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Synchronicity of LV Contraction as a Determinant of LV Twist Mechanics

Serial Speckle-Tracking Analyses in WPW Syndrome Before and After Radiofrequency Catheter Ablation

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OBJECTIVES This study set out to investigate the isolated impact of synchronous patterns of left ventricular (LV) contraction (i.e., LV synchronicity) on LV twist behavior.

BACKGROUND Although the relationships between LV loading status/LV contractility and twist are well-established, no data are available regarding the relation between LV twist and LV synchronicity, without any interference by changes in LV pre-load, afterload, and contractility. Serial assessment of patients with Wolff-Parkinson-White syndrome before and after radiofrequency catheter ablation (RFCA) allows this to be explored.

METHOD Of the 40 Wolff-Parkinson-White patients initially screened, 34 were enrolled. Two-dimensional and Doppler echocardiography along with speckle tracking-derived LV twist mechanics, apical-basal rotation delay, and left ventricular dyssynchrony index (LVdys) were obtained before and after RFCA. The LVdys was defined as the maximal delay in time-to-peak radial strain of different LV segments at the papillary muscle level.

RESULTS Overall, no significant changes were demonstrated in LV volumes, systolic and diastolic function, and end-systolic wall stress before versus after RFCA. After RFCA, median value of LVdys was attenuated from 33.5 (interquartile range [IQR]: 14.0 to 84.3) to 14.0 (IQR: 11.5 to 21.8) ($p = 0.002$), which was accompanied by a reduction in apical-basal rotation delay from 9.7% (IQR: 3.5 to 23.7) to 3.3% (IQR: 1.3 to 8.0) ($p = 0.004$). In contrast, LV twist increased from 14.2° (IQR: 9.1° to 18.4°) before to 19.7° (IQR: 15.0° to 22.6°) after RFCA ($p = 0.002$). Delta LV twist pre- to post-RFCA displayed a significant inverse correlation with changes in apical-basal rotation delay ($r = -0.42$, $p = 0.01$) and Delta LVdys ($r = -0.39$, $p = 0.02$).

CONCLUSIONS The LV synchronous contraction is significantly related to LV twist. (J Am Coll Cardiol Img 2011;4:338–47) © 2011 by the American College of Cardiology Foundation

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Since the first description of left ventricular (LV) twist motion by William Harvey in 1628 (1), cardiac twist coupled with ensuing untwist motion is known to play a critical role in the maintenance of LV systolic and diastolic function (2,3). LV twist and ensuing untwist are important aspects of cardiac mechanics. Aside from a progressive increase in LV twist from infancy to adulthood (4), a small number of physiological factors—such as LV pre-load, afterload, and contractility—are known to alter LV twist extent (5,6). Recently, Matsuoka et al. (7) reported that acute right ventricular apical pacing significantly decreased LV twist extent, largely because of the induction of LV dyssynchrony, suggesting that LV synchronous contraction plays a pivotal role in maintaining LV twist. However, they failed to address whether the magnitude of LV twist can be modified solely by changes in LV synchronicity without alterations of left ventricular ejection fraction (LVEF) and LV size.

Wolff-Parkinson-White (WPW) syndrome is characterized by a short PR interval, a wide QRS duration (>120 ms), and a delta wave with or without secondary ST-T change on electrocardiogram. The presence of accessory pathways is electrocardiographically manifested by a delta wave just before QRS initiation, leading to a short atrioventricular delay due to its uninhibited electrical conduction, and eventually to asynchronous contraction (8,9). Radiofrequency catheter ablation (RFCA) treatment of accessory pathways in WPW patients is an established curative therapy restoring normal atrioventricular conduction and LV synchronous contraction (8,9). Provided that LV twist can be serially measured before and after RFCA in WPW syndrome without significant changes in LVEF and dimensions, the "isolated" effect of LV synchronicity on the LV twist mechanics could be effectively addressed without biases occurring due to changes in LV pre-load, afterload, or contractility. Recently, 2-dimensional speckle tracking echocardiography (STE) has been applied to the assessment of LV twist and has been extensively validated and proven to be accurate and consistent (10), allowing LV twist to be repetitively and noninvasively examined. Accordingly, we attempted to examine whether synchronous patterns of LV contraction can exert a significant influence on the LV twist behavior, without changes in LV loading status and contractility.

METHODS

Study population. We consecutively recruited 40 patients with WPW syndrome with a delta wave on resting electrocardiogram, who were scheduled for an elective electrophysiological study and RFCA for 1 or a combination of the following clinical indications: paroxysmal supraventricular tachycardia, atrial fibrillation, syncope, or aborted sudden cardiac death. Patients that satisfied the following criteria were excluded: congenital heart disease (n = 1), any valvular heart disease of more than mild degree (n = 1), episodes of arrhythmic attacks within a week of RFCA (n = 1), or not in sinus rhythm (n = 1). Patients with echocardiographic images inadequate for speckle tracking (n = 2) were also excluded. After exclusion of 6 patients, 34 patients formed the final study group. The study protocol was approved by the institutional review board of our hospital, and written informed consent was obtained from all participants before study enrollment.

