ECHOCARDIOGRAPHIC EVALUATION OF A GROUP OF OBESE WOMEN

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INTRODUCTION

Obesity is associated with hyperglycemia, hypertriglyceridemia, hypertension, elevated low density lipoprotein cholesterol (LDL) value and depressed values for high density lipoprotein cholesterol (HDL). All those factors are also associated with an increased risk of chronic heart disease (CHD). In general, women prior to menopause have a relative immunity for atherosclerotic heart disease compared to men. Menopause, however, appears to increase female susceptibility to coronary heart disease (AHA, 1980).

Also, two recent clinical studies demonstrated that, in moderately overweight patients with hypercholesterolaemia, serum cholesterol and triglyceride levels usually fell markedly when body weight was sufficiently reduced (Olefsky and Farquhar, 1974). According to Singman et al. (1980), there was a lower incidence of heart disease in two active experimental groups who ranged in age from 40-49 and 50-59 due to the reduction in serum cholesterol level, with weight reduction.

It seems reasonable that obesity might increase the hemodynamic demands of the heart and the higher prevalence of angina pectoris in overweight subjects might thus be due to both metabolic and mechanical causes. Overweight women have higher blood pressure than women in the general

population. A relationship was found between overweight women and high serum cholesterol (Noppa et al., 1978).

The use of reflected ultrasound from external transducers to determine the cardiac chamber size is a relatively recent development. Echocardiography is a diagnostic examination using high frequency sound waves to visualize the heart and great vessels (Joffe, 1976). It is a safe, noninvasive, nonionizing examination that can be repeated frequently (Kotler and Segal, 1978). Work in the field of diagnostic ultrasound began in the mid-1950's with the work of Edler and Hertz, who focused attention on mitral stenosis. Today, echocardiography has proven to be an effective diagnostic evaluation in assessing many forms of acquired and congenital heart disease (Kotler and Segal, 1978).

At the present time, no study has defined the relationship between obesity and heart performance. However, previous echocardiographic studies demonstrated a growth related change on chamber size, heart wall thickness, and heart performance as a function of either body surface area (Epstein et al., 1975) or the weight of the individual (Lundstrom, 1974).

More recently, significant correlation between left ventricular mass and lean body mass was reported by Longhurst et al (1980) but the correlation coefficient was low ($r = 0.276$). Normalizing left ventricular mass by lean

body mass revealed a significantly higher mass for long-distance runners (LDR) compared to all other groups (weight lifters (WL), heavy controls (HC), and light controls (LC)). These data suggested that static training in weight lifters induced a cardiac hypertrophy related to increased body mass, especially that part due to skeletal muscle hypertrophy.

The purpose of this study is to evaluate the heart size and function for a group of obese women by using echocardiography.

LITERATURE REVIEW

It is known that obesity is a risk factor for all types of coronary heart disease (CHD), sudden death, angina, myocardial infarction (Kannel et al., 1967). In a recent study, Dayer et al. (1975) demonstrated that the body mass index was more strongly associated with cardiovascular mortality than relative weight.

Obesity is associated with hyperglycemia, hypertriglyceridemia, hypertension, elevated low density lipoprotein (LDL) value and depressed values for high density lipoprotein (HDL). All are associated with an increased risk of chronic heart disease (C.H.D.). In general, women prior to menopause have a relative immunity for atherosclerotic heart disease compared to men. Menopause, however, appears to increase female susceptibility to coronary heart disease (AHA, 1980). Women on estrogen also have an average high density lipoprotein cholesterol about 20% greater than women not taking estrogen (Gordon et al., 1977).

The American Heart Association has identified a number of other modifiable risk factors. These risk factors include cigarette smoking, elevated blood pressure, elevated blood lipid levels, and the presence of diabetes (Gordon et al., 1977). Essential hypertension is particularly prevalent in middle-aged and elderly people, blacks, obese

people, heavy drinkers, and women who are taking oral contraceptives (Kannel and Sorlie, 1975).

Berchtold et al. (1977) investigated cardiovascular risk factors in 500 obese patients. Of these patients, 88% had one or more cardiac risk factors. The most frequent was hypertension, followed by glucose intolerance, hypertriglyceridemia and hyperuricemia. Only 12% of the patients were without additional risk factors, and these patients were younger and and less obese than the patients with risk factors. The correlation between obesity and the sum of all the risk factors was higher ($r = 0.35$) than the correlation between age and the sum of all risk factors ($r = 0.23$).

Obesity and the Heart:

Since it seems reasonable that obesity might increase the hemodynamic demands on the heart, the higher prevalence of angina pectoris in overweight subjects might thus be due to both metabolic and mechanical causes. Overweight women have higher blood pressure than women in the general population. A relationship was found between overweight women and high serum triglyceride level but not between overweight and high serum cholesterol (Noppa et al., 1978). Cholesterol is the predominant lipid constituent of the atherosclerotic lesion.

Two recent clinical studies demonstrated that in moderately overweight patients with hypercholesterolaemia,

serum cholesterol and triglyceride levels usually fall markedly when body weight was sufficiently reduced (Olefsky and Farquhar, 1974). According to Singman et al. (1980), there was a lower incidence of heart disease in the active experimental subjects who ranged in age from 40-49 and 50-59 due to the reduction in serum cholesterol level, with weight reduction. Also, Blacker et al., (1979) suggested that there is a beneficial effect of weight loss and changes in dietary lipids on the serum cholesterol of obese men with hypercholesterolaemia.

In the Framingham autopsy series, body weight was more strikingly related to left ventricular weight and thickness than to coronary atherosclerosis, and the data were considered to indicate that obesity might represent more a hemodynamic than an atherogenic stress for the circulatory system (Kannel and Gordon, 1974). However, the Framingham study also showed that coronary mortality in general and sudden death in particular were substantially increased in the obese.

Relative weight and obesity in middle-aged men do not make an independent contribution to the risk of developing coronary heart disease. It is commonly seen, however, that when fat people with hypertension reduce, the blood pressure falls, so the observed relationship between relative weight and blood pressure is presumably causal.

Still 40% of the extremely obese people do not have high blood pressure (Whyte, 1959). The effects of both obesity and blood pressure on heart performance has not been evaluated. In summary, the influence of both obesity and blood pressure on heart size and function is unknown.

Hypertrophy - A Cardiac Adaption to Stress:

The normal heart muscle grows to match the workload imposed upon the ventricle since the work can vary both in systolic pressure produced and in the volume of blood ejected; different types of growth are produced. When the muscle is normal this growth results in a constant relation between systolic pressure and the ratio of wall thickness to ventricular radius, irrespective of heart size. It is proposed that this relation can, therefore, be used to determine the progression of hypertrophy, and deviation from the relation can be used as a measure of myocardial disease (AHA, 1976). Also, the degree of left ventricular hypertrophy was expressed as the ratio of left ventricular mass to end-diastolic volume by Feild et al., (1973). He reported that patients who have a mass to volume ratio < 0.99 may have been associated with abnormally high peak systolic stress values. Graham et al. (1970) have demonstrated that mass to volume ratio can be related to systolic pressures for humans. Muscle mass does not change

with acute dilation, and mass to volume ratio can however be used as an index of hypertrophy.

Grant et al. (1965), have compared the adaptations of the left ventricle to pressure load and to a volume load in a group of 25 patients with the normal function of the left ventricle. These data suggested that adaptation to a pressure load is by concentric hypertrophy which has been defined as increase in wall thickness without chamber enlargment. Filling pressure may be raised in these normal-sized chambers. With volume loading, there is an enlargement of the ventricle and proportionate increase in wall thickness. This adaptation has been termed "eccentric hypertrophy." End-diastolic pressure may be normal in these large chambers. Eccentric hypertrophy is analogous to normal growth, the process which converts a neonatal left ventricle into an adult chamber.

Grossman et al. (1975) recently measured the wall thickness to ventricular radius ratio (t/r) and left ventricular mass to volume ratio (m/v) in patients with both pressure and volume overload caused by aortic and mitral valve lesions. They reported that a volume overload caused a three-fold increase in end-diastolic volume did not substantially change t/r ratio or m/v ratio. Whereas a pressure overload produced an increased t/r and m/v ratio that was nearly proportional to the increased ventricular

systolic pressure. Therefore, it seems reasonable that obesity which is frequently associated with hypertension should also be associated with "concentric hypertrophy" (Gordon et al., 1977).

Echocardiography:

The use of reflected ultrasound from external transducers to determine cardiac chamber size and wall thickness is a relatively recent development. Echocardiography is a diagnostic examination using high frequency sound waves to visualize the heart and great vessels (Joffe, 1976). It is a safe, noninvasive, nonionizing examination that can be repeated frequently. It also provides an excellent noninvasive technique for the evaluation of left ventricular size and function which have previously required cardiac catheterization and angiography (Kotler and Segal, 1978). Work in the field of diagnostic ultrasound began in the mid-1950's with the work of Edler and Hertz, who focused attention on mitral stenosis. Today, echocardiography has proven to be an effective diagnostic evaluation for assessing many forms of acquired and congenital heart disease (Kotler and Segal, 1978).

At the present time, no study has defined the relationship between obesity and heart performance. However, previous echocardiographic studies demonstrated a growth related change in chamber size, heart wall thickness, and

heart performance as a function of either body surface area (Epstein et al., 1975) or the weight of the individual (Lundstrom, 1974).

