Cardiac Tamponade: Hemodynamic Observations in Man

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SUMMARY  Hemodynamic studies were performed before and after pericardiocentesis in 19 patients with pericardial effusion. Right atrial pressure decreased significantly, from 16 ± 4 mm Hg (mean ± SD) to 7 ± 5 mm Hg in 14 patients with cardiac tamponade. This change was accompanied by significant increases in cardiac output (3.87 ± 1.77 to 7 ± 2.2 l/min) and inspiratory systemic arterial pulse pressure (45 ± 29 to 81 ± 23 mm Hg). The remaining five patients did not demonstrate cardiac tamponade, as evidenced by lack of significant change in these hemodynamic parameters.

In all patients with tamponade, right ventricular end-diastolic pressure (rVEDP) was elevated and equal to pericardial pressure; equilibration was uniformly absent in patients without tamponade. During gradual fluid withdrawal in the tamponade group, significant hemodynamic improvement was largely confined to the period when right ventricular filling pressure remained equilibrated with pericardial pressure. In 10 patients with tamponade and pulsus paradoxus, pulmonary arterial wedge pressure (PAW) was equal to pericardial pressure except during early inspiration and expiration when it was transiently less and greater, respectively; however, inspiratory right atrial pressure never fell below pericardial pressure. In these 10 patients, rPAW decreased significantly following pericardiocentesis (P < 0.001). In the remaining four patients with tamponade but without pulsus paradoxus, all of whom had chronic renal failure, PAW was consistently higher than pericardial pressure or rVEDP and did not decrease after pericardiocentesis.

These data tend to confirm the hypothesis that in patients with tamponade, the venous pressure required to maintain any given cardiac volume is determined by pericardial rather than ventricular compliance. When pericardial compliance determines diastolic pressure in both ventricles, relative filling of the ventricles will be competitive and determined by their respective venous pressures (pulmonary vs systemic), which vary with respiration and alternately favor right and left ventricular filling. This results in pulsus paradoxus. However, if pulmonary arterial wedge pressure is markedly elevated before the onset of tamponade, as in patients with chronic renal failure, then pericardial compliance may only determine right ventricular filling pressure. In such cases, pulsus paradoxus may be absent.

MUCH OF OUR KNOWLEDGE of the pathophysiologic mechanisms in cardiac tamponade is from observations in experimental animals. Only a few clinical hemodynamic studies consisting of small numbers of patients have been reported in this disorder. This study attempted to extend these clinical hemodynamic observations in a larger number of subjects with pericardial effusion. While our findings tend to confirm previous experimental observations, the varied pathology associated with the effusion has allowed definition of hemodynamic subsets which have relevance to both the pathophysiology and clinical manifestations of cardiac tamponade.

Materials and Methods

The patient population of this report included 19 patients with pericardial effusion who underwent pericardiocentesis for diagnostic or therapeutic purposes. Clinical data for the 19 patients are summarized in table 1. All patients were in sinus rhythm.

Hemodynamic studies were performed in the supine position. All patients underwent right heart catheterization with No. 7F Lehman catheters. Retrograde left heart catheterization was performed in four patients. Systemic arterial pressure was recorded from a PE-160 catheter introduced percutaneously into the left brachial artery. Micromanometer pressures (8F Millar catheters) were also recorded from the right ventricle in five patients and from the left ventricle in one patient; these pressures were referenced to zero mm Hg by superimposing micromanometer and fluid-filled pressures during slow rates of pressure change. The zero reference level for Statham P-23 db strain gauges was 5 cm below the sternal angle. Cardiac output determinations were performed in duplicate by the dye dilution method; indocyanine green was injected into the pulmonary artery and sampled from the brachial artery using a Model 103 or 140 Gilford densitometer. Pressures were recorded on an Electronics for Medicine DR-12. Respiratory phase was determined by a nasal thermistor.

Following initial right heart catheterization, a no. 17 pericardiocentesis needle was introduced into the pericardial space using a subxiphoid approach and unipolar electrocardiographic monitoring. Under fluoroscopic control, a guide wire was introduced through the needle. The latter was withdrawn and a PE-160 radiopaque catheter was advanced over the guide wire into the pericardial space. This catheter had an end hole and six side holes, 1 cm apart, arranged in a spiral pattern. The catheter was then connected to a Statham strain gauge. Systemic arterial, right atrial, right ventricular, pulmonary arterial and wedge pressures were recorded simultaneously with pericardial pressure, and a baseline cardiac output was obtained. Pericardial fluid
was then aspirated as completely as possible in the supine position as well as the head end of the bed elevated to 30°. After a period of five to 15 minutes, pressure sampling at the above sites was repeated. Cardiac output was measured after pericardiocentesis in 13 patients.

