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Senior thesis on the artificial pneumothorax treatment of acute lobar pneumonia

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A Senior Thesis
on the
ARTIFICIAL PNEUMOTHORAX TREATMENT
of
ACUTE LOBAR PNEUMONIA

With a review of the literature

by

Lawrence L. Anderson.

1936.
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Introduction

The treatment of lobar pneumonia has constituted a harassing problem to the medical profession since the beginning of medicine. As Osler (39) said, "Ever since the days of antiquity, pneumonia has been observed and studied; while one method of treatment after another has been vaunted with enthusiasm, only to be abandoned in despair; the disease meanwhile pursuing the even tenor of its way with scant regard for the treatment directed against it." That this statement, made thirty years ago, is still applicable today, is shown by the present mortality rate in this country. While the mortality rate varies from year to year in the larger hospitals, it remains relatively fixed between 30 and 50 percent. Even in private practice, where the patient is given a more individualized type of treatment, the mortality is said to be no less than 25 percent. (11).

This paper is being written in an effort to establish, in an unbiased manner, the value of one of the latest methods to be advanced for the treatment of lobar pneumonia, namely; the therapeutic use of artificial pneumothorax.
While the advocates of this treatment do not claim that it is a positive cure for pneumonia, they do claim some advantages to be gained by its application, which in themselves are important enough to merit its being given a fair trial.
History of Pneumonia

The characteristic clinical picture so often seen in lobar pneumonia has been recognized since the earliest times by the members of the medical profession. Hippocrates discussed pneumonia at quite some length in his writings, although at this time there was much confusion, and no differentiation between pneumonia, pleuritis and other acute thoracic diseases. Aretaeus, Celsus and Thenison, four centuries after Hippocrates were the next writers to discuss pneumonia, contributing little however, to what was already known. Diokles of Karystus, one of the oldest writers, is credited with having pointed out the difference in the pathology of pneumonia and pleuritis. Although mentioned and described in the writings of Galen, Caelius Aurelianus, Aetius, Alexander Thrallianus, Rhazes, Avicenna and others, no great advance was made until the era which began about the time of Harvey, Sydenham and Malpighi, when physicians began to think and investigate for themselves.

The relation between the clinical manifestations of pneumonia and consolidation of the lungs was first pointed out by Morgagni, while Baillie described the morbid process as "Hepatization". Further
important studies were made by Laennec, Cruveilhier and Rokitansky. The diagnosis of pneumonia based on the physical signs is credited to Auenbrugger who discovered the possibilities of percussion, and to Laennec who fathered auscultation. Addison showed that the inflammatory exudate was extravesicular and not interstitial, while to Rokitansky belongs the credit of differentiating between the lobar and lobular types of pneumonia. (57)
History of Pneumonia Therapy

Pneumonia therapy has an ancient background fully in keeping with that of the disease itself. Hippocrates treated pneumonia by laxatives, expectorants, cups, or the actual cauterity and with baths on the fourth and seventh days and oil inunctions on the fifth and sixth days. The principal food allowed was of fatty and saline character. Galen, in 230 A.D., advocated venesection, a practice which was strenuously opposed by Epistretus. Laennec stated that "In every case, the more feeble the pulse, the less the indication for blood-letting". (39) In 1830 Demees stated that nearly all writers agreed that the proper treatment of pneumonia should consist of; venesection, local and general, purges, blisters, alteratives, antimony and tonics. At this time, blisters were considered as being, next to venesection, the most efficacious remedy for pneumonia. They were not only applied to the chest, but to the legs, thighs or inside the arms. When they failed to act as derivatives, they were supposed to act beneficially by exiting temporarily, the powers of the system, thereby making further bleedings possible.
Alkalies were used by the ancients to render the blood less plastic. Subcarbonate of soda, potassium or ammonium soap, also the sulfates of soda and potassium were used. The fact that hepatized portions of lung placed in an alkaline solution softened, lent strength to this opinion. As recently as 1919 alkalies were advocated by different men in this country. (50) (56).

Purgatives and emetics were used freely with the view of lessening the congestion within the chest or on the theory that the inflammation might be complicated with a bilious disorder.

