Mitral and Tricuspid Annular Velocities Before and After Pericardiectomy in Patients With Constrictive Pericarditis

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Background—Previous studies have demonstrated that mitral annulus early diastolic (e') velocity is increased in constrictive pericarditis (CP) and reduced in restrictive cardiomyopathy. However, those studies did not comprehensively evaluate mitral and tricuspid annular velocities before and after pericardiectomy.

Methods and Results—We performed comprehensive echocardiography before and after pericardiectomy in 99 patients with CP, 52 with primary (idiopathic or postpericarditis etiology) and 47 with secondary CP (due to surgery or radiation). Overall, mean±SD mitral medial, mitral lateral, and tricuspid lateral e’ velocities were 12.2±4.2, 10.0±5.4, and 11.6±3.5 cm/s, respectively; annular late diastolic velocities were 10.3±4.3, 12.2±4.9, and 11.7±5.4 cm/s, respectively; and annular systolic (s’) velocities were 7.8±2.8, 8.2±2.1, and 11.2±3.8 cm/s, respectively. Medial e’ was equal to or greater than mitral lateral e’ in 74% of analyzable cases. With the exception of tricuspid s’, there were significant differences in all s’ and e’ velocities between primary and secondary CP before pericardiectomy. After pericardiectomy, all annular velocities decreased significantly (P<0.02 for all comparisons). The reduction in medial e’ velocity was greater than that of mitral lateral e’ velocity (P<0.0001 and P=0.0004, respectively), and the mitral lateral/medial e’ ratio normalized (P=0.0002).

Conclusions—The mitral lateral/medial e’ ratio is reversed in three fourths of patients with CP. All annular velocities are lower in secondary compared to primary CP before pericardiectomy. After pericardiectomy, there is reduction of all annular velocities and normalization of the mitral lateral/medial e’ ratio. (Circ Cardiovasc Imaging. 2011; 4:399-407.)

Key Words: pericardium ■ echocardiography ■ pericarditis

Constrictive pericarditis (CP) is a disabling consequence of chronic inflammation and thickening of the pericardium. The encasement of the heart by a rigid, noncompliant pericardium results in characteristic hemodynamic consequences, including impaired diastolic filling of the ventricles, exaggerated ventricular interdependence, and dissociation of intracardiac and intrathoracic pressure changes during respiration.1,2 Pericardiectomy usually is the only accepted curative treatment for CP, and several studies have shown its efficacy in improving symptoms.3,4 CP frequently is difficult to diagnose, even after comprehensive evaluation, and often not considered in the differential diagnosis of patients with heart failure. Echocardiography has made a significant contribution to the accurate diagnosis of CP, being capable of demonstrating characteristic 2D features and respirophasic changes in Doppler echocardiographic flow velocities.

Clinical Perspective on p 407

Tissue Doppler imaging (TDI) has further facilitated detection of CP. TDI can be used to measure mitral and tricuspid annular motions, which reflect ventricular systolic and diastolic motions in the long axis. In CP, mitral medial early diastolic (e’) velocity is preserved or even increased5–10 because of the limitation of lateral expansion by the constricting pericardium, and mitral lateral e’ velocity tends to be lower than medial e’ velocity, which is a reversal of their normal relationship.11 This mitral annular velocity pattern is relatively specific for CP in patients with heart failure because e’ velocity usually is reduced in patients with myocardial disease whether left ventricular ejection fraction (LVEF) is preserved or reduced. However, there are no substantive data on mitral annulus systolic velocity and tricuspid annulus velocity in CP. The effect of pericardiectomy on mitral and tricuspid annular velocities, which may

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provide further insight into the mechanism of annulus motion in CP, is unknown. The aim of this study was to assess these annular velocity changes in patients with CP who underwent pericardiectomy.

