ECHOCARDIOGRAPHIC ASSESSMENT OF THE LEFT VENTRICLE IN THE SPINAL CORD INJURED PATIENT

THESIS

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By

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Ten caucasian male quadriplegics were compared with eight sedentary caucasian male controls in regards to left ventricular dimensions and mass obtained from echocardiograms. The interventricular septum (IVS), left ventricular posterior wall (LVPW) and left ventricular internal diameter (LVID) were within normal limits for both groups. However, the IVS in the SCI were significantly thicker than controls ($p < 0.05$). Myocardial thickness was larger in SCI subjects ($p < 0.05$). Absolute left ventricular mass (LVM) and total left ventricular volume was not different ($p > 0.05$), but SCI subjects had significantly greater LVM to lean body mass ratios. Echocardiographically, SCI patients demonstrate concentric hypertrophy. This suggests adaptive response to chronic increase in afterload pressure secondary to their daily activities and muscle spasticity.

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CHAPTER I

INTRODUCTION

The spinal cord injured (SCI) patient has posed many problems for the medical profession over the years. The advances in understanding of the physiological problems of the SCI have improved medical care and increased the SCI's lifespan. Twenty years ago the life expectancy of the SCI patient was less than one year (20, 47). Today, over ninety percent of the SCI patient population are alive and well ten years after injury and are expected to live almost a normal lifespan (47). However, even though SCI patients are living longer they may be prone to premature aging (45) and a higher average coronary risk profile (41). The extended life span in conjunction with the problems of premature aging and coronary artery disease has presented the medical profession with new questions to answer.

The primary causes of death in the chronic SCI patients are renal failure and cardiovascular disease (68). Specifically, the SCI appear at greater risk for myocardial infarctions and atherosclerotic heart disease than the general population (20, 47). It has been speculated that inactivity due to wheelchair confinement is a major contributor to the SCI's higher incidence of cardiovascular disease (7, 14, 20). To curtail and possibly prevent cardiovascular problems, many investigators have been investigating the SCI's responses to exercise and have developed exercise programs specific to their needs (7, 14). Many of these programs have been based on the aerobic concepts of exercise prescription (7, 8) and were designed to attenuate the degenerative changes within the cardiovascular system related to a limited activity lifestyle. Many investigations have evaluated the effects of aerobic exercise training on the cardiovascular system with respect

to blood pressure control (8). However, no specific anatomical description of changes in the heart after cord injury in relation to the level of injury has been reported.

When the heart works against increased preloads or afterloads (33, 34, 42, 63), or in cases of sympathetic denervation (40), left ventricular hypertrophy is a frequently noted anatomical change. In recent years, numerous studies on athletes have documented cardiovascular changes in relation to long term dynamic training. While relatively few studies have been conducted to determine the changes induced by chronic static exercise on the same cardiovascular parameters. Three of these investigations compared the effects on the heart between those athletes performing predominantly dynamic exercise and those who perform primarily static exercise (33, 42, 63). Anatomical differences in the cardiovascular system were noted between the two groups and the distinct modes of training were thought to be the primary reason for the differences.

Activities of daily living contain both static and dynamic components of muscle contraction and are generally performed without much thought. However, these muscular activities become increasingly more difficult for the ambulatory disabled (30, 31). Disabilities, as those seen in paraplegics and quadriplegics, lead to many changes in the way activities of daily living are performed. Due to paralysis below the level of injury, the SCI are confined to wheelchairs. Loss of sensation, decreased circulation in the paralyzed extremities, and confinement to sitting many hours a day make them prone to pressure sores on their buttocks (66). In order to help prevent this prevalent problem, the SCI are instructed during their rehabilitation to perform sitting push-ups and to hold them for 10-15 seconds every 15-30 minutes (22). This is to be done as long as they are confined to a wheelchair (22). An activity such as getting in and out of bed involves transferring from the bed to a wheelchair using the upper extremities only, resulting in the SCI performing several sideways sitting push-ups (22). The above two activities involve lifting and sustaining support of one's own body weight. Hence, these transfer activities

have a static component, because the body weight must be lifted with the smaller muscle groups, in addition to controlling their paralyzed trunk muscles to insure maintenance of balance (30, 31, 56).

In addition to the voluntary static muscle contractions, the SCI experience numerous involuntary muscle contractions due to spasticity and autonomic dysreflexia (7, 14, 18). Spasticity and autonomic dysreflexia have been shown to cause similar cardiovascular responses as those observed during voluntary isometric contractions (6, 33). The occurrence and intensity of spasticity and autonomic dysreflexia vary throughout the day and from day to day (8).

In addition to the increased afterload effects of the intermittent isometric activities and spasticity, another factor which may lead to changes in left ventricular anatomy and function is sympathetic denervation (30, 31, 44, 67). In patients with high cord lesions (ie. above T_5) the heart may be partially or completely denervated (42). In animal studies of chronic denervation increases in size of the left ventricle and an increased sensitivity to circulating catecholamines have been noted (30, 32, 44, 55, 67).

As stated before, cardiovascular changes are expected when the heart is subjected to different workloads in response to exercise, spasticity, or sympathetic denervation (30, 31, 44, 46, 67). In summary, the heart of the SCI, due to voluntary and involuntary isometric contractions, is subjected to chronic increases in afterload pressures, while additional influences of partial sympathectomy may also be present (7, 14, 18). Hence, one may propose that the SCI should have an increase in left ventricular mass and posterior wall thickness when compared to normal population values (30, 31, 46, 51, 66). This investigation was designed to determine if cardiac structural changes arise in the chronic SCI patient and to describe the changes in relation to gross anthropometric parameters such as body weight and lean body mass.

Statement of the Problem

The purpose of this investigation was to provide an "in vivo" description of the heart's anatomical components in order to compare the heart structure of the spinal cord injured (SCI) with that of healthy sedentary individuals.

Null Hypothesis

There will be no differences between the spinal cord injured population and the controls in left ventricular mass and posterior wall thickness.

Delimitation

The experimental population will be males between the ages of 20 to 35 with spinal cord injuries of C4-8 and T7-12. In addition, the subjects will have spasticity and be of varying levels of activity. The control subjects will be healthy males within the same age range of the spinal cord injured.

Limitations

1. The subjects were not selected on a random basis.
2. Results of the study will be limited to those levels of SCI involved in the study.

Definition of Terms

Activities of daily living: functions considered necessary for maintenance of a normal life; ie., eating, dressing, bathing, grooming, etc.

Autonomic dysreflexia: paroxysmal hypertension during certain physiological functions; abnormal reflexes elicited by bladder distention and micturition, or rectal distension and pressure, sweating, muscle spasms and headaches.

Chronic: an activity performed intermittently during the day for a period of time exceeding months or years.

Dynamic exercise: rhythmic contraction performed by large muscle groups against a light load.

Isometric contraction: development of tension without a change in muscle length.

Isometric exercise: slow contraction against a heavy load; high static component and low dynamic component.

Isotonic contraction: muscle length shortens with a constant tension.

Lean body mass: the weight of bone and muscle in the body.

Paraplegia: paralysis involving the trunk and both lower extremities.

Quadriplegia: paralysis of trunk, legs and partial to complete paralysis of both arms.

Sitting push-ups: while seated in a chair, lifting the buttocks from the surface using the upper extremities only.

Spasticity: involuntary muscular contractions caused by the severing of the spinal cord with subsequent loss of cerebral inhibitory effect on spinal reflexes.

Transfers: as related to the spinal cord injured population, the moving of oneself from one place to another by doing several sideways sitting push-ups.

CHAPTER II

REVIEW OF THE LITERATURE

This section discusses the validity of using echocardiography to determine myocardial dimensions and anatomy. It will also review the effects of various types of exercise on the normal cardiovascular system and then discuss how this may relate to alterations in the cardiac anatomy in persons with spinal cord injury.

