral oedema. As they expand, the cell-walls become thinner; the contained pigment-particles more scattered; and the minute granulation which results in the swollen cellular substance may be compared to the fine chemical precipitates obtainable in diluted solutions. The swelling which I have described, whilst it fills the alveolus, tends ultimately to burst the individual cells, and to weld them into an homogeneous mass, in which the scattered pigment is the only surviving witness to earlier conditions. This explanation of the compactness of the deposit as due to a swelling from endosmosis appears to me more natural than the suggestion made by Dr. Hamilton, that the healthy lung, acting by its respiratory efforts upon the caseous material after the fashion of a cheese-press, expels from it the fluid constituents; and it supplements the statement made by Rindfleisch, that a spontaneous contraction takes place in the alveolar walls after effusion has occurred within them.

In pure caseous pneumonia, the subsequent desiccative changes are carried out to perfection, owing to the complete stoppage of the supply of air and of blood; and the advent of excavation may be long delayed. Not so in many cases of catarrhal pneumonia of a low type, in which imperfect aëration persists side by side with an inveterate oedema. Here, molecular breaking down occurs early, and partakes less of the nature of fatty degeneration than of a maceration, which is alike destructive of the deposit and of the tissue. Thus, contrary to the opinion expressed by Dr. Hamilton, I consider oedema capable of exercising a most dangerous influence on the course of epithelial inflammations. The combination of an extensive catarrhal pneumonia occurring in separate foci, with severe oedema, commonly leads to what may be roughly termed a “dissecting excavation” of the lung. If the process be tolerably acute, and the softening rapid, the fibro-elastic framework of the lung is completely emptied of its parenchyma, and the lobular septa alone remain.

This condition of excessive moisture need not result from low oedema; it sometimes is produced by a localised congestion. To this class probably belong many of the apex-excavations. The apex-catarrh, which runs a rapid course and breaks down under our observation without passing through a stage of complete consolidation, furnishes us with daily recurring instances of this mode of formation of cavities, sometimes less disastrous than the rapidity of the softening might lead us to suspect. All observers agree in admitting that, *ceteris paribus*, the quicker and the more complete the softening, the more favourable, also, will be the chances of recovery.

It is especially in these moister, least caseous, forms of catarrhal destruction that *elastic tissue* is most abundantly ejected. Even true caseation does not completely destroy elastic fibres; I have found them repeatedly in ancient putrescent deposits, where all other elements were reduced to granular debris. The elastic structures, however, do not wholly escape the destructive influence of caseation; occasionally they are so much comminuted (probably as a result of the degeneration rather than of the original inflammation) that their recognition may become a matter of difficulty. Grancher, in his valuable article

in the *Revue de Physiologie*, ascribes their fragmentation to a mechanical, rather than to a chemical, agency: the strength of elastic tissue lies in its suppleness; if stretched unfairly, and embedded in a hard and brittle mass, it loses its resisting power together with its pliancy. This fact is demonstrated by Grancher by means of injections of the lung with paraffin. The toughness natural to the pulmonary tissue is, under these circumstances, completely abolished, and the lung becomes capable of fracture. Exactly similar conditions are present in caseous pneumonia, which is characterised by the stretching of the alveolar wall, and by the density of the infiltration; and under the microscope, it is difficult to find in advanced caseation the markings of the alveoli, although the larger elastic tracts remain conspicuous.

These remarks will explain the true significance of a discovery of elastic fibres in the sputum; if present, they constitute proof positive of softening, but their absence does not furnish us with any negative evidence. With regard to fully formed cavities, I may anticipate further details, by stating that we do not habitually find any elastic sediments among their contents.

I propose, after these general observations, to enter upon a short description of the modes of excavation special to the leading varieties of phthisis.

