Original Article

Usefulness of Pulsed-Wave Tissue Doppler Imaging of Mitral Valve Annulus for Assessment of Hypertensive Patients

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ABSTRACT

Aim: To assess the utility of tissue Doppler imaging (TDI) of mitral valve annulus (MVA) as preload-independent tool to quantify left ventricular function in long axis

Design: Cohort study conducted between January 2000 and March 2004

Setting: Non-invasive cardiac laboratory, Medicine department, Farwania Hospital, Kuwait

Patients and Methods: Ambulatory blood pressure (BP) monitoring, transthoracic echocardiography with Doppler study and treadmill exercise ECG test were done for 150 hypertensive patients and 50 normotensive subjects. There were two groups: group I: included 150 hypertensive patients and group II: included 50 normotensive subjects.

Results: There was a significantly decreased ventricular descent phase and recoil phase of M-mode of MVA ($p < 0.05$) and a significantly decreased E velocity and a significantly increased A velocity of mitral inflow pattern ($p<0.05$) in the hypertensive patients than the normotensives. Receiver operating characteristic (ROC) curve data of TDI indices in hypertensive patients revealed that systolic contraction velocity < 8 cm / second was a good indicator to detect the impaired LV longitudinal fiber shortening during systole (sensitivity = 84%, false +ve = 18% and area under curve = 0.837). Multivariate analysis revealed a significant relation between BP status and LV hypertrophy and impaired LV function in long axis in hypertensive patients ($p < 0.05$).

Conclusion: TDI can be considered as a clinically useful marker of impaired left ventricular function in long axis and an indicator of impaired relaxation of the longitudinal fibers of LV in hypertensive patients.

KEYWORDS: mitral valve annulus, tissue doppler imaging, ventricular function in long axis

INTRODUCTION

Annular ring motion of the mitral valve toward the ventricular apex begins during the isovolumetric contraction period and the subsequent ventricular contraction phase and the end of the J phase of pulmonary venous flow and is followed by the recoil or spring back of the annulus during the rapid phase of mitral blood flow into the ventricle. So, annular motion is essential for left atrial filling and this increases the net velocity of mitral flow and augments left ventricular filling process, as there is a 20% increase in relative velocity of blood from left atrium to left ventricle due to recoil motion of the mitral annulus[1].

Diastolic dysfunction of the LV refers to the impaired capacity to fill and maintain stroke volume, without a compensatory increase of atrial filling pressure, either at rest or during exercise. Impaired relaxation refers to the impairment of the rate or extent of relaxation of the examined ventricular wall or the entire unloaded ventricle during early diastole, defined according to the clinical and echocardiographic practice[2].

After more than two decades of experience, conventional Doppler echocardiography has become the principal tool in assessing diastolic function in daily practice. In addition, the anatomical and functional information provided by 2-dimensional echocardiography has reasonable accuracy in many forms of cardiac disease[3]. Preload dependency and the ensuing pseudo-normalization of flow patterns constitute major limitations of the technique to assess either ventricular relaxation or filling pressures separately.

In early diastole, left atrial pressure, ventricular relaxation and restoring forces interact on the atrioventricular pressure gradient that determines transmitral flow[4]. It is difficult to isolate the effects of these different factors in intact humans, not only because they mutually interact, but because they all are profoundly modified by ventricular filling. Tissue Doppler Imaging (TDI) has been used to assess regional myocardial wall motion and ventricular function[5].

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We began this study with the hypothesis that the TDI of mitral valve annulus (MVA) is a preload-independent tool and a valid technique to assess left ventricular (LV) longitudinal fiber function (shortening and lengthening) in hypertensive patients.

The aim of the study was to evaluate the utility of TDI of the MVA for the assessment of systolic function in long axis and diastolic relaxation of the left ventricle in hypertensive patients.

PATIENTS AND METHODS

Study patients:

One hundred and fifty hypertensive patients and 50 normotensive subjects were included in the study. All patients were referred by their physicians to cardiology clinic in Farwania Hospital with blood pressure more than 140/90 mmHg. Sixty patients complained of chest pain, 40 patients presented with shortness of breath (NYHA functional classification grade II), and 50 patients were referred for echocardiographic assessment of hypertension. All patients were evaluated clinically by looking at the history, physical examination, 12-leads ECG and routine laboratory investigations.

