

## CLINICAL STUDY

# Relation of various degrees of body mass index to systolic and diastolic dysfunctions

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**Abstract:** *Objectives:* This study was designed to identify any associations between left ventricular function and obesity using clinical two-dimensional echocardiographic and Doppler studies.

*Methods:* We retrospectively evaluated 260 consecutive clinical echocardiographic findings. Key echocardiographic variables of systolic and diastolic function were related to various degrees of body mass index.

*Results:* In multiple regression analysis in the whole group of patients there was significant relation of body mass index to left ventricular mass and its indexed value ( $p < 0.0001$ ). Multiple regression analyses in subgroups of patients according to systolic and diastolic function or dysfunction revealed similarly the strongest association of body mass index to mainly left ventricular mass. In subgroups of patients classified on the basis of their body mass indices significant differences were found also in case of left ventricular mass as well as left atrial volume ( $p = 0.0001$ , and  $p = 0.003$ , respectively). There was no association between body mass index and systolic or diastolic dysfunction.

*Conclusion:* We found strong association between obesity and left ventricular mass. Obesity was not related to systolic and diastolic function or dysfunction (Tab. 4, Fig. 1, Ref. 29). Full Text (Free, PDF) [www.bmj.sk](http://www.bmj.sk).

**Key words:** obesity, systolic dysfunction, diastolic dysfunction.

Average body weight has been increasing in the past decade all over the world (1). Obesity is associated with a preclinical manifestation of cardiovascular disease, which is prognostically relevant (2–6). Moreover, obesity is associated with increases in cardiovascular event rates (7, 8). This study has been undertaken to assess the relation of various degrees of body mass index to echocardiographic parameters.

## Patients and methods

We retrospectively evaluated 260 consecutive clinical echocardiographic findings from January to September 2006. Echocardiography and cardiac Doppler studies were performed using a Vivid Five (GE Medical Systems) or Sequoia (Siemens Medical Solutions) ultrasonographs with a 2.25–3.75 MHz transducer. The study group consisted of 260 patients (130 women, 130 men, mean age  $60.2 \pm 17.3$  years). Echocardiography was performed and echocardiographic measurements were obtained in accordance with American Society of Echocardiography criteria (9). Key echocardiographic variables assessed at examination are presented in Table 1. In patients with no wall motion abnormalities,

ejection fraction (EF) was assessed semiquantitatively and in patients with wall motion abnormalities it was assessed by using modified biplane Simpson's rule. Left atrial volume (LAV) and its indexed value were measured by monoplane Simpson's rule. Left ventricular mass (LVM) was calculated using the formula of Devereux et al and was indexed to body surface area (10). LV diastolic filling was assessed by pulse-wave Doppler echocardiography. The peak E-wave (early filling wave) velocity, peak A-wave (late filling wave) velocity, and the transmitral Doppler E-wave deceleration time were measured, as well as the E/A ratio was calculated. Color Doppler M-mode recordings of left ventricular inflow from an apical approach was used to measure the propagation velocity ( $V_p$ ) (11, 12). A measure of combined systolic and diastolic performance, the so called Tei index, was also assessed from transmitral Doppler flow using the formula  $(a - b)/b$ , where  $a$  is the time interval between the end of A wave and beginning of E wave, and  $b$  is the ejection time (13). Patients were divided into groups with normal systolic function ( $EF > 50\%$ ) and systolic dysfunction ( $EF < 50\%$ ). Other groups were classified according to different forms of diastolic dysfunction: impaired relaxation, pseudonormal filling, and restrictive physiology. Impaired relaxation was defined:  $E/A < 1$ , E-wave deceleration time  $> 240$  ms, isovolumic relaxation time (IVRT)  $> 100$  ms,  $V_p > 45$  cm/s. Pseudonormal filling was:  $E/A > 1$ , E-wave deceleration time was normal (160–240 ms), IVRT also normal (60–100 ms), but  $V_p < 45$  cm/s. Restrictive physiology was defined as follows:  $E/A > 2$ , E-wave deceleration time  $< 160$  ms, IVRT  $< 60$  ms, and  $V_p < 45$  cm/s. In addition,

