PRO

Determining Diastolic Dysfunction Has No Bearing on Management Decisions

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What Is Diastolic Dysfunction?

Diastolic heart failure is defined as heart failure in those patients with preserved left ventricular function. The prevalence of asymptomatic diastolic dysfunction in the general population is not insignificant (approximately one quarter to one third of individuals greater than fifty years of age). Note that diastolic dysfunction and diastolic heart failure are not interchangeable terms. Diastolic heart failure is used to describe clinically symptomatic individuals with the characteristic syndrome of heart failure in the setting of a normal ejection fraction whereas diastolic dysfunction simply denotes an abnormality of diastolic function and does not characterize the clinical status of an individual. The term “diastology” refers to the science and art of characterizing left ventricular relaxation, filling dynamics, and their integration into clinical practice. While determining diastolic dysfunction is germane to the practice of medicine, it has no bearing on the intraoperative management of patients undergoing cardiac surgery.

Pathophysiology Of Diastolic Dysfunction

Impaired diastolic filling initiates an upward shift of the left ventricular diastolic pressure-volume relationship. Common causes include the normal aging process and/or myocardial ischemia. Subsequent vascular and ventricular stiffness appear to be a precipitant to impaired cardiac performance and clinical prognosis. The change in cardiovascular elastance is a marker for increased cardiac morbidity and appears to be most prevalent in that segment of the community most likely to develop the clinical syndrome of heart failure independent of the underlying left ventricular ejection fraction. Diastolic dysfunction is directly related to the reduction in early left ventricular relaxation, compromising the effective transfer of the blood from the left atrial reservoir into the left ventricular cavity. There are two fundamental causes of delayed left ventricular relaxation and elevation of early left ventricular filling pressure:

1. Left ventricular distortion (infarction, hypertrophy, cardiomyopathy, etc.), imparing effective transfer of blood from mitral valve to left ventricular apex.
2. Left ventricular ejection period prolongation, due to increased ventricular-arterial afterload.

**Causes Of Diastolic Dysfunction**

There are many causes of diastolic dysfunction. Etiologies include systemic hypertension, ischemic heart disease, diabetic heart disease, obesity, numerous metabolic syndromes, sleep-disordered breathing, valvular heart disease, constrictive pericarditis, restrictive cardiomyopathy, hypertrophic cardiomyopathy, myocardial infiltrative disorders, and storage diseases.

**Assessment Of Diastolic Dysfunction**

The diagnostic standard is perhaps cardiac catheterization, which reveals increased ventricular diastolic pressure with preserved systolic function and normal ventricular volumes. There are numerous ways to assess diastolic dysfunction via echocardiography. There is no “gold standard” (all have substantial limitations):

<table>
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<tr>
<th>Method</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
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<tbody>
<tr>
<td><strong>Left Atrial Size/Volume</strong></td>
<td>Provides morphologic and physiological evidence of chronic elevation in filling pressure; severity scale based on clinical outcomes.</td>
<td>Chronic volume overload can result in an increase in LA volume yet normal LV filling pressure.</td>
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<td><strong>Transmitral Flow Pattern</strong></td>
<td>Easily obtainable; usually contains diagnostic and prognostic information.</td>
<td>Highly preload dependent; quantitative values influenced by PW sample volume placement; problematic at high heart rates, atrial fibrillation, heart block, and paced rhythms; measurement of DT when there is a nonlinear velocity decline.</td>
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<td><strong>Doppler Tissue Imaging</strong></td>
<td>Easily obtainable; early marker of diastolic dysfunction; not influenced by changes in heart rate; primarily load independent.</td>
<td>Influenced by local changes in wall motion; possibly influenced by numerous other factors.</td>
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<td><strong>Isovolumic Relaxation Time</strong></td>
<td>Represents earliest phase of diastole.</td>
<td>Requires timing of two separate events that may need different imaging planes; measurement reproducibility.</td>
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<td><strong>Mitral Inflow Propagation</strong></td>
<td>Provides temporal, velocity, and spatial information.</td>
<td>Observer reproducibility; dependent on preload and cardiac chamber size.</td>
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<td><strong>Pulmonary Vein Flow</strong></td>
<td>Relationship of PVAR to mitral A duration only marker specific for elevated LVEDP; complements PW mitral inflow; may be helpful when E-wave / A-wave fusion present.</td>
<td>May be difficult to obtain; influenced by changes in rhythm (atrial fibrillation).</td>
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An enlarged left atrium reflects chronicity of left ventricular filling pressure elevation. Doppler-derived left ventricular filling dynamics, which reflect acuity, can vacillate moment to moment. An integrative evaluation of acuity and chronicity (via numerous techniques) allows for clinical staging of diastolic dysfunction. Based on a variety of techniques, diastolic dysfunction is characterized:

