Constrictive pericarditis

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INTRODUCTION — The normal pericardium is a fibroelastic sac surrounding the heart which contains a thin layer of fluid. When larger amounts of fluid accumulate (pericardial effusion) or when the pericardium becomes scarred and inelastic, one of three pericardial compressive syndromes may occur:

- Cardiac tamponade — Cardiac tamponade, which may be acute or subacute, is characterized by the accumulation of pericardial fluid under pressure. Variants include low pressure (occult) and regional tamponade.
- Constrictive pericarditis — Constrictive pericarditis is the result of scarring and consequent loss of the normal elasticity of the pericardial sac. Pericardial constriction is typically chronic, but variants include subacute, transient, and occult constriction. Pathologically, this results in chronic inflammation and, frequently, calcification. Grossly, the pericardium is considerably thicker than normal in approximately 80 percent of cases.
- Effusive-constrictive pericarditis — This variant is characterized by constrictive physiology with a coexisting pericardial effusion, usually with cardiac tamponade [1]. Such patients may be mistakenly thought to have only cardiac tamponade; however, elevation of the right atrial and pulmonary wedge pressures persists after drainage of the pericardial fluid.

In both typical constrictive pericarditis and effusive-constrictive pericarditis, cardiac filling is impeded by an external force. The normal pericardium can stretch to accommodate physiologic changes in cardiac volume, and only after this reserve volume has been filled does the pericardium stiffen. In the pericardial compressive syndromes, the pericardium is inelastic, resulting in minimal ability to adapt to volume changes.

As a result, an important pathophysiologic feature of constrictive pericarditis is greatly enhanced ventricular interaction or interdependence, in which the hemodynamics of the left and right heart chambers are directly influenced by each other to a much greater degree than normal.

The physiology, clinical presentation, diagnosis, and treatment of constrictive pericarditis will be reviewed here. Issues related to cardiac tamponade, and the evaluation and management of pericardial diseases that do not compromise hemodynamics, are discussed separately. (See "Cardiac tamponade" and "Diagnosis and treatment of pericardial effusion" and "Evaluation and management of acute pericarditis".)

PHYSIOLOGY

Constrictive pericarditis — In constrictive pericarditis the upper limit of cardiac volume is constrained by the inelastic pericardium. Compression does not occur until the cardiac volume approximates that of the pericardium, which occurs in mid through late diastole. As a result, the bimodal pattern of venous return is preserved, since there is no compression in systole and early

diastole.

Venous return to the right heart does not increase during inspiration in constrictive pericarditis, because the respiratory variation in intrathoracic pressure with inspiration is not transmitted to the heart chambers. Thus, pulmonary venous pressure, but not left ventricular pressure, declines during inspiration, leading to a reduction in left ventricular volume. Because of ventricular interaction, the right heart volume expands via shift of the interventricular septum.

With constrictive pericarditis, early diastolic filling is even more rapid than normal. Compression does not occur until the cardiac volume approximates that of the pericardium, which begins in mid-diastole. As a result, the majority of ventricular filling occurs rapidly in early diastole. Neither ventricle fills in mid through end diastole. In severe cases ventricular volumes and stroke volumes are reduced.

The hemodynamic findings and distinction from restrictive cardiomyopathy are discussed in more detail elsewhere. (See "Hemodynamics in constrictive pericarditis versus restrictive cardiomyopathy".)

Comparison with tamponade — Tamponade and constriction, while having several features in common, differ in how they alter diastolic filling of the ventricles, leading to different clinical manifestations and different findings on physical examination, echocardiography, including Doppler echocardiography, and invasive hemodynamic assessment. (See "Cardiac tamponade".)

Features in common — Features that are common to tamponade and constriction include:

- Diastolic dysfunction and preserved ventricular ejection fraction. Constrictive pericarditis is an important cause of diastolic dysfunction or diastolic heart failure.
- Heightened ventricular interaction.
- Increased respiratory variation of ventricular inflow and outflow manifested by pulsus paradoxus. Pulsus paradoxus, however, is a less frequent finding in constrictive pericarditis than in tamponade.
- Equally elevated central venous, pulmonary venous, and ventricular diastolic pressures.
- Pulmonary hypertension; systolic 35 to 50 mmHg.

Distinctive features — Features that are distinct between tamponade and constriction include:

- In tamponade, the pericardial space is open and transmits respiratory variation in thoracic pressure to the heart, while in constrictive pericarditis the cavity is obliterated and the pericardium does not "see" fluctuation in thoracic pressure.
- In tamponade systemic venous return increases enlarging the right heart and encroaching on the left, while in constrictive pericarditis systemic venous return does not increase with inspiration. The mechanism of diminished left ventricular and increased right ventricular volume in constrictive pericarditis is impaired left ventricular filling due to a lesser pressure gradient from the pulmonary veins.
- Unlike in tamponade, equalization of right atrial, pulmonary venous and ventricular diastolic pressures is not present throughout the respiratory cycle in constrictive pericarditis. This is because right atrial pressure is not changed by inspiration, but pulmonary arterial wedge pressure falls.

ETIOLOGY — Constrictive pericarditis can occur after virtually any pericardial disease process, but only rarely follows recurrent acute pericarditis [2]. (See "Recurrent pericarditis".)

A few large series of patients with constrictive pericarditis diagnosed at pericardiectomy have been

described, including 95 from Stanford [3], 135 from the Mayo Clinic [4], and 163 from the Cleveland Clinic [5]. The frequency of various causes in these reports, which is influenced by referral bias, is as follows:

- Idiopathic or viral — 42 to 49 percent
- Following cardiac surgery — 11 to 37 percent
- Following radiation therapy — 9 to 31 percent, mostly for Hodgkin's disease or breast cancer
- Connective tissue disorder — 3 to 7 percent
- Postinfectious (tuberculous or purulent pericarditis) — 3 to 6 percent
- Miscellaneous causes (malignancy, trauma, drug-induced, asbestosis, sarcoidosis, uremic pericarditis) — 1 to 10 percent

Five cases of surgically-confirmed cases of constrictive pericarditis after orthotopic heart transplant were recently reported [6].

Tuberculosis accounted for 49 percent of cases of constrictive pericarditis in a series reported in 1962 [7] but is now only a rare cause of constrictive pericarditis in developed countries. However, this disorder may be increasing among immigrants from underdeveloped nations and patients with HIV infection. (See "Tuberculous pericarditis".)

CLINICAL EVALUATION

History — Patients with pericardial constriction may present with two types of complaints: those related to fluid overload, ranging from peripheral edema to anasarca; and those related to diminished cardiac output response to exertion, such as fatigability and dyspnea on exertion. Pericardial constriction should be considered in any patient with an unexplained elevation in jugular venous pressure, particularly if there is a history of a predisposing condition.

