# Constrictive Pericarditis After Coronary Artery Bypass

Ahmad Halawa, MD, Said Iskandar, MD, Israel Garcia, MD, FACC

East Tennessee State University, James H. Quillen College of Medicine, Johnson City, TN

*A 67-year-old male patient received a coronary artery bypass graft. Less than 2 months afterward, he presented with recurrent exacerbations of congestive heart failure. His response to a standard treatment regimen for heart failure was partly successful, but a few days after discharge he was readmitted for worsening dyspnea and edema. Doppler echocardiography suggested the hemodynamics of constrictive pericarditis. Magnetic resonance imaging showed thickened pericardium with exudates in the pericardial space. Cardiac catheterization confirmed the diagnosis, showing equalization of diastolic pressures of the left and right ventricles. The patient underwent subtotal pericardiectomy with resolution of the pericardial disease, but he died from respiratory insufficiency.*

[Rev Cardiovasc Med. *2006;7(4):238-243*]





**Key words:** Pericarditis • Coronary artery bypass graft • Echocardiogram • Thickened pericardium • Pericardiectomy

A 67-year-old male patient with known diabetes mellitus, dyslipidemia, peripheral vascular disease, and prior transient ischemic attacks was hospitalized for treatment of unstable angina and pulmonary edema. Left and right heart catheterization revealed severe multivessel disease. A left ventriculogram showed inferobasilar segmental akinesis with a left ventricular ejection fraction (LVEF) of 45%. Mild pulmonary hypertension with a mean pulmonary artery pressure of 29 mm Hg was also noted.

The patient had a history of moderate obstructive sleep apnea, for which he used a nasal continuous positive airway pressure mask at night. Pulmonary function tests performed before his coronary artery bypass graft (CABG) surgery showed mild restrictive lung disease. He did not have known tuberculosis exposure. His purified protein derivative status was unknown.

Two vessel coronary artery bypasses were performed without complication. Cold saline was used for washing the heart intraoperatively. The pericardial margins were approximated anteriorly and superiorly but left open inferiorly. The patient did well postsurgery and was discharged after 4 days.

Six weeks after the CABG procedure, the patient was admitted to the hospital with dull chest tightness and exertional shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, generalized weakness, and decreasing urine output. Pertinent examination findings included sinus tachycardia at 130/min and blood pressure of 150/90 mm Hg. Jugular venous pressure was elevated to 18 cm. He also had 2+ bilateral carotid bruits. Bibasal crackles were heard on chest examination. Cardiac auscultation showed normal S1 and S2 with no murmurs, gallops, rubs, or knocks. His abdomen was globular, but there was no apparent organomegaly. He had 2+ bipedal pitting edema up to his thighs, with scrotal swelling.

A chest x-ray showed cardiomegaly. An electrocardiogram showed sinus tachycardia with biatrial enlargement, nonspecific ST-T wave changes, old T wave inversions anterolaterally, and old Q waves inferiorly. A pharmacologic nuclear stress test showed normal homogeneous uptake by the left ventricle (LV) except for an old inferior wall defect. Two-dimensional echocardiography showed normal LV dimensions, with an LVEF of 50% and mild left atrial enlargement with a poor left atrial emptying index, suggesting LV diastolic dysfunction. Mild pulmonary hypertension was also present. A new finding of anterior echo-free space was noted, which had not appeared during a 2-dimensional echocardiography performed in 1998.

The patient's clinical symptomatology was thought to be secondary to LV diastolic dysfunction. The doses of his beta-blocker medication and diuretic were increased, and his symptoms resolved. He was discharged.

The patient was readmitted 5 more times for recurrence of his symptoms. On one of these occasions, because he was thought to have postpericardiotomy syndrome, he was given nonsteroidal anti-inflammatory drugs (NSAIDs). During his fourth and fifth admissions, he was hypotensive and had deteriorating renal function, and he was placed in the intensive care unit. Pulmonary artery catheterization revealed elevated right atrial, right ventricular, pulmonary arterial, and capillary wedge pressures, with means between 20 mm Hg and 30 mm Hg. The possibility of pulmonary embolism was considered but was essentially ruled out by a low-probability ventilation-perfusion scan.

