

The relationship between left ventricular late-systolic rotation and twist, and classic parameters of ventricular function and geometry

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Abstract

Background: Using speckle tracking echocardiography we investigated left ventricular (LV) twist and rotation (ROT) at the papillary muscle (PM) level and their correlation with standard echocardiographic and demographic parameters.

Aim: To assess whether the fulcrum of LV short axis ROT is shifted in myocardial disease.

Methods: The study group consisted of 33 patients (54±13 years old, 18 women). Left ventricular systolic function was normal in 6, and various degrees of wall motion abnormalities were present in the others [LV ejection fraction (LVEF) 49±15, wall motion score index (WMSI) 1.43±0.38]. Short axis images at basal, PM and apical level were analysed offline. The direction of ROT was determined from the apical aspect and expressed in degrees: clockwise (CW) in negative values, counter CW in positive. Twist is the arithmetic difference between apical and basal ROT.

Results: Left ventricular twist was in the range of 0.4-27.5 (14±7) degrees and correlated with LV systolic diameter (LVS), $r=-0.46$, 95% CI from -0.69 to -0.13, $p < 0.01$; LV diastolic diameter (LVD), $r=-0.40$, 95% CI from -0.65 to -0.06, $p=0.02$; and systolic motion score index of 6 mid segments (6S-MSI), calculated as WMSI at PM level, $r=-0.37$, 95% CI from -0.63 to -0.03, $p < 0.04$. Linear regression resulted in a model including interventricular septum systolic thickness (IVSS) and 6S-MSI, which predicted twist correctly in 21% of cases. Twist was independent of LVEF and overall WMSI. The PM ROT value correlated with: apical ROT, $r=0.36$, 95% CI 0.02-0.63, $p < 0.04$; posterior wall systolic thickness (PWS), $r=0.39$, 95% CI 0.05-0.64, $p < 0.03$. We distinguished Group A, $n=14$, with CW direction of PM ROT – negative values, range from -5.2 to -0.9; Group B, $n=19$, with counter CW, range 0.4-4.9. Apical ROT was 5 vs. 10 degrees, $p < 0.03$; PWS 14 vs. 15 mm, $p < 0.03$; diastolic posterior wall thickness 10 vs. 12 mm, $p < 0.04$, respectively. In univariate logistic regression, we identified independent factors related to counter CW PM ROT: apical ROT (OR=1.15, 95% CI 1.00-1.33, $p < 0.05$) and PWS (OR=1.71, 95% CI 1.03-2.84, $p < 0.04$). Multiple logistic regression resulted in a model predicting counterCW rotation at PM ($p < 0.01$) including: apical ROT (OR=1.18, 95% CI 1.00-1.38, $p < 0.05$) and PWS (OR=1.77, 95% CI 1.02-3.08, $p < 0.05$). ROC curves identified cut-off values of apical ROT > 11.3 deg and PWS > 13 mm. We found counterCW PM ROT in all patients with both conditions, 59% of patients with one, 22% with none.

Conclusions: Left ventricular twist is related to mid segments function and IVSS, while PM ROT value and its direction (associated with 'zero ROT level') is related to PWS and apical ROT, rather than to LVEF or WMSI. Thus twist and rotation may reflect novel aspects of LV function.

Key words: rotation, speckle tracking echocardiography, left ventricular function

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Introduction

Left ventricular (LV) twist results from apical and basal rotational motion in opposite directions (Figures 1 and 2). Speckle tracking echocardiography (STE) [1] has already been proven to be a reliable tool for the assessment of LV rotation. The method is angle-independent and shows

good correlation with measurements with tagged magnetic resonance imaging [2]. Ventricular twist, defined as the difference between apical and basal rotation, has been proposed as a novel marker of cardiac function [3]. In the literature, there is some inconsistency in using two different terms – 'torsion' and 'twist' which are used interchangeably. We decided to follow the definition

