THE EFFECT OF PULMONARY CONGESTION ON THE VENTILATION OF THE LUNGS.

By CECIL K. DRINKER, M.D., FRANCIS W. PEABODY, M.D., AND HERRMANN L. BLUMGART, M.D.

(*From the Laboratory of Applied Physiology of Harvard Medical School and the Medical Clinic of the Peter Bent Brigham Hospital, Boston.*)

PLATES 1 TO 3.

(Received for publication, June 21, 1921.)

Clinical observations have shown that in patients with heart disease the vital capacity of the lungs is frequently less than normal, and that the decrease in the vital capacity bears a close relation to the development of dyspnea. As the tendency to dyspnea increases the vital capacity usually falls, and in patients who are bedridden on account of shortness of breath the vital capacity is rarely more than 30 per cent of the normal. This lowering of the vital capacity expresses the impossibility of increasing the depth of respiration in a normal manner, and under the stress of exercise such subjects are unable to raise the volume of pulmonary ventilation so as to bring about the necessary gas exchange between lungs and blood. The determination of the vital capacity has, therefore, considerable practical significance, since it serves as a guide to the general condition of the patient and often indicates changes in the functional condition of the circulation and respiration that are in harmony with the symptoms but are not necessarily suggested by changes in the physical signs.

The cause of this decrease in the vital capacity of the lungs in heart disease has never been adequately explained. In advanced cases it is, of course, due in part to pulmonary edema, pleural effusion, hepatic enlargement, and similar obvious factors; but in many cases the vital capacity is decreased without any physical signs which account for it, or with physical signs which are certainly insufficient to explain the degree of decrease.

The suggestion has been made (1, 2) that in such cases there may be an increase of pressure in the pulmonary circulation with engorgement of the alveolar capillaries of the lungs. As the alveoli are extremely vascular such a condition might produce a stiffening or *Lungenstarrheit*, in the sense of von Basch (3), which would interfere with their easy expansion and collapse in respiration. There might also be some protrusion of the distended capillaries into the alveoli and thus a decrease in the volume of residual air of the lungs. Since it is impossible to measure pulmonary arterial pressure in human beings, no statements can be made as to the degree of pressure increase which may develop in cardiac disease. From our observations upon cats in which the pulmonary veins have been obstructed, it is apparent that a high degree of pulmonary stasis may be produced without causing much actual increase in pulmonary blood pressure. The lungs act as a slightly elastic sponge, and are able to take up a vast amount of blood without significant pressure change. Furthermore, the normal right ventricle is unable to sustain pressures of any magnitude. We have attempted to increase pressure in the pulmonary artery through a clamp placed upon this vessel just central to its division. It is impossible to adjust such a clamp so as to provide a sustained increase in pressure of more than a few millimeters of mercury in the right ventricle of the cat without causing the death of the animal. We may, therefore, consider that when blood accumulates in the pulmonary circuit with a normal heart there is little attendant pressure increase, and that accompanying changes in ventilation are incident upon volume rather than pressure changes in this circulation.

In chronic cardiac disease with hypertrophy of the right ventricle it is conceivable that pressure in the pulmonary capillaries may reach abnormal levels, but it should be remembered that at the time we are interested in the question—the moment when the vital capacity begins to diminish—the heart muscle is in all probability beginning to fail, and, as a consequence, we are inclined to believe that even in human beings with considerable cardiac hypertrophy the rise in pressure in the pulmonary circuit accompanying a much increased blood volume in this circuit is not great. We are at present engaged upon experiments in which the pressure changes in the pulmonary

artery and capillaries resulting from pulmonary vein compression are being followed, and the statements just made must, therefore, be taken as expressions of our opinion at the moment, not as facts of experimental observation.

Clinical experience contributes several facts which are in harmony with the theory that the interference with the ventilation of the lungs, which shows itself by a decrease in the vital capacity, is due to a chronically increased filling of the pulmonary veins and capillaries. Thus, mitral stenosis is characterized by an early onset of the tendency to dyspnea and an associated low vital capacity. In aortic insufficiency, on the other hand, there is little tendency to dyspnea, and the vital capacity remains high until a relative mitral insufficiency develops and the pulmonary circulation is affected. Again, pleural effusions and pulmonary edema with râles are accepted signs of cardiac weakness. These probably find their cause in congestion of the pulmonary capillaries, and it is logical to suppose that their appearance is preceded by a phase in which the pressure may be very slightly higher than normal, but not yet sufficient to bring about exudation into the alveoli with eventual production of râles or the passage of fluid into the pleural cavity. Before either of these processes takes place there are no physical signs to indicate definitely changes in the pulmonary blood circuit. According to the theory suggested, however, even at this stage the vascular engorgement might interfere with the movements of the lungs and with the size of the alveoli, and cause a decrease in the vital capacity. If this theory is true, its practical significance will be easily understood, for the determination of the vital capacity will give the earliest and most accurate information which can be obtained about the pulmonary circulation. The earliest symptom in most cases of heart disease is an increased tendency to dyspnea and throughout the course of the disease this tendency is usually one of the best guides to the functional efficiency of the heart. It seems quite possible that the development of dyspnea depends largely on the condition of the circulation in the lungs, and, if such be the fact, any method which will help to throw light on one of the most obscure portions of the circulation will be of great clinical importance.

The problem which presents itself, therefore, as the result of the foregoing clinical observations, is to determine whether blood stasis

in the pulmonary circulation produces any effect on the ventilation of the lungs. If such a change does produce a limitation or reduction of the ventilation, then the theory that the reduction of the vital capacity of the lungs in heart disease is due to an increased filling of the pulmonary circulation receives considerable support. It is obvious that the question cannot be decided by the study of patients; the animal experiments to be described in the present communication were, therefore, designed in order to elucidate it.

