The role of isovolumic acceleration in predicting subclinical right and left ventricular systolic dysfunction in hypertensive obese patients

Hipertansif obezlerde erken dönem sağ ve sol ventrikül sistolik disfonksiyonun değerlendirilmesinde izovolümik akselerasyonun önemi

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ABSTRACT

Objectives: Isovolumic acceleration assessed by tissue Doppler imaging has been proposed as a preload-independent indicator of left ventricular contractility. We investigated the utility of isovolumic acceleration in the prediction of preclinical right and left ventricular systolic dysfunction in hypertensive and obese subjects.

Study design: Seventy-eight obese subjects (BMI >30 kg/m^2; 57 women, 21 men; mean age 51±8 years) were prospectively enrolled. Fifty patients (64.1%) had hypertension and 33 patients (42.3%) had diabetes mellitus. All the subjects were assessed by conventional and tissue Doppler echocardiography. Myocardial velocities of the left ventricular septal and lateral mitral annulus and lateral tricuspid annulus were determined. Isovolumic contraction wave was defined as the preceding wave of the systolic wave that began before the peak of the R wave on the electrocardiogram. Myocardial isovolumic acceleration was measured by dividing the peak velocity by the time passed from the onset of the wave (zero-crossing) during isovolumic contraction to the peak velocity of the wave.

Results: Waist circumference was in positive correlation with left ventricular end-systolic (r=0.22, p=0.047) and end-diastolic (r=0.384, p=0.001) diameters, and in negative correlation with the peak systolic velocity of the tricuspid annulus (r=-0.311, p=0.006). Although hypertensive and normotensive (n=28) obese subjects had similar myocardial velocities, lateral tricuspid annular isovolumic acceleration (p=0.027), septal isovolumic acceleration (p=0.026), and septal isovolumic contraction myocardial velocity (p=0.018) were significantly lower in hypertensive patients.

Conclusion: Isovolumic acceleration and isovolumic contraction myocardial velocity analysis may be useful in the diagnosis of subclinical left and right ventricular dysfunction in hypertensive obese patients.

ÖZET

Amaç: Doku Doppler görüntüleme ile değerlendirilen izovolümik akselerasyon, sol ventrikül kasılmasının önyükten bağımsız bir göstergesi olarak önerilmektedir. Çalışmamızda, hipertansif obez kişilerde sağ ve sol ventrikül sistolik disfonksiyonun erken dönemde öngörülmesinde izovolümik akselerasyonun kullanımı araştırıldı.

Çalışma planı: Yetmiş sekiz obez kişi (beden kütle indeksi >30 kg/m^2; 57 kadın, 21 erkek; ort. yaş 51±8) ileriye dönük olarak çalışmaya alındı. Elli hastada (%64.1) hipertansiyon, 33 hastada (%42.3) diabetes mellitus vardı. Tüm katılımcılar konvansiyonel ve doku Doppler ekokardiyografi ile değerlendirildi. Sol ventrikül septal ve lateral halka ile lateral triküspit halkaya ait miyokart hızları ölçüldü. İzovolümik kasılma, elektrokardiyografide R dalgası zirvesinden önce başlayan ve sistolik dalganın öncesinde gelen dalga olarak tanımlandı. Miyokart izovolümik akselerasyonu, zirve hızın, izovolümik kasılma sırasında görülen dalganın başlangıcından (sıfır noktasından) bu dalganın zirve hıza ulaşmasına kadar geçen süreye bölünmesiyle hesaplandı.

Bulgular: Bel çevresi, sol ventrikül sistol sonu (r=0.22, p=0.047) ve diyastol sonu (r=0.384, p=0.001) çapları ile pozitif ilişki, triküspit halka zirve sistolik hızı ile negatif ilişki (r=-0.311, p=0.006) gösterdi. Hipertansif ve normotansif (n=28) obez kişilerde doku Doppler ile ölçülen miyokart hızları benzer bulunmasına karşın, hipertansif hastalarda triküspit lateral halka izovolümik akselerasyonu (p=0.027), septal izovolümik akselerasyon (p=0.026) ve septal izovolümik miyokart kasılma hızı (p=0.018) anlamlı derecede düşük bulundu.

