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ORIGINAL PAPER

Left ventricular remodeling and torsion dynamics in hypertensive patients

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Abstract Left ventricular (LV) torsion is a fundamental component of wall motion and plays an important role to optimize ventricular ejection fraction. The aim of our study was to calculate by speckle tracking echocardiography LV twist angle in patients with hypertension and LV remodeling, analyzing torsional indices in all patterns of hypertrophy, in comparison to torsional dynamics of age-matched healthy subjects. Hypertensive patients ($n = 202$) were divided in three groups, patients with concentric remodeling ($n = 70$), concentric hypertrophy ($n = 68$) and eccentric hypertrophy ($n = 64$), in relation to the echocardiographic measurements of relative wall thickness and LV mass, analyzing their torsional patterns by speckle tracking in comparison to age-matched control group. Compared to healthy controls, LV twist angle was increased in patients with hypertension and concentric remodeling (15.2° ± 1.9° vs. 11.0° ± 1.6°; $p < 0.001$), reaching the highest value in patients with concentric hypertrophy (19.4° ± 2.6°); instead LV twist angle presented depressed in the group of patients that presented eccentric hypertrophy (5.0° ± 1.1°). Regarding LV untwisting rate, it was higher in the concentric remodeling and concentric hypertrophy groups (−123.1°/s ± 12.1°/s and −145.1°/s ± 15.5°/s, respectively) in comparison with the controls (−90.0°/s ± 10.1°/s; $p < 0.0001$ for both). Instead, lower values of LV untwisting rate were observed in the eccentric remodeling group (−81.6°/s ± 8.1°/s), not significantly different to controls' values ($p = 0.09$). Enhanced LV twist angle appears to be a compensatory mechanism in hypertensive patients during the earlier stages of concentric remodeling and concentric hypertrophy; this hyper-torsion is inevitably loss in the more advanced stage of eccentric hypertrophy.

Keywords Echocardiography · LV function · LV torsion · Hypertension

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Background

Left ventricular (LV) torsion is described as the overall twisting or wringing motion of the heart caused by an opposite rotation of the base and apex, created by the contraction of its oblique spiral fibers [1]. It has been shown experimentally as well as clinically that the torsional behavior of the LV closely parallels changes in its global ejection performance [2]; in fact the dynamic interaction between subendocardial and subepicardial fiber helices plays an important role in optimizing LV ejection fraction [3]. Moreover, recent studies [3, 4] have demonstrated the influence of cardiac shape on LV function, showing that LV myocardial fiber architecture is crucial for an efficient LV performance. These researches have suggested that LV torsion is a fundamental component of LV wall motion that should be considered in assessing ventricular function and that various clinical conditions such as hypertrophy may affect such torsional movement and consequently LV function by altering the distribution of LV myocardial wall stress [5]. In fact in patients with severe LV remodeling in term of size and shape and severe systolic and diastolic dysfunction, torsional movement is depressed, due to the loss of the oblique architecture of the ascending and descending apical loop fibers [4]. In patients with hypertension, LV systolic pressure overload results in

various LV geometric changes, determining progressively an increase of the relative wall thickness (RWT) and/or of LV mass which are usually associated with alterations in diastolic function with preserved global systolic function until the latter stages of the disease [6, 7].

In echocardiographic practice, calculation of LV mass and RWT allows identification of concentric remodeling (normal LV mass with increased RWT) and permits categorization of an increase in LV mass as either concentric (RWT $\geq$ 0.42) or eccentric (RWT $\leq$ 0.42) hypertrophy.

The development of speckle tracking echocardiography (STE) has facilitated the simple and angle-independent measurement of all the components of LV myocardial deformation and it has recently been proposed and validated as a feasible method for measuring LV torsion [8–11].

The aim of our study was to evaluate by STE LV torsion in patients with hypertension and LV remodeling, analyzing LV torsion in all patterns of hypertrophy: concentric remodeling, concentric hypertrophy and eccentric hypertrophy, in comparison to torsional dynamics of age-matched healthy subjects.