Study protocol. To prove the critical role of LV synchronous contraction on the maintenance of LV twist, 2 serial echocardiographic examinations were performed. The first was conducted 18 to 24 h before RFCA, and the second was conducted 18 to 24 h after RFCA. Repetitive echocardiographic examinations with this short-time delay should help to isolate the impact of synchronous patterns of LV contraction on the magnitude of LV twist, because LV loading status (pre-load and afterload) and LV contractility do not seem to be altered over this short time delay. To minimize variability between echocardiographic examinations, all examinations were performed by 2 experienced cardiologists (S-A.C. and H-K.K.). Blood pressure and heart rate were measured after a 15-min resting period before echocardiographic examinations.

Electrophysiological study and RFCA procedures. Electrophysiological study and RFCA were simultaneously performed under fluoroscopic guidance. Antiarrhythmic agents were discontinued at least 48 h before electrophysiological study and RFCA in all patients. Six-F quadripolar electrode catheters were positioned at upper right atrium and right ventricular apex, whereas a 6-F decapolar electrode catheter was placed at coronary sinus or His bundle. The CardioLab electrophysiological system (GE

ABBREVIATIONS AND ACRONYMS

IQR = interquartile range
LV = left ventricle/ventricular
LVdys = left ventricular dyssynchrony index
LVEDV = left ventricular end-diastolic volume
LVEF = left ventricular ejection fraction
LVESV = left ventricular end-systolic volume
LV-ESWS = left ventricular end-systolic wall stress
RFCA = radiofrequency catheter ablation
STE = speckle tracking echocardiography
WPW = Wolff-Parkinson-White

Healthcare, Little Chalfont, United Kingdom) was used for detecting the location of accessory pathway by using programmed electrical stimulation. Bipolar and deflectable thermocatheters (bidirectional Livewire TC, St. Jude Medical, Minnetonka, Minnesota) with 4-mm tip electrode were used to ablate the accessory pathway under biplane fluoroscopic guidance.

Standard echocardiography. Transthoracic echocardiograms were obtained with commercially available equipment (Vivid 7, GE Medical Systems, Horten, Norway) with patients in a left lateral decubitus position. We initially determined the appropriateness of the patients for inclusion on the basis of 2-dimensional echocardiographic image quality. Patients with technically inappropriate image quality were excluded at this stage.

Standard echocardiographic examination involved measurements of LV wall thicknesses, left ventricular end-systolic (LVESV) and left ventricular end-diastolic volumes (LVEDV), LVEF, and a pulsed-wave Doppler examination of mitral inflow and mitral annular velocity. The modified biplane Simpson method was used to calculate LVESV, LVEDV, and LVEF.

Because systemic vascular resistance was reported to be an unreliable index of LV afterload (11), we calculated left ventricular end-systolic wall stress (LV-ESWS) as a reliable representative of LV afterload. LV-ESWS was calculated with the following formula (12,13):

$$LV-ESWS\,(\mathrm{g/cm^2}) = (Pes)\left[\frac{Des}{(Hes)(1 + Hes/Des)}\right](0.34)$$

LV-ESWS is in g/cm^2, Pes, which stands for LV end-systolic pressure, is in mmHg, Des and Hes are in LV end-systolicdimension and wall thickness in cm, and 0.34 is the factor for converting Pes from mmHg to g/cm^2.

Because there were no subjects with aortic stenosis, end-systolic pressure could be replaced with the noninvasively determined systolic blood pressure (13).

STE performance. After a standard echocardiographic examination, we scanned and recorded parasternal basal and apical short-axis planes to quantify basal and apical LV rotations without a dual-focusing tool. Sector width and image depth were optimized to maintain an adequate frame rate without losing 2-dimensional image quality. Frame rate (60 to 110 frames/s, mean 90 frames/s) and probe frequency (range 1.7 to 2.0 MHz) were adjusted. To standardize short-axis image planes among individuals, the basal level was defined as the point of the tips of mitral valve leaflets, and the apical level was defined just proximal to the level with LV luminal obliteration at the end-systolic period (2,3,13). To quantify radial left ventricular dyssynchrony index (LVdys), short-axis images from the mid-LV at the papillary muscle level were procured during end-expiratory breath hold. Care was taken not to get oblique LV short-axis images and to obtain short-axis images with the most circular geometry possible. To obtain longitudinal LVdys, apical 4-, 2-, and 3-chamber views were obtained. Three consecutive heart beats were digitally stored in cine-loop format and analyzed.

Image analysis. Image analysis was conducted by 1 independent cardiologist with a customized, dedicated software package (EchoPac 7.05 for PC, GE Medical Systems). Briefly, from an end-systolic single frame, a region of interest was manually defined on the endocardial cavity interface by a point-and-click approach. Then an automated tracking system followed the endocardium throughout the cardiac cycle. The validity of tracking was verified with a reliability parameter offered by the system and was again checked visually. Further adjustment of the region of interest was performed to ensure that all of the myocardial regions were included, as needed (2,3,13). The traced endocardium was automatically divided into 6 standard segments: inferoseptal, anteroseptal, anterior, anterolateral, posterior, and inferior.

Curves of averaged basal and apical rotations in 6 myocardial segments and their net difference at each corresponding time point (i.e., LV twist) can be directly generated from the current version of the EchoPac program and transported to an Excel worksheet (Microsoft Excel, 2007, Microsoft, Redmond, Washington) (Fig. 1). A similar pattern of analysis was applied to the LV twisting and untwisting rate calculations. To account for variable heart rates between patients, time points were normalized to the percentage of systolic duration (%).

