

Overview of Strain Imaging and Common Clinical Applications

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Learning Objectives

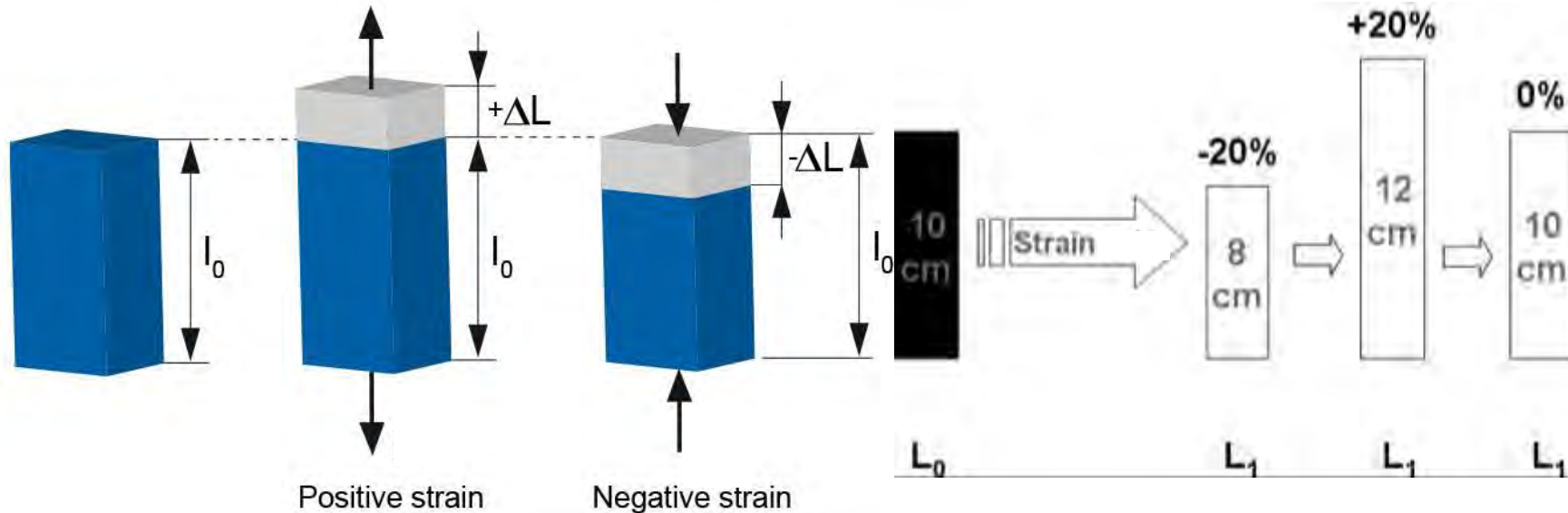
1. Discuss the basic principles of strain and myocardial deformation imaging
2. Describe common clinical applications of left ventricular strain



What is Strain?



Strain = Deformation

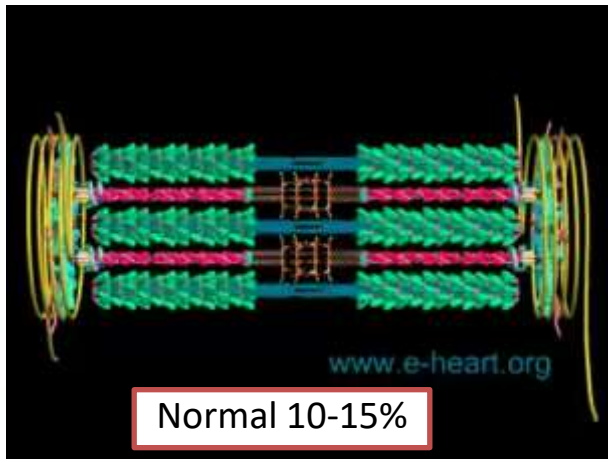


$$\text{Strain} = \frac{L_0 - L}{L_0} = \frac{\Delta L}{L_0}$$

Strain = Differential displacement of an object expressed as % (-ve \rightarrow shortening; +ve \rightarrow elongation)

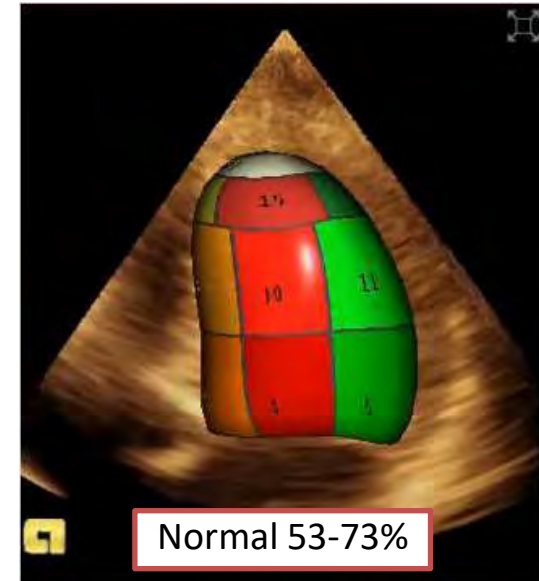


LV chamber deformation is determined by myofiber architecture



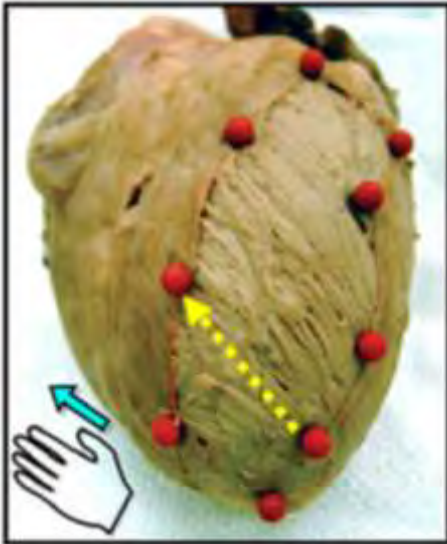
3D LV myocardial deformation is the result complex electrical and mechanical inter-connection of all fibers throughout the wall

$$\text{Strain} = \frac{L_0 - L}{L_0} = \frac{\Delta L}{L_0}$$

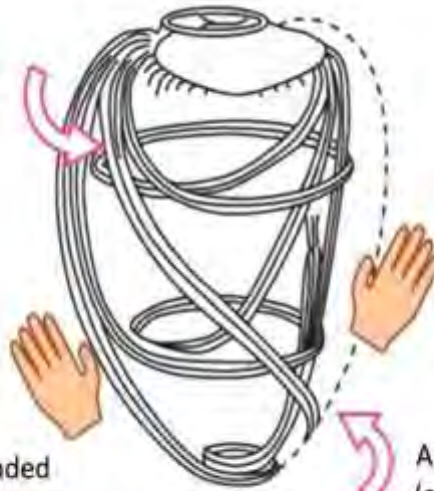


Cardiac Muscle Fiber Orientation

Epicardium [L]



Basal rotation (clockwise)



Right-handed helix

Left-handed helix

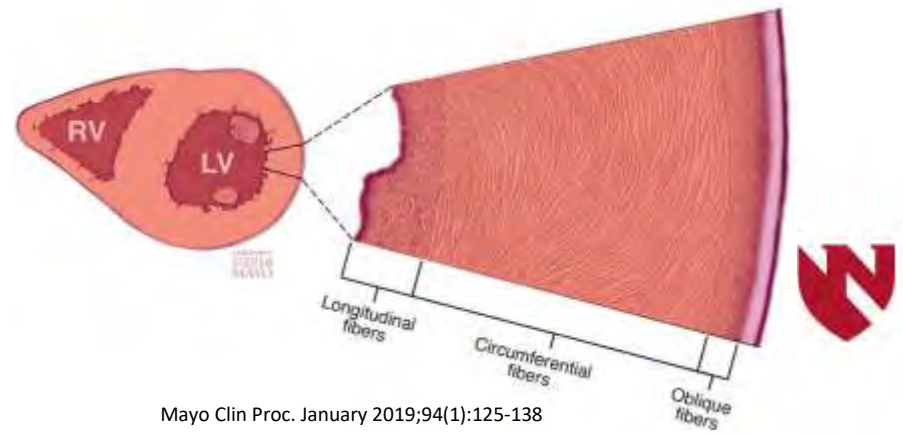
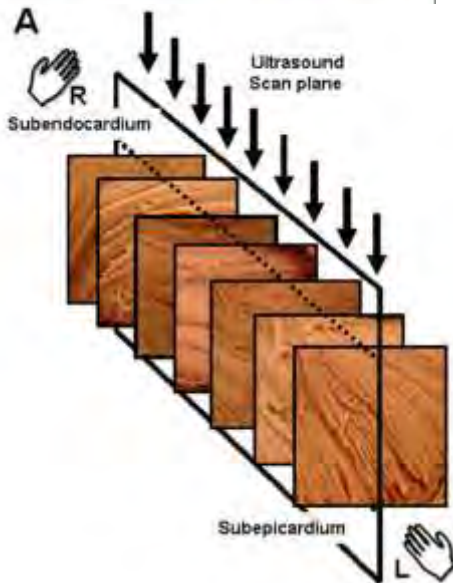
Apical rotation (anticlockwise)

Endocardium [R]

