Effect of Elevated Left Atrial Pressure and Decreased Plasma Protein Concentration on the Development of Pulmonary Edema

By Arthur C. Guyton, M.D., and Arthur W. Lindsey, M.D.

With the technical assistance of Johnnie O. Howell, John W. Williams and Malcolm A. Franklin

In 97 dogs left atrial pressure was elevated to various levels up to 50 mm. Hg by partial constriction of the aorta. The effect of these pressures for from 30 min. to 3 hours on the accumulation of lung edema was then studied. Edema was estimated by determining the ratio of the weight of the wet lung to the weight of the same lung after drying. In animals with normal plasma protein concentrations fluid began to transude into the lungs when the left atrial pressure rose above an average of 24 mm. Hg. In another series of animals the plasma protein concentrations were reduced by plasmapheresis at the beginning of each experiment until the plasma protein concentration averaged 47 per cent of the control value. In these animals fluid began to transude into the lungs when the left atrial pressure rose above a critical value of 11 mm. Hg. Furthermore, the rate at which fluid accumulated in the lungs, in all series of experiments, was directly proportional to the rise in left atrial pressure above the critical pressure at which fluid began to collect in the lungs.

In the past few years many different investigators have stressed the importance of even short periods of elevated pulmonary vascular pressure in the development of acute pulmonary edema. For instance, Gorlin and co-workers have demonstrated in patients with mitral valvular disease that left atrial pressures in the range of 10 to 20 mm. Hg are compatible with dry lungs, but that even a few moments of exercise can cause temporary elevation of the left atrial pressure to 35 mm. Hg or more and result in clinical signs of edema. Haddy has demonstrated in experimental animals that left atrial pressures below 20 mm. Hg are almost invariably accompanied by dry lungs, but left atrial pressures only slightly above this value cause death within a few hours. Even types of pulmonary edema not formerly believed caused by elevated pulmonary capillary pressures have more recently been ascribed to this cause. Many examples of this, including especially "neurogenic" pulmonary edema, have been reviewed in detail in a very thorough summary of the whole subject by Visscher, Haddy, and Stephens.

Despite this recent emphasis on pulmonary capillary pressure in relation to pulmonary edema, precise studies describing the dynamics of fluid exchange at the capillary membrane of the lungs have not been reported. For this reason we undertook the present project, in which the left atrial pressures of dogs were held at elevated but constant levels for long periods of time. In each dog the degree of fluid accumulation in the pulmonary tissues was assessed as accurately as possible. In an additional group of dogs the plasma protein concentration was decreased to approximately one half normal at the beginning of each experiment and the same studies with elevated left atrial pressure were repeated. Thus, in these two groups of experiments the effects of both pressure and plasma protein concentration on the dynamics of fluid exchange have been studied.
METHODS

Ninety-seven mongrel dogs anesthetized with 30 mg./Kg. sodium pentobarbital were used. The left chest was opened between the third and fourth ribs and a specially made adjustable clamp was placed around the ascending aorta. A cannula was tied into the apex of the left atrial appendage, and, in 15 of the experiments, a catheter was threaded down the right external jugular vein, through the right ventricle and into the pulmonary artery. The chest was closed leaving the stem of the adjustable clamp protruding to the outside. The lungs were reinflated to their full extent and a constant suction was maintained within the thoracic cage to remove any fluid that might have accumulated during the experiment.

Left atrial pulmonary arterial and systemic arterial pressures were recorded by means of mercury manometers. At the outset of each experiment the adjustable clamp was tightened on the aorta until the left ventricle began to fail, thus causing an elevation in left atrial pressure. The clamp was further adjusted until the left atrial pressure reached the desired level. During the course of the experiment the clamp was continually readjusted to maintain the appropriate pressure. When the left atrial pressure was elevated for more than a few moments above 20 to 25 mm. Hg, fluid was lost rapidly into the lungs. To offset deterioration of the circulation, a mixture of equal parts of heparinized blood and Tyrode's solution was infused as needed (up to 1 L.) through a catheter in a femoral vein.

At the end of an experiment the animal was sacrificed by rapid hemorrhage and the chest was immediately opened to remove the lungs. The vessels of the lungs were drained of their blood and, to assess the amount of edema in the lungs, three different procedures were carried out: First, the lungs were weighed and the ratio of lung weight to body weight was calculated. Second, the lungs were weighed, then dried to constant weight and the ratio of wet to dry lung weight was calculated. Third, prior to drying the lungs the trachea was connected to an apparatus for determining lung compliance. It will be evident later that the second of these procedures—the wet to dry weight ratio—proved to be by far the best measure of the degree of pulmonary edema. Therefore it was used almost exclusively in the later experiments.

