Left Ventricular Diastolic Collapse

An Echocardiographic Sign of Regional Cardiac Tamponade

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Background. Cardiac tamponade after cardiac surgical procedures is often associated with hemodynamically significant localized pericardial effusions. The localized collection of pericardial effusion in the postoperative period and the atypical presentation of cardiac tamponade limit the use of conventional clinical and echocardiographic signs usually seen with a circumferential pericardial effusion. Observation of left ventricular diastolic collapse in the echocardiogram of a patient with postoperative regional cardiac tamponade prompted us to explore the frequency of this sign in regional cardiac tamponade.

Methods and Results. We retrospectively analyzed the echocardiograms of 18 patients with postoperative cardiac tamponade for the following echocardiographic findings: right atrial collapse, right ventricular diastolic collapse, left atrial collapse, and left ventricular diastolic collapse. Three of the 18 patients had circumferential pericardial effusion, and 15 had loculated pericardial effusion; in 10, the effusion was predominantly posterior, and in the other five, it extended laterally or inferiorly. The conventional echocardiographic signs of cardiac tamponade such as right atrial collapse, right ventricular diastolic collapse, and left atrial collapse were present in only 3, 1, and 3 of these 15 patients, respectively, but all exhibited left ventricular diastolic collapse. Increasing pressure within the compartment of a loculated pericardial effusion reaching the limit of pericardial distensibility and consequent transient reversal of transmural left ventricular pressure during diastole are most likely the basis for diastolic collapse of the thick-walled ventricle in a setting of regional cardiac tamponade.

Conclusions. We conclude that left ventricular diastolic collapse is a frequent sign of regional cardiac tamponade and could be a useful marker of tamponade in postoperative patients. (Circulation 1991;83:1999–2006)

Pericardial effusion is commonly seen in patients after cardiac surgery. It is usually small in amount and inconsequential. However, in some patients, a pericardial effusion may be circumferential and quite large, or it may be regional and located in a strategic area, either of which may impede cardiac filling, reduce cardiac output, and lead to tamponade. Cardiac tamponade is an important consideration in the differential diagnosis of the low-output state, hypotension, or severe dyspnea in the postoperative period. Early recognition of tamponade is crucial for the optimal management of these critically ill patients. Clinical examination and echocardiography play an important role in the diagnosis of cardiac tamponade. In the setting of cardiac tamponade resulting from a circumferential pericardial effusion, echocardiography frequently reveals right ventricular diastolic collapse (RVDC) and right atrial collapse (RAC), which serve as useful signs of cardiac tamponade. These classic signs could, however, be absent in tamponade caused by a regional effusion. In patients who have had cardiac surgery, pericardial effusion is frequently regional and is often localized posteriorly. In these patients, right ventricular free wall and right atrial wall are commonly adherent to the anterior chest wall without any significant pericardial effusion in between. Clinical findings such as jugular venous distention, pulsus paradoxus, or hypotension may be insensitive, nonspecific, or difficult to evaluate in this circumstance. The usefulness of echocardiography in the evaluation of tamponade caused by a regional...
pericardial effusion has not been systematically evaluated, although isolated echocardiographic observations of abnormalities of left ventricular contour in patients with cardiac tamponade after cardiac surgery have been reported earlier.\textsuperscript{15–17} We noted an early diastolic invagination of the left ventricular free wall (left ventricular diastolic collapse, LVDC) in one of our patients with a localized posterior pericardial effusion after coronary artery bypass surgery (CABG). This led us to examine for the presence of LVDC in patients with regional cardiac tamponade after cardiac surgery and to compare the frequency of LVDC with that of other echocardiographic signs of tamponade.

