WAR MEDICINE

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OPEN PNEUMOTHORAX: ITS RELATION TO THE TREATMENT OF EMPYEMA.[1]

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INTRODUCTION. In the preliminary report of the Empyema Commission the opinion was given that in the type of cases of empyema which accompanied the prevailing streptococcus pneumonia, operation should not be undertaken early but should be deferred,

[1] This experimental work has been carried out at the Hunterian Laboratory, Johns Hopkins Medical School, through the courtesy of Dr. W. G. MacCallum, to whom we are indebted for many kindnesses.

in order to allow time for the pneumonia to subside and for the empyema to become more localized by the formation of adhesions. One of the principal considerations which led to this opinion was the question of the possible harm to the patient caused by the creation surgically of an open pneumothorax in the presence of an already existing state of severe asphyxia. Notwithstanding the apparently striking clinical confirmation of the soundness of this point of view, as shown by our remarkable reduction in the mortality of 4.3 per cent. as compared with the average mortality of 30.2 per cent. noted in the replies of the various camps to the Surgeon-General's questionnaire of February 21, 1918, it seemed desirable to submit to experimental test some of the considerations which theoretically had led the commission to the adoption of deferred operation. This seemed especially desirable, since there was a possibility that our low mortality might have been due in some measure to a decrease in virulence of the organism. Accordingly, work has been carried out along the following lines: (1) a study of the effects of open pneumothorax in normal and diseased chests, (2) the experimental production of a streptococcus pneumonia and empyema in dogs comparable with that in the human, (3) a study of the effect of early operation in dogs with experimentally produced streptococcus empyema. The limited amount of time available for these investigations prevented as complete a study of the problems as seemed desirable, but nevertheless certain facts have been established which seem to have an important bearing on the method of treating the condition.

PART I.—PNEUMOTHORAX.

Definition. Under normal conditions each pleural cavity is completely filled by its corresponding lung and the cavity of which one speaks is only a potential one. When air is present in a pleural cavity the condition of pneumothorax exists. Air may enter either from an opening through the thoracic wall or from a communication with the lung. If the opening remains patent so that air passes in and out freely the condition is known as an "open pneumothorax." If air is enclosed within a pleural cavity so that its exit is blocked a "closed pneumothorax" is produced. Another type of pneumothorax, commonly called "valve pneumothorax," occurs when air may enter freely but cannot pass out again. The latter type is found usually in connection with communications between the lung and the pleural cavity, especially in tuberculosis and in pulmonary abscess. It is the first type—namely, open pneumothorax—which is of primary interest in this discussion, for it is this condition which is produced by early operation and drainage in cases of empyema. If the pleural infection is not sharply walled off by adhesions the operation will create a pneumothorax, and the insertion of a drainage tube, by continuously allowing the free passage of air into and out of the chest, will maintain an open pneumothorax after the operation.

Importance of Negative Intrapleural Pressure in Maintaining Respiration. By far the most harmful effects, and perhaps the only harmful ones, of a pneumothorax are the changed pressure relationships within the thorax. Normally, each pleural cavity has a potential negative pressure, and it is owing to this negative pressure that respiration can go on. At each inspiration the thoracic cavity is enlarged. This enlargement and the accompanying tendency of the lungs to lag behind obviously makes the pressure still lower within the pleural cavities until after the lungs have been filled with air, expiration occurs and, with the diminution in size of the pleural cavity, the intrapleural pressure tends to approach the atmospheric pressure but does not actually reach that height unless the glottis is closed. These pressures, of course, even normally, are subject to wide variations, depending upon the amplitude of the respiratory movements. They have been measured repeatedly in various ways by numerous observers in human cadavers, in the living human and in animals. Donders,[2] who was apparently the first to measure them, considered the normal intrapleural tension in man at the end of quiet expiration to be −7.5 mm. Hg. and at the end of inspiration −9 mm. Hg. Aron[3] made determinations on a living normal man and found at the height of quiet inspiration an average reading of −4.64 mm. Hg. and at the height of quiet expiration −3.02 mm. Hg. For a more detailed discussion of these points reference may be made to the extensive works of Emerson,[4] Sauerbruch[5] and L. Mayer.[6] It is not within the province of this article to review the literature on pneumothorax exhaustively, since this has been well done by each of the three authors just mentioned. Instead, we shall be concerned here only with those literature references which deal particularly with the salient points of our problem—namely, the relative harmfulness of open pneumothorax.

Prevalent Conceptions of the Mechanics of Open Pneumothorax. In the light of our experiments the prevalent conceptions of open pneumothorax seem to be based on an incorrect understanding of the condition. Practically, without exception, they start from the apparently erroneous assumption that the mediastinal structures constitute an unyielding, almost rigid, partition between the two sides of the thoracic cavity. This assumption in turn has given rise to the idea that one lung is collapsed and that the other lung alone carries on respiration, although somewhat impeded, perhaps, if the opening be a large one, by the bulging of the mediastinum against

[2] Quoted by Heynsius, Arch. f. d. ges. Physiol., 1882, xxix, 267.

[3] Die Mechanik und Therapie des Pneumothorax, 1902, quoted by Emerson, p. 194.

[4] Pneumothorax, Johns Hopkins Hosp. Rep., 1903, xi, 1.

[5] Zur Pathologie des offenen Pneumothorax und die Grundlagen meines Verfahrens zu seiner Ausschaltung, Mitteil. a. d. Grenzgeb. d. Med. u. Chir., 1904, xiii, 399.

[6] Les Bases Physiologiques de la Chirurgie Pleuropulmonaire, Ann. d. l. Soc. Roy. d. Sciences Méd. et Nat. de Bruxelles, 1906, xv, 1.

it. Garré,[7] for instance, in a series of diagrams to represent the conditions in open pneumothorax, shows the mediastinum as a straight line, in cases in which the opening is small, with one lung collapsed and retracted and the other lung (on the unopened side) of normal size and apparently unaffected. In cases with a large opening he represents essentially the same condition except for a slight bulging of the mediastinum away from and toward the opening on inspiration and expiration respectively. Here, again, the lung on the affected side is shown as contracted into a small mass about the hilum, whereas the opposite lung appears to be relatively unaffected. L. Mayer, after reviewing the literature extensively, states the situation in regard to pressure relationships in open pneumothorax as follows: "On the healthy side the modifications due to the pneumothorax with a small opening ought to adjust themselves by a deviation of the mediastinal pleura, the two surfaces of which are no longer submitted to conditions of identical equilibrium; on inspiration the lowering of intrapulmonary pressure of −7 mm. Hg. not manifesting itself on the fistula side, the mediastinal pleura ought to become curved toward the healthy side and narrow the expansion of the other lung; the inverse should be produced at expiration. In reality these theoretical differences are minimal and are scarcely established by experimentation. If, on the contrary, the pleural opening is widely gaping the atmospheric air enters and goes out freely at each respiratory movement and the lung of this side will not be called upon at all to become distended. At each inspiration the pressure of the two sides of the mediastinal pleura will be different; on the healthy side, negative pressure of 7 mm. Hg., on the other side atmospheric pressure, with aspiration of the mediastinum toward the healthy side, toward which it ought to be markedly convex. In moderate and light expiration the pressure on the two sides remains equal; but if a forced or sudden expiration supervenes the pressure is raised in the normal lung while it remains constant on the incised side, giving a convexity of the mediastinum on the side of the pneumothorax." It is seen, therefore, in Mayer's resumé of the common conceptions regarding the pressure relationships in open pneumothorax that there are several assumptions ordinarily taken for granted: (1) that a marked inequality of pressure exists in the two pleural cavities after the establishment of a communication between one pleural cavity and the external air; (2) that this marked inequality of pressure results in the more or less complete collapse and retraction of the lung on the opened side while the other lung remains but slightly affected; (3) the mobility of the mediastinum is a relatively unimportant factor. These conceptions are so commonly accepted that one finds ingrained in all the literature of pneumothorax references to the "sound lung,"

[7] Garré's Diagrams are reproduced in L. Mayer's article (loc. cit.). The originals were inaccessible to us.

the "healthy lung," the "unaffected lung," etc., when what is meant is the lung in the unopened pleural cavity, as if it were little, or not all, affected by the opening in the opposite pleural cavity.

Prevalent Conceptions of Open Pneumothorax Seem to be Erroneous in Light of Our Experiments. In our experiments, on the contrary, we have found that normally the mediastinal structures are so mobile that they offer practically no resistance to pressure on either side. In the normal human (both adult and child) the resistance offered by the mediastinum, when one pleural cavity contains air at a known pressure equivalent to that of 10 cm. of water, corresponds to a pressure of only 1 cm. or less of water, which, of course, is practically negligible. For all practical purposes, therefore, from the standpoint of pressure relationships, 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 conceptions 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. Roughly speaking, also, the degree of asphyxia which will follow the creation of an open pneumothorax will depend upon (1) the relation of the amount of air entering through the pneumothorax opening to the amount of air permitted to pass down the air-inlet to the lungs, and (2) the ability of the individual to compensate by increasing either the rate or amplitude of his respiratory movements, or both. The dog's mediastinal structures have the same mobility as the human beings; therefore, experimental results obtained on the living dog are applicable to man. The importance of these considerations in regard to the surgical treatment of empyema will be shown later under "General Discussion."