To study the effect of diminished plasma proteins on the development of pulmonary edema, approximately one third the blood, calculated to be 5 per cent of body weight, was removed, centrifuged and the red cells reinfused suspended in Tyrode's solution. This procedure was repeated twice in each dog. Analysis of the plasma proteins showed that the protein content of the blood was reduced to an average of 47 per cent of the control value.

RESULTS

Effect of Elevated Pressure for Thirty Minutes

In each of 19 dogs the left atrial pressure was adjusted to a given value somewhere between 0 and 45 mm. Hg, which was maintained for a period of 30 min. At the end of this time the dog was sacrificed and the degree of edema in the lungs was assessed by the three methods outlined.

Lung Weight to Body Weight Ratio. Figure 1 illustrates the effect of a 30 min. exposure to various left atrial pressures, up to 45 mm. Hg, on the lung weight to body weight ratio. This illustrates a general increase in the ratio as the left atrial pressure increased. It also illustrates that the increasing ratio can be represented best by two different regression lines, one for the points below 25 mm. Hg and a second for the points above 25 mm. Hg. These data are therefore consistent with the idea that a rise in pressure above a critical value of 25 mm. Hg causes fluid to transude into the lungs and that the rate of transudation rises in proportion to the further increase in pressure above this level. In the pressure range below 25 mm. Hg there was no statistical proof from this study that any fluid at all transuded into the lungs.

Ratio of Wet Weight to Dry Weight. The results in figure 2 show much more vividly than those of figure 1 that the rate of fluid accumulation increases markedly as the left atrial pressure rises above the critical value of approximately 25 mm. Hg. Indeed, at 45 mm. Hg pressure, only 20 mm. Hg above the critical value, the wet to dry weight ratio was approximately double the control value, thus illustrating the rapidity with which fluid can enter the lungs at these high pressures.

Another fact illustrated in figures 1 and 2 is the far greater consistency of results when the data are analyzed on the basis of wet to
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dry weight ratio rather than on the basis of lung to body weight ratio. This was also evident in all the other groups of animals studied. In 27 different experiments the left atrial pressure was less than the calculated value of the colloid osmotic pressure of the plasma. In all of these the wet to dry weight ratio of the lungs averaged 5.4 ± 0.3. This finding illustrates how completely reproducible the control values throughout the entire series of experiments when this particular method was used to assess the degree of edema. (The importance of this ratio is emphasized because we have not found this method of analysis used previously to evaluate pulmonary edema. A number of investigators, however, have used the lung to body weight ratio, and Visscher, Haddy, and Stephens, in their extensive review on the mechanisms of pulmonary edema concluded that the lung to body weight ratio is the most valid procedure available. Perhaps one of the reasons why wet to dry weight ratio has proved to be a better measure of pulmonary edema than lung to body weight ratio is that the wet to dry weight ratio of blood itself, as shown by analyses during the course of these experiments, has almost exactly the same value as that of normal lungs themselves. Therefore, blood inadvertently retained in the vessels of the lungs does not greatly alter the wet to dry weight ratio, although it does alter the lung to body weight ratio extensively.)

Compliance Curves as a Measure of Pulmonary Edema. In the lungs from all 19 dogs in which the left atrial pressure had been held to various pressure levels for 30 minutes, compliance curves were measured by a standardized procedure. Proportional volumes of air in relation to the weight of the lungs were injected and then withdrawn at standard rates. Despite all possible precautions for standardizing the procedure, only general changes could be noted between lungs with and without pulmonary edema (fig. 3). The solid curve in figure 3 is the compliance loop of a normal lung which had

![Compliance Curves](image)

**Fig. 1** Top. Fluid in the lungs of 19 dogs at termination of one half hour experiments in which the left atrial pressure had been elevated to various values. The amount of fluid is expressed as the ratio of the wet weight of the lungs to the body weight.

**Fig. 2** Middle. Fluid of the lungs of the same animals as in figure 1 except that the data are expressed as the ratio of the wet weight to dry weight of the lungs. Note the small deviation of the points from the mean in this graph.