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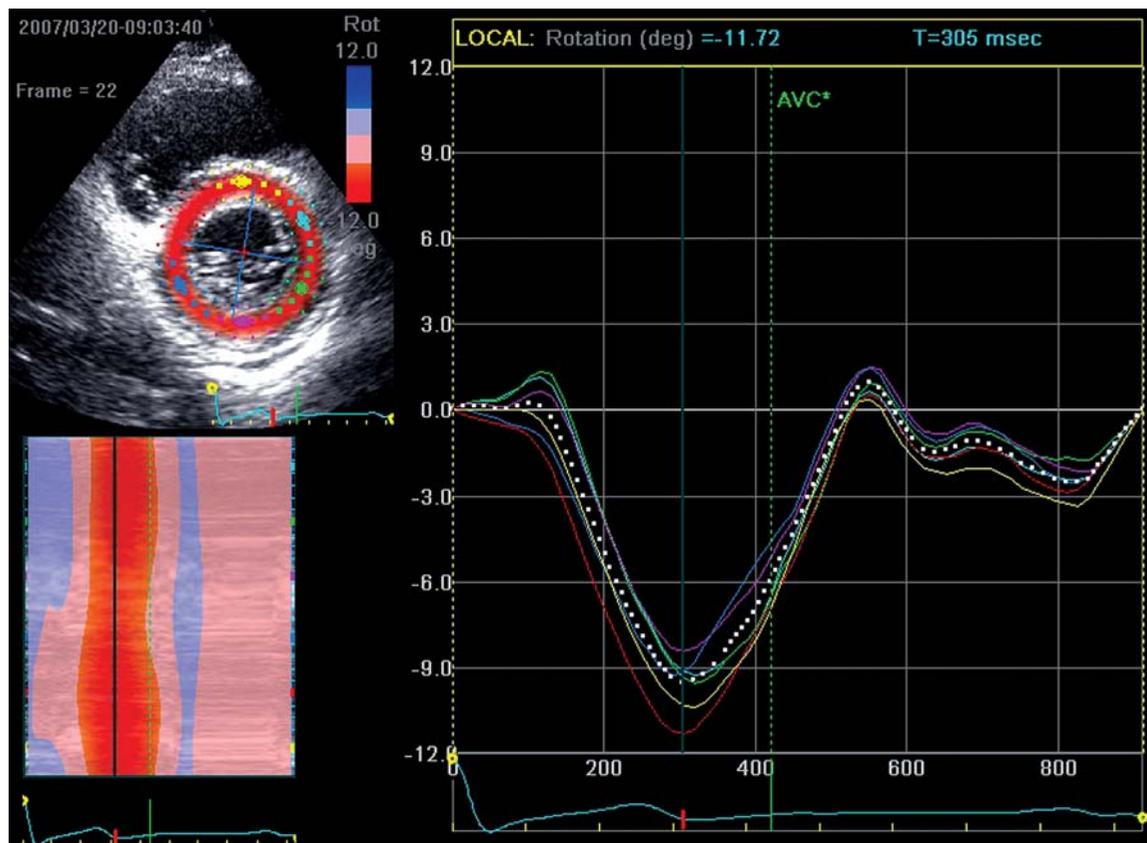


