

A novel technique for measurement of pericardial pressure

GWYNETH DEVRIES, DOUGLAS R. HAMILTON, HENK E. D. J. TER KEURS, RAFAEL BEYAR, AND JOHN V. TYBERG

Department of Medicine and Department of Physiology and Biophysics, University of Calgary, Calgary, Alberta, Canada T2N 4N1

Received 29 June 2000; accepted in final form 17 January 2001

deVries, Gwyneth, Douglas R. Hamilton, Henk E. D. J. Ter Keurs, Rafael Beyar, and John V. Tyberg. A novel technique for measurement of pericardial pressure. *Am J Physiol Heart Circ Physiol* 280: H2815–H2822, 2001.—To determine whether pericardial liquid pressure accurately measures pericardial constraint, we developed a technique in which a catheter was positioned perpendicular to the epicardial surface. This device, which occupies little or no pericardial space, couples the thin film of liquid to a transducer. In six open-chest dogs, we also measured left ventricular (LV) end-diastolic pressure (LVEDP) and anteroposterior and septum-to-free wall diameters. LVEDP was raised incrementally to ~25 mmHg by saline infusion. With the use of the product of the two diameters as an index of area (A_{LV}), LVEDP- A_{LV} relationships were obtained with the pericardium closed and again after the pericardium had been widely opened to obtain the isovolumic difference in LVEDP (Δ LVEDP). In all dogs, the technique yielded values of pericardial pressure equal to Δ LVEDP as well as equal to that measured using a previously placed balloon transducer in the same location and at the same A_{LV} . We conclude that, when the pressure of the pericardial liquid is appropriately measured, it (in addition to the balloon-measured contact stress) defines the diastolic constraining effect of the pericardium. Furthermore, we suggest that earlier measurements of pericardial “liquid pressure” were low, due to an artifact of measurement.

pericardium; mechanics; balloon; catheters; physiology

THE MEASUREMENT OF THE MAGNITUDE of pericardial constraint has been controversial because different techniques have yielded different pressures and no technique has escaped criticism. Early investigators (5, 6, 12, 22, 26, 30) appreciated that the mechanical effect of the pericardium was substantive, but they did not actually measure the pressure from the potential space between the heart and pericardium. The first widely quoted attempt to measure pressure in the pericardial liquid employed an end-hole catheter a few millimeters in diameter inserted between the heart and pericardium (21). Those investigators concluded that diastolic pericardial pressure was approximately equal to or just less than atmospheric pressure and that it did not vary

despite large changes in right atrial pressure, which generally have been interpreted to reflect large changes in cardiac volume.

As Tyberg et al. (35) originally proposed, the magnitude of diastolic pericardial constraint may be determined by comparing the shift in the left ventricular (LV) end-diastolic pressure (LVEDP)-volume relationships before and after pericardiectomy [i.e., pericardial pressure is equal to the isovolumic difference in LVEDP (Δ LVEDP) before and after removing the pericardium]. The logic of this strategy depends on the assumption that the stress-strain properties of the LV free wall are unchanged by removing the pericardium and that the right ventricular (RV) influence on the LV free wall stress-strain relationship is unaltered by removing the pericardium. In the intact human subject, an equivalent approach is to measure Δ LVEDP before and after pericardial effects have been eliminated by transient caval obstruction (20). Because this rationale for determining the true magnitude of pericardial constraint has been adopted by several investigators (3, 4, 16), we suggest that it provides an accepted measure of pericardial pressure.

Smiseth et al. (32) showed that a conventional end-hole catheter inserted between the heart and pericardium was adequate for measuring pericardial pressure only when excessive amounts (i.e., ≥ 30 ml) of pericardial fluid were present, because that method significantly underestimated Δ LVEDP. However, these investigators demonstrated that a small (3×3 cm) thin balloon transducer (14) placed within the pericardial space would give pressures equaling Δ LVEDP. Despite the findings of Smiseth et al. (32), the accuracy of the balloon transducer has been questioned (3, 4, 13), largely because it was assumed that the balloon would overestimate pressure because it was thicker than the normal pericardial space. Previous investigators (18, 25) have agreed that such devices should be flat and thin, but no agreement on critical dimension has been reached.

Similar concerns with device-induced distortion of the pleural space led Wiener-Kronisch et al. (36) to

Address for reprint requests and other correspondence: J. V. Tyberg, Professor of Medicine and Physiology and Biophysics, Univ. of Calgary Health Sciences Centre, 3330 Hospital Dr. NW, Calgary, Alberta, Canada T2N 4N1 (E-mail: jtyberg@ucalgary.ca).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