**Fig. 3** Bottom. Typical compliance loops measured in lungs removed respectively from 2 dogs, 1 with normal left atrial pressure and 1 exposed for 30 min. to 45 mm, Hg left atrial pressure and showing extensive edema.
not been subjected to elevated left atrial pressure; the *dashed curve* was obtained from an edematous lung that had been maintained at 45 mm Hg left atrial pressure for 30 min. It is evident that the intrapulmonary pressures required for given volume changes were usually considerably greater in the case of the edematous lung than the nonedematous lung. However, the curves illustrated are extreme values and intermediate curves overlapped these in both directions so that no valid assessment of the degree of edema could be attained by the compliance method, but only a general indication that edema increases the pressures needed to expand the lungs.

**Effect of Prolonged Elevated Left Atrial Pressure on the Development of Pulmonary Edema**

**Time of Survival.** In a series of 25 different dogs the effect of elevated left atrial pressure lasting up to 3 hours was studied. Figure 4 illustrates the survival times. All dogs with left atrial pressures of 24 mm Hg or less survived the entire experiment and were apparently still in good condition when sacrificed at the end of 3 hours. One animal with a left atrial pressure of 25 mm Hg survived the full 3 hours and 1 survived 2.3 hours. All animals with left atrial pressures above 25 mm Hg failed to survive the full 3 hours. The period of survival was inversely related to the degree of elevation of the left atrial pressure above the critical values of 25 mm Hg (fig. 4).

**Degree of Fluid Retention During Prolonged Elevation of Left Atrial Pressures.** Figure 5 illustrates the wet to dry weight ratios of the lungs after termination of the experiments. In all animals with pressures up to 25 mm Hg, the experiments lasted a total of 3 hours so that a long time had been available for fluid to transude into the tissues of the lungs. In all experiments with left atrial pressures above 25 mm Hg, the animals died prior to 3 hours with correspondingly less opportunity for fluid to transude into the lungs. Nevertheless, despite the difference in time available for fluid transfer, it is evident from this study that in the lower pressure ranges, particularly 21 mm Hg and less, there is no evidence at all of fluid transudation into the tissues, whereas above 21 mm Hg, and particularly above 25 mm Hg, all of the lungs proved to be pathologically edematous. These results again show that there is a critical pressure point in the range of 20 to 25 mm Hg above which fluid leaves the capillaries to enter the lung tissues and below which the lungs are maintained in a persistently dry state.

**Degree of Pulmonary Edema at Death.** In figure 5 all dogs represented by *black dots* were alive at the end of 3 hours, while all dogs represented by *crosses* had died during the 3-hour period. If one draws a regression line (*upper line*) through only those points represented by *crosses*, the line obtained is almost parallel to the Y-axis. This indicates that the average degree of edema in the lungs at the time of death was essentially the same regardless of the actual left atrial pressure. In other words, those animals with left atrial pressures in the range of 45 to 50 mm Hg lived only for a very short time, but the time factor was balanced by a high rate of fluid loss into the tissues. Conversely, those animals with left atrial pressures of only 25 to 30 mm Hg lived a long time, but again the time factor was balanced by a correspondingly slow rate of transudation of fluid into the lungs. From these data it would seem that the cause of death is principally the degree of edema itself and not the length of time required to develop the edema.

**Rate of Edema Formation.** The rate of pulmonary edema formation in the dogs with prolonged elevation of the left atrial pressure was calculated as follows: The wet weight of the average normal lung was subtracted from that of the edematous lung of comparable dry weight. This value was then divided by the number of hours that the animal had survived. The results are plotted as *crosses* in figure 6. They indicate that below approximately 23 mm Hg there was no net transfer.
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of fluid into the lungs, but above this level the rate of fluid flow into the lungs was approximately proportional to the additional rise in left atrial pressure. For each additional millimeter of mercury rise in pressure, the mean increase in rate of fluid transudation (dR/dP) was 0.21 Gm. of fluid/Gm. dry weight of lung tissue/hour.

Effect of Diminished Plasma Proteins on Development of Pulmonary Edema

In 53 dogs the plasma protein concentrations were decreased by plasmapheresis to an average of 47 per cent of the control value. The animals were then exposed to 30 min. of different elevations of left atrial pressure. The results from 33 of these dogs are illustrated in figure 7. Almost identical results were obtained in the other 20 dogs, but these were analyzed separately because the method of drying the lungs was slightly different.

