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The effects of interval resistance and interval aerobic exercise on oxygen pulse

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THE EFFECTS OF INTERVAL RESISTANCE AND INTERVAL AEROBIC
EXERCISE ON OXYGEN PULSE

A Thesis

Presented to

The Faculty of the Department of Human Performance

San Jose State University

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

by

Diane Augustine

May 2003

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ABSTRACT

THE EFFECTS OF INTERVAL RESISTANCE AND INTERVAL AEROBIC EXERCISE ON OXYGEN PULSE

By Diane E. Augustine

Oxygen pulse was measured during interval resistance and interval aerobic exercise. Oxygen pulse, defined as oxygen uptake per heart beat, is an indicator of stroke volume, and its rise may increase cardiovascular efficiency. The heart rate corresponding to each participant's 50% 1 repetition maximum during the interval resistance exercise was determined and maintained during the interval aerobic exercise. Oxygen pulse values were compared during the interval resistance and aerobic exercise and rest periods. Overall, oxygen pulse did not differ significantly between interval resistance and interval aerobic exercise. However, oxygen pulse values increased from rest to exercise during aerobic exercise, while the values decreased from rest to exercise during resistance exercise.

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CHAPTER I

INTRODUCTION

The concept of combining resistance and aerobic exercise into one training session developed to allow individuals to achieve the benefits of both aerobic and resistance exercise without the time commitment of separate programs. Interval resistance training, a program of moderate weight exercises consisting of 12 to 15 repetitions at 50% one repetition maximum (1RM) with 30 s rest between exercises, is the manifestation of this idea (Gettman & Pollock, 1981).

Strength exercise benefits peripheral musculature and other soft tissues in many ways, yet its effect on cardiac morphology and function is unclear (Cohen & Segal, 1985; Fleck & Falkel, 1986; George, Batterham, & Jones, 1998; Morganroth, Maron, Henry, & Epstein, 1975; Tesch, 1988; Urhausen & Kindermann, 1989, 1999; Urhausen, Monz, & Kindermann, 1996, 1997; Willoughby, Green, Sebesta, & Crouse, 1993). Previous work has focused on comparing physiological adaptations to exercise between traditional strength athletes and traditional aerobic athletes, supporting the belief that while strength exercise does cause hypertrophy of the peripheral musculature, it does not cause cardiac hypertrophy nor increase cardiovascular function to the extent of aerobic exercise (Gettman & Pollock, 1981; Haennel, Teo, Quinney, & Kappagoda, 1989; Kraemer, Deschenes, & Fleck, 1988). Since traditional strength exercise is viewed as having little effect on aerobic capacity, it has been neglected in programs that are focused on improving aerobic fitness.

The increase of oxygen pulse (O_2 pulse) during exercise has been identified as a possible mechanism to induce cardiac hypertrophy and, therefore, increase cardiovascular function. Oxygen pulse, oxygen uptake per heart beat, is calculated by dividing oxygen uptake (VO_2) by heart rate (HR), and is a valid predictor of stroke volume (Bhambhani, Norris, & Bell, 1994; Whipp, Higgenbotham, & Cobb, 1996). An increase in stroke volume at a given HR signifies greater cardiovascular fitness and efficiency; therefore, O_2 pulse can be used to determine cardiac efficiency (Bhambhani et al.). Interval aerobic programs, aerobic exercise intervals of 30 s to 5 min with equal or less rest, provoke a higher O_2 pulse response than continuous aerobic programs (Perry, Mosher, La Perriere, Roalstad, & Ostrovsky, 1988). Also, athletes participating in interval aerobic training traditionally display greater heart enlargement, or cardiac hypertrophy, as well as an increase in heart function as a result of this type of physical exercise (“The athlete’s heart”, 1996; DiBello et al., 1996; Morganroth et al., 1975; Perry et al; Richey & Brown, 1998; Snoeckx et al., 1982; Urhausen & Kindermann, 1999; Urhausen et al., 1996, 1997).

While traditional strength exercise may fail to cause cardiovascular adaptation, it is possible that interval resistance exercise may increase O_2 pulse through a similar mechanism as interval aerobic exercise. Indeed, interval strength exercise has induced small increases in aerobic capacity, possibly through a higher O_2 pulse response than traditional resistance exercise (Gettman & Pollock, 1981; Kraemer et al, 1988). Therefore, interval resistance exercise may influence cardiac hypertrophy. A program that increases the O_2 pulse response during exercise may induce cardiac hypertrophy, possibly leading to greater cardiovascular function. The ability to maintain or increase

cardiovascular function through interval resistance exercise could transform rehabilitation practices as individuals forced to suspend aerobic activity may delay both cardiovascular detraining and muscle atrophy. Moreover, interval resistance exercise may be used in conjunction with aerobic exercise to cause even greater cardiovascular and muscular gains. Incorporating resistance and aerobic exercise supports current exercise programming recommendations for increasing functional ability in special populations, such as individuals participating in cardiac and pulmonary rehabilitation. In fact, interval resistance exercise with 10 to 15 repetitions at 40 to 50% 1 repetition-maximum (1RM), or the maximum weight that can be lifted, is recommended by the American College of Sports Medicine (ACSM) to increase strength in patients with coronary conditions as early as 6 weeks after surgery (Moore & Roberts, 1997).

The purpose of this study was to observe the O_2 pulse response during interval resistance and aerobic exercise. The testing program used each individual's calculated HR average corresponding to interval resistance exercise at 50% of 1RM. This intensity was maintained during interval aerobic exercise. Oxygen uptake (VO_2), HR, and O_2 pulse values were recorded continuously.

Statement of Research Hypothesis

At the same submaximal HR, interval resistance exercise will elicit a similar increase in O_2 pulse as interval aerobic exercise.

Delimitations

All participants said they were free of any known chronic or acute disease or infection. All participants were free of anabolic steroids. Participants had participated in

a weight lifting class or personal training during the past 2 years. Participants were not professional body builders or power lifters. All participants were between 18 to 31 years of age. An equal number of females and males participated.

Limitations

The research design attempted to control for sources of invalidity that may have caused the results to be attributed to something other than the exercise modality. Oxygen uptake, HR, and O₂ pulse values were determined and recorded by the Medgraphics metabolic cart every 15 seconds. Oxygen pulse values were averaged over each 30-second rest period and 45-second exercise period. Data validity and reliability depended on the calibration of the metabolic cart, which was calibrated according to the procedures listed in the operator's manual (Medical Graphics Corporation, 2001). The participants were volunteers, possibly indicating a bias toward physical activity that other individuals may not possess, thus reducing the ability to generalize this study to a broader population beyond that represented by the participants. Exercise testing time was reduced to limit fatigue; thus, it only approached true exercise time. Oxygen uptake values did not approach maximum values during the interval aerobic exercise; therefore, the aerobic exercise portion of the study was not an accurate representation of high intensity interval aerobic exercise.

Assumptions

The participants understood the directions for performing the resistance and aerobic exercise. The participants followed the pretest guidelines. The participants answered the medical history form honestly and to the best of their knowledge. The exercise and rest

intervals represented a real interval training protocol. The interval resistance exercise protocol represented the type of program used when interval resistance training. The participants performed to the best of their ability. The exercise intensity typified the exertion commonly experienced during interval resistance training.

Definition of Terms

Aerobic training: exercise training that causes cardiovascular overload, increasing stroke volume and cardiac output, improving aerobic capacity; exercise using specific muscles to increase the cardiovascular system's ability to circulate more blood as well as to increase the muscular system's ability to aerobically generate more ATP (McArdle, Katch, & Katch, 1996).

Cardiac output: the volume of blood pumped by the heart, or arterial blood flow per minute; the product of stroke volume and heart rate (McArdle et al., 1996).

Cardiac hypertrophy: the enlargement of the heart, either in left ventricular internal cavity (eccentric), or wall thickness (concentric) to compensate for stretching and thinning of the ventricular wall as a result of physical exercise (Morganroth et al., 1975; Richey & Brown, 1998; Snoeckx et al., 1982).

End-diastolic volume: left ventricular blood capacity at the end of the diastolic phase of the cardiac cycle (McArdle et al., 1996).

HR rest: resting heart rate (ACSM, 2000).

Heart rate reserve: $HR_{max} - HR_{rest}$ (ACSM, 2000).

Interval aerobic exercise: aerobic exercise intervals of 30 s to 5 min with equal or less rest (Baechle & Earle, 2000; Snoeckx et al., 1982).

Interval resistance exercise: a program of moderate weight exercises consisting of 12 to 15 repetitions at 50% 1RM with 30 s rest between exercises (Gettman & Pollock, 1981).

Lean body mass: body weight minus fat weight (Foss & Keteyian, 1998).

Law of Laplace: an increase in left ventricular wall thickness to reduce the strain associated with an increase in ventricular internal diameter (Urhausen & Kinderman, 1989).

Maximum oxygen uptake (VO₂max): The point at which O₂ consumption by the body ceases to increase as workload increases. This point or score reflects the capacity for ATP resynthesis under aerobic conditions (McArdle et al., 1996).

O₂ pulse: oxygen uptake per heart beat. This value is calculated by dividing VO₂ by HR (Whipp et al., 1996).

Rate pressure product: product of systolic blood pressure and HR (McArdle et al., 1996).

Strength training: a method of conditioning to increase the ability to exert force (Baechle & Earle, 2000).

Target heart rate (THR): a HR range often used to prescribe exercise intensity. The Karvonen method uses both HR rest and HR max to determine THR.

$$THR = [(HR \text{ max} - HR \text{ rest}) \times (\% \text{ intensity})] + HR \text{ rest}$$
 (McArdle et al., 1996).

Operational Definition of Terms

For this study, terms were defined as follows.

Beginning weight lifting experience: determined by current or previous enrollment in weight-training classes and/or personal training, but not including professional body builders or power lifters. Participants displayed correct lifting biomechanics.