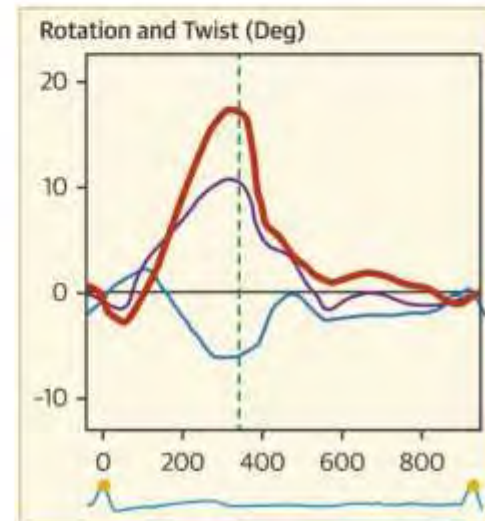
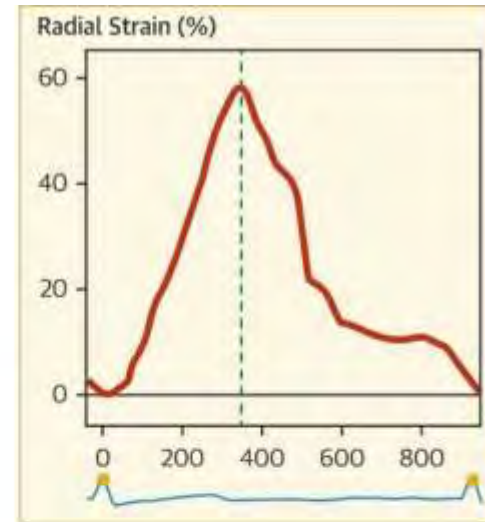
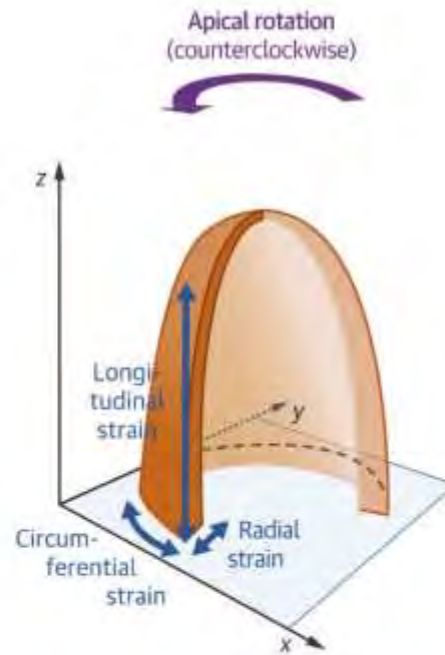
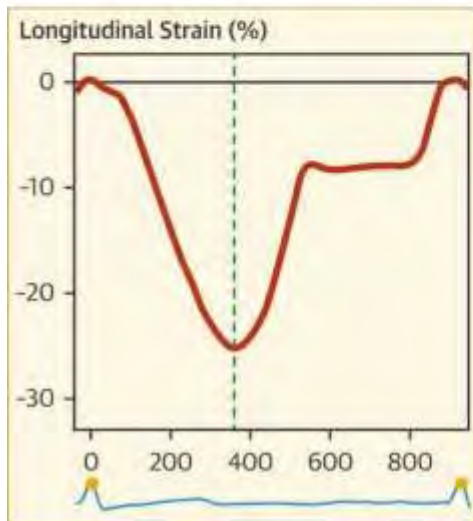
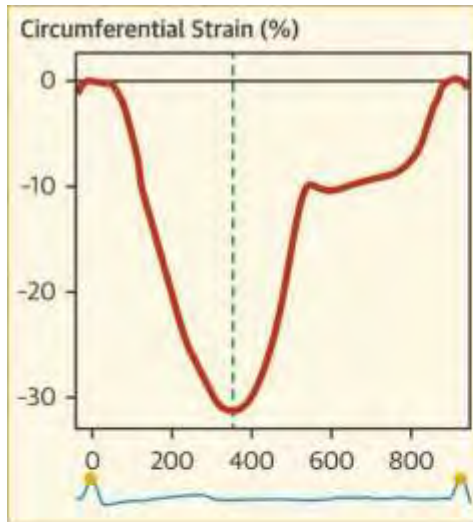


Journal of Cardiovascular Ultrasound 2011 19 1-6.

Fibers in the sub-endocardium are arranged in right-handed helix, then smoothly transition to a transverse circular arrangement in the midmyocardium and then finally a left-handed helix in the epicardium



Principle myocardial deformations

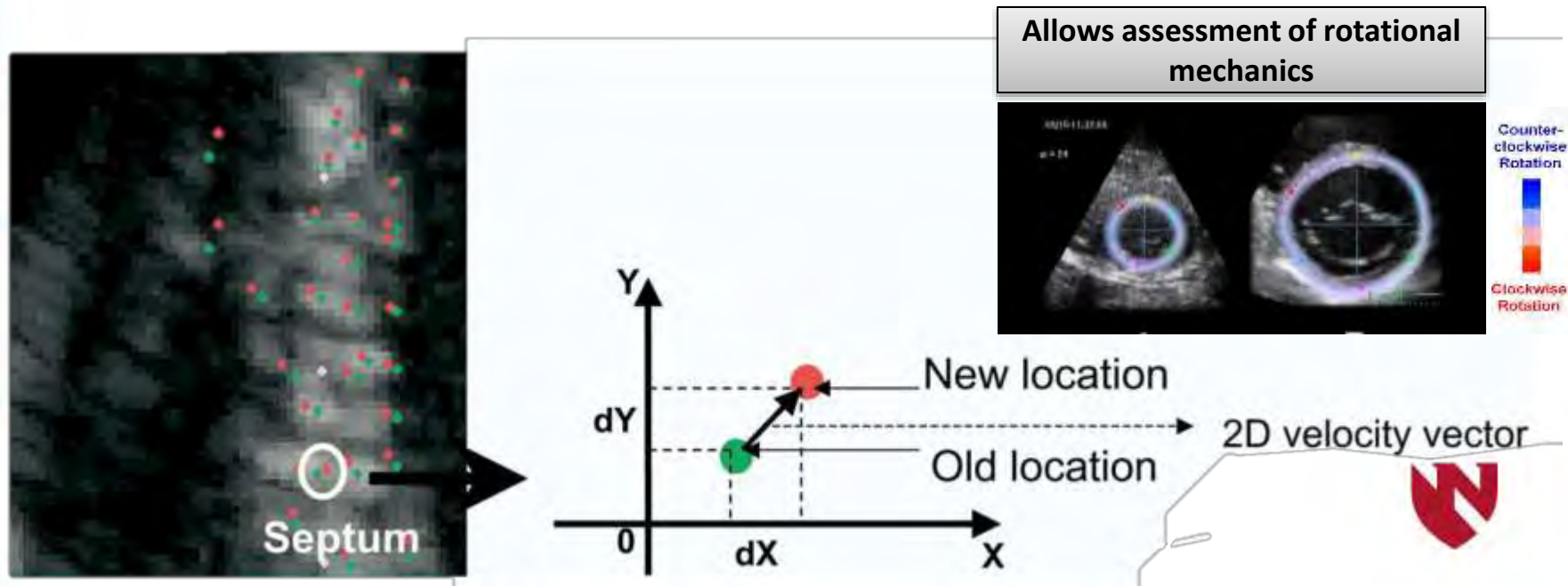


Acoustic Pattern (Speckle) Tracking

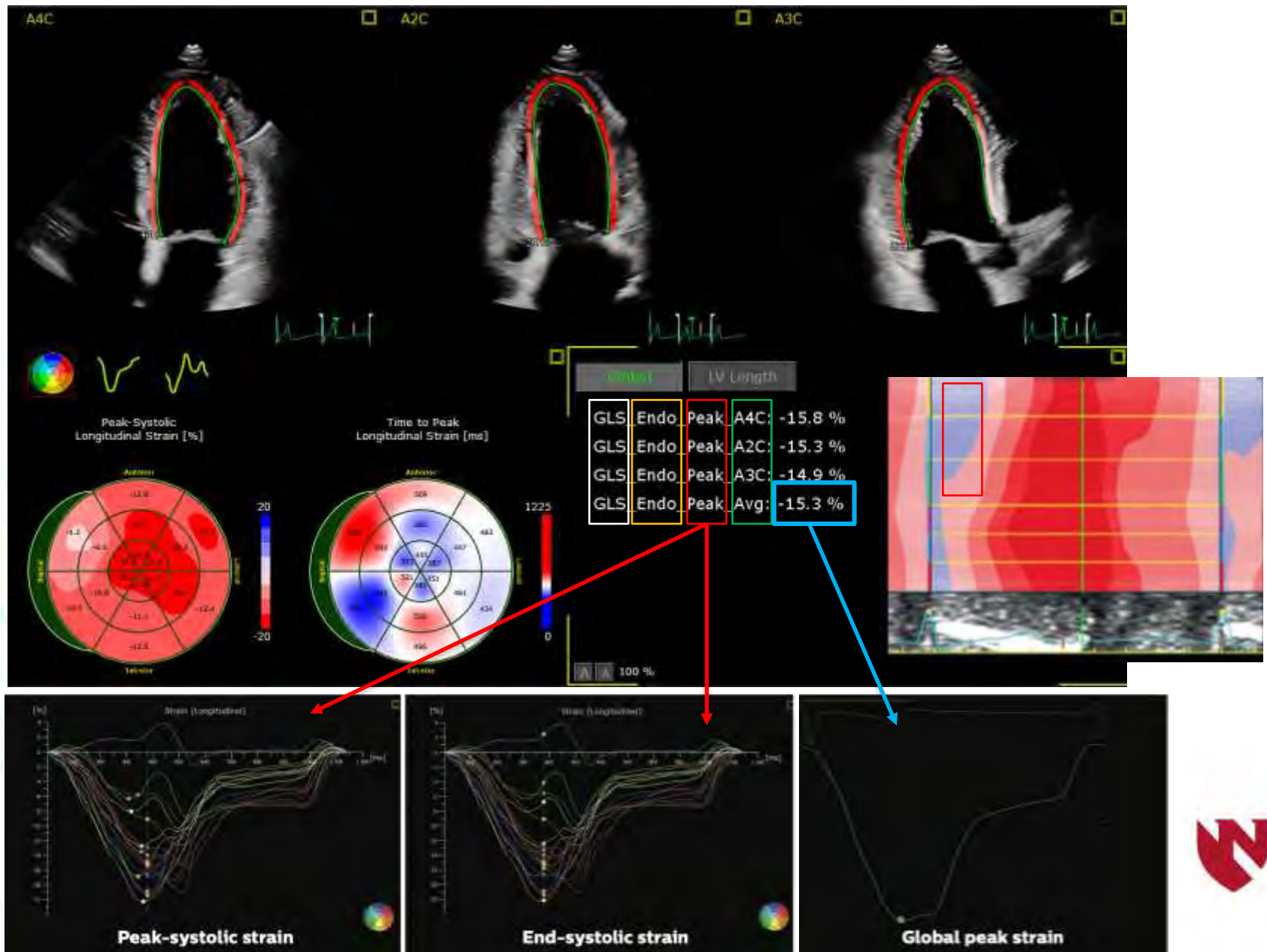
Velocity is estimated as a shift of each speckle divided by time between successive frames (or multiplied by Frame Rate)

