Key messages

- Patients undergoing hip fracture repair constitute a high risk group with considerable mortality and morbidity and an often protracted postoperative hospital stay
- These patients often have depleted intravascular volume in the perioperative period and rarely receive either invasive haemodynamic monitoring or high dependency care
- Haemodynamic optimisation guided by pulmonary artery catheter in the perioperative period has been shown to improve outcome in high risk patients undergoing major surgery, but this is not considered routinely practicable for hip fracture repair
- Intravascular volume optimisation directed by minimally invasive oesophageal Doppler monitoring in the intraoperative period significantly reduces hospital stay

postoperative period may be partly responsible for patients not benefitting from aggressive haemodynamic management after they have been admitted to an intensive care unit.[19 20] Use of this simple procedure could produce considerable cost benefit in terms of shorter hospital stays and improved patient outcome.

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Effects of obesity and weight loss on left ventricular mass and relative wall thickness: survey and intervention study

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continued over

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Abstract

Objectives: To investigate the consequences of longstanding obesity on left ventricular mass and structure and to examine the effects of weight loss on these variables.
Design: Cross sectional survey and controlled intervention study.
Setting: City of Gothenburg and surrounding areas, Sweden.
Subjects: 41 obese patients treated with weight reducing gastric surgery, 31 obese patients treated conventionally, and 43 non-obese subjects.
Main outcome measures: Changes in left ventricular mass and relative wall thickness.
Results: Obese patients had higher blood pressure, greater left ventricular mass, and increased relative wall thickness than did matched non-obese control subjects. Obese subjects treated with gastric surgery had a substantial weight loss and a significant reduction in all variables when compared with conventionally treated obese subjects. Univariate and multivariate analysis of pooled data from the two groups of obese subjects showed that changes in relative wall thickness and left ventricular mass were more closely related to the change in weight than to the concomitant change in blood pressure.
Conclusions: Structural heart abnormalities occurring in conjunction with obesity diminish after weight loss. The regression in these structural aberrations is better predicted by the weight loss than by the accompanying reduction in blood pressure. To prevent or improve abnormalities of heart structure in obese people, weight control should be the primary goal; it should be regarded as at least as important as regulating blood pressure.

Introduction

Obesity is related to several disturbances in cardiac structure.[1] Obese people have greater left ventricular mass, greater wall thickness, and larger chamber size than those who are not obese,[2 3] and the ratio between wall thickness and chamber radius (the relative wall thickness) is larger in obese people than in lean people.[1 4] These aberrations in left ventricular mass and structure are of great importance. Left ventricular hypertrophy is one of the strongest risk factors for cardiovascular morbidity and mortality,[5] and an increase in relative wall thickness has been shown to increase cardiovascular risk.[6 7]

The changes in left ventricular mass and structure with increasing body weight can be partially explained by the haemodynamic changes that accompany obesity.[8 9] As body weight increases, total blood volume and cardiac output rise. This leads to a volume overload that causes left ventricular dilatation and a parallel thickening of the ventricular wall (eccentric left ventricular hypertrophy). Obesity is also closely related to arterial hypertension,[10 11] a form of pressure overload that is followed by increased wall thickness without chamber dilatation (concentric left ventricular hypertrophy). Metabolic and hormonal factors can also influence the heart structure of obese people.[12]

Though it is well known that the medical treatment of hypertension can induce a regression in left ventricular hypertrophy,[13] little is known about the effect of weight reduction on left ventricular mass. The few studies on the subject have produced contradictory results.[14-17] The importance of relative wall thickness for estimating cardiovascular risk has only recently been spotlighted and is still under debate.[18] The effect of weight reduction on relative wall thickness has not previously been investigated. We investigated the consequences of longstanding obesity on left ventricular mass and structure and examined the effects of weight loss on these variables.

Subjects and methods

In total, 119 subjects from the city of Gothenburg and the surrounding areas were enrolled, comprising 61 men and 58 women with ages ranging from 37 to 61 years. The study population consisted of two groups of obese patients (body mass index 30-47 kg/m^2) and one group of non-obese subjects (body mass index 18-27 kg/m^2). The obese subjects were recruited from the ongoing Swedish obese subjects study, which is a nationwide trial designed to determine whether the mortality and morbidity among obese people who lose weight by surgical means differs from that in an obese reference group.[11] The non-obese subjects were recruited from a randomly selected sample of adults living in the municipality of Mölndal.