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**Tab. 1. Mean±SD of the assessed echocardiographic parameters classified according to systolic and diastolic function or dysfunction.**

	Normal findings	Impaired relaxation	Pseudo-normalized pattern	Restrictive physiology	EF>50%	EF<50%	Impaired relaxation with EF>50%	Impaired relaxation with EF<50%
Age	43.2±15.8	66.8±11.2	64.4±15.9	66.1±13.3	59.9±17	68.6±12	66.7±11.5	67.6±9.6
LVM (g)	144.7±43.7	208.9±62.6	273.4±98.9	330.4±113.4	190.2±69.7	276.2±94.7	199.9±55.8	271.4±74.6
LVMI (g/m <sup>2</sup> )	78.2±21.8	112±32.7	150.3±54.6	165±46.11	101.3±34.9	149.7±47.1	106.3±26.6	148.6±45.7
LAV (ml)	46.2±12.6	62.7±20.7	90.2±43.8	72.1±139.9	70±42.7	105±75.4	61.8±19.4	70.2±27.3
LAVI (ml/m <sup>2</sup> )	25.3±6.7	33.6±11.6	48.7±21.8	84.5±60.5	37.9±24.1	54.9±34.7	33.1±10.9	38.3±15.4
EF (%)	60.4±7.6	55.4±8.2	35.9±11.7	35.5±14.6	58.4±5	34.9±7.5	58±5	39±6
Tei index	0.2±0.1	0.4±0.2	0.4±0.3	0.7±0.4	0.3±0.1	0.6±0.2	0.3±0.1	0.6±0.2
E (cm/s)	79.6±16.7	59.5±15.1	76±11.7	123.7±17.7	74.8±22.8	79.3±29.6	60.9±14.7	51.1±15.6
DT E (ms)	186.7±36.8	260.2±59.7	152.3±53.8	105±23.5	210.7±66.2	189.7±83.4	259.6±58.7	264.3±70.7
A (cm/s)	64.3±14.9	84.1±16.8	67.2±19.5	48.7±14.8	75±22.2	70.6±23.5	84.6±16.8	82.5±16.4
E/A	1.2±0.4	0.7±0.1	1.1±0.4	2.6±0.6	1±0.5	1.2±0.9	0.7±0.1	0.6±0.1
IVRT (ms)	88±15	114.1±25.1	85.4±33.3	55.5±15.1	99.2±25.5	97.7±36.4	113±25.1	120.1±25.3
Vp (cm/s)	54±14.4	39.1±0.6	33.7±14.3	35.4±10.1	45.8±15.9	31.9±11.6	40.4±17	31.3±13.6
E/Vp	1.5±0.43	1.7±0.6	2.6±1.22	3.7±1.1	1.8±0.9	2.6±1.2	1.7±0.631	1.8±0.63

LVM – left ventricular mass, LVMI – left ventricular mass index, LAV – left atrial volume, LAVI – left atrial volume index, EF – ejection fraction, E – transmitral E wave, DT E – E wave deceleration time, A – transmitral A wave, IVRT – isovolumic relaxation time, Vp – transmitral flow propagation velocity

**Tab. 2. P values of multiple regression analyses in subgroups of patients classified according to systolic and diastolic function or dysfunctions.**

	LVM	LVMI	LAV	LAVI	EF	FS	Tei index	E	DTE	A	E/A	IVRT	Vp	E/Vp
All patients	0.0001	0.0001	0.3	0.2	0.7	0.01	0.4	0.7	0.7	0.0003	0.7	0.13	0.9	0.7
Normal findings	0.10	0.18	0.9	0.9	0.6	0.8	0.4	0.9	0.8	0.3	0.8	0.005	0.9	0.83
Impaired relaxation	0.01	0.02	0.3	0.5	0.9	0.5	0.9	0.5	0.6	0.14	0.6	0.7	0.8	0.33
Impaired relaxation with EF>50%	0.04	0.04	0.5	0.6	0.8	0.7	0.9	0.8	0.7	0.1	0.6	0.3	0.9	0.3
Impaired relaxation with EF<50%	0.03	0.04	0.2	0.2	0.2	0.5	0.6	0.3	0.2	0.4	0.3	0.5	0.2	0.3
EF>50%	0.03	0.04	0.04	0.1	0.6	0.2	0.6	0.4	0.5	0.0001	0.5	0.6	0.6	0.9
EF<50%	0.01	0.01	0.7	0.8	0.6	0.19	0.9	0.1	0.3	0.14	0.8	0.8	0.2	0.3