Echocardiography

Information in regards to cardiac anatomical variations associated with disease or athletics had been limited until the development of single M-mode echocardiography, a sophisticated noninvasive technique (42). Devereux and Reichek (1977) compared autopsy reports with echocardiographic results on patients who had died within four months of their performance of an echocardiographic evaluation of the heart. The authors reported that the Penn Convention method for determining left ventricular mass (LVM) was an accurate ($r = .96$) method for the study of left ventricular hypertrophy and were unable to identify any group in which the method was grossly inaccurate. Anatomical LVM can be calculated at autopsy by measurements of posterior wall thickness (LVPW), interventricular septal wall thickness (IVS), left ventricular internal diameter (LVID), apical myocardial thickness and hemimajor axis (15). The LPW, IVS and LVID can now be measured directly by echocardiography (9). However, without the measure of the hemimajor axis, LVM calculations were dependent on a cube formula that estimated the volume of the left ventricle (9). Determination of LVM also required measurements of myocardial thickness (9). According to Devereux, autopsy myocardial thickness

measurements have been considered to be very accurate (53, 69); however, Devereux (1977) stated "that LPW and IVS measurements by echocardiography are not absolutely accurate due to the inability to factor the gain settings of the ultrasonic probe into the individual recording and because of the structural complexity of the septum and posterior wall." However, Devereux further noted that there was a relationship between LPW and IVS and the mean myocardial muscle thickness (MMT) such that when the Penn Convention was combined with the formula, MMT = (IVS + LPW)/2, the results were an accurate estimate of LVM.

Exercise Training and Echocardiography

With the advent of echocardiography, numerous studies on athletes have been conducted investigating the effects of long term dynamic exercise training and chronic weight training (33, 42, 54, 63). These investigations have found cardiovascular differences between the two groups of athletes undergoing the two distinct modes of training. Morganroth, et al. (1975) used echocardiography to compare cardiovascular parameters in swimmers, long distance runners (LDR), and wrestlers (42). They found that the left ventricular mass (LVM) was enlarged in all of the athletes when compared to controls (301.9gms to 330.1 gms vs. 211.4 gms), but the cause of the increased mass differed according to the sport. Swimmers and runners had greater than normal left ventricular end-diastolic volume (LVEDV = 54.1 mm - 56.5 mm vs. 46.4 mm), but normal left ventricular or posterior wall thickness (LPW = 10.6 mm - 11.3 mm). Wrestlers, on the other hand, had a normal mean LVEDV of 47.8 mm vs. 46.4 mm, but an increased LPW of (13.7 mm vs. 10.3 mm). In a later investigation, Longhurst, et al. (1980) compared 17 competitive weight lifters (CWL), long distance runners (LDR), amateur weight lifters (AWL), and 24 sedentary controls [12 heavy controls (HC) and 12 lean controls (LC) of similar lean body mass (LBM)]. The absolute LVM was

significantly increased in the two competitive athlete groups compared with their respective controls (LDR 195 ± 12, CWL 190 ± 10, LC 122 ± 10 and HC 151 ± 9 gms). Normalization of the LVM measurement by accounting for LBM resulted in the LDR group having a greater LVM/LBM ratio than all other groups and equalized the LVM of the CWL and the matched HC (LDR 3.2 + 0.2, CWL 2.5 + 0.1, AWL 2.5 + 0.3, HC 2.3 + 0.2, and LC 2.0 + 0.3 gms). This data demonstrates that endurance athletes develop eccentric or appropriate hypertrophy with an increase in internal diameter without an increase in wall thickness.

In another study by Snoechex, et al. (1981), echocardiographic parameters of 16 long distance runners, 15 cycle racers, 14 weight lifters and 17 controls were compared. The results indicated increases in left ventricular diameter (10.7 - 16.3 percent) and wall thickness (37.8 percent) over controls of both the runners and cyclists with subsequent increases in LVM. In the weight lifting group, the left ventricular posterior wall thickness was significantly enlarged (17 percent), but LVM was not significantly increased in comparison to controls In summary, these studies (33, 42, 63) suggest that long term dynamic exercise such as running and cycling will result in an increase in LVM and LVEDV. However, while exercises such as wrestling and weight lifting, which have a high proportion of static exercise, will result in increased left ventricular posterior wall thickness and LVM/LVEDV ratio.

More recently, Shapiro, et al. (1983), has evaluated the rate of left ventricular structural changes during training and detraining. In this investigation, healthy, nonathletic males were placed on a running training program for twelve weeks. During the first two weeks, each running session was 15 minutes in duration and was repeated five times per week. At the end of each two weeks the duration was increased five minutes until 6 weeks of training was accomplished. The frequency of sessions were unchanged. At the end of six weeks the volunteers were asked to either continue running

five times per week for 30 minutes or to stop training completely. Echocardiographic assessment of the heart structures was performed at the end of 1, 2, 4, 6, and 12 weeks. At four weeks, increases in wall thickness of up to 30 percent were seen without changes in left ventricular cavity diameters. After six weeks the increase in LVM was similar to that observed in elite athletes. No further changes were observed at the end of the twelfth week of training when compared to the six week's measures. However, in the subjects who had ceased training at six weeks, small yet significant reductions in wall and septal thickness were observed. From this data, it would appear that the human heart structures adapt readily to moderate exercise training and its subsequent detraining. These findings confer with the data of Hickson et al. (1983) who, using a rat model, demonstrated that a chronic daily exercise program of swimming six hours per day, six days per week for three weeks resulted in a significantly increased ventricular weight of 19 - 29 percent, and further showed that after 50 days of detraining the ventricular weight returned to normal. The increase and decrease in ventricular weight was shown to be related to collagen synthesis and breakdown, respectively. Obviously, the changes in anatomic structure of the heart, as a result of exercise training, can be rapidly induced and are related to biochemical and physiological changes.

Cardiovascular Responses to Static Exercise

Activities such as running and cycling have been denoted as dynamic, isotonic, or endurance exercises (33, 42, 49, 63), whereas wrestling and weight lifting are denoted as isometric, static, or strength type activities (33, 42, 49, 63). The classical definition of an isometric contraction is the development of tension without a change in muscle length (38) Force-velocity curves, plotting speed against load, demonstrate that as the load is increased the speed of contraction decreases until the velocity is equal to zero and a pure isometric contraction is performed (4). Also, as the load increases the latent period

increases and the total amount of shortening decreases (4). It is clear that as the magnitude of the load moved is increased, the muscle spends more and more of its time contracting isometrically (4). Pure isometric exercise is uncommon outside of a research laboratory (32, 32, 49). Most daily activities are a mixture of the isometric and dynamic contraction components of exercise. For example, dynamic exercise at a light load and high speed has a low static component (49) while slow contractions against a heavy load have a high static component (49). A practical definition of an isometric exercise is an activity having a high static component and a low dynamic component, or a slow contraction against a heavy load (49).

Cardiovascular responses to static exercise have been extensively investigated. It was initially reported that when an isometric contraction was sustained at 18 percent MVC for 6 - 7 minutes, the following results were seen: mean arterial pressure (MAP) rose to 126 mm Hg, systemic vascular resistance (SVR) increased to 12 PRU, cardiac output increased 30 percent, and heart rate was elevated (56). In participants performing dynamic or supine bicycle exercises at 100 watts the MAP increased to 102 mm Hg, SVR decreased to 6.5, while heart rate and cardiac output increased. When the two activities were combined the MAP increased to 131 mm Hg., SVR decreased to 7.8, cardiac output increased to 15.91/minute and heart rate increased but did not achieve a steady state level in four minutes as noted in the dynamic activity. Oxygen uptake was greater in the static and static-dynamic exercises with the most significant increases occurring in static exercises (30, 31, 56). This data suggests that a greater load stress was placed on the heart during static and combined static and dynamic exercise than during dynamic exercises alone.

The cardiovascular responses to an isometric or static contraction are well documented (32, 40, 65). During a static contraction systolic, diastolic, and mean arterial pressure were elevated in proportion to the muscle mass and force involved (40).