The two groups of obese subjects comprised 41 consecutive patients referred for weight reducing gastric surgery (the "obese operation" group) and 35 matched control subjects who were treated with conventional dietary recommendations ("obese control" group). The non-obese group consisted of 43 subjects matched with the obese groups for sex, age, and height. Subjects in all three study groups were examined at baseline and those in the two obese groups were examined again after one year. Four of the obese control patients were excluded from the study as they did not participate in the follow up, leaving 31 subjects in the obese control group. Table 1 shows the clinical characteristics of the three study groups (115 subjects).

Body weight was measured with the subjects wearing light clothing and no shoes and was rounded to the nearest 0.1 kg. Height measurements were rounded to the nearest 0.01 m, and body mass index was calculated as the weight in kilograms divided by the height in metres squared.

Systolic and diastolic (phase V) blood pressure was measured in the right arm using a mercury sphygmomanometer with the subject in the supine position after 10 minutes of rest. An appropriate cuff was used, with a width of at least 40% of the circumference of the arm. Echocardiography was performed on each subject in the left lateral decubitus position, using a commercially available ultrasound system (Accuson 128 XP; Mountain View, CA) with 2.0-2.5 MHZ transducers. Two dimensional echocardiography registrations were obtained with short axis and four chamber views. From the left ventricular short axis view, epicardial and endocardial perimeters were traced and mean wall thickness and cavity radius were calculated. Relative wall thickness was defined as the ratio of mean wall thickness to chamber radius. Left ventricular mass was calculated according to the truncated ellipsoid algorithm from Byrd et al.[19] Left ventricular diastolic volumes were estimated from the four chamber view, using the disc summation method (modified Simpson's rule).

All recordings were performed by doctors experienced in echocardiography, and 75% of the registrations were made by one investigator (IW). Each reading was assessed before statistical analyses took place, and only subjects with recordings of excellent or good quality were included in data analyses. As a result, 9 (13%) of the obese patients were excluded from the analyses of left ventricular wall thickness and mass and 20 (28%) from the estimations of left ventricular

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Table 1 Clinical characteristics of study groups at baseline and one year follow up. Values are means (SD) unless specified otherwise

	Obese aubjects (n=72)			Non-obese subjects (n=43)	Difference between non-obese and obese (95% CI)
	Operation (n=41)	Control (n=31)	Difference (95% CI)		
No of men	21	16	NS	23	NS
Age (years)	48 (6)	49 (6)	NS	49 (7)	NS
Height (cm)	173 (10)	171 (9)	NS	173 (9)	NS
No (%) current smoker	11 (27)	6 (19)	NS	8 (19)	NS
No (%) receiving antihypertensive treatment	9 (22)	8 (26)	NS	1 (2)	P=0.002
Weight (kg):					
Baseline	117 (15)	112 (14)	–5 (–12 to 2)	70 (11)	45 (40 to 50)**
One year follow up†	84 (14)	115 (14)	31 (26 to 36)**		
Body mass index (kg/m^2):					
Baseline	39 (4)	39 (5)	0 (–2 to 2)	23 (2)	16 (14 to 18)**
One year follow up†	29 (3)	39 (3)	10 (8 to 12)**		
Systolic blood pressure (mm Hg):					
Baseline	143 (18)	139 (18)	–4 (–12 to 4)	116 (14)	25 (19 to 31)**
One year follow up†	125 (14)	141 (14)	16 (10 to 22)**		
Diastolic blood pressure (mm Hg):					
Baseline	88 (11)	83 (12)	–5 (–11 to 1)	72 (11)	14 (10 to 18)**
One year follow up†	75 (9)	85 (9)	10 (6 to 14)**		

**P<0.001.
†Adjusted mean (SD).

Table 2 Mean (SD) echocardiographic measurements at baseline and one year follow up

