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MASSIVE COLLAPSE OF THE LUNG

With Special Reference to Etiology
FOREWORD:

My object in reviewing the literature and presenting a thesis on the subject of Massive Collapse of the Lung is because it is an extremely interesting subject from the standpoint of etiology, and also because my interest was stimulated by the possibility that some day the Medical men may recognize the importance of bronchoscopic treatment in pneumonias.

This is not a new subject as it dates back to 1853, at which time much work was done on this subject. From that time on up to the present many theories have been presented as to the etiology of massive collapse of the lung, and what I aim to do in this review of the literature is to try to establish the most logical theory as to the etiology of massive collapse of the lung.

It will be impossible for me to state any of my own ideas on the subject as they are not backed up by research or clinical experience, which, after all, is the most important phase when conclusions are to be arrived at. I will, however, be able to judge for myself which of the many theories as to etiology is backed up by the most proof.

In this thesis I will also review the possibility of massive collapse of the lung as being an etiological factor in the production of post operative pneumonia.
HISTORY:

The rediscovery of massive collapse of the lung, particularly the relation of the condition to post operative and post traumatic states, dates from the paper written by W. Pasteur in 1910. The history of massive collapse goes back from 1910 sixty to seventy years or more to contributions of extraordinary merit.

Willshire writing in 1853 gives a detailed account of the events, beginning, he thinks, with Schenk in 1811, through which the differential diagnosis between lobar and lobular pneumonia had passed. He is said to have found evidence that Wilbrandt in 1816 and Eberhardt in 1817 and Lucae in 1819 followed with clear recognition of a post mortem state in the lungs of infants, which they regarded as congenital or due to the musculi bronchioles not being as yet sufficiently accustomed to the rhythmic order of respiration.

In 1830 Alderson, writing on "Hooping Cough", describes four autopsies upon children dying from that disease. He gives an accurate description of partial collapse of the lung, but to which he did not assign any name. Alderson distinguishes this condition from any inflammatory process of the lung.

Duges in 1821 and Shoelgruber in 1823 described several autopsies and gave very clear and accurate descriptions of massive collapse occurring in children up to the time the majority of the scientists held the theory of congenital non-expansion.

In 1834 Joerg wrote on a particular condition of the lungs of new born children and worked out this condition more fully in 1853. Willshire
believes that the term atelectasis was first applied at this time.

Barlow in 1841 reported a case of atelectasis following, or the result of, a dilated heart. In another case reported by Barlow, the condition of atelectasis seems to have been accentuated from an obstructive bronchitis.

Gairdner in 1854 in a review credits Duges, 1821, Louis, 1829 and Joerg in 1832, with recognizing the condition in autopsies and giving the name of carnification to the condition they found. These men all recognized the points of difference from pneumonia.

Louis found carnification, as he called it, in nineteen out of forty-six autopsies, the patients having died from typhoid fever. Louis did not offer any explanation as to the probable mechanism of it.

In 1841 Rilliet and Barthez gave the following description of carnification, i.e. their name for massive collapse of the lung: "The lung is soft, flaccid and extremely collapsed in contra-destination to pneumonia where it is full, hard and resistant. The color is violet marked by white lines disposed in lozenges or squares, defining the lobules. There is no crepitation on pressure. On incision the lung is found to be of a red color, smooth, resisting pressure of the finger so as to be penetrable with difficulty, exuding upon pressure a serous, bloody fluid which is destitute of air. Rilliet and Barthez suggested that the appearance resembled the close, compact fibres of muscle. As to distribution, these two scientists thought that the carnification might be marginal or some part of a lobe, the middle lobe being the only one that was ever seen to be entirely collapsed. (This may be the first mention of the term "massive collapse")."
Barthez considered the condition as a terminal phase of pneumonia.

In the year 1844 Legendre and Bailé established the autopsy identity of congenital non-expansion with a return to a similar state in lungs which had once expanded.

Mendelssohn and Traube in 1845, working upon dogs experimentally, produced collapse of the lung by occluding a bronchus with shot, paper and gum arabic. Traube was skeptical as to the mechanism of the experiment and also as to the importance of it.

The first systematic treatise originating in the English language was written by Charles West. He shows familiarity with the condition and distinguishes it from inflammatory states and also from pneumonia. Although West did not agree with Bailé and Legendre as to the etiology which they set forward or as to their explanation of their observations (they believe collapse of the lung is the result of engorgement of the pulmonary circulation); he, however, states that these two men have thrown more light on the affections of the lungs in infancy and early childhood than all the writers of the previous ten years taken together.

Fuchs in 1845 wrote a criticism on the then accepted conception of lobular pneumonia and considered it to be an inflammatory condition of the mucous membrane of the bronchi combined with a contingent lesion of the pulmonary vesicles similar to that existing normally in the fetus and proposed the name apneumatosis. By the year 1845 we see then that the three terms in use today had been proposed, namely atelectasis, collapse and apneumatosis.

In 1850 Gairdner brought forth the theory that lung collapse was the result of partially inspissated mucous which acted in the bronchi as
a "ball valve". He stated that the usual place where the ball valve would be effective would be at the bronchial bifurcations. Even though this theory is almost wholly disregarded today, it has still some worthy supporters, namely Chevalier, Jackson, Dixon and Brodie.

Two years after Gairdiner formulated his theory as to the etiology of massive collapse of the lung Meigs addressed the Philadelphia Medical Association on Atilectasis Pulmonum. In this address Meigs made a clear differentiation between massive collapse of the lung and bronchial pneumonia.

Stokes was the first man to really understand mediastinal displacement although it had been observed before. Stokes recognized mediastinal displacement as a sign and result of vesicular emphysema of Laennec.

It is very difficult to understand how Gairdiner, Jenner, Hewitt, Lichtheim and others before Pasteur's time failed to recognize mediastinal displacement, which is by far the most outstanding symptom of massive collapse of the lung.

The work of Meigs, West and Foster seems to represent the entire American knowledge at this time, whereas in Europe there was a great deal more thought given to the work. In Europe there were those that agreed with the work and findings of Legendre, and Bailey and those that agreed with Rilliet and Barthez, who took a middle ground.

In 1879 Lichtheim, not satisfied with the findings of Traube and Mendelssohn, decided to try their experiment. By instituting more accurate instruments and perfecting a technique he hoped to be able to come
to some definite conclusion after his experiment. He concluded that collapse would occur only when the blood stream to the lung was unimpeded.

Lichtheim's conclusion still holds good today as most recent observers have made the observation upon experimental animals that it is necessary to have the pulmonary circulation intact before collapse of the lung will take place.

It was not until 1890 that Pasteur published his first paper on respiratory paralysis as a cause of pulmonary complications. Pasteur's idea was that respiratory paralysis after diphtheriae (as he had noted it) was the cause of lung collapse, the diaphragm becoming immobile because of phrenic paralysis. Pasteur's idea had many supporters, but most of the scientists were very skeptical as to the value of his contribution to science.

In 1907 we have the first recognized and recorded case of heart displacement as seen in a case of lung collapse, i.e. after Stokes. Samuel West presents a case of plugging of the bronchus in plastic bronchitis. West sketched the displaced heart and mediastinum and the probable mode of mechanism. The conclusion reached by West is that which is generally accepted today, namely complete bronchial obstruction.

Again in 1908 Pasteur published his second paper. This paper was identical with his first publication, except that he reported more cases. He also included in this report several autopsies. In all of the cases which he presented phrenic disability was the outstanding feature. It was not until 1910 that Pasteur came out with his masterpiece of research work. In his paper on active lobar collapse after abdominal operations Pasteur makes the first recorded mention of heart displacement as a cardinal diagnostic
sign. Pasteur also makes here the first mention of massive collapse as being a type of post operative complication. Pasteur presented an analysis of 3,559 surgical operations between the years of 1906 and 1910. Out of this number of cases there were 201 pulmonary complications. Eighty-eight cases were classified as post operative pneumonias and twelve as massive collapse. Out of the 201 pulmonary complications there was a mortality of forty-five.

Although Pasteur in all of his work on the subject of massive collapse of the lung was very scientific and tried to justify his conclusions by clinical data and experiments, nevertheless, he was not quite satisfied with his theory of reflex diaphragm hypothesis. In spite of the fact that he was not able to feel satisfied with his theory, he did not believe in the bronchial obstruction theory and, therefore, he decided to leave further investigation to other scientists and follow his own theory in the meanwhile.

During the World War numerous cases of traumatic massive collapse were reported by Bradford and Crymble. There was much speculation concerning these cases because it was found that some of the cases presented contra-lateral collapse of the lung from the point of injury. Bradford and Crymble seemed to accept Pasteur's theory in part as the etiological factor in the production of the collapse. Why the condition appeared contra-lateral to the point of injury at times, however, was not explained by Bradford or Crymble.

In American literature the first communication after Pasteur was that made by Scrimger of Montreal, who, after reporting seven cases of
massive collapse of the lung out of 580 operations, came to the conclusion that the condition was the result of bronchial constriction due to a vagus reflex and followed by viscid secretions which brought about obstruction to the bronchi followed by absorption of the retained air in the terminal alveoli.

In 1921 Sewall of Denver, Colorado began to take an active interest in the study of massive collapse as related to physical examination of the chest in regard to pulmonary tuberculosis. Sewall studied Gairdener, Willshire and Lichtheim for a background and then proceeded with his study of massive collapse in relation to pulmonary tuberculosis. Sewall stressed the idea of keeping atelectasis as a possibility always in mind when examining the chest. He especially emphasized the frequency of non-traumatic massive collapse of the lung.

With the invention of the bronchoscope in 1897 by Killion and its improvement by Jackson in 1904 more understanding of intra-pulmonary conditions has been brought about, of which very little had been known before. The bronchoscope has not only become a valuable instrument in diagnosis, but it has also become a very valuable instrument in treatment, especially the removal of foreign bodies from the bronchi.

A brief outline of the history of massive collapse has been presented with the view in mind of establishing a background for the following discussion. The various theories which exist today in regard to the etiological factor or factors will be presented with discussions and criticisms of each. In this way I hope to establish to my satisfaction the most probable theory as to the etiology of massive collapse of the lung.
MECHANISM OF RESPIRATION:

A brief review of the mechanism and physiology of respiration is important here as the correct understanding of the mechanism and physiology will enable one to better understand the pathologic processes which are to be considered later in this paper.

