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Bronchiectasis

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BRONCHIECTASIS

by F. Glenn Warrick

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Introduction

This paper attempts to review the subject of bronchiectasis from the time when it was first recognized and described as a pathological and clinical entity by Laennec in 1819 to the present day.

The phases of etiology and pathology have been treated rather more fully in order to gain a better understanding to the pathogenesis of this disease. With the advent of bronchoscopy and Sicard's introduction of a radio-opaque contrast medium, diagnosis has been more or less well refined. Since removal of the diseased portion of lung is the only curative treatment, the only moot aspect of the disease is its mode of production. Current thought on this subject is presented in hope that the onset and development of the pathological process will be, in a degree, clarified.

DEFINITION

Ballou, Singer, and Graham (8) define bronchiectasis as bronchial dilatations associated with bronchial and peribronchial changes which may produce thickenings and thinnings of the bronchial walls.

Others say that it is a diseased state of the bronchi in which they become dilated and distorted producing dilatations and enlargements which may be simple or multiple, local or general. The term bronchiectasis or bronchiectasia, however, merely means a dilated bronchus or dilated bronchi, and according to their form one may recognize saccular, cylindrical, fusiform and varicose dilatations.

"It is only when such dilatation is accompanied by failure of adequate drainage facilities for the normal secretions and inflammatory exudates that the actual picture of the disease presents itself" Miller (76).

Head (53) states that bronchiectasis is second to tuberculosis in causing chronic cough. 7% of all autopsies in some hospitals show evidence of the disease.

ANATOMY AND PHYSIOLOGY

Robinson (91) states that the walls vary in structure from the bifurcation of the trachea to their terminations in the lung parenchyma. Throughout their course they are lined by a ciliated type of columnar epithelium supported by a stroma with a number of mucous glands opening on the surface. Elastic elements are distributed in the longitudinal as well as the circular bundles. There are several layers, the most conspicuous being situated in the mucosa and running longitudinally. They are branched and grouped in fasciculi, allowing thus for dilatation of the bronchi as well as a lengthening. Just outside the longitudinal elastic membrane of the mucosa is another layer running mainly circularly. These latter fibers are interwoven with the muscle bundles. Other layers of lesser importance are also present.

The bronchial musculature extends throughout the whole length of the air passages. In the main bronchus where the cartilage plates are distributed around the entire circumference of the wall, the muscle fibers form a complete layer located between them and the mucosa. The muscle bundles, while in main, encircling the tube, branch off diagonally in 'lazy tong' fashion to form a more or less continuous network, or as it

has been expressed in 'geodesic lines'. The walls of the main bronchus down to a point where they have a diameter of 0.5 mm. are reinforced by cartilage plates, which prevent them from collapsing on inspiration.

During inspiration bronchi both widen and elongate. During expiration they contract and shorten. Whether this is done simultaneously throughout the entire system, or passes as a wave from larynx to periphery of lung, or vice versa, it is not known. A wave passing from periphery to hilus would best serve in emptying the lung of air.

It is readily seen that bronchi and bronchioles are actually engaged in the respiratory movements. Their elasticity and resiliency are essential to normal function. Any damage which causes loss of contractile power will lead to permanent state of relaxation and dilatation.

ETIOLOGY

Theories concerning the mechanism of production of bronchiectasis have been many since the disease was first accurately described by Laennec in 1819. Those of the earlier writers follow here immediately:

Laennec (62) in 1819 regarded dilatation as a primary anomaly and considered them to be a consequence of chronic and repeated bronchitis. It is known as the theory of mechanical pressure of stagnant secretions.

Andral (3) in 1824 stressed the importance of nutritional changes in the bronchial wall. He thought that atrophy in the bronchial wall followed a hypertrophy.

Stokes (101) wrote that paralysis of the circular muscle fibers of bronchi with loss of ciliary action and atrophy of the musculature was causative.

Corrigan (29) noted cirrhosis in the lung. He said that parenchymal fibrosis was responsible.

Rokitansky (92) said that bronchi near the pleura and areas of emphysema are most likely to be involved. He noted that bronchiectasis followed infective lesions such as bronchitis. Bronchitis, he states, is a primary factor.

Most writers of the present day admit that infec-

tions of the respiratory system play an important role. Some state that bronchopneumonia predisposes while Hoffman (57) states it merely permits manifestation of a latent predisposition.

A bronchopneumonia following acute respiratory infections, including measles, may cause a suppurative bronchitis. The bronchial walls soften and are dilated by coughing (39, 91, 87).

Morse (78) found that 3% of admissions to a tuberculosis sanatorium were bronchiectasis. Antecedent respiratory disease in a series of 42 cases was as follows: 3--whooping cough, 1--measles, 1--pneumonia, 3--influenza, 17--had frequent head and chest colds. 3--pleurisy, 2--had associated abscesses of the lung, and 2--had associated pneumoconiosis. Warner (106, 107) states that 59% of bronchiectatics have some known prior illness. Pneumonia accounts for 30%, lung abscess for 12%, influenza 5%, whooping cough 4%, "acute bronchitis" 3%, measles 2%, bronchogenic carcinoma 2%, and foreign body for 1%.

Boyd (16) states that pneumonia is the commonest precursor in all types. Lobar atelectasis is the result of infection or injury of the bronchial wall, with secretion and consequent plugging of the bronchioles.

Bronchopneumonia in childhood causes an unequal-

ity of air distribution which may result in "bronchiectatic emphysema". As growth proceeds these dilated bronchioles move inwardly and may become the dilated bronchi of adults.

Many cases of bronchiectasis could be traced as a result of the influenza epidemic in 1918 (84). Bronchiectasis and influenza can and do occur independently. In influenzal pneumonia, streptococci are secondary invaders. These organisms cause much necrosis and, lymphatic and interstitial suppuration. This sequence of pathology leads to the production of bronchiectasis (9).

Chronic pneumonia is usually followed by multiple dilatations, or the initial pathology may cause destruction of fibroelastic tissue and coughing leads to distension of lung and bronchi. Many cases start insidiously. Compression of lung by pleural fluid or air does not seem to be a factor. (18, 43, 58, 67, 81)

Abscess of the lung and bronchiectasis have been noted to occur together in occasional cases (50).

Wm. Stokes(101) in 1844 blamed inflammation as a responsible agent. He felt that as a result of the inflammatory action in the bronchial wall, the muscular structure itself was paralyzed. He suggested that this paralysis might also contribute to the difficulty of expectoration in certain cases. The same process

would effect a loss of the vibratory action of the cilia which would tend to allow the accumulations of secretions.(101).

He states that whooping cough in children of two to three months is sufficient to produce fullest development of the disease. There is bronchial irritation accompanied by violent cough, and this occurring at a period when tissues are still imperfect, and the muscular apparatus not yet fully developed, is so powerful a cause, that it seems only wonderful that the lesion does not occur more often. (101).

In others bronchial irritation may continue for years. He also noted it as an accompaniment of tuberculosis of the lung (101).

According to Miller (76) any disturbance of the bronchial function may be causative. This function is achieved by broncho-motor activity and collateral respiration. With the loss of normal contraction and distension, power for emptying secretions is lost and secretions accumulate and stagnate in the affected air passages. This is also said to disturb the distribution of air and the equilization pressure within the lung. When air distribution becomes unequal, intrathoracic traction and intrabronchial pressure exert a distending effect upon the bronchial walls.

Developmental factors or interference with the functional efficiency of the bronchi lay the ground work for the destructive influences of infection. (82).

During childhood pulmonary diseases cause a production of greater amounts of interstitial tissue, There is proportionally greater amounts of lymphatic channels and lymphoid structures in children. In the adult the inflammatory exudate in the alveoli is almost completely absorbed when inflammation subsides. In children, on the contrary, the interstitial framework and its extensive interlobular and peribronchial tissues are more frequently invaded, and associated with this the lymph channels and neighboring lymph nodes are severely involved, blocking lymphatic drainage, and frequently interfering with the free intake of air into the bronchi more severely involved. Such conditions resolve with much greater difficulty and consequently pulmonary infections in childhood much more frequently results in chronic, more or less permanent, pulmonary sequelae. The above changes are most often due to influenzal, mixed influenzal and streptococcic infections and the bronchopneumonias associated with measles and pertussis.(82).

Development and continued growth may be capable of restitution and elimination of destructive processes

which on first impression might seem irreparable. (82).

Meiks (75) in a series of 71 cases, all children, fifteen years of age or under, had 49 that showed definite cavitation. 18 were merely symptomatic. The age of onset was found to be highest at 2 to 3 years and at 6 to 7 years, periods which correspond to the age of respiratory infection. 72% began following inflammatory lesions of the bronchi or pneumonia. 81% had sinus disease. The severity and duration of sinus and lung pathology had no definite relationship.

