Cardiac Tamponade
A Clinical or an Echocardiographic Diagnosis?
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At times, a patient with pericardial effusion demonstrates echocardiographic evidence of cardiac tamponade without any of the usual clinical signs of tachycardia, elevated venous pressure, falling blood pressure, or pulsus paradoxus. When this happens, the clinician may be confronted with a therapeutic dilemma as to whether or not to proceed with pericardial drainage.

Since a diagnosis of cardiac tamponade usually implies a need for pericardial drainage, there should be agreement on its definition. The term "cardiac tamponade" means that there is compression of the heart by fluid within the pericardial sac that impairs diastolic filling of the ventricles.1

Physiological Features

When liquid is injected into the pericardial sac of an experimental animal, intrapericardial and right and left atrial pressures begin to rise equally2 with addition of as little as 20–40 mL of fluid in dogs weighing approximately 20 kg.3 As more fluid is added, cardiac stroke volume falls. Cardiac output also falls, but less because of compensating tachycardia.2 When cardiac output falls, systemic blood pressure is at first maintained by increased systemic arterial resistance.4 Finally, when more fluid is added, one reaches the steep part of the pericardial pressure–volume curve, where a small volume increase results in a large increase in intrapericardial pressure. Blood pressure decreases, usually at intrapericardial volumes of 6–12 mL/kg in the 22–34-kg dog.5 Spodick et al.6 by study of left ventricular ejection times, found increased inspiratory fall in left ventricular stroke output in patients with large pericardial effusions but none of the clinical features of tamponade. Echocardiographic evidence of right atrial compression indicates that intrapericardial pressure exceeds right atrial pressure. This sign may be found in some patients who have no clinical evidence of cardiac tamponade.7 Thus, the hemodynamic effects of cardiac compression by fluid in the pericardial sac represent a continuum in the early phases of which even minor elevations of intrapericardial pressure produce some effects on ventricular filling. This raises the question as to when tamponade should be diagnosed in patients. Is it when intrapericar-

dial pressure first rises and the right atrium is first compressed, is it at the point when systemic blood pressure begins to fall, or is it at some intermediate point?

Clinical Features

Early clinical definitions of cardiac tamponade dealt largely with intrapericardial bleeding from cardiac trauma or aortic or cardiac rupture. These patients had falling blood pressure, elevated systemic venous pressure, and a small, quiet heart: hence, the well-known Beck’s triad of acute cardiac compression.8 Later, it was realized that medical patients with more gradual onset of tamponade might have large hearts with preserved blood pressure and heart sounds.9 Hence, our group proposed a clinical definition of tamponade consisting of moderate or large pericardial effusion, elevated systemic venous pressure, pulsus paradoxus, dyspnea and tachycardia and relief of venous hypertension, pulsus paradoxus and dyspnea, and tachycardia by pericardial fluid drainage.9 All except one of our patients with cardiac tamponade had pulsus paradoxus, and that one had left ventricular dysfunction, which may obviate pulsus paradoxus when left ventricular end-diastolic pressure exceeds intrapericardial pressure.10

Echocardiographic Features

Echocardiographic features of cardiac tamponade include abnormal inspiratory increase of right ventricular dimensions and abnormal inspiratory decrease of left ventricular dimensions,11,12 right atrial compression,13 right ventricular diastolic collapse,14,15 abnormal inspiratory increase of blood flow velocity through the tricuspid valve and abnormal inspiratory decrease of mitral valve flow velocity,16 dilated inferior cava with lack of inspiratory collapse,17 and swinging heart18 (Table 1). Regional tamponade may be associated with left atrial compression and left ventricular diastolic collapse in the absence of right atrial or right ventricular diastolic collapse.19

Exclusive use of either clinical or echocardiographic criteria poses a diagnostic problem in some patients. Right atrial and right ventricular diastolic collapse are perhaps the most commonly used echocardiographic evidence of cardiac tamponade. Changes in blood volume may affect the sensitivity and specificity of right heart collapse as a sign of tamponade. Klopfenstein and associates20 found that the onset of right ventricular diastolic collapse is associated with significant decrease in cardiac output but not in blood pressure in euvoiemic dogs. However, with hypovolemia, as might occur in

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patients with dehydration or acute blood loss, these hemodynamic variables were unchanged at the onset of right ventricular diastolic collapse.