Figure 1. Rotation at the basal level

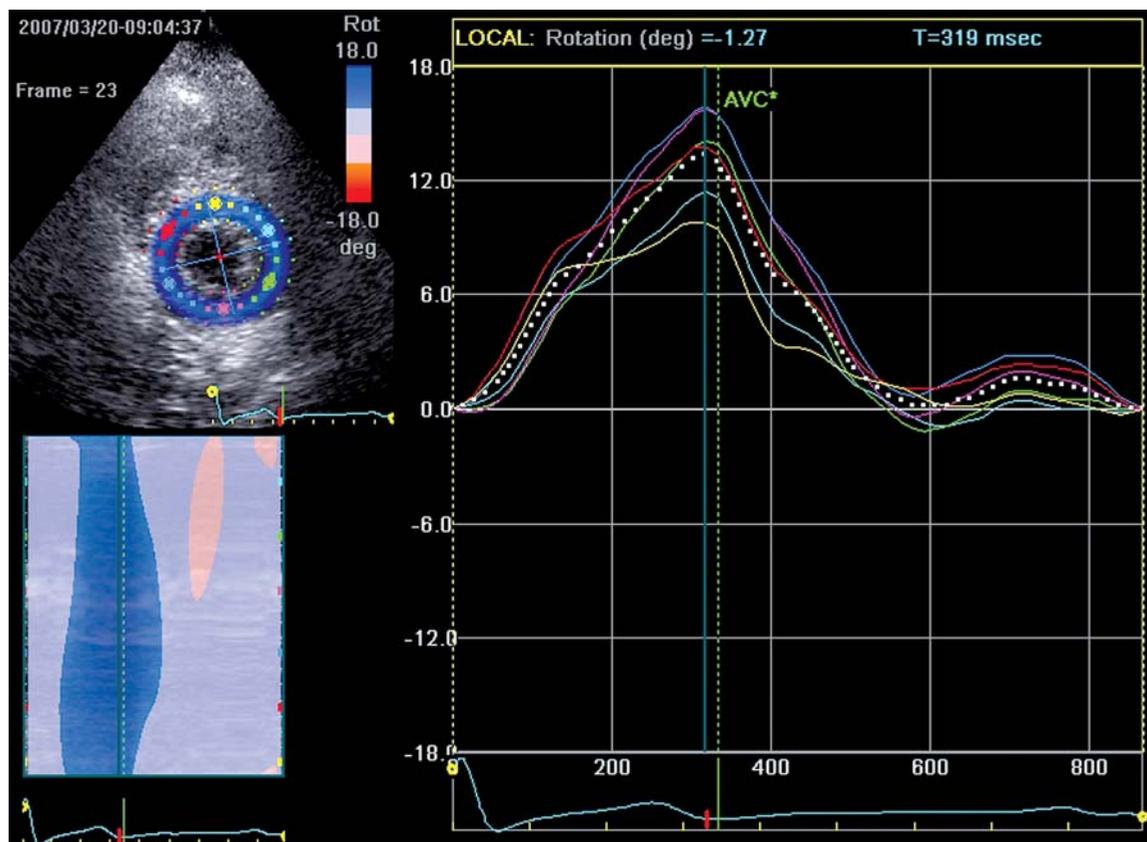


Figure 2. Normal, synchronous rotation at the apical level of the left ventricle

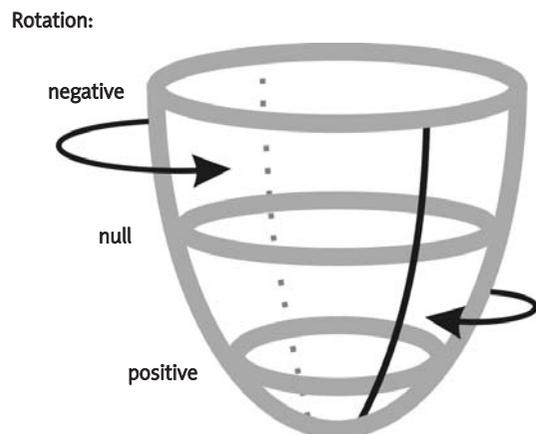


Figure 3. [After Helle-Valle T et al. [6], modified]

Table I. Demographic and baseline echocardiographic characteristics of the studied patients

Parameter	Value
Age [years]	54±13
Gender	
women	18 (54%)
men	15 (46%)
PM rotation direction	
counterclockwise	19 (58%)
clockwise	14 (42%)
Diastolic LV function (N=31)	
normal	12 (39%)
impaired	13 (42%)
pseudonormal	6 (19%)

Abbreviations: PM – papillary muscles, LV – left ventricle

of twist as the rotation difference, while torsion would be the twist value divided by the distance between the apical and basal level in centimetres [4, 5].

In this study, we aimed to define demographic and echocardiographic determinants of LV twist. Furthermore, we studied the LV rotation at the papillary muscle level, as we hypothesised that the equator or fulcrum (the relatively still area between the regions rotating in opposite directions – Figure 3) of LV short axis rotation is shifted in myocardial diseases.