Calomel and opium separately or combined, were extensively used in England. Calomel was often given to the point of salivation. Tartar emetic in large doses was used in Italy and also by Laennec in France. The drug was given in sufficient doses to cause the patient to vomit two or three times and to produce five to six stools a day. The Italians gave as high as several drachms in twenty-four hours and controlled the vomiting and diarrhea by syrup of poppies. (14).

What was undoubtedly one of the most salutary events, so far as treatment is concerned, was brought
out by the Viennese school of Therapeutic Nihilism, lead by Skoda. As a result of its doctrines the fact was thoroughly demonstrated that pneumonia is a self limited disease with a natural tendency toward recovery, and that nature can do more for the patient than the routine application of any one method of treatment indiscriminately applied.

Up to 1910 there were a host of vaunted specifics. Flint (22) once claimed that quinine in 20 to 40 grain doses would abort pneumonia. Schwartz (47) claimed that iodine was a specific, and at different times, veratrum viride, ammonium carbonate, ergot, salicylates, sodium benzoate, creosote carbonate and other drugs have claimed this distinction.

About 1910 vaccines and serums came into prominence. Although serum treatment in lobar pneumonia had been attempted as early as 1897 by Washbourne, (55) his serum treatment was a failure because he was unaware of the existence of the different strains of pneumococci. Today the serum treatment is used in pneumonia caused by the Type I and Type II pneumococci, and is said to reduce the mortality ten to fifteen percent. (52) Vaccines, while they are of some value in the treatment of bronchopneumonia, (31) are of little or no value in the treatment of lobar
pneumonia. Park (42) in referring to a series of cases of lobar pneumonia treated with mixed stock vaccine says, "In fact, if we had not known that the vaccine was being given to one-half of them we would never have suspected from the course of the disease that any special form of treatment was being given".

Oxygen therapy was first used by Chambers, (12) who reported on it in 1890. This method of treatment is still widely used at present as a method of symptomatic treatment. McCurdy, (35) summing up the present day status of oxygen therapy, states that it is neither a pneumonia cure, nor a resuscitator of moribund patients, yet properly used, should always be a valuable aid to recovery and yield gratifying relief of symptoms.
History of Artificial Pneumothorax

In 1821 James Garson, a Scotch physician and physiologist, working in Liverpool, recognized the therapeutic possibilities of artificial pneumothorax and proved its practicability by animal experimentation. In 1822 he published an article advocating its use in the treatment of pulmonary tuberculosis. There is however, no record of his having tried it out on a human subject. Artificial pneumothorax was next heard of in 1832 when McRuer of Bangor Maine, suggested its use for the immobilization of a diseased lung. Again no one actually tried to carry out the treatment and again it was forgotten; this time for fifty years, until in 1882 when Forlanini of Italy suggested its use in the treatment of tuberculosis. (9)

Although there is reference in the literature to the introduction of air into the chest for the treatment of disease by the Greek physicians in the 4th century B.C., the modern practice of artificial pneumothorax dates, either back to 1888, when Forlanini introduced air into the pleural cavity of a patient suffering from a pleural effusion, (40) or back, as some authorities claim to 1884 when Potain a French
physician replaced the effusion in a case of hyd­
ropneumothorax with sterile air. (9) (3) Be that as it may however, it was first used for the collapse of a diseased lung in a person suffering from tuberculous by Forlanini, in 1892.

Apparently not knowing of the work of Forlanini and from independent thinking and observations, Murphy, of Chicago, reported in 1898, five cases of tuberculosis that he had treated with artificial pneumothorax. (33) (54) Tice began to use artificial pneumothorax about the same time as Murphy. Lempke, one of Murphy's pupils, carried on the work with artificial pneumothorax when Murphy dropped it. Since that time artificial pneumothorax has become a stand­
dard method of treatment in tuberculosis.
Pneumothorax in Pneumonia
(a review of the literature)

Therapeutic pneumothorax as a method of treatment in lobar pneumonia has been used by European physicians since 1921 when Friedemann (23) reported on its use in seven patients. He is given credit for being the originator of this form of pneumonia therapy. He found that when artificial pneumothorax was instituted on the affected side, severe pain subsided and respiration became quiet, with evident improvement in the general condition. In 1921 David (19) also reported six cases with favorable results. Two of his patients recovered by lysis, the other four recovering by crisis, all without complications.

