Relation of Negative Intraventricular Pressure to Ventricular Volume

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Elastic forces of the ventricular walls produce a subatmospheric (negative transmural) pressure in the empty quiescent ventricle of open chest dogs. As fluid is added to the ventricle the negative intraventricular transmural pressures become 0 and finally positive. The resulting ventricular pressure-volume curves are S-shaped, having one limb in the negative and another in the positive pressure range. It is concluded that previously reported suction and negative intraventricular transmural pressures lasting throughout the entire diastole in ventricles with small residual volumes are caused by elastic forces which also prevail under static conditions.

It has recently been demonstrated that the nearly empty, rhythmically beating ventricle is capable of sucking fluid into its cavity. Negative intraventricular pressures and suction have been recorded not only in early diastole but throughout the entire diastole. This indicates that a sucking force exists even when the myocardium is in a relaxed state such as prevails under the relatively static conditions of late diastole. Since the ventricular residual volume was very small under the conditions of those experiments, a study was made of the relation of negative intraventricular pressure to ventricular volume under static conditions. This was done in the quiescent but viable ventricle.

Method

The precise establishment of the 0 level is important for the accurate recording of transmural intraventricular pressures, especially in the low pressure range. Pressure-volume determinations in the past have been done without establishing a 0 level suitable for transmural pressure measurements. Generally, previous workers considered the lowest pressure obtained in the ventricle as 0 pressure and used it as the 0 reference level. This may explain why the occurrence of negative transmural pressure in pressure-volume measurements has escaped observation in the past. In the present experiments the 0 pressure level was established precisely by submerging the ventricle in saline and taking the surface of the “saline lake” as the reference 0 point. Under such conditions subatmospheric pressures in the ventricle represent negative transmural pressures.

Nineteen acute experiments were performed in open chest dogs, ranging in weight from 9.2 to 25.9 Kg. The animals were anesthetized intravenously with sodium pentobarbital (30 mg./Kg.) and then heparinized. The experimental arrangement used for the left ventricle is illustrated in figure 1. The chest was entered between the fourth and fifth rib on the left side and the pericardium was widely incised. A rigid plastic cannula of 4 mm. internal diameter with 4 holes in the tip was inserted into the ventricle through the left atrial appendage. Through it the ventricle could be filled with known amounts of saline from buret B by opening and closing stopcock S (fig. 1). In 5 experiments intraventricular pressures were recorded with a Gregg type optical manometer M of high sensitivity. In 14 experiments pressures were read on water manometers. Pressures were registered via a polyethylene catheter (no. 260) passed inside the intraventricular cannula and extended 5 mm. beyond the cannula tip. Multiple holes at the catheter tip minimized occlusion by intraventricular structures. To establish 0 pressure, the heart was completely submerged by filling the chest with saline. A tube leading from the saline in the chest to the manometer M permitted recording of 0 pressure by closing stopcock V and opening stopcock Z.

Since it was the purpose of these experiments to determine the ventricular pressure-volume relationships under conditions of normal and near normal ventricular wall elasticity, all procedures up to this point were undertaken with the circulation still intact. The pressure-volume measurements were then started immediately after the circulation stopped, while the myocardium was still viable. The measurements were repeated twice to verify that no significant changes of the myocardium occurred. To achieve a quick circulatory standstill the ventricular inflow was stopped by tightening a ligature L around the atrioventricular junction, and the ventricular outflow was blocked by applying a clamp to the aorta and pulmonary artery at their roots. Under these conditions the ventricular...
myocardium either fibrillated, continued its rhythmic contractions, or became quiescent while the atria continued to beat. Immediately after stopping the circulation, the remaining ventricular content was expressed manually into the buret. Pressures were recorded first while the ventricle contained an inexpressible residual volume and then while it was filled from the buret with saline by increments of 1 ml. every 10 sec. In these intervals the pressures stabilized sufficiently for accurate measurements. Eight experiments were carried out for the left ventricle alone and 11 experiments for both ventricles simultaneously, using 2 burets. The saline was kept at body temperature throughout the experiment.

