Impact of Radiofrequency Catheter Ablation on Left Ventricular Twist, Synchronicity, and Performance in WPW syndrome

; 2D Speckle Tracking Echocardiographic Study

Division of Cardiology, Department of Internal Medicine

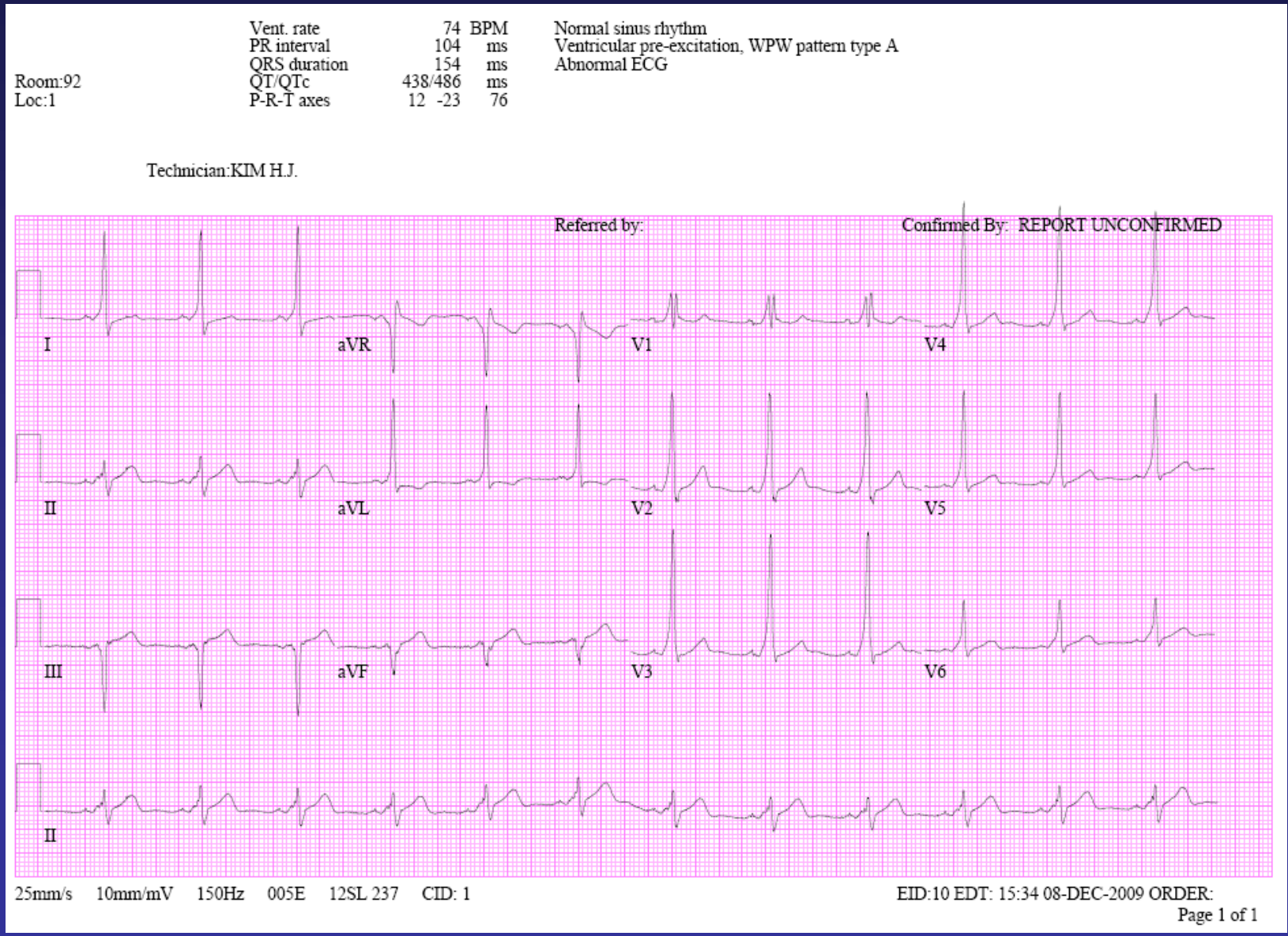
Seoul National University Hospital

Hyung-Kwan Kim, *Myung-Ki Seo, Sung-A Chang,*

Eue-Keun Choi, Yong-Jin Kim, Seil Oh, Goo-Yeong Cho

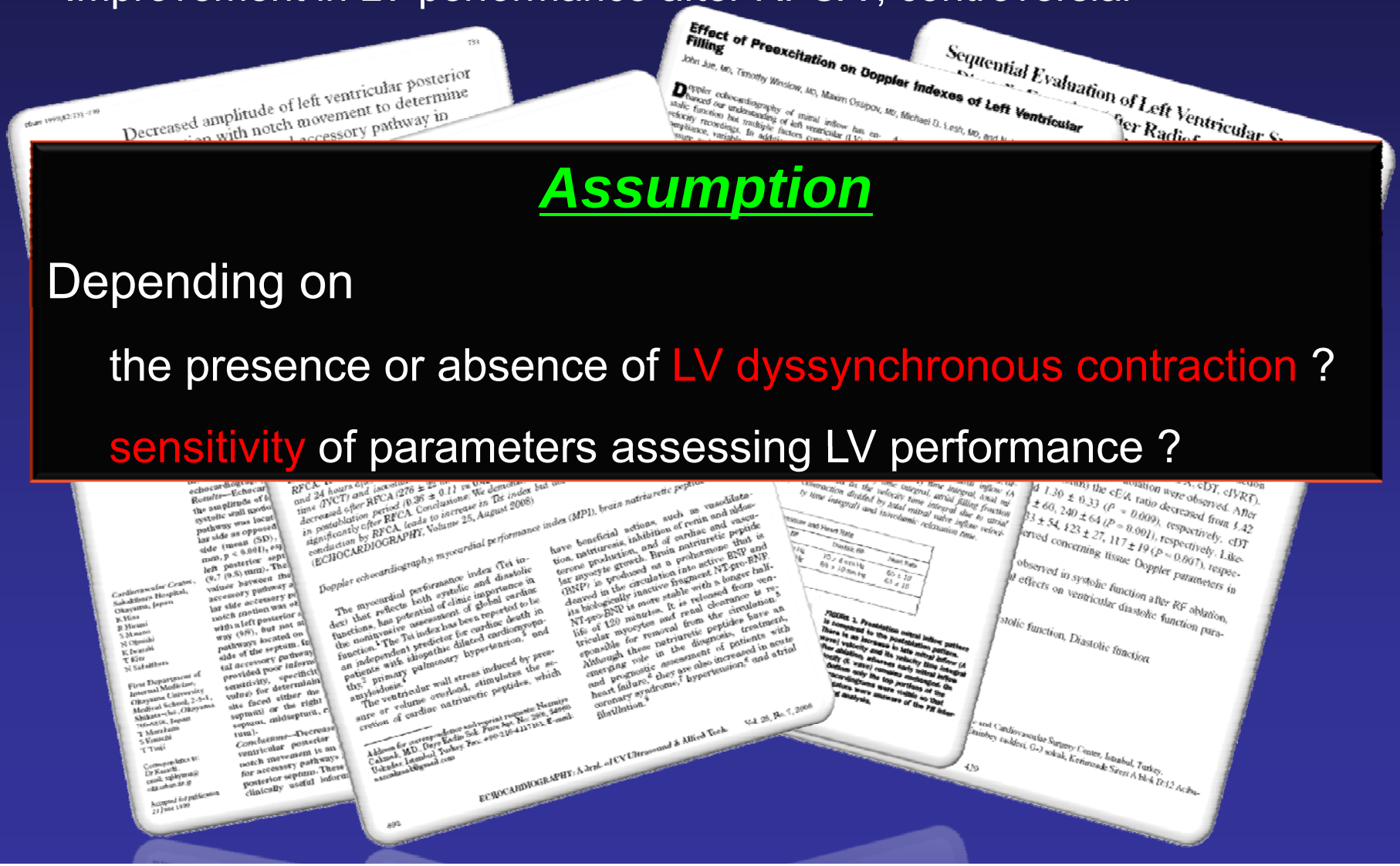
Dae-Won Sohn, Byung-Hee Oh, Young-Bae Park

WPW syndrome



WPW syndrome

- Dyssynchronous contraction before RFCA; relatively consistent
- Improvement in LV performance after RFCA ; controversial



