

# Diastology

Being able to determine diastolic dysfunction of the LV (left sided filling pressure) in patients can help us:

- Assess the fluid status of the patients
- Optimize diuretics management of Diastolic Congestive Heart Failure (HFpEF).
- Differentiate between cardiogenic & non-cardiogenic pulmonary edema aka acute respiratory distress syndrome (ARDS).

First, we will discuss about basic physiology, phases & LV compliance

## Physiology

Diastole starts immediately after systole. The initial relaxation is *isovolumetric*, meaning that relaxation occurs without changes in ventricular volume. The ability of the myocardium to relax during IVRT is governed by left ventricular compliance. The greater the compliance, the better the ability to relax and stretch out during diastole.

During IVRT when the pressure in the left ventricle is lower than left atrial pressure, the mitral valve opens, results in blood flowing into the LV. This defines the second phase of diastole—the **rapid filling**—which can be studied using pulsed Doppler. The sample volume (in apical view) is placed at the tips of the mitral valve leaflets. The rapid blood flow from the left atrium to the left ventricle results in a positive wave called the **E wave**.

If diastolic pressure rises too quickly, left ventricular filling will be reduced and prematurely terminated. If a compensatory increase in left atrial pressure is required to maintain left ventricular filling, pulmonary venous pressure will rise as a result, leading to symptoms.

As blood flows into the LV, the pressure gradient between the LA and the ventricle diminishes, and the passive filling subsides (**The greater the ventricular compliance, the larger the volume of blood that flows from the atrium to the ventricle during this phase. If the ventricle has poor compliance, then the passive filling will cease faster, which is explained by a faster equalization of the pressure gradient.**

## Phases of Diastole

PHASE	EVENT
IVRT (Isovolumetric Relaxation Time)	No Ventricular Filling
First Rapid Passive Ventricular Filling (Rapid Passive Filling)	Passive emptying of the Left Atrium (80%)
Diastasis	No ventricular Filling
Active Atrial Contraction	Active emptying of Left Atrium (20%)

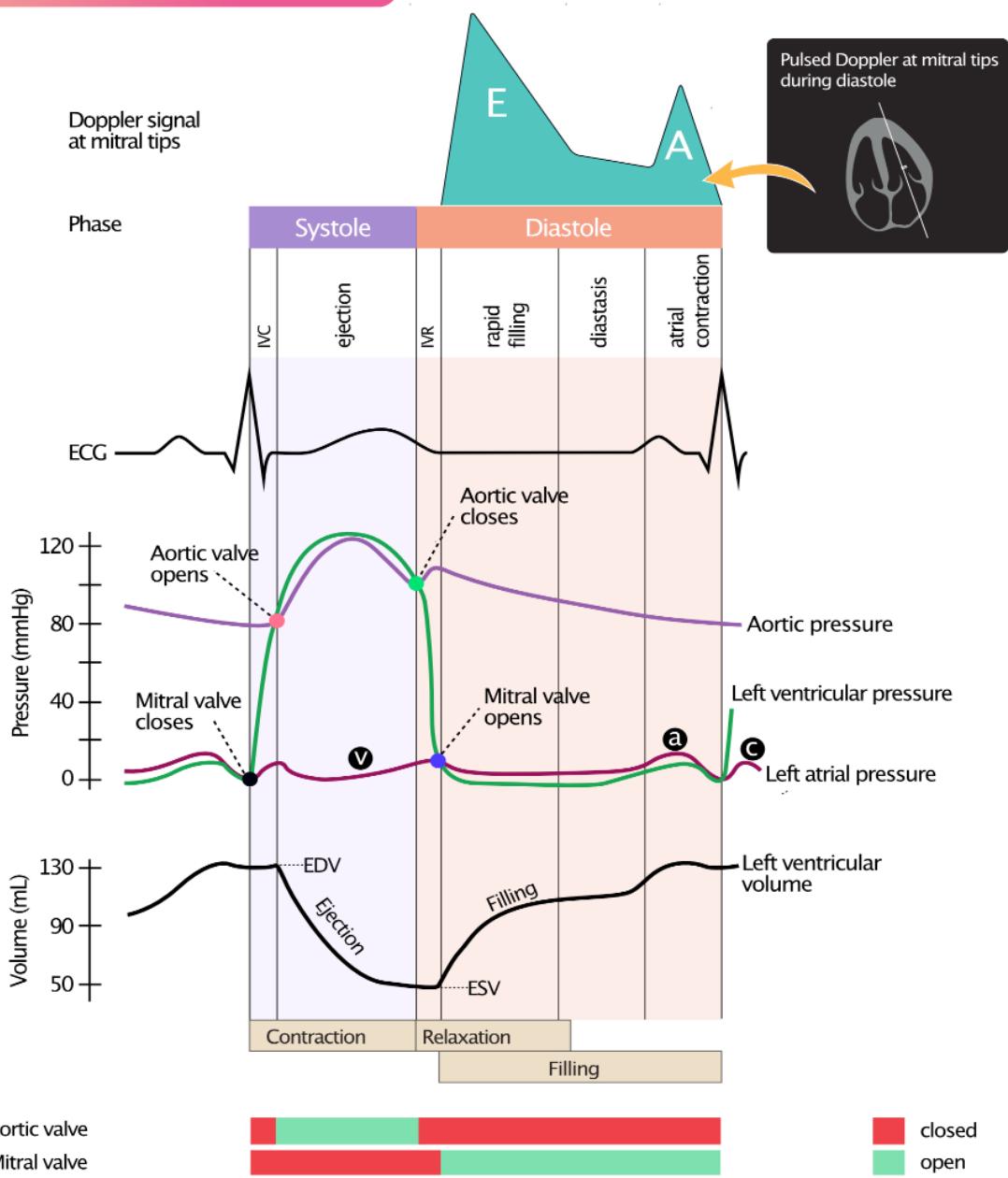
Diastasis ends when the LA starts to contract, which defines the fourth and final phase of diastole. Atrial contraction contributes to the final emptying of the atrium. This gives rise to an **A wave** on the spectral (Doppler) curve.

A modified Wiggers diagram, provides a comprehensive view of the phases and events during the cardiac cycle. Diastole begins when the aortic valve closes and it ends when the mitral valve closes. Systole occurs between mitral valve closure and aortic valve closure. On ECG, the R-wave apex coincides with the onset of systole, and diastole starts at the end of the T wave.

**Here diagram showing pressure, volume, Doppler signal, ECG and AV valves during the cardiac cycle. Doppler recording of mitral valve flow during diastole.**

- (a) = **active atrial filling;**
- (b) = **increased atrial pressure due to bulging of mitral valve into the left atrium, when valve closes;**
- (c) = **passive atrial filling**

### Extended Wiggers diagram



### Left Ventricular Relaxation & Compliance

The entire ventricular myocardium must relax rapidly during diastole, such that the ventricle can expand/relax & refill with blood.

