Pericardial and cardiac pressure

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ASSESSMENT OF pericardial pressure is beset with a number of conceptual and technical obstacles. One of the most formidable of these is that the clinical or experimental conditions under which a specific investigation is carried out strongly influence pericardial volume and the compliance of pericardium, myocardium, and the cardiac chambers. Of perhaps equal importance, operational conditions alter juxtapericardial pressure through alterations in pulmonary volume and transpulmonary pressure. Finally, comparison of results of different studies of pericardial pressure or restraint depends on knowledge of at what point on the pressure-volume relationship data have been obtained, and whether myocardial, pericardial, or juxtapericardial compliance dominated.

The publication of a series of articles dealing with the concept of pericardial surface contact pressure therefore has important implications for normal pericardial physiology and the pathophysiology of acute and chronic cardiac enlargement, cardiac tamponade, and constrictive pericarditis. The concept that conventional measurement of pressure in a film of liquid between two serous membranes may give a falsely low estimate of the contact force between them originated in discussions of pleural mechanics. However, in 1960 Holt et al. measured pericardial pressure in dogs with the use of open-ended catheters and cylindrical and flat balloons filled with liquid. They, like subsequent investigators, found that a cylindrical balloon seriously overestimated pericardial pressure, whereas a flat balloon yielded a pericardial pressure slightly higher than the pressure measured directly from the end of a catheter. These investigators assumed that the true pressure was that yielded by the catheter, and introduced a correction factor for use by investigators employing flat balloons.

When Holt et al. sequentially subjected dogs to acute hemorrhage and plethora they measured substantial changes in intrapericardial pressure without change in intrapleural pressure, and concluded that cardiac transmural pressure cannot be estimated from intrapleural pressure. This result emphasized that under all except the most abnormal circumstances, true ventricular transmural pressure is the difference between ventricular end-diastolic and pericardial end-diastolic pressure and does not usually exceed 2 or 3 mm Hg. This last observation takes measurement of intrapericardial pressure in experimental studies to the limit of accuracy, and beyond it in most clinical studies. This limitation explains why investigators tend to emphasize the extremes of hypovolemia and hypervolemia, thereby making specific and important observations, but at the risk of obscuring normal physiology.

The argument for surface contact pressure as opposed to liquid pressure is that in normal cardiac chambers there must exist a static equilibrium whereby the pericardial pressure is equal to ventricular pressure minus ventricular transmural pressure. Liquid pressure is exerted in all directions, obeying Pascal's law, but surface pressure is the sum of liquid pressure and the force exerted by the heart and pericardium on each other.

It is argued that the pericardial space is a potential one, because the film of normal pericardial liquid is too thin to separate the layers from one another; thus, introducing a catheter tip into the pericardial space creates an isolated area of artificial separation of the pericardial layers such that the catheter lumen is not in continuity with the pericardial liquid and yields an intrapericardial pressure too low to be meaningful. Unfortunately we lack an imaging technique that has
sufficient resolution to measure the space between the pericardial layers throughout the cardiac cycle in normal subjects. However, in a recent study, Constantin-Nescu et al,* demonstrated, with color video images made after intrapericardial injection of green dye, that fluid accumulated in the atrioventricular and interventricular grooves, but not over the ventricular surfaces. Thus it seems that the answer may be complex; the pericardial space is liquid filled over the grooves but potential over the lateral walls of the ventricles.

In canine experiments designed to determine how pericardial pressure should be measured, pericardial pressure in the control state was zero or subatmospheric when measured directly from the catheter, but close to right atrial and right ventricular diastolic pressure when measured from a flat balloon.1–4 The pericardial pressure that was deemed normal was obtained by balloon in open-chest dogs immediately after implantation of mechanical devices and after application of pericardial suction, which would have unduly emptied the pericardium, exaggerating the difference between liquid and contact pressures. Subsequent observations were made after intravenous infusions to maintain left ventricular end-diastolic pressure around 20 mm Hg, thereby creating acute rapid cardiac distension against the relatively noncompliant, recently incised and sutured pericardium, which would tend to exaggerate the effects of cardiac dilatation on pericardial pressure.