In 1922 Wynn, (59) in this country, while not actually using pneumothorax, says, "In a few cases with very severe pain (pleurisy) I have separated the pleural surfaces by the introduction of a small quantity of oxygen. This is not difficult to those accustomed to the artificial pneumothorax treatment, and I was favorably impressed with the immediate relief given."

Ibrahim and Duken (29) used it in three infants with pneumonia with recovery; and Duken (20) later successfully treated five other children with pneumonia by
this method. In these patients, treatment was given late in the disease (twelfth to the thirty-fifth day) and must be regarded as being given for a complication of lobar pneumonia rather than for the acute pneumonia itself.

Leiberman and Leopold (33) in 1928, reported a series of cases of artificially induced pneumonia in dogs, which they treated with artificial pneumothorax. In a group of 36 animals, one-half were used as controls and one-half were given artificial pneumothorax. Of the 18 untreated control dogs, 13 died and 5 recovered; of the 18 treated with artificial pneumothorax on the second day, 15 recovered and 3 died. These workers report, "Our experimental study indicates that the introduction of air in the pleural cavity in the proper amount on the affected side produces, temporarily at least, a picture comparable to the crisis in lobar pneumonia and achieves an artificial limitation of an otherwise self-limited disease. In addition to this, in the experimental animal, this treatment appears to have a favorable effect on the bloodstream invasion."

In 1931 Taylor (53) used artificial pneumothorax in the treatment of pleurisy in pneumonia in
three cases with immediate relief of pain. In 1932 Coghlan (13), the first to use it in this country, used it in six cases of lobar pneumonia with complete recovery in five cases. In the one fatal case, an attempt was made to produce an immediate and complete crisis because of the desperate condition of the patient. 750 cc. of air was injected at one time; a very violent crisis set in, the patient perspired freely and the cyanosis became markedly diminished. A few hours later the pulse began to rise and signs of failure of the right ventricle appeared, the patient dying the next day. He says, "The fatality in this case can be attributed to an error in judgement in a difficult situation and lack of knowledge of the technique, owing to inexperience rather than to a defect in the method of treatment itself, for it was obvious that the pneumonic process was as well controlled by the artificial pneumothorax in this instance as in the other cases."

Coghlan's work has stimulated others to use this form of therapy and in the past Hanau (26) and Guadarrama (25) have commented favorably on its use in lobar pneumonia and the latter has published one case. Li (32) reported six cases from China with
one fatality in a case that also had an effusion in
the right pleural cavity. Anderson (1) reported
three cases with complete recovery. Perlroth and
Topercer (43) treated seven patients with one death.
The fatality occurred in a chronic alcoholic with
confluent bronchopneumonia and jaundice. Blake et
al (7) report favorably on twenty-two cases treated
by artificial pneumothorax with three deaths from
septicemia. In their series, treatment was instit­
uted on or before the fifth day. Moorman (36) in
1934 reported two cases which he had treated in 1930;
both of these patients were seen late in the disease
end in a very toxic state. Both died, but rather
from the advanced state of the disease than from the
pneumothorax treatment. Later in the year he report­
ed (37) eight more cases with one death. This patient
died of a massive cellulitis in the region of the par­
otid gland which failed to localize. He states that
the interval between fillings should not exceed twelve
to twenty-four hours. Behrend and Cowper (4) treat­
ed eleven cases with two fatalities, one of which
was moribund when first seen, and all who saw him
thought that his life had been prolonged; the sec­
ond died of pneumococcic meningitis on the twenty­
first day of the disease. In neither of these two
cases can the cause of death be even remotely attributed to the pneumothorax treatment. Isaacs et al (30) reported seven cases with four fatalities, end of this method of treatment say, "The treatments were not associated with any untoward effects, and no deaths or complications could be attributed to its usage, thus proving that pneumothorax treatment in lobar pneumonia is not associated with symptoms of shock." Crowell (18) reported three cases, all of which recovered without complications. Riggins (44) reports six cases with no deaths, the treatment having been started on the second to the fourth days. Hines and Bennett (27) reported twelve cases treated with pneumothorax with four deaths. Holmes and Randolph (28) treated eighteen cases by this method with two deaths. They believe that the total mortality in pneumonia is reduced by the use of pneumothorax treatment, and the chance of late complications such as abscess, bronchiectasis or unresolved pneumonia is probably reduced. Stoll et al (51) treated twenty-five unpicked cases of pneumonia with pneumothorax and had seven fatalities. Fentress (21) treated eleven cases by this method and had two deaths. In his series, treatment was begun from the second
to the eleventh day of the disease. He says, "The
day of treatment makes no difference, yet, as in
serum therapy, treatment should be begun as soon as
the diagnosis is made." Robbins (45) reported five
cases in which treatment was begun on the fourth to
the eighth day, with definite improvement in each
patient, none of whom died. Shipman and Cox (49)
had twenty-two cases with four deaths. Two of the
patients were moribund when treatment was begun.
These men say, "We believe that the toxemia may be
diminished in certain instances and the subsequent
course changed for the better. It is our impression,
however, that once air is introduced into the pleural
cavity, it should be given for at least two weeks to
allow the inflammatory reaction in the pleura to sub-
side. Otherwise, sterile effusions, which prolong
the patients stay in the hospital, may be expected
to develop."
Pathogenesis of Pneumonia

