

University of Nebraska Medical Center DigitalCommons@UNMC

MD Theses

Special Collections

1931

Bronchiectasis

William J. Hervert University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search PubMed for current research.

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Part of the Medical Education Commons

Recommended Citation

Hervert, William J., "Bronchiectasis" (1931). *MD Theses*. 157. https://digitalcommons.unmc.edu/mdtheses/157

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

THESIS: BRONCHIECTASIS

Ъу

William J. Hervert

BRONCHIECTASIS

Definition

The term bronchiectasis comes from the Greek and means bronchial dilatation. It was first described as a clinical entity by Laennec in 1819. Pathologically, it is characterized by bronchial dilatation, and clinically, by a chronic cough and purulent sputum. Bronchial dilatations may be congenital or ac-The congenital type gives rise to characterquired. istic symptoms only following a persistent infection. The acquired type has a varied pathological anatomy, pathogenesis, and etiology. The condition is essentially one of bronchial disease in which bronchial dilatation is only one of the pathological changes. Incidence

Bronchiectasis is now known to be far more prevalent than it was formerly thought to be. Some state that it ranks next to pulmonary tuberculosis in incidence. Statistics show that it appears in from 2-4% of the General Hospital necropsies, and these reports are quite generally alike.¹ All these statistics have been compiled since the event of pulmonary radiography and particularly since the latter has become better understood. Heretofore, this disease was usually unrecognized and treated as pulmonary tuberculosis, chro1

nic bronchitis, asthma or lung abscess. The incidence of the disease, then, rose to a higher level as a consequence of closer study and a clearer knowledge of the disease, and better means of studying the contents of the thorax with the x-rays. The use of contrast media bronchography will no doubt increase the incidence of the disease still more.

Etiology

A. Predisposing Causes

1. <u>Climate</u>--Sudden fluctuations in temperature and humidity are known to have a definite influence upon the respiratory tract and sinus infection, which in turn tend to excite bronchiectasis. Experiments have proven that sudden cooling of the body surface is particularly provocative of this condition, but only in the presence of infection especially when different grades of virulent bacteria are present.

Inhalations of dust and other particles are known to spread tubercle bacilli, and so inhalations of pyogenic bacteria could likewise produce lung infections similarly. This has also been demonstrated experimentally. Lowered resistance and virulent organisms are very important in determining the onset of this disease.

2.<u>Age</u>--Of 512 patients observed at the Mayo Clinics, Wisconsin General Hospital, the Research and Educational Hospital of the University of Illinois, and

other Chicago Hospitals, during the last 10 years, the ages of the patients at the time they were first observed read as follows:

TABLE 1

AGES								CASES			
Under 5	yrs	. –	-						8		
6-10	11	-						-	44		
11-15	11			-		-			37		
16-20	11	-	-	-					69		
21-30	H			40100	-	-		-1	.27		
31-40	11	-	-				-		83		
41-50	11			-	-	-	-	-	65		
51-60	11	-	-	-	-	-		-	46		
61-70	11	-	-	-		-	-		28		
71-80	11	-		_	-	-	-	-	5		
					Total			512			

This table shows that only 17.4% were under 16 yrs., and 15.4% had passed the 5th decade. Clinical statistics show its presence commonly observed in middle life. This doesn't, however, help to determine its time of onset, since the latter is so prolonged and insiduous. The next table shows the ages at first observation, and the onset of symptoms as derived from the clinical histories of a series of 134 cases. They have been proven by contrast media bronchography. TABLE 111 (Continued)

 DURATION
 CASES

 21-30 yrs.
 ----5

 31-40 ----6

 41-50 ----3

 51-60 ----0

 (?)
 ----7

 134

It has been reported to be present in very early childhood, usually following some acute process such as, measles, whooping cough, grippe, broncho-pneumonis or chronic bronchitis. Often the etiology is unknown. The earliest symptoms are not infrequently mistaken for bronchitis and treated as such. The pediatricians at present are devoting their time to diagnosing this disease in its early beginning.^{4,23}.

3. <u>Sex</u>--In the adult, the disease is most prevalent in the male, while in children, the reverse of this is true⁵. The reason for this is not satisfactorily worked out. It is probable that the adult male comes into more contact with exposure, changes of temperature, infection and occupational bronchial tract irritation than the female or the child.

4. <u>Race</u>--This condition appears to be more common in negroes; however, it is not so limited. 5. <u>Heredity</u>--Heredity is used by some workers as an etiological factor of much gravity since it was seen to occur in several members of the same family, and in association with definite congenital defects. Arnheim reports one case associated with a hemilateral hypertrophy of the whole body. It is seen associated with hare-lip, web fingers and mental anomalies of different kinds.

6. <u>Occupation</u>--Occupational inhalations of organic dust from tobacco, textiles, cereals and of inorganic dust steel, stone, coal, etc., cause irritation of the bronchial mucosa leading often to a bronchitis followed by a connective tissue infiltration extending into the interlobar septa. Distal to the connective tissue contractions or cicatrices, is the most common site for bronchiectasis to develop. Bronchiectasis also probably develops secondarily to an extending infectious bronchitis. Some cases are reported to have followed pneumoconiosis. Mustard gas and other chemical irritations produce a severe bronchitis and may eventually terminate in bronchiectasis.

B. <u>Exciting Causes</u>--The essential cause of congenital bronchiectasis is infection of bronchial dilatations at birth. The respiratory tract diseases which bring about such dilatations are probably among those

which bring about bronchial damage in the acquired type.

The causes of bronchiectasis are inflammatory changes in the bronchial walls and mechanical stress incident to inhalation or cicatrical contraction. The bronchial lesion is the fundamental cause, but massive atelectosis or chronic fibrosis may play a primary role.

It is fairly well established that bronchitis is the essential cause of the changes in the bronchial wall incident to their dilatation. Brauer classifies these changes into three stages; first, a superficial catarrhal bronchitis, which may heal without scarring; second, an infiltrative bronchitis and peribronchitis, the first stage of which is an intra-mural process, the second a peri-bronchitis; and third an ulcerative bronchitis. A large part of the cases of bronchiectasis may be attributable to a recurrent "flu" and many of indeterminate origin, are attributable to such a primary bronchitis. Injury to the bronchial mucosa chemically, and by inhalation of organic and inorganic dust also leads to infection and finally bronchiectasis. At other times it follows such acute infectious diseases as measles, pertussis, whooping cough, etc., with an associated acute bronchitis.

1. <u>Bronchial</u> <u>Obstruction</u>--That foreign bodies which obstruct the lumen of bronchi act as etiological

factors in the formation of bronchiectasis is well established. Any foreign body can cause this as long as the patient isn't able to cough it out. The dilatations of the bronchi always occur distal to the foreign body. The secretions remain within the lumen of the bronchus since they are unable to pass out. Infection sets in subsequently and the bronchial wall suffers permanent damage with resultant dilatation. Other factors within this category are, pneumonia, broncho-pneumonia, infectious disease complicated by pneumonia, chronic pulmonary abscess, fibroid phthisis, fibrosis from syphilis and post operative infections.

C. <u>Indeterminate</u>--This includes cases where no infectious diseases are reported, where an unreliable history has been given, or where there are congenital malformations. Sauerbruch¹ believes that most of these are congenital in origin.

Pathology⁶

The location and extent of bronchial dilation are dependent upon the etiology and duration. In the acquired type the etiology determines the original site of the lesion. In case of tuberculosis bronchiectasis, the lesions usually involve the upper part of the lung. When due to a foreign body or a stenosis the dilation is at first circumscribed to the involved bronchi, but,

if secondary, to a diffuse fibrosis or extension atelectosis, it is diffuse from the onset. Secondary infection of adjacent bronchi and of those in the opposite lung from the purulent secretion and infectious processes in the parenchyma determines the spread of the original focus. Duration is an important factor in such extension.

