Diagnostic Value of Echocardiography in Cardiac Tamponade

Teresa S. M. Tsang, Jae K. Oh, James B. Seward, A. Jamil Tajik[1]

Abstract

Cardiac tamponade is a life-threatening condition. Accurate diagnosis and prompt intervention are necessary to prevent adverse outcomes. Clinical features of tamponade such as pulsus paradoxus, tachycardia, elevated jugular venous pressure, and hypotension are important clues to the diagnosis, but are non-specific.

Echocardiography allows rapid confirmation of the presence or absence of an effusion, and enables assessment of its hemodynamic impact. Decisions regarding treatment must take into account the clinical presentation and echocardiographic findings.

Echocardiographically-guided pericardiocentesis with catheter drainage is the primary treatment strategy of choice for most large or hemodynamically significant effusions. In contemporary clinical practice, echocardiography is the gold standard for diagnosis of tamponade and is essential for directing treatment.

Key Words: Cardiac tamponade · Doppler echocardiography · Pericardial effusion

Echokardiographische Diagnostik der Perikardtamponade

Zusammenfassung

Die Perikardtamponade ist ein lebensbedrohliches Ereignis. Eine rasche Diagnosestellung und eine sofortige Intervention sind erforderlich, um einen tödlichen Ausgang zu verhindern. Klinische Zeichen der Perikardtamponade sind die Beck'sche Trias mit Hypotonie, Halsvenenstauung und leisen Herztönen. Hinzu kommen der Pulsus paradoxus, definiert als Abnahme der Pulsamplitude (oder Abnahme des Blutdrucks) bei Inspiration, und eine Tachykardie. Beide Symptome weisen auf die Diagnose Perikardtamponade hin, sind aber unspezifisch, da bis auf den Pulsus paradoxus alle anderen Symptome auch bei einer (Links-)Herzinsuffizienz auftreten können. Selbst ein Pulsus paradoxus findet sich gelegentlich bei akuter und chronischer Rechtsherzbelastung, Lungenembolie, Pleuraerguss, konstriktiver Perikarditis und rechtsventrikulärem Infarkt. Umgekehrt kann er trotz bestehender Perikardtamponade fehlen, wenn eine Vorhofseptumdefekt, eine Aortenstenose oder ein vermehrtes intravasales Volumen („Low Pressure Tamponade") vorliegen.

Die Echokardiographie ermöglicht rasch eine Bestätigung der Verdachtsdiagnose und gibt Auskunft über die hämodynamische Relevanz. Zeichen der Perikardtamponade im zweidimensionalen Echokardiogramm sind: die rechtsatriale spätdiastolische Kompression, der rechtsventrikulärer Kollaps in der frühen Diastole, eine paradoxe Septumbewegung, eine abnorme Mitralklappenbewegung, die dilatierte Vena cava ohne inspiratorischen Kollaps und das „Swinging Heart". Die Doppler-Echokardiographie des tamponierenden Perikardergusses stützt sich auf das „konstriktive Muster" mit reduzierter E-Welle (frühe mitrale Füllungsgeschwindigkeit) bei Inspiration, einem reduzierten pulmonalvenösen diastolischen Vorwärtsfluss bei Inspiration, das Gegenteil beider bei Exspiration, der Zunahme des reversen hepatischen Venenflusses in der Diastole sowie dem Verlust des inspiratorischen Kollapses in der unteren Vena cava.

Die Therapieentscheidung hat vor allem die klinische Problematik, aber auch die echokardiographischen Kriterien einer Tamponade einzubeziehen. Die Perikardpunktion unter echokardiographischer oder angiographischer Kontrolle mit Entlastung des Perikardergusses ist die Therapie der Wahl für die großen und hämodynamisch wirksamen Ergüsse. Daher kommt der Echokardiographie als dem Standard in der Diagnostik auch bei der Einleitung der Therapie der Perikardtamponade durch Perikardpunktion entscheidende Bedeutung zu.

Schlüsselwörter: Perikardtamponade · Doppler-Echokardiographie · Perikarderguss

[1] Division of Cardiovascular Diseases and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