Sehrwald,[3] as long ago as 1889, arrived at some of the same conclusions which we have drawn from our work. His conclusions, however, seem not to have been based on much experimental work; and, perhaps for that reason, they have not influenced opinion so much as their soundness entitled them to do. For example, he stated that the influence of a unilateral open pneumothorax on respiration depends not on the absolute diameter of the opening but on the relation between the size of this opening and the diameter of the air passage to the lungs. He concluded also that the mobility of the mediastinum was a very important consideration; if it were delicate and easily stretched it would be aspirated so strongly against the "sound" lung that it would hinder its expansion. He made the important statement, furthermore, that very young individuals and those who previously had had normal thoracic organs, because of the delicacy and elasticity of their mediastinum, stand an open pneumothorax with much more difficulty than patients in whom,

[3] Zum Atmungsmechanismus beim offenen Pneumothorax, Deutsch. med. Wchnschr., 1889, xiv.

because of a strong thickening, the mediastinum has been converted into a firm, unyielding membrane. It is to be noted, however, that even he also had the conception of a "sound lung" as opposed to a collapsed lung, and that he did not recognize that the pressures in the two pleural cavities are practically equal, with the result that both lungs are collapsed to the same extent.

PART II.—EXPERIMENTAL RESULTS.

Parallelism of Thoracic Pressure Relationships in Dog and Human. Experiments have been carried out on both animals and human cadavers. In order to determine whether results obtained on living animals could be considered as applicable to the human, it seemed especially desirable to ascertain, if possible, the comparative mobility of the dog's mediastinum with that of the human under the same pressure relationships, since the dog seemed to be in general the most suitable animal for a study of the living conditions. The experiments which show this comparison will be described first. Since the human experiments could be carried out only on cadavers the procedure chosen was to compare the relationships in the human cadavers with those in recently killed normal dogs and then to compare these results with those obtained by similar means in living dogs. In this manner it was found that the experimental results obtained on living dogs were directly applicable to the living human. The experiments were carried out as follows:

A metal cannula (diameter 4 mm.) was inserted into each pleural cavity (usually in the fifth interspace in about the anterior axillary line). Each cannula was attached by rubber tubing of the same size and kind to a tambour which, by means of an indicator, registered the pressure changes on a smoked drum. In one rubber tube was interposed a T-tube, which was connected in one direction with a water manometer and in the other with a rubber atomizer bulb. By means of the bulb, air was forced into the corresponding pleural cavity until the manometer registered 10 cm. pressure. Simultaneously with the rise of the indicator on the tambour of that side the other indicator also became elevated above its base line. Calibration of the reading of the second indicator showed the equivalent of a pressure of 9 or 9.5 cm. of water. In other words, when a known pressure, equivalent to 10 cm. of water, was established in one pleural cavity there was practically the same pressure in the opposite pleural cavity. This experiment has been carried out on five human cadavers, two of which were infants of four months and fourteen months respectively, one a child, aged fourteen years, and two adults. Duplicate determinations on the same cadaver sometimes showed a variation of 0.5 cm. in the reading of the opposite side, due probably to experimental error: that is, when the pressure of the inflated side was the equivalent of 10 cm. of water,

that of the opposite side registered sometimes only 9 cm. and at other times 9.5 cm. No difference was noted whether the right or left pleural cavity was inflated. These results were strictly comparable with those which were obtained on normal dogs which had been killed with ether. It should be stated, however, that cadavers which had been kept in the refrigerator for several hours showed a difference in pressure sometimes of as much as the equivalent of 3 cm. of water, but control dead dogs kept in the same refrigerator for the same length of time always showed the same variations. Since the objects sought in the experiments was really to determine the extent of mobility of the mediastinum, it was necessary to rule out the possibility of pressure being transferred to the opposite side by being distributed through the diaphragm of the inflated side to the abdomen and from there through the diaphragm of

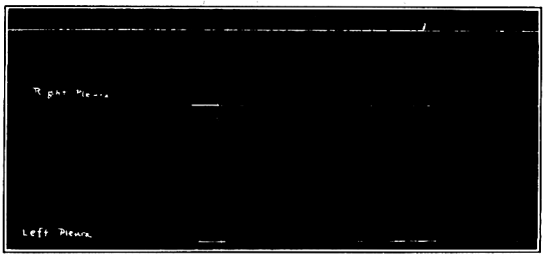


FIG. 1.—Tracing showing that when the left pleural cavity of a fresh adult human cadaver is inflated with air at a pressure of 10 cm. of water the right pleural cavity registers a pressure of 9 cm. The top line is a record of a pressure of 10 cm. made with the tambour attached to the right pleural cavity. Calibration showed that the actual pressure was 9 cm. of water.

the opposite side to that pleural cavity. In order to exclude such a possibility the abdomen was opened by a long median incision before inflation of the pleural cavity. By so doing the transfer of any extra pressure to the opposite pleural cavity by way of the abdomen was obviated. 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. of water the rigidity of the mediastinal partition between the pleural cavities in both dogs and the human amounts only to the equivalent of from 0.5 to 1 cm. of water pressure (0.4 to 0.8 mm. of mercury), a value which obviously is practically negligible. (See Figs. 1, 2 and 3.)

It was suggested to us by Dr. Howell that perhaps a more conclusive demonstration of whether or not both lungs are nearly

equally compressed by raising the pressure in one pleural cavity could be made by comparing the relative densities of the two lungs when one pleural cavity was inflated with air at a known pressure. If the pressure relationships in both pleural cavities are affected about equally by altering the pressure in one then both lungs

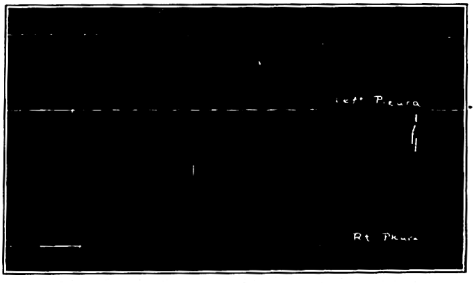


FIG. 2.—A similar tracing with the right pleural cavity inflated. Calibration showed that in this case also there was a difference in pressure of only 1 cm. of water (about 0.8 mm. of mercury).

should be about equally compressed and should show about equal relative densities.

In order to determine this point the following method was used: A normal dog was killed with ether or chloroform and into one pleural cavity air was injected until the pressure was equal to that of

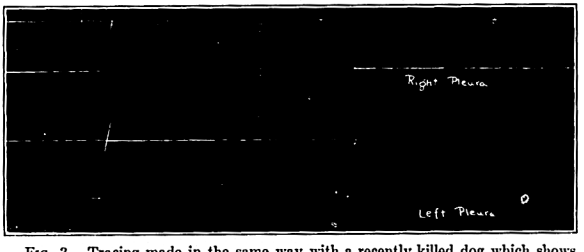


FIG. 3.—Tracing made in the same way with a recently killed dog which shows that the dog is strictly comparable with the human, since here also the difference in pressure between the two pleural cavities amounted to only 1 cm. of water.

10 cm. of water. Through a low tracheotomy in the neck, plaster-of-Paris paste was then pushed down into each main bronchus in order to block its lumen. After allowing the plaster to "set" for a few minutes the lungs were then removed. In this way it was possible to remove the lungs so that they remained in the actual condition

in which they were before the chest was opened. The weight and volume of each lung was then determined, the latter by measuring the amount of water displaced. The plaster was then thoroughly removed from the bronchi and its weight and volume subtracted from that of the corresponding lung. In this manner the specific gravity of each lung was approximately determined, which indicated fairly closely the degree of compression of each lung. When dogs are killed with ether or chloroform the condition of the lungs is not uniform even when nothing else is done to them. One lobe may be well inflated while another is partly collapsed even on the same side. The specific gravity of the two lungs may vary considerably. In some cases the left and in other cases the right lung is the denser. But when the lungs are collapsed by inflating one pleural cavity these variations are not so apparent. The experimental results on five dogs are as follows:

	Density of lungs. Left.	Right.
No. 1. Right pleural cavity inflated with air to a pressure equal to that of 10 cm. of water	.74	.74
No. 2. Left pleural cavity inflated	.73	.69
No. 3. Left pleural cavity inflated	.70	.72
No. 4. Control, not inflated	.52	.48
No. 5. Control, not inflated	.43	.51

The results thus demonstrate that the density of one lung practically equals that of the other when one pleural cavity is inflated with air—in other words, there is practically the same amount of compression of both lungs. These results, therefore, furnish additional evidence in favor of the principal conclusion to be drawn by our experimental work—namely, that when the intrathoracic pressure relationships are changed by the creation of an open pneumothorax both lungs are practically equally affected.

Experiments on the Living Dog. Experiments on the living dog not only demonstrate the fact that there is practically an equilibrium of pressure throughout the thorax, as already noted in the recently killed dog and in a fresh human cadaver, but they afforded an opportunity to investigate the extent to which an open pneumothorax produces dyspnea and asphyxia, the mechanism of the attempts at compensation, etc. The method used was in general the same in all the experiments. It consisted essentially in recording simultaneously on a smoked drum the pressure changes in each pleural cavity and in the trachea and the changes in the amplitude and rate of the respiratory movements. To record the variations in pleural pressure the same cannulas as already described above were inserted into the pleural cavities and connected with Marey tambours by means of rubber tubing. The changes of tracheal pressure were recorded in practically the same way by attaching a glass T-tracheal cannula to another tambour, and the variations in the rate and amplitude of the respiratory movements were noted by attaching a fourth tambour to a spring tambour, which was tied to the animal's chest.