Pulmonary cavities most commonly owe their existence to some form of *catarrhal pneumonia*. The ordinary type of the excavation is lobular. Even in larger cavities, this lobular origin remains for a long time evidenced by the persistence of trabeculae. The softening, which begins at a bronchial termination, gradually proceeds through the consolidated lobule as far as its fibrous boundaries, or it may even extend beyond the latter. But, sooner or later, the excavating process is interrupted by tissue-reaction, and a barrier is formed which resists the further encroachment of the disease. This reactive inflammation is probably due to the irritation set up within the alveolar wall by the pressure of foreign material, and especially of the products of disintegration.

Sections of this zone of irritation exhibit, under the microscope, the appearance of a sarcomatous or of a granulating tissue. Sprouting into the alveolar space, the new growth ultimately obliterates the air-cells, the epithelium of which may, for a time, assume undue activity and a cuboid shape; but all alveolar character is finally lost, except in a few air-sacs which had suffered collapse before they were reached by the infiltration. By degrees, the young connective tissue cells elongate into fibres, or become flattened into laminae, and thus a fibrous zone may be formed, which, for a time, delays the forward march of the softening. Active lymphatic functions also belong to this layer; for, in its fissures, streaks of a finely granular substance, mixed with pigment-particles, indicate the high road followed by absorption. A similar process takes place at the periphery of caseous nodular deposits, and of inflamed bronchioles.

Thus fibrosis, or its first stage, fibroid cell-growth, is inseparable from catarrhal softening; but it does not in every case lead to the formation of a distinct capsule. In a special class of cases, a membrane is developed at the periphery of the deposit prior to excavation. I believe these clean-cut cavities, isolated from healthy lung-tissue by a simple membrane, to be usually the outcome of slow caseation (it will be hereafter shown that they may also originate as a late result of hæmoptysis). Should the bronchus of the district affected be rendered impervious, the degenerative process may ensue without any excavation. Increasing inspissation takes place in the deposit, owing to the gradual absorption of fluids, and to the contraction of the membrane; and the latter becomes thicker and more fibrous, ultimately resembling a stout ring of hyaline cartilage. Such is the history of many an old broncho-pneumonia, which has never culminated in open excavation. The tissue has been destroyed by caseation, but has never been expectorated; and its remains exhibit under the microscope the *debris* of cells and abundant elastic fibres.

This occlusion of the bronchus need not, however, be permanent. From a variety of causes, the slender barrier separating the caseum from the atmosphere may ultimately break down. Rindfleisch ingeniously attributes the laceration to the force of the inspiratory current; but it is referred by Dr. Douglas Powell rather to the expiratory cough-pressure. This is an important, because a very common, mode of excavation. The softening may have progressed slowly, but the cavity is formed *ex abrupto*, the physical signs of an empty space becoming suddenly developed, after the expectoration of the caseous fluid. The rapidity of the process is one of the more favourable features in these cases.

When the healthy tissue-reaction (to which is due the formation of the pseudo-capsules of cavities) is absent or imperfect, as in cases presenting less consolidation than oedema, the invariable result is a wide-

spreading destruction. The same consequence follows even in the presence of moderately firm consolidation, should the oedema happen to be great; of this, we possess an instance in the catarrhal pneumonia of drunkards. But, more commonly, diffuse softening results from a variety of pneumonia, which has never reached consolidation, but remains in a state of congestive oedema. This condition, it will be observed, is but one step removed from alveolar catarrh. In the latter, the slight accumulation of cells within the alveoli, and the moderate oedema of the alveolar wall, may completely disappear under treatment. But, if the oedema should become severe and inveterate, the power of epithelial proliferation, and of vascular reaction, is completely extinguished; and the obliteration of a vessel of moderate size will suffice to establish a progressive softening, for which there is no longer any check.

