Pericardial and Ventricular Pressure

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In an earlier study of right ventricular pressure and volume, in animals subjected to plethora and hemorrhage, it was found that as right ventricular end-systolic volume (ESV) increased a small amount, from the lowest value consistent with life, end-systolic pressure increased greatly; further increase in ESV was associated with a smaller increase in end-systolic pressure until a plateau level of pressure was reached. If the plethora was large the end-systolic pressure was frequently elevated well above the plateau level of systolic pressure for a short time. It was felt that following plethora the distended atria and ventricles may have distended the relatively inelastic pericardium to the point where it would stretch no further. As a result, the pressure within the pericardial space may have increased and contributed to the high end-systolic pressure. Since the heart is contained within the relatively non-distensible pericardium, ventricular end-systolic pressure should be the algebraic sum of the pressure within the pericardial space and the pressure developed by the ventricular muscle during systolic contraction. In order to learn to what extent intrapericardial pressure contributed to ventricular pressure, it was decided to carry out the present series of experiments in which the pressure within the pericardial space and ventricular pressures were measured simultaneously.

Methods

In 17 anesthetized (subcutaneous injection of morphine sulfate 3 mg./Kg.; intravenous injection of dial urethane solution* 0.125 ml./Kg.; and pentobarbital sodium 7.5 mg./Kg.) dogs (6 open-chest and 11 closed-chest) catheters were passed into the right atrium and ventricle and pulmonary artery by way of the external jugular vein, and into the left ventricle and aorta by way of a common carotid artery, and intrapleural pressure was measured by cannulating the pleural space. With the animal under artificial respiration, a small opening was made in the left chest wall, and in some cases in both the right and left chest, generally between the fourth and fifth ribs. The pericardial space was either cannulated with a small open-end polyethylene tube, or a 1 cm. slit was made in the pericardium and a thin-walled rubber balloon passed into the pericardial space until it lay on the surface of the right or left ventricle or atrium. Preliminary experiments in several animals with a cylindrical balloon (4.2 cm. long x 0.7 cm. diameter) demonstrated that this type of balloon tended to exaggerate intrapericardial pressure as compared with an open-end tube. As a result, the experiments were carried out using a flat balloon (3.5 cm. X 4.5 cm. X 0.2 cm.) in 5 animals.

In 9 animals a puncture was made in the pericardium by means of a sharpened stylette (diameter of 1.7 mm.) and an appropriate size polyethylene tube with or without a rubber sleeve, which fit water-tight in the hole in the pericardium, was passed into the pericardial space. Following the cannulation of the pericardium the chest was closed, artificial respiration discontinued, and the animal allowed to breathe naturally; this was done in 4 dogs in which a balloon was used, and in 3 dogs in which an open-end tube was used. In 5 animals, in which the open-end tube was used and in 1 animal in which both an open-end tube and a balloon was used, the chest was left open throughout the experiment.

The pericardial balloons were filled with 0.9 per cent NaCl solution and the open-end tube with 0.9 per cent NaCl solution containing 5 per cent dextran. In the open-tube experiments, 5 to 10 ml. of this solution were injected into the pericardial space. Pressures were measured with Statham strain gages, Models P23D (right and

*Kindly supplied by Ciba Pharmaceutical Products, Inc.
Graph for converting pericardial pressures measured with a flat balloon to pericardial pressures measured with an open tube. See text for discussion.

Correction Factor for Pressures Measured with Balloons

It was found that the flat balloon always gave a pressure a few mm. Hg higher than the pressure within the bladder, and the cylindrical balloon gave a still higher pressure. A similar experiment was carried out in a living open-chest dog subjected to plethora and hemorrhage in which a series of pericardial pressures were measured with an open-end tube and right ventricular end-diastolic pressure was measured. Following these measurements, the above mentioned cylindrical balloon and flat balloon were placed in the pericardial space and the experiment repeated, measuring pressure simultaneously from both balloons and from the right ventricle, in the animal subjected to plethora and hemorrhage. Thus, we determined in the same animal the relationship between right ventricular end-diastolic pressure and pericardial pressure measured with an open-end tube, the same relationship when pericardial pressure was measured with a flat balloon, and the same relationship when pericardial pressure was measured with a cylindrical balloon. It was found that pericardial pressure measured with an open-end tube was only slightly less than right ventricular end-diastolic pressure, and that pressure measured with the flat balloon was a little greater than the pressure measured with the open-end tube, and that the pressure measured with cylindrical balloon was greater than the pressure measured with the flat balloon. In figure 1, values of pericardial pressure measured with the open-end tube are plotted against values of pericardial pressure measured with the flat balloon for various right ventricular end-diastolic pressures. Thus, any pericardial pressure measured with the flat balloon can be corrected to the true pericardial pressure (measured with an open-end tube) by reference to figure 1. In all of our experiments the pressures measured with the flat balloon were corrected in this manner. A similar larger correction can be made for pressure measured with a cylindrical balloon.