However, the pressor response was disproportionate to the increased oxygen consumption (40). The pressor response results from an increased cardiac output and a vasoconstriction of nonactive areas increasing SVR (32). The elevated cardiac output was caused by the increased heart rate since the stroke volume changes were minimal (48). Stefadouros et al. (1974) studied the left ventricular response to isometric hand grip exercise with echocardiography in 15 normal subjects. They found that heart rate increased from 79 to 97 beats per minute, cardiac index rose from 3.5 to 4.4 L/min./m^2, mean blood pressure increased from 87 to 120 mm Hg, and the product of stroke volume times the blood pressure (an index of stroke work) rose from 52 to 74 gm-m/m^2. They found no significant differences in the end-diastolic volume, stroke volume, or systemic vascular resistance from resting levels. The overall effect of an isometric contraction was to increase the pressure load, oxygen consumption and workload of the left ventricle.

Spinal Cord Injured Patients and Static Exercise

Although the cardiovascular responses to isometric contractions are well known in the normal population, very little is known in regards to the spinal cord injured (SCI) patient's response to isometric type activities. A few investigators have evaluated the SCI's response to involuntary or spastic contractions due to spasticity and autonomic dysreflexia (6, 14, 66). Incidences of spasticity and autonomic dysreflexia cause similar cardiovascular responses as those observed when normal healthy individuals perform voluntary isometric contractions (6, 14). In the SCI patient, spasticity resulted in increases in systolic, diastolic, mean arterial pressure and pulse pressures (6, 14) and the degree of the pressor response was related to the amount of musculature involved and whether the spasms were induced or spontaneous (6). Induced spasms cause approximately a 13 percent rise in blood pressure, a 9 percent rise in pulse pressure, and a 16 percent decrease in heart rate (6). Spontaneous spasms cause a 19 percent rise in mean

blood pressure, 17 percent rise in pulse pressure, and no change or slight increase in heart rate (6). The primary differences noted between the cardiovascular response to voluntary and involuntary isometric contractions were that heart rate decreased during the involuntary contractions and that the pressor response to the involuntary contractions lasted longer than during the voluntary contractions (6). The occurrence of involuntary muscle contractions or spasticity vary from day to day and from individual to individual (38). Many SCI patients are medicated to help control their involuntary contractions (38).

Partial or complete cardiac sympathetic denervation is another factor that the SCI patient has that can lead to changes in left ventricular anatomy and function (26, 37, 46, 57). High cord lesions, above T_5, result in partial to complete sympathetic denervation of the heart (41). Investigations using animal models of a denervated heart demonstrate increases in end-diastolic volume, myocardial muscle fiber length, coronary collateral circulation and hypertrophy of the left ventricle (26, 27, 46, 57), along with beneficial reductions in myocardial oxygen uptake and collateral resistance (26, 27). However, there are marked increases in sensitivity to circulating catecholamines (52). Denervated hearts increase cardiac output via increases in stroke volume through the Frank-Starling mechanism (52). However, during moderate exercise, the left ventricle was able to increase cardiac output beyond the levels available through the Frank-Starling mechanism alone. This increase in cardiac output was thought to be due to the increased sensitivity of the myocardium to the circulating catecholamines (52). In contrast, a major difference noted between animal studies and those investigations using the SCI patient is that denervated animals have an increased blood volume, whereas the SCI patients do not have increases in blood volume. Subsequently, it was shown that the SCI patient has diminished levels of red blood cells, hemoglobin, and a lower hematocrit (60).

In addition to differences between intact normals and the SCI cardiovascular systems, there are differences between paraplegics and quadriplegics. Quadriplegics do not have

full sympathetic innervation of the heart as seen in lower cord injured paraplegics (14). Resting blood pressure is lower in quadriplegics than paraplegics and intact normals, and furthermore it was shown that there was an increase in blood pressure of 1.97 mm Hg per cord segment below T1 (14). During maximal exercise, quadriplegics have significantly lower maximal heart rates, oxygen uptake and peak exercise capacity in comparison to paraplegics (7).

More recently Kessler et al. (1986) echocardiographically compared seven normal healthy subjects with 7 paraplegic and 7 quadriplegic patients (28). Quadriplegic patients had a 26 percent lower LVM index, lower blood pressure and calculated cardiac output ($P < 0.01$) compared to normals and paraplegic patients. Hemodynamic data for the paraplegic patients were similar to normal subjects while 3 paraplegics and 6 quadriplegic patients had LV asynergy. The authors suggested that a decrease in LV wall stress, mediated primarily by a decrease in venous action, appeared to result in an adaptive cardiac atrophy (28).

A clinically relevant problem seen in quadriplegics compared to normals and paraplegics is the greater incidence of orthostatic hypotension. Orthostatic hypotension has been reported upon head-up tilting and was accompanied by a fall in systolic, diastolic, pulse and mean blood pressures, and an increase in heart rate (14). It is generally accepted that the lack of sympathetic innervation is the cause of the greater incidence of orthostatic hypotension in the quadriplegic (14). Furthermore, plasma catecholamines were lower in the quadriplegics at rest and during exercise and may indicate a depletion or degeneration of sympathetic nerve activity (14, 72).

In summary, alterations in cardiac anatomy would be expected in spinal cord injured patients purely as an adaptive response to the increased static exercise activity of daily living, the involuntary and partial or complete spasticity and sympathetic denervation (26, 27, 33, 42, 46, 57). Specifically, these increases should include changes in LVM, LVEDV and LVPW (26, 27, 33, 42, 49, 63).

CHAPTER III

METHODS AND PROCEDURES

Subjects

Ten healthy spinal cord injured (SCI) caucasian male volunteers between the ages of 20-34 years with the injury levels of C5-8 were recruited from area universities, Texas Rehabilitation Commission, and the regional Paraplegic Society. Range of time from initial spinal cord injury to participation in the study was 2-15 years. Spasticity was present in all SCI volunteers. Eight healthy, sedentary caucasian male volunteers were used as controls.

Prior to the subjects participation in the study, they were informed of the purpose, protocol and experimental procedures. Each subject signed an informed consent form which had been approved by the TCOM Advisory Committee for Research Involving Human Subjects (see Appendix I). The subjects were asked to complete a medical history and activity questionnaire and were then provided a physical examination by a physician's assistant along with a 12 lead electrocardiogram. Manual muscle and sensory tests were performed to verify functional levels of injury. The SCI subjects were accepted into the study if their injuries were complete, as determined by muscle and sensory tests, and they had no history of hypertension, heart murmur, electrocardiogram abnormalities, or other psychophysiological condition that contraindicated their participation in the investigation. The control volunteer subjects were selected on the basis of having no cardiopulmonary or structural and functional disability, except a sedentary lifestyle.

Experimental Protocol

Following their preliminary screening and acceptance into the investigation all of the subjects were seen on two separate days. On the first day, the echocardiographic assessment of cardiac structure was performed by a cardiologist. The second session involved anthropometric measures of height, weight, and lean body mass and percent body fat using the deuterium dilution technique.

Echocardiographic Evaluation

Echocardiogram measurements were obtained using the procedures described by Longhurst et al. (1980). The subject, undressed to the waist, was positioned in a 30 degree left lateral decubitus position. The transducer, focused at 4-7 cm with a frequency of 2.25 mHz, was placed in the left third to fifth intercostal space adjacent to the sternum and directed posteriorly. A 3-lead electrocardiogram was superimposed on the echocardiograph. Speed of the paper was set at 100 mm/sec. Upon identification of the mitral valve leaflets, an M-mode sweep was recorded to include echoes from the interventricular septum and the posterior left ventricular wall (see Figure A). Diastolic dimensions were measured from the peak of the R wave, and the end-systolic diameter was measured at the anterior point of excursion of the posterior left ventricular wall (see Figure A). Left ventricular dimensions were taken at the mitral valve leaflets or just below the tips of the leaflets in the region of the chordae tendineae. The anatomical position selected was the one that demonstrated the widest internal cavity dimension and simultaneous endocardial septal and free wall echocardiogram reproduction.