The act of respiration consists of two parts, both parts being opposite movements, namely inspiration and expiration. The movements of inspiration result in an increase of the capacity of the thorax, which is partly brought about by muscles acting on the ribs, sternum and vertebral column and partly by the diaphragm. The transverse diameter of the thorax is increased by elevation and eversion of the ribs, and these movements are produced during quiet respiration by the intercostal both internal and external, subcostals and levatores costorum. As a result of the elevation of the ribs, the sternum is thrust forward, particularly in its lower part, thus increasing the thoracic capacity in an antero-posterior direction.

In forced inspiration the transverse and the antero-posterior diameters of the thorax are further increased by elevation of the first rib, elevation and forward movement of the upper part of the sternum, and diminution of the concavity of the thoracic part of the vertebral column accompanied by further elevation and eversion of the ribs. The elevation of the first rib is produced by the scaleni, anterior and medius muscles and also by the sterno mastoid acting through the sternum, the sterno clavicular joint and the costo clavicular ligament. Elevation and forward movement of the upper part of the sternum necessarily accompany this elevation of the ribs and this is produced by the action of the seratus anterior and pectoralis minor. For this
purpose it is necessary that the scapula should be fixed by the trapezius, the rhomboids and the levator scapulae. The effect of the diaphragm may be said to be three-fold. First, when it is relaxed, the fibres of the diaphragm curve upward over the liver, stomach and spleen, and the disappearance of this curve and consequent straightening of the fibres when the muscle contracts provide a great increase in the depth of the thorax. Secondly, the central tendon of the diaphragm descends somewhat. As a result of the contraction of the fibres of the diaphragm and, secondly, the descent of the central tendon, the abdominal viscera are thrust downward and the relaxed abdominal wall is thrust outward. The third effect of the diaphragm is that the eighth, ninth, tenth and eleventh ribs are slightly elevated by the diaphragm, but the lower ribs as a whole tend to be thrust outward by the abdominal viscera, and the twelfth rib is fixed by the quadratus lumborum muscle.

The movement of expiration is brought about by a diminution of the transverse and antero-posterior diameters of the thorax. This is effected by the elastic recoil of the costal cartilages and the weight of the thoracic cage. The depth of the thorax is diminished by the reproduction of the concavity of the cupola of the diaphragm. This result is brought about by the contraction of the abdominal muscles. In forced expiration these muscles are reinforced by the latissimus dorsi, which exerts a powerful compressing effect, and vertebral column is fixed, thus allowing the ribs to be still further approximated.

Now that the mechanism of respiration has been considered, it will be advisable to briefly discuss intra thoracic pressure.
At the end of expiration the lungs are in a stretched condition. This is shown by the fact that if in an animal or in a corpse an opening is made into the pleural cavity, air rushes into the opening and the lungs collapse, driving a certain amount of the air out through the trachea. Since the lungs are always tending to collapse, it is evident that they must exert a pull on the thoracic wall. This pull of the lung gives rise to a negative pressure in the pleural cavity. If we connect a mercurial manometer with the pleural cavity, we find that the pull of the lungs amounts in the corpse to 6 m.m. of mercury. If the lungs are fully distended as after full inspiration, the elastic forces are brought more into play, and the negative pressure in the pleura may amount to 30 m.m. of mercury. Since the lungs are always tending to collapse, respiration becomes impossible directly free openings are made into the pleural cavities on both sides. With each inspiratory movement air rushes in through these openings so that the thoracic movements can no longer exert any influence on the volume of the lungs. The negative pressure in the thorax is diminished by any factor decreasing the elasticity of the lung tissue. Thus, in an old man, when the elastic tissue is degenerated and the alveoli are also enlarged, giving rise to the condition known as emphysema, the lungs may collapse only slightly or not at all on opening the chest. The lungs do not collapse on making an opening in the chest of a new born mammal, but this owing to the fact that they completely fill the thorax in the expiratory position, and it is only later that with the growth of the ribs the thorax gets, so to speak, too large for the lungs, which are therefore stretched to fill it.

The force exerted by the inspiratory muscles is nearly all spent in overcoming the elastic resistance of the lungs and costal cartilages.
A free access of air is provided for by contractions of certain accessory muscles of respiration. With each inspiration the glottis is widened by abduction of the vocal cords. When the glottis is observed by means of a laryngoscope, a rhythmical separation and approximation of the vocal cords are observed, synchronous respectively with inspiration and expiration. When inspiration is labored, the alae nasi are dilated by the dilator nasi. This movement of the nostril, which is constant in many animals, becomes very marked in children suffering from any respiratory trouble.

If a manometer be connected with one of the nostrils so as to register the pressure in the air cavities, it is found that there is a negative pressure of 1 m.m. mercury with inspiration and a positive pressure of 2 to 3 m.m. mercury with expiration. With forced inspiration the negative pressure may amount to -57 m.m. mercury, and with forced expiration there may be a positive pressure of 87 m.m. mercury.

Under no circumstances can we by forced expiration empty the lungs of air. At the end of the most forcible expiration, if the pleura were perforated, the lungs would collapse and drive more air through the trachea. When breathing quietly a man takes and gives out at each breath about 500 c.c. of air measured dry and at 0 degrees C. If measured moist and at the temperature of the body, viz. 37 degrees C, the volume would be 600 c.c.. This amount is known as the tidal air. By means of a forcible inspiratory effort it is possible to take in about 1,500 c.c. more (complemental air). At the end of a normal expiration a forcible contraction of the expiratory muscles will drive out about 1,500 c.c. more (supplemental air). These three amounts together constitute the vital capacity of an individual. This total
may be determined by means of an instrument known as the spirometer, which is merely a small gas meter with a gauge by which the amount of air in it can be at once read off. The person to be tested fills his lungs as full as possible and then expires to the utmost into the spirometer. The air left in the lungs after a vigorous expiration is known as the residual air.

Normal respiration has been considered now from the standpoint of mechanism and intra-thoracic pressure. We are now ready to consider the changes which occur when the condition of massive collapse ensues.

Massive collapse brings about dyspnea, cyanosis and compensatory emphysema on the unaffected side. The lung that is involved hardly moves with respiration and the diaphragm remains in a high fixed attitude on the unaffected side. Negative intra pleural pressure is greatly increased and vital capacity is considerably diminished. Due to the ensuing massive collapse the normal mechanism of respiration on the affected side is completely lost and an immobile chest is found. Whether the immobility involves part of the lung or all depends upon the degree of the collapse.

Yandell Henderson-54-states that in every major operation there is not only a decrease in the volume of air breathed, but also a prolonged loss of tonus and relaxation in the thoracic muscles of the diaphragm. Vital capacity is decreased, lungs deflated, and occlusion of the pulmonary airways occurs often. The lungs normally are the best drained organs in the body. Their drainage is maintained by cilia, coughing and by muscular contraction of the walls of the airways. Shallow breathing predisposes to obstruction, and conversely deep breathing tends to distend atelectatic areas. The natural stimulant in producing normal respiration is CO2, and that is
produced within the body. Macklin states that the bronchi and bronchioles have a definite muscular tonus, contractile tissue and probably peristaltic. There are three methods by which the respiratory tract frees itself from foreign bodies: (1), cough reflexes; (2), action of cilia; (3), peristaltic movements in the air sacs, bronchioles and bronchi. The rate of peristalsis is one wave every seven or eight minutes.

DEFINITION:

By massive collapse, atelectasis or apneumatosis is understood the deflation of a large portion of the lung tissue to a retracted airless state. This is the definition given by Herman Hennell-21-in the "Archives of Internal Medicine for 1929". This is a fairly good definition but it is not as complete as the following definition, which is given by Dyke and Sosmen, Journal of S.G. & O. 1929-21. Post operative massive collapse is a reaction of an obstructive nature in which the affected part of the lung becomes airless, characterized by displacement of the mediastinal contents of the involved side, decreased radiability of the affected lung tissue, a high diaphragm and flattened chest on the same side, increased respiration and pulse rate, cyanosis and usually a rather abrupt onset within twenty-four to forty-eight hours after an operation.

The definition, as can be seen, is quite descriptive and covers the essential factors in massive collapse of the lung.

Dr. David R, Bowen-5, radiologist of the Pennsylvania Hospital gives a good definition of this condition and it is well worth mentioning. Acute massive collapse (atelectasis) of the lung is a definite clinical entity which is characterized by more or less airlessness of a lobule, a partial or
entire lobe, a whole lung or even of both lungs. It is rarely seen without some measure of increased or retained secretion which may be excessive and which rapidly becomes purulent. Dependent upon a variety of predisposing factors its final incidence is practically without exception due to complete occlusion of a bronchus or a bronchial branch with subsequent absorption of the retained air, and its area is definitely limited to that part of the lung served by the bronchus so occluded. The most important premonitory symptom is a moderate elevation of temperature; the most notable sign is dyspnoea; and the most reliable physical and roentgen finding is displacement of the heart towards the involved lung.

Massive collapse affects both male and female to the same degree, and there is no particular age that is immune from this pulmonary complication. It is hard to estimate the frequency of the condition as some hospitals confess that the condition occurs often, while other hospitals state that it does not occur at all. Dr. Bowen-5-persists in believing that the hospitals that do not find the pulmonary complication of massive atelectasis have not as yet begun to think atelectasis, and that as soon as they begin to look for the condition in the post operative cases, they will find it often. Cutler-55-states that one case of massive atelectasis occurs in every thirty to fifty post operative cases. Mastics, Spittler and McNamee-40-state that approximately seventy per cent of all post operative pulmonary complications are atelectasis. Gwyn-56-57-believes that acute collapse as a complication of pneumonia has been under our eyes from all times. The teaching of all this is that massive collapse occurs with sufficient frequency to make it distinctly important to think atelectasis whenever there is any post operative lung com-
plication to say nothing of its occurrence in lung disease generally.

