ORIGIN, DIAGNOSIS, AND MANAGEMENT OF EARLY BRONCHOGENIC TUBERCULOSIS

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(WITH SPECIAL PLATE)

There is scarcely a report by a medical officer of health, tuberculosis officer, or sanatorium superintendent that does not refer to the advanced stage of the disease in which those suffering from pulmonary tuberculosis first come for treatment under the tuberculosis scheme. The cry is, "If only we could get the cases early!" The emphasis laid on this plea implies that if people were seen early in the development of the disease transmission of infection to others would be avoided and the disease would be made to heal in all patients. It is not surprising, therefore, that some advocates of mass radiological examinations see in them the key to the control of tuberculosis. This should not, however, be taken for granted, because up to the present relatively few such early cases have been detected, treated, and followed up; indeed, no clear-cut conception of what constitutes an early case is being taught.

Definitions

The term "pulmonary tuberculosis" itself requires clarification. It usually refers to the disease commonly seen in adolescents and adults, characterized by gradual destruction of the lungs as a result of the intrabronchial spread of massive doses of bacilli following cavitation of a tuberculous focus. But other active tuberculous lesions are common in the human lung—those of the primary infection and of the dissemination that follows it. It would be well, therefore, to use the term "pulmonary tuberculosis" as implying any active lesion in the lung belonging to any phase in the evolution of tuberculosis, and to restrict the use of another term—that of "bronchogenic tuberculosis"—to the common type of bronchogenic disease.

In discussing the early stage of bronchogenic tuberculosis mention must be made of those lesions in the lung which occur during the phase of dissemination in the evolution of tuberculosis (Kayne, Pagel, and O'Shaughnessy, 1939) (of which chronic miliary tuberculosis is but one clinical type), for such haematogenous cases may simulate bronchogenic tuberculosis when the lesions cavitate (Case 1). These thin-walled "punched" cavities have, however, little caseous matter in their walls and probably only rarely lead to intrabronchial transmission of bacilli. The common

beginning of bronchogenic tuberculosis is a single focus, usually in the upper part of the lung, which enlarges, liquefies, and cavitates, when secondary foci develop as a result of intrabronchial transmission (this process has been accurately reproduced in animals). Since the disease takes on its grave aspect with liquefaction and cavitation of the first focus, early bronchogenic tuberculosis must be defined as the earliest recognizable caseous focus which has not yet opened into a bronchus. Such a focus does not give rise to abnormal physical signs, is often associated with no symptoms, either local or constitutional, and may even present no evidence of toxæmia as shown by a raised sedimentation rate or altered blood picture. But it can be detected in a radiograph of good quality when it is less than a quarter of an inch in diameter unless the focus is entirely hidden by the shadow of a bony structure. It may appear as a uniform cloudy haziness or as a group of nodules, some of which coalesce. To summarize, early bronchogenic tuberculosis is the smallest radiologically detectable focus with potentialities for liquefaction.

A small shadow in the upper part of the lung field may be caused by other tuberculous lesions (active or healed). Thus the pulmonary focus of an active primary infection may sometimes be seen as a distinct shadow in the lung

field. Nowadays the primary infection occurs often during adolescence and not very rarely even in adults, but, as in children, enlargement of at least the corresponding hilar shadow is then seen—a change not met with in the early lesion of bronchogenic tuberculosis. Again, the pulmonary foci of dissemination associated with and following the primary infection may resolve completely, or may leave behind a few tiny nodules scattered throughout the lungs, or a group of rather bigger nodules at the apices (Simon foci), or one or more larger foci a quarter of an inch or more in diameter. The latter foci usually consist of a caseous centre with fibrotic capsule,

and have been termed "round foci." Finally, quite sizable lesions of bronchogenic tuberculosis may develop and heal unsuspected, so that one is presented with a single residual non-active focus which may be mistaken for a first or early focus of bronchogenic tuberculosis.

