A Lecture
ON
BRONCHIECTASIS.

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BY

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BRONCHIECTASIS, or dilatation of the bronchi, both large and small, is a condition quite commonly met with among the in-patients and the out-patients at this hospital. Except perhaps in a few rare congenital cases, it is not a primary disease of the lungs, but is always secondary to some preceding disorder, such as pulmonary tuberculosis, bronchitis, pneumonia, or bronchial obstruction, and as a rule it is a chronic complaint. I make particular mention of the fact that in bronchiectasis both the large and the small bronchi are dilated, because there is a condition known as bronchiolectasis, or dilatation of the bronchioles, in which only the smaller bronchi are involved, the larger not being dilated. Bronchiolectasis is, generally speaking, an acute condition met with in fatal cases of bronchopneumonia, particularly in children; a good many instances of bronchiolectasis have been recorded in fatal cases of influenzal bronchopneumonia during the epidemics of influenza in this and other countries during the last few years. It is a condition secondary to the bronchopneumonia in which it occurs, and does not alter the clinical picture of the primary disease in any way. It is, however, evidence of the severity of the infection in the bronchioles in which it is seen.

For the purposes of this lecture I have made a study of the clinical notes and *post-mortem* records of over 100 cases of bronchiectasis treated at this hospital and at St. George's Hospital, excluding all instances in which the condition was due to chronic pulmonary tuberculosis. My remarks will be based mainly on the analysis of these records, and I wish to thank my colleagues at those institutions for permission to make use of them.

FREQUENCY.

During the last twenty years some 29,700 patients have been admitted to the Brompton Hospital, 567 of them with the clinical diagnosis bronchiectasis, or 1.9 per cent. But these figures do not by any means give a true picture of the frequency with which it occurs. Bronchiectasis is always secondary to some other pulmonary disease; and in the milder degrees is so overshadowed by the more obvious and important primary disorder as to escape comment. For example, a mild degree of bronchiectasis occurs in practically every case of fibroid phthisis, or chronic pulmonary tuberculosis of the fibrotic type, that comes up for examination in the *post-mortem* room. The physician will no doubt suspect bronchiectasis in such patients while they are in the wards, although he may find it very hard to decide in any given instance whether he has to do with bronchiectatic cavities or the common tuberculous excavation of lung tissue, because the physical signs of the two conditions are similar, and indeed the two conditions often occur together. In such cases the primary disease is so much the more important that the secondary bronchiectasis is apt to be disregarded. In fact, it is probable that, pathologically speaking, some degree of bronchiectasis is present in quite 5 per cent. of the patients admitted to the Brompton Hospital.

Sex and Age.—Bronchiectasis is commoner in men than in women; of the 567 cases mentioned above 332 (or 59 per cent.) were males, 235 females. Among 112 cases examined after death, 90 (or 80 per cent.) were males, 22 females; the ages of these patients were as follows:

Under 10	5	31–40	27
10–20	19	41–60	34
21–30	19	Over 60	8

The youngest patient was twelve months old, the oldest 72 years. In no case was the bronchiectasis thought to have been congenital.

PATHOLOGICAL ANATOMY.

The pathological anatomy of bronchiectasis is well described in the ordinary textbooks; it suffices to say here that there may be either a diffuse more or less uniform cylindrical widening of the tubes or else irregular and saccular dilatation. The distinction is quite unimportant clinically, and as a rule both conditions are present together, with more or less collapse and fibrosis of the intervening lung tissue. Special attention may be drawn to the apparently invariable presence of dense pleural adhesions over any lobes or areas of lung affected with bronchiectasis. In a few instances these adhesions precede the widening of the tubes, and are the chief cause of it, but in the majority of the cases now under consideration they are the result and not the cause of the bronchiectasis, and are due to the outward spread of infection from the bronchi. In all cases the dilated bronchi contain much septic secretion, usually offensive and highly tryptic.

