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Left ventricular twist in left ventricular noncompaction

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Aims Left ventricular (LV) twist is an important component of systolic function. The effect of abnormal LV twist on adverse remodelling of the heart in left ventricular noncompaction (LVNC) is unknown. This study used speckle-tracking echocardiography to evaluate LV twist in patients with LVNC and determine whether abnormal LV twist is associated with more adverse LV remodelling.

Methods and results Clinical, echocardiographic, and myocardial deformation characteristics were prospectively compared between 60 subjects diagnosed with LVNC and 59 age-matched healthy controls. Net instantaneous twist was defined as: peak apical rotation minus isochronous basal rotation. Normal rotation during systole was defined based on the 2010 ASE/EAE consensus document. Rigid body rotation (RBR) was determined present if the apex and base moved in the same direction during ejection. Rigid body rotation was found in 32 (53.3%) subjects with LVNC. The 28 subjects with LVNC and normal LV rotation had diminished apical rotation, basal rotation, and net twist compared with normal controls ($P < 0.0001$). Patients with LVNC and RBR had worse NYHA functional status ($P < 0.0001$), but similar echocardiographic indices of remodelling, ejection fraction, and strain parameters as those with LVNC and normal LV rotation.

Conclusion Left ventricular twist is diminished in subjects with LVNC and normal LV rotation. Rigid body rotation occurs in 53.3% of subjects with LVNC and is not associated with more adverse remodelling than subjects with LVNC and normal LV rotation.

Keywords Left ventricular noncompaction • Twist • Rigid body rotation

Introduction

Left ventricular (LV) twist is defined as the wringing motion of the heart during systole caused by rotation of the apex in a counterclockwise direction, as viewed from the apex, while the base moves in a clockwise direction. This wringing motion is an important contributor to the efficiency of blood ejection during systole.[1,2] Speckle-tracking echocardiography can be used to accurately assess LV twist and has been validated against tagged magnetic resonance imaging (MRI).[3]

Left ventricular noncompaction (LVNC) is considered a genetic cardiomyopathy that may be caused by intrauterine arrest of myocardial compaction.[4] Heart failure is the predominant clinical manifestation of this condition, which is often characterized by significant morbidity and mortality.[5] A recent report from Van Dalen *et al.*[6] documented that the apex and base of the left ventricle rotate in the same direction during systole in subjects with LVNC. This implies there is a loss of the wringing motion of the left ventricle during systole, which the authors termed rigid body rotation (RBR). They proposed this could be used as a functional criterion for the diagnosis of LVNC. In addition, RBR could be an important contributing factor of LV dysfunction and the burden of heart failure that characterizes the clinical presentation in this population.

Our group recently documented LVNC in subjects of African descent.[7] Heart failure due to systolic dysfunction was the most prevalent clinical manifestation of this cohort. Thus, we undertook this study to evaluate LV twist mechanics in our LVNC population using speckle-tracking echocardiography for the purpose of determining the prevalence of RBR and whether patients with RBR had more adverse LV remodelling.

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Methods

A total of 67 individuals diagnosed with LVNC were enrolled in Chris Hani Baragwanath Hospital's cardiomyopathy registry from July 2009 to September 2011. The Baragwanath cardiomyopathy registry was established in June 2009 after receiving approval from the University of the Witwatersrand Ethics Committee. From this cohort of 67 patients with LVNC, 60 patients fulfilled the study criteria and were included in this analysis. Inclusion criteria for this study were sinus rhythm, echocardiographic criteria that met a diagnosis of LVNC, and adequate echocardiographic image quality that allowed for complete assessment of LV myocardial mechanics. Exclusion criteria were documented hypertension (past or present), documented known coronary artery disease (past or on presentation), echocardiographic features suggestive of organic valvular disease, any systemic illness [e.g. human immunodeficiency virus (HIV)], thyroid disease, or any primary organ failure (e.g. chronic renal failure).

Control group

All 59 individuals that comprised the control group were of African descent. Patients with LVNC were matched to controls by age and gender. Individuals younger than 50 years were matched with a tolerance of 5 years in terms of age, whereas individuals older than 50 years were allowed a tolerance of up to 10 years. All controls were recruited from unrelated staff at Baragwanath Hospital as well as from local churches. The control group comprised individuals who were healthy (no known cardiac or systemic disease), normotensive, and not on any medication, and who had normal echocardiograms. These individuals are part of an ongoing prospective study to provide normal reference ranges for echocardiographic measurements in subjects of African ancestry, which has been approved by the University of the Witwatersrand Ethics Committee.

