Diastolic Heart Failure: Restrictive Cardiomyopathy, Constrictive Pericarditis, and Cardiac Tamponade: Clinical and Echocardiographic Evaluation

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An understanding of the basic principles of diastolic function is important in order to recognize diseases that may result in diastolic dysfunction and diastolic heart failure. Although uncommon, restrictive cardiomyopathy, constrictive pericarditis, and cardiac tamponade are among the disorders that may affect primarily diastolic function with preservation of systolic function. Diastolic heart failure may manifest with chronic nonspecific symptoms or may present with acute hemodynamic compromise. Echocardiography plays a vital role in the diagnosis of diastolic dysfunction and differentiation of these disease processes. It also provides a basis for clinical decisions regarding management and surgical referral. This review summarizes the clinical features, pathophysiology, and hemodynamic and echocardiographic signs of restrictive cardiomyopathy, constrictive pericarditis, and cardiac tamponade.

Key Words: Cardiac tamponade, Constrictive pericarditis, Diastolic dysfunction, Restrictive cardiomyopathy

Diastolic dysfunction is defined as the requirement for elevated filling pressures to maintain cardiac output. Augmented filling pressures may result in excessive cardiac volume and right-sided or left-sided congestion, the syndrome of diastolic heart failure (1). Although diastolic dysfunction contributes to heart failure in patients with abnormal systolic function, it may occur in patients with normal function (2). Numerous disease processes affecting the myocardium and pericardium are associated with diastolic dysfunction and heart failure. The clinical manifestations of these conditions are diverse, ranging from nonspecific, insidious symptoms such as exertional dyspnea to pulmonary edema or sudden hemodynamic collapse.

The prevalence of diastolic heart failure is variably reported depending on the age, diagnostic criteria, and referral patterns of the populations studied (2). Hypertension, coronary artery disease, valvular heart disease, and hypertrophic cardiomyopathy are the predominant myocardial disorders that result in diastolic dysfunction with relatively preserved systolic function (3). Restrictive cardiomyopathies are less common causes of diastolic dysfunction because of myocardial storage or infiltration. All of these myocardial conditions cause diastolic dysfunction largely by their effect on ventricular relaxation and compliance (4). Constrictive pericarditis and cardiac tamponade are pericardial disorders that impede ventricular filling by extrinsic constraint or limitation of chamber loading. Pericardial disorders may result in a spectrum of acute and chronic hemodynamic disturbance.

Distinguishing disorders that cause diastolic dysfunction is important so that management can be tailored to the specific disease. This review discusses the general principles of normal diastolic function and contrasts the abnormalities of diastolic dysfunction present with restrictive cardiomyopathy, constrictive pericarditis, and cardiac tamponade. The integral role of echocardiography in differentiating these diseases is highlighted.
**Diastolic Function: Physiology**

Diastolic filling of the left ventricle (LV) encompasses the period of the cardiac cycle between aortic valve closure and mitral valve closure. Four distinct phases are distinguished: (1) isovolumic relaxation (between aortic valve closure and mitral valve opening), (2) rapid filling, (3) diastasis (slow filling), and (4) atrial contraction (5). There are multiple physiologic determinants of diastolic function. The major myocardial factors that affect diastolic function include LV myocardial relaxation, compliance, and left atrial function (5). Other parameters include heart rate, preload, afterload, atrioventricular conduction, right ventricular (RV) function, intrathoracic pressure, viscoelastic properties, coronary engagement, neurohormonal activation, and pericardial constraint (6). RV diastolic function is concordant with LV diastolic function in healthy people, although discordant filling and differing external interactions may occur in some disease states.

After LV contraction, diastole begins with the energy-dependent cellular process of ventricular relaxation, which extends into diastasis. LV relaxation is best characterized by the time constant of relaxation (τ), which describes the rate of LV pressure decay in an exponential equation. Until recently, only invasive methods were available to estimate τ(7). Other, more dependent surrogate echocardiographic markers of relaxation include the isovolumic relaxation time (IVRT) and information obtained by color M-mode and Doppler tissue imaging. The IVRT characterizes the time when LV pressure declines and LV volume remains constant. When LV pressure drops below left atrial pressure, the mitral valve opens and the early rapid filling period begins.