**Methods**

From January 2006 through September 2008, 183 patients underwent pericardiectomy at the Mayo Clinic (Rochester, MN). We excluded 41 patients who had pericardiectomy due to recurrent or relapsing pericarditis but no evidence of CP. Also excluded were 43 patients who had concomitant valve surgery or coronary artery bypass grafting at the time of pericardiectomy. Hence, the study population consisted of 99 patients (72 men and 27 women; mean age, 58 ± 15 years) with surgically proven CP who had comprehensive echocardiographic examination before and after pericardiectomy. The study was approved by the Institutional Review Board. Because concomitant myocardial disease can affect annulus velocities, we divided the patients into 2 groups on the basis of the underlying etiology of CP (primary CP [idiopathic, postpericarditis, viral etiology], n = 52; secondary CP [due to surgery or radiation], n = 47). The clinical profile and echocardiographic findings for both groups were compared before and after pericardiectomy.

**Echocardiographic Examination**

All patients had comprehensive evaluation with M-mode, 2D, and pulsed Doppler echocardiography with a respirometer recording and TDI before and after pericardiectomy. Figures 1 to 3 show examples of mitral and tricuspid annular velocities before and after pericardiectomy. The clinical profile and echocardiographic findings for both groups were compared before and after pericardiectomy.

![Figure 1](image1.png)

**Figure 1.** A, Tissue Doppler imaging (TDI) at the medial annulus in a patient with constrictive pericarditis before pericardiectomy. All subsequent figures are from the same patient. Medial systolic annulus velocity (s') was 0.09 m/s; early diastolic annulus velocity (e'), 0.14 m/s; and late diastolic annulus velocity (a'), 0.11 m/s. B, TDI at the medial annulus after pericardiectomy. Medial s' was 0.08 m/s; e', 0.07 m/s, and a', 0.1 m/s.
measured: peak velocities of early (E) and late (A) filling and E-wave deceleration time. Peak annular velocities were measured from the apical 4-chamber view at systole (s’), early diastole (e’), and late diastole (a’) with a 2- to 5-mm tissue Doppler sample volume placed at the septal corner and at the mitral and tricuspid lateral annuli. In patients with atrial fibrillation, 5 consecutive signals were measured and averaged.

**Diagnosis of CP**
Clinical, hemodynamic, and echocardiographic findings were considered, but the final diagnosis of CP was confirmed at the time of surgery in all study patients.

**Operative Details**
Pericardiectomy was performed through a sternotomy or left thoracotomy incision. The standard pericardial resection at our institution is comprehensive pericardiectomy, with removal of the diaphragmatic component, anterior pericardium from phrenic nerve to phrenic nerve, and posterior pericardium to the left phrenic nerve. In most patients, radical pericardiectomy was performed, but where this was not achievable, as much pericardium was resected as possible. Visceral pericardium also was removed as required.

**Statistical Analysis**
Descriptive data are reported as mean±SD or count (percent), as appropriate. The Shapiro-Wilk test was used to check the normality of the outcome distributions. Paired t tests (or Wilcoxon signed rank tests for nonnormal data) were used to assess the echocardiographic parameters before and after pericardiectomy. In addition, comparisons between 2 groups were done with the t test (or Wilcoxon rank sum test for nonnormal data). Differences were considered statistically significant at P<0.05. For all outcomes, Spearman correlations were computed. All analyses

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*Figure 2. A, TDI at the lateral mitral annulus before pericardiectomy. Lateral s’ was 0.11 m/s; e’, 0.13 m/s; and a’, 0.16 m/s. B, TDI at the lateral mitral annulus after pericardiectomy. Lateral s’ was 0.9 m/s; e’, 0.1 m/s; and a’, 0.13 m/s. Abbreviations as in Figure 1.*
were performed using JMP version 8 (SAS Institute Inc; Cary, NC) statistical software.

