The clinical recognition of constrictive pericarditis (CP) is important but challenging. In addition to Doppler echocardiography, newer echocardiographic techniques for deciphering myocardial deformation have facilitated the noninvasive recognition of CP and its differentiation from restrictive cardiomyopathy. In a patient with heart failure and a normal ejection fraction, echocardiographic demonstration of exaggerated interventricular interdependence, relatively preserved left ventricular longitudinal deformation, and attenuated circumferential deformation is diagnostic of CP. This review is a concise update on the pathophysiology and hemodynamic features of CP, the transmural and torsional mechanics of CP, and the merits and pitfalls of the various echocardiographic techniques used in the diagnosis of CP. (J Am Soc Echocardiogr 2009;22:24-33.)

**Keywords:** Constrictive pericarditis, Echocardiography, Doppler tissue imaging, Restrictive cardiomyopathy, Left ventricular deformation

Constrictive pericarditis (CP) is characterized by impaired diastolic cardiac filling and elevated ventricular filling pressures (Figure 1) due to a rigid pericardium with fusion of the visceral and parietal layers. Patients present predominantly in heart failure with elevated jugular venous pressure, dyspnea, peripheral edema, hepatomegaly, and ascites. Pericarditis, cardiac surgery, and mediastinal irradiation are the leading identifiable causes of CP in the developed world. However, pericardial fibrosis and calcification are often idiopathic in origin. Pericardiectomy relieves pericardial restraint and, in the absence of concomitant myocardial dysfunction, effectively restores diastolic filling. CP due to cardiac surgery, especially coronary artery bypass grafting and mediastinal irradiation, can involve the pericardium and also the myocardium. Determining the relative contribution of pericardial restraint versus myocardial dysfunction in such patients remains a diagnostic challenge.

**PATHOPHYSIOLOGY OF CONSTRICTIVE PERICARDITIS**

In CP, the visceral pericardium and the parietal pericardium are fibrosed and fused together, although not necessarily always thickened (Figure 2A). Calcification can result in the formation of pericardial calcium plaques (eggshell appearance), which may penetrate into the myocardium. The incidence and prevalence of pericardial calcification is dependent on the underlying cause of CP and is less commonly encountered in developing nations as the incidence of tuberculous pericarditis has declined. A recent series of 136 patients reported radiographic evidence of pericardial calcification in approximately one third of patients. The myocardium is usually normal in CP, but concomitant myocardial involvement may be seen, for example, following mediastinal irradiation. Epicardial involvement may also result from subpericardial mycarditis.

Characteristic hemodynamic changes in CP are attributed to isolation of the cardiac chambers from intrathoracic respiratory pressure changes and a fixed end-diastolic ventricular volume (Figure 2B). Although normally, the pressure gradient between the pulmonary veins and the left atrium and left ventricle is relatively constant during respiration, this pressure gradient decreases with inspiration in CP, leading to reduced diastolic pulmonary vein flow and left atrial and left ventricular (LV) filling (Figure 1). The rigid, noncompliant fibrous pericardial sac couples both ventricles. Consequently, an increase in filling on one side of the heart impedes contralateral filling through the motion of the interventricular septum (IVS), thereby making both ventricles markedly interdependent. The inspiratory reduction in LV filling is therefore associated with a simultaneous increase in right ventricular (RV) diastolic filling and an IVS shift toward the left chamber. The opposite physiologic effects on filling gradients and IVS shift are seen in expiration, with increased pulmonary venous pressure and an attendant increase in LV diastolic inflow. This is associated with simultaneous decrease in right-sided filling. Because of elevated atrial pressures, ventricular filling is very rapid and predominantly in early diastole, abruptly declining by middiastole. This hemodynamic pattern is reflected in the jugular venous and right atrial pressure waveforms as elevated mean filling pressure with a prominent “a” wave, a sharp “y” descent reflecting early diastolic resistance-free rapid RV filling, and as well as a preserved “x” descent due to accelerated atrial relaxation. The RV hemodynamic waveform with a dip-and-plateau, or square root, pattern reflects rapid ventricular relaxation, with a subsequent sharp increase in filling pressure as the expanding ventricle meets the pericardium (Figure 1A). At end-diastole, the end-diastolic right atrial, RV, wedged pulmonary
arterial, and LV pressures are equally elevated. These physiologic abnormalities that occur as a result of the ventricular interdependence and respiratory influenced changes are the fundamental processes that account for the clinical, hemodynamic, and echocardiographic findings of CP.