Exclusion criteria included patients with history of myocardial infarction, diabetes mellitus, cerebrovascular disease, valvular disease, calcification of mitral leaflets or mitral valve annulus, prolonged PR interval, sinus tachycardia, patients with summation of transmitral flow pattern and pregnant women. Exclusion was based on: medical history, physical examination, fundus examination, urine analysis for proteinuria and 12-lead electrocardiogram to avoid confounding factors.

Blood pressure measurements:

Mercury sphygmomanometer was used to measure office systolic and diastolic BP (mmHg). At least two measurements were recorded between 8 AM and 11 AM with the patients in a sitting position with the legs uncrossed and the feet on the floor. BP was measured after the patient had rested for 15 minutes. Cuff inflation pressure was then determined by palpating the disappearance and appearance of the radial pulse. BP was recorded twice, with approximately a two-minute interval. Ambulatory blood pressure was recorded with an auscultatory device (Accutracker II). Correct position of the microphone was done by palpating the brachial artery. Ambulatory BP was recorded during the day (6 AM to 10 PM) at one-hour intervals and during the night (10 PM to 6 AM) at two-hour intervals. Blood Pressure Load is the percentage of all systolic and diastolic BP recordings exceeding the threshold of 140/90 mmHg.

Echocardiographic study:

Two-dimensional and M-mode echocardiography was performed for all patients in the study. The leading edge to leading edge convention was used. Left ventricular dimensions were measured at or immediately below the tips of mitral leaflets and averaged over five heart cycles. Left Ventricular Mass (LVM) and Left Ventricular Mass Index (LVMI) were calculated.

Pulsed Doppler echocardiography was obtained from the standard apical four chamber view. Mitral inflow velocity was recorded with the sample volume at mitral annulus level. The transducer was then manipulated to obtain the maximal flow velocity as assessed by the auditory and spectral outputs. The Doppler measurements were made during at least three cardiac cycles using the darkest part of the spectral recording and were then averaged. The following measurements were obtained: peak velocity of early left ventricular filling (E), peak velocity of late left ventricular filling (A) and the ratio between early and late flow velocity (E/A).

Mitral valve annulus motion:

This was obtained with the M-mode cursor directed from the apical four chamber view. The cursor was oriented toward the bright septal margin of the annulus (fibrous trigone) and then toward the lateral margin. On each side, the beam was oriented so that it was perpendicular to the descent motion of the annular structure. Multiple M-mode recordings of the septal and lateral margins of the mitral annulus were made. The motion of the septal and lateral margins was similar, and thus, only the motion of the septal margin was reported[6]. Mitral valve annulus descent is a motion of the mitral annulus toward the apex and mitral valve annulus recoil is a movement of the mitral annulus toward the atrium[7].

TDI pattern during sinus rhythm:

A normal pattern consists of three major signals: a single systolic signal (Sm) and two distinct signals in early (Em) and late (Am) diastole, timed by the onset of early inflow and atrial contraction, respectively. During isovolumic contraction time (IVCTm) and isovolumic relaxation time (IVRTm), the displayed, smaller biphasic signals are presumed to be the result of brief geometrical changes that occur in the LV (induced by different timing of long axis and circumferential axis dynamics and by ventricular interdependence[8].

Treadmill exercise ECG test protocol:

All patients in the study underwent the exercise

ECG test using standard or modified Bruce models. Resting ECG was done for all patients to exclude patients with significant ST- segment changes, left bundle branch block or tachyarrhythmias. Blood pressure was recorded midway through each stage and at peak exercise. ST- segment level was measured 60 ms after the J point in all 12 leads. Exercise induced significant ST- segment depression was defined as horizontal or downsloping ST-segment depression ≥ 1mm in any lead present during exercise test.

Stress thallium scintigraphy:

Only 62 patients were known to have undergone stress thallium - 201 scintigraphy in the course of their clinical management. The SPECT (single-photon emission computed tomography) in Egypt, India and England. Ischemia by SPECT was defined as a stress perfusion defect in an area with normal perfusion at rest.

