Echocardiographic Evaluation of Pericardial Disease

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Objectives:
1) Identify subcategories of pericardial disease
2) Apply Echo for quantitative analysis of cardiac function in patients with pericardial disease
3) Recognize pericardial tamponade

I. PERICARDIAL ANATOMY

The pericardium is a sac that normally consists of two apposed serous surfaces that surround a closed potential space. The pericardial space normally contains 5 to 50 mLs of serous fluid. This potential space is complex in that various reflections of pericardial tissue create sinuses (in this circumstance a sinus is defined as a depression or cavity formed by bending or curving) within the pericardial sac. The visceral (continuous with the epicardium) and parietal (thin, dense, fibrous structure that blends with the central tendon of the diaphragm inferiorly and is apposed to the pleural surfaces laterally) pericardium are apposed to create the pericardial space. Conjoined areas of both pericardial surfaces meet to close the ends of the pericardial sac in areas termed pericardial reflections. The reflections in the posterior region of the pericardium at the atrial level surround the venae cavae and the pulmonary veins. The reflections that surround the venae cavae and pulmonary veins create a dead ended pocket behind the left atrium termed the oblique sinus (Figure 1). A useful anatomical landmark to inspect for accumulated fluid within the oblique sinus can be obtained from TEE images of the left atrial appendage (LAA). At an omniplane of approximately 70° at the mid to high esophageal level the LAA appendage appears within the oblique sinus at screen right. An echolucent signal surrounding the LAA in this view represents fluid within the pericardial space. The reflections in the superior region of the pericardial sac surround the great vessels. The pericardial reflection around the great vessels creates the transverse sinus posterior to these structures (Figure 1).

II. PERICARDIAL PHYSIOLOGY

The definitive function of the pericardium is unclear but it is thought that the normal pericardium provides mechanical restraint to the cardiac chambers and inhibits chamber dilation, especially under conditions of acute volume increase in the heart chambers or within the pericardium. Additionally, the pericardium limits ventricular filling, and may protect against excessive incompetence of atrioventricular valves. Attachments of the pericardium at the diaphragm, at the atrial level and around the great vessels provide restraint to this structure in that expansion beyond these pericardial reflections is inhibited if the heart increases in size; the tough fibrous quality of the parietal pericardium contributes to the pericardium’s ability to provide restraint. The pericardium also isolates the heart from the surrounding mediastinal structures (e.g., the lungs and pleural spaces). The pericardium may also provide a lubricating function that facilitates normal myocardial rotation and translation during each cardiac cycle in that the mesothelial layers of the pericardium produce a serous fluid that serves as a lubricant. The pericardium is known to secrete biochemical substances (e.g., prostacyclin) that may play a role in sympathetic neural regulation, coronary vascular tone and cardiac contractility. The pericardium also has a fibrinolytic function in that it contributes to maintaining accumulated blood in a liquid state. Much of the microphysiologic function of the pericardium is not completely understood.

Normally during spontaneous respiration, changes in intrathoracic pressures are nearly equally transmitted to the pericardial space and intracardiac chambers. The pericardial pressure commonly approximates pleural pressure and will vary with the
respiratory cycle demonstrating pressures of approximately \( -6 \) mm Hg at end inspiration and \( -3 \) mm Hg at end expiration (NB – as measured by a fluid filled, non-balloon tipped catheter). It is the lowering of intrathoracic and pericardial pressure during inspiration that permits increased filling of the right-sided chambers such that venous return is augmented. The true filling pressure of the heart is determined by transmural pressure which is calculated by subtracting the pericardial pressure from the intracardiac pressure. For example, in a spontaneously breathing patient with a right atrial (RA) pressure of 6 mmHg and a pericardial pressure of \( -6 \) mmHg the actual filling pressure is \( 6 - (-6) = 12 \) mmHg, during inspiration. The same inspiratory negative intrapleural pressures create a pooling of the increased right ventricular (RV) output in the pulmonary circulation which reduces left atrial (LA) pulmonary venous return. The decrease in LA return results in a decrease in left ventricular (LV) stroke volume and output. Additionally, the decrease in intrathoracic pressure during spontaneous inspiration relative to higher extrathoracic systemic arterial pressures may contribute to a decrease in left heart output.\(^4,6\) Measurable Doppler changes in the transatrioventricular valvular velocities accompany these intrapericardial pressure dynamics. During the inspiratory phase of spontaneous respiration normal transtricuspid velocities increase by approximately 20% inspiration (Figure 2a). Transmural velocities normally decrease by approximately 10% during spontaneous inspiration (Figure 2b).\(^1\) Intermittent positive pressure ventilation (IPPV) will produce opposite changes in velocities (Figures 2c-d).\(^7\) The increase in intrathoracic pressures relative to extrathoracic systemic pressures during IPPV inspiration favors an increase in left heart output. Additionally, the increased intrathoracic pressures during IPPV inspiration expels blood from the pulmonary veins into the LA and thus augments left heart filling and output.\(^4\) The changes of transatrioventricular valvular velocities have been termed respirophasic variation.