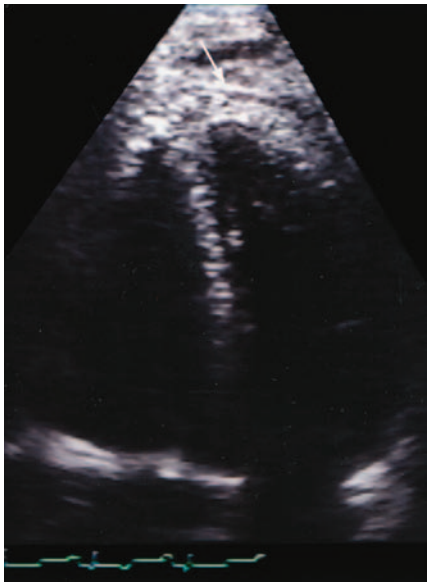


**Figure 1.** *Two-dimensional echocardiogram of the left ventricle in the apical 4-chamber view, showing thickened pericardium (arrow) with a small effusion.*
www.medreviews.com

Repeat echocardiography showed an LVEF of 60%, with evidence of grade 3 LV diastolic dysfunction, paradoxical interventricular septal

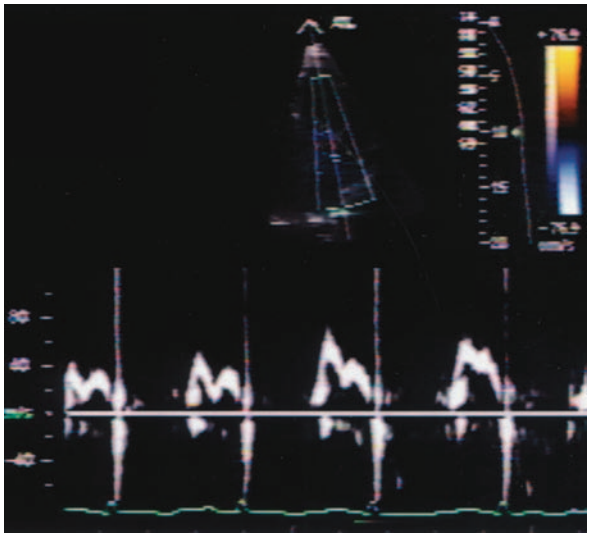


**Figure 2.** *Doppler echo across the mitral valve, showing reduction in the peak E velocity ≥ 25% during inspiration.*
www.medreviews.com

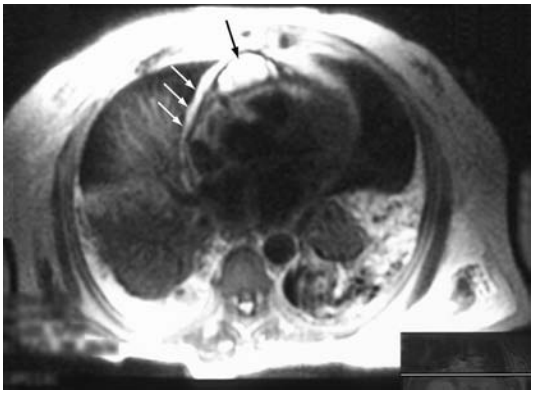


**Figure 3.** *Cardiac magnetic resonance image showing thickened pericardium (white arrows) and a localized fibrosis with fluid accumulation anteriorly (black arrow).*
www.medreviews.com

movement, and right ventricular (RV) enlargement. The pericardium was thick (Figure 1). Doppler also showed significant respiratory variation in the mitral valve inflow and LV outflow (Figure 2). A chest computed tomography (CT) scan showed a loculated-appearing pericardial process, raising the possibility of restrictive pericardial disease.

