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The Bacillary Origin
of Phthisis Pulmonalis

I certify that this Thesis has been composed by myself.
Auga Matheson.
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The Bacillary Origin of Phthisis Pulmonalis

There is no disease about whose pathology more has been written than that of Phthisis Pulmonalis. When we consider the various manifestations, the universal distribution of the disease, and how it affects the high and the low, the young and the old, selecting as its favourite victims those who are just entering the arena of life, and causing a mortality which has been computed by some to be as high as one seventh of the total death rate of this country—it can no longer be wondered at that men have always been striving to ascertain the nature and origin of such a scourge of the human race, in the hope that some means might thereby be found for averting it or at least checking it in its progress. In the endeavour to elucidate the problem, we should expect that many difficulties would present themselves, some of these have been overcome; but even at the present day with all our
improved methods of research, & knowing so much about the disease as we do, we are very far off indeed from the attainment of a satisfactory explanation of the manifold appearances presented to us. Proof of this is easily obtained by glancing at the various views that are held as to the etiology of the disease. Every year our knowledge is increasing; and though we are still a great distance from the goal of our aspirations, we must work steadily on, confident that at last the day will come, though we may not live to see it, when truth will prevail.

About sixty years have passed since Laennec in his Classical Work declared that Phtisis Pulmonalis consisted in the deposition in the tissues of the lungs of one morbid product which he named "Tubercle." This Tubercle presented itself in an infiltrated or in an isolated form; and in either case, caseation & softening were liable to ensue. Thus caseous matter came to be looked upon in every case as having arisen from Tubercle; and a
great advance was made when Virchow showed that calcification was merely a form of degeneration, which might take place in matters simply inflammatory in their origin, and in various other morbid products such as tumours. There then arose the dualistic school who taught that the different lesions in Phthisical lungs should be divided into two groups—those that were inflammatory & those that were tubercular; the characteristic & distinguishing feature being the anatomical structure. The doctrine that now came to the front, & of which the chief upholders were Bühl & Thiemeyer, was that tubercle were caused by preexisting caseous material inflammatory or otherwise in origin, as the two were frequently associated and distributed in such a manner, as to lead to the conclusion that they stood in one another in the relation of cause & effect. When Villemin in 1865 showed that a general tuberculous resulted on inoculation into animals, not only of tubercular material, but also of caseous matter from
a phthisical lung or a scrofulous gland, a certain revival of Laennec's views as to the "unity" of Phthisis came about. Thus a few years ago, just before Koch published his account of the Bacillus Tuberculosis, there were some who upheld the doctrine of the "unity" of Phthisis, while there were others who firmly believed in its duality. In April 1882 Koch gave forth to the world his important discovery of a specific Bacillus in Phthisis & certain other diseases, this conclusion concerning its significance. His experiments have since been verified, notably by Watson Cheyne in this country; & discussions have been held in the abodes of scientific enquiry in all the civilized countries of the world, as to the relations of the microorganism to the diseased tissues in which it occurs. Those who had previously believed in the unity of Phthisis now considered it satisfactorily demonstrated that their views had been correct; while very many of those whose views had been dualistic, enamoured with the glamour of a new discovery, unhesitatingly gave in their adherence.
to the most recent doctrine. Men's minds had now something tangible to work upon, in their efforts to understand a disease whose manifestations are both various and difficult to comprehend as Phthisis; as a result, the tendency has been to blame everything on it to explain everything by the presence of the Bacillus Tuberculosis. Nevertheless there were, and there still are, some men who have been unable to agree with the conclusions arrived at by Koch and his followers. Though they recognise the great value and importance of the discovery, they cannot bring themselves to say, as Klein has done, "No Bacillus without Tubercle; & no Tubercle without Bacillus."

I have carefully considered the anatomical manifestations of the disease, as given in standard works; I have maturely considered what to me has seemed of nearly equal importance in endeavouring to form correct ideas as to the nature of the disease, namely its Clinical History; and I have given my careful attention to
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the many discussions which have taken place of which reports have appeared in Medical Publications from time to time. What I have sought to do in the following pages is to give expression to those views which I have in consequence formed, as to the significance of the Bac. Tuberculosis in disease of the lungs, and my reasons for having formed these views.
Chapter 7

I am met upon the threshold of my discussion by what is not a very easy matter — the giving a good definition of Phthisis Pulmonalis, or Phthisis as I shall call it, without introducing the vexed question of its etiology. The tendency at the present day seems to be to make Phthisis synonymous with Pulmonary Tuberculosis, as used by those who look upon the Bacillus Tuberculosis as the exciting agent of the disease & call a disease Tuberculous whenever the Bacillus...
is present, no matter what the anatomical changes may be. I shall however limit the term **Pulmonary Tuberculosis or Tuberculosis or Tuberculous Disease of the lungs** to those diseases of the lungs which are characterized by the presence of Tubercles; and shall employ the term **Bacillary Phthisis** to signify a disease of the lungs in which Bacilli Tuberculosis are found in the tissues or pathological products; and thus avoid acknowledging that there is necessarily any further identity between Pulmonary Tuberculosis and Bacillary Phthisis than the presence of the **Bacillus Tuberculosis**. I have used the word **Tubercle**; by it I mean a small rounded nonvascular tumour, not growing beyond a certain size, made up of one or more giant cells from which a reticular tissue radiates; both in and on this reticular tissue are numbers of epithelioid cells, and, especially towards the periphery, small lymphoid cells. A young tubercle however has not such a structure — it merely consists of a collection of cells; its distinguishing feature is its inherent
tendency to develop into a typical tubercle. Tubercles are liable to undergo caseous degeneration, or on other occasions to develop into fibrous tissue; from either of these changes having occurred, the typical structure may not be seen. As a rule however, tubercles are readily recognised in tissues otherwise normal or diseased by their giant cells. It has been said there is nothing characteristic in the elements of a tubercle; for giant cells have been described by Liegler as occurring in simple chronic inflammations, and the other elements are found in various parts of the body under different conditions. But it is not any one element that is characteristic but its whole structure & the relation of its several parts to one another; or the inherent tendency to form such a structure. As well might it be said that there is nothing characteristic in a cancerous tumour, whose component elements occur in so many situations, but not with the same relations to one another as in the tumour. In Liegler's Pathology (P.166) it is stated that in the future we are to understand by a tubercle a cellular module containing
within it the Bacillus Tuberculosis; but I think such a definition by no means satisfactory, when Klein definitely declares that there are tubercles which contain no Bacilli.

Wishing then to avoid the question of Aetiology, I define Phthisis as a disease of the lungs in which interstitial changes, or intraalveolar and intrabronchial changes, or both, have led to caseation and destruction of lung tissue; it is usually progressive in character and unless in exceptional cases, accompanied by progressive wasting of the body. Thus are excluded acute Pneumonia, Pleurisy, Bronchitis, Catarrhal or Lobular Pneumonia, Emphysema, Collapse, Brown Induration from heart disease, tumors and parasites; but it does not of necessity follow that certain of these diseases may not be so modified in their course under certain conditions, as to lead to caseation and destruction of lung tissue. There is a disease of the lungs described by Dr. Fyffe (Pr. Med. P. 904) as Atrophy of the Lung or Chronic Pneumonia; it occurs very exceptionally and the only structural change is a marked increase of the fibrous tissue of the
lung; it arises secondarily to a croupous or catarrhal pneumonia. The diseases of the lungs induced by syphilis, Inhalations of Dust Particles, a chronic Pleurisy, or a chronic Bronchitis consist, at least in the early stages, of a very similar increase of fibrous tissue. If such a condition becomes complicated by caseation and destruction of lung tissue, then only then does it come under my definition of Phthisis.

"Miliary Tuberculosis" of the lungs, pure and simple, occurring as part of a general disease of which it is but a manifestation, I do not include under my definition of Phthisis.

Chapter II

The various terms which I shall have to employ having now been defined in the sense I shall use them, it is necessary for the better understanding of what is to follow, that I should briefly describe in a general way the structural changes that are found in the lungs of persons who have succumbed to Phthisis. The appearances presented are very various; and on first looking at an
advanced case of chronic phthisis, all kinds of lesions would seem to be inextricably mingled together. But by careful examination we shall find, by leaving out of consideration for the time being the tubercles which are so frequently present, that in every case the morbid changes before us may be divided into inflammation and necrosis. The inflammatory phenomena affect the interstitial tissues, the alveoli & the bronchi; the necrosis or caseation attacks both the inflammatory products & the lung tissue.