Statistical analysis:

Continuous variables are summarized as a mean ± standard deviation (SD). Comparison between two groups was performed with t-test for continuous variables and chi-square test for categorical variables. A p value < 0.05 was considered statistically significant and a p value < 0.01 was considered statistically highly significant. A stepwise multivariate regression model was used to identify possible independent variables associated with impaired TDI of the MVA. The strength of the association with impaired TDI - MVA was presented as 95% confidence intervals. Potential confounding of clinical variables was entered as independent variables.

Predictive indices: True positive (TP), true negative (TN), false positive (FP) and false negative (FN) were calculated. Sensitivity = TP / (TP+FN), specificity = TN / (TN+FP). False positive = 1- specificity.

Receiver operating characteristic (ROC) curve (grade of sensitivity versus false positive) was used to identify the sensitivity and false positive of certain value of the variable with area under curve and probability of error to detect the impaired LV in long axis with sensitivity at 100%.

Simple linear regression (Least-square method) was used for correlation of the variables of the study: Y = b + aX, where, a = slope and b = intercept; Dependent variable = intercept + (independent variable x slope).

RESULTS

Clinical characteristics:

As regards age and gender, there was no significant difference between both groups of the study (54.38 ± 7.22 versus 47.6 ± 3.23 years, respectively, p = NS, 132 (88%) versus 40 (80%) male, p = NS and 18 (12%) versus 10 (20%) female, p = NS respectively).

There was no significant difference between both groups regarding percentage of patients with history of smoking and hypercholesterolemia [48 (32%) versus 21 (42%) patients, p = NS and 32 (21.3%) versus 12 (24%) patients, p = NS respectively]. There was no significant difference regarding the resting heart rate between both groups (89.25 ± 5.93 versus 78.5 ± 8.72 beats / minute, respectively, p = NS) but there was a significant increase in the systolic and diastolic blood pressure in the hypertensive group than the normotensive group (179.5 ± 12.41 versus 122.35 ± 8.11 mmHg, and 104.8 ± 5.32 versus 76.84 ± 6.18 mmHg, respectively, p < 0.05).

Echocardiographic analysis:

There was a significant increase in the LVMI in the hypertensive patients than the normotensive subjects but there was no significant difference as regards EF of left ventricle between both groups (157.3 ± 5.47 versus 126.53 ± 4.28 gm / m^2, p < 0.05 and 60.61 ± 4.22 versus 62.52 ± 3.22% , p = NS respectively). In group I, 70 patients had left ventricular hypertrophy while 80 patients had no left ventricular hypertrophy.

As regards M-mode of MVA there was a significant decrease in the ventricular descent phase and ventricular recoil phase but no significant difference as regards atrial phase in the hypertensive group than the normotensive group (10.2 ± 1.3 versus 12.8 ± 1.4 mm, p < 0.05, 8.8 ± 0.91 versus 10.3 ± 1.6 mm, p < 0.05 and 1.92 ± 0.54 versus 2.4 ± 0.73 mm, p = NS, respectively). Mitral inflow pulsed wave Doppler revealed that there was a significant impaired diastolic function in the hypertensive group as there was a significant decrease in the early E velocity (48 ± 3.51 versus 66.1 ± 10.2, p < 0.05), a significant increase in the late A velocity (57.2 ± 4.61 versus 34.5 ± 8.4, p < 0.05) and a significant decrease in the E / A ratio (0.84 ± 0.12 versus 1.94 ± 0.21, p < 0.05), Table 1). Ninety seven (64.7%) hypertensive patients had an impaired diastolic function as detected by pulsed wave mitral inflow velocity and 53 (35.3%) hypertensive patients had a normal diastolic function.