Epstein et al. (1975) studied 205 normal, healthy children ranging in age from 6 months to 18 years to establish normal echocardiographic measurements of valvular motion, cavity dimensions, great vessel diameters, and septal wall thickness. The data were plotted for each body surface showing the measurements at the levels of 5, 50, and 95% of the total. Left ventricular cavity measurements in systole and diastole approximately doubled as the body surface area increased from 0.3 to 1.7 m^2. The growth rate of right ventricular cavity is very similar to that of the left ventricle, but the absolute values are only about 30% as large. The right ventricular anterior wall increases in thickness by only about 0.5 mm from 6 months to 18 years. The interventricular septum increases in thickness from approximately 0.5 cm in the 6 month-old to 0.65 cm in the oldest children. The thickest of normal septum was 0.8 cm. Left ventricular posterior wall measurements were very similar to those of the septum.

Furthermore, Henry et al. (1978) reported that echocardiographic measurements of the internal dimension of the left ventricle, the left atrium, the aortic root, and the mitral E-F slope are linearly related to the cube root of

the body surface. The wall thickness of the left ventricle, however, is linearly related to the square root of the body surface area, and estimated LVM varies directly with BSA. Ejection fraction, fractional shortening percentage, and percent thickening of IVS and left ventricle free wall are independent of BSA.

Longhurst et al. (1980) studied 60 individuals including 17 competitive weight lifters (CWL), 12 competitive long-distance runners (LDR), 7 non-competitive weight lifters (AWL), 14 heavy controls (HC), and 10 light controls (LC) at supine rest with echocardiographic determination of the left ventricular mass (LVM) by the methods of Penn-Devereaux and Reichek (1977). Lean body mass (LBM) was estimated by the methods of Wilmore and Behnke (1969). Left ventricular mass was increased in the two competitive athlete groups compared to controls. The AWL had a mass intermediate between the LDR-CWL and the HC-LC groups. A significant correlation between LV mass and LBM mass was reported by Longhurst et al. (1980) but the correlation coefficient was low ($r = 0.276$). Normalizing LVM by LBM revealed a significantly higher mass for LDR compared to all other groups. These data suggested that static training in weight lifters induced a cardiac hypertrophy related to increased body mass, especially that part due to skeletal muscle hypertrophy.

In summary, the chronic cardiac structural and functional adaptation induced by obesity in women have not been elucidated. In this study, M-mode echocardiography will be employed to clarify the role of obesity and hypertension as factors that influence chronic heart performance.

METHODS

A total of 20 adult premenopausal women between 19 and 36 years of age volunteered for an electrocardiographic and echocardiographic examination. The subjects were divided into two groups on the basis of percent body fat. Ten subjects served as controls having a body composition of less than 30% fat; 10 subjects served as obese, having a body composition of more than 30% fat. No subjects had a history of cardiovascular illness and all were in good health as determined by medical history, 12-lead EKG, and an echocardiographic examination. Informed consent was obtained from all subjects.

Standing height was measured to the nearest 0.1 cm and body weight was recorded to the nearest kg on standard scale. Skin fold measurements were made on the right side of each subject at the chest, axilla, triceps, subscapula, suprailium, abdomen and thigh with a Lange Skin Fold Caliper (Cambridge, Massachusetts). The body density of the subjects was calculated from the following formula of Andrew S. Jackson, 1980:

$$BD = 1.0970-0.00046971(X_1)+0.00000056(X_1)^2-0.00012828(X_4)$$

X_1 = Sum of all seven skin folds, mm

X_4 = Age

Percent body fat was calculated according to the formula of Sire, 1956:

$$\% \text{ Fat} = \frac{4.95}{D} - 4.5 \times 100$$

Lean body mass (LBM) of the subjects was calculated according to the formula of Sloan, 1967:

LBM = BW [1-[4.95/(1.10-0.00133(TSF)-0.00131(SSF))]-4.5]

TSF = Thigh Skin Fold

SSF = Scapular Skin Fold

A routine 12-lead electrocardiogram was obtained with the subject in a supine position. Resting heart rate, the direction of mean electrical axis of the heart, the maximum net magnitude and direction of the vector of the QRS complex and T-wave, and direction of the vector loop were determined from the electrocardiogram. Resting blood pressures were measured by means of electronic sphygmomanometer with the subject supine. Mean arterial blood pressures were calculated from the formula:

MAP = 1/3(Syst Bl. Pr. - Diast. Bl. Pr.) + Diast. Bl. Pr.

A comparison of age, height, weight, body surface area, lean body mass, and percent body fat is presented in Table 1.

Standard M mode echocardiogram was recorded on each subject with an Irex System II echocardiograph unit using a 2.25 MHZ transducer focused at 3-10 cm. Signals were recorded with a fiberoptic strip chart recorder at a chart speed of 25 mm/second using a thermal processor. A lead II electrocardiograph tracing was simultaneously

recorded with the echocardiogram. The transducer was positioned near the left sternal border with the subjects in the left lateral position. Once the anterior leaflet of the mitral valve was optimally recorded in this position, the ultrasonic beam was directed superomedially to observe the aorta and the aortic valve. The beam was then angled in an infero-lateral direction until the standard recording position for the left ventricle had been reached. This was the area where the recording was made for left ventricular size, volume, and function and also for right ventricular dimension. Fragments of the anterior mitral valve leaflet and chordae were frequently observed in the left ventricular area.

The echo parameters measured and derived for the left ventricle included: interventricular septal wall thickness (IST), determined as the vertical distance from the right ventricular side of the IVS to the left ventricular side of the IVS in end-diastole; left ventricular posterior wall end-diastolic thickness (PWT), determined as the vertical distance from the epicardium of LVPW to the endocardium in end-diastole; left ventricular end-diastolic dimension, identified as the vertical distance from the endocardium of LVPW to the endocardium of the IVS in end-diastole; left ventricular end-systolic dimension, determined as the vertical distance from the endocardium of LVPW at

the peak of its anterior motion during systole to the endocardium of the IVS; left ventricular end-diastolic $(LVIDd)^3$ and end-systolic volume $(LVID_s{}^3)$; stroke volume $(SV = LVIDd^3 - LVID_s{}^3)$ ejection fraction $(EF = SV/LVIDd^3)$; total left ventricular volume $TLVV = (LVIDd + 2PWT)^3$ and the left ventricular cavity volume to mass volume ratio: $\frac{LVIDd^3}{(LVIDd + 2PWT)^3}$. Figure 1 illustrates an echogram from the standard LV area (See Appendix).

Other echo parameters included: aortic root and diastolic dimension (A_o), determined as the vertical distance from the outer edge of the anterior aortic wall to the inner edge of the posterior aortic wall; left artial dimension (LAD), determined as the greatest vertical distance between the anterior side of the posterior aortic wall and posterior left atrial wall during ventricular systole when the aorta is in its maximum anterior position, mitral valve pliability (DE), anterior mitral valve leaflet excursion (CE), velocity of initial mitral valve closure $(EF_o$ slope) and maximal diastolic mitral valve excursion (EE^-) (Feigenbaum, 1973). Representative tracings of the aortic and mitral valve areas are shown in Figures 2 and 3 (See Appendix).

Heart rate, end-diastolic septum dimension, posterior wall end-diastolic dimension, left ventricular end-systolic dimension, left ventricular end-diastolic dimension, stroke

volume, and ejection fraction were averaged from a minimum of three consecutive cardiac cycles. The Student's t-test for group data was used in comparing absolute and standardized measurements for total body weight, body surface area, percent body fat, and lean body mass between the obese group and the control group. Probability of the differences in echo parameters between the obese and control was significant when it was less than 0.05 ($P<0.05$) in these data.

RESULTS

The mean value for total body weight (TBW) of the obese subjects (99.8 ± 4.1 kg) was greater ($P<0.001$) than the total body weight of the control (60.6 ± 2.1 kg). The height of the obese women (169.3 ± 2.4 cm) was not significantly greater than that of the control women (164 ± 1.9 cm). Significant difference ($P<0.001$) was observed between the body surface area (BSA) of the obese women (2.2 ± 0.1 m^2) and that of the control women (1.7 ± 0.03 m^2). The mean value for the lean body mass (LBM) of the obese subjects (47.4 ± 2.1 kg) was not significantly greater than that of the control women (44.9 ± 1.5 kg). However, the mean value for the percent body fat (% Fat) of the obese subjects (40.7 ± 1.0%) was significantly greater ($P<0.001$) than that of the control (22.5 < 1.3%). The age of the control women (25.4 ± 1.4 yr) was not significantly less than that of the obese women (25.8 ± 1.9 yr).

Table 1 presents the subject profile for the obese and control women. The mean value (± SEM) for the resting heart rate of the control (75 ± 4 bpm) did not differ significantly from that of the obese (78.4 ± 2.8 bpm). All the subjects have normal blood pressure but the mean value for systolic blood pressure of the control (114 ± 1.4 mm Hg)

Table 1

Subject profile for obese and control women ($\bar{X} \pm$ ESM)

	Control	Obese	P*
	(n = 10)	(n = 10)	
Heart Rate (bpm)	74.8±4.2	78.4±2.8	NS
Systolic Pressure (mm Hg)	114.4±1.4	132.8±0.9	0.001
Diastolic Pressure (mm Hg)	69.9±1.9	80.8±0.7	0.001
Mean Blood Pressure (mm Hg)	84.6±1.6	98.0±0.7	0.001
Age (yrs.)	25.4±1.4	25.8±1.9	NS
Height (cm)	164.4±1.9	169.3±2.4	NS
Weight (kg)	60.6±2.1	99.8±4.1	0.001
BSA (m^2)	1.7±0.03	2.2±0.1	0.001
Lean Body Mass (kg)	44.9±1.5	47.4±2.1	NS
% Body Fat	22.5±1.3	40.7±1.0	0.001

*P = Probability of the difference between obese and control

was significantly less ($P<0.001$) than that of the obese (132.8 ± 0.9 mm Hg). The mean value for mean blood pressure of the control (84.6 ± 1.6 mm Hg) was significantly less ($P<0.001$) than that of the obese women (98.0 ± 0.7 mm Hg). Also, the diastolic blood pressure of the control (69.9 ± 1.9 mm Hg) was significantly less ($P<0.001$) than that of the obese group (80.8 ± 0.7 mm Hg).

