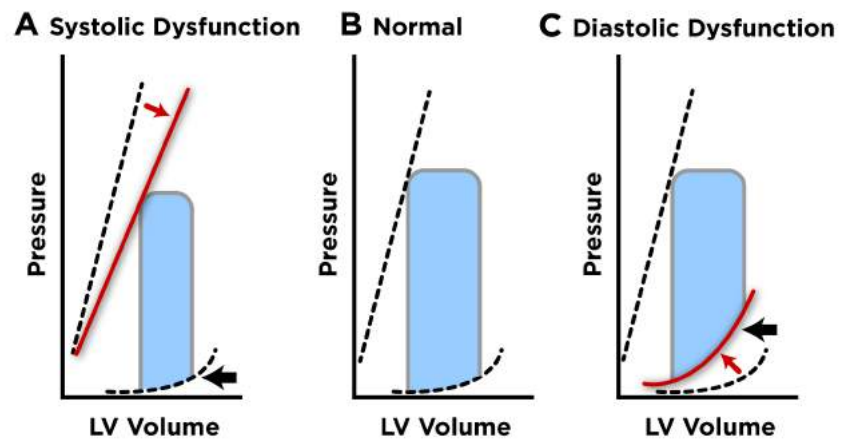


E. DIASTOLIC DYSFUNCTION

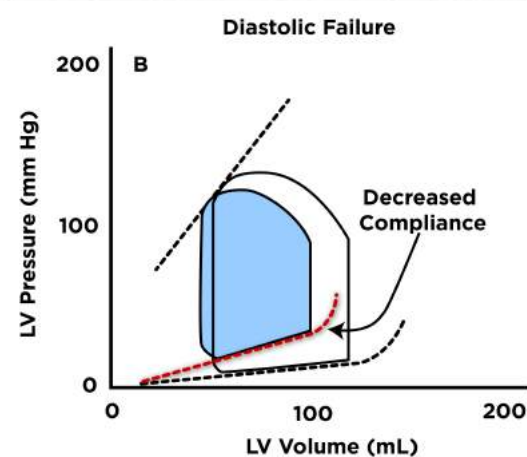
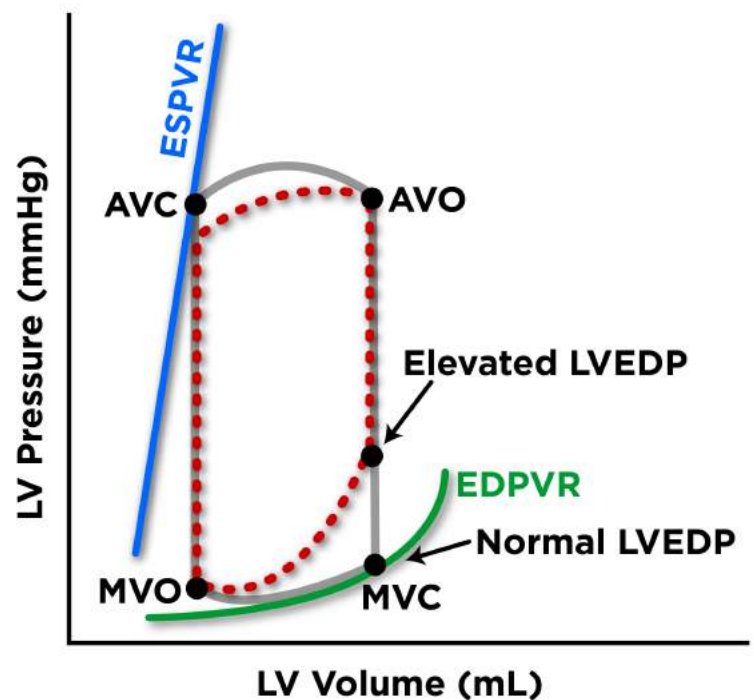
Diastolic Dysfunction is defined as an abnormality of diastolic distensibility, filling, or relaxation of the left ventricle. Normally, diastole occupies about 2/3 of the cardiac cycle. Diastolic relaxation, like systolic contraction, is an active process that requires energy, which is why moments of decreased oxygen delivery will worsen diastolic function. It is important to remember that this abnormality occurs in diastole and therefore is irrespective of systolic ejection fraction (These pathologies can coexist, but one does not require the other). Diastolic heart failure occurs when one's diastolic dysfunction is severe enough to cause dyspnea and decreased functional status. Remember that findings of dyspnea are secondary to venous congestion from elevated pulmonary venous pressure and pulmonary edema. Again, diastolic function results in a reduced cardiac output despite a normal ejection fraction. Also, the limited exercise tolerance is a result of elevated left ventricular diastolic and pulmonary venous pressure -> reduction in lung compliance -> increase in the work of breathing.