During the early filling period (mitral E wave), there is a rapid increase in the LV chamber volume, which contributes largely to the ventricular stroke volume (8). The amount of volume entering the LV cavity is influenced by many factors, including the pressure gradient between the pulmonary veins and LV, continued myocardial relaxation, elastic recoil (ventricular suction), and stiffness properties of the myocardium (5). LV stiffness (or its reciprocal, compliance) characterizes the change in pressure within the chamber with an increase in volume and is dependent on the many inherent properties of myocardial content, chamber dimension, and external constraints of the pericardium and thorax (5).

The left atrial and LV pressures are equalized during diastasis, resulting in slow or minimal filling. Diastasis is followed by atrial contraction (mitral A wave), which contributes variably to LV end-diastolic volume. Aside from the left atrium’s passive role as a conduit receptacle, it functions as a pump to regulate filling volume. When left atrial pressure is high at the end of diastole because of systolic heart disease, Frank-Starling mechanisms are activated (increased stretch, increased force of contraction), and atrial systole may contribute significantly to diastolic filling volume (9).

**Diastolic Function: Echocardiography**

The assessment of diastolic function has become an integral part of any echocardiographic study. Many causes of diastolic heart failure can be identified, including restrictive, hypertrophic, and ischemic cardiomyopathies and pericardial, valvular, and congenital heart disease. Diastolic filling patterns can be evaluated and stages of diastolic dysfunction determined that correlate with left-sided and right-sided filling pressures and disease-related prognosis (10–13). Other techniques such as radionuclide angiography, magnetic resonance imaging, and cardiac catheterization are more time consuming, invasive, and tedious, and are generally used only as adjuncts to echocardiography.

Traditionally, the diastolic function exam focuses on pulsed Doppler recordings of left-sided mitral inflow E and A waves and pulmonary vein systolic (S), diastolic (D), and atrial reversal (AR) waves and the corresponding right-sided tricuspid inflow and hepatic vein flow waves (14). Measurements of deceleration and IVRT times and responses to physiologic maneuvers such as Valsalva provide more information. Using these parameters, 4 stages of diastolic dysfunction can be determined: (1) impaired relaxation, (2) pseudonormal stage, (3) restrictive, reversible stage, and (4) restrictive, irreversible function (15) (Fig. 1).

Newer technologies have provided supplemental data to differentiate normal from pseudonormal patterns and to estimate left atrial and LV filling pressures more accurately (16) A color M-mode display shows a more precise spatial and temporal distribution of flow velocities propagating across the mitral valve into the LV. The flow propagation velocity (vp) provides a relatively preload-independent determination of relaxation in various disease states and can be used in a regression equation with the mitral inflow E wave.
to estimate pulmonary capillary wedge pressure (17). Doppler tissue imaging obtains the low velocity and high amplitude signals of the myocardium rather than the high velocity and low amplitude blood signals (16). With this technique, spectral Doppler or color Doppler encoded velocities can be recorded. Spectral Doppler myocardial velocities of the mitral annulus provide systolic and diastolic velocities (Em, early filling; Ea or Am, atrial contraction) similar to the mitral inflow profiles. Studies have demonstrated that Em is relatively preload-independent in many disease states. As with vp, Em can be used to differentiate disease states such as restrictive cardiomyopathy and constrictive pericarditis and to assess LV filling pressures more accurately (18). Additionally, recent reports have emphasized the importance of assessing regional myocardial function by Doppler echocardiographic strain rate imaging (19).

**Restrictive Cardiomyopathy**

As defined by the revised World Health Organization Task Force, restrictive cardiomyopathy is a disease characterized by “restrictive filling and reduced diastolic volume of either or both ventricles with normal or near-normal systolic function.” (20) Despite this rigid definition requiring that restrictive hemodynamics are present, several studies have demonstrated early forms of these disorders with earlier stages of diastolic impairment. These diseases may be idiopathic or associated with specific systemic conditions and may be further characterized as myocardial (noninfiltrative, infiltrative, and storage) or endomyocardial types (21). The prognosis of restrictive cardiomyopathies is generally poor, except for those with reversible causes such as hemochromatosis. Cardiac amyloidosis is the most prevalent restrictive cardiomyopathy, and the current literature addresses predominantly this specific disease (Fig. 2).

