Transmural Ventricular Pressures in Experimental Cardiac Tamponade

By Noble O. Fowler, M.D., Ralph Shabetai, M.D., and John R. Braunstein, M.D., Ph.D.

Experimental cardiac tamponade was produced in open-chest dogs. Left and right ventricular systolic pressures fell when intrapericardial pressure was increased only a few millimeters of mercury. Negative diastolic transmural pressures of 1 to 6 mm Hg developed in the left ventricle and negative diastolic transmural pressures of 2 to 8.5 mm Hg developed in the right ventricle. In some instances the period of right ventricular systolic ejection was greatly abbreviated at higher levels of intrapericardial pressure.

Bloom1 has shown that the excised beating mammalian heart placed in isotonic saline solution is capable of repeatedly filling and emptying the ventricles in the absence of the vis a tergo produced by atrial pressure. Measurements obtained under these circumstances showed that pressure within the left ventricle fell below that in the surrounding liquid during diastole.2,3 Brecher4 demonstrated that in dogs the left ventricle was capable of exerting an aspirating effect during diastole when obstruction was produced at the mitral orifice. Bloom and Ferris5 and Fowler, Couves and Bewick5 demonstrated subatmospheric right ventricular diastolic pressure during experimentally produced obstruction to inflow in the dog's right ventricle. Kraner and Ogden6 demonstrated diastolic suction in the excised turtle ventricle. Brecher and Kissin7 have concluded that the development of negative relaxation pressure in the manually compressed static dog ventricle is related to elastic recoil when there is a low residual volume. This study suggested that the negative diastolic pressure observed in the excised beating heart and in the beating heart of the intact animal with atrioventricular valve obstruction might be related to elastic recoil associated with abnormally low residual volumes and that these observations do not necessarily imply that diastolic suction is an important mechanism in cardiac filling under normal conditions. Fowler, Couves and Bewick5 showed that negative diastolic pressure developed in the left ventricle of the dog during rapid exsanguination, a finding consistent with Brecher's observation of negative relaxation pressure in association with low residual ventricular volume.

The production of cardiac tamponade by means of the injection of air or liquid into the pericardial sac is known to produce certain hemodynamic changes. Post8 demonstrated a rise in systemic venous pressure, a fall in systemic arterial blood pressure and cardiac output, an increase in systemic arterial resistance, a fall in glomerular filtration rate, and sodium retention during experimental cardiac tamponade in the closed-chest dog. Metcalfe, Woodbury, Richards, and Burwell9 produced cardiac tamponade in closed-chest dogs, and showed that right atrial and pulmonary venous pressures increased almost equally as pericardial pressures rose, with changes in pulmonary arterial pressures being relatively small. When
FOWLER, SHABETAI, BRAUNSTEIN

pulmonary venous pressure increased during progressive tamponade to approximately 20 mm. Hg, circulation ceased. Isaacs, Berglund and Sarnoff showed that pressures rose in both right and left atria of the dog during increasing cardiac tamponade, but that the effective atrial filling pressures fell, with a proportional decline in stroke work. The circulatory defect was believed related to a limitation of pericardial space available for diastolic expansion of the ventricles, resulting in shortening of diastolic ventricular fiber length, thus decreasing ventricular work.

Cardiac tamponade by pericardial fluid is thus an example of another situation in which the amount of blood in the ventricles in diastole may be less than normal because of impairment of cardiac filling. For this reason, it seemed desirable to investigate the transmural ventricular pressure in diastole during experimental cardiac tamponade in animals.

The following study reports the results of the transmural pressure in the right and left ventricles of dogs subjected to experimental cardiac tamponade and shows that a negative transmural ventricular diastolic pressure developed under these circumstances. The study also relates the level of intrapericardial pressure to changes in right and left ventricular systolic pressures. In some instances, right atrial pressure was also measured.