Diastolic function is determined by the efficiency of myocardial relaxation. The degree and velocity of relaxation are the key parameters. Ideally, relaxation should proceed rapidly and the ventricle should expand substantially. This requires that the myocardium has high *compliance*, a term used to describe myocardial elasticity. ***The greater the compliance, the more rapid and pronounced the relaxation (i.e., the stretching of myocardial fibers). The opposite***

*is also true; the stiffer the myocardium, the slower and less pronounced the relaxation.*

### **Factors affecting ventricular compliance:**

- **Age:** In healthy young individuals, the volume of blood transported during the rapid filling phase (E wave) is greater than the volume transported during atrial contraction (A wave). With age, however, the A wave becomes larger, which is explained by the fact that ventricular compliance diminishes and atrial contraction becomes increasingly important for atrial emptying.
- **Afterload:** it is the resistance that the LV must overcome to eject blood into the aorta. It can affect myocardial muscle fibers during systole & diastole. Afterload is the function of the following three variables:
  - Aortic resistance (pressure)
  - LV Volume
  - Ventricular wall thickness

The greater the pressure in the aorta, the greater the load on individual muscle fibers. It can affect as:

- Increase in load on muscle fibers increases ventricular volume.
- Increase load leads to increase wall thickness (hypertrophy).
- There is inverse relationship b/w wall thickness & load, such that greater wall thickness reduces the load on muscle fibers.

***Increased afterload causes impaired diastolic function.***

- **Desynchronization of Myocardial Activation:** Desynchronization of myocardial activation, such as occurs in conditions like bundle branch block (BBB), can lead to impaired ventricular relaxation through several mechanisms:
  - **Altered Electrical Activation:** In conditions like BBB, electrical activation of the ventricles becomes delayed or asynchronous, disrupting the coordinated contraction and relaxation of the myocardium. This delay in electrical activation can lead to delayed onset of relaxation during diastole, leading to inefficient ventricular emptying during systole and impaired ventricular filling during diastole.
  - **Changes in Myocardial Structure and Function:** Chronic desynchronization of myocardial activation can lead to remodeling of the myocardium, including changes in cell size, shape, and collagen deposition. These structural alterations can further impair ventricular relaxation and diastolic function over time.

***Desynchronization of myocardial activation causes impaired ventricular relaxation.***

- **Impaired Relaxation**: ventricular pressure should drop rapidly & substantially during diastole, but if relaxation is impaired, the drop in pressure will be slower & less pronounced. Ultimately leads to increased diastolic pressure in the LV.  
***Impaired relaxation results in increased ventricular diastolic dysfunction.***
- **Myocardial Stiffness**: myocardial stiffness is inversely related to compliance; stiffer the myocardium, the lower the compliance. Stiffness is determined by several factors, e.g.,
  - Ventricular geometry (a large ventricle yields greater afterload & consequently increasing diastolic pressure).
  - Sarcomere structure
  - The composition of extracellular matrix (extracellular fibrosis reduces compliance)
  - Pericardial status, etc.
- **Coronary Artery Disease**
- **Restrictive cardiomyopathy**
- **Myocardial Infiltration**
- **Pericardial Constriction**

## Assessment of Diastolic Function

### **Machine & Patient Preparation for Exam:**

Place the machine on the right side of the patient so you can scan with your right hand & manipulate ultrasound buttons with your left hand.

- Transducer: Phased array Probe
- Preset: Cardiac
- The indicator should be on the RIGHT side of the screen (cardiac mode).
- Place patient supine or left lateral decubitus position.
- Acquire the A4C view of the heart.
- watch video <https://www.youtube.com/watch?v=wxTHn5lvgXY>

In order to measure blood flow into the left ventricle & how well the left ventricular muscle is relaxing, we will be using Doppler function of the ultrasound machine.

The American Society for Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) stress that the following parameters are of particular importance for the evaluation of diastolic function:

- The ratio between **E-wave** and **A-wave (E/A ratio)**. The E/A ratio is derived by measuring flow velocities across the mitral valve using pulsed Doppler.
- Estimating left ventricular filling pressure via **e'**.

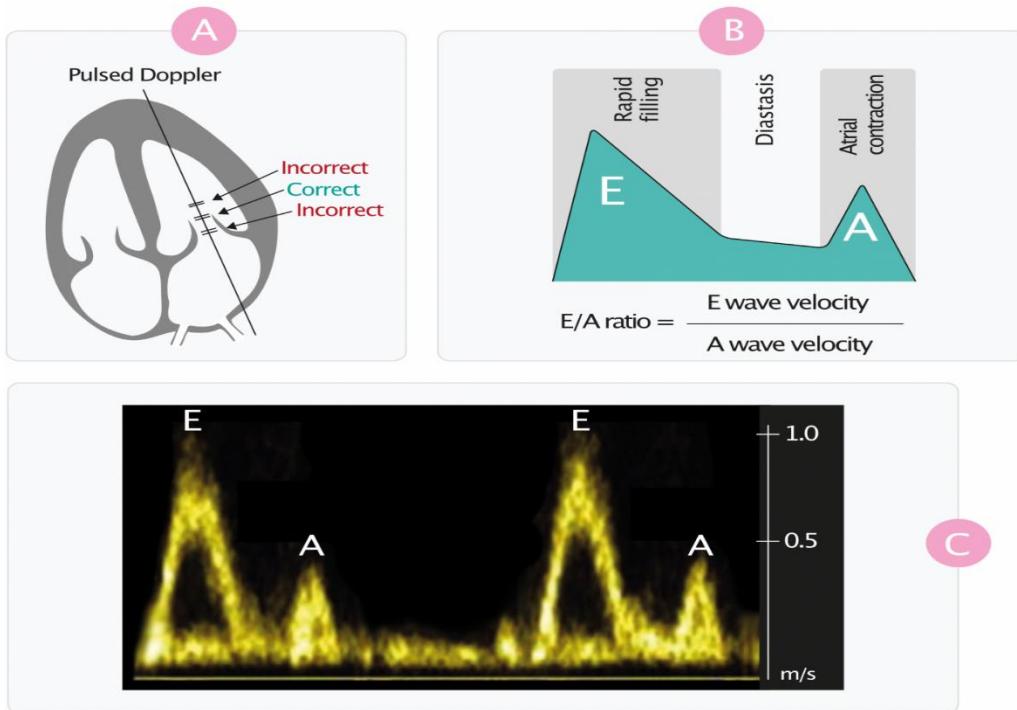
- Measurement of **deceleration time (DT)**.

These three methods, as well as several supplementary methods, will now be discussed in detail.

### **Using PULSE WAVE DOPPLER to measure BLOOD FLOW:**

Pulse wave doppler allows us to measure the velocity of blood flow (at a single point) as it enters the LV (there are 2 patterns of diastolic dysfunction: slow-relaxation pattern, restrictive pattern). A unique aspect of PWD is that we can specify to the ultrasound machine exactly where we would like the machine to measure the velocity using

**Sample Gate.** It is usually seen by two Horizontal lines along cursor. We can move cursor & sample gate & place it exactly where we want to measure blood velocity.

