May I first tell you how much I appreciate the honor that you have bestowed upon me in making me president of this Association. I regard it as one more example of the generosity and friendship which the people of this great country habitually extend to their neighbors to the north. In the thirty-five years of its existence this society, through the excellence of the contributions of its members, has gained a reputation as a scientific body of the first importance. To be its president is therefore not only an honor but a responsibility.

In searching for a subject upon which I might be qualified to address you it is natural that I should have reviewed the presentations of my predecessors. It was a somewhat discouraging task since I found that they were, for the most part, dissertations upon a subject of which the speaker was an undisputed master. Having reached a point in my surgical career at which I cannot conscientiously claim to possess unusual knowledge of any subject, it has not been possible to follow this example. It then occurred to me that it might be worth-while to consider how well the Association had justified the hopes of its founders by reviewing with you some of the contributions of surgery to our knowledge of pulmonary disease.

I must assure my conferees who are physicians, radiologists, bronchoscopists, or pathologists that it is with the contributions that surgery has made that I shall concern myself, those things which it has been possible to learn about disease when what Brock has called "the white light of surgery" has been cast upon them. The meanings of symptoms and signs and the interpretation of shadows in the x-ray films have been removed from the realm of conjecture. Opportunities have been provided to study disease in all its stages and not merely in the end stage as seen at autopsy. Long recognized changes have come to have new meaning to the pathologist, and stages which...
were obscured because of the advanced state at autopsy but obvious in the surgical specimens have made it possible to understand disease processes. The surgeon, noting things on the operating table that are not apparent elsewhere, has been able to understand and interpret for others what only he has occasion to observe.

The first presidential address was given in 1918 by Dr. S. J. Meltzer. He defined the aims of the Association as the advancement of the knowledge of and skill in thoracic surgery. He stated that to date there was no surgical experience to demonstrate whether or not the normal pleural cavity could be opened with safety since all patients operated upon had had diseased pleural cavities. He emphasized that the problems were essentially of a physiologic nature and that surgeons were well trained in anatomy, technique, and mechanical manipulations but that a lack of knowledge led to an undesirable form of conservatism when confronted with problems that were predominantly physiologic.

In the early years much discussion centered about the problems of the open pneumothorax and measures designed to overcome its dangers. Willy Meyer was an enthusiastic advocate of the virtues of the negative pressure chamber, and I was interested to read that in July, 1922, the Lennox Hill Hospital in New York was the only one in America that possessed such a chamber. Inventions and devices clustered around two opposite principles of differential pressure, the one typified by the minus pressure of Sauerbruch and the other by the plus pressure apparatus of Bauer, finally culminating in a single apparatus which combined both principles. Meltzer and Auer first used intratracheal ventilation in 1909, but Matas began experiments upon an apparatus for insufflation intratracheal anesthesia in 1898; he and his associates worked upon the problem for the next fifteen years and made their final report before the American Surgical Association in 1913. The development of a satisfactory method of intratracheal insufflation anesthesia made possible the great progress in thoracic surgery that has occurred during the life of this Association. In his remarks in 1918, Meltzer demonstrated an awareness of the dangers of hypoxia. He stated that when artificial respiration was carried out simultaneously with ether anesthesia the heart could be handled with practical impunity, but that if respiration was insufficient the heart responded to each touch with a series of irregular beats, which in some instances might lead to ventricular fibrillation and a cardiac death. It seems unfortunate that the dangers of oxygen lack and carbon dioxide retention are still insufficiently appreciated.