At the end of each experiment the content of the ventricle was again manually expressed and any remaining fluid recovered. This inexpressible amount of fluid is called “minimum residual volume.” It was taken as the base fluid content of the ventricle before any amounts of saline were added for the pressure-volume determinations.

Results

Figure 2 depicts a segment of an original record from a representative experiment illustrating the effect of increasing ventricular volumes on left intraventricular transmural pressures. This segment was taken 8 min. after the ventricular inflow and outflow tracts had been occluded and the ventricle had just stopped beating, although the atria were still contracting. The impacts of the atrial contractions on the manometer system can be seen as ripples in the horizontal part of the tracing. Part A is the tracing of 0 pressure. At the place marked B the ventricular content was expressed by hand whereupon the intraventricular transmural pressure fell to —128 mm. of water pressure as seen in part C. Upon the addition of 1 ml. of fluid the pressure rose from —128 to —56 mm. of water (marked D). Further increments in ventricular content of 1 ml. each resulted in relatively smaller pressure rises as illustrated by the progressively smaller steps of the tracing in part E. The intraventricular pressures remained negative until 7 ml. had been added. At that point, marked F, intramural and extramural pressures reached equilibrium and transmural pressure became 0. Further fluid additions led to positive pressures, as seen in part G.

From this one may conclude that under static conditions the negative intraventricular transmural pressure is caused by elastic forces of the ventricular wall and that the degree of negativity is inversely related to the ventricular volume.

It was of interest to see whether the elastic force would be greater immediately following ventricular contractions and would thereby alter the pressure-volume relationship existing in the quiescent ventricle. This point is illustrated in figure 3. It depicts another segment of the same record shown in figure 2. However, it was taken immediately after ventricular inflow and outflow were stopped, while the ventricles were still beating. Part A indicates 0 pressure. The tracing begins at B after the ventricular content had been expressed. Part C shows the lowest pressure level of —157 mm. water ob-
Fig. 3. Effect of stepwise increases in ventricular volume on intraventricular transmural pressures in the weakly contracting left ventricle of a dog. Calibration as in figure 2.

Fig. 4. Pressure-volume curves of the left ventricle (points and solid line) and right ventricle (open circles and broken line) of a dog heart (weight of dog 15 Kg). Abscissa, intraventricular transmural pressure in mm. of water, ordinate, intraventricular volume in milliliters.

weak myocardial contractions does not appear to alter significantly the general level of the negative intraventricular transmural pressures prevailing under static conditions.

The typical pressure-volume relationships in both ventricles are graphically represented in figure 4. After expressing both ventricles manually, a minimal residual volume of 0.3 ml. was found in the left ventricle and 2 ml. in the right ventricle. These figures were entered in the graph as the minimal volumes at the lowest obtained pressure. Pressures in both ventricles rose with fluid additions, at first steeply, then more gradually. When the right ventricle contained 7 ml. of saline the transmural pressure became 0, whereas with the identical fluid volume the left ventricular pressure was still on the negative side (—11 mm. of water). Left ventricular pressure reached the 0 level with 12 ml. filling, although the same content on the right side produced a positive pressure of 10 mm. of water. With further fluid additions the curves cross, since the left ventricular pressure-volume curve is steeper than the right. The final upturn of the curves is indicated in figure 4 by arrows only, since those parts of the curves are well-known.5,7

The plot in figure 4 reveals (1) that a complete ventricular pressure-volume curve is S-shaped with a negative limb for small

From this it may be concluded that any increase in the elastic force which might follow

The fact that this level is lower than that shown in part C of figure 2 may be explained by a slight difference in evacuating the ventricle by hand. The downward directed systolic spikes in this part of the record are attributable to movement artifacts when the cannula tip came in contact with some structures during the contraction of the empty ventricle.
volumes and the familiar positive limb for large volumes, (2) that the suction force of the left ventricle is greater than that of the right, (3) that the wall tension changes least in the range of 0 and slightly positive pressures (least inclination of slope) and (4) that conversely the modulus of elasticity of the walls increases with extreme degrees of emptying as well as filling.