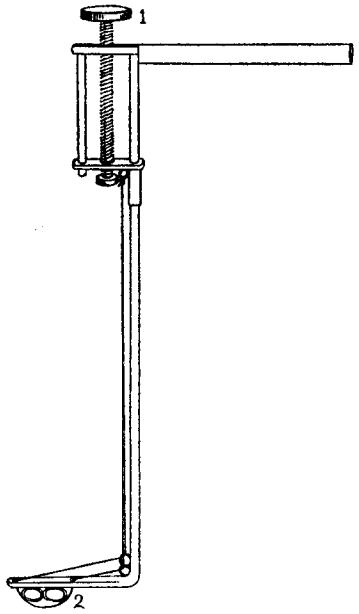
Technique.

The animals selected for study were cats. Because of the exquisite delicacy of the tissues to be studied and the relatively slight displacement in air space to be measured, certain experimental requirements were considered essential.

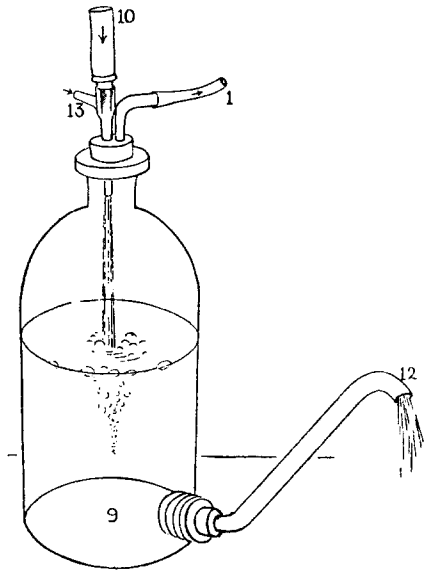
1. All spontaneous and reflex movements of the animal had to be eliminated. Even in the anesthetized animal compression of the pulmonary veins brings about active respiration, and such a reaction interferes with comparative measurements. Urethane anesthesia followed by curare obviates this difficulty.

2. A method had to be devised for compressing and releasing the pulmonary veins without any manipulation of the lungs. This was satisfactorily accomplished by the method described in detail previously (4). An oval section of the chest wall immediately over the heart is removed, the pericardium incised anteriorly, and the cut margins are reflected and sewed to the edges of the chest wall. With proper technique, the pleural cavity is rendered air-tight, permitting for hours independent respiration of the animal. With such a method the heart and great vessels lie exposed *in situ*, resting posteriorly on a pericardial sling. Advantage is then taken of the unusually high reflection of the pericardium in the cat, in which animal a ligature may be slipped about the pulmonary veins without in any way entering the mediastinum. The ligature is then threaded into the clamp as indicated in Text-fig. 1. The pulmonary veins, 2, represented in cross-section, can then be compressed and released by manipulation of thumb-screw 1. We have also found it possible to insert into the pulmonary artery a cannula of the type described by Schafer (5), in order to measure pressure in this artery during pulmonary vein compression.

All of these adjustments can be made after closing the thorax with the pericardium without the use of artificial respiration. In certain instances, of which Experiment 4 is an example, an intrathoracic cannula was inserted and connected either with a water manometer or with a delicately balanced volume recorder. Under such circumstances intrathoracic pressure or lung volume may be followed and the influence of blood in filling up thoracic space noted with accuracy.



TEXT-FIG. 1.



TEXT-FIG. 2.

TEXT-FIG. 1. Clamp for compression of pulmonary veins.
TEXT-FIG. 2. Apparatus for obtaining constant air pressure.

But in these instances the lungs are invariably collapsed away from the chest wall and are thus able to swell outwardly when they fill with blood. Such a condition is not permitted when the chest wall is properly closed and the lungs are well inflated against it.

3. The air delivered to the animal had to be at a relatively constant pressure and uniform rate of flow. This condition was satisfied by the apparatus illustrated in Text-fig. 2. The water flows down through tubing 10, under a constant head which is maintained by

a bottle suspended 9 feet above, kept full to overflowing. The water passes through an ordinary suction pump, draws in air through by-pass 13, and runs into an air-tight bottle, 9. Here air and water separate. Since the water level in the bottle is determined by the height of the top of the overflow tube, 12, the added amount of water passes out through this overflow tube, while the air is conducted out through tubing 1 to the animal.

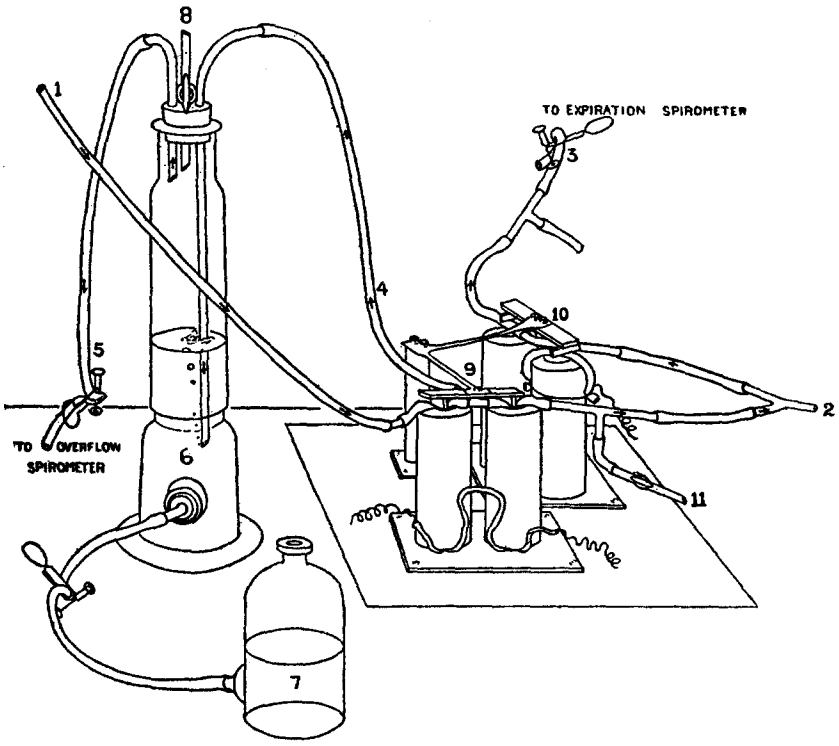
4. The apparatus had to be so designed as to register quantitatively any alteration in pulmonary ventilation. The artificial respiration machine indicated in Text-fig. 3 fulfills this need. It consists of two magnetic interrupters timed by a rotary switch to compress alternately the rubber tubing beneath them. The air passes to the animal from the bottle shown in Text-fig. 2 through an inspiratory tube, 1 (Text-fig. 3), to the cannula, 2, inserted into the trachea of the cat. During the period which constitutes inspiration, one hammer, 9, is raised, while a second hammer, 10, completely occludes the expiratory tube, 3. During expiration, the reverse situation exists, the expiratory air delivered through tube 3 being collected in a sensitive spirometer, known hereafter as the expiration spirometer.

