Low-Pressure Cardiac Tamponade
Clinical and Hemodynamic Profile

Jaume Sagristà-Sauleda, MD; Juan Angel, MD; Antonia Sambola, MD; Joan Alguersuari, MD; Gaietà Permanyer-Miralda, MD; Jordi Soler-Soler, MD

Background—Low-pressure cardiac tamponade is a form of cardiac tamponade in which a comparatively low pericardial pressure results in cardiac compression because of low filling pressure. This syndrome is poorly characterized because only isolated cases have been reported. We conducted a study of its clinical and hemodynamic profiles.

Methods and Results—From 1986 through 2004, we evaluated all patients at our institution with combined pericardiocentesis and cardiac catheterization. We identified those patients who fulfilled catheterization-based criteria of low-pressure cardiac tamponade and compared their clinical and catheterization data with those of patients with classic tamponade. A total of 1429 patients with pericarditis were evaluated, 279 of whom underwent combined pericardiocentesis and catheterization. Criteria of low-pressure cardiac tamponade were met in 29, whereas 114 had criteria of classic cardiac tamponade. Patients with low-pressure tamponade less frequently had clinical signs of tamponade, but the rate of constitutional symptoms, use of diuretics, and echocardiographic findings of tamponade were similar in both groups. Patients with low-pressure tamponade showed a significant increase in cardiac output after pericardiocentesis, but they usually had less severe cardiac tamponade compared with patients with classic tamponade. Prognosis was related mainly to the underlying disease.

Conclusions—Low-pressure cardiac tamponade was identified in 20% of patients with catheterization-based criteria of tamponade. Clinical recognition may be difficult because of the absence of typical physical findings of tamponade in most patients. Although some patients are critically ill, most show a stable clinical condition. However, these patients obtain a clear benefit from pericardiocentesis. (Circulation. 2006;114:945-952.)

Key Words: cardiac tamponade ■ catheterization ■ echocardiography ■ hemodynamics ■ pericardium

Low-pressure cardiac tamponade has been described as a form of cardiac tamponade in which a comparatively low pericardial pressure results in cardiac compression in patients with intravascular fluid depletion. Such a situation was well characterized in experimental preparations, but it is not systematically assessed in reviews of cardiac tamponade.4–7 Clinically, this syndrome was first described by Antman et al8 in 1979 in an apparently hypovolemic elderly man with tuberculous pericarditis who developed cardiac tamponade. Since this initial report, only a few individual cases have been reported.9–12 Remarkably, in some of these cases, some typical manifestations of tamponade such as jugular venous distention or pulsus paradoxus were not present. This may create some difficulties in recognizing the syndrome and accounts for both the low reported prevalence and the limited available information. To assess the prevalence, clinical findings, hemodynamic profile, and evolution of this poorly known type of cardiac tamponade, we identified the cases fulfilling its diagnostic criteria in a series of 279 consecutive patients who underwent combined pericardiocentesis and cardiac catheterization over a 19-year period.

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Definitions
There is no established definition of low-pressure cardiac tamponade. The classic clinical criteria of cardiac tamponade are inadequate for that purpose because classic physical signs may be absent and, on the other hand, there are no reliable clinical signs of the pressure level at which tamponade has developed. Accordingly, in this review, we used a conventional hemodynamic definition based on our own findings during combined pericardiocentesis and cardiac catheterization. The criteria required for the diagnosis of tamponade were equalization of intrapericardial pressure and right atrial pressure with right transmural pressure <2 mm Hg. In patients fulfilling such criteria, low-pressure cardiac tamponade was diagnosed when intrapericardial pressure was <7 mm Hg before pericardiocentesis and right atrial pressure became <4 mm Hg after intrapericardial pressure had been lowered to near 0 mm Hg by pericardiocentesis (Figure 1) (see Discussion). These patients were considered group 1. Patients with intrapericardial pressure ≥7 mm Hg before pericardiocentesis and right atrial pressure ≥4 mm Hg after pericardiocentesis were considered to have classic tamponade (group 2).
was considered significant. Statistical analysis was performed using the SPSS 11.0 statistical package.