The exiting cause of lobar pneumonia is the pneumococcus but, predisposing causes also play an important part. When virulent pneumococci get into the throat the germs do not pass down the bronchial tree to the lungs. In man accessory factors are necessary to lower the resistance of the patient. The best recognized of these are; profound fatigue, chill, injury to the chest, severe fractures, the inhalation of an anesthetic, and some other infection ( influenza, the infectious fevers, etc.).

The route by which the pneumococci reach the lung has long been a matter of dispute. The opposing views are; (I) That the infection is bronchogenic, passing down the bronchi into the lung; (II) that it is hematogenous, passing from the throat into the bloodstream and reaching the lung by that route. The work of Blake and Cecil (6) has proven quite conclusively that the former theory is correct.

The pneumococci penetrate the walls of the larger bronchi, enter the lymphatics of the interstitial framework of the lung, and by this route reach the walls of the alveoli. They pass through these walls and enter the alveoli in large numbers.
Meanwhile, invasion of the blood stream has occurred, probably by way of the lymphatics, the organisms being found in blood culture in from six to twenty-four hours after injection. It is this early blood infection that gave rise to the idea of the hemato-
genous mode of pathogenesis. The fundamental feature of the process is the interstitial invasion of the lung, and it is from the interstitial tissue, not from the bronchioles, that the alveoli are reached. The earliest lesion is an interstitial inflammation affecting the perivascular and peribronchial connective tissue, but this inflammation is not severe and there is no destruction or tissue necrosis.

According to Boyd, (8) the essential pathological feature of pneumonia is an out-pouring of an inflammatory exudate into the alveoli in response to the irritation produced by the pneumococci. The alveoli are filled by this exudate, the air is displaced, and the lung or part of it is converted into a solid and airless organ. This process is known as consolidation or hepatization because the lung becomes like liver in consistence. The process is a progressive one commencing at the hilus and sweeping out to the periphery, involving one or more lobes.
and sometimes both lungs. It follows that one part of the lung may be at one stage while another part is at another.

The gross appearance of lobar pneumonia is very characteristic. By the end of the second day the stage of red hepatization has been reached and the affected part is consolidated, swollen and reddish brown in color. Only one part of a lobe may be consolidated, and the line of separation between the two parts is remarkably sharp. When the stage of grey hepatization is reached, usually by the fifth day, the consolidated part is grey or brownish-grey in color, and it is now distinctly moist owing to softening of the exudate. The bronchial lymph nodes are enlarged and congested. In the stage of resolution, the lung has become soft and translucent, and may be almost jelly-like in consistence.

