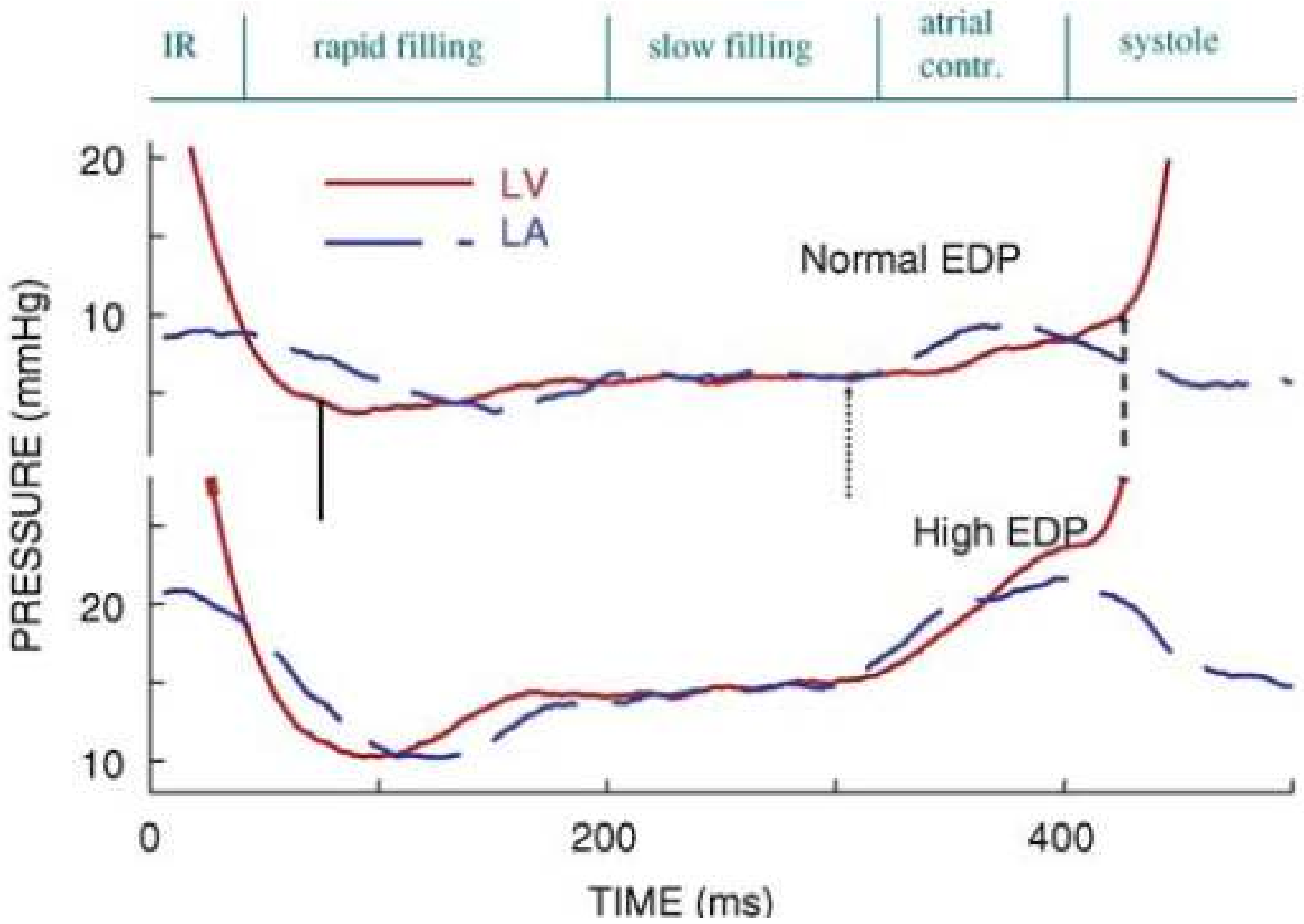
EAE/ASE RECOMMENDATIONS

Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography

J Am Soc Echocardiogr 2009;22:107-133

Eur J Echocardiogr 2009;10:165-193


IR
rapid filling
slow filling
atrial contr.
systole
LV
LA
Normal EDP
High EDP
PRESSURE (mmHg)
20
10
20
10
0
200
400
TIME (ms)

- The optimal performance of the left ventricle depends on

1. A compliant chamber in diastole that allows the left ventricle to fill from low LA pressure
2. A stiff chamber (rapidly rising pressure) in systole that ejects the stroke volume at arterial pressures

- Diastole starts at aortic valve closure and included LV pressure fall, rapid filing, diastasis (at slower heart rates) and atrial contraction

- Elevated filling pressures are the main physiologic consequence of diastolic dysfunction

- Filling pressures are considered elevated when
  - The mean PCWP > 12 mmHg or
  - The LVEDP > 16 mmHg

Morphologic and functional correlates of diastolic dysfunction

- LV hypertrophy
- LA volume
- LA function
- Pulmonary artery systolic and diastolic pressures

LV hypertrophy

- *Hypertensive heart disease* is the most common abnormality leading to diastolic heart failure.

- In pathologic hypertrophied myocardium, LV relaxation is usually slowed, reducing early diastolic filing

LA volume

- Using apical 4-chamber and 2-chamber views
- A significant relation between LA remodeling and echocardiographic indices of diastolic function
- LA volume index > 34 ml/m^2
  - An independent predictor of death, heart failure, atrial fibrillation and stroke
- Dilated left atrium also in the absence of diastolic dysfunction
  - Bradycardia and 4 chamber enlargement
  - Anemia and other high output states
  - Atrial flutter or fibrillation
  - Significant mitral valve disease
  - Athletes

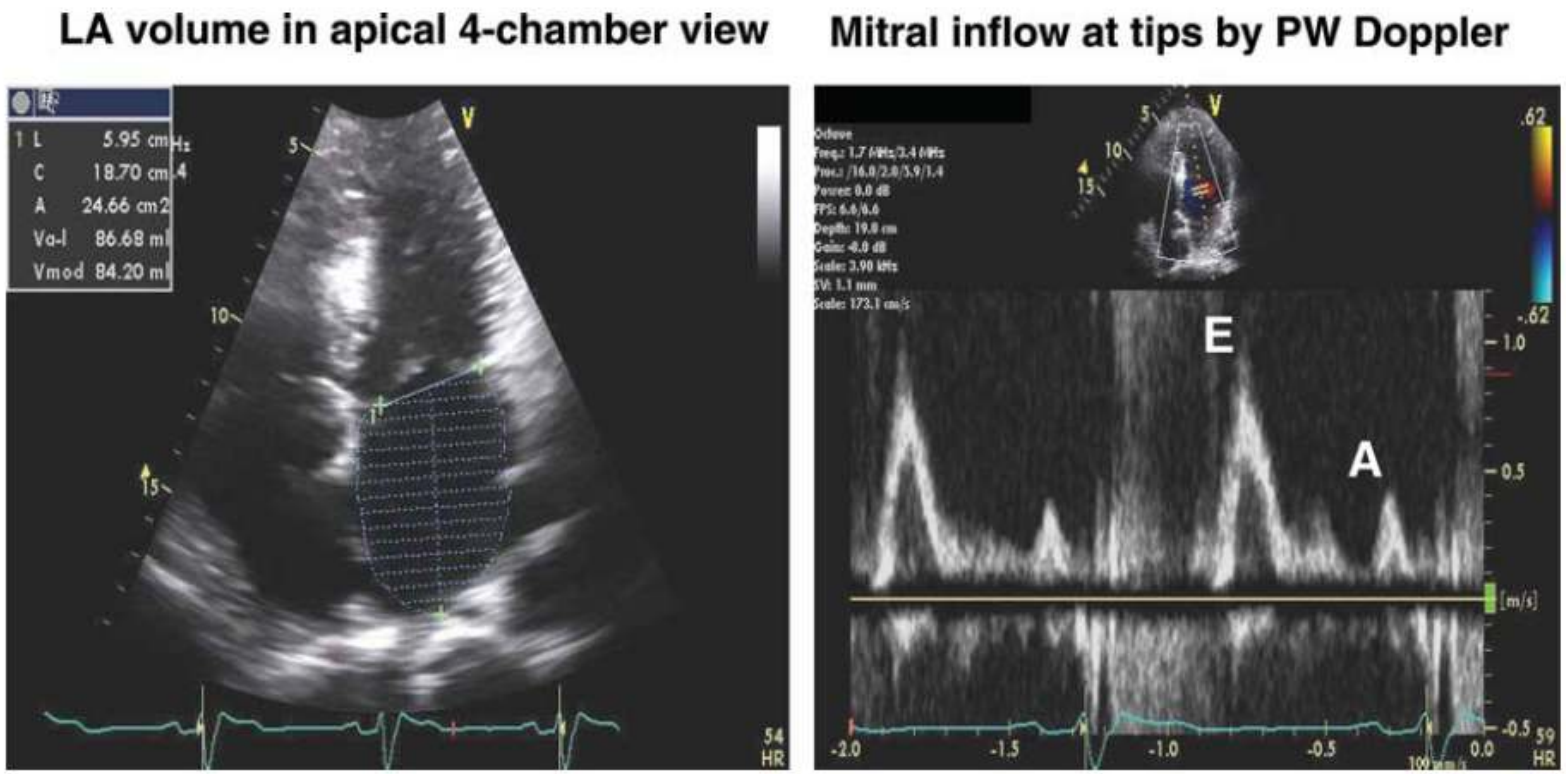


Figure 2 *(Left)* End-systolic (maximum) LA volume from an elite athlete with a volume index of 33 mL/m^2. *(Right)* Normal mitral inflow pattern acquired by PW Doppler from the same subject. Mitral E velocity was 100 cm/s, and A velocity was 38 cm/s. This athlete had trivial MR, which was captured by PW Doppler. Notice the presence of a larger LA volume despite normal function.

LA function

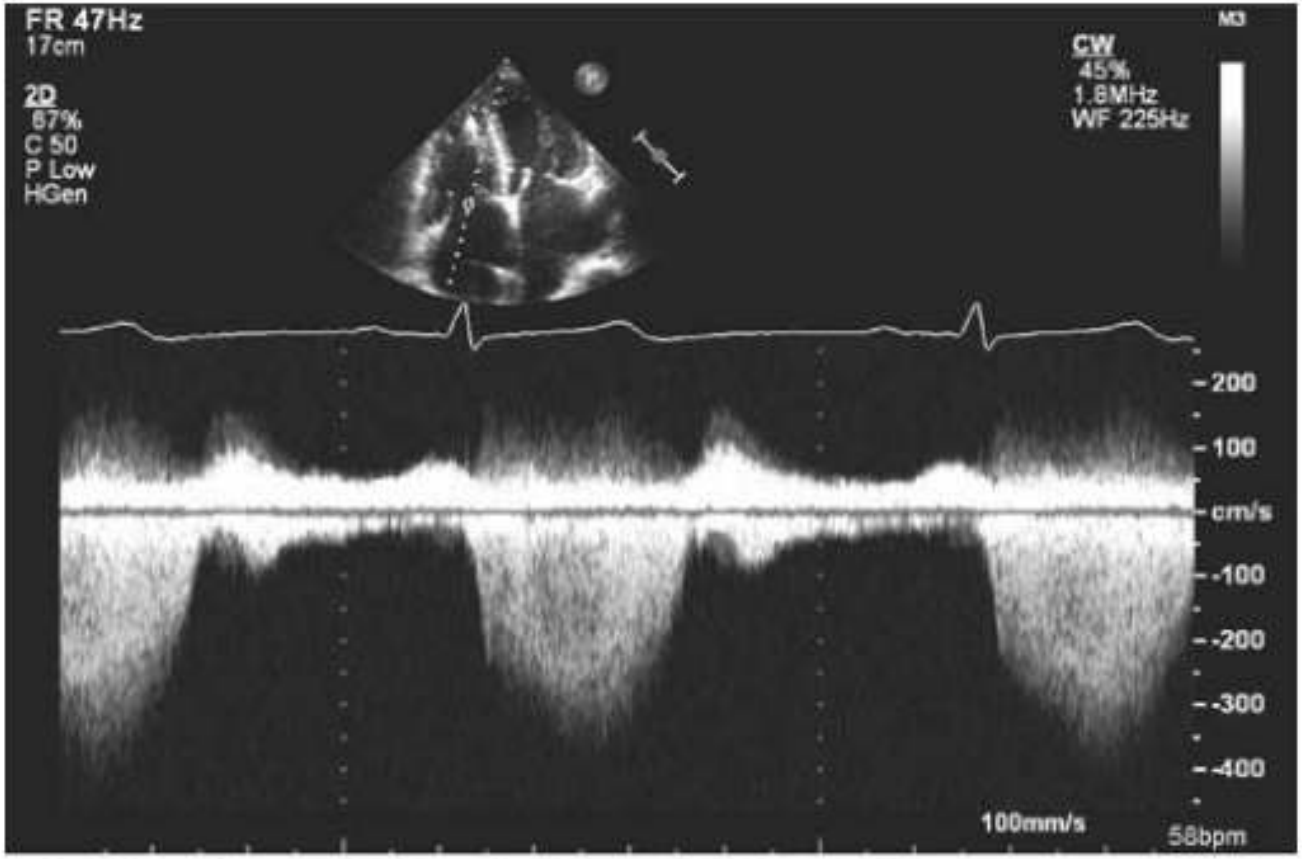
- The atrium modulates ventricular filling through its reservoir, conduit, and pump functions.
- During ventricular systole and isovolumic relaxation, when the AV valves are closed, atrial chamber work as distensible reservoirs accommodating blood flow from the venous circulation
  - Reservoir volume = LA passing empty volume – the amount of blood flow reversal in the pulmonary veins with atrial contraction
- The atrium is a pumping chamber, contributing to maintaining adequate LV EDV by actively emptying at end-diastole
  - LA stroke volume = LA volume at the onset of P wave- LA min. volume.
- The atrium behaves as a conduit that starts with AV valve opening and terminates before atrial contraction
  - The conduit volume = the LV stroke volume – (LA passive and active emptying volumes)

LA function

- Impaired LV relaxation is associated with
  - A lower diastolic AV gradient
  - A reduction in LA conduit volume
- The reservoir-pump complex is enhanced to maintain optimal LV end-diastolic volume and normal stroke volume.
- With a more advanced degree of diastolic dysfunction and reduced LA contractility, the LA contribution to LV filling decreases.