$$\mathbf{2D\ vector: (V_x, V_y) = (dX, dY) * FR}$$

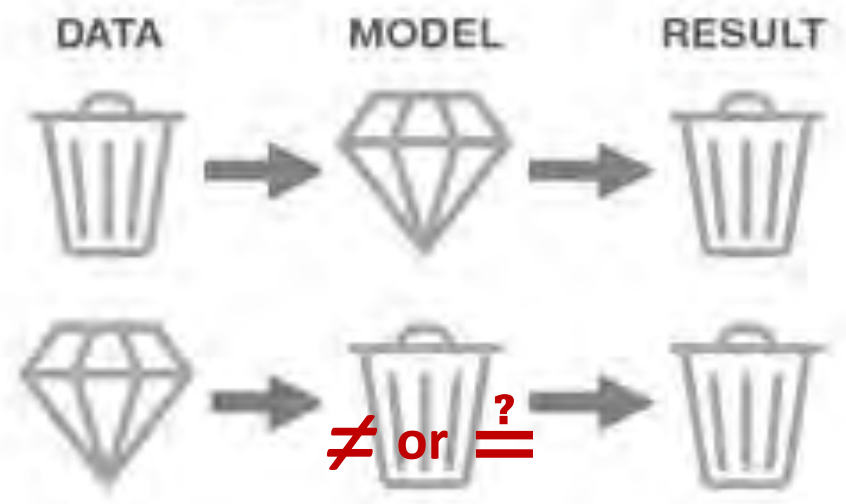
Improved signal noise levels, less angle dependency, & freedom to assess strain using regular B-Mode datasets in 2D, rather than a single dimension locked along the scan line



GLS Results Display

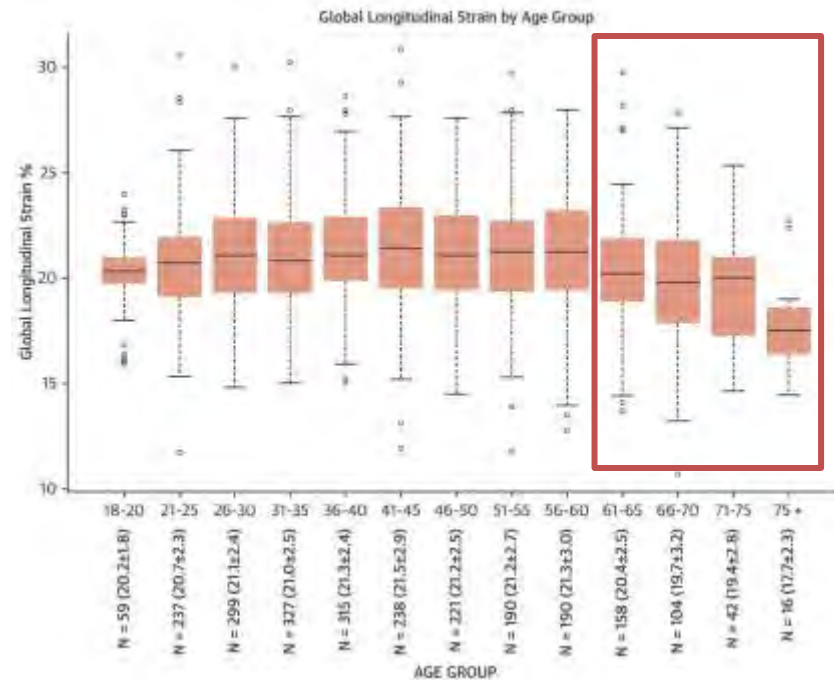


Technical Challenges and Barriers to Adoption



Age, Gender, & Vendor Variability

- Individuals > 60 years show clinically and statistically relevant impairment in GLS, compared with younger subjects ($19.9\% \pm 2.9\%$ vs $21.1\% \pm 2.6\%$, $P < .01$)
- **Normal** ranges for GLS also varied with covariates such as **weight** and **BP**
- Strain reported **lower in women** vs. men, presumably due to smaller female heart (



Regardless of vendor or clinical covariate

- ➔ **GLS <16%** is almost certainly **abnormal**
- ➔ **GLS >18%** is considered **normal**
- ➔ **GLS between -16% and -18%** is **ambiguous** (probably abnormal), although normal measurements may still be in this category due to increased afterload and older age



Definition of Cancer Therapeutics–Related Cardiac Dysfunction (CTRCD)

Established Criteria for Cardiotoxicity

Standard	Definition of Cardiotoxicity	Clinical and Imaging Features
American Society of Echocardiography and European Association of Cardiovascular Imaging (ASE/EACVI)	LVEF fall by >10% to absolute EF <53%	May be global or regional change in LV function Symptomatic or asymptomatic for HF
European Society of Cardiology (ESC)	LVEF fall by >10% from baseline to EF <50%	Symptomatic or asymptomatic for HF
National Cancer Institute (NCI)	HF grade 1–5 adopted in common terminology criteria for adverse events	Grade 1 (asymptomatic) Grade 2 (mild to moderate symptoms) Grade 3 (symptomatic on minimal exertion or at rest) Grade 4 (life threatening) Grade 5 (death)
Canadian Cardiovascular Society (CCS)	LVEF fall by >10% from baseline or LVEF <53%	Guidelines also recommend (1) 3D echocardiography or same imaging modality during cancer therapy, (2) myocardial strain imaging, and (3) cardiac biomarkers (N-terminal pro brain natriuretic peptide, troponin) for early detection
European Society of Medical Oncology (ESMO)	Symptomatic decline in LVEF of at least 5% to <55% or asymptomatic decline in LVEF of at least 10% to <55%	Symptoms for congestive HF with signs including but not limited to S3 gallop, tachycardia, or both Decline in LVEF either global or regional



Expert Consensus for Multimodality Imaging Evaluation of Adult Patients during and after Cancer Therapy: A Report from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

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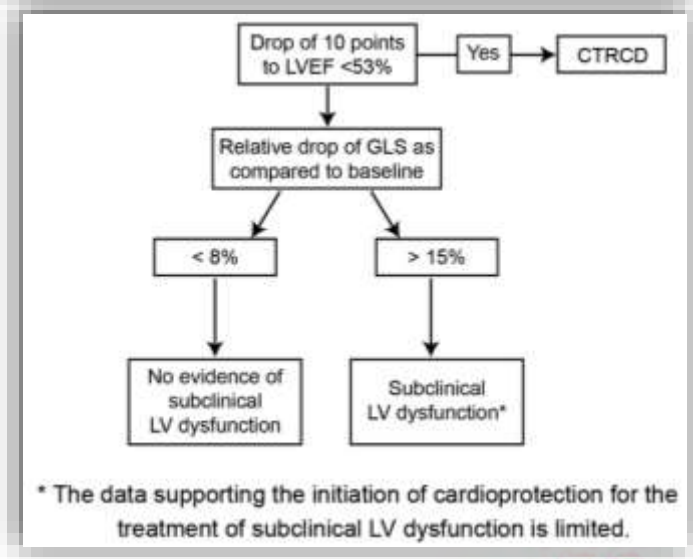
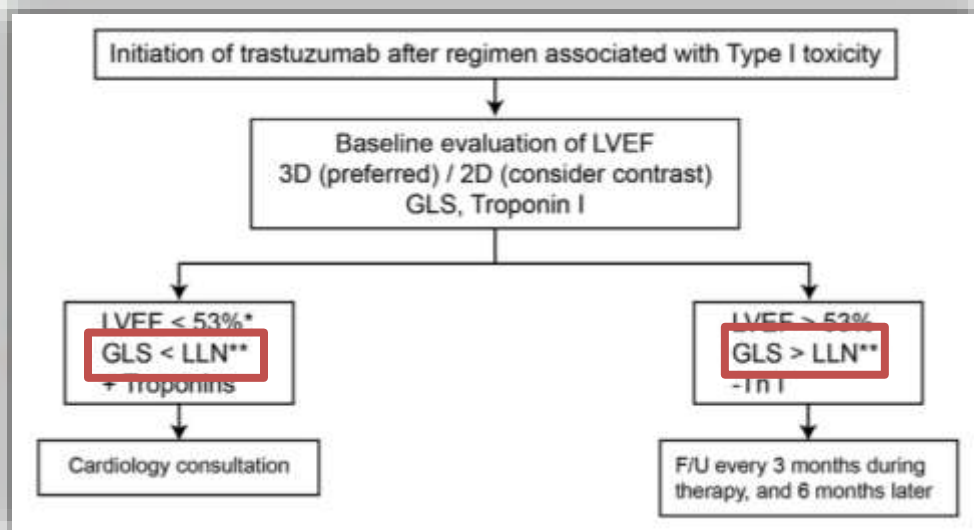
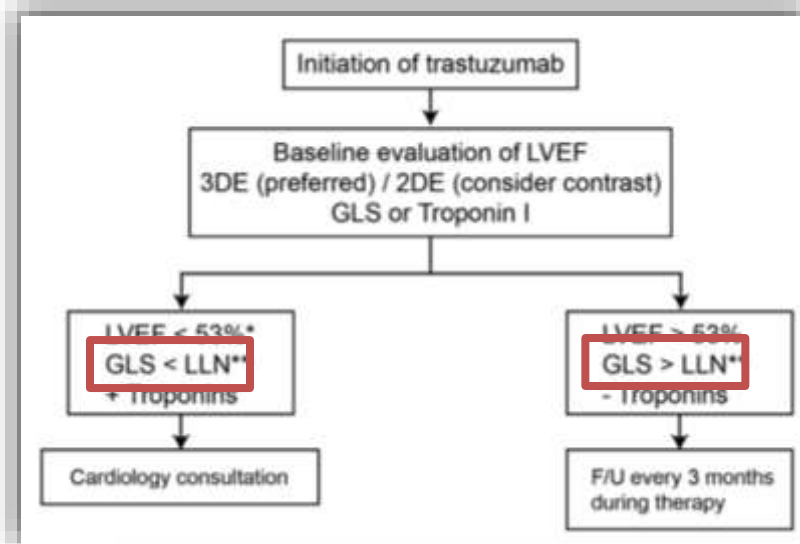
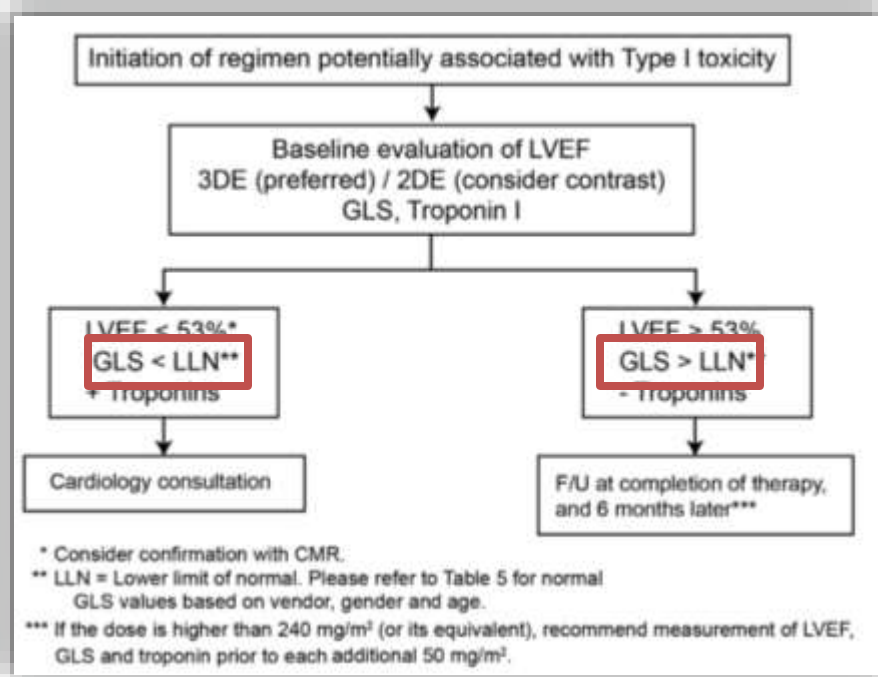
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<http://dx.doi.org/10.1016/j.echo.2014.07.012>