Results

Patient Characteristics
Of the 99 patients, CP was secondary to previous cardiac surgery in 34 (34.4%), previous radiation therapy in 13 (13.1%), and other causes (postpericarditis, autoimmune disease, etc) in 19 (19.2%) and idiopathic in 33 (33.3%). Mean patient age was 58±15 years; 72 (73%) were men. Patients in the secondary CP group were older (61±13 versus 53±16 years, \( P=0.0053 \)). Sinus rhythm was present in 83% of patients with primary CP and 83% of those with secondary CP. Body mass index was 28.5±5 versus 27.5±5 kg/m², and body surface area was 1.99±0.3 versus 1.98±0.2 m².

Two-Dimensional and Doppler Echocardiography
Follow-up echocardiograms were obtained 51±131 days (range, 0 to 778 days) after pericardiectomy. Table 1 lists 2D and Doppler echocardiographic data before and after pericardiectomy. Before pericardiectomy, atrial enlargement was reported in 26 (50%) patients with primary and 32 (68%) with secondary CP. Apart from mitral A velocity, there were no significant differences between the 2 subgroups before pericardiectomy. After pericardiectomy, only LV end-diastolic dimension was significantly different between these subgroups.

Before pericardiectomy, RV function was normal or mildly decreased in 95% of patients and moderately to severely decreased in 5%. After surgery, these proportions were 81% and 19%, respectively.
Tissue Doppler Imaging

Table 2 shows the TDI data points available for analysis, and Table 3 shows their overall mean values. Among analyzable patients, a medial e’ velocity ≥8 cm/s was present before pericardiectomy in 86% of the entire group, and in 100% of patients with primary and 75% with secondary CP. Between primary and secondary CP groups, there were significant differences in e’ velocities except at the tricuspid annulus (Table 4). In both primary and secondary CP groups, early annular diastolic velocities decreased significantly after pericardiectomy, whether medial e’ (P<0.0001 and P=0.0004, respectively), mitral lateral e’ (P=0.022 and P=0.013, respectively), or tricuspid lateral e’ (P=0.0005 and P=0.0028, respectively) (Table 4). Overall, the reduction in medial e’ was somewhat more significant than mitral lateral e’ velocity (P<0.0001 and P=0.0004, respectively) (Table 3).

Comparing radiation and postcardiectomy patients within the secondary CP group, there were no significant differences in tissue velocity characteristics before or after pericardiectomy. In both groups, medial and mitral lateral e’ velocities were lower after surgery. However, the decline in e’ appeared more significant in the radiation compared to the cardiac surgery group (P=0.0092 versus P=0.0383), consistent with greater myocardial disease in the former.

Table 2. Frequency of Baseline (Prepericardiectomy) and Matched Prepericardiectomy and Postpericardiectomy Tissue Velocity Data in 99 Patients With Constrictive Pericarditis

<table>
<thead>
<tr>
<th></th>
<th>Prepericardiectomy</th>
<th>Matched Prepericardiectomy and Postpericardiectomy Data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>s’</td>
<td>e’</td>
</tr>
<tr>
<td>Medial</td>
<td>92</td>
<td>92</td>
</tr>
<tr>
<td>Mitral lateral</td>
<td>48</td>
<td>49</td>
</tr>
<tr>
<td>Tricuspid lateral</td>
<td>39</td>
<td>40</td>
</tr>
</tbody>
</table>

The fewer a’ values reflect the occurrence of atrial fibrillation. a’ indicates late annulus diastolic velocity; e’, early diastolic annulus velocity; E’, early diastolic mitral annular velocity; s’, systolic annulus velocity.

Table 5 shows the E/e’ and mitral lateral/medial e’ ratios before and after pericardiectomy. The mitral lateral/medial e’ velocity ratio was reversed (ie, <1) in 74% of all analyzable cases before pericardiectomy. This reversed ratio prevailed equally in patients with primary (74%) and secondary (75%) CP and increased in both groups after pericardiectomy (P=0.0002 for the entire CP group). Notably, among patients with primary CP, this relationship changed from an overall reversed ratio to normal after surgery (P=0.0006).