Constrictive pericarditis was suspected because of the patient's worsening right-side pressure symptoms and signs and his lack of response to continued diuresis. Cardiac magnetic resonance imaging (MRI) showed prominent paracardial tissue/fluid collection anterior to the heart that had a maximum thickness of 24 mm, but there was no definite evidence of constriction (Figure 3).

Left and right heart catheterization showed patent grafts with normal LV systolic function. It also revealed equalization of all diastolic pressures in both the left- and right-side chambers. The ventricular diastolic waveforms had the characteristic dip and plateau pattern. The right atrial waveform showed prominent *y* descents (Figure 4).

Repeat PFTs were obtained before pericardiectomy. They showed severe restrictive lung disease.

The patient underwent reentry sternotomy. The heart was encased in a thick, fibrous shell of visceral pericardium, which in most places was densely adherent to the epicardium. The parietal pericardium was also thickened and extremely dense in focal areas, especially anteriorly, affecting the anterior aspect of the RV extending to the right atrium. A total pericardiectomy was performed. Gross and histologic examination of the pericardium showed marked fibrosis and chronic inflammation with organizing exudates (Figures 5 and 6).

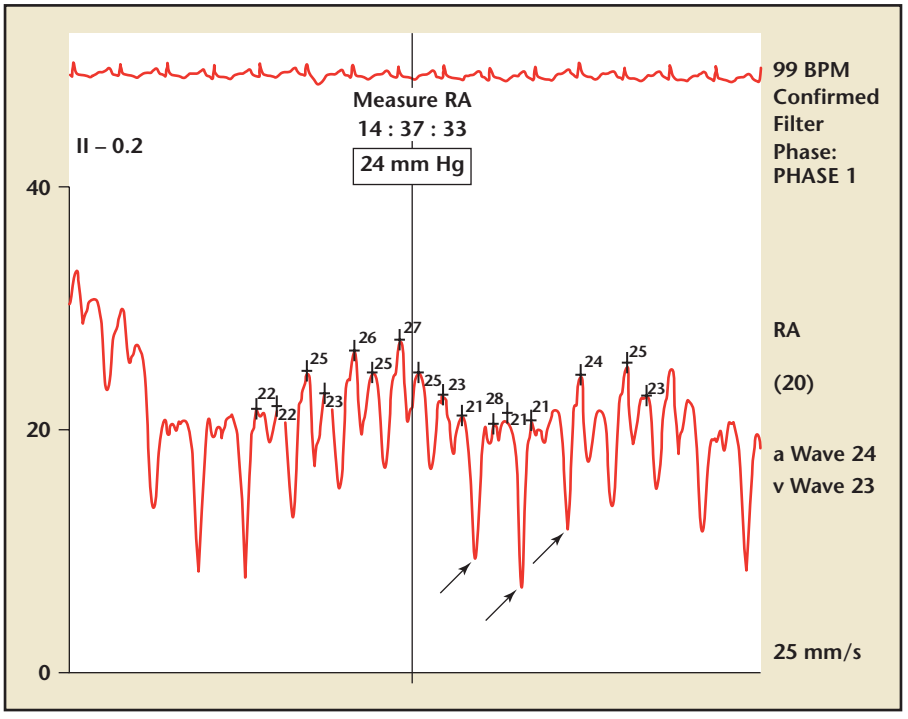
Intraoperatively, the patient received multiple blood products because of excessive bleeding, for which he was seen by a hematologist. The hematologist thought that disseminated intravascular coagulation might be starting. The patient underwent a reentry sternotomy with evacuation of mediastinal hematoma. The bleeding complication eventually resolved.

