1933

Postoperative atelectasis

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Senior Thesis

Submitted to the Faculty

of the

College of Medicine

University of Nebraska

by

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INTRODUCTION

Every physician and particularly every surgeon should be keenly interested in the prevention and treatment of postoperative pulmonary complications. Anesthesia, asepsis, and accurate hemostasis have removed most of the dangers of surgical intervention and have vastly increased the limits of surgery, particularly for operations of election. Only in the control of the postoperative pulmonary complications has little progress been made until recently, and in this field lie infrequent but probably preventable postoperative tragedies.

Atelectasis, as a causative factor in the production of postoperative pulmonary complications, has assumed an increasingly important role during the last few years. In fact, Mastics, Spittler and McNamee state that atelectasis accounts for about 70 per cent of all postoperative pulmonary complications.

For some time, there has been much discussion and experimental work on the mechanism, etiology, prophylaxis and treatment of postoperative pulmonary atelectasis. It is the purpose of this paper to make a review of the more important recent contributions to the literature on these various phases of the subject.

TERMINOLOGY

As Pasteur remarked in 1913, "much confusion has arisen from the careless use of the word 'collapse'" in relation to conditions of the lung. It should only be used to indicate the condition in which "a lung or a lobe of it is cimpletely deprived of its air contents." The expression "partial collapse" is misleading and should be replaced by "partial deflation." "Partial deflation of pulmonary bases", as it is used for bedridden patients, signifies the loss of efficient inspiratory force. It has little or nothing in common with true collapse, either clinically or anatomically, but it is doubtless predisposing to "patchy collapse" of the lung.

There is still a confusion of names which is a hindrance to study and perhaps even to understanding. Two terms, collapse and atelectasis, are in common use. Each word has the merit of correct derivation, common usage and clarity of meaning.

In order to avoid confusion arising from the use of the word "massive collapse", Scott proposes the term active collapse or active atelectasis in order to distinguish it from passive collapse or passive atelectasis, which is due to the compression of the lung by pneumothorax, the effusion of fluid in the pleura, or even to a tumor.

Jackson and Lee prefer the term "atelectasis" to "collapse" because the latter suggests the simultaneous collapse of alveolar tissue and bronchi as it occurs in all cases of increased and

positive intrapleural pressure, whereas in the so-called acute postoperative massive collapse of the lung, collapse of the brenchi does not take place. So far as "partial collapse" or "partial deflation" is concerned, Jackson and Lee consider it a common condition in the lung after death, being a lesser degree of postoperative collapse, and for that reason they do not consider it worth while to create a special terminology, thus unnecessarily increasing the already existing confusion of terms. Coryllos and Birnbaum suggest that the term "atelectasis" always be used instead of "collapse, because collapse can occur without atelectasis, but atelectasis can never occur without collapse. They further state that "even atelectasis is a misnomer, and 'apneumatosis' should be used in its stead. The most appropriate name for the condition is 'obstructive massive apneumatosis of the lung'".

In this paper, I originally decided to use the term apneumatosis as I believe it has a more correct derivation and to be more descriptive of the condition than atelectasis. However, because of the common usage and the fact that the Quarterly Cumulative Index lists both "massive collapse" and "apneumatosis" under the heading for Atelectasis, I later decided to use the latter term.

"Postoperative" is a widely employed and very useful adjective, even if it does lend distinction to a causative factor, which is not always operative in this condition,

HISTORY

The rediscovery of massive collapse of the lung and particularly the relating of this condition to postoperative and posttraumatic states dates from the 1910 paper of Pasteur, but we find that however its antecedents may differ, and whatever its surrounding circumstances may be, there is, in its pathology at any rate, nothing really new. With almost unbelievable lapses, its history goes back from that date for sixty years or more to contributions of extraordinary merit, and the more so if we consider the handicaps of that day.

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 found evidence that Wilbrandt, in 1816, Eberhard 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 bronchiales not being as yet sufficiently accustomed to the rythmic order of respiration."

In 1830, Alderson, writing on the "Pathology of Hooping Cough" describes four autopsies in children dying from that disease and gives an accurate description of partial collapse, which he clearly distinguishes from any inflammatory process, but to which he assigns no name. Willshire, however, credits him

with first linking the atelectasis of Joerg with the assumptory collapse of the lung; afterward, (as we shall presently find), playing so important a part in the hands of Bailly and Legendre.

Duges in 1821 and Shallgruber in 1823 also gave clear descriptions of autopsy findings. Up to this time, however, Willshire says that all held the theory in one way or another of congenital nonexpansion.

In 1834, Joerg wrote on a particular condition of the lungs of newborn children and more fully worked out this subject in 1835, at which time, so Willshire believes, was first applied the term atelectasis. Barlow in 1841 reported atelectasis from the pressure of dilated heart. In one case, this seems to have been accentuated by obstructive bronchitis.

Gairdner, in a review (1854) credits Duges (1821), Louis (1829) and Joerg (1832) with recognizing the condition post mortem, each of them calling it "carnification", and each realizing its points of difference from pneumonia. Louis found it in nineteen out of forty-six autopsies after death from typhoid fever. These were amplified in his work on fever in 1841. Louis offers no speculation as to the origin of the condition.

Rilliet and Barthey in 1841 give this description of carnification: "The lung in this state is externally collapsed, soft and flaccid instead of full, hard and resistant as in pneumonia. Its color is violet marked by white lines, disposed in lozenges or squares, defining the lobules. There is no crepitation on

pressure. On incision, red color, smooth, resisting pressure of finger, so as to be penetrable with difficulty; exuding upon pressure a serous, bloody liquid destitute of air. Its appearance, very like the close compact fibres of a muscle, has given it its name. The carnification may be marginal or some part of a lobe. The middle lobe is the only one we have seen entirely invaded." (Perhaps the first mention of a massive collapse.) They considered this condition as a terminal phase of pneumonia.

Legendre and Bailly in 1844 established the autopsy identity of congenital nonexpansion with a return to a similar state, "etat foetal", in lungs which had once expanded. Though they attributed the collapse to the elasticity of the lung parenchyma, acting because of lack of air entrance, their several articles created a profound impression and marks a distinct turn and advance in the literature.

Mendelssohn in 1845 and Traube in 1846, in very similar research, experimentally occluded bronchi with shot, paper and (Mendelssohn) gum arabic paste, and produced collapse.

Fuchs in 1845 wrote in criticism of the then accepted conception of "lobular pneumonia" and considered it to be a disease 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". Thus we see that the three terms proposed today, atelectasis, collapse and apneumatosis, were all used before 1850. Our choice

need not be prejudiced by any thought that one or the other has important priority.

