Iatrogenic Pericardial Effusion and Tamponade in the Percutaneous Intracardiac Intervention Era

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The number, specific type, and complexity of percutaneous intracardiac procedures continue to evolve. Many of these procedures require left atrial access using transseptal techniques. These approaches carry with them the potential for pericardial effusion (PE) and cardiac tamponade, particularly in the setting when intraprocedural anticoagulation is being administered. PEs and even cardiac tamponade have been documented with both diagnostic as well as therapeutic procedures. When the effusion is a complication of an intracardiac procedure, it is usually the result of a cardiac perforation. The presentation depends on several factors including the structure that is perforated, the device that caused the perforation, the baseline hemodynamic status of the patient, and the level of anticoagulation present. The incidence has varied substantially although it has been recorded with essentially all intracardiac procedures, both diagnostic and therapeutic on both the right and left side of the heart. Prompt recognition is essential so that prevention of the transition from effusion to tamponade can be attempted (e.g., by reversing anticoagulation) or the hemodynamic collapse can either be averted or treated. Clinical, radiologic, and echocardiographic assessment are each important. Pericardiocentesis can be life-saving and is a core competency for all laboratories performing invasive cardiac procedures. Systems of care must include the knowledge base, equipment, and expert echocardiographic and interventional personnel. Collaboration with noninvasive colleagues and training interventionalists who perform intracardiac interventions, both electrophysiologists and interventional cardiologists, should be required as part of every invasive program. (J Am Coll Cardiol Intv 2009;2:705–17) © 2009 by the American College of Cardiology Foundation

The number and complexity of percutaneous intracardiac procedures continue to increase as a variety of new technologies for structural heart disease and electrophysiology interventions are introduced. Many of these procedures have in common the need for intraprocedural anticoagulation, and a number require transseptal puncture. As a result, they all carry a risk of pericardial effusion (PE) and tamponade. The procedures include closure of patent foramen ovale, atrial and ventricular septal defects; percutaneous heart valve repair, balloon dilation or percutaneous valve replacement; and left atrial (LA) appendage occlusion. In the electrophysiology setting, an increased risk for effusion is associated with left-sided arrhythmia ablations, placement of biventricular pacemakers, and implantable cardioverter-defibrillators. This article reviews the presentation, frequency, timing, and outcome of PE and tamponade as a complication of intracardiac interventions.
Hemodynamics of Tamponade

The pericardium consists of a visceral and parietal segment that extends from the lower third of the superior vena cava to the apex of the heart (1,2). The parietal pericardium is normally <2 mm thick and is composed of collagen and elastin; it is relatively noncompliant and acts to limit abrupt expansion of the heart in response to a variety of loading conditions. Normally, a small amount of fluid is present ranging in volume from 20 to 50 cc; this may cause a slight separation between the 2 pericardial surfaces, typically not more than 5 mm.

The response of the pericardium to fluid accumulation has been well described (2–4). The compliance of the parietal pericardium increases slowly, although it can eventually accommodate large volumes; 500 cc or more may be tolerated without any hemodynamic compromise when accumulation occurs over weeks to months (Fig. 1A). However, in the setting of percutaneous intervention, fluid accumulation is abrupt and as little as 100 cc may result in hemodynamic decompensation (3,4). The noncompliant pericardium has a steep pressure volume curve (Fig. 1B); as intrapericardial pressure rises, the transmural pressure results in collapse of the right atrium (RA) first, followed by collapse of the right ventricle (RV) during progressively longer portions of diastole (Fig. 2). Compression of left-sided chambers usually occurs in severe tamponade or, in some cases, when loculation affects a discrete portion of the left heart only. Another pathophysiological mechanism may relate to lack of LA and left ventricular (LV) filling. There is a continuum of clinical manifestations that depends upon the speed of accumulation and the absolute volume that accumulates as well as the underlying presence or absence of associated cardiac disease (2–4); this variability ranges from early echocardiographic manifestations of right-sided chamber compression to shock and hemodynamic collapse.