Because of the resistance to biventricular filling, patients commonly develop a clinical profile related to congestive failure with fatigue, exercise intolerance, right-sided or left-sided heart failure, and atrial fibrillation (22,23). The clinical examination correlates with these symptoms. An elevated jugular venous pressure with an x and prominent y descent (early-stage, M wave), y descent only (late-stage), and Kussmaul’s sign may be present, although pulsus paradoxus is always absent. Depending on the stage of disease, an S4 (early) or S3 (late) may be present (21). The electrocardiogram of amyloidosis typically shows a

![Figure 1. Stages of diastolic function as assessed by Doppler echocardiography mitral inflow, pulmonary venous flow, tissue Doppler, and color M-mode. Using an integrated approach with these 4 modalities, the stages of diastolic dysfunction can be determined. A, mitral A-wave velocity; Am, tissue Doppler atrial contraction wave; CMM-vp, color M-mode flow propagation velocity; D, pulmonary venous diastolic wave; Em, tissue Doppler early filling wave; PV, pulmonary vein; Sm, tissue Doppler systolic wave; TDE, tissue Doppler echocardiography; vp, color M-mode flow propagation velocity. Reprinted with permission from Garcia MJ, Thomas JD, Klein AL: New Doppler echocardiographic applications for the study of diastolic function. J Am Coll Cardiol 1998;32:865–875.](image-url)
large discordance between echocardiographic wall thickness and voltage (low voltage and increased wall thickness) (24).

Pathophysiology
As a result of myocardial or endocardial involvement such as amyloid infiltration, the ventricle is noncompliant or stiff and resists ventricular filling. Therefore, as the volume of the heart is increased, there is a steep rise in the pressure. Diastolic right-sided or left-sided heart failure may ensue (22).

Hemodynamic Signs
In the cardiac catheterization laboratory, invasive determination of operating stiffness may be demonstrated by pressure-volume curves, LV and RV hemodynamic tracings showing a dip and plateau or square root pattern (21). Other, less specific findings include a RV systolic pressure of greater than 50 mmHg with a RV end-diastolic pressure usually less than one third of the RV systolic pressure. Typically, the LV end-diastolic pressure is greater than the RV end-diastolic pressure (25).

Echocardiographic signs
Echocardiography is uniquely suited as a diagnostic modality to detect the anatomic and functional abnormalities associated with restrictive cardiomyopathies. A combination of 2-dimensional imaging, M-mode, and Doppler information is important to identifying specific features of restrictive cardiomyopathy. Amyloidosis is primarily discussed here because it is the prototype of this class of diseases, and although endomyocardial disorders have very distinct features, they are less commonly encountered in this country.

Two-dimensional Imaging and M-mode
Increased wall thickness of the LV and often the RV is a hallmark of amyloidosis and almost all the myocardial disorders (26). Both ventricular cavities are typically small with a decreased volume and large atria. The atrial septum and cardiac valves may also be thickened. A characteristic “granular sparkling” myocardial texture was described before harmonic and high frequency imaging, but this may be a less sensitive and specific finding with current imaging modalities. A pericardial effusion may be present. The inferior vena cava is usually dilated and does not collapse with inspiration (plethoric), reflecting increased right atrial pressure.

Doppler Echocardiography
Diastolic dysfunction is a requirement for a restrictive cardiomyopathy, although not all patients will have a restrictive diastolic filling pattern. In earlier stages of disease an impaired relaxation pattern may be present before the onset of abnormal compliance. Determination of the severity of diastolic impairment is important not only for diagnostic reasons; it has been demonstrated that the prognosis of patients with restrictive filling patterns have a nearly fivefold higher mortality than the nonrestrictive patterns (27).