Definitions and Terminologies

Cardiac tamponade refers to cardiac compression caused by an accumulation of pericardial fluid that leads to increased intrapericardial pressure and progressive limitation of ventricular diastolic filling, resulting in hemodynamic compromise. That cardiac tamponade is not an all-or-none phenomenon has been demonstrated with the advent of echocardiography. A spectrum of abnormal hemodynamic changes associated with this condition has been identified [1, 2]. Since the term "cardiac tamponade" was well established before the development of echocardiography, it has generally been used to convey the presence of cardiac compression accompanied by overt clinical manifestations including dyspnea, pulsus paradoxus, elevated jugular venous pressure, tachycardia, and hypotension. With echocardiography, detection of the hemodynamic impact of an effusion is possible even in situations where overt clinical signs are absent. This has often been referred to as "tamponade physiology" or "echocardiographic evidence of tamponade". Therefore in the era of echocardiography, there is a distinct advantage in describing cardiac tamponade with overt clinical manifestations as "overt clinical tamponade", and distinguishing this from the scenarios where echocardiographic evidence of tamponade physiology is present without overt clinical manifestations. In addition, the term "low pressure tamponade" has been used to refer to cases where classical clinical findings of tamponade have been altered because of hypovolemia, such that elevated jugular venous pressure and pulsus paradoxus may not be present, although hypotension and tachycardia may be identified in association with echocardiographic evidence of effusion with tamponade physiology.

Pathophysiology

Cardiac tamponade develops secondary to increasing pericardial contents, which may be effusion, blood, clots, pus, gas or combinations of these, leading to raised intrapericardial pressure, progressive limitation of ventricular diastolic filling, and consequent reduction of stroke volume and cardiac output. Large or symptomatic pericardial effusions can develop in association with a wide spectrum of medical and surgical conditions. These include malignancy [3], postoperative state [4], perforation associated with catheter-based procedures [5], infections, ischemia, postradiation connective tissue diseases, and uremia. Some may be idiopathic in nature. Symptoms, however, do not always correlate well with the size of the effusion.

Large effusions that accumulate over an extended period of time, permitting stretching of the parietal pericardium, may be associated with minimal or no symptoms. In contrast, as little as 50 ml of fluid entering the pericardial space rapidly may impair cardiac filling sufficiently to produce marked hemodynamic compromise. Thus, both the volume of fluid and the distensibility of the pericardium contribute to the hemodynamic impact of the effusion.

When increasing pericardial contents move the intrapericardial pressure to the steep portion of the pressure-volume curve (Figure 1), the cardiac chambers must also operate on the steep portion of the pressure-volume curve. Progressive compression of atria and ventricles leads to increasing resistance to cardiac filling with reduction of stroke volume. Because the right atrium and right ventricle are generally at lower pressures than their left-sided counterparts, the diastolic pres-

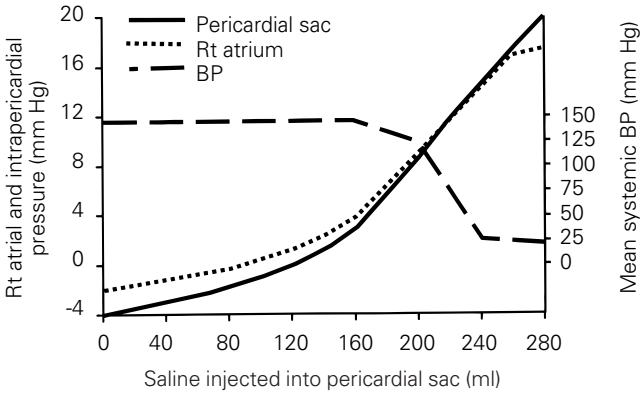

Figure 1
The production of cardiac tamponade by serial injections of saline solution into the pericardial sac of an anesthetized dog. As more than 160 ml saline was injected into the pericardial sac, right atrial and intrapericardial pressures rose more steeply and blood pressure fell more abruptly. Blood pressure (BP) fell to shock levels as intrapericardial pressure exceeded 14 mm Hg (from: Fowler NO, ed. Cardiac diagnosis and treatment, 3rd. edn. Hagerstown, Maryland: Harper & Row, 1980, with permission.)

Abbildung 1
Eine Perikardtamponade wurde tierexperimentell (instrumentierter Hund) durch eine serielle Injektion einer Kochsalzlösung in den Herzbeutel erzeugt: Sobald mehr als 150 ml Kochsalzlösung in den Herzbeutel eingebracht wurden, stieg der Druck im rechten Vorhof und Herzbeutel steiler an und der Blutdruck nahm besonders deutlich ab. Wenn der intraperikardiale Druck 14 mm Hg überstieg, kam es zum kardiogenen Schock.

sures in these chambers equilibrate with the rising intrapericardial pressure sooner, and collapse of these chambers usually precedes that of the left-sided chambers. This is not necessarily the case if the effusion is loculated over the left chambers, as occurs more commonly in postoperative patients.

When the intrapericardial pressure reaches the critical point of equilibrating with the ventricular diastolic pressures, systemic venous congestion occurs and cardiac output decreases, with a drop in blood pressure (Figure 1) [6]. With inspiration, the increase in right ventricular inflow leads to leftward shifting of the interventricular septum and decreased filling of the left side of the heart. That is, the filling of the right heart occurs at the expense of the left heart, thus reducing cardiac output, which is detected clinically as a significant drop in systolic blood pressure, or classically, the finding of pulsus paradoxus. The reverse occurs on expiration.

