Chapter 9
Diastology

Liem Nguyen, MD and Neal Gerstein, MD

Abstract Echocardiography can be used in real time to guide fluid responsiveness and assess the results of hemodynamic interventions perioperatively. Diastology, the assessment of intraoperative cardiac filling, can be studied by echocardiography using pulse-wave Doppler modalities. The speed at which blood enters a given chamber can be used to derive and estimate chamber pressures. There are two main properties that govern filling in a specific chamber. The first is the ability for the chamber to relax or drop its pressure. The second property is the ability of the chamber to maintain its compliance as filling proceeds. Filling abnormalities can be detected on echocardiography, yielding a spectrum of filling patterns that characterize the various stages of diastolic dysfunction.

Keywords Diastology · Diastolic dysfunction · Diastole · Cardiac filling · Pulse-wave doppler · Transmitral inflow · Pulmonary venous flow · Tissue doppler index

Introduction

In its simplest form, the study of diastolic function by echocardiography is a means to measure the speed at which blood moves or travels through the heart during diastole [1–5]. The pulse-wave Doppler serves as a "radar detector" as it measures the speed of blood moving through a given chamber. Information on the velocity of blood allows the echocardiographer to derive or estimate pressures, thus providing a means to measure the relationship between pressure and volume in a specific chamber of the heart [e.g., left atrium (LA) or left ventricle (LV)] [1, 4]. Beat to beat data on how the heart fills enables the anesthesiologist to optimize the determinants of stroke volume and thus, cardiac

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performance. The importance of understanding diastolic filling comes down to the most basic of concepts: *if the heart cannot fill adequately, it cannot eject adequately*.

There are two distinct properties that govern the normal filling of the heart. The first prerequisite to normal filling is an adequate drop or fall in pressure in the downstream receiving chamber (e.g., pressure drop in the LA or LV) as a result of the heart's intrinsic ability to relax. The pressure drop establishes the gradient necessary to drive blood downhill through the heart. The second property that governs normal adequate filling is the ability for the heart to fill with volume and simultaneously maintain a relatively low filling pressure. The heart's pressure response to increasing volume is referred to as compliance ($\Delta V/\Delta P$). Together, the heart's ability to relax (adequate pressure drop) and remain compliant (maintain low pressures despite high volumes) are the two main properties that regulate adequate filling. It is the presence of inadequate relaxation and/or poor compliance that characterizes the onset of impaired filling. The hallmark of diastolic dysfunction is therefore a result of the inability of the heart to relax and/or remain compliant [1, 4].

The echocardiographer uses the pulse-wave Doppler to detect changes or deviations from normal blood velocity, indicating the presence of an abnormal filling process. Using Doppler echocardiography, the anesthesiologist may be able to identify a shift from a normal filling state to an abnormal filling state. This data can then be used to determine how to improve and correct the filling abnormality [4, 5].

Phases of Diastole

There are four main phases of diastole (Fig. 9.1): Isovolumetric relaxation (IVRT), early rapid filling, diastasis, and atrial contraction [1, 2, 6].

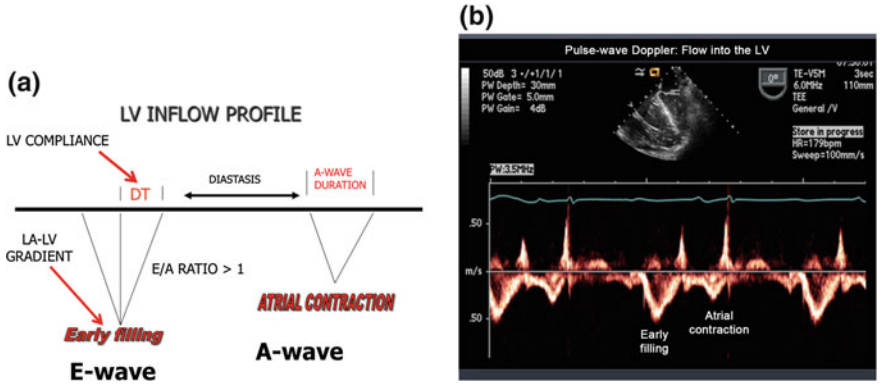


Fig. 9.1 **a** Diagram of a normal transmitral inflow profile. After IVRT, the mitral valve opens and early filling (E-wave) commences, followed by a period of pressure equalization (diastasis), ending with atrial contraction (A-wave). The peak E-wave velocity is proportional to the LA–LV gradient while the deceleration time (DT) is dependent on LV compliance. **b** Pulse-wave Doppler of normal transmitral inflow. The profile is obtained by placing the point of interrogation just below the mitral leaflet tips and utilizing pulse-wave Doppler echocardiography