Fordl (41) in a significant study of 100 cases reports the following findings:

1. 52 were male and 48 were female. 77 were under 30 years of age. 41 had the pathology in the left lung, 23 in the right lung and the disease was bilateral in 36.

2. He concluded that it was a chronic disease. 50 patients had symptoms for less than 5 years. 47 had the disease for more than five years and 3 were indeterminate.

3. The onset is early in life. 17 patients, ages 4 to 54, had had symptoms from infancy. 80% dated their symptoms from the first decade.

4. In 45 the disease followed an upper respiratory infection. It followed some infectious disease of childhood in 12.

5. 86% of those examined by x-Ray showed sinus changes. In 24% the changes were marked. F

Fordl (41) concluded and advised that Roentgenological and bronchoscopic examinations are needed for early diagnosis.

Gairdner (44) concluded that almost all so-called bronchial dilatations, particularly those of the abrupt, sacculated character, were the result of ulcerative excavations of the lung which communicated with a bronchus.

The lower lobes are affected in 80% of cases (44). It is interesting to note that Laennec (62), Stokes (101), and others wrote that the lesion was more common in the upper lobes.

Bronchitis has almost universally been blamed in a greater or lesser degree (7, 13, 42, 45, 88, 104). The trachea, large and medium sized bronchi are usually involved and is most often bilateral. There is a congestion of the superficial membranes, lining the walls. The walls themselves may be uninvolved. The walls may be trabeculated with new cells formed in the deeper epithelial layers. The lesions at post-mortem may not be marked. Connective tissue infiltration is the usual thing. The glands may atrophy or become cystic. One may see a putrid bronchitis without bronchiectasis.

There may also be associated disease in the pulmonary parenchyma with bronchitis.

Bronchial dilatations may be atrophic and dry. Hypersecretion may operate as an etiological factor. Asthmatic bronchitis has developed into bronchiectasis. However a putrid bronchitis with abundant pus in which the bronchi are literally drowned may not develop bronchiectasis.

Ferguson (39) states that a destructive bronchitis which causes a dissolution of the mucosa, submucosa, cartilage and muscle may be causative. It has been said that a bronchiectatic cavity is not a dilated bronchus but an excavation in the lung substance, starting in a bronchus.

Rokitansky (92) said that bronchitis was the more frequent primary cause. It causes an atony and paralysis of contractile elements. Dilatation is caused by inspiration and cough (to dispel secretions). Smaller bronchi were found to be completely filled with secretions.

He felt that the saccular form was due to occlusion of a bronchus to inspired air. The surrounding parenchyma collapses and the bronchus pushes into the space.

The co-existence of bronchiectasis and disease of

the accessory nasal sinuses has often been noted and recorded. Thompson (102) in 1914 suggested that bronchorrhoea might be due to chronic suppuration of sinuses. Hodge (56), Clerf (24), Webb (110), Quinn and Meyer (89), and McLaurin (74) have likewise noted the relationship.

Hodge (56) noted that paranasal sinus disease and bronchiectasis occurred together in 75% of his series of bronchiectasis. Clerf(24) found the relationship to exist in 82% in his series. Quinn and Myer (89) claimed a percentage of 58%.

Clerf (24) says the associated disease is usually bilateral while Adam (1) asserts that it is unilateral. Hodge (56) states that the maxillary sinuses are most frequently involved in the co-existent disease. Hodge (56) instilled iodized oil into the trachea. The coughing which resulted failed to spread the oil to the sinuses. The palate acts as an effective barrier.

Quinn and Meyer (89) demonstrated opaque oil in the thorax after instillation into the nasal passage of a sleeping patient.

McLaurin (74) also demonstrated that iodized oil instilled in the antra passed into the chest. The assumption may be made that following a sinusitis, chronic bronchitis develops which may later become bronchiectasis.

Clerf (25) noted that the repeated inspiration of infected secretions set up a chronic laryngotracheitis. He noted a change in the angle of the carina and that the bronchial spurs are widened. This is supposed to represent evidence of enlarged peritracheobronchial and peribronchial lymph nodes. This may be a cause of bronchitis which will lead to the formation of bronchiectasis.

Dunham and Skavlem (36) found in 25 cases of bronchiectasis that 8 antra, 4 ethmoids, and 13 antras plus ethmoids were involved.

Dennis (33), in a study of 50 cases of sinus disease with complications found that 24 had bronchiectasis.

Quinn and Meyer (89) observed patients for years being treated as tuberculous who were suffering from sinusitis and bronchiectasis. They found the two diseases associated in 21 of 38 patients. The average duration of symptoms was found to be from 5 to 7 years.

Davis (31) attempts to evaluate the role of nasal sinus infection in relation to bronchiectasis and makes the following points:

1. The infection may extend by direct extension along the mucosa, or spread through the blood or lymph stream.
2. The infection may aggravate or increase an ex-

isting lesion in the lower respiratory tract.

3. Or nasal infection may merely be a part of a generalized infection of the upper and lower respiratory tracts.

Graham (47) in observing a bronchial fistula noted that the bronchial mucosa reacted in 24 hours to infection in the nasal sinuses. Evidence of inflammation subsides in both regions at about the same time. The bacteria found in the two areas are always the same and usually streptococci.

Young (113) states that there are 6 mechanisms through which the respiratory system is affected through nasal disease. They are as follows:

- a. By direct spread of the infection.
 - b. By mechanical nasal obstruction, the air is not warmed and filtered, and causes a catarrhal inflammation in the alveoli.
 - c. Through the effect of inhalation of septic organisms from the mouth and throat and masses of infective material.
 - d. By altered blood immunity conditions with absorption from foci of the diseased areas.
 - e. When oral sepsis leads to septicemia.
 - f. By causing myositis, pleurodynia, etc.
- Lymphatic absorption from the antra and frontal

sinuses is by way of the submaxillary and internal jugular nodes. The lymph passes from there to the lymphatics and thence to the great veins and finally to the heart. The right heart pumps this lymphatic return into the lung fields (80). They were unable to demonstrate direct lymphatic communications from the plexuses of the nodes of the head and neck to the lungs and pleura.

Raffe (90) believes that the two processes are of simultaneous origin. When both are then existent, they exert an influence on each other.

Adam (2) suggests that aspiration of pus from the antrum during sleep may act as a factor. Mullin (79) states that sinuses may be diseased without causing symptoms.

Watkins (109) considers that oral, and nasal diseases play a prominent part in the production of bronchiectasis. He feels that the incidence of bronchiectasis in otorhinolaryngological practice is relatively high. He quotes several series of high incidence and gives his own series as having an incidence of 89.3% of concomitant disease. Watkins admits that the diseases may merely be coexistent and wholly unrelated. The sinusitis may also follow bronchial involvement. Once both processes are firmly established, one focus

keeps the other active by constant reinfection.

Atrophic rhinitis and extensive pyorrhea has been named as a causative factor. With sinusitis the disease in the lung is usually bilateral.

Ballou, Singer, and Graham (9) state that no operation on sinuses will cure bronchiectasis, which has been demonstrated by lipiodol. Bronchorrhea without bronchiectasis may improve markedly. We have little proof that nasal sinusitis may cause bronchiectasis. Nor do we have any evidence to support the claim that bronchiectasis causes sinusitis.

There is normal physiological dilatation of a bronchus during inspiration. This may permit permanent dilatation when constrictor properties of the bronchus are injured as by infection. The potentialities of this mechanism are still problematical (5).

The negative pressure of the pleural sacs do not act as a factor in the normally aerated lung because the pressure change is in the alveoli and not in the bronchus (4).

By bronchoscopy a bronchus is seen to dilate during inspiration and narrow on expiration. The forces acting are a direct pull of the expanding thorax on the bronchi, transmitted through the parenchyma, and second the differences in pressure in the lumen and out-

side its wall occurring on inspiration.

A greater dilatation occurs during inspiration when the normal elasticity of the bronchial wall has been destroyed by disease. The bronchus will not return to normal size with cessation of the dilating force since its power of contraction is diminished on account of the loss of elastic and muscle tissue in its wall (107).

Atelectasis which is common in bronchiectasis may exert an added effect. The pull of the expanding thorax on the bronchi will be transmitted directly to the bronchus through the solid atelectatic lung. There will result a greater pull on the bronchus. The normal telescoping movements of bronchi and passage of air are interfered with, so allowing the stagnation of secretions. Secretions themselves are probably a negligible dilating factor, but secretions do favor further infection and weakening of the bronchial wall.(106).

Contemporary observers, particularly those of the past ten years, assign considerable responsibility to atelectasis as an etiological agent in the production of bronchiectasis.

Warner (107) feels that atelectasis is a big factor in the establishment of permanent dilatations. Deflated parenchyma leads to an increase in negative pressure which results in an increased dilating force on

the bronchial walls. The movements of the bronchi are impaired and this allows the stagnation of secretions.

In atelectasis when collapse was complete and persisted, bronchial dilatation rapidly ensued. Secretions alone may cause obstruction as in post-operative atelectasis which leads to bronchiectasis (39).

