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**Bronchiectasis**

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BRONCHIECTASIS
Senior Thesis
1932

Erling S. Fugelso
BRONCHIECTASIS.

DEFINITION:

Bronchiectasis is an infection in a pathological dilatation of one or more bronchial segments, characterized pathologically by various co-existing stages of inflammatory thickening, ulcerative thinning and cicatricial contraction of the bronchial walls — these determine the clinical manifestations of the disease (Hedblom 13). Bronchiectasis may exist as pathological dilatations of the bronchial subdivisions, without infection, but the condition is not recognized clinically until infection supervenes. Whittemore (26) believes that a better term for this condition would be chronic broncho-pulmonary disease with bronchiec-tasis, thus including the concomittant pneumonitis.

HISTORY:

Laennec, in 1819, first recognized bronchiectasis as a clinical entity, describing two cases (24). Again in 1826, Laennec in "De Auscultation Mediate", under the title of "Dilatations of Bronchi", gave the history, clinical condition, and morbid anatomy of four cases. It is notable that three of the cases originated in childhood. He describes minutely the bronchial dilatations associated with fibrous contraction of the lung substance. The compatibility with long life, moderate health and capacity for work are noted. He ascribes the dilatation to be due temporarily, to the voluminous production of sputum and rendered permanent by the constantly successive secretions of similar dilatations. In 1883, Corrigan wrote his "Cirrhosis of the Lung", reporting four cases. He regarded the fibrous changes in the lung parenchyma as the primary change, which produced by traction the dilatations of the bronchial passages.
For a long period of time there was confusion between cases of true bronchiectasis and tuberculous cavitation. (There still is, as will be shown later in this paper.) This existed until the demonstration of the tubercle bacillus was made possible by Koch about 1865. In 1891 Clark, Hadley and Chaplin reported forty-five cases of bronchiectasis in which tuberculosis had been definitely ruled out by repeated sputum examinations. Of this series the majority were alive and enjoying excellent health. Eight were under ten years of age, but a majority of the others dated their original illness back to childhood. In thirty-three cases it had followed measles, whooping cough or both. (21).

In 1905 Clive Riviere published his "Pulmonary Fibrosis in Children", an analysis of thirty-three cases, all of which showed definite bronchiectasis. In twenty-three of this series the original illness occurred before five years of age (21).

The introduction by Sergent and Cothenot in 1922 of contrast-media x-ray for the diagnosis of bronchiectasis, is of particular importance in the early and positive diagnosis of the disease.

INCIDENCE:

This condition is not as rare as it is commonly believed to be, as it may exist without being evident clinically. It is, no doubt, more widespread than indicated by the literature. Ochsner (23), while admitting that bronchiectasis with marked anatomical changes as found at autopsy in advanced cases is relatively rare, states that bronchial dilatation is not rare. He states that bronchiectasis is the most frequently encountered pulmonary infection, occurring even more frequently than pulmonary tuberculosis. Hartung (11) claims that the introduction of iodized oil in roentgenography has shown bronchiectasis to rank second only to tuberculosis in frequency of chronic pulmonary infection. There
is no doubt that many cases of bronchiectasis, non-tuberculous in nature, at the present time, are being diagnosed and treated as tuberculosis, because many patients with persistent cough, lasting over a period of years, resistant to all therapy, have been considered as cases of tuberculosis and have been referred to tuberculosis sanitariums even though the sputum has been repeatedly negative. Hamilton (10) stated that between twenty-five percent and fifty percent of all patients admitted to tuberculosis sanitariums are not tuberculous.

Jex Blake (Priddle 24) in 1920 reported the incidence of bronchiectasis in hospital cases as one and nine-tenths percent and estimated that five percent would be more accurate, as it would include the milder undiagnosed forms of the disease.

Priddle (24) reports an incidence of seven and one-tenths percent of all adults in the Buffalo City Hospital in his series.

Lemon (14) found dilatation of the bronchi in four percent of all the children admitted to the Mayo clinic from 1920 to 1925.

Riviere, Sergent and Pottenger (24 Priddle) point out the frequency of onset in childhood, and Pottenger regards bronchiectasis developing after middle age either as tuberculous in nature or due to an occupational fibrosis.

The age of incidence, however, is in adult life:

<table>
<thead>
<tr>
<th>Jex Blake</th>
<th>series of 100 cases</th>
<th>40 - 60 years majority.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acland</td>
<td>&quot; &quot; &quot; &quot;</td>
<td>10-40 &quot; &quot; &quot;</td>
</tr>
<tr>
<td>Osler</td>
<td>&quot; &quot; &quot; &quot;</td>
<td>20-40 &quot; &quot; &quot;</td>
</tr>
<tr>
<td>Elliott</td>
<td>&quot; &quot; 40 &quot;</td>
<td>40-60 &quot; &quot; &quot;</td>
</tr>
</tbody>
</table>

Males seem to be more often affected than females, the ratio being placed as high as four to one (24). Lilienthal (15) says they are equally divided.
ETIOLOGY:

Bronchiectasis may be congenital or acquired. D. T. Smith describes a primary bronchiectasis, which also will be briefly considered in this paper.

I Congenital Bronchiectasis.

This type is not recognized clinically until it becomes infected, and naturally it cannot be prevented.

Some of the theories are as follows:

A. Kaufman calls it an arrested development, "an agenesis of the alveoli." (Hedblom 12).

B. Stoerch believes the histology in some cases is that of a congenital fetal adenoma (12).

C. Baker and Grandhomme believe intrauterine syphilis plays a part. (12)

D. Sauerbruch and Lotzin are especially firm believers in the congenital origin of bronchiectasis. They maintain it is due to an interference in the development of the lower main bronchus, usually the left. The bronchus rides upon Cuvier's duct (which connects the primitive peripheral veins with the venous sinuses of the heart), and a persistent or unusually high duct might interfere with the development of the bronchus. (Eloesser 6)

E. Eloesser (5) gives an interesting discussion of congenital cystic disease of the lungs and its relation to bronchiectasis. Cysts of the lungs are of two types: solitary cyst and cystic disease proper. The latter is the most frequent and is also termed congenital bronchiectasis, honeycomb lung and saccular degeneration of the lung. In this form a whole
or part of one lobe, or even a whole lung, may show various
degrees of cystic degeneration ranging from multiple miliary
cysts scattered through a normal parenchyma to conversion of
a lobe or an entire lung into a huge multi- or uni-lucular
cyst.