Only high quality echocardiograms were used for measurement determination. Some echocardiograms were repeated to obtain a better quality tracing. Penn methods of measurement (9) of the left ventricular dimensions were used to determine diastolic interventricular septal thickness (IVS), left ventricular internal diameter (LVID) and left

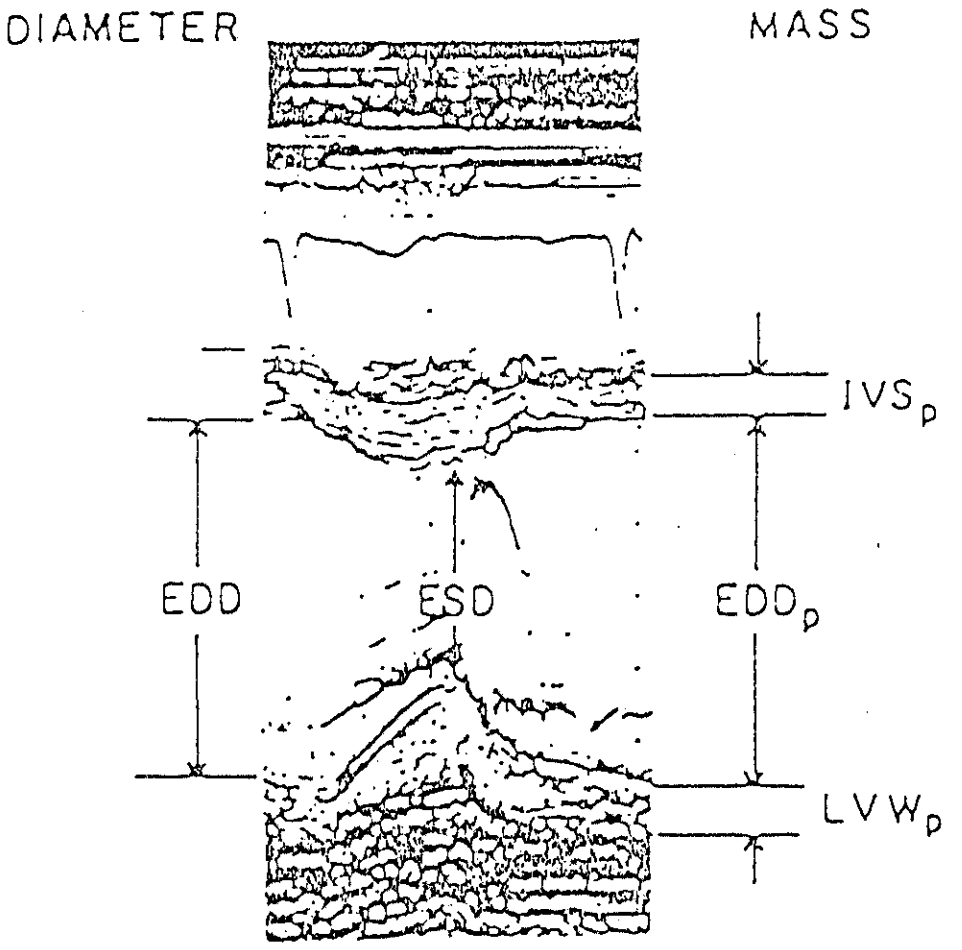

Fig. A--Illustration of M-mode echocardiographic measurements (Langhurst, et al. 1980).

ventricular posterior wall thickness (LVPW). Each echocardiogram was measured by the principal investigator and the consultant cardiologist, and recorded to the nearest 0.5 mm. A discrepancy of greater than 5 percent was re-evaluated by both individuals. The following equations were used to determine left ventricular mass, mean muscle thickness, left ventricular end-diastolic volume, and the ratio of left ventricular mass to end diastolic volume using Penn equations (9, 33).

Left ventricular mass (LVM) = $1.04\ [(LVID + LPW + IVS)^3-(LVID)^3] -13.6g$

Mean muscle thickness = IVS + LPW/2

Left ventricular end-diastolic volume(LVEDV) = $1.047\ (LVID)^3$

Lean Body Mass Determination

Accepted methods for determining lean body mass of normal sedentary subjects have not been verified in SCI population. A study of body composition changes in the SCI using skinfolds and heavy water methods found an increased sodium space and body fat, and a decreased bone density (8). Subsequently, calculated changes in lean body mass were found to be inconsistent (18); however, it was felt that the physiological changes were consistent with the enforced sedentary lifestyle. Other workers have found that underwater weighing and potassium 40 counting (k^+40) yield similar estimates of lean body mass, while skinfold measurements predicted a greater body of fat and had a larger variance of measurements between the SCI subjects (34).

As deuterium dilution techniques measure total body water (TBW) regardless of age, race or sex, and lean body mass is considered to be a constant function of TBW (3, 59); it was felt that the deuterium dilution technique would more accurately compare SCI patients with healthy normals. In the present investigation, the deuterium dilution technique was selected as the method of choice for determining lean body mass and calculated percentage of body weight as fat.

Deuterium Oxide Determination of Lean Body Mass

All subjects emptied their bladder and bowels and/or catheter bag and tubing prior to drinking the tracer, deuterium oxide. Each subject drank 4 ounces of 27 mg tracer, and then filled the 4 ounce bottle three-quarters full with tap water, mixed, and then drank the water. The bottle was refilled with tap water and then drunk. Participants were not allowed to drink or eat anything for the next 4-5 hours. At the end of the 4-5 hour period, a urine sample was taken from the control group. The SCI groups catheter leg bag and tubing were emptied and then an additional 30 minutes were allotted before a urine sample was taken from the leg bag. The urine samples were sent to Sometric Research Laboratories, Houston, Texas, for analysis. The samples were double vacuum distilled and the distillates were analyzed using an infrared spectrophotometer (MIRAN IA) with a fixed wavelength of 4.0 μm and a heat controlled sample cell. The resultant concentration aside was determined and was used to predict total body water (3,59). Fat percentage of body weight was determined by the formula:

$$\text{Fat \%} = 100 \times [\text{weight} - (\text{TBW}/.732)]/\text{weight}$$

Statistical Analysis

A Two-Tailed Student t test was used to determine group differences in the echocardiographic measurements and their ratios. Descriptive statistics such as regression equations and correlations were used as necessary. A significant difference was accepted at 5 percent level of probability, $P < 0.05$.

CHAPTER IV

RESULTS

The purpose of this section is to present the data collected during the investigation using the methods described in section 3. Fourteen spinal cord injured (SCI) individuals volunteered to participate in the study, and ten were selected to complete the study. The SCI volunteers not selected were rejected for heart abnormalities, bladder infections, the presence of incomplete lesions, or the lesion was below C8. All of the SCI volunteers were at least two years post injury with a mean of 4.6 years (range 2.08 to 12.17 years) and had cord lesions of C4-5 to C7-8. Eight normal sedentary young men volunteered to be controls and all were used in the study. A demographic description of the two groups is listed in Table I.

TABLE I

SUBJECT CHARACTERISTICS

		No.of Yrs. Past Injury	Age (Yrs.)	Weight (Kg)	LBM (Kg)
SCI Patients	x	4.60	23.7	70.8	48.6
	S.D.	± 1.80	± 3.3	± 16.1	± 7.2
Control	x	NA	26.8	67.2	56.1*
	S.D.		± 4.0	± 9.8	± 8.0

*$p < 0.05$

No statistical differences were noted between the two groups in regards to age, weight, or height. The SCI volunteers mean age, weight and height are as follows: 23.7 years (range 20 to 32 years), 70.8 kg (range 47.7 to 97.3 kg), and 158.4 cm (range 145.2 to 171.6 cm). The mean age, weight and height of the control group are 26.8 years (range 21 to 33 years), 67.2 kg (range 51.6 to 85.9 kg), and 156.2 cm (range 140.8 to 167.2 cm), respectively. The control group had a significantly greater lean body mass (56.1 ± 8.0 kg, range 39.2 to 69.6 kg) when compared to the SCI groups (48.6 ± 7.2 kg, range 41.0 to 59.4 kg).