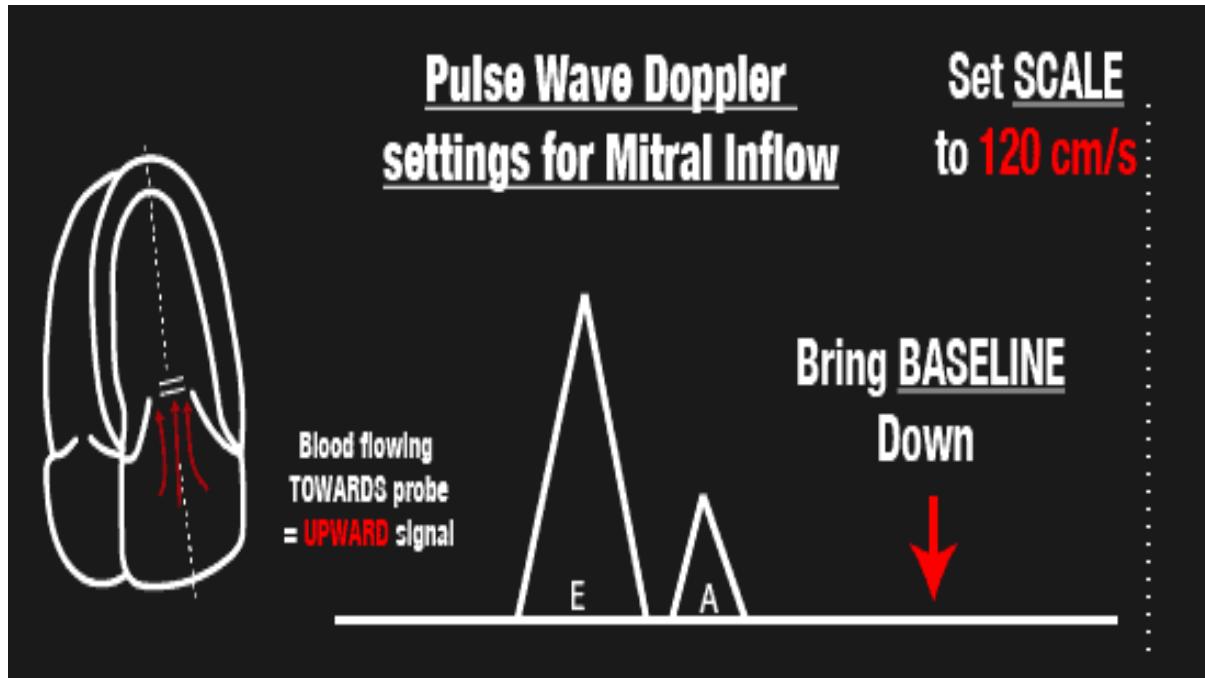


*Just keep it in mind that doppler just measures the speed of blood flow or speed of tissue movement either towards or away from the ultrasound probe. All the doppler modes do this, whether it is color doppler, PWD, CWD, Tissue Doppler. Concepts are all the same.*

*So, if blood flows towards the sample gate we will get a positive deflection above the baseline, and if blood flows away from the sample gate, we will get a negative deflection.*

In A4C view blood is flowing towards the LV from LA, so there will be UPWARD deflection of PWD signal. This will result in E & A waves.

Here we also need to optimize the baseline & scale of our PWD settings to best capture accurate mitral inflow signals. We can do this by bringing baseline towards the bottom of the screen & setting the scale to about 100-120cm/s. now we can start measuring the mitral inflow velocities to asses diastolic dysfunction. See video [https://youtu.be/\\_YXBCQldH1E](https://youtu.be/_YXBCQldH1E).



### The Push & Pull Phenomenon of Early Diastolic Filling (the E wave)

Blood can either be **passively pulled** through the mitral valve, by rapidly lowering left ventricular pressure below left atrial pressure (suction), or **actively pushed** through the valve by raising atrial pressure above ventricular pressure.

Both occur in the normal heart. In early diastole, flow is initiated by the rapidly relaxing left ventricle resulting in a suction of blood from the left atrium, through the mitral valve. In late diastole, the continued forward flow of blood is accomplished by a pushing mechanism, the result of atrial contraction.

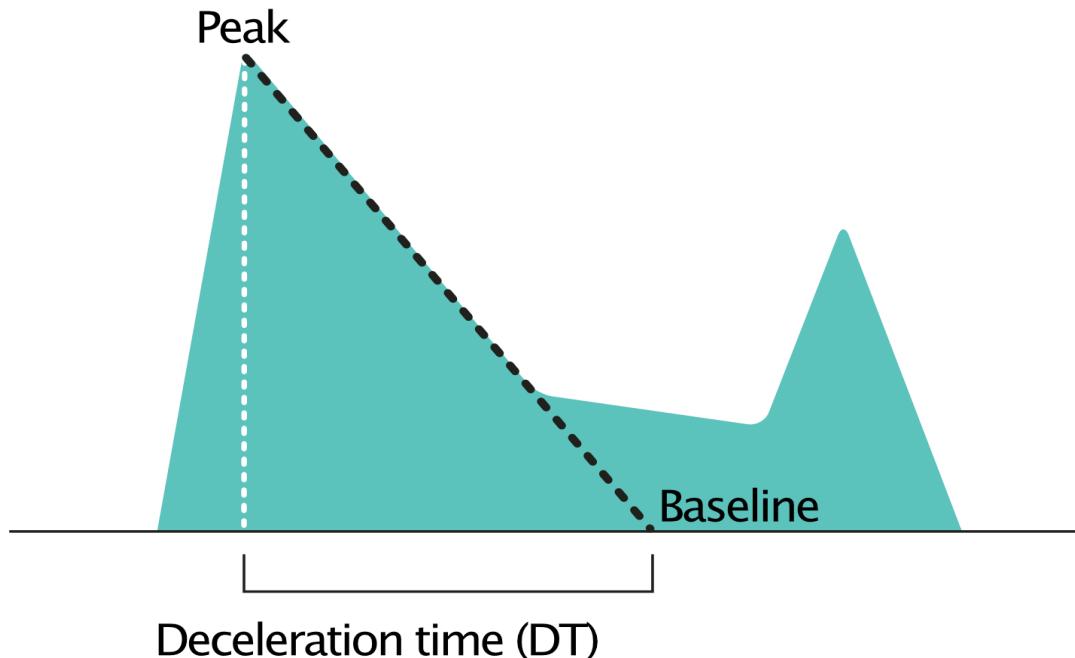
### **Deceleration time (DT)**

- The normal E-wave displays a rapid acceleration (ascending part) and rapid deceleration (descending part). Deceleration time (DT) is the time interval from the peak of the E-wave to its projected baseline.

**The E-wave deceleration time is normally between 150 ms and 240 ms**

- The deceleration time indicates the duration for equalizing the pressure difference between the LA and the LV. The deceleration time is prolonged in conditions leading to a delayed equalization of the pressure gradient.

Conversely, the deceleration time is shortened if left ventricular compliance is reduced, or if left atrial pressure is increased.



### Mitral E-wave velocity

The E-wave represents passive blood flow from the LA to the LV. This flow is propelled by the pressure gradient between the LA and the LV. The amplitude and form of the E-wave reflect the velocity and course of the flow. The main factors determining E-wave velocity and form are the following:

- **The pressure gradient** between the LA & the LV.
- **Left ventricular compliance** (i.e., the ability of the ventricle to relax and stretch out during diastole).

**Normally, the peak E-wave velocity is between 0.6 and 0.8 m/s and it occurs approximately 100 ms after the start of the E-wave.**

**Diastasis:** The phase following the E-wave is the **diastasis**, during which there is no significant flow across the mitral valve. The duration of diastasis is inversely related to heart rate (diastasis is shorter at higher heart rates and *vice versa*). The diastasis may disappear at very high heart rates.

