ABSTRACT

KARLSDOTTIR, A. E. Left ventricular function during aerobic and resistance exercise. MS in Adult Fitness/Cardiac Rehabilitation, December 2000, 63pp. (C.Foster).

Resistance exercise (RE) has become an important component of cardiac rehabilitation programs. However, the potential for exaggerated BP responses during RE raises concerns about the stability of LV function during RE. To address this, we studied healthy controls (HC) (N = 12, 8M/4F), patients with stable coronary artery disease (CAD) (N = 12, 11M/1F), and patients with stable heart failure (CHF) (N = 12, 7M/5F). Ss were studied during upright cycling at 90% of ventilatory threshold and during 1 set of 10 repetitions of RE (leg press, shoulder press, and biceps curls) in the upright posture. Left ventricular function was measured by echocardiography, BP was measured by auscultation, and HR by radiotelemetry at rest and peak exercise. Despite significant hemodynamic changes from rest to peak, the LV function remained stable with no significant changes from rest to peak in LV ejection fraction, end systolic and end diastolic dimensions, or systolic and diastolic wall thickness. There was no interaction between exercise type and group; all groups showed fundamentally similar behavior patterns during all exercises. This suggests that RE is as safe as aerobic steady-state exercise in these patient groups and may be included in their cardiac rehabilitation programs.

LEFT VENTRICULAR FUNCTION DURING AEROBIC AND RESISTANCE EXERCISE

A MANUSCRIPT STYLE THESIS PRESENTED

TO

THE GRADUATE FACULTY

UNIVERSITY OF WISCONSIN-LA CROSSE

IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE

MASTER OF SCIENCE DEGREE

BY

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DECEMBER 2000

COLLEGE OF HEALTH, PHYSICAL EDUCATION, AND RECREATION

UNIVERSITY OF WISCONSIN-LA CROSSE

THESIS FINAL ORAL DEFENSE FORM

Candidate: Arna E. Karlsdottir

We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree:

Master of Science in Adult Fitness / Cardiac Rehabilitation

The candidate has successfully completed the thesis final oral defense.

Thesis Committee Chairperson Signature — Date: 8/10/00

Thesis Committee Member Signature — Date: 8/10/00

Thesis Committee Member Signature — Date: 8-10-00

This thesis is approved by the College of Health, Physical Education, and Recreation.

Associate Dean, College of Health, Physical Education, and Recreation — Date: 8/23/00

Director of University Graduate Studies — Date: 8-23-00

ACKNOWLEDGEMENTS

This thesis project took a long time and many hurdles had to be passed. This could not have been done without the help of many people.

First and most of all I would like to thank my thesis chair, Dr. Carl Foster, for the enormous time and effort you put in throughout this thesis project. Your support, encouragement and humor kept me afloat during tough times. You should be awarded for your patience! Thank you very much.

Special thanks to my other committee members, to Dr. John P. Porcari for your valuable input and support throughout this project, and to Dr. Karen Palmer McLean for the time you gave me working on editorial revisions.

Thank you to my co-investigators at Franciscan Skemp Healthcare, Roseanne White-Kube, echotechnician, for acquiring and reading all the echo images and Dr. Richard Backes, cardiologist, for giving your time during exercise testing and for reading the echo images.

Thank you to the participants of this study. Thank you for giving me your time and for showing up so early in the mornings; this could not have been done without you.

Finally, extensive thanks to my family and friends for both encouraging me to come here and supporting me throughout the year.

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INTRODUCTION

Background

Cardiac rehabilitation recommendations have changed a great deal over the last 3 decades. In contrast to the almost complete bed rest prescribed 30 years ago for patients during the first weeks following a cardiac event, contemporary cardiac rehabilitation starts much earlier and progresses more rapidly. Earlier mobilization of patients enables them to return to work and recreational activities much sooner and minimizes the impact of heart disease on their quality of life.

Steady-state exercise has proven to be beneficial and safe for cardiac patients and is the type of exercise training that is most widely used. The focus of recent studies has been to determine the additional types of exercise that could be added to steady-state aerobic exercise to further increase functional capacity in a shorter time period without increasing the risk of cardiac complications. Meyer et al.[1] have demonstrated that interval training is well tolerated by coronary artery bypass graft patients and accelerates the rate of recovery.

Resistance training has traditionally been contraindicated for cardiac patients. It was not even mentioned in an exercise prescription chapter from 1980.[2] However, since it is clear that activities of daily living require both endurance capacity and muscle strength, recent studies have been conducted to examine the effects of resistance training on cardiac patients. The results have shown that resistance training appears to be safe for

low- to intermediate-risk cardiac patients. The hemodynamic loads are similar to those observed during traditional steady-state exercise training.[3-8] A combination of endurance and resistance training has been shown to enhance the recovery of functional capacity and is now widely recommended for cardiac rehabilitation patients.

Traditionally it was thought that rehabilitation was not of benefit to patients with congestive heart failure (CHF) because exercise placed too great of a strain on their heart muscle. However, recent studies have shown that patients with stable CHF benefit from exercise training, just as low- to intermediate-risk cardiac patients do. Patients with CHF have been shown to benefit from low-intensity endurance training and show increased exercise capacity, apparently because of an increase in mitochondrial density and oxidative capacity of the skeletal muscles. They also demonstrate a shift from type II to type I muscle fiber distribution after training.[9,10] Further, rehabilitation has been shown to improve the quality of life for patients with CHF.[11,12] Finally, interval training has been shown to be well tolerated by patients with CHF and to result in greater improvements in functional capacity[13], without inappropriately stressing the left ventricle.[14]

The safety of resistance training for CHF patients is still in question. It is known that endurance training alone does not enhance skeletal muscle strength.[15] On the other hand, structural and metabolic changes in the skeletal muscle account for much of the decrease in exercise capacity in patients with CHF.[16-18] Accordingly, resistance training would be expected to benefit this patient group. Recent studies including both endurance and resistance training for patients with CHF, have shown that both types of training are

well tolerated and result in an increase in muscular strength and endurance.[19-21] However, resistance training is still generally contraindicated in patients with CHF because the potential elevation in afterload during lifting is considered dangerous for this group. Limited data regarding central hemodynamics in normals[22] and in patients with heart disease[23] suggest that afterload remains high during upper-extremity exercise. At the same time, recent measurements of central hemodynamics during lower body resistance exercise in patients with CHF suggest an appropriate decrease in afterload.[24,25] Further, measures of left ventricular (LV) function in patients with coronary artery disease (CAD) during isometric weight lifting do not suggest serious dysfunction.[26]

The results of interval training studies imply that patients with CHF seem to be able to tolerate much higher exercise intensities than was previously thought; therefore, contemporary guidelines may underestimate their capacity. Since upper-body resistance exercise may further contribute to functional recovery in patients with CHF, this study was designed to evaluate the stability of LV function during upper and lower body resistance exercise in patients with CHF in comparison to cycle ergometry.

METHODS

The study protocol was approved by both the University of Wisconsin-La Crosse and Fransiscan Skemp Healthcare Institutional Review Boards for the Protection of Human Subjects prior to any contact with participants. Each subject provided informed consent (Appendix A) prior to participation. All testing was conducted at Franciscan Skemp Healthcare except for the incremental exercise test on the healthy subjects.

Subjects

Three groups of male and female subjects, aged 23 and older, were recruited for this study. Physical characteristics of all subjects are summarized in Table 1.

TABLE I Mean (± SD) Physical Characteristics of Participants

	CHF group	CAD group	Healthy group
Age (yr)	62 ± 9	69 ± 9	29 ± 8
Height (cm)	171 ± 10	177 ± 8	177 ± 7
Weight (kg)	85 ± 15	85 ± 14	75 ± 14
Maximum exercise capacity			
Power Output (W)	90 ± 34	132 ± 37	267 ± 58
Oxygen uptake (ml/kg/min)	15.3 ± 4.2	21.2 ± 3.7	41.8 ± 7.7
Heart rate (beats/min)	148 ± 33	140 ± 12	178 ± 11
Ventilatory Threshold			
Power Output (W)	50 ± 25	69 ± 22	145 ± 50
Oxygen uptake (ml/kg/min)	10.2 ± 2.5	13.5 ± 2.5	27.1 ± 7.1
90% of Ventilatory Threshold			
Work rate (W)	39 ± 19	59 ± 19	129 ± 44
Oxygen uptake (ml/kg/min)	9.1 ± 2.2	12.1 ± 2.3	24.2 ± 6.4

The group characteristics were as follows:

CHF group. Twelve subjects (7 males and 5 females) having stable CHF of NYHA class I - II were recruited from the Cardiology Department at Franciscan Skemp Healthcare in La Crosse, WI and from the phase III Cardiac Rehabilitation Program at the University of Wisconsin-La Crosse. The average (± SD) sitting resting LV ejection fraction was 35 ± 8%. All patients had experienced ≥ 1 episode of acute heart failure, but had been

clinically stable for ≥ 8 wks. Six patients were participants in a regular exercise training program prior to entering the study.

CAD group. Twelve clinically stable patients (11 males and 1 female) with ischemic cardiovascular disease but with minimal LV dysfunction and no history of CHF were recruited from the phase III Cardiac Rehabilitation Program at Franciscan Skemp Healthcare in La Crosse, WI or from the phase III Cardiac Rehabilitation Program at the University of Wisconsin-La Crosse. The average (± SD) sitting resting LV ejection fraction was 51 ± 11%.

Healthy group. Twelve clinically healthy subjects (8 males and 4 females) were recruited from the faculty and students of the University of Wisconsin-La Crosse. The average (± SD) upright resting LV ejection fraction was 63 ± 8%.

The CHF/CAD patients were on the following medications: angiotensin converting enzyme inhibitors 9/1, beta blockers 8/7, alpha blockers 0/1, calcium channel blockers 1/0, diuretics 9/1, digitalis 8/0, nitrates 1/4, anticoagulants 11/9 and anti-lipidemics 6/9. All patients continued with their usual medications during all procedures, as the purpose of this study was to evaluate responses under conditions representative of rehabilitation program conditions.

Two of the patients with CHF were in atrial fibrillation that was well controlled and did not interfere with any of the testing. No patient had angina, ECG-evidence of ischemia, hemodynamically significant valvular disease, serious ventricular arrhythmias, orthopedic disorders, neurological disorders or peripheral vascular disease that limited exercise.

Tests Prior to the Study

Resting spirometry was performed to exclude subjects with serious pulmonary obstructive disease. To be able to estimate the work load during cycling, and to rule out possible ischemia, all subjects initially underwent a maximal incremental cycle ergometer exercise test on an electrically braked cycle ergometer (Lode, Groningen NL) with measures of respiratory metabolism using open-circuit spirometry (Quinton Q-Plex, Seattle, WA). Participants were seated for the first 3 min to obtain resting oxygen uptake. After a 3-min warm-up of unloaded pedaling, the power output was increased by 10–25 W *every min*, depending on the participant. During this test the 12-lead ECG and blood pressure were monitored according to conventional guidelines.

Protocol and Procedures

The subject groups were tested in the following order: healthy controls, stable cardiac patients, and CHF patients. This sequencing was designed to insure that the technical hurdles to acquiring echocardiographic images during cycle and resistance exercise had been solved prior to studying the potentially more fragile patients with CHF. All participants were tested from 1–7 days after the incremental exercise test. Both the cycle resistance protocols were designed to be similar to a regular exercise training session. Subjects completed the 15-min cycling protocol first. A 3-min warm-up period on the cycle at about 35% of their maximal exercise capacity was followed by 12 min of cycling at a power output requiring approximately 90% of their ventilatory threshold, as determined from the incremental exercise test. After cycling they rested briefly. They then performed the resistance exercises in the following order: leg press, using a

machine, and shoulder press and biceps curls using free weights. The shoulder press and biceps curls were done unilaterally to facilitate echocardiography and blood pressure measurements. Prior to each resistance exercise, the 1 repetition maximum (RM) was determined. The exercise required for the 1 RM determination also served as a warm-up. Each participant then performed 1 set of 10 repetitions of each of the 3 exercises at a resistance of 60-70% of their 1 RM.

Measurements Taken During Testing

Resting blood pressure, heart rate, and echocardiographic images were obtained while sitting upright on the bicycle. Blood pressure and heart rate were measured after the 3rd min of warm-up and at the 5^{th}, 10^{th} and 15^{th} min of the cycling exercise bout. Echocardiographic images were obtained at the 5^{th}, 10^{th}, and 15^{th} min. Heart rate, blood pressure, and echocardiographic images were obtained at rest, prior to the resistance exercises, with the participant holding the weight. Heart rate, blood pressure, and echocardiographic images were also obtained during the last 5 repetitions of each 10-repetition set. The blood pressure was measured on the non-exercising arm (left arm).

Echocardiographic Technique

The LV functional images were acquired by using two-dimensional echocardiography (Acuson Corporation, Mountain View, CA). The left ventricular end-diastolic and end-systolic dimensions were measured from the parasternal long-axis view just below the level of the mitral valve. Anteroseptal and posterior systolic and diastolic wall thickness was also measured from the parasternal long axis just below the level of the mitral valve. Ejection fraction was calculated according to Schiller et al.[27] We chose

to make echocardiographic measurements in the upright position using the parasternal long axis view instead of the more echocardiographically optimal apical 4 chamber view in the supine left lateral decubitis position because we were primarily interested in optimizing the hemodynamic responses to exercise and in simulating the circumstances of rehabilitation exercise session. In our view this need took precedence over more optimal imaging conditions and is accepted as a limitation of the study.

Statistical Analysis

Statistical comparison between groups, exercise type, and trials (rest and peak) were made using a 3-way ANOVA with repeated measures. Because there were no significant 3-way interactions, a 2-way ANOVA with repeated measures was conducted for each group comparing exercise type and trials. Specific pairwise comparisons for trials (rest versus peak) were made using Tukey's test. Alpha was set at 0.05 to achieve statistical significance.

RESULTS

All participants completed the testing procedures without difficulties (e.g. untoward events). The patients with CHF complained of moderate muscle tiredness and 3 patients experienced moderate shortness of breath during steady-state cycling, which was managed with brief reductions in workload between echocardiographic images. Mean responses (± SD) for each group are presented in Tables II, III and IV.