A study was made of experimental cardiac tamponade in conscious dogs. The systemic blood pressure response to increasing pericardial pressure depended in part on the length of the recovery period after the catheter implantation procedure. Among other factors, the patient’s blood pressure response to tamponade depends on the rate of fluid accumulation, the state of the pericardium and the myocardium, and the presence or absence of preexisting hypertension.

In experimental animals with hypovolemia, right ventricular diastolic collapse occurred at lower intrapericardial pressures than during euvoeemia or hypervolemia. Singh et al found that right ventricular diastolic collapse was associated with significant decrease in cardiac output and blood pressure in patients with hemodynamic evidence of tamponade. However, with further pericardial drainage, hemodynamic improvement occurred even after right ventricular diastolic collapse was no longer present. In patients with moderate or large pericardial effusion, right atrial collapse may be found with none of the clinical features of cardiac tamponade. Gillam et al reported right atrial collapse in 19 of 19 patients with tamponade but also in 19 of 104 without tamponade. Hemodynamic compromise is more likely when right atrial inversion is prolonged. With hypovolemia, this sign of tamponade may precede hemodynamic alterations.

Echocardiographic Doppler criteria of tamponade may be too sensitive on the one hand and lack specificity on the other. The problem with right atrial and right ventricular diastolic collapse has already been mentioned. A dilated, noncollapsing inferior vena cava is often seen with congestive heart failure and with constrictive pericarditis as well as tamponade. An abnormal respiratory pattern of mitral and tricuspid flow may be seen in obstructive airway disease, pulmonary embolism, and constrictive pericarditis or in right ventricular infarction as well as in cardiac tamponade. Increased respiratory variation in ventricular dimensions is an inconstant finding. The echocardiographic sign of swinging heart is not highly sensitive and is of unknown specificity.

Reliance on the echocardiographic signs of right atrial collapse and right ventricular diastolic collapse may be misleading in patients with suspected cardiac tamponade. False positives or false negatives may occur. In an experimental study, Vaska and associates reported right ventricular diastolic collapse caused by increased intrapericardial pressure from large intrapleural effusion in the presence of trivial pericardial effusion. Another example of a false-positive echocardiographic sign of tamponade may be found when cardiac size is abruptly increased in the presence of a trivial pericardial effusion. False-negative signs, i.e., absence of right atrial or right ventricular diastolic collapse, may also occur. This evidence of tamponade may be absent with acute or chronic elevation of right heart pressures or with decreased right atrial or right ventricular compliance caused by hypertrophy of these chambers.

### Atypical Clinical Presentation

Reliance solely on the clinical signs of tamponade may also be a problem in several settings (Table 2). Pulsus paradoxus may be absent with tamponade caused by regional right atrial compression. When regional tamponade compresses the left heart, which is most commonly seen after cardiac surgical procedures, there may be severe hemodynamic compromise in some instances despite normal right atrial and peripheral venous pressure. Patients with left ventricular dysfunction and cardiac tamponade may lack pulsus paradoxus. Pulmonary paradoxus may not be present with cardiac tamponade in the presence of positive-pressure breathing, atrial septal defect, or aortic regurgitation. Pulmonary paradoxus may be absent with tamponade associated with pulmonary arterial obstruction. Patients with obstructive airway disease may have elevated venous pressure caused by cor pulmonale and pulsus paradoxus caused by obstructive airway disease in the absence of tamponade.

### Indications for Pericardial Drainage

Agreement on a definition of tamponade is desirable from the standpoint of better communication, more exact prognosis, and improved treatment of patients with pericardial effusion. One does not wish to diagnose cardiac tamponade with the implication of pericardial drainage in every patient with a large effusion nor necessarily proceed to pericardial drainage in every patient with right atrial collapse if there is no hemodynamic compromise. The decision for pericardial drainage will be strongly influenced by the etiology of the effusion as well as the clinical status of the patient; when hemodynamic compromise is severe with dyspnea or falling blood pressure, pericardial drainage will be needed regardless of etiology. On the other hand, when the etiology is uncertain, pericardial drainage may be indicated for diagnostic purposes when echocardiographic and clinical signs of tamponade are minimal or absent. However, in this study, the diagnostic yield was only 6% of 32 patients not suspected of tamponade but was 29% in 44 patients suspected of tamponade.
It seems clear that some patients with cardiac tamponade will require pericardial drainage despite absence of some of the clinical features, e.g., those with left ventricular dysfunction, positive-pressure breathing, or localized tamponade of the left heart. On the other hand, patients with right heart compression on the echocardiogram and no clinical signs of hemodynamic compromise may not require pericardial drainage when the etiology is clear and susceptible to medical management. Levine and associates33 described 50 patients with pericardial effusion and right heart collapse, all of whom had elevated intrapericardial pressure, but 94% had systolic blood pressure >100 mm Hg and 58% had a cardiac index >2.3 L/min/m². Pericardiocentesis lowered intrapericardial pressure and improved cardiac output, but in 28 of these patients with preserved systemic blood pressure and cardiac output, pericardiocentesis did not improve blood pressure or always relieve tachycardia or dyspnea. The authors commented that the appropriate timing of pericardiocentesis in patients with two-dimensional echocardiographic signs of tamponade and variable hemodynamic compromise is not yet known.33