All the subjects in this investigation had normal heart rate, normal direction for the mean electrical axis of the heart, normal magnitude for P, QRS and T waves, and normal direction for the vector loops as determined by 12 lead EKG. All subjects were in a normal sinus rhythm.

Table 2 presents the results for comparing the absolute and standardized measurements of the aortic size and left atrial dimension in control and obese women. The mean value (± SEM) for the absolute aortic size of the control women (2.3 ± 0.1 cm) was significantly less ($P<0.002$) than that of the obese women (2.6 ± 0.1 cm). Also, the absolute value for left atrial dimension (LAD) in the control group (3.1 ± 0.1 cm) was significantly less ($P<0.02$) than that of obese women (3.6 ± 0.1 cm). A significant difference ($P<0.003$) was observed in standardized absolute measurement of aortic size for BSA (Absolute/BSA) between the obese (1.2 ± 0.03 cm/m^2) and control women (1.4 ± 0.05 cm/m^2).

Table 2. Comparison of absolute and standardized measurements of the aortic size and left atrial dimension in control and obese women ($\bar{X} \pm$ SEM).

	Control	Obese	P*
Absolute			
Aorta (cm)	2.3±0.1	2.6±0.1	0.002
LAD (cm)	3.1±0.1	3.6±0.1	0.02
Absolute/BSA			
Arota (cm/m^2)	1.4±0.05	1.2±0.03	0.003
LAD (cm/m^2)	1.9±0.07	1.6±0.10	0.05
Absolute/LBM			
Aorta (10^{-2}cm/kg)	5.0±0.2	5.7±0.3	0.05
LAD (10^{-2}cm/kg)	7.0±0.3	7.7±0.5	NS
Absolute/TBW			
Aorta (10^{-2}m/kg)	3.8±0.1	2.9±0.2	0.001
LAD (10^{-2}m/kg)	5.2±0.2	3.7±0.2	0.001
Absolute/% Fat			
Aorta (cm/%)	10.2±0.6	6.9±0.3	0.001
LAD (cm/%)	14.2±0.8	8.9±0.3	0.001

*P = Probability of the difference between obese and control.

The mean value (± SEM) for (LAD/BSA) of the control subjects (1.9 ± 0.07 cm/m^2) was significantly greater ($P<0.05$) than that of the obese subjects (1.6 ± 0.10 cm/m^2).

The mean value (± SEM) for Aorta/LBM of the control subjects (5.0 ± 0.2 cm/kg) was significantly less ($P<0.05$) than that of obese subjects (5.7 ± 0.3 cm/kg). However, the mean value for LAD/LBM in control subjects (7.0 ± 0.3 cm/kg) did not differ from that of the obese subjects (7.7 ± 0.5 cm/kg).

A significant difference ($P<0.001$) was observed between the mean value (± SEM) for Aorta/TBW of control group (3.8 ± 0.1 cm/kg) and that of the obese group (2.9 ± 0.2 cm/kg). Also, the mean value (± SEM) for (LAD/TBW) of the obese (3.7 ± 0.1 cm/kg) was significantly less ($P<0.001$) than that of the control group (5.2 ± 0.2 cm/kg).

The mean value (± SEM) for Aorta/% Fat of the control (10.2 ± 0.6 cm/% Fat) was significantly greater ($P<0.001$) than that of obese (6.9 ± 0.3 cm/% Fat). Also, the mean value (± SEM) for LAD/% Fat of the control subjects (14.2 ± 0.8 cm/% Fat) was significantly greater ($P<0.001$) than that of the obese women (8.9 ± 0.3 cm/% Fat).

Table 3 compares the absolute and standardized measurements of left ventricular dimensions in control and

Table 3. Comparison of absolute and standardized measurements of left ventricular dimensions in control and obese women (X SEM).

	PWT		IVS		LVIDd		LVIDs	
	Control	Obese	Control	Obese	Control	Obese	Control	Obese
Absolute (cm)	0.6 ±0.03	0.9*** ±0.04	0.6 ±0.03	0.9*** ±0.03	4.7 ± 0.15	5.2* ± 0.19	3.2 ± 0.12	3.8* ±0.12
Absolute/BSA (cm/m^2)	0.4 ±0.02	0.4 ±0.02	0.4 ±0.02	0.4 ±0.02	2.9 ± 0.09	2.4** ± 0.10	1.9 ± 0.07	1.7* ±0.07
Absolute/LBM ($10^{-2}cm/kg$)	1.3 ±0.10	1.9*** ±0.1	1.3 ±1.0	2.0*** ±1.0	10.6 ± 5.0	11.3 ± 4.0	6.8 ± 4.0	8.0* ±3.0
Absolute/TBW ($10^{-2}cm/kg$)	1.0 ±0.1	0.9 ±0.04	1.0 ±0.1	1.0 ±0.04	8.0 ± 0.4	5.4 ± 0.3	5.3 ± 0.3	4.0*** ±0.3
Absolute/ % Fat (cm/%)	2.8* ±0.2	2.2 ±0.1	2.7 ±0.2	2.4 ±0.2	25.3 ± 3.3	13.0** ± 0.7	14.4 ± 0.9	9.4*** ±0.6

*p 0.05

**p 0.01

***p 0.001

obese women. The mean value (± SEM) for posterior wall thickness (PWT) of nonobese women (0.6 ± 0.03 cm) was significantly less ($P<0.001$) than that of obese (0.9 ± 0.4 cm). However, the mean value (± SEM) for standardized PWT for BSA (PWT/BSA) of control (0.4 ± 0.02 cm/m^2) was not significantly less than that of obese (0.4 ± 0.02 cm/m^2). Also, PWT/TBW of control (1.0 ± 0.1 cm/kg) was not significantly less than that of the obese subjects (0.9 ± 0.04 cm/kg). However, the PWT/% Fat for control subjects (2.8 ± 0.2 cm/% Fat) was significantly greater ($P<0.05$) than that of the obese women (2.2 ± 0.1 cm/% Fat). The mean value (± SEM) for PWT/LBM of control women (1.3 ± 0.10 cm/kg) was less ($P<0.001$) than that of the obese subjects (1.9 ± 0.1 cm/kg).

The mean value (± SEM) for IVS of normal subjects (0.6 ± 0.003 cm) was less ($P<0.001$) than that of the obese women (0.9 ± 0.03 cm). However, there was no significant difference for IVS/BSA between the mean value (± SEM) of the control (0.4 ± 0.02 cm) and that of the obese women (0.4 ± 0.02 cm). Also, there was no significant difference for IVS/TBW (control = 1.0 ± 0.1 cm/kg and the obese = 1.0 ± 0.4 cm/kg). The mean value (± SEM) for IVS/LBM of the nonobese (1.3 ± 1.0 cm/kg) was significantly less ($P<0.001$) than that of the obese women (2.0 ± 1.0 cm/kg).

Significant differences were observed for LVIDd,

LVIDd/BSA, LVIDd/TBW and LVIDd/ % Fat between the control and obese. The mean value (± SEM) for LVIDd of the control (4.7 ± 0.2 cm) was significantly less ($P<0.05$) than that of the heavier women (5.2 ± 0.2 cm). The LVIDd/BSA of control (2.9 ± 0.1 cm/m^2) was significantly greater ($P<0.01$) than that of the heavier (2.4 ± 0.1 cm/m^2). The LVIDd/TBW of the control (8.0 ± 0.4 cm/kg) was greater ($P<0.001$) than that of the heavier women (5.4 ± 0.3 cm/kg). Also, the LVIDd/% Fat of the lighter subjects (25.3 ± 3.3 cm/%) was significantly greater ($P<0.01$) than that of the obese (13.0 ± 0.7 cm/%). However, the LVIDd/LBM of control subjects (10.6 ± 5.0 cm/kg) was not significantly less than that of obese (11.3 ± 4.0 cm/kg).

Significant differences were observed for LVIDs, LVIDs/BSA, LVIDs/LBM, LVIDs/TBW and LVIDs/% Fat between the control and the obese. The mean value (± SEM) for LVIDs of control (3.2 ± 0.1 cm) was less ($P<0.05$) than that of obese women (3.8 ± 0.1 cm). The mean value for LVIDs/BSA of control (1.9 ± 0.1 cm/m^2) was greater ($P<0.05$) than that of the obese (1.7 ± 0.1 cm/m^2). Also, the controls had greater LVIDs/TBW and LVIDs/% Fat than the obese, and the mean values (± SEM) of the controls were 5.3 ± 0.3 cm/kg, 14.4 ± 0.9 cm/% Fat and that for the obese were 4.0 ± 0.3 cm/kg, 9.4 ± 0.6 cm/%. However, the LVIDs/LBM of control (6.8 ± 4.0 cm/kg) was less ($P<0.05$) than that of obese women (8.0 ± 2.3 cm/kg).

Significant differences ($P<0.05$) were observed for left ventricular end-diastolic volume ($LVIDd^3$) and $LVIDd^3$/LBM between the obese and the control subjects. The mean values (± SEM) for $LVIDd^3$ and $LVIDd^3$/LBM of the control were 106.7 ± 10.7 cc and 2.39 ± 0.24 cc/kg respectively, and for the obese were 149.0 ± 15.9 cc and 3.31 ± 0.2 cc/kg respectively. However, the $LVIDd^3$/BSA, $LVIDd^3$/TBW and $LVIDd^3$/% Fat of the controls were not significantly different than that of the obese women (Table 4).