Origin of Bronchogenic Tuberculosis

The early lesion of bronchogenic tuberculosis occurs in an individual who has already overcome his primary infection, though the interval between the onset of the first infection and the lesion of bronchogenic tuberculosis may be as short as a few months (Case 2). There are several possible theories of the origin of the early lesion, since it is known that bacilli may remain alive in an apparently healed primary pulmonary focus (rarely), a primary glandular focus (more commonly), and in foci of dissemination (quite commonly). As a result of lowering of resistance or fol-

LEGENDS FOR THE SPECIAL PLATE

FIG. 1.—Case 1 (May 31, 1939). Coarse miliary mottling in upper half of each lung.

FIG. 2.—Case 1 (June 11, 1940). In addition there is now a thin-walled irregular cavity in right upper zone.

FIGS. 3 and 4.—Case 2. Showing progression towards bilateral cavernous bronchogenic tuberculosis (films taken in August, 1939, and November, 1940).

FIG. 5.—Case 3. Round focus with calcified shell in left infraclavicular region.

FIG. 6.—Case 4. Primary calcified complex on left side (not reproduced) and roundish lesion in right mid-zone.

FIG. 7.—Case 5 (March 13, 1940). Faint circular shadow on right in fourth posterior interspace, and "filling" of right costo-phrenic angle (not reproduced).

FIG. 8.—Case 5 (November 4, 1940). Showing definite extension of shadow upwards and outwards.

FIG. 9.—Case 7. Roundish focus in left infraclavicular region with two tiny calcifications (marked C).

FIG. 10.—Case 7. Showing definite increase in size of shadow, six months later.

FIG. 11.—Case 9 (January, 1940). Calcified foci on both sides, with pleural thickening at left base (right upper zone only reproduced).

FIG. 12.—Case 9 (December, 1940). Showing a faint but definite shadow around a group of small calcified foci on right side.

* The views expressed in this paper are those of the author, and do not necessarily represent those of the Middlesex County Council.

lowing a contiguous non-tuberculous process, exacerbation might occur in any of these foci, which could themselves liquefy and cavitate, or, if the exacerbation occurs in the neighbourhood of a small bronchus, the discharge of a large number of bacilli into another part of the lung might give rise to a fresh focus, which would cavitate in due course. Neither of these alternatives seems to happen in the case of the primary pulmonary lesion, but both have been shown conclusively to occur in connexion with the remains of haematogenous disseminations. The apical remains (Simon foci) exacerbate and give rise to infiltrations lower in the lung, which may liquefy and cavitate; and "round foci" exacerbate and themselves cavitate. Another possibility is a new focus that develops as the result of a fresh exogenous infection. No pathological confirmation of this is available, but epidemiological and clinical evidence points strongly to its occurrence. Presumably a focus develops when massive and repeated doses of bacilli are inhaled. The lesion probably regresses spontaneously unless at the same time resistance has been lowered, when the lesion tends to progress to bronchogenic tuberculosis. Yet another possibility for which supporting evidence is scarce is superinfection. The mechanism would then be as follows. The introduction of a fresh massive dose would not itself give rise to a new focus in the lung, but the destruction of the bacilli so introduced would release toxins which could cause exacerbation of healed primary or disseminated foci.

To summarize, it may be stated with some assurance that the two common mechanisms from which arises the early lesion of bronchogenic tuberculosis are exacerbation of residual foci of dissemination and fresh exogenous reinfection with massive doses in people with lowered resistance, but the relative frequency of these two mechanisms cannot be established at present. A third, uncommon, origin is a bronchogenic sequel to a progressive chronic dissemination in the lungs.

