Does This Patient With a Pericardial Effusion Have Cardiac Tamponade?

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**Context**  
Cardiac tamponade is a state of hemodynamic compromise resulting from cardiac compression by fluid trapped in the pericardial space. The clinical examination may assist in the decision to perform pericardiocentesis in patients with cardiac tamponade diagnosed by echocardiography.

**Objective**  
To systematically review the accuracy of the history, physical examination, and basic diagnostic tests for the diagnosis of cardiac tamponade.

**Data Sources**  
MEDLINE search of English-language articles published between 1966 and 2006, reference lists of these articles, and reference lists of relevant textbooks.

**Study Selection**  
We included articles that compared aspects of the clinical examination to a reference standard for the diagnosis of cardiac tamponade. We excluded studies with fewer than 15 patients. Of 787 studies identified by our search strategy, 8 were included in our final analysis.

**Data Extraction**  
Two authors independently reviewed articles for study results and quality. A third reviewer resolved disagreements.

**Data Synthesis**  
All studies evaluated patients with known tamponade or those referred for pericardiocentesis with known effusion. Five features occur in the majority of patients with tamponade: dyspnea (sensitivity range, 87%-89%), tachycardia (pooled sensitivity, 77%; 95% confidence interval [CI], 69%-85%), pulsus paradoxus (pooled sensitivity, 82%; 95% CI, 72%-92%), elevated jugular venous pressure (pooled sensitivity, 76%; 95% CI, 62%-90%), and cardiomegaly on chest radiograph (pooled sensitivity, 89%; 95% CI, 73%-100%). Based on 1 study, the presence of pulsus paradoxus greater than 10 mm Hg in a patient with a pericardial effusion increases the likelihood of tamponade (likelihood ratio, 3.3; 95% CI, 1.8-6.3), while a pulsus paradoxus of 10 mm Hg or less greatly lowers the likelihood (likelihood ratio, 0.03; 95% CI, 0.01-0.24).

**Conclusions**  
Among patients with cardiac tamponade, a minority will not have dyspnea, tachycardia, elevated jugular venous pressure, or cardiomegaly on chest radiograph. A pulsus paradoxus greater than 10 mm Hg among patients with a pericardial effusion helps distinguish those with cardiac tamponade from those without. Diagnostic certainty of the presence of tamponade requires additional testing.

**See also Patient Page.**  
CME available online at [www.jama.com](http://www.jama.com)

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He is found to have a pulsus paradoxus of 18 mm Hg with a blood pressure of 130/70 mm Hg and a heart rate of 90/min. The remainder of the physical examination is unremarkable. The chest radiograph shows an enlarged cardiac silhouette. You order an echocardiogram that shows a moderate-sized, circumferential pericardial effusion with a left ventricular ejection fraction of 65%. The study is limited by body habitus and rapid respirations, and the right...

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atrium and right ventricle are poorly visualized. Do the physical examination findings alter your assessment of this patient’s pericardial effusion?

**WHY IS THE CLINICAL EXAMINATION FOR CARDIAC TAMPONADE IMPORTANT?**

Cardiac tamponade occurs when fluid trapped in the pericardial space compresses the heart and compromises cardiac output. The consequences of cardiac tamponade range from barely detectable effects to overt hemodynamic collapse.1,2 When a pericardial effusion becomes large enough or accumulates rapidly enough to cause hemodynamic consequences, readily observed symptoms and signs herald impending circulatory collapse that necessitates urgent therapeutic intervention.