When an opening was made into a pleural cavity, therefore, its effects could be at once determined by the movements of the various indicators on the revolving smoked drum. Most of the experiments were conducted under general anesthesia, either urethane or ether, but a few were performed with local cocain anesthesia. No important difference was observed between the changes in pleural pressure produced by the open pneumothorax when an animal was under the influence of general anesthesia and those produced when the opening in the pleural cavity was made under merely local anesthesia with cocain. The factor of general anesthesia, therefore, in most of the experiments can be considered as having no appreciable influence on the results. In the control experiments with cocain no attempt was made to record the tracheal pressure or the changes in the respiratory movements. In all thirty-eight dogs have been used for the work on pneumothorax. In addition, similar experiments have been carried out on three cats and two rabbits. No essential differences were observed regardless of the kind of animal used.

A marked difference in the character of the reaction by the animal occurs, depending upon whether a small or a large opening is made in a pleural cavity. By small or large opening is meant small or large in comparison with the air-inlet to the lungs. Normally the diameter of the air-inlet is the diameter of the opened glottis, but generally in our experiments it was the diameter of the tracheal cannula. A more detailed discussion of the important relationship which exists between the amount of air which enters the lungs and that which enters the pleural cavity will be given later in this article. The characteristic effects produced by a pleural opening smaller than the air-inlet to the lungs may be summarized thus: Immediately upon making the pleural opening there is an increase of pressure in both pleural cavities. Whereas, before making the opening both the inspiratory and expiratory pressures are entirely negative, immediately afterward the pressure rises so much that it is almost entirely positive; the indicators descend below the base line of atmospheric pressure only at about the end of the inspiratory effort. During this time there is noticeable a slight pause early in the course of the expiratory movement just before the pleural pressure reaches that of an atmosphere in its ascent from the negative phase. Simultaneously, also, the tracheal pressure changes in such a way that the indicator practically rests on the base line of atmospheric pressure instead of sweeping above and below the base line on expiration and inspiration respectively. This change is in accord with what is to be expected, since the tracheal pressure is a rough index of the amount of air passing down the trachea into the lungs, and since, obviously, with the pleural pressure almost entirely positive only a small amount of air can pass down the trachea into the lungs.

These changes are also coincidental with an effort on the part

of the respiratory mechanism to compensate by making a greater effort to get air into the lungs; accordingly there is a marked change in the respiratory movement. As a rule, this change is rather an increase in amplitude of respiratory movement than an increase in rate. If the amplitude is increased considerably the rate will of necessity be slower than before. If the pleural opening is small enough to allow compensation, the pleural pressures will gradually diminish so that negative pressure will be reëstablished and the tracheal pressure will rise simultaneously as the animal is enabled to get air into its lungs. The negative pressure, however, is never restored so completely as to reach the level at which it was before the opening was made, providing the opening remains in free communication with one pleural cavity. Instead of both the inspiratory and expiratory pleural pressures being negative, as in the normal state, the pressure at the height of expiration will be positive. The ability of the animal, therefore, to withstand an open pneumothorax is dependent upon his ability to compensate by increasing his respiratory effort. Obviously a strong, vigorous animal can compensate better than a weaker one. In our experience young adult dogs have uniformly withstood the harmful effects of an open pneumothorax better than old or weak and emaciated dogs.

It is a very striking fact that usually the restoration of negative pleural pressure can be instantly accomplished by simply closing the pleural opening, thus making a closed instead of an open pneumothorax. But here, again, the intrapleural pressure never becomes so low as it was before the opening was made. Presumably it would become normal as soon as the air contained in the pleural cavity was absorbed; but we have not tested this point. If the opening is made sufficiently large the animal will pass through a state of marked dyspnea and die of asphyxia within a few minutes, owing to his ability to compensate no matter how great his respiratory effort; for if the opening is so large that, despite his maximal respiratory efforts, he is unable to establish a negative pressure in the pleural cavity he will be unable to get air into his lungs, or if he is able to establish a slight negative pressure he may be able to get a little air into his lungs but not enough to maintain life. In this connection it is to be constantly remembered that, roughly speaking, an alteration of pressure in one pleural cavity is accompanied by an alteration of pressure to the same extent in the other pleural cavity. No such condition can be recognized as the collapse of the lung on the affected side and maintenance of respiration with only the other lung, as is the prevalent conception of open pneumothorax. Rather, both lungs must functionate about equally, since practically equal pressures are present in the two pleural cavities. In fact, if the opening into the pleural cavity be made in a favorable place for observation, and if it is not too large to prevent respiratory compensation, the lung on the affected side can

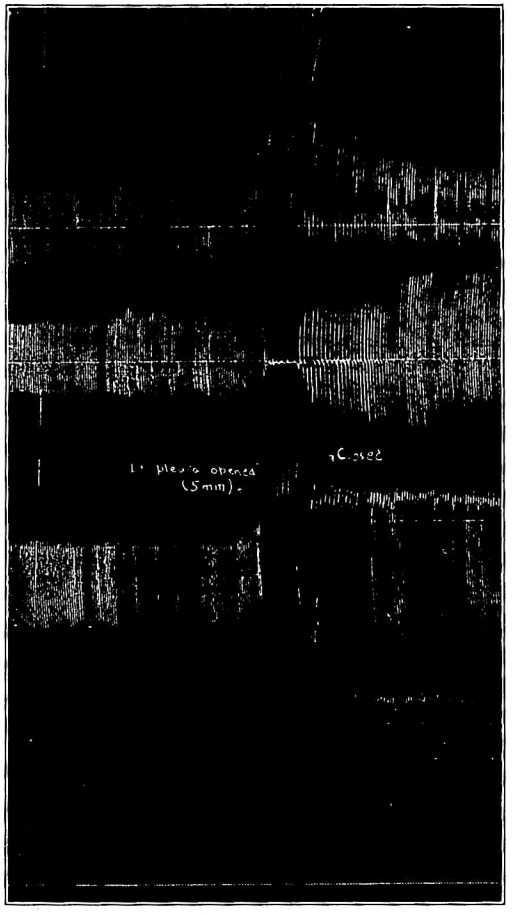
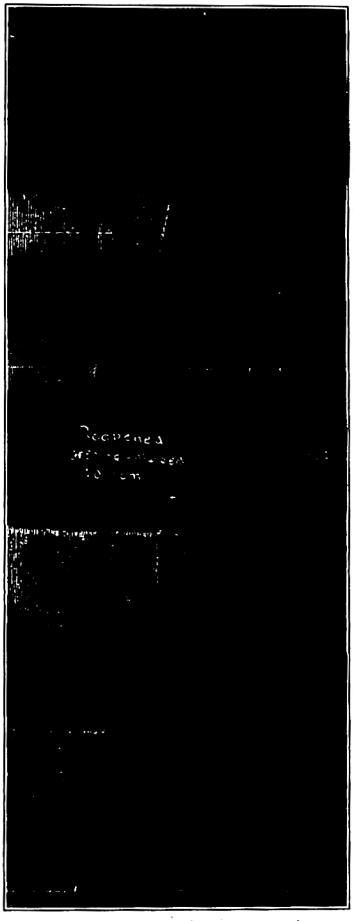


FIG. 4.—A tracing made on the living dog under ether anesthesia to show the nature of the reaction to an open pneumothorax with a moderate opening, as indicated in the changes in the respiratory movements, in the tracheal pressure and in the pressures in both pleural cavities. The upper tracings represents the respiratory movements, the next the tracheal pressure, the third the left pleural pressure and the fourth one the right pleural pressure. The lowest line indicates the time in seconds. The base lines were drawn at atmospheric pressure. The interval represents a duration of six minutes. Immediately after making the opening there is a simultaneous change of pressure in the pleural cavities from an entirely negative phase to one which



is mostly positive. The respirations are slowed but are increased in amplitude. Because the intrapleural pressure is largely positive, practically no air enters the trachea and the intratracheal pressure tends to be at equilibrium with atmospheric pressure. Immediately upon closure of the opening there is a simultaneous response in both pleural cavities, with restoration of negative pressure to a large extent, diminution of the amplitude of the respiratory movements and oscillations again of positive and negative intratracheal pressure, with inspiration and expiration. After an interval of six minutes during which time the air in the pleural cavities has probably been absorbed the intrapleural pressure has again become entirely negative.

be plainly seen to expand and contract with the inspiratory and expiratory efforts of the animal. Also, when a large opening is made, during the few minutes in which the animal remains alive the mediastinum can frequently be seen to flap from one side to the

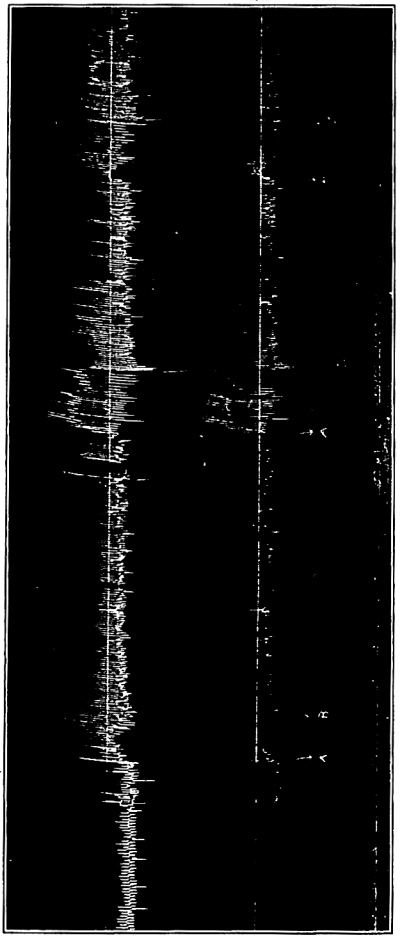


FIG. 5.—Tracing showing the simultaneous changes in intrapleural pressure in a dog when an opening of 5 mm. was made in the left pleural cavity with cocain anesthesia. The base lines again represent atmospheric pressure. The upper tracing is the right pleural pressure and the lower that of the left pleural cavity. At *A* and *B* in each case the chest was opened and closed respectively.

other with inspiratory and expiratory movements in much the same manner as a flapping sail caught in the breeze first on one side and then on the other. It sometimes happens that, with a severe expiratory effort, the mediastinal pleuræ are ruptured so that a double pneumothorax occurs. The various points about the changed pressure relationship may be best understood by reference to the accompanying reproduction of tracings. (See Figs. 4 and 5.)