In the Mayo Clinic series, 67 percent of patients presented with symptoms of heart failure (HF), 8 percent with chest pain, 6 percent with abdominal symptoms, 4 percent with atrial arrhythmia, and only 5 percent with symptoms of cardiac tamponade [4].

Physical examination — Except for patients with mild constriction, the physical examination reveals elevated jugular venous pressure, which was observed in 93 percent of the patients in the Mayo Clinic series [4]. The x and y troughs are more prominent than the a and v peaks and the inspiratory decline in venous pressure is confined to the depth of the y descent. (See 'Hemodynamic evaluation' below.) Occult constriction has been described in patients who are volume depleted; in this setting, the increase in venous pressure may not become apparent without rapid volume expansion [8].

Common findings with more severe constriction include peripheral edema, ascites, pulsatile hepatomegaly (part of the syndrome of congestive hepatopathy), and pleural effusion [9]. These findings may lead to the misdiagnosis of chronic liver disease. An important distinguishing feature is that, in patients with cirrhosis, the jugular pressure is normal or only slightly elevated, unless there is tense ascites [10]. (See "Clinical manifestations and diagnosis of edema in adults", section on 'Distribution of edema and central venous pressure' and "Congestive hepatopathy".)

Additional important features include:

- Pulsus paradoxus is not common in the absence of pericardial fluid or pulmonary disease [11], occurring in 19 percent of patients in the Mayo Clinic series [4].
- Kussmaul's sign (the lack of an inspiratory decline in jugular pressure) may be present but does

not distinguish constriction from severe tricuspid valve disease or right-sided HF. This finding was noted in only 13 percent of patients in the Stanford series [12] and 21 percent in the Mayo Clinic series [4]; in our experience, Kussmaul's sign is more common. (See "Examination of the jugular venous pulse".)

- A pericardial knock, which is nothing more than a third heart sound, may be audible and sometimes palpable. It was observed in 47 percent of patients in the Mayo Clinic series, while 16 percent had a pericardial friction rub [4].
- Profound cachexia occurs with late-stage disease; because of the associated ascites and hepatic dysfunction, the patient's appearance simulates that of severe malnutrition.

Electrocardiography — There are no specific electrocardiographic findings in pericardial constriction. Nonspecific ST and T wave changes are common. Low voltage is sometimes present, atrial fibrillation is common in advanced cases as is P mitrale in less severe and less chronic cases. In a series of 143 patients with surgically confirmed constriction, 22 percent had atrial fibrillation and 27 percent had low voltage [13].

Chest radiograph — The presence of a ring of calcification around the heart, best seen on lateral or anterior oblique chest radiograph projections, strongly suggests pericardial constriction in patients with symptoms of right HF (picture 1) [9]. Pericardial calcification can occur in the absence of constriction, but is usually less dense and has a patchy distribution. The clinical value of calcification of the pericardium in constrictive pericarditis was evaluated in a retrospective review of 135 patients with constrictive pericarditis, confirmed surgically or at autopsy; 27 percent had pericardial calcification [14]. Compared to those without calcification, patients with calcification had the following:

- A greater likelihood of having idiopathic pericardial disease (67 versus 21 percent)
- A longer duration of symptoms
- A greater likelihood of having a pericardial knock, larger atria, and atrial arrhythmias
- A higher perioperative mortality, but the same long-term survival

Echocardiography — Echocardiography is an essential adjunctive procedure in patients with pericardial constriction. The use of echocardiography for the evaluation of all patients with suspected pericardial disease was given a class I recommendation by a 2003 task force of the American College of Cardiology (ACC), the American Heart Association (AHA), and the American Society of Echocardiography (ASE) [15].

One or more of the following findings may be seen [2,9,13,16,17] (see "Echocardiographic evaluation of the pericardium"):

- Increased pericardial thickness, sometimes with calcification. Transthoracic echocardiography (TTE) is insensitive, as mildly increased pericardial thickening is often missed, and false positive results can be obtained if the gain is set too high.

  Measurement of pericardial thickness by transesophageal echocardiography (TEE) correlates strongly with that obtained by computed tomography [18]. The 2003 ACC/AHA/ASE task force gave a class IIb recommendation (usefulness/efficacy less well established by evidence/opinion) to TEE assessment of pericardial thickness to support a diagnosis of constrictive pericarditis [15].

- Abrupt posterior motion of the ventricular septum in early diastole with inspiration (septal shudder and bounce) is caused by underfilling of the left ventricle (due to the decreased pulmonary vein-left atrial gradient with inspiration), allowing redistribution of blood from the left to the right ventricle. Systemic venous return is not increased. Thus, respiratory variation in superior venal caval flow suggests obstructive lung disease rather than constrictive pericarditis [19].

- M-mode may show notching of the ventricular septum in early diastole or with atrial systole due to transient reversal of ventricular septal transmural pressure at these times in the cardiac cycle [20]. Other M-mode signs include rapid posterior motion of the left ventricular posterior wall in early diastole followed by a flattening (also a feature of restrictive cardiomyopathy), rapid left atrial emptying, and premature opening of the pulmonic valve (as right ventricular diastolic pressure rises above pulmonary arterial pressure).

  No sign or combination of signs on M-mode is diagnostic of constrictive pericarditis. However, a normal study virtually rules out the diagnosis [21].

- Two-dimensional echocardiography reveals dilation and absent or diminished collapse of the inferior vena cava and hepatic veins (plethora), moderate biatrial enlargement (severe enlargement is more compatible with restrictive cardiomyopathy), a sharp halt in ventricular diastolic filling, hypermobile atrioventricular valves, and an abnormal contour between the posterior left ventricular the left atrial posterior walls [22,23].

Left ventricular systolic function as judged by the ejection fraction is typically normal, but may be impaired in mixed constrictive-restrictive disease (eg, radiation-induced disease or prior cardiac surgery).

In the above report of 143 patients with surgically confirmed constriction, 138 underwent TTE [13]. Increased pericardial thickness was seen in 37 percent, abnormal septal motion in 49 percent, and atrial enlargement in 61 percent.

Doppler echocardiography is critically important for diagnosis, and usually shows the following findings:

- High E velocity of right and left ventricular (LV) inflow, due to the abnormally rapid early diastolic filling associated with the combination of a small volume and rapidly recoiling ventricle. Tissue Doppler shows a prominent E (unless slowed by mitral annular calcification), which is an important point of distinction from restrictive cardiomyopathy in which the transmitral E is tall and narrow but the tissue E is diminutive [24]. The usually positive linear relation between E/tissue E and left atrial pressure, which is useful for assessing left atrial pressure in cardiomyopathy is reversed (annular paradox) in constrictive pericarditis [25]. (See "Hemodynamics in constrictive pericarditis versus restrictive cardiomyopathy".)