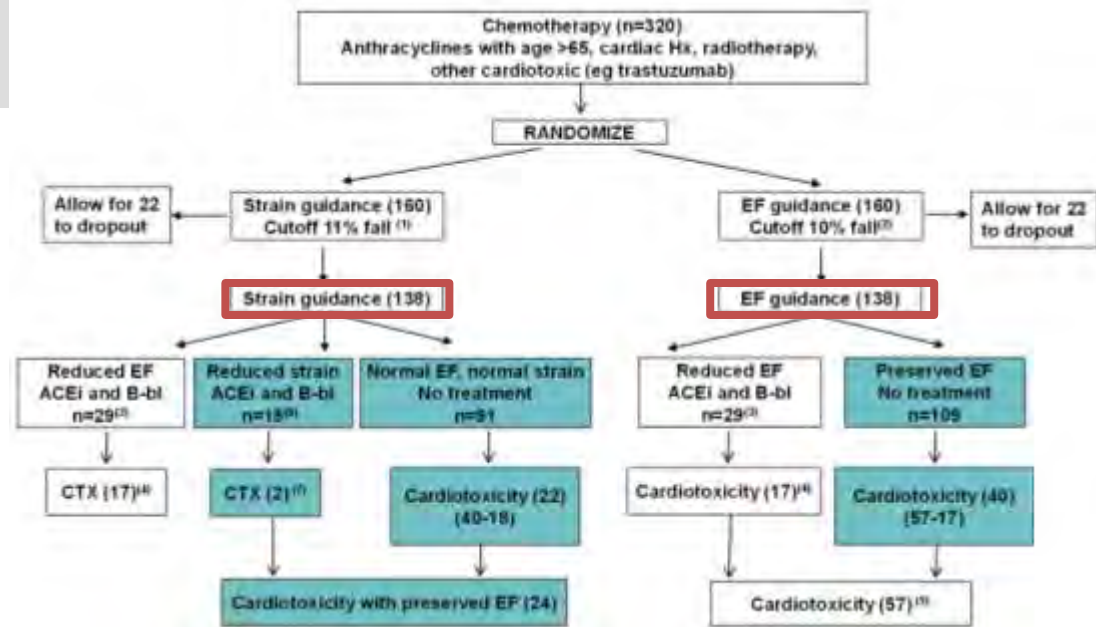


Expert consensus for screening & diagnosis of CTRCD



Strain sUrveillance during Chemotherapy for improving Cardiovascular Outcomes (SUCCOUR)

International, multicenter, prospective, RCT



Trial ID	ACTRN12614000341628
Ethics application status	Approved
Date submitted	21/03/2014
Date registered	31/03/2014
Type of registration	Retrospectively registered

Titles & IDs

Public title	Strain sUrveillance during Chemotherapy for improving Cardiovascular Outcomes
Scientific title	Randomised controlled trial (RCT) of chemotherapy patients at risk of cardiotoxicity undergoing cardioprotection guided by measurement of LV strain compared with cardioprotection guided by measurement of left ventricular (LV) ejection fraction for avoidance of cardiotoxicity
Secondary ID [s]	none
Universal Trial Number (UTN)	
Trial acronym	SUCCOUR Study
Linked study record	



Strain-Guided Management of Potentially Cardiotoxic Cancer Therapy



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Patients were followed for change in EF and development of CTRCD (symptomatic EF reduction of >5% or >10% asymptomatic to <55%) over 1 year

CENTRAL ILLUSTRATION: Various Surveillance Strategies, Initiation of Cardioprotective Therapy, and the Subsequent Response at the 1-Year Follow-Up

Baseline echocardiography

Echo surveillance during therapy

1-year follow-up

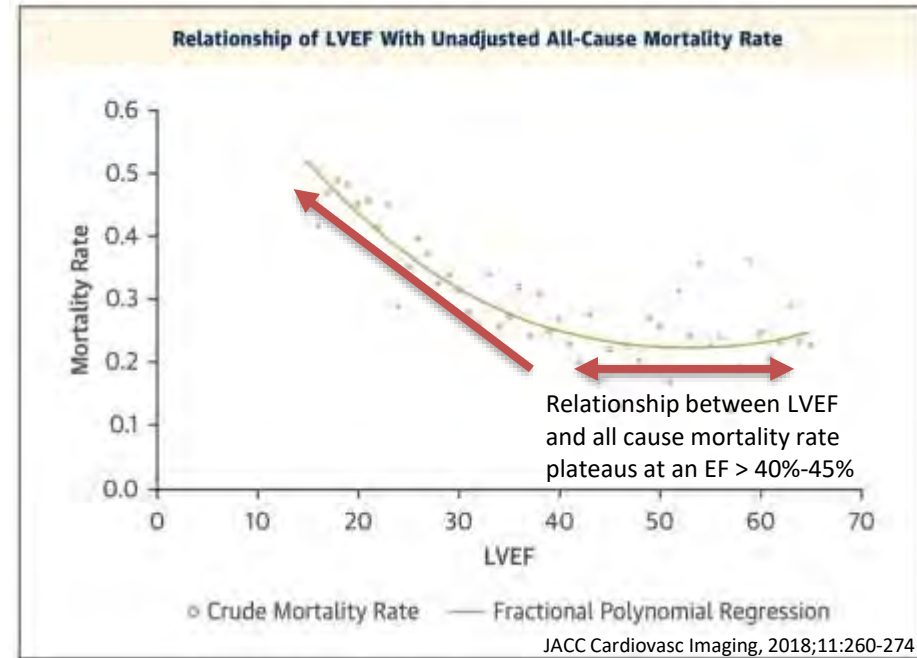
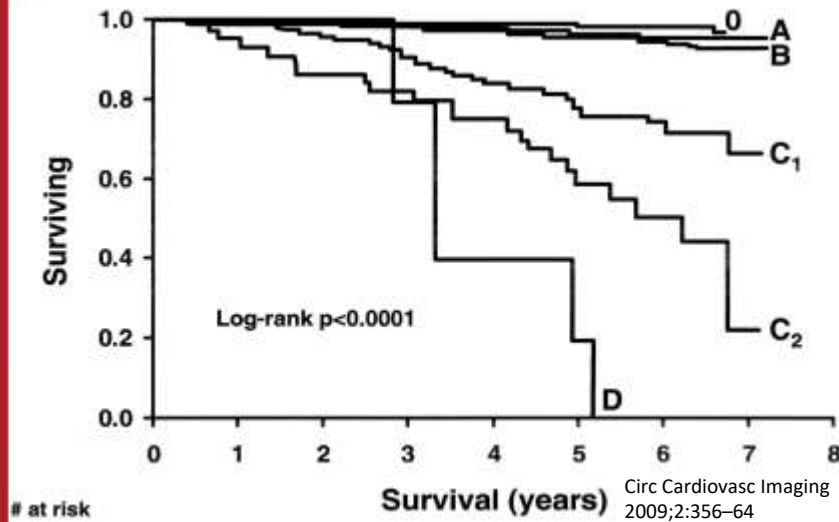
Although the **primary outcome of change in LVEF in both groups was similar**, LV surveillance with GLS is associated with:

- 1) Greater use of CPT
- 2) Higher final LVEF
- 3) Lower incidence of CTRCD (with a number needed to treat of 13)

Findings underscore the flaws of our current imaging practices and critical limitations of relying on LVEF as the main measure of CV risk

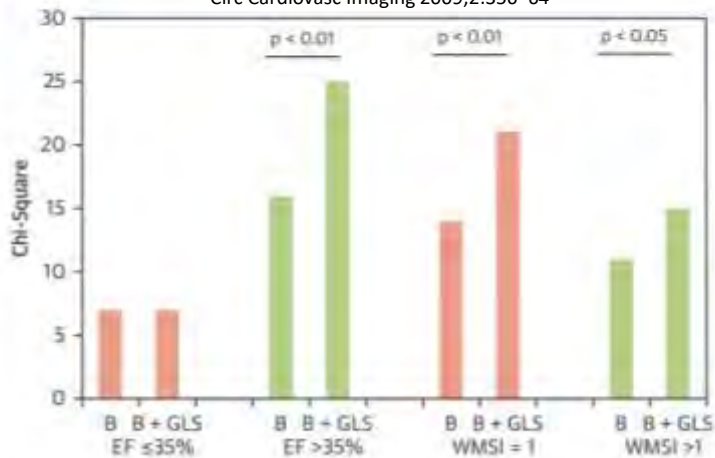


Prognostic value of Strain imaging



GLS adds significant incremental predictive value for mortality in patients with LVEF >35%, irrespective of WMSI

Circ Cardiovasc Imaging 2009;2:356-64



Baseline clinical variables: Diabetes, Age, & HTN were selected based on independent univariable predictors. Data from Stanton et al.