Detection of Early Bronchogenic Tuberculosis

It is again stressed that early bronchogenic tuberculosis can be detected only by radiological examination. Evidence for this will be found in the work of Braeuning in Stettin and of Malmros and Hedvall in Lund, both of whom published monographs in 1938 on their findings in students, nurses, and hospital personnel. Braeuning reported on 86 people who presented an abnormal radiological picture within one year or less of having given a normal radiograph; primary infection as a cause of the abnormal shadows seen in the later radiographs could be excluded with reasonable certainty. In no fewer than 50 of the 86 persons no symptoms whatever could be elicited. In 10 of the 86 the interval between a clear radiograph and the development of a focus was not more than three months, so that the question of symptoms could be examined with special care; in no fewer than 6 there were no symptoms whatever. Similarly, in the majority of 43 students and nurses who were tuberculin-positive and gave an abnormal radiograph after an interval following a normal one, Malmros and Hedvall found that the onset of the radiological shadow was not accompanied by symptoms.

It is clear that early bronchogenic tuberculosis will not be detected if one waits until the individual consults his doctor, that it can only be discovered by mass radiological examination of the apparently healthy, and that the most fruitful results will be obtained in those exposed to massive infection or conditions lowering general resistance, such as home and work contacts, medical students, nurses, industrial employees, etc. The method to be used requires careful consideration. If such examinations are merely for the purpose of detecting well-developed bronchogenic tuberculosis in people who delay consulting a doctor or in whom

the doctor fails to recognize this possibility, then any method will do, including miniature radiography and screening. If, however, the mass examination is intended to detect the early focus of bronchogenic tuberculosis, then two conditions are essential—a good radiograph, and repetition of the examination every six months or so; for it is most desirable to have a normal film to compare with, and too long a delay between examinations may allow cavitation to occur before the recently developed focus had been detected.

Diagnosis of Early Bronchogenic Tuberculosis

The recognition of a radiological shadow as one of early bronchogenic tuberculosis is easier if a radiograph and a tuberculin-test result, dating no further back than a few months, are available. When a previous film is not available the shadow must be differentiated from a primary pulmonary focus, Simon foci, round foci of dissemination (Cases 3 and 4), and the healed residua of previously unsuspected bronchogenic tuberculosis. It has been shown that the focus of early bronchogenic tuberculosis is found as often above the clavicle as below it, and that it appears as a group of small foci or a shadow with denser nodules in it as often as in the form of a homogeneous cotton-wool shadow. In the light of recent work the distinction (stressed by the late Fishberg, for instance) between a very benign apical tuberculosis and an acute progressive infra-clavicular *Frühinfiltrat*, or "early infiltration" (as worked out by Assmann and Redeker), is no longer tenable. In practice what needs to be decided is whether the radiological shadow represents an active lesion. It has already been stated that there may be total absence of symptoms. There may be slight pyrexia; but Braeuning found that in 24 of 43 patients, in whom axillary temperature was recorded several times a day, no pyrexia could be detected. Similarly, in 42 of 75 patients in whom the sedimentation rate was noted the figure was normal, and this series included some patients with early lesions which rapidly cavitated. Again, 7 of 48 patients with definitely active lesions showed a perfectly normal blood picture. Only in a few of his patients were all these three investigations carried out, but in 3 of them they were normal. Similarly, Malmros and Hedvall conclude from their findings in 43 students and nurses that whether the focus begins as a group of small nodules or as a so-called "early infiltration" the sedimentation rate is generally not raised. It appears, therefore, that help in deciding whether the lesion is active or not must often be obtained by noting changes in the radiological shadow after an interval (Cases 5, 6, and 7).

A month seems a suitable interval, since this would generally allow enough time to produce a radiological change, and would as a rule, but only as a rule, not be long enough to allow gross harmful changes to develop, unless the case is already one of those which deteriorate rapidly even under treatment.

As will be shown later, another film after a short interval is also of great help in the management of the case. But if one is to rely on these slight radiological changes the standard technique of the films must be good and uniform, and changes that might be caused by technical differences must be carefully appraised.