It is important to distinguish the hemodynamic features of CP from those of cardiac tamponade, a condition characterized by excess fluid in the pericardial sac that hinders diastolic filling. In cardiac tamponade, the intracardiac volumes are decreased on both sides of the heart, with elevation and equalization of diastolic filling pressures throughout the cardiac chambers, reflecting increased ventricular interactions between the right and left heart. Importantly, however, although early ventricular filling is resistance free in CP, the effusion in cardiac tamponade imposes pandiastolic resistance to ventricular filling.

Echocardiographic Diagnosis of Constrictive Pericarditis

The echocardiographic diagnosis of CP was originally based on M-mode echocardiographic findings and subsequently on 2-dimensional echocardiography and Doppler hemodynamics in response to the respiratory cycle. More recently, newer echocardiographic techniques, such as pulsed tissue Doppler, color Doppler tissue imaging (DTI), and speckle-tracking imaging, have been used to assess the unique changes in global and regional myocardial function seen in CP. However, while interpreting the role of various echocardiographic techniques and comparing individual studies, several facts must be kept in consideration: (1) CP is a heterogeneous disease, and study populations vary significantly regarding the distribution of underlying CP etiologies; (2) the confirmation of a firm diagnosis of CP is based on different “gold standards” (eg, surgical confirmation vs pericardial thickness on computed tomography); and (3) pericardial constriction may be localized, affecting the right ventricle more than the left ventricle or vice versa. Data on generalized versus localized pericardial constriction are sparse.

Doppler Hemodynamics

Mitral inflow as assessed by Doppler echocardiography demonstrates an increased early diastolic filling velocity followed by rapid deceleration, leading to a short filling period. Early mitral inflow deceleration time is usually, but not always, <160 ms. As first reported by Hatle et al and Oh et al in a larger cohort, dynamic changes with respiration occur in patients with CP (Figure 2D), but not in patients with restrictive cardiomyopathy (RCM; Figure 3B). Factors responsible for these respiratory driven changes are the dissociation of intrathoracic pressure from intracardiac pressure and enhanced ventricular interdependence. In CP, early diastolic mitral inflow is reduced with the onset of inspiration, and isovolumic relaxation time is prolonged. With expiration, mitral inflow returns to normal, and isovolumic relaxation time shortens. Typically, patients with CP demonstrate an increase in early diastolic mitral inflow velocity of ≥25% during expiration compared with inspiration. These changes can be seen with the first beat of inspiration, when a decrease in transmitral flow velocity is noted; the reverse occurs with expiration. Sensitivities and specificities of Doppler assessment of respiratory changes have been reported to be as high as 85% to 90%. Importantly, the percentage of respiratory change in patients with “mixed” constrictive and restrictive disease (eg, mediastinal radiation) is markedly lower. After complete pericardectomy mitral inflow patterns return to normal, and little respiratory variation is seen on Doppler echocardiography. Accordingly, Doppler assessment of respiratory variation has been reported to be useful for evaluating the outcome of pericardiectomy. Patients who had minimal respiratory variation after pericardiectomy were asymptomatic compared with those who continued to show respiratory variation.

Patients with RCM (Figure 3) and up to 20% of patients with CP may lack the typical respiratory changes in the presence of mixed constrictive-restrictive disease and/or markedly increased left atrial pressure. Typical respiratory changes in the latter situation may not be observed, because of mitral valve opening during the steep portion of the LV pressure curve, when respiration has little effect on
the transmitral pressure gradient. In these patients, maneuvers that decrease preload (head-up tilt or sitting) can unmask the characteristic respiratory variation in early mitral inflow velocity. Atrial fibrillation complicates the interpretation of respiratory variation of Doppler velocities, but respiratory variation can still be appreciated regardless of cardiac cycle length. Usually, this requires longer recording periods of Doppler tracings.

Phasic, respiratory changes in mitral inflow are not unique to CP, however. Patients with chronic obstructive pulmonary disease or severe RV dysfunction and large respiratory variations in intrathoracic pressure may also show inspiratory decreases in early mitral inflow velocities. Typically, these changes are more gradual and occur later in the respiratory cycle rather than during the first inspiratory beat. In such patients, a marked increase in inspiratory superior vena cava systolic forward flow can be helpful to rule out CP. In chronic obstructive pulmonary disease, there is a greater decrease in intrathoracic pressure in inspiration, which generates greater negative pressure changes in the thoracic cavity. This augments blood flow to the right atrium from the superior vena cava during inspiration.