METHODS

The studies described were performed upon open-chest dogs under sodium pentobarbital anesthesia (25 mg./Kg.) with positive pressure respiration. Studies were made in 20 dogs with satisfactory data being obtained in 13 animals. Pressures were recorded from the cardiac ventricles through no. 12 or 13 needle cannulas with side openings near the tip. In two instances no. 20 needles were used. Pressures were recorded from the right atrium with a no. 7, 100 cm. cardiac catheter or, in later experiments, with a no. 13 side-

![Image](https://example.com/image.png)
hole needle cannula. In one instance, left atrial pressure was recorded through a no. 20 side-hole needle cannula inserted into a pulmonary vein and advanced until its tip lay at the junction of a pulmonary vein and the left atrium. Intra-pericardial pressures were recorded through a no. 7, 100 cm. cardiac catheter with the catheter tip sutured to the pericardium at a point level with the entry of the left ventricular needle cannula, or, in later experiments, from a no. 18 needle and a 10 cm. section of no. 7 cardiac catheter at the level of the left ventricular needle cannula. Electrocardiograms and pressures were recorded simultaneously. Pressures were recorded by Statham P 23 D gages and Electronics for Medicine Research Recorder with the exception of the first 3 animals, in which the Sanborn Poly-Viso Recorder was used. Paper speeds were 100 or 200 mm./sec. The pressures were recorded at sensitivities of 2.5 to 3 mm. of paper for each millimeter of mercury pressure. For all strain gages a common zero reference point was selected at a fixed vertical distance above the top of the animal table. This reference point was 1.7 cm. superior to the level of the horizontal left ventricular needle. In later tracings, right ventricular, left ventricular and intrapericardial pressures were recorded at equal sensitivities, the strain gages being standardized by use of a mercury manometer. Tracings were checked at the end of each run for electrical drift, for gage balance and for return of the tracings to control configurations following removal of the pericardial fluid. Strain gages and needles were rigidly clamped to minimize vibration artefact.

Saline was injected into the pericardial space through a cardiac catheter inserted into the pericardium through a separate opening. Pressures were recorded continually in the pericardial space and in right and left ventricles and, in some instances, right atrium, as intrapericardial pressure rose. In some instances pressure was recorded also as fluid was removed from the pericardium. Intrapericardial pressure was increased until left ventricular systolic pressure fell to approximately 25 to 40 mm. Hg, at which time pressure recordings were made and fluid was removed from the pericardial space.

Results

The results are shown in figures 1 to 5. The pressure tracings of figures 2, 3, and 4 were retouched for better illustration. In general, the findings were consistent in the different animals. Satisfactory runs of left ventricular and pericardial space pressures were obtained in 20 instances in 12 animals and of right ventricular pressure in 10 instances in 7 animals. Intrapericardial pressures were often slightly negative initially, since the zero reference level was superior to the catheter or needle tip in the pericardium. During tamponade, systolic pressures fell and diastolic pressures rose in both left and right ventricles (fig. 1). A negative left ventricular diastolic transmural pressure, in other words, a higher intrapericardial than left ventricular diastolic pressure, was seen in each instance with the exception of one run in dog no. 42 (fig. 1). In this single instance the intrapericardial pressure during tamponade of 9/7.5 mm. Hg was the lowest obtained in this study. In the right ventricle, negative diastolic transmural pressure during tamponade was found in each instance (fig. 1). This negative pressure, unlike that in the left ventricle, was often restricted to early diastole.

Figure 2 is an illustration of left ventricular and intrapericardial pressures of dog no. 71 before, during and after the increase of pressure in the pericardial space by injection of physiologic saline solution. During tamponade, a negative left ventricular diastolic transmural pressure of 2.5 mm. Hg was produced.

Figure 3 is an illustration of left ventricular, left atrial and intrapericardial pressures of dog no. 79 before and during the production of cardiac tamponade by injection of physiologic saline into the pericardial space. Negative left ventricular diastolic transmural pressure of 5 mm. Hg developed; during tamponade diastolic left atrial pressure was 1 mm. Hg higher than left ventricular diastolic pressure, but was 4 mm. Hg lower than intrapericardial pressure.

