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Improvement of left ventricular morphology and function in obese subjects following a diet and exercise program

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The aim of this work was to compare left ventricular performance during weight reduction induced by either physical training and diet or diet alone.

Forty-three moderately obese subjects received a hypocaloric diet of 800 kcal/d for 4 weeks; 22 of them were also subjected to an exercise program.

By means of echocardiography, left ventricular dimensions and systolic time intervals were determined. Heart rate and blood pressure were measured at rest and during exercise.

The addition of physical training resulted in a more favourable change in weight loss (–8.3 *vs* –6.3 kg), heart rate (–14 *vs* –7 bpm), systolic (–17 *vs* –8 mm Hg), and diastolic (–11 *vs* –6 mm Hg) blood pressure. Left ventricular mass (LVM) was diminished more pronounced by combined therapy (–10.0%) as compared to diet alone (–4,7%). Changes in LVM were correlated with weight loss but not with alterations in heart rate and blood pressure. Fractional shortening and mean circumferential fiber shortening velocity did not improve significantly whereas the ratio of preejection period/left ventricular ejection time (PEPi/LVETi) was shortened in the diet and diet plus exercise group by –10.7 and –17.9%, respectively. It was concluded that exercise training in combination with a hypocaloric diet reduces left ventricular dimensions, LVM and PEPi/LVETi more distinctly than diet alone.

Keywords: left ventricular mass, left ventricular dimensions, exercise training, hypocaloric diet, blood pressure, systolic time intervals

Introduction

Obesity, especially abdominal obesity, is associated with an increased incidence of mortality. Besides coronary artery disease, sudden death, and metabolic complications, congestive heart failure is also an important cause of death, which also exists in the absence of associated diseases and may be characterized as obese cardiomyopathy.[2] In recent years many studies have demonstrated that obesity often goes along with an increased blood volume, stroke volume, and cardiac output pointing to a high output state.[1,6] Preload may be elevated as indicated by an increased pressure in the right heart and pulmonary artery bed[1] and accelerated flow across the mitral valve.[15] Endsystolic and enddiastolic diameters of the left ventricle are predominantly enlarged in grossly obese patients[18] and the thickness of the interventricular septum and the left ventricular posterior wall can be increased even in the absence of hypertension.[9] Except for obesity with excess fat mass and long duration, contractile function is usually not impaired.[2,14] The circulatory disturbances of obesity are at least partially reversible by weight loss. Left ventricular (LV) systolic and diastolic variables are improved and left ventricular mass (LVM) is diminished.[5,11] Besides caloric restriction, physical training is recommended for weight reduction, mainly for metabolic and psychological reasons; cardiac effects such as left ventricular size and contractility were not investigated. Detrimental consequences had to be assumed, since endurance athletes may have cardiac enlargement,[12] and obese children increase the left ventricular enddiastolic dimension following a jogging program.[8]

Subjects and methods

A total of 43 obese subjects, 12 women and 31 men, aged 30 to 55 years were investigated. The body mass index (BMI) varied betwen 30 and 36 kg/m^2. All patients were free of coronary and peripheral artery disease, heart failure, diabetes mellitus and major orthopedic problems. Twenty-four subjects were hypertensive according to WHO classification stage I; they did not receive any medication. Fat distribution was assessed by measurement of waist-to-hip ratio (WHR). The waist girth was measured at the level of umbilicus, and the hip girth at the level of the superior iliac spine. Men and women were classified as 'abdominal obese' with an WHR > 1.00 and > 0.85, respectively.

Treatment

The studies were carried out under supervised conditions on a metabolic ward. During a baseline period of 1 week the subjects received an isocaloric diet; weight changes were less than 2 kg. Thereafter they were randomly assigned to one of two treatment groups and informed consent was obtained. Group R received a conventional hypocaloric diet with 800 kcal/d containing approximately 90 g carbohydrates, 50 g protein, and 15 g fat.