Stokes (1837) understood mediastinal displacement as a sign and result of "vesicular emphysema of Laennec." Stokes' book must have been an authority in its day for it is more than worth while to read it now. With this in mind, it is difficult to understand how Gairdner, Jenner, Hewitt, Lichtheim, and others, before Pasteur, failed to recognize mediastinal displacement in the condition which may, for analogy, be termed the compensatory emphysema of massive collapse.

Hewitt wrote in 1857 of apneumatosis, preferring this term taken from Fuchs to the more commonly used atelectasis. Jenner (1857) wrote: "While complete obstruction of a bronchial tube is followed by collapse of the pulmonary tissue on its distal side; partial closure of a tube has as its consequence, dilation of some of the air cells of the part of the lungs from which it leads, etc." This observation was confirmed fifty years later by the research of MacCallum. In 1861 and again in 1867, Bartels made extended autopsy studies in the lung affections of croupous diptheria, finding atelectasis as a complication when the bronchi were closed by the membrane. Bartels accepted Gairdner's ball-valve theory but questioned whether complete collapse could come about in this way alone. In 1871, Hewitt furnished a chapter on apneumatosis for Reynolds' System of Medicine and Hertz wrote of Atelectasis in Ziemssen's Cyclopedia. It must be said, how-

ever, that with the exception of the work of Mendelssohn, Traube and Gairdner, atelectasis failed to attract general attention and that when noticed at all, it was considered as an affliction of children.

In 1879, Lichtheim repeated Traube's experiments more accurately by the use of laminaria tents. Combining bronchial occlusion with ligation of bronchial vessels, he proved conclusively that collapse would occur only when the blood circulation was unimpeded. Lichtheim also studied the rate of absorption of gases and found that oxygen or carbon dioxide was absorbed rapidly while the absorption of nitrogen was much slower. This remarkable research by Lichtheim seems not to have been given any clinical application.

The first recorded recognition of heart displacement in case of lung collapse after Stokes seems to be that reported by Samuel West, in 1907, in a case of plugging of the bronchus in plastic bronchitis. West sketched the displaced heart and mediastinum and studied its probable mechanism, arriving at the conclusion now generally accepted.

In 1910, Pasteur wrote under the title "Active Lobar Collapse of the Lung after Abdominal Operations", and here the real modern conception of this condition began. At this time, Pasteur makes the first recorded mention of heart displacement as a cardinal diagnostic sign. Also this is the first definite mention of collapse as a type of postoperative complication.

Pasteur here predicts that "active collapse of the lung from deficiency of inspiration power, will be found to occupy an important position among the determining causes of postoperative lung complications." In 1911, Pasteur delivered the annual oration before the Medical Society of London: "Post-operative Lung Complications." Here was presented an analysis of 3,559 surgical operations in Middlesex Hospital, 1906-1910. There were 201 pulmonary complications with 45 deaths; 88 cases were classed as pneumonia and 12 as massive collapse.

Pasteur's final contribution appeared in 1914, and in point of description and diagnosis but little has been added. The practical stimulus given by Pasteur's work can scarcely be overestimated.

During the World War, British surgeons, notably Bradford, and Crymble, reported numerous cases among the wounded and much speculation arose from the curious fact that many of these showed collapse contralateral to the injury. As to thought of etiology, Pasteur seems to have been followed generally, particularly so by Bradford and by Briscoe, who in 1920 published an elaborate study of the musculature of the thorax.

The first communication in American literature, after Pasteur, was the paper by Scrimger of Montreal in 1921. Reporting seven cases in a series of 540 operations, Scrimger tentatively held the theory of vagus reflex followed by viscid secretion and, finally, absorption of the retained air. In the

same year (1921) appeared the very important work of Sewall of Denver. Taking no notice of surgical or military reports, Sewall began his study with the works of Gairdner, Willshire and Lichtheim and developed his study as related to physical examination of the chest and particularly with regard to pulmonary tuberculosis. In this field, Sewall's contribution stands almost alone. His repeated slogan, "Think Atelectasis", in other words, approach no chest examination without the thought that here may be some appearance or phase of atelectasis, placed a much needed emphasis upon the frequency of nontraumatic massive collapse.

The invention of the bronchoscope by Killian in 1897 and its improvement by Jackson in 1904, and since, and the wide variety of conditions to which he has adapted it, began, even before Pasteur's work, to broaden the general understanding of intrapulmonary conditions of which very little had been known. This and Jackson's success as a teacher are certain to have an influence upon the subject in hand, the far-reaching value of which affords wide range for imagination.

The more recent history is well known, and its makers are liberally quoted elsewhere in this paper.

ETIOLOGY

The most fascinating part of this problem is its etiology, which has been heatedly discussed for over a hundred years. Up to the past decade, a great part of the discussion of

etiology has been on a theoretical basis. First, there have been put forward the causes external to the lung:

(A) Paralysis of the diaphragm or a part of it.

(B) Fixation of the diaphragm or a part of it.

(C) Reflex action from injury or insult to the vagus.

(D) Various nerve injuries or disturbances causing incoordination of the diaphragm or parts of it with the other respiratory muscles.

Second, causes within the lung causing obstruction either from:

(A) Partial plug acting as ball valve, the air finally leaving the lung because in these instances, the expiratory air stream is less impeded than the inspiratory.

(B) Actual plugging of the lung with collapse, caused specifically by circulatory absorption of the air distal to the obstruction.

The last theory is still in some dispute but there is an overwhelming array of evidence, both experimental and clinical, favoring the theory of bronchial obstruction as a prime factor in the production of pulmonary atelectasis whether it be postoperative or not.

As has been mentioned before, Mendelssohn, in 1845, showed experimentally that the introduction of foreign bodies--shot, paper ball, thick watery solution of gum arabic--into the air passages, narrowing of the trachea by means of a ligature,

opening of the pleural cavity or section of both recurrent laryngeal or both vagus nerves, was followed by atelectasis. Traube, in 1846, showed that the atelectasis following section of the vagus or recurrent laryngeal nerves was due to the entrance of buccal secretions into the air passages. In 1878-1879, a very illuminating series of experiments was published by Lichtheim, who obstructed the bronchus in rabbits by the introduction of a laminaria plug without opening the thorax. As the plug swelled, the lung beyond collapsed. Collapse did not occur when the blood vessels to the lung were tied. He also sectioned the phrenic nerve and the spinal cord at the second and third cervical segments. No collapse occurred. He went further and established the speed of absorption of the different gases. Thus, operating on rabbits, he found that after obstruction of the bronchus, pure oxygen is completely absorbed in forty-five minutes and carbon dioxide in from ten to thirty minutes, whereas the nitrogen is absorbed only after twenty-four hours.

