Atrial Transmural Pressures during Experimental Pericardial Tamponade

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ABSTRACT

To elucidate the mechanism responsible for the occurrence of negative transmural atrial pressures in pericardial tamponade, saline or air was progressively injected into the pericardial sac. In 14 anesthetized, open-chest dogs, left atrial (LA), left ventricular (LV), and pericardial pressures (PP) were measured with catheter tip manometers. In beating and fibrillated hearts, measurements were carried out at normovolemia, hypovolemia and hypervolemia. Transmural atrial pressure (TAP = LA — PP) became negative when the PP reached 130 mm H2O whether the heart was beating or not. In normovolemia the relation between TAP and PP was: TAP (mm H2O) = 38.1 — 0.237PP — 0.00042PP2. In anesthetized, closed-chest dogs, air inflation of pleural space produced pressures around the heart similar to those obtained in pericardial tamponade. With such pneumothorax, no negative TAP occurred either with beating or quiescent hearts. It is hypothesized that pericardial filling stiffens the pliable structures surrounding the heart and thereby diminishes the collapsibility of the atrial walls which form the inner part of the pericardial sac. Veins entering the atria through the filled pericardial sac are held open like the hole in an inflated inner tube. Through these mechanisms some cardiac filling is ensured even with marked pericardial tamponade.

KEY WORDS

ventricular transmural pressures atrial wall stiffness low pressure system manometry cardiac filling hypovolemia positive pressure pneumothorax ventricular fibrillation pericardial function dog

That negative transmural pressures during pericardial effusion can develop not only in the ventricles but also in the atria was noted by us in preliminary studies. Fowler et al. (1) studied in detail negative transmural pressures in the ventricles during pericardial tamponade. It is relatively easy to explain ventricular pressures lower than those in the pericardial sac since the ventricles with thick walls and small volumes can develop subatmospheric pressures that can aid ventricular filling, a variety of “suction” (2, 5). While negative transmural pressures across ventricular walls can be explained by the relative rigidity of the thick walls (3-5), it is more difficult to explain how negative transmural pressures could develop across the thin-walled atria, whose walls appear to be as pliable and collapsible as those of many veins. In freely collapsible veins, the transmural pressure can never fall below zero when the extramural pressure exceeds the intramural pressure (6-8). If the atria behaved similarly, their transmural pressures should not become negative. However, the occurrence of negative transmural pressures in the atria suggests the presence of a mechanism that counteracts the complete collapse of the atrial walls. The purpose of the present study was to find out whether such a mechanism exists.

Methods

To achieve the precision necessary for manometry in low pressure systems, a uniform zero pressure reference level was established by filling the open-chest dogs with saline (Fig. 1) as...
Experimental arrangement for pressure recording from heart and pericardium with high and low fidelity instrumentation. High fidelity is assured by intra-vascular manometers (MM) in left ventricle and left atrium. Low fidelity is brought about by the presence of the catheter lumens leading from the left ventricle, left atrium, and pericardial space to the respective extravascular manometers (EM) of conventional construction. The electrical connections from both intra-vascular and extravascular manometers are shown in broken lines leading to the electrical inputs of the recorder. The zero reference is the level of the saline lake.

previously described (3). This was done in 14 mongrel dogs weighing 14 to 22 kg and anesthetized with morphine (1.4-2.0 mg/kg) and chloralose (100 mg/kg). After a thoracotomy in the 5th intercostal space, a polyethylene cannula 42 cm long (i.d. 4.5 mm, o.d. 8 mm) with four large holes at the tip was inserted into the pericardial space and securely tied. A no. 7, 100-cm catheter tip manometer (Statham SF-1) was directed into the left atrium (LA) through a pulmonary vein. Another SF-1 manometer was inserted into the left ventricle (LV) via a carotid artery. Pressures obtained from the LA and LV were transmitted to Statham P23Gb transducers through the lumina of the SF-1 catheters. The pericardial cannula was also connected to a P23Gb transducer. The sensitivity of the pericardial and atrial pressure transducers was made equal to facilitate the measurement of atrial transmural pressures. The surface of the saline lake in the chest was used as the common zero pressure level for all three P23Gb manometers. The zero levels for the two SF-1 catheter tip manometers were established by superimposing their tracings with the respective ones obtained via the lumina of the LA and LV catheters. The passive effects of lung inflation upon the heart's position and upon the level of the “saline lake” were eliminated by disconnecting the respirator from the animal for 5 to 10 seconds. During these periods the zero level and all pressures were recorded. The tracings were taken at 180 mm/sec paper speed with an 8-channel photographic recorder (Electronics for Medicine). Fibrin deposits were prevented by heparinization (2.5 mg/kg). The saline in the chest cavity and the body temperature were kept at 37°C ± 0.5° to minimize temperature drift of the SF-1 manometers. Arterial blood gases were determined five times during each experiment, and sodium bicarbonate (average 44 mEq) was given as needed to maintain blood pH within the physiological range.