TABLE II Mean (± SD) hemodynamic and LV responses during cycling, leg press (LP), shoulder press (SP) and biceps curls (BC) in CHF group

	Cycle		LP		SP		BC	
	Rest	Peak	Rest	Peak	Rest	Peak	Rest	Peak
Heart rate (beats/min)	80 ± 11	113 ± 18	88 ± 13	94 ± 11	88 ± 10	100 ± 15	90 ± 10	96 ± 12
Systolic blood pressure (mmHg)	120 ± 17	146 ± 21	122 ± 18	136 ± 19	121 ± 14	137 ± 16	121 ± 14	137 ± 19
Diastolic blood pressure (mmHg)	77 ± 10	78 ± 11	82 ± 8	91 ± 14	81 ± 11	100 ± 12	84 ± 12	97 ± 17
Mean arterial pressure (mm Hg)	91 ± 11	100 ± 14	95 ± 10	106 ± 14	95 ± 10	112 ± 12	96 ± 11	110 ± 16
Ejection fraction (%)	35 ± 8	42 ± 11	38 ± 9	37 ± 10	38 ± 10	35 ± 7	35 ± 9	36 ± 11
End systolic dimension (mm)	46 ± 7	42 ± 7	44 ± 7	45 ± 8	44 ± 7	44 ± 7	44 ± 8	44 ± 8
Systolic post wall thickness (mm)	13 ± 2	12 ± 3	13 ± 2	12 ± 2	13 ± 3	13 ± 2	12 ± 2	12 ± 2
Systolic ant-sept wall thickness (mm)	13 ± 2	13 ± 3	14 ± 3	13 ± 3	12 ± 3	12 ± 2	12 ± 3	13 ± 3
End diastolic dimension (mm)	55 ± 5	54 ± 6	54 ± 7	55 ± 8	54 ± 6	53 ± 7	53 ± 7	53 ± 7
Diastolic post wall thickness (mm)	10 ± 2	11 ± 2	11 ± 2	11 ± 1	11 ± 2	12 ± 2	11 ± 2	12 ± 2
Diastolic ant-sept wall thickness (mm)	11 ± 2	11 ± 2	11 ± 3	11 ± 2	11 ± 3	12 ± 3	11 ± 3	12 ± 2

TABLE III Mean (± SD) hemodynamic and LV responses during cycling, leg press (LP), shoulder press (SP) and biceps curls (BP) in CAD group

	Cycle		LP		SP		BC	
	Rest	Peak	Rest	Peak	Rest	Peak	Rest	Peak
Heart rate (beats/min)	79 ± 11	109 ± 11	82 ± 13	95 ± 15	88 ± 13	96 ± 13	89 ± 12	94 ± 12
Systolic blood pressure (mmHg)	139 ± 16	171 ± 20	149 ± 16	164 ± 15	145 ± 17	167 ± 14	145 ± 15	158 ± 13
Diastolic blood pressure (mmHg)	88 ± 7	82 ± 11	93 ± 8	97 ± 10	92 ± 8	111 ± 6	92 ± 7	107 ± 9
Mean arterial pressure (mmHg)	105 ± 6	112 ± 12	112 ± 8	120 ± 9	110 ± 9	130 ± 6	110 ± 6	124 ± 6
Ejection fraction (%)	51 ± 11	57 ± 8	56 ± 8	55 ± 9	59 ± 12	53 ± 13	53 ± 12	54 ± 7
End systolic dimension (mm)	33 ± 6	30 ± 6	31 ± 5	33 ± 5	29 ± 8	30 ± 7	30 ± 6	29 ± 5
Systolic post wall thickness (mm)	14 ± 2	14 ± 3	14 ± 3	14 ± 2	15 ± 3	14 ± 3	15 ± 3	15 ± 2
Systolic ant-sept wall thickness (mm)	16 ± 3	16 ± 3	16 ± 3	16 ± 4	17 ± 3	16 ± 3	17 ± 3	17 ± 4
End diastolic dimension (mm)	44 ± 5	45 ± 6	46 ± 7	47 ± 7	43 ± 6	41 ± 6	42 ± 6	41 ± 5
Diastolic post wall thickness (mm)	11 ± 1	11 ± 2	11 ± 2	12 ± 2	13 ± 1	13 ± 2	13 ± 2	12 ± 2
Diastolic ant-sept wall thickness (mm)	13 ± 1	13 ± 2	13 ± 3	14 ± 3	14 ± 2	14 ± 2	14 ± 3	14 ± 3

TABLE IV Mean (± SD) hemodynamic and LV responses during cycling, leg press (LP), shoulder press (SP) and biceps curls (BC) in healthy group

	Cycle		LP		SP		BC	
	Rest	Peak	Rest	Peak	Rest	Peak	Rest	Peak
Heart rate (beats/min)	68 ± 12	135 ± 16	84 ± 14	117 ± 17	98 ± 14	110 ± 20	87 ± 12	109 ± 16
Systolic blood pressure (mmHg)	131 ± 14	175 ± 18	142 ± 21	173 ± 17	151 ± 8	166 ± 19	136 ± 18	166 ± 18
Diastolic blood pressure (mmHg)	89 ± 9	68 ± 12	86 ± 11	109 ± 9	95 ± 11	114 ± 11	90 ± 8	117 ± 9
Mean arterial pressure (mmHg)	106 ± 15	104 ± 12	105 ± 11	130 ± 9	113 ± 9	132 ± 10	105 ± 11	133 ± 11
Ejection fraction (%)	63 ± 8	69 ± 7	60 ± 10	59 ± 8	66 ± 5	65 ± 11	63 ± 10	58 ± 6
End systolic dimension (mm)	28 ± 6	25 ± 6	29 ± 5	28 ± 4	25 ± 5	25 ± 8	28 ± 6	27 ± 6
Systolic post wall thickness (mm)	15 ± 4	15 ± 3	15 ± 3	15 ± 4	15 ± 4	14 ± 3	14 ± 3	14 ± 3
Systolic ant-sept wall thickness (mm)	14 ± 3	15 ± 2	15 ± 3	15 ± 3	15 ± 3	15 ± 3	14 ± 3	15 ± 4
End diastolic dimension (mm)	44 ± 7	43 ± 7	44 ± 6	43 ± 6	39 ± 6	40 ± 8	42 ± 6	40 ± 8
Diastolic post wall thickness (mm)	12 ± 3	12 ± 2	11 ± 3	12 ± 2	12 ± 2	11 ± 2	11 ± 2	12 ± 1
Diastolic ant-sept wall thickness (mm)	11 ± 2	12 ± 2	12 ± 3	12 ± 2	12 ± 2	13 ± 3	12 ± 2	12 ± 2

There were no significant groups x exercise type x trials (rest and peak) interactions. We interpret this as indicative of the same pattern of response in all 3 groups. The heart rate increased significantly from rest to peak during all exercises in the healthy group and in the CAD group. In the CHF group, the heart rate increased significantly from rest to peak during the cycle exercise and during the shoulder press exercise, but not during the leg press or biceps curl exercises. The healthy group had significantly larger increases from rest to peak than the other two groups. During cycling the heart rate increased more than during the resistance exercises (Figure 1).

The systolic blood pressure increased significantly from rest to peak in all groups during all exercises. The systolic blood pressure during cycle exercise showed the greatest increase from rest to peak exercise in both the healthy and CAD groups. There was also a tendency for the systolic blood pressure to show the greatest increase during cycle exercise in the CHF group ($p = 0.08$). When comparing groups there was a significant difference in systolic blood pressure values, with the CHF group having significantly lower values than the CAD or healthy groups (Figure 2).

In the healthy group, there was a significant decrease in diastolic blood pressure from rest to peak cycle exercise while there was a significant increase during the 3 resistance exercises. In the CAD group, the diastolic blood pressure increased during the shoulder press and biceps curl exercises from rest to peak while the diastolic blood pressure remained constant during the other two exercises. The diastolic blood pressure during the resistance exercises increased significantly from rest to peak in the CHF

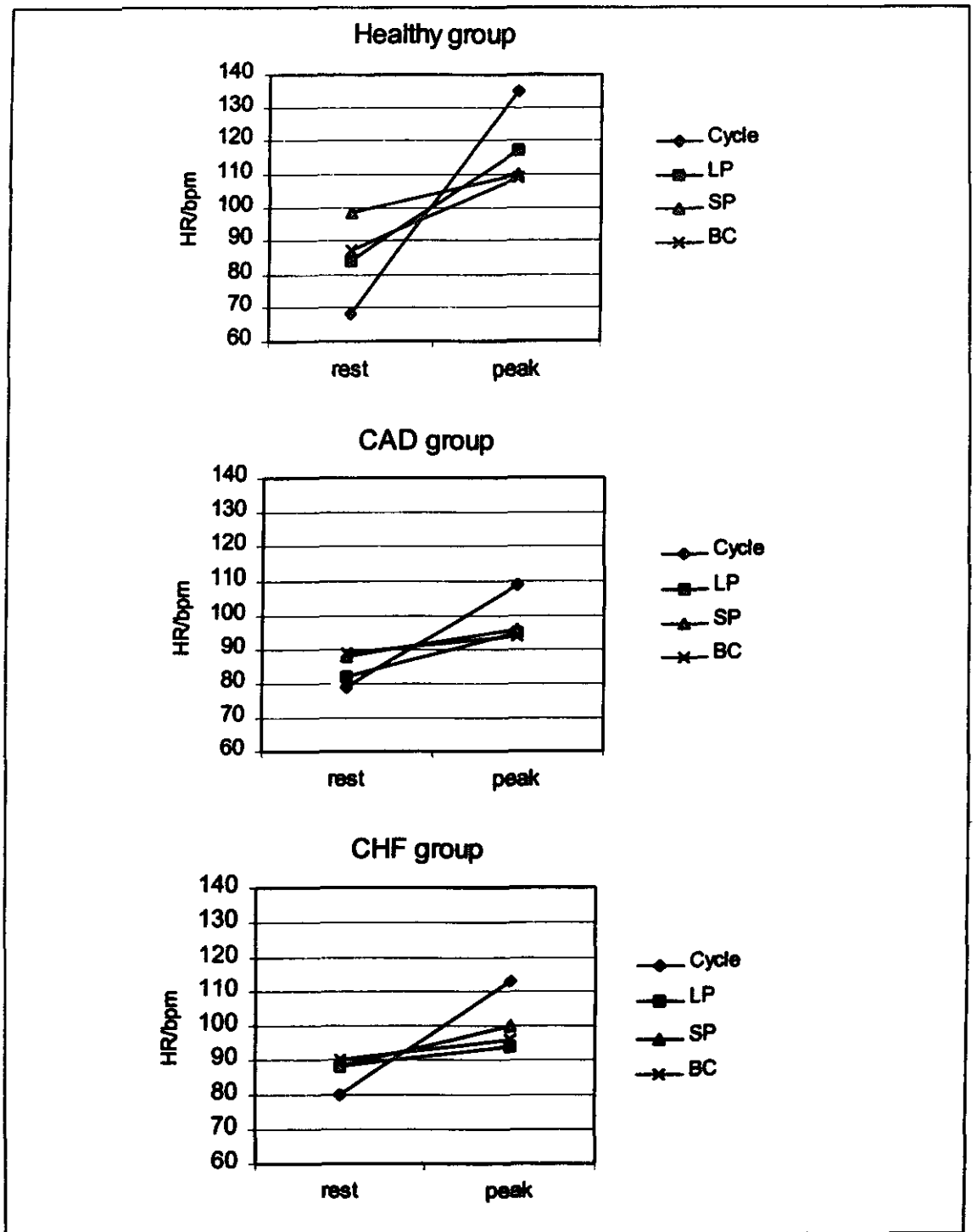

FIGURE 1. Heart rate (HR) responses during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

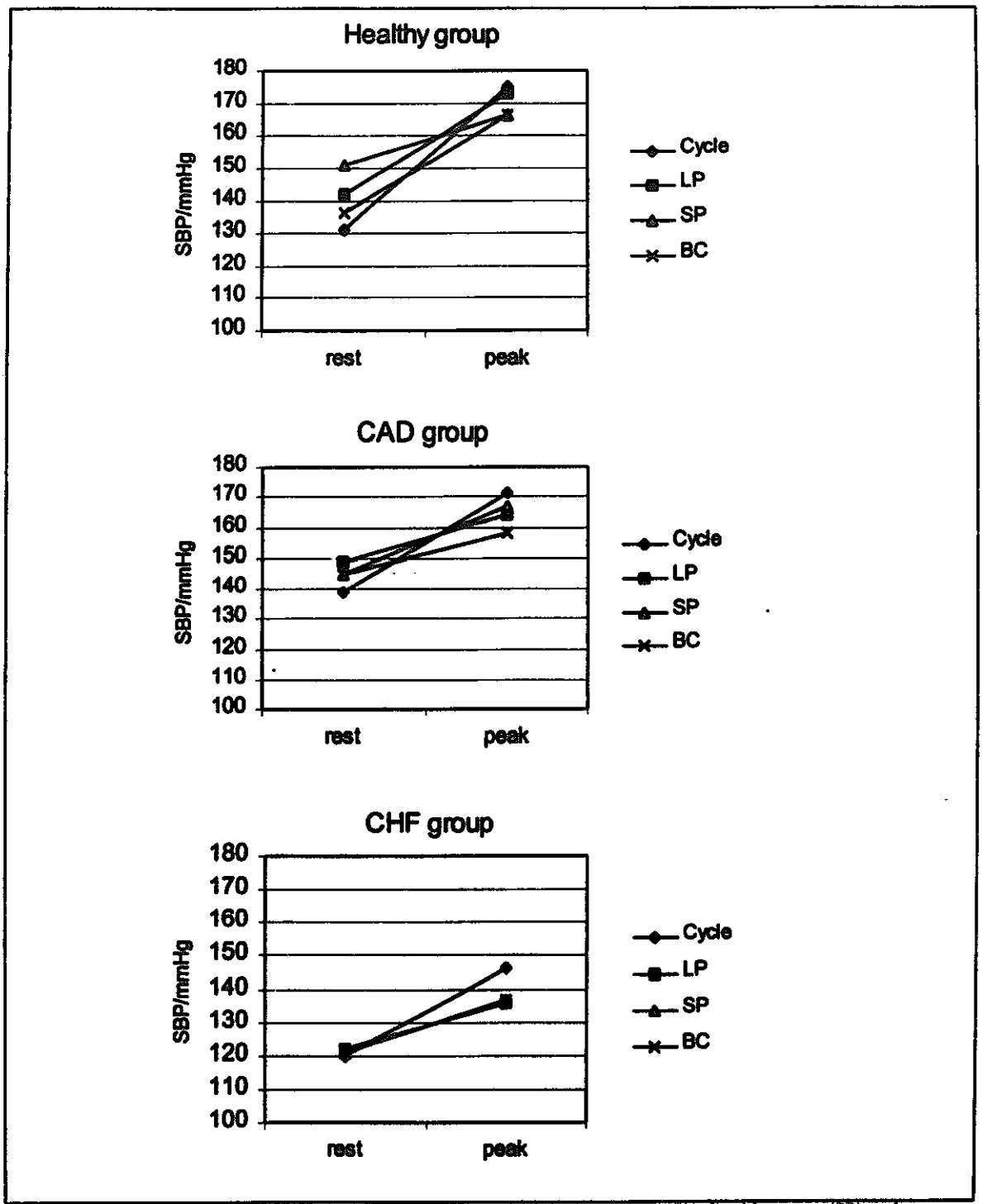

FIGURE 2. Systolic blood pressure (SBP) responses during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

group, while it did not change significantly during the cycle exercise. The diastolic blood pressure change during shoulder press was significantly different from the cycle exercise (Figure 3).

The calculated mean arterial pressure increased significantly from rest to peak during all exercises for both the CAD and the CHF groups. In the healthy group, the mean arterial pressure remained constant during the cycle exercise but increased significantly during the resistance exercises. In the CAD group, the mean arterial pressure was significantly lower during cycling than during shoulder press. When comparing groups, overall the mean arterial pressure in the CHF group was significantly lower than in the other two groups (Figure 4).