Mild degrees of cystic degeneration accompanied by more
or less atelectasis have escaped clinical recognition, but
have been found at post mortem. The more prominent cystic
changes with saculation or clubbed dilatation of the bronchial
endings are subject to controversy. Gräwitz and others believe
most of these to be congenital. They may be present at birth
or develop in the first few months of life. Heller, Orth and
others believe they are due to persisting fetal atelectasis
in which the expanding thoracic cage, finding no alveolar
tissue to exert its expansive force upon, expands the bronchi.
Sauerbruch, Kössling and Borst consider congenital cystic
disease much more frequent than ordinarily assumed. However,
Huebner and many other German pathologists believe congenital
atelectasis is rare.

The problem is difficult because the cysts of themselves
give no symptoms. The acute disease thought to be the cause
of the bronchial dilatation in a given case often is merely
the factor which changes an aseptic and symptomless cystic
lung into an infected and suppurating one, manifesting the
disease.

Thus, it is not always possible, in a given case, to determine
whether the origin was congenital or acquired. However, the
congenital origin, in some cases can be proven, for cystic
dilatation has been found in the fetus and in the new-born. Grawitz distinguishes two types; 1. a universal bronchiectasis involving a main bronchus and all its side branches, forming a central cyst with many small cysts debauching from it; 2. telangiectatic bronchiectasis in which smaller bronchioles are cystically dilated, some dilatations being open, some closed, and some strung bead-like after one another on the thread of an occluded bronchiole. Sauerbruch and Lotzin found that congenital cystic bronchiectasis frequently presented irregular conglomerations of cysts without relation to a bronchial tree, that these are lined by a single layer of ciliated epithelium, and that the walls are free from ulceration and inflammation. The cystic areas may be free from carbon and inhaled pigments in the congenital form, whereas, in the acquired form the bronchi are inflamed, ulcerated and pigment is present.

The congenital origin of the most marked degrees of sacculation admits of no doubt.

II. Causes of Acquired Bronchiectasis.

A. Within the lumen of the bronchus, the factors are chiefly due to aspiration of foreign bodies and infective material. Aspiration commonly occurs during tooth extraction, tonsillectomies, operations in the upper-respiratory tract, under general anaesthetics, during alcoholic intoxication, deep sleep, comas, epileptic seizures, submerging, as in the case of drowning, and in regurgitation of food, as from an esophageal diverticulum. The aspirated material may give rise to partial or complete obstruction of the bronchus, with dilatation and accumulation of secretions distal to the block.
Other factors within the bronchus are the irritation due to poisonous gases and stagnation of secretions.

B. Factors affecting the Bronchial Walls

1. Ochsner (23) believes that the bronchial secretion is normally bactericidal, and that an alteration of the secretion with lessening of the bactericidal power permits the growth of organisms tending toward weakening of the bronchial wall, resulting in dilatation.

2. The acute infectious diseases of childhood are important etiological factors. Bronchopneumonia, measles and pertussis are the chief causes, but diphtheria, bronchitis and others also may be factors. Thorpe (31) reports fifty-three cases under thirteen years of age as follows:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Cases alone</th>
<th>Cases accompanied by other diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchopneumonia</td>
<td>23</td>
<td>26</td>
</tr>
<tr>
<td>Pertussis</td>
<td>14</td>
<td>17</td>
</tr>
<tr>
<td>Measles</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td>Influenza</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Repeated upper respiratory</td>
<td>2</td>
<td>48</td>
</tr>
</tbody>
</table>

Of these, thirty-eight showed residual sinusitis, otitis and pharyngitis.

Boyd (2) also studying the disease in children, found a similar relationship between these factors, except that he places bronchitis next to broncho-pneumonia in the order of frequency. Several other writers also have made similar observations. Ochsner (23) notes a marked increase in bronchiectasis, dating from the influenza epidemic of 1918.

3. Severe bronchitis, peribronchitis, recurrent attacks of
these, recurrent attacks of bronchopneumonia, delayed resolution of lobar pneumonia, and similar infections damage the epithelial lining, weaken the walls and produce peribronchial fibrosis. (3). Ochsner (22) found ninety percent of so called chronic bronchitis showed evidence of bronchial dilatation on bronchographic examination. Edwards Graham and Singer (27) state that all chronic suppurative conditions of the lung show an associated bronchiectasis of a greater or less degree.

Riviere (Priddle 24) believes that many bronchopneumonias, in adults, are but acute exacerbations of bronchiectatic processes, following a childhood broncho-pneumonia which caused permanent fibrosis and bronchiectasia in the lung bases.

4. Rist (Ochsner 23), in 1916, is credited with first establishing the association between chronic cough and sinusitis. Brown (3) states that eighty percent of chronic pulmonary suppuration is accompanied by sinusitis. Graham and Singer (27) believe that chronic sinusitis increases the symptoms of bronchiectasis, and, conversely, the clearing up of the sinuses, markedly reduces the amount of sputum. Quinn and Meyer (25) found it present in fifty-seven and nine-tenths percent of their series and also observed that the majority so affected gave no symptoms of the sinusitis. The mode of infection is by aspiration and lymphatic drainage. Lilienthal (15), Eloesser (6), Priddle (24) and Whittaker (35) also stress the etiological importance of upper respiratory infection. Ochsner (23) goes so far as to state that there is a possibility of bronchiectasis developing in every persistent case of sinusitis.