Let us first consider the inflammatory processes occurring in the interstitial tissues. In the majority of cases, they consist in a chronic inflammatory condition, resulting in an increase of fibrous tissue in one or more of the situations where it naturally abounds—the pleura, interlobular septa, around the divisions of the bronchi & pulmonary artery, & the interalveolar septa. Microscopically all the stages of development of the new tissue may be traced: where the process is most recent an abundance of small round cells is presented to our view; where the process is more advanced many spindle shaped & branching cells are seen; while in the oldest part nothing but dense
cicatrical tissue is present. Bloodvessels are numerous in the part where the process is most recent, but in the fully formed tissue few or none are seen. Contraction takes place in this cicatrical tissue, and in consequence the chest wall is drawn in to a certain extent, and bronchiectatic cavities are formed from the traction exerted upon the bronchial walls. Other agencies entering into the causation of the formation of these bronchiectases are destruction more or less complete of the muscular tissue, yellow elastic fibres, cartilages of the bronchial wall, from the pressure of the small celled infiltration, retention of secretion from some obstruction, with consequent distension from the increasing accumulation (3) forced expiratory efforts acting on the bronchial wall weakened by the small celled infiltration (4) compensatory dilatation (5) ulceration of the bronchial wall, occurring in acute lobular pneumonia (6) tubercular or caseous ulcerations. In some of the bronchiectatic cavities is contained a putrid purulent material, while in others is a thick pulpy caseous cheesy looking mass composed of inspissated catarrhal secretion. These cavities frequently simulate those due to caseation & softening of lung
Tissue: there can be doubt of the real state of affairs however, if the basement membrane & columnar epithelium of the bronchus be found lining the wall. Still in some cases, there is a gangrenous inflammation of the bronchiectatic wall, & here it is almost impossible to distinguish such a cavity from one due to caseous destruction & softening of lung tissue the walls of which have become gangrenous.

But the interstitial inflammatory change are not always chronic, for occasionally it happens that in acute lobular pneumonia the small celled infiltration of the intralobular bronchi leads to suppuration: such an ulcerated bronchus tends to dilate. This supplicative condition of the bronchi is very frequently associated with a similar acute inflammation of the alveolar walls, which may thus aid in forming the resulting cavity or abscess. When the disease is so acute it is usually rapidly fatal, but in exceptional cases recovery occurs by discharge of the pus and cicatricial contraction of the surrounding tissue, or perhaps sometimes by the purulent material drying up.
and becoming caseous

Let us now turn our attention to the inflammatory changes affecting the alveoli and intralobular bronchi, for both these structures are usually in a similar manner if at the same time. They consist in a catarrhal inflammation with a lobular distribution, resulting in the accumulation in larger or smaller areas of inflammatory products in the alveoli and intralobular bronchi; there is present also a greater or less amount of small celled infiltration in their walls, which, as already mentioned, may in acute cases go on to actual suppuration. There is usually more or less fibrinous exudation along with the catarrhal changes, more especially in the zone of air vesicles around the central intralobular bronchi. In some cases a croupous inflammation may be the principal change but even then we can usually make out a lobular distribution. Cornil & Ranvier state that in Phthisical lungs the fibrinous change is usually quite as marked common as the catarrhal. In parts the inflammatory products, as well as the lung tissue
containing them, have undergone caseous degeneration. Microscopically, if we examine a lobule in which caseation is commencing, we shall find that the central bronchus with its contained inflammatory products presents a dark opaque appearance, due to caseous necrosis having occurred; the alveoli to a greater or less extent around the bronchus have also caseated, as well as the contained products of inflammation; further out the alveolar walls may be seen but the alveolar contents are caseous; towards the peripheral zone of the lobule, the principal change consists in the alveoli being filled to a greater or less extent with catarrhal epithelial cells perhaps becoming fatty. No bloodvessels are present in the central area, but as the periphery is approached where there is no caseation bloodvessels are seen. When the caseous matter softens perhaps for the same reason as maturation occurs in cheese) a cavity is formed. By increasing caseation and softening, and confluence of smaller cavities, larger ones are formed so that in time a great part of one lung may become converted into one large cavity. The
caseous masses however do not always soften: but the lobular pneumonia ceasing to extend and an interstitial inflammation around the caseous area being set up, it may become encapsulated; such encapsulated masses are liable to become infiltrated with lime salts & to form a calcareous mass which may lie innocuous in the tissues for many years. In other instances, the softened caseous debris may be removed and a surrounding interstitial inflammation form a lining of granulation or fibrous tissue to the resulting cavity.

There are frequently caseous masses to be seen, often in the thickened interlobular septa, which are not formed from a caseating lobular pneumonia. They are produced by a process like that which causes a similar condition in syphilitic gummata & in the fibrous nodules found in "stonemasons lung"—by the artery supplying a part becoming gradually obliterated by endarteritis obliterans.

The Bronchi always suffer from a more or less general catarrh, often localised however to that part of the lung
where the other morbid processes are found. There may be caseous material in the
dilated bronchi, & in the intralobular—
bronchi (associated with caseous lobular pneumonia)
In other bronchi are found ordinary
inflammatory products.

Though I have described separately
the changes affecting the different structure,
it must be borne in mind that they are
invariably combined to a greater or less
degree; and that according to the predomi-
nance of one or the other, special character
will be given to the appearances. Thus the
interstitial tissues may be most markedly
affected, as in Syphilis & the Pneumonokonios;
or it may be that the chief affect
consists in a lobular inflammation, as in
that variety of caseating lobular pneumonia
which frequently occurs in children after
measles or whooping cough; & in whom the
disease may run such a rapid course that
death ensues before softening has taken place
in the caseous masses. It is rare for this
latter to occur in an adult: softening
can nearly always occur be found in the
caseous areas at one or other part of the lung.

All the above inflammatory changes, whatever cause they may be due to, are such as occur in ordinary inflammations, from which they differ only in the fact that the inflammatory products of the lung tissue are liable to undergo caseous degeneration.

_Tubercles_ are not always present; whether they are or not, the inflammatory and caseous changes are as above described in varying combination. When tubercles are found in the more fibroid conditions, they are seen to occur in the pleura and interstitial tissues—

in short to follow the lines of the lymphatics. They are frequently in greatest numbers around the dilated bronchi and have a tendency in their further development to become converted into fibrous tissue. Tubercles are also frequently present when caseating lobular pneumonia is the prominent lesion. They occur away from as well as in the midst of the inflammatory areas. On the other hand, large tracts of the inflamed tissue may be examined with the result of finding few, if any, tubercles, either with the
naked eye or microscopically. The tubercles are liable to undergo similar degenerative changes to the surrounding inflam. products & to become caseous. In the bronchi tubercular ulcers are frequently present.

Chapter III

Before discussing what is believed by many to be the direct causal relationship of Bacillus Tuberculosus to Phthisis, let me describe in a few words what the life history of the Bacillus is, what Koch's experiments have been, and his conclusions.

Koch found that certain Bacilli were constantly or almost constantly present in tubercular & certain caseous matters from the lungs & other parts of the body. These Bacilli by giving a reaction to a special staining process are distinguished from all other known Bacilli except the Bacillus Leprae which however differs in shape & in being stained by other reagents. To this Bacillus he gave the name of Bacillus Tuberculosis.
broth or blood serum, but only at a constant temperature between 86° and 105.8° F.; it is of comparatively slow growth, and cannot continue its development in fluids which are decomposing from the presence in them of more rapidly growing putrefactive Bacteria. The Bacillus Tuberculosis however is very tenacious of its vitality, and preserves its power of development & its virulence for six weeks or even more in decomposing sputum; & for six months or longer in the dry state. This is due probably to the spores, which are formed in the process of development, being very much more resistant to injurious influences than the Bacillus itself. These spores are formed within the body; drying of phthisical sputum does not destroy its virulence, because of the presence of the resistant spores.

When caseous or tubercular matter containing the Bacillus Tuberculosis is inoculated into the tissues of an animal, an artificial general tuberculosis is produced; that is to say "swollen lymphatic glands near the seat of inoculation, with subsequent caseation & softening; enlargement of the spleen..."
due to numerous whitish tubercles, the larger ones caseous, enlargement of the liver which is mottled by the presence of uniformly distributed whitish points and streaks, which by and by become confluent caseous tuberculosis of peritoneum; isolated tubercles in the lungs at first grey and transparent then caseating in the centre; enlargement and subsequent caseation of bronchial glands (Klein, Microfascicular Disease p. 166): this artificial tuberculosis also results on inoculation of Bacilli from a pure cultivation. We may then look upon artificial tuberculosis as a chronic inflammation, going on to caseous degeneration characterized by the presence of tubercles which have the same appearance and structure as those occurring in the human body in acute general tuberculosis. When Bacilli containing caseous matter or a pure cultivation of the Bacilli is diffused in water and sprayed into the air of a chamber in which animals are placed, tuberculosis results in the lungs and bronchial glands; the intestine and mesenteric glands become tubercular when animals are made to eat Bacilli-containing material. In all these experiments, in all or nearly all of the resulting tubercles, Bacilli are present, and on inoculation of portions of these tubercles into other animals, an artificial tuberculosis is again
produced. These experiments have been verified by various observers.

In the majority of cases of Phthisis, Bacilli are present in the sputum; indeed they are said to be always found in well-marked cases, but it is more doubtful if they are commonly present in the very early stages when there are no signs characteristic of phthisical disease. They are found in great numbers invariably or almost invariably in phthisical cavities, and little masses may be detached from the walls of old cavities almost entirely composed of groups of Bacilli. Douglas Powell (Dis. of Lungs, p. 276) states that the Bacilli are scarce and rather difficult to find in cases of catarrhal pneumatic consolidation except in the immediate neighbourhood of cavities. Bacilli are very generally but not always present in the subacute and acute general Tuberculosis: Dr. Klein & Sebbas found in tubercular lesions produced in guinea pigs by inoculation of Bacilll-containing matters, but few or in many instances no Bacilli. In old tubercles they are to be found with difficulty.

Koch concludes that military & acute general tuberculosis, cheesy bronchitis, intestinal & glandular tuberculosis, bovine tuberculosis,
and spontaneous or inoculated tuberculosis of animals are all "tuberculous," taking as his diagnostic criterion not any special anatomical structure of the lesions but the presence of the Bacillus Tuberculosis, and he holds that the Bacilli present in these diseases not only accompany them but are the cause of them.