In figure 7 the two best fitting regression lines intersect at 11 mm. Hg. Here again there is a very critical pressure level above which fluid transudes into the tissues. But the critical point is now at a pressure level less than half that observed for animals with normal plasma proteins. Unfortunately, however, the scatter of the points on this graph is so great that its significance is not as satisfactory as would have been desired.

Rate of Edema Formation in Dogs with Lowered Plasma Colloid Osmotic Pressure.

Figure 7 is based on exposure of the dogs to elevated left atrial pressure for 30 min. By doubling the slope of the second half of the curve one obtains the rate of edema fluid collection per hour. When this is done it is found that dP/dR is 0.22 Gm. of fluid/Gm. dry weight of the lung tissue/hour. This value is almost identical with the 0.21 obtained above in animals with normal plasma proteins subjected to prolonged elevated left atrial pressures. Therefore, it appears that the rate of fluid transudation is not a function of the left atrial pressure itself, but rather a function of the left atrial pressure minus the colloid osmotic pressure of the blood.

FIG. 4 Top. Survival times of 25 dogs exposed to elevated left atrial pressures of different values. Arrows, dogs that lived the full duration of the 3-hour experiment; dots without arrows, dogs that died before completion of the experiment.

FIG. 5 Middle. Effect on the fluids of the lungs of prolonged exposure to elevated left atrial pressures. X, animals that succumbed before completion of the 3-hour experiment. Upper curve, regression line for the animals that succumbed, illustrating that the degree of edema in all animals that succumbed averaged almost the same amount at the time of death, regardless of left atrial pressure.

FIG. 6 Bottom. Rate of edema formation in dogs with normal plasma proteins and exposed to prolonged elevation of the left atrial pressure.
DISCUSSION

To study the physical principles of fluid exchange at the pulmonary capillary membrane one ideally should measure the pulmonary capillary pressure itself. Since this has never been accomplished satisfactorily we have chosen in this study to measure the left atrial pressure which, according to the best available estimates, is not more than one or two millimeters of mercury different from the pulmonary capillary pressure itself. This close correspondence between left atrial pressure and pulmonary capillary pressure has been adequately discussed many times in the past, particularly in a very complete review of the subject by Cournand.4 In essence, then, if one remembers that the pulmonary capillary pressure is usually only about one millimeter greater than left atrial pressure, one can think of the data presented in this paper as expressing the relationship of pulmonary capillary pressure to fluid exchange in the lungs.

In the two separate experimental groups of this study, the plasma proteins of the dogs were normal and the critical left atrial pressures above which fluid began to transude into the lungs were 25 and 23 mm. Hg respectively. If another millimeter of mercury should be added to each of these figures and the result considered to be the critical pulmonary capillary pressures at which fluid entered the lung tissues, the two values would be respectively 26 and 24 mm. Hg, or an average of 25 mm. Hg. This value is well within the range of the plasma colloid osmotic pressure expected for dogs with normal plasma proteins.5 Thus the usual principles ascribed to the law of the capillaries probably hold equally as well for the pulmonary circulation as for the systemic circulation.

In the 53 animals whose plasma proteins had been decreased to slightly less than one half normal, the critical left atrial pressure at which fluid began to transude into the lungs was 11 mm. Hg. If we should add 1 mm. Hg to this and assume the result to be the pulmonary capillary pressure, the critical value would now be 12 mm. Hg, which is approximately equal to the colloid osmotic pressure one would expect in dogs with half normal concentration of plasma proteins. This indicates that diminution of the colloid osmotic pressure of the blood causes a direct and equal decrease in the critical capillary pressure at which fluid begins to escape into the lungs.