5. Neoplasms of the bronchial wall narrowing the lumen is a rare cause.
C. Factors Extrinsic to the Bronchus.

1. Pulmonary fibrosis due to tuberculosis or occupational diseases may give rise to bronchiectasis in advanced years.
2. Atelectasis, either fetal, or due to a blockage of the bronchus.
3. Pleural adhesions, thickened pleura, empyema and pulmonary empyema.
4. Pressure on bronchi, due to mediastinal glands, pleural effusions, hepatic and splenic tumors, sub-diaphragmatic abscess, enlarged heart, pericardial effusions and ascites.

III. Etiology of Primary Bronchiectasis.

D. T. Smith (29) considers a bronchiectasis primary when the dilatation cannot be ascribed to any previous or underlying pathological process of lungs or bronchi, excluding that due to B. Influenza and the congenital cystic type. The essential lesion being a destruction of the elastic coat of the bronchi, due to focal necrosis.

The infecting organisms are the fusospirochaetal group of microbes composed of treponema microdentium, Trep. Macrodentium, S. Vincenti, S. Buccalis, fusiform bacilli, and vibrios and cocci all common to pyorrhea alveolaris, Vincent's agnina, pulmonary abscess, and pulmonary gangrene. These organisms are constantly present in the sputum of active bronchiectasis and can be found on deep sections of the diseased bronchi. They will reproduce the disease in rabbits.

J. K. Smith (30) believes the spirochaetes are merely ingrafted and are not the cause of the disease.

PATHOLOGY AND PATHOGENESIS:

Acute Bronchiectasis

According to McNeil et al (21) a common post mortem finding in
children dying of acute broncho-pneumonia is a widening of the lumina of the small bronchi, especially in the consolidated patches. This is termed acute bronchiectasis or bronchiolectasis. It may be produced either by ulcerative bronchitis, destroying the bronchial wall with permanent changes, or by dilatation due to weakening, without structural change. This later condition occurs in the smaller bronchioles and is analogous to acute emphysema of the alveoli, which often accompanies it. This condition is capable of complete, spontaneous recovery.

**Chronic Bronchiectasis**

Ochsner (23) believes there is a functional bronchiectasis, occurring before anatomic changes take place, due to atony of the bronchial musculature brought on by bacterial toxins aided and abetted by stagnation of secretions. Later anatomic changes take place.

The dilatations may be cylindrical, fusiform or saccular. The cylindrical type is more commonly the result of inflammation, whereas the saccular type is more often regarded as congenital in origin.

Ballon and Ballon (1) list five roentgenological types of dilatation based on injection of iodized oil:

- Grape
- Clubbing
- Cylindrical
- Saccular
- Beaded

The location may be unilateral, bilateral, localized or diffuse. It usually affects the lower lobes, because of stasis of secretions. The left lower lobe is most frequently involved (between eighty and ninety-five percent), because of congenital malformations, more acute angulation of the bronchus impeding the discharge of secretions and because of constriction due to the crossing of the left pulmonary artery over the main
bronchus. About fifty percent of the cases are bilateral. (23). In all cases the bronchial wall is intensely inflamed and thickened by the accumulation of inflammatory exudate. The mucosa is thick and ragged. The musculature and elastic tissue spread apart, torn and weakened, and the connective tissue, as well, is infiltrated with fluid and round cells. The cartilaginous plates tend to disappear and a newly formed connective tissue occupies the bulk of the wall—all tending to diminish the strength and elasticity of the wall. (McCallum 19)

The lung tissue is often emphysematous, but may be converted into a dense fibroid substance, partly by the organization of exudate in the alveoli and partly by scar tissue formation in the parenchyma itself (19).

McNiel, MacGregor and Alexander (21) have a different conception of the cavity formation. They believe that the initial change underlying the condition especially following acute respiratory diseases in children is an acute interstitial inflammation of the bronchial wall, which goes on to necrosis and suppuration. This causes the formation of a cavity by loss of tissue from the bronchial wall and adjacent alveolar tissue. The size, shape and location of the cavities depend entirely upon the extent of the lesion and its relation to the circumference of the bronchiols. Subsequently, the cavity is lined by granulation tissue, which becomes fibrous and is finally covered by modified bronchial epithelium. Thus, the principle mechanism in the cavity formation, is excavation of tissue, rather than, the usually accepted concept, of dilatation of a weakened bronchial wall.

Priddle (26) believes that the fact that the bronchial cartilaginous rings are not complete, and that the smaller bronchi the less cartilage in each ring, may be important factors in pathogenesis.
The factors usually considered in the pathogenesis of the bronchiectatic dilatations are: 1. Infection weakening the bronchial wall; 2. Mechanical force producing dilatation.

Sources of mechanical force:

1. In the absence of pathology in the parenchyma or pleura, atmospheric pressure is 10-20 cc of water more than the intra-bronchial pressure. Normally the elasticity of the bronchi compensate for this, but with inflammatory changes in its wall, the bronchi lose their elasticity and dilate.

2. The deep inspiration, just preceding a cough, produces a maximum difference between the intra-bronchial and intra-pleural pressure. The actual act of coughing has no dilating effect, as the increased intra-bronchial pressure during this act is supported from the outside, by the force causing the increase.

3. Partial obstruction of a bronchi may produce dilatation in a manner analogous to emphysema

4. Pulmonary atelectasis, congenital or acquired, by creating an increased negative pressure intra-pleurally is an important factor. Additional mechanical factors are the imposed stress in the shifting of the mediastinal structures to the affected side, elevation of the diaphragm and retraction of the thoracic wall. Atelectasis, if it persists, produces the most marked degrees of sacculation and dilatation observed.

5. Scar tissue retraction in the lung parenchyma is a source of extrabronchial traction resulting in dilatation. This is not as a rule very marked, due to the decreased expansion of the affected lung.