Clinical Diagnosis of Cardiac Tamponade

The classic Beck's triad [7], described in 1935, referred to acute cardiac compression accompanied by physical findings of hypotension, increased jugular venous pressure, and quiet (or muffled) heart sounds, than can occur in cardiac trauma, or rupture, where the increase of intrapericardial pressure is rapid. Tachycardia is often present in these patients. In contrast, these physical findings may be absent if an effusion develops insidiously, with a more gradual increase of intrapericardial pressure over a period of time, such as occurs in many medical patients. This was highlighted in the study by Guberman et al. [8], which demonstrated that the classic Beck's triad was not present in most patients with cardiac tamponade. The majority of the patients in that study had well-preserved blood pressure and heart sounds.

The combination of classic symptoms and signs such as dyspnea, tachycardia, elevated jugular venous pressure, pulsus paradoxus and hypotension leads the clinician to strongly suspect the diagnosis. There are, however, other conditions in which some of these findings are not present. Even though pulsus paradoxus is most frequently described in association with cardiac tamponade, it may occur in patients without tamponade as a result of acute or chronic obstructive airway disease [9], pulmonary embolism, tension pneumothorax, large pleural effusions [10], effusive constrictive pericarditis, right ventricular infarction, restrictive cardiomyopathy, extreme obesity, or tense ascites. Conversely, pulsus paradoxus may be absent during tamponade because of atrial septal defect, severe aortic stenosis, left ventricular dysfunction, or simply decreased intravascular volume (low pressure tamponade) [11, 12]. Except for the finding of pulsus paradoxus, many typical clinical signs and symptoms of cardiac tamponade, such as dyspnea, tachycardia, hypotension, and elevated jugular venous pressure can occur in a patient with congestive heart failure. In the case of pulmonary embolism, all clinical manifestations classically associated with tamponade may be present. Therefore, the signs and symptoms typically described in association with overt clinical tamponade are relatively non-specific, and may not necessarily be all present. The electrocardiogram may provide additional clues, such as findings of low voltage or electrical alternans. An enlarged cardiac silhouette on chest radiographs may be suggestive of the presence of an effusion, but is also non-specific and non-diagnostic.

Echocardiographic Diagnosis of Cardiac Tamponade

Two-Dimensional (2-D) Echocardiography

Echocardiography has become the standard modality for assessing pericardial effusion and cardiac tamponade [13, 14]. Morphologic features suggestive of tamponade physiology, and easily confirmed by 2-D echocardiography, include right atrial compression during late diastole [15] (Figure 2), right ventricular collapse during early diastole [16] (Figure 3), abnormal septal motion [17], abnormal mitral valve motion [18], dilated inferior vena cava with lack of inspiratory collapse [19], and a swinging heart [20, 21]. Isolated left atrial or left ventricular collapse may occur in localized left-sided compression or in severe pulmonary hypertension without any evidence of right atrial or right ventricular collapse [22, 23].

Doppler Echocardiography

More recently, Doppler echocardiography has defined the physiologic mechanisms of tamponade and has evolved to become a sensitive means of identifying compressive hemodynamics [24]. Doppler hemodynamic findings of tamponade are based on pressure variations between the thorax and cardiac chambers. The normal rise and fall of intrathoracic pressures create characteristic variations in atrial and ventricular filling relative to the respiratory cycle. Two principles regarding tamponade physiology are critical to its understanding: Firstly,