During the next fifty years, there was little experimental work on this subject. MacCallum, in 1908, had one of his students introduce a pea in such a manner as to completely obstruct the flow of air to that lobe. At autopsy, this lobe was found to be atelectatic and, it is interesting to note, the bronchus behind the obstruction was found to be filled with thick mucus.

In 1928, Corylles and Birnbaum reported the production of atelectasis by plugging the bronchus with a rubber balloon. By

placing the animal in a specially constructed negative pressure box with the chest opened, they were able to watch the development of the atelectasis in the involved lung or lobe. They found that the time necessary for the establishment of atelectasis when the lung was filled with oxygen was about one-half hour, but when the lung was filled with air, it took about twelve and one-half hours to cause a complete atelectasis, although the process was marked at the end of the six hours. Almost simultaneously, Lee, Ravdin, Tucker and Pendergrass produced massive atelectasis in the dog by obstructing the bronchus with the tenacious bronchial secretion removed from the bronchial tree of a patient with massive atelectasis. They emphasized the importance of inhibiting the cough reflex in their animals by the use of sodium amytal.

There have been various explanations as to the fate of the pent-up air in the obstructed lung. The theory that the air of the obstructed lung was absorbed by the blood was first suggested by Fuchs, in 1849, and again by Bartels, in 1861. That this was possible was shown by Lichtheim as mentioned, and, more recently, by VanAllen and Adams, Coryllos and Birnbaum. Gairdner, in 1850, suggested that the air was probably expelled by the plug in the bronchus acting as a ball valve, allowing the air to escape in expiration, but preventing the access of air in inspiration. Although Jackson and others also believed this to be possible, the theory has been generally given up by most writers on this subject.

Postoperative Atelectasis--Ellis

The recent experimental studies on the absorption of gases in the lung and pleural cavity by Coryllos and Birnbaum are outstanding. They showed that gases and anaesthetic vapors contained in alveolar cavities shut off by complete bronchial obstruction gradually leave the lung and finally disappear so that the lung becomes atelectatic. The speed of the disappearance of these gases is proportionate to their solubility coefficient, diffusion speed and chemical affinities for substances dissolved in the blood. Since ligature of the branches of the pulmonary artery corresponding to the obstructed lung prevents this disappearance of gases and vapors from the alveoli, it has been concluded that this disappearance is due to absorption by the blood circulating through the lung. The results obtained in closed as well as open chest experiments show that within two to seven minutes after bronchial obstruction, the oxygen percentage falls rapidly from fifteen to five or six per cent. It remains at these figures until complete disappearance of the alveolar air. In animals in which gas analyses were performed until complete atelectasis occurred, the curves plotted for percentage vary inversely. It is thus seen that the entrapped alveolar air rapidly undergoes marked quantitative changes, the percentage of oxygen dropping and carbon dioxide rising, so that their respective partial pressures tend to come into equilibrium with the corresponding gases of the venous blood. These changes occur in exactly the same way in animals as in man.

Determinations which were carried out by the same technique for anesthetic vapors and gases, namely, ether, ethyl chloride, nitrous oxide and ethylene, showed the great rapidity of their absorption. Integrity of the alveolar endothelium is just as necessary as integrity of the pulmonary circulation. Edema of the lung, produced by injection of concentrated ether vapors into the lung, instantaneously stops gas absorption. Comparative studies of absorption by the pleural cavity of oxygen, carbon dioxide, nitrogen, air, hydrogen, and helium showed that their absorption is regulated by the same physico chemical laws governing absorption of gases from the obstructed lung. Coryllos and Birnbaum concluded their paper by stating that "The aim of this investigation has been to offer direct experimental proof that complete bronchial obstruction is the exclusive cause of atelectasis".

The first to stress the importance of obstructing bronchial secretion in postoperative atelectasis were Elliott and Dingley, who studied very carefully eleven cases of postoperative massive collapse. They noted that the viscid muco-purulent expectoration was uniform in all their cases and that this sputum was different in type from pneumonic sputum. More convincing evidence was given by the bronchoscopists from Jackson's Clinic who were able to cure clinically the atelectasis by removing the obstructing tenacious secretion, or plug, by the bronchoscope.

There are still proponents favoring etiological theories,

other than bronchial obstruction, for the production of atelectasis, but as yet there has been no experimental evidence to substantiate their claims. There is very little in postoperative atelectasis, as we now know it, to support the theory of Pasteur that it is caused by paralysis of the diaphragm or chest musculature. Churchill reports a case of a woman, aged fifty-two, who sustained a fracture of the cervical spine with almost complete transverse paralysis of the respiratory muscles, except the diaphragm. She developed a partial collapse of the left lung. He states she was subject to repeated spells of dyspnea and unproductive cough. This case may then partially support Pasteur's hypothesis. This theory now has practically no supporters, however, although Bradford and Sotan both believe that a reflex paralysis of the muscles of respiration might produce atelectasis.

Besides this reflex action, there are two other reflexes which have to be considered. Probably the most important is a reflex by way of the vagus which directly affects the lung and so may cause a collapse. Secondly, there may be a vasomotor reflex which could cause an increased outpouring of secretion in the bronchial tree or some other circulatory change which might conceivably produce atelectasis. Such a mechanism has been suggested by Gwyn and Scott. A strong argument against the nervous origin of atelectasis is that the experimental worker has been unable to produce it by any nervous mechanism. Coryllos points out that no one has given "an exact and pre-

cise description of the reflex involved or of the nature of the stimulus itself."

There are certain factors, preoperative, operative and post-operative, which either aid the production of atelectasis or play a part in initiating the process. Pre-operatively, we should consider first the length of time and the position in which the patient is kept in the hospital. Schaack reports that a group of patients who were kept in the hospital two weeks or more had four times the incidence of post-operative pulmonary complications in comparison with a similar group who were in the hospital only two or three days. He stated further that the incidence of pulmonary complications in both groups was markedly decreased when pulmonary exercises were induced. It is well known that whenever any patient is kept quietly in bed for an extended period of time, localized areas of atelectasis are very prone to occur. This is especially true in young children, in the aged and debilitated.

The existence of previous pulmonary disease may play a part in any chest complication. There may be increased bronchial secretions in the bronchial tree favoring plugging. It is well known that in cases of bronchitis, there is often a profuse outpouring of secretion during and immediately after the operative procedure. There is always a possibility in a case of bronchiectasis or lung abscess for the pus to cause an atelectasis, whether the patient is operated upon or not. There is also the

possibility of the pulmonary lesion acting as a focus from which organisms may invade a beginning atelectasis, making it more extensive and more serious. This will be referred to later.