- GLS is a **stronger predictor than LVEF** of all-cause mortality and a composite of cardiac death, HF hospitalization, and malignant arrhythmias in **meta-analysis of 5,721 subjects** across 16 studies of various cardiac diseases
- In 603 patients in the VALIANT (Valsartan in **Acute Myocardial Infarction** Study) echocardiographic sub-study, GLS provided prognostic value for the **prediction of all-cause mortality**, independent of and incremental to clinical variables and LVEF, and circumferential strain rate identified patients at risk of LV remodeling.
- GLS **improved predictive ability** of the Framingham risk score in a **Danish population** when natriuretic peptides did not



Global Longitudinal Strain to Predict Mortality in Patients With Acute Heart Failure



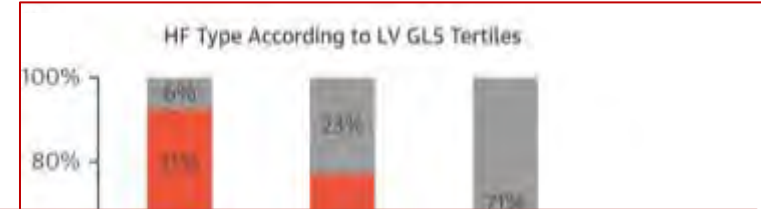
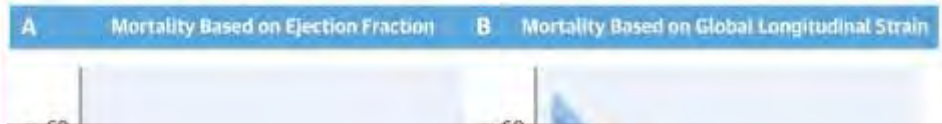
4,172 consecutive pts w acute HF

- HFrEF, HFmEF, HFpEF
- Mild, mod, severe GLS

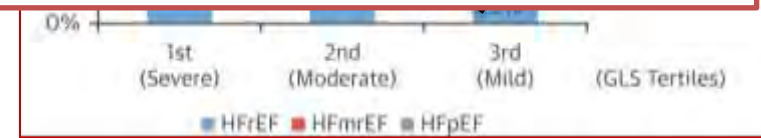
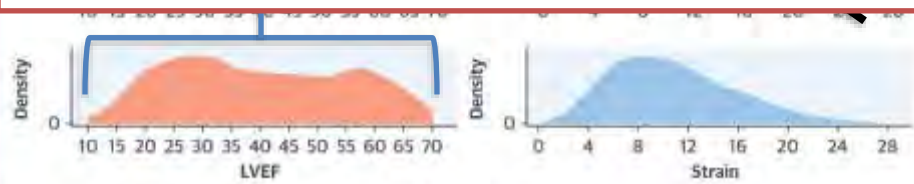
5-year all-cause mortality

Jin Joo Park, MD, PhD,¹ Jun-Bean Park, MD, PhD,¹ Jae-Hyeong Park, MD, PhD,² Goo-Yeong Cho, MD, PhD²

CENTRAL ILLUSTRATION: Prognostic Value of Strain in Acute Heart Failure: Probability Plot for 5-Year All-Cause Mortality



In patients with acute HF, GLS has greater prognostic value than LVEF supporting consideration for GLS as a standard measurement in all patients with HF



The proportion of patients with HFrEF increased as GLS decreased.

Park, J.J. et al. J Am Coll Cardiol. 2018;71(18):1947-57.

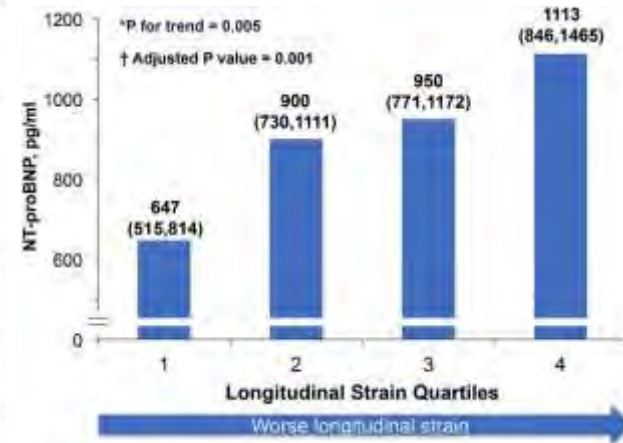
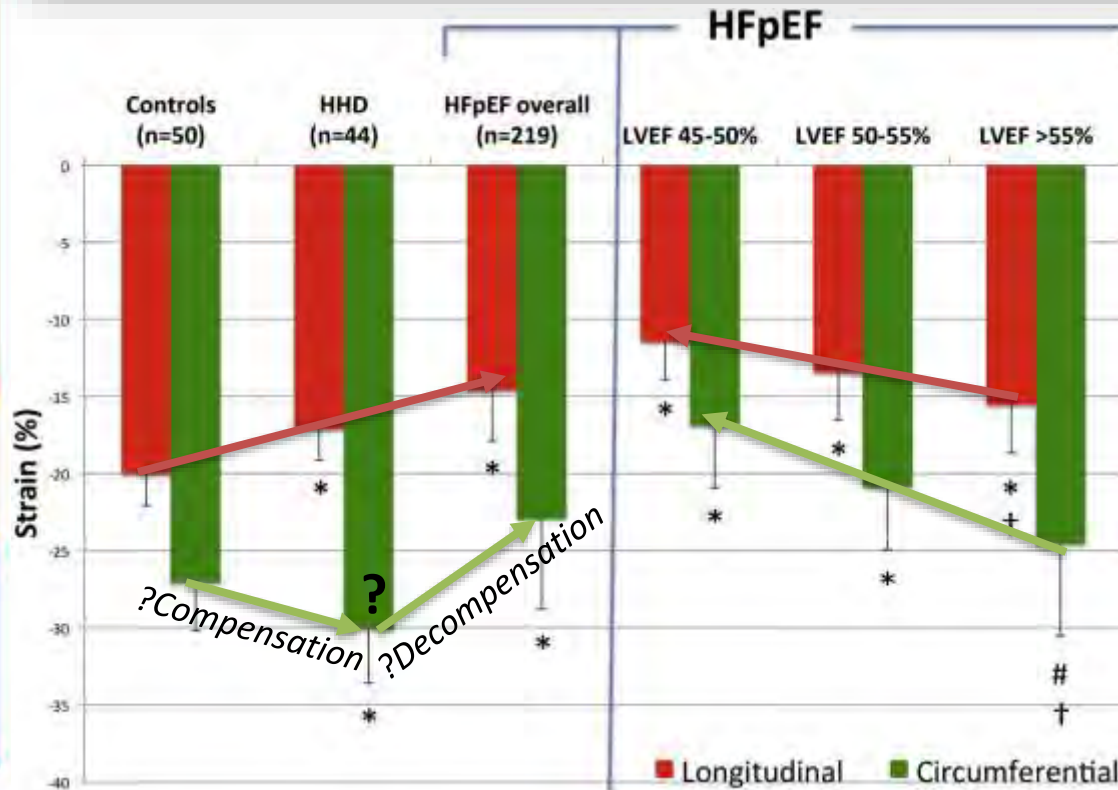
- Patients with HFrEF had slightly higher mortality than those with HFmEF or HFpEF, whereas patients with reduced strain had significantly higher mortality
- In multivariable analysis, each 1% improvement in GLS was associated with a 5% decreased risk for mortality



Impaired Systolic Function by Strain Imaging in Heart Failure With Preserved Ejection Fraction

Elisabeth Kraigher-Krainer, MD,* Amil M. Shah, MD, MPH,* Deepak K. Gupta, MD,* Angela Santos, MD,* Brian Claggett, PhD,* Burkert Pieske, MD,† Michael R. Zile, MD,‡ Adriaan A. Voors, MD,§ Marty P. Lefkowitz, MD,|| Milton Packer, MD,¶ John J. V. McMurray, MD,# Scott D. Solomon, MD,* for the PARAMOUNT Investigators

- 219 HFpEF patients from HFpEF PARAMOUNT trial
- 50 normal controls
- 44 pts with HHD but no HF



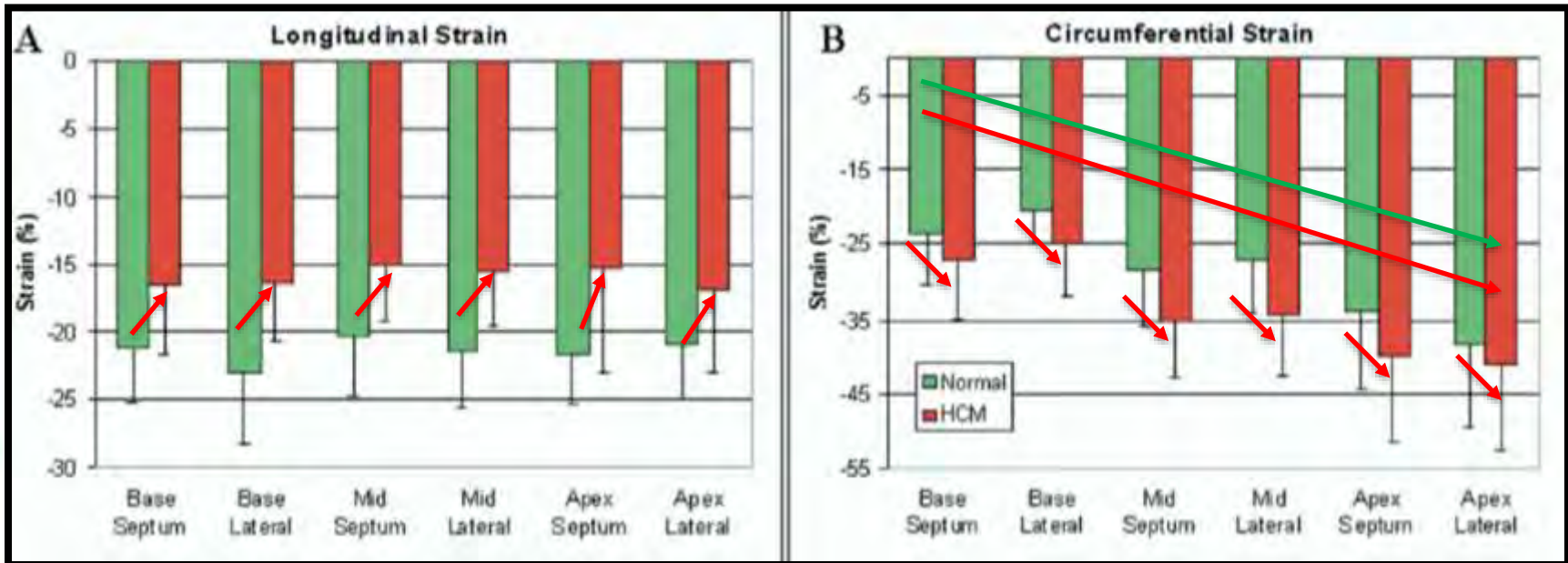
Lower LS was modestly associated with higher NT-proBNP, even after adjustment for 10 baseline covariates including LVEF, measures of diastolic function, and LV filling pressure