The invasive reference standard for the diagnosis of cardiac tamponade requires simultaneous measurement of intrapericardial and intracardiac pressures. The intrapericardial, right atrial, pulmonary artery diastolic, and pulmonary capillary wedge pressures are all elevated and equalized in cardiac tamponade (FIGURE 1). In the absence of other pathology (eg, effusive-constrictive disease), pericardiocentesis will normalize pressures and improve the cardiac output, confirming the diagnosis.4

Echocardiography is the diagnostic test used most frequently to evaluate cardiac tamponade when it is clinically suspected (FIGURE 2). While echocardiography is the noninvasive reference standard for the diagnosis of pericardial effusion,5 the mere presence of an effusion does not define cardiac tamponade. Rather, several echocardiographic findings, including right atrial systolic collapse, right ventricular diastolic collapse, inferior vena caval plethora, and exaggeration of respiratory changes in flow velocities across the tricuspid and mitral valves, are indicative of tamponade physiology and make the diagnosis with near certainty when the pretest probability is high. Because cardiac tamponade occurs on a continuum of hemodynamic effects, the findings on echocardiogram may be too sensitive and overdiagnose cardiac tamponade in patients with only subtle evidence of hemodynamic compromise.6,7 Nonetheless, because echocardiography is a noninvasive modality that is highly accurate, it is an essential component of the evaluation.8-11

There are several scenarios in which the clinical examination for cardiac tamponade could be useful. If the clinical examination could rule out tamponade with a high degree of certainty, it could preclude the need for echocardiography in some patients and allow the clinician to pursue other potential diagnoses. On the other hand, the clinician is sometimes faced with echocardiographic findings suggestive of cardiac tamponade, and clinical correlation is necessary to make the diagnosis with near certainty when the pretest probability is high.6,12

**The Pathophysiology of Cardiac Tamponade**

The pericardial sac consists of 2 layers: an outermost parietal pericardium and the inner visceral pericardium (epicardium) that reflects directly over the surface of the heart. Normally, no more than 15 to 30 mL of fluid exists between the 2 layers, held at pressures approximating the pleural pressure or approximately 5 mm Hg lower than central venous pressure.13,14

When pericardial fluid accumulates slowly, parietal pericardium compliance increases. Increasing amounts of fluid causes intrapericardial pressure to increase, while the central venous pressure responds by increasing to maintain a gradient that allows cardiac filling. When pericardial compliance can increase no more, the intrapericardial fluid pressure and cardiac filling pressures remain equalized. As intrapericardial fluid volume increases to the limit of pericardial compliance, intrapericardial pressure rises. Cardiac output begins to fall as intrapericardial pressure equals, then exceeds, central venous, right atrial, and right ventricular end-diastolic pressures (right heart filling pressures), then rapidly falls as it equalsizes with left atrial and left ventricular end-diastolic pressures (left heart filling pressures).

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pressure first equalizes with the right ventricular diastolic pressure and then with the left (Figure 1). At this threshold, the cardiac output drops and circulation is maintained only through an increase in heart rate, contractility, and peripheral arteriolar vasoconstriction. Because of pericardial compliance, large amounts of fluid may accumulate before overt hemodynamic effects occur. Rapid (ie, over minutes to hours) fluid accumulation may exceed the ability of the pericardium to stretch; if so, intrapericardial pressure increases rapidly and cardiac filling is impaired, with an often dramatic drop in cardiac output.

Causes of Cardiac Tamponade
The etiology of cardiac tamponade in patients admitted to medical services mirrors the various conditions that cause pericardial effusions. The absence of population-based incidence estimates of tamponade and the paucity of data about how often pericardial effusion leads to tamponade prevents establishment of pretest probability estimates. We do know that cardiac tamponade occurs so infrequently that the diagnosis should not be considered unless the setting is appropriate. As a result of different patient populations, practice settings, and the intensity of evaluation undertaken, the reported incidences of various etiologies of pericardial effusions vary.