Inferior vena cava dilation (Figure 2C) and pulsed wave Doppler of hepatic venous flow mirrors right atrial pressure. Pulsed Doppler recordings of hepatic vein flow in CP show marked diastolic flow reversal (Figure 2E), which increases in expiration compared with inspiration. In patients with advanced constriction or with mixed constrictive-restrictive physiology, it is not unusual to see significant diastolic flow reversals during both inspiration and expiration. In contrast, diastolic hepatic vein flow reversal is more prominent with inspiration in RCM.

Doppler evaluation of the pulmonary veins shows marked respiratory change in pulmonary venous flow in CP. The pulmonary venous systolic wave and early diastolic wave velocities, especially the early diastolic wave velocity, are increased during expiration and decreased during inspiration. The changes in pulmonary venous flow velocities have been reported to be more pronounced than changes in mitral inflow velocities. Similar respiratory variation can also be observed in patients with CP and atrial fibrillation. In contrast, patients with RCM show blunting of the systolic wave velocity and a decrease in the ratio of systolic to early diastolic wave velocity throughout the respiratory cycle, with a large atrial reversal wave and without any significant respiratory variation. (Table 1 summarizes sensitivities and specificities for hemodynamic CP Doppler findings.)

Color Doppler

Patients with CP typically demonstrate only mild mitral and tricuspid regurgitation. A pronounced increase in tricuspid regurgitant jet velocity from onset to peak inspiration can, however, help diagnose

Figure 2 Findings in patients with CP. (A) Computed tomography in a patient with CP shows the extent of pericardial thickening (red arrows). Apical 4-chamber view of the left ventricle (LV) (B) and dilated inferior vena cava (C) with increased respiratory variations in transmitral early diastolic flow (D) and hepatic venous Doppler flow (E) are shown for the same patient with CP (red arrows in D and E indicate respiratory-dependent change in Doppler flow). Exp, Expiration; Insp, inspiration.

Figure 3 Echocardiographic features in a patient with RCM due to cardiac amyloidosis. Apical 4-chamber view shows thick walled left ventricle (LV) (A) with a restrictive transmitral early diastolic flow pattern (B). Note the lack of increase in early diastolic transmitral velocity with the onset of expiration (Exp). Insp, Inspiration.
CP.29 Severe mitral and tricuspid regurgitation speak against isolated or predominant constrictive physiology and are markers for underlying myocardial disease. Color M-mode Doppler shows early-onset, elevated mitral inflow velocities in patients with CP. Rajagopalan et al30 reported sensitivity and specificity of 74% and 91%, respectively, to differentiate CP from RCM (mitral inflow slope /H11022.

LV Mechanics

Radial motion of the IVS. Abrupt anterior or posterior motion of the IVS in early diastole is common in patients with CP (Figure 4). The direction of early diastolic IVS motion may depend on a number of factors, including uneven distribution of fibrosis or calcification in the pericardial sac, the timing of mitral and tricuspid opening, the relative compliance of the right and left ventricles, and phase of respiration.31 In classic CP, the IVS shows a brisk, early diastolic motion toward the left ventricle during inspiration, followed by a rebound in the opposite direction during expiration.32 This septal bounce reflects exaggerated interventricular dependence combined with forceful early diastolic filling. Himelman et al13 reported in a large number of patients that an IVS bounce is the most consistent 2-dimensional echocardiographic sign for CP, with sensitivity of 62% and specificity of 93%. Recently, Sengupta et al34 assessed IVS motion by color DTI (Figure 4). In this study, the majority of patients with CP had high-velocity early diastolic biphasic IVS motion (>7 cm/s), a finding with 82.5% sensitivity and 92.7% specificity for CP (Table 1).

Radial motion of the LV posterior wall. In patients with CP, the LV posterior wall rapidly expands posteriorly during early diastole, followed by an abrupt cessation of such movement during middiastole and late diastole (Figure 4), which corresponds to the abrupt termination of rapid ventricular filling.35,36 This lack of motion, termed “flattening,” of the LV posterior wall during middiastole and late diastole can be best observed with M-mode echocardiography.37,38 Palka et al39 assessed relative subendocardial versus subepicardial LV posterior wall velocities using myocardial velocity gradient imaging. Patients with CP (n /H11005 10) had markedly faster moving subendocardium during rapid ventricular filling than normal subjects and patients with RCM (Table 1).

