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TWO-DIMENSIONAL ECHOCARDIOGRAPHIC EVALUATION OF UPRIGHT EXERCISE: COMPARISON OF LEFT VENTRICULAR VOLUMES IN NORMAL AND POST-MYOCARDIAL INFARCTION SUBJECTS

The Ohio State University

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OF UPRIGHT EXERCISE: COMPARISON OF LEFT VENTRICULAR
VOLUMES IN NORMAL AND POST-MYOCARDIAL INFARCTION SUBJECTS

DISSERTATION

Presented in Partial Fulfillment of the Requirements for
the Degree Doctor of Philosophy in the Graduate
School of The Ohio State University

By
Walter Rolph Thompson, B.S., M.A.

* * * * *

The Ohio State University
1983

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To the memory of

EDWARD L. FOX, Ph.D.

Teacher, Adviser, Friend
ACKNOWLEDGEMENTS

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CHAPTER I

INTRODUCTION

The evaluation of left ventricular function using non-invasive procedures has long been a problem for clinicians. Cardiac angiography, although invasive and quite costly, remains the standard by which most techniques are compared. However, because of the costs involved, the invasive nature, and the lack of facilities in most hospitals and medical centers, angiography has not been a procedure of first choice in the evaluation of borderline or questionable cases.

Another technique often used in the evaluation of cardiac function is echocardiography. Until recently, this procedure has been used primarily in the evaluation of valvular diseases. However, with the introduction of two-dimensional echocardiography, cardiac function may be evaluated qualitatively by describing ventricular wall motion abnormalities and quantitatively by measurement of volumes and volume changes. Inotropic responses (measured as ejection fraction, velocity of circumferential shortening, percent fractional shortening, etc.) can also be calculated from the two-dimensional echocardiogram.
Purpose of the Study

The purpose of this particular investigation is to study changes in left ventricular volume using two-dimensional echocardiography at rest and compare the findings with those immediately following physical stress in two groups of men:

Group A: Men who have had a documented myocardial infarction, and,

Group B: Men who have NOT had a documented myocardial infarction and are considered apparently normal (controls).

Limitations of the Study

Limitations exist in every study, this one is no exception. The following is a list of such limitations imposed by the echocardiographic technique. These are recognized and kept to a minimum in this present investigation by practicing standard and accepted procedures (21, 56).

1. Subject Size: Differences in body surface area are the most significant influence in determining chamber dimensions. The larger individual (i.e., those with a larger body surface area) has a greater chamber dimension. Our measurements normalize for differences in body surface area.

2. Subject Position: The upright sitting (or standing) position with its inherent reduction in systemic venous return decreases
ventricular chamber sizes.

3. Transducer Placement: Standard transducer placements have been recommended as suprasternal, apical, subcostal and parasternal. Long axis, short axis, two and four chamber refer to the anatomic planes.

4. Heart Rate: Alterations in left ventricular end-diastolic and end-systolic dimensions are predictably produced with increases in heart rate during exercise. Accounting for those adjustments requires alterations in the quantitative analysis.

5. Heart Rhythm: Diastole (or the diastolic filling time) is disrupted in the presence of changes in the heart rhythm. Especially true when the atria do not contract, left ventricular end-diastolic dimensions may be decreased in the presence of premature ventricular contractions. To correct for this, only sinus beats were analyzed quantitatively.

6. Left Ventricular Preload and Aortic Impedance: Systemic vascular resistance may decrease or increase ventricular dimensions. A decrease in venous return (left ventricular preload), as in the upright or standing position, decreases chamber size. An increase in systemic vascular resistance (i.e., afterload) increases chamber size.

7. Physical Conditioning: Decreases in heart rate at rest and submaximal exercise are well documented. As the resting heart rate decreases, the left ventricular diastolic dimension increases and systolic dimension decreases (thus increasing stroke volume).
8. Intraobserver and Interobserver Variability: As with all dimension studies, some variability will exist between and among observers. Absolute requirements must be followed by observers to promise accuracy in measurements.

Statement of the Problem

Only recently has the emphasis shifted from resting supine echocardiographic studies to conditions in which physical stress is imposed. The problem to which this study attempts to address is the assessment of left ventricular volumes in the two aforementioned groups of men under resting and exercise conditions. The advantages of exercise echocardiography are outlined in Table 1.

Definition of Specific and Related Terms

For an extensive list of definitions see Appendix A.
Table 1

Advantages of Exercise Echocardiography*

<table>
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<tr>
<td>Safe, non-invasive, repeatable</td>
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<tr>
<td>Can continuously image heart during and after exercise</td>
</tr>
<tr>
<td>Can be performed with equipment already available in most cardiology facilities</td>
</tr>
<tr>
<td>Directly images left ventricular wall motion</td>
</tr>
<tr>
<td>Can visualize wall thickening and thinning</td>
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* From: Meltzer, R. S., C. A. Visser, and J. Roelandt. 
CHAPTER II

REVIEW OF RELATED LITERATURE

A comprehensive review of the literature concerning exercise is not feasible due to the prodigious amount of information compiled. The amount of work reported about echocardiography is also monumental. This review will therefore, contain itself with two basic areas. First, it will deal with the uses of two-dimensional echocardiography. Secondly, this chapter will review the literature concerning two-dimensional echocardiography as a tool in investigating the exercise response.

A review of the literature in echocardiography includes the study of left ventricular function, infarct sizing, identification of right ventricular infarction, the coronary anatomy (especially identification of the left main coronary artery), complications of chronic coronary artery disease, valvular diseases (mitral valve, tricuspid valve, aortic valve, and pulmonary valve diseases) and other evaluations including the detection of endocarditis, pericardial effusion, septal defects and cardiac masses such as tumors. A comparison of echocardiography with other procedures follows this review. Contributions to the literature of exercise echocardiography reveals very little information regarding technique.
Left Ventricular Function

Since the early work of Tennant and Wiggers in 1935 (68), ligation (or occlusion) of a coronary artery was shown to cause abnormal contraction of the subsequently ischemic muscle. More recently, Goldstein and DeJong (25) observed decreases in systolic ventricular wall thickening after coronary arterial occlusion. After reperfusion of the coronary artery ligated, a return to normal contractile properties was observed. Both of these studies were performed utilizing open-chested anaesthetized animals. The consequences of regional wall motion abnormality were realized yet no non-invasive technique was yet available to detect such an abnormality until the invention and modernization of echocardiography.

Most recently, experimental studies have demonstrated echocardiographic evidence of wall motion abnormalities within five to ten beats after ligation of a coronary artery (22, 69, 81). Clinically, abnormalities of wall motion can now be detected almost immediately with onset of anginal pain in patients with unstable angina pectoris or acute myocardial infarction (55). A great deal of recent research activity has appeared in professional journals using animals (35, 36) and human patients during acute ischemia or myocardial infarction (13, 19, 29, 30, 31).

It may not be possible to obtain an echocardiogram on a patient during the acute attack of myocardial infarction because of the uncertain nature of the disease process. Almost all patients with acute transmural infarction demonstrate some type of regional wall
motion disturbance (7). The patient with a non-transmural or subendocardial infarction may not present with wall motion abnormalities (52). The echocardiogram recorded within minutes, or a very few hours, may diagnostically rule out transmural involvement owing to the absence of wall motion disturbance.

By defining the areas of segmental wall motion abnormality, echocardiography has been shown to correctly identify the location of the infarcted zone between 84 and 95% of the time (13, 29, 52, 70). These studies compared electrocardiographic evidence with data collected echocardiographically for comparison purposes. Postmortem examinations with subsequent location of the infarction, compared favorably to antemortem studies of segmental wall motion as described by echocardiographic evaluation (29, 77).

Infarction Sizing

Clinicians have found it very difficult to determine the extent of myocardial involvement during acute infarction, though several studies have made the attempt (30, 45, 52, 70). More recently, attempts have been made using radionuclide imaging procedures. Thallium 201 scintography has been used with some success (5, 51, 71, 73, 74) as well as Technetium-99m pyrophosphate scanning (10, 84) and radionuclide cineangiography (50). Morrison et al. (50) studied estimates of infarct size using Thallium 201 scans and gated blood pool radioangiograms and creatine kinase curves. They found good correlation in only 14 of 35 patients
between any of these procedures. Two-dimensional echocardiography has been utilized by studying the extent of wall motion abnormality before and after acute myocardial infarction (31) and the extent to which the segment of abnormality is involved. As has already been pointed out and well documented, the transmural infarction predisposes the infarcted area to dyskinesis and/or akinesis and can be visualized on the echocardiogram. As two-dimensional echocardiography becomes more sophisticated and accepted as a diagnostic instrument in the evaluation of acute myocardial infarction by virtue of its analysis of ventricular wall motion, further study may include its role in infarct sizing. Furthermore, infarct expansion has become of interest owing to the complications often observed without further necrosis of ventricular muscle mass (33).

Right Ventricular Infarction

Right ventricular infarction may be a complication associated with extensive left ventricular infarction and is thought to be uncommon (40, 64, 72). Wartman and Hellerstein (76) found true right ventricular infarction in only 2.2% of 160 autopsied patients. Other studies have shown no true right ventricular infarction but as extensions of left ventricular infarction, especially of the inferior variety (34, 72). D'Arcy and Nanda (15) describe two-dimensional echocardiographic features of ten patients right ventricular infarction which included akinetic diaphragmatic wall, akinetic free wall, right ventricular enlargement, abnormal ventricular septal
motion, tricuspid regurgitation, and dyskinetic free wall.

**Coronary Anatomy**

A recent application of two-dimensional echocardiography is the visualization of the left main coronary anatomy. The prognosis of patients with left main disease is not good (27). Therefore, it is important to the clinician to be able to identify left main coronary artery obstruction. Furthermore, a non-invasive procedure in its detection may decrease the number of cardiac angiograms performed in the search of left main disease. Weyman et al. (79) first reported visualization of left main obstruction in 1976. Subsequent studies have reported similar results (9, 10, 53). More recently, Rink et al. (61) studied 26 males and 5 females correctly identifying all four patients with significant lesions in the left main coronary artery. Of the twelve patients with angiographically documented proximal left anterior descending coronary artery disease, echocardiography revealed nine with no left main coronary artery obstruction, two with "diseased" left main coronary arteries and one with left main coronary artery obstruction. These results reveal the sensitivity of echocardiography in the detection of left main disease of significant obstruction, yet more distal arteries are not yet visualized with this technique.
Complications of Chronic Coronary Artery Disease

Left ventricular aneurysms are a complication of myocardial infarction and have been shown to predispose a patient to ventricular arrhythmias, congestive heart failure, and embolization (7). Weyman et al. (83) detected 100% of ventricular aneurysms using two-dimensional echocardiography whereas conventional M-mode echocardiography detected only 47%. Block and Popp (7) suggest two-dimensional echocardiography may be more sensitive to ventricular function with aneurysm than angiography. Aneurysmal obstruction of left ventricular function in angiography, not a problem encountered with echocardiography, may indicate the latter as the investigative procedure of choice.