Sonuç: İzovolümik akselerasyon ve izovolümik miyokart kasılma hızı analizi, hipertansif obez bireylerde subklinik düzeydeki sol ve sağ ventrikül disfonksiyonu tanısında yararlı olabilir.

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Obesity is the emerging public health problem in developed countries. It is very common and has significant morbidity and mortality.[1,2] The association between body weight and blood pressure is well-established.[3,4] Elevated intracardiac pressure and left ventricular hypertrophy impair left ventricular diastolic filling and eventually cause heart failure in hypertensive patients.[5-9] Obesity is also closely associated with right- and left-sided heart failure.[10-17] Myocardial acceleration during isovolumic contraction and isovolumic contraction myocardial velocity are two echocardiographic parameters that predict ventricular functions independent of preload.[18,19] These two parameters seem to reflect myocardial contractility with a better sensitivity than TDI-derived peak systolic velocities. We investigated the utility of IVA, IVV, and myocardial systolic and diastolic velocity measurements on the assessment of left and right ventricular functions among normotensive and hypertensive obese individuals.

Abbreviations:

IVA *Myocardial isovolumic acceleration*
IVV *Isovolumic contraction myocardial velocity*
LV *Left ventricular*
TDI *Tissue Doppler imaging*

PATIENTS AND METHODS

Seventy-eight obese subjects (body mass index >30 kg/m^2) were enrolled prospectively in this study. There were 57 women and 21 men with a mean age of 51±8 years. The patients were selected randomly from the population of internal medicine outpatient clinic in a consecutive manner and gave written consent to participate in the study. Exclusion criteria included the presence of the following: left ventricular ejection fraction <55%, history of coronary artery disease, evidence for ischemic heart disease on the electrocardiogram, echocardiography, or cardiac stress test; chronic obstructive lung disease, organic valvular heart disease, chronic renal failure, and poor echocardiographic image quality. All patients were in sinus rhythm. The local ethics committee approved this cross-sectional study.

Echocardiography was performed in all patients in the left lateral decubitus position with standard views using a Vivid 5 machine (GE Vingmed, Horten, Norway). Left atrial systolic dimension and LV internal dimensions and wall thicknesses were measured from two-dimensional guided M-mode tracings obtained at the midchordal level in the parasternal long-axis view according to the criteria of the American So-