Prognosis and Management

An attempt must next be made to estimate the outlook for the lesion. Serial radiographs will have already given some indication of its rate of progression. It will be of immense value to know whether the lesion developed as

a result of lowered resistance and exacerbation of old foci or as a new exogenous infection from massive contact with or without lowering of resistance. When only massive contact is a factor, interruption of this may be sufficient to allow natural regression of the lesion* (Case 8). If lowering of resistance is a factor, then clearly this will not be enough, and measures to increase this resistance, either in the home or in a sanatorium, will need to be taken. It may be very difficult or even impossible to find the incriminating factor or factors, but careful history-taking and investigation of the patients' environment should discover this with reasonable sureness in an important proportion of cases.

The first steps in the management of the individual patient are therefore serial films and investigation of aetiological factors. If the lesion is only doubtfully progressive, or is probably due to massive contact alone which can be interrupted, then it may be justifiable to allow the patient to carry on with his work, more rest in leisure hours being suggested, and monthly investigations with radiographs, sedimentation rates, etc., being continued. If the lesion is definitely progressive, and the factor of lowered resistance cannot be excluded, more is indicated. Before advising institutional treatment, measures short of this must be considered, because reluctance to give up work and leave home for several months is likely to be very great when the patient feels there is nothing the matter with him. When ceasing work is likely to be followed by the necessary restful conditions, good food, and discipline in the home or with relatives, then this home treatment might be tried in the slowly progressive case, provided monthly radiographs and other investigations are practicable. Failing this a stay in an institution is essential.

A digression on tubercle bacilli in the sputum is here necessary. The definition of early bronchogenic tuberculosis excludes radiologically detectable cavitation, but even a tiny focus, if situated in the neighbourhood of a small bronchus, would produce a slight amount of bacilli in any sputum expectorated, even if cavitation had not occurred, and therefore the presence of bacilli in the sputum does not necessarily remove the case from the group of early bronchogenic tuberculosis. On the other hand, the outlook for such a patient is worse, because extension to other parts of the lung is already possible. It may be that even among people with early foci who produce no sputum, bacilli would be grown from the gastric contents of some or many; this requires further investigation. In the American classification of pulmonary tuberculosis "minimal lesions" are defined as: "(a) slight infiltration without demonstrable excavation; (b) a small part of one or both lungs—total volume of involvement, regardless of distribution, shall not exceed the equivalent of the volume of lung tissue which lies above the second chondrosternal junction and the spine of the fourth or body of the fifth thoracic vertebra on one side"; tubercle bacilli may be present or absent. The official classification in this country is so antiquated that it still relies on physical signs and does not appear to recognize the existence of radiography: discussion of it is therefore futile. The presence or absence of tubercle bacilli in association with the early lesion will clearly affect the management of the patient. Thus, to begin with, their presence will indicate institutional treatment in every case.

Institutional treatment may consist of sanatorium regime alone, rest in bed, or rest in bed combined with collapse therapy; bed rest, continued until the lesion is quiescent, should be considered a *sine qua non* of properly conducted collapse therapy. Whatever benefits may be claimed for sanatorium regime alone, this is not justified in the cases discussed here, because it is so vital that the institutional stay of these patients, most of whom feel more or less well and are reluctant to give up work and most anxious to return to it, should be as short as is compatible with

safety. When there is no sputum, or no tubercle bacilli can be detected in the sputum (culture being of course employed), strict rest in bed, continued until there is evidence of quiescence as demonstrated by serial radiographs, sedimentation tests, and blood examinations, is sufficient unless the lesion shows signs of progression. If progression is evident at the end of a month's bed rest, or if there is no definite evidence of regression at the end of, say, two months, then continued bed rest alone is not justified. And to these patients, as well as to those whose sputum is positive, collapse therapy should be applied without delay. There is plenty of authoritative opinion to support this conclusion. Thus the following among American workers may be quoted: O'Brien (1933), Myers and Levine (1935), and John Alexander (1937). O'Brien favours phrenic interruption, permanent or temporary, according to the case. Turner and Collins (1936) show that artificial pneumothorax in these minimal cases is associated with very slight operative risk, that the percentage of cases with a free pleural space and therefore with an effective collapse is very high, and that the results are distinctly worse when the induction of a pneumothorax is delayed even by a few weeks. The general application of pneumothorax therapy for early bronchogenic tuberculosis does imply, however, that the treatment will be carried out both in institution and in clinic by experienced practitioners, or at least under their close supervision, and, if at all possible, by one physician throughout—the one who induced the pneumothorax.