Left ventricular mural thrombi are currently under investigation for detection by two-dimensional echocardiography (2, 3, 16, 44, 59). These studies show a high predictive accuracy of echocardiography in the detection of thrombi. The importance is quite apparent and a non-invasive technique in its evaluation is essential.

Left Ventricular Outflow Obstruction

Outflow tract obstruction includes aortic stenosis (60) or aortic coarctation (80). Both of these structural problems may be evaluated by two-dimensional echocardiography. The former can be further defined as subvalvular, hypertrophic or subvalvular. Coarc-tation of the aorta was correctly identified in 16 of 18 patients
Tricuspid Valve Disease

All three leaflets of the tricuspid valve can be identified by two-dimensional echocardiography (38). Further evaluation of the inferior vena cava and right atrium in evaluating tricuspid regurgitation by strategic placing of the transducer can also be accomplished. Tricuspid valve prolapse (41) and Ebstein's anomaly of the tricuspid valve (58) can also be diagnosed using this technique.

Pulmonary Valve Disease

Two-dimensional echocardiographic determination of pulmonary valve stenosis is thought to be more sensitive than M-mode echocardiography (38, 82). In the normal heart, pulmonary valve leaflets move apart rapidly at the onset of systole and remain parallel to the walls of the pulmonary artery the duration of systole. Two-dimensional echocardiography reveals a "doming" of the pulmonary valve leaflets during systole (82).

Other Uses of Two-Dimensional Echocardiography

Two-dimensional echocardiography has been used extensively in the past in the detection of endocarditis (48), in the evaluation of prosthetic valves (6, 32), and pericardial effusion (42). Cardiac masses such as tumors have been successfully diagnosed using
two-dimensional echocardiography in several studies (16, 57) as have aortic aneurysms (17, 47). Septal defects have been studied with some success as well (18, 39).

**Procedural Comparisons**

It is generally accepted that two-dimensional echocardiography underestimates left ventricular volume in comparison to ventriculography (8, 20, 54, 63). This underestimation is as high as 30 to 49% in some investigations. Schiller et al. (63) found a good correlation in the calculation of left ventricular end-systolic volume (r=.90), end-diastolic volume (r=.80) and ejection fraction (r=.87).

They compared phased-array echocardiography to biplane cineangiography in 30 patients with wall motion abnormalities (78). Similarly, Silverman et al. (65) reported a comparison in ten children. Two-dimensional echocardiography and biplane cineangiography were used for comparison. They found a good correlation in the calculation of left ventricular end-systolic volume (r=.94). Other investigations have used contrast two-dimensional echocardiography and contrast ventriculography (4), M-mode echocardiography and cardiac angiograms (26) and a description of ventricular asynergy utilizing real-time, two-dimensional echocardiography and cineangiography (37).

All of these reports indicate a perfect correlation may not be possible because of fluctuations of left ventricular volume between studies and a lack of precise endocardial definition by two-dimensional echocardiography (4).
Exercise Echocardiography

In evaluating left ventricular reserve, Sugishita and Koseki (67) compared 46 healthy persons and 47 cardiac patients performing bicycle exercise in the supine position. Both groups consisted of males and females and were neither age nor sex matched. Grouping the patients into various subgroups dependant upon functional classification allowed these researchers the opportunity to study a wide range of diseases affecting the left ventricle. For example, the cardiac patient group included history of hypertension, mitral valvular disease, aortic valvular disease, idiopathic cardiomyopathy and "various other conditions". Using the equation of Gibson (23) for the calculation of left ventricular end-diastolic volume and left ventricular end-systolic volume, Sugishita and Koseki (67) found a correlation coefficient of \( r = 0.96 \) between cardiac output measurements taken by echocardiography and the dye-dilution method when both techniques were performed simultaneously at rest and at steady-state exercise at a heart rate equal to 100 beats per minute. They further reported in the 83% of the patients in whom adequate studies could be performed, exercise echocardiograms were also adequate.

Demonstrating the difficulty in ascertaining good studies using echocardiography, 71% of the patients studied by Wann et al. (75) presented adequate exercise echocardiograms for study. They found dynamic echocardiography to be technically difficult yet feasible in the detection of exercise-induced myocardial ischemia. Volume
measurements were not attempted. Ventricular wall motion was described qualitatively by reporting motion patterns as normal, hypokinetic, akinetic or dyskinetic. Descriptions of wall motion were made at rest, immediately upon termination of exercise (symptom-limiting end-point or 85% of the age-predicted maximum heart rate), and after sublingual nitroglycerine administration. The authors conclude the use of exercise in conjunction with echocardiography expands the usefulness of echocardiography in the detection of ventricular wall motion abnormalities.

Mason and colleagues (43) from Johns Hopkins University initially examined 83 people using M-mode echocardiography. Fifty-four persons (65%) presented quality echocardiograms. A quality echocardiogram was described as one in which both sides of the interventricular septum and the posterior left ventricular free wall are visualized during the entire cardiac cycle. Only 24 of this group (44%) presented quality echocardiograms during exercise. It is this latter group which the authors describe in their report. Of the 24 individuals, eight were normal volunteers, three persons had exhibited chest pain on exertion with normal angiograms (chest pain was of "uncertain" etiology) and 13 were patients with documented coronary artery disease. The authors were investigating primarily contractility of the left ventricle. Percent systolic wall thickening and percent systolic change in diameter were the principle investigative measurements in addition to maximum velocities of wall thickening in systole, wall thinning in diastole,
decrease of diameter in systole, and increase of diameter in diastole. As one might assume, those patients with documented advanced coronary artery disease demonstrated negative inotropic responses immediately post-exercise by evidence of changes in the above-mentioned parameters. The authors conclude exercise echocardiography has the advantage over exercise cineangiography in that it is non-invasive and thus easily repeatable. Exercise echocardiography is comparatively inexpensive, and has the capability of comparing many cardiac cycles.

Studying ten healthy men aged 18 to 23 years, Stein et al. (66) used M-mode echocardiography during supine bicycle exercise. The authors studied the subjects previous to the exercise portion obtaining quantifiable echocardiograms on each. Their results indicated no change in the end-diastolic dimension during exercise, and a significant decrease in end-systolic dimension. The calculated stroke dimension (difference between end-diastolic dimension and end-systolic dimension) increased by 6.2% during exercise. Recovery data demonstrate a significant increase in the end-diastolic dimension in all subjects. A decrease in end-systolic dimension during recovery as the end-diastolic dimension increased was also shown.

In 1979, Amon and Crawford (1) studied upright exercise echocardiography in 18 normal males. The subjects were chosen for the study after adequate resting echocardiograms were analyzed. Approximately 20% of the subjects screened were accepted as subjects for this study. Of the initial 18 men chosen, only 12 subjects' data were chosen for statistical and comparative purposes. The results indicate no
differences in the end-diastolic dimension between upright and supine exercise. There was a progressive decrease in the end-systolic dimension under both conditions. The percentage shortening (ejection fraction) increased during exercise under both conditions. The authors admit the difficulty in obtaining quantifiable echocardiograms. They state only one in five studies (20%) were adequate for quantification. Various cardiopulmonary diseases complicate the recording. Second, these authors use M-mode echocardiograms which may influence the quality of the study. Third, only nine minutes of exercise were recorded for comparative purposes. Some of the more athletic subjects continued to pedal for nearly 18 minutes. Perspiration and depth of breathing complicated the echocardiogram procedures. The authors conclude the future of exercise echocardiography may be beneficial to assess the effects of the disease process and cardioactive drugs.

Zwehl et al. (85) studied ten healthy volunteers aged 16 to 48 years utilizing the supine position during exercise. Two-dimensional echocardiography revealed no difference in end-diastolic short axis area from rest to exercise. End-systolic area decreased significantly. This is in agreement with the aforementioned of Stein et al. (66). Reproducibility of two-dimensional echocardiographic measurements at rest demonstrated correlation coefficients ranging from .87 to .96 and standard errors of the estimate from .65 to 1.63 cm². During exercise, reproducibility correlation coefficients ranged from .90 to .99 with standard errors of the estimate ranging from
Morganroth et al. (49) achieved adequate echocardiographic studies in 78% of the 55 patients studied during supine exercise echocardiography. The primary reason for the remaining inadequate studies was due to interference of the breathing pattern (hyperventilation) immediately upon conclusion of the bout of exercise. Again in this study, nearly half of the subjects were receiving propranolol therapy. Although the investigators were primarily interested in wall motion abnormalities during exercise echocardiography and its correlation to coronary angiography, it is worthy to note the high sensitivity of echocardiography and the extent of coronary artery disease diagnosed by angiography. The authors conclude an important role exercise echocardiography may play in the non-invasive detection of coronary artery disease. Although volumetries were not considered, regional wall motion disturbances were highly predictive of coronary disease.

Recently, upright exercise echocardiography has been the interest of Crawford et al. (14). They report biapical echocardiograms on 18 of 25 subjects (72%) studied. Wall motion abnormalities were described as were volume determinations (left ventricular end-diastolic volume, left ventricular end-systolic volume, ejection fraction). Nitroglycerine administration to the subjects (25 men with angina pectoris) 30 minutes after an initial bout of exercise on the bicycle ergometer provided the intervention. The subjects repeated the bout of exercise under this condition. Ten males served as a control group. The results indicated an increase in ejection
fraction of the normal control subjects from 57% at rest to 71%
immediately after peak exercise. The patients showed the improve­
ment only after nitroglycerine administration. The importance of
this study appears to be the beneficial aid of nitroglycerine as
evidenced by the increase in ejection fraction during exercise in
response to the demand of physical work. Second, quantification
of left ventricular volumes by echocardiography is an acceptable
procedure to demonstrate exercise-related stress. The investig­
gators did not alter medications already taken regularly by the
subjects. Thirteen of the eighteen patients whose echocardiograms
were adequate to determine volumes were taking propranolol and
isosorbide dinitrate. Five of these patients were also taking
digoxin. Isosorbide dinitrate was discontinued 12 hours before
the test but propranolol and digoxin were taken as usual.