About one third of all patients with congestive heart failure have diastolic heart failure. Prevalence is highest in patients older than 75 years old. The mortality rate is about 5-8% annually as compared to 10-15% among patients with systolic heart failure and is directly related to age and the presence/absence of coronary disease. Factors that exacerbate diastolic heart failure include uncontrolled hypertension, atrial fibrillation, non-compliance with or inappropriate discontinuation of medications for heart failure, myocardial ischemia, anemia, renal insufficiency, use of NSAIDs or thiazolidinediones, and overindulgence in salty foods.

The pathophysiological features of diastolic dysfunction include 1) abnormal passive elastic properties of the left ventricle, 2) increased myocardial mass, 3) alterations in the extra myocardial collagen network, and 4) increased stiffness of the left ventricle. If you were to look at the pressure-volume curve in a patient with diastolic dysfunction, you would see the curve shift upward and to the left (see picture below).



Characteristics of Diastolic Heart Failure Compared with Those of Systolic Heart Failure		
Characteristic	Diastolic	Systolic
Clinical Symptoms (dyspnea)	Yes	Yes
LV Ejection Fraction	Normal	Decreased
Left Ventricular Mass	Increased	Increased
Wall Thickness	Increased	Decreased
End Diastolic Volume	Normal	Increased
Left Atrial Size	Increased	Increased
Exercise Capacity	Decreased	Decreased



This is because the chamber's diastolic compliance is reduced. This decreased compliance causes the time course of left ventricular filling to be altered. Specifically, the amount of blood and its velocity of flow are altered in early diastole and become more dependent on atrial kick.

Acute management of diastolic dysfunction is to prevent tachycardia and/or slow the heart rate. This will increase the diastolic time and therefore allow for the lowering of pressures to occur by giving more time for them to equalize. Since heart rate determines the length of coronary perfusion time, tachycardia causes a decrease in coronary perfusion time and increases the myocardial oxygen demand. This can be prevented by the use of beta-blockers and non-dihydropyridine calcium channel blockers. For long-term management please see the following:

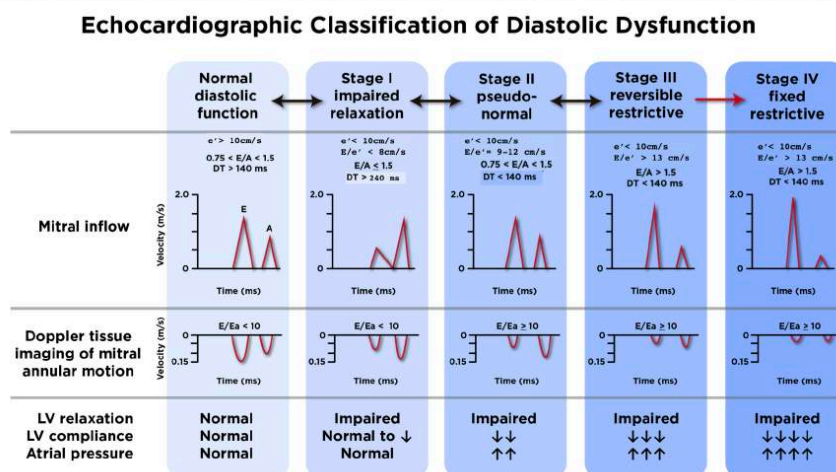
- To treat hypertension: ACE-Inhibitors or Angiotensin Receptor Blocker
- To promote regression of left ventricular hypertrophy: ACE-Inhibitors
- To prevent fibrosis: Spironolactone
- Control of ventricular rate: Calcium channel blockers
- Control of pulmonary congestion and peripheral edema: Diuretics and nitrates
- Coronary re-vascularization in patients with CHD in whom ischemia is judged to have an adverse effect on diastolic function.

Ultrasound Assessment of Diastolic Dysfunction

The modality used to assess diastolic function is pulse wave doppler echocardiography. Remember that pulse wave doppler allows one to assess the velocity in a specific area of the ultrasound image (provides depth and location). The drawback of pulse wave doppler is that there is a limited range of velocities that it can assess (range = Nyquist limit), so it cannot be used to quantify high velocity lesions (such as aortic stenosis). Please review the physics and color doppler chapters for further details on pulse wave doppler. Specifically, for diastolic function we will use PW doppler to assess the mitral inflow of blood from the LA to the LV.