Advice in regard to the treatment of early bronchogenic tuberculosis may thus be summarized: Get on with it at the beginning of the treatment, for as often as not it is the patient and not the physician who decides when it will end.

Mass Radiological Examinations

Unless the early cases of bronchogenic tuberculosis are properly managed and treated, little benefit will accrue from mass radiological examination: 15% of Braeuning's patients were sputum-positive within one year of a normal radiograph; 41% became sputum-positive within a short time of detection. A cavity was visible after a normal radiograph in 1 case within a month, in 7 within six months, and in 15 within a year. (Hence the importance of repeated radiographic examination at intervals of less than a year if infection from open cases to others is to be prevented.) Death took place from the time of a normal radiograph in 1 case within a month, and in 2 within fourteen months. In most cases, however, the development of the disease was slow at first.

From this it appears that while mass radiological examination will be of distinct benefit to individuals and will result in decreasing infection to others, as a method of eradicating tuberculosis it is likely to play a truly effective part only under the following conditions:

1. That the examinations will be carried out extensively.
2. That the method of radiological examination used will be the best available—that is, radiographs will be taken with a satisfactory technique.
3. That re-examinations will be carried out at no longer interval than six months or so.
4. That full facilities will be available for detailed investigation and observation of all cases with an abnormal or suspicious radiograph.
5. That all persons shown by this means to be suffering from early bronchogenic tuberculosis will have proper and adequate institutional treatment.
6. That such treatment will not interfere with the return of the patient to his work and with the economic security of the family during the absence of the breadwinner, housewife, or mother.

To secure these conditions in full would probably necessitate economic and social changes in our community that

* Persistent massive contact in such a case might be harmful not only by influencing this lesion but by giving rise to a fresh lesion which would progress if resistance were subsequently lowered.

would themselves do more than mass radiological examinations to reduce the incidence of tuberculosis.

Case Reports

Case 1.—A girl aged 8. Mother suffers from advanced bronchogenic tuberculosis. Tuberculin-positive; no symptoms; radiograph on May 31, 1939 (Special Plate, Fig. 1), showed coarse miliary mottling in upper half of each lung. Child not seen again till June 11, 1940, when she was stated to be well and had put on 10 lb. There was tiredness, however, and a radiograph (Fig. 2) showed in addition to the miliary mottling a thin-walled irregular cavity in the right upper zone, presumably due to coalescence and liquefaction of some of the larger foci.

Case 2.—A lad aged 16. Seen in a hospital in February, 1939, for unexplained fever: a radiograph on February 15 showed no abnormality, but one taken a week later revealed an opacity to right of superior mediastinum, and the tuberculin test was positive. Diagnosis: primary tuberculous infection with involvement of mediastinal glands. April 12: lymphocytic left pleural effusion; mediastinal shadow smaller. April 25 to June 7: in sanatorium. Radiograph on August 16 (Fig. 3) still showed enlargement of right paratracheal glands and remains of pleurisy at left base. Radiographs November 15 and January 31, 1940, revealed fresh foci at top of left apex, apparently unchanged in this interval, there being no symptoms, and sedimentation rate (S.R.)—Westergren 1 hour—remaining normal. Radiograph July 31 (Fig. 4) showed bilateral bronchogenic tuberculosis with cavitation. (Interval between onset of primary infection and fully developed bilateral cavernous bronchogenic tuberculosis thus not longer than seventeen months.)