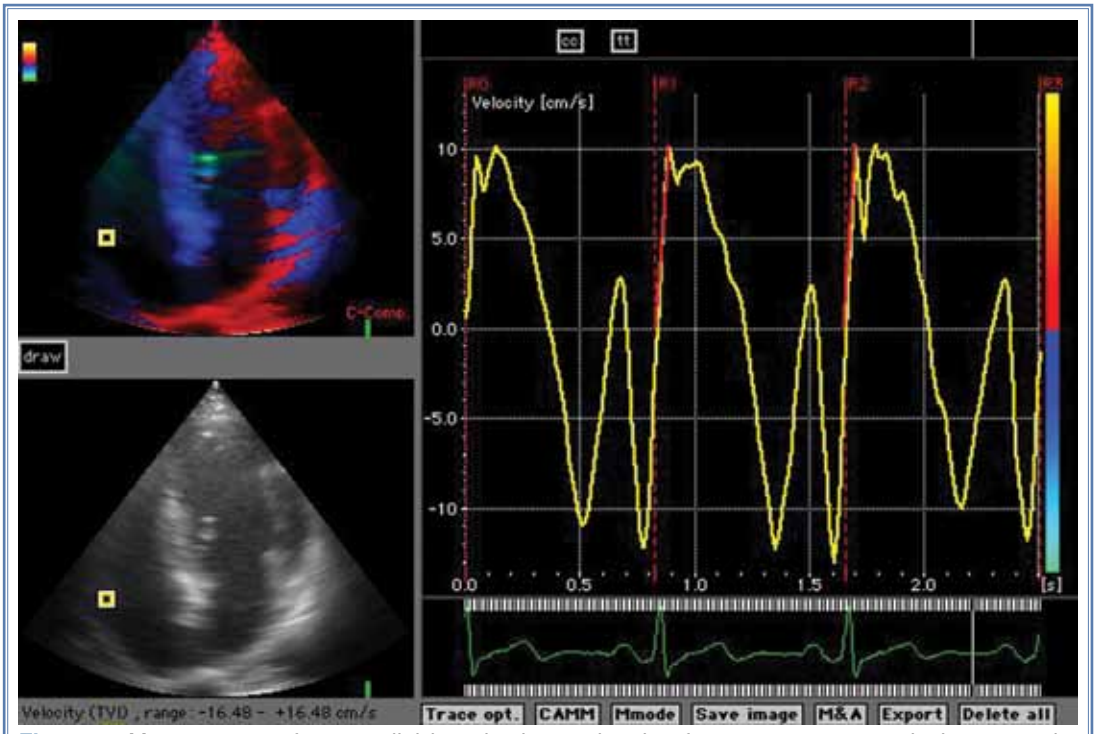

Figure 1. Measurement of myocardial isovolumic acceleration from two separate velocity curves by dividing the peak velocity by the time interval from the onset of the wave (zero-crossing) during isovolumic contraction to the peak velocity of the wave.

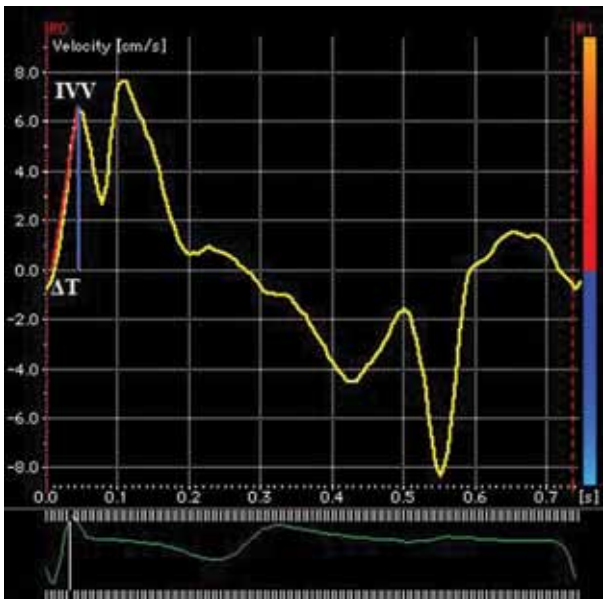

Figure 2. Isovolumic acceleration (IVA) is calculated by dividing the peak isovolumic velocity (IVV) by the delta time (ΔT).

ciety of Echocardiography.[20] Left ventricular mass was calculated using the method of Devereux et al.[21] and normalized to height in meters. Percent fractional shortening and ejection fraction were calculated using the Teichholz formula.[22] Mitral inflow velocities were obtained by pulsed-wave Doppler in the apical 4-chamber view with the sample volume placed at the tips of the mitral valve leaflets. The peak early (E) and late (A) diastolic mitral inflow velocities, deceleration time, E/A ratio, and isovolumic relaxation time were measured and averaged over three cardiac cycles according to the recommendations of the American Society of Echocardiography.[23] Color tissue Doppler imaging was performed from the apical 4-chamber view using a 2.5-MHz transducer and frame rates of >80/sec and the images were digitized. Derivation and analysis of TDI-derived velocity profiles were performed offline using a commercial computer software (Echopac, GE Vingmed). Myocardial velocity profiles of the basal septal and lateral mitral annulus were obtained by placing a 6-mm sample volume at the junction of the mitral annulus with the septum and lateral myocardial wall. Myocardial velocities of the lateral tricuspid annulus were obtained similarly by placing the sample volume at the junction of the tricuspid valve annulus and right ventricular free wall.