The classic Doppler pattern of advanced cardiac amyloidosis is as follows: (28)

1. Mitral inflow: large E wave and small A wave; E/A ratio greater than 2; short deceleration time (DT) less than 150 ms; short IVRT less than 60 ms; no significant change in mitral E wave, deceleration time, or IVRT with phases of respiration
2. Pulmonary veins: small S wave; large D wave;
S/D ratio less than 0.5; prominent AR (except with atrial systolic failure) width greater than mitral A wave and high amplitude; no significant respiratory variation of D wave

(3) Tricuspid inflow: similar to mitral inflow with an increased E/A ratio and short DT; with inspiration, there is further shortening of the DT and minimal change in E/A ratio

(4) Hepatic veins: similar to pulmonary vein flow with S/D ratio less than 0.5 and prominent atrial and ventricular reversals; with inspiration and expiration, there is increased prominence of the reversal waves

Superior vena cava flows are similar to hepatic vein flows, and right and left heart patterns are concordant in most patients. Other findings include the lack of respiratory variation of the tricuspid regurgitant jet with respiration and the presence of at least mild mitral and tricuspid regurgitation.

Doppler tissue imaging and color M-mode flow propagation have provided invaluable information for the differentiation of restrictive cardiomyopathy and constrictive pericarditis. Patients with restrictive cardiomyopathy have an abnormality of both relaxation and compliance, whereas those with constrictive pericarditis generally have normal relaxation and impaired compliance because of pericardial impedance to filling. For this reason, patients with constrictive pericarditis generally have a normal or fast color M-mode vp and a normal or increased Em, differing from patients with restrictive cardiomyopathy (16,18,29). A recent case report demonstrated that an exception to the rule of higher Em velocities with constrictive pericarditis may occur when pericardial calcification and tethering involve the annulus (30). In these cases, Doppler imaging of the apex and strain rate imaging may help confirm constriction.

**Differential Diagnosis**

Differentiation of restrictive cardiomyopathy and constrictive pericarditis is essential because of the potential to relieve constrictive pericarditis effectively with surgical pericardial stripping (Table 1). It should be appreciated that certain conditions such as radiation of the heart may cause a mixed disease of constrictive pericarditis and restrictive physiology.

Restrictive cardiomyopathy must also be distinguished from other pathologic conditions that cause an increase in wall thickness because of hypertrophy. These diseases are caused by either primary hypertrophy (hypertrophic cardiomyopathy) or secondary hypertrophy (ie, aortic valve disease, hypertension).

**Constrictive Pericarditis**

The majority of contemporary cases of nonidiopathic constrictive pericarditis are caused by previous cardiac surgery, radiation exposure, and chronic pericarditis with a myriad of etiologies (31). With the increasing rates of patients undergoing cardiac surgery, the prevalence of constrictive pericarditis may be increasing. Constrictive pericarditis can be surgically cured with pericardial stripping, with acceptable surgical mortality rates and symptomatic benefits in most patients (31). Therefore, clinicians must consider the diagnosis among patients seeking treatment with symptoms that may be consistent with the condition.

The clinical presentation of patients with constrictive pericarditis may be nonspecific and indolent in the early stages. Symptoms may include fatigue or decreased exercise tolerance. In more advanced stages, patients may develop both left and right heart failure, with the latter often predominating. Physical findings in advanced disease reflect the signs of cardiac congestion (32). The hallmarks of physical diagnosis include an elevated jugular venous pressure with a prominent y descent (Friedreich’s sign) reflecting the predominance of filling in early diastole. Kussmaul’s sign, an inspiratory increase in the jugular venous pressure, may be present but is not specific for constrictive pericarditis (32). Findings specific to constrictive pericarditis include a pericardial knock (occurs after an opening snap and before an S3 in timing) because of cessation in diastolic filling and retraction of the apical impulse in systole. In very advanced cases, patients develop severe right heart failure.

In advanced cases, noninvasive diagnostic testing may be supportive of the diagnosis. The electrocardiogram may be low in voltage because of the pericardial encasement. The chest radiograph may show calcification of the pericardium and pleural effusions.