Pulmonary artery systolic and diastolic pressures

- Symptomatic patients with diastolic dysfunction usually have increased PA pressures
- In the absence of pulmonary disease, increased PA pressures may be used to infer the presence of elevated LV filling pressures.
- The peak velocity of the TR jet by CW Doppler together with systolic RA pressure are used to derive PA systolic pressure.
  - In patients with severe TR and low systolic RV-RA pressure gradients, the accuracy of the PA systolic pressure calculation is dependent on the reliable estimation of systolic RA pressure.



4 $(V)^2$ of peak TR velocity = PAS – RAP
4 $(3.6)^2$ or 52 = PAS - 20
PAS = 52 + 20 = 72 mmHg

Figure 3 Calculation of PA systolic pressure using the TR jet. In this patient, the peak velocity was 3.6 m/s, and RA pressure was estimated at 20 mm Hg.

Pulmonary artery systolic and diastolic pressures

- The end-diastolic velocity of the PR jet can be applied to derive PA diastolic pressure.
- The estimation of RA pressure is needed for both calculations and can be derived using IVC diameter and its change with respiration, as well as the ratio of systolic to diastolic flow signals in the hepatic veins.
- PA diastolic pressure by Doppler echo usually correlates with well with invasively measured mean PCWP
  - Limitations:
    - in the lower feasibility rates of adequate PR signals (< 60%), particularly in ICU and without IV contrast agents.
    - Its accuracy depends heavily on the accurate estimation of mean PA pressure.
- The assumption of relating PA diastolic pressure to LA pressure has reasonable accuracy in patients without moderate or severe pulmonary hypertension.

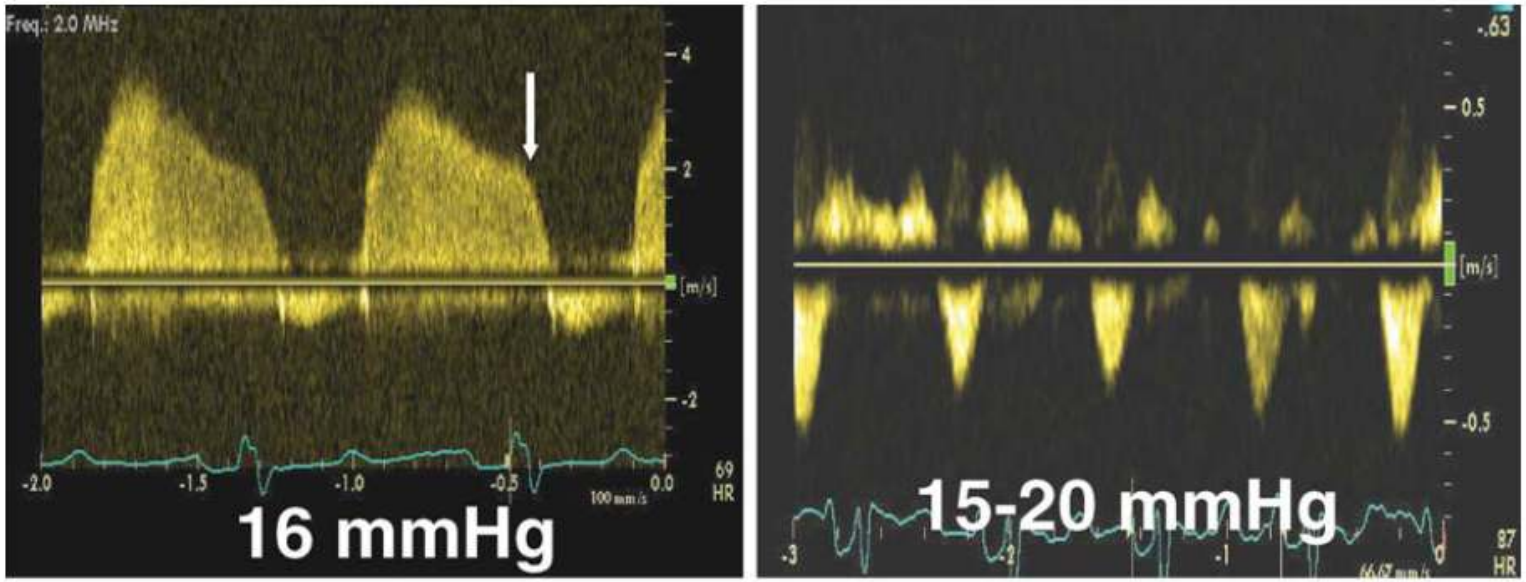


Figure 4 Calculation of PA diastolic pressure using the PR jet *(left)* and hepatic venous by PW Doppler *(right)*. In this patient, the PR end-diastolic velocity was 2 m/s *(arrow)*, and RA pressure was estimated at 15 to 20 mm Hg (see Quiñones et al[19] for details on estimating mean RA pressure).

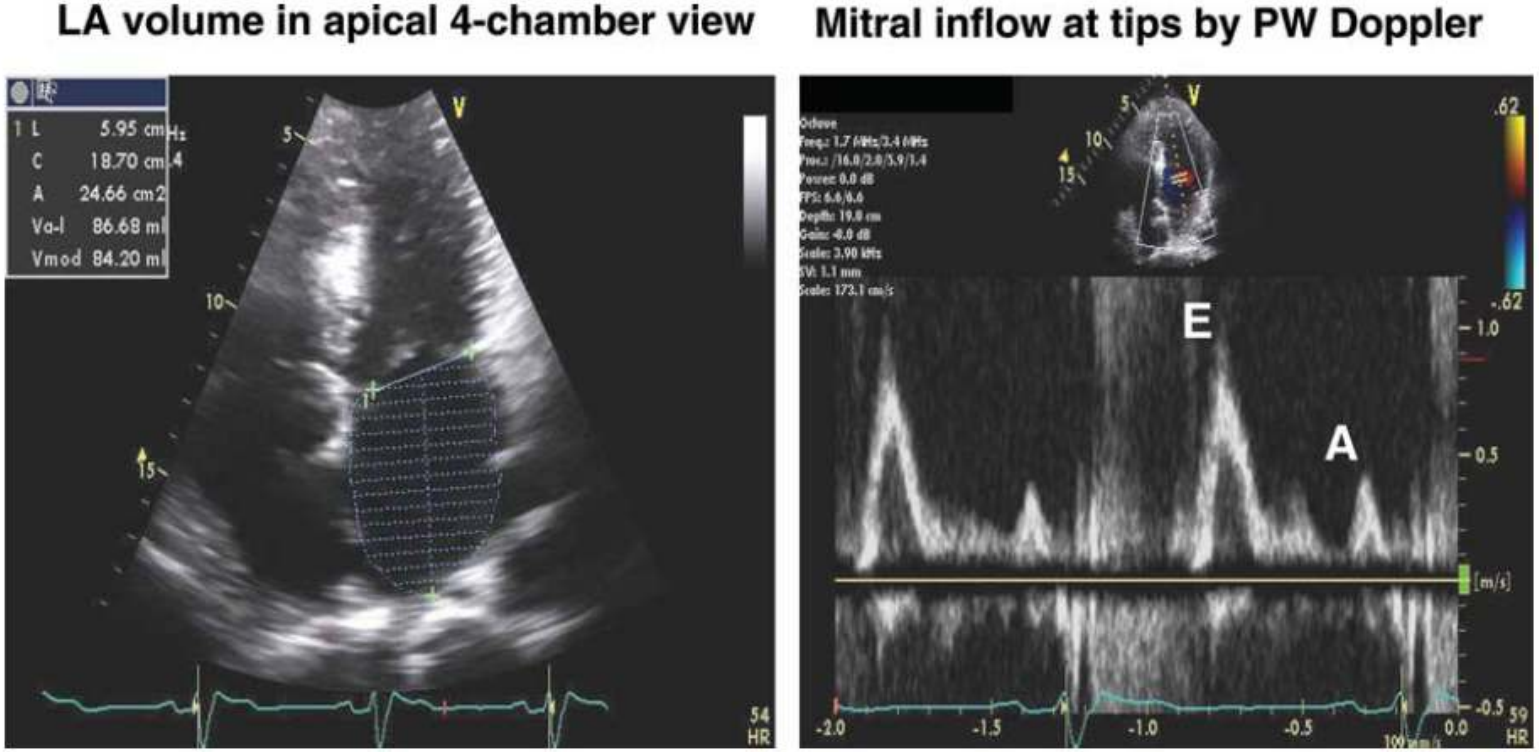
Estimation of LV diastolic function

- Mitral inflow
- Valsalva maneuver
- Pulmonary venous return
- Color M-mode flow propagation velocity
- Tissue Doppler annular early and diastolic diastolic velocities
- *Deformation measurements*
- *LV untwist*
- *Estimation of LV relaxation*
- *Estimation of LV stiffness*
- *Diastolic stress test*
- *others*

Mitral inflow

- Acquisition and feasibility

  - PW Doppler in the apical 4-chamber view to obtain mitral inflow velocities to assess LV filling
  - Performing CW Doppler to assess peak E and A velocities should be preformed before applying the PW technique to ensure that maximal velocities are obtained.
  - A 1-mm to 3-mm sample volume is then placed between the mitral leaflet tips during diastole to record a crisp velocity profile.
  - Spectral mitral velocity recordings should be initially obtained at sweep speeds of 25 to 50 mm/s for the evaluation of respiratory variation of flow velocities, as seen in the patients with pulmonary or pericardial disease
  - If variation is not present, the sweep speed is increased to *100mm/s at end-expiration,* and averaged over 3 consecutive cardiac cycles.



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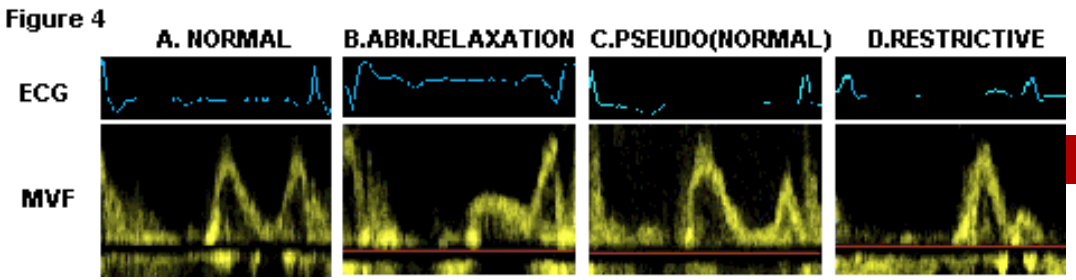
Mitral inflow

- Measurements
  - Primary measurements
    - The peak filing (E-wave) and late diastolic filling(A-wave) velocities
    - The E/A ratio
    - Deceleration time (DT) of the early filling velocity
    - The IVRT*
  - Secondary measurements
    - Mitral A duration obtained at the level of the mitral annulus
    - Diastolic filling time
    - The A-wave velocity-time integral
    - The total mitral inflow velocity-time integral with the sample volume at the level of the mitral annulus.

Mitral inflow



- Normal values
  - Age is the primary consideration when defining normal values of mitral inflow velocities and time intervals.
    - Age ↑, mitral E velocity ↓, E/A ratio ↓, DT ↑, mitral A velocity ↑
  - Variables affecting mitral inflow (other than LV diastolic function and filling pressures)
    - Heart rates and rhythm
    - PR interval
    - Cardiac output
    - Mitral annular size
    - LA function

Table 1 Normal values for Doppler-derived diastolic measurements

Measurement	Age group (y)			
	16–20	21–40	41–60	>60
IVRT (ms)	50 ± 9(32–68)	67 ± 8(51–83)	74 ± 7(60–88)	87 ± 7(73–101)
E/A ratio	1.88 ± 0.45(0.98–2.78)	1.53 ± 0.40(0.73–2.33)	1.28 ± 0.25(0.78–1.78)	0.96 ± 0.18(0.6–1.32)
DT (ms)	142 ± 19(104–180)	166 ± 14(138–194)	181 ± 19(143–219)	200 ± 29(142–258)
A duration (ms)	113 ± 17(79–147)	127 ± 13(101–153)	133 ± 13(107–159)	138 ± 19(100–176)
PV S/D ratio	0.82 ± 0.18(0.46–1.18)	0.98 ± 0.32(0.34–1.62)	1.21 ± 0.2(0.81–1.61)	1.39 ± 0.47(0.45–2.33)
PV Ar (cm/s)	16 ± 10(1–36)	21 ± 8(5–37)	23 ± 3(17–29)	25 ± 9(11–39)
PV Ar duration (ms)	66 ± 39(1–144)	96 ± 33(30–162)	112 ± 15(82–142)	113 ± 30(53–173)
Septal é (cm/s)	14.9 ± 2.4(10.1–19.7)	15.5 ± 2.7(10.1–20.9)	12.2 ± 2.3(7.6–16.8)	10.4 ± 2.1(6.2–14.6)
Septal é/á ratio	2.4*	1.6 ± 0.5(0.6–2.6)	1.1 ± 0.3(0.5–1.7)	0.85 ± 0.2(0.45–1.25)
Lateral é (cm/s)	20.6 ± 3.8(13–28.2)	19.8 ± 2.9(14–25.6)	16.1 ± 2.3(11.5–20.7)	12.9 ± 3.5(5.9–19.9)
Lateral é/á ratio	3.1*	1.9 ± 0.6(0.7–3.1)	1.5 ± 0.5(0.5–2.5)	0.9 ± 0.4(0.1–1.7)

Mitral inflow

- Inflow patterns and hemodynamics
  - Mitral inflow patterns (mitral E/A ratio and DT)
    - Normal. Impaired LV relaxation, pseudonormal LV filling (PNF) and restrictive LV filing
  - The most abnormal diastolic physiology and LV filling pattern
    - Elderly patients with severe and long-standing hypertension or
    - Patients with hypertrophic cardiomyopathy
  - Mitral E-wave velocity
    - Reflecting the LA-LV pressure gradient during early diastole
    - Affected by preload and alternations in LV relaxation
  - Mitral A-wave velocity
    - Reflecting the LA-LV pressure gradient during late diastole
    - Affected by LV compliance and LA contractile function
  - E-wave DT is influenced by
    - LV relaxation
    - LV diastolic pressures following mitral valve opening
    - LV compliance