In *caseous pneumonia* the cavities are influenced by the special character of the inflammation. The consolidation, which I have elsewhere described, rivals in extent that of fibrinous pneumonia, whilst in density it greatly exceeds the latter. As in fibrinous pneumonia, so here, the air-cells are not only filled, but distended, with the products of inflammation. Whereas, however, in fibrinous pneumonia, exudation invades suddenly a tissue structurally healthy, in caseous pneumonia the onset is gradual; numerous lobules have been for varying periods in a state of catarrhal pneumonia, and the intervening tissue deeply altered by chronic irritation, when the finishing stroke is given by the effusion of fibrin into the diseased districts. Our diagnosis between the two affections, when the consolidation occurs at the base, will, therefore, mainly rest upon the history of the case; but its correctness can only be tested by subsequent events. In common fibrinous pneumonia, the exudation, which consists of fibrin, of leucocytes, and of red blood-corpuscles, all readily liquefiable, clears up early and rapidly. The result is widely different in caseous pneumonia. The fibrin may be not inferior in quality, but it is mixed with cells of epithelioid type, the fatty degeneration of which is naturally slow, whilst the channels of absorption are blocked by cell-growth. Yet greater differences are found in the vascular conditions. The circulation in fibrinous pneumonia is, for a while, impaired by lateral pressure on the capillaries; but, in caseous pneumonia, the stoppage is permanent, and due to endothelial growth. This peculiarity is sufficiently well marked to establish a safe distinction between caseous disease, and not only fibrinous pneumonia, but also the ordinary forms of catarrhal inflammation. The obliteration is not restricted to the capillaries, but attacks the larger vascular branches, cutting off all blood-supply to the districts involved. This blocking of the blood-vessels, which has been described by Friedländer, by Grancher, by Hamilton, and others, is well illustrated in the diagram which I submit to you. I cannot agree with the view, that the softening in caseous pneumonia is determined by this obliterative arteritis. I hold that the latter is but an ascending change; that it is altogether secondary to the arrest of the circulation within the capillaries; and that it does not, therefore, possess the ordinary value of an arterial thrombosis.

Equally distinctive of caseous pneumonia is the obliteration of the bronchial tubes, a feature less important anatomically than as a valuable guide to a diagnosis between the two forms of basic pneumonia. Under the influence of the same deep-seated irritation which leads to obliteration of the blood-vessels, the bronchi become permanently plugged by their own secretion and by the irritative proliferation of their epithelium. The practical result of the plugging is a complete stoppage of the air-traffic in the diseased region, and a loss of all auscultatory sounds. Instead of yielding bronchial respiration as in fibrinous consolidation, the lung is silent, whilst giving a percussion-note of absolute dullness. From this combination arises, even for the trained observer, a danger of mistaking caseous pneumonia for a pleurisy. The only physical sign establishing a distinction between these two conditions is the increase of vocal resonance in pneumonia, and its diminution in pleuritic effusion; but if the pneumonia be accompanied with thickening and oedema of the fibrous layers, the physician is deprived of this last diagnostic help. Hérard and Cornil relate cases in which the presence of pneumonia was veiled in the manner I have described; and a striking instance of the confusion to which I have alluded has come under my own notice.

I need not insist at length upon the mechanism of the process of excavation, upon the combination of liquefaction with that "dry crumbling" so characteristic of the disease. From the moment that softening has gained access to it, the entire mass is at the mercy of purely chemical forces. Undermined in various directions, large blocks of dense tissue are loosened and cast into the cavity, where they suffer ultimate disintegration. The lung-tissue is destroyed, as it were, in its integrity; neither interlobular septa, elastic fibres, nor bronchi offer any lasting resistance to the softening; alone, the largest blood-vessels

survive, thanks to their *vasa vasorum*, and to the great thickening of their coats; whilst the diseased tissue falls away from them, as it does from the thickened pleura, much as a ripe fruit is detached from the branch. Thus dissected by the softening, the vessels may long remain sole occupants of the vomica.

From this brief description, it would be easy to recognise the typical case of pneumonic excavation. The main features of these cavities are their size, which is often considerable, the anfractuous condition of their walls, and the persistence within them of large vascular trunks absolutely cleared of all tissue. An arrest of the excavating process short of the boundaries of caseation is absolutely impossible, but in some cases caseation is not stopped, even by the spongy tissue around. In constitutions weakened by intemperance or undermined by kidney-disease, the œdematised alveolar tissue falls an easy prey to the advance of softening, which may end in the transformation of a whole lobe, or even of the lung, into a huge vomica.