Thus it is that he believes that Bacillary Phthisis is due to direct infection with the Bacillus Tuberculosis. According to Rindfleisch (Elem. of Path. 1876) the first changes due to the infection occur in the finest bronchial tubes just where they open out into the infundibula, and consist in a cellular deposition in the form of nodules ridges & other shaped circumscribed infiltrations in the connective tissue; hence the process spreads in either or both of two directions— from the peribronchial tissue to the neighbouring interstitial tissue, or directly to the alveoli giving rise to lobular pneumonia caseation. Watson Cheyne however (Brit. med. J. 1893 vol. 7, p. 339) states that the Bacilli act first on the epithelial cells of the alveoli, & that it is from here that the process spreads, inducing inflammatory changes more or less acute or chronic, depending on the number of the Bacilli—the suit—
ability of the soil.

In whatever way the Bacillus is regarded in its relations to Phthisis, it is agreed that it acts in some way as an irritant, setting up an inflammatory process, characterised by the presence of tubercles associated with inflammatory changes in which there is a great tendency to caseous degeneration.

It is supposed according to Woodhead and Hare (Practical Mycology p. 902) that the Bacillus Tuberculosis is an organism which acts chemically on the living tissues by its secreted or excreted products in such a manner as to alter them from their original resistant condition to one in which there is increased vegetative activity but a diminished power of resistance to the Bacillus itself. Thus the Bacillus is enabled to use up as food the highly organized substance of the weakened, causing caseation. Chemical formation takes place anew, fresh cells are acted on, so the process advances. It is said that a good example of such a process is seen in disseminated caseous bronchopneumonia or so-called "bronchopneumonic tubercle", where caseation has occurred of the intralobular bronchus.
of the surrounding air vesicles, and of the contained inflammatory products; as the periphery of the lobule is approached, the chief change consists in an active proliferation of the alveolar epithelium. Bacilli are abundant in the caseous centre but toward the periphery they gradually diminish in numbers, till in the zone where the cells are proliferating none whatever can be distinguished. If this supposed mode of action of the Bacillus upon living tissues be correct, it is easily understood how a caseous mass, containing not only the Bacillus but its food and its products, is able on reaching an alveolus or small bronchus to set up changes in the tissues, by the action of the products so altering these tissues as to render them fit for being acted upon by the Bacillus itself. It is also easily understood how a Bacillus, without its food and products would not be able to act upon living tissues, unless indeed the resisting power of these tissues could be diminished in some way so as to approximate them in their condition to those acted on by the products of the Bacillus.

It is right however to add that it is not universally admitted that the Bacillus
acts on the weakened cells so as to produce caseation: for Green in his Pathology, & others, maintain that the caseous degeneration is due to conditions induced by the Bacilli interfering with the blood circulation in the part.

Chapter IV

Are we justified in concluding from Koch's experimental inoculations that the various inflammatory lesions occurring in Phthisical lungs, with or without accompanying tubercles, owe their origin to the Bacillus Tuberculosis when it is present? When tubercles occur, I believe we are entitled to hold that they are due to the action of the Bacillus, as well as to some or all of the accompanying inflammatory changes; but I do not admit that all or any of the inflammatory changes in which Bacilli are present are necessarily caused by the Bacillus.

Germain Séé (Bac. Path. 5:109) "following Koch & other authors" states that the presence of Bacilli Tuberculosis in diarrhoeic stools is a sign of "Tubercular" or caseous ulceration of the intestine: in my opinion this cannot be a "certain"
sign of the existence of such a lesion, for it is admitted that Bacilli gain access to the intes-
tinal canal by swallowed phthisical sputum and it is quite possible that they gain access also, even in persons who do not swallow their sputum, by swallowed food removing from the mouth and pharynx Bacilli which have reached those parts in the expired air or in the sputum. Bacilli or at least their spores — as I shall show further on — must frequently be present in the secretions of the lungs of nonphthisical persons though in such small numbers as not to be recognisable by us; it can therefore be easily understood how these Bacilli might become so numerous as to be capable of ready recognition, if they were allowed to remain long enough in the secretions or usual inflammatory products to develop, and that without there being any disease caused by them, in the same way as Bacilli may be present in diarrhoic stools without necessarily having been the cause of the disease of the intestine producing the diarrhoic stools. Still the presence of Bacilli Tuberculosis in inflammatory products or lesions
Difficult to determine whether inflammation is due to Bacillus tuberculosis or ordinary inflammation.

The diagnosis can only be made by finding the Bacillus in the sputum. Distinctive symptoms are lacking, and pathological changes are not sufficient to determine ordinary tuberculosis.

The inflammatory lesions, which we know are due to the Bacillus, could be wrong.

The anatomical features of the disease, if it could be shown, would be strong presumptive evidence of their being the actual cause of the disease.
diffused consolidation (induration) of the lungs between the tubercles is partly effected by a "tubercular" (due to Bacillus), and partly by a simple, interstitial pneumonia in the septa, alveolar walls, etc.

And I do not admit, as Charcot has stated, that tubercles, & caseating catarrhal pneumonia are identical. It is true that in the early stage of tubercle formation, when it merely consists of a cellular nodule situated in one or more alveoli, it presents a somewhat similar appearance to a catarrhal pneumonia; but even then there is a difference between the two, inasmuch as the tubercle grows from the alveolar wall itself involving in the process, while the pneumonia consists in a proliferation of the alveolar epithelium, the alveolar wall, perhaps infiltrated with cells, remaining quite distinct (unless of course caseation has occurred). But it has been stated by Klein, Watson, Chaynes & others that true giant cells do arise from such proliferated alveolar epithelial cells; it is allowed however even by these observers that giant cells may perhaps also arise in the connective tissues, and Cornil & Ranvier, and
Hamilton give figures in their works which seem to prove that certainly in some instances at least Tubercles arise in the alveolar wall, in which case the giant-cells are developed from the endothelium of the lymphatics or bloodvessels or from connective tissue corpuscles, or according to Tiegerch chiefly from white blood corpuscles.

Now if we look upon giant-cells as sometimes originating from the alveolar epithelium thus entering into the formation of Tubercle, we are forced to believe that in a Tubercle an epithelial cell—or cells—is capable of undergoing a form of development which does not occur in epithelium in any other inflammatory condition, and which does not form a part or stage in the normal development of epithelial cells. We know however that giant-cells occur both in chronic inflammations in tumours & in bone, when they are derived from cells of the mesoblastic layer of the embryo; and we know that giant-cells are but a stage in the normal development of connective tissue. So that it seems to me that we are not justified in considering that giant-cells can arise from epithelial cells.
and that we can only admit their origin from cells derived from the mesoblastic layer of the embryo.

Thus anatomically there is no means of deciding whether an inflammatory lesion containing the Bacillus Tuberculosis is due to the Bacillus or not; and I hold that all we are justified in concluding from the presence of the Bacillus in inflammatory processes is that the Bacillus may be the cause of part or whole of the inflammation but not necessarily because of any of it. It will have been gathered from what I have already said that I do not include Tubercles in these inflammatory processes, when I say that such may be due to the Bacillus, for we know that Tubercles are caused by the Bacillus.

It is easy to understand however, how Bacilli, which in acute general Tuberculosis give rise to Tubercles and chronic inflammatory conditions by acting as an irritant, could on reaching the human lung in the inspired air give rise to the various inflammatory lesions with
or without tubercles, which occur in Phthisis. But as I have said, finding the Bacillus does not mean that it is necessarily the exciting cause. We must try to ascertain if the Bacillus, on entrance into the lungs, can induce similar conditions. Of course such inhalations can only be performed experimentally on animals; they produce in them tuberculosis of the lungs & bronchial glands, but I have not been able to see a full & detailed description of this 'tuberculosis'. Thus I do not know whether an inflammatory condition has resulted on some occasions, or whether an eruption of tubercles with a varying amount of accompanying inflammation has been the result. If the latter has been always what followed, it is evidence against rather than for Bacilli Tuberculi being the cause of an inflammatory condition of the lungs in man in which no tubercles are present, though Bacilli Tuberculi are, and leaves it an open question as to whether all inflammatory changes accompanying tubercles are due to the Bacilli or not. While if sometimes one, sometimes the other, condition was induced, it would be very strong presumptive evidence that similar diseases were due to the same cause—namely the inhalation of the Bacilli. But even then, since we know
that Bacidia or inoculation only give disease to certain animals. I hold that we must be able to trace some relationship of cause and effect between exposure of the lungs to the Bacilli and a resulting Bacillary Phthisis, before we have the right to say that Bacilli Tuberculoid can induce disease of the lungs in man, upon their reaching these organs. It will now be my endeavour therefore to search for any such relationship.