Longitudinal LV mechanics. The quantitative assessment of longitudinal mitral annular motion by pulsed tissue Doppler and color DTI estimates LV relaxation.40-42 In patients with CP, mechnoelastic properties of the myocardium are relatively preserved in the longitudinal direction, and therefore longitudinal deformation of the LV base (Figure 5) and longitudinal early diastolic velocities (Figure 6)

---

Table 1 Sensitivities and specificities of echocardiographic markers for CP

<table>
<thead>
<tr>
<th>Marker</th>
<th>Study</th>
<th>n</th>
<th>Secondary CP</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doppler echocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥25% respiratory variation of peak early diastolic MV inflow velocity; augmented hepatic vein diastolic flow reversals after the onset of expiration; ≥ 25% of forward diastolic velocity</td>
<td>Oh et al21</td>
<td>28</td>
<td>13</td>
<td>88%</td>
<td>67%</td>
</tr>
<tr>
<td>≥10% respiratory variation of peak early diastolic MV inflow velocity</td>
<td>Rajagopalan et al30</td>
<td>19</td>
<td>6</td>
<td>84%</td>
<td>91%</td>
</tr>
<tr>
<td>Color M-mode MV inflow propagation; first aliasing contour ≥ 100 cm/s</td>
<td>Rajagopalan et al30</td>
<td>19</td>
<td>6</td>
<td>74%</td>
<td>91%</td>
</tr>
<tr>
<td>Respiratory variation in PV systolic/diastolic flow ratio ≥ 65% in inspiration + % change of early mitral peak diastolic flow ≥ 40%</td>
<td>Klein et al27</td>
<td>14</td>
<td>10</td>
<td>86%</td>
<td>94%</td>
</tr>
<tr>
<td>Respiratory variation in PV peak diastolic flow velocity ≥ 18%</td>
<td>Rajagopalan et al30</td>
<td>19</td>
<td>6</td>
<td>79%</td>
<td>91%</td>
</tr>
<tr>
<td>Dilated hepatic veins, “W” wave pattern (reverse flow in late systole and diastasis)</td>
<td>von Bibra et al26</td>
<td>13</td>
<td>8</td>
<td>68%</td>
<td>100%</td>
</tr>
<tr>
<td>LV septal/posterior wall radial motion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M-mode</td>
<td>Engel et al31</td>
<td>40</td>
<td>NA</td>
<td>40%</td>
<td>80%</td>
</tr>
<tr>
<td>M-mode</td>
<td>Candell-Riera et al32</td>
<td>8</td>
<td>NA</td>
<td>88%</td>
<td>NA</td>
</tr>
<tr>
<td>2-dimensional</td>
<td>Himelman et al23</td>
<td>39</td>
<td>NA</td>
<td>62%</td>
<td>93%</td>
</tr>
<tr>
<td>Biphasic early diastolic IVS motion by color DTI (≥7 cm/s) motion</td>
<td>Sengupta et al34</td>
<td>40</td>
<td>30</td>
<td>82%</td>
<td>93%</td>
</tr>
<tr>
<td>Biphasic early diastolic IVS motion by pulsed tissue Doppler</td>
<td>Oki et al46</td>
<td>12</td>
<td>9</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>LV posterior wall flattening</td>
<td>Voelkel et al38</td>
<td>12</td>
<td>2</td>
<td>92%</td>
<td>100%</td>
</tr>
<tr>
<td>M-mode</td>
<td>Schnittger et al37</td>
<td>14</td>
<td>6</td>
<td>64%</td>
<td>90%</td>
</tr>
<tr>
<td>M-mode</td>
<td>Engel et al31</td>
<td>40</td>
<td>NA</td>
<td>85%</td>
<td>82%</td>
</tr>
<tr>
<td>Miscellaneous echocardiographic findings</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pericardial thickening</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M-mode</td>
<td>Engel et al31</td>
<td>40</td>
<td>NA</td>
<td>53%</td>
<td>100%</td>
</tr>
<tr>
<td>M-mode</td>
<td>Hütte et al18</td>
<td>7</td>
<td>4</td>
<td>100%</td>
<td>50%</td>
</tr>
<tr>
<td>2-dimensional</td>
<td>Oh et al21</td>
<td>28</td>
<td>19</td>
<td>36%</td>
<td>NA</td>
</tr>
<tr>
<td>Left atrial enlargement, M-mode</td>
<td>Engel et al31</td>
<td>40</td>
<td>NA</td>
<td>75%</td>
<td>100%</td>
</tr>
<tr>
<td>Premature PV opening, M-mode</td>
<td>Engel et al31</td>
<td>40</td>
<td>NA</td>
<td>14%</td>
<td>100%</td>
</tr>
</tbody>
</table>