**Methods**

**Patient Population**

We performed a retrospective analysis of the echocardiographic studies of 18 patients with documented cardiac tamponade after cardiac surgery who had undergone echocardiography before fluid drainage in two institutions from 1986 to 1990. Of these 18 patients, 12 had undergone surgery at the New England Medical Center, Boston, and six at the Veterans Administration Medical Center, Richmond, Va. Eight patients had undergone CABG; one, CABG and left ventricular aneurysmectomy; two, CABG and aortic valve replacement; one, CABG and mitral valve replacement; one, aortic and mitral valve replacement; three, cardiac transplantation; and two, aortic valve replacement. The pericardia were left open and not approximated in all patients. Seventeen patients had dyspnea as the predominant symptom, and one patient was excessively fatigued and had failed to thrive after surgery. Of the 18 patients included in the study, 15 had evidence of loculated regional pericardial effusion, and three had circumferential pericardial effusion. Because regional pericardial effusion is generally more common in postoperative patients than is circumferential effusion and because the diagnosis of regional cardiac tamponade caused by loculated effusion is more difficult than that caused by circumferential effusion, we focused our analysis on the 15 patients with loculated regional effusion. Analysis of these 15 patients revealed that 10 had hemodynamic criteria consistent with tamponade physiology (elevated right atrial and right ventricular diastolic pressures and elevated pulmonary capillary wedge pressure, with equalization [within 5 mm Hg] of right atrial and pulmonary capillary wedge pressures); two patients did not demonstrate diastolic pressure equilibration on right heart catheterization but did have increased diastolic pressures and exhibited significant clinical improvement after pericardial fluid drainage. Three other patients included in the study did not undergo catheterization. These three patients had large posterior pericardial effusions, were extremely unstable, required emergency drainage procedure without prior right heart catheterization, and demonstrated significant clinical improvement after the drainage procedure. The demographic data on the patients were obtained from the medical records (Table 1). The time of the drainage procedure from the date of the surgery was noted. Additional information on the type of pericardial drainage procedure, the amount of fluid obtained, and the symptoms before and after the drainage procedure were also recorded.

**Table 1. Clinical and Diagnostic Features**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Operation</th>
<th>Day</th>
<th>Pulsus paradoxus</th>
<th>RA (M)</th>
<th>RV (S/D)</th>
<th>PA (S/D)</th>
<th>PCW (M)</th>
<th>Drainage procedure</th>
<th>Fluid (ml)</th>
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<tbody>
<tr>
<td>1</td>
<td>62</td>
<td>M</td>
<td>CABG, ANR</td>
<td>23</td>
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<td>14</td>
<td>35/14</td>
<td>35/14</td>
<td>14</td>
<td>Pericardiocentesis</td>
<td>50</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>M</td>
<td>CABG, AVR</td>
<td>11</td>
<td>AFIB</td>
<td>19</td>
<td>37/16</td>
<td>37/25</td>
<td>22</td>
<td>Surgical drainage</td>
<td>200</td>
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<tr>
<td>3</td>
<td>56</td>
<td>M</td>
<td>CABG</td>
<td>18</td>
<td>20</td>
<td>12</td>
<td>25/10</td>
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<td>Pericardiocentesis</td>
<td>210</td>
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<tr>
<td>4</td>
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<td>CABG</td>
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<td>13</td>
<td>20</td>
<td>42/15</td>
<td>42/22</td>
<td>21</td>
<td>Surgical drainage</td>
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<td>34</td>
<td>M</td>
<td>CT</td>
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<td>50/30</td>
<td>25</td>
<td>Surgical drainage</td>
<td>600</td>
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<td>13</td>
<td>54</td>
<td>M</td>
<td>CABG</td>
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<td>AFIB</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>Surgical drainage</td>
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</tr>
<tr>
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<td>M</td>
<td>CT</td>
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<td>-</td>
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<td>25/15</td>
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<td>-</td>
<td>16</td>
<td>40/16</td>
<td>40/18</td>
<td>18</td>
<td>Surgical drainage</td>
<td>570</td>
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</table>

RHC, right heart catheterization; RA, right atrial; RV, right ventricular; PA, pulmonary artery; PCW, pulmonary capillary wedge pressure; M, mean; S, systolic; D, diastolic; CABG, coronary artery bypass; ANR, aneurysmectomy; AVR, aortic valve replacement; CT, cardiac transplant; AFIB, atrial fibrillation.
Echocardiographic Analysis

All the patients had undergone two-dimensional echocardiographic examination with a commercially available echocardiographic instrument. Imaging views consisted of the conventional two-dimensional echocardiographic views; that is, parasternal long-axis, short-axis at the basal, mid- and apical level, apical four-chamber, and subcostal views. Patients with technically suboptimal anterior acoustic windows underwent imaging from additional posterior acoustic windows from the back. While reviewing the echocardiograms, we specifically examined the following features: 1) location of the pericardial effusion, 2) approximate visual estimate of the size of the effusion, 3) RAC, 4) RVDC, 5) left atrial collapse (LAC), and 6) LVDC. The presence or absence of these signs was correlated with other available clinical and hemodynamic findings of tamponade observed in these patients. The studies were reviewed by two observers independently for assessing interobserver variability and on two occasions by one observer for assessing intraobserver variability in the identification of RAC, RVDC, LAC, and LVDC.