The LV ejection fraction did not change significantly from rest to peak during any of the exercises in any of the groups. However, there was a tendency ($p = 0.055$) for the cycle exercise LV ejection fraction to increase and the biceps curl fraction to decrease in the healthy group. In the CAD group, although there were no significant changes in LV ejection fraction, there was an interaction in the behavior of LV ejection fraction during cycling exercise (increasing) and the shoulder press (decreasing). In the CHF group, the cycle exercise LV ejection fraction response was significantly different from the resistance exercises, with a tendency to increase, but was unchanged during the resistance exercises. When comparing groups, the CHF group had significantly lower LV ejection fractions relative to the other two groups. The LV ejection fraction in the CAD group was also significantly lower than in the healthy group (Figure 5).

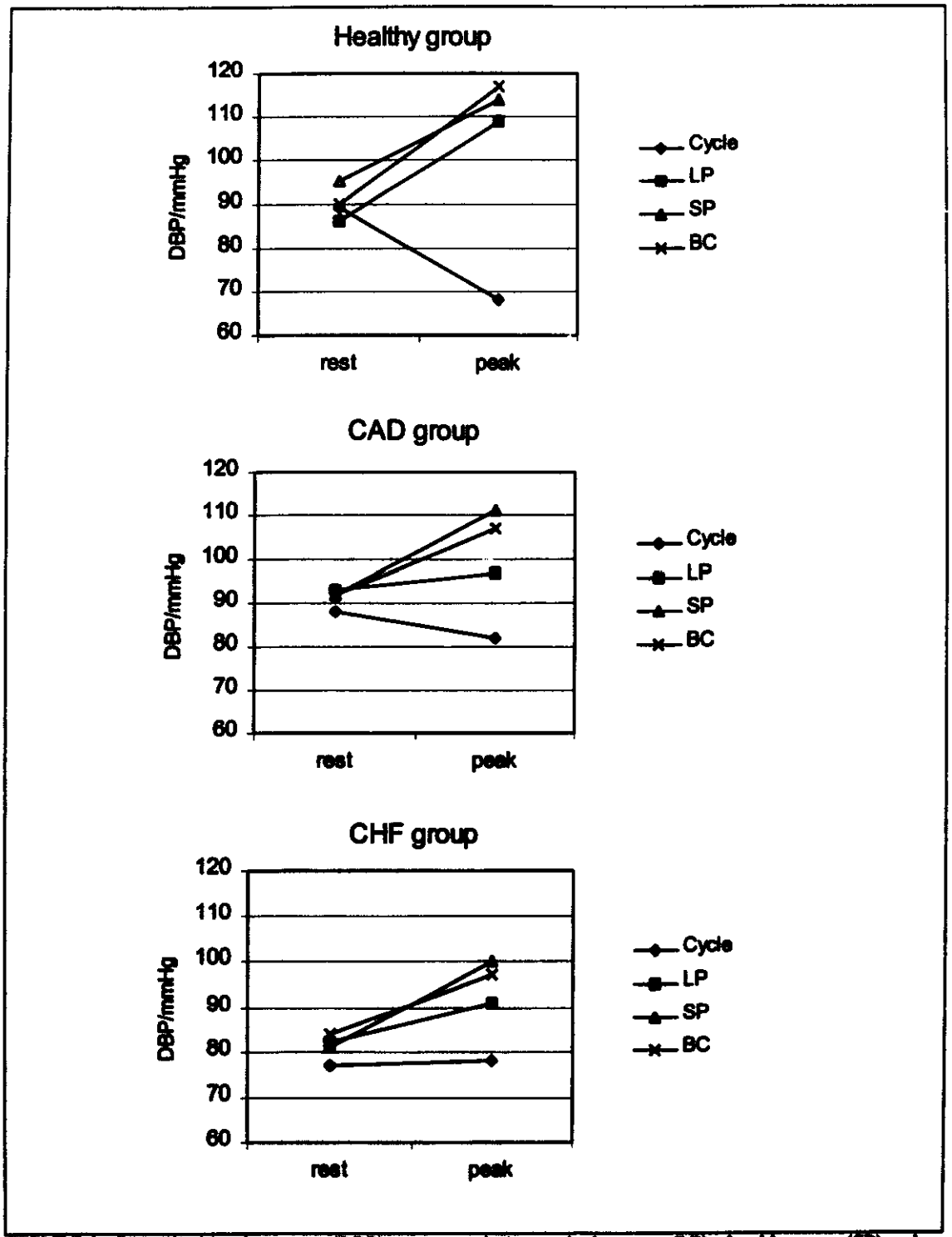

FIGURE 3. Diastolic blood pressure (DBP) responses during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise, in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

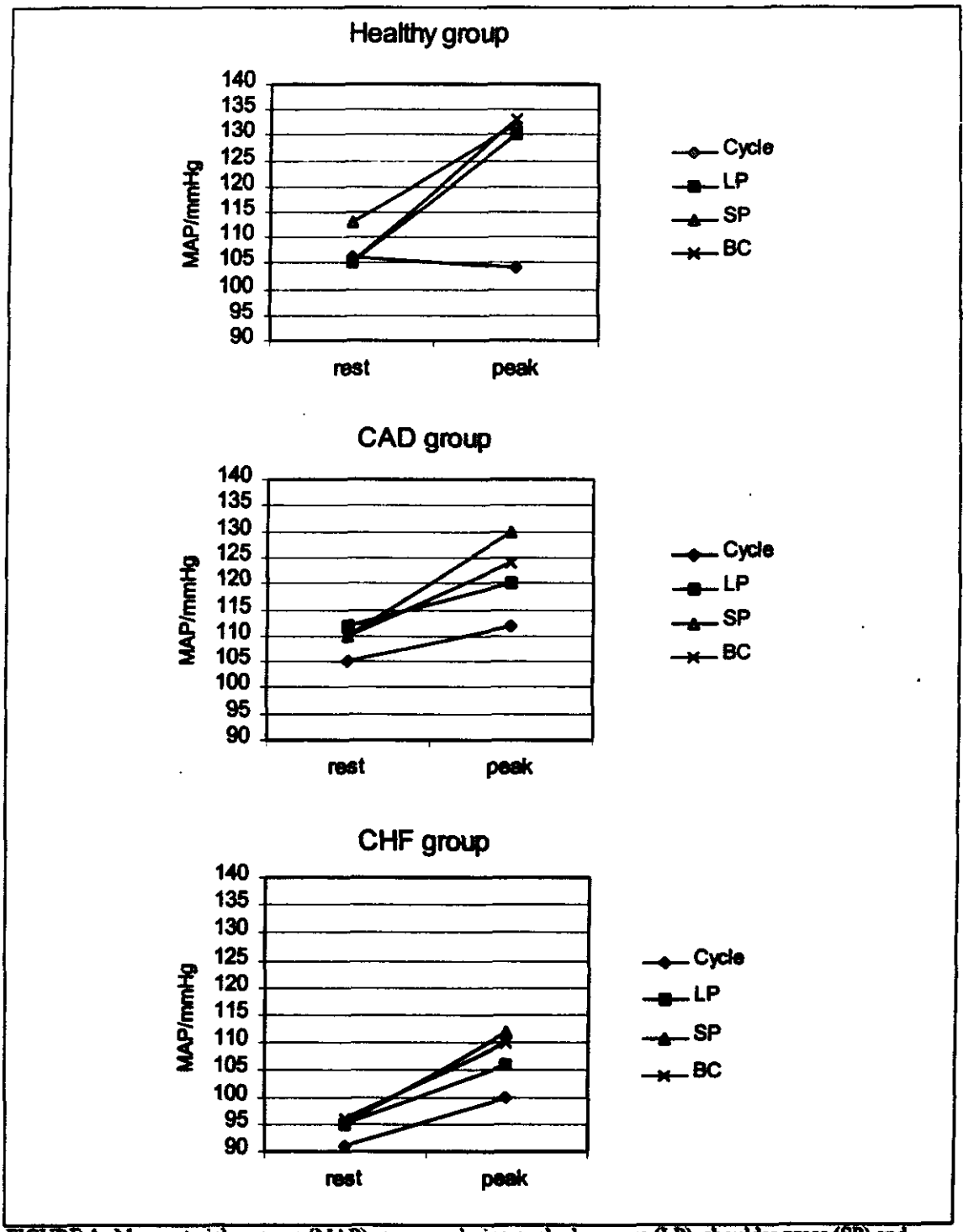

FIGURE 4. Mean arterial pressure (MAP) response during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise, in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

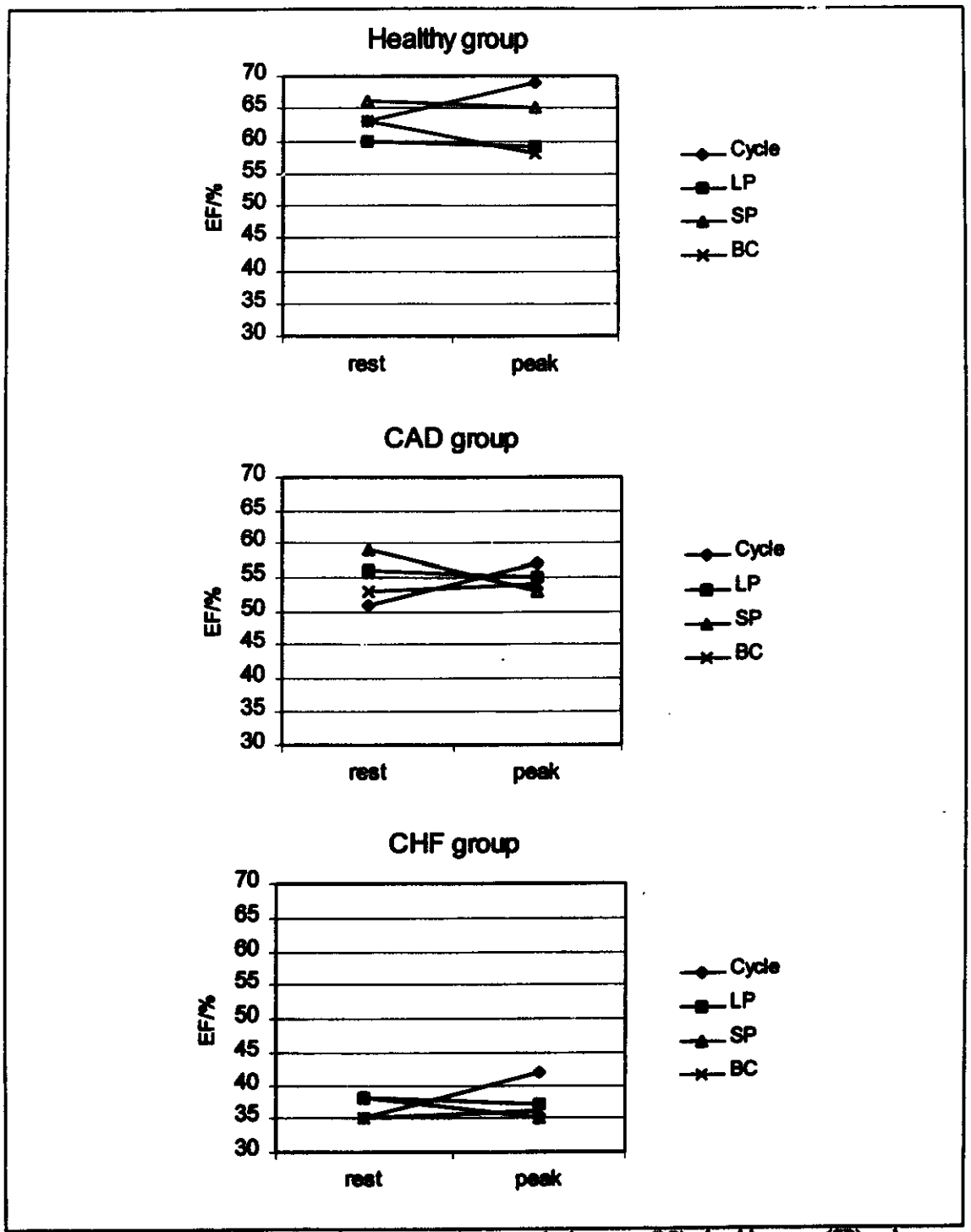

FIGURE 5. Left ventricular ejection fraction (EF) during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

The end-systolic dimension showed no significant change from rest to peak in any of the groups, although there was a tendency for it to decrease in the healthy group ($p = 0.077$). In the CHF group, the end systolic dimension also showed a tendency to decrease during cycle exercise ($p = 0.054$). There was a significant difference in the mean end-systolic dimension, with the CHF group showing significantly greater values (Figure 6).

The end-diastolic dimension showed no significant trials effect in any group. However, the CHF group showed significantly larger end-diastolic dimensions than the other two groups (Figure 7).

The systolic posterior wall thickness did not change significantly from rest to peak during any exercise in any group. However, the CHF group did show a tendency to decrease regardless of exercise type ($p = 0.064$). When comparing groups, the CHF group had significantly smaller posterior wall thickness than the other two groups (Figure 8).

The systolic anterior-septal wall thickness showed no significant change between trials, trials by exercise type, or for groups. The only significant difference was that the CHF group had significantly smaller systolic anterior-septal thickness than the other two groups (Figure 9).