There has been no conclusive study to date to determine which
parameter influences stroke volume the greatest under exercise
conditions. There has been some suggestion of a decrease in the
end-systolic dimension to increase stroke volume but these studies
have been under conditions in which end-diastolic volume were as­
sumed to be maximal (i.e., supine). This study will investigate
central circulatory changes in the upright position to determine
if end-diastolic volume or end-systolic volume changes to increase
stroke volume, cardiac output, and ejection fraction in two groups
of age-matched men.
CHAPTER III

METHODS AND PROCEDURES

The primary purpose of this study was to investigate the effects of an acute bout of exercise in the upright position on left ventricular volumes in two groups of age-matched men. The difference between the groups was the presence of myocardial scarring due to a previous myocardial infarction in one of the groups. The methods and investigative procedures will be explained in this chapter.

Subjects

The subjects for this study were male, aged 30 to 67 years. Group A consisted of volunteers from the Phase III Cardiac Rehabilitation Program of Swedish Covenant Hospital, Chicago, Illinois. A documented transmural myocardial infarction by virtue of pathologic Q waves on the electrocardiogram or elevation of cardiac enzymes (i.e., CPK, SGOT, LDH) consistent with myocardial infarction during the hospital stay made the men eligible for this study and inclusion in Group A. A further restriction was the absence of positive inotropic medications (i.e., digitalis). Negative inotropic drugs were allowed if it did not effect the chronotropic response to stress. All medications were taken as usual. This was determined by previous graded exercise testing (i.e., achievement
of age-predicted maximal heart rate). Group B consisted of normal volunteers (see Table 2). All subjects were instructed as to the changes that occur during an acute bout of exercise before obtaining written informed consent.

Echocardiographic Procedures

Advanced Technology Laboratories (P.O. Box 6639, Bellevue, Washington 98007) Mark 300 Cardiology Ultrasonic Scanning System for high resolution ultrasound cardiac imaging was used for the recording of the echocardiogram (figure 1). The Mark 300 C Ultrasound System is capable of scanning at 3.0 MHz and 5.0 MHz to ensure optimal resolution on all patients. Two-dimensional sector scanning allows for both real and freeze frame modes on the two-dimensional sector scanner. The viewing range starts at the chest wall. A fanning effect allows viewing from 3.5 to 22.5 centimeters in 16 depth settings. The Advanced Technology Laboratories 722A Ultrasound In-line Scan head allows for a 90 degree sector scan in line with the length of the scan head body at a frequency of 3.0 MHz.

A Panasonic VHS video cassette recorder stores all displays for subsequent playback and analysis on standard half-inch recording tape. The Advanced Technology Laboratories 130A Line Scan Recorder provides freeze frame hard copies of both two-dimensional and M-Mode prints. A nine inch video monitor located on top of the control panel allows the technician comfortable viewing during the recording session.
<table>
<thead>
<tr>
<th>Group</th>
<th>AGE (YEARS)</th>
<th>HEIGHT (CM)</th>
<th>WEIGHT (KG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>X 54.60</td>
<td>173.50</td>
<td>81.21</td>
</tr>
<tr>
<td>(n =15)</td>
<td>SD 7.80</td>
<td>5.88</td>
<td>11.59</td>
</tr>
<tr>
<td>Group B</td>
<td>X 48.64</td>
<td>172.50</td>
<td>75.65</td>
</tr>
<tr>
<td>(n =11)</td>
<td>SD 7.27</td>
<td>5.59</td>
<td>12.90</td>
</tr>
</tbody>
</table>
Figure 1. Advanced Technology Laboratories Mark 300 Cardiology Ultrasonic Scanning System
Figure 2 demonstrates transducer location illustrating two-dimensional echocardiographic features. The SUPRASTERNAL location is produced by placing the transducer in the suprasternal notch. Positioning of the transducer near the midline of the body beneath the lowest ribs is referred to as the SUBCOSTAL location. The APICAL position refers to the left apex unless otherwise described. The apical location is found by palpating the apical impulse and...
placing the transducer on that particular location. The entire left side bounded by the left clavicle, sternum and apical region is referred to as the PARASTERNAL position. Parasternal terminology assumes left parasternal if the apical impulse is palpated in the ordinary left side. Under the unusual circumstance of right apical positioning, the parasternal side will be assumed as the right side and is thus indicated. Under ordinary conditions, as the apical location is assumed to be left apical, parasternal will be assumed left parasternal. In those conditions where the apex is located on the right side, the apical location will be noted as right apical. The region bound by the right clavicle, sternum and right apex is known as the right parasternal region.

**Imaging Planes**

Imaging planes utilized by two-dimensional echocardiography include: long-axis, short-axis and four-chamber planes (figure 3). The LONG-AXIS transects the heart perpendicular to the dorsal and ventral surfaces of the body and from base to apex of the heart. The two-dimensional image, achieved by placing the transducer in the parasternal location (left side bounded by the left clavicle, sternum and apex), is a long-axis view (transecting the heart from base to apex) and is known as the "parasternal long-axis view". The imaging plane perpendicular to the long-axis view is referred to as the SHORT-AXIS. The FOUR-CHAMBER view is obtained by placing the transducer approximately parallel to the dorsal and ventral
Figure 3. Diagram of the three orthogonal imaging planes used to visualize the heart with two-dimensional echocardiography. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

surfaces of the body and, as the label suggests, images a four-chamber view of the heart. If the apical position is used to illustrate a four-chamber view, "apical four-chamber view" is the nomenclature used to describe such an image. The description of an imaging view, then, describes which view is being sought and the transducer position.
Transducer Orientation

Located on the housing of the transducer exists an index mark. The index mark is used to identify the image projected onto the imaging screen. The index mark is always pointed either in the direction of the patient's head or to the left side of the patient. When the transducer is oriented accordingly, images can be clearly located and identified.

Long-axis views

The long-axis can be viewed from the apical, parasternal or suprasternal location (figure 4). The view in this illustration is of the left ventricle, the object of this investigation. Note the index mark is always pointed toward the patient's head. Figure 5 A illustrates the apical long-axis image and resultant image display. Use of this transducer location will result in an image displayed with the apex of the heart located at the top of the display, the left atrium inferior and to the left, the aorta inferior and to the right, and the right ventricle to the right. The posterior wall of the left ventricle is seen on the extreme left. The parasternal long-axis view is demonstrated in figure 5 B. Again, the index mark is pointed toward the patient's head. The image display shows right ventricle at the top, the apex of the heart is located on the left side and the aorta to the right. The left ventricle (left ventricular posterior wall) appears at the bottom of the display. With the index mark again pointing toward the patient's head, the suprasternal long-axis image is demonstrated in figure 5 C. In this display, the
Figure 4. Diagram of the transducer orientation used to obtain long-axis views. Note that the transducer index mark is always pointed either in the direction of the patient's head or the patient's left side. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

aorta is visualized at the top of the image display, the left atrium at the right, the right ventricle at the left and the left ventricle in the middle with the posterior wall of the left ventricle on the right side. Slight modifications in the transducer location in the suprasternal long-axis view enable the observer to image the ascending aorta, transverse aorta (or aortic arch with major arterial branches), descending aorta, and pulmonary artery.

Short-axis views

The short-axis views are usually visualized with the parasternal or subcostal transducer location (figure 6). As with all transducer positions, the index mark is once again pointed toward the
Figure 5. Illustration of the long-axis, two-dimensional images that result when the transducer is used to visualize the apical long-axis view (A), parasternal long-axis view (B), and suprasternal long-axis view (C). These images were obtained with the transducer index mark pointing to the patient's head. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

patient's head. The parasternal short-axis view (figure 7 A) images the interventricular septum at the top and to the left of the image display. The lateral papillary muscles can be seen to the right and the medial papillary muscles to the left. The left ventricular posterior wall is seen at the bottom. The subcostal short-axis view (figure 7 B) illustrates the right ventricle at the top of the display, the interventricular septum in the middle, and the left
Figure 6. Diagram of the transducer orientations to obtain short-axis views. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

ventricle on the bottom. Note the posterior wall of the left ventricle appears on the bottom and to the right of the image display.

Four-chamber views

The four-chamber view can be visualized from either the subcostal or apical transducer locations (figure 8). The apical four-chamber view affords the viewer two options (figure 9 A). The only difference between options one and two is the orientation of the structures in question on the image display. Operating the transducer by internally switching it "on" or "off" (utilizing an image inversion switch) allows the technician to distinguish between option one and two. The choice of options is purely arbitrary
dependant upon the choice of the technician performing the test. In both displays the images are identical. The subcostal four-chamber view also has two options when utilizing the image inversion switch.

Figure 7. Diagram of the short-axis, two-dimensional images that result when the transducer is used to visualize the parasternal short-axis view (A) and the subcostal short-axis view (B). These images were obtained with the transducer index mark pointing to the patient's left side. Abbreviations are as in figure 2. From Circulation, 62(2):212-216, 1980.

and is shown in figure 9 B. In option one, the right ventricle is shown at the bottom of the image display with the left ventricle and left atrium at the top. The apex of the heart appears on the right side. By inverting the image, option two is visualized. The
Figure 8. Diagram of the transducer orientations to obtain four-chamber views. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

right ventricle appears at the top of the screen, the apex appears on the right side and the atria appear on the left side. The sub-costal four-chamber view, as in the apical four-chamber view, also displays the same images whether in option one or option two. The choice is, again, left to the discretion of the technician.

Variations between laboratories exist yet standard nomenclature and procedures should be followed to provide continuity between laboratories. The standard transducer locations and image planes with resulting transducer orientation that have been presented make it easier to compare existing studies, allowing a contiguous flow of information between hospitals, medical centers and institutions of research.
Figure 9. Four-chamber, two-dimensional images that result when the transducer is used to visualize the apical four-chamber view (A) and the subcostal four-chamber view (B). These images were obtained with the transducer index mark pointing to the patient's left side. Two options are included for each four-chamber view. In each case, option 1 is produced by activation of the image inversion switch which results in the near signals of the image being inverted from the top to the bottom of the display. Abbreviations are as in figure 2. From CIRCULATION, 62(2):212-216, 1980.

Exercise Testing Mode

To elicit the desired exercise response, Quinton Instrument Company Model 846-T Tilting Imaging Table (figure 10) was used. This
Figure 10. Quinton Instrument Company Model 846-T Tilting Imaging Table.

table allows flexibility of adjustment to accommodate individual subjects. A powered actuator allows an adjustment of the angle from supine (zero degrees) to full upright (90 degrees). Two types of bicycle seats are provided for comfort and ease of pedalling. Pedal length and angle are independent to suit individual subjects. Handgrips and restraints are also adjustable. Stability of the torso is provided for by these adjustable handgrips, restraints and shoulder
harness. Quinton Instrument Company Model 845 Electronic Ergometer allows a constant workload at a comfortable pedalling rate at workloads from 200 to 2400 kilogram meters per minute (kgm·min⁻¹) in 50 kgm·min⁻¹ increments.