HR max: the maximum rate the heart is able to achieve. This can be estimated by using the age-predicted equation: $220 - \text{age in years}$ (ACSM, 2000).

Resistance exercise: “A specialized method of conditioning that involves the progressive use of resistance to increase one’s ability to exert or resist force” (Baechle & Earle, 2000, p. 170).

CHAPTER II

REVIEW OF LITERATURE

Cardiovascular adaptation as a result of aerobic exercise is well understood, including the ability to increase VO_2 max and induce cardiac hypertrophy. The effect of strength exercise on cardiovascular adaptation is less clear, and warrants a closer look. Understanding previous research regarding physiological adaptations to aerobic and strength exercise may enable one to manipulate strength exercise to increase cardiovascular fitness, and thus induce cardiac hypertrophy.

Aerobic training causes an increase in blood volume which, in turn, increases the end-diastolic volume of the left ventricle as more blood is returned to the heart. The larger blood volume must then be pumped back into the circulation, causing an increase in stroke volume and cardiac output (DiBello et al., 1996). An increase in stroke volume at a given HR signifies greater cardiovascular fitness and efficiency (Bhambhani et al., 1994). Aerobic training also decreases resting and submaximal HR via down-regulation of the sympathetic nervous system (DiBello et al.). The combined effects of a decrease in HR and an increase in cardiac output indicate increased cardiovascular efficiency. An increase in O_2 pulse is associated with an increase in stroke volume and, thus, is also a valid estimate of cardiovascular efficiency (Bhambhani et al.; Thomas & Londeree, 1989; Whipp et al., 1996). Aerobic training increases O_2 pulse, as well as causes 10 to 30% increases in VO_2 max (ACSM, 2000), while traditional strength training has failed to elicit an equivalent response (Willoughby et al., 1993).

Although studies on strength training have failed to show cardiovascular adaptation, strength training does elicit many positive changes in the human body, in addition to increases in muscle strength. These changes include increases in bone mineral density and mass (Fleck & Falkel, 1986; Pruitt, Jackson, Bartels, & Lehnhard, 1992; Pruitt, Taaffe, & Marcus, 1995; Stone, 1988), increased connective tissue health (Fleck & Falkel; Kraemer et al., 1988), and favorable body composition changes (Kraemer et al.; Mayhew & Gross, 1974).

Muscle hypertrophy and strength gains from strength training are widely accepted (Tesch, 1988). Although strength training increased strength up to 50% in college-aged participants (Kraemer et al., 1988; Mayhew & Gross, 1974), neurological adaptations were the greatest contributor to strength gains in the first 3 to 4 weeks of training (Bell & Wenger, 1992; Kraemer et al.). An increase in lean body mass from strength training is accompanied by a reduction in fat mass (Kraemer et al.; Mayhew & Gross, 1974). Strength training increases tendon and ligament thickness, weight, and strength, thus reducing injury occurrence and decreasing connective tissue healing time (Fleck & Falkel, 1986; Kraemer et al.; Stone, 1988).

Intensity and duration are major factors in differentiating between gains in strength, endurance, and power (Costill, Coyle, Fink, Lesmes, & Witzmann, 1979; Dudley, 1988; Tesch, 1988). One factor that may explain why strength training has little influence on VO_2 max is the rest periods between sets in traditional strength training. The cessation of exercise may cause too dramatic a reduction in HR, lowering the intensity of the total program below that required to induce aerobic change (Gettman &

Pollock, 1981). Another factor is the difference in the methods used to determine VO_2 max between strength and aerobic athletes (Urhausen & Kindermann, 1999). Some studies tested aerobic athletes' VO_2 max by treadmill or upright bicycle, while strength trained athletes' values were determined by supine bicycle (Urhausen & Kindermann, 1989). Sagiv, Rotstein, Watkins, Climor, and Ben-Sira (1992) noted that O_2 pulse is lower when the body is in a supine rather than an upright position while performing the same exercise, indicating a discrepancy in physiological responses due to body position. Therefore, VO_2 max values from supine exercise may not represent the same values as upright exercise, misrepresenting a possible cardiovascular adaptation achieved through strength training at the appropriate intensity.

Interval Strength Training: Strength Training with Cardiovascular Benefits

Interval strength training is a specific strength exercise program that can improve strength 7 to 32%, as well as induce aerobic improvement through the manipulation of the work-to-rest ratio (Gettman & Pollock, 1981; Kraemer et al, 1988). Interval strength training is a sequence of strength exercises using 40 to 60% 1RM for 12 to 15 repetitions with little rest between exercises (Gettman & Pollock; Haennel et al., 1989). This system contrasts with traditional strength exercise, which dictates that the person performs multiple sets of one exercise before moving to another exercise. In interval strength exercise, the movement of the person to the next exercise and set up of the desired weight is considered the rest interval, and lasts 15 to 30 seconds, thus reducing the total duration of the workout (Gettman & Pollock; Haennel et al.). After a 12-week interval strength program of 15 exercises at 40% 1RM for 30 s work and 15 s rest, young men increased

their VO_2 max 6%, while the young women increased their VO_2 max 5%, and both groups increased muscular strength 10% (Gettman & Pollock). A 9-week study of low fit males performing nine exercises, alternating upper and lower body musculature at 70 to 85% heart rate reserve, noted a 9% improvement in aerobic capacity (Haennel et al.).

Interval strength exercise causes increases in strength comparable to those achieved through traditional strength exercise, yet fails to cause similar improvements in aerobic capacity as traditional aerobic programs (Gettman & Pollock, 1981). Since intensity and duration are both important factors in causing cardiovascular improvement (ACSM, 2000), the results from interval strength exercise may be attributed to the intensity of the protocol. Manipulating the work-to-rest ratio may cause aerobic improvements. More than 30 seconds of rest does not allow aerobic improvement (Gettman & Pollock), possibly due to an excessive decrease in HR during the rest interval. Women exercising at a higher percent of their VO_2 max and HR max, 47% and 88% respectively, showed an 11% improvement in VO_2 max, while men who were training at a lower percent of their VO_2 max and HR max, 41% and 78%, failed to show any improvement (Gettman & Pollock). The 34% higher initial fitness of the men compared to the women may explain the men's failure to increase aerobic fitness. Since the men had a greater initial fitness level, the absolute workload at which they were training represented a lower percent of their VO_2 max. Therefore, the men might have required a higher workload to induce aerobic improvement (Gettman & Pollock). As with aerobic exercise, interval strength exercise must maintain HR and VO_2 in the aerobic training zone to improve cardiovascular fitness, which corresponds to 40 to 85% heart

rate reserve (Moore & Roberts, 1997). Reducing the rest between strength exercises would maintain the higher HR, thus stimulating aerobic adaptation (Gettman & Pollock).

Traditional aerobic exercise, especially high intensity, is believed to cause the greatest improvements in aerobic fitness, yet interval strength exercise, as previously cited, has also resulted in small increases in VO_2 max and strength. This may be a result of the shortened rest periods and, therefore, higher aerobic intensity than traditional strength training. (Gettman & Pollock, 1981; Haennel et al., 1989). The combination of physical benefits from interval strength exercise promotes positive body composition changes favoring the increase of lean body mass and the reduction of fat mass (Gettman & Pollock; Haennel et al.). Furthermore, establishing an interval strength protocol that increases aerobic and strength fitness would allow the prescription of individualized programs that further manipulate the exercise as well as rest duration. Still to be determined are the cornerstones of interval strength exercise that will elicit the desired aerobic adaptation. Combining aerobic capacity and muscular strength improvements in a 30-min workout makes interval strength exercise a valuable alternative to the traditionally longer aerobic and strength programs.

Exercise-Induced Eccentric and Concentric Cardiac Hypertrophy

In addition to other physiological adaptations, nonpathologic cardiac enlargement, or hypertrophy, is hypothesized to be a result of physical exercise (Snoeckx et al., 1982). To maintain or enhance cardiac function during repeated bouts of strenuous activity, the heart undergoes a physiological remodeling process that results in hypertrophy of the cardiac muscle (Fleck, 1988; Kraemer et al., 1988; Morganroth et al., 1975; Richey &

Brown, 1998). Exercise-induced cardiac hypertrophy is a concurrent increase in wall thickness, cavity size, and heart function (“The athlete’s heart”, 1996; DiBello et al., 1996; Richey & Brown; Urhausen & Kindermann, 1999; Urhausen et al., 1997).

Sustained activity over a period of time may be required before significant hypertrophy is noted, since predominantly high caliber athletes develop hypertrophied hearts (“The athlete’s heart”; Ricci et al., 1982; Shapiro, 1984; Snoeckx et al.; Urhausen & Kindermann).

Although athletes display a greater wall thickness, internal heart dimension, and left ventricular mass when compared to nonathletes, determining the specific influence of either aerobic or strength exercise results in ambiguous findings (Abinader, Sharif, Sagiv, & Goldhammer, 1996; Cohen & Segal, 1985; DiBello et al., 1996; George et al., 1998; Shapiro, 1984). An increase in preload, or venous return, from an increased blood volume results in an increase in left ventricular cavity dimension, or eccentric hypertrophy. Furthermore, researchers have typically hypothesized that an increase in afterload, or peripheral resistance, results in an increase in left ventricular wall thickness, namely concentric hypertrophy (Abinader et al.; “The athlete’s heart”, 1996; Richey & Brown, 1998; Urhausen et al., 1997).

