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Left Ventricular Hypertrophy in Obese Hypertensives: Is It Really Eccentric? (An Echocardiographic Study)

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A B S T R A C T

In order to study left ventricular hypertrophy patterns in obese hypertensives, we examined 132 patients with essential hypertension by 2D, M-mode and Doppler echocardiography. The patients were classified in four comparable groups, corresponding to the values of Quetelet's body mass index (BMI) and grades of obesity. More obese hypertensives had on average larger left ventricles with thicker walls and larger left atria than less obese, or lean ones. Left ventricular mass increased significantly and progressively with advancing grades of obesity, but relative wall thickness (wall thickness/cavity size ratio) did not diminish.

Doppler echocardiography revealed significantly higher prevalence of left ventricular diastolic dysfunction among obese than among lean hypertensives.

In the second part of our study, we analyzed the subgroups defined by the severity of hypertension and the age of the patients. The correlation of the indices of left ventricular and left atrial hypertrophy with the BMI values was considerably better in the group of moderate than in the group of mild hypertension. The r values were 0.62 vs. 0.22 for left ventricular mass and 0.64 vs. 0.26 for left atrial dimension. The group of patients with severe hypertension was characterized by left ventricular cavity enlargement in correlation with increasing BMI values, but without corresponding left ventricular wall thickening. So called left ventricular »eccentricity index«, as the reverse value of relative wall thickness, correlated well (r = 0.76) with the BMI values. The indices of left ventricular hypertrophy correlated with the BMI values slightly better in middle age groups than in the groups of the youngest (≤ 30 years) or the eldest (≥ 61 years) hypertensives.

In conclusion, eccentric left ventricular hypertrophy does not seem to be a distinctive feature of hypertensive heart disease in obesity. There is only some tendency toward the »eccentricity« of left ventricular geometry which becomes more apparent in more severe forms of hypertension, especially in very obese persons.

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Introduction

It has been stated that the obese hypertensives are prone to the eccentric hypertrophy of the left ventricle[1–5]. This is in contrast to the lean hypertensives who develop concentric left ventricular hypertrophy as the most typical feature of hypertensive heart disease[6–8]. Eccentric hypertrophy may be defined by an increase in left ventricular mass due mainly to the cavity enlargement without pronounced thickening of the walls. At most, they are thickened proportionally to the increase in cavity size[8,9]. On the contrary, concentric hypertrophy is characterized by left ventricular wall thickening without increase in cavity size[8,9].

The assumption that eccentric left ventricular hypertrophy is typical for obese hypertensives is supported by the observations that marked obesity induces eccentric left ventricular hypertrophy even in the absence of arterial hypertension. This can be explained by increases in intravascular blood volume, cardiac output and stroke volume, necessary to meet metabolic demands of increased mass of adipose tissue[10,11].

These peculiarities of left ventricular geometry and haemodynamics in obese hypertensives are relevant for the choice of antihypertensive drugs[2]. The question arises: »Is the prevalence of eccentric hypertrophy among obese hypertensives distinctive enough to warrant specific therapeutic approach?« Evidence on this point is conflicting. We tried to throw little bit more light upon the question relevant for clinical practice: »Do obese hypertensives with left ventricular hypertrophy really have eccentric, instead of concentric hypertrophy?«

Patients and Methods

We examined 132 hypertensives by two-dimensional, M-mode and Doppler echocardiography. Among them were 71 males aged 53.11 12.55 years (mean SD) and 61 females aged 48.18 14.63 years. Corresponding values for the whole group were 50.83 13.2 years. Almost all age groups were included. The youngest patient was 17 and the eldest 90 years old, median value was 50 years. All the patients were supposed to have had essential hypertension on the basis of generally accepted clinical and diagnostic standards[12]. The patients with coronary and valvular heart disease were excluded from the study, as were the patients with atrial fibrillation. Only three patients had the signs of overt heart failure.

The severity of hypertension was assessed according to the criteria of Julius and Marinković[13,14]. It was classified as mild, moderate, or severe hypertension.

The values of Quetelet's body mass index (BMI) of weight/height^2, expressed in kg/m^2 and relative body mass (RBM) expressed in the percentages of ideal weight (»weight goal«), were estimated for each patient[15]. The patients were classified as: lean hypertensives (BMI 24.9 kg/m^2, RBM 110%), mildly obese hypertensives (BMI 25–27 kg/m^2, RBM 111–120%), moderately obese hypertensives (BMI 27.1–31.5 kg/m^2, RBM 121–140%) and extremely obese hypertensives (BMI > 31.5 kg/m^2, RBM > 140%). Ideal body weight (RBM 100%) corresponded to the BMI value of 22.7 kg/m^2 for males and 26.9 kg/m^2 for females. The RBM value for 40% overweight (RBM 140%) corresponded to the BMI values of 31.8 and 31.4 kg/m^2 for males and females respectively[15].

All the groups were mutually comparable in respect to the age and the severity of hypertension: the analysis of variance did not reveal any significant difference between the groups. The proportion of women in the group of markedly obese hypertensives was significantly higher than in the groups of lean and mildly

obese hypertensives (^{2} test). However, there was no significant difference between the lean hypertensives and obese hypertensives altogether, so that the groups were also comparable in respect to sex distribution.