III. PERICARDIAL PATHOLOGY

Echocardiographic evaluation of pericardial disease is performed to rule out specific pathologies that can affect myocardial function. There are five basic categories of pericardial pathology that one must consider (Table 1).
Table 1. General Categories of Pericardial Pathology

<table>
<thead>
<tr>
<th>Congenital Pericardial Defects</th>
<th>Pericarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial Effusion</td>
<td>Pericardial Tamponade</td>
</tr>
<tr>
<td>Pericardial Masses</td>
<td></td>
</tr>
</tbody>
</table>

Congenital pericardial defects are rare and consist of partial or total absence of the pericardium and Mulibrey nanism, an autosomal recessive disorder, primarily found in the Finnish population that can lead to congestive heart failure in early adult life and is partially characterized by the development of constrictive pericarditis in some affected individuals. TEE findings of constrictive pericarditis will be discussed in a later section. The significance of pericardial absence varies although appears minor in that most of these patients are clinically asymptomatic (Table 2). One small observational study (n=10) reviewing this disorder found that paroxysmal stabbing chest pain that mimicked coronary ischemia was the most common clinical presentation. Individuals with congenital absence of the pericardium are at increased risk for additional congenital abnormalities in that 30% of these patients will have some additional congenital pathology.

Table 2. Pathologic Characteristics of Forms of Pericardial Absence

<table>
<thead>
<tr>
<th>Form</th>
<th>Incidence</th>
<th>Clinical Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Bilateral Absence</td>
<td>Rare</td>
<td>Mostly asymptomatic</td>
</tr>
<tr>
<td>Partial Left Absence</td>
<td>70%</td>
<td>Increased risk for aortic dissection if augmented heart mobility;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>possible herniation or strangulation of heart structures through</td>
</tr>
<tr>
<td></td>
<td></td>
<td>the defect with chest pain, shortness of breath, syncope</td>
</tr>
<tr>
<td>Partial Right Absence</td>
<td>17%</td>
<td>Increased risk for aortic dissection if augmented mobility</td>
</tr>
<tr>
<td></td>
<td></td>
<td>of the heart exists as a result of the defect</td>
</tr>
<tr>
<td>Overall</td>
<td>0.01%</td>
<td></td>
</tr>
</tbody>
</table>

Incidence values represent a percentage of the overall incidence of 0.01%.

Echocardiographically, one may observe paradoxic ventricular septal motion, an exaggerated posterior left ventricular wall and possibly the appearance of a dilated right ventricle when assessed through the transthoracic parasternal window. A small series of patients (n=8) from a Japanese group reported more marked alterations in venous return in the right heart (measured in the superior vena cava) than in the left heart (measured in the pulmonary veins). CXR and cardiac MRI are the two imaging modalities that appear to be most useful in making the definitive diagnosis of congenital absence of the pericardium.