In the largest reported series, Sagristà-Sauleda et al reviewed the medical records of 322 Spanish patients with moderate or large effusions. The most common diagnoses were acute idiopathic pericarditis (20%), iatrogenic effusion (16%), malignancy (13%), chronic idiopathic effusion (9%), acute myocardial infarction (8%), end-stage renal disease (6%), congestive heart failure (5%), collagen vascular disease (5%), and tuberculosis or bacterial infection (4%). Pericardial effusions often result from primary cardiac disease, occurring in 14% of patients with congestive heart failure, 21% of patients with valvular heart disease, and 15% of patients with myocardial infarction. Effusions are extremely common after cardiac surgery, although large effusions causing tamponade are rare in this setting.

Figure 2. Echocardiogram of a 62-Year-Old Woman With Advanced Lung Cancer and Malignant Pericardial Effusion Causing Cardiac Tamponade

In this subcostal view captured in early ventricular diastole, a large, circumferential pericardial effusion measuring 3.3 cm in maximal diameter is compressing the heart, and the right ventricle is completely collapsed (see video at http://jama.com/cgi/content/full/297/16/1810/DC1).
cough, weakness, fatigue, anorexia, and palpitations also occur.\textsuperscript{23} Because patients with acute cardiac tamponade are critically ill and often in shock, the clinical history may not be obtainable.

\textbf{Signs}

The physical examination begins with an assessment of vital signs and, depending on the degree of hemodynamic compromise, may reveal tachycardia, hypotension, and tachypnea. The classic findings of tamponade were described in 1935 by thoracic surgeon Claude Schaeffer Beck.\textsuperscript{24} The Beck triad is characterized by decreasing arterial blood pressure, increasing jugular venous pressure, and a small, quiet heart. This triad was seen in surgical patients with acute tamponade from intrapericardial hemorrhage due to trauma or myocardial or aortic rupture. This “surgical tamponade” is distinct from that seen in medical patients, who generally develop pericardial effusions slowly, and in whom the findings of the Beck triad may not be present at all.\textsuperscript{25} Some medical patients, especially those with preexisting hypertension, may actually be hypertensive with tamponade physiology.\textsuperscript{26} A fever associated with a pericardial effusion suggests an infectious etiology but also could be associated with immune-mediated connective tissue disease such as systemic lupus erythematosus.

\textbf{Pulsus Paradoxus}

The examiner should evaluate the patient for the presence of pulsus paradoxus, a phenomenon originally described by Adolf Kussmaul in 1873 as a palpable diminution of the radial pulse on inspiration in patients with cardiac tamponade.\textsuperscript{27} Pulsus paradoxus is not a paradox at all, but an exaggeration of the normal inspiratory decrease in blood pressure.\textsuperscript{28} This decrease occurs because negative intrathoracic pressure during inspiration cause increased venous return and filling of the right heart, which result in bowing of the septum to the left, decreasing filling of the left heart. In the normal heart, the consequence of this phenomenon, known as ventricular interdependence, is a mildly decreased stroke volume and blood pressure during inspiration. Opposite changes occur during expiration. In cardiac tamponade, ventricular interdependence is exaggerated because high pericardial pressure compresses the entire heart; during inspiration, left heart filling is more dramatically restricted by right heart filling.\textsuperscript{29}

Pulsus paradoxus can be detected by palpating the radial pulse and noting an inspiratory diminution of the pulse during normal respirations. It is most commonly measured using a manual sphygmomanometer (FIGURE 3).\textsuperscript{30,31} During normal respirations, the examiner slowly deflates the sphygmomanometer cuff while listening for the first Korotkoff sounds. These sounds are initially intermittent and respirophasic, becoming audible with expiration and inaudible with inspiration. With further cuff deflation, the Korotkoff sounds become audible throughout the respiratory cycle. The clinician records the systolic pressure at which Korotkoff sounds are first audible and the systolic pressure at which they are audible constantly through a respiratory cycle. The presence of a pulsus paradoxus may also be detected by observing the inspiratory diminution of the peripheral pulse on an arterial catheter tracing or pulse oximeter (Figure 3).\textsuperscript{30,31}

