"The consumptive houses" in which people go to live and become infected, whose existence has been beautifully demonstrated by Sir Robert Phillip of Edinburgh, should be destroyed for two reasons. Firstly, such houses are frequently insanitary on many grounds. They are often the refuge of those whose lack of means renders resistance impossible of attainment. Secondly, as nests of bacilli they convey large doses of infection to their non-resistant inhabitants.

A second great use of the tuberculosis dispensary is that of reaching the various classes of disease and sorting them out for appropriate treatment. Without their first function, that of acting as centres of instruction, they cannot hope to catch a large number of cases in the earliest stages. Early application for treatment implies knowledge. From what I have said, you will have gathered that nowadays we are curing a great number of cases that formerly got well without treatment. But I think also we are curing a great number of cases that without our help would have utterly lost their resistance. The tuberculosis dispensary used intelligently will form a net to catch a considerable number of the latter class. It will send those, in whom it is believed that resistance can be regained, to the sanatorium; it will enable those too ill for the sanatorium to receive appropriate treatment at home or in the hospital; it will distribute the means of protection and disinfection to those watching the death-bed of the advanced case, and it will provide some of these dying people with gentle and kindly care in special institutions until the end.

HILUS TUBERCULOSIS ("ROOT PHTHISIS").

By R. MURRAY LESLIE,

M.A., B.SC., M.D.,

Physician to the Royal Hospital for Diseases of the Chest, London; Physician to the Prince of Wales's General Hospital.

Introduction.

THE region of the *hilus*, or root, of the lung has long been recognized as a common site of tuberculous deposit, more particularly in connection with the tracheo-bronchial lymphatic glands, but the immense importance of the hilus of the lung as an early and primary focus of pulmonary disease is only just beginning to be appreciated. The frequency with which the bronchial and mediastinal glands become affected may be readily understood when we recall the fact that these glands are the natural filters or sieves to which pass foreign particles, including tubercle bacilli, deposited on the tracheal and bronchial

mucous membranes or on the epithelial surface of the alveoli. These glands are, indeed, as Sir Douglas Powell well expresses it, "the dust-bins of the broncho-pulmonary tracts." The pleural lymphatics also drain into the glands at the root of the lung. The bronchial glands are also directly connected with the cervical and abdominal lymphatic systems, as has been definitely proved by Grober, Poirier,¹ and other observers.

In view of the above considerations, it is not surprising that the thoracic lymphatic glands should be frequently the seat of disease. So extraordinarily common, indeed, are tuberculous foci in the bronchial glands and surrounding lung-tissue, that the majority of all bodies examined post mortem, whatever the cause of death, present distinct evidence of old-standing or recent disease in these parts. Even when there are no tuberculous foci visible to the naked eye or on microscopic examination, the intra-peritoneal injection of the gland-tissue in guinea-pigs is usually followed by general tuberculosis. Naegeli found evidence of tuberculosis in no less than 97 per cent. of all adult post-mortem examinations, and in a large proportion of these the disease took the form of calcareous or caseous deposits in the glands situated either at the roots of the lungs or along the large air-tubes. The bronchial glands, indeed, are often the first tissues attacked by tuberculosis in early life, and may be the only foci of disease in the whole body.

Professor Nietner,² of Berlin, is of opinion that tuberculous deposit often commences in the second year of life, and continues in an increasing ratio up to and beyond the age of puberty. K. Preisich,³ as the result of his investigations on the frequency of tuberculosis in childhood, makes the following statement, which indicates the importance of peribronchial and hilus tuberculosis in children: "In children over eight years of age chronic tuberculosis of the lungs tending to recovery is no longer rare. Most frequently there are found peribronchially disseminated, lobular or confluent foci. *There is also caseous infiltration starting from the hilus and spreading over an entire lobe (probably starting from the hilus glands and spreading back along the lymphatics).*"

In England the radiographic investigations of Dr. Alfred Jordan,⁴ published under the title "Peri-bronchial Phthisis," have attracted widespread attention. He states that the great majority of a series of "healthy" lungs obtained by him from the post-mortem room and examined by the X rays showed calcareous deposits in the lymphatic glands, either at the roots or along the course of some of the larger air-

¹ Poirier: "Traité d'Anatomie Humaine," Paris, 1902.