It was determined from electrocardiographic precordial voltage criteria that there was no evidence of cardiac enlargement in either subject group. However, one of the SCI volunteers had a variable first degree AV block with a P-R interval ranging from 0.20 or less. Mean resting heart rates of the SCI subjects were 79.8 beats per minute and 68.7 beats per minute in the control subjects ($P > 0.05$). Mean resting blood pressure measurements of the SCI and control groups were 104/76 and 110/80, respectively and were not significantly different.

Echocardiographic Analysis

Technically adequate echocardiograms from the left decubitus position were obtained from all subjects at rest. In all echocardiograms, left ventricular posterior wall thickness (LVPW), left ventricular internal diameter (LVID), and interventricular septal wall thickness (IVS) were measured (Table II).

TABLE II

ECHOCARDIOGRAPHIC MEASUREMENTS
OF THE LEFT VENTRICLE

		LPW (mm)	IVS (mm)	LVID (cm)
SCI Patients	x	9.7	10.1*	4.8
	S.D.	± 1.4	1.4	0.5
Control	x	9.2	8.2	5.1
	S.D.	± 0.9	1.4	0.4

*$p < 0.05$

IVS was significantly greater in the SCI group than controls (10.1 ± 1.14 mm, range 9.0 to 12.0 mm vs. 8.21 ± 1.44 mm, range 6.0 to 11.0 mm, $P < 0.05$). The mean values of both groups were within the accepted values of normal range (7-11 mm). However, it was observed that the IVS of 8 of the ten SCI subjects was within the upper range of normal and that two of the SCI subjects values exceeded the normal range, see Figure 1.

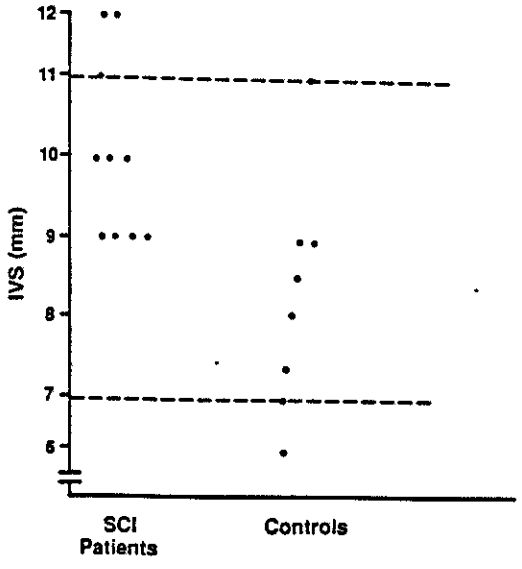

Figure 1: Interventricular Septal thickness measurements with the accepted normal range indicated by the horizontal lines (–).

LVID was not significantly different in the SCI subjects when compared to controls (4.8 ± 0.52 cm, range 4.0 to 5.8 cm vs. 5.06 ± 0.36 cm range 4.4 to 5.5 cm, $P > 0.05$) and were within normal limits. The LVPW thickness values were similar between the two groups (SCI 9.7 ± 1.27 mm, range 7 to 12 mm vs. controls 9.24 ± 0.94, range 8.0 to 11.0 mm, $P > 0.05$). The average values for LVPW thickness were also found to be within the normal accepted range of 7-11 mm. Again, a trend was seen in the LVPW measurements in that the SCI subjects values were in the upper normal limits, see Figure 2.

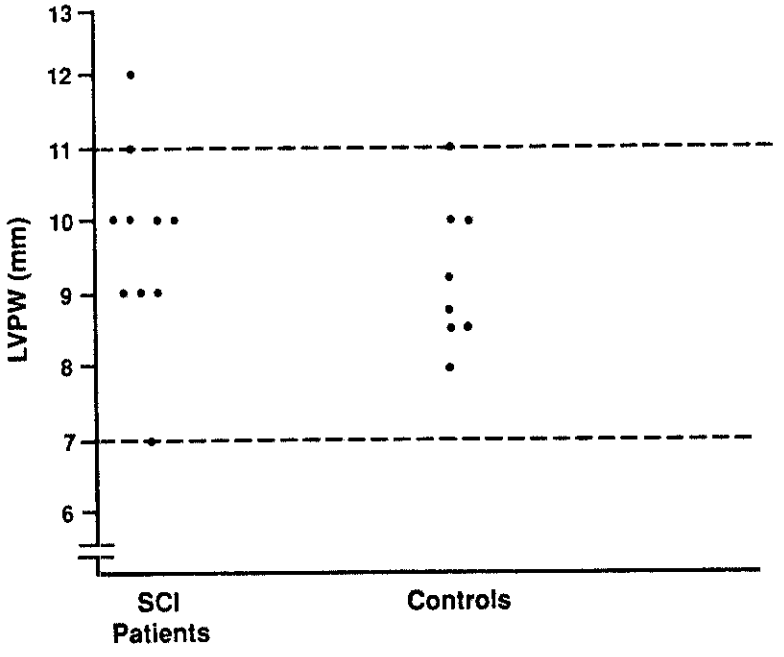

Figure 2: Left ventricular posterior wall (LVPW) thickness measurements with the accepted normal range indicated by the horizontal lines (--).

The average myocardial muscle thickness calculated from (IVS + LVPW)/2 indicated that the SCI subjects had a significantly greater average value than controls (0.985 $\pm$ 0.12 cm, range 0.8 to 1.2 cm vs. 0.88 $\pm$ 0.11 cm, range 0.75 to 1.1 cm, $p < 0.05$), see Figure 3.

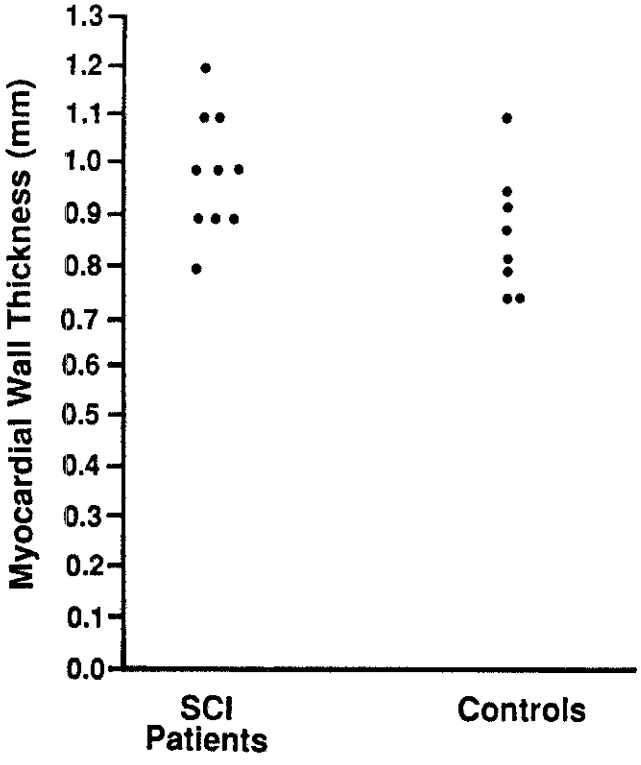

Figure 3: Average myocardial muscle thickness of the left ventricle calculated from (IVS + LPW/2).

The IVS/LVPW ratio was significantly greater in the SCI in comparison to controls (1.05 ± 0.14, range 0.8 to 1.3, vs. 0.89 ± 0.11, range 0.66 to 1.06, $P < 0.05$). Furthermore, eighty percent of the SCI subjects had a ratio of one or greater, see Figure 4.