The patients were not selected. They were included in the study as they were examined in our outpatient's department, providing that they had essential hypertension and no other cardiac disease, excepting hypertensive heart disease. Relatively high proportion of obese patients probably reflected the nutritional status of the hypertensives in our local population. The evaluation of the patients and diagnostic procedures were performed in our outpatient's department. The only exceptions were a few patients with severe hypertension who were admitted to our hospital department because of intensive treatment and complex diagnostic procedures.

Echocardiographic examinations were performed by Diasonics CV 400 echocardiographic equipment. Heart dimensions were measured in at least three consecutive cardiac cycles, according to the recommendations of European[16] and American[17] echocardiographic societies. The values of left ventricular internal diastolic dimension (LVIDd), diastolic left ventricular posterior wall thickness (PWTh), diastolic interventricular septum thickness (IVSTh), left ventricular mass, LVIDd/IVSTh + PWTh ratio and left atrial dimensions were analyzed in respect to the grades of obesity and hypertension. The age of the patient was taken into the account. Left ventricular mass was calculated from M-mode left ventricular dimensions, using Penn convention equation1[8–21].

The ratio LVIDd/PWTh + IVSTh could be called »the index of left ventricular eccentricity« and it actually represents the reciprocal value of the relative left ventricular wall thickness[22,23].

Systolic left ventricular function was represented by ejection fraction. It was preferred because of its widespread clinical use, although some other echocardiographic indices might be more correct from the aspect of methodology[24]. Left ventricular ejection fraction was calculated from left ventricular diastolic (LVIDd) and systolic (LVIDs) dimensions, using Teicholz's equation[25], but it was controlled by two-dimensional methods: Simpson's and Dodge's (»area-length«)[24,36–31]. The overlapping of M-mode and two-dimensional left ventricular ejection fractions' values was satisfactory, so that repeated estimation and correction were rarely needed.

Some aspects of left ventricular diastolic function and filling were studied by comparing transmitral peak flow velocities in early (PFVE) and late (PFVA) diastole[24,32–34]. Pulsed Doppler sample volume was placed in the mitral valve orifice, at the level of mitral valve ring. Diastolic left ventricular dysfunction was suspected if PFVE/PFVA ratio was more than 1.5, or if its reciprocal value PFVE/PFVA was less than 0.66. Markedly decreased early and mid-diastolic deceleration rate (represented by the slope following PFVE) was considered as an additional clue for the presence of left ventricular diastolic dysfunction, useful in dubious cases. Similar meaning was ascribed to the very slow increase in M-mode left ventricular dimension in early diastole, together with its marked increase after atrial contraction in late diastole[24].

In 98 patients with satisfactory suprasternal approach, cardiac output was estimated by Doppler method, using 2.25 MHz continuous wave nonimageing transducer (so called Pedoff probe). The equation used for the calculation of cardiac output was: SV HR = FVI CSA HR, where SV is stroke volume, HR heart rate, FVI flow velocity integral in ascending aorta from the suprasternal approach

and CSA cross sectional area at the level of aortic valve ring (annulus). Cross sectional area was calculated from the standard circle area equation r^2p, where r is the half value of aortic annulus diameter, measured from the two-dimensional parasternal long axis approach[24,35].

The statistical significance of differences between the groups was tested by variance analysis with Newman Keul's comparisons. The only exception was the analysis of left ventricular diastolic dysfunction, where c^2 test was used instead. Linear correlations were expressed as Pearson's correlation coefficients (r). Computer program »Quickstatt« was used for statistical analysis.

Results

Very small increments of LVIDd were noted, along with increasing grades of obesity, represented by BMI values (Figure 1). The differences were small and statistically insignificant, but the trend was steady. On the average, more obese hypertensives had slightly larger left ventricles than their less obese, or lean counterparts.

Obese hypertensives had significantly thicker left ventricular walls, represented by the IVSTh and PWTh values, than lean hypertensives (Figure 2). However, no differences between the groups with various degrees of obesity were noted.

The increase in left ventricular mass among the obese hypertensives, in respect to their less obese counterparts was highly significant (Figure 3). However, the differences between the subgroups of obese hypertensives were small. Extremely obese hypertensives had slightly higher left ventricular mass values than their less obese counterparts.

The ratio LVIDd/PWTh + IVSTh (»left ventricular eccentricity index«) was slightly

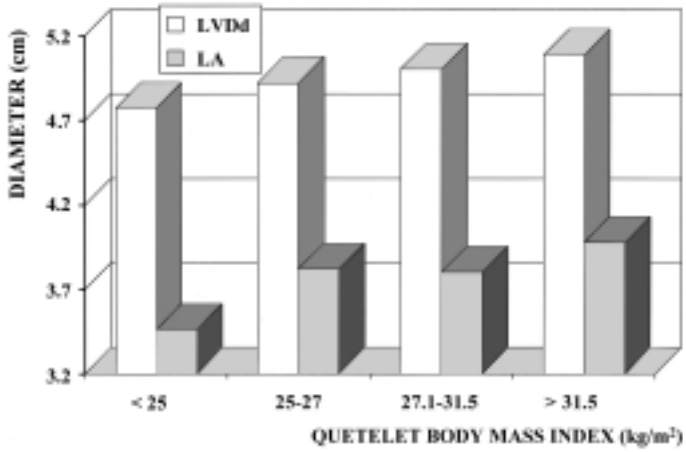

Fig. 1. Left ventricular (LVIDd) and left atrial (LA) diameter in the groups of hypertensives defined by the BMI values, from lean and mildly obese to moderately and extremely obese hypertensives. The group of lean hypertensives comprised 30 patients with LVIDd 4.78±0.55 cm (mean±SD) and LA 3.46±0.55 cm The respective values were 4.94±0.61 cm and 3.82±0.50 cm for 25 mildly obese, 5.02±0.51 cm and 3.81±0.67 cm for 42 moderately obese, as well as 5.09 ± 0.54 cm and 3.40±0.51 cm for 35 extremely obese hypertensives. The differences between the LVIDd values were not significant, but the level of statistical significance was reached for LA values ($p < 0.05$, analysis of variance).

