Reproducibility of a Continuous-Wave Doppler Ultrasound System for Assessment of Ascending Aortic Blood Flow Responses During Graged Exercise Testing with Healthy Individuals

by

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(ABSTRACT)

Continuous-wave (CW) Doppler recordings of ascending aortic maximal blood flow acceleration (PkA), maximal velocity (PkV) and systolic velocity integral (SVI) were taken at each stage of a graded exercise treadmill test on two separate days with 30 physically active adult males. Signals were measured (Quinton Exerdop) for all cardiac cycles in the 3rd minute of each stage using a hand-held probe positioned at the suprasternal notch. A dedicated microcomputer, programmed to select "valid" beats on the basis of value consistency in the sample set, determined the acceptability of signals. No significant differences were found between the three trial means within each stage on either day for PkA, PkV or SVI. Significant ($p < .01$) intraclass reliability estimates ranged from $r = 0.89$ to 0.97 (PkA), $r = 0.90$ to 0.98 (PkV) and $r = 0.85$ to 0.95 (SVI). Coefficients of variation were calculated at

each stage to estimate the relative consistency of each measure. A gradual reduction of the coefficient of variation was observed for each blood flow measure between stages one and four. The test-retest (between days) reliability coefficients for PkA, PkV and SVI for stages one to four ranged between r = 0.51 to 0.78 (P <.004), but correlations for the pre-exercise baseline and stages five and six were lower. These results indicate that (1) PkA, PkV and SVI demonstrate greater measurement stability within each stage of a graded exercise test than is the case between separate days of measurement at the same stage; and (2) there is modest day-to-day response stability for clinical testing with the Doppler parameter of PkV. Reliability/stability was best in exercise stages which encompass the speed and grade range of 45.0 $m \cdot min^{-1}$/10% - 111.7 $m \cdot min^{-1}$/14%, i.e., those in which all subjects can walk.

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CHAPTER I

Introduction

The primary disease associated with Western Society's high standard of living is coronary heart disease (CHD). Kannel (1981) reported that CHD affects 650,000 individuals each year and is the leading cause of death in men over 40 years of age and women over 55. The related health care cost accounts for approximately 10% of the annual Gross National Product. Of those men who die from heart attacks, it is estimated that one-half have clinical manifestations of CHD prior to death which potentially might have been diagnosed.

Various techniques have been developed to diagnose disease or predict the likelihood of a future heart attack. Bennett, Else, Miller, Sutton, Miller and Noble (1974) suggested that cardiac output and intracardiac pressure do not accurately predict myocardial dysfunction. Huntsman, Stewart, Barnes, Franklin, Colocousis and Hassel (1983) estimated that 25% of myocardial infarctions (MI) were not recognizable by standard clinical evaluation, and about half of the MI's have abnormal cardiac output. Many such tests, including radionuclide studies and coronary angiography, are relatively expensive and have a remote potential of being harmful to the patient (Elykayam,

Gardin, Berkley, Hughes & Henry, 1983). Equipping a laboratory for radionuclide angiography, in particular, often requires a large capital outlay, and the serial studies required in the procedure necessitate repeated injections of radionuclear material. It would thus seem important to develop alternative means for non-invasively measuring an individual's myocardial status to detect latent or evolving CHD which are less expensive and risky to the patient. The use of advanced technology in Doppler ultrasonics, supported by use of microprocessors, has made such applications feasible in association with exercise stress testing.

Statement of the Problem

One factor which has prognostic importance relative to forecasting cardiac mortality is the left ventricular ejection fraction of the heart. Stein and Sabbah (1976a) reported that the ejection fraction was a function of the tension, radius, speed of shortening and acceleration of cardiac fiber shortening. They stated that their studies on ventricular power allowed the development of formulas that characterize ventricular performance which are virtually free of assumptions. In their studies, Stein and Sabbah (1976b) compared healthy and coronary diseased subjects; diseased subjects were defined as those individuals with an ejection fraction less than 50% and

circumferential fiber shortening of less than 1.0 $m \cdot sec^{-1}$. They reported that ejection fraction was advantageous in evaluating ventricular performance as it could clearly distinguish between the two groups in terms of important diagnostic and prognostic criteria. In a third paper from that year, Stein and Sabbah (1976c) reported that their two previous articles had allowed them to derive a ventricular function formula based on the physiological characteristics of the myocardium. With this formula, they were able to determine left ventricular function (LVF) at peak tension which was termed the ejection rate of change of power at peak tension. They reported that this indicator would be able to distinguish between normal and non-normal individuals based on the rate and flow changes in the blood; their hypothesis was that this indicator would accurately reflect the pressure generated by the ventricle wall prior to ejection. Similarly, Bennett et al. (1974), reported that the occlusion of a coronary artery may reduce maximum acceleration of blood, even when there may be no significant changes observed in cardiac output or intracardiac pressure. Thus, knowledge of a patient's power characteristics associated with ejection fraction would appear to be of central importance in the diagnosis of CAD during clinical stress testing.

The "gold standard" used for determining the ejection fraction has been invasive ventriculography performed with

cardiac catheterization, dye solution, and x-ray imaging procedures. With this technique, a catheter with an electromagnetic transducer tip is inserted into the ventricle to measure maximum acceleration and peak velocity of blood flow. More recently, ejection fraction has also been measured by using a continuous wave (CW) Doppler system. Chandnarata, Silveira and Aronow (1980) reported that the CW Doppler was sensitive enough to measure blood flow indicators of LVF. The importance of knowing exactly which variable to measure was emphasized in a study by Lambert, Nichols and Pepine (1982). They analyzed 24 variables in an attempt to identify the most sensitive index of ventricular function. Their results indicated that Stein and Sabbah's (1976c) "ejection rate of change of power at peak tension" was the most promising variable for predicting intracardiac peak wall tension, independent of pre- and afterload.

Once the change of ejection in volume variable to be assessed had been determined, the dilemma was to determine which aspect of ventricular function provided the most accurate index. According to Stein and Sabbah (1976b,c), this is best achieved by assessing the combination of ventricular pressure and its derivitives within the systolic ejection period. Gardin, Burn, Childs and Henry (1984) compared the blood flow from the pulmonary artery and the aorta. They reported that data relative to blood flow

changes in the ascending aorta provided the most relevant indicator of ventricular wall tension, and that, at this site, peak acceleration was more reliable as an indicator than was average velocity. Reporting on findings which had a most important bearing on interpretation of peak aortic blood flow acceleration data in exercise, Daley, Sagar and Wann (1985) found that during maximal exercise, the aortic dimensions change little relative to resting conditions; this supports the assumption that changes in blood flow characteristics measured at the ascending aorta are not biased by transient changes in the vessel lumen. Consequently, the speed at which the blood flows is closely related to the rate at which it is ejected from the heart.

Huntsman et al. (1983) and Elkayam et al. (1983) sought diagnostic procedures that would be non-invasive and relatively inexpensive in their studies to test the effects of vasodilatory drugs, different pacemaker modes, and cardiotoxic drugs used in treating cancer. They reported that the change in aortic blood flow was an effective means of distinguishing between healthy patients and those with limited ventricular function. Sabbah, Khaja, Brymer, McFarland, Albert, Snyder, Goldstein and Stein (1986) used a CW Doppler (Quinton Exerdop) to collect data on subjects undergoing cardiac catheterization. They reported that peak acceleration was the most favorable indicator of left ventricular

function when this variable was compared against the patient's ejection fraction (r = 0.90). Sabbah et al. (1986) reported comparative results for maximal aortic blood acceleration using a CW Doppler and found that this measure was a useful indicator of overall LVF.

Significance of the Study

There is a growing body of evidence which supports the use of a CW Doppler as a means of non-invasively measuring LVF. Avenues now need to be explored which examine the application of this new "basic research tool" as a means for assessing LVF in the exercise laboratory. Most published studies to date using this technique have dealt with the assessment of ejection fraction at rest. By assessing LVF during exercise, additional information could potentially be obtained by the investigator. For example, one could compare the relative contribution of increased myocardial contractility versus ventricular pre-load to cardiac output under a variety of exercise conditions.

However, before one is able to investigate such questions, one must determine the reliability of the instrument under the laboratory conditions in which it is to be operated. Specifically, it is important to determine the measurement stability of testing exercising subjects with this instrument. Also, cardiologists need to have a particular level of assurance that such responses, if valid, will actually

represent a technically stable functional measure. Finally, from a research perspective, there is a question of this instrument's response stability when compared to that for other physiological responses typically monitored during exercise testing.

Research Hypotheses

The purpose of the study is reflected in the following hypothesis:

1. HO: There is no internal consistency for the Doppler variables of peak acceleration (PkA), peak velocity (PkV), or the systolic velocity integral (SVI) during selected time intervals within each exercise stage of a graded exercise treadmill test.

2. HO: There is no test-retest consistency for the variables of peak acceleration (PkA), peak velocity (PkV) and systolic velocity integral (SVI) during selected time intervals within each exercise stage of a graded exercise treadmill test.

Delimitations

1. The subjects were physically-active young adult males from the Virginia Tech community for whom maximum exercise was not medically contraindicated.

2. Measurements made during the treadmill test were taken with the Quinton Exerdop during the third minute of each stage of graduated exercise stress test.

3. Each subject was oriented to the treadmill during two submaximal trials on different days in order to achieve his proper understanding of the exercise expectations.

4. Each individual was instructed to exercise to the point of fatigue.

5. Verification of maximal exertion was done with reference to plateauing of the measured and normative referenced response comparisons for peak oxygen consumption, heart rate and rating of percieved exertion.

Limitations

1. At this time, the data will only identify the reliability of using a CW Doppler on healthy individuals.

2. The reliability of this instrument is limited to the third minute of each stage.

3. Despite the fact that the subjects are oriented to the treadmill, there may be some extraneous gait influence due to the nature of running on the treadmill (Charteris and Tavais, 1976).

4. Each subject may not have the same perception of a "pain threshold," thus each subject may not reach a true maximum point of fatigue.

5. The experimenter had no way on insuring that there were identical physiologic responses for the subjects prior to each test.

6. There may be variance in the accuracy of the Exerdop measurements, oxygen consumption ($\dot{V}O_2$) max and heart rate (HR) measurements due to the inaccuracy of the technicians.

Basic Assumptions

Prior to the investigation, the following assumptions were made pertaining to the collection of the data:

1. The subjects would comply to the requests of experimenter, including refraining from strenuous activity prior to the test and completing the test to maximal exertion;

2. The techniques of measurement (CW Doppler, $\dot{V}O_2$max, heart rate, blood pressure and skinfolds) are valid;

3. The subjects were free of coronary artery or other diseases which would affect the CW Doppler responses.

Definitions and Symbols

1. Borg Rating of Percieved Exertion (RPE)- a commonly used scale (6 to 20) developed to quanitify an individual's overall feelings of effort during exercise testing.

2. Bruce Protocol- the protocol to be used during the graded exercise testing.

3. Continuous Wave (CW) Doppler- a type of Doppler ultrasound that continually emits ultrasound waves from the transducer and receives reflected sound waves at the same transducer, simultaneously.

4. Ejection Fraction- the amount of blood that is ejected from the left ventricle during systole, expressed as a percentage of the total end-diastolic ventricular volume.

5. Exerdop- the manufacturer's product name of the CW Doppler (Quinton Instruments, an A. H. Robbins Company subsidiary).

6. Peak Acceleration (PkA)- the first derivitive of Peak Velocity, which is actually the highest instantaneous slope of the blood flow being ejected into the ascending aorta in each cardiac cycle (Gardin, 1984).

7. Peak Velocity (PkV)-the peak speed at which blood is ejected into the ascending aorta during systole (Gardin, 1984).

8. Stroke Velocity Integral (SVI)- the integral of the velocity x time response for blood flow in the ascending aorta during systole, identified as "stroke distance" by Quinton Instruments (Gardin, 1984).

CHAPTER II

Continuous-wave (CW) Doppler echocardiography has recently been utilized as an non-invasive instrument for determining the force generating capabilities of the left ventricle. The validity of this instrument for detecting aortic blood flow indicators of ventricular power has been demonstrated with invasive techniques in animal models. As a result of these investigations, opportunities have arisen with the Doppler that may permit this instrument to have important medical and research applications in assessment of left ventricular function during exercise stress tests.

Physiological Studies

Rushmer (1962) noted that at rest the cardiac output for coronary heart disease (CHD) patients was often the same as that of individuals who were healthy. He sought an instrument that would accurately discriminate between such individuals and could measure the cardiac reserve during maximal cardiac output. He hypothesized that by measuring the impulse of the blood within the cardiac cycle (caused by the force of contraction), he could determine the acceleration to establish the presence or absence of functionally significant cardiac dysfunction. Rushmer reported that the greatest aortic blood flow velocity is due

to the contraction (myocardial shortening), and the impulse is represented by the pressure exerted on the load in the ventricle. He stated that the dynamic characteristics of the ventricular ejection are greatly altered by autonomic control and simulated diseases. Using dogs, he developed a model showing the relationship of aortic blood flow acceleration due to contraction, but this needed to be refined because of species differences.

Noble, Trechard and Gus (1966) also used dogs to examine maximum aortic acceleration as an index of the myocardial contractile state. They placed an electromagnetic flowmeter around the base of the aortic to measure the impulse of the blood. Noble, Trechard and Gus isolated arteries on the myocardium and restricted blood flow to the left ventricle. By temporarily producing regional myocardial ischemia in this manner, they were able to induce decreases in maximal acceleration generated by contraction of the left ventricle. Their results suggested a maximum acceleration which was closely related to changes induced on the left ventricular muscle, but is insensitive to left ventricular endiastolic volume. Changes in maximum acceleration occurred prior to any other changes, and they determined this to be the most sensitive index of the myocardium. Based on these observations, they expressed the view that measures of maximal blood flow acceleration would be valuable in cardiac diagnosis. They hypothesized that

"defective" myocardial tissue would be easily detectable to assess if non-invasive technology could be developed.

Similarly, Nutter, Noble and Hurst (1970) noted that in dogs, the peak aortic blood flow and acceleration were sensitive measures regardless of the inotropic or contractile state. They also noted that both measures were sensitive to alterations in the load and inotropic state. Finally, they reported that the ratio of acceleration to peak flow may be useful as and indices which reflects the contractile state of the myocardium.

Light (1969) reported that a transcutaneous ultrasonic Doppler system could possibly noninvasively determine the blood velocity in the ascending aorta. By using a diverging ultrasonic beam, he hypothesized that one could quantify the peak velocity and peak acceleration of blood flow because the frequency change in returning signal should provide a close relationship to the blood's mainstream velocity.

Jewitt, Gabe, Mills, Maurer, Thomas and Shillingford (1974) reported the comparison of the blood flow values obtained by placing a flow transducer into the ascending aorta of 24 coronary artery disease (CAD) patients. They measured both peak velocity and peak acceleration. Jewitt et al. reported that peak acceleration was the most sensitive of the two variables. They suggested that this assessment would be valuable in the prognosis of patients with CAD. They noted that patients with peak blood flow

velocities measured below 400 $cm \cdot sec^{-1}$ and a maximum acceleration below 700 $cm \cdot sec^{-2}$ did not have a high survival rate. They also concluded that this would be of assistance in evaluating the post-operative assessment of CAD surgery patients should there be a suitable transcutaneous technique developed.

Bennett, Else, Miller, Sutton, Miller and Noble (1974) evaluated 12 CAD patients using a catheter-tip for changes in peak velocity and acceleration. They reported those individuals with a high functional status had a peak velocity of 1500 $cm \cdot s^{-2}$ and a peak acceleration of 60 cm•s, while those with moderate disease had PkV of 995-1100 $cm \cdot s^{-2}$ and a PkA of 32-58 cm•s, and those with a poor ejection fraction had a PkV below 850 $cm \cdot s^{-2}$ and a PkA below 41 cm•s. Bennett *et al*. reported that one can accurately determine the zero point of peak acceleration because the blood flow returns to zero, but that it is more difficult to determine the PkV. With PkV, one assumes that it is zero at the end of diastole, however, there may be some residual blood flow that confounds the starting point such as is found in individuals with CHD or a prolapsed valve.