It would appear that the reason the pressure recorded by means of a balloon in the pericardial space is greater than the true pericardial pressure is because the surface area of a cylindrical balloon is small relative to the cross section of the balloon, while in a flat balloon the surface area is large relative to the cross section. As a result, when such balloons are placed between the surface of the ventricle and the pericardium, the force exerted by a relatively larger surface area of the ventricle is applied to unit surface area of the cylindrical balloon than is applied to unit surface area of the flat balloon. As a result a higher pressure is developed in the cylindrical balloon than in the flat balloon. Thus, the flat balloon records a pressure that is only slightly higher than that in the pericardial space (fig. 1).
while the cylindrical balloon records a higher pressure which under certain circumstances may be higher than the pressure in the ventricle.

**Results**

The results of a typical experiment measuring intrapericardial pressure with a flat balloon in a closed-chest dog are shown in figure 2. It will be noted that following plethora right and left ventricular end-diastolic pressures increased greatly, and that pericardial pressure increased nearly the same amount; during systole the pericardial pressure was markedly elevated as was the right ventricular pressure, but there was little or no change in intrapleural pressure. From the beginning of systole to the end of systole the pericardial pressure decreased an amount equal to approximately 32 per cent of its value during diastole.

**Pericardium Relatively Nondistensible**

That the pericardium is relatively nondistensible is shown in figure 3 in which immediately after death the pericardium of a dog was cannulated, fluid injected slowly into the pericardial space and the pressure measured. In this experiment the heart was first drained of blood and the pulmonary artery, aorta and veins entering the right and left atria were tied. After obtaining the pressure-volume curve of the pericardium, the heart was removed from the pericardium and its volume determined. The volume of the heart was added to the volume of fluid injected into the pericardial space and in this way the pressure-volume curve of the total volume of the pericardial contents was obtained as shown in figure 3. It will be seen that as the volume of the pericardium increases from zero there is little or no pressure within the pericardial space; after the volume reaches a large value, further small increase in volume causes a nearly perpendicular rise in pericardial pressure. These results are in general agreement with those reported earlier by Barnard and Isaacs et al.\(^8\)

**Relationship Between Intrapericardial and Right Ventricular Pressure at the End of Diastole**

The results of an experiment showing this relationship in a closed-chest dog are shown in figure 4A. It will be noted that as the right ventricular end-diastolic pressure rises, the pericardial pressure rises nearly the same amount. Results similar to this were obtained in all open-chest and closed-chest animals studied both with open-end tubes and balloons in the pericardial space. In figure 4B are shown similar results obtained in experiments on 9 dogs. The linear relationship obtained by the method of least squares has the equation:

\[
P = 0.85V - 0.4
\]

which shows that on the average with zero pericardial pressure \((P)\) the right ventricular end-diastolic pressure \((V)\) is about 0.5 mm Hg, and a given rise in ventricular end-diastolic pressure is on the average associated with a rise in pericardial pressure equal to 85 per cent of the ventricular pressure increase. Since the "effective" or transmural pressure distending the right ventricle is the difference between the pressure inside the ventricle and the pericardial pressure, it is clear that the transmural pressure is very small, even though the right ventricular end-diastolic pressure may be very large; for example, when right ventricular end-diastolic pressure is 25 mm Hg, the right ventricular transmural pressure is only 4 mm Hg.