Methods

Study population

The study population included 202 patients with arterial hypertension (mean duration 8.9 $\pm$ 6.1 years), referring to our Echo Laboratory for a diagnostic examination from January of 2010 to September of 2011. In this study population we divided hypertensive patients in three groups according to the last guidelines, in relation to RWT and LV mass measurements: 70 patients with concentric remodeling, 68 patients with concentric hypertrophy and 64 with eccentric hypertrophy, Sixty age-matched healthy subjects, who did not have history of cardiovascular disease and HTN were recruited as controls. The control subjects had no abnormal findings at physical examination, electrocardiogram and baseline echocardiography and did not take any cardiac medications. To be eligible, all patients were required to have no: evidence of secondary hypertension by extensive clinical, laboratory and instrumental examinations; $\geq$2+ valvular regurgitation; any degree of valvular stenosis; overt coronary artery disease (defined by at least one of the following: history of effort angina, coronary acute syndrome or revascularization procedures; evidence of positive exercise stress test; segmental wall motion abnormalities at echocardiography); hypertrophic cardiomyopathy; atrial fibrillation or other major arrhythmias; previous pacemaker implantation or heart transplantation; inadequate acoustic windows. In addition, all were required to have preserved LV systolic function (LV ejection fraction (EF) $\geq$55 %). Hypertension was defined by anthypertensive treatment or recorded blood pressure measurements $\geq$140 mmHg systolic and/or $\geq$90 mmHg diastolic on $\geq$2 occasions. Type and number of anthypertensive medications used were recorded at clinical evaluation. All subjects gave their written informed consent for the participation to the study. All work was in compliance with the declaration of Helsinki.

Standard echocardiography

Echocardiographic studies were performed using a high-quality echocardiograph (Vivid 7, GE, USA), equipped with a 2.5 MHz transducer. Subjects were studied in the left lateral recumbent position. Measurements of LV and left atrial dimensions, LV ejection fraction, and diastolic LV filling velocities were made in accordance with current recommendations of ASE [12]. LV EF, measured using Simpson's method, was used as a standard index of global LV systolic function. For determination of the presence of LV hypertrophy, LV mass, measured following ASE criteria, was indexed to height, according to the Framingham convention [13], with a partition value of 100 g/m for women and 127 g/m for men. The ratio between peak early (E) and late (A) diastolic LV filling velocities was used as standard indices of LV diastolic function [14]. LA volumes were measured using the area-length method, from the apical four and two chamber views. LA volumes were subsequently indexed by body surface area (BSA). The time interval between the onset of the QRS on the electrocardiogram and the aortic and mitral valve opening and closure were measured using pulsed-wave Doppler from the LV outflow and inflow, respectively.

Tissue Doppler imaging and M-mode annulus excursion analysis

Left ventricular longitudinal function was explored by pulsed Tissue Doppler imaging, placing the sample volume at the level of mitral lateral annulus from the apical four-chamber view [15]. Peak systolic (Sm), early diastolic (Em), and late diastolic (Am) annular velocities were obtained. Sm was considered as a relatively load-independent index of LV longitudinal systolic function. Em and the derived Em/Am ratio were used as load-independent markers of ventricular diastolic relaxation [16]. The E/Em ratio was also calculated and used as a reliable index of LV filling pressures [17]. M-mode measurements of mitral annular plane systolic excursion (MAPSE) was performed by placing the cursor perpendicular to the lateral site of the annulus [18].

Speckle tracking echocardiography

For the assessment of LV twisting, LV short-axis planes were obtained at basal and apical levels at adequate frame rates (range 60–90 FPS). The basal plane was defined as that allowing visualization of the mitral valve, whereas the apical plane was acquired distally to the papillary muscles. Particular care was taken to obtain LV cross-sections as circular as possible. Three consecutive cardiac cycles were recorded during breath hold, and saved in digital cine-loop format for subsequent off-line analysis. STE analysis was performed using a dedicated software (Echo Pac, GE, USA). After manual demarcation of LV endocardial border using a point-and-click approach, a circular region of interest including the whole short-axis LV myocardial area was depicted by the software. The software then divided each region of interest in 6 radial segments and tracked myocardial speckles frame-to-frame within each segment, with the possibility of manual adjustment in case of segments with poor tracking quality. LV rotation curves for each segment and two mean curves representing basal and apical rotation were obtained. All curves were calculated as the average of three consecutive cardiac cycles. By convention, clockwise rotation as viewed from the apex was expressed as a negative angle, whereas counterclockwise rotation was expressed a positive angle. LV twist was calculated as the instantaneous net difference in mean rotation between the apical and basal levels. Peak LV twist angle was measured. LV untwisting rate was calculated as the early diastolic peak time derivative of the time-twisting angle curve.