As regards TDI of MVA, there was a significant decrease in the E velocity (10.1 ± 2.3 versus 13.6 ± 3.4 cm / sec, p < 0.05), a non- significant increase in the A velocity (8.9 ± 1.4 versus 7.2 ± 1.6 cm / sec, p = NS), a significant decrease in the E / Aratio (1.13 ± 0.41 versus 1.89 ± 0.37, p < 0.05), a significant increase in the isovolumetric relaxation time (129.9 ± 12.5 versus 110.2 ± 6.3 msec, p < 0.05), a significant

Table 1: M-mode of mitral valve annulus and pulsed Doppler mitral valve inflow velocity variables in both groups of the study

Variable	Group I	Group II	p-Value
Ventricular descent phase (mm)	10.2 ± 1.3	12.8 ± 1.4	< 0.05
Ventricular recoil phase (mm)	8.8 ± 0.91	10.3 ± 1.6	< 0.05
Atrial phase (mm)	1.92 ± 0.54	2.4 ± 0.73	NS
Mitral inflow E-velocity (cm / sec)	48.4 ± 3.51	66.1 ± 10.2	< 0.05
Mitral inflow A-velocity (cm / sec)	57.2 ± 4.61	34.5 ± 8.4	< 0.05
E / Aratio	0.84 ± 0.12	1.94 ± 0.21	< 0.05

Table 2: Parameters of TDI of MVA in both groups

Variable	Group I	Group II	p-Value
E-mitral valve annulus (cm / sec)	10.1 ± 2.3	13.6 ± 3.4	< 0.05
A-mitral valve annulus (cm / sec)	8.9 ± 1.4	7.2 ± 1.6	NS
Em / Am ratio	1.13 ± 0.41	1.89 ± 0.37	< 0.05
IVRT (msec)	129.9 ± 12.5	110.2 ± 6.3	< 0.05
SCV -mitral valve annulus(cm / sec)	8.2 ± 1.5	10.9 ± 2.1	< 0.05
SCT-mitral valve annulus (msec)	236 ± 13.2	208 ± 21.5	NS

IVRT = isovolumetric relaxation time, SCT = systolic contraction time, SCV = systolic contraction velocity

Table 3: Correlation of tissue Doppler imaging indices of MVA in hypertensive patients as dependent variables to the LVMI as an independent variable, where, dependent variable = Y + LVMI x Slope, (n=70)

Dependent variable	y Intercept	Slope	r	p-value
E-mitral valve annulus (cm / sec)	18.376	6.792	0.822	< 0.05
A-mitral valve annulus (cm / sec)	1.082	2.831	0.839	< 0.05
E / Aratio	8.215	0.913	0.782	< 0.05
IVRT (sec)	40.241	7.082	0.925	< 0.01
SCV -mitral valve annulus(cm / sec)	13.427	0.609	0.517	NS
SCT-mitral valve annulus (cm / sec)	290.38	23.615	0.492	NS

SCT = systolic contraction time, SCV = systolic contraction velocity, r = regression coefficient

Table 4: Receiver operating characteristic (ROC) curve data of MVA-TDI in hypertensive patients to predict impaired LV function in long axis (n=80)

Variable	Sensitivity	False +ve	AUC	POE
E-mitral valve annulus (<8 cm / sec)	85%	29%	0.796	34%
A-mitral valve annulus (>8 cm / sec)	82%	24%	0.748	38%
E / Aratio (<0.8)	80%	28%	0.774	39%
IVRT (>120 ms)	73%	46%	0.691	50%
SCV -mitral valve annulus(<8 cm / sec)	84%	18%	0.837	20%
SCT-mitral valve annulus (>230 msec)	87%	20%	0.872	24%

AUC=area under curve, IVRT = isovolumetric relaxation time, POE = probability of error with sensitivity 100%, SCT = systolic contraction time, SCV = systolic contraction velocity

decrease in the systolic contraction velocity (8.2 ± 1.5 versus 10.9 ± 2.1 cm / sec, p < 0.05) and a significant increase in the systolic contraction time (236 ± 13.2 versus 208 ± 21.5 msec, p < 0.05, Table 2).

Exercise ECG test:

There was an insignificant difference between both groups as regards the peak heart rate during exercise (165.8 ± 7.66 versus 158.4 ± 9.21 beats / minute, respectively, p = NS) but there was a significant increase in the peak blood pressure and a significant decrease in the duration of the exercise test in hypertensives with impaired heart rate recovery after exercise than those with normal heart rate recovery (219.6 ± 5.33 versus 197.6 ± 7.42 mmHg, p < 0.05 and 6.84 ± 1.92 versus 8.45 ± 2.17 minutes, p < 0.05) respectively. Forty five hypertensive patients had a positive test and 105 patients had a negative stress ECG test.