Pericarditis is an inflammation of the pericardium and can occur as the result of multiple pathologies. Pericarditis may present as either a chronic or acute process and is often associated with the development of a pericardial effusion. Clinically, one examines the patient suspected of having pericarditis for the presence of the classic triad of chest pain, ECG evidence of pericarditis (e.g., diffuse ST segment elevations), and a pericardial rub on auscultation. Etiologies of pericarditis include the following:

- Infections (e.g., postviral, bacterial-especially tuberculosis)
- Neoplastic (e.g., primary-mesothelioma or fibrosarcoma; secondary metastatic disease-melanoma, lymphoma, leukemia or direct extension of a pulmonic or breast tumor)
- Immune/Inflammatory (e.g., rheumatoid arthritis, systemic lupus erythematosus, scleroderma, acute rheumatic fever, dermatomyositis, Wegener’s granulomatosis, mixed collagen vascular disease; post-myocardial infarction (Dressler’s syndrome); uremia; postcardiac surgery
- Intracardiac-pericardial communications (e.g., chest trauma, post-interventional catheter procedures, postmyocardial ventricular rupture).

Echocardiographically, one inspects the pericardium for evidence of thickening that appears as an increase in the brightness, or echogenicity of the ultrasound signal. Normal pericardial thickness is approximately 2-3 mm. M-mode analysis of a patient with pericarditis will reveal multiple parallel ultrasound reflections and can be helpful to discern the thickened pericardium. Multiple echo windows should be obtained to verify the diffuse or localized nature of the pericarditis. Echocardiography lacks sufficient fidelity to consistently quantify the degree of pericarditis and thus carries an unreliable sensitivity and specificity for detecting this disorder compared to other imaging techniques (e.g, cardiac MRI or high resolution (64 slice) computed tomography).
Pericardial effusion is a collection of fluid within the pericardial sac that can either be diffusely distributed within the pericardium or localized. Depending on patient position and the amount of volume contained in the effusion different patterns of fluid distribution can be seen assuming that the pericardial space does not contain fibrous adhesions, or a mass that may redirect accumulating fluid. Echocardiographically, a pericardial effusion appears as an echolucent signal immediately adjacent to the epicardium. General guidelines on pericardial effusion size and fluid distribution patterns are presented in Table 3. Caution is warranted when diagnosing a pericardial effusion to rule out the presence of epicardial fat which can appear similar to a pericardial fluid collection at first glance. Close observation of epicardial fat demonstrates a weak echogenic signal compared to the more echolucent signal of a pericardial fluid collection.

Table 3. Pericardial Effusion Characteristics

<table>
<thead>
<tr>
<th>Severity</th>
<th>Width by Echo</th>
<th>Volume (mL)</th>
<th>Localization Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>&lt; 10 mm</td>
<td>&lt; 100</td>
<td>Behind posterior LV wall</td>
</tr>
<tr>
<td>Moderate</td>
<td>10-15 mm</td>
<td>100-150</td>
<td>Expand laterally and apically</td>
</tr>
<tr>
<td>Large</td>
<td>&gt; 15 mm</td>
<td>&gt; 500</td>
<td>Evenly distributed around the heart</td>
</tr>
</tbody>
</table>

The clinical significance of a pericardial effusion relates to the amount of fluid present and the time course over which it accumulates. Large, chronic effusions are frequently associated with excessive antero-posterior heart motion as well as counterclockwise rotation in the horizontal plane. Effusions can lead to cardiac translation within the pericardial space that can create the ECG finding termed electrical alternans (Figure 4). Electrical alternans is the regular occurrence of alternating heights of the QRS complex that may also involve the P, T, U waves and ST segments; this finding is not specific to pericardial effusions.