Aerobic exercise requires the left ventricle to accommodate a larger blood volume while maintaining a large cardiac output for a prolonged time period. Over time, the ventricle’s end-diastolic volume increases, which is observed as eccentric hypertrophy (Abinader et al., 1996; Agati, Fedele, Gagliardi, Sciomer, & Penco, 1985; “The athlete’s heart”, 1996; Cohen & Segal, 1985; Di Bello et al., 1996; Fleck, 1988; Morganroth et al.,

1975; Richey & Brown, 1998; Roy, Doyon, Dumesnil, Jobin, & Landry, 1988; Shapiro, 1984; Snoeckx et al., 1982; Urhausen & Kindermann, 1989, 1999; Urhausen et al., 1997; Wisloff, Helgenid, Stoylen, & Ellingsen, 2001). Studies have further demonstrated that some aerobic athletes display concentric hypertrophy when compared to nonaerobic athletes (Abinader et al.; DiBello et al.; Snoeckx et al.; Urhausen et al., 1997). Studies have revealed increased left ventricular mass in runners as a compensation for the increased end-diastolic volume (Abinader et al.), including increased septum thickness and left ventricular mass indices (DiBello et al.). Other studies have also found increased left ventricular posterior wall thickness in runners and cyclists when compared to nonaerobic athletes (Snoeckx et al.). Similarly, Urhausen et al. (1997) summarized findings stating that aerobic athletes display a concurrent increase in ventricular wall thickness and internal diameter, with wall thickness typically being higher in relation to internal diameter. These findings can be explained according to the Law of Laplace, as an increase in blood volume entering the heart causes the cardiac muscle walls to stretch. The stretching stimulates the muscle to increase the wall thickness in an effort to compensate for the increased strain, thus preventing muscle damage. As the heart volume increases, the thickness of the walls will increase as well (Urhausen & Kindermann, 1989). Left ventricular mass and mass indices were similar in aerobic and nonaerobic athletes (Abinader et al.), and both were greater than sedentary controls (DiBello et al.). Furthermore, maximum rate pressure product, an indication of myocardial oxygen uptake during maximal exercise, is not significantly different between aerobic and strength-trained athletes of similar heart size. This suggests that myocardial demand, and thus

hypertrophy, may be similar between strength and aerobic athletes of similar heart size (Willoughby et al., 1993).

An increased heart volume in athletes causes an increase in the stroke volume and, therefore, a greater VO_2 max than that of a sedentary person (DiBello et al., 1996; Snoeckx et al., 1982; Urhausen & Kindermann, 1989). Although aerobic athletes consistently display larger end-diastolic volumes than both strength trained and sedentary individuals (Morganroth et al., 1975; Snoeckx et al.), the data for strength athletes varies. Some studies noted that recreational and heavy weight lifters have slightly higher VO_2 max values than sedentary values (Yeater, Reed, Ullrich, Morise, & Borsch, 1996), while others determined that body builders display VO_2 max values within the sedentary range (Urhausen & Kindermann).

Some researchers believe that pressure overload causes concentric left ventricular hypertrophy since strength athletes display greater hypertrophy of the ventricular wall than aerobic athletes (Agati et al., 1985; Cohen & Segal, 1985; Menapace et al., 1982; Morganroth et al., 1975; Richey & Brown, 1998; Roy et al., 1988). During heavy strength exercise, a brief period of increased systolic blood pressure requires the ventricular muscle to overcome high peripheral resistance to maintain blood delivery to the working muscles. Therefore, the high pressure is hypothesized to stimulate an increase in ventricular wall thickness (Morganroth et al.). Other researchers agree that while the pressure stimulus could cause concentric hypertrophy, the blood pressure increase during exercise is too transient to stimulate morphological change (Fleck, 1988; Shapiro, 1984). Instead, researchers believe the influence of strength exercise on cardiac hypertrophy is

more attributable to gains in lean body mass (Abinader et al., 1996; Fleck; “The athlete’s heart”, 1996; George et al., 1998; Kraemer et al., 1988; Morganroth et al.; Ricci et al., 1982; Richey & Brown; Shapiro; Urhausen et al., 1997). Indeed, strength exercise causes an increase in peripheral skeletal muscle, which is matched by an increase in left ventricular muscle mass and internal diameter (Urhausen & Kindermann, 1989). Although studies report cardiac hypertrophy in strength athletes, the increase of lean body mass is believed to be the primary mechanism influencing cardiac morphological change in these athletes (Urhausen & Kindermann, 1999).

The underlying influence of body dimension on heart size led many researchers to disregard previous findings of hypertrophy with strength exercise (Urhausen & Kindermann, 1989; Urhausen et al., 1997). Similarly, Wolfe, Martin, and Seip (1985) reported that basketball player somatotype, rather than exercise, might have influenced cardiac hypertrophy. Therefore, the amount of mesomorphism, body surface area, and lean body mass should all be taken into consideration prior to interpreting the results of cardiac hypertrophy studies (Brown & Thompson, 1987; Shapiro, 1984; Urhausen et al., 1997; Wisloff et al., 2001). Similarly, athletes using anabolic steroids display the greatest cardiac hypertrophy (Di Bello et al., 1999; Ullrich, Younis, Yeater, Borsch, & Zanke, 1990; Urhausen & Kindermann, 1999; Yeater et al., 1996); therefore, cardiac hypertrophy induced by strength training alone may not be as common as once believed (Roy et al., 1988; Urhausen & Kindermann, 1999). Although athletes have greater cardiac hypertrophy than nonathletes, anthropometric influences must be considered when attempting to distinguish the effects of specific training techniques on cardiac

hypertrophy (Brown & Thompson; Fleck, 1988; George et al., 1998; Shapiro; Richey & Brown, 1998; Ullrich et al., 1990; Urhausen & Kindermann, 1989, 1999; Urhausen et al., 1997).

The definitive cause of cardiac hypertrophy from strength and aerobic exercise is still unknown (Urhausen et al., 1997). Studies have failed to control participants' exercise protocols, with some participants involved in combined strength and aerobic exercise, such as wrestlers, rowers, soccer players, and triathletes (Agati et al., 1985; Fleck, 1988; Menapace et al., 1982; Shapiro, 1984; Urhausen & Kindermann, 1989, 1999; Urhausen et al., 1997). Athletes participating in combined exercise programs may increase both their lean body mass and blood volume, leading to the inability to distinguish the individual influences of strength or aerobic exercise on cardiac hypertrophy. Since both strength and aerobic exercise may result in similar cardiac morphological adaptation (Brown & Thompson, 1987; Shapiro), intensity rather than specific training modality has been explored (Snoeckx et al., 1982).

Oxygen Pulse

Cardiac muscle responds to exercise intensity regardless of exercise type (Shapiro, 1984). Low intensity strength exercise does not cause significant cardiac hypertrophy (Ullrich et al., 1990; Van Den Broeke & Fagard, 1988). Yet, aerobic exercise produces a larger maximum O₂ pulse than power lifting, a traditionally high intensity strength program (Willoughby et al., 1993). This introduces the possibility that the HR during the strength workout session may be less than that for aerobic exercise (Roy et al., 1988; Shapiro, 1984; Snoeckx et al., 1982). A lower overall intensity may explain why some

strength athletes display normal sized hearts for their body weight and perform aerobically within the limits of sedentary controls, while aerobic athletes with hypertrophied hearts for their body weight perform aerobically above average (Hurley et al., 1984; Snoeckx et al.; Urhausen & Kindermann, 1989).

According to the Law of Laplace, an increase in O_2 pulse stimulates cardiac enlargement through increased venous return (Urhausen et al., 1996). Interval aerobic programs, such as those consisting of 10, 30 s sprints with 15 s rest (Snoeckx et al., 1982), cause a more pronounced rise in O_2 pulse than continuous exercise at the same HR, thus eliciting a greater VO_2 max increase (Perry et al., 1988). Furthermore, athletes who participate in high intensity interval aerobic training have more pronounced left ventricular hypertrophy than those in continuous programs (Perry et al.; Snoeckx et al.; Urhausen et al., 1996). Oxygen pulse has also been discovered to increase during the short rest periods following intense exercise commonly associated with ball games, causing more pronounced hypertrophy in soccer players than 400 m runners (Urhausen et al., 1996; Urhausen & Kindermann, 1999). Yet other studies of ball players show ambiguous findings, as basketball players do not display cardiac hypertrophy for their body size (Wolfe et al., 1985). Unfortunately, the type, intensity, and duration of training practices for these athletes are unknown, preventing conclusions regarding O_2 pulse and subsequent morphological change. Nevertheless, a program that increases O_2 pulse may stimulate cardiac hypertrophy, regardless of the mode of training.

Interval resistance exercise induces small increases in stroke volume for a given HR, indicating a cardiac adaptation that is related to O_2 pulse (Haennel et al., 1989).

Similarly, interval resistance exercise is able to maintain cardiovascular fitness after the cessation of a running program (Gettman & Pollock, 1981), suggesting that interval resistance exercise can be manipulated to elicit intensity sufficient to prevent detraining. Yet interval resistance exercise causes aerobic improvement that is consistently less than that from traditional aerobic exercise (Gettman & Pollock). An explanation may be the influence of the sympathetic nervous system on HR. Parker, Hurley, Hanlon, and Vaccaro (1989) noted that higher HRs were required during aerobic dance to reach a specific oxygen uptake value (VO_2) than during jogging. The discrepancy between VO_2 and HR during aerobic dance may be attributed to a higher sympathetic response to the smaller muscle mass involved with arm movements (Parker et al.; Taguchi & Horvath, 1987). Since arm exercise produces higher heart rates than leg exercise at similar VO_2 values (Parker et al.; Taguchi & Horvath), and more muscle mass causes a higher O_2 pulse at the same HR (Thomas & Londeree, 1989), HR alone may not be an accurate representation of exercise intensity when the exercise involves both arm and leg movement. Yet at the same absolute workload, arm exercise produces higher O_2 pulse values (Taguchi & Horvath), probably due to the higher intensity required for the smaller arm muscles to do the same work as the larger leg muscles (Parker et al.). Since interval resistance exercise relies on both upper and lower body muscles, it is possible that the target heart rate for interval resistance exercise stimulates a lower O_2 pulse than the same HR during aerobic exercise. The consistently lower O_2 pulse during interval resistance exercise would explain the lower VO_2 max gains. The behavior of O_2 pulse during all aspects of interval resistance exercise is still unknown; therefore, whether interval

resistance exercise causes an adequate increase in O_2 pulse to induce cardiac hypertrophy is likewise unclear. Determining the O_2 pulse response at the HR commonly used during interval resistance training will allow a comparison of the aerobic response between interval resistance and aerobic exercise to decide whether interval resistance exercise at that intensity is likely to stimulate cardiac hypertrophy. Furthermore, determining O_2 pulse behavior during this activity will allow HR adjustment to better elicit aerobic improvement. Athletes and trainers would then be able to monitor and maintain the intensity that would stimulate increases in O_2 pulse and, thus, cardiovascular adaptation.

