Left Ventricular Diastolic Collapse in Regional Left Heart Tamponade

An Experimental Echocardiographic and Hemodynamic Study

STEVEN L. SCHWARTZ, MD, FACC, NATESA G. PANDIAN, MD, FACC, QI-LING CAO, MD, TSUI-LIEH HSU, MD, MARK ARONOVITZ, BA, JAMES DIEHL, MD, FACC, FACS

Boston, Massachusetts

Objectives. This study was designed to describe the hemodynamic abnormalities associated with the appearance of left ventricular diastolic collapse in the setting of regional left heart cardiac tamponade.

Background. Cardiac tamponade after heart surgery is frequently associated with localized pericardial effusion. Although right ventricular diastolic collapse and right atrial collapse are reliable echocardiographic findings in patients with circumferential pericardial effusion and tamponade, they are often not present in postoperative patients with localized pericardial effusion and regional left heart tamponade. Left ventricular diastolic collapse has been described in such patients, but the degree of hemodynamic alteration that exists with this finding is not known.

Methods. Acute regional left heart tamponade was produced 14 times in seven spontaneously breathing anesthetized dogs by infusing fluid into an isolated compartment created in the pericardial space adjacent to the left ventricular free wall. Continuous echocardiographic imaging and hemodynamic monitoring of left ventricular, systemic arterial, right atrial, pulmonary capillary wedge and pericardial pressures were performed. Measurements at baseline were compared with those made at the onset of left ventricular diastolic collapse and at decompenated tamponade.

Results. Left ventricular diastolic collapse was noted in all 14 episodes of regional tamponade. It occurred when pressure in the left pericardial compartment exceeded left ventricular diastolic pressure by 3.0 ± 1.9 mm Hg. At the onset of left ventricular diastolic collapse, cardiac output and mean arterial pressure were significantly reduced from the control value (p < 0.05). Systolic hypertension was noted only twice at this stage, respiratory variation in systolic pressure >19 mm Hg only once. The appearance of this sign was also associated with elevated left heart filling pressures.

Conclusions. Left ventricular diastolic collapse is a reliable sign of regional left ventricular tamponade and is associated with a reduction in cardiac output. This echocardiographic finding usually occurs before the development of arterial hypertension and pulsus paradoxus. Thus, left ventricular diastolic collapse is potentially more reliable than hypertension or pulsus paradoxus in the diagnosis of regional left ventricular tamponade.

(J Am Coll Cardiol 1993;22:907-13)
Methods

Preparation. An animal model of acute regional cardiac tamponade was used. Nine male or female mongrel dogs (13 to 20 kg) were anesthetized with sodium pentobarbital (25 mg/kg body weight intravenously), intubated and ventilated on a Harvard volume cycle respirator. Fluid-filled catheters were advanced percutaneously into the left ventricle and right atrium and connected to pressure transducers. A balloon-tipped thermodilution catheter was placed in the pulmonary artery. Catheter position was confirmed using hemodynamic tracings and fluoroscopy. Arterial blood pressure was measured using an 8F sheath in the left femoral artery. The electrocardiogram was continuously monitored throughout the experiment.

A left lateral thoracotomy was performed. A small incision was made in the pericardium laterally, and a custom-designed 7F catheter with a 3 x 4-cm latex balloon at the distal end was inserted into the pericardial space posterior to the left ventricle and held in place with a purse-string suture (Fig. 1). This catheter was used for pressure measurement and infusion of saline solution. Balloon position was confirmed to be adjacent to the left ventricle with epicardial echocardiography. The pericardium was then sewn to the myocardium around the balloon. This created two pericardial compartments, one outside the left ventricle, which would be filled with fluid to create regional effusion and tamponade, and one outside the right ventricle. A 7F pigtail catheter was placed in the pericardial space anterior to the right ventricle to monitor pressure in this compartment. The chest was closed and the pneumothorax was reduced with a chest tube placed to underwater seal.

Experimental protocol. Intravenous sedation was adjusted to allow the dogs to be weaned from the ventilator. The protocol was carried out with the dogs breathing spontaneously. Heart rate and the following pressures were recorded at baseline: systemic arterial, left ventricular, pulmonary capillary wedge, right atrial, left pericardial (measured using the balloon catheter) and right pericardial. Cardiac output was measured by the thermodilution method. Echocardiographic imaging with a Hewlett-Packard Sonos 1000 imaging system was performed at baseline and continuously throughout the study from the right parasternal region. Long- and short-axis images of the left ventricle were obtained.