The pressures recorded when the pericardial cavity was empty served as control values. The experimental conditions were produced by step-wise installations of 25 or 50 ml of warm saline into the pericardial sac. Atrial and pericardial pressures were recorded at each volume of pericardial fluid after approximately 1 minute for stabilization of the hemodynamic alterations. When severe circulatory impairment occurred with marked tamponade, the saline was quickly removed and the animal allowed to recover. The stepwise production of pericardial effusion was repeated several times in most experiments.

All measurements were first made at normal blood volumes and then repeated in states of hypovolemia and hypervolemia. Hypovolemia was produced by removing 10 ml blood/kg body weight. Hypervolemia was brought about by additional infusion of homologous blood from a donor dog. After each blood volume change the animal was allowed to stabilize for several minutes.

Simultaneous left atrial and pericardial pressures were measured just before the onset of atrial systole. Left atrial transmural pressures were calculated as the difference between the intramural atrial pressure and the pericardial pressure.\(^1\)

To find out whether the flow of blood has an influence upon the development of the negative transmural atrial pressures during pericardial effusion, additional experiments were undertaken after stopping the circulation. In three anesthetized open-chest dogs, no. 9 multiholed catheters were placed into the LA and LV and connected to saline column manometers suitable for static pressure measurements. The pericardial cavity was cannulated as described above and this catheter was attached to another saline manometer. All three columns of saline were set at a common zero level. At this point, the heart was

\(^1\)The data were analyzed on a RCA Spectra 70 computer using a general linear model program.
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Fibrillated by a 60-cycle, 110-volt current. The pericardial sac was then filled with saline or inflated with air to various pressure levels. All three manometers were read directly in millimeters of saline. In low pressure systems the use of water rise tubes assures a greater sensitivity and accuracy than can be achieved with mercury manometers (5, 6).

Further experiments were designed to determine whether a negative atrial pressure is produced not only when the pericardial pressure is raised but also when the pressure surrounding the heart outside the normal pericardium is elevated. This situation would correspond to a positive pressure pneumothorax. In five anesthetized, closed-chest dogs a large cannula was inserted through a stab wound into each side of the thoracic cavity. The lumens of the cannulas were connected via a Y-tube to a saline manometer. Right atrial and right ventricular pressures were obtained through no. 9 catheters attached to saline manometers. Air was then blown into the chest cavity to produce a positive pressure surrounding the heart. The height of each saline column was measured and thereafter the air was withdrawn from the chest. This procedure was repeated several times to obtain a wide range of positive thoracic pressures. After completing the measurements with the chest closed, a thoracotomy was performed, a pericardial cannula inserted, and the RA, RV, and pericardial tamponade pressures were measured over a wide range of pericardial tamponade pressures. These experiments were performed with normal blood volumes only. The measurements were made in beating hearts using atrial and pericardial mean pressures as well as in quiescent hearts after induction of ventricular fibrillation.

Results

The tracings in Figure 2 illustrate the effect of pericardial tamponade on the pressure relations between the atria and the pericardium in a representative experiment. The control part of the figure shows that, under the normal condition of zero pericardial pressure, the tracing of atrial pressure lies above that of the pericardial pressure, indicating a positive left atrial transmural pressure. The middle part of the figure illustrates that, at a pericardial pressure induced by 100 ml of saline, there is still a positive transmural atrial pressure. The right part of the figure shows a negative left atrial transmural pressure associated with 200 ml of pericardial fluid, with the pericardial tracing lying above the atrial pressure tracing. Note that the atrial transmural pressure is negative throughout the entire diastole.

Figure 3 is a plot of the atrial transmural pressures calculated from all measurements in the beating hearts of 14 dogs with pericardial

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Since the difference in the height of a water or saline column is negligible, measurements are expressed in mm of H2O.