It is well known that lung lesions, cardiovascular disease and acute abdominal lesions--upper abdominal, in particular--may reduce the vital capacity markedly and, as may be readily understood, that reduced vital capacity would favor pulmonary complications and atelectasis. Muller, Overholt and Pendergrass, in a routine study of the chests of patients before and after abdominal operations, found a seventy-five per cent diminution of chest expansion after operation. The thoracic circumference became greater after operation.

They noted a surprising difference in roentgenograms taken in the same position, in the same phase of respiration and with the same exposure, before and after abdominal operations. The superior inferior diameter of the thorax was greatly diminished in the postoperative films because of a marked elevation of the diaphragm. Diaphragmatic excursions, as observed under the fluoroscope, were found to be reduced from 33 to 50 per cent after operation.

Several explanations for the altered position and restricted movement of the diaphragm are available. It is well known that distention of the abdominal viscera, splinting of the abdominal musculature because of pain, and surgical dressing are all factors. The disturbance of the normal pressure relationships

in the immediate subdiaphragmatic region that occurs after opening of the air-tight abdominal cavity must also be considered. The intraperitoneal pressure in the upper abdomen is subatmospheric. Overholt demonstrated in animals an upward shift of the diaphragm by changing the intraperitoneal pressure from a subatmospheric, or negative pressure to a positive pressure.

A study of the vital capacity in 218 patients was made before and after the performance of abdominal operations by Overholt. Following operations on the upper abdomen, the vital capacity was reduced sixty-four per cent of the pre-operative or normal value. Following lower abdominal operations, the average vital capacity was reduced forty per cent of the normal value.

Thoracic operations were not accompanied by such an extensive reduction of the vital capacity, which was low previous to operations. In twelve patients on whom a thoracoplasty was done, or a mediastinal tumor removed, the average postoperative vital capacity was seventy-two per cent of the pre-operative value. Head also found less variation in the vital capacity after thoracic operations.

All of the clinical and roentgenologic evidence in regard to the degree of postoperative pulmonary hypoventilation followed so closely the evaluation of the vital capacity that this test was used to determine the importance of various factors which influence respiratory action in the postoperative case. The relation of the position of the patient, the surgical dressing,

the presence of pain, and the effect of the laparotomy itself without the pain, were investigated

The vital capacity of a large number of patients was taken in the semi-Fowler and horizontal positions. The bed position influenced the breathing little. The vital capacity was decreased less than four per cent in the horizontal position.

Determinations of vital capacity were made in seventy nine patients before and after applications of a muslin, many tailed abdominal binder, and the results are given in the accompanying table. Only a slight reduction was noted in the pre-operative

Vital Capacity Averages. Position and Dressing

	Number of Patients	Bed Position Semi-Fowler CC	Flat CC
Pre-operative	58	2,759	2,695
Postoperative	43	1,786	1,718
		Abdominal Binder Before Application	After Application
Pre-operative	54	2,740	2,661
Postoperative	25	1,798	1,520

(Overholt)

group following the application of the binder. However, approximately a fifteen per cent reduction in the vital capacity could be attributed to a tightly fitting abdominal binder, applied postoperatively. When an ordinary surgical dressing of gauze and adhesive tape was applied, there was very little change in the vital capacity.

The effect of the partial abolition of pain by the administration of one-sixth grain (11 mg) of morphine sulphate hypodermically was studied in thirty-five patients. The vital capacity was definitely greater thirty minutes after the administration of the drug. In ten patients the drug was administered before and after operation and repeated determinations of the vital capcaity were made and compared. A slight depression was noted in the pre-operative patients, whereas, postoperatively a definite improvement was noted.

The effect of pain in limiting respiration after operation was also investigated by studying patients still under the influence of spinal anesthesia. Vital capacity determinations were made: (1) with the patients on the operating table before the administration of the spinal anesthetic; (2) ten minutes after induction of spinal anesthesia by administration of from 100 to 200 mg. of procaine hydrobromide; (3) after the abdominal operation, but before the return of cutaneous sensation, and (4) six hours later. The first reading served as a control while the second disclosed any alteration in vital capacity due to the spinal anesthesia itself. The third reading indicated the mechanical changes in respiratory activity due to the opening of the abdomen alone without the inhibiting effect of pain. The last reading taken after the disappearance of anesthesia indicated the extent of postoperative ventilation with all factors including pain being present.

Five patients on whom lower extremity or perineal operations were performed were used as controls. Vital capacity determinations were carried out in this group in a manner similar to that used in the abdominal group. In addition, a vital capacity reading was taken thirty minutes after the induction of anesthesia at the time the intercostal muscles were paralyzed. A fall in the vital capacity was noted while the intercostal muscles were not functioning, but a return to the normal level took place after the operation and after there had been a restoration of thoracic movements.

In the abdominal group, a slight reduction in the vital capacity followed spinal anesthesia alone. The operation itself caused a marked fall in the vital capacity, even though the operations merely consisted of an exploratory laparatomy. After the disappearance of the effects of spinal anesthesia, a still greater reduction was noted. Allen found that forty-three per cent of ninety-four surgical cases subjected to pre- and postoperative chest x-ray examinations have shown a unilateral high position of one hemidiaphragm and that fifty-seven per cent exhibited bilateral high positions of the domes. Y. Henderson states that: "The acapnial position of the lungs may be described as one near or below that of normal expiration, a condition of prolonged deflation. As long as this position is maintained, the thoracic, diaphragmatic, and abdominal movements of breathing are carried on at such a shallow leval that inspiration in-

flates the lung only slightly, and expiration deflates them almost as completely as the deepest voluntary expiration. This deflation leaves some parts of the lung unventilated, thereby permitting an accumulation of mucus, a blocking of airways, and the initiation of atelectasis."

It is fairly well agreed that bronchial obstruction, plus decreased aeration ability of the lung, is a prime cause of post-operative pulmonary atelectasis, and that obstruction is usually due to retained tracheo-bronchial secretions. Moreover, limitation of movement of the diaphragm for one cause or another greatly aids this process. Numerous other causes have been suggested without any general acceptance of any of them.

It appears that the role of the bronchial secretions, either in producing the atelectasis or in determining the specific type of atelectasis, which ensues, has not been given sufficient consideration. In a study of the action of cough on material in the tracheobronchial tract, Archibald and Brown observed that whereas the usual action of cough is to expel material from the tracheobronchial tree, it may, under certain conditions, actually bring about the opposite result; that is, drive material deeper. Moreover, they noted that the degree of penetration of the material into the smaller ramifications of the tracheobronchial tree was in direct proportion to the viscosity of the material in question; that is, the less viscid the material, the greater was the penetration and the greater the possibility of dispersion.