Reproducibility

The reproducibility of measurements was assessed in a subset of 20 patients randomly selected from the study population. For the assessment of inter-observer variability, images were independently analyzed by a second experienced investigator blinded to the results of standard echocardiography and not involved in image acquisition. To investigate the test retest reliability of all the LV twist indices mentioned above, we calculated the intraclass correlation coefficient (ICC), with an ICC value from 0.75 to 1.0 indicating excellent reliability [19].

Inter- and intra-observer variation coefficients of LV twist angle, untwisting rate, and time-to-peak LV twist were all <7.0 % and ICC of these parameters were all >0.85, demonstrating a low intra e inter-observer variability and a good test retest reliability of LV twist parameters.

Statistical analysis

Data are shown as mean ± SD. A p value <0.05 was considered statistically significant. Pearson's correlation coefficients were calculated to assess the relationships between continuous variables. Multiple regression analysis was performed to explore the independent determinants of speckle tracking measures of LV twist angle. Analyses were performed using the SPSS (Statistical Package for the Social Sciences, Chicago, IL, USA) software Release 12.0.

Results

Clinical and echocardiographic characteristics

Table 1 shows the clinical and standard echocardiographic characteristics of the study population. No significant difference was observed between groups regarding age, sex, heart rate and body mass index. LV ejection fraction was in the normal range and not significantly different between all pathological groups' patients and controls. As listed in Table 1, there were also no significant differences in medical therapy between groups.

Comparison of LV torsional parameters in controls and in patients with concentric remodeling, concentric hypertrophy and eccentric hypertrophy

Among a total of 3,144 segments analyzed, the software was able to correctly track 2,920 (92.9 %) segments. To examine the relationship between torsional indices and each underlying pattern of hypertrophy, LV torsional parameters of the three patients groups and controls were compared (Table 1; Figs. 1, 2). LV twist angle was higher in the concentric remodeling group (15.2° ± 1.9°) in comparison with the controls (11.0° ± 1.6°; $p < 0.001$); LV twist angle was further increased in the concentric hypertrophy group (19.4° ± 2.6°), instead presented lower values in eccentric hypertrophy group (5.0° ± 1.1°) (overall $p < 0.0001$ by ANOVA, $p < 0.05$ for all pair-wise comparisons). Regarding LV untwisting rate, it was higher in the concentric remodeling and concentric hypertrophy groups (−123.1°/s ± 12.1°/s and −145.1°/s ± 15.5°/s, respectively) in comparison with the controls (−90.0°/s ± 10.1°/s; $p < 0.0001$ for both). Instead, lower values of LV untwisting rate were observed in the eccentric remodelling group (−81.6°/s ± 8.1°/s), not significantly different to controls' values ($p = 0.09$) (Fig. 3).

Relationships of LV twist angle with clinical and echocardiographic variables in pathological groups

In three pathological groups, LV twist angle correlated significantly with LV mass index ($r = 0.27$; $p = 0.0005$),

Table 1 Characteristics of the study population ($n = 262$)