Case 3.—A man aged 32, referred by recruiting medical board. Mother suffers from advanced bronchogenic tuberculosis. No symptoms; radiograph October 15, 1940 (Fig. 5), showed round focus with calcified shell in left infraclavicular region; S.R. normal. Radiographs one month and three months later showed no change in appearances of this focus. (Diagnosis: "round focus" of dissemination; calcifying or partly calcified primary focus excluded, as no calcification seen in corresponding hilar glands.)

Case 4.—A girl aged 11. Father died of bronchogenic tuberculosis six years ago; now in contact with brother suffering from advanced bronchogenic tuberculosis. Slight cough; S.R. normal; radiograph, March 6, 1939 (Fig. 6), showed primary calcified complex on left side and roundish lesion in right mid-zone. Admitted to sanatorium for observation: tuberculin test found positive. Throughout stay there and after discharge, radiographs showed no change in appearances of this focus. (Diagnosis: "round focus" of dissemination; it is possible, however, that the focus represented an "early infiltration" which was already quiescent when the girl was first seen.)

Case 5.—A woman aged 33. Intermittent contact until Christmas, 1939, with sister suffering from bronchogenic tuberculosis. History of pain in right side of chest in March, 1939, when she remained in bed for a month. No symptoms; S.R. = 15. Radiograph on March 13, 1940 (Fig. 7), showed faint circular shadow on right, in fourth posterior interspace and partly covered by anterior part of second rib, and "filling" of right costo-phrenic angle. April 15: no symptoms; S.R. normal; radiograph, apparently no change. September 10: no symptoms; S.R. normal; radiograph showed some change in shape of shadow. November 4: no symptoms, but had a "cold" a week before; radiograph (Fig. 8) now showed definite extension of shadow upwards and outwards; recommended for sanatorium. (Diagnosis: probably exacerbation of "round focus" of dissemination; note (1) the pleural effusion a year before first seen, (2) the interruption of contact.)

Case 6.—A girl aged 18. Father suffered from advanced bronchogenic tuberculosis (died October, 1940). November, 1938: no symptoms; radiograph showed infraclavicular shadow on left. February, 1939: no symptoms; radiograph, shadow unaltered. February, 1940: no symptoms; weight about the same; radiograph showed increase in size of focus. April, 1940: confessed to fatigue during past two months; no abnormal physical signs in chest; S.R. = 16; radiograph showed further increase in infraclavicular shadow. In sanatorium from May to August. September, 1940: no symptoms; radio-

graphically, shadow was "harder" in appearance, indicating fibrosis. (Diagnosis—most likely sequence: (a) healed dissemination with residual small foci; (b) development of "round focus" which remained latent; (c) exacerbation of this focus due to ? superinfection and/or lowering of resistance.)

Case 7.—A man aged 25, referred by recruiting medical board. Two years previously radiographed at a tuberculosis dispensary elsewhere: this radiograph, inspected by me, revealed no abnormality in lung fields. First seen May 23, 1940: slight cough and sputum; radiograph (Fig. 9) showed roundish focus in left infraclavicular region with two tiny calcifications in one part of it; S.R. normal; sputum negative. June, 1940: very slight cough and sputum (negative); put on 2 lb.; S.R. normal; radiograph, apparently no change. August, 1940: no symptoms; very slight sputum (negative); lost 3 lb.; S.R. normal; radiograph, apparently no change. November, 1940: had a "cold" last week; slight cough and sputum; has put on 3 lb.; S.R. normal; radiograph (Fig. 10), when compared with film taken in May, showed definite increase in size of shadow. (Diagnosis: slowly progressive lesion, probably due to exacerbation of tiny round healed foci of dissemination. This case also illustrates the cumulative effect of very small increases in size of radiological shadows.)