Methods

We studied a group of 33 consecutive patients [54±13 years, 18 (54%) women], in whom we were able to obtain high-quality short axis images of LV. Besides basic two-dimensional examination, parasternal short axis images were taken with temporal resolution of 50-80 frames per second at 3 levels: basal (MV), papillary muscles (PM), apical (AP). Age, gender, body surface area

(BSA), LV mass, LV mass index, LV ejection fraction (LVEF), diastolic function (normal – E/A between 1 and 2 and E' >8 cm/s; impaired relaxation – E/A <1 or <0.5 in patients over 50 years of age; pseudonormal – enlarged left atrium and E/A between 1 and 2 but E' <8 cm/s, restrictive – enlarged left atrium, E/A >2 and E' <8 cm/s), diastolic diameter of LV (LVS, LVD), systolic and diastolic thickness of interventricular septum (IVSS, IVSD), systolic and diastolic thickness of LV posterior wall (PWS, PWD), diameter of left atrium (LA), diameter of right ventricle (RV), diameter of ascending aorta (Ao) as well as segmental systolic function of LV were assessed during the examination. Using the 16-segment model, we calculated overall wall motion score index (WMSI) and regional indexes for 3 levels (MV-, PM-, AP- WMSI) and also for regions of 3 coronary arteries (LAD, LCX, RCA region WMSI).

Echocardiography was performed with the GE Vivid 7 apparatus. Digitally stored data were analysed by one observer, using the speckle tracking principle with dedicated software: EchoPAC PC Version 6.1.0. Rotation values of three aforementioned LV levels were obtained.

The direction of systolic rotation was determined from the apical aspect, clockwise denoting negative values, expressed in degrees. Then we calculated LV twist according to the following formula: twist = apical rotation value – basal rotation value.

Of note, we always considered the maximum end-systolic rotation, but images at different levels were obtained during separate heart beats. Thus rotation values were not from the same heart beat, and moreover were not from the same moment of systole.

We also evaluated the direction of PM rotation (clockwise or counterclockwise) and the absolute value of this rotation, as well as correlation with other echocardiographic parameters.

Results

The left ventricular function was normal in 6 patients (LVEF ≥55% and WMSI=1). In the whole study group, mean LVEF was 49±15%. Mean WMSI was 1.43±0.38. Twenty two patients had ischaemic heart disease; the others presented various diseases such as dilated or hypertrophic cardiomyopathy, aortic stenosis, previous artificial valve implantation, patent foramen ovale (PFO) with atrial fibrillation (the last three patients were counted as 'normal'). Characteristics of the study group are presented in Tables I and II.

Left ventricular twist

We assessed the correlation between continuous variables and twist. Since not all variables presented with a normal distribution, Pearson's linear as well as Spearman's rank correlation coefficient were used when appropriate. The results are shown in Table III.

Table II. Detailed echocardiographic characteristics

Parameter	Whole group	Normal LV function	Abnormal LV function
Age [years]	54±13 (22-74)	42±12	55±12
TWIST [deg]	14±7 (0.4-27.5)	14.2±7.5	14±7
PM rotation [deg]	0.4±3.3 (-5.2-4.9)	0.8±4.6	0.3±3.0
MV rotation [deg]	-6.3±4.3 (-12.6-5.9)	-8.4±2.6	-5.8±4.4
AP rotation [deg]	7.7±6.3 (-5.8-21.1)	5.8±7.0	8.2±6.1
BSA (N=32)	1.9±0.3 (1.4-2.4)	1.7±0.2	1.9±0.3
LVEF [%]	49.1±15.0 (22-69)	65±1.1	45.6±14.4
E/A (N=31)	0.1±0.4 (0.4-2)	1.4±0.3	0.9±0.4
LVS [mm]	35.3±8.1 (25-56)	28.5±2.9	36.8±8.2
LVD [mm]	49.3±7.0 (38-68)	43.3±3.6	50.6±6.9
IVSS [mm]	14.4±2.1 (10-20)	13.2±1.7	14.6±2.1
IVSD [mm]	11.1±1.8 (8-16)	9.2±1.3	11.6±1.6
LA [mm]	40.4±6 (30-56)	36±4	41.4±6.0
PWS [mm]	14.4±1.8 (12-20)	13±1.1	14.7±1.8
PWD [mm]	11.1±1.6 (8-15)	9.2±1.0	11.6±1.4
Ao [mm]	32.9±5.1 (22-46)	29.8±5.6	33.6±4.8
RV [mm]	25±2.7 (18-30)	23.5±3.4	25.3±2.5
LV mass index (N=32)	131.6±40.2 (67-213)	87.5±21	141.7±36.6
LV mass (N=32)	252.5±94.4 (111-461)	147.5±42	276.8±86.2
WMSI (overall)	1.4±0.4 (1-2.4)	1±0	1.53±0.4
AP-WMSI	1.7±0.8 (1-2.9)	1±0	1.9±0.7
PM-WMSI	1.4±0.4 (1-2.4)	1±0	1.5±0.4
MV-WMSI	1.2±0.3 (1-2.4)	1±0	1.3±0.4
LAD region WMSI	1.5±0.5 (1-2.3)	1±0	1.6±0.5
LCX region WMSI	1.3±0.4 (1-2.7)	1±0	1.4±0.4
RCA region WMSI	1.5±0.5 (1-2.6)	1±0	1.6±0.5