**Mitral A-wave velocity:** The mitral A-wave reflects blood flow generated by active atrial contraction. The velocity and form of the A-wave are determined by atrial contractility and left ventricular compliance.

**Peak A-wave velocity is normally 0.2 ms/s to 0.35 m/s.**

**Mitral E/A ratio:** The ratio between the E-wave and the A-wave is the E/A ratio. Since the E-wave is normally larger than the A-wave, the ratio should be  $>1$ . The

E/A ratio is age-dependent. The E-wave becomes smaller and the A-wave becomes larger with age. At the age of 60 to 70 years, the E-wave and A-wave display similar amplitudes. Deceleration time and IVRT are also prolonged with age.

**Reduced E/A ratio is a hallmark of diastolic dysfunction.**

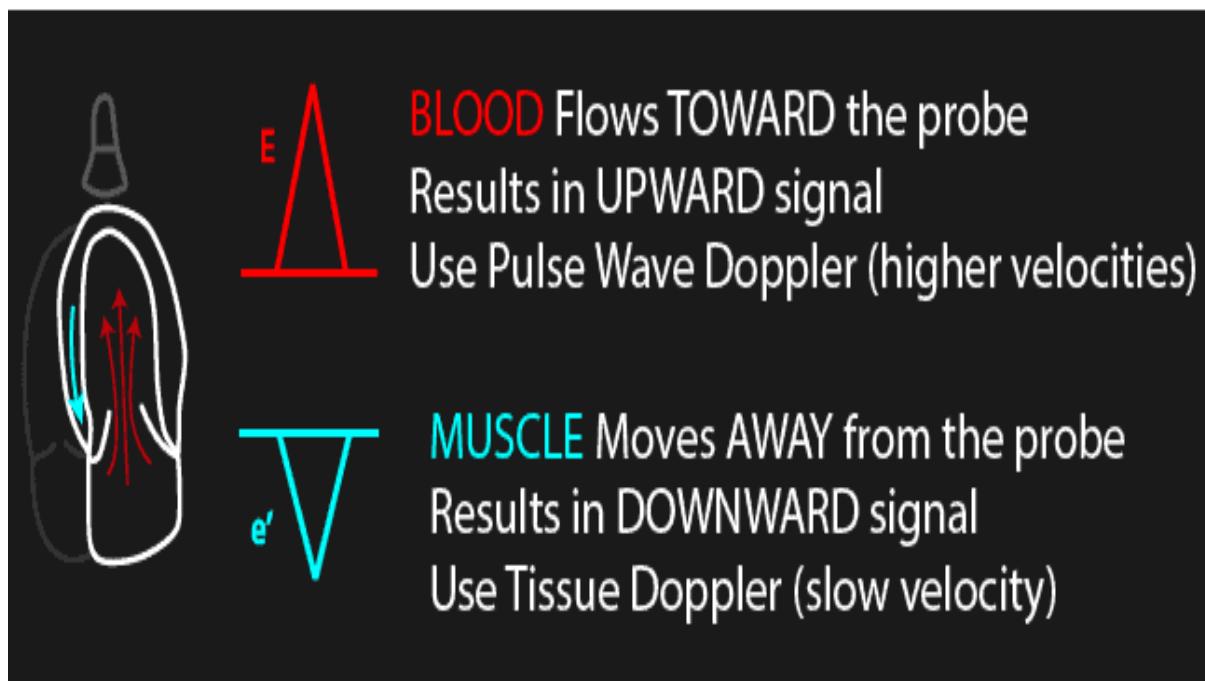
Reduced passive filling (i.e., smaller E-wave), which leaves a larger blood volume for the atrium to eject during contraction (i.e., larger A-wave).

**Using TISSUE DOPPLER to measure LV MUSCLE MOVEMENT:**

Tissue Doppler is just another form of PWD that allows us to measure the much slower speeds of tissue/muscle movement (from 1cm/s – 20cm/s) compared to PWD that measures the much faster speed of blood (30cm/s – 200cm/s).

For diastolic dysfunction, we will be using TDI to specifically measure the speed of LV muscle relaxation during diastole. This will allow us to see how "stiff" the LV is. A normal heart will relax quickly during diastole & stiff heart will have a slower speed of muscle relaxation.

The direction of diastolic LV movement (at the mitral annulus) is actually **AWAY** from the probe as it expands to allow blood to fill the LV. This is why we get downward signal when measuring the left ventricle using TDI.



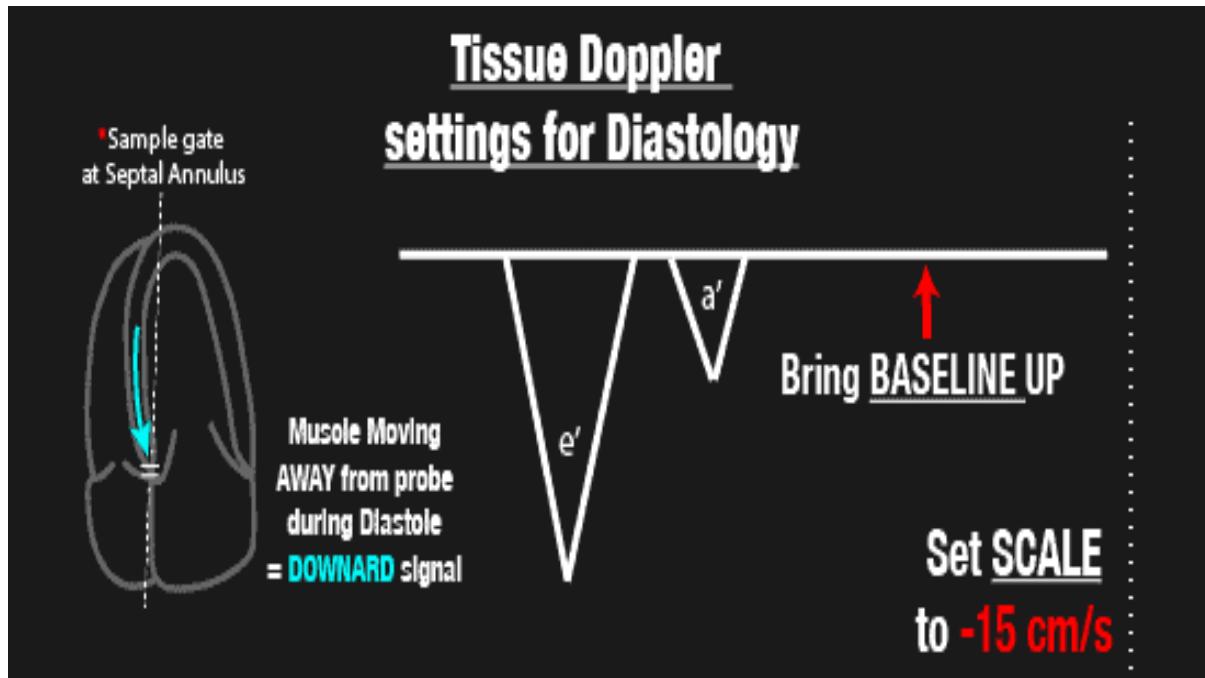
**Setting the Tissue Doppler Sample Gate, Baseline & Scale:**

**Sample Gate:** There are two validate areas where we can place the Tissue Doppler sample gate are at septal or lateral portions of the mitral annulus. Septal

annulus is usually easier to obtain and not obscured by lung parenchyma (refers to functional tissues of lungs like alveoli other authors may include interstitial tissue into it) like the lateral annulus may be.