Case 8.—A girl aged 17. Mother suffers from advanced bronchogenic tuberculosis and admitted to sanatorium. June, 1939: no symptoms; radiograph showed shadow in right second anterior interspace. July, 1939: radiograph suggests decrease in this shadow. October, 1939: radiograph, shadow smaller and "harder" in appearance. (It was therefore concluded that the shadow on the first radiograph had represented an *active* focus of early bronchogenic tuberculosis.) December, 1939: no symptoms; working. March, 1940: no symptoms; radiograph shows the shadow originally seen replaced by a very few fibrotic strands and nodules. (Diagnosis: early bronchogenic tuberculosis ("early infiltration"), ? origin, regressing without treatment, contact having been interrupted; therefore presumably arose as the result of massive infection without lowering of resistance.)

Case 9 (This case, though not referred to in the text, is included because it is closely connected with the subject of this paper, as it shows the really early stage of a true "relapse").—A woman aged 28. Case diagnosed as pulmonary tuberculosis in 1934, when a radiograph showed "(R.) slight infiltration of upper zone, (L.) infiltration of upper and middle zones with cavity in upper zone"; later admitted to sanatorium, where a left A.P. was induced, which was carried on until December, 1937. Seen in November, 1938, May, 1939, and January, 1940, when on each occasion a radiograph (Fig. 11) showed the same appearances—calcified foci on both sides, with pleural thickening at the left base. When seen in December, 1940, she was feeling fit but had a slight cough; she had lost over 6 lb. since January, and had been working seven days a week; S.R. normal; radiograph (Fig. 12) showed a faint but definite roundish shadow around a group of small calcified foci on the right side. Note: (1) the calcified foci on the right side were not present in the film of 1934, so that they did not represent a healed dissemination, but healed lesions of bronchogenic tuberculosis; and (2) the group of foci around which the recent shadow is seen had themselves changed in appearance. This suggests the diagnosis of recent exacerbation, due to lowered resistance, of a "healed" focus of bronchogenic tuberculosis, the exacerbation having caused some of the calcified foci to be "ejected" or at all events to change in position.

Summary

It is suggested that the term "pulmonary tuberculosis" should be used as implying any active lesion in the lung belonging to any phase in the evolution of tuberculosis, and the use of the term "bronchogenic tuberculosis" be restricted to the common type of bronchogenic disease.

"Early bronchogenic tuberculosis" is defined as the smallest radiologically detectable focus with potentialities for liquefaction.

The two common mechanisms leading to the early lesion of bronchogenic tuberculosis are exacerbation of residual foci of dissemination and fresh exogenous reinfection with massive

doses in people with lowered resistance. Another, uncommon, origin is a bronchogenic sequel to a progressive chronic dissemination in the lungs.

Early bronchogenic tuberculosis is generally *not* associated with symptoms or signs (including sedimentation rate and blood picture), and can be detected only by radiological examination of the apparently healthy, particularly among groups exposed to massive infection or circumstances lowering resistance.

The focus of early bronchogenic tuberculosis must be differentiated from other tuberculous lesions: a primary pulmonary focus, apical Simon foci, "round foci" of dissemination, and the healed residua of a previously unsuspected bronchogenic tuberculosis. It is found as often above as below the clavicle, and appears as a group of small foci or as a non-homogeneous shadow as frequently as in the form of a homogeneous cotton-wool shadow (*Frühlinfiltrat*, or "early infiltration"). Help in deciding whether the lesion is active or not must often be obtained from serial radiographs.

Help in assessing the outlook for the lesion is obtained from serial radiographs and by finding the causative factor or factors; mode of origin, massive contact, and lowered resistance must be considered.

Treatment will depend to some extent on these factors. Treatment in the home may be considered under certain circumstances, but institutional treatment is usually essential.

Rest in bed until the lesion is quiescent is indicated whenever institutional treatment is advised. Collapse therapy should be used in addition when there is no evidence of regression of the lesions within a very few weeks. Reasons for these conclusions are discussed.

The relations of tubercle bacilli in the sputum to the definition of early bronchogenic tuberculosis and the classification of tuberculosis are discussed, as well as the significance of this finding in connexion with treatment.