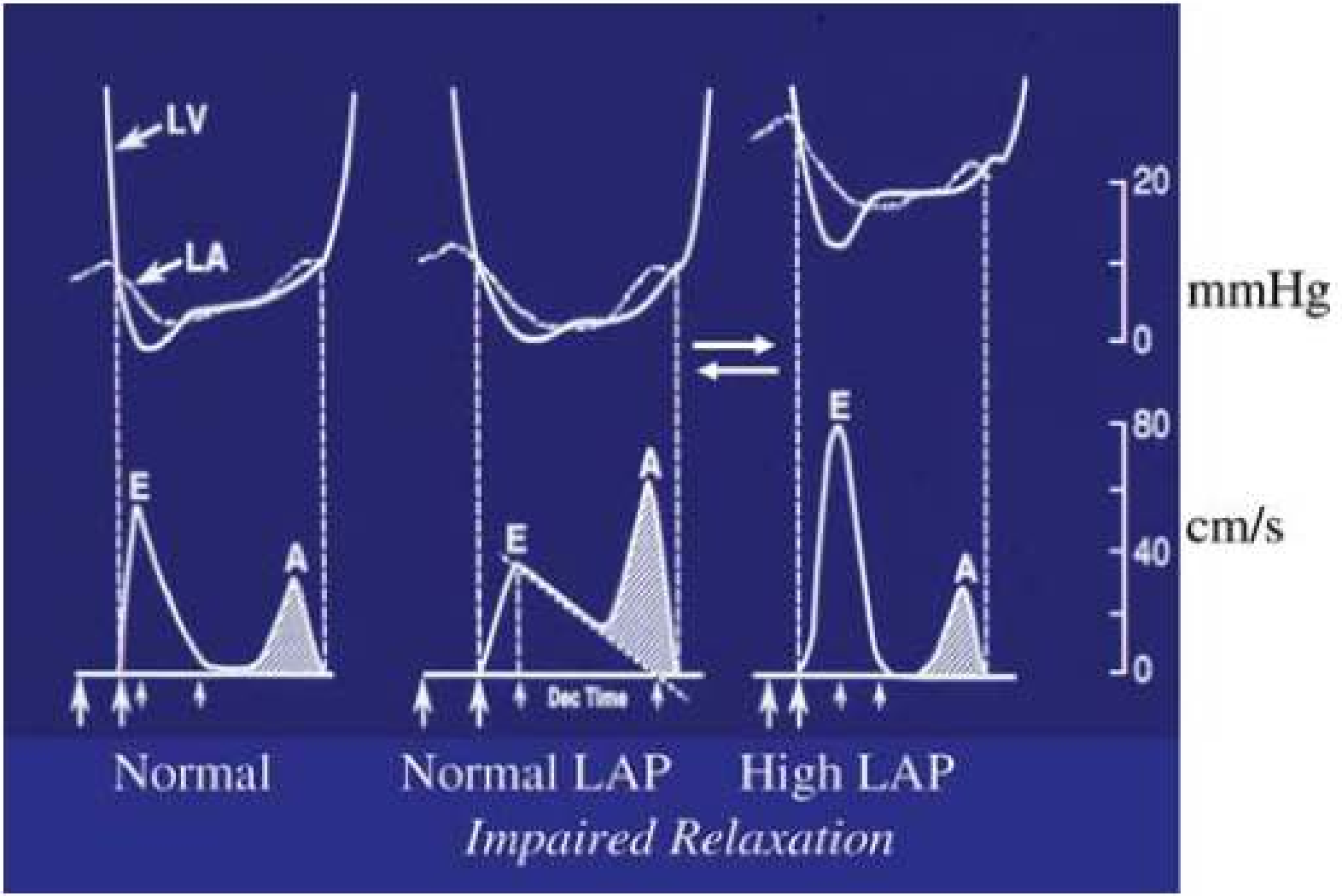


Figure 5 Schematic diagram of the changes in mitral inflow in response to the transmitral pressure gradient.

Mitral inflow

- Clinical applications
  - Patients with impaired LV relaxation filling are the least symptomatic, while a short IVRT, short mitral DT, and increased E/A velocity ratio characterize advanced diastolic dysfunction, increased LA pressure, and worse functional class.
  - A restrictive filling pattern is associated with a poor prognosis, esp. if it persists after preload reduction.
  - A pseudonormal or restrictive filling pattern associated with AMI indicates an increased risk for heart failure, unfavorable LV remodeling, and increased cardiovascular mortality, irrespective of EF.

Mitral inflow

- In patients with CAD or HCM, in whom LVEFs are $\geqq$ 50%, mitral variables correlate poorly with hemodynamics. **

- A restrictive filling pattern and LA enlargement in a patient with a normal EF are associated with a poor prognosis similar to that of a restrictive pattern in DCM.
  - Most commonly seen in restrictive cardiomyopathies, esp. amyloidosis
  - In heart transplant recipients.

Mitral inflow

- Limitations
  - Sinus tachycardia and first-degree AV block can result in partial or complete fusion of the mitral E and A waves.
  - With atrial flutter, LV filling is heavily influenced by the rapid atrial contractions,
    - No E velocity, E/A ratio, or DT is available for measurement.

Mitral inflow

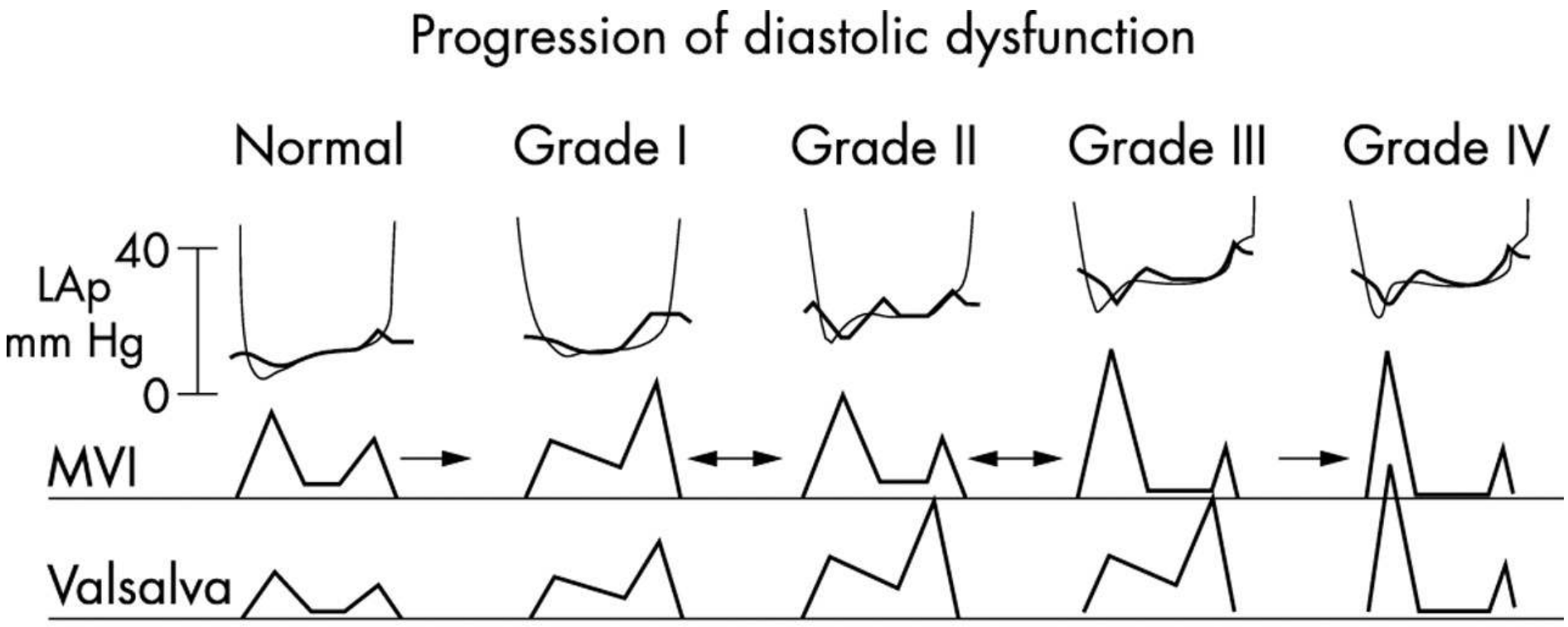
Key Points

(1) PW Doppler is performed in the apical 4-chamber view to obtain mitral inflow velocities to assess LV filling.
(2) A 1-mm to 3-mm sample volume is then placed between the mitral leaflet tips during diastole to record a crisp velocity profile.
(3) Primary measurements include peak E and A velocities, E/A ratio, DT, and IVRT.
(4) Mitral inflow patterns include normal, impaired LV relaxation, PNF, and restrictive LV filling.
(5) In patients with dilated cardiomyopathies, filling patterns correlate better with filling pressures, functional class, and prognosis than LV EF.
(6) In patients with coronary artery disease and those with hypertrophic cardiomyopathy in whom the LV EFs are $\geq$50%, mitral velocities correlate poorly with hemodynamics.

Valsalva maneuver

- Performance and acquisition
  - Performed by forceful expiration (about 40 mmHg) against a closed nose and mouth.
  - LV preload is reduced during the strain phase (phase II), and changes in mitral inflow are observed to distinguish normal from PNF patterns
  - A decrease of 20 cm/s in mitral peak E velocity is usually considered an adequate effort in patients without restrictive filling.

Valsalva maneuver



Valsalva maneuver

- Clinical applications
  - A pseudonormal mitral inflow pattern is caused by a mild to moderate increase in LA pressure in the setting of delayed myocardial relaxation.
  - Because the Valsalva maneuver decreases preload during the strain phase, pseudonormal mitral inflow changes to a pattern of impaired relaxation. ^
    - Mitral E velocity decreases with a prolongation of DT, whereas the A velocity is unchanged or increases, such that the E/A ratio decreases.
    - With normal mitral inflow velocity pattern, both E and A velocities decrease proportionately, with an unchanged E/A ratio.
  - In cardiac patients, a decrease of $\geqq$50% in the E/A ration is highly specific for increased LV filling pressures. ^^

Valsalva maneuver

- Limitations
  - Not everyone is able to perform this maneuver adequately
  - Not standardized.
  - Diminishing the value of distinguishing normal from pseudonormal mitral inflow since the introduction of tissue Doppler recordings of the mitral annulus to assess the status of LV relaxation and estimation filling pressures more quantitatively and easily .

Valsalva maneuver

Key Points

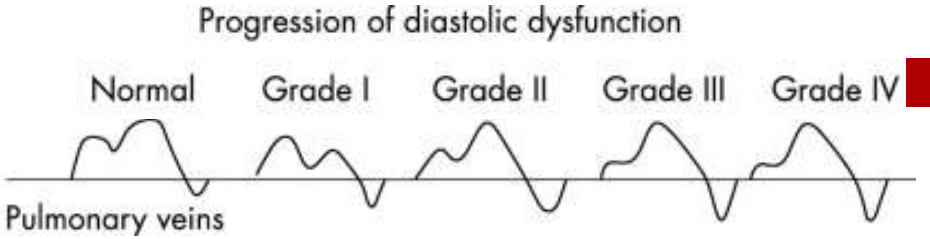
(1) The Valsalva maneuver is performed by forceful expiration (about 40 mm Hg) against a closed nose and mouth, producing a complex hemodynamic process involving 4 phases.

(2) In cardiac patients, a decrease of $\geq$50% in the E/A ratio is highly specific for increased LV filling pressures,[57] but a smaller magnitude of change does not always indicate normal diastolic function.

Pulmonary venous flow

- Acquisition and feasibility
  - Performed in the apical 4-chamber view*
  - A 2-3 mm sample volume is placed >0.5cm into the pulmonary vein for optimal recording of the spectral waveforms
  - The major technical problem is LA wall motion artifacts, caused by atrial contraction, interfering with accurate display of atrial reversal (Ar) velocity.
  - Spectral recordings at a sweep speed of 50-100mm/s at end-expiration
  - Measurements include the average of $\geqq$ 3 consecutive cardiac cycle.

Pulmonary venous flow

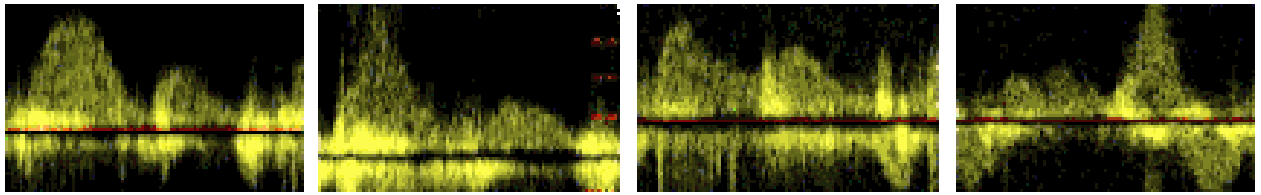


- Measurements include
  - Peak systolic (S) velocity
  - Peak anterograde diastolic (D) velocity
  - The S/D ratio
  - Systolic filling fraction
    - $S_{\text{time-velocity integral}}/(S_{\text{time-velocity integral}} + D_{\text{time-velocity integral}})$
  - The peak Ar velocity in late diastole
  - The duration of the Ar velocity
  - The time difference between The duration of the Ar velocity and mitral A-wave duration (Ar-A)
  - D velocity DT
- There are 2 systolic velocities (S1 and S2), most noticeable when there is a prolonged PR interval.
  - S1: related to atrial relaxation
  - S2; used to compute the ratio of peal systolic to peak diastolic velocity

Pulmonary venous flow

- Hemodynamic determinants
  - S1 velocity is primarily influenced by changes in LA pressure and LV contraction and relaxation.
  - S2 is related to stroke volume and pulse-wave propagation in the PA tree.
  - D velocity is influenced by changes in LV filling and compliance and changes in parallel with mitral E velocity.
  - Pulmonary venous Ar velocity and duration are influenced by LV late diastolic pressure, atrial preload, and LA contractility.
  - A decrease in LA compliance and an increase in LA pressure
    - ↓ S velocity, ↑ D velocity → S/D ratio < 1*
  - Atrial fibrillation results in a blunted S wave and the absence of Ar

PVF



With increased LVEDP, Ar velocity and duration increase, as well as the time difference between Ar duration and mitral A-wave duration