Twenty-one subjects performed in addition an endurance training program 6 days/week (group R+T). Exercise was carried out as bicycle ergometer training, swimming, gymnastics in water, and callisthenics. The duration of exercise sessions was gradually increased from 1 h daily in the first week to 2 h in the last week. The intensity was adjusted to approximately 70% of the heart rate achieved at maximal

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work. Group R and group R+T were similar with regard to the number of hypertensives (12 *vs* 12).

Exercise test

Maximal bicycle tests were performed before and at the end of the treatment period. Starting with 50 watts, the workload was gradually increased every minute by 10 watts until exhaustion. Blood pressure measurements were made by mercury sphygmomanometry. Diastolic blood pressure referred to Korotkoff phase IV.

Echocardiography

M-mode echocardiography was performed using a Hewlett Packard Sonos 1000 ultrasonograph with a 2.5 MHz scanning head. Echocardiograms were carried out in the semirecumbent and left lateral position. Key echocardiographic data were measured below the plane of the mitral valve in the left ventricular minor axis and included the left ventricular endystolic (LVESD) and enddiastolic (LVEDD) diameter, interventricular septal thickness in diastole (IVS) and left ventricular posterior wall thickness in diastole (LVPW). Left ventricular mass (LVM) was calculated according to the formula of Troy *et al.*[16] as following: $LVM = 1.05 \times [IVS + LVPW + LVEDD)^3 - (LVEDD)^3]$. To avoid observer bias the measurements were blinded. All determinations were done in triplicate. Systolic time intervals (STI) were assessed by echocardiography and electrocardiography. Preejection period (PEPi) was measured from the QRS onset to the earliest separation of the aortic leaflets and corrected for heart rate (HR) according to Weissler[19]; left ventricular ejection time (LVETi) was measured from that point to their closure. Velocity of mean circumferential fiber shortening (Vcf) was calculated from the data above.

Statistical analysis

Results were expressed as mean ± standard deviation. Student's *t*-test was applied for paired values (comparisons before and after the treatment) and unpaired values (comparisons between groups). Pearson's linear correlation coefficient (r) was used to test the correlation betwen specific variables. P values of less than 0.05 were considered to indicate statistical significance. In order to test the reproducibility and interobserver variation of the echocardiographic data, the triplicate measurements were tested for following variables: variance, standard deviation, and sum of squared deviation. The F value was > 0.05 for each variable.

Results

Anthropometric variables (Table 1)

Daily measurements showed an almost linear loss of body weight in both groups, indicating a good compliance with treatment. Interindividual weight loss differed considerably ranging from 4.1 to 12.0 kg. Training in addition to a reducing diet resulted in a more pronounced weight reduction (–8.3 *vs* –6.5 kg; Table 1) amounting to a surplus of 26%. Body fat distribution had no influence on body weight changes.

Stress test (Table 1)

At rest, heart rate decreased in both groups, the effect being

Table 1 Anthropometric data before and after 4 weeks of weight loss. Mean ± s.d. before *vs* after treatment $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$

		Reducing diet (R) $n = 22$	*Reducing diet + training (R+T)* $n = 21$
Age (years)		45.9 ± 8.0	43.9 ± 7.0
Height (cm)		171 ± 8.1	174 ± 8.2
Body Mass Index (kg/m^2)		32.9 ± 1.9	33.1 ± 1.9
Weight (kg)	before treatment	94.6 ± 9.9	98.6 ± 11.4
	after treatment	88.1 ± 9.1***	90.3 ± 10.6**
Heart rate (beats/min)	before treatment	72 ± 11	78 ± 15
	after treatment	65 ± 10**	64 ± 12***
Systolic blood pressure at rest (mm Hg)	before treatment	163 ± 21	172 ± 18
	after treatment	155 ± 17**	155 ± 18***
Diastolic blood pressure at rest (mm Hg)	before treatment	104 ± 13	108 ± 11
	after treatment	98 ± 9.1**	97 ± 10***
Systolic blood pressure at 150 watts (mm Hg)	before treatment	239 ± 25	241 ± 18
	after treatment	226 ± 22*	211 ± 22**
Diastolic blood pressure at 150 watts (mm Hg)	before treatment	122 ± 7.3	124 ± 8.6
	after treatment	119 ± 10**	111 ± 10***
Maximal working capacity at ergometry (watts)	before treatment	125 ± 19	150 ± 21
	after treatment	133 ± 18**	171 ± 19**