There were significant differences in all s’ velocities between the subgroups before pericardiectomy. After pericardiectomy, only lateral s’ was lower in the secondary group (Table 4). Moderate to strong correlations were observed between s’ and e’ as well as between s’ and a’ velocities before pericardiectomy (Table 6). These correlations were generally weaker after pericardiectomy.

Regional wall motion abnormalities were present in 3 (6%) and 9 (19%) patients with primary and secondary CP, respectively. No significant changes occurred in annular velocities after surgery in these patients probably because of their small number or lower baseline annular velocities. Conversely, among the patients without wall motion abnormalities, all annulus velocities decreased significantly (P<0.03 for all medial, mitral lateral, and tricuspid lateral velocities).

Postoperative echocardiography showed persistent features of CP (defined as ≥2 of the following: mitral E respiratory variation, hepatic vein diastolic flow reversals with expiration, and inferior vena cava plethora) in 7 patients. At the median annulus where postoperative data were available in all cases, s’, e’, and a’ were not significantly different between these patients and the 92 without persistent constrictive hemodynamics (all P>0.35).

Discussion

Garcia et al® were the first to report that the measurement of longitudinal axis expansion by TDI provided a clinically useful distinction between CP and restrictive cardiomyopa-
Thy. Subsequently, Rajagopalan et al\(^6\) showed that a peak e’ velocity \(\geq 8\) cm/s could discriminate between these entities with high sensitivity and specificity. We have confirmed that medial e’ velocity is relatively normal or even accentuated in patients with CP, irrespective of the presence or absence of characteristic respiratory variation in mitral E velocity.\(^7\) Other investigators\(^8,9\) recommended the routine measurement of e’ in the evaluation of heart failure and suspected CP, albeit with interpretative caution in the setting of myocardial disease, patchy fibrosis, or extensive annular calcification.\(^14–16\) Our group first described a reversal of the normal relationship of mitral lateral e’ velocity is lower than medial e’ velocity) and inverted lateral/medial e’ ratio.\(^11\) Few data exist on postoperative changes in TDI. Kim et al\(^10\) found in 16 patients that medial e’ decreased significantly after pericardiectomy. Building on these observations from a small number of patients, our aim was to provide a comprehensive evaluation of TDI at both mitral and tricuspid annuli in a larger number of patients and to follow their evolution after pericardial resection.

### Early Diastolic Annulus Velocity

We confirmed the presence of *annulus reversus* in patients with CP. Based on the hypothesis that lateral annulus motion is restricted by the constricting pericardium and that medial annulus diastolic motion increases in compensation, it may be anticipated that medial mitral annulus velocity decreases and lateral annulus velocity increases after pericardiectomy and that the mitral lateral/medial e’ ratio normalizes. Although the latter was confirmed, both medial and mitral lateral e’ velocities were found to decrease after pericardiectomy. Several mechanisms may explain these observations. Pericardiectomy removes constraint to lateral expansion of the annuli and nullifies the exaggerated longitudinal motion of the mitral annulus as well as the translational component of lateral e’ velocity related to increased medial excursion. In some patients, low annular velocities unmasked by pericardiectomy may reflect underlying myocardial damage or atrophy secondary to long-standing encasement and penetration of the myocardium by calcium spurs,\(^17\) persistent inflammation, and because the pericardium might be firmly adherent to the myocardium, additional injury at the time of surgery.

We also found reduced tricuspid lateral e’ velocity after pericardiectomy. The aforementioned mechanisms operative at the mitral annuli apply, but we note relevant and frequent association of CP with significant tricuspid regurgitation and, in some cases, further RV expansion and worsening of regurgitation following pericardiectomy.\(^18\)

All mitral annular velocities were higher in patients with primary CP than in those with secondary CP, an observation that can be explained by concomitant myocardial disease due to radiation or ischemic heart disease. A medial e’ value \(\geq 8\) cm/s was shown by Ha et al\(^7\) to differentiate CP from restrictive cardiomyopathy with a sensitivity and specificity of \(\geq 95\%\). In the present larger and more diverse patient pool,