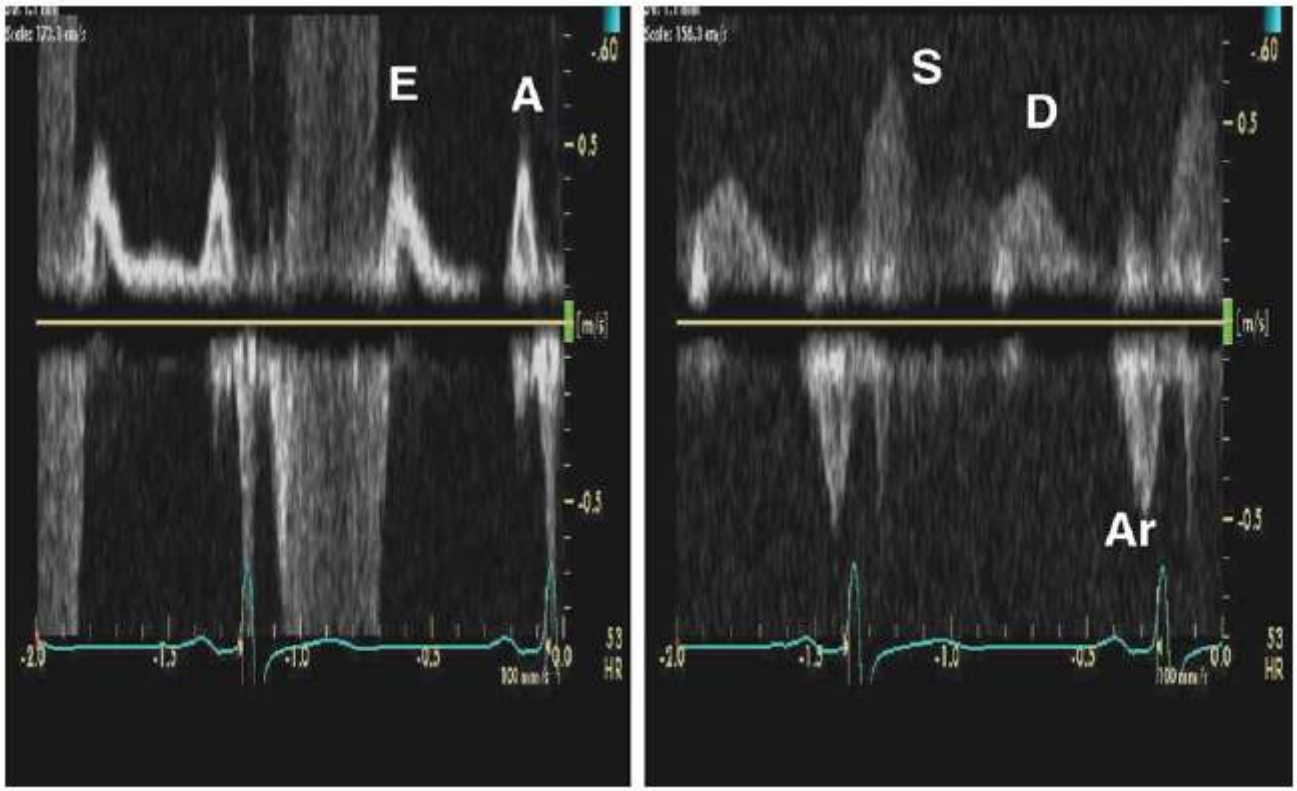


Figure 6 Recording of mitral inflow at the level of the annulus *(left)* and pulmonary venous flow *(right)* from a patient with increased LVEDP. Notice the markedly increased pulmonary venous Ar velocity at 50 cm/s and its prolonged duration at >200 ms in comparison with mitral A (late diastolic) velocity. Mitral A duration is best recorded at the level of the annulus.[22]

Pulmonary venous flow

- Normal values
  - Pulmonary venous inflow velocity are influenced by age.
  - Normal young subjects aged < 40 years usually have prominent D velocities, reflecting their mitral E waves
  - With increasing age, the S/D ratio increases.
  - In normal subjects, Ar velocities can increase with age but usually do not exceed 35 cm/s.
    - High values suggest increased LVEDP

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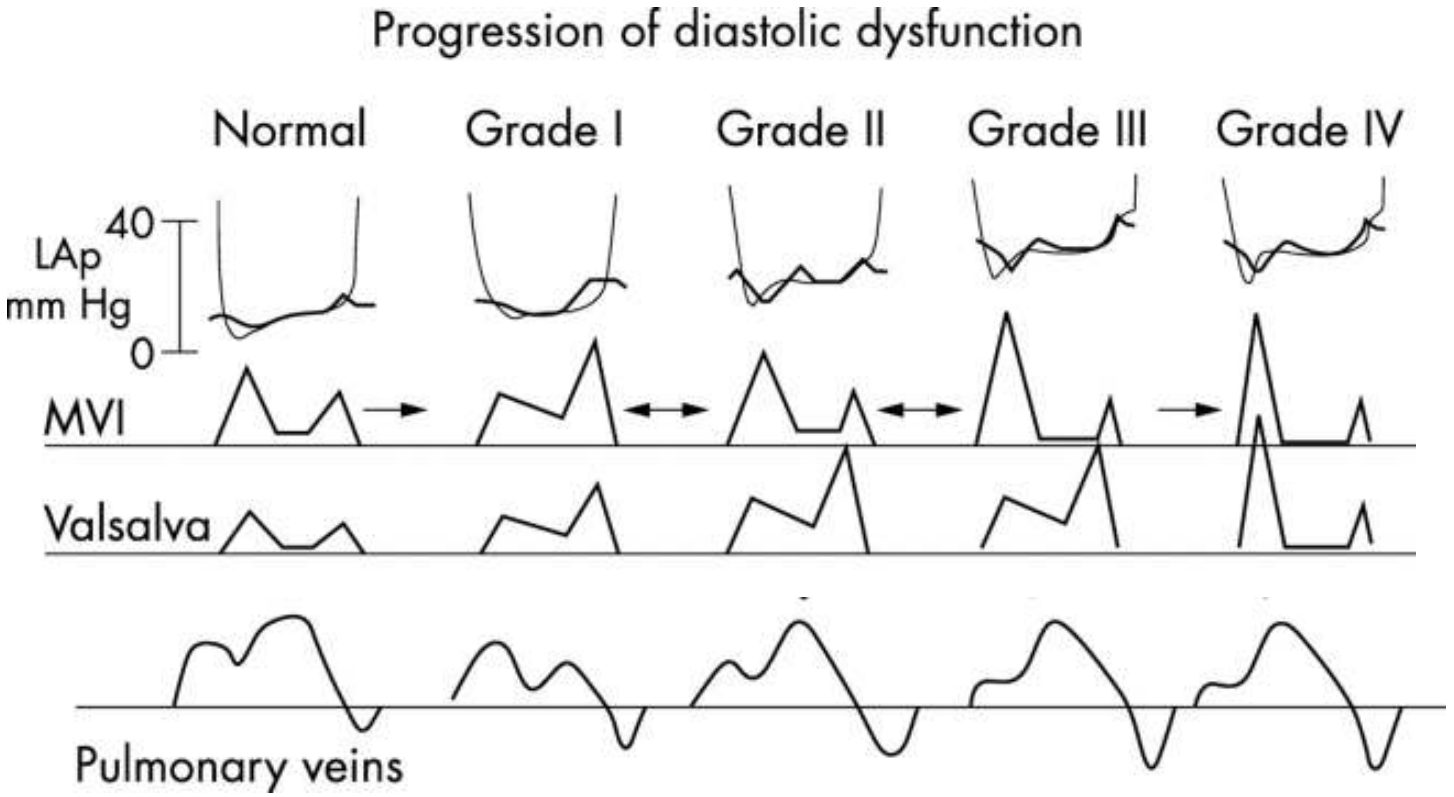

Progression of diastolic dysfunction
Normal
Grade I
Grade II
Grade III
Grade IV
LAp
mm Hg
40
0
MVI
Valsalva
Pulmonary veins

Pulmonary venous flow

- Clinical applications
  - In patients with depressed EFs, a reduced systolic fraction of anterograde flow (<40%) is related to decreased LA compliance and increased mean LA pressure.
    - Limited accuracy in patients with EF > 50%, atrial fibrillation, mitral valve disease and hypertrophic cardiomyopathy.
  - The isolated increase in LVEDP is the first hemodynamic abnormality seen with diastolic dysfunction.

Pulmonary venous flow

- Limitations
  - Difficulty in obtaining high-quality recording suitable for measurement
    - Esp. for Ar velocity*
  - Sinus tachycardia and first degree AV block often result in the start of atrial contraction occurring before diastolic mitral and pulmonary venous flow velocity has declined to the zero baseline.
    - →Increasing the width of the mitral A-wave velocity
    - →Decreasing the width of the reversal of in the pulmonary vein
    - →Making the Ar-A relationship difficult to interpret for assessing LV A-wave pressure increase
  - With atrial fibrillation, the loss of atrial contraction and relaxation reduces pulmonary venous flow systolic flow regardless of filling pressures.
  - With a first-degree AV block of $\geqq$ 300ms, flow into the left atrium with its relaxation(S1) cannot be separated from later systolic flow(S2) or can even occur in diastole

Pulmonary venous flow

Key Points

(1) PW Doppler of pulmonary venous flow is performed in the apical 4-chamber view and aids in the assessment of LV diastolic function.
(2) A 2-mm to 3-mm sample volume is placed >0.5 cm into the pulmonary vein for optimal recording of the spectral waveforms.
(3) Measurements include peak S and D velocities, the S/D ratio, systolic filling fraction, and peak Ar velocity in late diastole. Another measurement is the time difference between Ar duration and mitral A-wave duration (Ar − A).
(4) With increased LVEDP, Ar velocity and duration increase, as well as the Ar − A duration.
(5) In patients with depressed EFs, reduced systolic filling fractions (<40%) are related to decreased LA compliance and increased mean LA pressure.

Color M-mode flow propagation velocity

- Acquisition, feasibility and measurement
  - The most widely used approach for measuring mitral-to-apical flow propagation is *the slope method*
    - The least variability
    - Performed in the apical 4-chamber view.
  - The M-mode scan line is placed through the center of the LV inflow blood column from the mitral valve to the apex. *
  - Flow propagation velocity (Vp) is measured as the slope of the first aliasing velocity during early filling, measured from the mitral valve plane to 4cm distally into the LV cavity.
    - Vp > 50 cm/s is considered normal.

Color M-mode flow propagation velocity

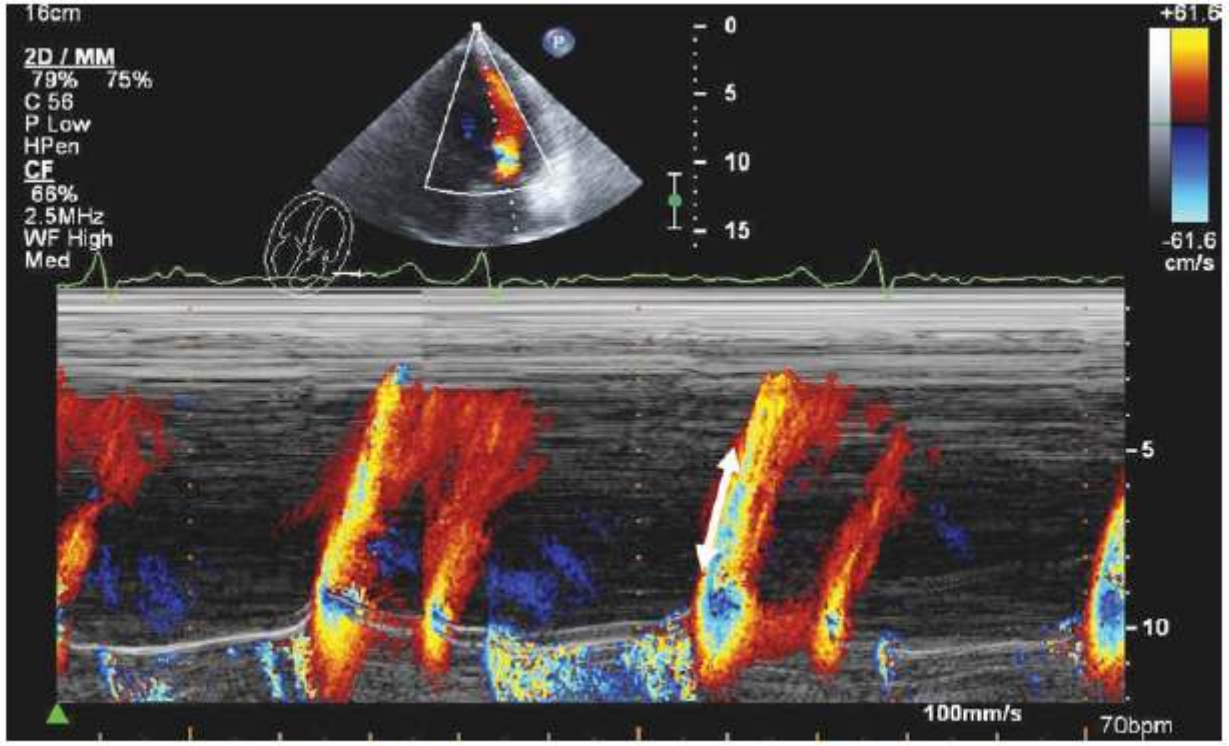


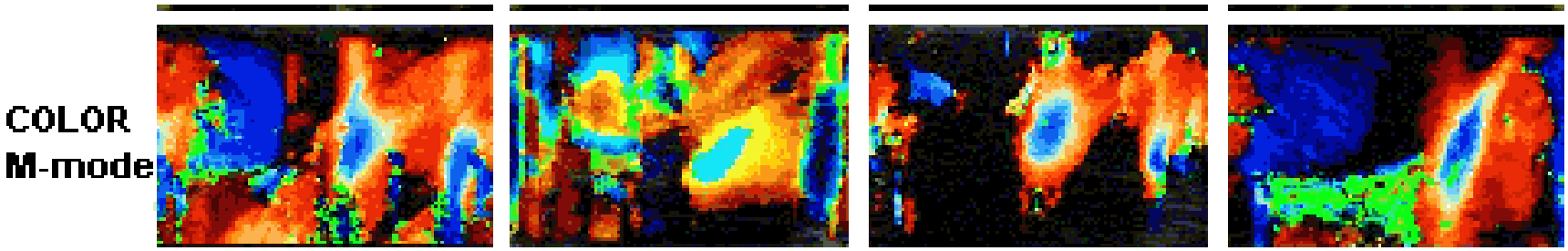
Figure 7 Color M-mode Vp from a patient with depressed EF and impaired LV relaxation. The slope *(arrow)* was 39 cm/s.