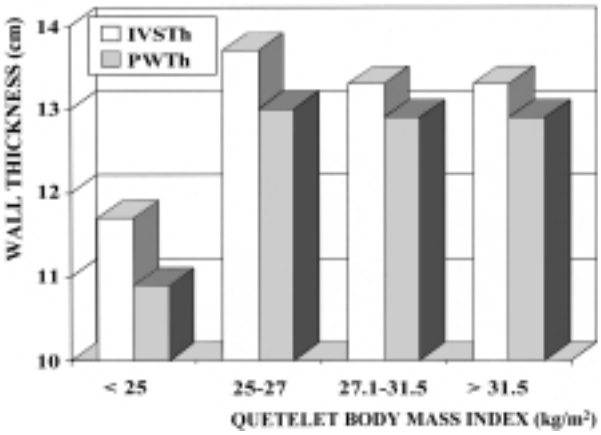

Fig. 2. Interventricular septum (IVSTh) and posterior left ventricular wall thickness (PWTh) in the groups of lean (11.77±2.22 mm vs. 10.9±2.54 mm), mildly obese (13.79±3.64 mm vs. 13.068±2.45 mm), moderately obese (13.33±2.24 mm vs. 12.94±1.88 mm) and extremely obese hypertensives (13.39±2.29 mm vs. 12.94±1.93 mm). The differences were statisticaly significant ($p < 0.05$ for IVSTh values and $p < 0.001$ for PWTh values according to the analysis of variance).

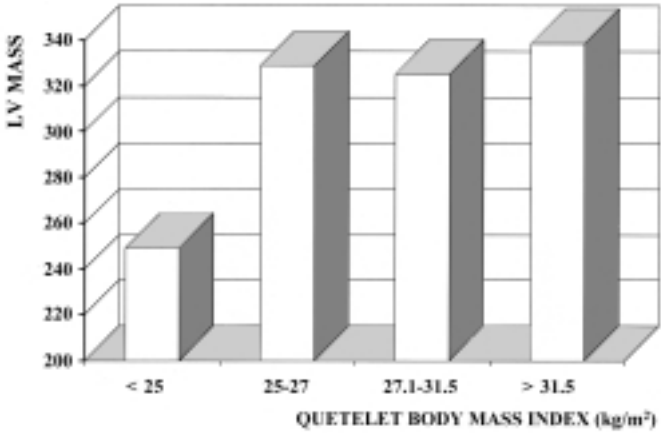

Fig. 3. Degree of obesity and left ventricular mass : 249.27±62.62 g in the group of lean hypertensives, 329.08±95.97 g for mildly obese, 325.21±89.86 g for moderately obese and 338.74±107.38 g for extremely obese hypertensives. The differences were highly significant ($p < 0.001$, analysis of variance).

and insignificantly higher in the group of lean hypertensives than in the subgroups of obese hypertensives (Figure 4). The mean values in the subgroups of obese hypertensives were almost identical.

Left ventricular ejection fraction values were normal in all the groups, being slightly and insignificantly higher among the lean hypertensives than among their less obese counterparts (Figure 5).

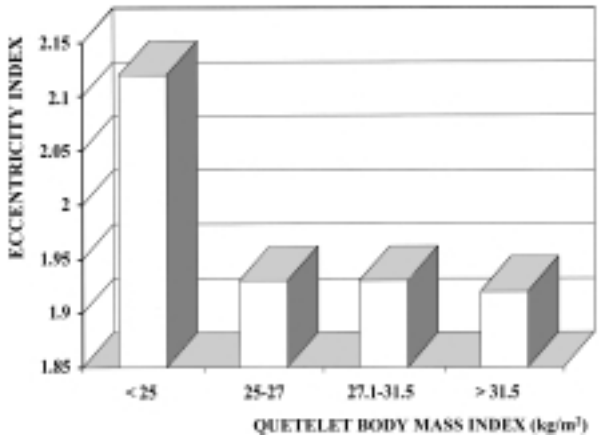

Fig. 4. Degree of obesity and »eccentricity index«: 2.124±0.476 for lean, 1.934±0.461 for mildly obese, 1931±0.351 for moderately obese and 1.930±0.385 for extremely obese hypertensives. The differences were not significant.