As regards the distribution of bronchiectasis in the lungs, the disease was bilateral in 47 cases and unilateral in 61 out of 108; the right lung was affected in 29 and the left in 32 of the 61. All five lobes of the two lungs were bronchiectatic in 18 of the patients, but there is a tendency for the lower lobes to be more affected than the upper, and in 34 patients with only a single lobe involved the bronchiectasis was in an upper lobe in 8 (right 2, left 6), in a lower lobe in 26 (right 11, left 15). The number of instances in which the different lobes of the lungs were involved in the 108 cases is shown in the following table:

Right upper lobe	50
Right middle lobe	51
Right lower lobe	61
Left upper lobe	49
Left lower lobe	73

Aspiration of the highly septic bronchiectatic sputum into previously uninfected parts of the lung may occur at any time, producing a local aspiration pneumonia or bronchopneumonia. If small in extent, this process may lead to gangrene and excavation of the lung; more or less extensive pulmonary cavities communicating with dilated bronchi were present in five cases. The diagnosis between bronchiectatic and pulmonary cavities is often impossible to the naked eye, though the walls of the latter are usually the more ragged and irregular. Microscopically, however, bronchial unstriped muscle or cartilage will be found in the walls of bronchiectatic cavities. But, if such an aspiration pneumonia is extensive, it is likely to be a terminal event, bringing about the death of the patient by asphyxia or exhaustion before the stage of pulmonary gangrene has been reached or cavities formed. Multiple pulmonary abscesses occurred in three cases.

As would be expected in a chronic septic condition like bronchiectasis, amyloid disease is not rare in it, and was recorded in six out of 103 *post-mortem* examinations. Bronchial calculi were present in the dilated tubes in one case of long standing.

The bronchial secretion in bronchiectasis is copious, purulent, and often possesses strong powers of tryptic digestion. This no doubt explains its highly solvent action on the bronchial walls, which commonly lose their lining of ciliated epithelium and much of the involuntary muscle and the cartilage in their walls in the process of dilating.

Bronchiectasis being always secondary to some other pulmonary disease, the following table shows the primary disorder from which it resulted in 105 fatal cases so far as could be judged from the histories recorded and *post-mortem* examinations:

Prime Cause of Bronchiectasis in 105 Fatal Cases.

Chronic bronchitis	41
Pleurisy or pneumonia	27
Bronchial obstruction by—	
New growth	27
Foreign body	6
Aortic aneurysm	3
Syphilitic stenosis	1

Apart from the fact that all the common cases due to fibrotic pulmonary tuberculosis are excluded from this list, it should be pointed out that these are figures from the *post-mortem* room, and do not represent the relative frequency of the causes of bronchiectasis among the living. Thus, speaking generally, the various forms of bronchial obstruction do not account for anything like one-third of the cases of bronchiectasis met with in the wards; but they lead to a progressive and rapidly fatal type of bronchiectasis, and are associated with other features of great clinical interest, and so occupy a disproportionately large share of the *post-mortem* statistics. The cases in which either pleurisy or pneumonia gave

rise to bronchiectasis have been grouped together, because it is often impossible to ascertain from a patient who had one or other of these diseases many years previously which it was.

With regard to the 27 cases of new growth in the lung compressing or directly obstructing bronchi, it is often impossible to say exactly where such neoplasms originate, owing to the extensive spread of the growth by the time death takes place. The following table can therefore be given only approximately correct:

Nature of New Growths causing Bronchial Obstruction and Bronchiectasis in 27 Fatal Cases.

Primary:	
Mediastinal sarcoma or lymphosarcoma	9
Sarcoma of the root of the lung	8
Carcinoma of the bronchus	5
Endothelioma of pulmonary alveolus	1
Direct spread of—	
Oesophageal carcinoma	1
Scapular perithelioma	1
Secondary:	
From carcinoma of the oesophagus	1
From carcinoma of the duodenum	1

The left lung was affected in 15 cases, the right in 10, and both lungs only in the two instances of secondary invasion. The upper lobes showed bronchiectasis in 3, the lower in 10, and both in 12 of the unilateral cases. In only a few of these 27 cases was the bronchiectasis either a prominent clinical feature or well developed from the pathological point of view by the time the patient came to die. The chief signs and symptoms were mostly those of intrathoracic new growth exciting pressure on adjoining structures, or cachexia. In many the presence of bronchiectasis was hardly suspected *ante mortem*.