Consequences when pericardial pressure can be measured directly from a catheter. There are several important consequences when the pressure measured at the end of a catheter in the pericardium is physiologically relevant. Since intrapericardial pressure measured by this technique is negative, significant transmural pressure would normally exist across the walls of cardiac chambers. Loss of this normal transmural pressure, as for instance occurs in cardiac tamponade, unloads the heart, creating important hemodynamic impairment. Furthermore, except for hydrostatic differences, pressure would be identical throughout the pericardial space save for local variations associated with the activity of adjacent cardiac chambers. Overall, the liquid that separates the pericardial surfaces would maintain equal gravitational force over the heart during acceleration. In addition, pericardial pressure measured directly from a catheter tip would be close enough to pleural pressure measured by cannula to allow substitution of pleural or esophageal pressure for true intrapericardial pressure.

Unfortunately, adequate measurements of intrapericardial pressure, especially in human subjects, are scanty. For precise intrapericardial pressure measurement with an open catheter and an external transducer, the catheter tip can be placed anywhere within the pericardial space, but the zero reference for the external transducer must be exactly level with the tip of the intrapericardial catheter as ascertained by biplane fluoroscopy. If a solid state device such as a Millar, or Camino catheter is used, the weight of the column of pericardial fluid must be taken into consideration, because it cannot be corrected for in the same way as is done with external transducers.

Consequences when contact pressure is assumed to be close to true pericardial pressure. The literature expounding the importance of surface pressure has emphasized that pericardial pressure and right atrial or right ventricular diastolic pressure are the same, or almost the same, rendering transmural right heart pressure zero or almost negligible over a substantial range for right heart volumes. Consequently, assuming that major differences in contact pressure over the two sides of the heart do not ordinarily exist, right atrial pressure can be substituted for intrapericardial pressure to calculate left ventricular preload and diastolic compliance. However, recent studies have indicated that regional differences in pericardial contact pressure can be measured, particularly after massive selective distention of one side of the heart,5 and that right ventricular transmural pressure is uniformly present.8,9 Furthermore, contact pressure should vary with the degree of fit between the contacting layers, and could therefore be very different when the angle of curvature is sharp, as at the cardiac apex compared with the relatively flat ventricular surfaces. Finally, it is important to appreciate that pressure on a flat balloon may, in any ratio, be exerted either by the heart or the pericardium.

Physiologic interpretation. The question for normal physiology is whether we should consider the pericardium to exceed the normal diastolic heart in volume and to contain sufficient liquid to keep its cavity open, or consider the heart surrounded by a stiff closely but variably fitting membrane that, as though it were a series of rubber bands, must be actively stretched from systole to diastole, creating varying contact pressure throughout the cardiac cycle, and ultimately end-diastolic surface contact pressure such that right-sided and end-diastolic transmural pressure becomes negligible or zero.

Technical limitations. It is impossible to place a pressure-measuring device within the pericardial space without altering intrapericardial pressure. The chest or

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the abdomen and diaphragm may be opened, a needle or cannula may be passed from the exterior or via puncture of a cardiac chamber or great vessel, and fluid may be inadvertently added or allowed to escape from the pericardial cavity. If the pericardial space is indeed only potential, an appropriate location in which to situate a catheter tip or solid-state transducer does not exist. On the other hand, it is equally impossible to place noninvasively a balloon or balloons in the pericardial space that would conform perfectly to the surfaces of the visceral and parietal pericardium. Investigators who have used gas- rather than liquid-filled balloons report lower values for pericardial surface pressure, a finding that reemphasizes that we still have much to learn about surface contact forces. Neither the lumen nor the balloon technique eliminates the effects of the weight of the heart, fluctuations related to the respiratory and cardiac cycles, or regional cardiac activity.