Scrimger-44 states that the frequency with which massive collapse or atelectasis occurs can not be recognized with any certainty since it only now is being recognized, not as a curiosity, but as one of the most frequent of post operative complications. As Sewall of Denver said, every time a physical examination is made the examiner should think atelectasis. If this were done, the frequency would be alarming, but when it is not sought for, the cases are classed among the post operative pneumonias or as pulmonary emboli. Sante-45 finds that the condition of massive collapse is most usually seen as a post operative complication of abdominal and rectal surgical procedures, especially following appendectomy. Its occurrence seems to be independent of the anasthesia used, and it has even followed spinal anesthesia. Massive collapse has been found to occur after fracture of the pelvis or of the femur and after injuries apparently trivial to the chest, abdomen and buttocks.

Pasteur found massive collapse in twelve out of 201 post operative cases, and H. H. Davis of Omaha made the statement that massive collapse occurred in fifty to seventy-five per cent of operative cases, but most of them are never known to be present.

INVolVEMENT:

As to type of involvement, the condition of massive collapse may be considered as (1) massive, involving a lobe or lung, and (2) partial, lobular or patchy when smaller areas are involved. The second type has an especial importance, Lee-58, in that it almost certainly affords frequently a lodging for minute emboli which, as Cutler-55 has shown, are a constant result
of all operative procedures in which blood vessels are opened. Mastics, Spittler and McNamee suggest the following classification:

(1) Fulminant, with sudden onset and a stormy course ending by crisis, lysis or with complications.

(2) Moderate with fairly well marked differential signs but of distinctly lessened severity.

(3) Latent with little or no subjective symptoms but demonstrable clinically by physical examination and by roentgen ray appearance.

(4) Evanescent without subjective symptoms and with physical signs suggestive only or more commonly undiscovered but demonstrable by roentgen ray.

Sante states that there are two types of involvement in massive collapse of the lung. The disease manifests itself in two more or less defined forms. In the first form the respiratory distress predominates, and in a case of this nature the onset is sudden within one to four days after operation or trauma, coming on with pain in the chest, cyanosis, dyspnoea and a rapid pulse. The temperature may be normal at first but usually ranges from 98 to as high as 103 within a few hours. In a few days there is a productive cough expelling muco purulent sputum. The sputum is never a prune juice variety. The development of the fever seems to run parallel to the quantity and character of the expectoration. The white blood count is really somewhat higher than normal. With the appearance of the muco purulent sputum the white blood count reaches as high as 20,000. In the second type according to Sante the pulmonary symptoms are entirely secondary to other symptoms. In this type there seems to
be little respiratory disturbance, very little pain in the chest and little change in the pulse note although there may be fever, cough and leucocytosis later, they are usually attributed to the inciting condition.

INCIDENCE:

The date of incidence is frequently within twenty four hours and commonly within seventy-two hours although occasionally it is reported seven to ten days after the operation. The site of election has been reported as the right lower lobe although other investigators such as Crymble have found the larger numbers of cases occurring in the left lower lobe. Most of Crymble's cases were war wounded, several of them with contralateral collapse. Contralateral collapse was found commonly during the war and is not uncommon among the post operative cases. Because of the contralateral appearance the theory of reflex etiology has appealed to many. Bowen believes that Scott and Joelson offer a more credible theory in asserting that the position which the patient assumes because of the discomfort following the abdominal operation is the largest factor in determining the site of collapse and therefore producing this contralateral phenomenon.

Since massive collapse occurs most usually as a result of a post operative condition, it is interesting to investigate the incidence of massive collapse after certain types of operations.

OPERATION:

In reviewing the literature I find that the majority of the contributors seem to think that the incidence of massive collapse is higher from appendectomies and herniotomies than any other operation although, as Bowen
states, that because this is the observation made by several men, it does not follow that this would be any convincing argument for the reflex theory.

Scott and Joelso have done much to clear the atmosphere by pointing out that in some cases at least these operations entail a naturally selected post operative posture and that this posture may be one of the causes of contra lateral collapse.

Massive collapse has occurred from so trivial and remote an operation as circumcision-50- and despite the oft repeated contrary statement, it has occurred after tonsillectomy-60-and there is still some doubt as to whether massive collapse is not one of the antecedents of post tonsillar lung abscess. Perhaps the reason that massive collapse has not been seen oftener in post tonsilectomized patients is because there has been no systematic roentgen examination or a study of the premonitory signs and onset of post tonsillar lung abscess made.

ANAESTHETICS:

That massive collapse has occurred quite often with the use of local anaesthesia has been definitely shown by Elwyn-11-and also Bowen-5-states that massive collapse has occurred where no anaesthetic has been used. However, regardless of these findings, it does not absolve ether as being a suspicious etiological factor. Bowen-5-, Mastics, Spittler and McNamee-40-have watched the administration of ether over a period of years, and they all agree that time and time again they have observed profuse secretion and signs of respiratory irritation. The conclusions reached by Bowen-5-seem to be quite fair; (1) ether per se is probably not a direct cause of pulmonary inflammation; and (2) respiratory troubles during anaesthesia, whether due to faulty administration,
unsuitable method, surgical manipulation or the quantity of the anaesthetic itself, favor the aspiration of saliva, etc. into the lungs; and that, in this way, the anaesthetic may become an important contributing cause of lung trouble. At any rate, ether, since it is so generally used, must be considered as a potential factor in the etiology of massive collapse although it perhaps could be spoken better of as a contributory factor rather than inciting.

MORPHINE AND ATROPINE:

The routine giving of morphine $\frac{1}{4}$ and atropine sulphate $\frac{1}{20}$ pre-operatively and morphine $\frac{1}{4}$ post operatively has been in vogue for such a long time, that any criticism offered against the use of these drugs would at once meet with unfavorable opinion. Nevertheless, Bowen-5-states the pre-anaesthetic use of morphine and atropine has a real etiologic relation to the occurrence of massive collapse.

Jackson-61-62-speaks of cough as the watch dog of the respiratory system. "There are certain circumstances in which the normal agencies are inefficient in ridding the bronchial system of excess secretion. Various drugs, and especially antibiotics, hinder the action of the normal agencies and hence should always be avoided. Bowen-5-has always opposed their use in all laryngical and tracheal surgery and in bronchoscopy.

It is a question whether or not many of the post anaesthetic pneumonias in surgery remote have not been the result of abolition of those agencies by which secretions are normally removed from the air passages.

The literature affords the scantiest of information, and the practice of giving morphine and atropine is all but universal. Lord-13-says, "Probably administration of morphine after operation favors collapse by diminish-
ing the cough." Hewlett-64-says, "Morphine and other opiates are usually to be avoided during bronchial obstruction and atelectasis. They decrease the tendency to cough." Pasteur-65-states that since the practice of giving an injection of morphine and atropine before anaesthesia has become general at the Middlesex Hospital, there has been a striking disappearance of respiratory difficulties and aspiration during anaesthesia and a corresponding diminution of post operative bronchial complications after major operations.

Mason-66-thinks that morphine should be given to control pain incident to the operation, and Gwyn-67-says that in the majority of cases the use of morphine in large doses is imperative.

In spite of all the opposition, Bowen-5-still believes that morphine and atropine have a real etiologic relation to the occurrence of massive collapse.

ETIOLOGY:

As mentioned before the subject of massive collapse of the lung has been attacked from almost every angle with the hope of clearing up its etiological factor or factors. In the last ten years the literature has increased three-fold, and the subject now becomes one of especial interest to every doctor regardless of whether he be a surgeon, internist or a research worker. It is definitely recognized now as a clinical entity. There are no diverse views as to the symptomology, diagnosis and treatment, but when the etiology of massive collapse of the lung is mentioned, there immediately arise many different theories and diverse views.

In reviewing the literature I find that there are about seven different theories advanced as to the etiology of massive collapse of the lung.
Each theory is based on experimental studies, clinical cases and autopsies; the information derived by such means is brought forth by the supporters of the various theories as proof.

The first theory that must be considered, of course, is that of Pasteur. His theory is that of paralysis, i.e. collapse of the lung as due to pressure exerted by the collapsed chest wall and to the elevation of the diaphragm, which is brought about by a paralysis of these structures. The resulting sluggishness of respiration, resulting from the paralysis plus the collection of secretion in the bronchioles, is the first factor in the production of massive collapse of the lung. The deciding factor as to whether there will be massive collapse or not is that the air remaining distal to the obstruction must be absorbed by the circulating blood stream. The primary cause is the pressure of the chest wall and diaphragm and the lung collapse is secondary. The paralysis theory which was advanced by Pasteur was the result of his early work with post diptheritic paralysis. He found many cases of bilateral and a few unilateral cases of atelectasis. When a few years later he began to study post operative complications, he still retained his theory of paralysis of the diaphragm. Pasteur was never very satisfied with his own theory, but, since he could not find a better one, he was content with his own.

The original contention of Pasteur that massive collapse of the lung was due to the pressure of the depressed chest wall brought about by paralysis of the diaphragm and accessory muscles of respiration is criticised by Sante-45—because it is a well known fact that in patients suffering from pulmonary tuberculosis, phrenioctomy as a therapeutic measure does not bring about massive collapse of the lung; therefore, some other factor must have been instrumental in bringing about this condition. Elliot, Dingley and Briscoe have
cut the phrenic nerve in animal experimentation as well as other nerves which influence respiration, and they have come to the conclusion that massive collapse of the lung never results from this procedure.

Sante-45-on reporting an autopsy says that upon opening the chest of a patient who had died from massive atelectasis, the chest at once assumed a normal position and contour, the lung failing to fill the thoracic cage. The conclusion reached by this finding was that the collapse of the chest wall was secondary to the lung collapse and not the primary factor in the production of massive collapse as Pasteur thought.

Elkin measured the intra thoracic pressure in a patient suffering from massive collapse of the lung and found the negative intra pleural pressure increased. Normally the intra pleural pressure varies from 9m.m. hg. on inspiration to 2m.m. hg. on expiration. In the three cases cited by Elkin the negative pressure was increased in both chests. The largest variation was 12m.m. hg. on inspiration and 15m.m. hg. on expiration on the involved side. On the uninvolved side, 6m.m. hg. on inspiration and 8m.m. hg. on expiration. The findings of Elkin would also tend to show that the collapse of the chest wall is secondary to the lung collapse.

