REPORT

of

Committee on Thesis

The undersigned, acting as a Committee of the Graduate School, have read the accompanying thesis submitted by John William Snyder for the degree of Master of Science in Surgery. They approve it as a thesis meeting the requirements of the Graduate School of the University of Minnesota, and recommend that it be accepted in partial fulfillment of the requirements for the degree of

Master of Science in Surgery.

Carl A. Hedblom

F. C. Mann

Willis S. Lemon

F. H. Scott.

Graduate School, University of Minnesota.

Date:

This is to certify that John William Snyder, a candidate for the degree of Master of Science in Surgery, has passed the final written examination for the major in the Department of Surgery.

E A Graves

For the Major Department.

Graduate School, University of Minnesota.

Date: Nov. 28, 1922.

This is to certify that John William Snyder, a candidate for the degree of Master of Science in Surgery, has completed the requirements for the minor in the Department of Pathology.

Louis B. Wilson

For the Minor Department.

THE UNIVERSITY OF MINNESOTA

GRADUATE SCHOOL

Report

of

Committee on Examination

This is to certify that we the undersigned, as a committee of the Graduate School, have given John William Snyder final oral examination for the degree of Master of Science in Surgery.

We recommend that the degree of Master of Science in Surgery be conferred upon the candidate.

Earl G. Hedblom, Chairman

C. M. Jackson

R. H. Scott

H. C. Mann

C. L. Cameron

Willis S. Lemon

E. J. Bell.

Date Dec. 15, 1922

10-20 5M

THESIS

# STUDIES IN INTRAPLEURAL TENSION.

John William Snyder, M.D.

Submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

October, 1922.

MOM

Intrapleural tension may be defined as that force which maintains expansion of the lungs under normal conditions. From another viewpoint intrapleural tension may be considered as a potential negative pressure existing within the pleural cavities dependent upon and caused by the normal elasticity and contractability of the lungs.

Our studies have been concerned with changes produced in the normal intrapleural tension by various pathological conditions. In addition we have studied the mediastinum in the dog and man with the view of determining:

1. If there were any essential difference in the mobility of the mediastinum in the dog and man.

2. If the stability and relative strength of the structure in the two instances were comparable.

3. If a change in the intrapleural tension of one side produced an equal change on the opposite side and if so by what type of mechanism.

4. If it were possible to produce a unilateral pneumothorax in a dog.

5. If experimental results obtained in the dog, especially in relation to surgical pneumothorax, could be applied to man.

The present day conception of intrapleural tension and the mechanics of respiration have passed through many stages of development. We will briefly review the important work on this subject.

In ancient times the view was held that inspiration and expiration were due to active contraction and relaxation of the lungs similar to the movements of the heart. In the second century Galen stated that this was not true but that the lungs followed the movements of the thorax in a passive manner. He also noted the muscular contraction of the diaphragm.

For the following 1500 years this view was disputed and not generally believed. The phenomena of respiration were explained in three ways.

1st. Expansion of the chest was thought to push the surrounding air away from the chest and this force was in turn transmitted to the adjacent air until it was literally driven into the lungs.

2nd. That the chest behaved like a bellows which became filled because it was dilated.

3rd. That the lungs resembled a bladder whichwas dilated because it was filled.

In 1667 Richard Lower described the act of respiration and produced respiratory difficulty in a dog by division of the phrenics.

For the following century hypotheses and speculations prevailed. Air was generally believed to be present between the two pleural surfaces. Hamberger as late as 1751 held that a layer of air was present between the visceral and parietal pleuras and that the lung functioned by active contraction.

Borelli in 1743 clearly described the passive movements of the lung in respiration.

Boyle in 1744 adopted the view of the bellows action of the lungs. He believed the diaphragm the principal instrument in ordinary respiration although the intercostal and perhaps other muscles might assist.

Haller in 1746 staunchly combated the view that the pleural cavities normally contained air. He also confirmed the passive role of the lung in respiration. His conclusions were reached by animal experimentation. The

parietal pleuras in living animals were freed from overlying structures without injury of the pleura. He was able to see the lungs in contact with the pleura and could follow their movements with respiration.

Meckel, 1759, described a case of spontaneous pneumothorax which resulted in death. He concludes, "This very rare observation is not useless to refute the opinion of the presence of an elastic fluid between the lung and the pleura, and necessary for the mechanism of respiration. It is rather clear - - - that the expanded air between the pleura and the lung, far from aiding respiration, placed there an obstacle by compression of the lung."

Hewson, 1767, proposed paracentesis for evacuating air from the pleural cavity which he believed could cause compression of the lung and dangerous respiratory symptoms in a manner similar to effusions of serous or purulent material. He tried to produce pneumothorax in animals by wounding the lung but was unsuccessful. Regarding wounds of the chest he states, "It has been observed that in gunshot wounds where the ball has passed through the lungs, and in other large penetrating wounds of the chest the patient breathes more easily when the external wound has been covered and has hardly been able to breathe when it was open. In these circumstances the difficulty in breathing seems to be owing to the air getting into the cavity of the thorax in inspiration instead of entering the lungs by the trachea so that the lungs are not distended by the expansion of the chest and the patient, while such a wound is uncovered, is deprived of the use of the lobes of that side either partially or entirely according as the wound of the thorax bears a greater or less proportion to the branch of the trachea of that side in which the wound is." It will be noted that Hewson's conclusions regarding wounds of the chest, bear a striking similarity to the conclusions arrived at by military surgeons during the late war.

John Bell seems to have had a very clear conception of the mechanics of the thorax. Speaking of a large open pneumothorax he states, "The thorax

therefore, has nothing to do with the lungs but like a pair of bellows having a large air hole which admits air every time the breast rises - - -. There is no vacuum to move the lungs. The lungs whether they be entire or whether they be wounded always lie collapsed."

Again speaking of closed pneumothorax, he states: "In a punctured wound (of the lung) - - - at every stroke of respiration more and more air is drawn out from the lung till at last the blood and especially the air, are so condensed that they not only oppress that side of the lung, but by hindering the free play of the diaphragm and loading the mediastinum, they oppress also the other lung - - - - and the patient dies." This is the first mention so far as we have been able to discover by a review of the literature of a mechanism by which the opposite lung is interfered with in pneumothorax.

During the past century investigation of the normal and pathological physiology of the respiration has been carried out along many lines. Researches of this period which have to do with the special field of this paper namely, Intrapleural Tension, can be more conveniently considered under separate divisions depending upon the special methods employed or the condition investigated. We may arbitrarily divide them as follows:

A - Determinations of pulmonary elasticity.

B - Investigation of pressure relations in pleural effusions.

C - Indirect estimation of intrapleural pressures.

D - Direct measurements of intrapleural pressures.

E - Pressure relations in pneumothorax.

A - Determinations of Pulmonary Elasticity.

The elasticity of the lungs of animals was first studied by James Carson in 1820. The trachea of dead animals were connected by a tube of dried intestine to a glass globe and this in turn, to a water manometer. The diaphragm was then incised from the abdominal surface and both pleural cavities

opened. The amount of pressure necessary to prevent collapse of the lungs was then determined by the manometer. In three freshly killed oxen, a pressure equivalent to one and one-half feet of water was not sufficient to do this. In a calf he found fourteen inches of water prevented pulmonary collapse. A large dog required ten inches of water pressure while rabbits and cats varied from six to ten inches.

Donders, 1849, employed human cadavers in a somewhat similar manner. He fastened a tube tightly in the trachea and connected it with a manometer. Both pleural cavities were then opened and the lungs allowed to collapse. The elevation of the column of fluid in the manometer registered the elastic recoil of the lung. For man he found it to be 3 to 7 cm. of water.

Hutchinson repeated these experiments and obtained slightly higher values 13.4 mm. Hg. or 18.2 cm. of water. He, however, believed that the lungs of cadavers contained only residual air and therefore first inflated the lungs with 1500 cc. of air making up the supplemental air which he thought absent. Heynsius later claimed this to be an error.

Perl, twenty years later, did similar experiments on 100 consecutive cadavers opening one pleura at a time and observing the pressures obtained from the collapse of each lung. He found that the pressures from opening each pleura were sometimes about equal but frequently the opening of one pleura went a long way to satisfy the elasticity of both lungs.

Heynsius adapted this method to animals. The animals were first killed and traction on the diaphragm used to simulate inspiration. For dogs the average inspiratory pressure was 7 mm. Hg. and expiration 4 mm. Hg. Rabbits gave inspiratory pressures of 7.6 mm. Hg. and expiratory of 2.5 mm. Hg.

Sussdorf in 1890 applied the same method to horses and found with inspiration a pressure of 28 mm. Hg. and with expiration 7 mm. Hg.

The study of the intrapleural tension in infants furnishes an

interesting contrast to the results obtained in adults.

Bernstein, using Donders method in still born children, found that there was no elastic recoil of the lungs, but by first distending the lungs with air until the thorax was noticeably distended, a pressure of 6 to 7 mm. Hg. could be obtained. After opening the pleural cavities the lungs collapsed but not to their original volume.

L. Herman claimed that in infants who lived for some time after birth, the lungs entirely filled the thorax and did not collapse on opening the pleural cavities. Employing Donders method he was unable to obtain any pressure as shown by the manometer. He stated that Bernstein's results were due to over distention of the lungs. Later Lehmann, a student of Bernstein, and Bernstein himself confirmed Herman's results.

B – Investigation of Pressure Relations in Pleural Effusions.

The investigation of conditions existing in the pleural cavities under pathological conditions was probably stimulated by the introduction of aspiration as a treatment for pleural effusions. H. Quincke in 1882, under title of "Zur Behandlung des Pleuritis", discusses the treatment of effusions by aspiration. He also states that in a case of empyema with a persistent sinus he was able to determine the intrapleural pressure by means of a manometer. In a later article he gives the pressures obtained in pleural effusions at the time of aspiration. He found the initial pressures to vary from 0 to plus 26 mm.Hg. with an average of plus 10 mm. Hg. and only one case showed a negative pressure with inspiration. After aspiration most of the cases showed a negative pressure of minus 12 to minus 15 mm. Hg. He states that the pressure depends on three factors:

1. Elasticity of the lung.
2. Elasticity of the chest wall.
3. Height of the column of fluid.

Peyrot in 1876, studied the effects of pleural effusions on the lungs, thorax, mediastinum and diaphragm but made no manometric observations, except in one case of pyopneumothorax.

Leyden, in 1878 found the initial pressures in pleural effusions varied from 0 to plus 28 mm. Hg. and only a few showed negative pressure on inspiration. After removal of the fluid he obtained a negative pressure of minus 24 to minus 28 mm. Hg which increased with inspiration to minus 32 to minus 42 mm. Hg.

Homolle one year later reported initial pressures in similar cases of minus 2 mm. Hg. to plus 30 mm. Hg with a negative pressure after aspiration of minus 33 to minus 38 mm. Hg.

Schreiber in 1883 reports three cases with sanguinous and purulent exudates in which the initial pressures varied from 0 to plus 12 mm. Hg. but which with ordinary respiration changed to minus 4 to minus 8 mm. Hg. and with deep inspiration to minus 24 to minus 32 mm. Hg. All became markedly negative with removal of 400 to 1,000 c.c. of fluid.

E. Aron in 1891 reported the pressure findings in cases of empyema using a manometer containing glycerine. Graphic tracings were taken of the respiratory oscillations. He found the pressure constantly negative varying from minus 3.9 with expiration to minus 6.85 with full inspiration. He also found variations depending on whether the patient were sitting up or lying down.

James Calvert in 1892 points out that the pressure at the surface of an effusion, where the liquid is in contact with the lung, must be different from the pressure at the level of the diaphragm by the weight of the superincumbent fluid so that the pressure upon the lung may be negative or zero when the pressure at the diaphragm is positive.

Pitres, 1896, reports a series of cases with effusions of varying size. He believes the pressures obtained are proportional to the amount of fluid

present. Cases with more than 2,000 c.c. show a positive pressure of plus 20 to plus 48 mm. Hg. while effusions of 1,000 to 2,000 c.c. give pressures from plus 8 to plus 22 mm. Hg. Effusions below 1,000 c.c. were often negative or weakly positive. There are frequent exceptions to this general rule but in the main he believes it holds good. After evacuation of the fluid the pressures become negative.

West found initial pressures of minus 1 to plus 18 mm. Hg. in serous effusions and plus 3 to plus 16 mm. Hg. in purulent effusions.

L. Bard criticises the instruments and methods previously used in determination of the tension of pleural effusions. He finds many possible causes for error. To obviate these objections he employed a tube of glass connected with a trochar and permitted the fluid contained in the pleural cavity to rise in the glass tube on making the pleural puncture. The point of puncture was taken as zero and the fluid elevation above this point represented the positive pressure existing at that point. In case of negative pressure he took the precaution of emersing the open end of the glass tube in water before puncturing the pleura.

The pressure found is, he believes, the sum of two factors, the one always positive which is the height of the column of fluid above the point of puncture and the other always negative which is the surface pressure or normal physiological tension. This latter is the result of the normal elasticity of the lung and the chest wall itself. He found that pressures varied with the point of puncture and with the attitude of the patient whether sitting or lying. The surface pressure could be approximated by determining the upper limits of the effusion by percussion and subtracting the distance between this point and the point of puncture from the pressure obtained by his manometer. If he found the surface tension to be positive it indicated a complete suppression of the pulmonary elasticity.

In seventeen cases of pleurisy, he found the average pressures varied

from plus 2 to plus 8 becoming negative with inspiration in some cases.

Weitz using Bard's method reports pleural pressures in 108 cases. He divides the exudates into large, medium, and small, according to the amount of fluid obtained. The large exudates were constantly positive, the medium less markedly so and the small exudates frequently negative. Exudates of some duration as a rule, gave higher positive values than the more recent, and high pressures were especially marked in empyemas and effusions resulting from malignant disease. After removal of the exudate in the majority of cases the pressures became negative averaging minus 11 cm. water. In very large exudates, he believes the opposite lung exerts a negative pressure through the mediastinum while with smaller exudates where the lung is not collapsed it retains considerable elastic tension within itself and is the main factor in the negative tension present.

Flurin and Rousseau also report measurements of pleural pressures in effusions. They employed an aeronoid type manometer which only registered positive pressures so that their observations are of limited value.

C - Indirect Estimation of Intrapleural Pressures.

Estimations of the negative tension existing within the chest but outside of the pleura cavity itself have been made by various observers.

Adamkiewicz and Jacobson fastened a blunt cannula in the pericardial cavity of various animals, sheep, dogs, and rabbits. The cannula was connected to a manometer containing Hg. With quiet breathing, he found a negative pressure of 3-5 mm. Hg.

Heger and Spehl introduced a tube into the pericardium and connected it with a manometer. In three experiments they found a respiratory oscillation of minus 5, minus 2, minus 4 mm. of Hg. averaging 3.6 mm. Hg.

Luciani and Rosenthal introduced an elastic tube into the esophagus of animals and man, and connected it to a manometer. In animals the average pressure was minus 3 mm. Hg. In man they found a negative pressure of 4-6 cm.

of water but by deep inspiration a negative pressure of minus 10 to minus 12 cm. $H_2O$ could be obtained. In a later article, Rosenthal amplified these results and showed that while in the esophagus the tube always recorded a negative pressure as soon as it passed into the stomach it became positive.

Julius Schreiber followed Rosenthal's methods but obtained slightly higher results in man. With quiet breathing he found a pressure of minus 6 to minus 7 cm.

S. J. Meltzer, believing the thickness of the esophageal wall interfered with accurate measurements, introduced a catheter into the posterior mediastinum beside the esophagus. He found variable pressures at different levels.

D - Direct Measurements of Intrapleural Pressures.

Direct measurement of the intrapleural tension was first attempted by D'Arsonval in 1877. He placed a blunt trochar in the right pleural cavity and arranged to draw off the air admitted by this procedure. He then connected the trochar to a manometer. In a dog, inspiration produced a pressure of minus 20 to minus 22 cm. of water and expiration minus 15 cm., an average respiratory oscillation of 6 cm. of water or 4.4 mm. Hg.

Fredericq followed a similar procedure introducing a glass tube into the pleural cavity without admitting air. He observed only slight respiratory oscillations but does not give any figures.

E. Aron made observations on a 34 year old healthy man. He found the absolute value of the intrapleural pressure impossible to give as it depended on the depth of breathing and this was variable. The average of thirty-six measurements for quiet breathing gave inspiration minus 4.64 mm. Hg. and expiration minus 3.02 mm. Hg. The highest pressure with inspiration was minus 5.09 mm. Hg. and the lowest with expiration was minus 2.54 mm. Hg.

Van der Brugh experimented with narcotized dogs and found in six dogs an average expiratory pressure of minus 7.9 cm. of water.

R. Bendele obtained the pleural pressures in living horses. He found it varied from minus 50 to minus 60 mm. Hg. With forced expiration it could become positive but always less so than the intrapulmonary pressure.

V. Muralt in conjunction with artificial pneumothorax therapy for pulmonary tuberculosis made graphic curves of the normal intrapleural pressures. He found the pressure varied from minus 10 to minus 2 cm. of $H_2O$ reducing to minus 14 with a deep inspiration and changed to plus 20 by coughing. On holding the breath in expiration the pressures varied minus 2 to minus 3 cm. $H_2O$.

Burrell and Macaulty report pressure readings in four cases during the introduction of artificial pneumothorax as follows:

| | Case 1. Initial operation | Case 2. Initial Operation | Case 2. Three months later |
|---|---|---|---|
| Initial pressures | -14 -12 cm. $H_2O$ | -7 -5 cm. $H_2O$ | -3 -0 cm. |
| After 100 cm. air. | -10 - 9 " | -7 -2 " | -3 -0 " |
| " 200 " " | -10 - 8 " | -7 -2 " | -2 Plus 2 cm. |
| " 300 " " | -9 - 6 " | -6 -2 " | 0 " 2 " |
| " 400 " " | -8 - 6 " | -6 -1 " | Plus 1 " 4 " |
| " 500 " " | | | " 2 " 5 " |
| " 600 " " | | | " 4 " 7 " |
| " 700 " " | | | " 6 " 9 " |
| " 800 " " | | | " 8 " 11 " |
| " 900 " " | | | " 10 " 13 " |

| | Case 3. Refill | Case 4. Refill |
|---|---|---|
| Initial pressures | -12 -8 cm. $H_2O$ | -6 -0 cm. $H_2O$ |
| After 600 cm. air | -3 -0 " | |
| " 700 " " | -2 -0 " | |
| " 800 " " | Plus 2 Plus 2 cm. | -2 plus 4 cm. |
| " 900 " " | " 4 " 2 " | -2 " 10 " |
| " 1000 " " | " 4 " 6 " | -2 " 10 " |

| continued – | Case 3. Refill | Case 4 Refill |
|---|---|---|
| After 1100 cm. air | - - - - - - | plus 4 plus 10 cm. |
| " 1200 " " | | " 6 " 11 " |

It will be noted that the initial pressures vary from minus 6 to minus 14 on inspiration and that when larger amounts of air are employed both inspiration and expiration become definitely positive.

E - Pressure Relations in Pneumothorax.

Although pneumothorax began to be recognized as a clinical entity about the middle of the eighteenth century very few investigations of pressure relations in this condition are to be found until one hundred years later.

We have already noted that Hewson in 1767 unsuccessfully attempted to produce the symptoms of emphysema and pneumothorax in animals by direct injury of the lung through the chest wall. He found that animals, if left to themselves, had no marked respiratory difficulties and would recover.

Cruveilhier in 1836 opened both pleural cavities in a dog and after a temporary cessation of respiration the dog completely recovered when left to itself. This the author believed was contrary to the earlier teachings of Van Swieten that opening of both sides of the pleura was fatal when the combined openings were larger than the glottis. In other experiments the pleural openings were held open with the hand or tubes introduced and the animals quickly died of asphyxia unless the hands were removed or the tubes were closed. A single tube in one pleura was tolerated by the animal although the respirations were increased in rate. The author did not think that the size of the tube was of any significance.

Powell[58] in 1868 demonstrated a positive pressure of four inches of water in a case of tuberoular pneumothorax at autopsy. He found an opening in the lung with a valve arrangement of the pleura. This was the first demonstration of a positive pressure in pneumothorax. In a later article, Powell[59]

considers the displacement of the heart in pneumothorax. The cause of the displacement, he was first to prove, is not positive pressure but a diminution of the negative tension on one side. Therefore, the heart balanced between two negative tensions will be drawn to the normal side. This, he showed on a human cadaver and a dog by thrusting a needle through the chest wall and into the heart and then opening one pleura. He further reports eight cases of pneumothorax with positive pressures of 1½ to 5.3 inches of water at autopsy.

Weil divided spontaneous pneumothorax into three types.

1. Closed pneumothorax in which case the pulmonary fistula had become closed and allowed no air to enter in either inspiration or expiration.