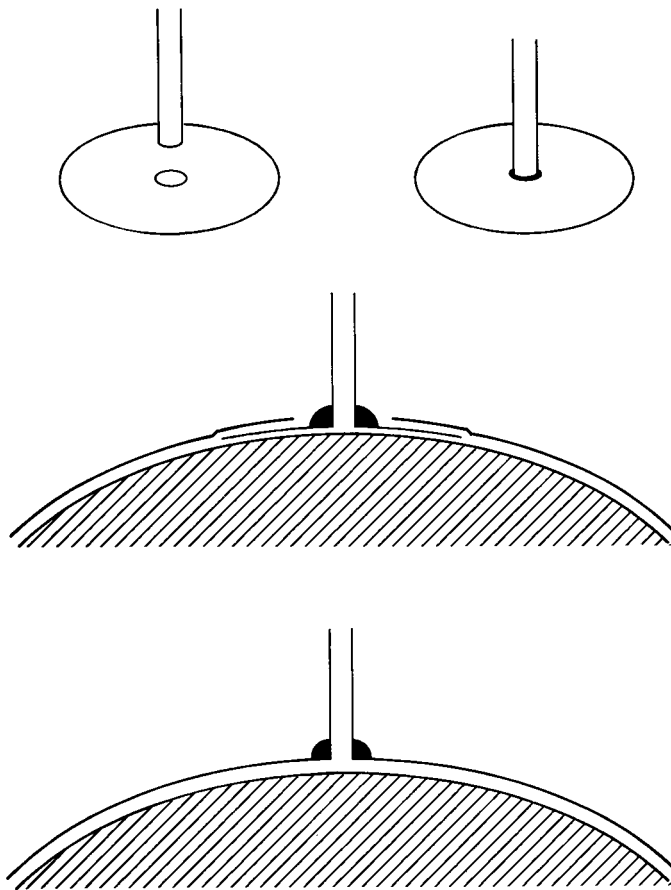


Fig. 1. *Top*: fabrication of the orthogonal catheter from tubing and a membrane; *middle*: the relation of this device to the pericardium and the left ventricular (LV) surface; *bottom*: relation of the extrapericardial modification of the catheter (fabrication not illustrated) to the pericardium. Both systems are connected to a pressure transducer and flushed with saline.

devise a simple method for measuring pressure. A needle was passed through a rib such that it communicated with the pleural fluid but did not actually enter the pleural space. The pressures measured by this so-called rib capsule were shown to be the same as the accepted measure of pleural pressure (i.e., the static recoil pressure of the lung).

With the use of a new technique fundamentally similar to the rib capsule, the purpose of this investigation was to determine whether pressure measured from a thin layer of pericardial liquid would be substantively equal to Δ LVEDP and to the pressure measured using a balloon transducer, not to reverify the balloon transducer measurements or to precisely define any small second-order differences between them and the measurements using the new technique.

MATERIALS AND METHODS

To allow access to the pericardial space with minimal distortion, we adapted a Silastic tube to enable it to stand perpendicular to the epicardial surface. A disc of thin (0.3 mm) flexible Silastic (compound AR131, Armet Industries; Ontario, Canada) rubber sheeting (diameter = 3 cm) with a 1-mm hole in the center was glued to the end of a small

Silastic tube (length = 8 cm, inner diameter = 1 mm) in such a way that the result resembled a thin flexible wheel at the extreme end of an axle (see Fig. 1). This tube was connected to a pressure transducer (model P23ID, Gould Statham Instruments; Oxnard, CA) using a 30-cm 8-Fr cardiac catheter.

To allow access to the pericardial space without introducing any extraneous material, an equivalent extrapericardial device was constructed. We modified the orthogonal catheter by trimming the disc to a diameter of 8 mm and glued it (Vetbond tissue adhesive 1469, Animal Care Products-3M; St. Paul, MN) to the pericardium of two dogs (Fig. 1, *bottom*). The hole in the catheter was fixed directly above a 2- to 3-mm hole in the pericardium, which had been cut previously.

In Vitro Study

A Plexiglas device adapted from Lai-Fook et al. (23) consisting of a reference chamber and a test space was used to compare different pressure measurements from liquid in the test space (Fig. 2). The airtight reference chamber could be accessed by a single port; pressure in the chamber could be altered and measured via that port. A thin Silastic rubber diaphragm separated the reference chamber from the test space, which was bounded on the other side by an immediately adjacent Plexiglas plate, thus defining a potential space. Twenty 1.6-mm holes in the Plexiglas plate allowed excess air or water in the test space to escape freely. The orthogonal catheter technique was simulated by sealing a Silastic tube into one of the holes in the Plexiglas plate such that no part of the tube actually entered the test space. To measure pressure in the test space by a method simulating the insertion of a catheter into the pericardial space (21, 32), a second 3-cm long catheter was inserted "tangentially" into the test space via a channel, its long axis being parallel to the surfaces of the membrane and the Plexiglas plate. The reference pressure was varied while recording pressure data from both the orthogonal and tangential catheters and the reference chamber. All pressures were monitored using liquid-filled catheters zeroed to a common level and connected to external transducers (model P23ID, Gould Statham Instruments). Conditioned signals (model VR16, Electronics for Medicine; White Plains, NY) were passed through antialiasing filters with low-pass cutoff frequencies of 100 Hz. The filtered signals were then digitized with 12-bit resolution at a

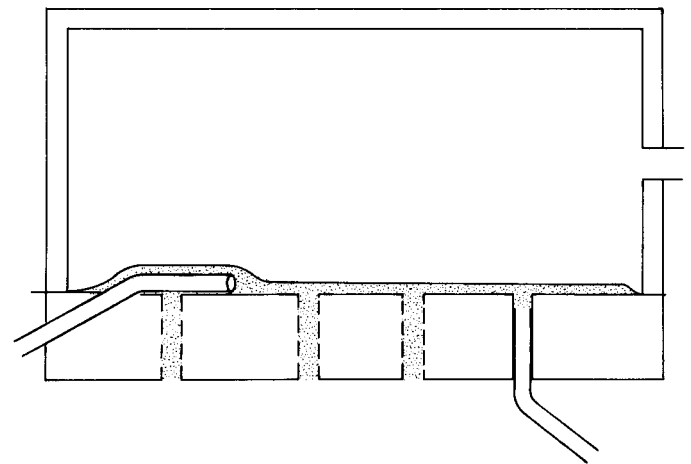


Fig. 2. Apparatus designed to compare the performance of a tangential catheter (*left*) to that of the orthogonal catheter (*right*). The pressurized reference chamber (*top*) is separated from the wet test chamber by a Silastic membrane.

sampling rate of 200 Hz using an IBM personal computer. Data acquisition and analysis were performed using CVSOFT (Odessa Computer Systems; Alberta, Canada).