When PE is a complication of an intracardiac procedure, the effusion is usually the result of a perforation and may develop rapidly. The perforation may be caused by a guidewire, dilator, sheath, balloon or guiding catheter, pacemaker lead, or excessive ablation energy. The presentation depends in part on at least 5 factors: the size of the device responsible for the perforation; the structure that is perforated, such as atrial versus ventricular myocardium, RA or LA, RV or LV; the hemodynamic state at the time of perforation; properties of the pericardium itself; and the coagulation status. Full anticoagulation in particular is a major risk factor for both PE and its sequelae. After cardiac surgery, the pericardium may be absent or adherent to the myocardial reflection and may prevent the development of tamponade, although this is not the case universally because tamponade and hemodynamic compromise can result from a posterior localized effusion (and may be particularly difficult to reach during attempted pericardiocentesis).
The pressure within the structure that is perforated is a major determinant of the development and severity of tamponade. Thus a small perforation of the RV in an unanticoagulated patient may not be clinically apparent; in contrast, perforation of the RV in the setting of pulmonary hypertension or anticoagulation can be catastrophic. These same considerations apply for both the RA and LA as well.

The specific chamber involved is very important in determining not only presentation but also outcome. The RV typically has a transmural thickness of $\leq 4$ mm and placement of temporary and permanent pacemakers as well as electrophysiology catheters is associated with a small but significant perforation rate, the frequency of which is probably underestimated because of lack of clinical sequelae in many unanticoagulated patients. The thicker wall of the LV ($\geq 10$ mm) may act to seal small perforations, balancing the higher intrachamber pressures. However, even a small guidewire perforation may not be tolerated if it occurs in patients with resistance to LV outflow, such as in severe aortic stenosis. In this case, very high LV pressure and high afterload result in the newly created hole in the chamber acting as a “pop-off” valve, with blood preferentially driven toward an initially low pressure intrapericardial space. The possibility of tamponade is substantially increased, as was seen with early experiences of aortic valvuloplasty (5), for which a 1.8% rate of tamponade was reported in 492 patients, and caused by guidewire, catheter, or balloon perforation. In contrast, a small RA perforation may be well tolerated in some patients not on anticoagulants because of low intracardiac pressures and therefore low driving pressure. Perforation of the LA is potentially much more serious, primarily because procedures involving the LA are always associated with anticoagulation, and because LA pressure is typically higher than RA pressure. Finally, the geometry of the perforation may also be important. A slitlike perforation may result in different patterns of fluid accumulation than a circular hole, particularly if the wall of the affected chamber is thin.

**Recognition of PE and Tamponade**

**Clinical.** The occurrence of myocardial perforation and subsequent fluid accumulation within the pericardium should be suspected clinically during the time of catheterization if the patient begins to complain of chest pain. The typical pain of a new pericardial irritation is that of substernal discomfort sometimes radiating up into the neck and jaw. Acute pericardial irritation can also present with atypical symptoms such as shoulder discomfort, abdominal discomfort, or even nausea. In some instances, the patient may describe a sense of doom even before hemodynamic changes are observed.

Central aortic pressure and RA pressures can provide excellent indirect evidence of perforation and subsequent tamponade. In the very early stages, the blood pressure response is variable: although hypotension is a hallmark of tamponade, systemic aortic pressure may actually increase initially along with an increase in heart rate due to a sympathetic response to the initial pericardial irritation. On occasion, the earliest finding is acute bradycardia and hypotension, reflecting a vasovagal reaction to sudden pericardial stretch. Other symptoms may include nausea or chest discomfort. As tamponade begins to develop, systemic pressure may be sustained transiently by increased adrenergic stimulation and peripheral vasoconstriction. However, pulse pressure will decrease and pulsus paradoxus develop reflecting an exaggerated decrease in pulse pressure during inspiration. At this time, RV filling pressures will begin to elevate. The contour of the RA pressure will subsequently change during early stages of tamponade. There will be a loss of the $y$ descent and in patients who remain in sinus rhythm, and there will be a more prominent $a$ wave at the time of atrial contraction (Figs. 3A and 3B). As pericardial tamponade develops, there will then be a significant decrease in aortic systolic pressure and rise in RA pressure (Fig. 4). The pulse pressure may be so narrow during inspiration that the observer will be unable to detect individual heart beats (Fig. 5).
laboratory and occurs concomitantly with significant hemodynamic deterioration. Intervention is indicated in the early hemodynamic stages, once there is a noticeable pulsus paradoxus, even in the absence of a drop in overall systemic pressure.