The mean values (± SEM) for left ventricular end-systolic volume (32.5 ± 3.7 cc) and $LVIDs^3$/LBM (0.72 ± 0.08 cc/kg) of the control women were less ($P<0.05$) than that of the obese women (59.3 ± 10.3 cc and 1.21 ± 0.18 cc/kg). However, the mean values (± SEM) for $LVIDs^3$/BSA, $LVIDS^3$/TBW, and $LVIDs^3$/% Fat for the control (19.4 ± 2.2 cc/m^2, 0.55 ± 0.06 cc/kg, and 147.4 ± 16.3 cc/% Fat) were not significantly less than that of the obese subjects (26.0 ± 3.7 cc/m^2, 0.61 ± 0.12 cc/kg, and 148.4 ± 31.0 cc/% Fat). A difference ($P<0.05$) was observed for stroke volume (SV) standardized for % Fat (SV/% Fat) between the control and the obese, and the mean value (± SEM) of the control (336.9 ± 36.8 cc/% Fat) was greater than that of the obese (228.8 ± 22.3 cc/% Fat). However, the mean values (± SEM) for absolute SV, SV/BSA, SV/LBM

Table 4. Comparison of the absolute and standardized measurements of $LVID^3$ and SV in control and obese women ($\bar{X} \pm$ SEM).

	$LVIDd^3$ (cm^3)		$LVIDs^3$ (cm^3)		SV (cm^3)	
	C	O	C	O	C	O
Absolute (cm)	106.7 ± 10.7	149.0* ± 15.9	32.5 ± 3.7	59.3* ±10.3	74.2 ± 7.9	90.6 ± 7.8
Absolute/BSA (cm/m^2)	64.5 ± 6.0	66.9 ± 6.0	19.4 ± 2.2	26.0 ± 3.7	44.9 ± 4.6	40.8 ± 3.5
Absolute/LBM (cm/kg)	2.39 ± 0.24	3.31* ± 0.23	0.72 ± 0.08	1.21 ± 0.18	1.67 ± 0.19	1.94 ± 1.6
Absolute/TBW (cm/kg)	1.79 ± 0.18	1.49 ± 0.18	0.55 ± 0.06	0.61 ± 0.12	1.25 ± 0.14	0.93 ± 0.10
Absolute/% Fat (cm/%)	484.4 ± 48.5	377.1 ± 47.5	147.4 ± 16.3	148.4 ± 31.0	336.9* ± 36.8	228.8 ± 22.3

*p 0.05

**p 0.01

***p 0.001

and SV/TBW of the control group were 74.2 ± 7.9 cc, 44.9 ± 4.6 cc/m^2, 1.67 ± 0.19 cc/kg and 1.25 ± 0.14 cc/kg respectively. These values for the obese women were 90.6 ± 7.8 cc, 40.8 ± 3.5 cc/m^2, 1.9 ± 1.6 cc/kg and 0.93 ± 0.10 cc/kg, respectively.

The controls had a greater ($P<0.001$) cavity volume to mass volume ratio than the obese women (Table 5). The CV/MV for the control was 0.51 ± 0.02 and for the obese women was 0.4 ± 0.02.

The ejection fraction of the controls (69.3 ± 2.3) was not significantly greater than that of the obese women (61.6 ± 2.7).

Correlations were made to determine if left ventricular end-diastolic and left ventricular end-systolic dimensions were related to TBW, BSA, % Fat or lean body mass (Tables 6-9). The only significant correlation ($P = 0.010$ and $P = 0.015$) was with lean body mass. The linear coefficients of correlation of LVIDd and LVIDs with LBM were $r = 0.763$ and $r = 0.736$ respectively. Correlation of left ventricular end-diastolic, left ventricular end-systolic volumes, and stroke volume with BSA, TBW, % Fat, and LBM also revealed significant correlations with LBM. The linear coefficients of correlation for $LVIDd^3$, $LVIDs^3$, and SV with LBM were $r = 0.773$, $r = 0.715$, and $r = 0.634$ (Table 7).

Table 5. Comparison of CV/MV and EF in control and obese Women ($\bar{X} \pm$ SEM)

	Control	Obese	P*
	(n = 10)	(n = 10)	
CV/MV	0.51	0.42	0.001
	± 0.02	± 0.02	
EF	69.3	61.6	0.043
	± 2.3	± 2.7	

*P = Probability of the difference between obese and control

Significant correlation (r = 0.799) was observed between aortic root end-diastolic dimension (Ao) and body surface area (BSA) (Table 6). However, there were no significant correlations between Ao and the other parameters (LBM, TBW or % Fat) (Tables 7-9).

There was a significant correlation (r = 0.689) between left atrial dimension and TBW (Table 9). No correlation was found between LAD and LBM, % Fat or BSA (Tables 6-8).

Table 6. Correlation between echo parameters and BSA.

	Control			Obese		
	y = a+bx	r	p	y = a+bx	r	p
Ao	1.324+(0.1448)X	0.417	0.231	-0.7900+(1.150)X	0.799	0.0032
LAD	1.369+(0.08997)X	0.354	0.3156	3.379+(-0.3192)X	-0.404	0.2180
PWT	1.485+(0.2750)X	0.263	0.4632	1.153+(1.235)X	0.464	0.1505
IVS	1.599+(0.08695)X	0.077	0.8322	0.7793+(1.625)X	0.468	0.1467
LVIDd	1.326+(0.06900)X	0.330	0.3522	1.537+(0.1343)X	0.270	0.4228
LVIDs	1.331+(0.1012)X	0.385	0.2726	1.462+(0.2052)X	0.420	0.1986
$LVIDd^3$	1.544+(0.0009915)X	0.339	0.3385	1.944+(0.001990)X	0.338	0.3096
$LVIDs^3$	1.543+(0.003309)X	0.393	0.2606	1.995+(0.004153)X	0.458	0.1566
SV	1.571+(0.001068)X	0.272	0.4480	2.152+(0.0009874)X	0.083	0.8094
EF	1.785 (-0.001946)X	-0.141	0.6981	3.327 (-0.01761)X	-0.503	0.1147

Table 7. Correlation between echo parameters and LBM.

	Control			Obese		
	y = a+bx	r	p	y = a+bx	r	p
Ao	34.87+(4.441)X	0.246	0.4613	11.37+(13.45)X	0.401	0.2513
LAD	32.92+(3.826)X	0.311	0.3820	65.16+(-4.977)X	-0.308	0.3872
PWT	32.79+(20.12)X	0.397	0.2558	41.64+(6.349)X	0.116	0.7490
IVS	35.15+(16.46)X	0.302	0.3968	32.64+(16.10)X	0.216	0.5495
LVIDd	38.73+(1.305)X	0.129	0.7229	7.260+(7.610)X	0.763	0.0103
LVIDs	29.17+(4.980)X	0.391	0.2641	20.06+(7.185)X	0.736	0.0153
$LVIDd^3$	42.94+(0.01793)X	0.127	0.7269	33.43+(0.09142)X	0.773	0.0087
$LVIDs^3$	39.92+(0.1520)X	0.373	0.2880	39.61+(0.1294)X	0.715	0.0202
SV	44.91+(-0.0007178)X	-0.004	0.9918	32.96+(0.1553)X	0.634	0.0490
EF	64.97 (-0.2901)X	-0.433	0.2109	65.28 (-0.2888)X	-0.402	0.2500

Table 8. Correlation between echo parameters and % fat.

	Control			Obese		
	y = a+bx	r	p	y = a+bx	r	p
Ao	11.88+(4.267)X	0.025	0.5701	32.86+(2.918)X	0.172	0.6345
LAD	7.084+(4.941)X	0.474	0.1668	30.84+(2.733)X	0.335	0.3447
PWT	27.30+(-8.00)X	0.186	0.6064	31.23+(10.58)X	0.384	0.2735
IVS	26.78+(-7.246)X	-0.157	0.6656	34.25+(7.029)X	0.186	0.6060
LVIDd	17.28+(1.110)X	0.129	0.7220	43.97+(-0.6315)X	-0.125	0.7300
LVIDs	19.71+(0.8854)X	0.083	0.8219	43.97+(-0.8278)X	-0.168	0.6431
$LVIDd^3$	22.03+(0.004576)X	0.044	0.9033	42.05+(-0.009239)X	-0.155	0.6696
$LVIDs^3$	20.99+(0.04637)X	0.134	0.7114	41.81+(-0.01950)X	-0.213	0.5539
SV	20.80+(0.02296)X	0.142	0.6952	41.02+(-0.003968)X	-0.032	0.9299
EF	19.43+(0.04426)X	0.078	0.8304	36.88 (0.06059)X	00.167	0.6449

Table 9. Correlation between echo parameters and TBW.

	Control			Obese		
	y = a+bx	r	p	y = a+bx	r	p
Ao	65.32+(-3.412)X	-0.457	0.1838	63.52+(13.76)X	0.218	0.5188
LAD	23.09+(11.87)X	0.689	0.0275	62.08+(10.58)X	0.306	0.3599
PWT	52.42+(12.87)X	0.182	0.6158	76.20+(26.75)X	0.230	0.4965
IVS	57.88+(3.826)X	0.050	0.8907	93.04+(7.500)X	0.049	0.8854
LVIDd	49.12+(2.345)X	0.165	0.6481	56.50+(8.254)X	0.379	0.2508
LVIDs	54.26+(1.866)X	0.105	0.7737	82.34+(4.592)X	0.215	0.5260
$LVIDd^3$	57.60+(0.03222)X	0.162	0.6541	87.15+(0.08432)X	0.327	0.3260
$LVIDs^3$	57.68+(0.07576)X	0.133	0.7144	96.34+(0.05824)X	0.147	0.6666
SV	56.65+(0.04567)X	0.170	0.6396	77.48+(0.2464)x	0.471	0.1440
EF	55.75 (0.06338)X	0.068	0.8527	88.98 (0.1754)X	0.115	0.7372

DISCUSSION

Previous echocardiographic studies demonstrated a growth related change in chamber size, heart wall thickness, and heart performance as a function of either body surface area (Epstein et al., 1975) or the weight of the individual (Lundstrom, 1974). More recently Longhurst et al. (1980) reported a significant correlation between left ventricular mass and lean body mass. His data suggested that static training in weight lifters induced a cardiac hypertrophy related to increased body mass, especially that part due to skeletal muscle hypertrophy. Correlations were found between left ventricular mass and lean body mass, but no correlations were found between left ventricular mass and other parameters (body surface area, total body weight or percent body fat).