In Vivo Studies

The experimental protocol was approved by the Animal Care Committee of the Faculty of Medicine. Mongrel dogs were premedicated with morphine (0.75–1.0 mg/kg im), followed by thiopental sodium (10–15 mg/kg iv). Anesthesia was maintained by a continuous infusion of fentanyl citrate (induction dose 50–75 mg/kg; maintenance dose 20–50 mg·kg⁻¹·h⁻¹). The animals were ventilated with oxygen and nitrous oxide (1:2) using a constant volume respirator (model 607, Harvard Apparatus; Millis, MA). Blood gas levels, pH, and body temperature were monitored. Aortic pressure was monitored using a liquid-filled catheter inserted into the right femoral artery. RV and LV pressures were measured with 8-F micromanometer-tipped catheters with reference lumens (Millar Instruments; Houston, TX) inserted through the right jugular vein and right carotid artery, respectively. Pressure measurements were referenced (zeroed) to the mid-LV level. Before each data acquisition interval, the pressure wave forms from the micromanometers were compared with those from their respective reference lumens. Any baseline shift in the micromanometer wave form was corrected by manipulation of a manual balance control. A large-bore catheter was inserted into the left jugular vein for fluid administration. A thoracotomy through the left fifth interspace was performed. A ~7-cm pericardial incision was made along the atrioventricular sulcus to allow placement of epicardial ultrasonic crystals for measurement of LV anteroposterior and septum-to-free wall diameters (Triton Technology; San Diego, CA) (32). A flat 3 × 3-cm liquid-filled balloon transducer (14, 32) was positioned in the pericardial space at a site over the LV free wall approximately halfway between the base and apex and in the same cardiac plane as the ultrasonic crystals. The balloon was loosely tethered to the epicardium. With the use of interrupted sutures, the pericardium was reapproximated to its original volume, with care being taken to avoid overlapping the edges and compromising the original pericardial volume (31); between the sutures, the edges remained gaping, and liquid could escape freely. Later, the balloon was removed, and a small 2- to 3-mm hole was cut in the pericardium at the site that previously overlaid the balloon; the location chosen was one free of underlying major epicardial coronary vessels and remote from the pericardial incision. The orthogonal catheter was introduced into the pericardial cavity, and the tube led out through the small hole; therefore, the disc portion was left lying flat between the heart and pericardium. If necessary, the hole created to allow passage of the orthogonal catheter was loosely sutured to minimize movement of the device. The tube was connected to a pressure transducer, and all air bubbles were purged from the system using saline flushes. Fluid introduced into the pericardial space by flushing dispersed immediately and escaped freely through the gaps between the cut edges of the pericardium. A pneumatic constrictor was positioned around the inferior vena cava to impede venous return temporarily and reduce LVEDP. Throughout the experiment, the chest retractor remained in place to maintain convenient access to the heart and to avoid destabilizing or kinking the orthogonal catheter.

Experimental protocol. Pressures, dimensions, and electrocardiograms were recorded during changes in cardiac size that were induced by caval occlusion, intravenous saline infusion, and withdrawal of blood as described below.

In six dogs, pressure recordings using the orthogonal catheter were obtained over a range of cardiac sizes and LVEDPs. To study the effect of flushing the catheter, two protocols were carried out. *Protocol A* (used in four dogs) involved flushing immediately before each data acquisition interval. *Protocol B* (used in two of the *protocol A* dogs and in two other dogs) involved intermittent flushing. In *protocol A*, LVEDP was raised by 5-mmHg increments from baseline values (0–5 mmHg) to peak values of 20–25 mmHg. The orthogonal catheter was flushed with saline immediately before each data acquisition; hemodynamic data were acquired for 15 s with the ventilator stopped at end expiration. In *protocol B*, LVEDP was raised through the same total range of pressure by a 2-min continuous saline infusion; the orthogonal catheter was flushed only once, just before the infusion. Data were acquired throughout the infusion while the dog was normally ventilated.

To determine transmural LVEDP as a function of LV volume and thereby calculate Δ LVEDP (32), the pericardium was opened widely and a pericardial cradle was constructed. Hemodynamic parameters were then measured over the course of volume loading and occlusion of the inferior vena cava. Monitoring LV dimensions ensured that they were comparable in pericardium-closed and pericardium-open states.

Data acquisition and analysis. For *protocol A*, end-diastolic data points from several normal cardiac cycles were selected and averaged. For *protocol B*, end-diastolic points that corresponded to end expiration were selected. End diastole was defined as the relative minimum of LV pressure after atrial systole just preceding the rapid increase in LV pressure. If this pressure was difficult to discern, the peak of the R wave of the electrocardiogram was used as a temporal estimate of end diastole. As a surrogate for LV volume, an index of LV area (A_{LV}) was calculated as the product of the LV anteroposterior and septum-to-free wall diameters.

To obtain Δ LVEDP, cubic curves were fitted to LVEDP- A_{LV} data points collected after the pericardium had been opened. (The introduction of the cubic term significantly reduced the amount of unexplained variance in each animal's data.) Thus, for a given A_{LV} , there were two corresponding LV intracavitary pressures: one recorded when the pericardium was closed and another (found by interpolation using the fitted curve) recorded after the pericardium had been widely opened. Subtraction of the pericardium-open pressure from the pericardium-closed pressure yielded Δ LVEDP at that A_{LV} . (In *dog 6*, Δ LVEDP was calculated with respect to the LV septum-to-free wall diameter only because the anteroposterior diameter signal was unsatisfactory.)