6. Chronic fibroid phthisis causes the condition due to scar tissue
retraction.

7. Chronic pulmonary abscess combines scar tissue pull and atelectatic pull. In chronic fibroid diseases of the lungs and in some types of atelectasis, dilatation may precede infection, but infection usually precedes as it is concomitant with the mechanical factors (12).

SYMPTOMOLOGY:

The symptoms vary with the site of the lesion, the adequacy of drainage and the severity of the superimposed infection.

Acute bronchiectasis is rarely recognized due to the presence of the acute exciting disease.

**Cough:** The cough and expectoration are quite characteristic. The cough is chronic with periods of amelioration. It is paroxysmal in character, usually on arising in the morning or brought on by change of posture. With the evacuation of the sputum in the morning, the patient may pass the day in comfort. Lying down often aggravated the cough, due to the overflow of pus from its cavity.

**Sputum:** The amount varies from a few c.c. to a liter. Children tend to swallow it. It is usually purulent, may be blood streaked or contain frank hemorrhage. Theoretically, it separates into three layers -- a thick granular layer below, a thin mucoid layer in the middle and capped by a brownish froth. It may or may not have a foul odor.

**Hemorrhage:** Occurs in about fifty percent and varies from a trace to several ounces of blood. It is due to the erosion of a blood vessel.

**Dyspnoea** is only found in advanced cases and is a sign of complications, such as dilated heart, emphysema, extreme fibrosis of the lung or fixation of the diaphragm.

**Cyanosis gives a poor prognosis.**

Dyspnoea and cyanosis occurring in spells, is characteristic of congenital
cystic disease in children.

Pain only occurs in cases in which the pleura is involved.

Circulatory changes are noted in the form of clubbing of the fingers and toes. While this occurs also in other conditions, it is one of the commonest findings in bronchiectasis, in advanced stages.

Temperature in moderate degrees of bronchiectasis, ranges from normal to 100. Children are more likely to show a fever than adults. The temperature rises with impairment of drainage and in complications.

Pulse and Respiration are normal in cases of moderate degree.

Blood count shows slight reduction in hemoglobin and a moderate polymorphonuclear leukocytosis. In children a rather marked anemia is commonly found. Constitutional state in moderate degrees may be good. With progression of the condition, toxic absorption takes place, general debility and finally cachexia occurs.

Physical signs:

These vary greatly depending on the extent and location of the lesion, and the co-existence of complicating conditions. The signs are usually found in the bases. The commonest finding is the persistant presence of coarse, moist rales in the bases. There may be dragging of the affected side more noticeable at the base. Percussion changes only occur late, in the presence of pneumonitis (23). Lemon (14) claims curvature of the spine occurs in children.

Course:

The course is usually protracted. Lemon (14) and Thorpe (31) observe that children are stunted, underweight, impaired in strength and more susceptible to epidemic and endemic infections, being generally handicapped for life. A large percentage of children so affected show a marked anemia. In general, it may be said that the condition is generally progressive, unless adequate treatment is instituted.
DIAGNOSIS:

1. A history of a chronic cough with purulent or muco-purulent sputum, expectorated during a paroxysmal attack of coughing, especially incident to stooping or change of posture, is the characteristic syndrome. The onset may date back to an acute respiratory disease, but the etiology in at least twenty-five percent is obscure. General health is usually rather good. Some cases are characterized by recurring attacks of chills and fever (18)

Boyd (2) believes that bronchiectasis should be diagnosed in children when:

a. Cough, particularly with sputum, persists more than a few months after such illnesses as pertussis, measles, influenza or broncho-pneumonia; particularly if the cough is accompanied by hemoptysis and night sweats.

b. In so called chronic bronchitis, especially if not relieved after a clean up of the upper respiratory tract.

c. In repeated attacks of broncho-pneumonia, after the eradication of foci of infection.

A history of prolonged bronchitis demands x-ray studies. (16 and 22)

2. Always inquire as to surgical procedures of the naso-pharynx preceding the attack.

3. Make routine examination of the accessory sinuses and sputum.

4. Bronchoscopy (4, 16, 17)

Locate presence, extent and site of lesion and the patency of the bronchi. It permits of the evacuation of the secretion from the pocket, detects any inflammatory condition along the course of the bronchial tree. It permits of equal and bilateral placing of iodized oil for roentgenography. It is a means of obtaining uncontaminated cultures and biopsy material.
5. Physical signs are inconstant and only suggestive.

6. Roentgonological examination (Hartung 11)

Findings on plain film are:

a. Increased linear markings -- the commonest finding.

b. Irregular densities obscuring heart outline or dome of diaphragm -- correspond to cul-de-sacs shown by iodized oil injections.  
(Singer and Graham (20) and Thrope (31) note that the right cardiac angle is often obscured by an atelectatic lobe.)

c. Irregular densities in lower lobes.

d. Displacement of heart shadows without demonstrable cause.

e. Localized densities suggestive of atelectasis.

f. Generalized densities and radiolucent areas.

g. Honey-comb appearance of advanced cases.

Concludes that:

a. Sixty-percent of bronchiectasis can be diagnosed on plain film.

b. Majority of the remaining forty percent will show suggestive findings.

c. Contrast media roentgonography is absolutely essential for obtaining accurate information relative to the nature, location and extent of bronchiectatic dilatations, and with its use, early detection, in a stage amenable to treatment, will be possible.

DIFFERENTIAL DIAGNOSIS: 
(3, 12, 35, 20)

1. Chronic bronchitis is usually considered a concomitant condition -- particularly so in view of Ochsner's findings (22) in cases of chronic bronchitis studied by contrast media roentgonography, showing that ninety percent of his series showed bronchial dilatation.