### Table 3. Overall Mean Long-Axis Annular Velocities Before and After Pericardiectomy

<table>
<thead>
<tr>
<th></th>
<th>Before Pericardiectomy, cm/s</th>
<th>After Pericardiectomy, cm/s</th>
<th>(P^*)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>s’</td>
<td>e’</td>
<td>a’</td>
</tr>
<tr>
<td>Medial</td>
<td>7.0 (6.0, 9.0)</td>
<td>12.2</td>
<td>8.0 (7.0, 11.5)</td>
</tr>
<tr>
<td>Mitral lateral</td>
<td>8.0 (6.3, 9.8)</td>
<td>11.9</td>
<td>10.0</td>
</tr>
<tr>
<td>Tricuspid lateral</td>
<td>12.1</td>
<td>13.1</td>
<td>13.8</td>
</tr>
</tbody>
</table>

Nonnormally distributed data are presented median (25th percentile, 75th percentile). Abbreviations as in Table 2.

*Comparing before and after pericardiectomy.

### Table 4. Long-Axis Systolic, Early Diastolic, and Late Diastolic Annular Velocities Before and After Pericardiectomy

<table>
<thead>
<tr>
<th></th>
<th>Before Pericardiectomy, cm/s</th>
<th>After Pericardiectomy, cm/s</th>
<th>(P^*)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All Patients</td>
<td>Primary CP</td>
<td>Secondary CP</td>
</tr>
<tr>
<td>Medial s’</td>
<td>7.0 (6.0, 9.0)</td>
<td>9.0 (7.0, 10.0)</td>
<td>6.0 (5.0, 8.0)</td>
</tr>
<tr>
<td>Mitral lateral s’</td>
<td>8.0 (6.3, 9.8)</td>
<td>9.1 ± 2.2</td>
<td>7.0 (6.0, 9.0)</td>
</tr>
<tr>
<td>Tricuspid lateral s’</td>
<td>11.2 ± 3.8</td>
<td>12.4 ± 3.1</td>
<td>9.7 ± 4.0</td>
</tr>
<tr>
<td>Medial e’</td>
<td>12.2 ± 4.2</td>
<td>14.6 ± 3.4</td>
<td>10.3 ± 3.5</td>
</tr>
<tr>
<td>Mitral lateral e’</td>
<td>11.6 ± 3.5</td>
<td>12.8 ± 3.8</td>
<td>10.3 ± 2.8</td>
</tr>
<tr>
<td>Tricuspid lateral e’</td>
<td>12.2 ± 4.9</td>
<td>13.1 ± 4.8</td>
<td>11.2 ± 4.8</td>
</tr>
<tr>
<td>Medial a’</td>
<td>8.0 (7.0, 11.5)</td>
<td>10.0 (7.0, 13.0)</td>
<td>8.0 (7.0, 10.0)</td>
</tr>
<tr>
<td>Mitral lateral a’</td>
<td>10.3 ± 4.3</td>
<td>10.5 ± 4.9</td>
<td>10.1 ± 3.5</td>
</tr>
<tr>
<td>Tricuspid lateral a’</td>
<td>11.7 ± 5.4</td>
<td>13.1 ± 5.7</td>
<td>9.6 ± 4.5</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD or median (25th percentile, 75th percentile). Abbreviations as in Tables 1 and 2.

*Comparing primary and secondary CP.
†Comparing prepericardiectomy and postpericardiectomy values in primary CP.
‡Comparing prepericardiectomy and postpericardiectomy values in secondary CP.
including a large secondary subgroup, a medial e’ cutoff <8 cm/s had a lower negative predictive value.

Systolic Annulus Velocity

The s’ by TDI reflects the peak velocity of myocardial fiber shortening in the longitudinal direction and provides a more sensitive assessment of global LV systolic function than 2D or M-mode imaging. The s’ has been correlated with peak positive dP/dt and LVEF in patients with dilated cardiomyopathy, hypertensive heart disease, and myocardial infarction. There is little information on mitral and tricuspid s’ velocities in patients with CP. Studies in very small patient populations compared either s’ velocity between CP and restrictive cardiomyopathy or changes in s’ before or after pericardiectomy.