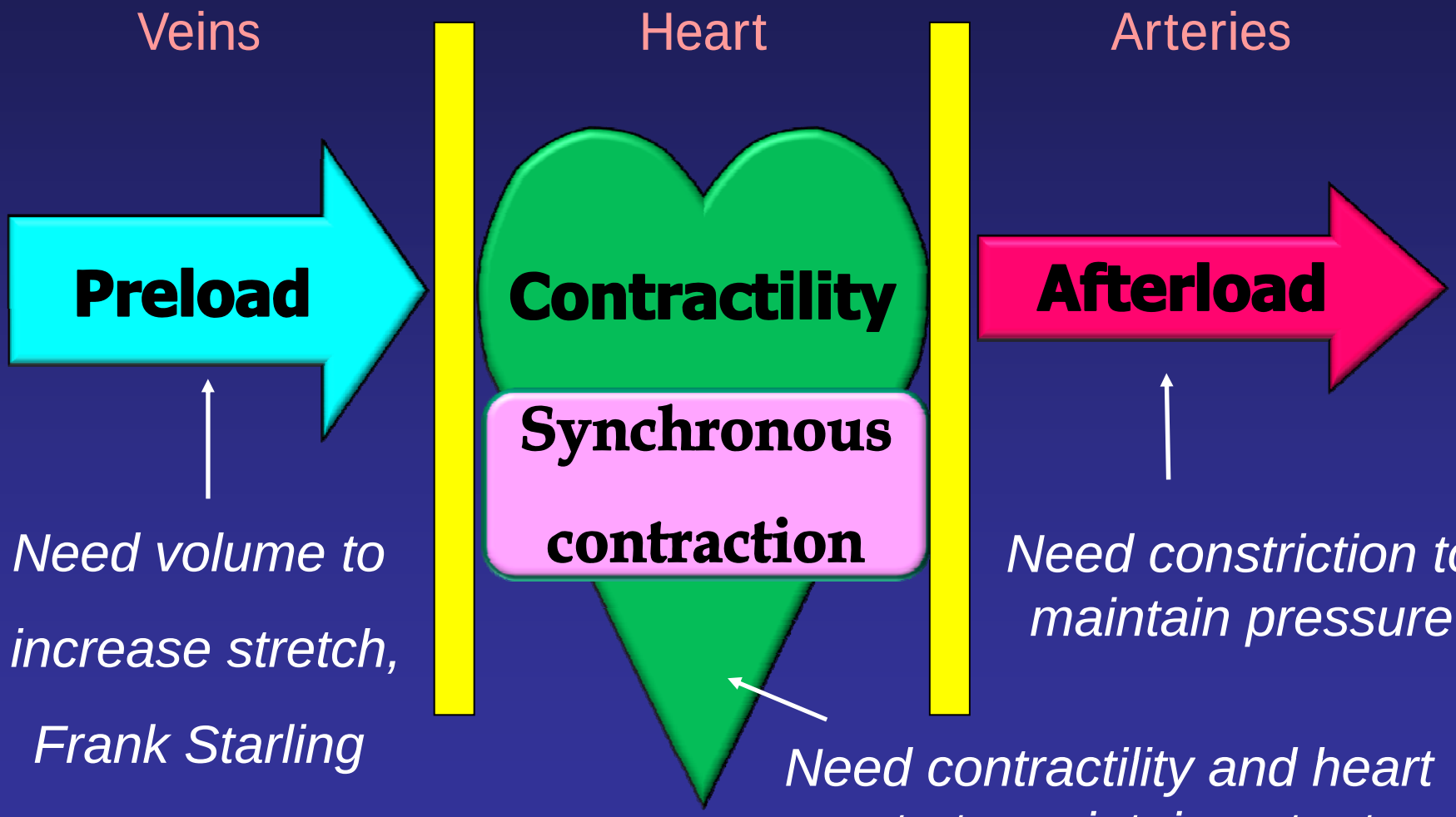
Assumption

Depending on

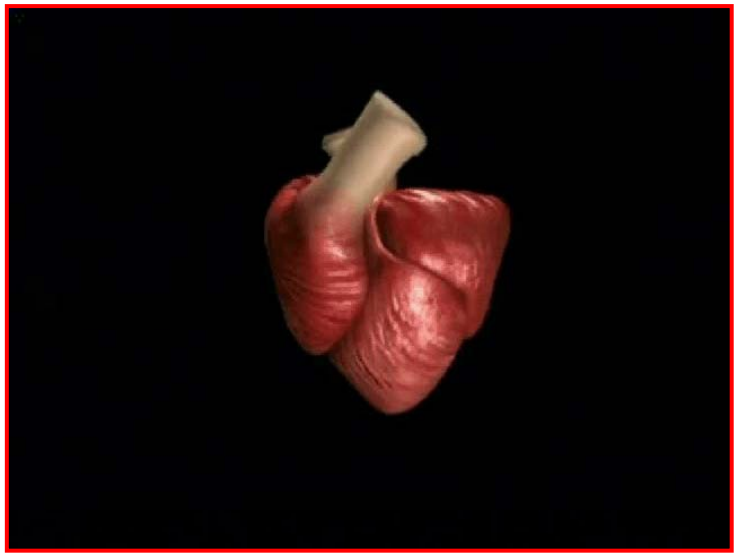
the presence or absence of LV dyssynchronous contraction ?

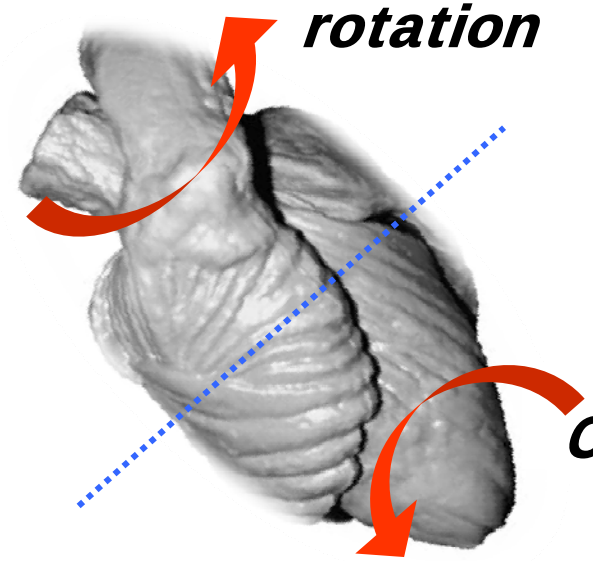
sensitivity of parameters assessing LV performance ?

Heart Functions



LV twist





Twist

as viewed from the apex

Circulation
Cardiovascular Imaging
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association
Learn and Live

Apical Rotation Assessed by Speckle-Tracking Echocardiography as an Index of Global Left Ventricular Contractility

Won-Jang Kim, MD; Byeong Han Lee, DVM; Yun Jeong Kim, RN, RDCS; Jee Hye Kang, RN, RDCS; Yoo Jin Jung, RN; Jong-Min Song, MD; Duk-Hyun Kang, MD; Jae-Kwan Song, MD

Background—Left ventricular (LV) apical rotation and twist can be estimated noninvasively by speckle-tracking echocardiography (STE). In this study, we tested whether apical rotation is an accurate index of LV contractility.

Methods and Results—We measured LV basal and apical rotation by STE in 11 open-chest anesthetized mongrel dogs under 8 different inotropic stages before and after ligation of either left anterior descending (n=6) or circumflex coronary artery (n=5). We measured LV pressure simultaneously with a high-fidelity pressure catheter and calculated LV ejection fraction (EF) with the biplane Simpson method and 2D echocardiography. Maximal positive dP/dt (dP/dt_{max}) was used as the gold standard measurement of LV contractility. We compared LV twist and apical rotation and EF against dP/dt_{max} by linear mixed model. LV apical rotation and twist showed dose-dependent increases and decreases after dobutamine and esmolol infusion, respectively. However, basal rotation did not change significantly during different inotropic conditions. There was a stronger association between dP/dt_{max} and LV twist (R^2=0.747, P<0.001) and apical rotation (R^2=0.726, P<0.001) than between dP/dt_{max} and EF (R^2=0.408, P<0.001), and this trend was more apparent with coronary ligation irrespective of the ligation site. There was also a high association between dP/dt_{max} and apical rotation alone, both with (R^2=0.805, P<0.001) and without (R^2=0.748, P<0.001) coronary ligation. Apical rotation alone showed comparable accuracy to LV twist. Apical rotational velocity also showed a high association with dP/dt_{max} (R^2=0.669, P<0.001) and LV twist (R^2=0.892, P<0.001).

Conclusions—Apical rotation assessed by STE is an effective noninvasive index of global LV contractility and is more closely related to dP/dt_{max} than LV EF. (***Circ Cardiovasc Imaging*. 2009;2:123-131.**)

Key Words: contractility ■ ventricular rotation ■ echocardiography

Physiologic variables influencing LV twist mechanics

Physiologic variables	LV twist	Er*
Increasing preload	↑	↓
Increasing afterload	↓	↓
Increasing contractility	↑	↑
Exercise	↑	↑

*** Early diastolic untwisting velocity**

1) Buchalter MB et al. Cardiovasc Res 1994;28:629–35.
2) Dong SJ et al. Am J Physiol 1999;277:H1053-60
3) Hansen DE et al. Circulation 1991;83:1315-26
4) MacGowan GA et al. Cardiovasc Res 1996;31:917-25
5) Gibbons Kroeker CA et al. Circulation 1995;92:130-41
6) Moon MR et al. Circulation 1994;89-142-50

LV twist-volume loops

Dong SJ et al. *Am J Physiol Heart Circ Physiol.* 1999;277:H1053-60.

Independent effects of preload, afterload, and contractility on left ventricular torsion

SHENG-JING DONG, PAUL S. HEES, WEN-MEI HUANG, SAM A. BUFFER, JR., JAMES L. WEISS, AND EDWARD P. SHAPIRO
Division of Cardiology, Johns Hopkins University School of Medicine, Johns Hopkins Bayview Medical Center, Baltimore, Maryland 21224

Dong, Sheng-Jing, Paul S. Hees, Wen-Mei Huang, Sam A. Buffer, Jr., James L. Weiss, and Edward P. Shapiro. Independent effects of preload, afterload, and contractility on left ventricular torsion. *Am. J. Physiol.* 277 (*Heart Circ. Physiol.* 46): H1053–H1060, 1999.—Shortening of oblique left ventricular (LV) fibers results in torsion. A unique relationship between volume and torsion is therefore expected, and the effects of load and contractility on torsion should be predictable. However, volume-independent behavior of torsion has been observed, and the effects of load on this deformation remain controversial. We used magnetic resonance imaging (MRI) with tagging to study the relationships between load and contractility, and torsion. In ten isolated, blood-perfused canine hearts, ejection was controlled by a servopump: end-diastolic volume (EDV) was controlled by manipulating preload parameters and end-systolic volume (ESV) by manipulating afterload using a three-element windkessel model. MRI was obtained at baseline, two levels of preload alteration, two levels of afterload alteration, and dobutamine infusion. An increase in EDV resulted in an increase in torsion at constant ESV (preload effect), whereas an increase in ESV resulted in a decrease in torsion at constant EDV (afterload effect). Dobutamine infusion increased torsion in association with an increase in LV peak-systolic pressure (PSP), even at identical EDV and ESV. Multiple regression showed correlation of torsion with preload (EDV), afterload (ESV), and contractility (PSP; $r = 0.67$). Furthermore, there was a close linear relationship between torsion and stroke volume (SV) and ejection fraction (EF) during load alteration, but torsion during dobutamine infusion was greater than expected for the extent of ejection. Preload and afterload influence torsion through their effects on SV and EF, and there is an additional direct inotropic effect on torsion that is independent of changes in volume but rather is force dependent. There is therefore potential for the torsion-volume relation to provide a load-independent measure of contractility that could be measured noninvasively.

left ventricle; twist; magnetic resonance imaging

CARDIAC TORSION is the relative rotation of the left ventricular (LV) apex with respect to the base, about LV long axis. During the past decade, this deformation has

(2). The effects of load and contractility on torsion should therefore be predictable. However, these remain controversial. Studies in transplanted human hearts (13, 21) suggest that torsion, as measured by radiopaque markers, is not affected by pressure or volume loading, whereas experiments in dog models report that torsion, as measured by markers (2) or an optical device (10, 11), is primarily a function of volume at end diastole and end systole and is preload and afterload dependent. Furthermore, although it has been well demonstrated that torsion is very sensitive to changes in contractility (6, 11, 13, 14, 21) (positive inotropic interventions increase torsion, whereas negative inotropic interventions decrease torsion), it has been difficult to distinguish whether this inotropic effect is mediated through (11) or is independent of (13) changes in volume.

A volume-independent component of torsion has been clearly observed; substantial recoil of torsion is known to occur between the time of aortic valve closure and mitral valve opening, when cavity volume is fixed (10, 11, 23), and systolic torsion has been demonstrated in isolated isovolumic beating hearts (20). Thus under some circumstances, torsion may vary with force, as well as with volume. Because inotropic stimulation enhances pressure (or force) generation at any given end-diastolic volume (EDV) and end-systolic volume (ESV; 17, 18), it may therefore have a direct effect on torsion that is not mediated through changes in volume but through changes in force.

To examine further the individual effects of preload, afterload, and contractility on torsion, we used magnetic resonance imaging (MRI) with tissue tagging (29), which permits the accurate measurement of torsion, to study an isolated, blood-perfused, ejecting heart model that allows independent control of these factors. We hypothesized that *1*) preload and afterload, through their effect on volume, affect torsion and *2*) there is a further direct inotropic effect on torsion that is independent of changes in volume but rather is force dependent.

rather is force dependent. There is therefore potential for the torsion-volume relation to provide a load-independent measure of contractility that could be measured noninvasively.

esthetized with pentobar-
e smaller dog of the pair
wt 21.2 ± 1.5 kg) and the
nean wt 30.1 ± 1.5 kg).
hydrocortisone (250 mg),
ministered to the support
lated (model 613; Harvard
Apparatus, South Natick, MA).

solely to indicate this fact.

LV twist-volume loops

Imaging

Enhanced Ventricular Untwisting During Exercise

A Mechanistic Manifestation of Elastic Recoil Described by Doppler Tissue Imaging

Yuichi Notomi, MD; Maureen G. Martin-Miklovic, RDCS; Stephanie J. Oryszak, BA, CCRP; Takahiro Shiota, MD; Dimitri Deserranno, PhD; Zoran B. Popovic, MD; Mario J. Garcia, MD; Neil L. Greenberg, PhD; James D. Thomas, MD

Background—The cascade of events by which early diastolic left ventricular (LV) filling increases with exercise is not fully elucidated. Doppler tissue imaging (DTI) can detect myocardial motion, including torsion, whereas color M-mode Doppler (CMM) can quantify LV intraventricular pressure gradients (IVPGs).

