Volume Pressure Relationships of the Pulmonary-Left Heart Vascular Segment

Evidence for a “Valvelike” Closure of the Pulmonary Veins

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Dynamic considerations indicate that the pulmonary veins, left atrium, and left ventricle are a freely communicating system during diastole. This concept suggests that the distensibility of the pulmonary-left heart structures are an important determinant of left heart diastolic pressure. Most studies of this parameter have been carried out in isolated perfused preparations or deduced from indirect measurements. Accordingly, this investigation was undertaken to quantify, in the living animal, the elastic properties of this segment of the vascular system.

In the course of these studies, it began to appear that at low atrial pressure levels the pulmonary-left heart vascular segment was not a freely communicating unit. This observation is in agreement with the findings of Borst and co-workers, who presented evidence that the pulmonary vascular pathways tend to close at low pressure levels and suggested that this was due to critical closing. The precise location of this closure was not delineated in their study. Burton has suggested, on theoretical grounds, that critical closing is most likely to occur in the arterioles. Our preliminary observations indicated, however, that the block in the pulmonary system occurred in the pulmonary veins or at their junction with the left atrium. Therefore, the investigation was extended to include a study of this discontinuity in the pulmonary-left heart system.

Methods

Two experimental preparations were utilized in this study. The first was developed to measure the distensibility of the pulmonary-left heart unit in the living animal. The second preparation was used to investigate more directly the apparent discontinuity in this vascular segment.

First Experimental Preparation

This preparation has been described in abstract form and is given here in detail. Mongrel dogs were anesthetized with sodium pentobarbital (30 mg./Kg. intravenously) and the chest opened, using a sternal splitting procedure. The pericardium was opened. Respiration was maintained artificially but was interrupted, during the experimental procedures, to avoid artifacts due to lung expansion. The azygos vein was ligated, and tapes were placed around the superior and inferior vena cavae and the pulmonary artery. The left atrium was doubly cannulated through 2 pulmonary veins. One cannula was used for pressure recordings and the second for injection. In a few experiments, a cannula was also placed in a peripheral pulmonary vein and directed toward the left atrium. The right atrium was cannulated via the jugular vein. A sound was introduced via the carotid artery to the arch of the aorta. Phasic right and left atrial, pulmonary vein and aortic pressures were measured electrically, using Statham strain gages and recorded photographically by an Electronics-for-Medicine Research Recorder. The zero level for these pressure recordings was the hydrostatic level of the midleft atrium.

During the experimental procedure, ventricular asystole was produced by electrical stimulation of the left vagus nerve or, in a few experiments, by injecting potassium chloride into the left heart cavity. Dilation of the right heart was prevented by occluding the cavae during the period of cardiac standstill. The maintenance of essentially normal right atrial pressure levels during the period of asystole indicated that pooling of blood did not occur in the right heart. During the experimental period, inflow into the pulmonary-left heart system was prevented by occluding the pulmonary artery. A measured volume of saline or heparinized blood was then injected rapidly into the left heart through one of the cannulae in the left atrium. In those cases where vagal stimulation was used, ventricular asystole lasted for as long as 8 to 10 seconds after the injection. When ventricular...
escape occurred, vagal stimulation was discontinued, the tapes occluding the cavae and pulmonary artery were loosened, and the circulation returned to normal. In this way, a number of different injections was made in each preparation. When cardiac standstill was produced chemically, serial injections of 1 or 5 ml units were employed and the entire study completed within 1 or 2 minutes while the preparation was viable.

**Second Experimental Preparation**

Dogs were anesthetized and the chest opened as described above. The right atrium was doubly cannulated via 2 pulmonary veins. A third cannula was placed in the periphery of a different pulmonary vein and directed toward the left atrium. The animal was heparinized, and the heart stopped by electrically induced ventricular fibrillation. The pulmonary artery and the proximal aorta were clamped. The pulmonary-left heart vascular space was emptied, as completely as possible, by manual compression of the heart and the evacuation of blood from the left atrium and pulmonary vein. The thorax was filled with water until it covered the heart and lungs. The upper level of this fluid was used as a common zero hydrostatic level for all pressure measurements.

Heparinized blood was injected into the left atrium, and simultaneous measurements were made of left atrial and pulmonary vein pressure, using the recording equipment described in the first experimental preparation. The injections were made in 1 or 5 ml units with a few seconds between injections, or, in some experiments, a single injection of 50 ml was used.

**Results and Discussion**

A typical record of left atrial and aortic pressure during an injection of 25 ml of saline into the left atrium of the first experimental preparation is shown in figure 1. Left atrial pressure rose rapidly during the injection and remained constant at its higher level for the remainder of ventricular asystole. Aortic pressure gradually fell during this period; however, it was always higher than intracardiac pressure. This aortic-intracardiac pressure gradient prevented outflow through the closed aortic valves. The constant atrial pressure following the injection indicated that the system did not have significant leaks during the period of cardiac standstill.