2. Pulmonary phthisis - In bronchiectasis the history is of a chronic
cough over a period of years, with expectoration, yet with the patient in a fairly good state of health. The absence of tuberdl bacilli in the sputum is valuable. Tuberculosis is usually located in the upper lobes, while bronchiectasis is usually basal, but either may be diffuse. Clubbing of the fingers is more frequently found in bronchiectasis. The x-ray is very valuable. Tuberculosis shows a leukopenia while bronchiectasis tends to a polymorphonuclear leukocytosis, ranging from ten to eighteen thousand.

3. Pulmonary abscess - In this condition the patient is usually acutely ill, or has just recovered from an acute illness. There is sudden expectoration of a large quantity of muco-purulent material with a characteristic sweetish odor. The patient appears ill, runs a septic temperature and shows a higher leukocytosis than bronchiectasis, as a rule. The sputum is more of a prune-juice variety. Diagnostic puncture and contrast media x-ray should be employed. Multiple small abscesses are difficult to differentiate from bronchiectasis.

4. Empyema, with a bronchial fistula, has a history of acute onset and is usually preceded by pleurisy or pneumonia. There may be displacement of the mediastinal structures. Again x-ray should be resorted to.

5. Pulmonary gangrene has a history of sudden onset. It may be a sequela of pulmonary infarct, tuberculosis, broncho-pneumonia or spirachaetal bronchitis. It may be difficult to differentiate - presence of elastic tissue in the sputum favors this condition.

PROGNOSIS:

In childhood Findlay and S. Graham (8) draw the following conclusions:

1. In children the prognosis is grave, as the condition usually gets worse and leads to a fatal termination.
2. Undoubted bronchiectasis (bronchiolectasis or acute bronchiectasis) following a chronic pneumonia may disappear, but only when the degree of dilatation has been slight.

3. The age of the onset seems to influence the course -- recovery being more probable in cases developing later in childhood.

4. In childhood the duration of the disease is of no prognostic aid.

Lilienthal (15) states that the outlook in the long established case of suppurative bronchiectasis is far from cheerful. He has seen but few cases improve under medical treatment. The mechanism of spontaneous healing is not known for sure, but surely can not occur after fibrotic changes have occurred to prevent the collapse of the cavity. The majority, however, while showing temporary remissions at times, progress steadily downward. Without surgery there is continued sepsis, more or less severe (with its dangers including abscess of the brain), with pains in the joints and epiphyses, hemoptysis, secondary lung abscess, secondary empyema and nephritis.

The only hope in chronic cases, without surgical intervention, is for the gradual development of fibrosis, which will get rid of much of the constitutional disturbances and greatly reduce the amount of sputum, but cannot completely stop the cough. There may be a diminution, but not a disappearance of the bronchial dilatations.

COMPLICATIONS:

The commonest complications are broncho-pneumonia, chronic fibrous pleurisy and pulmonary abscess. Less commonly empyema, meningitis and the arthritides occur. (24, 15).

Wm. B. Faulkner (7) claims internal drainage as the chief cause of death, post operatively in bronchiectasis and other thoracic surgery. Internal drainage is the spilling over of intra-bronchial secretions from
the diseased bronchi into neighboring bronchi of either lung. This results in a widespread dissemination of infection and mechanical obstruction to the flow of air. The mechanical action of the secretion, depending on position and amount, may result in an emphysema, obstructive atelectasis or even massive collapse of the lung. Faulkner believes many deaths previously ascribed to cardiac failure, bronchopneumonia, cerebral or pulmonary emboli and operative shock were primarily due to internal drainage, which can be prevented by careful choice of anaesthetic, selection of proper posture, attention to hemostasis and removal of excess intra-bronchial secretions.

**TREATMENT:**

**Prophylaxis.**

This may be accomplished in a certain class of cases by oral hygiene previous to operation, and by prevention of aspiration during surgical procedures in the naso-pharynx, during tooth extractions, and during acute illnesses.

**Medical Treatment:**

The value of non-surgical treatment of bronchiectasis is subject to much controversy. Whittemore (36) states "the medical treatment can be dismissed by saying that it cannot cure any case, although, if the patient can devote his life to taking care of his health, spending his winters in a dry, warm climate, using postural drainage, it may be that he will live a long and fairly comfortable life. There is no drug that has any influence upon the disease."

Riviere (26) on the other hand champions the medical treatment, believing it best to be satisfied with a moderate result without endangering the life of the patient.

A. Postural drainage, accomplished by hanging over the side of the bed ten to fifteen minutes a day, aids in evacuation of the cavity
and is usually practiced by the patient without instruction. It is merely palliative.

B. Lipiodal is not only invaluable as a diagnostic measure, but also is accorded considerable merit by a host of observers in the treatment of the condition in that it:
   1. lessens the amount of expectoration.
   2. reduces the number of bacteria in the sputum.
   3. lessens the toxic symptoms, and
   4. reduces the dilatation.

Weinberg (34) believes that although lipiodal does not exert a bacteriocidal action, as such, it does do so when it comes into contact with tissue, due to the liberation of iodine. He reports eight cases improved by this treatment in conjunction with phrenic nerve exeresis.

Reports from the University of Minnesota (33) state that the known good results obtained there have been with lipiodal and that all radical measures have proved fatal.

The contraindications for the use of lipiodal are:
   1. acute pulmonary tuberculosis,
   2. extensive advanced general pulmonary suppuration,
   3. angina pectoris and cardiac decompensation,
   4. aneurysm,
   5. hemoptysis,
   6. acute inflammatory involvement of the upper respiratory tract.

Eloesser (6) and others do not believe that it has any therapeutic value.

C. Bronchoscopic treatment is advocated by many. Bronchoscopic
removal of foreign bodies, secretions plugging a bronchus, new growths, granulations and stenosis, while the alveoli are still intact, will result in their reinflation and this will relieve the existing dilating strain (14). With the bronchoscope, one can locate the size and extent of the lesion and the patency of the bronchi. One may also aspirate the contents of the cavities and make local application of medicaments (4, 16, 17).