Clinical studies as to site and extent of involvement, based on physical and ordinary roentgenographic studies, have been at best only approximately correct. Lately with the introduction of contrast media roentgenography we have a fairly accurate means of determining the localization of bronchiectasis.⁵ The methods of using contrast media, of course, also have their limitations.

From a series of 113 cases studied by contrast media bronchiography the localizations discovered are as follows:

SITE			CASES							PERCENT			
Left	Lower	Lung -	-	-		46				-			40.1
Right	Lower	Lung -		-		31			-	****	-		27.1
Both	Lower	Lungs-		-		32	-	-	-	-		-1929	28.0
Whole	Left	Lung -		_	-	4		-		-		-	3.5
Whole	Right	Lung -	***		-	0	_	-	-			-	0
Total 113													

From all the studies so far, it becomes clear that

there is a left sided preponderance of bronchiectasis. There is, as yet, no convincing explanation for this. It isn't to be explained on the basis of obsturuction by foreign bodies since they usually lodge on the right Lober pneumonia likewise is usually found to be side. right sided. Duken explains the left sided preponderance of bronchiectasis as due to a mechanical predisposition to dilatation on account of the greater deviation of the left bronchus from the axis of the trachea, and also on account of the narrowing of the left main bronchus just proximal to the origin of the upper lobe subdivi-The further support of such a view is the clinsion. ical observation that primary tuberculosis in the right upper lobe spreads first to the left upper lobe, while a tuberculosis that is primary in the left upper lobe as rule involves the left lower lobe before involving the opposite lung. That is why a generalized tuberculosis on the left side may frequently be seen and a similar situation on the right side is a rarity.

Sauerbach believes that the left sided preponderance is due to a mechanical interference to the development of the bronchi on that side by the heart and Cuvier's duct.

A. Types of Bronchiectasis

1. <u>Congenital</u>--Congenital bronchiectasis may

be due to a congenital malformation or to foetal disease. These anomalies are described under such terms as cryptic malformation, foetal bronchiectasis, polycystic lung, and papillary bronchial adenoma. These anomalies may involve any part, or all of one lobe of the lung. It may be found in the lungs of premature babies to such an extent that respiration is so inadequate that life cannot be amintained.

A variety of anomalies has been described. One of these is described as "Universal" bronchiectasis dilatation of the bronchi to the involved lobe communicating with the multilocular cystic dilatations lined with cuboidal cystic dilatations of the bronchi of the third and fourth order, some ending blindly and some communicating with cysts. Pappenheemeir describes a case involving the lower part of the upper lobe. The cysts in this case were as large as one cm. in diameter, lined by epithelium (or columnar), mostly of the ciliated variety. The cyst wall consisted of this epithelium, in an elastic tissue stroma with smooth muscle and cartilage found here and there. Serial sections showed these capillary spaces in direct communication with the alveoli and larger bronchi.

These have been variously explained as due to simple arrested development, agenecis of the alveoli, collection of fluid in foetal bronchioles or as being essentially

cystic foetal bronchial adenoma. Many think that syphilis plays a big part, since the spirochaeta jallida have been demonstrated in some cases.

2. <u>Acquired</u>--Studies in clinics and pathology show that the majority of bronchial dilatations occurs in lungs which were normal at birth, the bronchial dilatations resulting from a combination of mechanical and infectious factors. Some of the mechanical factors are foetal atelectasis and atelectasis acquired later in life. In a majority of cases, infection plays an important role. The foetal atelectatic bronchiectasis results from a failure of the alveoli of the lungs to open at birth or their collapse thereafter.

Acquired bronchiectasis in contradistinction to that due to the congenital failure of development of the alveoli was first described in 1885. The characteristics of atelectatic bronchiectasis acquired at birth or in early life are the absence of pigment in the affected lobe, the absence of inflammatory signs, and the absence of alveoli in the parenchymal portion of the lung. There are also characteristic changes in the walls of the bronchi. This condition is usually seen in chronic form.

3. <u>Acute</u>--This type of bronchiectasis is usually spoken of as following some acute infections disease such as whooping cough or measles. The dilatations

end in cul-de-sacs of the diameter of a little finger. Reports have been made of this form of bronchiectasis following acute capillary bronchitis which only lasted about a week or ten days. Whooping cough which is severe and rapidly progressive is responsible for many early cases of bronchiectasis; the symptoms which are characteristic follow much later in the disease.

4. <u>Chronic</u>-Early and uncomplicated bronchiectasis may not be recognized from a normal lung by inspection and palpation. When emphysema is present, the lung may be enlarged and feathery to palpation. In the atelectatic lung the lobe seems smaller than the normal size. When the atelectasis has been present from early childhood, the lung tissue will have the characteristics mentioned above; namely, absence of pigmentation, absence of alveoli, no evidence of active inflammation and characteristic atrophic conditions of the bronchial and alveolar walls. A lung of this type will not crepitate on palpation, and considerable enduration will be in evidence.

The shape, size, and distribution of the bronchial dilatations will vary in different individuals and even in different lobes in the same person. The classical morphological description based on necropsy studies includes the cylindrical, fusiform, saccular, and monili-

form types. Since contrast media bronchography has been introduced, studies show that a combination of the cylindrical and sacculated varieties is the most common. Pure saccular or moniliform types are relatively uncommon. The fusiform type is spoken of as a grade of sacculation of the cylindrical type.

Cylindrical dilatation may involve the larger, more proximal bronchioles only, or chiefly the medium-sized and terminal bronchi. Some writers state that the larger bronchi are involved in primary bronchitis, and in bronchitis due to foreign body or stenosis from other causes, but that bronchioles and smaller bronchi are primarily involved in cases due to measles, whooping cough, and influenza. The degree of dilatation varies from that which is barely perceptible to that of a diameter two to three times the normal. The shape of the enlargement also varies; in some the sacculation is uniform and in others it tapers distally.

The combined saccular and cylindrical variety of ampullor dilatations consist of a typically cylindrical proximal and ampullor distal extremity, usually situated at the basal medial portion of the lung. The ampullae may be regular or asymmetrical. The degree of dilatation varies from a slight clubbing to an ampullar enlargement many times the diameter of the widest portion

of the cylindrical enlargement.

The alveoli between the dilated bronchi are collapsed and may have undergone cicatrical atrophy or obliteration, so that the walls of adjacent ampullae may lie in direct contact. Sometimes the walls of the saccules fuse, later rupture and form a large cavity. When the proximal portion of the bronchiole is closed up, such cavities may be converted into cysts. When bronchiectasis is associated with fibroid phthisis, one almost always finds cylindrical bronchial dilatations, often cone shaped, with lateral and occasionally large vestibular cavities, or fusiform inlargements where the bronchus passes through a cavity. In these cases, accumulations of mucous and muco-purulent secretions are seen behind scar tissue or stenosed parts.

The chronic inflammatory changes involve the whole thickness of the bronchial wall and may extend to a greater or lesser degree to the surrounding penbronchial tissue and parenchyma. Some portions of the same dilated bronchus may be hypertrophic, and in others atrophic. In the hypertrophic portions, the mucus membrane is reddened, swollen, velvety, villous or polypoid and more or less thrown into folds. Ulcerated areas may be found where the mucosa has sloughed away. In the atrophic portions, on the other hand, it is thin,

smooth or trabeculated from the projection of interlacing transverse longitudinal ridges.