Statistical Methods

Least squares linear regression was used to correlate measured pericardial pressures to calculated values. Differences in slope and intercept obtained between groups were analyzed by one-way analysis of variance (ANOVA) and Student-Newman-Keuls test for multiple comparisons between groups. A value of $P < 0.05$ was considered significant.

RESULTS

In Vitro Study

Pressures measured by the orthogonal catheter or tangential catheter were plotted against the pressure recorded from the reference chamber. Figure 3 shows that pressures recorded using the orthogonal catheter

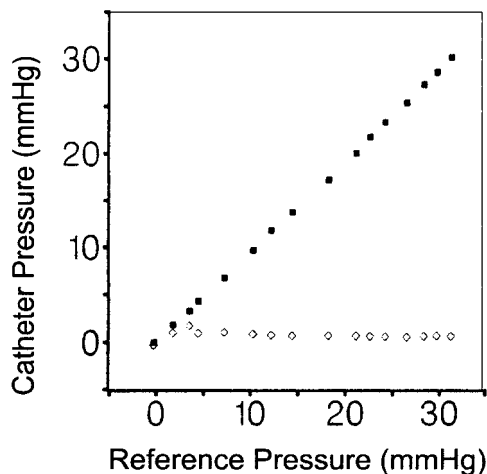


Fig. 3. Pressures recorded using the orthogonal catheter (■) and the tangential catheter (◇) plotted against the reference pressure (see Fig. 2). Only the orthogonal catheter measured pressure accurately.

were identical to the reference pressure over the given range. Pressures measured using the tangential catheter were independent of the reference pressure.

In Vivo Studies

The effect of flushing the orthogonal catheter system was investigated by using two slightly different protocols. No difference was observed in data collected 2 min after flushing (*protocol B*) compared with the data collected within several seconds of flushing (*protocol A*), and so the data were combined for subsequent analyses.

The orthogonal catheter, when positioned in the pericardial space as described, gives a characteristic wave form during the cardiac cycle (see Fig. 4). In general, the wave form parallels LV intracavitary pressure during diastole and falls quite sharply after the beginning of ventricular ejection. Although our experience with this device is not nearly so great as with the balloon transducer and although rigorous comparisons have not been made, the systolic decrease in pressure

Table 1. Pericardial pressure correlations: orthogonal catheter and balloon transducer vs. Δ LVEDP

Dog	Orthogonal Catheter			Balloon Transducer		
	<i>m</i>	<i>b</i>	<i>r</i> ²	<i>m</i>	<i>b</i>	<i>r</i> ²
1	0.78	-0.42	0.99‡	0.77	2.79	0.94‡
2	0.7	2.26	0.85*	1.05	-2.29	0.99†
3	0.96	0.16	0.99‡	0.94	0.84	0.99‡
4	0.96	1.65	0.98‡			
5	0.94	0.99	0.94‡	0.94	1.93	0.98‡
6	0.9	0.23	0.98‡	1.04	-1.46	0.99‡
mean	0.87	0.81		0.94	0.36	
SE	0.05	0.45		0.09	1.09	

m, Slope; *b*, y-intercept; *r*, correlation coefficient; Δ LVEDP, isovolumetric difference in left ventricular pressure. **P* < 0.05; †*P* < 0.005; ‡*P* < 0.001.

appears somewhat more prominent than when using the balloon. Depending on the volume-loading state, decreases of 5–25 mmHg were seen during LV ejection. The wave form measured using the extrapericardial orthogonal catheter (data not shown) was not different.

For each dog, pericardial pressures measured using the orthogonal catheter and the balloon transducer were correlated with Δ LVEDP calculated at the respective A_{LV} (Table 1). Figure 5 (*top*) shows the pooled orthogonal catheter data and the pooled LV balloon transducer data, both sets of data plotted against Δ LVEDP. There was no statistically significant difference in the pericardial pressures measured using the two methods. Figure 5 (*bottom*) shows data from the extrapericardial orthogonal catheter. There was no statistically significant difference in the regression coefficients obtained from data using either orthogonal catheter method.

DISCUSSION

Because it is still not clear whether the pressure of the pericardial fluid accurately reflects pericardial constraint to diastolic filling, we developed a novel “orthogonal catheter” technique by which the long axis of a liquid-filled catheter is positioned perpendicular to