PATHOGENESIS.

The pathological anatomy of these 105 cases of bronchiectasis having been outlined, it is now possible to consider the pathogenesis of the condition. In all cases two main factors are at work in the production of bronchial dilatation:

I. Softening and disorganization of the bronchial wall by virulent bacterial infection, acute or chronic.
II. Dilatation of the weakened bronchi by—
  (*a*) Pressure from within (retained secretion, frequent cough, with its high intrabronchial air pressure).
  (*b*) Traction from without (forced inspiration, pleural adhesions, pulmonary collapse and fibrosis).

The way in which these various factors work can best be shown by considering pathogenetically different examples of bronchiectasis.

1. In *chronic bronchitis and emphysema* the patient normally lives for many decades without the supervention of bronchiectasis. Should his lungs, however, become infected by some unusually virulent strain of bacteria, a more acute and destructive inflammation of the bronchi sets in. The sputum increases and becomes more purulent and finally offensive, tending to accumulate and stagnate in the more dependent parts of the lungs. Such a patient is likely to get a symmetrical bronchiectasis, at first of the lower lobes only but later throughout the lungs, the inflamed and softened bronchial walls yielding under the combined strains of retention of secretion, repeated prolonged bouts of coughing, and violent inspiratory efforts.

2. In *pleurisy* the *primum movens* of bronchiectasis is the formation of dense pleural adhesions. Thus fixed the lung is unable to expand, and hence cannot be adequately ventilated by respiration, so that the bronchial secretions cannot be coughed up, but tend to accumulate in the tubes and weaken their walls. The adjacent pulmonary alveoli collapse for want of air, and ultimately the affected part of the lung becomes fibrosed by the slow outward spread of infection from the bronchi. The same condition is reached in one step by *slowly resolving pneumonia*. In both cases the newly formed fibrous tissue in the lung contracts, with the result that the softened bronchial tubes are dilated by traction from without. Bronchopneumonia is often bilateral, and so may give rise directly to bilateral bronchiectasis. Pleurisy (with or without effusion) and lobar pneumonia are usually unilateral, and so are likely to give rise to bronchiectasis on one side only. But in such cases the virulently infected bronchiectatic secretion may be spread, by coughing, into the sound lung, with the production of bilateral bronchiectasis later.

3. In *bronchial obstruction* by the pressure of new growths or aneurysms from without, or by the growth of, say, a carcinoma originating in the bronchial wall and obstructing its lumen, the first impulse toward bronchiectasis comes from stagnation of the infected bronchial secretion behind the obstruction. Such secretion is often not sterile; the infection spreads outwards through the corresponding lung tissue, which becomes collapsed for want of proper ventilation, till finally pleurisy and pleural adhesions are set up. The collapsed lung tissue passes into a state of chronic interstitial pneumonia with the formation of fibrous tissue; this inflammatory tissue contracts, and as it does so increases the dilatation of the septic and softened bronchi. It is in this way that the "aneurysmal phthisis" of the older pathological textbooks, or conversion of one or more lobes of a lung (usually the left) into a matted collection of bronchiectatic cavities, takes place when an aortic aneurysm compresses and obstructs a bronchus. In the three such cases tabulated above the bronchiectasis was confined to the left lower lobe in each; the left upper lobe escaping no doubt because the expectoration of its bronchial secretion was aided adequately by gravity.

It must be mentioned that bronchiectasis is not by any means always seen in cases of bronchial obstruction by new growth or aneurysm. If the bronchi distal to the obstruction are sterile they do not become the seat of bronchitis followed by bronchiectasis and chronic pneumonia; instead, the lung merely collapses.

4. In the case of *foreign bodies in the bronchi*, in addition to the resulting bronchial obstruction, which produces the effects described just above, must be considered the fact that the foreign body is invariably charged with bacteria of all sorts, and is, indeed, highly septic. Thus in one of the cases included in the statistics the sputum was fetid on the second day, and the pulmonary infection ran a rapid course, proving fatal in four months. In such instances bronchiectasis, usually a very chronic process, may truly be described as acute in its onset and course.