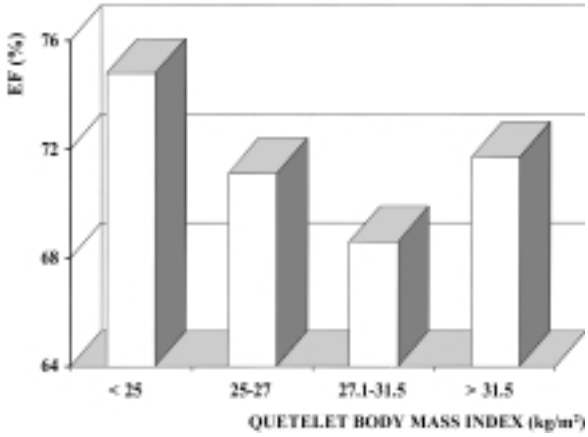

Fig. 5. Degree of obesity and ejection fraction: 74.83±4.62% for lean, 71.12±6.84% for mildly obese, 68.67±14.24% for moderately obese and 71.66±8.30% for extremely obese hypertensives. The differences were not significant.

The prevalence of left ventricular diastolic dysfunction was significantly higher among the obese, than among the lean hypertensives (Figure 6). It was two times higher in the group of extremely obese hypertensives than in the group of lean hypertensives.

Left atrial dimension was significantly bigger in the group of obese hypertensives than in the groups of their non-obese counterparts (Figure 1). The differences between the subgroups of obese hypertensives were small, but on average, extremely obese hypertensives had the largest atria.

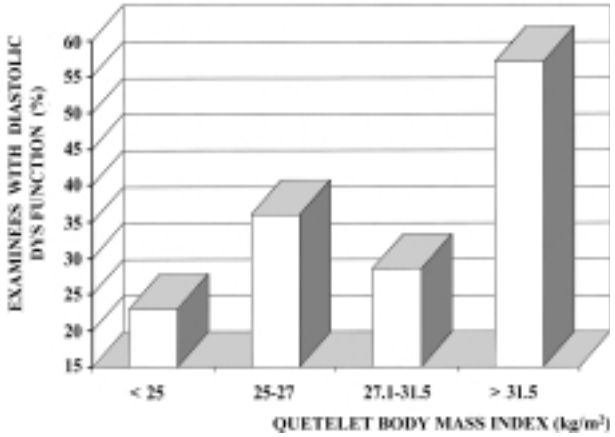

Fig. 6. Degree of obesity and left ventricular diastolic dysfunction: 23% for lean hypertensives had the signs of left ventricular diastolic dysfunction (7 out of 30). The respective values were 36& (9 out of 25) for mildly obese, 29% (12 out of 42) for moderately obese and 57% (20 out of 30) for extremely obese hypertensives. The differences were significant ($p < 0.05$, χ^2 test).

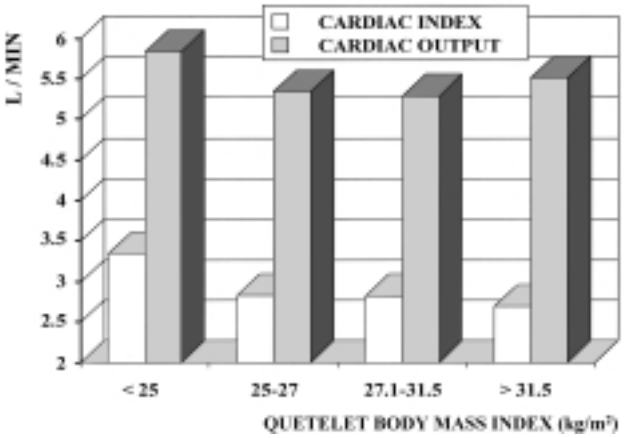

Fig. 7. Degree of obesity and cardiac output / cardiac index values in 98 hypertensives. In the group of 26 lean hypertensives, cardiac output was 5.85±1.44 l / min (mean value±SD) with cardiac index of 3.36±0.92 l / min / m². The group of mildly obese hypertensives included 16 patients with the cardiac output of 5.34±1.23 l / min and cardiac index of 2.84±0.61 l / min / m². The respective values for 25 moderately obese hypertensives were 5.29±1.40 l / min and 2.83±0.60 l / min / m², followed by 29 extremely obese hypertensives with 5.52±1.32 l / min and 2.69±0.59 l / min / m². The differences in cardiac output were insignificant, while all the groups of obese hypertensives had significantly lower values of cardiac output than the group of lean hypertensives ($p < 0.05$, analysis of variance).

In the group of 98 hypertensives, the values of cardiac output measured by Doppler method were very similar in all the groups arranged about their BMI values and grades of obesity (Figure 7). Corresponding to the definition, cardiac

TABLE 1
THE CORRELATIONS OF MORPHOMETRIC AND HAEMODYNAMICAL INDICES WITH THE BMI VALUES

	No. of pts.	r	p
LVIDd	132	0.1938	0.026*
LV mass	132	0.2823	0.001*
LA	132	0.2472	0.004*
PWTh	132	0.2598	0.003*
IVSTh	132	0.1428	0.102
LVIDd/IVSTh+PWTh	132	–0.1366	0.118
Cardiac output	98	0.0931	0.362
Cardiac index	98	0.0964	0.345

Asterisk (*) denotes statistical significance with p value < 0.05.