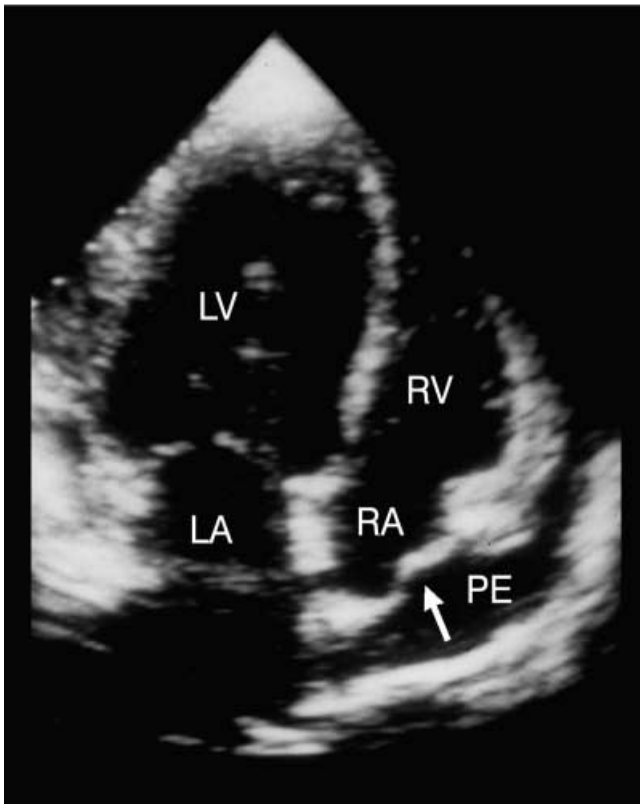

Figure 2
Two-dimensional echocardiography showing late diastolic right atrial inversion from apical 4-chamber view. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; PE = pericardical effusion (from: Oh JK, Seward JB, Tajik AJ. The echo manual, 2nd edn. Philadelphia: Lippincott-Raven, 1999:183, with permission of Mayo Foundation).

Abbildung 2
Die zweidimensionale Echokardiographie zeigt im Vier-Kammer-Blick bei Perikardtamponade eine Einziehung des rechten Vorhofs. LA = linker Vorhof; LV = linker Ventrikel; RA = rechter Vorhof; RV = rechter Ventrikel; PE = Perikarderguss.

the dissociation of intracardiac and intrathoracic pressures, and secondly, the increased ventricular interdependence during diastolic filling. Thus, on inspiration, there is a decrease in intrathoracic pressure, but the negative intrathoracic pressure is dissociated from the intracardiac pressure because of the intervening pericardial fluid and increased intrapericardial pressure. Consequently, there is less reduction in intracardiac pressure, resulting in an attenuated decrease in the left sided filling gradient. In addition, on inspiration, there is increased right ventricular filling, "shifting" the septum to the left, and further compromising left ventricular filling. These factors contribute to decreased mitral early filling (E) velocity (Figure 4) and decreased pulmonary venous diastolic forward flow on inspiration (Figure 5). On expiration, exactly the opposite sequence of events occurs (Figures 4 and 5) [24, 25], accompanied by an increase in hepatic venous diastolic flow reversal [24–26] (Figure 6) and a decrease or loss of change in superior vena cava flow velocity during inspiration and expiration [26]. Because of an increase in right atrial pressure, the inferior vena cava is characteristically dilated, with decreased or loss of normal inspiratory collapse (Figure 6). The physiology involved in tamponade, as assessed by Doppler echocardiography, is identical to that described for constrictive pericarditis (tamponade or constrictive physiology).

Transesophageal Echocardiography (TEE)

When transthoracic echocardiography is inadequate for confirmation of the presence of a pericardial effusion or evaluation of its hemodynamic impact, TEE may be considered. Transesophageal echocardiography is particularly valuable in some postoperative patients who

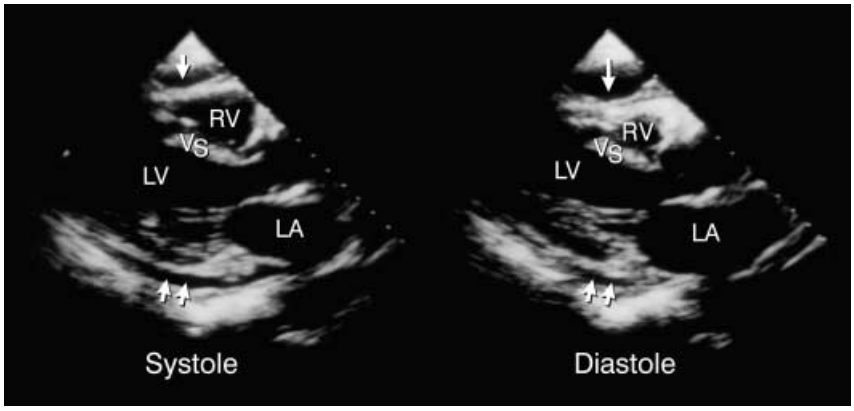

Figure 3
Two-dimensional echocardiographic assessment of hemodynamic impact of a pericardial effusion. Parasternal long-axis view shows evidence of tamponade during systole and diastole. Although the PE (double arrowhead) appears small, right ventricular (RV) free wall collapse is clearly present (arrow) (LA = left atrium; LV = left ventricle; VS = ventricular septum) (from: Oh JK, Seward JB, Tajik AJ. The echo manual, 2nd edn. Philadelphia: Lippincott-Raven, 1999:183, with permission of Mayo Foundation).