Table 2 Echocardiographic data before and after 4 weeks of weight loss

		Reducing diet (R) $n = 22$	*Reducing diet + training (R+T)* $n = 21$	*R+T vs R* *P*
Left ventricular end-diastolic diameter (mm) middle of the term	before treatment	53.5 ± 4.7	52.9 ± 3.3	n.s.
	after treatment	53.2 ± 4.5	50.6 ± 3.5	< 0.05
	Difference	-0.6%	-4.3%	
	P	n.s.	< 0.001	
Left ventricular end-systolic diameter (mm)	before treatment	33.4 ± 4.1	32.7 ± 4.3	n.s.
	after treatment	32.2 ± 4.4	30.7 ± 4.0	n.s.
	Difference	-3.6%	-6.4%	
	P	< 0.05	< 0.001	
Fractional shortening (%)	before treatment	37.7 ± 4.5	38.2 ± 5.6	n.s.
	after treatment	39.6 ± 5.3	39.6 ± 4.9	n.s.
	Difference	+5.0%	+3.4%	
	P	< 0.05	n.s.	
Interventricular septal thickness (mm)	before treatment	8.8 ± 1.4	9.0 ± 1.1	n.s.
	after treatment	8.5 ± 1.4	8.8 ± 1.2	n.s.
	Difference	-4.5%	-2.2%	
	P	< 0.05	n.s.	
Left ventricle posterior wall thickness (mm)	before treatment	9.3 ± 1.2	9.8 ± 1.1	n.s.
	after treatment	9.2 ± 1.3	9.5 ± 1.0	n.s.
	Difference	-1.1%	-2.0%	
	P	n.s.	< 0.05	
Mean velocity circumferential fiber shortening (circ/sec)	before treatment	1.21 ± 0.16	1.28 ± 0.19	n.s.
	after treatment	1.21 ± 0.16	1.23 ± 0.16	n.s.
	Difference	-1.9%	-3.9%	
	P	n.s.	n.s.	
Left ventricular mass (g)	before treatment	238 ± 57	244 ± 44	n.s.
	after treatment	227 ± 48	219 ± 38	n.s.
	Difference	-4.7%	-10%	
	P	< 0.05	< 0.001	

more distinct in group R + T as compared to group R (–17.9 *vs* –9.7%). Similar results could be obtained with regard to blood pressure. Systolic blood pressure was reduced by 17 and 8 mm Hg and diastolic pressure by 11 and 6 mm Hg in group R + T and group R, respectively. During exercise (150 watts), the blood pressure was even more diminished: 30 and 13 mm Hg systolic as well as 13 and 3 mm Hg diastolic. Physical working capacity increased considerably in the training group (+ 14.0%) and slightly in the dieting-only group (+ 6.6%). Differences between hypertensive and normotensive subjects could not be detected.

Echocardiography (Table 2)

LVESD decreased almost twice as much in group R + T as compared to group R (–6.4 *vs* –3.6%). LVEDD altered little; the difference to pretreatment values (–4.3%) was only significant in group R + T. Changes in LVEDD were weekly correlated with changes in body weight ($r = 0.22$; $P < 0.05$) but not to changes in blood pressure or heart rate. Fractional shortening (FS) tended to be increased in both groups as one can assume from changes in LVEDD and LVESD.