TABLE 2
CORRELATIONS OF THE MORPHOMETRIC INDICES WITH THE BMI VALUES IN THE SUBGROUPS DEFINED BY THE SEVERITY OF HYPERTENSION

hypertension	mild			moderate			severe		
	n	r	p	n	r	p	n	r	p
LVIDd	93	0.1502	0.151	35	0.6656	0.000*	5	0.6370	0.248
LV mass	93	0.2167	0.037*	35	0.6199	0.000*	5	0.0760	0.904
LA	93	0.2590	0.012*	35	0.6360	0.000*	5	0.1320	0.832
PWTh	93	0.2414	0.020*	35	0.7018	0.000*	5	–0.2674	0.664
IVSTh	93	0.1212	0.247	35	0.5892	0.000*	5	–0.7061	0.183

Asterisk (*) denotes statistical significance with p value < 0.05.

index values were considerably lower in obese than in lean hypertensives (Figure 7). However, the differences did not reach the level of statistical significance. Unexpectedly, the mean cardiac index values did not diminish further, proportionally to the increasing grades of obesity.

The majority of aforementioned cardiac morphometric and functional parameters were analyzed by linear regression procedures in the whole group of hypertensives. Morphometric indices were also analyzed in the subgroups defined by the age of the patient and by severity of hypertension.

The data for the whole group are presented in the Table 1. Evidently, all the morphometric indices (LVIDd, PWTh, LV mass and LA), excepting IVSTh and LVIDd/PWTh + IVSTh, correlated significantly with the BMI values, although the Pearson's correlation coefficients were very modest. Functional indices, i.e., cardiac output and cardiac index did not correlate with the BMI values.

Analysis of the subgroups regarding the severity of hypertension is presented in the Table 2. Obviously, the correlation of the morphometric indices with the BMI values was much better in the subgroup with moderate hypertension than in the one with mild hypertension. The subgroup of the patients with severe hypertension was small, so that even quite high correlation coefficients did not reach the level of statistical significance. The

best correlation (r = 0.76) was between the LVIDd/PWTh + IVSTh ratio and the BMI, but the significance was borderline (p = 0.05). It is depicted separately in the Figure 8, while the characteristic regression line for the BMI/LVIDd relation in the subgroup of moderate hypertensives is presented in the Figure 9.

The Table 3 presents the analysis of the morphometric indices in relation to the BMI values in the subgroups arranged according to the age. The level of statistical significance was reached only for posterior wall thickness in the fourth, left atrial dimension in the fifth and left ventricular mass in the sixth decade of life. The relationship between left atrial size and BMI in the forth decade of life is presented in the Figure 10.

Discussion

The principal questions demanding answers in our study might be defined as

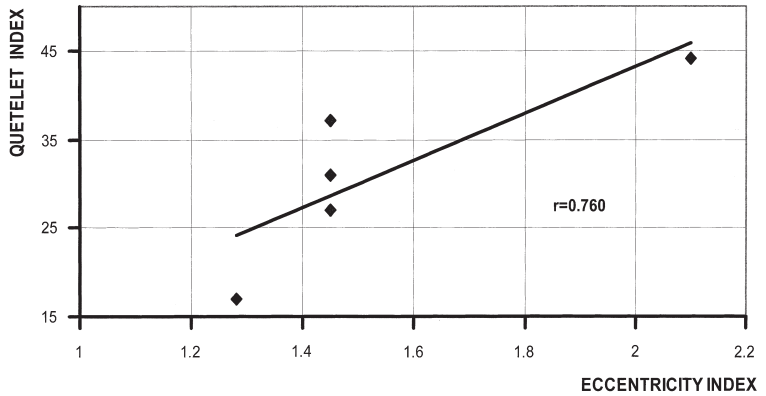

Fig. 8. The correlation between the BMI and »eccentricity index« values in the group of severe hypertension.

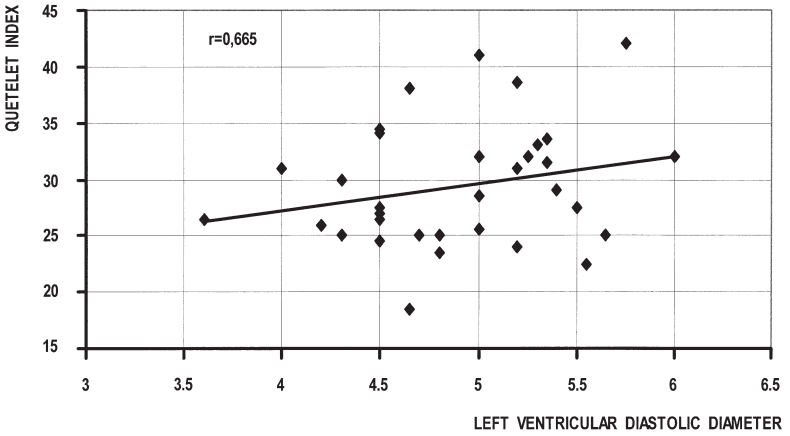

Fig. 9. The correlation between the BMI and LVIDd values in the group of moderate hypertension.