On microscopic examination of the hypertrophic portion, the mucosa is found to be thickened, uneven, and thrown into folds and papillae. The blood supply is increased, and this especially in the papillary outgrowths. The epithelium is often irregular, showing various grades of transformation from the columnar to the cuboidal of flattened cells. In some cases the epithelial metaplasia occurs--the cylindrical epithelium is replaced by cuboidal, pavement of cornified epithelium. One may find adjacent areas of ulcerative loss of epithelium, areas of hypertrophy and islands of normal ciliated epithelium. Some of these islands may even show neoplastic changes. There is a marked round cell infiltration about the glands, muscle bundles, elastic fibers and cartilage. Calcification occurs in the tissues as a later stage of the inflammatory process. This is seen mostly in the degenerated areas especially in the cartilage. Sometimes it occurs even in the mucosa and fibrous portions. In later stages the elastic fibers and muscle fibers become dissociated and replaced by fibrous tissue.

The walls of the saccular or ampullar enlargements consist of a thin layer of scar tissue lined by

epithelium. There may remain no vestige of the normal bronchial structures.

In the presence of bronchial cicatrical tissue, with the formation of obstruction, the retained secretions may ulcerate their way through the bronchial wall, producing extrabronchial gangrene or empyema.

Performnchial infiltration and thickening occur in varying degree and extent. The amount of lung involvements varies greatly in greatly in different types of bronchiectasis. In the early cylindrical type there may be none. Bronchiectasis may exist in a healthy lung. In the atelectatic type of long standing, there is often complete fibrosis or "Cornification" of the involved lung parenchyma. In cases of bronchiectasis in which fibrosis is one of the pre-existing and etiological factors the changes in the lung parenchyma are characteristic of the pre-existing disease condition.

Pleural adhesions develop at different stages in the progress of the disease. Early cases show no evidence of adhesions or thickening as shown by x-ray or physical findings; this is also borne out by the fact that artificial pneumothorax can be produced. In the terminal stages of the disease, as shown by necropsies, adhesions and pleural thickenings occur in great abundance.

Symptomatology

The primary and usually the most outstanding symptom of bronchiectasis is a chronic, recurrent productive cough. In most instances this cough can be traced back to an attack of La grippe or bronchopneumonia or in children it is a residual of whooping cough, measles, bronchopneumonia or influenza.

In typical bronchiectasis, the cough is typically paroxysmal in character or may be induced voluntarily such as upon bending over. This spasm of coughing is accompanied by the production of varying amounts of purulent sputum. The most severe attack of coughing in most instances takes place when the patient arises in the morning. The cough does not disturb the sleep, but the patient usually lies on the affected side to immobilize it and prevents coughing.

The explanation of the periodicity of the cough and evacuation of sputum is a gradually acquired lowering of the irritability of the dilated bronchi to the pressure of the purulent secretion. Only when it overflows into the more sensitive portions of the bronchial tree or other bronchi does it bring on the cough reflex. The overflow of the lipiodol from the dilated bronchi into other portions of the bronchial tree has been observed by fluoroscopic methods.

One of the very striding ovservations on bronchiectatic patients is the amount of sputum they produce. The amount of it varies greatly with different cases. Many cases proven to be bronchiectasis by contrast media bronchography will raise only 30-60cc. of sputum in 24 hours; others with apparently the same grade of bronchiectasis will raise 200-500cc., and in some cases as high as 1000cc. has been reported, or even more in 24 There is considerable variation in the amount of hours. sputum from day to day without any explainable cause for it. Some causes will increase the amount such as, for example, the common colds. The consistancy of the sputum varies with the amount of mucus in it. The character is that of frank pus of a liquid consistency especially when large in amount or when the larger bronchi are extensively involved, but it may be almost pure pus when small in amount. The color of it is characteristically of a grayish green, unless mixed with changed blood, when it is brown. The odor is usually very musty, and may be putrid and very offensive. The breath may have that same odor. An unbearable stench characteristic of gangrene may occur without other evidence of gangrene.

Hemoptysis is another common symptom of bronchiectasis. Reports have been made that it is more frequent in this disease than in tuberculosis. The in-

tensity of the hemorrhage varies with the individual. It may merely stain the sputum slightly or prove fatal. The time of it also varies greatly for it may appear as one of the primary things or appear late when the disease is greatly advanced. The bleeding is thought to arise from the dilated capillaries within the raw ulcerated mucosa of the bronchi. The profuse hemorrhage comes from the branches of the pulmonary artery.

In the earliest stages of the disease, fever in adults is rather the exception than the rule, but is found to be quite common in children. The fever may linger quite constantly or be of an intermittant or remittant character. Later on when the pathology extends farther into the parenchyma, a mild grade of fever is usually observed. Sometimes there are acute exacerbations of chills and fever in the more early stages of the process.

Pleuritic pains are also present in some cases. This may be due to an intercostal toxic neuritis. Dyspncea and cyanosis are usually not common except on exertion and in the later stages of the disease. Vomiting and diarrhea also occur probably from toxicity.

1. <u>Physical Findings</u>--Patients suffering with bronchiectasis often show no evidence of it by their personal appearance. It would seem, however, that

greater evidences of it would be presented since there is so much opportunity for the system to absorb toxic material from the bronchi. Those that present physical evidences of the disease manifest it from a state of undernourishment, anaemia to semi-invalidism.

The physical findings in most instances are very nearly like the normal. There may be a few creaky rales heard over the involved areas upon deep inspiration with coughing and that's all. But when these creaky rales are persistently heard over the same areas, it should arouse suspicion that bronchiectasis is present. Where the involvement becomes more advanced, the rales become of the sonorous, or loud bubbling type. The area is then increased also. The percussion note may be intirely normal. When the percussion note is normal and the rales are loud, it is suggestive of bronchiectasis. When the rales are bilateral, the condition is usually one of bronchiectasis on the left side and bronchitis of a chronic type on the right side. Loud cavernous breathing is heard when there are large saccular dilatations, and especially so when these are near the chest wall. Consolidations within the lung parenchyma would tend to intensify these still more. The percussion note, as stated, may be quite normal; but there may also be a flatness on percussion when the dilated bronchi are full of the liquid purulent material.

If the patient should get a paroxysm of coughing, and raise the sputum, then the quality of the percussion note will be changed.

Later in the course of the disease, other changes seem to occur which change the picture entirely. When the pleur becomes thickened and the parenchyma of the lung becomes more involved, more bronchial breathing is heard. Associated with that, there is a greater transmission of whispered and spoken voice and an increase in tactile and vocal fremitus. There is usually a dullness on percussion, which varies with the quantity of secretion. Fibrosis of the lung and pleura causes a narrowing of the intercostal spaces, retraction of the chest wall, lagging motion on inspiration, and deviation of the trachea.

Characteristic changes take place in the extremities. The hands become cold and clammy and are cyanosed. Similar changes are seen about the ears and nose. Clubbing of the fingers and toes, which occur in disturbances in circulation are also manifested in this disease. In many instances it becomes very apparent. The terminal phalanges are enlarged in both diameters, and the area over the matrix of the nail is elevated, especially at the proximal border, giving the nail an increased convexity and an inward tilt to the tip like a parrot's

beak.

There are several thories advanced to account for the clubbing of the fingers and toes in bronchiectasis. Bamberger, who first spoke of it, attempted to explain it on a basis of toxic absorptions, and Loeschke to an angiospasm, which would also explain the cyanosis of the ears and nose. The tissues of different fingers, with different degrees of clubbing have been examined histologically and no changes could be seen in the capillaries. Plesch explains this condition on the basis of increased resistance to blood flow within the pulmonary circulation in which condition the extremities tend to suffer the most. The time of onset of clubbing also varies. In some cases the symptoms of bronchiectasis and clubbing appear at the same time. In other instances, the clubbing may follow the original symptoms by one or ten years. When clubbing and bronchiectatic symptoms are seen at the same time or nearly so, it is usually seen to follow a severe attack of whooping cough.