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

### Results

#### Clinical Findings

Among 1429 consecutive patients with pericarditis of any type, 279 underwent a first combined pericardiocentesis and cardiac catheterization according to our protocol. During the same period, 32 additional patients with severe clinical tamponade underwent emergency pericardiocentesis without hemodynamic monitoring in other hospital areas. Forty-seven patients were excluded because of incomplete clinical or hemodynamic information (most had been referred from other institutions only for pericardiocentesis). Fifty patients did not fulfill our hemodynamic criteria of tamponade. Thirty-nine patients were submitted to volume expansion before pericardiocentesis and thus were excluded because this maneuver precluded reliable hemodynamic evaluation after pericardiocentesis. Twenty-nine patients (10 women, 19 men; age range, 24 to 84 years; median age, 55±18 years) fulfilled our criteria for low-pressure cardiac tamponade (group 1). Pericardiocentesis had been indicated because of clinical signs of tamponade in 7 patients and dyspnea in 15 patients and for diagnostic purposes in 7 patients. Catheterization criteria of classic tamponade were fulfilled in 114 patients (56 women, 58 men; age range, 15 to 89 years; median age, 58±18 years) (group 2). In this group, pericardiocentesis had been indicated because of clinical signs of tamponade in 81 patients and dyspnea in 20 patients and for diagnostic purposes in 13 patients. Clinical findings of both groups are shown in Table 1. The only significant differences between them were higher heart rate in group 2 (P=0.0001), classic findings of cardiac tamponade (24% in group 1, 71% in group 2; P=0.0001), and origin of pericarditis, with neoplastic pericarditis and iatrogenic pericarditis being more frequent in group 2 (P=0.039). Remarkably, there were no differences in the prevalence of dehydration or in the use of diuretic, vasodilator, or antihypertensive drugs.

#### Findings at Catheterization

Before pericardiocentesis, group 1 patients had lower pulsus paradoxus (10.41±5.33 versus 23.67±12.47 mm Hg; P=0.0001), intrapericardial pressure (2.52±1.68 versus 12.93±5.42 mm Hg; P=0.0001), right atrial pressure (3.59±1.57 versus 14.63±5.42 mm Hg; P=0.0001), and left ventricular diastolic pressure (11.22±5.16 versus 18.62±5.46 mm Hg; P=0.0001) than group 2 patients, but there were no significant differences in cardiac index (2.96±1.26 versus 2.69±1.79 L/min; P=0.291) or right transmural pressure (0.07±0.26 versus 0.35±1.70 mm Hg; P=0.093) (Table 2). After pericardiocentesis, patients in both groups showed a significant increase in cardiac index (2.96±1.26 to 3.22±1.11 versus 2.69±1.78 to 3.48±1.97 L/min, respectively), although the gain was greater in group 2 (12.49±15.30% versus 35.06±37.73%; P=0.0001); group 1 patients had lower pulsus paradoxus (5.76±2.60 versus

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**Patients**

This series includes all patients meeting the criteria of low-pressure cardiac tamponade from 1986 through 2004. We identified these patients by reviewing all pericardiocentesis procedures performed in the catheterization laboratory. All patients had been admitted to our hospital ward with a diagnosis of pericardial effusion or clinical tamponade requiring pericardiocentesis according to our protocol (clinical findings of hemodynamic embarrassment or, for diagnostic purposes, with suspected purulent pericarditis or evidence of large chronic pericardial effusion). Patients with acute traumatic tamponade and associated massive hemorrhage were not included. Written informed consent for pericardiocentesis and cardiac catheterization was obtained from all patients. Since 1986, our routine practice has been to perform pericardiocentesis in the catheterization laboratory. All patients had been admitted to our hospital and submitted to volume expansion before pericardiocentesis. The number of triangles is smaller because identical values were found in 36 patients.

**Follow-Up**

Patients were monitored in the outpatient clinic by one of us after discharge and every 3 months during the first year. Subsequent follow-up depended on the underlying condition and symptomatic status.

