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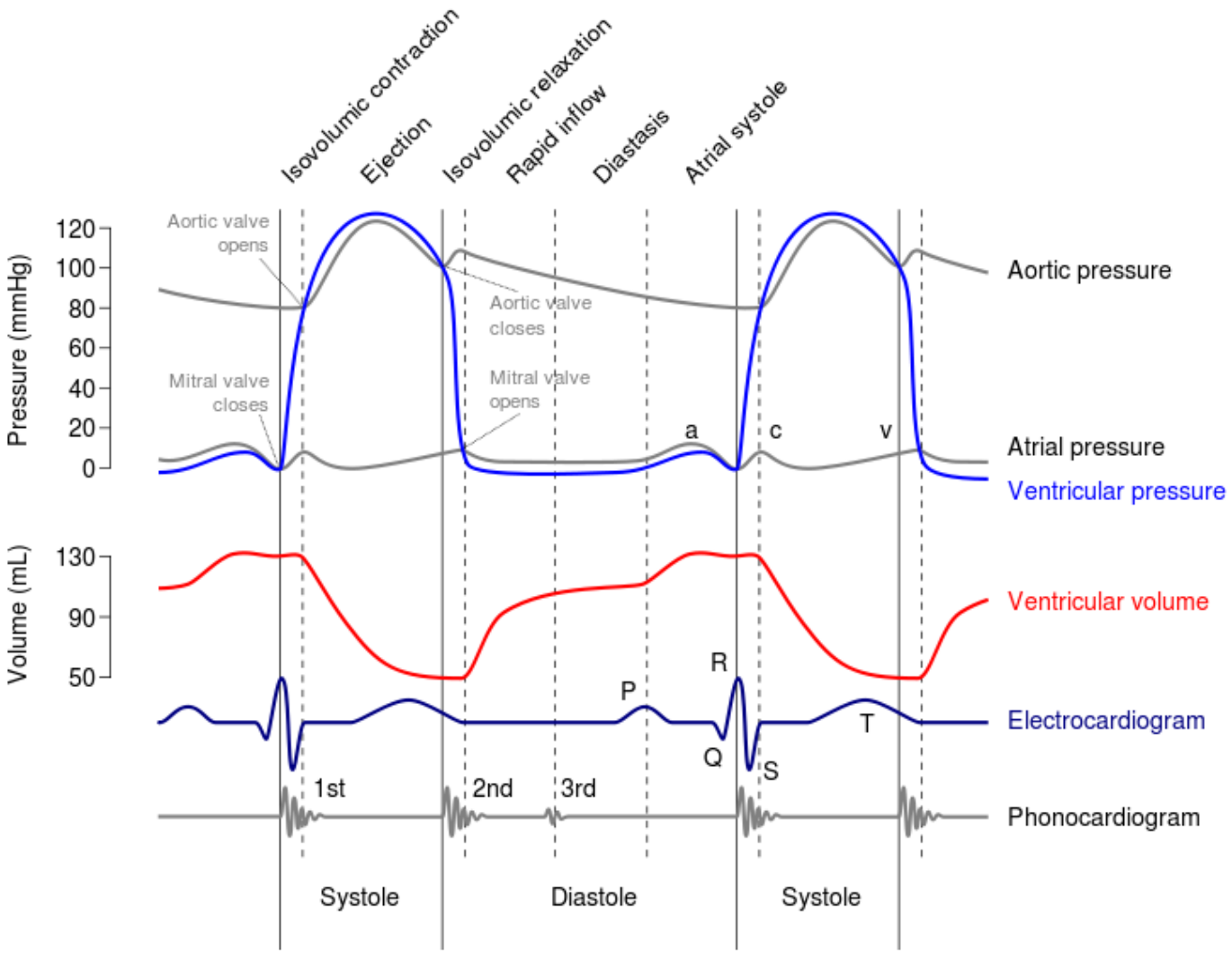
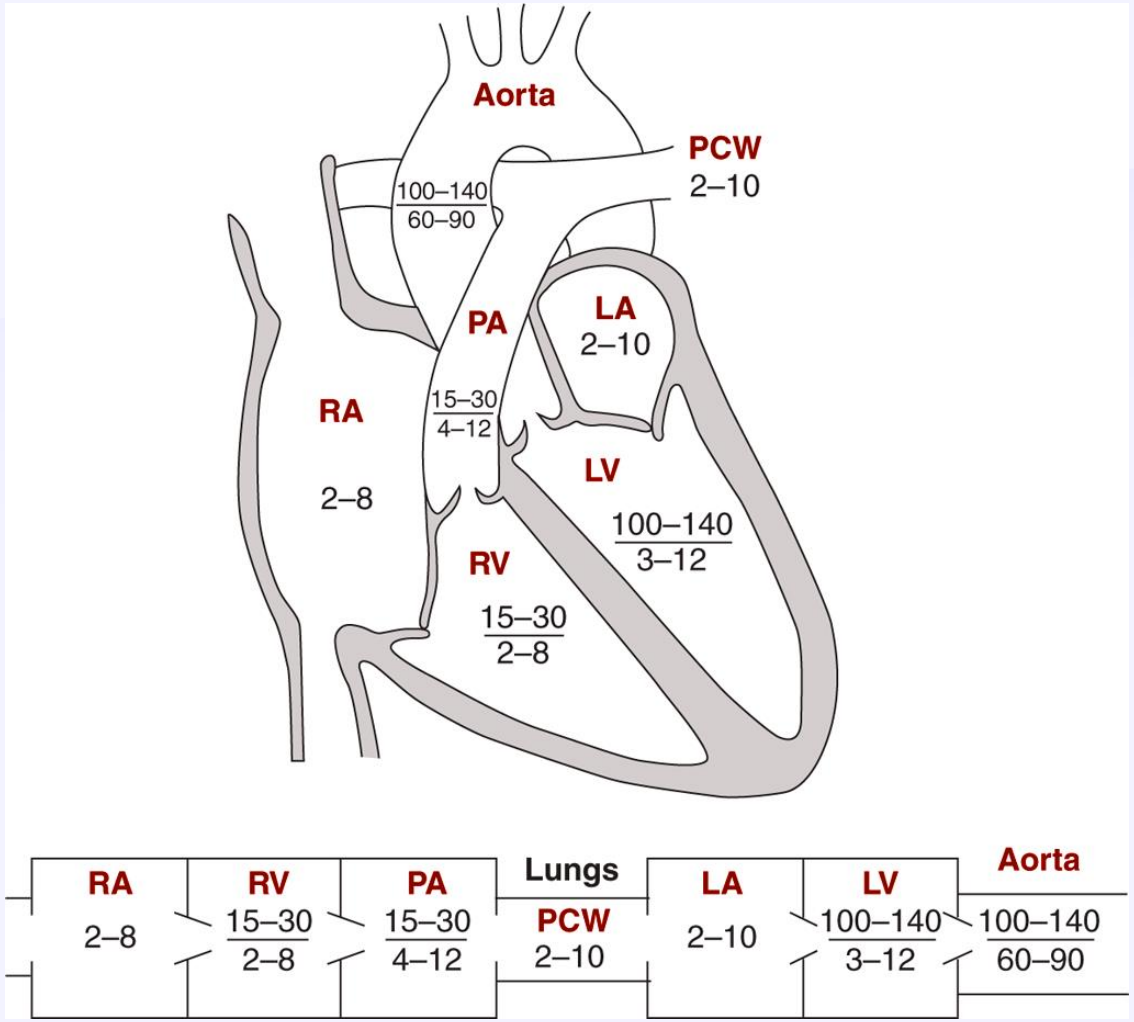
Diastology, Cardiomyopathies (Except HOCM)

July 3, 2019
Toronto General Hospital

1883- 1963

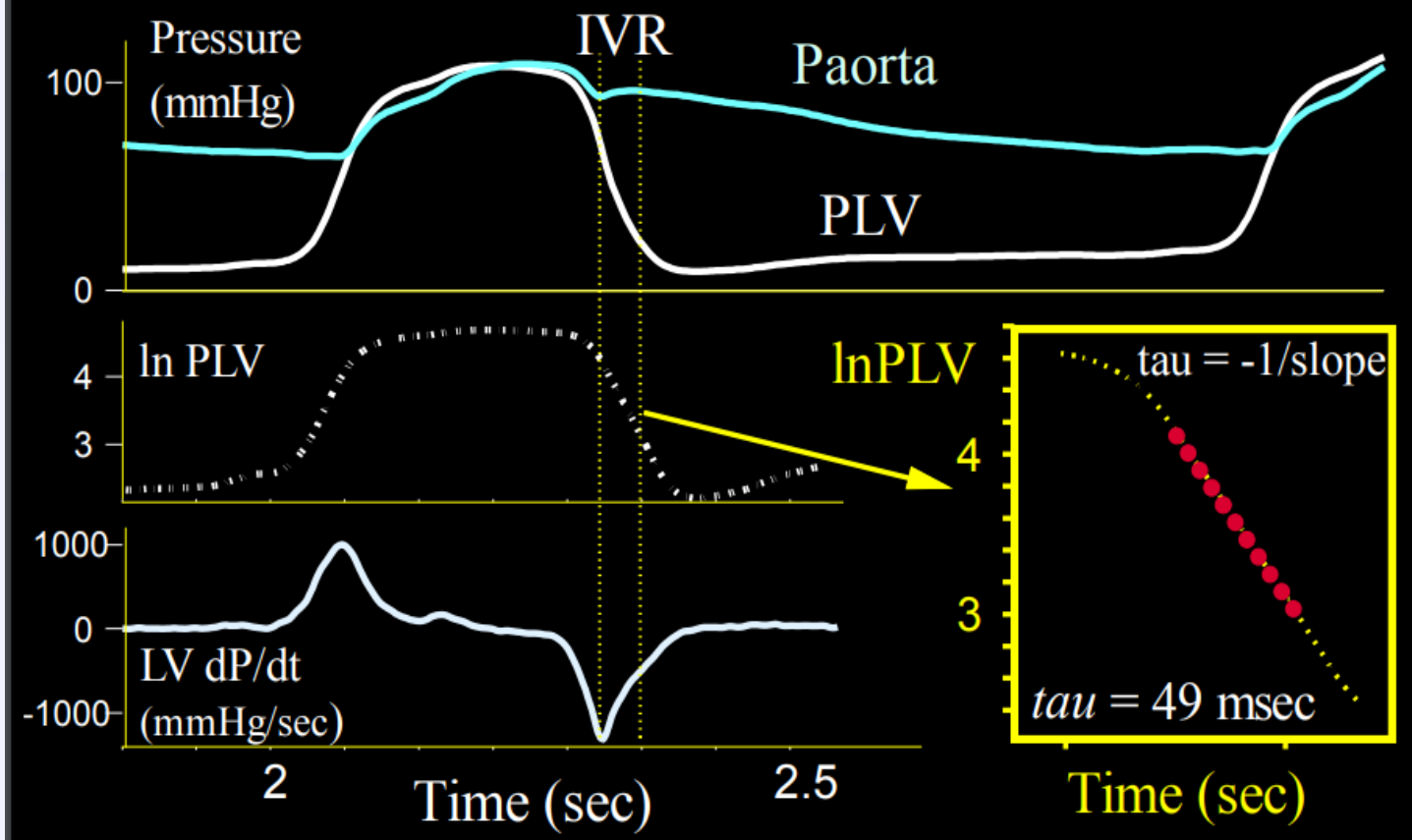
Wiggers diagram for cardiac events (left heart)





Normal pressures in cardiac chambers

Invasive measure of LV relaxation (*tau*)

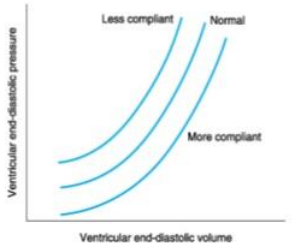


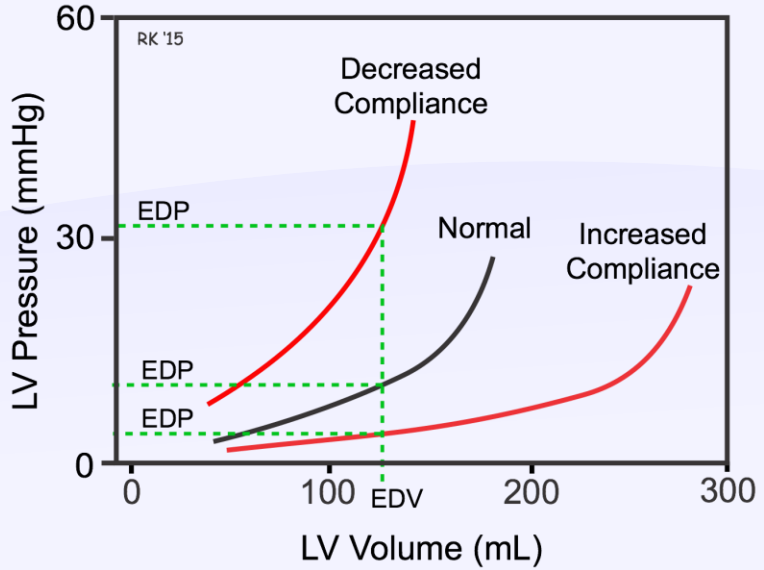
Tau (T, *t*) is time constant of LV relaxation

Smiseth et al, 2002.

(B) Cardiac Compliance:

- Technically compliance means change in volume for a given change in pressure .
- Ventricular compliance is normally nonlinear .
- With normal compliance, large increases in volume leads to a relatively small increases in pressure.
- Whereas in a less-compliant ventricle, a greater pressure is generated with very little increase in volume.





Estimation of Left Ventricular Stiffness

- LV stiffness is defined as the ratio of LV diastolic pressure and LV diastolic volume (LV dP/dV) at any given LV diastolic volume.
- LV compliance is the reciprocal of stiffness (LV dV/dP)

ASE/EACVI GUIDELINES AND STANDARDS

Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

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(J Am Soc Echocardiogr 2016;29:277-314.)

JASE 2016

The term *LV filling pressures* can refer to:

- Mean pulmonary capillary wedge pressure (PCWP) (which is indirect estimate of LV diastolic pressures)
- Mean left atrial pressure (LAP)
- LV pre- A pressure
- Mean LV diastolic pressure
- And LV end- diastolic pressure (LVEDP)

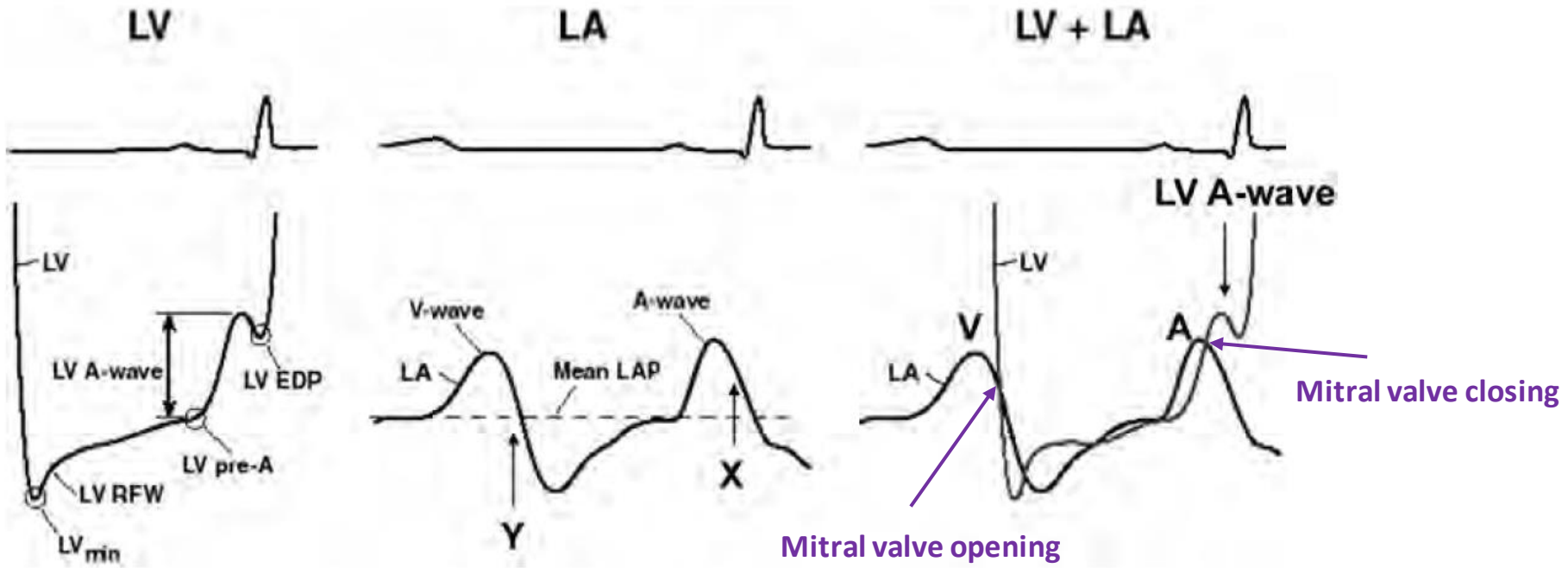

Figure 1 (*Left*) LV diastolic pressures recording. *Arrows* point to LV minimal pressure (min), LV rapid filling wave (RFW), LV pre-A pressure (pre-A), A wave rise with atrial contraction and end-diastolic pressure (EDP). (*Middle*) LAP recording showing "V" and "A" waves marked along with Y and X descent (*Right*) Simultaneous LV and LAP recording showing early and late transmitral pressure gradients. Notice that LA "A wave" pressure precedes the late diastolic rise (LV A wave) in LV pressure.

Table 1 Two-dimensional and Doppler methods for assessment of LV diastolic function

Variable	Acquisition	Analysis
Peak E-wave velocity (cm/sec)	1. Apical four-chamber with color flow imaging for optimal alignment of PW Doppler with blood flow. 2. PW Doppler sample volume (1–3 mm axial size) between mitral leaflet tips. 3. Use low wall filter setting (100–200 MHz) and low signal gain. 4. Optimal spectral waveforms should not display spikes or feathering.	Peak modal velocity in early diastole (after ECG T wave) at the leading edge of spectral waveform
Peak A-wave velocity (cm/sec)	1. Apical four-chamber with color flow imaging for optimal alignment of PW Doppler with blood flow 2. PW Doppler sample volume (1–3 mm axial size) between mitral leaflet tips. 3. Use low wall filter setting (100–200 MHz) and low signal gain. 4. Optimal spectral waveforms should not display spikes or feathering.	Peak modal velocity in late diastole (after ECG P wave) at the leading edge of spectral waveform
MV A duration (msec)	1. Apical four-chamber with color flow imaging for optimal alignment of PW Doppler with blood flow. 2. PW Doppler sample volume (1–3 mm axial size) at level of mitral annulus (limited data on how duration compares between annulus and leaflet tips) 3. Use low wall filter setting (100–200 MHz) and low signal gain. 4. Optimal spectral waveforms should not display spikes or feathering.	Time interval from A-wave onset to end of A wave at zero baseline. If E and A are fused (E velocity > 20 cm/sec when A velocity starts), A-wave duration will often be longer because of increased atrial filling stroke volume.
MV E/A ratio	See above for proper technique of acquisition of E and A velocities.	MV E velocity divided by A-wave velocity
MV DT (msec)	Apical four-chamber: pulsed Doppler sample volume between mitral leaflet tips	Time interval from peak E-wave along the slope of LV filling extrapolated to the zero-velocity baseline.

Table 1 Two-dimensional and Doppler methods for assessment of LV diastolic function **(Continued)**

Variable	Acquisition	Analysis
Pulsed-wave TDI e′ velocity (cm/sec)	1. Apical four-chamber view: PW Doppler sample volume (usually 5–10 mm axial size) at lateral and septal basal regions so average e′ velocity can be computed. 2. Use ultrasound system presets for wall filter and lowest signal gain. 3. Optimal spectral waveforms should be sharp and not display signal spikes, feathering or ghosting.	Peak modal velocity in early diastole at the leading edge of spectral waveform
Mitral E/e′	See above for acquisition of E and e′ velocities	MV E velocity divided by mitral annular e′ velocity
LA maximum volume index (mL/BSA)	1. Apical four- and two-chamber: acquire freeze frames 1–2 frames before MV opening. 2. LA volume should be measured in dedicated views in which LA length and transverse diameters are maximized.	Method of disks or area-length method and correct for BSA. Do not include LA appendage or pulmonary veins in LA tracings from apical four- and apical two-chamber views.
PV S wave (cm/sec)	1. Apical four-chamber with color flow imaging to help position pulsed Doppler sample volume (1–3 mm axial size). 2. Sample volume placed at 1–2 cm depth into right (or left) upper PV. 3. Use low wall filter setting (100–200 MHz) and low signal gain. 4. Optimized spectral waveforms should not display signal spikes or feathering.	Peak modal velocity in early systole at the leading edge of spectral waveform

Table 1 Two-dimensional and Doppler methods for assessment of LV diastolic function **(Continued)**

Variable	Acquisition	Analysis
PV D wave (cm/sec)	Same as for PV S wave.	Peak modal velocity in early diastole after MV opening at leading edge of spectral waveform
PV AR duration (msec)	Apical four-chamber: sample volume placed at 1–2 cm depth into right (or left) upper PV with attention to presence of LA wall motion artifacts	Time interval from AR-wave onset to end of AR at zero baseline
PV S/D ratio	See above for acquisition of pulmonary vein S and D velocities.	PV S wave divided by D-wave velocity or PV S wave time-velocity integral/PV D wave time-velocity integral.
CW Doppler: TR systolic jet velocity (m/sec)	1. Parasternal and apical four-chamber view with color flow imaging to obtain highest Doppler velocity aligned with CW. 2. Adjust gain and contrast to display complete spectral envelope without signal spikes or feathering	Peak modal velocity during systole at leading edge of spectral waveform

Table 1 (*Continued*)

Variable	Acquisition	Analysis
Valsalva maneuver	Recording obtained continuously through peak inspiration and as patient performs forced expiration for 10 sec with mouth and nose closed.	Change in MV E velocity and E/A ratio during peak strain and following release
Secondary measures		
Color M-mode Vp (cm/sec)	Apical four-chamber with color flow imaging for M-mode cursor position, shift color baseline in direction of mitral valve inflow to lower velocity scale for red/yellow inflow velocity profile	Slope of inflow from MV plane into LV chamber during early diastole at 4-cm distance
IVRT	Apical long-axis or five-chamber view, using CW Doppler and placing sample volume in LV outflow tract to simultaneously display end of aortic ejection and onset of mitral inflow.	Time between aortic valve closure and MV opening. For IVRT, sweep speed should be 100 mm/sec.
TE-e′	Apical four-chamber view with proper alignment to acquire mitral inflow at mitral valve tips and using tissue Doppler to acquire septal and lateral mitral annular velocities.	Time interval between peak of R wave in QRS complex and onset of mitral E velocity is subtracted from time interval between QRS complex and onset of e′ velocity. RR intervals should be matched and gain and filter settings should be optimized to avoid high gain and filter settings. For time intervals, sweep speed should be 100 mm/sec.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function

Variable	Utility and physiologic background	Advantages	Limitations
Mitral E velocity	E-wave velocity reflects the LA-LV pressure gradient during early diastole and is affected by alterations in the rate of LV relaxation and LAP.	1. Feasible and reproducible. 2. In patients with dilated cardiomyopathy and reduced LVEF, mitral velocities correlate better with LV filling pressures, functional class, and prognosis than LVEF.	1. In patients with coronary artery disease and patients with HCM in whom LVEF is >50%, mitral velocities correlate poorly with LV filling pressures 2. More challenging to apply in patients with arrhythmias. 3. Directly affected by alterations in LV volumes and elastic recoil. 4. Age dependent (decreasing with age).
Mitral A velocity	A-wave velocity reflects the LA-LV pressure gradient during late diastole, which is affected by LV compliance and LA contractile function.	Feasible and reproducible.	1. Sinus tachycardia, first-degree AV block and paced rhythm can result in fusion of the E and A waves. If mitral flow velocity at the start of atrial contraction is >20 cm/sec, A velocity may be increased. 2. Not applicable in AF/atrial flutter patients. 3. Age dependent (increases with aging).
Mitral E/A ratio	Mitral inflow E/A ratio and DT are used to identify the filling patterns: normal, impaired relaxation, PN, and restrictive filling.	1. Feasible and reproducible. 2. Provides diagnostic and prognostic information. 3. In patients with dilated cardiomyopathy, filling patterns correlate better with filling pressures, functional class, and prognosis than LVEF. 4. A restrictive filling pattern in combination with LA dilation in patients with normal EFs is associated with a poor prognosis similar to a restrictive pattern in dilated cardiomyopathy.	1. The U-shaped relation with LV diastolic function makes it difficult to differentiate normal from PN filling, particularly with normal LVEF, without additional variables. 2. If mitral flow velocity at the start of atrial contraction is >20 cm/sec, E/A ratio will be reduced due to fusion. 3. Not applicable in AF/atrial flutter patients. 4. Age dependent (decreases with aging).
Mitral E-velocity DT	DT is influenced by LV relaxation, LV diastolic pressures following mitral valve opening, and LV stiffness.	1. Feasible and reproducible. 2. A short DT in patients with reduced LVEFs indicates increased LVEDP with high accuracy both in sinus rhythm and in AF.	1. DT does not relate to LVEDP in normal LVEF 2. Should not be measured with E and A fusion due to potential inaccuracy. 3. Age dependent (increases with aging). 4. Not applied in atrial flutter.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function **(Continued)**