Normal saline solution was infused into the left pericardial compartment at 5-ml intervals. Repeat hemodynamic and echocardiographic measurements were obtained at each period. The onset of left ventricular diastolic collapse, defined as a discrete, transient, inward motion of the left ventricular free wall adjacent to the pericardial effusion in diastole, was noted (Fig. 2). The infusion was terminated when the mean arterial pressure was reduced to 75% of that in the control state, a condition defined as decompressed cardiac tamponade (19).

The fluid was removed and the dogs were allowed to recover for 30 min. The protocol was repeated. The animals were killed with an anesthetic overdose after completion of the experiment. Balloon position in the pericardium was visually confirmed. The balloon was removed from the pericardium, attached to a pressure transducer and filled with saline solution as in the protocol to determine its pressure-volume characteristics.

The experimental protocol was approved by the New England Medical Center Animal Research Committee. The study conforms to the "Position of the American Heart Association of Research Animal Use" adopted by that Association in November 1984.

Data analysis. The following hemodynamic measurements were recorded at each stage: heart rate, arterial pressure, respiratory variation in systolic pressure or pulsus paradoxus, left ventricular diastolic pressure, pulmonary capillary wedge pressure, right atrial pressure and pressure in the left and right pericardial compartments. Cardiac output was determined by the average of at least three measurements using a thermistor-tipped catheter. Stroke volume was calculated by dividing the cardiac output by the heart rate. From the echocardiographic images, left ventricular cavity area was measured at end-diastole in the short-axis view off-line using the analysis package contained within the imaging system. Images were viewed by two observers unaware of the experimental protocol to determine the interobserver variability for the presence of left ventricular diastolic collapse. Data obtained at baseline and at the onset of left ventricular diastolic collapse and of decompressed tamponade were compared using repeated measures analysis of variance testing with use of a commercially available statistical program (Statview II, Abacus Concepts Inc.). The Fisher least-square difference test was used to determine differences between groups. All data are presented as mean value ± SD. Data were considered significantly different if p was < 0.05.
Results

Hemodynamic measurements. Regional cardiac tamponade was produced 14 times in seven dogs (two dogs died before completing the protocol). Left ventricular diastolic collapse was noted in all 14 episodes and occurred when pressure in the left pericardial compartment exceeded left ventricular diastolic pressure (Fig. 3). In 13 of 14 episodes the appearance of left ventricular diastolic collapse preceded the stage of decompensated tamponade; in 1 episode the echocardiographic sign appeared simultaneously with the defined hemodynamic decompensation (mean arterial pressure =75% of the control value).

The hemodynamic data obtained at baseline and at the onset of left ventricular diastolic collapse and of decompensated cardiac tamponade are summarized in Table 1. At the onset of left ventricular diastolic collapse, cardiac output was reduced 20 ± 4.3% from baseline (p < 0.05). Although both the mean and systolic arterial pressures were also significantly reduced, they were both in the normal range. Systolic hypotension was noted only twice at this stage. The increase in respiratory variation in systolic pressure did not achieve significance; there was only one instance in which it was >10 mm Hg. Both left ventricular diastolic pressure and pulmonary capillary wedge pressure had increased significantly, whereas right atrial pressure was unchanged.

There was a further reduction in cardiac output and blood pressure with continued infusion of fluid into the pericardial space. Although the average systolic arterial pressure at this stage was 80 ± 20 mm Hg, hypotension was present in only 8 of 14 cases. Respiratory variation in systolic pressure increased to 8.5 mm Hg but was >10 mm Hg in just three episodes. As expected, there was a further increase in left ventricular diastolic pressure. Only at the final stage of decompensated tamponade did the right atrial pressure exhibit a significant increase from baseline.
Pressure measurement in the right and left pericardial compartments demonstrated that the two were isolated. The pressures were no different from each other at baseline. At the appearance of left ventricular diastolic collapse, the mean pressure was 21 ± 10 mm Hg in the left pericardial compartment but only 3.5 ± 3.5 mm Hg (p < 0.05) in the right pericardial compartment.