**Relationship Between Intrapericardial and Left Ventricular Pressure at the End of Diastole**

The results of an experiment showing this relationship in a closed-chest dog are shown in figure 4C, and the results obtained in experiments on 9 dogs in figure 4D. Results similar to these were obtained in all animals studied. The linear relationship shown is similar to that for the right ventricle, except that for any given ventricular end-diastolic pressure the transmural pressure is slightly greater for the left ventricle than for the right. The equation describing this relationship (fig. 4D) is:

\[
P = 0.78V - 0.8
\]

which shows that on the average with zero pericardial pressure \((P)\) left ventricular end-diastolic pressure \((V)\) is about 1 mm Hg and a given rise in left ventricular end-diastolic pressure is associated with a rise in pericar-
Effect of plethora on the pressure in the pericardial space, right ventricle, right atrium, left ventricle, and intrapleural space in a closed-chest dog. Pericardial pressure was measured with a flat balloon, three-fourths of which lay on the surface of the right ventricle and one-fourth on the right atrium. Between the arrows 1,000 ml of 5 per cent dextran in 0.9 per cent NaCl solution was injected intravenously over a period of a few minutes. Note the rise in all pressures during systole and diastole except the pleural space pressure. See text for discussion.

Dial pressure equal to 75 per cent of the ventricular pressure increase. Thus, the "effective" or transmural pressure distending the left ventricle is small even though the left ventricular end-diastolic pressure may be quite large; for example, when left ventricular end-diastolic pressure is 25 mm. Hg, the transmural pressure is only 6 mm. Hg.

Relationship Between Pericardial Pressure at the End of Diastole and at the End of Systole

Pericardial pressure decreases during systole as shown in figure 2. The relationship between the end-diastolic pericardial pressure and the end-systolic pericardial pressure in a closed-chest dog subjected to plethora and hemorrhage is shown in figure 4E, and the results of experiments in 9 dogs in figure 4F. Results similar to these were obtained in all animals studied. It will be noted that there is an approximately linear relationship with the pericardial end-systolic pressure increasing a smaller amount than the pericardial end-diastolic pressure. The equation describing these results is:

\[ S = 0.68P - 0.5 \]

which shows that on the average for a given pericardial end-diastolic pressure (P) the pericardial end-systolic pressure (S) is 68 per cent of the pericardial end-diastolic pressure.

Relationship Between Right Ventricular End-Systolic Pressure and Pericardial End-Systolic Pressure

The results of an experiment showing this relationship in a closed-chest animal subjected to plethora and hemorrhage are given in figure 5A. It will be noted that, beginning with the smallest ventricular systolic pressure consistent with life, up to approximately 29 mm. Hg the pericardial pressure remains small and constant; beyond 29 mm. Hg the pericardial pressure rises as the systolic pressure rises. Thus, the measured ventricular end-systolic pressure is the algebraic sum of the pressure developed by the ventricular muscular contraction and the pressure in the pericardial space; the transmural pressure developed by the ventricular muscular contraction during systole is the measured ventricular end-systolic pressure minus the pericardial pressure at that time. When the transmural pressure is calculated in this way for the data in figure 5A, the results obtained are shown in figure 5B. It will be noted that as the measured right ventricular systolic pressure rises, the transmural pressure rises, and after the ventricular systolic pressure has reached a value of approximately 29 mm. Hg, further rise in ventricular systolic pressure is associated with no further rise in transmural pressure. Results similar to these were obtained in all animals studied with the peak transmural pressure varying between 49 and 29 mm. Hg in different experiments. In figure 5C is shown...
the linear relationship between pericardial end-systolic pressure and right ventricular end-diastolic pressure in experiments on 9 dogs. The equation describing this relationship is:

\[ S = 0.58V - 0.7 \]

which shows that on the average for a given rise in right ventricular end-diastolic pressure (V) the pericardial end-systolic pressure (S) rises 58 per cent of the right ventricular end-diastolic pressure. Thus, if the right ventricular end-diastolic pressure is known, the end-systolic pressure may on the average be corrected to right ventricular transmural pressure by reference to the above equation or figure.