**Pathophysiology**

Most conditions that lead to constrictive pericarditis result in inflammation, fibrosis, adhesion, thickening, and calcification of the pericardium. Isolated constrictive pericarditis is characterized by an abnormality in compliance. Ventricular compli-
ance is impaired because of external interference to diastolic filling from a rigid pericardium. The myocardium is unaffected in isolated constrictive pericarditis; therefore, systolic function and early diastolic filling are normal. Once ventricular diastolic filling reaches the limitations of the pericardial restraint, the pressure and volume in the cavity rise, filling ceases, and congestion occurs (33). Because of the isolated encasement of the pericardium and not the systemic veins or lungs, there is a dissociation between intrathoracic and intracardiac pressures with marked interventricular dependence, respiratory variation, and discordance in right and left heart filling (34).

Table 1. Differentiation between restrictive cardiomyopathy and constrictive pericarditis.

<table>
<thead>
<tr>
<th>Evaluation Type</th>
<th>Restrictive Cardiomyopathy</th>
<th>Constrictive Pericarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical Examination</td>
<td>Kussmaul’s sign may be present</td>
<td>Kussmaul’s sign usually present</td>
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<tr>
<td></td>
<td>Apical impulse may be prominent</td>
<td>Apical impulse usually not palpable</td>
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<tr>
<td></td>
<td>S3 (advanced disease), S4 (early disease)</td>
<td>Pericardial knock may be present</td>
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<tr>
<td></td>
<td>Regurgitant murmurs common</td>
<td>Regurgitant murmurs uncommon</td>
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<tr>
<td></td>
<td>Paradoxical pulse absent</td>
<td>Paradoxical pulse rare</td>
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<tr>
<td>Electrocardiography</td>
<td>Low voltage (especially in amyloidosis), pseudoinfarction, left-axis deviation, atrial fibrillation, conduction disturbances common</td>
<td>Low voltage (&lt;50 percent)</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>Absent calcification</td>
<td>Calcification sometimes</td>
</tr>
<tr>
<td>Echocardiography</td>
<td>Small LV cavity with large atria</td>
<td>Normal wall thickness</td>
</tr>
<tr>
<td></td>
<td>↑ wall thickness sometimes present (especially thickened interatrial septum in amyloidosis)</td>
<td>Pericardial thickening seen</td>
</tr>
<tr>
<td></td>
<td>Thickened cardiac valves (amyloidosis)</td>
<td>Prominent early diastolic filling with abrupt displacement of interventricular septum</td>
</tr>
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<td></td>
<td>Granular sparking texture (amyloidosis)</td>
<td></td>
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<tr>
<td>Mitral inflow</td>
<td>No resp variation of mitral inflow</td>
<td>Inspiration = ↓ mitral inflow</td>
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<tr>
<td></td>
<td>E wave, IVRT, E/A ratio &gt;2</td>
<td>E wave, prolonged IVRT</td>
</tr>
<tr>
<td></td>
<td>Short DT</td>
<td>Expiration = opposite changes</td>
</tr>
<tr>
<td></td>
<td>Diastolic regurgitation</td>
<td>Short DT</td>
</tr>
<tr>
<td>Pulmonary vein</td>
<td>Blunted S/D ratio (0.5), prominent &amp; prolonged AR</td>
<td>Diastolic regurgitation</td>
</tr>
<tr>
<td></td>
<td>No resp variation D wave</td>
<td></td>
</tr>
<tr>
<td>Tricuspid inflow</td>
<td>Mild resp variation of tricuspid inflow E wave</td>
<td>Inspiration = ↑ tricuspid inflow E wave, ↑ TR peak velocity</td>
</tr>
<tr>
<td></td>
<td>E/A ratio &gt;2</td>
<td>Expiration = opposite changes</td>
</tr>
<tr>
<td></td>
<td>TR peak velocity no significant resp change</td>
<td>Short DT</td>
</tr>
<tr>
<td></td>
<td>Short DT with inspiration</td>
<td>Diastolic regurgitation</td>
</tr>
<tr>
<td>Hepatic vein</td>
<td>Blunted S/D ratio, inspiratory ↑ reversals</td>
<td>Inspiration = Minimally ↑ HV, S and D reversals</td>
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<tr>
<td></td>
<td></td>
<td>Expiration = ↓ diastolic flow &amp; ↑ reversals</td>
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<tr>
<td>Inferior vena cava</td>
<td>Plethoric</td>
<td>Plethoric</td>
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<tr>
<td>Color M-mode</td>
<td>Slow flow propagation</td>
<td>Rapid flow propagation (≥100 cm/s)</td>
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<tr>
<td>Mitral annular motion</td>
<td>Low velocity early filling (&lt;8 cm/s)</td>
<td>High velocity early filling (≥8 cm/s)</td>
</tr>
<tr>
<td>Cardiac catheterization</td>
<td>“Dip and plateau”</td>
<td>“Dip and plateau”</td>
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<td></td>
<td>LVEDP often &gt;5 mmHg greater than RVEDP, but may be identical</td>
<td>RVEDP and LVEDP usually equal</td>
</tr>
<tr>
<td></td>
<td>RV systolic pressure &gt; 50 mmHg</td>
<td></td>
</tr>
<tr>
<td>Endomyocardial biopsy</td>
<td>RVEDP &lt; one-third of RV systolic pressure</td>
<td>Inspiration = ↑ in RV systolic pressure ↓ in LV systolic pressure</td>
</tr>
<tr>
<td></td>
<td>May reveal specific cause of restrictive cardiomyopathy</td>
<td>Expiration = opposite changes</td>
</tr>
<tr>
<td>CT/MRT</td>
<td>Pericardium usually normal</td>
<td>Pericardium may be thickened</td>
</tr>
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</table>