Stein and Sabbah (1976a,b) suggested that blood flow data relative to ventricular ejection was clearly valuable in assessing functional status of the myocardium. Using dogs, they measured the aortic and left ventricular pressures using a catheter-tip probe, while aortic flow was

measured by a magnetic flow transducer placed around the root of the aorta. They noted that the change in power of the contracting myocardium produces power that is related to the length, tension and velocity of the shortening of myocardial tissues. The experimenters both augmented and decreased the contractility of the myocardial tissue and measured the responses. They reported that the peak rate of change was linearly related to the peak rate of change of the aortic flow ($r = 0.99$) for both experimental conditions and were not affected by drugs, preload or afterload. No other indices showed a similar response. They reported that the internal chamber pressure the contracting muscle produces during systole was related to LaPlace's law regarding circumferencial fiber length and the velocity of fiber shortening. This was possible because tension related to the Laplace equation and circumference fiber length and velocity related to flow produced an exact relationship which depended upon the shape of the venticle (which was assumed to be spherical). This evaluation can then be applied to a cardiac patient for determining PkA or PkV assuming a flat profile and and a constant cross-sectional area are utilized when assessing the instantaneous rate of change of power.

In their later paper, Stein and Sabbah (1976b) evaluated the rate of change of power during ejection in 22 patients during a cardiac catheterization. They used a

catheter-tip velocity sensor at the root of the aorta to distinguish between a normal and abnormal ejection rate based upon the differences of the blood flow into the aorta. They assumed the profile to be flat and the cross-sectional area constant. Stein and Sabbah reported that those individuals with a normal ejection fraction had a mean value of 25 x 10^8 dyne cm, while those with abnormal ejection fractions had mean values of 11 x 10^8 dyne cm. They concluded that the peak ejection rate of change of power correlated well with the mean velocity of circumferential fiber shortening ($r = 0.86$), while the correlation between peak ejection rate of change of power and ejection fraction was $r = 0.71$.

Stein and Sabbah (1976b) noted that the isovolumic pressure in the ventricle was related to the tension in the wall (LaPlace's Law). They deducted that in order to determine the effectiveness of the pump, it was essential to evaluate the ventricular performance prior to ejection. They examined several indices. In their earlier dog study, they reported the rate of change of flow was the only ejection index that was responsive to changes in the myocardial contractile state. In their latter study, they hypothesized that the rate of change of power indicates acceleration of energy expended on production of useful work by the ventricle during ejection. Stein and Sabbah (1976b) derived a formula based on the tension, radius,

acceleration of shortening and rate of shortening; assuming a spherical shaped ventricle. They concluded that the rate of change of ventricular power, tied in with the dog study, showed that it reflected alterations in the inotropic state, which was relatively independent of preload or afterload.

In a third paper from that year, Stein and Sabbah (1976c) noted that by deriving their formula for the rate of change of power at peak tension, they were able to reduce the characteristics of the myocardial contraction to a rather non- complex summary if one assumed that the ventricle was to be a thin-walled spherical. The ejection rate of change could then be calculated based on the rate of shortening of the contractile element, tension, fiber length and relative acceleration of circumfrencial fiber shortening. They suggested that this could be of benefit in diagnostic procedures because it could distinguish between a normal and abnormal heart based upon the velocity of the contractile element. This, in turn, results in a characteristic ejection rate of change at peak tension of the ventricle which can be calculated by measuring the blood flow and its changes.

Lambert, Nichols and Pepine (1982) attempted determine a parameter which would accurately reflect the myocardial contractile state. When examining 24 parameters, they reported that the ejection rate of power

at peak tension to be the most sensitive index independent of the pre- and afterload.

Principles of Ultrasonics

Doppler instrumentation is based upon the premise estabilished by Christian Johan Doppler more than 100 years ago. Doppler ultrasound emits sound waves to detect changes in blood flow characteristics. The sound waves are produced at a rate based upon the frequency of the transmission, generally between two and ten mega Hertz (MHz).

At the present time, there are several diagnostic tools that emply ultrasound as a means of assessing cardiac function. The 2-D and M-mode echocardiograms function in a manner similar to sonar in that they provide a visual means of assessing the heart structure. Specifically, these instruments allow imaging which documents the dimensions and movements of the valves and function of the heart walls. Doppler ultrasound emits sound waves that are classified in two categories. Pulse gated Dopplers emit waves a selected distance from the probe. They are received back to the probe at intermittent cycles. By determing the timing of the signals being returned, the instrument is able to determine where the plaque is in the artery due to an increased turbulence which causes the signal to be dispersed over a wider area.

The sound waves in a continuous wave (CW) Doppler are produced by a crystal to be transmitted through a resonating substance (Sumner, 1983). The probe is placed on the skin with an acoustic gel that is applied to the tip of the probe to enhance signal conductance. The sound waves travel toward the aorta. The amount of interference that impedes the progress of the sound wave is called attenuation. This is found to be quite high in the lungs because air does not transmit the waves very well, and in bone because of the high concentration of calcium. Calcium is also quite prevalent in atherosclerosis, so Doppler ultrasound may not be quite as reliable in those patients with advanced aortic atherosclerotic disease (Sumner, 1983). However, CW Dopplers are of use for diagnostic purposes in that they allow the overall flow disturances to be readily assessed (Come, 1986).

Once the sound wave reaches the intended object, in this case the red blood cell, the sound wave is reflected at various angles. This disperison is known as "Rayleigh scattering" (Hill, 1978). Waves that are reflected back to the probe are picked up by a receiver crystal, also located in the tip of the transducer. The velocity of the blood is determined by the difference in the rate of frequency of the original sound beam and the sound wave that is received by the probe after it has bounced off the blood cell.

The rate at which the blood is travelling is determined by measuring the systolic interval proceeding through the aorta (Gardin, 1984) describes the peak flow velocity at the midpoint of maximum systolic blood flow. Peak acceleration was determined from the onset of ejection, while the total ejection time was measured from the onset of ejection to the end of the systolic ejection.

Validity of Doppler

The Doppler, by emitting an ultrasonic beam, is capable of determining the velocity of a flow stream, it's first derivitive: acceleration, and the integral of the 'velocity' x time curve for systole.

Chandraratra, Silveira and Aronow (1980) questioned the potential of CW Doppler placed transcutaneously for generating accurate data relative to LVF. They correlated acceleration measured invasively with that measured with the acceleration measured non-invasively using a CW Doppler. They reported a moderately high correlation, $r = 0.83$, and concluded that the Doppler was a useful non-invasive method for measuring PkA as it relates to the detection of impaired left ventricular performance.

Huntsman, Stewart, Barnes, Franklin, Colocousis and Hessel (1983) emphasized the need for developing an accurate instrument for non-invasively assessing cardiac function. They noted that 25% of the M.I.'s are unrecognizable with

the present diagnostic criteria which physicians currently have available. Huntsman et al. suggested that when cardiac output declines, so then does the prognosis for CHD patients. They wanted an ultrasound instrument that would use a flat profile to measure the highest blood flow velocity. The flat profile is required because the velocity of blood that measures the highest velocity. If the profile is skewed, the result will be an overestimation of flow. Huntsman et al. reported an moderately high correlation, $r = 0.83$, between the Doppler and the thermodilution (TDCO) technique. They suggested Doppler to be an excellent tool for measuring the therapeutic intervention of vasodilatory drugs or a pacemaker.

Bennett, Barclay, Davis, Mannering and Mehta (1984) also echoed the concerns of Huntsman for developing an accurate assessment for LVF because the alternative methods of echocardiography and nuclear imaging and coronary angiography were expensive. They reported a correlation of $r = 0.91$ between Doppler and TDCO. Bennett et al. concluded that the non-invasive measure of the ascending aorta could provide useful monitoring of the inotropic state and Starling mechanism.

With the technical advancement of the Doppler system which occured in the early 1980's, there was the need to determine in which anatomical position the beam should actually be directed to determine the most accurate

assessment of blood flow. Gardin, Burn, Childs and Henry (1984) compared several places for determining the blood flow. When comparing the ascending aorta, aortic arch, and proximal descending aorta for peak flow velocity, they determined the ascending aorta to be the area most indicative of the myocardial contractile state.

Ihlem, Myhre, Amlie, Forfang and Larsen (1985), in conducting similar research on 20 CAD patients, measured the stroke volume (SV) simultaneously using Doppler transcutaneously and TDCO intraveneously. The SV was calculated at the aortic orifice. Ihlem et al. found the aortic orifice to be the most ideal for assessing the blood flow because the profile is flat, which was one of Huntsman's concerns for determining an accurate blood flow. Ten patients were given an injection of dobutaime to induce rapid changes in the stroke volume. Doppler had a close correlation with TDCO ($r = 0.92$), suggesting that Doppler could reliably detect changes in SV.

Stein, Sabbah, Albert and Snyder (1985) summed up the concerns expressed by several other investigators by conducting their own validity studies with the Doppler system. They simultaneously measured compared the maximum aortic blood acceleration with electromagnetic flow transducer placed around the root of the aorta to a Doppler placed transcutaneously 16 open-chested dogs. The linear correlation coefficients for maximum velocity and the

maximum acceleration determined by the two methods were r = 0.95 and r = 0.96, respectively.

Daley, Sagar and Wann (1985) suggested that the Doppler may be useful for the non-invasive detection of exercise induced left ventricular dysfunction. They reported that when exercising 10 males to maximum using the Bruce protocol, neither the ventricular stroke volume, nor peak aortic blood flow changed from significantly different between the upright and supine positions (PkV: 0.91 to 1.36 $m \cdot s^{-1}$ supine and 0.75 to 1.39 $m \cdot s^{-1}$ upright, SV: 54 to 63.5 $ml \cdot m^{-2}$ supine and 38 to 63.3 $ml \cdot m^{-2}$ upright). They hypothesized that the changes in gravity probably enhanced myocardial contractility and the Frank-Starling (F-S) mechanism, leading to an increased SV during exercise. It is the inability of one of these two mechanisms to meet the increased cardiac demand during exercise that leads to impaired function. Daley, Sagar and Wann concluded that the measurement of PkV and PkA using a Doppler were similar to those found using an electromagnetic flow meter. However, limitations exist in determining the SV because of inaccuracies in determining the aortic diameter, and their may be problems in older populations with aortic stenosis.

Experimental Uses of CW Doppler in Medical Diagnosis and Treatment

As mentioned earlier, this instrument was hypothesized

to be useful in assessing the efficacy of vasodilator therapy. Elkayam, Gardin, Berkley, Hughes and Henry (1983) also agreed with this position as it was a going to be opposed to the M-mode echocardiography and radionuclide studies (effectiveness and cost). Echocardiography is limited in determining cardiac output because it can only measure changes as the cube root of ventricular volumes which limits assessment of vasodialator therapy. Radionuclide treatment, aside from requiring a special building, also subjects the patient to repeated infusions of radioactive material In their study, Elkayam et al. cited the high reliability that was noted with dog research using a Doppler to measure peak flow. Their study was aimed at determining the usefulness of this instrument in distinguishing between those 13 CHD individuals who had received 18 vasodilator drug interventions and the controls. Elkayam et al. hypothesized that with drug intervention one could alter the systemic vascular resistance (SVR), and improve the ability of the left ventricle to empty. In their findings, they reported a good correlation existed between peak flow velocity and the three Doppler measures. SVR had an inverse relationship ($r=-0.89$) with peak aortic blood flow during therapy. The Doppler aortic flow velocity integral and SV were showed a moderately high correlation ($r=0.88$).

Vijayaragharan, Singham, Tei, Wong, Wong & Shah (1984) noted several problems in using the Doppler in older subjects. They reported that there were anatomic changes, an increase in atherosclerosis, development of systemic hypertension and emphysema that all either produced changes in the aorta or limited clear access to the vessel by the ultrasonic beam, thus making accurate assessment difficult. However, the concerns about accuracy in the presence of CAD were soon reduced by the continued success of several authors who used this instrument for diagnostic purposes.

Khaja, Sabbah, Brymer, Albert, Goldstein & Stein (1986a,b) reported that one could assess global left ventricular performance before, during and immediately after blood occlusion of the coronary artery during coronary angioplasty using a CW Doppler (EXERDOP). By non-invasively measuring peak blood flow acceleration, they was able to identify global left ventricular dysfunction caused by ischemia. At the same time, Khaja et al reported, that in a small sample of CHD subjects, he was able to distinguish between myocardial dysfunction accompanied by chest pain and those individuals in whom coronary occlusion does not influence regional function due to preexistance of collateral coronary circulation.

There seems to be some question as to which of the flow parameters measured by CW Doppler (Exerdop) actually provides the most useful information in determining cardiac

dysfunction. Harrison, Smith, Friedman, Kwan and DeMaria (1986) compared the blood flow velocity values between patients with who had ischemia or had infarcted. They reported that there was a wide variability in the response of this method and that the velocity changes due to exercise do not allow the detection of myocardial ischemia. Kelly, Rothbart, Partone, Moore, Watson and Gibson (1986), in contrast noticed that older individuals have lower PkV values, both at rest and exercise, but he was able to determine that there was a decreased velocity during exercise which appears to distinguish between a true and false positive GXT.

The most successful diagnostic results appear to be with those researchers who have used the peak acceleration (PkA) as the means for determining left ventricular function (LVF). Sabbah, Khaja, Brymer, McFarland, Albert, Snyder, Goldstein and Stein (1986) noted that PkA was used to differentiate between patients with normal and abnormal LVF. Mehta, Bennett, Mannering, Dawkins and Ward (1986) reported that when patients were measured post-MI using a Doppler, the finding of a combination of an attenuated Doppler monitored hemodynamic response and early ischemia was able to predict those individuals who had three vessel coronary disease. In a later paper, Mehta, Bennett, Dawkins, Ward and Mannering (1986) all three Doppler ejection values (PkA, PkV, SVI) were found to be lower in those

individuals with a positive ischemic response. The maximum acceleration signal was lower in those patients with three-vessel CAD than those with one- and two- vessel CAD. They noted that when predicting three-vessel coronary disease, using PkA and PkV values resulted in a predictive accuracy of 65%. By comparison, the onset of ST depression (ECG) was 74% predictive and the combination of the two yielded a predictive accuracy of 80%. He suggested that the Doppler may be a useful adjunct test during a routine exercise stress test in identifying high mortality risk patients in the setting of an M.I.

Similarly, Sabbah, Przybylski, Albert and Stein (1987) used Evan's blue dye to assess the percentage of induced ischemic mass in dogs who had had their coronary arteries partially occluded. They reported a correlation of $r = 0.88$ for percentage of ischemic mass at risk and percentage of change in the PkA measured in the aorta by the Doppler. Other correlations were $r = 0.84$ for the percent change in the ejection fraction and the Doppler PkA response, $r = 0.77$ for the percent change in velocity and $r = 0.17$ for the percent change in the stroke volume. They concluded that among the various global indices of left ventricular performance that have been used to non-invasively, acceleration correlated most closely with the extent of left ventricular ischemia mass at risk.

Summary

Rushmer and other pioneering investigators in cardiac physiology saw the practicality for employing an instrument capable of non-invasively assessing myocardial contractile properties. The establishment of Doppler responses as valid indicators of LVF in animal models and in recent human research studies has provided such an opportunity. Initial testing seems to suggest the merit of such an instrument in the medical field; however, future reseach must focus on the measurement stability of this technology in the non-invasive clinic, as well as with the capacity of these instruments to with discriminate between healthy individuals and those with varying degrees of CAD. Before this instrument can be used for further medical and research applications under exercise stress conditions, it is critical that measurement stability be demonstrated with the most common mode for clinical exercise evaluation, i.e., graded exercise testing with the treadmill.

