Cardiac Tamponade: Diagnosis and Treatment

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Table of Contents

Introduction ............................................. 2
Patient Presentation .................................... 2
Physiology and Pathology ............................... 2
Approach to Diagnosis ................................. 3
Treatment ................................................ 7
Summary Points ........................................ 9
References ............................................. 9

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Cardiac Tamponade: Diagnosis and Treatment

Fadi M.F. Alameddine, MD, and Andro G. Kacharava, MD, PhD

INTRODUCTION

Cardiac tamponade is a compression of the heart caused by an excessive accumulation of contents in the pericardium. These contents can include fluid, blood, pus, or gas resulting from inflammation, trauma, or rupture of the heart. The 3 principal features of cardiac tamponade are elevation of intracardiac pressures, limitation of ventricular filling, and reduction of cardiac output. Severe cardiac tamponade can lead to hemodynamic deterioration and shock. The quantity of fluid necessary to produce this critical condition may be as small as 150 mL if the effusion develops rapidly, or more than 2000 mL if the effusion develops slowly and the pericardium has time to stretch and adjust to the increasing volume.1

Multiple medical conditions can lead to cardiac tamponade. The 4 most common causes of accumulation of fluid in the pericardium in an amount sufficient to cause serious obstruction to the cardiac inflow of blood are neoplastic and infectious diseases, idiopathic pericarditis, and uremia. Cardiac surgery and percutaneous coronary interventions are frequent iatrogenic causes of cardiac tamponade.

PATIENT PRESENTATION

A 57-year-old man with a 3-hour history of worsening shortness of breath is brought by ambulance to the emergency department. The patient reports a 1-month history of chest discomfort and mild shortness of breath that has become progressively worse within the past several days. His last general medical examination was performed 3 months earlier. At that time, he was in good health, and his primary care physician reemphasized the need for him to comply with his diabetes, hypertension, and hyperlipidemia medications.

On initial physical examination in the emergency department, the patient is in respiratory distress, tachycardic, and hypotensive with peripheral cyanosis. Cardiovascular examination is remarkable for attenuated heart sounds and extreme elevation of venous pressures. Breathing sounds are normal. An electrocardiogram (ECG) shows decreased amplitude of QRS complexes but otherwise is unremarkable. Echocardiography is performed and confirms the diagnosis of cardiac tamponade. Emergent needle pericardiocentesis is performed, and 1.5 L of serosanguinous fluid are drained. Immediately following pericardiocentesis, the blood pressure and heart rate normalize.

A catheter is left in the pericardial space, and over the next 48 hours an insignificant amount of fluid (< 80 mL) is drained. The catheter is removed. Further workup to determine the cause of the pericardial effusion does not identify a specific cause. The patient is treated with nonsteroidal anti-inflammatory medications and is discharged from the hospital. Predischarge echocardiogram does not reveal further accumulation of pericardial fluid.

PHYSIOLOGY AND PATHOLOGY

The pericardium is an elastic, closed fibroserous sac that envelops the entire heart and much of the ascending aorta, the main pulmonary artery, all pulmonary veins, and portions of the inferior and superior vena cavae. It consists of an inner serous layer and an external layer composed of fibrous tissue. The pericardial space between the 2 layers contains a small amount of ultrafiltrate of plasma that acts as a lubricant and normally drains through the right lymphatic duct via the right pleural space and through the thoracic duct via the parietal pericardium.23

The pericardium is relatively inextensible and has a limited ability to expand acutely in the face of rapidly increasing pericardial contents. The exponential volume–pressure relationship for the pericardium is shown in Figure 1. The flat portion of the J-shaped volume–pressure curve represents intrapericardial...
pressure when the pericardial content and cardiac volumes are at normal, physiologic levels (ie, pericardial reserve volume). According to the concept of the pericardial continuum, steady increases in pericardial content cause pericardial pressure to increase once the relatively small pericardial volume reserve is filled. Increases in pericardial content that exceed the rate at which the pericardium stretches will place the pericardial pressure on the steep portion of the volume-pressure curve and cause a parallel increase in intracardiac pressure. In this situation, the cardiac chambers must compete with pericardial content for relatively finite intrapericardial volume to avoid progressive cardiac compression and eventual hemodynamic failure.