There is one point in the pathogenesis of bronchiectasis that affords matter for dispute, and that is whether it is the abnormal raising of the intrabronchial air pressure during coughing and forced expiration, or the abnormal lowering of this pressure during violent inspiratory efforts, if either, that most tends to dilate the weakened bronchial tubes in bronchiectasis. This is a hard matter to decide: a question for physicists. Again, how far may the slight but steady outward pull of the elastic tissue in the lungs be considered responsible for bronchiectasis when the two layers of the pleura are united by adhesions?

SYMPTOMS AND SIGNS.

Bronchiectasis, as we have seen, always occurs as a complication of some other disease of the lungs. The patient is already unwell, and in its early stages bronchiectasis will only add to his troubles the spitting up of increased quantities of sputum which is at times, or in parts, offensive in smell and unpleasant in taste. When the bronchiectasis is well established the patient is often cachectic, with irregular fever, sweating, and all the signs of prolonged septic absorption. He has violent bouts of coughing, particularly on change of position, and may bring up as much as 10 oz. of sputum at a single effort. The sputum is highly offensive—its smell is often compared with justice to that of Limburger cheese—and copious, as a rule, particularly in cases where the bronchial dilatation follows on chronic bronchitis, pleurisy, or pneumonia; here it averages perhaps a pint in the twenty-four hours, and reached 71 oz. in one of the patients in this series. Placed in a tall glass such sputum settles into three layers, as is well known to you. The offensive odour of bronchiectatic sputum appears to be due to the setting free of butyric, caproic, and other volatile acids of the paraffin series from the fatty substances in the purulent sputum by the bacteria of putrefaction it commonly contains.

Haemoptysis is of common occurrence in bronchiectasis, and is often copious. As in pulmonary tuberculosis, it may be an early and frequently repeated sign throughout the course of the disease. It was the immediate cause of death in 5 of the 105 patients tabulated above; in one, however, being due to the rupture of an aortic aneurysm into the left bronchus it was compressing. It had occurred in over 90 per cent. of the patients, and may be taken as proof of bronchial ulceration.

Except for its variety and richness, I believe there is nothing characteristic about the bacterial flora in bronchiectatic sputum, unless it be the frequent presence of influenza bacilli. The offensive sputum contains in addition pus cells, crystals of fatty acids, small yellowish masses known as Dittrich's plugs, and in cases where the dilated bronchi have given way with the formation of pulmonary cavities, alveolar elastic tissue also.

The pulmonary physical signs in the early stages are likely to be those of chronic bronchitis in the symmetrical cases; in the unsymmetrical pulmonary tuberculosis is well imitated. When the bronchiectasis is well developed and the sputum copious, the physical signs vary widely with the amount of sputum present in the dilated tubes at the moment of examination. If the tubes are full the vocal fremitus is lessened, resonance on percussion is much diminished, and on auscultation the breath sounds—which are bronchial, tubular, or even amphoric—will be distant and accompanied by comparatively few adventitious sounds. But if the tubes have been emptied by recent cough and expectoration the physical signs will be very different, because the dilated bronchi are now full of air and in free communication with the trachea. The percussion note over the bronchiectatic lung will be more resonant, the vocal fremitus will be increased and much more than the normal, and on auscultation the loud breath sounds and the extraordinary variety of metallic moist sounds suggest the diagnosis of pulmonary consolidation and excavation. Here again pulmonary tuberculosis may be well imitated; I have met with two cases of obstruction of the bronchus to the left upper lobe by new growth, and in each there proved to be extensive bronchiectasis confined to that lobe, but the physical signs were precisely those of tuberculous infiltration and excavation, though tubercle bacilli could not be found in the sputum.

Some degree of clubbing of the finger-tips was recorded in 70 out of 103 of the cases of bronchiectasis, and may well have been present in more. Clubbing of the toes was present in a few of the patients, and clubbing of the end of the nose also was recorded in two instances. In two cases the clubbing of the fingers was associated with Marie's chronic pulmonary hypertrophic osteo-arthropathy.

Complications.