Conclusion

The body adapts to exercise by reducing the relative amount of strain placed on the heart, while maintaining adequate blood delivery to the working muscles. Cardiac enlargement, either in cavity dimension or wall thickness, is compensation for an increased load experienced by the heart (Kraemer et al., 1988; Richey & Brown, 1998). The influence of training on cardiac adaptation has been a concern of many researchers. Maximal oxygen uptake (VO_2 max) is a valid measure for assessing aerobic ability (DiBello et al., 1996), and is influenced by the size of the left ventricular cavity. A larger left ventricular cavity translates into greater stroke volume, and a larger cardiac output, thus increasing cardiovascular ability (Morganroth et al., 1975; Shapiro, 1984). A long-held belief has been that cardiac morphological change depends on the type of exercise (Agati et al., 1985; Brown & Thompson, 1987; Fleck, 1988; Morganroth et al.; Snoeckx et al., 1982). Since exercise intensity is a major influence on cardiac functional and morphological adaptation (Shapiro; Van Den Broeke & Fagard, 1988), determining the

intensity that induces these changes would streamline exercise programming, ultimately benefiting athletic performance.

An intensity-driven interval resistance exercise program designed to manipulate both upper and lower body large muscles in a rhythmic, strength-building fashion should bridge the gap that separates traditional strength exercise from traditional aerobic exercise. Interval resistance exercise is a specific program that has demonstrated an ability to induce some aerobic improvements, possibly through the manipulation of O_2 pulse (Kraemer et al, 1988). Although aerobic improvement has been less than that after traditional aerobic exercise, the combination of improvements in aerobic capacity as well as strength makes interval resistance exercise a valuable, timesaving alternative to traditional aerobic and strength exercise programs. Furthermore, once the behavior of O_2 pulse is determined during interval resistance exercise, the intensity and/or duration of the exercise can be adjusted to elicit or maintain a rise in O_2 pulse to stimulate left ventricular hypertrophy. Oxygen pulse is a tool that can differentiate cardiovascular functional ability and exercise-induced versus pathological cardiac hypertrophy (Whyte, Sharma, George, & McKenna, 1999). Therefore, a rise in O_2 pulse during interval resistance exercise similar to that observed during interval aerobic exercise would indicate increased cardiovascular function, and potentially indicate exercise-induced cardiac hypertrophy.

CHAPTER III

METHODS

The purpose of this study was to determine the magnitude of oxygen pulse increase as a result of interval resistance exercise, and to compare the results with the oxygen pulse response of interval aerobic exercise at the same heart rate. Since O_2 pulse is an indicator of stroke volume, information about O_2 pulse behavior during exercise may help clarify the effects of resistance exercise on aerobic fitness and cardiac hypertrophy.

Pilot Study Data Collection

The Institutional Review Board approved this study on October 28, 2002 (Appendix A). After approval, two participants were tested for 1RM on the latissimus pull-down and seated leg press (ACSM, 2000). Each participant performed a light warm-up of 5 to 10 repetitions at 40 to 60% perceived maximum on the latissimus pull-down. The latissimus pull-down bar was lowered to chin level. Following 1 min rest with light stretching, the participant performed three to five repetitions at 60 to 80% of perceived maximum. After adding 10 to 20 lb (4.5 to 9 kg), a 1RM lift was attempted. If the lift was successful, 3 to 5 min of rest were provided. The process of adding 10 to 20 lb was continued until the participant failed to lift the weight. The 1RM was the last completed lift. The goal was to find the 1RM within three to five maximum efforts (ACSM, 2000). The determination of the 1RM for the seated leg press was done in the same manner for each participant.

After calculating 50% 1RM for both resistance exercises, the first participant was instructed to lift the 50% 1RM weight for 15 repetitions on the latissimus pull-down machine during which his or her heart rate and VO_2 were recorded via the Medgraphics CPX/D metabolic cart. This instrument was previously determined to produce reliable and valid data by both Jones and Wasserman (as cited in Medical Graphics, 1996). The flow device has an accuracy of $\pm 3\%$ or 50 ml, whichever is greater, meeting American Thoracic Society and European Respiratory Society clinical performance standards (Medical Graphics, 2001). The oxygen analysis has an accuracy of $\pm .03\%$, and the carbon dioxide analysis has an accuracy of $\pm .05\%$. The metabolic cart measured oxygen consumption (VO_2 in ml/min and ml/kg/min), carbon dioxide production (VCO_2 in ml/min), and expired ventilation (V_e), recorded heart rate (HR), and calculated respiratory exchange ratios (VCO_2/VO_2) and oxygen pulse (VO_2/HR). Oxygen uptake was also recorded during the 30 s rest period while the participant advanced to the seated leg press machine. After 30 s, the participant lifted 50% 1RM for 15 repetitions during which his or her heart rate and VO_2 were recorded. This interval resistance exercise at 50% 1RM for 15 repetitions with 30 s rest continued for three circuits. The other participant completed the same testing with the same data collection protocol. The latissimus pull-down was always performed before the seated leg press. After the completion of the interval resistance testing for both participants, individual HRs were averaged over the exercise and rest periods, and were used to determine the average HR to be used for the interval aerobic exercise. Each participant exercised on the treadmill for 45 s, the length of time to complete 15 repetitions, at the HR that he or she experienced during the

resistance exercise. After each interval of treadmill exercise, participants straddled the treadmill to simulate a 30 s rest period. After the rest period, the participants again walked on the treadmill for another 45 s exercise interval. The total exercise time was the same between resistance and treadmill exercise, 7 min and 30 s. Oxygen uptake and HR values were recorded continuously during both exercises. The protocol was reversed on a third day to examine whether resistance exercise affected the O₂ response during aerobic exercise. No order effect was detected. The results of the pilot study were reviewed by an exercise physiologist, and the protocol was revised by increasing the exercise intervals from 30 s to 45 s to allow 15 repetitions with a 1 s concentric lifting phase, and a 2 s eccentric lifting phase.

Interval Resistance and Aerobic Exercise Testing

The heart rate corresponding to 50% 1RM was determined during the interval resistance exercise and was maintained during the interval aerobic exercise. The interval resistance exercise consisted of the latissimus pull-down (upper body resistance exercise) and the seated leg press (lower body resistance exercise).

The participants were familiarized with the exercise protocol and the use of the three exercise machines prior to testing. The first testing session consisted of an explanation of the study, an overview of the exercise modalities, measurement of resting heart rate, height, weight, percent body fat, and 1RM determination as described in the pilot study.

The second testing session included both interval resistance and interval aerobic exercise. Oxygen uptake and HR values at 50% 1RM were determined using the

Medgraphics metabolic cart in the same manner as described in the pilot study. After the interval resistance exercise was completed, the participants were instructed to stretch until their HR lowered to near baseline levels. The participants were then instructed to sit in a chair for 15 min. The participants concluded the testing session by participating in interval aerobic exercise on the treadmill for 7 min and 30 s at the average HR corresponding to 50% 1RM, with 45 s exercise intervals and 30 s of rest between intervals. The O₂ pulse values were averaged for the 7 min and 30 s of exercise and rest for the interval resistance exercise. The O₂ pulse values were similarly averaged for the interval aerobic exercise.

Participants

A sample of healthy, college-age participants, 18 to 31 years of age, was obtained. All participants met ACSM guidelines for low risk individuals, and were free of contraindications that would prohibit submaximal exercise testing (ACSM, 2000; Moore & Roberts, 1997; Appendix B). Participants had at least beginning weight lifting experience determined by current or previous enrollment in weight-training classes and/or personal training, but not including professional body builders or power lifters. Twenty participants were recruited, but only 18 participated in the study, 7 females and 11 males. One participant dropped out of the study due to facial injury that inhibited her from completing the testing. One participant was eliminated from the study due to a faulty electrode causing HR readings that could not be interpreted.

Pretesting

Participants completed a medical and activity history form (Appendix C). Only those participants designated as low risk were allowed into the study. Procedures, risks, and benefits were explained to each participant, after which informed consent was obtained (Appendix D). Consistent with ACSM recommendations (2000), participants were asked to refrain from eating, smoking, and ingesting caffeine 3 hr prior to testing, and to refrain from ingesting alcohol or other drugs, exercising, and using body lotion on the day of testing. They were asked to drink plenty of fluids for 24 hr prior to testing, and to obtain 6 to 8 hr of sleep the night before the test. Participants were asked to wear loose exercise clothing, including a short-sleeved shirt to facilitate blood pressure (BP) assessment, and running shoes.

Day 1

Physiological and Anthropometric Measures

Prior to testing, the participants were asked to change into appropriate clothes and to void their bladders. Participants (dressed in light exercise clothes, but without shoes) were weighed on a calibrated, beam-type scale. Weight was recorded to the nearest 0.1 kg. Height was assessed using a stadiometer that consisted of a vertical board with an attached rule, and a horizontal headboard that was brought into contact with the most superior point on the participant's head. Participants were instructed to stand without shoes in the center of the flat stadiometer with their heels together and weight evenly distributed on both feet. Their feet were spread at a 60° angle, and their head, scapula, buttocks and heels were touching the vertical board. Their arms were placed at their sides

with palms facing thighs. Their head was erect with eyes looking forward. The wooden level was placed on top of the participant's head, and the participant was asked to inhale, maintaining a fully erect position without changing his or her feet. The headboard was lowered onto the most superior point of the head, compressing the hair. Measurements were recorded to the nearest 0.5 cm.