Echocardiography

Comprehensive transthoracic echocardiography was performed using a commercially available system (iE33 xMATRIX, Philips Healthcare, Best, The Netherlands) equipped with an S5-1 transducer (frequency transmitted 1.7 MHz, received 3.4 MHz), according to a standardized protocol. All data were transferred to an Xcelera workstation (Philips Healthcare) and analysed offline.

Measurements relating to chamber size and function were performed in accordance with the American Society of Echocardiography (ASE) chamber quantification guidelines of 2006 and the 2010 ASE guidelines on right heart assessment.[8,9] The severity of mitral and tricuspid regurgitation was analysed in accordance with ASE guidelines on native valvular regurgitation.[10] The left ventricular sphericity index (LVDSI) was calculated by dividing the left ventricle's maximal long-axis internal dimension by its maximal short-axis dimension at end-diastole.[11]

Diagnosis of LVNC was based on a combination of the Jenni[12] and Stöllberger[13] criteria:

(i) The ratio of the noncompacted to compacted layer width had to be >2 when measured at end-systole.
(ii) Presence of more than three prominent trabeculations in the LV apex that did not originate from the septum.
(iii) Deep intertrabecular recesses that filled with blood from the ventricular cavity as visualized on colour Doppler ultrasound.
(iv) No evidence of congenital or acquired heart disease.

Only subjects definitively satisfying all these criteria were diagnosed with LVNC. Requiring all these criteria be met for diagnosis is a documented method for accurately differentiating between normal subjects of African descent who could have variants of hypertrabeculation and those with true LVNC.[7] No individuals in the control group fulfilled any of these criteria for diagnosis of LVNC.

The location of noncompaction was described using a nine-segment model proposed by Jenni and co-authors.[12] The apex was defined as caudal to the papillary muscles, the base defined as the area of the left ventricle cranial to the closed tips of the mitral valve, and the midventricle defined as the area between the apex and base. The entire apex was regarded as one segment, whereas the base and midventricle were divided into four segments each (inferior, lateral, anterior, and septal).

Speckle-tracking analysis

Analysis of all cases was performed by a cardiologist experienced in speckle-tracking echocardiography (F.P.) using QLAB Advanced Quantification software (Version 8.0, Philips Healthcare). The assessment of LV twist using speckle tracking was previously validated against tagged MRI.[14] To optimize speckle tracking, images were obtained at a frame rate of 50–80 frames/s. All images were obtained during sinus rhythm with $<10\%$ variability in heart rate. Parasternal short-axis images at the basal LV level showing the tips of the mitral valve leaflets were obtained with as circular of a cross-section as possible. To obtain a short-axis image at the apical LV level, the transducer was positioned one or two intercostal spaces more caudally and acquired in a manner described by Van Dalen *et al.*[15]

To assess LV rotation, tracking points were placed on an end-diastolic frame in each parasternal short-axis image. In areas of hypertrabeculation, the tracking points were placed in the compacted part of the muscle. Tracking points were separated about 60 degrees from one another to fit the total LV circumference. After positioning these points, the program tracked them. In keeping with the standard ASE/EAE consensus document on myocardial deformation as viewed from the apex, counterclockwise rotation was assigned a positive value and clockwise rotation a negative value.[16] The peak systolic bulk rotation was measured at the apex. Thereafter, the basal bulk rotation at a time isochronous to that of the peak apical rotation was measured. Net instantaneous twist was calculated as peak apical rotation minus basal rotation at a time interval corresponding to the occurrence of peak apical rotation. In addition to quantifying apical and basal rotation, we analysed the direction of rotation in both isovolumic contraction and the rest of systole. We focused on the pattern of rotation during the ejection phase of systole and identified rotation as either normal or having evidence of RBR, which was either entirely counterclockwise or clockwise at both the base and apex during the ejection phase of systole. Subepicardial and subendocardial rotation was measured at both the apex and the base. Diastolic rotation parameters were not examined in this study.

Longitudinal strain was measured using an apical four-chamber image; radial and circumferential strain were measured using a short-axis view of the midventricle (at the level of the papillary muscles). In all instances, the inability to track more than one segment resulted in the study being deemed suboptimal. The average longitudinal, radial, and circumferential strain values for each patient were calculated by adding the respective segmental strain values in a view and dividing by the number of segments analysed.

Reproducibility

The pattern of rotation during the ejection phase was assessed in 30 subjects (15 controls and 15 subjects with LVNC) by a blinded individual experienced with speckle tracking (N.M.). There was 100% agreement in all cases with regard to patterns of rotation.

Statistics

Statistical analyses were performed using an SAS statistical program (Version 9.12, SAS Institute Inc., Cary, NC, USA). Descriptive statistics are presented as means ± standard deviation for continuous variables or frequencies and percentages for categorical variables. Comparisons between the LVNC group and controls were evaluated using analysis of covariance techniques with age and gender as covariates. Categorical variables were compared using the chi-square test. Differences between LVNC subjects with RBR or normal twist patterns were assessed using the Mann–Whitney–Wilcoxon test for non-normal distributions. Statistical significance was defined as two-tailed P-value < 0.05.