Isovolumic contraction wave was determined as the preceding wave of the systolic wave that began before the peak of the R wave on the electrocardiogram. Myocardial isovolumic acceleration was measured by dividing the peak velocity by the time interval from the onset of the wave (zero-crossing) during isovolumic contraction to the peak velocity of this wave as previously described (Fig 1, 2).[19] The ratio of peak early diastolic mitral inflow velocity by pulsed-wave Doppler to peak early diastolic mitral annular velocity by TDI was calculated as a measure of LV filling pressure. Peak tricuspid annular systolic, early diastolic, and late diastolic velocities were also measured from three consecutive cardiac cycles and averaged.

Descriptive statistics were shown as mean ± standard deviation. Parameters with and without normal distribution were compared with the Student's t-test and Mann-Whitney U-test, respectively. Categorical variables were compared with the Fisher's exact test (chi-square). The Pearson's correlation coefficient was used to assess the association between anthropometric and echocardiographic data. A p value <0.05 was accepted as significant for all statistics.

RESULTS

Demographic, clinical, and echocardiographic characteristics of the study population are shown in Table 1. Fifty patients (64.1%) had a history of hypertension and 33 patients (42.3%) had diabetes mellitus. Of the diabetic patients, 23 were hypertensive, and 10 were normotensive. There were no significant correlations between body mass index and echocardiographic parameters. However, waist circumference was in positive correlation with left ventricular end-systolic (r=0.22, p=0.047) and end-diastolic (r=0.384, p=0.001) diameters, and in negative correlation with TDI-derived peak systolic velocity of the tricuspid annulus (r=-0.311, p=0.006).

The patients were divided into two groups based on the presence (n=50) or absence (n=28) of hypertension (Table 1). Compared to normotensive obese subjects, hypertensive patients exhibited significantly lower values of right ventricular IVA (p=0.027), septal IVA (p=0.026), and septal IVV (p=0.018). The remaining echocardiographic characteristics were similar in the two groups.

DISCUSSION

In our study, we did not find any significant difference in the TDI-derived myocardial systolic velocities between hypertensive and normotensive individuals. However, right and left ventricular IVA values were

Table 1. Demographic, clinical, and echocardiographic characteristics of the obese subjects

	Total (n=78)			Hypertensives (n=50)			Normotensives (n=28)			
	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (years)			51±8			50±9			51±8	0.875
Gender										0.065
Female	57	73.1		40	80.0		17	60.7		
Male	21	26.9		10	20.0		11	39.3		
Hypertension	50	64.1								
Diabetes	33	42.3		23	46.0		10	35.7		0.378
Body mass index (kg/m^2)			34±5			34.5±5			33.8±4	0.524
Waist (cm)			109±9			110±10			108±9	0.291
Basal heart rate			86±16			91±14			89±16	0.544
Left atrium (cm)			3.5±0.4			3.4±0.4			3.5±0.4	0.676
Left ventricle										
End-diastolic diameter (cm)			5.0±0.5			5.0±0.5			4.9±0.5	0.395
End-systolic diameter (cm)			3.1±0.5			3.0±0.5			3.0±0.4	0.817
Ejection fraction (%)			70±7			70±7			70±6	0.972
Mass (g/m^2)			149±48			150±45			147±53	0.789
Interventricular septum (cm)			1.3±0.2			1.3±0.2			1.3±0.2	0.594
Posterior wall (cm)			1.1±0.2			1.1±0.2			1.1±0.2	0.830
Mitral E velocity (m/sec)			0.7±0.1			0.7±0.1			0.7±0.1	0.811
Mitral A velocity (m/sec)			0.8±0.2			0.8±0.2			0.8±0.2	0.564
E/A			0.9±0.3			0.9±0.3			0.9±0.7	0.704
E-wave deceleration time (msec)			282±92			282±88			283±101	0.980
Isovolumic relaxation time (msec)			118±25			118±25			116±27	0.727
Right ventricle										
Systolic velocity (cm/sec)			10.7±1.9			10.6±2.2			10.8±1.5	0.629
Isovolumic acceleration (cm/sec^2)			210±80			196±74			237±84	**0.027**
Isovolumic contraction velocity (cm/sec)			8.4±2.4			8.2±2.4			8.7±2.4	0.361
Septum										
Septal annular systolic velocity (cm/sec)			6.1±1.1			5.9±1.0			6.3±1.0	0.158
Septal annular early diastolic velocity (cm/sec)			5.1±1.6			4.9±1.4			5.4±2	0.191
Septal annular late diastolic velocity (cm/sec)			7.9±1.6			7.8±1.6			8.2±1.6	0.260
Isovolumic acceleration (cm/sec^2)			143±53			133±56			161±43	**0.026**
Isovolumic contraction velocity (cm/sec)			3.4±1.7			3.6±1.7			4.6±1.4	**0.018**
Tricuspid lateral annulus										
Systolic velocity (cm/sec)			6.9±1.7			6.9±1.8			7.0±1.7	0.656
Early diastolic velocity (cm/sec)			7.1±2.7			6.8±2.6			7.2±2.1	0.190
Late diastolic velocity (cm/sec)			8.6±2.0			8.6±1.8			8.5±2.3	0.874
E/E'			15.2±7.0			15.2±5.0			15±10	0.916