**Empyema.**—When the Association was founded it was debated whether the question of empyema should be included in thoracic surgery. After some discussion it was decided to include empyema as one of the subdivisions in the surgery of the chest. One wonders if this indicates that surgeons of the day failed to appreciate how bad the treatment of empyema really was and if it required the influenza epidemic of 1918 to impress it upon them.
One of my earliest recollections as a house physician in the winter of 1915 and 1916 is the deplorable treatment of empyema. The patient, with pneumonia who developed a cloudy fluid in the pleural cavity, was regarded as a surgical emergency and transferred to the surgical side where he had an open drainage done under general anesthesia. Of course, the mortality was very high and if by good luck the patient survived this assault there was often prolonged morbidity. My only comfort now is derived from the knowledge that we were not alone in our stupidity. In 1920 Moschowitz published an article giving mortality figures from various centers. For children it gave a mortality of 8 to 25 per cent but in certain hospitals it reached 55 per cent. The average mortality for twenty-five American base and general hospitals was 30 per cent but it reached a maximum of 65 per cent. The recurrence rate probably amounted to 20 to 25 per cent.

The empyema commission set up by the Surgeon General of the United States made contributions to an understanding of the pathologic and physiologic processes involved which completely changed the picture. It was demonstrated that the source of most empyemas complicating a lobular pneumonia was the rupture of a subpleural abscess into a pleural effusion and that except in cases in which the rupture was interlobar in location or pre-existing pleural adhesions prevented a spread, there was a general involvement of the pleural space. Immediate open drainage produced a pneumothorax and collapse of uninvolved lung, and displacement of the mediastinum reduced the effectiveness of the opposite lung. The increase in toxemia and decrease in oxygenation that occurred were often lethal. It was laid down that with the exception of those cases in which empyema was the result of rupture of a gross lung abscess with a bronchial communication it was never a surgical emergency. Treatment was aspiration at intervals frequent enough to control toxemia and the respiratory embarrassment which came from compressions of sound lung and displacement of the mediastinum, until the patient recovered from pneumonia. Usually all that was necessary at that stage was the drainage of a local collection of pus which had become walled off by pleural adhesions. When toxemia was not controlled by aspiration and drainage of a thin pus became necessary, the closed method was advocated. The use of local anesthesia was taught and became standard practice. Almost at once the mortality from empyema dropped to a fraction of what it had been; indeed the loss of a patient because of empyema became a reflection upon his management. Controversy continued upon the virtues of closed and open drainage and the value of irrigation of the pleural space with various solutions but the fundamental principles had been established.

The contributions of members of this association to the management of chronic empyema have been of great importance but their effects have been less dramatic than those which led to an understanding of acute empyema. Today acute infections of the pleural spaces are held by the physicians to be a medical problem and are, for the most part, prevented or cured by aspiration and the use of antibiotics. Only when these fail is the help of the surgeon requested. The teaching of these fundamental concepts continues, however, to be important.
Atelectasis.—It is natural that the problem of postoperative pulmonary complications should have received attention in the earlier years of the Association. What we now know as postoperative pulmonary atelectasis was first recognized in adults by M. Louis in 1829. Schenck in 1811 had described a peculiar condition of the lungs in children dying shortly after birth. The term atelectasis was first applied by Jorg in 1835. Clinical interest in the condition was aroused in 1890 by William Pasteur of the Middlesex Hospital, London, when he published a description of certain pulmonary complications associated with respiratory paralysis after diphtheria. Seringer of Montreal is usually credited with having published the first description on this continent in 1921. Cutler and Hunt published a review of 2,000 surgical admissions from the Peter Bent Brigham Hospital in 1920 but they were impressed by the importance of the embolic factor and stated “We believe that embolism from the operative field is the chief factor in the etiology of such a complication.” Sepsis, trauma, and immobility of the patient were listed in that order as predisposing to atelectasis, with pre-existing lung disease, irritation of the anesthetic and such general factors as old age, chilling, and poor general condition as contributors. They seem to have failed to recognize that the real cause was bronchial obstruction. Scott reviewed twenty cases of postoperative pneumonia at the Peter Bent Brigham Hospital in 1925. One of his conclusions is interesting: “The mechanism of massive atelectasis appears to be a reflex blocking of the finer air passages in the affected lung tissue quite possibly of vasomotor origin.” Infarction in an atelectatic area is suggested as the usual mechanism of postoperative bronchial pneumonia. The differentiation between atelectasis and infarction may pose a problem even today, but it is apparent that embolism played too large a part in the thinking of these observers.