TABLE 3
THE CORRELATIONS BETWEEN THE LEFT VENTRICULAR AND LEFT ATRIAL MORPHOMETRIC INDICES WITH THE BMI VALUES IN THE DIFFERENT AGE GROUPS

age	30			31–40			41–50		
	n	r	p	n	r	p	n	r	p
LVIDd	9	0.0807	0.837	14	0.3865	0.172	46	0.1628	0.280
LV mass	9	0.2366	0.540	14	0.4462	0.110	46	0.2580	0.083
LA	9	0.5787	0.103	14	0.0666	0.821	46	0.4467	0.002*
PWTh	9	0.1536	0.693	14	0.5540	0.040*	46	0.2645	0.076
IVSTh	9	0.3660	0.333	14	0.0902	0.759	46	0.1245	0.410

age	51–60			61		
	n	r	p	n	r	p
LVIDd	35	0.2820	0.101	28	–0.0010	0.996
LV mass	35	0.4290	0.010*	28	0.0240	0.910
LA	35	0.2837	0.099	28	0.1415	0.473
PWTh	35	0.3195	0.061	28	0.1070	0.588
IVSTh	35	0.3014	0.078	28	0.0175	0.930

Asterisk (*) denotes statistical significance with p value < 0.05.

follows: »Do obese hypertensives really develop eccentric left ventricular hypertrophy as a typical form of hypertensive heart disease?«. Are they distinctively different from lean hypertensives who usually develop concentric left ventricular hypertrophy? Could be assumed with reasonable certainty that left ventricular hypertrophy in an obese hypertensive would be eccentric, instead of concentric hypertrophy?«

Intravascular volume and cardiac output are increased in obese persons (compared with lean ones) to fulfill the metabolic demands of increased adipose tissue mass[10,11]. Cardiac index is actually decreased[1,10,36]. As no essential changes in the basic heart rate are usually present, the principal determinant of the increase in cardiac output has to be an increase in stroke volume. It is due to increased left ventricular filling, providing that there are no substantial changes in myocardial contractility and peripheral vascular resistance. In the circumstances of long-standing increase in left ventricular filling, development of eccentric left ventricular hypertrophy, seems to be a logical consequence[10,11]. It is presumably due to the replication of sarcomeres in length, perhaps with some myocardial cell slippage[9].

Classical post-mortem studies confirmed that eccentric left ventricular hypertrophy is typical for very obese persons[10]. Echocardiographic studies demonstrated that on average, obese persons had larger left ventricles with thicker walls and bigger stroke volumes than non-obese controls[37,38]. This was especially true in the obesity of visceral type and long-standing duration[37,38]. However, the data on left ventricular geometry were conflicting. Until recently, the view prevailed that the increase in left ventricular mass in obese persons was mainly due to the cavity enlargement[39,40]. The echocardiographic evidence supporting this view was

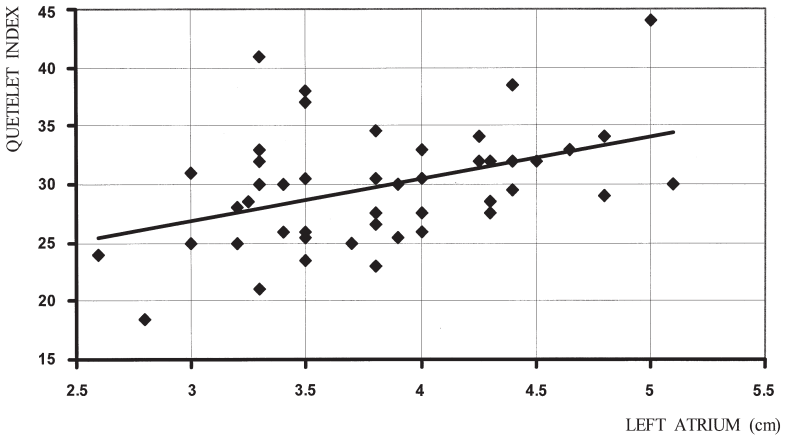

Fig. 10. The correlation between the BMI values and left atrial size in the fourth decade of life.

not very convincing for us. The overlapping of cardiac dimensions between the groups of obese and lean persons was substantial, despite the statistical significance of differences. Moreover, the data published after the termination of our study indicated that the relative wall thickness might be actually increased in obese persons[41].

The next step in our deduction is a shift from obese normotensives to obese hypertensives. Obesity and arterial hypertension have much in common, from epidemiological features and physiological derangements to the clinical, therapeutic and prognostic aspects[4,5,10,42,43]. Insulin resistance, a common metabolic denominator of both conditions has been much discussed lately[44–47]. In the clinical practice, both conditions can be found in the some patient so often, that they almost form a third entity: an obese hypertensive. The effects of hypertension and obesity on the increase in left ventricular mass are additive, both favoring the development of left ventricular hypertrophy[48–50].

The shape of the hypertrophied left ventricle has significant clinical implications. Not a long time ago, a distinguished author recommended diuretics for the basic antihypertensive treatment of obese persons, supposing that volume overload and eccentric left ventricular hypertrophy were the main features of the condition[2,39,51]. However, this appears to be a matter of controversy. While the most authors supported the common belief that left ventricular hypertrophy in obese hypertensives is mostly eccentric[39,52], some have recently found that concentric one is more common[50].

Our results suggest that eccentric left ventricular hypertrophy is not very typical for obese hypertensives in general. On average, they may have somewhat larger left ventricles than non-obese hypertensives, but the features of cavity size are not distinctive enough. It cannot be presumed without diagnostic (mainly echocardiographic) evaluation that particular hypertensive and obese hypertensive has eccentric, instead of concentric left ventricular hypertrophy[53–55]. Making presumptions on the left ventricular geometry just because of the patient's obesity does not seem to be justified, especially if therapeutic consequences are anticipated[56,57].