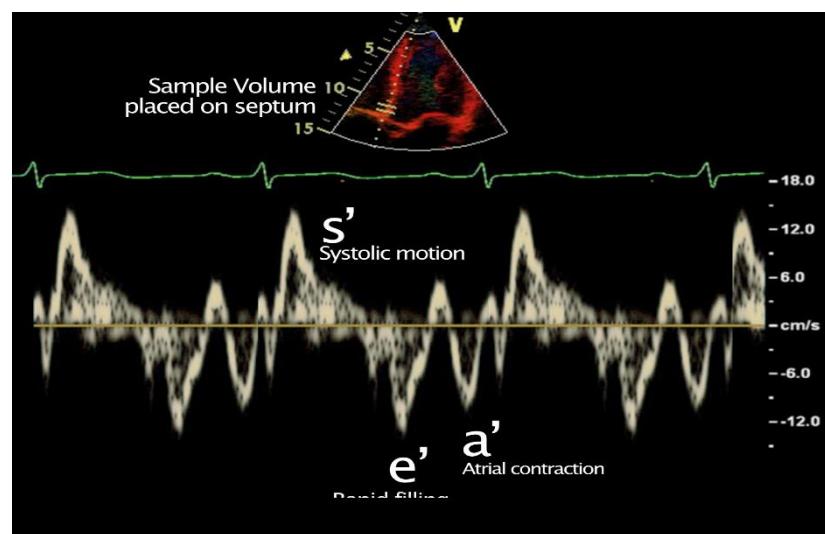
**Baseline:** for tissue doppler baseline, bring it up towards the top of the screen since we are looking for a negative deflection. Set the scale to around -15cm/s (muscle has lower velocity).

Watch video <https://youtu.be/agndqftTpqo>



### Mitral annular velocity with tissue Doppler imaging (TDI)

Two dominant waves are observed during diastole, namely **e'** and **a'**. These waves reflect the same events as the E-wave and A-wave, respectively.



**The mitral annular velocity during systole and diastole, recorded with tissue Doppler sampling 1 cm below the mitral plane. The sample volume can be placed either medially or laterally.**

Mitral annular velocities can be measured medially (i.e., in the septum) or laterally. Medial velocity is normally <8 cm/s and lateral velocity is normally <10 cm/s. Young healthy people may display higher velocities. If medial and lateral velocities are measured, the mean value is used.

$$\text{Medial velocity} + \text{Lateral velocity} / 2$$

### **E/e' ratio and LVEDP**

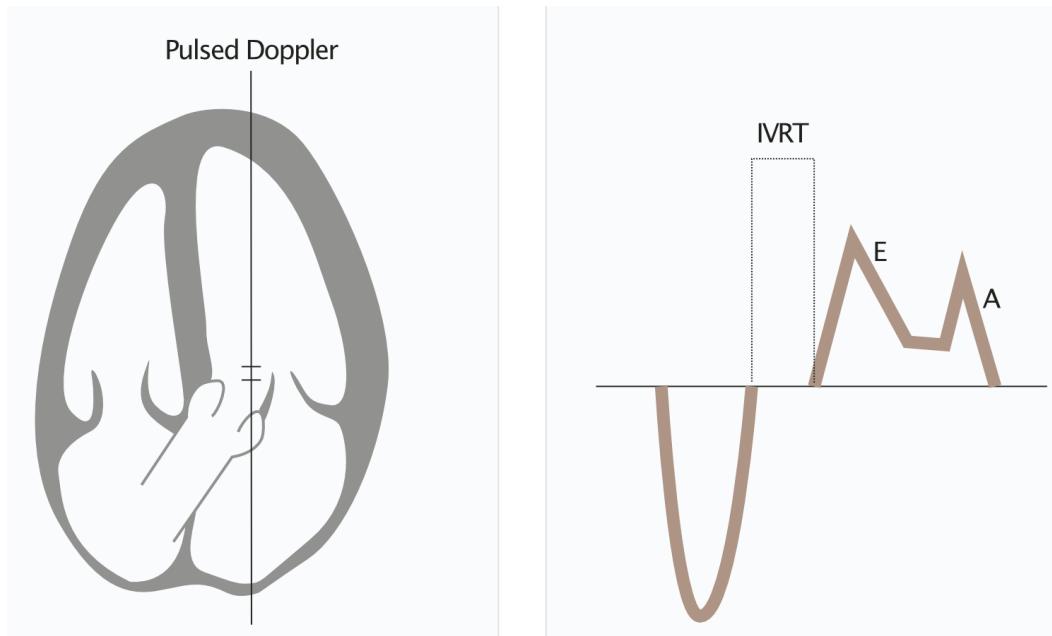
- By dividing the peak E-wave velocity by the peak e' velocity, left ventricular end-diastolic pressure (LVEDP) can be estimated. This is the E/e' ratio and it is normally <15. Values greater than 15 suggest that LVEDP is elevated. Diastolic dysfunction leads to larger E/e' ratio, which is explained by the fact that diastolic dysfunction leads to impaired ventricular relaxation, and thus a smaller e' wave. Conversely, the E-wave tends to become larger at elevated LVEDP.

Currently, the ASE recommends measuring the E/e' [**E/ (e' medial + e' lateral)/2**] ratio both medially (septal) and laterally, and calculate the mean value. An E/e' ratio (mean value)  $\geq 13$  suggests elevated ventricular filling pressure (LVEDP). Note that the E/e' ratio is less reliable in the following situations:

- Healthy individuals
- Left ventricular dysfunction
- Constrictive pericarditis
- Mitral valve disease (mitral valve regurgitation, mitral valve stenosis)
- Mitral valve surgery
- Acute heart failure
- CRT treatment
- Hypertrophic obstructive cardiomyopathy (HOCM)

### **IVRT (isovolumetric relaxation time)**

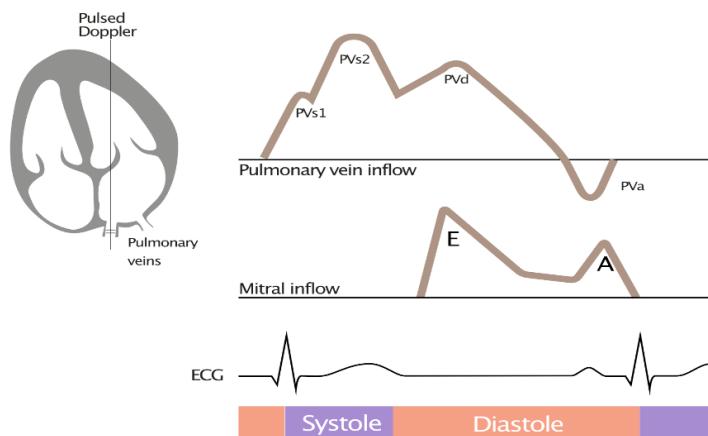
IVRT is the period from aortic valve closure to mitral valve opening. IVRT is measured in apical five-chamber view (A5C), using pulsed Doppler. Sample volume is placed between the aortic valve and the mitral valve, which allows it to record both the closure of the aortic valve and the opening of the mitral valve.