In 1928 Lee, Tucker, and Clerf reported that in thirty-three cases which they had been able to follow three factors had been constant: (1) a thick, viscid, bronchial secretion, (2) some inhibition of coughing, (3) interference of respiratory movements. They aspirated 7 c.c. of thick, viscid secretion from the bronchial tree of a patient within a few hours after the development of postoperative atelectasis, injected it into the bronchus of a dog, and thus produced massive atelectasis of the whole right lung. Of course, since that time there have been innumerable cases in which atelectasis has been relieved dramatically by bronchoscopic aspiration and instances in which it has been produced by occlusion of the lumen of a bronchus. Unfortunately, some of these were accidental experiments in which a bronchus was occluded at operation. Many have contributed to a solution of the problem. The publications of Brunn and Brill in 1930 were of importance calling attention as they did to the role of infection, smoking, and sedation as predisposing factors, and the use of the bronchoscope in demonstrating such etiological factors as mucus plugs, blood clots, and foreign bodies and the exclusion of tumors.

The clinical picture and x-ray changes of this condition are now so familiar to all of us that it must be difficult for the younger men to understand why its recognition and an understanding of its etiology ever presented difficulty.
Thoracic surgeons have contributed largely to the elucidation of its etiology and pathology, to the development of measures which may be employed to lessen its incidence, and the treatment of those patients in whom, in spite of these precautions, it still occurs. Although incidence of atelectasis has been much reduced by better preparation of patients before operation, better anesthesia, attention to posture, and the encouragement of activity (including deep breathing and coughing in the postoperative period), it is still not rare. The frequency with which it is recognized when still minimal in extent bears a direct relation to the astuteness and diligence of the house surgeon. An awareness and understanding of the condition by physicians and surgeons has played a major role in the reduction of postoperative morbidity in all fields of surgery.

**Lung Abscess.**—In the earlier years of the Association there was much controversy regarding the etiology and pathology of lung abscesses. In spite of the contributions made by animal experiments I believe that an understanding of the disease has been reached through clinical observation rather than experimentation. Undoubtedly the postoperative abscess was much more common twenty-five years ago than today. I suppose it was natural that the possibility of an embolic origin should have received support since, although more common after operation upon the upper respiratory tract, an abscess might follow any operation.

The experimental work of Cutler and his associates demonstrated that a single lung abscess could be produced by an embolus from the jugular vein. This seems, however, to have been an artificial way of initiating the process. That the majority of abscesses were at first single rather than multiple as might be expected from septic emboli and that the bacterial flora was so frequently mixed rather than single would appear to have attracted insufficient attention. The hematogenous origin received support from such worthy champions as Howard Lilienthal and Evarts Graham. Dr. Graham mentioned a triangular area of density near the periphery of the lung which he had observed in the roentgenogram following laparotomy and preceding the development of a lung abscess which he thought could be explained more readily on the basis of embolism than aspiration. I have no doubt that this was a very early recognition of a shadow which many of us have seen since that time and accept as an area of atelectasis which has resulted from the plugging of a small bronchus by a bronchial embolus. Lambert and Miller, in 1924, reported that anaerobic organisms had predominated in ten cases upon which they had done an open drainage; Flick, in 1926, referred to these findings and gave aspiration a prominent place but conceded the possibility of a vascular origin in certain cases. Ambrose Lockwood supported the aspiration origin in 1922. Frederick T. Lord was one of the earliest proponents of the theory of aspiration. He stated in 1925 that aspiration of infected material from above into the deeper parts of the respiratory tract appeared to be the chief
cause. He also said that lung abscess rarely followed lobar pneumonia and was not the result of a failure of resolution but a special kind of pneumonia due to aspiration.