The Bacilli or their spores are present in great numbers in the sputum of very many cases of Phthisis, and are stated to occur in the air expired by such patients. When the sputum is expectorated it dries up, & the dust containing the still vivulent spores of the Bacilli is carried about by currents of air; so that it is easily understood how the Bacilli or their spores must be present in great numbers in the atmosphere of towns & of all places where people are congregated together with a phthisical patient among them, as in workshops, hospitals, barrackes. When particles of Carbon are floating about in the air, they are inhaled & some of them penetrate to the inmost recesses of the lungs; they may be found in the
alveoli & in the alveolar walls, and can be traced through the lymphatics to the bronchial glands. There are also certain experiments which show that fine particles do enter the lung tissues. So that it may be assumed that such minute germs as the Bacillus Tuberculosis also reach the alveoli & lymphatics of the lungs. That the Bacilli do enter into the lymphatics at some part of the body & probably those of the lung, is borne out by the fact that the Bacilli are found in certain joint diseases, which situation they must have reached by means of the blood. Bacilli Tuberculosis also sometimes penetrate by means of the lymphatics of the intestine to the mesenteric glands: and putrefactive Bacteria are found in the urine in the urine of animals who have been fed on putrid material. I have therefore no doubt whatever, that the Bacilli Tuberculosis are not only frequently inhaled into the alveoli but that they also pass on many occasions into the lymphatics of tissues of the lungs. Since then such is the case, we would expect, looking upon the Bacillus as the cause of Bacillary Pneumonia, that the world would soon become depopulated. But it is certain that all who inhale the
Bacillus do not become phthisical, indeed much the greater number do not. How can this be explained if the Bacillus is the exciting cause of Phthisis? The difficulty is sought to be got over by supposing that there is a predisposition on the part of the tissues of certain individuals so that there has been some state of the lungs present which allowed the Bacillus to obtain the necessary condition of rest. Let one endeavour to ascertain if the difficulty be satisfactorily explained in this way.

Germain Sée (Bac. Ph. 645) and Landouzy & Martin (Sanct 504) hold that the virus can be actually transmitted from the parent to the foetus, and they state that the Bacilli are found in the placenta, foetal fluid, which on inoculation will produce an artificial tuberculosis. But it can hardly be believed that this transmission occurs to any extent if at all, when we consider how seldom children are born with tubercular or phthisical disease.

It cannot be doubted that in certain families there is a great tendency for the various members to become phthisical.
— a tendency which is hereditary. The Bacillus is said to be able to attack such persons because they have inherited a "weakness of the tissues" of the lung, alone or in common with the rest of the body, consisting in a condition which renders them peculiarly incapable of resisting the action of the Bacillus. It has been suggested that this "weakness" of the tissues is very similar to what occurs in certain persons with regard to infectious diseases, such as diphtheria & scarlet fever; for it is a well-known fact that some people are frequently exposed to the contagion of these diseases & yet escape infection, while others are infected on almost the first exposure. But this comparison does not hold good for whereas with the zymotic diseases, one exposure may be sufficient for infection, those persons "predisposed" to Phthisis may have been exposed to the possibilities of infection by the Bacillus, & even had the Bacilli actually in their tissues on countless occasions, & yet escape. It may be objected that the Bacilli, though they reached the tissues, did not obtain rest; but this objection is not valid for how then do the Bacilli gain a footing in animals on experimental inhalation, and
in man & animals give rise to the tubercles of general tuberculosis, when the Bacilli must be under the same conditions as regards rest as they are when inhaled into the tissues of the lungs in ordinary condition of life. How frequently do we see "predisposed" persons living in close association with those affected with Bacillary Phthisis, and exposed to the possibilities of infection with the Bacillus, in both large & small quantities as we would think, month after month or for years; yet they may escape till after the death of the patient?—Scott states that brothers & sisters frequently die in succession & have a considerable interval between their illnesses. I therefore cannot believe that there is a "weakness of the tissues" inherited, which will allow the Bacillus to obtain a footing on reaching the lungs: I cannot regard the hereditary tendency to phthisis as anything more than an inherited condition of the tissues, which renders them liable to be altered in some way so that the Bacillus can obtain settlement, or a condition which on being altered by some accidental local or general affection will allow the Bacillus to
obtain a settlement.

Since then we cannot explain by an "inherited weakness" of the tissues alone, why the Bacillus should attack certain persons; let us see if there cannot be acquired some condition which will allow it to get a footing. Koch states that denudation of epithelium of the small bronchi, and imperfect movements of the lungs, retained secretions, are favourable to if not necessary for the onset of the Bacillus. But these acquired predisposing conditions are so intimately associated with those which are said to favour the onset of the Bacillus at the apex in so many cases, that I shall consider them all together.

Germainée (Bac. Ph. 540), a firm believer in the Bacillary origin of Phthisis, gives the following alternative reasons for the frequency with which Phthisis is localised at the apex, at least in the early stages, as the conformation of the chest, that is, the incomplete inspiratory distension at the upper segments, from which an accumulation of mucus & epithelium in the bronchioles results; the idea is that the upper portions of the musculous wall do not expand properly during inspiration.
Gauchard admits functional inertia of the superior lobes as the cause. Peter says that in the lung the apex is the least living part, for it is the least acting; and it is here that air circulates with such difficulty; consequently tubercles occur where we find minimum of haemoptysis. Let us direct our attention to each of these in turn.

An accumulation of mucus and epithelium will tend to occur in the apex of the lung, because there the respiratory movements are least; it will also be liable to take place when there is a diminution of the lumen of a large bronchus from pressure, or when there are adhesions binding down the lung, the remains of a chronic pleurisy, or when the chest walls are deformed or ill-developed. That mucus and epithelium ever accumulates remains as such for any length of time I very much doubt; we never hear of an accumulation of mucus and epithelium in the apex becoming putrid from infection with septic germs, which may be said to develop more rapidly than the Bacillus Tuberculosis. What seems to me to be the most likely course of events, when from
impaired movements of some parts of the lungs, there is a tendency to an accumulation of secretion, so that the secreted matters will not be entirely retained but will be gradually removed as they are formed, perhaps slowly but yet sufficiently rapidly to prevent decomposition from development in them of putrefactive germs, let alone of the slow-growing Bacillus Tuberculosis. For we know that in the bronchial secretion, both in ordinary catarrhs and normally, there are found numerous Bacteria, but the secretion does not remain long enough for putrefaction to occur. Or if the matters are removed with difficulty, & accumulate & remain for some time, I would expect putrefaction to occur, when inflammatory changes would be set up, and we know the Bacillus Tuberculosis cannot grow in decomposing matters. Or perhaps the secretions in some cases, along with inflammatory products produced by their presence, may gradually lose their fluid parts & become caseous; in this last condition alone can I conceive that retained secretions can afford a suitable nidus for the Bacillus Tuberculosis.

I take what Jacquin calls the
functional inertia of the superior lobes: to mean the imperfect movements of these parts in respiration; a similar state of affairs occurs when there is pressure on a large bronchus, old pleuritic adhesions binding down the lung, or when there is imperfect development or formation of the chest.

It is true Bacilli entering such a part would probably be allowed to remain longer in the alveoli than in a part with good movements; but the Bacillus requires something more than rest—a suitable nidus is necessary before it can develop. And indeed I cannot than any such condition of "rest" will have any influence in favouring the development of the Bacillus when I consider that in every lung it gets all the rest it requires if it gains access to the tissues—an access which I have tried to show must very frequently be taking place without giving rise to Phthisis. As I have already said, this is all the rest it obtains in inhalation experiments on animals, & when it causes tubercles in man & animals in acute general tuberculosis. Let me ask if women are less liable to Phthisis of the apex than men, & they use the upper parts of the lungs
much more extensively than men.

I now come to Peter's statement that the Bacillus attacks the least living part of the lung. Green also seems to think there is some truth in this explanation. But such affections, occurring in people of delicate constitution with a hereditary history of phthisis, must have had the Bacillus in them over and over again and yet did not allow it to develop; as evidenced by Fagg's statement that brothers and sisters dying from phthisis frequently have an interval between their illnesses. The apex from the fact of its being the least living part of the lung will not therefore of itself, even in those with hereditary history, be sufficient to account for the localisation; it is quite possible however it may have some influence in leading to some other condition which will favour the settlement of the Bacillus, or it may favour the onset of the Bacillus when it has been altered by some accidental complication affecting it.

It has been said that simple inflammations or catarrh of the respiratory passages predispose to Bacillary Phthisis, by acting in some way so as to allow of the
settlement of the Bacillus. Now it cannot be that the secretions can have any influence one way or another, for usually they are removed so fast that putrefaction does not occur in them; unless indeed we could believe that these same secretions could be so altered and remain so long as to allow of the Bacillus, but not the putrefactive organisms, developing in them. Nor do I well see how catarrhs can affect the tissues, as to diminish their resisting power to the Bacillus in a somewhat similar way to that in which we would expect conditions lowering or depressing the general health would act—a state of health such as occurs in the serofulmin who are also liable to respiratory catarrhs. For them surely we would find that persons who have just recovered from an acute illness—a state especially from acute pneumonia when the tissues both locally and generally must be very much lowered in vitality—would be very frequent subjects of Bacillary Phthisis; but I am not aware that such is the case. Rather it is after or during the course of subacute or chronic affections of the lungs, when there
is the possibility of altered secretions being present which may afford a suitable nidus, that Bacillary Phthisis occurs. And if the Bacillus could act on cells whose vitality has been lowered by inflammation, surely Tubercle would not be found as such limited nodules, in which there are cells weakened by inflammation & usually Bacilli to act upon them, but it seems as if the Bacillus could not so act, certainly at least at the periphery, for new tubercles often develop there, by budding as it were, but the tubercle never increase individually beyond a certain size. We must however, when talking of the limited nature of a tubercle, be careful to discriminate between a caseating catarhal nodule & a tubercle; the former (often called “Tubercle”) certainly increases at its periphery, but we do not know that the Bacillus is the cause of the catarrh, while we do know the Bacillus is the cause of the Tubercle, which is quite limited & defined at the periphery. With reference to the state of the general health as being a condition favouring the attack of the Bacillus on the tissue, it is instructive to hear what
G. Lée (Bac. Phil. 5175) says:—that among families with family history or predisposition, there are some with good looks, good health & good hygiene who become subjects of Bacillary Phthisis, while a brother or a sister, with a miserable constitution exposed to all the dangers of the malady, escapes.