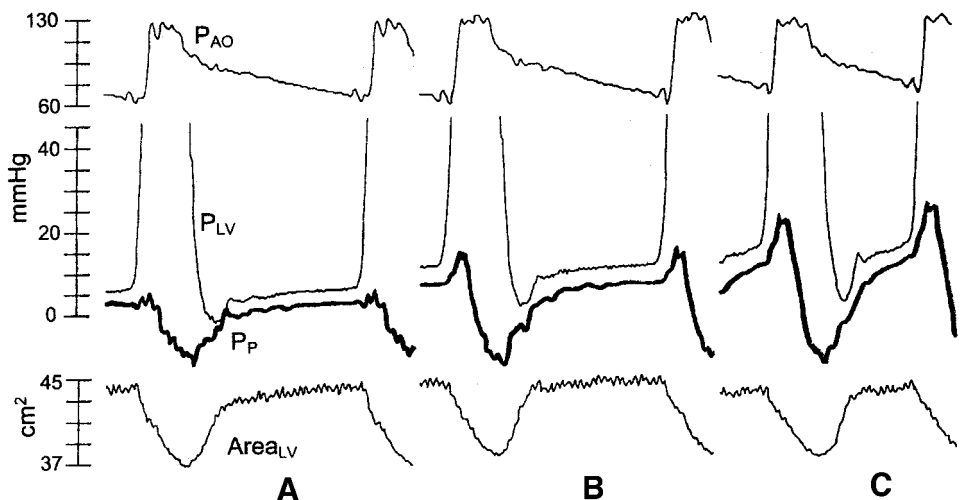


Fig. 4. Pericardial pressure (P_p) recorded over the LV free wall using an orthogonal catheter. Aortic pressure (P_{AO}), LV pressure (P_{LV}), and LV area ($Area_{LV}$; as an index of LV volume) are shown for comparison. A–C: results of progressive volume loading.

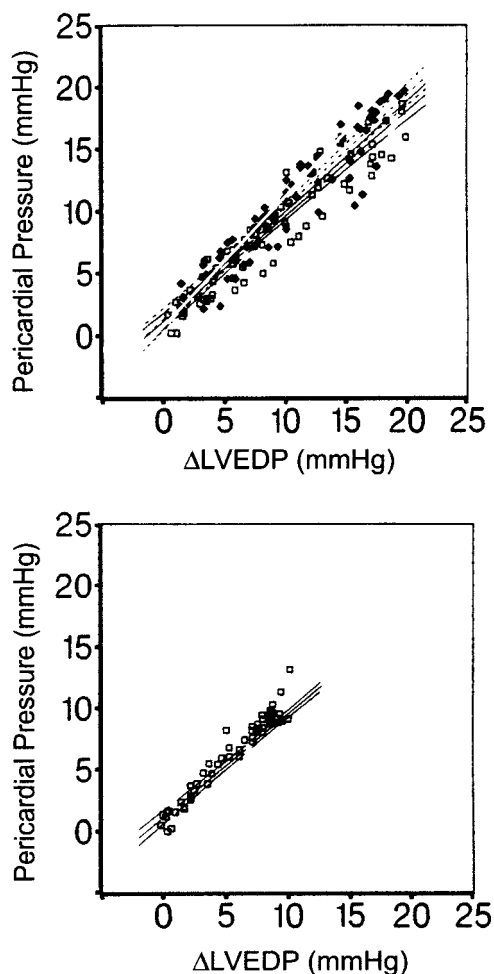


Fig. 5. *Top*: comparison of pericardial pressure measured by the orthogonal catheter (\square and solid lines; pooled data from 6 dogs) and the balloon transducer (\blacklozenge and dashed lines; data from 5 dogs), both plotted against the isovolumetric difference in LV end-diastolic pressure (Δ LVEDP). The 95% confidence intervals for the regression lines are shown; they overlap over the whole range of observations. *Bottom*: pressure recorded using the extrapericardial orthogonal catheter (\square) glued to the outside of the pericardium plotted against Δ LVEDP. Data were from a single dog. The regression line and its 95% confidence intervals are shown (solid lines). There was no statistically significant difference in the regression coefficients obtained by these two methods.

the epicardial surface of the heart, thereby coupling a pressure transducer to the pericardial liquid while only minimally changing the normal heart-pericardium relation. Its low-profile construction should introduce no more than negligible LaPlacian stresses on the pericardium, and, in its extrapericardial modification, such stresses should be completely absent. Therefore, neither modification is likely to alter pericardial pressure. The technique provides a direct measurement of pressure from the pericardial liquid layer that, in magnitude, approximates the theoretical value of pericardial pressure Δ LVEDP. It also approximates the magnitude of pericardial pressure measured by the balloon transducer (14); both are much greater than the pressure measured by a liquid-filled tangential catheter lying within the pericardial space (32).

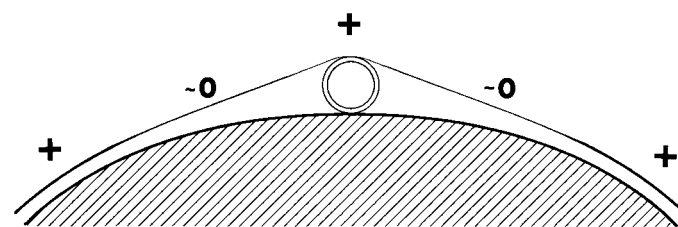


Fig. 6. A schematic diagram showing that a tangential catheter that is large relative to the thickness of the pericardial space produces a "tented" region of pericardium, over which the curvature (~ 0) must be substantially less than that over the heart (+, left and right). For simplicity, we assumed that the chest is open, that the lungs are retracted, and that the catheter lies in a horizontal plane. If surface properties produce a region of negative curvature (not shown) at the "base" of the tent, the curvature must then be zero between regions of positive and negative curvature. Thus the Law of LaPlace dictates that pericardial pressures recorded using a large tangential catheter must be lower than that over the heart or, indeed, zero.