Most textbooks define a greater than 10-mm Hg difference between the initial detection of sounds on expiration and the constant presence of sounds with each heartbeat through the respiratory cycle as a “pulsus paradoxus.”\textsuperscript{32} Some experts suggest that the absolute value of pulsus paradoxus should be interpreted as a percentage of the pulse pressure or as a percentage of the expiratory systolic pressure.\textsuperscript{15,33,34} “Total paradox,” also known as “pulse obliteration,” is defined as inspiratory disappearance of the brachial and radial pulses, with total disappearance of the Korotkoff sounds.\textsuperscript{25}

Many conditions may mask the presence of pulsus paradoxus, including hypotension, pericardial adhesions, aortic regurgitation, atrial septal defects, and right ventricular hypertrophy.\textsuperscript{23} Likewise, other conditions can create a pulsus paradoxus, including severe obstructive pulmonary disease, congestive heart failure, mitral stenosis, massive pulmonary embolism, severe hypovolemic shock, obesity, and tense ascites.\textsuperscript{13,23}

\textbf{Other Signs}

Patients with cardiac tamponade often have an elevated jugular venous pressure at bedside examination, but the sensitivity of this finding may be reduced by the patient’s body habitus and, theoretically, in the setting of hypovolemia. The Kussmaul sign, an inspiratory elevation of the jugular venous pressure, should not be seen unless underlying constrictive disease exists.\textsuperscript{13,35}

On cardiac auscultation, the examiner may note diminished heart sounds, although this finding may also be observed in patients with large chests or chronic obstructive pulmonary disease. Although pericardial friction rubs are often believed to occur only in patients with pericarditis and relatively small effusions, some patients with tamponade have a pericardial rub.\textsuperscript{8,25,35} One study demonstrated that a rub may be present with very large effusions, so the presence of a rub is not a useful clue to the size of the effusion.\textsuperscript{36}

\textbf{Electrocardiogram and Chest Radiograph}

The electrocardiogram and chest radiograph support the diagnosis of a large pericardial effusion causing tamponade. Findings on an electrocardiogram indicating a large pericardial effusion include low QRS voltage, electrical alternans (changes in the amplitude or morphology of the P, QRS, and ST-T waves from one beat to the next, resulting from cardiac oscillation within the pericardial fluid\textsuperscript{17}), atrial arrhythmias, and, if pericardial inflammation is present, ST-segment elevation and PR-segment depression. Depending on the size of the effusion, the chest radiograph may be normal or may demonstrate an enlarged, globular cardiac silhouette and/or an epicardial fat stripe or “double lucency” sign on lateral views.\textsuperscript{38}
METHODS

Literature Search and Data Collection

We performed a structured MEDLINE search (January 1, 1966, to December 31, 2006) to identify English-language articles pertinent to the clinical examination of patients with cardiac tamponade. Keywords used included physical examination, medical history taking, predictive value, decision support techniques, Bayes theorem, cardiac auscultation, professional competence, sensitivity and specificity, reproducibility of results, observer variation, diagnostic tests, routine, electrocardiography, pericardium, pericardial disease, pericarditis, pericardial effusion, pericardial tamponade, tamponade, and cardiac tamponade. Two authors (C.L.R., M.A.M.) independently reviewed the abstracts of the search and retrieved potentially relevant articles. To identify additional articles, we reviewed the reference lists of articles that were retrieved and references cited in cardiology and physical diagnosis textbooks.32,39-41

We included articles reporting the results of original studies that evaluated the history, physical examination, and routine diagnostic tests such as electrocardiography or plain chest radiography compared with a reference standard for the diagnosis of cardiac tamponade.

