The Problem of Bronchiectasis*
A Review

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Bronchiectasis, like pulmonary tuberculosis, is a killer of men; but bronchiectasis, unlike tuberculosis, kills on a fairly definite schedule. Tuberculosis, unless in a terminal state, tends to heal if given a chance; and we see many people who once had far advanced active pulmonary tuberculosis, who have regained a measure of health and economic independence; but we see very few bronchiectatic patients with far advanced disease who are ever able to do more than live a miserable, dependent existence; the tendency of bronchiectasis is to progress downward. Head¹ found, in a group of two hundred bronchiectatic patients, who had acquired their disease in the first ten years of life, that few were living after forty years of age. Riggins,² from observations at the Bellevue and Lennox Hill Hospitals, New York City, agreed with Head for the most part; but also saw a fair sprinkling of bronchiectatic patients in their sixth or seventh decades. While Roles and Todd³ observed a forty-seven per cent mortality in forty-nine non-surgically treated patients that were followed for six years. Clagett and Deterling, Jr.,⁴ stated that some bronchiectasis was found in two per cent of all the necropsied cases at the Mayo Clinic.

The bronchiectasis found in the right upper lobe and in the upper portion of the left upper lobe due to atelectasis, bronchial stenosis, and contracting scar tissue and following various inflammatory conditions of the lung (including tuberculosis), is relatively benign because the drainage is downhill; the drainage of bronchiectatic lesions in the lower lobes, right middle lobe, and lingula of the left upper lobe is uphill, and more liable to pursue a progressive course. Also, there may be a more or less temporary bronchiectasis following acute respiratory infections as bronchitis, virus pneumonia, and which follows enlarged hilar glands as from primary phase tuberculosis; in these cases the bronchiectasis follows bronchial stenosis with its tendency to trap inspired air and prevent its egress, combined with areas of atelectasis which increase the negative pull of the pleural space; this type of bronchiectasis has a tendency toward recovery when the

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causative conditions are removed, provided the bronchial walls do not become grossly infected.

Bronchiectasis, as we ordinarily understand it, is an acquired dilatation of the bronchi and bronchioles and the primary lobules or lung units of the pulmonary parenchyma. But before going further into the etiology, pathogenesis, pathology, and treatment of bronchiectasis, it seems advisable to briefly review the developmental and final anatomy of the tracheo-bronchial tree and lungs in order that these phases may be better understood.

The anlage of the larynx, trachea, bronchi, and lungs arises about the fifteenth day (embryo of 23 segments; 3.2 mm. in length) from an outgrowth in the ventral wall of the entodermal tube (the foregut; the primitive oesophagus). This anlage is known as the laryngotracheal groove. The caudal end of this outgrowth promptly becomes rounded and marks the future development of the bronchi and lungs; constriction of the groove proceeds cephalad and it becomes separated from the primitive oesophagus, the cephalic portion becoming the larynx and the intermediate part becoming the trachea. The rounded, caudal portion, the lung bud, becomes bilobed in four to five millimeter embryos. Both lung buds elongate, the right being slightly larger than the left at first. These lung buds (stem buds, main bronchial stems) grow downward, lateralward and backward, branching monopodially to give rise to the future bronchi. In the later stages of development there is probably dichotomous branching of the smaller bronchi.

In the seven millimeter embryo, from the stem bronchi, two bronchial buds arise on the right side (the apical bud, future right upper lobe bronchus; and a ventral bud, the future right middle lobe bronchus); a ventral bud also arises on the left bronchus (the future left upper lobe bronchus); the main bronchial stems or buds become the lower lobe bronchi. These future bronchi grow into a mass of mesenchymal cells and carrying before them folds of mesoderm. The mesenchyme forms the cartilage plates, muscle, connective tissue of the lungs, and tracheal and bronchial walls. Blood vessels and nerves grow into this developing mass. The visceral (splanchnic) mesoderm forms the visceral pleura, and the somatic (parietal) mesoderm forms the parietal pleura. The entoderm of the primitive trachea and bronchi forms the epithelium lining of these and the lining of the lung units. The air cells or alveoli begin to form at about the sixth month; and the lung is fully formed when birth takes place.