Another suggested favourable condition for the attack of the Bacillus is denudation of the epithelium of the lesser bronchi — by which it has been sought to partly account for the frequency of Bacillary Phthisis after measles. Whooping cough. It cannot be that thus the entrance into the tissues of the Bacillus is favoured to any extent whether by causing a "soo" on the mucous membrane or by causing the bronchial epithelial cells to lose their ciliary action so carbon & other particles enter the lung tissues without any such denudation. If such were really the case, surely attacks of simple catarrh affecting the lesser tubes would more frequently lead to Bacillary Phthisis than they do. And I cannot conceive of any other way by which such a denudation might have any influence in favouring the settlement of the Bacillus.
It has been suggested that possibly a diet with excess of Potash salts can so alter the tissues as to render them fit to be acted on by the Bacillus; this, or any other "chemical predisposition" of the tissues requires no further consideration, as there is no evidence for a against.

And now I cannot do better than quote Herman Weber (Seemann, 1861) who states that it is not yet clear why the Tubercle Bacillus thrives in some persons not in others, or why in some persons it thrives at one time and not at another; and Dr. Pagge whose words are that "Clinical observation is altogether opposed to the idea that infection from without is the most essential part of the Etiology of Phthisis." Green also states that "the chemical evidence of clinical experience is altogether against the contagious nature of Phthisis" (Brit med. J., 1883, Vol. 1, p. 193)

I have now done my best to show that we are not justified, from Koch's experiments, or from the similarity of the anatomical changes in general tuberculosis in Phthisical lungs, in concluding
that the Bacillus is necessarily the cause of all or part of the inflammatory lesions in Bacillary Phthisis. And I have not been able to satisfactorily explain why the Bacillus can affect some persons and not others, at one time and not another, or in other words to trace any causal relationship between the Bacillus and Bacillary Phthisis. I therefore conclude that there is not the least evidence to show that Bacilli Tuberculosis, as they enter the lungs under the ordinary conditions of life, ever induce Bacillary Phthisis (with or without tubercle).

What the significance of the Bacilli is, which are found so frequently in Phthisical disease, and the explanation I would give to account for their presence, I shall reserve for consideration in the following chapter.

**Chapter V**

It has been satisfactorily demonstrated that the Bacillus Tuberculosis when inoculated into the tissues, or inspired or swallowed experimentally, gives rise to an artificial tub-
exudation: that thus there has been introduced into the tissues some material or substance which has so acted in virtue of its irritant nature as to give rise to nodular hyperplastic growths (similar anatomically to those which result on injecting small irritating foreign bodies) and to more or less chronic inflammation with a great tendency to caseation. The important question is then presented to us: What is the virus that has been introduced and has been the cause of these changes? Is it the Bacilli themselves? or has there been something introduced along with the Bacilli, that we are unable to recognise? Which simply have been conveyed by the Bacilli from the original Bacilli-containing material, from which the cultivations were obtained? or is there some chemical substance produced by the Bacilli, which we are unable to recognise?

Dr. Greighten, in the discussion on Dr. Kidd's paper on the "Distribution of Bacilli Tuberculosi in Phthisis," expressed himself as not being sure that Koch's dry methods of cultivation were quite free from fallacy, inasmuch as it was possible for the Bacilli to be simple carriers of
some virus which existed in the original Bacilli-containing material. But after consideration of Watson Cheyne's description of his method of making cultures, similar to Koch's, I cannot conceive such to be the case. Beside, from pure cultures from tubercles of an artificially produced tuberculosis, other animals can be successfully inoculated.

If we are to look upon the Bacillus itself as the direct cause of the new growth, it is not easy to understand why Bacilli should be present in greater or less abundance in the tubercles of acute general tuberculosis, but not invariably according to Klein and other authors; indeed in the inoculation experiments of Klein and Gibbs already referred to, the resulting tubercles contained but few and in many instances no Bacilli. Klein also states that the "tuberculous deposits" in artificial tuberculosis stand in no relation to the number of Bacilli in them. And if the Bacilli alone acted on cells, how do they disappear after a time from the giant cells in Bovine Tuberculosis, as Koch says they do? They can hardly act mechanically as an irritant, for nonirritating foreign bodies.
in the tissues set up no irritation.

We must therefore suppose that there is some chemical substance produced by the Bacilli, which acts as an irritant to the tissues, giving rise to the tubercle or diffuse inflammation. This chemical substance may either be produced by the action of the Bacilli upon their food, in a somewhat similar way to that in which alcohol results from the action of yeast upon sugar, or it may be of the nature of an unorganised ferment evolved by the Bacilli. Admitting as I shall do that it is this chemical substance which acts as an irritant, I think we must also then admit that caseation is not due to the action of the Bacilli upon cells prepared for them by the chemical substance, but that it is due to simple nonspecific causes, since caseation takes place in tubercles in which there are no Bacilli. Granting that these statements are correct, the question is then introduced Can Bacilli tuberculosis utilise as food, from which they may evolve or produce their chemical substance, living tissues? I hold
that we have no reason for believing that they can do so; and by supposing that they can use as food only dead matter. I think that many things are explained which can be explained in no other way.

I shall here insert some of Klein's views with regard to the significance of the presence of septic organisms in diseased tissues in the living body; and I shall then endeavor to show that a somewhat similar theory accounts for the presence of Bacilli Tuberculosis in the living body in certain lesions, and also explains certain facts in regard to inoculation and infection.

Klein (Minor, 1st Ed., p. 234) states that it is not at all uncommon to find organisms in tissues which, during the life of the subject, have become dead, or so severely injured by inflammation or otherwise that they may be considered practically dead; and that these organisms bear no intimate relationship to disease, merely finding in the dead and severely diseased tissues a suitable nidus for their growth and development.

He also says that the spread of purulent
inflammation in connective tissues, in parenchymatous organs is often, if microorganisms are present in the original focus, associated with corresponding spreading of the microorganisms, but whether this spreading of the microorganisms is merely concomitant with or subsequent to the spreading of the inflammation or whether it is the primary cause, requires definite experimental proof. Then again he states that it is not difficult to explain that if a focus or necrosis be set up at various internal places in consequence of emboli carried from an inflammatory focus to which microorganisms have access from the outer world, these internal places or metastases would harbour the same microorganisms, and as soon as disintegration — abscess caseation or necrosis — took place in these metastases, also the imported organisms would multiply to a great extent the tissue being shut out from the circulation and practically dead.

Can we not then understand how it may be the same with Bacilli tuberculosis — with this difference, however, that the Bacilli produce a chemical substance
which acts in the same way as the emboli in so far as it causes the tissues to become practically dead by setting up an inflammation in which easeous degeneration takes place; and can we not understand that it is of the power of forming this chemical substance that Bacilli Tuberculosis, when once they have gained a footing in the living body, are able to develop & spread from place to place causing a local or general tuberculosis, or in other words to be locally or generally infective

Let me now apply this theory to various points in Bacillary Tuberculosis & show how it satisfactorily explains these points.

A Tubercle occurs as a module limited in size: it is produced by the irritant action of the Bacillary chemical substance. Because the Bacilli cannot grow in living tissues, the tubercle does get beyond a certain size; but their chemical substance, not being a powerful diffusible irritant, acts locally & gives rise to a circumscribed inflammatory module or tubercle. When caseation occurs in the centre of the tubercle, the Bacilli
Infect grow in the dead matter and produce some chemical substance, which passes slowly outwards into the neighbouring tissues, giving rise to further inflammation and tubercles.

Experimental inhalation of Bacilli-containing material always induces in rabbits tuberculosis of the lungs; rabbits are very susceptible to the disease. Then how is it they ever escape infection of the lungs in a laboratory where experiments are being made with the Bacilli, since the Bacilli or their spores must be floating about in the atmosphere? The only way I can explain this is by supposing there is something not introduced in the latter case along with the Bac. or their spores, which is introduced in the former case and prepares the tissues for the action of the Bacilli—this something is the chemical substance, which must have been inhaled along with the Bacilli or must have been produced from food (dead matter) inhaled at the same time in which the spores developed when they obtained a suitable temperature and moisture. This explanation holds good whether fresh or dried material...
is used for the inhalation experiments — that is to say that the Bacilli or their spores cannot be separated experimentally from the chemical substance of their food. That spores do float about in a laboratory seems to be evident when we consider how Cohnheim inoculated simple nonbacillary bodies and yet produced an artificial Tuberculosis; it could not well have been through his instruments carrying the Bacilli from other Bacillary matters for simple nonbacillary bodies did not produce Tuberculosis at his own house. And Klein gives two instances in which he inoculated rabbits with a culture of bacilli anthracy, which he did not expect to produce anthrax; instruments never used before were employed but the animals became tuberculous; some other inoculation had been going on on the same day with bacillary material, so that the Bacilli Tuberculosis must have reached the inoculation wound through the atmosphere (June 8, 1878).