Figure 3. Measurement and Mechanism of Pulsus Paradoxus

A. Measuring Pulsus Paradoxus

B. Ventricular Interdependence

A. The examiner inflates the sphygmomanometer cuff fully, listens for Korotkoff sounds as the cuff is slowly deflated, and then notes the pressure at which Korotkoff sounds are initially audible only during expiration. As the cuff is further deflated, the examiner notes the pressure at which Korotkoff sounds become audible during expiration and inspiration. The difference between these 2 pressures is the pulsus paradoxus. In cardiac tamponade, the pulsus paradoxus measures greater than 10 mm Hg. Inspiratory diminution in the pulse wave amplitude seen on this arterial tracing demonstrates pulsus paradoxus. A similar phenomenon may be observed on a pulse oximeter waveform. B. During inspiration in the normal heart, negative intrapleural pressures increase venous return to the right ventricle and decrease pulmonary venous return to the left ventricle by increasing pulmonary reservoir for blood. As a result of increased right ventricular distention, the interventricular septum bows slightly to the left, and the distensibility, filling, and stroke volume of the left ventricle are mildly reduced. In expiration, these changes are reciprocal, resulting in the septum bowing to the right and a mild reduction in right ventricular filling. In the presence of cardiac tamponade, the reciprocal changes seen in the normal heart are exaggerated when the pericardial sac is filled with fluid, thus limiting distensibility of the entire heart. This results in a more dramatic reduction in filling of the left ventricle during inspiration, exacerbating the normal inspiratory decrease in stroke volume and blood pressure.
tamponade. Acceptable reference standards included pericardiocentesis with right heart catheterization and echocardiography. We excluded studies dealing with pericardial diseases other than cardiac tamponade, as well as studies of patients with cardiac tamponade after cardiac surgery. We also excluded those with fewer than 15 patients; similar exclusions of small studies have been used in prior reviews in The Rational Clinical Examination series.62,63

Articles were graded for methodological quality.44 Level 1 studies independently compare a symptom or sign with an acceptable reference standard of diagnosis among a large number of consecutive patients. Level 2 studies meet the criteria for level 1 studies but have fewer patients. Level 3 studies examine nonconsecutive patients suspected of having the target disorder, but, as in level 1 and 2 studies, the patients are identified independently of the reference standard and the results of the reference standard are interpreted blinded to the signs and symptoms. Level 4 studies are nonindependent comparisons of signs and symptoms vs a gold standard among patients with, and perhaps without, the target condition. Level 4 studies may therefore include implicit retrospective reviews of small groups and case series literature.

Data Analysis

When 3 or more studies presented data on a particular finding, we calculated pooled sensitivities and confidence intervals (CIs) using a random effects model; otherwise, we report the point estimate or range. The data were analyzed using R version 2.2.0.65

RESULTS

Study Characteristics

Of the 787 studies identified using our search strategy, 8 that included a total of 300 patients met our inclusion criteria (TABLE 1). All studies were of level 4 quality. No studies assessed the precision of the clinical examination in cardiac tamponade.

Six of the 8 studies used right heart catheterization and pericardiocentesis as the reference standard for the diagnosis of cardiac tamponade. Guberman et al25 included patients with clinical findings consistent with tamponade (elevated jugular venous pressure and pulsus paradoxus) and the documentation of pericardial effusion by echocardiography or other means. Gibbs et al17 included patients diagnosed with cardiac tamponade using echocardiographic criteria. One study included control patients without pericardial effusion to evaluate the sensitivity and specificity of pulsus paradoxus in diagnosing tamponade.9,33

Accuracy of Symptoms for Cardiac Tamponade

No studies provide data for calculating the likelihood ratios (LRs) of symptoms that suggest cardiac tamponade. However, 2 studies examining 80 patients report the sensitivities of the clinical history among patients with pericardial effusion referred for pericardiocentesis (TABLE 2).6,47 Most patients with cardiac tamponade have dyspnea (sensitivity, 87%-88%). Chest pain, cough, fever, lethargy, and palpitations occur in 25% or less of patients.