For the production of the acute syndrome known as lobar pneumonia, another factor besides the presence of the pneumococcus is necessary; this is the occlusion of a bronchus by the pneumococccic bronchial exudate. (17) This marks the onset of a clinical syndrome in which are united the clinical features of an acute obstructive atelectasis and an acute
obstructive atelectasis and an acute pneumococcic cellulitis. The factors which make this occlusion possible are, the viscid and tenacious nature of the pneumococcic sputum, the diminished force of expectoration in a toxic individual, perhaps, the edema of the bronchial mucosa, and possible damage to the ciliated epithelium. Coryllos and Birnbaum (15) (16) (17) have done a great deal of work, principally animal experimentation, on massive atelectasis and lobar pneumonia. In massive atelectasis, they find, the bronchus is often occluded by a relatively poorly infective secretion, and the toxic symptoms are absent; whereas in lobar pneumonia, the viscid occluding secretion is infected with pneumococci from the start, the number and virulence of the organisms determining the degree of toxicity of the disease. The obstruction having once set in, absorption of the alveolar air proceeds; simultaneously the infection is spreading to the periphery of the lobe by septal and interalveolar lymphatics.

Absorption of endotoxins in pneumonia explains the toxic element which is usually absent in simple atelectasis. The topography and size of the bronchus occluded, the duration of the occlusion,
the virulence of the infecting organisms and the rapidity of the development of the pneumococcic cellulitis will determine the features of lobar pneumonia. Occlusion of a large bronchus with pneumococcus of a low virulence will produce a syndrome closer to a massive atelectasis than to lobar pneumonia, while occlusion of a bronchus with highly virulent pneumococcus will cause a more typical and toxic lobar pneumonia.

In its earliest period, lobar pneumonia presents radiographic and auscultatory symptoms of a lobar atelectasis, namely; a wedge shaped shadow, more or less marked, absence of respiratory breathing, with a rather tympanitic dulness over the effected area, and often a homolateral displacement of the mediastinum and elevation of the diaphragm on the affected side. Simultaneously, the cellulitis spreads from the hilar portion of the lobe to the periphery accompanied by the phenomena of pneumococcic inflammation, namely; congestion, interstitial and lymphatic infiltration, vascular engorgement and production of the characteristic fibrinous exudate.

The roentgram and physical signs of lobar atelectasis, which are due to the obstruction of a
central bronchus, appear to proceed from the periphery to the center of the lobe, because the absorption of the alveolar air is more marked in the periphery of the lobe, where the alveoli are more numerous. The symptoms of the pneumococcic cellulitis, on the contrary, spread from the central infectious "plug" to the periphery, following the lymphatic and interstitial tissue, so that after the initial and transitory period of simple atelectasis there is a period of pneumococcic invasion. Engorgement, red hepatization, grey hepatization and resolution, follow one another from the center to the periphery. When the hilar portion of the lobe shows engorgement and even red hepatization, in the periphery, we still find alveoli, more or less atelectatic, but free from exudate. When grey hepatization starts in the hilar portion of the lobe, the more peripheral parts show red hepatization; when, after the resolution, the proximal portion of the lobe has drained and become aerated, the peripheral part still contains fibrinous exudate, more or less liquified by the action of the proteolytic enzymes produced by the white blood cells. Thus the shadow persists even after the crisis in pneumonia and gradually disappears from the center.
to the periphery.

The simultaneous development of atelectasis and pneumococcic cellulitis explains the less marked displacement of the mediastinum in pneumonia than in massive atelectasis. Besides, the lesion is often limited to one lobe, and the inflammatory swelling of the interstitial tissue and alveolar exudation prevent shrinkage of the lung to the extent found in simple atelectasis.

The crisis or lysis is due to the disintegration of the fibrinous exudate which characterizes grey hepatization, and to the sudden liberation of a large bronchus, thus allowing sudden drainage of the affected lobe. If the liberation is complete and rapid, a crisis follows; if it is gradual, the healing occurs by lysis. (17)
Rationale

Coghlan, (13) in his original paper claimed the following advantages for the treatment of lobar pneumonia with artificial pneumothorax; (I) that it separated the inflamed pleural surfaces, relieving pain and allowing easy respiration, (II) that it put the inflamed lung at rest, and (III) limited the flow of blood through the pneumonic lung, thereby diminishing anoxemia and interfering with the passage of toxins into the general circulation. Bullowa and Meyer (10) state that the mode of action of artificial pneumothorax is "a natural surgical method of placing the lung at rest, and is comparable to splinting an infected hand to avoid blood invasion and spread of the infection."