**Pulmonary vein flow:** Pulmonary vein velocities are difficult to perform and they should be considered as supplementary methods.

The pulmonary veins transport oxygen-rich blood from the lungs to the left atrium. The pulmonary veins have no valves. Flow through the pulmonary veins can be used as a supplementary method to examine diastolic function. The pulmonary veins are visualized in apical four-chamber view (A4C). The upper right pulmonary vein is often easiest to visualize. Pulmonary vein velocities are recorded using pulsed Doppler, with sample volume located approximately 1 cm into the pulmonary vein.

During systole, two positive waves may be seen, namely **PVs1** and **PVs2**. PVs1 and PVs2 may be fused, making them difficult to distinguish. PVs1 and PVs2 represent flows into the left atrium during systole. The force behind PVs1 is atrial relaxation, which leads to lower atrial pressure. PVs2 is due to an increase in pressure in the pulmonary circulation, caused by right ventricular contraction. **PVd** occurs during diastole and coincides with the mitral E-wave. For simplicity, PVs1 and PVs2 are henceforth referred to as **PVs**.



The final wave, **PVa** (or **PVAR**) is negative, which implies that the flow is reversed (blood flows from the left atrium back into the pulmonary vein). This reversal of flow is caused by atrial contraction, which squeezes blood back into the pulmonary veins.

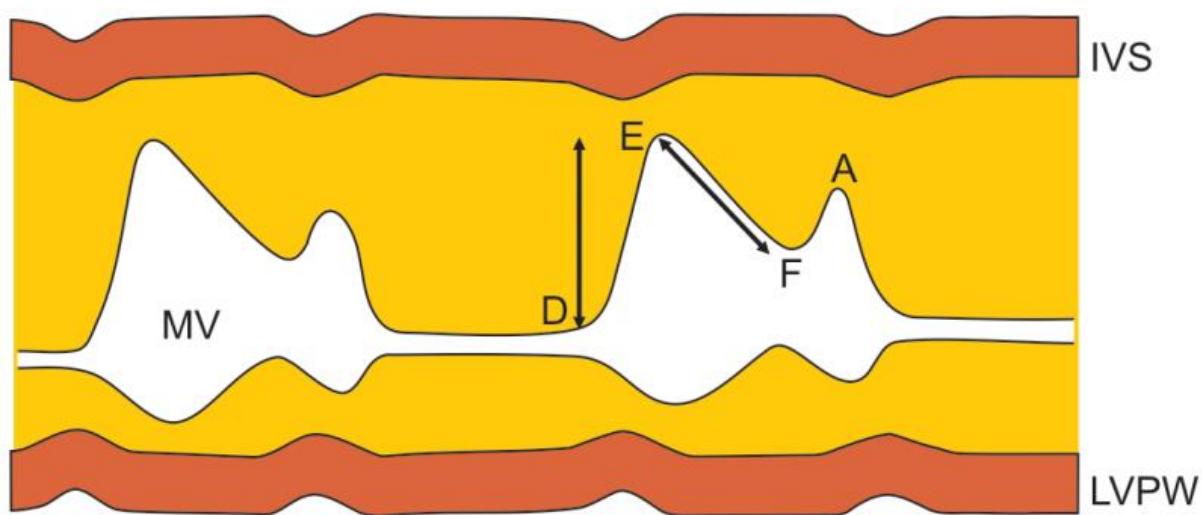
PVs is normally larger than PVd, leading to a PVs/PVd ratio  $>1$ . Diastolic dysfunction results in increased atrial pressure, which affects left atrial filling. This leads to characteristic changes in PVs and PVd, such that PVs become smaller and PVd becomes larger, leading to a PVs/PVd ratio  $<1$ . It should also be noted that young people may display a PVs/PVd ratio  $<1$  as a normal finding.

If left ventricular compliance decreases, the atrium will encounter a higher resistance to atrial contraction, resulting in increased reversed flow in the pulmonary vein. This yields a larger and wider PVa wave. PVa velocity greater than  $>35$  cm/s suggests increased left ventricular end-diastolic filling pressure. PVa duration becomes longer than mitral A-wave duration. If the difference between the PVa duration and the A-wave duration is  $>30$  ms, end-diastolic filling pressure is likely  $>20$  mmHg.

### Echo features of LV Diastolic Dysfunction

**M-Mode MV Level:** motion of the AML during normal diastole has a characteristic M-shape (E-A pattern). In the presence of LV diastolic dysfunction, AML excursion is diminished, a wave is taller than the E wave & E:A ratio is reduced.

These signs are neither highly sensitive nor specific for the presence of Diastolic Dysfunction.



### Pitfalls in the Diagnosis of LV Diastolic Dysfunction

- The mitral inflow pattern of LV filling is influenced by a large number of factors besides myocardial relaxation & distensibility. Therefore, it is

inappropriate to rely only on E:A ratio as an indicator of LV diastolic dysfunction.

- Factors that influence the mitral inflow pattern include:
  - Volume loading (preload & afterload)
  - Heart Rate & cardiac rhythm
  - Left atrial systolic function
  - The phase of respiration
- Volume overloading due to mitral or aortic regurgitation attenuates the A wave since atrial contraction cannot affect forward flow if the ventricle is already maximally distended. The E wave peak is also increased.
- In the presence of tachycardia, A wave is more prominent since the diastole is shortened (greater atrial contribution). When there is bradycardia, A wave is small since diastolic filling is prolonged (lesser atrial contribution). Therefore, a tall A wave carries greater significance in the presence of bradycardia.
- The E:A ratio, as an indicator of diastolic dysfunction, is invalid in the presence of atrial fibrillation (there is no atrial contribution to ventricular filling), CHB (CHB with A-V dissociation, the E wave & A wave occur at different times) or a prolonged P-R interval (the E wave & A wave occur at the same time & appear as a single wave).
- In an elderly person, the A wave is dominant. If the E:A ratio is  $>1$  or E=A with short deceleration time (DT), it indicates the presence of an elevated LVEDP. This is referred as pseudo-normalization of MV inflow pattern.