Accuracy of the Physical Examination for Cardiac Tamponade

One study adequately evaluated pulsus paradoxus among patients with and without cardiac tamponade (TABLE 3), allowing calculation of LRs. Curtiss et al33 studied 65 patients with known pericardial effusion referred for pericardiocentesis for suspected tamponade and documented 13 patients with pulsus paradoxus and 52 without pulsus paradoxus. The study included control patients without cardiac tamponade (elevated jugular venous pressure, pulsus paradoxus, and documentation of pericardial effusion in 9 patients diagnosed with cardiac tamponade using echocardiographic criteria. One study included control patients without pericardial effusion to evaluate the sensitivity and specificity of pulsus paradoxus in diagnosing tamponade.9,33

Table 1. Studies Assessing the Accuracy of the Clinical Examination for Cardiac Tamponade

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of Patients</th>
<th>Study Design/ Patient Population</th>
<th>Reference Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reddy et al,66 1978</td>
<td>19</td>
<td>Retrospective chart review of patients with pericardial effusion referred for pericardiocentesis</td>
<td>Hemodynamic findings before and after pericardiocentesis</td>
</tr>
<tr>
<td>Guberman et al,25 1981</td>
<td>56</td>
<td>Retrospective chart review of patients with cardiac tamponade</td>
<td>Clinical criteria including elevated jugular venous pressure, pulsus paradoxus, and documentation of pericardial effusion</td>
</tr>
<tr>
<td>Singh et al,8 1984</td>
<td>16</td>
<td>Prospective cohort of patients with pericardial effusion referred for pericardiocentesis</td>
<td>Hemodynamic findings before and after pericardiocentesis</td>
</tr>
<tr>
<td>Curtiss et al,33 1988</td>
<td>65</td>
<td>Prospective cohort of patients with pericardial effusion referred for pericardiocentesis</td>
<td>Hemodynamic findings before and after pericardiocentesis</td>
</tr>
<tr>
<td>Levine et al,6 1991</td>
<td>50</td>
<td>Prospective cohort of patients with suspected cardiac tamponade referred for pericardiocentesis</td>
<td>Hemodynamic findings before and after pericardiocentesis</td>
</tr>
<tr>
<td>Brown et al,66 1992</td>
<td>18</td>
<td>Prospective cohort of patients with cardiac tamponade referred for pericardiocentesis</td>
<td>Hemodynamic findings before and after pericardiocentesis</td>
</tr>
<tr>
<td>Cooper et al,66 1995</td>
<td>30</td>
<td>Retrospective chart review of patients with pericardial effusion referred for pericardiocentesis</td>
<td>Relief of symptoms of dyspnea by pericardiocentesis</td>
</tr>
<tr>
<td>Gibbs et al,7 2000</td>
<td>46</td>
<td>Retrospective chart review of patients with pericardial effusion referred for pericardiocentesis</td>
<td>Echocardiographic evidence of cardiac tamponade</td>
</tr>
</tbody>
</table>

*All studies were quality level 4.
used a 20% or greater increase in cardiac output after pericardiocentesis as the reference standard for tamponade. Pulsus paradoxus was measured using simultaneous recordings of brachial or femoral artery pressure, electrocardiogram, and a thermistor output for respiratory phase identification. The LR for a pulsus paradoxus greater than 12 mm Hg was 5.9 (95% CI, 2.4-14) and for a cutpoint of greater than 10 mm Hg was 3.3 (95% CI, 1.8-6.3).[^33] When the pulsus paradoxus was 10 mm Hg or less, cardiac tamponade was unlikely (LR, 0.03; 95% CI, 0.01-0.24) and was similarly unlikely when a higher pulsus paradoxus threshold of 12 mm Hg or less was used (LR, 0.03; 95% CI, 0-0.21).[^33]

Eight studies of 295 patients reported the sensitivity of various components of the physical examination in cardiac tamponade (Table 4). All but 1 of these studies[^25] were in patients with pericardial effusion referred for pericardiocentesis. The most common findings among patients with cardiac tamponade were tachycardia, elevated jugular venous pressure, and pulsus paradoxus, with pooled sensitivities ranging from 76% to 82%. Hypotension and diminished heart sounds were insensitive (26% and 28%, respectively). Hypertension can occur in patients with tamponade but primarily occurs in patients with preexisting hypertension (sensitivity, 33%; 95% CI, 11%-55%).[^26] Guberman et al[^25] noted that tachypnea was a common finding in their study of 56 patients (sensitivity, 80%; 95% CI, 70%-90%).