To assess synchrony between the LV basal and apical rotation, the time interval from Q-wave onset on the electrocardiogram to peak rotation (Q-peak rotation interval) was obtained from basal and apical short-axis images (Fig. 1) (7). To avoid errors derived from variable heart rates between patients, time points were normalized to the duration of the whole RR interval, and as such, Q-peak rotation

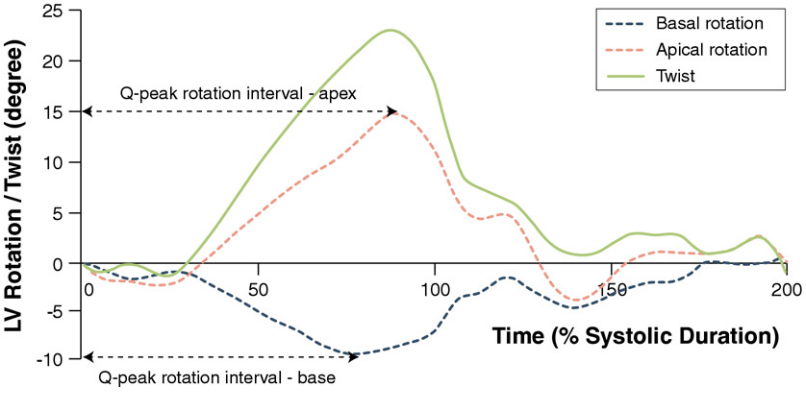

Figure 1. Curves for LV Basal and Apical Rotation and Twist

The time from the Q-wave on the electrocardiogram to peak rotation was defined as Q-peak rotation interval. Instead of being normalized to the percentage of systolic duration, Q-peak rotation interval was expressed in reference to the duration of the whole RR interval, with end-diastolic time point as 100%. LV = left ventricular.

interval at the LV base or apex was expressed as percentages. The difference between basal and apical Q-peak rotation intervals was employed as an indicator of apical-basal rotation delay (7). An example of the LV rotation and twist curve and radial strain analysis in a 21-year-old female patient is displayed in Figure 2.

To determine LVdys, time-radial strain curves for all the 6 myocardial segments were automatically constructed from LV short-axis images obtained at the papillary muscle level with the speckle tracking algorithm, by which the time from QRS onset to peak radial strain was obtained. The heterogeneity in time-to-peak radial strain for the 6

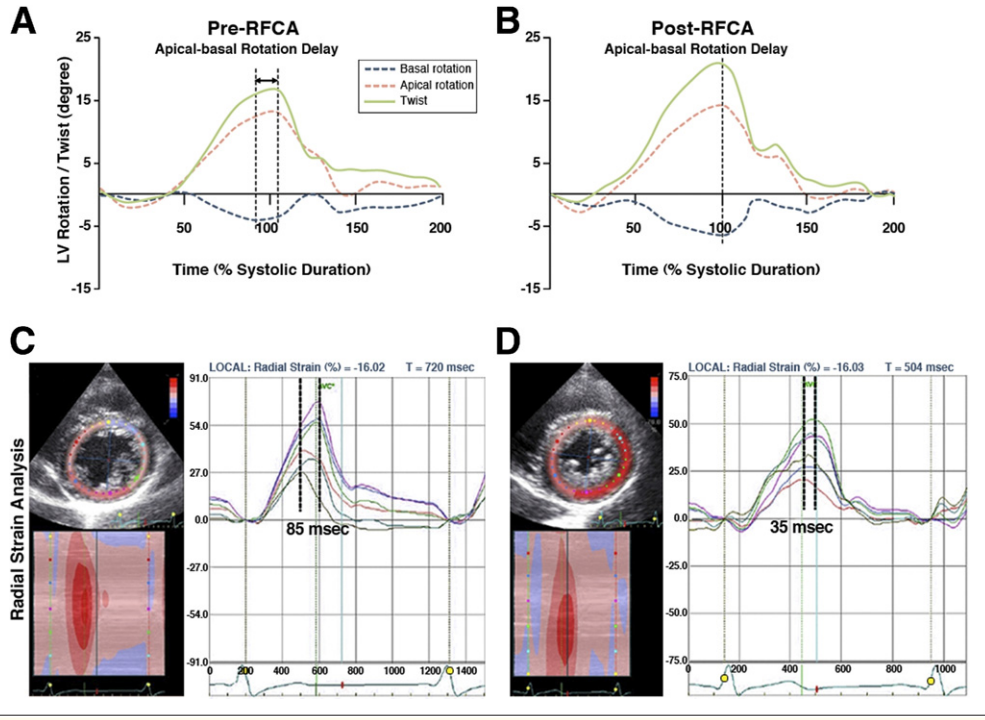

Figure 2. Changes in LV Rotation/Twist, Apical-Basal Rotation Delay, and Radial LVdys

LV rotation/twist curves before **(A)** and after **(B)** radiofrequency catheter ablation (RFCA) were shown in conjunction with corresponding radial strain curves before **(C)** and after RFCA **(D)**. Significant decreases in apical-basal rotation delay and radial left ventricular dyssynchrony index (LVdys) were noted with a rise in LV rotation/twist. Abbreviation as in Figure 1.

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myocardial segments was determined, whereby LVdys was calculated as the maximal time delay between the first and last segments to reach the peak radial strain (14). In addition, we also measured longitudinal LVdys with the apical 4-, 2-, and 3-chamber views. Standard deviation of the time to peak longitudinal systolic strain for 6 basal myocardial segments from 3 apical views was employed to obtain longitudinal LVdys.