Methods and Results—Twenty healthy volunteers underwent echocardiographic examination with DTI at rest and during submaximal supine bicycle exercise. We assessed LV long-/short-axis function, torsion, volume, inflow dynamics, and early diastolic IVPG derived from CMM data. LV torsion and untwisting velocity increased with exercise (torsion, 11±4° to 24±8°; untwisting velocity, −2.0±0.7 to −5.6±2.3 rad/s) that was associated with an increase in IVPG (1.4±0.5 to 3.7±1.2 mm Hg). Untwisting in normal subjects occurred during isovolumic relaxation and early filling, significantly before long-axis lengthening or radial expansion. The clinical feasibility of this method was tested in 7 patients with hypertrophic cardiomyopathy (HCM); torsion was higher at rest but did not increase with exercise (16±4° to 14±6°), whereas untwisting was delayed and unenhanced (−1.6±0.8 to −2.3±1.2 rad/s). In concert, IVPG was similar at rest (1.2±0.3 mm Hg), but the

Valvular heart disease

Left ventricular torsion in primary chronic mitral regurgitation

A N Borg, J L Harrison, R A Argyle, S G Ray

ABSTRACT

Background: Torsion is essential for normal systolic and diastolic function of the left ventricle (LV), and is known to be abnormal in animal models of mitral regurgitation (MR). There are no comparable data in humans.

Objectives: To study LV torsion in humans with chronic primary MR using speckle-tracking echocardiography.

Methods: Rotation and rotation rate were measured

METHODS

Patient population

Our study population consisted of patients with chronic moderate-severe MR due to mitral valve prolapse and age-matched healthy controls. MR patients were selected on the basis of the presence of MR scoring ⩾3+ on colour Doppler imaging. All subjects were in sinus rhythm. Patients with significant disease of other valves, a history of

Heart failure and cardiomyopathy

Left ventricular twist mechanics in patients with apical hypertrophic cardiomyopathy: assessment with 2D speckle tracking echocardiography

S-A Chang,[1] H-K Kim,[2] D-H Kim,[2] J-C Kim,[3] Y-J Kim,[2] H-C Kim,[3] D-W Sohn,[2] B-H Oh,[2] Y-B Park[2]

ABSTRACT

Objective: Left ventricular (LV) apical rotation significantly contributes to LV twist, which has been reported to have a vital role in maintaining LV systolic and diastolic function. Apical hypertrophic cardiomyopathy (ApHCM) is a unique disease with pathological LV hypertrophy at the apex. We aimed (1) to evaluate LV twist mechanics in ApHCM and (2) to demonstrate the influence of predominantly local, pathological involvement of the apical myocardium on LV twist mechanics.

Methods: 21 patients diagnosed with ApHCM were consecutively enrolled and compared with normal controls. After a standard echocardiographic examination, we scanned parasternal basal and apical short-axis planes to quantify LV rotations and LV twist using the speckle tracking technique. For better understanding of LV twist mechanics in ApHCM, LV radial and biplanar strains and LV twist-volume curve were also evaluated.

stenosis[9 10] and asymmetric septal hypertrophic cardiomyopathy.[11]

Hypertrophic cardiomyopathy (HCM) is a genetic disorder that is characterised by a hypertrophied and non-dilated LV with variable myocardial involvement. Although HCM is classified based on the anatomical location of myocardial involvement, apical hypertrophic cardiomyopathy (ApHCM) was not included in the conventional classification.[12] Recently, ApHCM has been widely accepted to be a unique phenotype of HCM with a giant negative T wave in anterior leads of the electrocardiogram, pathological hypertrophy of the LV apex and a "spade-like" configuration of the LV cavity at end-diastole by two-dimensional (2D) echocardiography.[13] Although LV twist mechanics have been investigated in HCM patients with asymmetrical septal hypertrophy,[11] our under-

Original articles

Role of pericardium in the maintenance of left ventricular twist

Sung-A Chang, Hyung-Kwan Kim, Yong-Jin Kim, Goo-Yeong Cho, Seil Oh, Dae-Won Sohn

ABSTRACT

Background The role of pericardium in left ventricular (LV) twist has not been directly investigated. We sought to determine the role of pericardium in maintenance of LV twist function in an animal experiment, before and after pericardial opening.

Methods 13 mongrel dogs were initially operated on, but two dogs were excluded from the final analyses owing to poor speckle tracking. Intraoperative echocardiography for conventional and speckle tracking measurements was performed at baseline with intact pericardium, and after pericardial opening. Using the speckle tracking technique, LV twist and strains were obtained before and after pericardial opening in 11 animals and additionally after pericardial repair in five animals.

Left ventricular (LV) twist is a complex myocardial motion that is generated by the dynamic interaction between oppositely wound epicardial and endocardial myocardial fibre helices.[6 7] From the functional perspective, LV twist with ensuing untwist motion has been reported to contribute significantly to maintain LV systolic and diastolic functions.[8–13] Recently, a decrease in LV twist was reported in a small number of patients with congenital total absence of pericardium.[6] We believed that this finding requires confirmation by human study or animal experiment, because in this earlier study an indirect comparison of changes in the extent of LV twist were conducted in completely different populations—that is, in normal controls

Questions ?

independently of preload or afterload……

RFCA *in WPW patients*

LV dyssynchrony improvement ?

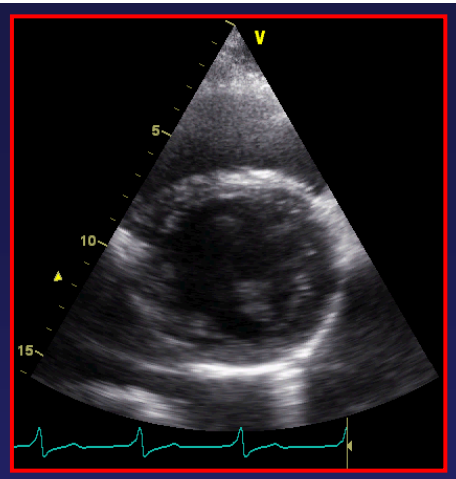
LV twist ?

Correlation ?

LV performance ?

Using LV twist-volume curve

WPW syndrome patients with clinical indication of RFCA



Pre-excitation (+) heart

RFCA

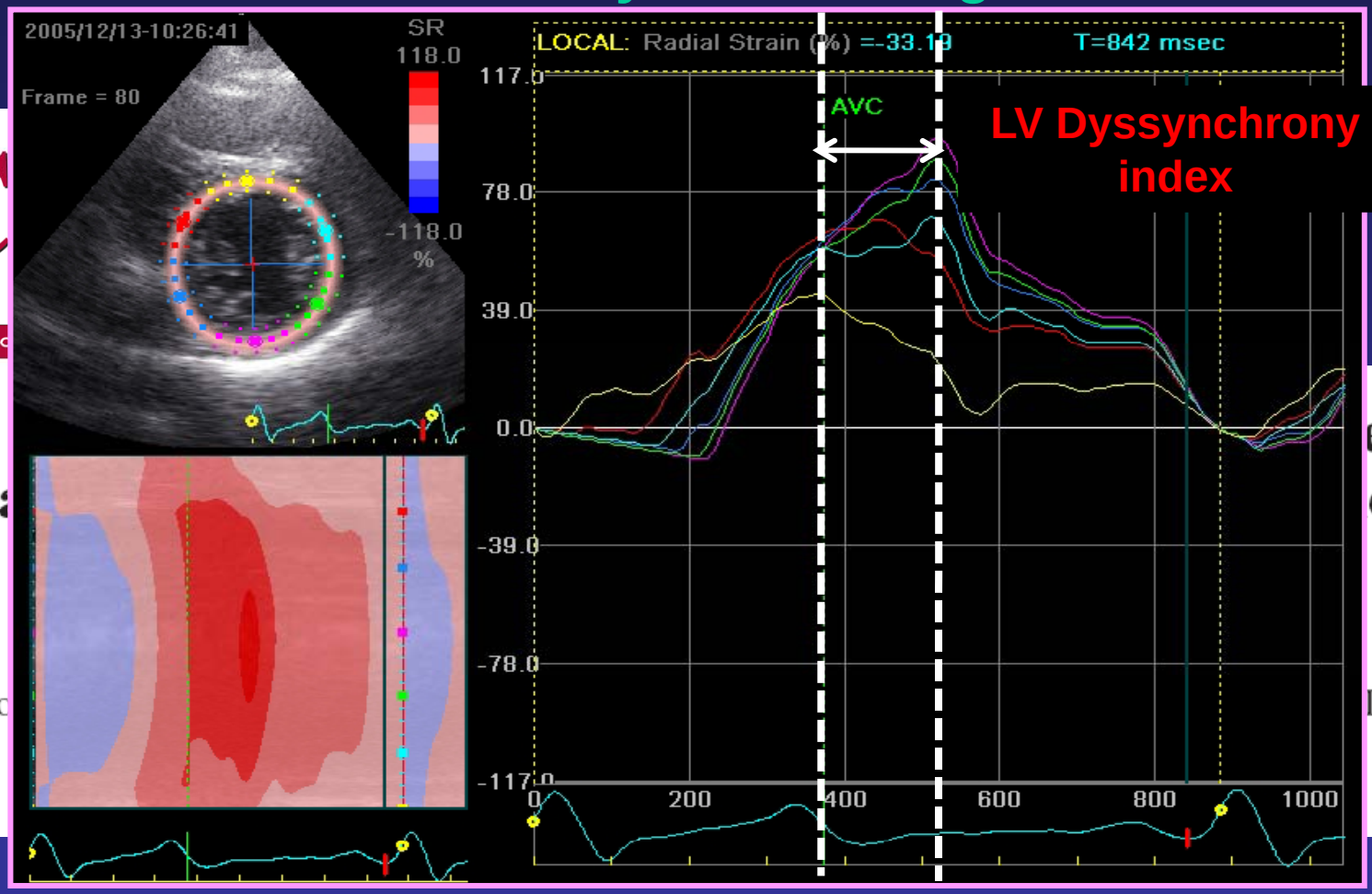
- **LV contraction pattern**
  - ✓ Heterogeneity in time-to-peak radial strain for 6 mid-myocardial segments
  - ✓ Synchrony between the basal and apical rotation
- **LV twist**
- **LV systolic function**
- **LV diastolic function**