significantly lower in the hypertensive group. This finding suggests that basal septal and tricuspid annular IVA measurements may be superior to conventional techniques for the diagnosis of preclinical myocardial systolic dysfunction.

Segmental LV shortening that occurs before the onset of LV ejection represents an active contraction; it not only results in changes in the global LV geometry by mobilizing blood and allowing the protrusion of the mitral leaflets into the left atrium during systole, but also contributes to the isovolumic contraction velocities measured by TDI.[24,25] Peak endocardial acceleration is caused by vibrations that are generated and transmitted throughout the heart by the isometric contraction of the myocardium during isovolumic contraction.[26] Vogel et al.[18,19] measured tricuspid and mitral ring velocities by TDI and observed a relationship between IVA and the global right ventricular and LV contractility. Lyseggen, et al.[25] also found a correlation between IVA and LV dP/dt_{max} during incremental dobutamine infusion and during mild reductions in preload, and the responses were similar for measurements taken near the mitral ring (basal segments) and more distally in the LV wall.

Although TDI-derived systolic myocardial velocities are accepted as reliable, sensitive, and relatively load-independent parameters for the detection of preclinical LV systolic dysfunction,[27] measurements of IVA and IVV may better predict myocardial contractility. Considering that TDI-derived parameters were similar for both right and left ventricles of hypertensive and normotensive obese subjects, we hypothesize that IVA analysis may provide further information on myocardial contractility, ventricular function, and preclinical systolic dysfunction in obese individuals.

Obesity is an independent risk factor for the development of hypertension.[4,28,29] The prevalence of hypertension in overweight or obese individuals is as high as 50%, and this is proportional to the severity of obesity.[28] Hypertension is one of the most common causes of heart failure. Diastolic dysfunction is common among hypertensive individuals due to left ventricular hypertrophy and increased intracardiac pressures. The E/E' ratio obtained from TDI is a reliable parameter in predicting elevated left ventricular filling pressures.[6,9,30,31] However, in our study, the conventional and TDI-derived echocardiography parameters indicating diastolic dysfunction were similar in hypertensive and normotensive obese subjects. On the other hand, obesity itself is associated with impaired diastolic function independent of the presence of hypertension.[12,13]

Studies on right ventricular functions in obese patients reported conflicting results. Otto et al.[15] found that right ventricular relaxation and filling were impaired in obesity. However, this study did not find a significant difference in the TDI-derived tricuspid annular peak systolic velocity between obese and non-obese groups. On the other hand, Wong et al.[17] demonstrated that increased body mass index was associated with right ventricular dysfunction and this finding was independent from sleep apnea. Willens et al.[16] reported improvement in right ventricular dysfunction following weight loss. We did not find any significant difference in the TDI-derived peak systolic velocities of the tricuspid lateral annulus between hypertensive and normotensive subjects. However, we found that right ventricular IVA was significantly lower in hypertensive patients. As right ventricular dysfunction is a major determinant of impaired functional capacity in obese individuals, early diagnosis of right ventricular dysfunction by IVA analysis may be useful in the management of these patients.