2. Open pneumothorax in which air has free entrance to and exit from the pleural cavity.

3. Valvular pneumothorax in which the fistula is open on inspiration and closed on expiration.

Pneumothorax was produced in rabbits by injecting measured quantities of air into the pleura. The average normal intrapleural tension in rabbits, he found to be minus 6.7 cm. of water with a respiratory oscillation of 4.9 cm. of water. Air introduced until the tension became zero was considered to correspond to the closed type of pneumothorax. In this condition he found the average pressure on inspiration to be minus 7 cm. water and on expiration plus 3 cm. of water. Introducing double the amount of air previously employed he considered equivalent to valvular pneumothorax. In this condition the average inspiratory pressure was minus 1.1 cm. water and expiratory plus 5 cm. In open pneumothorax the respiratory oscillations were slight and the mean pressure zero. Dogs were employed in a similar series of experiments and like results obtained. He noted, however, that dogs did not stand pneumothorax as well as rabbits.

Seifert reported four cases of pneumothorax in which the type of pneumothorax could be diagnosed from manometrical pressure determinations according to Weil's method. Two cases showed zero pressure and proved to be of the open type while two others gave pressures of plus 3.5 cm. $H_2O$ and proved to be of the closed type. One of the latter cases, while still alive gave an inspiratory pressure of plus 5 cm. $H_2O$ and expiratory pressure of plus 6.5 cm.

West reports pressure determinations in two cases of pneumothorax. One case gave plus $2\frac{1}{2}$ inches $H_2O$ on expiration and plus $\frac{1}{2}$ inch $H_2O$ on inspiration while the second case gave plus 33.5 cm. $H_2O$ on expiration and plus 17.25 cm. $H_2O$ on inspiration.

Weil also reports seven additional cases of tubercular pneumothorax with positive pressure of plus 2 to plus 10 mm. Hg.

Bard in 1901 and Aron in 1902 discussed at length the mechanism of valvular pneumothorax and the significance of positive pressures in this condition. Aron insisted on the danger of increasing intrapleural pressures and advised paracentesis or an open drainage while Bard thought the condition an advantage as it tended to the closure of the fistula.

Sehrwald, as the result of experimental investigation on animals, drew the following very accurate conclusions as to the mechanics of open pneumothorax.

1. The effect on respiration of a unilateral pneumothorax opening externally depends, not on the absolute size of the opening, but on the relation of its size to that of the bronchus of that lung.

2. The second determining factor for the ventilation of the sound lung is the nature of the mediastinum. If it is thin and distensible each inspiration will aspirate it so much to the good side that it hinders the dilatation of that lung.

3. For this reason the young and persons with a normal mediastinum

endure pneumothorax worse than those in whom it has become a thick, stiff membrane.

4. In inspiration the descent of the diaphragm puts the mediastinum on the stretch and thus aids the sound side.

5. To force the collapsed lung to again take some part in respiration it is well to close the thoracic opening at the end of forced expiration.

Dwyer, by a few well selected experiments, demonstrated that in pneumothorax the collapsed lung is expanded by the air forced into it from the sound lung. This cannot occur if by free incision into the trachea constriction of the glottis is prevented.

Reinboth discussed the importance of the size of the opening in the chest wall in pneumothorax and concluded that any hole in the pleura causes collapse of the lung and the time required varies inversely with the size of the hole. With the opening larger than the bronchus in ordinary respiration there are no volume changes in the collapsed lung. Forcible inspiration causes further collapse and expiration an inflation provided the glottis is narrowed or the trachea clamped. If the opening is smaller than the bronchus the reverse is true and the lung expands with inspiration.

Auquier in 1882 emphasized the importance of another factor besides negative intrapleural tension in maintaining the expansion of the lungs in the thoracic cavity. He states that Dolbeau and Smith, 1868, maintained that a molecular force greater than the elasticity of the lungs existed between visceral and parietal pleuras and kept them in contact. This force they likened to the cohesion between two plates of wet glass. Auquier corroborates this view and states that an intrapleural vacuum does not exist but that when a trocar is introduced between the pleural layers normal conditions are altered and a pneumothorax is produced. All measurements so obtained, he believes, are false and do not show the true state of affairs.

West, in 1887, also subscribes to this view and made experiment with discs covered with smooth membrane and determined the force necessary to separate them. He believed pneumothorax was brought about by the forcible separation of the two pleuras in one area after which the elastic recoil of the lung sufficed to peel one pleura from the other.

Pique made experiments which seemed to corroborate the existence of cohesion between the two pleuras. He found an incision one centimeter long could be made in the pleura without collapse of the lung.

Northrup could find no evidence of cohesion between the two pleural layers. On making a pin hole opening in the parietal pleura, collapse of the lung occurred.

Tendeloo, Brauer, Macewen and Roth, as well as others, sustained the cohesion theory in numerous articles. They insisted that the parietal pleura could not be opened without mechanically causing a slight local separation of the two pleural surfaces and a resultant small pneumothorax after which further separation of the pleuras could occur by a process of rolling backward from each other and the pulmonary elasticity then could become effective.

Charles Line and Dionys Hellin in 1907 are the most recent advocates of cohesion as the important factor in pulmonary expansion although they added no new evidence in their articles.

Barr states that the illustration of cohesion commonly employed, namely two wet pieces of glass or discs covered with smooth, moist membrane, are not examples of cohesion but examples of surface tension and atmospheric pressure. Even when such experiments are carried out in a vacuum, such a vacuum cannot be made perfect and some portion of an atmosphere remains. If cohesion of the pleural surfaces was present all lateral movements would be abolished.

Murphy could find no evidence of such a cohesive force. He also

discussed various factors concerned in surgical pneumothorax. He found the anterior mediastinum thin in dogs and sometimes with an opening between the two pleural cavities. In certain types of animals he stated such an opening was normally present. When such an opening was not present in the dog the mediastinum is easily ruptured. In expiration with an open pneumothorax, the lung appears to come closer to the chest wall while in reality it contracted the hilus. If there was resistance to the exit of air from the trachea in expiration as by narrowing of the glottis then air from the uninjured lung was forced across and distended the collapsed lung. This was noted in three human cases. He concludes "I am convinced that the dyspnea, following opening of the pleural cavity is due to the vibration of the mediastinal septum and its contents, destroying the piston action of the diaphragm". Under such conditions a "pendulum movement" of air ensues from one lung to the other and no effective exchange of air occurs with the outside. This condition can be relieved by covering the pneumothorax opening or by traction on the collapsed lung to steady the mediastinum.

Edmunds studied the resistance offered by the vocal cords to the passage of air in pneumothorax. By means of an endoscope the larynx of anesthetized animals was brought under direct observation. He found that normally the cords move freely and never actually touch. When however, the pleura is opened the cords take on a more vigorous movement, at one stage being in contact and at another widely separated. Coincident readings of intratracheal pressures showed that an increased pressure was maintained during part of the respiratory cycle. At this time the glottis was seen to be closed and the collapsed lung became inflated. He also observed in a child in whom decortication had been done for empyema, three phases to respiration.

1. Inspiration which filled the sound lung and the affected lung collapsed.

2. Expiratory effort and the diseased lung was seen to fill with air.

3. Glottis was opened and air expelled from both lungs.

He therefore recognized three respiratory phases.

1. Inspiration, 2. Distribution, 3. Expiration.

In 1902 Gerulanus recommended tamponade and suture of visceral and parietal pleuras to prevent surgical pneumothorax.

In 1904 a negative pressure cabinet was introduced by Sauerbruch and positive pressure apparatus by Brauer for the same purpose. From this time on much has been written regarding the surgical management of thoracic diseases and the preventions of serious complications.

The work of Garre and Quincke in 1912 may be taken as typical of the general opinions held regarding pneumothorax. The size of the opening in the pleura was stated to be of decisive importance. With an opening smaller than the trachea a slight negative pressure was produced in the affected side with each inspiration and some air entered the collapsed lung. The function of the other lung depended upon the position of the mediastinum and this depended on the pressures exercised upon it. With equal pressures on the two sides it would not change. However, in pneumothorax with a small opening there is a marked difference in the pressures of the two sides and the mediastinum is displaced toward the sound side and a complete expansion of the sound lung cannot occur. In expiration, especially if forced, the pressures become positive and the mediastinum is forced away from the sound lung and bulges toward the pneumothorax. Some air from the sound lung will also enter the collapsed lung. In spite of the difficulties an opening smaller than the glottis can be compensated for and dangerous symptoms do not appear. However, with a wide open pleura the air enters and leaves the pleura so easily that the lung is functionless. Mediastinal flutter is more marked and inspiration and expiration become insufficient. There occurs a pendulum movement of air

from one lung to the other with respiratory efforts and adequate gas exchange with the exterior does not occur.

Graham and Bell in 1918 brought forward as result of their experimental investigations radically different conclusions. They stated, "For all practical purposes - - - the thorax may be considered as one cavity instead of two. Any change of pressure in one pleural cavity will affect also the other one almost equally. The common conception of collapsed lung on one side and "healthy" or normal lung on the other, in the condition of open pneumothorax in the otherwise normal chest, must be erroneous. - - - - The dogs mediastinal structures have the same mobility as the human being's; therefore, experimental results obtained on the living dog are applicable to man." Parallelism of thoracic pressure relation in dogs and humans was demonstrated by injecting air into one pleural cavity until a pressure of plus 10 cm. water was reached and observing the pressure recorded by the opposite pleura. Observation on human cadavers and dead dogs showed that the opposite pleural cavity showed pressures of plus 9 to plus 9.5 cm. $H_2O$ in each case. Five human cadavers were employed, two of which were infants of four months, and fourteen months respectively, one a child aged fourteen years and two adults. They state,"It is clear, therefore, that the distribution of the pressure from one pleural cavity to the other under the conditions of the experiment, was due to the fact that the mediastinal structures were pushed over against the opposite lung. Furthermore, under a pressure equivalent to 10 cm. $H_2O$, the rigidity of the mediastinal partition between the pleural cavities in both dogs and the humans - - - is practically negligible". The relative density of the lungs of dogs and humans, under the same degree of collapse, was compared and found to be very similar.

As similar results were obtained in living and dead dogs, it was thought that experimental results obtained on living dogs could be directly applied to the living human. They found a marked difference in the behavior

of the animal depending on the size of the pleural opening in relation to the glottis. With a large opening, despite maximal respiratory efforts, the animal was unable to establish a negative pressure in the pleural cavity, and therefore was unable to get air into the lungs or if it was able to establish a slight negative pressure it might still be unable to obtain sufficient air to maintain life. No such thing as a unilateral collapse was possible for the two lungs must function equally since practically equal pressures are present in the two pleural cavities. "In the normal chest the action of the lungs, as a whole, proceeds practically as if no mediastinum were present, since the pressures in the two pleural cavities are always the same." Again, "A double open pneumothorax in a normal chest is more dangerous to life than a unilateral open pneumothorax merely because usually the combined areas of the two openings - - is greater than a single opening on one side is likely to be." The size of a pleural opening compatible with life, they found bore a definite relation to the area of the glottis and this could be expressed by a mathematical formula. For man the maximum size opening compatible with life for short periods was calculated to be 5 x 10 cm.

In a later article Graham corrects there figures giving an area of 101.3 sq. cm. as the maximum non fatal opening.

Graham's conclusions, from a clinical standpoint, seem open to question. The experience of military surgeons during the late war seems to emphasize the relative safety of unilateral surgical pneumothorax. Matas, in a recent article, discusses the apparent indifference of the French surgeons generally to the dangers of pneumothorax.

Hedblom, in speaking of open pneumothorax, states, "It is obviously not only possible but reasonably safe, so far as the immediate risk to the life of the patient is concerned, to open wide the pleural cavity without differential pressure anesthesia."

We have confined our studies of intrapleural tensions to two main divisions; one the study of pleural pressures in the presence of effusions and secondly the study of pressure relations in pneumothorax.

Pleural Pressures in Pleural Effusions.

For some time, we have carried out as a matter of routine the estimation of pleural pressures in all cases of pleural effusion requiring aspiration. As a result, various types of pathology are included in this report. The simplest type of portable apparatus was employed, namely a water manometer of the U type mounted on an upright stand and connected by stiff walled tubing to an ordinary aspirating needle by means of a slip-on metallic union. This tube could be connected directly with the needle or the tubing of a Patain aspirator interposed, the metallic connections fitting in either case. In actual use, a large sized aspirating needle fitted to a Record Syringe was inserted between the ribs. By withdrawing the piston of the syringe slightly and so creating a vacuum, entrance of the needle into the pleural effusion was readily noted by the appearance of fluid in the syringe. The syringe was then quickly disconnected from the needle and the opening in the butt of the needle closed by the finger to prevent the entrance of air until the manometer tubing could be connected to the needle. The column of air in the manometer tubing served to convey the pleural pressures to the water manometer. Care was taken to prevent the entrance of fluid into the tubing but if this did occur the tubing was maintained on a level with the point of puncture. The punctures were made quite uniformly in the ninth interspace in the posterior axillary line. The pressures are given in centimeters of distilled water.

Case 1. Miss R.A. - nurse - age 25.

Diag. Left pleural effusion (tuberculous)

Patient lying on right side.

Initial pressure - minus 5 cm. water.

1,000 c.c. straw-colored fluid removed.

Case 2. Mrs. Wm. L. #396042. Age 74.

Diagnosis: Right pleural effusion (tuberculous)

Patient sitting erect.

Initial Pressure: minus 5 to minus 6 cm. water.

700 c.c. straw-colored fluid removed.

Case 3. Mr. W. M. #388765. Age 59

Diagnosis: Exophthalmic goiter, pneumonia and right pleural effusion.

Patient lying on left side.

Initial Pressure: minus 6 to minus 7 cm. water.

After removal 250 c.c. turbid bile stained fluid.

Terminal Pressure: minus 12 cm. and with deep breath gave minus 18 cm.

Case 4. Miss M. G. #372930. Age 20.

Diagnosis: Exophthalmic goiter. Lt. Empyema.

Patient lying on right side.

Initial Pressure: minus 6 to minus 8 cm. water.

Case 5. Mr. A. W. V. #385875. Age 28.

Diagnosis: Biliary fistula. Postoperative pleuritis. Left.

Patient lying on right side.

Initial Pressure: minus 4 cm. water.

After removal of 800 c.c. turbid yellow fluid.

Terminal Pressure: minus 10 cm. water.

Case 6. Mr. J.G. #386051. Age 61.

Diagnosis: Acute empyema, right.

Patient lying on left side.

Initial Pressure: plus 3 cm.

2,000 c,c. thin foul pus evacuated.

Case 7. Mr. G. R. #138023. Age 33.

Diagnosis: Left empyema with bronchial fistulas.

Patient lying on right side.

Initial Pressure: minus 2 to 0 cm.

No change from coughing or deep breathing.

Case 8. Mr H. R. #386643. Age 28.

Diagnosis: Splenic anemia. Acute empyema. right.

Patient turned partly on left side.

Initial Pressure: minus 2 to minus 8 cm. increasing with deep breath to minus 10 cm.

800 c.c. foul pus evacuated.

Case 9. Mrs. A.P.A. #382100. Age 50.

Diagnosis: Exophthalmic goiter. Right pleural effusion.

Patient lying on left side.

Initial Pressure: minus 4 to minus 6 cm. water.

250 c.c. yellow bile stained fluid removed.

Case 10. Mrs. E. L. #401953. Age 40.

Diagnosis: Cardiac decompensation. Right pleural effusion.

Patient lying on left side.

Initial Pressures: Quiet breathing, minus 5 to minus 7. Deep respiration, minus 14 cm.

Cough: plus 12 cm.

After removal 900 c.c. clear yellow fluid.

Terminal Pressures: Quiet breathing, minus 2 to minus 8 cm. Deep respiration, minus 12.

Cough: plus 50 cm.

Case 11. Mr. Wm. F. #382822. Age 40.

Diagnosis: Polyserositis (Pick's Disease)

Sitting erect.

Left pleura Initial Pressures: Quiet breathing plus 10 to plus 12 cm. Cough plus 50 cm.

After removal 1,100 c.c. from left pleura. Quiet breathing plus 6 to plus 8 cm.

The next seven cases are examples of intrathoracic malignancy with effusions.

Case 12. Mr. W. H. #398137. Age 49.

Diagnosis: Malignancy of chest with right pleural effusion and cerebral metastasis.

Patient sitting erect.

7/12/22 Initial Pressures:

A. Quiet breathing plus 5 to plus 6 cm. Cough plus 24 cm.

After removal of 1,000 c.c. of sero-sanguinous fluid.

Terminal Pressure: minus 2 to minus 3 cm.

7/18/22 Initial Pressure: 0 to plus 1.

B. 850 c.c. sero-sanguinous fluid.

Case 13. Mr. T.K. #387269. Age 51.

Diagnosis: Mediastinal tumor. Right hydrothorax.

Patient sitting erect.

Initial Pressures: Quiet breathing plus 1 to plus 2 cm. Cough plus 22 cm.

After removal 1,000 c.c. clear yellow fluid: Quiet breathing minus 8 to minus 10 cm. Deep breath minus 40 cm. Cough, plus 10 cm.

After removal of 1500 c.c.: Quiet breathing minus 18 to minus 20 cm. Deep breathing minus 30 cm. Cough, zero.

Case 14. Mr. W. H. #377816. Age 40.

Diagnosis: Mediastinal tumor and left pleural effusion.

Patient sitting erect.

Initial Pressure: Quiet breathing minus 1 to minus 3 cm. Cough plus 24 cm.

1700 c.c. sero-sanguinous fluid removed.

Case 15. Mrs. D. B. #32481. Age 63.

Diagnosis: Cancer of breast with pulmonary metastasis. Right pleural effusion..

Patient lying on left side.

Initial Pressure: plus 7 to plus 8 cm.

1,000 c.c. cloudy fluid removed.

Case 16. Mrs. H. T. #232334.

Diagnosis: Cancer of breast with pulmonary metastasis and right pleural effusion.

Patient sitting erect.

Initial Pressures: Quiet breathing minus 5 to minus 6 cm. Cough plus 26 cm.

Removal of 1,000 c.c. clear yellow fluid.

Case 17. Miss I.K. #382098. Age 20.

Diagnosis: Hodgkins Disease. Bilateral pleural effusion.

1/24/22. Lying on left side.

Aspiration of right pleura.

Initial Pressure: minus 7 cm.

500 c.c. cloudy yellow fluid removed.

1/29/22. Lying on left side.

Aspiration of right pleura.

Initial Pressure: minus 9 cm.

450 c.c. removed.

2/2/22. Lying on right side.

Aspiration right pleura.

Initial Pressure: plus 7 to plus 9 cm.

After aspiration 1,000 c.c.

Pressure: minus 6 to minus 8 cm.

2/5/22. Lying on right side

Aspiration of right pleura.

Initial Pressure: plus 2 to plus 4 cm.

After aspiration 1,000 c.c.

Pressure: minus 9 to minus 11 cm.

2/8/22. Lying on right side.

Aspiration left pleura. Initial Pressure: minus 3 to minus 4 cm.

After aspiration 200 c.c., pressure minus 3 to minus 4 cm.

2/13/22. Lying on right side.

Aspiration right pleura.

Initial Pressure: plus 1 to plus 3.

Cough plus 18.

After aspiration 1,400 c.c., pressure minus 10 to minus 12.

2/18/22. Lying on right side.

Aspiration right pleura.

Initial Pressure: plus 4 to plus 6.

After aspiration 700 c.c., pressure minus 3 to minus 1 cm.

Case 18. Mr. A. M. #385580. Age 70.

Diagnosis: Lymphosarcoma chest wall. Hydropneumothorax left side following first stage operation for removal of growth.

Aspiration yielded nothing but air.

Quiet breathing minus 1 to minus 14 cm.

Suspended respiration minus 3 cm.

Deep breath minus 24 cm.

Cough plus 54 cm.

In the three following cases the positions of the patients were changed after the pleural puncture had been made to observe possible effects on the pleural pressures.

Case 19. Mr. J. N. #369132. Age 42.

Diagnosis: Cardiovascular lues with right pleural effusion.

Sitting erect.

Initial Pressure: plus 6 to plus 7 cm.

4/6/22. Lying on right side plus 7 to plus 8 cm.

Lying on left side minus 5 to minus 7 cm.

After removal 1,150 cc. clear yellow fluid.

Sitting erect plus 2 to plus 3 cm.

Lying on right side plus 1.

Lying on left side minus 1.

Case 20. Mr. H. A. S. #388873. Age 37.

Diagnosis: Chronic myocarditis, decompensation and right hydrothorax.

4/20/22 Initial Pressures: Sitting erect plus 6 to plus 6.5.

Lying on left side minus 6.

Lying on right side plus 8 to plus 9.5.

After removal of 1,000 c.c. fluid.

Sitting erect 0 to plus 1.