Pre-excitation (-) heart

LV dyssynchrony assessment

Heterogeniety in time-to-peak radial strain

for 6 mid-myocardial segments



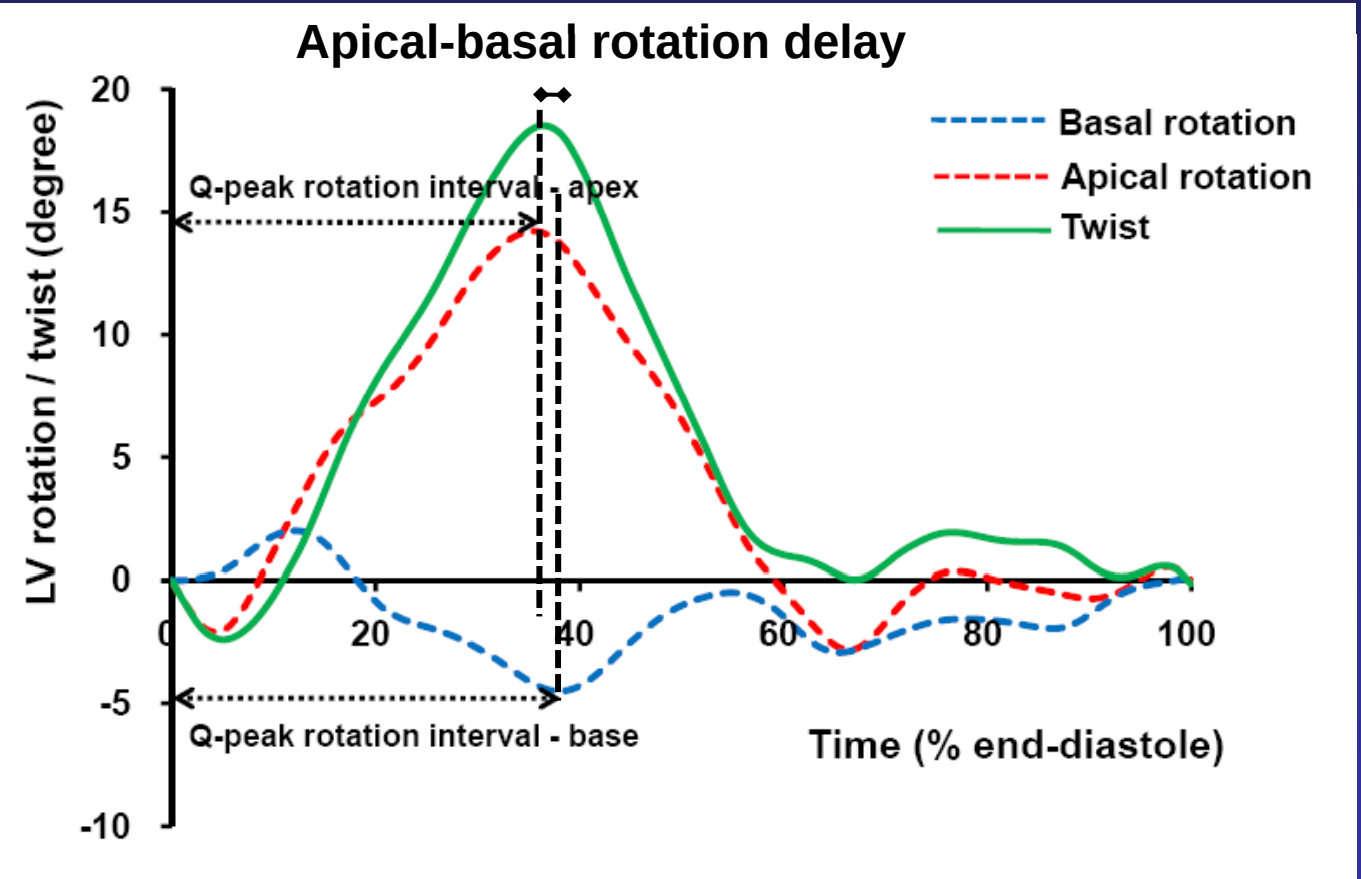
LV dyssynchrony index : maximal delay among 6 segments at the level of papillary muscle

LV dyssynchrony assessment

Synchrony between the basal and apical rotation

(Q-peak rotation interval)

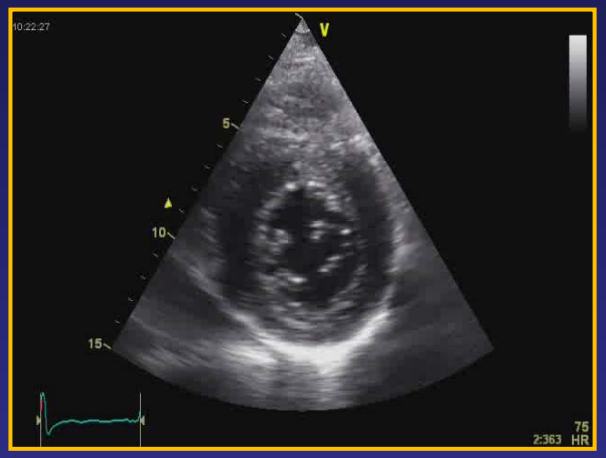
; difference in time interval from Q wave onset to peak rotation



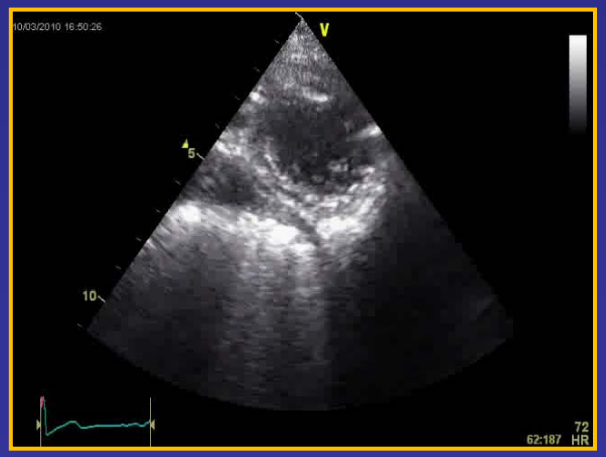
LV Twist

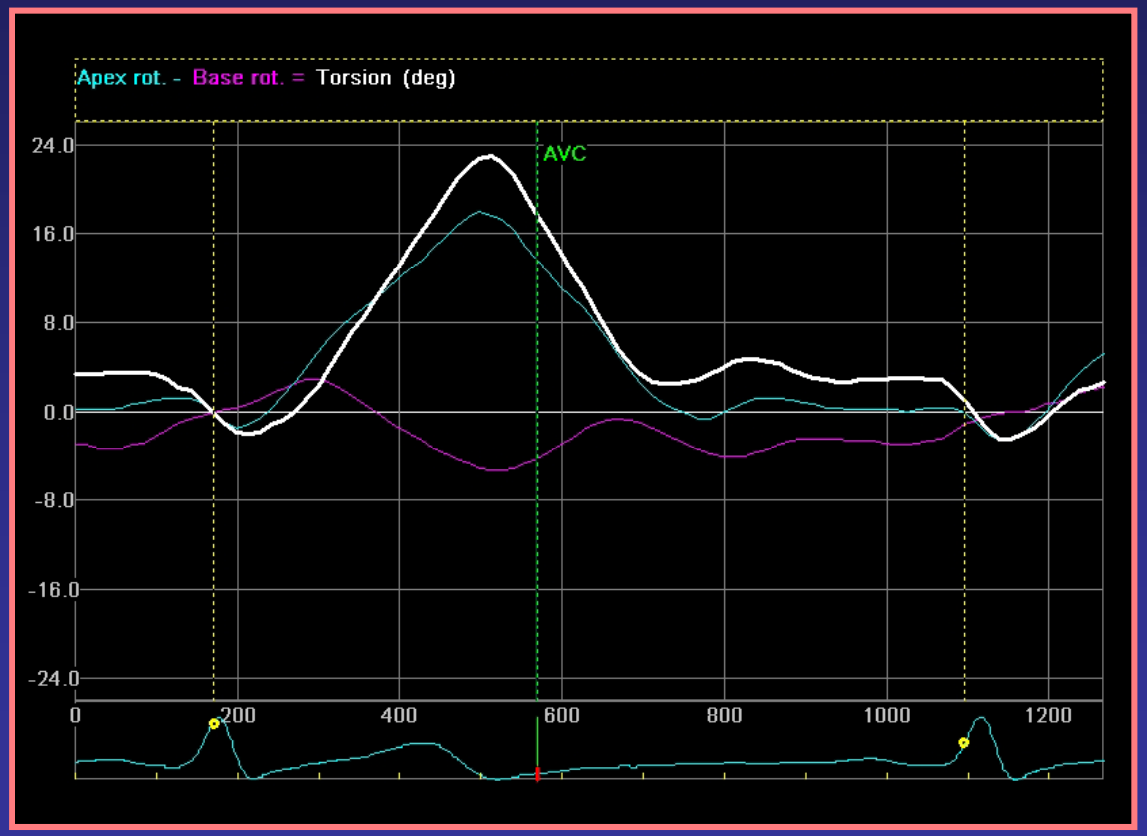
"*Net difference*" between apical and basal rotation

Basal level



Apical level





LV twist-volume loops

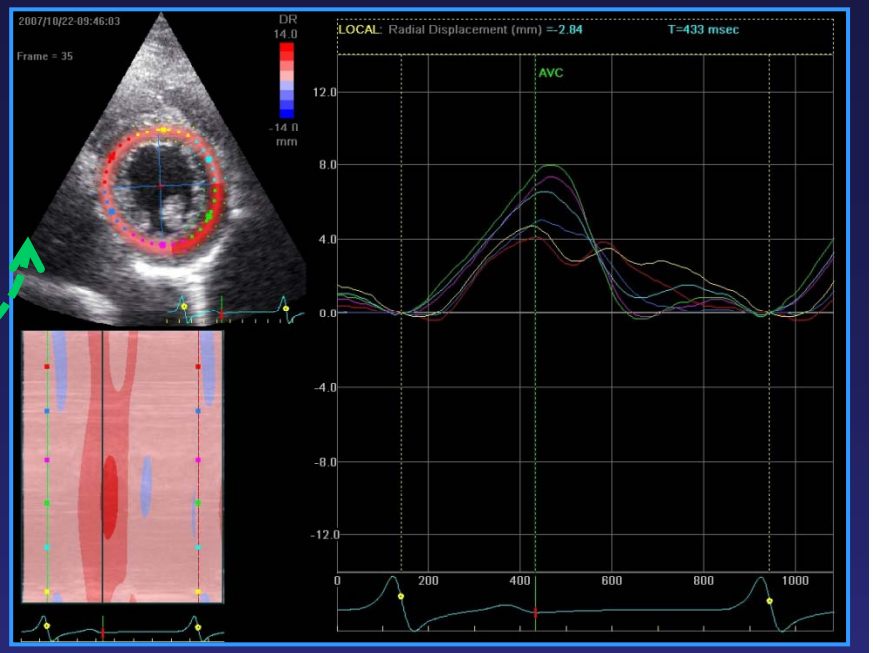
Volume calculation

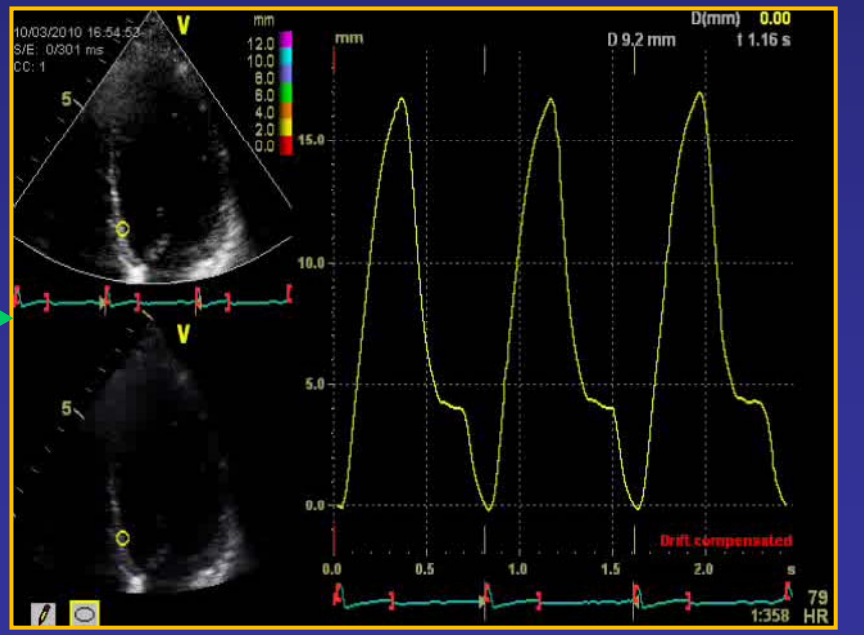
$$V(t) = \pi/6 \times L(t) \times S(t)^2$$