Abbreviations: see 'Methods' section

In univariate analysis, beside the expected correlation with AP rotation value and MV rotation, LV twist showed a correlation with LVS, LVD, and PM-WMSI. Other parameters showed a non-significant trend for a linear correlation with twist: PM rotation, LVEF, IVSS, RV and RCA region WMSI.

Subsequently, stepwise multiple linear regression analysis resulted in a model explaining 21% of twist variability. The model included IVSS and PM-WMSI and was described with the following formula:

$$\text{twist} = 7.17 + 1.19 \times \text{IVSS} - 7.18 \times \text{PM-WMSI}, p=0.01.$$

Additionally, we distinguished a subgroup with WMSI=1 and with WMSI >1. TWIST did not differ between these subgroups (14.7±8.9 vs. 13.8±6.4 respectively, p=0.77). Also, twist did not differ significantly between the subgroup with LVEF ≥55% and with LVEF <55% (16.6±6.2 vs. 12.1±7

respectively, p=0.07). Moreover, twist did not differ between the subset of patients with normal LV systolic function (defined as both LVEF ≥55% and WMSI=1, n=6) and the rest of the study group (14.2±7.5 vs. 14±7, respectively, p=0.9) (Table II).

Determinants of rotation at the papillary muscle level

Among the studied continuous variables, only AP rotation (r=0.36, 95% CI 0.02-0.63, p <0.04) and PWS (r=0.39, 95% CI 0.05-0.64, p <0.03) were correlated with PM rotation.

Next, we distinguished 2 subsets of patients: group A (n=14) with clockwise direction of PM rotation (negative values) and group B (n=19) with counterclockwise direction of PM rotation (positive values). The subgroups differed significantly with regard to AP rotation, PWS and PWD (Table IV).

Table III. Results and correlation analysis between TWIST and echocardiographic variables

Parameter	r	95% CI	p
Age	-0.11	(-0.43, 0.25)	0.56
PM rotation	0.30	(-0.05, 0.58)	0.09
MV rotation	-0.46	(-0.70, -0.14)	0.01
AP rotation	0.80	(0.62, 0.90)	<0.01
BSA	-0.23	(-0.54, 0.13)	0.12
LVEF	0.34	(-0.01, 0.61)	0.05
E/A	0.09	(-0.27, 0.43)	0.63
LVS	-0.46	(-0.69, -0.13)	0.01
LVD	-0.40	(-0.65, -0.06)	0.02
IVSS	0.29	(-0.06, 0.58)	0.10
IVSD	0.14	(-0.22, 0.46)	0.44
LA	-0.25	(-0.54, 0.10)	0.16
PWS	0.10	(-0.25, 0.43)	0.57
PWD	0.03	(-0.32, 0.37)	0.88
Ao	-0.19	(-0.50, 0.17)	0.29
RV	-0.30	(-0.58, 0.05)	0.09
LV mass index	-0.15	(-0.47, 0.21)	0.42
LV mass	-0.23	(-0.54, 0.13)	0.20
WMSI	-0.26	(-0.55, 0.09)	0.14
AP-WMSI	-0.05	(-0.38, 0.30)	0.79
PM-WMSI	-0.37	(-0.63, -0.03)	0.04
MV-WMSI	-0.23*	(-0.53, 0.12)	0.19
LAD region WMSI	-0.15	(-0.47, 0.20)	0.40
LCX region WMSI	-0.17	(-0.48, 0.19)	0.35
RCA region WMSI	-0.33	(-0.60, 0.02)	0.06