Inspiratory and expiratory systemic arterial pressures were the average lowest and highest systolic values, respectively, recorded during five consecutive quiet respiratory cycles. Pulsus paradoxus was calculated as:

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Pulsus \ paradoxus \% = \left( \frac{\text{expiratory systolic pressure} - \text{inspiratory systolic pressure}}{\text{expiratory systolic pressure}} \right) \times 100
\]

Diagnostic pulsus paradoxus was greater than 10%. Statistical comparisons were performed using paired and grouped data. The significance of differences was assessed using the two-tailed Student \( t \) test.

Results

Two groups of patients could be distinguished based upon the hemodynamic response to pericardiocentesis. Fourteen of 19 subjects (table 1) manifested cardiac tamponade as evidenced by a significant \((P < 0.001)\) decrease in right atrial pressure averaging 10 mm Hg (range: -6 to -15 mm Hg); this decrease was accompanied by significant \((P < 0.001)\) increases in inspiratory systemic arterial pulse pressure and cardiac output (fig. 1). In the remaining five patients, no significant changes in these hemodynamic parameters were observed. In all patients with tamponade, right ventricular end-diastolic pressure \((RVEDP)\) exceeded 7 mm Hg, and was equal to pericardial pressure. (fig. 2)

Pericardial fluid was gradually removed in 50 ml aliquots in five patients with tamponade. The most significant hemodynamic improvement occurred during the initial withdrawals (figs. 3 and 4). After the decrease of pericardial below right atrial pressure or \(RVEDP\), changes in stroke volume and systolic arterial pressure were insignificant. Although pericardial pressure continued to decline after this separation occurred, right atrial pressure showed essentially no further change.

In 10 of 14 patients with tamponade, left ventricular filling pressure, measured as pulmonary arterial wedge pressure, also tended to equilibrate with pericardial pressure except during the early portion of each respiratory phase. During early inspiration and expiration, wedge pressures were transiently less and greater than pericardial pressure, respectively (fig. 5). In contrast, inspiratory mean right atrial pressure was never observed to fall below pericardial pressure.
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Figure 1. Hemodynamic changes before and after pericardiocentesis in patients with (closed circles) and without (open circles) cardiac tamponade. The horizontal bars indicate mean values; changes (paired data) were significant only for those with tamponade. Pre = pre-pericardiocentesis; Post = post-pericardiocentesis; RAm = mean right atrial pressure; CO = cardiac output; Insp Puls Press = inspiratory arterial pulse pressure. *P < 0.001.

Figure 2. Pressure tracings before pericardiocentesis in a patient whose hemodynamic data are illustrated in figure 4. Upper panel: simultaneous intrapericardial (IPP) and micromanometer right ventricular (RV) and left ventricular (LV) pressures. All pressures are equilibrated during ventricular diastole. Lower panel: brachial arterial pressure; pulsus paradoxus is present, as evidenced by a 28% decrease in systolic pressure during inspiration. BA = brachial arterial pressure.
Following pericardiocentesis, these 10 patients demonstrated a significant decrease in left ventricular filling pressure averaging 10 mm Hg (range: -2 to -20 mm Hg; \( P < 0.001 \)). All patients initially manifesting equilibration of both ventricular filling pressures with an elevated pericardial pressure had diagnostic levels of pulsus paradoxus as illustrated in figure 2, where right and left ventricular pressures were recorded using micromanometer catheters. When serial determinations of left ventricular filling pressure were obtained in two patients with pulsus paradoxus, this arterial pressure sign was found to persist as long as the decreasing ventricular filling and pericardial pressures remained equilibrated. When pericardial fell below left ventricular filling pressure, diagnostic levels of pulsus paradoxus disappeared (fig. 4).