- The propagation velocity of early diastolic transmitral flow on color M-mode is normal or increased.

- Mitral inflow velocity falls as much as 25 to 40 percent and tricuspid velocity greatly increases in the first beat after inspiration. The respiratory variation in pulmonary venous flow is even more pronounced [26]. These phenomena, which are manifestations of greatly enhanced ventricular interaction, are not present in either normal subjects or patients with restrictive cardiomyopathy [27]. Increased respiratory variation of mitral inflow may be missing in patients with markedly elevated left atrial pressure, but can sometimes be brought out in such patients by preload reduction with head up tilt or diuretic administration [28].

- Hepatic vein flow reversal increases with expiration, reflecting the ventricular interaction and the dissociation of intracardiac and intrathoracic pressures [2].

CT scan — Computed tomographic (CT) scanning of the heart, obtained by rapid scanning gated to the cardiac cycle, is extremely useful in the diagnosis of constrictive pericarditis. Findings include increased pericardial thickness and calcification [9]. In the review of 143 patients with surgically confirmed constriction cited above, 97 underwent CT imaging. A pathologically thickened pericardium (>4 mm) was seen in 72 percent and pericardial calcification in 25 percent [13]. A normal appearance of

nonvisualization of the pericardium does not rule out the diagnosis.

Other findings include dilatation of the inferior vena cava, deformed ventricular contours, and angulation of the ventricular septum. Nonvisualization of the posterolateral left ventricular wall on dynamic CT may indicate myocardial fibrosis or atrophy and be associated with a poor surgical outcome [29].

CT imaging may also be used to examine the effect of cardiac motion transmitted to the surrounding pulmonary parenchyma. Failure of the immediately adjacent pulmonary structures to pulsate during the cardiac cycle, in the presence of a regionally or globally thickening pericardium, is virtually diagnostic of constrictive physiology.

CT may assist in the preoperative planning of pericardiectomy in patients with a history of previous cardiothoracic surgery given its ability to identify critical vascular structures [30]. Additionally, in selected patients, CT offers the ability to:

- Assess the extent of lung injury in patients with previous radiation exposure
- Evaluate the location and extent of pericardial calcification
- Avoid the need for coronary angiography in those with low-to-intermediate coronary risk.

Magnetic resonance imaging — Gated cardiac magnetic resonance imaging (CMR) provides direct visualization of the normal pericardium, which is composed of fibrous tissue and has a low MRI signal intensity [31]. CMR is claimed by some to be the diagnostic procedure of choice for the detection of certain pericardial diseases, including constrictive pericarditis [32-34]. Characteristic features include increased pericardial thickening and dilatation of the inferior vena cava, an indirect sign of impaired RV diastolic filling [9]. (See "Clinical utility of cardiovascular magnetic resonance imaging".)

While computed tomography (CT) is superior to CMR in detecting calcification, CMR better differentiates small effusions from pericardial thickening. CMR also has the potential to resolve hemodynamic events such as septal bounce and to better identify pericardial inflammation and pericardial-myocardial adherence [30].

Hemodynamic evaluation — Invasive hemodynamic evaluation is occasionally needed for the diagnosis of constrictive pericarditis [2]. (See "Hemodynamics in constrictive pericarditis versus restrictive cardiomyopathy".)

The major findings include:

- Increased right atrial pressure.
- Prominent x and y descents of venous and atrial pressure tracings (figure 1). The y descent of diastolic filling is absent in tamponade. (See "Cardiac tamponade", section on 'Physical findings'.)
- Kussmaul's sign (the lack of an inspiratory decline or an inspiratory increase in central venous pressure). This classic sign became less important with the advent of Doppler studies.
- Increased RV end-diastolic pressure, usually to a level one-third or more of RV systolic pressure.
- "Square root" signs in the RV and LV diastolic pressure tracings (an early diastolic dip followed by a plateau of diastasis; the last stage of diastole just before contraction), often with an absent a wave [9]. This finding, also called dip and plateau, reflects rapid early diastolic filling of the ventricles, followed by lack of additional filling due to compression in mid and late diastole (figure 2).
- A greater inspiratory fall in pulmonary capillary wedge pressure compared to left ventricular diastolic pressure.
- Equalization of LV and RV diastolic plateau pressure tracings, with little separation with exercise,

since filling, and therefore diastolic pressure, in both ventricles is constrained by the inelastic pericardium [9]. In some patients, this finding is seen only during inspiration (figure 3).

- Discordance between RV and peak LV systolic pressures during inspiration, another sign of increased ventricular interdependence. During peak inspiration, an increase in RV pressure occurs when LV pressure is lowest [35]. These changes can be detected by both invasive hemodynamic monitoring and Doppler echocardiography.

In the series of 143 patients with surgically confirmed pericardial constriction cited above, 78 underwent cardiac catheterization [13]. The mean right atrial pressure was 21 mmHg. A dip and plateau pattern was seen in 77 percent, diastolic equalization of pressures in 81 percent, and respiratory variation in the RV-LV pressure relationship in 44 percent. Tachycardia, which abbreviates or abolishes diastasis, is the usual explanation for the absence of the plateau configuration of ventricular mid through late diastolic pressure. The dip but not the plateau persists.

Differential diagnosis — An important step in the differential diagnosis is the distinction between constrictive pericarditis (which is treated by pericardiectomy) and restrictive cardiomyopathy or cirrhosis with ascites [2]. (See "Hemodynamics in constrictive pericarditis versus restrictive cardiomyopathy".)

Patients with both constrictive pericarditis and restrictive cardiomyopathy have elevated left and right sided filling pressures, often of equal magnitude, and usually have normal systolic ventricular function. Findings that distinguish the two conditions include:

- The history may provide helpful clues, such as prior pericarditis, a systemic disease predisposing to pericardiopathy, or a cause of restrictive cardiomyopathy (eg, diabetes mellitus or amyloidosis).

- A pericardial knock favors constriction, but is difficult to distinguish from the third heart sound of heart failure (HF).

- Increased thickness or calcification of the pericardium favors the diagnosis of constriction, while thickening of the ventricular wall and septum, abnormal myocardial texture and, to a lesser extent, mitral or tricuspid regurgitation favor restrictive cardiomyopathy.

  Although increased pericardial thickness is a classic finding in constrictive pericarditis, it is not present in all patients. This was illustrated in the review cited above of 143 patients with surgically confirmed constriction [13]. Pericardial thickening was seen in only 37 percent by TTE and 72 percent by CT. On histopathologic examination of pericardiectomy specimens, 26 patients (18 percent) had normal pericardial thickness. Important causes of constriction in this series included chest surgery, mediastinal irradiation, prior infarction, and idiopathic pericarditis. Pericardiectomy was equally successful in those with and without increased pericardial thickness.