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Transmural left atrial pressure plotted against pericardial pressure. Values near the ordinate are at low pericardial pressures and positive transmural left atrial pressures. Values to the right side are at higher pericardial pressures and show negative transmural left atrial pressures. For any value above the zero line of the ordinate, left atrial pressure is greater than pericardial pressure; for any value below the zero line of the ordinate, left atrial pressure is less than pericardial pressure. See text for further details.

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FIGURE 2

Representative pressure tracings showing control and pericardial tamponade of 160 ml and 200 ml. (L.V.P. = left ventricular pressure, L.A.P. = left atrial pressure, P.P. = pericardial pressure). Time lines are 0.1 second in this figure. See text for discussion.

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Intraatrial pressure and atrial transmural pressure at increasing pericardial pressure in the nonbeating heart with normal blood volume, hyper- and hypovolemia. In A the absolute values of the intraatrial pressures are plotted at increasing pericardial pressures. The level of the atrial pressures in hypovolemia is lower and in hypervolemia higher than at a normal blood volume. In all three blood volume conditions, the intraatrial pressures tend to rise with increasing pericardial effusion. Part B illustrates the relation of the calculated transmural atrial pressures to the degree of pericardial tamponade. Each point represents the difference between the values shown for the intraatrial pressures in A and the pericardial pressures. As in the beating hearts, it is evident that negative transmural atrial pressures can also develop when blood flow has stopped.

Figure 5 shows the measurements from a representative experiment designed to determine whether the negative transmural atrial pressures are caused by an elevation of the extramural cardiac pressure per se or by the specific character of a pressure rise in the effusion. The solid line represents the transmural pressures in normovolemia. With the empty pericardial sac, the mean of the normovolemic left atrial transmural pressure is about 40 mm of H2O. The transmural pressures become smaller as the pericardium is progressively distended with saline. At approximately 130 mm H2O pressure in the pericardium, the atrial transmural pressures tend to be zero. At still higher pericardial pressures the transmural pressures become consistently negative. The light dashed line in Figure 3 is derived from the measurements in 12 dogs during hypervolemia and is not significantly different from the values in normovolemia. With hypovolemia, the transmural atrial pressures become zero at about 70 mm H2O of pericardial pressure and are substantially more negative than those with normovolemia at equivalent degrees of pericardial tamponade.

Figure 4 shows the measurements from a representative experiment with a nonbeating heart at normovolemia, hypovolemia, and hypervolemia. In A the absolute values of the intraatrial pressures are plotted at increasing pericardial pressures. The level of the atrial pressures in hypovolemia is lower and in hypervolemia higher than at a normal blood volume. In all three blood volume conditions, the intraatrial pressures tend to rise with increasing pericardial effusion. Part B illustrates the relation of the calculated transmural atrial pressures to the degree of pericardial tamponade. Each point represents the difference between the values shown for the intraatrial pressures in A and the pericardial pressures. As in the beating hearts, it is evident that negative transmural atrial pressures can also develop when blood flow has stopped.

Intraatrial pressures (A and C) and atrial transmural pressures (B and D) in the beating heart during inflated chest (A and Ti) and inflated pericardium (B and D). See text for explanation.

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Intraatrial pressures (A) and atrial transmural pressures (B) in the quiet heart during bilateral pneumothorax. See text for explanation.

The results of these experiments demonstrate that negative transmural pressures can develop across the thin, pliable atrial walls during pericardial tamponade. In fact, the degree of negativity in the present studies established with the simple and direct pressure measurements is so marked that this peculiar phenomenon cannot be attributed to artifacts or instrumentation errors which can easily lead to faulty measurements in low pressure systems (6, 9).

Since we have established a systematic quantitative relation of the negative transmural atrial pressures to the extent of pericardial tamponade in both the beating and quiescent heart (Figs. 2, 3, 4B, 5B), this information permits us to advance a hypothesis that may serve as an explanation.