LVM – left ventricular mass, LVMI – left ventricular mass index, LAV – left atrial volume, LAVI – left atrial volume index, EF – ejection fraction, E – transmitral E wave, FS – fractional shortening, DT E – E wave deceleration time, A – transmitral A wave, IVRT – isovolumic relaxation time, Vp – transmitral flow propagation velocity

we analysed two subgroups of patients with impaired relaxation, one with preserved systolic function and another with systolic dysfunction. Subjects were classified on the basis of their BMIs (weight in kilograms divided by the square of height in meters) as normal (18.5 to <25 kg/m<sup>2</sup>), overweight (>25 and <30 kg/m<sup>2</sup>), obese (>30 and <35 kg/m<sup>2</sup>), or severely obese (>35 kg/m<sup>2</sup>).

**Statistical analysis**

Continuous variables are reported as mean±SD. Differences among the variables were assessed by one-way analysis of variance. If significant difference was found by one-way analysis of variance, Tukey-Kramer’s post hoc test for intergroup comparisons was used. Multiple linear regression models were used to predict the independent contribution of factors that influenced

the BMI. A p value of <0.05 was considered significant for all analyses.

**Results**

In multiple regression analysis in the whole group of patients there was a significant relation of BMI to LVM (p<0.0001), LVMI (p<0.0001), fractional shortening (FS) (p=0.01), and transmitral A wave (0.0003). There was no relation of BMI to other echocardiographic parameters: LAV (p=0.3), LAVI (p=0.2), EF (p=0.7), Tei index (p=0.4), E wave (p=0.7), E-wave deceleration time (p=0.7), E/A ratio (p=0.7), IVRT (p=0.13), Vp (p=0.9), and E/Vp (p=0.7). The regression equation was:

$$BMI = 17.93869 - 0.060966 LAV + 0.144506 LAVI - 0.009961 EF + 0.060361 FS - 0.900227 TEI + 0.007814 E - 0.001786$$

**Tab. 3. Mean±SD of the assessed echocardiographic parameters classified according to BMIs.**

	BMI			
	18–25	25–30	30–35	>35
LVM (g)	166.6±74.3	203.9±72.5	228.7±79.6	254.8±59.9
LVMI (g/m <sup>2</sup> )	97.4±41.5	108.2±34.1	115±35.3	121.94±26.2
LAV (ml)	57.9±33.3	63.4±24.5	80.3±61	74.7±26.6
LAVI (ml/m <sup>2</sup> )	34.8±20.5	33.9±13.5	40.5±28.2	35.7±12.5
EF (%)	55±12.2	55.4±9.7	55.9±9.1	51.6±10.8
FS (%)	37.4±12	40±11	39.8±11.9	37.1±17.4
Tei index	0.39±0.2	0.40±0.19	0.39±0.25	0.34±0.14
E (cm/s)	23±21.6	70.4±23	72.4±23.2	70±24.3
DT E (ms)	209.5±82	220.5±61.2	217.6±61.4	244.4±76.3
A (cm/s)	72.8±24.1	77.7±22.2	78.2±21.2	76.9±13.8
E/A	1.12±0.53	1.78±0.39	1.06±0.7	0.96±0.52
IVRT (ms)	95.9±27.2	102.5±25.4	199.7±22.1	108.23±19.1
Vp (cm/s)	41.7±14.4	42.6±16.7	44.3±15.3	36.9±9.5
E/Vp	1.9±0.73	1.8±1.01	1.8±0.9	2.06±1