**Exercise Test Protocol**

The Quinton Instrument Company Model 846-T Tilting Imaging Table was set approximately 70 degrees in the upright position. (figure 11 and 12). The volunteer subjects were appropriately strapped at the pelvic area by way of a side-to-side 2 inch strap. The head was placed in a comfortable position between two shoulder braces. A pillow was provided for comfort. The hands rested on handgrips projecting from each side of the table. The feet were placed in boot-like straps on the pedals. The appropriate length of seat to pedal was adjusted for leg length for each subject. Conformance to these procedures insured comfort for the subject and minimal movement of the torso (i.e., region of the thorax) during exercise.

A pre-determined heart rate equal to 85% of the maximal heart rate achieved during previous graded exercise testing was the determining end-point. The subject was instructed to respond to questions concerning perceived exertion during the test. Terms such as "mild", "moderate", "severe", and "exhausted" were used. None of the subjects reached the "exhausted" stage although the greatest complaint following the testing procedure was localized leg fatigue. The patients were also instructed to pay particular attention to
Figure 11. Quinton Instrument Company Model 846-T Tilting Imaging Table set at approximately 70 degrees (front).
Figure 12. Quinton Instrument Company Model 846-T Tilting Imaging Table set at approximately 70 degrees (side).
indications of angina pectoris.

The testing protocol began with no resistance at 50 revolutions per minute (rpm) for two minutes. Increments of 200 to 300 kgm\(\cdot\)min\(^{-1}\) provided the work (speed was held constant at 50 rpm). Heart rates were recorded at the end of each minute on a PhysioControl Life Pak V defibrillator-monitor. Arterial blood pressure was obtained at the end of each two minute stage in the right arm by auscultation and duly recorded.

An echocardiogram was recorded in the upright sitting position (in the exercise mode) at rest. The apical four-chamber view was recorded followed by the apical two-chamber view (slight clockwise rotation of the transducer recording only the left atrium and left ventricle). The technician identified the appropriate location for duplication immediately post-exercise. At least one minute of each view was recorded before the exercise test. Immediately post-exercise, the subject remained as motionless as possible while the technician recorded first an apical four-chamber view followed by an apical two-chamber view. Fifteen seconds of each view were recorded for one minute post-exercise.

At the conclusion of the one minute echocardiographic recordings, the subject was allowed to pedal slowly against no resistance until the heart rate approximated the pre-exercise rate. None of the subjects complained of adverse symptoms with the exception of localized leg fatigue.
Quantification Procedures

The area-length method of calculating ventricular volumes was utilized in this study (21, 78). Assuming the left ventricle resembles a prolate ellipse, the mathematical formula for calculating its volume is:

\[
\text{Volume} = \frac{4}{3} \pi \left( \frac{L}{2} \right) \times \left( \frac{D_1}{2} \right) \times \left( \frac{D_2}{2} \right)
\]

where L is the long-axis, and D_1 and D_2 are the areas of the long-axis and short-axis dimensions. An Apple II Plus computer with the appropriate software package for quantification of the above equation was employed. Calculations included: end-diastolic volume (measured at the peak of the simultaneously recorded R wave on the electrocardiogram), end-systolic volume (measured at the peak of the T wave), stroke volume (difference between end-diastolic volume and end-systolic volume), cardiac output (product of heart rate and stroke volume), and ejection fraction (measured as the stroke volume divided by the end-diastolic volume). All measurements were made during end-expiration to avoid lung movements and for standardization purposes.

Reliability and Validity

Eight patients representing "worst case" studies were independently catheterized (i.e., cardiac angiograms) with subsequent
calculation of ventricular volumes. Two-dimensional echocardiograms provided comparative ventricular volumes. The observer measured ventricular volumes twice independently and blindly using the above equation. Validity correlation was calculated between the angiographic and echocardiographic volumes. The echocardiographic volumes were then tested for reliability by the independently determined second volume.

Statistical Analysis

The two-way analysis of variance (ANOVA) for repeated measures was used as the statistical analysis. A 2 X 2 factorial design for rest and exercise measurements was established. The F ratio needed to meet or exceed the .05 level to be significant. The Newman-Kuels procedure was used to determine which differences between means were significant.
Quality (i.e., measurable) echocardiograms were recorded on 13 of 15 (87%) subjects in Group A and 11 of 13 (85%) subjects in Group B. Validity and reliability data reflect measurements on six independent subjects in whom angiographically calculated volume determinations were made at rest. Echocardiographic volumes were determined with subsequent correlation coefficients calculated and compared for validity and reliability.

**Validity and Reliability**

Validity correlations were calculated based on angiographic data and echocardiographic observations. Table 3 demonstrates the correlation coefficients and the mean absolute difference for each of the measured variables.

Reliability data and respective correlations are presented in Table 4. These data are echocardiographic measurements made by the observer under independent and blind conditions.

**End-Diastolic Volumes**

Table 5 indicates the end-diastolic volumes for both the experimental and control groups at rest and after exercise conditions.
Table 3. Validity data showing correlation coefficients and mean absolute difference between angiographically determined volumes and volumes calculated from echocardiography. EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, CO = cardiac output, and EF = ejection fraction.
Table 4. Reliability data showing correlation coefficients and mean absolute difference between two echocardiographically determined series of volumes calculated blindly and independently. EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, CO = cardiac output, and EF = ejection fraction.

<table>
<thead>
<tr>
<th></th>
<th>Echocardiography I</th>
<th>Echocardiography II</th>
<th>r</th>
<th>mean absolute difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDV (ml)</td>
<td>160.33 ± 55.95</td>
<td>147.33 ± 57.19</td>
<td>.81</td>
<td>31.67</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>81.30 ± 48.42</td>
<td>68.33 ± 33.75</td>
<td>.97</td>
<td>13.33</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>67.00 ± 42.45</td>
<td>74.50 ± 27.15</td>
<td>.81</td>
<td>21.50</td>
</tr>
<tr>
<td>CO (L·min⁻¹)</td>
<td>4.02 ± 2.55</td>
<td>4.47 ± 1.63</td>
<td>.81</td>
<td>1.29</td>
</tr>
<tr>
<td>EF (%)</td>
<td>44.33 ± 16.95</td>
<td>45.50 ± 10.07</td>
<td>.87</td>
<td>11.17</td>
</tr>
<tr>
<td>Group</td>
<td>Rest</td>
<td>Exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------</td>
<td>------------</td>
<td>------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group A</td>
<td>125.38 ± 23.69</td>
<td>150.31 ± 33.91</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group B</td>
<td>119.89 ± 17.05</td>
<td>171.11 ± 32.91</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 5. End-diastolic volumes for Group A (patients) and Group B (normals). Data represent means ± standard deviation and are expressed in milliliters.
Two-way ANOVA for repeated measures indicated a significant interaction ($p < .05$) (see Appendix J). The Newman-Kuels post-hoc comparison demonstrated the following (see Appendix K).

1. There were no significant differences between the groups at rest ($p < .05$),
2. Group A (patients) did not significantly increase ($p < .05$) end-diastolic volume from rest to exercise and,
3. Group B (normals) did increase end-diastolic volume ($p < .05$).

**End-Systolic Volumes**

Table 6 indicates the end-systolic volumes for both groups at rest and after exercise conditions. The two-way ANOVA for repeated measures indicated no significant differences ($p < .05$) in main effects or interaction (see Appendix J).

**Stroke Volume**

Data calculated for stroke volume are shown in Table 7. Stroke volume was calculated by use of the following formula:

$$SV = EDV - ESV$$

where $SV$ = stroke volume, $EDV$ = end-diastolic volume, and $ESV$ = end-systolic volume. The two-way ANOVA for repeated measures revealed a significant interaction effect ($p < .05$) (see Appendix J). The Newman-Kuels post-hoc comparison disclosed the following (see Appendix K):
Table 6. End-systolic volumes for Group A (patients) and Group B (normals). Data represent means ± standard deviation and are expressed in milliliters.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>47.54 ± 12.98</td>
<td>47.69 ± 14.63</td>
</tr>
<tr>
<td>Group B</td>
<td>40.89 ± 14.65</td>
<td>37.67 ± 12.95</td>
</tr>
</tbody>
</table>
Table 7. Stroke volume calculated by the difference between end-diastolic volume and end-systolic volume and reported in milliliters. Data represent means ± standard deviation.
1. At rest, stroke volume was the same for both groups,

2. Group A (patients) did not change stroke volume in response to exercise conditions,

3. Group B (normals) increased stroke volume significantly (p < .05), and

4. There is a significant difference between the groups during exercise conditions (p < .05).

Ejection Fraction

Ejection fraction is calculated from the following formula:

\[ \frac{EDV - ESV}{EDV} = \frac{SV}{EDV} \]

where EF = ejection fraction, EDV = end-diastolic volume, ESV = end-systolic volume, and SV = stroke volume. Table 8 indicates the calculated ejection fractions of both groups under two conditions previously described. The two-way ANOVA for repeated measures on the data for ejection fraction indicated significance (p < .05) (see Appendix J). The Newman-Kuels post-hoc comparison revealed the following (see Appendix K):

1. Ejection fraction at rest is not significantly different between the two groups,

2. Group A (patients) did not alter ejection fraction in response to exercise,

3. Group B (normals) did change ejection fraction significantly (p < .05) as a response to exercise, and,
Table 8. Ejection fraction measured for Group A (patients) and Group B (normals) under resting and exercise conditions. Reported values are in percents. Data represent the means ± standard deviation.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>62.08 ± 7.24</td>
<td>67.23 ± 10.56</td>
</tr>
<tr>
<td>Group B</td>
<td>66.11 ± 9.71</td>
<td>77.67 ± 7.75</td>
</tr>
</tbody>
</table>
4. There exists a significant \( (p < .05) \) difference between Group A and Group B during exercise conditions.

Heart Rate

A two-way ANOVA for repeated measures was calculated for resting and exercise heart rates. A significant increase \( (p < .05) \) in heart rate was observed between resting and exercise conditions. No interaction effect was established. Table 9 summarizes heart rate data.

Cardiac Output

Table 10 summarizes cardiac output data for both groups under resting and exercise conditions. The following formula was used in the calculation:

\[
CO = HR \times SV
\]

where \( CO = \) cardiac output, \( HR = \) heart rate, and \( SV = \) stroke volume.