Macklin considers the tracheo-bronchial tree as far as the respiratory bronchiole, as merely a conduit system; while the
part beginning with the respiratory bronchiole and ending with the pulmonary alveoli is the functional part. The bronchi, when they enter the lungs, become cylindrical and gradually acquire a circular layer of smooth muscle on the outer border of the tunica propria. These muscle fibers become more and more prominent as the smaller bronchi are reached, and are found to extend as far as the atria. The other elements of the bronchial wall (cartilage, white fibrous tissue, elastic tissue, glands, etc.) decrease as the smaller bronchi are reached. The circular muscle is best developed in the terminal bronchiole, and can exert a strong sphincteric action in bronchospasm; as the bronchi grow smaller, the cartilagenous rings change to small plates or flakes, and disappear in bronchi of about one millimeter diameter. The mucus glands also disappear about here (Fig. 1).

Stratified, ciliated, columnar epithelium lines the trachea and larger bronchi; as the bronchi grow smaller, this becomes simple, columnar, ciliated epithelium; then ciliated cuboidal epithelium; when the respiratory bronchiole is reached, the epithelium becomes simple cuboidal with areas of squamous or respiratory epithelium. The remaining portion of the tract (alveoli, air cells) are lined with squamous epithelium; probably deficient in places (Fig. 2).

Diagram of origin of lungs and bronchi.
1. Entodermal tube (fore-gut); 2. primitive trachea; 3. lung bud from ventral aspect of entodermal tube; 4. mesenchyme; 5. mesothelium (visceral), future visceral pleura; 6. primitive esophagus from fore-gut; 7. ventral bud (future left upper lobe bronchus); 8. stem buds (main bronchial stems); 9. ventral bud (future middle lobe bronchus); 10. apical bud (future right upper lobe bronchus). On left side, apical bud in ventral bud; no separate lobe on left side comparable to right upper lobe; 11. septum transversum (future diaphragm).
Miller's lung unit begins with the respiratory bronchiole (bronchiolus respiratorius) which gives off several alveolar ducts (ductuli alveolares; alveolar passages; vestibules); each alveolar duct gives rise to several irregularly spherical dilated parts, the atria; each atrium originates several alveolar sacs (sacculi alveolares; infundibula; air sacs); and in each alveolar sac wall, there arise a number of alveoli (alveoli pulmonis; air cells). Best and Taylor's description of a lung unit or primary lobule begins with the respiratory bronchiole, which is a continuation of the terminal bronchiole, and has the same length (0.2 - 0.5 mm.) and diameter (0.3 - 0.4 mm.); five or six alveolar ducts arise from one respiratory bronchiole; and an alveolar duct gives origin to three to six alveolar sacs after a variable number of rebranchings; an alveolar sac contains a number of small pouches, the alveoli. As the bronchioles approach the periphery of the lung, they grow shorter, but maintain about the same diameter as the earlier ones (0.3 - 0.4 mm.), the first branches being about 1.5 millimeters in length.

**Diagram of cross section of medium-sized, intrapulmonary bronchus.**

1. Stratified, ciliated, columnar epithelium; 2. basement membrane (membrana propria); 3. goblet, mucus cell; 4. outer layer of tunica propria, shows longitudinal elastic fibers cut across, and white fibrous tissue, inner layer shows fine, loose fibers and basement membrane; 5. racemose mucus and serous gland opening on mucus surface and extending into adventitia; 6. mucus membrane; 7. tunica propria (cortum); 8. lymph gland; 9, 10, 12, blood vessels. All layers contain blood vessels, nerves, lymphatics and secreting glands; 11. hyaline cartilage plate in dense fibro-elastic layer; 13. fat cells; 14. submucosa of coarse, loose areolar tissue with glands, vessels, nerves and lymphatics; 15. adventitia of loose areolar tissue with glands, vessels, nerves and lymphatics; 16. fibro-elastic layer of white fibrous tissue and yellow elastic fibers densely arranged; 17. circular, smooth muscle layer in mucus membrane.
The essential etiology and pathogenesis of bronchiectasis can be summed up in a few words: Stenosis of a bronchus with retained infective secretions permit infection of the bronchial walls, with weakening of such stromal elements as elastic tissue and muscle, and permit the rythmical pull of inspiration, possibly combined with the weight of the pooled secretions, gradually to dilate the walls of the bronchi and lung units into permanent or potential cavities. When there is atelectasis of portions of the surrounding lung; or when there is contracting, inelastic, fibrous tissue in the surrounding lung, this inspiratory pull is greatly increased; when there are also partly occluded bronchi which permit air to enter, but which exert Chevalier Jackson's well known check-valve action in preventing egress of some of the air, the dilating effect on the infection-weakened walls is heightened.