* Spearman's rank correlation
Abbreviations: see 'Methods' section

In univariate logistic regression, AP rotation (OR=1.15, 95% CI 1.00-1.33, $p < 0.05$) and PWS (OR=1.71, 95% CI 1.03-2.84, $p < 0.04$) predicted counterclockwise direction of PM rotation. A non-significant trend was observed for IVSS and PWD.

Multiple logistic regression resulted in a model (Table V) predicting counterclockwise rotation at PM ($p < 0.01$) including: AP rotation and PWS.

The ROC curves identified cut-off values of apical rotation > 11.3 deg and posterior wall systolic thickness > 13 mm, to optimally predict PM rotation direction. We found counterclockwise rotation at the PM level in all patients fulfilling both criteria, in 59% of patients with one and in 22% with none.

Discussion

Left ventricular rotation and twist

The main finding of our study is the independence of LV rotation and standard parameters of LV function. Left

Table IV. Comparison and echocardiographic variables between patients with clockwise (Group A) versus counterclockwise (Group B) direction of PM rotation

Parameter	Group A	Group B	p
Female gender	57% (n=8)	53% (n=10)	
Age [years]	49.6±12.0	57.0±12.9	0.11
TWIST [deg]	12.0±6.3	15.5±7.2	0.15
PM rotation absolute value [deg]	3.00±1.4	2.9±1.6	0.92
MV rotation [deg]	-6.9±4.7	-5.8±4.0	0.45
AP rotation [deg]	5.0±5.5	9.7±6.1	0.03
BSA*	1.9±0.3	1.9±0.2	0.98
LVEF [%]	47.7±17.4	50.1±13.5	0.66
E/A**	1.0±0.3	0.9±0.4	0.45
LVS [mm]	36.8±9.1	34.3±7.4	0.39
LVD [mm]	50.6±7.7	48.3±6.5	0.35
IVSS [mm]	13.6±1.9	14.9±2.1	0.07
IVSD [mm]	10.5±1.6	11.6±1.9	0.09
LA [mm]	38.9±6.9	41.6±5.1	0.19
PWS [mm]	13.6±1.5	14.9±1.8	0.03
PWD [mm]	10.4±1.5	11.6±1.6	0.04
Ao [mm]	32.6±3.9	33.0±5.9	0.82
RV [mm]	25.0±1.6	24.9±3.4	0.96
LV mass index*	126.4±41.6	135.6±39.7	0.53
LV mass*	241.9±92.3	260.8±97.9	0.58
WMSI (overall)	1.4±0.4	1.4±0.3	0.99
AP-WMSI	1.7±0.8	1.7±0.7	0.99
PM-WMSI	1.4±0.5	1.4±0.4	0.96
MV-WMSI	1.2±0.4	1.2±0.3	0.63
LAD region WMSI	1.5±0.6	1.5±0.4	0.97
LCX region WMSI	1.3±0.5	1.4±0.4	0.75
RCA region WMSI	1.5±0.5	1.4±0.4	0.86

* $n=14$ in Group A, $n=18$ in Group B;
** $n=13$ in Group A, $n=18$ in Group B
Abbreviations: see 'Methods' section

Table V. Results of multiple logistic regression analysis

Parameter	OR	95% CI	p
AP rotation	1.18	(1.0001, 1.3851)	0.0498
PWS	1.77	(1.0170, 3.0822)	0.0434

ventricular hypertrophy and apical function seem to determine the level of zero rotation ('fulcrum') of LV.

Left ventricular twist is believed to play an important role in LV systolic function and filling. Twist is dependent on active myocardial performance as well as anatomical

conditions. In healthy humans, LV twist assessed by STE was reported to be -14.5 ± 3.2 degrees [6]. Twist is supposed to increase in normal human hearts during maximal exercise [7].