In some instances, particularly in bronchiectasis which follows bronchial obstruction, the changes in the peripheral parts may be so great as to make the patient appear as though he were suffering from acromegaly rather than from bronchiectasis.

Autopsies usually reveal a distinct periosted1 thickening with new bone formation. In some instances, the synovial membranes are thickened and cartilages eroded.

The sputum, as alluded to above, is very characteristic. If allowed to stand it usually separates into three layers, a frothy mucus on top, pus which contains the Dietrich corpuscles at the bottom, and saliva remains in the middle layer. It has no diagnostic significance since it also occurs in tuberculosis, pulmonary abscess, and empyema with bronchial fistula.

Elastic tissues may or may not be found. Microscopic examination shows leukocytes in varying degrees of disintegration, mucous plugs, fat goblets, fatty acid crystals, and bacteria.

It is evident that infection is the most important causative factor in bronchial dilatation, and also in the manifestation of the disease; so it is interesting to find out what particular organism it is that exerts this profound influence. Thus far, bacteriological examinations show that it can't be ascribed to any particular organism. A mixed infection usually is the case. The study of the organisms can be carried out in any one of several different ways: (1) By the

23

an

study of the sputum, (2) By obtaining culture specimens from the bronchi by bronchoscopy, (3) By the study of necropsies, and (4) By the study of pathological specimens. By all these methods a mixed infection has been found to be the rule and some of the organisms seen were the influenza bacillus, pneumococcus, streptococci, staphylococci, micrococcus catarrhalis, and the diphtheroids. Others have made reports of the Friedlander's bacillus, pneumonia bacillus, colon bacillus, pseudotuberculosis bacillus, tetrogens and anoerobes. Still others have given reports of bacillus fluorescens liquifaciens and nonliquifaciens, and also leptrothrix.

In purulent bronchitis, the spirochaete of Vincent has been found to be the causative organism. This disorder draws a close analogy to the purulent form of bronchiectasis.

In some of the latest works, the influenza bacilli were found practically in pure culture in the sputum over a long period of observations. This includes both the sputum, as well(as, the specimens of the teased bronchial wall.

2. <u>The Blood</u>-Leucocytosis is usually the rule in bronchiectatic conditions; if not continuously, at least at intervals. The count may be anywhere from

12,000 to 25 or 30,000. In children, a leucocyte count of 12,000 along with the other symptoms, is usually strongly indicative of bronchiectasis. Secondary anaemia is usually seen also.

3. <u>X-ray Findings</u>--The roentgenograms in a well advanced case of bronchiectasis are very characteristic. In the more early cases, the pathology is quite obscure although some haziness will usually be shown. This is because the mucus and purulent material which has accumulated within the dilated bronchi has very nearly the same density as body fluids and normal tissue. Unless there is some calcification present or solid foreign bodies introduced, they will not show up in the picture.

A typical picture will show an extensive thickening of the lung markings along the course of the larger bronchi. This thickening usually begins some distance above the hilus and continues from there downwards to the base of the lung, and in many instances also some distance lateralward. This will not appear definitely as dilatated tubes, but rather as a haziness of an indefinite course or outline. In case of diseases of long standing and chronicity there is usually an enlargement of the parabronchial lymph glands and this is very true of bronchiectasis. The hilar glands also show up

very plainly in the picture. In the more advanced cases when a great deal of fibrosis and infiltration has taken place areas will appear, single or multiple, of increased density in the lung fields near the bronchi, which may show considerable change in films obtained before and after evacuation. Cavities can be demonstrated frequently.

In the early stages the picture is much less characteristic, and depends upon the demonstration of small ringlike shadows of dilated bronchi, which, however, are generally obscured by the infiltrated lung surrounding them. There is an associated emphysema in the longstanding cases.

The introduction of an opaque substance such as lipiodol in cases of bronchiectasis permits of a complete study of the size, shape, and distribution of the damaged bronchi. Lipiodol is 10% iodized poppy seed oil, or contains about 9.8 to 11.2 percent of iodine (0.11gm of iodine per cc.) in organic combination.

Ochener has made a wider use of lipiodol injections perhaps than any other man in this country, and he uses the "passive method" of its introduction. Its introduction into radiographic work is also very interesting. The idea was suggested by an accident which occurred while he was serving as an exchange assistant in the

Zurich Surgical Clinic under Professor Clairmont. Following an esophagoscopy for carcinoma of the esophagus, preceding which the pharynx had been completely anaesthetized, the patient was given a barium meal in order to visualize the tumor radiographically. Much to the surprise of the observers, the barium meal was seen to pass into the trachea instead of the esophagus and caused a paroxysm of coughing. By following up the ideas suggested by this accident, the technique for the insufflation of lipiodol has been developed. Briefly stated, the procedure is as follows: Following the use of an antiseptic mouth wash the anterior surface of the anterior tonsillar pillars is painted with a 10 per cent cocaine solution. The anesthesia is continued until the swallowing reflex is abolished, as evidenced by immobility of the larynx on attempted deglutition. As soon as the anesthesia is complete, the patient is instructed to take about 3 cubic centimeters of a 3 per cent procaine hydrochloride solution into his mouth. He then tips his head backward, protrudes his tongue so that the procaine solution may enter the pharynx, leans slightly to the side which one desires to fill, and breathes. The procaine solution is used as an anesthetic to the tracheobronchial mucosa. As the anesthesia of the anterior pillar is only temporary, it is essential again

to paint the anterior pillars with the 10 per cent cocaine solution. The patient is then placed behind the fluoroscopic screen and given 10 cc. of iodized oil and is instructed to assume the same position that he assumed while inspiring the procaine solution. The entrance of the oil into the trachea and bronchi is observed fluoroscopically. The patient expectorates the saliva that has collected in the mouth, following which he aspirates another 10 cc. of iodized oil.

In some cases it may be necessary to swab the anterior pillars a number of times in order to abolish the reflex. It is important not to allow more than about thirty seconds to elapse between applications of the cocaine and also to be sure that the swab is pressed. well into the angle at the junction of the anterior pillars and the tongue. The saliva which accumulates should be expectorated each time before pillars are swabbed to svoid diluting the anesthetic. If the chin is elevated and the head held well back it is not always necessary to anesthetize completely the pillars as swallowing in this position is impossible even with only a partial anesthesia. It frequently aids to have the patient pull his tongue out and hold it, especially when the oil is being given therapeutically and no fluoroscopic observation is being made. We have used

Ker

a locc. syringe to introduce the iodized oil over the posterior part of the tongue. This will avoid any unnecessary waste of oil.

Following a diagnostic insufflation a radiogram should be made to confirm the flurorscopic findings and also as a permanent record. This should be done immediately since frequently the lapse of four or five minutes is enough to cause the radiogram to be worthless, especially should the patient cough.

The advantages of the above described technic are several. The filling of the bronchi can easily be observed fluoroscopically and in this manner dilatation will be observed which cannot be shown in the radiogram. Because of its simplicity the method can be used in out patient or office practice, thus lessening the expense and inconvenience for the patient. It can be carried out by the internist or roentgenologist, the skill of the bronchoscopist being unnecessary. Because of the fact that there is no discomfort associated with the procedure, patients readily submit to repeated insufflations when necessary for therapeutic purposes.