Fontaine and Hermann-12-tend to support Pasteur's theory by experimental work on partially denervated lungs. Three days after severing the extrinsic nerves to the left lung, one of the experimental dogs developed massive collapse. The post operative course of the dog had been uneventful and studies of the CO-2 combining power of the blood was normal as was the frequency and character of respiration.
The animal was placed in a reclining position and put on the operating table for the purpose of obtaining a specimen of blood for the estimation of the CO-2 combining power of the plasma. The dog was placed on its back and immediately it became cyanotic. Respiration ceased and next the heart ceased and the animal was pronounced dead. Roentgenograms showed elevation of the diaphragm on the left side but no displacement of the mediastinum. When the dog was autopsied; massive collapse of the entire left lung was found, as well as the lower lobe of the right lung. Microscopically and macroscopically the bronchus and bronchi contained no secretion.

Pasteur's theory says that Scrimger-44-, holds good providing that we are talking about the right variety. According to Pasteur, there are two varieties of massive collapse. First the variety in which demonstrable lesions are present, which bring about paralysis of the muscles of respiration. The second variety of massive collapse according to W Pasteur, were those cases in which collapse supervenes suddenly without any premonitory signs or evidence of respiratory paralysis, and in which the lesions are always unilateral. This group includes all of the cases, which are classified as post operative massive collapse. Pasteur's first variety is given due weight and is generally accepted upon the basis of respiratory paralysis, but his second variety of massive collapse can hardly be explained upon the same basis.

The next theory that must be discussed is that held by J. Charlton Briscoe, which is the Diaphragmatic infection theory. The supporters of this theory hold that due to the onset of inflammation affecting the muscles of the crus of the diaphragm situated behind the peritoneum, one half of the diaphragm and its synergistic and antagonistic muscles are thrown out of action. This condition is brought about by inflammation of the muscle
or the pleural membrane covering it. This inflammatory process results in a disturbance of respiration which in turn leads to massive collapse of the lung.

In criticising Pasteur's theory of diaphragmatic paralysis Briscoe-47-says that the symptoms and signs of the syndrome known as diaphragmatic paralysis are, therefore, to be attributed to an exaggeration of the normal phenomenon of breathing when supine, such exaggeration being due to prolonged maintenance of that posture owing to general weakness and toxaemia. The normal inspiratory action of the diaphragm in the supine posture is carried out by the contraction of the crura, the costal portion being in abeyance. The inactivity of the costal portion is greater according to the approach of the individual to the type of breathing designated as crural, and the tone of this part of the muscle will be diminished in accordance with the degree of toxaemias and the weakness of the individual. The phenomena of the post operative collapse attack are produced by the onset of inflammation affecting the pleura covering the diaphragm, especially that part in the region of the crura, or affecting the muscle of the crus situated behind the peritoneum in an individual in whom the lower lobes are already deflated.

Briscoe's basis for this assumption of infection of the crura of the diaphragm as being the predominating factor in the etiology of massive collapse of the lung lies in the detection at autopsy of a thick, fibrinous exudate over the pleurae of the diaphragm. A similar observation was made by Pasteur who found the thick, fibrinous exudate on the pleura and on the diaphragm of patients who had died from diphtheritic paralysis. This finding has also been confirmed by Sante-45-in one of the cases which he presented. However, in the case presented by Sante-45-there was the history of a previous infection. In no
case presented by Sante-45—which occurred suddenly was there any of the thick, fibrinous exudate found at autopsy.

From a comparative standpoint it will be seen that there is not a whole lot of difference between the theory put forward by Pasteur and that held by Briscoe. Whereas Pasteur attributes the condition of the lungs found in diphtheria to paralysis of the diaphragm, Briscoe considers that it is the result of a natural condition in which the costal portion of the diaphragm is in abeyance, the crural portion alone contracting. This type of breathing occurs in individuals who have a particular form of chest. The post operative collapse attack is regarded by Pasteur as due to a reflex arrest of one half of the diaphragm, whereas Briscoe's contention is that one half of the diaphragm and its synergistic and antagonistic muscles are out of action owing to the inflammation of the muscle or of the pleural membrane covering it, and generally where Pasteur invokes inspiration as most important, Briscoe is lead to emphasize the importance of posture and alterations in the action of muscles accessory to the diaphragm.

This theory as held by Briscoe is hardly tenable because if true (Sante-45-) that a lack of synchronism between the diaphragmatic movements brought about by a retroperitoneal infection of the crus, of one part or the other part of the diaphragm it would only explain the abdominal cases and postoperative cases where there had been an infection. Also it would be hard to explain massive collapse following trauma and fractures of the pelvis, falls upon the buttocks and so forth (Elwyn-11-). It would be rather difficult to try and explain massive collapse upon Briscoe's theory, the rapidity of infection where the condition of massive collapse comes on quickly as the result of trauma (gun shot wounds). Sante-I8-, Bergamini and Shephard-24-,
have reported several cases in which massive collapse occurred while the patient was still on the operating table. Such evidence, it would seem to me, would rule out infection of the crus of the diaphragm as being the etiological factor in the production of massive collapse of the lung.

The conclusion reached as to this theory advanced by Briscoe must be that although cases have been found to have the fibrinous exudate present on the muscles of the diaphragm or upon the pleura covering them at the time of autopsy, it is the result of an established infection, and, further, that in cases where massive collapse occurs very suddenly there surely is not sufficient time for a primary infection to bring about a condition of this nature.

The spasmodic reflex theory has been proposed by Sir John Rose Bradford as a probable etiological factor in the production of massive collapse of the lung.

This theory holds that there is a reflex spasmodic contraction of the bronchioles which causes them to become occluded, and combined with a subsequent absorption of alveolar air by the circulatory system, a condition of collapse of the lung occurs. There is ample experimental data to be found of the existence of constrictor nerves and muscle fibres of the bronchioles sufficiently strong to produce complete and prolonged constriction of the bronchioles. Auer and Lewis in working with anaphylaxis in the guinea pig have definitely proven the existence of constrictor nerves and muscle fibres of the bronchioles sufficiently strong to bring about constriction of the bronchioles.

Sir John Rose Bradford in 1918 presented his views on massive collapse of the lung as a result of gun shot wounds with special reference to the chest. "When massive collapse of the lung is present, the chest on the affected
side is immobile, and the causation would seem to center around the significance of the immobility." The question to be answered is: Is the chest immobile because the lung is collapsed or is the lung collapsed because of the immobility of the chest wall? If the collapse is primarily of lung origin, the only obvious mode of production would seem to be that of obstruction of the bronchi. Scott in the Archives of Surgery 1925-68-concludes from the work of Bradford that there must be some reflex which causes a constriction of the air passages, probably affecting the bronchioles and not dependent originally an infection, and acting on both lungs to some extent.

Bradford-43-, in conclusion, states that the mode of production of massive collapse of the lung is obscure. Theoretically, it might ensue either as a result of bronchial obstruction or as a sequel to impairment of the efficiency of the respiratory movements. This in turn might result from a definite respiratory paralysis as in paralysis of the diaphragm in diphtheritic neuritis or possibly from a temporary paralysis of the inspiratory mechanism of reflex origin. There are difficulties in accepting either of these views.

That there is often no clinical or post mortem evidence of bronchial obstruction makes it particularly difficult to explain the frequent absence of symptoms if the obstruction is of a bronchial origin. On the other hand, it is not easy to understand why a comparatively trivial injury on one side of the chest should produce a reflex palsy of the respiratory muscles on the opposite side of the chest. On the whole, suggests Bradford, it would seem that the best explanation would be that of reflex constriction of the bronchioles.

That constriction of the bronchioles such as presented in this theory ever leads to atelectasis has never been proven.—Sante-45. One would be
lead to believe from the frequency with which atelectasis follows abdominal operations that perhaps the condition is brought about by insult to the vagus nerve, but Briscoe-47-performed numerous experiments on animals in order to try to produce this reflex spasm. He irritated the vagus directly as well as the peritoneum but failed to get the reflex spasm.

It would appear that there is very little support for this theory that reflex bronchial spasm is the cause of this condition, although there is far more than enough to show that the bronchial musculature is sufficiently strong to cause bronchial contraction and thus shut off the bronchi.

Considering next the angioneurotic theory: This theory is supported in the main by Bergamini and Shephard and also by Gwyn and Scott to a lesser extent.

The angioneurotic theory is based upon the fact that the condition of massive collapse is usually of a rapid development and that often there is found dilatation and engorgement of the blood vessels. This rapidity of development and the finding of engorgement and dilatation of the capillaries suggests the possibility of a vaso motor disturbance as being the etiological factor in the production of massive collapse of the lung. Bergamini and Shephard-24-state that the uniform dilatation of the capillaries, venules and arterioles and the rapidity of the condition would tend to rule out bronchial obstruction as the causative factor and favor the idea of a vaso motor disturbance.

Bergamini and Shephard-24-report two cases where sudden death occurred during operation, and at autopsy massive collapse was found to be present.

The first case was a married white female forty-four years of age
admitted to the Bellevue Hospital complaining of pains in the lower abdomen. There was also the presence of a bloody watery vaginal discharge. There was a history of irregular menses previous to entrance into the hospital. A diagnosis of fibromyomata was made. Physical examination at the time of entrance proved negative with exception of tenderness in the lower part of the abdomen. Heart and lungs were negative. Three days after entrance a supra vaginal hysterectomy and appendectom was done. As the last sutures were being made, it was noticed that the patient had stopped breathing. All attempts at resuscitation failed, and the patient was pronounced dead. Ether anaesthesia had been used, and the patient was reported in good condition until the close of the operation.

The patient was autopsied, and the left lung was found to be collapsed completely; the right lung was partially collapsed. There was moderate engorgement. No aspirated food was found in the bronchi. The tongue was normal. A fairly well marked laryngeal oedema was found.

Histological examination showed a tissue which was solid, epithelial cells packed closely together, the alveolar arrangement being entirely lost. The individual cells were swollen and hydropic, and the cell outlines were rather indistinct. Capillaries, arterioles and venules were all uniformly dilated and filled with blood.