Brown observed the different types of secretion in the cases of postoperative pulmonary atelectasis apparently due to bronchial obstruction. He states: "The usual type of secretion consists of a thick, viscid material, not necessarily present in large amounts, and broken up with difficulty. On bronchoscopic examination, this material is found to plug a bronchial orifice, atelectasis occurring distal to the plug. If the material is slightly less viscid, one or more smaller bronchi may become plugged, and as the material found is again less viscid, smaller bronchi still are seen to be filled or plugged, and the possibility of obstruction in more than one bronchus increases. Finally, when one notes a secretion of almost watery consistency, then one obtains clinically the condition which has been spoken of as 'drowned lung'".

Brown also gained the impression that the incidence of pulmonary atelectasis is greater following spinal anesthesia than following any form of inhalation or regional anesthesia. He gives several reasons why spinal anesthesia might predispose to this complication: "First, spinal anesthesia definitely inhibits the depth and force of respiratory movements, not only during the operation itself, but for a considerable period thereafter. It is these respiratory movements (both intrinsic and extrinsic) which tend to rid the tracheobronchial tree of foreign matter or secretions. Second, the normal viscosity of the secretions of the tracheobronchial tree appear to be increased, i.e., the mater-

ial is more tenacious following operation under spinal anesthesia. Third, following operation under spinal anesthesia, the patient tends to remain relatively quiet for a number of hours. One has, then, a more tenacious sputum and decreased or impaired factors that might tend to free the tracheobronchial tree of this material. The increased possibility for this material to obstruct or plug a bronchus and the subsequent development of atelectasis appear reasonable."

In an analysis of four hundred ninety seven cases of operations with spinal anesthesia, Arnheim reports twenty-one cases of pneumonia. He states that: "The high incidence of pneumonia, twenty-one cases (4.2 per cent), was somewhat of a surprise. These pneumonias were all verified by x-ray or post-mortem examination. We soon learned that spinal anesthesia did not prevent postoperative pulmonary complications."

RELATIONSHIP TO POSTOPERATIVE PNEUMONIA

Postoperative pneumonia, despite the study it has received, remains a dreaded complication of surgical therapy. The cause remains much discussed, and as a knowledge of etiology is the key to prevention, it is the problem we are chiefly concerned with. The principal theories of causation are: atelectatic, anesthetic, embolic and inhalation.

Two principal theories with respect to the initial mode of infection have existed. Some writers have advocated the theory

that lobar pneumonia is hematogenous in origin, basing this belief on the fact that pneumococci have been isolated from the blood prior to the development of symptoms or physical signs of pneumonia. This point of view, however, has received no experimental support, since attempts to produce lobar pneumonia by intravenous inoculation have consistently failed. On the other hand, the more commonly accepted view that the mode of infection is by way of the air passages has received a certain amount of confirmation in the experimental production of pneumococcus pneumonia in animals by various methods of intratracheal or intrabronchial inoculation.

Elwyn, in 1924, after twenty-eight months' study of postoperative pneumonia, concluded that following operations on the abdomen, there occur small or large areas of atelectasis in the lungs, especially in the lower lobes. When a bronchitis is present at the time of the operation, or irritation by the anesthetic prepares the bronchi for infection, any such infection is liable to spread to the atelectatic lung, with the production of pneumonia.

The recent experimental work of Henderson, Haggard, Coryllos and Birnbaum confirms the clinical studies of Elwyn and others. Coryllos and his collaborators found that if the occluding plug in a bronchus contained a highly virulent pneumococcus organism that following atelectasis, pneumonia would develop. Furthermore, x-ray studies and physical findings could not differentiate

between a pneumonia and atelectasis. Many questions arise in such an investigation and discussion so I will only give a summary of Coryllos' conclusions, which are theoretical but have considerable clinical and experimental data to support them.

He is of the opinion that lobar pneumonia is a pneumococcic atelectasis due to bronchial obstruction by mucous exudate infected with virulent pneumococci. Atelectasis (generally but not always postoperative pulmonary complication) is due to bronchial exudate usually infected with pneumococcus Group IV, an organism of low virulence. Postoperative pneumonia, or pneumonitis is produced by the same mechanism as atelectasis and is due to the same infective organisms, pneumococcus Group IV, which is, however, of higher virulence. The study of atelectasis gives us a better insight into the relation between atelectasis and postoperative pneumonia, and in the broader sense, between three forms of the same pathologic process, namely, atelectasis, postoperative pneumonitis, and lobar pneumonia. On such a basis, atelectasis bears the same relationship to bronchopneumonia that lobar atelectasis bears to lobar pneumonia or massive atelectasis bears to massive pneumonia. Finally, one can state that the sole and relative position of bronchitis in the evolution of the disease syndroms is represented by: bronchitis, atelectasis and pneumonia.

SYMPTOMS AND PHYSICAL SIGNS

The symptoms of atelectasis vary greatly in different cases,

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 at rest in bed. It is important to recognize that massive atelectasis may involve the whole of one lung without the presence of any urgent symptoms, and the condition may be overlooked unless the chest is carefully examined, and this is more especially the case in the contralateral type. Dyspnea is the most constant symptom. It is usually of moderate severity but is greatly increased on exertion and even by such slight exertion as sitting up in bed. Cough, usually slight, but often repeated and persistent, may be present, together with expectoration of a muco-purulent character. Pyrexia is often absent, and when present may be due to some other lesion, but pyrexia is necessarily present in the cases where pneumonia, pleurisy, etc., develop as complications of atelectasis.

The signs are most marked and most easily recognized in cases of contralateral collapse involving the whole of one lung. The cardiac impulse is greatly displaced toward the affected side. The displacement is lateral and upward, the lateral displacement being usually far the greater. The area of visibility of the cardiac impulse may also be increased greatly in extent. The affected side of the chest is retracted and immobile, and the ribs can be seen and also felt to be closer together than on the normal side. The dome of the diaphragm on the affected side is also much higher than normal and the displaced diaphragm is

immobile on the affected side. The high level of the diaphragm can be readily demonstrated on the left side by percussion; this method is not so satisfactory on the right side, but x-ray observation not only demonstrates the high level of the diaphragm but also reveals its immobility on the affected side. The percussion note is impaired all over the affected side, and dullness may be present up to the level of the clavicle. Tactile vocal fremitus is either diminished, absent or increased. If diminished or absent, the breath sounds are also diminished or absent; if increased, the breath sounds are tubular or amphoric in character. In such cases, bronchophony and pectoriloquy are exceedingly well marked and whispering pectoriloquy may be heard with great distinctness over a wide area. Thus two groups of cases may be recognized, one with diminished or absent tactile fremitus and breath sounds, and one with increased tactile fremitus, together with tubular or amphoric breathing, and with bronchophony and pectoriloquy. The great bulk of cases conform to the type with increased tactile fremitus and tubular breathing, but some cases, and more especially those seen early, have weak or absent tactile fremitus and breath sounds. The signs, however, are apt to change in one and the same case; thus at first, weak or absent breath sounds may be present, and twenty-four hours later, these are replaced by loud tubular or amphoric breathing, with increased tactile fremitus. Such changes may occur without any alteration in the degree of displacement of the heart. The alterations in physical

signs are commonly of the nature just described, but sometimes a change takes place so that tubular breathing, etc., is replaced by weak or absent breath sounds, and such alterations may occur more than once in the clinical course of the case. These repeated alterations are evidently dependent upon the patency of the bronchial tubes, and are seen in the later stages of the condition when the chest wall is no longer immobile and some degree of re-expansion of the lung is taking place.