² Nietner: *Lancet* November 16, 1912.

³ Preisich: *Wiener Medizinische Wochenschrift*, 1911, Nos. 3 and 5.

⁴ Jordan: *Practitioner*, February, 1912, and *British Medical Journal*, August, 1912.

tubes. On the other hand, Dr. Horton-Smith-Hartley¹ has recently stated that, whatever may be true of children, his pathological experience has shown that in the case of adults dying from chronic pulmonary tuberculosis caseation of the bronchial glands is the exception rather than the rule. In 263 post-mortem examinations which he has conducted at the Brompton Hospital, in only 9·1 per cent. were the bronchial glands found caseous. Dr. Hartley probably refers to readily visible gross changes, such as definite softening and enlargement. Careful dissection is often necessary to expose to view the smaller indurated, caseous or calcareous glands situated on the bronchi and below the tracheal bifurcation, where they are often concealed by the over-lying hilus tissue, though during life they may have stood out clearly in a radiogram as definite rounded dark shadows.

Fortunately, there is a marked tendency to spontaneous cure in the case of glandular tuberculosis, as is evidenced by the frequency with which such calcified bronchial glands are met with post mortem in cases where there had been no suspicion during life of any tuberculous disease. At the same time, it must not be forgotten that the disease may lie latent in the glands for long periods, and may be followed many years later by important and serious consequences.

As regards the incidence of the disease, evidence is accumulating that catarrh of the bronchial mucous membrane is one of the most important predisposing causal factors in connection with hilus tuberculosis. Sir William Osler in this way explains the frequent occurrence of tuberculosis *adenitis of the bronchial glands as a sequela of whooping-cough and measles*. Increased vulnerability of the tissue, according to the late Professor Virchow, is the important factor in producing the disease, and such vulnerability would naturally occur as the result of injury or breach of continuity in connection with the bronchial mucosa.

The Primary Site of Chronic Pulmonary Tuberculosis : Apex versus Hilus.

Hitherto the apex has always been regarded as the most frequent primary focus of pulmonary tuberculosis. In 1880 Sir James Kingston Fowler² stated that a spot situated 1 to 1½ inches below the actual apex was the primary seat of disease in the large majority of cases. This observer also drew attention to the fact that tuberculous disease in its progress through the lungs followed a distinct "line of march," particularly in the chronic and fibroid varieties, the disease spreading in each lobe from above downwards, hardly ever from below upwards. It must be admitted, however, that Fowler makes no attempt whatever to explain the rationale of the "line of march" which he so carefully

¹ Horton-Smith-Hartley : Trans. Roy. Soc. Med., vol. vi., No. 6, April, 1913.

² Fowler : "Localization of Lesions in Phthisis," 1880.

describes, and it would seem that a more or less constant "line of march" must have some rational explanation.

On the other hand, the view is now held by many, including the writer,¹ that the *hilus*, and not the apex, is in general the primary focus of disease—certainly in children, and probably also in adults. The strongest evidence has come from the radiographers.