CHAPTER III

JOURNAL MANUSCRIPT

Reproduciblity of a Continuous Wave Doppler Ultrasound for Assessment of Ascending Aortic Blood Flow Responses During Graded Exercise Testing

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(abbreviated running head)

Reproducibility of a CW Doppler during Graded Exercise Testing

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(ABSTRACT)

Continuous-wave (CW) Doppler recordings of ascending aortic maximal blood flow acceleration (PkA), maximal velocity (PkV) and systolic velocity integral (SVI) were taken at each stage of a graded exercise treadmill test on two separate days with 30 physically active adult males. Signals were measured (Quinton Exerdop) for all cardiac cycles in the 3rd minute of each stage using a hand-held probe positioned at the suprasternal notch. A dedicated microcomputer, programmed to select "valid" beats on the basis of value consistency in the sample set, determined the acceptability of signals. No significant differences were found between the three trial means within each stage on either day for PkA, PkV or SVI. Significant ($p < .01$) intraclass reliability estimates ranged from $r = 0.89$ to 0.97 (PkA), $r = 0.90$ to 0.98 (PkV) and $r = 0.85$ to 0.95 (SVI). Coefficients of variation were calculated at each stage to estimate the relative consistency of each measure. A gradual reduction of the coefficient of variation was observed for each blood flow measure between stages one and four. The test-retest (between days) reliability coefficients for PkA, PkV and SVI for stages one to four ranged between $r = 0.51$ to 0.78 ($P < .004$), but correlations for the pre-exercise baseline and stages five and six were lower. These results indicate that (1) PkA, PkV and SVI demonstrate greater measurement stability

within each stage of a graded exercise test than is the case between separate days of measurement at the same stage; and (2) there is modest day-to-day response stability for clinical testing with the Doppler parameter of PkV. Reliability/stability was best in exercise stages which encompass the speed and grade range of 45.0 $m \cdot min^{-1}$/10% - 111.7 $m \cdot min^{-1}$/14%, i.e., those in which all subjects can walk.

Introduction

Various techniques have been developed to diagnose disease or predict the likelihood of a future heart attack. One factor which has prognostic importance relative to forecasting cardiac mortality is impairment of left ventricular ejection fraction (EF) of the heart. Bennett, E.D., Else, W., Miller, G.A.H., Sutton, G.C., Miller, H.C., & Noble, M.I.M. (1974) reported that occlusion of a coronary artery may reduce maximum acceleration of blood; however, there may be no significant change observed in cardiac output or intracardiac pressure. Stein and Sabbah (1976a,b,c) derived a formula that characterizes ventricular performance. Stein and Sabbah (1976b) determined that comparisons of left ventricular function (LVF) at peak tension would allow them to distinguish between healthy and coronary diseased patients (diseased subjects were those with EF < 50% and circumferential fiber shortening of less than 1.0 $m \cdot sec^{-1}$) based upon the rate and flow changes in the blood.

Previously, ejection fraction and its power derivatives have been determined by invasive ventriculography (Noble, 1966). Ejection fraction has also been measured by using a continuous wave (CW) Doppler system. Chandnarata, Silveira and Aronow (1980) reported that the CW Doppler was sensitive enough to measure blood flow indicators of LVF. The importance of knowing exactly which variable to measure

was determined by Lambert, Nichols and Pepine (1982). They analyzed 24 variables in an attempt to identify the most sensitive index of ventricular function. Their results indicated that Stein and Sabbah's (1976c) peak rate of change of ejection at peak tension, or blood flow acceleration, was the most promising variable for predicting intracardiac peak wall tension.

Once the change of ejection in volume variable to be assessed had been determined, the dilemma was to determine which aspect of ventricular function provided the most quantitatively accurate index. Gardin, Burn, Childs and Henry (1984) compared blood flow from the pulmonary artery and the aorta. They reported that data relative to blood flow changes in the aorta provided the most specific indicator of ventricular wall tension, and that, at this site, peak acceleration was more reliable as an indicator than average velocity. This finding was corroborated by the findings of Daley, Sagar and Wann (1985) who reported that during maximal exercise, aortic dimensions change little relative to resting conditions, thus supporting the assumption that changes in flow characteristics measured at this site were not biased by vessel lumen changes. Consequently, the speed at which the blood flows in the aorta is closely related to the rate at which it is ejected from the heart.

Sabbah, Khaja, Brymer, McFarland, Albert, Snyder, Goldstein and Stein (1986) using a CW Doppler (Quinton Exerdop) to collect data on subjects at rest, reported that peak acceleration was the most favorable indicator left ventricular function. They (1986) reported comparable results for maximal aortic blood flow velocity and acceleration using a CW Doppler compared to an electromagnetic flow transducer; the correlations were r = 0.96 and r = 0.95, respectively. However, despite the advances in determining the validity of a CW Doppler, we have been unable to document any studies which have determined the stability/reliability of these measurements within a graded exercise test protocol or between repeated graded exercise tests (GXT) given on separate days.

Thus, the purpose of this study was to determine if the Quinton Exerdop would provide reproducible measures within each stage of an exercise stress test and between successive test administrations with the same subjects.

Methodology

Duplicate trials (separate days) were conducted on a group of healthy male subjects (19-43 years of age) who were accustomed to regular vigorous exercise. During the first 2 minutes of each stage, blood pressure, heart rate

(HR), maximal oxygen consumption ($\dot{V}O_2$max), and rating of perceived exertion (RPE) were measured. Blood pressure was determined according to the Krokoroff sounds used in exercise evaluations (ACSM, 1986). Heart rate was determined by counting the R to R intervals, as these subjects had normal sinus rhythms. $\dot{V}O_2$max was measured using gas analysis of expired air. RPE was measured using the Borg Rating of Perceived Exertion on the 6 to 20 scale for exercise. In each trial, LVF measurements were taken after two minutes of standing rest and then, throughout a series of progressively increasing speed/grade stages (final minute of each three minute stage) in a clinical treadmill test, i.e., the Bruce protocol. During the third minute of the stage, the Exerdop (Quinton Instruments, Seattle, WA.) probe was placed above the suprasternal notch and aimed toward the ascending aorta prior to the beginning of the final minute of each stage. This enabled the technician to determine which position of the probe produced the sound most likely to record a "valid" signal. Thus, the signal to be used was determined before the final minute of each stage began. The technician pressed a remote foot pedal that triggered the instrument to start recording measurements. At 20 and 40 seconds into the minute, the experimenter would quickly lift his foot off the pedal and put it back down again. This would create a data set for each 20 second interval of the third minute of the stage.

If it did not appear that the individual was going to complete the third minute of the stage, such as was found at the higher stages, then the experimenter attempted to record a complete minute data during the final stage. Also, during the last minute of each stage, the heart rate was measured during the 20th, 40th and 50th seconds of the stage so as to provide heart rates that corresponded to the Exerdop measurements that were being made. Each subject continued to exercise until he had reached his functional capacity ($\dot{V}O_2max$) (Appendix C).

Results

PkA, PkV, and SVI Responses

The physiological characteristics for all subjects are presented in Table 1. The group mean EXERDOP responses and related standard deviations for test two are depicted in Figures 1-3.

Intraclass reliability

The intraclass correlation for PkV during test one was above R = 0.81 ($p < .01$) at all stages, and greater than R = 0.90 at all stages for test two (Table 2). For SVI, the intraclass correlation was above R = 0.84 during test one and R = 0.89 or greater during test two (Table 3). The intraclass reliability for PkA was above R = 0.79 ($p < .01$) for all stages during test one, while in test two,

values above R = 0.89 (p <.01) were found for all stages (Table 4).

Test-Retest Reliability

For Doppler all variables, there were statistically significant correlations (r = 0.50 to 0.78, p <.01) from stage one through stage four (Figures 4-6). However, there were no significant associations between response at the pre-exercise stage or after the fourth stage for any variable. The correlational values for PkV plateaued earlier during the test than did PkA or SVI.

Discussion

For the results presented in Tables 2-4, it was found that that were statistically significant intraclass correlational values for all variables during every exercise stage. The lowest correlations for any measurement occurred during the 6th stage of exercise. This may be attributed to the fact that the testor had difficulty maintaining probe contact on the suprasternal notch while the subject was running, introducing mechanical artifact, thus reducing the number of "valid" beats and affecting the testors ability to maintain the proper angle of interrogation for Doppler measurement in the ascending aortic blood flow. Generally, the highest correlational values for all variables were found during the middle stages

(stages two to four) of the GXT. There may be several reasons for this. The technician was able to maintain good probe placement during the walking stages. Also, the measurements were taken for 20 second intervals which were separated only by a one second delay.

In terms of day-to-day response stability, the results presented in Figures 4-6 showed only modest correlations from stage one to stage four for each Doppler variable. Reproducility coefficients were especially low during the pre-exercise stage or after the fourth stage. Also, between the fourth and fifth stages, there was a decline in the coefficient from non-determination was observed from 60% to 1.5%. The lack of a statistically significant correlation during the pre-exercise stage may be due to fluctuations of HR, LVF and vasoconstriction as these are influenced by catacholamines (Ihlen, 1985). The decrease in the correlation coefficient after the fourth stage may be due to substantial within subject physiological variation on a day-to-day basis, i.e., lack of uniform condidtions for sleep, diet, pre-test physical activity, arousal and habituation to the protocol. As shown in Table 5 and Figure 7, there were considerable fluctuations in each of the physiological variables measured during the GXT. In a study using graded exercise testing, Moore (1987) reported that there were low to modest correlation coefficients for physiological variables that

were measured on seperate days (HR, r = 0.50-0.86, $\dot{V}O_2$, r = 0.35-0.79, and SBP, r = 0.18- 0.77). Thus, although there does not appear to be an overwhelming stability/reliability, the values obtained by EXERDOP appear to be consistent with other commonly used indices such as heart rate, blood pressure and oxygen consumption measured during a GXT when testing has been done under conditions typical of those that prevail in the clinical laboratory.

An important point is that these tests were conducted with a modified Bruce protocol (Appendix C) on healthy individuals. Using a Bruce protocol, Kelly (1986) reported that he was able to distinguish between healthy and abnormal (multivessel CAD) individuals using a CW Doppler during graded exercise testing. The most stable results from this study occurred when the individuals were able to maintain a moderate walking pace on the treadmill. Those stages in which stable measurements were found may not necessarily be the same stages which are now gaining acceptance as part of the clinical protocol for physician diagnosis of latent ischemic heart disease. Individuals with advanced CAD tend to have a lower ejection response Kelly, (1986), so these data suggest that cardiologists might improve test sensitivity by actually tracking stage-by-stage PkA or PkV changes instead of just examining maximal exercise versus resting responses. The stages that may produce the most consistent measures may

occur during the middle stages of a lower intensity test, i.e. the Naughton protocol (ACSM, 1986).

The data from this study suggest that EXERDOP does produce statistically significant reproducibilty during repeated GXTs on seperate days. The correlations are similar to other physiological measurements taken during the same tests (Moore, 1987). Given the instability observed for several of these important diagnostic variables, it would seem more appropriate to combine several physiological variables to provide a non-invasive tool for assessing an individual's LVF.

In conclusion, several questions remain unanswered and several questions have arisen from these results. Standard scores for different aged males and females need to be developed for the Doppler variables that would be applicable to individuals with- or without symptoms of CHD. Also, there needs to be a determination of the amount of attenuation of the Doppler signal that is due to atherosclerotic conditions present with increased calcium deposits. This is of concern because calcium is known to attentuate the Doppler signal (Sumner, 1983). Answers to these and other questions will ultimately define the diagnostic limitations of this non-invasive instrument during graded exercise testing.

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Index of Terms for Physical Characteristics of 30 Subjects

Height = subject's height in cm

Body Fat = estimation of total body fat (Jackson & Pollock, 1978)

$\dot{V}O_2$ = oxygen consumption in liter•min^{-1}

R = respiratory quotient

HR = heart rate

Table 1. Mean Physiological Responses Of 30 Subjects.

	Height (cm)	Body Fat %	$\dot{V}O_2$ ($ml \cdot kg^{-1}$)	R	HR ($b \cdot min^{-1}$)
$\bar{x}$ =	178.00	15.43	48.25	1.29	193.00
SD =	±7.12	±5.58	±7.25	±0.11	±10.92

Table 2. Intraclass correlations (R) and Coefficients of Variation (CV) for Peak Velocity During Graded Exercise Testing for 30 Subjects

Stage	Test		Retest	
	R	CV	R	CV
0	0.93	0.23	0.90	0.21
1	0.94	0.20	0.95	0.24
2	0.94	0.16	0.98	0.22
3	0.94	0.17	0.96	0.18
4	0.94	0.17	0.93	0.16
5	0.93	0.21	0.96	0.22
6	0.81	0.15	0.96	0.24

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$/10% grade
2 = 66.7 $m \cdot min^{-1}$/12% grade
3 = 90.0 $m \cdot min^{-1}$/14% grade
4 = 111.7 $m \cdot min^{-1}$/16% grade
5 = 133.3 $m \cdot min^{-1}$/18% grade
6 = 146.7 $m \cdot min^{-1}$/20% grade

Table 3. Intraclass Correlations (R) and Coefficients of Variation (CV) for Systolic Velocity Integral During Graded Exercise Testing for 30 Subjects

	Test		Retest	
Stage	R	CV	R	CV
0	0.88	0.26	0.94	0.28
1	0.86	0.25	0.89	0.23
2	0.85	0.18	0.93	0.22
3	0.93	0.18	0.89	0.19
4	0.95	0.19	0.92	0.21
5	0.85	0.21	0.93	0.25
6	0.84	0.21	0.91	0.26

Stage:
0 = standing rest
1 = 45.0 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 4. Intraclass Correlations (R) and Coefficients of Variation (CV) for Peak Acceleration During Graded Exercise Testing for 30 Subjects

Stage	Test		Retest	
	R	CV	R	CV
0	0.93	0.38	0.94	0.22
1	0.91	0.37	0.89	0.36
2	0.93	0.32	0.93	0.36
3	0.93	0.28	0.97	0.38
4	0.91	0.25	0.96	0.27
5	0.91	0.24	0.94	0.28
6	0.79	0.18	0.90	0.26

Stage:
0 = standing rest
1 = 45.0 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 5. Means ($\bar{x}$) and Standard Deviations (SD) for Physiological Responses of 30 Subjects at Each Stage of a Graded Exercise Test

Stage		$\dot{V}O_2$	R	HR	SBP
0	$\bar{x}$	-	-	76.82	120.74
	SD			±11.84	±12.72
1	$\bar{x}$	14.68	0.80	97.25	135.15
	SD	±2.32	±0.10	±12.00	±14.49
2	$\bar{x}$	19.82	0.85	113.07	145.16
	SD	±2.78	±0.06	±12.86	±14.85
3	$\bar{x}$	26.74	0.95	135.63	158.65
	SD	±3.12	±0.08	±15.04	±14.55
4	$\bar{x}$	34.34	1.07	162.14	172.53
	SD	±7.23	±0.11	±25.93	±14.41
5	$\bar{x}$	43.85	1.20	183.58	186.57
	SD	±5.26	±0.12	±13.87	±14.18
6	$\bar{x}$	47.59	1.24	189.83	197.80
	SD	±9.42	±0.12	±10.83	±15.56

Stage:
0 = standing rest
1 = 45.0 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

