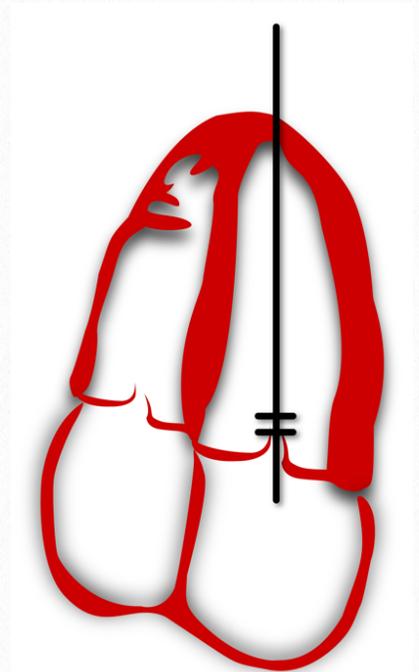# 13

Cardiac Ultrasound: 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	0.75 < E/A < 1.5 DT > 140 ms	E/A ≤ 1.5	0.75 < E/A < 1.5 DT > 140 ms	E/A > 1.5 DT < 140 ms	E/A > 1.5 DT < 140 ms
Doppler tissue imaging of mitral annular motion	E/Ea < 10	E/Ea < 10	E/Ea ≥ 10	E/Ea ≥ 10	E/Ea ≥ 10
LV relaxation	Normal	Impaired	Impaired	Impaired	Impaired
LV compliance	Normal	Normal to ↓	↓↓	↓↓↓	↓↓↓↓
Atrial pressure	Normal	Normal	↑↑	↑↑↑	↑↑↑↑
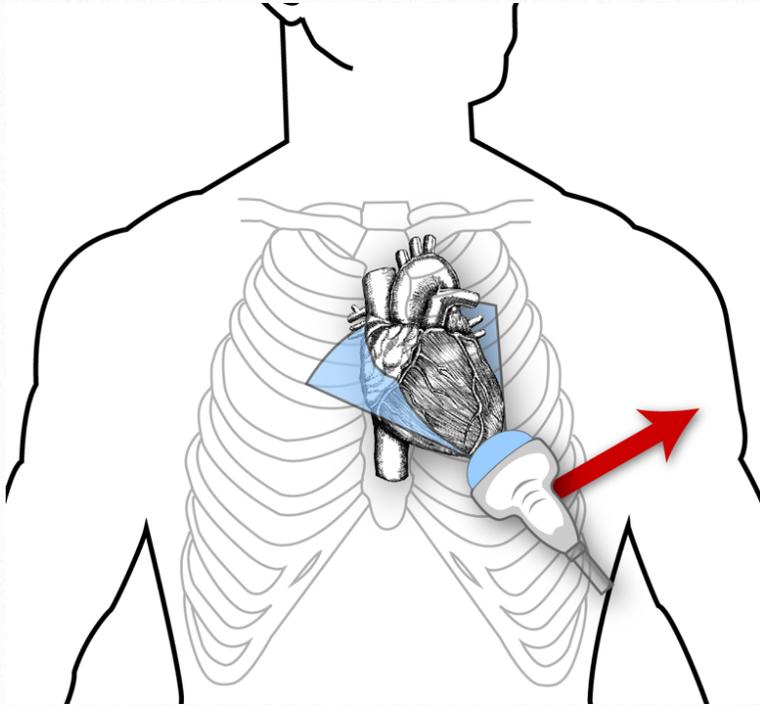
III. CARDIAC: Ultrasound is an excellent modality to assess cardiac function/abnormalities. Surface ultrasound can provide an excellent minimally invasive tool to determine the mechanisms of the patient's current hemodynamic status. Each subsection will cover a cardiac ultrasound technique used to answer these questions.