Martin (17) reports sixty-three cases treated, with fifteen cures. He claims its value is only in early cases and is surely more effective than postural drainage. Bronchoscopy is contraindicated in advanced cardiac disease, advanced pulmonary gangrene, emphysema, pneumothorax and hypertension. It has the additional disadvantage, in that it requires a trained specialist, and even in such hands, is traumatic to some degree.

D. D.L. Smith (29) and Graham (9) recommend careful sputum examinations for spirochaetal organisms, for they believe, that in a few selected cases, the use of neosalvarsan or other arsenicals have been of value.

E. Other palliative therapeutic measures that have been employed are: Ammonium chloride to thin out secretions and make coughing easier; monochlorphenol 2% in an atomizer to relieve upper respiratory irritation; compound tincture of Benzoin, used to relieve bronchial tree irritation; cresote, used in the hope that it would exert an antiseptic action, on being excreted thru the lungs. A thirst cure was advocated at one time. However, the dehydration thus produced to lessen secretions is not desirable.

**Surgical Treatment:**

A. General considerations.
In the past the operative treatment was doubtful procedure. The operative mortality was high and the results, in a majority of cases, were merely palliative, due to the incomplete and faulty diagnosis of the extent and location of the pathology and the consequent inability to choose the type of surgery best adapted to the case. Also, (due to the belated diagnosis) thickening, ulceration and cicatricial stenosis in the bronchi accompanied by sclerosis of the lung parenchyma, incident to the prolonged suppuration, combined to lessen the effectiveness of operative compression of the lung.

Myocarditis and nephritis (due to chronic toxic absorption) and dilatation of the right heart (from increased venous pressure) have contributed, largely, to the post-operative mortality. Injury to these vital organs is perhaps greater than clinical manifestations might indicate.

The introduction of the contrast-media x-ray, making possible early diagnosis and aiding the selection of suitable surgical methods for the individual case, while the process is limited and the tissues elastic and before the vital organs are severely injured, should tend to improve the effectiveness of the surgical treatment.

The principles of treatment are: Drainage, Pulmonary compression and extirpation of the diseased portion of the lung.

A. Drainage may be accomplished by posture and bronchoscopy, as discussed under the medical treatment. Surgical drainage or bronchostomy (15) consists of a deliberate formation of a muco-cutaneous opening into a good sized bronchial
branch. It is usually followed by a relief of the symptoms, but the fistula does not heal spontaneously, and no attempt should be made to close it, until one can be reasonably sure that sufficient contraction of the entire tract has taken place and that the adjoining bronchiectatic condition has undergone fibrosis. The indications for this operation are: 1. temporary expedient, preceding more radical procedures; 2. in a sudden change in the clinical picture, characterized by high fever and very foul sputum, indicating perforation of the bronchiectatic cavity into the lung parenchyma, with resulting gangrene, where prompt drainage may save life; 3. in bilateral bronchiectasies, with a bad turn in the clinical picture, where bronchiostomy, upon the worst side, may preserve life, indefinitely. Meanwhile, fibrosis may arrest the disease in the opposite lung.

B. Compression treatment (6, 12, 13, 15, 27, 30).

The older literature maintained that the bronchiectatic lung could not be collapsed, due to the marked sclerosis of the parenchyma and the incompressability of the dilated bronchi. However, we now know these tenets are not true, because x-ray shows that the sclerosis, as a rule, is not marked, the smaller bronchioles, with the least cartilage, are affected and finally, necropsies have revealed extreme degrees of fibrosis and shrinkage, with obliteration of the affected bronchi, following the collapse technique. The principles of pulmonary collapse are:

a. To put the diseased lung at rest, slowing the lymph stream and thus the rate of toxic absorption, the
presence of which is evidenced by the myocarditis, nephritis, arthritis and amyloidosis in advanced cases.
b. Fibrous tissue is permitted to contract and the collapsed state brings about further fibrosis.
c. The collapse of the bronchiectatic cavities, even though only partial, lessens the amount of purulent secretion, markedly.
d. Mechanical and cicatrical contraction of the dilatations promotes healing of the opposing ulcerated surfaces.
e. The lessened cough and secretions along with the fibrous occlusion of the bronchial lumen prevent the dissemination of the disease and tend to lessen hemorrhage.
f. Surgery thus neutralizes mechanical dilating stress, whose tendency is to change the persistent stress even in comparatively inactive cases.
g. If scar tissue stress is due to fibroid phthisis, this is merely an added indication for collapse therapy.

Methods employed in collapsing the lung are:

a. artificial pneumothorax,
b. phrenico-exeresis,
c. extra pleural thoracoplasty,
d. major thoracotomy,
e. upward displacement of the lung by Garre.

1. Artificial pneumothorax is of value in early unilateral disease, particularly in children. It is relatively safe, simple and permits of ultimate restoration of lung function. It can not be used in the presence of adhesions or very marked parenchymatous changes in the lungs. Its disadvantages are the need of frequent refills and the results
are merely palliative.

2. Phrenico-exersis, extraction of the phrenic nerve, results in paralytic relaxation of the diaphragm, on the affected side, bringing about a partial collapse of the lung, estimated at fifteen to thirty percent of its original volume or equal to a pneumothorax of two hundred and fifty to five hundred c.c. It is simple and safe and is of value where pneumothorax is not possible, due to adhesions. The rise of the diaphragm, compressing the lung facilitates the evacuation of sputum and tends to lessen the cough.

Alexander (Riviere 26) is of the opinion that this procedure makes for greater ease in expectoration due to the fact that the abdominal muscles can exert a greater propulsive force through the paralyzed leaf of the diaphragm, in coughing.

Singer and Graham (27), while advocating this procedure, in early lesions, warn us that it is not without danger, as it sometimes seriously interferes with the evacuation of pus, by coughing and occasionally results in severe suppurative pneumonia.