Chest radiography may be helpful in some settings (6,7). Typically, large effusions present as globular cardiomyopathy with sharp margins, sometimes referred to as a “water bottle” silhouette. If the effusion develops during catheterization, it may also be identified by the development of lucent lines in the cardiopericardial silhouette—the so-called epicardial halo sign or fat pad sign.

Echocardiography should be performed as soon as there is a suspicion of PE or tamponade, such as with onset of chest discomfort after trans-septal puncture, even in the absence of hemodynamic abnormalities, because early diagnosis of tamponade should prompt the operator to discontinue the intervention and if appropriate, reverse the anticoagulation. In many instances, there may not be a large PE but rather a small circumferential effusion that was not present before the procedure. Although not all patients will have had a baseline echocardiogram, many patients have that as part of their diagnostic cardiac workup. In addition, in many
centers, pre-procedural echocardiography is an integral part of procedural planning, such as LA appendage obliteration or mitral valve dilation. Loculated PEs may also be seen, primarily in the posterior region of the pericardium (Fig. 6). The typical 2-dimensional echocardiographic findings of RV collapse and right atrial invagination may not be seen in the early stages of tamponade, because they require transmural pressures greater than the intracardiac chamber pressures; thus these findings may also not be present or may be delayed in patients who have specific forms of underlying heart disease where intracardiac chamber pressures are elevated. This may be particularly the case in patients with RV hypertrophy where pericardial fluid may not result in initial RA and RV diastolic collapse. One of the most sensitive methods for detecting early pericardial tamponade is the finding of a new septal shift on 2-dimensional echocardiography, indicating ventricular interdependence. Pulse-wave Doppler interrogation of the mitral inflow velocity is also important to detect early subclinical stages of tamponade, with dissociation of intrathoracic and intracardiac pressures as well as enhancement of ventricular interaction. This will result in a decrease in the initial E velocity during inspiration on the transmitral flow velocity curve. In typical tamponade, there is an overall decrease in the E:A ratio, due to a low early diastolic filling (Fig. 7). However, the initial E velocity will decrease further with inspiration due to decreased filling in the left side countered by increased filling of the right side during inspiration. One caveat must be kept in mind. Echocardiography initially universally documents a free space. However, as anticoagulation is stopped in a specific patient or if coagulation of the pericardial fluid starts, the free space may not be as visible. Careful analysis of the images and hemodynamic monitoring can identify and resolve this issue.

**Transseptal Puncture**

After its initial introduction (8) for diagnostic assessment of left heart pressures, transseptal catheterization nearly disappeared from the catheterization laboratory armamentarium (9) as indirect measurement of LA pressure by the pulmonary arterial wedge technique and retrograde LV catheterization were introduced. The last 2
decades have seen a rebirth of interest in transseptal left heart catheterization, occasioned primarily by LA structural and electrophysiologic interventions. A survey of hospitals in Italy showed a 60-fold increase in transseptal punctures by electrophysiologists alone between 1992 and 2003 (10) (Fig. 8).

Ross (11) recently reviewed the origins and development of the technique. In 1962, the Clinical Center at the National Institute of Health published the first 450 cases and reported no procedure-related mortality; subsequently in 1966, Ross (12) reported on 350 additional patients. At that time, relative contraindications to transseptal puncture included anticoagulation, LA thrombus, and anatomic abnormalities such as severe kyphoscoliosis or marked dilation of the ascending aorta. Some of these relative contraindications remain operative. The learning curve for transseptal procedures particularly when structural heart disease is present can be steep (13). Use of echocardiographic guidance has added a measure of safety to the operator learning curve (14) and allows for safer puncture in settings of anatomic deformity as well. It also allows more accurate determination of transseptal needle location in the fossa ovalis as opposed to the more muscular atrial septum; although entry into the LA through the latter is adequate for many purposes, passage of devices across the septum and manipulation of catheters through a thickened, sometimes calcified septal wall may seriously impair performance of some procedures. In contrast, some interventions, such as percutaneous mitral valve repair, may benefit from higher entry (15) and others, such as the percutaneous metal mitral valvulotome technique (16), required a relatively low puncture.