To understand ultrasound assessment of diastolic function, let's first talk about the normal process that occurs in diastole. Diastole consists of 4 phases: 1) isovolumic relaxation, 2) early rapid diastolic filling, 3) diastasis (period of no flow), and 4) late diastolic filling due to atrial contraction. In diastole, isovolumic relaxation (1) is the period during which the LV pressure becomes less than LA pressure and the MV opens. Once the MV opens, rapid early diastolic filling begins (2). The driving forces are predominantly elastic recoil and relaxation of LV muscle. 80% of LV filling normally occurs during this phase. The ultrasound doppler waveform that this phase represents is called the **E wave**. It is important to realize that when both LA and LV pressures at the end of diastole (LVEDP) are normal (low), the **deceleration time** of the E wave, which is the time it takes for the E wave to go back to zero from its peak velocity (see images below), is offset a certain time period (150-220ms). When the LVEDP and thus LA pressures are elevated, the peak velocity of the E wave will be higher (since it has a higher forward pressure) and will cause the deceleration time to be faster (<140ms). There is normally a period of no flow, called diastasis (3). This is followed with late diastolic filling which results from atrial contraction, creating the **A wave** (4). This normally accounts for <20% of LV filling, but as the early filling phase (2) is more and more impaired, the atrial kick takes on a greater role of importance.

The normal E, A, and deceleration time patterns are shown below:



Tissue Doppler: Use of TDI or PW doppler modalities can also be used to measure diastolic function by assessing the motion of mitral annulus during diastole (termed e' / a' to match mitral inflow). This can be measured by placing the doppler signal at either the septal or lateral mitral annulus. Use of this parameter is a good screening technique for diastolic dysfunction. Comparison of the mitral inflow early filling wave velocity (E wave) to the early diastolic mitral annulus motion (e' wave) can be used to stratify degree of diastolic dysfunction (see table) . Remember that the waveform will be in the opposite direction as mitral inflow assessment. In addition, it will require 1/10 the scale compared to mitral inflow assessment.

normalization, the deceleration time will be less than normal (140 ms), because the elevated pressure from the LA and the elevated pressure in LVEDP cause a more rapid time of pressure equalization.

Grade III: This stage is simply a worsening of the phenomena described in Grade II. Now the LA and LVED pressures are even higher. One defines this “worsening” by looking at the E/A ratio. If this ratio is >2 , it has reached grade III diastolic dysfunction.

Grade IV: This stage is the same as grade III, the only difference is that in grade IV there is no change in severity by altering the patient’s preload, while in grade III there is.

One may assess diastolic function by placing the pulse wave doppler signal just distal to the mitral valve. Looking at the resulting waveform, if one sees $E < A$ then it is mild diastolic dysfunction. If one sees $E/A > 2$ with a deceleration time < 140 ms, it is severe diastolic dysfunction. Also, one should examine the left atrium for enlargement to help with the diagnosis as well, since a large atrium may be secondary to an elevated left ventricular end diastolic pressure from diastolic dysfunction.

Echocardiographic Classification of Diastolic Dysfunction

	Normal diastolic function	Stage I Impaired relaxation	Stage II pseudo-normal	Stage III reversible restrictive	Stage IV fixed restrictive
Mitral inflow	$e' > 10$ cm/s $0.75 < E/A < 1.5$ DT > 140 ms	$e' < 10$ cm/s $E/e' < 8$ cm/s $E/A < 1.5$ DT > 240 ms	$e' < 10$ cm/s $E/e' = 9-12$ cm/s $0.75 < E/A < 1.5$ DT < 140 ms	$e' < 10$ cm/s $E/e' > 13$ cm/s $E/A > 1.5$ DT < 140 ms	$e' < 10$ cm/s $E/e' > 13$ cm/s $E/A > 1.5$ DT < 140 ms
Doppler tissue imaging of mitral annular motion	$E/Ea < 10$	$E/Ea < 10$	$E/Ea \geq 10$	$E/Ea \geq 10$	$E/Ea \geq 10$
LV relaxation	Normal	Impaired	Impaired	Impaired	Impaired
LV compliance	Normal	Normal to ↓	↓ ↓	↓ ↓ ↓	↓ ↓ ↓ ↓
Atrial pressure	Normal	Normal	↑ ↑	↑ ↑ ↑	↑ ↑ ↑ ↑