- Doppler studies are helpful, revealing no abnormal increase in respiratory variation in Doppler measured ventricular inflow velocities in patients with restrictive cardiomyopathy.

- Hepatic vein flow reversal is expiratory in constrictive pericarditis, but inspiratory in restrictive cardiomyopathy. Diastolic mitral regurgitation suggests restrictive cardiomyopathy.

- The early diastolic Doppler tissue velocity at the mitral annulus (E') can also be helpful in diagnosis. The transmitral E' is decreased in restrictive cardiomyopathy, due to an intrinsic decrease in myocardial contraction and relaxation, while the transmitral E' is increased in constrictive pericarditis, since the longitudinal movement of the myocardium is enhanced because of constricted radial motion (figure 2) [25].

A high E' velocity (>12 cm/sec) usually indicates constrictive pericarditis while a low E' velocity (<8 cm/sec) usually indicates restrictive myocardial disease. However, a large number of patients fall in between these numbers in whom diagnosis is still not clear; despite excellent specificity of E' for differentiating restrictive cardiomyopathy from constrictive pericarditis, its sensitivity is more modest.

In a study of 17 patients with constrictive pericarditis, 12 patients with restrictive cardiomyopathy, and 15 controls, measurement of systolic mitral annular velocity (S') and the time difference between the onset of transmitral flow and the onset of E' (T(E'-E)) was shown to increase sensitivity and provide incremental diagnostic discriminating information to E' [36].

- Right and left ventricular end-diastolic pressures (RVEDP and LVEDP) are equal or very nearly so in constrictive pericarditis, while LVEDP is usually higher in restrictive cardiomyopathy. However, in many cases of restrictive cardiomyopathy the plateau of diastolic pressure is equally elevated in the two ventricles as occurs in constrictive pericarditis. If the pressures are approximately equal, a fluid bolus, exercise, or pharmacologic maneuver should theoretically increase LVEDP above RVEDP in restrictive cardiomyopathy [37-39], but the data supporting this theory are not convincing. The sensitivity and specificity of such maneuvers are not known and they cannot be relied upon.

  The predictive accuracy of conventional catheterization criteria was 75 percent in 100 consecutive patients undergoing cardiac catheterization to differentiate constriction (n= 59) from restrictive cardiomyopathy (n=41). Use of a novel "systolic area index" ratio (right ventricular to left ventricular systolic pressure-time area during inspiration versus expiration) that estimated enhanced ventricular interdependence had a sensitivity of 97 percent and a predictive accuracy of 100 percent for the identification of patients with surgically documented constrictive pericarditis [40].

- Endomyocardial, or less commonly, pericardial biopsy may be helpful when hemodynamic and imaging studies fail to establish the diagnosis [41,42].

Patients with constrictive pericarditis presenting with ascites as the main manifestation are often thought to have cirrhosis, sometimes considered cryptogenic because the history does not reveal an obvious cause. The main clue to a pericardial or myocardial etiology is marked elevation in jugular venous pressure with characteristic changes in the morphology of the pressure pulse. Cirrhosis is characterized by normal or only slightly elevated jugular venous pressures in the absence of tense ascites; the jugular venous pressure falls rapidly in such patients with removal of some of the ascitic fluid [10]. (See "Clinical manifestations and diagnosis of edema in adults", section on 'Distribution of edema and central venous pressure'.)

Plasma BNP — Plasma concentrations of brain natriuretic peptide (BNP) are increased in patients with left ventricular dysfunction; as a result, plasma BNP is used in the evaluation of dyspnea and to assess the efficacy of therapy and estimate prognosis in patients with heart failure (HF). (See "Brain natriuretic peptide measurement in left ventricular dysfunction and other cardiac diseases".)

Among patients with restrictive cardiomyopathy, BNP is released in response to left ventricular dysfunction and wall stretch. However, wall stretch is limited in constrictive pericarditis by the thickened stiff pericardium. These physiologic differences suggest that the elevation in plasma BNP in constrictive pericarditis should be much less than in restrictive cardiomyopathy. (See "Hemodynamics in constrictive pericarditis versus restrictive cardiomyopathy", section on 'Plasma BNP'.)

- An initial report of six patients with constrictive pericarditis and five with restrictive cardiomyopathy diagnosed by hemodynamic assessment provided support for the value of plasma BNP [43]. Plasma BNP was markedly elevated in patients with restrictive cardiomyopathy

and just above normal in those with constrictive pericarditis (825 versus 128 pg/mL; normal ≤100 pg/mL).

- Similar results were noted in a series of 22 patients with surgically-confirmed constrictive pericarditis. Patients were divided into those with idiopathic (n = 11) and those with secondary constrictive pericarditis (n = 11, previous surgery, chest irradiation), and these groups were compared to 11 patients with restrictive cardiomyopathy. Plasma BNP levels were statistically higher in restrictive cardiomyopathy than in idiopathic, but not secondary constrictive pericarditis. However, all patients with BNP <150ng/l had constrictive pericarditis [44].

- In a subsequent study, although BNP was higher in patients with restrictive cardiomyopathy (n=15) than constrictive pericarditis (n=16), there was significant overlap, particularly in those with secondary constriction and when BNP was less than 400 pg/ml. Interestingly, early diastolic Doppler tissue velocity could more accurately separate the groups (area under the curve 0.97 and 0.76 using cutoff values of 6.6 cm/s for E' and 308 pg/ml for BNP) [45].

These intriguing observations need to be confirmed in a larger number of patients to determine the accuracy of BNP testing. However, because the physiologic rationale is strong, we recommend measurement of plasma BNP early in the evaluation of patients with suspected constrictive pericarditis.

Treatment and outcome — In a minority of patients with constrictive pericarditis constriction is transient or reversible, but in most patients the disorder is permanent unless surgically treated.

Transient constrictive pericarditis — A subset of patients with constrictive pericarditis undergo spontaneous resolution of constriction or respond to medical therapy [46,47]. In a review from the Mayo Clinic of 212 patients with echocardiographic findings of constrictive pericarditis, 36 (17 percent) had follow-up studies showing resolution at an interval ranging from two months to two years [47]. The most common cause of transient constrictive pericarditis was caused by pericardial inflammation after pericardiotomy in nine cases (25 percent); infection (viral, bacterial, or tuberculous), idiopathic, collagen vascular disease, trauma, and malignancy accounted for the remaining cases. Treatment had included nonsteroidal antiinflammatory drugs, a steroid, antibiotics, chemotherapy, and angiotensin converting enzyme inhibitors plus diuretics. Five patients had resolution of constriction without any specific therapy.