1. Isovolumetric relaxation
   The start of IVRT corresponds to the onset of aortic valve closure. With both the tricuspid and mitral valves closed, the chamber pressure in both ventricles fall, creating a gradient to pull blood downward into the receiving ventricle when the atrioventricular valves are open in the next phase of diastole. This active phase of relaxation is energetically consuming, with the inactivation of the contractile apparatus driven by sequestration of Ca^{2+} ions into the sarcoplasmic reticulum. The pressure drop in the ventricle is the prerequisite for establishing an adequate atrial-ventricular pressure gradient for normal filling [1, 2].
2. Early rapid filling
   As the tricuspid and mitral valves open, early filling of the ventricle commences as blood is pulled downward into the downstream chamber. Pulse-wave Doppler interrogation of the blood flowing into the ventricle produces the characteristic E-wave (velocity of the early filling wave) on the LV or right ventricle (RV) inflow profile (Fig. 9.1). The peak E-wave velocity is primarily dependent on the LA–LV gradient. The LA–LV gradient is a function of the large pressure drop in the LV during the IVRT phase, and *not* a result of a rise in LA pressure. The deceleration time (DT) is the time it takes for the atrial and ventricular pressure to equilibrate and is predicated upon the volume–pressure relationship or compliance ($\Delta V/\Delta P$) of the downstream receiving ventricle. That is, the DT is the time for the peak velocity (E-wave) of blood moving into the ventricle to reach zero [1, 4].
3. Diastasis
   Diastasis marks the period in which the atrium and ventricle are in equilibrium with very little flow moving into either chamber. This period is often truncated or shortened during higher heart rates (shorter time in diastole) and is more prolonged during lower heart rates.
4. Atrial contraction
   Atrial contraction (A-wave) occurs at the latter stage of diastole and corresponds to the p-wave on the electrocardiogram. The atrial contraction or "atrial kick" mechanism transiently increases the atrial pressure and generates a "push" of blood into the receiving ventricle. The velocity of blood being "pushed" downward into the ventricle is described by the peak A-wave velocity. Diastole is finally complete when the atrioventricular valves close [1, 4].

Assessment of Chamber Filling: Pulse-Wave Doppler Echocardiography

Transmitral Inflow: Measuring Flow into the Left Ventricle

The LV or transmitral inflow profile (Fig. 9.1) is obtained with pulse-wave Doppler echocardiography by setting the point of interrogation just below the leaflet tips of

the mitral valve in the midesophageal 4-chamber view at zero degrees [1, 2, 4, 6]. This modality measures the velocity of blood traveling from the left atrium to the ventricle during the early filling phase of diastole. In other words, it is a measurement of how fast blood enters the LV. The speed at which blood enters the LV is primarily dependent on the LA–LV gradient (Fig. 9.1), which under normal conditions is a result of LV relaxation. As in any Doppler assessment, it is essential to align the pulse-wave Doppler parallel to the direction of blood flow to obtain the most accurate results. The normal LV filling pattern (Fig. 9.1) is described by a higher velocity E-wave (early filling), a period of diastasis (equilibrium), and a lower velocity A-wave (atrial contraction and late filling). The higher velocity E-wave is a consequence of the larger LA–LV gradient during the initial mitral valve opening. By contrast, at late diastole, the gradient between the LA–LV gradient is smaller due to the almost filled LV (from the early filling phase), resulting in a lower velocity A-wave. The end result is an E-wave velocity that is $\sim$1.5 times greater than the A-wave velocity or E/A ratio of $\sim$1.5:1.0. The DT marks the time for the peak E-velocity to reach baseline (zero velocity) and defines the time it takes for the LA and LV pressures to equilibrate. The DT mathematically describes how long the early filling occurs before diastasis begins and is a measure of LV compliance (Fig. 9.1). A short DT signifies a truncated filling period where the mitral valve opens and flow ceases quickly due to rapid atrial–ventricular pressure equilibration often from poor ventricular compliance. Normal deceleration times indicate that the early filling occurs longer and may represent adequate ventricular filling and compliance [1, 4, 5].