Pericardial effusions are highly common in cardiac surgical patients (85%) and generally peak by the 10th postoperative day. The relevance of a pericardial effusion relates to the volume of fluid that accumulates and the chronicity with which it accumulates (Figure 5a). Over time the pressure in the pericardial space will increase as the effusion grows in volume and eventually intrapericardial pressure will rise above the cardiac chamber pressures. When intrapericardial pressure increases to the point that cardiac chamber filling is compromised the patient is considered to have developed tamponade physiology resulting in the clinical finding of pericardial tamponade. Additionally, when the amount of accumulating fluid causes a more extreme increase in intrapericardial pressure (i.e. the pressure-volume relationship has reached the steep point on the curve which relates these two physiologic variables (Figure 5b)) ventricular interdependence will be manifest. Note the adaptive nature of the pericardium that is observed with slowly accumulating effusions in Figure 5b. The mesothelial cells that comprise the pericardium have the ability to hypertrophy and over time accommodate greater amounts of pericardial fluid provided that the fluid is slowly accumulating. With ventricular interdependence as the right ventricle fills the interventricular...
septum will shift towards the left and thus compromises left ventricular filling and left ventricular output as evidenced by a decrease in systemic blood pressure (Figure 5c)\(^2\). Loss of the y-descent in the CVP tracing is also characteristic of tamponade.

Tamponade physiology occurs when the pericardial pressure exceeds cardiac chamber pressure; lower pressure chambers (e.g., RA in systole) will be affected initially followed eventually by all cardiac chambers if pericardial pressures continue to rise. In fully developed tamponade all cardiac chambers will have elevated and equal diastolic pressures. Loculated effusions can elicit tamponade physiology with a considerably smaller volume of fluid than unloculated effusions if the fluid collection is strategically located near a low-pressure cardiac chamber (e.g., the right or left atrium). Echocardiographic examination must involve a 2D interrogation from multiple windows to assess for the potential presence of one or more loculated effusions.

Pericardial effusions can create a host of echocardiographic findings that include:
- An echolucent signal of variable width (Table 3) adjacent to the pericardium (note that the echolucent signal can brighten if the pericardial space is filled with blood that has clotted as may be seen with hemopericardium)
- Fibrinous stranding within the pericardial fluid may be seen in patients with long-standing pericardial disease
- Regional septal wall motion abnormalities
- MV & TV prolapse
- Systolic anterior motion (SAM) of the anterior mitral leaflet
- Early systolic closure of the aortic valve
- Mid-systolic notching (partial closure) of either the aortic valve or pulmonic valve

When pericardial effusions grow to an extent that elicits pericardial tamponade there are a host of echocardiographic findings that can be observed\(^2\):
- RA systolic collapse, or inversion (inversion for > 1/3 of systole has a 94% sensitivity and 100% specificity for tamponade)
- RV diastolic collapse (sensitivity 60-90% and specificity 85-100% for the diagnosis of tamponade)
- Reciprocal respiratory changes in RV and LV volumes (during spontaneous inspiration an increase in RV volume will occur that will result in a shift of the interventricular septum towards the left ventricle in diastole; these changes reverse
during expiration—*pulsus paradoxus* (Figure 5c) will be observed on the arterial waveform under these physiologic conditions.

- Inferior vena cava (IVC) plethora (dilated IVC with < 50% inspiratory reduction in diameter near the IVC-RA junction; this a sensitive (97%) but nonspecific indicator of tamponade)

Note that Doppler evaluation of tamponade physiology in spontaneously breathing patients can also provide useful information to confirm the diagnosis of pericardial tamponade. As seen during spontaneous respiration in normal trans-atrioventricular valve Doppler profiles there is a decrease in transmitral velocity flow profiles during inspiration (Figure 2ba). In severe tamponade this decrease can be as much as 37% (Figure 6a). The transtricuspid valve velocities increase by as much as 77% with tamponade physiology during spontaneous respiration (Figure 6b). The magnitude of the transatrioventricular velocities will be reduced in absolute value although the respirophasic variation will be accentuated in pericardial tamponade during spontaneous respiration. The mechanism accounting for the exaggerated respirophasic variation in tamponade is an insulation of the heart from the intrathoracic pressure changes associated with the respiratory cycle.