The diastolic posterior wall thickness only changed significantly from rest to peak in the CHF group (increased) with all exercise types behaving the same. In the healthy group, the diastolic posterior wall thickness during shoulder press exercise showed a tendency to decrease ($p = 0.058$). When comparing groups there was a

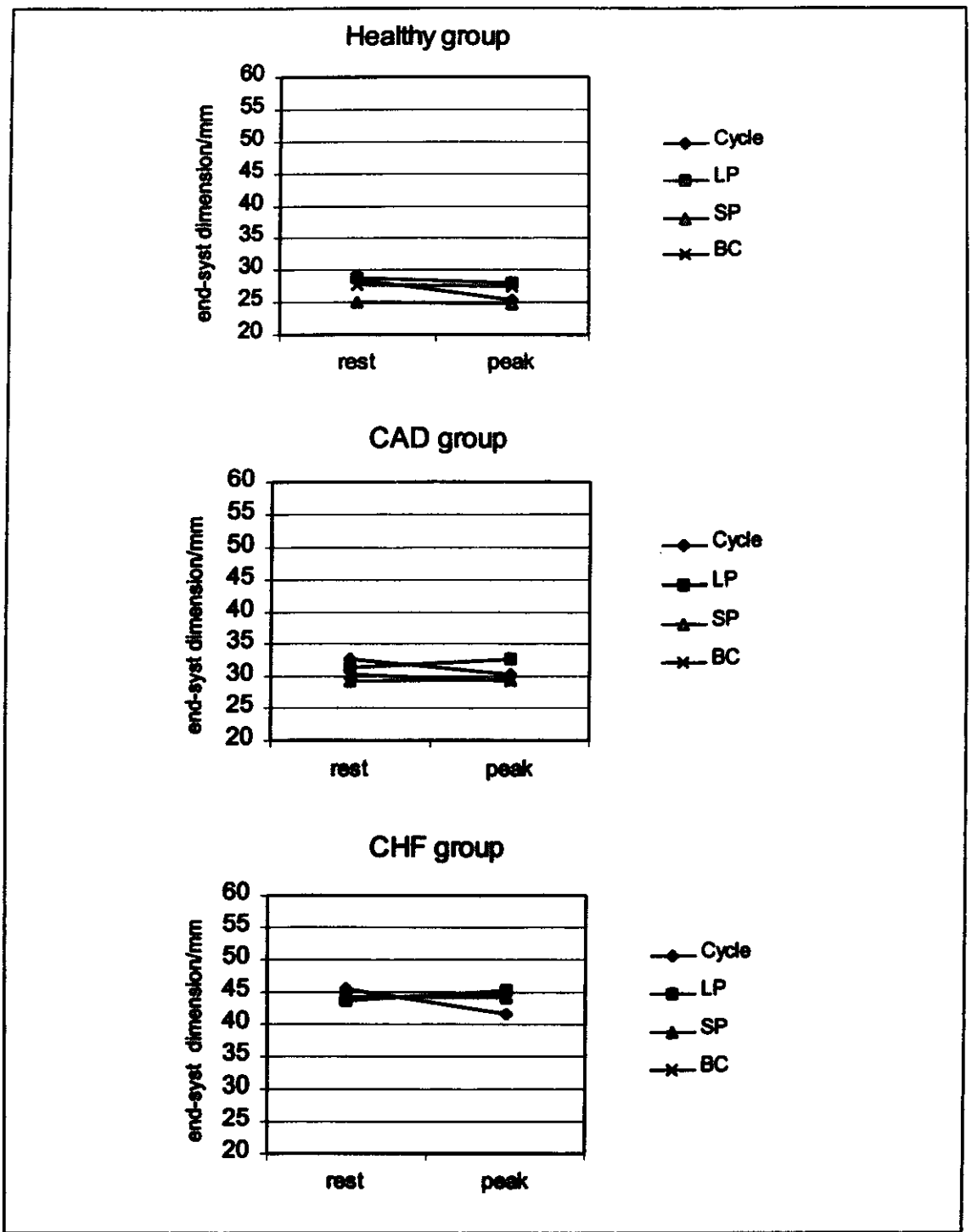

FIGURE 6. End-systolic dimension during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

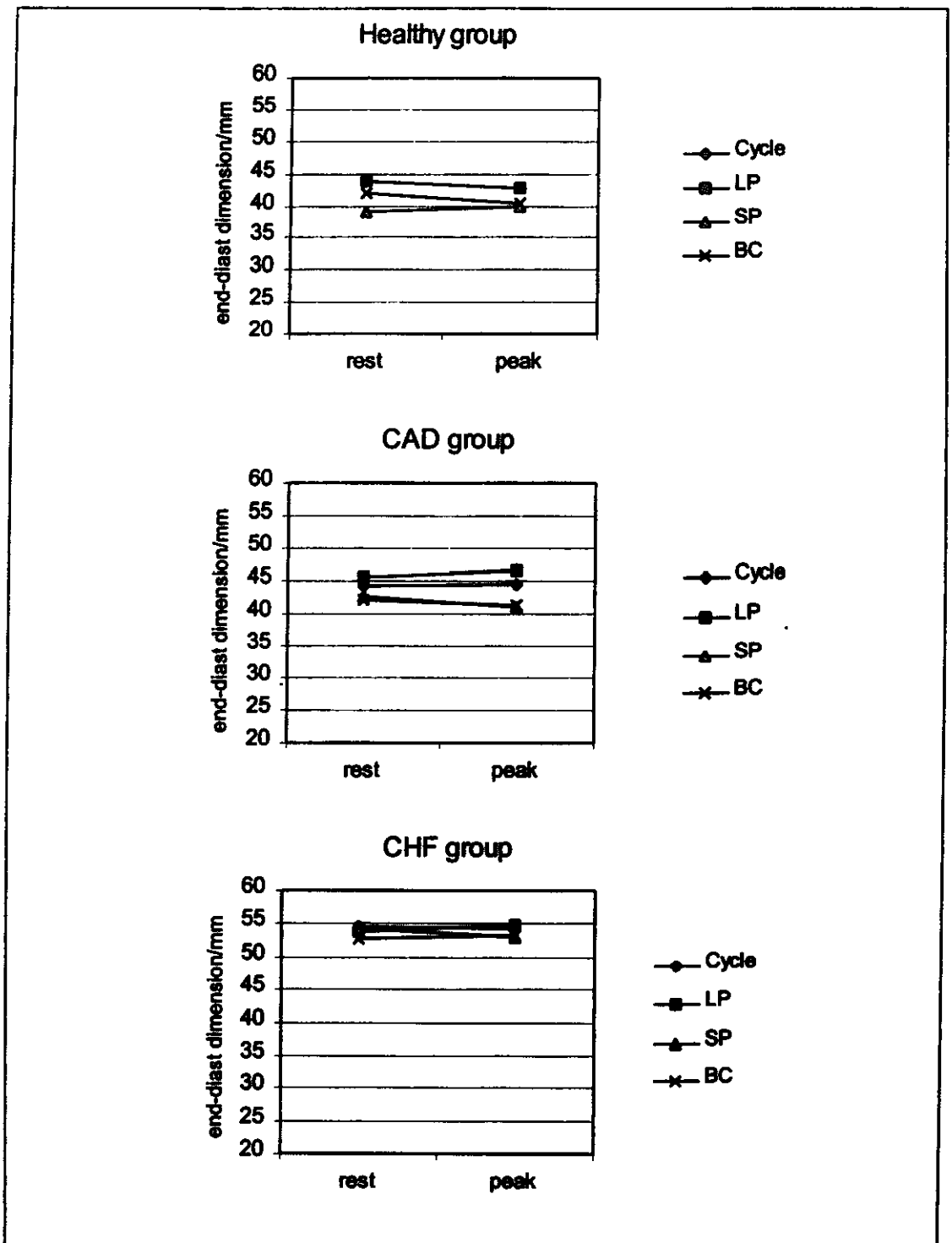

FIGURE 7. End-diastolic dimension during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

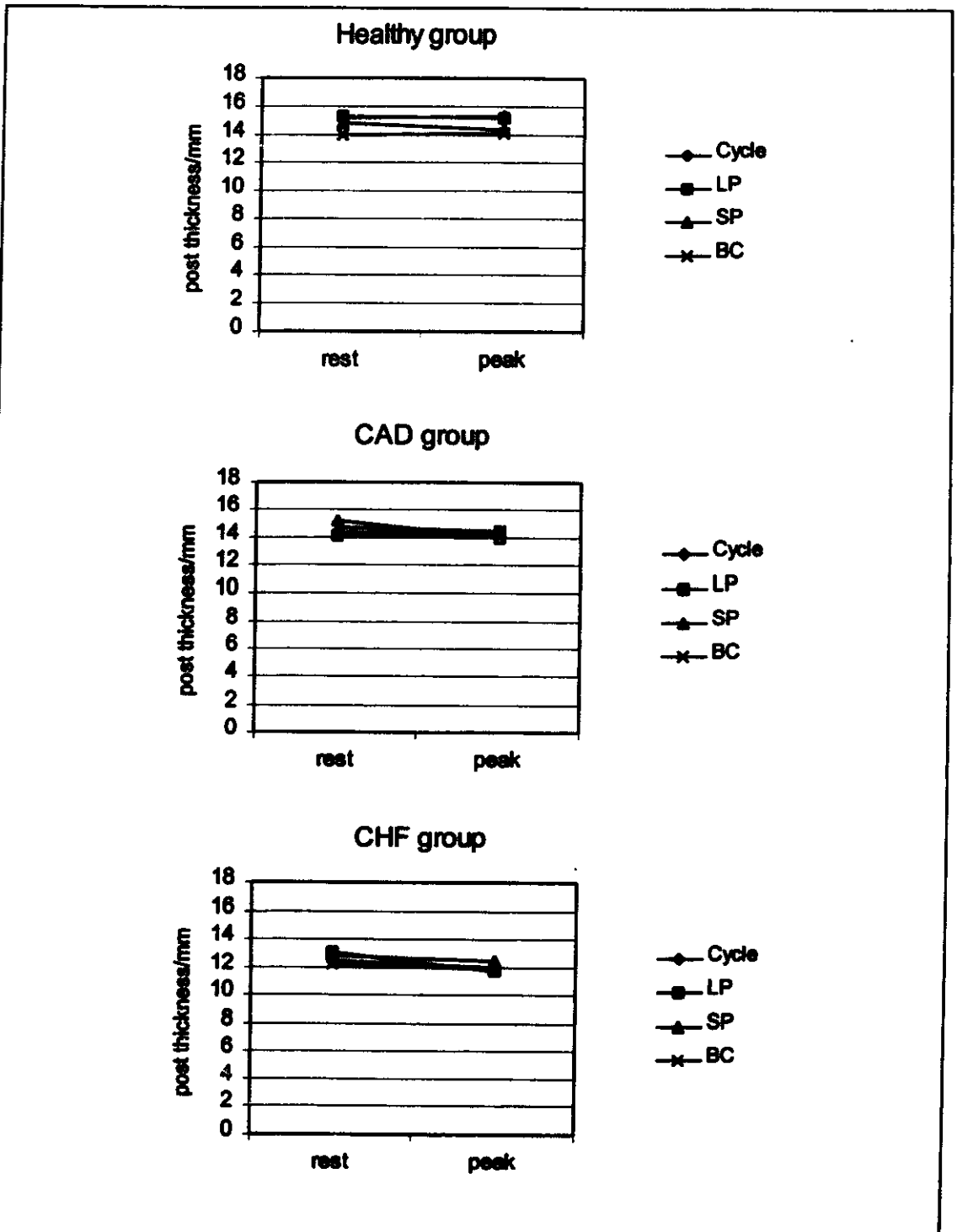

FIGURE 8. Systolic posterior wall thickness during cycle, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

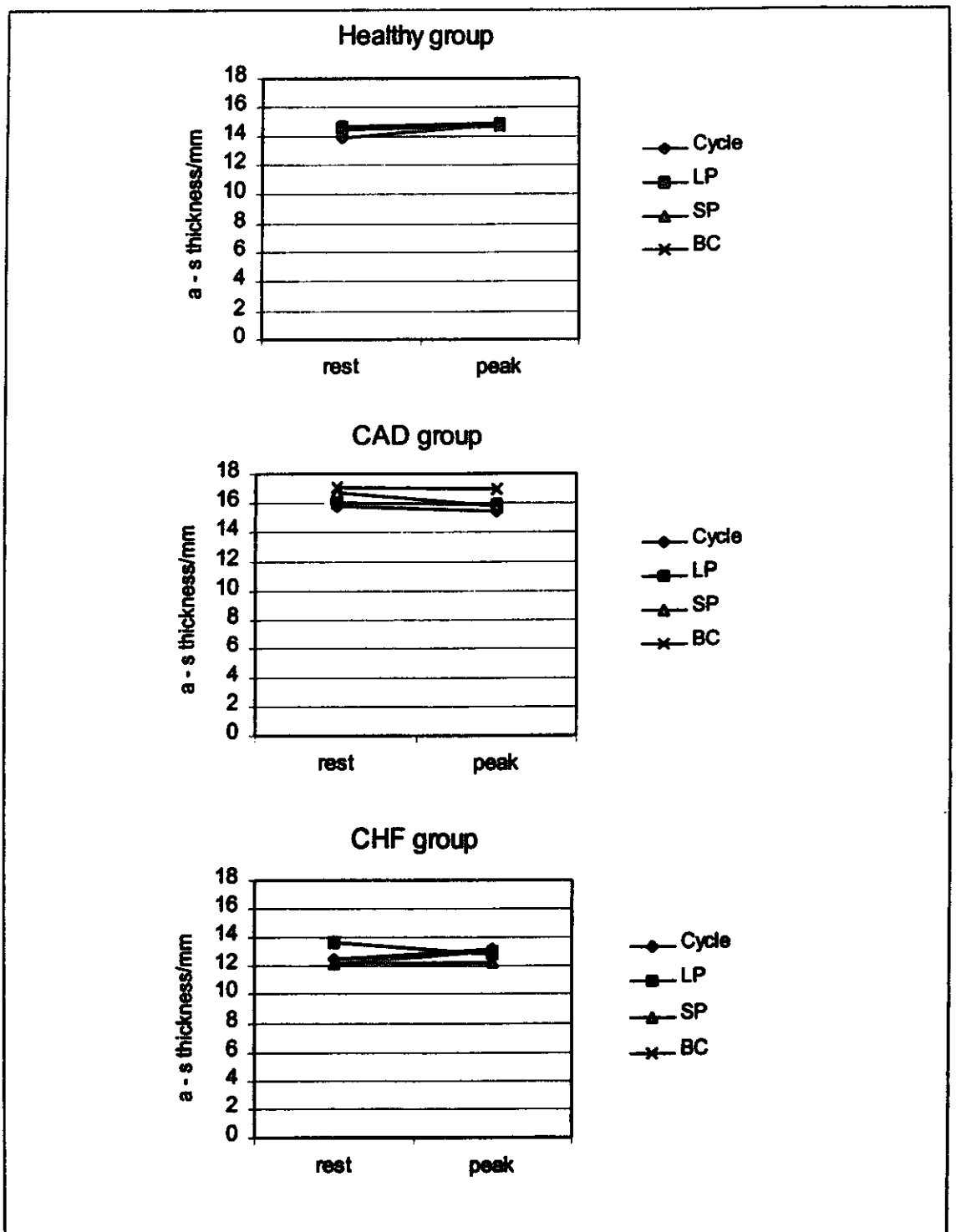

FIGURE 9. Systolic anterior-septal wall thickness during cycling, leg press (LP), shoulder press (SP) and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

tendency in the CHF group to show the smallest diastolic posterior wall thickness ($p = 0.075$) (Figure 10).

The CHF group showed significantly lower value than the other two groups in diastolic anterior-septal thickness when groups were compared. The difference between the CAD group and the healthy group was also significant (Figure 11). No other differences were found.

DISCUSSION

The primary purpose of this study was to evaluate LV function during both aerobic and resistance exercise in patients with CAD and in patients with CHF, and to compare this response to that of healthy controls. The major finding of this study was that LV function remained essentially stable during both aerobic and resistance exercise, suggesting the safety of resistance training in well-compensated patients with CAD or CHF. This suggests, in turn, that the already proven clinical efficacy of resistance training in the rehabilitation of patients with CAD may be extended to patients with CHF. Lastly, this study suggests that the traditional prohibition to overhead work by cardiac patients needs to be reconsidered.