The clinical course in these patients implies the presence of acute inflammatory pericarditis with constriction due to inflammation that resolved once the inflammatory process was treated. Thus, in the absence of evidence that the condition is chronic (eg, cachexia, atrial fibrillation, hepatic dysfunction, or pericardial calcification), patients with newly diagnosed constrictive pericarditis who are hemodynamically stable may be given a trial of conservative management for two to three months before pericardiectomy is recommended.

Such patients require frequent observation for signs that constriction has become chronically established. Worsening systemic congestion, as manifested by increased symptoms, otherwise unexplained weight gain, and/or a new or increased pleural effusion or ascites, is an indication for pericardiectomy. (See "Evaluation and management of acute pericarditis" and "Etiology of pericardial disease", section on 'Acute pericarditis'.)

Chronic constrictive pericarditis — Surgery in experienced centers is the accepted standard of treatment in patients with chronic constrictive pericarditis who have persistent and prominent symptoms. However, as will be described below, surgery should be considered cautiously in patients with either mild or very advanced disease and in those with radiation-induced constriction, myocardial dysfunction, or significant renal dysfunction. As an example, patients with a history of prior cardiac surgery should be evaluated for myocardial dysfunction, a common finding in this population, to help determine whether cardiomyopathy or constrictive pericarditis is the primary cause of symptoms.

Preoperative diuretics should be used sparingly with the goal of reducing, not eliminating elevated venous pressure, ascites, and edema.

Surgical removal of the pericardium has a significant operative mortality. In the Stanford series of patients who underwent surgery between 1970 and 1985, the operative mortality was 12 percent [3]. A lower mortality rate of about 6 percent has been noted in patients who underwent pericardiectomy between 1977 and 2000 at the Mayo Clinic, the Cleveland Clinic, the Johns Hopkins Hospital, or the All India Institute [4,5,48,49].

The pericardiectomy must be as complete as is technically feasible [5,13,49]. This should be performed by surgeons with considerable experience with this procedure, which may require referral to a center with a special interest in pericardial disease.

Most patients have relief of symptoms after pericardiectomy. In the Mayo Clinic experience, for example, functional class improved markedly among the 90 long-term survivors, and 83 percent were free of clinical symptoms [4]. Another study evaluated 58 patients with constrictive pericarditis who underwent pericardiectomy and had at least one follow-up Doppler echocardiographic study [50]. The following findings were noted:

- During the first three months after surgery, diastolic function was normal in 41 percent of patients, while a restrictive or constrictive filling pattern was present in 41 and 19 percent, respectively.
- A repeat echocardiographic study was performed in 35 patients after a mean of 21 months; diastolic function remained abnormal (restrictive or less often constrictive) in 15 (43 percent). Among these 15 patients, 11 were in NYHA class II and four in NYHA class III.
- At late follow-up at a mean of 4.2 years, 14 patients (24 percent) remained symptomatic, 12 of whom had a persistently abnormal diastolic filling pattern. The poorest outcome was in patients with radiation-induced disease. The next poorest outcome was in patients who had previously undergone complicated cardiac surgery.

Patients with mild constriction, advanced disease, or mixed constrictive-restrictive disease may not benefit from pericardiectomy:

- Constrictive pericarditis in patients whose only abnormality is a mild to moderate increase in central venous pressure with little or no edema can be followed, provided their circumstances make this a reliable option.
- Patients with "end-stage" constrictive pericarditis derive little or no benefit from pericardiectomy and the operative risk is inordinately high. Manifestations of end-stage disease include cachexia, atrial fibrillation, a low cardiac output (cardiac index ≤1.2 L/m2 per min) at rest, and hypoalbuminemia due to protein losing enteropathy and/or impaired hepatic function due to chronic congestion or cardiogenic cirrhosis.
- Symptoms may persist after successful pericardiectomy in patients with mixed constrictive-restrictive disease (eg, radiation disease) because of abnormalities in intrinsic myocardial compliance. In these patients it is important to assess the extent of myocardial damage. Tissue Doppler is very helpful for this, and endomyocardial biopsy is useful in some cases.

Long-term survival after pericardiectomy is inferior to that of an age- and sex-matched population. In the Mayo Clinic series, the 5 and 10 year survival rates were 78 and 57 percent (figure 4) [4]. Independent adverse predictors of long-term outcome included older age and worse NYHA class. In the Cleveland Clinic series, independent adverse predictors included age, renal dysfunction, pulmonary hypertension, left ventricular dysfunction, and hyponatremia [5].

The etiology of the pericardial disease is also an important determinant of survival [4,5]. Prior ionizing radiation, because it induces cardiomyopathy as well as pericardial disease, is associated with a poor long-term outcome. This was illustrated in the Cleveland Clinic series; the seven year survival rates after surgery for patients with idiopathic, postsurgical, and radiation-induced constrictive pericarditis were 88, 66, and 27 percent, respectively [5].

Preoperative indices of contraction and relaxation also predict postoperative prognosis. An observational study of 40 patients undergoing preoperative cardiac catheterization reported that patients with both an abnormal + LV dP/dt (<1200 mmHg/s) and an abnormal time constant of LV isovolumic relaxation (tau >50 ms) required more frequent postoperative inotropic support, had higher immediate postoperative mortality, and significantly lower long-term survival (median follow-up 2.4 years) than patients with either two normal values or one abnormality [51].

OCCULT CONSTRICTIVE PERICARDITIS — A report in 1977 described 19 patients with a syndrome called occult constrictive pericardial disease [8]. The symptom complex of this proposed syndrome was comprised of chest pain, dyspnea, and fatigue, which may appear nonspecific. However, 12 of the 19 had a history of prior acute pericarditis (which was recurrent in five). In addition, two patients had pericardial calcification and, in 16, the ECG showed nonspecific repolarization changes. A reasonable cause for pericardial disease was present in 10.

The authors proposed that very mild constrictive pericarditis can cause these symptoms in the absence of abnormal physical or hemodynamic findings when the patient is evaluated in the basal state. To test this hypothesis, they measured hemodynamics invasively before and after infusing a liter or warm saline over a period of six to eight minutes to determine if occult constriction would then become overt. Six patients known not to have heart disease and 12 patients with myocardial disease served as controls.

The results can be summarized as follows:

- Saline infusion caused an elevation and equalization of ventricular filling pressures, and development of pressure waveforms in diastole characteristic of constriction (figure 5) in the patients with occult constriction.
- Ventricular filling pressures and diastolic waveform were unaltered in the subjects free from heart disease.
- The patients with myocardial disease developed elevated ventricular filling pressures, but unequally on the two sides.