The most interesting of the complications of bronchiectasis is intracranial abscess, 15 instances of which occurred in 108 fatal cases of the disease. The abscesses were cerebral in 9 instances, cerebellar in 3, both in 2, and in one case meningitis and ependymitis were present, but no intracranial abscess was located. The abscess was single in 9 patients, multiple in 6, and in one instance from 20 to 30 abscesses were present throughout the brain. In addition two cases of secondary intracranial new growth were recorded in this series: one patient had a primary new growth at the root of the left lung, with secondary deposits in the pancreas and brain; the other a primary endothelioma of a pulmonary alveolus compressing the left bronchus, with secondary growth in the brain.

Apart from these intracranial complications, the common complications of bronchiectasis are inhalation or aspiration bronchopneumonia, empyema, gangrene and abscess of the lung, and old or recent pulmonary tuberculosis.

Empyema apparently secondary to the bronchiectasis occurred in 17 of the 108 cases, and its occurrence may be regarded as due to the spread of a more than usually virulent bacterial infection from a dilated bronchus to the pleura; in the other 91 patients the organisms reaching the pleura set up no more than an adherent pleurisy. The empyema discharged itself through a dilated bronchus in 4 cases; pyopneumothorax was present in 3, in 2 the empyema was interlobar, in 1 apical, and in 2 was operated on and drained by the surgeon. In 2 other cases that came up for operation pulmonary abscess, not empyema, was found and drained. Extensive gangrene of the lung was noted in 5 instances, multiple small pulmonary abscesses in 3. Old pulmonary tuberculosis independent of the bronchiectasis was present in 5 cases; recent miliary or caseating tuberculosis was seen in 5 also. Acute pleuropericarditis—an unusual complication—was present in the case of a girl of 15, who had a primary periosteal sarcoma of the femur with secondary deposits in both lungs; the left bronchus was completely obstructed, and the left lung converted into a honeycombed mass of bronchiectatic and bronchiolectatic cavities.

Course and Duration.

The course and duration of bronchiectasis are very variable, depending as they do upon the severity of the bacterial infection present in the dilated tubes. This in turn depends upon the pathogenesis of the condition to a large extent, as will be seen on consideration of the following figures:

Duration of Cough in 105 Cases of Bronchiectasis.

Prime Cause of Bronchiectasis.	No. of Cases.	Duration in Months.		
		Average.	Maximum.	Minimum.
Bronchitis	40	110	600	4
Pleurisy and pneumonia	30	39	144	2
New growth...	24	9	24	2
Foreign body	8	23	60	4
Aortic aneurysm	3	15	24	7

These figures can only be offered with the reservation that they are but very approximately correct. It is not possible to say at what moment bronchitis ends and bronchiectasis begins in any series of cases, which is what the above table attempts to do. There is no clinical criterion to mark the onset of bronchial dilatation. Still, the figures in the column showing the average duration of cough in the cases may fairly be taken as indicating that bronchiectasis secondary to bronchitis or to pleurisy or pneumonia is likely to last for several years; whereas the duration of that due to bronchial obstruction is more likely to be a matter of months.

The causes of death common in bronchiectasis are set out below. This table, too, must be offered with reservations. Thus, the term "exhaustion" might from certain points of view be replaced by "chronic septic absorption," with a low or falling terminal temperature.

The Cause of Death in 110 Cases of Bronchiectasis.

Bronchopneumonia	34
Exhaustion	34
Exhaustion and asphyxia	8
Intracranial abscess	15
Intracranial new growth	2
Haemoptysis	5
Heart failure	3
Intercurrent disease	3
Post-operative collapse	3
Septic diarrhoea	1
Lobar pneumonia	1
Influenzal bronchopneumonia	1

The deaths from exhaustion and asphyxia were all in cases of bronchial obstruction. The intercurrent diseases were uraemia, haematemesis from gastric ulcer, and oxalic acid poisoning.

Diagnosis.