Two-way ANOVA for repeated measures indicated significant differences \( (p < .05) \) in the data (see Appendix J). The Newman-Kuels procedure revealed the following (see Appendix K):

1. No significant difference \( (p < .05) \) exists between the groups during resting conditions,

2. A significant increase \( (p < .05) \) exists in cardiac output for both groups in response to exercise, and

3. A significant difference \( (p < .05) \) exists between the groups during exercise conditions.
Table 9. Heart rate reported in beats per minute for Group A (patients) and Group B (normals). Data represent the means ± standard deviation.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>65.92 ± 12.46</td>
<td>126.85 ± 15.49</td>
</tr>
<tr>
<td>Group B</td>
<td>73.89 ± 6.85</td>
<td>137.44 ± 13.68</td>
</tr>
</tbody>
</table>

HEART RATE (b·min⁻¹)
(\(\overline{x} \pm SD\))
<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>$5.19 \pm 1.71$</td>
<td>$12.94 \pm 4.19$</td>
</tr>
<tr>
<td>Group B</td>
<td>$5.84 \pm 1.13$</td>
<td>$18.26 \pm 4.48$</td>
</tr>
</tbody>
</table>

Table 10. Cardiac output data reported in Liters per minute. Group A = patients, Group B = normals. Data represent the means ± standard deviation.
The purpose of this investigation was to examine changes in left ventricular volumes between rest and exercise conditions in a group of middle-aged men who had suffered myocardial infarctions and their normal counterparts. The exercise was performed in the upright position on a modified bicycle ergometer. Left ventricular volumes were calculated from measurements obtained through two-dimensional echocardiography.

The difficulty in obtaining quality (i.e., measurable) echocardiograms has been described elsewhere (14, 43, 66, 67, 75, 85). This present study obtained measurable left ventricular volumes in 87% of patients with myocardial infarctions and 85% of normal volunteers. Wann et al. (75) measured volumes in only 71% of patients in their study. Mason et al. (43) obtained quality echocardiograms on 65% at rest and 44% during exercise. Morganroth et al. (49) achieved measurements in 78% of patients in a study of supine exercise. Crawford et al. (14) reported 72% of those subjects studied were adequate for measurement. Other studies reported only those values of subjects in whom adequate volume determinations could be made (66, 85). All of these studies make an attempt to further describe the adaptability of the heart to changes in cardiac output between rest and exercise.
conditions. The high percentage of measurable echocardiograms in this study may reflect the progressive technology in this field.

A distinction needs to be made between the different positions in which exercise effects have been studied and their influence on both heart rate and stroke volume. Most of the earlier studies of ventricular volumes were performed in the supine position (66, 67, 85). The technology of the time required strict adherence to established guidelines and protocols to gather valid and reliable data. The data accumulated allowed researchers to conclude several important details in the adapting heart. Supine exercise in normal subjects demonstrated that in response to a greater demand for cardiac output during exercise, the heart adapted by increasing the rate only, while stroke volume remained unchanged. Because the supine position allows for greatest ventricular filling at rest, end-diastolic volume is said to be maximal. With increased stretch on the myocardial fibers due to greater ventricular filling, force of myocardial contraction is enhanced resulting in maximal stroke volume. The demands of exercise and concomitant demand for an increase in output by the heart can then only be met by an increase in heart rate in the supine position.

An increase in heart rate may be considered the most effective method of increasing cardiac output in the supine position for normal subjects. However, cardiac contractions beyond a certain heart rate (which may be inherent to the individual patient), may diminish cardiac output. An increase in heart rate results in a shortening
of diastole which limits ventricular filling time. Furthermore, coronary blood flow occurring during diastole is also limited. The results in victims of coronary artery disease is ventricular dysynchrony and a decrease in cardiac output. Therefore, in these patients supine exercise may be contraindicated because heart rate alone contributes to changes in cardiac output.

As the exercising subject becomes upright, several important factors need to be considered. Cardiac output must be maintained and increase in response to the greater demands of exercise. It is changes in stroke volume in the upright position, as well as heart rate, that affect cardiac output. The upright position lends itself to the effects of gravity by diminishing venous return and limiting end-diastolic volume. Thus, if end-systolic volume remained unchanged from the supine to the upright position, cardiac output (which is the product of two factors: heart rate and stroke volume) would be expected to decrease. If in the upright position stroke volume were decreased by a decrease of end-diastolic volume, an increase in heart rate would increase cardiac output. However, it has been shown that stroke volume also increases in response to upright exercise conditions. It is this response which this investigation addresses and the mechanism of stroke volume regulation.

During exercise, a redistribution of systemic blood flow away from tissues not being utilized in favor of enhanced flow to the exercising muscles occurs. In addition, venoconstriction, mediated by the sympathetic nervous system, and vasodilatation within the active
tissues shifts blood from the venous reservoirs (i.e., splanchnic area) to the heart, arterial system, and active muscle tissues. Since there is considerable decrease in arteriolar resistance caused by vasodilatation, these factors create a favorable effect on the pumping mechanism. An increase in venous return by vеноconstriction and enhanced by the so-called abdominothoracic pumps, and vasodilatation of metabolically active tissue has a favorable effect on stroke volume in the upright position.

The results of this study indicated that during upright exercise conditions, the normal group was able to capitalize by enhancing venous return and increasing end-diastolic volume from rest to exercise conditions \( (p<.05) \). This increased blood volume further enhanced stroke volume \( (p<.05) \) and together with the increased heart rate augments cardiac output. End-systolic volume remained unchanged indicating arteriolar vasodilatation of active tissues without interfering with systolic emptying. This study demonstrated that in the upright position an increase in end-diastolic volume from rest to exercise may be related to increased venous return. The ratio of stroke volume to end-diastolic volume (ejection fraction) also increased \( (p<.05) \) by this mechanism.

In those patients who have suffered a myocardial infarction, no changes in either end-diastolic volume or end-systolic volume were noted from rest to exercise conditions. The result of a myocardial infarction is an area of the left ventricular myocardium which necrosis resulting in scar tissue. As such it loses its distensibility.
The extent of ventricular involvement (i.e., the region of infarcted tissue) may influence how compliant the left ventricle is to changes in end-diastolic volume. This loss of compliance in the left ventricle may be the single most important difference between this population and the normals studied. In the normal population, an increase in venous return to the heart may stretch the myocardial muscle fibers enhancing end-diastolic volume and, thus, stroke volume. This will result in an improved cardiac output due to both increased heart rate and stroke volume. In the cardiac population, myocardial scarring may not allow stretching of myocardial fibers, thus no changes in end-diastolic volume or end-systolic volume. Therefore, during exercise conditions, cardiac output increases due to changes in heart rate with no apparent increase in stroke volume.

The single difference between the cardiac patients and normal subjects is the myocardial infarction. It can be assumed that under exercise conditions, sympathetic nervous system activity is the same for both groups. The mechanisms involved in mediating venous return are not influenced by the infarcted area. Redistribution of blood away from inactive tissues occurs followed by vеноconstriction and is assumed to be the same for both groups. The limiting factor between the groups does not appear to be neural. Vasodilatation of the active tissues also can be assumed to be the same. The limiting factor and the difference between the groups is how the myocardium (or, specifically the left ventricle) reacts to an increased venous return and the ability to concomitantly increase stroke volume and cardiac
output. It has been suggested that the loss of distensibility due to myocardial scarring occurs in the group having had a myocardial infarction. The results of this study support that theory. During upright rest both groups had identical end-diastolic volumes, end-systolic volumes, and stroke volumes. Under identical heart rate-related exercise conditions, only the normal group could increase end-diastolic volume enough to influence stroke volume and meet statistical significance.

To summarize, exercise in the upright position causes an increase in cardiac output in normal subjects by an increase in heart rate and stroke volume. The increase in stroke volume is due to an augmented end-diastolic volume with no change in end-systolic volume. Cardiac output during upright exercise in cardiac patients is a function of increased heart rate alone. Neither stroke volume nor end-diastolic volume changed in this group in response to upright exercise. Maximal heart rate values were not significantly different (p < .05) between the groups either at rest or exercise. If it is assumed that the only difference between the groups is the scarred left ventricle or a loss of ventricular compliance, even in the presence of an enhanced venous return in the upright position, may predispose this group to a cardiac output controlled only by changes in heart rate.
CHAPTER VI

SUMMARY AND CONCLUSIONS

The purpose of this study was to measure left ventricular volumes in two groups of men of comparable age using two-dimensional echocardiography. One group was classified by prior history as having normal cardiovascular status, the other group having had a previous myocardial infarction. End-diastolic and end-systolic volumes were measured at rest and immediately following an acute bout of exercise equivalent to 85% of maximal heart rate. These measurements and heart rate data were then utilized to calculate stroke volume, cardiac output and ejection fraction.

Maximal heart rate values were not statistically different (p < .05) for either group. In the normal group, end-systolic volume did not change. End-diastolic volume increased from rest to exercise conditions. Stroke volume increased significantly (p < .05) with exercise. Calculated ejection fraction also increased. In the cardiac group, neither end-diastolic volume nor end-systolic volume changed. Perhaps a loss of ventricular compliance to augment end-diastolic volume in this group results in no change in either stroke volume or ejection fraction.

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Conclusions

Within the limitations of this study, the following conclusions can be substantiated:

1. At rest in the upright position, there are no differences (p < .05) in heart rate, stroke volume, end-diastolic volume, end-systolic volume, cardiac output, or ejection fraction between apparently normal middle-aged males and their counterparts who have suffered myocardial infarction.

2. After a bout of acute upright exercise on the bicycle ergometer, there is a significant increase (p < .05) in cardiac output to meet the metabolic demands of the work task for both groups.

3. In the normal population, cardiac output is increased by both an elevation of the heart rate and increased stroke volume. Stroke volume is significantly increased (p < .05) in this population by a significant augmentation (p < .06) of the end-diastolic volume with no concomitant decrease in end-systolic volume.

4. In the cardiac patients, cardiac output is increased to meet metabolic demands of the work task primarily by an increased heart rate. Stroke volume did not change (p < .05) under exercise conditions in this population. No changes (p < .05) in end-diastolic volume or end-systolic volume were observed for this group.

5. Ejection fraction for the normal group increased (p < .05)
from rest to exercise conditions.

6. Ejection fraction did not change (p < .05) in the group of cardiac patients.

**Future Direction**

Future study in the area of rehabilitation after myocardial infarction may reveal central adaptation in response to chronic exercise training even in the presence of myocardial scarring. Intrinsic changes in cardiac performance of this group have not been documented. Perhaps as non-invasive procedures become even more sophisticated, myocardial adaptation and central circulatory changes can be documented in individuals who have had a myocardial infarction.