	Obese subjects			Non-obese subjects	Difference between non-obese and obese subjects(95% CI)
	Operation	Control	Difference (95% CI)		
Truncated ellipsoid model	(n=38)	(n=25)		(n=42)	
Chamber radius (mm):					
Baseline	22.4 (2.5)	23.0 (3.3)	0.6 (–0.9 to 2.1)	21.9 (2.9)	0.8 (–0.3 to 1.9)
One year follow up†	24.1 (2.2)	23.3 (2.2)	–0.8 (–1.9 to 0.3)		
Average wall thickness (mm):					
Baseline	13.2 (2.3)	12.6 (2.3)	–0.6 (–1.8 to 0.6)	10.8 (0.19)	2.2 (1.4 to 3.0) (P< 0.001)
One year follow up†	11.1 (2.1)	12.7 (2.1)	1.6 (0.6 to 2.6) (P=0.005)		
Relative wall thickness:					
Baseline	0.60 (0.13)	0.57 (0.16)	–0.03 (–0.10 to 0.04)	0.50 (0.10)	0.09 (0.04 to 0.14) (P=0.001)
One year follow up†	0.47 (0.12)	0.55 (0.12)	0.08 (0.02 to 0.14) (P=0.01)		
Left ventricualr mass (g):					
Baseline	193 (57)	188 (44)	–5 (–31 to 23)	138 (40)	53 (34 to 72) (P< 0.001)
One year follow up†	165 (41)	189 (41)	24 (3 to 45) (P=0.03)		
Apical four chamber view	(n=29)	(n=23)		(n=42)	
Left ventricular volume diastole (ml):					
Baseline	104 (27)	97 (23)	–7 (–23 to 9)	80 (20)	21 (11 to 31) (P< 0.001)
One year follow up†	93 (23)	96 (23)	3 (–11 to 17)		

†Adjusted mean (SD).

volumes. Only 1 (2%) of the lean subjects was excluded from data analyses because of deficient registrations. The standard error of a single determination of left ventricular mass among obese subjects was 17%, assessed by a double determination in nine patients.

Statistical analyses were performed with the Statview (Abacus Concepts; Berkeley, CA) and SAS (SAS Institute, Cary, NC) statistical software packages. The data are summarised as means (SD). At baseline, differences between non-obese and obese groups were assessed with χ^2 or unpaired t tests, and at the one year follow up, differences between the obese operation and the obese control groups were investigated with analyses of covariance with adjustment for baseline values. After data in the obese groups were pooled, associations between changes in body weight and blood pressure and changes in left ventricular measurements were evaluated with univariate and multivariate regression analyses. All probability values were derived from two tailed tests, and a P value <0.05 was considered significant.

Results

At baseline, there were no differences in sex ratio, age, height, or smoking habits between obese and non-obese subjects. By definition, obese subjects had a significantly higher body weight and body mass index than lean ones; they also had higher blood pressure, and a greater proportion were receiving antihypertensive treatment (table 1). At baseline, clinical and echocardiographic variables were similar in the two obese groups (tables 1 and 2).

Although short axis measurements at baseline showed no difference in chamber radius between obese and non-obese subjects, measurements from the four chamber view showed that obese subjects had a larger left ventricular volume than lean ones (table 2). Moreover, in comparison to non-obese subjects, obese patients had increased wall thickness, increased relative wall thickness, and a greater left ventricular mass (table 2).

At the one year follow up, the surgically treated obese patients showed substantial reductions in weight and body mass index and significant decreases in systolic and diastolic blood pressure in comparison to conventionally treated obese patients (table 1). They also had significant reductions in wall thickness, relative wall thickness, and left ventricular mass. Left ventricular dimension and volume were similar in the two groups of obese patients (table 2).

Analyses of pooled data from the obese operation and obese control groups showed that changes in weight and changes in relative wall thickness and left ventricular mass were significantly correlated (figure). Changes in blood pressure correlated significantly with changes in relative wall thickness ($r=0.33$; $P<0.05$) but not with changes in left ventricular mass. Multiple regression analysis showed that changes in relative wall thickness and left ventricular structure were predicted by baseline relative wall thickness and baseline left ventricular mass respectively, as well as by changes in body weight. Changes in blood pressure did not contribute to the variation of left ventricular structure explained by these analyses (table 3). The findings persisted even after adjustment for age, sex, and antihypertensive treatment.