LVM – left ventricular mass, LVMI – left ventricular mass index, LAV – left atrial volume, LAVI – left atrial volume index, EF – ejection fraction, E – transmitral E wave, FS – fractional shortening, DT E – E wave deceleration time, A – transmitral A wave, IVRT – isovolumic relaxation time, Vp – transmitral flow propagation velocity

**Tab. 4. Multiple comparison of the assessed echocardiographic parameters between subgroups of patients according to BMIs.**

	P value (ANOVA)
LVM	0.0001
LVMI	0.008
LAV	0.003
LAVI	0.23
EF	0.4
FS	0.4
Tei index	0.6
E	0.8
DT E	0.2
A	0.3
E/A	0.6
IVRT	0.13
Vp	0.2
E/Vp	0.73

LVM – left ventricular mass, LVMI – left ventricular mass index, LAV – left atrial volume, LAVI – left atrial volume index, EF – ejection fraction, E – transmitral E wave, FS – fractional shortening, DT E – E wave deceleration time, A – transmitral A wave, IVRT – isovolumic relaxation time, Vp – transmitral flow propagation velocity

$$DTE + 0.048537 A + 0.020865 E/A + 0.018132 IVRT - 0.002364 Vp - 0.221381 E/Vp + 0.166552 LVM - 0.296392 LVMI$$

Multiple regression analyses in subgroups of patients according to systolic and diastolic functions or dysfunctions revealed similarly the strongest association of BMI to mainly LVM and LVMI. The data are presented in Table 2.

The mean±SD of the analysed echocardiographic parameters in subgroups of patients classified on the basis of their BMIs are presented in Table 3. One-way analysis of variance showed significant differences between these subgroups of patients only in case of LVM, LVMI and LAV ( $p=0.0001$ ,  $p=0.008$ , and  $p=0.003$ , respectively). There were no significant differences in intergroup

comparisons of other parameters. The data are presented in Table 4. The increase of LVM and LVMI depending on the increase of BMI is presented in Figure 1.

## Discussion

This study was designed to identify any associations between left ventricular function and obesity. Our analysis showed that in subgroups of patients classified according to systolic and diastolic functions or dysfunctions, the increasing BMI was associated mainly with LVM and LVMI. Only in subgroups of patients with normal systolic function and normal echocardiographic findings, there was a significant relation of BMI to LAV, transmitral A wave and IVRT. In the whole group of patients we found a significant association of BMI with fractional shortening and A wave. There was no relation between obesity and other echocardiographic parameters of systolic and diastolic functions, such as EF, Tei index or global myocardial performance index, and left ventricular filling. Similarly, in subgroups of patients classified according to BMI, we found a significant association of BMI only with LVM, LVMI and LAV. Our results are partly in concordance with the study by Powell and associates, who analysed 4,281 patients without coronary artery disease and found a significant relation between BMI and LVM; there was no association between BMI and EF. Contrary to our results, the authors revealed a relation of BMI to left ventricular diastolic function assessed either by echocardiography or invasively (14). In a similar study by Dorbala and associates, left ventricular EF was not adversely affected, not even in cases with severe degrees of obesity (15).

Although obesity has adverse effects on cardiovascular structure and function and may be a risk factor for heart failure, several recent studies have suggested that in patients with chronic systolic heart failure, obesity is actually associated with trends