Left ventricular wall stress is especially high in hypertensives with dilated left ventricle, without sufficient wall thickening[58,59]. The resulting increase in myocardial oxygen consumption with the metabolic energy demand-supply imbalance may cause myocardial failure[6,60,61]. In such circumstances, the effects of antihypertensive drugs that may cause regression of the left ventricular hypertrophy with wall thinning may be unfavorable[62–64]. The bouts of hypertension after cessation of therapy may be deleterious. Therefore, some authors regarded eccentric left ventricular hypertrophy in arterial hypertension as an unfavorable condition with predisposition to systolic heart failure[58]. Others pointed to the specific adverse aspects of concentric left ventricular hypertrophy[50,65,66]. Left ventricular filling patterns are presumably different in hypertensives with concentric and eccentric left ventricular hypertrophy[23].

The adverse prognostic significance of left ventricular hypertrophy is well defined[65,67–69]. The eccentric hypertrophy in hypertensives may be in some aspects even more ominous than its concentric counterpart[47].

The lack of correlation between left ventricular dimensions and the BMI values is probably not surprising. »Pure« haemodynamical models are rare in clinical studies. »Pure« obesity may be characterized by left ventricular volume overload, but the features of established essential arterial hypertension are the decreases of intravascular volume and cardiac output with an increase in peripheral vascular resistance[1,2,70]. Besides of this, haemodynamical features are not the only determinants of left ventricular hypertrophy. Factors influencing the type and extent of left ventricular hypertrophy are manifold with complex interactions[5,70–74]. Physical activity also influences left ventricular mass[75] and the form of left ventricular hypertrophy. The example is physiological left ventricular hypertrophy in athletes[76–84]. Long distance runners and swimmers develop eccentric hypertrophy, while weight lifters are prone to the concentric hypertrophy[77]. Presumably, not all of our hypertensive patients were devoid of significant physical activity.

Relative wall thickness is not increased in eccentric left ventricular hypertrophy. In our study, it was slightly higher in obese than in non-obese hypertensives, while the eccentricity index was little bit lower. This was not surprising considering some published data[41] and the predominance of mild hypertension in our study. Left ventricular dimensions in the group of non-obese hypertensives were not in the range of hypertrophy.

This explanation was confirmed in part by the analysis of the subgroups. More obese patients with severe arterial hypertension had more pronounced »eccentricity« of the left ventricular hypertrophy than their less obese counterparts. The correlation's between left ventricular and left atrial dimensions with the BMI values were much better among the patients with moderate and severe hypertension than in the subgroup with mild hypertension. The influence of mild hypertension on left ventricular hypertrophy and geometry may be weak[85,86], while more severe forms of hypertension predispose to the eccentric hypertrophy development and left atrial enlargement in markedly obese persons. Previous data on this point are conflicting[50,85,86]. Moderate and severe forms of arterial hypertension in obese people must not be simply identified with advanced stages of essential arterial hypertension characterized by intravascular volume decrease and high peripheral vascular resistance[2,87].

In our study, left ventricular diastolic dimension and mass correlated with BMI values somewhat better in middle aged,

than in younger or elderly hypertensives. The correlations for all left ventricular and left atrial morphometric indices were especially poor among the patients over sixty years of age. The temporal patterns of arterial hypertension and obesity development in our patients were not defined precisely enough to allow the exact analysis of their mutual relationship through the process of aging[88,89].

It was found earlier that eccentric left ventricular hypertrophy is uncommon in persons under 50 years of age, but it is not rare in elderly hypertensives who are over 60[5,90]. However, this refers not to the particular group of predominantly obese hypertensives without coronary heart disease that was analyzed in ours study.

The finding that left ventricular filling impairment paralleled with advancing grades of obesity in our hypertensive patients was not surprising. Obesity can be associated with impaired left ventricular diastolic function even in normotensive subjects[9,91].

Cardiac index values were lower in our obese hypertensives than in their non-obese counterparts, as expected[4,10,11]. Unexpectedly, cardiac output values did not rise with advancing obesity. This was not much surprising, considering very small increments in left ventricular diastolic dimensions, slight decrease in ejection fraction and from other point of view, limited accuracy of Doppler cardiac output estimation[35,92]. Therefore, we abandoned idea of cardiac output analysis in the subgroups.

Our study is limited by a few possible shortcomings. One of them is the lack of a control group of obese normotensives. However, our study was basically designed to analyze the patterns of left ventricular hypertrophy in obese hypertensives, contrasted with the hypertrophy patterns in non-obese hypertensives. The introduction of the second (obese normotensives), or even the third (lean normotensives) control group may be confusing.

We are aware that Quetelet's body mass index is far from being the optimal indicator of body fat content and distribution. There are much more accurate methods nowadays[93,94]. However, more reliable techniques are also more expensive, possibly with some irradiation (CT scans). In our future studies, we shall include at least waist/hip ratio as a simple index of central obesity[39].

Our patients were not distributed evenly in the subgroups concerning the severity of hypertension. Mild hypertensives prevailed, moderate hypertension was quite frequent, while only a few patients had severe arterial hypertension. This probably reflects the prevalence of various grades of arterial hypertension in clinical practice[95,96]. The selection in favor of more severe forms of hypertension would bias the study. Nevertheless, the subgroup with severe hypertension was too small and some further investigation of the whole problem »on the large scale« would be desirable.

