Heart Failure

Hemodynamic Effects of Volume Expansion in Patients With Cardiac Tamponade

Jaume Sagristà-Sauleda, MD; Juan Angel, MD; Antonia Sambola, MD; G. Permanyer-Miralda, MD

Background—Volume expansion has been proposed as an alternative treatment for cardiac tamponade; however, the scientific evidence for this recommendation is very poor.

Methods and Results—Forty-nine unselected patients (23 males; age 55±16 years) with large pericardial effusion and hemodynamic tamponade underwent fluid overload with intravenous administration of 500 mL of normal saline over 10 minutes. Cardiac index and intrapericardial, left ventricular end-diastolic, right atrial, and right ventricular end-diastolic pressures were measured during basal state (tamponade), after fluid overload, and after pericardiocentesis. Twenty-eight patients (57%) had physical signs of tamponade, and 10 (20%) were hypotensive. Size of pericardial effusion was 31±13 mm. Initial mean arterial pressure was 88±21 mm Hg, and cardiac index was 2.46±0.80 L min⁻¹ m⁻². Intrapericardial pressure was 8.31±5.98 mm Hg. Volume expansion caused a significant increase in mean arterial pressure (from 88±21 to 94±23 mm Hg, $P=0.003$) and cardiac index (from 2.46±0.80 to 2.64±0.68 L min⁻¹ m⁻², $P=0.013$), as well as in intrapericardial pressure (from 8.31±5.98 to 11.02±6.27 mm Hg, $P=0.0001$), right atrial pressure (from 9.76±5.91 to 12.82±6.34 mm Hg, $P=0.0001$), and left ventricular end-diastolic pressure (from 14.21±5.97 to 19.48±6.19 mm Hg, $P=0.0001$). Cardiac index increased by >10% in 23 patients (47%), remained unchanged in 11 (22%), and decreased in 15 (31%). No patient developed clinical complications. Predictors of this favorable response were systolic blood pressure <100 mm Hg and low cardiac index.

Conclusions—Approximately one half of patients with cardiac tamponade develop a significant increase in cardiac output after volume overload. Low systolic blood pressure (<100 mm Hg) at baseline was the simplest clinical finding that was predictive of this favorable response. (Circulation. 2008;117:1545-1549.)

Key Words: cardiac tamponade ■ hemodynamics ■ pericardium

D ecompensated cardiac tamponade is a medical emergency that requires urgent therapy. Once diagnosed on a clinical and echocardiographic basis, therapy must consist of rapid pericardial drainage by needle pericardiocentesis or a surgical procedure. Occasionally, a situation may be encountered in which these procedures cannot be readily performed, and alternative methods could then be considered as interim therapy. Intravascular volume expansion, alone or in combination with vasodilator drugs, is the most common therapeutic procedure proposed as emergency therapy for cardiac tamponade as a useful temporizing measure while the patient is prepared for pericardiocentesis. Although this indication is mentioned in most cardiology textbooks and in the recent guidelines on the diagnosis and management of pericardial diseases of the European Society of Cardiology, there is scanty scientific evidence for this recommendation. In fact, only experimental studies in animals have shown an increase in cardiac output and improvement of blood pressure with expansion of central blood volume, but the validity of this experience in humans with cardiac tamponade is doubtful. Surprisingly, there is a striking paucity of relevant data on human beings. A limited number of isolated case reports have shown no benefit of volume expansion in patients with low-pressure cardiac tamponade, a situation in which some benefit of this therapeutic procedure could have been expected. We have found only a single series of 11 patients with cardiac tamponade treated with volume expansion. The authors found no significant changes in mean values of heart rate, right atrial and arterial pressure, systemic vascular resistance, pulsatia paradoxus, or cardiac output, but individual values for each patient and intrapericardial pressure were not provided in the report.

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Here, we report our experience with volume expansion in a large series of patients with tamponade across a wide range of values of intrapericardial (and accordingly, intracavitary) pressures. We especially wished to investigate whether some subgroups of patients with tamponade could obtain benefit from (or be harmed by) this therapeutic procedure.
Methods