And does not the theory that the Bacilli can only grow in dead matter explain why so many persons such as physicians
attendants on consumptives whether predisposed or not, frequently inhale the Bacilli & yet do not allow settlement; because the Bacilli have been introduced without their food & without their chemical substance & because they have not obtained food in the lungs ready provided for them.

Let me now describe what I suppose to take place in inoculation experiments. When a portion of Bacillary material is inoculated into the tissues, there is introduced at the same time, some of the chemical substance or some food, or perhaps the food may actually be provided by the inoculation wound depriving some cells of their vitality. The Bacilli are thus able to grow for they have dead matter to feed on, and they produce a quantity of their chemical substance which passes into the neighbouring tissues, sets up irritation & a tubercle results, which tubercle may contain some Bacilli which have passed through the tissues along with the chemical substance: indeed the frequency with which Bacilli are found
in tubercles would seem to show that they act as carriers of their chemical substance in the tissues. Besides the tubercle, there may also be some diffused inflammation from the passage of the irritating chemical substance through the tissues. From the peculiar nature of the inflammatory nodule and the diffuse inflammation, caseation is very apt to ensue, thus providing a further supply of food for the Bacilli which are present, and a further quantity of chemical substance is formed. This passes into the neighbouring tissues, sometimes with sometimes without accompanying Bacilli, and tubercles and inflammation are again induced. So the process may continue extending further and further from its starting point till finally perhaps some of the Bacilli with their chemical substance gain entrance into the blood-vessels, with the result of inducing a general tuberculosis from the virus being conveyed to various organs or parts of the body.

From what I have said, it can readily be inferred what I believe to take place when the Bacilli reach the lungs.
of an individual. Bacilli - or more frequently their spores - are often inhaled; many are immediately exhaled while others reach the alveoli & intralobular bronchi and are removed by expiration after having made a longer or shorter stay. Others pass into the lymphatics & tissues, and possibly are there destroyed, or they may be carried to the bronchial glands & to the blood to be finally removed in the urine or other eliminatory secretion. Such at least must be the course of events in those who do not become Phthisical, and the Bacilli have not obtained a settlement either because they were introduced without their chemical substance or their food, or because they did not reach soil which would supply them with food.

So that the only circumstances under which I can conceive that Bacilli could develop on reaching a lung & remaining in contact with living tissue, are that, as in inhalation experiments, they should carry along with them either some of their chemical substance or some food from which they might form it. If the chemical substance
& the Bacilli should pass into the tissues.

A tubercle or local inflammation would result; if they remained in the alveoli, a tuberculous pneumonia or fibrous would result; in either instance when caseation occurred the process would be liable to extend indefinitely. But that the introduction into the lung, not only of the Bacillus Tuberculosis but also of its chemical substance or food, takes place but very seldom or never must be evident when it is considered that there are many people who are almost constantly exposed to the possibilities of such an infection & yet escape. It is not impossible however that it may occur occasionally; & this may perhaps be the true explanation of some of the cases of apparent infection, such as have been recorded in the report of the Collective Investigation Committee of the Brit. Med. Assn. and in H. Weber's series of cases.

Thus since Bacilli are found in certain diseases of the lung, we must admit that they have obtained a soil suitable for their settlement. Let the Bac.
illi or their spores gain access, let them remain in contact with dead matter which will allow of their development, & they will flourish. And where can they find a more suitable soil than dead caseous matter, the result of previous pathological processes? Hence it is that Bacilli are found in such enormous masses in the walls of some phthisical cavities. Let the caseous matter soften, the Bacilli & their chemical substance are absorbed & induce a tubercular inflammation. And an inflammation truly Bacillary in origin is liable to be set up whenever caseous matter has become infected with Bacilli, apart from the inflammation induced by absorption: for the softened caseous debris, by the force of gravitation & from the respiratory movements, tends to be carried to other parts of the lung or lungs & there set up a catarrhal or fibrinous inflammation, from the irritant action of the Bacillary chemical substance. Partly in this way do I account for the liability of Phthisical inflammatory lesions to extend, often by fits & starts, and for the frequency with which an acute
Pulmonary Tuberculosis being used synonymously with Bacillary Phthisis, for, apart from the fact that Tubercles may not be present in the latter, we are not able to decide whether a lobular pneumonia, resulting in caseation with Bacilli in the necrotic masses, with the accompanying interstitial changes, is partly or not at all due to the action of the Bacilli. I would however point out that these views do not detract from the value of Bacilli in the sputum being...
important evidence that the subject is affected with Tuberculosis; for their presence shows that they have found suitable food for their development; therefore that caseation is present, as well as the fact that such persons, if not already tuberculous, are very liable to become so at any moment, either locally or generally.

Chapter vi

There is no doubt as I have already mentioned that certain individuals have a predisposition to phthisical disease, which may be either hereditary or acquired. I have been unable to explain this liability to the disease by supposing that the living tissues can be so constituted or modified as to afford a place of settlement for the Bacillus Tuberculosis. I hold that the real causes of this predisposition are to be found in the tendency to caseation of inflammatory products and in the tendency to cætarrh of the air passages.

Cornelius Ranvier quoting Foster define Caseous Inflammation as follows in
their Manual of Pathology. In this the products of inflammation are not eliminated. They become atrophied, broken up, filled with fine fatty granules, destruction of the part affected being the result. The lungs and the lymphatic glands are the usual seat of this inflammation, but it may occur in all the organs. The essential condition of its production is that the infiltrated pus should remain long enough in such abundance in an organ, that the vessels are compressed by the inflammatory elements. The same result occurs when vessels are themselves the seat of endarteritis— to put it in as few words as possible, when the blood supply of a part is gradually cut off by accumulation of inflammatory products or from endarteritis or I would add—from any other cause, caseation ensues in the part. I may as well state here that I believe the reason of tubercles caseating is their being extravascularly; consequently the new growth is not sufficiently nourished; especially in acute general tuberculosis does caseation occur early, since the mode of formation of the tubercles necessitates...
the early destruction of the bloodvessels. And in the diffuse inflammation induced by Bacilli, caseation occurs from the inflammatory products gradually interfering with the blood supply.

In the lungs, when a catarrhal or fibrinous inflammation has occurred, the inflammatory products degenerate and become fatty, and very frequently are removed by expectoration and absorption. But sometimes they remain so long and in such abundance in the affected part that the blood supply is gradually cut off and caseous degeneration ensues. Thus any condition tending to prevent or hinder the removal of the inflammatory products and any condition which will allow the blood supply to be more easily interfered with than in ordinary circumstances, will favour the occurrence of caseation. Hence the liability in debilitated individuals, especially those of a scrofulous constitution, for inflammation to assume a subacute or chronic form, is an important factor in rendering them more liable to caseation of the inflammatory products.
for when an inflammation is slow & insidious in its origin, there is a tendency for the exudate to contain little fluid & to be removed with difficulty. It is also easily understood that, in these same debilitated individuals, the bloodpropelling power of the heart is more or less impaired, thus allowing of interference with the bloodcirculation in a part to take place more readily; and that in them the coughing or forced expiratory efforts are weakened or impaired, thus rendering the removal of inflammatory products more difficult, especially of catarrhal pneumatic products which are naturally rather viscid & adhesive from the quantity of mucin they contain. Indeed the forced expiratory efforts, not being successful in removing the inflammatory products in the alveoli & lesser bronchi, will tend to still further insipissate them by the increased pressure exerted by the efforts driving the liquid part into the absorbents of the lung. Thus the great & predisposing cause of cæsation of inflammatory products in the lungs is debility of the individual,
either inherited or acquired; and it is on this account that we meet with caesation in the lungs so frequently in children, whose blood-propelling power of the heart, whose muscular system are not very powerful at the best of times.

But there is another predisposing cause of phthisis: for it is evident that any condition of the lungs or any state of the constitution, which renders an individual more liable to suffer from respiratory catarrh, will supply an important predisposing element by furnishing the inflammatory product which may cause it. Many people are subject to respiratory catarrhs on slight exposure to cold, and this susceptibility seems to be inherited in many cases. Phthisis is common in low-lying damp badly-drained situations, according to the report of Dr. Buchanan: this frequency of the disease in such places is probably to be explained by an increased susceptibility to the influence of cold on the respiratory passages being produced; perhaps because
a "germinal" condition of the alveolar epithelium is produced similar to what is found normally in children (Hamilton) who are subject to cataracts.

Those individuals therefore who from hereditary or acquired causes are liable to respiratory exanthems, who are more or less debilitated are specially liable to become the subjects of Phthisis.

I insert here an account of some experiments on animals by Cornel & Ranvier (Path. and Vol. 44) which seem to me to indicate that the above explanation of caseous inflammation in the lung is the true one. They say that the resolution of pneumonia, the result of injection of solid substances suspended in fluid, of which the lung frees itself with difficulty, is always slow; and, especially in the rabbit, a condition is induced which to the naked eye appears like caseous pneumonia. When the two recurrent laryngeal nerves are divided in the rabbit, thus facilitating the entrance of foreign bodies, such as buccal mucus and fragments of alimentary matter, into the
larynx & bronchi, a slow & insidious bronchitis & pneumonia are induced; some weeks after the operation a more or less complete lobar or lobular hepatisation will be found at the autopsy, & the surface of a section of the diseased parts shows a yellowish or grey colour resembling that of caseous hepatisation in man—a condition which is exactly similar to caseous pneumonia in "tuberculosis."