The bronchiectasis, mainly in the upper lobes, caused by the irregularly developing fibrosis of chronic tuberculosis produces dilatation in the distorted, gnarled bronchi which does not present the regular pattern seen in ordinary lower lobe bronchiectasis with its more evenly developing fibrosis. Even so, this upper lobe bronchiectasis of tuberculous fibrosis is usually relatively benign because the drainage is downhill; but the bronchiectasis that follows invasion of the bronchial walls by tubercle bacilli, with possible secondary infection by ordinary pyogenic bacteria, is one of the severer complications of pulmonary tuberculosis, and may require surgical removal.

Bronchiectasis can develop at any age; but a large percentage of cases develop during the first ten years of life. Perry and King claimed that the onset of 42 per cent of their patients was during the first ten years, and that the onset occurred in 27 per cent during the second ten years. Farrell stated that 80 per cent of his bronchiectatic patients acquired their disease during the first ten years.

Singer and Graham pointed out that the dense, triangular shadows seen along the mediastinal borders with bases on the diaphragmatic leaflets, apices in the hill, and hypotenueses facing peripherally were due to atelectasis of lower lobes. These are seen principally in infants and young children. There are similar, but larger and less dense triangular shadows which are due to interstitial infiltration and fibrosis in bronchiectasis.

McNeill, MacGregor and Alexander, Richards, and Anspach showed that these triangular areas of atelectasis frequently accompanying pneumonia, were often followed by bronchiectasis later, especially in infants and children. The smaller bronchi of infants and children are especially susceptible to plugging, from thick, tenacious secretions with attendant atelectasis. It is a law
of the cube that the smaller bronchi would present a relatively greater surface for adherence per unit of volume than the larger bronchi.

Anything which causes bronchial stenosis predisposes to bronchiectasis. The inflamed bronchi of whooping cough, measles, bronchopneumonia, and bronchitis, are special sources in childhood; enlarged tracheobronchial and hilar glands following acute and chronic infections, and tuberculous adenitis of these glands may cause stenosis of the bronchi with development of bronchiectasis. The inhalation of foreign bodies with the drowned lung beyond the obstruction are frequent sources of bronchiectasis, particularly in children; and as Chevalier Jackson pointed out long ago, the vegetable body as the peanut is more liable to encourage bronchial and pulmonary suppuration than the metallic one.

There is a form of chronic bronchitis and bronchiectasis occurring in children having cystic fibrosis of the pancreas. The lack of exocrine secretion of the pancreas with failure to digest fats and other nutritional constituents causes lack of absorption of vitamins A and D and malnutrition; these in turn permit infection of the respiratory tract with pyogenic organisms, espec-

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Diagram depicting William S. Miller's primary lobule or lung unit.

1. atria; 2, alveolar sacs (infundibula); 3, alveolar ducts (vestibuli); 4, respiratory bronchiole; 5, terminal bronchiole; 6, alveoli (air-cells); 7, pulmonary artery accompanying terminal bronchiole, and giving a branch to each atrium; then forming capillary plexuses around alveoli; 8, pulmonary venule collecting from plexuses; then following partly independent course back to heart, but joining other venules.
ially staphylococci; as with other vitamine A deficiencies, there is a piling up of epithelial debris in the salivary gland ducts, trachea and bronchi.

Kartagener\textsuperscript{20} found bronchiectasis and sinusitis frequently associated with dextrocardia and situs inversus, the "Kartagener triad." He cited this in support of the theory that bronchiectasis is congenital. Adams and Churchill\textsuperscript{21} reported five cases of this syndrome. Olsen\textsuperscript{22} reviewed 85 cases of dextrocardia seen at the Mayo Clinic from 1920 to 1941, inclusive; evidence of bronchiectasis was found in 16.5 per cent of these cases in contrast to less than 1 per cent of bronchiectasis in all other patients registered there. He also mentioned bronchiectasis occurring in pairs of identical twins as further evidence for the congenital theory of bronchiectasis.

Any condition of the lungs and bronchi that causes fibrosis or bronchial stenosis or both as pneumoconoisis, corrosive gases, fungus infections, bronchopneumonia, bronchitis, pleural empyema, lung abscess and the like, may be causative of bronchiectasis. Benign or malignant intrabronchial tumors; extrabronchial tumors; large glands, or fibrotic bands pressing on the bronchial walls all encourage the development of bronchiectasis. Uncomplicated pneumococcus lobar pneumonia rarely causes bronchiectasis, because the involvement here is primarily parenchymal and not bronchial. But the virus types of pneumonia are frequently followed by the development of bronchiectasis. Here, there is an intense inflammatory involvement of the bronchial and peribronchial tissues as well as an alveolar exudate and hemorrhagic pneumonitis. The partly occluded bronchi with their damaged walls and retained infective secretions, the scattered areas of atelectasis all favor the invasion of secondary bacterial pathogens into the bronchial walls and surrounding tissues.