In the *in vitro* study, the orthogonal catheter faithfully recorded the reference chamber pressure, just as Lai-Fook et al. (23) observed during the development of the rib capsule technique that we emulated. We also found that the tangential catheter could not be used to assess reference chamber pressure. In 1969, McMahon et al. (25) demonstrated that an end-hole cannula inserted into a model of the pleural space would underestimate the reference pressure and concluded that an appropriate sensor to measure pleural pressure should be thin and flat. On the basis of experiments using a plastic model of the pleural space, Lai-Fook et al. (23) also discounted the technique of using tangential catheters to measure pressure directly from liquid in a thin space. They concluded that when the diameter of a tangential catheter was much greater than the thickness of the space, it distorted the space and caused a liquid column to develop along the catheter. Because such a column would not be able to maintain a pressure difference (i.e., fluid would move freely), it follows that the pressure measured at the end of the catheter would have to be the same as that prevailing proximally along the catheter and not the pressure borne by a lubricant film. These concepts are supported by the present *in vitro* results and are consistent with the findings of Smiseth et al. (32): that the tangential catheter underestimated pericardial pressure when only small (i.e., normal) amounts of fluid were present in the pericardial space of a dog.

These findings can also be supported theoretically (24). When a tangential catheter that is large relative to the thickness of the space lies between the pericardium and the ventricular epicardium, the pericardium must become "tented" along the side of the catheter (see Fig. 6). The curvature of the tented pericardium would, necessarily, be less than that of the pericardium overlying the ventricle and may approach zero (i.e., this region might be flat). Because the pericardial curvature over the ventricle would be associated with the appropriate pericardial pressure, the pressure in the tented region would be less and probably negligible. Furthermore, if surface properties cause the curvature

of the pericardium at the “bottom” of the tent to become negative, the curvature of the “wall” of the tented region would indeed be zero, because curvature must be zero between regions of positive and negative curvatures. If curvature is zero at any point, transmural pressure must be zero, according to the Law of LaPlace. Only if the tangential catheter is sufficiently small enough to produce no distortion will the curvature be uniformly positive and, therefore, pericardial transmural pressures will be positive at every location over the heart; such a tangential catheter should behave as well as the orthogonal catheter and register the correct local pressure. However, when the pericardial fluid volume is increased sufficiently [in dogs, more than 30 ml (32)], even a ordinary tangential catheter will float freely in the liquid and register the correct pressure that will be uniform throughout, consistent with a pericardial curvature that is presumably uniform except for local external constraint.

The effect of flushing the orthogonal catheter system was investigated by using two slightly different protocols, which revealed no difference. This suggests that the flushing volume was quickly distributed away from the catheter and, ultimately, out of the pericardial space through gaps in the pericardium, leaving only a lubricant film. This lubricant film appeared to be maintained for periods of a few minutes, although the necessity of intermittently reflushing and reestablishing this liquid continuity made using these devices less reliable and convenient than the pericardial balloon.

Each of the two versions of the orthogonal catheter have advantages and disadvantages. The original version requires less surgical dexterity but does involve the introduction of an extraneous device. Because it is noninvasive, the extrapericardial version would seem to be theoretically superior, but the application of glue could have stiffened the pericardium and, thereby, altered its internal surface. Given that comparable results were obtained with both modifications and that those results were equal to Δ LVEDP and to the results of balloon measurements, we conclude that neither the presence of the Silastic disc within the pericardial space nor the application of glue had any measurable effect.

The typical pressure wave form recorded using the orthogonal catheter was similar to that recorded using a pericardial balloon except that the decrease in pressure during systolic ejection was somewhat larger. This decrease seemed to be related to instantaneous LV volume in that pressure fell rapidly during ejection. The minimum systolic pericardial pressures varied from dog to dog, from near 0 mmHg in some to as low as -10 mmHg in others. The sole objective of this investigation was to evaluate these devices in terms of their ability to measure end-diastolic pericardial pressure, and the dynamic systolic performance of the orthogonal catheter requires further study. We (14) have recently shown that the pericardial balloon transducer spatially integrates over its 9-cm² surface very well. That is, when known stresses are applied over as little as 25% of its surface, the balloon records a pres-

sure equal to the correct average stress. The orthogonal catheter may reflect the pressure from a much smaller area and so might provide information about focal pericardial pressures that cannot be detected using the balloon. With the use of other techniques, it has been shown that pericardial pressure varies at different locations (15, 17, 33), but this device might measure pressures with even more spatial precision.

Because our technique measures pressure from a thin fluid film, it may be useful to consider this fluid as a lubricant, as Santamore et al. (29) have done. Similar to synovial joints in which moving surfaces may be separated by a thin fluid film at different stages of stance and walking (10, 11, 37), the heart and pericardium might be viewed as a load-bearing system in which deformable epicardial and pericardial sliding surfaces are separated by a lubricant. Several specific theoretical subtypes of lubrication may apply to the pericardial system. Broadly speaking, “fluid-film” lubrication occurs when there is a thin film of lubricant between the sliding surfaces. The load on the bearing is supported by a pressure in this fluid film (7, 9, 19, 27). A subset of fluid-film lubrication is “squeeze-film” lubrication, which pertains when two surfaces approach each other perpendicularly. As the surfaces become closely opposed, a pressure is generated in the fluid that is sufficient to support high loads for short durations. Squeeze-film lubrication may describe the establishment of relatively high pressure in the fluid between the opposing epicardial and pericardial surfaces for a brief moment at end diastole but, because the pericardium obviously slides over the epicardial surface, “elastohydrodynamic lubrication,” in which the deformability of relatively soft sliding surfaces results in a longer-lasting lubricant film that enhances load-carrying capacity (9), may be more relevant to explain the persistence of the lubricant effect in the pericardium. This concept has been extended more recently to “microelastohydrodynamic lubrication,” which applies to soft layers with “rough” surfaces whose roughness is small (10, 11). A final possibility is some form of boundary lubrication (37), in which surface-active lipids accumulate on the opposing surfaces (perhaps in combination with a thin film of interstitial fluid) and provide easy movement of one surface relative to the other. However, before the mode of lubrication can be identified, the composition of pericardial fluid needs to be defined in addition to the pressure distribution over the epicardial surface and the relative speeds of the two surfaces.