Results

The mean age of the 15 patients was 57±9 years (range, 34–69 years). There were 14 men and one woman (Table 1). The mean time of the diagnoses of tamponade after surgery was 82±95 days (range, 11–330 days). The mean right atrial pressure was 15±5 mm Hg (range, 12–24 mm Hg), mean right ventricular diastolic pressure was 16±4 mm Hg (range, 10–24 mm Hg), mean pulmonary artery diastolic pressure was 19±5 mm Hg (range, 13–30 mm Hg), and the mean pulmonary capillary wedge pressure was 19±3 mm Hg (range, 14–25 mm Hg). Only nine patients had pulsus paradoxus exceeding 10 mm Hg, and among them, three had pulsus paradoxus of only 12 mm Hg. All patients were symptomatic (dyspnea in 14 and failure to thrive in one), and all demonstrated relief of symptoms and clinical improvement after pericardial fluid drainage. Seven patients underwent pericardiocentesis, two patients underwent pericardiocentesis followed by surgical drainage, and six patients had surgical drainage only. The amount of pericardial fluid drained was 662±464 ml (range, 50–1,495 ml). In one patient (patient 13), the amount of free fluid that could be removed was only 50 ml, although the echocardiogram had revealed a moderate-sized posterior effusion. The remainder of the material removed consisted of an unmeasured mixture of blood and clots. This patient had marked clinical improvement after the surgical drainage.

Of the 15 patients, five patients were receiving anticoagulant therapy. Of the six patients who had late presentations (98–330 days after surgery), one patient was receiving anticoagulants. None of the 15 patients had overt evidence of postpericardiotomy syndrome.

Echocardiographic Findings

Two-dimensional echocardiograms in these patients (Table 2) revealed a large pericardial effusion in 10 and a moderate-sized effusion in five patients. The location of the pericardial effusion was posterior in 10 patients (mostly loculated). Three patients had lateral extension of the effusion, and in two patients, the effusion extended inferiorly. Eleven of the 15 patients showed adhesions between the anterior wall of the right ventricle and the posterior surface of the sternum without any intervening pericardial fluid (Figure 1). Of the 15 patients, only three had echocardiographic evidence of RAC, and these three had a minimal amount of effusion adjoining the right atrium. One patient, who had a large posterior pericardial effusion, also had a small amount of

<table>
<thead>
<tr>
<th>Patient</th>
<th>Effusion size</th>
<th>Effusion location</th>
<th>RV adherence</th>
<th>RA</th>
<th>RV</th>
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<tbody>
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<tr>
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<td>Posterior/inferior</td>
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<td>+</td>
<td>+</td>
<td>+</td>
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</tr>
<tr>
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<td>Large</td>
<td>Posterior/lateral</td>
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<td>+</td>
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<td>Large</td>
<td>Posterior</td>
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<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>Posterior</td>
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<td>+</td>
<td>+</td>
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<td>Posterior</td>
<td>+</td>
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<tr>
<td>8</td>
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<td>Posterior</td>
<td>+</td>
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<tr>
<td>9</td>
<td>Large</td>
<td>Posterior</td>
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<td>Posterior/lateral</td>
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<td></td>
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<tr>
<td>12</td>
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<tr>
<td>13</td>
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<td>15</td>
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<td>Posterior</td>
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RV, right ventricular; RA, right atrial; LA, left atrial; LV, left ventricular.
pericardial fluid adjoining the right ventricle and demonstrated RVDC. LAC was evident on echocardiograms of three patients. All 15 patients showed LVDC (Figures 1–3). Interobserver and intraobserver concordance were excellent in the identification of not only RAC, RVDC, and LAC, but also LVDC.