Results

Baseline clinical and echocardiographic characteristics of the 60 subjects with LVNC are compared with the cohort of 59 controls in *Tables 1* and *2*. No significant differences were noted with regard to age, sex, diastolic blood pressure, and body surface area between patients with LVNC and controls. Heart failure (based on Framingham criteria[17]) was the most prevalent clinical manifestation of patients with LVNC and occurred in 58 (96.7%) subjects. Standard medical therapy included beta blockers and angiotensin-converting enzyme inhibitors in all patients with LVNC. All subjects were on furosemide at the time of evaluation. Most subjects with LVNC (33/60; 55%) were New York Heart Association (NYHA) Class II.

As expected, patients with LVNC had significantly greater LV dilatation, LV dysfunction, E/A ratios, and left atrial volume indices compared with the control group (*Table 2*). The right ventricle was found to be dysfunctional in 44 (73.3%) patients with LVNC; mean right ventricular S' (9.4 ± 3.04 cm/s) was significantly lower than that of the control group.

Left ventricular rotation

A normal pattern of LV rotation was found in 28 (46.7%) patients with LVNC (*Figure 1*), whereas RBR was noted in the remaining 32 (53.3%) (*Table 3*). In the group with RBR, 23 subjects (71.9%) had clockwise rotation (*Figure 2*) and 9 (28.1%) had counterclockwise rotation at both the base and apex. All subjects in the control group had a normal pattern of rotation at the base and apex. In the 28 subjects with LVNC and a normal pattern of LV rotation, apical rotation, basal rotation, and net twist were all diminished compared with the control group ($P < 0.0001$). In both the control group and all patients with LVNC irrespective of their pattern of rotation, the subendocardial and subepicardial patterns of rotation were in the same direction as global apical and basal rotations, respectively.

Table 1 Clinical features of the study population

Characteristic	LVNC ($n = 60$)	Controls ($n = 59$)	P-value
Age, years[a]	47.01 ± 12.8	48.68 ± 10.7	0.28
Female sex, no. (%)	33 (55%)	32 (54.2%)	0.15
Body surface area, m^2[a]	1.75 ± 0.16	1.71 ± 0.18	0.34
Systolic blood pressure, mmHg[a]	111.3 ± 15.2	127.86 ± 6.7	<0.0001
Diastolic blood pressure, mmHg[a]	75.7 ± 11.2	77.98 ± 7.11	0.17

LVNC, left ventricular noncompaction; NYHA, New York Heart Association.
[a]Mean ± standard deviation.

Strain

Longitudinal, radial, and circumferential strains in patients with LVNC were all significantly diminished compared with controls (*Table 3*).

Clinical and echocardiographic characteristics based on rotation patterns

Subjects with LVNC who displayed RBR did not differ from LVNC subjects who had a normal pattern of twist with respect to age, indices of LV remodelling, or ejection fraction (*Table 4*). In addition, no difference was noted in right ventricular function ($P = 0.53$), degree of tricuspid regurgitation ($P = 0.16$), and degree of pulmonary hypertension ($P = 0.25$). The major difference between these two groups was that radial strain was significantly greater in those with RBR compared with those with normal twist ($P = 0.024$), and that NYHA functional class was worse in those with RBR ($P = 0.04$). No significant differences were noted for longitudinal and circumferential strain.

Discussion

One of the main findings of this study is that RBR was found in 53.3% of subjects diagnosed with LVNC based on strict echocardiographic criteria. Two patterns of RBR were found—clockwise rotation in 23

Table 2 Echocardiographic features

Characteristic	LVNC ($n = 60$)	Controls ($n = 59$)	P-value
LVEDD, mm	60.3 ± 8.3	43.1 ± 3.9	<0.0001
LVESD, mm	52.8 ± 9.4	29.2 ± 4.6	<0.0001
LVEF, %	26.7 ± 10.7	62.6 ± 6.5	<0.0001
IVSd, mm	8.9 ± 1.9	10.01 ± 1.3	<0.0001
LVPWd, mm	9.9 ± 2.3	8.6 ± 0.9	0.002
End-diastolic volume, mL	190.4 ± 69.8	74.3 ± 12.8	<0.0001
End-systolic volume, mL	139.6 ± 71.4	30.4 ± 7.6	<0.0001
LVDSI, ratio	1.4 ± 0.16	1.9 ± 0.2	<0.0001
E/A ratio	1.8 ± 0.9	1.17 ± 0.3	<0.0001
Left atrial volume index, mL/m^2	42.4 ± 18.6	20.6 ± 1.1	<0.0001
Pulmonary arterial pressure, mmHg	45.5 ± 17.7		
Right ventricular S' velocity, cm/s	9.4 ± 3.04	12.3 ± 2.3	<0.0001

Data reported as mean ± standard deviation.
IVSd, interventricular septal thickness at diastole; LVDSI, left ventricular sphericity index; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVNC, left ventricular noncompaction; LVPWd, left ventricular posterior wall thickness at diastole; S', systolic tricuspid annular motion detected using tissue Doppler imaging.