Sample size calculation. We estimated study sample size, assuming an alpha-error of 0.05 and a beta-error of 0.20 at a statistical power of 80%. We presumed that an approximately 4° change in the LV twist could be found for an SD of 6°, on the basis of our earlier experiences. As a result, a total of 30 patients should be enrolled. When we supposed that approximately 10% of patients would be excluded during analysis, the number of patients required was approximately 35.

Statistical analysis. Data are expressed as mean ± SD and/or median with interquartile ranges for continuous variables on the basis of normality testing results and as numbers (%) for categorical variable. After evaluating the normality of continuous variables with Shapiro-Wilk test, the 2-sided Wilcoxon signed rank test or the paired *t* test was used to compare continuous variables before versus after RFCA. Differences between groups before or after RFCA were compared with the Student *t* test or the Mann-Whitney *U* test. For comparison of categorical variables, Fisher exact test was used. The relationships between continuous variables were analyzed with Spearman rho test. To identify independent determinants of a change in LV twist before versus after RFCA, we performed multivariate linear regression analysis with changes in LVEDV, radial LVdys, and apical-basal rotation delay as independent variables. All statistical analyses were performed with SPSS (version 17.0, SPSS, Inc., Chicago, Illinois), and a p value of <0.05 was considered statistically significant.

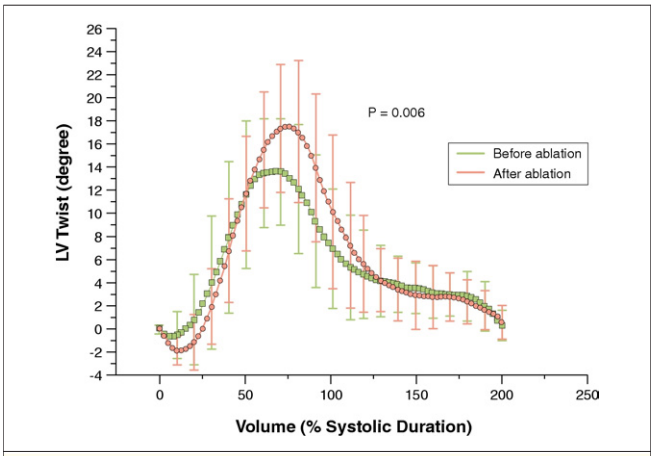

Figure 3. LV Twist Curves Before and After RFCA

Error bars of each curve drawn at 10% volume interval represent 1 SD. Abbreviations as in Figure 1 and 2.

RESULTS

Effects of RFCA on LV twist mechanics indexes and synchronous patterns of LV contraction. The RFCA was successfully performed in all 34 patients (median age 32 years; range 17 to 62 years; 19 men) without any complication. The accessory pathway was located on the right side of the heart in 6 patients (17.6%), on the septal side in 6 (17.6%), and on the LV lateral side in the remaining 22 patients (64.7%). Diabetes mellitus was present in only 1 patient, and only 2 patients had a diagnosis of hypertension. In all, there were no significant changes in terms of LV dimensions, LVESV, LVEDV, LVEF, systolic mitral annular velocity, global longitudinal strain, or LV-ESWS before versus after RFCA. Similarly, variables representing LV diastolic function (i.e., E, A, E/A ratio, E', and E/E' ratio) showed no changes after RFCA. By contrast, mean QRS duration on electrocardiogram before RFCA was 134.4 ± 18.8 ms, which was significantly decreased to 93.7 ± 12.2 ms after RFCA ($p < 0.001$). Of note, the median value of LVdys reduced from 33.5 (14.0 to 84.3) before to 14.0 (11.5 to 21.8) after RFCA ($p = 0.002$), whereas LV twist increased from 14.2° (9.1° to 18.4°) to 19.7° (15.0° to 22.6°) ($p = 0.003$) (Fig. 3). The RFCA induced no changes in basal or apical Q-peak rotation intervals, although apical Q-peak rotation interval tended to be shorter; however, apical-basal rotation delay became more simultaneous after RFCA (9.7% [3.5% to 23.7%] before vs. 3.3% [1.3% vs. 8.0%] after RFCA, $p = 0.004$) (Table 1).

Differences in LVdys (radial and longitudinal) were shown according to the location of accessory pathway in Table 2.

Relationship between LV twist mechanics indexes and LV synchronicity indexes. Because longitudinal LVdys was not affected by RFCA, as shown in Table 1, most results described hereafter will be limited to the radial LVdys.

When values obtained before and after RFCA were pooled, QRS duration on 12-lead surface

Table 1. Clinical and Echocardiographic Characteristics of the Entire Population (n = 34)