The second case which Bergamini and Shephard present in support of the angioneurotic theory is that of a man aged 69 who was admitted into the Bellevue Hospital with a chief complaint of pain and swelling in the left hypochondrium. Physical examination showed the presence of an abdominal tumor in the region of the spleen. The rest of the physical examination was essentially negative. A clinical diagnosis of "Bantis Disease" was made. An operation was
decided upon; ether anaesthesia was to be used; the patient was in good physical health with the exception of his complaint. The operation was started, a left rectus incision being made. A large spleen was found, which was removed. The wound was closed and saline clysis and transfusion under way as the operation ended. The total time of the operation was sixty-five minutes. Suddenly, as the patient was to be taken to the ward, he stopped breathing. Resuscitation was of no avail, and the patient died. The pulse was perceptible ten minutes after cessation of respiration. The autopsy findings on this patient were as follows: When the peritoneal cavity was opened, 50 c.c. of free blood was found. The diaphragm was found to be at the upper border of the fourth rib. No defect was found in the diaphragm. No free pleural fluid was found. Both lungs were found to be completely collapsed and were found to be lying close to the vertebrae. Each lung weighed about 50 gms. The only crepitant part of the lung was the apices of each, the rest of the lung tissue being solid and non-crepitant.

Histological section showed tissue firm and homogeneous and similar in appearance to the histological findings in the previous case. The pulmonary vessels were empty, and no secretion was found within the bronchi.

Bergamini and Shephard have presented two cases wherein death occurred during operation. Autopsy findings showed that massive collapse was present. These two cases show with what rapidity massive collapse of the lung may occur. The chest signs were perfectly normal before operation and throughout the two operations respiration was absolutely natural. There was not the slightest indication of respiratory embarrassment; in fact, the patient in the first case was in such a good condition that the anaesthetist had left her to start another anaesthesia when the patient suddenly stopped breathing. It would seem
that this extreme rapidity would in itself rule out bronchial obstruction with subsequent absorption of alveolar air. Further, the gross examination of the collapsed lungs gave no evidence of bronchial obstruction, and no gross particles were seen in the bronchi. Elliot, Dingley and Churchill emphasize very strongly the presence of muco purulent secretion in the bronchi in the cases of massive collapse, but this was not found in the two cases of Bergamini and Shephard. The lung tissue was not oedematous, and did not contain any excess fluid.

To repeat, these two cases strongly suggest a vaso motor disturbance; the rapidity and autopsy findings, coupled with the histological hydropic appearance of the epithelial cells lining the alveoli and bronchioles, gives the impression of interstitial oedema; and, in conclusion, Bergamini and Shephard ask the question as to whether or not the condition observed in these two cases might not be closely related to angio-neurotic oedema.

The pathological autopsy findings of Lee and Paul do not seem to bear out the autopsy report of Bergamini and Shephard. The case was reported by Hoge of Pennsylvania Hospital and was the fatal result of an unrecognized massive collapse. In this case the lower left lobe was pitch black, a picture of complete atelectasis. Compression of the tissue produced an extrusion of thick, muco purulent pus from the bronchioles. Tenacious, purulent mucous filled the entire bronchi leading to the lower lobe. Bergamini and Shephard did not find any of the muco purulent secretion in the bronchi.

The findings of this autopsy do, however, agree as to the histological appearance of the lung tissue. Lee and Paul state that there was a marked dilatation of the alveolar capillaries, all the smaller vessels of the
of the lung being congested with distended red blood clots, the antithesis of pulmonary consolidation.

The angio-neurotic theory of massive collapse of the lung is not generally accepted, because the majority of the clinicians and research workers have found that in the vast number of cases of massive collapse of the lung the findings of Bergamini and Shephard in their two cases are not present. The rapidity of massive collapse and the engorgement of the capillaries, venules and arterioles as suggested by Bergamini and Shephard in their two cases might just as well be present in cases of massive collapse where bronchial obstruction had been definitely ascertained. The only thing that lends credence to the hypothesis of the angio-neurotic theory is the autopsy findings in which no obstruction was found in the bronchi.

Coryllos and Birnbaum-10-state that the existence of an angio-neurotic oedema of the lung in cases of massive atelectasis has never been proved either experimentally or clinically. In the cases of acute anaphylactic shock in the guinea pig emphysema and not collapse is produced even when oedema occurs.

Scott and Joelson-68-are the supporters of the posture theory. Their contention is that massive collapse of the lung is due to the position which the patient assumes during post operative convalescence. These two research workers cite an unusual case which is as follows:

History. J. M., a Greek aged 24, was admitted to the Lakeside Hospital on February 22, 1925, complaining of abdominal pain. His family history was good. He had never had any respiratory symptoms or distress. The present illness started ten years before when the patient began to have recurring
attacks of abdominal pain, which extended to either or both lumbar regions and
to the back. The pain was severe and colicky and would usually last for several
hours. In addition to the pain the patient also had frequency of micturition
and nocturia. Hematuria did not appear until about six years after the onset of
the attacks of pain but it had recurred frequently since then. At the onset of
the present illness the attacks came on about once every three months, but they
became steadily more severe and more frequent.

Physical examination was essentially negative with exception of
slight tenderness in the left lumbar region.

On February 28 a left pyelotomy was done under nitrous oxide,
oxygen and ether anaesthesia. The patient was placed on his right side, and the
usual position for an operation on the kidney was obtained by means of a kidney
elevator. The operation was completed in one hour and twenty minutes. The time
of the anaesthesia was two hours. There was nothing unusual about the anaesthesia;
the patient's condition and color remained good throughout the procedure. The
patient made a good immediate recovery from the operation. Toward evening, how­
ever, the patient's temperature went up to 39.8 C., pulse 112, with respiration
remaining at 22 per minute. The next morning the patient presented the classic
picture of post operative atelectasis of the right lung. Temperature was 39.4 C.,
pulse rate of 140 and respiration 40. Physical findings of atelectasis were
present. X-ray confirmed a diagnosis of post operative massive atelectasis of
the right lung. Three weeks after the operation the condition of the chest had
returned to normal. The patient was discharged from the hospital and told to re­
turn in about two months for removal of the calculus in the right kidney.

The patient was re-admitted to the hospital on May 14th for
pyelotomy on the right side. He had been well except for one attack of renal colic on the right side. He had not had any respiratory symptoms, and physical examination of the chest did not disclose any abnormalities of the heart or lungs. Roentgenograms of the kidneys showed the calculus in the right one to be the same as on previous examinations. A right pyelotomy was done on May 16th under nitrous oxide and ether anaesthesia. The patient was placed on his left side, and the usual position for an operation on the kidney was obtained by means of a kidney elevator. The stone was removed from the right kidney and the wound was closed in the usual way with drainage. The total duration of the anaesthesia was one hour and twenty-five minutes, whereas the operation itself lasted for one hour. The anaesthesia was taken well, and the patient's color and condition remained good throughout the entire procedure.

On May 17th, (the first day after operation) the patient's temperature rose to 39.6 C., pulse rate of 120 and respirations 40; and a slight cough was present. On the next day the patient showed evidence of a post operative atelectasis of the left lung. Roentgenograms confirmed the diagnosis. The symptoms and signs gradually disappeared, and nineteen days after the operation the patient's temperature, pulse rate and respirations were normal. The patient was discharged on June 10 (twenty-five days after operation) feeling well and in good condition.

There are several points of interest in this case, and the most obvious fact is that, following separate operation on each kidney, the patient developed massive atelectasis of the opposite lung. Not only during the operation on the kidney was the position of the patient such that the contra-lateral lung was dependent, but the lumbar wound resulted in his remaining at least partially
turned on the opposite side during the immediate post operative interval, in which massive atelectasis developed.

In order to determine whether there might be a relationship between posture and the occurrence of massive atelectasis, Scott and Jaelson present twenty-two cases of massive atelectasis, analyzed for the factor of posture.

In the majority of cases, the character of the operation did not necessitate lying on one side. In two instances, however, this condition was fulfilled. These two cases, however, were the only ones that were observed in which massive atelectasis developed after an operation which required the patient to be on one side.

Scott and Jaelson present four instances in which massive atelectasis has occurred in the contra-lateral lung from the wound. A review of the literature by Scott and Jaelson revealed five more cases which were similar. Altogether, then, nine cases have developed atelectasis following operations that necessitated the patient's lying on his side. In each instance the collapse occurred in the dependent lung. The evidence that this is not merely coincidence is stronger for two facts: (1) in seven of the nine cases the atelectasis occurred on the side contra lateral to the operation, whereas the homo lateral lung is ordinarily the one involved; (2) in the majority, the collapse was on the left side, which is the side much less commonly involved in post operative cases.

The conclusion thus reached by Scott and Jaelson is that when the patient has been consistently lying on one side, if unilateral massive atelectasis develops, it will occur in the dependent lung.

The posture of the patient under such circumstances appears to
determine the localization of the complication. However, it certainly is not the primary cause of atelectasis. The fact that atelectasis follows only a small minority of the operations on the kidney is ample evidence that there is a more fundamental factor than the position alone. Scott and Jaelson favor the view that the fundamental condition which initiates massive atelectasis is a nervous reflex which affects both lungs and probably occurs frequently in a minor degree.

We are now ready to discuss the bronchial obstruction theory of massive collapse of the lung. This theory is supported by Jackson and Lee in the main and also by Coryllos, Birnbaum and Churchill, with reservations.

The theory is that obstruction of the bronchioles by secretions is the entire explanation, such a condition being all that is necessary for the development of the other features of this condition. Any disturbance of the respiratory function is purely incidental and is not the primary factor. Long before this theory was advanced by Jackson and Lee it was known that when a foreign body completely occluded the bronchi, the portion of the lung supplied by the obstructed bronchus became atelectatic. Mendelssohn and Traube in 1845, doing experimental work on animals showed that when the bronchus was occluded by packing paper wads, shot and gum arabic into it, atelectasis followed.

Lichtheim in 1879 produced experimental proof of the existence of atelectasis after plugging a bronchus with a stick of laminaria. This experiment was performed upon rabbits. Lichtheim's observations and conclusions were that atelectasis took place when the bronchus was occluded providing the pulmonary circulation was left undisturbed. Lichtheim showed conclusively that if the pulmonary vessels were ligated, the residual air left distal to the obstruction
was not absorbed, and atelectasis did not occur. Similar observations have been made by Chevalier Jackson working upon foreign body atelectasis in human beings.