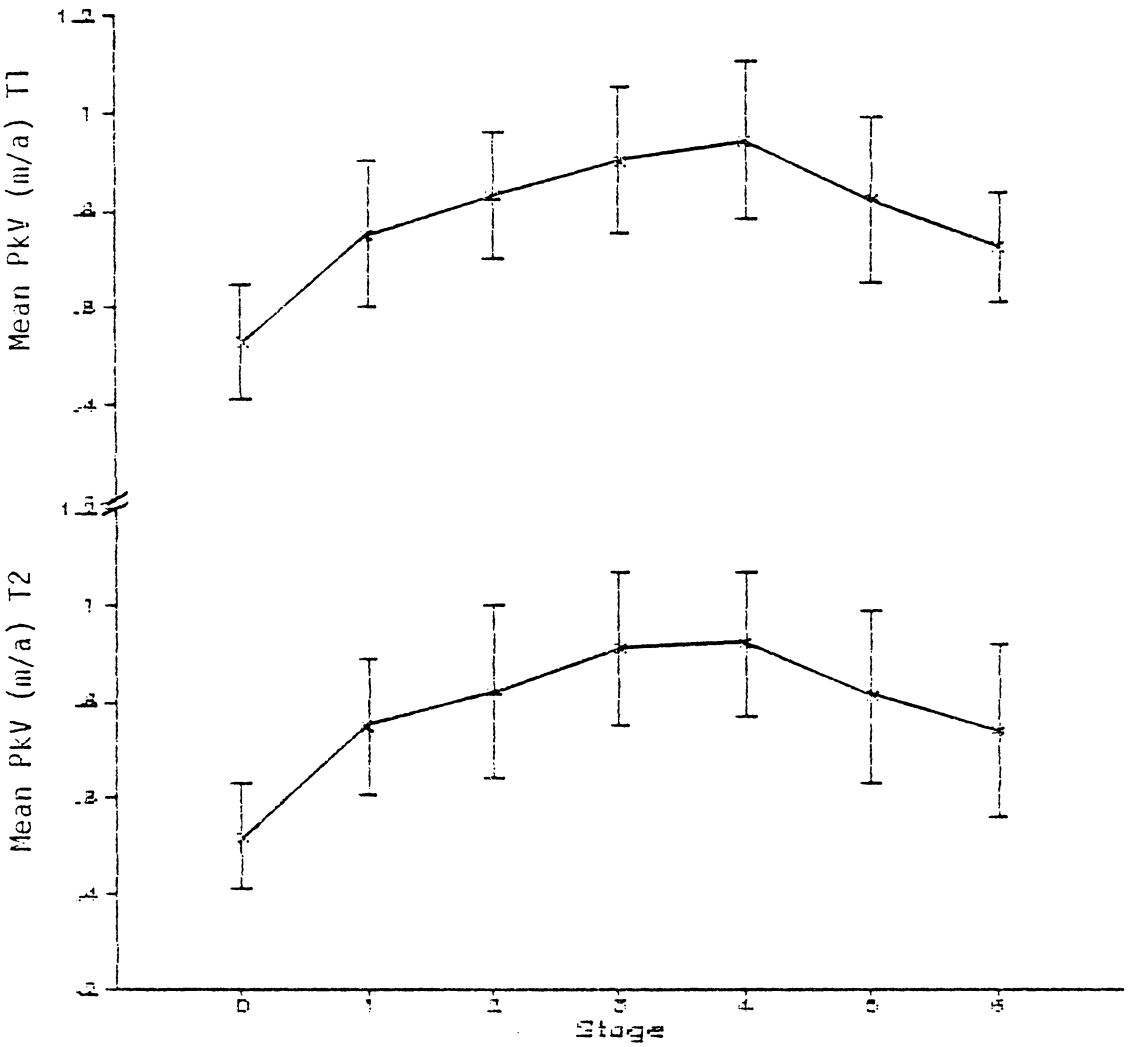

Figure 1. Means and Standard Deviations of PkV during GXTs.

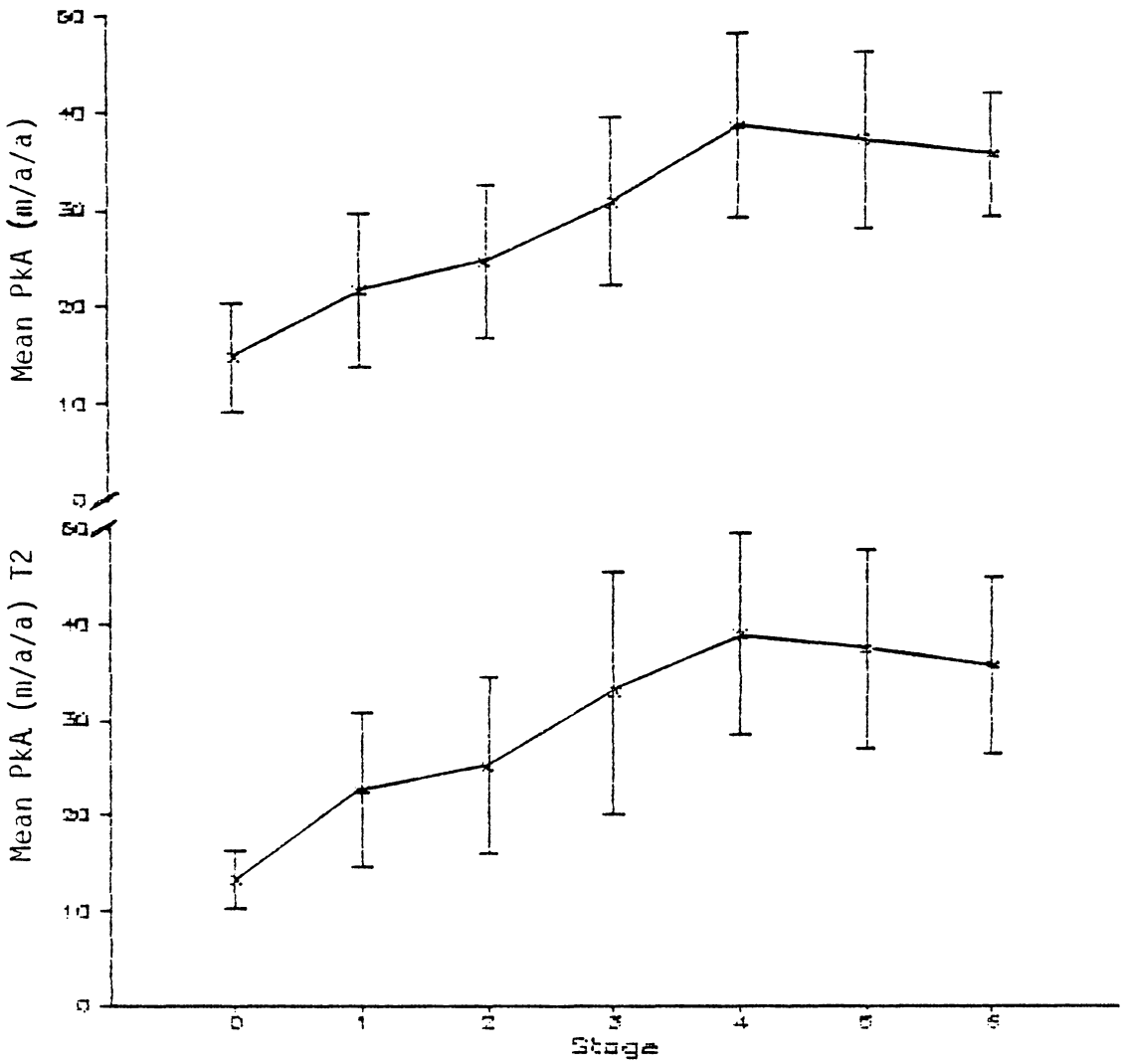

Figure 2. Means and Standard Deviations of PkA during GXTs.

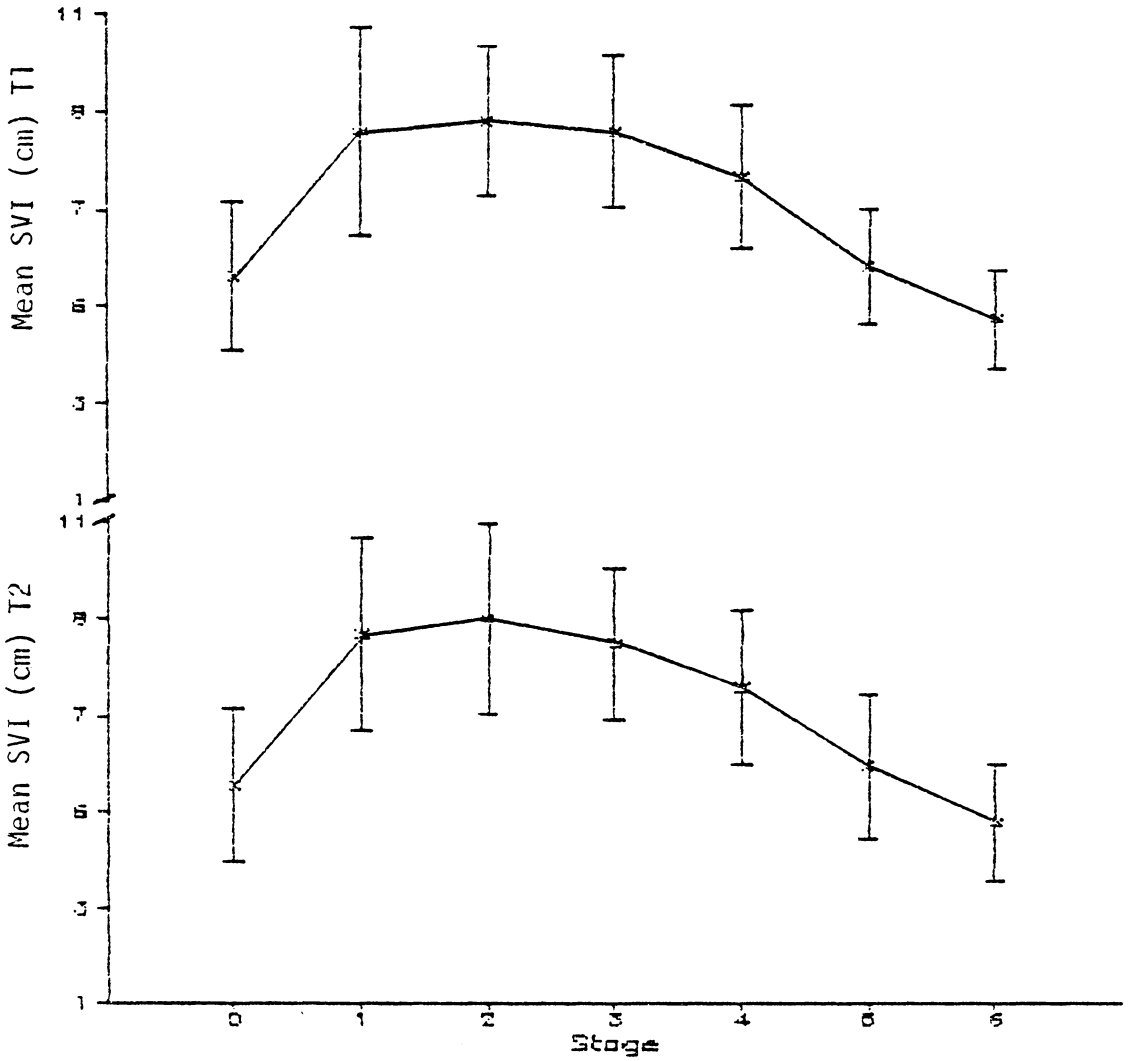

Figure 3. Means and Standard Deviations of SVI during GXTs.

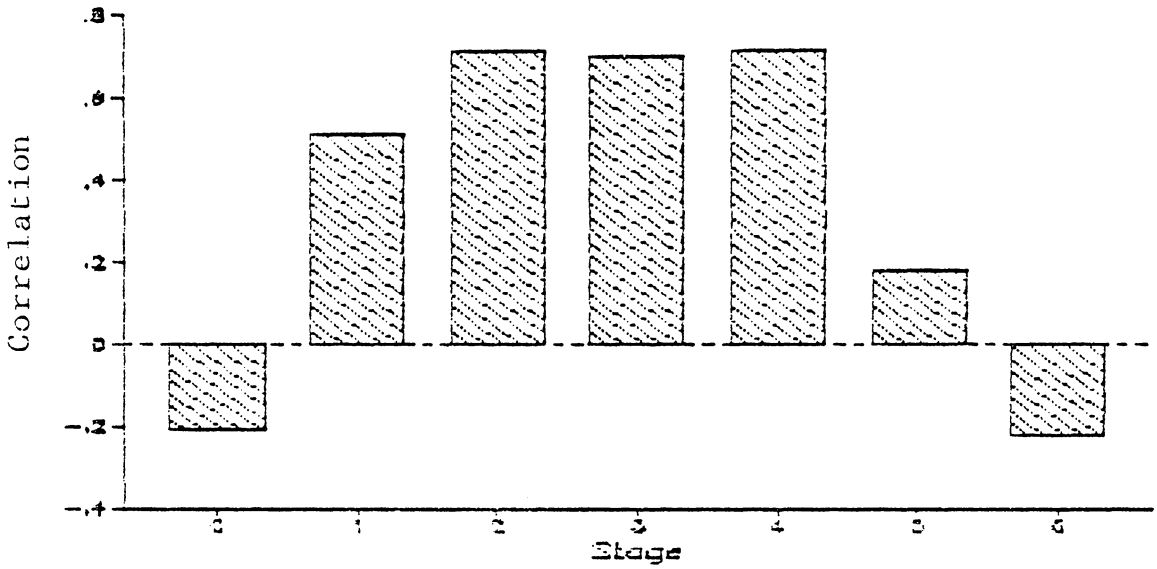

Figure 4. Correlations for test-retest reliability for PkV.

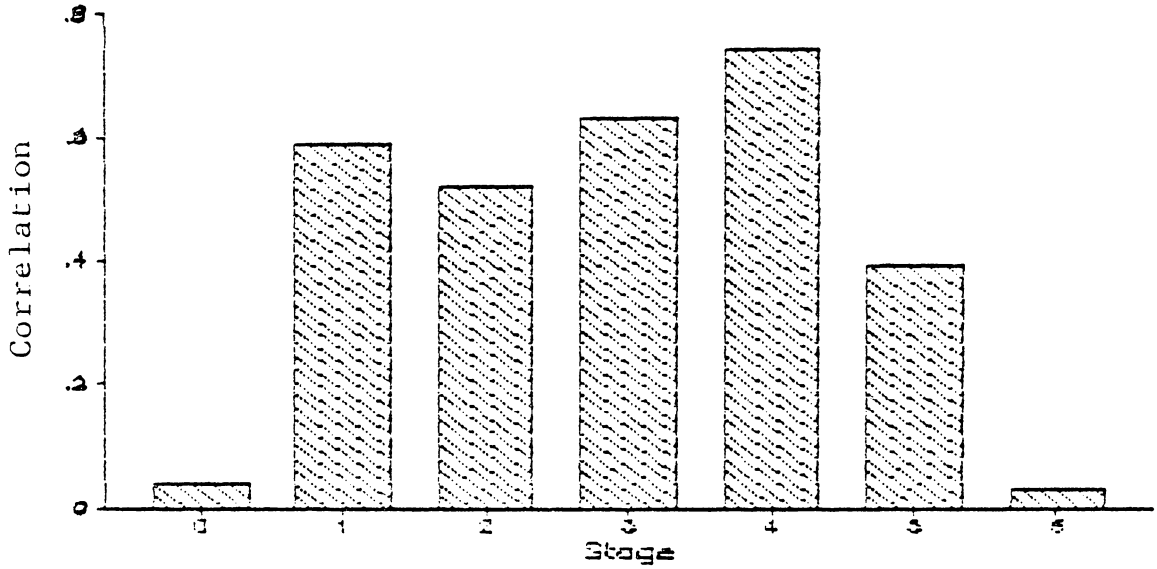

Figure 5. Correlations for test-retest reliability for PkA.