During the first phase of cardiac tamponade, intrapericardial and ventricular diastolic pressures rise but do not equilibrate. In phase 2, the more rapidly rising intrapericardial pressure equilibrates with right ventricular diastolic pressure. During this phase, the parallel rise of the intrapericardial and right ventricular diastolic pressures continue until they equilibrate with left ventricular diastolic pressure (phase 3). Cardiac chamber collapse occurs at the moment when the myocardial transmural pressure (the intracardiac pressure minus the pericardial pressure) becomes transiently negative. Collapse of the thinner right atrium tends to occur earlier than collapse of the right ventricle.

Like most tamponade-induced abnormalities of pressure and flow, transmural pressures are reciprocally reduced and increased during the respiratory phases (variation in intrathoracic pressure) due to the effect of respiration on systemic and pulmonary venous pressures. These respiratory pressure changes and the phenomenon of ventricular interdependence are mechanisms underlying the clinical finding of pulsus paradoxus, defined as an abnormally large (> 10 mm Hg) decrease in systolic blood pressure on inspiration. The origin of pulsus paradoxus is complex and multifactorial. Because both ventricles share a tight pericardial sac, the inspiratory enlargement of the right ventricle in cardiac tamponade compresses the left ventricle and reduces left ventricular volume. Leftward bulging of the interatrial and interventricular septa further increases the volume of the right side of the heart at the expense of the left side, decreasing left ventricular inflow. Thus, in cardiac tamponade the normal inspiratory augmentation of right ventricular volume causes an exaggerated reciprocal reduction in left ventricular volume. In addition, the negative thoracic pressure produced by inspiration is transmitted to the aorta, increasing left ventricular afterload and reducing stroke volume.

**APPRAOCH TO DIAGNOSIS**

**CLINICAL AND PHYSICAL FINDINGS**

Cardiac tamponade should be viewed hemodynamically as a continuum ranging from mild to severe. Mild cardiac tamponade is frequently asymptomatic, whereas moderate and severe tamponade produce precordial discomfort, dizziness, and dyspnea at rest. Patients frequently complain of palpitations and dyspnea on exertion that progresses to shortness of breath at rest. They also may complain of general weakness, poor appetite, abdominal distention, atypical chest pain, and cough. When cardiac tamponade complicates a diagnostic procedure, common symptoms include vague discomfort, generalized uneasiness, and precordial pain. Fluoroscopy may show an enlarged cardiac silhouette and diminished pulsations.

Similar to the clinical findings, most physical findings in cardiac tamponade are nonspecific. Tachycardia and hypotension (which can be profound) are seen in most patients with clinically significant cardiac tamponade. The triad of hypotension, quiet heart sounds, and increased jugular venous pressure described by Beck in 1935 is present in only 30% of patients with acute cardiac tamponade. Careful inspection of the jugular venous pulse waveform is essential for the
In cardiac tamponade, compression of the heart by pericardial fluid results in a characteristic loss of the y descent, but the x descent is preserved. However, the venous pressure may be normal in early tamponade, and extreme elevations of venous pressure may go unrecognized in a recumbent or semirecumbent patient. Peripheral venous distention also is noted in the forehead, scalp, and ocular fundi unless the patient is hypovolemic. A pericardial rub is a frequent finding in patients with inflammatory effusions. Heart sounds may be attenuated due to the muffling effects of the pericardial fluid and reduced cardiac function. An apical beat is occasionally palpable, and patients with cardiomegaly or apical pericardial adhesions may have active pulsations.

An inspiratory decline of systolic arterial pressure exceeding 10 mm Hg (ie, pulsus paradoxus) may be quantified with sphygmomanometry as the patient breathes normally through a respiratory cycle. The size of the pulsus is the difference between the systolic pressure at which Korotkoff sounds are heard only during expiration and the systolic pressure at which sounds are heard through the respiratory cycle. Although pulsus paradoxus is neither sensitive nor specific for cardiac tamponade, in the appropriate clinical setting it is a key finding that signifies cardiac tamponade. Pulsus paradoxus also may be observed in hypovolemic shock, acute and chronic obstructive airways disease, and pulmonary embolus. In certain conditions, cardiac tamponade can occur without the presence of pulsus paradoxus (Table 1).