I have purposely drawn an extreme picture of this affection. In many instances, however, the extent and the intensity of the lesion are limited, so that it may become quite impossible to draw an absolute distinction between a catarrhal and a caseo-fibrinous consolidation. Again, in the less severe cases, the slowness of the process and the occurrence of tissue-irritation, to which I have already alluded, favour the formation of a limiting membrane, which protects the lung against further damage.

In the common form of phthisis known as *tuberculo pneumonic*, owing to the diversity of the morbid conditions, it is difficult to describe any special type of excavation. The following varieties are included in this group.

1. Limited inflammatory processes leading to a generalised tuberculosis.

2. Catarrhal phthisis followed by tubercular infection of a local character.

3. Tubercular phthisis from the first complicated with much inflammation, which often specially affects the peribronchial tissue.

The process of softening may similarly partake of the features of the tubercular, or of the catarrhal, or of the caseous varieties. Many of the excavations which will most engage our attention belong to this class; they need not, therefore, be further described at this stage of my subject.

In the *tubercular* variety of phthisis, characterised by the large size and spongy state of the lung, and by the presence of bunched masses of pigmented fibroid tubercle, yielding a dry surface on section, excavation is a much less prominent feature than in the pneumonic forms. The cavities are often small; their peculiarities are chiefly those of the dense tubercular tissue in which they occur—a tissue where proneness to decay is often surpassed by the tendency to fibrosis. Their walls are thick, abruptly cut, and usually provided with a pseudo-capsule. Tubercular softening is, under normal circumstances, a very slow process, strictly localised, owing to the perfect isolation of the caseum at the centre of a fibrous capsule. Consequently, the work of repair progressing at the periphery is allowed time to prevent, or at least to check any great extension of the cavities beyond the original tubercular deposit. These vomicae, however, may occasionally attain a considerable size from a successive implication of neighbouring tubercular masses.

This part of my subject would be incomplete, were I not to allude to the cavities found in *chronic pneumonia* and in *acute phthisis*. Neither of these forms, strictly speaking, belong to the groups I have hitherto discussed.

Chronic irritation of the bronchi complicated with pleuritic adhesions is at the root of the changes known as chronic pneumonia, which generally include irregular dilatation of the bronchi. Bronchiectasis in this variety is obviously secondary to the fibrosis; it is especially prone to degenerate into phthisical excavations. We are familiar, on the other hand, with a form of bronchiectasis where fibrosis is superadded as a consequence of the bronchial disease. I believe that these two conditions, although extremely different both in their origin and in their clinical history, have been frequently confounded. In respect of the chronic pneumonic form, I am prepared to adopt the able description given by Dr. D. J. Hamilton, and to refer the bronchiectatic dilatations to the fibrosis resulting in the interlobular tissue and in the pleura from the irritation of an inveterate bronchitis.

The pathology of acute phthisis is far from complete, but the connection of the process with a purulent bronchitis of virulent type is generally admitted by the side of complications, sometimes more tubercular, and sometimes rather pneumonic. The cavities in this disease are barely worthy of the name of vomicae, but resemble the sinuous burrowings of chronic abscesses. This feature is illustrated in the diagram which I have borrowed from Hérard and Cornil's work on Phthisis. Softening is invariably accompanied with much redness,

inflammation, and œdema. The excavating process thus derives considerable assistance from the friability of the neighbouring textures, and in the worst cases leads to an extensive tunnelling of the lung. The affection is distinguished by the great rapidity of the softening, by the formation of pus, and by the redness, often erysipelatous, of the bronchial membrane. In this variety of the disease, pneumothorax is often threatened as a consequence of the insidious march of the ulceration, and of the lack of interstitial reaction. Life, however, usually drops before this point is reached, and only at the necropsy do we recognise the multiplicity of the patient's dangers.