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**Figure 6a** – Spont. Respiration: Tamponade – Transmitral

![Figure 6a](image)

**Figure 6b** – Spont. Respiration: Tamponade - Transtricuspid

![Figure 6b](image)
Doppler interrogation of transmitral valve velocities during IPPV will reveal a markedly different pattern than seen with spontaneous respiration. According to work done in a canine model the normally observed dynamics in transmitral valvular velocities seen in pericardial tamponade will be opposite in direction and markedly attenuated (Figure 6c)\(^1\). Thus the characteristic respirophasic variation in transatrioventricular velocities seen with tamponade in spontaneously breathing patients is absent during IPPV. However, consistent with the pattern seen in spontaneously breathing patients with tamponade there will be an overall decrease in the amplitude of the flow velocities during mechanical ventilation compared to values obtained in individuals without tamponade\(^5,7\).

In addition to interrogation of the trans-atrioventricular valve velocities, assessment of the hepatic vein flow profiles can also provide useful diagnostic information in spontaneously breathing patients suspected of having tamponade physiology. Normally the pulse wave Doppler profile of the hepatic vein is bimodal with the forward flow during systole being equal to or greater than the forward flow during diastole throughout the respiratory cycle. The systolic and diastolic forward velocities increase during inspiration with spontaneous respiration. Under conditions of tamponade physiology the systolic and diastolic forward flow velocities also increase in magnitude during inspiration but during expiration there is a marked reduction, or reversal of both the S and D waves which is not seen in normal subjects (Figure 7)\(^{13}\). Data on respirophasic changes in the hepatic veins during IPPV is not available and thus evaluation of the hepatic vein profiles with TEE during positive IPPV is not useful at present.

A final Doppler echocardiographic quantitative method that can be integrated into the assessment of a pericardial effusion and tamponade physiology is measurement of the isovolemic relaxation time (IVRT). In one transthoracic study that compared spontaneously breathing normal adults to individuals with tamponade as well as to individuals with an effusion, but no tamponade, a significantly prolonged IVRT was observed in patients with tamponade compared to normal individuals. Normally the IVRT is < 100 msec in most age groups (mean value of 82 msec was observed in this study) and a mean value of 104 msec was observed in the tamponade group that was studied (\(P < 0.01\))\(^{13}\). Changes in the IVRT in patients with pericardial tamponade that are receiving IPPV have not been described.

The evaluation of a subtype of pericarditis termed Constrictive pericarditis is important to be aware of when diagnosing pericardial disease. Clinically significant constrictive pericarditis (CP) will affect ventricular filling in that it can mimic restrictive diastolic dysfunction. CP has been reported to occur in 0.2 – 0.3% of cardiac surgical patients and has a number of pathophysiologic features that include: fibrotic pericardium, inflamed pericardium, calcific thickening of the pericardium, abnormal diastolic filling, a narrow RV pulse pressure (i.e. the systolic RV pressure is normal but the diastolic RV pressure is elevated), a prominent early diastolic RV pressure dip and later plateau (termed the “square root sign” (Figure 8))\(^{1,14}\).
Additionally, the RA pressure exhibits a pronounced systolic drop (y descent followed by an increase and then plateau) that appears as an “M-like” wave on the CVP tracing (Figure 8).

The diagnosis of CP is made by using a combination of 2D echocardiographic analysis and Doppler analysis. The 2D echocardiographic exam will generally reveal a thickened, highly reflective pericardium in patients with CP. TEE can detect the thickened pericardium but computed tomography and magnetic resonance imaging are considered to provide more accurate measurements of the pericardial thickness. Table 4 lists a number of nonspecific echocardiographic findings associated with CP.