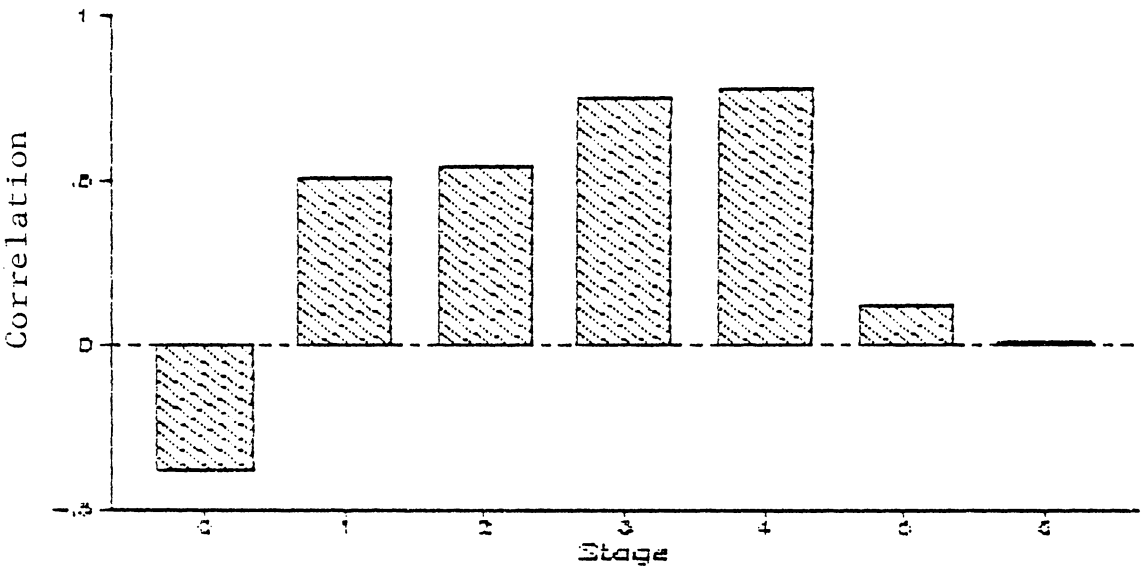

Figure 6. Correlations for test-retest reliability for SVI.

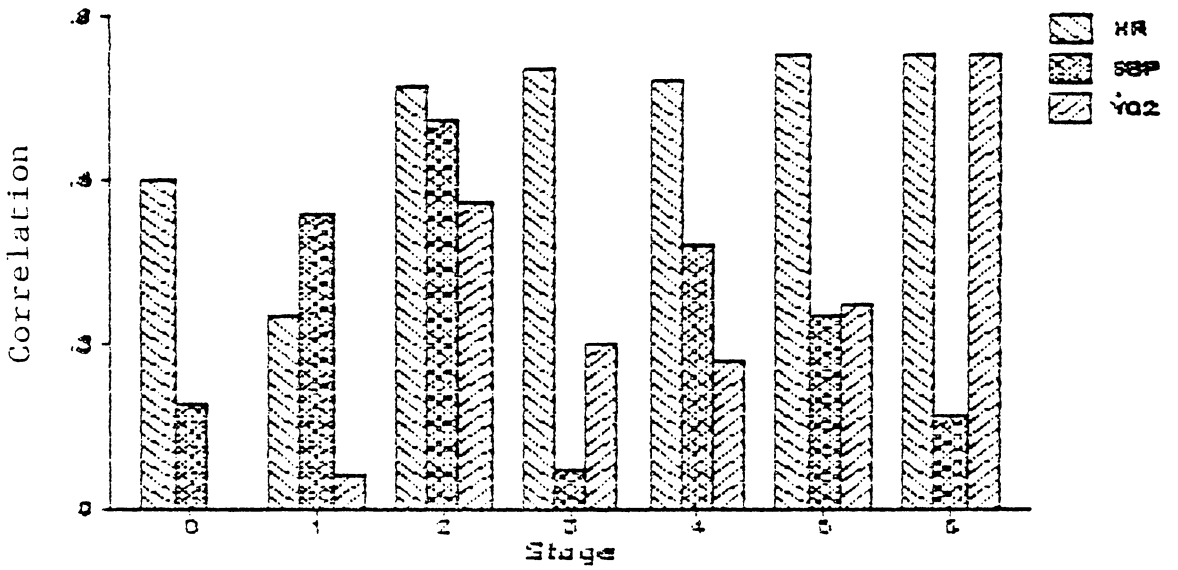

Figure 7. Correlation coefficients of physiological reponses of 30 subjects at each stage of graded exercise test.

CHAPTER IV

Summary

Various techniques have been developed to diagnose disease or predict the likelihood of a future heart attack. One factor which has prognostic importance relative to forecasting cardiac mortality is impairment of left ventricular ejection fraction (EF) of the heart. Stein and Sabbah (1976a,b,c) determined left ventricular function (LVF) at peak tension would allow them to distinguish between healthy and coronary diseased patients based upon the rate and flow changes in the blood. Chandnarata, Silveira and Aronow (1980) reported that the (CW) Doppler was sensitive enough to measure the blood flow indicators of left ventricular function.

Gardin, Burn, Childs and Henry (1984) and Sabbah, Khaja, Brymer, McFarland, Albert, Snyder, Goldstein and Stein (1986) reported that peak acceleration was more reliable as an indicator of LVF than average velocity in the pulmonary artery and the aorta. This finding was corroborated by the findings of Daley, Sagar and Wann (1985) who reported that during maximal exercise, aortic dimensions change little relative to resting conditions, thus supporting that changes in

flow characteristics measured at this site are not biased by vessel lumen changes. Consequently, blood flow velocity in the aorta is closely related to the rate at which it is ejected from the heart.

Despite the advances in determining the validity of a CW Doppler to date, this author has been unable to document any studies which have determined the reliability of these responses during exercise testing in healthy subjects.

The purpose of the study, then, was to determine if the a CW Doppler could provide reproducible measures within each stage of a modified Bruce protocol and also test-retest conditions on succesive days with the same subjects.

Duplicate maximal exercise trials (separate days) were conducted on a group of healthy male subjects (19-35 years of age) who were accustomed to regular vigorous exercise. During the first 2 minutes of each stage, blood pressure, HR, $\dot{V}O_2$, and RPE were measured (Tables 5-35). In each trial, LVF measurements were taken after two minutes of standing rest and then, throughout a series of progressively increasing speed/grade stages (final minute of each three minute stage) in a clinical treadmill test, i.e., the Bruce protocol (Appendix C). During the third minute of the stage, the Exerdop (A.H. Robbins, Co.) probe was placed

on the suprasternal notch of the individual and aimed towards the ascending aorta. The technician determined the placement of the probe by the auditory cues of the blood flow, and proceeded to record the data at the beginning of the final minute in a stage. The technician created a data set for each twenty second interval during that final minute of the stage. If it did not appear that the individual was going to complete the third minute of the stage, such as was found at the higher stages, then the experimenter attempted to record data for the final 60 seconds of the peak exercise.

Intraclass reliability was estimated to determine whether the measures of the different variables were stable during each stage of a graded exercise test. As noted for each of the values, there were moderately high correlations for all values, with PkV and PkA showing increases up to stage four, while SDI only increased up to stage three. PkV also had the most consistent values and the lowest values for the coefficient of variation.

Test-retest reliability was conducted to determine the consistency of the EXERDOP variables between repeated GXTs. As noted earlier, there was a low correlation for all values during the pre-test stage. Once the test began, there was an increase

in the correlations of all values up to the 4th stage. This consisted of a brisk walk for all subjects (111.7 $m \cdot min^{-1}$/ 16% grade). After the fourth stage, there was a decrease in the correlational value for both the 5th and 6th stages.

The objectives of this study were to determine the reliability of an EXERDOP during each stage of a graded exercise test and repeated test days. All values (PkV, PkA, and SVI) were statistically significant during the three measurements taken during the third minute of the stage. The third stage was selected because it was assumed that if the subjects could meet the workload, they should have been at physiological steady- state values. Also, the reliability was increased not only because of stable physiological values, but also because of the time intervals in which the measurements were made.

The test-retest reliability showed progressive increases in the correlation coefficient after the pretest stage and up to the 4th stage of exercise on each test day. The reliability may have been lower during the pre-test stage due to any number of factors, but probably due to a fluctuation in the catacholamine response which has also been suggested by Ihlen (1985). After the fourth stage, there was

a decrease in the correlation. This may possibly have been attributed to two factors. First, this was the beginning of the first running stage of the test. Consequently, it became increasingly difficult to maintain probe contact above the suprasternal notch while attempting to obtain accurate values. Secondly, with the increased workload, there should be an increase in the ejection fraction and aortic blood flow velocity. This would produce a greater amount of turbulence in the aorta, which may confound the values because the CW Doppler measures of blood flow are based upon an assumption of laminar flow.

From this study, it appears that non-invasive CW Doppler echocardiography may potentially be a useful instrument during repeated GXT's. Of the Doppler values measured (PkV, PkA, and SVI), PkV appears to be the most consistent measure within each stage, while all values for the healthy males tested in repeated GXTs were significant during the walking stages of a modified Bruce Protocol.

Implications for Future Research

From this study, there are still several questions that remain unanswered, and several questions that have arise from the results. The questions that need to be addressed are as follows:

1. What is the reliability of the EXERDOP during each minute of the test? As not all subjects were able to complete each minute of the final stage they began, there may have been some inaccuracies due to validity of the minute in which the measurements were taken.

2. What causes the plateauing effect of the response for the variables during the higher stages of exercise? Are they related to additional turbulence that produces artifact, or is it related to the subject's psychological perception that the test is about to end?

3. There needs to be the determination of whether postural changes or changes in the tension of muscle at the neck with increased exercise influence the reflection of the Doppler sound wave.

4. There needs to be an assessment of whether there are any extraneous signals being produced that confound the data when the Doppler is mounted as it is advertised on the top of an oscilloscope. It is conceivable that cathode rays may be produced that confound the averaging signals being produced by the EXERDOP.

Recommendations for Clinical Application

Although the use of this instrument is still in its infancy, there are several recommendations that should be considered as a result of this investigation:

1. This instrument appears to be most consistent when it measures blood flow velocities at lower exercise intensities. The most consistent value of those recorded by the EXERDOP appears to be PkV.

2. The subjects should not be permitted to hold onto the handrail while using this machine on the treadmill during a graded exercise test. There is an increase in the tension of the neck muscles which makes it hard to maintain contact between the probe and the surface of the skin. Also, there may be restriction for the sound waves to travel.

3. There needs to be the development of a regression formula based upon age/sex norms that would be applicable for individuals without symptoms of CHD and those who had been diagnosed as symptomatic. Because of discrepancies in the mean scores between this study (Figures 1-3) and Kelly's (1986) values for healthy individuals, each clinic may want to develop its own norms/values that are acceptable limits for the population it is assessing.

Recommendations for Future Development with the EXERDOP

There are several recommendations that the author would like to suggest for improving the technical performance of the EXERDOP:

1. The paper drive is set upside down. It should be reversed so that gravity will assist in driving the paper out of the machine and that a display of printed recording while testing is in progress be available.

2. Because the machine determines the values for a particular test segment based on an averaging of beats, the machine should have a digital display that notes when a specified number of "valid" beats has been reached. There should also be an adjustment which the experimenter can manipulate to set his/her own standard for the minimum number of valid beats.

3. There should be a structural reinforcement of the probe where the cord attaches to the handle. After many tests, this will be the point that deteriorates first. It may be that the handle should be detachable for easy storage.

4. There should be a protective cover for the probe to store it in when not in use.

5. The manufacturers should eliminate the LED on the handle because it is not of practical use during exercise testing.

6. The trigger on the probe should be moved to the side of the handle because it can not be manipulated during exercise testing in its present position.

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APPENDIX A

Selection of Subject

The experimenter recruited 30 male volunteer subjects of varying body types, age and functional capacity to participate in this study. The subjects ranged in age from 18 to 35 years, with no known risk factors as specified by the American College of Sports Medicine (1986) during exercise.

General Method

During two preliminary orientation periods, each subject was briefed as to the nature of the study, its purpose, and the possible research implications for such a study. The subject was shown how to walk on the treadmill, how to adjust their gait and how to balance themselves should they deem it necessary during the test.

During the first orientation trial the subjects were permitted to perform the first two stages of the test in their entirety; then, to assist with the orientation, the experimenter exposed each subject to the various speeds and grades that would be encountered during the experimental trials. These

stages were performed for approximately 15 seconds each, through the sixth stage.

During the second orientation trial, the subject was exposed to the same treatment as given in the first, with the addition of a mouthpiece and a noseclip. This increased the subjects' familiarity with the physiological responses to be measured in the experimental trials. This amount of time spent on the treadmill coincided with the work of Charteris and Taves (1978) who took novice treadmill walkers, they determined how long it took them to assume normal gait patterns. They reported gait stability in those subjects who had been on the treadmill 15 minutes, but that stride variance was present to some extent even after orientation. This was important because the less difficulty an individual had maintaining his gait during the test, the easier it was for him to control his upper body movement. This aided the technician by allowing him to maintain the EXERDOP probe contact on the suprasternal notch. Also, skinfold measurements were taken at the sites described by Jackson and Pollock (1978) for healthy young males. If the readings did not vary more than 10 percent during the first three trials at each site, then two additional trials were taken, with

the sites averaged to develop one score. This score was then compared to calculated norms for age and sex.

The subjects were informed that the experimental exercise trials were to include a maximal exercise test and that they were to refrain from eating a large meal on the day of the test, to avoid vigorous exercise on the day before or the day of the test, and to refrain from drinking alcohol prior to taking the test. These instructions followed the guidelines established by Goldstein et al.(1971) that development of angina in CHD patients during exercise after a meal was due to an increase in HR and BP. Jones and Haddon (1971) reported that post prandial exercises one hour after eating resulted in mobilization of free fatty acids and an increased ventilation, with no apparent affect on cardiovascular changes. Compliance was evaluated by the subject's self-report prior to the test. Prior to each test, the subject was asked about his diet and exercise pattern for the 24 hours prior to the GXT. Also he was asked about any medications or orthopedic problems that might have hindered his performance.

Experimental Procedures

The experiment consisted of thoroughly acquainting the subject with treadmill walking as found in the Bruce protocol. To ensure that the individual did not experience habituation effects that would bias data in the subsequent experimental trials, the heart rate (HR) was taken at rest and during the second minute of the second stage in this preliminary trial. This HR value was compared with the HRs of the second minute of the second stage in each of the two experimental trials to determine if there large discrepancies in the HR for the two tests.

Additionally, during a second orientation trial, each subject wore a mouthpiece and noseclip to better approximate the conditions of the experimental trial. Finally, the individuals were acquainted with the other stages of the test for about 15 seconds each during the second orientation trial.

At the outset of each experimental trial, technicians noted the barometric pressure in the room, and this was recorded on the data analysis sheet. The gas analyzers (AMETEK O_2 and CO_2 Analyzers) were calibrated to known room temperatures after the flow meters had been in use for at least 15 minutes for stabalization. The temperature of the oxygen sensor

was set at 6.790mV. The partial pressure of the oxygen (pO_2) analyzer was set at 20.93% pO_2 (room air), while the partial pressure of carbon dioxide (pCO_2) analyzer was set to fluctuate between 0.03 and 0.04% pCO_2 as the air at room temperature was assumed to contain 0.035% pCO_2.

The gas analyzers were then calibrated with two know gases as determined by standard Haldane procedures. The partial pressures of the gases in the first tank were 15.65% pO_2 and 5.10% pCO_2 and 18.05% pO_2 and 3.07% pCO_2 for the second tank.