48. Mintz, G. S., M. N. Kotler, B. L. Segal, and W. Parry. A
comparison of two-dimensional and M-mode echocardiography in the evaluation of patients with infective endocarditis. Am.J.Cardiol, 43:738-744, 1979


APPENDIX A

DEFINITION OF SPECIFIC AND RELATED TERMS
Definition of Specific and Related Terms*

* Taken in part from:


A. Orifices and Valves of the Heart and Great Vessels

1. Atrioventricular orifices and valves — openings and cuspid valves between atria and ventricles.
   
   a. mitral valve, bicuspid valve — valve between left atrium and left ventricle.
   
   b. tricuspid valve — three endothelial folds or cusps which guard the right atrioventricular orifice.

2. Semilunar valves — half moon-shaped flaps within the aorta and pulmonary trunk which prevent the blood from flowing back into the ventricles.

B. Diagnostic Terms

1. Aneurysm — dilation or bulging out of the wall of the heart, aorta or any other artery.

2. Coronary atherosclerosis — chronic disorder characterized by the presence of lipid deposits which form fibrous plaques within the intima and inner media of the coronary arteries.

3. Coronary atherosclerotic heart disease — the most common heart condition. Progressive thickening of the intima of the coronary arteries leads to occlusion, caused by narrowing of the lumen and intravascular clotting.

4. Ischemic heart disease — cardiac disease in which the prominent feature is a markedly reduced blood supply to the heart muscle, generally due to coronary atherosclerosis.

5. Myocardial infarction, acute — clinical syndrome manifested by persistent, usually intense cardiac pain, unrelated to exertion and often constrictive in nature followed by diaphoresis, pallor, hypotension, dyspnea,
faintness, nausea and vomiting. The underlying disease is usually atherosclerosis which progresses to coronary thrombosis and occlusion and results in a sudden curtailment of blood supply to the heart muscle and myocardial ischemia.

a. inferior wall infarction, diaphragmatic infarction — infarction due to occlusion of the right coronary artery.

b. lateral wall infarction — infarction resulting from occlusion of diagonal branch of left anterior descending artery or of left circumflex.

c. posterior wall infarction — infarction precipitated by occlusive lesions of right coronary artery or circumflex coronary artery branch.

6. Pericarditis — inflammation of the covering membranes of the heart.

7. Valvular heart disease, chronic — disorder referring to any permanent organic deformity of one or more valves.

a. Aortic stenosis — reduction in the valve orifice interfering with the emptying of the left ventricle.

b. mitral stenosis — a very common sequela of rheumatic fever, marked by the development of minute vegetations and thrombi which narrow the orifice of the valve leaflets. Calcifications form as the disease progresses.

c. tricuspid disease — defect associated with mitral stenosis. It reduces the valve to a small triangular opening.

8. Valve replacement surgery — removal of incompetent or stenotic valve.

9. Valvotomy — incision into a valve.

a. mitral valvotomy — splitting the two commissures (areas of fusion) of the mitral valve to widen its opening.

b. pulmonary valvotomy — incising the valve of the
10. Ventricular aneurysmectomy — excision of aneurysm which may harbor large ventricular thrombi, decreasing the pumping efficiency of the heart.

11. Transducer, medical use — a transforming device which transmits energy from a patient to a monitoring machine.

12. Ischemia — reduced blood supply to an organ usually due to arterial narrowing or occlusion in advanced atherosclerosis.
   a. Cerebral ischemia — local anemia in the brain.
   b. Myocardial ischemia — inadequate blood supply to the heart muscle.

13. Murmur — blowing sound heard on auscultation.

14. Palpitation — subjective awareness of skipping, pounding or racing heart beats.

C. Angiocardiography

1. Peripheral angiocardiography — injection of contrast medium into the basilic or cephalic veins of the arm for examining the chambers of the heart and pulmonary circulation.

2. Selective angiocardiography — injection of contrast material through a catheter placed into the chamber or vessel of interest.

3. Selective retrograde aortography and left ventriculography — retrograde aortic catheterization for passing a radiopaque catheter across the aortic valve into the left ventricle. The correct position of the catheter is ascertained either by fluoroscopic or television guidance or radiogram. This method discloses ventricular septal defects, aneurysms, mitral and aortic regurgitation and other pathology.

4. Selective right ventricular angiography — visualization of the anatomic structures involved in tetrology of Fallot, transposition of the great
vessels, patent ductus arteriosus and similar conditions.

D. Terms Related to Diagnostic Ultrasound of Cardiovascular Disorders

1. Doppler ultrasonic flowmetry — ultrasound techniques for obtaining phasic, continuous and instantaneous measurement of velocity (speed) of blood flow in various cardiovascular disorders.

2. Echoaortography — ultrasonic study of the aortz.

3. Echocardiography — graphic recording of ultrasound waves reflected from the heart for the purposes of:
   a. studying the development of mitral stenosis from the onset of rheumatic heart disease.
   b. determining the severity of mitral stenosis.
   c. appraising the leaflet motion after mitral valvotomy.
   d. testing the functional capacity of the prosthetic valves following surgical valve replacement.
   e. reflecting the left ventricular outflow tract and aortic valve.
   f. delineating pericardial effusion and pericardial absence.
   g. aiding in the diagnosis of congenital and acquired heart disease.

E. Terms Related to Cardiac Catheterization and Coronary Artery Catheterization

1. Cardiac Catheterization — a procedure of diagnostic value in detecting various cardiac defects and diseases. A radiopaque cardiac catheter is inserted into an accessible vein and passed into the heart and pulmonary artery.

2. Cardiac Pressures — pressures created by the force of the contraction within the chambers of the heart upon the circulating blood.
APPENDIX B

HUMAN SUBJECT CONSENT FORM

Cardiac Rehabilitation Center
Swedish Covenant Hospital
5145 North California Avenue
Chicago, Illinois 60625
FORM OF INFORMED CONSENT FOR EXERCISE TESTING

I understand this test is being done to: (1) detect the possible presence of heart disease, (2) provide guidelines for prescription of an exercise program, (3) determine an appropriate plan of medical management to complete my recovery from my recent heart illness, or (4) this may be a re-test and re-evaluation after a period from a previous exercise test.

I hereby consent to voluntarily engage in an exercise test to determine the state of my heart and circulation. The information thus obtained will aid my physician(s) in advising me as to the activities in which I may engage.

The test which I will undergo will be performed by Dr. __________ and his staff. I will perform the test on an ergometer; the work levels will begin at a level which I can easily accomplish and will be advanced in stages, depending on my work capacity. I may ask to stop the test at any time because of fatigue or because of personal feelings of discomfort. I understand that I will not be exercised at a level which is abnormally uncomfortable for me. The supervising physician may also decide to stop the test because certain observations he may make may require or indicate the need to terminate the test.

Before I undergo the test, I will have an interview and will be examined by a physician to determine if I have a condition which
would indicate that I should not engage in this test.

During the performance of the test, a physician or trained observer will keep under surveillance my pulse, blood pressure and electrocardiogram. At the peak of exercise, collection of exhaled air may be collected for eventual calculation of my peak exercise oxygen consumption. Skinfold measurements to determine my percent body fat may also be done.

There exists the possibility of certain changes occurring during the exercise test. They include abnormal blood pressure, very rapid or very slow heart rate, and in rare instances, a heart attack. Every effort will be made to minimize them by the preliminary examination and constant surveillance during testing. Emergency equipment and trained personnel are available to deal with unusual situations which may arise, and these personnel have my consent to institute appropriate measures as needed.

The information which is obtained will be treated as privileged and confidential and will not be released or revealed to any person other than my physician without my expressed written consent. The information obtained, however, may be used for statistical or scientific purposes with my right of privacy retained.

I have read the foregoing and I understand it and consent to said testing. Any questions which may have occurred to me have been answered to my satisfaction.

Patient's signature ___________________________ Date ___________________________ Witness ___________________________

Patient's printed name ___________________________ Supervising physician ___________________________
CONSENT TO SPECIAL TREATMENT OR PROCEDURE

I, ____________________, hereby authorize to direct ____________ or associates or assistants of his choosing, to perform the following treatment or procedure and such additional services as they may deem reasonably necessary in its performance: a graded exercise test (stress test) performed on a bicycle ergometer with monitoring by electrocardiography (ECG) and echocardiography upon ____________________ (myself or name of subject).

The experimental (research) portion of the treatment or procedure is performed during a bout of exercise while in the upright position. This is a noninvasive procedure so no needles or intravenous devices will be used.

This is done as part of an investigation entitled: "Exercise Cross-Sectional Echocardiography in the Detection of Left Ventricular Dysfunction".

(1) Purpose of the procedure or treatment: (a) to detect the presence of heart disease, (b) to determine an appropriate plan of medical management for an existing heart illness, (c) to determine the function (or dysfunction) of the heart while under physical stress.

(2) Possible appropriate alternative methods of treatment include other procedures to evaluate heart function or not to participate in this study.

(3) Discomforts and risks reasonably to be expected: the patient will perform the test in a bicycle ergometer, the work stages beginning at a level which can easily be accomplished and will be advanced in stages, depending upon the working capacity of the subject (patient). The patient may ask to stop the test at any time because of fatigue, shortness of breath, chest pain or irregular heart rhythm (which may require electric shock) or because of personal feelings of discomfort. The patient will not be exercised at a level which is abnormally uncomfortable. The supervising physician may also decide to stop the test because certain observations he may make require or indicate the need to terminate the test. There exists a potential risk of abnormal responses to a graded exercise test including hypertension, hypotension,
bradycardia, tachycardia, and in rare instances, myocardial infarction. Every effort will be made to minimize them by a preliminary examination and constant surveillance during the test. Emergency equipment and trained personnel will be available to deal with unusual situations which may arise.

(4) Possible benefits for subjects/society: Exercise echocardiography is a safe, inexpensive investigative procedure that continuously images the heart during physical stress.

(5) Anticipated duration of subject's participation: The patient will be asked to visit the Cardiac Rehabilitation Center of Swedish Covenant Hospital, Chicago, Illinois on two separate occasions, each visit lasting approximately two hours. The first visit will include a complete physical examination by a physician including past medical history, resting electrocardiogram. A graded exercise test performed on the bicycle ergometer will be performed at this time without echocardiography to measure the patient's tolerance to the procedure. The second visit will include the exercise echocardiographic procedure.