Variable	Utility and physiologic background	Advantages	Limitations
Changes in mitral inflow with Valsalva maneuver	Helps distinguishing normal from PN filling patterns. A decrease of E/A ratio of ≥50% or an increase in A-wave velocity during the maneuver, not caused by E and A fusion, are highly specific for increased LV filling pressures.	When performed adequately under standardized conditions (keeping 40 mm Hg intrathoracic pressure constant for 10 sec) accuracy in diagnosing increased LV filling pressures is good.	1. Not every patient can perform this maneuver adequately. The patient must generate and sustain a sufficient increase in intrathoracic pressure, and the examiner needs to maintain the correct sample volume location between the mitral leaflet tips during the maneuver. 2. It is difficult to assess if it is not standardized.
Mitral "L" velocity	Markedly delayed LV relaxation in the setting of elevated LV filling pressures allows for ongoing LV filling in mid diastole and thus L velocity. Patients usually have bradycardia.	When present in patients with known cardiac disease (e.g., LVH, HCM), it is specific for elevated LV filling pressures. However, its sensitivity is overall low.	Rarely seen in normal LV diastolic function when the subject has bradycardia but it is then usually <20 cm/sec.
IVRT	IVRT is ≤70 msec in normal subjects and is prolonged in patients with impaired LV relaxation but normal LV filling pressures. When LAP increases, IVRT	1. Overall feasible and reproducible. 2. IVRT can be combined with other mitral inflow parameters as E/A ratio to estimate LV filling pressures in patients with HFrEF.	1. IVRT duration is in part affected by heart rate and arterial pressure. 2. More challenging to measure and interpret with tachycardia.
	shortens and its duration is inversely related to LV filling pressures in patients with cardiac disease.	3. It can be combined with LV end-systolic pressure to estimate the time constant of LV relaxation (τ). 4. It can be applied in patients with mitral stenosis in whom the same relation with LV filling pressures described above holds. 5. In patients with MR and in those after MV replacement or repair, it can be combined with $T_{E-e'}$ to estimate LV filling pressures.	3. Results differ on the basis of using CW or PW Doppler for acquisition.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function **(Continued)**

Variable	Utility and physiologic background	Advantages	Limitations
Pulsed-wave TDI-derived mitral annular early diastolic velocity: e′	A significant association is present between e′ and the time constant of LV relaxation (τ) shown in both animals and humans. The hemodynamic determinants of e′ velocity include LV relaxation, restoring forces and filling pressure.	1. Feasible and reproducible. 2. LV filling pressures have a minimal effect on e′ in the presence of impaired LV relaxation. 3. Less load dependent than conventional blood-pool Doppler parameters.	1. Limited accuracy in patients with CAD and regional dysfunction in the sampled segments, significant MAC, surgical rings or prosthetic mitral valves and pericardial disease. 2. Need to sample at least two sites with precise location and adequate size of sample volume. 3. Different cutoff values depending on the sampling site for measurement. 4. Age dependent (decreases with aging).
Mitral E/e′ ratio	e′ velocity can be used to correct for the effect of LV relaxation on mitral E velocity, and E/e′ ratio can be used to predict LV filling pressures.	1. Feasible and reproducible. 2. Values for average E/e′ ratio < 8 usually indicate normal LV filling pressures, values > 14 have high specificity for increased LV filling pressures.	1. E/e′ ratio is not accurate in normal subjects, patients with heavy annular calcification, mitral valve and pericardial disease. 2. "Gray zone" of values in which LV filling pressures are indeterminate. 3. Accuracy is reduced in patients with CAD and regional dysfunction at the sampled segments. 4. Different cutoff values depending on the site used for measurement.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function **(Continued)**

Variable	Utility and physiologic background	Advantages	Limitations
$T_{E\text{-}e'}$ time interval	Can identify patients with diastolic dysfunction due to delayed onset of e′ velocity compared with onset of mitral E velocity.	1. Ratio of IVRT to $T_{E\text{-}e'}$ can be used to estimate LV filling pressures in normal subjects and patients with mitral valve disease. 2. $T_{E\text{-}e'}$ can be used to differentiate patients with restrictive cardiomyopathy who have a prolonged time interval from those with pericardial constriction in whom it is not usually prolonged.	More challenging to acquire satisfactory signals with close attention needed to location, gain, filter settings as well as matching RR intervals.
LA maximum volume index	LA volume reflects the cumulative effects of increased LV filling pressures over time. Increased LA volume is an independent predictor of death, heart failure, AF, and ischemic stroke.	1. Feasible and reproducible. 2. Provides diagnostic and prognostic information about LV diastolic dysfunction and chronicity of disease. 3. Apical four-chamber view provides visual estimate of LA and RA size which confirms LA is enlarged.	1. LA dilation is seen in bradycardia, high-output states, heart transplants with biatrial technique, atrial flutter/fibrillation, significant mitral valve disease, despite normal LV diastolic function. 2. LA dilatation occurs in well-trained athletes who have bradycardia and are well hydrated. 3. Suboptimal image quality, including LA foreshortening, in technically challenging studies precludes accurate tracings. 4. It can be difficult to measure LA volumes in patients with ascending and descending aortic aneurysms as well as in patients with large interatrial septal aneurysms.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function **(Continued)**

Variable	Utility and physiologic background	Advantages	Limitations
Pulmonary veins: systolic (S) velocity, diastolic (D) velocity, and S/D ratio	S-wave velocity (sum of S1 and S2) is influenced by changes in LAP, LA contractility, and LV and RV contractility. D-wave velocity is mainly influenced by early diastolic LV filling and compliance and it changes in parallel with mitral E velocity. Decrease in LA compliance and increase in LAP is associated with decrease in S velocity and increase in D velocity.	1. Reduced S velocity, S/D ratio < 1, and systolic filling fraction (systolic VTI/total forward flow VTI) < 40% indicate increased mean LAP in patients with reduced LVEFs. 2. In patients with AF, DT of diastolic velocity (D) in pulmonary vein flow can be used to estimate mean PCWP.	1. Feasibility of recording PV inflow can be suboptimal, particularly in ICU patients. 2. The relationship between PV systolic filling fraction and LAP has limited accuracy in patients with normal LVEF, AF, mitral valve disease and HCM.
Ar-A duration	The time difference between duration of PV flow and mitral inflow during atrial contraction is associated with LV pressure rise because of atrial contraction and LVEDP. The longer the time difference, the higher LVEDP.	1. PV Ar duration > mitral A duration by 30 msec indicates an increased LVEDP. 2. Independent of age and LVEF. 3. Accurate in patients with MR and patients with HCM.	1. Adequate recordings of Ar duration may not be feasible by TTE in several patients. 2. Not applicable in AF patients. 3. Difficult to interpret in patients with sinus tachycardia or first-degree AV block with E and A fusion.
CW Doppler TR systolic jet velocity	A significant correlation exists between systolic PA pressure and noninvasively derived LAP. In the absence of pulmonary disease, increased systolic PA pressure suggests elevated LAP.	Systolic PA pressure can be used as an adjunctive parameter of mean LAP. Evidence of pulmonary hypertension has prognostic implications.	1. Indirect estimate of LAP. 2. Adequate recording of a full envelope is not always possible, though intravenous agitated saline or contrast increases yield. 3. With severe TR and low systolic RV-RA pressure gradient, accuracy of calculation is dependent on reliable estimation of RA systolic pressure.

Table 2 Utility, advantages and limitations of variables used to assess LV diastolic function **(Continued)**

Variable	Utility and physiologic background	Advantages	Limitations
CW Doppler PR end-diastolic velocity	A significant correlation exists between diastolic PA pressure and invasively as well as noninvasively derived LAP. In the absence of pulmonary disease, increased diastolic PA pressure is consistent with elevated LAP.	Diastolic PA pressure can be used as an adjunctive parameter of mean LAP. Evidence of pulmonary hypertension has prognostic implications.	1. Adequate recording of a full PR jet envelope is not always possible though intravenous contrast increases yield. 2. Accuracy of calculation is dependent on the reliable estimation of mean RAP. 3. If mean PA pressure is >40 mm Hg or PVR >200 dynes·s·cm^{-5}, PA diastolic pressure is higher by >5 mm Hg over mean PCWP.
Color M-mode Vp: Vp, and E/Vp ratio	Vp correlates with the time constant of LV relaxation (τ) and can be used as a parameter of LV relaxation. E/Vp ratio correlates with LAP.	1. Vp is reliable as an index of LV relaxation in patients with depressed LVEFs and dilated left ventricle but not in patients with normal EFs. 2. E/Vp $\geq$ 2.5 predicts PCWP >15 mm Hg with reasonable accuracy in patients with depressed EFs.	1. There are different methods for measuring mitral-to-apical flow propagation. 2. In patients with normal LV volumes and LVEF but elevated LV filling pressures, Vp can be misleadingly normal. 3. Lower feasibility and reproducibility. 4. Angulation between M-mode cursor and flow results in erroneous measurements.

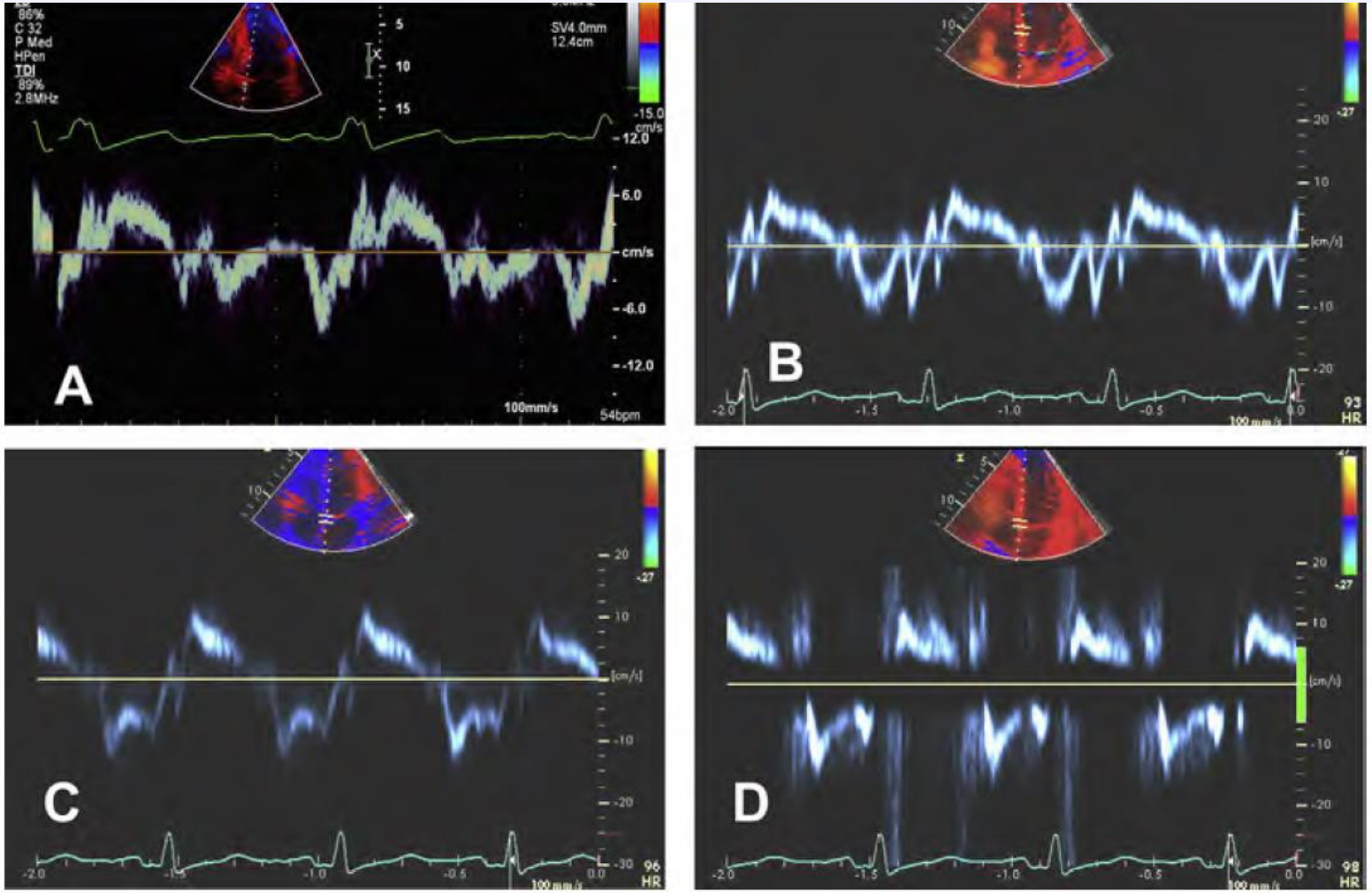

Figure 2 Tissue Doppler recordings of septal mitral annular velocities. In **(A)**, Doppler settings and sample volume location are optimal, whereas in **(B)** the sample volume is placed in the ventricular septum (not annulus). Doppler setting are suboptimal in **(C)** with low gain and in **(D)** with high filter.

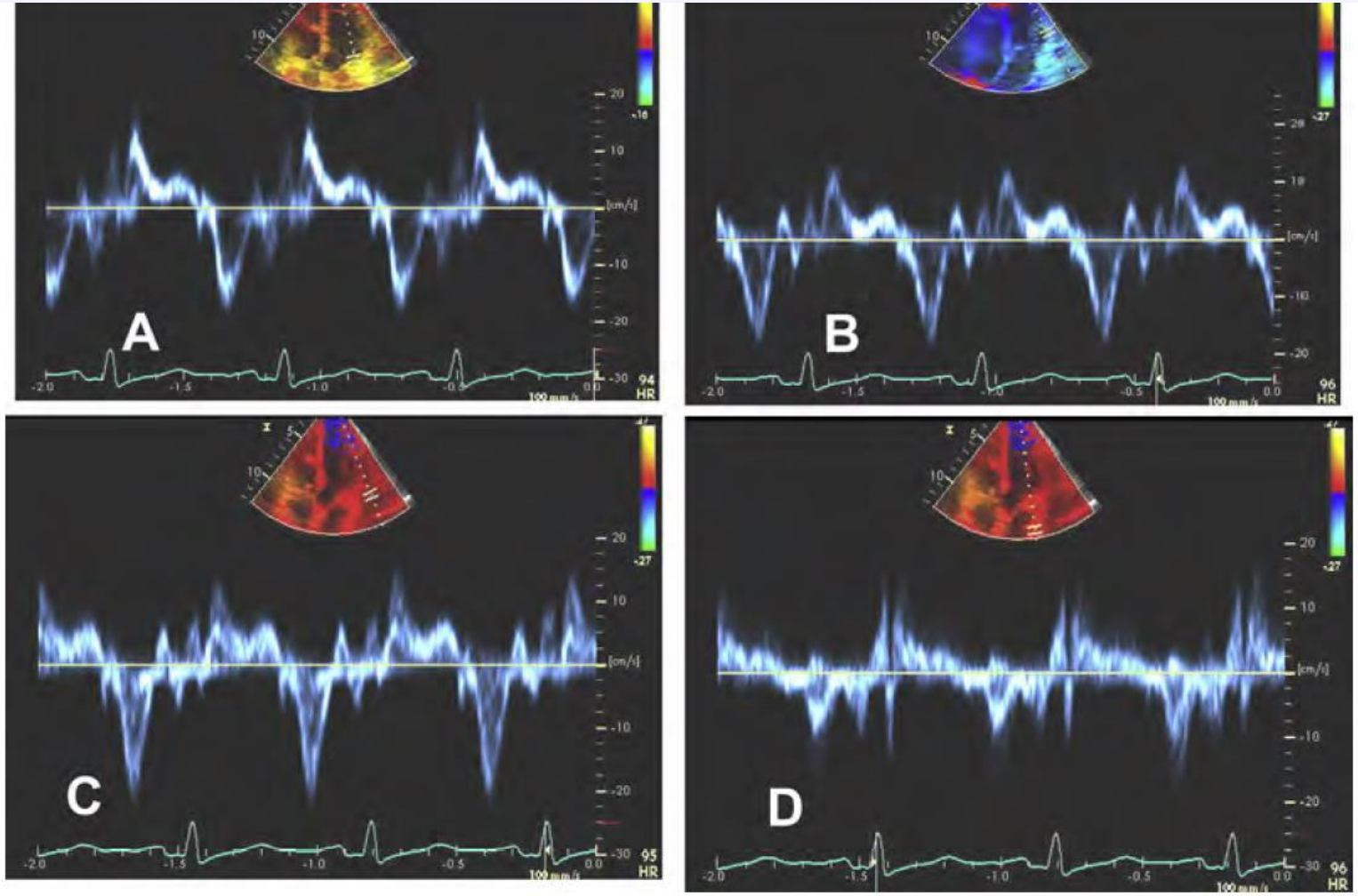

Figure 3 Tissue Doppler recordings of lateral mitral annular velocities. In **(A)**, Doppler sample volume is located in part in LV cavity. In **(B)** the sample volume is in basal segment of lateral wall, in **(C)** the location is partly outside the heart altogether, and in **(D)** it is located in the left atrium above the mitral annulus.

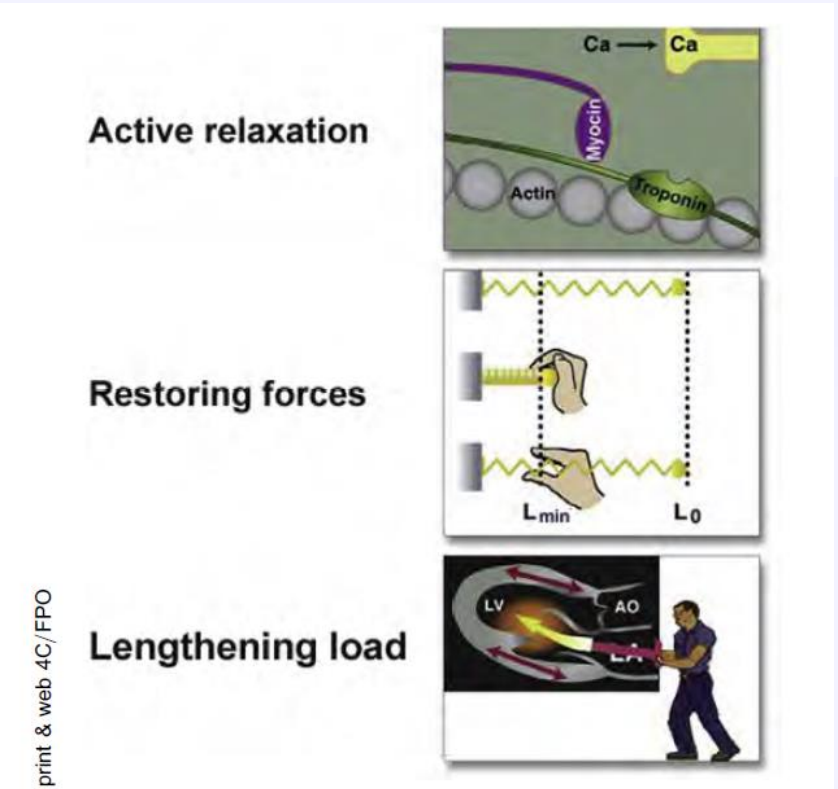

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Figure 4 The figure shows the three independent determinants of e′, which are LV relaxation, restoring forces, and lengthening load. Rate of relaxation reflects decay of active fiber force. Restoring forces which account for diastolic suction, are illustrated by an elastic spring which is compressed to a dimension (L_{min}) less than its resting length (L_0) and recoils back to resting length when the compression is released. Lengthening load is the pressure in the left atrium at mitral valve opening, which "pushes" blood into the left ventricle and thereby lengthens the ventricle. The figure is based on data from Opdahl *et al*.[35]

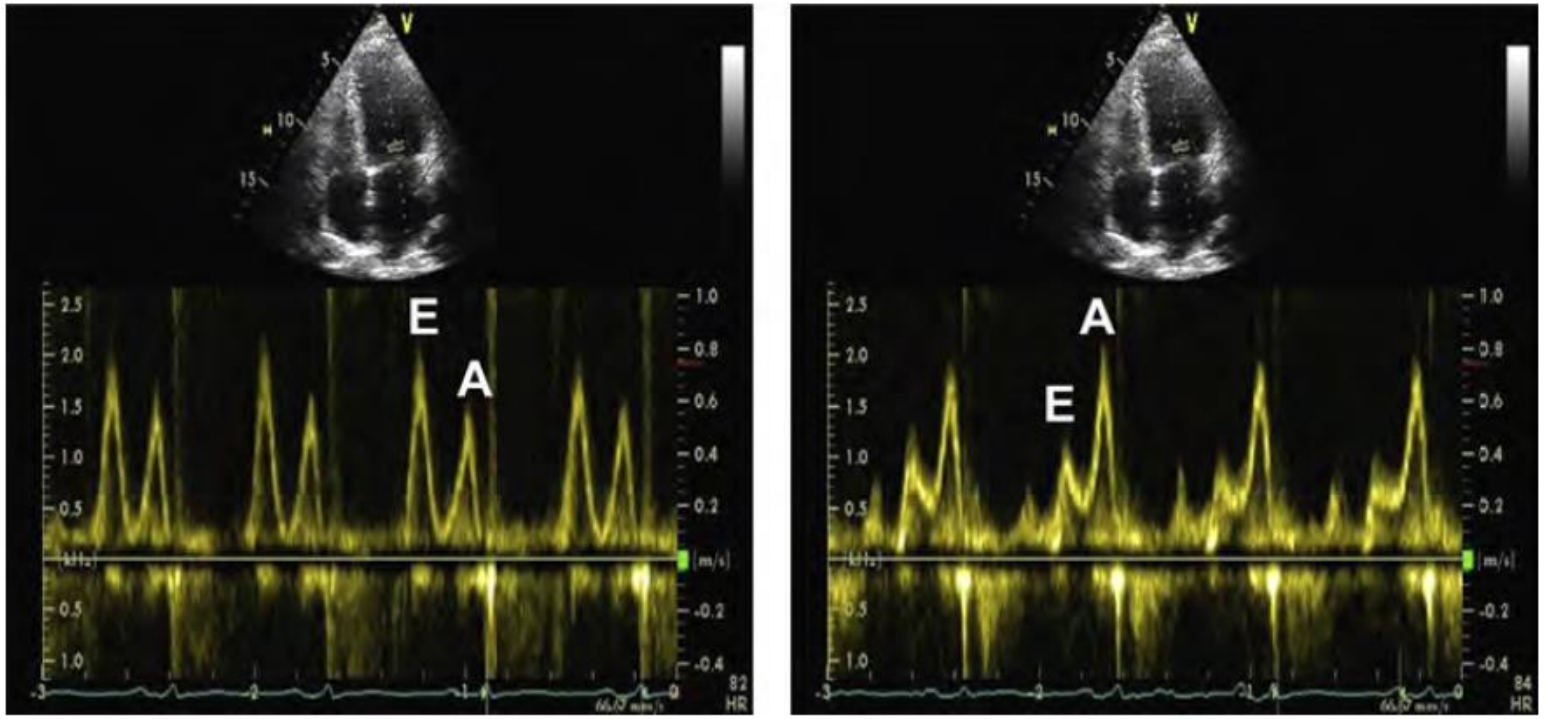


Figure 5 Valsalva maneuver in a patient with grade II diastolic dysfunction. At baseline, E/A ratio is 1.3 (*left*) and decreases to 0.6 (impaired relaxation pattern) with Valsalva.

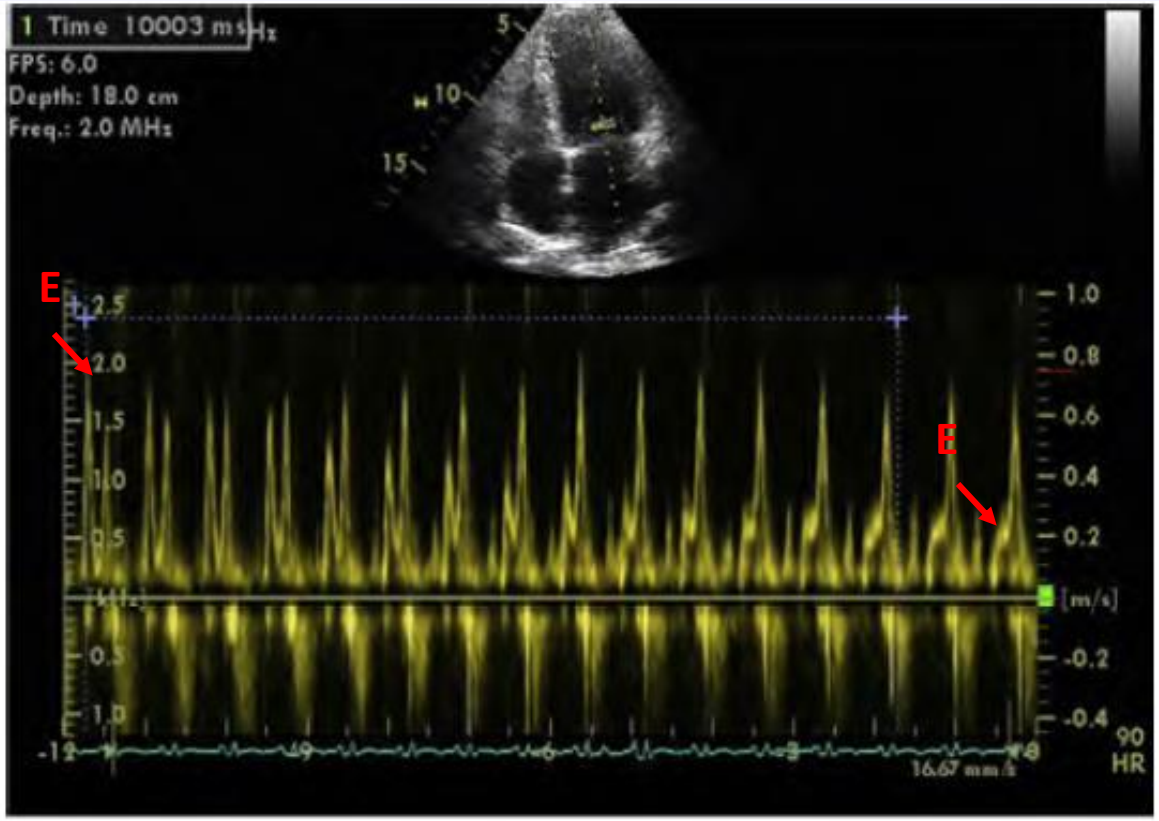


Figure 6 Continuous recording of mitral inflow during standardized Valsalva maneuver for 10 sec showing the decrease in E/A ratio with straining, which is consistent with elevated LV filling pressures.