Hemodynamic Signs
Cardiac catheterization is usually reserved for unclear cases of constrictive pericarditis in which the diagnosis is uncertain. The classic feature of constrictive pericarditis is near equalization of chamber pressures between the left atrium, right atrium, LV and RV in end-diastole (33). Other hemodynamic findings include an elevated right atrial pressure, a pulmonary artery pressure of less than 50 mmHg, and a RV diastolic pressure exceeding one third of the RV systolic pressure (25). Characteristic pressure tracings include the atrial waveform showing the W wave configuration with preserved x and prominent y descent and the simultaneous ven- tricular pressure waveform showing the dip and plateau configuration (33). Reciprocal changes with respiration can be observed, with the RV systolic pressure increasing with inspiration and the LV pressure decreasing.

Echocardiographic Signs
An integrated and dedicated echocardiographic examination including 2-dimensional imaging, M-mode, and Doppler are capable of diagnosing the anatomic and pathophysiologic features of constrictive pericarditis in most patients with suspected disease.

Two-dimensional Imaging and M-mode
The anatomic features of constrictive pericarditis may be recognized by 2-dimensional imaging (35). These include pericardial thickening, calcification, and localized tethering of the atrial or ventricular cavities (Fig. 3). A pericardial effusion may be present. The inferior vena cava is usually dilated (plethoric). The septal bounce is a classic feature of abnormal diastolic septal motion caused by abrupt termination of ventricular filling (35). LV and RV function are usually preserved in isolated cases. M-mode signs such as flattening of the LV free wall after early diastole are less well appreciated (36).

Doppler Echocardiography
Elucidation of the pathophysiologic features of constrictive pericarditis requires a comprehensive dedicated Doppler examination. The hallmark of the examination is reciprocal respiratory variation of right and left heart flows caused by interventricular dependence (37). Because the heart is encased in a rigid shell, when the right heart fills during inspiration, the left heart filling is restricted by the shift of the septum to the left. The opposite changes occur with expiration.

The classic Doppler pattern of constrictive pericarditis is as follows (37–39) (Fig. 4):

1. Mitral inflow

Figure 3. Example of the utility of transesophageal echocardiography and magnetic resonance imaging for demonstrating the location and extent of pericardial thickening and calcification with constrictive pericarditis. Reprinted with permission from Klein AL, et al: Am Heart J 1999;138:880–889.
Inspiration: smaller E wave greater than A wave, longer IVRT and, usually shorter DT
Expiration: larger E wave greater than A wave, shorter IVRT and usually longer DT; E wave is typically increased more than 25% with expiration, and the IVRT increased more than 50% with inspiration

(2) Pulmonary veins
Inspiration: S and D waves near equal in size
Expiration: larger S and D waves

(3) Tricuspid inflow: same pattern, with reciprocal changes compared with mitral inflow; tricuspid E wave increase is typically more than 40% with inspiration

(4) Hepatic vein
Inspiration: S greater than D wave, with small AR and ventricular reversals
Expiration: S greater than D wave, with smaller or absent D wave and larger AR and ventricular reversals

Also described in constrictive pericarditis is an inspiratory increase in the tricuspid regurgitant jet velocity and duration of the signal (40). Superior vena cava flow has been useful to distinguish respiratory changes caused by chronic obstructive lung disease from constrictive pericarditis, with the latter having less significant changes in systolic forward flow during inspiration (41).