	Controls ($n = 60$)	Concentric remodeling ($n = 70$)	Concentric hypertrophy ($n = 68$)	Eccentric hypertrophy ($n = 64$)
Age	65.0 ± 15.5	64.8 ± 8.8	66.1 ± 10.6	65.4 ± 8.5
Gender (% female)	47.9	48.2	46.9	46.8
Height (cm)	166.5 ± 8.4	164.9 ± 7.4	166.4 ± 7.5	168.1 ± 10.0
Weight (kg)	74.2 ± 9.9	73.6 ± 10.8	76.4 ± 14.9	76.6 ± 12.8
Body surface area (m^2)	1.78 ± 0.2	1.78 ± 0.15	1.79 ± 0.19	1.76 ± 0.3
Body mass index (kg/m^2)	27.2 ± 2.2	27.0 ± 3.2	27.4 ± 5.0	27.1 ± 3.1
Systolic blood pressure (mmHg)	121.0 ± 10.1	135.5 ± 11.9*	136.2 ± 10.1*	131.6 ± 12.2*
Diastolic blood pressure (mmHg)	77.3 ± 5.5	79.6 ± 6.0	80.3 ± 5.5	77.7 ± 5.3
Heart rate (bpm)	74.0 ± 9.3	76.1 ± 9.8	76.5 ± 10.1	73.6 ± 9.4
Echocardiographic data				
End-diastolic LV diameter (mm)	42.2 ± 4.3	42.3 ± 3.5	49.0 ± 3.4*	55.5 ± 6.4*, ‡
End-diastolic LV volume (ml)	84.1 ± 25.1	85.3 ± 29.0	98.7 ± 30.1*	105.7 ± 33.7*, ‡
Relative wall thickness	0.39 ± 0.06	0.52 ± 0.04*	0.52 ± 0.04*	0.39 ± 0.04
LV mass index (g/m)	83.6 ± 26.3	98.2 ± 15.8*	152.9 ± 27.8*, †	148.9 ± 38.7*, †
LV ejection fraction (%)	59.0 ± 7.2	58.3 ± 5.4	57.5 ± 6.4	58.6 ± 7.2
Left atrial area (cm^2)	13.9 ± 3.8	14.4 ± 3.6	19.1 ± 3.2*	19.5 ± 3.9*
Left atrial volume (ml)	43.5 ± 8.6	46.2 ± 7.8	68.3 ± 10.1*	70.0 ± 12.1*
Mitral E/A ratio	1.02 ± 0.41	0.77 ± 0.3*	0.87 ± 0.49*	0.98 ± 0.42†
E/Em ratio	5.45 ± 3.32	6.81 ± 3.40	8.6 ± 4.4*	7.4 ± 4.30*
LV twist angle (°)	11.2 ± 3.2	15.6 ± 3.4*	19.4 ± 3.5*, †	5.1 ± 3.1*, †, ‡
LV untwisting rate (°/s)	−90.3 ± 8.5	−123 ± 11.5*	−145 ± 13.6*, †	−81.3 ± 9.2†, ‡
Medical therapy				
Ace-inhibitors or ARB	–	30 (42.8 %)	30 (44.1 %)	28 (43.8 %)
Beta-blockers	–	16 (22.8 %)	19 (27.9 %)	19 (29.7 %)
Calcium antagonists	–	20 (28.5 %)	19 (27.9 %)	16 (25.0 %)
Loop diuretics	–	13 (18.6 %)	15 (22.0 %)	16 (25.0 %)
Statins	–	30 (42.8 %)	32 (47.0 %)	29 (45.3 %)

* $p < 0.05$ versus controls by the Scheffé pairwise comparison test

† $p < 0.05$ versus concentric remodeling group by the Sheffè pairwise comparison test

‡ $p < 0.05$ versus concentric hypertrophy group by the Sheffè pairwise comparison test

relative wall thickness ($r = 0.25$; $p = 0.001$), body mass index ($r = 0.14$; $p = 0.01$) and heart rate ($r = 0.12$; $p = 0.05$). Other clinical and echocardiographic parameters were not related to LV twist angle. Stepwise multivariate regression analysis was performed among clinical and echocardiographic indices to detect independent determinants of LV twist angle; the parameters analyzed were age, gender, systolic blood pressure, heart rate, end-diastolic LV diameter and volume, relative wall thickness, LV mass index, LV ejection fraction, LA area e volume, mitral E/A ratio e E/E′ ratio. The analysis showed that only LV mass index ($\beta = 0.240$, $p = 0.0005$) and relative wall thickness ($\beta = 0.210$, $p = 0.001$) were independently associated with LV twist angle, among all the parameters explored. The model explained 22.7 % of the variability in LV twist angle (overall model $p < 0.0001$).

Discussion

In our study, we demonstrated that LV torsion was increased in patients with hypertension and LV concentric remodeling, reaching the highest value in patients with concentric hypertrophy; with the progression of the hypertensive heart disease, LV torsion became depressed in the group of patients presented LV eccentric hypertrophy at the echocardiographic examination.

Our results were in agreement with previous researches [20–22] which have showed that among patients with diastolic dysfunction, the extent of LV torsion is dependent on the stage of diastolic dysfunction with an increase in torsion during the early stage and with its lessening in the advanced degree of diastolic dysfunction and in systolic heart failure.