**Statistical Analysis**

For descriptive analysis, mean±SD and range for continuous variables and absolute and relative frequencies of patients in each category for categorical variables were used. Differences between study groups were evaluated with the use of unpaired Student’s t test for continuous variables and χ² test for categorical variables. A paired t test was used when appropriate. A 2-tailed value of P<0.05 was considered significant. Statistical analysis was performed using the SPSS 11.0 statistical package.

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

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TABLE 1. Clinical Findings for the Study Groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=29)</th>
<th>Group 2 (n=114)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female/male, n</td>
<td>10/19</td>
<td>56/58</td>
<td>NS</td>
</tr>
<tr>
<td>Age, y</td>
<td>55±18</td>
<td>58±18</td>
<td>NS</td>
</tr>
<tr>
<td>General illness, n (%)</td>
<td></td>
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<tr>
<td>Diabetes</td>
<td>4 (15)</td>
<td>26 (24)</td>
<td>NS</td>
</tr>
<tr>
<td>Renal insufficiency (diabetes)</td>
<td>4 (15)</td>
<td>13 (12)</td>
<td>NS</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>4 (15)</td>
<td>34 (30)</td>
<td>NS</td>
</tr>
<tr>
<td>Dehydration</td>
<td>5 (17)</td>
<td>13 (14)</td>
<td>NS</td>
</tr>
<tr>
<td>Previous treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretic drugs (n, %)</td>
<td>5 (21)</td>
<td>31 (34)</td>
<td>NS</td>
</tr>
<tr>
<td>Vasodilators (n, %)</td>
<td>2 (8)</td>
<td>8 (9)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypotensive drugs (n, %)</td>
<td>12 (36)</td>
<td>27 (25)</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>82.5±16.5</td>
<td>95.1±16.0</td>
<td>0.0001</td>
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<tr>
<td>Arterial pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>138.9±39</td>
<td>136±33</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>69.6±14</td>
<td>73.5±12.9</td>
<td>NS</td>
</tr>
<tr>
<td>Clinical status, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>6 (20.7)</td>
<td>17 (14.9)</td>
<td></td>
</tr>
<tr>
<td>Exertional dyspnea</td>
<td>14 (48.3)</td>
<td>47 (41.2)</td>
<td></td>
</tr>
<tr>
<td>Rest dyspnea</td>
<td>5 (17.2)</td>
<td>37 (32.5)</td>
<td></td>
</tr>
<tr>
<td>Hypotension</td>
<td>4 (13.8)</td>
<td>13 (11)</td>
<td></td>
</tr>
<tr>
<td>Acute inflammatory signs*</td>
<td>10 (40)</td>
<td>54 (52)</td>
<td>NS</td>
</tr>
<tr>
<td>Clinical tamponade</td>
<td>7 (24)</td>
<td>81 (71)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Jugular venous distension</td>
<td>6 (22)</td>
<td>60 (55)</td>
<td>0.003</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>2 (7)</td>
<td>53 (50)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td></td>
<td></td>
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<tr>
<td>Amount of pericardial effusion, mm</td>
<td>30.4±11.9</td>
<td>33.6±14.3</td>
<td>0.0001</td>
</tr>
<tr>
<td>No collapses, n (%)</td>
<td>7/26 (27)</td>
<td>22/105 (21)</td>
<td>NS</td>
</tr>
<tr>
<td>Right atrial collapse, n (%)</td>
<td>20/27 (74)</td>
<td>79/104 (76)</td>
<td>NS</td>
</tr>
<tr>
<td>Right ventricular collapse, n (%)</td>
<td>9/27 (33)</td>
<td>54/105 (51)</td>
<td>NS</td>
</tr>
<tr>
<td>Exaggerated respiratory changes in mitral and tricuspid flow/n (%)</td>
<td>15/28 (54)</td>
<td>63/106 (59)</td>
<td>NS</td>
</tr>
<tr>
<td>Origin of pericarditis, n (%)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Neoplastic pericarditis</td>
<td>11 (38)</td>
<td>32 (28)</td>
<td></td>
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<tr>
<td>Renal insufficiency</td>
<td>3 (10)</td>
<td>31 (27)</td>
<td></td>
</tr>
<tr>
<td>Chronic idiopathic pericardial effusion</td>
<td>3 (10)</td>
<td>12 (11)</td>
<td>0.039</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>1 (3)</td>
<td>13 (11)</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>8 (29)</td>
<td>10 (9)</td>
<td></td>
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*Pericardial chest pain, friction rub, or fever.