The patient's Swan-Ganz parameters postsurgery showed improvement in cardiac pressures, although mildly elevated mean pulmonary artery pressures remained. Renal function normalized. He remained ventilator dependent postoperatively and subsequently developed methicillin-resistant *Staphylococcus aureus* and *Pseudomonas* pneumonia, and he eventually died 51 days after the first pericardiectomy.

## Discussion

Constrictive pericarditis is a pericardial disease related to the compression

**Figure 4.** *Right atrial (RA) waveforms on right heart catheterization, showing prominent y-wave descents (arrows). BPM, beats per minute.* www.medreviews.com



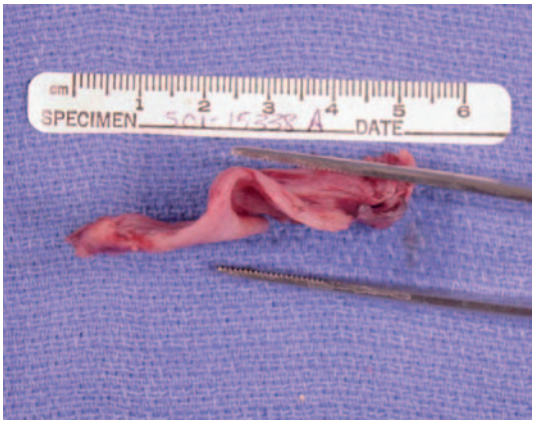



**Figure 5.** *Gross histology of an excised part of the pericardial sac, showing thickening with excessive fibrosis.* www.medreviews.com

and restriction of the heart secondary to a thickened pericardium—either by the visceral layer or by an adherent parietal layer—which causes hemodynamic abnormalities that manifest as equalization and elevation of the diastolic pressure of the heart chambers. This condition resolves after successful and adequate pericardiectomy.

In 1980, Hancock[1] discussed the difference between subacute effusive constrictive pericarditis and rigid, chronic constrictive pericarditis. The subacute effusive form includes the presence of a pericardial effusion with visceral pericardial constriction of the heart; patients often present with pulsus paradoxus and a prominent systolic dip (*x* descent) of the jugular venous pressure. Rigid, chronic constrictive pericarditis results from progression of the subacute effusive form, with eventual formation of a rigid shell around the heart. Affected patients have Kussmaul's sign, prominent diastolic dip of the neck veins, and a classic dip and plateau pattern of RV and LV diastolic pressures.

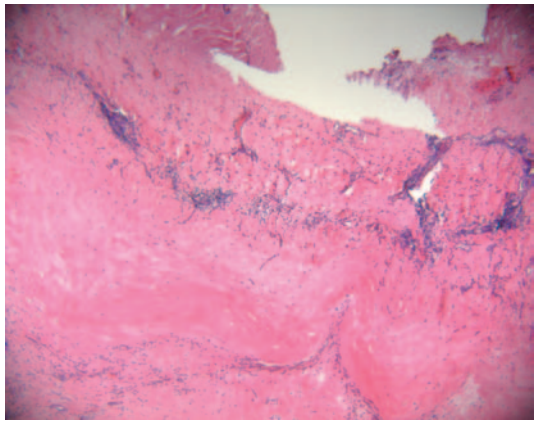
Constrictive pericarditis was first reported in 1972.[2] It occurs in 0.2% to 0.3% of cases after cardiac surgery.[2] It may manifest as early as 2 weeks[3] or as late as 21 years[4] after surgery. As noted above, the patient described here presented with symptoms about 6 weeks after his original heart surgery.

Tuberculosis was once the classic etiology of chronic calcific constrictive pericarditis. Currently, most cases are idiopathic (or patients have a history of previous viral infection). Some cases are related to inflammatory connective tissue disease, chest irradiation, bacterial and fungal infection, chronic renal failure, trauma, and metastatic neoplastic conditions. Postpericardiotomy syndrome occurs in as many as 30% of patients after cardiac surgery; if not adequately treated, this condition may eventually cause constriction. Patients typically present with fever and serositis a few weeks after the original surgery. Some patients may have subclinical symptoms, and these cases may be missed. Whether the patient described here had this postoperative complication is unclear. He did not have any flu-like symptoms before or during his original presentation after the open heart surgery. Nevertheless, he was given NSAIDs for possible postpericardiotomy syndrome, as recommended in the literature.