These experiments seem to me to show that in the rabbit a subacute pneumonia, in which the inflammatory products are removed with difficulty, leads to caseous degeneration: and therefore that in man similar conditions would in all probability tend to induce caseation.

As I have now given the agencies which I believe to be instrumental in inducing caseation, I am in a position to state what I believe to be the cause or causes of the localization of Phthisis at the apex in so many cases.

According to Hamilton the chief cause is the natural dryness of that part of the lung; for when products of inflam.
are present the fluid parts tend to gravitate & drain off, and to sink to the lower parts of the lung. Thus aiding aiding the removal of inflammatory products there by rendering them more fluid, but tending to hinder their removal from the upper parts by rendering them more viscous & insipid, & more apt to dry up and become caseous.

Another cause is said to be the imperfect movements of the upper parts of the lung. In ordinary respiration, the respiratory movement can have little or no effect in assisting or retarding the removal of accumulated secretions or inflammatory products, except in so far as expiration will pass alveolar contents on to the bronchi; when the cilia of the bronchial epithelium and forced expiratory efforts will be made to come into play. In ordinary respiration, inspiration neutralizes any effect expiration has of itself in expelling inflammatory products or secretions, but it is far different when forced expiratory efforts are made. In coughing or deep inspiration is taken, which tends to draw any matter present in the bronchi still deeper into the lung— then the glottis
is closed preparatory to a very sudden & forcible expiratory blast. Such an expiratory effort must not only return matter to where they were before inspiration was made, but from its sudden & forcible nature must tend to propel them into the larger bronchi, to be finally expectorated. But at the apex, with its slight expansile capacity, both in women & men, the forced expiratory efforts will act to a less degree than in other parts of the lung, & thus will have less effect in bringing about the removal of inflammatory products contained in the alveoli & small bronchi. Deformities of the chest, pleuritic adhesion, bending down the lung, & pressure on a bronchus, will each in this way be favouring cessation of inflammatory products.

Another agency tending to induce cessation at the apex of the lung is the greater ease with which the circulation in the part will be interfered with than elsewhere, by an accumulation of inflammatory products in the alveoli & lesser bronchi. This is due to the fact that the apex is what
Peter calls the least living part of the lung, where the circulation is the most sluggish.

Chapter VII

I have now described the structural lesions occurring in Phthisical lungs, and have endeavoured to show why certain individuals & certain parts of the lungs are predisposed to cavitation & destruction of lung tissue, which, by affording a supply of foodstuff, allow the Bacillus Tuberculosis to settle & grow, thus rendering the subject liable to tuberculosis local & general. I intend in the present chapter to briefly refer to the different varieties of Phthisis & to trace the relations between non-tubercular, tubercular, & tubercular Phthisis.

Douglas Bowells classification of Phthisis is the following (see pp. 129 & 29): —

Pneumonic Phthisis

1. alveolar nature
2. Catarhal Phthisis
3. Acute Pneumonic Phthisis
4. Chronic "
5. Fibroid Phthisis
Tubercular Phthisis
(1) acute
(2) chronic
(3) acute tuberculous
Other varieties separable for clinical convenience
(4) Bronchial tubercular Phthisis
(5) Diabetic Phthisis
(6) Dust Phthisis
(7) Abdominal Phthisis
(8) Laryngeal Phthisis
(9) Syphilitic disease of the lung
(10) Syphilitic Phthisis

In my opinion this classification requires some amendment; for we are not justified in regarding an "alveolar cataract" of the apex or other part of the lung as "Phthisis," unless there is caseation and destruction of lung tissue. And I am unable to see why "catarrhal phthisis" should be classified as a separate form, for both in acute and chronic pneumatic phthisis this is frequently the prevailing lesion: it is quite possible however that in some cases of what would clinically be called Phthisis catarrhal products alone have ascended, but such must very rarely happen and...
practically the lung tissue is always involved in the degenerative process more or less. I also would exclude Acute Tuberculosis when merely a part of acute general Tuberculosis for it is then rather a manifestation of a general disease than a disease of the lungs. The varieties which he separates for clinical convenience really belong to some one or other of the other forms, they are worthy of special names only in so far as this is shown some prominent clinical or pathological feature. Abdominal & Laryngeal Phthisis are of course not diseases of the lungs. Phthisis is used by Powell as being a disease which may occur in any organ. Diabetic Phthisis is Phthisis occurring in a diabetic individual; such persons seem peculiarly liable to suffer from Phthisis, perhaps because Diabetes is such a chronic & debilitating disease, and so favours of cessation in inflammatory products.

I will therefore classify Phthisis as follows, though it must be borne in mind that there is not a hard & fast line to be drawn between the different varieties, one passing by insensible gradations.
into another.

**Nontubercular or Pneumonic Phthisis**

1. Acute Pneumonic Phthisis
2. Chronic " "
3. Tubercular Phthisis

**Tubercular Phthisis**

1. Acute Tubercular Pneumonic Phthisis
2. Chronic " "
3. Tubercular fibroid Phthisis

The confluent pneumonic form of tubercle resembles acute pneumonia of the alveoli in the suddenness of its onset, the acuteness of the symptoms, and the extent of the consolidation. Other cases do not begin so acutely, and the consolidation may not be so extensive but it soon advances with rapidity.

The chief anatomical change consists in a pneumonic condition, either chiefly exudative or not infrequently consisting in great part of fibrinous exudation: the two are very often combined. Resolution and removal of the inflammatory products may take place more or less extensively. But there are areas in which such removal does not occur: caseation ensues, "breaking down" and softening usually follows with the formation of cavities. The disease is usually
rapidly fatal, perhaps by extension.

Bacilli usually gain access to the caseous masses, and the Phthisis becomes Bacillary.

If now some of the Bacilli & their chemical substance are absorbed in tubercular inflammation
with tubercles result, converting the case into one of acute Tuberculo Pneumonic Phthisis.

A favorable result may occur in the non
Tubercular form, by the caseous masses being
removed after they have softened: and the
the greater the strength of the patient, the more
likelihood is there, from the strength of his
forced expiratory efforts, of his getting rid of
the debris before infection of the tissues by the
Bacilli has occurred. But there is always the
great risk present that some of the caseous
matter containing the Bacilli may be carried
to other parts of the lung or to the other lung
and there set up further inflammation.

The Post Mortem lesions of dis-
seminated acute Pneumonic Phthisis are very
similar in their nature to those found in
the confluent form: the affected areas however
are small in size & scattered over the lung,
simulating in the form of distribution acute
disseminated broncho-pneumonia from which the disease differs essentially, the fact that the affected areas have gone on to caseation and destruction of living tissue. Bacilli may obtain settlement, and then the disease may be converted into a Tuberculo Pneumonic Pithesis. The mistake is frequently made, more especially when a secondary acute general tuberculosclerosis has been produced, of looking upon the caseous pneumonic nodules as Tubercles, and they are often described as miliary Tubercles; they are however easily distinguished microscopically by their structure. The course of the disease is similar to that of the confluent form, with the differences resulting from the different distribution. A possible explanation of the wide and scattered distribution is that the inflammatory products in the alveoli have arisen to a large extent from the bronchi; the patient often a child, inspires these into the alveoli but is unable to dislodge them from the weakened state of the forced expiratory efforts. There is then set up some irritation in the alveoli, catarrh ensues, and caseation takes place in the inflammatory products derived alveoli & bronchi. That bronchial inflammatory products do reach
the alveoli is proved by finding the bronchial columnariliated epithelial cells in the alveoli in some cases of bronchopneumonia. Hamilton has seen a similar disseminated distribution take place of blood which had been effused into a primary bronchus of the lung.

**Chronic Pneumonic Phthisis.** occurs very frequently, and often begins with a bronchial cataract which extends to the alveoli to a greater or less extent. The cataract is recovered from in some parts but not completely throughout the lung; some inflammatory products remain long enough in such abundance in the alveoli & bronchioles for impairment of the circulation in the affected parts to take place, and cessation of destructive of lung tissue results. Infection of the caseous matter with Bacilli frequently occurs, and perhaps absorption of the Bacilli & their chemical substance takes place, and the production of Chronic Tuberculous Pneumonic Phthisis, is the result.

Recovery may occur before the tissues have become Tubercular, either before or after infection of the caseous masses with the Bacilli by the caseous debris being removed by expectoration & cicatricial changes taking place in the walls
of the cavity; or by the caseous mass becoming encapsulated & calcareous.

The most frequent course of events however is for the pneumonic & caseous changes to gradually extend & to appear in other parts of the lungs. The fresh inflammatory processes seem frequently to be induced by repeated exposures to cold, to the influence of which the lungs are apt to become more susceptible the more it gets involved. From the blood circulation being interfered with in certain parts & thus a greater strain thrown upon the still precious blood vessels. It is for this reason that pleurisy is so common: and that haemorrhage, or fibrinous exudation into the alveoli may take place on any slight sudden exertion. The extension of the disease is also largely due to the Bacteary caseous debris being carried to other parts of the lungs & there setting up inflammation. There is always more or less fibrosis or increase of connective tissue from the reactive inflammation set up by the pneumonic protoplasm in the neighbouring tissues. In the tubercular form, the tubercular inflammation will also aid in the fibrosis. There is always also a more or less general bronchitis, or perhaps limitted
So these bronchi in connection with the diseased areas this bronchiitis may have been present from the first or may have been induced by the passage over the bronchi of irritating maters. More or less peribronchitis occurs along with the bronchitis a fibrosis is produced round the bronchi extending into the neighbouring fibrous tissue aiding to increase the general fibrosis of the organ. In the bronchial walls tubercles are often seen but all cellular nodules in the bronchial walls are not tubercles for they are often the peribronchial lymphatic structures which normally are found there in a state of inflammation.