LV wall thickness showed small reductions between 1.1 and 4.5% with variable significance concerning both IVS and LVPW. LVM, however, lessened considerably (Figure 1). The reduction in group R + T (– 25 g) was twice as high as in group R (– 11 g). The most distinct diminution was seen in hypertensive (– 27 g) and abdominal obese subjects (– 32 g) under combined therapy. Reductions in LVM were mainly due to decreases in LVEDD and LVESD in both groups and to a lesser extent to changes in IVS as well as LVPW thickness.

Systolic time intervals (Table 3)

PEPi was shortened by – 16.1 and – 9.8% in group R + T and group R, respectively. LVETi was lengthened only by combined treatment (+ 3.1%). The quotient PEPi/LVETi changed considerably in group R + T (–17.9%) and moderately in group R (– 10.7%; Figure 2). Alterations in normotensive subjects (– 22.9 and – 13.7%) were more pronounced as compared to hypertensive patients (– 14.3 and – 9.2%) in the training and dieting group, respectively. Mean Vcf did not change in either group.

Table 3 Heart rate corrected systolic time intervals before and after 4 weeks of weight loss

		Reducing diet (R) $n = 22$	*Reducing diet + training (R+T)* $n = 22$	*R+T vs R* *P*
Preejection period (msec)	before treatment	119 ± 14	118 ± 16	
	after treatment	107 ± 15	99 ± 12	
	Difference	-9.8%	-16.1%	< 0.05
	P	< 0.001	< 0.001	
Left ventricular ejection time (msec)	before treatment	425 ± 19	420 ± 17	
	after treatment	428 ± 18	433 ± 21	
	Difference	+0.8%	+3.1%	n.s.
	P	n.s.	< 0.01	
Preejection period / Left ventricular ejection time	before treatment	0.28 ± 0.04	0.28 ± 0.05	
	after treatment	0.25 ± 0.04	0.23 ± 0.03	
	Difference	-10.7%	-17.9%	
	P	< 0.001	< 0.001	< 0.001

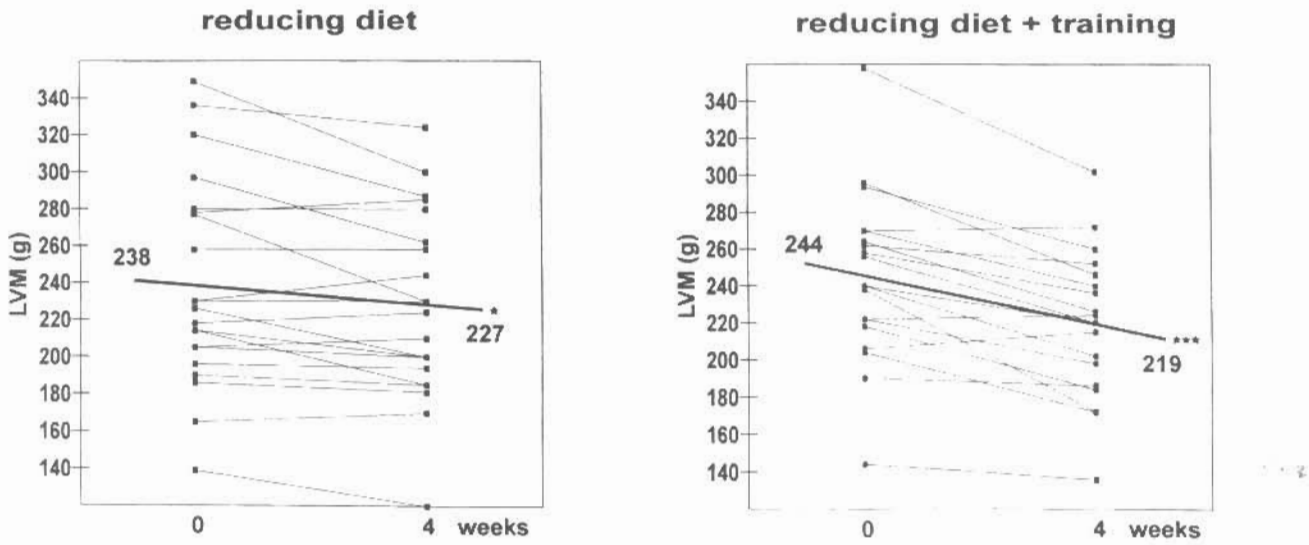

Figure 1 Echocardiographically determined left ventricular mass (LVM) before and after a 4-week period with a reducing diet (R) and training in addition (R+T) Treatment related differences: * = p < 0.05; ** 0 p < 0.01; *** = p < 0.001.