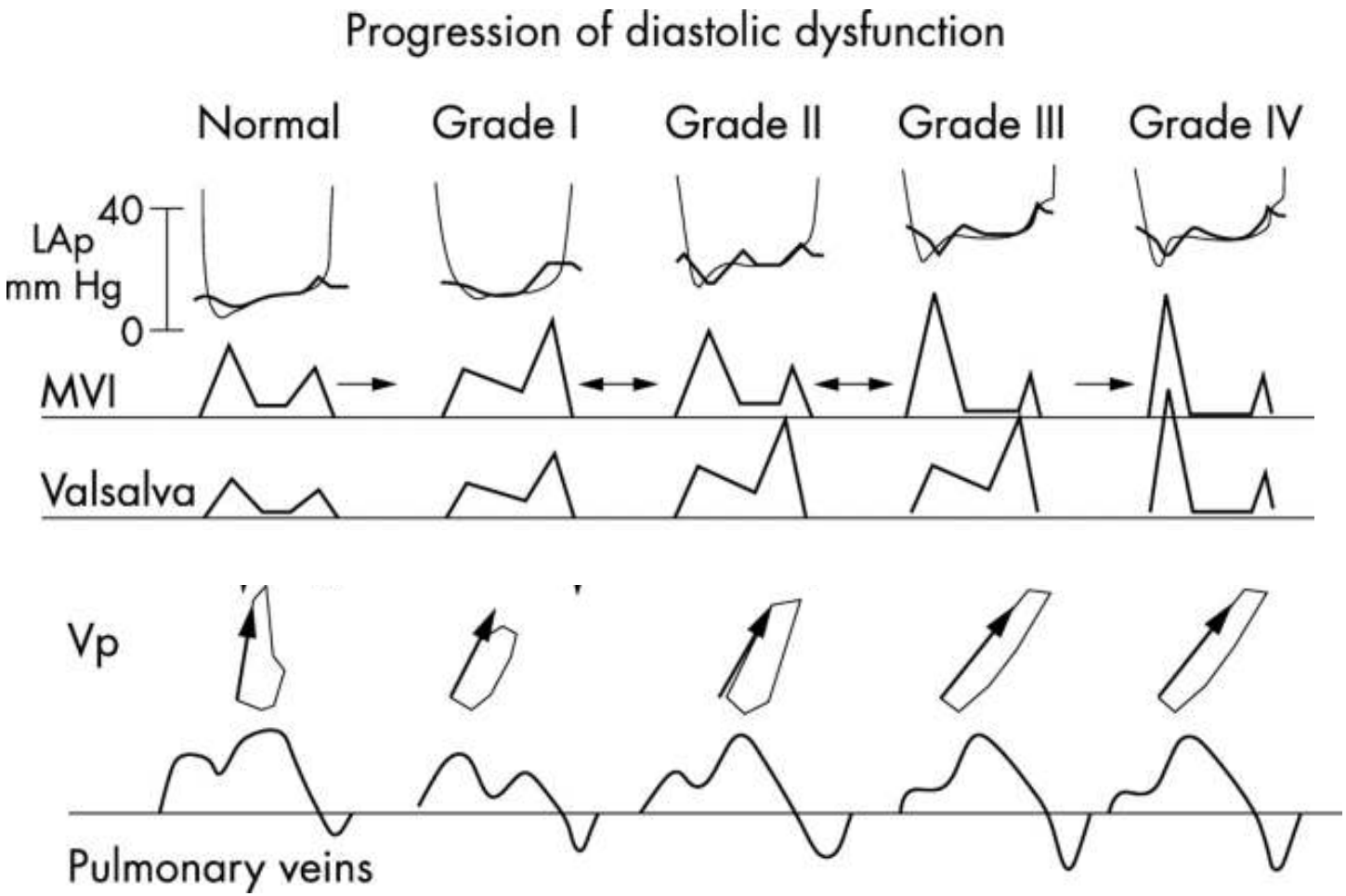
Color M-mode flow propagation velocity

- Hemodynamic determinants
  - Dominated by an early wave and an atrial-induced filling wave.
  - In the normal ventricle, the early wave propagates rapidly toward the apex and is driven by a pressure gradient between the LV base and the apex.
    - The gradient represents a suction force and has been attributed to LV restoring forces and LV relaxation.
  - During heart failure and during myocardial ischemia, there is slowing of mitral-to-apical suction.

Color M-mode flow propagation velocity

- Clinical applications
  - A semiquantitative marker of LV diastolic dysfunction.*
  - E/Vp to predict LV filling pressures




Progression of diastolic dysfunction
Normal
Grade I
Grade II
Grade III
Grade IV
LAp
mm Hg
40
0
MVI
Valsalva
Vp
Pulmonary veins

Color M-mode flow propagation velocity

Key Points

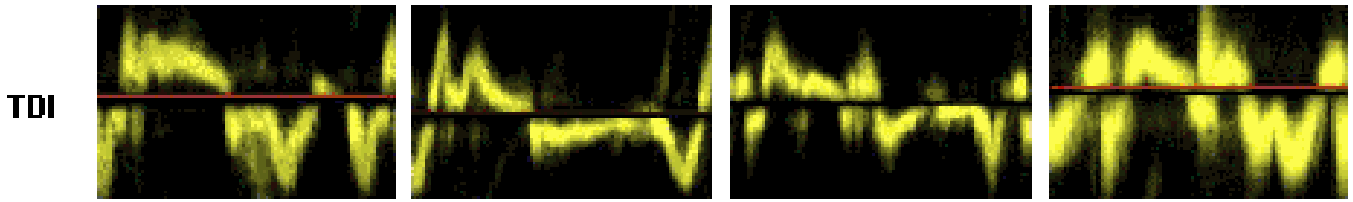
(1) Acquisition is performed in the apical 4-chamber view, using color flow imaging.
(2) The M-mode scan line is placed through the center of the LV inflow blood column from the mitral valve to the apex, with baseline shift to lower the Nyquist limit so that the central highest velocity jet is blue.
(3) Vp is measured as the slope of the first aliasing velocity during early filling, measured from the mitral valve plane to 4 cm distally into the LV cavity, or the slope of the transition from no color to color.
(4) Vp $>$ 50 cm/s is considered normal.
(5) In most patients with depressed EFs, Vp is reduced, and should other Doppler indices appear inconclusive, an E/Vp ratio $\geq$ 2.5 predicts PCWP $>$ 15 mm Hg with reasonable accuracy.
(6) Patients with normal LV volumes and EFs but elevated filling pressures can have misleadingly normal Vp.

Tissue Doppler annular early and late diastolic velocities

- Acquisitions and feasibility
  - PW tissue Doppler imaging (DTI) is performed in the apical views to acquire mitral annular velocities.
  - The sample volume should be positioned at or 1cm within the sepal and lateral insertion sites of the mitral leaflets.
  - Minimal angulation(<20 ◦) should be present.
  - Spectral recordings at a sweep speed of 50-100 mm/s at end-expiration
  - Average of ≧3 consecutive cardiac cycles.

Tissue Doppler annular early and late diastolic velocities

- Primary measurements
  - The systolic velocity: S
  - Early diastolic velocity: Ea,Em, E'or ***e'***
  - Late diastolic velocity: Aa, Am. A'or ***a'***
  - e'/a' ratio
  - E/e' ratio: estimation of LV filling pressures



Tissue Doppler annular early and late diastolic velocities

- Hemodynamic determinants of e' velocity
  - LV relaxation, preload, systolic function and LV minimal pressure
  - For preload, LV filling pressures have a minimal effect on e' in the presence of impaired LV relaxation.
  - In patients with cardiac disease, e'velocity can be used to correct for the effect of LV relaxation on mitral E velocity
  - The E/e' ratio can be applied for the prediction of LV filling pressrues

Tissue Doppler annular early and late diastolic velocities

- Hemodynamic determinants of a' velocity
  - LA systolic function, and LVEDP
  - Increased LA contractility leads to increased a' velocity
  - Increased LVEDP leads to a decrease in a'

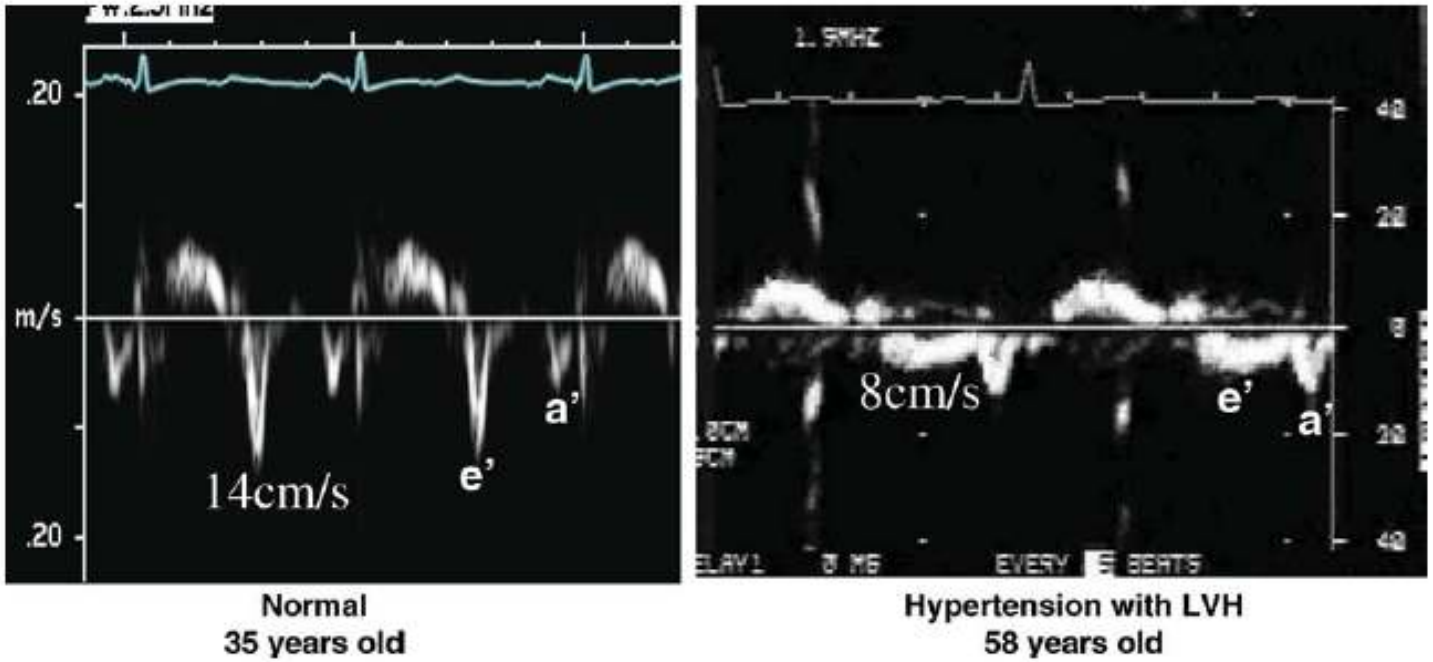


Figure 8 Tissue Doppler (TD) recording from the lateral mitral annulus from a normal subject aged 35 years *(left)* (é = 14 cm/s) and a 58-year-old patient with hypertension, LV hypertrophy, and impaired LV relaxation *(right)* (é = 8 cm/s).

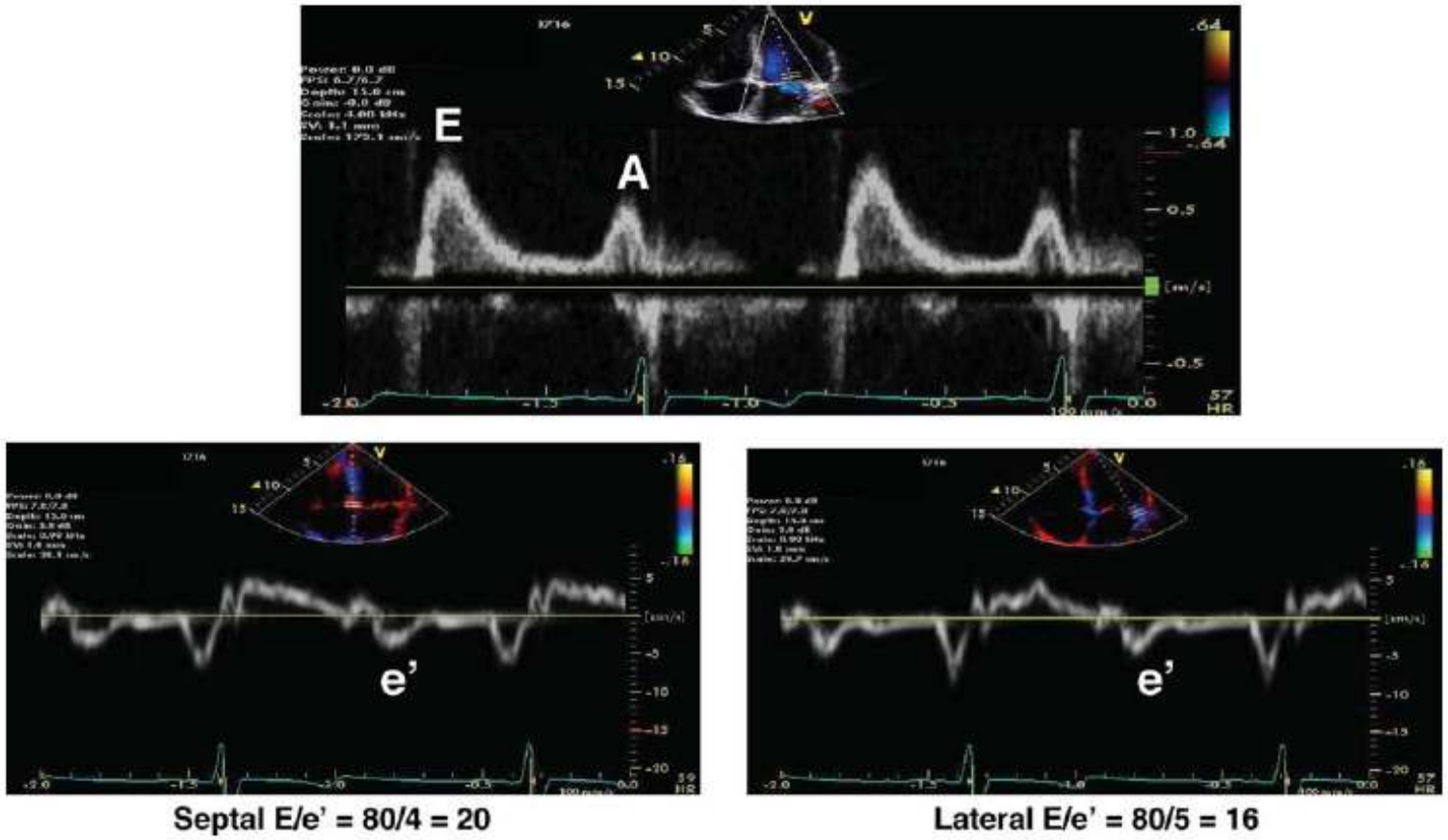
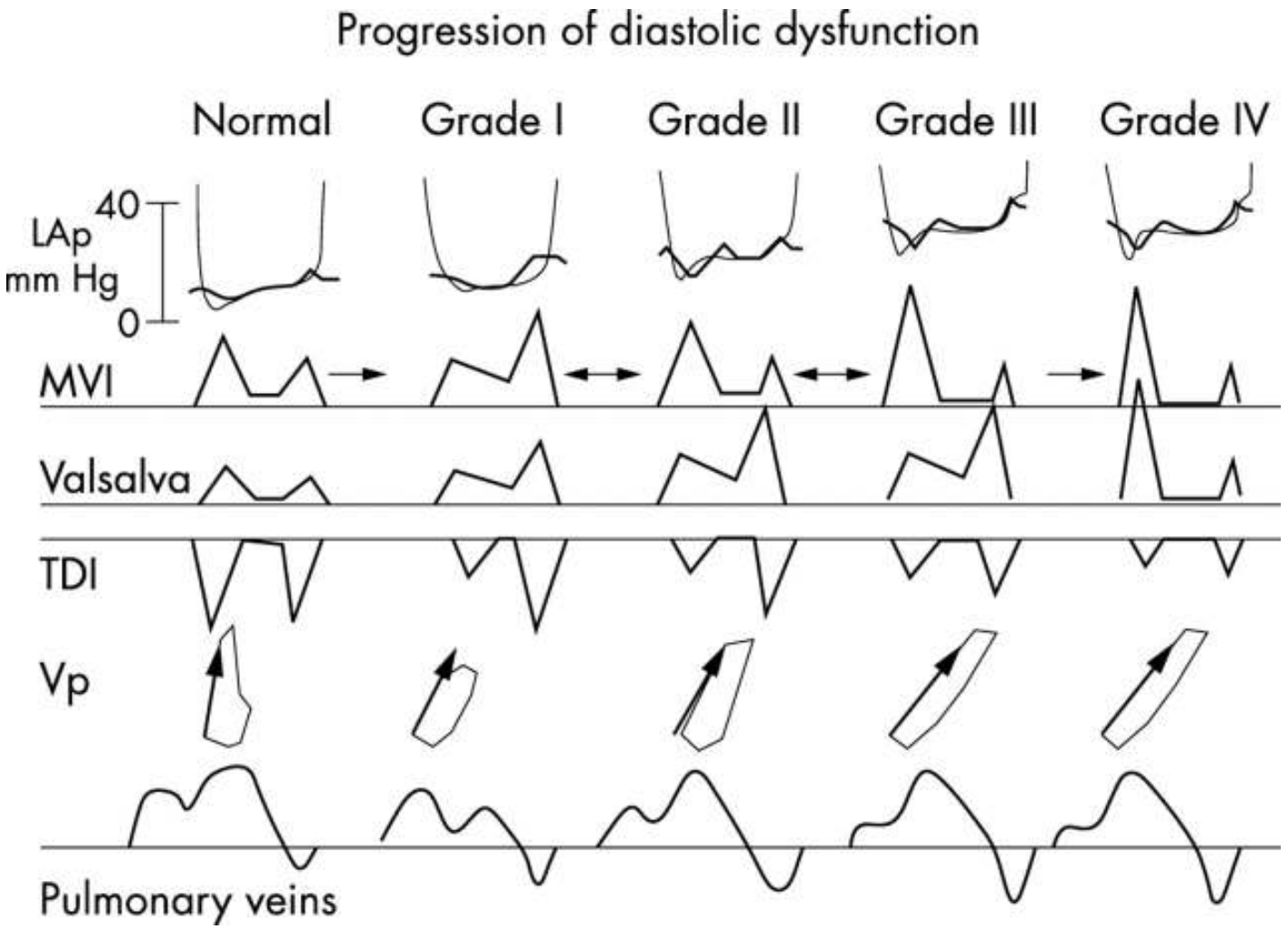