LVDC was characterized by a transient regional invagination of the left ventricular posterior wall during early diastole. In contrast to the normal smooth convex contour of the left ventricular wall seen throughout diastole, the left ventricular posterior free wall in our patients with regional tamponade displayed a different motion pattern: The wall contour was convex toward the pericardial effusion at the end of systole and at the very beginning of diastole, but immediately thereafter, instead of moving posteriorly as the ventricle relaxed, there was an abrupt, sudden inward invagination or buckling of the wall toward the ventricular cavity. This inward invagination was transiently noted during the early or mid-diastolic phase; by mid- or late diastole, the wall resumed its expected, normal posterior motion, and the ventricle resumed its normal shape. The degree of LVDC was variable, ranging from mild inward bowing of a limited region of the posterior wall in some patients to a striking invagination of the whole posterior wall in some others. Similarly, the LVDC was noted only during early diastole in some cases.

**Figure 1.** Serial echocardiographic images in the parasternal long-axis view from a postoperative patient with cardiac tamponade shows a large posterior pericardial effusion (PE). Right ventricle (RV) is adherent to the anterior chest wall. Contour of the left ventricular (LV) free wall is normal at end systole. During early diastole, however, LV posterior wall invaginates inward, which is identified by the arrow as LV diastolic collapse (LVDC). This LVDC is only transient; by late diastole, LV has assumed its normal contour. LA, left atrium; Ao, aorta.

**Figure 2.** Two-dimensional echocardiographic image (parasternal long-axis view, early diastolic frame) in a postoperative patient with cardiac tamponade. There is a large loculated posterior pericardial effusion (PE). Image depicts a localized invagination of the posterobasal portion of the left ventricular (LV) free wall, which is identified by the arrow as LV diastolic collapse (LVDC). RV, right ventricle.
whereas the duration extended to mid-diastole in others. On M-mode echocardiography, LVDC was seen as an inward motion of the left ventricular posterior wall during early diastole (Figure 4), followed by resumption of the normal posteriorward diastolic motion in the later part of diastole. After the evacuation of the pericardial fluid, LVDC disappeared in all cases.

Discussion
Our study shows that LVDC is a common echocardiographic sign of regional cardiac tamponade caused by posteriorly loculated pericardial effusion in postoperative patients. While the utility and limitations of echocardiographic signs such as RVDC and RAC have been studied in detail, the potential value of LVDC is not well known. This study demonstrates that this finding is a frequent one in postoperative regional tamponade. Cardiac tamponade in patients after cardiac surgery continues to be a cause of excessive morbidity. Recognition of cardiac tamponade in these patients is often difficult because of the frequent lack of conventional clinical and echocardiographic signs. LVDC may prove to be a valuable marker of tamponade in this clinical scenario.

Regional Cardiac Tamponade in Postoperative Patients
Although large circumferential pericardial effusions do occur in patients after cardiac surgery, the pericardial effusion is frequently loculated posteriorly. Postoperative adherence of the anterior right ventricular and right atrial wall and the anterior pericardium to the anterior chest wall, and adhesions that develop in the surrounding area do not generally allow space for fluid collection anteriorly. When a pericardial effusion develops because of inflammation or bleeding in these patients, the fluid tends to collect posteriorly and laterally. The loculated nature of pericardial effusion was seen in our patients not only in the early postoperative period, but some were seen months after surgery. Because many cardiac patients may already have abnormalities in the size and function of cardiac chambers resulting from the valvular or ischemic heart disease for which they have undergone surgery, and because cardiac surgery may itself result in dysfunction of the cardiac structures such as the right and left ventricles, the pathophysiology of cardiac tamponade in these patients may not always be associated with the conventional clinical and hemodynamic findings of tamponade. The regional nature of the effusion may further compound the evolution of hemodynamic derangement and its clinical expression. In our patients, pulsus paradoxus was marginal in three, and a certain degree of disparity between right atrial pressure and left-sided filling pressure was noted in two patients. In these two patients, the pulmonary artery diastolic or pulmonary capillary wedge pressure was higher than the right atrial pressure. In view of the lack of reliability of the classic clinical and hemodynamic criteria and of conventional echocardiographic signs to diagnose postoperative regional cardiac tamponade, any additional or new diagnostic marker would be of value in the recognition of this condition. Two-dimensional echocardiography, which can disclose the presence

**Figure 3.** Two-dimensional echocardiographic images obtained from the posterior region of the thorax (back) from a postoperative patient with cardiac tamponade. In this patient with technically difficult anterior acoustic windows, the presence of a left pleural effusion (PL E) allowed adequate imaging from the back. A large loculated pericardial effusion (PE) is present posteriorly. In early diastole, the left ventricular (LV) free wall is noted to collapse inward, that is, LV diastolic collapse (LVDC). By late diastole, the LV contour has become normal. RV, right ventricle.
and location of pericardial effusion, is extremely useful in these patients. Its value in the detection of cardiac tamponade caused by localized regional pericardial effusion has not been evaluated thus far.