_Tuberculosis._—Increase in the knowledge of pulmonary tuberculosis has been a very gradual process in which many have played a part. The bronchoscopist, the radiologist, and the surgeon have made it possible with the help of the internist and pathologist to bridge the gap that existed when sole dependence had to be placed in the clinical picture and autopsy findings. The lungs of patients coming to autopsy exhibited an end-stage disease from which it was difficult to reconstruct a picture of successive changes which had led to that final state. It seems probable also that the manner in which the lungs were examined, by making leaflike sections from the periphery to the hilum, focused attention upon the changes in the lung parenchyma rather than those in the bronchi. The pathologic material available in most sanatoriums must, however, have been studied insufficiently; otherwise cases of chronic bronchiectasis of nontuberculous origin, bronchiogenic tumors, and various other nontuberculous lesions would have been sorted out sooner. Perhaps to some extent the mere possibility of the employment of surgical therapy led in many instances to a more careful assessment of the anatomic location of the disease and the pathologic processes at work.

Although knowledge of the occurrence of tuberculous tracheobronchitis is by no means recent, it would appear that a general awareness of its frequency and importance dates from the contributions of Eloesser in 1931 and 1932. Samson’s paper, in 1936, added to a knowledge of the bronchoscopic picture. The feeling that bronchoscopy might aggravate the disease persisted for a considerable time; it was only with the gradual realization that no ill effects resulted, provided the procedure was carried out expertly and without trauma, that the bronchial changes came to be observed in various stages of progression and healing. As lobectomy and pneumonectomy came to occupy an increasingly important place in therapy, opportunities were provided to study not only the changes in the major bronchi but those in minor bronchi beyond the vision of the bronchoscopist. Atelectasis of lobes or portions of lobes as seen in the x-ray film came to have new significance to the radiologist and clinician and to be regarded as an indication of bronchial disease. It was realized that what had been interpreted as pulmonary fibrosis was often atelectasis due to bronchial occlusion. The persistence of a cavity in an apparently completely collapsed lobe under pneumothorax carried a new import. When a positive sputum persisted in spite of the complete collapse of a lobe under a thoracoplasty, the demonstration of widespread disease of bronchi in the lobe when it was removed surgically explained the occurrence.

Thoracotomy upon patients thought suitable for lobectomy or even local resection has led to an increasing awareness of the limitations of the roentgenogram. Who among us has not been amazed and chagrined to find that widely dispersed tuberculous nodules existed in pulmonary tissue which the films had led us to consider free from disease. An apparently normal roentgenogram
in a patient with positive sputum may suggest that the patient has tuberculous bronchitis but tuberculosis of the lung, not demonstrable by x-ray picture may also be present. The failure of the x-ray examination to demonstrate parenchymal changes in the patient with a pleural effusion can no longer be interpreted as excluding pulmonary disease. Indeed it can be assumed that there is a primary lung focus which the x-ray photograph has failed to show. To that extent primary tuberculous pleural effusion is a misnomer.

What the surgeon has seen at thoracotomy and the study of the pathologic material which his efforts have made available have led to increased accuracy in diagnosis and consequently better and more rational treatment of the individual patient.

**Bronchiectasis.**—Dilatation of the bronchi was mentioned by Storeck in 1761, and the first anatomic and clinical account of the disease was given by Laennec in 1819. I have often wondered why the disease failed to make a greater impression upon the student of my day. It is probable that in the majority of cases the diagnosis was chronic bronchitis and that the pathologist saw only the far-advanced or terminal disease and its complications. It was not possible to study the disease in its various stages of development until surgical material became available. The first really good pathologic study, published in 1922 by Paul M. Aschner, was made upon operative specimens provided by Lilienthal. Multiple stage operations did not yield material suitable for study, but as the one-stage procedure gained in popularity further studies became possible. Robinson made an excellent report upon the early specimens obtained by the one-stage technique in Toronto. Greer studied the bacteriology of the disease from the same material. Perhaps the most striking bacteriologic evidence produced was that if there were a common causative factor it was not bacterial since there was no common organism. Occasionally the bronchi contained only a single variety of organism; in others there was a mixed flora in which spirochetes might or might not be present.