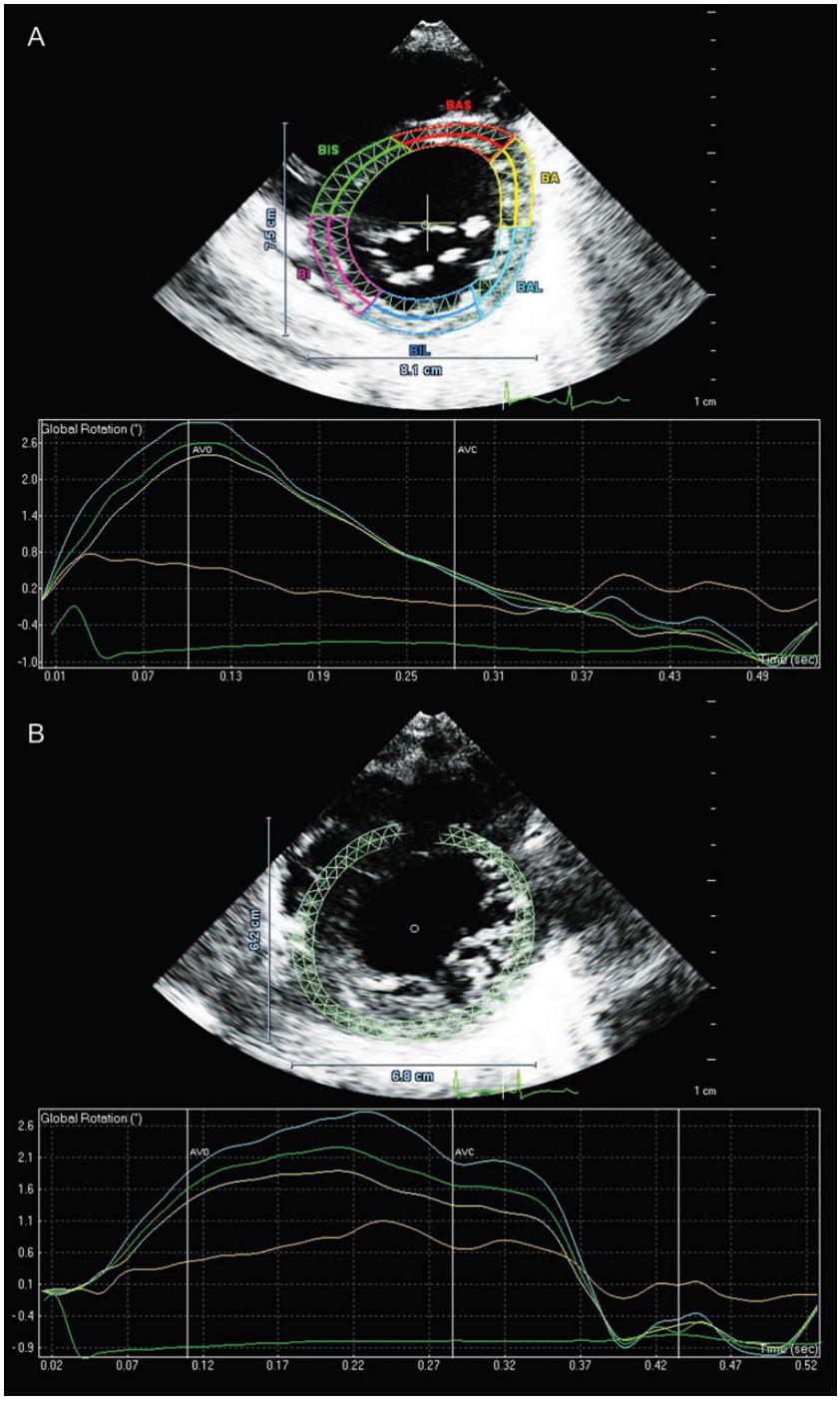

Figure 1 (*A*) Basal rotation of a patient with left ventricular noncompaction (LVNC) and a normal pattern of rotation. Bulk rotation is displayed in green. The subepicardial (gold) and subendocardial (blue) rotations move in the same direction as the bulk rotation, which is akin to global rotation at the base when using other vendors' software. (*B*) Apical rotation of a patient with LVNC and a normal pattern of rotation. Bulk rotation is displayed in green. The subepicardial (gold) and subendocardial (blue) rotations move in the same direction as the bulk rotation at the apex.