MV, Mitral valve; NA, data not available; PV, pulmonary valve.
are either normal or exaggerated. Conversely, patients with RCM and intrinsic myocardial abnormalities have reduced longitudinal deformation of the LV base and reduced early diastolic longitudinal velocities. Pulsed tissue Doppler and color DTI have been shown to be helpful to diagnose CP. A lateral or septal early diastolic mitral annular velocity of $>$8 cm/s on pulsed tissue Doppler is in general the accepted cutoff value to distinguish patients with CP from those with RCM. Mitral annular velocities are particularly useful in CP when pronounced respiratory variations in peak early mitral inflow velocities are not seen. The additional use of pulsed tissue Doppler to hemodynamic Doppler and 2-dimensional and M-mode echocardiography increases predominantly the sensitivity of echocardiography.

Figure 4 Color-DTI for quantifying abnormal septal and posterior wall motion in CP. Characteristic findings on M-mode echocardiography (A) are septal notching (arrow 1) and the abrupt flattening of the posterior wall (arrow 2). Color M-mode echocardiography (B) shows high-velocity motion (arrow 3) in early diastole. Myocardial velocity tracings from the septum and posterior wall (C) identify components of abnormal septal motion in constriction. The posterior wall shows rapid early diastolic radial expansion (blue tracing), which corresponds to the abrupt outward motion seen on M-mode. The septum shows high-velocity early diastolic fluttering (yellow tracing), which corresponds to the septal notching seen on M-mode. $A_{m}$, Radial late diastolic velocity; $E_{m}$, radial early diastolic velocity; Exp, expiration; LV, left ventricle; RV, right ventricle; $S_{m}$, radial systolic velocity.

Figure 5 Longitudinal deformation of the left ventricle in CP and RCM. Longitudinal shortening strains were obtained by 2-dimensional speckle-tracking imaging. Speckle-tracking imaging is different from DTI in that strain data are angle independent. Myocardial deformation is computed from continuous frame-by-frame tracking of a small image block of “natural acoustic markers.” Tracking is based on searching the new location of the marker in the subsequent frame using a block-matching algorithm. The basal septal and basal lateral wall segments show normal longitudinal shortening strains in CP (A, B) and reduced longitudinal shortening strains in RCM (C, D). Apical longitudinal shortening strains are reduced in CP (B), whereas longitudinal strain at the LV apical septum is preserved in RCM (D).
phy to diagnose CP. Table 2 summarizes studies that have explored the diagnostic role of early diastolic mitral annular velocity in CP. Attention needs to be paid to the differing patient age groups (mitral annular velocities decrease with age) and different percentages of underlying CP etiologies, with varying patient numbers with presumed “mixed” constrictive and restrictive physiology. These factors may explain the spread of early mitral annular velocities and reported sensitivities and specificities.

When recording and interpreting early mitral annular velocities, caution is warranted in the setting of extensive mitral annular calcification, LV systolic dysfunction, or segmental nonuniform myocardial velocities. Averaging additional mitral annular sites is