We studied 49 unselected, nonconsecutive patients with large pericardial effusion (sum of anterior and posterior echo-free spaces at end diastole >20 mm) who were referred to the cardiac catheterization laboratory for pericardiocentesis and who met hemodynamic criteria of tamponade, that is, equalization between intrapericardial and right atrial pressure with right transmural pressure <2 mm Hg. Patients in a critical situation, patients with additional heart disease, and those undergoing hemodialysis were not included in the study. During the period in which the present series was collected, 11 additional patients underwent pericardiocentesis at the catheterization laboratory without fluid overload because of the presence of valvular mitroaortic disease in 2 patients, effusive-constrictive pericarditis in 2, left ventricular dysfunction in 3, pulmonary hypertension in 1, uremia in 2, and unknown reasons in 1 patient. The present study included 23 men and 26 women with an age range of 23 to 83 years (median age 55±16 years). Twenty-eight patients (57%) had physical signs of tamponade (jugular venous distension, pulsus paradoxus in 21 patients, pulsus paradoxus in 22, and both in 15), and 10 patients (20%) were hypotensive (systolic arterial pressure <100 mm Hg). The cause of pericardial effusion was acute idiopathic pericarditis in 17 patients (35%), neoplasia in 7 (14%), renal insufficiency in 2 (4%), chronic idiopathic pericardial effusion in 16 (33%), and miscellaneous in 7 (14%). No patient had hyperacute tamponade caused by cardiac rupture, aortic dissection, or thoracic trauma. The size of pericardial effusion as measured by echocardiographic criteria (sum of anterior and posterior echo-free spaces at end diastole) was 31±13 mm; 18 patients (37%) had right atrial collapse, 10 (20%) had right ventricular collapse, and 9 (18%) had combined right atrial and right ventricular collapses.

All patients gave informed, written consent for pericardiocentesis and cardiac catheterization. Before the performance of this procedure, a venous line was implanted, but a substantial amount of fluid was not administered to any patient. In brief, the protocol for the catheterization procedure was as follows.16 Two 5F pigtail angiographic catheters were used for pericardiocentesis and to record intrapericardial and left ventricular pressures simultaneously. Right atrial or right ventricular pressures were recorded with a multipurpose 5F catheter. Hemodynamic pulsus paradoxus was measured from a recording of femoral artery pressure obtained during spontaneous respiration. The average of the differences of all measurements of instantaneous right atrial and intrapericardial pressures during 3 spontaneous respiratory cycles (taken every 0.2 seconds during the second half of expiration) was considered the right atrial transmural pressure. Cardiac output was measured by the thermodilution technique.

After all initial measurements were performed, we manually administered 500 mL of intravenous normal saline over a 10-minute period, because this was a standard therapeutic recommendation for the emergency treatment of acute cardiac tamponade.15 All pressures were then remeasured, and cardiac output was again calculated. Thereafter, the pericardial fluid was completely withdrawn, and a final set of pressure and cardiac output measurements were taken.

Statistical Analysis

Descriptive analysis was performed with mean±SD and range for continuous variables and absolute and relative frequencies of patients in each category for categorical variables. Differences between baseline and postintervention parameters in the same group were evaluated with the use of the paired Student’s t test for continuous variables and the χ2 test for categorical variables. The Kruskal-Wallis test was used to compare the influence of volume expansion on continuous variables among the 3 subgroups of cardiac index. A 2-tailed value of P<0.05 was considered significant. Statistical analysis was performed with the statistical package SPSS 11.0 (SPSS Inc, Chicago, Ill). We investigated whether certain clinical (presence of jugular venous distension, pulsus paradoxus, arterial pressure), echocardiographic (amount of pericardial effusion, right atrial or right ventricular collapse), and hemodynamic (right atrial pressure, left ventricular end-diastolic pressure, cardiac index) variables were predictors of hemodynamic benefit (defined as an increase in cardiac index >10%).

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

All 49 patients met hemodynamic criteria of tamponade by showing near equilibration within 2 mm Hg of mean right atrial pressure and intrapericardial pressure, with right transmural pressure <2 mm Hg. Twenty-one patients fulfilled 1 criterion for low-pressure cardiac tamponade, ie, intrapericardial pressure <7 mm Hg at baseline during tamponade.17 Hemodynamic parameters during pericardiocentesis are summarized in Table 1. Volume expansion caused a significant increase in mean arterial pressure (from 88±21 to 94±23 mm Hg, P=0.003). Heart rate did not change. Intrapericardial pressure rose from 8.31±5.98 to 11.02±6.27 mm Hg (P=0.0001), which paralleled the increase in right atrial pressure (from 9.76±5.91 to 12.82±6.34 mm Hg, P=0.0001) and left ventricular end-diastolic pressure (from 14.21±5.97 to 19.48±6.19 mm Hg, P=0.0001). Right atrial pressure and left ventricular end-diastolic pressure increased in all patients except 3, in whom it remained unchanged. Hemodynamic pulsus paradoxus decreased from 6.19±5.98 to 4.07±3.26 mm Hg (P=0.0001). Heart rate decreased from 94±23 to 88±18 bpm (P=0.013). Cardiac index increased from 2.64±0.68 to 3.14±0.85 L·min⁻¹·m⁻² (P=0.0013). Cardiac index increased by >10% in 23 patients (47%), remained unchanged (increase of <10%) in 11 patients, and decreased in 15 patients (Table 2). As shown in Table 2, compared with patients whose cardiac output did not increase after volume expansion, patients in whom volume expansion resulted in an