In dogs studied with conventional pressure-measuring devices, pleural and pericardial pressures remain equal throughout the respiratory cycle distension, and when the subject performs the Valsalva or Mueller maneuver, findings are at variance with those reported from studies of pericardial pressure measured by a flat balloon. Liquid rather than contact pressure has been measured to demonstrate that external force on the surface of the heart remains constant up to acceleration of 7 g. Before concluding that only surface contact pressure is relevant to cardiac restraint, it must be recalled that a sharp rise in intrapericardial pressure resulting from acute cardiac dilatation can be demonstrated equally well with either pressure-measuring technique.

**Pericardial pressure in the presence of pericardial fluid.** When approximately 30 ml or more of pericardial fluid is present in a canine pericardial cavity, contact pressure, catheter tip pressure, and theoretical pressure in the pericardial space become essentially equal. Protagonists of the importance of surface contact pressure emphasize the differences in pericardial pressure obtained on the one hand via a catheter tip, or on the other hand by measurement from a balloon or calculation from the static equilibrium. With the pericardium empty, this difference may amount to 8 to 10 mm Hg in a normal dog, whereas once the critical 30 ml content of pericardial fluid has been achieved, the difference becomes negligible. However, the curves are far from parallel, but converge rapidly from the point of their widest separation when the pericardium is suctioned empty, to their convergence when the critical pericardial fluid volume is established. A more pragmatic approach is to add 5 or 10 ml of fluid to the pericardial space before making observations that depend on accurate measurement of pericardial pressure. This solution is particularly relevant to normal physiology, since neither right atrial nor pericardial pressure exceeds 2 or 3 mm Hg, and therefore, while the percent difference between transmural and cavity pressures may appear significant, important absolute differences do not exist.

**The pericardium restraints acute cardiac distension.** A uniform finding when total cardiac volume is immediately increased by rapid infusion is that much of the increased ventricular diastolic pressure is borne by the pericardium, with the result that minimal change in transmural ventricular diastolic pressure takes place. This observation goes far to explain shifts in the entire ventricular diastolic pressure-volume relationship observed during acute alteration of preload. The same phenomenon may also be observed in subacute volume overload such as that produced by right ventricular infarction, carcinoid of the tricuspid valve, and creation of an aortocaval fistula in experimental animals.

**The pericardium in chronic heart failure.** When dogs with surgically induced aortocaval fistula are followed for several weeks or months, the restraining effect of the pericardium and cardiac dimension can no longer be demonstrated, because the pericardium hypertrophies and its compliance increases. Nevertheless, evidence exists that under appropriate circumstances, the pericardium may limit the dimension or increase the diastolic pressure of the ventricles in patients with heart failure. The most compelling evidence is that interventions that create an immediate drastic decrease in preload shift the ventricular pressure volume relationship downward on its pressure axis, a finding highly consistent with the hypothesis that diminished preload acts to free the heart from restraint by the pericardium and cardiac fossa.

Similarities between the hemodynamics of constrictive pericarditis and right heart failure have prompted some investigators to assign a prominent role to the pericardium in the hemodynamics of heart failure. The principal findings shared by the two conditions are absence of an inspiratory decrease in mean right atrial pressure, the development of a deep y descent of atrial pressure that deepens and steepens during inspiration, and equal diastolic pressure recorded from the left and right atria, either throughout the respiratory cycle, or confined to the inspiratory phase. Inherent to this concept is that in these cases, intrapericardial pressure is greatly elevated, being the same as the increased ventricular diastolic pressure and would require the insertion of a flat balloon for measurement.