Rales and adventitious sounds may be present, but they are often absent throughout the clinical course of the most marked cases, even those involving the whole of one lung. Adventitious sounds may, however, be a prominent sign in the later stages of some cases when the lung is re-expanding. They are also present when inflammatory complications, such as pneumonia, develop in the collapsed lung. These signs are not essential signs of atelectasis as such; they are more usually to be associated either with re-expansion of the lung or with the development of inflammatory complications.

In cases of massive collapse of less extent and involving only one lobe or a portion of one lobe, the signs are similar to those just described, but they are necessarily more limited in area, and the cardiac displacement, although present, is not so great as that seen in cases of collapse involving the whole of one lung. The high level and immobility of the dome of the diaphragm are, however, always striking features of these cases.

The physical signs may be summarized by saying that the pulmonary signs present a considerable resemblance to the well-known signs of consolidation; if anything, they are rather more marked, especially in the tubular or amphoric character of the breath sounds; these signs are, however, accompanied with retraction and immobility of the chest wall, together with displacement of the heart and of the diaphragm. Sewall made the following apt statement: "If the examiner but thinks atelectasis, bears in mind the conditions of its occurrence, and applies the simple tests for its recognition, physical diagnosis of the lungs will gain much in certainty."

TREATMENT

Lee made the first suggestion for any treatment which seemed to have an effect on fully established massive atelectasis. On the basis of the assumption that the obstruction in the respiratory tree occurred in the main bronchi, Jackson and Lee advised bronchoscopic aspiration and in several cases carried out the procedure with improvement both in the patient's condition and in the degree of atelectasis present. There was a marked tendency toward recurrence of the atelectasis, for which they sometimes repeated the therapeutic measure. Two other measures have been advocated more recently which are extremely simple. Sante has advised rotating the patient on the unaffected side and has

reported the striking improvement obtained by this simple procedure in at least some cases.

The postoperative use of carbon dioxide as a prophylaxis is now recognized as the ideal method of preventing atelectasis and subsequent postoperative pneumonia. In 1920, Henderson, Haggard and Coburn published their important work, which showed that ether and other inhalation anesthetics could be rapidly removed from the body by inhalation of carbon dioxide. Graham and others used carbon dioxide continually after abdominal operations following the publishing of this paper. At first, this was done without any reference to the question of preventing pneumonia, but later, after the stimulating publication of Coryllos and Birnbaum, it seemed that possibly it might have been of benefit for that purpose.

Van Allen points out that coughing cannot be effective unless the lungs are well inflated. The forcible expiration of a considerable amount of air is necessary to clear the bronchi of obstructing viscid secretions. Scott and Cutler found that the deep breathing or hyperventilation, which is induced by inhalation of carbon dioxide distends the lungs so effectively that postoperative atelectasis is avoided or counteracted and they were the first to report the use of carbon dioxide for this purpose. Brown reported that carbon dioxide inhalations, when observed through the bronchoscope, were seen to: (a) increase the rate and depth of respiration; (b) produce violent movements

in the tracheo-bronchial tree and alterations in the shape of the lumens of its branches, thereby tending to free adherent mucous; (c) induce a distinct blanching of the mucous membranes of the trachea and bronchi.

Henderson, Haggard, Coryllos and Birnbaum, in their experimental work induced atelectasis and pneumonia in dogs and then used carbon dioxide in treatment. As a background for this study, they cited the following facts:

"(a) If the lungs are not fully distended soon after birth, pneumonia is likely to occur.

"(b) After surgical operations, massive lobar or lobular atelectasis of the lung is a rather frequent occurrence, and is the condition from which postoperative pneumonia develops. This atelectasis is prevented and relieved, and the risk of pneumonia eliminated, by the inhalation of carbon dioxide.

"(c) The inhalation of 5 per cent carbon dioxide in oxygen, which is now the standard treatment for carbon monoxide asphyxia, is also an effective prevention of postasphyxial pneumonia.

"(d) In pneumonia, it is the blocking of the lung airways, bronchi or bronchioli, by plugs of thick and sticky secretion, which is the critical morbidic factor producing atelectasis and the conditions characteristic of an undrained infection."

They further stated that their experimental work demonstrated these facts:

"(a) Atelectasis that is induced experimentally in dogs by

blocking a bronchus is quickly cleared up and the lung is redistended by the deep breathing induced by inhalation of carbon dioxide in proper dilution.

"(b) Pneumonia that is induced in dogs by insufflation of a virulent culture of pneumococci is generally overcome, the lung is redistended and the animal is restored to health by inhalation of carbon dioxide sufficient to cause deep breathing and continued until the pneumonic area is cleared."

There are now numerous reports to be found in the literature that when the routine postoperative use of carbon dioxide has been used, the incidence of postoperative atelectasis and pneumonia has been reduced to a minimum.

Hyperventilation with carbon dioxide is easily carried out after operation. Henderson and Haggard devised for this purpose a special mask which mixed pure carbon dioxide with inspired air. There is a safety valve to prevent the patient from getting too much of the gas. This type may be best when it is necessary to have one piece of apparatus for hyperventilation alone. However, carbon dioxide is administered easily and safely from any of the modern gas machines. The following is the procedure Scott and Cutler use, no other special apparatus being necessary: The gas from a tank of 30 per cent carbon dioxide and 70 per cent oxygen is delivered undiluted to the mask; the anesthetist varies the amount of carbon dioxide given by allowing the admixture of air around the mask, and by the interval the latter is held in place.

Postoperative Atelectasis

The whole purpose of the procedure is to hyperventilate fairly vigorously but not sufficiently to tire the patient or to raise the blood pressure excessively. After the patient begins to breathe deeply and at a slightly increased rate, the mask is removed or lifted and the hyperventilation diminishes. Usually several such waves of increased depth of respiration are produced in this manner over a period of from five to ten minutes. The use of carbon dioxide following operation should be supplemented by the elimination of the factors, so far as possible, that tend to diminish the vital capacity as reported by Overholt and others, such as flat position in bed, tight abdominal binders and pain.

