The Nuts and Bolts of Diastology

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Left ventricle function is determined by the ability to perform two tasks: (1) raise intraventricular pressure during systole to eject blood into the systemic circulation and (2) relaxation during diastole to accept blood flow from the left atrium. Although most clinicians focus on systolic function when assessing ventricular performance, both systolic ejection and diastolic relaxation are vital for optimal ventricular performance. Optimal diastolic relaxation entails an instantaneous decrease in pressure from the peak left ventricular systolic pressure to the low diastolic pressure to maximize time for left ventricular filling. Passive LV properties also contribute to diastolic function and can be described as chamber stiffness (ΔP/ΔV). Like systolic dysfunction, diastolic dysfunction has varying degrees of severity. The grading scheme is mild (impaired relaxation), moderate (pseudonormal flow pattern), and severe (restrictive filling). {Nagueh, 2009 #8} Consistent in all degrees of dysfunction is the presence of elevated filling pressure and is defined by a pulmonary capillary wedge pressure (PCWP) > 12 mm Hg or a left ventricular end diastolic pressure > 16 mm Hg. {Paulus, 2007 #3}

Before an echocardiographic assessment of diastolic function can be performed, an understanding of diastolic physiology is required. Diastole begins with closure of the aortic valve and concludes with closure of the mitral valve. Phases of diastole include ventricular relaxation, rapid filling, diastasis and atrial contraction. During isovolumic relaxation, the first phase of diastole, pressure in the left ventricle falls rapidly. Relaxation involves the ventricle returning to an unstressed state and is an ATP dependent process by which intracellular calcium is sequestered in the sarcoplasmic reticulum. As left ventricular pressure falls below left atrial pressure, the mitral valve opens and passive ventricular filling begins. Pressures in the left atrium and left ventricle soon equilibrate and LV filling is diminishes. This phase is known as diastasis. The last phase of diastole is atrial contraction, where left atrial pressures increase above LV pressures. This results in ~10-20% of filling in a normal heart, but increases towards 50% in a heart with diastolic dysfunction. {Plotnick, 1989 #9}

Echocardiography plays a critical role in the diagnosis of diastolic dysfunction. Assessment of diastolic function should include morphologic and functional correlates such as left ventricular wall thickness, left atrial volume and pulmonary artery pressure. Although many patients with diastolic heart failure do not have an increase in LV wall thickness or left ventricular hypertrophy (LVH), LVH is an important contributor to diastolic heart failure. Hypertension commonly leads to LVH and is a very common disease in our aging population. Left atrial volume is also an important correlate. Left atrial enlargement is not exclusive to diastolic dysfunction, but in patients with diastolic heart failure the presence of left atrial
enlargement is known to have worse outcomes. {Abhayaratna, 2006 #11} Lastly, estimation of pulmonary artery pressures should be obtained. In the absence of pulmonary disease, elevated pulmonary artery pressures may indicate elevated left ventricular filling pressure.

Transmitral inflow velocities provide valuable information about diastolic function. Transmitral inflow patterns are best obtained in a midesophageal 4-chamber view by placing the pulse wave doppler sample volume at the mitral leaflet tips to obtain a crisp velocity profile. Measurements from this velocity profile include early ventricular filling velocity (E wave), late diastolic filling velocity (A wave), the E/A ratio, and deceleration time (DT) of early filling. Inflow patterns are determined by the E/A ratio and DT. These patterns include normal, impaired relaxation, pseudonormal LV filling, and restrictive filling. Impaired relaxation is identified by an E/A ratio < 0.8 and a DT > 200 ms. Pseudonormal pattern is identified by an E/A ratio of 0.8-1.5. Lastly, a restrictive filling pattern is identified by an E/A ratio > 2 and a DT < 160 ms. {Nagueh, 2009 #8} Unfortunately, mitral inflow has limitations. The most significant limitation is the inability to differentiate normal diastolic dysfunction from a pseudonormal filling pattern. Additionally, transmitral inflow velocities are affected by loading conditions. Any variable affecting loading conditions will alter the pressure gradient and ultimately affect the inflow pattern. These variables include sinus tachycardia, atrial arrhythmias, mitral valve disease and conduction abnormalities.

When interpreted in combination with mitral inflow patterns, pulmonary venous flow aids in the diagnosis and grading of diastolic dysfunction. Acquisition of doppler velocity profiles is obtained by placing the pulse wave doppler sample volume 0.5 cm into a pulmonary vein. Measurements obtained include the peak systolic (S) velocity, peak diastolic (D) velocity, the S/D ratio, and the peak Ar velocity. As diastolic dysfunction worsens and left atrial pressures increase, systolic velocities decrease and result in a S/D ratio < 1. A S/D ratio of < 1 used in combination with mitral inflow allows for the differentiation of a pseudonormal filling pattern from a normal pattern on a mitral inflow study. As with mitral inflow, pulmonary venous doppler flow is dependent on loading conditions and has many of the same limitations as mitral inflow.

Tissue doppler of the mitral annulus is a newer echocardiographic study that is less susceptible to varying loading conditions. Tissue doppler has many similarities to routine doppler techniques, but the doppler ultrasound settings are shifted to monitor the lower velocity frequency shifts of the mitral annulus. Tissue doppler is acquired in the midesophageal 4-chamber. The tissue doppler pulse wave sample volume is placed on the lateral or septal insertion site of the mitral valve. Primary measurements include the early diastolic (e') and late diastolic (a') velocities. Calculating an E/e' ratio provides valuable information about LV filling pressures. An E/e' < 8 of the lateral wall is consistent with mild diastolic dysfunction and an E/e' > 12 lateral wall is consistent with severe diastolic dysfunction. {Nagueh, 2009 #8} Application of the E/e' ratio is limited in patients without diastolic dysfunction and patients with heavy annular calcification or mitral valve disease.
Propagation velocity (Vp) across the mitral valve is also unaltered by variations in loading conditions. Unlike mitral inflow measurements where pulse wave doppler measures blood flow only at a single location, Vp offers the benefit of measuring velocities along a path from the LA to the LV apex. Vp is measured in the midesophageal 4-chamber view. The M-mode scan line is placed on a line connecting the center of the mitral inflow and the LV apex and color flow dopper is applied. The color flow scale is shifted to enable visualization of the aliasing velocity. The Vp is determined by measuring the slope of the aliasing velocity from the mitral leaflets to the LV. Normal Vp is > 50 cm/s and as diastolic severity worsens, propagation velocity decreases. Caution should be used in interpreting propagation velocities in patients with normal ejection fractions and LV volumes.

Diastolic heart failure has a high prevalence in the elderly patient population undergoing cardiac surgery. Furthermore, many patients develop worsening diastolic function in the post cardiopulmonary bypass period. Assessment of diastolic dysfunction in the perioperative period identifies higher risk patient populations and potentially helps in the decision making about use of pharmacologic agents.