Watson and Kibler\textsuperscript{23} reiterated an earlier theory of theirs that allergy of the bronchial tubes with its attendant bronchial stenosis and retention of secretions was the most frequent cause of bronchiectasis. It is true that the bronchial swelling and muscle spasm of allergy furnish a good foundation for the development of bronchiectasis; but is not the only mechanism that does so.

The pathological changes in bronchiectasis are varied. The invasion of the bronchial walls and surrounding lung tissue by the pathogenic bacteria and defence white cells of the blood is followed by varying degrees of destruction and replacement by inelastic, white fibrous scar tissue. The scar tissue, when young and gelatinous, is easily stretched by inspiration and the weight of retained secretions; but becomes hard and contracted when older. There is loss of the elastic elements in the bronchial walls.
The ciliated, columnar epithelium may be eroded in places, leaving granulation or scar tissue in its place; or it may be replaced by flattened epithelium devoid of a ciliary fringe with its self cleaning action. Blocked, infected secretions may extend into the lung, producing lung abscess or pleural empyema.

After each bout with an acute respiratory infection, the chronic bacterial infection spreads more extensively into the bronchial walls and lung parenchyma, leaving more destruction and scar tissue formation. A greater and greater load is added to the right side of the heart by the mechanical obstruction to blood flow and the replacement of functional units in the lung. This recurs until the right ventricle fails and eventually congestive heart failure develops. This heart failure responds to cardiac treatment for a time; but if the patient does not die subsequently of metastatic abscess, pneumonia amyloid disease, nephritis, or massive hemorrhage, he will eventually die of heart failure from the chronic overwork, and infectious or toxic myocarditis from the pulmonary infection.

The left lower lobe is the one most frequently involved in bronchiectasis; and when bronchiectasis develops here, the lingular segment of the left upper lobe frequently becomes affected. Churchill and Belsey found the lingular segment sufficiently involved to require surgical removal in eighty per cent of cases requiring removal of the left lower lobe. This frequent left sided localization is probably mainly a matter of drainage; the left main bronchus and its branches make more of an angle with the trachea than does the right one, which is more of a continuation of the trachea. Clagett and Deterling found the following lobar distribution in 471 bronchiectatic patients seen at the Mayo Clinic: the right lower lobe in 19 per cent; the left lower lobe in 35 per cent; both lower lobes in 19 per cent; the right middle lobe seldom. While these statistics agree fairly with those of others, there are a considerable number of patients who have right middle lobe bronchiectasis accompanying bronchiectasis of the right lower lobe.

There is no micro-organism distinctive of bronchiectasis; but streptococci (including viridans or alpha type, hemolyticus or beta type, non hemolyticus or gamma type and probably some anaerobic types), seem to play an especially prominent role in bronchiectasis. The pathogenic significance of streptococcus viridans is not yet settled. Monilia albicans is found occasionally as a secondary saprophytic invader, but may possibly at times assume pathogenic importance. The fusiform-spirilla group is only rarely found in bronchiectasis; and then mainly as a saprophytic invader. The bad odor found in the sputum of some bronchiectatic patients
is now thought to be rarely caused by this group. This odor is now attributed by many, to other organisms, as the anerobic streptococcus. The Neisseria catarrhalis is considered by many to play little or no part in the disease process.

In some quarters, there is a marked swing away from the former tenet that sinusitis is a cause of bronchiectasis; and some present day observers even claim that the sinusitis present is caused by the bronchiectasis. I have found that sinusitis is very frequently associated with bronchiectasis; and that very little permanent result can be secured by either medical or surgical treatment in bronchiectasis if the accompanying sinusitis cannot be controlled; and very often a case of early bronchiectasis can be improved by merely clearing up the sinusitis present. The protagonists of the sinusitis etiology of bronchiectasis postulate two possible routes of spread of infection to the bronchi: the inhalation route, and the lymphatic route, the former having the most supporters.