**Discussion**

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péricardial sac. Part A is a plot of the right intraatrial pressures measured in the beating heart at stepwise increases of intrathoracic pressure in the closed-chest dog. The pericardium is left untouched in its normal position. At the normal negative intrathoracic pressure of —90 mm H2O during the respiratory pause, the atrial pressure is —5 mm of H2O. With thoracic positive pressure inflation, the intra-atrial pressures rise conspicuously. Part B gives the values for the calculated transmural atrial pressures at various levels of pressure surrounding the heart. Although the transmural atrial pressure decreases with increased thoracic inflation, it does not become negative. After these measurements were made the thorax was opened and the pericardium cannulated, and approximately the same extramural cardiac pressures were produced by inflating the pericardial cavity. Part C shows a slight rise of the intrathoracic pressure with pericardial tamponade. Part D is a plot of the calculated values of the transmural atrial pressures when the extramural pressure is elevated in the pericardium. In this situation the transmural pressures become markedly negative as contrasted to the positive transmural atrial pressures shown in B.

Figure 6 shows the results of a representative experiment designed for studying the problem of whether the absence of negative transmural atrial pressure in the tension pneumothorax (Fig. 5B) can be attributed to the hemodynamics of the intact circulation or not. In the closed-chest dog, the heart was electrically fibrillated. The pericardium was left intact while right atrial and intrathoracic pressures were recorded. Part A of Figure 6 is a plot of the intraatrial pressure measured at increasing elevations of intrathoracic pressure. Part B of Figure 6 contains the values of the calculated transmural atrial pressures. They are consistently positive and correspond to those found in the beating heart (Fig. 5B). After opening the chest and inflating the pericardium, the transmural atrial pressures also become negative in this particular experiment. The values were similar to those depicted in Figure 4B for a normovolemic dog with a nonbeating heart.
FIGURE 7

In a schematic form typical filling pressures (mm Hg) are represented here for the normal (A) and for pericardial tamponade (B). See text for discussion.

The occurrence of a negative transmural pressure in the atria during pericardial effusion indicates the existence of a force that counteracts the complete collapse of the atrial walls. It must be assumed that the distended pericardium not only compresses the atria but at the same time becomes a rather stiff structure surrounding the atrial lumen. This structure consists of the distended pericardial sac of which the atrial walls form the inner aspect. The increase in the overall pericardial stiffness may act as a force that prevents the complete collapse of the atrial walls. Although the atrial lumen is decreased, it is not entirely obliterated and therefore the atria continue to be filled from the extrapericardially situated veins. Furthermore, the distended pericardial sac does not cause a total compression of the veins at their entry into the atria. By inspection and by using a probe, one can observe that, although the lumen of the veins becomes smaller, they remain patent as they enter and penetrate the effused pericardium. It is as if the area of the pericardium surrounding the veins becomes inflated like the inner tube of a tire. The overall diameter of this doughnut-shaped structure increases with inflation, whereas the diameter of the "hole" decreases. The concept presented here is visualized in Figure 7.

The results of our experiments also cast a light on the effects of different filling states of the circulatory system on the development of negative atrial transmural pressures in pericardial effusion. The less well filled the circulatory system is, the greater becomes the negativity of atrial transmural pressures (Figs. 3, 4B). We cannot explain why there was no significant difference in the pressure relations during normovolemia and hypervolemia in the beating hearts but such a difference was evident in the quiescent hearts (Fig. 4B). The development of negative atrial transmural pressure did not depend on the movement of blood as revealed by the measurements undertaken when blood circulated normally and after it had been stopped. From this finding one must conclude that primarily mechanical alterations during pericardial effusion produce negative atrial transmural pressures. This evidence supports the hypothesis advanced as an explanation at the beginning of our discussion (Fig. 7).

It is obvious that the negative transmural pressures do not result from the compression of all cardiac structures by an overall higher extracardial pressure (pressure pneumothorax) but from the compression by a structure of a specific configuration (pericardium) (Figs. 5B and D, 6). This observation lends further support to the mechanical explanation elaborated on in this discussion (Fig. 7).

One may conclude from the results of this investigation that in addition to the well known hemodynamic functions of the normal pericardium and those in pericardial effusion, there is another function, which has remained unknown in the past. It has been established that the normal pericardium restrains the heart and its cavities from overdistension.
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11). It has also been shown that moderate pericardial effusion interferes primarily with ventricular filling (12) but marked effusion involves numerous hemodynamic changes (13). The contribution of the experiments reported here is that even in very marked pericardial effusion the veins entering the atria are not fully occluded nor are the atrial cavities obliterated by a complete collapse of the atrial walls. Through this mechanism (Fig. 7) a small amount of ventricular filling and output is assured.

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