Moorman (37) gives five indications for pneumothorax in the treatment of lobar pneumonia. These are, (I) atelectasis, (II) pleurisy, (III) to separate inflamed surfaces, (IV) to provide local rest and (V) early treatment.

Atelectasis constitutes a very definite indication for artificial pneumothorax in pneumonia. This condition, depending upon the degree and extent, may give rise to symptoms and physical signs ranging
from slight intrathoracic distress with possibly slight deviation of mediastinal structures toward the affected lung, to pronounced pain and distress (so-called respiratory catastrophe) with marked displacement of heart, great vessels and trachea toward the affected side.

Since the above phenomena are due to intrathoracic negative pressure, the introduction of a few hundred cubic centimeters of air into the pleural space will promptly restore the normal relationship of the mediastinal structures and relieve the pain and distress. Griffith, (24) Wu, (58) and Robertson et al, (46) have shown by their studies that atelectasis is not uncommon in pneumonia.

Pleurisy pain which is so common and often so persistent in pneumonia may be accepted as a direct indication for pneumothorax. The relief of pain and the resulting comfort with peaceful sleep, obviating the necessity of large doses of opium and other sedatives, must favorably influence prognosis. There is a marked reduction in the frequency and severity of cough and the associated stabbing pain is reduced.

It may be possible to limit the spread of pleural inflammation and decrease the incidence of
empyema by interposing a cushion of air between the two pleural surfaces and thus intercepting the constant friction and agitation which must result in aggravation and dissemination of infection.

When we consider the nature of the infection, the structure of the lungs, and the influence of gravity, plus the possible peripheral suction due to atelectasis, it seems quite reasonable to believe that by means of collapse therapy, secretions and inflammatory exudate may be expressed and their progress toward the periphery retarded. The absorption of toxins will be lessened due to the diminished flow of blood and lymph. (48) (2) (36).

Artificial pneumothorax not only brings rest to the inflamed lung and pleura, but through relief of pain and added comfort it favors general rest. Local rest is important in the treatment of inflammation in any organ; it is doubly important in the inflamed lung where, in spite of the ultimate inhibitory action on the part of nature, function goes on until curtailed by artificial means.

It is generally agreed that artificial pneumothorax is particularly indicated early in the course of pneumonia. It seems reasonable to expect better
results if collapse can be induced before the lung
tissue is completely consolidated and the inflamma-

dory process is widely distributed; before the patients
vital forces become depleted by toxemia and before
the development of cardio-respiratory distress due
to atelectasis and pleurisy. Clinical experience,
plus roentgen ray evidence indicates that the more
complete the consolidation, the more incomplete the
collapse from pneumothorax. Lieberman and Leopold
(34) state that artificial pneumothorax should be
used early to be effective; in fact, they find evi-
dence to justify its being contraindicated after the
third day of the disease.

However, one must not lose sight of the fact
that various complications, disadvantages and pos-
sible dangers have been mentioned in the literature.
Coghlan, (13) in his paper mentions pyopneumothorax
and cardiac collapse as possible dangers in the use
of this method of treatment. Pyopneumothorax is
easily prevented by the use of aseptic technique, as
was pointed out by Coghlan himself. Cardiac collapse
can occur as a result of a crisis which is too severe.
This can be controled safely by lessening the amount
of air administered at the first injection.
Bullowa and Meyer, (10) in their paper on the hazards of this method of treatment, list eight complications and disadvantages, which are: "(a) Late invasion of the bloodstream in spite of collapse, (b) Invasion of another lobe on the collapsed side, (c) Invasion of the contralateral side, (d) Empyema, (e) Induced rupture of the lung, (f) Extreme mediastinal shift, with death, (g) Harmful delay in application of serum therapy in suitable cases, (h) Fatigue of patients by frequent manipulations and roentgen examinations."

Late invasion of the bloodstream in spite of collapse treatment does not occur when collapse is obtained on or before the third day of the disease. According to Lieberman and Leopold, (33) (34) tendency toward bloodstream invasion is lessened with pneumothorax, although it probably has no effect upon a preexisting bloodstream infection.