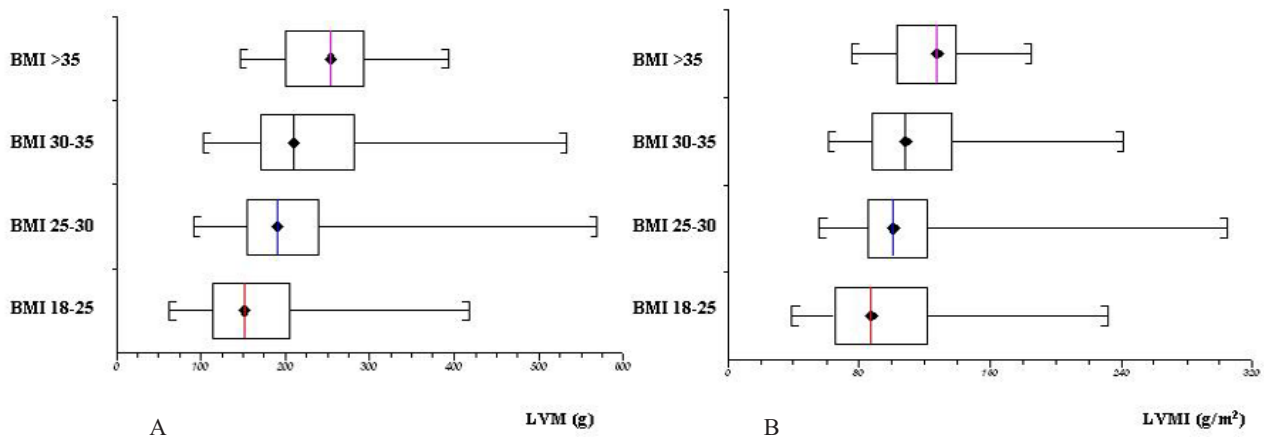


Fig. 1. Box and whisker plots of the relation between various degrees of body mass index (BMI) and left ventricular mass (LVM) and left ventricular mass index (LVMI) (A, B).

for better survival (16–21). This obesity paradox or „protective effect“ of a greater BMI against systolic dysfunction seems to be proved also in our study – no relation between systolic dysfunction and high BMI. There is no explanation how can high BMI protect the heart. It has been shown that adipose tissue produces soluble tumor necrosis factor receptors, which are believed to neutralize the deleterious effects of tumor necrosis factor on the myocardium; thus, the possible protective effect of obesity (22). On the other hand, lower body weight is associated with an increased catabolic state, which is related to higher levels of tumor necrosis factor and other cytokines and increased cortisol/dehydroepiandrosterone balance (23, 24). The strong relation between the increasing degree of obesity and LVM proved in our study indicates on the other hand some harmful effect of obesity on the heart. Alexander and Alpert described the cardiomyopathy of obesity, which is characterized by increased cardiac output and left ventricular volume. These changes in the early stage could theoretically lead to left ventricular diastolic dysfunction and the eventual impairment of systolic function (25–27). Our study supports this theory only regarding the left ventricular hypertrophy. We did not prove any relation between obesity and systolic and diastolic dysfunctions. However, the association between obesity and cardiomyopathy is still being debated. Coughlin and Rice performed a pooled analysis of 2 case-control studies of idiopathic dilated cardiomyopathy (28). The authors did not find any association between obesity and idiopathic dilated cardiomyopathy.

In subgroups of patients classified on the basis of their BMIs, beside significant differences in LVM and LVMI, we found significant differences also in LAV. Intergroup analysis showed that in obese and severely obese patients mean LAV ( $80.3 \pm 61$  ml, and  $74.7 \pm 26.6$  ml, respectively) was significantly larger than in patients with normal BMI ( $57.9 \pm 33.3$  ml). However, this finding was not proved in case of LAVI. This is partly in concordance with the study by Pritchett and associates, who found no big differences in LAVI between lean ( $BMI \leq 28$ ) and overweight

or obese normal individuals ( $BMI \geq 28$ ). The LAVI in subgroup of 300 obese participants ( $22.85 \pm 5.40$  ml/m<sup>2</sup>) was only slightly larger than for lean participants ( $21.46 \pm 5.14$  ml/m<sup>2</sup>) (29). The LAVI of our group of lean subjects ( $34.8 \pm 20.5$  ml/m<sup>2</sup>) is larger than the LAVI of lean participants in the above mentioned study. The reason is the difference in population studied. While our group of lean patients includes heterogeneous subjects with systolic or diastolic dysfunctions, the group of patients studied by Pritchett and associates includes subjects with no cardiovascular disease and normal systolic and diastolic functions.

Our study supports works proving obesity to be related to left ventricular mass, but not related to systolic function or dysfunction. Unlike the findings in previous reports we found no relation between obesity and diastolic function or dysfunction.

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