Another interesting observation in these studies was that once the critical pressure had been exceeded, the mean rate of fluid transudation into the lungs of animals with normal plasma proteins (0.21 Gm./mm. Hg/hr./Gm. of lung) was essentially the same as that in animals with diminished plasma proteins (0.22 Gm./mm. Hg/hr./Gm. of lung). To state these results another way, the rate of fluid transudation in an animal with a plasma colloid osmotic pressure of approximately 12 mm. Hg and a pulmonary capillary pressure of 17 mm. Hg seems to be essentially the same as that in another animal having a plasma colloid osmotic pressure of 25 mm. Hg and a pulmonary capillary pressure of 30 mm. Hg. That is, the rate of fluid transfer is approximately proportional to pulmonary capillary pressure minus plasma colloid osmotic pressure.
The significance of tissue pressure and tissue colloid osmotic pressure in the dynamics of pulmonary capillary fluid exchange is not yet known. Unfortunately, neither the tissue pressure nor the tissue colloid osmotic pressure has ever been measured in lungs. However, the highly distensible nature of the interstitial spaces would indicate that the lung tissue pressure could hardly rise to more than a few millimeters of mercury. On the other hand, the tissue colloid osmotic pressure could easily become extremely high should the lymphatics become occluded or should capillary permeability become greatly enhanced. Yet in the present experiments the changes which occurred in fluid exchange could be explained by the balance between capillary pressure and plasma colloid osmotic pressure of the plasma without invoking either tissue pressure or tissue colloid osmotic pressure. This indicates that the tissue pressure and the tissue colloid osmotic pressure in these experiments were not greatly different from each other. And, if we assume that the pulmonary tissue pressure was only about 1 mm. Hg, we must also assume that the tissue colloid pressure was likewise in this low range.

The ability of the lungs to maintain the alveoli in a “dry” state has often been explained by the large differential between plasma colloid osmotic pressure and mean capillary hydrostatic pressure. A generally accepted value for plasma colloid osmotic pressure in the human being is about 30 mm. Hg and for pulmonary capillary pressure about 5 mm. Hg. If one also assumes that the pulmonary tissue pressure and pulmonary tissue colloid osmotic pressure are equal to each other, a net diffusion pressure of approximately 25 mm. Hg would exist at all times, tending to pull fluid out of the alveoli and tissue spaces into the circulation. For this reason any fluid that might exude into the alveoli, or enter them in any other way, would immediately come under the influence of this diffusion force so that the alveoli would soon be dry once more. In fact, many observers have already noted the rapidity with which fluid is absorbed from the normal lung.

Yet, despite the fact that the alveolus is normally filled with air, its walls are still coated with fluid. One questions why this remaining portion of fluid is not also absorbed into the capillaries as a consequence of the extensive differential between plasma colloid osmotic pressure and mean capillary pressure. This remaining film of fluid can undoubtedly be explained on the basis of the well-known phenomenon of surface tension, for, as the last remnants of fluid begin to be removed from the alveoli, intermolecular attraction along the surface of the alveolar wall begins to exert an opposing force. If we assume the pores to be cylindrical and 60 Å in diameter, as calculated by Pappenheimer, and also the fluid lining the alveoli to be pure water, the force that would have to be overcome before the surface film could be broken would be $1.5 \times 10^8$ dynes/cm$^2$ or 116,000 mm. Hg. Obviously, this is many times the 25 mm. Hg absorption pressure and, even allowing for tremendous error in the calculation or changes in the fluid composition, the force still should be far more than adequate to maintain a wet surface in the alveoli.

But why does surface tension not keep the alveoli filled completely with fluid rather than simply maintaining only a surface film over the alveolar walls? To answer this, one must remember that surface tension depends on cohesion of molecules only at the surface of the fluid. Consequently, any molecules that lie beneath the surface are subject to usual tissue fluid forces and can be absorbed into the capillaries, but as soon as all of the fluid beneath the surface has been absorbed so that only the surface layer itself remains, the forces caused by surface tension then oppose further absorption.

Presumably the lymphatics could act as a safety valve for removal of fluid from the lungs even after the pulmonary capillary pressure rises above the plasma colloid osmotic pressure. However, in the present experiments no animals with left atrial pressures
above 25 mm. Hg lived a full 3 hours. This indicates that the lymphatic safety factor is very minor indeed and that the lymphatics, rather than acting as a safety valve for fluid removal, have essentially the same function in fluid dynamics as elsewhere in the body—removal of proteins and thereby maintenance of the tissue colloid osmotic pressures near to zero.