**Relationship Between Left Ventricular End-Systolic Pressure and Pericardial End-Systolic Pressure**

The results of an experiment showing this relationship in a closed-chest dog subjected to plethora and hemorrhage are shown in figure 5D. It will be noted that when the ventricular pressure increased from 38 to 125 mm. Hg, there was little or no change in the pericardial end-systolic pressure; as the ventricular systolic pressure rose above 125 mm. Hg, the pericardial pressure rose. Since the end-systolic pressure measured in the left ventricle is the algebraic sum of the pressure developed by the ventricular muscular contraction and the pressure in the pericardial space, the true ventricular pressure resulting from muscular contraction is less than the measured pressure for pressures greater than 125 mm. Hg in this experiment. For example, a pressure of 140 mm. Hg measured in the left ventricle (fig. 5D) is, in actuality, a transmural pressure of 108 mm. Hg. Results similar to this were obtained in all animals studied except in some experiments the increase in measured ventricular systolic pressure, associated with the increase in pericardial end-systolic pressure, was greater than that shown in figure 5D. In figure 5E is shown the relationship between left ventricular end-diastolic pressure and pericardial end-systolic pressure. It will be noted that there is a slightly curvilinear relationship, concave toward the pericardial axis, with the pericardial pressure increasing as left ventricular end-diastolic pressure increases. Results similar to this were obtained in most of the animals studied except a few dogs in which the relationship was linear. In figure 5F is shown the relationship between pericardial end-systolic pressure and left ventricular end-diastolic pressure in experiments on 9 dogs. Although the relationship is slightly curvilinear in many of the individual experiments, as a first approximation the relationship in figure 5F is considered to be linear and the equation describing the relationship is:

\[ S = 0.51V - 0.7 \]

where V is left ventricular end-diastolic pressure and S is pericardial end-systolic pressure. Thus, if the left ventricular end-diastolic pressure is known, the measured left ventricular end-systolic pressure can, as a first approximation, be corrected to left ventricular transmural pressure by means of the above equation.

**Right Atrial Filling Pressure**

At the end of ventricular diastole, right atrial and ventricular pressures are approximately the same (figs. 2 and 6A). During the early part of ventricular systolic ejection, pericardial pressure falls (fig. 2). This is in agreement with the observations of Nerlich's that in experimental pericardial effusion peri-
Cardiac pressure falls during early ventricular systole. Since the atria are located within the pericardial sac, the pressure on the outside of the right atrium decreases markedly during early ventricular systole. As a result, the pressure in the right atrium decreases in early systole and the atrium becomes distended by blood rushing into it from the great veins. A measure of the degree of this atrial "filling pressure" is the difference between right atrial end-diastolic pressure and the pericardial pressure in early systole. The results of an experiment showing the relationship between right atrial end-diastolic pressure and the difference between right atrial end-diastolic pressure and pericardial pressure in early systole, as well as the relationship between right atrial end-diastolic pressure and the difference between right atrial end-diastolic pressure and pericardial end-diastolic pressure, are shown in figure 6B. The length of the vertical lines in the figure is equal to the "filling pressure" which resulted from the decrease in pericardial pressure in early systole. It will be seen that this "filling pressure" of the atrium is considerable, even with a normal right atrial end-diastolic pressure, and that the "filling pressure" is markedly increased as the atrial and end-diastolic pressure increases. Results similar to these were obtained in all animals studied.

In all animals in which the pericardial pressure was measured with a flat or cylindrical balloon lying on the right or left ventricular surface, midway between the base and apex of the heart, the pericardial pressure increased at the onset of systole and fell during the latter part of systole. This is shown in figure 2 in which pericardial pressure was measured with a flat balloon lying over the right ventricle. In all experiments in which pericardial pressure was measured with an open tube in the region of the atria or apex, there was generally no increase in pressure at the onset of systole, and when present was quite small. Although the reason for the increase in pericardial pressure at the onset of systole is not entirely clear, it is felt that it is caused by the ventricles becoming more spherical during isometric contraction and increasing the pericardial pressure locally, and that the decrease in pressure in the region of the apex and base of the heart in early systole is caused by the decrease in ventricular volume occurring with the first part of systolic ejection from the ventricles. After a certain amount of blood is ejected from the ventricles during systole, the volumes of the ventricles have decreased to the point where the pressure on the entire surface of the ventricles decreases. It should be pointed out that some part of the increase in pericardial pressure during early systole may be caused by an artifact resulting from the movement of the ventricular surface against the surface of the balloon.

**Relationship Between Intrapericardial Pressure and Intrapleural Pressure**

It will be noted in figure 2 that, although the pericardial pressure at the end of systole and diastole is markedly elevated following plethora, there was little or no change in intrapleural pressure. Results similar to this were obtained in all animals studied in which intrapleural pressure was measured.