Abbildung 3
Zweidimensionale Echokardiographie eines hämodynamisch relevanten Perikardergusses. In der parasternalen langen Achse finden sich Tamponadezeichen während der Diastole und Systole. Obwohl der Perikarderguss (Doppelpfeil) klein erscheint, kollabiert der rechte Ventrikel (Pfeil) (LA = linker Vorhof; LV = linker Ventrikel; VS = Ventrikelseptum).

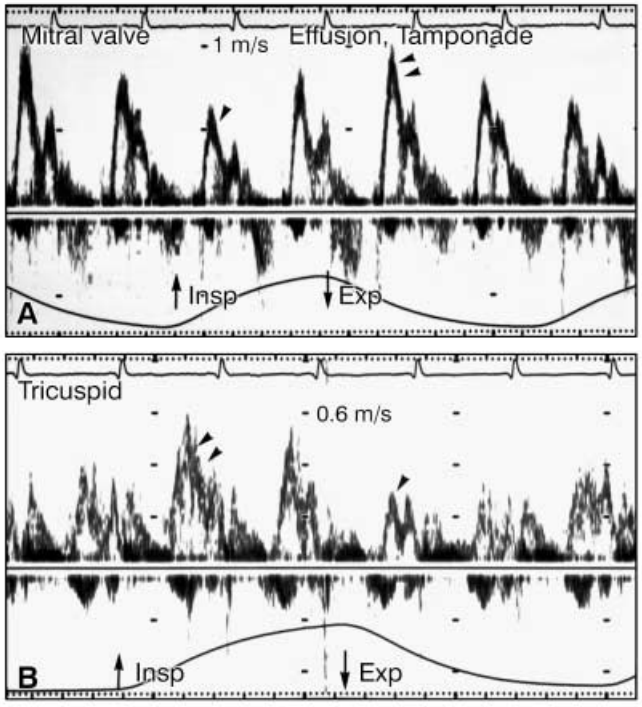

Figure 4
Doppler assessment of hemodynamic impact of a pericardial effusion: Top (A) and bottom (B) ventricular filling pattern in cardiac tamponade. On inspiration (Insp) (arrowhead), mitral E velocity decreases significantly and opposite changes occur on expiration (Exp) (double arrowhead). Reciprocal changes are identified on the bottom (from: Oh JK, Hatle LK, Mulvagh SL, et al. Transient constrictive pericarditis: diagnosis by two-dimensional Doppler echocardiography. Mayo Clin Proc 1993;68:1158–64, with permission).

Abbildung 4
Doppler-echokardiographischer Nachweis der hämodynamischen Relevanz von Perikardergüssen: (A) und (B) Ventrikelfüllungsmuster bei Perikardtamponade. Bei Inspiration (Insp) (Pfeil) sinkt die E-Geschwindigkeit der Mitralklappe signifikant ab. Gegensätzliche Veränderungen finden sich bei Exspiration (Exp) (Doppelpfeil). Reziproke Veränderungen sind im unteren Anteil der Abbildung sichtbar. Insgesamt handelt es sich um das „konstriktive Muster".

develop tamponade due to a loculated pericardial effusion or intrapericardial clot, which may not be readily appreciated with transthoracic echocardiography [27–29]. The hemodynamic significance of an effusion can be determined during TEE by assessing the transmitral and pulmonary venous flow patterns with simultaneous respirometry [30, 31].

Diagnostic Value of Echocardiography in Tamponade

Historically, the decision to initiate drainage of a pericardial effusion relied principally on the clinical findings of tamponade. However, since many of these physical findings lack specificity [8], a decision to intervene

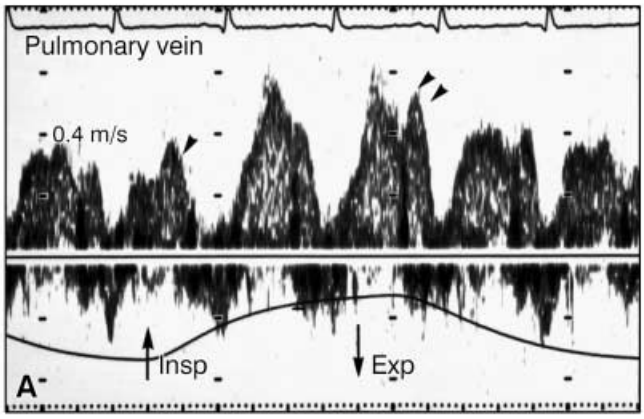

Figure 5
Doppler assessment of hemodynamic impact of a pericardial effusion: characteristics of tamponade include inspiratory (Insp) decrease (arrowhead) and expiratory (Exp) increase (double arrowhead) in diastolic pulmonary venous forward flow (from: Oh JK, Hatle LK, Mulvagh SL, et al. Transient constrictive pericarditis: diagnosis by two-dimensional Doppler echocardiography. Mayo Clin Proc 1993;68:1158–64, with permission).