Transseptal puncture has been an important source of PE and tamponade in procedures requiring LA access even in a primarily diagnostic catheterization setting: a review of 278 transseptal punctures revealed a 3.2% incidence of pericardial puncture with incipient or clinically significant tamponade (17). In general there has been a growing base of experienced operators in the past decade. In addition, newer techniques (18–21), such as computed tomography guidance, radiofrequency, and laser-facilitated puncture are being developed along with technology for safer access via the jugular vein.

**Effusion and Tamponade as a Function of Specific Procedures**

The specific incidence of PE complicating intracardiac procedures varies depending on the procedure. Some of these procedures are diagnostic procedures such as myocardial biopsies or transseptal catheterization for assessment of aortic stenosis. However, they also occur with therapeutic procedures such as ablations, mitral or aortic valve dilation, or LA appendage occlusion devices. In general, mild to moderate effusions occur several times more frequently than tamponade. The latter ranges from <1% to 6% in most series depending on the technique for assessment, level of anticoagulation, and type of procedure among other factors. In reviewing the statistics that follow, it is important to note that most of the studies described did not prospectively screen for PE; thus small to moderate effusions, late effusions, and those that did not produce significant symptoms or hemodynamic compromise could have been missed in many of these series. To evaluate the exact incidence, studies would need to perform serial echocardiograms progressively screening from a pre-procedure baseline through post-hospital discharge follow-up. In the absence of such protocols, the overall numbers in many series may represent a small percentage of the actual effusions that occurred. An important additional reason that the incidence of effusion is underestimated is the timing. Although the effusion typically develops during the procedure, it may occur days later, particularly if the patient is subsequently placed on anticoagulant or intensive antiplatelet therapy (22,23).
Atrial Fibrillation Ablation

Ablation of left-sided pathways is a technically demanding and frequently prolonged procedure, requiring transseptal puncture and extensive catheter manipulation, prolonged anticoagulation, and multiple energy deliveries into the wall of the heart. Complications include pulmonary vein stenosis, esophageal injury, thromboembolism, phrenic nerve injury, and a significant incidence of tamponade (24). In 2007, the Heart Rhythm Society, European Heart Rhythm Association, and the European Cardiac Arrhythmia Society published an expert consensus statement on ablation for atrial fibrillation (25). This document described cardiac tamponade occurring in up to 6% of procedures and identified it as the most common potentially life-threatening complication associated with this procedure.
The specific type and power of the ablation protocol contributes to tamponade risk (26). The sites of perforation include the RA, roof or free wall of the LA, and the LA appendage. Bunch et al. (27), reviewed 632 procedures performed from January 1999 to October 2004, 2.4% of which were complicated by perforation requiring pericardiocentesis. The most common site of perforation was the LA (60%), followed by the RV (33%), and the RA (6.7%). All were initially managed with pericardiocentesis, but 13% subsequently required surgery because of persistent bleeding. Of importance is the fact that in >90% of the patients, routine use of intracardiac echocardiography documented an effusion before the onset of overt hemodynamic instability. Recently, Fagundes et al. (28) reported on 1,150 consecutive patients undergoing single transseptal punctures in anticipation of atrial fibrillation ablation. In this series, which enrolled patients from 2003 to 2005, a PE was documented in 2.7% and tamponade requiring pericardiocentesis in 1%.

Other recent series include a worldwide survey of atrial fibrillation ablation that reported an incidence of tamponade from 1% to 3% (29). The variability in reported incidence may result from several factors including different ablation energy strategies, routine use of intracardiac ultrasound, and operator learning curve. The incidence of effusion not resulting in tamponade is likely several times higher; detection in this setting depends upon how often routine echocardiographic screening is performed.