L (t) : long axis length

S (t) : short-axis dimension

V (t) : volume at time of t





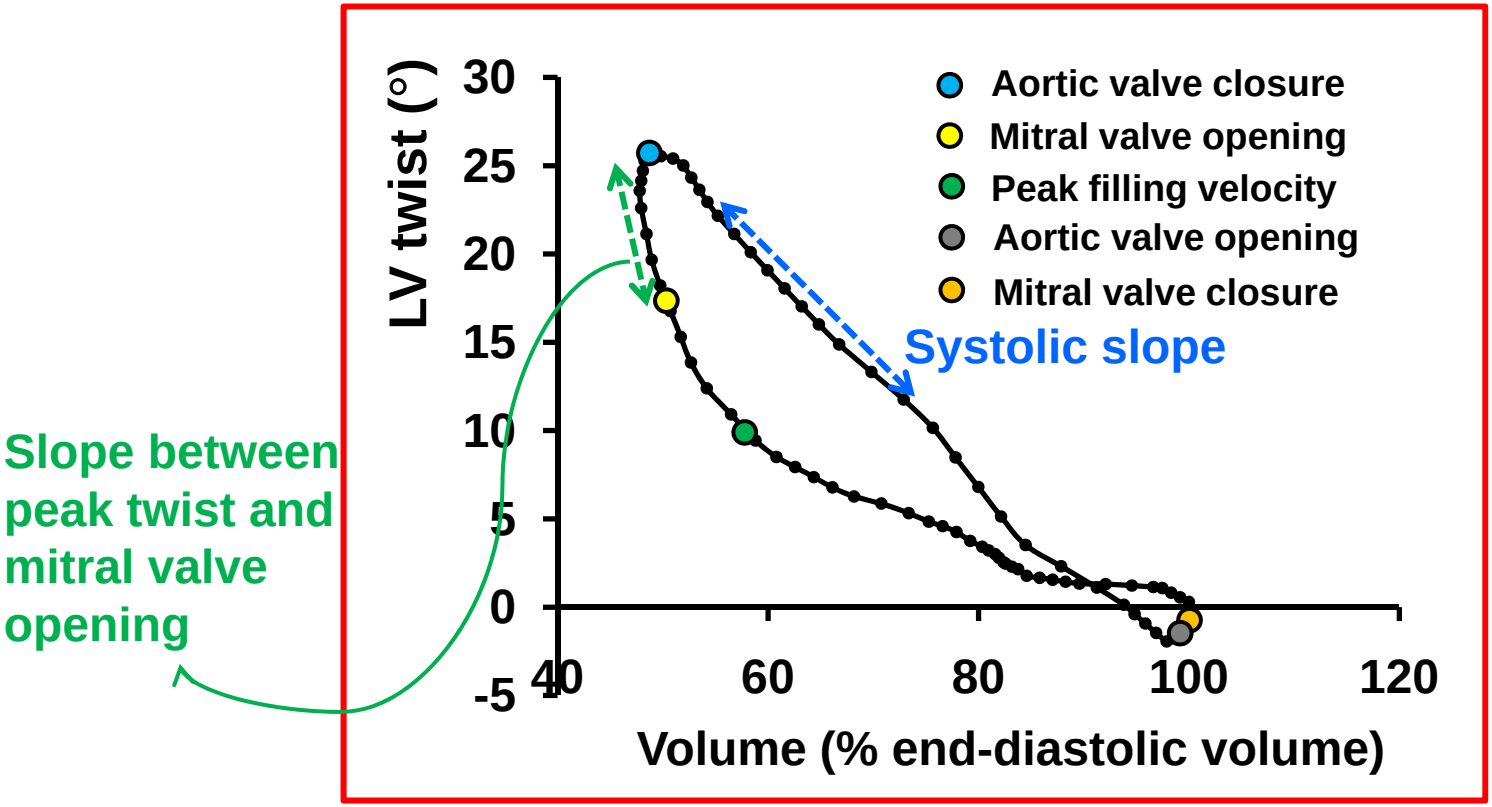
Notomi et al. *Circulation* 2006;2534-41.

Borg AN et al. *Heart* 2008;94:597-603.

Chang SA and Kim HK et al. *Heart* 2010;96:49-55.

Chang SA and Sohn DW et al. *Heart* 2010. *In Press*

LV twist-volume loops



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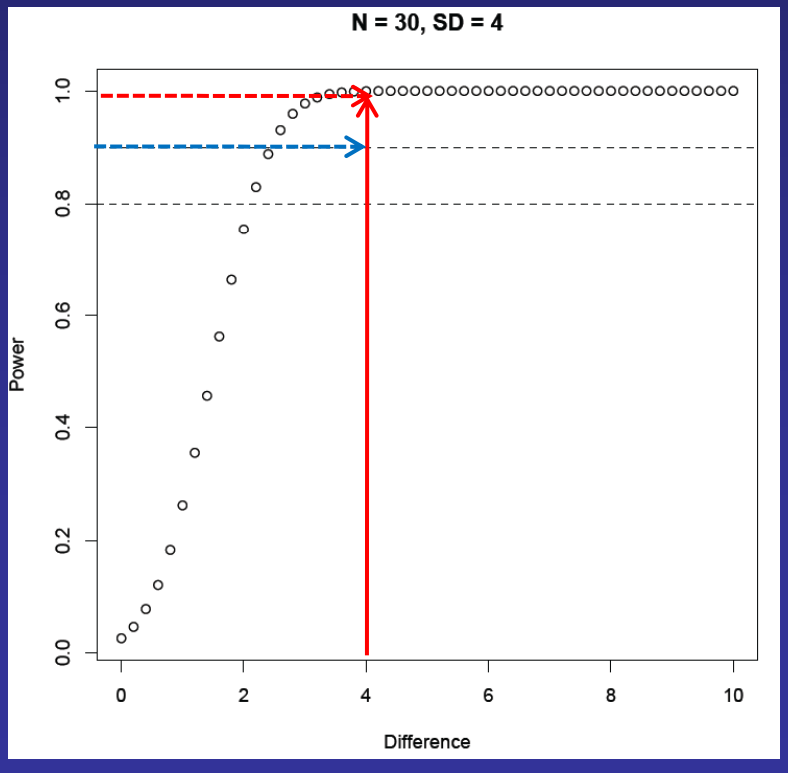
Sample size calculation

- α error = 0.05, β error 0.20, statistical power = 90%
- about 4° change in the LV twist could be found

  for a SD of 6°, based on our earlier experiences **



20% exclusion rate

due to difficulty in speckle tracking technique

40 patients should be enrolled

** Chang SA and Kim HK et al. *Heart* 2010;96:49-55.

Chang SA and Sohn DW et al. *Heart* 2010. *In Press*

Demographics

Initially 40 patients → Finally 34 patients

clinically indicated for RFCA

Congenital heart disease (n=1)

Coronary artery disease (n=1)

Not in sinus rhythm (n=1)

Arrhythmic attack within 1 weak of RFCA (Af) (n=1)

Inadequate speckle tracking (n=2)

- BMI ; 23.0 ± 3.6 Kg/m^2
- Age ; 32±14 years
- Accessory pathway
  - Left side ; 27 (79.4%)
  - Right side ; 17 (20.6%)

Clinical characteristics

In entire population…

N = 34	Pre-RFCA	Post-RFCA	*P* value
Clinical variables			
Age (years)	32 ± 14		…
Median (interquartile range)	27 (20, 43)		
Male (%)	19 (55.9%)		…
BMI (kg/m^2)	23.0 ± 3.6		…
SBP (mmHg)	123 ± 16	122 ± 11	0.62
DBP (mmHg)	71 ± 12	71 ± 8	0.74
Heart rate (bpm)	68 ± 10	68 ± 7	0.86
Median (interquartile range)	67.5 (62, 70)	67 (63, 71)	
Diabetes mellitus (%)	1 (2.9%)		…
Hypertension (%)	2 (5.9%)		…
QRS duration (msec)	133.4 ± 19.6	93.6 ± 12.3	< 0.001

Conventional echocardiography

In entire population…

N = 34	Pre-RFCA	Post-RFCA	*P* value
Conventional echocardiographic variables			
IVSd (mm)	8.4 ± 1.4	8.4 ± 1.4	0.95
LVPWd (mm)	8.1 ± 1.4	8.2 ± 1.5	0.61
LV-EDV (mL)	115.6 ± 24.1	113.6 ± 20.3	0.34
LV-ESV (mL)	45.4 ± 13.6	43.7 ± 10.9	0.31
LV-EF (%)	61.0 ± 6.2	61.9 ± 5.1	0.38
E (m/sec)	0.71 ± 0.13	0.71 ± 0.14	0.65
A (m/sec)	0.46 ± 0.12	0.46 ± 0.12	0.79
E/A ratio	1.65 ± 0.56	1.68 ± 0.66	0.55
DT (msec)	169.0 ± 28.4	169.5 ± 29.4	0.94
S' (m/sec)	0.07 ± 0.01	0.08 ± 0.01	0.51
E' (m/sec)	0.09 ± 0.02	0.10 ± 0.02	0.17
A' (m/sec)	0.07 ± 0.02	0.07 ± 0.02	0.42
E/E' ratio	7.5 ± 1.3	7.4 ± 1.7	0.58
LV-ESWS (g/cm^2)	52.5 ± 14.9	50.6 ± 10.0	0.41