Although treatment with NSAIDs or steroids, or both, has proved helpful, this approach does not necessarily prevent the formation of cardiac constriction.[5] More recently, clustering of recurrent pericarditis with effusion and constriction was reported in one family, suggesting autosomal dominant inheritance with incomplete penetrance.[6] The patient in our report was the only member of his family known to have this condition.

The mechanism of constrictive pericarditis requires both the presence of blood in the pericardial sac and associated injury to the serosal lining.[7] These 2 events set the stage for inflammation and the resulting fibrosis of the pericardial sac. An earlier study alluded to an increased incidence of this complication when the pericardium is left open after open heart surgery because blood usually originates from the extracardiac surface and eventually passes to the pericardium.[8] Cunningham and

**Figure 6.** *Pathology of excised pericardium, showing lymphocytic infiltration with fibrosis suggestive of inflammation.* www.medreviews.com



colleagues[9] suggested that closure of the pericardium decreased the incidence of intrapericardial blood accumulation. Whether leaving the pericardium open decreases postoperative constrictive pericarditis was not demonstrated in subsequent studies.[3,10-15] Although another study suggested a better outcome if the pericardium was closed,[5] it remains unclear whether pericardial closure might change the incidence of constrictive pericarditis.

Serosal injury can be caused by antibiotics, povidone-iodine, or iced saline wash. The patient in our report received iced saline irrigation during his operation. Mere air drying has also been implicated,[7] probably because it causes the loss of mesothelial cells that possess fibrinolytic activity. In 1929, Beck[16] suggested that the irritant effect of Dakin's solution in the pericardium caused constrictive pericarditis. Marsa and colleagues[17] confirmed the association of this condition with postoperative use of povidone-iodine as a pericardial irrigant. Likewise, there has been a report of fibroblastic proliferation of the serosal surface related to iced saline wash, especially if the wash is cooled to 0.5°C.[18] A Japanese study suggested that the presence of normal LV systolic function, use of coumadin anticoagulation, and early postoperative pericardial effusion in patients undergoing CABG increase the risk of this dreaded complication.[19]

Affected patients typically present with dyspnea on exertion, fatigue, and chest pain, as did the patient described in this report. The physical examination is notable for recurrent lower-extremity edema, jugular venous distension, hepatomegaly, S3 on cardiac auscultation, ascites, friction rub, pulsus paradoxus, and Kussmaul's sign, in that order.[5] These findings seem to be refractory to usual diuresis.

Twelve-lead electrocardiograms may show nonspecific ST-T changes, low voltage, and, typically, in-sinus tachycardia or atrial dysrhythmia. Chest x-ray traditionally shows pericardial calcification and thickening, although, as noted above, this finding is less common today because of the decreased incidence of tuberculosis. Chest CT may show pericardial thickening, pericardial calcifications, loculated effusions, and fibrotic changes.[15] MRI allows better definition of the pericardium and may even delineate whether the pericardial fluid is serous, exudative, or hemorrhagic. With advances in cardiac ultrasonography, 2-dimensional echocardiography, and, particularly, traditional spectral Doppler, with the addition of tissue Doppler, constrictive pericarditis can now be differentiated from restrictive cardiomyopathy and cardiac tamponade by the bedside. Two-dimensional images may show pericardial thickening or pericardial effusion. More importantly, spectral Doppler shows restrictive physiology, with $\geq$ 25% respiratory variation in mitral inflow E velocity, and increased diastolic flow-reversal velocity in the hepatic vein with expiration. Lung-related conditions may also be differentiated from constrictive pericarditis by systolic flow changes in the superior vena cava. Finally, the advent of tissue Doppler technology affords further differentiation of restrictive cardiomyopathy from the pericardial disease. In constrictive pericarditis, early and late diastolic tissue Doppler patterns will appear normal.