Dr. Alfred Jordan² states categorically that *chronic pulmonary tuberculosis does not generally commence at the apex, but begins at the roots and extends along the branchings of the large bronchial tubes*. Dr. Jordan inclines to the view that the primary tuberculous deposit does not take place in the bronchial glands, but in the peribronchial tissues in the form of broncho-pneumonic foci, and that the glandular enlargement is secondary. He believes that the tuberculous process generally spreads by broncho-pneumonic extension in the peribronchial tissues, and that these deposits in favourable cases cicatrize into fibrous bands, appearing as dark, radiating shadows on X-ray examination of the affected lungs. He further states that in advancing cases the disease, commencing as a definite peribronchial infiltration, spreads from the hilus in all directions, but most rapidly along the ascending and descending branches of the main bronchus, and that sooner or later the disease reaches the apex by way of the ascending bronchiole. When the apex is reached, the disease, he says, may there progress rapidly, so that the apex appears to be clinically the chief seat of invasion, the primary disease at the root being so deeply placed as to escape detection from without. He explains purely apical cases on the hypothesis that the tuberculous infection has travelled up from the hilus along the main ascending bronchiole to the apex in an early stage, and progressed there, while the track of infection from the root to the apex has healed up, leaving only an excess of fibrous tissue along the ascending bronchiole. Dr. Jordan bases his conclusions mainly on his interpretations of radiographic shadows in the lungs of living subjects, and of similar shadows obtained by the X-ray examinations of lungs taken from the post-mortem room.

Dr. Bythell³ goes so far as to state that *in children pulmonary tuberculosis invariably starts at the roots of the lungs*. Out of 300 successive cases in children examined clinically and radiographically, he had never seen a single case in which the disease had not obviously originated at the hilus of the lung, nor met one in which the sole or even the primary lesion had been at the apex. Whenever there had been an apical lesion, there was in every case revealed by the X rays a far more advanced lesion at the root of the lung, which had escaped

¹ Leslie: "Hilus Tuberculosis," *Clinical Journal*, January 22, 1913.

² Jordan: *Practitioner*, February, 1912, and *British Medical Journal*, August, 1912.

³ Bythell: *Proc. Roy. Soc. Med.*, vol. vi., No. 5, pp. 77 and 92, March 13, 1913.

detection by the ordinary clinical methods of examination, but which was clearly indicated by definite mottling and nodules in the region of the hilus.

Mode of Extension of Hilus Tuberculosis.

In a number of cases the infection seems to spread directly to the lungs from the caseous bronchial glands, often apparently by direct contact. Indeed, as Sir William Osler has pointed out, and his statement is confirmed by the writer's observations, it is sometimes difficult to determine in a section through the affected lung where the caseous bronchial glands terminate and where the pulmonary tissue begins. In the greater number of cases, however, the extension is probably by way of the lymphatic network surrounding the pulmonary vessels and bronchi.

Kingston Fowler¹ himself, in discussing the pathology of chronic fibroid phthisis, admits that the extension of the disease is more towards the peribronchial tissue than towards the alveoli, and states that the bronchi may be thickened from small-celled infiltration, while actual tubercles may be present both in the walls of the bronchi and in the peribronchial tissue, in which cases groups of granulations having a racemose arrangement are usually present. Fowler also found that the lining membranes of bronchi which pass through such tuberculous broncho-pneumonic areas are often intensely injected and swollen, and may even have undergone extensive tuberculous ulceration. He further found that tuberculous infiltration of the peribronchial sheath may lead to marked thickening of the tube and narrowing of its lumen. In some of these cases this fibrous constriction and narrowing of the bronchus may lead to bronchiectatic dilatation beyond the site of constriction.

Dr. Jordan's² views on the mode of extension of hilus tuberculosis are expressed in the following paragraph: "The first stage is catarrh of the air-tubes; this gives no X-ray evidence of its existence. The next stage is catarrhal broncho-pneumonia; in this stage the lumen of the air-tubes is found to contain catarrhal cells (epithelioid plates), while the tissue surrounding the bronchial cartilages is found infiltrated with leucocytes. This peribronchial small-cell infiltration gives rise to the X-ray mottling along the air-tubes which is the characteristic feature of peribronchial phthisis. The glandular involvement at the roots is a secondary change. The glandular enlargement is usually an early result."

Dr. Bythell,³ on the other hand, supports the present author's views expressed in the *Clinical Journal* of January 22, 1913, respecting

¹ Fowler: "Diseases of the Lungs." Fowler and Godlee.

² Jordan: Proc. Roy. Soc. Med., vol. vi., Nos. 5 and 6, 1913.