The rough sketch which I have endeavoured to give of some of the forms of excavating disease will convince you that, if attempted separately from a description of the respective pulmonary affections, a classification of cavities can possess but a limited value. For practical purposes, some such classification is, however, required; and its most appropriate basis is probably to be found in a description of the cavity-walls.

In whichever process they may have originated, cavities pass through a *period of formation*, during which their walls consist of inflamed or necrosing tissue, incapable of prolonged vitality. Cruveilhier has described this stage under the term "gécide". This, in the typical vomica, is but a state of transition, sooner or later to be succeeded by the development of a lining membrane, for the fibroid material thrown out for the protection of the lung becomes the wall of the cavity when the original consolidation has been softened and removed. The more quickly this change is completed, so much more favourable will be the prospects of the case. Too frequently, however, owing to the progressive crumbling of caseous masses, and to the coalescence of neighbouring caverns or cavernules, a limiting membrane is slow to be developed, or is carried away by the rapidly advancing wave of destruction. This formation of large cavities by a progressive encroachment is clearly to be distinguished from the excavating process in caseous pneumonia, where, large tracts having been simultaneously involved in consolidation and lost from the first for the purposes of respiration, disintegration may proceed to an extraordinary extent without entailing additional strain upon the system.

The absence of a limiting membrane in cavities usually implies that the excavating process is not completed; and that, actively or slowly, decay is progressing, laying bare successive strata of consolidation, and not infrequently exposing the calcified remains of earlier disease. The expectoration of chalky particles gives us a proof of tissue-destruction occurring in a fibrotic district, just as the detection of elastic fibres in the sputum points to the softening of a tissue where inflammatory changes bear a more recent date. In all these non-circumscribed cavities, the surrounding tissue is caseous, fibrotic, or pneumonic, and is doomed, wholly or in part, to destruction. The line of demarcation has not yet been drawn; and too often, as in surgical gangrene, it fails to be drawn before the powers of life are exhausted.

It is, however, in the *mature* cavities that centre our chief interest and our hopes of successful treatment. I would again refer to the vomicae surrounded by well-defined capsules, and developed in the abrupt manner to which I have alluded. The wall of such a cavity usually presents internally a pyogenic lining, and externally it is provided with a substantial fibroid coat. Owing to the suddenness with which it is relieved from internal pressure, and to the spongy character of its environment, the capsule is placed under extremely favourable conditions for contraction. A large proportion of the healed cavities are to be reckoned to this class. This happy termination does not, however, obtain in every case. The caseous abscess may fail to contract, and, after a varying period of suppuration, may assume the character of a chronic vomica.

I agree with Dr. Reginald E. Thompson in thinking that a large proportion of these well defined encapsulated cavities take their origin in hæmorrhagic deposits. I shall have occasion elsewhere to insist upon the remarkable degree of fibrotic action set up at the periphery of the deposits of blood in the lung. It may be incidentally observed that, in simple hæmoptysis, this "reactive" fibrosis is carried out with the full energy of a tissue thoroughly healthy as yet; hence the abrupt transition, not elsewhere observed, of spongy tissue into a tough fibroid capsule. These capsules are in other respects sufficiently characterised by their pigmentation and by the radiating puckering which invariably occurs at their surface.

Whether originally caseous, tubercular, or hæmorrhagic, the cavities ultimately acquire denser walls, and the secretion becomes more free from solid debris. The following is roughly the aspect presented by a cavity in this stage. Internally, the surface is lined with a greyish *false membrane*, often of appreciable thickness, but, in other cases, possessing little more substance than the bloom on a fresh fruit. In either case, it is readily detached, and exposes a red layer, which constitutes

the inner or vascular portion of the capsule, the outer portion of which it purely fibrous. The relative thickness of these three coats varies according to the age of the cavities, and to the degree of irritation under which they may be placed.