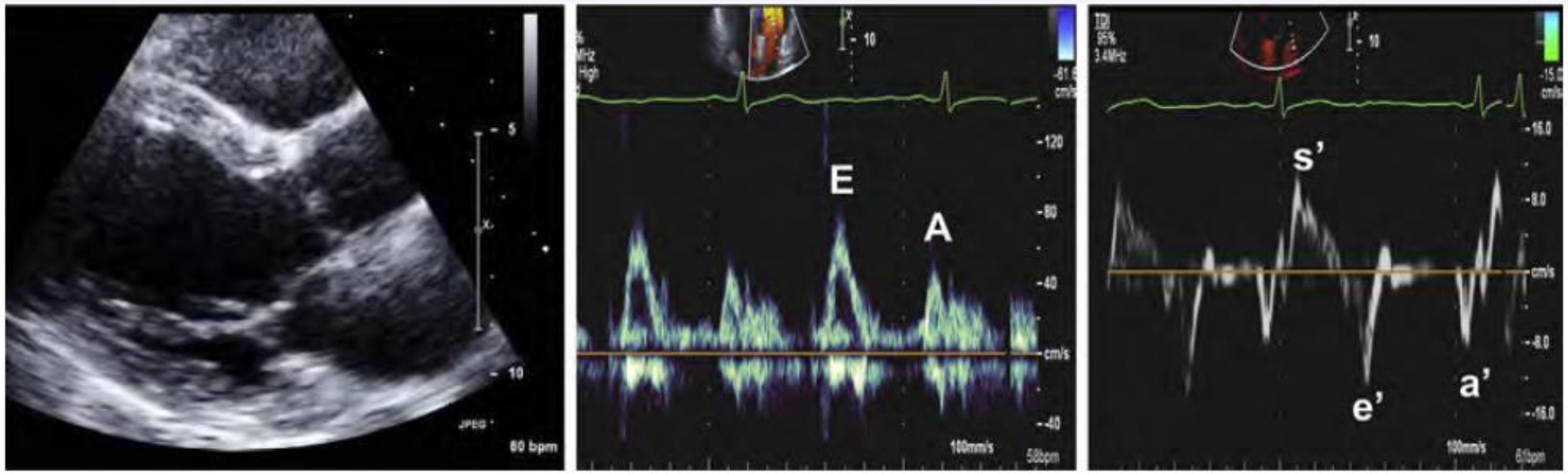
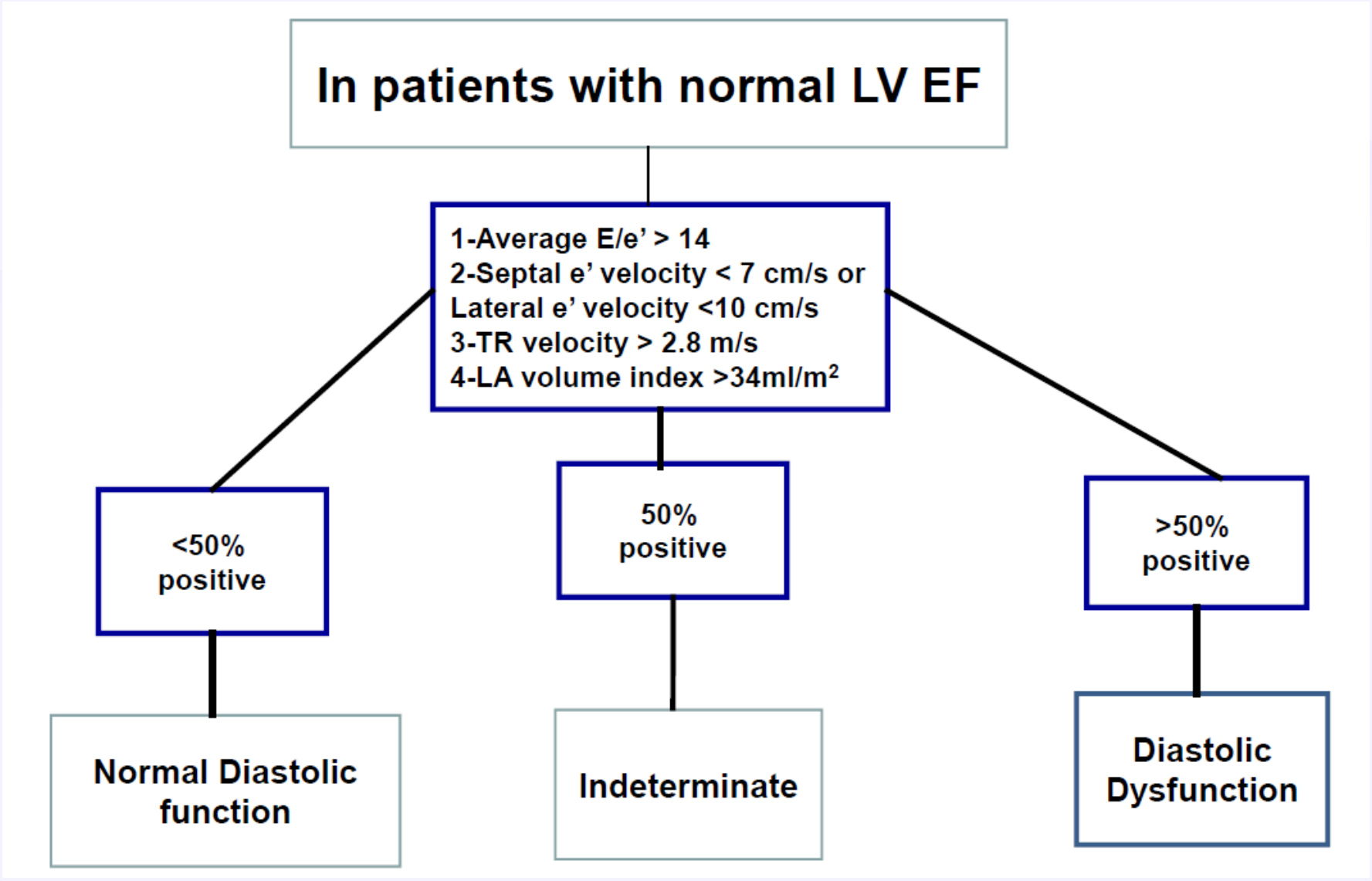


Figure 7 Example of normal findings from a young subject. *Left* shows normal LV size in parasternal long-axis view, with a normal mitral inflow pattern and E/A ratio > 1 in *middle panel*. Lateral e′ velocity is normal at 12 cm/sec (*left*).

Key Points

1. The four recommended variables for identifying diastolic dysfunction and their abnormal cutoff values are annular e′ velocity: septal e′ < 7 cm/sec, lateral e′ < 10 cm/sec, average E/e′ ratio > 14, LA volume index > 34 mL/m^2, and peak TR velocity > 2.8 m/sec.
2. LV diastolic function is normal if more than half of the available variables do not meet the cutoff values for identifying abnormal function. LV diastolic dysfunction is present if more than half of the available parameters meet these cutoff values. The study is inconclusive if half of the parameters do not meet the cutoff values.

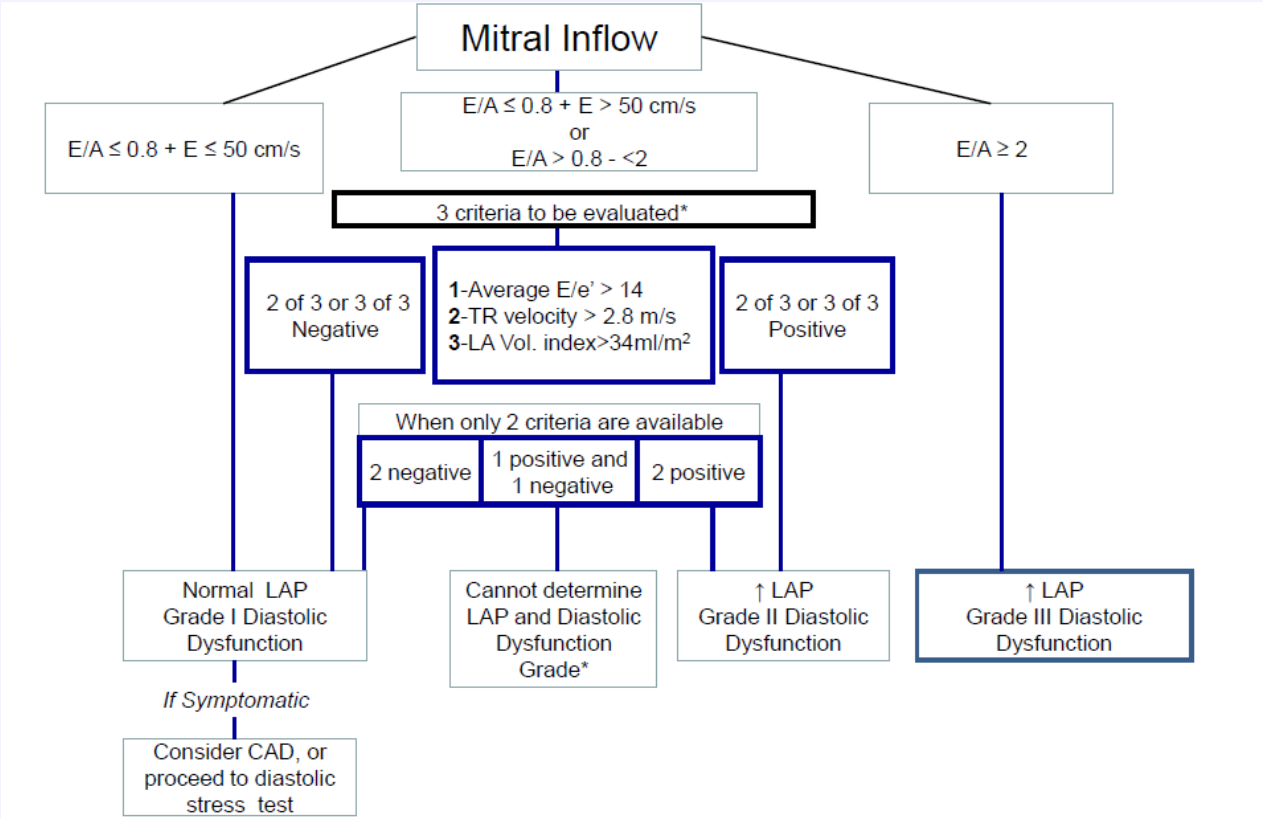
Diagnosis of diastolic dysfunction in the presence normal LVEF


In patients with normal LV EF
1-Average E/e' > 14
2-Septal e' velocity < 7 cm/s or
Lateral e' velocity <10 cm/s
3-TR velocity > 2.8 m/s
4-LA volume index >34ml/m2
<50% positive
50% positive
>50% positive
Normal Diastolic function
Indeterminate
Diastolic Dysfunction

Key Points

1. In patients with reduced LVEFs, transmitral inflow pattern is usually sufficient to identify patients with increased LAP and DT of mitral E velocity is an important predictor of outcome.
2. In patients with preserved LVEFs, several parameters, including 2D variables, are often needed to estimate LAP.
3. In patients with depressed EFs and in patients with normal EFs and myocardial disease, if E/A ratio is ≤0.8 along with a peak E velocity of ≤50 cm/sec, then mean LAP is either normal or low and patient has grade I diastolic dysfunction.
4. In patients with depressed EFs and in patients with normal EFs and myocardial disease, if E/A ratio is ≥ 2, LA mean pressure is elevated and grade III diastolic dysfunction is present. DT is usually short in patients with HFrEF and restrictive filling pattern (<160 msec). However, in patients with HFpEF, DT can be normal despite elevated LV filling pressures.
5. In patients with depressed EFs and in patients with normal EFs and myocardial disease, E/A ratio ≤ 0.8 along with a peak E velocity of >50 cm/sec, or an E/A ratio > 0.8 but < 2, additional parameters are needed. These include peak TR velocity, E/e′ ratio and LA maximum volume index. Their cutoff values to conclude elevated LAP are peak velocity of TR jet >2.8 m/sec, average E/e′ ratio>14, and LA maximum volume index > 34 mL/m^2. If more than half or all of the variables meet the cutoff values, then LAP is elevated and grade II diastolic dysfunction is present. If only one of three available variables meets the cutoff value, then LAP is normal and grade I diastolic dysfunction is present. In case of 50% discordance or with only one available variable, findings are inconclusive to estimate LAP.
6. In patients with depressed LVEFs, pulmonary vein S/D ratio may be used if one of the three main parameters is not available. A ratio < 1 is consistent with increased LAP.

Diagnosis of diastolic dysfunction in the presence of reduced LVEF



Algorithm for estimation of LV filling pressures and grading LV diastolic function in patients with depressed LVEF and patients with myocardial disease and normal LVEF after consideration of clinical and other 2D data.

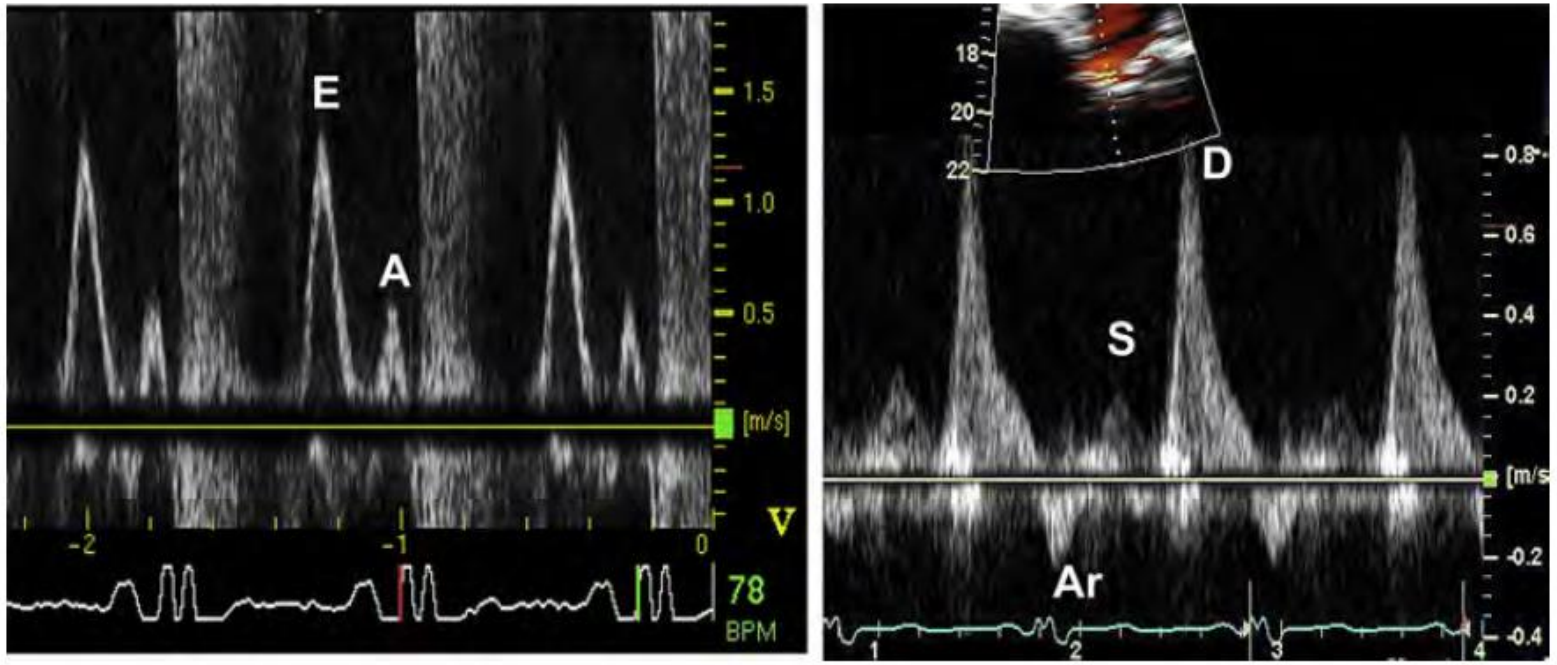


Figure 10 Mitral inflow (*left*) and pulmonary venous flow (*right*) from a patient with HFrEF. Notice the increased E/A ratio >2 and reduced S/D ratio in pulmonary venous flow. Both findings are consistent with increased LAP in this patient population.

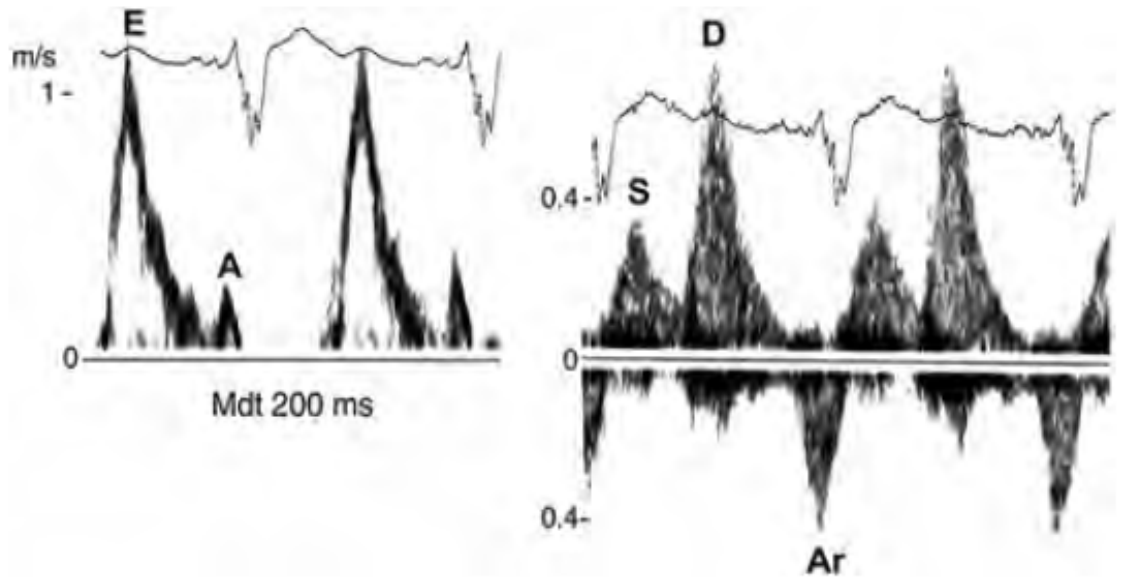


Figure 12 (*Left*) Mitral inflow from a patient with HFpEF. Mitral inflow pattern is consistent with elevated LV filling pressures. Notice the abbreviated mitral A velocity with short duration. DT of mitral E velocity (Mdt) measured at 200 msec. This is seen in patients with markedly delayed LV relaxation such that LV diastolic pressure continues to decline after mitral valve opening. (*Right*) Pulmonary venous flow from the same patient. Notice the decreased S/D ratio and the increased amplitude and velocity of Ar signal consistent with increased LVEDP. Abbreviations as in other figures.

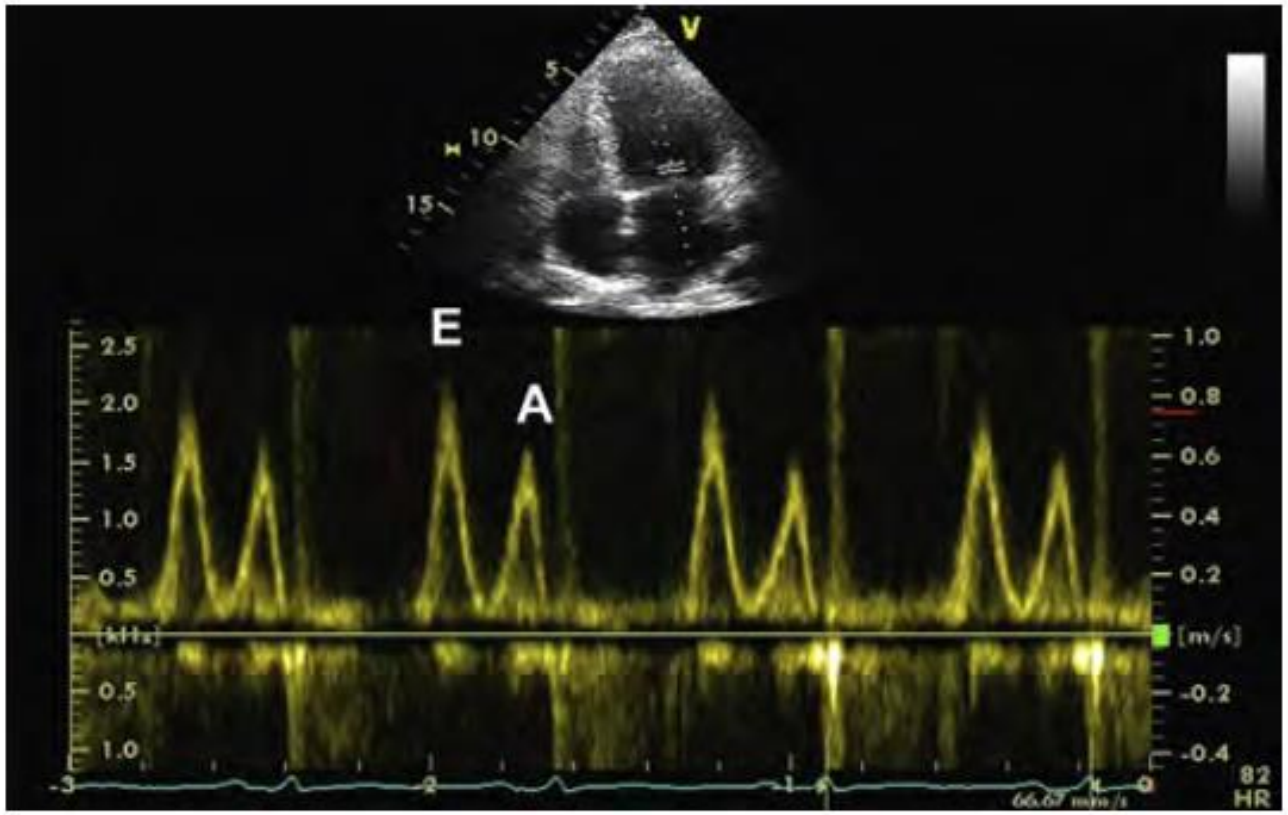


Figure 13 Mitral inflow from a patient with hypertensive heart disease with normal EF. Patient has LV hypertrophy and a moderately enlarged left atrium. Mitral inflow shows pseudonormal LV filling pattern consistent with elevated LV filling pressures and grade II diastolic dysfunction.

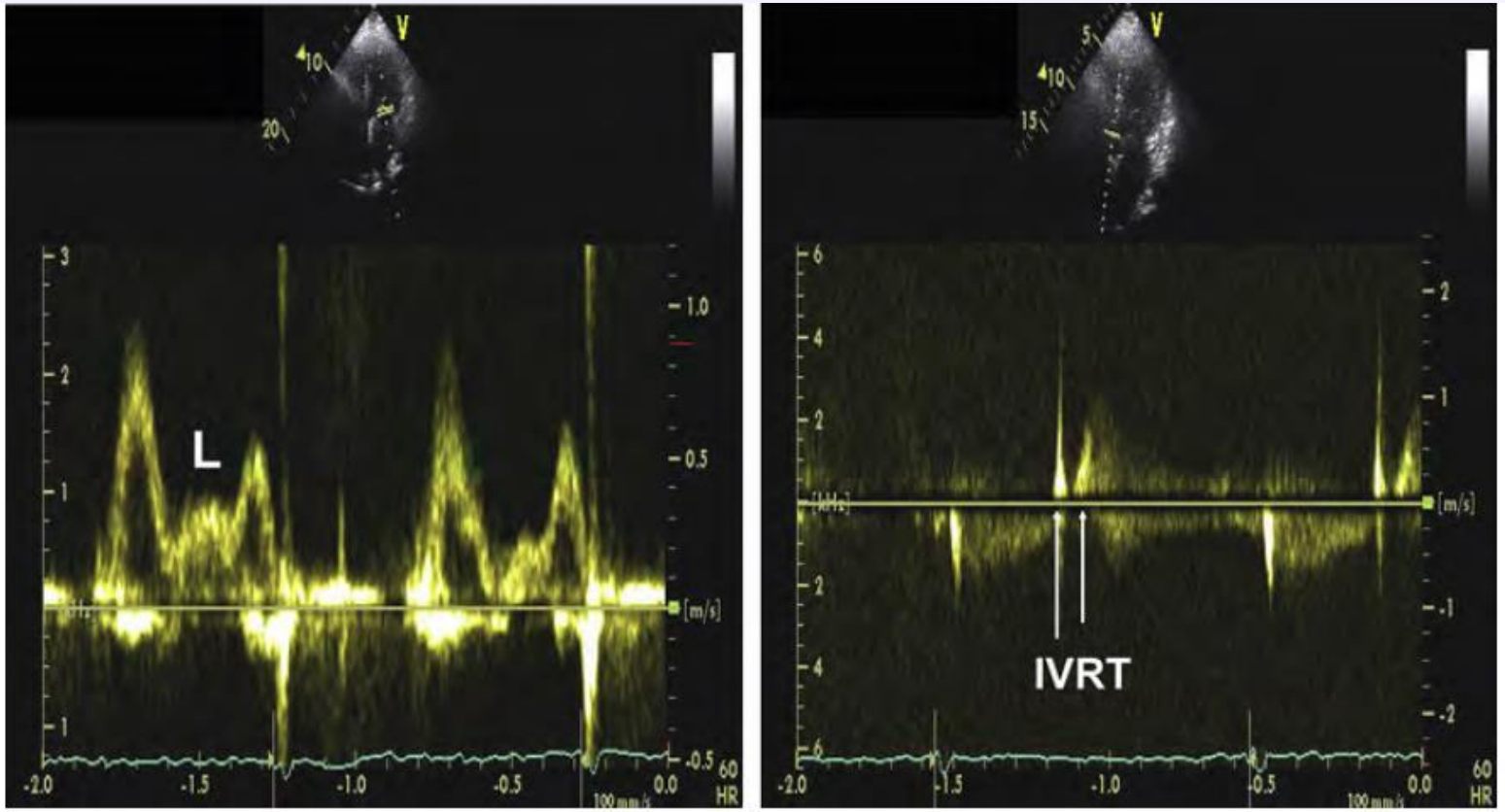


Figure 14 Mitral inflow (*left*) and IVRT (*right*) from another patient with HFpEF and heart rate 60 beats/min. E velocity was 96 cm/sec with A velocity of 65 cm/sec. Mid-diastolic flow (L velocity) is present because of the slow and impaired LV relaxation and the increased LAP. The arrows in the right panel point to IVRT between aortic valve closure and mitral valve opening. IVRT was short at 48 msec consistent with increased LAP.