	Pre-RFCA	Post-RFCA	p Value
Clinical variables			
Age (yrs)	32 ± 14		—
Median (IQR)	27 (20–43)		
Male (%)	19 (55.9%)		—
BMI (kg/m^2)	23.0 ± 3.6		—
SBP (mm Hg)	123.0 ± 16.0	122.0 ± 11.0	0.62
DBP (mm Hg)	71.0 ± 12.0	71.0 ± 8.0	0.74
Heart rate (beats/min)	68.0 ± 10.0	68.0 ± 7.0	0.86
Median (IQR)	67.5 (62–70)	67 (63–71)	
Diabetes mellitus (%)	1 (2.9%)		—
Hypertension (%)	2 (5.9%)		—
QRS duration (ms)	134.4 ± 18.8	93.7 ± 12.2	<0.001
PR interval (ms)	116.2 ± 23.7	152.3 ± 21.0	<0.001
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
LVEDV (ml)	115.6 ± 24.1	113.6 ± 20.3	0.34
LVESV (ml)	45.4 ± 13.6	43.7 ± 10.9	0.31
LVEF (%)	61.0 ± 6.2	61.9 ± 5.1	0.38
E (m/s)	0.71 ± 0.13	0.71 ± 0.14	0.65
A (m/s)	0.46 ± 0.12	0.46 ± 0.12	0.79
E/A ratio	1.65 ± 0.56	1.68 ± 0.66	0.55
DT (ms)	169.0 ± 28.4	169.5 ± 29.4	0.94
S′ (m/s)	0.07 ± 0.01	0.08 ± 0.01	0.51
E′ (m/s)	0.09 ± 0.02	0.10 ± 0.02	0.17
A′ (m/s)	0.07 ± 0.02	0.07 ± 0.02	0.42
E/E′ ratio	7.5 ± 1.3	7.4 ± 1.7	0.58
LVESWS (g/cm^2)	52.5 ± 14.9	50.6 ± 10.0	0.41
Speckle tracking echocardiographic variables			
Frame rate	93 ± 22	88 ± 19	0.21
Median (IQR)	86 (70 to 119)	86 (70 to 104)	
Radial LVdys (ms)	49.4 ± 42.9	20.4 ± 21.4	0.002
Median (IQR)	33.5 (14.0 to 84.3)	14.0 (11.5 to 21.8)	
Longitudinal LVdys (ms)	42.5 ± 19.2	34.6 ± 15.8	0.16
Median (IQR)	39.0 (25.9 to 55.1)	31.5 (24.5 to 44.0)	
Global longitudinal strain (%)	−18.6 ± 1.8	−19.0 ± 1.89	0.48
Basal rotation (°)	−4.3 ± 2.5	−5.8 ± 2.4	0.002
Median (IQR)	−3.5 (−6.8 to −2.4)	−6.3 (−7.5 to −4.1)	
Apical rotation (°)	11.3 ± 4.9	13.8 ± 5.2	0.013
Median (IQR)	10.4 (7.5 to 13.8)	12.9 (10.6 to 17.7)	
LV twist (°)	14.6 ± 6.1	18.9 ± 6.0	0.002
Median (IQR)	14.2 (9.1 to 18.4)	19.7 (15.0 to 22.6)	
LV twisting rate (°/s)	102.3 ± 36.0	125.3 ± 34.2	0.003
Median (IQR)	98.1 (76.8 to 126.9)	123.1 (104.4 to 147.1)	
LV untwisting rate (°/s)	−115.7 ± 42.7	−146.5 ± 57.1	0.004
Median (IQR)	−111.6 (−134.4 to −83.1)	−138.8 (−194.0 to −101.3)	
Q-peak rotation interval–base (%)	41.6 ± 18.4	43.7 ± 11.3	0.28
Median (IQR)	37.7 (31.9 to 47.7)	42.1 (35.0 to 48.2)	
Q-peak rotation interval–apex (%)	42.9 ± 15.1	38.2 ± 5.7	0.34
Median (IQR)	38.9 (33.6 to 44.0)	37.4 (34.6 to 41.6)	
Apical–basal rotation delay (%)	15.8 ± 14.9	6.8 ± 9.7	0.004
Median (IQR)	9.7 (3.5 to 23.7)	3.3 (1.3 to 8.0)	

A = late diastolic mitral inflow velocity; A′ = late diastolic mitral annular velocity; BMI = body mass index; DBP = diastolic blood pressure; DT = deceleration time of E-wave; E = early mitral inflow velocity; E′ = early diastolic mitral annular velocity; ESWS = end-systolic wall stress; IQR = interquartile range; IVSd = end-diastolic interventricular septal thickness; LV = left ventricular; LVdys = left ventricular dyssynchrony index; LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; LVESV = left ventricular end-systolic volume; LVPWd = left ventricular end-diastolic posterior wall thickness; RFCA = radiofrequency catheter ablation; S′ = systolic mitral annular velocity; SBP = systolic blood pressure.

Table 2. Differences in LVdys According to the Location of Accessory Pathway

	RV Accessory Pathway (n = 6)	Septal Accessory Pathway (n = 6)	LV Free Wall Accessory Pathway (n = 22)	p Value by ANOVA
Pre-RFCA radial LVdys	44.3 ± 30.3	64.3 ± 34.3	46.8 ± 45.9	0.65
Post-RFCA radial LVdys	16.8 ± 14.3	23.0 ± 18.2	20.7 ± 24.2	0.89
Pre-RFCA longitudinal LVdys	37.0 ± 35.3	51.3 ± 7.9	42.2 ± 15.6	0.64
Post-RFCA longitudinal LVdys	37.6 ± 15.4	45.8 ± 26.2	31.4 ± 13.4	0.34
Changes in radial LVdys	−27.5 ± 40.9	−41.3 ± 53.6	−30.6 ± 47.6	0.86
Changes in longitudinal LVdys	0.6 ± 31.6	−5.5 ± 30.8	−10.8 ± 19.2	0.68

ANOVA = analysis of variance; RV = right ventricular; other abbreviations as in Table 1.