The chronic cardiac structural and functional adaptations induced by obesity in women have not been elucidated. By using M-mode echocardiography this study reports the cardiac adaptations induced by obesity.

The obese women in this study had a greater total body weight and a greater body surface area than the control subjects. However, the height and the lean body mass of the obese women were not significantly greater than that of the control women. Nor was the heart rate of these

women significantly greater than that of the control subjects. However, the obese women in this study had greater systolic, diastolic and mean blood pressures.

This study reports that the absolute measurements of the aortic root, left atrial dimension, left ventricular end-diastolic dimension, left ventricular end-systolic dimension, posterior wall thickness, interventricular septal wall thickness, left ventricular end-diastolic volume and left ventricular end-systolic volume of the obese women were greater than that of the control women.

Epstein et al., 1975, and Lundstrom, 1974, suggested a correlation in the adult population in both aortic size and left atrial dimension when compared with body surface area or total body weight. This study indicates that no correlation is present for these parameters and body surface area in control women. However, a positive correlation was found between aortic size and body surface area in the obese women. Also, a positive correlation was observed between left atrial dimension and total body weight in control women. Further discrepancy between this study and previous studies was found when the control aortic size and left atrial dimension were standardized. This suggests that either the correlation of these parameters for the women studied differ from the other population previously

reported or that the population studied was too small to permit a comparison.

The mean value for left ventricular end-diastolic dimension standardized for lean body mass of the control was not significantly less than that of the obese. This study suggests positive correlations between lean body mass and these parameters, left ventricular end-diastolic dimension, left ventricular end-systolic dimension, left ventricular end-diastolic volume, left ventricular end-systolic volume and stroke volume. This data generally agree with the data recently reported by Longhurst et al., 1980. Furthermore, this study suggests a correlation in both posterior wall thickness and interventricular septal wall thickness when compared with body surface area or total body weight.

In 1970 Graham et al., have demonstrated that mass to volume ratio can be related to systolic pressures in humans. His data suggested that mass to volume ratio can however be used as an index of hypertrophy. The obese women in this study had higher systolic, diastolic and mean blood pressures than the control subjects, and had a greater mass volume to cavity volume ratio than the control subjects. This data suggest that the symmetrical increase in left ventricular wall thickness without chamber

enlargement is an adaptation to pressure load. This adaptation has been termed "concentric hypertrophy." Therefore, the primary cardiac adaptation demonstrated in the obese women is concentric hypertrophy.

In summary, this study has shown a correlation between left ventricular dimensions and lean body mass in the obese women, but no correlations were found between left ventricular dimensions and the other parameters (body surface area, total body weight or percent body fat). Also, this data suggest that the primary cardiac adaptation demonstrated in the obese women is concentric hypertrophy. Therefore, when echocardiographic studies are performed on obese women, the influence of blood pressure and body size must be considered when comparing data with that of the normal subjects. Further study on normotensive obese women will be necessary to confirm these observations and to characterize the possible influence of high blood pressure on chronic ventricular performance.

APPENDIX

Table 10(A). Individual subject data for normal population description

Subject No.	Age (yrs)	Height (cm)	Weight (kg)	LBM (kg)	B.S.A. (m^2)	Bl. Pr. (mm Hg)	H.R. (bpm)
1	27	170.5	58.8	48.8	1.68	115/75	70
2	30	168.0	72.9	53.3	1.83	108/65	85
3	19	159.0	56.6	42.4	1.58	110/68	86
4	21	168.0	57.7	46.6	1.65	118/70	88
5	27	160.0	67.3	43.6	1.68	115/70	70
6	22	168.0	67.1	48.9	1.76	118/68	71
7	31	170.5	57.9	43	1.68	110/58	79
8	24	162.8	55.5	44.7	1.58	120/80	79
9	31	151.3	55.0	36.3	1.50	120/75	73
10	22	166.0	51.4	41	1.56	110/70	67

Table 10(B). Individual subject data for normal population description.

Subject No.	Age (yrs)	Height (cm)	Weight (kg)	LBM (kg)	B.S.A. (m^2)	Bl. Pr. (mm Hg)	H.R. (bpm)
1	36	170.5	96.4	56.8	2.7	130/80	68
2	21	168.5	105.5	48.9	2.14	136/84	54
3	19	155.5	80.2	36.4	1.79	133/80	83
4	28	174	102.3	46.5	2.17	135/84	66
5	27	185.5	113.4	56.1	2.36	135/82	93
6	28	160	76.8	44.6	1.8	128/78	81
7	21	168.7	92.3	52.9	2.3	135/80	66
8	35	174.5	92.9	44.3	2.8	130/80	80
9	20	164.5	115.9	45.8	2.19	132/78	63
10	23	171	117.5	40.6	2.26	134/82	94

Table 11(A). Individual subject data for percent body fat calculation.

Subject No.	Triceps (mm)	Axilla (mm)	Chest (mm)	Scapula (mm)	Abdomen (mm)	Iliac (mm)	Thigh (mm)	% Fat (%)
1	13.0	8.2	7.2	10.2	7.7	13.8	20.0	17.2
2	21.6	14	16.0	11.4	22.0	22.0	35.5	27
3	15.9	6.2	5.0	10.0	7.0	9.2	33.8	18
4	14.1	8.5	8.9	9.5	16.5	8.5	24.5	18.6
5	22.1	10.2	15.2	19.1	14.2	9.2	41.0	25.2
6	8.6	20.4	12.8	20.0	31.0	15.2	27.2	25.6
7	21.0	21.0	9.0	9.0	16.5	9.3	36	24
8	11.0	12.4	8.0	14.2	22.0	8.6	20.2	24.1
9	23.4	13.5	8.2	18.6	25.4	15.1	39.8	27.3
10	13.5	9.6	8.8	8.6	12.5	7.4	27	18.1

Table 11(B). Individual subject data for percent body fat calculation.

Subject No.	Triceps (mm)	Axilla (mm)	Chest (mm)	Scapula (mm)	Abdomen (mm)	Iliac (mm)	Thigh (mm)	% Fat (%)
1	19.7	22.3	22.7	36.7	47.3	36	33	37
2	35	32.3	31.3	44	40.3	43	44.7	41
3	31.7	41.7	31	35	40.7	43	55	39
4	35.3	36	45	39.3	42	45.3	50.7	43
5	47	41.3	40	33	50.7	48	51	44
6	22.7	29.7	32	24	34	30	46.7	36.5
7	26	15.7	25.3	29	40	40	43	36
8	43.7	32	30.3	36.7	45	42.3	50	42
9	34.3	36.7	40.3	43.7	49	41	55	43
10	35.7	45.7	44	48.7	54	50	57	45

Table 12. Individual subject data for aorta and LAD.

Subject No.	Aorta (cm)		LAD (cm)	
	C	O	C	O
1	1.7	2.9	3.0	3.5
2	2.7	2.6	3.7	3.5
3	2.5	2.5	2.9	3.5
4	2.3	2.7	3.1	4.2
5	2.5	2.7	3.7	3.3
6	2.2	2.3	3.2	3.7
7	2.3	2.8	2.4	3.3
8	2.2	2.9	3.0	2.9
9	2.0	2.5	3.3	3.8
10	2.1	2.8	2.9	4.2

Table 13. Individual subject data for IVS and PWT.

Subject No.	IVS (cm)		PWT (cm)	
	C	O	C	O
1	0.6	1.0	0.6	1.0
2	0.6	1.0	0.7	1.0
3	0.7	0.9	0.7	0.9
4	0.5	1.0	0.5	1.0
5	0.5	0.9	0.5	0.9
6	0.7	0.8	0.7	0.7
7	0.5	0.8	0.5	0.7
8	0.7	1.0	0.6	1.0
9	0.5	0.9	0.5	0.9
10	0.6	0.8	0.7	0.8

Table 14. Individual subject data for LVIDd, LVIDs, $LVIDd^3$ and $LVIDs^3$.

Subject No.	LVIDd (cm)		LVIDs (cm)		$LVIDd^3$ (cm^3)		$LVIDs^3$ (cm^3)	
	C	O	C	O	C	O	C	O
1	4.2	6.4	3.1	5.3	74.2	262.4	29.8	148.9
2	5.3	5.4	3.7	3.9	148.9	157.5	50.7	59.3
3	4.7	4.3	2.8	3.1	103.9	79.5	22.0	29.8
4	4.5	4.8	3.0	3.2	91.1	110.6	27.0	32.8
5	4.7	5.9	2.8	4.5	103.8	205.4	22.0	91.1
6	4.3	5.3	2.9	3.8	79.5	148.9	24.4	54.9
7	5.6	5.4	3.7	3.5	175.6	157.5	50.6	42.9
8	4.5	4.3	3.4	3.3	91.1	79.5	39.3	35.9
9	4.2	5.3	2.7	3.5	74.2	148.9	19.7	42.9
10	5.0	5.5	3.4	3.8	125.0	166.4	39.3	54.9

Table 15. Individual subject data for SV, EF and CV/MV.