Discussion

The Framingham heart study has clearly shown that both obesity and hypertension are associated with increased left ventricular mass and that left ventricular hypertrophy is one of the strongest risk factors for cardiovascular morbidity and mortality.[5 20] Several studies have shown that left ventricular hypertrophy is reduced after the pharmacological treatment of hypertension,[21] but results of studies on the effect of weight

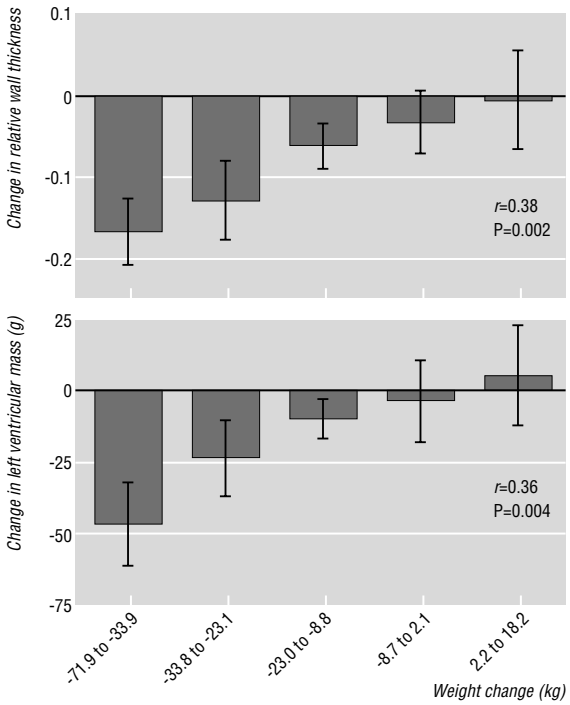
loss on left ventricular mass have been scarce and inconsistent.

In 1972 Alexander and Peterson reported that raised left ventricular filling pressure in obese subjects persisted three years after weight loss and concluded that myocardial hypertrophy did not regress after weight reduction.[14] Likewise, Alpert et al reported that surgically induced weight loss (mean 56 kg) in a group of obese patients had no effect on septal or posterior wall thickness.[15] In contrast, MacMahon et al found that a weight loss of only 8 kg in mildly obese patients with hypertension was associated with a significant decrease in left ventricular mass,[16] and more recently, Alpert et al observed a reduction in left ventricular mass after weight loss in obese subjects with pre-existing left ventricular hypertrophy.[17]

To explain the discrepancy in these studies it has been suggested that the effects of weight loss on left ventricular measurements occur only if obesity is mild or of short duration.[22] However, our results show that weight loss in subjects with long term morbid obesity is associated with reduced left ventricular wall thickness and left ventricular mass. Moreover, we conclude that improvements in left ventricular structure after weight loss are related to both the magnitude of weight reduction and the initial degree of left ventricular hypertrophy.

Left ventricular chamber size is known to be larger in obese subjects than in lean ones, as a result of the volume overload that occurs with obesity.[23] In our group of obese patients the increased chamber volume did not regress significantly in conjunction with weight loss, which indicates that chamber dilatation related to obesity may be less reversible than left ventricular wall thickening.

Recent trials have shown that a high relative wall thickness (concentric left ventricular pattern) is associated with increased cardiovascular risk.[6 7] Relative wall thickness was higher in our obese subjects than in lean subjects and, because wall thickness decreased more than cavity dimension after weight loss, relative wall thickness also decreased significantly. To our knowledge, this has not been reported previously.



Mean (SEM) changes in relative wall thickness and left ventricular mass as a function of weight change (in fifths) in obese subjects. Correlation coefficients are based on all individual observations (n=63)

Table 3 Multiple regression analyses of changes in relative wall thickness and left ventricular mass on changes in weight and systolic blood pressure after adjustment for baseline values*

	Dependent variables			
	Change in relative wall thickness		Change in left ventricular mass	
Independent variables	Standardised regression coefficient	P value	Standardised regression coefficient	P value
Baseline relative wall thickness	−0.62	<0.001		
Baseline left ventricular mass			−0.68	<0.001
Baseline weight	0.17	0.10	0.31	0.004
Baseline systolic blood pressure	0.19	0.12	0.27	0.04
Change in weight	0.32	0.007	0.37	0.002
Change in systolic blood pressure	0.07	0.63	0.10	0.49
P value for model	<0.001		<0.001	
Adjusted R^2 (%)	48		47	

*Data from the obese operation and obese control groups are pooled in these analyses.