1. *False Membrane*.—Most cavities are for a time the seat of a supuration which gradually diminishes, and ultimately ceases, unless called into renewed existence by some fresh irritation. During this suppurative stage, the innermost lining of the cavities presents pyogenic characters; but, at all other periods, it can more aptly be designated as a necrotic layer. If examined under the microscope, it is found to consist mainly of caseous particles and of decaying cells, and to be merely an exfoliation from the inner surface of the cavity wall. In this particular, the wall of a vomica is comparable to the wall of the medullary cavity of bone, for it sheds its waste products internally, and acquires thickness from without.

2. *Fibro-vascular Layer*.—The real gauge for the activity of a cavity is given in the state of the vascular layer, which, in irritative conditions, becomes extremely congested. The remarkable polish of this surface, and its uninterrupted continuity with the surface of the air-tube, have often deluded observers into mistaking the cavity for a bronchial dilatation. The smoothness, the firmness, the bright arterial hue, are the same as naturally belong to the bronchial membrane; and had we trusted to the naked eye alone, we must have concluded, with some eminent observers, that, far from being rare, bronchiectasis is among our most common diseases. But the specific constituents of the bronchial tube are not continued into the cavity. The mucous membrane, with its epithelium and its characteristic limiting structure, is stretched for a short distance over the infundibular entrance into the cavity, but it is ultimately thinned away. Scattered islands of mucous membrane, it is true, may be noticed where outlying bronchi have been intersected by the cavity-wall; and to the continued activity of these bronchial segments is doubtless to be ascribed a great proportion of the secretion commonly referred to the latter; from islands of this nature were probably obtained the cells which have been adduced as evidence of the bronchiectatic nature of cavities formed in tubercular tissue. With the progress of time, the thickness of the vascular layer is gradually lessened, its exposed surface becomes more tolerant of irritation, and the secretion less abundant, whilst the fibrous membrane acquires increasing strength.

In comparatively rare instances, the lining of the cavity becomes purely fibrous, and the space is ultimately encircled by a continuous layer of wavy fibres. The diagram which represents this somewhat unusual condition was taken from a case of chronic apex cavity. More commonly, the fibres are interrupted and intermixed with a varying proportion of vascular tissue; but, in all cases, this layer is in contact with a fibro-nuclear zone, which becomes denser and more fibrous on the cavity side, whilst its outer boundary is lost in the spongy substance. Invariably, also, lymphatic spaces are enclosed in the meshes of the fibrous layer; they are readily identified, owing to the pigment-particles which they usually contain.

I would call attention incidentally to a peculiar appearance, not hitherto described, presented by cavities of old standing, which have reached the pleural boundary. In these I have often noticed a fine fasciculation, closely resembling the muscular coat of an intestine from which the peritoneum has been detached. The fasciculation appears to consist mainly of fibrous tissue; it is striking only in respect to the arrangement of the fibres, which is almost invariably horizontal. Having found the same appearance in the lining membrane of an empyema, I do not look upon this formation as special to pulmonary cavities, but more probably referable to the pleural membrane.

The chief features of the walls of cavities in phthisis are briefly the following:

1. Absence of protecting epithelium.
2. Gradual decay within, leading to the formation of a necrotic layer (pseudo-pyogenic).
3. Gradual fibroid growth from without, constituting the so-called capsule.

I have made it sufficiently clear that, by whichever process produced, the cavity-membrane is never a separable entity, but a surface-induration. Loose capsules have been described by Dr. Reginald E. Thompson as due to the retraction of a layer of blood which originally adhered to the cavity-wall. For such an appearance, I can conceive but two other explanations: either the loose capsules must be due to a pulmonary aneurysm which has expanded into a cavity, and furnished it with an accurate internal lining; or we must imagine the unlikely event of the necrotic zone special to the cavity having been shed in its integrity, and in a layer of considerable thickness.