Speckle tracking echocardiography

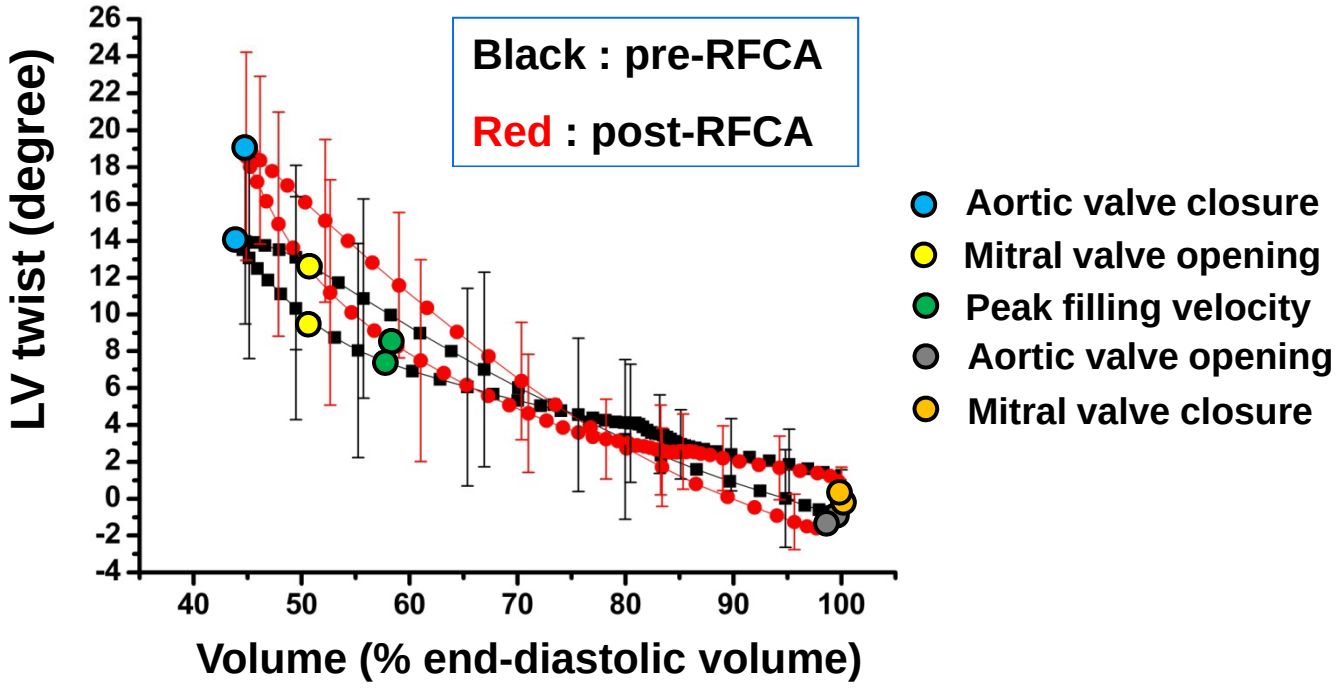
In entire population…

N = 34	Pre-RFCA	Post-RFCA	*P* value
Speckle tracking echocardiographic variables			
Frame rate	93 ± 22	88 ± 19	0.21
Median (interquartile range)	86 (70, 119)	86 (70, 104)	
LVdys (msec)	49.4 ± 42.9	20.4 ± 21.4	0.002
Median (interquartile range)	33.5 (14.0, 84.3)	14.0 (11.5, 21.8)	
Basal rotation (degree)	-4.3 ± 2.5	-5.8 ± 2.4	0.002
Median (interquartile range)	-3.5 (-6.8, -2.4)	-6.3 (-7.5, -4.1)	
Apical rotation (degree)	11.3 ± 4.9	13.8 ± 5.2	0.013
Median (interquartile range)	10.4 (7.5, 13.8)	12.9 (10.6, 17.7)	
LV twist (degree)	14.6 ± 6.1	18.9 ± 6.0	0.002
Median (interquartile range)	14.2 (9.1, 18.4)	19.7 (15.0, 22.6)	
LV twisting rate (degree/sec)	102.3 ± 36.0	125.3 ±34.2	0.003
Median (interquartile range)	98.1 (76.8, 126.9)	123.1 (104.4, 147.1)	
LV untwisting rate (degree/sec)	-115.7 ± 42.7	-146.5 ±57.1	0.004
Median (interquartile range)	-111.6 (-134.4, -83.1)	-138.8 (-194.0, -101.3)	
Q-peak rotation interval – base (%)	41.6 ± 18.4	43.7 ± 11.3	0.28
Median (interquartile range)	37.7 (31.9, 47.7)	42.1 (35.0, 48.2)	
Q-peak rotation interval - apex (%)	42.9 ± 15.1	38.2 ± 5.7	0.34
Median (interquartile range)	38.9 (33.6, 44.0)	37.4 (34.6, 41.6)	
Apical – basal rotation delay (%)	15.8 ± 14.9	6.8 ± 9.7	0.004
Median (interquartile range)	9.7 (3.5, 23.7)	3.3 (1.3, 8.0)	

Speckle tracking echocardiography

In entire population…

N = 34	Pre-RFCA	Post-RFCA	*P* value
LV twist-volume curve			
Systolic slope	-0.31 ± 0.10	-0.39 ± 0.10	< 0.001
Median (interquartile range)	-0.30 (-0.35, -0.23)	-0.39 (-0.48, -0.30)	
Early diastolic slope	-0.96 ± 0.57	-1.37 ± 0.74	0.02
Median (interquartile range)	-0.75 (-1.28, -0.48)	-1.24 (-1.74, -0.66)	



Clinical characteristics

Gr1 ; with LVdys improvement

Gr2 ; without LVdys improvement

with cutoff value of 20 msec improvement

Variables	Gr1 (n=17)			Gr2 (n=17)		
	Pre-RFCA	Post-RFCA	*P* value	Pre-RFCA	Post-RFCA	*P* value
Clinical variables						
Age (years)	32 ± 14		…	32 ± 14		…
Median (interquartile range)	27 (21, 40)			23 (20, 44)		
Male (%)	8 (47.1%)		…	11 (64.7%)		…
BMI (kg/m^2)	22.4 ± 3.0		…	23.6 ± 4.1		…
SBP (mmHg)	124 ± 18	119 ± 9	0.18	122 ± 14	124 ± 12	0.30
DBP (mmHg)	73 ± 12	71 ± 10	0.35	69 ± 11	70 ± 7	0.79
Heart rate (bpm)	68 ± 12	68 ± 8	0.53	70 ± 8	70 ± 7	0.93
Median (interquartile range)	66 (58, 69)	67 (62, 70)		69 (67, 71)	69 (67, 75)	
Diabetes mellitus (%)	1 (5.9%)		…	0 (0%)		…
Hypertension (%)	1 (5.9%)		…	1 (5.9%)		…
QRS duration (msec)	137.2 ± 23.7	93.3 ± 13.6	< 0.001	129.5 ± 14.1	93.4 ± 11.3	< 0.001

Conventional echocardiography

Gr1 ; with LVdys improvement

Gr2 ; without LVdys improvement

Variables	Gr1 (n=17)			Gr2 (n=17)		
	Pre-RFCA	Post-RFCA	*P* value	Pre-RFCA	Post-RFCA	*P* value
Conventional echocardiographic variables						
IVSd (mm)	8.5 ± 1.3	8.4 ± 1.4	0.68	8.2 ± 1.5	8.3 ± 1.5	0.68
LVPWd (mm)	8.3 ± 1.7	8.1 ± 1.6	0.63	8.0 ± 1.1	8.3 ± 1.5	0.14
LV-EDV (mL)	112.6 ± 22.0	112.1 ± 17.1	0.87	118.6 ± 26.4	115.1 ± 23.5	0.21
LV-ESV (mL)	42.4 ± 12.1	41.9 ± 9.3	0.83	48.3 ± 14.8	45.4 ± 12.4	0.24
LV-EF (%)	62.4 ± 6.6	62.8 ± 5.6	0.81	59.6 ± 5.7	60.9 ± 4.4	0.33
E (m/sec)	0.75 ± 0.13	0.75 ± 0.15	0.98	0.66 ± 0.12*	0.68 ± 0.13	0.60
A (m/sec)	0.43 ± 0.10	0.45 ± 0.11	0.45	0.49 ± 0.13	0.48 ± 0.14	0.75
E/A ratio	1.84 ± 0.55	1.83 ± 0.76	0.92	1.46 ± 0.51*	1.53 ± 0.54	0.06
DT (msec)	167.9 ± 19.2	163.7 ± 23.9	0.55	170.2 ± 36.0	175.4 ± 33.6	0.63
S' (m/sec)	0.08 ± 0.02	0.08 ± 0.02	0.79	0.07 ± 0.01	0.08 ± 0.01	0.52
E' (m/sec)	0.09 ± 0.02	0.10 ± 0.02	0.46	0.09 ± 0.02	0.10 ± 0.02	0.25
A' (m/sec)	0.07 ± 0.02	0.07 ± 0.02	0.37	0.07 ± 0.02	0.07 ± 0.02	0.93
E/E' ratio	7.7 ± 0.9	7.6 ± 1.7	0.64	7.3 ± 1.7	7.2 ± 1.7	0.75
LV-ESWS (g/cm^2)	49.4 ± 15.8	49.3 ± 9.0	0.97	55.6 ± 13.6	51.8 ± 11.2	0.26

* $P<0.05$ vs. Pre-RFCA value for Gr1

† $P<0.001$ vs. Pre-RFCA value for Gr1

‡ $P<0.05$ vs. Post-RFCA value for Gr1

§ $P<0.01$ vs. Post-RFCA value for Gr1

Speckle tracking echocardiography

Gr1 ; with LVdys improvement

Gr2 ; without LVdys improvement

Variables	Gr1 (n=17)			Gr2 (n=17)		
	Pre-RFCA	Post-RFCA	*P* value	Pre-RFCA	Post-RFCA	*P* value
Speckle tracking echocardiographic variables						
Frame rate	93 ± 24	85± 20	0.15	92 ± 21	91 ± 18	0.93
Median (interquartile range)	88 (70, 119)	85 (70, 104)		85 (71, 118)	85 (73, 107)	
LVdys (msec)	83.2 ± 36.1	16.6 ± 15.2	< 0.001	15.7 ± 8.5†	24.2 ± 26.0	0.24
Median (interquartile range)	84.0 (59.0, 88.5)	14.0 (6.5, 16.0)		14.0 (12.0, 22.5)	17.0 (11.0, 27.5)	
Basal rotation (degree)	-4.9 ± 2.4	-6.8 ± 2.4	0.025	-3.6 ± 2.5	-4.9 ± 2.1‡	0.04
Median (interquartile range)	-4.9 (-6.9, -2.9)	-6.4 (-8.8, -5.2)		-2.7 (-6.1, -1.7)	-4.6 (-6.5, -3.3)	
Apical rotation (degree)	11.5 ± 5.5	15.6 ± 5.5	0.006	11.0 ± 4.5	11.9 ± 4.1‡	0.52
Median (interquartile range)	10.3 (6.6, 14.4)	15.1 (11.9, 19.0)		10.5 (7.7, 13.2)	11.8 (8.8, 14.9)	
LV twist (degree)	16.0 ± 6.7	22.1 ± 4.5	0.006	13.2 ± 5.4	15.8 ± 5.6§	0.14
Median (interquartile range)	13.9 (12.0, 18.5)	22.2 (19.1, 24.6)		14.5 (8.4, 17.9)	15.7 (9.7, 20.0)	
LV twisting rate (degree/sec)	100.2 ± 34.3	132.9 ± 26.0	0.005	104.3 ± 38.6	117.7 ± 40.1	0.19
Median (interquartile range)	95.2 (78.6, 118.0)	127.3 (107.8, 159.5)		101.1 (75.2, 127.0)	111.1 (86.2, 129.7)	
LV untwisting rate (degree/sec)	-122.9 ± 49.6	-158.6 ± 58.5	0.06	-108.5 ± 34.5	-134.5 ± 54.8‡	0.06
Median (interquartile range)	-112.6 (-148.9, -83.1)	-142.2 (-194.7, -112.2)		-105.1 (-129.4, -79.6)	-124.1 (-179.5, -95.3)	
Q-peak rotation interval – base (%)	38.2 ± 16.5	39.1 ± 6.6	0.33	45.0 ± 19.9	48.4 ± 13.1‡	0.49
Median (interquartile range)	36.2 (30.9, 43.9)	37.3 (33.2, 46.1)		39.0 (32.5, 53.8)	46.4 (39.4, 56.7)	
Q-peak rotation interval - apex (%)	47.6 ± 18.4	37.8 ± 4.8	0.04	38.1 ± 9.4	38.6 ± 6.5	0.38
Median (interquartile range)	39.6 (34.6, 60.9)	37.3 (35.2, 40.2)		38.8 (33.1, 42.0)	37.8 (32.8, 43.2)	
Apical – basal rotation delay (%)	19.2 ± 16.5	3.5 ± 2.7	0.002	12.5 ± 12.6	10.2 ± 12.7	0.46
Median (interquartile range)	20.0 (4.0, 37.9)	2.9 (1.3, 5.2)		7.5 (2.6, 21.3)	3.4 (0.7, 19.2)	