**Echocardiography in Regional Cardiac Tamponade and a Possible Mechanism Behind LVDC**

In the setting of cardiac tamponade caused by a circumferential pericardial effusion, RVDC, RAC, and LAC have proven to be extremely valuable diagnostic signs. Flow abnormalities associated with tamponade such as exaggerated inspiratory cyclical variation in intracardiac flow velocities provide further aid in recognizing a hemodynamically significant effusion. In most nonsurgical conditions, the pericardial effusion is circumferential and nonloculated. Under these circumstances, the thinner-walled right atrium and right ventricle are more compliant than the left ventricle and are also usually free from the adjoining structures. The frequently noted signs of cardiac tamponade such as RVDC and RAC occur at a time in the cardiac cycle when the intrapericardial pressure transiently exceeds or nearly exceeds the intracavitary pressure. These signs have proven to be useful diagnostic signs of a hemodynamically significant pericardial effusion except in circumstances of pulmonary hypertension and isolated instances of regional tamponade.15,17,21

The thicker and relatively less-compliant left ventricle usually does not show a regional diastolic collapse when the effusion is circumferential. A posteriorly loculated effusion surrounding the left ventricle creates a different hemodynamic environment. The presence of adhesions between the right ventricular free wall and the chest wall may prevent the collapse of the right ventricular free wall. As more and more fluid collects within the posterior region, the ability of pericardium to distend and accommodate the fluid can halt at one point, and the intrapericardial pressure can increase. As the intrapericardial pressure increases beyond the distending limit of the parietal pericardium, the only structure that can yield is the adjoining left ventricular wall. If the pressure inside the compartmentalized effusion exceeds the intracavitary diastolic pressure of the left ventricle disproportionately, then the left ventricular wall yields secondary to the localized compressive effect and invaginates toward the cavity of the chamber.

In an echocardiographic analysis of seven patients with postoperative tamponade associated with a loculated pericardial effusion, D'Cruz and coworkers noted an abnormal contour of the left ventricular posterior wall in all seven and a “paradoxical" motion of the left ventricular posterior wall in two. They interpreted this finding as a phenomenon secondary to alterations in the shape of the loculated pericar-
dial effusion during the cardiac cycle. A similar finding was observed in the M-mode echocardiogram of a patient reported with late isolated left ventricular tamponade by Jones and colleagues.16 Steele and Perez17 encountered LVDC in a patient with cardiac tamponade but believed that the LVDC actually provoked cardiac tamponade. A report by Hsu and colleagues,21 though dealing with a single case, illustrated the importance of the regional nature of tamponade for development of LVDC. In their patient, who presented with a circumferential malignant pericardial effusion and tamponade, echocardiography revealed RVDC but no abnormalities in the left ventricular wall dynamics. The patient underwent pericardiocentesis and improved. When she developed severe dyspnea due to recurrent tamponade 4 weeks later, echocardiography revealed a posteriorly loculated pericardial effusion, and although there was no RVDC, there was regional paradoxical motion of the left ventricular posterior wall. Inspection of the figure in this report reveals that this abnormal motion is very similar to the LVDC we observed in our patients. Our present study follows our earlier preliminary observations of LVDC in a small number of patients with regional tamponade. We believe that LVDC is an important feature of a hemodynamically significant effusion loculated posterior to the ventricle and that it could serve as a valuable diagnostic feature of such regional cardiac tamponade.

That regional tamponade can be caused by a compartmentalized posterior pericardial effusion is supported by recent experimental observations by Fowler and his associates.22 In canine experiments, they demonstrated a reduction in cardiac output and stroke volume with regional left heart tamponade, although the reduction in cardiac output was of significantly greater magnitude with right-sided tamponade or with tamponade of both the right and left sides. Cardiac imaging was not used in their study, and consequently, the effect of regional tamponade on cardiac wall dynamics is not known. We believe that cardiac tamponade as seen in our patient population after cardiac surgery is different from the above-described canine preparation because most of our patients showed some evidence of adhesions of the right atrium and right ventricle to the posterior sternal wall, whereas in the experimental preparation of Fowler et al, the right ventricular and right atrial walls were free to expand without any external restriction. Anterior adhesions in patients could potentially limit the right heart expansion. This, coupled to some possible postoperative impairment of right heart function, could lead to significant hemodynamic compromise when LVDC also occurs because of a tense posterior pericardial effusion. If fluid accumulates behind the left atrium as well, LAC can also occur. LAC was noted in three of our patients in whom the effusion was present behind the left atrium. In most of our patients, the pericardial effusion did not surround the left atrium, and LAC was not seen.