Pulmonary Venous Flow: Measuring Flow into the Left Atrium

LA inflow or pulmonary venous flow can be measured by placing the pulse-wave Doppler point of interrogation into the left or right upper pulmonary vein [1, 2, 6]. The point of interrogation is placed approximately one centimeter into the pulmonary vein near the entry point of the left atrium. The resulting velocity profile (Fig. 9.2) consists of higher velocity systolic wave (S-wave) and lower velocity diastolic wave (D-wave). The higher velocity S-wave is a result of active left atrial relaxation promoting a larger gradient for forward flow from the pulmonary veins into the left atrium during early systole. As the mitral valve opens during early diastole, forward flow from the pulmonary veins travels downhill into the LA and eventually into the LV, corresponding to the D-wave on the LA inflow profile. The higher velocity S-wave is often affected by either the inability of the left atrium to relax adequately and/or an increase in left atrial pressure. Accordingly, a blunted or reduced S-wave velocity is often a marker of an abnormality of left atrial relaxation and/or compliance (Fig. 9.2) [4].

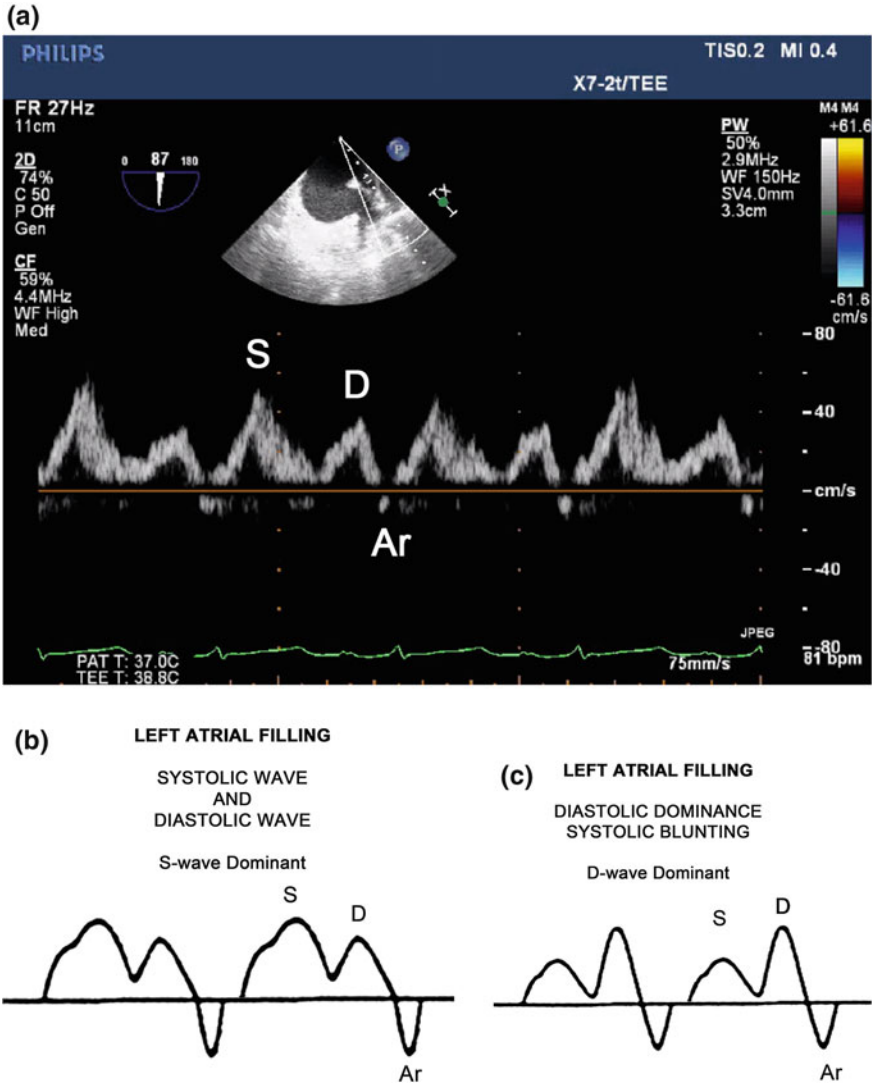


Fig. 9.2 **a** Left atrial filling or pulmonary vein flow profile obtained by placing point of interrogation ~1 cm into the left upper pulmonary vein near the entry of the left atrium. **b** Left atrial filling or pulmonary vein flow profile consists of a higher velocity and more dominant systolic (S-wave) component and a lower velocity diastolic (D-wave) component. **c** The blunted S-wave velocity is a result of a poor left atrial relaxation and/or a rise in LA pressure

Tissue Doppler Imaging: Measuring the Velocity of Myocardial Tissue

During the first stage of diastole, IVRT results in a net drop in LV pressure creating a downhill pressure gradient. During this period of LV relaxation, the lateral aspect of the mitral annulus and atrial myocardium moves upward (the LV is "snapping open"). The direction, movement, and velocity of the atrial