The Doppler findings of constrictive pericarditis with the hallmark of reciprocal respiratory variation contrast with the typical findings of restrictive cardiomyopathy. Doppler tissue imaging and color M-mode techniques provide more information for distinguishing these disorders. In addition, transesophageal echocardiography may be required for a more optimal Doppler examination and assessment of anatomic features of constriction.

Differential Diagnosis

There are limitations to the Doppler echocardiography differentiation of constrictive pericarditis and restrictive cardiomyopathy, and special clinical situations should be appreciated.

Particular attention must be paid to the loading conditions and respiratory effort when performing a study. A respirometer is used to assess the timing of inspiration and expiration. The first beat after inspiration is measured because it is least likely to be affected by factors affecting the respiratory effort such as chronic obstructive lung disease (34). The optimal examination is performed during a period of euovolemia, possibly requiring saline loading for hypovolemic patients and maneuvers or diuresis for hypervolemic patients (42).
The many special situations make interpretation particularly challenging (43). Patients with irregular heart rhythms like atrial fibrillation may require ventricular pacing to equalize the R-R intervals. Conditions such as mixed restriction and effusive and constrictive states have features of both diseases. Other diseases that exaggerate the rate or force of breathing such as pulmonary embolism, obesity, and chronic obstructive lung disease may cause reciprocal changes in right and left heart flows that must be differentiated from constrictive pericarditis.

**CARDIAC TAMPOONADE**

Cardiac tamponade is a medical emergency caused by the inability of the heart to fill adequately to maintain cardiac output. Although the causes of pericardial effusions are numerous, in daily practice, most cases occur after cardiac surgery or with malignancy and any cause of acute or chronic pericarditis. Clinicians must be able to recognize and treat cardiac tamponade rapidly. Echocardiography is an essential modality to guide the choice of management. Patients may seek treatment with symptoms of low cardiac output or unheralded hemodynamic collapse. The medical or surgical history of the patient should provide ample clues to the potential for this event, because many events occur in patients after procedures or with known effusions.

The bedside diagnosis of cardiac tamponade should be considered in a patient with elevated jugular veins, quiet heart sounds, tachycardia, and hypotension. The hallmark of the jugular venous waveform is the loss of the y descent with a maintained x descent (33). A pulsus paradoxus may be present but is not specific for cardiac tamponade and may be absent in many disorders in which the LV or RV diastolic pressure is high or there is decompression of respiratory changes in pressures, as with an atrial septal defect (44). Other supportive noninvasive tests include an electrocardiogram with low voltage and electrical alternans, and a chest radiograph with cardiomegaly.

**Pathophysiology**

A small volume of fluid is present in a normal pericardial sac. When fluid accumulates because of injury inflammation or hemorrhage, the intrapericardial volume and pressure rise proportionally with the compliance of the pericardial lining and the intracardiac pressures. When intrapericardial pressure exceeds intracardiac pressure, there is interference of diastolic filling and subsequent decreased cardiac output (44). With inspiration, intrathoracic pressure and pulmonary capillary wedge pressure decrease, whereas intracardiac pressure is unchanged because of the pericardial effusion. A decrease in gradient between the pulmonary veins and LV results in a decreased mitral inflow E wave with inspiration and an increase in tricuspid E-wave flow (45). Reciprocal changes occur during expiration. Because the cardiac chambers are in a finite space within the pericardium, there is equalization of pressures in all cardiac chambers.

**Hemodynamic Signs**

Right heart catheterization is an important tool to diagnose cardiac tamponade, particularly in patients in the intensive care unit setting, where physical diagnosis and echocardiography may have limitations. The typical hemodynamic signs include elevation of the central venous pressure with a prominent x descent and loss of the y descent and near equalization of right atrial, RV end-diastolic, pulmonary capillary wedge, and LV diastolic pressures (33). Tachycardia, a decreased stroke volume, cardiac output, and hypotension with a pulsus paradoxus may be present.