**Critique of Our Study**

Our experience indicates that LVDC is commonly associated with posteriorly loculated pericardial effusion causing regional tamponade in postoperative patients. However, our study is a retrospective investigation in a selected patient population. Therefore, information on the precise frequency of LVDC, its sensitivity, specificity, and predictive accuracy as a diagnostic sign of cardiac tamponade cannot be determined from this investigation. Our study reveals that RVDC, RAC, and LAC are unreliable markers of regional cardiac tamponade. We were also not able to ascertain the usefulness of Doppler analysis of flow velocity recordings in this circumstance because Doppler recordings were not systematically acquired in our patients. Furthermore, whether LVDC occurs only in the setting of posterior regional tamponade or whether it can occur in other types of tamponade cannot be determined from this study. Previous investigations of circumferential tamponade have not highlighted this sign, but it is conceivable that the finding may not have been carefully looked for. An interesting case report of tamponade in a patient with primary pulmonary hypertension and right ventricular hypertrophy by Frey and associates20 suggests that LVDC may occur as the sole echocardiographic marker of tamponade due to a circumferential pericardial effusion. In their patient, LVDC was present but RVDC and RAC were absent. Prospective investigations of patient groups with tamponade resulting from regional and circumferential pericardial effusions are necessary to accurately define the value of LVDC as a diagnostic sign. Because of the lack of simultaneous left ventricular and intrapericardial pressure recordings, our observations do not allow us to characterize the exact mechanism behind LVDC. A prospective study with detailed intrapericardial and intracavitary hemodynamic measurements with simultaneous echocardiographic imaging before, during, and after withdrawal of pericardial fluid is required to clarify the pathophysiological basis for LVDC.

In this study, we did not correlate the frequency of the regional cardiac wall changes, namely the RAC, RVDC, LAC, and LVDC, with a multitude of other M-mode and two-dimensional echocardiographic findings previously described in the setting of cardiac tamponade such as changes in mitral valve motion, leftward septal shift with inspiration, and cyclical respiratory variations in ventricular dimensions.23 Although these abnormalities were reported in the early 1970s, subsequent work and clinical experience have documented the insensitivity, nonspecificity, and the lack of clinical usefulness of these findings in the diagnosis of tamponade. On the other hand, RVDC and RAC have emerged as useful markers, and hence, we correlated the presence of LVDC in our study primarily with these findings.
Clinical and Research Implications

Despite the drawbacks associated with the retrospective nature of our investigation, our observations demonstrate that patients who have had cardiac surgery can develop cardiac tamponade because of a loculated regional pericardial effusion, and that LVDC is a common accompaniment of this type of regional tamponade. Because the conventional diagnostic criteria for tamponade may not always be present in these patients, the paucity of classic clinical or echocardiographic signs could give a false sense of security if tamponade is not recognized. In this setting, the presence of LVDC could suggest the presence of hemodynamically significant pericardial effusion. Therefore, if tamponade is even remotely suspected in patients who have had cardiac surgery, echocardiography should be performed not only to detect and localize a pericardial effusion, but also to determine whether LVDC is present. This sign could conceivably also be useful in tamponade occurring in other clinical settings such as those due to traumatic or malignant pericardial effusion and in tamponade occurring in patients in whom right-sided findings may be absent because of pulmonary hypertension or right ventricular hypertrophy. Our study stresses the need for further research in regional cardiac tamponade caused by loculated pericardial effusions, both in patients and in experimental animal models that simulate the human scenario realistically. We believe that our investigation also raises questions about the clinical and hemodynamic criteria used for the diagnosis of cardiac tamponade in patients with various cardiac diseases that by themselves may alter the intracardiac hemodynamics. Studies exploring these issues could further our understanding of the pathophysiology of cardiac tamponade.

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References


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