Table 2 Studies assessing longitudinal early diastolic tissue velocities in CP

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Mean ± SD age (y)</th>
<th>Secondary CP</th>
<th>Mitral annular sample site</th>
<th>Comparison group</th>
<th>Cutoff value (cm/s)</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Garcia et al44</td>
<td>8</td>
<td>62 ± 13</td>
<td>6</td>
<td>Lateral*</td>
<td>CA (n = 4), nonspecific fibrosis (n = 1), DM/small-vessel disease (n = 1), cardiac transplantation (n = 1)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Rajagopalan et al50</td>
<td>19</td>
<td>56 ± 13</td>
<td>6</td>
<td>Lateral*</td>
<td>CA (n = 8), advanced HTN (n = 1), DM/small-vessel disease (n = 1), idiopathic (n = 1)</td>
<td>≥8</td>
<td>89%</td>
<td>100%</td>
</tr>
<tr>
<td>Ha et al45, Sohn et al48</td>
<td>23</td>
<td>59 ± 13</td>
<td>8</td>
<td>Septal*</td>
<td>CA (n = 38), RCM (n = 14) Normal (n = 35), Normal (n = 35), Normal (n = 35), Normal (n = 35)</td>
<td>≥8</td>
<td>95%</td>
<td>96%</td>
</tr>
<tr>
<td>Sengupta et al47</td>
<td>45</td>
<td>24 ± 12</td>
<td>30</td>
<td>Septal and lateral*</td>
<td>RCM (n = 10), CA (n = 1)</td>
<td>≥8</td>
<td>89%</td>
<td>73%</td>
</tr>
<tr>
<td>Choi et al43</td>
<td>17</td>
<td>55 ± 12</td>
<td>12</td>
<td>Septal*</td>
<td>RCM (n = 12), normal (n = 15)</td>
<td>&gt;8</td>
<td>70%</td>
<td>100%</td>
</tr>
<tr>
<td>Sengupta et al51</td>
<td>16</td>
<td>62 ± 13</td>
<td>9</td>
<td>Septal*</td>
<td>CA (n = 15)</td>
<td>&gt;6.6</td>
<td>93%</td>
<td>93%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Averaged from 4 corners of MV annulus†</td>
<td></td>
<td>&gt;5</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Sengupta et al57</td>
<td>26</td>
<td>56 ± 13</td>
<td>13</td>
<td>Septal and lateral</td>
<td>CA (n = 15), RCM (n = 4)</td>
<td>&gt;5</td>
<td>92%</td>
<td>90%</td>
</tr>
</tbody>
</table>

CA, Cardiac amyloid; DM, diabetes mellitus; HTN, hypertension; MV, mitral valve; NA, data not available.
*Pulsed tissue Doppler.
†Mean velocity by color DTI obtained in 11 of the 16 patients.
of incremental value to differentiate CP from RCM. Although patients with CP and RCM had overlapping values of early diastolic pulsed tissue Doppler velocities, clear separation of groups was evident with averaged color DTI early diastolic annular velocities (averaged from 4 walls). However, when using color DTI, one must note that resulting tissue velocities represent mean velocities, which are significantly lower compared with peak velocities derived on pulsed tissue Doppler. This is important because most CP studies and early diastolic mitral annulus cutoff values are based on pulsed tissue Doppler velocities (Table 2). Furthermore, in RCM, mitral annular velocities may not be representative if the disease process is heterogeneous, as reported previously in patients with endomyocardial fibrosis. Thus, hypokinetic areas accompanied by hyperkinesis in neighboring areas could result in relatively unaltered overall annular motion. This limits the ability of pulsed tissue Doppler to differentiate RCM because of endomyocardial fibrosis from CP.

**Circumferential LV mechanics.** Because of pericardial restraint and potential epicardial involvement, LV expansion in CP may be limited in the circumferential rather than the longitudinal direction. Accordingly, patients with CP were found to have reduced circumferential strain in combination with preserved longitudinal early diastolic velocities. This concept is further supported by the significant positive correlation of circumferential strain and radial displacement.

**Torsional LV mechanics.** By speckle-tracking imaging, Sengupta et al showed that patients with CP have significantly reduced net LV twist and torsion compared with normal subjects and patients with RCM. While the base and mid cardiac rotation was relatively preserved, apical rotation was markedly reduced, including the peak systolic and diastolic rotation rates (Figure 7). Early diastolic recoil, LV untwisting, and the magnitude of circumferential and longitudinal expansion of the left ventricle are modulated by pericardial tissue properties. A recent study showed that the congenital absence of the left pericardium results in reduced LV torsion. The reduction of apical rotation in CP could be due to a lack of normal pericardium, enabling swift and friction-free apical motion and potential scarring and inflammation extending into the epicardial layer of the myocardial wall. Pericardial tethering and/or diminished epicardial fiber function manifest as reduced basal, mid, and apical circumferential strain and also reduced longitudinal strain at the apex, which may be susceptible to additional endocardial involvement due to the close spatial relationship of epicardial and endocardial fibers. Table 3 summarizes LV mechanics in CP versus RCM.