There are reports that twist is related to LVEF [8]. Possible changes of twist with age are still a matter of discussion [8, 9]. Some authors have observed a significantly higher peak of LV twist with advancing age, while the rate of LV untwisting was significantly reduced and delayed [9].

Although our study group was small, it was representative for a range of varying grades of LV dysfunction. In our group, with the majority of patients presenting LV systolic and diastolic dysfunction, mean twist (14 ± 7) was surprisingly close to the proposed normal value. Also our 'normal LV function' and 'abnormal LV function' subgroups presented similar LV twist (Table II).

Our results suggest that twist is not simply predicted by standard LV function parameters. Moreover, in our group we did not find a contribution of aging to LV twist, even though this was outside the main scope of our study.

According to our results, LV twist is determined by systolic function of middle-level LV segments and interventricular septum systolic thickness. In our group LV twist was not related to standard parameters of LV systolic function such as LVEF or WMSI. Also we found no correlation with diastolic function, assessed with normal /impaired /pseudonormal /restrictive classification. Further studies are needed to expand the evidence on determinants of LV rotation and twist.

Rotation at the papillary muscle level

It has been shown that LV rotation is minimal at the short-axis equatorial level, located approximately one third from the base to apex. Since basal and apical regions of LV rotate in opposite directions, a fulcrum of this movement must exist representing the zero rotation level [10]. The translation of this fulcrum level could reflect abnormal LV rotation, resulting from myocardial pathology (considered as LV geometry and function rather than aetiology due to a particular disease). Using short axis images acquired at 3 levels, we assumed that rotation at the PM level potentially interacts with the LV fulcrum, so that minimal rotation at the PM level may indicate that the equator is shifted to this position.

According to our results, PM rotation is determined by myocardial thickness and amplitude of apical rotation. We found counterclockwise rotation at the PM level in all patients with both apical rotation value >11.3 deg and posterior wall systolic thickness >13 mm. The direction of PM rotation may therefore reflect apical LV function and myocardial thickness. Papillary muscle rotation is not related to LVEF or WMSI; thus possibly it may reflect novel aspects of LV function.

Magnetic resonance imaging (MRI) was the first modality to visualise rotational deformation of LV [11].

However, it is not easily accessible and thus there are only a few published studies using this technique. Speckle tracking echocardiography, a simple and reliable method, validated with MRI [6, 12], allows more intense studies of rotation and twist. Previously proposed Doppler tissue imaging, though showing good correlation with MRI, has the shortcoming of angle-dependency and requires complicated calculations [13].

Limitations

We are aware of several limitations of our study. The study group was small. Importantly, the 'normal' subgroup was also very small. We used only 3 sampling planes and no long axis parameters were studied. The images were recorded during one examination but not during the same heart cycle. Rotation at different levels was in fact assessed in different cardiac cycles at variable time points. This may influence the exactitude of the calculated twist value. The measurements of rotation were made by a single observer.

Also, we have no comparison with other methods of rotation assessment.

Conclusions

We found that the direction of papillary muscle rotation is related to apical LV function and myocardial thickness. In other words, equatorial level appears to be shifted, depending on ventricular geometry and function. Rotation and twist are independent of standard left ventricular function parameters and may reflect different aspects of its performance. Further studies are necessary to understand their utility and practical value.

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Skrećenie i późnoskurczowa rotacja lewej komory – porównanie ze standardowymi wskaźnikami echokardiograficznymi funkcji i geometrii lewej komory

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Streszczenie

Wstęp i cel: Metodą śledzenia plamki (ang. *speckle tracking echocardiography*) ocenialiśmy skrećenie (*twist*) lewej komory (LV) i późnoskurczową rotację (ROT) na poziomie mięśni brodawkowatych (PM) oraz ich korelację ze standardowymi parametrami echokardiograficznymi i demograficznymi. Ponadto wysunęliśmy hipotezę, że równik rotacji LV może być przesunięty w chorobach miokardium.