Hemodynamic changes in the postsurgical period may delay the appearance of clinical signs of cardiac tamponade such as jugular venous distention, quiet heart sounds, and pulsus paradoxus. In these patients, the appropriate imaging modality is needed for a correct and quick diagnosis. A high index of suspicion for cardiac tamponade is required because right ventricular myocardial infarction and constrictive pericarditis can resemble cardiac tamponade. The differences between these conditions are shown in Table 2.

### Table 1. Conditions Leading to the Absence of Diagnostic Pulsus Paradoxus in Cardiac Tamponade

<table>
<thead>
<tr>
<th>Condition</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marked LV or RV dysfunction</td>
<td>Decrease myocardial compliance or hypertrophy; heart failure</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>Can produce sufficient regurgitant flow to the left ventricle to damp respiratory fluctuations</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>Sufficiently large defects cause increased venous return to the right atrium to be balanced by right-to-left shunting of blood</td>
</tr>
<tr>
<td>Extreme hypotension</td>
<td>Respiratory pressure changes become unmeasurable, especially by cuff</td>
</tr>
<tr>
<td>Local cardiac adhesions and compression (eg, with loculated fluid)</td>
<td>May be related to LV stiffness for uncertain reasons</td>
</tr>
<tr>
<td>Acute LV myocardial infarction</td>
<td></td>
</tr>
</tbody>
</table>

LV = left ventricular; RV = right ventricular.


### Diagnostic Modalities

#### Electrocardiogram

There are no specific ECG signs in cardiac tamponade. The ECG may show tachycardia, atrial arrhythmias, low-voltage, P-R segment and ST-T changes of pericarditis, and electrical alternation, which may affect any electrocardiographic waves. Occurring in only approximately 20% of cardiac tamponade cases, electrical alternans is an insensitive sign, unless complete alternans is present.

#### Chest Radiograph

Similarly, there are no specific signs of cardiac tamponade on chest radiograph. A large cardiac silhouette in a patient with clear lung fields may suggest the presence of a pericardial effusion. Chest radiographs may not be helpful early in the course of acute cardiac tamponade because at least 200 mL of fluid must accumulate before the cardiac silhouette is affected.

#### Computed Tomography/Magnetic Resonance Imaging

Computed tomography scanning can easily detect as little as 50 mL of pericardial effusion, reflecting better resolution of boundaries between cardiac, pleural, pericardial, and mediastinal structures. Magnetic resonance imaging is also a very sensitive tool in the diagnosis of pericardial effusion. This modality has the same advantages of computed tomography as well as the potential...
to characterize the nature of the pericardial content in certain cases. In a study comparing magnetic resonance imaging and 2-dimensional echocardiography for diagnosis of pericardial effusion, Mulvagh and colleagues found that MRI’s ability to easily visualize epicardial borders and pericardial fat pad enabled detection of small pericardial effusions that were not detected by echocardiography.19 Despite these advantages, both computed tomography and magnetic resonance imaging are often less readily available and more costly and inconvenient in an acutely ill patient. These imaging modalities usually are not required unless echocardiography is not feasible.

### Echocardiography

Echocardiography plays a significant role in the diagnosis of pericardial effusion and tamponade. It is a non-invasive test that can be performed quickly and frequently in a critically ill patient. Echocardiography can detect pericardial effusion by identifying a fluid collection around the heart. The presence of pericardial effusion can be confirmed by the change in the shape of the heart chambers and the presence of a pericardial knock. The key characteristics of echocardiography include:

- **Clinical**: Pulsus paradoxus, Jugular veins, Prominent y descent, Prominent x descent, Kussmaul’s sign, S₃, Pericardial knock, Low ECG voltage, Electrical alternans.
- **Electrocardiogram**: Low ECG voltage, Electrical alternans.
- **Echocardiography**: Thickened pericardium, Pericardial effusion, RV size, Myocardial thickness, Right atrial collapse, Increased early filling, Increased MFV, Exaggerated respiratory variation in flow velocity.
- **CT/MRI**: Thickened/calcific pericardium, Cardiac catheterization, Cardiac biopsy.