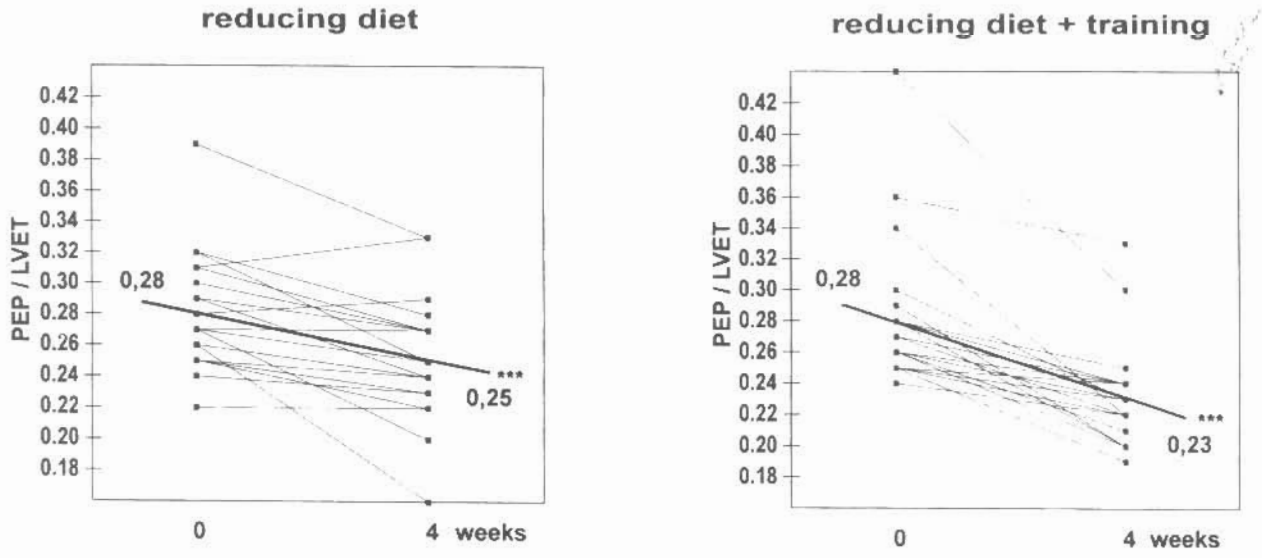

Figure 2 Echocardiographically determined quotient of preejection period (PEP) and left ventricular ejection time (LVET) before and after a 4-week period with a reducing diet (R) and training in addition (R + T).

Discussion

The present study confirms that physical training in combination with a reducing diet accelerates weight loss more effectively than dieting alone.[20] This fact and the more pronounced reduction in heart rate and blood pressure as well as an increase in working performance indicate that subjects in group R+T had attended a comprised training program.

The internal diameter of the LV tended to decrease by diet but became significantly smaller by diet combined with physical training. On the basis of radionuclide ventriculography, Ramhamadany *et al.*[13] reported similar changes after a 4-week period using a very low calorie diet. Other investigators, however, failed to demonstrate a diminution of the LV chamber size by similar weight loss[11] or even after tremendous weight reduction by gastric restriction;[3] only patients with enlarged ventricles profited from weight loss. The fact that exercise in addition to a hypocaloric diet reduces LV dimensions more effectively than a diet alone was totally unexpected. The result was surprising since athletes often develop enlarged cardiac chamber sizes.[12] The applied intensity of training in the present study was, of course, certainly far below that in competition sports. A reduction in LVEDD was seen in all subjects in group R+T. The decrease was neither correlated with the absolute LVEDD nor with changes in systolic blood pressure or HR but with changes in weight loss ($r = 0.22$; $P < 0.05$).