Skinfolds were measured using a Lange skinfold caliper. A calibration block of known thickness was used to check calibration of the caliper. The same test administrator and same caliper were used for all participants. Since every participant was Caucasian, skinfold sites were determined according to the participant's gender and athletic level, and were taken on the right side of the body. The sites were identified using anatomical reference points, marked by a pen, and measured. The skinfold was grasped by the thumb and index finger of the left hand and lifted 1 cm above the site that was measured. The calipers were placed perpendicular to the fat fold and 1 cm below the fingers. The caliper spring was released slowly, and the dial was read to the nearest .1 mm about 4 s after the release. All skinfolds were measured sequentially two times. If the measurements varied by more than 10%, additional measurements were taken. If the measurements varied less than 10%, the two measurements were averaged. Body fat percentage was calculated using the prediction equations for gender and athletic level (Heyward & Stolarczyk, 1996).

Resting HR via a Polar heart rate monitor was obtained prior to testing. Participants sat in an armchair for 15 min prior to recording HR rest. Resting BP was obtained using a mercury sphygmomanometer. Systolic BP was assessed at the first

Korotkoff sound, and diastolic BP was assessed at the fourth Korotkoff sound. If the fourth phase of Korotkoff was not discernible, the fifth phase was used. Appropriate technique was followed for cuff and stethoscope placement, and for sphygmomanometer reading (ACSM, 2000). Participants sat in an armchair for 15 minutes prior to measuring resting BP. Only those participants with resting systolic measures below 200 mmHg and diastolic below 110 mmHg were accepted into the study (ACSM, 2000).

1-RM Testing and HR Response to Treadmill Intensity

As previously described in the pilot study, each participant's 1RM was determined according to ACSM guidelines (2000). To facilitate setting the treadmill on Day 2, each participant walked on the treadmill to determine his or her HR response to varying treadmill intensities. Each participant began by walking at 1.5 mph (2.4 km/hr) until his or her HR response stabilized, at which time the speed was increased to 2.0 mph (3.2 km/hr). After the HR again stabilized, the speed was increased and maintained in this step-wise manner until 3.5 mph (5.6 km/hr) was reached. If the HR response was below 140 bpm, the incline was increased in 1% increments until 140 bpm was achieved or exceeded.

Day 2

Interval resistance exercise

Prior to testing, the participants were asked to change into appropriate clothes and to void their bladders. The instrument used to determine VO_2 and HR was the Medgraphics CPX/D metabolic cart, and was calibrated prior to measurement of the dependent variable to ensure valid data collection. This instrument had been shown to

produce reproducible and consistent data (Medical Graphics, 1996, 2001). Oxygen uptake values were corrected to Standard Temperature and Pressure Dry (STPD) conditions. The pneumotach, used to measure flow rate, was calibrated over a range of flow rates. The O₂ and CO₂ analyzers were calibrated with two gas mixtures containing known concentrations of O₂ and CO₂, representing upper and lower gas concentrations expired by the participant during testing. The sampling line was calibrated to determine the time required for gas to travel from the participant's mouth to the cart. Ten electrodes were placed on the participant's chest to obtain a 12-lead electrocardiogram.

Each participant began with treadmill walking at 3.0 mph (4.8 km/hr) at 0% incline for 5 min as a warm-up. After completing the warm-up, the participant was instructed to dismount the treadmill and to prepare for the resistance testing by placing an air-tight facemask fitted with a calibrated pneumotach and sampling line over his or her mouth and nose. Gas samples were drawn from the participant's exhaled air, and delivered directly to the metabolic cart. A metronome was set to beat every second to regulate the lifting rhythm corresponding to 1 s exertion, the concentric phase, and 2 s relaxation, or the eccentric phase. The 12-lead electrocardiogram wires were attached to the participant's electrodes. The weight was adjusted on the latissimus pull-down machine to the 50% 1RM weight determined by the previous day's testing. Participants were instructed to place their palms in the correct position on the latissimus pull-down bar. The participant was instructed to lift the 50% 1RM weight for 15 repetitions, or 45 s, according to the metronome rhythm. Oxygen uptake and HR values were obtained via the Medgraphics CPX/D metabolic cart throughout exercise. Heart rate and VO₂ values

were also recorded during the 30 s rest periods during which the participant advanced to the seated leg press machine. After the 30 s rest period, the participant lifted 50% 1RM on the seated leg press for 15 repetitions during which his or her HR and VO_2 values were recorded. This interval resistance exercise at 50% 1RM for 45 s with 30 s rest continued for 7 min and 30 s, completing three sets of alternating latissimus pull down and seated leg press exercises with rest intervals. Heart rates for each participant were averaged over the total testing period. The VO_2 and HR values averaged over the interval resistance exercise were used to calculate O_2 pulse. The participant rested sitting in an armchair for 15 min after the completion of the interval resistance exercise, after which he or she began the interval aerobic exercise on the treadmill as described below.

Interval aerobic exercise

Each participant began with treadmill walking at 1.5 mph (2.4km/hr) and 0% incline for 5 min as a warm-up. After 5 min, the speed and the incline were adjusted to elicit the HR corresponding to 50% 1RM as established during the interval resistance exercise. This exercise was maintained for 45 s, after which the participant straddled the treadmill for a 30 s rest period. Once the 30 s rest period was over, the participant was instructed to resume walking for another 45 s. The 45 s of exercise with 30 s of rest continued for 7 min and 30 s, or six sets of alternating aerobic exercise and rest intervals.

Posttest Procedure

Each participant was instructed to perform a cool-down exercise of walking and stretching until HR stabilized near baseline levels. Results were available to the participant, if requested. Average HR over each testing session (interval aerobic and

resistance exercise) allowed the descriptive analysis of relative HR intensity differences between interval resistance exercise and ACSM recommendations for increasing cardiovascular fitness (ACSM, 2000). The following equation was used to calculate the intensity of exercise for each participant: $[(\text{HR average} - \text{HR rest}) / (\text{HR max} - \text{HR rest})] \times 100$ (McArdle et al., 1996, p.266). Maximum HR was estimated using the equation $220 - \text{age in years}$ (ACSM, 2000), HR rest was determined via Polar heart rate monitors on Day 1, and HR average was calculated by averaging the HR response during the interval resistance exercise.

Research Design

One group of 18 participants was tested. Oxygen pulse values for two forms of interval exercise, resistance and aerobic, were compared. As previously determined during the pilot study, no order effect was observed; therefore, the interval aerobic and resistance exercise testing was completed on the same day. The resistance exercise was always performed first to allow the calculation of the average exercise HR, which was used to determine intensity during the interval aerobic exercise. Error associated with lack of familiarity with the exercises was reduced, as the participants had previous experience through either a beginning weight lifting class or personal training, and were able to perform 7 min and 30 s of interval resistance and aerobic exercise. The O₂ pulse values from the arm and leg exercises were averaged over 45 s, and the rest periods were averaged over 30 s. Similarly, the O₂ pulse values for the interval aerobic exercise were averaged over 45 s, and the rest periods were averaged over 30 s. Aerobic efficiency, as measured by O₂ pulse, was attributed solely to the modality of exercise since the

participants were exercising at their individual HR associated with 50% 1RM during interval resistance and aerobic exercise.

CHAPTER IV

RESULTS

Raw data are presented in Appendix E. Mean descriptive data, including age, height, weight, and percent body fat of the participants, are presented in Table 1. Participants were asked to report their current activity level ranging from inactive, “most of your day is spent sitting and you do no regular physical activity”, to very active, “your day requires extensive physical activity and/or you exercise moderately to vigorously for more than 30 minutes, 4 or 5 times each week”. All participants reported the three most active levels; somewhat active ($n = 4$) “your day requires some physical activity and you occasionally are involved in recreational activities (e.g., tennis, golf) or you occasionally do physical activities such as gardening, housecleaning, dancing, etc.”, moderately active ($n = 4$) “your day requires physical activity and/or you participate in regular recreational or fitness activities (walking, jogging, swimming, cycling, weight lifting) for at least 20 minutes, 3 days each week”, and very active ($n = 10$). None of the participants designated “inactive” or “relatively inactive” as their current level of physical activity. The minutes per session of resistance training ranged from 0 to 15 min per session ($n = 4$) to 31-60+ min ($n = 12$). Additionally, the participants reported that they participated in zero to three ($n = 10$), four to five ($n = 4$), or six to seven ($n = 4$) aerobic training sessions per week. The interval resistance exercise elicited a HR response that ranged from 19 to 73% of predicted heart rate reserve ($M = 44\%$, $SD = 14\%$).

Table 1

Mean Descriptive Data

	All Participants		Male ^a		Female ^b	
Variable	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age (years)	24.8	3.4	24.9	3.3	24.6	3.7
% BF	21.5	8.9	16.4	6.5	29.4	5.8
Ht (cm)	171.6	9.8	174.9	10.3	166.4	6.4
Wt (kg)	77.1	15.5	84.8	14.0	65.1	9.1

^a n = 11

^b n = 7

According to previous research in this field, alpha was set at .05. The sample size recommended by power software was 24 when alpha = .05, effect size = .8 to produce a power of .96 for a 2-way ANOVA, but actual sample size was 18, with power = 0.89, critical $F(1,16) = 4.49$, and lambda = 11.52. The 2-way repeated measures ANOVA failed the normality and equal variance assumptions for parametric tests; however, results for practical significance showed that 7.5% of the variance in O₂ pulse was explained by the interaction between exercise modality (aerobic versus resistance) and exercise or rest intervals. Also, 16.4% of the variance in O₂ pulse between arm and leg resistance exercise was attributable to time. Oxygen pulse values for resistance and aerobic interval exercise are presented in Table 2. When plotted, the mean O₂ pulse values between aerobic and resistance modalities during exercise and rest appeared to represent opposite behaviors, as depicted in Figure 1.