Robinson (91) states that a persistent fetal atelectasis when the chest cavity later expanded beyond the capacity of the atelectatic lung is a mechanism of production.

Hedblom (54) records that atelectasis in adults may produce bronchiectasis by virtue of an increased negative pleural pressure, lessening as it does the support to the outside of the bronchial wall and allowing atmospheric pressure to exert its dilating effect.

Royles and Todd (93) express the belief that collapse of the lung, if maintained for any length of time, is a prominent factor in causation of the disease.

,Lee (63) was able to produce massive atelectasis experimentally by bronchoscopic introduction of human bronchial secretions into the bronchus of a dog. The cough reflex was depressed by the use of a barbiturate. This experiment suggests that retained secretions can cause atelectasis which will lead to bronchiectasis if

the lung is not re-aerated rather soon.

Anspach (6) asserts that atelectasis precedes and plays a prominent and most constant role in the development of a common form of bronchiectasis of the lower lobe.

It has been noted that a primary acquired atelectasis may secondarily lead to bronchiectasis. A fertile source of bronchiectasis in children is pulmonary collapse. When mucous or secretions plug a bronchus, then each expiratory effort must compress a vesicular structure. Peripheral bronchial tubes will in course of time become dilated. Blood vessels may be narrowed and obliterated during the emphysema and bronchiectasis. So massive atelectasis and patchy atelectasis may play a part. (50).

Andrus (5) lists pulmonary atelectasis as an important causative agent. In atelectasis other thoracic viscera and the over-expansion of the remaining pulmonary tissue fill the space deficit. This causes an elastic hypertension in the pulmonary tissues. The normal negative intra-pleural pressure is doubled in atelectasis. So, since negative pressure is due to elastic recoil of the lung, this property must be doubled to increase the negative pressure.

Elastic hypertension exerts pull and tug on lung

parenchyma and conducting tubes. The direction of the maximum pull is horizontal and to the lateral side. The diaphragm compensates vertically, and antero-posterior over-expansion results in no readily observable effects. For the lower lobe the force is essentially at right angles to the long axis of the lower lobe bronchi. Thus the force is concentrated at a site where dilatation commonly occurs.

Bronchi in atelectasis are specifically and selectively exposed to simultaneous weakening by infection, and gross abnormal dilating stress. First, over-expansion occurs in the respiratory air chambers but the bronchi are influenced by the elastic hypertension factor still. A constant pull on the walls, even in absence of infection, may cause dilatation and the "dry" form of bronchiectasis.

The elastic hypertension causes emphysema. Emphysema is frequently seen in bronchiectasis. So both may be the end results of the same cause, and as would be expected the emphysema is most marked around the vicinity of bronchiectatic or atelectatic disease.

Atelectatic collapse of bronchial wall from gas pressure may result in a pneumonic area. The exudate is absorbed but the bronchiole remains plugged. Then it collapses if the surrounding tissue is soft. If the

surrounding area is rigid, then bronchiole might collapse into the cavity producing the saccular type of bronchiectasis.

The clinicopathological correlation of atelectasis and bronchiectasis is the only relationship not controversial. An acute pneumonic origin can be traced in all but a few cases of bronchiectasis. Fibrosis is now recognized as a result of bronchiectasis and atelectasis. Only patchy atelectasis is necessary, not lobar (5).

Corrigan (29) felt that the contraction of fibrous tissue around the bronchi exerted an additional influence upon dilatation.

Van Allen and Wang (104) have demonstrated that extensive fibrosis of visceral pleura will cause marked contraction and cirrhosis of a lung lobe independent of any compression, or of bronchial obstruction.

Bronchiectasis may occur without either fibrosis or pleural adhesions. A consistent pathological finding is chronic inflammation of the bronchial wall, often with complete destruction of elastic tissue and muscle. So it must be due to initial damage and subsequent weakening by non-specific infection (108).

Pulmonary fibrosis has often been mentioned as a factor. The fibrosis of tuberculosis in adults may

operate to produce dilatations; this relationship is much more significant when found in an upper lobe (8).

Fibrosis in interstitial tissues is a factor in producing dilatation by contraction and distortion. Inflammation may spread from bronchi to peribronchial tissues, especially from nose and throat infections. Lung abscesses often are seen as a result of bronchiectasis (87).

Warner (107) is of the opinion that neither fibrosis of parenchyma nor pleural adhesions need to be present in production of bronchiectasis. He states that the bronchial wall is at fault. There is at first a weakening in the bronchial wall and then secondary changes in the parenchyma.

It has been shown that fibrosis and pleural adhesions were absent in lobes removed at operation. They also observed that atelectatic bronchiectasis developed so rapidly that fibrotic changes could not occur (107).

Inflammatory reaction in the walls of the bronchioles caused them to swell and form obstruction which leads to atelectasis. Central obstruction could produce both dilatation and atelectasis. The most common mode of production is obstruction in the terminal bronchioles.

Contraction of fibrous tissue would cause a constriction of the bronchi and protect them from dilata-

tion. If fibrous bands are attached to opposite sides of a bronchus and then anchored to fixed points, dilatation might occur, as between the spine and the postero-lateral thorax in lower lobes. The direction of the axis of anchorage is at right angles to the bronchi at the base and this is a common seat of bronchiectasis. There is usually atelectasis which withdraws the lung from the thoracic wall and does not permit anchorage. Respiratory excursion is limited when extensive fibrosis occurs. This does not permit intrapulmonic pressure to exceed a certain maximum. So the lung is protected from materially increased stress. Fibrosis is often lacking in early bronchiectasis. The onset may be rather sudden which is not compatible with the picture of fibrosis. Fibrosis can act as a cause but does not do so clinically in many cases. It may be more of a protective factor against dilatation.(5).

Obstruction by foreign bodies, new growths, aneurysms, enlarged tuberculous nodes, and pulmonary secretions are known to be causative. There may be also an innate susceptibility coupled with impaired capacity for removing secretions; repeated infections would serve to aggravate this tendency. Obstruction seems to be a major factor, from whatever cause (39).

Bronchostenosis may be a factor. Usually a stone

forms an ulcer which heals. Scar tissue formation with subsequent contraction is the mechanism (50).

Metastatic carcinoma may operate by infiltrating peri-bronchial nodes and causing narrowing. Non-specific inflammation accompanying tumors may cause fibrosis and shrinking of lung parenchyma with bronchiectasis.

Various workers have demonstrated a metaplasia of bronchial epithelium as caused by chemical irritants and infections but as yet there has not been sufficient evidence to suggest that proliferative changes in the bronchial epithelium are responsible for the production of bronchiectasis (50).

A few have suggested that carcinoma occasionally develops in bronchial dilatations but only a few authentic cases have been reported. The relationship is not extremely significant.

Rokitansky (92) and Cohnheim (26) demonstrated that purely mechanical factors act, by occlusion of a bronchus with foreign bodies. Paul Claisse (23) also wrote many years ago of the same thing.

Broncholithiasis is known to be causative. Stokes (101) and Rokitansky (92) stated they had never seen broncholiths without corresponding dilatation. If a stone is in the parenchyma, a lung abscess is more

likely to be the effect.

Benign and malignant tumors of the bronchi and peribronchial tissues can act by causing an occlusion of the bronchus from within or without. All forms of pulmonary carcinoma are likely to result in dilatations, cavities and atelectasis. If a tumor rather suddenly occludes a bronchus, the corresponding portion of lung tissue becomes atelectatic. If growth is slow, dilatations may develop beyond the site; and cough and expectoration of bronchitis may develop. As size of dilatations increase, secretions stagnate and increase in amount (50).

Weinberg (111) attempted to evaluate the role of obstruction experimentally. Using an aseptic technique he introduced sterile sand burs into the bronchi of rabbits. He was able to produce bronchiectasis in this manner. He concluded that bronchial obstruction is a primary factor. He feels that in the presence of a simple obstruction, the normal bacterial flora of the bronchial tree is capable of setting up an infection in the bronchi which will weaken the wall, and the surrounding atelectasis permits dilatation.

In speaking of partial or fixed obstruction Andrus (5) states that during inspiration the bronchus does not fill well and the normal parenchyma which sur-

rounds, expands and encroaches upon the space of the obstructed portion. Hence it is specifically protected against mechanical injury. Normal bronchi of the same lung will be overdistended in an effort at compensation. However the respiratory air chambers are those influenced and emphysema results.

During expiration normal elastic tension and recoil are responsible chiefly for expelling air from the lung. Some of the normal elasticity may be lost and air is expelled more slowly from the obstructed portion.

Valvular obstruction should obstruct equally in both directions as a rule and be the same as in partial obstruction. So obstruction does not constitute an appreciable dilating force, and that dilatation distal to obstruction is explained only by complete obstruction and atelectasis (5).