Results ranging from no improvement to complete symptom-free cures have been reported by Chaufford, Ravina, Davies, Rist, Bishop and others (Hedblom 13). It should be employed in early unilateral lesions. Best results are obtained in the dilatations of larger bronchial subdivisions, laterally situated. While it does not have such a direct effect on centrally located
lesions, it, at least, lessens mechanical tension, thus retarding the progress of the disease. It is also indicated as a preliminary procedure to thoracoplasty and is employed in cases, in which operative procedures on the thoracic wall are contraindicated, due to toxicity, sepsis, cyanosis and marked dyspnoea.

3. Extra-pleural thoracoplasty (13) involves the collapse of the diseased lung, by means of extensive rib resections. The collapse is relatively complete and permanent, is not limited by adhesions, presents but little danger of emphysema and paves the way for later lobectomy or cautery extirpation. It is preceded by phrenico-exeresis. Sauerbruch does a posterior para-vertebral resection of the upper eleven ribs -- procedure favored by Hedblom and Eloesser, but not by Edvart Graham. Sauerbruch also combines ligation of the pulmonary artery with secondary thoracoplasty, thus obtaining shrinkage of the lung due to fibrosis. This operation is done in several stages. Other operators resect several of the lower ribs. It is indicated in the early peripheral unilateral cylindrical lesions, not helped by phrenico-exeresis. Cylindrical involvement of the larger bronchial subdivisions, while less influenced directly, shows less peripheral extension.

The nodular type of bronchiectasis, associated with tuberculosis, presents a double indication. Marked mediastinal displacement and marked retraction of the chest wall, due to fibrous contraction in the cirrhotic type of bronchiectasis, constitutes an additional mechanical
indication. Early thoracoplasty in an etiological atelectasis, with great increase in intra-pleural tension, will prevent the formation of large sacules.

Thoracoplasty is least valuable in the large sac- cular dilatations, lying posteriorly and close to the mediastinum.

4. Major thoracotomy (15) may be done in several stages, depending on what pathology is encountered. It is primarily an exploratory operation. It consists of a long intercostal incision with resection of a few inches of one rib with its periosteum. Adhesions are broken down to permit the lung to collapse and to do away with intra-pleural negative pressure. If small abscesses are present, they may be opened, and one may even open into a bronchus (bronchostomy). The lung is then collapsed by gauze packing. This procedure is best suited to lesions situated at or near the hilus. It is an operation adapted to old age. The benefits, of course, are due to the collapse and subsequent cicirization of the affected lobe, yielding a permanently decreased volume. The patients blood pressure must be over one hundred and twenty-five systolic, and he must be in general good condition to withstand a subsequent anaerobic infection. This procedure is recommended by Lilienthal.

5. The upward displacement of Garre is accomplished by suturing the base of the lung to the thoracic wall above the level of the dome of the diaphragm and pack beneath it with gauze. At a later date this lobe
may be resected (13).

6. Extirpation Treatment

All the preceding methods, with the possible exception of Gare's, have been merely palliative in intent although, sometimes they resulted in unexpected cures. The one procedure calculated to produce a complete restoration of health is that of resection of the complete diseased lobe. This procedure (lobectomy) in Lilienthal's (15) hands carries a mortality of forty-five percent. The risk is one which, if successful, offers a cure with little or no apparent deformity and with excellent function, physical and general. Also, resection prevents malignant degeneration of the lung.

The types of lobectomy vary. Lilienthal (15) prefers lobectomy done in several stages. Sauerbruch and Edwart Graham use a cautery extirpation, following rib resection. Graham reports a series of twenty cases, in which thirty percent were cured, twenty percent were symptom free, with a fistula, fifteen percent were improved, fifteen percent not fully determined and twenty percent operative mortality. Whittemore (37) reports four cases in which he sutured the lobe externally to the thorax and permitted it to slough away. Two cases were cured, one well with fistula and one died. Sauerbruch reported twenty-three cases, in which he caused the lobe to slough out by compression, around the mobilized lobe, with a rubber band, the gangrenous mass sloughing in two to three weeks. Three died and the
others healed completely.

Lobectomy is indicated where a lobe is totally replaced by undrainable bronchiectatic channels and cavities and by fibrosis. Restoration of function is not possible, nor is obliteration. Simple drainage is useless and the focus of infection can only be cleared up by extirpation of the lobe. The patient must be under thirty-five years of age and in good condition. Affection of other lobes need not be a contraindication, if the other lung is sound. The pathology encountered will determine the stages of the operation. Cardiac, renal, or grave metabolic diseases, pulmonary tuberculosis and Wasserman positive lues are strong contraindications for lobectomy.

II. In general, before treating bronchiectasis, in any manner, one should investigate and clean up any existing nasal-sinus infection. Simple uninfected cases of bronchiectasia should have attention only to the upper respiratory tract. Simple infected bronchiectasia may be given a trial pneumothorax and phrenic avulsion, before more radical surgical measures are employed, except in rapidly fulminating cases. Postural drainage, for a few days preceding operative intervention, relieves the patient of fatigue, due to excessive coughing and lessens the danger of internal drainage during and following the procedure (6).
CASE REPORTS.

Case 1.

A white school boy, age 16, entered the hospital 2/2/31, complaining of:

1. Cough, most productive in the morning.
2. Foul smelling sputum and breath, making school attendance, impossible.
3. Pain in the lower right chest.

The onset was in 1926. Following an attack of influenza, he developed a slight, chronic cough, with foul sputum, occasional fever, and weakness. His doctor suspected tuberculosis and treated him accordingly, without results. A year and a half later, he changed doctors and was x-rayed and a diagnosis of lung abscess was made. He began treatment with postural drainage and cod liver oil and began to show some improvement.

Past History: Measles, mumps, influenza, occasional attacks of tonsillitis and sore throats.