D. 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 dia-
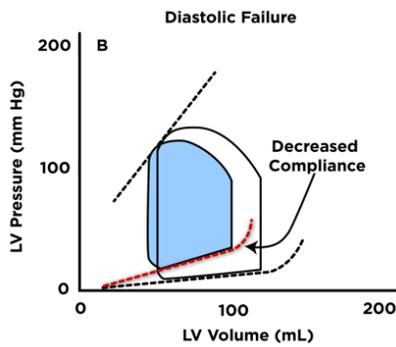

Diastolic Failure

stolic 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

Characteristics of Diastolic Heart Failure Compared with Those of Systolic Heart Failure		
Characteristic	**Diastolic**	**Systolic**
Clinicial 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
Exercse Capacity	Decreased	Decreased
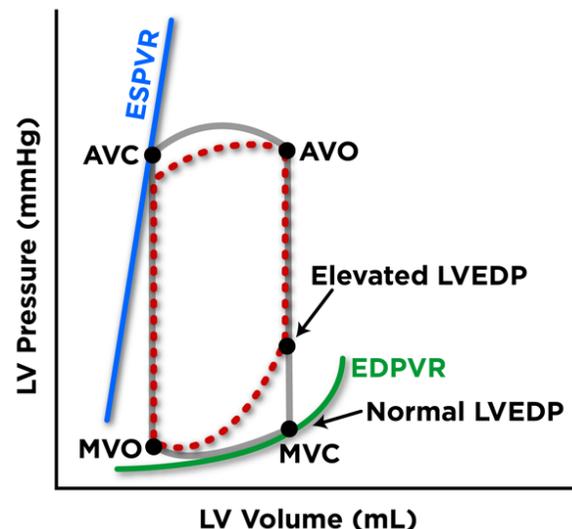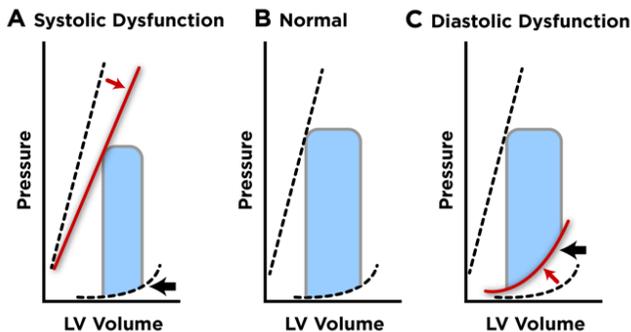
the curve shift upward and to the left (see pic below). This is because the chamber's diastolic compliance is reduced. This decreased compliance the time course of the filling of the left ventricle to be altered. Specifically, the amount of blood and its velocity of flow are altered in early diastole 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 with diuretics and nitrates

Coronary revascularization in patients with CHD in whom ischemia is judged to have an adverse effect on diastolic function

### Ultrasound Assessment of Diastolic Function:

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 filing beings (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 E wave, which is the time it takes for 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

Echocardiographic Classification of Diastolic Dysfunction
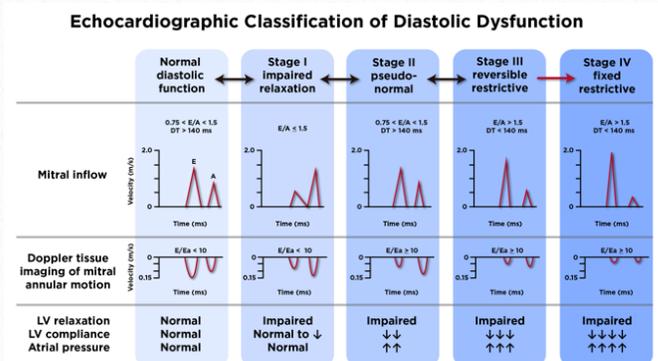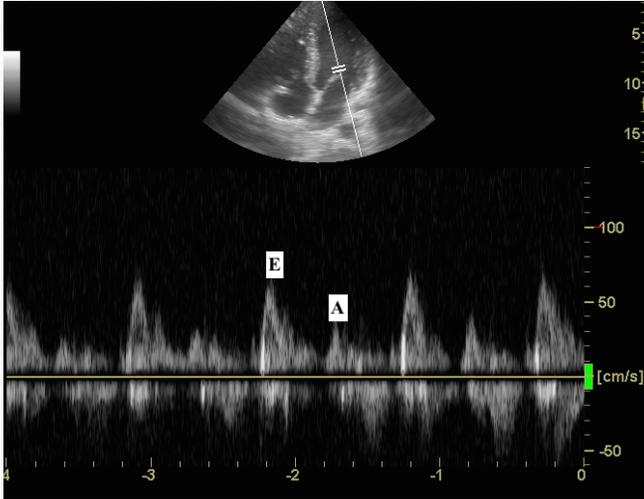
When it comes to diastolic dysfunction, its severity is graded in **four stages.**
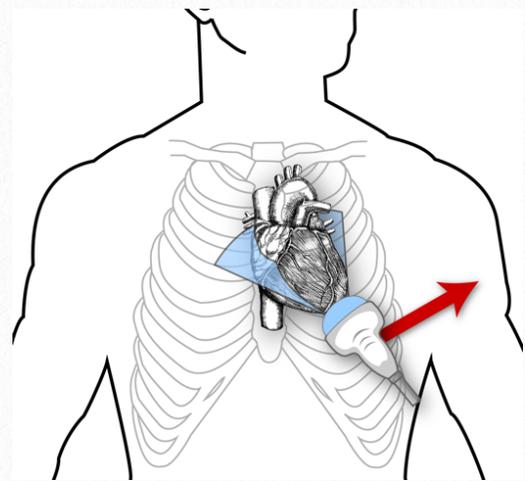
Grade I: First, in the impaired relaxation state (grade I), there is **no elevation of LA pressure**, but rather the LV simply takes more time to fully relax such that there is no diastasis stage. Because the E wave is stretched out over a longer period of time the peak E velocity is reduced and is actually 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 pseudonormalization, the deceleration time will be less than normal (140ms) because the elevated pressure from LA and the elevated pressure in LVEDP cause a more rapid time of pressure equalization.

Grade III: This stage is simply a worsening of 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, this has reacehd 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 alerting the patients 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 <140ms it is severe diastolic function. 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. Please see the table below for further details.

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 felling 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 location of PW doppler signal.

Location of PW Doppler Signal