Spread of infection, either homolateral or contralateral, is probably not influenced by this method of treatment, when the treatment is instituted before the third day. However, there is some evidence to prove that the chances of spreading the infection are increased by artificial pneumothorax after the third day.
According to Roorman (37) the prevention of empyema is one of the indications for the use of artificial pneumothorax. In children however, this treatment is contraindicated because it has been found that it seems to increase the occurrence of empyema. Also, the tendency toward spontaneous pneumothorax is greater in children than in adults.

Induced rupture of the lung is a more or less mythical complication or danger. Its possibility being practically precluded by the proper use of the manometer while the treatment is being given.

Extreme mediastinal shift as a dangerous complication and a cause of death can be dismissed by reiterating the necessity for taking frequent manometer readings during the injection of the air. Paying close attention to the patients subjective complaints at this time is another safety factor and as such is probably as important as the manometer readings, because pain and distress are the first signs of mediastinal shift. Injection of more than two or three hundred cubic centimeters of air is not necessary for adequate collapse, and if exceeded carries with it the danger of mediastinal shift.

That harmful delay in the application of serum
therapy is entailed by the use of artificial pneumothorax is a mistaken claim because serum treatment need not be omitted or delayed when artificial pneumothorax is used. In fact, it may be soon found that a combination of these two methods of treatment is the best yet to be proposed.

Fatigue of the patient by frequent manipulations and roentgen examinations need not be a disadvantage, as claimed, because of the simplicity of these manoeuvres, and the infrequency with which they must be carried out. The x-ray is only needed for confirming the diagnosis and locating the exact site of the consolidated area. This can be accomplished by the portable equipement at the patient's bedside and with no discomfort or exertion on the part of the patient. The pneumothorax itself requires only about five minutes for induction and is done without moving the patient from his bed.
Technique

It should be remembered that the induction of artificial pneumothorax, even in the hands of a skillful operator, is a hazardous procedure. We cannot escape the danger of sudden death from air embolism, or so-called pleural shock. Traumatic or spontaneous pneumothorax with bronchial fistula may be accompanied by extreme shock, and empyema is a dangerous complication. These are the most hazardous complications and their incidence may be greatly increased by inexperience, lack of skill, and improper technic. A working knowledge of the pneumothorax apparatus to be used is prerequisite.

It is important to use at the first operation a needle of proper size, gauge 18, with a blunt point or a bevel approximating 45 degrees. The stilet should be well fitted. If these precautions are disregarded, grave damage to the visceral pleura may result. Such accidents may occur even though every safeguard is employed.

In pneumonia it is not necessary to turn the patient on his side with a pillow under his thorax in order to bring the field of operation into bold relief and to widen the interspaces. Pneumonia is
an acute inflammatory disease and demands gentle handling. With rare exceptions, the best position for the operation is the position assumed by the patient when left to his own wishes. The field of operation should be preferably in the lower half of the thorax, anywhere above the diaphragm between the mammary line and the paravertebral line. If the patient is lying on his back, the needle may be introduced in the anterior axillary line, in the third to the fifth interspaces. If the patient is in the lateral position, affected side down, because of the pleural pain, a change of position may be necessary in order to select a desirable field for operation.

The patient's interests are safeguarded by attempting, as far as possible, to keep the field of operation away from the location of gross disease as determined by the signs of consolidation, pleural friction, and roentgen ray findings. If introduction of the needle should traumatize the visceral pleura, repair without infection and serious damage will be more likely to occur if the point of attack is outside the field of acute inflammatory reaction.

The field is prepared with alcohol and iodine and at the chosen point the local anesthetic is
introduced. (novocaine, $\frac{1}{2}$ of 1% or procaine hydrochloride 2%) With a small hypodermic syringe and a fine sharp needle, the skin wheal is made and then the needle is directed perpendicularly toward the pleura, with progressive infiltration, until the parietal pleura is anesthetized. When the parietal pleura is penetrated, the piston of the syringe voluntarily descends with each inspiration in response to the intrathoracic negative pressure. However with increased resistance due to thickened pleura, there is no indication of negative pressure manifest at this time.