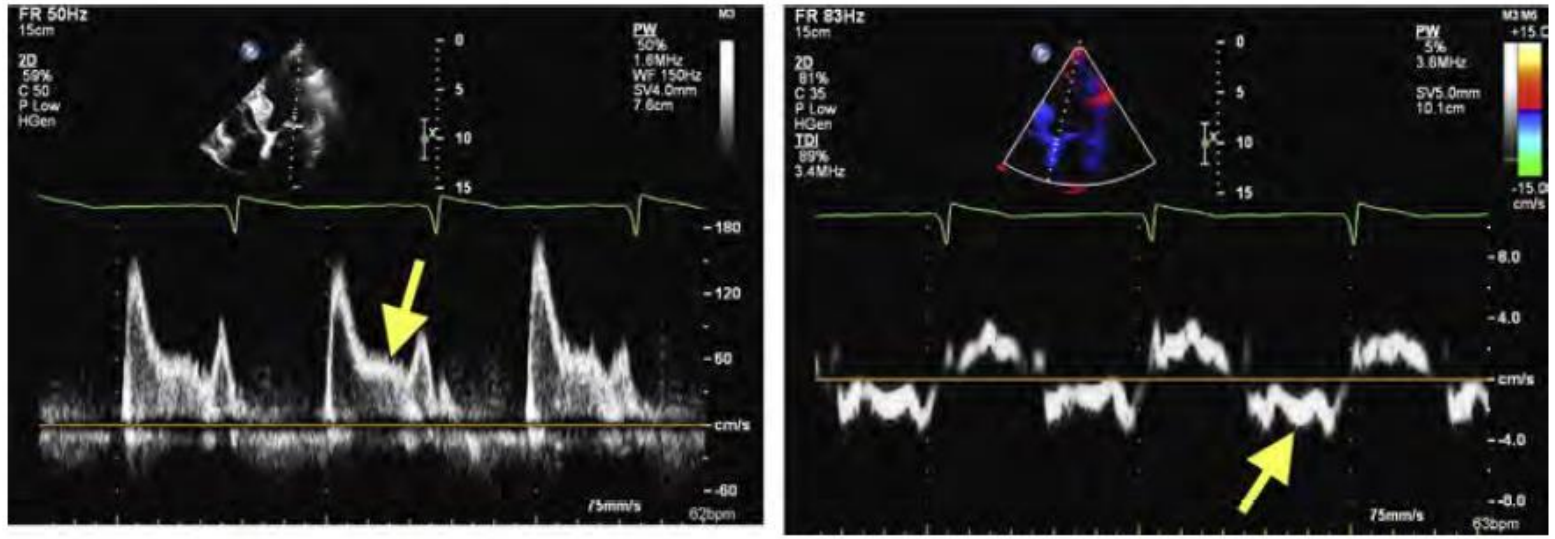


Figure 15 L velocity from a patient in sinus rhythm and increased LAP. Notice the presence of L velocity in mitral inflow and septal tissue Doppler signals (*arrows*).

Table 4 LV relaxation, filling pressures and 2D and Doppler findings according to LV diastolic function

	Normal	Grade I	Grade II	Grade III
LV relaxation	Normal	Impaired	Impaired	Impaired
LAP	Normal	Low or normal	Elevated	Elevated
Mitral E/A ratio	≥0.8	≤0.8	>0.8 to <2	>2
Average E/e′ ratio	<10	<10	10–14	>14
Peak TR velocity (m/sec)	<2.8	<2.8	>2.8	>2.8
LA volume index	Normal	Normal or increased	Increased	Increased

Following patients SHOULD have transmitral flow or TDI for the purpose of diastolic function assessment, despite noted limitations:

1. Tachycardia (difficult interpretation or impossible)
2. Atrial and atrioventricular pacing (changing PR interval or A-V delay may make interpretation difficult)
3. Atrial fibrillation or atrial flutter (No A wave in mitral Doppler inflow and no A' in TDI, interpretation of the pulmonary venous flow during arrhythmia is difficult). Note: E/ e' ratio still can be used for assessment of LV filling pressure

Following patients SHOULD NOT have transmitral flow or TDI for the purpose of diastolic function assessment:

1. Mitral stenosis
2. S/P Mitral annuloplasty ring (? Severe MAC)
3. S/P Mitral valve replacement
4. LVAD
5. Ventricular pacing

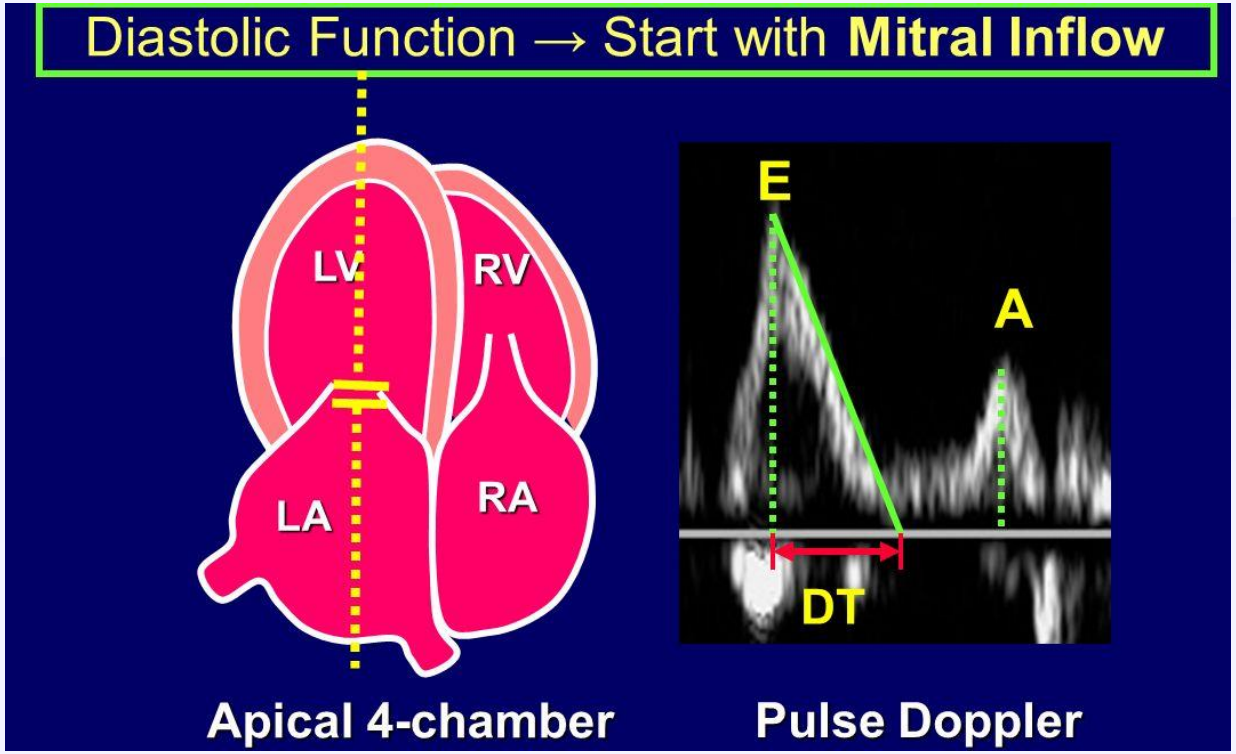
Table 6 Assessment of LV filling pressures in special populations

Disease	Echocardiographic measurements and cutoff values
AF[43,94-99]	Peak acceleration rate of mitral E velocity (≥1,900 cm/sec^2) IVRT (≤65 msec) DT of pulmonary venous diastolic velocity (≤220 msec) E/Vp ratio (≥1.4) Septal E/e′ ratio (≥11)
Sinus tachycardia[41,44]	Mitral inflow pattern with predominant early LV filling in patients with EFs <50% IVRT ≤70 msec is specific (79%) Pulmonary vein systolic filling fraction ≤40% is specific (88%) Average E/e′ >14 (this cutoff has highest specificity but low sensitivity) When E and A velocities are partially or completely fused, the presence of a compensatory period after premature beats often leads to separation of E and A velocities which can be used for assessment of diastolic function
HCM[100-106]	Average E/e′ (>14) Ar-A (≥30 msec) TR peak velocity (>2.8 m/sec) LA volume (>34 mL/m^2).

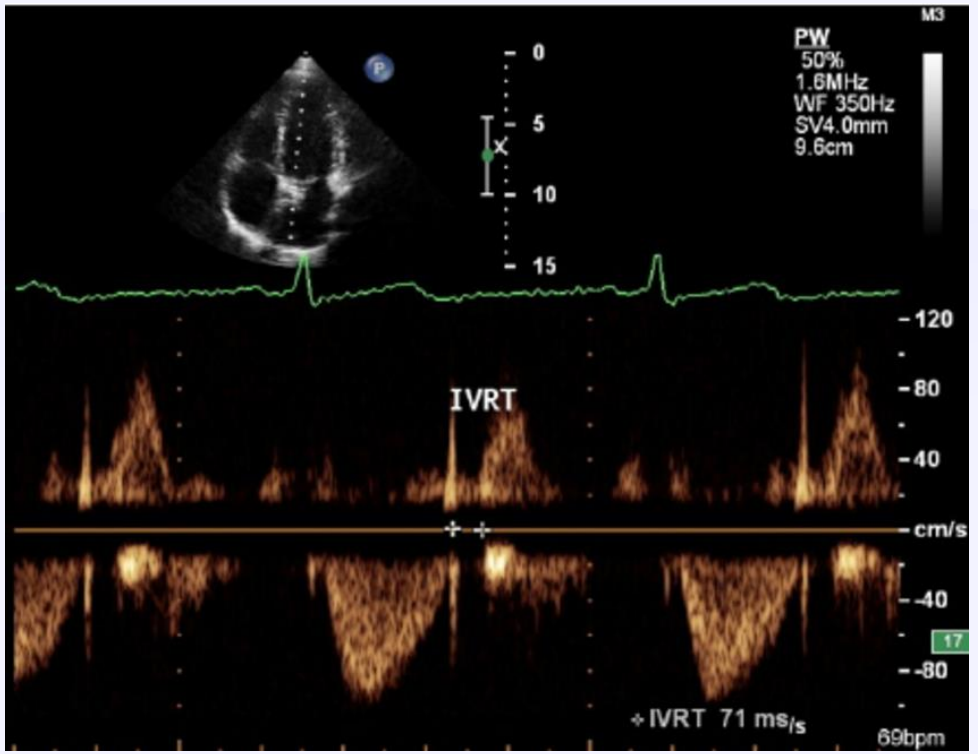
Table 6 Assessment of LV filling pressures in special populations **(Continued)**

Disease	Echocardiographic measurements and cutoff values
Restrictive cardiomyopathy[13,107-109]	DT (<140 msec) Mitral E/A (>2.5) IVRT (<50 msec has high specificity) Average E/e′ (>14)
Noncardiac pulmonary hypertension[32]	Lateral E/e′ can be applied to determine whether a cardiac etiology is the underlying reason for the increased pulmonary artery pressures When cardiac etiology is present, lateral E/e′ is >13, whereas in patients with pulmonary hypertension due to a noncardiac etiology, lateral E/e′ is <8
Mitral stenosis[110]	IVRT (<60 msec has high specificity) IVRT/$T_{E\text{-}e'}$ (<4.2) Mitral A velocity (>1.5 m/sec)
MR[110-112]	Ar-A (≥30 msec) IVRT (<60 msec has high specificity) IVRT/$T_{E\text{-}e'}$ (<5.6) may be applied for the prediction of LV filling pressures in patients with MR and normal EFs Average E/e′ (>14) may be considered only in patients with depressed EFs

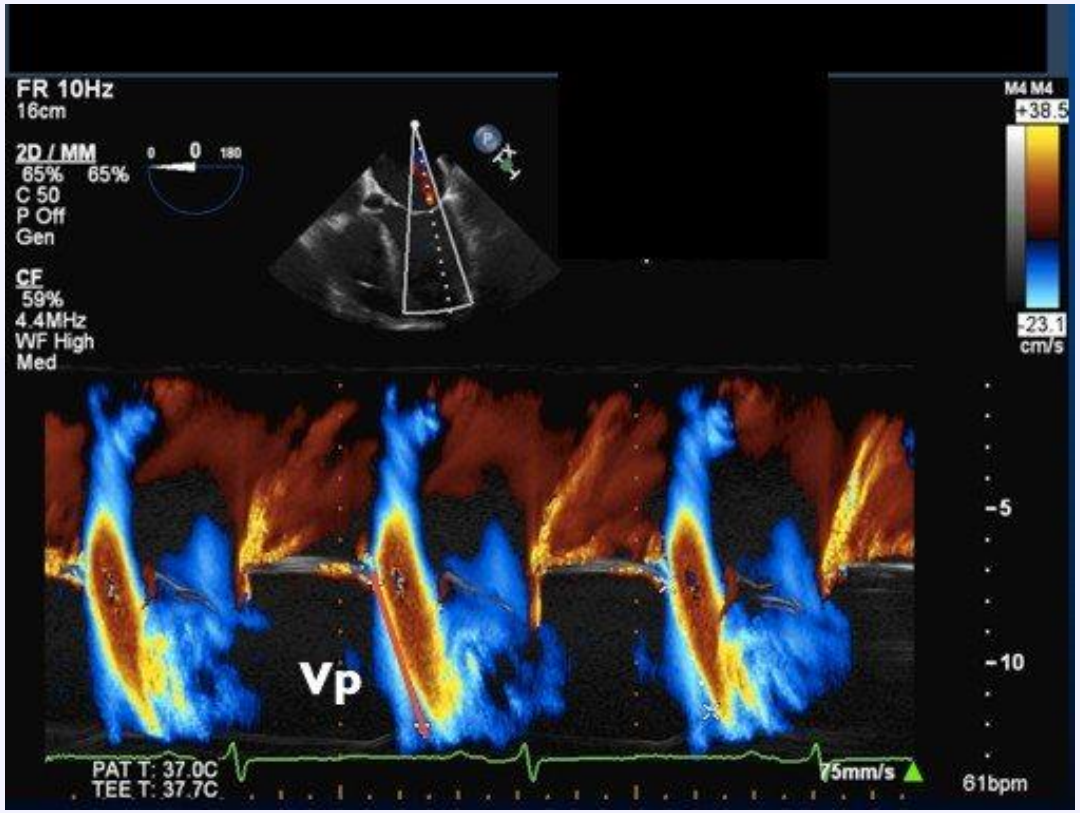
A comprehensive approach is recommended in all of the above settings, which includes estimation of PASP using peak velocity of TR jet (>2.8 m/sec) and LA maximum volume index (>34 mL/m^2). Conclusions should not be based on single measurements. Specificity comments refer to predicting filling pressures > 15 mm Hg. Note that the role of LA maximum volume index to draw inferences on LAP is limited in athletes, patients with AF, and/or those with mitral valve disease.



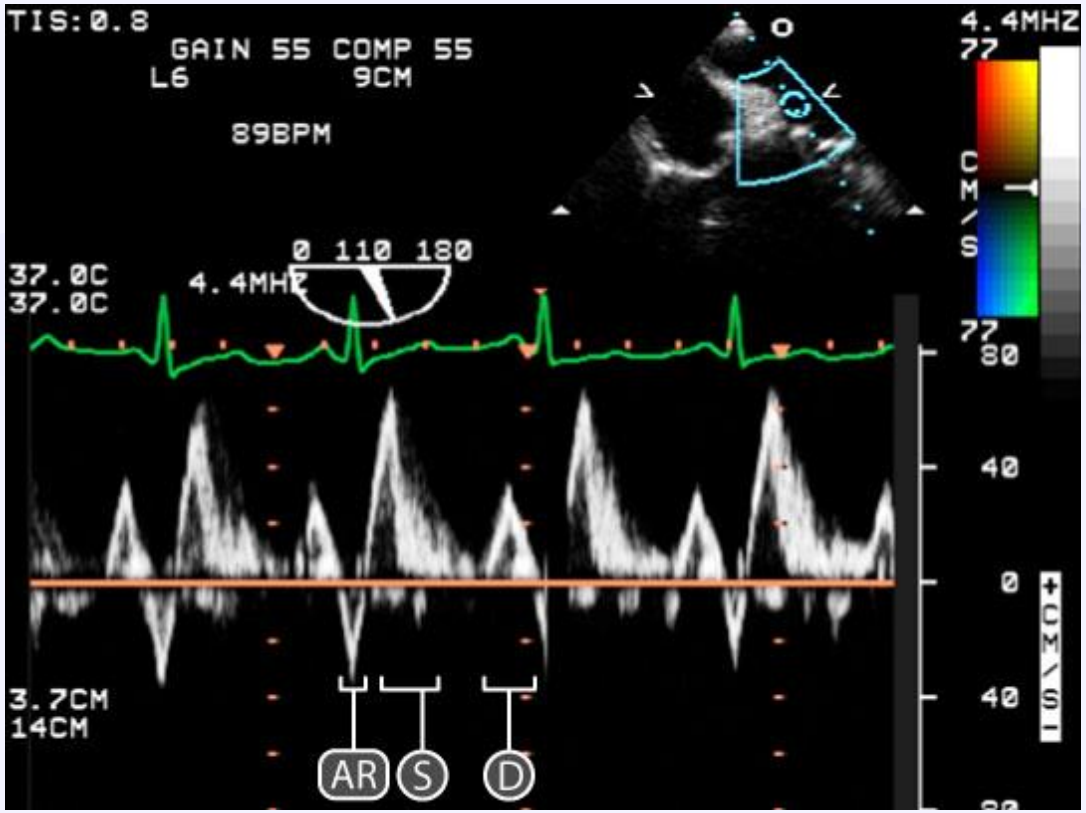
Deceleration time (DT)



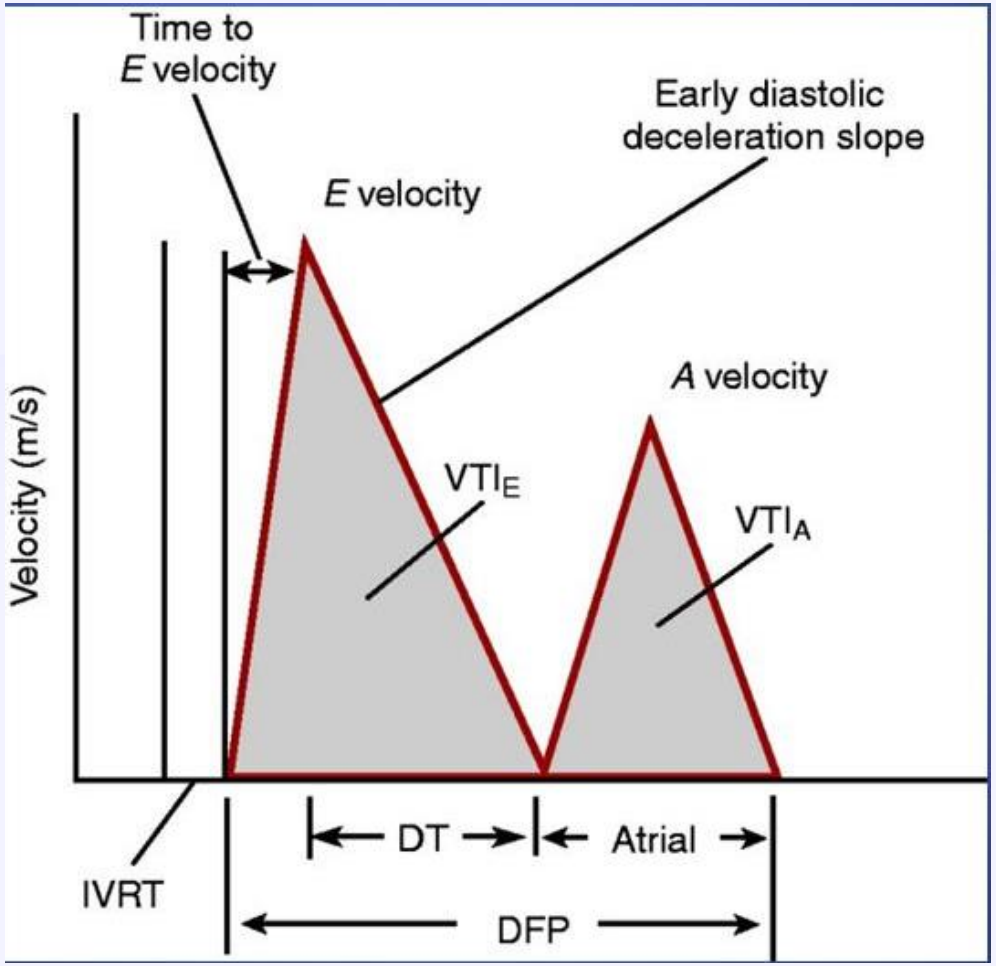
Isovolumic relaxation time (IVRT)

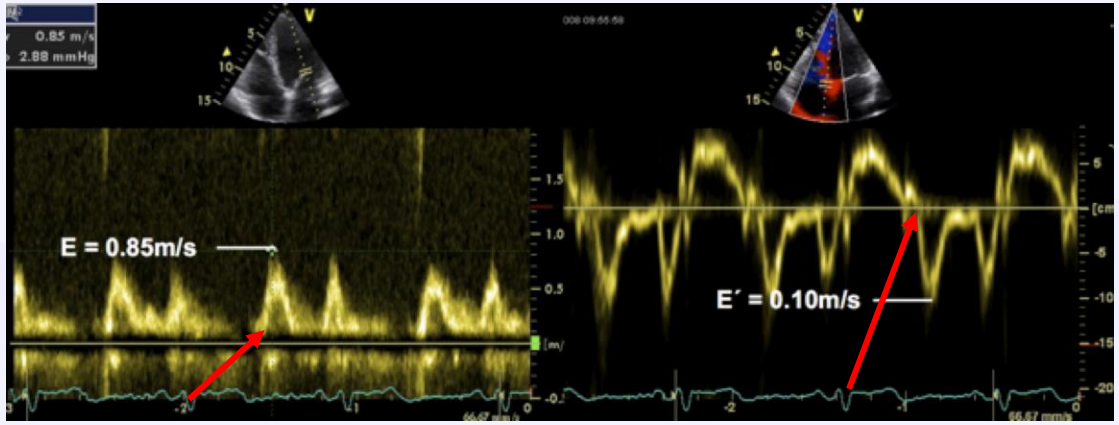


Colour flow velocity propagation time (Vp)
Normal > 45 cm/s



Pulmonary A wave reversal time

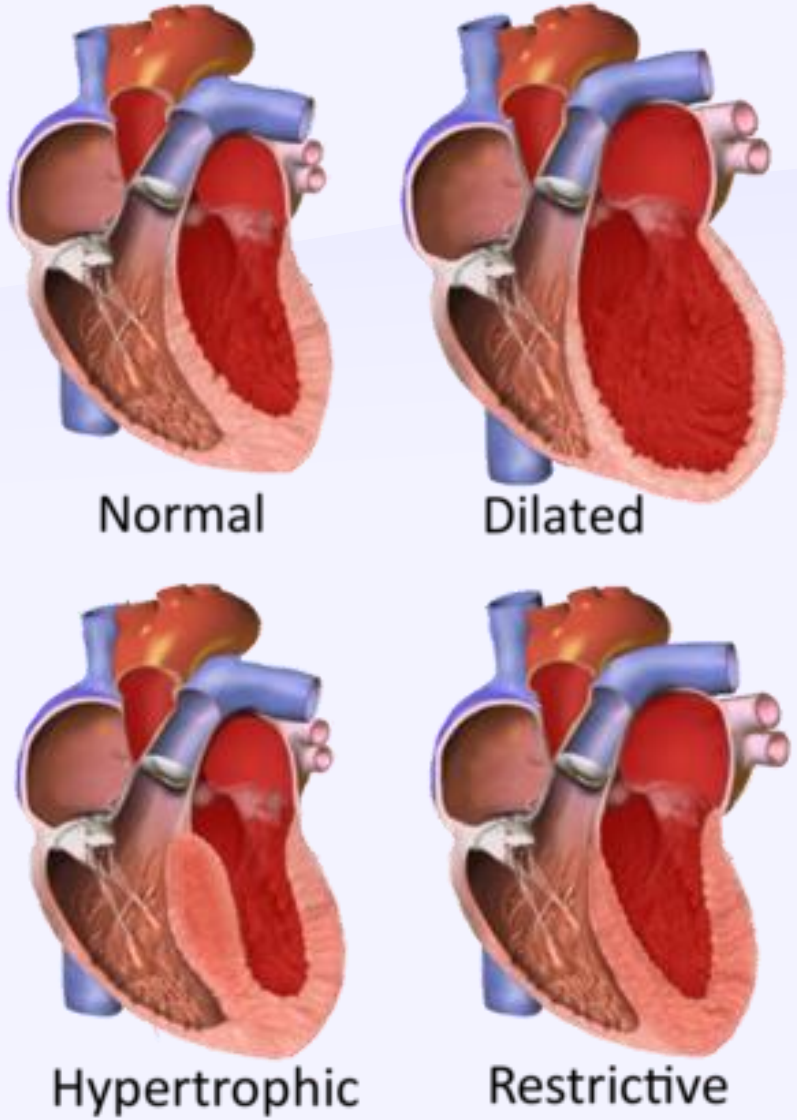




T (E- e') = Time from peak R wave of ECG to the beginning of the E wave in mitral inflow – time from peak R wave of ECG to the e' in TDI of mitral annulus. In severe diastolic dysfunction e' has significant delay compared to E wave.

