Ventricular Function

Role of the Pericardium in Regulation of Cardiovascular Hemodynamics

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The influence of the pericardium on cardiovascular phenomena has been examined and evaluated in the light of both ventricular function (modified Starling) curves and pressure-volume relationship of the pericardium. When the left ventricle is stressed, as by an increased aortic resistance, it dilates. The resulting pericardial tension limits right ventricular diastolic expansion and right ventricular work. By this mechanism pulmonary blood volume and pressures are kept at lower levels than would otherwise be present. The pericardium also protects against ventriculo-atrial regurgitation which may account for the observed descending limb of the classically conceived Starling curve.

This communication will deal with the influence of the pericardium on cardiovascular hemodynamics especially when the circulation is subjected to abnormal conditions such as pronounced stress of the left ventricle. Data will be presented which appear to support the following:

The pericardium restrains diastolic expansion and, therefore, ventricular work at high atrial or end-diastolic ventricular pressures.

When the left ventricle is severely stressed and dilated, the pericardium restrains diastolic expansion of the right ventricle and, therefore, limits right ventricular work; the consequences are lower pulmonary capillary and left atrial pressures than would result if the pericardium were not present.

The pericardium protects against tricuspid or mitral valve regurgitation at high ventricular filling pressures; the significance of this for the so-called descending limb of the Starling curve will be discussed. A preliminary report has been presented elsewhere.

The terms "effective" and "apparent" ventricular filling pressures will be used as indicated in previous publications.

METHOD

Mongrel dogs weighing between 16.2 and 27.2 kilograms were anesthetized with morphine-chloralose-urethane and maintained on positive pressure breathing with a constant tidal volume. The methods used have been previously described, and discussed in detail. Briefly, they involve the continuous registration of systemic blood flow (cardiac output minus coronary blood flow), right and left atrial and pulmonary artery and aortic pressures. From these data the stroke work for each ventricle was calculated over a wide range of filling pressures, and right and left ventricular function curves were constructed.

The blood volume of the dog and, thus, the atrial pressures were altered in a stepwise fashion by infusion from a reservoir containing dextran and blood. The latter was connected to the right or left atrium or, in later experiments, to a femoral vein. When the atrium was used a small hole was made in the pericardium for the tubing connecting the reservoir. Pericardial constriction was produced by pulling together two rows of sutures previously attached two to four cm. apart on the surface of the pericardium. When the pericardium was opened it was cut wide open. A standard aortic constriction was obtained by completely occluding the main lumen of the aortic tubing and thereby diverting the total aortic blood flow through a narrow bypass. The
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Before After

Fig. 1. Effect of Pericardium on Left Atrial Pressure Elevation Following an Acute Increase in Aortic Resistance. Exp. #49. Vagi intact. Standard aortic constriction with normal pericardium (O • • • O), with open pericardium, (X———X). Blood then infused so that “initial” left atrial pressure before aortic constriction was higher. Aortic constriction then repeated with open (X———X) and closed, slightly constricted (□———□) pericardium. Note more marked elevation of left atrial pressure following aortic constriction with pericardium open. Heart rate 160–182/min. Ordinate, mean left atrial pressure in cm. H2O. Values at left before values at right after aortic constriction.

RESULTS

Effect of Pericardium on Left Atrial Pressure Elevation Following an Acute Increase in Aortic Resistance

When left ventricular output was suddenly diverted through the high resistance bypass, left atrial pressure rose. Fig. 1 shows the effect of the pericardium on the magnitude of this rise. At initial levels of 13 to 16 cm. H2O, the rise in left atrial pressure after aortic constriction was somewhat larger with an open pericardium than with the normal pericardium. At higher initial levels of left atrial pressure, the resultant elevation was still more pronounced with an open pericardium. This rise, from 21.5 to 29 cm. H2O, could be abolished by closing and slightly constricting the pericardium. It would appear, therefore, that the pericardium is, in some way, involved in preventing excessively high pulmonary vascular pressures when the left ventricle is stressed.