### Table 2. Features That Distinguish Cardiac Tamponade from Constrictive Pericarditis and Similar Clinical Disorders

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Tamponade</th>
<th>Constrictive Pericarditis</th>
<th>Restrictive Cardiomyopathy</th>
<th>RVMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>Common</td>
<td>Usually absent</td>
<td>Rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Jugular veins</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prominent y descent</td>
<td>Absent</td>
<td>Usually present</td>
<td>Rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Prominent x descent</td>
<td>Present</td>
<td>Usually present</td>
<td>Present</td>
<td>Rare</td>
</tr>
<tr>
<td>Kussmaul’s sign</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>S₃</td>
<td>Absent</td>
<td>Absent</td>
<td>Rare</td>
<td>May be present</td>
</tr>
<tr>
<td>Pericardial knock</td>
<td>Absent</td>
<td>Often present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low ECG voltage</td>
<td>May be present</td>
<td>May be present</td>
<td>May be present</td>
<td>Absent</td>
</tr>
<tr>
<td>Electrical alternans</td>
<td>May be present</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Echocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickened pericardium</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Pericardial calcification</td>
<td>Absent</td>
<td>Often present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>RV size</td>
<td>Usually small</td>
<td>Usually normal</td>
<td>Usually normal</td>
<td>Enlarged</td>
</tr>
<tr>
<td>Myocardial thickness</td>
<td>Normal</td>
<td>Normal</td>
<td>Usually increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Right atrial collapse and RVDC</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Increased early filling, increased MFV</td>
<td>Absent</td>
<td>Present</td>
<td>Present</td>
<td>May be present</td>
</tr>
<tr>
<td>Exaggerated respiratory variation in flow velocity</td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>CT/MRI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickened/calcific pericardium</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Cardiac catheterization</td>
<td>Usually present</td>
<td>Usually present</td>
<td>Usually absent</td>
<td>Absent or present</td>
</tr>
<tr>
<td>Equalization of diastolic procedures</td>
<td>Usually present</td>
<td>Usually present</td>
<td>Usually absent</td>
<td>Absent or present</td>
</tr>
<tr>
<td>Cardiac biopsy helpful?</td>
<td>No</td>
<td>No</td>
<td>Sometimes</td>
<td>No</td>
</tr>
</tbody>
</table>

CT = cardiac tamponade; ECG = electrocardiogram; MFV = mitral flow velocity; MRI = magnetic resonance imaging; RV = right ventricle; RVDC = right ventricular diastolic collapse; RVMI = right ventricular myocardial infarction.

Cardiac Tamponade: Diagnosis and Treatment

**Figure 2.** Echocardiographic images of cardiac tamponade. (A) Subcostal long-axis view demonstrating a large effusion (arrows) and severe right ventricular compression. (B) Apical 4-chamber view demonstrating right atrial compression (arrow). LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle. (Adapted with permission from Alexander RW, Schlant RC, Fuster V, eds. Hurst’s the heart. 9th ed. New York: McGraw-Hill; 1998:2184.)

There are multiple M-mode and 2-dimensional signs of cardiac tamponade (Figure 2). There are multiple M-mode and 2-dimensional signs of cardiac tamponade (Table 3), but the 2 most suggestive of the presence of hemodynamic compromise are late diastolic collapse of the right atrium and an early diastolic right ventricular compression. Right ventricular collapse is a less sensitive but more specific finding. Transesophageal echocardiography can assist with the diagnosis of posterior loculated effusions, which occasionally produce left atrial and left ventricular diastolic collapse.

Pulsed wave Doppler echocardiography has been used to measure exaggerated respiratory variation of transvalvular blood flow through the left and right heart valves in patients with cardiac tamponade. An inspiratory decrease in peak mitral inflow of more than 30% (normal, < 10%) and inspiratory increase of peak tricuspid inflow velocity of more than 50% (normal, < 25%) had a good correlation with clinically significant cardiac tamponade plus a greater sensitivity than right ventricular early diastolic collapse and greater specificity than right atrial late diastolic collapse.