### Pacemakers

The incidence of cardiac perforation with permanent pacemaker implantation also varies widely. Mahapatra et al. (30) evaluated 4,280 permanent pacemakers implanted from 1995 to 2003 and found an incidence of suspected perforation within 7 days of the procedure in 1.7%. Of 50 patients with chest pain or hypotension without known prior effusion, tamponade physiology was demonstrated by echo in 60%, 83% of whom had hypotension; 70% required pericardiocentesis. Multivariate predictors of perforation included use of a temporary pacemaker, helical active fixation leads, and steroids. In a similar type of analysis, Navarrete et al. (31) reported on their experience of cardiac tamponade requiring pericardiocentesis or surgical drainage from 1985 to 2002 and found that 5.4% of these cases were related to pacemaker implantation. Danik et al. (32) reported an incidence of 3.8% with a specific implantable cardioverter-defibrillator lead, 40% of whom required pericardiocentesis. The time course is occasionally significantly delayed (33), including reports of tamponade occurring >30 days after implantation (22).

The etiology of the perforation varies in this setting. PE may result from attempts to position a lead in the tributaries of the coronary sinus or during attempted removal of failed leads. As previously mentioned, the apex of the RV is often very thin walled, particularly in older women. Placement of a permanent or a temporary pacemaker may result in PE or tamponade even without adjunctive anticoagulation. Techniques to prevent this include the use of biplane fluoroscopy or echo to ensure that the lead is placed against the septum instead of at the free wall. Small 3-F temporary “screw-in” leads can aid in avoiding perforation with temporary pacemaker insertion.

### Percutaneous Mitral Valvuloplasty

This procedure began the modern structural heart disease intervention era. It is associated with up to 4% risk of tamponade, a level reported in both a 146-patient series from the Beth Israel Hospital in Boston (34) and the 738-patient National Heart, Lungs, and Blood Institute Mitral Valvuloplasty Registry (35) (Table 1 [36–45]). The causes of tamponade are multifactorial, including transseptal puncture, and, in the era of single or double cylindrical balloon use, rare but catastrophic “harpooning” of a balloon through the apex of the LV. Catheter and sheath manipulation in the LA were culprits as well. A review of 10 cases of tamponade from a series of 903 mitral valvuloplasty patients (43) demonstrated the following mechanisms: perforation during transseptal puncture (2 cases), harpooning of the LV apex (3 cases), apical guidewire perforations (2 cases), stitch perforation of the RA caused by low transseptal puncture with fenestration of the posterior RA wall (2 cases), and temporary pacing catheter-induced RV perforation (1 case). The investigators demonstrated an inverse
relationship between incidence of tamponade and operator experience. The percutaneous metal commissurotomy device designed by Cribier et al. (16) caused tamponade secondary to trauma from a stiff guidewire and rigid metal bulb designed to exert traction on the metal valvulotome.

**LA Appendage Occlusion**

This procedure involves placement of a nitinol frame structure with a polyester membrane and fixation barbs inside the LA appendage. The device is designed to be partially compressed and to be held in place by a combination of radial force and the fixation barbs that protrude approximately 1 mm into the wall of the appendage. The LA appendage itself can be extremely thin-walled. In the initial 66-patient pilot experience with the Watchman device (Atritech, Plymouth, Minnesota), there were 2 cases of tamponade (3.0%) and 3 additional effusions that were described as minor (46). The use of a predecessor device, PLAATO (Percutaneous Left Atrial Appendage Transcatheter Occlusion) (Appriva Medical Inc., Sunnyvale, California), made of a nitinol cage with a polytetrafluoroethylene fabric and 3 rows of fixation barbs was associated with pericardiocentesis in 2 cases of tamponade (1.8%) and 1 of 2 nontamponade effusions in a 111-patient series; in addition 1 hemotherax occurred (47). With increasing operator experience, manipulation of the delivery sheath and occluder device inside the appendage has become more atraumatic; in addition both the sheath and occluder have been modified to minimize trauma to the appendage. These maneuvers and approaches have appeared to decrease the frequency of PEs.

**Miscellaneous**

There are an increasing number of other therapeutic invasive cardiac procedures. Many of these involve transseptal puncture or entry into the LA via a patent foramen ovale. Each of these has the potential to have PE and tamponade as complications.