Although the techniques used in this study do not provide the basis of a detailed understanding of the physics of the lubricated interface between the heart and the pericardium, our results led us to some tentative interpretations. First, because the saline could escape freely from the pericardium and because others (29) have observed substantial convective movement of pericardial liquid, we believe that the flushing volume of saline dispersed immediately and the orthogonal catheter simply became hydraulically coupled to the thin layer of fluid normally present. (Corollary: we do

not believe that the flushing liquid remained loculated in the region of the catheter, thus simulating the balloon transducer.) Second, we believe that the pressure that we measured is a true local pressure in the sense of being “locally scalar” and obeying Pascal’s Law. That is, we are measuring the local hydrostatic component of stress over a representative region of the LV. However, we do not believe that a simple gravitational gradient in absolute pressure would be found; Lai-Fook et al. (23) were unable to demonstrate such a gradient for the pleura. If the previous interpretations are correct, this leaves the question of how a pressure gradient can exist between, for example, the region over the ventricular free walls where end-diastolic pericardial pressure has been found to exceed 20 mmHg (34) and the large spaces within the pericardium where pressure is near zero (21). In agreement with Santamore et al. (29), we conclude that this pressure gradient would tend to diminish after some long time, a time that is very substantial compared with the duration of the cardiac cycle. Therefore, that this pressure is relatively high and that it has been shown to be equal to that measured using a balloon transducer is only due to the viscosity of the liquid and the fact that the layer is extremely thin. Because much of the liquid must be loculated in the large spaces (e.g., over the atrioventricular groove and between the great vessels), its average thickness over the ventricles is probably much less than 0.34 mm (the average thickness found by dividing the volume of liquid by the total pericardial surface area) (29). Thus its thickness is potentially similar to the 20- μ m thickness that was estimated for the pleural space (23).

Finally, the terminology in this field of study should be addressed. The concepts of “liquid pressure” and “surface pressure” were developed by Agostoni and others (1, 2, 8, 18, 28) and were based on an assumed microscopic structure of the pleural interface. Smiseth et al. (32) adopted the terms, equating surface pressure with the pericardial pressure measured by a balloon transducer inserted into the pericardial space and liquid pressure with that measured via a tangential catheter. In that we have shown here that the pressure of the thin film of pericardial liquid is equal to that measured using the balloon, we suggest that the liquid pressure-to-surface pressure distinction (24) is unnecessary and may be inappropriate, at least with respect to pericardial pressure.

In conclusion, we measured a pressure directly from the pericardial liquid that is equal to Δ LVEDP (the theoretical value of pericardial pressure) and to the pressure measured by a balloon transducer. This new method of measuring pericardial pressure is analogous to methods used to measure pleural pressure.

We thank Cheryl Meek and Gerald Groves for excellent technical assistance and Rozsa Sas for preparing the illustrations. We also thank Drs. Nigel Shrive and William Whitelaw for helpful criticism.

H. E. D. J. ter Keurs and J. V. Tyberg are Heritage Medical Scientists of the Alberta Heritage Foundation for Medical Research (AHFMR). R. Beyar, on sabbatical leave from Technion (Haifa, Israel), was a Visiting Scientist of the Medical Research Council of

Canada (Ottawa, Canada) and the AHFMR and was also supported in part by donations from Alvin and Mona Libin and Ted and Lola Rozsa. This study was supported by a Heart and Stroke Foundation of Alberta (Calgary, Canada) grant-in-aid (to J. V. Tyberg).