**Table 4. 2D-Echocardiographic Findings Associated with CP**

<table>
<thead>
<tr>
<th>Paradoxical ventricular septal motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal “bounce”</td>
</tr>
<tr>
<td>Diastolic flattening of the posterior LV</td>
</tr>
<tr>
<td>Premature mid-diastolic pulmonary valve opening</td>
</tr>
<tr>
<td>Spontaneous inspiratory leftward shift of the atrial &amp; ventricular septum</td>
</tr>
<tr>
<td>Enlarged hepatic veins</td>
</tr>
<tr>
<td>Dilated IVC without variation in size during respiration</td>
</tr>
<tr>
<td>Normal ventricular size</td>
</tr>
<tr>
<td>Normal or enlarged atria with reduced wall excursion</td>
</tr>
</tbody>
</table>

Doppler assessment of patients with CP is complex in that multiple Doppler modalities are recommended to accurately diagnose this pathology. Doppler modalities used to assess CP include: pulse wave Doppler transmitral tracings, pulse wave Doppler pulmonary vein tracings, tissue Doppler imaging of the mitral annulus and color Doppler M-mode (flow propagation, $V_p$) of the transmitral inflow. Transmitral blood flow has proven to be a useful diagnostic and prognostic tool for individuals with CP. By color Doppler assessment patients with CP may exhibit tricuspid or mitral valve incompetence. Similar to pericardial tamponade, the transmitral pulse wave Doppler profile of most patients with CP will demonstrate an exaggerated respirophasic variation (i.e. it will decrease by approximately 25% during spontaneous inspiration). Note that up to 20% of patients with CP will not exhibit this Doppler finding although the application of preload reducing maneuvers (e.g., reverse Trendelenberg position) may be useful to amplify the transmitral velocity respiratory variation that is expected in these patients. The thickened pericardium insulates the intrapericardial structures from the intrathoracic pressure changes associated with the respiratory cycle and produces an exaggerated respirophasic variation as a result (as seen in tamponade); similar respirophasic variation is observed in the pulmonary veins with CP and the S:D ratios observed are similar to those of patients with restrictive myocardial pathology (e.g., the pulmonary vein diastolic flow velocity will be greater than the systolic flow velocity in patients with a restrictive LV filling pattern, or S:D ratio < 1). Additionally, unique to CP relative to restrictive cardiomyopathy is that the peak amplitude of the pulmonary venous D wave has been noted to exhibit pronounced respiratory variation (> 18% increase upon inspiration in patients receiving IPPV) in CP.

During mechanical ventilation the transmitral E-wave velocity will increase with early inspiration in CP, thus the exaggerated respirophasic variation seen with spontaneously breathing CP patients is also observed with IPPV, although the direction of change in velocity will be opposite in direction. The increase in intrathoracic pressure expels blood from the extrapericardial pulmonary veins into the left atrium which results in an increase in transmitral flow velocity. Again the mechanism for the exaggerated respirophasic variation observed in CP patients is that the thickened pericardium insulates the intracardiac chambers (the LA in this case) from the changes in intrathoracic pressure thus increasing the gradient between the pulmonary veins and the LA during inspiration. Overall, as with pericardial tamponade, the transmitral velocities will be reduced in amplitude in CP. Given that the CP limits the volume of both cardiac chambers the inspiratory increase in transmitral E wave velocity will be accompanied by a simultaneous decrease in transtricuspid E wave velocity for two reasons. First, the increase in intrathoracic pressure will limit right sided filling and second, the increased filling velocity and increased amount of blood in the LV will result in a shift of the interventricular septum to the right and thus compromises RV filling during inspiration (i.e. ventricular interdependence is demonstrated). These changes in the transmitral velocities appear to be reversible with surgical treatment (pericardial stripping or pericardiectomy) of CP although this therapy has been associated with LV dilation and transient ventricular diastolic dysfunction.