### Accuracy of Electrocardiography and Chest Radiography

The electrocardiographic findings of cardiac tamponade lacked sensitivity (Table 5). The pooled sensitivity of low QRS voltage was only 42% (95% CI, 33%–51%).

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**Table 2. Sensitivity of the Clinical History in the Diagnosis of Cardiac Tamponade**

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>88</td>
<td>87</td>
</tr>
<tr>
<td>Fever</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Chest pain</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Cough</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Lethargy</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Palpitations</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 3. Sensitivity, Specificity, and Positive and Negative Likelihood Ratios (LRs) of Pulsus Paradoxus in the Diagnosis of Cardiac Tamponade[^a]**

<table>
<thead>
<tr>
<th>Pulsus Paradoxus, mm Hg†</th>
<th>&gt;12</th>
<th>&gt;10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity, %</td>
<td>98</td>
<td>98</td>
</tr>
<tr>
<td>Specificity, %</td>
<td>83</td>
<td>70</td>
</tr>
<tr>
<td>LR (95% CI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>5.9 (2.4-14)</td>
<td>3.3 (1.8-6.3)</td>
</tr>
<tr>
<td>Negative</td>
<td>0.03 (0-0.21)</td>
<td>0.03 (0.01-0.24)</td>
</tr>
</tbody>
</table>

**Table 4. Sensitivity of the Physical Examination in the Diagnosis of Cardiac Tamponade**

<table>
<thead>
<tr>
<th>Sign</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulsus paradoxus &gt;10 mm Hg</td>
<td>71±‡</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>77§</td>
</tr>
<tr>
<td>Hypotension</td>
<td>35</td>
</tr>
<tr>
<td>Hypertension[^¶]</td>
<td>80</td>
</tr>
<tr>
<td>Diminished heart sounds</td>
<td>34</td>
</tr>
<tr>
<td>Elevated JVP[^p]</td>
<td>88</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>21</td>
</tr>
<tr>
<td>Pericardial rub</td>
<td>29</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>55</td>
</tr>
<tr>
<td>Kussmaul sign</td>
<td></td>
</tr>
<tr>
<td>Pulse pressure, mm Hg</td>
<td></td>
</tr>
<tr>
<td>&gt;0</td>
<td>54</td>
</tr>
<tr>
<td>&gt;100</td>
<td>12</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; JVP, jugular venous pressure.

[^a]: All data from Curtiss et al (N = 65).[^33]
[^¶]: Measured using an intra-arterial transducer.
[^p]: Systolic blood pressure >140 mm Hg.
[^33]: Defined pulsus paradoxus as expiratory systolic pressure-Inspiratory systolic pressure/expiratory systolic pressure >10%.
[^25]: Pulsus paradoxus measured with intra-arterial transducer.
[^19]: Pulsus paradoxus measured with sphygmomanometer or intra-arterial transducer.
DOES THIS PATIENT WITH A PERICARDIAL EFFUSION HAVE CARDIAC TAMPOONADE?

Table 5. Sensitivity of the Electrocardiogram in the Diagnosis of Cardiac Tamponade

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Low voltage</td>
<td>40</td>
<td>50</td>
<td>56</td>
<td>22</td>
<td>39</td>
<td>42 (32-53)</td>
<td></td>
</tr>
<tr>
<td>Atrial arrhythmia</td>
<td>0</td>
<td>9</td>
<td>4</td>
<td>6</td>
<td>1</td>
<td>6 (1-11)</td>
<td></td>
</tr>
<tr>
<td>Electrical alternans</td>
<td>21</td>
<td>16</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td></td>
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<tr>
<td>ST-segment elevation</td>
<td>30</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>PR-segment depression</td>
<td>18</td>
<td>18</td>
<td>18</td>
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<td>18</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: CI, confidence interval.