The groups of lean and obese hypertensives were comparable, with similar proportions of both sexes. The analysis of sexual differences would surpass the scope of our present study, but it may be the aim of our future studies. Sexual dimorphism is expected in normal hearts from the puberty[97–100]. On average, males have somewhat larger left ventricular mass than females, even if normalized to the body surface area[58,72]. In hypertensives, premenopausal women are less prone to the left ventricular hypertrophy than men[72]. Thus, the preponderance of women in the group with severe hypertension may have only diminished the extent of hypertrophy.

Our study was lacking the follow-up of the patients. Such approach would be very complex and probably surpass our

basic aim, defined by the title of this article. Our present study might be viewed upon as an impetus for the future longitudinal studies.

Our estimation of left ventricular diastolic function was incomplete, limited to the methodology used in everyday clinical routine. A complete estimation would include isovolumic relaxation time measurements and more elaborate analysis of left ventricular filling[23,24,32–34,101–104]. However, this would surpass the basic aim of the study. The E/A ratio, as a single index of diastolic performance has become the most popular method for clinical detection of left ventricular diastolic dysfunction[34]. Our E/A criterion f or diastolic dysfunction was rigorous to avoid false positive estimations and to overcome the uncertainties due to insufficient standardization of normal values and age influence[105–108]. The point of evaluation was not the estimation of the true diastolic dysfunction prevalence, but its relation to the grades of obesity. Our methodology was probably not free of some pitfalls that were not recognized at the time of initiation of the study. Some of them could have been avoided by the analysis of pulmonary venous flow velocity patterns[34].

Turning back to our basic question, whether obese hypertensives in clinical practice usually develop eccentric, instead of concentric left ventricular hypertrophy, we could probably offer some answers. There is a tendency in obese hypertensives towards the eccentric left ventricular hypertrophy, however, it is mostly weak and non discriminative. This is especially true for the mild hypertensives. In the cases of moderate and severe hypertension, the tendency towards eccentric left ventricular hypertrophy appears to be considerably more pronounced. The severity of arterial hypertension appears to be a major determinant for the left ventricle and left atrium enlargement in obese hypertensives. The ideal candidate for eccentric left ventricular hypertrophy appears to be a markedly obese, middle aged person with moderate, or severe arterial hypertension. It appears that eccentric left ventricular hypertrophy is not very typical for the most of obese hypertensives. However, it is quite common in certain subgroups whose characteristics we tried to define in this study.

Updating this manuscript since the time of submission, we have to comment a new large Norwegian study on left ventricular hypertrophy in population[100]. BMI and systolic pressure were confirmed as strong determinants of left ventricular mass, while the influence of age was remarkable only in persons with hypertensive, or some other heart disease. Left ventricular geometry in obese hypertensives was not analyzed. It remains the matter of controversy which we tried to elucidate by our modest contribution.

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DA LI JE HIPERTROFIJA LIJEVE KLIJETKE U PRETILIH HIPERTONIČARA: UISTINU EKSCENTRIČNA? (EHOKARDIOGRAFSKA STUDIJA)

S A Ž E T A K

U studiji oblika hipertrofije lijeve klijetke u pretilih hipertoničara, pregledali smo 132 bolesnika dvodimenzionalnom, M-mode i Doppler ehokardiografijom. Ispitanici su podijeljeni u četiri podjednake skupine prema vrijednostima Queteletovog indeksa tjelesne mase i stupnjevima pretilosti. Izrazitije pretili hipertoničari imali su u prosjeku malo veću lijevu klijetku s debljim stijenkama i veći lijevi atrij nego manje pretili i mršavi hipertoničari. Masa lijevog ventrikula se povećavala usporedo s povećanjem stupnja pretilosti, ali se relativna debljina stijenke (omjer debljine stijenke i veličine ventrikula) nije smanjivala.

Doppler ehokardiografija je pokazala značajno veću učestalost dijastoličke disfunkcije lijeve klijetke među pretilim, negoli među mršavim hipertoničarima.

U drugom dijelu studije, analizirali smo podskupine definirane težinom hipertenzije i dobi bolesnika. Korelacija pokazatelja hipertrofije lijeve klijetke i pretklijetke s vrijednostima Queteletovog indeksa je bila znatno bolja u skupini s umjerenom, nego li u skupini s blagom hipertenzijom. Vrijednosti koeficijenta r su bile 0,62 i 0,22 za masu lijeve klijetke te 0,64 i 0,26 za promjer lijevog atrija. Skupina bolesnika s teškom hipertenzijom isticala se povećanjem šupljine lijeve klijetke, usporedo s povećanjem vrijednosti Queteletovog indeksa, ali bez odgovarajućeg zadebljanja stijenke. Tzv. indeks ekscentričnosti, kao recipročna vrijednost relativne debljine stijenke lijeve klijetke, dobro je korelirao s Queteletovim indeksom (r = 0,76). Pokazatelji hipertrofije lijeve klijetke su malo bolje korelirali s Queteletovim indeksom u srednjim dobnim skupinama, nego u skupinama najmlađih (30 g) i najstarijih (60 g) hipertoničara.

U zaključku, ne čini se da je ekscentrična hipertrofija lijeve klijetke karakteristična osobina hipertenzivne bolesti srca u pretilih. Samo je naznačena tendencija »ekscentričnosti« geometrije lijevog ventrikula koja postaje izrazitija u težim oblicima hipertenzije, posebno u vrlo pretilih osoba.