Limitations

Our study population was not homogenous and had a disproportionate number of female subjects and diabetics. In addition, the patients were taking antihypertensive medications, and these medications were not stopped before the study. These drugs may have effects on echocardiographic measurements. Finally, we did not exclude patients with obstructive sleep apnea. However, Wong et al.[17] demonstrated that subclinical right ventricular dysfunction was independent from obstructive sleep apnea, diabetes, hypertension, and other comorbidities in obesity.

In conclusion, IVA and IVV analysis may be useful in the diagnosis of subclinical left and right ventricular dysfunction in hypertensive and obese subjects.

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REFERENCES

1. Noppa H, Bengtsson C, Wedel H, Wilhelmsen L. Obesity in relation to morbidity and mortality from cardiovascular disease. Am J Epidemiol 1980;111:682-92.
2. Allison DB, Fontaine KR, Manson JE, Stevens J, van Itallie TB. Annual deaths attributable to obesity in the United States. JAMA 1999;282:1530-8.

3. Dustan HP. Mechanisms of hypertension associated with obesity. Ann Intern Med 1983;98:860-4.
4. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. JAMA 1978;240:1607-10.
5. Inouye I, Massie B, Loge D, Topic N, Silverstein D, Simpson P, et al. Abnormal left ventricular filling: an early finding in mild to moderate systemic hypertension. Am J Cardiol 1984;53:120-6.
6. Fouad FM, Slominski JM, Tarazi RC. Left ventricular diastolic function in hypertension: relation to left ventricular mass and systolic function. J Am Coll Cardiol 1984; 3:1500-6.
7. Hartford M, Wikstrand J, Wallentin I, Ljungman S, Wilhelmsen L, Berglund G. Diastolic function of the heart in untreated primary hypertension. Hypertension 1984; 6:329-38.
8. Senni M, Tribouilloy CM, Rodeheffer RJ, Jacobsen SJ, Evans JM, Bailey KR, et al. Congestive heart failure in the community: trends in incidence and survival in a 10-year period. Arch Intern Med 1999;159:29-34.
9. Vasan RS, Benjamin EJ, Levy D. Prevalence, clinical features and prognosis of diastolic heart failure: an epidemiologic perspective. J Am Coll Cardiol 1995;26:1565-74.
10. Kenchaiah S, Evans JC, Levy D, Wilson PW, Benjamin EJ, Larson MG, et al. Obesity and the risk of heart failure. N Engl J Med 2002;347:305-13.
11. Maniscalco M, Arciello A, Zedda A, Faraone S, Verde R, Giardiello C, et al. Right ventricular performance in severe obesity. Effect of weight loss. Eur J Clin Invest 2007;37: 270-5.
12. Peterson LR, Waggoner AD, Schechtman KB, Meyer T, Gropler RJ, Barzilai B, et al. Alterations in left ventricular structure and function in young healthy obese women: assessment by echocardiography and tissue Doppler imaging. J Am Coll Cardiol 2004;43:1399-404.
13. Sharpe JA, Naylor LH, Jones TW, Davis EA, O'Driscoll G, Ramsay JM, et al. Impact of obesity on diastolic function in subjects < or = 16 years of age. Am J Cardiol 2006;98:691-3.
14. Willens HJ, Chakko SC, Lowery MH, Byers P, Labrador E, Gallagher A, et al. Tissue Doppler imaging of the right and left ventricle in severe obesity (body mass index >35 kg/m2). Am J Cardiol 2004;94:1087-90.
15. Otto ME, Belohlavek M, Khandheria B, Gilman G, Svatikova A, Somers V. Comparison of right and left ventricular function in obese and nonobese men. Am J Cardiol 2004;93:1569-72.