**Cardiac Catheterization**

Cardiac catheterization can be used to confirm the diagnosis of cardiac tamponade. Catheterization shows equilibration of average diastolic pressure and characteristic respiratory reciprocation of cardiac pressures: an inspiratory increase in right-sided pressures and a concomitant decrease in left-sided pressures, the proximate cause of pulsus paradoxus. On hemodynamic tracings during cardiac catheterization in patients with cardiac tamponade, the right atrial, pulmonary capillary wedge, pulmonary artery diastolic, and pericardial pressures are elevated, usually between 15 and 30 mm Hg, and are equal within 5 mm Hg. The right atrial and wedge pressure tracings reveal an attenuated or absent y descent (Figure 3) secondary to impaired late diastolic filling of the ventricles. Neither Kussmaul sign (ie, absence of decline in jugular venous pressure with inspiration) nor the early ventricular diastolic dip and plateau (ie, the square root sign) characteristic of pericardial constriction are seen in cardiac tamponade. Cardiac output is reduced, despite the presence of compensatory tachycardia, and systemic vascular resistance is usually elevated.

Vasoconstriction can be a prominent part of the hemodynamic response in some patients due to severe activation of the adrenergic system. In these patients, blood pressure may be normal or even elevated despite hemodynamics consistent with tamponade. In the rare cases of low-pressure cardiac tamponade, which can be observed in the presence of significant volume depletion, all the hemodynamic signs of cardiac

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**Table 3. Echocardiographic Features of Cardiac Tamponade**

<table>
<thead>
<tr>
<th>Feature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swinging heart</td>
</tr>
<tr>
<td>Abnormal respiratory changes in ventricular sizes</td>
</tr>
<tr>
<td>Dilated inferior vena cava with absence of inspiratory collapse</td>
</tr>
<tr>
<td>Late diastolic right or left atrial collapse</td>
</tr>
<tr>
<td>Early diastolic right or left ventricular collapse</td>
</tr>
<tr>
<td>Paradoxical septal motion</td>
</tr>
</tbody>
</table>

---
tamponade may be seen with intrapericardial pressures below 15 mm Hg.\textsuperscript{4,24}

**TREATMENT**

Before making decisions on the selection and timing of therapy in patients with cardiac tamponade, the physician must assess the clinical status of the patient and consider the source of the pericardial content. One may consider close observation and medical therapy if hemodynamics are not significantly compromised and the etiology of pericardial content is responsive to medical therapy. However, once hemodynamic deterioration is obvious, the physician must determine how to quickly and safely relieve intrapericardial pressure with the equipment that is available and prevent subsequent hemodynamic collapse.

**MEDICAL TREATMENT**

Inotropic support, vasodilators, and volume infusion have a limited role in the medical treatment of cardiac tamponade, especially in normovolemic and hypervolemic patients, because volume infusion can decrease transmural pressures, resulting in worsened hemodynamics.\textsuperscript{25} On the other hand, blood volume expansion to maintain the venoatrial gradients may be effective in patients suffering from low-pressure cardiac tamponade.

**NEEDLE PERICARDIOCENTESIS**

Percutaneous needle pericardiocentesis under echocardiographic guidance is the preferred therapy for cardiac tamponade that is causing hemodynamic compromise (Figure 4).\textsuperscript{24,26} The first needle pericardiocentesis was performed in 1840 by Franz Shuh, a physician from Vienna, and the method has undergone multiple modifications since then. It is best to perform the procedure in the cardiac catheterization laboratory under echocardiographic guidance so that cardiac hemodynamics can be monitored. Real-time cardiac ultrasound imaging allows one to track the needle tip and assure correct location by following the injection of normal saline into the pericardial space. It also allows the physician to assess the cardiac response to drainage and measure the volume of the residual pericardial content after drainage. When the cause of accumulated pericardial
content is uncertain, fluid obtained at pericardiocentesis should be sent for diagnostic studies. A study by Ziskind et al showed that percutaneous pericardial biopsy at the time of pericardiocentesis significantly increased diagnostic yield in malignant and nonmalignant pericardial effusion.27