From the abundance of pathologic material made available in the last twenty years it has been possible to recognize certain etiological factors. Partial obstruction of the bronchus, whether by enlarged lymph nodes or other structures pressing upon it from without or by a foreign body (a plug of mucus, an adenoma, or carcinoma) in its lumen, may be the initiating factor. The addition of an infection which destroys the elastic and muscle tissues in the wall of the bronchus seems to be essential. The development of bronchiectasis after a virus pneumonia in so many soldiers whose chest films were normal on induction into the army has raised again the possibility of a virus origin for the disease, a suggestion made at the time of the 1918 to 1919 influenza epidemic by Lord of Boston and Vogues of Johns Hopkins. The dilatation seems to develop occasionally with great rapidity. Whether a true bronchiectasis once formed in such cases is ever recoverable remains a moot point. The possibility that the presence of a specific agent, most probably a virus, is necessary for the destruction of the muscle and elastic tissue would explain certain observations that are otherwise difficult to understand. Why, for
example, does bronchiectasis complicate certain cases of long-standing lung abscess but fail to occur in others though the conditions for its development seem the same.

Surgery has made it possible for us to learn much about the more obvious changes in this disease and to relate them, in at least certain instances, to apparent etiological factors. The specific etiological factor, if such there be, still awaits discovery.

**Arteriovenous Aneurysm of the Lung.**—Surgeons have had a part not only in the therapy but in the study of that interesting condition known variously as hemangioma, arteriovenous fistula, or arteriovenous aneurysm of the lung. The gross pathology of the lesion is more evident as it is observed through the open chest than at any other time. The thin-walled sac can be observed, the blood can be seen coursing through it, a thrill can be felt which explains the bruit that was heard through the chest wall, and the sac can be made to collapse by compressing the branch or branches of the pulmonary artery entering it. An opportunity is provided to appreciate the relatively enormous quantity of blood that is being by-passed from the arterial to the venous side of the pulmonary circulation and to understand why such an apparently insignificant anomaly should have such a profound effect upon the physiology of the individual.

Following excision or at autopsy the thin walls collapse and the lesion may be difficult to find and its importance still more difficult to understand. It does not require great imagination to realize that small arteriovenous communications of this nature may account for an otherwise inexplicable pulmonary hemorrhage even though it has not been possible to find a source of bleeding in the surgically removed lung or the autopsy material. Various members of this Association have contributed to an understanding of the physiologic disturbances that may be associated with the condition and considerations upon which surgical therapy should be based.

**Bronchiogenic Carcinoma.**—That the incidence of cancer of the bronchus has increased can no longer be questioned. It is probable, however, that much of the apparent increase is to be attributed to the ability of the clinician, the radiologist, and the pathologist to recognize the many and varied manifestations of the disease. It is not hard to realize that it may have been difficult to diagnose many cases before the radiograph became available and when exploration of the thoracic cavity was rarely undertaken. It is less easy to understand why it was not discovered at autopsy by the painstaking morbid anatomist of the past. On reflection one appreciates that the situation is by no means unique. Pulmonary embolism was thought by most pathologists to be a rare finding until Belt published his monograph in 1934. I am told that pernicious anemia was considered rare until Minot and Murphy's discovery in 1926. For more than 400 years it was known that one of the most common causes of death among the miners in Schneeberg in Saxony was a progressive, wasting pulmonary disease presumed to be tuberculosis or silicosis. Osler stated in
his first textbook, published in 1894, "It is a remarkable fact that the workers in the Schneeberg mines are very liable to primary cancer of the lungs. It is stated that in this region a considerable proportion of all deaths in persons over forty are due to this disease." In 1922 Schmorl attracted attention to the disease and vital statistics at once demonstrated a great increase in its incidence. Books on pathology published in the latter part of the last and the early part of this century mention the disease as a rule only to comment upon its rarity. One receives hints suggesting why it may have been missed. The projection into the bronchus was regarded by some at least as a late manifestation of a tumor which had secondarily invaded the bronchus; the involvement of the lymph nodes was regarded as the primary rather than the secondary disease; the extensive broken-down masses in which at times cavities, that communicated with the bronchus, had formed were mistaken readily for advanced tuberculosis. Anyone interested in this aspect of pathology should read Boyd's monograph.  