Influence of Posture on Effects of Open Pneumothorax. Elsberg[9] has raised the point that an animal with an open pneumothorax may be favorably or unfavorably influenced by posture. He quotes Depage as saying that the prone posture is the best in thoracic surgery. As a result of experiments on dogs he concludes that the prone position enables the animal to withstand an open pneumothorax much better than the supine or a lateral position. In three experiments of our own on dogs we tested out the influence of a prone and supine position respectively and we were unable to note any beneficial influence exerted by the prone position. In fact, with the animal lying prone the difficulties of compensating for the opening in the pleura were increased owing to the necessity for the animal with each inspiratory effort to raise his weight from the table in order to enlarge his thorax with his inspiratory muscles. It is interesting, furthermore, in this connection that Emerson[10] after a most exhaustive review of the literature of pneumothorax states that "while, as a rule, the patient prefers to lie on the affected side there are two remarkable cases on record in which the knee-elbow position was chosen." Presumably the individual would choose whatever position would give him the most relief from his respiratory embarrassment, and the fact that there are only two cases on record in which preference was shown for a posture somewhat analogous to the prone would indicate that there is no decided relief gained usually from that posture. Probably the position which in a given case gives the greatest relief is somewhat dependent on the location of the opening and is brought about by the attempt on the part of the body to close the opening and thus to make a closed pneumothorax which, as will be shown later, causes the least respiratory embarrassment of all the types of pneumothorax if the amount of air enclosed is not too great. It seems possible that in the two exceptional cases quoted by Emerson the individuals had anterior openings.

Relation of Amount of Air Entering Thorax Opening to Amount of Air Entering Lungs. As has been mentioned earlier in this article the immediate influence of an open pneumothorax in producing respiratory difficulties depends upon the relation of the amount of air entering the pleural cavity to the amount of air entering the lungs.

[9] Pneumothorax and Posture, Jour. Exper. Med., 1909, xi, 444.
[10] Loc. cit., p. 390.

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. If an opening is made into the chest cavity, part of the air which enters the chest during inspiration will pass in through the trachea and part will come through the chest opening. Only that part of the air which enters through the trachea is effective in carrying on respiration, and to prevent asphyxia the animal must increase either the rate or depth of respiration. If we assume that the rate of respiration remains constant and that all compensation is effected by increasing the depth of respiration, then the change of chest volume which occurs at each inspiration must be made greater by the amount of air which enters through the opening. That is, if the animal requires a volume of air (T), at each inspiration, and if a volume (B) enters through the opening, then the increase in chest volume at each inspiration must be $T + B$ after the opening is made.

If, however, the rate of inspiration is increased after the opening is made the volume of air required at each inspiration will be reduced. If the rate was R_1 before and R_2 after the opening was made then a volume of only

$$\frac{R_1}{R_2} T$$

will be required at each inspiration, and the required increase in volume of the chest to secure the same amount of air per minute will be—

$$\frac{R_1}{R_2} T + B$$

There is, however, a limit to the possible compensation. The change in the chest volume from maximum expiration to maximum inspiration is the same as the "vital capacity" V. The maximum possible compensation is reached when the increase in chest volume is V and when R_2 is the greatest possible rate comparable with this depth of respiration. Under these conditions it is evident that—

$$B = V - \frac{R_1}{R_2} T$$

represents the greatest volume of air that can enter through the thoracic opening if the animal is to survive even for a short time.

The ratio of the volume of air entering the chest through the thoracic opening to the volume entering the lung through the natural air passages would be equal to the ratios of their areas if it were not for two facts: (1) the natural air passages to the lungs have a considerable length which causes appreciable friction and a resistance to the entrance of air, and (2) the natural elasticity of the lungs

offers further resistance. On account of these two factors an opening in the chest wall of a somewhat smaller size than the narrowest part of the natural air passages will admit the same amount of air. If the area of this narrowest part, which is the glottis, is C, and if the resistance to the passage of air makes this opening equivalent to an opening of an area aC located in the thoracic wall (a being less than 1), then the entire ratio of the volume of air entering the chest through the thoracic opening to the volume entering the lung through the natural passages will be—

$$\frac{X}{aC}$$

X being the area of the thoracic opening. But from the equation above the maximum of this same ratio is—

$$\frac{B}{\frac{R_1}{R_2}T} = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T}$$

and substituting we have—

$$\frac{X}{aC} = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T}, \quad \text{or} \quad X = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T}aC$$

In a normal human chest during rest the maximum area (X) of a thoracic opening compatible with life for even a short time can be obtained by substituting numerical values in this expression. The average vital capacity V and the tidal air T are given by Howell[11] as 3700 c.c., and 500 c.c., respectively. The normal rate of respiration R_1 during complete rest is about 15 per minute and the maximum rate R_2 for the greatest possible depth of respiration is about 60 per minute.

$$\frac{R_1}{R_2}T$$

then equals 125 and

$$X = \frac{3700-125}{125}aC = 28.6\,aC$$

The area of the human glottis may be taken as 2.25 sq. cm., and the value of a may be assumed to be about 0.8 for the maximum rate and depth of respiration. Therefore, $aC = 1.8$ sq. cm. and $X = 51.5$ sq. cm. In other words, an opening about 5 x 10 cm. (2 x 4.1 inches) is the

[11] Text-book of Physiology, Philadelphia, 1911, p. 646.

largest for which compensation can theoretically be established in the human even for short periods if the mediastinum has normal mobility. Practically, the opening must be somewhat smaller, since the extra work performed by the muscles of the chest to establish compensation increases the amount of air required. The presence of toxemia, infection or any other cause which increases the level of metabolism will decrease the safety limits of the maximum size of

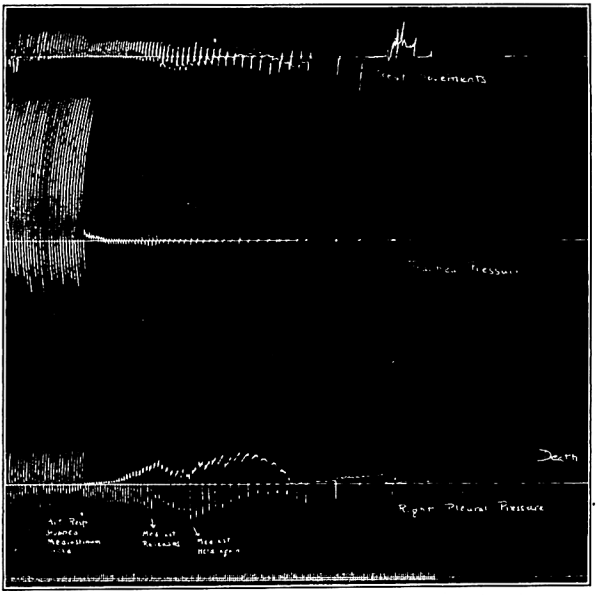


FIG. 6.—Showing slightly advantageous effect of holding of mediastinum in case of large opening into the left pleural cavity made by turning down a flap of pleura with six ribs while the dog was given artificial respiration by intratracheal insufflation.

the opening as will also any condition reducing the available breathing space of the lung. The use of general anesthesia will probably also act in the same way.