None of our patients had increased LV wall thickness (> 12 mm). Reductions induced by either treatment were only marginal as noticed in other studies.[13] In young overweight hypertensive patients weight loss of 8.3 kg diminished LV septal and posterior wall thickness by 14 and 11%, respectively.[11]

BMI is a strong independent predictor of LVM as shown in the Framingham Study,[9] the age-adjusted prevalence of LV hypertrophy was 15–17 fold increased in the most obese compared to the lean. In obese hypertensive patients, LVM can clearly be decreased by a reducing diet; this effect is probably more distinctive than the use of a beta-adrenergic blockade stressing the importance of weight loss.[11] Surprisingly again, the exercise group (R+T) lost more (– 25 g) LVM than the diet group (– 11 g; $P < 0.05$), the loss being mainly reflected by decreases in LV dimensions than by changes in LV wall thickness. The difference between both groups was neither due to blood pressure nor to body composition. Similar to LVEDD, changes in LVM were not correlated with changes in systolic blood pressure and HR, but with weight changes. These findings raise the question of mechanisms involved. Since sympathetic activity has been implicated in the development of LV hypertrophy, and HR decreased more pronounced in group R + T compared to group R, plasma norepinephrine may have played a role. On the other hand, weight reduction and training decrease plasma renin activity and suppress aldosterone levels which might also be relevant for the pathogenesis of LV hypertrophy.[17]

In overweight and moderately obese subjects, LV contractile function is at most slightly impaired. Morbid obese patients, however, may have a distinct reduction in ejection fraction.[1,10] In the present study, echocardiographically determined FS was normal (> 25%) in all subjects; following treatment it increased minimally. Considering the LV contractility assessed by STI, a clear improvement could be ascertained in both groups. PEP, a summation of electrical and mechanical events, which reflects mainly isovolumetric contraction time, is related to heart rate, preload and myocardial contractility.[7] Following treatment, heart rate corrected PEPi was shortened significantly with more distinct changes in the combined treated group. LVETi is usually shortened only at the stage of overt heart failure;[7] it was prolonged after 4 weeks of training. The ratio of PEPi/LVETi, probably a sensitive index of LV contractility, clearly improved in group R and even more distinctly in group R+T. These results are in agreement with studies of Caviezel *et al.*[4] investigating the effect of energy restriction.

It was remarkable that physical working capacity improved not only in training subjects but also in patients without exercise. Assuming that skeletal muscle strength and nerve function were not improved by energy restriction, adaptations in cardiac function may have been responsible for better working capacity. Therefore, at least one of the three determinants of left ventricular function (preload, afterload, contractility) has been involved. Afterload was apparently reduced as indicated by reduced systolic as well as diastolic blood pressure. Contractile function assessed by FS and STI was improved although significance was apparent only in the latter variable. Both reduction in afterload and increase in contractility were more pronounced by combined therapy as compared to diet alone. Preload was not a target of the present investigation; available results are scanty. By means of right heart catheterization, pulmonary wedge pressure was elevated in relation to obesity; it decreased following weight loss.[1] The peak early filling velocity across the mitral valve is positively correlated with obesity;[15] whether or not it may be reduced by energy restriction or physical training remains to be shown.

References

1 Alaud-din, A., Meterissian, S. Lisbona, R. MacLean, L.D., (1990): Assessment of cardiac function in patients who were morbidly obese. *Surgery* **108**, 809–820.

2 Alexander, J.K. (1991): Cardiac structure and function in obesity. *Medicographia* **13**, 9–12.

3 Alpert, M.A., Terry, B.E. Kelly, D.L. (1985): Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. *Am J Cardiol* **55**, 783–786.