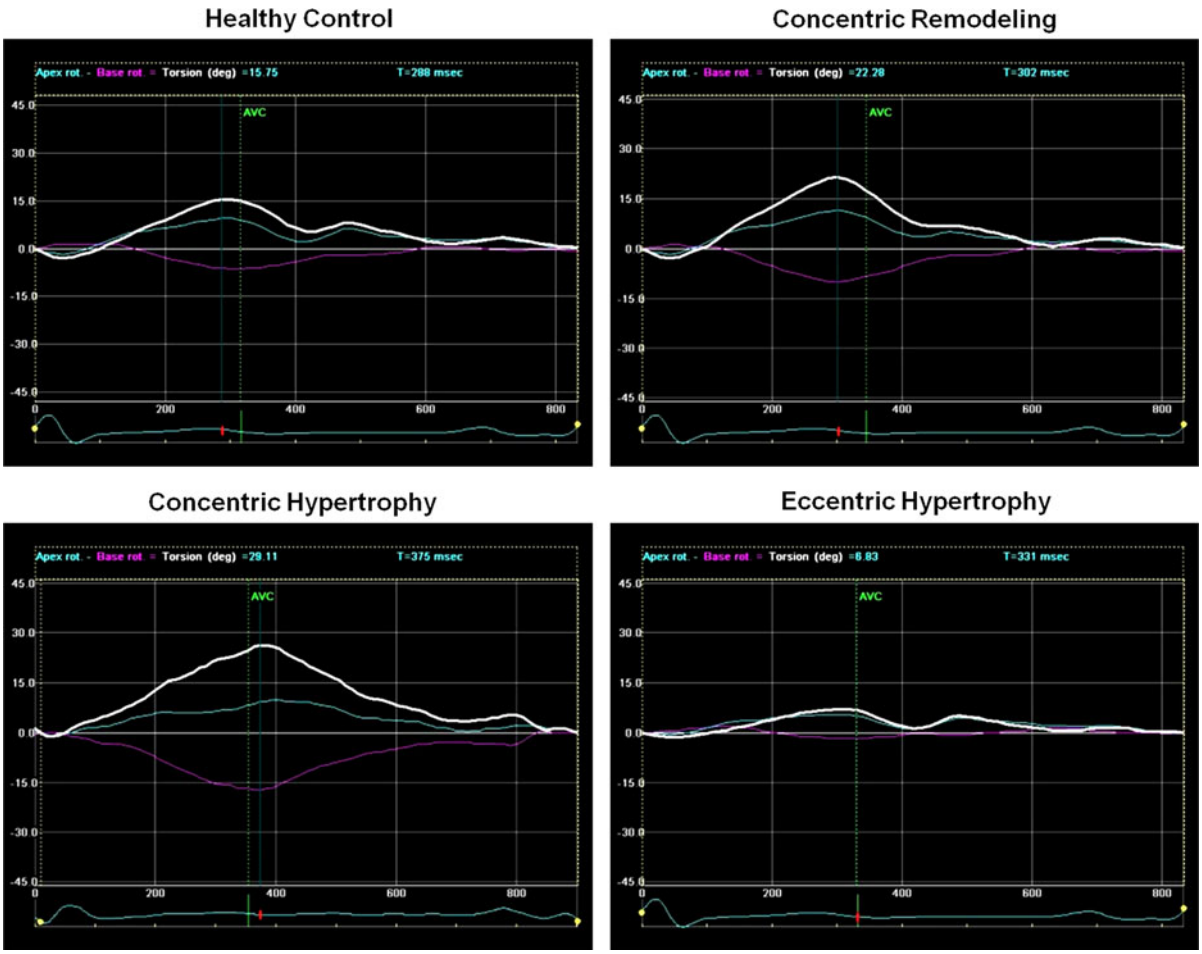

Fig. 1 LV twisting measured by STE in healthy subjects, in patients with concentric remodeling, concentric hypertrophy and eccentric hypertrophy. The *white line* represents LV twist angle, automatically calculated as the net different between the clockwise rotation of the base (*pink line*) and the counterclockwise movement of the apex (*blue line*)

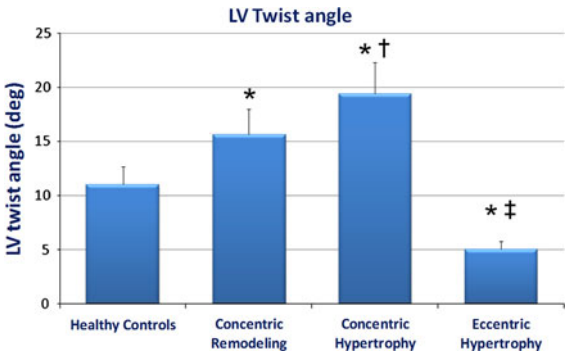

Fig. 2 LV twist angle values in accordance to different patterns of LV hypertrophy. Compared to healthy controls, left ventricular twist angle was increased in patients with hypertension and concentric remodeling, reaching the highest mean value in patients with concentric hypertrophy; instead LV twist appears depressed in the group of patients presenting eccentric hypertrophy. $^{*}p < 0.05$ versus controls by the Scheffé pair-wise comparison test. $^{\dagger}p < 0.05$ versus concentric remodeling group by the Scheffé pair-wise comparison test. $^{\ddagger}p < 0.05$ versus concentric remodeling and versus concentric hypertrophy by the Scheffé pair-wise comparison test