It has been suggested that hypertension in obesity is a result of complex interactions between weight related volume overload and changes in hormonal factors.[24 25] In addition to promoting hypertension, volume overload and hormonal aberrations may directly modulate myocardial structure.[26 27] Although both obesity and hypertension are associated with left ventricular hypertrophy, our results showed that weight reduction itself was a better predictor of changes in left ventricular structure than the concomitant decrease in blood pressure. Our findings thus imply that the effects of weight loss on cardiac structure may be primarily mediated by a reversal in volume overload or hormonal aberrations, or both, rather than by a reduction in blood pressure.

Limitations of the study

Before generalising the results of this study, certain limitations in the design and methods should be taken into account. Firstly, the study was neither randomised nor blinded; this was not possible for practical and ethical reasons. However, the subjects in each group were carefully matched for clinical variables, thereby improving the credibility of the results. Secondly, it was difficult to evaluate obese subjects echocardiographically, leading to missing data, especially with respect to measurements of left ventricular volumes. Nevertheless, the exclusion rate was similar in both obese groups, and patients with unacceptable readings had similar clinical characteristics to those of the remaining obese patients. Furthermore, the main effect of missing data is in cross sectional data analyses; longitudinal analyses are less sensitive. A third limitation is the problem of measuring blood pressure precisely in obese subjects, as well as the assumption that single measurements are representative of blood pressure over time. These factors could explain the relatively weak association between the changes in blood pressure and the changes in left ventricular structure observed in our study. Even so, optimal measurement techniques were used in all study subjects, and average

Key messages

- Obesity and hypertension often coexist, leading to various degrees of eccentric and concentric left ventricular hypertrophy
- These structural heart changes are in turn powerful risk factors for cardiovascular morbidity and mortality
- Weight loss is followed by a reduction in left ventricular mass and relative wall thickness
- Changes in left ventricular structure are better predicted by the weight loss than by the accompanying reduction in blood pressure
- To prevent or improve abnormal heart structure in obese people, weight control should be the primary goal and should be regarded as at least as important as regulating blood pressure

24 hour ambulatory blood pressure registrations have not been shown to correlate much more strongly with left ventricular mass than one-off blood pressure measurements.[28] Our study included an obese control group,which strengthens our conclusions.

Conclusion

We have confirmed that obese people have abnormalities of heart structure which are associated with increased cardiovascular risk, and we have shown that these structural aberrations diminish after weight loss. The regression of abnormal heart structure should be regarded as favourable, even though the question of whether it can reduce morbidity and mortality remains unanswered. One important finding in this study is that the regression in abnormalities of heart structure is better predicted by weight loss than by reduction in blood pressure. To prevent or improve abnormal heart structure in obese subjects, weight control should be the primary goal and should be regarded as being at least as important as regulating blood pressure.

We thank Lauren Lissner for her help in recruiting study subjects form the municipality of Mölndal.

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Correction

Bone density and risk of hip fracture in men and women: cross sectional analysis

Owing to an editorial error the authors' corrections to the figures were not incorporated in this paper by Chris E D H De Laet and colleagues (26 July, pp221-5). Figure 2 is corrected below; the heading of the key to figure 3 should read "Range (– 2 SD to 2 SD) of density".

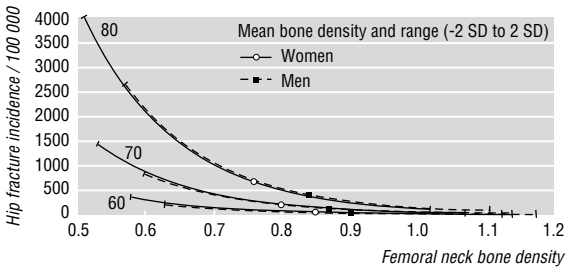

Fig 2 One year cumulative incidence of hip fracture by femoral neck bone density at ages 60, 70, and 80 in women and men