The mean s’ velocity in patients with secondary CP was lower both before and after pericardiectomy than published normative values and lower, especially prepericardiectomy, than in patients with primary CP. These observations concord with coexisting myocardial disease in this subgroup. In both CP subgroups, all s’ velocities decreased after pericardiectomy, consistent with previous smaller studies. This finding seems counterintuitive because s’ velocity is expected to increase with augmented stroke volume after pericardiectomy. To explain this paradoxical relationship between s’ and stroke volume in CP, we postulate that systolic and diastolic motion of the mitral annulus are closely coupled, in part, through elastic recoil mechanisms. Thus, in the prepericardiectomy setting, both longitudinal systolic and diastolic motion of the annulus are exaggerated, whereas after release of pericardial constraint, both decrease in tandem. This hypothesis is supported by the moderate to high correlation between annular s’ and e’ as well as s’ and a’, especially before pericardiectomy when restorative forces may be most operative.

There appeared to be proportionately greater postoperative reduction in tricuspid lateral or RV s’ and e’ compared to mitral annulus values. Because the pericardial process often is asymmetrical, being most pronounced over the RV, annular motion here may be expected to be most exaggerated before pericardiectomy and after decortication, approximate normality. However, the disproportionate reduction in tricuspid lateral s’ and e’ probably also stems from postoperative RV dysfunction, which was moderate to severe in 1 in 5 patients.

LVEF did not change despite the expected increase in stroke volume after pericardiectomy. We postulate that after pericardial resection, LV filling increases and other elements of LV shortening, including torsion, are recruitable, contributing to better cardiac output and compensating for abnormal longitudinal function. Sengupta et al found higher net twist but no significant increase in torsion postpericardiectomy, a conclusion limited by small patient numbers and early timing of the postoperative studies when restoration of function may have been incomplete. To confirm our hypothesis, detailed analysis of myocardial mechanics in a larger number of patients before and after pericardiectomy will be required.

A subset of patients with CP continue to experience heart failure symptoms after pericardiectomy, 1 cause being residual constriction. In the present study, we did not find any significant difference in annulus velocities between patients with and without postoperative constrictive hemodynamics. The utility of TDI in identifying residual CP should be further assessed in larger cohorts with clinical outcomes correlation.

Table 5. E/e’ Ratios and Mitral Lateral/Medial e’ Ratios Before and After Pericardiectomy

| Table 5. E/e’ Ratios and Mitral Lateral/Medial e’ Ratios Before and After Pericardiectomy |
|------------------|------------------|------------------|------------------|------------------|
|                   | Before Pericardiectomy | After Pericardiectomy |
|                   | All Patients | Primary CP | Secondary CP | P* |
| E/medial e’       | 7.5 (4.8, 10) | 6.7 ± 4 | 8.9 (6.4, 14.0) | 0.0008 |
| E/mitral lateral e’ | 8.3 ± 4 | 7.5 ± 4 | 9.4 ± 5 | 0.12 |
| E/tricuspid lateral e’ | 8.0 (6.0, 10.0) | 7.1 (5.0, 9.0) | 9.2 (6.9, 16.5) | 0.048 |
| Mitral lateral/medial e’ | 0.93 (0.78, 1.07) | 0.9 ± 0.2 | 0.94 (0.85, 1.2) | 0.52 |

Data are presented as mean ± SD or median (25th percentile, 75th percentile). Abbreviations as in Tables 1 and 2.