myocardium-mitral annulus can be measured using tissue Doppler imaging [1, 4, 7, 8]. Tissue Doppler imaging is a mode of spectral Doppler with the filter simply reversed. Doppler evaluation of blood flow utilizes a filter to remove slow moving bright echoes (i.e., tissue) to focus on fast moving low echoes (i.e., blood). Tissue Doppler inverses the filter to focus on the velocity of slower moving bright echoes, the tissue velocity and filters out the faster moving lower echoes, the blood. Analogous to measuring the velocity of blood moving across the mitral valve, pulsed-wave tissue Doppler interrogation can be utilized to measure the velocity of myocardial tissue, or specifically, the translational movement of the atrial myocardium or mitral annulus during diastole.

The tissue Doppler profile (Fig. 9.3) of the lateral mitral annulus-atrial myocardium is described by two positive deflections, a positive signal in early diastole,

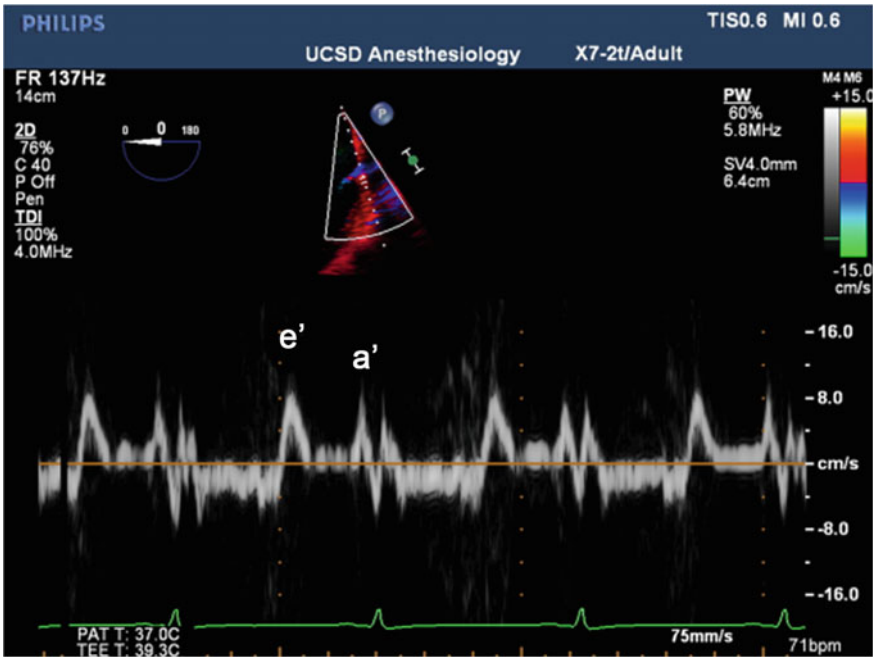


Fig. 9.3 Tissue doppler interrogation measuring the upward velocity of the atrial myocardium and mitral annulus during diastole. The e′-wave corresponds to ascension of the atrial myocardium and mitral annulus during IVRT. The a′-wave corresponds to movement of the myocardium just before atrial contraction

carrying the designation of e′ (e prime), and another wave in late diastole with the designation of a' (a prime). The onset of the e'-wave occurs just before the mitral valve opens and represents the rate of relaxation of the myocardium during the isolumetric relaxation period (IVRT). The e′-wave velocity is therefore an index of myocardial relaxation. The tissue Doppler e′-wave velocity (normally ~10–12 cm/s) is much lower than the LV inflow E-wave velocity (~50–100 cm/s) of blood during early diastole, with a normal E/e′ ratio of ≤8 [7]. An e′-wave velocity of less than 10 cm/s is abnormal and suggests that impaired myocardial relaxation is present [7]. The a′-wave represents upward motion of the atrial myocardium and lateral annulus just before atrial contraction [1, 4].