Lying right side plus 5 to plus 6.

Lying left side minus 7 to minus 8.

After removal 1800 c.c. fluid.

Sitting erect minus 7 to minus 8 cm.

Lying right side plus 8 cm.

Lying left side minus 26 to minus 27 cm.

5/1/22. Initial Pressure: Sitting erect plus 5 cm.

Lying left side 0 to plus 1 cm.

Lying right side plus 9 cm.

After removal of 1,130 c.c. fluid.

Sitting erect plus 2 to plus 3 cm.

Lying left side minus 9 cm.

Case 21. Mr. L. R. T. #388238. Age 37.

Diagnosis: Right pleural effusion (tuberculous).

Initial Pressures:

Sitting erect - Quiet breathing minus 4 to minus 24 cm.

Deep breath minus 36 cm.

Cough plus 56 cm.

Lying on left side -

Quiet breathing minus 4 to minus 30 cm.

Lying on right side -

Quiet breathing minus 2 to minus 16 cm.

Deep respiration minus 24 cm.

Cough plus 54 cm.

The two following cases were aspirated at the same level on either side for a comparison of the pressure relations existing in bilateral effusions.

Case 22. Mr. J. S. #390463. Age 49. Blacksmith.

Diagnosis: Malignant hypertension. Cardiac hypertrophy and dilitation. Left hydrothorax.

Patient sitting erect.

Initial Pressures: Right plus 3 to plus 2 cm.

Left plus 6 to plus 8 cm.

900 c.c. aspirated from left pleura.

20 c.c. aspirated from right pleura.

Case 23. Mr. J. B. #397332. Age 68. Farmer.

One year ago this patient had attack of dyspnea, tachycardia and fever lasting for one month from which he fully recovered. Six weeks ago developed cough, dypsnea and slight hemoptysis since which time he has become progressively weaker with the development of anemia and general anasarca.

Diagnosis: Chronic myocardial degeneration. Auricular fibrillation, decompensation. Pleural effusion, right.

7/12/22 Initial Pressures:

Patient sitting erect.

Pressure left, plus 3 to plus 4 cm.

Pressure right, plus 8 to plus 9 cm.

10 c.c. fluid, clear yellow, from left pleura.

850 c.c. serosanguinous fluid from right pleura.

7/14/22 Initial Pressures:

Patient sitting erect.

Pressure left, minus 2 to minus 3 cm.

Pressure right, plus 5 to plus 6.cm.

10 c.c. fluid from left, clear yellow color.

735 c.c. serosanguinous fluid from right.

At autopsy four days later pressure on the right was plus 12 cm. and left minus 3 cm.

Air was then pumped into the right pleura until a pressure of plus 47 was shown. At this time the pressure on the left became plus 7 cm. The heart showed hypertrophy and dilitation, there was hemorrhagic infarction of both lungs and the right pleura presented a hemorrhagic putrefactive empyema with 1,000 to 1,500 c.c. of fluid present. A few adhesions were present about the hilus of the left lung. About 100 c.c. of serous fluid was found on the left side.

▲ ▲ ▲ ▲

The majority of the above cited patients were quire sick so that frequently it was necessary to do an aspiration in the recumbant position. Respiration in these patients was more or less feeble and the pressures recorded do not represent the amplitude or negativity which would prevail in patients of more vigorous constitutions.

The etiology of the first group of eleven patients may be considered as inflammatory or due to venous stasis. Of these, nine showed negative pressures

averaging minus 4 to minus 7 cm. of water. Those in whom records were made after aspiration showed negative pressures of minus 10 to minus 12 cm. $H_2O$ with the exception of Case 11 in which aspiration was discontinued early because of pain and a pressure of plus 6 to plus 8 remained.

Patient #10 could produce a negative pressure of minus 12 both before and after aspiration by a deep inspiration while coughing produced a positive pressure of over 50 cm. $H_2O$.

In patient #7, who had an empyema with open bronchial fistulae coughing or deep inspiration produced no effect on the manometer.

Of the second group, containing seven patients with intrathoracic malignancy, three showed positive pressures averaging plus 4 to plus 5 cm. while two showed negative pressures. Coughing produced positive pressures averaging plus 26 in four cases in which it was recorded, while one case reached minus 40 cm. by deep inspiration after removal of 1,000 c.c. of fluid. All cases became negative after aspiration.

In general, the main difference noted between this group and the preceding is the greater frequency of positive pressures.

Patient # 18, as already stated, was a case of hydropneumothorax following a first stage operation for removal of a lymphosarcoma from the left anterior chest wall. He was a vigorous old man and quiet respiration gave an oscillation of 13 cm. $H_2O$. This may be explained partly by the fact that air can pass readily through the aspirating needle, while fluid has more inertia and tends to dampen the height of respiratory oscillations. In this case nothing but air was encountered although fluid was found at a secondary stage operation. By deep inspiration a negative pressure of minus 24 cm. was produced and coughing caused a positive pressure of plus 54 cm. $H_2O$.

Case #17 brought up the question of the position of the patient in relation to the intrapleural pressures obtained. It was noted that when the

patient was lying on the side to be aspirated, the pressure was positive, while if lying on the opposite side the pressure was negative.

The next group of three patients were utilized to decide the effect of posture on intrapleural pressure. The puncture was made in the usual manner with the patient sitting erect and readings were taken after which they were tipped to one side for a second reading and then to the opposite side for a third, without disturbing the needle. As will be recalled, punctures were made as a rule in the posterior axillary line at the level of the ninth interspace, which brought the needle well out to the lateral portion of the chest.

In patient #19, the pressures determined while sitting erect were plus 6 to plus 7 cm. $H_2O$ increasing to plus 7 to plus 8 cm. when tipped toward the affected side and changing to minus 5 to minus 7 when tipped toward the opposite side.

The other two cases behaved in a very similar manner. (#20 & #21)

The last two cases, #22 and #23, were bilateral aspirations at the same level with the patient sitting erect having a large effusion on one side and a smaller effusion on the other. The question was whether the large effusion could produce sufficient displacement of the mediastinum to equalize the pressure on the two sides. As will be seen there is still an appreciable difference between the pressures of the two sides in spite of displacement of the mediastinum.

In considering the pressures obtained in pleural effusions the point of puncture, in relation to the surface of the fluid, is of primary importance, as was first stated by James Calvert and later developed by Bard, The elastic tension of the lungs and the chest wall are the negative components while the height of the column of fluid is the positive element. A puncture near the surface of an effusion will show a more negative or less positive pressure than a puncture several centimeters lower in the fluid body. By tipping our patients

from one side to the other, we alternately placed the needle near the surface of the effusion at one time and its base at another with resultant marked differences of pressure. Another factor to be considered when the patient is lying on the affected side is the added weight of the heart and other mediastinal structures which tends to increase the positive value of the intrapleural pressure. When lying on the unaffected side,however, these structures exert the opposite effect as their weight tends to carry them away from the side of the effusion.

By aspiration two factors are changed, first expansion of the lung increases its elastic tension while removal of fluid lowers the height of the column of fluid present in the chest.

With increasing large effusions, the lung on the affected side becomes collapsed, its elasticity is overcome, and the lung on the opposite side is the sole negative factor remaining. Itwould seem reasonable to suppose that with a patient lying on the sound side if aspiration of the affected side disclosed a positive pressure which did not become negative on inspiration, the conclusion could be drawn that the elasticity of the lung on that side was nullified and a complete collapse was probably present.

Displacement of the mediastinum away from the affected side may cause notable embarrassment of respiration. This occurs by partial satisfaction of the pulmonary elasticity of the opposite lung. The chest must then be maintained in the state of partial expansion in order to produce a sufficient negative intrapleural tension to insure adequate function of that lung. The accessory muscles of respiration are called upon and if this is not sufficient to produce an inspiratory negative tension death occurs. In the two cases in which bilateral pleural pressures were taken the displacement of the mediastinum very noticeably diminished the normal negative tensions in the opposite pleura. Small positive pressures were in fact present. This does not signify, however, that negative inspiratory pressures did not exist above the fluid level on that side.

There are so many variable and uncertain factors concerned in pressure relations in pleural effusions that definite conclusions are difficult to draw. Adhesions between the two pleural surfaces preventing collapse of the lung and the exercise of its elasticity in whole or in part, definite walling in of a collection of fluid, thickening of the mediastinum, the position of the patient and the point of puncture are all variable factors to be considered. In addition the consistency of the fluid and the inertia or friction of fluid passing through a narrow aperture such as a needle tend to make manometric readings inaccurate. The pressures recorded vary with the depth of respiration and, the type of individual, and conclusions relative to the normal range of intra-pleural tension should only be drawn in a very general way.

Pneumothorax.

Our studies in pneumothorax were undertaken with the object of comparing the pressure relations in the dog and human, paying especial attention to the behavior of the mediastinum in each case. The previous work of Graham and Bell along this line was most fortunate and opportune, undoubtedly being responsible for the saving of many lives during the recent influenza epidemic. We have sought to reexamine the mechanics involved in pneumothorax and to see if any new facts could be elicited. If the conclusions of these authors were correct, we felt that the mediastinum must behave in a similar manner in both the dog and man under equal unilateral changes of pressure. The evidence seemed to prove that such a similarity existed not only between the dead dog and the dead human but also between the dead dog and the living dog. Results obtained on the living dog could therefore very justly be applied to the living human.

Our first experiments were carried out on dead dogs usually within one to six hours after death. The dogs were placed directly on their backs on the table of an x-ray machine and retained in this position by supports and straps. A plate changing tunnel was placed beneath the thorax and a Coolidge tube

centered over the sternum. In this way without disturbing either animal or tube, multiple plates could be taken with the animal in the same position. We hoped by this to show the deviation of the mediastinal structures on production of a unilateral pneumothorax. Two water manometers were connected by thick walled tubing to two fifteen-gauge needles. A Y was interposed in the center of the tubing and to one branch of the Y a hand pressure bulb was attached. After clamping off each pressure bulb to prevent leakage, the needles were inserted into each pleural cavity. When the pleural cavity was reached a negative pressure was registered by the manometer and intermittent pressure on the thorax caused fluctuations of the manometer.

The pressures obtained from each pleural cavity were first noted and an x-ray picture taken of the thorax. Then the clamp was removed from one hand bulb and air was pumped into the pleural cavity until a positive pressure was reached. The readings on each side were then noted and a second x-ray exposure of the thorax made. Following this one side of the thorax was opened and the mediastinum brought under direct observation. Air was then pumped into the opposite pleura with the idea of producing displacement of the mediastinum and finally rupture.

The first four experiments on dead animals, were not entirely satisfactory because of technical faults. The abdomen was opened in each case to obviate abdominal transmission of pressures.

Dog 1. Dead 1 hour.

Abdomen opened.

Pressure - left pleura, zero.
right pleura, 1 cm. water.
Chest ray taken.

Forced air into the right pleura.

Pressure - left, plus 10 cm. water
right, plus 11 cm. water.

Chest ray taken.

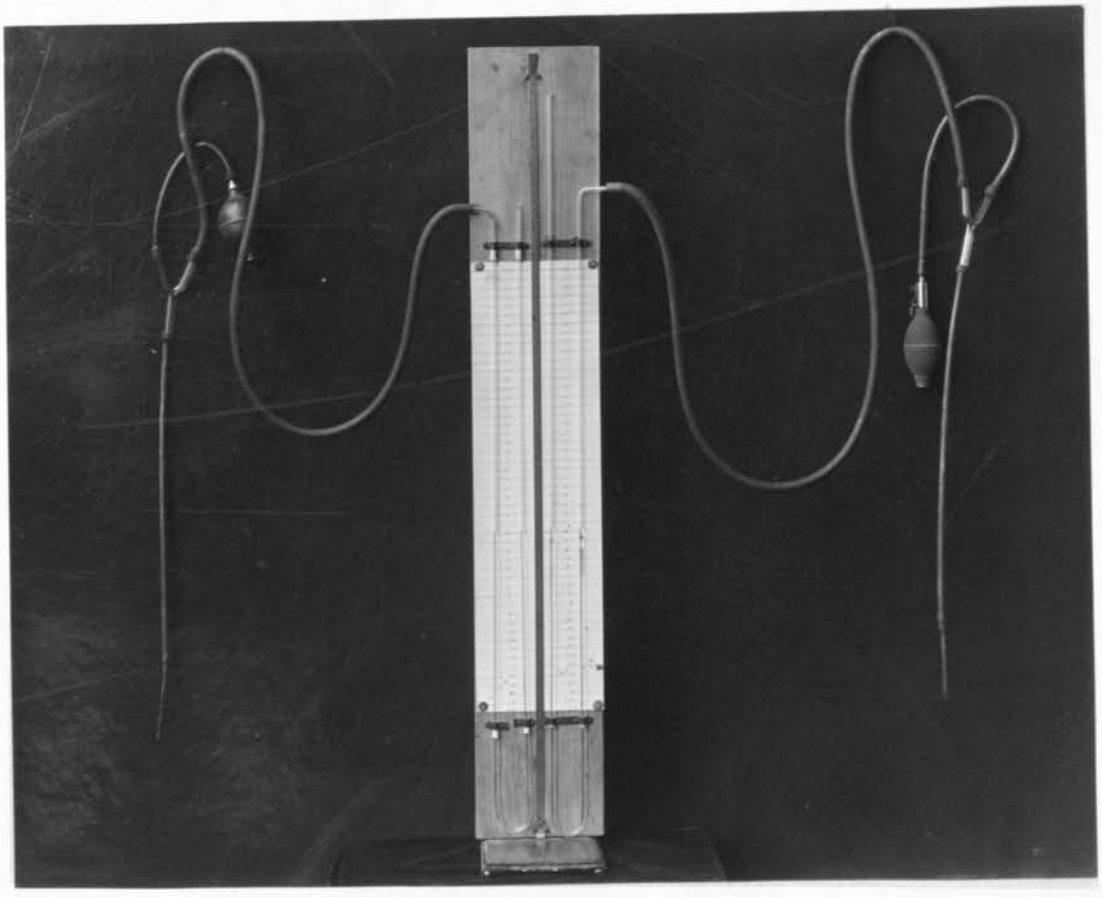


Fig. 1. Water manometers assembled with Y tubes and pressure bulbs for bilateral pressure determinations in artificial Pneumothorax.

10-20 SM

I then opened the right thorax, well on the lateral aspect, and by forcing air into the left pleura attempted to displace or rupture the mediastinum. Found this to be impossible as there seemed to be a communication between the two sides of the thorax. Search was made for this opening but it could not be found. Pathologically, lungs and pleura were negative. No deviation of the mediastinum was noted in the x-ray plates.

Dogs 2, 3 and 4 were failures as I was not certain that the needles had reached the pleural cavities.

Dog 5, E 966. Weight, 5.9 kg. Dead for three hours. Unused animal.

Abdomen opened.

Pressure - right, minus 8 cm. water
left, minus 8 cm. water.

Chest ray A.

Forced air into left pleura.

Pressure - left, plus 27 cm.
right, plus 27 cm.

Chest ray B taken.

I then opened the right pleura and by pumping air into the left pleura attempted to displace or rupture the mediastinum as in Case 1. Again, I was unsuccessful but could demonstrate no opening between the two pleuras. Pathologically, lungs and pleura were negative. The chest rays showed no deviation of the mediastinal structures and a bilateral pneumothorax was present in plate B.

Dog. 6. (Unused animal) Dead for six hours.

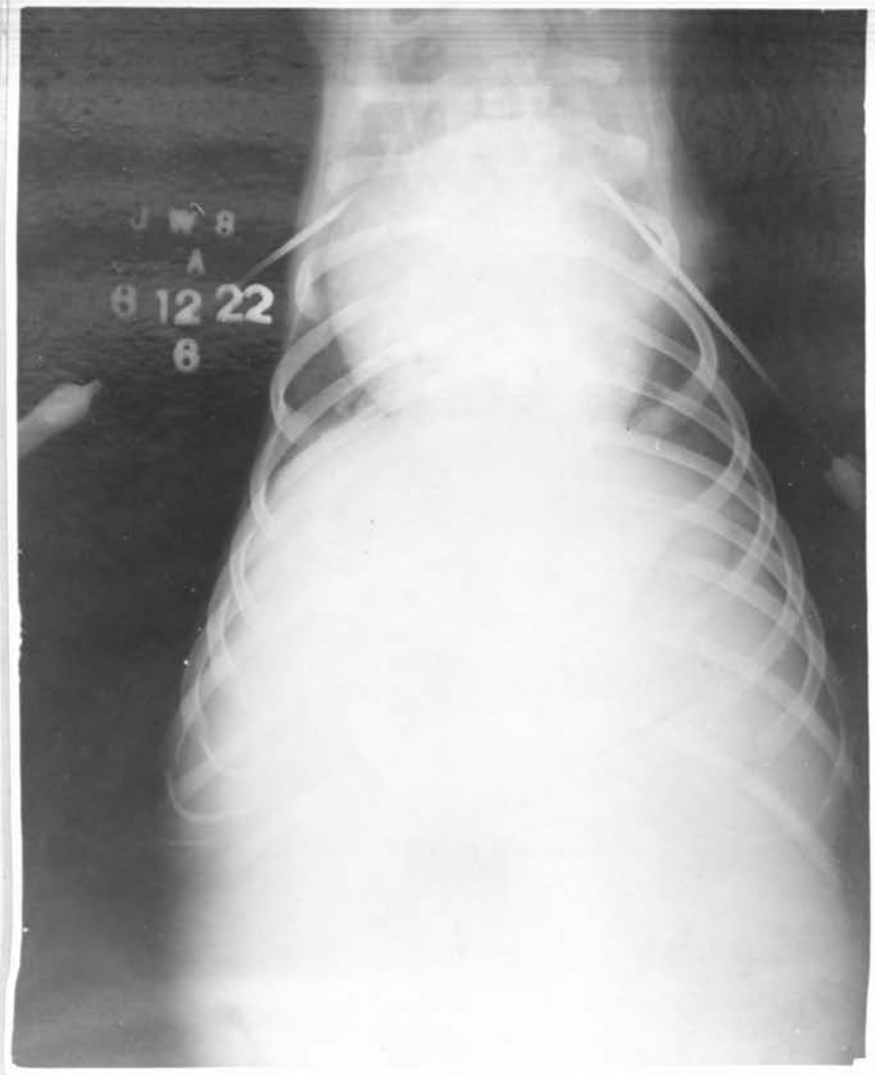
Abdomen opened.

Pressure - right, minus 4 cm. water
left, minus 4 cm. water.

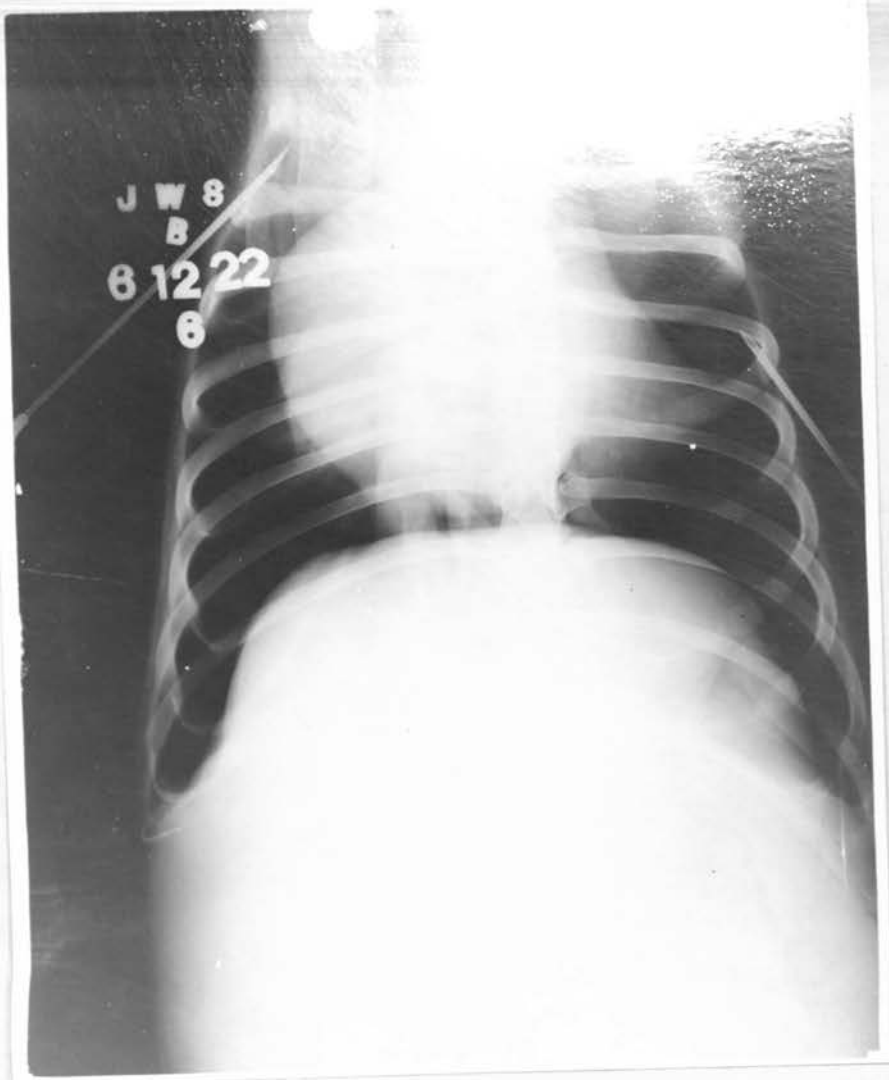
Chest ray A.