The results of the cycle exercise are similar to those of prior studies. In healthy individuals, the LV ejection fraction is usually thought to increase somewhat during submaximal steady-state exercise.[28-31] Our results are consistent with these observations. In patients with CAD, without active ischemia, the LV ejection fraction is generally thought to increase slightly during the course of steady-state exercise.[14,30] Again, the present results support this expectation, even though we did not see a significant change

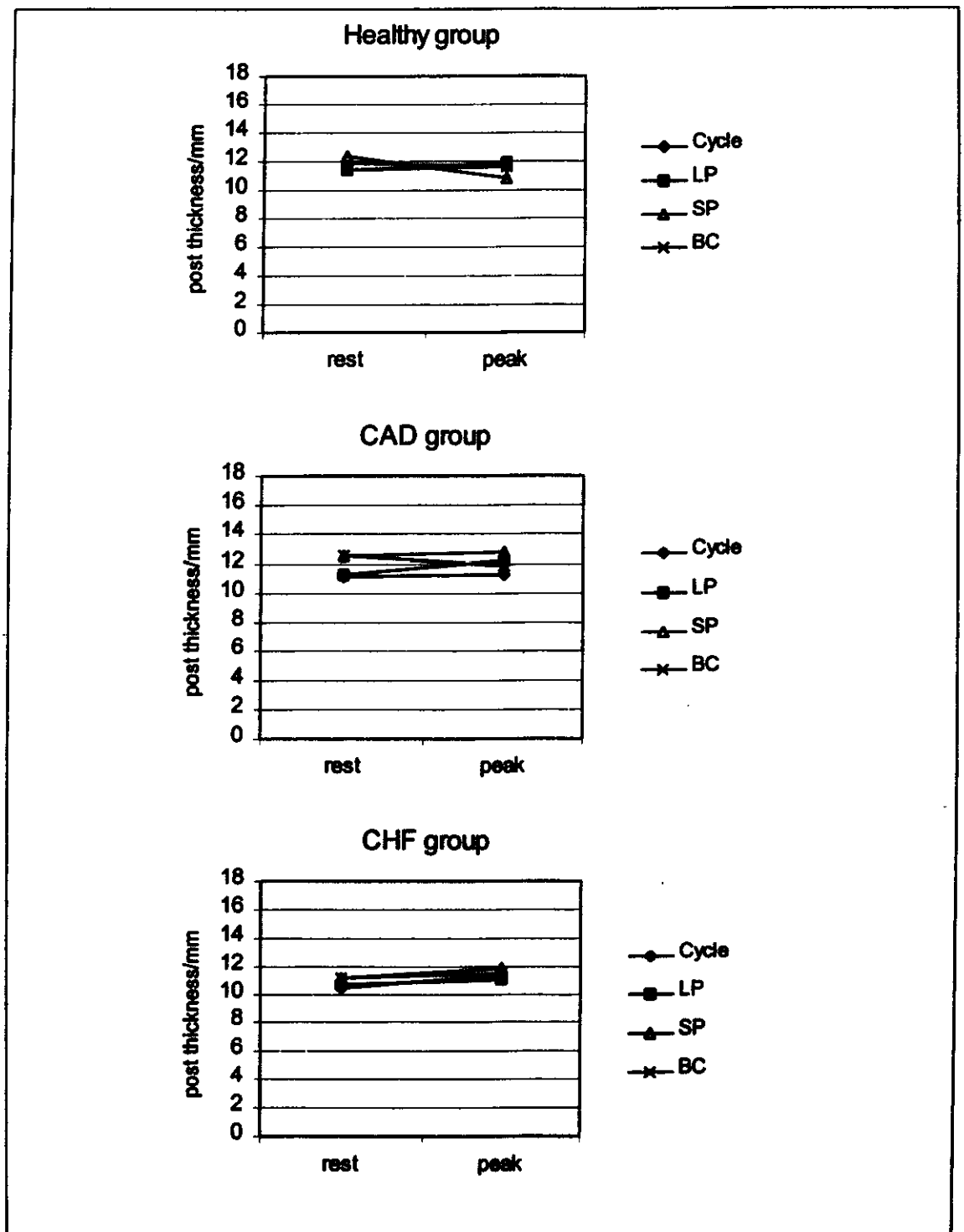

FIGURE 10. Diastolic posterior wall thickness during cycle, leg press (LP), shoulder press (SP), and biceps curl (BC) exercise in healthy group, in patients with coronary artery disease (CAD), and in patients with congestive heart failure (CHF).

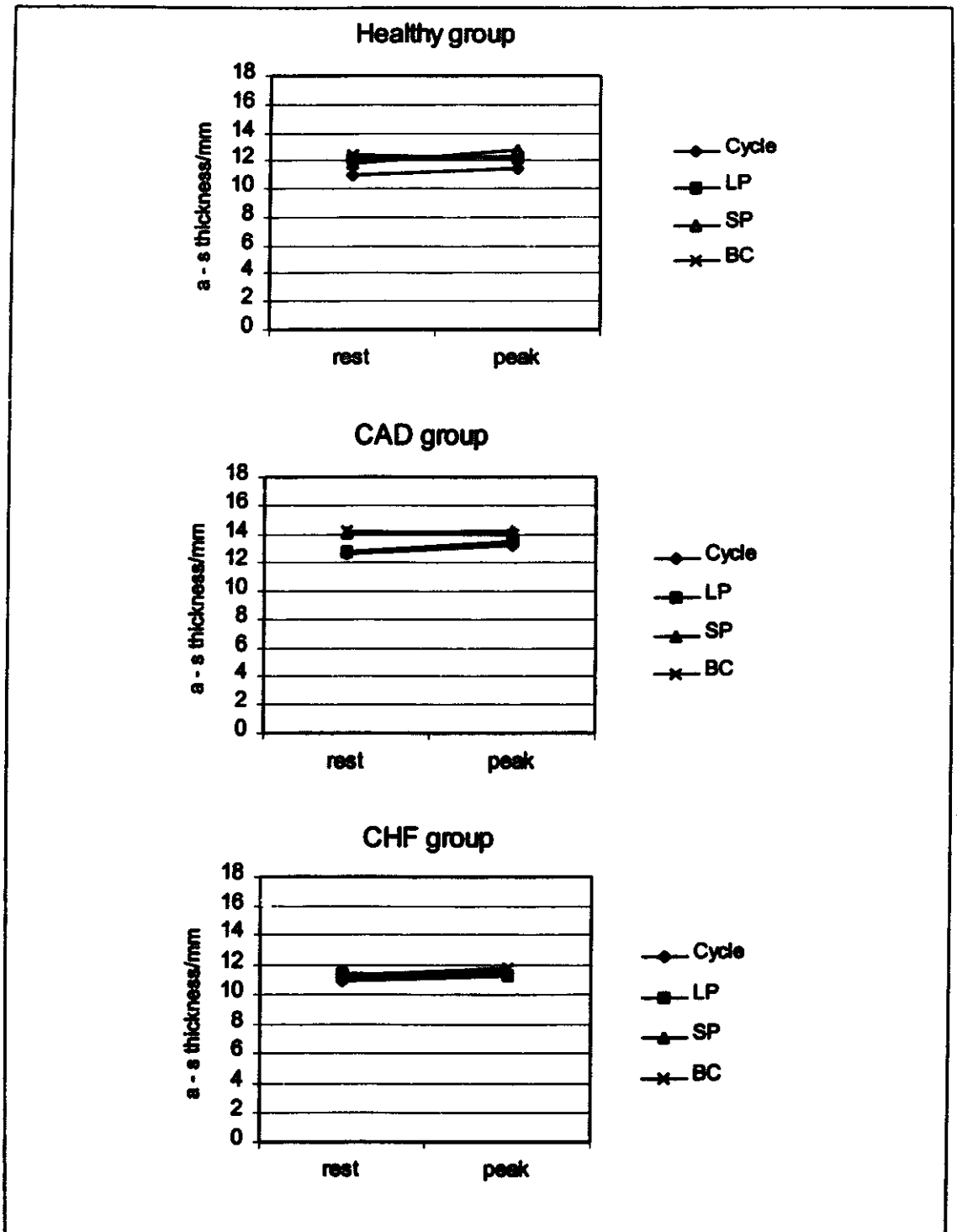

FIGURE 11. Diastolic anterior-septal wall thickness during cycle, leg press (LP), shoulder press (SP) and biceps curl exercise in healthy group, in patients with coronary artery disease (CAD) and in patients with congestive heart failure (CHF).

in LV ejection fraction. In patients with stable CHF, the LV ejection fraction is thought to increase somewhat or remain constant during steady-state exercise.[14,24,32] Our present results are consistent with these previous studies.

In healthy individuals, the end-diastolic volume is usually thought to increase somewhat at the beginning of steady-state exercise and then remain constant or decrease during the course of the exercise bout (due to continually falling systemic vascular resistance). At the same time, the end-systolic volume has a tendency to decrease throughout steady-state exercise (implying increased contractility).[28-31] In the present study, using dimensions as a surrogate for volumes, the end-systolic dimension showed a tendency to decrease in the control group, while the end-diastolic dimension remained constant. This implies similar responses as in previous studies. Foster et al.[30] showed very similar volume changes during steady-state exercise in healthy individuals and in patients diagnosed with CAD but exhibiting no active ischemia. However, Meyer et al.[14] showed no significant changes in volumes, which is consistent with the results of the present study. In patients with stable CHF, studies have shown both a proportional increase in end-diastolic volume and end-systolic volume[32] or no significant change in volumes during steady-state cycle exercise.[14,24] The results of our study imply constant volumes during steady-state cycle exercise. The results from these studies suggest that in well-compensated patients with CHF, aerobic exercise does not aggravate LV dysfunction. Dyspnea can be managed by brief rests or reductions in load with no further sequele.

In healthy individuals, our results during resistance exercise are similar to other studies in terms of large increases in systolic blood pressure, diastolic blood pressure, and mean arterial pressure.[22,32,33] The magnitude of the hemodynamic response was smaller than in previous studies and much less than in the classic work of MacDougall et al.[22] involving competitive body builders. Lentini et al.[33] measured LV function during the leg press exercise and found decreases in both end-diastolic volume and end-systolic volume, resulting in an increase in the LV ejection fraction. Those results are similar to the results of our study, which showed a tendency for the end-systolic dimension to decrease during resistance exercise. In patients with CAD but no active ischemia, Sagiv et al.[26] found increases in heart rate, systolic blood pressure and diastolic blood pressure similar to the present study, and no change in LV ejection fraction during isometric exercise. Featherstone et al.[34] showed systolic blood pressure and diastolic blood pressure responses during upper and lower body dynamic resistance exercise similar to the present study. In patients with stable CHF, resistance exercise studies have only been conducted using the leg press exercise. In these studies the systolic blood pressure, diastolic blood pressure, and mean arterial pressure responses during resistance exercise have been very similar to results of the present study.[24,25] Despite these hemodynamic increases, LV function remained stable during resistance exercise, consistent with our results.

This present study was limited by the echocardiographic imaging technique. No volumes were calcuable as only the diameters from the parasternal long axis view were measured. This was done purposely as we chose to optimize the exercise component at

the expense of better echocardiographic imaging (e.g. post exercise, supine, apical 4 chamber view), as our primary purpose was to measure function during exercise. Both hemodynamic and LV responses would have been compromised if a protocol more optimal for imaging had been chosen. Using LV dimensions as a surrogate of volume, our results suggest no major LV dilation and normal thickness of walls during exercise.

This study shows stability of LV function during aerobic and resistance exercise in patients with CAD or CHF and the fundamental similarity of the pattern of these responses to those of healthy individuals. This suggests that resistance exercise is as safe as aerobic steady-state exercise in these patient groups, e.g. CAD patients with essentially normal resting LV function, and NYHA class I-II CHF patients. Resistance exercise may therefore be included in a rehabilitation program in well compensated patients, increasing the quality and effectiveness of the rehabilitation. Further studies need to be done on patients with CHF of NYHA class II-III, and in patients with LV ejection fraction < 30.

REFERENCES

1. Meyer K, Lehmann M, Sunder G, Keul J, Weidermann H. Interval versus continuous exercise training after coronary bypass surgery: A comparison of training-induced acute reactions with respect to the effectiveness of the exercise methods. *Clin Cardiol* 1990;13:851-861.

2. Pollock ML, Ward A, Foster C. Prescription of exercise in a cardiac rehabilitation program. In: Fardy PS, Bennett JL, Reitz NL, Williams MA, eds. Cardiac Rehabilitation: Implications for the nurse and other health professionals. St. Louis: The Mosby Company, 1980:73-89.

3. Butler RM, Beierwaltes WH, Rogers FJ. The cardiovascular response to circuit weight training in patients with cardiac disease. *J Cardiopulm Rehabil* 1987;7:402-409.

4. Kelemen MH, Stewart KJ, Gillilan RE, Edwart CK, Valenti SA, Manley JD, Kelemen MD. Circuit weight training in cardiac patients. *J Am Coll Cardiol* 1986;7:38-42.

5. Maiorana AJ, Briffa TG, Goodman C, Hung J. A controlled trial of circuit weight training on aerobic capacity and myocardial oxygen demand in men after coronary artery bypass surgery. *J Cardiopulm Rehabil* 1997;17:239-247.

6. McCartney N, McKelvie RS, Haslam DRS, Jones NL. Usefulness of weightlifting training in improving strength and maximal power output in coronary artery disease. *Am J Cardiol* 1991;67:939-945.

7. Stewart KJ, McFarland LD, Weinhofer JJ, Cottrell E, Brown CS, Shapiro EP. Safety and efficacy of weight training soon after acute myocardial infarction. *J Cardiopulm Rehabil* 1998;18:37-44.

8. Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, Limacher M, Pina IL, Stein RA, Williams M, Bazzarre T. Resistance exercise in individuals with and without cardiovascular disease, benefits, rationale, safety and prescription. An advisory from the committee on exercise, rehabilitation and prevention, council on clinical cardiology, American Heart Association. *Circulation* 2000;101:828-833.

9. Belardinelli R, Demetrios G, Scocco V, Barstow TJ, Augusto P. Low intensity exercise training in patients with chronic heart failure. *J Am Coll Cardiol* 1995;26:975-982.

10. Hambrecht R, Fiehn E, Yu J, Niebauer J, Weigl C, Hilbrich L, Adams V, Riede U, Schuler G. Effects of endurance training on mitochondrial ultrastructure and fiber type distribution in skeletal muscle of patients with stable chronic heart failure. *J Am Coll Cardiol* 1997;29:1067-1073.

11. Belardinelli R, Georgiou D, Cianci G, Purcaro A. Randomized controlled trial of long-term moderate exercise training in chronic heart failure: effects on functional capacity, quality of life and clinical outcome. *Circulation* 1999;99:1173-1182.

12. Kavanagh T, Myers MG, Baigrie RS, Mertens DJ, Sawyer P, Shephard RJ. Quality of life and cardiorespiratory function in chronic heart failure: Effects of 12 months' aerobic training. *Heart* 1996;76:42-49.

13. Meyer K, Samek L, Schwaibold M, Westbrook S, Hajric R, Beneke R, Lehmann M, Roskamm H. Interval training in patients with severe chronic heart failure: analysis and recommendations for exercise procedures. *Med Sci Sports Exerc* 1997;29:306-312.

14. Meyer K, Foster C, Georgakopoulos N, Hajric R, Westbrook S, Ellestad A, Tilman K, Fitzgerald D, Young H, Weinstein H, Roskamm H. Comparison of left ventricular function during interval versus steady-state exercise training in patients with chronic congestive heart failure. *Am J Cardiol* 1998;82:1382-1387.

15. McKelvie RS, Teo KK, McCartney N, Humen D, Montague T, Yusuf S. Effects of exercise training in patients with congestive heart failure: A critical Review. *J Am Cardiol* 1995;25:789-796.

16. Drexler H, Riede U, Munzel T, Koning H, Funke E, Just H. Alterations of skeletal muscle in chronic heart failure. *Circulation* 1992;85:1751-1759.