The expired air samples were collected with the subject wearing a noseclip and a head gear apparatus that contained a mouthpiece and a Daniel's low resistance low dead space flutter one-way valve for collection of expired air. The expired air then flowed through a flexible 3.49 cm hose and three liter mixing chamber and passed over the heated sensor of the pneumotach (Hewlett Packard 47303A Digital Pneumotach). It is the cooling effect of the air passing over the heat sensors that determines the volume of gas that is being collected. The pneumotach was calibrated periodically using a three liter syringe to measure the amount of air flow going into the pneumotach for known periods of time. This calibration demonstrated a constant error which was used

to correct all subsequent calculations of ventilation at standard temperature, pressure and dry air (VESTPD) and $\dot{V}O_2$, i.e. 1.07. This remained constant throughout the test.

The air then passes into a 3 liter mixing chamber. This small chamber contained a sample port and small bore tyram tubing through which the sample of air was then drawn through Aquasorb absorbent into the gas analyzers by sealed pumps (AMETEK model P-G1) which were calibrated to both draw 500 $ml \cdot min^{-1}$ of air into the O and CO sensors (AMETEK models P-61B for CO_2, and N-22M for O_2 simultaneously). Visual averaging (DeBoever, 1985) was conducted at 15 seconds past the exercise minute in order to provide time for the indicative expired gas to be drawn into the sensor. Because it was the expired air that was being measured, two additional constants were incorporated into the conversions leading to calculation of VESTPD. The temperature of the gas was 27.2°C. The other constant was the water vapor pressure of O_2 at room temperature, i.e. 25.8°C.

The subjects were then fitted with the open circuit breathing system (described in the foregoing paragraph) and a blood pressure sphygmometer (Trimline, by PyMaH, Sommerville, N.J.). Baseline measurements

were taken with the subject standing in an upright position on the treadmill.

Also, baseline data were taken with the Exerdop (Quinton, A.H. Robbins, Co.). Placement of the Exerdop probe was just above the suprasternal notch and the probe was held at this site during the resting and exercise measurements by the experimenters. The angle at which the probe was held was determined by the auditory signal that was proportional to the reception of the sound waves being reflected. The experimenter monitored this with the assistance of headphones.

Once the baseline data were taken, the individual began a two minute walking warm-up in which they were not required to used the gas analysis equipment. They were then instructed to put in the mouthpiece and wear the noseclip just prior to the beginning the first test stage.

During the first minute of each stage, blood pressure and HR were measured (Hewlett Packard 1514C ECG system) after 50 seconds had elapsed. Ventilation and the partial pressure of oxygen were measured once during each minute of the test. During the second minute of the stage, HR was again measured at 50-60 seconds into the minute. During the third minute of the stage, the Exerdop probe was placed on the

individual. The technician pressed a remote foot pedal that triggered the instrument to start recording measurements. At 20 and 40 seconds into the minute, the experimenter would quickly lift his foot off the pedal and put it back down again. This would create a data set for each twenty second interval of the third minute of the stage. If it did not appear that the individual was going to complete the third minute of the stage, such as was found at the higher stages, then technician attempted to record the final exercise minute. Also, during the last minute of each stage, the heart rate was measure during the 20th, 40th and 50th seconds of the stage to provide heart rates that corresponded to the Exerdop measurements that were being made.

Statistical Procedures

The data from the Exerdop final report was separated into three variables: peak acceleration, peak velocity, and stroke distance. The data from the two readings during a submaximal stage was analyzed to estimate intraclass reliability using a repeated measures ANOVA. Next, the data were analyzed for test-retest reliability between different test days using Pearson Product Moment Correlation.

Table 6. Intraclass Correlations (R) and Coefficients of Variation (CV) for Peak Velocity During Graded Exercise for 30 Subjects

Stage	Test		Retest	
	R	CV	R	CV
0	0.93	0.23	0.90	0.21
1	0.94	0.20	0.95	0.24
2	0.94	0.16	0.98	0.22
3	0.94	0.17	0.96	0.18
4	0.94	0.17	0.93	0.16
5	0.93	0.21	0.96	0.22
6	0.81	0.15	0.96	0.24

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 7. Intraclass Correlations (R) and Coefficients of Variation (CV) for Systolic Velocity Intergral During Graded Exercise Tests for 30 Subjects

Stage	Test		Retest	
	R	CV	R	CV
0	0.88	0.26	0.94	0.28
1	0.86	0.25	0.89	0.23
2	0.85	0.18	0.93	0.22
3	0.93	0.18	0.89	0.19
4	0.95	0.19	0.92	0.21
5	0.85	0.21	0.93	0.25
6	0.84	0.21	0.91	0.26

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 8. Intraclass Correlations (R) and Coefficients of Variation (CV) for Peak Acceleration During Graded Exercise Tests for 30 Subjects

Stage	Test		Retest	
	R	CV	R	CV
0	0.93	0.38	0.94	0.22
1	0.91	0.37	0.89	0.36
2	0.93	0.32	0.93	0.36
3	0.93	0.28	0.97	0.38
4	0.91	0.25	0.96	0.27
5	0.91	0.24	0.94	0.28
6	0.79	0.18	0.90	0.26

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 9. Test-Retest reliability estimates for Peak Velocity

Stage	r	p
0	0.21	0.38
1	0.54	0.004
2	0.71	0.001
3	0.70	0.001
4	0.71	0.001
5	0.18	0.36
6	-0.81	0.60

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 10. Test-Retest reliability estimates for Systolic Velocity Integral

Stage	r	p
0	-0.38	0.11
1	0.51	0.004
2	0.54	0.001
3	0.75	0.001
4	0.78	0.001
5	0.12	0.59
6	0.006	0.99

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

Table 11. Test-Retest Reliability Estimates for Peak Acceleration

Stage	r	p
0	0.04	0.86
1	0.59	0.001
2	0.52	0.001
3	0.63	0.001
4	0.74	0.001
5	0.39	0.04
6	0.03	0.94

Stage:
0 = standing rest
1 = 45.7 $m \cdot min^{-1}$ / 10% grade
2 = 66.7 $m \cdot min^{-1}$ / 12% grade
3 = 90.0 $m \cdot min^{-1}$ / 14% grade
4 = 111.7 $m \cdot min^{-1}$ / 16% grade
5 = 133.3 $m \cdot min^{-1}$ / 18% grade
6 = 146.7 $m \cdot min^{-1}$ / 20% grade

APPENDIX B

LABORATORY FOR EXERCISE, SPORTS AND WORK PHYSIOLOGY

Division of Health, Physical Education and Recreation
Virginia Polytechnic Institute and State University

Informed Consent

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by the personnel of the Human Performance Laboratory of the Division of Health, Physical Education and Recreation of Virginia Polytechnic Institute and State University.

Title of Study: Reliability of Exercise Doppler Echocardiography.

The purpose of this experiment is to examine the reliability of a new Continuous-Wave echocardiographic device during maximal treadmill testing.

I voluntarily agree to participate in this testing program. It is my understanding that my participation will include: Two tests on a treadmill to maximal exercise levels. Each test will last from 12-18 min. During these tests the investigators will constantly monitor heart rate and rhythm, will measure blood pressure once every 3 min., will continuously collect my expired respiratory gases, and will determine

aortic blood flow with an echocardiographic device every 3 min. The blood flow determination will be made by a technician placing a hand-held probe above my sternum (the area where my neck and chest meet).

I understand that participation in this experiment may produce certain discomforts and risks. These discomforts and risks include: Abnormal changes in heart rate and /or rhythm, abnormal changes in blood pressure, fainting, very rare instances of heart attack, leg fatigue, skin irritation due to skin preparation for ECG monitoring, and minor soreness above the sternum from the pressure of the technician holding the probe in place for the blood flow measurements.

These risks will be minimized by screening for contraindications for me to exercise. The primary investigator and two of the laboratory technicians will follow the guidelines established by the American College of Sports Medicine (ACSM), and all of the technicians are certified in CPR.

Certain personal benefits may be expected from participation in this experiment. These include: Knowledge of my maximal oxygen uptake, which is the criterion measures of aerobic fitness.

I understand that any data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statements and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Scientific inquiry is indispensable to the advancement of knowledge. Your participation in this experiment provides the investigator the opportunity to conduct meaningful scientific observations designed to make significant educational contribution.

If you would like to receive a copy of the results of this investigation, please indicate this choice by marking in the appropirate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

____ I request a copy of the results of this study.

Date_____ Time _____a.m./p.m.

Participant Signature_____

Witness _____
HPL Personnel

Project Director Dr. William Herbert Telephone 961-6565

Section Investigation: Lindsay Wetherill Telephone 961-4900

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Dr. Charles Waring, Chairman, International Review Board for Research Involving Human Subjects. Phone 961-5283

APPENDIX C

SUBJECT CHARACTERISTICS AND PHYSIOLOGICAL RESPONSES

Index of Terms for Raw Data Set

$\dot{V}O_2$ = oxygen consumption in liter•min^{-1}

R = respiratory quotient

HR = heart rate

SBP = systolic blood pressure

DBP = diastolic blood pressure

Stage = 0 = standing rest
= 1 = 45.0 m•min^{-1}/10% grade
= 2 = 66.7 m•min^{-1}/12% grade
= 3 = 90.0 m•min^{-1}/14% grade
= 4 = 111.7 m•min^{-1}/16% grade
= 5 = 133.3 m•min^{-1}/18% grade
= 6 = 146.7 m•min^{-1}/20% grade

Table 12. Physiological Responses of Subject 1 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0		-	-	118	60
2	0		-	60	108	82
1	1	13.65	-	77	136	66
2	1	15.69	-	77	-	-
1	2	19.14	-	108	140	60
2	2	19.73	-	98	130	60
1	3	28.04	-	120	150	60
2	3	30.78	-	122	146	60
1	4	33.34	-	146	170	60
2	4	38.52	-	137	180	60
1	5	49.17	-	170	190	60
2	5	48.52	-	167	190	-
1	6	50.19	1.32	190	-	-
2	6	54.11	1.12	186	-	-

Table 13. Physiological Response of Subject 2 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			82	118	84
2	0			73	128	82
1	1	14.28	0.84	89	136	90
2	1	13.51	0.76	94	140	66
1	2	18.10	0.93	106	150	60
2	2	18.82	0.77	111	150	66
1	3	26.03	0.93	136	150	60
2	3	26.46	0.93	139	160	66
1	4	34.59	1.19	166	160	60
2	4	34.08	1.12	166	160	66
1	5	44.01	1.16	176	-	-
2	5	29.78	1.35	179	-	-

Table 14. Physiological Responses of Subject 3 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			69	118	80
2	0			-	122	72
1	1	12.51	0.90	87	134	76
2	1	13.52	0.83	83	-	-
1	2	18.50	0.96	101	146	74
2	2	18.47	0.93	101	-	-
1	3	25.40	1.12	123	160	72
2	3	26.30	1.05	127	150	64
1	4	33.60	1.33	159	-	-
2	4	31.05	1.29	162	170	64
1	5	35.54	1.48	170	-	-
2	5	37.53	1.47	170	-	-

Table 15. Physiological Responses of Subject 4 During GXTs

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			82	108	78
2	0			72	96	70
1	1	13.26	0.80	96	110	60
2	1	13.21	0.80	91	110	70
1	2	19.03	0.86	112	130	60
2	2	18.95	0.86	105	120	60
1	3	22.40	1.11	132	148	60
2	3	22.31	1.11	125	130	60
1	4	28.12	1.13	150	160	60
2	4	28.01	1.13	141	150	60
1	5	39.78	1.23	185	180	-
2	5	42.70	1.14	177	160	-
1	6	38.62	1.29	180	-	-
2	6	38.47	1.29	-	-	-

Table 16. Physiological Responses of Subject 5 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			67	-	-
2	0			68	120	70
1	1	14.05	0.76	105	-	-
2	1	15.35	0.66	98	132	66
1	2	22.48	0.81	120	-	-
2	2	20.71	0.81	112	140	66
1	3	25.14	0.96	143	-	-
2	3	31.52	0.89	137	150	66
1	4	33.10	1.07	161	-	-
2	4	44.81	0.97	159	180	60
1	5	44.83	1.24	179	-	-
2	5	49.02	1.30	179	200	60
2	6	49.25	1.31	185	-	-

Table 17. Physiological Responses of Subject 6 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			71	-	-
2	0			-	132	88
1	1	13.88	0.82	84	-	-
2	1	13.45	0.82	77	134	88
1	2	18.14	0.90	90	152	72
2	2	17.81	0.87	99	144	88
1	3	22.24	0.96	105	-	-
2	3	24.55	0.87	110	150	60
1	4	31.15	0.98	130	170	70
2	4	31.35	0.94	142	162	70
1	5	40.46	1.07	162	190	68
2	5	44.14	1.05	167	-	-
1	6	46.14	1.12	175	-	-
2	6	44.78	1.23	180	-	-

Table 18. Physiological Responses of Subject 7 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			82	160	86
2	0			100	150	90
1	1	14.34	1.08	107	160	86
2	1	14.03	0.96	106	168	88
1	2	21.62	0.88	119	200	70
2	2	19.48	0.92	118	180	72
1	3	29.11	0.97	147	190	70
2	3	26.17	1.02	137	190	70
1	4	37.79	1.11	178	210	-
2	4	37.01	1.18	177	200	80
1	5	46.93	1.25	191	210	-
2	5	42.56	1.34	189	200	-
1	6	23.39	1.36	196	210	-
2	6	34.83	1.26	200	-	-

Table 19. Physiological Responses of Subject 8 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			71	126	74
2	0			82	118	82
1	1	14.47	0.69	89	-	-
2	1	13.04	0.78	87	132	72
1	2	19.45	0.80	110	140	70
2	2	18.14	0.75	99	134	70
1	3	25.08	0.98	137	158	68
2	3	22.22	1.05	122	164	70
1	4	30.34	1.22	173	168	68
2	4	32.45	1.21	171	184	70
1	5	39.11	1.31	193	-	-
2	5	39.79	1.31	191	194	70
2	6	36.43	1.50	195	-	-

Table 20. Physiological Responses of Subject 9 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			72	126	80
2	0			66	116	76
1	1	15.20	0.70	101	140	72
2	1	13.89	0.79	91	126	70
1	2	20.99	0.77	124	150	70
2	2	17.22	0.91	106	140	60
1	3	27.20	0.86	150	160	60
2	3	26.73	0.99	132	150	60
1	4	28.95	1.04	175	170	60
2	4	28.28	1.04	159	160	60
1	5	41.79	1.37	196	-	-
2	5	39.24	1.39	189	-	-

Table 21. Physiological Responses of Subject 10 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			90	122	88
2	0			82	132	94
1	1	16.35	0.79	101	148	86
2	1	15.42	0.68	100	142	90
1	2	20.72	0.85	112	150	76
2	2	23.19	0.71	117	150	90
1	3	28.46	0.98	137	160	76
2	3	31.04	0.82	145	168	88
1	4	34.30	1.07	169	170	70
2	4	31.30	1.38	182	190	84
1	5	44.04	1.40	196	180	-
2	5	50.67	1.17	200	202	78

Table 22. Physiological Responses of Subject 11 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			60	-	-
2	0			60	120	82
1	1	19.74	0.75	90	136	74
2	1	12.58	0.83	90	156	-
1	2	22.48	0.92	104	132	84
2	2	17.40	0.88	-	-	-
1	3	25.06	1.01	130	-	-
2	3	22.42	1.08	-	150	66
1	4	29.39	1.15	155	-	-
2	4	33.22	1.03	-	-	-
1	5	29.39	1.15	-	-	-