*Not all patients had documentation of clinical findings.

32%-53%), and 2 studies reported that electrical alternans had a sensitivity of only 16% to 21%. Atrial arrhythmias are infrequent in cardiac tamponade, as are findings associated with acute pericarditis (PR-segment depression and ST-segment elevation).

Cardiomegaly on chest radiography is fairly useful in the diagnosis of cardiac tamponade, with a pooled sensitivity of 89% (95% CI, 73%-100%) in the 4 studies of 165 patients that evaluated this finding (TABLE 6).

SCENARIO RESOLUTIONS

Case 1

The patient has several important negative findings, notably an absence of a pulsus paradoxus, a jugular venous pressure that is not elevated, and no enlargement of the cardiac silhouette on chest radiograph. Knowing that these are relatively sensitive findings, you are reassured that despite her known effusion, cardiac tamponade is unlikely, and you entertain other possibilities to account for her symptoms.

Case 2

The patient has a pulsus paradoxus in the setting of a known pericardial effusion, and although the echocardiogram is nondiagnostic and the rest of his examination is normal, this clinical finding alone increases the likelihood of tamponade physiology. You arrange for urgent pericardiocentesis, which demonstrates elevated intrapericardial pressures and equalization with intracardiac pressures. Drainage of the effusion leads to resolution of the elevated pulsus paradoxus and improvement in symptoms.

CONCLUSIONS

Based on our review of the literature, dyspnea, tachycardia, elevated jugular venous pressure, pulsus paradoxus, or cardiomegaly on chest radiograph is seen in 70% or more of patients with a known pericardial effusion and cardiac tamponade. However, 50% or less of patients will have diminished heart sounds, hypotension, or low voltage on electrocardiogram. Based on 1 study, the presence of a pulsus paradoxus greater than 10 mm Hg increases the likelihood of cardiac tamponade, while a pulsus paradoxus of 10 mm Hg or less decreases the likelihood. The presence and degree of pulsus paradoxus may be helpful to predict the degree of hemodynamic compromise in tamponade in patients with an effusion who are suspected of having this condition. In general there is a paucity of evidence supporting the diagnostic accuracy of the clinical examination for cardiac tamponade, and it must be stressed that all studies enrolled patients with a previously established pericardial effusion. We found no studies that evaluated interrater reliability of clinical findings or that included patients not already known or highly suspected to have cardiac tamponade. As a result, we are unable to comment on whether the clinical examination is useful in other scenarios (eg, to exclude tamponade in patients who have not yet had an echocardiogram). In addition, the studies reviewed all took place in academic medical centers, raising concerns of referral bias and questions about generalizability to the community setting. The available evidence is also limited in that no studies have analyzed the predictive value of a combination of multiple findings. Finally, cardiac tamponade is a continuum of hemodynamic effects and the patients included in published studies are likely heterogeneous as to their position on that continuum, so the sensitivities and specificities of findings on clinical examination will vary depending on the severity of patients studied.

Two reviewed studies explicitly used invasive monitoring (ie, intra-arterial transducers), rather than a sphygmomanometer, to measure pulsus paradoxus.33,34 While one would expect that invasive measurement would be more accurate than manual measurement, the literature lacks studies directly comparing the accuracy of these methods. Nonetheless, we
found the sensitivity of pulsus paradoxus was similar in studies using different measurement techniques. In summary, when faced with a patient with a known pericardial effusion, the clinical examination may help guide decisions about the appropriate-ness of expectant management or more urgent, invasive intervention.

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