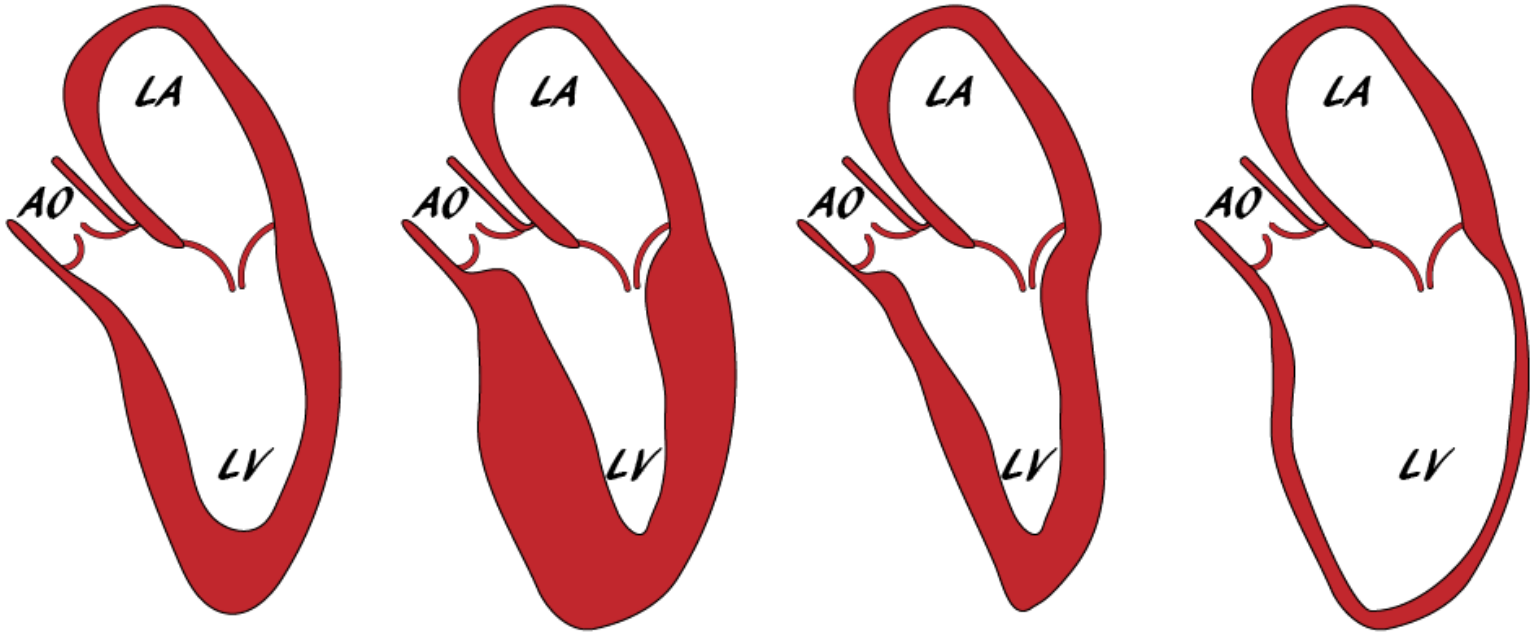
E wave acceleration rate (time to peak E velocity) > 1900 cm/s2 c/w severe diastolic dysfunction

Cardiomyopathies


Normal
Dilated
Hypertrophic
Restrictive

Cardiomyopathy

Apical, Long Axis, Three Chamber View



Normal Heart

Hypertrophic Cardiomyopathy

Restrictive Cardiomyopathy

Dilated Cardiomyopathy

WHO definition of cardiomyopathy:

"Disease of the myocardium associated with cardiac dysfunction"

World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies

- Dilated Cardiomyopahty
- Hypertrophic Cardiomyopathy
- Restrictive Cardiomyopahty
- Arrhythmogenic Right Ventricular Cardiomyopathy
- Unclassified Cardiomyopathies
  - Fibroelastosis
  - Noncompacted myocardium
  - Systolic dysfunction with minimal dilatation
  - Mitochondrial involvement
- Specific Cardiomyopahties
  - Ischemic cardiomyopathy
  - Valvular cardiomyopahty
  - Hypertensive cardiomyopathy
  - Inflammatory cardiomyopathy
  - Metabolic cardiomyopathy
  - General system disease
  - Muscular distrophies
  - Neuromuscular disorders
  - Sensitivity and toxic reactions
  - Peripartum cardiomyopathy

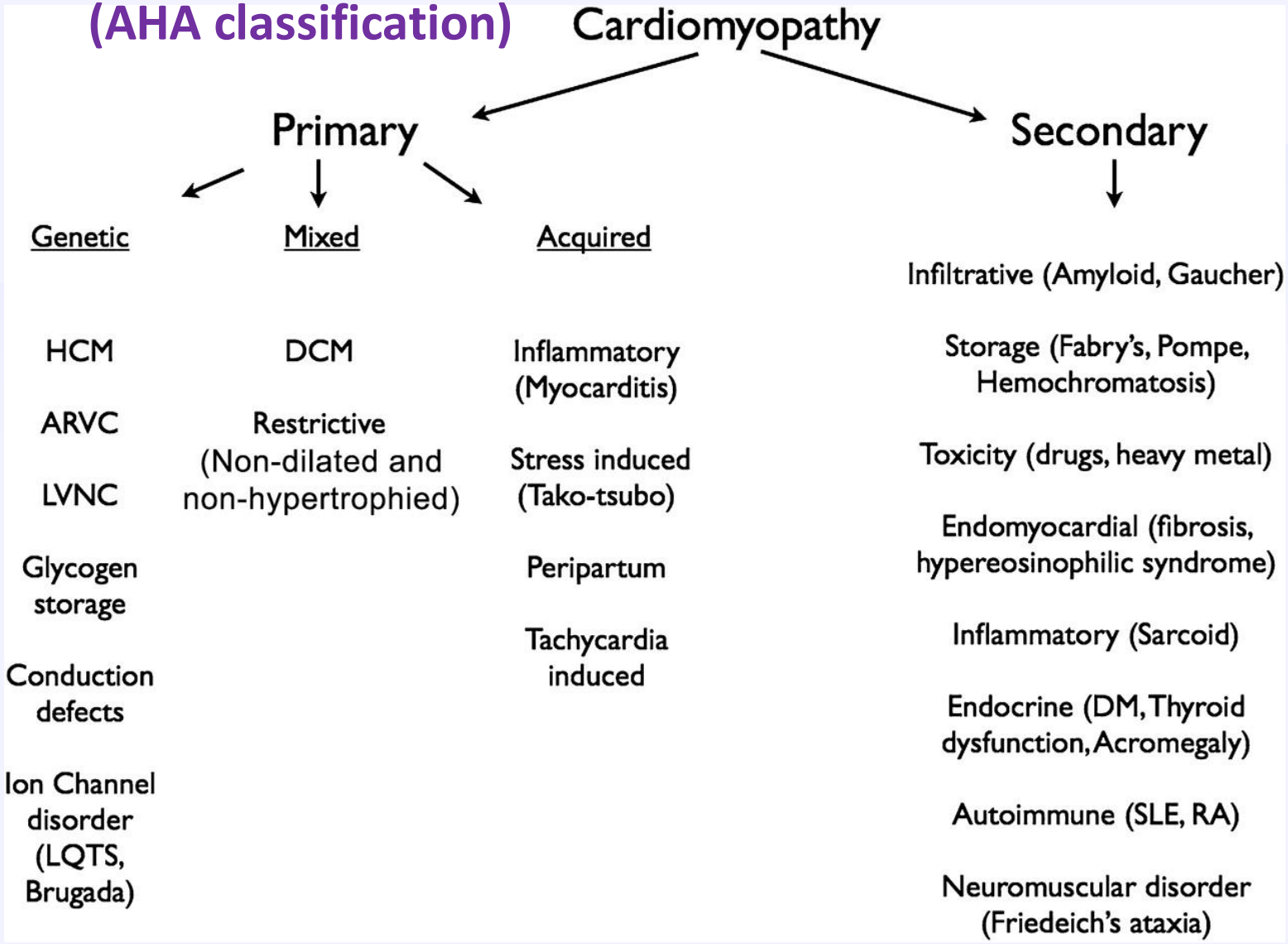
AHA Expert panel definition of cardiomyopathy:

- "A heterogeneous group of disease of the myocardium associated with mechanical and or / electrical dysfunction that usually (not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to variety of causes that frequently are genetic

- Cardiomyopathies are either confined to the heart or are part of generalized systemic disorders often leading to cardiovascular death or progressive heart failure- related disability".

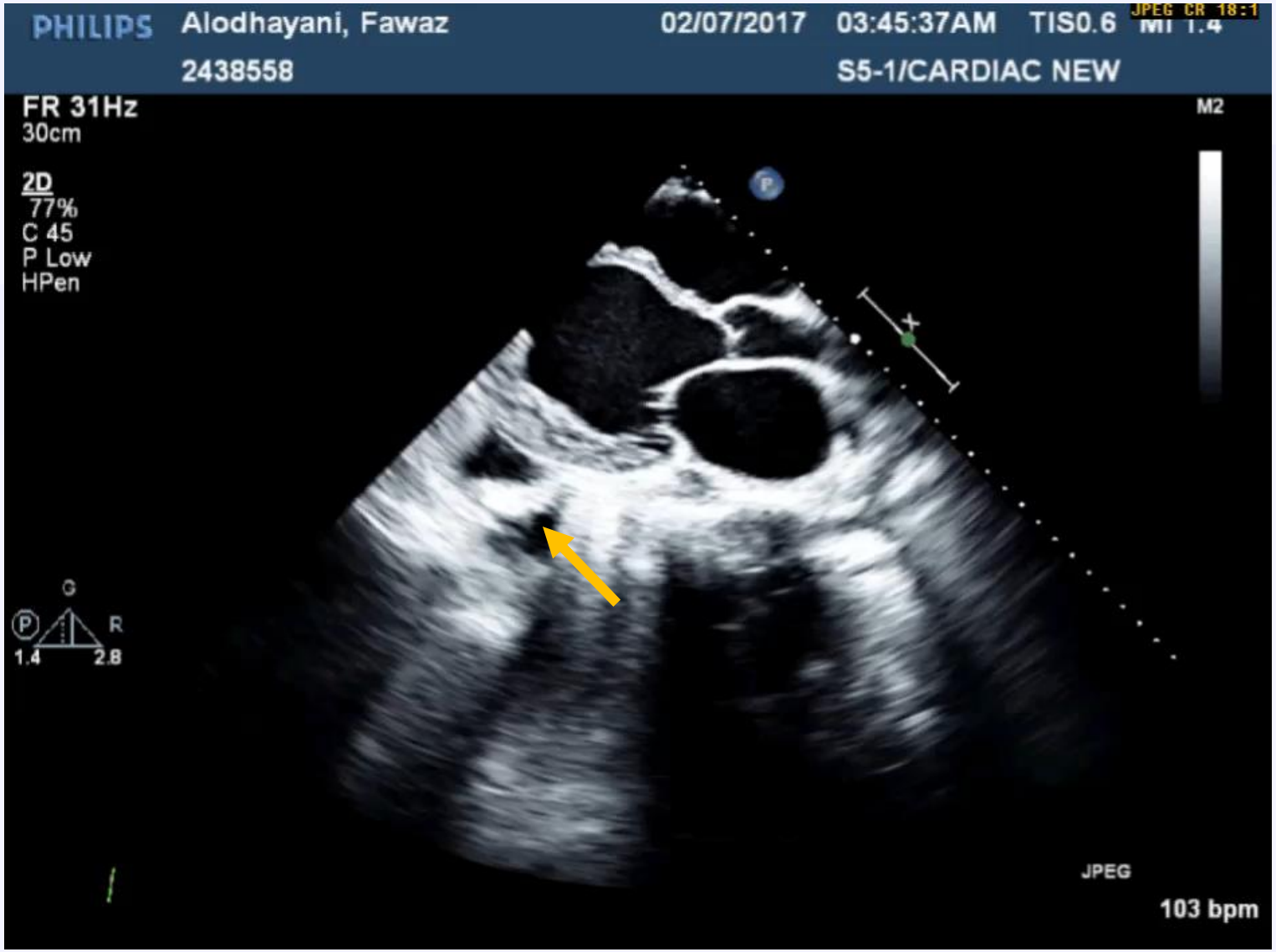
Maron BJ, et al. Circulation 2006

(AHA classification)