Stress thallium-201 scintigraphy:

Out of 62 patients who had undergone thallium stress test, only 39 patients had a positive test with cold spot defects. Out of these 39 patients, 38 patients had an impaired TDI of the MVA and impaired M-mode motion of mitral valve ring. Out of 24 patients who had a negative test, eight patients had an impaired TDI of the MVA and impaired M-mode motion of MVA.

There was a significant positive correlation between ventricular descent phase of M-mode MVA (mm) and systolic contraction velocity of TDI of mitral annulus (cm / sec), (y =1.3x+6, r = 0.911, p < 0.05) and the systolic velocity of TDI (9 cm / sec) of dependent Y-axis corresponded ventricular contraction phase of M-mode (8mm) of independent X axis (Fig. 1). There was a significant negative correlation between ventricular recoil phase of M-mode MVA (mm) and systolic contraction time of TDI of mitral annulus (cm / sec), (y = 244 - 2.31x, r = 0.962, p < 0.05) and the systolic time of TDI (227 msec) of dependent Y axis corresponded with ventricular recoil phase of M-mode (9 mm) of independent X axis (Fig.2).

There was a significant correlation between the TDI indices of MVA in the hypertensive patients as dependent variables and the LVMI as an independent variable but IVRT and SCT-MVA had an increased correlation coefficient (r) than other dependent variables (Y-axis) of correlation (r = 0.925 & r = -0.936 , p < 0.01 & p < 0.05, respectively, Table 3).

Receiver operating characteristic (ROC) curve data of MVA- TDI in hypertensive patients revealed that systolic contraction velocity < 8 cm / sec was an indicator for detection of the impaired LV shortening fiber lengthening during systole and the systolic contraction time > 230 msec was an indicator for detection of the impaired LV

Table 5: Stepwise logistic analysis of hypertensive patients versus those without impaired TDI of MVA as regards age, gender, smoking, left ventricular hypertrophy, BP load and hypercholesterolemia

Independent Variable	r	SE	p value	95% CI
Age	0.1922	0.0978	< 0.05	1.054 ---- 1.731
Gender	0.0258	0.0772	NS	0.923 ---- 1.176
Smoking status	0.0632	0.0649	NS	0.976 ---- 1.023
LVMI	0.1732	0.0786	< 0.05	0.761 ---- 0.892
BPLoad Status	0.1984	0.0641	< 0.05	1.761 ---- 2.892
Hpercholesterol	0.0678	0.0378	NS	0.654 ---- 1.531

No. of observations =150, BP=blood pressure, CI = confidence interval, LVMI = left ventricular mass index, r = regression coefficient, SE = standard error

Table 6: Stepwise logistic analysis of hypertensive patients versus those without impaired TDI of MVA as regards antihypertensive drugs

Independent Variable	r	SE	p value	95% CI
Beta Blockors	0.1823	0.0871	< 0.05	1.214 ---- 1.936
ACE inhibitors	0.0356	0.0789	NS	0.721 ---- 1.270
CCB	0.0439	0.0657	NS	0.572 ---- 1.422
AR antagonists	0.0241	0.0174	NS	0.621 ---- 1.096
Nitrates	0.0973	0.0364	NS	0.751 ---- 1.832

No. of observations = 150, ACE = angiotensin converting enzyme, AR = angiotensin receptors, CCB = calcium channel blockers, CI = confidence interval, LVMI = left ventricular mass index, r = regression coefficient, SE = standard error

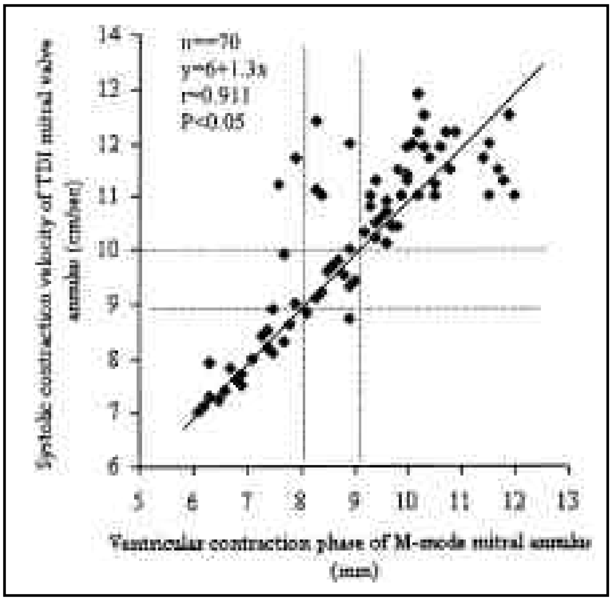

Fig. 1: Correlation between ventricular phase of M-mode mitral annulus and systolic contraction velocity of TDI mitral annulus