(71.9%) and counterclockwise RBR in 9 (28.9%). The major abnormality in twist mechanics in all subjects with LVNC and a normal pattern of rotation related to a reduction in both apical and basal rotation. There were no differences relating to clinical features, echocardiographic remodelling parameters, or ejection fraction between subjects with LVNC who had a normal pattern of twist or RBR except for two parameters—patients with RBR had worse NYHA class and statistically significant greater radial strain.

Left ventricular twist is thought to be an important component of LV systolic function. The normal LV myocardial wall is characterized

Table 3 Myocardial deformation parameters in LVNC and controls

Parameter	LVNC ($n = 60$)	Controls ($n = 59$)	*P*-value
Peak apical rotation, degrees[a]	1.74 ± 0.9	6.6 ± 1.9	<0.0001
Basal rotation, degrees[a]	−1.2 ± 0.9	−3.4 ± 0.6	<0.0001
Net twist, degrees[a]	2.9 ± 1.3	10.02 ± 2.1	<0.0001
Twist pattern			
Normal, no. (%)	28 (46.7%)	59 (100%)	<0.0001
Rigid body rotation, no. (%)	32 (53.3%)		
Longitudinal strain, %[a]	−5.2 ± 3.3	−14.83 ± 2.4	<0.0001
Circumferential strain, %[a]	−5.9 ± 4.4	−14.91 ± 2.6	<0.0001
Radial strain, %[a]	1.4 ± 5.2	14.9 ± 2.6	<0.0001

LVNC, left ventricular noncompaction.
[a]Mean ± standard deviation.

by a right-handed helix in the subendocardial region and a left-handed subepicardial helix. The interaction between these two helices is responsible for the net apical counterclockwise and basal clockwise rotations that occur in these regions during the ejection phase of systole. The net normal motion occurs because the wider radius of rotation of the subepicardial fibres dominates the subendocardial motion.[2] In this study, we have shown that LV twist in patients with LVNC and a normal pattern of rotation is diminished compared with controls ($P < 0.0001$). A decrement in both apical and basal rotation may be due to either a decrease in function of the subepicardial fibres or a combination of both helices having decreased function.

The process of myocardial compaction *in utero* proceeds from base to apex and from epicardium to endocardium. We can only postulate that differences relating to the timing of arrest in maturation could cause varying degrees of apical dysfunction as well as varying degrees of abnormality in the two helices. Abnormality of basal rotation also could be caused by varying degrees of maturation of the two helices at the base or by subendocardial ischaemia. The latter is a well-documented finding on thallium scintigraphy, positron emission tomography, and MRI, and occurs in the absence of any epicardial coronary abnormality.[18–21] Varying combinations of these abnormalities may account for either a normal pattern of LV twist or RBR.

In this study, RBR of two distinct patterns occurred in 53.3% of patients with LVNC (*Table 3*). The prevalence of RBR in this cohort of LVNC patients differs from two prior reports from Van Dalen *et al.*, who found that 83% of subjects with LVNC had evidence of RBR.[6] There are important similarities in the study design of Van Dalen *et al.*[6] and this study with regard to the use of similar vendor technology (QLAB analysis software), the placement of tracking points, and the same apex imaging technique utilized. However, there are important differences between these two studies, and we entertain three possible reasons for the lower prevalence of RBR in our cohort.

The possibility of overdiagnosis of LVNC in our cohort was taken into consideration, since a prior report suggested that normally healthy blacks could have more prominent trabeculation than the overall population, and could therefore satisfy general criteria for LVNC.[22] In this study, we only included individuals who satisfied all four inclusion criteria, unlike the less exclusive criteria applied by Kohli *et al.*[22] Our previous findings in a large cohort of normal controls from our study population showed that no normal individuals fulfill all four inclusion criteria. In addition, since 96.7% of our patients had LV dysfunction, we excluded individuals with other causes of LV dysfunction, e.g. HIV, hypertension, postpartum cardiomyopathy, etc.

A second possible explanation for our results is that RBR was underdetected. In this study, we diagnosed RBR only if either the base or apex was moving completely in the opposite expected direction during the entire ejection phase. Partial abnormal movement early in the ejection phase followed by the normal, expected direction of rotation was not diagnosed as RBR. This parameter was set based on our findings that this partial abnormal pattern can infrequently be observed in normal controls from our population.