Ferguson (39) mentions that pre-natal and post-natal infections and atelectasis may prevent the attainment of adult bronchial and parenchymal structure, by disturbing the normal course of differentiation in the development of the lung from infantile to adult type bronchi. The terminal bronchi end in buds which distend with the first few breaths and are comparatively large and few in number. As the lung develops, new branches are formed in the peripheral part of the bron-

chial tree, and from these new alveoli are constantly developing. The original alveoli of the infant become the non-respiratory bronchioles of the adult, and the peripheral bronchioles become the reinforced cartilaginous bronchi near the root of the adult lung. As growth continues the preponderant interstitial tissue thins out to attain the adult relation.

Pulmonary diseases during childhood influences the rate and character of differentiation, and also establishes a predilection for these same areas for subsequent development of disease (39).

Syphilis as a cause of bronchopneumonia is not common. Balzer (11) states that syphilis is more likely to be a factor through causing a bronchostenosis. The relationship of syphilis to bronchiectasis has been mentioned by many, but definite proof is still lacking.

Smith (100) feels that fusospirochetal disease in the respiratory tract is often times directly a cause. He thinks there is a parallelism between this and aneurysms of Treponema pallidum infections. He was able to produce lesions in rabbits similar to those found in man with Tr. microdentium, and Tr. macrodentium. The organisms found in the sputum from the depths of the lung included the above as well as S. Vincenti, S. Buccalis, fusiform bacillus, vibriosis and cocci.

The pathology is a focal necrosis of elastic tissue in the bronchial wall.

He found these organisms by culture, one or more, in 49 out of 60 cases. In using Levaditi's stain on tissues he found the fusospirochetal organisms in 8 out of 12 specimens. These cases were noted to respond to therapeutic administration of neosalvarsan.

Ferguson (39) also mentions the feeble cough mechanism in children which allows secretions to accumulate and cause obstruction.

Miller (76) states that cough is an important mechanism in discharging bronchial contents. Cough may also serve to reinflate collapsed parts. If a bronchus is surrounded by a deflated area, the air cushion effect is lost and distension obtains. Cough becomes effective as a dilating force when the normal contracting force of the bronchus is lost. He feels that it is a great pressure and acts by its cumulative effect.

The role of cough is questioned. The lung is expanded by the mechanical traction of the thoracic wall. During inspiration the pressure is slightly higher in the bronchi. However full inspiration is accompanied by dilatation of the bronchi which provides for increased gas flow without raising the pressure. During expiration the bronchi are exposed to a compensatory

compressing force. So cough alone is not a real factor. Clinically 2,000 soldiers with chronic pulmonary disease, followed for 15 years, failed to show development of bronchiectasis from cough due to chronic bronchitis. (5).

In pulmonary tuberculosis a mixed infection may result. Ulceration and scar tissue formation in bronchi follows. The peribronchial glands may be involved. McCrae and Funk (71) have noted in rather frequent association of apical bronchiectasis and tuberculosis.

Osler (86) stated that dilated bronchi are often seen in tuberculous lungs.

Dickey (34) reports 2 cases of coincident pulmonary tuberculosis and bronchiectasis in children. He feels that tuberculosis may be the cause of bronchiectasis in certain cases. He feels that in the presence of bronchiectasis with a tuberculous history one should always suspect the presence of both.

Morse (78) states that bronchiectasis in tuberculosis may be related in one of the following ways:

a. The dilatation may occur immediately in the pathological area of the tuberculous process, the local pathology causing a destruction of the bronchial wall, atelectasis, fibrosis, obstruction and eventually dilatation.

b. When the two exist independently in separate parts of the lung.

c. A post-tuberculous bronchiectasis.

Dean (31) found that avitaminosis A will cause bronchiectasis in guinea pigs, and sinusitis in monkeys. He also found that a high vitamin diet and a deficient protein diet also caused a suppurative sinusitis and otitis media.

Sippe (99) claims that hypoglycemia and ketosis are etiological factors. He gives nine cases in which by use of dextrose therapy a relief of symptoms was achieved.

Allergy has also been incriminated as an etiological factor.

Rachitis chests, weak respiratory efforts, or malformed chests have been blamed, but bronchiectasis occurs in normally shaped chests also (9).

Pneumonconiosis has been suggested as a factor. The particles are absorbed by lymphatics, go to nodes and there obstruct the lymphatic return. This leads to fibrosis. Its role in bronchiectasis is not very certain. It takes years to develop. Anthracotic glands may compress a bronchus sufficiently to produce dilatations early (50).

Poisonous gas may produce ulceration and necrosis.

The slough may form a block, causing obstruction.

Healing with scar formation may cause stenosis. Either of these conditions may lead to a bronchiectasis.

Mediastinal pleurisy has been mentioned as a cause. It is usually non*tuberculous in children. It is supposed to operate by causing an external pressure on the bronchi, effecting a collapse of them (50).

PATHOLOGY

Four distinct types of bronchiectasis have been described:

1. Pure atrophic (cylindrical). Here the bronchus is dilated in its entirety to the pleura. The epithelium is normal, the elastic tissue is torn and cartilage is destroyed. There is not cellular infiltration or connective tissue overgrowth.

2. Atrophic with inflammation. This type in addition to the above shows evidence of an inflammatory reaction in the subepithelial layers.

3. Sclerosing type. Here the bronchi are simply sclerotic.

4. Saccular. These may show elastic tissue in the dilated cavity or the cavities may be irregular and rigid, devoid of any vestiges of elastic tissue elements.

Andral (3) in 1843 described three types:

1. One or more bronchi present through their entire extent a greater or less increase of capacity. since the bronchial parietes is thicker than normal, he says that the cause of dilatation must be due to something more than the mechanical effect of cough, or the accumulations of mucous. There is in addition a

sort of hypertrophy of the tissue of the bronchial parietes.

2. This type is merely an enlargement at one point. The dilated portion pushes into the parenchyma.

3. This type consists of a succession of dilatations and narrowings along the course of a bronchus. The cause of these is a mere mechanical effect of accumulated mucous. The parietes of the bronchial wall is thin. This type is more common in children.

Robinson (91) asserts that temporary dilatations do occur which probably return to normal if no disintegration takes place in the wall. Irreparable damage to elastic and muscular elements leads to dilatations which become inexorably fixed. The essential pathology is a weakening or loss of the integrity of the musculo-elastic system.

Gaylord and Aschoff (46) state that round cell infiltration may become more extensive and permeate between mucous glands which ultimately atrophy and disappear. The round cells and granulation tissue invade the cartilage and split up and destroy the elastic tissue structure. As a result the wall of the bronchus becomes softened and loses its elasticity and when exposed to excessive intrabronchial pressure, dilates, producing the condition known as bronchiectasis.

Ferguson (39) states that there is first a violent inflammatory reaction in the bronchus with destruction of tissues. There is frequently necrosis which extends into the parenchyma forming the pulmonary "excavation". There is a leucocytic infiltration. The smaller bronchi are filled with purulent secretions.

Following the acute process there is some healing. Granulations start to become epithelialized from remaining islands of epithelium. New cells are changed from ciliated columnar to squamous or cuboidal type. There is scar tissue formation and few or none functioning alveoli remain.

Weinberg (111) in producing experimental bronchiectasis by causing bronchial obstruction noted the following significant pathological changes in the bronchial wall with special consideration for the time element.

1. Early, that change noted after 20 minutes was an enlargement and breaks in the alveolar walls with some congestion.

2. In six to forty hours he found a mucopurulent membrane around the foreign body, with an associated peribronchial atelectasis. There was a leucocytic infiltration and tearing of the alveoli.

3. Late. During this phase there was complete bronchial obstruction and massive atelectasis. The

non-obstructed lobes showed early bronchiolitis, peribronchial atelectasis and pneumonitis. He states this is due to spread of pus from the affected lobes by the way of the bronchial tree. Degeneration and necrosis was observed in the bronchial wall.

Robinson (91) made his observations on sixteen surgical specimens, all material was fresh, and some represented cases of fairly early development. All specimens were of the lower lobes and some included portions of upper lobes. The dilatations were fusiform or cylindrical in type measuring from 40 mm. to 1.5 cm. in diameter. The secondary branches were most frequently involved. The mucosa was a peculiar pinkish-yellow color, and was soft, succulent, almost redundant in appearance. The lumina for the most part contained in their proximal portions very little secretion. The finer bronchioles were usually well filled with a yellowish mucoid material; the walls were usually thicker than normal. In the vast majority of them there was little gross evidence of involvement of neighboring lung tissue beyond a variable amount of lung collapse. There was no evidence of tuberculosis.

There were definite inflammation of the bronchial wall in all cases with degenerative changes in certain essential structures added thereto. The more acute and

early cases showed a marked hyperplastic state of the mucosal epithelium. The cells were large, swollen, irregular in outline and size, and tended to grow up in a polypoid manner increasing thereby the thickness of the mucosa. In all but 2 cases the cilia of the lining epithelial cells were intact. Considerable quantities of exudate, consisting of mucous with large numbers of small polymorphonuclear leucocytes, occluded the smaller bronchi. In view of frequency of hemoptysis in these cases it was interesting to note that very few ulcers of mucosa were found. These occurred in the most acute cases, and were microscopic in size. (91).