Fibroid Phthisis - a term introduced by Dr A Clark - is characterized by the great increase which takes place in the fibrous tissue of the lungs. There are present also bronchiectasis with in addition some cheessymasses in the lung tissue and usually some small cavities. It is a very chronic disease and occurs secondarily to chronic bronchitis, chronic pleurisy, chronic pneumonia or other chronic disease of the lung. A local fibrosis may result from injury to the lung, or from impaction of a foreign body in one of the smaller bronchi; it may ultimately lead to phthisis. The presence of
Caseous pneumonia masses, when not the cause of the fibrosis, is an accidental complication, is no more than we would expect to find in a lung whose capacity for the removal of inflammatory products must be diminished by the fibrosis present and whose blood-circulation must be interfered with to a greater or less extent. When caseous pneumonia has once supervened, it aids in increasing the fibrosis, affecting especially the walls of the alveoli and intralobular bronchi.

Caseous masses are frequently present in the dilated bronchi, more especially in the apex. The reasons for this caseation taking place in the bronchial inflammatory products are much the same as those which lead to caseation of inflammatory products in the alveoli—difficulty of removal and draining off of the fluid parts leading to inspissation and drying up. In the base of the lung the secretions of dilated bronchi often become putrid, as their fluid parts do not drain off.

Caseous masses are also formed by obliteration of bloodvessels by endarteritis obliterans. When fibroid phthisis has lasted for some time, it usually becomes tuber-
cular (Tuberculo-fibroid Phthisis) from Bacilli 4  
their chemical substance being absorbed by the  
brines from infected caseous masses in the  
lung or dilated bronchi. These tubercles do not  
tend to caseate rapidly, and, with or without  
caseation in the centre, they are frequently con-  
verted into a little fibrous thickening. Hence  
when great numbers of these fibroid tubercles  
are present, they materially aid in increasing  
the general fibrosis.

Under the head of fibroid phthisis I  
include dust phthisis & syphilitic phthisis, both  
of which are characterised by the preponderating  
lesion being a great increase of fibrous tissue;  
in both tubercles are liable to occur during  
some time in their progress.

Green & others have attempted to make  
out that the smallcelled infiltration of the  
alveolar walls is different from that which  
occurs in the interlobular septa & other fibrous  
tissues of the lung. There is, as far as I can  
judge, no reason for such a statement; for  
the alveolar smallcelled infiltration is nothing  
more than a young stage in the develop-  
ment of fibrous tissue, and we can see
are exactly similar condition in the interlobular septa where the process is most recent. This alveolar small-celled infiltration frequently develop into cicatricial tissue & often leads to obliteration of the alveoli.

Chapter VIII

Are there any indications for treatment afforded by the views given in the preceding chapters of the nature & origin of emphysema? for it is of such an important end that all medical enquiries aim ultimately to arrive. It seems to me that *prophylactic* treatment specially is indicated in the following directions (1) To maintain the general health at par, as far as it is lies, by general hygiene: thus avoiding impairment of the blood-propelling power of the heart, & debility of the respiratory muscles. Respiratory gymnastics will be specially indicated in some cases (2) To avoid exposure to cold & wet, or any irritant that may induce an inflammatory state of the respiratory passages: or to remove an individual from any condition or situation...
which renders him more liable to suffer from such inflammations (3) To endeavour in all cataracts & inflammations of the lungs, especially in debilitated people & in children, to prevent the affection becoming chronic (4) To prevent as far as possible the exposure of any individual who is suffering from an inflammatory affection of the lungs to the possibility of infection of the inflammatory products with the Bacillus Tuberculosis. It is only in the earliest stages of Phthisis, before Bacilli have gained a settlement, that I can imagine that antiseptic inhalations (and especially by constantly worn inhalers for residence in an aseptic atmosphere, will have any influence upon the development of Bacillary Phthisis, and that by preventing the Bacilli gaining access to dead matter which will afford them a suitable nidus.

I believe the beneficial influence of high altitude climates such as Davos St. Moritz Colorado &c. to be chiefly owing to their general fortifying & invigorating effect upon the body; also to the fact that the respiratory muscles tend to become well developed from
& the lungs to expand well, from the mode of life engendered by living in these parts, perhaps also from the action of the rarefied air.

The Riviera, Madeira, & other mild winter residences I believe to act specially in a beneficial manner by removing the tendency to respiratory & catarrh; and to a certain extent by improving the general nutrition but not so markedly as high climates.

Each kind of climate is indicated for special classes of cases.

Chapter IX

Let me say a few words as to the significance of the presence of Bacilli Tuberculosis or certain other pathological processes, than Phthisis & I am done.

When a child suffers from some slight irritation, as a carious tooth or an eczema, one or more of the lymphatic glands in the neighbourhood frequently enlarges. If the source of irritation be removed, the gland may return to its normal condition. Sometimes however it remains enlarged, becomes caseous & finally may soften.
On examining such a gland there are found inflammatory & cæsion changes, & in a considerable number of case tubercles. Bacilli Tuberculosis are frequently found whether tubercles are present or not, but not always.

Are we to believe that Bacilli are the cause of all these lesions in a gland? They cannot well have been the cause of the enlargement, which perhaps lasted for a very considerable time, & then disappeared; but it may be said that Bacilli, if they found entrance to such an inflamed gland, would be able to act on the weakened tissues. Surely if such were the case the Bacilli would also show traces of their presence, similar to those in the lymphatic glands, in the lymphatic vessels by which they gained entrance & which are also in a weakened state. Rindfleisch (Einf. Pfl. p. 231) admits the possibility of simple serofolous inflammations & then because Bacilli are present in the secondary products of inflam (cæsion gland) he says the Bacilli are the cause of this inflammation & cæsion. He also states that there is a frequent commingling of the phenomena of serofolous & of local Tuberculosis in boxes & brain kidneys &c. To me it is most reasonable to suppose that the cause of event has been that the simply inflamed gland did not recover, but that
from the slow & increasing accumulation of inflammatory products, caseation took place to a greater or less extent, that Bacilli gained access to the caseation matter in which it found suitable food & developed. And then that the scrofulous & tubercular phenomena became con-

mingled.

Scrofulous disease of a joint in which tubercles & Bacilli are present, may continue for years. Amputation is performed & the patient makes a good recovery. How is this to be explained or other than the specific origin of the disease? To me the course of events seems to be that a person receives a slight injury at or near a joint; a healthy person suffers from no ill effects, for either no inflammation ensues, or the inflammatory products are soon removed or organs. But in a "scrofulous" person— that is one with a "frailty" or "vulnerability" of the tissues to slight injuries, so that an inflammation is set up which tends to become chronic & in which there is no tendency to absorption or organisation but rather to accumulation of the inflammatory products— the resulting inflammatory products are slowly & gradually formed & are with difficulty removed; and caseation ensues from the gradual interference with the blood supple.

Bacilli tuberculosus reach this caseous mass, prob-
ably by means of the blood & obtain a settlement; there is then a commingling of the scrofulous & the tubercular phenomena. Amputation is performed; here there are tissues which must be enflamed & weakened; but the inflammatory products are removed as they are formed by free drainage from the surface of the wound; hence there will be no accumulation in the tissues & caseation will not ensue, and the Dauelli Tuberculi will not obtain a nucleus for developing in; and the parts heal.

I conclude with a list of the works I have consulted, on which I have drawn for many of the foregoing statements. I have not always been able when quoting an author to give his exact words; this has been owing to my not having been in a position at the time of writing to refer to the works in question. I believe however that all quotations & references are pretty accurate as my plan has been when reading any work to take copious notes as I went along for future reference. The list is the following—

Brissot's Practice of Medicine; Fauconer's Practice of Medicine; Walsh's Diseases of the Chest; J. Henry Bennett's Treatment of Consumption; J. Hughes Bennett
(Phthisis; Reynolds' Syr: met; Hamilton's Branch): 
Coates, Manual of Pathology; Woodhead, Practical Pathology; 
Woodhead & Mar's Pathological Myology; Jones, Summing's 
Pathological Anatomy; Lancer, Diseases of the Eye; Colin- 
hein's Insane. Diseases of Tuberculosis; Cullenon's Treat- 
ment of Phthisis; Ingle's Pathological Anatomy (1842); 
Rinse, Lenz's Elements of Pathology; James's Treatment 
of Phthisis; Germain & C.'s Pulmonary Phthisis; CPhrys 
Manuel's Pathographical Histology (Vol 19, 1889); Beog's 
Diseases of Lung, & Pleura; Hunter, Medical Surgery. 
Hassell, Inhalation Treatment of Respir. Organs; Delafield 
& Prudden's Pathological Anatomy & Histology; Herman, Weller's 
Lectures on the Treatment of Phthisis; Greens, Pathology 
Mark's Anatomy; Fagg's Practice & Principles of Medicine; 
Klein's Microorganisms & Disease; Gradle's Thrombus & Gumm 
Theory of Disease; and Medical Journals.