Case 1

- 29-year-old man presented with SOB. Echocardiography was requested to rule out cardiac tamponade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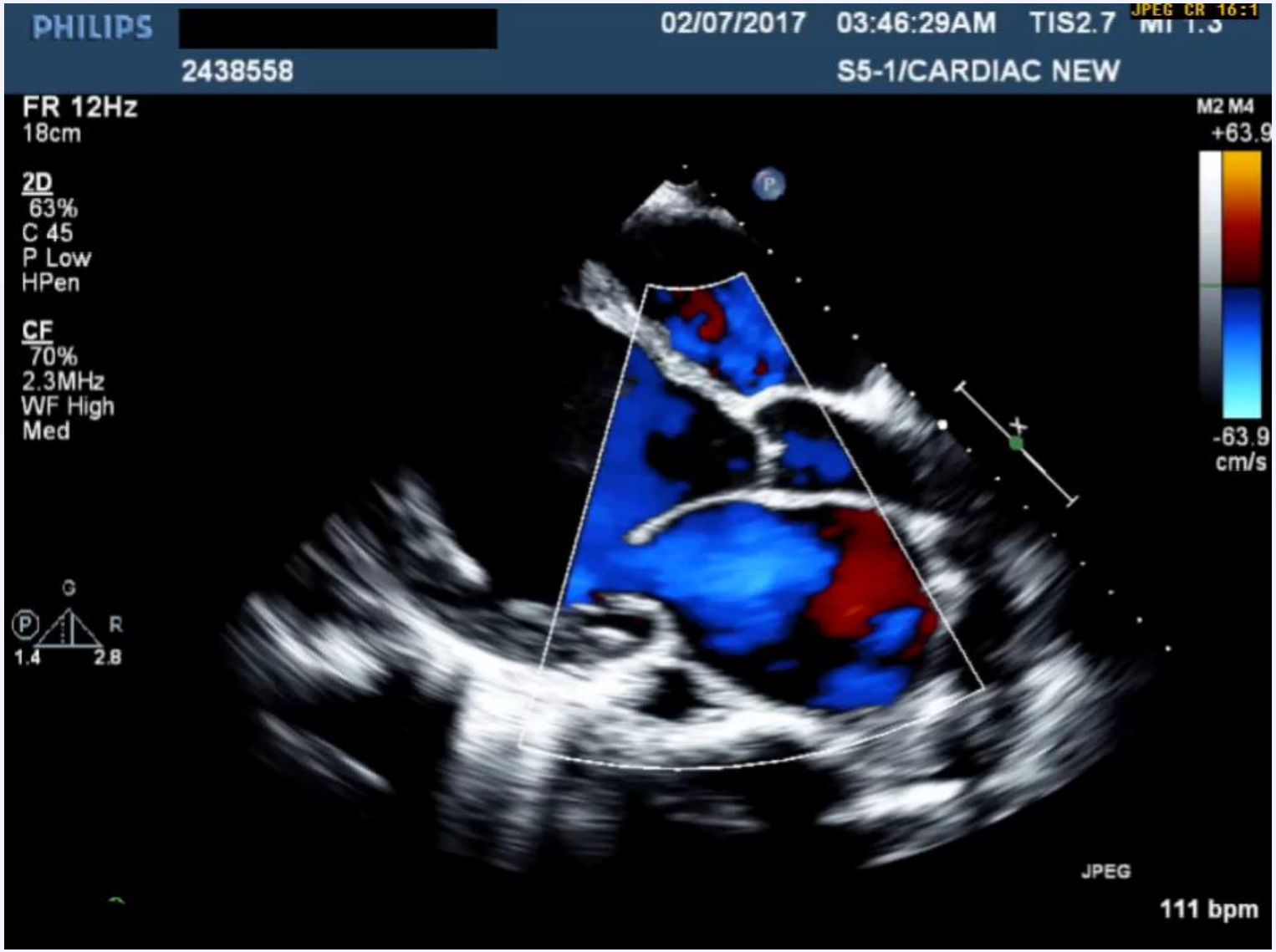
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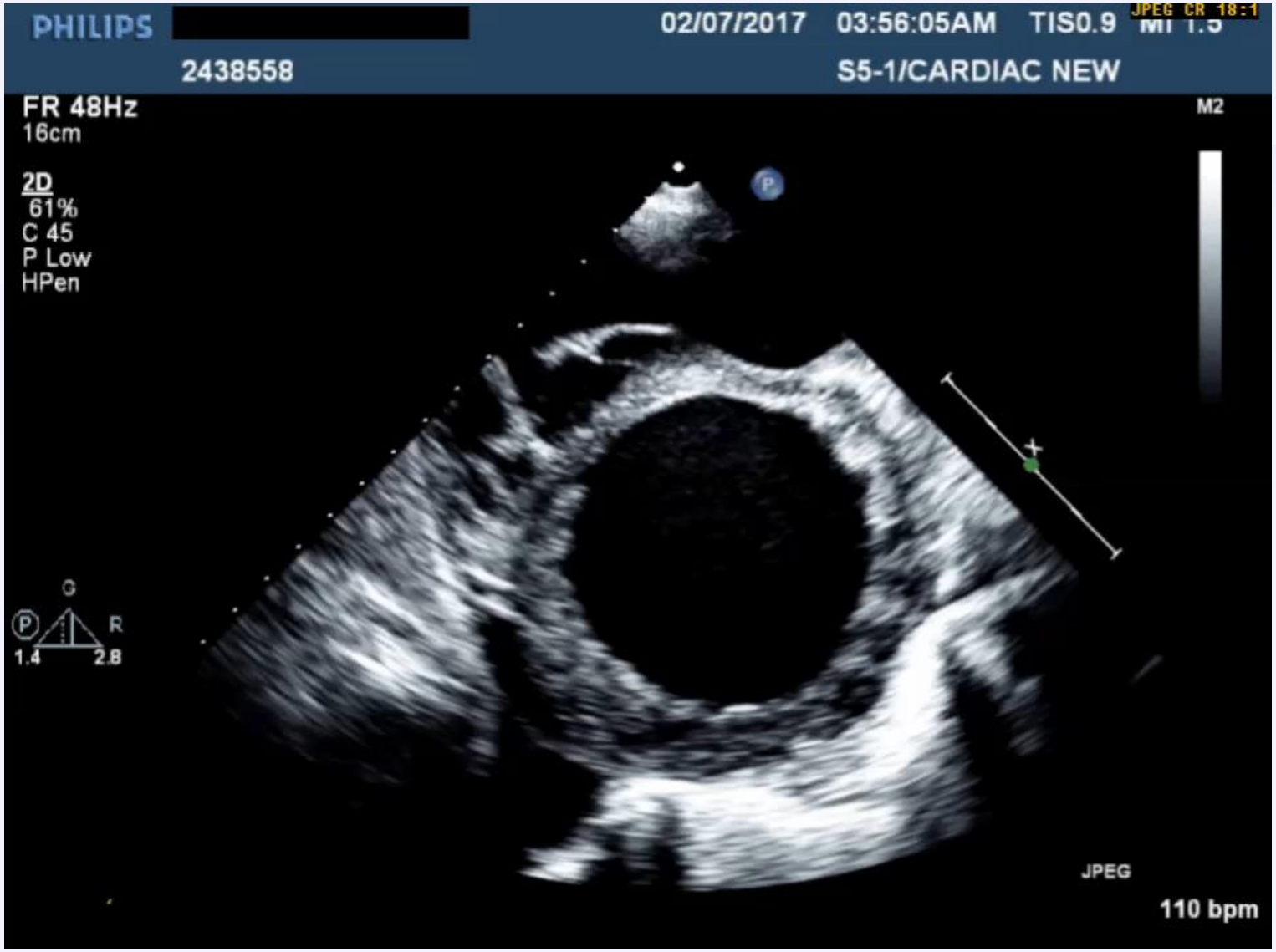
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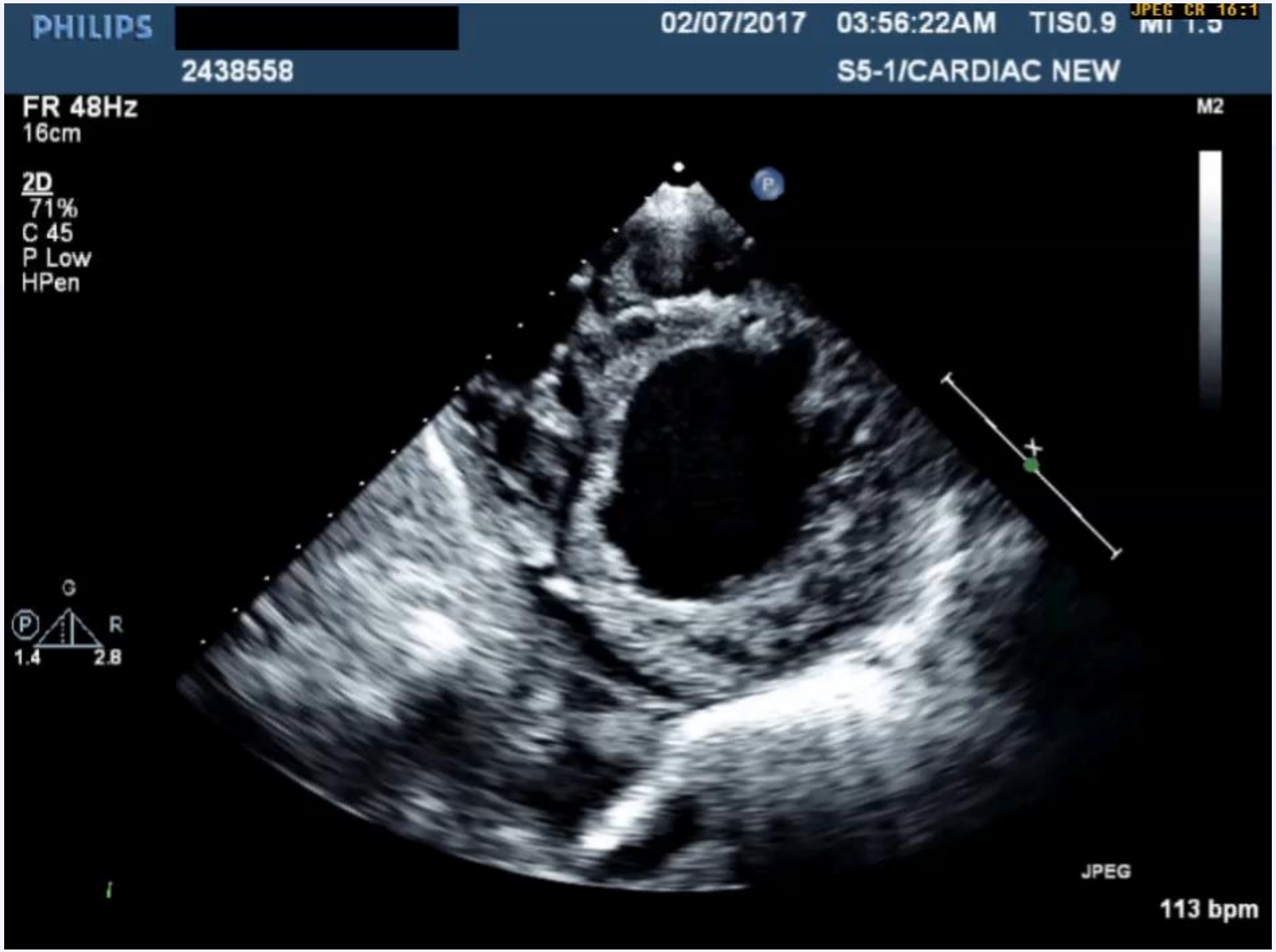
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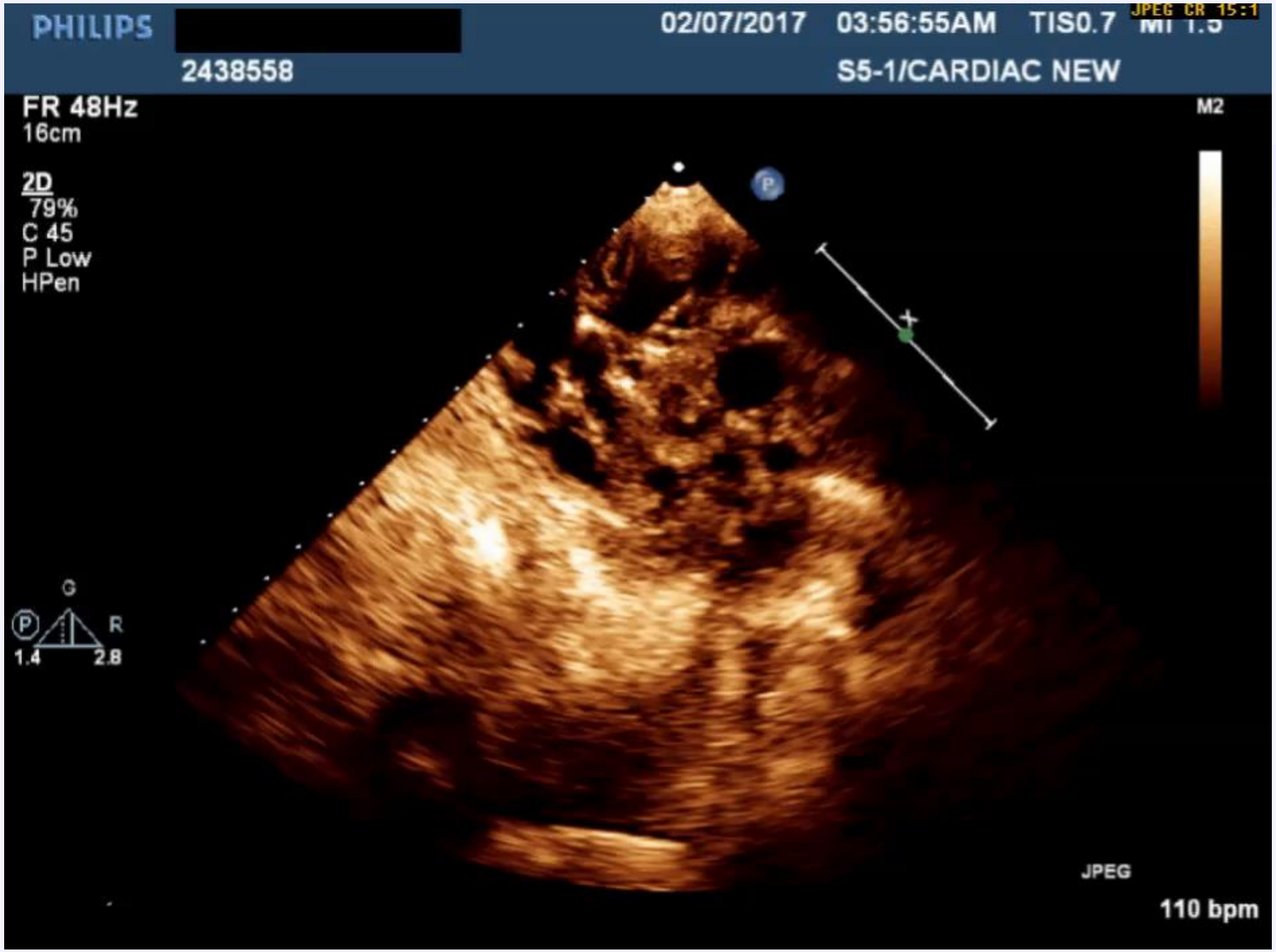
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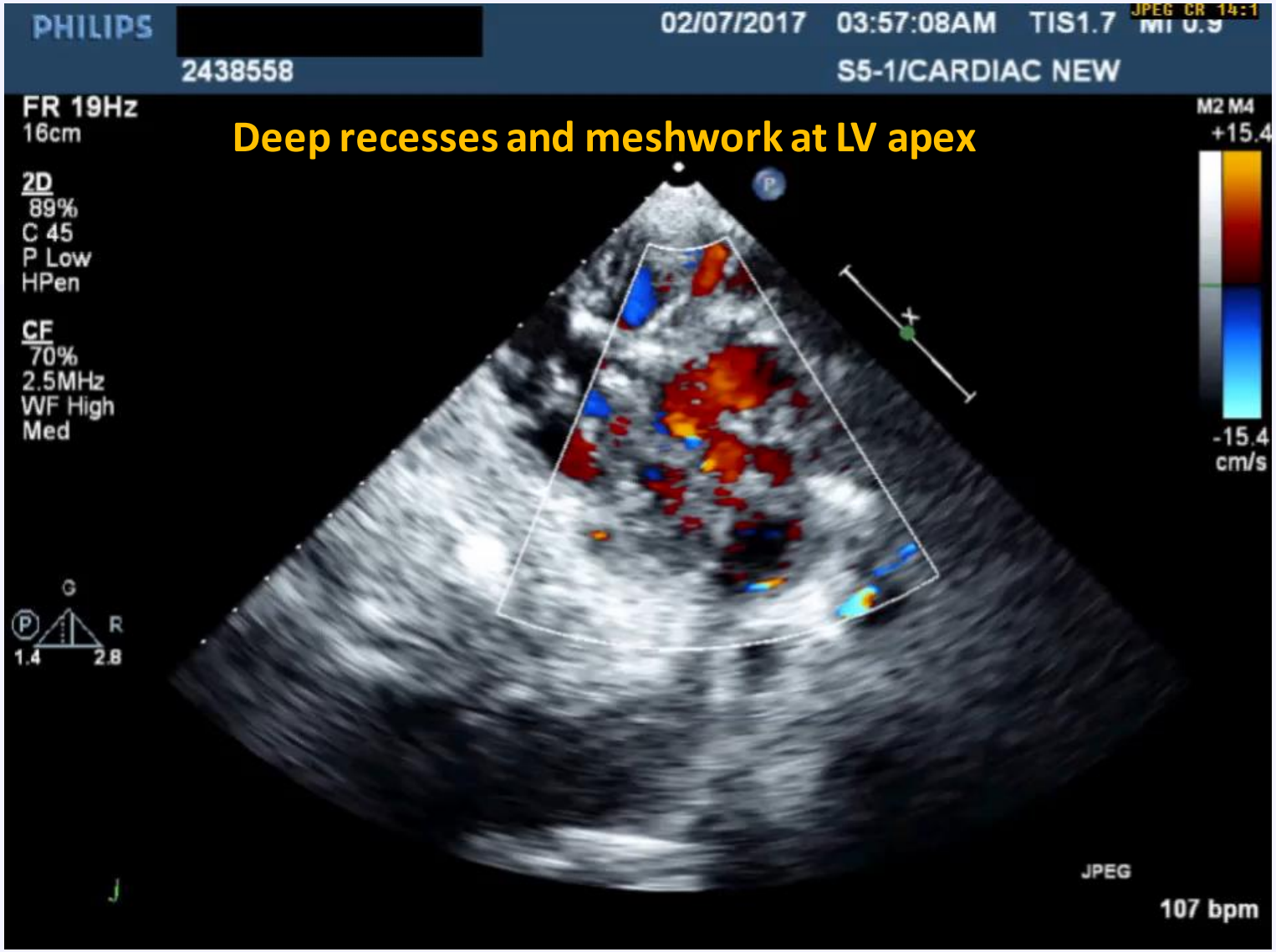
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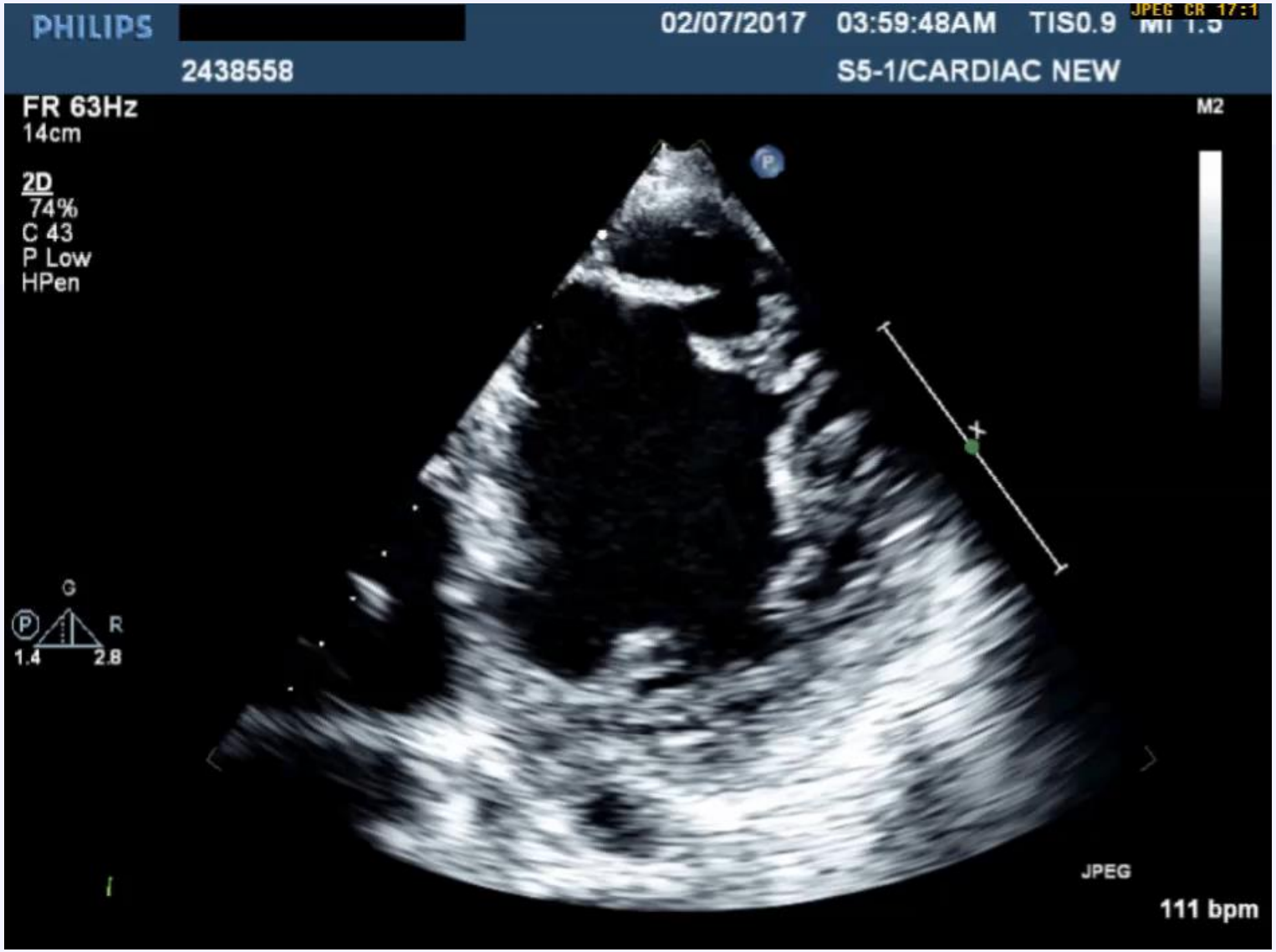
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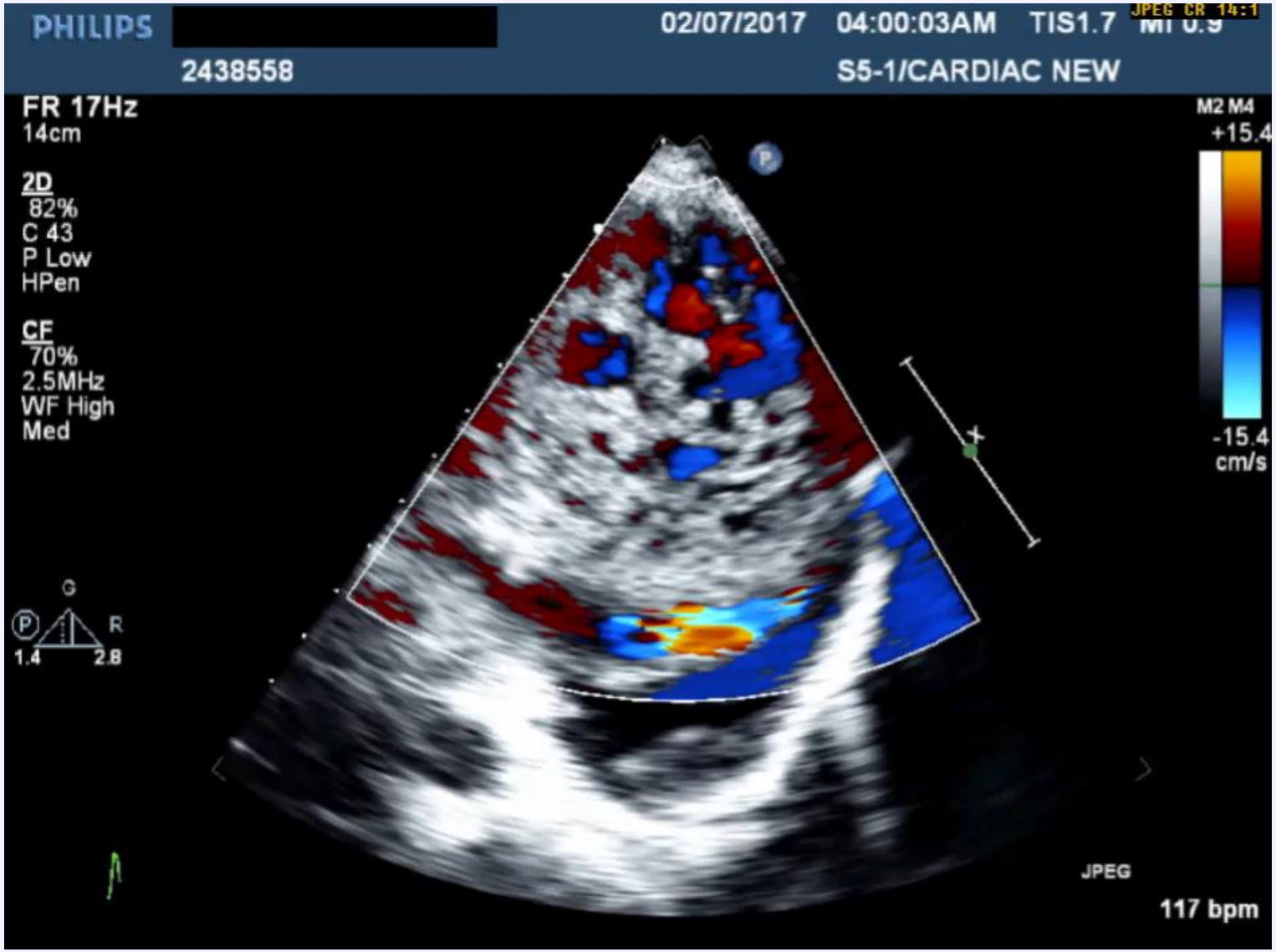
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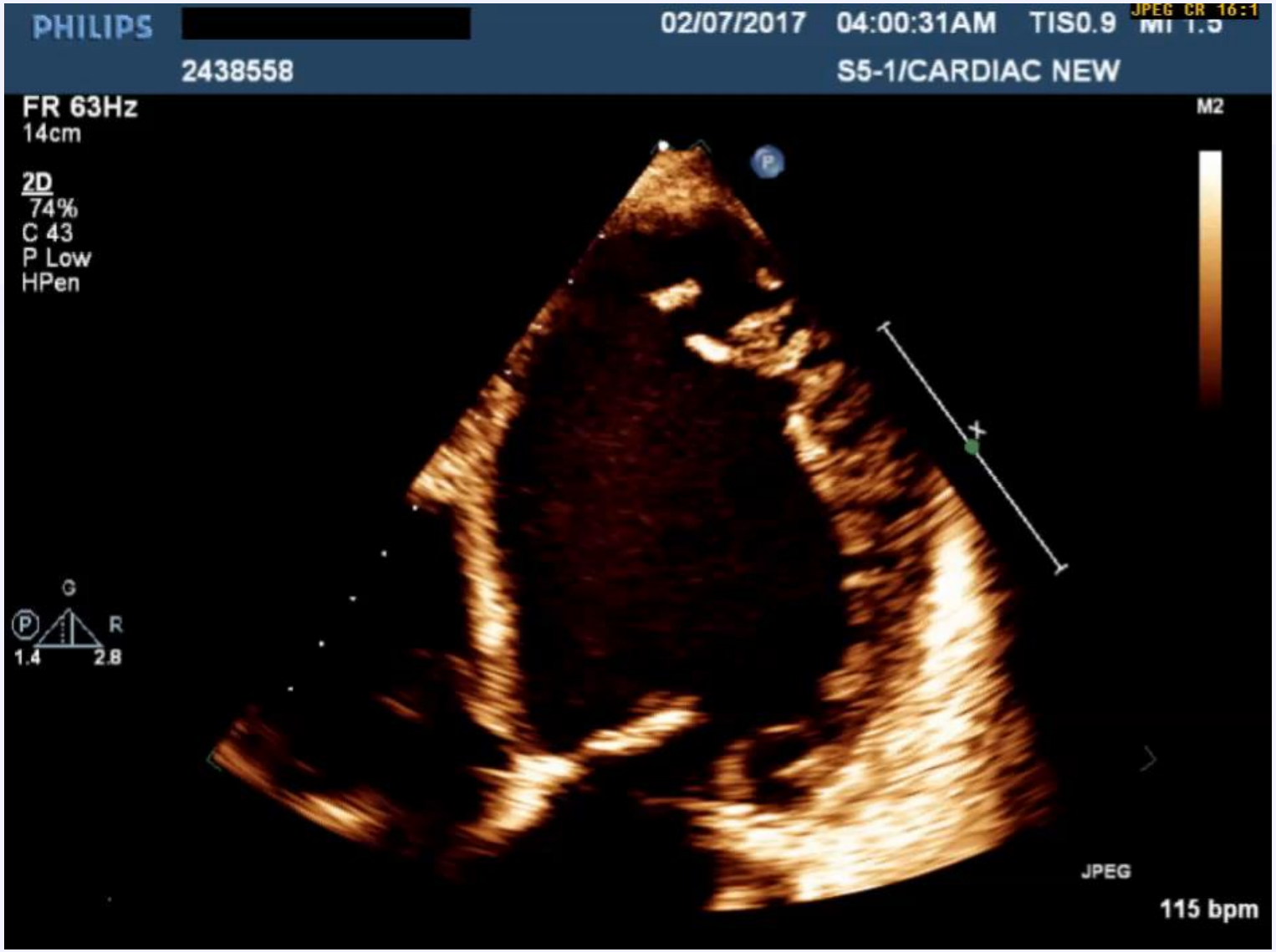
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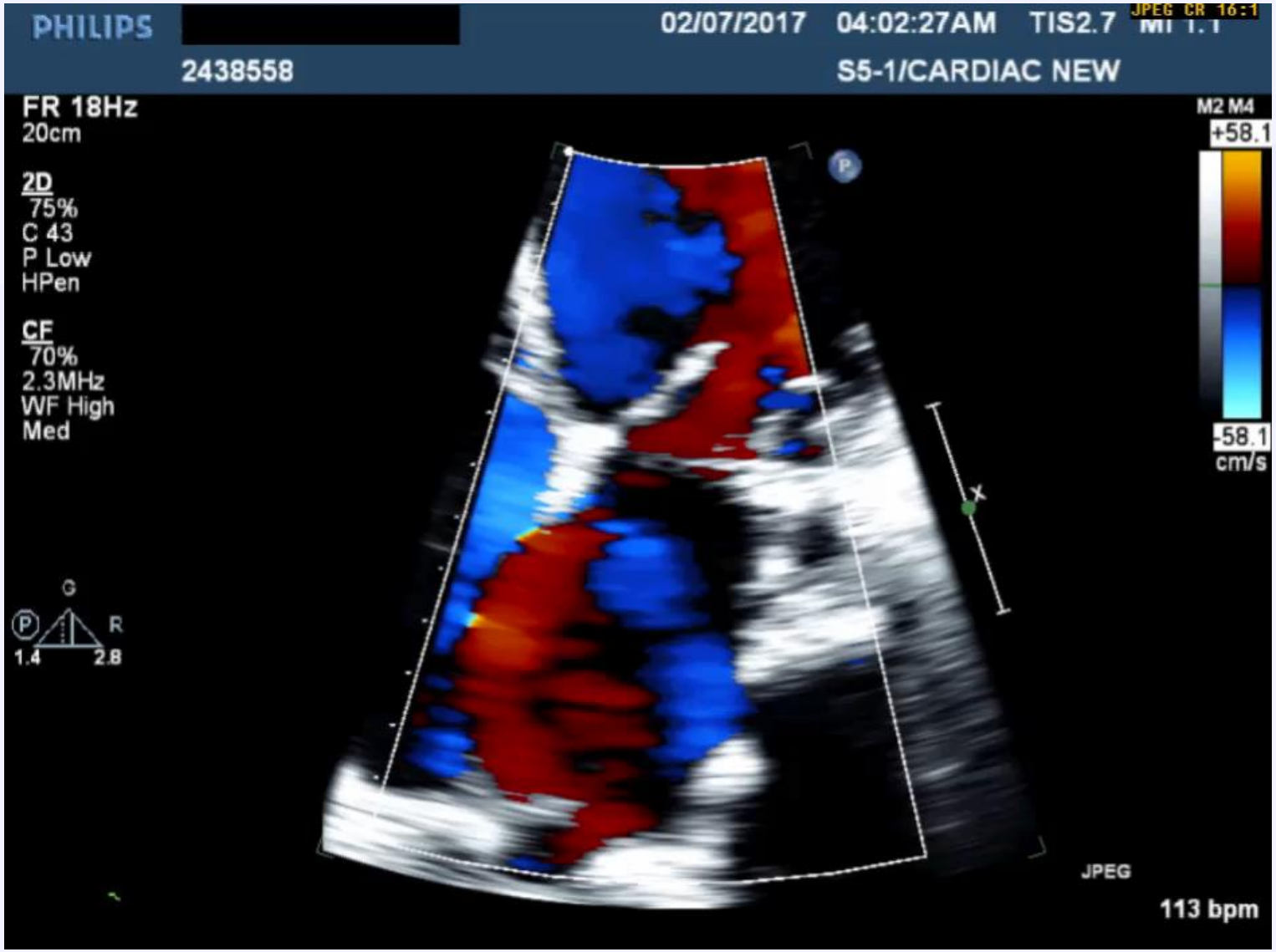
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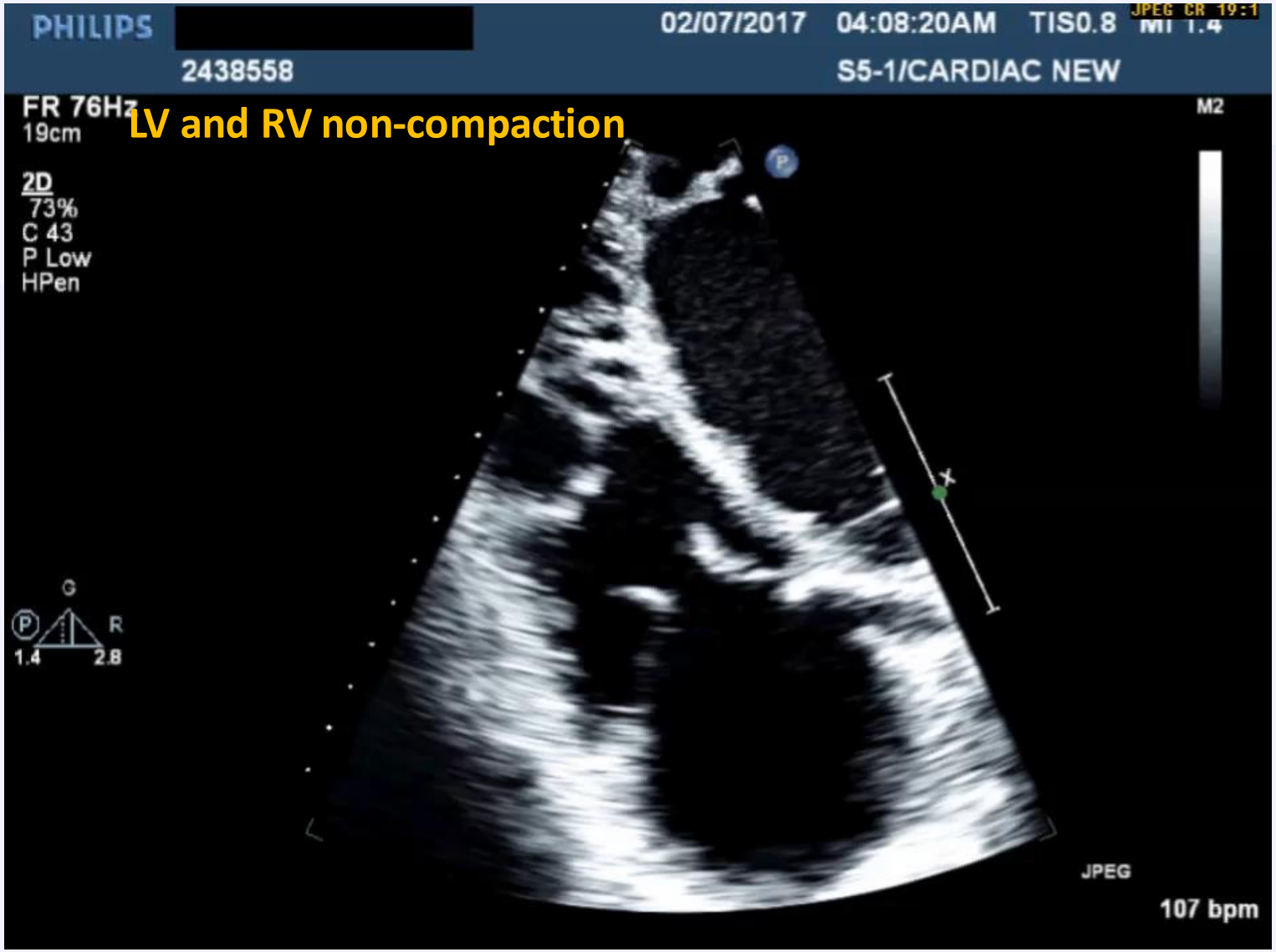
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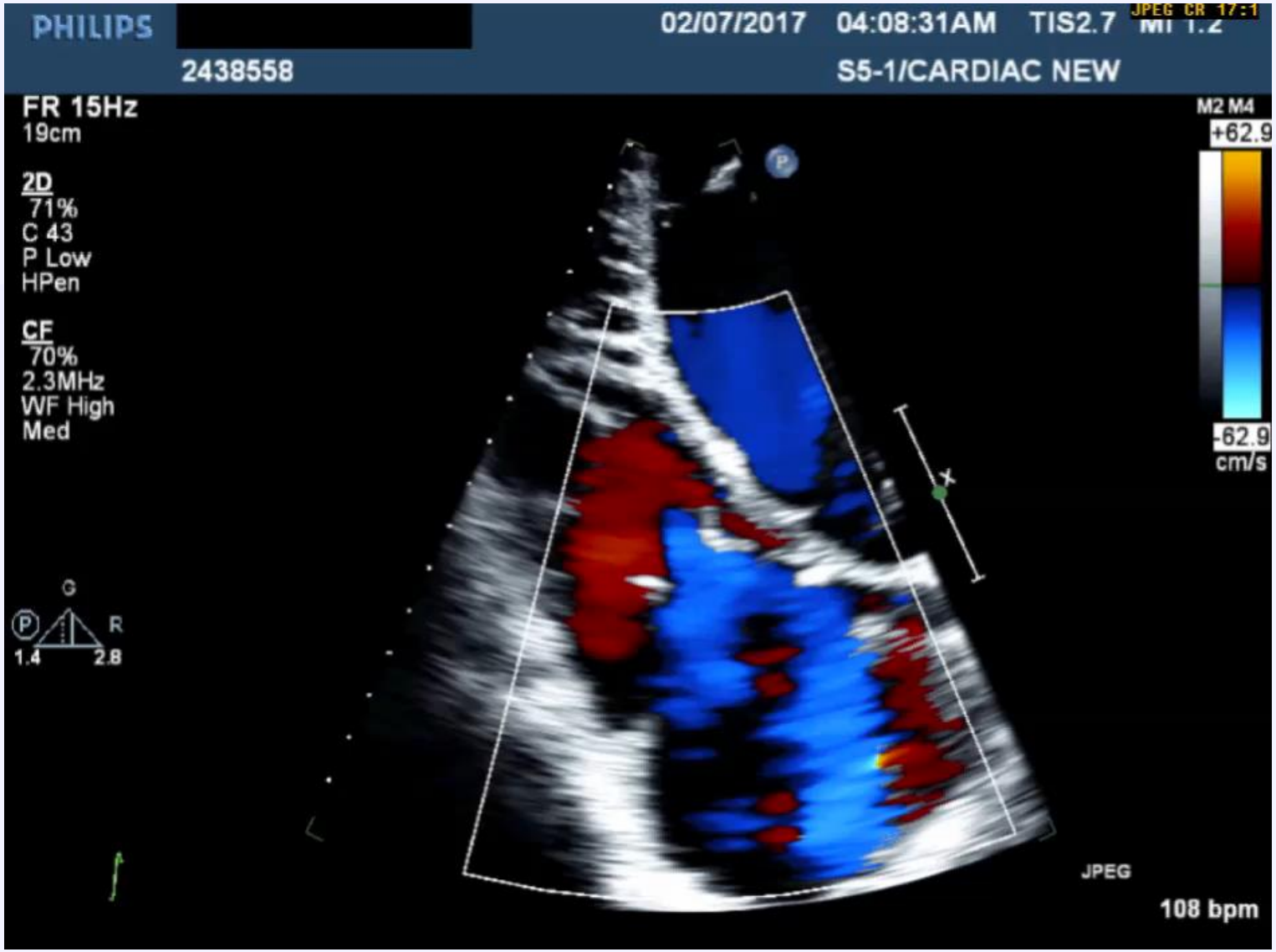
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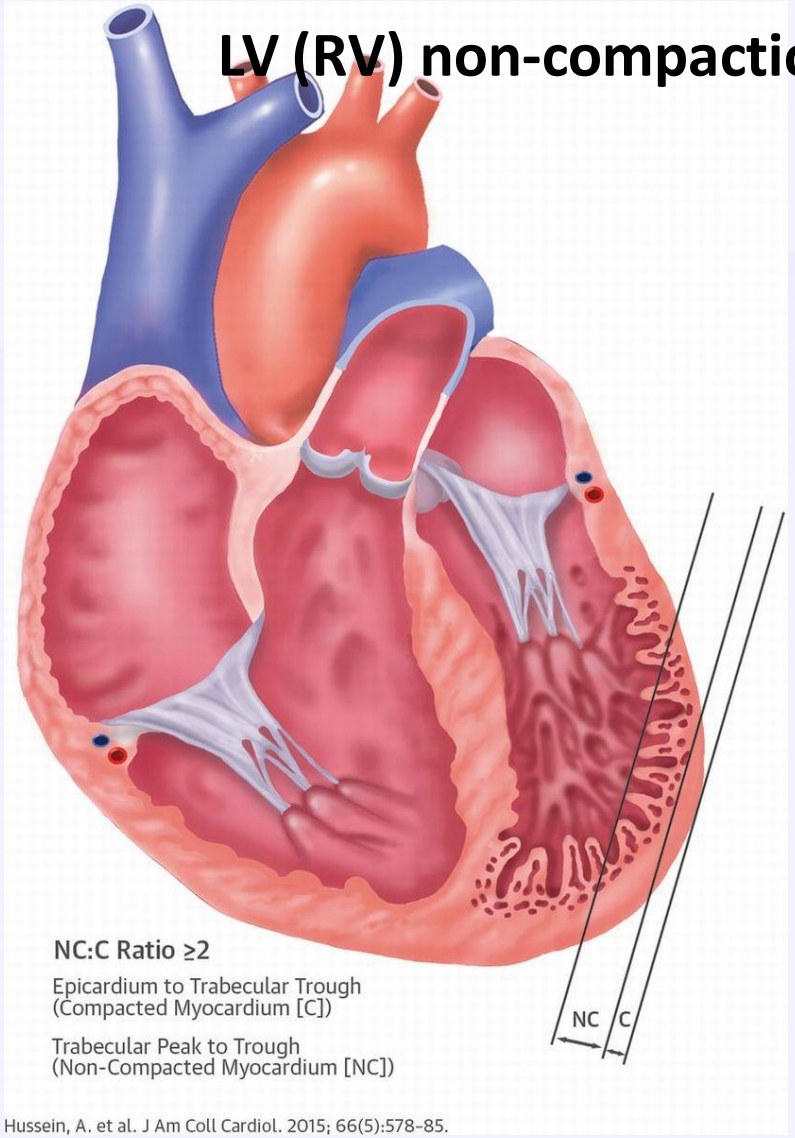
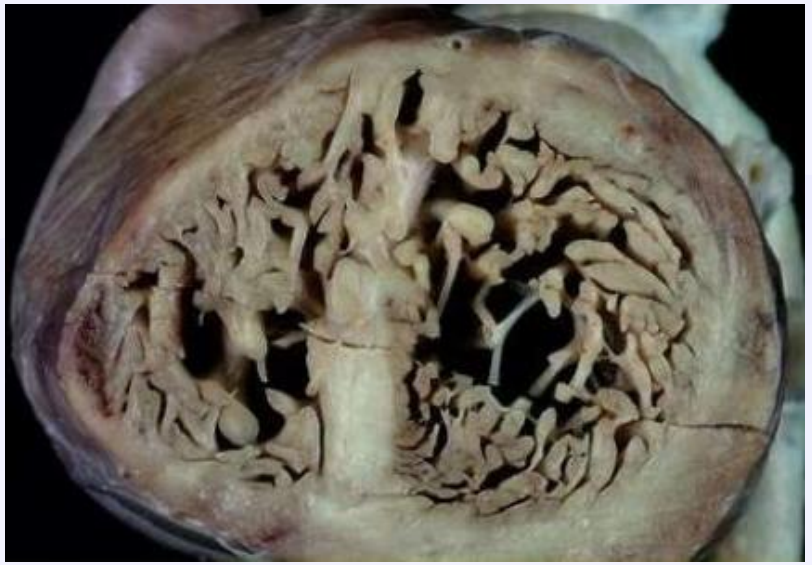
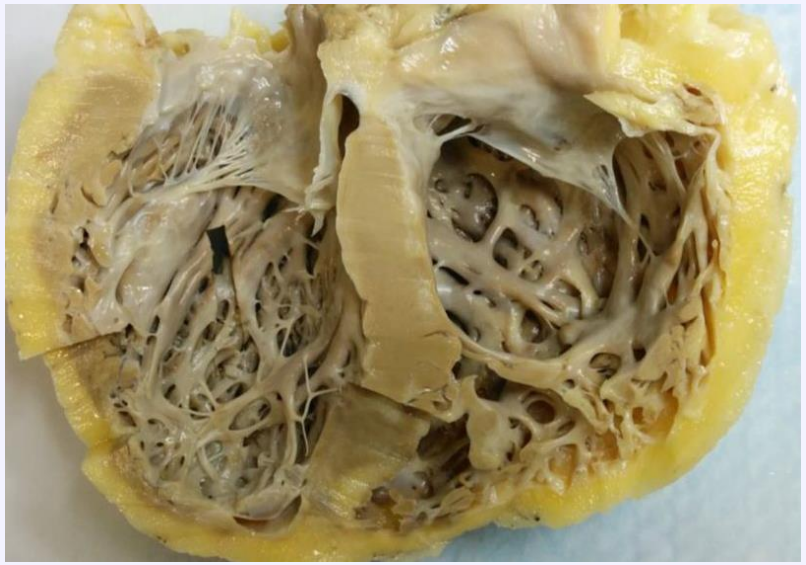
LV (RV) non-compaction