Cardiac catheterization remains the gold standard. It reveals the elevation and equalization of diastolic pressure in the cardiac chambers and the classic dip and plateau pattern of the ventricular waveform. This pattern reflects the change in early diastolic filling, which is unimpeded until the rigid shell abruptly terminates the mid- to late diastolic filling—a development appreciated clinically as a pericardial knock. Because patients with this condition are usually volume depleted, intravenous infusion during the cardiac catheterization has been advocated in order to diagnose occult constrictive pericarditis.[20] Preoperative cardiac catheterization can also confirm the patency of grafts, as these tend to be occluded in patients who develop postoperative cardiac constriction.[21] Pulsus paradoxus may also be demonstrated by the observation of aortic pressure, depending on whether an effusive component is present.

Patients who develop early post-CABG pericardial effusion should be considered at high risk for constrictive pericarditis. A study of steroid versus nonsteroid and diuretic treatment for this condition showed steroids to be effective in reducing the pericardial effusion in the early postoperative period, but it did not demonstrate any improvement in pericardial fibrosis.[19] Drainage of the effusion is mandatory, regardless of whether steroids are administered.[19,21]

*Treatment*

Whenever the diagnosis of this serious complication is confirmed by cardiac catheterization, important questions about appropriate therapy arise. In the early stage of the disease, during the predominantly inflammatory phase, medical therapy with NSAIDs together with increased diuretic and aggressive steroid administration may resolve constriction signs, especially when given within the first 2 months of the initial operation.[5,10,18] Steroids alone, in different doses, were tried in 3 cases and showed significant improvement in all, and cardiac catheterization confirmed recovery in 2 of the cases.[10]

Clinical improvement was reported when using diuretics alone.[13] As noted earlier, medical treatment does not preclude the development of pericardial fibrosis, and, in most cases, it has been tried in an attempt to avoid the aggressive surgical procedure.

Once the fibrosis and calcification phase has occurred and a "rigid shell" is present, visceral and parietal pericardiectomy is the treatment of choice, with mortality ranging from 5% to 11%.[15] Decortication of the heart with removal of the entire parietal pericardium and decompression of the involved grafts, or reconstruction of new bypass grafts, should normalize the cardiac pressures immediately after surgery. If an incomplete normalization of cardiac pressures is observed, a relook and repeat pericardiectomy may be indicated to address inadequate decortication. There are also reports of the efficacy of steroid treatment for recurrent constriction induced by inflammation after pericardial stripping.[5,18] Delayed recovery after prolonged constriction has also been attributed to secondary myocardial atrophy.[3]

## Conclusion

Constrictive pericarditis is a rare but critical complication of open heart surgery. A high index of suspicion coupled with a rigorous transthoracic echocardiogram, along with cardiac MRI, remains the best strategy for diagnosis. The optimal technique of open heart surgery to decrease the risk of this complication remains debatable. Treatment of constrictive pericarditis includes use of NSAIDs and steroids. Pericardiectomy has a high mortality, but it may be the only treatment choice in cases of advanced disease. ■