Table 23. Physiological Responses of Subject 12 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			81	114	50
2	0			80	120	88
1	1	14.50	0.90	99	148	60
2	1	12.83	0.87	112	136	70
1	2	18.60	0.93	119	148	60
2	2	16.62	0.87	118	154	70
1	3	24.90	1.04	149	164	70
2	3	19.62	1.04	143	156	68
1	4	34.23	1.13	192	180	70
2	4	27.43	1.14	180	160	76
1	5	44.87	1.26	211	190	-
2	5	34.33	1.12	195	-	-

Table 24. Physiological Responses of Subject 13 During GTXs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			70	118	60
2	0			73	126	68
1	1	15.74	0.77	115	130	64
2	1	15.66	0.78	108	120	70
1	2	19.53	0.83	130	142	70
2	2	18.81	0.89	131	154	68
1	3	27.91	0.91	158	170	66
2	3	25.51	0.96	155	176	68
1	4	-	-	188	174	70
2	4	34.73	1.05	172	188	68
1	5	42.84	1.12	190	180	60
2	5	47.16	1.12	190	194	68

Table 25. Physiological Responses of Subject 14 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			64	112	78
2	0			68	138	88
1	1	13.90	0.75	85	130	70
2	1	15.27	0.67	85	-	-
1	2	15.83	0.88	93	148	70
2	2	19.53	0.81	100	150	88
1	3	25.71	0.91	104	152	70
2	3	30.84	0.83	120	-	-
1	4	34.10	0.96	150	172	76
2	4	40.29	0.91	150	190	90
1	5	50.61	1.06	171	170	-
2	5	47.97	1.14	176	196	80
1	6	60.62	1.10	190	-	-
2	6	53.51	1.19	189	-	-

Table 26. Physiological Responses of Subject 15 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			72	108	72
2	0			83	126	76
1	1	22.69	0.81	94	128	72
2	1	14.45	0.79	101	150	80
1	2	23.57	0.87	104	-	-
2	2	20.08	0.82	115	160	70
1	3	30.79	0.97	130	-	-
2	3	28.77	0.92	136	166	70
1	4	37.51	0.97	146	-	-
2	4	34.49	1.14	165	190	70
1	5	48.53	1.14	170	-	-
2	5	48.75	1.17	178	200	-
1	6	51.87	1.15	180	-	-

Table 27. Physiological Responses of Subject 16 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			57	122	76
2	0			66	108	80
1	1	14.56	0.91	90	142	64
2	1	12.72	0.83	94	120	76
1	2	20.80	0.94	112	150	68
2	2	17.24	0.92	106	146	76
1	3	28.24	0.98	136	152	68
2	3	23.53	1.00	126	146	70
1	4	35.44	1.07	150	182	68
2	4	35.62	1.09	150	160	70
1	5	45.03	1.35	187	198	68
2	5	34.71	1.41	187	180	-

Table 28. Physiological Responses of Subject 17 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			91	106	74
2	0			85	134	90
1	1	24.27	0.44	100	124	74
2	1	14.39	0.84	105	150	80
1	2	26.42	0.77	125	124	68
2	2	27.44	0.85	125	160	76
1	3	32.66	0.92	150	162	74
2	3	28.68	1.03	150	170	76
1	4	47.11	0.94	184	174	74
2	4	29.71	1.15	166	186	76
1	5	39.69	1.20	188	186	74
2	5	44.44	1.28	188	190	-
2	6	43.81	1.34	198	-	-

Table 29. Physiological Responses of Subject 18 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			71	128	82
2	0			60	132	78
1	1	14.91	0.77	92	166	80
2	1	14.75	0.74	83	152	68
1	2	19.05	0.80	111	174	74
2	2	19.11	0.86	105	172	70
1	3	27.50	0.86	136	182	70
2	3	25.72	0.93	122	186	68
1	4	34.24	0.91	150	194	68
2	4	34.82	0.97	150	194	68
1	5	44.46	0.99	167	202	70
2	5	49.19	1.11	176	210	70
1	6	50.26	1.17	187	216	68
2	6	55.29	1.22	187	214	70

Table 30. Physiological Responses of Subject 19 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			100	118	80
2	0			88	108	80
1	1	9.90	0.82	111	130	70
2	1	16.53	0.84	107	120	76
1	2	19.68	0.84	120	144	68
2	2	21.07	0.91	115	146	76
1	3	29.25	0.98	142	152	68
2	3	27.81	0.99	136	146	76
1	4	38.33	1.15	176	-	-
2	4	39.46	1.09	175	160	70
1	5	50.89	1.23	187	-	-
2	5	48.15	1.20	185	180	-

Table 31. Physiological Responses of Subject 20 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			65	104	70
2	0			88	104	80
1	1	15.39	0.66	127	124	70
2	1	14.70	0.71	115	124	80
1	2	21.63	0.82	148	124	70
2	2	21.63	0.78	136	136	80
1	3	27.81	0.91	167	140	70
2	3	27.76	0.89	167	154	80
1	4	34.38	1.02	172	154	66
2	4	33.48	0.95	186	164	80
1	5	43.63	1.06	187	164	60
2	5	45.31	1.14	200	174	80
1	6	43.13	1.42	214	-	-

Table 32. Physiological Responses of Subject 21 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			80	130	90
2	0			71	120	84
1	1	13.73	0.84	107	156	90
2	1	13.06	0.84	100	130	70
1	2	17.44	0.93	115	150	88
2	2	16.91	0.90	115	134	70
1	3	25.63	0.96	136	170	86
2	3	19.62	1.04	143	156	68
1	4	34.23	1.13	192	180	70
2	4	27.43	1.14	180	160	76
1	5	44.87	1.26	211	190	-
2	5	34.33	1.12	195	-	-

Table 33. Physiological Responses of Subject 22 During GTXs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			65	114	70
2	0			49	-	-
1	1	15.20	0.81	87	134	70
2	1	16.33	0.82	75	120	80
1	2	21.87	0.84	107	140	70
2	2	18.24	0.92	95	124	60
1	3	31.09	0.91	130	160	70
2	3	26.90	0.95	122	142	60
1	4	44.80	0.96	170	190	70
2	4	41.88	1.02	155	184	60
1	5	49.18	1.03	180	-	-
2	5	49.42	1.19	176	194	60
1	6	66.38	1.10	190	-	-
2	6	66.38	1.29	187	208	60

Table 34. Physiological Responses of Subject 23 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			94	106	84
2	0			-	120	82
1	1	18.09	0.64	98	120	74
2	1	20.55	0.71	110	120	74
1	2	23.85	0.74	115	130	74
2	2	28.62	0.76	125	132	70
1	3	23.41	0.86	136	152	72
2	3	33.17	0.83	136	144	70
1	4	34.99	1.03	167	160	70
2	4	34.21	0.97	167	150	66
1	5	47.56	1.27	192	170	70
2	5	47.45	1.09	188	166	60
1	6	52.67	1.27	200	166	60
2	6	51.36	1.17	200	170	60

Table 35. Physiological Responses of Subject 24 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			75	-	-
2	0			88	102	80
1	1	16.08	0.67	90	132	76
2	1	14.09	0.83	90	130	80
1	2	22.92	0.77	107	140	74
2	2	19.58	0.85	106	136	80
1	3	28.17	0.87	125	158	74
2	3	29.99	0.86	130	156	76
1	4	31.40	0.99	150	172	68
2	4	35.08	1.04	150	170	80
1	5	48.47	1.08	167	180	70
2	5	49.78	1.10	178	190	-
1	6	47.29	1.10	187	190	70
2	6	46.29	1.33	190	-	-

Table 36. Physiological Responses of Subject 25 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			100	124	74
2	0			94	124	78
1	1	16.48	0.73	118	136	78
2	1	15.07	0.81	107	132	78
1	2	21.60	0.94	136	146	74
2	2	21.82	0.86	125	140	78
1	3	30.76	1.07	167	162	76
2	3	32.88	0.88	162	152	76
1	4	36.40	1.19	205	168	72
2	4	36.60	1.20	187	166	76
1	5	45.39	1.19	214	182	72
2	5	36.64	1.14	-	-	-

Table 37. Physiological Responses of Subject 26 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($m \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			83	120	80
2	0			94	112	80
1	1	15.39	0.74	111	140	80
2	1	14.46	0.83	125	118	80
1	2	19.14	0.86	130	144	80
2	2	22.40	0.92	150	144	80
1	3	29.48	0.97	136	-	-
2	3	23.35	1.06	167	154	78
1	4	36.33	1.32	164	152	74
2	4	-	-	191	184	80
1	5	41.47	1.29	191	156	70
2	5	41.96	1.32	205	202	80

Table 38. Physiological Responses of Subject 27 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			82	154	80
2	0			88	142	80
1	1	14.78	0.84	114	174	78
2	1	14.77	0.77	112	154	76
1	2	20.02	0.82	134	-	-
2	2	19.06	0.83	115	168	78
1	3	25.98	1.05	155	190	74
2	3	25.47	0.88	145	174	78
1	4	38.75	0.99	187	-	-
2	4	31.49	0.98	167	184	78
1	5	48.28	1.03	187	210	74
2	5	39.12	1.09	187	198	76
1	6	43.88	1.42	187	-	-
2	6	46.24	1.05	200	202	78
2	7	51.76	1.04	187		

Table 39. Physiological Responses of Subject 28 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($m \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			68	-	-
2	0			65	126	72
1	1	13.69	0.90	88	130	74
2	1	14.79	0.81	85	148	70
1	2	18.06	0.82	98	154	76
2	2	18.08	0.80	94	158	70
1	3	23.50	0.84	115	156	70
2	3	25.03	0.84	110	170	70
1	4	29.17	0.91	127	170	70
2	4	31.31	0.89	127	176	70
1	5	38.22	1.01	152	194	60
2	5	42.58	1.02	134	184	70
1	6	44.45	1.13	167	200	60
2	6	47.99	1.14	167	196	72
1	7	46.92	1.12	167	150	60
2	7	46.85	1.12	187	-	-

Table 40. Physiological Responses of Subject 29 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			93	110	68
2	0			75	108	76
1	1	13.70	0.85	88	118	62
2	1	12.92	1.00	87	122	70
1	2	18.04	0.81	103	122	68
2	2	21.49	0.77	102	124	68
1	3	23.39	0.90	120	128	70
2	3	28.36	0.98	123	136	70
1	4	38.36	1.07	150	164	68
2	4	33.41	1.13	152	146	70
1	5	49.99	1.13	187	176	68
2	5	46.35	1.29	187	152	70

Table 41. Physiological Responses of Subject 30 During GXTs.

Test	Stage	$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	R	HR ($b \cdot min^{-1}$)	SBP (mmHg)	DBP (mmHg)
1	0			71	132	66
2	0			88	112	62
1	1	11.12	0.98	102	136	68
2	1	14.72	0.83	98	114	62
1	2	18.54	0.83	107	152	72
2	2	17.06	0.85	107	136	66
1	3	25.96	0.95	125	162	68
2	3	24.69	0.90	136	-	-
1	4	34.07	0.97	152	174	68
2	4	31.75	1.02	167	158	66
1	5	45.69	1.17	187	-	-
2	5	51.05	1.12	187	182	60
1	6	49.56	1.37	197	-	-
2	6	64.97	0.97	187	-	-

Table 42. Physical Characteristics of Subjects

Subject	Height (cm)	Body Fat %	$\dot{V}O_2$ ($ml \cdot kg^{-1}$)	R	HR ($b \cdot min^{-1}$)
1	195	8.2	54.11	1.32	190
2	178	18.0	44.01	1.35	179
3	180	25.4	37.53	1.48	170
4	166	16.9	42.70	1.29	185
5	163	16.0	49.25	1.31	185
6	181	13.4	46.14	1.23	180
7	180	9.4	46.93	1.36	200
8	174	13.6	39.79	1.50	195
9	177	10.2	41.79	1.39	196
10	176	12.5	50.67	1.40	200
11	177	10.7	33.22	1.15	-
12	170	12.5	44.87	1.26	211
13	189	14.3	47.16	1.12	190
14	183	24.4	60.62	1.19	190
15	169	8.0	51.87	1.17	180
16	180	16.2	45.03	1.41	187
17	180	24.0	44.44	1.34	198
18	181	8.9	55.29	1.22	187
19	180	20.9	50.89	1.23	187
20	188	24.0	45.31	1.42	214
21	173	10.7	44.87	1.26	211
22	175	11.6	66.38	1.10	190
23	171	13.4	52.67	1.27	200
24	187	15.7	49.78	1.33	190
25	167	13.1	45.39	1.19	214
26	183	13.4	51.96	1.32	205
27	185	13.4	51.76	1.42	200
28	179	17.6	47.99	1.14	187
29	175	30.0	49.99	1.29	187
30	178	16.5	64.97	1.37	197
$\bar{x}$ =	178.00	15.43	48.25	1.29	193.00
SD =	±7.12	±5.58	±7.25	±0.11	±10.92

APPENDIX D

Subject Doppler Responses

Table 43. EXERDOP Values for Subject 1.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		11	0.50	5.5	9	0.41	4.7
1	a	17	0.64	7.5	13	0.59	7.4
	b	16	0.72	8.4	15	0.64	9.0
	c	15	0.79	9.3	12	0.54	7.4
2		24	0.79	11.6	15	0.66	8.0
3		21	0.72	10.7	19	0.80	9.1
4		28	1.02	10.4	26	0.97	10.3
5		38	0.96	8.5	28	0.76	6.0
6	a	35	0.91	6.1	23	0.61	6.0
	b	31	0.60		26	0.56	3.9
	c	33	0.66	3.5	27	0.68	4.8

Table 44. EXERDOP Value for Subject 2.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		11	0.54	5.6	13	0.57	5.6
1	a	19	0.86	9.1	20	0.88	8.3
	b	18	0.78	8.2	19	0.84	8.8
	c	21	0.88	9.0	22	0.87	8.9
2		27	1.10	11.0	28	1.15	10.8
3		35	1.26	10.9	32	1.12	9.1
4		52	1.17	8.4	60	1.33	9.4
5	a	43	0.89	5.5	60	1.15	7.8
	b	56	1.14	5.9	58	1.19	8.5
	c	61	1.19	7.1	56	1.16	7.5

Table 45. EXERDOP Values for Subject 3.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					15	0.44	5.7
1	a	30	0.75	10.5	18	0.60	10.4
	b	20	0.77	10.0	18	0.69	9.5
	c	17	0.73	9.9	18	0.68	10.0
2		24	0.76	9.6	24	0.88	11.0
3		38	0.87	11.0	38	0.99	10.8
4		39	0.87	8.0	35	0.91	9.2
5	a	31	0.84	6.7	33	0.79	6.9
	b	37	0.88	7.2	29	0.75	6.3
	c	30	0.88	6.6	31	0.80	6.6

Table 46. EXERDOP Values for Subject 4.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		14	0.50	7.1	14	0.50	7.1
1	a	15	0.61	6.4	15	0.61	6.4
	b	16	0.61	7.2	16	0.61	7.2
	c	16	0.59	7.2	16	0.59	7.2
2		19	0.70	7.4	19	0.70	7.4
3		26	0.78	7.2	26	0.78	7.2
4		49	1.02	9.2	50	1.02	9.2
5		41	0.84	6.2	41	0.84	6.2
6	a	40	0.84	5.5	36	0.84	5.5
	b	32	0.77	4.8	32	0.77	5.0
	c	41	0.84	5.2			

Table 47. EXERDOP Values for Subject 5.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					12	0.51	8.3
1	a	22	0.78	10.2	17	0.76	10.5
	b	20	0.79	10.6	18	0.82	10.8
	c	21	0.75	10.1	18	0.82	10.2
2		38	0.91	10.3	25	0.82	9.7
3		44	0.90	8.0	40	0.91	8.2
4		41	0.84	6.5	44	0.95	7.6
5	a	35	0.66	4.6	28	0.63	3.7
	b	29	0.55	4.4	23	0.52	4.2
	c	31	0.63	3.8	41	0.71	4.5