![X-ray studies of bronchial movements. Charles C. Macklin.](image)

Knowledge of the various clinical manifestations and of the gross and microscopic appearance which may be presented by the disease developed from Graham's first successful operation. Physicians, surgeons, bronchoscopists, and radiologists have all played their part in building up the clinical picture, and the pathologist, provided by the surgeon with growths in all stages of development, has benefited from the opportunity. The knowledge thus gained has thrown new light upon and has led to a complete change in the interpretation of autopsy findings. It is unfortunate that our efforts to cure the disease have not been as fruitful.

I cannot refrain from suggesting to the younger men the pleasure and the value that come from a survey of the literature of the past. It may be
discouraging at times to discover that an idea is not new at all but was presented so long ago that it has been forgotten, but it at least saves one from the embarrassment of presenting it as original. On the other hand the germ from which many important contributions have developed has come not infrequently from such reading.

Studies on the behavior of the bronchial tree in inspiration and expiration published by Macklin in 1925 make it easier to interpret the changes in bronchograms in the presence of an elevated diaphragm, pleural fluid, or other space-filling substance (Fig. 1). I believe that Willy Meyer was the first to suggest the use of a lung tourniquet in 1923. He had several “Baby-Sehrt” clamps made, a modification of the larger clamp Sehrt had devised for limb amputations (Fig. 2). He says, “Having watched the working and many advantages of Sehrt’s metallic tourniquet in limb amputations I planned to try small metallic tourniquets manufactured on the same principle, ‘Baby-Sehrts,’ in lung extirpations.” Unfortunately he apparently never used the clamps, and Dr. Shenstone and I were unaware of his suggestion at the time of our original publication.

I suspect that Schlueter and Weidlein published the first description of a one-stage dissection lobectomy in connection with experimental lobectomy and pneumonectomy on dogs in 1926 (Fig. 3). The vessels were dissected and individually ligated and the bronchus crushed, ligated with chromic catgut, and invaginated. I have often wondered whether Whittemore read Paget’s book before he devised his technique for lobectomy. He could certainly have obtained his idea from the case report which Rolandus published in 1499. It may be that the first description of a resection of a cancer of the lower
esophagus with anastomosis of the cut end of the esophagus to the stomach in the chest is to be found in an obscure footnote in a paper read by Dr. Herman Fischer at the 1925 meeting of this Association. Dr. F. N. G. Starr of Toronto performed the operation in November, 1922.

It has been possible for me to allude very briefly to some of the foundations upon which our present-day concepts of disease and therapy are based, Churchill reminded us in his address, in 1948, that we have thus far worked only the surface and that much that has been contributed has been in the nature of the obvious. The presentations upon the program of this society, not the least those of this year, assure us of continued progress.

![Fig. 3.—Experimental one-stage lobectomy. Schlueter and Weldlein, 1926.](image)

I believe, however, that it is important that the activities of today are based upon a knowledge of the past as well as faith in the future. I should like in concluding to quote from a recent issue of the *London Times Weekly Review*. "If the past be treated as nonexistent, then, since the future is not yet, while the present vanishes instant by instant into the nothingness of the past, there can be no reality. Thus while much of the past is lost in the oblivion of human forgetfulness, it can never be as though it never was."\(^{34}\)

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