<table>
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<tr>
<th>Grade</th>
<th>Description</th>
<th>Severity</th>
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<tbody>
<tr>
<td>I</td>
<td>Abnormal relaxation</td>
<td>[Mild]</td>
</tr>
<tr>
<td>II</td>
<td>Pseudonormalization</td>
<td>[Moderate]</td>
</tr>
<tr>
<td>III</td>
<td>Restriction (reversible)</td>
<td>[Severe]</td>
</tr>
<tr>
<td>IV</td>
<td>Restriction (irreversible)</td>
<td>[Severe]</td>
</tr>
</tbody>
</table>

**The Problem With Assessment Of Diastolic Dysfunction**

Diastolic filling of ventricular chambers is very complex and diastolic dysfunction is oftentimes difficult to diagnose. The relationship between diastolic function and systolic function is often blurred because of co-existent systolic dysfunction. A patient may be clinically symptomatic (diastolic heart failure) yet have a normal echocardiographic examination. Diagnosis of diastolic dysfunction (heart failure) in these patients requires provocation (“diastolic stress test”). This entails echocardiographic evaluation of the patient during aerobic stress. In summary, at the present time, we have no way of truly assessing diastolic function (all current methods have limitations). A patient may have diastolic heart failure (clinical symptoms) yet normal baseline examination (require provocative testing).

**Treatment Of Diastolic Dysfunction**

Recommendations for treatment of heart failure and normal LVEF include: (ACC/AHA 2005 Practice Guidelines)

- Control systolic / diastolic hypertension (Level of Evidence A)
- Control ventricular rate (Level of Evidence C)
- Use diuretics to control edema (Level of Evidence C)
- Coronary revascularization? (Level of Evidence C)
- Restoration of sinus rhythm (Level of Evidence C)
- Beta-adrenergic blocker (Level of Evidence C)
- Angiotensin converting enzyme inhibitor (Level of Evidence C)
- Angiotensin II receptor blocker (Level of Evidence C)
- Calcium antagonist (Level of Evidence C)
- Digitalis (Level of Evidence C)

Level of Evidence A: Multiple randomized clinical trials/meta-analyses
Level of Evidence B: Single randomized trial/nonrandomized studies
Level of Evidence C: Consensus expert opinion/case-studies/standard-of-care
Thus, initial treatment of diastolic dysfunction involves treating the underlying disorder and optimizing preload (volume reduction), controlling heart rate and blood pressure, maintaining sinus rhythm, and avoiding myocardial ischemia. Neurohormonal modulation of the renin-angiotensin-aldosterone system is currently the only drug therapy with a potentially beneficial effect on some of the pathophysiological mechanisms responsible for the increase in vascular and ventricular stiffness. At present, drug therapy may help alter symptoms yet is not linked to improved survival.

The Problem With The Intraoperative Environment

Clinical intraoperative management of patients undergoing cardiac surgery involves continuous optimization of preload, myocardial contractility, and afterload, guided by sophisticated monitors (pulmonary artery catheter, transesophageal echocardiography). Most often, we battle hypotension and administer therapy to increase preload (crystalloid/colloid), increase myocardial contractility (beta-receptor agonists, phosphodiesterase inhibitors, etc.) and/or increase systemic vascular resistance (alpha-receptor agonists, vasopressin, etc.). The only drug therapy with a potentially beneficial effect on some of the pathophysiologolical mechanisms involved with diastolic dysfunction (angiotensin converting enzyme inhibitors, angiotensin receptor blockers) has no place in this scenario. In fact, most clinicians now choose to discontinue these drugs during the preoperative period in order to decrease the incidence of intraoperative hypotension.

Summary

1. Diastolic dysfunction is common, caused by numerous things, and adversely affects outcome.
2. At present, there is no reliable way to accurately assess diastolic dysfunction via transesophageal echocardiography.
3. Treatment involves correction of underlying disorder and optimizing hemodynamics.
4. At present, there is no proven effective drug therapy. Intraoperative use of angiotensin converting enzyme inhibitors and angiotensin receptor blockers is not recommended.
5. Thus, diastolic dysfunction is a long-term medical problem, not an acute intraoperative problem and intraoperative assessment has no bearing on clinical management decisions.
References

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