A final important difference between our study and Van Dalen's relates to study population and methodology. Our study is a much larger cohort comprised of only individuals of African descent. Differences relating to race may have implication in terms of differing genetic profiles that can cause LVNC and in the manner of remodelling that might subsequently occur. Selection bias in both studies may contribute to differences observed. Van Dalen *et al.* included 17 cases of familial LVNC out of their total cohort of 36 individuals with LVNC. All of their familial LVNC patients showed RBR. Thus, in unrelated individuals with LVNC, RBR occurred in 13 of 19 cases (68.4%). This contrasts with the fact that, in our study, no familial LVNC patients were included. A second important consideration is the marked referral bias in our study population due to various socioeconomic resource limitations, which results in only the most symptomatic patients being evaluated. This may also account for the fact that our study subjects had a much lower ejection fraction and greater remodelling (larger LV volumes) than Van Dalen's cohort.

A major finding of this study is that there were no significant clinical and echocardiographic differences between patients with RBR and those with normal twist patterns. The only significant difference between these two groups in terms of their mechanics was that radial strain was higher in those with RBR. Despite the radial strain being statistically significantly higher, it is nevertheless considerably lower than expected and cannot be viewed as a partial compensatory mechanism for the absence of twist in subjects with RBR. This suggests that there are other factors that contribute to the adverse remodelling that characterizes LVNC when LV dysfunction occurs, such as wall stress, oxidative stress, neurohumoral factors, cytokines, and apoptosis. Differences relating to these factors could be important and result in the similar clinical and echocardiographic findings despite very different twist mechanics. Two important factors not evaluated in this study also may influence LV systolic function and possibly LV twist: (i) the influence of varying loading conditions on LV twist was not documented and may vary among individuals such that, despite similar EF, their clinical statuses and myocardial mechanics differ. The latter is particularly true regarding LV twist,