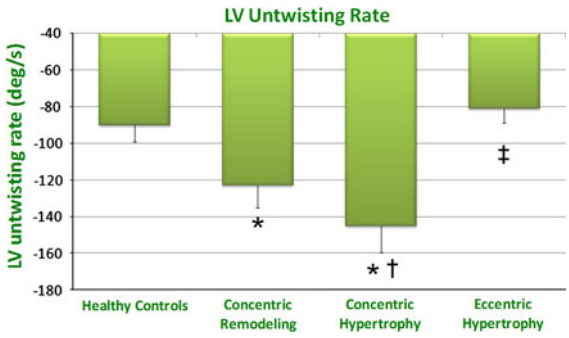

Fig. 3 Trend of LV untwisting rate respect to LV remodeling in the study population. LV untwisting rate appears higher in concentric remodeling and concentric hypertrophy groups in comparison to the controls; lower values of LV untwisting rate is observed in eccentric hypertrophy group, not significantly different to controls' values. $^{*}p < 0.05$ versus controls by the Scheffé pair-wise comparison test. $^{\dagger}p < 0.05$ versus concentric remodeling group by the Scheffé pair-wise comparison test. $^{\ddagger}p < 0.05$ versus concentric remodeling and versus concentric hypertrophy by the Scheffé pair-wise comparison test

Left ventricular twist dynamic represents the instantaneous rotational motion of the apex with respect to the base of the heart, able to generate a wringing movement of the LV that propels the blood out of LV cavity [23]; normally, the subendocardial and subepicardial layers oppose each other during contraction due to their oblique fiber angle orientations but greater force is generated in the epicardial layers as a result of the longer distance from the centre of the ventricle [24]. In such way, the epicardial fibers govern the direction of LV twist, mainly owing to their longer arm of movement, determining a counterclockwise torsional movement during systolic ejection. An increase in RWT with hypertrophy produces larger radius differences between endocardium and epicardium, resulting finally in an augmentation of peak torsion [25].

Furthermore, it is well known that in LV hypertrophy myocardial fibrosis, related to pressure overload, appears frequently in the subendocardial layer, leading to the impairment of longitudinal LV function and to a further dominance of subepicardial contraction [2].

It might potentially explain why in our study the LV torsion dynamics appeared increased in patients with concentric remodeling and much more in concentric hypertrophy; in fact when the LV walls are thicker in comparison to LV cavity dimension, the epicardial fibers become even more dominant and consequently the transmural gradient of myofibers shortening is greater than that in control subjects, resulting finally in an enhanced LV torsion [25].

With the progression of the hypertensive heart disease, as a consequence of LV dilatation, the LV takes on a more spherical geometry and this advanced LV remodeling and the consequent loss of the oblique architecture of loop fibers, leads to impair the LV torsional movement.

The reduction of twisting motion would increase endocardial stress and, therefore, increase oxygen demand, thereby reducing the efficiency of LV systolic function [26].

This result was in agreement with the recent study of van Dalen et al. [3] who have studied patients with dilated cardiomyopathy, demonstrating that an optimal myofiber helix angle exists and is related to a certain value of LV sphericity index; in fact, either an increase or decrease in LV sphericity index would result in a decrease in LV twist, confirming the hypothesis that a change in cardiac shape may indeed lead to a change in the arrangement of myocardial fibers and thereby to a change in LV torsion.

So at the an early stage of diastolic dysfunction, as in hypertensive patients with LV concentric remodeling and LV concentric hypertrophy, LV torsion is increased and represents the major determinant for maintaining LV pump function [27]; instead, in the latter stages of the disease, patients present eccentric hypertrophy, and thus this compensatory mechanism of hypertorsion is loss, leading to an impairment of LV systolic function.

Regarding LV untwisting rate, we found that it was higher in the concentric remodeling and concentric hypertrophy groups in comparison with the controls; instead, lower values of LV untwisting rate were observed in the eccentric remodelling group not significantly different to controls' values.