Failure of atrial pressure, in figure 1, to decline during the short interval of post-injection cardiac standstill may not be at variance with the stress-relaxation which has been reported for the pulmonary system, but rather reflects the relatively low level of atrial pressure. Some decline in the postinjection atrial pressure did occur with time in those records in which atrial pressures exceeded 12 to 15 mm Hg.

Records that showed preinjection atrial pressures outside the normal range (0 to 3 mm Hg) or that showed variation within any experiment of more than ± 0.5 mm Hg from the other preinjection pressures were discarded. The remaining records were utilized to construct a volume-pressure curve for each dog. These were made by plotting the change in atrial pressure against the volume injected. A total of 10 such curves was suitable for analysis. Seven curves represented animals in which vagal stimulation was used to produce ventricular asystole and in which saline was injected. Three curves were constructed from experiments in which cardiac arrest was produced with potassium chloride and blood was the fluid injected. All curves showed the same essential features. A representative curve is shown in figure 2.

A regression line for each volume-pressure plot was determined by the method of least squares. In each case, this line was essentially linear and intercepted the pressure axis of the plot. Failure of the regression line to pass through the origin indicates a change in pressure without a change in volume, which in
Figure 2

Volume-pressure relationships shown by plotting change in pressure against the volume injected (change in volume).

Figure 3

Plot of left atrial and pulmonary vein pressures compared to volume of blood injected into the left atrium during ventricular arrest produced by potassium chloride. (See text for discussion.)

this situation is untenable. Critical examination of the data indicated that 2 regression lines could be drawn. These lines join at pressure changes of 0.2 to 4 mm. Hg. Above this point the relationship is linear; below this point the relationship cannot be determined with accuracy, due to the limited number of determinations. However, it too appears to be linear, or nearly linear.

The abrupt break in the volume-pressure relationship for the pulmonary-left heart unit suggests a sudden change from one volume-pressure system to another. Several possibilities present themselves. For example, this could represent a transition from a partially filled venous reservoir to a distended elastic system. Inspection of the data, however, indicated that at pressures below the break point, the system is relatively rigid, in that small changes in volume give large changes in pressure. Above the break point, the system is more elastic, with the same change in volume giving smaller changes in pressure. These findings are not in accord with a change from a partially filled to a more rigid system, as would occur in the filling of a collapsed venous segment.8

A second possible explanation for the discontinuity of the volume-pressure curve is that the total volume of the system might be increased at the break point in the plot. If this were the case, the change in slope of the second part of the curve would represent a transformation of the volume scale. Such a transformation would result from the smaller per cent increment in volume for each unit volume change due to the larger initial volume. Thus, the break in the volume-pressure curve might represent the opening of additional vascular space and not a marked change in the distensibility of the system.

It is interesting that a reduction of the ordinate scale of figure 2 by 37.5 per cent at and above the break point in the curve will give a single linear relationship over the entire plot. If an initial volume of 10 ml. is assigned to the atrial-ventricular cavity of the heart, the calculated increase in the total volume necessary to give the break in the curve of figure 2 is 16.6 ml.

The mechanism for such an augmentation of the vascular volume is not clear from these data. If closing occurred in the pulmonary veins at or near the left atrium at low atrial pressure levels, this closure would prevent fluid from entering the pulmonary vascular system with small changes in left atrial pres-
sure. Higher atrial pressure levels would overcome this block and permit perfusion of the pulmonary vascular pathways. Such a mechanism would be consistent with the observed break in the volume-pressure curve.

A second possibility is that the valvelike openings, reported at the entrance of the pulmonary veins into the left atrium, might prevent regurgitation into the pulmonary venous system at low pressures but become incontinent at higher atrial volumes.

It, therefore, became important to determine more precisely if blockage of the pulmonary system occurs at low pressure levels and, if it does, to delineate the point of closure. This was first studied by recording pulmonary vein pressure in addition to left atrial pressure in 3 animals. In these studies, the experimental preparation was modified in that the animals were heparinized (8 to 10 mg./Kg.) and cardiac arrest was produced with potassium chloride to avoid any reflex effects of vagal stimulation. Prior to starting the injection into the quiescent heart, left atrial pressure was reduced to zero by aspirating through the injecting cannula. Figure 3 shows a plot of left atrial and pulmonary vein pressures as a function of the volume of blood injected. At the onset of the injection, a pressure gradient of 5 mm. Hg was present between the pulmonary vein and the left atrium. During the injection, left atrial pressure increased, first, until it reached a slightly higher level than the pressure of the pulmonary vein. At this point, pulmonary vein pressure began to increase, and the 2 pressures rose together.