<table>
<thead>
<tr>
<th>Table 1. Effects of Volume Expansion and Pericardiocentesis in Patients With Cardiac Tamponade (Hemodynamic Parameters)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
</tr>
<tr>
<td>Intrapерicardial pressure, mm Hg</td>
</tr>
<tr>
<td>Mean right atrial pressure, mm Hg</td>
</tr>
<tr>
<td>Right transmural pressure, mm Hg</td>
</tr>
<tr>
<td>Left ventricle diastolic pressure, mm Hg</td>
</tr>
<tr>
<td>Hemodynamic pulsus paradoxus, mm Hg</td>
</tr>
<tr>
<td>Cardiac index, L·min⁻¹·m⁻²</td>
</tr>
</tbody>
</table>

*Differences are calculated versus baseline parameters. Values are expressed as mean±SD. *P<0.05.
increase in cardiac index > 10% had a lower arterial pressure and lower cardiac index before volume expansion. By contrast, the presence of jugular venous distension or pulsus paradoxus, the amount of pericardial effusion by echocardiography, the presence of right atrial or right ventricular echocardiographic collapse, and the level of right atrial pressure and left ventricular end-diastolic pressure were not associated with a change in cardiac output.

Next, we analyzed the response of cardiac output to volume expansion in 3 subsets of patients according to the level of arterial pressure (<100 mm Hg, between 100 and 120 mm Hg, and >120 mm Hg) before volume expansion (Table 3). Only those patients with a baseline arterial pressure <100 mm Hg showed a significant increase in cardiac output with volume expansion (increase in cardiac index from 2.24±1.03 to 2.66±0.81 L · min⁻¹ · m⁻², P=0.014). Remarkably, left ventricular end-diastolic pressure increased significantly in all 3 subsets of patients (P=0.0001). No patient reported either subjective improvement or worsening of symptoms with saline administration. No patient developed clinical complications. In contrast to the modest changes after volume expansion, pericardiocentesis was followed by a dramatic decrease in intrapericardial pressure, right atrial pressure, left ventricular end-diastolic pressure, and pulsus paradoxus and by a marked increase in cardiac index (from 2.45±0.80 to 3.13±0.85 L · min⁻¹ · m⁻², P=0.0001; Table 1).

### Discussion

The optimal treatment of cardiac tamponade is pericardial drainage, either by needle pericardiocentesis or by a surgical procedure. However, occasionally, these procedures are not readily available, and alternative methods can be considered as interim therapy. Intravascular volume expansion is the measure usually contemplated for this purpose. It is mentioned in most cardiology textbooks¹⁻⁵ and in the recent guidelines on the diagnosis and management of pericardial diseases of the European Society of Cardiology.⁶ However, the scientific evidence for its clinical effectiveness is weak, because only isolated case reports¹⁰⁻¹⁴ and a small series of 11 patients¹³ have been published. Accordingly, we attempted to investigate the possible clinical benefits, as well as the possible harmful effects, of intravascular volume expansion in a larger series of patients with medical cardiac tamponade.

The most remarkable finding of the present study (Table 1) is that in patients with cardiac tamponade, volume expansion actually results in some rise in arterial pressure and cardiac index, but as a whole, the magnitude of these changes is small. In fact, no patient reported symptomatic improvement with this maneuver. On the other hand, volume expansion consistently caused a significant increase in intrapericardial pressure, right atrial pressure, and left ventricular end-diastolic pressure. Although no patient reported a worsening of dyspnea or the