Attention should be drawn to the shunt interposed between the two bars of hammer 9. This shunt, 4, by virtue of its position, operates only during inspiration and is designed to conduct all the air not accommodated by the cat's lungs through bottle 6 to another finely balanced spirometer, termed the overflow spirometer. If the available air space in the lungs is diminished, or the elasticity of the lungs becomes decreased so that they no longer expand as much under the same air pressure, more air will be collected in this overflow spirometer while less air will be collected in the expiration spirometer.

The construction and purpose of bottle 6 interposed in the overflow circuit deserve further comment. The air flows in through tubing 4, bubbles through the water in bottle 6, and escapes to the room through a stop-cock, 8, or, if this be closed and the clamp released, through tubing 5 to the overflow spirometer. Regardless of the avenue of escape of this air, however, the height of the water column in bottle 6, adjustable through container 7, determines the amount of air delivered to the animal. Thus, if the water column is high, little or no air will escape during inspiration into the overflow spirometer and

all the air will pass into the animal to be delivered to the expiration spirometer.

In brief, provision is made to collect all the air delivered to an anesthetized, curarized animal. This air reaches the animal rhythmically but under relatively constant conditions of pressure and flow.



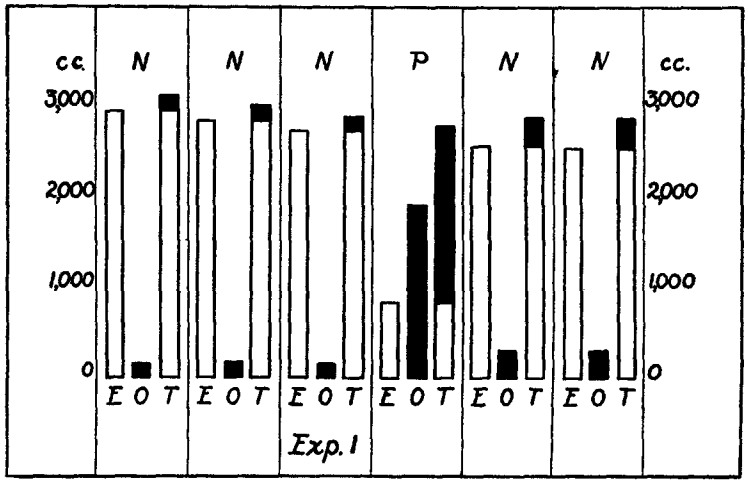
TEXT-FIG. 3. Magnetic interrupter for artificial respiration, with connections to spirometers.

It is collected in two spirometers and the total amount of air which these two receive will check closely in different periods of equal length. But if the pulmonary air space is decreased or if the movements of the lung tissue are restricted, the distribution of the collected air will at once change, a smaller amount being received by the expiration spirometer, and a larger amount by the overflow spirometer.

EXPERIMENTAL.

Experiment 1.—February 7, 1921. Cat; weight 2.8 kilos. 12.05 p.m. 28 cc. of 25 per cent urethane by stomach tube. 3.05 p.m. Operation on thorax completed. 3.30 p.m. 1 cc. of 2 per cent curare intravenously. Cardiometer and pulmonary vein clamp adjusted. Arrangements made for recording rate of respiration and femoral blood pressure.

The further course of this experiment is illustrated by Text-fig. 4 and Fig 1. Text-fig. 4 is a graphic representation of the six collection periods which comprised the experiment. There are, first, three normal periods, labeled *N*, in which air



TEXT-FIG. 4. Graphic representation of Experiment 1. Six 5 minute periods are shown. In this and the following text-figures *N* indicates normal period; *P*, period of pulmonary vein compression; *E*, air collected in expiration spirometer; *O*, air collected in overflow spirometer; *T*, total air collected by both spirometers.

delivered to the animal was collected in the expiration and overflow spirometers without any obstruction of the pulmonary circulation; then one period, labeled *P*, in which the pulmonary veins were compressed by means of the clamp shown in Text-fig. 1; and finally, two more periods, labeled *N*, prior to which the pulmonary vein clamp was released and the animal permitted to return to normal conditions. All periods in this and other experiments were 5 minutes in length, and in Text-figs. 4 to 7 the same method of representation is used. In every case the column marked *E* is the amount of air which actually entered the lungs of the cat and was collected in the expiration spirometer; the column marked *O* is the amount received in the overflow spirometer; and the column marked *T* is the

total amount of air delivered by the respiration apparatus during the 5 minute period, a total obtained by superposing the overflow column upon the expiration column.

Fig. 1 is a kymograph record of conditions during parts of the experiment. Section 2 covers the second period charted in Text-fig. 4. Tracing 1 is the cardiometer record made with the usual type of cardiometer transmitting through air to a large tambour. Tracing 2 is made by means of a second, rather stiff tambour and indicates the rate of respiration. A rubber tube (11, Text-fig. 3) on the inspiratory side of the respiration apparatus leads to this second tambour. Tracing 3 is the blood pressure record taken from the femoral artery by means of a mercury manometer. Tracing 4 is the base-line for blood pressure. Downward marks upon this line indicate 15 second intervals, but in this case are too irregular to be of value since the time-clock was out of order. Upward marks signal various events in the experiment.