The importance of mass radiological examinations in connexion with the detection of early bronchogenic tuberculosis is stressed, but the method to be used is critically examined. The conditions under which mass radiological examinations are likely to play a decisive part in the eradication of tuberculosis of human origin are brought forward.

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The papers by Hart, Hilton, and Morland deal with tuberculosis in medical students detected by radiological examination. The papers by Schemmel, by Uehlinger, and by Voigtman deal with "round foci."

J. Fénéon (*Thèse de Paris*, 1940, No. 189) states that it has been well known since Fournier's time that syphilis may give rise to osteo-articular lesions. They are rare, however, and occur in the course of active syphilis. The part played by syphilis in the aetiology of chronic rheumatism is much more important, hydrarthroses and arthritis of the hip-joint being the most frequent manifestations. It is often difficult to determine the existence of syphilis in an old-standing case which has been inadequately treated and may be associated with other infections. Inconstant results are yielded by chemotherapy, which should be employed with caution as the patients are affected by diffuse sclerosis. Orthopaedic surgery is important in the correction of deformities.

EMPLOYMENT OF AIR-RAID NOISES IN PSYCHOTHERAPY

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A large proportion of patients admitted to a neuropathic military hospital revealed that their neurotic breakdown was either determined or in part precipitated by the unaccustomed stress of noises, such as gun-fire, shell-bursts, exploding bombs, sirens, planes, and dive-bombing. Some of these soldiers were Dunkirk casualties, but the majority were men who had not served outside England. Experience showed that treatment along orthodox psychotherapeutic lines, which included physical training, occupational therapy, route marches, games, etc., was impeded in these cases by too quiet an environment in hospital. While other symptoms had been resolved there remained a certain hypersensitiveness to air-raid noises.

The type of patient who came into this group and who formed the material for the treatment here outlined consisted of those in whom anxiety was the main presenting symptom. Their symptoms were related closely to experiences of a traumatic nature—physical or psychological—when in the "front line." It was considered that a close approximation between hospital life and life in the Army was necessary if these men were to remain well when returned to their units. An attempt was made to "de-condition" the state of hypersensitiveness to warfare sounds by reproducing the disturbing noises till the patients gradually became more normal in their reactions.

Method

At first the apparatus used for reproducing these noises was rather crude, consisting of a small portable field siren and an assortment of tin boxes and sticks. Later, with the helpful co-operation of the B.B.C., it was possible to make use of gramophone records of actual warfare. Technicians made to our specification records of sirens, the firing of A.A. and machine guns, sounds of planes, dive-bombing, and explosions. Individual sounds or a composite effect could be reproduced when required. Records combining reassurance talks with actual bombing noises were also made. The following technique was employed:

The patient was given a simple explanation of the treatment and its object. He was placed in a state of relaxation, and details of the pulse rate, respiration, blood pressure, presence of tremors, restlessness, and the like were recorded. He was then subjected to the particular sound deemed to be the precipitant in his case. Notes were made of changes occurring in the physical and mental state, and after a short rest period the stimulus was again given. Depending on the response obtained, this was repeated three to seven times at each session, at the end of which the mechanism and meaning of fear were explained. The record chart, revealing a gradual improvement in responses—a phenomenon almost invariably present—was shown to the patient after the first session.

Reassurance and encouragement on common-sense lines were given to certain patients. They were told: "Men, women, and children in our badly bombed cities have developed a new lease of life. They are in better mental trim than those in safe areas. They have faced the raids, have found them not so bad as anticipated, and have got over them. They carry on with their jobs

G GREGORY KAYNE: EARLY BRONCHOGENIC TUBERCULOSIS
(For legends see text)

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

FIG. 7

FIG. 8

F. JACOBY, P. B. MEDAWAR, and E. N. WILLMER: TOXICITY OF SULPHONAMIDE DRUGS TO CELLS IN VITRO
(For legends see text)

FIG. 1

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

FIG. 7

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FIG. 9

FIG. 11

FIG. 10

FIG. 12