Effects of Immobilization of Mediastinum and Adhesions. Since the equalization of pressure in the thorax is dependent upon the mobility of the mediastinal structures, it would seem reasonable to suppose that any means of stabilizing the mediastinum would reduce the respiratory difficulties by confining the effects of the pneumo-

thorax to one pleural cavity. Old inflammation of the mediastinum with extensive induration will of itself result in a more rigid partition between the two pleural cavities. If, in addition to its being actually more rigid, the mediastinum is supported by strong adhesions the effect of an open pneumothorax on one side will be still less felt on the other. This is the condition which is often encountered in performing secondary operations of the type of Estlander, Schede, Delorme, etc., on old cases of empyema, and its presence probably accounts for the fact that in these operations the thorax can be opened widely with much less danger of respiratory embarrassment than is the case with the relatively normal mediastinum. These same factors also tend to make the deferred operation in acute empyema more safe than one performed too early. Experimentally, it can be shown that, furthermore, when the mediastinum is more or less fixed by pulling the lung well out of the pleural opening there is less pressure in the unopened pleural cavity than in the opened one. (See Fig. 6.) It is impossible, however, effectively to immobilize the normal mediastinum in that way because its extreme delicacy (especially in the anterior portion) allows it to be stretched and flapped about in spite of fixing it at one point by holding the root of the lung tight. It was hoped to demonstrate conclusively the influence which adhesions produced in various ways might have, but limitations of time have prevented it. The record of a single experiment, however, is presented. (See Fig. 7.) A few adhesions were produced between the lung and thoracic wall by the injection of 1 c.c. of turpentine. The record shows that during the intervals in which the left pleural cavity was open the right pleural pressure was distinctly less affected. On inspiration, for example, the pressure was twice as low in the right pleural cavity as in the left. This obviously enabled the dog to get more air into his lungs than he would have been able to do if the adhesions in the left pleural cavity had not to this extent prevented the aspiration of the mediastinum to the other side. Because of the elasticity of the lung tissue it is probable that extensive adhesions and those which involve the mediastinum itself are necessary in order to produce a more marked protective effect.

Closed Pneumothorax. In the description of the characteristic changes produced by making a free opening into the pleural cavity it was stated that the closure of the opening resulted in a sudden restoration of negative pleural pressure and a prompt relief from dyspnea. Obviously, after making the closure, air is still retained in the pleural cavity, so that the very striking difference in the phenomena observed must be due merely to the fact that an open pneumothorax has been converted into a closed one. Although air is absorbed from the pleural cavity, it disappears only slowly; and the sudden benefit noted by the closure of the opening cannot be attributed to the immediate disappearance of the air. Apparently

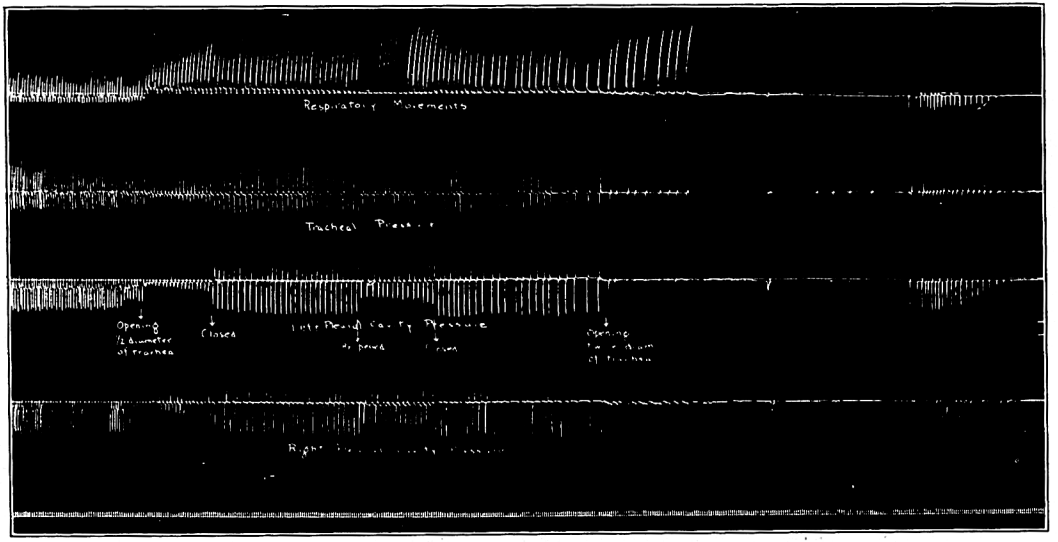


FIG. 7.—Adhesions between the left lung and thoracic wall. Opening made into left pleural cavity. Dog under ether anesthesia. Because of partial fixation of the mediastinum by the adhesions the right pleural cavity is less affected than the left. Respiration is maintained longer with the right lung than with the left, and there is more negative pressure on the right side than on the left when the left pleural cavity is opened.

the explanation of the relative harmlessness of a closed in comparison with an open pneumothorax lies rather in the fact that the ability to compensate for interference with the aëration of the lungs is limited. In a closed pneumothorax, no matter how much air is contained in a pleural cavity, no additional air can enter. It is necessary, therefore, only for the animal to increase his respiratory effort sufficiently to create enough negative pressure to allow him to take in the requisite amount of air to maintain aëration of his blood. Under conditions of rest this amount is equivalent only to the "tidal air," which in the human is about one-seventh of the total lung capacity. Accordingly, therefore, asphyxia should not occur until more than six-sevenths of the total lung capacity has been replaced by air in the pleural cavity, provided that the animal is at rest, that his ability to compensate by increasing his respiratory effort is good and that there is no extra abnormal demand for air such as might arise from toxemia. When an open pneumothorax is converted into a closed one, particularly if the closure is made at the end of expiration, the amount of air enclosed in the pleural cavity is very much less than six-sevenths of the total lung capacity, and naturally, therefore, there is comparatively little dyspnea. On the other hand in the case of an open pneumothorax there is an active competition for air going on between the trachea and the pleural opening. At each inspiration air not only enters the trachea but also enters the pleural cavity, and if the diameter of the pleural opening is the same as that of the trachea the same amount of air will enter the pleural cavity as enters the lungs and the animal will be compelled to increase his respiratory effort to get the "tidal air" in his lungs. If the opening is considerably larger than the trachea it will be still more difficult to get the required amount of air into the lungs, until when the pleural opening is of such size that more air than six-sevenths of the total lung capacity enters the pleural cavity with each inspiration the animal will no longer be able to obtain the requisite tidal air and he will die of asphyxia. This theoretical discussion is, of course, only an approximation and is not strictly accurate, since, as already shown in the paragraph dealing with the relationship of the diameter of the pleural opening to that of the air inlet to the lungs, it is necessary to consider the actual amount of air entering by each opening rather than merely the differences in diameter, a consideration which involves also the relative lengths of the pleural opening and the trachea with the resultant friction to the passage of the air. Observations, however, both clinical and experimental tend to confirm the truth of this explanation. For example, it is well known that an adult human can have as much as 2000 c.c. of fluid in his chest without producing alarming dyspnea so long as he remains at rest. Theoretically, he should be able to contain from 2500 to 3000 c.c. before developing a dangerous asphyxia if he remained quiet, and if it were not for the fact that his associated condition of toxemia and fever, uncompensated heart lesion, etc.,

demands a greater amount of oxygen than normal. Experimentally, we have injected into the pleural cavity of a dog of 8 kilos, through a small needle, as much as 1800 c.c. of air over an interval of twenty minutes without producing any marked asphyxia. Beyond that point, however, additional injections of only 50 c.c. at a time each had a very noticeable effect in increasing the dyspnea, and the animal died after about 2100 c.c. of air had been injected. The accompanying tracing illustrates the comparative harmlessness of a closed pneumothorax in which a moderate amount of air is present. Lest it should seem surprising that a dog of 8 kilos could withstand so large an amount of air as 1800 c.c. in his pleural cavity, it should be recalled that in proportion to the size of the rest of the body the dog has a much larger thorax than the human. (See Fig. 8.)

Effects of Open Pneumothorax other than Respiratory Disturbances. Besides its effects on respiration an open pneumothorax also induces other changes productive of harm to the body. These have been summarized by Sauerbruch[12] as heat loss, danger of infection and disturbances of the circulation. Sauerbruch himself has made some very important observations on the amount of heat loss. In unanesthetized rabbits in which he had established an open pneumothorax at a room temperature of from 20° to 22° C., the body temperature sometimes dropped as much as 3.5° C. within forty-five minutes. In dogs, also unanesthetized, within a half-hour he observed the body temperature fall 2° C. after making a pleural opening and rise again 1.6° C. within an hour after closing the opening. He also made the very striking observation that the heat loss in open pneumothorax is greater than in extensive laparotomy with eventration of the intestine for the same length of time. For example, a dog's temperature fell only 1° C. after having his intestine lying out of his abdomen for forty-five minutes, and a rabbit subjected to the same experiment showed a heat loss of only 1.3° C. after forty-five minutes.

The danger of infection is very great, since the ease with which organisms may be aspirated into the pleural cavity renders possible the infection of a relatively enormous amount of exposed surface. Naturally, also, a pleura already infected is exposed to secondary infection. That this readily occurs has been a frequent observation at Camp Lee in patients who had been operated on, and is a common experience to every surgeon. It explains, in many cases at least, the putrescent odor of the discharge so often present in old empyema cases. At Camp Lee we often found a great variety of bacteria after a pleural opening had been present for several days if means had not been taken to prevent the secondary infection, as by the use of Dakin's solution, etc. Ordinary aseptic dressing will usually not suffice to prevent the entrance of secondary invaders.

Marked disturbances in the general circulation have been noted by Sauerbruch as a result of the change of pressure relationships in the thorax, due to the pleural opening. He states that "in pneumo-

[12] Loc. cit.