8.77±4.03 mm Hg; P=0.0001), intrapericardial pressure (−3.45±1.84 versus −0.93±3.35 mm Hg; P=−0.0001), right atrial pressure (1.21±1.40 versus 9.11±4.65 mm Hg; P=0.0001), right transmural pressure (4.03±2.69 versus 7.10±4.36 mm Hg; P=0.0001), and left ventricular diastolic pressure (10.22±5.68 versus 15.19±5.68 mm Hg; P=0.0001), but there were no significant differences in cardiac index (3.22±1.11 versus 3.48±1.97 L/min; P=0.504). Figure 2 shows characteristic catheterization tracings in low-pressure cardiac tamponade and classic cardiac tamponade. Findings at pericardiocentesis and cardiac catheterization for each of the 29 patients with low-pressure cardiac tamponade are shown in Table 3, and values of intrapericardial pressure and right transmural pressure before and after pericardiocentesis are shown in Figure 3.

Therapeutic Procedures

Patients with inflammatory symptoms (pain, rub, or fever) were given oral nonsteroidal anti-inflammatory drugs. Pericardiocentesis was uneventful in all patients. The volume of pericardial fluid removed ranged from 210 to 1500 mL (mean, 607.55±267.07 mL) in group 1 and from 200 to 2000 mL (mean, 762.68±347.41 mL) in group 2. Ten patients (35%) in group 1 and 39 patients (34%) in group 2 (P=0.57) required new pericardial drainage because of pericardial fluid reaccumulation. Wide anterior pericardiotomy was performed in 6 group 1 patients 6 to 60 months (mean, 34±20 months) after pericardiocentesis (because of effusive-constrictive pericarditis in 3 patients and chronic pericardial effusion in 3) and in 16 group 2 patients 0.2 to 36 months (mean, 9.8±14.45 months) after pericardiocentesis (because of effusive-constrictive pericarditis in 5 patients, chronic pericardial effusion in 10, and recurrent cardiac tumor in 1).

Follow-Up

**Group 1**
Overall follow-up ranged from 8 days to 14.08 years (mean, 4.37±4.64 years). Two patients were lost to follow-up. Seven died between 7 months and 14 years after admission from neoplasia (5 patients) and coronary artery disease (2 patients).

**Group 2**
Overall follow-up ranged from 4 days to 14.86 years (mean, 3.02±3.91 years). Three patients were lost to follow-up. Thirty died between 9 months and 12.81 years after admission because of neoplasia (24 patients), heart failure (2 patients), coronary artery disease (2 patients), pericardietomy (1 patient), and cerebrovascular disease (1 patient).