During the first three normal periods, a constant amount of air was collected in the overflow spirometer but the total amount was slightly reduced. The change was, however, so much less than that occurring when pressure was applied to the pulmonary veins as to be negligible.

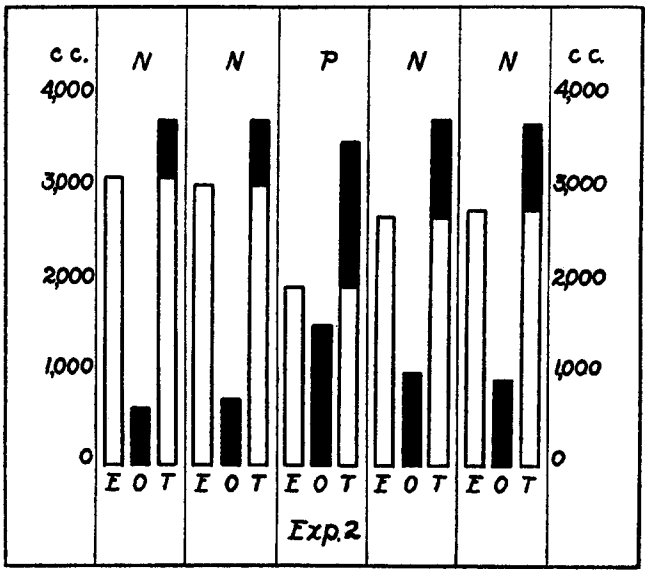
The first upward mark on the base-line in section 4, Fig. 1, indicates the beginning of pulmonary vein compression, brought about by tightening the clamp shown in Text-fig. 1. Between the second and third upward marks in this section of tracing, the air collection shown in *P*, Text-fig. 4, was made, and at the fourth upward mark the pulmonary vein pressure was released. The rise in the cardiometer record during this period shows a certain amount of cardiac dilatation; and the diminished movements of the writing point, a restriction of the individual beats. The fall in blood pressure indicates the degree of interference with filling of the left ventricle. The shift in distribution of the air during this period is clearly shown in *P*, Text-fig. 4. The great reduction in the amount of air collected in the expiration spirometer was due to pulmonary congestion incident upon the pulmonary venous obstruction, and the rise in the overflow collection merely accounts for the air which could not enter the lungs under the conditions imposed.

The two last normal periods *N* and *N*, Text-fig. 4, indicate an almost complete recovery after pulmonary vein release. The fact that this was not absolutely complete probably indicates a certain amount of intraalveolar extravasation which was not at once removed and which occupied space available for air in the first three normal periods. It is, however, clear that a very large measure of recovery did take place, and this means that the changed distribution of air noted in period *P*, Text-fig. 4, was due to intravascular blood.

The lungs, when removed at the close of this experiment, showed narrow longitudinal areas of congestion along the most posterior and consequently dependent portions of each lobe. The degree of change was comparable to that shown in Fig. 5, taken, it is true, from another experiment, but fairly indicative of the pathological changes occurring in experiments accepted by us as picturing the true relation of vascular filling and ventilation. On microscopic examina-

tion one finds epithelial desquamation with red cells and fluid in the alveoli making up the narrow area commented upon in the gross description of the lungs, and normal appearances in the rest of the tissue.

Experiment 2.—February 19, 1921. Cat; weight 3 kilos. 30 cc. of 25 per cent urethane. Operation and preparations for recording similar to those in previous experiment. Text-fig. 5 shows two normal periods, then a period of pulmonary vein compression, and finally, two more normal periods. The result is identical with that noted in Experiment 1. Fig. 2 is a graphic record similar to that in Fig. 1. Section 1 corresponds with the first normal period. Since pulmonary vein compression was produced rather slowly, section 3 has been



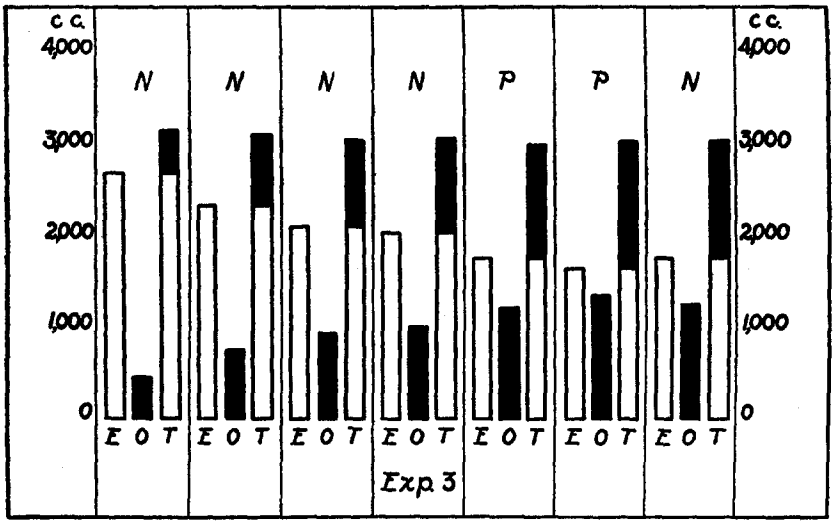
TEXT-FIG. 5. Graphic representation of Experiment 2. Five 5 minute periods are shown.

shortened to save space. The first upward mark on the base-line indicates the beginning of compression. Between the second and third, the air collection noted in *P*, Text-fig. 5, was made, and at the fourth mark the veins were released. Section 4 corresponds with the first normal period *N* after *P* in Text-fig. 5, and displays the condition of the circulation at that time.

On examination of the lungs an area of dark red congestion—not hard nor raised—was noted along the posterior surface of the right lung, and a few similar very small congested areas, chiefly along the margins of the lungs. It is interesting to note that the amount of overflow air in the last collection was somewhat less than in that just preceding, indicating a gradual tendency to return to normal.

In this case, the pulmonary vein clamp was tightened at 12.47 p.m., and collection period *P* took place between 12.56^{30} and 1.01^{30} p.m. The first normal collection period after clamping fell between 1.11^{10} and 1.16^{10} p.m. and the last one between 1.23 and 1.28 p.m.