Subject No.	SV (cm^3)		EF		CV/MV	
	C	O	C	O	C	O
1	44.3	113.3	59.7	43.2	0.47	0.44
2	98.2	98.2	66.0	62.3	0.50	0.38
3	81.8	49.7	78.8	62.5	0.46	0.35
4	64.1	77.8	70.4	70.4	0.55	0.35
5	81.8	114.3	78.8	55.6	0.56	0.45
6	55.1	94.0	68.0	63.1	0.43	0.49
7	125.0	114.6	71.2	72.8	0.61	0.50
8	51.8	43.6	56.9	54.8	0.50	0.32
9	54.4	106.0	73.3	71.2	0.53	0.42
10	85.7	111.5	68.8	67.0	0.48	0.45

Table 16. Individual subject data for aorta and LAD standardized for BSA.

Subject No.	A_o/BSA (cm/m^2)		LAD/BSA (cm/m^2)	
	C	O	C	O
1	1.0	1.1	1.8	1.3
2	1.5	1.2	2.0	1.6
3	1.6	1.4	1.8	2.0
4	1.4	1.2	1.9	1.9
5	1.5	1.1	2.2	1.4
6	1.3	1.3	1.8	2.1
7	1.4	1.2	1.4	1.4
8	1.4	1.0	1.9	1.0
9	1.3	1.1	2.2	1.7
10	1.4	1.2	1.9	1.9

Table 17. Individual subject data for IVS and PWT standardized for BSA.

Subject No.	IVS/BSA (cm/m^2)		PWT/BSA (cm/m^2)	
	C	O	C	O
1	0.36	0.37	0.36	0.37
2	0.33	0.47	0.38	0.47
3	0.44	0.50	0.44	0.50
4	0.30	0.46	0.30	0.46
5	0.30	0.38	0.30	0.38
6	0.40	0.44	0.40	0.39
7	0.30	0.35	0.30	0.30
8	0.44	0.36	0.38	0.36
9	0.33	0.41	0.33	0.41
10	0.39	0.35	0.45	0.35

Table 18. Individual subject data for LVIDd, LVIDs, $LVIDd^3$, $LVIDs^3$ and SV standardized for BSA.

Subject No.	LVIDd/BSA (cm/m^2)		LVIDs/BSA (cm/m^2)		$LVIDd^3$/BSA (cm^3/m^2)		$LVIDs^3$/BSA (cm^3/m^2)		SV/BSA (cm^3/m^2)	
	C	O	C	O	C	O	C	O	C	O
1	2.5	2.4	1.9	2.0	44.2	97.1	17.7	55.1	26.4	42
2	2.9	2.5	2.0	1.8	81.4	73.6	27.7	27.7	53.7	45.9
3	3.0	2.4	1.8	1.7	65.7	44.4	12.0	16.6	71.8	27.8
4	2.7	2.2	1.8	1.5	55.2	51.0	16.4	15.1	38.9	35.9
5	2.8	2.5	1.7	1.9	61.8	87.0	13.1	38.6	48.7	48.4
6	2.4	2.9	1.7	2.0	45.2	82.7	13.9	30.5	31.3	52.2
7	3.3	2.3	2.2	1.5	104.5	68.5	30.1	18.6	74.4	49.8
8	2.9	1.5	2.2	1.2	57.7	28.4	24.9	12.8	32.8	15.2
9	2.8	2.4	1.8	1.6	49.5	68	13.1	19.6	36.3	48.4
10	3.2	2.4	2.2	1.7	80.1	73.6	25.2	24.3	54.9	49.3

Table 19. Individual subject data for aorta and LAD standardized for LBM.

Subject No.	Aorta/LBM (cm/kg)		LAD/LBM (cm/kg)	
	C	O	C	O
1	0.035	0.05	0.061	0.062
2	0.051	0.053	0.069	0.072
3	0.059	0.069	0.068	0.096
4	0.049	0.058	0.067	0.090
5	0.057	0.048	0.085	0.059
6	0.045	0.052	0.065	0.083
7	0.053	0.053	0.056	0.062
8	0.049	0.065	0.067	0.065
9	0.055	0.055	0.091	0.083
10	0.051	0.069	0.071	0.010

Table 20. Individual subject data for IVS and PWT standardized for LBM.

Subject No.	IVS/LBM (cm/kg)		PWT/LBM (cm/kg)	
	C	O	C	O
1	0.012	0.018	0.012	0.018
2	0.011	0.020	0.013	0.020
3	0.017	0.025	0.017	0.025
4	0.011	0.022	0.011	0.022
5	0.011	0.016	0.011	0.016
6	0.014	0.018	0.014	0.016
7	0.012	0.015	0.012	0.013
8	0.016	0.023	0.013	0.023
9	0.014	0.020	0.014	0.020
10	0.015	0.020	0.017	0.020

Table 21. Individual subject data for LVIDd, LVIDs, $LVIDd^3$, $LVIDs^3$, and SV standardized for LBM.

Subject No.	LVIDd/LBM (cm/kg)		LVIDs/LBM (cm/kg)		$LVIDd^3$/LBM (cm^3/kg)		$LVIDs^3$/LBM (cm^3/kg)		SV/LBM (cm^3/kg)	
	C	O	C	O	C	O	C	O	C	O
1	0.086	0.11	0.064	0.093	1.52	4.62	0.61	2.62	0.91	1.99
2	0.099	0.11	0.069	0.080	2.79	3.22	0.95	1.21	1.84	2.01
3	0.111	0.12	0.066	0.085	2.45	2.18	0.52	0.82	1.93	1.37
4	0.097	0.10	0.064	0.069	1.96	2.38	0.58	0.70	1.38	1.67
5	0.108	0.10	0.064	0.080	2.38	3.66	0.51	1.62	1.88	2.04
6	0.088	0.12	0.059	0.085	1.63	3.34	0.50	1.23	1.13	2.11
7	0.130	0.10	0.086	0.066	4.08	2.98	1.18	0.81	2.91	2.17
8	0.101	0.097	0.076	0.074	2.04	3.36	0.88	0.81	1.16	0.98
9	0.116	0.12	0.074	0.076	2.04	3.25	0.54	0.94	1.5	2.3
10	0.122	0.14	0.083	0.094	3.05	4.1	0.96	1.35	2.09	2.75

Table 22. Individual subject data for aorta and LAD standardized for TBW.

Subject No.	Aorta/TBW (cm/kg)		(LAD/TBW (cm/kg)	
	C	O	C	O
1	0.029	0.030	0.051	0.036
2	0.037	0.025	0.051	0.033
3	0.044	0.031	0.051	0.044
4	0.040	0.026	0.054	0.042
5	0.037	0.024	0.055	0.029
6	0.033	0.030	0.048	0.048
7	0.040	0.03	0.041	0.036
8	0.040	0.03	0.054	0.031
9	0.036	0.02	0.060	0.033
10	0.041	0.02	0.056	0.036

Table 23. Individual subject data for IVS and PWT standardized for TBW.

Subject No.	IVS/TBW (cm/kg)		PWT/TBW (cm/kg)	
	C	O	C	O
1	0.010	0.010	0.010	0.010
2	0.008	0.009	0.010	0.009
3	0.012	0.01	0.012	0.010
4	0.009	0.01	0.009	0.01
5	0.007	0.008	0.007	0.008
6	0.010	0.009	0.010	0.010
7	0.009	0.008	0.009	0.009
8	0.013	0.011	0.011	0.011
9	0.009	0.008	0.009	0.008
10	0.012	0.007	0.014	0.007

Table 24. Individual subject data for LVID, LVIDs, $LVIDd^3$, $LVIDs^3$ and SV standardized for TBW.

Subject No.	LVIDd/TBW (cm/kg)		LVIDs/TBW (cm/kg)		$LVIDd^3$/TBW (cm^3/kg)		$LVIDs^3$/TBW (cm^3/kg)		SV/TBW (cm^3/kg)	
	C	O	C	O	C	O	C	O	C	O
1	0.071	0.066	0.053	0.055	1.262	2.72	0.507	1.55	0.753	1.17
2	0.073	0.051	0.051	0.037	2.043	1.48	0.695	0.56	1.347	0.92
3	0.083	0.054	0.049	0.039	1.827	0.99	0.387	0.37	1.44	0.62
4	0.078	0.047	0.052	0.031	1.579	1.08	0.468	0.32	1.129	0.78
5	0.070	0.052	0.042	0.040	1.542	1.81	0.327	0.81	1.215	1.01
6	0.064	0.069	0.043	0.049	1.185	1.94	0.364	0.71	0.819	1.22
7	0.097	0.059	0.064	0.038	3.033	1.70	0.874	0.46	2.159	1.24
8	0.081	0.046	0.061	0.036	1.641	0.86	0.708	0.39	0.933	0.462
9	0.076	0.046	0.049	0.030	1.349	1.29	0.358	0.41	0.989	0.916
10	0.097	0.047	0.066	0.032	2.432	0.99	0.765	0.47	1.677	0.949

Table 25. Individual subject data for aorta and LAD standardized for % body fat.

Subject No.	Aorta/% Fat (cm/%)		LAD/% Fat (cm/%)	
	C	O	C	O
1	9.9	7.8	17.4	9.5
2	10.0	6.3	13.7	8.6
3	13.9	6.4	16.11	9.0
4	12.4	6.3	16.7	9.8
5	9.9	6.1	14.7	7.5
6	8.6	8.8	12.5	10.1
7	9.6	7.8	10.0	9.2
8	9.1	6.9	12.5	6.9
9	7.3	6.5	12.1	8.8
10	11.6	6.2	16.0	9.3

Table 26. Individual subject data for IVS and PWT standardized for % body fat.