Figure 4 shows a record of right ventricular pressure and intrapericardial pressure in dog no. 69 before, during and after production of cardiac tamponade. In early diastole, a negative transmural diastolic pressure of 6 and 7 mm. Hg was recorded.
Figure 5 is a graph of pressure readings during injection of saline solution into the pericardial space in dog no. 67 and demonstrates that right ventricular and left ventricular systolic pressures fell with a rise in intrapericardial pressure from -2 to 2 mm. Hg. Furthermore, it may be seen that the left ventricular diastolic pressure became lower than intrapericardial pressure very early during the rest of the fluid injection and remained below the intrapericardial pressure during the rest of the fluid injection with a maximum transmural pressure difference of 5.8 mm. Hg. Right ventricular diastolic pressure, on the other hand, remained higher than intrapericardial pressure until relatively high levels of intrapericardial pressure, 10.8 mm. Hg, were reached. As intrapericardial pressure was increased further, right ventricular diastolic pressure was below intrapericardial pressure, reaching an eventual difference of 8.4 mm. Hg. Right ventricular systolic pressure exceeded intrapericardial pressure at first, but did not at the higher intrapericardial pressure levels of 16.5 and 19.6 mm. Hg.

**Discussion**

The results obtained are consistent with the theory that the development of negative transmural ventricular diastolic pressure is related to abnormally low ventricular volume. However, since measurement of ventricular volumes and of flows were not made, it is impossible to state whether or not the difference in pressure observed were, in fact, an expression of diminished ventricular volume. These observations do suggest, however, that with sufficient increase in intrapericardial pressure and with the chest open, ventricular diastolic suction may be an aid to ventricular filling. This mechanism seems

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**Fig. 3** Top. Left ventricular, left atrial and intrapericardial pressures of dog no. 79 before (above) and during (below) production of cardiac tamponade by injection of physiologic saline into the pericardial space. Ventricular fibrillation developed shortly after lower record was taken. *Pressures, millimeters of mercury; paper speed, 200 mm/sec.* Left ventricular pressure recorded at twice control sensitivity during tamponade.

**Fig. 4** Bottom. Right ventricular and intrapericardial pressures of dog no. 69 before (above), during (center) and after (below) the production of cardiac tamponade by injection of physiologic saline into the pericardial space. *Pressures, millimeters of mercury; paper speed, 200 mm/sec.* Pressures in right ventricle and pericardial space are recorded at the same sensitivity, but there is a slight hydrostatic difference in the zero reference level.
VENTRICULAR PRESSURES DURING TAMPONADE

Fig. 5. Relationship between left ventricular systolic and minimum diastolic pressures, right ventricular systolic and minimum diastolic pressures, right atrial pressure (ordinate), and intrapericardial pressure (abscissa) of dog no. 67 as pericardial pressure is increased continuously by injection of physiologic saline into the pericardial space.

to occur in the left ventricle at lower intrapericardial pressure than in the right.

An early diastolic dip was observed in the left ventricular pressure pulse curve in only one instance, in which intrapericardial pressure was increased to 35 mm. Hg. In this animal, the left ventricular systolic pressure rise was of brief duration and increased intraventricular pressure by only a few millimeters of mercury. Thus, the intraventricular pressure pulse did not resemble the right ventricular pressure pulse and associated early diastolic dip described by Hansen, Eskildsen and Gotzsche11 in patients with constrictive pericarditis. However, the present studies are acute, short term experiments in the open-chest animal and may not be related to what is seen in the chronic human disease.

These studies show that with high intrapericardial pressure, right ventricular systolic pressure may not exceed that in the pericardial space. A similar observation was made by Burwell12 who stated that the contribution of the right ventricle to the pressure head in the pulmonary circulation was not great in experimental cardiac tamponade. Another interesting observation with regard to the right ventricular pressure pulse is an early decline in pressure during left ventricular systole observed in some experiments, suggesting that right ventricular ejection may be of very brief duration under conditions of very high intrapericardial pressure in the open-chest dog. This apparent very short duration of systole may be related to a diminished volume of systolic discharge against relatively low pressure since it is
known that the duration of systolic ejection is related to the stroke volume and to the resistance to ejection. Measurements of pulmonary arterial pressure in 2 animals demonstrated no similar early decline in pulmonary arterial pressure. Study of the right atrial pressure curves in those animals showing early decline in pressure following a very brief systole showed a decline of right atrial pressure only during the early portion of this period. Under these circumstances of short systolic ejection, the right ventricle may be almost completely emptied by the pericardial tamponade. It seems likely that the thinner-walled right ventricle may be compressed by tamponade to a relatively smaller volume than the thicker-walled left ventricle.