I hereby acknowledge that has provided information about the procedure described above, about my rights as a subject, and he answered all questions to my satisfaction. I understand that I may contact him should I have additional questions. He has explained the risks described above and I understand them; he also offered to explain all possible risks or complications.

I understand that the information obtained from me, or from the person I am authorized to represent, will remain confidential unless I specifically agree otherwise by placing my initials here. I understand that, where appropriate, the U. S. Food and Drug Administration may inspect records of this research project.

I understand that I am free to withdraw my consent and participation in this project at any time after notifying the project director without prejudicing future care. No guarantee has been given to me concerning this treatment or procedure.

In the unlikely event of physical injury resulting from participating in this study, I understand that immediate medical treatment is available. Questions about this should be directed to the person named above. I also understand that the costs of such treatment will be at my expense and that financial compensation is not available.
I have read and fully understand the consent form. I sign it freely and voluntarily. A copy has been given to me.

DATE: ___________ TIME: ___________  AM

SIGNED: ___________________________  (subject)

______________________________  (person authorized to Consent for Subject - if Required)

WITNESSES: _______________________

______________________________

I certify that I personally completed all blanks in this form and explained them to the subject or his representative before requesting the subject or his representative to sign it.

SIGNED: ___________________________  (Project Director)
APPENDIX D

MEDICAL HISTORY AND PHYSICAL EXAMINATION FORM

Cardiac Rehabilitation Center
Swedish Covenant Hospital
5145 North California Avenue
Chicago, Illinois 60625
SWEDISH COVENANT HOSPITAL

MEDICAL HISTORY AND PHYSICAL EXAMINATION FORM

NAME: ________________________________

DATE: _______________ AGE: __________

I. GENERAL MEDICAL HISTORY

Any medical complaints? ____________________ YES NO

Any major illnesses in the past? (Give dates) YES NO

Any hospitalization? ________________________ YES NO

Smoke now? ___________ Packages per day ______ YES NO

Smoked in past? _______ Packages per day ______ YES NO

Weight gained in past ten years ______

Weight at age 20 _____ 30 _____ 40 _____ 50 _____

Diabetes? ________________________________ YES NO

Family history of diabetes? Who? ________ YES NO

Family history of heart disease? Who? ________ YES NO

Family history of high blood pressure? ________ YES NO

Who? __________________________________
Family history of muscular illness? _______ YES  NO
Who? ________________________________

2. CARDIO-RESPIRATORY HISTORY

Any heart disease now? YES  NO
Any heart disease in past? YES  NO
Heart murmurs? YES  NO
Occasional chest pains? YES  NO
Chest pains on exertion? YES  NO
Chest pressure on exertion? YES  NO
Fainting? YES  NO
Daily coughing? YES  NO
Coughs produces sputum? YES  NO
High Blood pressure? YES  NO
Shortness of breath at rest? YES  NO
Shortness of breath supine? YES  NO
Shortness of breath after two flights of stairs? YES  NO

3. MUSCULAR HISTORY

Any muscle injuries or illnesses now? YES  NO
Any muscle injuries or illnesses in the past? YES  NO
Muscular weakness now? YES  NO
Muscular illness now?  YES  NO
Muscle pain at rest?  YES  NO
Muscle pains at exertion?  YES  NO

4. BONE-JOINT HISTORY

Any bone or joint (including spine) injuries or illnesses now?  YES  NO
Any bone or joint (including spine) injuries or illnesses in past?  YES  NO
Ever had swollen joints?  YES  NO
Ever had painful joints?  YES  NO
Flat feet?  YES  NO
Athletics in past?  YES  NO
Specify: __________________________________________

_________________________________________________

5. LABORATORY EXAMINATION

Note: these tests are not required for this exam but should be recorded if the information is available. An SMAC 12/60 will be done as part of the regular evaluation.

Height ___________ Weight ___________ Waist girth _________
Vital capacity _______________ FEV 1.0 _________________
ECG rate _________________ Rhythm _________________
Interpretation __________________________________________

Blood hematocrit ________________ Hemoglobin ________________
White cell count ________________ Blood sugar ________________
Cholesterol ________________ Triglycerides ________________
Urinalysis specific gravity
Protein ________________ Sugar ________________
Chest X-ray
Interpretation

6. PHYSICAL EXAMINATION
Thyroid normal? YES NO
Chest auscultation abnormal? YES NO
Heart size normal? YES NO
Murmurs present? YES NO
Peripheral pulses absent? YES NO
Gallops, abnormal heart sounds? YES NO
Any joints abnormal? YES NO
Abnormal masses? YES NO
Hernias? YES NO

7. SUMMARY IMPRESSION OF PHYSICIAN
Comments of any history of physical finding: ________________

Diagnosis: ________________

I hereby release the above information to the Project Director.

SIGNED: ____________________________, M.D.
APPENDIX E

CONTRAINDICATIONS FOR EXERCISE TESTING
CONTRAINDICATIONS FOR EXERCISE TESTING

1. Contraindications

a. Acute myocardial infarction
b. Unstable or at-rest angina pectoris
c. Dangerous arrhythmias (ventricular tachycardia or any rhythm significantly compromising cardiac function)
d. History suggesting excessive medication effects (digitalis, diuretics, psychotropic agents)
e. Manifest circulatory insufficiency (congestive heart failure)
f. Severe aortic stenosis
g. Severe left ventricular outflow tract obstructive disease (HfSS)
h. Suspected or known dissecting aneurysm
i. Active or suspected myocarditis or cardiomyopathy (within the past year)
j. Thrombophlebitis - known or suspected
k. Recent embolism, systemic or pulmonary
l. Recent or active infectious episodes (including upper respiratory infections)
m. High dose of phenothiazine agents

2. Relative Contraindications

a. Uncontrolled or high-rate supraventricular arrhythmias
b. Repetitive or frequent ventricular activity
c. Untreated severe systemic or pulmonary hypertension
d. Ventricular aneurysm
e. Moderate aortic stenosis
f. Severe myocardial obstructive syndromes
g. Marked cardiac enlargement
h. Uncontrolled metabolic disease (diabetes, thyrotoxicosis, myxedema)
i. Toxemia or complications of pregnancy
3. Conditions Requiring Special Consideration and/or Precautions

a. Conduction disturbances
   1) Complete atrioventricular block
   2) Left bundle branch block
   3) Wolff-Parkinson-White anomaly or syndrome
   4) Lown-Ganong-Levine syndrome
   5) Bifascicular block (with or without 1st block)

b. Controlled arrhythmias

c. Fixed rate pacemaker

d. Mitral valve prolapse (click-murmur) syndrome

e. Angina pectoris and other manifestations of coronary insufficiency

f. Certain medications
   1) Digitalis, diuretics, psychotropic drugs
   2) Beta-blocking and drugs of related action
   3) Nitrates
   4) Antihypertensive drugs

g. Electrolyte disturbances

h. Clinically severe hypertension (diastolic above 110, grade III retinopathy)

i. Cyanotic heart disease

j. Intermittent or fixed right-to-left shunt

k. Severe anemia (hemoglobin below 10 gm/dl)

l. Marked obesity (20% above optimal body weight)

m. Renal, hepatic, and other metabolic insufficiency

n. Overt psychoneurotic disturbances requiring therapy

o. Neuromuscular, musculoskeletal, orthopedic or arthritic disorders which would prevent activity

p. Moderate to severe pulmonary disease

q. Intermittent claudication

r. Diabetes

*In the practice of medicine the benefits of evaluation often exceed the risks for patients with these relative contraindications*
APPENDIX F

CRITERIA FOR TERMINATION OF EXERCISE TEST
CRITERIA FOR TERMINATION OF EXERCISE TEST

Graded exercise tests should be stopped for the reasons listed. Physicians conducting graded exercise tests may use other criteria according to clinical considerations. The signs and symptoms of exercise intolerance presented are a guide to assist in a decision to terminate a graded exercise test.

1. Signs and symptoms of exertional intolerance
   a. Dizziness or near syncope
   b. Angina, regardless of the presence or absence of ECG abnormalities consistent with myocardial ischemia. (subjective angina ratings of the +3 level may be used if the exercising subject has been evaluated previously by a physician and the reliability of the subjective ratings has been established.)
   c. Nausea
   d. Marked dyspnea
   e. Unusual or severe fatigue
   f. Severe claudication or other pain
   g. Staggering or persistent unsteadiness
   h. Mental confusion
   i. Facial expression signifying severe distress
   j. Loss of sustained vigor of palpable pulse
   k. Cyanosis or severe palor
   l. Lack of rapid erythematous return of skin color after brief frim compression

2. Electrocardiographic changes
   a. ST-T segment horizontal or "divergent" displacement of 0.2 mV above or below the resting isoelectric line for at least 0.08 second duration after the junction ("J") point
   b. Ventricular arrhythmia
      1) Ventricular tachycardia (three or more successive ectopic ventricular complexes)
      2) Continuous bigeminal or trigeminal ectopic ventricular complexes
3) Frequent unifocal or multifocal ectopic ventricular complexes amounting to greater than 30% (trigeminy) of the total beats per minute

4) Due to the difficulty in differentiating between supraventricular and ventricular rhythms, unless well interpreted, supraventricular atrial complexes with aberrant ventricular conduction should be interpreted in the same way as ectopic ventricular beats

c. Atrial-ventricular or ventricular conduction disturbances
1) Second degree AV Block, Mobitz Type I (Wenckebach)
2) Second degree AV Block, Mobitz Type II
3) Third degree (complete) AV Block
4) Sudden left bundle branch block

3. Blood Pressure Responses. If systolic blood pressure (SBP) fails to rise with increasing exercise intensities (except as a result of familiarization in the early stages) or if SBP shows a drop of 10 mmHg or more, termination of the exercise test is usually indicated. An increase in SBP to the range of 250 mmHg or above is considered by some authorities as an indication for stopping exercise, but there is a lack of published reports indicating complications associated with high SBP during exercise. A diastolic rise of more than 20 mmHg or a rise above 110 to 120 mmHg is often considered an indication that the test should be terminated.

4. Heart Rate Response. Exercise heart rate will vary according to age, anxiety, disease medications, and functional capacity. Maximal heart rates for apparently healthy individuals 15 years and over may be estimated by either subtracting the participant's age from 220 (low estimate) or one half their ages from 210 (high estimate). Although these estimates may be used as a guide in test termination, they should not be used as predetermined termination points. It is important to remember that even for asymptomatic adults, the range of maximal heart rates for any one age is quite large (standard deviation: ±10 beats per minute) and that patients may have a much lower maximal rate. Symptom and sign limited tests are likely to be more useful. Once the maximal heart rate has been determined experimentally by a graded exercise test, it may usually be used effectively in exercise prescription.
5. Respiratory Responses. Marked dyspnea or cyanosis may be observed when individuals with pulmonary impairment exercise. Termination of the exercise test is recommended when the above symptoms or signs are observed.