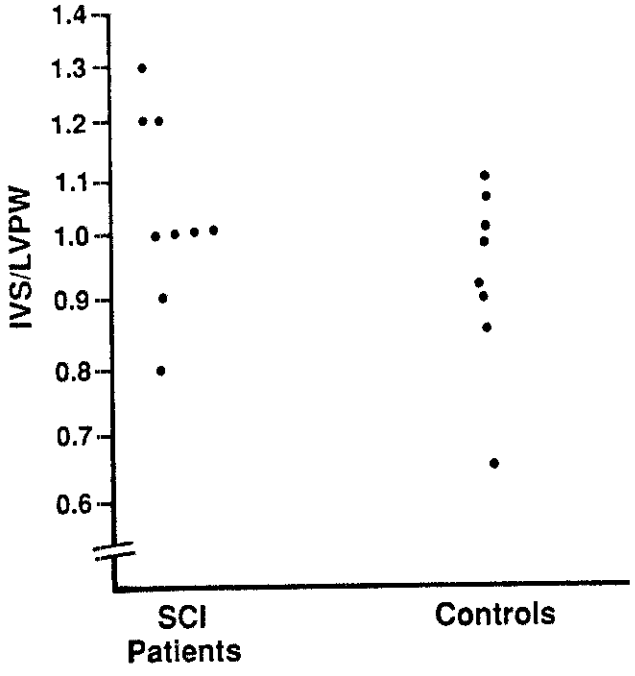

Figure 4: Interventricular Septal (IVS) thickness to Left Ventricular Posterior Wall (LVPW) thickness ratio.

Left ventricular mass (LVM) was calculated using the Penn equation and is represented in Table III. LVM was then corrected by total body weight (TBW) and lean body mass (LBM). Absolute mean LVM measurements did not differ significantly between the SCI group and controls, 180 g and 195 g, respectively. The SCI group had a significantly greater ratio in comparison to controls when LBM was considered (4.03 $\pm$ 0.77 g/kg, range 3.32 to 5.91 g/kg vs. 3.01 $\pm$ 0.68 g/kg, range 2.53 to 3.46 g/kg, $P<0.05$). LVM/TBW showed no significant difference (SCI 2.87 $\pm$ 0.68 g/kg, range 1.78 to 3.89 g/kg vs. controls 2.80 $\pm$ 0.28, range 2.13 to 3.78 g/kg).

TABLE III

MEAN LEFT VENTRICULAR MASS (gm)*

		LVM (g)	LVM/TBW (g/kg)	LVM/LBM (g/kg)
SCI Patients	x	195	2.87	4.03**
	S.D.	$\pm$ 46	$\pm$ 0.68	$\pm$ 0.77
Control	x	180	2.80	3.01
	S.D.	$\pm$ 45	$\pm$ 0.28	$\pm$ 0.76

* Values calculated using the Penn equation and then normalized by total body weight and lean body mass.

**$p < 0.05$

LVM to left ventricular volume ratio was not different for the SCI subjects compared to the controls (1.74 $\pm$ 0.43, range 1.18 to 2.69, vs. 1.64 $\pm$ 0.28, range 1.16 to 2.02, $P > 0.05$). Wall thickness to radius ratio was not significantly greater in the SCI subjects (Table IV). Although there was no significant difference between the two groups, the SCI subjects mean value of 0.41 mm/cm was greater than the normal accepted range of 0.32 to 0.36 mm/cm.

TABLE IV

LEFT VENTRICULAR POSTERIOR WALL THICKNESS (mm)
TO LEFT VENTRICULAR RADIUS (cm) RATIO

Subject	SCI Patients	Control
1	0.36	0.38
2	0.41	0.34
3	0.28	0.36
4	0.38	0.35
5	0.35	0.33
6	0.47	0.39
7	0.40	0.34
8	0.47	0.44
9	0.55	
10	0.47	
x	0.41	0.36
S.D.	0.07	0.03

Total left ventricular volume (TLVV) was not significantly different between the two groups (SCI 323 $\pm$ 83, range 249 to 536 cm^3, vs. 326 $\pm$ 65 cm^3, range 236 to 421 cm^3), Table V. When TLVV was normalized for differences in TBW, the control subjects demonstrated no significantly greater ratio than the SCI subjects (4.8 $\pm$ 0.85, range 2.9 to 6.3 cm^3/kg vs. 4.7 $\pm$ 1.2, range 3.7 to 5.7 cm^3/kg, $P > 0.05$); but when the TLVV was corrected by LBM, the SCI subjects had a significantly greater ratio of 6.8 $\pm$ 1.4 cm^3/kg, range 5.0 to 10.1 cm^3/kg vs. 5.6 $\pm$ 1.0 cm^3/kg, range 4.3 to 6.2 cm^3/kg.

TABLE V

MEAN TOTAL LEFT VENTRICULAR VOLUME (cm^3)
NORMALIZED BY TOTAL BODY WEIGHT
AND LEAN BODY MASS

		TLVV (cm^3)	TLVV/TBW (cm^3/kg)	TLVV/LBM (cm^3/kg)
SCI Patients	x	323	4.70	6.8*
	S.D.	± 83	± 1.20	± 1.4
Control	x	326	4.80	5.6
	S.D.	± 465	± 0.85	± 1.0

*$p < 0.05$

In summary, the measurements of interventricular septal wall thickness, left ventricular posterior wall thickness, and left ventricular internal diameter of the sample echocardiograms were within acceptable limits of normal. However, the septal wall thickness in the SCI was significantly thicker than matched controls. Furthermore, mean muscle thickness was also significantly larger in the SCI subjects. Absolute left ventricular mass and total left ventricular volume did not differ significantly between groups and when corrected by total body weight no differences were noted. However, when the lean body mass was used as the correction factor, the SCI subjects evidenced significantly greater ratios.

CHAPTER V

DISCUSSION

The purpose of this discussion will be to evaluate the results of this investigation in relation to the cardiac anatomical changes observed in the spinal cord injured patient when compared to a healthy normal sedentary individual. Also in this section, implications with respect to differences in cardiac anatomic structure and spinal cord injury will be proposed. Subsequently, recommendations for future investigations are presented.

The findings of the present investigation clearly identify that the measurements of IVS and LVPW thickness, as well as LVID of the SCI and normal healthy sedentary subjects were all within the accepted range of normal values (35). However, both IVS and LVPW of the SCI patients were in the upper range of normal thickness values and were similar to values reported for weightlifting athletes (33, 35, 42). Confirmation of this similarity was demonstrated in the significantly increased ratio of IVS/LVPW of the SCI patients compared to the healthy control subjects, see Figure 4. Previously, Menapase (1982) reported that weightlifting athletes had IVS/LPW ratios of greater than 1.3, while other researchers reported findings ranging from 0.95 to 1.01 (33, 42, 63). In the present study, the average IVS/LVPW ratio of the SCI patients was 1.05 and would suggest IVS thickening as a result of resistive exercise performance. However, Marcomechelabis (1983) has reported that IVS, CVPW and the IVS/LVPW ratio increase directly with increasing age and provides population normal values based on age. In this study, the values of IVS and LVPW of the control subjects were equivalent to the 50th percentile of their age group, whereas, the SCI patient values were equivalent to the 95th percentile values. IVS/LVPW ratio comparisons for the SCI patients indicated values at the 50th

percentile, whereas the control group's ratio values were less than the 50th percentile. Based on the above discussion, it was concluded that the greater thickness of IVS and LVPW of the SCI patients was probably secondary to daily resistive type activities.

The possible presence of cardiac muscle hypertrophy in the SCI patients as a result of resistive type activities was further evaluated by comparing LVID and mean muscle thickness relationships, and LVM and TLVV in relation to LBM. It has been previously reported that LVID increases in weightlifters, but the mean myocardium thickens out of proportion to the changes in the LVID (33, 37, 42). These changes are consistently reported for heavy resistance type athletes (weightlifting, wrestling and body building) and presumed to be related to the heart's adaptation to increased afterload pressures. In the present investigation, LVPW thickness of the SCI patients was greater than their matched controls, yet the LVID's were similar to the controls and population norms. Calculated ratios of LVPW/LVID were greater for the SCI patients than their matched controls and were similar to previous reports for resistance trained athletic groups (33, 42, 63). An increase in LVPW thickness without a change in radius of the ventricular chamber is consistent with concentric hypertrophy (17). It has been repeatedly demonstrated that concentric cardiac hypertrophy is a response to chronic repetitive isometric skeletal muscle contractions or a response to hypertension.