The eminently practical classification of cavities given by Dr. Douglas Powell in his book *On Consumption* (second edition, page 102), is based upon the pathological states of the cavity-wall. Cavities are divided

into four classes: (1) the recent; (2) the quiescent; (3) the secreting; (4) the active or ulcerous. As expressions of various phases of development and of morbid action, this nomenclature is complete, and it corresponds to equally well defined clinical states.

From a different standpoint, that of anatomical description, I would propose, side by side with this useful division, the following classification of cavities:

1. Cavities devoid of limiting membrane: in these excavation takes place by a crumbling in the dry variety, by a liquefaction in the moist variety. The usual type is the moist. Reaction is at a minimum, and disintegration often overtakes the fibroid zone before it has become a protection to the tissue. *Special subvarieties*: a. Suppurative necrotic form, commonly present in acute phthisis, where it is apt to occasion pneumothorax; b. Simple liquefaction, apparently from soakage, most frequently witnessed in compressed devitalised tissues when attacked by oedema; c. Ichorous ulceration, observed in the rigid and sinuous burrowings of indurative chronic pneumonia.

2. Cavities possessing a distinct capsule in the midst of spongy lung-tissue: excavation is due to limited caseous abscesses, frequently also to the softening of hæmorrhagic foci.

3. Chronic cavities with thick lining, continuous, with more or less induration, partly inflammatory, but chiefly due to collapse.

4. Chronic cavities with thick lining, surrounded by a rim of tubercle.

[To be continued.]

ABSTRACT OF LECTURES

ON THE

MORPHOLOGY OF THE MAMMALIAN SKULL.

Delivered at the Royal College of Surgeons of England.

By W. K. PARKER, F.R.S.,
Hunterian Professor of Anatomy in the College.

LECTURE III.—ON THE SUPERFICIAL CARTILAGINOUS SKELETON: LABIAL, ORAL, AND PHARYNGEAL; ON THE LIMBS; AND ON THE DERMAL (BONY) SKELETON.

THERE are certain parts of the vertebrate framework which cannot be classed with the endoskeleton; these are both cartilaginous and osseous. The former are usually developed in the lower types, and are manifestly archaic; they seem to represent elements which probably formed the principal part of the skeleton in types which we must suppose to have existed as the precursors of our present vertebrates.

The most simple and primitive of these parts are the barbels or oral palpi. Next to these come the cartilage formed in the substance of the lip, and the separate cartilaginous valves lying around the openings of the nasal capsules. The proper lip-cartilages evidently represent the proximal ends of barbels, which have more or less lost their distal parts. All these cartilages are classed together as labials.

In *Amphioxus*, the main skeletal parts are a fringe of labial palpi which surround the mouth. In the most abortive forms of fishes—*Myxine* and *Bdellostoma*—there is no proper suctorial mouth; but that opening is surrounded by barbels, which have a cartilaginous pith with a broad base. Instead of the detached nasal valves, which we find in *Selachians*, *Anura*, etc., these *Myxinoids* have a long passage leading to their single nasal aperture which is supported by a series of rings of cartilage, imperfect below, forming a structure very much like that of the mammalian trachea.

In the next form above these, the lamprey, in its larval condition as an *Ammocete*, we have a very simple *myxinoid* condition, there being no suctorial disk; the mouth is surrounded by palpi, but there is no trachea-like prenasal tube. In the adult lamprey, the skeleton—which is composed entirely of cartilage—is mainly superficial. There is a very perfect suctorial mouth, surrounded by one solid ring of cartilage. This latter, which is a lower labial, has another median T-shaped piece behind it, and a styloid cartilage on each side. It is surmounted above by a large median shield-shaped upper labial, and a styloid and a subrescent piece behind this. The visceral arches in this type are only partially differentiated from the basis cranii; there is no proper mandible, but an immense lingual cartilage in the floor of the mouth represents the basi-hyo-branchial. These, and the imperfect neural arches, constitute the whole of the proper endoskeleton; but a large and highly complex basketwork of soft cartilage encloses