Eleven of the symptomatic patients underwent pericardiectomy with dramatic improvement. All 11 cases had mild gross or histologic evidence of pericardial disease. The fluid challenge was repeated postoperatively in five of the patients with negative results.

Based upon these findings, the authors recommended pericardiectomy for disabling symptoms. However, we have strong reservations about this study, even though it was conducted using high fidelity pressure tracings in a laboratory well known for high quality hemodynamic studies. It is unclear why or how such mild constriction could cause disabling symptoms, and why chest pain was a feature. Furthermore, the dramatic relief by pericardiectomy is unexplained in these patients with low normal venous pressures that were modestly elevated by a rapid large fluid challenge, and in whom the cardiac output was normal at rest and was not changed by the infusion.

Our recommendation is that the frequently performed saline infusion test is seldom required and, if performed, the results should seldom provide the reason for pericardiectomy. Patients suspected of having occult pericardial constrictive disease should undergo cardiac catheterization, including measurement of oxygen consumption during progressive bicycle exercise. A study of this nature would

document exertional dyspnea and fatigue, and may help clarify the responsible mechanism. Rather than infusing fluid, diuretic therapy can be suspended for several days before cardiac catheterization.

EFFUSIVE CONSTRICTIVE PERICARDITIS — The pericardial cavity is typically obliterated in patients with constrictive pericarditis. Thus, even the normal amount of pericardial fluid is absent. However, pericardial effusion may be present in some cases. In this setting, the scarred pericardium not only constricts the cardiac volume but can also put pericardial fluid under increased pressure, leading to signs suggestive of cardiac tamponade. This combination is called effusive constrictive pericarditis [1,3,52-54].

Incidence and etiology — Effusive constrictive pericarditis appears to be relatively uncommon, although there are only limited published data. In one series from Stanford of 95 patients undergoing surgery for constrictive pericarditis, 23 (24 percent) were diagnosed with effusive constrictive disease [3]. In another report from Spain of 190 patients with cardiac tamponade who underwent pericardiocentesis and cardiac catheterization, the disorder was diagnosed in 15 (8 percent) [53].

Most cases of effusive constrictive pericarditis are idiopathic, reflecting the frequency of idiopathic pericardial disease in general [3,53]. Other reported causes include radiation, neoplasia, chemotherapy, infection, and postsurgical pericardial disease. Tuberculosis is a frequent cause of effusive constrictive disease in regions where the disease is common [55]. (See "Tuberculous pericarditis".)

Clinical features — Patients with effusive constrictive pericarditis usually present with clinical features of pericardial effusion or constrictive pericarditis or both. In the series of 15 patients cited above, the following clinical observations were made [53]:

- Symptoms were usually present for less than three months (range four days to 26 months)
- All patients had jugular venous distention and hepatomegaly
- Eight had pericardial chest pain, fever, and a pericardial rub
- Pulsus paradoxus was seen in ten
- All were in normal sinus rhythm
- None had pericardial calcification

A number of clinical clues suggest that a patient with manifestations of constrictive pericarditis may actually have effusive constrictive pericarditis:

- Pulsus paradoxus is often present; as noted above, this finding is uncommon in classical constrictive pericarditis because the inspiratory decline in intrathoracic pressure is not transmitted to the right heart chambers
- A pericardial knock is absent
- The Y descent is less marked than expected
- Kussmaul's sign is frequently absent

Diagnosis — The diagnosis of effusive constrictive pericarditis often becomes apparent during pericardiocentesis in patients initially considered to have uncomplicated cardiac tamponade. Unexpected persistence of the v wave of right atrial pressure is a clue to the possibility of effusive constrictive pericarditis that may be present before pericardiocentesis. After pericardiocentesis, despite lowering of the pericardial pressure to near zero, persistence of elevated right atrial pressure suggests the presence of effusive constrictive disease (figure 6) [53,54]. The diagnosis has been defined by failure of the right atrial pressure to fall by 50 percent or to a level below 10 mmHg after pericardiocentesis [53]. The right atrial pressure remains abnormal due to constriction by the visceral pericardium. For these reasons, we recommend that right heart pressures and systemic arterial blood pressure be monitored during elective pericardiocentesis [56].

A persistently elevated right atrial pressure after pericardiocentesis may also be due to right heart failure or tricuspid regurgitation. Thus, appropriate studies should be performed to exclude these

disorders before making the diagnosis of effusive constrictive pericarditis. Other features of the right atrial pressure tracing after pericardiocentesis that may suggest effusive constrictive disease include the development of a marked, rapid y descent and the lack of an inspiratory decline in right atrial pressure.

Noninvasive imaging is not useful in the diagnosis of effusive constrictive pericarditis [54]. The epicardial layer of pericardium, which is responsible for the constrictive component of this process, is not typically thickened to a degree that is detectable on imaging studies.

Treatment and course — Pericardiocentesis alone may produce at least temporary relief of symptoms in patients with effusive constrictive pericarditis, although it does not correct the underlying condition. In a series of 15 patients, marked improvement after pericardiocentesis was noted in five, mild improvement in eight, and no benefit in two [53].

The clinical course after pericardiocentesis depends upon the cause of the pericardial disease and the patient's general clinical condition. In the report of 15 patients [53]:

- Three patients with idiopathic pericarditis had spontaneous resolution, while one with malignant pericarditis responded to chest radiation.
- Seven patients with persistent disease underwent wide anterior pericardiectomy between 13 days and four months after pericardiocentesis.
- Four patients had progressive disease but were too ill to undergo surgery.

In effusive constrictive pericarditis, it is the visceral layer of pericardium, not the parietal layer, that constricts the heart. Thus, if surgery is required, it is visceral pericardiectomy that must be performed [54]. However, the visceral component of the pericardiectomy is often difficult, requiring sharp dissection of many small fragments until an improvement in ventricular motion is observed [53]. Thus, pericardiectomy for effusive constrictive pericarditis should be performed only at centers with experience in pericardiectomy for constrictive pericarditis.

INFORMATION FOR PATIENTS — Educational materials on this topic are available for patients. (See "Patient information: Pericarditis".) We encourage you to print or e-mail this topic, or to refer patients to our public web site www.uptodate.com/patients, which includes this and other topics.

SUMMARY

- The critical pathophysiologic features of constrictive pericarditis, which are responsible for the physical exam, hemodynamic, and imaging findings, are:

  Greatly enhanced ventricular interaction or interdependence
  The dissociation of intracardiac and intrathoracic pressures

  (See 'Physiology' above.)