Experiment 3.—February 19, 1921. Cat; weight 2.9 kilos. 29 cc. of 25 per cent urethane by stomach tube. A technique exactly similar to that of the two preceding experiments was used in this case except that two periods of pulmonary vein compression (*P, P,* Text-fig. 6) were employed. The result shows the effect of a progressively weakening heart and the production of extensive pulmonary edema. The heart was unusually large at the beginning of the experiment and



TEXT-FIG. 6. Graphic representation of Experiment 3. Seven 5 minute periods are shown, two of them in this instance being periods of vein compression.

there was more pericardial fluid than usual. Four normal periods were run and the volumes of air collected in the overflow spirometer showed an increase from 473 in the first period to 1,023 cc. in the fourth period. This increase was probably due to the development of pulmonary edema. In the third and fourth normal periods the volume in the overflow spirometer became more constant than it was in the first two, so that light compression of the pulmonary veins was tried in the 5th period.

The condition of the animal at this time is shown in section 5, Fig. 3. Poor adjustment of the cardiometer with leakage explains the atypical cardiometer record. Light compression caused a characteristic effect as noted in the first *P*, Text-fig. 6, and was followed in 9 minutes by a period of greater compression,

the second *P* in Text-fig. 6, and section 6 in Fig. 3. A greater degree of the typical change occurred, and that this was due in part to vascular engorgement, and not wholly to the progressive development of pulmonary edema, is indicated by the fact that release of the vein compression resulted in the last normal period (Text-fig. 6) in a slight return towards normal conditions. Section 7 in Fig. 3 indicates the condition of the animal at this time—a condition of progressively dilating heart and falling arterial pressure, the full extent of which was obscured by a clot which occurred near the end of the tracing.

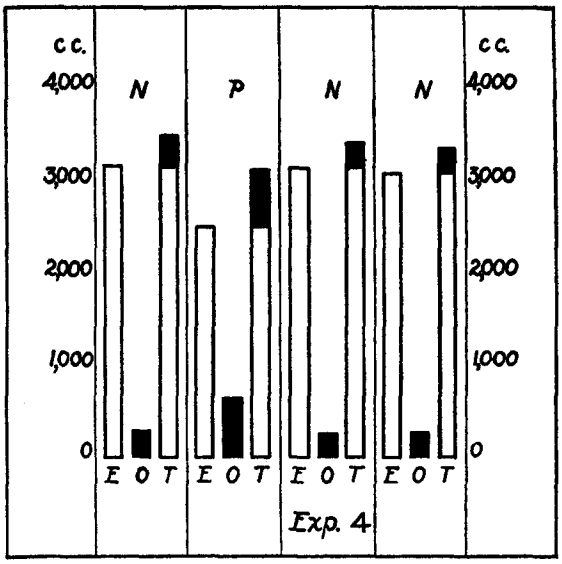
The lungs at the termination of this experiment were in bad condition, over two-thirds of the posterior surface being congested and dark red. There was, thus, extensive permanent damage. The experiment is published in order to display the course of events when pulmonary air space is steadily encroached upon by something more than intravascular blood. Such an animal is perhaps in a similar condition to that of the patient with reduced vital capacity and clear-cut physical signs in the lungs.

Experiment 4.—May 2, 1921. Cat; weight 2.7 kilos. 27 cc. of 25 per cent urethane by stomach tube. All preparations were exactly the same as in preceding experiments with one exception. The line of tracing numbered 2 in Fig. 4 indicates in this instance, intrathoracic volume changes. With the preparation completed in other respects, a glass tube was thrust through the chest wall and tied in place by a purse-string suture. Lung collapse at once occurred, probably bilateral. The intrathoracic cannula was connected with a delicate volume recorder constructed to write on a kymograph. On calibration it was found that a movement of the writing lever of 0.30 cm. recorded a volume change of 1 cm.

Text-fig. 7 is a chart of this experiment and shows a smaller but characteristic result during the period of compression, due probably to the fact that in this case the lungs were free to expand within the thorax, having been collapsed away from the chest wall, whereas in former instances practically all space occupied by blood was alveolar.

Section 1, Fig. 4, shows the condition of the animal during the first normal collection indicated in Text-fig. 7. Section 2 of the same figure is the period of pulmonary vein compression. At the first upward mark on the base-line, tightening of the pulmonary vein clamp was begun. Between the second and third upward marks the air collection period *P* of Text-fig. 7 took place, and at the fourth upward mark the pulmonary vein compression was released. The cardiometer and blood pressure effects are as in former experiments. The tracing of intrathoracic volume rises promptly, indicating a swelling of the lungs with blood, and the individual excursions of the recorder become less owing to less extensive movements of the lungs with each respiration. The maximum rise is equivalent to an encroachment on the intrathoracic space of 6.6 cc. and the individual excursions are diminished by an amount equivalent to 1 cc. Section 4, Fig. 4, represents the conditions during the final normal collection period of Text-fig. 7.

Fig. 5 is a drawing of the posterior aspect of the lungs from this animal. The broken lines lead to darkened sections which indicate the extent of a slight area of congestion—an area not great enough, however, to cause any effect upon ventilation, since it is noticeable that the overflow collections in the last two periods of Text-fig. 7 are not greater than that obtained in the first period of the experiment.



TEXT-FIG. 7. Graphic representation of Experiment 4. Four 5 minute periods are shown.

DISCUSSION.

As indicated at the outset, these experiments were designed to determine whether pulmonary congestion interferes with the entrance of air into the lungs. Congestion was produced by compression of the pulmonary veins at their entrance into the left auricle, and the effect on the air entering the lungs was determined by means of a sensitive artificial respiration apparatus which delivered a constant volume of air with each inspiration. Any obstruction to the entrance of air into the lungs of the animal was indicated by a smaller volume of air being collected on expiration and a larger volume being collected in a spirometer connected with the inspiratory tube, arranged to receive the "overflow" which did not enter the lungs.