Compared with normal controls & pts with hypertensive heart disease, HFpEF/HFmEF pts demonstrated significantly lower longitudinal strain



Strain Abnormalities in HCM vs. Healthy Controls

72 patients with HCM and 32 controls evaluated using 2-dimensional velocity vector imaging

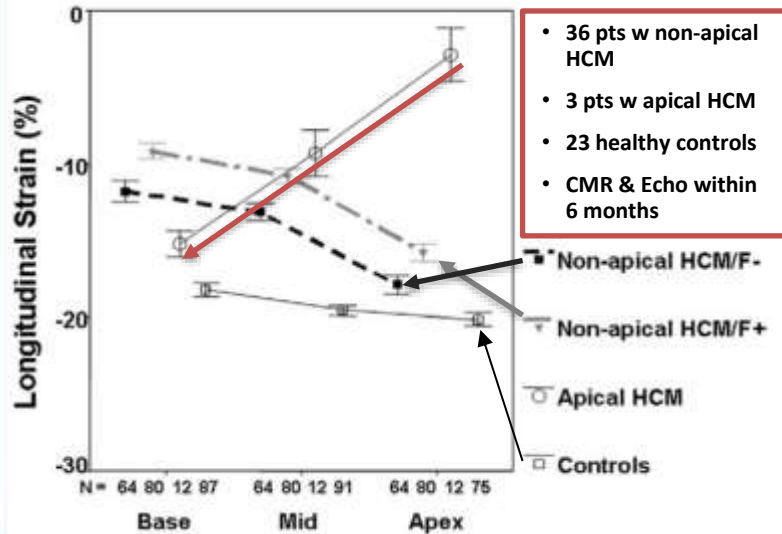


Longitudinal strain in Hypertrophic Cardiomyopathy is significantly **lower** than in control subjects **without** notable regional variability

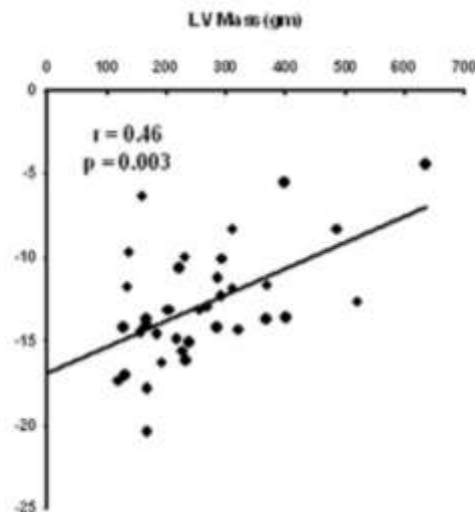
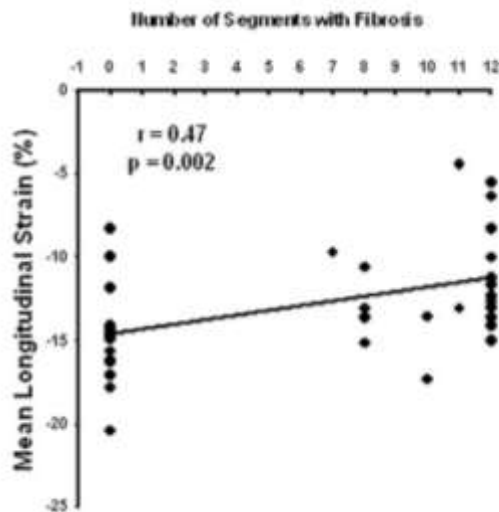
Circumferential strain **increased** from base to apex, & was significantly **higher** in HCM for all segments except lateral apex



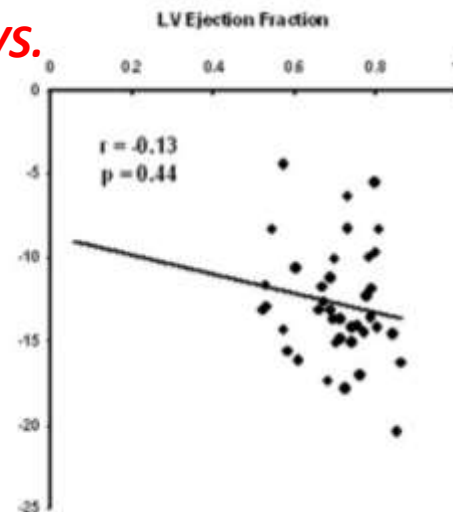
Association Between Regional Strain and Myocardial Fibrosis in HCM



- LS significantly decreased from apex-to-base in all pts w non-apical HCM
- Apex-to-base decrease of LS was greatest in HCM w fibrosis > HCM wo fibrosis > healthy controls
- All 3 patients with **apical form of HCM** (all with fibrosis) had an **apex-to-base increase** of LS
- Mean ES LS had moderate correlation with # of fibrotic segments ($r = 0.47$, $P = .002$) and total LV mass ($r = 0.46$, $P = .003$)
- *Fibrosis & wall thickness were multivariate predictors of lower segmental longitudinal strain ($P = .003$)*

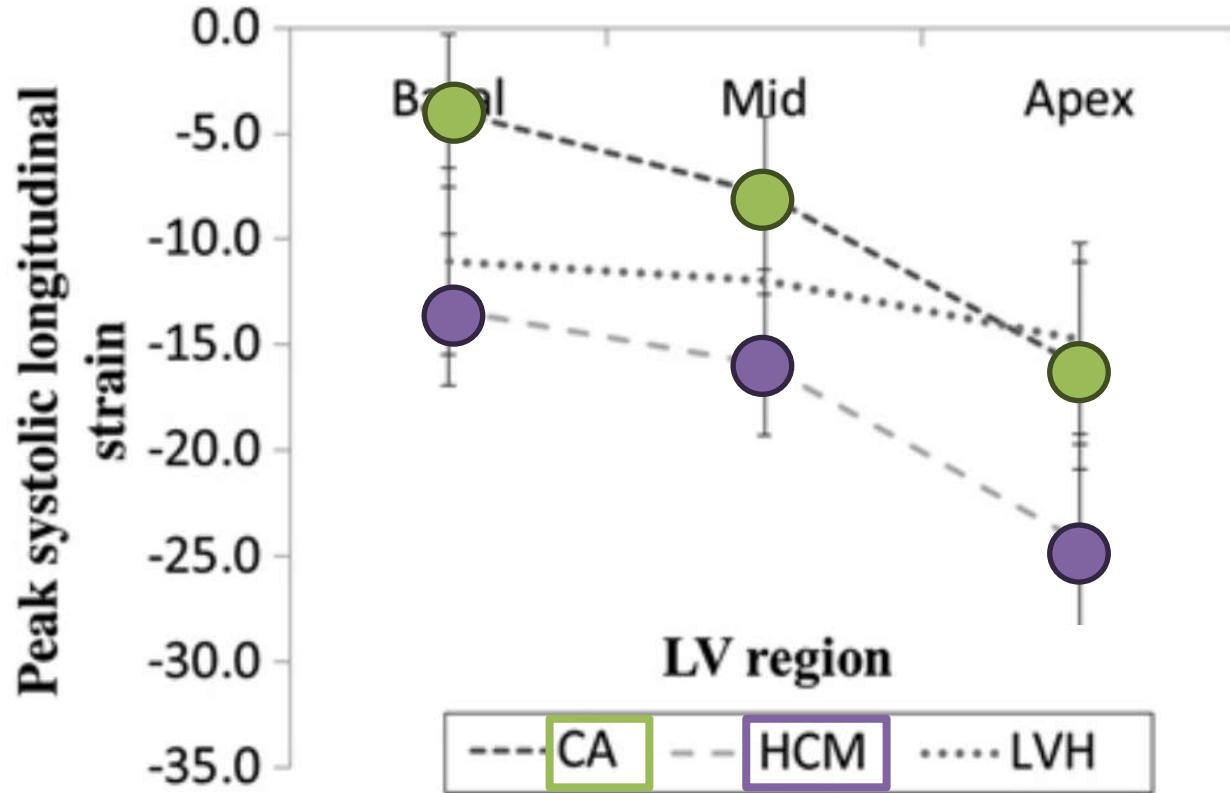


vs.



GLS for the Diagnosis of Cardiac Amyloid

- 55 pts with CA
- 15 pts w HCM
- 15 pts w AS



- Apex-to-base gradients in absolute regional strain were present in all groups
- **Absolute GLS** in CA \ll HCM or AS ($p < 0.001$)
- **GLS Gradients** in CA \gg AS ($p < 0.001$), but no different than HCM
- **Regional LS** is significantly lower in CA vs HCM groups at all 3 levels ($p < 0.001$)



EXPERT CONSENSUS DECISION PATHWAY

2023 ACC Expert Consensus Decision Pathway on Comprehensive Multidisciplinary Care for the Patient With Cardiac Amyloidosis

A Report of the American College of Cardiology Solution Set Oversight Committee

Endorsed by the American Association of Neuromuscular & Electrodiagnostic Medicine, Heart Failure Society of America, and International Society of Amyloidosis. The American Academy of Neurology affirms the value of this statement.