electrocardiograms displayed correlations with LVdys (rho = 0.32, p = 0.009) and apical-basal rotation delay (rho = 0.40, p = 0.001). Although PR interval showed a significant increase after RFCA from 116.2 ± 23.7 ms to 152.3 ± 21.0 ms (p < 0.001) (Table 1), we could not find any correlations between a change in PR interval and changes in radial LVdys (p = 0.11) or apical-basal rotation delay (p = 0.19). As was expected, LV twist showed a stronger correlation with LV apical rotation (rho = 0.86, p < 0.001) than with LV basal rotation (rho = −0.53, p < 0.001), suggesting that the extent of LV twist is more dependent on the degree of LV apical rotation, which is in keeping with previous investigations (8,27). The LV twist also showed a significant inverse correlation with LVdys (rho = −0.26, p = 0.04) (Fig. 4A), electrocardiogram-derived QRS duration (rho = −0.33, p = 0.006) (Fig. 4B), apical-basal rotation delay (rho = −0.35, p = 0.004) (Fig. 4C), and LV untwisting rate (rho = −0.62, p < 0.001). Of note, a difference in LV twist between before and after RFCA displayed a significant inverse correlation with a change in apical-basal rotation delay (rho = −0.42, p = 0.013) (Fig. 5A) and with a LVdys change (rho = −0.39, p = 0.023) (Fig. 5B) after RFCA (Table 3).

On multivariate linear regression analysis, we found that changes in radial LVdys (p = 0.046) and apical-basal rotation delay (p = 0.049) emerged as 2 variables showing independent associations with the change in LV twist after RFCA (r^2 for model = 0.20).

Measurement variability. The interobserver and intraobserver variabilities for measurements of LV twist in our echocardiographic laboratory were reported previously (2). Ten randomly selected clips were reviewed by a same observer more than 1 month apart after first measurement and independently by a second observer, with recorded images. The measurements made by the 2 independent observers correlated with the *r* value of 0.92 (standard error of the estimate = 1.6). In terms of intraobserver variability, the correlation was found to be *r* = 0.96 (standard error of the estimate = 1.2) without a trend for overestimation or underestimation.

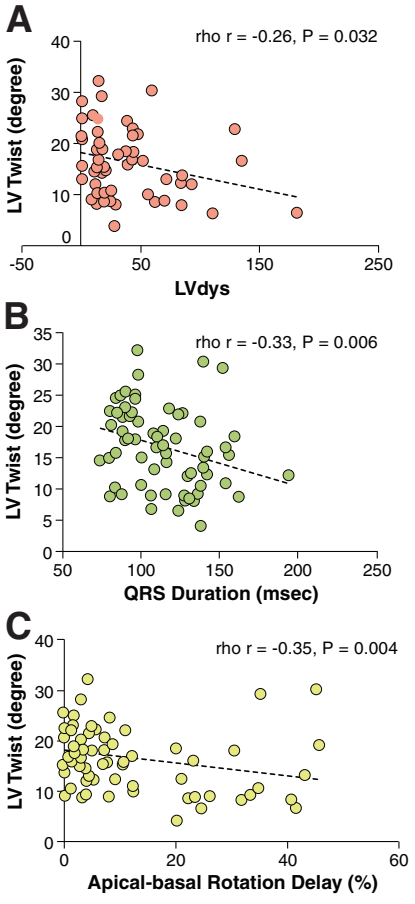

Figure 4. Scattergrams Showing the Relationships Between LV Twist and Other Variables

Relationships between LV twist and LVdys (**A**), QRS duration on surface electrocardiogram (**B**), and apical-basal rotation delay (**C**) were depicted. Abbreviations as in Figure 1 and 2.

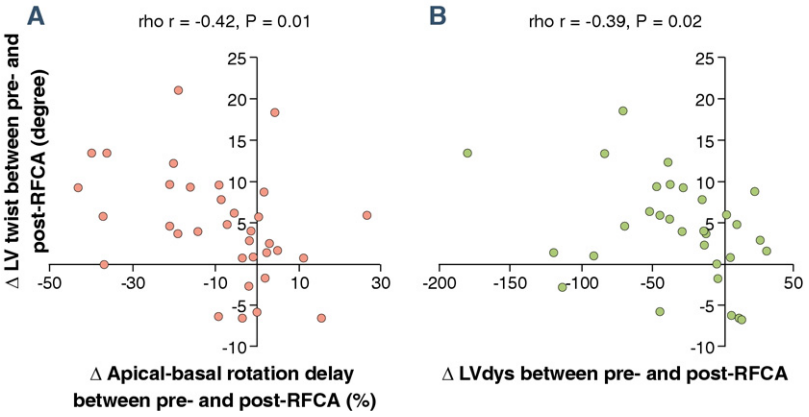

Figure 5. Relationship Between Delta LV Twist and LV Synchronicity

The difference in LV twist between pre- and post-RFCA was significantly correlated with Delta apical-basal rotation delay **(A)** or Delta LVdys **(B)** between pre- and post-RFCA. Abbreviations as in Figures 1 and 2.

DISCUSSION

LV twist mechanics. From a hemodynamic viewpoint, LV twist is dependent on LV loading conditions, such as LV pre-load and afterload (5). Similar to changes in LV loading status, LV twist progressively increases in parallel with an increment in LV contractility (5,6). The LV twist is well-known to be of utmost importance in maintaining LV systolic and diastolic function (2,3,15–17).