Echocardiographic measurements. As already stated, left ventricular diastolic collapse was noted in every episode of tamponade produced and was observed when the pressure in the left pericardial compartment exceeded that of the left ventricle. An example of the changes in the appearance of the left ventricle as pericardial volume increases is illustrated in Figure 3. At baseline, the left ventricle appears normal. A small volume of pericardial fluid does not change the ventricular shape. Once pericardial pressure is greater than left ventricular pressure, an inward concavity of the posterior wall adjacent to the effusion (left ventricular diastolic collapse) occurs. Initially, this finding is noted within the one half to three fourths of diastole. At this stage, mean left ventricular end-diastolic cavity area had decreased from the control value of 8.9 ± 2.0 cm² to 6.6 ± 1.4 cm² (p < 0.05), an 11% reduction. As more fluid is added to the pericardial space, the deformation of the left ventricular wall is more pronounced and lasts throughout diastole. Cavity area at end-diastole was further reduced at the stage of decompensated tamponade to 5.3 ± 1.6 cm² (p < 0.05 vs. baseline and left ventricular diastolic collapse).

In 13 of 14 instances of left ventricular tamponade, there was agreement between the two observers regarding the appearance of left ventricular diastolic collapse. The sole disagreement was resolved by consensus.

Discussion

The present study demonstrates that left ventricular diastolic collapse is a reliable indicator of regional left ventricular tamponade, occurs when pericardial pressure is greater than left ventricular pressure in diastole and is associated with a significant reduction in cardiac output. Although arterial pressure is lower than observed at baseline, it is usually within the normal range. These observations imply that in regional tamponade, left ventricular diastolic collapse is an earlier marker than clinical signs of tamponade of a hemodynamically significant effusion.

In the typical situation in which cardiac tamponade is caused by circumsferential effusion, an increase in pericardial fluid volume and pressure is associated with a gradual increase in right heart filling pressures and a decrease in cardiac output (20). As pericardial pressure continues to increase, it transiently exceeds right ventricular diastolic pressure, resulting in an inward motion of the right ventricular wall known as right ventricular diastolic collapse (8). In both experimental and clinical studies (7,19), the onset of right ventricular diastolic collapse has been associated with a significant reduction in cardiac output without substantial changes in blood pressure. It has been hypothesized (19) that the left ventricle does not demonstrate this motion because of its greater thickness and its symmetric shape.

However, when the fluid collection is loculated posteriorly such as in the postoperative patient, collapse of these chambers is not observed on the echocardiogram. The increased volume and pressure within the loculated effusion have little, if any, direct effect on right atrial pressure as demonstrated in this study and in the model of left heart tamponade described by Fowler et al. (18). This increased...
pressure is transmitted across the adjacent left ventricular wall resulting in higher diastolic pressure, reduced end-diastolic volume and reduced stroke volume. Eventually, as in circumferential effusions, pressure within the pericardial compartment transiently exceeds that of the left ventricle itself, and the left ventricular wall is then shifted toward the lower pressure ventricular cavity. As the situation progresses, there is a continued increase in pericardial and left ventricular pressure, together with a more pronounced deformation of the left ventricular wall and even further reductions in cardiac output and arterial pressure. Right atrial pressure is increased at this later stage, possibly more as a backward reflection of elevated left heart filling pressures than of localized compression.

Previous studies of regional tamponade. Prior experimental evidence has suggested that right heart compression is more important than left heart compression in cardiac tamponade (18,21,22). Initial studies concluded that reduction in cardiac output is primarily the result of right heart (21) and atrial (22) compression. In contrast, Carey et al. (23) reported a significant reduction in both arterial pressure and cardiac output with localized tamponade of the left ventricle alone. In a study comparing right heart tamponade, left heart tamponade and combined tamponade, Fowler et al. (18) reported that left heart tamponade did cause a reduction in cardiac output, but the hemodynamic alterations of right heart tamponade were more dramatic. However, the most marked hemodynamic disturbances were observed in combined right and left heart tamponade.