Abbildung 5
Dopplerechokardiographischer Befund eines hämodynamischen relevanten Perikardergusses: Die Charakteristika einer Tamponade umfassen einen inspiratorischen (Insp) Abfall (Pfeil) und expiratorischen (Exp) Anstieg (Doppelpfeil) des distolischen Vorwärtsflusses in der Pulmonalvene.

based solely on their presence or absence is not optimal. For instance, patients with preexisting hypertension may demonstrate an elevated blood pressure despite the presence of cardiac tamponade [32]. Patients who are hypovolemic and have low pressure tamponade may not present with classic clinical features [12]. In patients with atrial septal defects, a paradoxical pulse may not be present due to equilibration of flow across the atrial septum [33]. In contrast, tamponade physiology, such as right atrial compression identified by echocardiography, can occur in the complete absence of any clinical sign of tamponade [34]. Despite the existence of a good correlation between absence of chamber collapse and absence of clinical tamponade, the correlation between the presence of chamber collapse and clinical tamponade is poor [35]. In one study of patients with moderate or large pericardial effusions, 34% of patients had collapse of one or more cardiac chambers by echocardiography in the absence of overt clinical tamponade [35]. Echocardiography is safe, and can provide rapid confirmation of presence or absence of pericardial effusion, as well as precise hemodynamic assessment, allowing the most appropriate management decision to be taken.

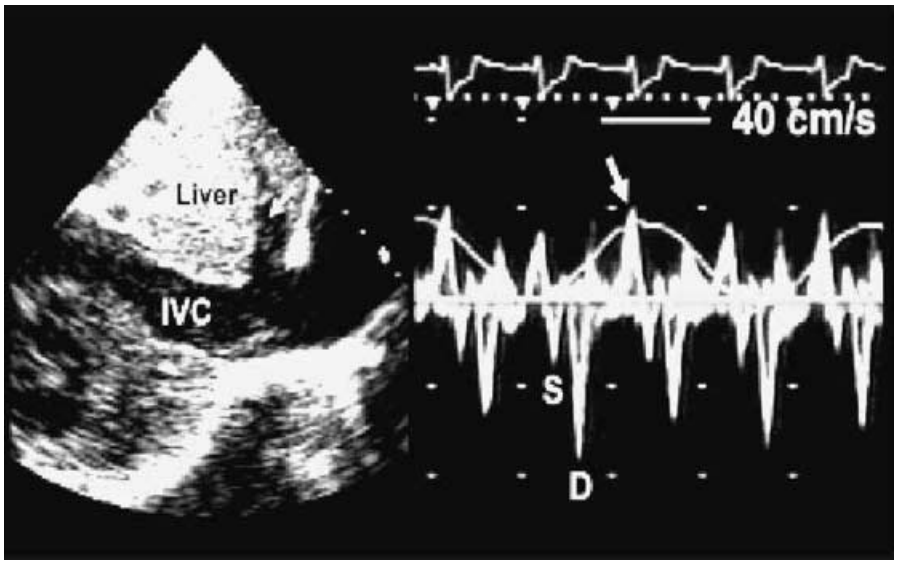

Figure 6
Dilated inferior vena cava (left) in tamponade consistent with elevated right atrial pressure. Increased hepatic venous diastolic flow reversal in tamponade (right) (IVC = inferior vena cava; S = systole; D = diastole).

Abbildung 6
Dilatierte inferiore Vena cava (links) bei Perikardtamponade infolge des erhöhten rechtsatrialen Drucks. Erhöhter reverser diastolischer Fluss in der Lebervene bei Tamponade (rechts) (IVC = inferiore Vene cava; S = Systole; D = Diastole).

Treatment

Detailed discussion regarding treatment options for pericardial effusion and tamponade is beyond the scope of this review. However, especially for effusions that are large or hemodynamically significant (evidence of raised intrapericardial pericardial pressure), evacuation of the pericardial fluid can be life saving and provide relief of symptoms.

Blind pericardiocentesis, which was fraught with high morbidity and mortality [36, 37] is now considered obsolete. Although both percutaneous techniques, such as pericardiocentesis guided by electrocardiography, fluoroscopy, or computed tomography, and surgical techniques, including balloon pericardiotomy, subxiphoid pericardial drainage, video-assisted thoracoscopy, pericardial-peritoneal drainage, pericardial window surgery, or pericardiectomy (partial or complete), are all used in the management of effusions, echo-guided pericardiocentesis with pericardial catheter drainage has been shown to be a safe and effective primary management strategy for most pericardial effusions [3–5], even in children [38]. The procedure is simple, well-tolerated, and complication rates are low. Details regarding the techniques have been published [3, 39]. Potential major complications requiring treatment, although infrequent, may include cardiac chamber puncture, vessel injury, pneumothorax, arrhythmias, infection, and, very rarely, death. Minor complications include transient cardiac chamber entry, vasovagal reactions, pneumopericardium, transient arrhythmia, and small pneumothorax, generally resolve spontaneously and require only appropriate monitoring and follow-up.