It is of interest to note that in these studies the left ventricular systolic pressure began to decline during pericardial fluid injection at a much lower level of intrapericardial pressure than was observed by Metcalf, Woodbury, Richards and Burwell in their animals subjected to experimental cardiac tamponade, where no fall in femoral arterial pressure was seen until intrapericardial pressure rose to at least 10 mm. Hg. However, there was a rapid decline of femoral arterial pressure as intrapericardial pressure rose to 15 to 20 mm. Hg. Their studies were performed in a closed-chest animal in whom the respiratory pump could act as an aid to right heart filling. The earlier decline in systolic ventricular pressures observed in our study in relation to slight elevation of intrapericardial pressure may be due to the loss of the respiratory pump as an aid in return of blood to the right heart and to the impairment of venous return by positive pressure respiration. This concept is consistent with the observations made by Brecher in his studies of the venous return in open-chest animals. The decline in right ventricular diastolic pressure below intrapericardial pressure found by us was not noted by Evans, Walter and Hellems in their study of experimental cardiac tamponade in dogs. However, their study was made with the chest closed, when the respiratory pump may prevent depletion of ventricular diastolic volume sufficient to produce negative transmural pressure.

**SUMMARY**

Transmural ventricular pressures during experimental cardiac tamponade were measured in 13 open-chest dogs. In 20 studies negative diastolic left ventricular transmural pressures of 1 to 6 mm. Hg were found during tamponade. In 10 studies negative right ventricular diastolic transmural pressures of 2 to 8.5 mm. Hg were observed during tamponade.

In the left ventricle, negative transmural diastolic pressures developed when the intrapericardial pressure was increased by a few millimeters of mercury and was sustained throughout diastole. In the right ventricle, negative diastolic transmural pressure was seen only after intrapericardial pressure rose to higher levels, approximating 10 mm. Hg, and was often present only in early diastole.

Right and left ventricular systolic pressures declined in these animals when intrapericardial pressure was increased by only a few mm. mercury. At higher levels of intrapericardial pressure (10 to 20 mm. Hg) the period of right ventricular systolic pressure rise became greatly abbreviated in some animals and right ventricular pressure decline during left ventricular systole was observed.

The results are consistent with the concept that impairment of ventricular filling during cardiac tamponade in the open-chest animal leads to diminished diastolic volume, with the development of negative diastolic transmural pressures from elastic recoil of the ventricles.

**Summario in Interlingua**

Le pressiones transmural ventricular esseva mesurate durante un tamponage cardiac experimental in 13 canes a thorace...
VENTRICULAR PRESSURES DURING TAMPONADE

In 20 studies, negative pressiones transmural sinistro-ventricular in diastole amontante a inter 1 e 6 mm de Hg esseva constatate durante le tamponage. In 10 studios, negative pressiones transmural dextero-ventricular in diastole amontante a inter 2 e 8,5 mm de Hg esseva constatate durante le tamponage.

In le ventriculo sinistre, negative pressiones transmural in diastole se disveloppava quando le pression intrapericardial esseva augmentate per alicue mm de Hg e mantenite assi a transverso le diastole. In le ventriculo dextere, negative pression transmural in diastole esseva constatate solmente post que le pression intrapericardial habeva montate a plus alte nivellos, i.e. a circa 10 mm de Hg. Iste pression negative esseva frequentemente presente in le prime portion del diastole.

Le pressiones systolic dextero- e sinistro-ventricular declinava in iste animales quando le tension intrapericardial esseva augmentate per solmente pauc mm de Hg. A plus alte nivellos de pression intrapericardial (i.e. a inter 10 e 20 mm de Hg), le periodo del augmento de pression dextero-ventricular in systole se mostrava grandemente abbreviata in certe animales, e occurrence de un reduction del pression dextero-ventricular esseva observate durante le systole sinistro-ventricular.

Le resultatos de iste studios se trova de acordo con le conception que le dysfuncion del plenation ventricular durante le tamponage cardiac in animales a thorace aperte resulta in un reduction del volumine diastolic, con le disveloppamento consequente de negative pressiones transmural diastolic per le resalto elastic del ventriculos.

REFERENCES

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NOBLE O. FOWLER, RALPH SHABETAI and JOHN R. BRAUNSTEIN

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