The clinical occurrence of regional left heart tamponade after cardiac surgery has previously been reported (2,10,11,14–17,24). Paradoxic motion and compression of the posterior wall of the left ventricle have been observed in patients with posterior effusion (10,11,14–17). Left ventricular diastolic collapse has also been noted in a patient with a circumferential effusion and tamponade who had severe pulmonary hypertension (25). In the largest published series to date, Chuttani et al. (14) reported on 15 patients with clinical evidence of tamponade and loculated effusion after cardiac surgery; all exhibited left ventricular diastolic collapse. Thus, not only is regional tamponade clinically important, but the echocardiographic marker of this event, left ventricular diastolic collapse, appears to reliably indicate the presence of a hemodynamically significant effusion.

Pulsus paradoxus in regional tamponade. One finding in this study was the low magnitude of respiratory variation in systolic pressure despite a reduction in cardiac output and arterial pressure. This is consistent with the prior observation that right ventricular diastolic collapse is a more reliable sign of cardiac tamponade than is pulsus paradoxus (26). One criterion necessary for pulsus paradoxus to occur is that both ventricles must be filling against a common stiffness (27). Total cardiac volume expansion must be limited by the pericardial effusion; therefore, any increase in right ventricular volume with inspiration impedes left ventricular filling. In our model, the absence of pericardial fluid anteriorly allowed for the right heart chambers to freely expand without interfering with left ventricular filling, so the relative absence of pulsus paradoxus is not surprising. This observation is consistent with much of the clinical experience of patients with loculated effusion and tamponade (2,11,15,24). In those studies, 8 of the 10 patients studied had clinical evidence of tamponade without pulsus paradoxus. One possible difference between regional tamponade in postoperative patients and our model is the frequent occurrence of adhesions around the right atrium and right ventricle in patients, which may limit the expansion of these chambers. In the presence of adhesions and posterior pericardial effusion, it is conceivable that in some patients, an inspiratory increase in right ventricular volume expansion does impinge on left ventricular filling, resulting in pulsus paradoxus.

Critique of the study. Acute regional tamponade was produced in an animal preparation and therefore does not precisely parallel regional tamponade in patients. Although allowed to breathe spontaneously, the animals were anesthetized, which may have altered the hemodynamic state as well as the depth and rate of respiration. As inspiratory augmentation of right ventricular volume is a prerequisite for pulsus paradoxus (28), lack of sufficient changes in right ventricular volume with respiration could diminish the observed frequency of pulsus paradoxus.

By design, we produced tamponade of the left ventricle only, whereas some patients with loculated effusion have fluid around other cardiac chambers as well. Thus, the relation of left ventricular diastolic collapse to other findings such as left atrial collapse was not explored. The presence or absence of previously described respiratory variations in flow velocities using spectral Doppler analysis (29–31) was not evaluated as part of this study.

Regional tamponade was produced by inserting a balloon in the pericardial space. This was done to ensure that the compartment was free of leakage. By measuring pressure in the pericardium anterior to the right ventricle, we were able to determine that the increased pericardial pressure was truly a local effect. One cannot comment on whether a localized effusion produced with a balloon catheter behaves in the same manner as one lined by pericardium, clots and adhesions. However, the hemodynamic conditions of the regional tamponade in this model—elevation of left ventricular diastolic pressure and pulmonary capillary wedge pressure in parallel with increasing pericardial pressure that was associated with reduced cardiac output—are evidence that tamponade was truly produced. The 20% reduction in cardiac output that accompanied the left
ventricular diastolic collapse we observed compares well with the previously reported 21% decrement in output that accompanied right ventricular diastolic collapse in circumferential effusion (19).

When interpreting the results of this study, one also must consider that the animals studied were otherwise normal, which is not always the case in patients undergoing cardiac surgery. The presence of cardiac pathologic findings may alter left ventricular diastolic pressure. Prior experimental and clinical experience with cardiac tamponade caused by circumferential effusion in the setting of elevated right heart filling pressures has shown that the appearance of right ventricular diastolic collapse might be delayed or absent despite significant hemodynamic alteration (19,32–35). It is possible, therefore, that the relation between left ventricular diastolic collapse and the hemodynamic abnormalities described in this study would not apply if the diastolic properties of the left ventricle were altered at baseline. Preliminary data suggest that the appearance of left ventricular diastolic collapse is delayed when left ventricular diastolic pressure is abruptly elevated by volume expansion (36). Further study of regional tamponade in the presence of preexisting cardiac abnormalities is required.

Clinical implications. Assessment of the patient with localized pericardial effusion is frequently difficult as the circumferential effusion (19).

References