Forced air into the left pleura.

Pressure - right, plus 10 cm.
left, plus 10 cm.




Dog 6. Chest ray A before inducement of pneumothorax.




Chest ray B after air was injected into the left pleural cavity with the production of a bilateral pneumothorax.

I then opened the right pleura very carefully, well on the side of the thorax, carefully preserving the mediastinum and met with the same experience as in Dogs 1 and 5. With a view to find the natural opening which is stated sometimes to exist between the two pleural cavities, I then opened the left side of the thorax so preserving the mediastinum as an intact septum between the two sides. On turning the dog slightly to one side and filling one pleural cavity with water it passed freely through the anterior mediastinum into the opposite pleura. I could find no natural opening but the anterior mediastinum seemed to be generally freely permeable to water. Pathologically, the lungs showed moderate bronchopneumonia. Chest ray B showed no deviation of the mediastinum and and a bilateral pneumothorax was present.

Dogs 7, 8 and 9 were repetitions of experiment on Dog 6.

Dog 7. F 396. Weight 13.3 kg. Dead for thirty minutes.

Animal sacrificed because of poor condition.

Abdomen opened.

Pressure- right, minus 2 cm.
left, minus 2 cm.

Chest ray A.

Forced air into right pleura.

Pressure - right, plus 10 cm.
left, plus 10 cm.

Chest ray B.

Carefully opened left pleura and again found that by the most strenuous pumping of air into the right pleura with the hand bulb it was impossible to cause more than a slight bulging of the anterior mediastinum, which as quickly subsided. Opened right pleura preserving mediastinum, then turned the dog on his side and by pouring water on the surface of the mediastinum, found it passed through very freely.

Pathologically, the lungs and pleura were negative.

Chest ray B showed a bilateral pneumothorax with no deviation of the

mediastinum.

Dog 8. F 543. Weight 12.4 kg. Dead for two hours.

Abdomen opened. Pancreas removed and animal sacrificed.

Pressure - right, minus 10 cm.
left, minus 8 cm.

Chest ray A.

Forced air into left pleura.

Pressure - right, plus 10 cm.
left, plus 10 cm.

Chest ray B.

Carefully opened right pleura preserving the mediastinum and by forcing air into left pleura, had the same experience as before. After opening left pleura, I poured in a dilute methylene blue solution and found it passed freely through the anterior mediastinum. Pathologically, the lungs and pleura were negative.

Chest ray B showed a bilateral pneumothorax with no deviation of the mediastinum.

Dog 9. F 540. Weight 6.9 kg. Dead for four hours.

Abdomen opened from previous surgery. Pancreas removed and animal sacrificed.

Pressure - right, minus 4 cm.
left, minus 5 cm.

Chest ray A.

Forced air into left pleura.

Pressure - right, plus 14 cm.
left, plus 14 cm.

Chest ray B.

I carefully opened right pleura and on forcing air into the left pleura I found it impossible to secure an elevation of pressure of .5 cm. water by the most vigorous pumping. No deviation of the mediastinum occurred. Water

passed through the anterior mediastinum freely. Pathologically, the lungs and pleura were negative.

Chest ray B showed a bilateral pneumothorax with no deviation of the mediastinum.

Dog 10. E 142. Weight 5.1 kg. Dead eight hours.

Abdomen opened from previous surgery. Died from etherization.

Pressure - right, minus 4 cm.
left, minus 4 cm.

Chest ray A.

Ran 350 c.c. of 10% K.Br. Solution into the left pleural cavity from an elevation of 25 cm. in the space of 10 minutes.

Pressure - right, plus 16 cm.
left, plus 20 cm.

Chest ray B.

Opened right pleura and found it partly full of fluid and about an equal amount on the left.

Pathologically, lungs and pleura were negative except for a small adhesion at the left base.

Chest ray B showed about an equal amount of the opaque solution in each pleural cavity.

Dog. 11. F 152. Weight 9.7 kg. Dog alive following abdominal operation.

Removal of both kidneys. Abdomen opened. Full ether anesthesia.

Pressure - right, minus 4 to minus 8 cm. water.
left, minus 4 to minus 8 cm. water.

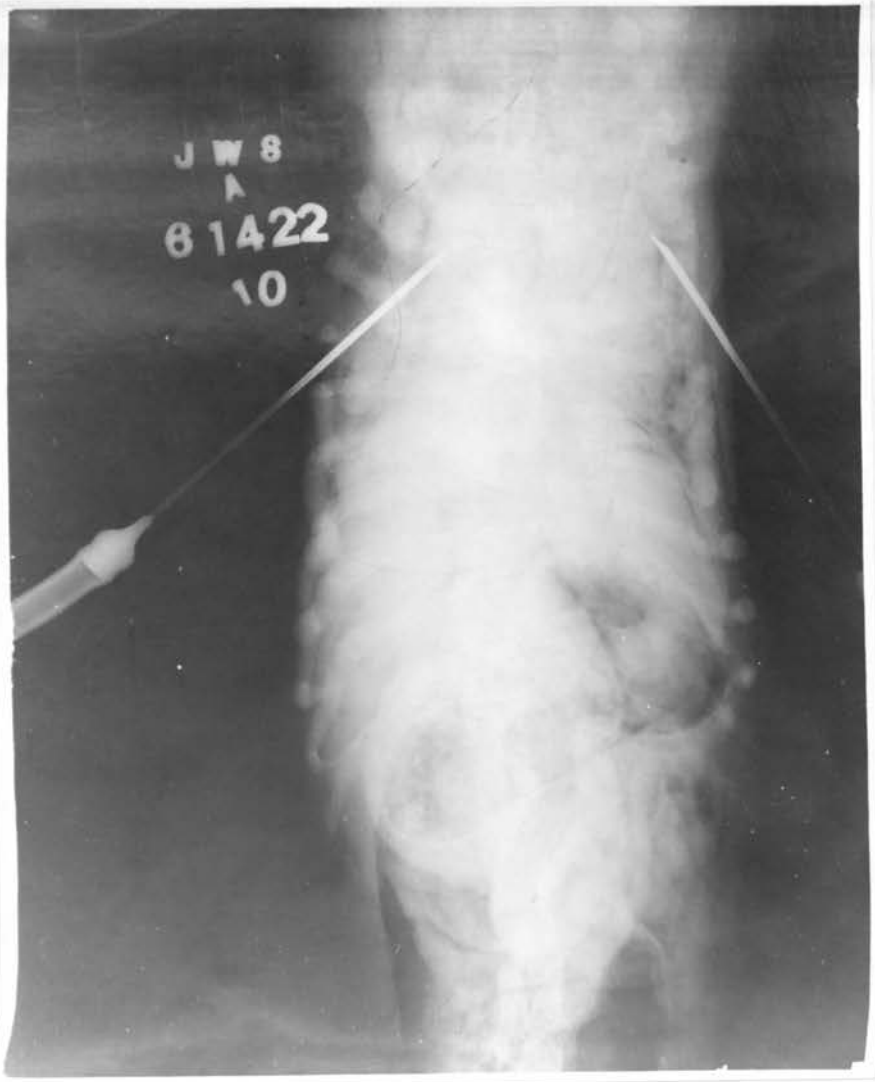
Chest ray A.

Dog died at this time.

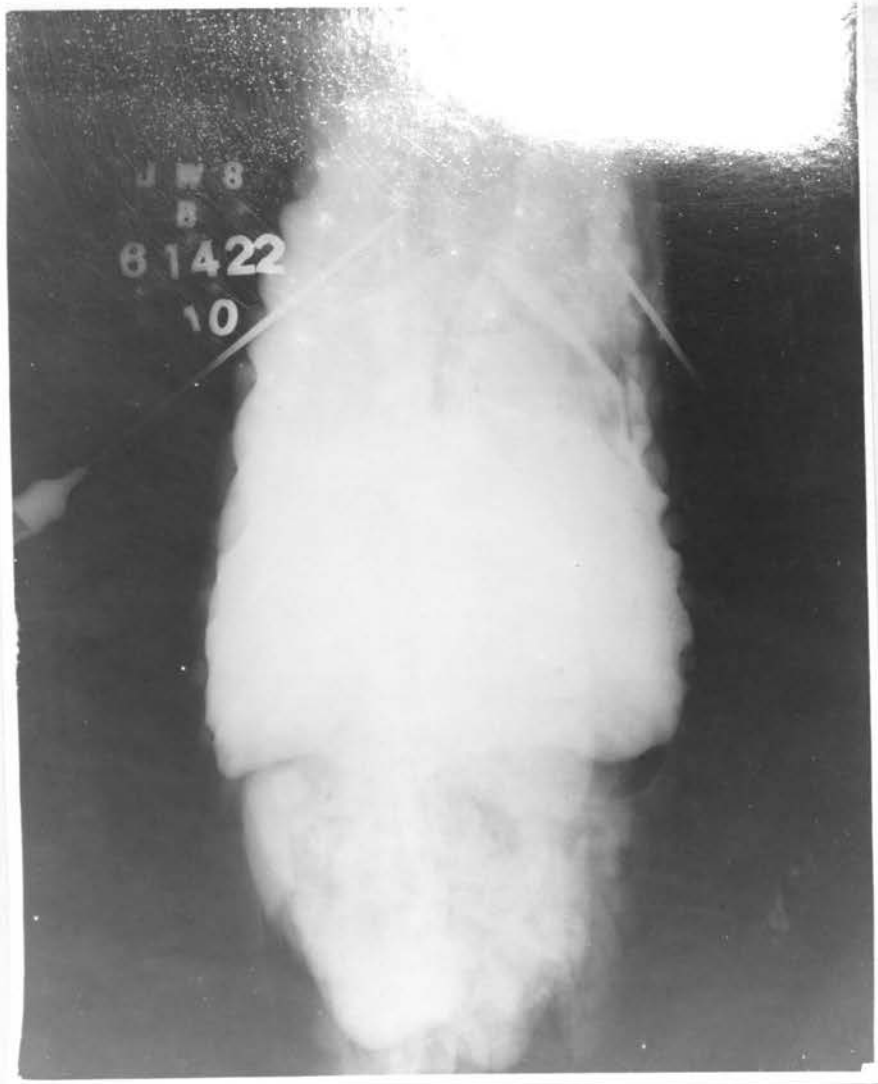
Forced air into left chest.

Pressure - right, plus 4 cm.
left, plus 4 cm.

Chest ray B.




Dog 10. Chest ray A before the introduction of Potassium Bromid solution.




Dog 10. Chest ray B after introducing Potassium Bromid solution into the left pleural cavity and showing its bilateral distribution.

On opening right pleura, the pressure on the left fell at once to zero. By pumping air into left pleura, I was unable to raise the pressure .5 cm. $H_2O$ in height or cause an appreciable displacement of the mediastinum. By running water into the left pleura through the needle already in place, it could be seen to pass through the posterior mediastinum just back of the heart and appear in the opposite pleura. By displacing the heart and vessels against the fluid, it could be forced up against the anterior mediastinum and there passed through very freely to the opposite side.

Pathologically, the lungs and pleura were negative.

Chest ray B showed a bilateral pneumothorax with no displacement of the mediastinum.

Dog 12. F 552. Weight 6.1 kg. Dog gasping slightly following removal of kidneys and severance of the abdominal aorta.

Abdomen opened.

Pressure - right, minus 6 cm.
left, minus 4 cm.

Chest ray A.

Ran 100 c.c. of 10% K Br solution into the left pleura from an elevation of 15 cm. in three minutes' time.

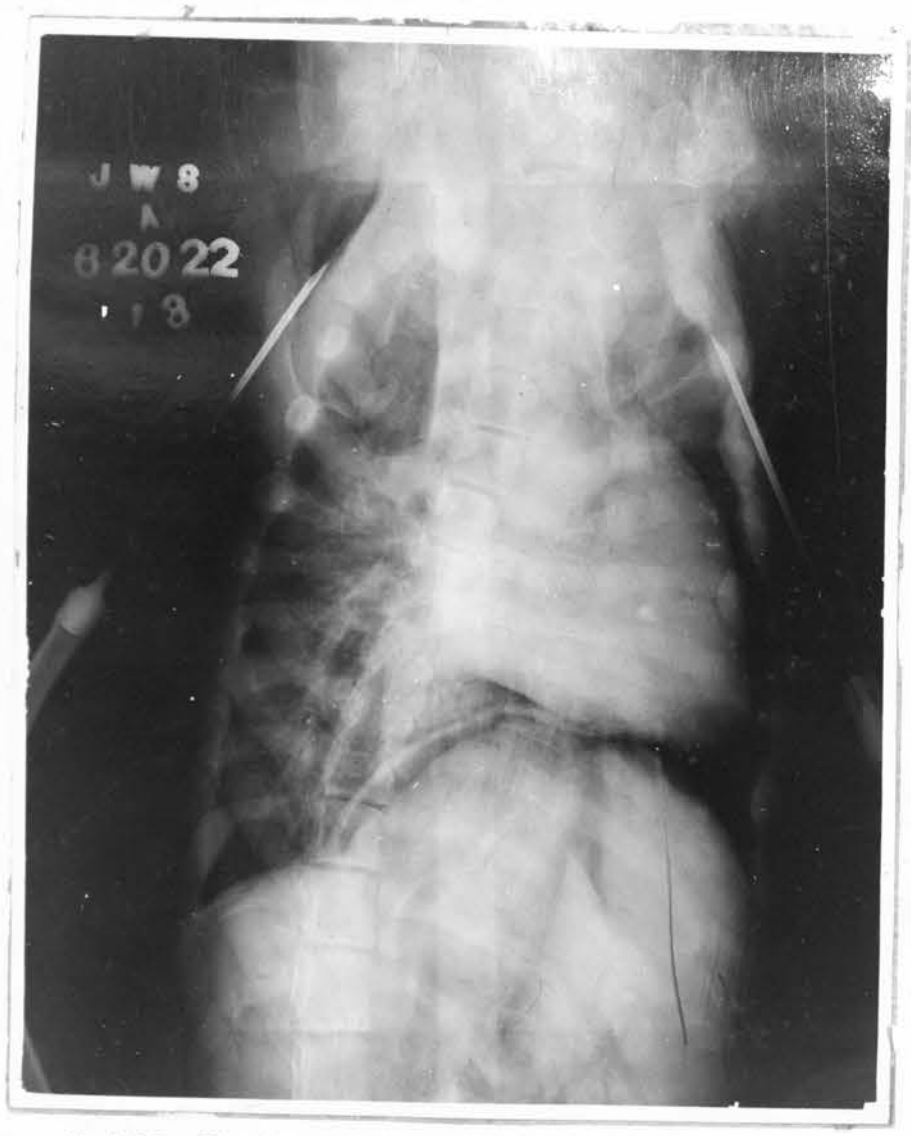
Chest ray B.

Ran in 100 c.c. additional in two minutes' time.

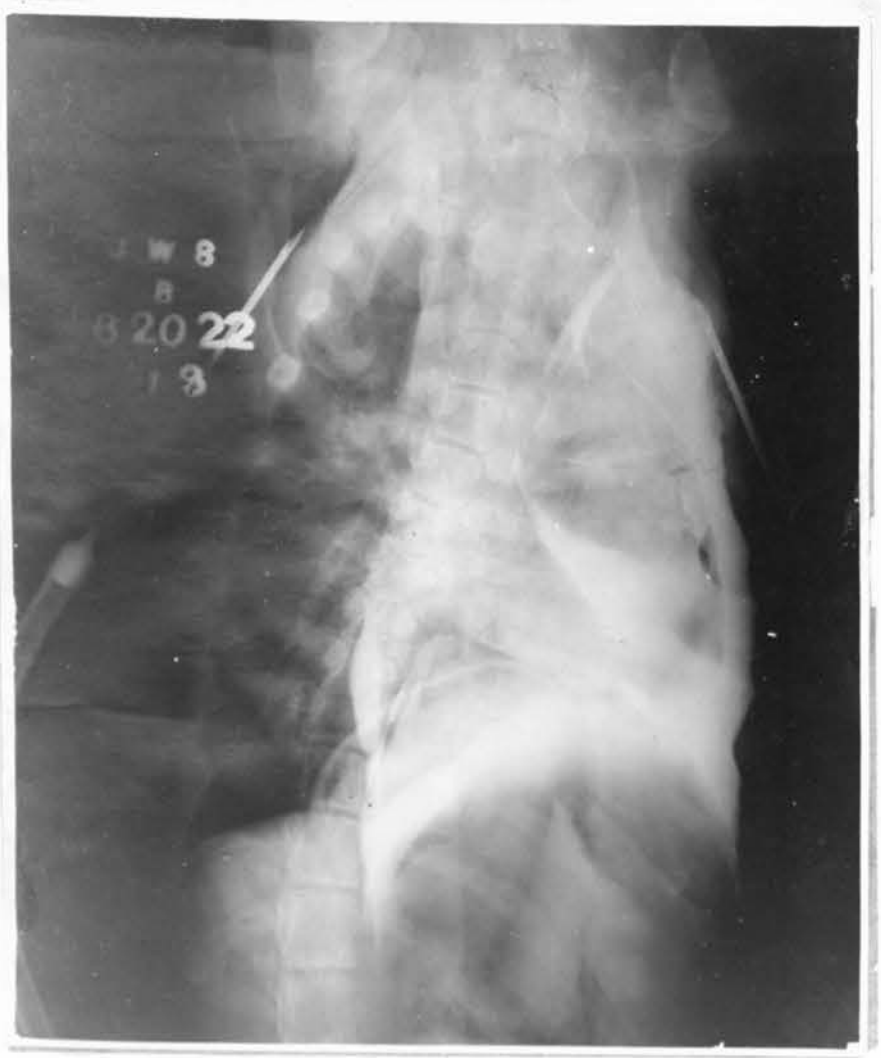
Chest ray C.

Opened right pleura and found about 75 c.c. of fluid present. Then ran larger amounts of fluid into the left pleura and while some came through the posterior mediastinum, most of it passed through the anterior mediastinum. Pathologically, the lungs and pleura were negative.

Chest ray C showed the presence of a portion of the opaque solution on the right side.




Dog 13. Chest ray A. Before the introduction of potassium bromide solution.




Dog 13. Chest ray B. After introducing 100 c.c. of potassium bromide solution into the left pleural cavity.


Dog 13. Chest ray C. After introducing 200 c.c. of potassium bromide solution into the left pleural cavity.

Dog 13. D 467. Weight 8.3 kg. Dead 1½ hours. Over-etherization.

Abdomen open from previous surgery.

Pressure – right, minus 13 cm.
left, minus 10 cm.

Chest ray A.

Ran 100 c.c. of 10% K Br solution into left pleura from 15 cm. elevation in two and one-half minutes' time.

Chest ray B.

Ran 100 c.c. more in two and one-half minutes' time.

Chest ray C.

Ran in 150 c.c. more in four minutes' time.

Chest ray D.

Total time to introduce solution, nine minutes. Then opened the right pleura and found it full of fluid. Pathologically, lungs and pleura negative. Chest rays C and D showed the bromide solution about equally distributed in the two sides.

Dog 16. F 249. Weight 9.2 kg. Dead for two hours.

Abdomen opened. Removal of both kidneys, animal then sacrificed.

Pressure – right, minus 4 cm.
left, minus 4 cm.

Chest ray A

Ran 100 c.c. of the 10% solution K Br. into the right pleural cavity in two minutes' time from an elevation of 15 cm.

Pressure – left, minus 2 cm.

Chest ray B.

Ran in 150 c.c. additional fluid in nine minutes' time.

Pressure – left, zero.

Chest ray C

Opened left chest and found about 100 c.c. of fluid. Introducing more fluid caused it to pass freely through the anterior mediastinum. Pathologically lungs and pleura were negative. Chest ray B showed some bromide solution in the lower portion of the left pleura while ray C showed a more equal distribution of the solution.

Dog. 15. F 402. Weight 15.7 kg. Dead one-half hour.

Abdomen not opened. Ligation of portal vein with resultant portal thrombosis.

Pressure – right, minus 5 cm.
left, minus 5 cm.

Chest ray A.

Forced air into the right pleura.