**Discussion**

These results show that in the normal animal the high end-diastolic pressure in the right atrium and ventricles following plethora is associated with a high intrapericardial pressure, and that the intrapleural pressure is little changed. That these high intrapericardial pressures were not caused by our technique of measuring pericardial pressure is indicated by the fact that the total volume of the pericardial contents in dogs, such as we studied, is of the order of 225 ml. (fig. 3). In the experiments in which a balloon was used, the volume of the balloon placed in the pericardial space was no more than 5 ml., and the few ml. of pericardial fluid normally present in the pericardial space flowed from the pericardium when it was opened to insert the balloon; in the experiments in which pericardial pressure was measured with an open-end tube, only 5 or 10 ml. of fluid was injected into the pericardial space and in many cases.
much of this fluid was removed during the course of the experiment without affecting the results.

On the basis of our results, it appears that the practice of defining the "effective" ventricular or atrial pressure as being the difference between ventricular end-diastolic pressure and intrapleural pressure gives erroneous values with the slightest elevation of ventricular end-diastolic pressure. The true "effective" diastolic pressure is the difference between atrial or ventricular end-diastolic pressure and pericardial end-diastolic pressure and has a value of no more than 2 or 3 mm. Hg under all but the most abnormal circumstances, i.e., a measured left ventricular end-diastolic pressure above 10 mm. Hg. Although we have studied only normal animals following plethora and hemorrhage, it would appear that similar results would be obtained under any circumstances when ventricular transmural end-diastolic pressure was greater than 1 mm. Hg, the only necessary condition being that the pericardium was intact. In severe states of congestive heart failure, extending over a long period of time and associated with a very large heart, there is the possibility that the pericardium has become stretched to the point where it no longer functions as above. In addition, there is the
Figure 5

A. Relationship between right ventricular end-systolic pressure (ESP) and pericardial end-systolic pressure (ESP) measured with a flat balloon in a closed-chest dog subjected to plethora and hemorrhage. See text for discussion. B. Relationship between right ventricular end-diastolic pressure (EDP) and right ventricular end-diastolic pressure for the data shown for figure 5A. The transmural pressure is the difference between right ventricular end-systolic pressure and pericardial end-systolic pressure. C. Relationship between right ventricular end-diastolic pressure (EDP) and pericardial end-systolic pressure (ESP) measured with an open tube in open-chest dogs and with a flat balloon in closed-chest dogs. The equation of the line was calculated as in figure 4B. The standard deviation was ± 5.6 mm Hg. S, pericardial end-systolic pressure; V, right ventricular end-diastolic pressure. See text for discussion. D. Relationship between left ventricular end-systolic pressure (ESP) and pericardial end-systolic pressure (ESP) measured with a flat balloon in a closed-chest dog subjected to plethora and hemorrhage. See text for discussion. E. Relationship between left ventricular end-diastolic pressure (EDP) and pericardial end-systolic pressure (ESP) measured with a flat balloon in a closed-chest dog subjected to plethora and hemorrhage. See text for discussion. F. Same as E except that the results are given for 9 dogs in which pericardial pressure was measured with an open tube in open-chest animals and with a flat balloon in closed-chest dogs. The equation of the line was calculated as in figure 4B. The standard deviation was ± 6.5 mm Hg. S, pericardial end-systolic pressure; V, left ventricular end-diastolic pressure.

Possibility that in certain individuals, as a result of a developmental or other abnormality, the pericardium is too large and as a result no longer provides the protective functions described above. Experimental evidence on these points is lacking.

These results confirm those reported earlier by us1–5 that in the intact closed-chest dog, horse, bovine and swine, the right and left ventricles during diastole, beginning with the smallest volume consistent with life, behave more or less as a flaccid bag having little or no increase in end-diastolic pressure as the end-diastolic volume increases. After a certain large end-diastolic volume is reached ventricular end-diastolic pressure increases markedly, although the end-diastolic volume increases little, because the pericardium has become stretched and the pericardial pressure has risen. It would appear that in all normal animals, the relatively nondistensible pericardium prevents the ventricles and atria from becoming markedly distended by a high transmural pressure, and that all of the experiments which have been carried out on the heart by various investigators, after re-
moval of the pericardium, in which the ventricular end-diastolic transmural pressure was increased above approximately 3 mm Hg (fig. 4, B and D) were carried out under circumstances of distension that do not occur in the normal animal. This applies to the studies of Starling and associates on the heart-lung preparation 46 years ago, and all similar studies by others up to the present day. Our results are in general agreement with those of Kuno who stated "a heart in the absence of the pericardium is already in danger when it is submitted during diastole to a diastolic force of venous pressure which is only one-third or one-half of that found under normal conditions."