Discussion

Cardiac tamponade has classically been considered a syndrome in which cardiac chambers are compressed by a tense pericardial effusion, resulting in limited cardiac inflow. This situation causes the typical findings of jugular venous distension, liver enlargement, hypotension, and pulsus paradoxus. However, clinical tamponade represents the tip of the iceberg when tamponade is understood as a severity continuum. Echocardiographic manifestations of cardiac compression and particularly hemodynamic findings of tamponade are already apparent before clinical signs develop.14,16,17 In the present study, we used a catheterization-based definition of tamponade that constitutes the most accurate diagnostic method. Experimental16 and clinical studies17 have demonstrated subtle abnormalities in cardiac filling in very early stages of pericardial effusion when intrapericardial pressure is lower than right atrial and pulmonary artery wedge pressures; however, in this study, we demanded equilibration of intrapericardial and right atrial pressures as a diagnostic criterion of cardiac tamponade, which is more accepted and more accurate.6
Low-pressure cardiac tamponade has been described as a syndrome in which a comparatively low pericardial pressure causes tamponade because of intravascular fluid depletion. Although some clinical findings may be suggestive, an adequate clinical definition is lacking. Therefore, its firm diagnosis must be based on catheterization data, as is effusive-constrictive pericarditis. However, no precise criteria emerge from the revision of the literature as the cutoff point to establish the diagnosis of low-pressure rather than classic tamponade. For instance, Spodick stated recently that “low pressure tamponade occurs at diastolic pressures of 6 to 12 mm Hg.” Shabetai commented on the concept of the syndrome but did not provide precise hemodynamic criteria. In our 143 patients with tamponade, the plot of individual values of intrapericardial pressure before pericardiocentesis and right atrial pressure after pericardiocentesis (Figure 1) shows a continuum of hemodynamic values. Our definition of low-pressure cardiac tamponade includes the patients with low filling pressure (arbitrarily defined as right atrial pressure <4 mm Hg after pericardiocentesis) and with preprocedural intrapericardial pressure of <7 mm Hg. Although the criteria we used may result in the selection of the most characteristic cases, we chose such cutoff points to ensure the specificity of our description and because of the observation (Figure 1) that most patients with right atrial pressure <4 mm Hg after pericardiocentesis had preprocedural intrapericardial pressure <7 mm Hg.

Low-pressure cardiac tamponade has been well described in experimental preparations but is mentioned only briefly in clinical reviews of cardiac tamponade. Its clinical and hemodynamic features were carefully described by Antman et al in 1979 in a presumably dehydrated and hypovolemic elderly man with tuberculous pericarditis. Jugular venous distension was not apparent, and pulsus paradoxus was only 5 mm Hg. Hemodynamic findings before pericardiocentesis included a cardiac output of 2.3 L/min and equalization of mean right atrial, intrapericardial, and pulmonary capillary wedge pressures at 8 mm Hg. After removal of 600 mL of pericardial fluid, both mean right atrial and intrapericardial pressures fell to 0 mm Hg, with marked improvement in clinical status. The authors emphasized the difficulties in recognizing low-pressure cardiac tamponade because some of

<table>
<thead>
<tr>
<th>TABLE 2. Hemodynamic Findings in the Study Groups</th>
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<tr>
<td>Group 1</td>
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<tr>
<td>Mean arterial pressure, mm Hg</td>
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<td>Before</td>
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<td>After</td>
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<td>Heart rate, bpm</td>
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<td>Before</td>
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<tr>
<td>HPP, mm Hg</td>
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<td>Before</td>
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<td>After</td>
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<tr>
<td>Cardiac index, L/min</td>
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<td>Before</td>
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<td>After</td>
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<tr>
<td>IP, mm Hg</td>
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<td>Before</td>
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<td>After</td>
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<td>RA, mm Hg</td>
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<td>RAT, mm Hg</td>
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<td>LVD, mm Hg</td>
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<td>Before</td>
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<td>After</td>
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</tbody>
</table>

HPP denotes hemodynamic pulsus paradoxus (measured in the femoral artery); IP, mean intrapericardial pressure; RA, mean right atrial pressure; RAT, right atrial transmural pressure; and LVD, left ventricular end-diastolic pressure. Probability values in the right column related to comparisons between groups 1 and 2. Statistical intragroup comparisons (before and after pericardiocentesis) are also shown.

*P<0.0001; †P<0.007; ‡P<NS.
features have been reported. These patients probably illustrating clinical, hemodynamic, and echocardiographic
dyspnea and arterial hypotension in both low-pressure tam-
(dry syndrome. Analysis of global clinical severity (Table 1) shows a
suspicion index is required for the recognition of this syn-
was found in only 2 patients. Therefore, a high clinical
degree of jugular venous distension, and pulsus paradoxus
were present in 24% of patients with low-pressure tamponade
and in 71% of patients with classic tamponade. Remarkably,
differences were in the clinical features of tamponade, which
in both patients and disappears after pericardiocentesis. Similar hemo-
dynamic compromise in both patients is apparent despite the
markedly different intrapericardial pressure level. FA indicates
femoral artery pressure; RA, right atrial pressure; and IP, intra-
pericardial pressure.