Fig. 2. Effect of Constricting and Opening the Pericardium when the Left Ventricle is stressed. Exp. #122. Vagotomized. Dog weight 17.0 kg., Heart weight 145 gm. From top to bottom, mean atrial and arterial pressures, stroke volumes and stroke work of right and left ventricles. Heart rate 190–198/min. Left atrial pressure high due to aortic constriction and increased blood volume. Note that pericardial constriction in this stage lowers left atrial pressure, and pericardiotomy increases it. See text.
The Effect on Cardiovascular Hemodynamics of Constricting and Opening the Pericardium While the Left Ventricle is Stressed

Figure 2 shows the hemodynamic consequences of constricting, releasing and finally opening the pericardium after high left atrial pressures had been produced by increasing the resistance to left ventricular outflow and by increasing blood volume. At the left, the pericardium is normal. Constricting the pericardium produced a lowering of left atrial pressure while right atrial pressure rose. The stroke work of both the right and left ventricle fell. The pericardial constriction was then released and all values returned to their previous levels. The pericardium was then opened. Left atrial pressure rose while left ventricular work was unchanged; right atrial pressure fell as right ventricular stroke work increased. The data presented below are aimed at explaining these phenomena.

Right and Left Ventricular Function Curves with a Normal, Open and Constricted Pericardium

Two different types of right and left ventricular function curves were obtained with the pericardium normal, open and constricted (fig. 3). The first type was with a normal left ventricular outflow resistance (solid line) and the second with a high left ventricular outflow resistance (broken line). With the normal pericardium, both the right and left ventricular function curves are depressed when aortic resistance is high. The depression of the left ventricular function curve that occurs with a high outflow resistance is clearly a real one since this depression is also present after the pericardium is removed. However, the depression of the right ventricular function curve is only an apparent or reflected one, since, after removal of the pericardium, it is no longer present. Conversely, after the pericardium is

![Diagram of hemodynamic consequences](https://example.com/diagram.png)
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NORMAL PERICARDIUM

EXP H

OPEN PERICARDIUM

RIGHT ATRIAL MEAN PRESSURE CM. HO

Fig. 4. Comparison of the Effect of Aortic Constriction on Right and Left Ventri cle Function Curves with Open and Normal Pericardium. Symbols same as in figure 3. All dogs except #31 vagotomized. Note that depression of the right ventricle function curve with aortic constriction was either diminished or abolished by pericardiotomy.

constricted, the reflected depression of the right ventricular function curve with a high aortic resistance is relatively greater than that on the left.

This type of comparison of ventricular function curves with a normal and an open pericardium and normal and high aortic resistance was done in six dogs (fig. 4). With a normal pericardium a depression of the right ventricular function curve was always present when a high aortic resistance was applied. After the pericardium was opened, this depression of the curve was either abolished or diminished.

Protection of Ventriculo-Atrial Regurgitation by the Pericardium

In some experiments tricuspid or mitral regurgitation occurred with an open pericardium at higher atrial pressure levels; whereas at similar or higher levels, it did not occur with an intact pericardium. An example of this is shown in fig. 5. In the two right atrial pressure tracings with an open pericardium (C and D) there is a marked positive pressure wave almost coincident with ventricular systole; this was not present with an intact pericardium (A and B).

When studying ventricular function it is important to note that normally the mean atrial pressure approximates end-diastolic atrial pressure, while, in the presence of valvular regurgitation, the mean atrial pressure is substantially higher than the end-diastolic. Consequently, mean atrial pressure under those circumstances should not be used, either as an index of diastolic ventricular volume or
Fig. 5. Ventriculo-Atrial regurgitation with open pericardium. Exp. 122. Dog weight 17 Kg. Heart weight 145 gm. Vagotomized. Note: with open pericardium a marked positive systolic pressure wave in right auricle which is absent when the pericardium is closed.

Discussion

It has been amply demonstrated that the potential volume of the pericardium is larger than that occupied by the normal heart in diastole. However, circumstances have been encountered in which the pericardium does influence heart size and cardiovascular dynamics. This has been attributed by Nelemans to the structural composition of the pericardium, namely, the interwoven pattern of elastic fibers and less distensible collagen fibers. It was felt that distention of the pericardium is accompanied by stretching of the elastic fibers, and, after these are stretched, further dilatation is hindered by the collagen fibers. The pressure-volume curves of the pericardial sac or length-tension curve of pericardial strips do show only an initial slight rise of pressure with substantial initial increases in volume, and this pressure rise then becomes increasingly large with further small volume increments. The restraining effect of the pericardium on ventricular diastolic size and consequently on ventricular work is therefore more pronounced at increased ventricular pressures and volumes. These data may explain the variability in the results of Gibbon and Churchill on hemodynamic effects of the pericardium.

The apparent lowering of the right ventricular function curve, when the left ventricle is severely stressed with an intact pericardium, is a direct consequence of Starling’s law of the heart. When the left ventricle is dilated to a point where diastolic intrapericardial pressure is elevated, the effective filling pressure and stroke work of the right ventricle are lower than that expected from the mean right atrial or apparent filling pressure.