The most striking changes, however, were found in the subepithelial layer. Here, lying between the lining epithelium and the cartilage plates are found important structures, namely the bronchial musculature and elastic fibers. In this zone a most marked inflammatory reaction occurred. The inflammation was somewhat granulomatous in character, the growth of new blood-vessels and connective tissue being a prominent part of the process. In the earlier stages, as seen in the acute cases, this granulomatous feature was not so apparent, and the muscle and elastic fibers were found for the most part intact.

Here and there, however, beginnings of a degener-

ative process has involved the septa between two bronchi, tending to a melting down of the intervening wall. In the rest of the cases, where the process was more definitely established, it was interesting to note that the lining epithelium was intact throughout, and consisted of normal stratified columnar epithelial cells with well-defined cilia. Fresh scrapings were made from some of these and examined with dark-field illumination. The cilia were observed to continue their whipping movements for some hours after removal (91).

A definite thickening of the intima with stenosis of the bronchial arteries was found in 65% of cases. These lesions were found only in the bronchial arteries and only in those of the dilated bronchi. The thickening of the walls were intimal and therefore of the type of endarteritis obliterans. Whether this is the cause or effect of bronchiectasis is difficult to say.

Robinson (91) believes that infection is of prime importance in the development of this disease. In early stages it is probably a paresis of the muscular system leading to loss of tone with consequent dilatation.

If the infection is of short duration, recovery may ensue. If long continued permanent damage follows with loss of resiliency leading to dilatation. This marks the onset of a vicious cycle. Secretions accumulate,

become infected and spread to adjacent walls.

Loss of cilia or metaplasia of epithelium to the squamous type may develop and permit accumulations to develop, a definite physiological block. Robinson observed one dilated bronchus completely lined with squamous epithelium (91)4

Anspach (6) noted these early pathological changes in bronchiectasis:

1. An intact bronchial wall.
2. Absence of bronchial distortion by enlarged glands.
3. An absence of dilatations in the bronchi.
4. Intact condition of lining membrane, muscle and cartilage.
5. Unusually thick exudate filling the smaller bronchi.
6. A slight microscopic induration of the para-bronchial tissues.
7. Frequent small areas of pneumonic consolidation of recent origin in other lobes.
8. Atelectasis of lower lobe.

Anspach thinks that the pathogenesis of the disease is as follows:

There is first an atelectasis due to an obstruction of the bronchi with tenacious secretions. Radiat-

ing bronchi fold together in a parallel manner. This causes poor drainage. The bronchial walls become weakened and the increased negative intrathoracic pressure permits a bulge or stretch of the bronchial wall. With dilatation some air enters and may partially re-expand the lung. Atelectasis precedes and plays a prominent and most constant role in the development of a common form of bronchiectasis in the lower lobe.

Opie (85) asserts, "When the epithelial lining of a bronchus is destroyed, coagulative necrosis of the underlying tissue occurs, and may extend a variable distance into the bronchial wall, not infrequently penetrating into or entirely through the muscular layer. These changes furnish an explanation of bronchiectasis following influenza."

Erb (37) states that bronchiectasis has its onset with an acute upper respiratory infection. The earliest lesion is an ulcerative bronchitis.

Erb (37) divides the pathological process into the destructive stage and the stage of repair. In the first the following observations were made:

The small bronchioles were filled with a purulent exudate. The epithelium is partially destroyed and there was considerable polymorphonuclear infiltration of the wall but some muscle fibers remain intact. The

Surrounding alveoli are filled with exudate and some are collapsed. Necrosis of the wall advances until no original elements of the wall are discernible. Then a little later bronchial dilatation occurs. Necrosis may extend rather unevenly into pulmonary tissue. This irregular distribution may explain the saccular type. Rents or fissures through the entire wall may extend to alveoli also giving unequal dilatation.

Purulent exudate of the lumen may be invaded by fibroblasts, causing obstruction. One of Erb's cases had a partial bronchial obstruction due to a large caseous gland which later eroded into a bronchus. This caused a destructive process in the bronchi followed by dilatation. "In six cases there has been no evidence of epithelial regeneration or of fibrosis of the intervening parenchyma" Erb (37).

In the stage of repair, little evidence of any reparative processes were noted until the sixth week after the onset. Islands of epithelium approaching the squamous type appeared; Erb thinks this presents evidence of regenerated epithelium. Re-epithelialization is usually preceded by the formation of a zone of highly vascular granulation tissue in the bronchial wall from which muscle and elastic fibers have wholly or partially disappeared.

As repair goes on, the relining of these cavities continues until it is more or less complete. Microscopically these are lined with a continuous sheet of epithelium. In one case the epithelium approached the normal ciliated variety. In all cases the parenchyma may be replaced by a dense connective tissue.

In replacing parenchyma by fibrous tissue a certain amount of shrinkage occurs around the bronchi, from side to side and parallel. In the shrinkage of the underlying fibrosing granulation tissue, the mucosa is thrown into transverse folds to accomodate itself to the shortened bronchus. Bronchial dilatation can and does take place long before the fibroid lung contracts, and the dilatation is, therefore, quite independent of such contraction.

An obstruction in a bronchus Erb says, "it is conceivable that ulcerative bronchitis without obstruction may heal without bronchial dilatation, and this probably frequently occurs. But in obstruction, destructive forces are enhanced and due to inadequate drainage, permanent damage results."

Obstruction may be caused by foreign bodies, caseous glands and tumors, and inflammatory exudates. Due to the edema of an inflammatory process plus the exudate, a bronchus may occlude itself. Cilia are destroyed and drainage impaired. A thick tenacious, stag-

nant exudate may later become organized.

Inadequate drainage can be attributed to a loss of muscle fibers and peristaltoid movements, loss of elastic fibers, and replacement of ciliated epithelium by a modified, non-ciliated epithelium. The contour of saccular or fusiform cavities themselves are an impediment to drainage. As fibrotic lung contracts bronchial mucosa is thrown into transverse ridges, adding thereby, to the drainage difficulties. (37).

Schneider (cited by Miller) gives two phases of pathology. In every bronchiectatic lung with lesions, no matter how extensive, there can always be demonstrated adjacent bronchi in the purely atrophic state of bronchial dilatation. There can always be found bronchi showing the first phases of the infectious pathological lesions, and that these are found in the subepithelial layer of the bronchial wall under the intact mucous membrane, as well as over intact subjacent structures of the bronchial wall.

The sequence of events first shows a purely atrophic, cylindrical bronchiectasis, that is followed by an atrophic bronchiectasis with chronic inflammatory changes in the subepithelial layer, which lead to cellular infiltration of the bronchial wall which he terms hypertrophic bronchiectasis. The end result is an or-

ganization of this hyperplastic tissue into fibrous tissue developing what he call cirrhotic bronchiectasis. Late, two classes are described, those with elastic walls and other with rigid cavity walls.

These atrophic processes may be the remains of previous inflammation, perhaps dating back to childhood. In atrophic and hyperplastic stages, the adjacent and tributary lung areas are usually found in hyperinflation and hypoinflation. During the cirrhotic phase, inflammatory changes frequently invades the pulmonary parenchyma, and from them repeated bronchopneumonic processes may be found. Such processes may extend to the pleura often ending with permanent adhesions and induration of surrounding lung and pleural structures. It may falsely appear as though the process were reversed. Spread of the pathology from the center to the periphery seems to be the usual mechanism.

Boyd (16) in studying four pathological specimens, found that all four showed a greatly shrunken lobe with the upper lobes enlarged to fill the space ordinarily occupied by the diseased lobe. In three of these the only pathological basis for the triangular shadow was the collapse. In the fourth some fibrous adhesions from the hilus to the base may have been responsible. The cut surfaces showed saccular and cylindrical bron-

chial dilatation. The bronchial mucosa was thrown in transverse ridges. The parenchyma was reduced and nearly airless. All showed some fibrosis.

The microscopic study revealed no normal lung tissue. The alveoli were collapsed and thick-walled. There was some pigment in each case except one infant. The dilated bronchi had stratified columnar epithelium. Some areas had a lining of granulation tissue. Only a few islands of cartilage remained. Some bronchi had ciliated epithelium. A few had hypertrophied mucous glands.

Woodward (112) found an occasional pure growth of staphylococcus in early bronchiectasis from endobronchial material. He states that the staphylococcus toxin can cause a fragmentation and degeneration of muscular and elastic tissue with dilatation of the bronchi, due to the toxin's necrotizing action when combined with increased intrapulmonary pressure.