4 Caviezel, F., Margonato, A. Slaviero, G. Bonetti, F., Vicedomini, G. Cattaneo, A.G. & Pozza, G. (1986): Early improvement of left ventricular function during caloric restriction in obesity. *Int J Obes* **10**, 421–426.

5 DasGupta, P.E., Ramhanmdany, E., Bridgen, G. Lahiri, A., McLean Baird, I. Raftery, E.B. (1992): Improvement in left ventricular function after rapid weight loss in obesity. *Eur Heart J* **13**, 1060–1066.

6 Devitis, de O., Fazio, S. Petitto, M. Maddalena, G. Contaldo, F. & Mancini, M. (1981): Obesity and cardiac function. *Circul* **64**, 477–482.

7 Hamada, M., Hiwada, K. & Kokubu, T. (1990): Clinical significance of systolic time intervals in hypertensive patients. *Eur Heart J* **11**, (Suppl. I): 105–113.
8 Hayashi, T., Fujino, M. Shindo, M. Hiroki, T. & Arakawa, K. (1990). Echokardiographic and elektrocardiographic measurements in obese children after an exercise program. *Int J Obes* **11**, 465–472.
9 Lauer, M.S., Anderson, K.M., Kannel, W.B. & Levy, D. (1991): The impact of obesity on left ventricular mass and geometry. *JAMA* **266**, 231–236.
10 Licata, G., Scaglione, R. Barbagallo, M. Parrinello, G., Capuana, G., Lipari, R., Merlino, G. & Ganguzza, A. (1991): Effect of obesity on left ventricular function studied by radionuclide angiocardiography *Int J Obes* **15**, 295–302.
11 MacMahon, St.W., Wilcken, D.E.L. & Macdonald, G.J. (1986): The effect of weight reduction on left ventricular mass. A randomized controlled trial in young, overweight patients. *N Engl J Med* **314**, 334–339.
12 Morganroth, J., Maron, B.J. Henry, W.L. Epstein, S.E. (1990): Comparative left ventricular dimensions in trained athletes. *Hypertension* **15**, (Suppl. I), I-153-I-6.
13 Ramhamndany, E., DasGupta, P., Brigden, G., Lahiri, A., Raftery, E.B. & McLean Baird, I. (1989): Cardiovascular changes in obese subjects on very low calorie diet. *Int J Obes* **13** (Suppl. 2), 95–99.
14 Scaglione, R., Dichiara, M.A., Indovina, A., Lipari, R., Ganguzza, A., Paririnello, G., Capuana, G., Merlino, G. & Licata, G. (1992): Left ventricular diastolic and systolic function in normotensive obese subjects: influence of degree and duration of obesity. *Eur Heart J* **13**, 738–742.
15 Stoddard, M.F., Tseuda, K., Thomas, M. Dillon, S. & Kupersmith, J. (1992): The influence of obesity on left ventricular filling and systolic function. *Am Heart J* **124**, 694–699.
16 Troy, B.L., Bombo, J. & Rackley, C.E. (1972): Measurement of left ventricular thickness and mass by echokardiography. *Circulation* **45**, 602–611.
17 Tuck, M.L., Sowers, J. Dornfeld, L. Kledzik, G. & Maxwell, M. (1981): The effect of weight reduction on blood pressure, plasma renin activity, and plasma aldosterone level in obese patients. *N Engl J Med* **304**, 930–933.
18 Warnes, C.A. & Roberts, W.C. (1984): The heart in massive (more than 300 pounds of 136 kilograms) obesity: analysis of 12 patients studied at necropsy. *Am J Cardiol* **54**, 1087–1091.
19 Weisler, A.M. (1977): Current concepts in cardiology. Systolic time intervals. *N Engl J Med* **296**, 321–324.
20 Wirth, A., Vogel, I., Schömig, A. & Schlierf, G. (1987): Metabolic effects and body fat mass changes in obese subjects on a very low-caloric diet and without intensive physical training. *Ann Nutr Metab* **31**, 378–386.