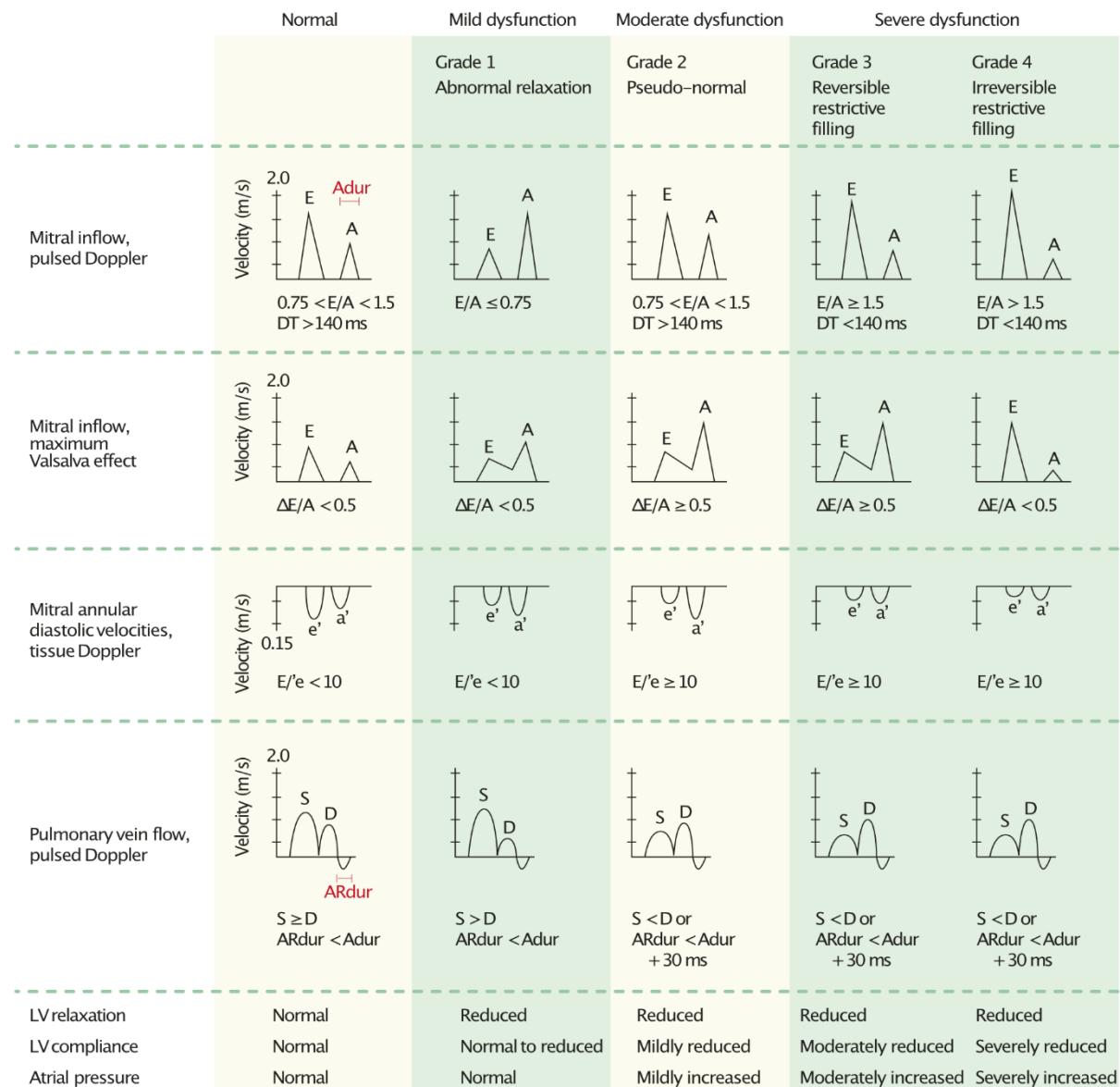
## Grading of Diastolic Dysfunction

- **Grade 1 diastolic dysfunction (abnormal relaxation)** – this condition is characterized by a E/A ratio is  $<1$ . The deceleration time is prolonged (usually  $>240$  ms) and IVRT is  $>90$  ms (a normal IVRT is about **70 ± 12 ms** (approximately 10 ms longer in people over forty years).
- **Grade 2 diastolic dysfunction (pseudo normal pattern)** — Mitral inflow displays normal appearance, with E/A ratio between 1 and 1.5. The deceleration time is between 150 and 200 ms and IVRT  $>90$  ms
- **Grade 3 diastolic dysfunction (restrictive filling)** — This condition is characterized by high E-wave amplitude and low A-wave amplitude, with reduced deceleration time ( $<150$  ms). E/A ratio is  $>2$ . IVRT is  $>70$  ms . Restrictive filling is defined as either irreversible or reversible, depending on whether the pattern disappears during Valsalva maneuver. If the pattern is normalized during Valsalva maneuver, it is classified as reversible restrictive filling.

- If a grade 3 pattern persists despite Valsalva maneuver, then it is classified as **irreversible restrictive filling**, which also defines **grade 4 diastolic dysfunction**.

### Valsalva maneuver and assessment of diastolic function

- Valsalva maneuver can be performed to further evaluate diastolic dysfunction. The Valsalva maneuver is performed by moderately forceful attempted exhalation against a closed airway, usually done by closing one's mouth, pinching one's nose shut while expelling air out as if blowing up a balloon. This leads to decreased preload and reduced left atrial pressure. The peak E-wave and A-wave velocities decrease by roughly 20%.*
- The Valsalva maneuver is useful because the reduction in preload and left atrial pressure affects the E/A ratio in a characteristic way, depending on the degree of diastolic dysfunction. Individuals with pseudonormal pattern (grade*



*2 diastolic dysfunction) will exhibit grade 1 dysfunction (abnormal relaxation) when performing the maneuver. Individuals with grade 3 dysfunction (restrictive filling) may exhibit grade 1 dysfunction (abnormal relaxation) or grade 2 dysfunction (pseudonormal pattern). If a grade 3 dysfunction is not affected by the maneuver, the condition is classified as grade 4 diastolic dysfunction (irreversible restrictive filling).*

## **Grade 0 – Normal Diastolic Function**

### **Mitral Inflow Pattern for Grade 0 Normal Diastolic Function**

- There are two mitral inflow waves that represent blood flow coming into the left ventricle. The first is the **E wave** (early diastolic filling) and the second is the **A wave** (atrial kick). You just have to remember that capital E and A represent mitral inflow patterns using Pulse Wave Doppler.

### **Grade 0: Normal Mitral Inflow**

- E wave: majority of blood flow resulting from Passive PULL of left ventricle relaxation (large E wave)
- A wave: Atrial kick with small amount of Blood Flow (small A wave)
- **E/A  $\geq 0.8$**

### **Tissue Doppler Pattern for Grade 0 Normal Diastolic Function**

- There are also two Tissue Doppler waves and they are **e' wave** (early diastolic filing) and **a' wave** (atrial kick). They are referred to as "e prime" and "a prime." Luckily, only the e' wave is commonly needed to assess for diastolic dysfunction. Recall that we will be using the SEPTAL e' and its corresponding values for this post.
- Since the myocardium of the left ventricle is relaxing normally, you will expect it to relax/expand quickly, hence the e' velocity on Tissue Doppler will be relatively quick (compared to someone with abnormal diastolic function) and you will expect a large negative deflection of the e' wave ( $\geq 8\text{cm/s}$  for septal e'). *(Note: Technically it would be -8cm/s since it is a negative deflection but e' is commonly referred to in absolute values. Also, this is assuming you use the septal e'. If you use the lateral annulus e' the normal value is  $\geq 10\text{ cm/s}$ )*

### **Grade 0: Tissue Doppler**

- Normal LV muscle relaxation
- **e'  $\geq 8\text{cm/s}$**  (septal annulus)

### **E/e' Ratio for Grade 0 Normal Diastolic Function**

- The E/e' ratio has been found to be fairly sensitive in evaluating the degree of diastolic dysfunction. The LOWER the ratio the better the diastolic function. This makes sense since the ratio is inversely proportional to the e'. So, the higher the e' (better diastolic function/muscle relaxation) the lower E/e' ratio.
- A patient with Grade 0 Diastolic Function should have an **E/e' ratio < 8**.

For Summary See Table.