To further analyze the behavior of O₂ pulse during resistance and aerobic exercise and rest, three separate paired *t*-tests were performed, adjusting alpha from .05 to .017. The first *t*-test did not find a difference in O₂ pulse between aerobic and resistance exercise when both exercise and rest were averaged for each participant ($t(17) = -1.029$, $p = .32$, and power = .05). The second *t*-test found a statistically significant difference ($t(17) = 3.847$, $p = .001$, and power = .95) between mean aerobic and resistance O₂ pulse exercise values. The last *t*-test did not find a difference when mean O₂ pulse resting values were compared ($t(17) = -2.413$; $p = .027$, alpha = .017, and power = .54).

As depicted in the upper panel of Figure 2, mean O₂ pulse during aerobic and resistance exercise behaved in an opposite manner initially, then leveled off as exercise

Table 2

Mean O₂ Pulse During Two Types of Interval Exercise

Exercise Modality	Exercise Interval		
	Rest + Exercise	Exercise	Rest
Aerobic	10.6 ± 3.4	11.5 ± 3.8	9.8 ± 3.2
Resistance	10.2 ± 3.0	9.6 ± 2.4	10.8 ± 3.6

Values are $M \pm SD$

Figure 1 Mean O₂ Pulse of Aerobic and Resistance Modalities During Exercise and Rest

progressed, with resistance values consistently lower than aerobic values. Although the overall rise and fall of the O₂ pulse values is similar during aerobic and resistance rest intervals, the magnitude of change was less during the aerobic intervals than during the resistance intervals (see Figure 2, lower panel). Oxygen pulse values for aerobic rest are consistently lower than for resistance rest. Although the general trend of O₂ pulse during aerobic exercise was to increase from rest to exercise while the opposite took place for resistance exercise, η^2 for the interaction of aerobic/resistance and exercise/rest was only 7.5%; therefore, practical significance was low.

The upper panel of Figure 3 shows the O₂ pulse behavior during interval resistance exercise. It is interesting to note how O₂ pulse initially rises rapidly and then levels off as exercise progresses. Because O₂ pulse is expressed as oxygen uptake per heart beat, the lower panel of Figure 3 shows the influence of HR and VO₂ during interval resistance exercise. Oxygen uptake and HR values increase rapidly at the onset of exercise, and follow a similar pattern of peaks and troughs as exercise continues. Heart rate appears to be higher than VO₂ values during most of the arm intervals, which would normally translate into lower O₂ pulse values, yet the rise in HR is matched by a rise in VO₂ values for arm intervals that are consistently higher than those for the leg intervals. Also, as exercise progresses, HR and VO₂ increase until steady state is reached.

Figure 2 Oxygen Pulse During Aerobic and Resistance Exercise

Oxygen Pulse During Aerobic and Resistance Rest

Figure 3 Oxygen Pulse During Resistance Exercise

Oxygen Uptake and HR During Resistance Exercise

During the rest intervals, O_2 pulse values are higher following arm exercise, and lower following leg exercise (see upper panel, Figure 4). As shown in the lower panel, VO_2 values increase gradually during rest intervals, while mean HR values display a larger increase during the rest intervals that follow leg exercise than following arm exercise (see Figure 4).

During aerobic exercise, mean O_2 pulse values decrease initially and then level off as exercise continues (see upper panel, Figure 5). Similarly, VO_2 and HR values decrease after the initial exercise interval to level off as the exercise progresses (see lower panel, Figure 5).

During the rest intervals of interval aerobic exercise, O_2 pulse values initially fell, and then maintained a level, steady state value (see upper panel, Figure 6). Oxygen uptake and HR values also fell initially, and then maintained fairly level values, but HR increased slightly more than VO_2 with each rest interval (see lower panel, Figure 6).

The upper panel of Figure 7 displays the rise and fall of O_2 pulse during aerobic exercise and rest intervals. Oxygen pulse increased during the exercise intervals and dropped during the rest intervals. The difference between the peaks and troughs of O_2 pulse was lessened as exercise continued. As shown in the lower panel, VO_2 also increased during the exercise intervals and decreased during the rest intervals, yet as exercise progressed, VO_2 values during the exercise interval slowly decreased as the values during the rest intervals slowly increased. Heart rate, on the other hand, showed an opposite behavior, increasing during rest intervals, and decreasing during exercise intervals.

Figure 4 Oxygen Pulse During Resistance Rest

Oxygen Uptake and HR During Resistance Rest

Figure 5 Oxygen Pulse During Aerobic Exercise

Oxygen Uptake and HR During Aerobic Exercise

Figure 6 Oxygen Pulse During Aerobic Rest

Oxygen Uptake and HR During Aerobic Rest

Figure 7 Oxygen Pulse During Aerobic Exercise and Rest

Oxygen Uptake and HR During Aerobic Exercise and Rest

Oxygen pulse values during interval resistance exercise increased rapidly as exercise was started, then increased and decreased during the 7 min and 30 s exercise session (see upper panel, Figure 8). Oxygen pulse was higher during the rest intervals than during the exercise intervals, but was higher than resting levels throughout the exercise session. Three peaks in the O_2 pulse occurred during the rest intervals following arm exercise (see upper panel, Figure 8). Oxygen uptake increased as the resistance exercise and rest intervals progressed, with VO_2 lower during leg exercise. Similarly, the three most noticeable rises in HR occurred during the arm exercise intervals, and the three most noticeable drops in HR occurred during the rest periods following arm exercise (see lower panel, Figure 8).

Figure 8 Oxygen Pulse During Resistance Exercise and Rest

Oxygen Uptake and HR During Resistance Exercise and Rest

CHAPTER V

DISCUSSION

Previous research supports the thinking that although resistance training does cause hypertrophy of peripheral musculature, it does not cause cardiac hypertrophy nor increase cardiovascular function to the extent of aerobic exercise (Gettman & Pollock, 1981; Haennel et al., 1989; Kraemer et al., 1988). Aerobic training increases O_2 pulse, as well as causes 10 to 30% increases in VO_2 max (ACSM, 2000), while traditional strength training has failed to elicit an equivalent response (Willoughby et al., 1993). Since eccentric cardiac hypertrophy results in an increase in stroke volume and, therefore, an increased VO_2 max, an increase in O_2 pulse has been identified as a possible mechanism to induce cardiac hypertrophy and, therefore, increase cardiovascular function regardless of the mode of training. (DiBello et al., 1996; Snoeckx et al., 1982; Urhausen & Kindermann, 1989).

Interval aerobic programs provoke a higher O_2 pulse response than continuous aerobic programs (Perry et al., 1988); therefore, interval resistance training may also induce a greater increase in O_2 pulse than traditional strength training. Interval resistance training is a system of sequential resistance exercises using 40 to 60% 1RM for 12 to 15 repetitions with 15 to 30 s of rest between sets (Gettman & Pollock, 1981; Haennel et al., 1989). This system contrasts with the traditional strength program, in which multiple sets of one exercise are performed before moving to another exercise (Gettman & Pollock; Haennel et al.). A rise in O_2 pulse during interval resistance exercise similar to that

observed during interval aerobic exercise at the same HR could indicate increased cardiovascular function, and potentially stimulate exercise-induced cardiac hypertrophy.

Oxygen Pulse and Exercise Modality

A difference in O_2 pulse values could not be detected between resistance and aerobic interval exercise when the exercise and rest periods were averaged, indicating that at the same exercise HR, both resistance and aerobic interval exercise elicited similar O_2 pulse values. At first glance, this appears to support the theory that interval resistance exercise has the ability to increase O_2 pulse to the same level as interval aerobic exercise. Yet these findings obscure the underlying behavior of O_2 pulse, demonstrated by the statistically different O_2 pulse values between aerobic and resistance interval exercise when only the exercise phase was analyzed. Oxygen pulse is dependent on the mode of exercise, interval aerobic or interval resistance exercise. Oxygen pulse was higher during each 45 s of interval aerobic exercise than during each 45 s of interval resistance exercise. Oxygen pulse was lower during the rest periods following aerobic exercise intervals than during the rest periods following resistance exercise intervals. Although O_2 pulse was higher during each 45 s of interval aerobic exercise, O_2 pulse started at a high value and then leveled off at a lower value during interval aerobic exercise, while O_2 pulse started at a low value and then leveled off at a higher value during interval resistance exercise (see Figure 2). On the other hand, the O_2 pulse behavior was higher during interval resistance rest, compared to interval aerobic rest, and approached statistical significance. Previously, O_2 pulse has been shown to increase during the short rest periods following intense exercise, such as soccer, causing more pronounced cardiac hypertrophy (Urhausen

& Kindermann, 1999; Urhausen et al., 1996). Therefore, the trend of increasing O₂ pulse during interval resistance rest may indicate a stimulation of the heart to cause cardiac hypertrophy.

Heart Rate and Energy Systems

At the onset of exercise, the parasympathetic nervous system's influence is reduced to increase HR. Furthermore, as exercise intensity increases, exercise byproducts are produced, activating muscle chemoreceptors that signal to the brain to further reduce parasympathetic influence as well as to activate the sympathetic nervous system to increase HR (Foss & Keteyian, 1998). One factor that may influence O₂ pulse during interval aerobic and resistance exercise is the different energy systems utilized during each exercise. Resistance exercise primarily uses anaerobic energy systems. During rest periods, resistance exercise relies on blood circulation to remove excess lactic acid, to supply glucose for energy, and to provide oxygen to oxidize lactic acid (McArdle et al., 1996). Aerobic exercise primarily uses an aerobic energy system, which relies on blood circulation to provide an adequate supply of oxygen and glucose to oxidize lactic acid at the same rate as it is formed during steady state exercise (McArdle et al.). During short bouts of intense exercise lasting less than 90 s, the anaerobic system does not require oxygen for energy production. By the time local chemoreceptors notify the cardiovascular system to increase the HR to supply oxygen to oxidize lactic acid at the working muscles, the exercise interval is over. In exercise greater than 90 s, the build-up of exercise byproducts activates muscle chemoreceptors that send slow signals to the brain to increase HR (Foss & Keteyian). Accordingly, during the rest periods following

anaerobic exercise, metabolic byproducts need to be removed to prepare for the next bout of exercise, increasing HR. During aerobic exercise, the energy system requires oxygen for exercise to proceed; therefore, HR may be increased to meet the muscles' demand for oxygen.