Weight: 130 pounds; Best weight 132 pounds.

Physical examination: Very foul breath; injected throat, hypertrophied and infected tonsils. Chest revealed rales, in both bases posterior, more marked on the right, with increased breath sounds and impaired resonance in the right base posterior. Fingers are clubbed. Nutrition is poor.

Laboratory: Urine essentially negative

Blood count - Reds 4,500,000; whites 8,400 to 9,400.

Polys 69%; Lymphos 28%; Monos 1%; Eosinos 2%.
X-ray: Chest - Lipiodol injection revealed saccular dilatations of the bronchi in both lower lobes, more marked in the left.

Sinuses: Bilateral maxillary sinusitis, with involvement of the right ethmoidal cells.

Treatment: Antra drained and tonsillectomy performed.

Discharged with slight improvement 3/10/31.

Case 2.

White, married woman, age 32, entered the hospital 4/10/31, complaining of:

1. Chronic productive cough.
2. Foul smelling sputum in the morning.
3. Chills and fever.
5. Pain in the left chest.

The onset was nine years ago, when she developed a lung abscess, following scarlet fever. This was drained. Six years ago, she had typhoid and scarlet fever again. Since this time, she has had pleuritic pains in the left chest, and a persistent cough, aggravated by frequent colds and attacks of "flu". She has had two hemorrhages, the first, eighteen months ago and the second, twelve months ago. In December 1930, she had a severe attack of influenza, with spasmodic coughing and marked debilitation. Another attack, one month before entrance, has confined her to bed since. For the past few years, she has raised large amounts of sputum, especially upon change of posture, and upon rising, in the morning. Occasional hemoptysis noted.

Past History: Measles, mumps, pertussis, scarlet fever, typhoid and
several attacks of otitis media. Asthma, since three years of age.


Weight: Best 135 pounds. Has lost eleven pounds in past year.

Family history not essential.

Physical Examination: Reveals well developed, fairly well nourished, white woman of about thirty-five years of age. There is diminished expansion of the left lower chest, associated with decreased breath sounds and impaired resonance. Moist rales heard over both bases, posteriorly chiefly over the left. Clubbing of fingers noted.

Laboratory: Urine - Albumen and red blood cells.

Blood count - R.B.C. 5,000,000; W.B.C. 12,000.

X-ray: Lipiodal reveals saccular dilatations of left lower bronchus in posterior third of lung. Only a moderate rise of the diaphragm, with but slight flattening of cavities.

Treatment: Phrenic nerve exersis done 4/17/31. Lipiodal injections every two weeks to three injections.

Dismissed improved.

Case 3.

White, married woman, age 37, entered the hospital 4/22/31, complaining of:

1. Mass in right breast of eleven years duration.
2. Cough - non-productive.
3. Weakness.
4. Anorexia.
5. Insomnia.

She has not been well since childhood, because of chronic cough and stomach disorder. The cough and weakness are aggravated in the winter time. The cough is non-productive and there is no history of hemoptysis.

Family History: Father - asthma.
Brother - chronic sinusitis.
History of cancer and diabetes in the family.

Physical Examination: Reveals poorly nourished and poorly developed woman with foul breath. Asthmatic type of chest, expansion equal; no percussion changes, rales, nor change in fremitus. No clubbing of the fingers.

Laboratory: R.B.C. 4,650,000; W.B.C. 10,000.
Polys 64%; Lymphos 36%.

X-ray: Lipiodal injection revealed bronchiectasis in the lower left lobe posterior of cylindrical type.

Treatment: Three lipiodal injections - dismissed improved 5/18/31. Returned for phrenic nerve exercise 9/10/31, and thirty cm. of the nerve was obtained.

Case 4.

A white, married housewife, age 67, entered the hospital 8/18/31, complaining of:

1. Cough, worse in the morning.
2. Foul sputum.
3. Anorexia.
4. Pain over left chest, on coughing.
5. Smothering sensation on slight exertion.
Onset three years ago, following an attack of influenza, which
confined her to bed for three weeks. Has had cough ever since, was
greatly weakened for three months. About two years ago, she began
to spit up a thick yellow, foul smelling material, chiefly in the
morning.

**Past History:** Measles, scarlet fever, chicken pox, influenza three
years ago.

**Weight:** Best 145 pounds, twenty years ago.

Present 95 pounds.

**Family History:** Father died of cancer of the rectum.

Mother died at age of forty five with bronchitis.

**Physical Examination:** Reveals an emaciated old woman of about seventy
years of age, propped up in bed. Shows an upper dorsal
kyphosis. Rales are heard, generally, over the chest on deep inspiration and after coughing. There is no impairment of resonance. Breath sounds are transmitted through the bases. B.P. 114/74; Heart sounds - first sound roughened, pulmonary second accentuated.

**Laboratory:** Urine - negative.

Blood count: R.B.C. 4,120,000 to 4,930,000.

W.B.C. 12,200 to 15,800.

Sputum - Negative for tubercle bacilli and spirochaetes.

**X-ray:**

Suggests bronchiectasis of right, lower lobe and fungus infection in left, lower lobe. Lipiodal injection was not made.

**Treatment:** Ultra violet light, postural drainage, and creosotal for one week.

Dismissed, improved with five pounds gain in weight 9/30/31.
Case 5.

White, boy, age 19, entered the hospital 11/7/31, with the chief complaint, that of several successive attacks of appendicitis.

Past History: Reveals attack of pneumonia at one year of age and another attack at eleven years of age, followed by chronic cough, without hemoptysis.

Chest findings were negative and an appendectomy was done 11/17/31. Following the operation, he shot a fever and raised thick purulent sputum, associated with increased breath sounds, decreased fremitus, and dullness over the base of the right lung posteriorly.

Diagnosis of bronchiectasis not confirmed by x-ray.

Dismissed 11/24/31, cough subsiding.
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