McCordock (72) demonstrated intranuclear inclusion bodies in the lungs in pertussis. He believes that the interstitial bronchopneumonia in such cases is due to a filterable virus with secondary bacterial infection. Destruction of the bronchial wall predisposes to bronchiectasis. Such changes are not observed in the ordinary bronchopneumonias in which bacteria alone are con-

cerned.

Robinson and Greey (51) took smears and cultures from lobes obtained at operation. In 8 cases they found nothing specific. Only 3 of this series showed spirochetes.

Ertmatinger (38) found that regardless of the predisposing cause most cases had Staphylococcus aureus, Streptococcus viridans and Micrococcus catarrhalis. She found Staphylococcus aureus in 75%. She states that no definite conclusions were possible in her studies.

DIAGNOSIS

The highest incidence of the disease occurs in the second and third decades with 70% of all cases occurring before 40. The symptoms had been present for 15 to 20 years in many (8).

In 83 of 149 cases, the disease was unilateral. It is essentially a disease of the lower lobe. If apical in location it suggests a chronic fibroid phthisis. The disease was bilateral in 66 of the 149 cases.

The most frequent and constant complaints were cough, expectoration, fever, hemoptysis and fetor.

a. Cough. It may be severe, hacking and persistent. Codeine and other drugs may fail to alleviate. Phrenicotomy has failed to provide relief in a few stubborn cases.

b. Expectoration. The sputum has no characteristic composition or appearance. It has much more water than tuberculous sputum. The amount may exceed 1500 cc. per day. The odor is due to the fatty acids, ammonium carbonates, sulphides, indol and other products of putrefaction and autolysis. Ferments are also present. There may be a 3-layered sputum. 1. is white or green-yellow. Is mixed with air bubbles, pus and mucous. 2. This layer is thinner, contains the same products

as the first layer with less air. 3. Contains pus cells, detritus, fat rests and Dittrich plugs. Lymphocytes do not predominate as in tuberculous sputa. Stones and pieces of cartilage have been found.

c. Hemoptysis. This finding is said to be more frequent in bronchiectasis than tuberculosis. It is said to be due to bleeding granulations. It may be the first sign to appear and the last to go. If the bleeding is severe, death may follow. It may be the only complaint.

d. Fever, sweats, chills. These are quite significant. They may suffer from repeated attacks of pneumonia. A pneumonitis with delayed healing will result in connective tissue proliferation. It becomes vascularized. Leucocytic infiltration occurs and effects the phagocytosis of fibrin. The process lasts for weeks and the red connective tissue finally becomes white. The connective tissue never disappears. The lung tissue so involved is no longer capable of function. Scar tissue around the bronchus pulls in all directions. Adherent pleura helps in the shrinkage process. The bronchi are filled with secretions and are veritable culture tubes. So fever may be due to a pneumonitis and not to retained secretions. This explains those cases which do not respond to adequate drainage (50).

e. Pain. This is not a striking feature and if present is most likely due to some other pathology.

f. Gastro-intestinal disturbances. If present and associated with the disease, it is probably due to amyloid degeneration. It is an ill omen for prognosis.

g. Skin eruptions. May be seen as a toxic dermatitis due to inadequate drainage.

h. Joint pain. The process causing this is the same as that which causes clubbing of the fingers.

An accurate diagnosis will include the location, size and distribution of the dilatations. In addition one should look for evidences of fibrosis (a shifted mediastinum), or atelectasis, tuberculosis, foreign body, abscess, bronchial stenosis, carcinoma, empyema, brain abscess and amyloid disease may be other concomitant processes which may point the way to diagnosis or offer differential diagnostic problems. Diagnosis is made on clinical history, physical examination, laboratory, x-Ray, fluoroscopy, bronchography, bronchoscopy and diagnostic pneumothorax.

A carefully taken and evaluated clinical history is said to comprise about 50% of the diagnosis. A careful elicitation of respiratory disease history is essential. Inquire about influenza, the exanthemata, the pneumonias and pertussis. Repeated upper respira-

tory infections may have significance. A history of an inspired foreign body will be extremely significant. Headache, as a part of the disease, may be due to brain abscess. Cyanosis and dyspnea may be due to a damaged myocardium. Urinary frequency may be attributed to amyloid disease.

The physical examination reveals no constant or typical findings. There may be signs of atelectasis and cavitation. There may be an increase in whispered voice or diminished breath sounds. Rales are nearly always present. Hypertrophic osteoarthropathy was first described by Bamberger (cited by Graham) in 1889. He thought that it was due to a venous congestion, a peripheral hyperemia due to increased resistance in the pulmonary capillary bed. The dilatations were thought to compress the capillary bed in the lung. He also admitted that a toxin might be the responsible agent.

Campbell (19) said that clubbing of the fingers was due to low oxygen tension in the arterial blood. The aerating surfaces of the lung are reduced. He noted that there was no obstruction to venous return from an extremity.

Chernyk (20) states that clubbing of the fingers is probably more pronounced in bronchiectasis than any

other chronic pulmonary disease.

Diagnostic pneumothorax is not used now since lipiodol has been introduced but it is a good method to bring out atelectatic bronchiectatic lobes which may be hidden by the heart.

Singer and Graham (98) in a Roentgen-ray study of bronchiectasis found a triangular shaped area of density from the hilum to the diaphragm in the bases of the lungs. The shadows at operation were found to be produced by atelectatic bronchiectatic lower lobes. Broncho-pneumonic areas in the opposite lung were thought to be due to "spill over" from the diseased side.

Roentgen ray findings usually reveals an increased hilus detail which extends to base; and "circular" shadows are suggestive. Fluoroscopy will give information concerning the condition of the diaphragm. Expiratory dyspnea with the diaphragm low is caused by edema of the mucous membranes in the terminal bronchioles, and secretions retained in them. This causes an alveolar collapse.

Warner (107) states that the x-Ray shows whorl-like shadows from apex to base with the mediastinum shifted to the affected side in atelectatic bronchiectasis. Parenchymal changes may be fibrotic, secondary to infection in the alveoli. 6% of all bronchiectatic

tasis show triangular basal shadow at the cardio-diaphragmatic angle, and when seen is practically diagnostic of bronchiectasis.

Boyd (16) describes a basilar triangular shadow as a homogeneous opaque shadow in the form of a right angled triangle having for its base the diaphragm, one side the mediastinum, and a hypotenuse formed by a line extending from the hilus to a point somewhere on the diaphragm. The latter may be straight, concave or convex, or slightly irregular in its outline. If the shadow is due to effusion, the hypotenuse is usually convex. The shadow occurs most often in bronchiectasis but may be due to pleural effusion or congenital heart disease.

The only constant and significant physical signs is cardiac displacement to the diseased side. It may be sufficient in some cases to suggest lobar collapse. There may be some dullness and altered breath sounds but they are not sufficiently characteristic to differentiate bronchiectasis from other lower lobe lesions.

12 out of 14 children with basal shadows had bronchiectasis. There was a morbidity of 7% in the cases studied. The shadows were permanent in all but one case. Reinflation of the lung with the use of CO₂ to effect forced breathing was not possible in one case.

Anspach (6) in a roentgenological study of fifty cases of bronchiectasis made this observation:

In early roentgenograms of more than fifty children with proved bronchial dilatations, this shadow was found to be present within a few hours or days after the onset of the first symptoms.

In a study of the histories of these cases an acute pulmonary process was noted at the onset.

A temperature of 103 to 105 degrees F. was often preceded by a chill. Decreased breath sounds were heard at the bases, along with moist rales. The temperature would drop in 48 hours. There would be frequently a hacking cough followed later by expectoration. The triangular basal shadow appears early. The bronchi dilate early. Infants in whom the triangular basal shadow was constantly present in the first year of life invariably died.

If the shadow was small and of a fluid-like density and remained so, bronchiectasis developed rapidly. If the shadow disappeared and recurred the dilatations were slow and not prone to progress. Constant postural drainage may cause a decrease in the shadow and an improvement in the clinical picture.

There is always present some cardiac and mediastinal displacement. Adjacent lobe or lobes were found

to pass nearly completely around the collapsed lobe, anteriorly, laterally and posteriorly. No adhesions or exudate were found.

Bronchography reveals the presence of dilatations. This technique will show changes in the dilated portions during inspiration and expiration.

Jacobaeus (59) used lipiodol in bronchography and he thought that he could distinguish two phases in expectoration. First was an expression of the emptying capacity of the bronchi and second, corresponds with the emptying time of the finest bronchioles and alveoli. The second phase was much slower and incomplete. The primary phase lasts from 4 to 8 hours followed by a transitional period of eight to twelve hours during which time globules of lipiodol were still coughed up. The primary phase may be absent. The second stages may show streaks of the medium in the sputum for several days. Expectoration time in normal cases was four to eight hours. Bronchi filled with lipiodol may remain unchanged for several days yet there will be abundant expectoration of sputum. One can not conclude that the lipiodol containing bronchi are the source of sputum. Lipiodol may merely coat a bronchus and sputum comes through.