The remaining four patients (patients 11–14) with tamponade all had chronic renal failure. Before fluid was aspirated from the pericardium, left ventricular filling pressure was consistently higher than right (fig. 6). These four patients did not manifest pulsus paradoxus, despite elevations of pericardial pressure ranging from 11–18 mm Hg (fig. 7). Left ventricular filling pressure was not changed by pericardiocentesis and averaged 17 mm Hg (range: 14–19 mm Hg) as compared to a value to 6 mm Hg (range: -2 to 12 mm Hg) after pericardiocentesis in those with paradoxus. Post-pericardiocentesis decreases in right atrial pressure in these four patients averaged 8 mm Hg (range: -6 to -11 mm Hg), which was not significantly different from the pulsus paradoxus group.

Five patients with pericardial effusion did not have tamponade, and none evidenced equilibration of pericardial and RVEDPs. The maximal change in right atrial pressure with pericardiocentesis was -1 mm Hg. Three had elevated RVEDPs of 11, 14 and 14 mm Hg, but diastolic pericardial pressure was less than this value by 4, 7 and 9 mm Hg, respectively.

**Discussion**

A uniform hemodynamic finding in patients with cardiac tamponade was equilibration of elevated pericardial and RVEDPs. During gradual pericardial fluid withdrawal, rises in stroke volume and systemic arterial pressure were most marked during equilibration, and tended to become negligible after the fall of pericardial below ventricular filling pressure. Hemodynamic improvement was not observed after fluid withdrawal in other subjects who manifested elevation but not equilibration of ventricular filling and pericardial pressures.

Consideration of the pericardial volume-pressure relationship suggests the characteristic elevation and equilibration of pericardial and ventricular filling pressures would be expected to occur together in cardiac tamponade. Pericardial pressure is determined by the compliance characteristics of the pericardium and the total intrapericardial volume. This volume consists of the pericardial fluid and heart volumes. When accumulation of intrapericardial fluid occurs, the total pericardial volume increases and venous pressure remains relatively constant until it equilibrates with rising pericardial pressure. Beyond this point, the pericardial volume-pressure curve is equal to or steeper than that of the ventricles. With further pericardial fluid increments beyond the equilibration point, pericardial pressure will remain constant if there is a corresponding decrease in cardiac pressure.
Figure 4. Hemodynamic changes during serial fluid withdrawals in a 22-year-old male with tamponade due to uremic pericarditis. Diagnostic levels of pulsus paradoxus persist as long as left ventricular end-diastolic pressure (LVEDP) remains equilibrated with pericardial pressure. RVEDP = right ventricular end-diastolic pressure; EXP = expiration; INSP = inspiration.

Figure 5. Relation between intrapericardial pressure (IPP), right atrial (RA) and pulmonary arterial wedge (PAW) pressures in a 53-year-old woman with tamponade and pulsus paradoxus due to uremic pericarditis. Mean and phasic pressures are shown in the upper and lower panels, respectively. During inspiration, mean RA pressure is essentially equal to pericardial pressure while PAW pressure transiently falls below pericardial pressure. During expiration, RA pressure remains essentially equal to pericardial pressure while PAW transiently rises above the latter. INSP = inspiration; EXP = expiration.
volume. If this situation were obtained in cardiac tamponade, there would be a precipitous fall in stroke volume and no further rise in pericardial pressure. On the other hand, if cardiac volume is maintained, pericardial pressure will rise steeply without a change in stroke volume. Cardiac volume can only be maintained if the necessary pressure required by the tense pericardium is generated by the venous bed. If the compensatory rise in venous pressure is insufficient to maintain cardiac volume, then stroke volume will fall precipitously.

FIGURE 7. Brachial arterial pressure pre- (left panel) and post-pericardiocentesis (right panel) in a patient whose other hemodynamic findings are illustrated in fig. 6. Brachial arterial pressure (BA) and right atrial pressure (RA) were recorded at a full scale of 200 mm Hg and 40 mm Hg, respectively. Left panel: pulsus paradoxus is absent despite the presence of tamponade; the inspiratory decline in arterial pressure is 10 mm Hg, or 6% of the expiratory arterial pressure. Right panel: pulsus paradoxus is still absent, but inspiratory arterial pulse pressure has increased from 54 to 79 mm Hg. EXP = expiration; INSP = inspiration.
decreases in spite of the rise in pericardial pressure; this is the situation actually encountered and illustrated in figure 4. Thus, in cardiac tamponade, the absolute level of pericardial pressure is passively determined by venous pressure, and the two are identical.