³ Bythell, *ibid.*

the hilus glands being themselves in many cases the direct or indirect origin of chronic pulmonary tuberculosis. It is the glands, he says, that are the actual danger spots. Dr. Bythell summarizes the present writer's views, which he strongly supports from his own personal experience, in regard to the primary glandular origin—direct or *indirect*—of chronic pulmonary tuberculosis and the mode of extension into the lung-tissue, in the following paragraph: "The lung may become infected either by the direct spread of the tubercular foci from the breaking down of the caseous glands, or by the bacilli being carried backwards along the lymphatics by what is known as the retro-impulsion of lymph, owing to the block that has taken place at the bronchial glands. Later, as the disease becomes more established, the apices may become infected either by this direct extension along the lymph channels, or as independent foci by one of the other methods, such as autogenous blood infection, aspiration broncho-pneumonia, or by bronchial embolism of infective particles detached from an open lesion."

The writer cannot accept in its entirety Dr. Jordan's view regarding the universality of peribronchial extension, and certainly not in the later stages of pulmonary tuberculosis, when we have to deal with an "open," and not a "closed," disease, and when there is accordingly a constant liability to such accidental contingencies as autogenous blood infection or bronchial embolism and aspiration broncho-pneumonia. In the "closed" stages, however, there seems little doubt that the extension does take place along the perivascular and peribronchial lymphatics. In the writer's opinion this occurs only to a limited extent and in the earliest stages as the *direct* result of primary bronchial infection, and then only in the form of small isolated deposits; though even a few such foci, however quickly they heal up, may serve to reinforce the virulent activity of the originally inspired bacilli, few of which probably in their original active state succeed in surviving the passage through the various lines of defence on their way to the root glands. It is the perivascular and peribronchial interstitial tissue rather than the pulmonary tissue proper which is mainly affected at this very early stage, when the lymphatic exits to the glands are freely open and the glands themselves functionally active. Small isolated foci of broncho-pneumonia do, however, occur, and usually heal up rapidly. The fine radiating hilus streaks seen with the X rays even in healthy children may have originated from a fibrous transformation of this small-celled perivascular and peribronchial connective-tissue infiltration. On the other hand, the mottling, blotching, and other evidences of gross hilus lesions, are probably later phenomena associated with the stagnation or backward flow of lymph consequent on the blocking resulting from glandular disease, whether active or quiescent.

The present writer inclines to the view that the bacilli which pro-

duce the actual pulmonary disease do not necessarily or even generally come from the infected glands, which probably act more as blocks, causing stagnation of the lymph stream, which contains bacilli derived from inhalation, peribronchial deposit, and other sources. These bacilli find their way through the distended afferent lymphatics and so invade the pulmonary tissue. This process would tend to extend gradually farther, and so in this way the disease would slowly advance from hilus to periphery. On this retro-impulsion of lymph theory (fully described in "Hilus Tuberculosis," ¹ *Clinical Journal*, January 22, 1913) a healed

FIG. 1.—SKIAGRAM OF CHEST FROM SUPPOSED CASE OF SPASMODIC ASTHMA FOLLOWING WHOOPING-COUGH IN BOY AGED NINE YEARS. IN REALITY A CASE OF HILUS TUBERCULOSIS.

Note blotching (*H*) at roots of both lungs (most distinct on right side) lying external and parallel to the narrow, vertically placed cardiac shadow. At the right hilus there are two small opaque spots suggesting old calcified foci (*C*). Linear shadows may be traced passing towards the periphery and extending in the direction of the apices as high as the level of the second rib in front. Here and there throughout both lungs may be seen small rounded opacities (*F*) arranged in clusters, indicating foci of tuberculous broncho-pneumonia. N.B.—After three months' open-air treatment at Bournemouth patient gained one stone in weight, and the asthmatic attacks entirely disappeared.

calcareous gland with no active bacilli might act as an indirect cause of pulmonary affection simply due to its blocking action.