Exploiting the observation that right atrial pressure
and pericardial contact pressure measured by balloon are equal or close, it has been proposed that in chronic congestive heart failure, right ventricular diastolic pressure and intrapericardial pressure are elevated and equal, resulting in the absence of a diastolic pressure difference across the right ventricular wall. Left ventricular diastolic pressure is also elevated, but owing to the lesser compliance of the left ventricle, its diastolic pressure commonly exceeds that of the right ventricle and the postulated intrapericardial pressure. Working from this framework, it has been further suggested that when an inspiratory drop in left and right ventricular diastolic pressure to a common level occurs, the left ventricle is transiently underfilled, allowing its diastolic pressures to equilibrate with the pressure common to the right ventricle and pericardium.

**Cardiac tamponade.** Clinicians and investigators have become increasingly cognizant of a wide spectrum of cardiac tamponade. We now expect elevation of intrapericardial and both ventricular diastolic pressures to a common level and pulsat paradoxx only in the absence of preexisting cardiac dilatation or hypertrophy or pericardial scarring. When these conditions are met in severe cardiac tamponade and pericardiocentesis is performed, the intrapericardial and the two ventricular diastolic pressures fall but remain equal, while cardiac output and arterial blood pressure increase and pulsus paradoxus decreases. This result is obtained when pericardial pressure is measured by an open catheter tip or by a flat balloon. After a continuous fall in right atrial pressure during pericardiocentesis, a pericardial fluid volume is arrived at when right atrial pressure remains constant in spite of removal of further quantities of pericardial fluid. At this volume, pericardial pressure measured by a catheter continues to fall, eventually becoming subatmospheric, but intrapericardial pressure measured by flat balloon, in contrast, remains equal to right atrial pressure, even though relief of severe distension of the pericardium cannot have reduced its surface area to normal.

If an open catheter in the pericardial space yields true pericardial pressure from the onset of pericardiocentesis to the appearance of negative pericardial pressure, the hemodynamics of cardiac tamponade and its relief by pericardiocentesis are explained by unphysiologic abolition of zero right atrial transmural pressure created by increased intrapericardial pressure that is exactly matched by increased right atrial pressure. The higher the common right atrial and pericardial pressure, the more severe the tamponade, but tamponade exists to some degree until pericardial pressure falls below that in the right atrium, and therefore no longer directly determines right atrial pressure. This explanation is based on the view, for which considerable experimental evidence exists, that diminished left ventricular volume in cardiac tamponade results primarily from reduced pulmonary venous return secondary to atrial and right ventricular compression, and that the thick-walled left ventricle is not significantly compressed by pericardial fluid.

If a flat balloon yields true pericardial pressure, a radically different explanation is needed, because a major consequence of the concept of surface contact pressure is that right heart diastolic transmural pressure is normally close to zero. It is then proposed that pericardiocentesis progressively increases left ventricular transmural diastolic pressure; in other words it progressively relieves direct compression of the left ventricle until the normal pericardial and right atrial common pressure is obtained. Here, it should be recognized that this normal common pressure considerably exceeds liquid pericardial pressure in mild and low-pressure cardiac tamponade, and that the pericardial cavity immediately after relief of tamponade may be too lax to forcefully contact the heart.

In conclusion, the present state of knowledge does not permit a simple unified answer to the question of the nature of pericardial pressure. Liquid-filled balloons probably exaggerate surface contact pressure, and the studies of Santamore and Sinker and their colleagues disprove the concept of zero right ventricular transmural pressure. Gas-filled balloons yield lower pericardial pressures than liquid-filled balloons and may be more accurate. The pericardium immediately after pericardiocentesis may not regain its normal size and contact, so that caution is necessary before extrapolating from postpericardiocentesis pericardial dynamics to the normal situation. The evidence that acute cardiac distension causes the heart to engage the pericardium and raise pericardial pressure is clear, and should be accepted. The evidence that the same phenomenon occurs in chronic heart failure is more indirect and is not elicited in all cases, being perhaps a manifestation of acute exacerbation of chronic cardiac enlargement. In normal subjects surface contact and liquid pressures may exist side by side, but since normal right atrial pressure is only 3 to 5 mm Hg, normal pericardial pressure is probably small, however it is measured.

**References**


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