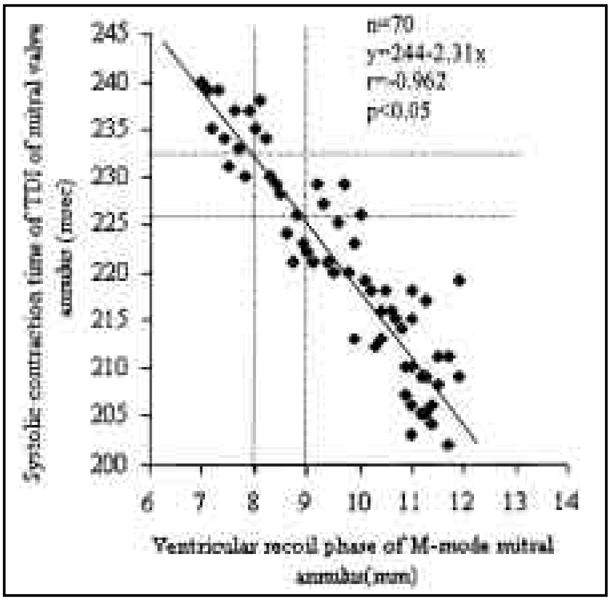

Fig. 2: Correlation between ventricular recoil phase of M-mode mitral valve annulus and systolic contraction time of TDI mitral valve annulus

longitudinal fiber lengthening during diastole (sensitivity = 84% Vs 87%, false +ve = 18% Vs 20%, area under curve=0.837 Vs 0.872 and probability of error = 20% Vs 24%, respectively, Table 4).

Stepwise logistic multivariate analysis revealed a significant relation between the age, blood pressure load status and LVMI as independent variables and impaired TDI of MVA (r = 0.1922, 0.0984 & 0.1732, 95% CI = 1.054 - 1.731, 1.109 - 2.093 & 1.761 - 2.892 respectively, $p < 0.05$). However, there was no significant relation as regards the gender, smoking status, and hypercholesterolemia (Table 5). As regards the antihypertensive drugs there was a significant relation between beta blockers and the impaired function of longitudinal fibers of left ventricle but no significant relation between AEC inhibitors, calcium channel blockers, angiotensin receptors antagonists and nitrates and the impaired LV function in long axis (p = NS, Table 6).

As regards reproducibility, there was no significant difference in intra-observer variability (p = NS).

DISCUSSION

Although longitudinal directed fibers situated mainly in the subepicardium and subendocardium regions of the left and right ventricular free walls and the papillary muscles comprise only a small proportion of the total ventricular myocardial mass, they play a major role in the maintenance of normal ejection fraction and in determining atrioventricular interactions. So, not surprisingly, loss of longitudinal fiber function leads to characteristic disturbances. It has been suggested that the movement of the annulus is dependent on the shortening and lengthening of the longitudinally oriented myocardial fibers[9].

Longitudinal function is always reduced when ventricular cavity size is increased, in addition ejection fraction is reduced. This relation is consistent enough for long axis amplitude or its equivalent, the amplitude of atrioventricular ring motion, to be used as an index of ejection fraction. It applies not only to the left ventricle, where it can be shown to relate to prognosis but also to the right ventricle, where it provides a simple method of assessing right ventricular function. When overall long axis amplitude is low, peak shortening and lengthening rates are reduced. In restrictive left ventricular disease, long axis amplitude is low even when cavity size is normal at end diastole, although the effects of this reduction are apparent in a reduced amplitude of wall thickening and thus of shortening fraction[10].