Table 6. Spearman Correlation of Annular Velocities Before and After Pericardiectomy

| Table 6. Spearman Correlation of Annular Velocities Before and After Pericardiectomy |
|------------------|------------------|------------------|------------------|------------------|
|                   | Before Pericardiectomy, cm/s | After Pericardiectomy, cm/s |
|                   | s’ and e’ | s’ and a’ | s’ and e’ | s’ and a’ |
| Medial            | 0.62 (<0.0001) | 0.66 (<0.0001) | 0.34 (0.001) | 0.38 (0.001) |
| Mitral lateral    | 0.49 (0.0004) | 0.41 (0.007) | 0.48 (0.0007) | 0.44 (0.005) |
| Tricuspid lateral | 0.34 (0.037) | 0.81 (<0.0001) | 0.27 (0.19) | 0.45 (0.034) |

Data presented as ρ (P value). Abbreviations as in Table 2.

Limitations

The present study was retrospective and has the inherent limitations of this design. Notwithstanding, these observations of CP on TDI represent, to our knowledge, the largest data set published to date.

We only recorded TDI of longitudinal axis motion in the 4-chamber view. Because of the local tethering effect, analysis of multiple annular regions could have provided additional helpful data. It would also be useful to characterize radial and circumferential function for a better understanding of the mechanics of the unique annulus motion in CP and the effects of pericardiectomy. Because not all patients had mitral
lateral and tricuspid lateral TDI recordings, certain conclusions were drawn from subsets with comparable data. Although this limits power, we do not envisage specific biases in the selective analysis.

The comparison of TDI in patients with primary and secondary CP is confounded by the older age of the latter group. Age influences both systolic and diastolic myocardial velocities arising from differences in myocyte integrity, fibrosis, and β-adrenergic receptor density. However, normal TDI reference values indicate that within the 1-decade difference in age between patients with primary and those with secondary CP, a percentage difference between 0% (for medial e') and 13% (for lateral e') can be expected. This contrasts with the observed 19% to 30% difference in TDI values between these groups prepericardiectomy and from 6% to 32% postpericardiectomy, suggesting a relatively minor influence of age compared with CP etiology.

**Clinical Implications**

In patients with heart failure and normal LVEF, preserved or increased medial e’ velocity strongly suggests CP. The diagnosis is further supported if medial e’ is higher than mitral lateral e’. With a mitral inflow profile of increased filling pressure and expiratory hepatic vein diastolic reversals as well as abnormal ventricular septal motion, CP can be diagnosed by echocardiography. This characteristic pattern of annulus velocities reverts to normal after pericardiectomy. It will be of clinical interest to investigate whether the extent of postoperative change in annulus velocities can predict clinical outcome after pericardiectomy.

**Conclusions**

This study demonstrates that in patients with CP, (1) medial e’ velocity usually is higher than mitral lateral e’ velocity, which is a reversal of the observed relationship in normal individuals and patients with restrictive cardiomyopathy; (2) all mitral and tricuspid annular velocities (e’, a’, and s’) are higher in primary CP compared to secondary CP; and (3) all mitral and tricuspid annular velocities decrease after pericardiectomy with normalization of the mitral lateral/medial e’ velocity ratio.

**Disclosures**

None.

**References**


The diagnosis of constrictive pericarditis often is challenging even after multiple diagnostic tests. Tissue Doppler imaging of the mitral annulus has facilitated the identification of constriction, which potentially is curable by pericardiectomy. Preserved or augmented medial annulus early diastolic velocity in a patient with heart failure and normal ejection fraction points to the diagnosis of constriction. However, this finding is not that helpful in young patients who normally have preserved early diastolic mitral annulus velocity. The additional finding of lower mitral lateral annulus early diastolic velocity compared with the medial annulus increases confidence in the diagnosis of constriction. Although normal or increased mitral medial annulus velocity strongly suggests the diagnosis of constriction, it may be reduced if there is superimposed myocardial disease. Even in this situation, the mitral lateral annulus velocity usually is lower than that at the medial annulus. The fact that annular velocities return to lower values after pericardiectomy confirms that the characteristic annulus velocity pattern observed is a product of constrictive pericardium. We need to take advantage of this simple measurement when evaluating a patient with heart failure and normal left ventricular ejection fraction.
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