When it comes to diastolic dysfunction, its severity is graded in four stages:

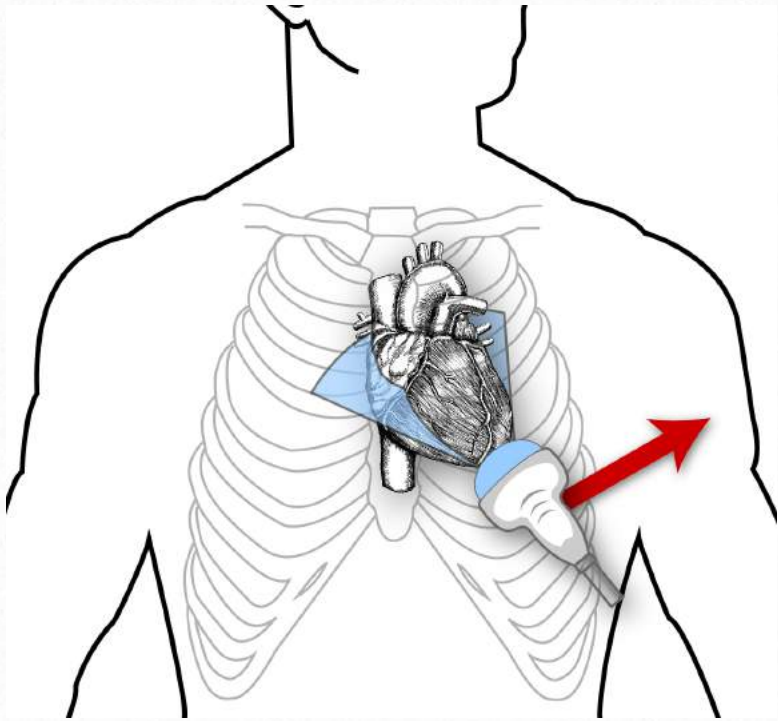
Grade I: First, in the impaired relaxation state (grade I), there is no elevation of LA pressure, but rather the LV simply takes more time to fully relax such that there is no diastasis stage. Because the E wave is stretched out over a longer period of time, the peak E velocity is reduced and is actually less than the A velocity. Also, because the E wave takes longer to return to zero from its peak, the deceleration time is increased (defined as being greater than 220ms).

Grade II: In this stage, the LV does not relax fully, and therefore there is an elevation in LVEDP and secondarily in LA pressures. This increase in pressure causes what is termed *pseudonormalization* of the E and A waves. This is because the now elevated LVEDP (that occurs from incomplete ventricular relaxation) causes the LA pressure to increase. This elevated pressure now causes the early filling stage of diastole (E wave) to shoot into the LV since it has higher LA pressure to drive it. *The way to tell the difference from normal and pseudonormalization is the deceleration time.* With pseudo-

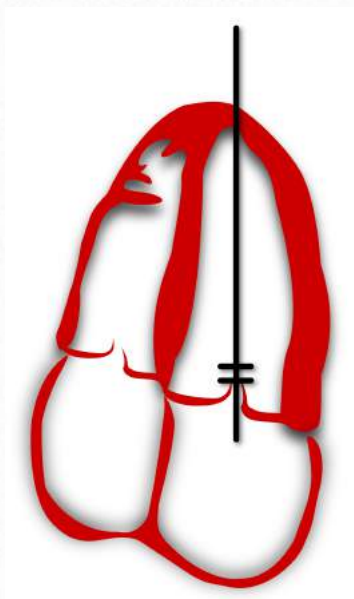
Patient Position Apical Window Views: Left-Lateral with L arm extended.

Probe Type: Phased array cardiac probe (small footprint / low frequency).

Probe Position: The apical window is usually found in the left lateral portion of the chest at the apex of the heart. This can sometimes be located by placing your hand lightly in area of the apex and feeling for the point of maximal intensity (PMI). The PMI will serve as your starting point; however, small adjustments will need to be made to the transducer location to optimize the image. Another good starting point is to go one to two rib spaces below, but in the same plane as the nipple. Also, please see the diagram below for the location of PW doppler signal.



Location of PW Doppler Signal for Mitral Inflow



Location of PW Doppler Signal for Mitral Annulus Motion (tissue doppler)