The diagnosis of bronchiectasis is easy enough in a typical case of long standing, with its copious offensive sputum, occasional haemoptysis, its fever and wasting, and the signs of excavation and fibrosis at the base of one or both lungs. But in its early stages there may be nothing to distinguish such a bronchiectasis from chronic bronchitis. Again, when the disease occurs at the apex of a lung, the physical signs may be precisely those of the common apical tuberculosis, the only *ante-mortem* distinction being the absence of tubercle bacilli in the case of bronchiectasis; and even then it must be remembered that an acute pulmonary tuberculosis is not rare as a complication of bronchiectasis. And wherever the bronchiectasis is located in the lung, the physical signs may be just those of tuberculous consolidation, fibrosis, and excavation.

The diagnosis may be very difficult in cases of bronchial obstruction by aneurysm or new growth. It must be remembered that in some such patients the lung merely collapses, and the bronchi, probably for want of infection with sufficiently virulent bacteria, do not develop bronchiectasis at all. In others the bronchiectasis may develop, but little or no sputum (and that inoffensive) may be able to make its way past the obstruction. In all these difficult cases good *x*-ray pictures of the lungs are of great help in diagnosis, the thickened and dilated bronchi being distinguishable as opaque strands converging towards the hilus of the lung.

To illustrate the difficulty of diagnosing acute bronchiectasis such as may follow the inhalation of a foreign body,

I have the permission of a colleague to quote the following case, which I saw while it was under his care at another hospital in 1906:

July 12th. A. E. S., aged 21 months, inhaled a pea into the left bronchus; had a very violent bout of coughing, turned black in the face.

July 19th. Admitted to dispensary; diagnosis of pneumonia made; later, dry paracentesis.

July 28th. Sent on to hospital; diagnosis, (?) empyema.

August 5th. History of inhalation of pea picked up from the floor obtained for the first time from the parents. Skiagram shows opacity of left lung, displacement of the heart to the left.

August 6th. Low tracheotomy, exploration of left bronchus, no obstruction or foreign body found.

August 28th. Paracentesis of left chest, half a drachm of blood-stained serum removed. Impaired note, weak breath sounds, occasional râle over the whole of the left lung; (?) pulmonary tuberculosis.

September 19th. Paracentesis, a little pus withdrawn.

September 20th. Operation, piece of sixth left rib removed; half an ounce of pus drained from a ragged cavity, no empyema found. Tube.

October 2nd. Death from wasting and exhaustion; irregular hectic fever since July 19th, occasional bouts of cough ending in vomiting. The left lung was found to be densely adherent, enlarged, airless, and on section composed of a honeycombed series of small bronchiectatic cavities filled with thick inoffensive green pus, with collapsed lung tissue in between. No bronchial obstruction or ulceration found. Corresponding to empyema operation, a small ragged cavity, into which pus could be squeezed from the adjoining lung.

I imagine that the interpretation of this case would be that the child inhaled a dirty and insalivated pea into its left bronchus on July 12th and developed an acute septic bronchitis on the left side, passing rapidly on to bronchiectasis. The pea must have been got rid of before August 6th.

Treatment.

In all cases inversion of the patient, or letting him cough with his head and chest hanging downwards so as to facilitate the emptying of his bronchiectatic cavities, is a serviceable practice, best carried out on waking. For the rest the treatment of bronchiectasis may be either medical or surgical. The *medical treatment* aims at combating the infection in the bronchial tubes by antiseptics, the most widely used of which is creosote. The creosote may be administered in three ways: (1) By the mouth in capsule form; absorbed from the alimentary tract, some of the creosote is excreted into the lungs, and may there act as an antiseptic. I have never seen much good result from this mode of treatment, and it has the disadvantage of tending to upset the stomach. (2) By intratracheal injection; creosote, thymol, menthol, or some other organic antiseptic, dissolved in five or ten parts of olive oil, is injected into the trachea through the curved nozzle of a syringe, below the vocal cords, after cocainization of the pharynx and larynx. A few drachms of the oily antiseptic can be introduced thus daily, in the hope that some of it will gravitate into the dilated tubes and help to sterilize them and their contents. In the few cases in which I have seen it tried this treatment has not proved strikingly successful. (3) By inhalation; up to a point this method gives admirable results, of a palliative order. The creosote, mixed perhaps in equal parts with eucalyptus oil and oleum pini silvestris, may be given on a Burney Yeo inhaler, worn for many hours a day. A more effective method of administration is to have a small closed chamber in which creosote can be volatilized by heat; the patient, his eyes protected from the pungent vapour by goggles, inhales the white clouds of creosote fumes deep into his lungs. The fumes bring on violent coughing which empties the dilated tubes, and no doubt also exert a beneficial antiseptic action on their infected and inflamed walls. A creosote vapour bath can be given daily, at first for five minutes only, but after practice for fifteen or twenty minutes; care should be taken to see that the fumes are not too strong, and that the creosote is not carbonized by overheating the dish from which it is evaporated in the creosote chamber. The benefits to be expected from this treatment are a great reduction in the quantity of the sputum, which often falls from 20 to 30 ounces a day to 2 or 3; loss of its offensive odour; diminution in the patient's fever; and much improvement in his appetite and general condition. But in the great majority of cases it is palliative only, and relapse is likely to follow its discontinuance. It would be interesting to know the result of the treatment of bronchiectasis by the inhalation of a 2 per cent. solution of, say, Dakin's chloramine-T sprayed into the air by a steam atomizer—a treatment that proved successful a few years ago in the sterilization of the nasopharynx of carriers of the meningococcus.