- Constrictive pericarditis can occur after virtually any pericardial disease process, but most often follows viral or idiopathic pericarditis and cardiac surgery. (See 'Etiology' above.)
- Characteristic hemodynamic changes that differentiate constrictive pericarditis from restrictive cardiomyopathy are detected at catheterization and during Doppler ultrasound interrogation. Imaging with echocardiography, magnetic resonance, and computed tomography often provide the critical anatomic information needed for diagnosis. Uncommonly, endomyocardial or pericardial biopsy is needed to establish the diagnosis. (See 'Clinical evaluation' above.)
- The diagnosis of effusive constrictive pericarditis most often becomes apparent by the

unexpected persistence of an elevated right atrial pressure following pericardiocentesis in patients initially considered to have uncomplicated cardiac tamponade. (See 'Effusive constrictive pericarditis' above.)

- Surgery in experienced centers is the accepted standard treatment in patients with chronic constrictive pericarditis who have persistent and prominent symptoms, but a subset of patients with constrictive pericarditis undergo spontaneous resolution of constriction or respond to medical therapy. (See 'Treatment and outcome' above.)

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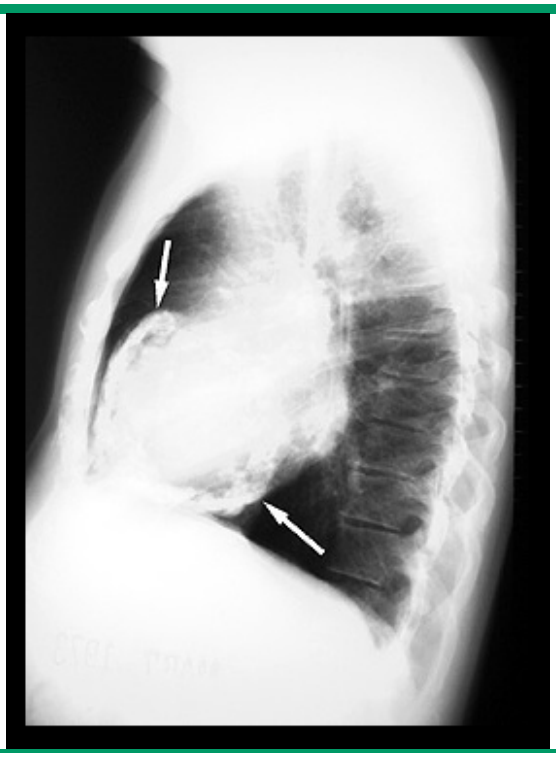
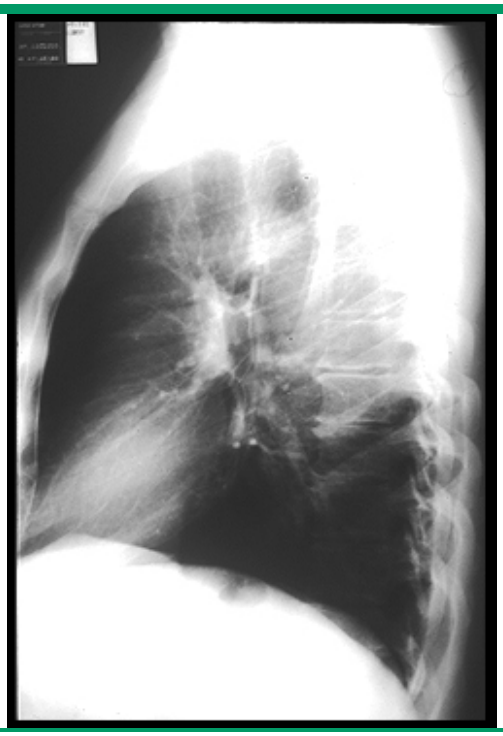
GRAPHICS

Pericardial calcium



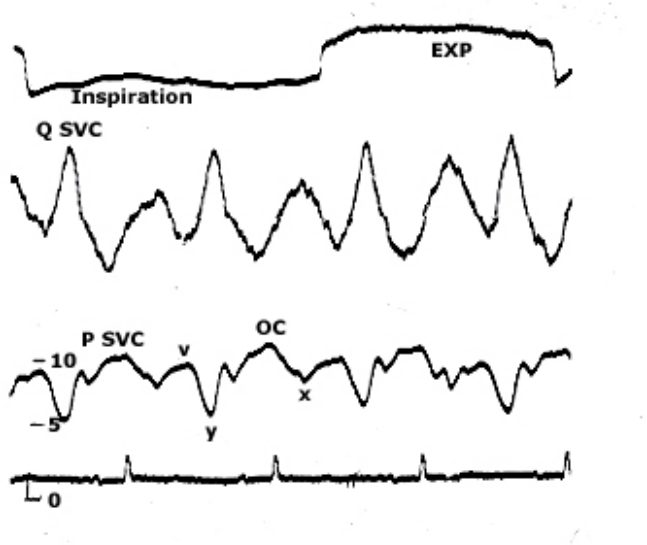
This lateral chest radiograph demonstrates coarse calcification involving the entire pericardium (arrows) in this patient who has a history of prior hemopericardium. *Photo courtesy of Jonathan Kruskal, MD.*

Normal lateral chest radiograph



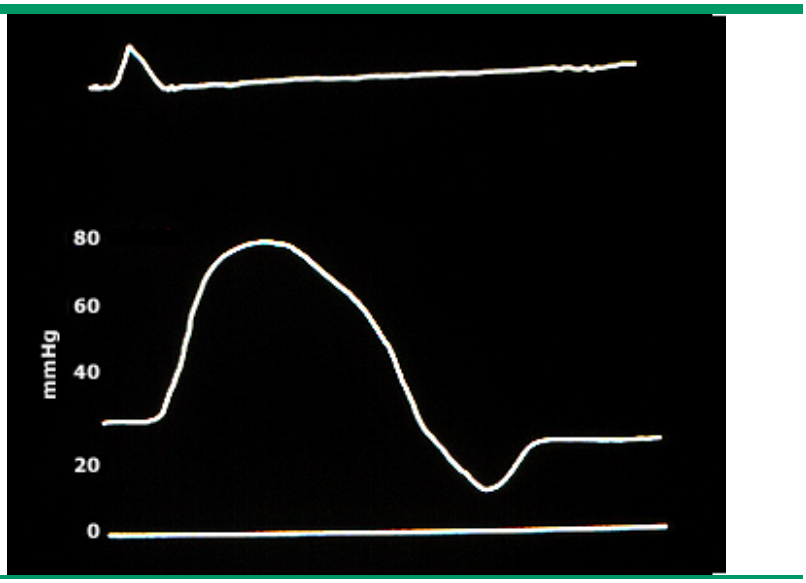
Courtesy of Steven Weinberger, MD.