**Percutaneous Patent Foramen Ovale Closure**

Percutaneous closure of patent foramen ovales has been performed with increased frequency—both for prevention of stroke and more recently treatment of recurrent migraine (48–53). The specific frequency of PE or tamponade is difficult to ascertain from the literature. In a systematic review of the literature, which included 10 studies and 1,355 patients, Khairy et al. (48) classified major complications of the procedure as death, hemorrhage requiring blood transfusion, massive pulmonary emboli surgical intervention, and cardiac tamponade as major events, which occurred in 1.5% of cases.

A number of other procedures require transseptal puncture. These include LV assist using the TandemHeart Device (Cardiac Assist, Pittsburgh, Pennsylvania) and prosthetic mitral paravalvular leak closure. Both have been associated with tamponade although reports are anecdotal (54). Early reports of complications after percutaneous aortic valve replacement via the transseptal technique have included up to a 7.4% incidence of mortality related to periprocedural tamponade (55).

**Percutaneous Valve Repair and Replacement**

There is burgeoning interest in the percutaneous repair or replacement of valves—both mitral regurgitation and aortic stenosis. Both procedures have the potential to result in the complication of PE. For each of these procedures, although the number of patients is increasing, there is limited information on that specific complication. The pathophysiology of the PE varies. With percutaneous mitral valve repair, a transseptal approach is required unless only a coronary sinus approach is used. As is true with any transseptal procedure, a PE is possible. With percutaneous aortic valve replacement using a retrograde aortic approach, the guidewires used a pre-valve replacement balloon dilation that may result in perforation of the LV (56,57).

**Percutaneous Coronary Intervention**

PES have also been reported as a result of percutaneous coronary interventions (58–63). These are infrequent events. Shirakabe et al. (61) evaluated 3,415 percutaneous coronary intervention procedures between 1991 and 2005, during which time, perforation occurred in 12 cases (0.35%). Javaid et al. (62) evaluated a 9-year period and found 72 cases (0.19%). These occurred in several settings. During the time when directional atherectomy was used frequently, excessive tissue removal was the cause. It may also be seen with other atheroablative approaches such as rotational atherectomy, particularly as a result of treatment of angulated lesions with guidewire bias. Perforation may also occur in the setting of treatment of chronic total occlusions using either antegrade or retrograde approaches with a variety of special guidewires. Guidewire coronary perforation in this setting may be a benign event and may be under-reported unless a balloon is inflated in the tract; in which case, tamponade may ensue. The definitive treatment of coronary perforations is different than with perforations.
of a cardiac chamber. With coronary perforations, a variety of approaches are possible including prolonged balloon inflation in addition to reversing anticoagulation, covered stents if the artery is large enough to accommodate these devices, or embolization for small vessels. If tamponade develops as a result of these perforations, obviously that must be treated urgently when definitive percutaneous treatment of the coronary perforation is undertaken.

**Pericardiocentesis**

Early diagnosis and prompt treatment of PEs with pericardiocentesis is life-saving and should be a core competency for all laboratories performing invasive cardiac procedures (64–66). Systems of care must include knowledge base, equipment, and expert echocardiographic and interventional personnel. All catheterization laboratories should have a pericardial puncture “tray set-up” and standard operating procedures. In 1 of the largest series of pericardiocenteses, Tsang et al. (64) evaluated clinical profile, practice patterns, and outcomes in 1,127 therapeutic echocardiograph-guided pericardiocenteses on 977 patients over a 21-year period. Of patients undergoing pericardiocentesis, 30% had echocardiographic evidence of tamponade physiology, 44% had evidence of clinical tamponade, and 10% had evidence of hemodynamic collapse; the remaining 16% had evidence of effusions without tamponade physiology.

The treatment of iatrogenic PE varies. Typically, when a new effusion is documented, the procedure should be terminated and any anticoagulation should be reversed. In some patients, when only a small effusion is present, these 2 maneuvers are sufficient to prevent hemodynamic deterioration. At the other end of the spectrum is tamponade requiring urgent resuscitation with pericardiocentesis and placement of an indwelling catheter for continued evacuation. If there has been a tear in a cardiac structure, percutaneous drainage may not successfully resolve the problem and, although infrequently, surgery may be required.