Figure 9 Mitral inflow *(top)*, septal *(bottom left)*, and lateral *(bottom right)* tissue Doppler signals from a 60-year-old patient with heart failure and normal EF. The E/é ratio was markedly increased, using é from either side of the annulus.

Tissue Doppler annular early and late diastolic velocities

Table 1 Normal values for Doppler-derived diastolic measurements

Measurement	Age group (y)			
	16–20	21–40	41–60	>60
IVRT (ms)	50 ± 9(32–68)	67 ± 8(51–83)	74 ± 7(60–88)	87 ± 7(73–101)
E/A ratio	1.88 ± 0.45(0.98–2.78)	1.53 ± 0.40(0.73–2.33)	1.28 ± 0.25(0.78–1.78)	0.96 ± 0.18(0.6–1.32)
DT (ms)	142 ± 19(104–180)	166 ± 14(138–194)	181 ± 19(143–219)	200 ± 29(142–258)
A duration (ms)	113 ± 17(79–147)	127 ± 13(101–153)	133 ± 13(107–159)	138 ± 19(100–176)
PV S/D ratio	0.82 ± 0.18(0.46–1.18)	0.98 ± 0.32(0.34–1.62)	1.21 ± 0.2(0.81–1.61)	1.39 ± 0.47(0.45–2.33)
PV Ar (cm/s)	16 ± 10(1–36)	21 ± 8(5–37)	23 ± 3(17–29)	25 ± 9(11–39)
PV Ar duration (ms)	66 ± 39(1–144)	96 ± 33(30–162)	112 ± 15(82–142)	113 ± 30(53–173)
Septal é (cm/s)	14.9 ± 2.4(10.1–19.7)	15.5 ± 2.7(10.1–20.9)	12.2 ± 2.3(7.6–16.8)	10.4 ± 2.1(6.2–14.6)
Septal é/á ratio	2.4*	1.6 ± 0.5(0.6–2.6)	1.1 ± 0.3(0.5–1.7)	0.85 ± 0.2(0.45–1.25)
Lateral é (cm/s)	20.6 ± 3.8(13–28.2)	19.8 ± 2.9(14–25.6)	16.1 ± 2.3(11.5–20.7)	12.9 ± 3.5(5.9–19.9)
Lateral é/á ratio	3.1*	1.9 ± 0.6(0.7–3.1)	1.5 ± 0.5(0.5–2.5)	0.9 ± 0.4(0.1–1.7)


Progression of diastolic dysfunction
Normal
Grade I
Grade II
Grade III
Grade IV
LAp
mm Hg
40
0
MVI
Valsalva
TDI
Vp
Pulmonary veins

Tissue Doppler annular early and late diastolic velocities

- Clinical applications
  - Mitral annulus velocities can be used to drew inferences about LV relaxation
  - E/e' ratio can be used to predict LV filling pressure
  - Preferable to use the average e' velocity obtained from the septal and lateral sides of the mitral annulus.
    - Because septal e' is usually lower than lateral e'velocity, the E/e' using septal signals is usually higher
  - Although single-site measurements are sometimes used in patients with globally normal or abnormal LV systolic function, it is imperative to use the average e' velocity in the presence of regional dysfunction.
  - The septal E/e' ratio
    - <8 → associated with normal LV filling pressures
    - >15 →associated with increased LV filling pressures
    - 8-15 → other echo indices should be used
  - In patients with normal EFs, the lateral tissue Doppler signals (E/e' and e'/a') have the best correlations with LV filling pressures and invasive indices of LV stiffness.

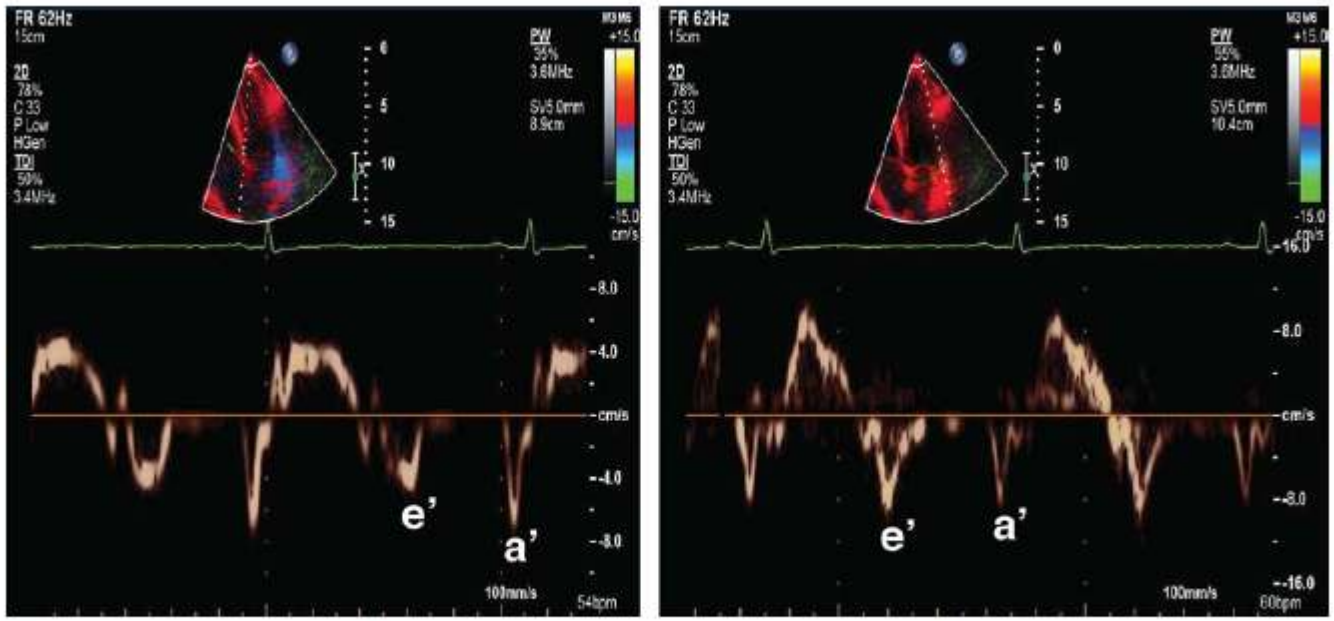


Figure 10 Septal *(left)* and lateral *(right)* tissue Doppler recordings from a patient with an anteroseptal myocardial infarction. Notice the difference between septal é (5 cm/s) and lateral é (10 cm/s). It is imperative to use the average of septal and lateral é velocities in such patients to arrive at more reliable assessments of LV relaxation and filling pressures.

Tissue Doppler annular early and late diastolic velocities

- For technical limitations
  - Proper attention to the location of the sample size, as well as gain, filter and minimal angulation with annular motion is essential for reliable velocity measurements
- With experience,
  - These are highly reproducible with low variability.

Tissue Doppler annular early and late diastolic velocities

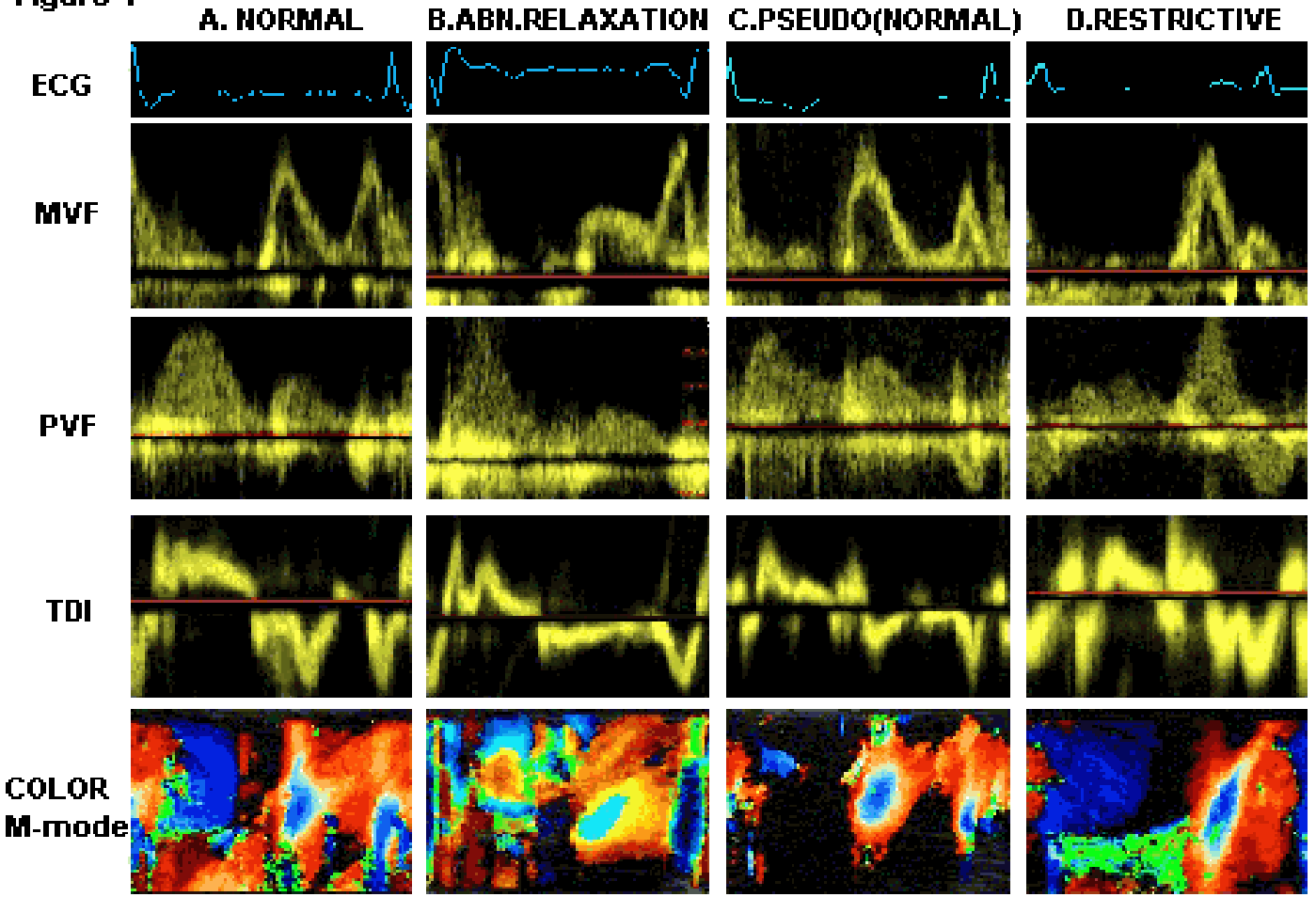
- Annular velocity measurements and the E/e'ratio should not be used in the following
  - In normal subjects, e'velocity is positively related to preload, and the e/e'ratio may not provide a reliable estimation of filling pressure.
  - e' velocity is usually reduced in patients with significant *annular calcification, surgical rings, mitral stenos is and prosthetic mitral valves*.
  - e' velocity is increased in patients with moderate to severe primary MR and normal LV relaxation due to increased flow across the regurgitant valve.