Speckle tracking echocardiography

Gr1 ; with LVdys improvement

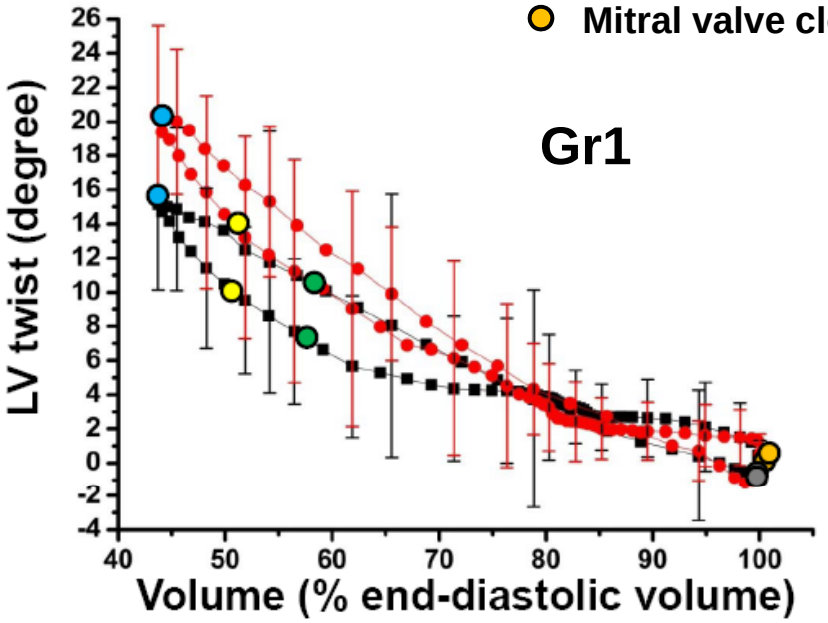
Gr2 ; without LVdys improvement

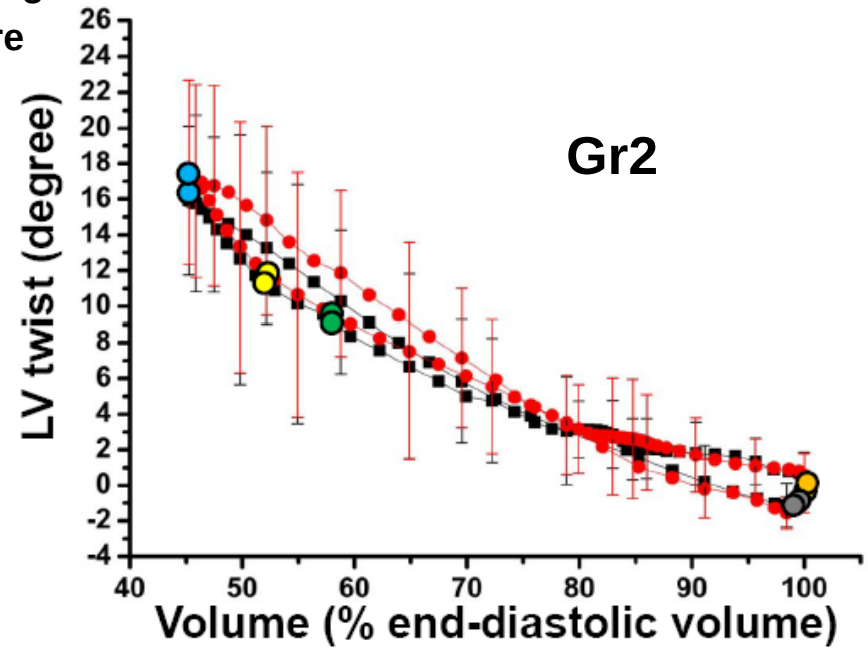
Variables	Gr1 (n=17)			Gr2 (n=17)		
	Pre-RFCA	Post-RFCA	*P* value	Pre-RFCA	Post-RFCA	*P* value
LV twist-volume curve						
Systolic slope	-0.33 ± 0.12	-0.44 ± 0.09	0.002	-0.28 ± 0.07	-0.32 ± 0.09	0.09
Median (interquartile range)	-0.30 (-0.37, -0.29)	-0.44 (-0.49, -0.38)		-0.29 (-0.32, -0.22)	-0.30 (-0.38, -0.25)	
Early diastolic slope	-0.91 ± 0.52	-1.49 ± 0.81	0.046	-1.01 ± 0.66	-1.22 ± 0.65	0.29
Median (interquartile range)	-0.69 (-1.33, -0.49)	-1.17 (-1.75, -0.70)		-0.80 (-1.26, -0.47)	-1.17 (-1.70, -0.59)	

- Aortic valve closure
- Mitral valve opening
- Peak filling velocity
- Aortic valve opening
- Mitral valve closure

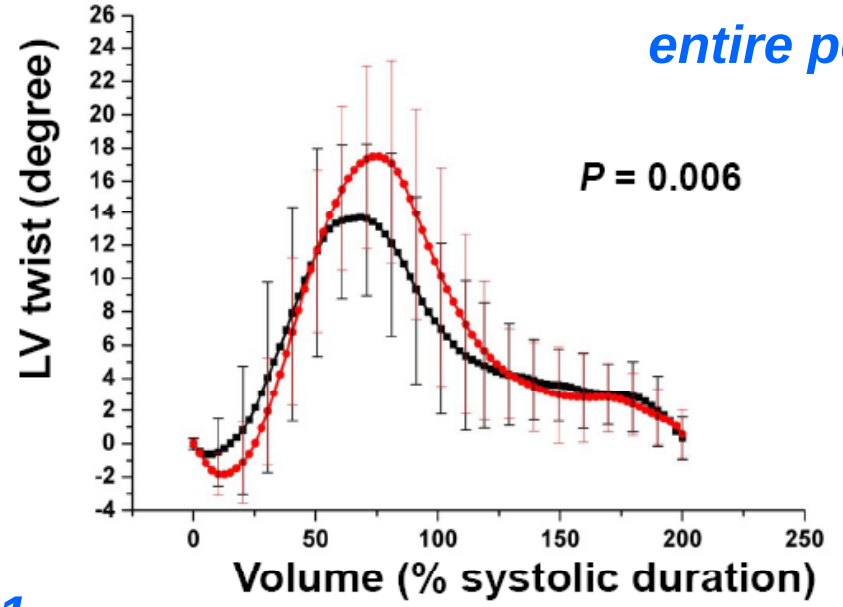
Black : pre-RFCA

Red : post-RFCA



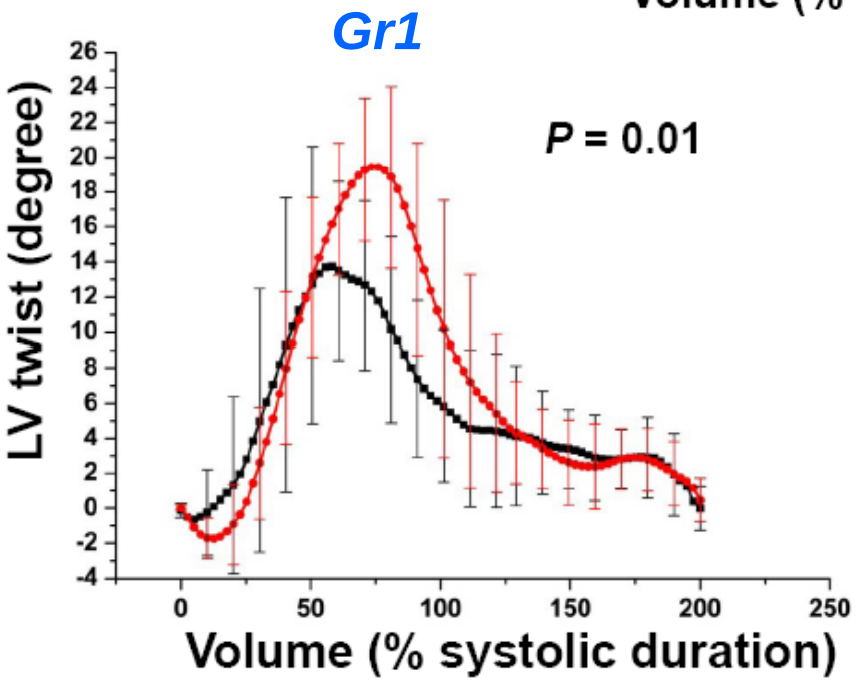


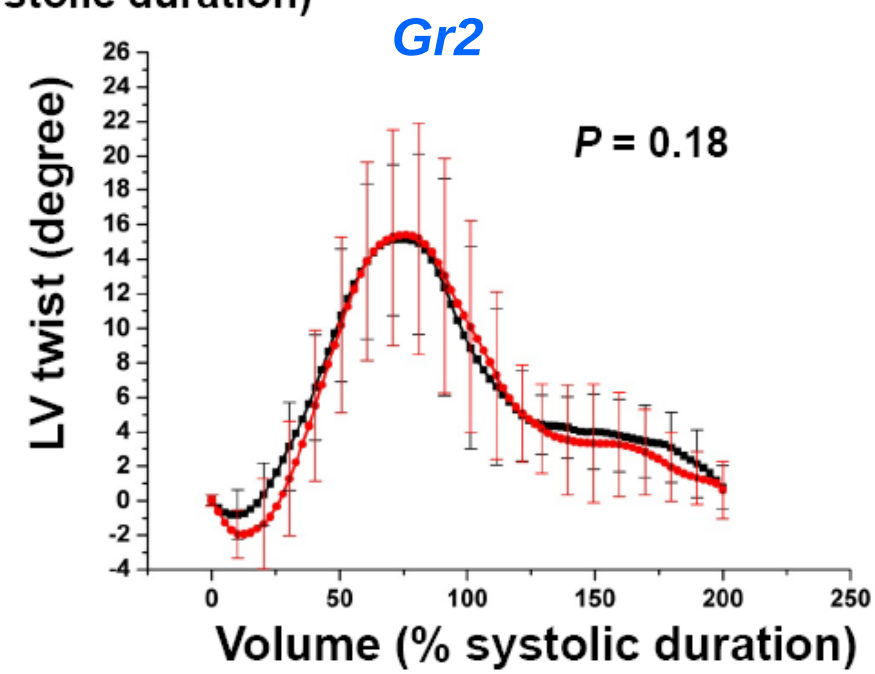
LV twist before vs. after RFCA



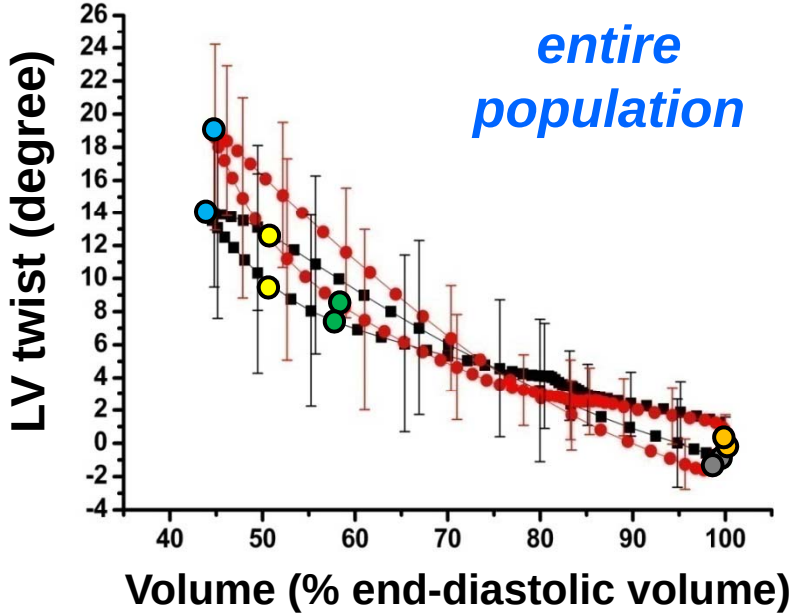
Black : pre-RFCA

Red : post-RFCA





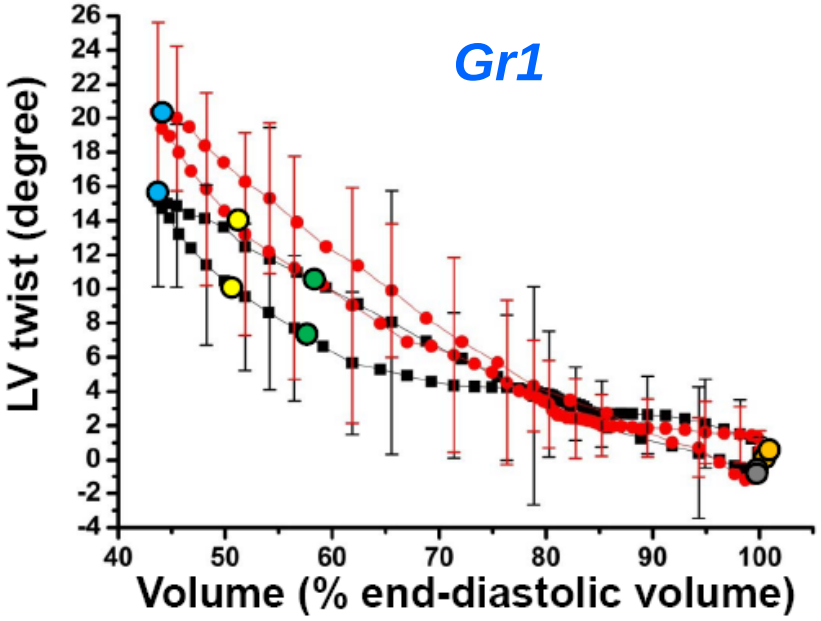
LV twist-volume loops before vs. after RFCA