17. Mancini, DM, Walter G, Reichek N, Lenkinski R, McCully KK, Mullen JL, Wilson JR. Contribution of skeletal muscle atrophy to exercise intolerance and altered muscle metabolism in heart failure. *Circulation* 1992;85:1751-1759.

18. Sullivan MJ, Green HJ, Cobb FR. Altered skeletal muscle metabolic response to exercise in chronic heart failure. *Circulation* 1991;84:1597-1607.

19. Delargadelle C, Feiereisen P, Krecke R, Essamri B, Beissel J. Objective effects of a 6 months' endurance and strength training program in outpatients with congestive heart failure. *Med Sci Sports Exerc* 1999;31:1102-1107.

20. Hare DL, Ryan TM, Selig SE, Pellizzer AM, Wrigley TV, Krum H. Resistance exercise training increases muscle strength, endurance and blood flow in patients with chronic heart failure. *Am J Cardiol* 1999;83:1674-1677.

21. Bowerman JA, Barnard KL, Adams KJ, Swank AM, Denny DM, Dushnick MR. Effect of resistance training (RT) on muscular strength in heart failure (HF) patients [abstract]. *Med Sci Sports Exerc* 1999;31:S368.

22. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol* 1985;58:785-790.

23. Freedson P, Chang B, Katch F, Kroll W, Rippe J, Alpert J, Byrnes W. Intra-arterial blood pressure during free weight and hydraulic resistive exercise [abstract]. *Med Sci Sports Exerc* 1984;16:131.

24. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall D. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol* 1995;76:977-979.

25. Meyer K, Hajric R, Westbrook S, Haag-Wildi S, Holtkamp R, Leyk D, Schnellbacher K. Hemodynamic responses during leg press exercise in patients with chronic congestive heart failure. *Am J Cardiol* 1999;83:1537-1543.

26. Sagiv M, Hanson P, Besozzi M, Nagle F, Zager L. Left ventricular responses to upright isometric handgrip and deadlift in men with coronary artery disease. *Am J Cardiol* 1985;55:1298-1302.

27. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, Gutgesell H, Reichek N, Sahn D, Schnittger I, Silverman NH, Rajik AJ. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommitte on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989;2:358-367.

28. Upton MT, Rerych SK, Roeback JR, Newman GE, Douglas JM, Wallace AG, Jones MD. Effect of brief and prolonged exercise on left ventricular function. *Am J Cardiol* 1980;45:1154-1160.

29. Foster C, Gal RA, Port SC, Schmidt DH. Left ventricular ejection fraction during incremental and steady-state exercise. *Med Sci Sports Exerc* 1995;27:1602-1606.

30. Foster C, Gal RA, Murphy P, Port SC, Schmidt DH. Left ventricular function during exercise testing and training. *Med Sci Sports Exerc* 1997;29:297-305.

31. Foster C, Meyer K, Georgakopoulos N, Ellestad AJ, Fitzgerald DJ, Tilman K, Weinstein H, Young H, Roskamm H. Left ventricular function during interval and steady-state exercise. *Med Sci Sports Exerc* 1999;31:1157-1162.

32. MacDougall JD, McKelvie RS, Moroz DE, Sale DB, McCartney N, Buick F. Factors affecting blood pressure during heavy weight lifting and static contractions. *J Appl Physiol* 1992;73:1590-1597.

33. Lentini AC, McKelvie RS, McCartney N, Tomlinson CW, MacDougall JD. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. *J Appl Physiol* 1993;75:2703-2710.

34. Featherstone JF, Holly RG, Amsterdam EA. Physiologic responses to weight lifting in coronary artery disease. *Am J Cardiol* 1993;71:287-292.

APPENDIX A

INFORMED CONSENT

INFORMED CONSENT FOR:

Left Ventricular Responses During Cycling and During Upper and Lower Body Resistance Exercise in Patients with Congestive Heart Failure.

I, _____ consent to participate in a study comparing heart rate, blood pressure and the pumping action of the heart during cycling and during resistance exercise in patients with congestive heart failure and controls.

I have been informed that the purpose of this study is to evaluate heart rate, blood pressure and the pumping action of the heart during resistance exercise and during steady-state exercise. This is being done to evaluate the safety of these types of exercises for patients with congestive heart failure. I consent to publication or presentation of results as long as I am not identified.

I have been informed that two separate visits to the Fransiscan Skemp Healthcare will be required of me. The purpose of the first visit is to determine the weights I will be lifting and the resistance on the cycle during the actual testing day. At the second visit, the actual testing will be done. Each visit will last around 60-90 minutes.

I have been informed that at my first visit, I will be required to undergo two different tests. One is a graded cycle exercise test where my oxygen consumption and exercise capacity will be measured to estimate the load I will be working at during cycling. I will be connected to a twelve lead electrocardiogram and my blood pressure will be measured at every stage. This is done to exclude any signs that my heart muscle is not getting enough blood during exercise or is showing any irregularities. During this test I will have a noseclip on my nose and I will breathe through a mouth-piece so that my oxygen consumption can be measured. The other test will be done to determine the weights I will be lifting during the resistance exercise on the testing day. For this, a weight that I can lift only once will be measured and then on the actual testing day I will be lifting 70% of this weight. The total time for these tests will be around 60-90 minutes.

I have been informed that on my second visit, the actual testing day, the session will consist of 18 minutes of cycling,(3 minutes warm-up and a 15 minutes of steady-state cycling). Steady-state cycling means that the load is the same for these 15 minutes. After the cycling I will get a 15 minute rest. Then I will do three different types of resistance exercises, biceps curls, shoulder press and leg press, 10 repetitions for each. For the arms I will be using dumbbells and for the legs I will be working on a machine. I will be doing these three different exercises while sitting. I will do the resistance exercises for the upper body with only one arm so that the echocardiographic measurements can be done. Two minutes rest will be given between the exercises. The total time for all these tests will be around 60 minutes.

I have been informed that during testing I will be connected to a twelve-lead electrocardiogram so my heart rate and rhythm can be examined. Echocardiography will also be done during the cycling and resistance exercises. Blood pressure will be measured both during cycling and during the resistance exercises. My blood pressure will be measured three times during the cycling and when I have 5 repetitions left of the lifting, the blood pressure will be measured while I am doing these last five repetitions. These blood pressure measurements will be done on the arm that I am not using. For the echocardiographic studies a transducer about the size of a cellular phone will be placed against the front of my chest. I should not experience any sensation from the high frequency sound waves used, but I may be aware of some mild pressure as the transducer will be held firmly against my chest wall.

I have been informed that both the tests prior to the study and the tests on the actual testing day include some risks. The graded exercise test risks include abnormal heart rate or blood pressure responses, irregular heart rhythms, heart attack, stroke and in very rare instances, death. The risk is estimated as 6 moderate and 1 serious complications per 10,000 tests. It should be noted that it would not be abnormal to feel some muscle soreness after the tests. Every effort will be made to minimize these risks.
Arna E Karladottir, an Advanced Cardiac Life Support certified graduate student in the AF/CR Masters Program at UW-L will be conducting all the testing. The electrocardiogram, blood pressure measurements and echocardiography add safety in detecting some abnormalities. Any unusual and uncomfortable signs or

symptoms I feel, I will have to report to the testers immediately. I have been informed that a physician will be immediately available during all testing.

I have been informed that my alternative is to not participate in this study.

I have been informed that if the investigators will be unsuccessful in echocardiographing my heart muscle my participation in the study will be terminated by the investigators.

I have been informed that although neither I nor my insurance carrier will be billed for these research procedures, either I or my insurance carrier will be responsible for the cost of treating complications that could occur as part of this study.

I have been informed that individual information obtained during these tests will be kept confidential. I will be informed about my results as will my personal physician, if I so desire.

I have been informed that my contact persons who will be available to answer any of my questions regarding this study are:
Arna E. Karlsdottir graduate student in the AF/CR program in UW-L, telephone: (608) 796-9178.
Dr.Carl Foster, professor in the College of HPER, UW-L and Arna's chairperson in this study, telephone : (608) 785-8687.
Dr. Richard J. Backes, the chair of Cardiology at Fransiscan Skemp Healthcare, telephone: (608) 785-0940.

I have been informed that questions regarding the protection of human subjects may be addressed to Dr. Garth Tymeson, chair of UWL-IRB for protection of human subjects, telephone: (608) 785-8155, to Terri Pedaci RN Research Coordinator at Fransiscan Skemp Healthcare, telephone: (608) 791-9462, and to Dr. Richard J Backes chair, Cardiology Department, Fransiscan Skemp Healthcare, telephone: (608) 785-0940.

I have read all the above and I have been informed what is expected of me and I consent to participate in the study.
All my questions have been answered to my satisfaction. I therefore voluntarily agree to be tested, and I have been informed that I am allowed to withdraw from these tests at any time without otherwise affecting any other treatment that might be available to me.

Signed:_____ Date:_____

Researcher:_____ Date:_____

APPENDIX B

REVIEW OF RELATED LITERATURE

REVIEW OF RELATED LITERATURE

The purpose of this paper is to review the literature related to left ventricular (LV) function during steady-state exercise and resistance exercise in apparently healthy people, patients with coronary artery disease (CAD) and patients with congestive heart failure (CHF).

Left Ventricular Responses During Exercise in Apparently Healthy People

During steady-state exercise, heart rate (HR) increases constantly as does the left ventricular ejection fraction (LVEF). The Frank-Starling mechanism seems to be utilized at first (increase in end-diastolic volume (EDV), stroke volume (SV) and LVEF), but as exercise is continued the EDV seems to decrease, suggesting that a continually falling systemic vascular resistance (SVR) allows maintenance of SV without use of the Frank-Starling mechanism.

During dynamic resistance exercise, the concentric work increases HR and both systolic blood pressure (SBP) and diastolic blood pressure (DBP). The blood pressure (BP) also increases with increasing repetitions. The rise is greatest at the beginning of exercise, where the muscle is in its weakest portion of the range of motion, requiring a high power production. A mechanical compression of the skeletal vasculature and elevated intrathoracic pressure appear to account for these hemodynamic increases. The HR and arterial pressure responses increase in proportion to the load. When comparing individuals with markedly different lifting capacities, the circulatory response seems to be more dependent on the relative rather than the absolute load. In the lockout phase,

when the muscle is in its strongest position, the hemodynamic values remain constant or return to almost resting values. When the eccentric work takes place, the hemodynamic response rises again but is significantly lower than during concentric work. The muscle is stronger during eccentric work, and the relative work is less. After exercise the BP falls rapidly due to the sudden release of muscle tension and hyperemic dilation of previously compressed vascular elements. The EDV decreases because of decreased venous return due to vascular dilation. The end-systolic volume (ESV) decreases because of decreased afterload and increased contractility. Thus the LVEF increases because of both enhanced contractility and a reduction in systemic arterial blood pressure. In healthy people these changes do not seem to cause deteriorating effects on LV function, even during rather strenuous exercise. In competitive body builders who work against very high resistance, the extreme rise in BP does not seem to have deteriorating effects on LV function. During both high and low resistance isometric work, hemodynamic responses are also large but not great enough to produce deterioration of LV function.

Left Ventricular Responses During Steady-State Exercise

Upton et al.[1] studied LV responses during prolonged exercise. The exercise lasted 120 min on an upright bicycle and the workload was 60-70% of maximum achieved during a preliminary stress test. The cardiac output increased primarily due to a gradual increase in HR, but there was no significant change in SV. The SV increased during the early minutes of exercise because of increases in both LVEF and EDV. The LVEF increased during the early phase because of an increase in EDV and myocardial

contractility observed by decrease in ESV. There was a slight decrease in mean blood pressure (MBP) over the course of exercise. They concluded that the normal response to prolonged exercise seems to rely on tachycardia, enhanced contractility and an early operation of Frank-Starling mechanism.

Foster et al.[2] studied LV function during steady-state exercise typical of that used in exercise training. The first 10 min of the protocol served as a warm-up with the workload increasing by 25 W every 2 min. The subjects then cycled for 20 min at a power output equivalent to their ventilatory threshold (VT). Subjects' HR increased significantly from rest to VT and showed a trend to continue to increase throughout the course of the steady-state exercise, which resulted in a significantly higher HR at 30 min than at 10 min. Blood pressure increased significantly during warm-up and had a tendency to decrease with continuation of steady-state exercise, resulting in a significantly lower MAP at 30 min than at 10 min. The LVEF increased significantly during warm-up and had a tendency to increase with continuation of steady-state exercise. A weak correlation was evident between the BP decrease and the increase in LVEF after the warm-up period, the authors suggested that a decrease in afterload may have importance in augmenting LV systolic function during steady-state exercise.

Palatini et al.[3] studied LV systolic performance in young subjects with mild hypertension (hypertensives) and compared it to subjects with normal BP (normotensives), during prolonged exercise at anaerobic threshold to exhaustion. Systolic BP increased gradually in both groups for the first 9 min and then progressively decreased throughout the remaining exercise period. The decrease was greater in

subjects with hypertension. The change in EDV was different between groups; the end-diastolic (ED) diameter declined in hypertensives while it was unchanged in normotensives. The end-systolic (ES) diameter decreased in both groups. The LVEF steadily increased up to the 20th min of effort and then leveled off in both groups. There was a statistical difference between the groups throughout exercise with LVEF being higher in the hypertensives. The SBP/ESV ratio leveled off much later in hypertensives than in normotensives. Changes in SV, cardiac output and total peripheral resistance (TPR) were similar in both groups. The authors concluded that the increased LV performance in hypertensives, both at rest and during prolonged exercise, was partially attributed to their increased LV wall thickness compared to normotensives. The authors hypothesized that the enhanced LV contractility in hypertensives relative to normotensives, was due to increased sympathetic activity in hypertensives. The reduced BP response in the hypertensive subjects appeared to reflect a limited ability to increase SV. The LV end-diastolic dimension tended to decrease slightly in the hypertensives, who emptied their ventricles more completely at end-systole to compensate for a lower end-diastolic volume. The authors concluded that the reduced early LV diastolic filling observed in the hypertensives at rest could be the reason for the tendency of the end-diastolic dimension to decrease during exercise. The TPR decreased similarly in both groups, leading to the conclusion that in the early stages of hypertension, the LV contractile reserve is increased to compensate for the initial decline in diastolic function. This response allows cardiac output to be maintained during vigorous exercise. As LV

hypertrophy gradually develops to reduce the wall stress, a progressive deterioration in systolic function may occur.

Foster et al.[4] studied LV responses during steady-state exercise in older participants. They used the same exercise protocol as in 1995[2] but made more extensive LV measurements. They found the same consistent drift in HR during steady-state exercise. The LVEF increased significantly. Although BP did not change significantly, it had a tendency to decrease. The cardiac output increased during warm-up and then remained unchanged, while SV increased significantly during warm-up. There was a tendency for SV to decrease during steady-state exercise. Calculated EDV did not change significantly during warm-up, thereafter it decreased significantly to below resting values. Calculated ESV decreased throughout steady-state exercise. Calculated SVR decreased during warm-up and then displayed a non-significant trend to decrease further. The authors concluded that an augmentation in EDV is important during incremental exercise, probably as a mechanism to augment SV and cardiac output. On the other hand, with continued exercise, EDV returns to or below resting values, suggesting that the continually falling SVR allows maintenance of SV without requiring Frank-Starling augmentation.