The diagnosis of advanced bronchiectasis is usually easy; the coughing up of large amounts of purulent sputum; the dyspnoea and cyanosis; the clubbed fingers; the low grade temperature with acute exacerbations; the history of frequent pneumonia like attacks, especially during the cold months; the hemoptysis varying from slight streaking, to massive pulmonary hemorrhage, all give definite leads to the diagnosis. There is present at some time, a myofascitis, especially along the paravertebral muscles, and often segmental neuralgia; frank rheumatoid arthritis is also seen, but less often.

To diagnose early bronchiectasis, or the prebronchectatic state, constant diagnostic suspicion and readiness to act are necessary; what seems to be unresolved pneumonia may be an atelectatic lobe with bronchiectasis developing; the subacute sinusitis, or bronchitis, may already be complicated by early bronchiectasis; the persistent low grade temperature, weight loss, and listlessness, with or without marked cough, that hangs on after an acute respiratory infection, may be the first vague hint of developing bronchiectasis. In any suspected case, bronchoigraphy should be done. If there is any suspicion of atelectasis from a mucus plug, growth, or pressure from outside the bronchus, a bronchoscopy should be done.

The treatment of advanced bronchiectasis is surgical removal of the affected areas if this can still be done; lobectomy, lingulectomy and other segmental operations, or even pneumonectomy occasionally are the surgical methods in use. The operation of lobectomy twenty-five years ago, carried a mortality rate of about 50 to 60 per cent, in general. In 1940, Churchill reported a series of 124 lobectomies, with a 2.4 per cent mortality. Others reported
no mortality from this operation during recent years. Churchill and Belsey\(^2\) gave impetus to the tissue saving operation of segmental resection, particularly as it applied to the lingular segment. Graham\(^3\) reported the successful removal of both lower lobes, the right middle lobe, and the lingula of the left upper lobe, in one patient.

The treatment of bronchiectasis that is too advanced for surgery is palliative: postural and bronchoscopic drainage; penicillin, sulpha drugs, and streptomycin by parenteral or aerosol administration; or sulfa drugs by mouth are all helpful during the acute episodes; vaccines (stock and especially autogenous); general hygienic measures as proper nutrition, including adequate protein and vitamins; attention to the cardiac complications; residence in a warm, dry climate are all useful. Climate itself may have an ameliorating influence on bronchiectasis. The prompt treatment of acute or chronic sinusitis and tonsillitis cannot be over emphasized; and in spite of the pernicious tendency of many people to overuse vasoconstricting nasal drops until the mucus membranes become swollen and boggy with paralyzed vasoconstrictors, in drops or by local application with a cotton tipped applicator, in sinusitis is as well indicated as is incision and drainage for an abscess elsewhere in the body. Also, removal of bony or hyperplastic obstructions in the nose are necessary.

The proper use of vaccines in early bronchiectasis often gives surprisingly good results at all ages, but especially in children. In children, the regenerative power is superior, and a defect in a child will be relatively smaller when the part has reached adult growth. In using vaccines, it will sometimes be found that the stock vaccine contains antigens closely enough related to the patient's own pathogens that an autogenous vaccine may not be necessary; but more often an autogenous vaccine made from the patient's own sputum becomes preferable.

There are several points in the use of vaccines in bronchiectasis that may make the difference between success or failure; in the first place, these treatments must be kept up for long periods of time, several years at least; however, after a time, such as a year, the periods between doses, which may have been one week, may be lengthened to two weeks or even a month; and rest periods of a couple of months may be taken. The administration should be started with doses small enough to avoid large general and local reactions.

While allergy to such extrinsic agents as foods, epidermals, or pollens, often precedes infection in the respiratory tract and acts by preparing the tract for chronic sinusitis, bronchitis, or bron-
chiectasis, there are a lesser number of patients in whom the intrinsic infection seems to act as a basic sensitizer, making the patient more susceptible to the extrinsic allergens; and when this happens, the patient may be treated for years with extrinsic allergens without results until the intrinsic bacterial antigen is added. The asthma that develops in middle age after an acute respiratory infection and for which no extrinsic allergenic cause can be found, supports this explanation.