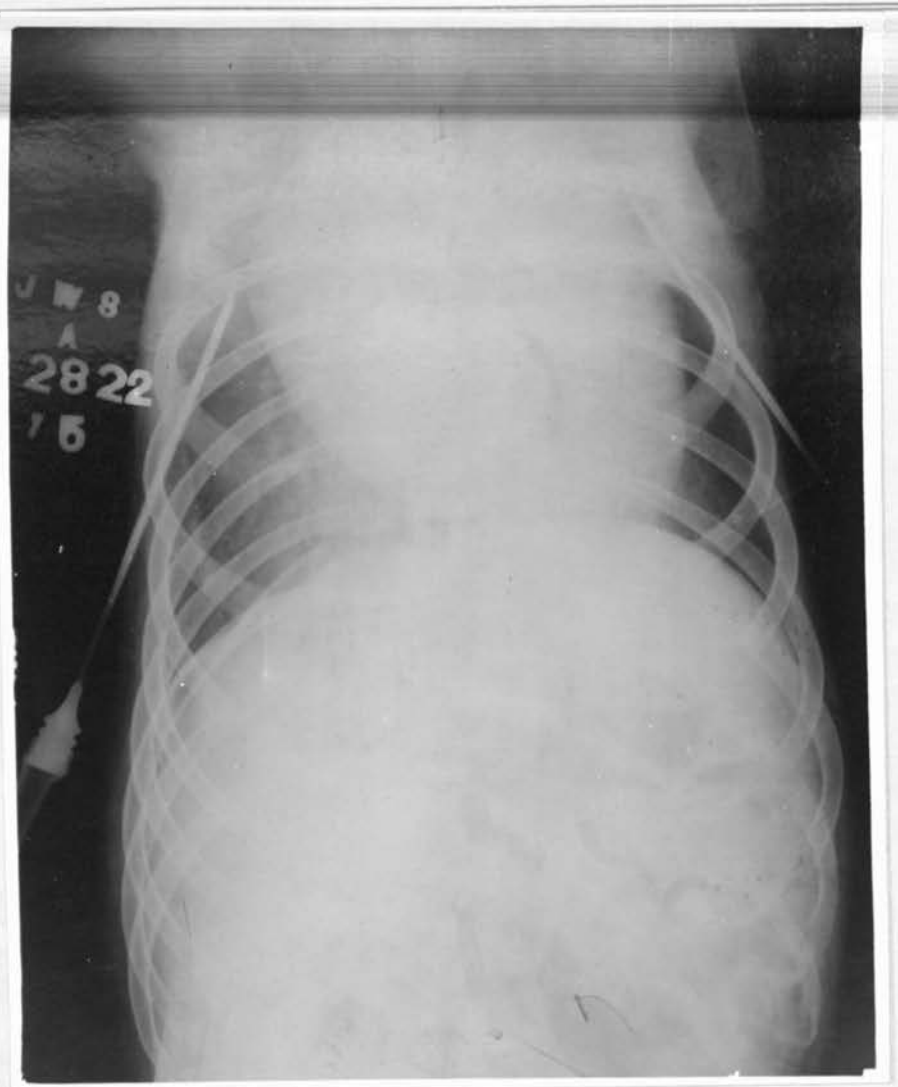
Pressure – right, plus 6 cm.
left, plus 6 cm.

Chest ray B.

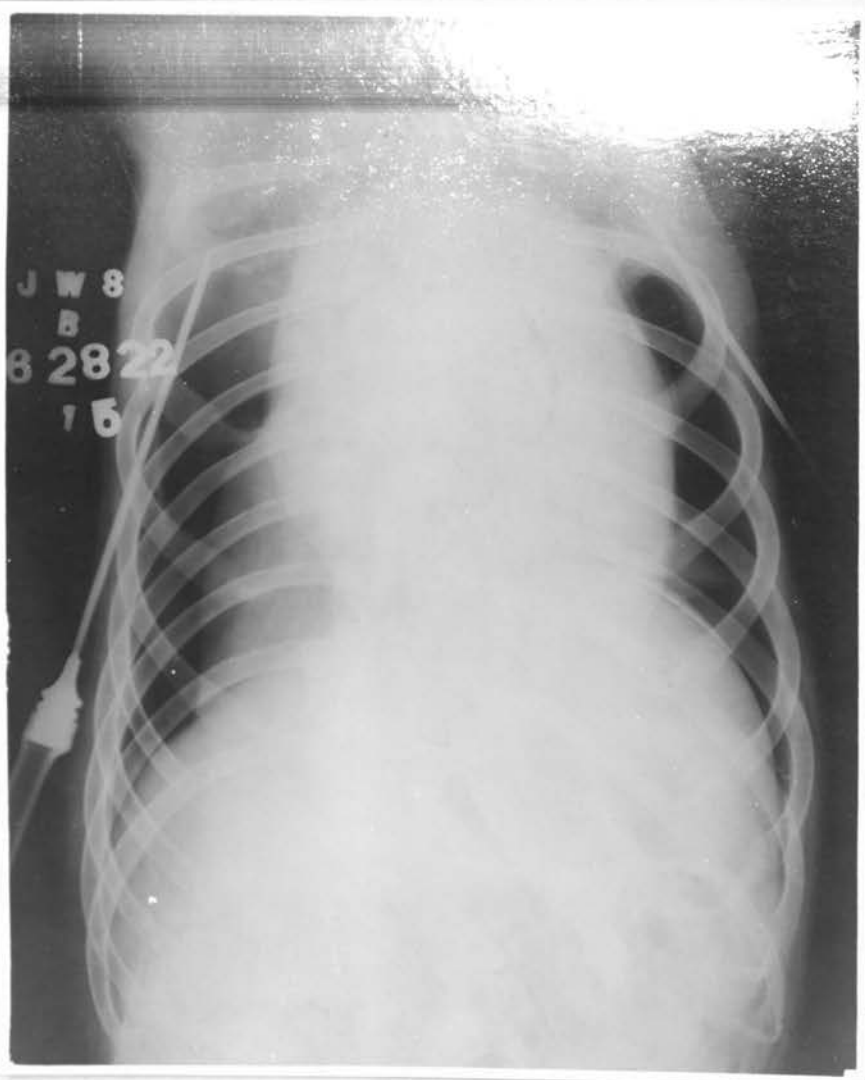
Opened right pleura and pressure on left fell to zero. Then forced air into the left pleura and by the most forcible pumping could barely cause the heart to move and was unable to raise the pressure .5 cm. $H_2O$ within the left pleura. Pathologically, the lungs and pleura were negative. Chest ray B showed a bilateral pneumothorax with no displacement of the mediastinal structures. – – – –

Presuming from the experiments already given that a unilateral pneumothorax could not be produced in the dead dog but that it was always bilateral, we proceeded to test this in a different manner.

By freeing the trachea of a dog and tying it tightly about a large glass tube, it could be connected to our water manometer. By now opening one pleura, we could produce a collapse of the lung and a positive pressure in the trachea as shown by the manometer. This method it will be seen is similar to that employed by Donders in the human. If by opening one pleural cavity in the dog,




Dog 15. Chest ray A before induction of pneumothorax.




Dog 15. Chest ray B after injecting air into the right pleural cavity with the production of a bilateral pneumothorax.

a bilateral pneumothorax is produced the opening of the second pleural cavity should cause no change in the pressure level already obtained by opening the first pleura.

Dog. 14. (no number) Dead one and one-half hours.

Pancreas had been removed. Trachea connected with manometer.

Opened right pleural cavity.

Tracheal pressure, plus 5 cm. water.

Opened left pleura.

Tracheal pressure unchanged.

Dog 17. F 483. Weight 8 kg. Dead three hours. Exsanguination. Blood taken for laboratory purposes. Trachea connected with the manometer.

Opened left pleura. Tracheal pressure, plus 7 cm.water.

Opened right pleura. Tracheal pressure, plus 7 cm.water.

Dog. 24. F 250. Dead one hour. Weight 7.6 kg.

Animal sacrificed. Kidney operation three months previously.

Trachea connected with manometer.

Opened right pleura. Tracheal pressure, plus 9 cm.

Opened left pleura. Tracheal pressure, plus 9 cm.

Dead animals were employed up to this time. We now repeated our experiments on living animals.

Dog. 18. F 99. Weight 13 kg. Both kidneys removed.

Abdomen opened from surgery. Full ether anesthesia.

Time 3:00 p.m. Pressure - right, minus 4 to minus 11 cm.
left, minus 1 to minus 10 cm.

Chest ray A.

Injected 100 c.c. of air into right pleural cavity.

3:05 p.m. Pressure - right, 0 to minus 10 cm.
left, 0 to minus 9.

Chest ray B.

Injected 100 c.c. air right pleura.

3:09 p.m. Pressure,-right, plus 1 to minus 9 cm.
left, 0 to minus 8 cm.

Chest ray C.

Injected 100 c.c. air right pleura.

3:11 p.m. Pressure - right, plus 2 to minus 8 cm.
left, plus 1 to minus 7 cm.

Chest ray D.

3:14 p.m. Injected 100 c.c. air right pleura.

Pressure - right, plus 3 to minus 8 cm.
left, plus 2 to minus 7 cm.

Chest ray E.

3:17 p.m. Injected 100 c.c. air right pleura.

Pressure - right, plus 2 to minus 8 cm.
left, plus 1 to minus 6 cm.

Chest ray F.

Injected 100 c.c. air right pleura.

3:20 p.m. Pressure - right, plus 5 to minus 8 cm.
left, plus 5 to minus 8 cm.

Chest ray G. Animal died at this time and after death pressure was plus 2 on each side.

Chest ray A 1.

Immediately ran 200 c.c. 10 % K Br solution into the left pleural cavity from an elevation of 15 cm. Time required, three minutes.

Pressure - right, plus 4 cm.

Chest ray A 2.

Ran in 200 c.c. more of K Br. solution in three minutes' time.

Pressure - right, plus 6 cm.

Chest ray A 3.

Pathologically, the lungs and pleura were negative. The chest rays again demonstrate a bilateral pneumothorax without deviation of the

mediastinal structures. The chest also showed an increasing distention, evidently the animal's attempt to compensate for the pneumothorax and maintain a negative pressure within the pleural cavity. It will be noted that a negative inspiratory pressure was maintained as long as the animal lived. After death the pressure became positive.

On opening the right pleural cavity bromide solution was present but this failed to show in the chest rays A 2 and A 3 probably because the animal in its agonal struggles turned to the left side and so pocketed the solution in the left pleura.

Dog 19. E 838. Weight 5.8 kg.

Dog alive. Abdomen not opened. Previous duodenectomy. Ether anesthesia.

3:12 p.m. Pressure - right, minus 4 to minus 10 cm. water.
left, minus 3 to minus 9 cm. water

Chest ray A 1.

3:14 p.m. Injected 100 c.c. air left pleura.

Pressure - right, minus 4 to minus 7 cm.
left, minus 4 to minus 7 cm.

Chest ray A 2.

3:16 p.m. Injected 100 c.c. air left pleura.

Pressure - right, minus 1½ to minus 6 cm.
left, minus 1½ to minus 6 cm.

Chest ray A 3.

3:17 p.m. Injected 100 c.c. air left pleura.

Pressure - right, 0 to minus 6 cm.
left, 0 to minus 6 cm.

Chest ray A 4.

3:19 p.m. Injected 100 c.c. air left pleura.

Pressure,- right, plus 1 to minus 2 cm.
left, plus 1 to minus 2 cm.

Chest ray A 5.

3:21 p.m. Injected 100 c.c. air left pleura.

Pressure - right, plus 1 to minus 1½ cm.
left, plus 1 to minus 1½ cm.

Chest ray A 6.

Animal breathing shallow, ether discontinued.

3:24 p.m. Injected 100 c.c. air left pleura.

Pressure - right, plus 1 to minus 1 cm.
left, plus 1 to minus 1 cm.

Chest ray A 7.

3:26 p.m. Injected 100 c.c. air left pleura.

Pressure - right, plus 11 to minus 3 cm.
left, plus 11 to minus 3 cm.

Chest ray A 8.

Breathing forced and violent, slight struggling.

3:28 p.m. Injected 100 c.c. air left pleura.

Pressure - right, plus 12 to minus 3 cm.
left, plus 12 to minus 3 cm.

Chest ray A 9.

Breathing slow, forced and labored.

3:29 p.m. Pressure - right, plus 9 to minus ½ cm.
left, plus 9 to minus ½ cm.

Dog practically ceased breathing. No air injected.

After death, 3:32 p.m.

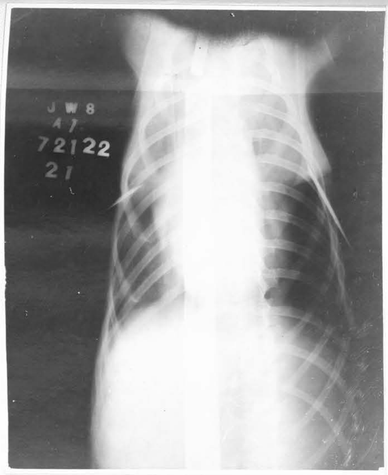
Pressure - right, plus 6 cm.
left, plus 6 cm.

Chest ray A 10.

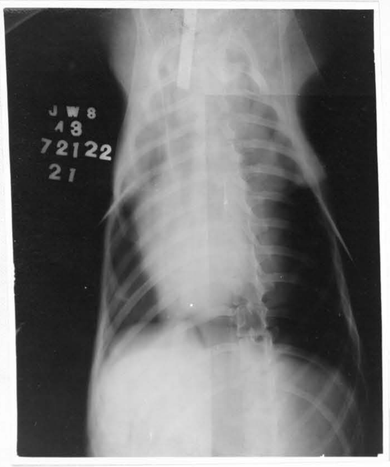
Pathologically, lungs and pleura were negative. The chest rays show a bilateral pneumothorax with a marked distention of the chest and no deviation of the mediastinum. It will again be noted that the dog was able to maintain a negative inspiratory pressure up to his death.

Dog 21. F 45. Weight 11.9 kg.

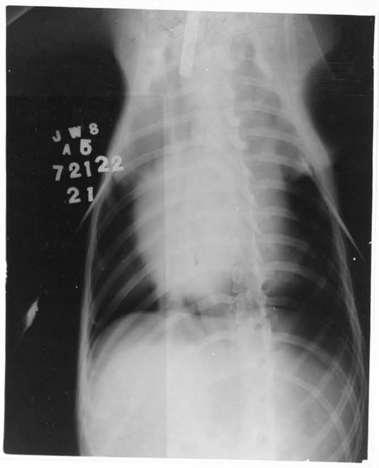
Bladder operation six months previously.



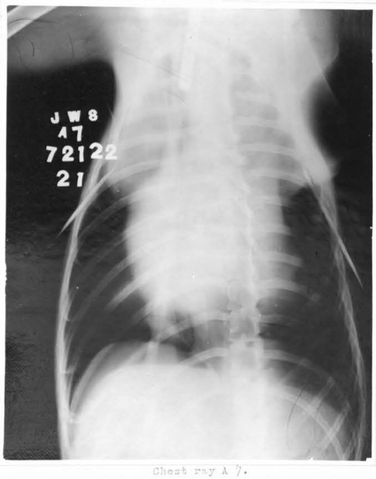

Dog 21. (Living animal) Chest ray A 1 - Before the introduction of pneumothorax.



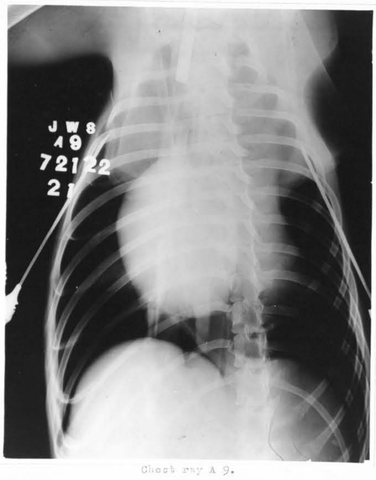

Chest ray A 3.




Chest ray A 5.




Chest ray A 7.




Chest ray A 9.

Chest rays A 3, 5, 7 and 9 show the production of a bilateral pneumothorax by repeated injections of air into the right pleural cavity. It will be noted that there is no displacement of the heart or other mediastinal structures.

Dog alive, abdomen not open. Ether anesthesia.

3:56 p.m. Pressure - right, minus 4 to minus 13 cm.
left, minus 4 to minus 13 cm.

Chest ray A 1.

3:58 p.m. Injected 200 c.c. air into right pleural cavity.

Pressure - right, 0 to minus 10 cm.
left, 0 to minus 10 cm.

Chest ray A 2.

4:00 p.m. Injected air into right pleura.

Pressure - right, plus 1 to minus 10 cm.
left, plus 1 to minus 10 cm.

Chest ray A 3.

4:04 p.m. Injected 200 c.c. air into right pleural cavity.

Pressure - right, plus 3 to minus 12 cm.
left, plus 3 to minus 12 cm.

Chest ray A 4.

4:06 p.m. Injected 200 c.c. air into right pleural cavity.

Pressure - right, plus 5 to minus 11 cm.
left, plus 5 to minus 11 cm.

Respiration slower and deeper.

Chest ray A 5.

4:10 p.m. Injected 200 c.c. air into right pleura.

Pressure - right, plus 6 to minus 14 cm.
left, plus 6 to minus 14 cm.

Chest ray A 6.

4:12 p.m. Injected 200 c.c. air into right pleura.

Pressure - right, plus 9 to minus 15 cm.
left, plus 9 to minus 15 cm.

Chest ray A 7.

4:15 p.m. Injected 200 c.c. air into right pleura.

Pressure - right, plus 10 to minus 11 cm.
left, plus 10 to minus 11 cm.

Chest ray A 8.

Injected 200 c.c. air into right pleura.

4:17 p.m. Death occurred.

Pressure - right, plus 10 cm.
left, plus 10 cm.

Chest ray A 9.

Opened abdomen and both pleural pressures fell to plus 7 cm. Opened right pleura and left pleural pressure fell to 0. Pumped air into the left pleura but could not raise the pressure .5 cm. $H_2O$ in height. Pathologically, the heart and lungs were negative. The chest rays showed a bilateral pneumothorax as before.

Dog 23. E 825: Weight 7.6 kg.

Previous cholecystectomy and appendectomy. Ether anesthesia.

11:36 a.m. Pressure - right, minus 7 to minus 9 cm. water.
left, minus 7 to minus 9 cm.

Chest ray A 1.

11:40 a.m. Injected 200 c.c. air right pleural cavity.

Pressure - right, minus 4 to minus 7 cm.
left, minus 4 to minus 7 cm.

Chest ray A 2.

11:42 a.m. Injected 200 c.c. air right pleural cavity.

Pressure - right, minus 2 to minus 5 cm.
left, minus 2 to minus 5 cm.

Chest ray A 3.

11:44 a.m. Injected 200 c.c. air right pleural cavity.

Pressure - right, plus 1 to minus 5 cm.
left, plus 1 to minus 5 cm.

Chest ray A 4.

Followed by deep forcible breathing.

11: Pressure - right, plus 10 to minus 14 cm.
left, plus 10 to minus 14 cm.

Injected 200 c.c. air right pleural cavity.

Animal ceased breathing.

Pressure - right, plus 4 cm.
left, plus 4 cm.

Chest ray A 5.

Opened right pleura and pressure on left fell to 0. Pumped air into the left pleura but could not raise the pressure .5 cm. in height. Ran milk into left pleura and after 100 c.c. ran in, it began to pass through the posterior mediastinum into the right pleura. Pathologically, the heart and lungs were negative. The chest rays again demonstrated a bilateral pneumothorax with no deviation of the mediastinal structures.

Dog 20. F 341. Weight 15.3 kg.

Bladder operation three months previously.

Ether anesthesia, abdomen opened.

3:14 p.m. Pressure-right, minus 5 to minus 7 cm.
left, minus 5 to minus 7 cm.

Chest ray A 1.

3:17 p.m. Ran 200 c.c. 10% K Br. solution into the left pleural cavity from an elevation of 15 cm.

Pressure - right, minus 3 to minus 9 cm.

Chest ray A 2.

3:21 p.m. Ran in 200 c.c. additional K Br. solution.

Pressure - right, minus 1 to minus 7 cm.

Chest ray A 3.

3:25 p.m. Ran in 200 c.c. additional K Br. solution.

Pressure - right, minus 1 to minus 8 cm.

Chest ray A 4.

Ran in 200 c.c. additional K Br. solution.

Death at 3:29 p.m.

Chest ray A 5.

Opened thorax and found both pleural cavities filled with fluid. Pathologically, the heart and lungs were negative. The chest rays showed the bromide solution in both pleural cavities.

Dog 22. F 480. Weight 9.6 kg.

Ether anesthesia, abdomen not opened.

12:00 p.m. Pressure - right, minus 5 to minus 7 cm.
left, minus 5 to minus 7 cm.