The principal evidence in support of the hilus as a primary focus of disease is obtained from *radiographic examinations*. In regard to the important question of the right interpretation of the various root shadows, including the diagnosis of acute and chronic tuberculosis, the

¹ See "Hilus Tuberculosis," *Clinical Journal*, pp. 247-250, January 22, 1913.

reader is referred to the writer's original article already alluded to and to the recent discussion at the Royal Society of Medicine¹ on "X Rays in Pulmonary Tuberculosis."

The accompanying radiograms illustrate an early and a late stage of hilus tuberculosis.

The *pathological evidence* of the spread from the hilus is, in the nature of the case, necessarily scanty. Patients do not die in the early stages of chronic pulmonary tuberculosis, when the disease is limited to the root area. At death the lungs are generally infiltrated throughout,

FIG. 2.—SKIAGRAM FROM CASE OF CHRONIC FIBRO-CASEOUS TUBERCULOSIS (SUPPOSED ASTHMA).

Case of gentleman, aged thirty-nine years, with twelve months' history of asthmatic attacks. Had occasional cough and expectoration, which contained tubercle bacilli. The physical signs consisted of a few crepitations at the right anterior apex, numerous rhonchi scattered over both lungs, and impaired expansion of both sides of the chest. Note the elongated cardiac opacity, the extensive hilus blotches on both sides, the well-marked striation and numerous rounded opacities in both lungs, and particularly the finger-like processes extending from the hilus to the right apex.

so that it is almost impossible to tell whether the disease started at the root or at the periphery. Still, occasional cases have arisen (and will be forthcoming more and more as time goes on) when the patients have died from some intercurrent disease or complication, and where the pathological evidence has strongly supported X-ray findings.

In one case published by Dr. Jordan it was possible to compare the previous X-ray pictures with the post-mortem findings, the patient having died of tuberculous ulceration of the intestines before the disease

¹ See Proc. Roy. Soc. Med., vol. vi., Nos. 5 and 6, 1913.

of the lungs had advanced beyond recognition. In this case the pathological changes corresponded precisely with the radiographic appearances.

It is not at all uncommon to meet post mortem with instances of extensive disease more or less limited to the root area, while the apices and peripheral portions of the lungs may be wholly or comparatively free from tuberculous infiltration. Fig. 3 is a good illustration of this type of case, of which the writer has seen a considerable number,

FIG. 3.—CHRONIC FIBROID PHTHISIS COMMENCING AS HILUS TUBERCULOSIS.

This drawing was made from a specimen in Brompton Hospital Museum (Catalogue No. 116), and represents a complete section through right lung. It is an excellent example of chronic fibro-caseous phthisis, the disease having evidently commenced at the hilus and extended outwards into the surrounding tissue in a wonderfully symmetrical fashion. *P*, Pulmonary vessel cut longitudinally; *B*, air-tubes cut across; *G*, caseous bronchial glands at hilus; *T*, tuberculous fibrous thickening and infiltration extending outwards from hilus; *R*, racemose tubercle in perihilus area; *L*, imaginary line circumscribing hilus of lung; *H*, peripheral zone of healthy lung-tissue $\frac{1}{4}$ inch wide. (Drawing by author.)

in which extensive central disease is surrounded by a peripheral zone of healthy pulmonary tissue.

It is a drawing of a specimen in the Brompton Hospital Museum from the case of a man, aged thirty-four years, who died in the hospital. The following is the description of the naked-eye characters of the right lung taken from the museum catalogue published in 1904: "Numerous recent racemose tubercles with pigmented centres and caseating circumferences are seen affecting the central portions of the upper

and lower lobes, a shell of practically unaffected lung-tissue about $\frac{3}{8}$ inch thick separating them from the pleural surface. This specimen explains how in some cases extensive tuberculous disease of the lungs may be present, and yet the physical signs may be scanty or altogether absent." The disease in this case seems to have extended more or less continuously in all directions, though the principal extensions are along the pulmonary vessels and air-tubes.