Further support for the existence of concentric hypertrophy in the group of SCI patients is seen when values of TLVV and LVM, normalized by LBM, are compared with control or athletic populations. TLVV/LBM ratio of the SCI patients was significantly greater ($P < 0.05$) than the control subjects ratios and similar to values of the elite athletic runners reported by Longhurst et al. (1980). Similarly, LVM/LBM values of the SCI patients were significantly greater ($P < 0.05$) than the control subjects. These same indices of cardiac hypertrophy of the SCI patient were greater than the reported indices of athletic cardiac hypertrophy reported by Longhurst.

In summary, echocardiographically the SCI patient has strong cardiac anatomical structural data indicative of concentric heart hypertrophy. This is suggestive of an adaptive response to chronic isometric exercise activities. Mechanistically, isometric tension within skeletal muscle results in an increased afterload and sympathetic cardiac stimulation of chronotropy and inotropy (40, 65, 71). In keeping with these responses, ventricular wall stress increases. Ventricular wall tension is directly related to ventricular pressure and internal radius of the ventricle. In order to keep systolic tension constant per cross sectional area of muscle, the ventricular wall thickness must increase as the radius increases (48). It is postulated that the increased systolic wall tension of the left ventricle associated with increased afterload stimulates the synthesis of sarcomeres in parallel to existing sarcomeres and produces concentric hypertrophy. Therefore, possibly the daily activities (transfers and sitting push-ups) of the SCI patient combined with his involuntary muscle spasms increases the afterload pressure on the heart which subsequently stimulates sarcomere synthesis in parallel with resultant concentric hypertrophy (19).

Another possible mechanism producing concentric cardiac hypertrophy within the SCI patient could be the existence of complete cardiac denervation. Each of the SCI patients had cord lesions above T1 and were regarded as having cardiac sympathectomies. Patients with cardiac denervation resulting from allograft transplantation have been shown to increase their diastolic and systolic blood pressure during isometric exercise as seen in innervated normal hearts but there was no concomitant increase in heart rate as seen in normals (57). Corbett (1971) reported that the involuntary muscle contractions associated with spasticity in the quadriplegic had similar cardiovascular effects as those seen in a normal healthy individual performing an isometric contraction; i.e., increased diastolic and systolic blood pressure, except there was no heart rate response. This evidence supports the presence of a denervated heart in quadriplegic patients similar to that seen in cardiac transplant patients. Unfortunately, cardiac transplant patients usually exist with

cyclosporin induced chronic hypertension, hence even though cardiac anatomic changes were observed in the transplant patient, it was difficult to assess whether the increased afterload was pharmacologically induced (cyclosporin) or intrinsic changes in the resistance vessels following transplantation (2). However, the SCI patients evaluated in the present study were less than thirty years of age, normotensive, and free of any ausculatory, electrocardiographic or echocardiographic signs of cardiac disease. Therefore, the presence of concentric cardiac hypertrophy was probably a result of frequent episodes of increased afterload pressure secondary to chronic daily activities involving numerous isometric muscle contractions and prolonged bouts of muscle spasticity.

Finally, it was postulated that because the SCI patients of this investigation were cardiac denervated, sympathetic augmentation of contractility and chronotropy would be compromised. Subsequently, the SCI patient would rely more heavily on the Frank-Starling mechanism to maintain or increase the cardiac output, thereby requiring a greater preload and greater diastolic filling. This expected increase in preload and diastolic filling would lead to increased LVID as seen in long distance runners (33, 42, 54), but the SCI individuals in this study had LVID values within the normal limits and did not differ from matched normal controls. Kessler et al. (1986) demonstrated that the more severely compromised SCI patient (i.e., the quadriplegic) had cardiac atrophy and postulated that it was secondary to chronic decreases in venous return. In conclusion, the data supports the premise that the SCI patient has developed concentric hypertrophy as an adaptation to the chronic increases in afterload secondary to their daily activities and muscle spasticity.

Recommendations for Future Research

Future research should extend the current investigation to include more subjects with the addition of comparing echocardiographic measurements in relation to levels of injury.

In addition, serial echocardiograms of SCI patients from the time of injury until a couple of years post-injury to determine if, when and how much anatomical change occurs in the left ventricle. Furthermore, serial echocardiograms in quadriplegic patients before, during and after various exercise protocols to demonstrate whether the denervated heart adapts to exercise. Finally, a more definitive investigation would be to complete an autopsy study of the heart in the SCI population and directly determine the anatomical changes in cardiac cells and their arrangement.

APPENDIX

QUESTIONNAIRES

LAY SUMMARY

The aim of this study is to identify structural differences in left ventricular size (the large chamber on the left side of your heart) in relation to lean body mass (weight of your bones and muscles) in the spinal cord injured and intact individuals.

You will undergo an initial screening procedure requiring a resting 12-lead electrocardiogram, blood pressure determination, and a medical examination with muscle and sensory tests to verify level of lesion.

The study will be carried out in two parts. The first part requires the determination of your lean body mass. Three different methods will be used to determine your lean body mass. The first involves skinfold measurements. While you are seated an investigator will measure the thickness of skinfolds in 10 different sites on your body with calipers. This procedure is painless and involves minimal assistance from you.

The second method is underwater weighing and determination of residual volumes. The amount of fluid and air in your body affects the measured weight of your body. To determine the amount of air in your lungs, you will be placed in a body box. A nose clip will be placed on your nose and you will be asked to breath normally for three minutes. Then you will hold your breath for five seconds. Upon completion you will be attached to a mouthpiece and asked to breath normally. You will then be asked to pant three (3) times. This whole procedure will be repeated three to five times. The procedures has minimal risk although you may feel light headed.

Once your residual volume in your lungs is determined, you will be weighted under water. Prior to weighing you will be asked to empty your bladder and bowels and don swimming trunks. You will be placed in a chair and lowered into a tank of water. You will be asked to exhale maximally then allowed to inhale a known quantity of oxygen.

You will then be immersed completely for 10-15 seconds. This will be repeated five to ten times. The third method for lean body mass determination involves drinking deuterium oxide, a natural part of your drinking water. Then a urine sample will be taken 4 hours later. This test will have absolutely no effect on your body, since the deuterium acts like any other water in your system.

The final part of this study is an echocardiogram. Echocardiography is a noninvasive technique used to study heart structure and function. It is based on the recording of high frequency sound waves reflected from the various structures of the heart (similar to the use of sonar in underwater searches). There are no risks and is of minimal inconvenience to you.

All parts of the study will have a physician on-call, an exercise physiologist, physical therapist, and experienced technicians. We will make every effort to prevent physical injury that could result from this research. Compensation for physical injuries incurred as a result of participating in this research is not available. Investigators are prepared to advise you about medical treatment in case of injuries or adverse effects from these procedures which you should report immediately. If hospitalization is necessary, you will have to provide your own insurance coverage.

You must feel free to terminate and withdraw from the study at any time.

If you do not fully understand this explanation of the objective and techniques of this experiment, please ask questions to make this procedure clear.

I have read and understand the description of the study, including the detailed explanation of each procedure. I have been given the opportunity to ask questions and discuss the study and its potential risks and benefits. I understand that my request for my

information that has been gathered will be fulfilled. I understand that I may withdraw from the study at any time. I hereby give my consent to the study.

_____

Name

_____

Date

HEALTH QUESTIONNAIRE

Read the instructions provided and follow directions carefully. All information is strictly confidential and will not be released without your written consent.

Identification

Name _____

Date of Birth _____ Sex _____

Height _____ Weight _____ lbs.

