

## Function of the Normal Pericardium

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**Summary:** Until recently, instrumenting the pericardium was possible only when a pericardial effusion is present or by surgical exposure of the pericardium. Techniques are now being developed to instrument the normal pericardium. This development will allow clinicians and investigators to study pericardial fluid in health and in a variety of disorders not associated with pericardial effusion. It will also be possible to improve our knowledge of pericardial pressure and the pericardial restraints on the heart.

**Key words:** pericardial physiology, transmural pressure, contact pressure

### Introduction

What I have to say is tangential to the main thrust of this meeting which will concern delivery of agents to the myocardium using the pericardial space as a depot. I think, however, that the opportunity to enter the pericardial space that does not contain and has never contained a pericardial effusion offers a unique opportunity for better understanding of the function of the normal pericardium.

In spite of much research, many questions about the function of the pericardium regarding restraint of cardiac volume and enhancing interaction between the two sides of the heart remain unanswered.

### Background

Until recently, physiologists did not doubt that pericardial and pleural pressures are essentially the same. Thus, both were regarded as intrathoracic pressure, so either one could be used to calculate ventricular transmural pressure, the only difference between the two pressures being the greater deflections relating to the cardiac cycle seen in pericardial pressure tracings. Of particular importance, earlier investigators found that inspiration lowered pericardial pressure almost as much as it did pleural pressure.

We must now consider a seminal paper published many years ago. The investigators infused a liter of saline into the pericardium of dogs. What was observed would not then have been anticipated: the right atrial pressure increased from 3 or 4 millimeters of mercury to 20 millimeters of mercury and its morphology assumed a more dynamic waveform, with large X and Y descents, simulating constrictive pericarditis. A very similar increase in right ventricular diastolic pressure also occurred. The most important finding in the experiment, however, was that pericardial pressure increased almost as much as right atrial pressure and pleural pressure did not change at all. Thus, when one acutely distends the heart by means of a fluid challenge and uses conventional means to measure pericardial pressure with a catheter in the pericardial space, one observes an increase in pericardial pressure, because all biological tissues resist acute strain. The pericardium, however, is stiffer than the myocardium, so when the heart is distended, it engages the pericardium. With further loading of the heart, pericardial pressure rises progressively, so that the change of transmural cardiac pressure is really quite small and much less than the change in absolute, or luminal pressure. Because fluid challenge does not alter pleural pressure when one needs to determine the transmural pressure in ventricular chambers in acute volume overload states, one cannot use intrathoracic pressure but must use pericardial pressure because they would be substantially different. Preload is the transmural pressure, not the absolute pressure. When one wants to study the compliance of the left ventricle, which is critically important in the physiology of diastole, one needs to know the transmural pressure, and to know the transmural pressure one needs to know the pericardial pressure.

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## The Concept of Contact Force

Pulmonary physiologists state that a true pleural space does not exist because the two pleural layers are tightly apposed to each other, so that there is only a *potential* pleural space. It therefore becomes necessary to measure the so-called contact pressure, and that requires a different technique. The technique is to place an unstressed flat balloon in the pleural space and to measure the pressure in the balloon.

Computed tomography of the heart shows minimal pericardial space in normal subjects. It has been proposed that the pericardial space, like the pleural space, is only a potential space and that all classical measurements of pericardial pressure were basically wrong, because liquid pressure, not contact pressure, was measured. Liquid pressure is independent of wherever the catheter tip lies in the space in which pressure is being measured, and consequently is the same wherever the catheter tip happens to lie in the pericardial space. Contact pressure does not share these characteristics.

It has been suggested that the pericardium is like the pleura and that if one introduces a catheter tip into the pericardial space one produces an artifact and would measure a pressure that only exists because the pericardium has been invaded. Contact pressure would be the true constraining force. By analogy, consider, for instance, the contact pressure between the condyle of the femur and the head of the tibia which must be of great magnitude; yet, a needle in the knee joint attached to a transducer would yield only atmospheric pressure. Clearly there are differences between contact pressure and liquid pressure, and the question, of course, is which is relevant to cardiac physiology?

Experiments have been reported in which left ventricular diastolic volume was held constant and left ventricular diastolic pressure was measured before and immediately following pericardiectomy. Pericardial removal caused a substantial drop in left ventricular diastolic pressure, even though the chamber volume was not allowed to change. The difference in left ventricular diastolic pressure before and after pericardiectomy was thus a measure of the pericardial restraining pressure, hereafter termed the theoretical pericardial pressure. This theoretical pericardial pressure was very close to contact pressure measured by a flat balloon placed between the left ventricular wall and the pericardium and was substantially higher than the pericardial liquid pressure measured by conventional catheter technique. The question for the physiologist is which of these different pressures is relevant to normal and abnormal cardiac physiology.

## The Influence of Pericardial Effusion

Before the development of techniques to instrument the normal pericardial cavity, the only opportunity for clinical investigators to assess the pressure in a dry pericardial sac was to make the measurement after tapping a pericardial effusion. After pericardiocentesis, however, although the pericardium

no longer contains a significant volume of fluid, the pericardium cannot be considered normal, because it was stretched before the tap and it does not recoil after the tap. Pericardial contact pressure would therefore be less than normal.

In animal experiments performed after the pericardium has been aspirated as completely as possible, liquid pericardial pressure becomes markedly subatmospheric, but contact pressure is several millimeters of mercury higher than atmospheric pressure. These low pericardial pressures likely do not exist in normal healthy subjects in whom pericardial fluid is present. When as little as 20 ml of saline is added to the pericardial space, the difference between pressure measured by these different methods disappears. When considering this information, it must be recalled that the human pericardium can normally contain up to 50 milliliters of fluid.

## Transmural Cardiac Pressure

Not only is pericardial contact pressure measured from a balloon significantly higher than liquid pressure, it is also virtually the same as right atrial and right ventricular diastolic pressure, which would mean that these chambers operate at a near to zero transmural pressure, defying our concept of preload. How does the idea that contact pressure, not liquid pressure, is the true operating pressure explain why liquid pressure in the pericardium increases during volume overload?

## Regional Transmural Pressure

It has long been considered that a film of pericardial fluid serves to equalize gravitational forces around the heart, preventing regional differences in transmural pressures during acceleration and deceleration. This formulation does not apply to contact pressure. Furthermore, when two flat unstressed balloons are placed in different regions of the pericardium, their contact pressures are unequal and change differently during interventions.

## Conclusion

The ability to invade the normal pericardium opens opportunities to understand the role of the pericardium in health and in heart disease. Pericardial pressure can now be assessed in euolemic patients with or without cardiac dilation or hypertrophy, and in acute and chronic overload states. Measurements will be made without altering the preexisting pericardial fluid volume and will be made simultaneously with tiny balloons and by conventional catheter methods.

These advances should improve our understanding of the normal function of the pericardium and its influence when the heart is enlarged, and provide fresh insights into diastolic function of the heart.