The experiments fall into two groups—those in which there was essentially no permanent change in the lungs as a result of the manipulations, and those in which extensive pulmonary congestion with edema into the air passages was produced. In the former, illustrated by Experiments 1, 2, and 4, constriction of the pulmonary veins and the consequent overfilling of the pulmonary circulation caused definite interference with the entrance of air into the lungs. In the different experiments the quantitative effect naturally varied within wide limits. That the effect on pulmonary ventilation was the result of vascular change alone is indicated by the fact that release of pressure on the veins resulted in a practically complete return to the previous normal conditions. The normal appearance of the lungs at the end of the experiments is also evidence that the whole effect was a vascular one, which corrected itself as soon as the obstruction to the circulation was removed.

In the second group of experiments, typified by Experiment 3, compression of the pulmonary veins produced not only the simple vascular effect which appeared in the first experiments, but also caused extensive pulmonary congestion with edema into the air passages. Marked permanent damage was done to the lungs. In these experiments constriction of the pulmonary veins produced an interference with the entrance of air into the lungs, as it did in the first group of experiments, but release of pressure was not followed by a return to normal conditions. The changes in the lungs resulting from the passage of fluid out of the vessels into the pulmonary tissues and the air spaces were not relieved when the pressure in the vessels was allowed to return to normal. In these experiments the blood pressure in the greater circulation fell progressively throughout the observation, and it seems probable that the more extensive and permanent damage to the lungs was in part due to the development of cardiac weakness. This was especially evident in Experiment 3, in which the extensive exudation was undoubtedly due to the combination of circulatory obstruction and weak heart muscle. The results obtained in this second group of experiments are in no way opposed to those obtained in the first group. On the contrary, they supplement them. The two sets of experiments show that if the pulmonary veins are obstructed to such a degree that congestion of the pulmonary

vessels without exudation is produced, there results interference with the entrance of air into the lungs, which is relieved as soon as the obstruction is removed, but that if the circulatory conditions are such that exudation of fluid out of the vessels into tissues and air passages is produced, a permanent interference with the entrance of air into the lungs results.

There appear to be two ways in which pulmonary obstruction can act in order to encroach upon air space in the lung. First, the dilatation of the capillaries may actually take up alveolar space which air could occupy under normal alveolar conditions. This is a simple explanation of the observed change and readily accounts for the fact that less air entered the lungs on each inspiration. When the chest is closed and the lungs expanded so as to fill all the available space. it is clear that extra room which excess blood may occupy can only be obtained at the expense of the alveolar air space. The size of the lungs can only be changed, under these conditions, by pressures great enough to move the chest wall and diaphragm, and it is doubtful whether the right ventricle is capable of accomplishing such a result. When the chest is closed the lungs swell inwardly and large encroachment upon ventilation is the result. This change is illustrated by Experiments 1 and 2. When, however, the chest is open, blood may cause the lungs to swell outward, and under such circumstances the alveolar space may be very little changed and the amount of air entering the lungs not much altered. Experiment 4 illustrates just such a result and brings us to the second method by which pulmonary congestion may reduce the air space. It can be seen that in this experiment the expansibility of the lungs was diminished. The effect is, perhaps, as though the lung were erectile tissue rendered rigid and inelastic through vascular turgescence. Von·Basch (3) suggested such an effect many years ago, and Experiment 4 is direct evidence of its existence but under abnormal conditions, the chest in this case being open. We are unable to say in what proportion these two factors operate to reduce ventilation in the closed chest, but are of the opinion that the first is the more important. It is possible that experiments now in progress, in which changes in capillary pressure are being measured, may throw light upon the situation.

We may now consider whether the facts derived from our experiments on animals afford any explanation of the conditions observed in patients with heart disease—conditions which were described at the beginning of this paper. In brief, it has been found clinically that patients with heart disease who become short of breath on exertion are unable to increase the depth of respiration, and that the vital capacity of the lungs is low. Experimentally it has been demonstrated that abnormal amounts of blood in the pulmonary circulation interfere with the entrance of air into the lungs. The clinical condition which has the closest analogy to our experiments is mitral stenosis. They are, indeed, essentially similar, for in mitral stenosis there is an obstruction to the flow of blood in leaving the left auricle, while in our experiments an obstruction was produced to entrance of blood into the left auricle. Both conditions, the clinical and the experimental, produce the same changes in the hemodynamics of the circulation in the lungs. In the early stages of mitral stenosis, when the obstruction to blood flow is slight, there is no abnormal tendency to dyspnea and no decrease in the vital capacity of the lungs. As the degree of stenosis increases, however, a tendency to dyspnea on exertion develops, and the vital capacity of the lungs is less than normal. Physical examination of the lungs reveals nothing to account for this interference with deep respiration. There is no exudation of fluid into the pleural cavities or into the air spaces, and thus no dullness on percussion, no alteration in breath sounds, and no râles. The conditions resemble those in Experiments 1, 2, and 4, in which there was engorgement of the pulmonary vessels with little or no exudation.

The inability to breathe deeply and the low vital capacity, which is one of the outstanding features of mitral stenosis at this stage, may well be explained by engorgement of the pulmonary vessels and lung rigidity, just as these factors explained the interference with the entrance of air into the lungs in our experiments. At a later stage of mitral stenosis, physical examination reveals râles in the air passages and fluid in the pleural cavities. These indicate actual exudation of fluid out of the vessels, and the conditions are similar to those typified by Experiment 3, in which extensive areas of pulmonary congestion were found. In the clinical disease, as well as in the experiments, the development of actual exudation depends on two factors,

the degree of obstruction to blood flow and the weakening of the heart muscle. At this period in clinical cases a still greater fall in vital capacity is found, and a greater tendency to dyspnea. The subject is unable to increase the depth of respiration enough to secure the degree of pulmonary ventilation necessary for aeration of the blood when the needs of the organism for oxygen are increased.