The amount of fluid, contained in the ventricles when transmural pressure became 0 after saline additions, is given in table 1 for 17 technically reliable experiments. The average volume at 0 transmural pressure was 5.1 ml. in the right ventricle and 8.2 ml. in the left ventricle. The average heart weight was 126.2 Gm. In no case did the right or left negative intraventricular transmural pressure exceed —300 mm. of water when the ventricle was emptied as completely as possible under the experimental conditions. There does not appear to be a close correlation between ventricular volume at 0 transmural pressure and heart weight. The small sample number may explain the lack of correlation.

**DISCUSSION**

It has been demonstrated by these experiments that under static conditions elastic forces of the ventricular walls tend to expand the ventricular cavities and thereby produce negative intraventricular transmural pressures. The effect of these forces becomes greater the smaller the ventricular volume.

These observations can be applied with some reservation to the beating heart. They indicate that a ventricle with a small residual volume would create a negative transmural pressure and hence suction when the myocardium is relaxed, such as would be the case toward the end of diastole. The existence of an elastic force under static conditions explains the previously described phenomenon of negative intraventricular pressures and ventricular filling by suction which lasted throughout the entire diastole. The elastic forces may be the same as those which Rushmer and his co-workers have postulated to occur during ventricular contractions because of stretching of connections between different ventricular muscle layers and because of the compression of the myocardial fibers of the inner layers of the myocardium by the relatively greater contraction of the outer as compared to the inner layers. Rushmer has called those forces "interfascicular tension" which tend to restore the ventricular cavities.

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**TABLE 1.—Ventricular Volume Present at Zero Intraventricular Transmural Pressure**

<table>
<thead>
<tr>
<th>Experiment number</th>
<th>Negative intraventricular pressure after expressing ventricle</th>
<th>Minimal residual volume after expressing ventricle (ml.)</th>
<th>Added volume to attain zero pressure (ml.)</th>
<th>Total volume to attain zero pressure (ml.)</th>
<th>Weight of heart (Gm.)</th>
<th>Weight of dog (Kg.)</th>
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<tbody>
<tr>
<td>1</td>
<td>—23</td>
<td>0.7</td>
<td>5.8</td>
<td>6.5</td>
<td>87.0</td>
<td>12.4</td>
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<td>2</td>
<td>—53</td>
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<td>20.2</td>
<td>22.5</td>
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<tr>
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<td>—208</td>
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<td>3.3</td>
<td>4.8</td>
<td>20.0</td>
<td>14.0</td>
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<tr>
<td>4</td>
<td>—70</td>
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<td>15.0</td>
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<tr>
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<td>8.5</td>
<td>63.0</td>
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</tr>
<tr>
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<td>2.8</td>
</tr>
<tr>
<td>7</td>
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<td>2.0</td>
<td>12.0</td>
<td>123.0</td>
<td>7.0</td>
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<tr>
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<td>8.0</td>
<td>9.6</td>
<td>6.3</td>
</tr>
<tr>
<td>10</td>
<td>—300 —80</td>
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<td>6.4</td>
<td>9.2</td>
<td>10.0</td>
<td>10.0</td>
</tr>
<tr>
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<td>1.3</td>
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<tr>
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<td>164.0</td>
<td>20.6</td>
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<tr>
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<td>0.6</td>
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<td>4.0</td>
</tr>
<tr>
<td>14</td>
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<td>4.2</td>
</tr>
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<td>5.6</td>
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<tr>
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<td>1.2</td>
<td>7.0</td>
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</tr>
<tr>
<td>17</td>
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<td>0.4</td>
<td>5.0</td>
<td>5.6</td>
<td>4.2</td>
</tr>
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</table>

Average —192 —157

1.2 1.8
7.0 3.4
8.2 5.1
126.2 16.9
toward their diastolic dimensions. It is reasonable to assume that similar physical forces of interfascicular tension would develop regardless of the process which induces the compression and wrinkling of the deeper layers of the myocardium, that is, whether it is brought about by artificial means such as manual expression or aspiration of the ventricular content or by a relatively greater contraction of the external layers of the myocardium. In either case some portion of the myocardium is elastically deformed in such manner that it tends to expand the ventricular cavity.