TABLE 1 Clues Suggesting a Diagnosis of Cardiac Amyloidosis

Cardiac Manifestations

Clinical

- Fatigue
- Heart failure symptoms
- Family history of heart failure

Electrical

- Conduction system disease/pacemaker
- Atrial fibrillation
- Pseudoinfarct pattern
- Discordant QRS voltage for degree of increased left ventricular wall thickness on imaging

Imaging

- Increased left ventricular wall thickness
- Grade 2 or worse diastolic function
- Abnormal longitudinal **strain** with apical sparing
- Diffuse subendocardial or transmural late gadolinium enhancement on cardiac magnetic resonance imaging with increased extracellular volume fraction

Laboratories

- Persistent low-level troponin elevation
- Elevated B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide

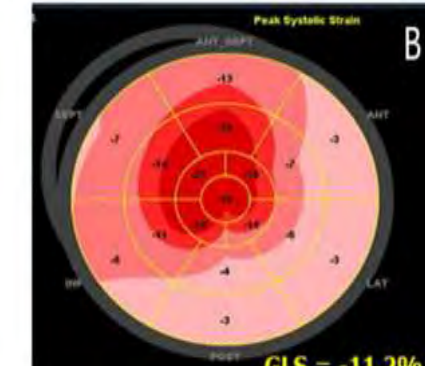
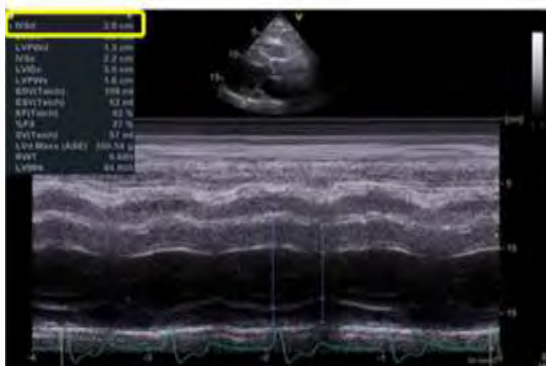
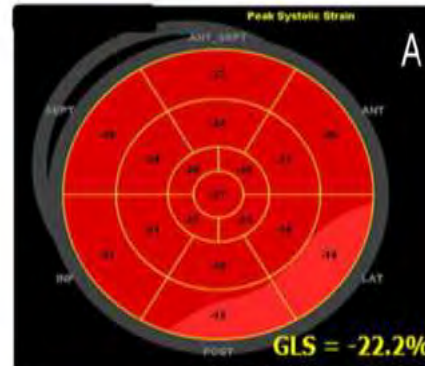
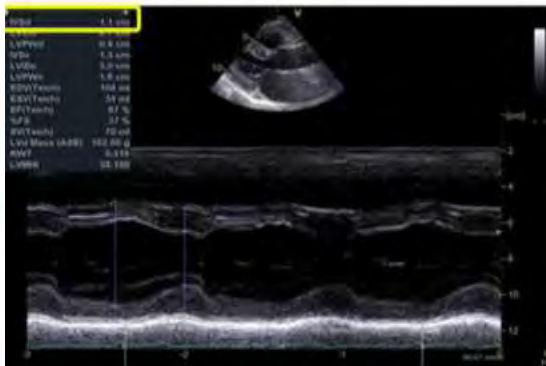
Necessary clinical testing will include, at a bare minimum, a screening ECG, cardiac biomarkers (natriuretic peptide and troponin), and an echocardiogram, ideally with **strain** imaging. If there are symptoms or abnormal

Treatment efficacy will likely be determined by a combination of circulating markers (prealbumin, cardiac troponins, natriuretic peptides, kidney function), imaging markers (the best candidates appear to be echocardiographic global longitudinal **strain** and CMR extracellular volume fraction),²³⁸ as well as conventional HF metrics (functional status, hospitalizations, and survival).



Anderson-Fabry Disease

X-linked lysosomal storage disorder caused by deficiency in α -galactosidase A enzyme



- Complex and heterogeneous strain patterns
- Greater impairment of basal vs. apical segments
- Reduced inferolateral strain despite septal hypertrophy
- Reduced strain in all basal segments occurs with advanced disease



Application of a Parametric Display of Two-Dimensional Speckle-Tracking Longitudinal Strain to Improve the Etiologic Diagnosis of Mild to Moderate Left Ventricular Hypertrophy

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Background: The distinction of hypertrophic cardiomyopathy (HCM) or cardiac amyloidosis (CA) from hypertensive heart disease may be difficult. The aim of this study was to determine the impact of parametric (polar) maps of regional longitudinal strain on identification of the etiology of mild to moderate left ventricular hypertrophy (LVH).

Methods: Twenty-four consecutive echocardiographic studies with mild to moderate LVH (eight with CA, eight with HCM, and eight with hypertensive heart disease) were selected on the basis of the availability of adequate images to assess longitudinal strain and absence of electrocardiographic criteria for low voltage or LVH or a pseudoinfarct pattern. Twenty level 3-trained readers provided the most likely of three diagnoses (CA, HCM, or hypertensive heart disease) and scored their confidence in making the diagnosis from two-dimensional images and diastolic parameters. A teaching exercise was provided on the interpretation of longitudinal strain in these cohorts, and interpretation was repeated with the addition of the strain polar map.

Results: Baseline concordance among the readers was poor ($\kappa = 0.28$) and improved with the addition of strain data ($\kappa = 0.57$). Accuracy was improved with the addition of polar maps for the entire study cohort ($P < .001$), with 22% of cases reclassified correctly. The largest improvements in sensitivity (from 40% to 86%, $P < .001$), specificity (from 84% to 95%, $P < .001$), and accuracy (from 70% to 92%, $P < .001$) were seen for CA. The strain polar map significantly improved reader confidence in making the correct diagnosis overall ($P < .001$).

Conclusions: Regional variations in strain are easily recognizable, accurate, and reproducible means of differentiating causes of LVH. The detection of LVH etiology may be a useful clinical application for strain. (J Am Soc Echocardiogr 2014;27:888-95.)

Keywords: LV hypertrophy, Hypertension, Hypertrophic cardiomyopathy, Amyloidosis, Strain



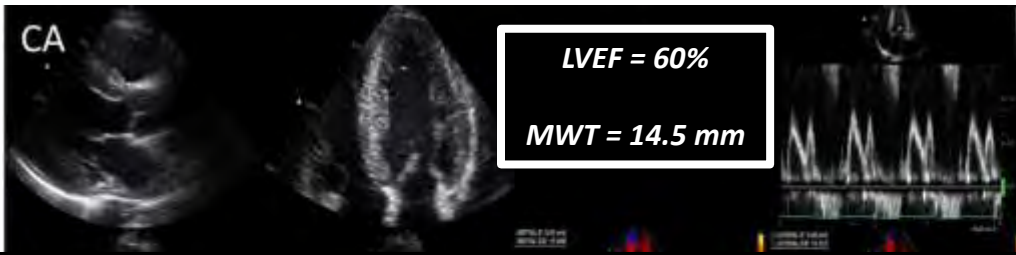


Table 3 Diagnostic accuracy in patients with mild to moderate LVH with and without strain polar map

Diagnosis	Sensitivity (%)	Specificity (%)	Accuracy (%)	PPV (%)	NPV (%)
CA					
Baseline read	40	84	70	55	75
Strain read	86	95	92	92	94
<i>P</i>	<.001	.002	<.001	<.001	<.001
HCM					
Baseline read	44	75	65	45	73
Strain read	52	84	73	63	78
<i>P</i>	.054	.01	.001	<.001	.005
HHD					
Baseline read	60	59	60	42	72
Strain read	70	74	73	59	84
<i>P</i>	.061	.002	.001	.001	.004

NPV, negative predictive value; PPV, positive predictive value.

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Detection of LVH etiology may be a useful clinical application for strain

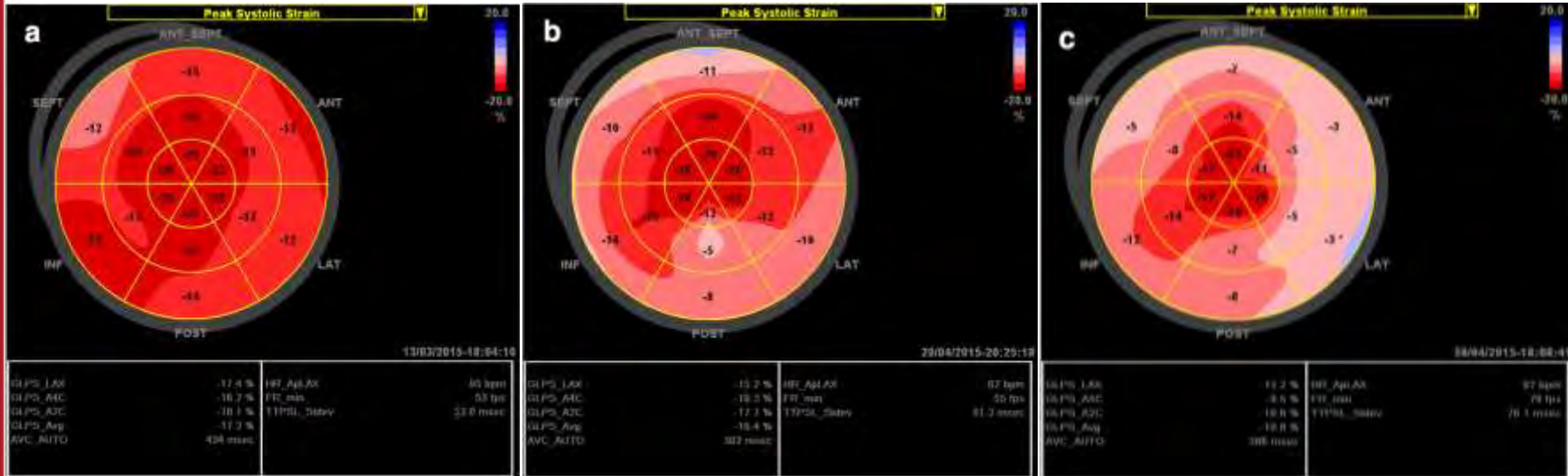


Key take home messages

- **Strain imaging** reflects the direction of the **wall movement** rather than the direction of the muscle fibers themselves
- **GLS** is the most robust & widely used strain parameter in clinical practice
- Despite being systolic phase parameters, **LV-GLS** and **LVEF** do **not** necessarily correlate
- In **HFrEF**, strain may not be needed for the diagnosis, but provides incremental **prognostic** value
- In **HFmrEF**, strain may be useful for the **diagnosis** when LVEF is difficult to assess
- In **HFpEF**, strain can provide significant **diagnostic** and **prognostic** value
- In **HCM**, the degree of **wall thickening**, and severity of **fibrosis** correlate closely with abnormalities in regional and global **longitudinal** strain
- Regional variations in strain can be easily recognizable, accurate, & reproducible means of differentiating causes of LVH