Figure 2. Findings at catheterization in a patient (patient 14)
with low-pressure tamponade (top) and in a patient with classic
tamponade (bottom). The left side of the figure corresponds to
catheterization data before pericardiocentesis; the right part,
data after pericardiocentesis. A marked pulsus paradoxus in the
femoral artery is apparent before pericardiocentesis in both
patients and disappears after pericardiocentesis. Similar hemo-
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pericardial pressure.

the most characteristic clinical findings may be absent. Since
the initial report by Antman et al., only 4 isolated cases
illustrating clinical, hemodynamic, and echocardiographic features have been reported. These patients probably
represent the most severe and paradigmatic cases. However,
because clinical series have not been reported in the literature,
the prevalence and the whole clinical spectrum of the syn-
drome are unknown. The present study is the first series of
patients with low-pressure cardiac tamponade who were
identified and followed up over a 19-year period in a single
general tertiary hospital.

We found low-pressure cardiac tamponade to be present in
9.6% of patients (29 of 279) with large pericardial effusion
and in 20% of patients (29 of 143) who fulfilled the
hemodynamic criteria of tamponade. Patients with low-
pressure tamponade showed a prevalence of underlying general illnesses and previous use of diuretic, hypotensive, or
vasodilator drugs similar to that of patients with classic tamponade (Table 1) but a lower prevalence of neoplastic and
iatrogenic pericardial effusion. The rates of collapse of the
right heart chambers and of exaggerated respiratory fluctua-
tions of mitral and tricuspid flow in the echocardiography-
Doppler study also were similar. The most remarkable
differences were in the clinical features of tamponade, which
were present in 24% of patients with low-pressure tamponade
but in 71% of patients with classic tamponade. Remarkably,
only 6 of 29 patients with low-pressure tamponade had some
degree of jugular venous distension, and pulsus paradoxus
was found in only 2 patients. Therefore, a high clinical
suspicion index is required for the recognition of this syn-
drome. Analysis of global clinical severity (Table 1) shows a
gradient of clinical severity from asymptomatic status to rest
dyspnea and arterial hypotension in both low-pressure tam-
ponade and classic tamponade patients, suggesting that the
clinical severity is, to some extent, independent of the
pressure level at which tamponade occurs.

A theoretical matter of clinical concern could be the
differentiation of mild classic tamponade from severe low-
pressure cardiac tamponade because jugular venous disten-
tion and pulsus paradoxus may be absent in both situations.
Echocardiographic findings are of limited value because the
prevalence of right-chamber collapse is similar in both groups
(Table 1). This differentiation should essentially rely on
accurate clinical evaluation. Patients with mild classic tam-
ponade have a good clinical condition, whereas patients with
severe low-pressure cardiac tamponade show evident find-
ings of hemodynamic embarrassment.

Even if their hemodynamic embarrassment was apparently
small in many patients, all patients fulfilled our hemodynamic
criteria of tamponade, ie, elevation of intrapericardial pres-
sure equilibrated with right atrial pressure with a correspond-
ing reduction in right transmural pressure and characteristic changes after pericardiocentesis (Figure 3). Furthermore,
a significant increment in cardiac output was achieved by
pericardiocentesis (2.96±1.26 to 3.22±1.11 L/min; P=0.0001). The analysis of individual changes in cardiac
index (Table 3) showed that cardiac index increased by 5% to
10% in 7 of 29 patients (24%), by 10% to 20% in 7 patients
(24%), and by >20% in 8 patients (28%). The corresponding
numbers in the group of patients with classic cardiac tam-
ponade were 14%, 16%, and 65%, suggesting more severe tamponade in the latter. Cardiac index did not increase after
pericardiocentesis in 7 patients with low-pressure tamponade
and in 8 patients with classic tamponade despite a clear
reduction in intrapericardial pressure and an increase in right transmural pressure. We have no explanation for this unex-
pected finding.