After anesthesia, the needle used to insert air is introduced. Since the point of the needle is relatively blunt, the skin may be penetrated with a cataract knife or preferably a sharp pointed needle with caliber corresponding to that of the needle that is to be used. When the pneumothorax space is sufficient to obviate the danger of contact with the visceral pleura, an ordinary needle may be employed for refills. Manometer readings bear the same significant indications routinely accorded them in tuberculosis cases and should be more highly respected because of the increased danger of dealing with an acute
infectious disease. Free oscillations should be secured before the air is introduced. As air is admitted, frequent manometer readings are necessary to avoid the danger of unexpected positive pressures. Behrend and Cowper (4) advocate manometer readings after every 50 cc. of air is admitted. It may be safe to close with neutral pressures, but, with few exceptions, positive pressures should be avoided.

There can be no inflexible rule with reference to the amount of air to be given, due to the difference in, the size of the thoracic cages of the various patients, the elasticity of the pleura, and the stage of development of the consolidation process. Experience with pneumothorax in other conditions suggests that the closing pressure accompanying the initial filling should be negative, or at least not above neutral, and the amount of air introduced should not exceed 300 cc., even though the pressure remains negative. Behrend and Cowper (4) however, feel that from 400 to 500 cc. of air will usually produce the desired effect without producing a mediastinal shift. This would seem a safe rule to follow in pneumonia. While there is some difference in opinion, a review of the reported cases indicates that with few
exceptions, these bounds have not been exceeded. The amount of air introduced has varied from 80 to 700 cc., and the number of fillings from one to three. The interval between fillings has varied from twelve to thirty-six hours. The probable increase in the rate of absorption in pneumonia, (13) (34) and the desirability of maintaining a fair degree of collapse, suggest that the interval between fillings should not exceed twelve to twenty-four hours. (37)

Worthy of consideration is the fact that in the employment of artificial pneumothorax the operator can promptly retrace his steps by the withdrawal of the air from the pleural space.
Summary

In 1892, Osler (41) said, "Pneumonia is a self-limited disease, and runs its course uninfluenced in any way by medicine. It can neither be aborted nor cut short by any known means at our command." Surgery now has a way to cut short the course of pneumonia and to relieve its attendant suffering.

At the present time, about 200 cases of lobar pneumonia which have been treated with artificial pneumothorax have been reported in the literature. The mortality for this group of cases has been practically the same as the general hospital mortality from this disease under all other forms of treatment. It seems likely however, that this mortality rate will decline as this form of treatment gets out of the experimental stage which it is in at present. Moreover, it has not been a standardized or even thoroughly understood method of treatment. Everyone using it had his own ideas about how and when to use it. As a result many mistakes in judgement and technique have undoubtedly been made. There have been a sufficient number of cases published now, that there should no longer be an excuse for patients dying due to lack of clinical skill and judgement on the part of the operator. One should keep in mind Coghlan's
(13) admonition that, "Lobar pneumonia is emphatically not the disease on which to gain a knowledge of the technique of artificial pneumothorax.", which contains as much truth today as the day he wrote it.

It seems fairly well established that the pneumothorax treatment does not increase the amount or number of complications of pneumonia. In fact, when used properly, it will without a doubt reduce the number of complications.

The only contraindications to its use seem to be in small children, and in any one after the third day of the disease. When used in children, it seems to increase the tendency to form spontaneous pneumothorax, a fact which is against its use. Unless used before the third day, there seems to be a slightly higher incidence of spread of the involvement, either to another lobe on the same side, or to the other side.

The greatest obstacle to effective early treatment is the presence of preexisting fibrous adhesions in almost 50% of adults of middle age. This barrier is almost insurmountable in patients treated after the third day because the pleural reaction after this time adds new adhesions to the predictable 50% already present, thus making complete compression
and artificial crisis virtually impossible.

Artificial crisis can only occur with a free pleural cavity and may be expected in 50% of cases treated before the fourth day. This statement is predictable and is in accord with clinical experience. Nothing is accomplished by compression treatment unless artificial crisis is achieved. (34)

Specific serum therapy is applicable to about 50% of all patients with lobar pneumonia. Artificial pneumothorax is capable of producing an artificial crisis in about the same proportion of cases and can be used in any and all types of lobar pneumonia if the involvement is unilateral. Both specific serum and pneumothorax must be used early to be effective. Artificial pneumothorax, properly used is a real and permanent addition to the treatment of lobar pneumonia.
Appendix
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