Echo-guided pericardiocentesis is not considered as the primary or definitive treatment of choice in a few situations. In patients with myocardial rupture or Type A aortic dissection, the relief of tamponade during pericardiocentesis may potentially extend the rupture or dissection [40]. Also, tamponade coagulum from any cause is rarely correctable with pericardiocentesis alone. Emergency surgery is usually necessary in these situations, and the mortality rate is high. The balance of risks and benefits of emergency pericardiocentesis in these patients must be assessed on a case-by-case basis. Other unusual clinical situations, such as prior pneumonectomy or severe pulmonary hypertension, may potentially convert a procedural complication that is generally minor, such as pneumothorax or right heart puncture, into a much more serious condition.

Conclusion

The spectrum of hemodynamic abnormalities associated with cardiac tamponade is wide. Classic physical findings of overt tamponade may not be all present. Echocardiography, which allows rapid confirmation of the presence of an effusion and precise assessment of its hemodynamic impact, is the gold standard both for diagnosis and for guidance of treatment of an effusion in contemporary clinical practice.

References

1. Reddy PS, Curtiss EI, Uretsky BF. Spectrum of hemodynamic changes in cardiac tamponade. Am J Cardiol 1990;66:1487–91.
2. Levine MJ, Lorell BH, Diver DJ, et al. Implications of echocardiographically assisted diagnosis of pericardial tamponade in contemporary medical patients: detection before hemodynamic embarrassment. J Am Coll Cardiol 1991;17:59–65.
3. Tsang TSM, Seward JB, Barnes ME, et al. Outcomes of primary and secondary treatment of pericardial effusion in patients with malignancy. Mayo Clin Proc 2000;75:248–53.
4. Tsang TSM, Barnes ME, Hayes SN, et al. Clinical and echocardiographic characteristics of significant pericardial effusions following cardiothoracic surgery and outcomes of echo-guided pericardiocentesis for management. Mayo Clinic experience, 1979–1998. Chest 1999;116:322–31.
5. Tsang TSM, Freeman WK, Barnes ME, et al. Rescue echocardiographically guided pericardiocentesis for cardiac perforation complicating catheter-based procedures: the Mayo Clinic experience. J Am Coll Cardiol 1998;32:1345–50.
6. Sharp JT, Bunnell IL, Holland JF, et al. Hemodynamics during induced cardiac tamponade in man. Am J Med 1960;29:640–6.
7. Beck CS. Two cardiac compression triads. JAMA 1935;104:714–6.
8. Guberman BA, Fowler NO, Engel PJ, et al. Cardiac tamponade in medical patients. Circulation 1981;64:633–40.
9. Hartert TV, Wheeler AP, Sheller JR. Use of pulse oximetry to recognize severity of airflow obstruction in obstructive airway disease: correlation with pulsus paradoxus. Chest 1999;115:475–81.
10. Alam H, Levitt A, Molyneaux R, et al. Can pleural effusions cause cardiac tamponade? Chest. 1999;116:1820–2.
11. Antman EM, Cargill V, Grossman W. Low-pressure cardiac tamponade. Ann Intern Med. 1979;91:403–6.
12. Hayes S, Freeman W, Gersh B. Low pressure cardiac tamponade: diagnosis facilitated by Doppler echocardiography. Br Heart J 1990;63:136–40.
13. Feigenbaum H, Waldhausen JA, Hyde LP. Ultrasound diagnosis of pericardial effusion. JAMA 1965;191:711–4.
14. Schutzman JJ, Obarski DO, Pearce GL, et al. Comparison of Doppler and two-dimensional echocardiography for assessment of pericardial effusion. Am J Cardiol 1992;70:1353–7.
15. Kronzon I, Cohen ML, Winer HE. Diastolic atrial compression: a sensitive echocardiographic sign of cardiac tamponade. J Am Coll Cardiol 1983;2:770–5.
16. Armstrong WF, Schilt BF, Helper DJ, et al. Diastolic collapse of the right ventricle with cardiac tamponade: an echocardiographic study. Circulation 1982;65:1491–6.
17. Cosio FG, Martinez JP, Serrano CM, et al. Abnormal septal motion in cardiac tamponade with pulse paradoxus. Echocardiographic and hemodynamic observations. Chest 1977;71:787–8.