Foster et al.[5] studied LV responses in 8 healthy individuals, using a shorter duration of exercise (15 min) at a somewhat lower intensity (90% of individual anaerobic threshold). Their results supported their previous findings. Heart rate increased throughout the exercise bout. End-diastolic volume and SV first increased and then remained constant. The ESV had a tendency to decrease throughout exercise. The

cardiac output increased throughout the exercise bout, with a constantly decreasing SVR, and increasing LVEF.

Left Ventricular Responses During Resistance Exercise

MacDougall et al.[6] recorded BP response during heavy weight lifting exercise in 5 experienced body builders. The exercises involved different muscle groups. Direct arterial BP and intrathoracic pressure were constantly recorded during the lifts. The subjects performed single arm curls, single overhead presses and both double and single leg presses in the sitting position. Each exercise was performed to concentric contraction failure with 80, 90, 95 and 100 % of maximum single lift. The results showed that with the initiation of each lift, there was a large increase in both SBP and DBP (up to fourfold), which persisted for the 2-3 s required raising the weight. As the weight was lowered, both SBP and DBP declined rapidly towards pre-exercise levels. The BP increased progressively with each repetition of the lift but after exercise, both SBP and DBP fell below pre-exercise values and it required approximately 10 s to return to normal values. The HR increased over the duration of each exercise bout. The highest absolute pressures occurred during the double leg press (mean = 320/250 mmHg). The peak blood pressure responses, when a relatively small muscle mass was employed, was still extremely high although less than elicited by either a single- or double leg press. The authors concluded that this supports the concept that contraction of a larger muscle mass is supposed to exert compression on a greater proportion of the vasculature. The elevation in BP caused solely by mechanical compression of vessel walls would be proportional to the size of the active muscle mass, as well as the absolute force of the

contraction. The authors' opinion was that although the largest pressures occurred with the double leg press and lowest with the single arm curl, this could not be the major cause for these extreme elevations; a more direct relationship with increasing active muscle masses would have to occur. Their opinion was that progressive increases in active muscle mass throughout repetitive lifts, might in part account for the progressive elevation in SBP. As muscles are stronger eccentrically, the degree of muscle excitation during this phase of the lift would be less than during the concentric phase. This clearly took place in the study, implying that BP response is more dependent on the degree of effort or percent of maximal voluntary contraction (MVC) than on the absolute mechanical force developed. They further concluded that another explanation for this increase in BP might be due to an increase in both HR and SV, as well as by a vasoconstriction in non-exercising areas. The progressive increase in HR which was seen as subsequent repetitions were performed, probably also contributed to the progressive increase in BP found with each repetition. The authors concluded that the rapid drop in BP immediately after exercise was probably caused by the sudden perfusion of a large vasodilated muscle mass, which was previously occluded. It could also be caused by a transient pressure undershoot initiated by baroreceptor and cardiopulmonary reflexes responding to the extreme elevation in pressure with the last repetition.

Sagiv et al.[7] compared hemodynamic and LV functional responses in healthy non-athletic males and females during isometric exercise requiring activation of a large muscle mass. The subjects performed isometric deadlift, involving arm, shoulder, lower back, hip and leg muscle groups, for 3 min at 30% of MVC. The results showed that HR,

SBP and DBP increased during the exercise bout. The BP increased to a mean of 196/118 mmHg. The total systemic vascular resistance increased only slightly from rest to maximal exercise in both groups. Only the males increased their LVEF. No change in end-diastolic dimension occurred in either group. The end-systolic dimension was significantly smaller during the deadlift than at rest in both groups. The contractility index in the two groups was similar at rest and increased significantly and similarly during the deadlift. The SV did not change, so a marked increase in HR was needed to maintain adequate cardiac output. The authors concluded that the pressure increment during static contraction elevates afterload by increasing both SBP and DBP. The increase in afterload may affect cardiac output, LVEF and heart volumes during isometric exercise.

MacDougall et al.[8] studied factors affecting BP during heavy weight lifting and static contractions. The subjects were 31 healthy male volunteers. All subjects were physically active and had had previous exposure to heavy resistance training. The factors studied were BP responses, intrathoracic pressure, effects of joint angles, effects of muscle size and strength, effects of contraction type, effects of the Valsalva maneuver, and the effects of muscle fatigue. The main result was that the peak BP was independent of muscle size. Although absolute force production varied between different muscle work, when contractions were made at the same relative intensity, BP and intrathoracic pressures were the same. Subjects did not seem to use the Valsalva maneuver except when the required force output exceeded 80% of MVC or the repetitions of lighter loads were continued to failure. The BP tended to increase when effects of muscle fatigue were

studied, although the force production declined over time. The authors' conclusion was that the magnitude of the BP response is related to the intensity of the effort and is independent of absolute force production or interindividual differences in muscle size. During different types of exercise the magnitude of the pressor response is primarily determined by the degree of central command or effort rather than feedback from the contracting muscles. The pressor response depends on the degree of effort involved in the lift and not to the absolute force of the contraction, as muscles are not evenly strong throughout the lifting range. The main finding was that increased BP is maintained and even increases over time although absolute force decreases which illustrates that it is the central drive to the muscle that determines the magnitude of the pressor response. The authors concluded that the tendency for BP to increase over the latter part of the contraction is probably the result of increased cardiac output due to increased HR and the probable recruitment of additional musculature in an attempt to maintain contraction force.

Sullivan et al.[9] studied 10 healthy young men when they performed serial isometric deadlifts at 50% of MVC for 90 s and at 100% of MVC for 30 s. Echocardiographic imaging, arterial BP and ECG monitoring were recorded throughout the deadlift and for 30 s of recovery. When the deadlift was performed at 50% of MVC, the BP and HR increased significantly but within 2-5 s after cessation of the deadlift, an abrupt fall in MBP was seen such that after 30 s of recovery it was close to resting values. The SV and LVEF decreased initially during the deadlift but after 15 s of exercise it went up again and approximated resting values before the termination of the deadlift. When

the deadlift had been performed for 15 s, the cardiac output started to increase and increased throughout the deadlift exercise. Calculated peripheral resistance increased slightly during the initial 15 s of the deadlift but returned to control values at the end of the deadlift. With cessation of the deadlift, peripheral resistance fell rapidly and remained significantly reduced at 30 s after the cessation of the lift. The initial decline was associated with a significant increase in ESV. A small nonsignificant increase in EDV at the end of the deadlift could have accounted for the restoration of LVEF to resting values by the end of the deadlift. The LVEF increased significantly within 5 s of recovery and the increased value was maintained at 30 s of recovery. The ESV and EDV declined rapidly after cessation and then partially recovered by 30 s. The calculated SBP/ESV ratio was unchanged at 15 s and 30 s of the deadlift but increased significantly at 5 s and 30 s of recovery. The patterns for all measured parameters during 100% of maximal deadlift were similar to the 50% of maximal deadlift. The authors' conclusion was that the initial decline in SV and LVEF was due to an increase in ESV secondary to the abrupt rise in systemic arterial pressure and corresponding LV wall stress. This initial decrease in LVEF was restored by augmentation of EDV, which indicates reliance on the Frank-Starling mechanism. As the SBP/ESV ratio did not increase during the lifts, the authors found it impossible to derive any definite conclusions about changes in contractility. The rapid fall in BP was attributed to the sudden release of muscle tension followed by hyperemic dilation of previously compressed vascular elements within the large mass of skeletal muscles used in maintaining the effort needed. The rapid LVEF increase after the lifts is a response to the reduction in systemic arterial blood pressure.

Both EDV and ESV decreased the EDV probably because of impaired venous return due to acute dilation of vascular capacitance vessels within skeletal muscle, and the standing posture. The decrease in ESV was attributed to the combined effects of afterload reduction and enhanced contractility. The calculated SBP/ESV ratio remained increased throughout the recovery in both types of lifts, which the authors suggested to be a sign of increased inotropic status. The cardiac output and MAP seemed to be protected by sustained increases in HR and enhanced LV performance during the recovery phase after muscular relaxation.

Lentini et al.[10] studied LV responses throughout a lifting phase in 5 healthy young males. These responses were compared to resting values. Subjects worked at 95% of 1 repetition maximum (1 RM) during double leg press. The SPB and DBP increased significantly during the lift, decreased in the lockout phase and increased significantly again during the lowering phase. The MAP increased significantly during the lift but went to resting levels during the lockout and lowering phases. There was an increase in TPR during lifting, during the lockout phase the resistance was significantly lower than the pre-exercise values and then reached pre-exercise values in the lowering phase of the lift. The HR increased during the lifting phase and remained elevated in the other two phases of the lift. The EDV and ESV decreased significantly during the lifting phase, increased significantly during the lockout phase and decreased again significantly during the lowering phase; the ESV reached lower values than pre-exercise. The SV decreased non-significantly during the lifting phase, increased during the lockout phase and decreased during the lowering phase, to lower values than pre-exercise. The cardiac

output increased throughout the first two phases of the lift and then decreased during the lowering phase, compared to the lockout phase. The SBP/ESV ratio increased significantly during the lifting phase declined during the lockout phase and increased again during the lowering phase to greater values than pre-exercise. The LVEF increased significantly during the lifting phase and remained elevated throughout the two other phases of the lift, significantly higher than the resting valuc. During the lockout phase there was a significant decline in BP and HR and an increase in cardiac volumes to control values. During the lowering phase the cardiac volumes tended to decrease whereas both HR and BP increased. During concentric and eccentric phases SV tended to decrease. The main results were that rapid changes in cardiac volumes and contractility occur related to the different phases of the lift. Greater effort was needed during the lifting phase than the lowering phase and a greater effort was required during both lifting- and lowering phases compared to the lockout phase. The authors concluded that this matched with the well-known fact that the muscle can generate greater force during maximum eccentric contraction than during maximum concentric contraction. The BP changes and the changes in the SBP/ESV ratio were in concert with the level of effort. The authors concluded that the changes in cardiac volumes might have been due to changes in central motor command but the role of alterations in venous impedance and the influence of the skeletal muscle pump function could not be assessed in this study.

Left Ventricular Responses During Exercise in Patients with Coronary Artery Disease

During steady-state exercise, ST segment changes tend to modulate in patients with documented CAD. No evidence of progressive LV dysfunction is shown when

steady-state exercise is continued, even though incremental warm-up shows dysfunction. Patients with prior myocardial damage without evidence of ischemia show responses similar to healthy people.

During isometric resistance exercise, no significant correlation between the resting LVEF or magnitude or direction of change in LVEF has been shown. A comparison of dynamic resistance exercise and treadmill exercise in trained subjects has shown more ischemic ECG findings during treadmill exercise. Myocardial oxygen demand seems to be similar during treadmill exercise and dynamic resistance exercise but the DBP is greater during lifting, suggesting that increased myocardial perfusion pressure contributes to a reduced ischemic response. The oxygen supply/demand ratio seems to be better during lifting than during treadmill exercise, implying no further risk in performing resistance exercise compared to treadmill exercise.

Left Ventricular Responses During Steady-State Exercise

Foster et al.[4] studied LV responses during steady-state exercise in patients with documented CAD. The patients were divided into two groups. One group demonstrated significant ST-segment depression on a screening incremental exercise test (+ ischemia group). The other group did not demonstrate ST-segment depression on a screening incremental exercise test (- ischemia group). Eight of 10 subjects in the + ischemia group had angina during steady-state exercise. Exercise was performed at a power output predicted to yield the VO_2 associated with VT. The power output was incrementally increased so that the power output associated with VT was first achieved between 8-10 min of exercise. Thereafter, the power output remained constant. The subjects were

allowed to decrease the power output momentarily as dictated by anginal pain and/or fatigue. During exercise, ST-segment depression observed during the first 8-10 min tended to modulate. There was a consistent drift in HR during the 30 min of exercise but the BP did not change significantly. The LVEF in the – ischemia group increased to the 10th min of exercise and thereafter it remained constant throughout the 30 min of steady-state exercise. In the + ischemia group, no significant change in LVEF from rest throughout the 30 min of steady-state exercise occurred. Wall motion improved in the – ischemia group from rest to the 10th min and then remained unchanged throughout the 30 min of steady-state exercise. In the + ischemia group, wall motion deteriorated significantly from rest to the 10th min of exercise and then it remained stable throughout the 30 min of steady-state exercise. Calculated cardiac output increased significantly from rest to the 10th min and then it remained constant in both groups. Calculated SV increased significantly from rest to the 10th min of steady-state exercise in the – ischemia group. Beyond 20 min of exercise there was a tendency for SV to decrease in the – ishemia group. The calculated SV remained constant in the + ischemia group. Calculated EDV did not change from rest to the 10th min in the – ischemia group but thereafter it decreased significantly to below resting values. No significant change in EDV was observed in the + ischemia group. The calculated ESV decreased throughout steady-state exercise in the – ischemia group but it did not change in the + ischemia group. Calculated SVR decreased in both groups from rest to the 10th min of exercise. Thereafter there was a non-significant trend for it to decrease somewhat more with continued exercise. The authors concluded that there was no evidence of progressive LV

dysfunction when steady-state exercise is continued although the incremental warm-up shows dysfunction. Patients that have prior myocardial damage but no evidence of ischemia showed no evidence of deterioration of myocardial function. The authors concluded that with continued exercise EDV returns to or below resting values, suggesting that the continually falling SVR allows maintenance of SV without using Frank-Starling augmentation, which could be more expensive in terms of increased wall stress and myocardial oxygen uptake. Their final conclusion was that the LV function seems to become nearly normal during sustained steady-state exercise. It seems that exercise under ischemic conditions shows essentially stable pattern of LVEF during 30 min of rather strenuous steady-state exercise without progressive deterioration of LVEF.

Left Ventricular Responses During Resistance Exercise

Sagiv et al.[11] studied 15 males with documented CAD or myocardial infarction (MI), who were already in a supervised exercise program. All of them had a functional capacity at or above 9 METS. The left ventricular function was evaluated during handgrip exercise and during deadlift exercise. Both exercises were performed at 30% of maximum effort for a total of 3 min. The LV responses were compared to these responses in apparently healthy individuals. The patients with CAD showed significant increases in HR while the healthy individuals did not. The BP increased significantly in both groups during the isometric handgrip exercise and also during the deadlift exercise. The LVEF, which was significantly lower in the CAD patients, remained constant in both groups during both types of exercise. There was no significant correlation between the LVEF at rest and the magnitude or direction of its change during handgrip- or deadlift

exercise. During handgrip exercise the ESV and the EDV showed a parallel non-significant increase in both the apparently healthy individuals and in the CAD group. At rest, wall motion abnormalities were detected in 9 out of 14 CAD patients. During the handgrip exercise, wall motion abnormalities developed in 2 patients that had normal wall motion at rest. During the deadlift exercise, LV wall motion abnormalities occurred in 13 out of 14 CAD patients. The apparently healthy individuals showed no evidence of wall motion abnormalities. The main result was thus that LV function remains stable during submaximal isometric exercise. The authors attributed the development of new LV wall motion abnormalities to ischemia that was induced by increased rate pressure product (RPP) or possibly by increasing dyskinesia in previously abnormal regions.