Chest ray A 1.

12:05 p.m. Ran in 200 c.c. of 10% K Br. solution from an elevation of 15 cm. into the right pleural cavity.

Pressure - left, minus 2 to minus 3 cm.

Chest ray A 2.

12:10 p.m. Ran in 200 c.c. additional K Br solution.

Pressure - left, plus 2 to minus 3 cm.

Chest ray A 3.

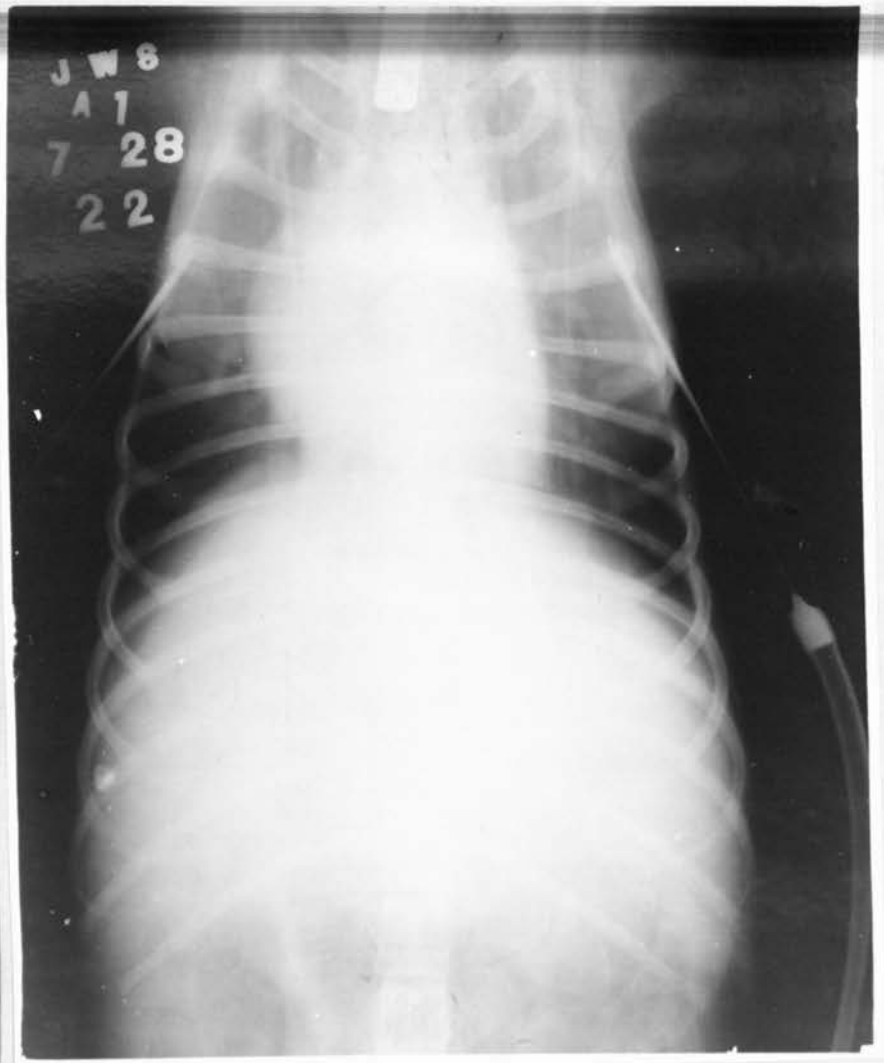
Death at 12:16 p.m.

Chest ray A 4.

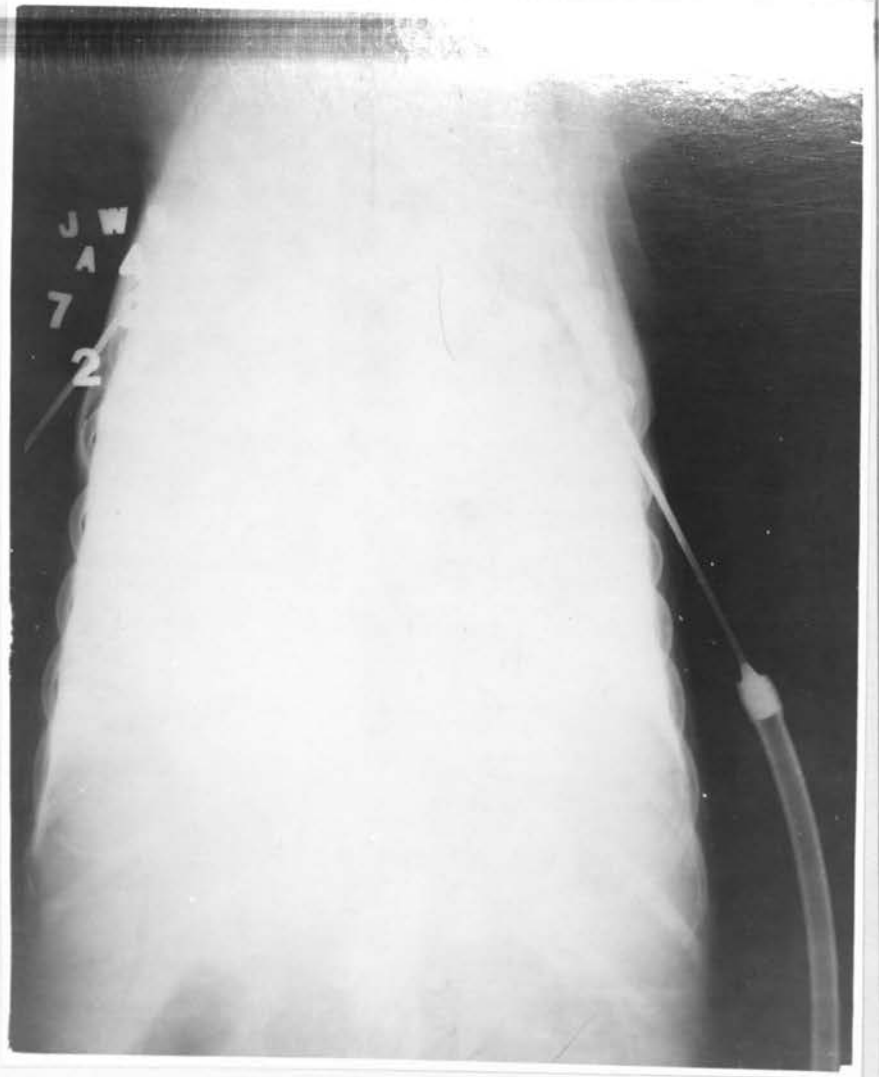
Opened chest and found fluid in each pleural cavity. Pathologically the lungs and pleura were negative. The chest rays showed bromide solution in both pleural cavities.

- - -

On three dogs graphic tracings of the pleural pressures were made. This was accomplished by arranging two water manometers having floats attached to writing arms. The points of the arms were placed in contact with the smoked drum of a kymograph. By this arrangement all oscillations of the manometer could be directly recorded on the drum of the kymograph.




Dog 22. (Living animal) Chest ray A 1. Before the introduction of potassium bromid solution.




Chest ray A 4. After introducing potassium bromid solution into the right pleural cavity and showing its bilateral distribution.

Dog 25. F 684. Weight 5.5 kg.

Ether anesthesia. Abdomen open. Pancreas previously removed.

2:45 p.m. Pressure - right, minus 6 to minus 7 cm.
left, minus 6 to minus 7 cm.

Chest ray A 1.

2:49 p.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 1 to minus 5 cm.
left, minus 1 to minus 5 cm.

Chest ray A 2.

2:51 p.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, 0 to minus 5 cm.
left, 0 to minus 5 cm.

Chest ray A 3.

2:52 p.m. Injected 200 c.c. of air into left pleura.

Pressure - right, minus 6 to plus 9 cm.
left, minus 6 to plus 9 cm.

Chest ray A 4.

2:55 p.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 8 to plus 10 cm.
left, minus 8 to plus 10 cm.

Chest ray A 5.

2:57 p.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 9 to plus 8 cm.
left, minus 9 to plus 8 cm.

Chest ray A 6.

2:59 p.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 5 to plus 8 cm.
left, minus 5 to plus 8 cm.

Chest ray A 7.

Forced labored respiration. 3:02 p.m. Injected 200 c.c. of

air into left pleura.

Pressure - right, minus 5 to plus 3 cm.
left, minus 5 to plus 3 cm.

Chest ray A 8.

Death of animal - 3:05 p.m.

Pressure - right, plus 4 cm.
left, plus 4.

Chest ray A 9.

Pathologically the lungs and pleura were negative. The chest rays showed a bilateral pneumothorax with no displacement of the mediastinum.

- - -

Dog 28. F 681. Weight 11.8 kg. 8/15/22

Ether anesthesia, abdomen open. Pancreas previously removed.

10:52 a.m. Pressure - right, minus 8 to minus 4 cm. water.
left, minus 8 to minus 4 cm.

Chest ray A 1.

10:54 a.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 2 to minus 7 cm.
left, minus 2 to minus 7 cm.

Chest ray A 2.

10:56 a.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 1 to minus 6 cm.
left, minus 1 to minus 6 cm.

Chest ray A 3.

10:58 a.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 7 to plus 8 cm.
left, minus 7 to plus 8 cm.

Chest ray A 4.

11:00 a.m. Injected 200 c.c. of air into left pleura.

Pressure - right, minus 7 to plus 10 cm.
left, minus 7 to plus 10 cm.

Chest ray A 5. - Breathing slower and deeper.

11:03 a.m. Injected 200 c.c. of air into the left pleura.

Pressure - right, minus 9 to plus 10 cm.
left, minus 9 to plus 10 cm.

Chest ray A 6.

11:05 a.m. Injected 200 c.c. of air left pleura.

Pressure - right, minus 7 to plus 11 cm.
left, minus 7 to plus 11 cm.

Chest ray A 7.

11:06 a.m. Injected 200 c.c. of air left pleura.

Pressure - right, minus 4 to plus 12 cm.
left, minus 4 to plus 12 cm.

Chest ray A 8.

11:07 a.m. Injected 200 c.c. of air into left pleura.

Pressure - right, minus 1 to plus 12 cm.
left, minus 1 to plus 12 cm.

Chest ray A 9.

Respiration ceased.

11:10 a.m. Pressure - right, plus 11 cm.
left, plus 11 cm.

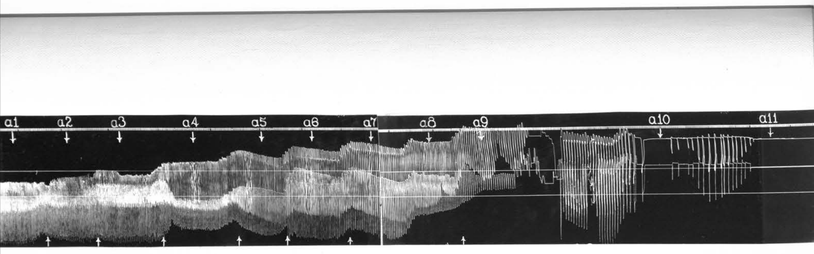
Chest ray A 10.

Few respirations occurred and again ceased.

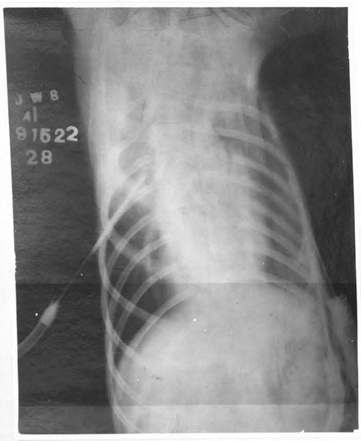
11:13 a.m. Pressure - right, plus 10 cm.
left, plus 10 cm.

Chest ray A 11.

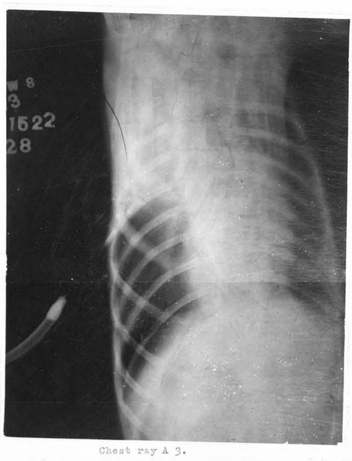
Opened right pleura and pressure on the left fell to zero. Then tried to raise pressure in the left pleura by injecting air but found this impossible. Very slight movement of the anterior mediastinum occurred. Then ran fluid into the left pleura. It did not pass through the posterior mediastinum but passed readily through the anterior mediastinum. Pathologically, the lungs and pleurae were negative. The chest rays showed a bilateral pneumothorax with no deviation of the mediastinum.



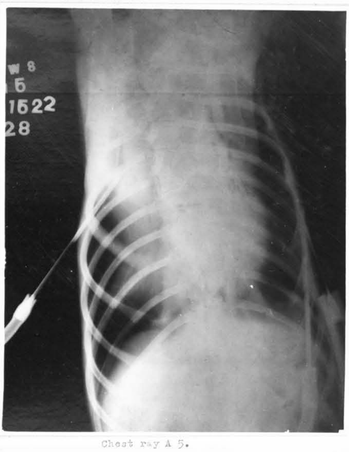

Dog 28. (Ether anesthesia) Simultaneous manometrical tracings of the pleural pressures of each side during the injection of air into the left pleural cavity. The top line represents the time in seconds. The upper curve represents the left pleural pressure and the lower curve the right pleural pressure. The base lines for each pleural cavity are drawn at atmospheric pressure. At the points designated A 1, A 2, A 3, etc. the corresponding chest rays were made. The arrows below the curves designate the points at which air in 200 c.c. amounts was injected into the left pleural cavity.



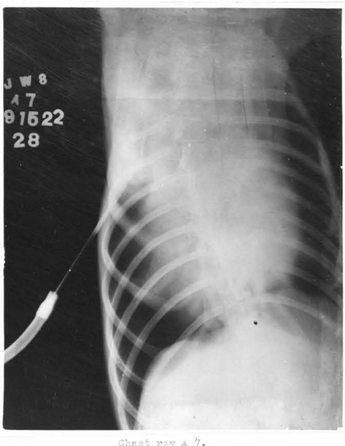

Chest ray A 1



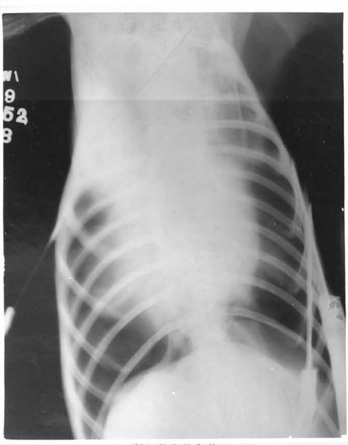

Chest ray A 3.



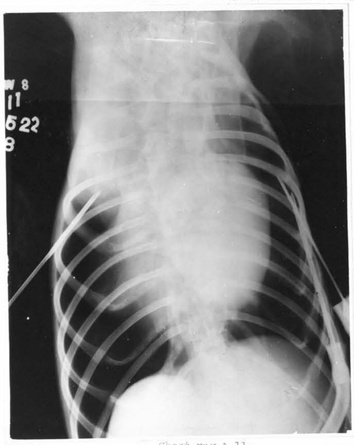

Chest ray A 5.




Chest ray A 7.




Chest ray A 9




Chest ray A 11.

Dog 28. Chest rays A 1, A 3, A 5, A 7, A 9 and A 11. The curves of the two pleural pressures are not identical probably because of technical faults. Their close similarity, however, will be readily seen and we may conclude that the pressures on the two sides are virtually identical. The injection of air into the left pleural cavity causes an immediate and corresponding alteration in the pleural pressure of the opposite side. As may be seen from the chest rays this is accomplished not by a displacement of the mediastinum but by the development of a bilateral pneumothorax.

Dog 29. F 201. Weight 7.8 kg. 9/18/22.

Abdomen open. Pancreas previously removed. Ether anesthesia.

3:04 p.m. Pressure - right, minus 9 to minus 5 cm. water.
left, minus 9 to minus 5 cm.

Chest ray A 1.

3:06 p.m. Injected 200 c.c. of air into the right pleural cavity.

Pressure - right, minus 7 to minus 2 cm.
left, minus 7 to minus 2 cm.

Chest ray A 2.

3:08 p.m. Injected 200 c.c. of air into the right pleural cavity.

Pressure - right, minus 5 to 0 cm.
left, minus 5 to 0 cm.

Chest ray A 3.

3:10 p.m. Injected 200 c.c. of air into the right pleural cavity.

Pressure - right, minus 3 to 0 cm.
left, minus 3 to 0 cm.

Chest ray A 4.

Injected 200 c.c. of air into the right pleural cavity.

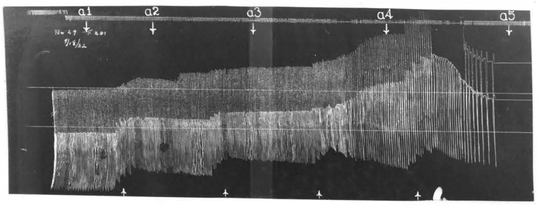
Produced death of animal 3:11 p.m.

Pressure - right, plus 4 cm.
left, plus 4 cm.

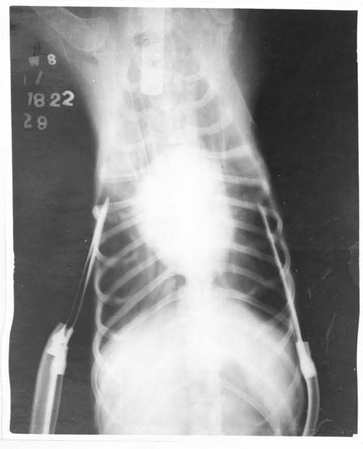
Chest ray A 5.

Opened the right pleura and pressure on the left fell to zero. Could not raise pressure in the left pleura by pumping air into the cavity. Pathologically, lung and pleura were negative. Chest rays showed a bilateral pneumothorax with no displacement of the mediastinum.

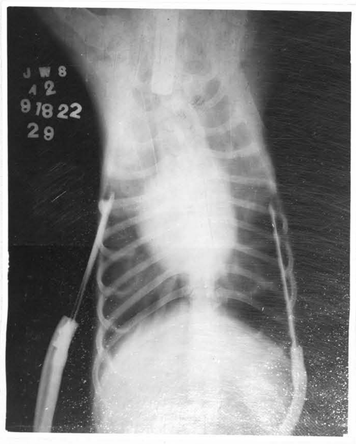
- - -



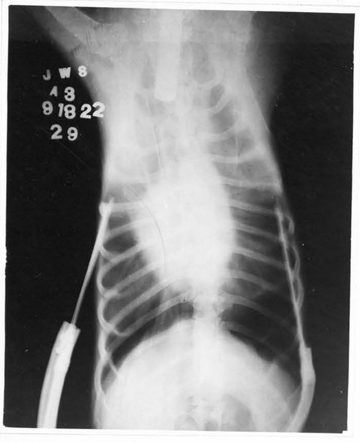

Dog 29. (Ether anesthesia) Simultaneous tracings of the pleural pressures of each side during the injection of air into the right pleural cavity. The upper curve represents the left pleural pressure and the lower curve the right pleural pressure. The corresponding base lines were drawn at atmospheric pressure. The points A 1, A 2, A 3, etc. indicate the time at which the corresponding chest rays were made. The arrows below the curves designate the points at which air in 200 c.c. amounts was injected into the right pleural cavity.



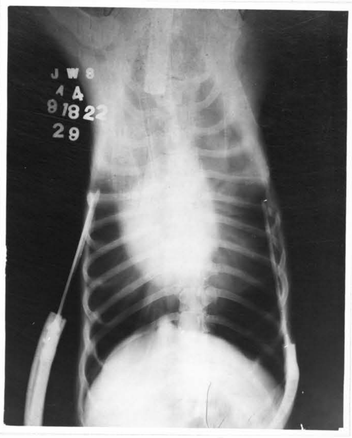

Chest ray A 1.



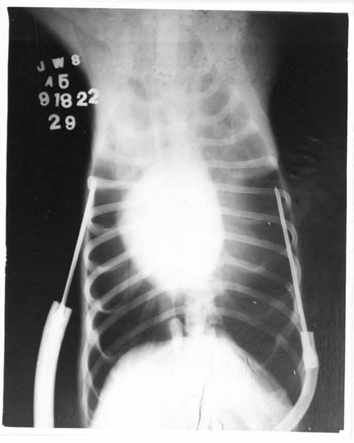

Chest ray A 2.




Chest ray A 3.




Chest ray A 4.




Chest ray A 5.

Dog 29. Chest rays A 1, A 2, A 3, A 4 and A 5. The close similarity of the curves of the two pleural pressures is evident in this experiment. The development of a bilateral pneumothorax without displacement of the mediastinum may be noted from the corresponding chest rays.