18. Settle HP, Adolph RJ, Fowler NO, et al. Echocardiographic study of cardiac tamponade. Circulation 1977;56:951–9.
19. Himelman RB, Kircher B, Rockey DC, et al. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. J Am Coll Cardiol 1988;12: 1470–7.
20. Feigenbaum H, Zaky A, Grabhorn L. Cardiac motion in patients with pericardial effusion: a study using ultrasound cardiography. Circulation 1966;34:611–9.
21. Kronzon I, Cohen ML, Winer HE. Contribution of echocardiography to the understanding of the pathophysiology of cardiac tamponade. J Am Coll Cardiol 1983;1:1180–2.
22. Fusman B, Schwinger ME, Charney R, et al. Isolated collapse of left-sided heart chambers in cardiac tamponade: demonstration by two-dimensional echocardiography. Am Heart J 1991;121:613–6.
23. Frey MJ, Berko B, Palevsky H, et al. Recognition of cardiac tamponade in the presence of severe pulmonary hypertension. Ann Intern Med 1989;111:615–7.
24. Appleton CP, Hatle LK, Popp RL. Cardiac tamponade and pericardial effusion: respiratory variation in transvalvular flow velocities studied by Doppler echocardiography. J Am Coll Cardiol 1988; 11:1020–30.
25. Burstow DJ, Oh JK, Bailey KR, et al. Cardiac tamponade: characteristic Doppler observations. Mayo Clin Proc 1989;64:312–24.
26. Oh JK, Seward JB, Tajik AJ. The echo manual, 2nd edn. Philadelphia: Lippincott-Raven 1999.
27. Berge KH, Lanier WL, Reeder GS. Occult cardiac tamponade detected by transesophageal echocardiography. Mayo Clin Proc 1992;67:667–70.
28. Hutchinson SJ, Smalling RG, Albornoz M, et al. Comparison of transthoracic and transesophageal echocardiography in clinically overt or suspected pericardial heart disease. Am J Cardiol 1994;74: 962–5.
29. Kochar GS, Jacobs LE, Kotler MN. Right atrial compression in postoperative cardiac patients: detection by transesophageal echocardiography. J Am Coll Cardiol 1990;16:511–6.
30. Castello R, Pearson AC, Lenzen P, et al. Evaluation of pulmonary venous flow by transesophageal echocardiography. J Am Coll Cardiol 1991;18:65–71.
31. Nishimura RA, Abel MD, Hatle LK, et al. Relation of pulmonary vein to mitral flow velocities by transesophageal Doppler echocardiography. Effect of different loading conditions. Circulation 1990;81: 1488–97.
32. Brown J, MacKinnon D, King A, et al. Elevated arterial blood pressure in cardiac tamponade. New Engl J Med 1992;327:463–6.
33. Winer HE, Kronzon I. Absence of paradoxical pulse in patients with cardiac tamponade and atrial septal defects. Am J Cardiol 1979;44:378-80.
34. Gillam LD, Guyer DE, Gibson TC, et al. Hydrodynamic compression of the right atrium: a new echocardiographic sign of cardiac tamponade. Circulation 1983;68:294–301.
35. Jorde M, Sgrista-Sauleda J, Permanyer-Miralda G, et al. Correlation between clinical and Doppler echocardiographic findings in patients with moderate and large pericardial effusion: implications for the diagnosis of cardiac tamponade. Am Heart J 1999;138: 759–64.
36. Moores DW, Dziuban SW. Pericardial drainage procedures. Chest Surg Clin North Am 1995;5:359–72.
37. Buzaid AC, Garewal HS, Greenberg BR. Managing malignant pericardial effusion. West J Med 1989;150:174–9.
38. Tsang T, El-Najdawi E, Freeman W, et al. Percutaneus echocardiographically guided pericardiocentesis in pediatric patients: evaluation of safety and efficacy. J Am Soc Echocardiogr 1998;11:1072-7.
39. Tsang TSM, Freeman WK, Sinak LJ, et al. Echocardiographically guided pericardiocentesis: evolution and state-of-art technique. Mayo Clin Proc 1998;73:647–52.
40. Isselbacher EM, Cigarroa JE, Eagle KA. Cardiac tamponade complicating proximal aortic dissection. Is pericardiocentesis harmful? Circulation 1994;90:2375–8.

Address for Correspondence:
Teresa S. M. Tsang, MD, FRCPC, FACC, Mayo Clinic,
200 First Street SW, Rochester, MN 55905, USA,
Phone (+1/507) 2664130, Fax 2840537,
e-mail: Tsang.Teresa@mayo.edu