CASES

Case I:- The diagnosis before bronchoscopy was atelectasis of a portion of the right lower lobe; after bronchoscopy, atelectasis of two tertiary lobules, right lower lobe.

A 10 mm. Bruning's bronchoscope was inserted. A small amount of thin mucous material was found in the trachea. The left primary bronchus was clear; no blocks were noted. At a distance of 37 cm. from the mouth down the right primary bronchus and into the anterior secondary bronchus, two posterolateral tertiary bronchi were noted to have their orifices completely blocked by small plugs of thick, tenacious sputum. The patient was asked to take deep breaths, but these plugs were immovable, whereas the thin mucous secretion of the bronchi moved in and out with respiration. The entire area was sprayed with 10 per cent cocaine and 1:1000 solution of epinephrine, equal parts. The aspirator applied directly to these plugs of mucous and each was removed. The air was seen to enter the bronchi involved. The orifices of each were then touched with the mixture of cocaine and epinephrine, and before withdrawal were seen to be about one-third larger than at the beginning of the procedure. After withdrawal of the bronchoscope, it was noted that the physical signs had changed, and that respiration was much easier.

Roentgenograms taken immediately before and after bronchoscopy showed clearing of the atelectatic area after bronchoscopy.

Case II:- The patient, a poorly nourished Jewish boy, 14 years of age, was admitted to the Hospital on October 16, 1924, with a diagnosis of appendiceal abscess and probably generalized peritonitis. At the time of admission, he also gave definite evidence of having bronchitis.

He was immediately operated upon under gas-oxygen anesthesia by Dr. Robbins, a gangrenous, perforated appendix was removed and a localized abscess drained with one cigarette drain placed in the pelvic cavity.

He made a satisfactory recovery from the operation, although it was noted that expectoration of thick, greenish, purulent material was rather profuse during the first few days. On the fifth day after the operation, he had a severe attack of coughing and subsequently became noticeably cyanosed. His temperature rose promptly five degrees, his pulse to 152, his respirations to 52, and he complained of pain in the right chest. Physical signs at this time showed the right side of the chest to be sunken, the intercostal spaces retracted and expansion to be practically absent. The heart was displaced to the right, the right border being 6 cm. to the right of the mid-sternal line. Dullness was present over the entire right chest but heavy percussion gave a somewhat tympanic note. Vocal fremitus was increased over the right, upper chest, but absent below. The breath sounds were loud and amphoric over the entire right upper chest diminishing in intensity towards the axilla and posterior aspect. No crackling

rales were heard anywhere although a few scattered bronchial mucous rales were heard in both lungs.

The diagnosis of massive collapse of the right lung was made, which was corroborated by a roentgenogram of the chest.

On the subsequent days, the patient's cough became more productive and the expectoration of thick green sputum, which had been noted from the beginning, increased in quantity. Almost daily, roentgenograms were taken, which showed a slow return of air to the collapsed portion of the middle and lower right lobes and the gradual return of the heart shadow to its normal position. By November 27, 1924, there was practically restoration to the normal picture.

Case III:- The patient, a well-mourished and developed young Negro girl, 18 years of age, was admitted to the Philadelphia Lying-In Charity Hospital on February 17, 1925, having been in labor for twenty-four hours.

She had bronchitis at this time, was coughing occasionally, and her temperature was slightly elevated.

A pelvic examination showed evidences of a markedly contracted pelvis; the foetal head was floating and the membranes were unruptured. She was operated upon the evening of admission, a Cesarean section being performed by Dr. Edmund Piper. Three days after the operation her temperature became elevated and she presented some of the signs of a postoperative pneumonia. She was seen by Dr. Wolwerth in consultation at this time and the possibilities of a collapse of the lung were suggested although the

most likely condition seemed to be that of a postoperative pneumonia.

The patient died five days after the operation.

Necropsy. (A2988) Within the peritoneal cavity, the small bowel was dilated; there was a moderate amount of localized peritonitis about the operative incision in the abdominal and uterine walls, and the loops of bowel in this region were bound together by plastic exudate. A small amount of purulent fluid was present in the pouch of Douglas.

One was struck by the greatly reduced size of the chest. The diaphragm occupied an unusually high position, as the dome reached to the third interspace on the right and to the fifth on the left. The heart was drawn slightly to the left, its right border measuring 3.5 cm. and the left 8 cm. from the mid-line. Both lungs appeared small, the upper lobes were crepitant and moderately air-containing, the lower lobes being partially atelectatic. The atelectatic areas were demarcated from the upper half of both lower lobes by irregular horizontal lines below which the pulmonary tissue was reduced to about one-half of the usual size. Atelectasis was, however, limited in some degree to definite lobules in most of which the alveoli were completely collapsed, in others they were distended. The inferior edge of the lung presented a distinct lip of dark limp non-air-containing tissue. On section the lower lobes were quite black in their dependent

portions and presented the typical picture of atelectasis. Manual compression of this pulmonary tissue caused small plugs of thick, white, purulent mucous to exude from the bronchioles and smaller bronchi. Dissection of the bronchial tree revealed the main bronchus leading into the lower lobe to be practically filled with extremely tenacious and purulent mucous. Interestingly enough this picture was more marked on the right side than the left although the degree of associated atelectasis did not show this difference.

Microscopical studies of sections from the atelectatic areas of these lungs showed the alveoli to be universally collapsed, the tissue being entirely composed of closely packed alveolar walls. One of the most noticeable features was the marked dilatation of the capillaries in the alveolar walls which were distended to several times their normal width. All of the bronchioles contained exudate which was rich in fibrin in the meshes of which were closely packed polymorphonuclear leucocytes, generally completely plugging the bronchiolar lumen. The bronchial wall itself peribronchial spaces did not show evidence of infection comparable to the usual picture of broncho-pneumonia. Associated with the terminal bronchi, however, were evidences of exudate definitely spreading out through the atria and neighboring alveoli.

Case IV:- The patient, K. M., a well developed and nourished young white girl, aged 24 years, was admitted to the Pennsylvania Hospital on February 1, 1925, with the typical signs of an attack

of acute appendicitis. Her past history was essentially negative and at the time of admission, she did not show evidences of any respiratory infection.

She was operated upon on the day of admission and a gangrenous appendix was removed. Advanced generalized peritonitis was present at the time of operation. For the first six days subsequent to the operation, she did moderately well. On February seventh, her abdomen became distended and vomiting started, which was persistent up until the time of death on February tenth.

Necropsy. (A2977) Within the abdominal cavity, about one-half of the small bowel was found to be greatly distended, proximal to a point of mechanical obstruction in which small loops of gut had become twisted and bound to other adjacent coils of intestine in the ileocecal fossa. Loculated pockets of pus were present in the right renal fossa and between the right lobe of the liver and the diaphragm, forming an early subphrenic abscess.