Wang et al. [28] has previously demonstrated that LV untwisting rate was primarily determined by LV systolic twist and LV end-systolic volume.

The clockwise recoil of twist, or untwisting, constitutes the deformation that largely occurs during the period of isovolumic relaxation; this recoil represents the release of restoring forces accumulated during systole and contributes towards diastolic suction, which is a major determinant of early LV filling [7].

Considering that increased LV twisting and increased diastolic untwisting have been proposed to be a compensatory mechanisms for impaired LV myocardial relaxation in the early stage of diastolic dysfunction [20], our findings may support the hypothesis that a physiological interaction between LV twisting during ejection and LV relaxation could modulate LV diastolic performance in hypertensive patients. Our group of study has previously demonstrated the positive association of LV twisting with heart rate [9] but in this study, heart rate was similar in all subgroup of patients, consequently LV untwisting rate followed closely the modifications of LV twist angle.

Thus, the assessment of torsional recoil, or untwisting, should provide an estimation of LV relaxation and may represent an additional parameter of diastolic dysfunction in hypertensive patients.

Advantages and clinical applications

Speckle tracking echocardiography is able to measure LV torsional deformation noninvasively by semi-automated tracking of speckles from apical and basal short-axis recordings. The validity of this technique was tested with sonomicrometry and MRI tagging as reference methods in animal and human models [29]. LV twisting assessment by STE shows dynamics, magnitudes and timing of peak basal and apical rotation, without the problem of angle-dependency and cardiac translation movement. The typical time spent for data analysis was <1 min; moreover the inter- and intra-observer variation coefficients of LV twist parameters were very low [29].

Due to its ability to differentiate between active and passive movement of myocardial segments and to evaluate components of myocardial function that are not visually assessable, STE allows comprehensive assessment of myocardial function

and the spectrum of potential clinical applications is actually very wide [8, 30].

In particular, a growing body of evidence suggests that the assessment of LV twisting is feasible and useful in several clinical settings [9, 26, 31]. The relationship between longitudinal and torsional mechanics of the LV provides insight into the transmural heterogeneity in myocardial contractile function. The presence of a sub-endocardial-to-subepicardial gradient in LV mechanics may provide a useful clinical measure for early recognition of a subclinical state that is likely to progress into either systolic or diastolic heart failure.

So LV twisting dynamics appears useful in detecting early LV dysfunction in the setting of systemic diseases with cardiac involvement such as arterial hypertension [26].

As a result, twisting assessment has been slow to get incorporated into everyday clinical practice. Despite the growing evidence in support of clinical implications of LV twist measurements using 2D STE, routine clinical use of this methodology is not recommended at this time [31]. Ongoing research and further technical development are likely to improve the quality of the data and the more general acceptance of this new modality of imaging in echocardiography.

Limitations

Some limitations need to be acknowledged in this study. LV longitudinal motion is a confounding factor for the assessment of LV torsion by two-dimensional STE, as different cross-sectional levels are explored during the cardiac cycle, particularly at the LV base [29]. Normalization of LV twist angle by end-diastolic length is to date the most used method to estimate LV torsion, but the possibility of measuring the true distance between the basal and apical levels used in the analysis would improve the accuracy of LV torsion measurements. Although we tried to define each slice by anatomical landmarks of the LV, we could not measure the exact distance between the scanned two levels. To overcome this problem, further developments in three-dimensional echocardiography may allow an even more comprehensive assessment of ventricular function [32].

Another important limitation of the clinical routine use of STE is that speckle quality in some cases was suboptimal in the tracking and visualization of the endocardium layer of the LV, so speckle tracking curves can vary even in the same patient, depending on 2D gray-scale resolution, and can determine in such way variant results, contributing to the variability of this parameter. Although there was a limited sample size, we were able to obtain novel findings; however this novel index needs further validation with larger and prospective studies also in the setting of hypertensive heart disease.

Conclusions

Enhanced LV torsion appears to be a compensatory mechanism in hypertensive patients during the earlier stages of concentric remodeling and concentric hypertrophy; but this hyper-torsion is inevitably loss in the more advanced stage of eccentric hypertrophy, probably because of the lack of the oblique architecture of myocardial fibers which happens as a consequence of LV remodeling in this kind of patients.

Conflict of interest The authors declare no conflict of interest.

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