In this regard, knowledge concerning the hemodynamic and/or mechanical factors modulating LV twist extent is of clinical value, because recognition of those factors should extend our understanding of the LV twist mechanics and thus could lay a cornerstone for developing a novel therapy that helps the LV to work more effectively. On top of physiologic factors including LV pre-load, afterload, and contractility (5,6), it is conceivable that the heterogeneity in LV basal and apical rotation or LV synchronicity might also be a pivotal factor for determining LV twist magnitude, given that LV twist is determined by the degree of basal as well as the degree of apical rotation of the LV. As far as we are aware, however, no study has previously dealt with the impact of LV synchronicity per se on LV twist mechanics in the absence of a concomitant LVEF change—especially in normal, stable sinus rhythm rather than in pacing rhythm. Thus, the "isolated" effect of the heterogeneity in the systolic timings of myocardial segments (i.e., LV synchronicity) on LV twist mechanics remains unclear.

To decipher this enigma, WPW syndrome patients with a delta wave on resting electrocardiogram and sinus rhythm seemed to be an ideal model, because RFCA effectively and safely eliminates pre-excitation and, as such, restores LV synchronous contraction without directly altering LVEF or LV volumes (8,9). Therefore, patients with WPW syndrome offer a unique opportunity to assess the "isolated" impact of LV contraction pattern on LV twist mechanics. For this purpose, we adopted 2 independent indexes in the current study: 1) LVdys from radial strain analysis; and 2) apical-basal rotation delay. The comprehensive assessment of the extent of LV dyssynchrony with 2 independent variables might help to consolidate the potential association between LV twist mechanics and LV synchrony. It is also conceivable that apical-basal rotation delay is more representative of global LV synchronicity in relation to LV twist than LVdys, because LV twist is by definition an index incorporating information on both LV basal

Table 3. Correlations Between Indexes Representative of LV Twist Mechanics and Indexes Representative of the Extent of LV Dyssynchrony

Variables		LV Twist	LV Twisting Rate
QRS duration	r*	−0.33	−0.27
	p	0.006	0.03
Radial LVdys	r*	−0.26	−0.35
	p	0.03	0.003
Longitudinal LVdys	r*	0.02	0.07
	p	0.90	0.64
Apical-basal rotation delay	r*	−0.35	−0.25
	p	0.004	0.04

*The r value was derived with Spearman's rho test.
Abbreviations as in Table 1.

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and apical rotations simultaneously, whereas STE-derived LVdys employed is simply an indicator calculated from the 6 myocardial segments at the papillary muscle level. However, unexpectedly, the association of LV twist differences pre- to post-RFCA with changes in apical-basal rotation delay ($r = -0.42$, $p = 0.01$) was very similar to that of radial LVdys ($r = -0.39$, $p = 0.02$), underscoring the notion that LVdys obtained from only 6 mid-myocardial segments can be regarded as a proxy for global LV synchronicity in relation to LV twist, with an accuracy comparable to that of apical-basal rotation delay.

Changes in LV twist mechanics after RFCA. In the present study, LVEDV (an index of LV pre-load), LV-ESWS (an index of LV afterload), and systolic mitral annular velocity, global longitudinal strain, or LVEF (indirect indexes of LV contractility) all remained unaltered by RFCA, implying that LV loading status and LV contractility were stable. Nevertheless, RFCA led to a significant change in variables representing LV twist mechanics (namely, LV basal and apical rotation, LV twist, and LV twisting/untwisting rate), and this was accompanied by significant changes in the heterogeneity in time-to-peak radial strain of the 6 LV segments at the papillary muscle level (or radial LVdys) as well as in the heterogeneity in time-to-peak rotation of the LV base and apex (or apical-basal rotation delay). Given that there were significant correlations, albeit moderate in degree, between the RFCA-induced change in LV twist and changes in radial LVdys (Fig. 5A) or apical-basal rotation delay (Fig. 5B), LV synchronous contraction can be considered to be tightly coupled with LV twist mechanics.

There is a continuing controversy with regard to the relationship between LV twist mechanics and dyssynchrony. Ashikaga et al. (18) reported no improvement in LV rotational mechanics with cardiac resynchronization therapy in an animal model, whereas Sade et al. (19) and Bertini et al. (20) showed a significant association between LV twist and LV mechanical dyssynchrony in patients with heart failure. Because the current study enrolled WPW patients with a normal LVEF and without any evidence of heart failure, the direct comparison of the results with other aforementioned studies is impossible.

Given that the dependency of LV twist on LV synchrony might be augmented in patients with ischemic or nonischemic heart failure, in whom the typical counterclockwise apical rotation might be absent or even be reversed (21), it would seem that the correction of LV dyssynchrony by cardiac resynchronization therapy emerges as a treatment option for heart failure patients, particularly patients with normal LVEF (i.e., heart failure with preserved LVEF), who do not meet the current indications for cardiac resynchronization therapy.