The following two dogs were employed to investigate the existence of cohesive force between visceral and parietal pleurae, as described by Auquier.

Dog 30. F 596. Dead twelve hours.

Chest ray A 1.

Opened right pleural cavity.

Chest ray A 2.

Dog.31. F 714. Weight 6.7 kg.

Animal sacrificed after removal of pancreas and kidney.

Dead three hours.

Chest ray A 1

Opened pleura.

Chest ray A 2.

In both dogs a bilateral pneumothorax was present occurring as readily on the side opposite to the injury as on the side of injury itself. This would seem to disprove the existence of a cohesive force existing between the two pleural surfaces.

– – – –

The following series of experiments were carried out on human cadavers to ascertain as closely as possible the parallelism between pressure relations in the dog and man.

Case 1.H.W.R. #394767. Age 6 yrs.

6/30/22 Death from operative repair of spina bifida with septic cerebro-spinal meningitis. Autopsy two hours after death.

Pressure – right, pleura – minus 3 cm. water.
left, pleura – minus 3 cm.

Forced air into the right pleura.

Pressure – right, plus 10 cm.
left, minus ½ cm.

Forced more air into the right pleura.

Pressure – right, plus 26 cm.
left, plus 1 cm.

After few seconds pressures changed to

Pressure – right, plus 20 cm.
left, plus 2 cm.

Abdomen then opened.

Pressure – right, fell to plus 10 cm.
left, fell to minus 1 cm.

Forced air into right pleura.

Pressure – right, plus 15 cm.
left, plus 1 cm.

Opened left pleura.

Pressure on right fell to plus 11 cm.

Then forced air into the right pleural cavity until a pressure of plus 60 cm. $H_2O$ was reached. The liver was forced well down into the abdomen, and the heart displaced beyond the left chest wall.

Pathologically both lungs were free from adhesions and the pleural cavities contained no fluid. No evidence of pneumonia.

Case 2. Mr. C.E.F. #392787. Age 64.

7/16/22. Death 7:05 p.m. Autopsy 9:00 p.m. Body warm, rigor mortis absent.

Cause of death: Primary squamous cell carcinoma of right pulmonary apex (7 cm. diameter) with generalized metastases.

Abdomen opened.

Pressure – right, plus 1½ cm.
left, plus 1 cm.

Forced air into the left pleural cavity.

Pressure – right, plus 9 cm.
left, plus 5 cm.

Again forced air into the left pleura.

Pressure – right, plus 14 cm.
left, plus 6 cm.

Then opened right pleura.

Forced air into the left pleura until pressure of plus 46 cm.$H_2O$ was reached. The diaphragm bulged into the abdomen on the left and slight displacement of the heart to the right occurred. The anterior mediastinum bulged as a tense membrane. A few adhesions were found at the right apex and anterior margin of the upper lobe. Left lung free.

Case 3. Mrs. G.G. #398339. Age 57.

7/24/22. Death 4:15 p.m. Autopsy 5:30 p.m.

Cause of death: Carcinoma of the gall bladder with metastasis to liver. Obstruction of the common duct by a stone, jaundice and purpura.

Pressure - right, minus 4 cm. water.
left, minus 3.5 cm. "

Forced air into the left pleura.

Pressure -left, plus 8 cm.
right, minus 1 cm.

Forced more air into the left pleura.

Pressure - left, plus 28 cm.
right, plus 1 cm.

Opened right pleura and pressure on the left fell to plus 20 cm.

Forced air into the left pleura until pressure of plus 50 cm. was reached.

The left diaphragm was forced into the abdomen and the mediastinal structures displaced about 5 cm. to the right.

On removing the sternum the anterior mediastinum was seen to be about 1 cm. in width in its thinnest place. Both lungs were free from adhesions.

Case 4. Baby H. #401005. Age 3 days.

8/9/22. Died 7:00 ;.m. Autopsy 9:00 p.m.

Cause of death: Melena Neonatorum.

Pressure - right, 0 cm.
left, 0 cm.

Forced air into right pleura.

Pressure - right, plus 22 cm.
left, plus 1 cm.

Then opened abdomen and pressure on right fell to plsu 14 cm.

Then opened left pleura and pressure on right fell to plus 10 cm.

Then pumped air into the right pleura up to a pressure of plus 50 cm.$H_2O$. The liver was forced well into the abdomen and the heart was displaced outside the left chest wall. The lungs were found to be free from adhesions and the anterior mediastinum in its thinnest place appeared as a relatively substantial membrane.

Case 5. Mr. W. W. #403062. Age 56.

9/13/22. Died 11:30 p.m. Autopsy 10:00 a.m. 9/14/22

Cause of death: Postoperative pulmonary embolism. Gastro-enterostomy for duodenal ulcer.

Abdomen not opened.

Pressure - right, minus 2 cm. water.
left, minus 2 cm.

Pumped air into the left pleura.

Pressure - left, plus 50 cm.
right, plus 5 cm.

Both pleural cavities were free from adhesions.

Case 6. Mrs. J.C.B. #403720. Age 56.

9/11/22. Died 4:00 p.m. Autopsy 6:15 p.m.

Cause of death: Pulmonary embolism. Bilateral carcinoma of ovaries with metastases.

Pressure - right, minus 6 cm.
left, minus 6 cm.

Forced small amount of air into the right pleura.

Pressure - right, plus 2 cm.
left, minus 1 cm.

Forced small amount of air into the left pleura.

Pressure - right, plus 2 cm.
left, plus 5 cm.

Forced more air into the left pleura.

Pressure - right, plus 4 cm.
left, plus 15 cm.

Then opened abdomen.

Pressure - right, minus 3 cm.
left, plus 8 cm.

Forced more air into the left pleura.

Pressure - right, plus 1 cm.
left, plus 30 cm.

Then pumped small amount of air into the right pleura.

Pressure - right, plus 18 cm.
left, plus 30 cm.

Forced air into the left pleura.

Pressure - right, plus 20 cm.
left, plus 50 cm.

Opened right pleura and pressure on left fell to plus 20 cm.

Found the mediastinum bulging into the right pleural cavity and diaphragm forced down into the abdomen on the left. No adhesions found in either pleural cavity.

Case 7. Mr. J.S. #264780. Age 45.

7/28/22. Death at 7:25 a.m. from cardiovascular lues with cardiac decompensation. Autopsy 9:15 a.m.

Abdomen opened.

Pressure - right pleura, minus 4 cm. water.
left pleura, minus 4 cm.

Chest ray A.

Forced air into the left pleura.

Pressure - right, 0 cm.
left, plus 18cm.

Chest ray B.

Forced more air into left pleura.

Pressure - right pleura, 0 cm.
left pleura, plus 50 cm.

Chest ray C.

Opened right pleura and pressure fell to plus 40 on the left. Forced more air into the left pleura until pressure plus 45 cm $H_2O$ was attained. This caused noticeable displacement of the heart and mediastinum to the right but even with this pressure a large right pleural cavity remained. The anterior mediastinum was tense but no distinct bulging occurred. On opening both pleurae the anterior mediastinum was seen to be about 1½ cm. in thickness in its thinnest place. Both lungs were free from adhesions. The heart was of the cor bovinum type.

Case 8. Mr. J.E.G. #115495. Age 42.

9/13/22. Death from cerebral tumor 9:00 p.m.

9/14/22 Autopsy, 8:30 a.m.

Pressure - right pleura, minus 3 cm. water
left pleura, minus 4 cm.

Chest ray A.

Opened abdomen.

No change in pressures.

Forced air into the left pleural cavity.

Pressure - right, plus 10 cm.
left, plus 11 cm.

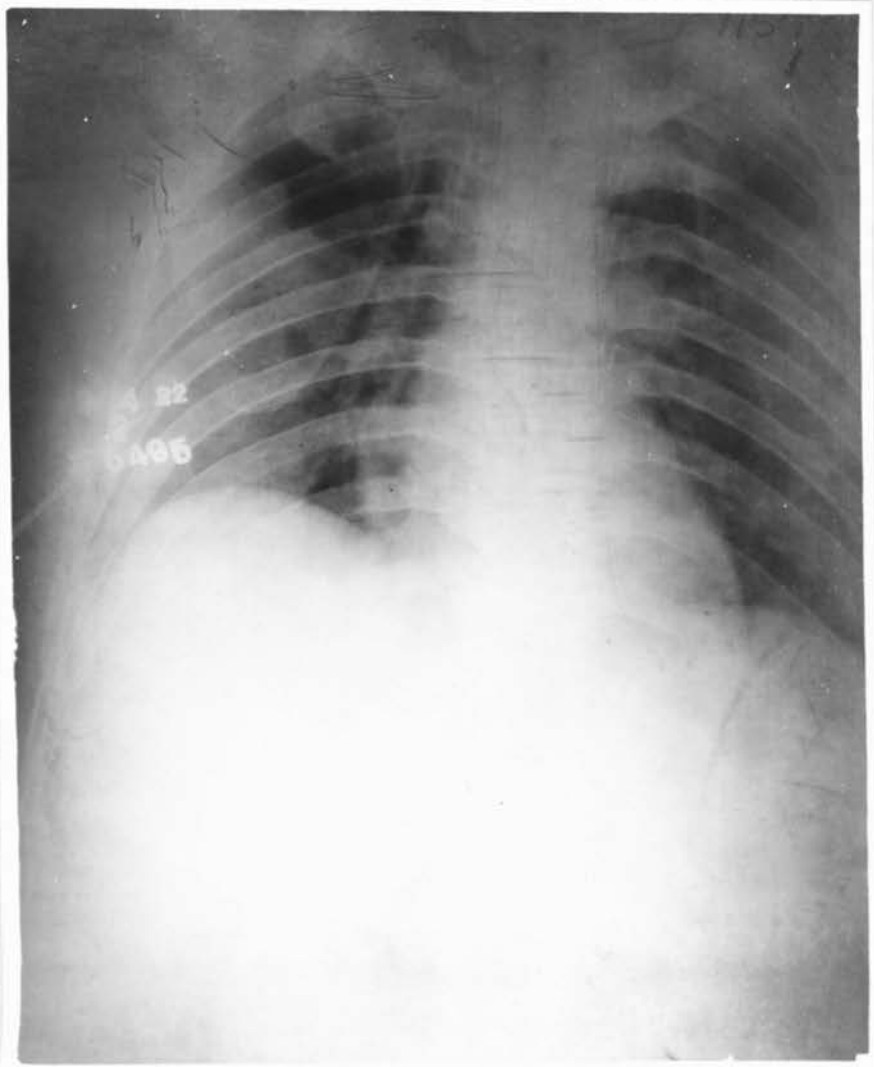
Chest ray B.

Forced more air into the left pleura.

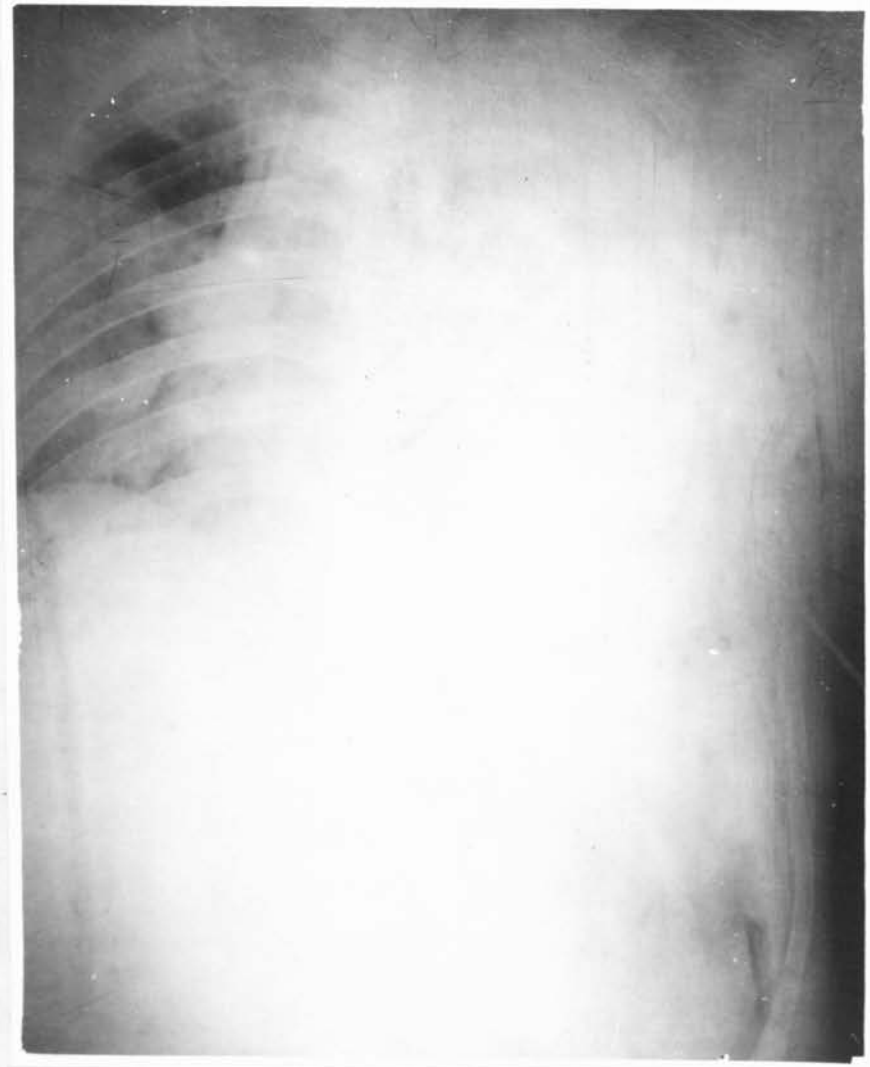
Pressure - right, plus 20 cm.
left, plus 32 cm.

Chest ray C.

Ran 200 c.c. of 10% K Br solution into the left pleural cavity.



*Case 8. #115495. Chest ray A. Before introducing potassium bromid solution.*



*Chest ray B. After introducing potassium bromid solution into the left pleural cavity and showing its unilateral distribution.*

Chest ray D.

From the chest rays a more marked displacement of the mediastinum was evident in this case than in case 6. The bromide solution appeared to be confined to the left pleural cavity. Both pleural cavities were free from adhesions.

Case 9. Mr. J.W.K. #403291. Age 62.

Died 6:45 p.m. Autopsy 9:00 p.m. 9/8/22.

Cause of death: Choledochotomy for stones in a dilated common duct.

Postoperative general peritonitis.

Pressure - right pleura, minus 3 cm. water
left pleura, minus 3 cm.

Forced small amount of air into each pleural cavity.

Pressure - right, minus 3 cm.
left, minus 2 cm.

Chest ray 1.

Forced more air into the right pleura.

Pressure - right, plus 8 cm.
left, 0 cm.

Chest ray 2.

Forced more air into the right pleura.

Pressure - right, plus 32 cm.
left, plus 4 cm.

Chest ray 3.

After few seconds pressures fell to

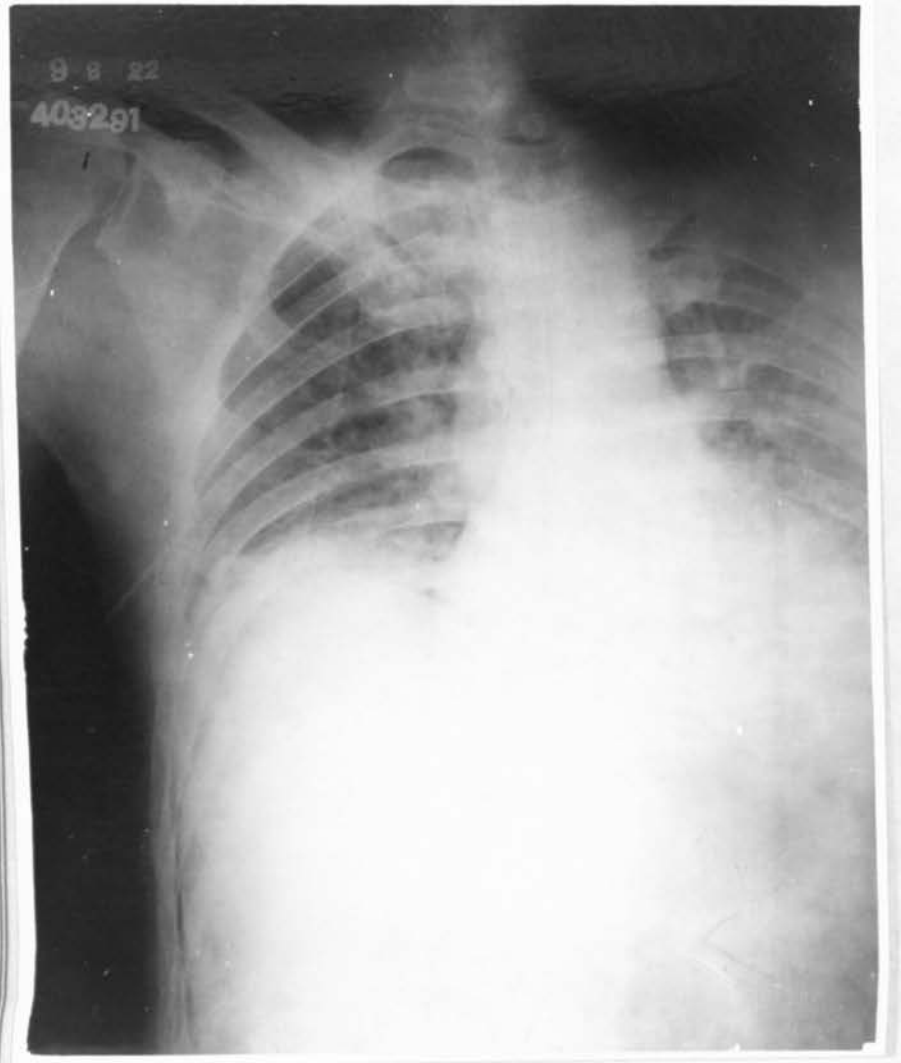
Pressure - right, plus 22 cm.
left, 0 cm.

Forced more air into the right pleura.

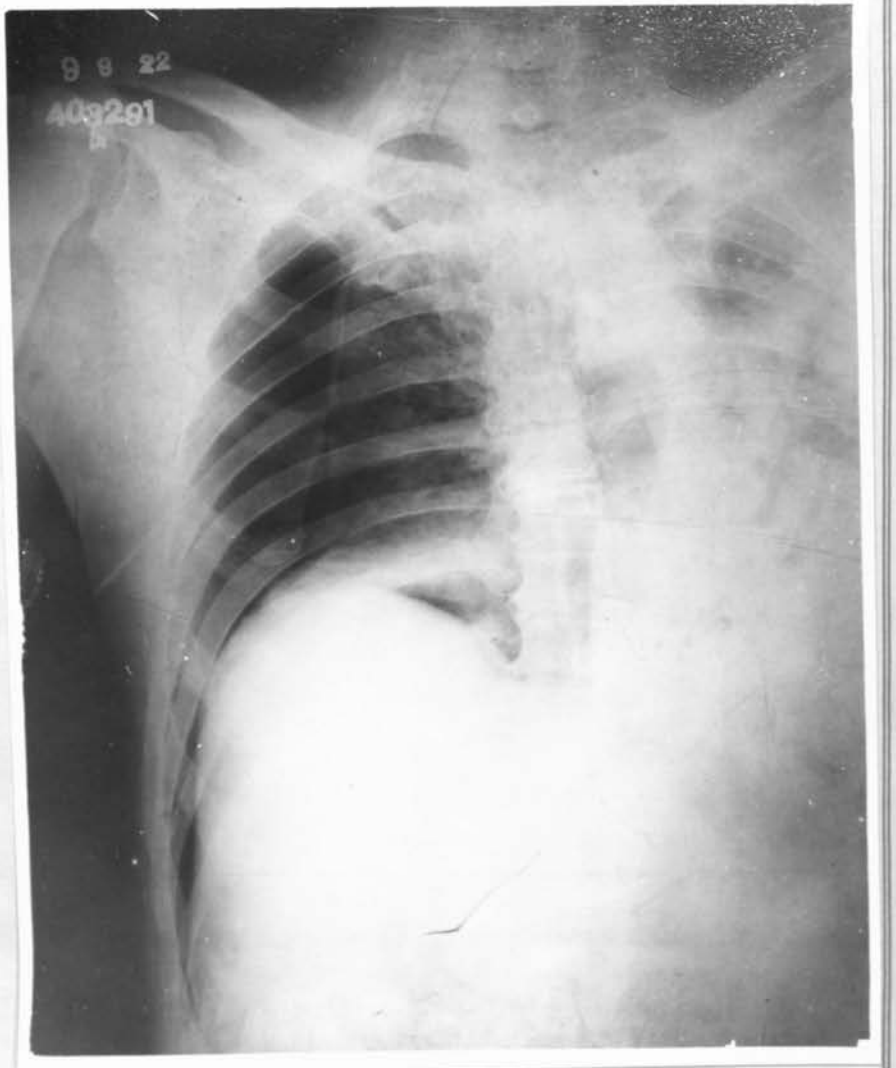
Pressure - right, plus 54 cm.
left, plus 10 cm.