Diagnosis

There is, perhaps, no other chest condition which has the marked characteristics so often encountered in this disease in which the diagnosis is so commonly overlooked. The importance of keeping in mind the significance of marked clubbing of the fingers in association with lesions at the base of the lung, especially those associated with signs of a cavity in the area about the angle of the scapula, cannot be overemphasized.

Inspite of the classical symptoms presented by this disease, there are several other conditions which occur in the lungs, that must be kept in mind to arrive at a correct diagnosis. Some of the most outstanding, and likewise most confusing conditions will be mentioned.

1. <u>Tuberculosis</u>--There are many more cases of bronchiectasis mistaken for tuberculosis than for any other condition. The percentage of cases runs quite high. It is stated that at the White Haven Sanitorium during one year there were no less than 6 cases typical of the condition sent into the institution under the belief that they were suffering from advanced pulmonary tuberculosis. Records from other hospitals show that this percentage is representative and, in many instances, is even exceeded. This mistake is inexcuseable when the dilatated bronchi are situated in the lower lobes. It is true that there is coughing, large amounts of blood streaked sputum and hemoptypes, there should really be no difficulty if one is ever mindful of the fact that tuberculosis rarely occurs in the lower lobes of the lungs. The sputum should be examined very frequently and the condition not pronounced as case of tuberculosis until the acid fast bacilli are found.

Bronchiectasis is often found to be associated with streptothrix organisms, which are almost as acid fast as the tubercle bacilli and hence, the mistake in diagnosis is made on that grounds. The streptothrix threads are broken up into small pieces and when stained with Gabbetts stain, appear just like tubercle organisms. A condition of this nature came under observation at the Phillips Institute with extensive involvement of one entire lung was believed to be tuberculous in nature from the positive sputum report for the presence of tubercle bacilli. Subsequently, a sputum was was cultured with the hopes that a vaccine could be obtained to give the patient some relief. After all this had been done, it was found that the sputum contained streptothrix organisms which were acid fast. No tubercle bacilli were found.

The dilatations of the bronchi do not always occur in the lower lobes of the lungs. If the dilatations

occur within, or near the apices it is practically impossible to differentiate this from pulmonary tuberculosis. These cases are found mostly where the person in question had been exposed to inorganic dust with great frequency. As the physical signs over the remainder of the lungs are indicative of very extensive pathological changes, and as tuberculosis is frequently found to be superimposed on a pneumoconiosis, it is very difficult, often, to determine whether pneumoconiosis alone is present or in company with tuberculosis. X-ray examinations of the lung pneumoconiosis will not show tubercle bacilli.

X-rays are very beneficial in diagnosis of bronchiectasis. Out of every 25 cases usually about 19 are made as a positive diagnosis and the remainder are either called possibilities or missed entirely. The cylindrical and sacculated varieties are most readily recognized. The third type, infiltrative, is characterized by a more or less stringy increase in density along the course of the bronchi, usually in the lower lobes, and radiating from the hilus to the periphery. This may be mistaken for a mere thickening along the bronchial wall.

2. <u>Abscess of the lung</u>--Occasionally a rapid and pronounced clubbing of the fingers occurs in cases of

pulmonary abscess. Although there may be reasons for some confusion in diagnosis, there should be no prolonged debate over the condition as the characteristic feature of an abscess is the sudden expectoration of a large quantity of muco-purulent material. This expectorate has a sweltish rather than a fetid odor so characteristic of bronchiectasis. The duration of an abscess is not as long as that of bronchiectasis and the condition which brings it about is almost always so acute illness. Small multiple abscesses are more difficult to diagnose than a single, large abscess. The x-rays in this case are of very much assistance.

3. Loculated Empyema--This condition draws a close analogy to a pulmonary abscess except that it is extrapulmonary. Loculated empyema may erode the periphery of the lung and gain entrance into the bronchi after which large quantities of purulent material are expelled after coughing. This condition is of short duration and follows acute illnesses.

4. <u>Gangrene of the Lung</u>-This condition is likewise very much on the same order as empyema and lung abscess. There isn't much chronicity to it, and although the sputum is very offensive, it shouldn't be confused with bronchiectasis as the latter is very chronic. Gangrene of the lung is thought by many to

be the terminal event of bronchiectasis.

Prognosis

A moderate degree of bronchiectasis may occur as a sequel to whooping cough, measles, influenza or irritant gases. The large portion of these cases probably clear up without any serious residuals except a persistent cough and lingering bronchitis. This usually occurs during the winter months. If the dilatations occur following acute infectious diseases such as whooping cough, permanent conditions usually result. In most of the cases the disease runs a very chronic course. Bronchiectasis is not incompatible with life necessarily for people are known to have lived with the condition for forty years after its onset. A true recovery rarely occurs in the chronic deep seated cases. Death in indiviauals occurs from complications of bronchiectasis and some of these are brain abscess, gangrene of the lung, bronchopneumonia, and hemmohrage. Abscess of the brain is probably the most frequent cause of death. In some cases the health gradually fails, and death results from amyloid changes in the liver and kidneys, or cardiac failure.

Complications

There are many complications which can result from bronchiectasis, and it is readily conceivable when we

consider the large surface, within the lungs, from which the remainder of the body may draw the infection. It is likewise surprising to note that in a large percent of the cases the patients show no ill effects whatever, and there are many cases that do not meet with serious complications. The most interesting of the complications of bronchiectasis is intracranial abscess, fifteen instances of which occurred in 108 fatal cases of the disease. The abscesses were cerebral in nine instances, cerebellar in three, both in two, and in one case meningitis and ependymitis were present, but no intracranial abscess was located. The abscess was single in nine cases, multiple in six, and in one instance from twenty to thirty abscesses were present throughout the brain. In addition two cases of secondary intracranial new growths 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, emphema, gangrene and abscess of the lung, and old or recent pulmonary tuberculosis.

The most frequent causes of death were: bronchopneumonia, 34 cases; exhaustion, 34 cases; exhaustion and asphixia, 8 cases; intracranial abscess, 15 cases.

A case of multiple abscesses secondary to bronchiectasis caused by the wedging of the lower lobe of the right lung into a pocket formed by a Ryphoscoliosis has been described. The causative agents isolated and cultivated from both the abscesses and the suppurating lung were B. fuseformis and anaerobic streptococci. The author, Saelhof, believes that the most probable route by which the infection traveled from the primary focus was the blood stream. There is no doubt that in any extension of infection from the bronchiectatic field to other parts of the body is chiefly by the blood stream or direct extension.

In one of Davies' cases the accessory nasal sinuses became infected, requiring operation for empyema of the antra of Heghmore. The fetid pus from the pulmonary cavities was often expelled in quantity and profected suddenly with much force into the mouth and nose, and infection of the accessory sinuses arose in this way. In a second case cited, the complications which chiefly deserve notice, were chronic glossitis, and a persistent severe albuminuria from chronic nephritis, both of which were probably of toxic origin from the

chronic infection.

One of Bray's patients presented evidences of a localized pulmonary lesion at the base of the left lung for a period of at least seven years. This was attended by a productive cough and mucopurulent sputum, partly residual and negative for tubercle ba-The woman's features were somewhat cyanosed cilli. and her fingers clubbed. These clinical manifestations suggested some condition other than tuberculosis, probably bronchiectasis. This suspicion was heightened by absence of any striking change in the clinical manifestations of the diease during this period, by the negative radiographic examinations, and finally by the contrast in the clinical picture, physical and radiographic findings when the condition became complicated by phthisis florida.

From all the complications presented, both the common and the unusual, it is clearly seen that they play the primary role as far as the death of the patient is concerned. There are many instances reported where patients suffering from bronchiectasis only, have withstood its effects over a period of many years often presenting no outward manifestations of the disease. They were, however, ousted from society and even from their homes because of the fetid odor

assiciated with this disease.