Stages of Diastolic Dysfunction: From Mild to Severe Dysfunction (Table 9.1)

Diastolic Dysfunction

2D	• Left Ventricular Hypertrophy • Left Atrial Enlargement • Secondary right heart dysfunction from pHTN
CFD	• Typically not utilized
Spectral	• Assess severity – PWD: Mitral Inflow – PWD: Pulmonary Venous Inflow – TDI: Mitral Annular Velocity

pHTN Pulmonary hypertension, *PWD* Pulsed-wave Doppler, *TDI* Tissue Doppler imaging

The term diastolic dysfunction encompasses a spectrum of abnormal filling stages of increasing severity (Fig. 9.4) [1, 2, 4, 6]. On the other hand, the term diastolic heart failure describes a state of decompensation due to diastolic dysfunction as a result of redistribution of fluid into the pulmonary venous system despite a normal ejection fraction [3, 4]. At the left side of the spectrum, the mildest stage of diastolic dysfunction is designated as *impaired relaxation*, which is characterized by the inability of the ventricle to adequately relax or drop its pressure. The next stage of the spectrum is designated as *pseudonormalization*, which is a moderate or intermediate stage of diastolic dysfunction characterized by the presence of *impaired relaxation* and a compensatory elevation in left atrial pressure. The final and most severe stage of diastolic dysfunction is referred to as the *restrictive* form. The *restrictive form* is manifested by the presence of impaired relaxation, elevated LA pressures, and elevated LV pressures (from poor compliance) [1, 4, 5].

Table 9.1 Stages of diastolic dysfunction

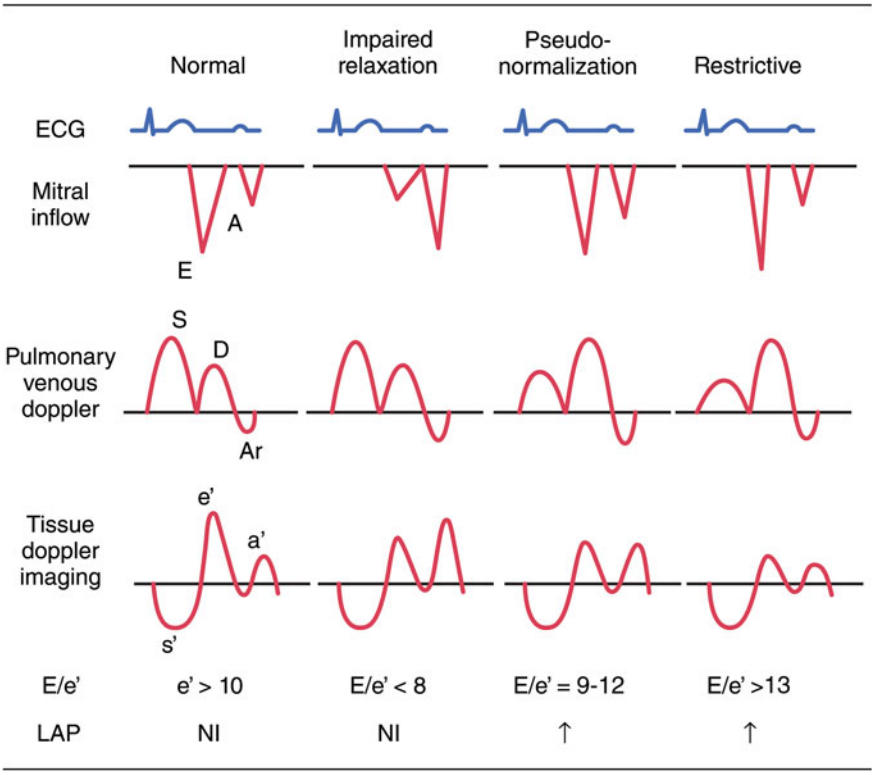
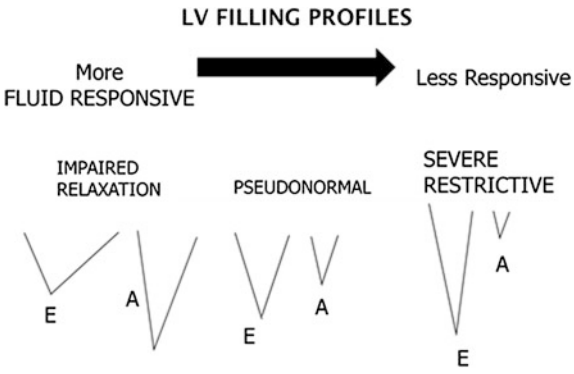