The relation of the conditions in the pulmonary circulation to the production of dyspnea is also suggested by other clinical observations. Mitral stenosis is the disease in which the most immediate effect on the pulmonary circulation would be expected, and dyspnea is an early symptom in mitral stenosis. Mitral insufficiency alone may not produce dyspnea for a long period of time, or apparently not until the heart muscle begins to weaken. The same is true of aortic insufficiency alone, but in association with a mitral lesion and involvement of the pulmonary circulation dyspnea begins to appear.

In aortic stenosis the situation is analogous to mitral stenosis, and when the pressure conditions are transmitted back to the left auricle and the pulmonary circulation, dyspnea becomes a prominent symptom. In all these diseases involving the heart valves the conditions are complicated by a coincident gradual degeneration or weakening of the heart muscle. No completely satisfactory analogy to this exists in our experiment for we were always dealing with a healthy myocardium. It was quite apparent, however, that the weakening of the heart in Experiment 3 acted in the same way as constriction of the pulmonary veins and served to increase the effects due to changes in the pulmonary circulation. It is not at all improbable that weakening of the myocardium alone may be enough to produce the same effects, and there is clinical evidence in harmony with this. Thus, in cases of myocardial weakness in later life the development of dyspnea on exertion is often the first symptom. This is usually accompanied by a fall in vital capacity, but at first there are no physical changes of importance in the lungs. Later, râles and pleural effusion appear. Dyspnea commonly develops early in such cases, while edema of the legs and hepatic enlargement come later. This suggests that in many cases of myocardial weakness the failure of the circulation takes place first in the pulmonary circuit. Clinical experience, as well as the experiments which have been described, indicates that in the

first stages there may be interference with the entrance of air into the lungs without the production of physical signs of lung involvement. At a later stage pulmonary edema and pleural effusion will take place.

SUMMARY.

1. A method is described for producing pulmonary congestion, together with what may be termed a differential spirometer method for studying lung ventilation.

2. The method utilized permits an approximately accurate prediction of degrees of pulmonary edema in the living animal, and suggests avenues of approach for the very difficult problems of pulmonary capillary pressure.

3. It is shown that intravascular blood can encroach markedly upon the pulmonary air space. Although the methods used in these animal experiments do not resemble vital capacity measurements in man, their result is so definite that their applicability to clinical conditions may be considered.

4. The similarity between the experiments described and certain conditions of cardiac decompensation, of which mitral stenosis is the best example, is pointed out.

BIBLIOGRAPHY.

1. Siebeck, R., *Deutsch. Arch. klin. Med.*, 1910, c, 204.
2. Peabody, F. W., *Am. J. Med. Sc.*, 1918, clv, 100.
3. von Basch, S., *Verhandl. deutsch. Cong. inn. Med.*, 1889, viii, 384.
4. Drinker, C. K., *J. Exp. Med.*, 1921, xxxiii, 675.
5. Schafer, E. S., *Quart. J. Exp. Physiol.*, 1918–20, xii, 133.

EXPLANATION OF PLATES.

PLATE 1.

FIG. 1. Kymographic record covering collection periods 2, 4, and 5 of Experiment 1. Upper line of tracing, marked 1, is the cardiometer record; tracing 2, the rate of artificial respiration; tracing 3, arterial pressure, mercury manometer, femoral artery; tracing 4, the base-line for blood pressure. Upward marks on this line signal events referred to in text; downward marks, 15 second intervals—inaccurate in this instance owing to defective clock. The tracing is reduced to about $\frac{1}{4}$ (it is actually $\frac{5}{27}\frac{4}{3}$) of the original size.

FIG. 2. Kymographic record covering collection periods 1, 3, and 4 of Experiment 2. Lines of tracing similar to those in Fig. 1. The tracing is reduced to about $\frac{1}{4}$ (it is actually $\frac{64}{278}$) of the original size.

PLATE 2.

FIG. 3. Kymographic record covering collection periods 4, 5, 6, and 7 of Experiment 3. Lines of tracing similar to those in Figs. 1 and 2. The tracing is reduced to $\frac{64}{278}$ of the original size.

FIG. 4. Kymographic record covering collection periods 1, 2, and 4 of Experiment 4. Lines of tracing similar to those in Figs. 1 to 3, except that line 2 records intrathoracic volume changes and time marker records 5 second intervals. The tracing is reduced to about $\frac{1}{4}$ (actually it is $\frac{64}{278}$) of the original size.

PLATE 3.

FIG. 5. The lungs of the animal used in Experiment 4. Dotted lines lead to area of congestion described in text.

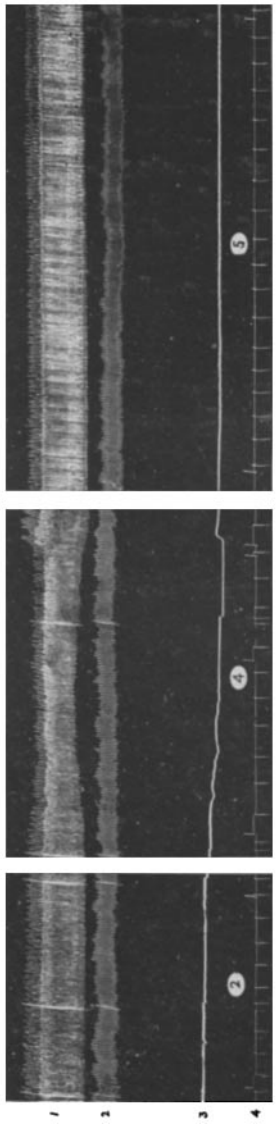


FIG. 1.

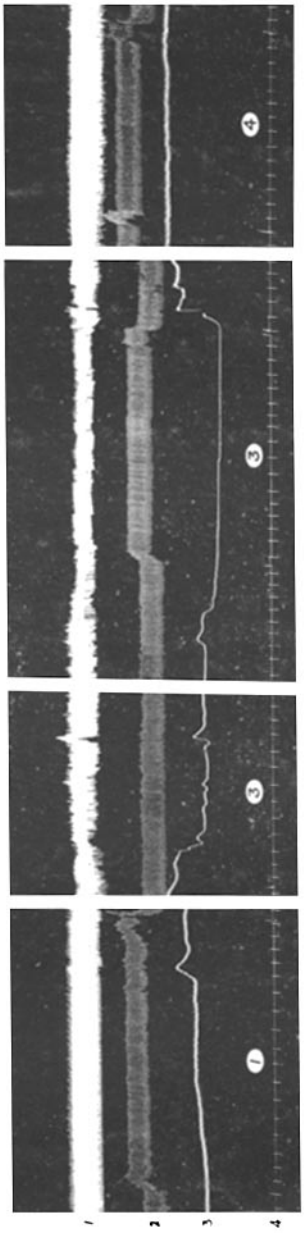


FIG. 2.