This pressure relationship is consistent with a block in the pulmonary vein near or at its junction with the left atrium. However, the differences in the hydrostatic level of the 2 cannulae might account for at least part of the initial pressure gradient. In order to control this factor, the second experimental preparation was developed. In the use of this second preparation, every effort was made to conduct the studies as soon as possible after cessation of cardiac activity. However, the heart was not considered to be viable at the time of study.

Figure 4 shows a record of left atrial and pulmonary vein pressure during an injection of 50 ml. of blood into the left atrium of the second experimental preparation. As the zero pressure level for both the left atrial and pulmonary vein are related to the top of the surrounding fluid lake, any positive pressure recorded is independent of the level of the cannula tip and represents a distending intravascular force. Left atrial pressure rose rapidly during the first part of the injection. During this interval, pulmonary vein pressure showed only a slight increase. During the second part of the injection, pulmonary vein pressure rose very rapidly, while the rate of increase in left atrial pressure was reduced. Left atrial and pulmonary pressure equalized and then increased together during the last phase of the injection.

The variation in the rate of pressure increase is more apparent in figure 5, where the volume of blood injected in a different experiment is plotted against the left atrial and the pulmonary vein pressure. The failure of pulmonary vein pressure to equal left atrial pressure during the initial phase of the injection is indicative of some block between these 2 structures. However, when left atrial pressure reached a critical level, pulmonary vein pressure suddenly increased to equal left atrial pressure, presumably due to removal of this block.
The mechanism responsible for this block remains speculative. The myocardial bands, reported to close off the venous openings during atrial systole, do not seem to be implicated as the block occurs during diastole and also in the dead heart. Similarly, reflex constriction of the veno-atrial junction does not seem to be a factor. Although this block may represent critical closing of the pulmonary veins, it seems more likely that this valvelike action is due to a collapse of the veno-atrial junction. Such a mechanism would explain the free communication that occurs with mild distention of the atrial system. It should be pointed out, however, that these studies were carried out in open-chest preparations. The effect, if any, of normal intrapleural pressure relationships on such a "valve" mechanism cannot be answered by this investigation.

It is interesting that the atrial volume-pressure diagram for the dead heart shows the same change in slope as demonstrated in the living animal. As predicted, this break coincides with the rapid rise in pulmonary vein pressure.

These observations give indirect evidence that the pulmonary-left heart vascular segment is not a freely communicating unit under all conditions, but rather has a valvelike mechanism which may normally prevent backflow from the atrium into the lungs. This mechanism may offer an explanation for the failure of rapid phasic changes in atrial pressure to be faithfully transmitted to the pulmonary capillaries (wedge pressure) particularly at low atrial pressure levels.

The essentially linear relationship above the break in the volume-pressure curve reported here is at some variance with the curvilinear relationship reported by others. This discrepancy may be due to the smaller volumes used in this study. For example, Sarnoff and Berglund utilized volumes of the order of 175 ml. to perfuse the pulmonary vascular bed. The pressure encountered in their study was considerably above that normally found in the pulmonary venous system.

Summary

Volume-pressure curves were determined for the living left heart vascular segment by injecting fluid into the left atrium during momentary cardiac asystole and measuring left atrial pressure. Such curves show 2 essentially linear relationships with a break at a low pressure level. It has been suggested that this break is due to closure of the pulmonary veins or the pulmonary-left atrial junction at low pressure levels. This closure prevents regurgitation into the pulmonary veins. Higher atrial pressures overcome this block and permit retrograde perfusion of the pulmonary pathways. This suggestion was tested by measuring left atrial and pulmonary vein pressure during the injection of blood into the atrium of the quiescent heart. The results are consistent with such a collapse of the veno-left atrial junction at low pressure levels.

Summario in Interlingua

Curvas del relation inter volumino e tension osserva determinate pro le intacte segmento vascular del corde sinistre per injicer liquido a in le atrio sinistre durante un momentane asystole cardine, sequite per le mensuration del tension sinistro-atrial. Tal curvas representa 2 relationes de conformation essencialmente linear, con un ruptura a un basse nivello de tension. Il ha esseite suggesto que iste ruptura es le effecto de un clausura del vonas pulmonar o del junction de pulmon e atrio sinistre occurrente a basse nivello de tension. Iste clausion preveni regurgitation a in le vonas pulmonar. Plus alte tensiones vinse iste
bloco e permite a perfusão retrograda dos vasos pulmonares. Esta sugestão foi testada para medir a tensão sinistro-atrial e pulmôno-venosa durante a injeção de sangue no átrio do coração em estado de quiescência. Os resultados suportam a teoria de um colapso da junção entre os vasos e o átrio esquerdo a baixos níveis de tensão.

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