![Figure 7](image.png)

**Figure 7** Twist mechanics of the left ventricle in CP and RCM. Short-axis cross-sectional views are divided into 6 segments, and 2-dimensional speckle-tracking imaging is used for computing LV rotation from 6 segments in each cross-sectional view. LV apical and basal rotation is averaged from 6 segments in each view, and the difference of apical and basal rotation provides the net twist angle of the left ventricle. LV apical rotation and net twist angles are reduced in CP (A). Similarly, apical early diastolic untwisting velocities obtained from 6 segments and the averaged value (dotted line) are also reduced in CP (B). Apical and basal LV rotation is normal in RCM with a normal net twist angle (C) and normal early diastolic apical untwisting velocities (D). $E_r$, Early diastolic apical untwisting velocity.

**Table 3** Myocardial mechanics in CP and RCM

<table>
<thead>
<tr>
<th>Deformation parameter</th>
<th>CP</th>
<th>RCM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal strain</td>
<td>Normal*</td>
<td>Decreased</td>
</tr>
<tr>
<td>Longitudinal early diastolic velocity</td>
<td>Normal or increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Circumferential strain</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Net twist angle</td>
<td>Decreased</td>
<td>Normal †</td>
</tr>
<tr>
<td>Apical untwisting velocity</td>
<td>Decreased</td>
<td>Normal †</td>
</tr>
</tbody>
</table>

*Except apical segments.
†Although normal in magnitude, early diastolic apical untwisting velocities may be delayed in timing.
ANCILLARY ECHOCARDIOGRAPHIC FINDINGS IN CONSTRUCTIVE PERICARDITIS

Pericardium
The detection of increased pericardial thickness may be of help in making a diagnosis of CP, but M-mode and 2-dimensional echocardiography show poor sensitivity and correlation with pathologic specimen measurements. Transesophageal echocardiography, on the other hand, because of superior resolution, showed high sensitivity (95%) and specificity (86%) to detect a pericardium ≥3 mm thick. Nevertheless, about 1 in 5 patients with CP have normal pericardium, and the isolated finding of thickened pericardium does not imply constrictive physiology.

Recently, Lu et al evaluated the relative motion of pericardium versus myocardium in patients with CP. By quantitative DTI, epicardial motion was found to be reduced, approaching that of the endocardium, whereas the endocardium was moving more vigorously. These findings are interesting because they seem to support the hypothesis that, whereas the endocardium is affected, the pericardium is not.

Cardiac Valves
A steep E-F slope on mitral valve M-mode tracing is suggestive of rapid early diastolic filling and can therefore be seen in CP. Premature opening of the pulmonary valve due to increased middiastolic RV pressure can be observed in patients with CP, if RV pressure exceeds pulmonary artery diastolic pressure.

Aorta
Another M-mode sign of the rapid early ventricular filling in CP is a sharp downward motion of the posterior aortic root in early diastole.

Posterior LV Wall and Posterior Left Atrium Angle
In CP, the angle between the posterior LV wall and posterior left atrium can be blunted, because the thickened, constricting pericardium affects the posterior left ventricle more than the posterior left atrium, which then expands at a more acute angle respective to the LV wall.

Intervertricular Septum
Displacement of the interventricular septum toward the left atrium during inspiration is another reported sign of CP. (Table 1 summarizes sensitivities and specificities for the miscellaneous echocardiographic findings of CP.)

CONCLUSION
Multiple echocardiographic findings are used to confirm a diagnosis of CP, but demonstration of enhanced cardiac intracavitary blood flow variations during respiration, abnormal interventricular septal motion, and normal longitudinal mitral annular velocity provide the greatest diagnostic yield. In the presence of a mixed physiology (pericardial constraint combined with myocardial dysfunction), the diagnosis and therefore referral for pericardectomy remain challenging. In such patients, the assessment of LV longitudinal, circumferential, and torsional mechanics by 2-dimensional speckle-tracking imaging can potentially decipher the extent of endocardial, midmyocardial, or epicardial involvement. Further prospective studies are warranted to address the potential clinical utility of such diagnostic algorithms.

REFERENCES


64. Hoit BD. Imaging the pericardium. Cardiol Clin 1990;8:587-600.