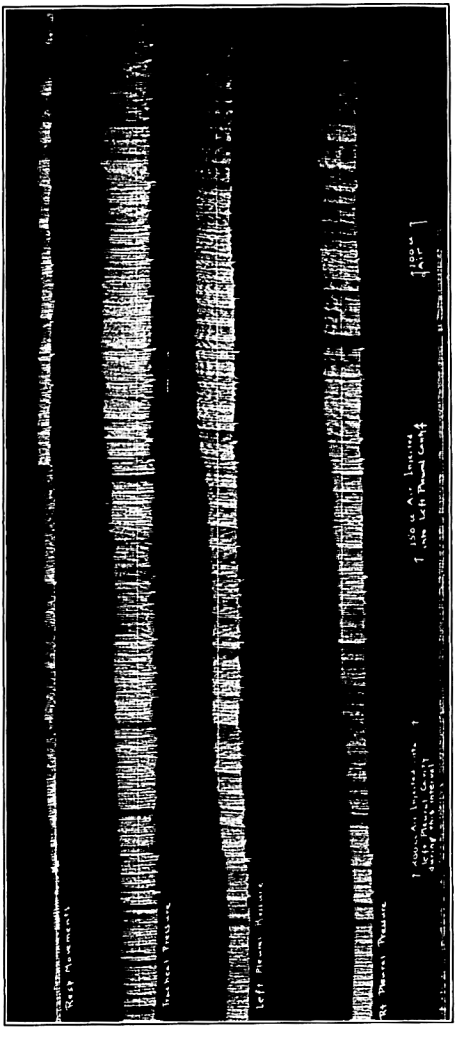


FIG. 8.—Closed pneumothorax in a 7-kilo dog under urethane anesthesia. It is to be noted that 450 c.c. of air can be injected into a pleural cavity without producing much distress, but again that the changes which result are strictly comparable in both pleural cavities.

thorax the aspiration of the heart fails; a stasis results in the venous system. Measurements of the venous pressure in the femoral vein give in fact an increase of the pressure." As a rule, there is not noteworthy change in the arterial pressure. When asphyxia occurs there is evident a rise in the carotid pressure, as might be expected. Sackur[13] claims to have found a marked diminution in the amount of oxygen in the blood, in some instances to only one-half.

PART III.—EXPERIMENTAL EMPYEMA.

In order to test the applicability of these ideas of open pneumothorax to the surgical treatment of empyema a series of twenty dogs in which empyema had been produced was used. After several preliminary failures to produce an empyema by the intrapleural injection of from 10 to 15 c.c. of pure broth cultures of a virulent strain of hemolytic streptococci given to us by Major R. A. Kinsella,[14] a successful result was finally accomplished by injecting into the pleural cavity of a 6-kilo dog 30 c.c. of a broth culture of the same strain which was highly virulent for mice. The dog died in about twelve hours after the injection, and at autopsy the left pleural cavity (the injected side) was found to contain about 200 c.c. of slightly blood-stained, serofibrinous fluid which contained myriads of streptococci and a few necrotic leukocytes. This exudate resembled in every respect the exudate obtained from the early human cases of streptococcus empyema at Camp Lee. Subsequently this exudate was used successfully in doses of from 1 to 5 c.c. for the production of empyema in the series of twenty dogs. In ten of the twenty dogs intercostal pleural drainage with cocain (0.5 per cent.) anesthesia was established from four to twenty-four hours after the operation. To the other ten dogs which served as controls nothing was done. The operations were all carried out under strictly aseptic precautions and sterile dressings were later applied. A stiff rubber drainage tube with a one-quarter inch (6 mm.) lumen was used for each dog and the tube was stitched to the skin in order to hold it in place. The point selected for the drainage in each case was the sixth interspace in the anterior axillary line, since this seemed to be the most dependent portion of the thorax with the dog in the natural position. Sixteen of the dogs each received 2 c.c. of the exudate, two each received 3 c.c. and two each received 1 c.c. All the injections were made into the left pleural cavity. The dogs were paired as carefully as possible according to weights, so that each dog operated upon had a control which was not only of approximately the same weight but which also had been injected with the same amount of pleural exudate. In general, out of each pair, the dog which seemed the stronger and the better operative risk was selected for the oper-

[13] Weiteres z. Lehre von Pneumothorax, Arch. f. path. Anat. u. Physiol., 1897, cl, 151.

[14] The particular strain used was the one which had been obtained at autopsy from the only fatal case of our series at Camp Lee. The patient had not only an empyema but also suppurative pericarditis and multiple small lung abscesses.

ation in order to give the operation every possible chance, since many of the dogs were poorly nourished and were victims of mange. In spite of all this, however, of the ten operated dogs all but one died (a mortality of 90 per cent.), and of the unoperated dogs only seven died (a mortality of 70 per cent.). Of the series of unoperated dogs also one fatality was in a dog which on the fourth day, while apparently well, was badly injured in a fight with another dog and received extensive lacerations in the neck, the back and the hind legs. Death occurred two days later and at autopsy the pleural cavity contained no exudate and appeared normal. It seems probable that it would not have died if it had not been for the wounds received in the fight.

It was a striking fact that of every pair of dogs which died the one which had been drained died from one to two days before its control with two exceptions, in which both the operated and the control dogs died at about the same time. It never happened that the unoperated dog of a pair died before the operated one. The only dog of the operated series which survived pulled its dressings off and pulled out the tube on the next day after the operation. When seen shortly afterward (within three hours after the dressings had been pulled off) the pleural opening had already closed so that the dog no longer had an open pneumothorax. This was the only dog which disturbed his dressings. As a rule, death occurred in the operated dogs from forty-eight to seventy-two hours after the operation, and in the control series from the fourth to the fifth day. Of one pair, each of which had been injected with 2 c.c. of the pleural exudate, both the control and the operated dog died after about twenty-four hours following the injection. The operated dog in this case had been drained four hours after receiving the injection. The operated dogs, as a rule, not only died more quickly than their controls but they all seemed much sicker than the unoperated dogs from the time that the pleural opening was made. They would lie quietly curled up, refusing food and resenting disturbance. The unoperated dogs were much more active. Immediately after operation each dog was placed in a cage by himself, was furnished with an abundance of water and food and the cage was kept clean. In almost every instance the dressings were changed daily, dry sterile dressings being substituted for the soiled ones, with rigid aseptic precautions, even to the extent of our wearing sterile gowns and sterile rubber gloves during the procedure. Each of the operated dogs had a profuse drainage of a thin serofibrinous discharge, sometimes slightly blood-stained, which microscopically showed innumerable streptococci and only a few leukocytes which were usually necrotic.

Each dog that died was examined at autopsy within a few hours after death. All of the operated dogs had been drained well, as shown by the absence of appreciable amounts of exudate in the pleural cavity. In practically every instance there was an extensive deposit of fibrin on the pleura, and there were many recent adhesions.

In general there were more adhesions in the dogs that had been drained than in the controls. Two dogs showed in addition to a left pleuritis involvement of the pericardium and the other pleura also. It was interesting that the pericardial fluid contained a noticeably smaller amount of organisms than the left pleural cavity and that the right pleural exudate contained even fewer. In other words, it appeared as if the infection had passed right through the pericardium and mediastinum from the inoculated left side. Of these two dogs, one had been drained and the other had not been. Three of the dogs that had been operated upon showed at autopsy pockets of pus behind the upper part of the sternum which resembled very much those which had been described frequently in the human cases. This condition has never been observed in any of the dogs which were not drained.

The fact that in this series there was a slightly higher mortality in the dogs that had had early drainage than in a control series of the same number which received no treatment of any kind seems to indicate very strongly that under the conditions of the experiment, early drainage at least is of no benefit to the animal, and if anything it is rather a source of harm. Presumably the harm is produced by the open pneumothorax, with the train of resulting effects which have been discussed earlier in this article. Any comparison, furthermore, with the condition of streptococcus empyema in man must carry with it the important consideration that theoretically this experimental empyema in dogs does not begin to contra-indicate the early establishment of an open pneumothorax to the same extent as the human condition for the reason that the dogs do not have the associated pneumonia which seems to have been universally present in the human cases and which necessarily lowers the threshold of safety for the establishment of an open pneumothorax. It was hoped to test out early aspiration with later operation on another series, but time was not available for the experiment.

PART IV.—EXPERIMENTAL PNEUMONIA.