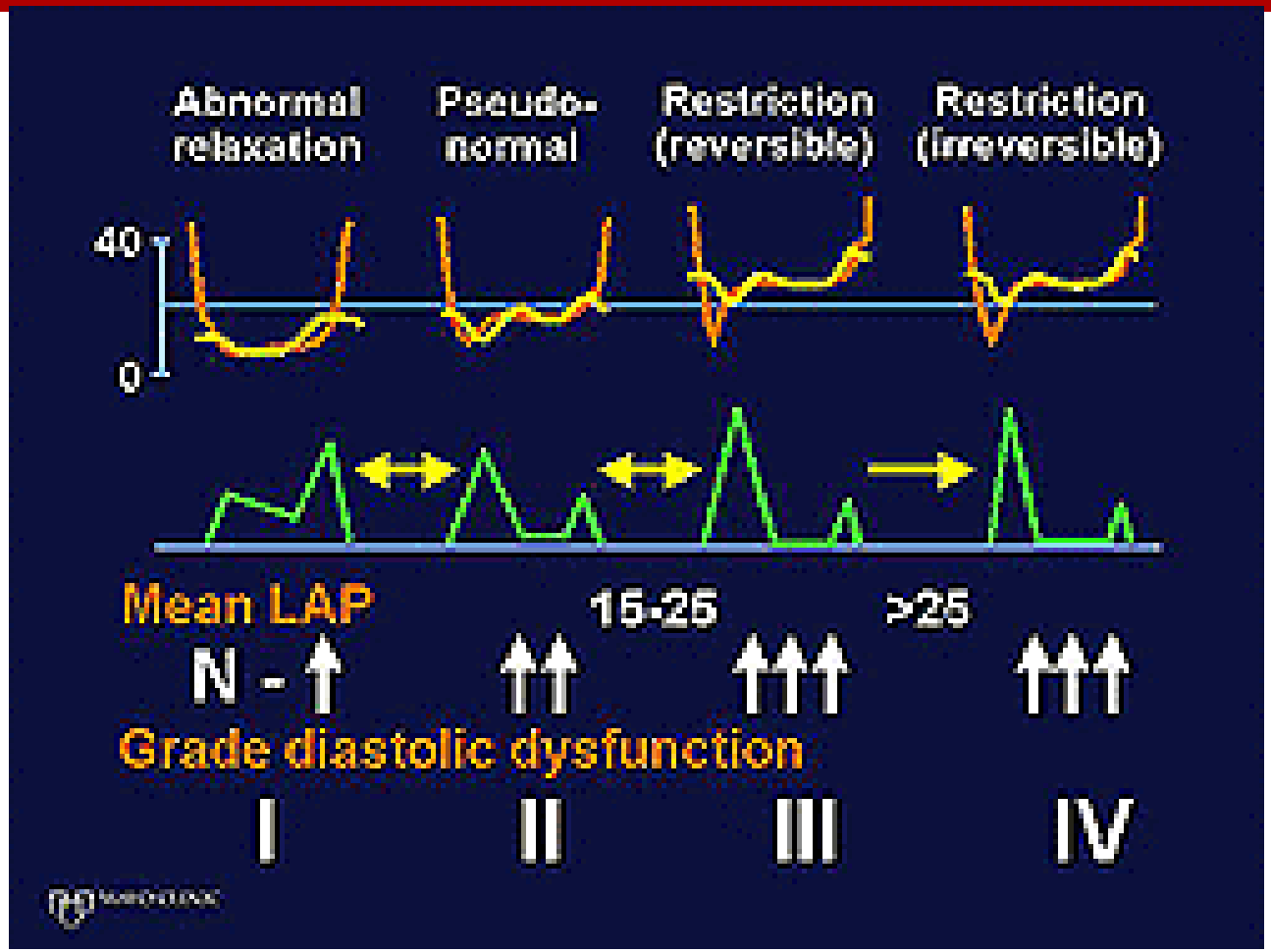
Tissue Doppler annular early and late diastolic velocities

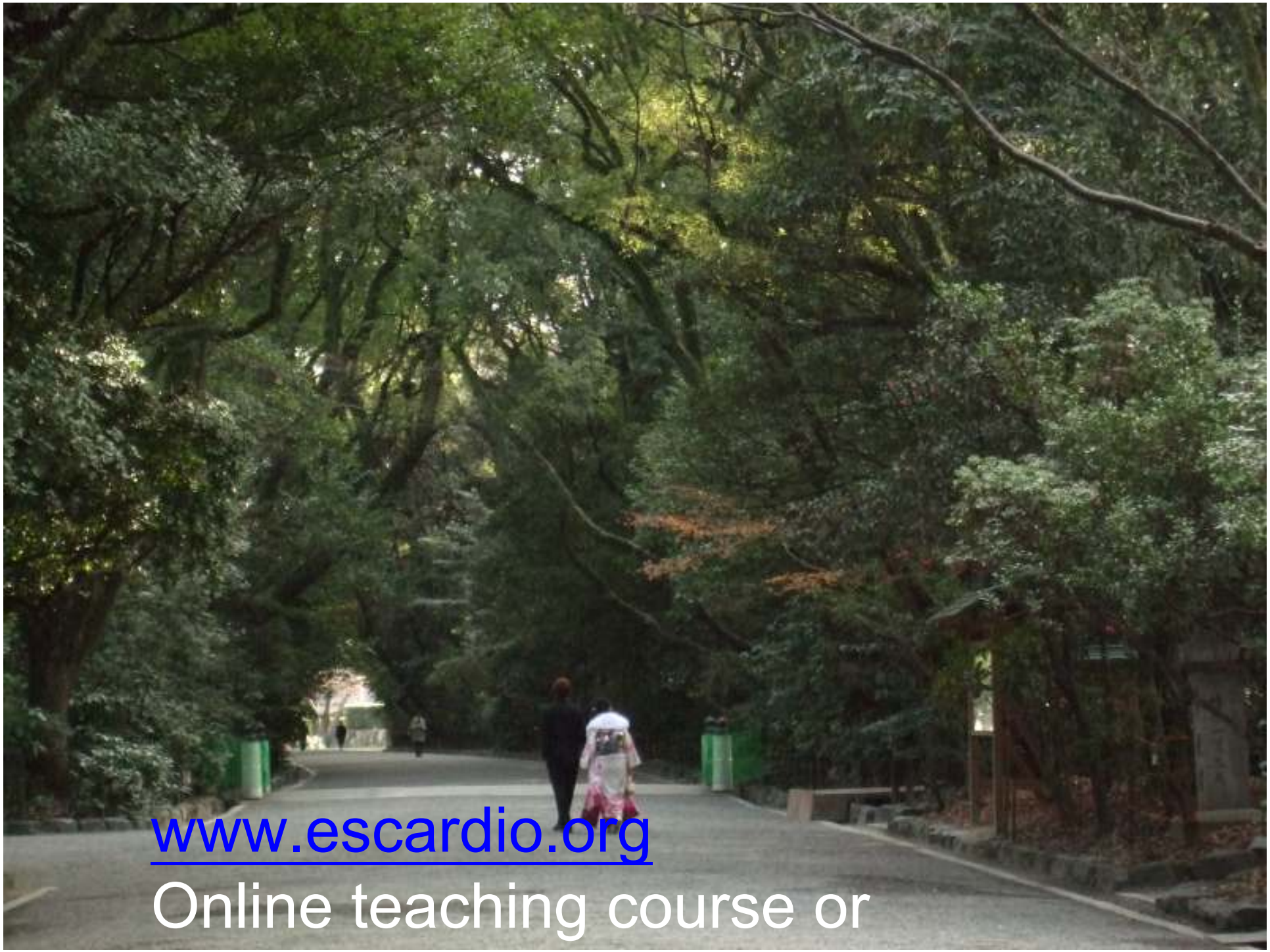
Key Points

(1) PW DTI is performed in the apical views to acquire mitral annular velocities.
(2) The sample volume should be positioned at or 1 cm within the septal and lateral insertion sites of the mitral leaflets.
(3) It is recommended that spectral recordings be obtained at a sweep speed of 50 to 100 mm/s at end-expiration and that measurements should reflect the average of ≥ 3 consecutive cardiac cycles.
(4) Primary measurements include the systolic and early (é) and late (á) diastolic velocities.
(5) For the assessment of global LV diastolic function, it is recommended to acquire and measure tissue Doppler signals at least at the septal and lateral sides of the mitral annulus and their average.
(6) In patients with cardiac disease, é can be used to correct for the effect of LV relaxation on mitral E velocity, and the E/é ratio can be applied for the prediction of LV filling pressures.
(7) The E/é ratio is not accurate as an index of filling pressures in normal subjects or in patients with heavy annular calcification, mitral valve disease, and constrictive pericarditis.

Figure 4


A. NORMAL
B.ABN.RELAXATION
C.PSEUDO(NORMAL)
D.RESTRICTIVE
ECG
MVF
PVF
TDI
COLOR
M-mode


Abnormal relaxation
Pseudo-normal
Restriction (reversible)
Restriction (irreversible)
40
0
Mean LAP
N -
15-25
>25
Grade diastolic dysfunction
I
II
III
IV


www.escardio.org
Online teaching course or

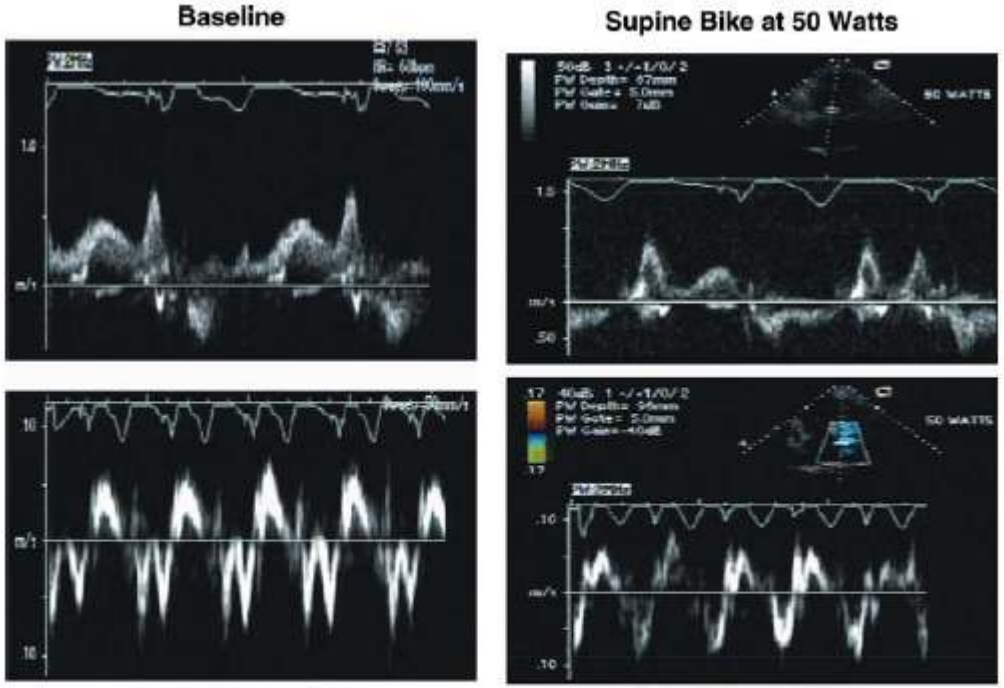


Figure 11 Exercise Doppler recordings from a patient with reduced diastolic reserve. At baseline, mitral inflow shows an impaired relaxation pattern, with an E/é ratio of 7, and the peak velocity of the TR jet was 2.4 m/s (PA systolic pressure ≥23 mm Hg). During supine bike exercise, mitral E velocity and the E/A ratio increase with shortening of DT. The E/é ratio is now 11, and the PA systolic pressure is increased to ≥58 mm Hg (TR peak velocity = 3.8 m/s).

Table 2 Changes in mitral and tissue Doppler septal velocities with exercise in normal subjects (mean age, 59 ± 14 years)[145]

Variable	Baseline	Exercise
E (cm/s)	73 ± 19	90 ± 25
A (cm/s)	69 ± 17	87 ± 22
DT (ms)	192 ± 40	176 ± 42
é (cm/s)	12 ± 4	15 ± 5
E/é	6.7 ± 2.2	6.6 ± 2.5

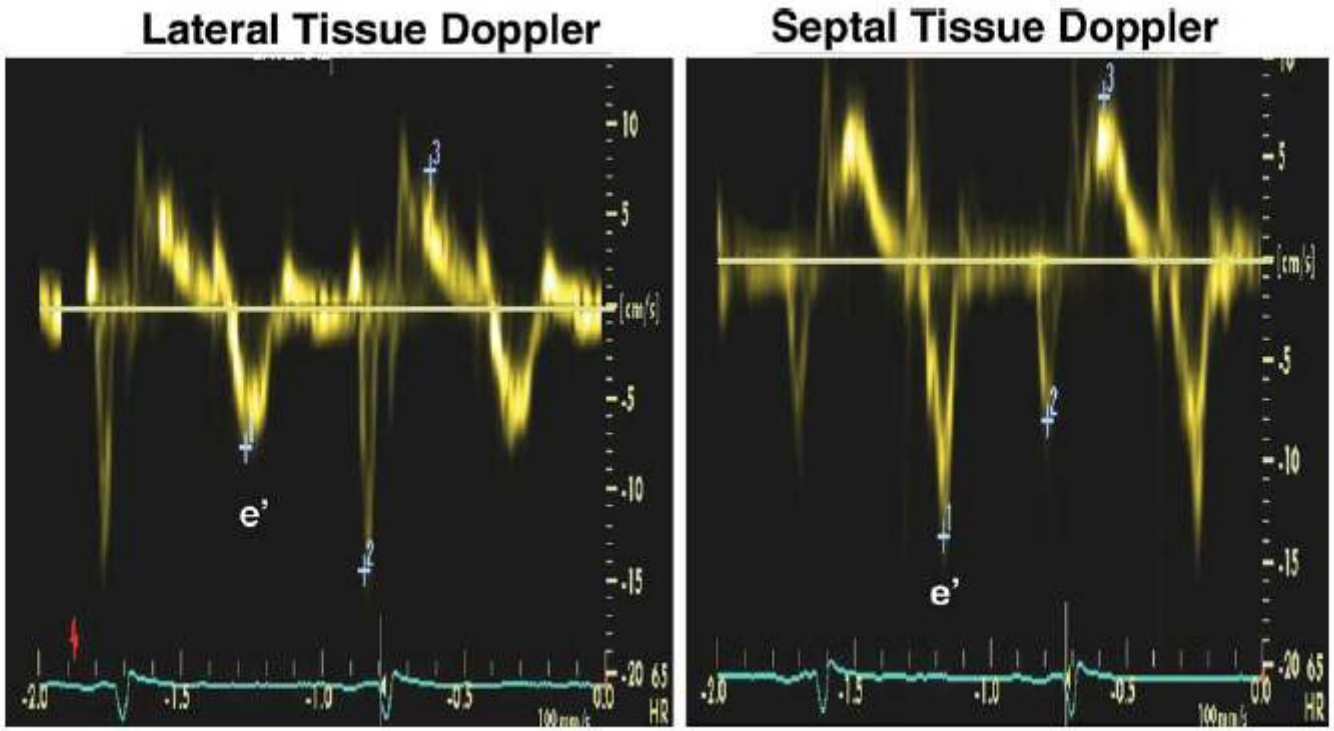


Figure 12 Lateral *(left)* and septal *(right)* TD velocities from a patient with constrictive pericarditis. Notice the higher septal é at 14 cm/s in comparison with lateral é at 8 cm/s. 1 = é, 2 = á, and 3 = systolic velocity.