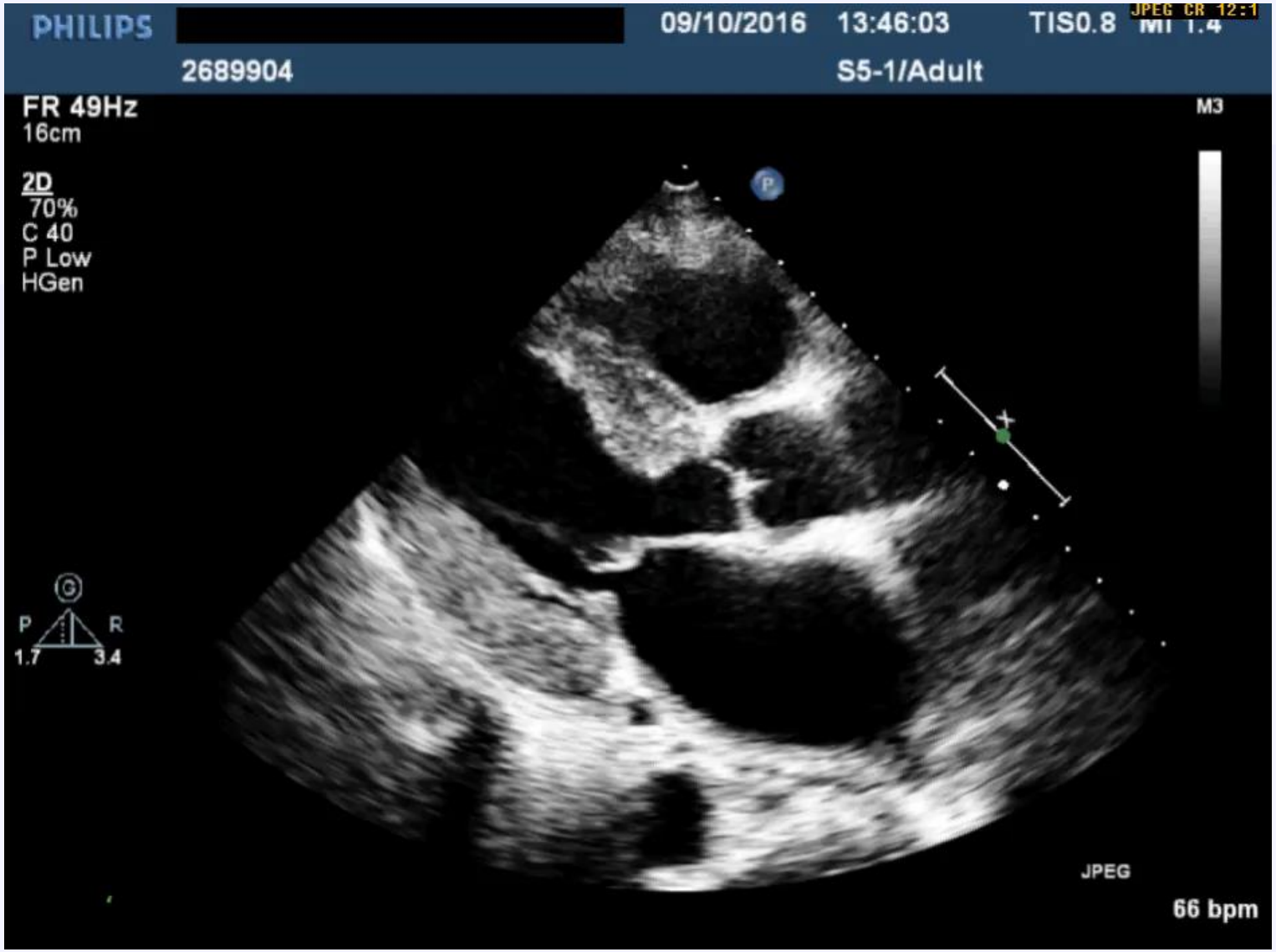
Hussein, A. et al. J Am Coll Cardiol. 2015; 66(5):578–85.





Case 2

- 82-year-old man presented to our center due to severe SOB and atrial fibrillation. No history of hypertension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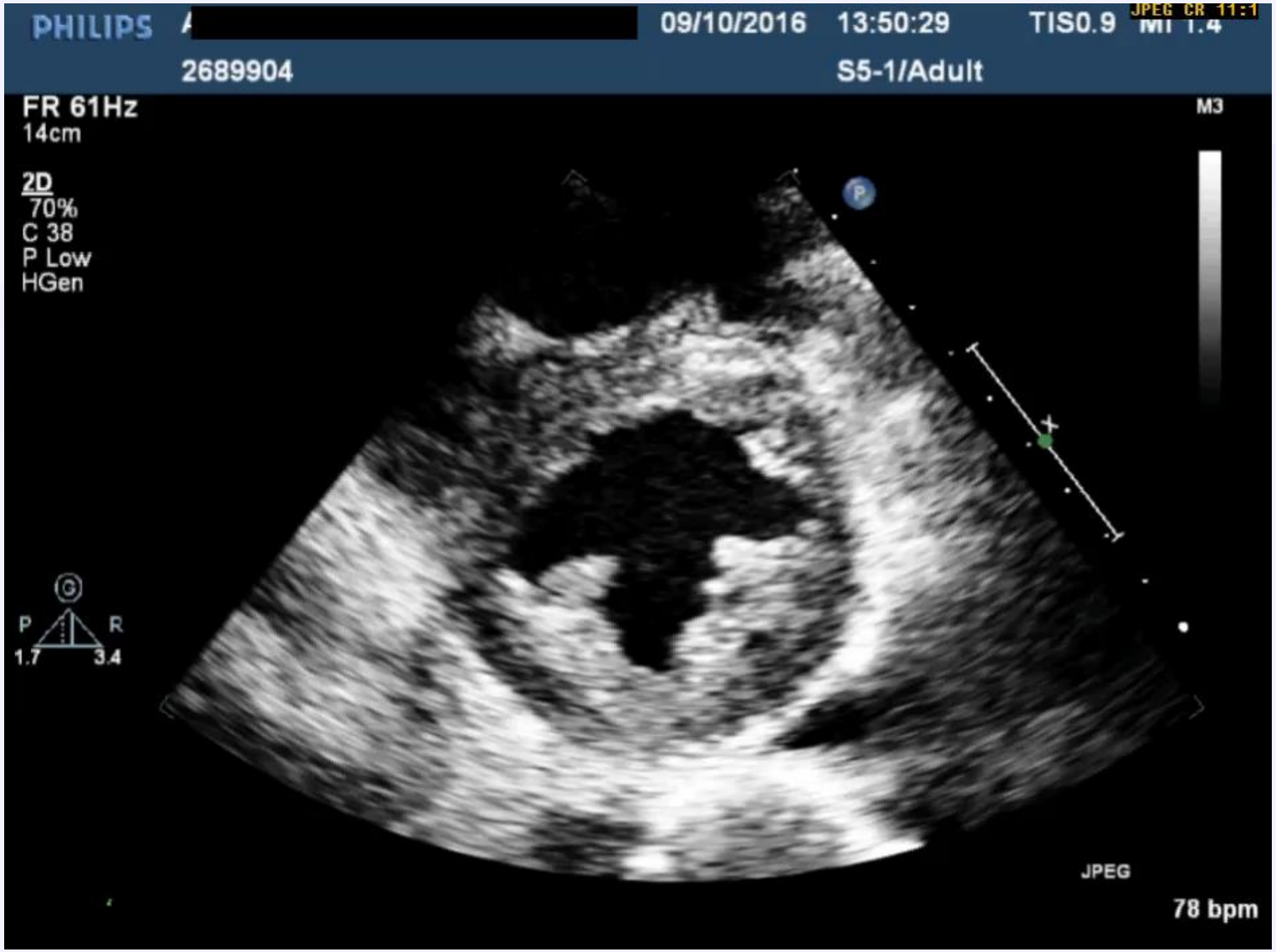
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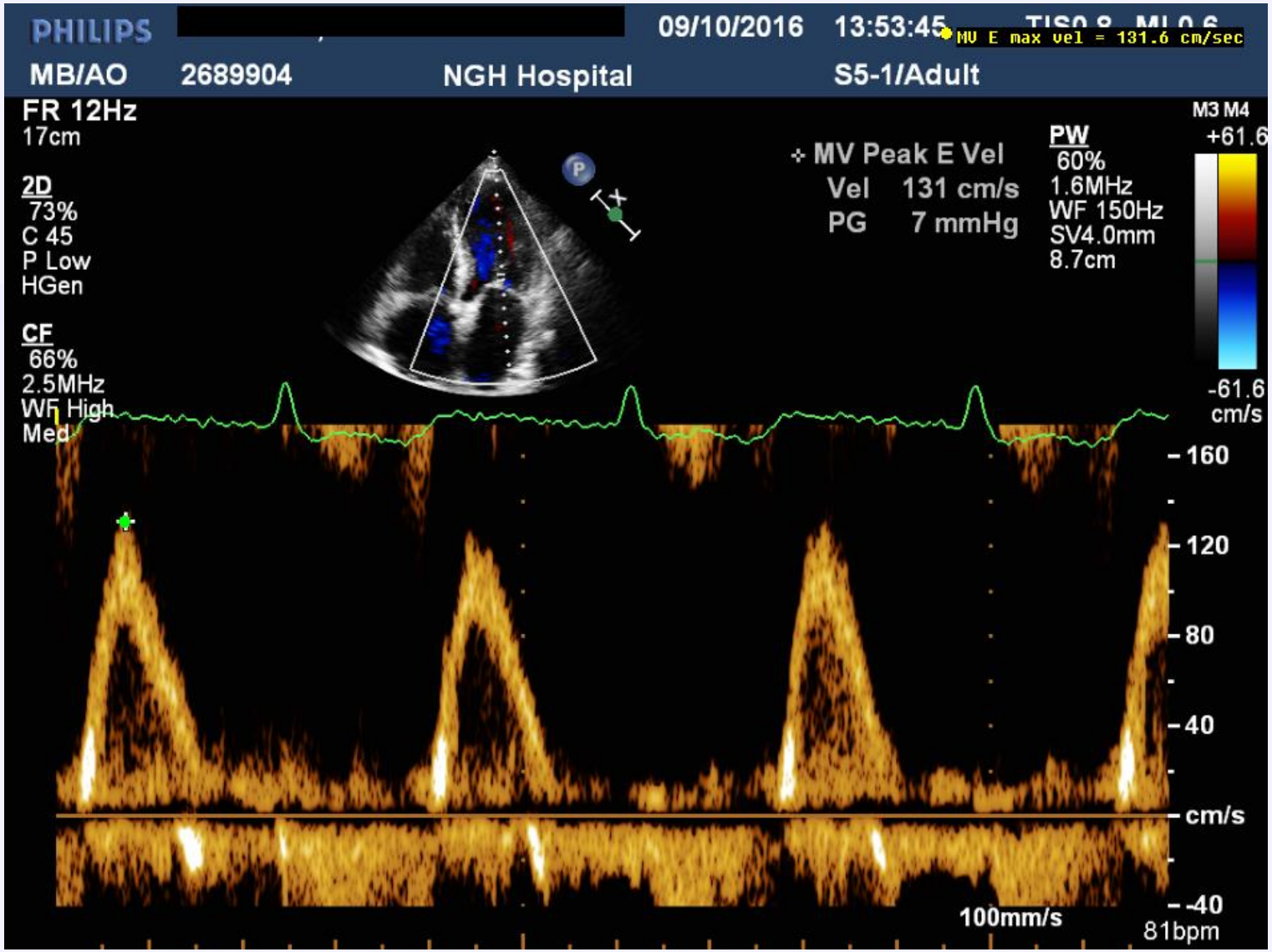
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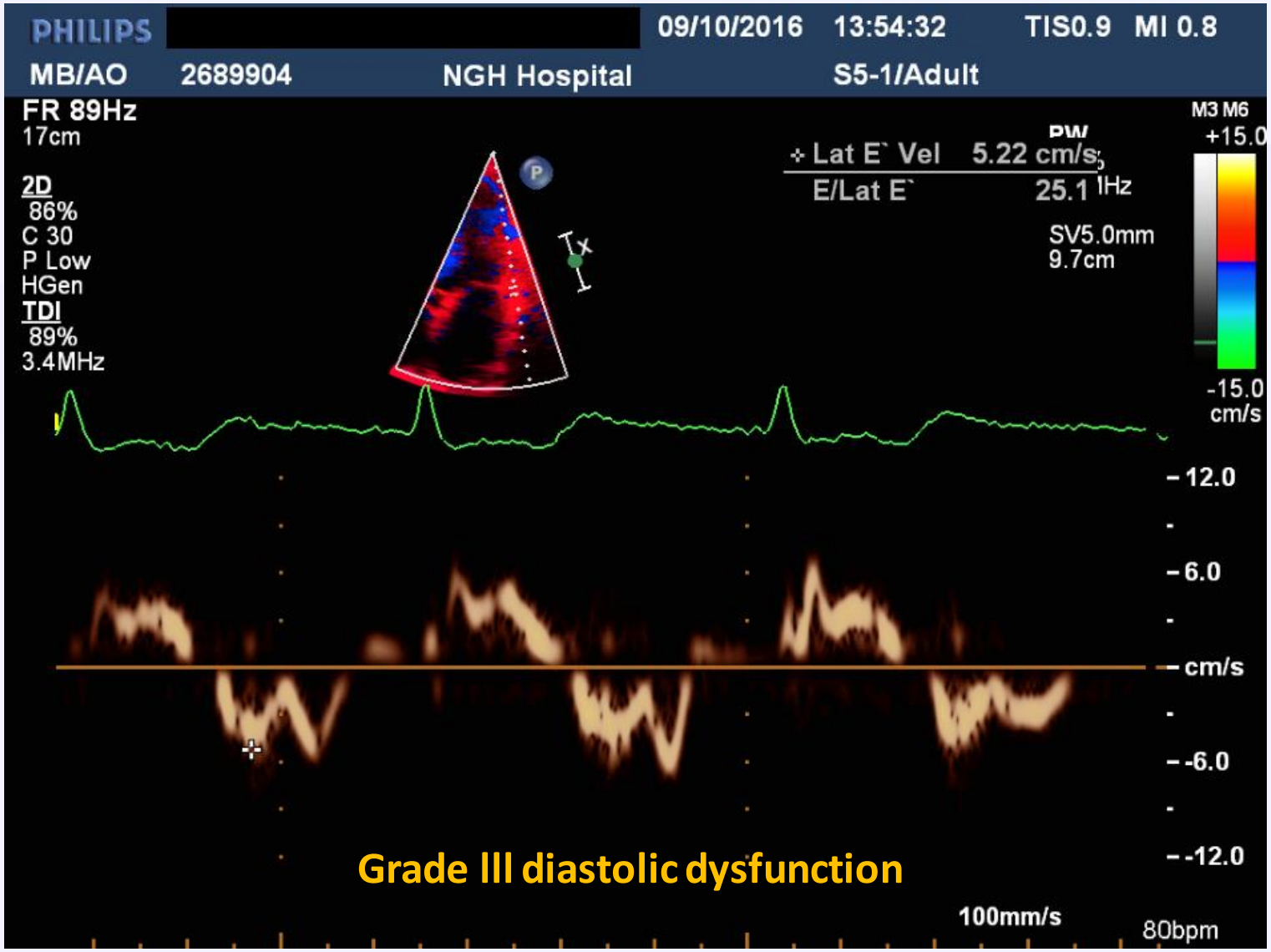
73%
C 45
P Low
HGen
CF
66%
2.5MHz
WF High
Med
MV Peak E Vel
Vel 131 cm/s
PG 7 mmHg
PW
60%
1.6MHz
WF 150Hz
SV4.0mm
8.7cm
M3 M4
+61.6
-61.6
cm/s
160
120
80
40
cm/s
-40
100mm/s
81bpm


PHILIPS
09/10/2016 13:54:32 TIS0.9 MI 0.8
MB/AO 2689904 NGH Hospital S5-1/Adult
FR 89Hz
17cm
2D
86%
C 30
P Low
HGen
TDI
89%
3.4MHz
Lat E` Vel 5.22 cm/s
E/Lat E` 25.1
SV5.0mm
9.7cm
M3 M6
+15.0
-15.0
cm/s
12.0
6.0
cm/s
-6.0
-12.0
100mm/s
80bpm
Grade III diastolic dysfunction

20-OCT-1933 (85 yr)
Male Unknown

Room: 07
Loc: 71

Vent. rate	76	BPM
PR interval	144	ms
QRS duration	112	ms
QT/QTc	482/543	ms
P-R-T axes	47 -86	31

Sinus rhythm with 1st degree A-V block
Left axis deviation
Low voltage QRS
Inferior infarct , age undetermined
Possible Anterolateral infarct , age undetermined
Prolonged QT
Abnormal ECG

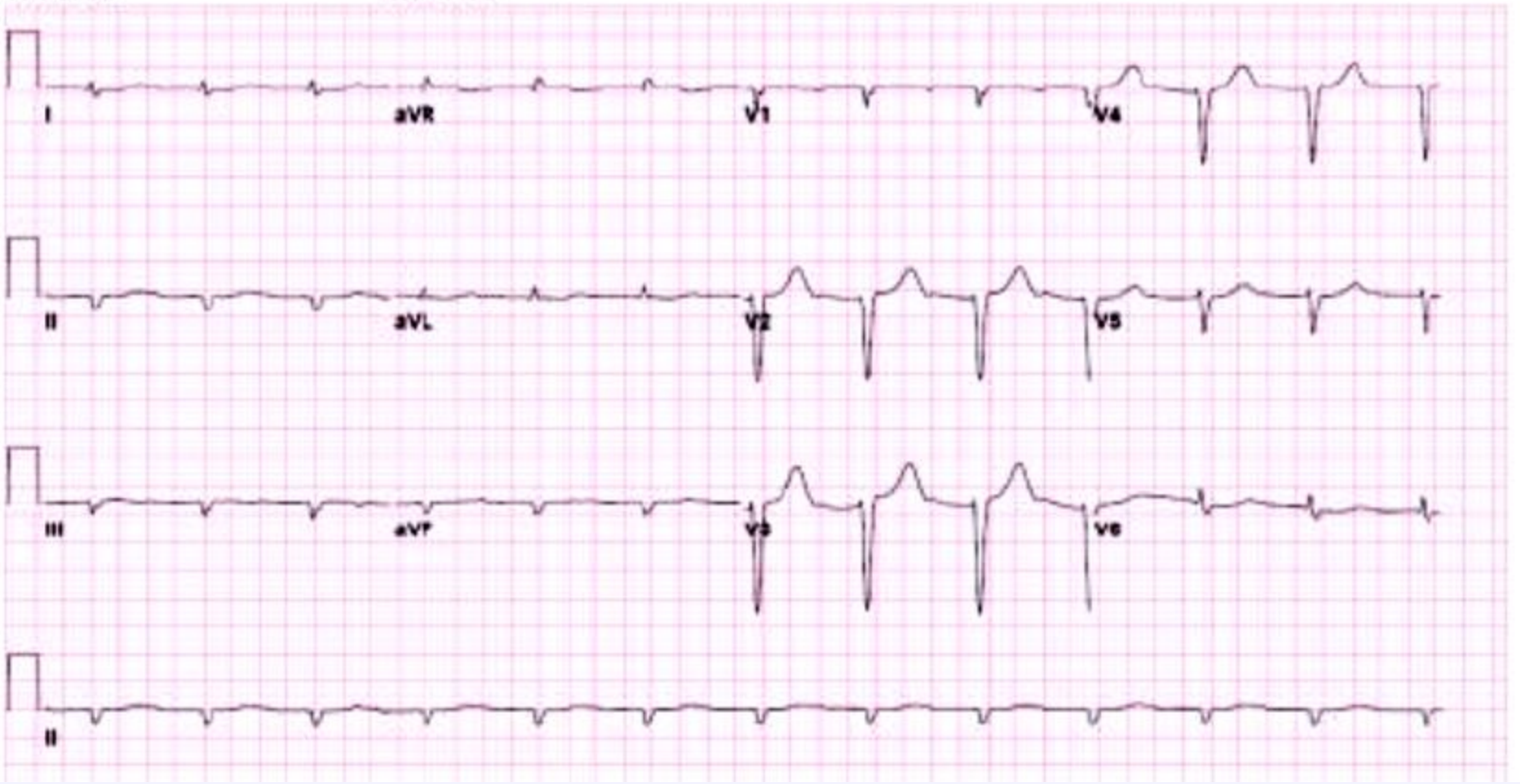
Technician: LOLITA DIZON
Test ind: POST AICD

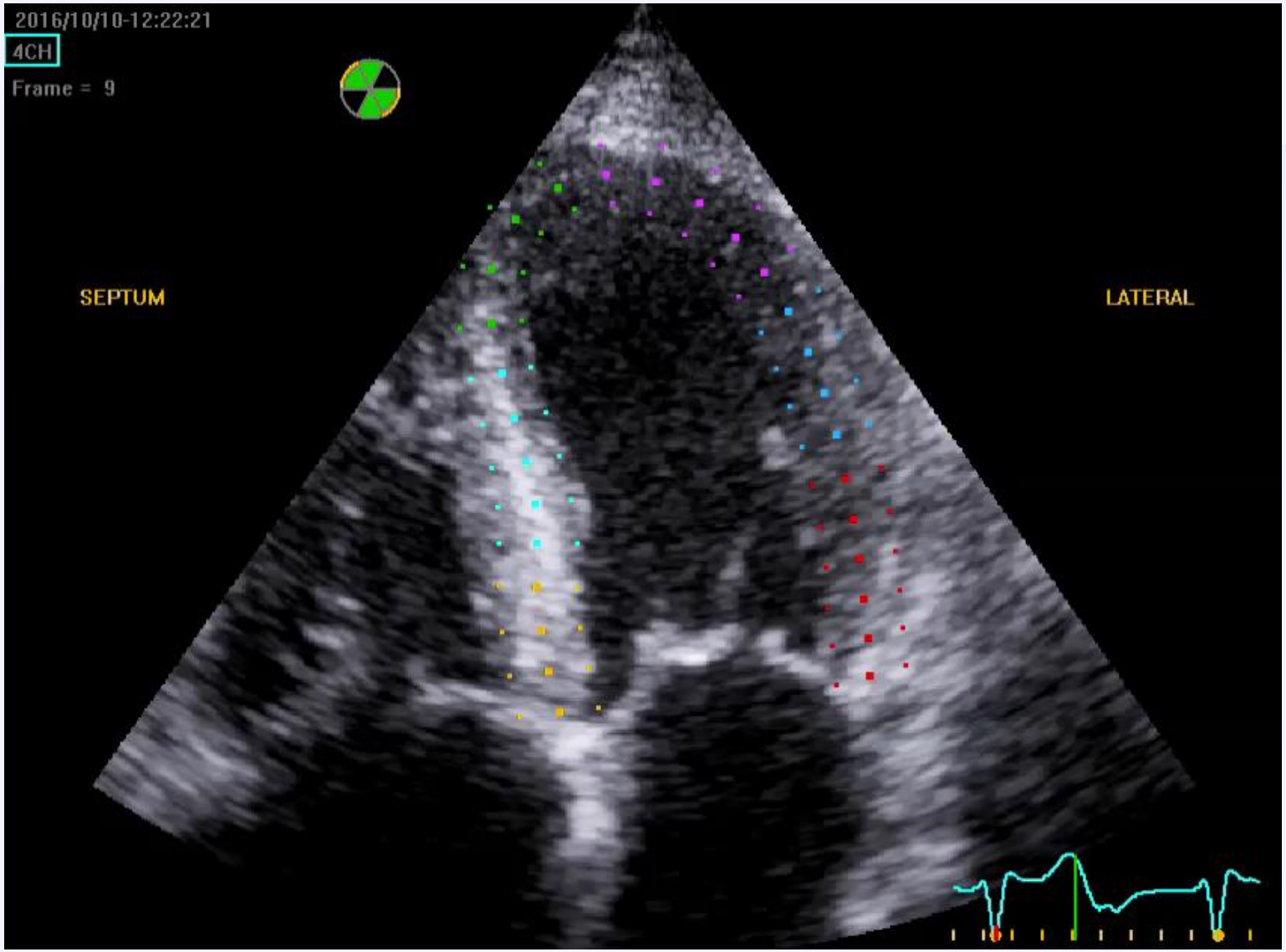
Referred by: Sadaf Al Kahan

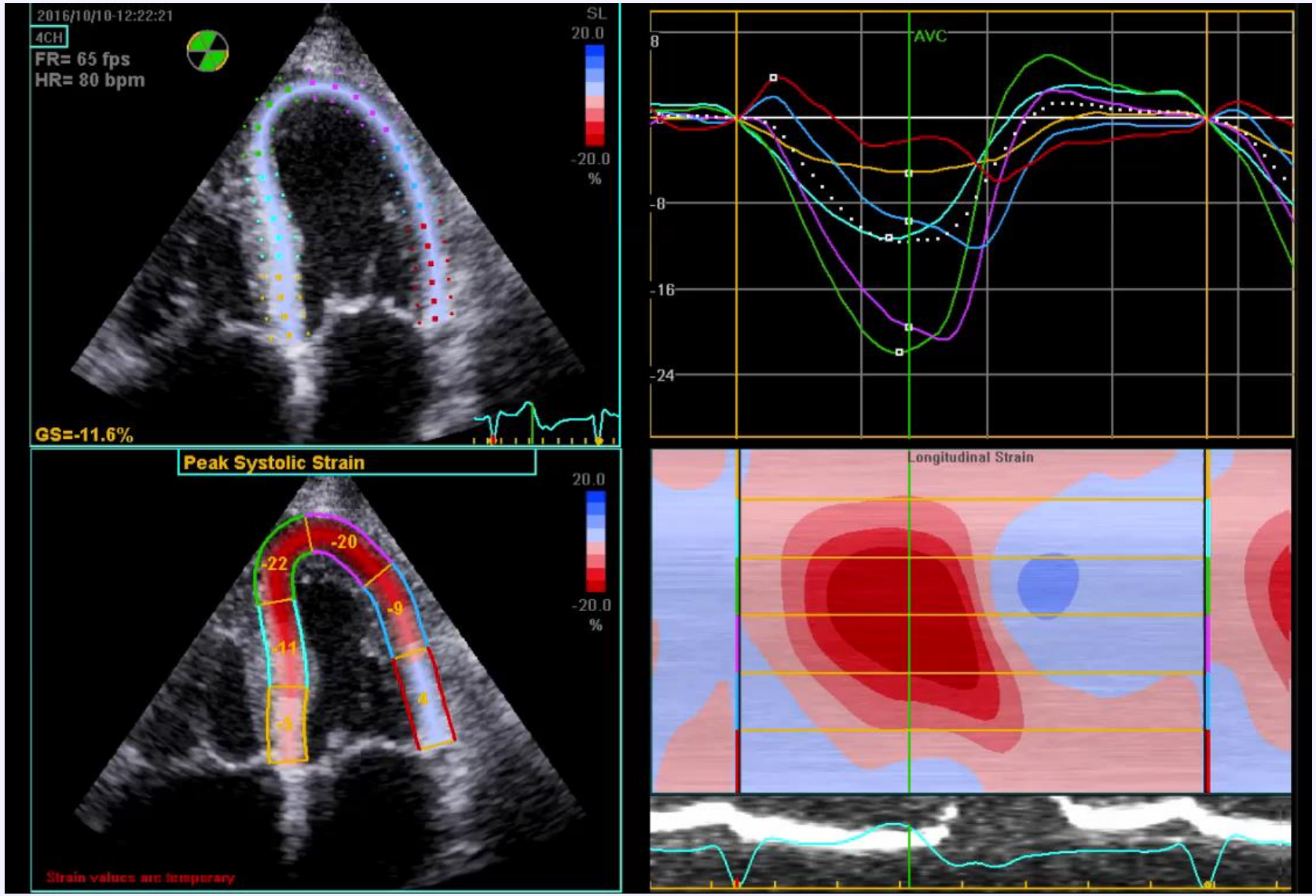
Confirmed By:

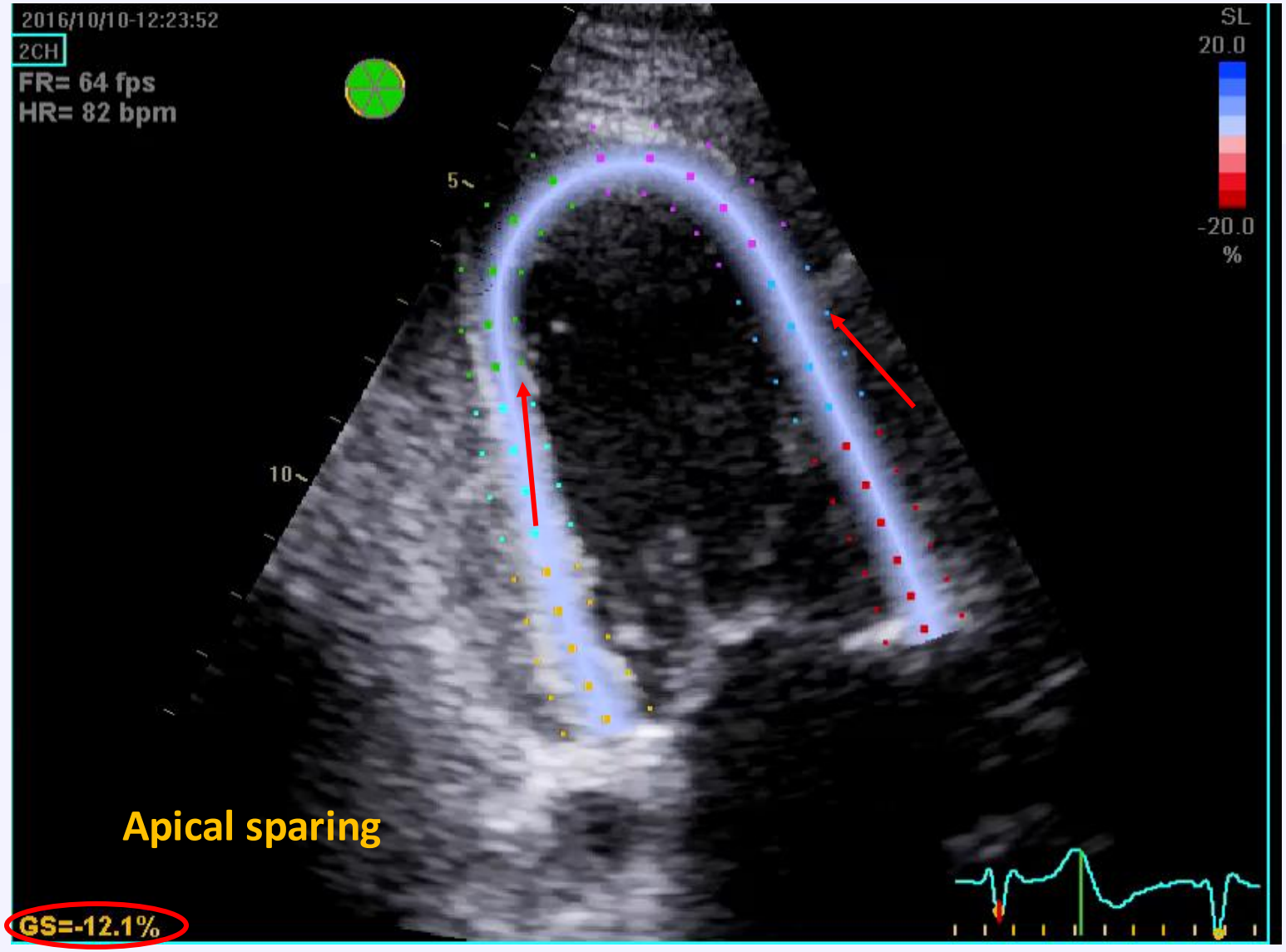
COMMENTS:

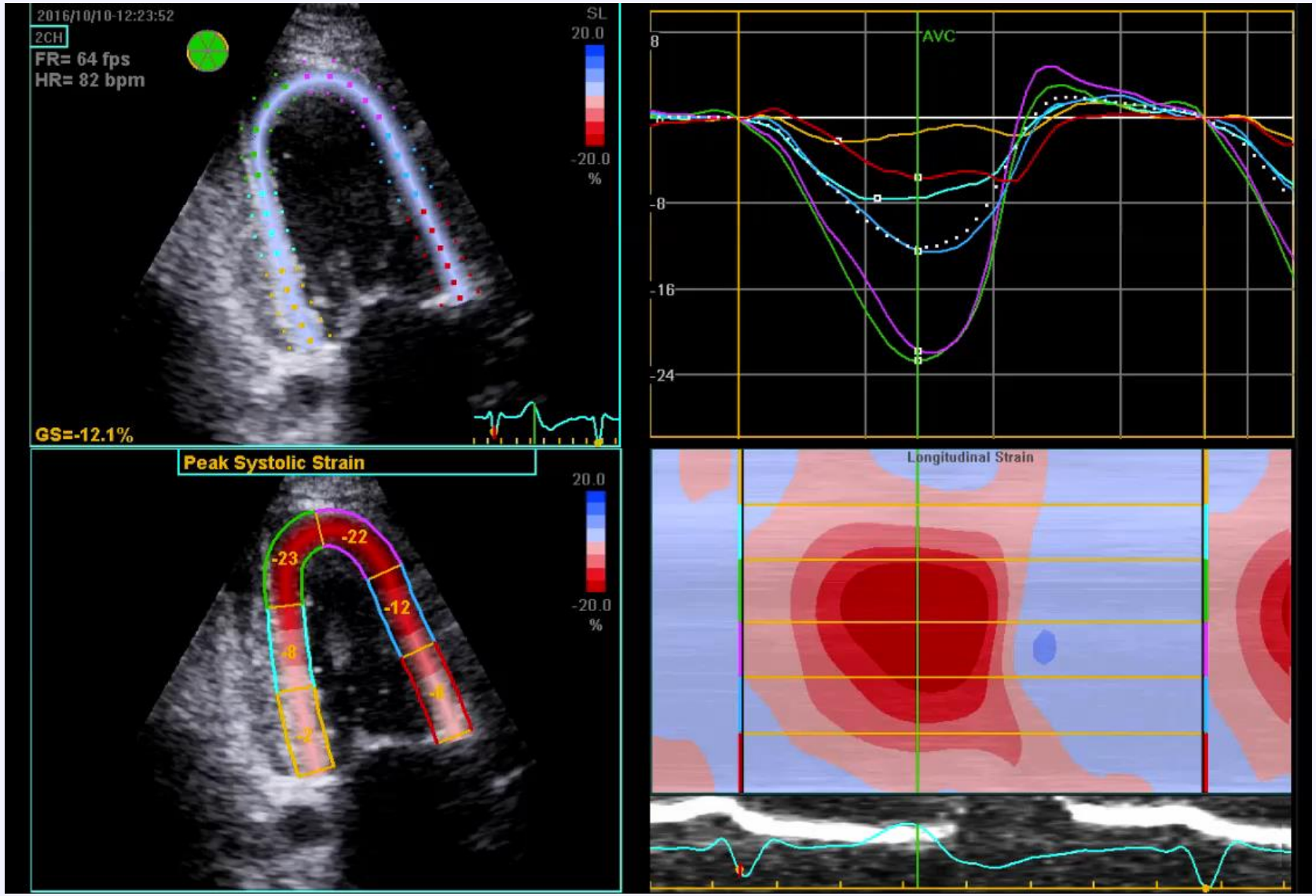
MACE-14: Yes

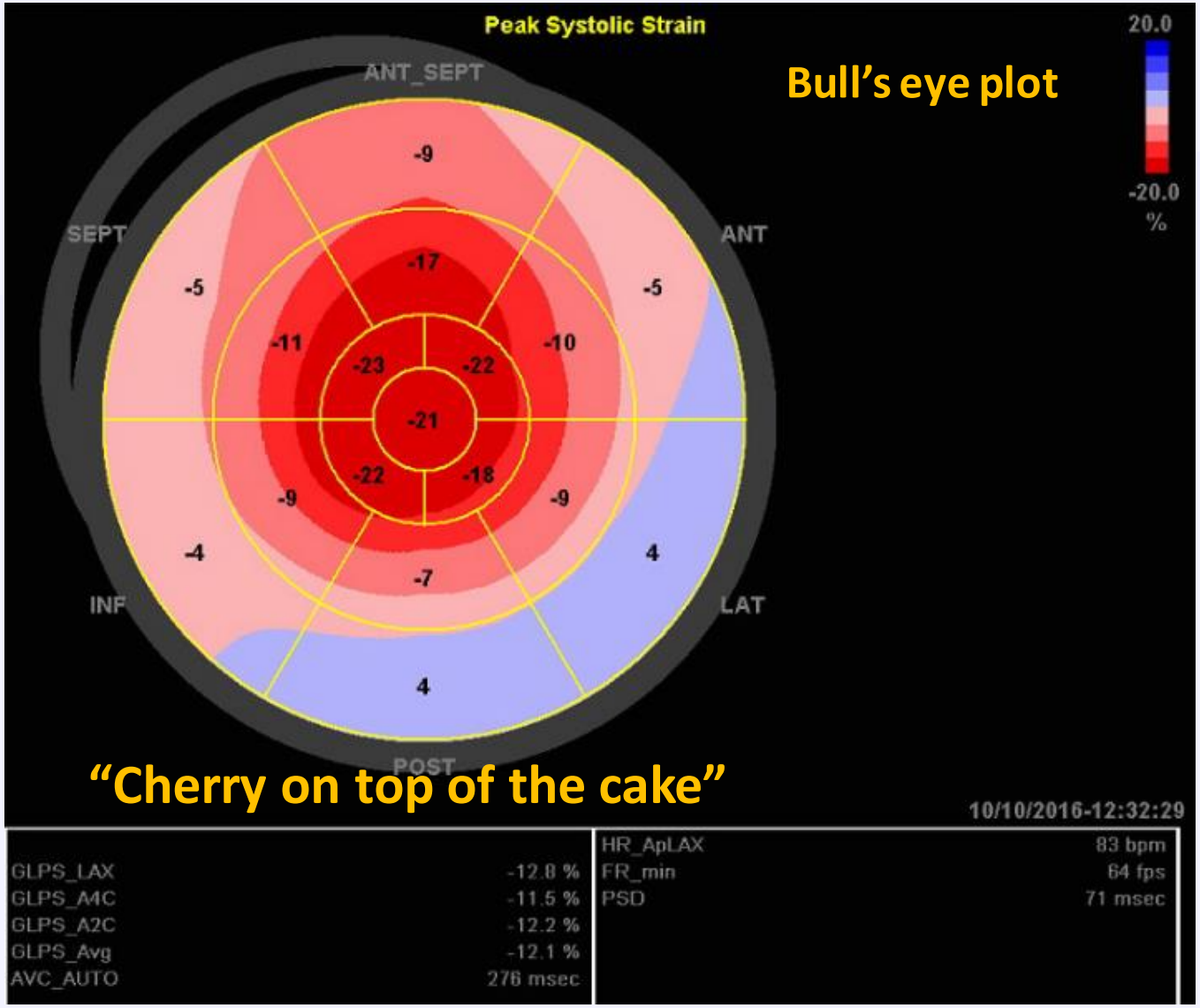



2016/10/10-12:22:21
4CH
Frame = 9
SEPTUM
LATERAL


2016/10/10-12:22:21
4CH
FR= 65 fps
HR= 80 bpm
SL
20.0
-20.0
%
GS=-11.6%
AVC
8
-8
-16
-24
Peak Systolic Strain
20.0
-20.0
%
-20
-22
-9
-11
-4
-4
Strain values are temporary
Longitudinal Strain

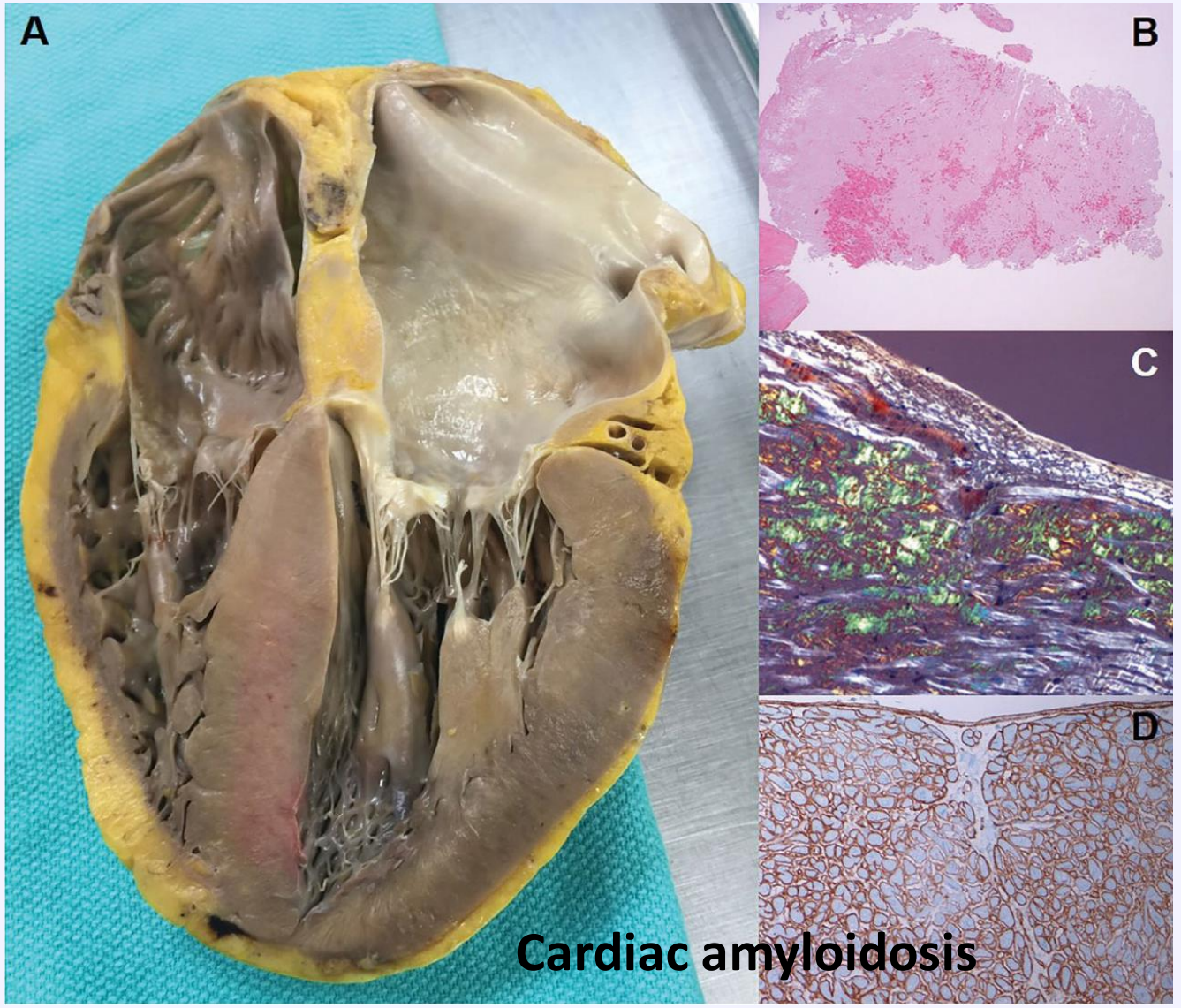

2016/10/10-12:23:52
2CH
FR= 64 fps
HR= 82 bpm
5
10
SL
20.0
-20.0
%
Apical sparing
GS=-12.1%


2016/10/10-12:23:52
2CH
FR= 64 fps
HR= 82 bpm
SL
20.0
-20.0
%
GS=-12.1%
AVC
8
-8
-16
-24
Peak Systolic Strain
20.0
-20.0
%
-22
-23
-12
-8
-4
-2
Longitudinal Strain



Bull's eye plot

"Cherry on top of the cake"



Cardiac amyloidosis


Toronto
Thank you.