Subject No.	IVS/% Fat (cm/%)		PWT/% Fat (cm/%)	
	C	O	C	O
1	3.5	2.7	3.5	2.7
2	2.2	2.4	2.6	2.4
3	3.9	2.3	3.9	2.3
4	2.7	2.3	2.7	2.3
5	2.0	2.1	2.0	2.1
6	2.7	2.2	2.7	1.9
7	2.1	2.2	2.1	1.9
8	2.9	3.8	2.5	2.4
9	1.8	2.1	1.8	2.1
10	3.3	1.8	3.9	1.8

Table 27. Individual subject data for LVIDd, LVIDs, $LVIDd^3$, $LVIDs^3$, and SV standardized for % body fat.

Subject No.	LVIDd/% fat (cm/%)		LVIDds/% fat (cm/%)		$LVIDd^3$/% fat (cm^3/%)		$LVIDs^3$/% fat (cm^3/%)		SV/% fat (cm^3/%)	
	C	O	C	O	C	O	C	O	C	O
1	24.4	17.3	18.0	14.3	431.4	708.4	173.3	402.4	257.6	306.2
2	19.6	13.2	13.7	9.5	551.5	384.2	187.8	144.6	263.7	239.5
3	26.1	11.0	15.6	8.0	576.7	203.9	122.2	76.4	454.4	127.4
4	24.2	11.2	16.1	7.4	489.8	257.2	145.2	76.3	344.6	180.9
5	18.7	13.4	11.1	10.2	411.9	466.8	87.3	207.1	324.6	260.2
6	16.8	14.5	11.3	10.4	310.6	408.0	95.3	150.4	215.2	257.5
7	23.3	15.0	15.4	9.7	731.7	437.5	210.8	119.2	520.8	318.3
8	18.7	10.2	14.1	7.9	378.0	189.3	163.1	85.5	214.9	103.8
9	53.4	12.3	9.9	8.1	271.8	346.3	72.2	99.8	199.3	246.5
10	27.6	12.2	18.8	8.4	690.6	369.8	217.1	122.0	473.5	247.8

Figure 1. Left ventricular area .

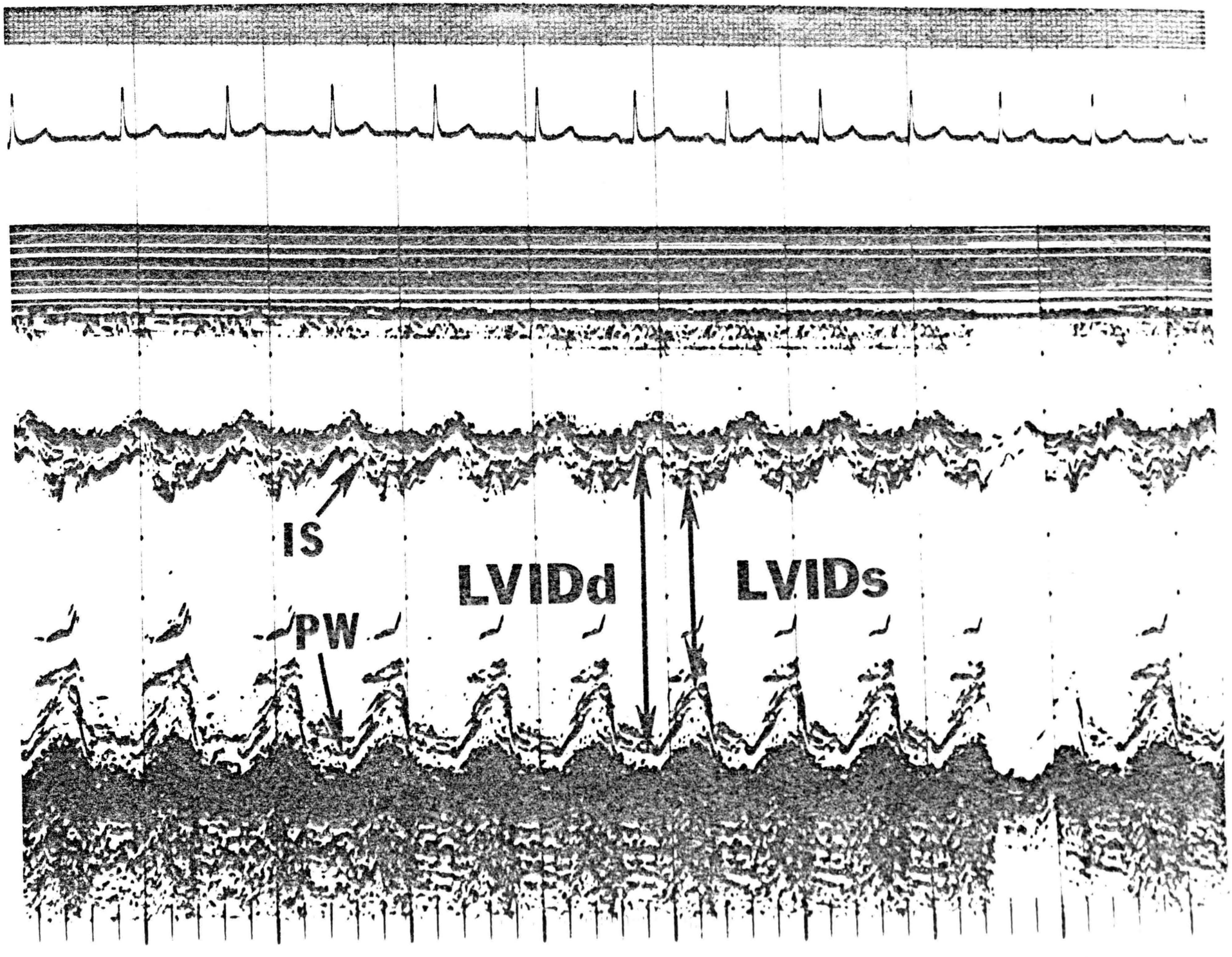

IS
LVIDd
LVIDs
PW

Figure 2. Aortic area.