Muscle Mass and Fiber Type

Interval resistance exercise causes small increases in stroke volume for a given HR, indicating a cardiac adaptation that is related to O_2 pulse (Haennel et al., 1989). Similarly, interval resistance exercise is able to maintain cardiovascular fitness after the cessation of a running program (Gettman & Pollock, 1981). Yet interval resistance exercise causes less aerobic improvement than traditional aerobic exercise (Gettman & Pollock), possibly due to the influence of the sympathetic nervous system on HR. During interval resistance exercise, VO_2 values increased from rest during the first arm and leg intervals and then leveled off at a higher value as exercise continued. Heart rate, on the other hand, while increasing during the entire interval resistance exercise, increased to a greater extent during arm exercise, and then decreased momentarily during the rest periods following arm exercise. This could be explained by the smaller arm muscle mass causing a greater sympathetic HR response during exercise (Parker et al., 1989; Taguchi & Horvath, 1987). Arm exercise produces higher HR values than leg exercise at similar VO_2 values (Parker et al.; Taguchi & Horvath), and the larger leg muscle mass causes a higher O_2 pulse at the same HR (Thomas & Londeree, 1989). Furthermore, the cessation of exercise allows the HR to quickly decrease during arm exercise since the smaller muscle produces less byproduct, requiring less oxygen and blood circulation to replenish

its energy stores, thus reducing sympathetic stimulation. On the other hand, HR values not only increase during leg exercise, but increase to an even greater value during the rest periods following leg exercise. This behavior is similar to that of aerobic interval exercise. The larger leg muscle mass requires an increased blood circulation to remove exercise byproducts and to supply oxygen; therefore, HR is increased to meet the needs of the muscles. Yet, VO_2 is increased during the rest intervals, possibly because the muscles extract oxygen from the blood to complete the metabolic processes that were started anaerobically. Also, VO_2 may be related to the muscle fiber types activated during the resistance exercise, with the majority of fibers recruited being type II, which have a smaller oxidative capacity than type I fibers (Foss & Keteyian, 1998). Determining which muscle fibers are recruited during 50% 1RM interval resistance exercise would help to answer the question as to why VO_2 behaves as it does during this interval resistance exercise.

The observation that VO_2 and HR values decreased after the first aerobic exercise interval and leveled off at a higher than resting value supports the theory that the oxygen supply and demand is balanced. Heart rate showed a slight upward drift as exercise progressed. Interestingly, HR values increased during the rest periods while VO_2 decreased, possibly indicating a delayed sympathetic HR response to the aerobic exercise as the circulatory system was stimulated during exercise to deliver oxygen to the working muscles. Yet during rest, the muscles were relatively inactive, thus reducing VO_2 . Also, during low intensity aerobic exercise, primarily type I fibers are recruited, which have a larger aerobic capacity compared with the type II fibers recruited during resistance

exercise (Foss & Keteyian, 1998). The type I fibers may be better able to extract oxygen, increasing VO_2 during exercise, while the type II fibers, those used during resistance exercise, are not able to utilize oxygen until exercise has ceased.

Oxygen Pulse and Exercise Intensity

The opposite behavior of HR and VO_2 between resistance and aerobic interval exercise may also be related to the intensity of exercise. The less rhythmic resistance exercise, as opposed to the rhythmic aerobic exercise, may alter the needs of the muscles. Also, the perception of exertion during resistance exercise may be different than during aerobic exercise at the same HR. Participants commented that interval resistance exercise was perceived as being much more difficult than interval aerobic exercise. Recruitment of different muscle fiber types may explain the observed behavior of O_2 pulse during the two interval exercises. This difference on the metabolic level may explain the reduced effect that resistance exercise has on altering VO_2 max.

Another factor that may explain the limited gains in VO_2 max as a result of resistance training is the rest periods between sets in traditional resistance training. The cessation of exercise may cause too dramatic a reduction in HR, lowering the intensity of the total program below that required to induce aerobic change (Gettman & Pollock, 1981). The question then is whether the HR during interval resistance exercise is sufficient to cause cardiovascular improvement. To examine this question, the interval resistance HR was compared to the age-predicted HRR. This was calculated by $[(\text{HR interval resistance} - \text{HR rest}) / (\text{HR max} - \text{HR rest})] \times 100$ (McArdle et al., 1996, p. 266). To improve cardiovascular fitness, exercise must be maintained at a level to elicit a HR

response in the aerobic training zone, or 40-85% of heart rate reserve (Moore & Roberts, 1997). The HR response during interval resistance exercise was between 19-73% ($M = 44\%$) of the predicted HRR, demonstrating that interval resistance exercise does have the ability to elicit a HR response that can positively influence cardiovascular fitness for some individuals. The percent of predicted HRR does not appear to be dependent on aerobic or resistance training volume. The participant whose HR response was 19% of his HRR considers himself very active, participating in five resistance training sessions per week for 31-60+ min per session, and five aerobic training sessions per week. Yet, the participant whose HR response was 73% of the HRR considers himself very active, participating in three to four resistance training sessions per week for 31-60+ min per session, and three to four aerobic training sessions per week. Similarly, a 58% HRR response was associated with six resistance-training sessions per week for 31-60+ min per session and five aerobic training sessions per week.

Sources of Error

Possible sources of error that could have compromised the reliability of the data were associated with the participant and the testing environment. Anxiety can increase HR and VO_2 . The study attempted to minimize the influence of anxiety by requiring that each participant had been enrolled in a beginning weight lifting class or personal training within the past year to assure familiarity with proper lifting technique. Each participant was also given pretest instructions, including recommendations on proper hydration, sleep, and clothing considerations, and was familiarized to the testing equipment prior to testing. The test administrators provided complete directions to each participant, allowing

time to answer any questions before, during, and after testing. These factors may have reduced anxiety that often accompanies this type of physiological testing.

Conclusion and Recommendations

The behavior of O_2 pulse during interval resistance exercise and interval aerobic exercise has been examined; therefore, the influence of interval resistance exercise on cardiac hypertrophy through the examination of O_2 pulse behavior is less obscure. Determining the O_2 pulse response at the HR commonly used during interval resistance exercise has allowed the comparison of aerobic responses between interval resistance and aerobic exercise. This has led to the determination that interval resistance exercise can produce an intensity sufficient to increase O_2 pulse and, thus, possibly influence cardiac hypertrophy, if the HR is sufficient according to ACSM (2000) guidelines. Therefore, determining O_2 pulse behavior during interval resistance exercise has defined the need for better HR control to elicit aerobic improvement. Many factors influence the various responses of the cardiovascular system to exercise, including muscle fiber type and nervous system response; therefore, determining which single factor most influences O_2 pulse and cardiovascular change is daunting, if not impossible, at this time. This study has provided athletes and trainers with more information to better monitor and maintain an intensity that may stimulate increases in O_2 pulse and, thus, cardiovascular adaptation to exercise, and possibly stimulate cardiac hypertrophy. Interval resistance exercise is a valuable tool that integrates resistance and aerobic exercise concepts, thereby increasing muscle and aerobic fitness without the time commitment of two separate programs. Future studies should further examine O_2 pulse behavior in a variety of exercise

conditions, including comparing interval resistance exercise to traditional strength exercise as well as observing O_2 pulse behavior with a variety of aerobic athletes. Also, observing the O_2 pulse behavior in conjunction with VO_2 max change during a long-term training study will further illuminate the influence of O_2 pulse on cardiovascular change. This study demonstrated that interval resistance exercise can increase O_2 pulse to the same extent as interval aerobic exercise at the same HR; thus, interval resistance exercise may positively influence VO_2 max and possibly stimulate cardiac hypertrophy if a minimum target HR is maintained.

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APPENDIX A

IRB Approval

San José State
UNIVERSITY

Office of the Academic
Vice President
Associate Vice President
Graduate Studies and Research
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To: Diane Augustine
4702 Parkwest Drive
San José, CA 95130

From: Nabil Ibrahim
AVP, Graduate Studies & Research

Date: October 28, 2002

The Human Subjects-Institutional Review Board has approved your request to use human subjects in the study entitled:

"The effects of interval strength and interval
treadmill exercise on oxygen pulse."

This approval is contingent upon the subjects participating in your research project being appropriately protected from risk. This includes the protection of the anonymity or confidentiality of the subjects' identity when they participate in your research project, and with regard to any and all data that may be collected from the subjects. The approval includes continued monitoring of your research by the Board to assure that the subjects are being adequately and properly protected from such risks. If at any time a subject becomes injured or complains of injury, you must notify Nabil Ibrahim, Ph.D. immediately. Injury includes but is not limited to bodily harm, psychological trauma, and release of potentially damaging personal information. This approval for the human subjects portion of your project is in effect for one year, and data collection beyond October 28, 2003 requires an extension request.

Please also be advised that all subjects need to be fully informed and aware that their participation in your research project is voluntary, and that he or she may withdraw from the project at any time. Further, a subject's participation, refusal to participate, or withdrawal will not affect any services that the subject is receiving or will receive at the institution in which the research is being conducted.

If you have any questions, please contact me at (408) 924-2480.