In the thoracic cavity, the left lung appeared normal. The right lung was partially atelectatic, being rather clearly demarcated from air-containing pulmonary tissue by a horizontal line. A number of small scattered patches of atelectasis were present along the entire posterior surface of the lower right lobe and also at the base of the middle lobe. There was no exudate either upon the parietal, or visceral pleura, but the vessels of the diaphragmatic pleura were considerably congested,

as were also those of the diaphragmatic surface of the lung. On section, the atelectatic portion was quite prominent. There was a slight excess of thick mucous in the bronchial tree of the lower and mid lobes on the right side.

Case V:- Mr. F. H., aged 50, Immanual Hospital 36995, in October, 1930, had a cholecystectomy. He had a rather stormy convalescence with vomiting, some abdominal distention and hiccough beginning the second day postoperative. On the fourth day, for the first time, his temperature became over 100, hiccoughing and vomiting continued. His temperature then ran from 100 to 102, his wound was not infected and the abdominal symptoms definitely improved, but hiccoughing continued. On the ninth day, his condition became definitely worse with temperature to 102.8 and on the tenth day 103.2, with decreased respiration on the right side and dullness at the right base. An x-ray showed cloudiness at the right base 3 plus. At this stage, we began carbon dioxide inhalations with immediate improvement of all his symptoms. The hiccoughing immediately stopped, the next day his highest temperature was 100 and after that continued normal with a perfectly uneventful convalescence. I believe that about the ninth day, the mucous plug was formed and his more acute symptoms were due to atelectasis, which was immediately relieved by carbon dioxide before definite pneumonia had developed.

Case VI:- Mrs. A. S., aged 52; height 5 feet, weight 170. This patient was a strong, well, German woman, the wife of a

retired farmer. She had always been well until two years ago when she developed what was regarded as an acute apendicitis. When first seen, I regarded a perforation of the appendix as probable. She was immediately removed to the hospital where a median incision was made. The appendix had ruptured and drainage was instituted. The recovery was uneventful, but about six months later, a postoperative ventral hernia was evident at the sight of the operation, and a repair was advised.

She did not consent until February, 1930, at which time she consulted Doctor L. O. Hoffman and myself. Pre-operative examination showed no contra-indication for surgery, except overweight. The heart and lungs, blood pressure and kidneys were normal. No x-ray examination of the chest was made. She was operated upon February 20th, 1930, at eight a.m. by Doctor Hoffman. The large hernial defect was closed and a Penrose drain was inserted through a stab wound. Nitrous oxide and ether were used and she was on the table about one hour and fifteen minutes. The first thirty hours postoperative were uneventful except for the common complaint of backache. At five o'clock p.m. of the second day, she rather suddenly complained of difficult breathing. The pulse rate was 125; the respiration 32, and the temperature 99.8. During the night the nurse recorded difficult respirations when she moved. I saw her the following morning, forty-eight hours after the operation. At this time there was some cyanosis and marked dyspnoea, no fever

and little cough. There was a very definite impairment of resonance of the entire left lung, except the apex. No breath sounds were audible, and the left chest seemed fixed. Resonance was normal over the right chest and there were rales at the right base. Postoperative massive collapse was suspected and x-ray ordered. The report of Doctor Charleton B. Pierce is as follows:

"Study was made at the bedside of the patient's thorax. This presented a gross accumulation of air in the cardia of the stomach and the splenic flexure of the colon. The diaphragm with the gas-filled stomach beneath it reached the fifth rib posteriorly and the second anteriorly. Above this, there was a small amount of relatively aerated lung, which we would interpret as the upper lobe, with a density at the hilum continuous with that on the right side, which we would interpret as collapsed lower left lobe. In the right thorax, there was observed a relatively homogeneous mass showing two roundish portions in profile at about the anterior axillary line. Lateralward there was aerated lung but definitely diminished aeration. The diaphragm is visualized at the level of the right rib.

"Our interpretation is that at the time of onset of the terminal difficulty, there occurred an occlusion of the left lower main bronchus with resultant atelectasis of the left lower lobe. Incident upon that respiratory difficulty, the patient gulped air contributing to the gastric dilation. Following this, in the absence of a cough reflex, an accumulation of mucous in the

right bronchus contributed to a diminution in aeration of the right lung, which permitted the distended stomach to displace the heart, mediastinum and collapsed lower left lobe into the right thorax, increasing the respiratory embarrassment.

Atelectasis of the left lower lobe.

Gastric dilation. Partial atelectasis of the right lung.

Mediastinal displacement.

Feb. 24, 1930."

Postural treatment and inhalations of carbon dioxide and oxygen had no effect whatever, the condition becoming gradually but progressively worse, and the next day Doctor J. B. Potts did a bronchoscopic examination. Tenacious, molasses-like mucous was removed from the right bronchus, but the left bronchus seemed completely occluded one inch below the bifurcation. Doctor Potts described the obstruction as a red mass, which pulsated markedly, and he felt that this mass was either the heart or possibly aneurysm. The x-ray picture was entirely atypical, as the mediastinal structures pushed away from the side where the chief collapse showed clinically.

The patient seemed to breathe more easily after returning from the operating room, due probably to the removal of mucous from the right bronchus, which probably improved somewhat the atelectasis in a portion of the right lower lobe, as nothing was removed from the left bronchus.

This case was, in our opinion, one of a double massive collapse

with the major collapse on the left side. The apparent improvement after the bronchoscopic treatment was short; the pulse became more rapid; respirations more labored; cyanosis more marked and the patient sank into a coma and died about five hours later. Unfortunately, a post mortem was not obtainable.

COMMENT ON CASES

Case I:- This case demonstrates that the cause of atelectasis may be an occluding plug or plugs of thick tenacious secretion. It further demonstrates the value of bronchoscopy in the treatment of the condition.

Case II:- This case demonstrates the relationship of bronchitis to the production of atelectasis and the particular need of phrophylactic treatment in any patient in which it is necessary to operate when suffering from an upper respiratory infection. It further gives the typical physical signs.

Case III:- This case was reported because of the very splendid pathological description. It also demonstrated again the etiological factor, that is bronchitis followed by occlusion of the air passages with subsequent absorption of the enclosed air by the blood stream.

Case IV:- This case again gives the pathology and demonstrates the occluding mucous plug as the etiological factor in the production of atelectasis.

Case V:- This case was cited in order to show the efficacy of

carbon dioxide in the treatment of atelectasis.

Case VI:- This case has been cited because of its unusual character. It is to be regretted that a necropsy was unobtainable because to know the exact nature of the pulsating red mass occluding the left bronchus would have been most interesting.

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