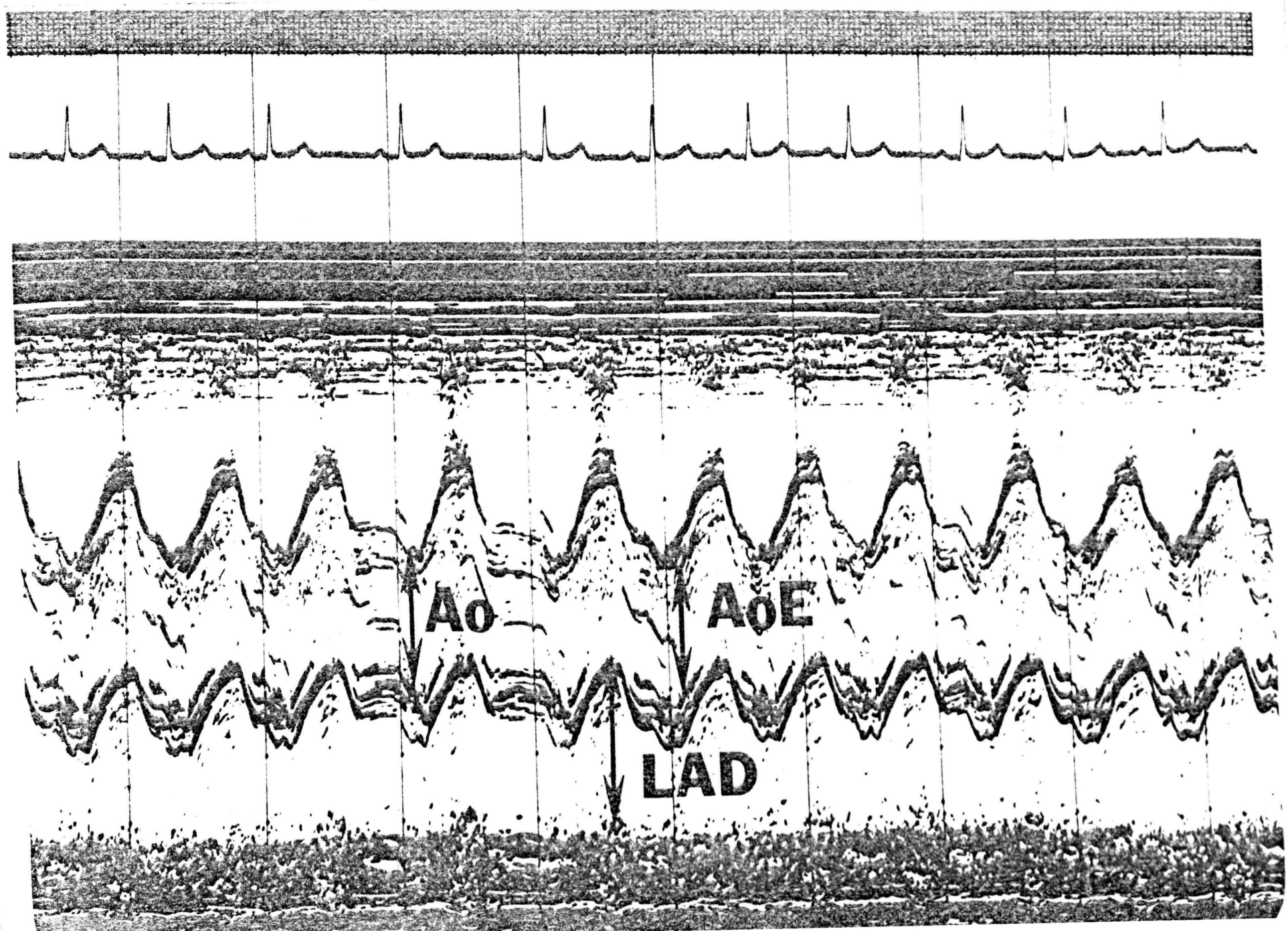
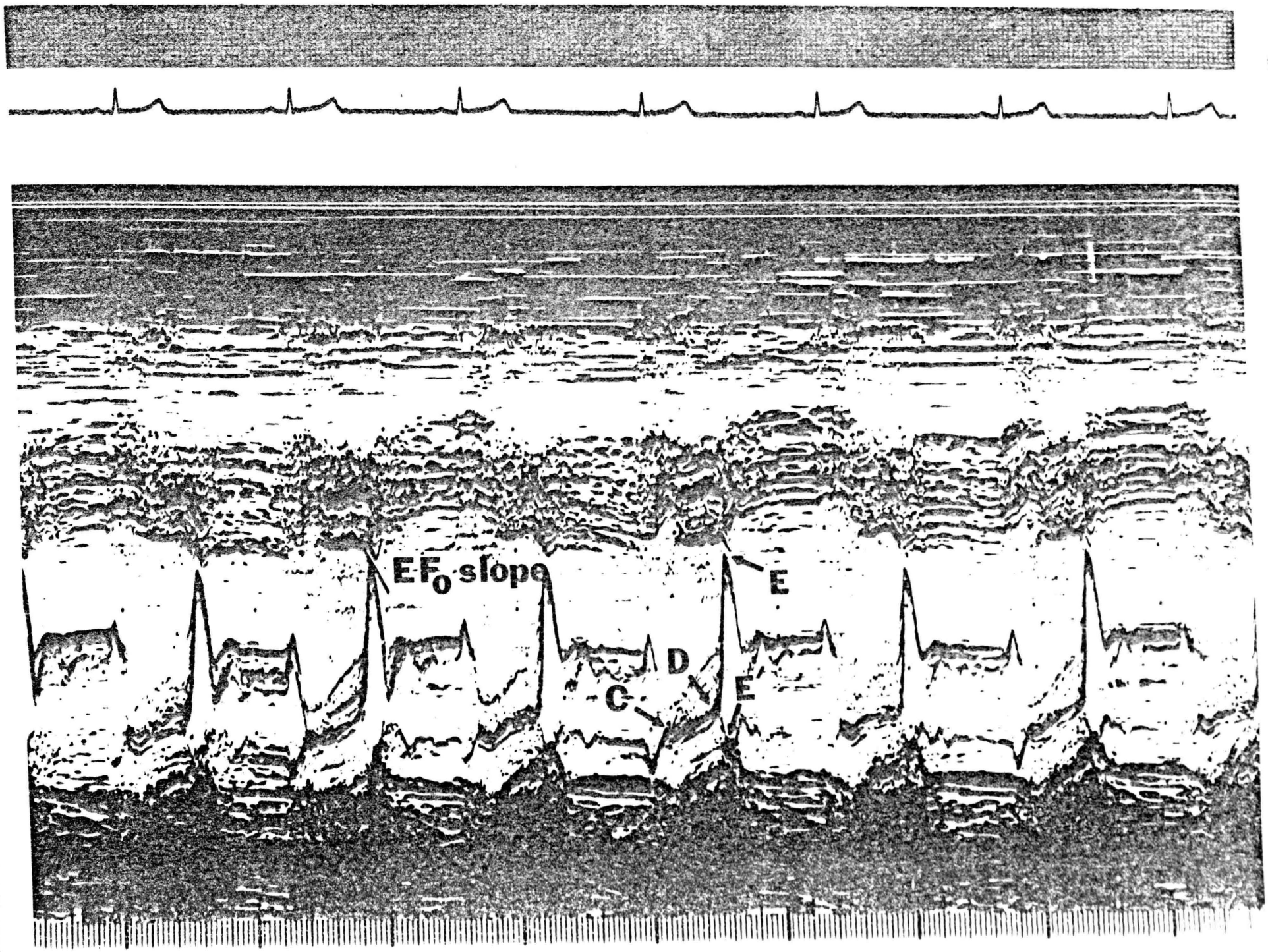

Ao
AoE
LAD

Figure 3. Mitral valve area.


EF$_0$ slope
E
D
C
E

REFERENCES

American Heart Association. 1980. Risk Factors and Coronary Disease. Circulation. Vol. 62, pp. 449A-455A.

Andrew, S. J., M. L. Pollock and A. Ward. 1980. Generalized Equations for Predicting Body Density of Women. Med. and Sci. Sports Exer. Vol. 12(3), pp. 175-182.

Berchtold, P., M. Berger, E. Greiser, M. Dohse, K. Irmscher, F. A. Gries and H. Zimmerman. 1977. Cardiovascular Risk Factors in Gross Obesity. Int. J. Obes. Vol. 1, pp. 219-229.

Blacker, R. B., B. Leelarthaepin, C. A. McGilchrist, A. J. Palmer and J. M. Woodhill. 1979. The Synergistic Effect of Weight Loss and Changes in Dietary Lipids on the Serum Cholesterol of Obese Men with Hypercholesterolemia; Implications for Prevention of Coronary Heart Disease. Aust. N. Z. J. Med. Vol. 9, pp. 521-529.

Dayer, A. R., J. Stamler, D. M. Berkson and H. A. Lindberg. 1975. Relationship of Relative Weight and Body Mass Index to 14-Year Mortality in the Chicago People Gass Company Study. J. Chronic. Dis. Vol. 28, p. 109.

Devereaux, P. and M. Reichek. 1977. Echocardiographic Determination of Left Ventricular Mass in Man. Anatomic Validation of the Method. Circulation. Vol. 55, pp. 613-618.

Epstein, M. L., S. J. Goldberg, H. D. Allen, L. Konecke and J. Wood. 1975. Great Vessel, Cardiac Chamber and Wall. Gross Patterns in Normal Children. Circulation. Vol. 51, p. 1124.

Feigenbaum, H. 1973. Echocardiography. Lea & Febiger, Philadephia, Pennsylvania.

Feild, B. J., W. A. Baxley, R. O. Russel, Jr., W. P. Hood, Jr., J. H. Holt, J. T. Dowling and C. E. Rackley. 1973. Left Ventricular Function and Hypertrophy in Cardiomyopathy with Depressed Ejection Fraction. Circulation. Vol. 47, pp. 1022-1031.

Gordon, T., W. P. Castelli, M. C. Hjortland, W. B. Kannel and T. R. Dawber. 1977. Predicting Coronary Heart Diseas in Middle-aged and Older Persons. JAMA. Vol. 238, p. 497.

Gordon, T., W. P. Castelli, M. C. Hjortland, W. B. Kannel and T. R. Dawber. 1977. The Framingham Study. Ann. Int. Med. Vol. 87, pp. 393-397.

Graham, T. P., B. W. Lewis, M. M. Jarmakani, R. V. Canent and M. P. Capp. 1980. Left Heart Volume and Mass Quantification in Children with Left Ventricular Pressure Overload. Circulation. Vol. 41, pp. 203-212.

Grant, C., D. G. Greene and I. L. Bunnell. 1965. Left Ventricular Enlargement and Hypertrophy. Amer. J. Med. Vol. 39, pp. 895-904.

Grossman, W., D. Jones and L. P. McLaurin. 1975. Wall Stress and Patterns of Hypertrophy in the Human Left Ventricle. J. Clin. Invest. Vol. 56, pp. 56-64.

Henry, W. L., J. Ware and J. M. Gardin. 1978. Echocardiographic Measurements in Normal Subjects; Gross Related Changes that Occur Between Infancy and Early Adulthood. Circulation. Vol. 57, pp. 278-285.

Joffe, C. D. 1976. Practical Echocardiography. F. A. Davis Company, Philadelphia 2-3, 199-205.

Kannel, W. B., E. J. LeBauer and T. R. Dawber. 1967. Relation of Body Weight to Development of Coronary Heart Disease. Circulation. Vol. 35, p. 734.

Kannel, W. B. and T. W. Gordon. 1974. Some Characteristics Related to the Incidence of Cardiovascular Disease and Death. (Framingham Study). NIH. pp. 74-599.

Kannel, W. B. and P. Sorlie. 1975. Hypertension in Framingham. In Epidemology and Control of Hypertension, pp. 553-592.

Kotler, M. L. and T. B. Segal. 1978. Clinical Echocardiography. The Charles Press Publishers; Bowie, MD., pp. 8-9.

Lincoln, E. F. 1976. Heart Size. Amer. Heart Assoc. Vol. 39(3), p. 297.

Longhurst, J. C., A. R. Kelly, W. J. Gonyea and J. H. Mitchell. 1980. Echocardiographic Left Ventricular Masses in Distance Runners and Weight Lifters. J. Appl. Phys. Vol. 48(1), pp. 154-162.

Lundstrom, M. 1974. Clinical Applications of Echocardiography in Infants and Children Without Heart Disease. Acta. Paediat. Scand. Vol. 63, p. 123.

Noppa, H., C. Bengtsson, p. Bjorntopp, A. Smith and E. Tibblin. 1978. Acta. Med. Scand. Vol. 203, pp. 135-141.

Olefsky, G. M. and J. W. Farquhar. 1974. Effects of Weight and Reduction on Obesity. J. Clin. Invest. Vol. 53, pp. 64-76.

Singman, H. S, S. N. Berman, C. Cowell, E. Maslansky and M. Archer. 1980. The Anti-Coronary Club. Circulation. Vol. 62(2), p. 449A.

Siri, W. E. 1956. Body Composition from Fluid Spaces and Density. Med. Physics. Rept.

Whyte, H. M. 1959. Blood Pressure and Obesity. Circulation. Vol. 19, pp. 511-516.

Wilmore, J. H. and A. R. Behnke. 1969. An Antropometric Estimation of Body Density and Lean Body Weight in Young Men. J. Appl. Phys. Vol. 27, pp. 25-31.