The elastic force demonstrated under static conditions may be smaller than that existing immediately following ventricular relaxation, especially if the relaxation occurs quickly after a strong myocardial contraction. Our experiments cannot reveal information on this point. It can only be stated that weak myocardial contractions do not appear to change the elastic forces from those observed under static conditions (compare figs. 2 and 3).

Our results indicate that the elastic forces responsible for ventricular filling by diastolic suction under relatively static conditions can only come into play when the residual volume of the ventricle is below a certain amount, and that knowledge of this volume would aid in predicting the possible contribution of diastolic suction to ventricular filling under various conditions.

**SUMMARY**

The relationship of negative intraventricular transmural pressure to ventricular volume was studied under static conditions in acute experiments on anesthetized open chest dogs. Ventricular inflow and outflow were blocked and intraventricular transmural pressures of the quiescent but still viable ventricles were measured after 0 transmural pressure was established by submerging the heart in saline.

Manual expression of the ventricular content always resulted in negative intraventricular transmural pressures. For 17 experiments the average amount of fluid added to the right and left ventricles to raise negative transmural pressures to 0 was 5.1 and 8.2 ml, respectively, in hearts of 126.2 Gm. average weight.

The relationship of volume to negative transmural pressure represents a negative limb in addition to the known positive limb of the ventricular pressure-volume curve. Both limbs form an S-shaped curve in which the least pressure-volume change occurs approximately at 0 transmural pressure.

It is concluded that elastic forces tend to restore diastolic ventricular dimensions under static conditions in the nearly empty ventricle and that these forces are in part responsible for the intraventricular suction, which has been demonstrated previously to occur throughout the duration of diastole when the ventricular residual volume is small.

**SUMMARIO IN INTERLINGUA**

Le relation inter negative pression transmural intraventricular e volumine ventricular eseva studiate sub conditiones static in experimentos acute con anesthesiate canes a thorace aperte. Influxo e effluxo ventricular eseva blocate, e le pressiones transmural intraventricular del quiescente sed ancora viabile ventriculos eseva mesurate post que le pression transmural zero habeva essite establite per submerger le corde in solution salin.

Le expression manual del contento ventricular resultava semper in negative pressiones transmural intraventricular. In 17 experimentos, le quantitate medie de fluido addite al dextere e sinistre ventriculos pro elevar negative pressiones transmural al nivello zero eseva 5,1 e 8,2 ml respectivemente, con cordes de un peso medie de 126,2 g.

In le curva pro pression e volumine ventricular, le relation inter volumine e negative pression transmural permette le addition de un branca negativa al cognoscite branca positive. Le duo brancas insimul produce un curva in forma de S. In illo le alteration minimal de pression como function de volumine occurre approximativamente al nivello zero del pression transmural.

Es formulate le conclusion que fortias elastic tende a restaurar le dimensiones ventricular diastolic sub conditiones static in le quasi vacue ventriculo e que iste fortias es es parte responsabile pro le succion intraventricular que ha previemente essite demonstrate como occurrentia coincidente con le integre duration del diastole quando le residue volumine ventricular es parve.
Medical Reading and Writing

The success of a scientific article is not measured by the number of reprints requested, nor by the number of persons who read the communication. Its impact is determined by the number who read it critically and gain by comparing what they and the author, respectively, have observed and inferred.

In an essay on how to read medical literature Allan Gregg has stressed the point that "knowing what not to read becomes a cardinal point of progress in the general task of learning how to read." Contributors to the cardiovascular literature who hope that their work will add a brick or two to the building of the wall of knowledge, can profit from perusal of Gregg's admirable exposition. He examines a number of faults of medical writing:

1. Expression of ideas in terms familiar to the author but not to most of his readers.
2. Conversion of the simple into the more complex by means of formulas, instead of the reverse.
3. Failure to make the vague more precise.
4. Tiresome use of notational systems difficult to grasp.
5. Lack of correct modifiers of the subject, verb and object, such as all or some, always or sometimes, only, etc.
6. Obliviousness of the fact that "results usually come from many causes not one."
7. Use of "good horse sense" instead of laws of probability in assessing the plausibility of deductions.
8. Inability to keep on the track in reasoning, facetiously styled "derailment of reason."

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