Jackson-15-and Lee recognize acute massive collapse as a clinical entity, and probably the result of bronchial obstruction primarily and the embarrassment of the respiratory mechanism incidentally. The embarrassment of the respiratory mechanism may be either the result of posture after the operation during convalescence, voluntary or reflex inhibition from post operative pain of abdominal operations or even the result of paralysis which occurs in diphtheria. The nature of the obstructing agent is said by Jackson and Lee to be bronchial secretion, foreign bodies or extra bronchial pressure produced by a tumor.

At the Bronchoscopic Clinic in Philadelphia, says Jackson, hundreds of cases of atelectasis affecting one or more lobes and often the entire lung have been seen. Usually the etiology is bronchial obstruction, but all bronchial obstructions do not produce atelectasis, some of the bronchial obstructions producing exactly the opposite condition, namely emphysema.

According to Chevalier Jackson-15-there are three types of bronchial obstruction: (1) the "by pass valve", (2) the "check valve" obstruction, (3) the "stop valve" obstruction.

The by pass valve obstruction is the type of partial obstruction which allows or permits ingress and egress of air past the obstruction.

The check valve obstruction allows the air to pass in but not to emerge. This is the type of obstruction that brings about emphysema. Dr. Jackson states that he has seen hundreds of these cases. The condition was first observed in a patient who had a peanut kernel in the main bronchus. Iglauer observed the condition first. Obstructive emphysema has also been seen in cases
of swollen bronchial mucosae and in cases of plugs of tough secretion. Aspiration of plugs from a diphtheritic membrane has also been reported.

The mechanism of the check valve obstruction is the normal enlargement of the diameters of the bronchial lumen during inspiration which opens a space for passage of air around the obstructive mass, this space being promptly obliterated by the normal expiratory diminution of the bronchial diameter. The pathological factor is supplied not only by the bulk of the plug of mucous or other foreign body, but by the swollen mucosae of the bronchial wall, which makes a soft cushion-like a valve seat. The reverse of this check valve obstruction, i.e. where air can get out but cannot get in again is rare because the lumen of the bronchi diminishes upon expiration.

Jackson's third type of valvular obstruction is the stop valve. In this type of obstruction air can neither enter past the obstruction nor get out. As a consequence of this total obstruction, the residual air left distal to the obstructive agency is absorbed by the pulmonary circulation, and the tributary lung collapses.

Golden-48-in studying the effect of bronchostenosis upon the roentgen ray shadows in Ca. of the bronchus, shows by a group of cases that when complete occlusion of the bronchus occurs, there is atelectasis of the corresponding part of the lung because the air remaining distal to the obstruction is quickly absorbed by the blood stream. When, however, the obstruction is incomplete, the bronchi distal to the obstruction become dilated; therefore, in the gradual developing stenosis of a bronchial carcinoma, the condition is first that of incomplete obstruction, and bronchiectasis follows with infection in its train. When the occlusion becomes more marked, atelectasis with its
ensuing fibrous changes complicates the picture. As a result of the atelectasis there is a marked thickening of the pleura.

In conclusion, Golden says that as far as physical effect is concerned, bronchial carcinoma may be considered as developing in two stages: (1) stage of invasion; (2) stage of broncho-stenosis, which is an incomplete occlusion or obstruction of the bronchi, and, due to the dilatation of the bronchi plus infection, bronchiectasis results. The third stage of complete bronchial obstruction is where the picture of massive collapse manifests itself.

That complete obstruction is necessary before atelectasis will follow has been quite definitely shown by Manges-49 and by Coryllos and Birnbaum-14. Jackson's observation of obstructive emphysema is the basis for Manges'-49-diagnostic sign for non opaque foreign bodies in the lung. Manges-49 presents a number of cases of foreign body in the bronchus, and he finds that the most constant diagnostic sign of foreign body of the bronchus is obstructive emphysema. Upon full inspiration the aeration of the lungs in the majority of cases is good, but upon expiration evidence of obstructive emphysema is striking in the affected lung.

Atelectasis has been present in a few of Dr. Jackson's cases in which the foreign body was also opaque. When the foreign body is definitely proven to be present, atelectasis is diagnostic of foreign body, as well as to its location.

The excellent experimental work of Coryllos and Birnbaum-13-in which a bronchus was obstructed by using balloons of varying size, introduced into the bronchi of dogs by the use of the bronchoscope and then filled with opaque sodium bromide so that x-ray could be taken serves to confirm previous observations and establish beyond a doubt the relationship between complete
bronchial obstruction and atelectasis. But, as Sante-45-says, "It leads no
closer to a solution of the problems in spontaneous massive collapse of the lung
when no evidence of bronchial obstruction produced by a foreign body can be
found."

The question will voluntarily arise that aspiration of secretions
from the mouth could produce the obstruction by acting as a foreign body, but in
a review of the literature Sante-45-says that patients aspirating barium sul-
phate through a broncho-esophageal fistulae, never develop atelectasis. Sante-45
makes the statement that no case of massive atelectasis is reported after
tonsillectomy. This is the opposite finding from what Solem-60-found. However,
the cases of massive collapse that occurred after tonsillectomies were not backed
by autopsy findings.

That it would be possible for this condition (atelectasis) to
result from thick fluids within the bronchi is possible. Hickey in discussing a
recent communication by Dr. Preston B. Forestier, "In Hickey's patient" massive
collapse followed the intra tracheal injection of iodized oil used for diagnostic
purposes. Massive collapse has also followed the injection of lipiodol into the
bronchi. Jacoboeus-23-presents four cases of massive collapse as produced by
lipiodol injections.

Hearn and Clerf-14-present a clinical case of massive collapse.
A girl eight years of age was admitted to the Pennsylvania General Hospital for
the purpose of closing a fistula, which had been present for six years as the
result of a gastrostomy, which had been performed because of stricture of the
oesophagus. This case developed massive collapse of the left lung. There was
no doubt that bronchial obstruction was present in this case and that the
obstruction consisted of thick, tenacious secretion.

Bronchoscopic removal of the obstruction was followed by a partial return of pulmonary function. Hearn and Clerf advise bronchoscopic examination and removal of the obstructive secretion in cases of post operative pulmonary collapse.

Lee, Tucker and Clerf-16-present a case of massive post operative pulmonary atelectasis with a follow-up experiment on a dog which has real value in explaining the "Etiology of Massive Collapse". A patient was operated on for right inguinal herniae under ether anaesthesia. During the administration of the anaesthesia, the anaesthetist noticed that there was more mucous in the respiratory tract than usual. The operation was well stood by the patient, and no symptoms developed until twenty-four hours after the operation, at which time the patient exhibited distress in breathing. It was quite evident that breathing was voluntarily restrained because of pain in the operative wound. There was a beginning rise in temperature and some mid-sternal pain. Forty-two hours after the operation there was more severe respiratory distress, the cough was weak and shallow, and there was a slight production of a thick, tenacious sputum.

Physical examination showed heart displaced to the left and dullness over the left lung. The X-ray showed the mediastinum displaced to the left. The patient was bronchosceded, and 9 c.c. of a thick, tenacious sputum was drained from the left main bronchus. Bacteriologically the secretion gave a pure culture of pneumococcus. Following bronchoscopy the patient had immediate relief.

In order to follow up the etiology of this case it was decided to subject a dog to a similar operative wound, narcotize the cough by injection of
sodium amytol intra-peritoneally, and by use of a bronchoscope 7 c.c of the thick, tenacious secretion was injected (which previously was bronchoscooped from the patient) into the right main bronchus of the dog. In a few minutes after the completion of the experiment, the animal developed a severe respiratory distress. Movements of the right side of the chest were soon lost while there was exaggeration on the left side of the chest. The dog was kept on his right side for three hours, and at the end of that time roentgen ray examination showed that there was complete massive atelectasis of all the lobes of the right lung with a definite transposition of the heart to the right beyond the spine.

According to Lee, Tucker and Clerf this is the first successful case in which bronchial secretion taken from a patient presenting the clinical signs of massive collapse (post operative) has been used to produce the same condition in an animal. This experiment proves definitely that massive collapse does occur as a result of bronchial occlusion by secretions. The reason for the excess of secretions in the bronchioles after a major operation still remains a matter of conjecture.

Churchill differs from Lee, Coryllos, Birnbaum and Jackson in his theory of massive atelectasis in that he stresses impaired respiration as the primary factor and bronchial obstruction as secondary. Churchill-7-said in 1925 that massive collapse was the continuation of two things; first, weakened respiratory force; and secondly, bronchial obstruction. That these two factors play roles of varying importance in different cases is probably true; thus extreme debility plus even normal bronchial moisture may result in areas of atelectasis, and conversely a foreign body may cause collapse with normal respiration. In most operative cases the collapse is the result of the combined action
of the two factors.

A consideration of the facts shows that there is very little clinical or experimental evidence for thinking that massive collapse can occur under any other condition than by actual mechanical bronchial obstruction. Assuming that bronchial obstruction is necessary for the production of massive collapse, then the manner in which the obstruction is brought about alone remains to be considered.

The question might be asked, "What extraneous factors or influences are there which could account for the accumulation of secretion in the larger bronchi?"

Sante-45-states that the loss of sensation of the trachea and larger bronchi with a consequent interference with the expulsive mechanism which is normally present (cough, reflex and ciliary action of the mucous membrane) might explain the developing atelectasis. In order to accept this idea it would be necessary to delve deeper into it and try to find the cause of the loss of sensation to the trachea and larger bronchi. Those cases that are definitely infectious might be explained in this manner as there is abundant evidence that extreme toxicity can abolish the tracheal reflex. An example of this is seen in those cases where individuals toxic from intestinal obstruction or septicemia aspirate barium sulphate into the lungs without any apparent discomfort. Sante-50-in Case I of his series confirms this statement.

In operative cases where massive collapse most frequently occurs, the question will be asked, "What abolishes the tracheal reflex in these cases especially where the abolition of the tracheal reflex cannot be ascribed as being due to toxicity. A possible explanation might be the use of morphine to quiet
the patient, especially so if the patient were allowed to remain in one position for an extended time after the operation.

The pre-operative administration of morphine has been offered as a possible explanation of post operative abscess of the lung, the analgesia produced presenting the normal expulsion of infected material aspirated at the time of operation. This idea would explain contralateral involvement which is occasionally encountered in post operative atelectasis as seen in Case III of Leroy Sante's-50-series.