Is low-pressure cardiac tamponade a separate entity? Pre-
vious case reports on low-pressure tamponade have des-
cribed patients with severe general illnesses with presumed
dehydration and hypovolemia, assuming that these conditions
were prerequisites for the diagnosis of low-pressure tampon-
ade. Objective methods to precisely quantify hypovolemia
were not used in any of them. Since then, however, hypovo-
lemia has been assumed to underlie the mechanism of
low-pressure tamponade, which might also develop with low
filling pressures by hypothetical alternative mechanisms. In
our study, patients who fulfilled the hemodynamic criteria of
low-pressure tamponade had, by definition, low right filling
pressures, but its cause was unknown in most of them because
severe general illness or clinical findings of dehydration were
present in a minority of patients. These patients recovered
uneventfully without fluid replacement therapy. Furthermore,
most patients (20.7%) in our series were asymptomatic or
complained of only exertional dyspnea (48.3%), showing a
global clinical status similar to patients with classic tampon-
ade despite the different pressure levels at which tamponade
occurs. Thus, low-pressure tamponade should not be consid-
ered a separate entity, although its clinical recognition is more
difficult because only 24% have typical clinical findings of
tamponade. Indeed, the central clinical consideration is the
recognition of the clinical severity of the tamponade, whether low pressure or classic.

Our findings improve the knowledge of the spectrum of cardiac tamponade, showing that tamponade may occur over a wide range of intrapericardial pressures with no clear correlation with symptoms. In addition, our data can help to explain the symptomatic benefit of pericardiocentesis in some patients with pericardial effusion who do not have characteristic physical or echocardiographic signs of tamponade.18

The retrospective nature of the review of the clinical records is the main limitation of this study. However, in our hospital, special attention has been given to pericardial syndromes since 1977,19 and clinical records have been fulfilled under a specific guideline since that time, thus increasing the reliability of our data. It should also be noted that our estimation of the frequency of low-pressure cardiac tamponade in the whole range of cardiac tamponade may be influenced by our strict criteria for selecting patients for pericardiocentesis. Another limitation of our study was that the mechanism of low filling pressure could not be established because no estimation of central blood volume was attempted in routine clinical practice.

Conclusions
Low-pressure cardiac tamponade is not infrequently encountered among patients who are submitted to pericardiocentesis,
although its clinical recognition may be difficult. Therefore, in patients with pericardial effusion who manifest dyspnea or clinical signs of hemodynamic embarrassment, tamponade should be ruled out and pericardiocentesis should be considered despite the absence of typical clinical findings of tamponade.

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**Disclosures**
None.

**References**
Cardiac tamponade may be a life-threatening syndrome. Its recognition is based on well-recognized physical findings such as pulsus paradoxus and jugular vein distension. However, it has been observed that in some patients with tamponade, eg, those with hypovolemia, classic signs may be absent and thus a severe condition requiring urgent pericardial drainage may be unrecognized. Because the hallmark of this condition is low cardiac filling pressure and comparatively low intrapericardial pressure, it has been called low-pressure cardiac tamponade. Knowledge is limited to isolated case reports, so the frequency and severity profile of low-pressure cardiac tamponade has been poorly defined. The present report is based on 279 patients who underwent combined pericardiocentesis and cardiac catheterization from 1986 through 2004. Twenty-nine of these patients fulfilled conventional catheterization criteria of low-pressure tamponade; classic tamponade was present in 114. Thus, low-pressure tamponade was found in 20% of patients fulfilling hemodynamic tamponade criteria. In most of these patients, classic signs of tamponade were absent and, although their clinical status as a rule was less severe than in patients with classic tamponade, they could be hemodynamically compromised. The rates of constitutional symptoms, diuretic use, and echocardiographic findings were similar in both groups. The cause of low filling pressure was unknown in most patients. Thus, our findings suggest that low-pressure cardiac tamponade is not a rare finding. Its recognition may require a high suspicion index in hemodynamically compromised patients with suspected or established pericardial disease because classic findings of tamponade are rare in these patients.
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