The sudden decrease in pericardial pressure that occurs with early ventricular systolic ejection is of particular interest because it lowers the pressure around the atria. As a result, the pressure in the atria is suddenly reduced (fig. 2) and the pressure gradient from the great thoracic veins to the right atrium is markedly increased. This appears to be a mechanism by which blood is drawn into the atrium during ventricular systole, and in this way blood is ready to fill the ventricles immediately on cessation of ventricular systole. Thus, with the pericardium intact, the act of ventricular systole draws blood to the ventricle and insures ventricular filling in early diastole. These results are in agreement with those of Böhme and Brecher who showed that there was a large sudden flow of blood through the superior vena cava toward the heart during early ventricular systole. This has been attributed by several investigators, and most recently by Brecher, to the sudden piston-like downward movement of the atrioventricular junction attracting blood from the central veins into the right atrium. Our data indicate that the increased flow into the right atrium is caused by the sudden decrease in pericardial pressure with ventricular systolic ejection, and that this factor becomes greater with higher ventricular diastolic pressures. Confirmation of the importance of the pericardium in this connection is the observation of

![Figure 6](http://circres.ahajournals.org/)

**Figure 6**

A. Relationship between right ventricular end-diastolic pressure (EDP) and right atrial end-diastolic pressure (EDP) in 9 dogs subjected to plethora and hemorrhage. The solid circles are closed-chest dogs in which pericardial pressure was measured with a flat balloon; the open circles are open-chest dogs in which pericardial pressure was measured with an open tube. The equation of the line was obtained as in figure 4B. The standard deviation was ± 3.5 mm Hg. V₂ right ventricular end-diastolic pressure; A₂ right atrial end-diastolic pressure. B. Relationship between "filling pressure" of the right atrium in early ventricular systole and right atrial end-diastolic pressure (EDP) in an open-chest dog subjected to plethora and hemorrhage. The points marked with the open circles have a value on the ordinate equal to the difference between right atrial EDP and pericardial end-diastolic pressure (measured with an open tube located over the right atrium), and the points marked with (x) have a value on the ordinate equal to the difference between right atrial EDP and pericardial pressure measured early during the following ventricular systole. The increase in right atrial "filling pressure" caused by the decrease in pericardial pressure in early systole is represented by the length of the vertical lines; note that this "filling pressure" increases markedly as the right atrial EDP increases.
Brecher\(^6\) that the acceleration of venous flow toward the right atrium during ventricular systole is decreased by opening the pericardium. It would appear that the increased flow into the right atrium during ventricular systole was caused in large part by the decrease in pericardial pressure during early ventricular systole. The question as to how much of this flow is caused by a downward movement of the atrioventricular junction remains unanswered. Quantitative data on this point could be obtained by measuring the flow into the right atrium in the open-chest dog, with the pericardium intact and after complete removal of the pericardium.

Our data indicating that there is an increased rate of flow of blood from the great veins into the right atrium during ventricular systole is consistent with the evidence that there is an increased venous flow into the chest during ventricular systole.\(^11-13\) However, it should be pointed out that, insofar as our data is concerned, the increased flow into the right atrium could come entirely from the thoracic great veins and that there was no increased flow into the chest during systole; this would appear to be the case under those conditions in which the veins entering the chest were "collapsed" beyond the depleting stage of "collapse."\(^9\)

In addition, the pericardium appears to have a particular protective function for the atria. If the pericardium were not present, then during plethora with high right atrial end-diastolic pressure the right atrium, as well as the rest of the heart, would remain greatly distended and stretched by a high transmural pressure throughout the cardiac cycle. As a result it might be damaged by stretch. With the pericardium intact, the right atrium is protected during ventricular diastole from overdistension, and tends to be greatly distended by a high transmural pressure only during part of ventricular systole.