POST OPERATIVE PNEUMONIA - RELATION TO MASSIVE COLLAPSE:

The various theories as to the etiology of massive collapse have now been discussed, and it will now be advisable to present a brief survey of the relationship which exists between what is commonly termed post operative pneumonia and clinical post operative atelectasis. There is no doubt but what many of the post operative complications that are termed post operative pneumonia are nothing more nor less than massive atelectasis of the lung.

Elwyn in 1924 proceeded with a course of research to try to find out (1) incidence of pneumonia following operations under general anaesthetic; (2) the etiologic factors concerned in its production. The conclusions that Dr. Elwyn reached are herewith presented: (1) Post operative pneumonia occurs frequently, possibly more frequently than is thought, upon operations involving the gastro intestinal tract. On other parts of the body the frequency is less than one percent. (2) The etiologic factors concerned in the production of post operative pneumonia are the presence of infection in the respiratory tract at the time of operation; hypostatic congestion in old and weakened persons; and irritation of the respiratory tract by the too liberal use of anaesthetic.
As to the mode of production, aside from the occasional production of pneumonia by the aspiration of contents from the mouth and pharynx, Dr. Elwyn thinks that the post operative complication is due to an area or areas of atelectasis or collapse, usually in the lower lobes. When a bronchitis has been present before operation or results from the irritation produced by the anaesthetic, the infection spreads to the atelectatic area and forms a locus minoris resistentiae, and due to this the condition known as post operative pneumonia develops.

Churchill-7 does not seem to agree with the hypothesis as set forward by Elwyn, that an atelectasis is a locus minoris resistentiae for the development of pneumonia. He states that is hard to understand why the whole rationale of treatment of tuberculosis is benefited by pneumothorax. The pneumothorax favors a limitation of the infectious process and promotes healing of the diseased lung.

Elwyn presents some interesting statistics in connection with post operative pneumonia and a study of these statistics reveals the similarity existing between post operative pneumonia and massive collapse of the lung.

Table I. Incidence of pneumonia following operations under general anaesthesia, and the organs or regions of the body involved.

<table>
<thead>
<tr>
<th></th>
<th>No of Operations</th>
<th>No Cases Pneumonia</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hernia</td>
<td>200</td>
<td>9</td>
<td>4.5</td>
</tr>
<tr>
<td>Appendix</td>
<td>309</td>
<td>10</td>
<td>3.2</td>
</tr>
<tr>
<td>Gall Bladder</td>
<td>114</td>
<td>10</td>
<td>8.7</td>
</tr>
<tr>
<td>Stomach</td>
<td>63</td>
<td>10</td>
<td>14.2</td>
</tr>
<tr>
<td>Exp. Laparotomy</td>
<td>55</td>
<td>4</td>
<td>7.2</td>
</tr>
<tr>
<td>Small Intestine</td>
<td>15</td>
<td>1</td>
<td>6.6</td>
</tr>
<tr>
<td>Colon &amp; Rectum</td>
<td>35</td>
<td>2</td>
<td>5.7</td>
</tr>
<tr>
<td>Operation</td>
<td>No of Operations</td>
<td>No Cases Pneumonia</td>
<td>Percentage</td>
</tr>
<tr>
<td>------------------</td>
<td>------------------</td>
<td>-------------------</td>
<td>------------</td>
</tr>
<tr>
<td>Bladder</td>
<td>23</td>
<td>1</td>
<td>4.3</td>
</tr>
<tr>
<td>Kidney</td>
<td>72</td>
<td>1</td>
<td>1.3</td>
</tr>
<tr>
<td>Spleen</td>
<td>7</td>
<td>2</td>
<td>28.5</td>
</tr>
<tr>
<td>Gyn Operations</td>
<td>358</td>
<td>2</td>
<td>0.55</td>
</tr>
<tr>
<td>Extremities</td>
<td>182</td>
<td>1</td>
<td>0.75</td>
</tr>
</tbody>
</table>

From this group of statistics it will be seen that the incidence of post operative pneumonia after operations on the appendix and after repair of herniae is very similar to the incidence of post operative massive collapse after operations on these two regions.

Table No. II. Incidence of pneumonia following operations under local anaesthesia and the organs or regions of the body involved.

<table>
<thead>
<tr>
<th>Operation</th>
<th>No of Operations</th>
<th>No Cases Pneumonia</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hernia</td>
<td>37</td>
<td>3</td>
<td>8.1</td>
</tr>
<tr>
<td>Appendix</td>
<td>4</td>
<td>1</td>
<td>8.1</td>
</tr>
<tr>
<td>Stomach</td>
<td>13</td>
<td>2</td>
<td>15.3</td>
</tr>
<tr>
<td>Small Intestine</td>
<td>6</td>
<td>2</td>
<td>15.3</td>
</tr>
<tr>
<td>Colon &amp; Rectum</td>
<td>3</td>
<td>1</td>
<td>15.3</td>
</tr>
<tr>
<td>Bladder</td>
<td>25</td>
<td>2</td>
<td>8.0</td>
</tr>
</tbody>
</table>

As in post operative massive atelectasis the type of anaesthesia used whether general or local does not seem to have much influence as to the frequency of post operative pneumonia.

Table No. III. Lobes involved in all cases of pneumonia following operations performed under general anaesthesia.
Right Lower Lobe  
Left Lower Lobe  
Both Lower Lobes  
Right Upper Lobe  
Left Upper & Left Lower Lobe  
Right Upper & Both Lower Lobes

An analysis of the statistics just presented will show that postoperative pneumonia and postoperative massive collapse seem to favor the right lower lobe as to site of involvement.

Table No. IV. Number of days elapsing between operation and onset of pneumonia.

<table>
<thead>
<tr>
<th>No. of Days</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
</tr>
</tbody>
</table>

The conclusion is justifiable that postoperative pneumonia seems to occur in the majority of cases during the first and second day postoperative. This is the finding also in postoperative massive collapse.

From the series of statistics presented by Dr. Elwyn on post
operative pneumonia it is very interesting to note the striking similarity to post operative massive collapse. It leads one to believe that we are dealing with the same condition under two different terms.

Coryllos and Birnbaum-10-state that the relationship between lobar pneumonia and obstructive atelectasis is suggested because it would help to explain the anatomic lobar distribution of the disease in relation to the obstruction of a bronchus by (1) the pneumonic sputum, which is so viscid and tenacious; and (2) the diminished force of respiration.

Coryllos and Birnbaum-9-doing experimental work on dogs, noticed such a striking similarity between massive atelectasis and lobar pneumonia that they decided to investigate the problem further. The fruit of their experimentation on dogs resolved itself into such conclusions as this: "That lobar pneumonia must be considered as a pneumococci atelectasis of the lung and that a simple pneumococci infection of the bronchial tree is not sufficient to produce a lobar pneumonia, but that there must be complete occlusion of a bronchus exactly as in massive atelectasis, the obstruction resulting from the tenacious, fibrinous pneumococci exudate.

The experimental work on dogs was done by the use of a small elastic balloon in which was incorporated a one-way valve by means of which the balloon could be filled with concentrated solution of sodium bromid which would be opaque to X-rays.

In considering the similarity of lobar pneumonia and massive atelectasis we may set down a few of the more important similarities:

1. Lobar distribution.
2. Rapid onset of symptoms.
3. Almost identical clinical symptoms except the more toxic element in pneumonia.

4. Rapid improvement after the release of the obstruction in atelectasis and after the crisis in pneumonia.

5. The more usual localization in the inferior lobes particularly the right.

6. X-ray findings.


Two problems confronted Coryllos and Birnbaum; namely, why is there a lobar distribution in lobar pneumonia exactly as in massive atelectasis; secondly, if lobar pneumonia is due to a similar mechanism, there should be at least at its onset signs of an atelectasis which must be revealed by X-ray.

In the literature nothing of an explanation is offered as to the lobar distribution of pneumonia. Coryllos and Birnbaum offer this explanation. Lobar pneumonia most often is the result of the pneumococcus, and this organism is the greatest producer of fibrin known. The collection of this fibrin in a large bronchus would soon become obstructive and account for the lobar distribution.

In the differential diagnosis between post operative massive collapse and post operative pneumonia a great difficulty arises. Coryllos and Birnbaum thought that shifting of the mediastinum means but one thing (and elevation of the diaphragm), namely decrease in size of the affected lung, in other words, massive atelectasis. Coryllos and Birnbaum have proved their point as to the decrease in the size of the affected lobe by using a tracheal clamp and clamping the trachea before removing the lung from the chest of a dog having lobar pneumonia.
Coryllos and Birnbaum cite a human case. A male patient aged 23 was brought into the hospital with a complaint of a slight cold, mild rhinitis, occasional cough which was slightly productive. Forty-eight hours before entrance into the hospital the patient had chills, fever, vomiting and pain in the chest (left). On physical examination the patient was found to be acutely ill, coughing up orange colored sputum.

The heart was normal, and the apex was found to be in the 5th I.S. 9 cm to the left of the sternum. The right lung was found to be normal. Percussion note of the left lung was resonant anteriorly, and dullness was found over the lower lobe posteriorly. Fremitus was increased over this area as was the whispered voice. Bronchial breath sounds were heard distinctly and a few rales were made out. No sounds were heard in the left axillae.

The patient's temperature was 103.6, pulse 94 and respirations 30. The blood count was 23,000 WBC of which 97% were polymorphonuclear leucocytes. The sputum was positive for pneumococcus type III.

Roentgenograms showed increased density of left lower lobe and showed the heart to be in the midline with elevation of the left part of the diaphragm with an accompanying deviation of the trachea to the right.

Bronchoscopy was decided upon, and one teaspoonful of the thick tenacious rusty colored exudate was recovered. All of the involvement was limited to the left lower bronchus.

Progress notes:


December 31. Consolidation signs present in left lower lobe.
General condition of patient good. T - 104. Roentgenograms show no morbid change.

January 1, 1928. Temperature falling 43 hrs. after bronchoscopy, reaching normal within 12 hrs. Crisis within less than four days after onset of disease.