Much has been written about making the distinction between CP and restrictive cardiomyopathy (RCM). Both of these pathologies share the common pathophysiology of decreased left ventricular compliance, but different mechanisms account for the decreased compliance in each case. In CP the decreased compliance relates to the restrictive nature of the thickened pericardium while in RCM the restriction is related to pathology within the myocardium (e.g., infiltrative disease of the muscle or hypertrophy). Recall that severely decreased LV compliance will present a restrictive LV filling pattern (Figure 9).
The differentiation of CP from RCM is best approached by assessing the patient for the described exaggerated respiratory variation in transmitral Doppler flow velocity profiles and for echocardiographic, or cardiac MRI/high resolution CT scan evidence of pericardial thickening. As previously discussed above this exaggerated respirophasic variation has been demonstrated in anesthetized patients using TEE\(^1\). Newer echocardiographic modalities that have been used to differentiate CP from RCM include color M-mode flow propagation velocities (Vp), Doppler tissue imaging (DTI) at the level of the mitral annulus, and Doppler myocardial velocity gradients (MVGs)\(^1^9^\)-\(^2^2\). DTI has been demonstrated to provide a highly sensitive and specific means to differentiate CP from RCM in one study of a homogeneous CP patient population\(^2^0\). See Table 5. DTI has been shown to be less sensitive to preload (a limitation of transmitral pulse wave Doppler) in differentiating CP from RCM. DTI at the level of the lateral mitral annulus provides a reproducible means to align the pulse wave tissue Doppler cursor with the longitudinal axis of myocardial excursion during relaxation of the muscle. Patients with CP generally have preserved myocardial diastolic function that therefore will demonstrate normal (E\(_m\) > 8 cm/sec) myocardial velocities. The converse is true for patients with RCM in that the muscle is inherently pathologic in these patients with infiltrative cardiomyopathies and thus will demonstrate abnormal (E\(_m\) < 8 cm/sec). See Figure 10.

Color M-mode, or the velocity of propagation (V\(_p\)) of transmitral flow is also recommended to be incorporated into the assessment of patients suspected of CP. Although the results of color M-mode are not as easily reproduced as DTI it is advantageous in that it appears to be preload independent and has the benefit of providing both excellent spatial and temporal resolution of diastolic mitral inflow. Color M-mode assessment of patients with CP will generally demonstrate high values of flow propagation toward the LV apex while those with RCM will have decreased V\(_p\) values. See Figure 11.
**Figure 11.** Transesophageal echocardiographic Color M-mode (Vp) of a patient with CP. Note the steep slope of the first aliasing velocity of mitral inflow (140 cm/sec).\(^1\)

Transthoracic echocardiographic Color M-mode (Vp) of a patient with restrictive physiology. Note the much more gradual slope (35 cm/sec) of the first aliasing velocity as compared to the tracing displayed above.

The relative sensitivities and specificities of each technique to distinguish CP from RCM are presented in Table 5.

**Table 5.** Relative Sensitivity and Specificity of Various Doppler techniques to distinguish CP from RCM.

<table>
<thead>
<tr>
<th>Technique</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>E wave peak (MV) (resp. variation ≥ 10%)</td>
<td>84</td>
<td>91</td>
</tr>
<tr>
<td>D wave peak (Pulmonary Vein) (resp. variation ≥ 18%)</td>
<td>79</td>
<td>91</td>
</tr>
<tr>
<td>Color M-mode Vp (slope ≥ 100 cm/s)</td>
<td>74</td>
<td>91</td>
</tr>
<tr>
<td>Tissue Doppler, (Em ≥ 8 cm/s)</td>
<td>89</td>
<td>100</td>
</tr>
</tbody>
</table>

Note that although these newer Doppler techniques are considered validated for use in determining diastolic dysfunction direct validation studies for TEE and patients receiving IPPV have not been performed.

**Pericardial masses** include benign pericardial cysts, pericardial neoplasms (mesothelioma, sarcoma, and teratomas are all gratefully rare), and tumors that invade the pericardium from neighboring tissue (e.g., breast or lung). Pericardial cysts are usually round and asymptomatic but can be associated with chest pain, dyspnea, cough, arrhythmias and compression of the left atrium or pulmonary vein(s). Any of these masses may inhibit cardiac chamber filling and result in hemodynamic compromise if they grow large enough in a critical location. Pericardial thrombus formation is another pericardial mass that may precipitate hemodynamic compromise, most commonly in the post-surgical state. Hemopericardium may result from a number of different pathologies (e.g., aortic dissection with extravasation of blood into the transverse sinus, percutaneous catheter interventions, pacer wires or post-infarction myocardial necrosis and rupture).
References: