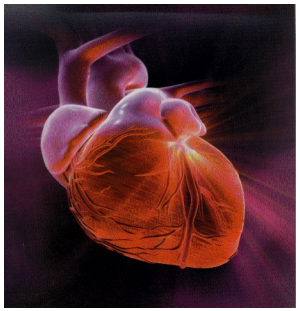
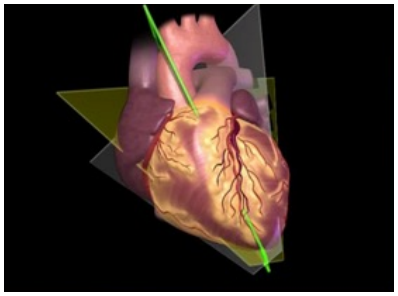
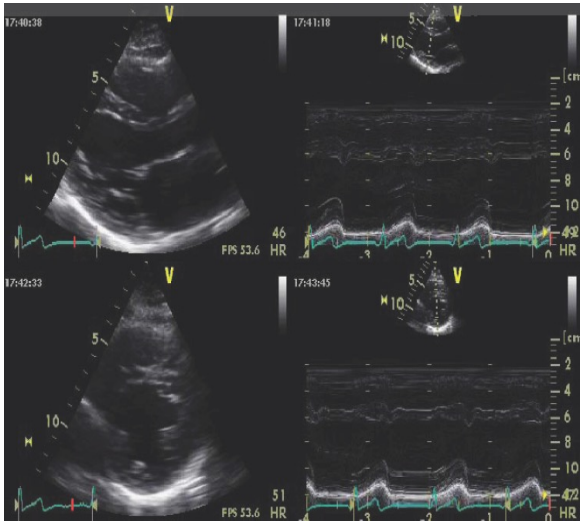


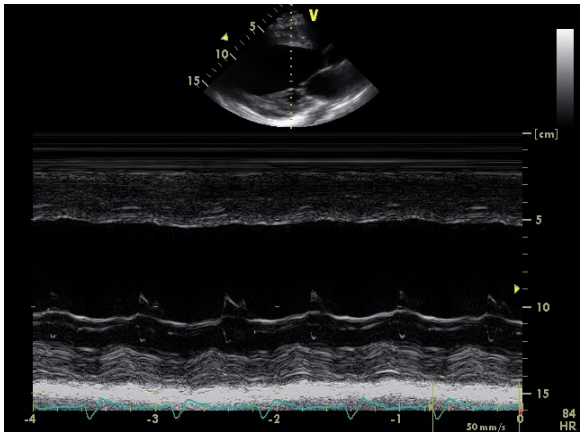


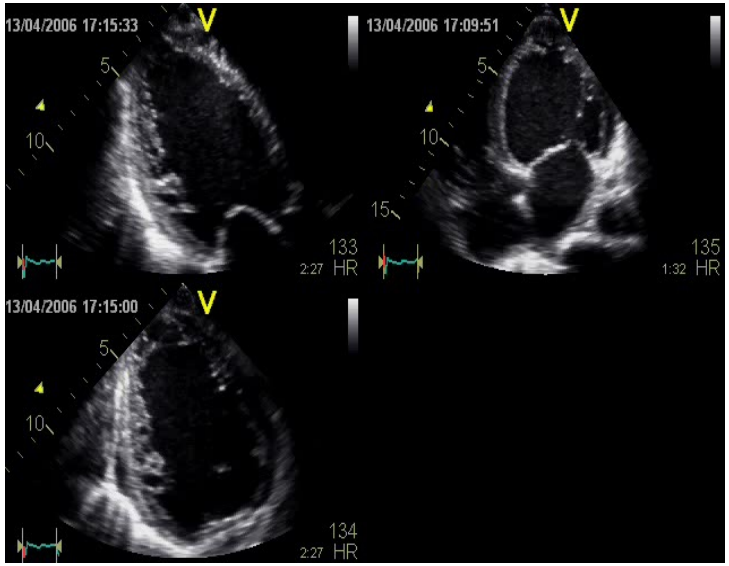
Νέες μέθοδοι απεικόνισης με υπερήχους στην καρδιολογία

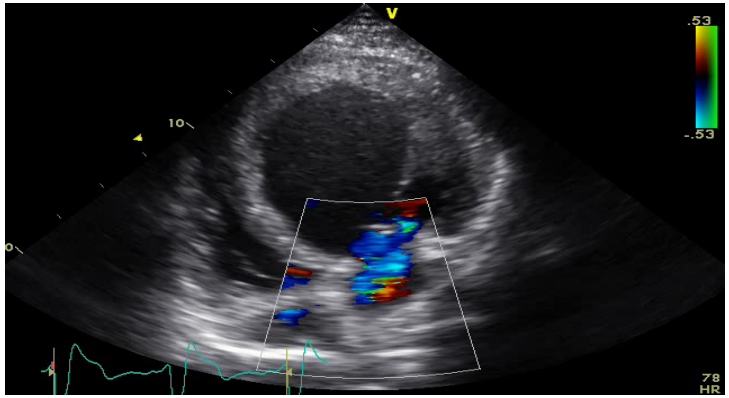
Τσούγκος Ηλίας MD PhD
Επ. Καθηγητής Καρδιολογίας
Διευθυντής ΣΤ Καρδιολογικής Κλινικής Υγεία
Καρδιολόγος Εθνικής Ολυμπιακής Επιτροπής

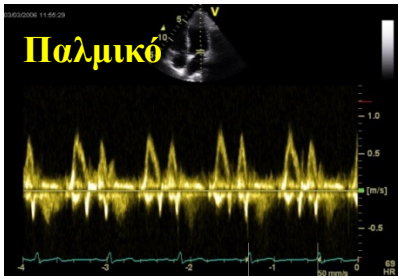
Καθιερωμένες ηχωκαρδιογραφικές τεχνικές

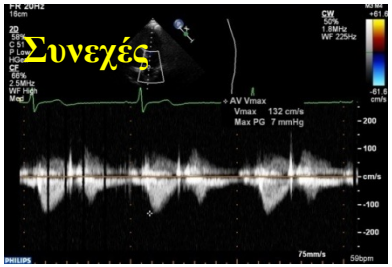


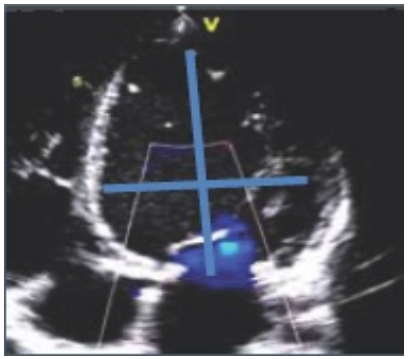






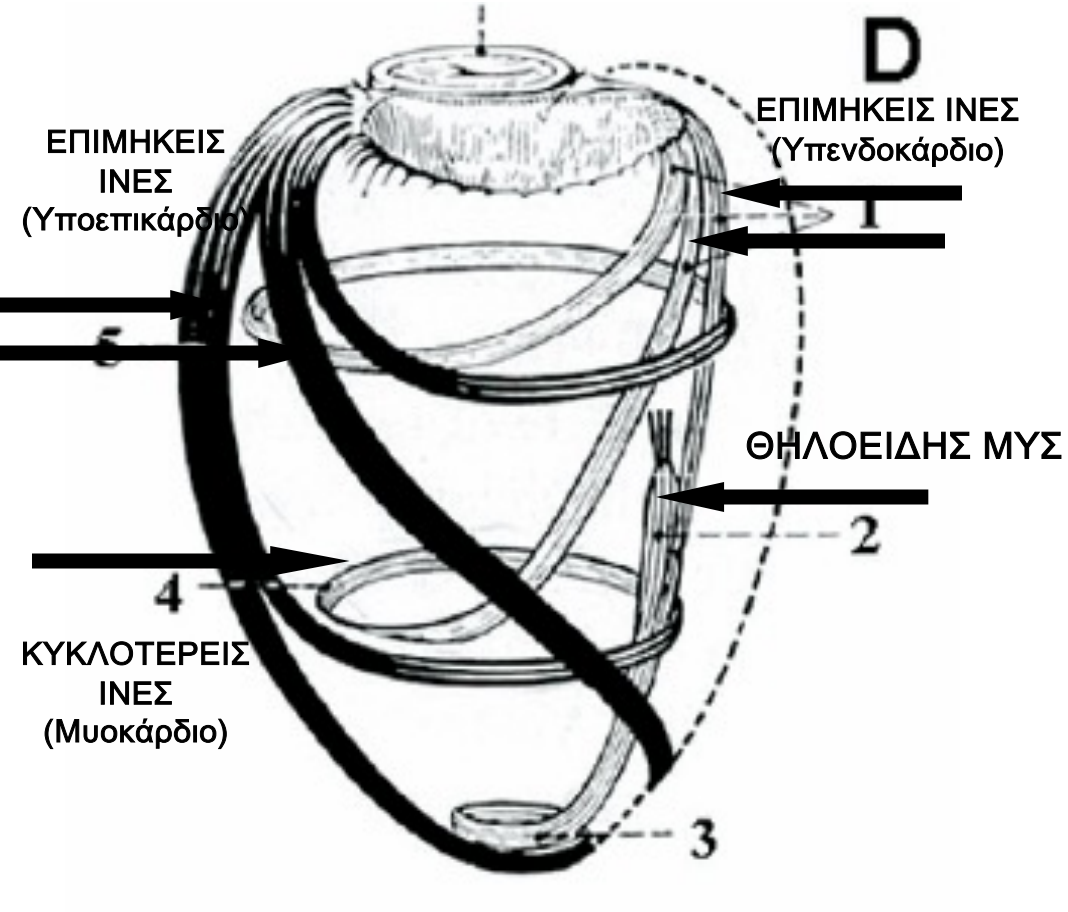


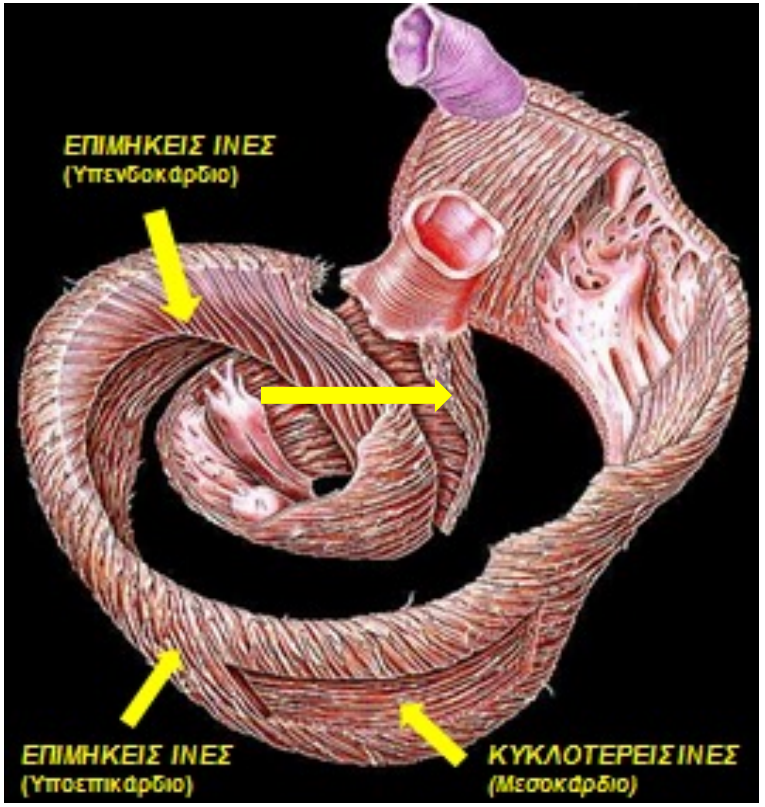




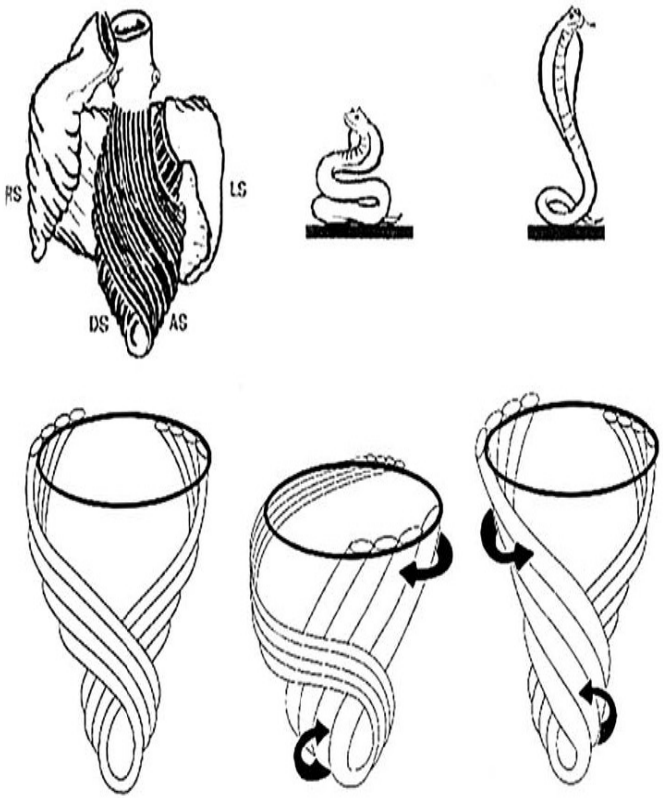
ΠΟΛΥΠΛΟΚΗ ΑΡΧΙΤΕΚΤΟΝΙΚΗ

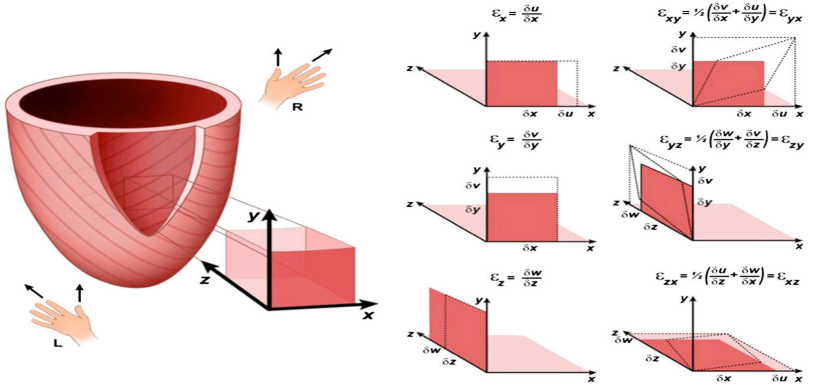
ΤΡΙΔΙΑΣΤΑΤΟ ΟΡΓΑΝΟ

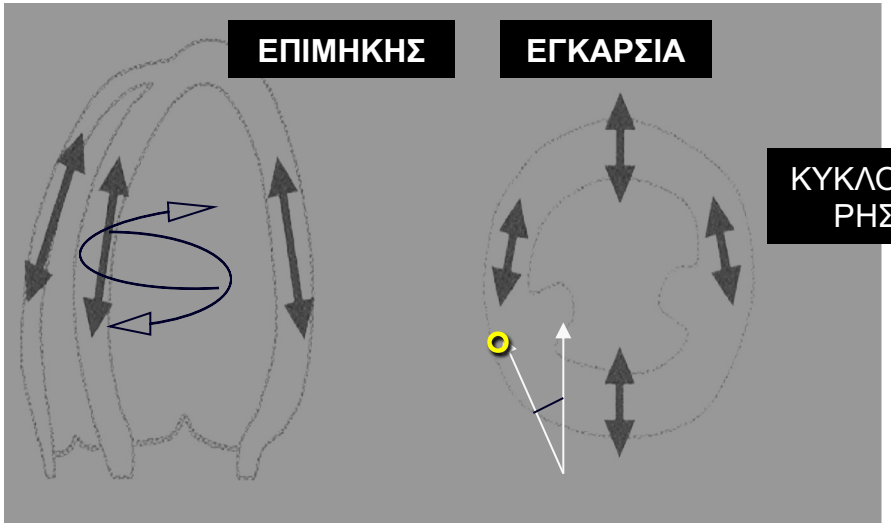




Κίνηση Καρδιακών Τοιχωμάτων







Περιορισμοί συμβατικών ηχωκαρδιογραφικών δεικτών

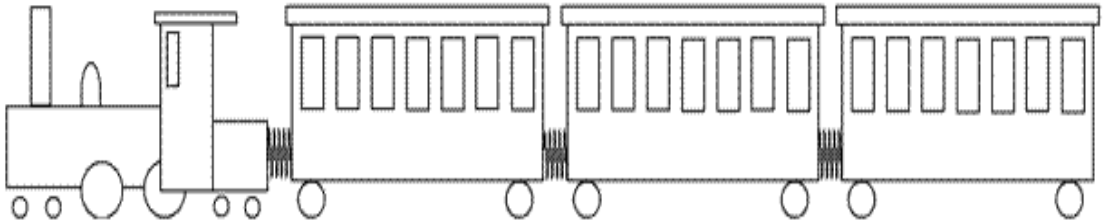
Προσπάθεια απεικόνισης ενός τριδιάστατου οργάνου σε 1 ή 2 διαστάσεις

Παραδοχή ότι η μορφή των καρδιακών δομών είναι συμμετρική

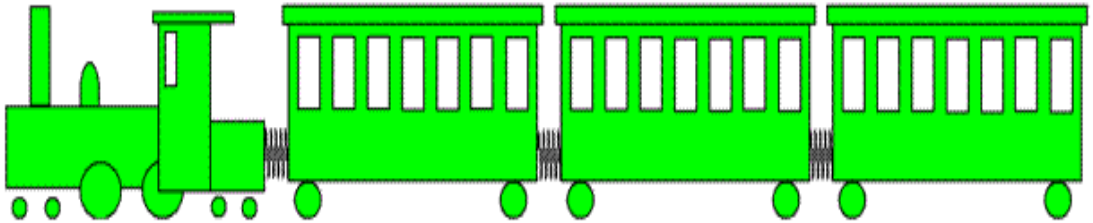
Αδυναμία απεικόνισης σύμπλοκων ανατομικών δομών, και σχέσης τους στο χώρο (πχ δεξιά κοιλία)

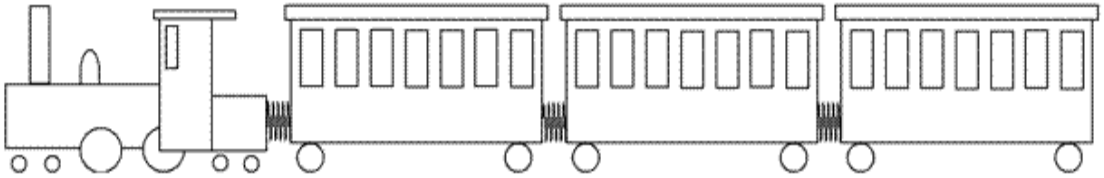
Περιορισμένη ακρίβεια - Μεγάλη διάρκεια πλήρους μελέτης

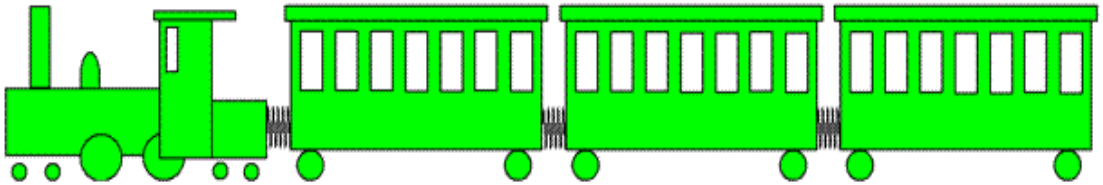
ΚΙΝΗΣΗ



ΠΑΡΑΜΟΡΦΩΣΗ - STRAIN



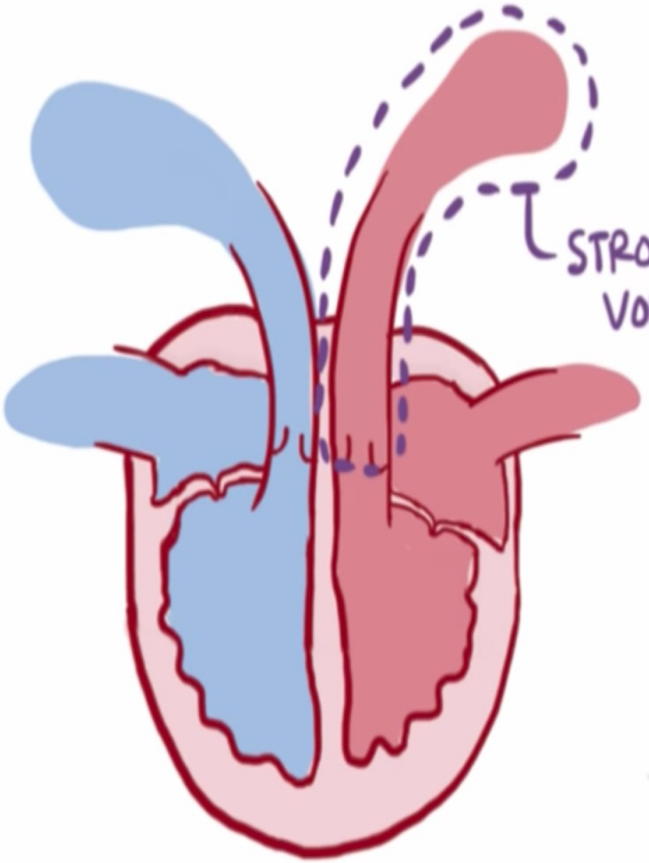




Ejection Fraction

Systolic Heart Failure

* can't pump hard enough



$$\left[\frac{\text{VOLUME BLOOD}}{\text{MINUTE}}\right] \text{ CARDIAC OUTPUT}$$

$$\Downarrow$$

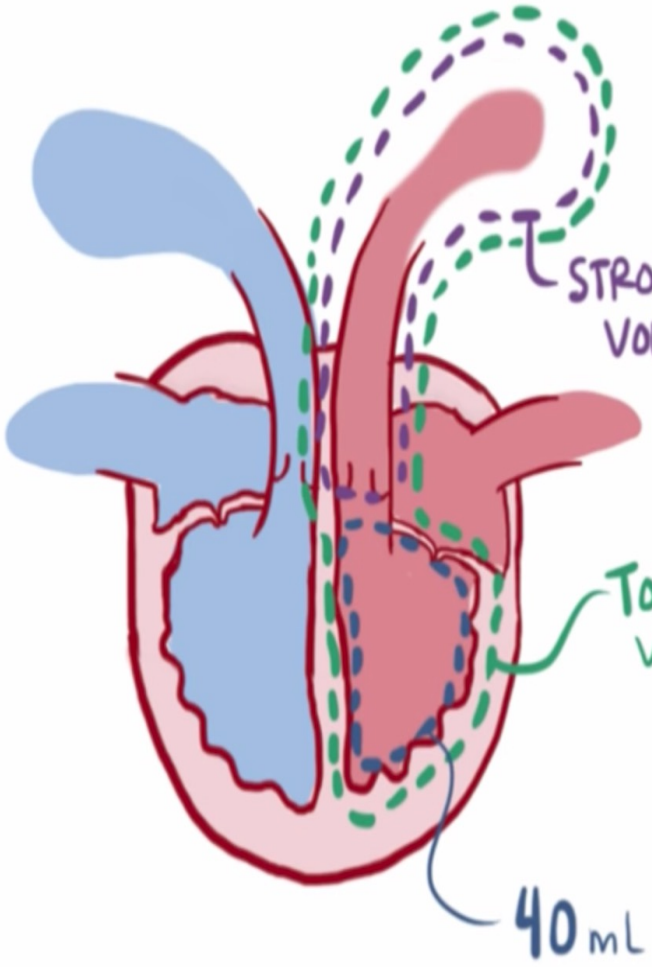
$$\left[\frac{\text{BEATS}}{\text{MINUTE}}\right] \times \left[\frac{\text{VOLUME BLOOD}}{\text{BEAT}}\right]$$

$$70 \frac{\text{beats}}{\text{min}} \times 70 \frac{\text{mL}}{\text{beat}} = 4900 \frac{\text{mL}}{\text{min}}$$

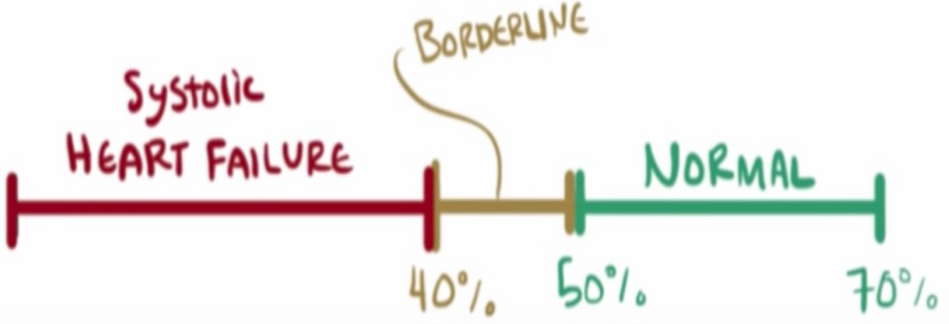
$$\approx 5 \frac{\text{L}}{\text{min}}$$

Systolic Heart Failure

* can't pump hard enough

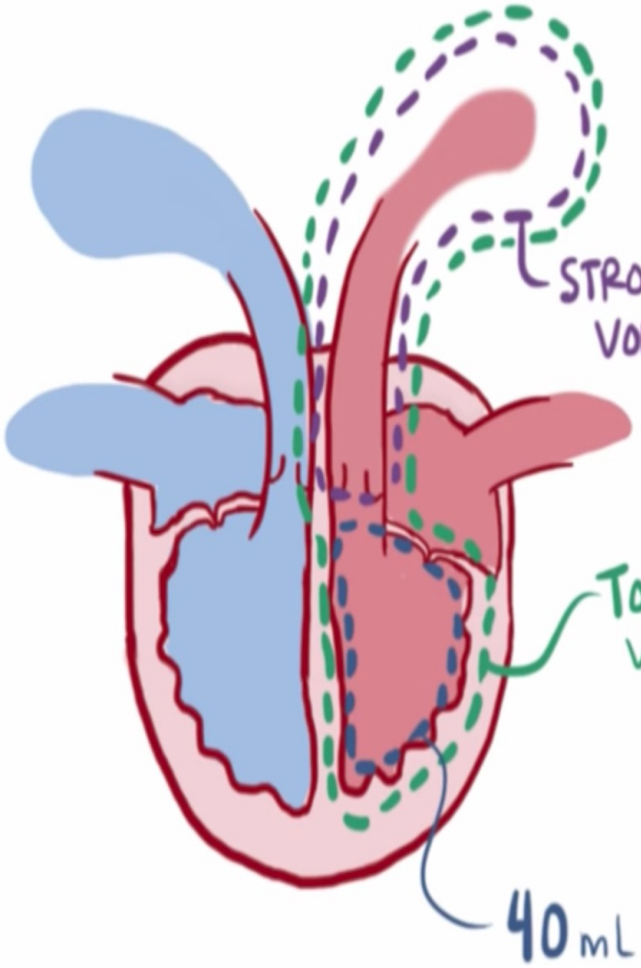


$$\frac{\text{STROKE VOLUME } 70\text{ mL}}{\text{TOTAL VOLUME } 110\text{ mL}} = \text{EJECTION FRACTION} \approx 64\%$$

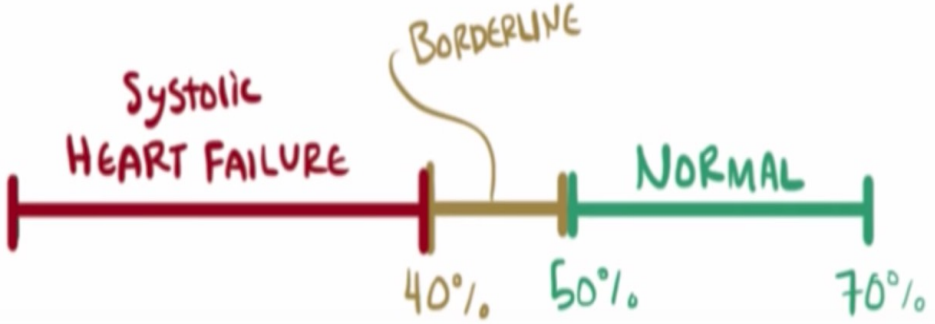


Systolic Heart Failure

* can't pump hard enough

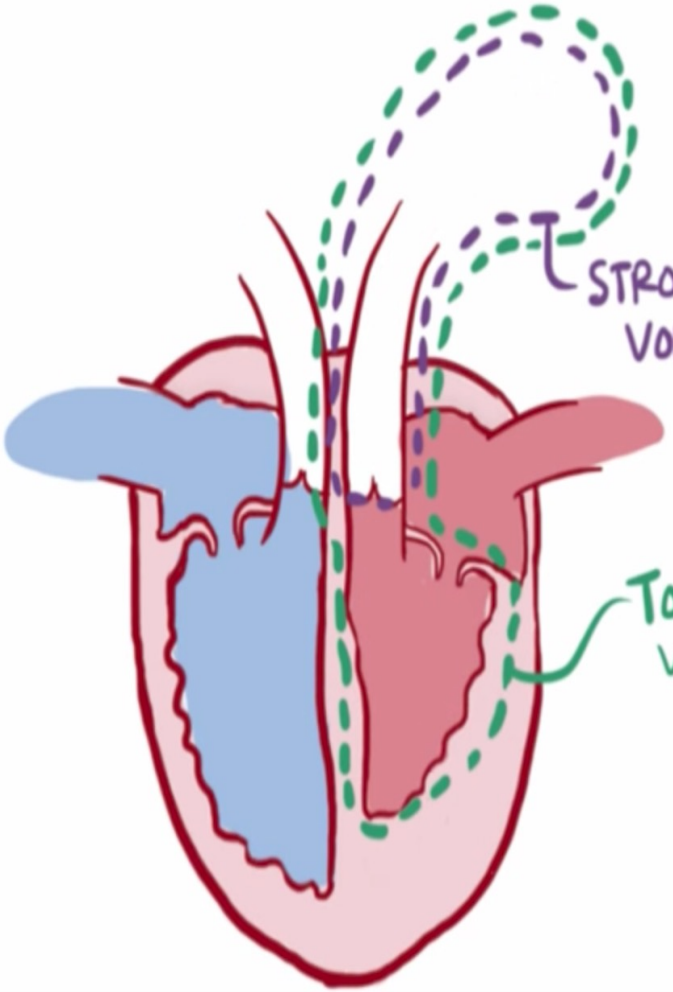


$$\frac{\text{Stroke Volume } 44\text{ mL}}{\text{Total Volume } 110\text{ mL}} = \text{Ejection Fraction} \approx 40\,\%$$



Diastolic Heart Failure

* not filling enough



Still low

Stroke Volume: 44 mL

Total Volume: 69 mL

$$\frac{44\ \text{mL}}{69\ \text{mL}} = \text{Ejection Fraction} \approx 64\ \%$$

NORMAL !

Ejection Fraction: A Chimeric Index

$$\text{Ejection Fraction (\%)} = \frac{\text{EDV-ESV (SV)}}{\text{EDV}}$$

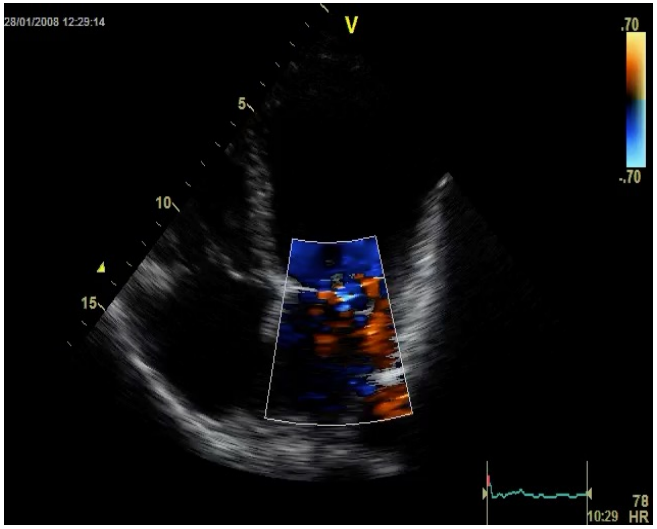
Preload
Afterload
Inotropy

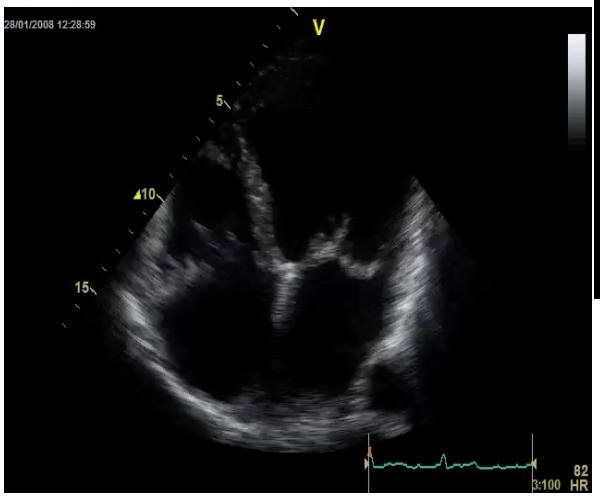
"Physiological variables"

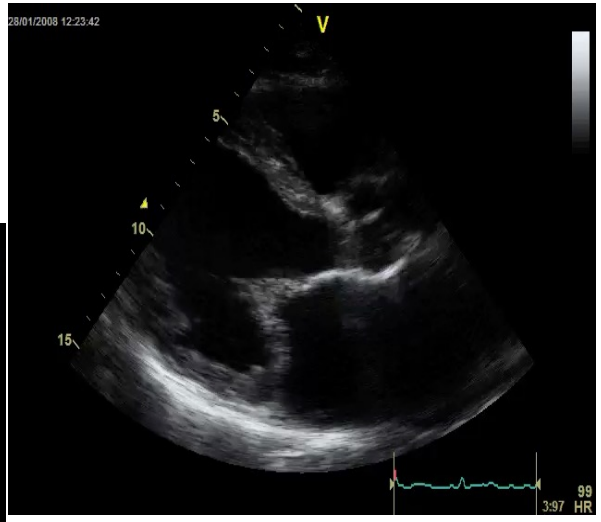
LV wall thickness
LV size and shape

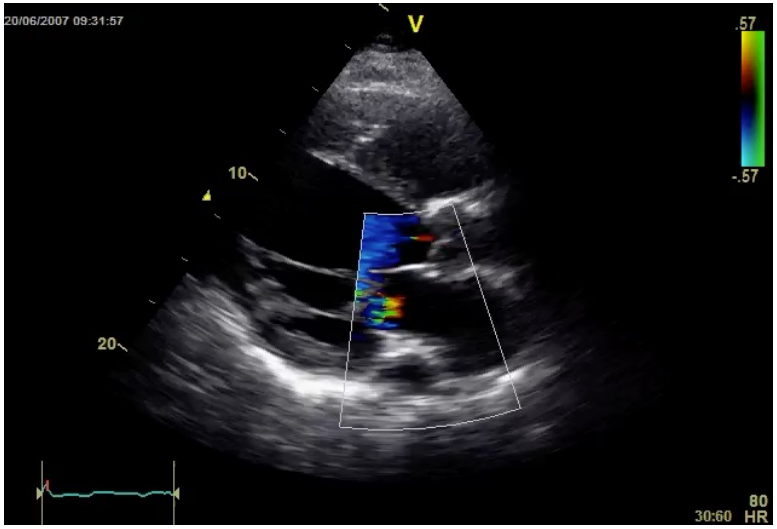
"Architectural variables"

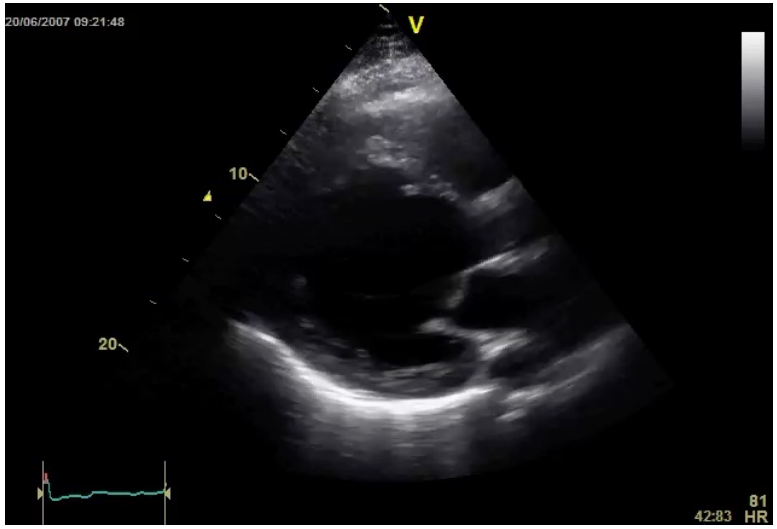
Katz AM, et al. Eur Heart J. 2016;37:449–454

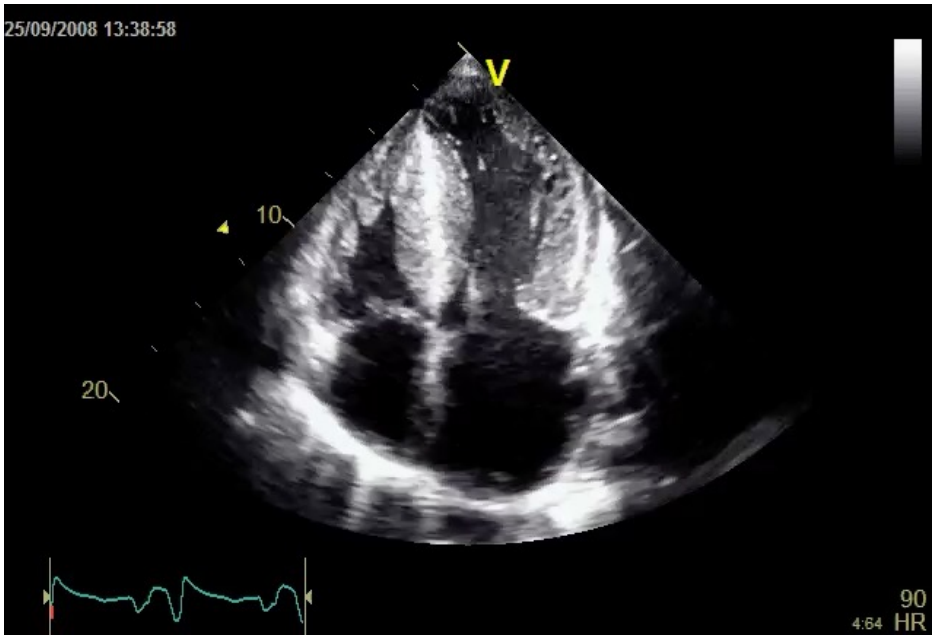

28/01/2008 12:29:14
V
.70
-.70
5
10
15
78
10:29 HR

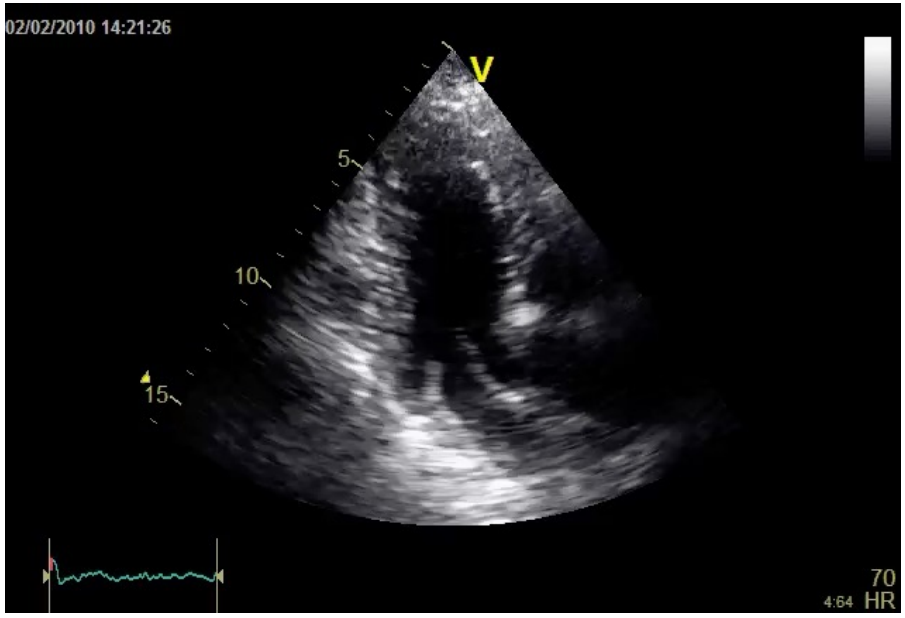

28/01/2008 12:28:59
V
5
10
15
82
3:100 HR

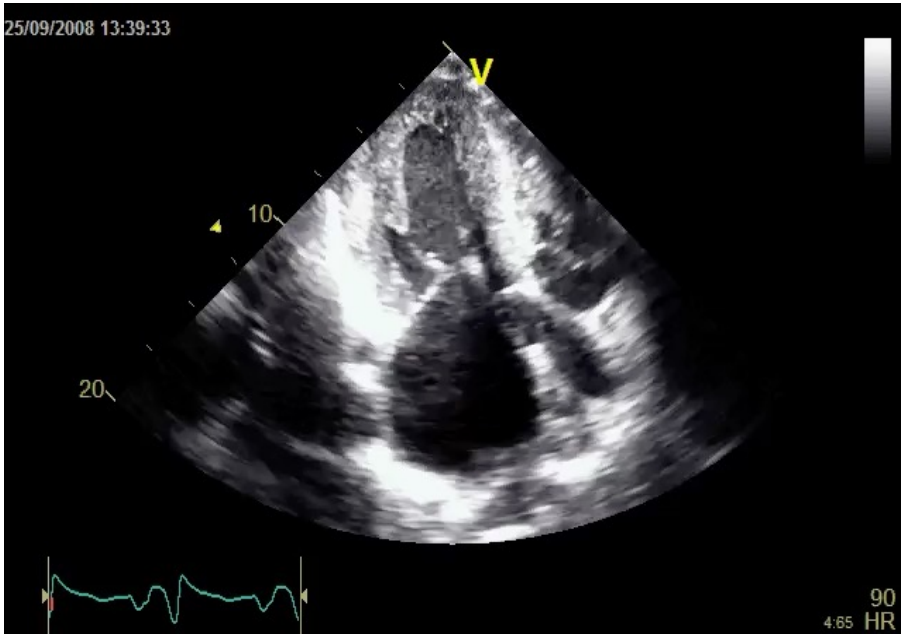

28/01/2008 12:23:42
V
5
10
15
99
3:97 HR


20/06/2007 09:31:57
V
.57
-.57
10
20
80
30:60 HR


20/06/2007 09:21:48
V
10
20
81
42:83 HR


25/09/2008 13:38:58
V
10
20
90
4:64 HR


02/02/2010 14:21:26
V
5
10
15
70
4:64 HR


25/09/2008 13:39:33
V
10
20
90
4:65 HR

Conclusion I

1. Sensitive to loading conditions for instance mitral insufficiency with mild dysfunction means actually great loss of EF
2. The term preserved could be misleading confusing and mistakenly reassuring

Intra and inter observer variability relatively unreliable

JAMA Network Open

Original Investigation | Cardiology

Variability in Ejection Fraction Measured By Echocardiography, Gated Single-Photon Emission Computed Tomography, and Cardiac Magnetic Resonance in Patients With Coronary Artery Disease and Left Ventricular Dysfunction

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Abstract

IMPORTANCE Clinical decisions are frequently based on measurement of left ventricular ejection fraction (LVEF). Limited information exists regarding inconsistencies in LVEF measurements when determined by various imaging modalities and the potential impact of such variability.

OBJECTIVE To determine the intermodality variability of LVEF measured by echocardiography, gated single-photon emission computed tomography (SPECT), and cardiovascular magnetic resonance (CMR) in patients with left ventricular dysfunction.

DESIGN, SETTING, AND PARTICIPANTS International multicenter diagnostic study with LVEF imaging performed at 127 clinical sites in 26 countries from July 24, 2002, to May 5, 2007, and measured by core laboratories. Secondary study of clinical diagnostic measurements of LVEF in the Surgical Treatment for Ischemic Heart Failure (STICH), a randomized trial to identify the optimal treatment strategy for patients with LVEF of 35% or less and coronary artery disease. Data analysis was conducted from March 19, 2016, to May 29, 2018.

MAIN OUTCOMES AND MEASURES At baseline, most patients had an echocardiogram and subsets of patients underwent SPECT and/or CMR. Left ventricular ejection fraction was measured by a core laboratory for each modality independent of the results of other modalities, and measurements were compared among imaging methods using correlation, Bland-Altman plots, and coverage probability methods. Association of LVEF by each method and death was assessed.

RESULTS A total of 2032 patients (mean [SD] age, 60.9 [9.6] years; 1759 [86.6%] male) with baseline LVEF data were included. Correlation of LVEF between modalities was $r = 0.601$ (for biplane echocardiography and SPECT [n = 385]), $r = 0.493$ (for biplane echocardiography and CMR [n = 204]), and $r = 0.660$ (for CMR and SPECT [n = 134]). Bland-Altman plots showed only moderate agreement in LVEF measurements from all 3 core laboratories with no substantial overestimation or underestimation of LVEF by any modality. The percentage of observations that fell within a range of 5% ranged from 43% to 54% between different imaging modalities.

CONCLUSIONS AND RELEVANCE In this international multicenter study of patients with coronary artery disease and reduced LVEF, there was substantial variation between modalities in LVEF determination by core laboratories. This variability should be considered in clinical management and trial design.

Key Points

Question What is the variability in left ventricular ejection fraction (LVEF) as measured by different cardiac imaging modalities?

Findings In this multicenter diagnostic study of 2032 patients with coronary artery disease and LVEF of 35% or less with imaging interpreted by core laboratories, correlation of LVEF between modalities ranged from $r = 0.493$ (for biplane echocardiography and cardiovascular magnetic resonance) to $r = 0.660$ (for cardiovascular magnetic resonance and gated single-photon emission computed tomography). There was no systematic overestimation or underestimation of LVEF for any modality.

Meaning There is substantial variability in LVEF assessment between modalities, which should be considered in trial design and clinical management.

+ Supplemental content

Author affiliations and article information are listed at the end of this article.

Pelikka PA, et al. JAMA Netw Open. 2018 ;1(4):e181456

VIEWPOINT

Redefining Heart Failure With a Reduced Ejection Fraction

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Baylor Heart and Vascular Institute, Baylor University Medical Center, Dallas, Texas; and Imperial College, London, United Kingdom.

The current management of patients with chronic heart failure depends on the noninvasive measurement of left ventricular ejection fraction (LVEF). In patients with an LVEF of 40% or lower, large-scale randomized clinical trials have demonstrated the benefits of inhibitors of the renin-angiotensin system, sympathetic nervous system, aldosterone, and neprilysin in reducing the risk of cardiovascular death and hospitalization for heart failure. Because these trials only enrolled patients with an LVEF of 40% or lower, a value of 40% has been used to define patients with heart failure and a reduced ejection fraction (HFrEF) for the past 30 years. Current guidelines strongly recommend the use of combination treatment with neurohormonal antagonists for patients with HFrEF.[1] By contrast, there are no evidence-based recommendations concerning the treatment of patients with LVEF greater than 40%, who have been conventionally referred to as having heart failure with a preserved ejection fraction (HFpEF). This lack of guidance is a concern because such patients now represent a majority of those with heart failure in the general community, particularly among women.[2]

How Should Patients With Impaired Systolic Function Be Identified?

Despite its historical use, a value for LVEF of 40% does not distinguish patients with heart failure who have normal LVEF from those who have abnormally low LVEF values. Like many measurements in medicine, LVEF is a continuous variable, and the identification of normal values is dependent on various variables including sex and age. Guidelines indicate that the low end of normal for LVEF is 52% in men and 54% in women[3]; an LVEF of 41% to 51% in men and 41% to 53% in women is regarded as mildly reduced. However, despite having meaningful systolic dysfunction, these patients were not enrolled in trials of HFrEF because those studies were designed to have high event rates to make their sample sizes financially feasible.

Because an LVEF of 40% or lower was used as a criterion for enrollment in studies of HFrEF, when trials of patients with HFpEF were first conducted, they focused on patients who had been excluded from trials of HFrEF, ie, they required patients to have an LVEF higher than 40%.[4] Early investigators deemed such patients to have preserved ejection fraction because they understood that the group included patients with a subnormal LVEF (<50%-55%) as well as patients with an LVEF in the normal range (>50%-55%). In 2013, the ACCF/AHA (American College of Cardiology Foundation/American Heart Association) guidelines classified patients with heart failure who had an LVEF of 41% through 49% as having "HFpEF, borderline" and considered them to be distinct from those with HFrEF.[1] More recently, the 2016 ESC guideline classified patients with an LVEF of 40% through 49% as having "heart failure with a mid-range ejection fraction."[5] The authors formulated this category to encourage further study of this intermediate group. However, this intent was widely misunderstood, and many physicians considered this mid-range group to represent a new distinct clinical entity.

Any classification of heart failure that relies on LVEF has inherent limitations. First, the measurement of LVEF is highly dependent on the method used for imaging, and even when the same method is used, there is considerable intraobserver and interobserver variability. Repeat measurements of LVEF in the same patients using the same methods by experts in echocardiography routinely vary by 7%; the variability is greater in clinical practice. When the echocardiograms of patients enrolled in clinical trials are reviewed using standardized criteria, differences between the values obtained by site investigators and the core laboratory routinely vary as much as 15% when reading the same images. Furthermore, the quality of images is highly operator-dependent, and the values for LVEF depend on loading conditions, ie, volume status and blood pressure. Hence, it is likely that a meaningful proportion of patients with an LVEF of 40% to 50% would be reclassified as having an LVEF of lower than 40% or higher than 50% if the measurement were repeated.

Perhaps more important, when assessed using biomarkers that reflect potential disease mechanisms, patients with HFrEF typically show evidence of increased circulating levels of proteins that reflect the occurrence of cardiomyocyte injury, loss, and stretch. In contrast, patients who have heart failure and an LVEF higher than 50% typically show biomarkers that reflect systemic inflammation and evidence of endothelial injury and myocardial fibrosis. It is therefore noteworthy that, in these studies, patients with an LVEF of 40% to 50% exhibit a pathophysiological profile that closely resembles patients with an LVEF lower than 40%, but manifest a profile that differs from patients with heart failure and an LVEF higher than 50%.[6]

Benefit of Neurohormonal Antagonists In Patients With an LVEF of 40%-50%

The concept that patients with heart failure and an LVEF of 40% to 50% have similar clinical features as those with an LVEF lower than 40% is strongly supported by the results of several large-scale randomized trials that enrolled patients with preserved ejection fraction. Each trial enrolled patients who had chronic mild to severe symptoms of heart failure, including those with and without underlying coronary artery disease or hypertension.

The CHARM-Preserved trial[4] evaluated the effects of the angiotensin receptor blocker candesartan in 3023 patients with heart failure and LVEF higher than 40%. Compared with placebo, candesartan reduced the risk

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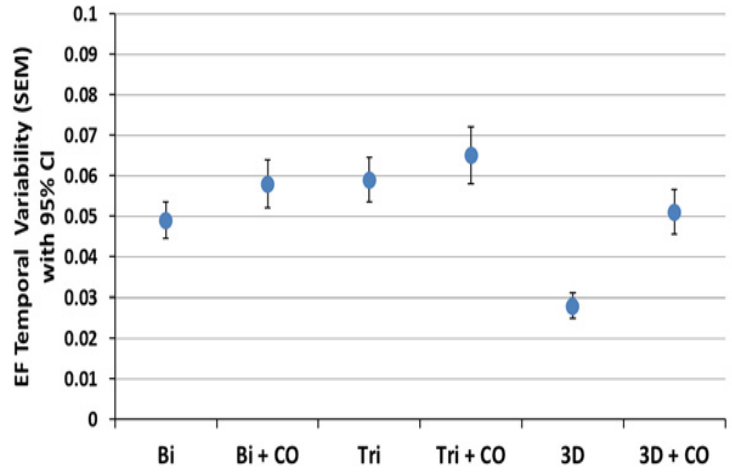
Butler J, et al. JAMA. 2019;322(18):1761-1762

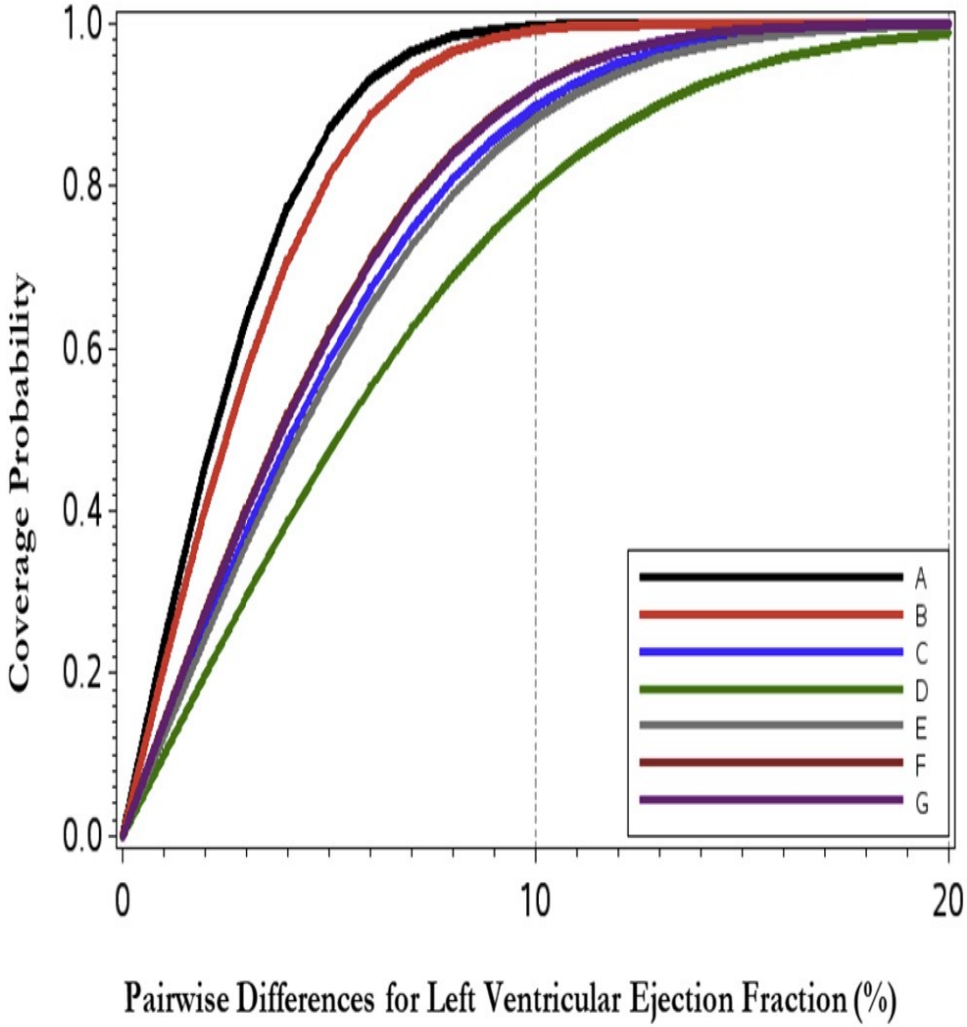
	Volumetric MRI	Biplane MRI	Volumetric Echocardiography	Biplane Echocardiography
Interobserver				
Variability (%)	3.6	13.4	8.3	17.8
Mean ± SD (%)	0.5 ± 1.5	−1.4 ± 5.9	−0.1 ± 3.8	1.3 ± 8.8
SEE	1.6	4.3	3.7	9.2
r^2	0.99	0.94	0.96	0.82
Intraobserver				
Variability (%)	5.1	13.0	6.9	13.4
Mean ± SD (%)	−1.1 ± 2.1	−2.0 ± 5.6	−0.4 ± 3.1	−0.9 ± 6.8
SEE	2.1	5.4	3.3	6.7
r^2	0.99	0.91	0.97	0.90

M. L. Chuang, et al J ACC 2000;35:477– 84



EF SEM 95% CI	0.049 (0.045–0.054)	0.058 (0.053–0.065)*	0.059 (0.054–0.065)	0.065 (0.058–0.072)	0.028 (0.025–0.031)	0.051 (0.046–0.057)*

P. Thavendiranathan et al. J Am Coll Cardiol 2013;61:77–84

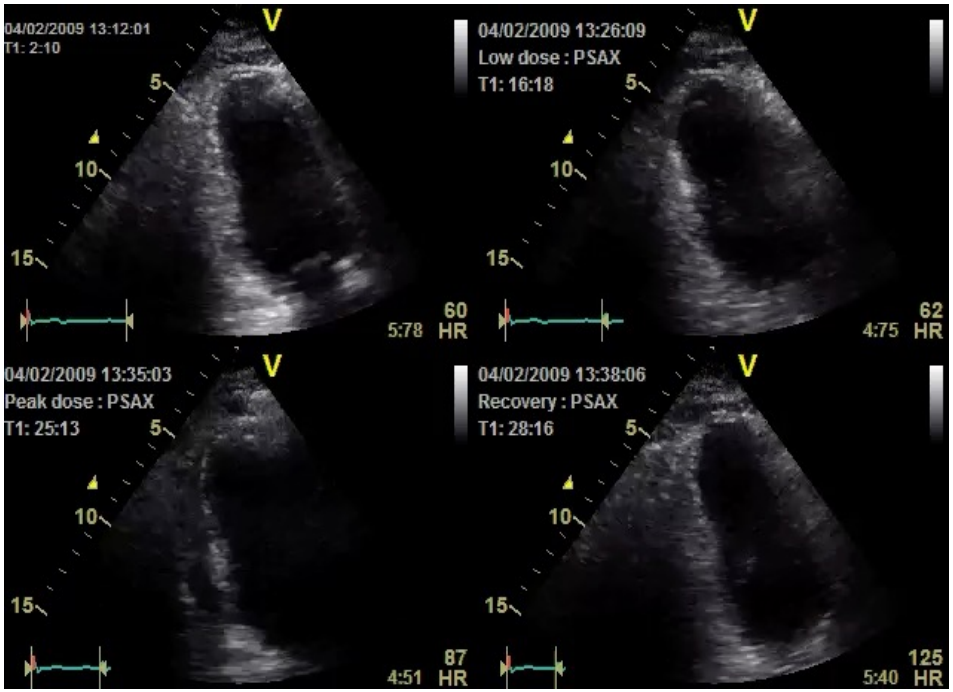


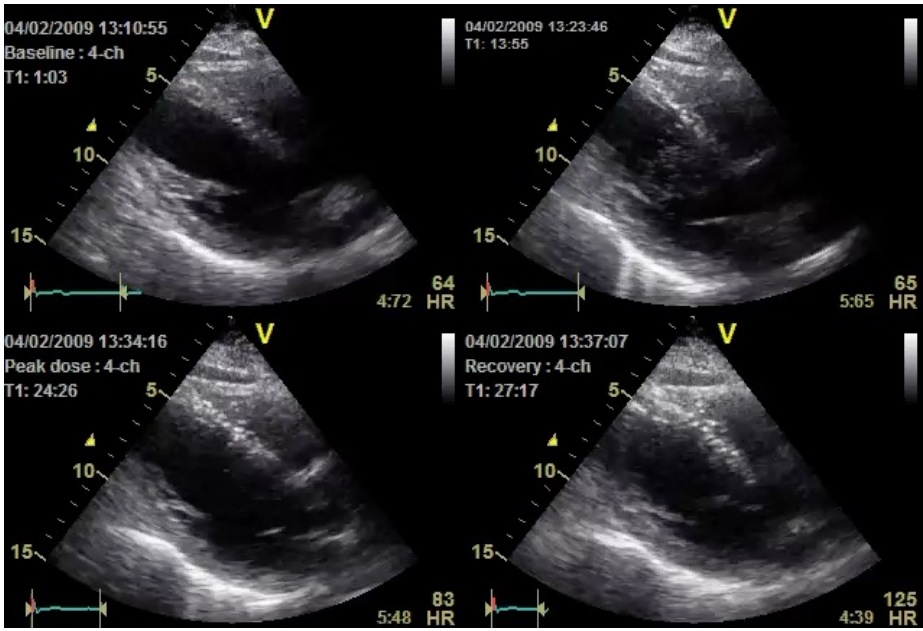
A.L. Crowley et al. J Am Soc Echocardiogr 2016;29:1144-54.)

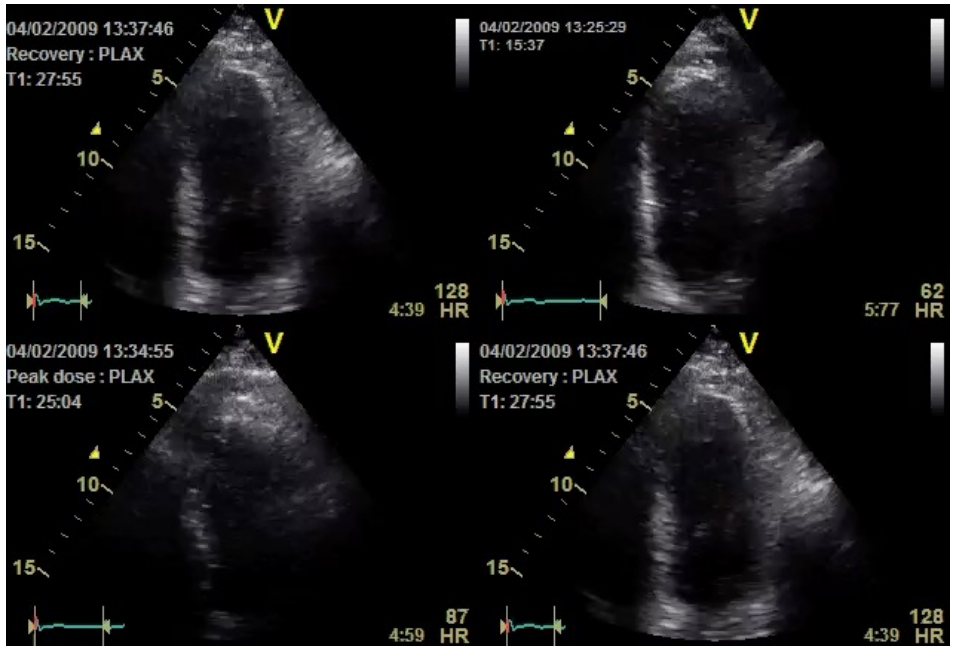
Conclusion II

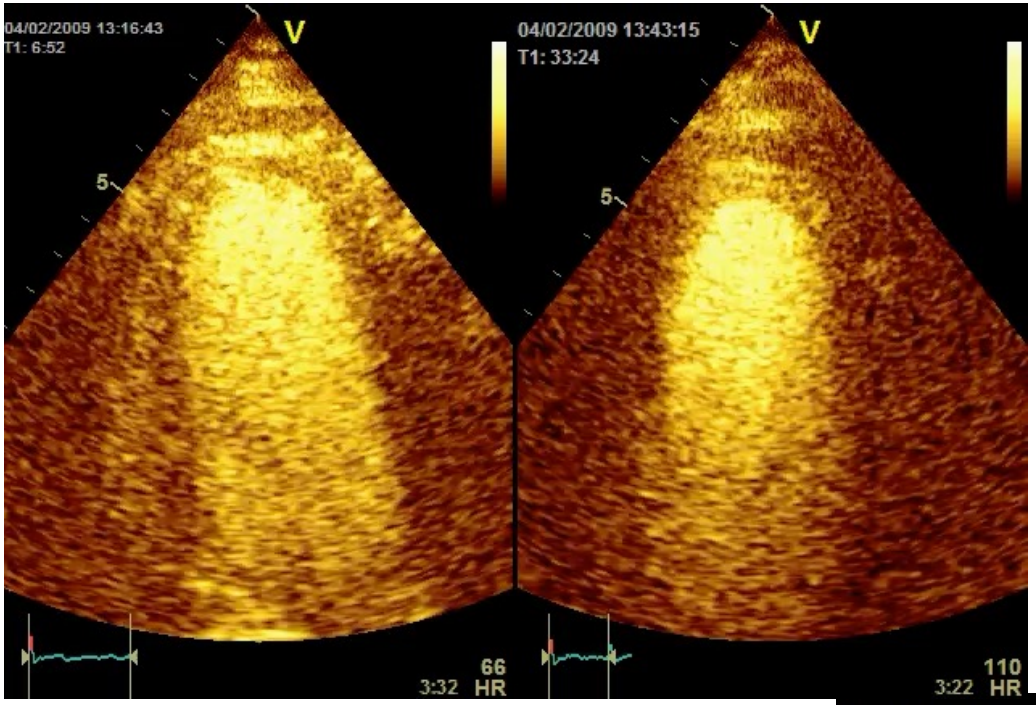
1. Intra and inter observer variability relatively unreliable
2. Unable to display complex anatomical structures - It hides structural functional heart abnormalities
3. Systolic and diastolic dysfunction is often present in HEpEF or HErEF

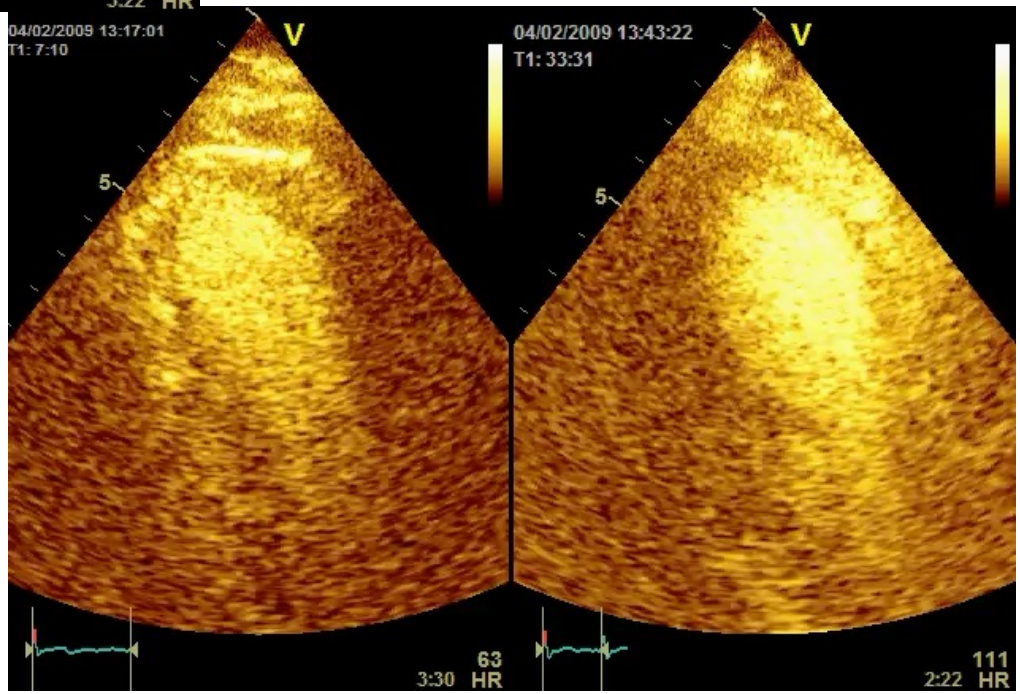
New Techniques
3D Echo


04/02/2009 13:12:01
T1: 2:10
5:78
60 HR
04/02/2009 13:26:09
Low dose : PSAX
T1: 16:18
4:75
62 HR
04/02/2009 13:35:03
Peak dose : PSAX
T1: 25:13
4:51
87 HR
04/02/2009 13:38:06
Recovery : PSAX
T1: 28:16
5:40
125 HR

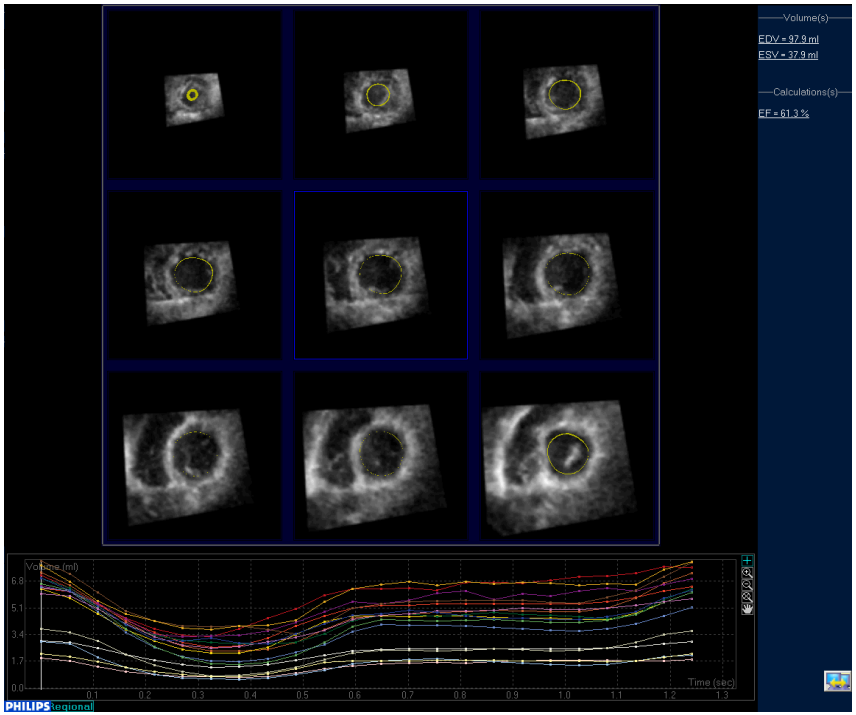

04/02/2009 13:10:55
Baseline : 4-ch
T1: 1:03
4:72
64 HR
04/02/2009 13:23:46
T1: 13:55
5:65
65 HR
04/02/2009 13:34:16
Peak dose : 4-ch
T1: 24:26
5:48
83 HR
04/02/2009 13:37:07
Recovery : 4-ch
T1: 27:17
4:39
125 HR

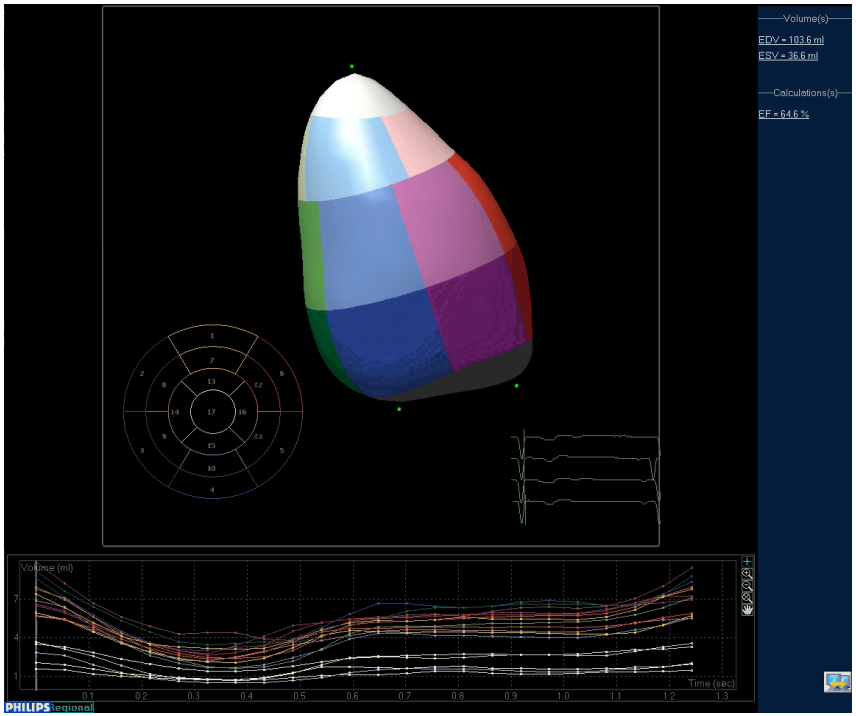

04/02/2009 13:37:46
Recovery : PLAX
T1: 27:55
4:39
128 HR
04/02/2009 13:25:29
T1: 15:37
5:77
62 HR
04/02/2009 13:34:55
Peak dose : PLAX
T1: 25:04
4:59
87 HR
04/02/2009 13:37:46
Recovery : PLAX
T1: 27:55
4:39
128 HR


04/02/2009 13:16:43
T1: 6:52
V
5
66
3:32 HR
04/02/2009 13:43:15
T1: 33:24
V
5
110
3:22 HR

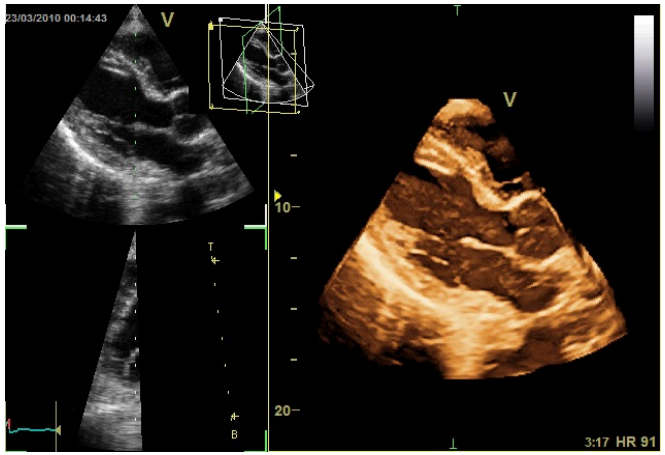

04/02/2009 13:17:01
T1: 7:10
V
5
63
3:30 HR
04/02/2009 13:43:22
T1: 33:31
V
5
111
2:22 HR

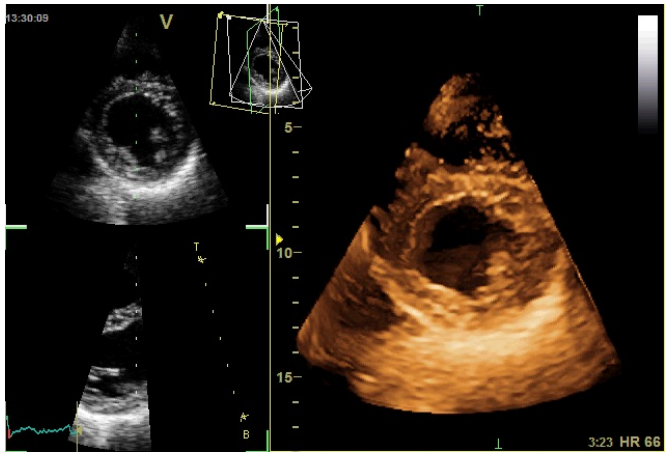
New Techniques
3D Echo

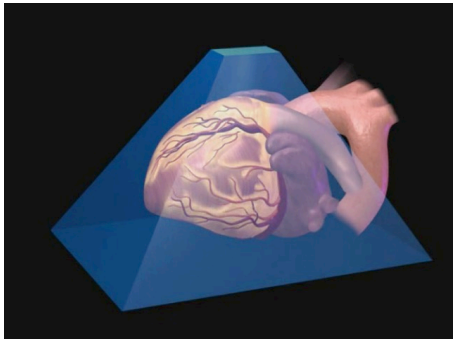


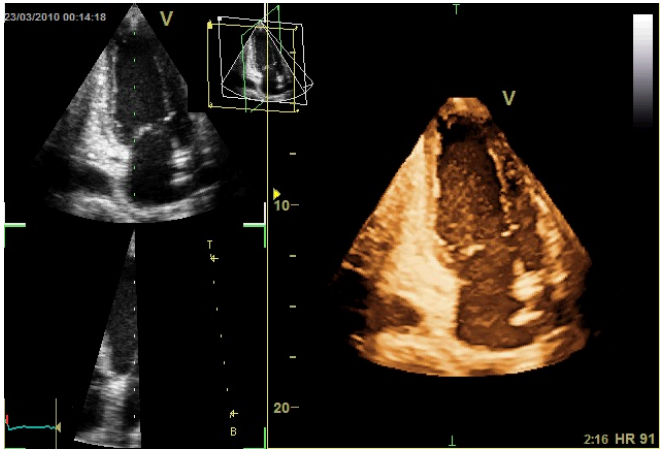


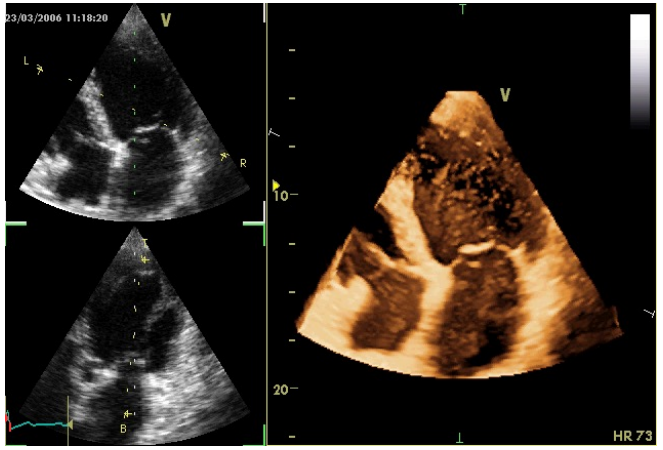
New Techniques
3D Echo



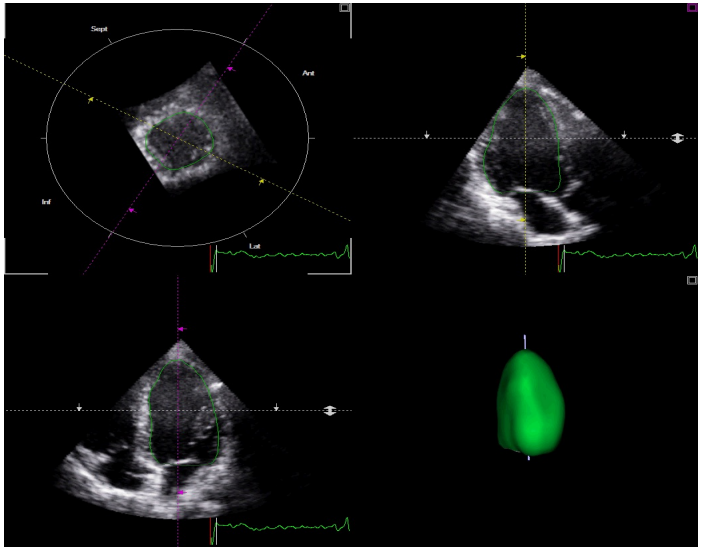




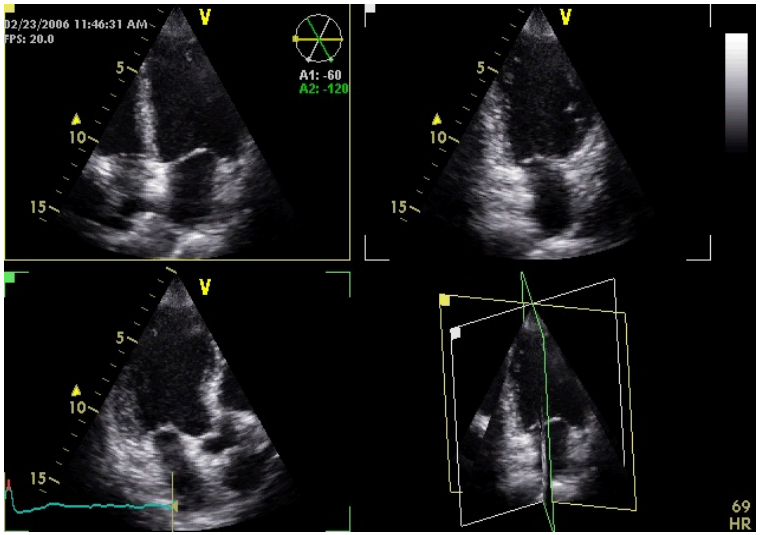


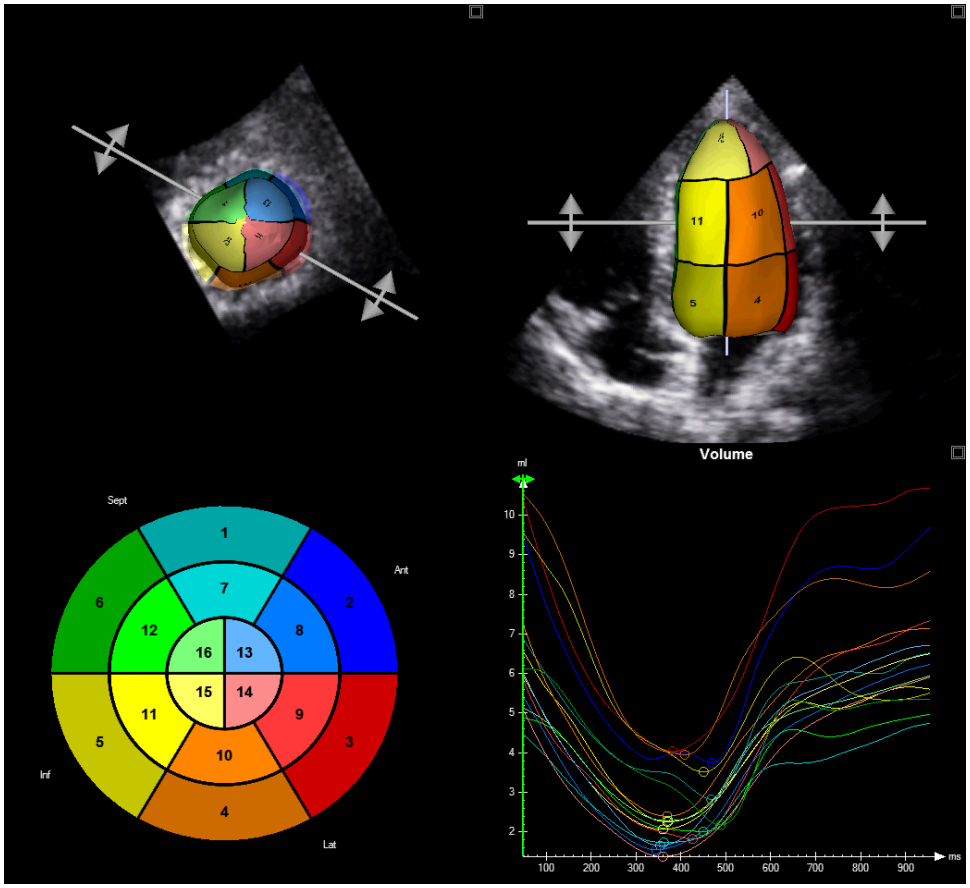


Estimation of the volume and functionality



EDV	109.44 ml
ESV	41.98 ml
SV	67.46 ml
EF	61.64 %
SDI16	49 ms



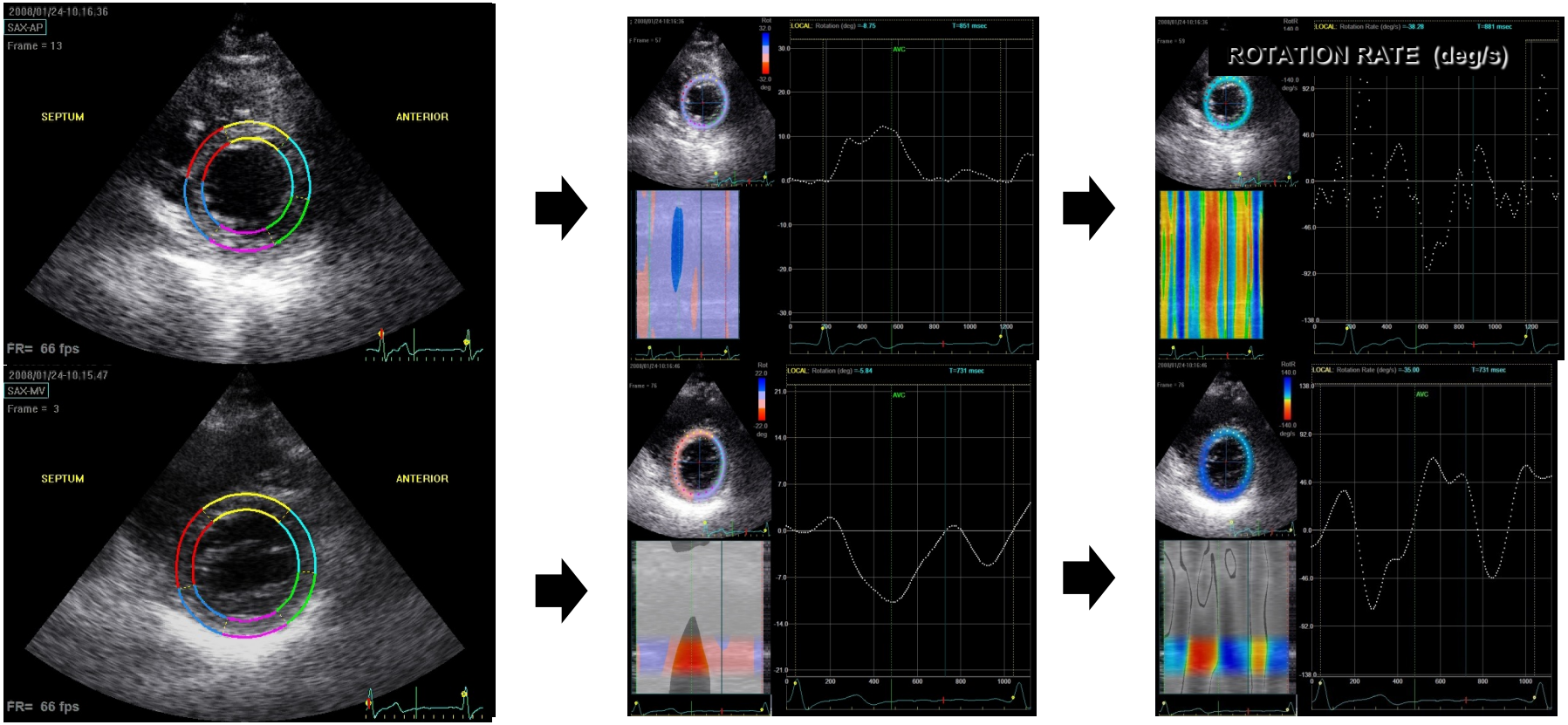


Left Ventricular Rotation

Counterclockwise movement

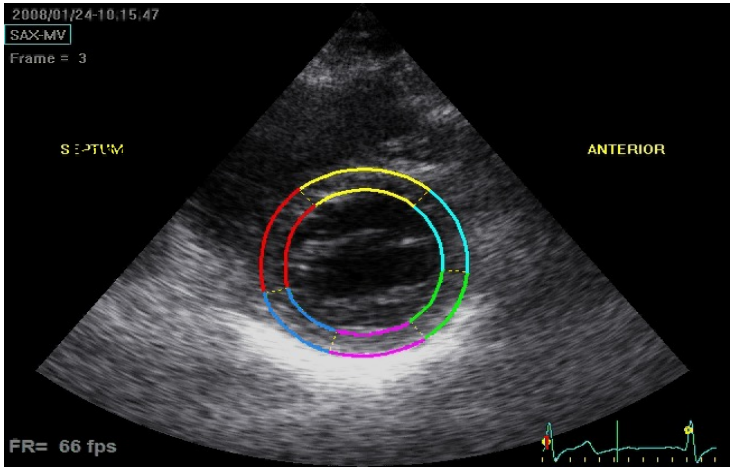
Top (A)

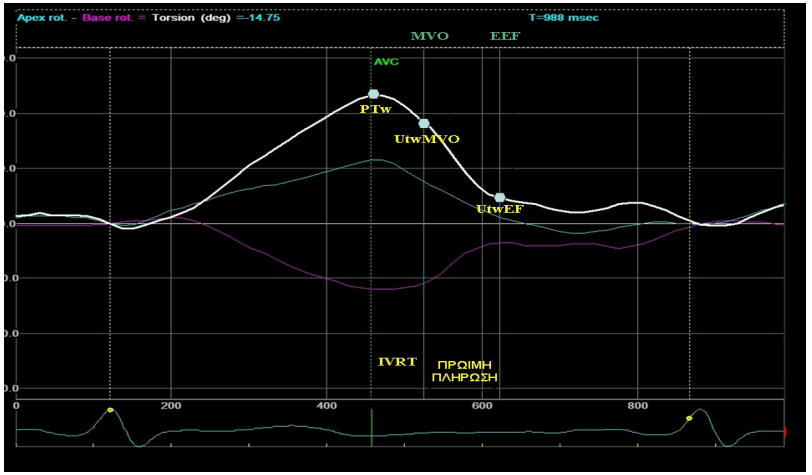
Base (B)

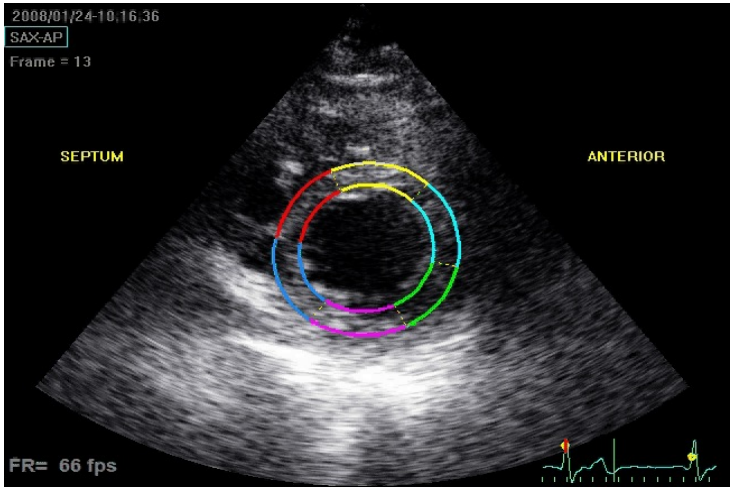


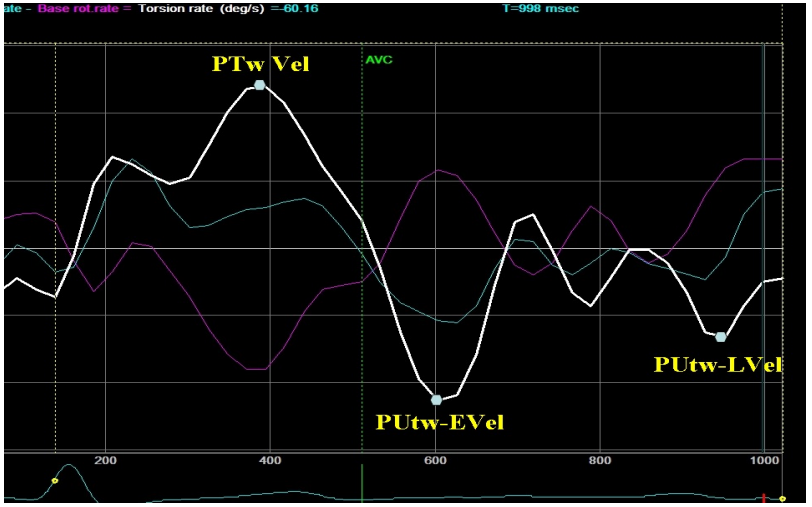
Clockwise movement

From Rotation to Torsion



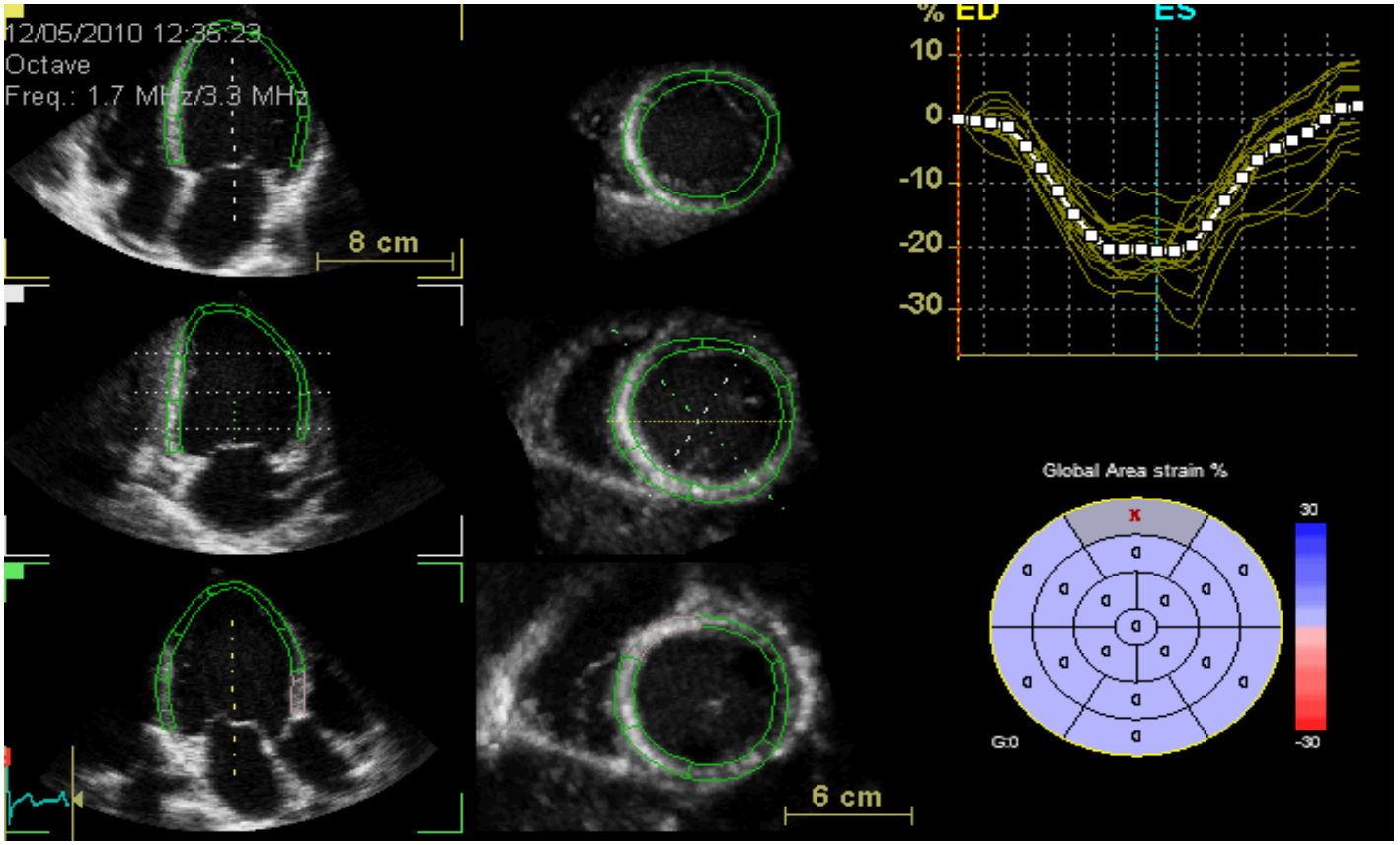






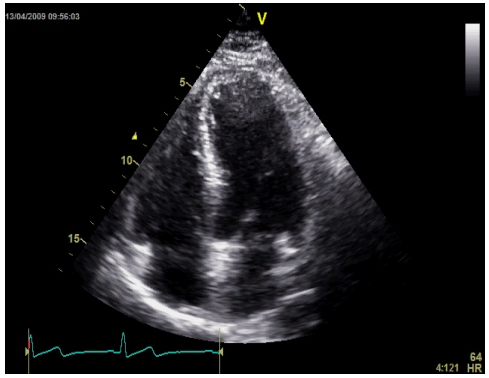
Helical cardiac motion where the top is directed counterclockwise and the base clockwise with longitudinal shortening and simultaneous transverse movement (thickening) of their walls

3D STRAIN
(Wall Motion Tracking)

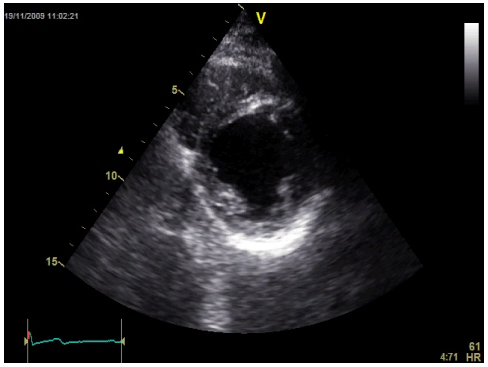


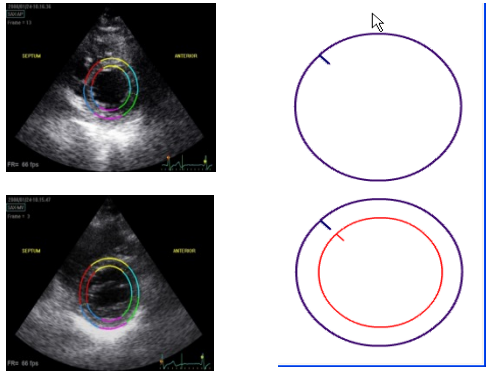
Time-strain curves from each of 16 segments

Longitudinal shortening



Cicumferential Radial shortening





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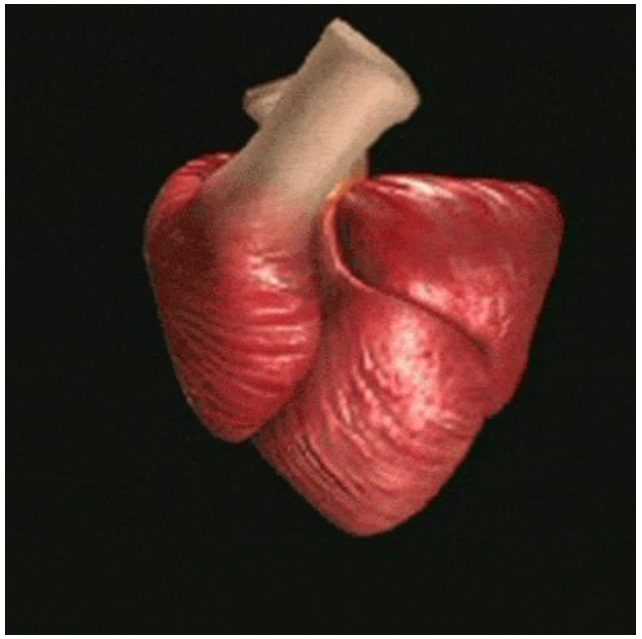
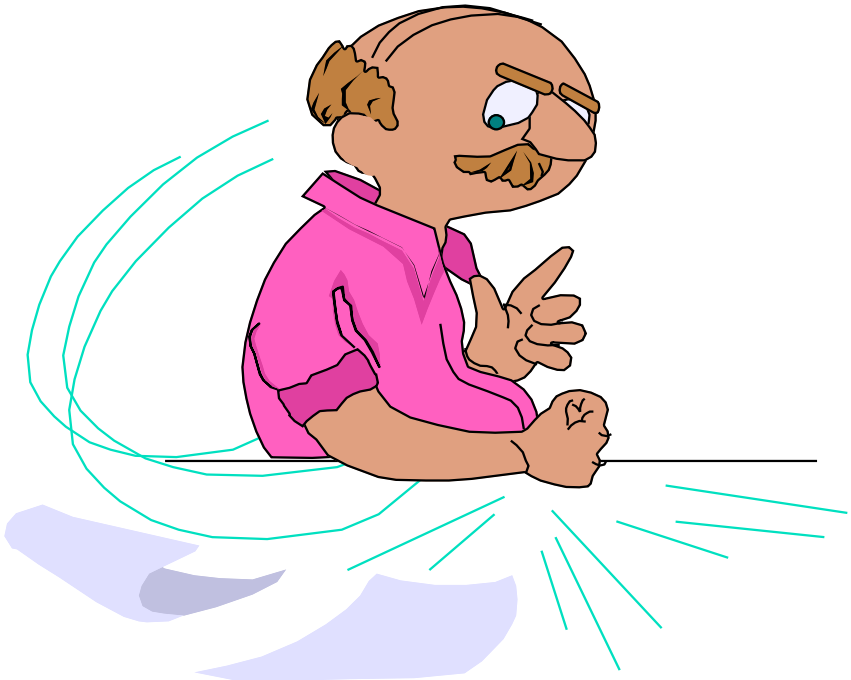
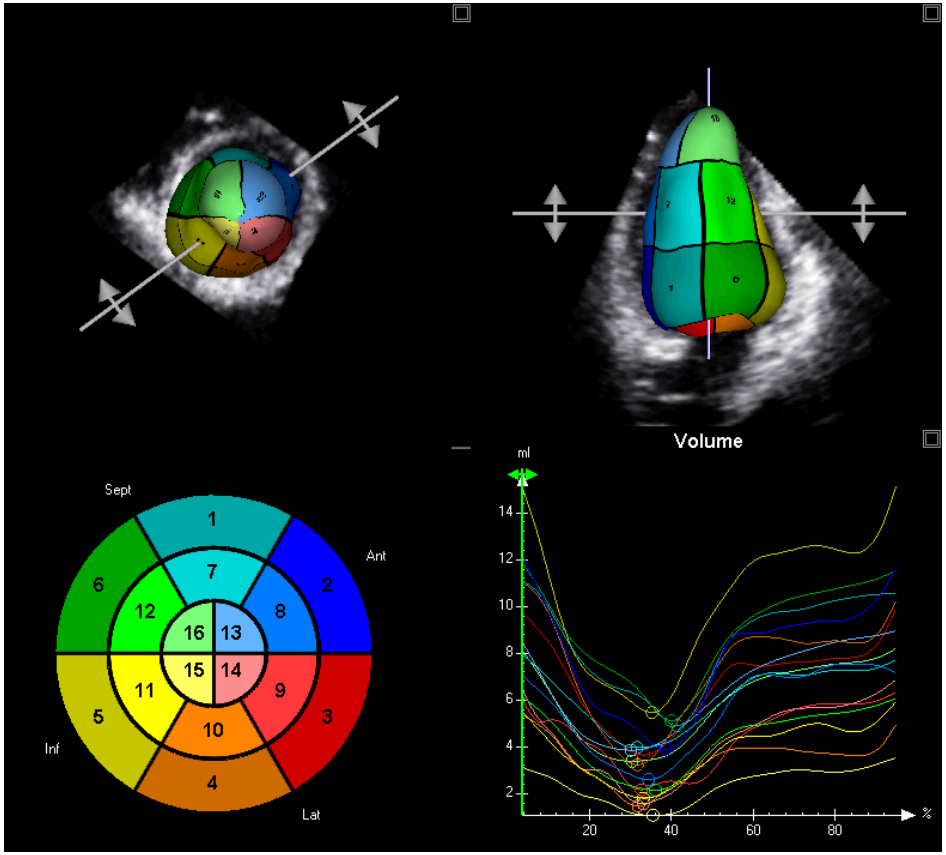




4D Quantification of LV function

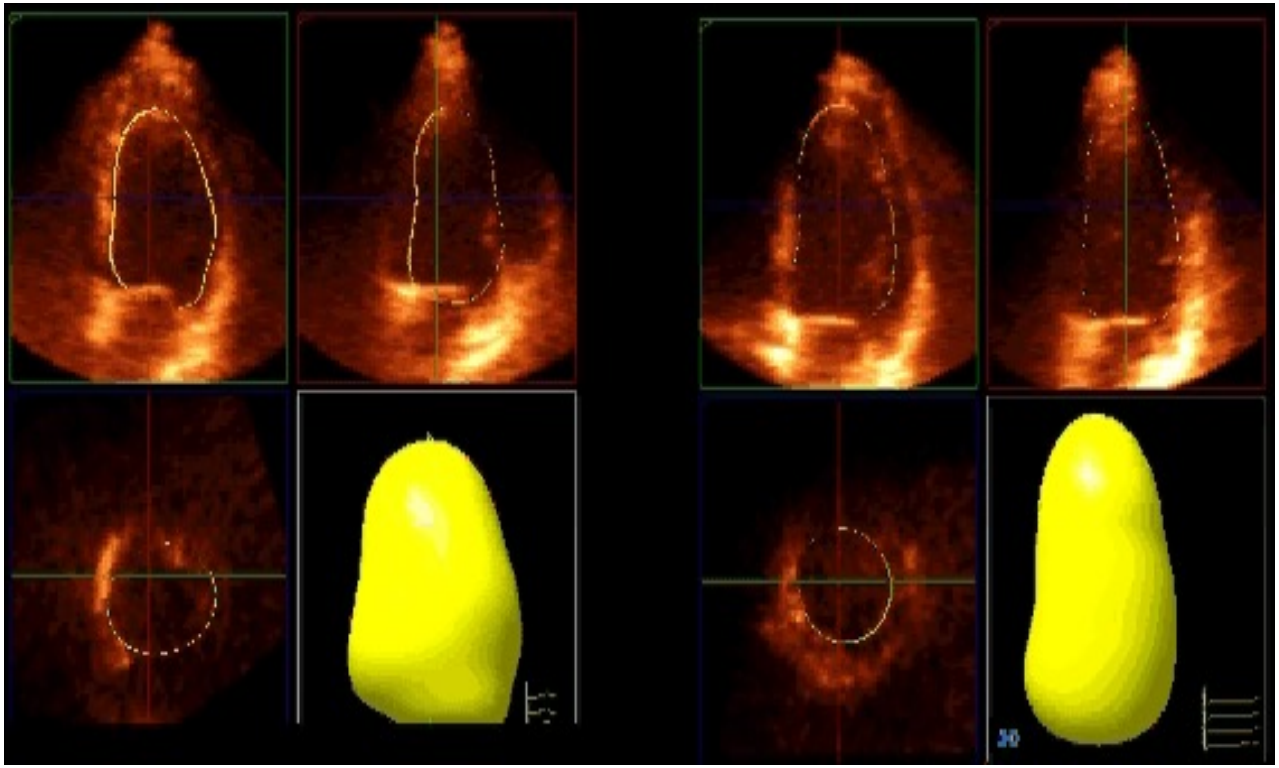
		Mean difference ± SD vs MRI	
		RT3DE	**2DE**
Jenkins 2004	EDV (ml)	-4±9	-54±33
	ESV (ml)	-3±18	-28±28
	EF (%)	0±7	-1±13
Caiani 2005	EDV (ml)	-4±29	-23±86
	ESV (ml)	-4±33	-19±60
	EF (%)	-8±14	+4±16
Jacobs 2006	EDV (ml)	-14±17	-23±29
	ESV (ml)	-7±16	-15±24
	EF (%)	-1±6	1±9



LV Remodeling Index (LVRi)
In Various Conditions by RT-3D Echo

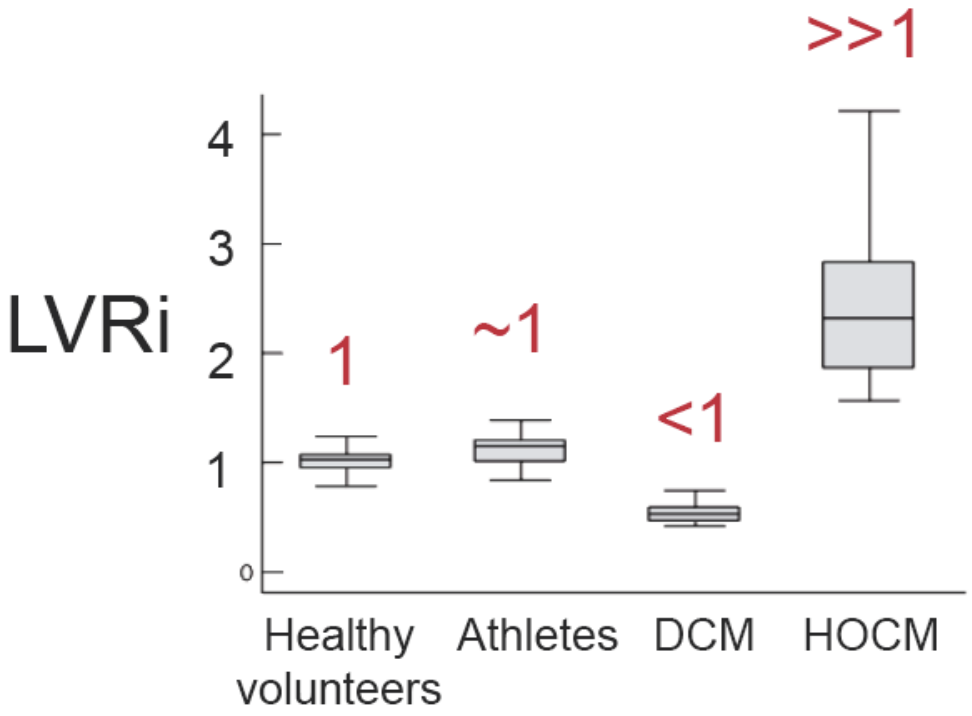
220 subjects:
- normal athletes
- DCM HOCM

3D:
Of line endocardial and epicardial border tracing



Nagueh S, et al. J Am Soc Echocardiogr 2016.

RT-3D Echo-Derived LVRi in Various Pathophysiological Conditions



LV Remodelling Index

$$LVRi = \frac{\text{LV mass}}{\text{LVEDV}}$$

Nagueh S, et al. J Am Soc Echocardiogr 2016.

Clinical Decision Making by 3D Echo

- *220 pts, 3 hospitals*
- *3D: 83% feasible; add time 5.4±2 min*

Clinically-relevant measurement thresholds:

- LVESV >50ml/m^2
  (surgery for regurgitant valve lesions)
- LVESV >30ml/m^2
  (prognosis after MI)
- LVEF <35%
  (indication for ICD)
- LVEF <40%
  (indication for HF treatment)

Reallocation by 3D
8.5%, (5/59)
12.4%, (13/105)
17.5%, (11/63)
16.1%, (13/81)

Hare JL et al, Heart 2007

Estimation of Diastolic and Systolic function

HEART FAILURE

Left Ventricular Mechanics in Idiopathic Dilated Cardiomyopathy: Systolic-Diastolic Coupling and Torsion

Jaroslav Meluzin, MD, PhD, FESC, Lenka Spinarova, MD, PhD, FESC, Petr Hude, MD, Jan Krejci, MD, Hana Poloczkova, MD, Helena Podrouzkova, MD, Martin Pesl, MD, Marek Orban, MD, Ladislav Dusek, MD, PhD, and Josef Korinek, MD, PhD, *Brno and Prague, Czech Republic; Rochester, Minnesota*

Background: In idiopathic dilated cardiomyopathy (IDC), myocardial deformational parameters and their mutual relationships remain incompletely characterized.

Methods: Thirty-seven patients with IDC underwent two-dimensional speckle-tracking echocardiography (2D-STE) to assess left ventricular rotation, torsion, and longitudinal, circumferential, and radial systolic and diastolic strains and strain rates. Additionally, 2D-STE was performed in 14 controls.

Results: All deformational parameters on 2D-STE were significantly lower in patients with IDC compared with controls. Seven patients exhibited opposite basal (positive, counterclockwise) and 11 patients exhibited opposite apical (negative, clockwise) rotation at end-systole. Circumferential, radial, and longitudinal early diastolic strain rates were correlated most strongly with the corresponding spatial components of systolic deformation.

Conclusion: In patients IDC, all torsional, systolic, and diastolic deformational parameters were decreased. Corresponding three-dimensional components of systolic and diastolic deformations were closely coupled. Considerable variation in the direction of basal and apical rotation exists in a subset of patients with IDC. (J Am Soc Echocardiogr 2009;22:486-493.)

Keywords: Left ventricular rotation, Torsion, Two-dimensional strain echocardiography

EUROPEAN SOCIETY OF CARDIOLOGY®

European Journal of Heart Failure (2009) **11**, 945–951
doi:10.1093/eurjhf/hfp124

Left ventricular remodelling and torsional dynamics in dilated cardiomyopathy: reversed apical rotation as a marker of disease severity†

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[1]'Carol Davila' University of Medicine and Pharmacy, 'Prof. Dr C. C. Iliescu' Institute of Cardiovascular Diseases, Sos. Fundeni 258, sector 2, 022328 Bucharest, Romania; and [2]Prof. Dr C. C. Iliescu' Institute of Cardiovascular Diseases, Bucharest, Romania

Received 18 June 2009; revised 29 July 2009; accepted 7 August 2009

Aims Decreased left ventricular (LV) rotation and torsion and even reversed systolic apical rotation have been described in patients with dilated cardiomyopathy (DCM). We sought to test in patients with DCM whether reversed apical rotation with loss of LV torsion is related to the extent of LV remodelling and to the severity of LV dysfunction.

Methods and results Fifty consecutive patients with DCM (aged 49 ± 13 years) were enrolled prospectively. Forty-seven healthy volunteers served as controls. All subjects underwent clinical examination, 12-lead electrocardiography, and a comprehensive echocardiogram. Basal and apical LV rotation and LV torsion were quantified by speckle tracking echocardiography. Left ventricular systolic rotation and torsion were reduced in patients, compared with controls ($P < 0.001$). Normally directed (counterclockwise) apical rotation was found in 24 patients (group 1), whereas 26 had reversed (clockwise) apical rotation (group 2). Patients in group 2 had larger LV volume, increased LV sphericity ($P \leq 0.02$), more severe systolic dysfunction (ejection fraction 26 ± 7 vs. 33 ± 12%), and higher filling pressures (E/E' ratio 19 ± 10 vs. 14 ± 6; $P < 0.05$). The main correlates of LV apical rotation were LV volume, sphericity index, and QRS duration.

Conclusion Reversed apical rotation and loss of LV torsion in patients with DCM is associated with significant LV remodelling, increased electrical dyssynchrony, reduced systolic function, and increased filling pressures, indicating a more advanced disease stage.

Keywords Dilated cardiomyopathy • Left ventricular remodelling • Apical rotation • Left ventricular torsion • Speckle tracking echocardiography

Estimation of Diastolic and Systolic function

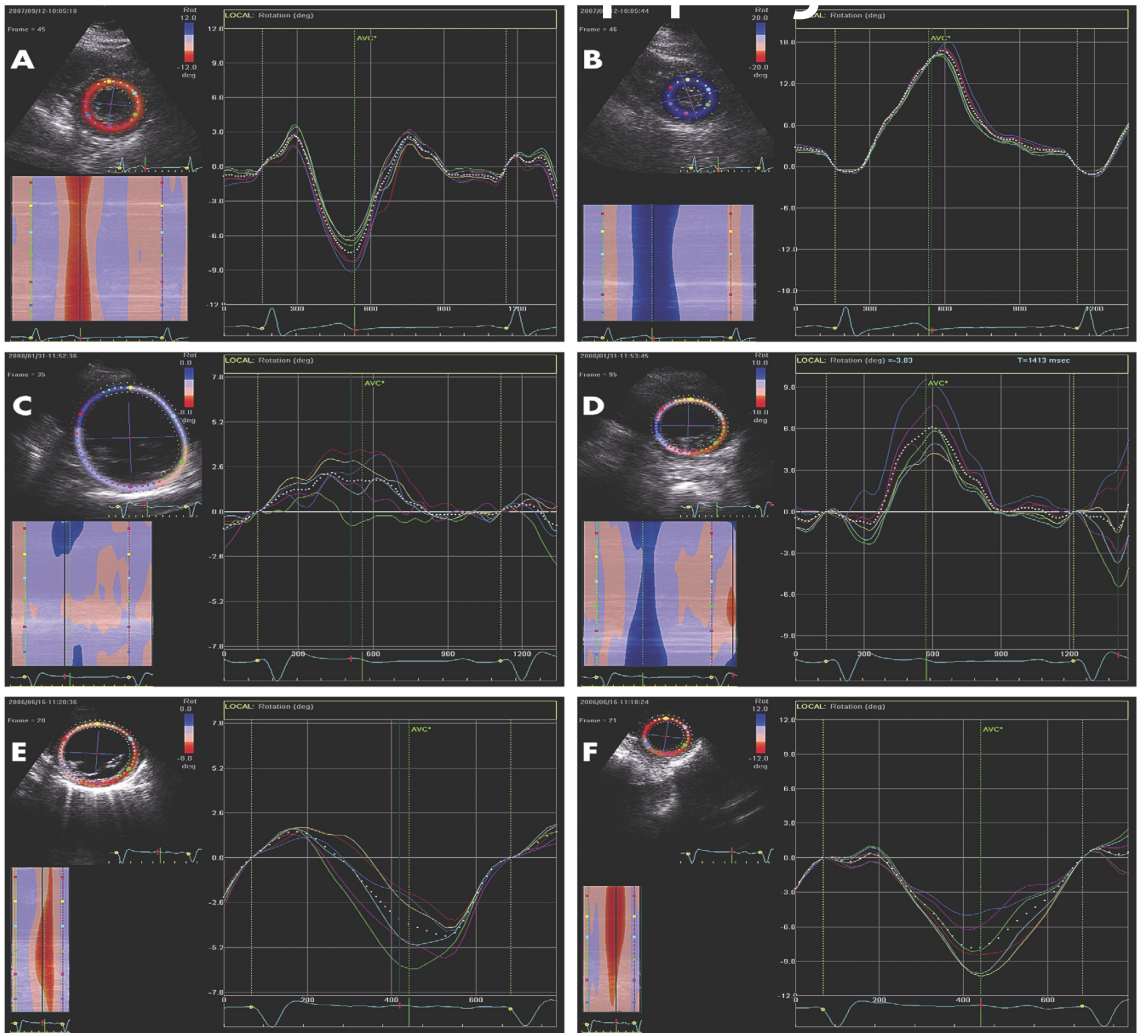


Figure 1 Examples of normal and opposite basal and apical rotation curves obtained from a healthy volunteer and from two patients with IDC. Each color of the deformational curve represents one segment of the left ventricle, and the *dashed white curve* depicts mean rotation of 6 segments. *(Top)* Normal negative (clockwise) late systolic and end-systolic basal rotation **(A)** and normal positive (counterclockwise) late systolic and end-systolic apical rotation **(B)** in a healthy volunteer. *(Middle)* A patient with IDC with opposite (positive) late systolic and end-systolic basal rotation **(C)** and normal positive direction of apical end-systolic rotation **(D)**. *(Bottom)* A patient with IDC with normal negative direction of basal end-systolic rotation **(E)** and opposite (negative) end-systolic apical rotation **(F)**.

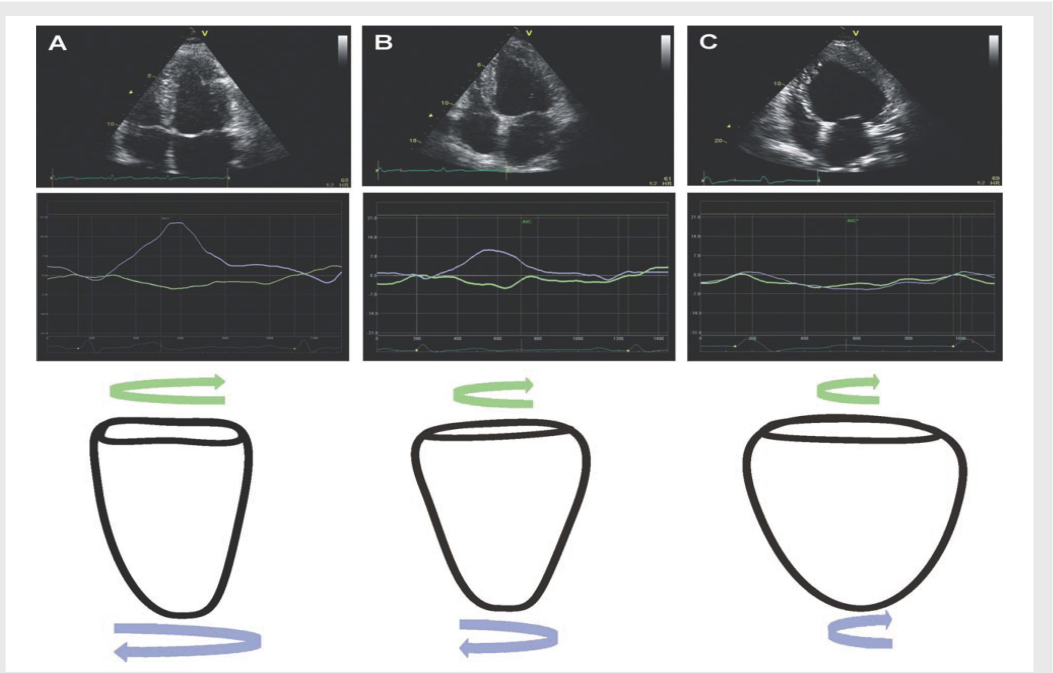
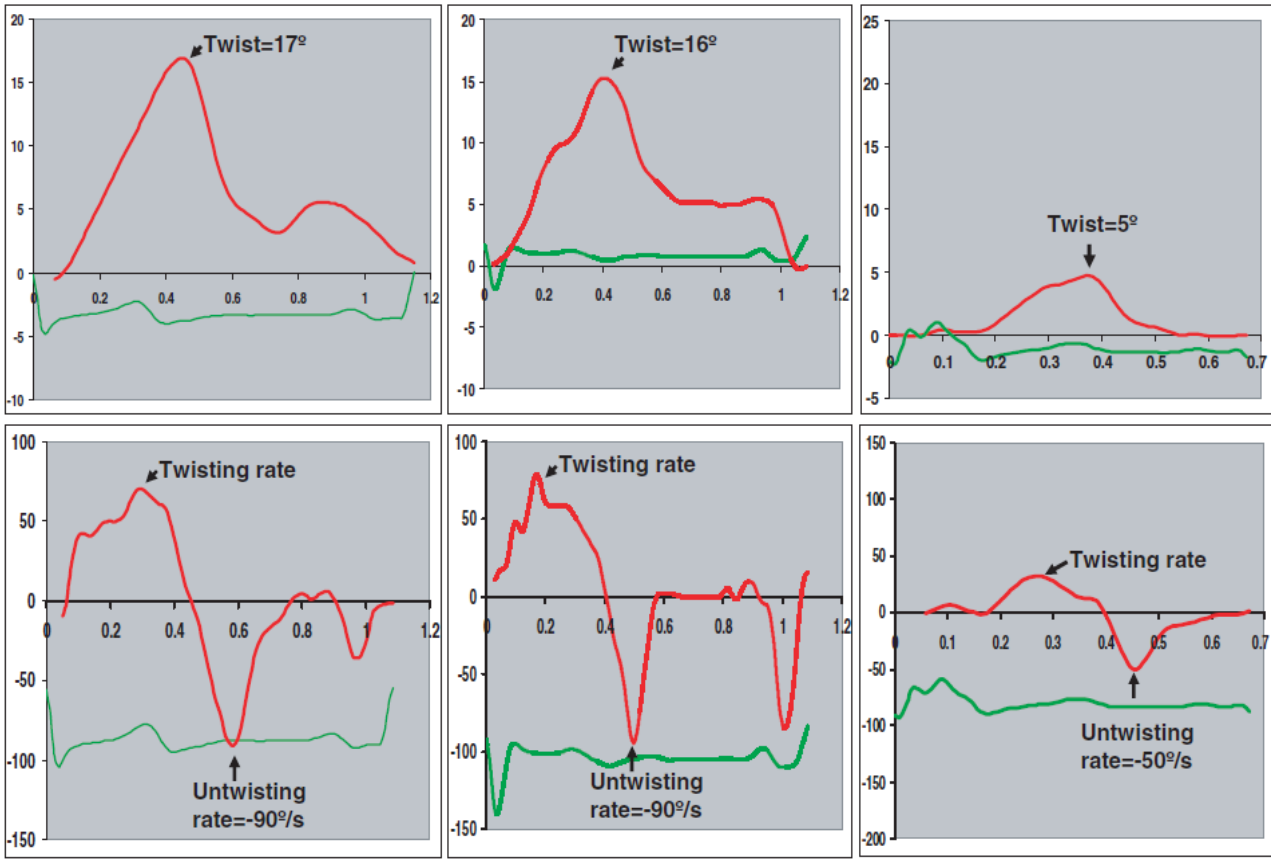


Figure 1 Two-dimensional apical four-chamber views (top), speckle-tracking left ventricular (LV) rotation curves (middle), and schematic representations of LV geometry and rotation (bottom) in a normal subject (*A*); a DCM (+) patient (*B*); and a DCM (−) patient (*C*). Top row: progressive increase in LV dimensions and sphericity, from normal (A) to DCM with severe LV remodelling (C). Middle row: (A) normal LV apical (blue) and basal (green) rotation curves; (B) normally directed, but reduced in amplitude LV rotation curves; (C) reversed apical rotation with clockwise (−) motion of both apex and base in the patient with severely dilated LV. Bottom row: changes in LV geometry and LV rotation at basal (green arrow) and apical (blue arrow) levels from normal (A) to mildly dilated (B), and severely dilated LV (C). The amplitude of both basal and apical rotation is reduced but normally directed in the patient with DCM and mildly dilated LV (B). Reversed apical rotation is seen in association with severe LV remodelling (C).

Conclusions

Reversed apical rotation and loss of LV torsion in patients with DCM is associated with significant LV remodelling, altered ventricular geometry, increased electrical dyssynchrony, reduced systolic function, and increased LV filling pressures. These findings identify a subgroup of patients with more advanced disease.

Torsion Behavior

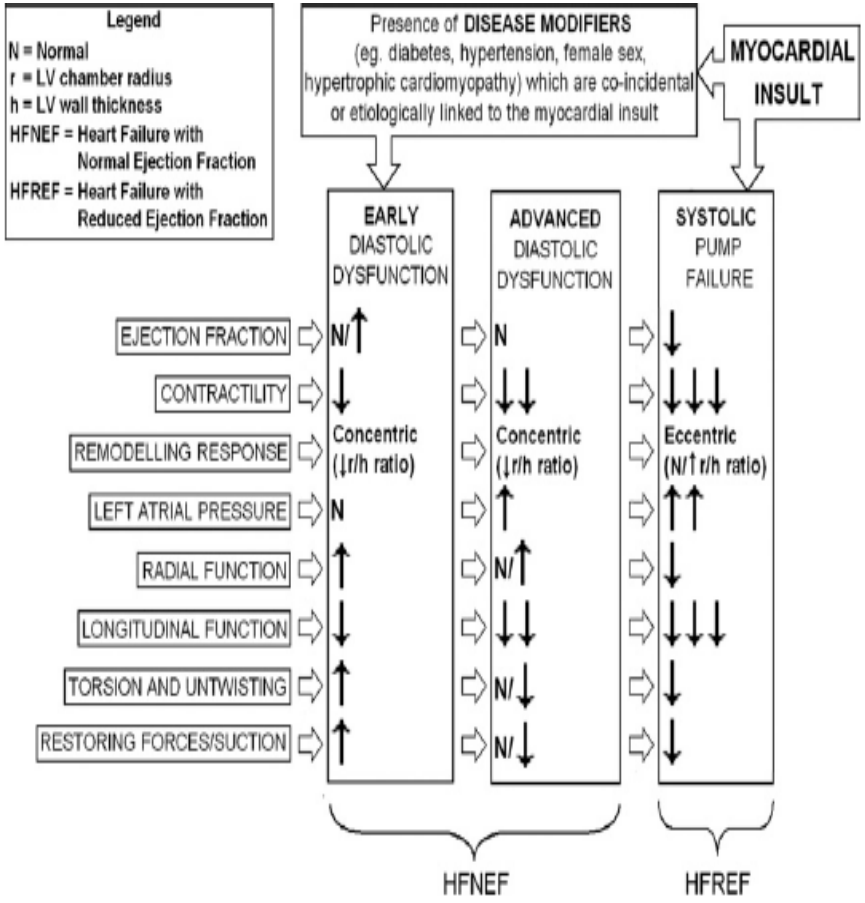


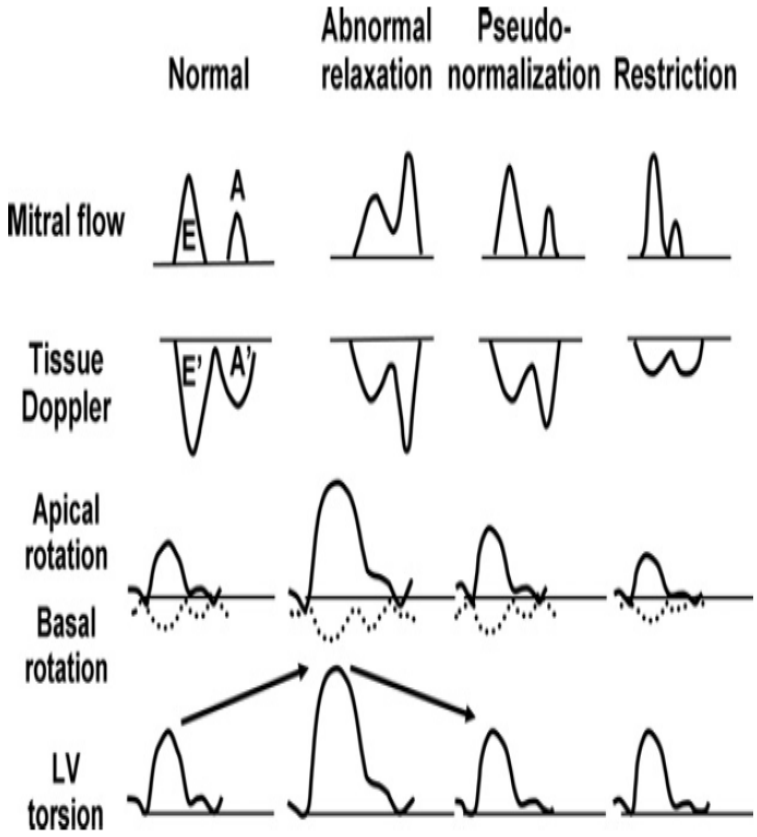
Control

Diastolic Dysfunction
EF 70%

Systolic Dysfunction
EF 33%

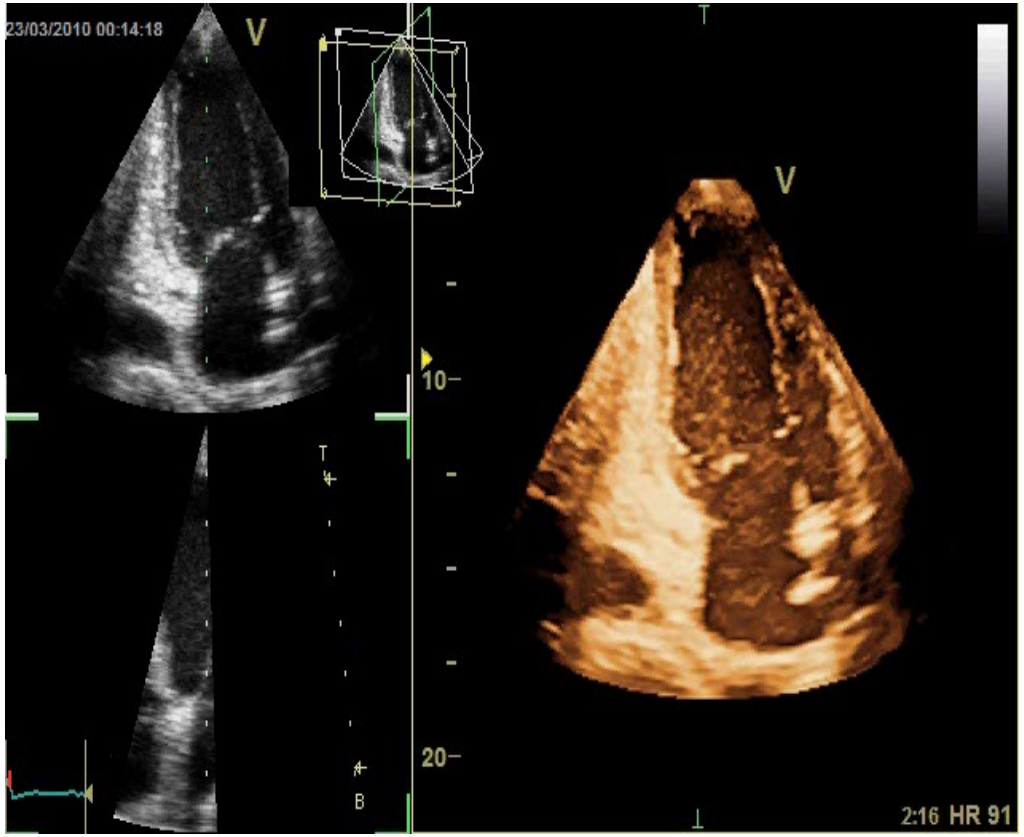
Torsion & HF


Legend
N = Normal
r = LV chamber radius
h = LV wall thickness
HFNEF = Heart Failure with Normal Ejection Fraction
HFREF = Heart Failure with Reduced Ejection Fraction
Presence of DISEASE MODIFIERS (eg. diabetes, hypertension, female sex, hypertrophic cardiomyopathy) which are co-incidental or etiologically linked to the myocardial insult
MYOCARDIAL INSULT
EARLY DIASTOLIC DYSFUNCTION
ADVANCED DIASTOLIC DYSFUNCTION
SYSTOLIC PUMP FAILURE
EJECTION FRACTION
CONTRACTILITY
REMODELLING RESPONSE
LEFT ATRIAL PRESSURE
RADIAL FUNCTION
LONGITUDINAL FUNCTION
TORSION AND UNTWISTING
RESTORING FORCES/SUCTION
N/↑
N
Concentric (↓r/h ratio)
Concentric (↓r/h ratio)
Eccentric (N/↑r/h ratio)
N/↓
HFNEF
HFREF


Normal
Abnormal relaxation
Pseudo-normalization
Restriction
Mitral flow
E
A
Tissue Doppler
E'
A'
Apical rotation
Basal rotation
LV torsion

Criteria to establish or Confirm a Diagnosis of Cardiac Dysfunction Cardioncology

- A decline in LVEF of >5% to <55% with signs and symptoms of HF or a decline in LVEF of >10% to <55% without signs or symptoms of HF.

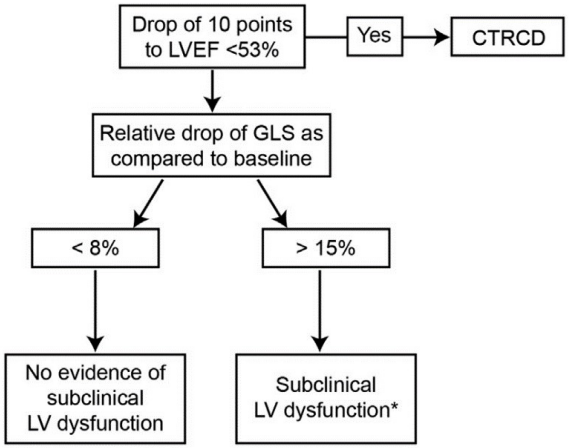


As a decrease in the LVEF of >10 percentage points, to a value <53%. This decrease should

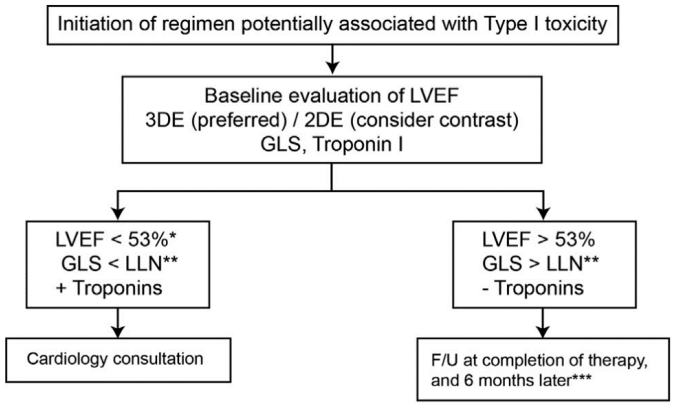
be confirmed by repeated cardiac

imaging (2 to 3 weeks after the baseline).

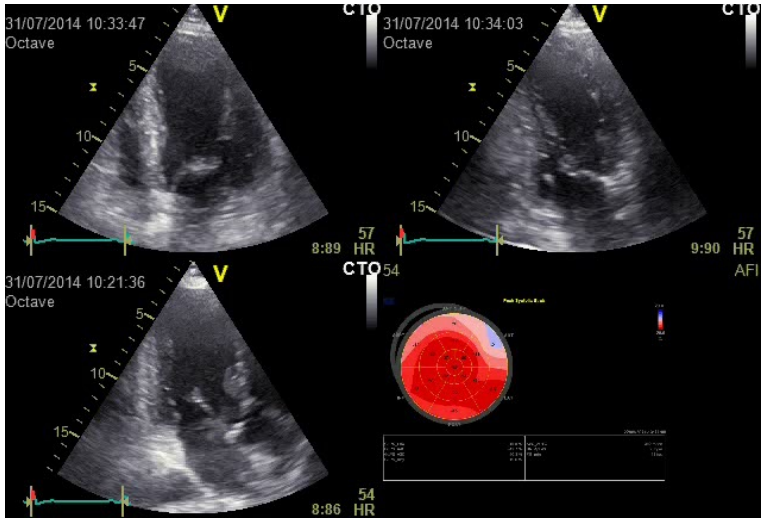
Expert consensus. EHJ – Cardiovascular Imaging 2014; 15: 1063–1093



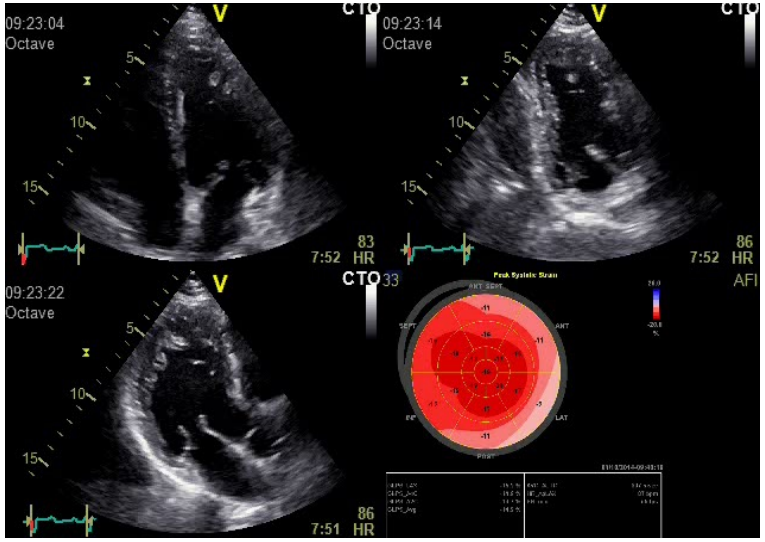
* The data supporting the initiation of cardioprotection for the treatment of subclinical LV dysfunction is limited.



* Consider confirmation with CMR.
** LLN = Lower limit of normal. Please refer to Table 5 for normal GLS values based on vendor, gender and age.
*** If the dose is higher than 240 mg/m² (or its equivalent), recommend measurement of LVEF, GLS and troponin prior to each additional 50 mg/m².



PRE
GLS:-19%



POST
GLS:-13%

1. The neurohormonal overactivity is present in all heart failure phenotypes been highest on the low ejection fraction side and lowest on the high ejection fraction side of spectrum
2. Myocardial fibrosis and left ventricular remodelling are independent of ejection fraction

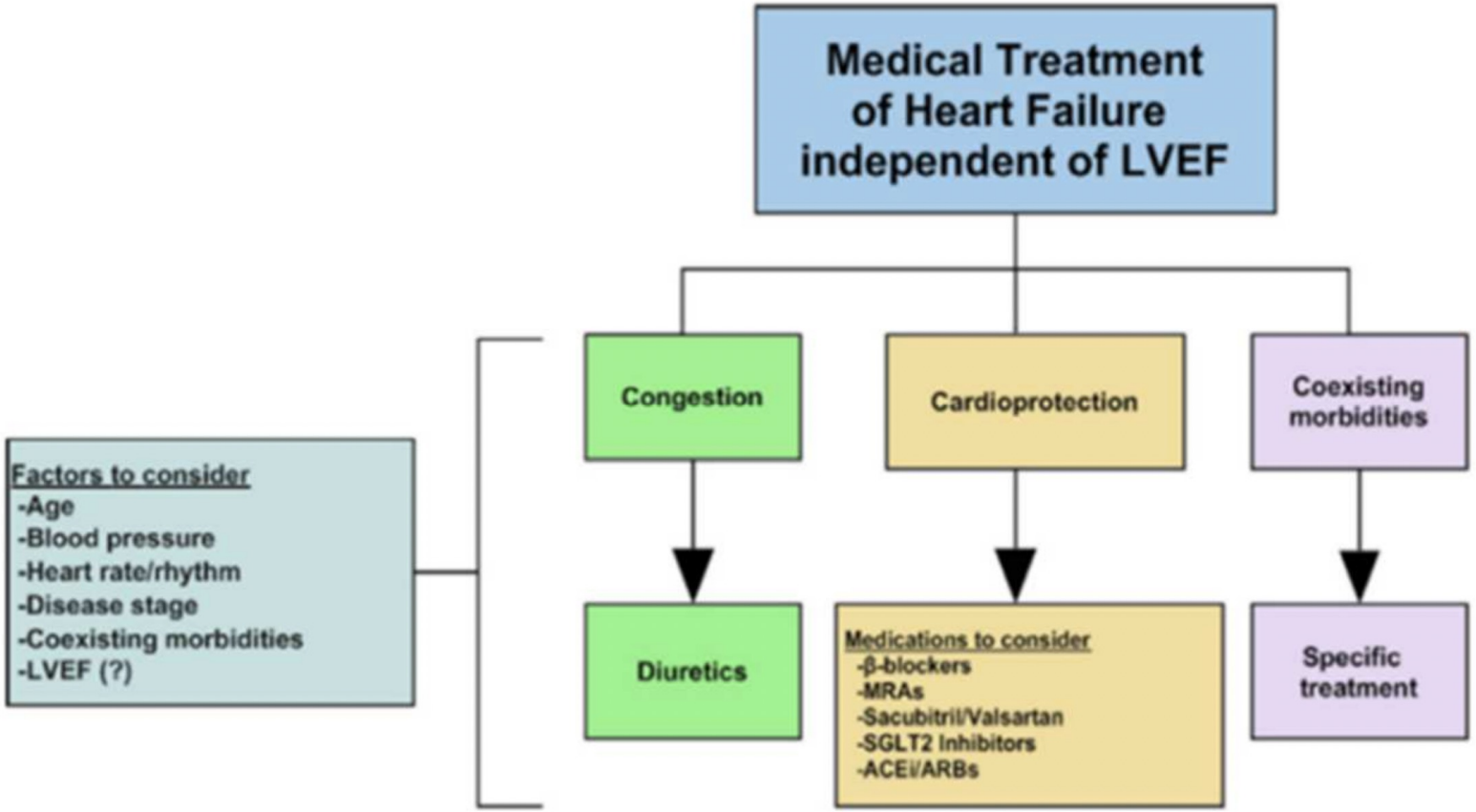
Journal of
Clinical Medicine

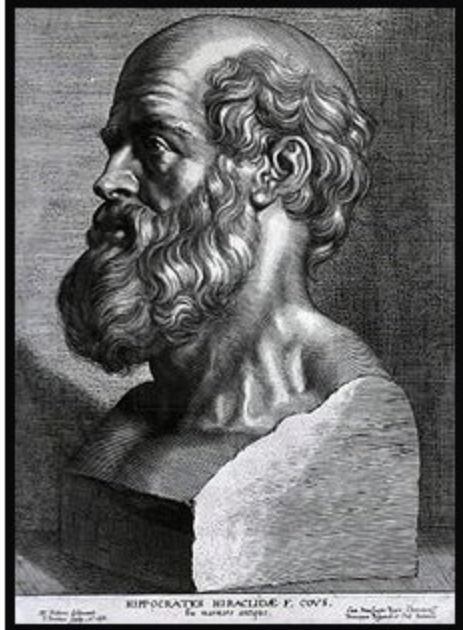
MDPI

Review

Reshaping Treatment of Heart Failure with Preserved Ejection Fraction

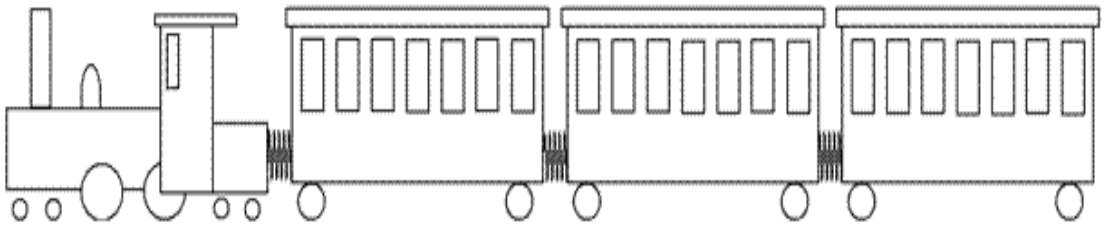
Nikolaos Karamichalakis [1], Andrew Xanthopoulos [2], Filippos Triposkiadis [2,*], Ioannis Paraskevaidis [1] and Elias Tsougos [1]

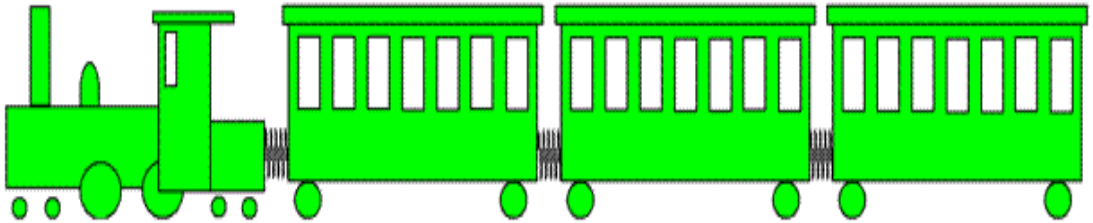


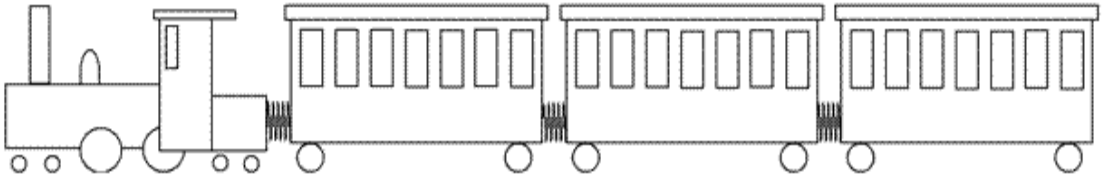


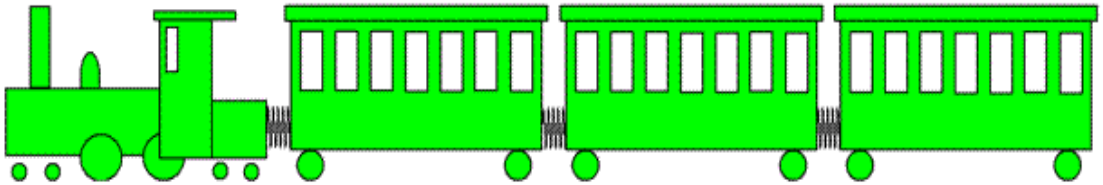
Wherever the art of medicine is loved, there is also a love of humanity.

(Hippocrates)

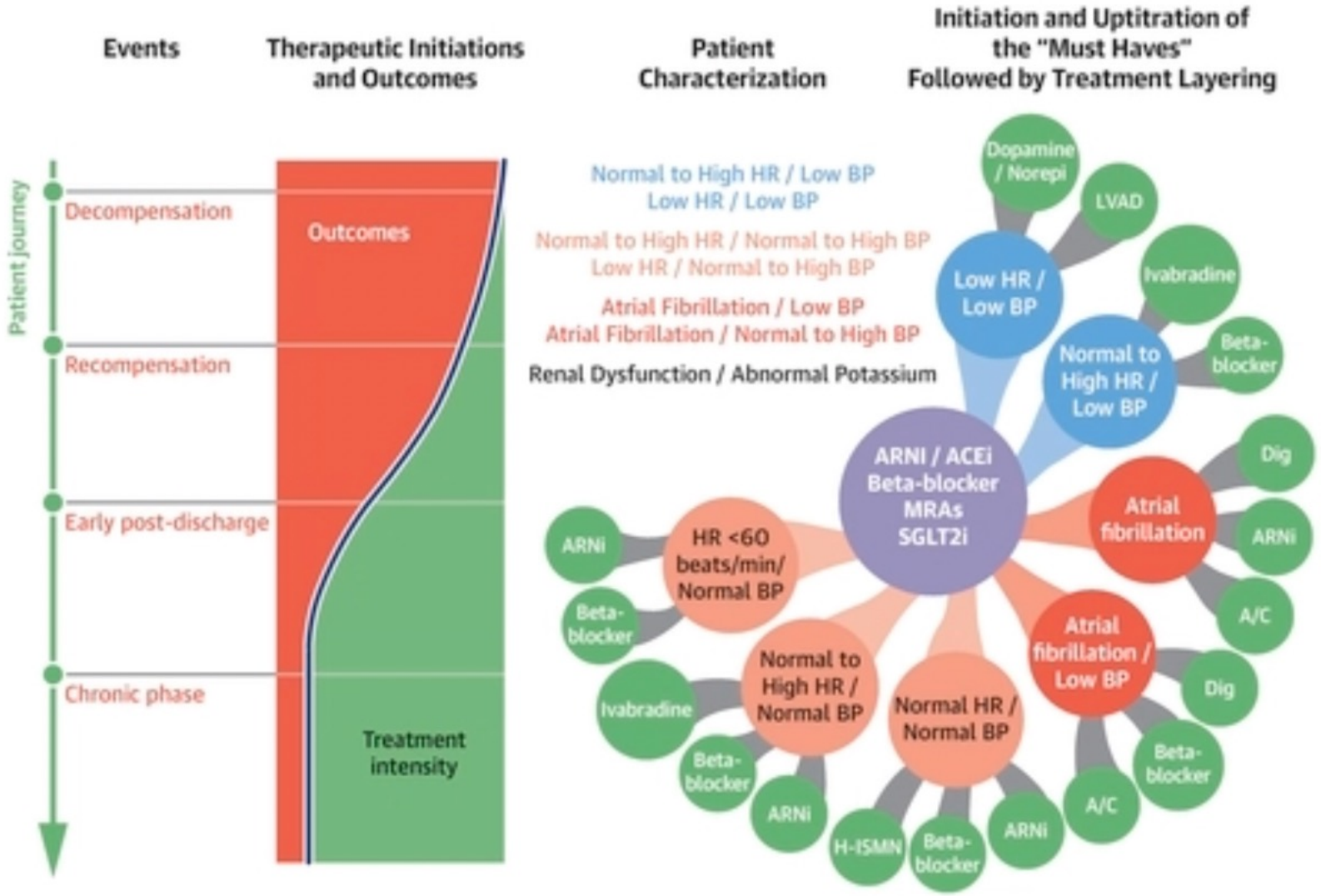




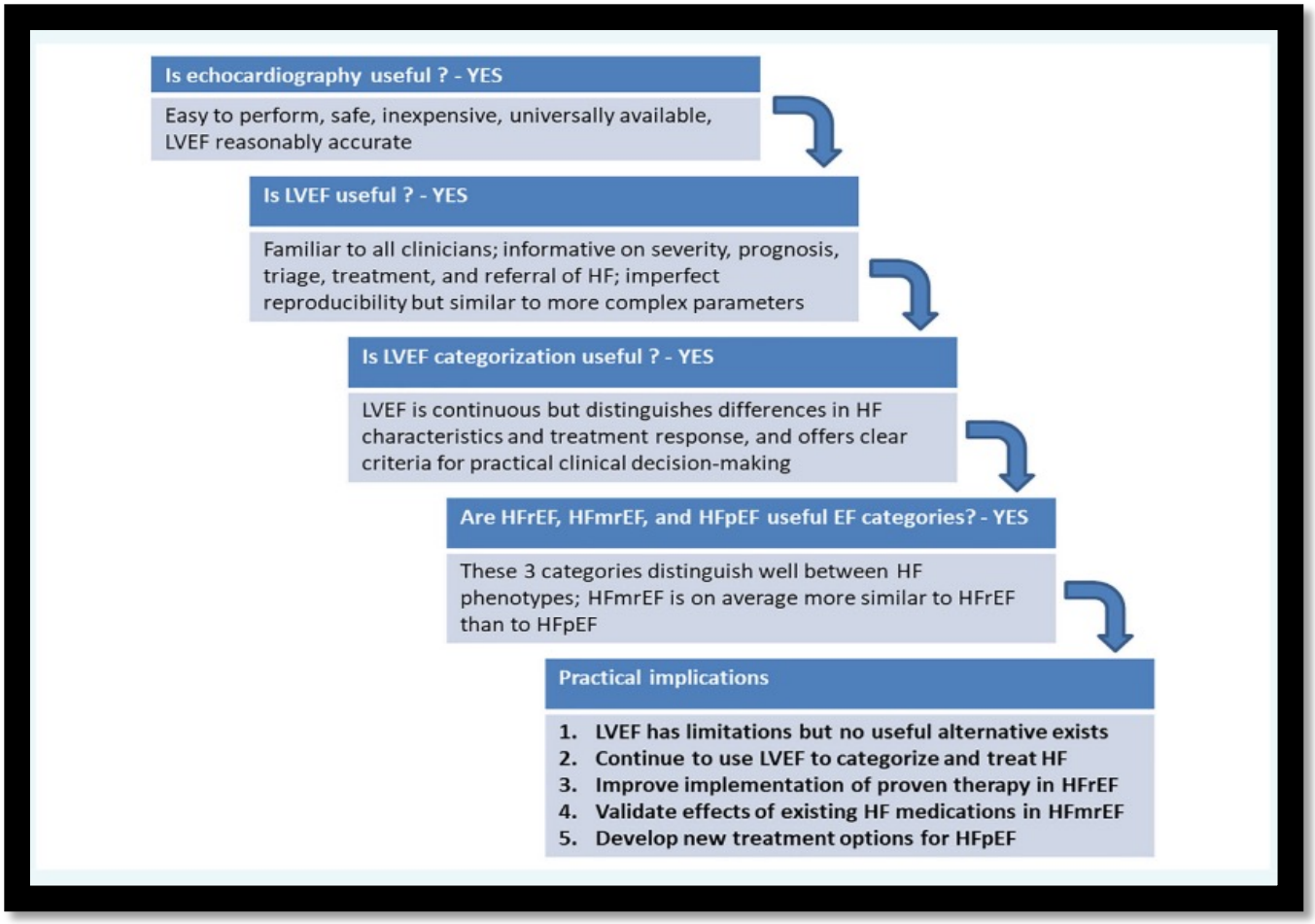




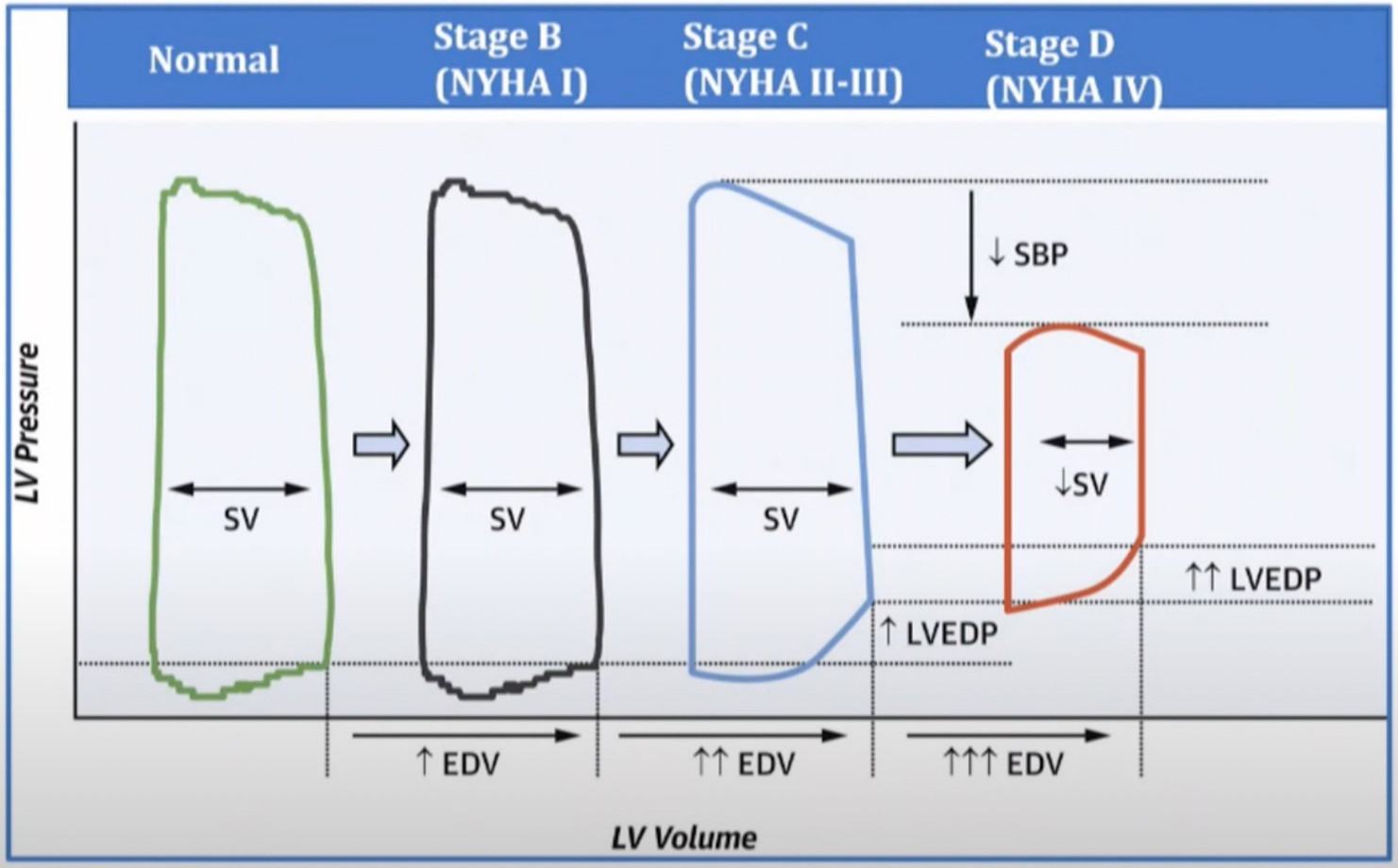
CENTRAL ILLUSTRATION: Treatment Layering According to Patient Characterization Alone: Patient Journey



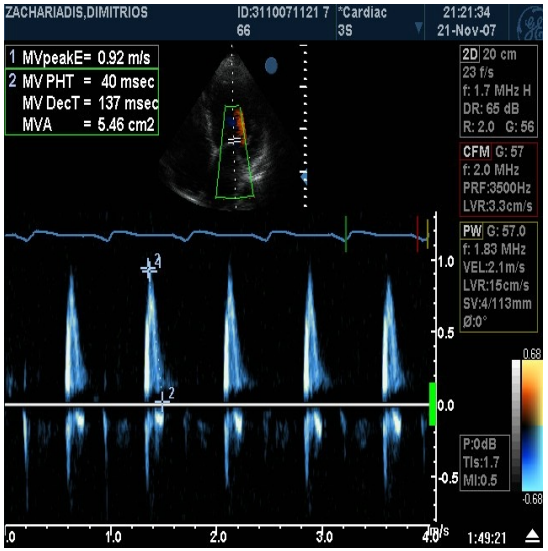
Rosano, G.M.C. et al. J Am Coll Cardiol HF. 2021;9(11):775-783.

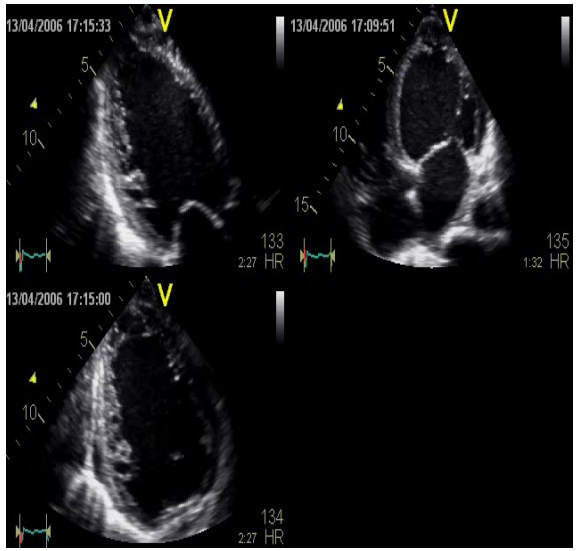


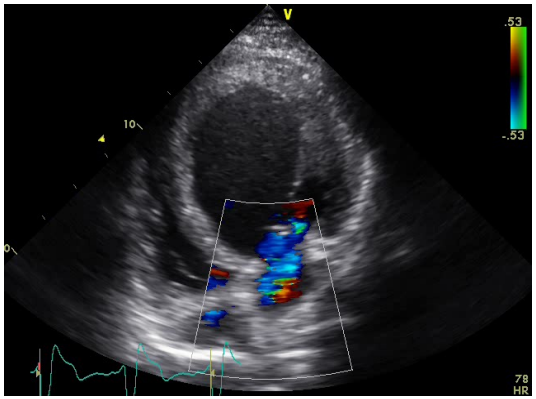
European Journal of Heart Failure

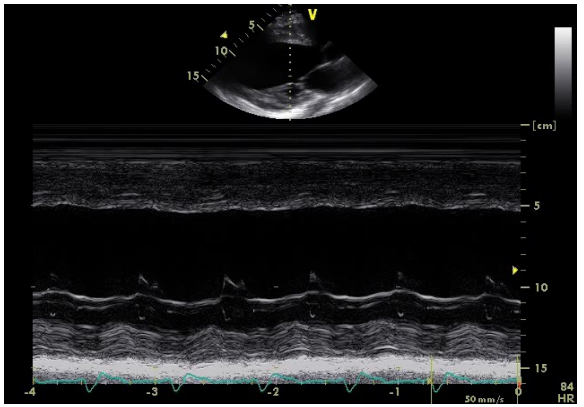


Guglin M, et al. J Am Coll Cardiol 2020; 75: 1471-87


ZACHARIADIS,DIMITRIOS
ID:3110071121 7
66
*Cardiac
3S
21:21:34
21-Nov-07
1 MVpeakE= 0.92 m/s
2 MV PHT = 40 msec
MV DecT = 137 msec
MVA = 5.46 cm2
2D 20 cm
23 f/s
f: 1.7 MHz H
DR: 65 dB
R: 2.0 G: 56
CFM G: 57
f: 2.0 MHz
PRF:3500Hz
LVR:3.3cm/s
PW G: 57.0
f: 1.83 MHz
VEL:2.1m/s
LVR:15cm/s
SV:4/113mm
Ø:0°
P:0dB
TIs:1.7
MI:0.5
1.0
0.5
0.0
-0.5
0.68
-0.68
.0
1.0
2.0
3.0
4.0
m/s
1:49:21


13/04/2006 17:15:33
13/04/2006 17:09:51
13/04/2006 17:15:00
133
2:27 HR
135
1:32 HR
134
2:27 HR


.53
-.53
78
HR


[cm]
5
10
15
-4
-3
-2
-1
0
50 mm/s
84
HR