If Coryllos and Birnbaum are right in their interpretation, a number of features inexplicable in lobar pneumonia may be explained, namely, (1) lobar distribution; (2) sudden onset; (3) crisis; (4) abortive forms; (5) predominance of localization in the inferior lobes; (6) the often found displacement of the heart and diaphragm to the affected side. From the possibility that both lobar pneumonia and massive atelectasis are secondary to bronchial obstruction may arise the possibility of an additional mode of treatment, i.e. a bronchoscopic treatment of pneumonias rather than medial.

SYMPTOMATOLOGY:

The symptoms of massive collapse vary greatly in severity and, on the whole, tend to be most marked at the onset of the condition, subsiding early and even disappearing provided the patient is kept in bed and at rest. Usually in the post operative variety of massive collapse the symptoms appear the first three days after operation. It is important to recognize that massive collapse may involve the whole of one lung with out the presence of any urgent symptoms.
and that the condition may be overlooked unless the chest is carefully examined, and this is more especially the case in the contra lateral variety. If such cases are not recognized the chest may be opened, pleura opened freely and death may occur suddenly.

Dyspnoea is the most constant symptom in massive collapse usually of a moderate severity, but is greatly increased upon the slightest exertion. Cough is usually slight, but often repeated and persistent, and is many times associated with a thick, tenacious sputum. Cough and expectoration is more marked in post operative cases than in cases of massive collapse after gun shot wounds. In gun shot wounds there may be a negative history of cough and expectoration. This is sometimes a valuable point in determining causation of massive collapse.

Absence of symptoms, as has been said before, is most marked in those cases where massive collapse involves the whole lung on one side or the other.

Pyrexia is not a constant symptom. The temperature is usually of a low grade variety when present. When pyrexia is present it might be due to some other lesion rather than massive collapse itself. Pyrexia is necessarily present when massive collapse is the result of a complicated pneumonia or pleurisy. Along with the symptoms already mentioned there is usually pain and lightness in the chest and profuse perspiration.

PHYSICAL SIGNS:

The patient lies on his back or on the affected side with the shoulder on the affected side drawn down. The respiration is rapid and labored. The skin is moist and cyanotic. The expression is anxious, the pulse rapid and bounding. The chest on the affected side is contracted, and there may be visible
retraction of the interspaces. Respiratory excursion is absent on the affected side. The diaphragm is high and excursion limited on the uninvolved side.

Tactile fremitus is reduced and the percussion note is flat. Breath sounds are usually absent. There is hyperexcursion of the unaffected chest, almost an emphysematous appearance. There seems to be some difference in opinion as to whether the emphysematous appearance is the same, as in case of "By valve" emphysema as described by Chevalier Jackson. There is increased respiration and increased breath sounds on the unaffected side.

As dyspnoea is the most important symptom of massive collapse, so is mediastinal displacement the most important physical and roentgen finding. The heart and mediastinum in massive collapse is always displaced to the affected side.

ROENTGEN FINDINGS:

1. Displacement of the heart and mediastinum toward the affected side.

2. Marked increase in density of the affected lung. This increased density is said to be the result of engorged blood brought about by an increase in the negative pressure outside of the blood vessels.

There has been considerable speculation as to what was the cause of mediastinal displacement in massive collapse of the lung. According to Santé-45-the lung does not leave the chest wall in massive collapse, but in its atelectatic state the lung occupies a smaller space than it did when fully expanded. To compensate for the space lost from collapse of the lung, the chest wall is depressed; trachea, heart and mediastinum are drawn to the involved side, and the diaphragm is pulled upward. The object of all this filling in is to take the place of the collapsed lung. The loss of air content produces con-
solidation of the lung fully as dense as that seen in pneumonia.

Scrimger-44-states that mediastinal displacement can only be explained as a passive response to a diminished intrathoracic pressure.

The four cardinal diagnostic physical signs are:
1. Contraction of one side of the chest.
2. Limited respiratory movement on involved side.
3. Dullness or flatness, absent or diminished breath sounds.
4. Displacement of heart to the affected side.

PATHOLOGY:

There are very few autopsies recorded in which the true pathology of massive collapse can be established.

Pasteur recorded eight autopsies in which massive collapse followed diphtheritic paralysis of the diaphragm, in five of which massive atelectasis was demonstrated. Pasteur was unable to find any evidence of bronchial obstruction. In every case he found a thick fibrinous exudate over the diaphragmatic surface of the pleura. Bradford, Briscoe, Bergamini and Shephard have made similar observations. Bradford failed to find any evidence of bronchial obstruction interfering with the entry of free air.

Elliot and Dingley emphasize the presence of large amounts of muco purulent secretion in the bronchioles, whereas Bergamini and Shephard failed to find any evidence of obstruction. Gross examination of the lung gave no evidence of obstruction. Neither was there any muco purulent secretion so strongly emphasized by Elliot, Dingley and Churchill. They found, however, uniform dilatation and engorgement of the capillaries, arterioles and venules.

Santee records findings similar to that observed by Elliot and
Dingley.

TREATMENT:

Since the condition of massive collapse of the lung, usually clears up spontaneously, there is very little in the literature as to treatment. Prophylactic treatment is the most important.

Among the various forms of treatment advised are (1) rolling of the patient on the affected side as suggested by Sante; (2) puncture and insufflation of air. This form of treatment is not advised.

As has been mentioned previously, the prophylactic treatment is the most important phase of treatment. Every post operative case developing fever, cough or pain in the chest should make the doctor suspicious of massive collapse, and he should watch the case closely for any premonitory symptoms of massive collapse. Local and spinal anaesthesia have been suggested as a prophylactic measure in place of ether anaesthesia. However, it has been definitely shown by Sante-45-and Elwyn-ll-that massive collapse does occur when local and spinal anaesthesia is used although the occurrence of massive collapse is probably greater after ether anaesthesia. This statement, however, is by no means authentic.

One good prophylactic measure that can be practiced by all physicians alike is to avoid operations on patients who have a slight upper respiratory infection.

Since it is definitely known that the vital capacity of respiration is decreased after operations, it is a good idea to encourage deep breathing post operatively. The patient will voluntarily inhibit deep respirations because of the pain and discomfort produced by mobility of the abdominal incision;
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consequently, it has been found a good plan to give CO₂ inhalations. Carbon
dioxide is the normal respiratory stimulant, and it promotes deep breathing.
Dr. H.H. Davis of Omaha reported at a recent medical meeting that he has used
carbon dioxide inhalations after all post-operative cases during the past nine
months, and as yet he has not observed a single case of post-operative pneumonia
or massive collapse of the lung.

Dr. Yandell Henderson, 54, upon the basis of his acapnia theory,
suggests that CO₂ should be used post-operatively routinely to insure against
post-operative complications.

Scott and Cutler-19-present the following conclusions as to the
effect of hyperventilation with CO₂:

1. Hyperventilation with CO₂ at the close of operation combined with a
semi-Fowler position has apparently reduced the incidence of massive
collapse as a post-operative complication.

2. Hyperventilation in one case of fully developed massive atelectasis has
caused an immediate diminution in the opacity of the lung and a decrease
in the displacement of the heart as seen under the fluoroscope. Im-
provement was only temporary, however.

3. Scott and Cutler suggest a post-operative regimen of CO₂ to diminish
the incidence of massive atelectasis.

The consensus of opinion as to the use of morphine and atropine
pre-operatively and post-operatively is that although these two drugs are almost
indispensable in operative cases, their use should be restricted as much as
possible since the use of these two drugs promiscuously might be a factor in the
production of massive collapse.
Chevalier Jackson, as well as Hearn and Clerf advocate bronchoscopic removal of obstructive mucous in the treatment of massive collapse and also as a prophylactic measure in preventing the occurrence of post operative pneumonia.
CONCLUSIONS:

I. The history of massive collapse is very enlightening, because it gives an idea of the extensive research work that was carried on sixty to seventy years ago on this subject. The history is important in gaining a complete knowledge of this subject, because all of the work done in the last fifteen or twenty years in this country on this condition is based upon the various theories as set forward by the older clinicians and research men. W. Pasteur should be given the honor of being called "the father of massive collapse of the lung".

II. The understanding of the pathologic process known as massive collapse of the lung is impossible unless a good knowledge exists of normal respiration, i.e. the anatomic and physiological properties of normal respiration. Vital capacity in massive collapse is decreased, and the negative pressure is always increased.

III. Massive collapse is a frequent complication after operation especially after appendectomies and herniotomies. It has also been shown to be a frequent complication in gun shot wounds. Massive collapse may occur after fractures of the pelvis, femur or from falls upon the buttocks. The right lower lobe of the lung seems to be the one more frequently involved although the entire lung may be collapsed.

IV. Massive collapse usually occurs within the first forty-eight hours after operation.

V. There is still much debate as to the influence of anaesthetics on the production of massive collapse. Although ether has not been definitely proven to be a factor in the etiology of massive collapse, I think it should be
regarded as a suspicious factor until definitely proven otherwise.

VI. The time honored usage of morphine and atropine preoperatively and post operatively must be regarded as having an etiologic relationship to the appearance of massive collapse.

VII. After quite a careful survey of the literature on the various theories concerning the etiology of massive collapse, I have come to the conclusion that the most probable theory and the one that is supported by the most clinical and experimental proof is the theory of complete bronchial obstruction plus respiratory embarrassment, supported by Coryllos, Birnbaum and Churchill.

VIII. The relationship which post operative pneumonia bears to massive collapse of the lung must be seriously considered as it is very possible that we are dealing with the same condition under two different terms. The arguments brought forward by Elwyn, Coryllos and Birnbaum are quite convincing that both conditions are one and the same thing.

IX. Bronchoscopic treatment of post operative pneumonia where the presence of a bronchial obstruction is certain seems to be logical.

X. Dyspnoea is the outstanding symptom of massive collapse, and mediastinal displacement is the outstanding physical and roentgen finding.

XI. Very few autopsies are recorded in the literature, and thus the true pathology of massive collapse is still somewhat undetermined. The weight of evidence seems to favor the finding of muco purulent secretion in the bronchi at the time of autopsy.

XII. Treatment of massive collapse is prophylactic rather than therapeutic. However, when it has occurred, CO2 inhalations and bronchoscopy
are of value. The majority of the cases recover spontaneously. Routine post operative inhalations of CO2 in all post operative cases is suggested as the best prophylactic measure.
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