Match the strain plot to the correct patient



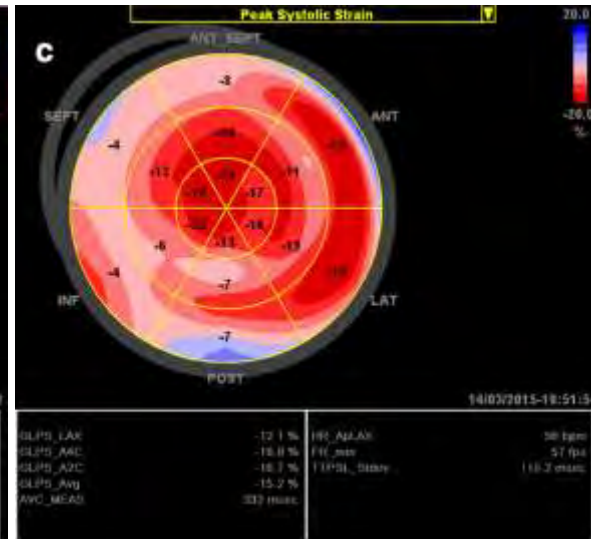
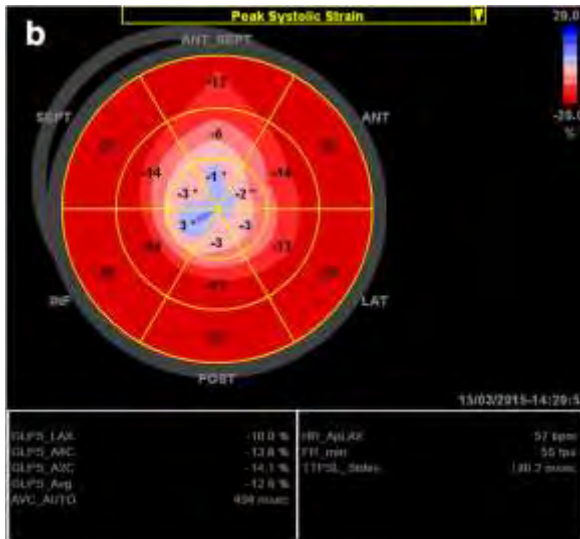
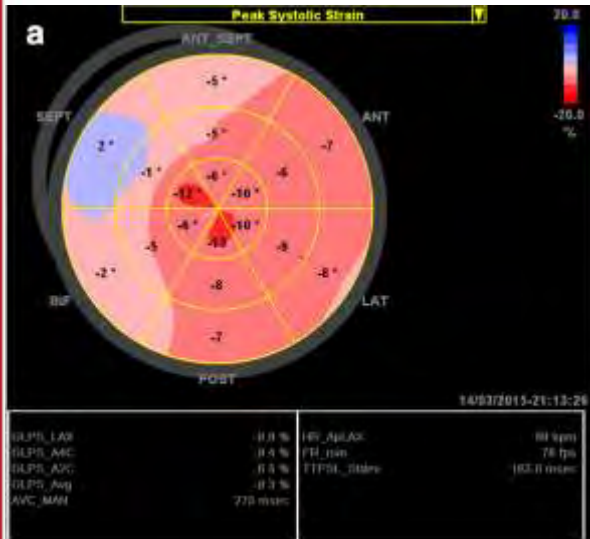
• In hypertensive patients with septal bulge, the bull's eye plot is characterized by a significantly reduced longitudinal strain (light red) at the basal part of the septum
58 yo F w HTN
LVPWd = 15 mm
IVSd = 15 mm
LVEF = 43 %
LV mass = Elevated

• In hypertensive patients with concentric LVH and normal EF, average GLS usually remains near normal, but significantly reduced LS patterns may be detected on multiple basal & mid myocardial levels
49 yo F w HTN
LVPWd = 15 mm
IVSd = 15 mm
LVEF = 75 %
LV mass = Elevated

• In cases with concentric LVH and reduced EF, reduced average global and segmental longitudinal strains are the usual findings
61 yo F w HTN & septal bulge
LVPWd = 9 mm
IVSd = 13 mm
LVEF = 65 %
LV mass = Normal



Match the strain plot to the correct patient



• In HCM patients with an asymmetric LVH, typical LS bull's eye plot pattern is characterized by significantly reduced average GLS with significantly reduced strain in hypertrophic regions

76 yo F w HCM
 LVPWd = 22 mm
 IVSd = 18 mm
 LVEF = 70 %
 LV mass = Elevated

• In apical HCM, the bull's eye plot displays blue or pale pink color at the apex with the loss of longitudinal deformation, surrounded by the red regions with normal strain

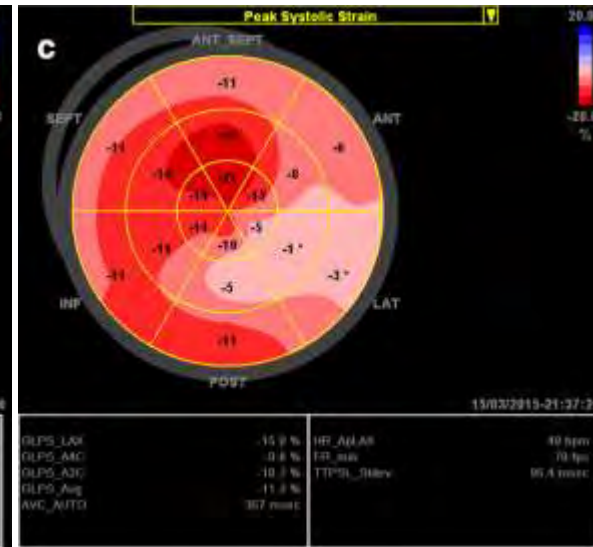
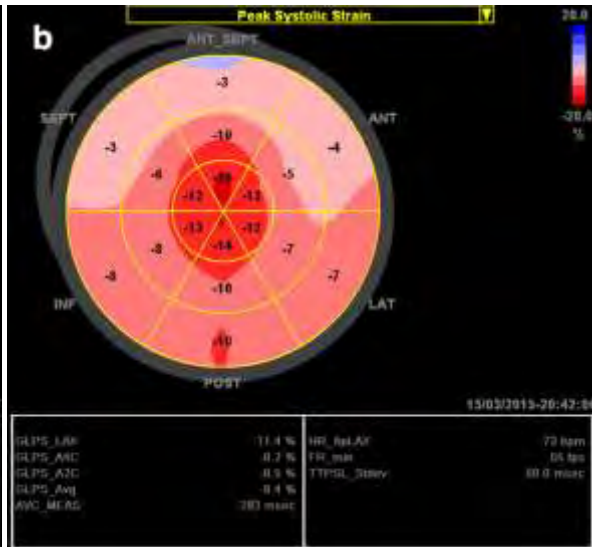
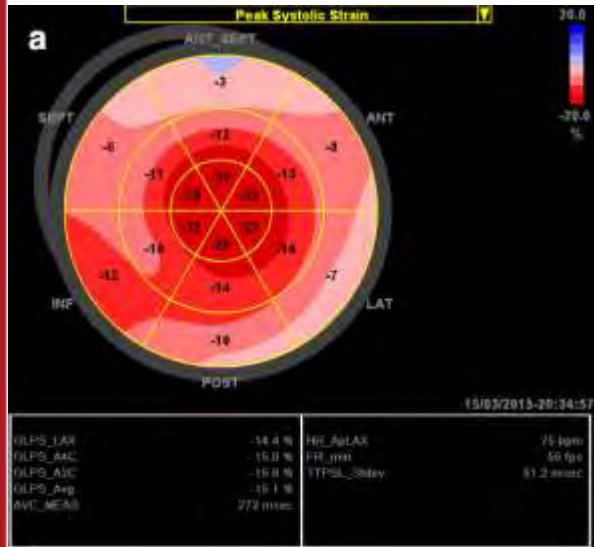
70 yo M w HCM
 LVPWd = 17 mm
 IVSd = 17 mm
 LVEF = 60 %
 LV mass = Elevated

• In HCM patients with concentric hypertrophy and normal EF (typically the bull's eye plot is characterized by a mildly reduced average GLS and prominently reduced strain in the hypertrophic segments)

59 yo M w HCM
 LVPWd = 9 mm
 IVSd = 9 mm
 LV apex = 20 mm
 EF = 60 %



Match the strain plot to the correct patient



• In CA w/normal EF, the bull's eye plot shows normal or slightly reduced average LS, normal LS at the apex (bright red), and a significantly reduced strain at all basal segments (pale pink to light red).
 • LVPWd = 14 mm
 IVSd = 14 mm
 • LVEF = 50 %
 Local thinning in the mid regions is also reduced in some individuals.

• With progressive CA, along with a decrease in LVEF, CA patients present with a reduced average GLS and gradual deterioration in apical LS on serial follow-up.
 • LVPWd = 13 mm
 IVSd = 13 mm
 LVEF = 65 %
 • As a result, the base-to-apex strain gradient is flattened as the segments to become smaller in the late stage of the disease in CA.
 • LV mass is Elevated

• In late stage ED, average GLS is reduced and the low LS (pale pink) could be detected in the basal and mid posterolateral segments due to progressive local myocardial thinning and replacement fibrosis.
 • LVPWd = 13 mm
 IVSd = 18 mm
 Basal Lateral wall = 11 mm
 EF = 72 %



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