Bronchoscopy is valuable as a diagnostic aid be-

cause:

1. It reveals foreign body, new growths or bronchostenosis.
2. Permits the observation of the bronchus discharging pus.
3. It is a good method to instill lipiodol. Also removes secretions and granulations.
4. Can obtain uncontaminated cultures from the bronchi.

Bonner (15) advises the use of the bronchoscope in diagnosis of bronchiectasis, and feels that the bronchoscope is an important adjunct in the diagnosis.

Christie (21) explains well the technique of lipiodol instillation into the bronchi for diagnosis. Lipiodol is a compound of iodine and poppy seed oil, which contains 40% iodine by weight. His technique which is essentially the same as first described by Sicard and Forestier (97) first in 1922 is as follows:

A 2% cocaine spray is used on the anterior pillars of the fauces, the vestibule of the larynx and the tracheo-bronchial mucosa. The patient inhales the cocaine spray at five minute intervals for thirty minutes. This abolishes (1) Gagging, (2) The swallowing reflex, (3) The cough reflex. With a curved cannula warm oil is dropped at the base of the tongue. The patient breathes deeply as the oil is introduced. About 10 cc. is used.

The patient must be tranquil and cooperative. Anesthesia must be sufficient to abolish swallowing and cough reflexes. The oil should be injected slowly. Hold the tongue forward and incline the patient to the side the oil is to fill. This is called the "passive" method of instillation. The oil may also be introduced intratracheally, with a catheter in the nose, and by use of the bronchoscope. It may be also injected through the cricothyroid membrane into the trachea (21).

Morse (78) in an interesting study of 42 cases found the following symptoms prevailing:

100% had cough.

57% had pulmonary bleeding at some time.

16% had night sweats.

66% complained of weakness.

46% had dyspnea.

69% had fever. (Only 33% had fever in the hospital).

28% had repeated attacks of pneumonia.

86% show upper respiratory disease---colds, chronic tonsillitis, carious teeth, and x-Ray evidence of paranasal sinusitis.

28% showed clubbing of the fingers.

Bronchograms revealed unilateral disease in 26 and bilateral in 16. 40 of the 42 had lower lobe involvement.

TREATMENT

Most forms of treatment merely deal with symptoms. Treatment can be divided into two types, operative and non-operative.

Non-operative measures which have been employed from time to time may be listed as follows:

1. Bed rest. This has only a temporary effect and the underlying pathology remains the same.
2. Diet. No diet yet devised possesses any curative merits. A nutritious, easily assimilable diet with a high vitamine content is considered indispensable in management of these cases.
3. Climate. This offers nothing specific or particularly helpful. One should merely avoid irritating gases and air that is heavily laden with dust.
4. Postural drainage probably is the most efficacious and respectable form of non-operative treatment. It should be done daily and continuously. This mode of therapy may suffice in mild cases. It should always be given a fair trial. It may be supplemented with profit by bronchoscopic aspirations. The use of an expectorant, potassium iodide, has been recommended. This was followed by use of an emetic; this is purported to aid in draining the bronchi by compressing them.
5. The thirst cure may be mentioned only to em-

phasize its worthlessness.

6. Heliotherapy likewise has no distinct effect.

7. When the disease process seems to be caused by the fusiform bacillus and spirochetes, intravenous use of neocarsphenamine may abate the symptoms but will not cure the disease. The dilatations persist.

8. The direct intrabronchial application of lipiodol by syringe or spray has been claimed by some to be beneficial. Lipiodol has no definite bactericidal effect and probably no therapeutic effect. There is some absorption of iodine from lipiodol when instilled into the trachea. Iodine concentration in the blood rises following intrabronchial instillation of lipiodol. The lipiodol may act mechanically by displacing secretions. Some patients treated with lipiodol instillations claim marked amelioration of symptoms. (8).

9. Bronchial irrigation or lavage was at one time recommended by a few workers who claimed good results. It is now known that bronchoscopy will accomplish the same results and is much easier to use. One series claimed a 35% symptomatic cure in a 100 cases. (41).

10. Vaccine therapy, although highly praised by some does not seem to have a definite place in treatment. It may occasionally help when other systemic reactions occur such as arthritis(9).

Moore and Love (77) made a study of polyvalent staphylococcus bacteriophage as used in conjunction with bronchoscopy for the treatment of bronchiectasis. They first tested the potency of the bacteriophage used. A potent phage was then instilled into the bronchi following bronchoscopic aspiration. They concluded that it was of value in certain cases. They were able to effect a change in the character and quantity of the secretions.

Woodward (112) thinks that a staphylococcus toxoid is a good adjunct to treatment. He bases this on finding the staphylococcus in pure culture from endobronchial material and in cultures from the sphenoidal region.

Balyeat (10) feels that intratracheal instillation of iodized oil does offer some palliative relief. Poor results are obtained when bronchiectatic areas were communicating with a large bronchus. It acts merely in a mechanical manner by lubricating tubes and floating up secretions and pus. It also facilitates drainage.

Clerf (24) stresses the importance of providing adequate drainage. If obstructive in type the obstruction should be removed. He thinks associated pathology in the accessory paranasal sinuses which is a com-

mon concomitant of bronchiectasis, should be treated likewise. It has been suggested by many that the two foci act deleteriously, one on the other, so it is essential to combat the two simultaneously. Postural drainage should always be tried. Bronchial irrigations have proven beneficial in some hands.

Sippe (99) claims good results in nine cases by use of dextrose therapy. The rationale of his treatment was based on his assumption that hypoglycemia and ketosis are factors in production of bronchiectasis.

Berck and Harris (12) proposed the following rationale as their basis for x-Ray therapy:

The x-Ray acts on leucocytic and lymphocytic infiltration causing a destruction of these extraordinarily sensitive cells with ensuing phagocytosis and connective tissue proliferation.

They did not treat those cases with acute infections or those which had exacerbations. An exact pre-therapeutic diagnosis was made by bronchoscopy and bronchography. No other type of treatment was used during an x-Ray course. They were treated as ambulatory out-patients. Large doses from anterior, lateral, and posterior aspects were used, obtaining the "cross-fire" effect. Each treatment gave 75 Roentgens to 2 or 3 fields. Treatments were done 2 or 3 times per

week for 3 months.

During the course the temperature may go up and there may be an exacerbation of symptoms, sometimes severe. Improvement was noted when the course was about three-fourths completed. Criteria for improvement were lessened cough and sputum, and subjective signs of feeling well. The tabulated results in a series of 30 cases are as follows.

Group I Chronic Bronchiectasis

Group II Odorless Bronchiectasis

Group III Foul Bronchiectasis

I	II	III	
7	1	7	Greatly improved
2	1	1	Moderately improved
4	1	3	No improvement
1	0	2	Deaths

Clubbing of the fingers was noted to improve markedly in a good many cases. This therapy merely changes the "wet" type to the "dry". There may or may not be a concomitant shrinking of the bronchial tubes.

Jackson (60) states that the bronchoscope is the most useful in bronchiectasis. It has its chief indication here.

Bonner (15) recommends ameliorative treatment

by the use of the bronchoscope.

Jackson (60) states that the chief object of bronchoscopic treatment is to promote drainage. Postural drainage is a good procedure but never is as thorough as bronchoscopic drainage. Neither postural nor bronchoscopic drainage can cure dilatations but they may alleviate symptoms. The value of bronchoscopy lies chiefly in removing or treating local causes such foreign bodies, bronchial stones, dilating strictures, and prevent progression by spilling over of secretions into unaffected bronchi.

Martin (68) reported 15 clinical cures in 61 bronchiectatics by use of the bronchoscope.

Oleoathorax achieves about the same results as pneumothorax. The collapse is more easily maintained for longer periods. This gives symptomatic relief only. Its position of worth is rather indeterminate. It may cause some dyspnea (50).

Morse (78) states that partial pneumothorax in hemorrhage may be life saving. As a curative measure collapse is not good therapy because the cavities have epithelial linings and consequently will not fibrose and heal.

Ballou, Singer and Graham (50) state that few patients with true bronchiectasis respond to pneumothorax

treatment even though collapse may be almost complete. This method compresses everything but the dilatations. Pneumothorax may change the position of the lobe so that the dilatations become more horizontal instead vertical. This treatment rarely does harm. If improvement does not take place in 3 to 6 months, then it is not likely to occur. Expectoration is rarely completely abolished by pneumothorax. It may be a good pre-operative measure so that the mediastinum may become stabilized. They conclude that according to their own results pneumothorax in treating bronchiectasis can not be recommended as a very useful procedure.

It would seem that certain acute inflammatory conditions of the lung which are likely to become chronic and lead to the development of bronchiectasis, might, with profit, be prophylactically treated by pneumothorax.

Cure can only be considered when the diseased portion of the lung has been removed or destroyed (50).