The *surgical treatment* of bronchiectasis consists in the operative removal of many inches each from a large number of ribs over the bronchiectatic lung, in order that it may be made to fall in completely and obliterate the bronchiectatic cavities by fibrosis and collapse. The operation is obviously very severe, and it has been practised on the Continent, and particularly in Scandinavia, much more frequently than in this country, and usually under local—not general—anaesthesia. It is applicable only to unilateral cases of bronchiectasis, preferably those in which the lower lobe of the lung is involved, and to those not due to bronchial obstruction by new growth or aneurysm. I have hitherto had occasion to recommend it in two cases only, in 1917; in one the result was unfortunate, in the other excellent; the patient has been able to return to work for the last six months, and the sputum has been reduced to half an ounce of inoffensive muco-pus brought up on rising. Three successive partial removals of ribs were performed in this instance, and extensive collapse of the affected lung has been effected.

THE EPIDEMIOLOGY OF PHTHISIS.

BY

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I HAVE read with great interest Dr. Brownlee's recent papers on "The epidemiology of phthisis in Great Britain and Ireland,"[1] and, as they bear upon some of my own work on the climatology of phthisis, I should like to say something about them.

Part I.—Phthisis Death Rates at Different Ages.

First of all I would congratulate Dr. Brownlee on having introduced into the investigation of phthisis-prevalence a new method of comparing death rates at successive age periods—a method likely to yield very interesting and valuable information.

Phthisis not a Single Disease.

Dr. Brownlee concludes, from a study of the age curves of male mortalities, that phthisis can no longer be regarded as a single disease, but that two or perhaps three types are to be recognized, "distinguished by the characters of their incidence at different ages"; death most commonly occurring in one type between 20 and 25, in another type between 45 and 50, and perhaps in a third type between 55 and 65.

Obviously this is a proposition which can only be accepted on the most convincing evidence, for the conception of *age selective* types is quite a different thing from the conception of such types as are met with in the diseases to which reference is made in his paper—namely, enteric, bacillary dysentery, tetanus, pneumonia, and cerebro-spinal fever—types which do not select different ages for their attack. Moreover, since we shall find that the male curves differ from the female curves for the same districts, in such a way that the further assumption is needed that his types of phthisis are not merely age selective, but to some extent *sex selective* as well, the difference between them and the types of those other diseases becomes still more pronounced.

Also it must be remembered that neither clinicians nor bacteriologists have any knowledge of the existence of separate types of "human" tubercle bacilli.

Now nothing has impressed itself so strongly on my mind, in the course of my work on phthisis environment, as the conviction that, if the subject is to be raised out of the chaos of mere surmise in which it has always been involved, and if it is to be placed on a solid scientific basis, a principle, which I have called "the principle of the approximate isolation of influences," must be recognized and applied.[2] By such "approximate isolation" I mean the enumeration of all possible appreciable conflicting influences and their successive elimination from the problem so far as that is possible. I need not illustrate the various ways