Table 48. EXERDOP Values for Subject 6.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
1	a	15	0.66	8.0	42	0.85	12.8
	b	17	0.64	7.4	25	0.59	6.3
	c	21	0.75	8.6	20	0.60	5.9
2		30	0.85	11.9	29	0.84	8.8
3		27	0.86	10.3	25	0.82	9.5
4		22	0.73	7.9	27	0.83	7.7
5		25	0.68	6.1	26	0.72	6.9
6	a	37	0.82	6.2	22	0.56	4.9
	b	41	0.79	5.3	17	0.50	4.3
	c	36	0.83	5.9	19	0.52	4.4

Table 49. EXERDOP Values for Subject 7.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		21	0.90	8.9	17	0.69	6.5
1	a	22	0.96	10.4	30	1.04	10.9
	b	22	0.94	10.4	24	1.02	10.4
	c	25	1.01	10.6	21	0.96	9.8
2		24	0.94	9.8	31	1.12	12.3
3		28	0.98	9.3	33	1.31	11.7
4		35	1.13	9.5	38	1.23	11.5
5		33	0.93	6.6	38	1.07	8.1
6	a	35	0.83	6.4	45	0.91	5.6
	b	40	0.76	4.4	43	0.91	5.4
	c	43	0.83	4.6	38	1.10	7.8

Table 50. EXERDOP Values for Subject 8.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					11	0.43	3.9
1	a	14	0.61	6.6	42	0.90	15.0
	b	18	0.70	7.1	27	0.66	9.5
	c	18	0.72	6.6	33	0.73	9.4
2		22	0.77	7.2	27	0.77	9.1
3		33	0.83	7.1	32	0.91	8.6
4		40	0.86	6.0	42	0.91	7.5
5	a	32	0.76	6.3	42	0.86	5.9
	b	34	0.78	5.5			
	c	36	0.81	5.5			
6	a				39	0.86	5.8
	b				54	0.91	5.7
	c				47	0.85	5.6

Table 51. EXERDOP Values for Subject 9.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		9	0.43	4.1	13	0.54	5.4
1	a	19	0.78	7.8	15	0.70	7.6
	b	20	0.85	8.8	18	0.76	7.6
	c	22	0.92	9.2	19	0.77	7.6
2		28	1.06	9.9	17	0.74	8.1
3		30	0.99	8.0	23	0.83	8.5
4		41	1.13	8.6	23	0.72	6.3
5	a	44	0.80	4.7	36	0.85	6.4
	b	35	0.76	3.9	41	0.84	5.8
	c	44	0.87	4.7	39	0.89	6.2

Table 52. EXERDOP Values for Subject 10

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		11	0.48	4.3	17	0.46	5.0
1	a	18	0.73	8.1	35	0.95	11.5
	b	17	0.71	7.0	31	0.68	9.1
	c	16	0.69	6.5	30	0.68	7.3
2		18	0.73	6.7	18	0.52	7.1
3		23	0.87	7.1	23	0.80	7.1
4		20	0.74	6.1	23	0.72	6.3
5	a	18	0.48	4.2	19	0.55	6.0
	b	18	0.48	4.7	19	0.58	6.4
	c	17	0.51	5.1	19	0.65	5.1

Table 53. EXERDOP Values for Subject 11.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					14	0.65	9.8
1	a	39	1.11	12.8	38	0.98	12.9
	b	56	1.30	16.0	42	1.12	14.5
	c	60	1.35	17.0	37	1.11	13.9
2		57	1.14	11.2	50	1.23	13.4
3					62	1.22	10.3
4		58	1.13	7.6	47	0.99	6.8
5	a	46	1.06	6.8	42	0.88	6.9
	b	49	1.04	6.2	48	1.04	6.8
	c	51	1.10	6.6	51	1.04	6.7

Table 54. EXERDOP Values for Subject 12.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
1	a	25	0.81	10.2	28	0.91	12.0
	b	19	0.73	7.8	31	0.94	10.8
	c	16	0.75	7.8	29	1.06	11.6
2		21	0.92	9.1	29	0.97	11.5
3		37	1.11	9.1	33	1.08	12.2
4		43	1.12	8.1	53	1.13	10.0
5	a	39	1.20	7.2	32	0.68	6.9
	b	47	1.04	6.9	21	0.53	5.5
	c	44	1.13	7.5	24	0.56	5.1

Table 55. EXERDOP Values for Subject 13.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		18	0.41	5.3	18	0.67	7.4
1	a	40	0.84	8.2	44	1.01	11.5
	b	41	0.77	8.3	28	0.75	7.3
	c	35	0.66	9.6	33	0.87	8.8
2		23	0.67	7.3	27	0.81	7.9
3		22	0.71	6.3	48	0.79	6.7
4		31	0.76	6.1	46	0.74	5.8
5	a	38	0.73	5.2	44	0.75	6.0
	b	34	0.73	5.5	37	0.75	5.0
	c	37	0.75	4.3	41	0.74	5.6

Table 56. EXERDOP Values for Subject 14.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		10	0.40	3.6	14	0.48	5.5
1	a	18	0.71	7.6	18	0.76	9.2
	b	18	0.70	8.0	18	0.75	9.0
	c	19	0.73	8.5	25	0.80	9.4
2		20	0.80	8.8	23	0.88	10.3
3		26	0.94	10.2	28	0.89	9.5
4		40	0.98	9.7	33	1.02	9.1
5		36	0.78	5.8	36	0.81	5.7
6	a	31	0.63	4.8	38	0.77	4.0
	b	22	0.51	3.0	38	0.73	3.8
	c	31	0.63	3.6	43	0.77	4.6

Table 57. EXERDOP Values for Subject 15.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		35	0.60	6.7	10	0.39	3.1
1	a	17	0.76	8.3	13	0.57	5.8
	b	17	0.70	8.5	13	0.56	5.8
	c	18	0.75	8.1	14	0.57	5.8
2		19	0.77	8.8	15	0.52	5.8
3		21	0.89	8.4	19	0.75	6.2
4		39	1.00	8.8	37	0.87	6.9
5	a	40	0.95	6.9	34	0.69	4.4
	b	48	1.12	7.5	32	0.59	3.6
	c	37	0.89	7.0	40	0.80	4.8

Table 58. EXERDOP Values for Subject 16.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		14	0.63	6.6	15	0.60	5.6
1	a	20	0.82	8.9	20	0.86	8.8
	b	22	0.85	9.5	28	1.04	10.9
	c	22	0.82	8.7	24	0.85	9.0
2		22	0.84	8.7	21	0.72	8.2
3		35	1.02	9.6	23	0.79	7.1
4	a	42	1.03	9.0	27	0.78	6.6
	b	35	0.97	9.2			
	c	33	0.90	8.2			
5	a				31	0.66	4.5
	b				28	0.83	6.8
	c				39	0.79	5.4

Table 59. EXERDOP Values for Subject 17.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		15	0.53	5.1	13	0.53	5.7
1	a	42	1.11	21.0	18	0.69	7.0
	b	27	0.73	13.7	20	0.73	7.6
	c	17	0.73	7.6	19	0.71	7.0
2		22	0.76	6.9	24	0.74	7.2
3		30	0.82	6.3	42	0.85	6.7
4		31	0.72	4.9	45	0.86	6.4
5	a	44	0.73	4.6	34	0.66	4.4
	b	43	0.84	5.6	38	0.77	6.2
	c	45	0.83	4.9	40	0.71	5.0

Table 60. EXERDOP Values for Subject 18.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		12	0.50	5.1	10	0.58	4.6
1	a	24	0.93	10.6	20	0.82	9.4
	b	22	0.85	9.6	17	0.74	8.6
	c	21	0.85	9.8	15	0.66	7.0
2		28	0.98	11.0	26	0.97	10.8
3		45	1.04	9.9	38	1.04	10.3
4		47	1.02	9.0	53	1.10	8.6
5	a	44	0.99	8.8	48	0.99	7.4
	b	52	1.03	7.8	43	0.98	7.4
	c	43	0.94	8.2	47	0.87	5.3
6		45	0.90	6.6	47	0.87	5.3
7		48	0.96	6.6			

Table 61. EXERDOP Values for Subject 19.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		12	0.41	4.2	14	0.49	4.9
1	a	24	0.70	7.3	20	0.71	7.2
	b	15	0.57	5.7	17	0.69	7.0
	c	15	0.55	5.8	14	0.63	6.4
2		20	0.75	7.3	19	0.72	7.4
3		22	0.82	7.9	20	0.68	6.5
4		28	0.83	7.2	40	0.91	6.9
5	a	28	0.59	4.6	28	0.55	4.2
	b	27	0.67	4.0	25	0.57	4.2
	c	26	0.57	5.2	26	0.56	5.0

Table 62. EXERDOP Values for Subject 20.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					11	0.35	4.1
1	a	20	0.65	6.2	11	0.51	4.9
	b	21	0.61	6.4	13	0.51	5.4
	c	14	0.50	5.2	13	0.48	4.8
2		18	0.67	7.1	15	0.63	6.2
3		22	0.68	5.7	20	0.80	6.1
4		30	0.80	5.9	24	0.82	5.7
5	a	27	0.53	4.2	20	0.64	5.4
	b	19	0.55	5.1	22	0.51	3.7
	c	25	0.66	5.6	15	0.39	4.5
6		23	0.53	3.6			

Table 63. EXERDOP Values for Subject 21.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					11	0.39	3.5
1	a	18	0.57	7.4	19	0.60	6.4
	b	19	0.61	6.7	14	0.59	6.9
	c	17	0.58	6.6	14	0.63	6.9
2		18	0.63	7.8	14	0.55	6.7
3		20	0.75	8.3	18	0.74	7.6
4		24	0.84	7.9	24	0.79	7.7
5	a	21	0.54	3.6	30	0.62	4.6
	b	24	0.65	5.0	27	0.54	3.4
	c	32	0.72	5.2	21	0.49	2.6
6		26	0.47	2.6			

Table 64. EXERDOP Values for Subject 22.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		16	0.67	7.8	12	0.48	6.4
1	a	23	0.69	9.4	41?	0.89	9.6
	b	17	0.62	9.4	17	0.67	8.5
	c	16	0.62	8.6	13	0.84	7.5
2		20	0.81	9.3	18	0.75	10.6
3		27	0.73	8.1	18	0.65	8.7
4		38	0.63	5.8	27	0.68	6.4
5	a	39	0.79	5.2	24	0.51	4.2
	b	26	0.53	3.2	31	0.56	3.8
	c	26	0.60	3.8	30	0.56	3.0
6		33	0.61	3.5			

Table 65. EXERDOP Values for Subject 23.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		16	0.56	5.1	16	0.45	5.1
1	a	25	0.71	6.6	20	0.77	8.0
	b	15	0.64	6.1	19	0.78	8.0
	c	20	0.74	7.1	20	0.77	7.6
2		22	0.83	8.1	26	0.85	8.8
3		37	1.13	10.2	32	0.96	8.6
4		44	1.19	9.7	44	1.15	9.3
5	a	42	1.00	8.2	49	1.06	7.6
	b	34	0.83	6.0	41	0.99	7.6
	c	54	1.05	7.0	58	1.23	8.6
6		41	0.77	4.5	44	1.02	7.0

Table 66. EXERDOP Values for Subject 24.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0					14	0.55	5.7
1	a	15	0.66	6.0	31	0.91	10.0
	b	13	0.58	5.8	28	0.92	9.6
	c	14	0.60	6.0	28	0.92	10.0
2		18	0.77	7.2	52	1.05	10.3
3		27	0.89	7.7	56	1.08	8.8
4		56	1.19	7.1	53	1.01	6.5
5	a	54	1.00	5.0	41	0.78	5.2
	b	44	0.86	4.6	35	0.75	4.6
	c	53	0.97	6.4	40	0.79	5.0

Table 67. EXERDOP Values for Subject 25.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		9	0.31	2.6	6	0.27	2.5
1	a	13	0.50	5.8	20	0.53	5.2
	b	14	0.54	6.2	9	0.40	5.2
	c	12	0.51	5.6	10	0.43	6.0
2		16	0.62	6.4	16	0.62	6.9
3		20	0.77	6.7	20	0.76	7.0
4		28	0.82	6.0	26	0.81	6.1
5	a	34	0.85	5.4	29	0.73	5.4
	b	33	0.86	4.4	38	0.81	5.0
	c	32	0.88	5.2	25	0.67	5.1

Table 68. EXERDOP Values for Subject 26.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		14	0.46	6.1	16	0.59	6.2
1	a	20	0.74	8.8	19	0.77	7.6
	b	17	0.77	9.0	18	0.75	7.2
	c	13	0.75	9.2	18	0.71	7.0
2		30	0.88	9.4	28	0.84	7.4
3		45	0.98	7.8	44	0.94	7.4
4		49	1.01	5.7	46	0.85	4.7
5	a	28	0.58	4.0	31	0.65	3.8
	b	21	0.55	6.0	41	0.88	5.2
	c	19	0.44	2.8	45	0.83	4.6

Table 69. EXERDOP Values for Subject 27.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		21	0.68	5.9	15	0.59	5.4
1	a	39	0.84	7.6	31	0.86	8.6
	b	37	0.84	7.6	33	0.90	8.8
	c	28	0.77	7.2	29	0.85	8.2
2		33	0.75	7.1	27	0.75	6.9
3		43	0.87	7.2	44	0.93	7.4
4		40	0.71	5.9	42	0.86	6.3
5	a	42	0.86	6.2	47	0.95	6.8
	b	44	0.97	7.4	54	1.04	6.5
	c	45	0.89	6.3	50	0.99	6.4
6		36	0.69	4.4	24	0.47	2.5

Table 70. EXERDOP Values for Subject 28.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		13	0.54	4.9	21	0.83	8.3
1	a	26	0.97	10.2	38	1.10	12.0
	b	35	1.06	11.2	40	1.16	12.0
	c	29	1.02	11.0	51	1.27	13.9
2		27	0.85	8.8	41	1.05	11.7
3		50	1.21	11.5	54	1.18	12.2
4		39	0.99	8.8	51	1.18	10.0
5	a	66	1.29	10.4	58	1.19	11.6
	b	76	1.29	10.4	67	1.20	10.6
	c	66	1.17	9.0	49	1.14	10.1
6		56	1.13	6.7	45	0.86	5.1
7		67	1.31	8.6			

Table 71. EXERDOP Values for Subject 29.

Stage	Meas.	Test PkA	Test PkV	Test SVI	Retest PkA	Retest PkV	Retest SVI
0		13	0.50	4.9	11	0.44	4.7
1	a	22	0.73	7.7	18	0.69	7.2
	b	19	0.78	8.9	17	0.69	7.2
	c	20	0.81	9.8	19	0.73	8.0
2		21	0.83	9.1	22	0.85	9.2
3		25	0.90	9.3	27	0.94	9.0
4		42	0.92	7.3	44	0.94	7.7
5	a	48	0.87	5.6	48	0.95	6.9
	b	42	0.91	6.4	51	0.97	6.7
	c	50	0.96	7.2	50	0.89	5.8

Table 72. EXFRDOP Values for Subject 30.

Stage	Meas.	Test pk A	Test pk V	Test pk SD	Retest pk A	Retest pk V	Retest pk S
0		17	0.67	8.1	14	0.54	5.3
1	a	21	0.71	8.0	20	0.82	9.0
	b	15	0.60	6.8	20	0.78	8.6
	c	18	0.63	7.0	21	0.79	8.8
2		26	0.85	9.1	23	0.85	9.6
3		30	0.83	8.8	27	0.87	8.4
4		34	0.84	7.4	40	0.91	7.3
5	a	50	0.89	5.4	43	0.88	4.4
	b	37	0.72	3.8	49	1.03	6.1
	c	35	0.68	5.6	51	1.00	6.2
6		26	0.59	3.8	36	0.78	4.6

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