Over twenty years ago, while I was practicing in Colorado Springs, Colorado, the chest specialists there tried out pneumothorax and phrenic paralysis for bronchiectasis; at the same time, these methods were tested extensively elsewhere. Our results were very poor, probably mainly because we chose old, advanced cases with hard, thick, unyielding scar tissue around the dilated bronchi. On the other hand, Hennell reported excellent results recently in four cases of early bronchiectasis by using pneumothorax. This is understandable if the disease is early enough; the collapse squeezes out the infective secretions; it stops the rythmical, respiratory pull on the infection-weakened bronchial walls; it approximates the walls of the infected lung units, and smaller bronchi, which contain little or no cartilage, and thus reduces space for secretions to collect as well as aids fibrotic obliteration; it produces a relative anoxia which inhibits the growth of aerobes; and it produces a slowed circulation with its bacteriostatic and local fibrosing effect. Of course, if the bronchial infection were mainly anaerobic, the collapse therapy in bronchiectasis might encourage the growth of the organisms.

However, minor collapse procedures as Banyai's pneumoperitoneum, phrenic paralysis, and pneumothorax have a very important place as emergency treatments for massive pulmonary hemorrhage in bronchiectasis. Probably the most universally useful one of these is pneumoperitoneum: 1) because it is not always possible to determine immediately which lung is bleeding; 2) the basal portion of the lung, the frequent bleeding site, is often uncollapsible by pneumothorax because of pleural adhesions; 3) pneumoperitoneum is usually the least dangerous and most reversible of these procedures.

**SUMMARY**

The incidence of bronchiectasis is greater than generally realized. Bronchiectasis runs a progressively downward course from its inception, and usually claims its victims in the third decade. Its serious complications are: heart failure, metastatic abscesses to the brain and other parts of the body, amyloidosis, lung abscess, pleural empyema and attacks of pneumonitis or pneumonia.
Leaving aside congenital bronchial cysts and other developmental malformations, the cause of bronchiectasis is respiratory infection; stenosis of the bronchi; lack of drainage; infection of the bronchial walls with weakening and destruction of the elastic supporting elements; the rythmical inspiratory pull on the weakened walls, which may be augmented by atelectasis or a check-valve mechanism, which increase the stretching effect on the walls.

The best treatment for bronchiectasis is prevention or treatment of the early phase: removal of a plug of mucus causing atelectasis following bronchial or virus pneumonia or other respiratory infections; prompt treatment of a subacute or chronic bronchitis; prompt treatment of a sinusitis and tonsillitis; attention to respiratory allergies; removal of extrinsic foreign bodies; treatment for endobronchial tumors, extrabronchial tumors and enlarged tracheobronchial and hilar glands; drugs such as sulpha drugs or antibiotics; general hygiene and good nutrition; vaccines (autogenous or occasionally stock) and sometimes a warm, dry climate.

The treatment for advanced bronchiectasis is mainly surgical, if the patient has not advanced to a terminal stage. This consists of lobectomy, segmental resection, and occasionally pneumonec- tomy. The younger patient with his superior regenerative power and his greater anatomical and physiological reserves is the preferred surgical risk in operations for bronchiectasis.

RESUMEN

La frecuencia de la bronquectasia es mayor de lo que generalmente se supone.

Desde sus principios la bronquectasia prosigue un curso progresivamente descendente y, por lo general, reclama sus víctimas en la tercera década. Sus complicaciones graves son: insuficiencia cardíaca, abscesos metastásicos al cerebro o a otras partes del cuerpo, amiloidosis, absceso pulmonar, empiema pleural y ataques de neumonitis o neumonia.

Pasando por alto los quistes bronquiales congénitos y otras anomalías del desarrollo, la causa de la bronquectasia es la infección respiratoria; la estenosis de los bronquios; la falta de canalización; la infección de las paredes bronquiales con debilitamiento y destrucción de los elementos elásticos de soporte; la tensión respiratoria rítmica sobre las paredes debilitadas, lo que puede ser aumentado por atelectasia o un mecanismo de válvula de retención que aumenta el efecto de la tensión sobre las paredes.

El mejor tratamiento de la bronquectasia es la profilaxia, o sea, el tratamiento de la fase temprana: la extracción de un tapón de moco que cause atelectasia subsiguiente a una bronconeumonía o neumonía de virus o a otras infecciones respira-
El tratamiento de la bronquectasia avanzada es principalmente quirúrgico, si el paciente no ha avanzado a un estado terminal. Este tratamiento consiste de lobectomía, resección segmentaria y, ocasionalmente neumonectomía. El paciente más joven, con su superior poder regenerativo y sus mayores reservas anatómicas y fisiológicas es el riesgo quirúrgico preferido en operaciones para la bronquectasia.

REFERENCES

6 “Cunningham’s Text Book of Anatomy,” Edited by Arthur Robinson; *Williams and Wilkins Co.*, New York City, 1921.
THE PROBLEM OF BRONCHIECTASIS