Treatment "

1. Medical--Lipiodal Methods.

The treatment of bronchiectasis in the past has been very unsatisfactory partly due to the resistance of the disease to all types of therapy and partly due to the fact that the diagnosis of the condition is often made relatively late. Chronic bronchitis or recurrent attacks of acute bronchitis is in a majority of cases the result of bronchial dilatations. While repeated introductions of iodized oil are of value in bronchiectasis, it must be remembered that this type of therapy is only a paliative one, and the underlying lesion, the bronchial dilatation is not affected.

The infection, however, which is present within the bronchiectatic cavity is controlled, and as the symptoms are the results of this infection the patients become symptomatically well. Of course, a definitely rigid, fibrotic bronchiectatic cavity cannot be transformed into a normal bronchus by the introduction of oil, but the dilatation does disappear after one or more introductions of the oil.

There are four methods of introducing iodized oil into the bronchial tree: namely, the supraglottic, the

subglottic and the bronchoscopic. In the first and second procedures, a curved cannula is used. In the subglottic method, a hollow curved meedle is passed through the cricothyroid membrane into the trachea, while in the fourth, the oil is introduced through a bronchoscope.

The following general principles should be observed in using any of the methods:

1. The use of discolored oil should be avoided since it is known that iodine in a free state has been liberated and is toxic.

2. Warm oil is to be preferred, as it flows more freely and lessens the tendency toward cough.

3. Solutions for anaesthesia should be warmed.

4. Roentgenographic exposures should be made as soon after the injections as possible and cough should be prevented by any unnecessary movements of the patient.

A. Methods of Introduction of the Oil

(1) <u>Supraglottic</u>--In some patients the introduction can be done without local anaesthesia, but a better procedure is to swab the pharynx, soft palate (velum) and the base of the tongue with a 10% cocaine solution. After an interval of three minutes, 1 cc. of a warmed 1 percent cocaine solution should be dropped into the glottis with the aid of a laryngeal mirror, syringe and a curved cannula. Five minutes later the injection can be made. The patient sits facing the operation can be made. The patient sits facing the operator, and slightly inclined toward the side to be injected, and is instructed to pull the tongue forward and breathe normally throughout this stage of the operation. A 20 cc. syringe filled with warmed iodized oil is firmly attached to a 6 in. cannula having its distal end curved to a right angle or less. With the aid of a laryngeal mirror, the tip of the cannula is introduced behind the base of the tongue held over the glottis, and the oil slowly expelled from the syringe into the larynx. The patient is then instructed to breath deeply when cough threatens.

(2) <u>Transglottic</u>--This method of injection is made with the tip of the cannula passed through the glottis into the trachea. The pharynx, velum and the base of the tongue are anaesthetized as in the preceding method, but for the larynx and trachea the quantity of 1% cocaine is increased to 1-3 cc. on account of the deeper degree of anaesthesia required.

(3) <u>Subglottic</u>--In this method, any difficulty passing the larynx is avoided, and the oil is deposited directly into the trachea. After anaesthesia of the skin and subjacent tissue, a hollow needle

(curved) attached to a metal guard is pushed through the cricothyroid membrane and into the lumen of the The position of the needle should be verified trachea. by attaching a syringe and aspirating, the withdrawal of air or mucus showing the needle to be in the trachea. From 1-2 cc. of warmed 1% cocaine solution is then slowly injected through the needle, in order to anaesthetize the mucosa of the trachea and bronchi of the desired area. The patient may cough for a short time. After from three to five minutes the iodized oil should be injected by means of a metal syringe firmly connected with the needle by a piece of strong rubber tubing. The tubing allows a certain motility of the syringe and must be allowed to withstand the pressure necessary to force the oil through the needle.

The patient's position during injection is important as the distribution of the oil is determined by gravitation and the aspirating power of the lung. If one observes the progress of the oil on a fluoroscopic screen during injection, the greater portion may be seen to follow the most dependent bronchial trunks. For this reason the lower lobes are most easily filled. This is done in the sitting position. When the oil is desired to go into the right or left direction during the injection, the patient is inclined in that direction.

When the upper lobes are being injected the patient should be in a lying position. The supraglottic method can still be employed. The transglottic, subglottic, and bronchoscopic methods, however, may be employed. The patient is put on an inclined table, lying on the affected side, head downward. After the injection of the oil, the table is lowered at the head end and the oil passed down into the bronchi.

The amount of oil used to completely visualize the bronchial tree varies. At least 10 to 20 cc. are used, and when the dilatations are great enough, 20 to 40 cc. are accepted by the bronchi without difficulty. When more than 20 cc. are to be used, the transglottic, subglottic or bronchoscopic methods are preferable to use.

In using these methods, it should always be explained to the patient what is being done, its purposes and so forth, so that the operation may be assured of complete cooperation and saving of time. The patient should have been given a complete physical and roentgenological examination before oil injection is done. This also applies to the time immediately following the oil injection since the difference in the findings will lend to considerable valuable information. The connection between lipiodol and radiography as been referred

to before and will not be discussed here.

The exact method of lipiodol action is not definitely known at present, however, it may be based on the fact that free iodine is liberated within the bronchiectatic cavities in sufficient quantities to exert a bactericidal effect. It prevents the growths and multiplication of bacteria and alleviates the formation of mucopurulent material in the damaged bronchi. None of the methods referred to are intended to bring on a permanent cure, but serve only to make the patient symptomatically relieved. There are always opportunities for re-infection when the iodine has become liberated by the body, and if proper care and hygiene are not employed, the same difficulty will ultimately recur.

(4) <u>Bronchoscopy</u>--Bronchoscopy will not cure this disease, but if the patient is willing to be bronchoscoped at regular intervals, there is no question but what his condition will be improved, in that the septic conditions will largely subside and the amount of sputum raised will be diminished. It seems that the greatest benefit from bronchoscopy is obtained from aspiration of the pus from the bronchial tree, by the dilatation of any stricture or strictures of the bronchus and by the removal of any

granulation tissue that is tending to obstruct the bronchus. Some do not believe that irrigation of the bronchi or the injection of medicated with oils is of any value, although reports have been made of symptomatic cure. The early cases of bronchiectasis which are caused by the lodgement of a foreign body in a bronchus and which are promptly cured by its removal are not to be included in this discussion.

(5) Postural Drainage-A most important adjunct to the treatment of bronchiectasis is the postural method. This is probably the oldest method used since its mechanics are based entirely upon the effects of gravity upon the muco-purulent material within the bronchi. In this method of treatment, the patient should be encouraged to make systematic efforts at stated intervals to empty the cavities in order to prevent stagnation of the secretions, with resulting toxic absorption. Such posture should be assumed as is found in the individual case to give best results. The patient may hang the head over the edge of a bed or bend over the back of a chair with the head as low as possible, or he may simply lie on one side or the other with the head lower. The bronchi are simply relieved of the secretions, and if nothing else is done, the bacteris will continue to go on growing

within them and more secretions will accumulate.

Burrell in 1926 described a simple treatment, consisting of draining the cavities by posture, and the administration of creosote by the mouth or by inhalation, and claims that it is often sufficient to cure even bad cases of bronchiectasis, especially in children, and early cases. Aspiration, lavage and artificial pneumothorax have proved successful in skilled hands. Anything pertaining to surgical methods will be discussed later.

Schafer⁴⁶ tried Quincke's method on eleven of his cases suffering mainly from bronchiectasis, with very good results. The patient remains, after the usual expectoration in the morning, for two hours in a horizontal position and, when accustomed to it, the foot end of the bed is raised for about 30 cm. This method is good only in localized disease of the lower lobes.