Conditions such as hypertension, which differentially stress the left ventricle, may cause it to dilate and occupy a disproportionately larger part of the pericardial space. At such times, if the pericardium is on relatively steep portion of its pressure-volume curve, the effective right ventricular diastolic pressure and stroke work are limited. As a consequence, the pulmonary blood pressures and volume are held at lower levels than would be the case if the restraining influence of the pericardium were not present (fig. 1). Further after the left ventricle had been stressed, constricting the pericardium elevated right atrial or apparent right ventricular filling pressure (fig. 2). However, the effective right ventricular filling pressure, which determines diastolic fiber length, was decreased since right ventricular stroke work fell. When the pericardium was opened, right ventricular stroke work rose even though its apparent filling pressure fell. This is attributable to the fact that the effective filling pressure rose when the pericardium was removed even though the measured right atrial pressure was lower. The left ventricular stroke work did not rise even though the left atrial pressure did; at these levels, the left ventricle was on the flat part of its ventricular function curve as determined in the same dog.

These data are entirely consonant with the findings of Rössler and Unna. They found in heart-lung preparations a rise of left atrial pressure after pericardiotomy when this pres-
sure was initially high. They felt that in the presence of left ventricular strain the pericardium exerted a restraining influence predominantly on the right ventricle and thereby protected the left ventricle from overload and the lungs from congestion. The above analysis gives a somewhat more complete understanding of these phenomena. The above data are also in agreement with the findings of Evans and Matsuoka and of Kuno, that pericardiotomy in the heart-lung preparation was followed by lower filling pressures, larger heart volume, increased oxygen consumption and increased ventricular work.

It will be noted from fig. 4 that the depression of the right ventricular function curves accompanying the high aortic resistance was sometimes abolished, but sometimes only diminished after pericardiotomy. This suggests that some mechanism other than the pericardial one may also be operating the experiments of Henderson and Prince are informative in this regard. In the isolated cat heart without a pericardium, marked overdistention of the left ventricle impaired the stroke amplitude of the right ventricle. It may be that the septal bulge from left to right varies with the diastolic pressure gradient between the right and left ventricles. That this gradient varies markedly with aortic constriction has already been shown. From fig. 4, however, it would appear that if this type of mechanism is operative, it is of secondary rather than of primary importance.

Regurgitation through the A-V valves in dilated hearts was reported in experiments with open pericardium. Winiwarter was able to prevent this regurgitation in the frog heart by constricting the A-V ring. He later emphasized that the descending limb on the “Starling Curve” that regularly occurred with an open pericardium, was due to valvular regurgitation. Frank has also emphasized the importance of excluding regurgitation in the analysis of ventricular performance. The descending limb of the “normal” Starling curve was demonstrated in preparations with an open pericardium and rare was not taken to ascertain or exclude the presence of mitral or tricuspid regurgitation. In this laboratory a true descending limb was found only when the metabolic needs of the myocardium were compromised as by induced coronary stenosis or severe anemia. By contrast, an apparent descending limb may occur in the presence of valvular regurgitation.

The therapeutic effects of positive pressure breathing in acute lung edema, when they occur, may perhaps be explained in part by a consideration of the phenomena described above. In some types of pulmonary edema the left atrial and pulmonary capillary pressures are markedly elevated and it is very likely that the left ventricle is on the flat part of its ventricular function curve or on its descending limb. The right ventricle, however, is still on the ascending part of its function curve. The application of positive pressure breathing results in a decrease of the net or effective filling pressure of both ventricles. This diminution of effective right ventricle filling pressure decreases its stroke work while the diminution of effective left ventricle filling pressure will either increase its stroke work or leave it unchanged. The physiologic consequence would be a lowering of pulmonary blood volume and pulmonary capillary pressure.

SUMMARY AND CONCLUSIONS

When the left ventricle is differentially stressed, as by substantially increasing its outflow resistance, it dilates and occupies a disproportionately large part of the potential pericardial space. Under these circumstances, when the pericardium is on a relatively steep portion of its pressure-volume curve, the effective right ventricular diastolic pressure and expansion is limited. As a consequence, the resulting right ventricular stroke work is also limited and pulmonary blood volume and pressures are held at lower levels than would be the case if the restraining influence of the pericardium were not present.

The action of the pericardium might be construed as limiting the elevation of pulmonary capillary pressure and the edemogenic influence thereof when the left ventricle is stressed. It also protects against ventriculo-atrial regurgitation at high filling pressures.
The regurgitation which occurs in the absence of the pericardium may in part help to explain the supposed descending limb of the classically conceived Starling curve.

REFERENCES

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