Address _____

_____

Illness and Medical Problems

Have you had any of the following: (Circle A, B, or C)

	Yes	No	Uncertain
Alcoholism	A	B	C
Anemia	A	B	C
Asthma	A	B	C
Bronchitis	A	B	C
Cancer of the:			
Colon	A	B	C
Lung	A	B	C
Other	A	B	C
Cirrhosis - Liver	A	B	C
Colitis	A	B	C
Decubitus Ulcers	A	B	C
Depression	A	B	C
Diabetes	A	B	C
Emphysema	A	B	C
Epilepsy	A	B	C
Frequent Episodes of Bleeding	A	B	C

	Yes	No	Uncertain
Heart Attack	A	B	C
Coronary Disease	A	B	C
Rheumatic Fever	A	B	C
Rheumatic Heart Disease	A	B	C
Heart Valve Problem	A	B	C
Heart Murmur	A	B	C
Enlarged Heart	A	B	C
Heart Rhythm Problems	A	B	C
Other Heart Problems	A	B	C
High Blood Pressure: controlled	A	B	C
High Blood Pressure: uncontrolled	A	B	C
Pneumonia	A	B	C
Polyps in Colon	A	B	C
Stroke	A	B	C
Arthritis	A	B	C
Tuberculosis	A	B	C
Urinary Tract Infection	A	B	C

In the past year, have you had: (Circle A or B)

	Yes	No
Seizures	A	B
Chest Pain on Exertion: (relieved by rest?)	A	B
Shortness of Breath (lying down, relieved by sitting up?)	A	B
Unexplained Weight Loss (more than 10 lbs)	A	B
Unexplained Rectal Bleeding	A	B
Urinary Tract Infection	A	B
Decubitus Ulcers	A	B

	Yes	No
Smoking		
Do you currently smoke cigarettes?	A	B
Do you have a history of cigarette smoking, but stopped?	A	B
Chest X-Ray		
Have you ever had a chest x-ray?	A	B
Have you had an abnormal chest x-ray?	A	B
Have you ever had an EKG?	A	B
Have you had an abnormal EKG?	A	B
Have you ever had an exercise stress test?	A	B

Medications

List all medications you are taking presently, include vitamins, iron, allergy, sleeping pills, ect.

_____

_____

_____

_____

LIFESTYLE QUESTIONNAIRE

Demographic Information

Circle the appropriate response.

How would you describe yourself?

Black Caucasian Latino Oriental American Indian Other

Income

less than $10,000 $25,000 to $40,000

$10,000 to $25,000 More than $40,000

Educational Level

K to 6 7 to 9 10 to 12 College (no degree)

Associate degree Bachelor's degree Graduate Work (no degree)

Master's degree Doctoral degree Professional degree

Family Medical History

Have any of the following had heart disease? (Circle A, B, or C).

	Yes	No	Uncertain
Father	A	B	C
Mother	A	B	C
Brother or Sister	A	B	C

Have any of the following had high blood pressure? (Circle A, B, or C).

	Yes	No	Uncertain
Father	A	B	C
Mother	A	B	C
Brother or Sister	A	B	C

Circle as applicable.

	Yes	No	Uncertain
Father died of heart disease before age 60	A	B	C
Mother died of heart disease before age 60	A	B	C

Have any of the following occurred in your immediate family? (Parents, brothers sisters) Circle A, B, or C.

	Yes	No	Uncertain
Sickle Cell Anemia	A	B	C
Diabetes	A	B	C
Cerebrovascular disease (stroke)	A	B	C
Cancer	A	B	C

Smoking

Have you ever smoked cigarettes? Yes_____ No_____ (If no, skip this section.)

How old were you when you began to smoke? _____

Have you quit smoking? Yes_____ No_____

If you have quit, how much did you smoke?

1-20 per day _____ 21-40 _____ 41-60 _____ more than 60 _____

For how many years had you smoked prior to quitting?

less than a year _____ 1-4 yrs. _____ 5-9 yrs. _____ 10-14 yrs. _____

15 - 19 yrs. _____ 20 or more yrs. _____

If you still smoke, how much do you smoke on the average?

1-20 per day _____ 21-40 _____ 41-60 _____ more than 60 _____

Do you inhale? Yes _____ No _____

Have you ever tried to stop smoking Yes _____ No _____

Do you smoke cigars? Yes _____ No _____

How many cigars to you smoke per day?

1-2 _____ 3-5 ____ 6-10 ____ More than 10 _____

Do you inhale? Yes _____ No _____

Do you smoke a pipe? Yes _____ No _____

How many pipes per day do you smoke?

1-2 _____ 3-5 _____ 6-10 _____ More than 10 _____

Do you inhale? Yes _____ No _____

Sport	# of years	Approximate age when participating
Golf	_____	_____
Field Hockey	_____	_____
Ice Hockey	_____	_____
Skiing	_____	_____
Volleyball	_____	_____
Other	_____	_____

Circle A for those activities in which you participated during the past year. In addition, indicate in the space provided, the approximate number of times you engaged in these activities, the number of minutes per session, and the intensity of your participation.

	Yes	# of times per month	Approx. Min. per session	How vigorous 1=low; 3=high
Lawn work and yard care	A	_____	_____	_____
Work in garden	A	_____	_____	_____
Archery	A	_____	_____	_____
Skating	A	_____	_____	_____
Dancing	A	_____	_____	_____
Fishing	A	_____	_____	_____

	Yes	# of times per month	Approx. Min. per session	How vigorous 1=low; 3=high
Hunting	A	_____	_____	_____
Basketball	A	_____	_____	_____
Volleyball	A	_____	_____	_____
Softball or Baseball	A	_____	_____	_____
Golf	A	_____	_____	_____
Handball, Tennis, Racquetball, or Squash	A	_____	_____	_____
Badminton	A	_____	_____	_____
Table Tennis	A	_____	_____	_____
Sailing	A	_____	_____	_____
Canoeing or rowing	A	_____	_____	_____
Downhill Skiing	A	_____	_____	_____
Cross Country Skiing	A	_____	_____	_____
Water Skiing	A	_____	_____	_____
Walking/Strolling	A	_____	_____	_____
Housework	A	_____	_____	_____
Calisthenics or Exercises	A	_____	_____	_____
Swimming	A	_____	_____	_____
Bicycling	A	_____	_____	_____
Running/Jogging/ Wheelchair	A	_____	_____	_____
Bowling	A	_____	_____	_____
Pool/Billiards	A	_____	_____	_____
Video Games	A	_____	_____	_____

Other Activities

Are you presently employed? Yes_____ No_____

If employed, how far do you travel to work in one day? _____ Miles

What is your usual mode of transportation? _____

How would you estimate the level of occupational physical activity?

Inactive _____

Moderately Active _____

Very Active _____

What is your occupation? _____

Emotional Well-Being

Circle the response which most appropriately describes you.

	Never	Seldom	Occasionally	Often
I feel sad or depressed.	A	B	C	D
I am under considerable stress.	A	B	C	D
I feel tense and anxious.	A	B	C	D
I worry about things	A	B	C	D
I am hard driving.	A	B	C	D
I have intense desire to achieve	A	B	C	D
I am a restless sleeper.	A	B	C	D
I am worried about my health.	A	B	C	D
I feel like I cannot cope with daily stress.	A	B	C	D
I feel like I need to get away	A	B	C	D

Tetraplegics and Paraplegics, please fill out the following information.

Level of injury: _____ Sensory Level: _____

Date of injury: _____ Functional Level: _____

Cause of injury: _____

Initial hospitalization

Name of facility: _____

Location: _____

Surgeries: _____

_____

Complications: _____

_____

Did you receive rehabilitation?

Name of facility: _____

Location: _____

Length of Stay: _____

Have you received any vocational training?

Name of facility: _____

Location: _____

Type of Training: _____

Other hospitalizations since your injury?

Reason for hospitalization: _____

When: _____

Number of times: _____

Can you drive?

Car adaptations: _____

Type of vehicle: _____

Where did you learn? _____

Spasticity

Do you spasticity?_____

Are you on any medications to control your spasticity?_____

If so, what are they and the dosages_____

_____

_____

Severity of spasms (Please describe)_____

_____

_____

ADL's Skills

Do you require assistance in any of the following:

Transfers	Yes _____	No _____
Dressing	Yes _____	No _____
Catheritizations	Yes _____	No _____
Suppositories	Yes _____	No _____
Wheelchair propulsion	Yes _____	No _____

How often do you do a sitting push-up? _____

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