It is clear, we believe, that right ventricular and left ventricular end-systolic pressures are the algebraic sum of the pressure developed by ventricular muscular contraction. Thus, it would appear that the values reported by all investigators for right ventricular systolic and left ventricular systolic pressures, when associated with high right or left ventricular end-diastolic pressures, have been too high insofar as such systolic pressure is supposed to represent the pressure developed by the ventricular muscular contraction. This error is relatively small for the systemic circulation because the left ventricular systolic pressure is relatively large compared to even high ventricular end-diastolic pressures; however, the error may be relatively large in the case of the pulmonary circulation where the normal right ventricular systolic pressure has a value of approximately 25 mm. Hg and is of the same order of magnitude as that of high right ventricular end-diastolic pressures. Since the pressure in the pericardial space at the end of systole is on the average 58 per cent of the right ventricular end-diastolic pressure when the end-diastolic pressure is elevated above a few mm. Hg, a reasonably accurate correction of ventricular end-systolic pressure can be made by subtracting from the right or left ventricular end-systolic pressure 58 per cent of the right ventricular end-diastolic pressure.

Although we have not carried out studies on the left atrium, it appears that it would in general function in a manner similar to that of the right atrium. The possibility of significant local variations in pericardial pressure exists and further studies are needed to clarify this matter.

Summary
In anesthetized open-chest and closed-chest dogs subjected to plethora and hemorrhage, pressures were measured simultaneously in the right atrium, right and left ventricles, and the pleural and pericardial spaces. It was found that an increase in ventricular end-diastolic pressure above approximately 1 mm. Hg was associated with a rise in pericardial pressure of nearly the same amount. Thus, the practice of defining "effective"
ventricular end-diastolic pressure as the difference between the ventricular and intrapleural pressure gives erroneous values when ventricular end-diastolic pressure is greater than a few mm. Hg. The true ventricular transmural pressure is the difference between ventricular end-diastolic and pericardial end-diastolic pressure and has a value of no more than 2 or 3 mm. Hg under all except the most abnormal circumstances. When ventricular end-diastolic pressure is greater than a few mm. Hg, the pericardial pressure at the end of systole is likewise elevated, but to a lesser degree. Thus, when ventricular end-diastolic pressure is elevated, the measured ventricular end-systolic pressure is greater than the pressure developed by ventricular muscular contraction because end-systolic pressure is the algebraic sum of the pressure developed by ventricular muscular contraction and the pressure in the pericardial space. This error in ventricular systolic pressure may be relatively large for the right and small for the left ventricle. Pericardial pressure decreases during ventricular systole, lowers the pressure on the outside of the atria, and thus, tends to draw blood into the right atrium during ventricular systole.

**Summario in Interlingua**

In aneaesthésique canis a thorace aperte e a thorace claudite, que esses subjecitos a plethora e a hemorragia, le tensiones essesu mesurate simultaneamente in lo atrio dexter, le ventriculos dexter e sinistre, e le spatio pleural e pericardial. Esses constatate que un augmento del tension ventricular termino-diastolic per plus que circa 1 mm de Hg essesu asociate con un augmento del tension pericardial per approximativemente le mesma quantitate. Assi le practica de definir le 'effective' tension termino-diastolic ventricular come le differentia inter le tension ventricular e intrapleural resulta in erronees valores quando le tension termino-diastolic ventricular es plus que aficam mm de Hg. Le ver tension transmural ventricular es le differentia inter le tension termino-diastolic ventricular e le tension termino-diastolic pericardial e mounuta a non plus que 2 o 3 mm de Hg sub onse circumstantias, exepute le plus anormale. Quando le tension termino-diastolic ventricular es plus que aficam mm de Hg, le tension pericardial al fin del systole es equalmente elevate ben que non al mesmo grado. Assi, quando le tension termino-diastolic ventricular es elevate, le mesurate tension termino-systolic ventricular es plus grande que lo pression disveloppate per contraction ventriculo-muscular, proque le tension termino-systolic es le summa algebraic del tension disveloppate per le contraction ventriculo-muscular e le tension in le spatio pericardial. Iste error in le tension ventriculo-systolic potess esser relativemente grande pro le ventriculo dexter e relativamente miere pro le ventriculo sinistre. Le tension pericardial decline durante le systole ventricular, reduce le pression al exterior del atrios, e nasi tende a tirar sanguine a in le atrio dexter durante le systole ventricular.

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J. P. HOLT, E. A. RHODE, HELGA KINES and Hawkins Ruth

Circ Res. 1960;8:1171-1181
doi: 10.1161/01.RES.8.6.1171

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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