Table 3 Differentiation of constrictive pericarditis from restrictive cardiomyopathy

Variable	Restriction	Constriction
Septal motion	Normal	Respiratory shift
Mitral E/A ratio	>1.5	>1.5
Mitral DT (ms)	<160	<160
Mitral inflow respiratory variation	Absent	Usually present
Hepatic vein Doppler	Inspiratory diastolic flow reversal	Expiratory diastolic flow reversal
Mitral septal annular é	Usually <7 cm/s	Usually >7 cm/s
Mitral lateral annular é	Higher than septal é	Lower than septal é
Ventricular septal strain	Reduced	Usually normal

Table 4 Assessment of LV filling pressures in special populations

Disease	Echocardiographic measurements and cutoff values
Atrial fibrillation[68,104,159]	Peak acceleration rate of mitral E velocity ($\geq$1,900 cm/s^2), IVRT ($\leq$65 ms), DT of pulmonary venous diastolic velocity ($\leq$220 ms), E/Vp ratio ($\geq$1.4), and septal E/é ratio ($\geq$11)
Sinus tachycardia[102,105]	Mitral inflow pattern with predominant early LV filling in patients with EFs < 50%, IVRT $\leq$ 70 ms is specific (79%), systolic filling fraction $\leq$ 40% is specific (88%), lateral E/é > 10 (a ratio > 12 has highest the specificity of 96%)
Hypertrophic cardiomyopathy[50]	Lateral E/é ($\geq$10), Ar − A ($\geq$30 ms), PA pressure (>35 mm Hg), and LA volume ($\geq$34 mL/m^2)
Restrictive cardiomyopathy[51,52,160]	DT (<140 ms), mitral E/A (>2.5), IVRT (<50 ms has high specificity), and septal E/é (>15)
Noncardiac pulmonary hypertension[163]	Lateral E/é can be applied to determine whether a cardiac etiology is the underlying reason for the increased PA pressures (cardiac etiology: E/é > 10; noncardiac etiology: E/é < 8)
Mitral stenosis[69]	IVRT (<60 ms has high specificity), IVRT/TE-é (<4.2), mitral A velocity (>1.5 m/s)
MR[69,70,157]	Ar − A ($\geq$30 ms), IVRT (<60 ms has high specificity), and IVRT/$T_{E\text{-}é}$ (<3) may be applied for the prediction of LV filling pressures in patients with MR and normal EFs, whereas average E/é (>15) is applicable only in the presence of a depressed EF

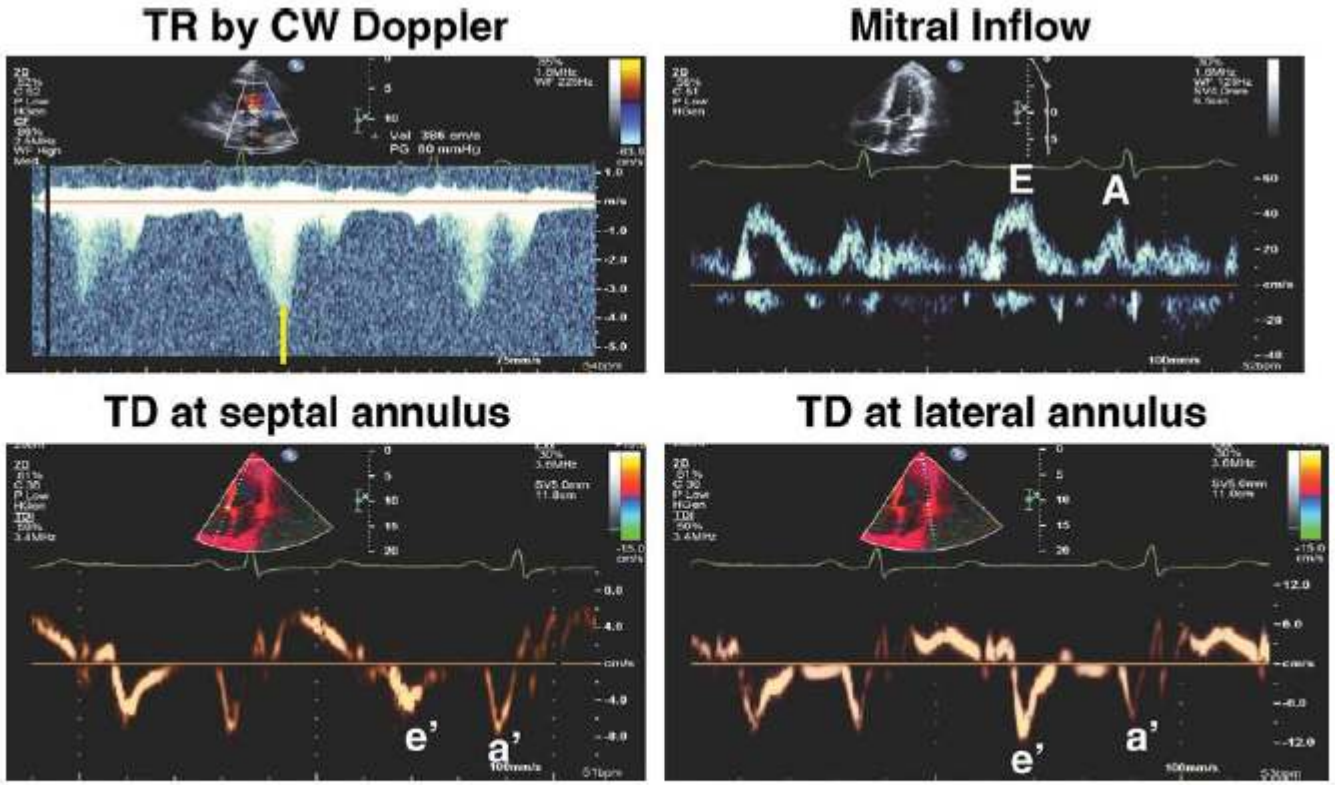


Figure 13 *(Top left)* Recording of TR jet by CW Doppler (peak velocity marked by yellow arrow) from a patient with primary pulmonary hypertension. The right ventricular–RA systolic pressure gradient is 60 mmHg. *(Top right)* Mitral inflow at the level of the leaflet tips with mitral E velocity of 50 cm/s. *(Bottom left)* Recording of septal tissue Doppler velocities with é of 5.5 cm/s. *(Bottom right)* Lateral tissue Doppler signals with a normal é velocity of 11.5 cm/s.

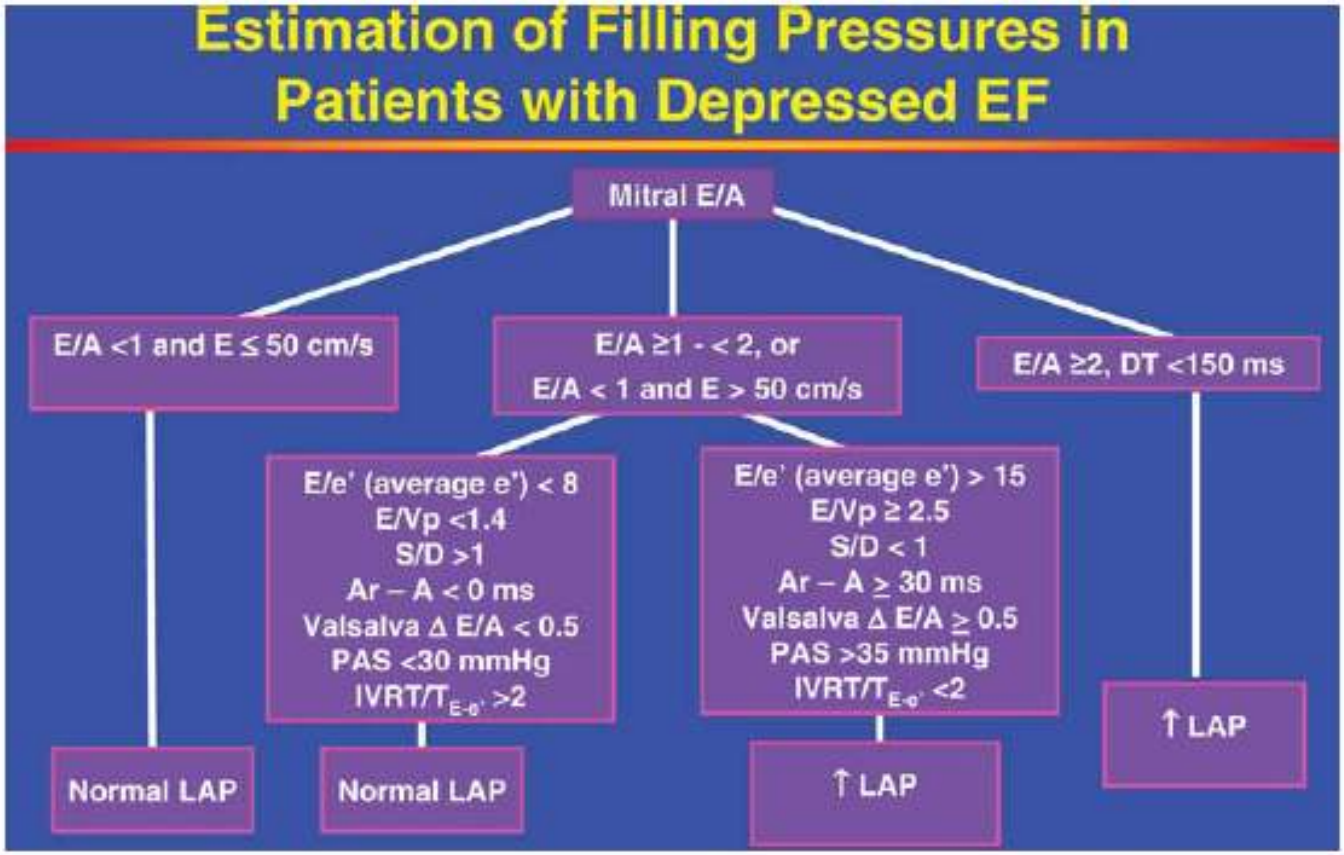


Figure 14 Diagnostic algorithm for the estimation of LV filling pressures in patients with depressed EFs.

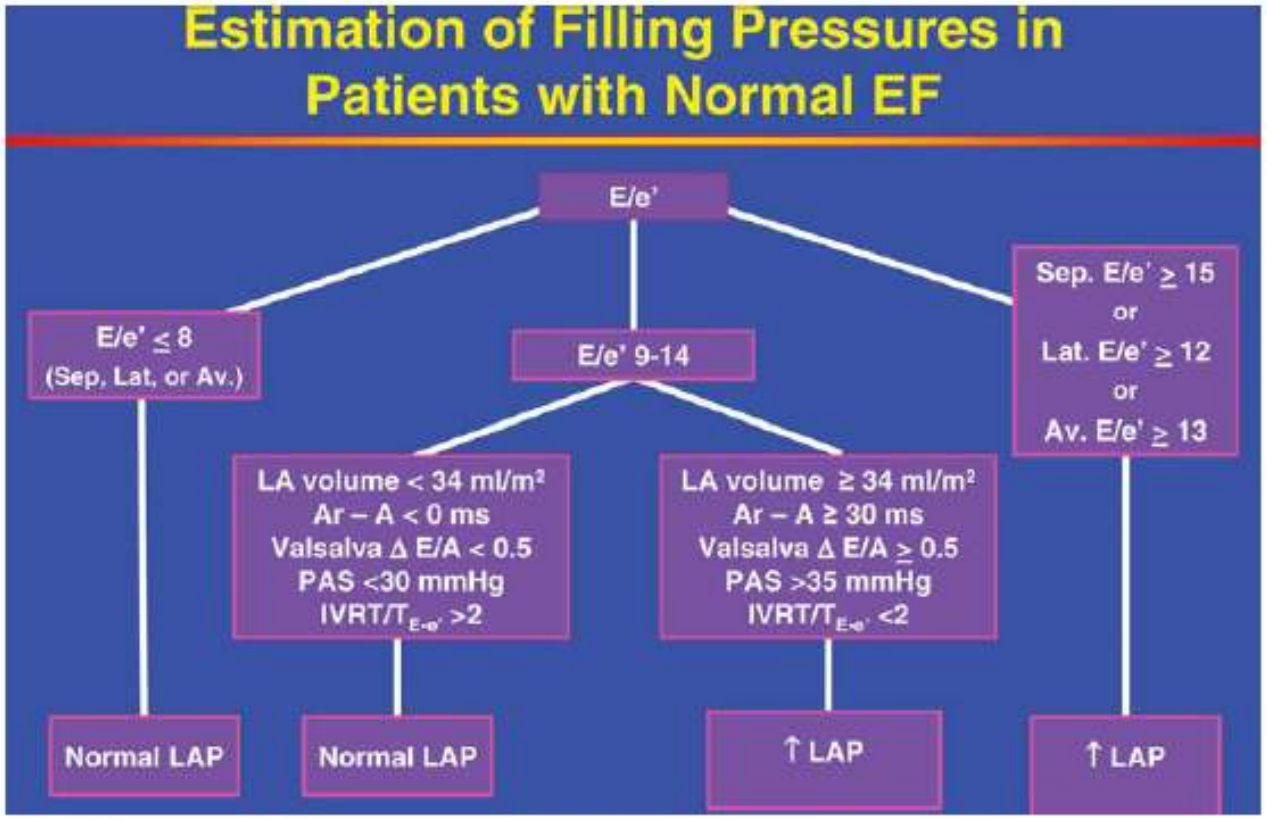


Figure 15 Diagnostic algorithm for the estimation of LV filling pressures in patients with normal EFs.