Medical treatment may be dismissed by saying that it will never cure this condition completely and permanently, although if the patient can devote his time to taking care of his health, spending his winters in a warm dry climate, and using postural drainage or some other of the methods described, it may be that he will live a long and fairly comfortable life. 2. Surgical--Surgical treatment of this disease

has probably been less successful than the medical treatment. This can be attributed to any one of several factors. The pathological anatomy of bronchiectasis is very peculiar and the diagnosis of the condition is usually made late. This has been more true before contrast media radiography was introduced into the diagnostic methods. It has often been impossible to differentiate between unilateral and bilateral involvement. The kind of pathology existing is always hard to discern. All these conditions tended to delay surgical procedures until it was too late. Later operation in the presence of inflammatory thickening, secondary suppurative foci in the lung parenchyma, often on patients with vital organs weakened by prolonged toxic absorption, resulted usually in only partial relief of symptoms from the more conservative operative procedures, and in a relatively high mortality from radical surgical measures. Such conditions prompted surgeons to hesitate and the patients were difficult to convince having it done. The principles of surgical treatment are drainage, pulmonary compression and pulmonary extirpation.

A. <u>Drainage</u>--This procedure is effective only in drainage of a single bronchiectatic cavity or a closely circumscribed group of such cavities. These are often mistaken for pulmonary abscess and treated as such; such a mistake, however, is justifiable. The cough and sputum cease almost instantly and the condition clears up.

The indication for a drainage operation is the . presence of a circumscribed group of dilatations determined by the contrast media roentgenogram, or acute secondary abscess or gangrene. The technique for this procedure is merely a simple incision the same as is done for the drainage of a pulmonary abscess. One should make certain that the pleurae are adherent and if they aren't the periphery of the lung may be sutured to the parietal pleura, through a wide window, before establishing drainage. The latter is usually done when conditions take place acutely with severe/constitutional symptoms threatening life and drainage is urgent. Infection of the remainder of the pleura is certain to take place if proper precautions aren't undertaken and the gravity of an empyema in such a condition is very great.

Should any of these bronchiectatic cavities spontaneously perforate the visceral pleura and drain into the pleural cavity, then, out of necessity, immediate drainage must be instituted. The opening should not be closed since the drainage will continue as long as there is an infection present.

When such a condition as the above mentioned occurs, there is an intense destruction of the lung parenchyma and exposure of the bronchi. The outer wall is formed by the parietal pleura which holds the drainage within a pocket formed in this way. The exposed bronchi become extremely dilated. The procedure to undertake with such an accident, is extensive thoracotomy for drainage. The bronchi will eventually close and can be hastened to do so by frequent cauterization. The scar tissue then obliterates them.

During these operations, the larger veins of the lung are held open by scar tissue and form an open passage way for pulmonary emboli. This is best avoided by the use of a hot cautery and burning them. The dangers of cerebral embolism are lessened by having the patient lie with his head down.

A very difficult situation to handle is the diffuse type of bronchiectasis since it cannot be drained by a single incision. This procedure has been followed by a high percent of mortality. This is overcome by making multiple incisions into the lung with a cautery at different stages. This drains all the cavities. The greatest drawback to this operation is the inability to reach certain of the cavities particularly those lying close to the hilus or the pericardium.

B. <u>Pulmonary Compression</u>--The object of pulmonary compression is to accomplish three purposes. First of all the mechanical tension which tends to dilate the bronchi which are weakened by disease is counteracted. Second, the secretions within them is lessened and third, the scar tissue which later follows, shrinks and obliterates these dilated bronchi in a course of time.

The release of this tension on the weakened bronchi will check further progress of the disease. If this method were recognized early, and attempts undertaken to check it, there are no doubts that the disease would never produce the morbidity that it does.

Extrabronchial inflammation determines the degree of compression of the lung which one will get. There is still another factor which determines the degree of pulmonary compression and that is secondary sclerosis of the lung parenchyma. Contrast media radiography should be employed before and after the undertaking to determine the degree of compression attained.

The maximum degree of compression of the bronchi results from fibrosis that seems to be due to venous and lymphatic stosis. The diseased bronchial walls in favorable cases are often more than compressed; they actually disappear.

C. <u>Artificial Pneumothorax</u>--Pneumothorax in treatment of bronchiectasis is the same as that undertaken in the treatment of unilateral cases of pulmonary tuberculosis. The same precaution must be kept in mind, that is, to be positive that the condition isn't bilateral, and that the two pleurae are not adherent. In some instances the parenchyma of the lung may be involved and under those conditions hesitation to preform a pneumothorax is to be exercised.

Many cases have been reported with inprovement, and others symptomatically cured. These cases embrace many which were less than a year's duration and others more than that. Pneumothorax collaps will probably find a definite field of usefulness in early cases, particularly in children.

D. <u>Phrenic Neurectomy</u>--Extirpation of the phrenic nerve on the affected side causes a paralysis of the diaphragm and relaxation. With this event, there is a consequent diminution of the pleurae cavity estimated to be about 15-30% of its original volume. A greater degree of compression of the lung also results from the relaxation of the diaphragm particularly during the expiratory phase of coughing. It is less difficult for the patient to cough and more sputum is cleaned out of the cavities.

The degree of improvement as measured by the decrease in cough and sputum depends on the site of involvement and on the height to which the dome of compression of the lung. When the dilatations are peripheral and more recent, the prognosis immediately becomes better. When, on the other hand, the dilatations are more medial and larger, they are, of course, less affected by the attempt at compression. Various men in this field have reported different results as well as opinions, however the tendency in general seems to be in favor of this operative procedure. Most of the patients have become symptomatically cured anywhere from three months to three years or more.

It has not been my intention to go into the discussion of the different operative techniques because they are too extensive. So, suffice it for me to say that phrenic neurectomy is so simple and safe an operation, and the probability of improvement is so considerable that it should be done in all early unilateral cases. Even though it isn't so effective with centrally located lesions, it will lessen mechanical irritation and control the further progress of the disease.

E. <u>Graded Extra Pleural Thoracoplosty</u>-Extrapleural thoracoplosty involves the collapse of the

lung by extensive rib resection. It is not an ideal operation as it does not remove the disease, but in properly selected cases it produces improvement approximating a cure of all methods of compression, it seems to be the most generally applicable. It produces a degree of collapse almost like that produced by artificial pneumothorax, and is not interfered with by the presence of adhesions. Even though this operation is an extensive one, shock is avoided by doing the operation in several stages. If the operation, when complete, doesn't produce sufficient collapse, a way is prepared to do a lobectony or cautery extirpation.

The type of bronchiectasis which seems to be the most responsive to this method of treatment is the early unilateral peripheral cylindrical type. Usually this method of treatment is employed when phrenic neurectomy has failed to produce sufficient collapse of the lung. The type that is least affected by this operation is the posterior saccular type lying close to the mediastinum. This operation tends to release the intense negative pressure within the pleural cavity which is so instrumental in production of the dilatations and this prevents their formation. The nodular type of bronchiectasis associated with a tuberculous fibrosis presents a double indication in that thoracoplasty is

indicated for the fibrous type of tuberculosis.

A considerable proportion of such cases of long standing have developed so much sclerosis secondary to the retraction that the degree of pulmonary collapse attainable will not materially influence the symptoms. Patients who show marked symptoms of toxicily, high leucocyte count, temperature, or multiple abscess formation are considered bad surgical risks to benefit from a thoracoplasty operation.

The thoracoplasty may be performed by a typical vertebral resection of the upper eleven ribs after the method of Sauerbruch, or Griedrich, or by a resection of the whole length of the lower ribs. The objection here is that the whole lung is collapsed when only the lower one is affected. If only the lower ribs are resected, then there isn't sufficient collapse of the lower lobe.