## **Grade 1 Diastolic Dysfunction: Impaired Relaxation**

### **Mitral Inflow Pattern for Grade 1 Diastolic Dysfunction**

- The first stage of diastolic dysfunction is impaired relaxation of the left ventricle. This results in a decrease of the normal "PULL" during early ventricular diastole and you will end up seeing a smaller amplitude E wave. The remaining blood is then pushed into the left ventricle during the atrial kick, resulting in a larger A wave.
- So, Grade 1 Diastolic Dysfunction will give you a very distinct mitral inflow pattern with an E/A ratio  $<0.8$ . It's actually the ONLY mitral inflow pattern that has a ratio below 0.8. So, if you see this, your patient has Grade 1 Diastolic Dysfunction. You can still measure the e' measurements if desired, but it is not necessary.
- (*Note: Just be aware that Grade 1 Diastolic Dysfunction can be a normal finding as patients age.*)

### **Grade 1 Diastolic Dysfunction: Mitral Inflow**

- **E wave:** Decreased "PULL" from left ventricle due to impaired relaxation (small E wave).
- **A wave:** Atrial kick pushes in remaining excess blood (large A wave)
- **E/A < 0.8**

### **Tissue Doppler Pattern for Grade 1 Diastolic Dysfunction**

- Since the left ventricular muscle relaxation is impaired in Grade 1 Diastolic Dysfunction, you will see an **e' < 8cm/s**. As diastolic dysfunction grade worsens you will see even smaller amplitude e' velocities in Grade 2 and Grade 3 diastolic dysfunction.

### **Grade 1 Diastolic Dysfunction: Tissue Doppler**

- Impaired left ventricular muscle relaxation
- **e' < 8cm/s**

### **E/e' Ratio for Grade 1 Diastolic Dysfunction**

- A patient with Grade 1 Diastolic Dysfunction will have an  **$E/e'$  ratio  $\leq 8$** . This is because both the E and e' waves decrease and the relative ratio is preserved.
- Since both Grade 0 and Grade 1 Diastolic Dysfunction will have  $E/e' \leq 8$ , you will have to rely on your E/A ratio. Grade 0 diastolic function will have  $E/A \geq 0.8$  and Grade 1 diastolic dysfunction will have an E/A ratio of  $< 0.8$ .

For summary see Table.

## **Grade 2 Diastolic Dysfunction: Pseudonormal**

*The primary problem in **Grade 2 Diastolic Dysfunction** is worsening left ventricular stiffness leading to increased Left Atrial Pressure +/- Left Atrial Enlargement.*

### **Mitral Inflow Pattern for Grade 2 Diastolic Dysfunction: Pseudonormal**

- As diastolic dysfunction progresses and the left ventricle becomes stiffer, the left atrial pressures start to increase to accommodate the higher left ventricular diastolic pressures.
- Therefore, once the mitral valves open in a patient with Grade 2 diastolic dysfunction, the elevated left atrial pressures will cause a significant amount of blood to flow into the left ventricle during early diastolic filling. This will result in an increased E wave on your mitral inflow pattern.
- The KEY here is that the increased E wave is due to an **increased PUSH from the left atrium**, NOT from the PULL of the left ventricle (normal heart). This is what results in the term "Pseudo normalization" because the E and A waves pattern look like a normal waveform ( $E/A \geq 0.8$ ).
- So, it will be necessary to also perform Tissue Doppler whenever you have a "normal" looking E/A ratio to be able to differentiate between Grade 0 versus Grade 2 Diastolic Dysfunction.

### **Grade 2 Diastolic Dysfunction: Mitral Inflow**

- **E wave:** Increased "PUSH" from left atrium due to increased Left Atrial Pressure (large E wave)
- **A wave:** Most blood pushed in during early diastole (small A wave)
- **$E/A \geq 0.8$**  (looks like Grade 0)

### **Tissue Doppler Pattern for Grade 2 Diastolic Dysfunction**

- As the left ventricle becomes stiffer there will be slower relaxation resulting in a slower e'. A decrease in e' ( $< 8\text{cm/s}$ ) is the defining feature between Grade 0 (normal) and Grade 2 Diastolic Dysfunction.

### **Grade 2 Diastolic Dysfunction: Tissue Doppler**

- Moderately Reduced LV muscle relaxation
- **e' < 8cm/s**

### **Grade 2 Diastolic Dysfunction: Tissue Doppler**

- Severely reduced LV muscle relaxation
- **e' << 8cm/s**

### **E/e' Ratio for Grade 2 Diastolic Dysfunction**

- A patient with Grade 2 Diastolic Dysfunction will have an **E/e' ratio between 8 -15**.

## **Grade 3 Diastolic Dysfunction: Restrictive**

*The primary problem in Grade 3 Diastolic Dysfunction is continued worsening left ventricular stiffness leading to SEVERE increased Left Atrial Pressure with Left Atrial Enlargement.*

### **Mitral Inflow Pattern for Grade 3 Diastolic Dysfunction**

- As diastolic dysfunction becomes more chronic and the left ventricle becomes stiffer, the left atrial pressures must increase to accommodate the higher left ventricular diastolic pressures. This is especially seen in Grade 3 Diastolic Dysfunction or a restrictive filling pattern.
- In Grade 3 diastolic dysfunction, there is severe increase in the left atrial pressure due to the significant decrease in left ventricular compliance.

In the early diastolic filling of a patient with Grade 3 diastolic dysfunction, almost all of the blood will fill the left ventricle from the **PUSH** of the extremely high pressures in the left atrium. You should see a very large and narrow E wave with an E/A ratio  $\geq 2$ .

### **Grade 3 Diastolic Dysfunction: Mitral Inflow**

- **E wave:** Significant Increased "PUSH" from left atrial due to Severely increased Left Atrial Pressure (large and narrow E wave)
- **A wave:** small A wave
- **E/A  $\geq 2$**

### **Tissue Doppler Pattern for Grade 3 Diastolic Dysfunction**

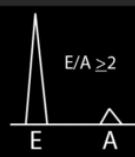
- In Grade 3 Diastolic Dysfunction, the muscle of the left ventricle will have severely reduced motion given the non-compliance of the LV. This will result in a very small e' wave.

## E/e' Ratio for Grade 3 Diastolic Dysfunction

- Since the E wave will be elevated and the e' wave will be depressed, a patient with Grade 3 Diastolic Dysfunction will have an **E/e' ratio > 15**.

For summary see Table

Watch Video <https://youtu.be/76xiXuN9tqA?si=-oC5r3KD28OZpnvC>

<u>INTERPRETATION:</u>	Grade 0 (Normal)	Grade 1 (Impaired Relaxation)	Grade 2 (Pseudonormal)	Grade 3 (Restrictive)
<b>MITRAL INFLOW</b> Measures <b>BLOOD FLOW</b> coming into the Left Ventricle  PULSE Wave gate at Mitral Valve Tips	E/A $\geq 0.8$ e' $\geq 8 \text{ cm/s}$ E/e' $< 8$	E/A $< 0.8$ e' $< 8 \text{ cm/s}$ E/e' $< 8$	E/A $\geq 0.8$ e' $< 8 \text{ cm/s}$ E/e' $8 - 15$	E/A $\geq 2$ e' $<< 8 \text{ cm/s}$ E/e' $> 15$
<b>TISSUE DOPPLER</b> Measures <b>MUSCLE MOVEMENT</b> of the Left Ventricle AWAY from probe during Diastole  Tissue Doppler gate at Septal Annulus	 	 	 	 

 POCUS 101