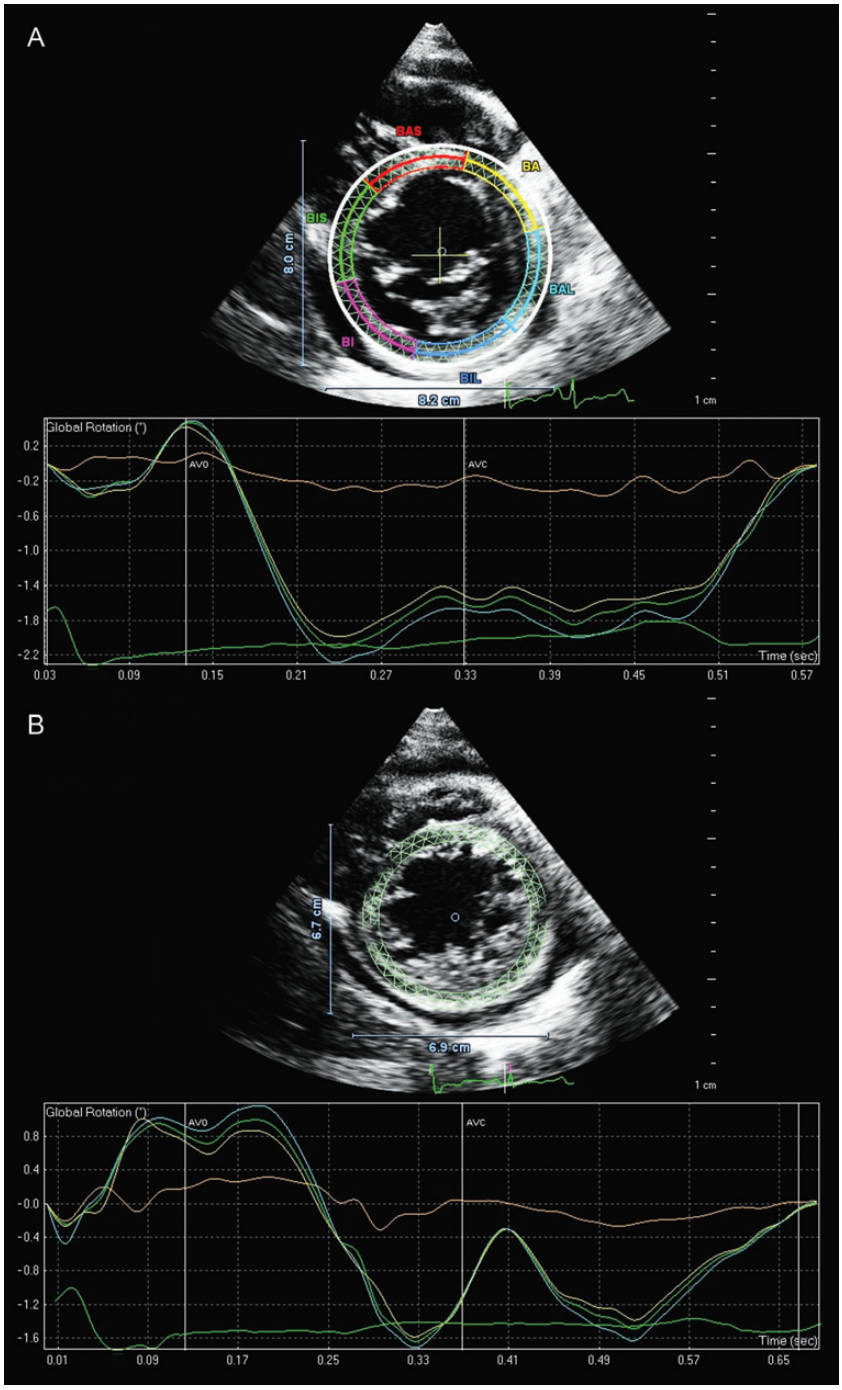

Figure 2 (*A*) Basal rotation in clockwise rigid body rotation (RBR). Bulk rotation at the base is depicted by the green curve, subepicardial rotation by the gold curve, and subendocardial rotation by the blue curve. During the ejection phase of systole, rotation at the base is in a clockwise direction. (*B*) Apical rotation in clockwise RBR. Bulk rotation at the apex is depicted by the green curve, subepicardial rotation by the gold curve, and subendocardial rotation by the blue curve. During the ejection phase of systole, rotation at the apex is in a clockwise direction.

which varies with changes in loading conditions. Physiological variables such as preload, afterload, and contractility alter the extent of LV twist.[23,24] Twist is greater with higher preload when end-systolic volume is held constant, whereas twist decreases if afterload increases when end-diastolic volume is constant.[2] (ii) The presence and severity of LV dyssynchrony, which is an additional important factor that could modulate LV remodelling and ejection fraction, was not documented.

One limitation of this study is that this work provides only a snapshot in time. A more meaningful conclusion can only be made after

Table 4 Clinical features, echocardiographic parameters, and strain in subjects with LVNC based on differences in twist patterns

Characteristic	RBR ($n = 32$)	Normal ($n = 28$)	*P*-value
Age, years[a]	47.3 ± 13.1	46.8 ± 12.7	0.80
Female sex, no. (%)	19 (59.4%)	14 (50%)	0.60
Body surface area, m^{2a}	1.72 ± 0.19	1.71 ± 0.16	0.62
Systolic blood pressure, mmHg[a]	113.1 ± 14.9	109.4 ± 15.6	0.60
Diastolic blood pressure, mmHg[a]	75.3 ± 10.2	76 ± 12.3	0.80
NYHA class, no. (%)			0.04
I	9 (28.1%)	8 (28.6%)	
II	14 (43.8%)	19 (67.8%)	
III	9 (28.1%)	1 (3.6%)	
LVEDD, mm[a]	59.3 ± 8.4	61.5 ± 8.1	0.13
LVESD, mm[a]	52.2 ± 10.3	53.5 ± 8.4	0.40
E′ M, cm/s[a]	5.5 ± 3.43	4.5 ± 1.8	0.26
E′ L, cm/s[a]	6.7 ± 3.0	7.28 ± 3.9	0.87
Deceleration time, ms[a]	100.0 ± 43.8	99.1 ± 41.9	0.76
E/A ratio[a]	1.7 ± 0.9	1.9 ± 0.8	0.30
End-diastolic volume, mL[a]	188.2 ± 71.6	192.9 ± 68.8	0.86
End-systolic volume, mL[a]	136.4 ± 82.4	143.2 ± 57.6	0.30
Left ventricular ejection fraction, %[a]	27.9 ± 9.7	24.9 ± 11.7	0.20
Left atrial volume index,mL/m^{2a}	43.1 ± 18.2	41.7 ± 19.4	0.70
LVDSI, ratio[a]	1.4 ± 0.2	1.4 ± 0.2	0.90
Right ventricular S' velocity, cm/s[a]	9.5 ± 2.8	9.3 ± 3.4	0.50
N/C myocardium ratio			
Apex[a]	3.27 ± 1.04	3.40 ± 1.19	0.70
Midlateral wall[a]	2.90 ± 1.04	2.43 ± 0.32	0.03
Midinferior wall[a]	2.70 ± 0.50	2.50 ± 0.40	0.05
Longitudinal strain, %[a]	−5.5 ± 6.6	−5.9 ± 4.3	0.80
Circumferential strain, %[a]	−5.2 ± 3.3	−5.2 ± 3.3	0.60
Radial strain, %[a]	4.2 ± 4.9	1.38 ± 5.2	0.024

[a]Mean ± standard deviation.
E′ L, peak E′ lateral wall velocity; E′ M, peak E′ medial wall velocity; LVDSI, left ventricular sphericity index; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVNC, left ventricular noncompaction; N/C, noncompacted/compacted; NYHA, New York Heart Association; RBR, rigid body rotation; S′, systolic tricuspid annular motion detected using tissue Doppler imaging.

long-term follow-up of this cohort determines the natural evolution of these abnormalities. Do subjects with diminished twist ultimately progress to RBR? Follow-up will provide insight into whether RBR has more deleterious clinical consequences compared with subjects who have only a decrement in LV twist.