### Table 2. Effect of Volume Expansion on Cardiac Index: Changes in Cardiac Index With Volume Expansion in 49 Patients

<table>
<thead>
<tr>
<th>Clinical Echocardiographic and Hemodynamic Data at Baseline</th>
<th>&gt;10% Increase in CI (n=23)</th>
<th>&lt;10% Increase in CI (n=11)</th>
<th>Decrease in CI (n=15)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jugular venous distension</td>
<td>7 (33)</td>
<td>6 (29)</td>
<td>8 (38)</td>
<td>NS</td>
</tr>
<tr>
<td>Paradoxical pulse</td>
<td>9 (41)</td>
<td>8 (36)</td>
<td>5 (23)</td>
<td>NS</td>
</tr>
<tr>
<td>Arterial pressure, mm Hg</td>
<td>114±26</td>
<td>148±44</td>
<td>131±26</td>
<td>0.017</td>
</tr>
<tr>
<td>Amount of pericardial effusion, mm</td>
<td>35.2±8.7</td>
<td>27±2.5</td>
<td>26±20.8</td>
<td>NS</td>
</tr>
<tr>
<td>RA collapse</td>
<td>6 (26)</td>
<td>5 (45)</td>
<td>7 (46.7)</td>
<td>NS</td>
</tr>
<tr>
<td>RV collapse</td>
<td>2 (8.6)</td>
<td>3 (27.3)</td>
<td>5 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>RA collapse and RV collapse</td>
<td>2 (8.7)</td>
<td>3 (27.3)</td>
<td>4 (26.7)</td>
<td>NS</td>
</tr>
<tr>
<td>RAP, mm Hg</td>
<td>8.57±5.06</td>
<td>10.45±7.97</td>
<td>11.07±5.44</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>12.96±4.79</td>
<td>15.45±7.63</td>
<td>15.29±6.34</td>
<td>NS</td>
</tr>
<tr>
<td>CI, L · min⁻¹ · m⁻²</td>
<td>2.13±0.54</td>
<td>2.28±0.60</td>
<td>3.08±0.94</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Values are expressed as mean±SD or n(%). CI indicates cardiac index; RA, right atrial; RV, right ventricular; RAP, right atrial pressure; and LVEDP, left ventricular end-diastolic pressure.

### Table 3. Effect of Volume Expansion on Cardiac Index and Left Ventricular End-Diastolic Pressure in Different Subgroups of Patients According to Systolic Blood Pressure

<table>
<thead>
<tr>
<th>SBP baseline</th>
<th>Before Volume Expansion</th>
<th>After Volume Expansion</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;100 mm Hg</td>
<td>2.24±1.03</td>
<td>2.66±0.81</td>
<td>0.014</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>12.78±5.23</td>
<td>17.89±4.01</td>
<td>0.0001</td>
</tr>
<tr>
<td>100–120 mm Hg</td>
<td>2.33±0.70</td>
<td>2.57±0.73</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>14.36±5.6</td>
<td>19.71±6.83</td>
<td>0.0001</td>
</tr>
<tr>
<td>&gt;120 mm Hg</td>
<td>2.64±0.75</td>
<td>2.65±0.66</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>14.91±6.75</td>
<td>20.35±6.66</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Values are expressed as mean±SD. SBP indicates systolic blood pressure; LVEDP, left ventricular end-diastolic pressure.
development of features of pulmonary edema, the possibility that the increase in left ventricular diastolic pressure could cause pulmonary congestion is a matter of some concern, especially if pericardiocentesis is not rapidly available. Therefore, although some experimental studies in animals with tamponade have reported an increase in cardiac output and blood pressure with expansion of central blood volume,7–9 this maneuver appears to afford a limited clinical benefit in the overall population of patients with cardiac tamponade.

The present findings closely reproduce the observations reported by Kerber et al,15 who found very limited hemodynamic benefits of volume expansion in a small series of 11 patients with cardiac tamponade. Those authors found no significant increase in mean arterial pressure or cardiac output; however, in their study,15 individual values of cardiac output, right atrial pressure, left ventricular end-diastolic pressure, and intrapericardial pressure were not reported. In the present study, we systematically recorded the entire set of hemodynamic data in all patients to investigate whether some subgroups of patients with cardiac tamponade benefited from volume expansion. It could be hypothesized, for instance, that patients in whom tamponade is established at a relatively low intrapericardial and right atrial pressure (the so-called low-pressure cardiac tamponade) could experience improvement with volume expansion. Accordingly, we assessed whether certain clinical, echocardiographic, or hemodynamic data during tamponade predicted benefit from volume expansion. Only 23 (48%) of 49 patients obtained a theoretically significant benefit in terms of a gain in cardiac index (>10%) after volume expansion in patients with lower arterial blood pressure and lower cardiac index during tamponade (Table 2). In fact, this benefit was only seen in patients with arterial pressure <100 mm Hg during tamponade. Neither the amount of pericardial effusion measured by echocardiography or the presence of right atrial or right ventricular echocardiographic collapse was predictive of a favorable response. In the design of the present study, only clinical and catheterization data were considered, because the procedure had to be performed in the catheterization laboratory, and echocardiographic data were retrieved retrospectively from clinical records. Therefore, the latter should be considered with some caution.