Exaggerated y descent in constrictive pericarditis



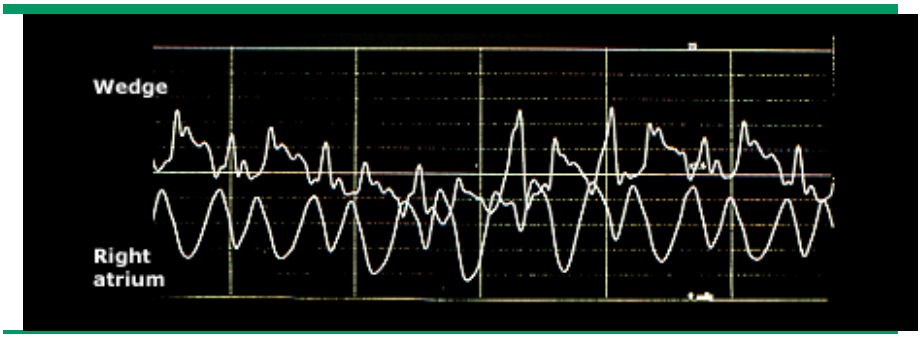
Tracing from a patient with mild constrictive pericarditis shows prominent y descent which becomes deeper during inspiration. PSVC and QSVC represent the pressure and velocity recorded from the superior vena cava. *Courtesy of Ralph Shabetai, MD.*

True dip and plateau in constrictive pericarditis



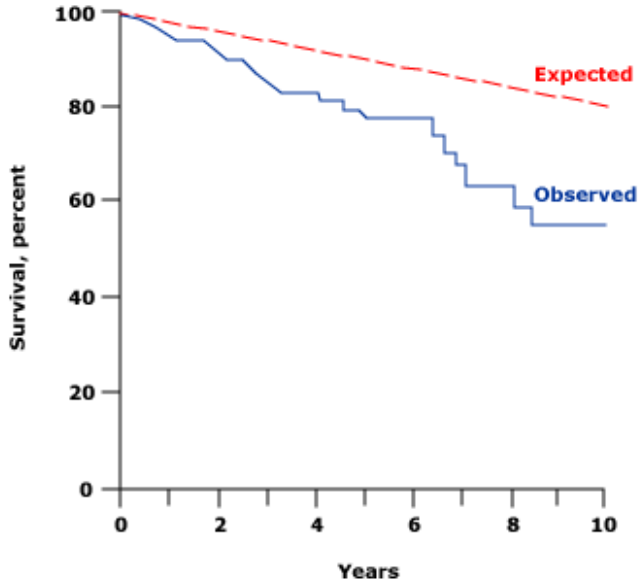
High fidelity left ventricular pressure tracing from a patient with severe calcific constrictive pericarditis showing dip and plateau pattern. *Courtesy of Ralph Shabetai, MD.*

Equalization of pressures in severe constrictive pericarditis



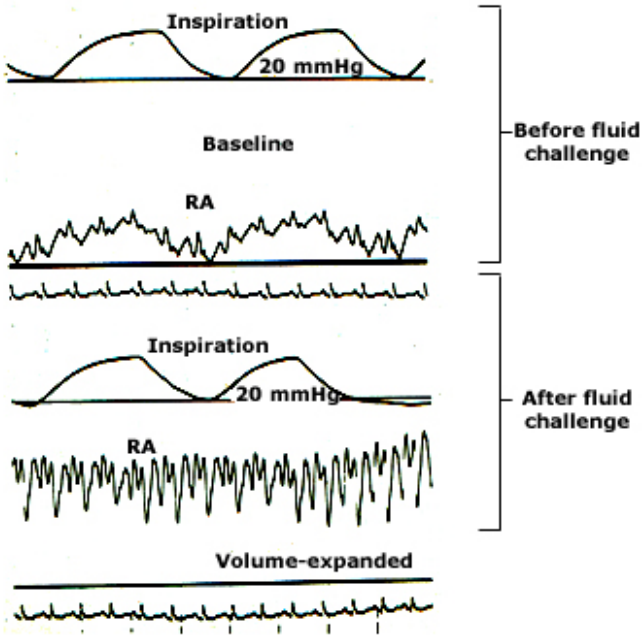
Tracing from a patient with severe constrictive pericarditis shows that the wedge and right atrial pressures are equal during inspiration, but that the wedge pressure is higher during expiration. *Courtesy of Ralph Shabetai, MD.*

Late survival after pericardiectomy for constrictive pericarditis is reduced



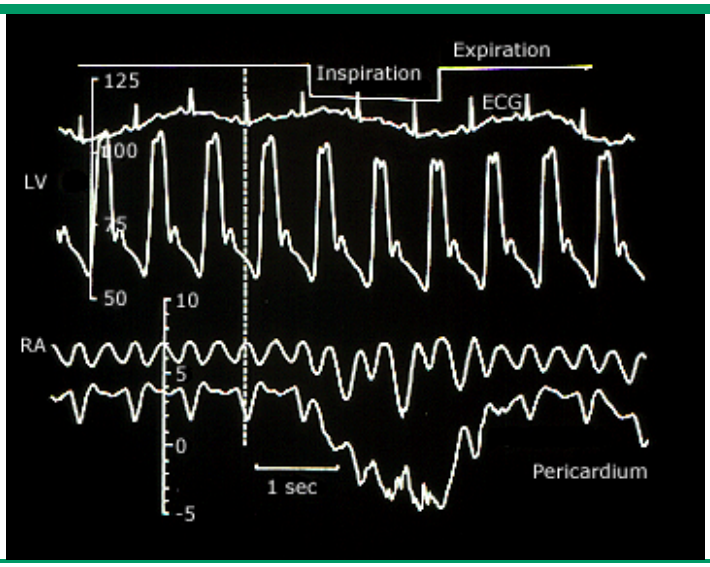
Among 135 patients who underwent pericardiectomy for constrictive pericarditis, the long-term survival was poorer when compared with that expected in an age- and sex-matched US population (57 versus 81 percent, $p < 0.001$).
Data from Ling, LH, Oh, JK, Schaff, HV, et al. Circulation 1999; 100:1380.

Saline loading unmasks occult constrictive pericarditis



Right atrial pressure tracings before (top panel) and after (bottom panel) saline loading in a patient with occult constrictive pericarditis. The previously normal right atrial pressure developed the characteristics of constrictive pericarditis (with equalization of pressures after fluid challenge. *Redrawn from Bush, CA, Stang, JM, Wooley, CF, Kilman, JW, Circulation 1977; 56:924.*

Effusive constrictive pericarditis after pericardiocentesis



Hemodynamic tracing in a patient with effusive constrictive pericarditis after pericardial pressure has returned to normal after pericardiocentesis. Right atrial pressure remains elevated and has the features of constrictive pericarditis.
Courtesy of Ralph Shabetai, MD.