However, the major area in need of further research is determining the extent of RBR in other populations with LVNC and doing so using another vendor's software. The findings relating to RBR in our environment imply that RBR cannot be implemented as an additional functional diagnostic criterion. It may be inferred that normal black individuals who do not satisfy the established criteria for LVNC would have normal myocardial mechanics; however, this postulate needs to be validated in a larger study. Further work will determine whether RBR is indeed a robust characteristic of subjects with LVNC, or if it is indicative of some remodelling abnormality that occurs in LV dysfunction, since it has also been found in dilated cardiomyopathy[25] and in hypertensive patients with low ejection fraction.[26]

Conclusions

Left ventricular twist is impaired in patients with LVNC regardless of rotation pattern. In patients with LVNC and a normal pattern of rotation, a decrement in both apical and basal rotation results in diminished twist. Rigid body rotation occurs in 53.3% of subjects and can be clockwise or counterclockwise. The presence of RBR is not associated with worse LV remodelling compared with subjects with normal twist.

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IMAGE FOCUS

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Coronary aneurysm mimicking a five chamber heart

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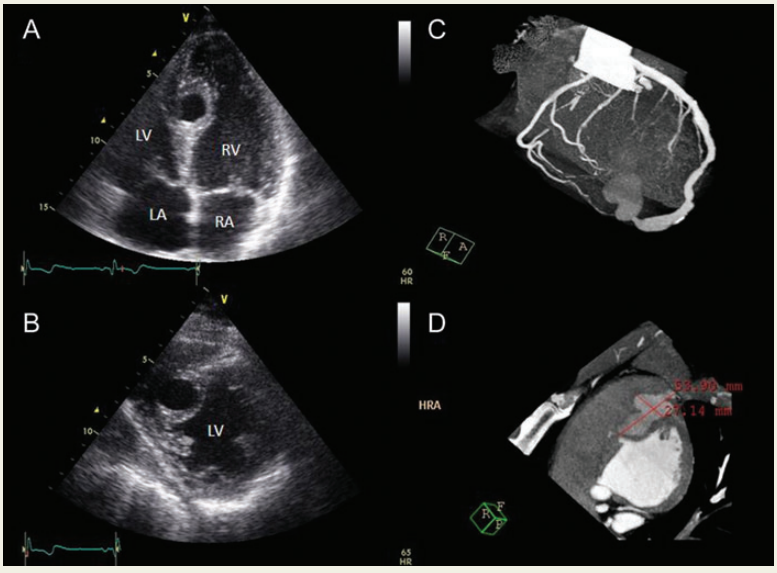
A 47-year-old soccer trainer was referred for a precordial murmur incidentally noted on routine physical examination. Cardiac auscultation revealed a grade 4/6 continuous murmur heard best on the left sternal border. No other sign was detected on physical examination. Chest X-ray and ECG showed no abnormalities. Transthoracic echocardiography revealed an echo-free cavity measuring 32 × 14 mm within the interventricular septum (*Panels A* and *B*; Supplementary data online, *Movie S1*). Colour Doppler imaging showed a continuous, mainly diastolic flow (Supplementary data online, *Movie S2*) within the structure, and a hyperaemic left anterior descending artery flow entering its cavity. The heart chambers, valves, and left ventricular performance were entirely normal.



Computed tomography angiography demonstrated a dilated left anterior descending artery streaming into a distinct cavity measuring 5 × 3 cm near the cardiac apex (*Panels C* and *D*). The diagnosis of communication between the artery and that structure was confirmed by conventional coronary angiography.

Giant coronary artery aneurysms (defined as aneurysms > 20 mm in diameter) are extremely rare. Therapeutic options include surgery or transcatheter closure. Although no clear consensus exists on the management of such a rare condition, clinically silent coronary artery aneurysms are reported to be well tolerated. A conservative approach is usually preferred.

Apical four chamber (*Panel A*), parasternal short-axis (*Panel B*) computed tomography angiography images of the cavity (*Panels C* and *D*).

Supplementary data are available at *European Heart Journal – Cardiovascular Imaging* online.