The apex of the lung may be held expanded by resecting ribs from the fourth down to, and including the eleventh; this means the entire length of the ribs. The remaining upper ribs maintain the apex expanded and also a portion of the middle lobe.

F. Extirpation of the Diseased Lung--Considering the pathological anatomy found in bronchiectasis, it would seem that this phase of treatment would be made

the one of choice, but the mortality is high and not all who survive the operation are entirely cured. The mortality rates from different men have been reported to range anywhere from 26 to 61 per cent which is relatively high to warrant the risk of the operation. Some of the causes for this high rate of mortality are secondary hemorrhage, tension pneumothorax from leakage at the bronchial stump, secondary infection of the pleura, abscess formation and a severe empyema. From the figures presented, and the conditions necessary to avoid, it would seem that until the technique of primary lobectomy can be so perfected as to reduce this high operative mortality and bring about healing of the bronchial tree, and fistula in the majority of the cases, it will be largely abandoned for safer, even though from the standpoint of the pathology, less rational procedures.

G. Lobectomy after Extrapleural Thoracoplasty It has been demonstrated that extrapleural thoracoplasty is a relatively safe operation resulting in improvement approximating a symptomatic cure in a large proportion of the cases. In those instances when relief is not affected, the way is paved for the performance of a lobectomy. After a thoracoplasty operation the pleural cavity is obliterated lessening the chances for an

empyema. The disturbance to respiration and circulation incident to a primary lobectomy is obviated. Ample exposure may easily be secured by a wide incision without any marked symptoms of pneumothorax. Sauerbach states that the marked shrinkage of the lung and hilus is very favorable for lobectomy, and didn't observe any tendency for the stump to retract into the hilus. This procedure as well as the others mentioned before carries a relatively high death rate from post operative accidents. Those that are relieved do not suffer anywhere from 1 to 6 years following the operation and the amount of sputum coughed up is diminished from 500 to less than 100 cc. The chief complaints of the patient upon return is the beginning of a chronic cough and increase of sputum. Many of those people so treated have done 6 or 7 years of hard manual labor. It is true that all these methods have a high mortality rate, but it likewise must be understood that bronchiectasis is a very resistant disease and working within the thoracic cage is no simple matter.

H. <u>Compression by Upward Displacement of the</u> <u>Diseased Lung</u>-Garre has mobilized the lung after an extensive rib resection for exposure, and sutured its edge to the chest wall at a level above the dome of the diaphragm, packing gauze into the space below. This

procedure results in marked improvement. Later, the diseased lobe is resected.

I. <u>Cautery Extirpation of the Diseased Lobe</u> Resection of ribs for exposure, and cautery destruction of the diseased lobe has been practiced by many men. There is, however, great danger of hemorrhage and embolism. This is attended by quite a high death-rate but those who recover from the operation seem to be possessed with a fairly definite cure.

Whittmore reports that he has accomplished a cure by displacing the diseased lobe through the chest wall and suturing it, allowing the projecting portion to slough away spontaneously. This was done upon several patients most of whom recovered. Most of those that didn't recover, died of pneumonia.

Conclusions

1. Bronchiectasis is now known to be more prevalent than was formerly supposed.

2. Acute infectuous diseases occuring in early childhood, and other varied etiological factors are known to be very important, and the disease is at first characterized by frequent recurrent attacks of bronchitis.

3. Bronchiectasis is not incompatable with life, under proper treatment.

4. When diagnosed early, the disease is amendable to treatment if the latter is properly administered.

5. There is no permanent cure for the disease, but patients are usually symptomatically relieved.

6. Medical treatment is inadequate in that the pathology is usually too far along before a correct diagnosis is made.

7. Surgical treatment is too dangerous to be properly carried out as regards the removal of the pathological portions of the lung.

8. With the event and introduction of contrast media bronchoscopy and radiography, we may look forward to an early recognition of the disease, and be able to institute early treatment and arrest its progress. A permanent cure will probably be arrived at in the near future.

Bibliography

- 1. Bronchiectasis--Carl E. Hedblom. Article in Dean Lewis' Surgery--Vol.5, Pages 1-60.
- Iodized Oil in Radiography--Nicarid and Dollfus. J.A.M.A.--Vol.83-No.21-11/22/24. Page 1720.
- Lipiodol in X-ray Diagnosis--Armand, Dille and Moncreiff. J.A.M.A.--Vol.83-No.9-8/30/24. Page 716.
- Diagnosis of Bronchiectasis in Children-Armand, Dille and Gelston. J.A.M.A.--Vol.83-No.25-12/20/24. Page 2045.
- 5. Bronchiectasis in Adults--DeJong and Hutine. J.A.M.A.--Vol.79-No.26. Page 80.
- The Diagnosis and Treatment of Bronchiectasis with Lipiodol by the Passive Method--J.C.Gant, M.D., and J.L.Harvey, M.D. Colorado Medicine, Vol.27, No.6:185-June, 1930.
- Use of Iodized Oil in Bronchiectasis--Achsner, A.
 S. Clinic North Am., 1930.
- 8. Brain Abscess Due to Bronchiectasis--Jex-Blake. J.A.M.A.--Vol.74-No.26-6/26/20. Page 1695.
- 9. Multiple Brain Abscesses Secondary to a Kyphoscoliosis--Saelhof. Vol.75-No.26-12/25/20. Page 1203.
- 10. Bronchiectasis and Bronchitis Associated with Sinus Disease--Webb and Gilbert. Page 714. Restriction of Fluids In, Page 1807. J.A.M.A.--Vol.76-No.26-6/25/21.
- 11. Cerebral Abscess Complication--Gibbs. J.A.M.A.--Vol.79-No.9-1/26/22. Page 771.
- 12. Bronchiectatic Lung Abscess--Operation-Recovery Kinghorn and Meyer. J.A.M.A.--Vol.79-No.24-12/9/22. Page 1989.
- 13. Bronchiectasis with Unusual Complications--Davies. J.A.M.A.--Vol.80-No.15-4/14/23. Page 1103.

- 14. Ohronic Bronchiectasis Ending in Phthisis--Bray. J.A.M.A.--Vol.80-No.10-3/10/23. Page 721.
- 15. Treatment of Bronchiectasis--Burrell. J.A.M.A.--Vol.87-No.26-12/25/26. Page 1686.
- 16. Postural Treatment of Bronchiectasis--Schaefer. J.A.M.A.--Vol.80-No.2-5/19/23. Page 1493.
- 17. Bronchiectasis, Surgical Treatment of, --Graham. Unilateral Graded Thoracoplasty--C.E.Hedblom. J.A.M.A.--Vol.79-No.26. Pages 1080 and 796.
- Lobectomy in Bronchiectasis--Graham. J.A.M.A.--Vol.80-No.2-1/13/23. Page 723.
- 19. Surgical Treatment of, -- Ribadeau, Dumas and Mocquot. J.A.M.A.--Vol.80-No.8-2/24/23. Page 587.
- 20. Extrapleural Thoracoplasty--Hedblom. J.A.M.A.--Vol.83-No.4-7/26/24. Page 305.
- 21. Phreniocotumy in Bronchiectasis--Rist. J.A.M.A.--Vol.84-No.26-6/27/25. Page 403.
- 22. Treatment of Such Cases of Bronchiectasis As Are Limited to One Lobe of the Lung--Wyman Whittmore, M.D. Boston, Mass.
- 23. Bronchiectasis in Childhood--Reichle, H.S. Ohio State Med. Jour., 25:357, May, 1929.