Metodyka: Do analizy włączono 33 chorych (wiek 54 ± 13 lat, 18 kobiet). Funkcja skurczowa LV była prawidłowa u 6, pozostali mieli różnego stopnia zaburzenia kurczliwości [frakcja wyrzutowa LV (LVEF) $49 \pm 15\%$, wskaźnik kurczliwości lewej komory (WMSI) $1,43 \pm 0,38$]. Zarejestrowano obrazy w krótkiej osi na 3 poziomach – podstawy, PM i koniuszka LV. Analizowano je w trybie *offline* przy użyciu specjalistycznego oprogramowania. Kierunek ROT oznaczany jest z perspektywy koniuszka i wyrażany w stopniach – zgodny z kierunkiem wskazówek zegara (CW) przyjmuje wartości ujemne, przeciwny – dodatnie. *Twist* to arytmetyczna różnica wartości między ROT koniuszka i podstawy.

Wyniki: *Twist* LV zawart się w przedziale $0,4$ – $27,5^\circ$ (średnia $14 \pm 7^\circ$) i korelował z wymiarem skurczowym LV (LVS), $r = -0,46$, 95% CI $(-0,69, -0,13)$, $p < 0,01$; wymiarem rozkurczowym LV (LVD), $r = -0,40$, 95% CI $(-0,65, -0,06)$, $p = 0,02$; i indeksem kurczliwości 6 środkowych segmentów (6S-MSI) obliczonym analogicznie do WMSI na poziomie PM, $r = -0,37$, 95% CI $(-0,63, -0,03)$, $p < 0,04$. Możliwe było utworzenie modelu uwzględniającego skurczowy wymiar przegrody międzykomorowej (IVSS) i 6S-MSI, prawidłowo przewidującego *twist* w 21% przypadków. *Twist* nie wykazywał zależności od LVEF ani od całkowitego WMSI. Wartość PM ROT korelowała z: koniuszkową ROT, $r = 0,36$, 95% CI $(0,02, 0,63)$, $p < 0,04$; skurczowym wymiarem tylnej ściany LV (PWS), $r = 0,39$, 95% CI $(0,05, 0,64)$, $p < 0,03$. Wyróżniliśmy 2 grupy – grupę A, $n = 14$, ze zgodnym z CW kierunkiem PM ROT – wartości ujemne, zakres od $-5,2$ do $-0,9$; grupę B, $n = 19$, z przeciwnym do CW kierunkiem PM ROT, zakres $0,4$ – $4,9$. Koniuszkowa ROT wynosiła odpowiednio 5 vs 10° , $p < 0,03$; PWS 14 vs 15 mm, $p < 0,03$; rozkurczowa grubość tylnej ściany 10 vs 12 mm, $p < 0,04$. W analizie jednoczynnikowej znaleźliśmy niezależne czynniki związane z kierunkiem PM ROT przeciwnym do CW: koniuszkowa ROT [OR=1,15, 95% CI (1,00, 1,33), $p < 0,05$] i PWS [OR=1,71, 95% CI (1,03, 2,84), $p < 0,04$]. Model przewidujący przeciwny do CW kierunek PM ROT ($p < 0,01$) wykorzystywał koniuszkową ROT [OR=1,18, 95% CI (1,00, 1,38), $p < 0,05$] i PWS [OR=1,77, 95% CI (1,02, 3,08), $p < 0,05$]. Krzywe ROC wyznaczyły wartości odcięcia dla koniuszkowej ROT $> 11,3^\circ$ i PWS > 13 mm. Stwierdziliśmy przeciwny do CW kierunek PM ROT u wszystkich chorych spełniających oba warunki, u 59% spełniających jeden i u 22% niespełniających żadnego.

Wnioski: *Twist* LV jest związany z funkcją środkowych segmentów i IVSS, natomiast wartość i kierunek PM ROT (powiązane z poziomem „równika” – wypadkowego braku rotacji) są związane z PWS i koniuszkową ROT, nie zależą od LVEF ani od WMSI. Zatem *twist* i rotacja mogą odzwierciedlać nowe aspekty funkcji LV.

Słowa kluczowe: rotacja, echokardiografia, funkcja lewej komory

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