Featherstone et al.[12] studied hemondynamic responses in 12 male volunteers with documented CAD during weight lifting at 40, 60, 80 and 100% of MVC. These responses were compared to hemodynamic responses during a maximal treadmill exercise test. All participants had been participating in a cardiac rehabilitation program for at least the preceding 4 months. Free weights were used and each participant performed supine press, seated overhead press, seated biceps curl and seated double leg quadriceps extension. The BP was always measured during the last repetition of each lift and completed before the end of the lift. No patient developed chest pain or significant ventricular ectopy during either lifting- or treadmill exercise. No signs of ischemia appeared on the ECG while lifting, whereas 5 out of 12 patients developed ischemic changes during maximal treadmill exercise. Peak HR during the resistance exercises was less than during the treadmill exercise, whereas the peak SBP was similar for all exercise

types. The RPP was also less during all lifts than during treadmill exercise, suggesting less myocardial oxygen demand. The DBP was greater during all lifts than during the treadmill exercise except during the 100% MVC biceps curl and quadriceps extension exercises. When estimates of myocardial oxygen supply were compared with those of myocardial oxygen demand, all lifts yielded significantly higher supply/demand ratios than did treadmill exercise. The RPP of the various lifts tended to be less at 100% MVC than at other intensities which the authors attributed to a limited time for HR and SBP to respond during one repetition. The main conclusion was therefore that resistance training appears to be safe for this population, which were trained individuals with documented CAD.

Left Ventricular Responses During Exercise in Patients with Congestive Heart Failure

During steady-state exercise patients with CHF show similar cardiovascular responses as healthy people but their metabolic levels are higher than in healthy people. The LVEF during isometric exercise has been controversial with studies showing decreases and increases in LVEF during exercise. Dynamic resistance exercise seems to be well tolerated by patients with CHF. They seem to be able to use the Frank-Starling mechanism and increases in the SBP/ESV ratio and stroke work during dynamic resistance exercise indicate increased contractile function during this type of exercise. Patients with CHF have not shown evidence of LV deterioration during dynamic resistance exercise.

Left Ventricular Responses During Steady-State Exercise

Delahaye et al.[13] assessed and compared LV responses during 3 different modes of testing. They compared maximal upright bicycle exercise test, the 6-minute walk test and stair climbing exercise test. Thirteen patients with stable CHF and LVEF < 35% were tested. All participants had normal coronary arteries. During the 6-minute walk test, patients walked as quickly as possible for 6 min without running. The BP was recorded at the beginning and at the end of the test while other measurements were done during exercise. The HR, SBP, ESV and EDV all increased significantly during the 6-minute walk test. The ESV and EDV increased proportionately resulting in no significant changes in LVEF. The authors concluded that despite hemodynamic challenges and increases in cardiac volumes during the 6-minute walk test, there were no signs of LV deterioration.

Meyer et al.[14] studied LV responses during steady-state exercise in 11 males with clinically stable CHF. The resting LVEF was 37 ± 3 %. For a control group, 8 males with documented CAD but only minimal LV dysfunction participated. The LVEF of the control group at rest was 58 ± 5%. Every participant underwent maximum incremental cycle ergometry test (10 – 15 W/min increments) to exclude myocardial ischemia and to measure exercise tolerance. Thirty minutes later a steep ramp exercise test (25 W/10 s increments) was performed to determine maximum short time exercise capacity. This study compared LV responses during interval exercise and during the steady-state exercise. The workload for the steady-state exercise was derived from the mean power output during the interval exercise part. The workload during the work phases in the

interval exercise part was set at 50% of the maximum workload capacity attained during the steep ramp exercise test. The work rate level for the steady-state exercise was the mean power output performed during the interval exercise part. During the steady-state exercise there were significant increases in LVEF, HR, SBP, cardiac output, lactate levels, ratings of leg fatigue and dyspnea in the patients with CHF. In the control group, the LVEF and SBP were the only variables that increased significantly. The magnitude of LVEF increase did not differ between the groups. No significant changes in EDV or ESV were seen in the two groups. The authors concluded that the pattern of the LVEF response during exercise in patients with CHF was similar to those responses reported during steady-state exercise in healthy people and in patients with stable CAD.

Left Ventricular Responses During Resistance Exercise

Elkayam et al.[15] evaluated hemodynamic effects of isometric exercise in 53 patients with stable NYHA class III and IV CHF. The resting LVEF ranged from 8 - 41% in the patient group. The hemodynamic effects were compared to the hemodynamic effects in 10 healthy subjects during the same exercise. Subjects were studied during isometric handgrip exercise at 30% of their maximal compression pressures. The duration of the handgrip ranged from 5-7 min. In the patients with CHF the HR, MAP, and SVR increased significantly during exercise. The cardiac output and SV decreased, but the stroke work did not change even though the group showed varied responses. Those patients who experienced the greatest decrease in stroke work had both lower cardiac output and SV than those who did not. The healthy subjects differed in that they increased their cardiac output and SV slightly but not significantly and their SVR

remained constant. The increased SV and cardiac output were therefore responsible for the increase in BP. The increase in BP in the patients with CHF, on the other hand, was primarily due to increased SVR. This increase in SVR was responsible for decreased SV and stroke work, resulting in either no change or an increase in LV filling pressure. The authors found a significant relationship between SVR changes and SV and cardiac output changes, and concluded that increases in afterload can result in deteriorating effects of LV function in patients with CHF. The hemodynamic deterioration could not be attributed to the patients' resting LVEF.

Reddy et al.[16] studied hemodynamic, ventilatory and metabolic effects of light isometric exercise in 20 patients with clinically stable CHF. The mean LVEF was 24 ± 9%. This was done in an attempt to explain why light exercise in these patients often results in dyspnea. These responses were compared to those responses in 17 normal individuals who served as a control group. The handgrip exercise load was 25% of MVC and was performed for 6 min. The HR increased significantly in both groups. The cardiac output increased by 58% in the control group but failed to increase in the CHF group. The SV also decreased in the CHF group. Oxygen extraction in the exercising forearm in the patients with CHF was higher than in the normal subjects but the difference was not significant. The patients with CHF increased their lactate concentration significantly whereas the control group did not. The patients with CHF increased their CO_2 production during the recovery and therefore increased their ventilation in an attempt to increase their VO_2. The authors concluded that this was

probably the reason why patients perceive themselves to be breathless during or after daily activities.

McKelvie et al.[17] studied acute cardiovascular responses during resistance exercise in 10 males with CHF and compared them to the same responses during cycle ergometry. The mean LVEF was 27 ± 2% (NYHA class I - III). The patients performed 5 min of cycle ergometry at 70% of peak capacity and performed 2 sets of 10 repetitions of single leg presses at 70% of 1 RM. The authors observed no adverse symptoms, arrhythmias or ST segment abnormalities during either type of exercise. The significant increase in SBP was not significantly different between the different exercise modes and the DBP was significantly higher during the resistance exercise. The HR increased significantly during both exercise modes and was significantly higher during cycling thus the rise in RPP was greater during cycling. There were no significant changes in EDV or ESV during either exercise mode. The cardiac output increased significantly in both modes and was significantly higher during the cycle exercise. The SV increased significantly during the cycle exercise and the TPR decreased significantly. The LVEF did not change significantly in either exercise modes. The SBP/ESV ratio increased significantly during both modes but was significantly higher during cycle exercise. The authors opinion was that while these two different exercise types produced similar increases in afterload, the resistance exercise showed lower RPP and thus lower myocardial oxygen demand. The increase in was HR less during the resistance exercise but the DBP increased more, suggesting better myocardial perfusion during resistance exercise than during cycling. The rise in cardiac output during the resistance exercise

occurred mainly because of increased HR . The main result was that single leg press does not produce adverse effects of LV function in patients with CHF.

Meyer et al.[18] assessed hemodynamic responses during double leg press exercise at 2 different loads in patients with stable CHF and LVEF 26 ± 3%. The responses were compared to the hemodynamic responses in 6 male patients with mild LV dysfunction and LVEF 68 ± 2%. The participants performed double leg presses at 60% and 80% of MVC. Each participant performed 2 series of four sets each. One set lasted 60 s and it included 12 repetitions. This was compared to 5 min of cycling at 25 W. This was done in order to compare the beginning of resistance exercise to the beginning of isotonic exercise. During the resistance exercise at 60% of MVC, there was a significant increase in HR, BP, pulmonary artery pressure (PAP) and cardiac index. The mixed venous oxygen saturation also decreased significantly. In addition to the same changes during 80% load as during 60% load, there was a significant increase in SV, stroke work and MAP and a significant decrease in SVR. The magnitude of change between different loads showed a significant difference in SVR (decreased more during 80% of MVC), SV (increased more during 80% of MVC), stroke work (increased more during 80% of MVC), cardiac index (increased more during 80% of MVC) and mixed venous O_2 saturation (decreased more during 80% of MVC). During the 60% load, the CHF group showed significantly higher HR but significantly lower SBP and MABP than the control group. The magnitude of change was similar in the two groups. The SV, cardiac index and stroke work were significantly lower and a smaller magnitude of change occurred in the CHF group. The SVR changed to a lesser extent in the CHF group. Both the CHF

group and the control group had similar reponses at the beginning of steady-state cycling exercise. The authors concluded that the increase in cardiac index was primarily due to the increase HR and the increase in BP was due to increased cardiac index because no significant change in SVR occurred. When compared to controls, the patients with CHF had significantly lower HR and MABP and a smaller change in BP during exercise, which the authors explained by lower SV, cardiac index, stroke work and a smaller decrease in SVR. No deterioration of LV function was seen. When the authors compared 60% and 80% loads, they concluded that a significantly greater increase in cardiac index in response to a greater load was probably due to a significantly more decrease in SVR during 80% of MVC. The 80% load showed greater cardiovascular and metabolic strain shown by significantly lower venous O_2 saturation. The authors concluded that the significant increases in SV and stroke work during 80% suggested enchanced contractile function of the left ventricle because preload and afterload conditions were similar in 60% and 80% loads. The patients with CHF showed a marked increase in diastolic pulmonary artery pressure, but a relatively small increase in HR suggesting Frank-Starling mechanism in elevating SV and stroke work. The control group had a rapid increase in diastolic PAP and a relatively small increase in HR, which was similar to that seen in patients with CHF. The main result of this study was that LV function remains stable during lower body resistance exercise in well-compensated patients with CHF.

Summary

The main result of reviewing the literature is that LV responses during steady-state exercise and resistance exercise are surprisingly similar in healthy people and in patients with CHF. The capability for patients with CHF to work against heavy loads during lower body resistance exercise has not been shown to result in deterioration of LV function. During steady-state exercise, healthy people seem to use the Frank-Starling mechanism at the beginning of the exercise, but as the SVR falls continuously during exercise the maintenance of the SV is possible without a further increase in EDV. They therefore do not need to use the Frank-Starling mechanism any further. The patients with CAD and patients with CHF compensate well to steady-state exercise and respond in a similar way as healthy subjects.

The two studies that have studied the responses to dynamic resistance exercise in patients with CHF have only utilized lower body resistance exercise. The same responses have been seen as in healthy people, although the magnitude of the cardiovascular changes has been smaller in patients with CHF. During resistance exercise, an increase in SV is mainly due to increased HR, and increases in the SBP/ESV ratio could indicate increased contractile function in patients with CHF during lower body resistance exercise.

REFERENCES

1. Upton MT, Rerych SK, Roeback JR, Newman GE, Douglas JM, Wallace AG, Jones MD. Effect of brief and prolonged exercise on left ventricular function. *Am J Cardiol* 1980;45:1154-1160.

2. Foster C, Gal RA, Port SC, Schmidt DH. Left ventricular ejection fraction during incremental and steady-state exercise. *Med Sci Sports Exerc* 1995;27:1602-1606.

3. Palatini P, Bongiovi S, Mario L, Schiraldi C, Mos L, Pessina AC. Above-normal left ventricular systolic performance during exercise in young subjects with mild hypertension. *Eur Heart J* 1995;16:232-242.

4. Foster C, Gal RA, Murphy P, Port SC, Schmidt DH. Left ventricular function during exercise testing and training. *Med Sci Sports Exerc* 1997;29:297-305.

5. Foster C, Meyer K, Georgakopoulos N, Ellestad AJ, Fitzgerald DJ, Tilman K, Weinstein H, Young H, Roskamm H. Left ventricular function during interval and steady-state exercise. *Med Sci Sports Exerc* 1999;31:1157-1162.

6. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol* 1985;58:785-790.

7. Sagiv M, Metrany R, Fischer N, Fisman EZ, Kellermann JJ. Comparison of hemodynamic and left ventricular responses to increased after-load in healthy males and females. *Int J Sports Med* 1991;12:41-45.

8. MacDougall JD, McKelvie RS, Moroz DE, Sale DG, McCartney N, Buick F. Factors affecting blood pressure during heavy weight lifting and static contractions. *J Appl Physiol* 1992;73:1590-1597.

9. Sullivan J, Hanson P, Rahko PS, Folts JD. Continuous measurement of left ventricular performance during and after maximal isometric deadlift exercise. *Circulation* 1992;85:1406-1413.

10. Lentini AC, McKelvie RS, McCartney N, Tomlinson CW, MacDougall JD. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. *J Appl Physiol* 1993;75:2703-2710.

11. Sagiv M, Hanson P, Besozzi M, Nagle F. Left ventricular responses to upright isometric handgrip and deadlift in men with coronary artery disease. *Am J Cardiol* 1985;55:1298-1302.

12. Featherstone JF, Holly RG, Amsterdam EA. Physiologic responses to weight lifting in coronary artery disease. *Am J Cardiol* 1993;71:287-292.

13. Delahaye N, Cohen-Solal A, Faraggi M, Czitrom D, Foult JM, Daou D, Peker C, Gourgon R, Le Guludec D. Comparison of left ventricular responses to the six-minute walk test, stair climbing, and maximal upright bicycle exercise in patients with congestive heart failure due to idiopathic dilated cardiomyopathy. *Am J Cardiol* 1997;80:65-70.

14. Meyer K, Foster C, Georgakopoulos N, Hajric R, Westbrook S, Ellestad A, Tilman K, Fitzgerald D, Young H, Weinstein H, Roskamm H. Comparison of left ventricular function during interval versus steady-state exercise training in patients with chronic congestive heart failure. *Am J Cardiol* 1998;82:1382-1387.

15. Elkayam U, Roth A, Weber L, Hsueh W, Nanna M, Freidenberger L, Chandraratna AN, Rahimtoola SH. Isometric exercise in patients with chronic advanced heart failure: hemodynamic and neurohumoral evaluation. *Circulation* 1985;72:975-981.

16. Reddy HK, Weber KT, Janicki JS, Mcelroy PA. Hemodynamic, ventilatory and metabolic effects of light isometric exercise in patients with chronic heart failure. *J Am Coll Cardiol* 1988;12:353-358.

17. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall JD. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol* 1995;76:977-979.

18. Meyer K, Hajric R, Westbrook S, Haag-Wildi S, Holtkamp R, Leyk D, Schnellbacher K. Hemodynamic responses during leg press exercise in patients with chronic congestive heart failure. *Am J Cardiol* 1999;83:1537-1543.