Fig. 9.4 The spectrum of diastolic dysfunction



Impaired Relaxation

The inability of the LV to relax marks the first stage of diastolic dysfunction and is referred to as *impaired relaxation* [1, 4, 6]. In this mild form of diastolic dysfunction, isolated impaired relaxation of the ventricle is present in the setting of

normal LA and LV compliance. Due to a smaller drop in LV pressure (smaller $\Delta P/\Delta T$), the LA–LV gradient is decreased leading to a reduction in the velocity and amount of blood moving into the ventricle during early filling (reduced E-wave velocity). As less blood fills the LV during the early filling phase, more volume is left in the LA during diastasis. The increased residual LA volume leads to a larger LA–LV gradient during late diastole, corresponding to a higher A-wave velocity and greater dependence on atrial contraction ($\sim$30–40 % contribution vs. $\sim$20–30 %) for adequate filling. In the earliest stage of diastolic dysfunction, impaired LV relaxation results in a *decrease* in the E-wave velocity and an *increase* in the A-wave velocity (E/A ratio <1 or E to A reversal, Fig. 9.4).

Pulsed-wave tissue Doppler interrogation of patients with *impaired relaxation* generally show reduced myocardial tissue velocity signals (<10 cm/s) of the lateral mitral annulus or atrial myocardium. This corresponds with an impaired ability of the atrial myocardium-mitral annulus to move upward during LV relaxation. However, since the LA pressures are normal in *impaired relaxation*, the velocity of blood flowing into the LA remains normal, as evidenced by a normal left atrial inflow pattern (S-wave dominant, Fig. 9.2). In summary, impaired relaxation is characterized by a *decreased* E-wave velocity, *increased* A-wave velocity, abnormal tissue Doppler velocities (<10 cm/s), in the presence of normal LA and LV compliance [1, 4].

Pseudonormalization: Impaired Relaxation and Elevated LA Pressure

The moderate or intermediate stage of diastolic dysfunction can be simplified by superimposing the presence of elevated left atrial pressures onto a background of poor LV relaxation. This defines the transition of the *impaired relaxation* stage to the next stage of diastolic dysfunction, designated as *pseudonormal*. The increase in left atrial pressure in the setting of impaired relaxation generates a reduction or blunting of the S-wave velocity ($S_{velocity} < D_{velocity}$) in the left atrial inflow (pulmonary venous flow) Doppler profile. As a result of poor left atrial relaxation and/or a rise in LA pressure, the pressure gradient dictating forward flow from the pulmonary veins into the left atrium is reduced, yielding a diastolic (D-wave) dominant profile (Fig. 9.2) [1, 4]. The additional increase in LA pressures (larger LA–LV gradient) in the setting of impaired relaxation (E < A) produces an increase in the E-wave velocity, resulting in reversal and therefore, normalization of the E and A transmitral velocities (E > A). The resulting "pseudonormal" E > A pattern and systolic blunting of the left atrial inflow profile are the defining characteristics of this intermediate stage of diastolic dysfunction. Furthermore, as the *pseudonormal* stage combines impaired relaxation and the presence of elevated LA pressures, tissue Doppler interrogation of the atrial myocardium and mitral annulus is abnormal (<10 cm/s), consistent with an LV relaxation defect. In summary, the

pseudonormal stage of diastolic dysfunction is an intermediate stage characterized by impaired relaxation and elevated LA pressures. Accordingly, the distinguishing features of the *pseudonormal* stage are the presence of abnormal myocardial velocities (<10 cm/s) and blunted S-wave velocities (diastolic dominance) on tissue Doppler imaging and left atrial inflow, respectively [1, 4].