What are the clinical circumstances that require the clinician to consider the possibility of cardiac tamponade? These include unexplained elevation of systemic venous pressure, unexplained low or falling blood pressure, pulsatd paradoxus, unexplained tachycardia, and unexplained dyspnea or tachypnea. Tamponade should also be considered in patients with evidence of pericarditis, i.e., pericardial rub, ECG changes of acute pericarditis, unexplained cardiac enlargement on chest radiogram, or echocardiographic evidence of pericardial effusion.

When a patient has one or more of the above findings, the possibility of tamponade is strengthened when certain predisposing conditions are present. These include patients who have had recent cardiac surgery, especially if receiving anticoagulants; patients who have had chest trauma; those after cardiac infarction, especially if receiving anticoagulant or thrombolytic therapy; patients with sepsisemia or other evidence of infectious disease; and patients with malignant neoplasm, renal failure, or connective tissue disease. Patients with hyperalimentation by means of a catheter tip in the right atrium are also at risk.

When systemic venous pressure can be reliably estimated at the bedside or is found to be normal by central venous pressure line, cardiac tamponade can usually be eliminated, although low-pressure tamponade may occur, and, rarely, regional left heart tamponade with normal central venous pressure must be considered, especially after cardiac surgery. Clinical evidence of cardiac enlargement, murmurs of significant valvular disease, rales of pulmonary congestion, and/or presence of ventricular gallop rhythm as a rule mean that elevated venous pressure is due to ventricular dysfunction and not tamponade. Asthmatic wheezes suggest that pulsatd paradoxus is due to obstructive airway disease.

When the symptoms and signs mentioned above are present and unexplained, a two-dimensional echocardiogram should be obtained. As a rule, cardiac tamponade can be eliminated as a possibility unless the echocardiogram demonstrates a moderate or large pericardial effusion, but localized effusions can produce tamponade, especially after cardiac surgery, chest trauma, or irradiation therapy. As a rule, tamponade will be associated with echocardiographic evidence of right atrial collapse, and the absence of this sign ordinarily excludes tamponade. Exceptions are cited earlier in this article. When there is doubt, hemodynamic investigation or pericardial drainage may be necessary, especially when there is echocardiographic evidence of ventricular enlargement or dysfunction in the presence of sizeable pericardial effusion.

**Summary**

In most patients, cardiac tamponade should be diagnosed by a clinical examination that shows elevated systemic venous pressure, tachycardia, dyspnea, and paradoxical arterial pulse. Systemic blood pressure may be normal, decreased, or even elevated.34 The diagnosis is confirmed by echocardiographic demonstration of moderately large or large circumferential pericardial effusion and in most instances, of right atrial compression, abnormal respiratory variation in right and left ventricular dimensions, and in tricuspid and mitral valve flow velocities. Pulsus paradoxus may be absent with left ventricular dysfunction, atrial septal defect, regional tamponade, and positive-pressure breathing. Systemic venous pressure may be normal with localized tamponade of the left atrium or ventricle.

Patients with moderately large or large pericardial effusions may have echocardiographic evidence of right atrial compression without clinical signs of elevated venous pressure or pulsus paradoxus. The majority of these patients have mild or moderate tamponade and if not subjected to pericardial drainage, should be observed closely. In some of these patients, when the etiology is known and the disease can be treated effectively with medication, e.g., nonsteroidal anti-inflammatory agents or adrenal corticosteroids in Dressler's syndrome or relapsing pericarditis, pericardial drainage may not be necessary.

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