16. Willens HJ, Chakko SC, Byers P, Chirinos JA, Labrador E, Castrillon JC, et al. Effects of weight loss after gastric bypass on right and left ventricular function assessed by tissue Doppler imaging. Am J Cardiol 2005;95:1521-4.
17. Wong CY, O'Moore-Sullivan T, Leano R, Hukins C, Jenkins C, Marwick TH. Association of subclinical right ventricular dysfunction with obesity. J Am Coll Cardiol 2006;47:611-6.
18. Vogel M, Cheung MM, Li J, Kristiansen SB, Schmidt MR, White PA, et al. Noninvasive assessment of left ventricular force-frequency relationships using tissue Doppler-derived isovolumic acceleration: validation in an animal model. Circulation 2003;107:1647-52.
19. Vogel M, Schmidt MR, Kristiansen SB, Cheung M, White PA, Sorensen K, et al. Validation of myocardial acceleration during isovolumic contraction as a novel noninvasive index of right ventricular contractility: comparison with ventricular pressure-volume relations in an animal model. Circulation 2002;105:1693-9.
20. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989;2:358-67.
21. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450-8.
22. Teichholz LE, Kreulen T, Herman MV, Gorlin R. Problems in echocardiographic volume determinations: echocardiographic-angiographic correlations in the presence of absence of asynergy. Am J Cardiol 1976;37:7-11.
23. Quiñones MA, Otto CM, Stoddard M, Waggoner A, Zoghbi WA. Recommendations for quantification of Doppler echocardiography: a report from the Doppler Quantification Task Force of the Nomenclature and Standards Committee of the American Society of Echocardiography. J Am Soc Echocardiogr 2002;15:167-84.
24. Rankin JS, McHale PA, Arentzen CE, Ling D, Greenfield JC Jr, Anderson RW. The three-dimensional dynamic geometry of the left ventricle in the conscious dog. Circ Res 1976;39:304-13.
25. Lyseggen E, Rabben SI, Skulstad H, Urheim S, Risoe C, Smiseth OA. Myocardial acceleration during isovolumic contraction: relationship to contractility. Circulation 2005;111:1362-9.
26. Rickards AF, Bombardini T, Corbucci G, Plicchi G. An implantable intracardiac accelerometer for monitoring myocardial contractility. The Multicenter PEA Study Group. Pacing Clin Electrophysiol 1996;19:2066-71.
27. Fang ZY, Yuda S, Anderson V, Short L, Case C, Marwick TH. Echocardiographic detection of early diabetic myocardial disease. J Am Coll Cardiol 2003;41:611-7.
28. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282:1523-9.
29. Kannel WB, Brand N, Skinner JJ Jr, Dawber TR, McNamara PM. The relation of adiposity to blood pressure and development of hypertension. The Framingham study. Ann Intern Med 1967;67:48-59.
30. Ommen SR, Nishimura RA, Appleton CP, Miller FA,

Oh JK, Redfield MM, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation 2000;102:1788-94.

31. Ersoylu ZD, Tuğcu A, Yıldırımtürk O, Aytekin V, Aytekin S. Comparison of the incidences of left ventricular hypertrophy, left ventricular diastolic dysfunction, and arrhythmia between patients with dipper and non-dipper hypertension. [Article in Turkish] Türk Kardiyol Dern Arş 2008;36:310-7.

Key words: Blood flow velocity; body mass index; echocardiography, Doppler/methods; myocardial contraction/physiology; obesity/complications; ventricular function, left; ventricular function, right.

Anahtar sözcükler: Kan akım hızı; beden kütle indeksi; ekokardiyografi, Doppler/yöntem; miyokart kasılması/fizyoloji; obezite/komplikasyon; ventrikül fonksiyonu, sol; ventrikül fonksiyonu, sağ.