Restrictive: Impaired Relaxation, Poor LV Compliance, Elevated LA and LV Pressures

The progression from *pseudonormal* to the most severe or *restrictive* stage of diastolic dysfunction is marked by the addition of poor LV compliance on the background of impaired relaxation and elevated LA pressures. The *restrictive* stage features the addition of a stiff and noncompliant LV in the setting of already present impaired relaxation and high LA pressures [1, 4, 6]. Marked elevations in LV pressure from poor LV compliance result in restricted LV filling and a major redistribution of volume to the left atrium. The large left atrial volume thus provides the basis for pulmonary venous congestion and symptomatic diastolic heart failure. Marked elevations in LA volume and pressure generate a much larger LA–LV gradient and faster E-wave velocity ($E \ggg A$) during early filling compared to normal (Fig. 9.4). In addition, as the pressure in the LA rapidly increases, the mitral valve is forced open before the LV can adequately relax. The velocity of blood moving into the LV is very fast as the elevated LA pressure forces the mitral valve open and pushes blood downstream into the LV. However, the overall volume of filling is extremely restricted as the LV pressures rise abruptly and rapidly equalize with the LA, generating a very short DT (time from peak E-wave velocity to zero). The end result is a small and quick "pulse" of volume that is delivered to the LV as the filling rapidly begins and ends during the early filling phase. After a period of diastasis, the elevated LV pressures are not conducive to late filling, resulting in a very small or negligible A-wave velocity during atrial contraction. This provides the basis for the unique pattern of a very fast E-wave, low velocity A-wave ($E \ggg A$, or $E/A > 2$), and short DT ($DT < 150$) on the LV inflow or transmitral profile (Fig. 9.4) [1, 4, 6].

Clinical Implications of Diastolic Dysfunction: Real-Time Measurements

Pulsed wave Doppler allows the echocardiographer to measure the velocity of blood moving into a given chamber. This is the main clinical value of diastology. It confers the echocardiographer the ability to evaluate how the heart fills and responds to volume on a beat-to-beat basis [9]. This modality allows the clinician to identify which patients are at risk of developing heart failure [4], providing the

foundation to optimize hemodynamics and cardiac performance in high risk patients [4, 10]. For example, in patients with impaired relaxation (i.e., E/A reversal or E/A ratio <1) and normal LA and LV pressures, the A-wave, or "atrial kick" component plays a larger role in LV filling compared to a normal profile. An increase in left atrial pressure with fluid therapy will therefore improve LV filling. Taken together, this early stage of diastolic dysfunction may be improved by maintaining a slow sinus rhythm, allowing more time for the LV to relax and fill, and by gentle administration of fluid to increase the LA–LV gradient [4]. Medications that may enhance LV relaxation such as milrinone, nitroglycerin, or levosimendan may also improve the relaxation abnormality and augment LV filling [4, 11–13]. On the other end of the spectrum, patients with a severe restrictive pattern with poor compliance and marked elevations in LA and LV pressures display a fixed end diastolic volume and stroke volume. In this case, slower heart rates may actually compromise cardiac output, whereas relative tachycardia may be more beneficial [1, 4]. Additionally, the elevated left ventricular end diastolic pressure (LVEDP) observed in patients with a restrictive pattern may necessitate an equivalent rise in aortic pressure to optimize coronary perfusion. Judicial use of diuretics may also be warranted as marked elevations in LA and LV pressure portends a greater risk of developing heart failure. Patients with a restrictive pattern may therefore benefit from relative tachycardia, higher aortic pressures, and avoiding further increases in LA or LV pressure [4]. The dynamic spectrum of diastolic dysfunction can be appreciated in the operating room (Fig. 9.4) [4, 7–10]. A patient with a pseudonormal pattern of filling may be pushed to the right of the spectrum and progress to a restrictive form following a fluid bolus, sudden decrease in coronary perfusion pressure, or acute onset valvular regurgitation. On the other hand, a patient with a restrictive pattern may improve and move to the left of the spectrum after hemodynamic optimization with diuretic therapy, careful initiation of inotropic therapy, and an increase in coronary perfusion pressure. Patients demonstrating impaired relaxation may benefit from slight increases in LA pressure and are therefore more fluid responsive compared to pseudonormal or restrictive patterns (Fig. 9.4). An impaired relaxation pattern may be moved to the left of the spectrum by the administration of nitroglycerin or a lusitropic agent to improve relaxation and b-blockers to increase diastolic filling by maximizing early filling and atrial kick [4].