Using ultrasound guidance, the optimal window for pericardiocentesis can be determined; in many instances, the apical and not the subcostal approach is superior. This is particularly true in acute tamponade, where most of the effusion is best visualized from an apical window on the posterior portion of the pericardium, necessitating an apical entry with the needle directed posteriorly. In some instances, a subcostal or lower parasternal approach is best if the effusion cannot be reached via an apical approach. In general, the specific route for needle placement should be that route that provides the shortest and easiest access to the pericardial space. If a subxyphoid approach is used, care should be taken to avoid trauma to the left lobe of the liver. For intercostal approaches, care must be taken to place the needle superior to the specific rib margin to avoid damage to the intercostal areas. Adequate local anesthesia is important. Some operators insert a long sheathed 16- to 18-gauge needle, whereas others prefer a micropuncture approach. Limited aspiration throughout the insertion of the needle with a 20-cc syringe is useful. Once blood is withdrawn through the needle, agitated saline should be injected under echocardiographic guidance to ensure that the entry point is within the pericardium and not within the ventricular chambers. Once the entry has been identified, the sheath is advanced into the pericardial space. Alternatively, a guidewire is inserted into the pericardium under fluoroscopy (to ensure that there is free flotation of the wire and that the course is consistent with extracardiac insertion and position). With either technique, a pigtail catheter is inserted and connected to continuous negative pressure. In certain situations, a “cell saver” can be used so that the blood drawn from the pericardial space can be reinfused back into the patient.

Although echo-directed pericardiocentesis is optimal, the procedure should not be delayed if an echo machine is not available and hemodynamic collapse is imminent. Straightening and immobility of the left heart border, especially if mobility was confirmed to be normal before transseptal puncture, is usually pathognomonic for tamponade. If the patient is rapidly deteriorating, a “blind” pericardiocentesis from a subcostal approach should be attempted by an experienced operator rather than waiting for echocardiography during hemodynamic collapse. Intubation should be avoided if at all possible, because the increased transthoracic pressure may lead to cardiac arrest (65). Temporizing medical therapy is controversial: rapid infusion of fluids is advocated by most operators, although the hemodynamic benefits have been called into question because rapid volume infusion indirectly increases intrapericardial pressure. Intravenous nitroprusside to reduce afterload and/or dobutamine may also be of benefit in some patients until a pericardiocentesis can be performed (Figs. 9A and 9B).

**Conclusions**

PEs and tamponade remain an important source of complications associated with a wide variety of structural heart disease and electrophysiologic interventions. Because of the growing number and types of these procedures, and the significant learning curves involved, establishing optimal training programs in transeptal puncture and in catheter manipulation in the LA will be important. Collaboration with noninvasive colleagues and training interventionalists who perform intracardiac interventions to interpret echocardiograms should be a part of every invasive program.
Early detection of PE before onset of hemodynamic manifestations and prompt pericardiocentesis may prevent further complications in many patients. The utility of the ever-increasing number of intracardiac interventions will likely continue to offset the associated risks, even as the risks will likely decrease with increasing operator experience and improving technology.

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Figure 9. High-Fidelity Pressure Traces Before and After Intravenous Fluids and NTP Treatment in a Patient Who Has Developed Pericardial Tamponade

(A) High-fidelity left and right ventricular pressure traces in a patient with pericardial tamponade. There is marked elevation and equalization of diastolic pressures with loss of early diastolic filling. The left ventricular end-diastolic pressure is 32 mm Hg. During the fourth beat, there is a marked drop in left ventricular systolic pressure and a rise in right ventricular systolic pressure, indicating enhancement of ventricular interaction. Overall, the systolic pressure is <100 mm Hg. (B) The same patient after receiving emergency treatment with intravenous fluids and nitroprusside (NTP). Although there is still evidence of enhancement of ventricular interaction and a pulsus paradoxus, the systolic pressure of the left ventricle has significantly increased to 120 mm Hg. These medical treatments are a “temporizing” approach to stabilize hemodynamics before emergency pericardiocentesis is performed.


Key Words: cardiac tamponade ■ pericardial effusion ■ pericardiocentesis.