Study limitations. Several limitations of the present study require consideration. First, the number of subjects enrolled was relatively small. Nevertheless, the study was sufficiently powered to reveal LV twist differences between pre- and post-RFCA. Second, we assumed that the only change in the study population from pre- to post-RFCA was the loss of pre-excitation. However, it is possible that RFCA per se might have induced edematous changes in myocardium or hyperthermal damage to myocardium, which could have caused deterioration in LV systolic and diastolic properties (22). However, we could not find any change in LV systolic and diastolic parameters. Moreover, if RFCA-induced myocardial injury had been present, LV twist and/or LV twisting/untwisting rate might have been reduced after RFCA, which is in contrast to our results. Thus, we do not believe that RFCA-induced myocardial damage, even if present, altered the results of our study. Finally, although a change in LV volumes after RFCA did not reach a statistical significance, LV volumes tended to slightly decrease after RFCA, possibly due to a 12- to 18-h fast before and during RFCA, which might exert a significant influence on the extent of LV twist.

CONCLUSIONS

LV synchronous contraction is significantly related to LV twist.

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REFERENCES

1. Harvey W. An anatomical disposition on the motion of the heart and blood in animals, 1628. Cardiac Classics. London: Henry Kimpton 1941;19–79.
2. Chang SA, Kim HK, Kim DH, et al. Left ventricular twist mechanics in patients with apical hypertrophic cardiomyopathy: assessment with 2D speckle tracking echocardiography. Heart 2010;96:49–55.
3. Kim HK, Sohn DW, Lee SE, et al. Assessment of left ventricular rotation and torsion with two-dimensional speckle tracking echocardiography. J Am Soc Echocardiogr 2007;20:45–53.
4. Notomi Y, Srinath G, Shiota T, et al. Maturational and adaptive modulation of left ventricular torsional biomechanics: Doppler tissue imaging observation from infancy to adulthood. Circulation 2006;113:2534–41.
5. Dong SJ, Hees PS, Huang WM, et al. Independent effects of preload, afterload, and contractility on left ventricular torsion. Am J Physiol 1999;277:H1053–60.
6. Gibbons Kroeker CA, Tyberg JV, Beyar R. Effects of load manipulations, heart rate, and contractility on left ventricular apical rotation. An experimental study in anesthetized dogs. Circulation 1995;92:130–41.
7. Matsuoka K, Nishino M, Kato H, et al. Right ventricular apical pacing impairs left ventricular twist as well as synchrony: acute effects of right ventricular apical pacing. J Am Soc Echocardiogr 2009;22:914–9.
8. Jue J, Winslow T, Ossipov M, Lesh MD, Schiller NB. Effect of preexcitation on Doppler indexes of left ventricular filling. Am J Cardiol 1993;71:1462–4.
9. Hina K, Murakami T, Kusachi S, et al. Decreased amplitude of left ventricular posterior wall motion with notch movement to determine the left posterior septal accessory pathway in Wolff-Parkinson-White syndrome. Heart 1999;82:731–9.
10. Notomi Y, Lysyansky P, Setser RM, et al. Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. J Am Coll Cardiol 2005;45:2034–41.
11. Lang RM, Borow KM, Neumann A, Jazen D. Systemic vascular resistance: An unreliable index of left ventricular afterload. Circulation 1986;74:1114–23.
12. Grossman W, Jones D, McLaurin LP. Wall stress and patterns of hypertrophy in the human left ventricle. J Clin Invest 1975;56:56–64.
13. Chang SA, Kim HK, Kim YJ, Oh S, Cho GY, Sohn DW. Role of pericardium in the maintenance of left ventricular twist. Heart 2010;96:785–90.
14. Park HE, Kim HK, Chang SA, et al. Impact of loading condition on the two-dimensional speckle tracking-derived left ventricular dyssynchrony index in non-ischemic dilated cardiomyopathy. Circ Cardiovasc Imaging 2010;3:272–81.
15. Kim WJ, Lee BH, Kim YJ, et al. Apical rotation assessed by speckle-tracking echocardiography as an index of global left ventricular contractility. Circ Cardiovasc Imaging 2009;2:123–31.
16. Sengupta PP, Tajik AJ, Chandrasekaran K, Khandheria BK. Twist mechanics of the left ventricle: principles and application. J Am Coll Cardiol Img 2008;1:366–76.
17. Park SJ, Miyazaki C, Bruce CJ, Ommen S, Miller FA, Oh JK. Left ventricular torsion by two-dimensional speckle tracking echocardiography in patients with diastolic dysfunction and normal ejection fraction. J Am Soc Echocardiogr 2008;21:1129–37.
18. Ashikaga H, Leclercq C, Wang J, Kass DA, McVeigh ER. Hemodynamic improvement in cardiac resynchronization does not require improvement in left ventricular rotation mechanics: three-dimensional tagged MRI analysis. Circ Cardiovasc Imaging 2010;3:456–63.
19. Sade LE, Demir O, Atar I, Müderrisoglu H, Ozin B. Effect of mechanical dyssynchrony and cardiac resynchronization therapy on left ventricular rotational mechanics. Am J Cardiol 2008;101:1163–9.
20. Bertini M, Marsan NA, Delgado V, et al. Effects of cardiac resynchronization therapy on left ventricular twist. J Am Coll Cardiol 2009;54:1317–25.
21. Bertini M, Nucifora G, Marsan NA, et al. Left ventricular rotational mechanics in acute myocardial infarction and in chronic (ischemic and nonischemic) heart failure patients. Am J Cardiol 2009;103:1506–12.
22. Eksik A, Gorgulu S, Eren M, et al. Sequential evaluation of left ventricular systolic and diastolic function after radiofrequency catheter ablation. Jpn Heart J 2004;45:429–40.

Key Words: echocardiography ■ speckle-tracking ■ synchronicity ■ twist ■ Wolff-Parkinson-White syndrome.