Doppler analysis of flow into the left ventricle or left atrium can also be individually performed to estimate changes in LV or LA filling pressures to optimize intraoperative hemodynamics as well. Using this approach, targeting a specific filling abnormality can be exploited to optimize cardiac output in real time (Fig. 9.5) [4, 9, 10]. By taking serial measurements of the velocity of blood moving into the left ventricle and obtaining an LV inflow profile before and after a therapeutic intervention, the clinician can track changes in the peak E-wave velocity, A-wave velocity, or deceleration times. For example, if the DT shortens after multiple fluid boluses, this suggests a right shift (Fig. 9.4) to a less fluid responsive state or less compliant portion of the starling curve. Similarly, flow into the left atrium and the left atrial inflow profile can detect changes in left atrial pressure after

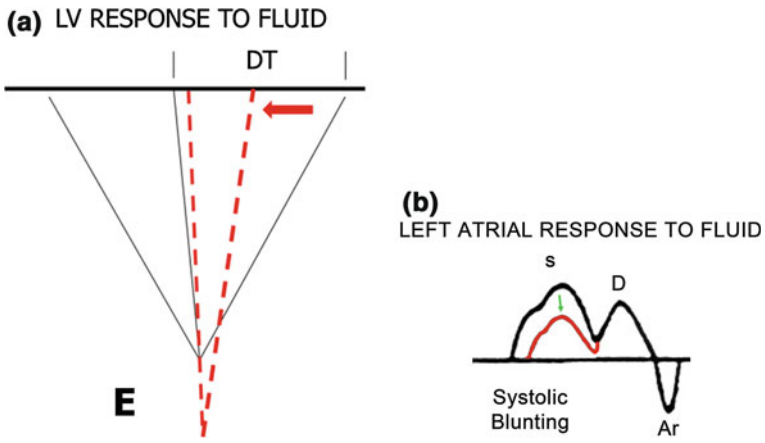


Fig. 9.5 **a** LV inflow profile at baseline (*black line*) and after a fluid challenge (*red dashed line*) demonstrating an increase in the peak E-wave velocity and shortening of the DT, corresponding to an increase in LA pressure and poor LV compliance. **b** LA inflow profile before (*black line*) and after (*red line*) a fluid challenge demonstrating systolic blunting and an increase in LA pressure

fluid administration. A normal left atrial inflow pattern exhibiting an S-wave dominant pattern (S > D) indicates normal left atrial pressures. If the S-wave begins to blunt and a D-wave dominant pattern (S < D) emerges after a fluid bolus, a rise in left atrial pressure is the likely cause of the change in the left atrial inflow profile (Fig. 9.5). A sudden increase in LA pressures signifies a shift to a less fluid responsive state and carries a greater risk of pulmonary congestion [9, 10].

The use of intraoperative echocardiography assisted hemodynamic management may confer additional advantages when compared to traditional static indices of chamber filling pressures [9, 10, 14]. One of the main advantages of intraoperative echocardiography is the ability to estimate the filling pressures of the left side of the heart and combine the data with stroke volume measurements by echocardiography or by other modalities [9, 10, 14]. The other main advantage of Doppler echocardiography is the ability to easily and repeatedly measure flow velocities and chamber pressures in real time. In a few studies, dynamic markers of LA and LV filling by echocardiography have been shown to be a viable alternative to conventional catheter-based techniques to measure intracardiac filling pressures. The use of intraoperative echocardiography-guided hemodynamic management was shown to reduce the amount of overall fluid administration when compared to the control group in one study [9]. At present, the ability to monitor dynamic indices of cardiac performance in real time to optimize hemodynamics is thoroughly lacking. With further study, spectral Doppler ultrasound has the potential to be an excellent dynamic cardiovascular monitor.

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