(Drinker, Peabody, and Blumgart: Pulmonary congestion.)

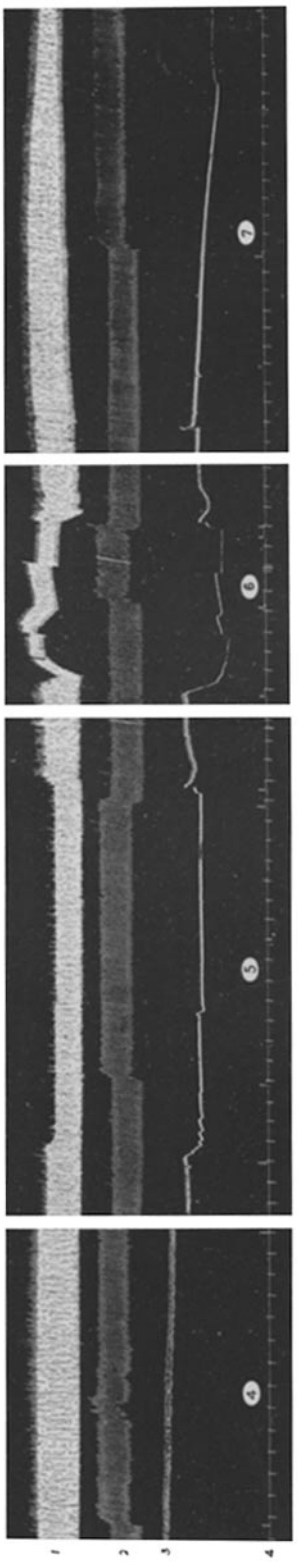


FIG. 3.

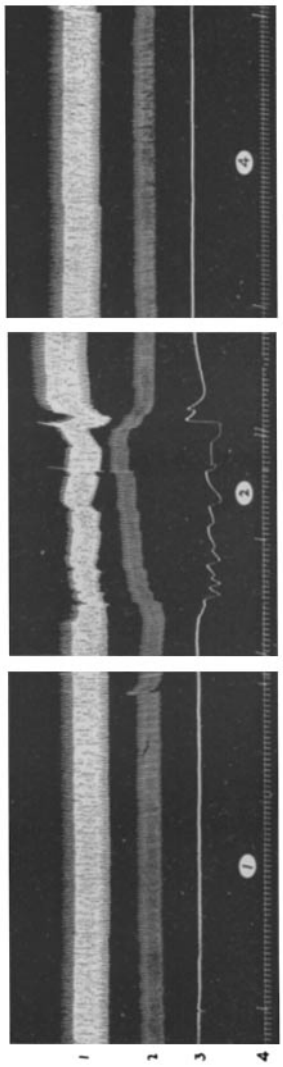


FIG. 4.

(Drinker, Peabody, and Blumgart: Pulmonary congestion.)

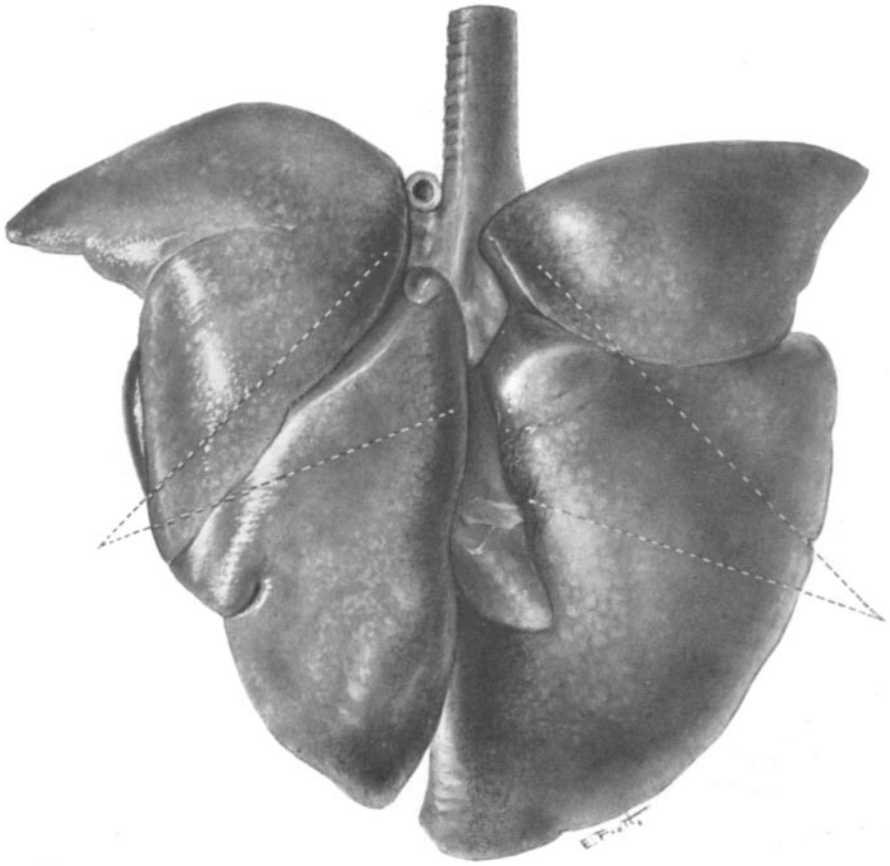


FIG. 5.

(Drinker, Peabody, and Blumgart: Pulmonary congestion.)