Removing fluid by pericardiocentesis in patients with cardiac tamponade usually leads to normalization of intracardiac pressures and cardiac output, returns pericardial pressure to the intrapleural pressure level, and normalizes the right atrial waveform with reappearance of the diastolic y descent wave. If the right atrial pressure remains elevated and prominent y descent appears after pericardiocentesis, the diagnosis of effusive constrictive pericarditis must be considered.28,29

Due to the high reoccurrence rate of pericardial effusion after initial pericardiocentesis, it is recommended that an indwelling pigtail drainage catheter be left in the pericardial space to monitor the rate of accumulation of pericardial content. If the drained amount is less than 50 mL over 24 hours, the catheter can be removed. Otherwise, the catheter should be kept in place so it can be used for intrapericardial delivery of sclerosing agents.30,31

In certain clinical presentations of cardiac tamponade, percutaneous balloon pericardiectomy (PBP) is used in order to avoid the morbidity and discomfort of surgical drainage of the pericardial content.27 With the PBP method, a pericardial “window” is created between the pericardium and the absorbing surface of the pleura or peritoneum.32,33 Typically performed in the cardiac catheterization laboratory, PBP is safe and effective and is usually well tolerated by patients. This procedure may be especially useful for patients with malignant pericardial effusion and a short life expectancy.

SURGICAL DRAINAGE

Open surgical drainage offers several advantages, including complete drainage of the pericardial contents, access to pericardial tissue for histopathologic and microbiologic diagnoses, and the ability to drain loculated effusions. The 3 most common surgical approaches to treating cardiac tamponade include the subxiphoid pericardial window, thoracostomy with creation of a pericardial window, and thoracotomy with
complete pericardiectomy. A pericardial window is the most commonly used technique and may be performed through subxiphoid median sternotomy or left thoracotomy. The subxiphoid approach provides the same degree of long-term survival as other approaches with significantly less operative morbidity and mortality.34

Recently, camera-assisted thoracic surgical techniques have been introduced, where a pericardial window is created under direct thoracoscopic vision.35,36 This method provides effective drainage of loculated pericardial effusions complicated by cardiac tamponade.

**SUMMARY POINTS**

- The fibrous pericardium envelops the entire heart and much of the ascending aorta, the main pulmonary artery, all pulmonary veins, and portions of the inferior and superior venae cavae.
- Cardiac tamponade is a life-threatening compression of the heart caused by accumulation of contents in the pericardium.
- The 4 most common causes of accumulation of fluid in the pericardium in an amount sufficient to cause serious obstruction to the cardiac inflow of blood are infectious and neoplastic diseases, idiopathic pericarditis, and uremia.
- Patients with cardiac tamponade may complain of severe shortness of breath accompanied by chest tightness and dizziness.
- Cardiac tamponade should be considered in any patient with tachycardia, hypotension, and elevation of jugular venous pressure with a prominent X descent and absence of the Y wave. The latter finding differentiates cardiac tamponade from constrictive pericarditis, where the Y descent is prominent. Kussmaul’s sign is generally not seen in pure cardiac tamponade.
- Pulsus paradoxus is a key diagnostic finding of cardiac tamponade. It is defined as an inspiratory fall in systolic arterial pressure greater than 10 mm Hg during normal breathing.
- There are no specific electrocardiogram signs of cardiac tamponade, although low voltage and other signs of pericarditis, including electrical alternans, may be present.
- There are no specific signs of cardiac tamponade on chest radiograph, although cardiomegaly in the presence of clear lung fields may be suggestive.
- Computed tomography and magnetic resonance imaging are inconvenient techniques in patients with acute cardiac tamponade.
- Echocardiographic signs of cardiac tamponade are right atrial and ventricular collapse and significant respiratory variation in tricuspid inflow (> 50%) and mitral inflow (> 30%).
- On hemodynamic tracings during cardiac catheterization in patients with cardiac tamponade, elevation of cardiac diastolic pressures and equalization of those with elevated mean pericardial pressure can be seen. In addition, right atrial and pulmonary capillary wedge pressure tracings show an attenuated or absent Y wave.
- The treatment of cardiac tamponade is urgent percutaneous or surgical drainage of the pericardial content.

**REFERENCES**