Attempts were made to produce in dogs the typical pneumonia and empyema of the human cases. It was felt that success in the experimental production of the condition would not only allow a better opportunity to determine by experiment the best treatment of the condition, but that also an opportunity would be presented of perhaps studying the pathology in all of its various stages with particular reference to the factors underlying the occurrence of empyema. Here, again, unfortunately, it has been impossible to carry this part of the work to a conclusion because of lack of time. We have, however, been able in three out of six dogs to reproduce a condition experimentally which seems to be in every way identical with the pathological picture of the human pneumonia and empyema which has been so well described by Dr. W. G. MacCallum. The

method used was tracheal insufflation of pleural exudate from other dogs into a dog deeply anesthetized with ether. The amount of pleural exudate used has varied from 2.5 to 5 cm. One of the dogs in which a successful result was obtained also was moderately ill with distemper at the time the insufflation was made. A brief summary of one of the experiments with a gross description of the viscera of the dog as noted at autopsy is as follows:

An 8.5-kilo dog on August 28 was given 2 c.c. of pleural exudate by method just described above. On August 31 the dog died. There was a very extensive pneumonia of both lower lobes with scattered areas of consolidation in the other lobes. Bilateral empyema (200 c.c. thin, slightly purulent fluid in left pleural cavity and 100 c.c. in right pleural cavity) was found, as well as suppurative pericarditis (about 50 c.c. of exudate in the pericardial sac similar to the pleural exudate) with extensive deposition of fibrin on the pericardium. Extensive fibrinous adhesions were present in both pleural cavities with massive fibrinous deposits on the posterior surfaces of both lower lobes. Each lower lobe also showed posteriorly a necrotic patch about 1 cm. in diameter which was found to allow air to escape when attempts were made to inflate the lungs, according to MacCallum's method. It seemed in fact that on the right side there was a pneumothorax in addition to the empyema, as air apparently escaped from that side when it was opened. We were not sure, however, of this point. The tracheobronchial glands were much enlarged and there was a moderate tracheitis. The other organs showed no important changes. The lungs were not cut but the thoracic viscera were placed *in toto* in Kaiserling solution. A description of the appearance of the cut surface of the lungs will be included in the report on the microscopic examination by Dr. MacCallum.[15]

[15] Incision into the several lobes on each side reveals areas of reddish-gray consolidation most extensive in the lower lobes. There is on each side in the central portion of the area in the lower lobe a ragged cavity filled with fluid and necrotic material and extending to the pleura, which is torn so that the abscess cavity appears in both cases to have communicated with the pleura. Other smaller areas appear opaque and necrotic, but are not liquefied. Microscopically it is found that the pleura is thickened and covered with a fibrinous exudate. No broad lymphatics are traceable into the substance of the lung. The bronchi are filled with leukocytes, among which are very numerous streptococci. Their walls are not markedly altered, although the epithelium is frequently desquamated. No conspicuous or thrombosed lymphatics are seen in their walls nor in the walls of the bloodvessels. The alveoli are filled with an exudate of polymorphonuclear leukocytes, with some red corpuscles and great numbers of streptococci. Their walls are not infiltrated or thickened. Great areas in the midst of such consolidated tissue have become necrotic *en masse* and are surrounded by a zone of closely packed leukocytes. These areas, rich in streptococci, are sometimes intact, but the larger ones show a complete disintegration of the structure of the necrotic lung and of the enclosed exudate, and the softened, partly liquefied debris is seen to have retracted from the living tissue, which remains as a densely infiltrated wall. The consolidation has little in common with the interstitial broncho-pneumonia observed in the recent epidemic, but is almost precisely like the condition also observed then which was referred to as lobular pneumonia. In that condition large numbers of streptococci invaded the lungs, causing a widespread intra-alveolar exudate, with hemorrhage and subsequent extensive necrosis of the infected tissue. The sections from the second dog repeat this picture so precisely that they need not be described.—W. G. M.

PART V.—GENERAL DISCUSSION.

Application of Experimental Results to Problem of Treatment of Empyema. In the light of all these observations it would seem definitely established that an operation for empyema performed too early in the course of the disease is accompanied by such very grave danger that in our opinion the risk of harm by the operation outweighs any advantages which it may have. These observations apply particularly to the type of empyema due to the hemolytic streptococcus prevalent in the epidemic last winter, but apparently they would apply equally well to any type of empyema. The principal determining factors to be considered in deciding whether the time is too early for operative interference are the relative immobility of the mediastinum, the presence of active severe pneumonia and the amount of asphyxia present. Practically it may be stated that, in general, the safest time to operate is when the exudate has become frank pus instead of being merely serofibrinous, for the following reasons: (1) There is less danger of creating an open pneumothorax because, in our experience, there is likely to be a circumscribed abscess shut off by adhesions from any communication with the free pleural cavity so that during the operation the pleural cavity, properly speaking, is not entered. (2) Even if an open pneumothorax is created the patient is in much better condition to withstand its harmful effects, because (*a*) the subsidence of the active pneumonia has the effect of making the area of the air-inlet to the lungs larger than when many of the bronchioles and much of the lung parenchyma are blocked by the pneumonic process, so that the pleural opening is incapable of producing the same amount of harm, (*b*) the presence of adhesions and the inflammatory thickening and induration of the mediastinum tend to make it less mobile, (*c*) the patient's need of oxygen is less because of a more nearly normal metabolism, (*d*) the respiratory compensation is more efficient since, owing to a diminished toxemia, the respiratory muscles will not become so easily fatigued. (3) The patient is in better condition to withstand whatever shock there is connected with even so slight an operation as pleural drainage. (4) There is probably less risk of creating a septicemia from absorption of organisms from the fresh operation wound, an occurrence which seemed to have happened a few times at Camp Lee by the finding of positive blood cultures a few hours after operation in cases which previously had shown negative blood cultures, the operation having been done very early in the disease.[16]

On the other hand the only advantage to be gained from an early operation is drainage which theoretically accomplishes the removal of both toxic material and living organisms and in addition relieves

[16] For our usual procedure in deciding when to operate upon cases of streptococcus empyema at Camp Lee, see "Preliminary Report of Empyema Commission," Review of War Medicine and Surgery, Office of the Surgeon-General, Washington, 1918, i, No. 6, also Jour. Am. Med. Assn., lxxi, 1918, 366 and 443.

mechanical embarrassment to respiration caused by the presence of a large amount of fluid. Practically, however, the mechanical embarrassment to respiration is almost sure to be aggravated instead of improved because of the creation of an open pneumothorax; and, moreover, the fluid can be withdrawn by aspiration as often as it accumulates in any considerable amount, a procedure which, of course, also removes some of the toxic material and living organisms. The apparently theoretically ideal method of continuous drainage under negative pressure involves the necessity of a special attendant to prevent a delirious patient from interfering with the apparatus and thus running the risk of creating an open pneumothorax. It should not be forgotten also that in many of these streptococcus cases operation will be unnecessary. Of our cases at Camp Lee, 13 per cent. recovered merely with aspiration.

If, in spite of consideration of all the relative dangers of an early operation, it should be decided nevertheless to establish drainage in a certain case, it seems apparent that only a small opening should be made during the stage of active pneumonia. Resection of ribs and the creation of large openings in the pleura should be reserved if necessary, until later, after adhesions and induration have changed the mediastinum into a relatively immobile partition.

In considering the mathematical expression given in a previous section it will be seen that any condition which increases the values of

$$\frac{R_1}{R_2}T$$

or which decreases the value of V or of aC will decrease the size of the maximum opening compatible with life. Thus any obstruction of the natural air passages, any interference with maximum contraction or expansion of the chest or any condition which increases the demand for air will tend to decrease the limit of safety in the size of the opening. In the early stages of a streptococcus pneumonia several such factors are present. There is great obstruction of the air passages, and the vital capacity is reduced by the consolidation of the lung and by the exhausted condition of the muscles of respiration. The amount of air required is greatly increased since the general metabolism is nearly doubled. All these factors tend to reduce the size of the thoracic opening for which compensation can be made by the patient. In fact, some cases have already so much interference with respiration that they are partially asphyxiated and a thoracic opening of any appreciable size must inevitably cause death.

On the other hand a patient who has recovered from the pneumonia and presents only an empyema is in a much more favorable condition to stand an open pneumothorax. The air requirement has fallen considerably, the obstruction of the air passages and the consolidation have disappeared, and the muscles of respiration have recovered from their exhaustion. The mediastinum has become thickened and stiffened by exudate and immobilized by adhesions so that it no longer has normal mobility, thus reducing greatly the effect of a

pneumothorax on the lung of the unoperated side. The presence of this condition may make the patient even a better operative risk than a normal man.

There is no Discrepancy between our Experimental Results and the Clinical Findings in War Wounds of the Thorax. During the past year the idea has become prevalent in this country that operations upon the thorax which permit of the free entrance and exit of air into the pleural cavity during the operation can be performed with about the same impunity as abdominal operations. In fact, the belief has arisen that surgeons at the front have found that alterations of the intrathoracic pressure by the admission of air through extensive incisions or large gaping wounds are in themselves not dangerous, and a feeling of optimism exists that now bold surgical intervention on the thorax may be carried out. It has been said consequently that the danger from creating an open pneumothorax in early operations for empyema with permanent drainage is negligible. These conceptions apparently have been largely based upon the address made a year ago at the Clinical Congress of Surgeons by Sir Berkeley Moynihan, a Colonel in the Royal Army Medical Corps. In the printed address Colonel Moynihan[17] bases his remarks chiefly upon articles by Duval, Depage, Gregoire, Elliott, etc. He says practically nothing about how extensive his own experience with wounds of the lungs and pleura has been, but he makes some remarkable statements which give the impression that the operator may attack the lungs boldly without heed to the danger of an open pneumothorax. For example, in discussing the open operation for extraction of foreign bodies he states that "no shock follows this operation," also, that during the operative incision of the pleura the free admission of air, "as a rule, causes no disturbance and does not alter the rate of the respirations or of the pulse."

On the other hand, reference to the work of Duval[18] and of others who have had extensive personal experience with this type of cases gives one a very different impression from that obtained from Moynihan's address, although at their hands also results were obtained which seem startling when compared with our ideas of the limitations of thoracic surgery before the present war. For instance, Duval's article, which is based on 3453 cases, shows that of all types of wounds of the chest the mortality is nearly twice as great in the gaping as in the closed wounds (27 and 15 per cent. respectively). The larger mortality of the gaping wounds is ascribed by Duval chiefly to the element of infection, but it seems highly probable that many of these may have died of the open pneumothorax, especially since, according to Duval, 50 per cent. of all lung wounds die within the first day, before the results of infection could have manifested themselves. Piéry states that the two gravest prognostic

[17] Gunshot Wounds of Lungs and Pleura, Surg., Gyneo. and Obst., 1917, xxv, 605.