MVA motion, which is recorded by TDI with high feasibility and reproducibility has been studied in the evaluation of the left ventricular function[5]. It has long been recognized that the left ventricular diastolic function may be abnormal in patients with left ventricular hypertrophy at a time when systolic function is preserved. This has been documented by contrast and nuclear angiography and by M-mode echocardiography. All these methods demonstrate that the velocity of early diastolic filling is reduced with or without superimposed asynchrony. These findings are mirrored in long axis function when both extent and peak velocity of early diastolic lengthening are reduced and that during atrial systole they are increased[11]. The overall amplitude of motion is however, normal. In left ventricular hypertrophy, therefore, long axis function corresponds closely to conventional views of diastolic function and in these circumstances it may be appropriate to calculate the ratio of early to late diastolic lengthening. It is also reasonable to suggest that peak diastolic lengthening rate determined by TDI may be an index of early diastolic function[12]. However, these conclusions are limited to cases without asynchrony. In addition, the most common cause of a reduction of early diastolic lengthening rate is low overall amplitude of ring motion, which is characteristic of systolic left ventricular disease. Considering peak velocities of long axis motion in isolation, disregarding overall amplitude and timing is thus likely to lead to misleading conclusions[13].

Our study revealed that both M-mode echocardiogram and TDI of MVA were markers of left ventricular longitudinal fibers shortening and lengthening with subsequent effect on the diastolic filling of LV and revealed that impaired longitudinal fibers function was due to ischemia independent of the presence or absence of left ventricular hypertrophy and this in agreement with previous studies[5,10]. Haluska *et al*,[1] from the university of Queensland, Australia, reported that the longitudinal function was the only parameter to be significantly abnormal at rest in patients without contractile reserve and the failure to increase TDI velocity significantly with stress corresponded to the EF response.

Contrary to the frequent appearance of apical movement in the apical views, caused by movement of the heart within the scan plane, the apex is normally in a fixed position. During systole, contraction of subendocardial and subepicardial fibers, which follow a helical course leads to movement of the base of the heart toward the apex[14]. The contribution of this longitudinal shortening to overall function probably varies according to the circumstances and pathology but its role appears to be substantial. This aspect of LV function has received limited attention in the past, perhaps reflecting the difficulties experienced in its measurement. However, recent studies have shown the TDI measurements of the base-apex function to be a sensitive marker of ischemia[15] and Haluska *et al*[1] reported that the results of their study suggest it is a sensitive marker of latent LV dysfunction.

Henein and Gibson reported that prolonged left ventricular long shortening and delayed onset of lengthening effectively suppress early diastolic transmitral flow even though the minor axis increases and mitral cusps separate apparently normally and this grossly asynchronous left ventricular relaxation may interfere with filling by dissipating normal ventricular restoring forces[16]. They suggested that delayed and prolonged long axis shortening is the primary disturbance, but this may have been the result of activation disturbance, subendocardial ischemia[17,18] or other causes still to be identified, either alone or in combination. This prolonged shortening interacts with rapid thinning of the posterior left ventricular wall, a process that we have already suggested to be autonomous and a major site of restoring forces[19] and the energy normally coupled to filling is thus dissipated as a change in cavity shape.

Methodological considerations:

1. M-mode echocardiogram of mitral valve is a valid and useful method to evaluate left ventricular function[20].
2. Pulsed wave TDI of MVA is an advanced method to study the systolic and diastolic LV function[3,5,6].

Limitations of the study:

1. Relatively small number of patients.
2. Echocardiogram was done by one observer, so intra-observer variability was evaluated but it is difficult to evaluate inter-observer variability.
3. Study was not completely blind to the observer.

4. Pulmonary venous flow velocity was detected only in 20 patients to estimate left ventricle end-diastolic pressure with TDI mitral annulus[14].
5. Only 31 patients were known to have undergone cardiac catheterization and coronary angiography in the course of their clinical management.
6. Isotope cardiac scanning is more accurate and valid method to evaluate diastolic function[5], but it was not easily available .

CONCLUSION

Mitral valve annular motion velocity measurements using TDI should be employed routinely during the evaluation of the hypertensive patients.TDI of mitral valve ring is a clinically useful marker for impaired LV function in long axis and an indicator of impaired relaxation of LV in hypertensive patients independent of the presence or absence of the left ventricular hypertrophy.

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