**Echocardiographic Signs**

In most patients with adequate image quality, echocardiography can rapidly and accurately make the diagnosis of cardiac tamponade. Causes of hemodynamic compromise other than cardiac tamponade may be excluded. The decision can also be made to evacuate the fluid via pericardiocentesis or surgery.

An integrated echocardiographic study incorporating 2-dimensional imaging and Doppler must be performed to assess the presence of a pericardial effusion and the pathophysiologic consequences. In the intensive care unit setting, a transesophageal echocardiogram may be required if transthoracic imaging is suboptimal.

**Two-dimensional Imaging and M-mode**

A pericardial effusion is present in almost all cases of cardiac tamponade. Most pericardial effusions appear as an echocardiography-free space that is localized or circumferential, free-flowing or organized. Scanning in and out of the plane may be required to separate the visceral and parietal pericardium in order to distinguish a pericar-
dial effusion from a pleural effusion. Another clue is the presence of fluid anterior to the descending aorta in the parasternal view. Even small effusions can cause tamponade, depending on their location and rate of accumulation.

Cardiac chamber collapse is a critical feature of cardiac tamponade. The chambers with the lowest intracardiac pressure are most likely to be compressed. Right atrial collapse is the earliest sign of tamponade, occurring toward the end of ventricular end-diastole and extending into ventricular systole. Because the right atrium may often invert even in normal hearts with low right atrial pressure, the specificity of right atrial collapse may be low (46). Extension of collapse greater than 1/3 of the cardiac cycle increases the sensitivity of this finding to more than 90% (46). Although RV diastolic collapse is generally a more specific indicator of tamponade, the sensitivity can be reduced because of conditions that increase RV pressure (47). Left atrial and LV collapse are unusual and are observed generally with massive effusions or, in the case of left atrial collapse, loculated effusions.

A plethoric inferior vena cava (dilated with failure to collapse by more than 50%) is a highly sensitive sign of cardiac tamponade. This finding confirms high right atrial pressure, invariable with few exceptions in cardiac tamponade (48).

**Doppler Echocardiography**

The Doppler examination confirms the pathophysiologic hemodynamic aberrations that occur during cardiac tamponade.

The classic Doppler pattern of cardiac tamponade is as follows: (37,45)

1. Mitral inflow: for inspiration, E wave decreases and IVRT increases by more than 30% compared with expiration
2. Tricuspid inflow: for inspiration, E wave increases more than 70% compared with expiration
3. Pulmonary vein: for inspiration, D wave decreases compared with expiration
4. Hepatic vein: for inspiration, S is greater than D; for expiration, an absent or very diminished D wave with prominent reversals

**Differential Diagnosis**

Aside from the differentiation of pericardial from pleural effusions, other echocardiography-free spaces around the pericardium must be recognized. These include pericardial fat pads (more often anterior and echocardiography-dense), vascular structures (descending aorta, pseudoaneurysm, coronary sinus), other pericardial structures (cysts found in right cardiophrenic angle, hematoma, or tumor), and noncardiac structures (hiatal hernia) (49).

In many situations, an incorrect diagnosis of cardiac tamponade is made based on the presence of confounding factors that affect respiratory variation or chamber collapse (50). Clinical history and other echocardiographic findings are important in differentiating these conditions.

**Conclusion**

The clinical presentation of restrictive cardiomyopathy, constrictive pericarditis, and cardiac tamponade may vary vastly from chronic nonspecific symptoms to acute hemodynamic deterioration. As diseases of diastolic dysfunction, there are common hemodynamic and echocardiographic features of these diseases. A comprehensive echocardiographic evaluation includes two-dimensional imaging and pulsed Doppler with a respirometer. Color M-mode and Doppler tissue imaging should be performed. Volume loading maneuvers or transesophageal echocardiography may also be required. Together, these techniques help to distinguish anatomic and pathophysiologic findings specific to each process and provide the basis for clinical decisions regarding management.

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