## References


1. Hancock W. On elastic and rigid forms of constrictive pericarditis: fundamentals of clinical cardiology. *Am Heart J*. 1980;100:917-923.
2. Kendall M, Rhodes G, Wolfe W. Cardiac constriction following aorta to coronary bypass surgery. *J Thorac Cardiovasc Surg*. 1972;64:142-154.
3. Rubeiro P, Sapsford R, Evans T, et al. Constrictive pericarditis as a complication of coronary artery bypass surgery. *Br Heart J*. 1984;51:205-211.
4. Schiavone WA. The changing etiology of constrictive pericarditis in a large referral center. *Am J Cardiol*. 1986;58:373-375.
5. Cimino J, Kogan A. Constrictive pericarditis after cardiac surgery: report of three cases and review of the literature. *Am Heart J*. 1989;118:1292-1301.
6. Deline J, Cable D. Clustering of recurrent pericarditis with effusion and constriction in a family. *Mayo Clin Proc*. 2002;77:39-42.
7. Cliff W, Grobety J, Ryan G. Postoperative pericardial adhesions: the role of mild serosal injury and spilled blood. *J Thorac Cardiovasc Surg*. 1973;65:744-751.
8. Ng A, Dorosti K, Sheldon W. Constrictive pericarditis following cardiac surgery—Cleveland Clinic experience: report of 12 cases and review. *Cleve Clin Q*. 1984;51:39-46.
9. Cunningham J, Spencer F, Zeff R, et al. Influence of primary closure of the pericardium after open-heart surgery on the frequency of tamponade, postcardiotomy syndrome, and pulmonary complications. *J Thorac Cardiovasc Surg*. 1975;70:119-126.
10. Ansinelli R, Weeks K, Key T. Effects of steroids on postoperative constrictive pericarditis. *Am J Cardiol*. 1983;51:1238-1241.
11. Sohi G, Flowers N, Ward W, Gray L. Constrictive pericarditis following heart surgery. *J Ky Med Assoc*. 1981;79:591-594.
12. Halon D, Koren G, Kriwisky M, et al. Constrictive pericarditis following coronary artery bypass grafting in a patient with chronic asymptomatic pericardial disease. *Cardiology*. 1983;70:280-284.
13. Ayzenberg O, Oldfield G, Stevens J, Beck W. Constrictive pericarditis following myocardial revascularization. *S Afr Med J*. 1984;65:739-742.
14. Bewtra C, Shultz R. Constrictive calcific pericarditis following coronary arterial bypass surgery. *Hum Pathol*. 1985;16:522-526.
15. Killan D, Furiasse J, Scanlon P, et al. Constrictive pericarditis after cardiac surgery. *Am Heart J*. 1989;118:563-569.
16. Beck C. The effect of surgical solution of chlorinated soda (Dakin's solution) in the pericardial cavity. *Arch Surg*. 1929;8:1659-1672.
17. Marsa R, Mehta S, Willis W, Bailey L. Constrictive pericarditis after myocardial revascularization. *Am J Cardiol*. 1979;44:177-184.
18. Kutcher M, King S, Alimurung B, et al. Constrictive pericarditis as a complication of cardiac surgery: recognition of an entity. *Am J Cardiol*. 1982;50:742-748.
19. Matsuyama K, Matsumoto M, Sugita T, et al. Clinical characteristics of patients with constrictive pericarditis after coronary bypass surgery. *Jpn Circ J*. 2001;65:480-483.
20. Bush C, Stang J, Wooley C, Kilman J. Occult constrictive pericardial disease: diagnosis by rapid volume expansion and correction by pericardiectomy. *Circulation*. 1977;56:924-931.
21. Kabbani S, Bashour T, Ellertson D, et al. Constrictive pericarditis following myocardial revascularization: a possible cause of graft occlusion. *Am Heart J*. 1985;110:493-496.


## Main Points

- In constrictive pericarditis, compression and restriction of the heart by thickened pericardium causes equalization and elevation of diastolic pressures of the heart chambers.
- Postpericardiotomy syndrome occurs in up to 30% of patients after cardiac surgery; if not adequately treated, it may eventually cause constriction.
- Cardiac catheterization remains the gold standard for diagnosis.
- Patients who develop early postsurgical pericardial effusion should be considered at high risk for constrictive pericarditis.
- In the early, inflammatory phase of the disease, medical therapy may resolve inflammation and constrictive signs, but it does not preclude development of pericardial fibrosis. Once fibrosis and calcification occur, visceral and parietal pericardiectomy is the treatment of choice.