The great majority of acute and chronic *pleuritis*, whether dry or accompanied by sero-fibrinous effusion, are tuberculous in origin, although there may be nothing in the lungs or elsewhere to suggest the presence of tuberculosis; and Sir William Osler states that *in almost every instance of tuberculous pleurisy there are tuberculous foci in the bronchial glands*. Applying here also the doctrine of the retro-impulsion of lymph, the ready extension of the tuberculous process from the hilus to the pleura along the terminal pleural lymphatics is obvious. The extensive tuberculosis of the pleuræ and the mediastinal glands so frequently found in cattle is significant in this connection. There is also a close association between *asthma*, particularly in children, and hilus tuberculosis (see Figs. 1 and 2). According to C. Weigert, *acute miliary tuberculosis*, whether pulmonary or general, is often the direct result of penetration of branches of pulmonary veins by caseating bronchial glands.

The rapidity of the extension of the tuberculous process in the lungs is, as a rule, inversely proportional to the degree of immunity—natural or acquired—the determining factors being not so much the numbers or virulence of the invading bacilli, as the resistive power of the invaded tissues on the one hand, and the degree of vulnerability and phagocytic efficiency of the bronchial mucous membrane on the other.

The fact that most of us have had at one time tuberculous deposit now represented by calcified glands or fibrous thickening at the roots may in reality be an advantage. A definite degree of immunity due to the presence of antibodies has thus been gradually established during childhood, and we are actually in a better condition as adults to resist any future invasion than if we had never contracted the disease at all. It is probable, indeed, that the gradual reduction in tuberculosis mortality in civilized countries is largely due to the fact that the population has become gradually immunized in this way. This theory will serve to explain the alarming spread of and mortality from tuberculosis among the native adult populations of Bengal, the West Indies, Australia, and elsewhere, who have not been immunized.

As regards the *value of early diagnosis* of hilus tuberculosis, it is impossible to over-estimate its importance. The late Dr. Gee was of opinion that, "if physical signs of pulmonary tuberculosis were present,

the probabilities were against complete recovery; but if the disease were detected before this, there was hope that it might be cured." This statement is as true now as it was twenty years ago. In modern instantaneous radiography we have a method of diagnosing early cases of pulmonary tuberculosis that Dr. Gee in his day did not possess, it being now possible to discover deep-seated central tuberculous disease in the hilus and surrounding pulmonary tissue before the tubercle bacilli appear in the sputum and long before physical signs manifest themselves.

THE ADJUNCT TREATMENT OF TUBERCULOSIS WITH CERTAIN ORGANIC EXTRACTS.

By HENRY R. HARROWER,

M.D.,

Late Professor of Clinical Diagnosis, Loyola University, Chicago, Illinois.

As the years go by our ideas regarding the treatment of tuberculosis are modified, and in no phase of therapeutics do opinions change so much. About 1890 Professor Koch discovered a therapeutic wonder in tuberculin, but its vogue was short-lived, for the intricacies of dosage had not then been learned. Less than ten years ago hyperalimentation was the fashion, and, as with fashions in other things, it was carried to extremes. Nowadays tuberculin has regained its place, but its application is almost an obsession, and the devotion of some practitioners to tuberculin in one form or another seems to show that the fashion has changed again.

There appears to be an idea, almost universal, that as tuberculosis is an invasion of the body by a certain bacillus, the treatment must needs be directed primarily towards the destruction of these organisms and the neutralization of their toxic products. That this is essential none will deny, but, unfortunately, the view is much too narrow. Tuberculosis is first and foremost a condition of lowered resistance in which the infective process has become so prominent as quite to overshadow the original and by far the most important defect. The following extract from a recent editorial¹ aptly states the matter in the plainest of language:

"The cause of active tuberculosis must now be sought in something which destroys our tolerance, and permits our own lesions to

¹ See *American Medicine*, New York, 1913, xix. 1, 5.