[18] The original was inaccessible to us, but extensive abstracts of the work of Duval and others may be found in the Review of War Surgery and Medicine, Office of the Surgeon-General, Washington, 1918, i, No. 4, 1–27.

elements are the large, open chest wound and generalized infection. "In discussing operative indications, Piéry[19] sounds a moderate note of conservatism when he points out that he considers it unfortunate that the early optimistic reports regarding the safety of lung surgery must in a measure be modified—in other words, one must bear in mind that surgery of the lung, in spite of the remarkable recent advance, is still surgery of a very grave sort." Both Duval and Piéry consider that the gaping wound must be closed in order to overcome the resultant mechanical embarrassment of respiration and the resultant pleural infection. As a matter of fact, according to them, closure is to be considered as an emergency operation.

As regards the operative attack on the lung itself it is to be noted that most surgeons with wide experience in war wounds of the lungs adopt measures which of necessity tend to limit the actual area of the opening into the pleural cavity and also to immobilize the mediastinum somewhat. In other words, they follow either consciously or unconsciously methods which our experiments show to be effective in reducing the danger arising from an open pneumothorax. For example, as a rule, the lung is drawn out through the thoracic incision a procedure which not only diminishes, if it does not actually plug, the opening into the pleura, but also tends to immobilize the mediastinum. Moreover, generally, the thoracic opening is plugged by a thick gauze compress after the delivery of the lung in order to obviate the to-and-fro movements of air during the operation. "It[20] is necessary to operate as gently and quickly as possible and to get the opening in the chest wall closed at the earliest possible moment. In case there should be any evidence of mechanical interference with breathing it is advisable rapidly to deliver the lung, plugging the chest wall with gauze. This procedure is usually followed by cessation of all respiratory embarrassment." Duval says that the closure of the chest wall must be done very carefully in order to avoid the leakage of air; the resected rib ends should be covered with muscle in order to get an air-tight wound. "Although the operation is described as sometimes remarkably simple it is, nevertheless, exceptional for these patients to run other than a rather stormy postoperative course." Roberts and Craig,[21] in a report based on 199 cases state that they operated upon "open" cases so soon as the condition of the patient permitted and that before doing that the great majority of cases in this class never reached the base. Anderson[22] in an experience in a British casualty clearing station found that the closure of an aperture in the wall of the thorax more than compensates for any risk which is accepted, and he emphasizes that the important matter is to get the chest completely closed. Gask and Wilkinson[23] in a report based on 500 cases of this type, also at a British casualty clearing station, adopted

[19] Quoted from Review of War Surgery and Medicine, loc. cit, p. 10.
[20] Quoted from the combined abstract in the Review of War Surgery and Medicine, loc. cit., p. 14. [21] Ibid, p. 18. [22] Ibid., p. 20. [23] Ibid., p. 22.

the routine of always closing large openings into the pleura through which air was sucked by temporary skin suture without an anesthetic. This was found to give the patient immediate relief.

It is evident, therefore, that from the experience of many surgeons in a very large series of war wounds of the chest the dangers arising from an open pneumothorax cannot be disregarded. On the contrary it is not surprising that otherwise healthy men can stand during the period of an operation a relatively large opening in the thorax, for in a previous section of this article it is shown that on the basis of calculations based on our experiments a healthy adult, without pneumonia and with good respiratory muscles, should be able to stand for a short time without death an opening of about 51.5 sq. cm. (5 x 10 cm., or 2 x 4.1 inches). It should be emphasized that, on the basis of our calculations, this is the largest possible opening compatible with life in the average healthy adult with a normal mediastinum, and that life can be maintained with so large an opening for only a short time. It is larger, however, than the usual area of an opening into the chest when an operation is made on the lung, especially when the opening is partly closed by the presence in the incision of a hand, the lung, a gauze pad or a number of instruments. *There is, therefore, no discrepancy between the deductions drawn from our experiments and the clinical observations on war wounds of the chest.*

Mechanism of Reaction to Change of Intrathoracic Pressure not Clear. The almost instantaneous response in the character of the respiratory movements which follows the creation of an open pneumothorax cannot be satisfactorily explained at the present time. It is very unlikely that asphyxia can account for it because the reaction occurs before any appreciable effect of asphyxia on the respiratory center can manifest itself. Also, that it is not due to the stimulation of nerve endings by the admission of cold air into the pleural cavity is shown by the fact that on several days when experiments were performed the temperature of the room was between 95° and 100° F. No noticeable difference was detected at this temperature and at lower temperatures.

Summary and Conclusions. From the standpoint of pressure relationships the normal thorax may be regarded as one cavity instead of two. Any change of pressure in one pleural cavity is accompanied by practically an equal change in the other so that an equilibrium of pressure exists at all times throughout the whole thorax. In our experiments the changes of intrapleural pressure have been accomplished only with air, but probably the same conclusions would hold for those effected by fluid.

The prevalent conceptions of pneumothorax are erroneous in that they are based on the assumption that when an opening is made into the chest one lung is collapsed and the other maintains respiration. This assumption implies that the mediastinum constitutes a rigid partition between the two pleural cavities. On the

contrary the mediastinum is so mobile that any increase of pressure in one pleural cavity pushes it over into the opposite one so that both lungs are compressed practically equally. No such condition is possible, therefore, of collapse of one lung and maintenance of respiration with the other in a chest with a normal mediastinum.

If, on the other hand, the mediastinum has been made rigid by induration as a result of long-standing inflammation, or if it has become fixed by adhesions, then a pleural opening on one side will not produce the same pressure changes in both pleural cavities.

The maximum opening into a pleural cavity compatible with life depends upon a definite relationship which exists between the amount of air entering the lungs and the amount which enters the pleural opening. The maximum opening compatible with life may be approximately determined for the normal chest by the mathematical expression given in the text. By this mathematical expression it is found that a normal human adult should be capable of withstanding for a short time an opening of about 51.5 sq. cm. (5 x 10 cm., or 2 x 4.1 inches). There is no discrepancy, therefore, between our experimental results and the finding at the front that men are capable of maintaining respiration with gaping thoracic wounds, which seem surprisingly large.

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 (and therefore the amount of air admitted into the pleural cavities) is greater than a single opening on one side is likely to be. Theoretically and experimentally effects of practically the same severity result in the case of one or more openings into one pleural cavity as follow the creation of a double pneumothorax, provided that in each case the combined areas of the various openings are equal.

The bearings of these results and deductions upon both the treatment of acute empyema and upon thoracic surgery in general is obvious. Whenever the amount of air taken into the lungs is limited by the presence of an active pneumonia, with plugging of both air channels and alveoli, whenever there is an excessive demand for air, or whenever there is sufficient weakening of the respiratory muscles to impair compensation, the size of a pleural opening compatible with life becomes smaller; and if any or all of the above factors are present in sufficient intensity, even a very small opening into the pleural cavity will produce death from asphyxia. Since all of these factors are likely to be present to a high degree during the early stage of an empyema of the streptococcus type, early operation with the establishment of an open pneumothorax carries with it an unwarrantable danger. Aspiration is indicated instead until after the above dangerous factors have disappeared.

Special emphasis has been placed in this article on the changes of intrathoracic pressure induced by an open pneumothorax, but other results such as heat loss, danger of infection and disturbance of the systemic circulation are of great importance.

IRRITABLE HEART OR EFFORT SYNDROME.

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The medical work incidental to the selection of men for our new armies has brought new problems and novel experiences for all engaged in it, even for those who by reason of years of service in civil or military hospitals might justly consider themselves familiar with the various fields of clinical medicine. Especially is this true with relation to the physical signs and phenomena of heart affections. Not indeed that anything essentially new has been discovered, but rather that the methods employed, applied as they have been to many thousands of men called from civil life to the Colors, have brought new appreciation of the extent to which certain physical defects or disturbances prevail among our people and have compelled us to study these conditions from an entirely new view-point, that of the acceptability of the subject of such disabilities for military service. The problem has concerned prognosis rather than diagnosis, and its solution has been hedged about with anxiety, both for the welfare of the individual concerned and for the soundness and vigor of our armies.

To rightly value the facts set forth in this report certain considerations must be borne in mind. In the first place the men summoned for military service must have been subjected to most careful and repeated physical examinations. It is doubtless true that no army in the world has been put through so many tests to assure the physical soundness of its personnel. The men of the National Guard were not only examined by their regimental surgeons before being mustered into the Federal service, but were also gone over, man by man, by experts in the diagnosis of diseases of the heart and lungs. The drafted men were examined at the place of their enrolment and again at the camp to which they were assigned. In the case of any suspicion of disability they underwent a third examination at the hands of specialists in the particular field concerned. Finally, in all the camps during the past six or seven months, boards of specialists in tuberculosis have been engaged in going over the forces, man by man, for disease of the heart or lungs. In consequence of these methods it may safely be assumed that few indeed of the recruits presenting any abnormality of heart sound or action have escaped observation. As a corollary the numbers of those found or believed to be suffering from disease or disturbance of the heart has been impressively large.