Chest ray 4.

Permitted air to escape from right pleura through needle. Pressure




Case 9. #403291. Chest ray 1. Before the induction of a unilateral pneumothorax.




Chest ray 4. After inducing a right pneumothorax and showing the displacement of the mediastinum.

on left fell to 0. Ran in 1000 c.c. of 10% K Br. solution into the right pleural cavity. Pressure on left became plus 10 cm.

Chest ray 5.

Turned body on face.

Chest ray 6.

Opened right pleura and found it full of fluid. The heart and mediastinum were displaced to the left. The left pleura contained no fluid. There were a few adhesions on the posterior surface of the right hilus and right apex. The chest rays showed a marked displacement of the mediastinal structures to the left.

Case 10. Mr. H.D. #405132. Age 18.

9/18/22 Death 6:10 p.m. Autopsy 10:00 p.m.

Cause of death: Anterior poliomyelitis.

Pressure - right pleura, minus 2 cm. water.
left pleura, minus 5 cm.

Injected small amount of air into the right pleura.

Pressure - right, minus 1 cm.
left, minus 5 cm.

Injected small amount of air into the left pleura.

Pressure - right, minus 1 cm.
left, minus 4 cm.

Injected more air into right pleura.

Pressure - right, plus 4 cm.
left, minus 4 cm.

Injected more air into right pleura.

Pressure - right, plus 10 cm.
left, minus 3 cm.

Injected more air into right pleura.

Pressure - right, plus 18 cm.
left, minus 2 cm.

Injected more air into right pleura.

Pressure - right, plus 36 cm.
left, 0 cm.

Opened abdomen.

Pressure - right, plus 2 cm.
left, minus 4 cm.

Injected more air into right pleura.

Pressure - right, plus 14 cm.
left, minus 0 cm.

Opened left pleura, pressure on right fell to plus 4 cm.

Pathologically both lungs were free from adhesions.

It will have been noted that in several of the preceding cases air was first injected in small amounts into each pleural cavity. This was done with the object of creating a sufficient space about each lung to prevent closure of the needle by a displacement of the lung when the pressure was increased on the opposite side. In the following case this precaution was observed.

Case 11. Mr. S.W. #404109. Death 12:36 a.m. 9/20/22. Autopsy 8:30 a.m.

Cause of death: Exophthalmic goiter.

Pressure - right, minus 3 cm. water.
left, minus 3 cm.

Injected small amount of air into right pleural cavity (100 c.c.)

Pressure - right, 0 cm.
left, minus 3 cm.

Injected small amount of air into left pleural cavity (100 c.c.)

Pressure - right, minus 2 cm.
left, minus 2 cm.

Injected more air into left pleura.

Pressure - right, 0.
left, plus 5 cm.

Injected more air into the left pleura.

Pressure - right, plus 2 cm.
left, plus 12 cm.

Opened abdomen.

Pressure - right, 0.
left, plus 7 cm.

Injected more air into left pleura.

Pressure - right, plus 6 cm.
left, plus 12 cm.

Injected more air into left pleura.

Pressure - right, plus 8 cm.
left, plus 54 cm.

Opened right pleura and pressure on left fell to plus 30 cm.

Pathologically there was a small adhesion at the apex of the left lung.

- - - -

Granting that a negative tension must be established within the pleural cavity at some stage of respiration, the following case is an example of the tremendous handicaps for which the body can compensate.

Case 12. Mr. E.K. #404313. Age 15.

9/17/22 Died 6:00 p.m. Autopsy 7:30 p.m.

Cause of death: Acute gangrenous appendicitis, generalized peritonitis, bilateral hydrothorax.

Pleural pressure - right, plus 24 cm. water.
left, plus 24 cm.

Opened abdomen and pressures fell to

Pressure - right, plus 11 cm.
left, plus 11 cm.

Forced a small amount of air into left pleural cavity.

Pressure - left, plus 16.
right, plus 12.

At this time the left pleura was inadvertently opened and no further readings were possible. The abdomen showed a gangrenous appendix with a generalized sero-purulent peritonitis and marked tympany. Both pleural cavities contained about 1000 c.c. of cloudy serous fluid.

## Discussion

From our experiments it would seem evident that a unilateral pneumothorax does not occur in the dog. Conclusions drawn from experiments on the dog in which the parallelism of the two pleural pressures was explained by a displacement of the mediastinum are evidently erroneous. A bilateral pneumothorax must exist in each case. The anterior mediastinum of the dog is of paper thinness, permeable to both air and fluids while in man it is a membrane of some thickness and considerable resistance. A "weak place" is described in the posterior mediastinum in both the dog and man situated posterior to the heart. In the dog this area is sometimes permeable to fluids and in other cases not. Permeability of the dog's mediastinum to fluids is,we find,not a new observation. Garland in 1877, after injecting melted cocoa butter into a dog's pleura noted a bilateral distribution and Calvert states "The mediastinum of a dog readily permits fluid to flow from one pleura into the other".

In man neither the anterior nor posterior mediastinum is permeable to air or fluids. The changes of pleural pressures in man on the side opposite to a pneumothorax are due to displacement of the mediastinum. While the pressures on the two sides are comparable they are not identical as in the dog. Burrell and Salisbury as the result of taking bilateral pressures in a case of induced pneumothorax state "It may be concluded therefore, that in compressing one lung by artificial pneumothorax one does not interfere with the pressure in the opposite pleural cavity, even if the mediastinum is displaced." In contrast to this, Simon, as the result of bilateral pressure observations in three cases of artificial pneumothorax, states, "An artificial pneumothorax on one side of the chest affects the intrapleural pressure on the opposite side and this effect is probably transmitted to the corresponding lung". He found an average difference of pressure of only 1 cm. $H_2O$ between the two sides.

We have had no opportunity of taking bilateral pleural pressures in cases with artificial pneumothorax. However, the observations on Cases 22 and 23 with bilateral pleural effusions showed a notable difference of pressure on the two sides at the same level. If the mediastinum could be ignored as a factor the pressures would be identical. Weitz, in one case of bilateral effusion, found a difference of 6 cm. $H_2O$ between the two sides increasing to a difference of 11.5 to 13.5 cm. after aspiration.

We believe that with a small variation in the intrapleural tension on one side a shift in the mediastinum will compensate for it and equal pressures will again be established on the two sides.

However, with a decided change on one side as with open pneumothorax the natural resistance of the human mediastinum is exerted and equal pressures do not exist. We have never been able to rupture a human mediastinum with pressures up to 50 cm. $H_2O$ so that it seems to have considerable stability. We have noted that the thickness of the anterior "thin portion" of the mediastinum varied largely depending on the amount of adipose tissue between the two pleural surfaces but of fibrosis which is mentioned so frequently we have not seen an example.

Perl's early experiments on 100 consecutive cadavers in which one pleura was opened at a time and each lung in turn, unless firmly adherent, registered its own elastic tension on the manometer, would seem to bear out our contention. If the pressures on the two sides were identical then opening of the second pleural cavity should show no further change in the level of the manometer. It is true that the first lung frequently registered twice or three times as much as the second but if the mediastinum could be ignored the second lung would never register an additional pressure.

Therefore, we believe that experiments conducted on dogs with relation to pneumothorax may not be applied directly to man but due attention should be given to the differences in anatomy and physiology in the two instances.

10-20 5M

Cohesion as a factor in maintaining expansion of the lung may, we believe, be ignored. No evidence could be found of its existence in the dog, for collapse of the lung on the side which had incurred no injury occurred in a regular manner.

Normal negative intrapleural tension is dependent on the elastic recoil of the lungs. By expansion of the chest or descent of the diaphragm the lungs are stretched and their power of elastic recoil increased and with it the negative intrapleural tension. Coughing or other forced expiratory acts, by decreasing the capacity of the thorax, produce a positive intrapleural pressure and with it a compression of the lungs. The normal range of intrapleural tension in the adult during quiet respiration varies from minus 7 to minus 9 mm.Hg. with inspiration to minus 3 to minus 5 mm. Hg. with expiration. In the infant the intrapleural tension is zero as the lungs completely fill the thorax. With increasing age the thorax seems to grow away from the lungs and a negative intrapleural tension is established. Normally there is a perfect balance in the tensions of the two sides of the thorax. Unequal expansion of the two sides of the thorax may, however, cause a slight difference. Changes in the intrapleural tensions on one side of the thorax of moderate degree can be compensated for by a deviation of the mediastinum, and equal tensions again become established. Larger differences in the two sides compensate with greater difficulty depending on the resistance of the mediastinum.

## Summary

To summarize we may state:

A. Normal intrapleural tension is dependent upon the elastic recoil of the lungs.

B. Pathological conditions alter the intrapleural tension.

1. In pleural effusions pulmonary elasticity is partially nullified and the resultant tension is the residuum of unsatisfied

elasticity of the affected or the contralateral lung.

2. The pressure exerted at any point in the body of an effusion is the algebraic sum of the negative intrapleural tension and of the height of the column of fluid which is always positive.

3. Pressure determinations vary with the point of puncture in relation to its depth in the fluid and with the position of the patient.

C. Comparison of the mediastinum in the dog and man have shown that:

1. There was an essential difference in the mobility of the mediastinum in the dog and man.

2. A comparison of the stability and strength of the structure showed a marked difference in the two instances.

3. A change in the intrapleural tension of one pleural cavity produced a corresponding change on the opposite side but of different degree in the two instances. In man such a change was more or less compensated for by a deviation of the mediastinum. In the dog the mediastinum was not only freely mobile but freely permeable to air and fluids and an equalization of the intrapleural tension occurred to a very exact degree.

4. An induced unilateral pneumothorax in the dog cannot be produced. A bilateral pneumothorax uniformly results because of the permeability of the mediastinum.

5. Experimental results obtained in the dog with relation to surgical pneumothorax may not be applied to man without recognition of the difference in the structure of the mediastinum in the two instances.

6. Pneumothorax in man, if unilateral in origin, remains unilateral and the pleural tensions may or may not become equal on the two sides depending on the resistance of the mediastinum and the initial differences in tension between the two sides.

In addition we may conclude that cohesion is probably not a factor in maintaining expansion of the lungs.

# BIBLIOGRAPHY


1. Adamkiewicz and Jacobson: Centralbl. f. d. med. Wissensch.,1873, 483.

2. Aron, E.: Ueber einen Versuch, den intrapleuralen Druck am lebenden Menschen zu messen. Arch. f. path. Anat.,1891, cxxvi, 517-542.

3. Aron, E.: Der intrapleurale Druck bein lebenden gesunden Menschen. Arch. f. path. Anat., 1900, clx, 226-234.

4. Auquier, E.: Du rôle des tensions et des pressions intrathoracique dans le pneumothorax traumatique. Gaz. hebd. d. sc. méd. de Montpel., 1882, iv, 557; 598; 609; 617.

5. Bard, L.: Sur la pression intrapleurale dans le pneumothorax. Rev.de méd., 1901, xxi, 449-576.

6. Bard, L.: Pression des epauchements pleuraux. Rev. de méd.,1902, xxii, 269-340.

7. Barr, J.: The pleura: Pleural effusion and its treatment. Brit. Med. Jour., 1907, ii, 1289.

8. Bell, J.: The principles of surgery. 8 ed. 1826.

9. Bendele, R.: Der Druck im Cavum pleurae des Pferdes. Arch. f. d. ges. Physiol., 1911, cxxxix, 593-610.

10. Bernstein, J.: Ueber die Entstehung der Aspiration des Brustkorbes bei der Geburt. Arch. f. d. ges. Physiol., 1878, xvii, 617-623.

11. Bernstein, J.: Weiteres über die Entstehung der Aspiration des Thorax nach der Geburt. Arch. f. d. ges. Physiol., 1884, xxxiv, 21-37.

12. Borelli: De motu animalium. 1743.

13. Boyle: Quoted by Hutchinson.

14. Brauer, L.: Die Ausschaltung der Pneumothoraxfolgen mit Hilfe des Ueberdruckverfahrens. Mitt. a.d.Grenzgeb. d. Med. u. Chir.,1904, xiii, 483-500.

15. Brauer, L.: Der Druck zwischen den beiden Pleurablättern. Beitr. z. path. Anat. u. z. allg. Path., 1905, 7th. Suppl., 762-776.

16. Burrell and Salusbury: Report on artificial pneumothorax. Medical Research Council, London, 1922.

17. Calvert, J.: On the intrapleural pressure and manner of contraction of the lung in pleural effusion. St. Bartholomew's Hosp. Rep., 1892, xxviii, 131-135.

18. Carson, J.: Phil. Tr. Roy. Soc. London, 1820, i, 42.

19. Cruveilhier, M.: De l'empyeme et de quelques experiences sur les animaux. Bull. de l'Acad. de med.,Par., 1836, i, 280.

20. D'Arsonval, A.: Recherches théoriques et expérimentales sur le rôle de l'élasticité du poumon dans les phénomènes de la circulation. Theses, Paris, 1877.

21. Donders: Bijdrage tat het mechanisme von ademhaling en bloedsomloop. Ned. Lancet, 1849, v. Translation in Ztschr. f. rational Med.,1953, iii, 287.

22. Dwyer: The mechanism by which the lung is reinflated. Researches of the Loomis Laboratory of the Medical Department of the University of the City of New York, 1890, No. 1.

23. Edmunds, A.: The mechanism of respiration in pneumothorax. Brit. Med. Jour., 1903, ii, 1322.

24. Flurin and Rousseau: Les tensions intra-pleurales. Ann. de Med., 1920, vii, 325.

25. Fredericq: Arch. de biol., 1882, iii, 55.

26. Garland, G.M.: Pneumodynamics. New York, 1878, p. 53.

27. Garre, C. and Quincke, H.: Lungenchirurgie. Jena, Fischer, 1912, p.21.

28. Gerulanus, M.: Ueber die chirurgische Behandlung von Lungenkrankungen. Deutsch. Aerzte-Zeitung, 1902, i, 193-197; 225-229.

29. Graham, E.A.: The maximal nonfatal opening of the chest wall. Jour. Am. Med. Assn., 1919, lxxiii, 1934.

30. Graham and Bell: Open pneumothorax: Its relation to the treatment of empyema. Am.Jour. Med. Sc.,1918, clvi, 839.

31. Haller: De respiratione esperiment anatomica. Pars I and II. Gottingen, 1746-1747.

32. Hamberger: Physiologia medica sen de actionibus corporus humani, etc. Jena, 1751.

33. Hedblom, C.A.: Open pneumothorax in its relation to the exstirpation of tumors of the bony chest wall. Arch. Surg.,1922, iv, 588.

34. Heger and Spehl: Recherches sur la fistule pericardique chez le lapin. Arch. de biol., 1881, ii, 153.

35. Hellin, D.: Der doppelseitige Pneumothorax und die Unabhängigkeit der Lungenrespiration von den Druckverhältnissen. Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1907, xvii, 414-430.

36. Hermann, L.: Das Verhalten des kindlichen Brustkastens bei der Geburt. Arch. f. d. ges. Physiol.,1883, xxx, 276-287.

37. Hewson: The operation of paracentesis thoracis proposed for air in the chest; with some remarks on emphysema and on wounds of the lung in general. Med. Observations and Inquiries, 1767, iii.

38. Heynsius, A.: Ueber die Grosse des negativen Drucks im Thorax beim ruhigen Athmen. Arch. f. d. ges. Physiol.,1882, xxix, 265-311.

39. Homolle, G.: De la tension intrathoracique dans les épanchments pleuraux et de l'emploi du manomètre dans la thoracentèse. Rev. méns. de méd. et de chir., 1879, iii, 81-126.

40. Hutchinson, J.: Capacity of the lungs and the respiratory function. Med.-Chir. Tr., 1846, xxix, 137.

41. Hutchinson, J.: Thorax. Todd Encyclopedia of Anat. and Physiol., 1849-1850, 1059.

42. Lehmann: Beitrage zur Kenntnis des Donderschen Druckes, etc. Arch. f. d. ges. Physiol., xxx.

43. Leyden: Manometrischen Messung über den Druck innerhalb der Brust. Charite Ann., 1876, iii, 264.

44. Line, C.: Pneumothorax chirurgical. Theses, Paris, 1907.

45. Lower, R.: Quoted by Hutchinson. Phil. Tr., 1667, i, 179.

46. Luciani and Rosenthal: Arch. f. Physiol. von duBois-Reymond, 1880, Suppl. Bd., 34.

47. Macewen, W.: Some points in the surgery of the lung. Brit. Med. Jour., 1906, ii, 1.

48. Matas, R.: The value of artificial aids to respiration. Arch. Surg.,1922, v, 110-133.

49. Meckel: Sur de l'air repandu ramasse dans la cavite du thorax qui arretait la respiration et qui a cause la mort. Mem. de l'Acad. Roy. de sc. de Berlin, 1759, xv.

50. Meltzer, S.J.: Intrathoracic pressure. Jour. Physiol., 1892, xiii, 218.

51. Muralt, L.v.: Manometrische Beobachtung bei der Ausübung der Therapie der kunstlichen Pneumothorax. Beitr. z. Klin. d. Tuberc., 1911, xviii, 539.

52. Murphy, J.B.: Surgery of the lung. Jour.Am.Med.Assn., 1898, xxxi, 151.

53. Northrup: The effect of opening the pleural cavity. Researches of the Lommis Laboratory of the Medical Department of the University of the City of New York, 1890, i.

54. Perl: Deutsch. Arch. f. klin. Med., 1869,vi, 1.

55. Peyrot: Sur les tensions intra-thoraciques dans les epauchements de la pleura. Arch. gen de med., 1876, ii, 47.

56. Pique: The chest. Dict. Encyclop. d. sc. Med., 1888, xxvi, 481.

57. Pitres, A.: Les signes physiques des e'panchements pleuraux. Arch. clin. de Bordeaux, 1896, v, 65.

58. Powell: Lung showing perforation from a case of pneumothorax. Tr. Path. Soc. London, 1868.

59. Powell: Notes on pneumothorax occurring in phthisis. Med. Times and Gaz., 1869.

60. Quincke, H.: Zur Behandlung des Pleuritis. Berl. klin. Wchnschr.,1872, ix, 65.

61. Quincke, H.: Ueber den Druck in Transudaten. Deutsch. Arch. f. klin. Med., 1878, xxi, 453.

62. Reinboth: Deutsch. Arch. f. klin. Med., 1897, lviii.

63. Rosenthal: Ueber den intrathoracalen Druck. Arch. f. Anat. u. Physiol., 1882, 152.

64. Roth, J.: Ueber den interpleural Druck. Beitr. z. Klin. d. Tuberc., 1905, iv, 437.

65. Sauerbruch: Zur Pathologie des offenem Pneumothorax und die Grundlagen meiner Verfahrens zur server Ausschaltung. Mitt. a. d. Grenzgeb. d. Med. u. Chir.,1904, xiii, 399.

66. Schreiber, J.: Ueber Pleural und Peritoneal Druch unter pathologischen Verhaltnissen. Deutsch. Arch. f. klin. Med.,1883, xxxiii, 485.

67. Schreiber,J.: Zur physikolioschen Untersuchung des Oesophagus und des Magens. Deutsch. Arch. f. klin. Med.,1883, xxxiii, 425.

68. Sehrwald: Zum Atmenmechanismus bei offenem Pneumothorax. Deutsch. med. Wchnschr.,1882, Aug. 22.

69. Seifert: Pneumothorax. Deutsch. Arch. f. klin. Med.,1883, xxxiii, 157.

70. Sussdorf: Die Atmung. Ellenberger, Handbuch der vergbickenden Physiol. d. Haussangetiere, 1890, 630.

71. Tendello: Studien in der Ursachen der Lungenkrankheiten. Weisbaden bei Bergman, 1902.

72. Van der Brugh, J.P.: Ueber eine Methode zur Messung der interpleural Druckes. Arch. f. d. ges. Physiol., 1900, lxxxii, 591.

73. Weil: Zum Lehre vom Pneumothorax./ Deutsch. Arch. f. klin. Med.,1879, xxv, 1.

74. Weil: Weitere Mittheilungen über Pneumothorax. Deutsch. Arch. f. klin. Med., 1886, xl.

75. Weitz, W.: Über den Druck in Pleuraegüssen. Deutsch. Arch. f. klin. Med., 1908, xcii, 526.

76. West: Cases of pneumothorax. Tr. Clin. Soc. London, 1886, xix, 227.

77. West: Pneumothorax. Brit. Med. Jour., 1887, ii, 393.

78. West: Intrapleural tension. Méd.-Chir. Tr., 1898, lxxxi, 273.