In view of the extremely rapid appearance of pulmonary edema in certain clinical conditions, one would suspect that even a few millimeter's rise of left atrial pressure above plasma colloid osmotic pressure would cause almost instantaneous pulmonary edema. In the present experiments it was found that this was not true. Indeed, animals with elevation of left atrial pressures to approximately 5 mm. Hg above the calculated plasma colloid osmotic pressure lived an average of 2.5 hours before dying of pulmonary edema. To explain the development of pulmonary edema within a matter of minutes during certain hemodynamic abnormalities it is necessary to propose levels of left atrial pressure that are extremely high. In the present studies levels as high as 50 mm. Hg usually caused sufficient pulmonary edema to kill the animals in approximately 30 min. Pressure levels as high as 75 mm. Hg in the left atrium have actually been measured in experimental animals under certain acute hemodynamic conditions. If the rates of transudation of fluids that occurred in the present experiments should also hold when the pressure rises as high as 75 mm. Hg, one would expect the lung edema to reach lethal levels in an average of 12 min. Clinical signs of pulmonary edema should be evident within 5 to 7 min. Even without invoking any special mechanisms for acute pulmonary edema, the data of these studies, coupled with those obtained by others, indicate that acute pulmonary edema can certainly result from abnormal hemodynamic factors alone.

This study has demonstrated rather precise applicability of the well-known laws of capillary fluid exchange to the pulmonary circulation and that the major difference between the systemic circulation and the pulmonary circulation lies in the mean capillary pressure. In the lungs this pressure is low enough to keep the alveoli dry. In the systemic capillaries it is so high that the tissues remain wet.

**Summary**

The effect of prolonged elevation of left atrial pressure on the development of pulmonary edema in dogs has been studied under several conditions. In one series the left atrial pressure was elevated for 30 min. to values between 0 and 45 mm. Hg. By studying lung weight to body weight ratios and wet lung weight to dry weight ratios it was shown that atrial pressures below 24 mm. Hg did not cause significant transudation of fluid from the pulmonary capillaries, but left atrial pressures above this value caused fluid transudation at rates directly in proportion to the additional rise in pressure.

In similar studies in another series the left atrial pressure was maintained at elevated levels for as long as 3 hours. No animals died of pulmonary edema as long as the left atrial pressure was 24 mm. Hg or less. All animals with left atrial pressures of 26 mm. Hg or above failed to survive the full 3 hours of elevated pressure.

In an additional series of animals the plasma protein concentration was first decreased to about one half normal and the left atrial pressure was then elevated for prolonged periods of time. In this series no edema appeared when the left atrial pressure was less than 12 mm. Hg, but edema appeared in the other animals in proportion to the additional rise in pressure above the 12 mm. mark.

In the dogs with normal plasma proteins and with diminished plasma proteins the rates of fluid transudation into the lungs were respectively 0.21 and 0.22 Gm./mm. Hg/Gm. dry weight of lung tissue, in which mm. Hg is expressed as the actual left atrial pressure minus the critical pressure point at which edema began to appear.
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SUMARIO IN INTERLINGUA

Le effecto de un prolongate elevation del tension sinistro-atrial super le disveloppamento de edema pulmonar in canes esseva studiate sub varie conditiones. In un serie le tension sinistro-atrial esseva elevate durante 30 minutas a magnitudes de inter 0 e 45 mm de Hg. Per studiar le proportion inter le pesos de pulmon e de corpore e inter le pesos de pulmon humid e sic, il esseva possibile demonstrar que tensiones atrial de infra 24 mm de Hg non causa un grado significative de transsudation de liquido ab le capillares pulmonar, durante que tensiones atrial de plus que 24 mm de Hg causava un transsudation de liquido con intensitates directemente proportional al augmento additional del tension.

In simile studios in un altere serie, le tension sinistro-atrial esseva mantenite durante periodos de usque a tres horas. Nulle del animales moriva in consequentia de edema pulmonar, providite que le tension sinistro-atrial esseva 24 mm de Hg o minus, sed omne le animales con tensiones sinistro-atrial de 26 mm de Hg o plus moriva ante le fin del complete periodo de tres horas de tension elevate.

In ancora un altere serie de animales, le concentration del proteina in le sero esseva redueite a circa un medietate le valor normal, e posteas le pression sinistro-atrial esseva elevate durante prolongate periodos de tempore. In iste serie nulle edema apareva quando le pression sinistro-atrial esseva minus que 12 mm de Hg, sed edema esseva presente in animales con plus alte pressiones e isto in proportion con le augmento del pression supra le marca de 12 mm.

In le canes con normal proteinas del plasma e con reduceite proteinas del plasma, le transsudation de liquido a in le pulmones amontava, respectivamente, a 0,21 e 0,22 gramma per mm de Hg per gramma de peso sic de histo, con mm de Hg representante le total pression sinistro-atrial minus le pression critie al qual le edema comenciava monstrar se.

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