REFERENCES

1. **Agostoni E.** Mechanics of the pleural space. In: *Handbook of Physiology. The Respiratory System. Mechanics of Breathing.* Bethesda, MD: Am. Physiol. Soc., 1986, sect. 3, vol. III, pt. 2, chapt. 30, p. 531–560.
2. **Agostoni E and D’Angelo E.** Thickness and pressure of the pleural liquid at various heights and with various hydrothoraces. *Respir Physiol* 6: 330–342, 1968.
3. **Applegate RJ, Santamore WP, Klopfenstein HS, and Little WC.** External pressure of undisturbed left ventricle. *Am J Physiol Heart Circ Physiol* 258: H1079–H1086, 1990.
4. **Assanelli D, Lew WYW, Shabetai R, and LeWinter MM.** Influence of the pericardium on right and left ventricular filling in the dog. *J Appl Physiol* 63: 1025–1032, 1987.
5. **Barnard HL.** The functions of the pericardium. *J Physiol (Lond)* 22: 43–47, 1898.
6. **Berglund E, Sarnoff SJ, and Isaacs JP.** Ventricular function: role of the pericardium in regulation of cardiovascular hemodynamics. *Circ Res* 3: 133–139, 1955.
7. **Bray R, Frank C, and Miniaci A.** Structure and function of diarthrodial joints. In: *Operative Arthroscopy.* New York: Raven, 1991, p. 79–116.
8. **D’Angelo E, Bonanni MV, Michelini S, and Agostoni E.** Topography of the pleural surface pressure in rabbits and dogs. *Respir Physiol* 8: 204–229, 1970.
9. **Dowson D.** Mineral oil and scientific studies of lubrication. In: *History of Tribology.* New York: Longman, 1979, p. 308–327.
10. **Dowson D and Jin ZM.** An analysis of micro-elastohydrodynamic lubrication in synovial joints considering cyclic loading and entraining velocities (Abstract). *Proceedings of the 13th Leeds-Lyon Symposium on Tribology*, 1987, p. 375–386.
11. **Dowson D and Jin ZM.** A full numerical solution to the problem of micro-elastohydrodynamic lubrication of a stationary, compliant, wavy, layered surface firmly bonded to a rigid substrate with particular reference to human synovial joints. *Proc Inst Mech Eng [H]* 206: 185–194, 1992.
12. **Gibbon JHJ and Churchill ED.** The mechanical influence of the pericardium upon cardiac function. *J Clin Invest* 10: 405–422, 1931.
13. **Goto Y and LeWinter MM.** Nonuniform regional deformation of the pericardium during the cardiac cycle in dogs. *Circ Res* 67: 1107–1114, 1990.
14. **Hamilton DR, deVries G, and Tyberg JV.** Static and dynamic operating characteristics of a pericardial balloon. *J Appl Physiol* 90: 1481–1488, 2001.
15. **Harasawa H, Li KS, Nakamoto T, Coghlan L, Singleton HR, Dell’Italia LJ, and Santamore WP.** Ventricular coupling via the pericardium: normal versus tamponade. *Cardiovasc Res* 27: 1470–1476, 1993.
16. **Hoit BD, Dalton N, Bhargava V, and Shabetai R.** Pericardial influences on right and left ventricular filling dynamics. *Circ Res* 68: 197–208, 1991.
17. **Hoit BD, Lew WYW, and LeWinter M.** Regional variation in pericardial contact pressure in the canine ventricle. *Am J Physiol Heart Circ Physiol* 255: H1370–H1377, 1988.
18. **Hoppin FG, Green ID, and Mead J.** Distribution of pleural surface pressure in dogs. *J Appl Physiol* 27: 863–873, 1969.
19. **Hou JS, Mow VC, Lai WM, and Holmes MH.** An analysis of the squeeze-film lubrication mechanism for articular cartilage. *J Biomech* 25: 247–259, 1992.
20. **Kass DA, Midei M, Brinker J, and Maughan L.** Influence of coronary occlusion during PTCA on end-systolic and end-diastolic pressure-volume relations in humans. *Circulation* 81: 447–460, 1990.
21. **Kenner HM and Wood EH.** Intrapericardial intrapleural, and intracardiac pressures during acute heart failure in dogs studied without thoracotomy. *Circ Res* 19: 1071–1079, 1966.
22. **Kuno Y.** The significance of the pericardium. *J Physiol (Lond)* 50: 1–36, 1915.

23. **Lai-Fook SJ, Price DC, and Staub NC.** Liquid thickness vs. vertical pressure gradient in a model of the pleural space. *J Appl Physiol* 62: 1747–1754, 1987.
24. **Lai-Fook SJ and Rodarte JR.** Pleural pressure distribution and its relationship to lung volume and interstitial pressure. *J Appl Physiol* 70: 967–978, 1991.
25. **McMahon SM, Permutt S, and Proctor DF.** A model to evaluate pleural surface pressure measuring devices. *J Appl Physiol* 27: 886–891, 1969.
26. **McMichael J.** Heart. *Annu Rev Physiol* 10: 201–224, 1948.
27. **Mow VC and Mak AF.** Lubrication of diarthrodial joints. In: *Handbook of Bioengineering*. New York: McGraw-Hill, 1987, 5.1–5.34.
28. **Permutt S, Caldini P, Bane HN, Howard P, and Riley RL.** Liquid pressure versus surface pressure of the esophagus. *J Appl Physiol* 23: 927–933, 1967.
29. **Santamore WP, Constantinescu MS, Bogen D, and Johnston WE.** Nonuniform distribution of normal pericardial fluid. *Basic Res Cardiol* 85: 541–549, 1990.
30. **Sarnoff SJ.** Myocardial contractility as described by ventricular function curves; observations on Starling's law of the heart. *Physiol Rev* 35: 107–122, 1955.
31. **Scott-Douglas NW, Traboulsi M, Smith ER, and Tyberg JV.** Experimental instrumentation and left ventricular pressure-strain relationship. *Am J Physiol Heart Circ Physiol* 261: H1693–H1697, 1991.
32. **Smiseth OA, Frais MA, Kingma I, Smith ER, and Tyberg JV.** Assessment of pericardial constraint in dogs. *Circulation* 71: 158–164, 1985.
33. **Smiseth OA, Scott-Douglas NW, Thompson CR, Smith ER, and Tyberg JV.** Nonuniformity of pericardial surface pressure in dogs. *Circulation* 75: 1229–1236, 1987.
34. **Traboulsi M, Scott-Douglas NW, Smith ER, and Tyberg JV.** The right and left ventricular intracavitary and transmural pressure-strain relationships. *Am Heart J* 123: 1279–1287, 1992.
35. **Tyberg JV, Misbach GA, Glantz SA, Moores WY, and Parmley WW.** A mechanism for the shifts in the diastolic, left ventricular, pressure-volume curve: the role of the pericardium. *Europ J Cardiol* 7, Suppl: 163–175, 1978.
36. **Wiener-Kronish JP, Gropper MA, and Lai-Fook SJ.** Pleural liquid pressure in dogs measured using a rib capsule. *J Appl Physiol* 59: 597–602, 1985.
37. **Williams PF, Powell GL III, and LaBerge M.** Sliding friction analysis of phosphatidylcholine as a boundary lubricant for articular cartilage. *Proc Inst Mech Eng [H]* 207: 59–66, 1993.