Ordinarily, left ventricular filling pressure is higher than right ventricular filling pressure, but the difference is small. Therefore, as cardiac tamponade progresses, rising pericardial and right ventricular diastolic pressure will quickly equilibrate with left ventricular filling pressure. Since both ventricles must fill against the same pericardial stiffness, the relative filling of these chambers is determined by their respective filling pressures. At this point, the filling pressures of the right and left ventricles must be equal, or a dys-equilibrium will result. Hence, a characteristic hemodynamic finding in the catheterization laboratory is equalization of diastolic pressures in all chambers of the heart. This was true for most, but not all, patients with tamponade. Some patients with chronic renal failure had significantly higher left ventricular filling pressures; the elevated and equilibrated pericardial and right ventricular filling pressures had not yet risen to the level of left ventricular diastolic pressure. In these subjects, pericardiocentesis resulted in a significant decrease in right, but not left, ventricular filling pressures. However, hemodynamic embarrassment requiring pericardial fluid aspiration was clearly present in these patients. These data suggest that when left ventricular pressure is markedly elevated before tamponade, as might be encountered in uremic subjects, the compensatory rise in systemic venous pressure during pericardiocentesis may fail to maintain cardiac output before RVEDP rises to the level of left ventricular filling pressure. In this situation, cardiac compression is largely restricted to the right side of the heart.

A characteristic feature in patients with isolated right heart tamponade was the absence of pulsum paradoxus. The mechanism of pulsum paradoxus in cardiac tamponade has been extensively studied, using a variety of techniques over many years. While the details differ, two major pathophysiologic alterations emerge from the published data: 1) a limitation of ventricular volume expansion so that inspiratory increases in right ventricular filling tend to decrease that of the left,\(^5\)\(^,\)\(^6\)\(^,\)\(^7\)\(^,\)\(^8\) and 2) an inspiratory decrease in pulmonary venous below pericardial and left atrial pressure impeding filling of the left ventricle.\(^9\)\(^,\)\(^10\)\(^,\)\(^11\)

Our data are compatible with the following hypothesis for pulsum paradoxus production, and suggest the above theories are complementary rather than mutually exclusive. When cardiac tamponade is severe enough to embarrass both ventricles, the operative portion of the pericardial volume-pressure curve is steeper than that of either ventricle; therefore, flow into both ventricles takes place against a common stiffness. The relative filling of these cardiac chambers will be competitive and determined by their respective filling pressures. During inspiration, pulmonary venous pressure transiently decreases below systemic, favoring greater filling of the right ventricle which, in turn, impedes left ventricular filling. Our findings did not directly document this respiratory venous pressure differential. During inspiration, pulmonary arterial wedge pressure briefly fell below pericardial pressure, which was shown to closely approximate right atrial pressure. Expiration results in a reversal of inspiratory changes, i.e., pulmonary arterial wedge rises to or above systemic venous pressure with relative increases and decreases in left and right ventricular filling, respectively. These respiratory changes are transient and limited to the initial phases of inspiration and expiration. The demonstrated pressure differences between wedge and pericardial pressures were probably exaggerated, as they were measured with fluid-filled catheters. The actual differences between extrapericardial pulmonary venous and pericardial pressure might be minimal and difficult to quantify with conventional techniques.

The two conditions apparently required for the production of pulsum paradoxus in tamponade are: 1) filling of both ventricles against a common stiffness, and 2) respiratory changes in venous pressure differential (systemic vs pulmonary), alternately favoring right and left ventricular filling. In cardiac tamponade restricted to the right heart, pericardial is equal to right ventricular filling pressure since both are determined by the compliance of the fluid filled pericardium; left ventricular filling pressure, however, is determined by the compliance of the left ventricle. Since the ventricles are not filling against a common pericardial stiffness and left ventricular filling pressure is constantly higher than the right regardless of respiratory phase, pulsum paradoxus is absent. In the presence of a common stiffness to ventricular filling determined by the pericardium, pulsum paradoxus may not appear if respiratory alterations in ventricular volume produced by the venous pressure differential are abolished. Shabetai has shown\(^7\) that pulsum paradoxus will disappear when a constant return to the right heart is maintained during tamponade. The clinical implication of our finding is that cardiac tamponade requiring pericardiocentesis may be present in the absence of pulsum paradoxus when it is largely confined to the right heart. Further elevations of pericardial pressure eventually cause significant compression of the left heart; this event will be associated with pulsum paradoxus, implying a more severe state of cardiac tamponade.

References
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