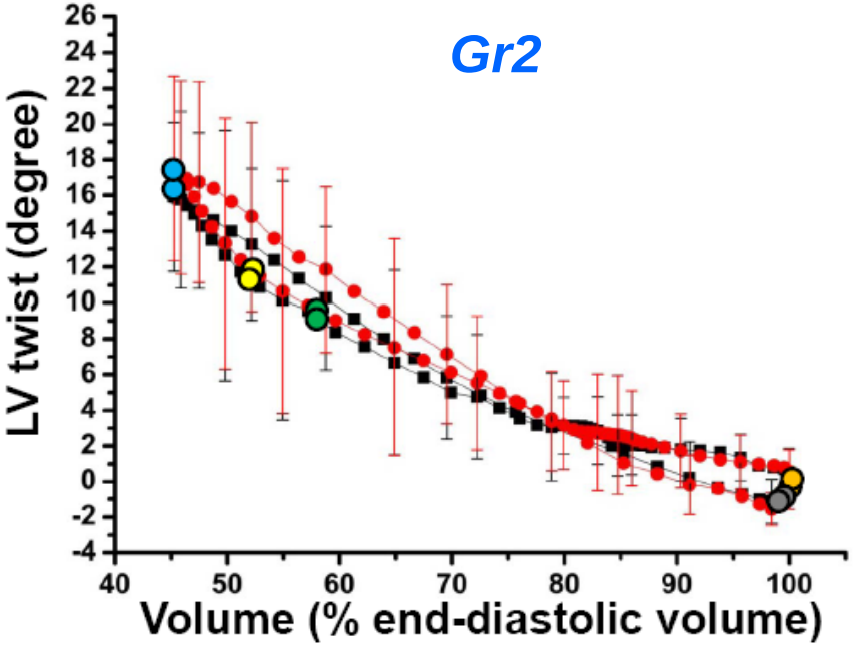


- Aortic valve closure
- Mitral valve opening
- Peak filling velocity
- Aortic valve opening
- Mitral valve closure

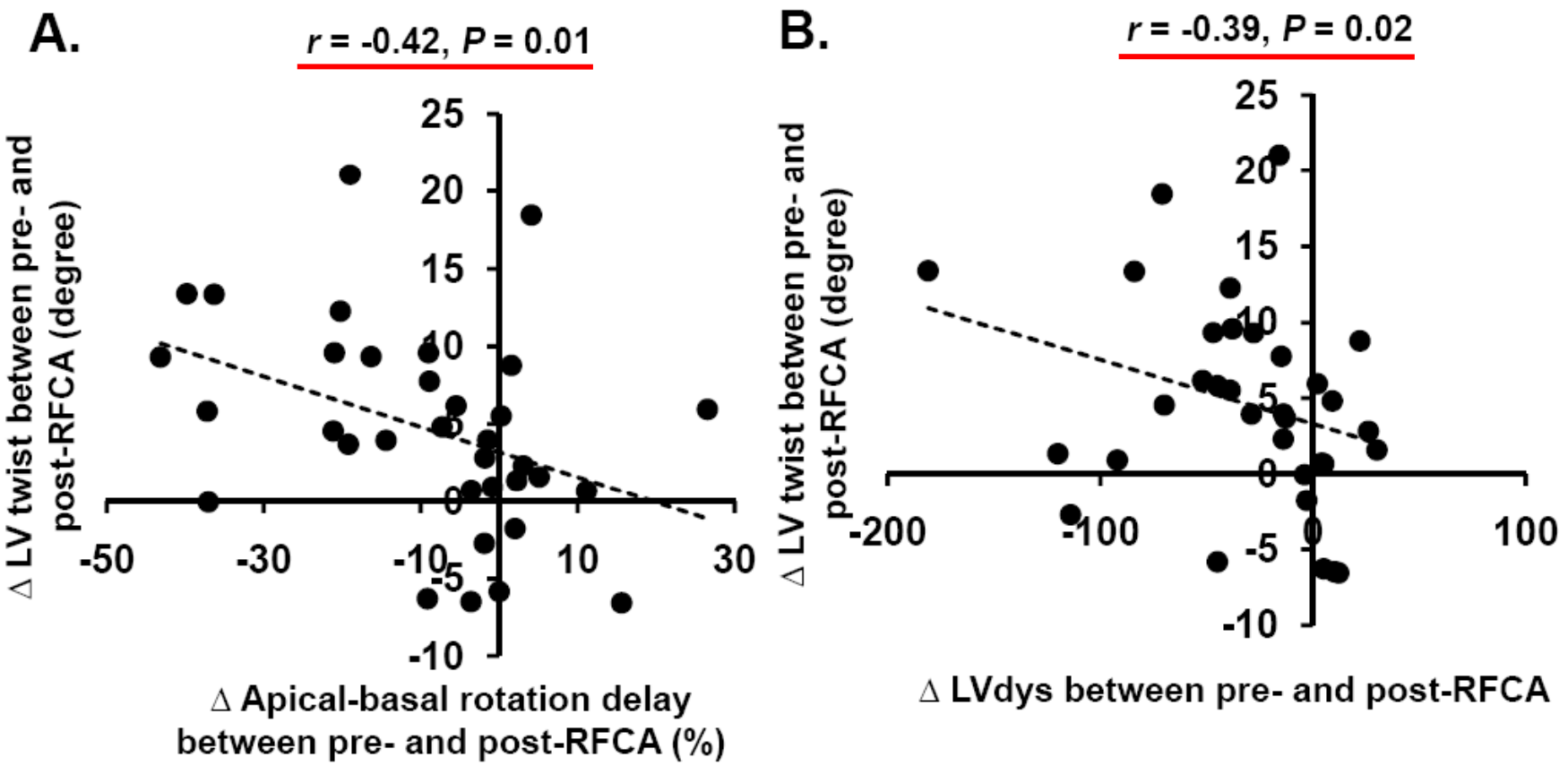
Black : pre-RFCA

Red : post-RFCA

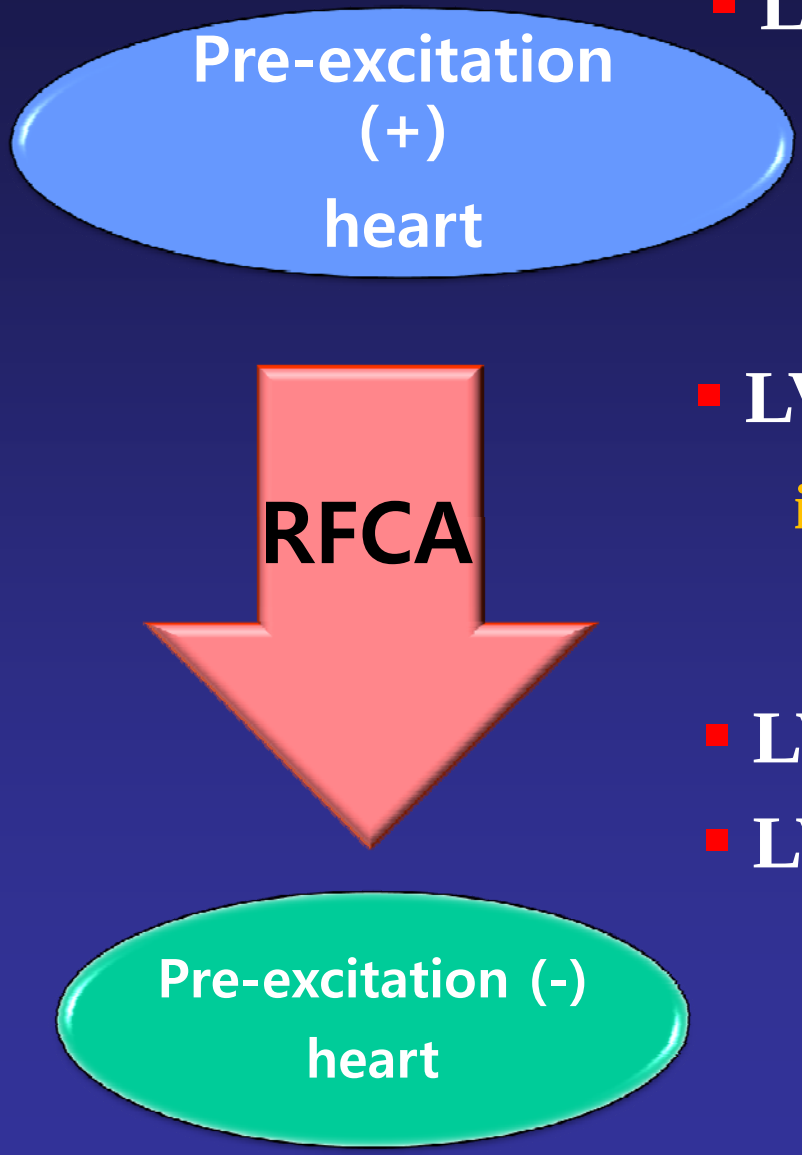




Δ LV twist in relation to LV synchronicity after RFCA



In Summary



- **LV contraction pattern**
  - **improved in some patients**
- **LV twist**
  - **improved in patients showing improvement in LV synchronicity**
- **LV systolic function**
- **LV diastolic function**
  - **improved in patients showing improvement in LV synchronicity**

Conclusions

- ***LV synchronicity***

  is an independent determinant of ***LV twist extent***,

  independently of preload or afterload

- ***RFCA*** leads to ***improvement in LV performance in a certain proportion of WPW patients***, another benefit of RFCA beyond elimination of sudden, fatal arrhythmic attack

*LV end-systolic wall stress (LV-ESWS)***

$$\text{LV end-systolic wall stress (gm/cm2)} = (\text{Pes}) \left[\frac{\text{Des}}{(\text{Hes})\,(1 + \text{Hes} / \text{Des})} \right] (0.34)$$

[Pes, LV end-systolic pressure ; Des, LV end-systolic dimension ; Hes, LV wall thickness]

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Lang RM et al. *Circulation.* 1986;74:1114-23.

Park HE and Kim HK et al. *Circ Cardiovasc Imaging*. 2010 *In press*.

Sohn DW and Kim HK et al. *Circ J.* 2008;72:251-5.