6. Malfunctioning Equipment. In the event that there is an equipment malfunction or the ECG monitoring system fails to give an interpretable ECG, the test will be terminated and the problem corrected before proceeding with the graded exercise test.
APPENDIX G

EMERGENCY EQUIPMENT AND DRUGS
EMERGENCY EQUIPMENT AND DRUGS

1. Defibrillator - monitor with ECG electrodes - defibrillator paddles or portable DC defibrillator and portable ECG monitor
2. Airways - nasopharyngeal and oral (endotracheal)
3. Face mask and Robert Shaw valve
4. Oxygen
5. Suction apparatus
6. Syringes and needles
7. Intravenous sets
8. Intravenous stand
9. Adhesive tape
10. Laryngoscope
11. Drugs
   a. Sodium bicarbonate (IV)
   b. Catecholamine agents
      1) Epinephrine (IV)
      2) Isoproterenol (IV)
      3) Dobutamine (IV)
   c. Atropine sulfate
   d. Antiarrhythmic agents
      1) Lidocaine (IV)
      2) Procainamide (IV)
      3) Propranolol (IV/oral)
   e. Morphine sulfate
   f. Calcium chloride
   g. Vasoactive agent (Norepinephrine)
   h. Corticosteroids
      1) Methylprednisol
      2) Sodium Succinate
      3) Dexamethasome phosphate
   i. Digoxin (IV/oral)
   j. Lasix (IV)
   k. Dextrose 5% in water
   l. Nitroglycerine tablets
   m. Amyl nitrite pearls
APPENDIX H

PHYSICAL CHARACTERISTICS OF SUBJECTS
### PHYSICAL CHARACTERISTICS OF SUBJECTS

#### GROUP A

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (Kg)</th>
<th>Medications</th>
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<tbody>
<tr>
<td>TC</td>
<td>39</td>
<td>180.3</td>
<td>94.5</td>
<td>Timolol - 10 mg BID</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Coumadin - 7.5 mg daily</td>
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<td>42</td>
<td>175.0</td>
<td>92.7</td>
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<td>152.5</td>
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<td>86.4</td>
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<td>54</td>
<td>180.0</td>
<td>102.7</td>
<td>Aldochlor - 2 Tabs daily</td>
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<td>MB</td>
<td>54</td>
<td>150.0</td>
<td>85.0</td>
<td>Diazide - 25 mg daily</td>
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<td>57</td>
<td>165.0</td>
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<td>GB</td>
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<td>177.5</td>
<td>80.9</td>
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### Physical Characteristics of Subjects

#### Group B

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APPENDIX I

RAW DATA
RAW DATA FOR SUBJECTS IN GROUP A
RESTING VALUES

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## RAW DATA FOR SUBJECTS IN GROUP B

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END-DIASTOLIC VOLUME

ANOVA

TWO-FACTOR EXPT.
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

GROUP MEAN OF GROUP
GROUP (1, 1) 119.8889
GROUP (1, 2) 171.1111
GROUP (2, 1) 125.3846
GROUP (2, 2) 150.3077

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F4 (1, 20) = 0.5348
F6 (1, 20) = 34.6499 (p < .01)
F6 (1, 20) = 4.4714 (p < .05)
END-SYSTOLIC VOLUME

ANOVA

TWO-FACTOR EXPT.
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

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\[
\begin{align*}
F_a (1, 20) &= 2.6861 \\
F_b (1, 20) &= 0.1546 \\
F_{ab} (1, 20) &= 0.2828
\end{align*}
\]
STROKE VOLUME
ANOVA

TWO-FACTOR EXPT.
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

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\[ F_a (1, 20) = 2.5740 \]
\[ F_b (1, 20) = 43.4036 \quad (p < .01) \]
\[ F_{ab} (1, 20) = 6.4933 \quad (p < .05) \]
EJECTION FRACTION

ANOVA

TWO-FACTOR EXPT.
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

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\[
F_a (1, 20) = 4.6784 \quad (p < .05) \\
F_b (1, 20) = 16.1704 \quad (p < .01) \\
F_{ab} (1, 20) = 2.6516
\]
HEART RATE
ANOVA

TWO-FACTOR EXPERIMENT
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

GROUP   MEAN OF GROUP
GROUP (1, 1) 73.8889
GROUP (1, 2) 138.4444
GROUP (2, 1) 65.9231
GROUP (2, 2) 126.8482

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
</tr>
</thead>
<tbody>
<tr>
<td>BET.SUBJECTS</td>
<td>5406.5455</td>
<td>21</td>
<td>257.4545</td>
</tr>
<tr>
<td>A</td>
<td>916.3916</td>
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<td>916.3916</td>
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<tr>
<td>SUBJ.W.GRPS</td>
<td>4990.1538</td>
<td>20</td>
<td>249.5077</td>
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<td>W/I SUBJECTS</td>
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<td>42284.0000</td>
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<td>B x SUBJ.W.GRPS</td>
<td>2121.5726</td>
<td>20</td>
<td>106.0786</td>
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<tr>
<td>TOTAL</td>
<td>49830.5455</td>
<td>43</td>
<td></td>
</tr>
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</table>

F_A (1, 20) = 4.0818
F_B (1, 20) = 398.6100 (p < .01)
F_{AB} (1, 20) = 0.1737
CARDIAC OUTPUT

ANOVA

TWO-FACTOR CRV.
(1) REP. MEAS. ON ONE FACTOR
(2) UNEQUAL OR EQUAL GROUP SIZE

LEVELS OF FACTOR A = 2
LEVELS OF FACTOR B = 2

<table>
<thead>
<tr>
<th>GROUP</th>
<th>MEAN OF GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>GROUP (1, 1)</td>
<td>5.8356</td>
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<tr>
<td>GROUP (1, 2)</td>
<td>18.2611</td>
</tr>
<tr>
<td>GROUP (2, 1)</td>
<td>5.1900</td>
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<tr>
<td>GROUP (2, 2)</td>
<td>12.9454</td>
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<tr>
<td>B</td>
<td>1027.7278</td>
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<td>1027.7278</td>
</tr>
<tr>
<td>AB</td>
<td>57.9961</td>
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<td>57.9961</td>
</tr>
<tr>
<td>B x SUBJ.W.GRPS</td>
<td>128.9515</td>
<td>20</td>
<td>6.4476</td>
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<tr>
<td>TOTAL</td>
<td>1597.5155</td>
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<td></td>
</tr>
</tbody>
</table>

F_a (1, 20) = 6.5544 (p < .05)
F_b (1, 20) = 153.3976 (p < .01)
F_ab (1, 20) = 8.9350 (p < .01)
APPENDIX K

STATISTICAL ANALYSIS

Newman-Kuels Multiple Comparisons
NEWMAN-KUELS MULTIPLE COMPARISONS

In analyzing the data, the analyses of variance indicated significant interaction for end-diastolic volume, stroke volume, ejection fraction, and cardiac output which required multiple comparisons to locate the source of the observed variance. Newman-Kuels multiple comparisons were conducted on the individual cell means representing group interactions.

END-DIASTOLIC VOLUME (n = harmonic mean of cells for unequal n's).

<table>
<thead>
<tr>
<th>cell*</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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<tbody>
<tr>
<td>n</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
</tr>
<tr>
<td>(\bar{x})</td>
<td>125.38</td>
<td>150.31</td>
<td>119.89</td>
<td>171.11</td>
</tr>
</tbody>
</table>

* cell numbers represent Group A - rest (cell 1), Group A - exercise (cell 2), Group B - rest (cell 3), Group B - exercise (cell 4).
Table of Q -- End-Diastolic Volume

<table>
<thead>
<tr>
<th>cell</th>
<th>4</th>
<th>2</th>
<th>1</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>-</td>
<td>3.33</td>
<td>7.32*</td>
<td>4.86*</td>
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<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>2.37</td>
<td>4.87**</td>
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<tr>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.88</td>
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<tr>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
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</table>

* significant (p < .01)
** significant (p < .05)

STROKE VOLUME (n = harmonic mean of cells for unequal n's).

<table>
<thead>
<tr>
<th>cell*</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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</thead>
<tbody>
<tr>
<td>n</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
</tr>
<tr>
<td>(\bar{x})</td>
<td>77.85</td>
<td>102.85</td>
<td>79.00</td>
<td>133.00</td>
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</table>

* cell numbers represent the same cells as for end-diastolic volume.
### Table of Q -- End-Systolic Volume

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<thead>
<tr>
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<th>2</th>
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<th>4</th>
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</thead>
<tbody>
<tr>
<td>4</td>
<td>-</td>
<td>5.27**</td>
<td>5.51*</td>
<td>9.64*</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>4.17**</td>
<td>2.55</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.20</td>
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<tr>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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* significant \( p < .01 \)
** significant \( p < .05 \)

### Ejection Fraction (n = harmonic mean of cells for unequal n's)

<table>
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<tr>
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<tbody>
<tr>
<td>n</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
</tr>
<tr>
<td>( \bar{x} )</td>
<td>62.08</td>
<td>67.23</td>
<td>66.11</td>
<td>77.67</td>
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* cell numbers represent the same cells as for end-diastolic volume.
### Table of Q -- Ejection Fraction

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<tbody>
<tr>
<td>4</td>
<td>-</td>
<td>5.28**</td>
<td>3.43**</td>
<td>7.89*</td>
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<tr>
<td>2</td>
<td>-</td>
<td>-</td>
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<td>1.53</td>
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</table>

* significant (p < .01)
** significant (p < .05)

### CARDIAC OUTPUT (n = harmonic mean of cells for unequal n's)

<table>
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<tr>
<th>cell*</th>
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<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
<td>10.53</td>
</tr>
<tr>
<td>$\bar{x}$</td>
<td>5.19</td>
<td>12.95</td>
<td>5.84</td>
<td>18.26</td>
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</table>

* cell numbers represent the same cells as for end-diastolic volume.
Table of Q -- Cardiac Output

<table>
<thead>
<tr>
<th>cell</th>
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<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>-</td>
<td>6.79*</td>
<td>10.62*</td>
<td>16.70*</td>
</tr>
<tr>
<td>2</td>
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<td>-</td>
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<td>6.63*</td>
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<tr>
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<td>-</td>
<td>-</td>
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</tr>
<tr>
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<td>-</td>
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<td>-</td>
<td>-</td>
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</table>

* significant (p < .01)