Clerf (25) states that lobectomy is the only sure means of cure.

Mason (69) reports four typical cases of bronchiectasis completely cured by pneumonectomy. He concludes that the operation is beneficial and justifiable.

Churchill (22) reports the following results in

lobectomy and pneumonectomy in bronchiectasis:

Mortality of 6.1% in 49 patients subjected to lobectomy and pneumonectomy.

5.0% in 40 lobectomies.

2.6% in 38 lobectomies under now recommended procedure.

Results:

30 cured.

5 improved.

5 multi-lobar, were improved; had a reduction in amount of sputum.

3 operative deaths.

Hedblom (54) did thoracoplasties on 32 cases and 21 were still alive several years after operation. Most were considerably improved. He claims the procedure reduces negative intrapleural pressure and reduces the formation of sacculations. The type of patient who has septic manifestations is not amenable to thoracoplasty. It is not a valuable procedure considering the difficulty and the risk.

Pneumonotomy has been more or less discarded. It is almost impossible to incise all dilataions. The procedure may be justified in a single bronchiectatic abscess. Mortality rates in pneumonotomy vary from 23% to 73%. Good results obtained vary from 9.4% to

33% in different series.

One can hardly expect much from pneumotomy unless when dealing with a single bronchiectatic abscess. A satisfactory result is seldom obtained because of associated bronchiectasis for which simple drainage does not suffice.

Coquelet (28) employs a surgical procedure known as "cuneo-pneumonectomie" in which he removes a wedge of lung tissue in the diseased area. In a series of six cases he cured three without residual fistulas. One patient was cured of the suppurative aspect of the disease but still had an open cavity. The fifth case was still under treatment and the sixth died of massive bronchopneumonia of the opposite side.

Lewis (64) performed a successful bilateral lobectomy for bronchiectasis and mentions the following indications:

There must be either single lower lobe involvement or both lower lobes, or lower and middle lobes on the right. Careful pre-operative visualization with lipiodol of both right and left bronchial trees is essential. Spinal anesthesia is to be preferred, since the positive pressure type tends to disseminate pus and particulate matter through the finer radicles of the bronchial tree.

bronchial tree.

Bohrer (14) performed lobectomies in four cases with bronchiectasis. They were cured or greatly improved. He advises early operation on the diagnosed cases for the following pertinent reasons:

1. Children withstand the operation well; better than adults.
2. The tissues in the young are more elastic than in the adult.
3. The resulting anatomical deformity is reduced when the operation is done early in the young.
4. Many die before reaching adult life.
5. The disease processs may extend to other/lobes.
6. By effecting a cure the patient is returned to society a useful member.

Shenstone (95) reported 16 lobectomies. His results had three death, six cures, 2 were improved and 3 were unimproved or worse.

Brunn (17) advises a preliminary phrenicotomy. In 6 of his cases there were four cures, one improvement and one death.

Coryllos (30) reported 7 cases of pneumonectomy for bronchiectasis with 2 deaths. Death came from pneumonia in one and cerebral embolism in the other. Six of these cases had bronchial fistulas which per-

sisted from 4 to 6 months.

The most successful cases of lobectomy are performed on children and young adults. The operation seems to have a definite place although it still has a high mortality. Bilateral lobectomy in extensive bilateral disease is not advised nor indicated.

Of 212 collected cases, 34% have died apparently from the operation of lobectomy. 47% had satisfactory results. The operation offers a 15 to 20% chance of dying and only a 65% chance of becoming cured (50).

Lobectomy was attempted early in this century as a cure for bronchiectasis. It was abandoned as an impracticable procedure because of the excessively high mortality. Until 1914 only sixteen pneumonectomies had been reported as a treatment for bronchiectasis. There were eight deaths. Lillenthal (66) in a series of forty-two patients in which sub-total and total resections were used, reported a mortality rate of 64.3%. Fifteen cases recovered and are well. Four had remaining bronchial fistulas after operation. Lillenthal (66) advised a two-stage operation, the first aimed merely to establish firm adhesions between the lung and pleura, and the second stage was for removal of the lobe.

Some operators favor the method of first exteriorizing the affected lobe and then later doing the resection.

Cautery pneumonectomy as described and employed by Graham (50) seems to be a worthwhile procedure. Indications include multiple lung abscesses and chronic lung abscesses with secondary bronchiectasis. These types respond best to this form of treatment. Thoracoplasty and pneumonotomy are not amenable because of the septic condition which leads to a high mortality rate. It is not a good measure for congenital and wide dilatations of the lower lobes.

Cautery pneumonectomy is recommended to those suffering from unilateral types of bronchiectasis who have not responded well to simpler forms of therapy, for whom thoracoplasty is either not indicated for various reasons or has failed to relieve the symptoms, and upon whom the performance of a lobectomy, although desirable, is unwise or technically impossible.

Technique of cautery pneumonectomy combines drainage, compression and extirpation. Exposure is made by resection of 2 or 3 ribs. The lung is made firmly adherent to the pleura. In the second stage an excavation is made into the lung tissue with a red hot soldering iron. Follow draining sinuses eccentrically. In bronchiectasis an effort is made to expose a large cross-section of the bronchial tree. One may establish 10 or 15 bronchial fistulae from each of which pus exudes.

Then wait for several weeks before repetition of the process. This establishes good bronchial drainage. Hemorrhage can be controlled by packs of gauze.

Cough and sputum may disappear in 24 to 48 hours. On the 3rd post-operative day the patient develops a little fever, malaise, loss of appetite and sometimes nausea and vomiting. These symptoms are probably due to intoxication from the burned lung tissue. A slough separates on the 10th to the 12th day. Toxic symptoms may increase until the slough separates, and then there is a gradual return to normal as bronchioles begin discharging.

Graham's results in 20 cases in 1925 were:

30 % were symptom free and healed in 6 months. 30% were symptom free but had fistulas. 15% were markedly improved. Some progress noted in 15%. There was a 20% mortality.

In 1929 in a series of 54 cases Graham reduced his operative mortality to 11%. 66% had definite improvement but 47% had persistent bronchial fistulas. These fistulas may persist for several months and cause little or no inconvenience. They do not predispose to respiratory infections and can be closed by cautery and plastic surgery.

PROGNOSIS

The duration of the disease does not influence prognosis in every case. The age at which dilatations develop is not always significant. Early removal of a foreign body may arrest the process before irreparable damage has been done. A patient may carry bronchial dilatations for years.

If the trachea is in the midline and there is little or no fever and no atelectasis, then the outcome is not nearly so bad as when the dilatations are associated with atelectasis and when there is fever. We are more often concerned with parenchymal and peribronchial changes than we are with the dilatations.

One may occasionally see saccular type dilatations in one lobe and atelectatic type in the opposite lower lobe. There can be little doubt that the manifestations of sepsis are then due to the acquired bronchiectasis since it is easy for the patient to empty the saccular dilatations. It is also easy to fill such dilatations with lipiodol. In addition, the sum total of diameters of such dilatation usually indicates that there can be little pulmonary parenchyma in such a lung and consequently little room for parenchymal involvement, for pneumonia and abscess formation.

The most unfavorable type in children is that in

which the mediastinum is displaced to the affected side. If this is associated with much fibrosis and progressive displacement of mediastinal contents, the process usually ends fatally in a few years. The cause of death is usually pneumonia, multiple lung abscesses or both (50).

Many children are treated for unresolved pneumonia. The underlying disease, however, is usually empyema, multiple abscesses or bronchiectasis. The course is usually down hill when the trachea and mediastinal contents are shifted to the affected side.

A midline trachea and the absence of visible dilatations does not permit one to conclude that the changes in the parenchyma are not striking or that they will not progress. It is essential to have good films to follow the progress of the disease. Both lungs should be injected with the opaque material. Physical examination and ordinary x-Ray may show less on the side most involved, particularly if the area is obscured by the heart shadow. Mediastinal thickening and dextrocardia should always suggest the possibility of bronchiectasis in children. Early treatment of a sinusitis may be extremely beneficial (50).

In adults the disease may run fifteen years or more. Purely bronchial involvement produces only cough

and expectoration, while chills and fever usually denotes parenchymal involvement. The dry form may persist for years and the only symptom be hemoptysis. The outlook is grave if there are multiple peribronchial abscesses.

Royles and Todd (93) state that those cases managed under a medical regime show a mortality of 51% while those treated surgically have a mortality rate of 30%. They conclude that lobectomy is the best treatment.

Anspach (6) stated that infants in whom a triangular basal shadow in the lungs was constantly present by x-Ray in the first year of life, invariably died.

Clerf (24) says that prognosis in bilateral bronchiectasis is always unfavorable, regardless of associated findings.

Chernyk (20) in regard to prognosis says that the untreated or neglected cases can carry on for many years, progressing downwards until secondary degeneration occurs in other vital organs of the body and lead towards a hopelessly incurable condition with a fatal prognosis.

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