Interestingly, we found that low right atrial pressure during tamponade was not predictive of a significant gain in cardiac index with volume expansion. This observation is in agreement with isolated case reports that found no benefit of volume expansion in patients with low-pressure cardiac tamponade.10–14 A putative explanation for this phenomenon is that although volume expansion forces the filling of right cardiac chamber and consequently might be expected to cause an increase in cardiac output, the increase in right-chamber volume in a limited total pericardial volume can produce a compression of left cardiac chambers, with a corresponding fall in cardiac output. In fact, in the present series, cardiac output actually decreased in 15 (30%) of the 49 patients. Another remarkable finding is that in the whole series and in all subsets of patients we analyzed, left ventricular end-diastolic pressure consistently rose after volume expansion. The Figure shows an illustrative example of this hemodynamic phenomenon.

It could be hypothesized that a greater amount of volume expansion (for instance, 1000 mL of saline) would have resulted in a greater increase in cardiac output. This hypothesis was not tested in the present study. However, filling
pressures increased in nearly all patients after the infusion of 500 mL of saline; therefore, an undue amount of volume expansion could have resulted in an excessive and perhaps dangerous elevation in left ventricular diastolic pressure.

No patient in the present series had acute tamponade that resulted from stab wounds of the chest or cardiac rupture with severe hypovolemia, a situation in which symptomatic improvement and increases in arterial pressure with saline infusion have been reported.16 The patients included in the present series were not consecutive, because the performance of intravascular volume expansion was dependent on the schedule and availability of a skilled clinical operator. However, these patients were not selected on any clinical basis, and only those with hyperacute tamponade secondary to chest wounds, aortic dissection, or cardiac rupture and patients with very poor clinical tolerance and in whom urgent pericardial drainage was mandatory were excluded from the present study. We believe, therefore, that patients included in the present series were representative of the entire population of patients with moderately severe medical cardiac tamponade.

In summary, in some patients with medical tamponade, volume expansion causes a significant rise in arterial pressure and cardiac output. Low systolic blood pressure was the only clinical predictor of such a favorable response, and low cardiac output as measured in the catheterization laboratory was also predictive. Therefore, volume loading can be offered to some patients as an emergency temporary measure to help stabilize a hypotensive patient while they are prepared for pericardiocentesis. This beneficial effect can be offset by a consistent increase in left ventricular end-diastolic pressure. Thus, this maneuver should not be considered for all patients with cardiac tamponade on a systematic basis.

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Disclosures
None.

References

CLINICAL PERSPECTIVE
Cardiac tamponade may be a life-threatening syndrome that requires urgent therapy. The optimal treatment is pericardial drainage, either by needle pericardiocentesis or by a surgical procedure; however, these procedures may not be readily available, and alternative methods can be considered as interim therapy. Intravascular fluid expansion has been proposed as a potentially useful procedure for this purpose, but the scientific evidence for its clinical effectiveness is very poor. The present report is based on 49 patients with large pericardial effusion and hemodynamic tamponade who underwent combined pericardiocentesis and cardiac catheterization and were submitted to fluid overload with administration of 500 mL of intravenous normal saline over a 10-minute period. At baseline, all patients met hemodynamic criteria of tamponade (equalization of intrapericardial and intracavitary pressures). Volume expansion caused a significant increase in mean arterial pressure and cardiac index, but only half of the patients had an increase in cardiac index >10%. The only predictor of this favorable response was low systolic blood pressure (<100 mm Hg). Volume expansion caused a significant increase in intrapericardial, right atrial, and left ventricular end-diastolic pressures. Remarkably, cardiac index decreased in 30% of patients, although no patient developed clinical complications. In conclusion, volume expansion should not be considered for all patients with cardiac tamponade on a systematic basis, but it can help to stabilize hypotensive patients while they are being prepared for pericardiocentesis. This beneficial effect can be offset by a consistent increase in left ventricular end-diastolic pressure.
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