The California State University:
Chancellor's Office
Bakersfield, Chico, Dominguez Hills,
Fresno, Fullerton, Hayward, Humboldt,
Long Beach, Los Angeles, Maritime Academy,
Monterey Bay, Northridge, Pomona,
Sacramento, San Bernardino, San Diego,
San Francisco, San José, San Luis Obispo,
San Marcos, Sonoma, Stanislaus

APPENDIX B

ACSM Risk Stratification (ACSM, 2000)

Low Risk

Younger individuals (Men < 45 years of age; Women < 55 years of age) who are asymptomatic and meet no more than one risk factor threshold from the following list.

Positive Risk Factors

Defining Criteria

Family history

Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or other male first-degree relative (i.e., brother or son), or before 65 years of age in mother or other female first-degree relative (i.e., sister or daughter)

Cigarette smoking

Current cigarette smoker or those who quit within the previous 6 months

Hypertension

Systolic blood pressure $\geq$ 140 mm Hg or diastolic $\geq$ 90 mm Hg, confirmed by measurements on at least two separate occasions, or on antihypertensive medication

Hypercholesterolemia

Total serum cholesterol > 200 mg/dl (5.2 mmol/l), or on lipid-lowering medication. If low-density lipoprotein cholesterol is available, use > 130 mg/dl (3.4 mmol/L) rather than total cholesterol of > 200 mg/dl

Impaired fasting glucose	Fasting glucose ≥ 110 mg/dl (6.1 mmol/L) confirmed by measurements on at least two separate occasions
Obesity	Body Mass Index ≥ 30 kg/m ² , or waist girth > 100 cm
Sedentary lifestyle	Persons not participating in a regular exercise program or meeting the minimal physical activity recommendations from the U.S. Surgeon General's report.

APPENDIX C

Medical and Activity History Form

Please complete this form to the best of your ability. Please make this record as accurate and complete as possible. Please print or type for easy legibility. All information will be kept confidential.

Name _____ Address _____

Telephone: (home) _____ (work) _____

Person to contact in case of an emergency: _____

Relationship to you: _____ Daytime phone: _____

Personal physician (local): _____

Address _____ Telephone _____

Demographic Information:

Age: _____ Date of Birth (month/day/year): _____

Ethnic/Racial Background:

☐ Anglo	☐ African American	☐ Hispanic
☐ Japanese	☐ American Indian	☐ Other

1. Personal Medical Information:

In the past have you ever had:

YES	NO		YES	NO	
☐	☐	Heart attack or stroke	☐	☐	Elevated cholesterol
☐	☐	Heart surgery	☐	☐	Low HDL cholesterol
☐	☐	Diseases of arteries	☐	☐	Severe leg pain while exercising
☐	☐	Asthma	☐	☐	Diabetes (onset age_)
☐	☐	Heart rhythm problems	☐	☐	Orthopedic Problems
☐	☐	Heart murmur	☐	☐	Arthritis
☐	☐	High blood pressure	☐	☐	Injuries to back, knees, ankles
☐	☐	Peripheral circulatory disorders	☐	☐	Osteoporosis
☐	☐	Pulmonary (lung) disorder			

Have you recently (in the past 12 months) had:

YES	NO		YES	NO	
☐	☐	Pain or discomfort in chest or arms	☐	☐	Lightheadedness or dizziness
☐	☐	Back pain	☐	☐	Unusual fatigue with usual activities
☐	☐	Heart palpitations or rapid heart beat			

YES	NO		YES	NO	
☐	☐	Shortness of breath at rest	☐	☐	Awakened short of breath
☐	☐	Ankle edema			
☐	☐	Pain or discomfort with mild exertion	☐	☐	Loss of coordination/balance
☐	☐	Shortness of breath with usual activities	☐	☐	Chronic muscle soreness
☐	☐	Unexplained loss of consciousness	☐	☐	Unexplained extreme weakness
☐	☐	Joint soreness or swelling			

Describe any recent (in the past 12 months) illnesses, hospitalizations, medical problems, or surgeries you have had.

Do you have any disabilities that will affect your ability to exercise? ☐ YES ☐ NO
If yes, please explain.

Are you currently under a physician's care? ☐ YES ☐ NO
If yes, please explain.

Do you have any other problems that will affect your ability to exercise that we should know about? ☐ YES ☐ NO
If yes, please explain.

List any allergies you have (including drug allergies):

List all medications (prescribed or self-prescribed) you currently take.

Drug Taken	Reason for taking
_____	_____
_____	_____

3. Lifestyle Information

Have you ever smoked cigarettes, cigars, or a pipe? ☐ YES ☐ NO

Do you presently smoke? ☐ YES ☐ NO

If no, when did you quit? (year) _____

How many years did you smoke? _____

Please check the one category that best describes your current level of physical activity.

- ☐ **Inactive:** most of your day is spent sitting and you do no regular physical activity.
- ☐ **Relatively Inactive:** your day includes 3-4 hours of standing or walking, but you do very little physical activity.
- ☐ **Somewhat Active:** your day requires some physical activity and you occasionally are involved in recreational activities (e.g., tennis, golf) or you occasionally do physical activities such as gardening, housecleaning, dancing, etc.
- ☐ **Moderately Active:** your day requires physical activity and/or you participate in regular recreational or fitness activities (walking, jogging, swimming, cycling, weight lifting) for at least 20 minutes, 3 days each week.
- ☐ **Very Active:** your day requires extensive physical activity and/or you exercise moderately to vigorously for more than 30 minutes, 4 or 5 times each week.

If you are moderately or very active, please indicate the type and amount of exercise you do.

Type of Activity	Frequency (days/wk)	Duration (min)	Intensity (light, mod, hard)
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

When you exercise, how many days during the week do you strength train?

- ☐ 0-1 ☐ 2-3 ☐ 4+

If you strength train, how many minutes do you spend lifting weights?

- ☐ 0-15 ☐ 16-30 ☐ 31-60+

Do you participate in organized athletics? ☐ Yes No ☐

If yes, please explain _____

Briefly write your current primary exercise goal. _____

Briefly write your current strength-training goal. _____

How would you describe your general health? (check one)

- ☐ Excellent ☐ Good ☐ Average ☐ Fair ☐ Poor

Has a physician ever advised you not to exercise because of a medical problem? ☐ YES

☐ NO

If yes, please explain. _____

I certify that, to the best of my knowledge, the above answers are accurate and true.

Signature _____ Today's Date _____

APPENDIX D

Informed Consent Form

San José State
UNIVERSITY

**College of Applied
Sciences and Arts
Department of Human
Performance**

One Washington Square
San José, CA 95192-0054
Voice: 408-924-3010
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Dominguez Hills, Fresno, Fullerton,
Hayward, Humboldt, Long Beach,
Los Angeles, Marine Academy,
Monterey Bay, Northridge, Pomona,
Sacramento, San Bernardino, San Diego,
San Francisco, San José, San Luis Obispo,
San Marcos, Sonoma, Stanislaus

Agreement to Participate in Research

Principal Investigator: Diane Augustine

Responsible Faculty Member: Peggy Plato, Ph.D.

Title of Protocol: The effects of interval strength and interval treadmill exercise on oxygen pulse.

1. I have been asked to participate in a research study investigating the oxygen pulse response of interval strength training and interval treadmill exercise.
2. I will be asked to participate in an assessment of body composition, cardiovascular function, and muscular strength through these four tests at San Jose State University:
 - The BODY COMPOSITION test will use a skinfold caliper to estimate my percent body fat. The investigator will measure the thickness of the skinfold (two layers of skin and the fat lying just beneath the skin) at three sites, and then use a formula to calculate my percent body fat.
 - The 1-RM MUSCLE STRENGTH test will be performed on two exercise machines, the latissimus pull-down and the leg press. I will begin with a light warm-up, stretching, and practice lifts, after which I will attempt to lift heavier loads until I am unable to lift the weight. The last weight I successfully lift will be my 1-RM.
 - The SUBMAXIMAL INTERVAL STRENGTH test will use 50% of my 1-RM on both the latissimus pull-down and the leg press. I will exercise for 15 repetitions on one machine before moving to the next machine for the same number of repetitions, with a 30-second rest between sets. I will perform three sets, and the test will last approximately 9 minutes.
 - The SUBMAXIMAL TREADMILL test will use the exercise heart rate determined by the submaximal interval strength test. I will exercise for about 9 minutes on the treadmill at the predetermined heart rate, in intervals of approximately 1 minute of exercise with 30 seconds rest.
3. The following risks may be associated with this testing. I may experience some discomfort and possibly bruising during the body composition testing as my skin is slightly lifted (pinched) by the calipers. However, the discomfort is momentary, lasting only 4 seconds. I may experience the discomforts commonly associated with exercise during the 2 submaximal exercise tests (e.g., sweating; increased heart rate, breathing rate, and temperature; and fatigue). I may experience muscle soreness (occurring as much as 48 hours after testing), muscle cramping, muscle strains (tears), and/or ligament or tendon damage. In addition, lifts with the upper body and arms may cause abnormally high blood pressure and abnormal heart beats. These risks are minimized by using an appropriate warm-up period, and allowing me to judge when I have reached my maximum effort.

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4. These tests will allow the investigator to estimate my body composition—amount of fat and fat free mass, to estimate the muscle strength of selected muscles or muscle groups, and to compare my cardiovascular response during two types of submaximal exercise. I will benefit from this testing by learning the following: my body fat percent, my ideal body weight range and the need for weight gain or loss, my maximum strength on two strength exercises, and recommended training heart rate.
 5. All results, as well as any conversations I may have with the investigator, are strictly confidential unless I consent to release this information with a specific "release to publish statement".
 6. Questions about the research can be addressed to the principal investigator (Diane Augustine, (408) 374-6904). Complaints about the research may be presented to Greg Payne, Ph.D. (408) 924-3028, Human Performance Department Chair. Questions about research, subjects' rights, or research-related injury may be presented to Nabil Ibrahim, Ph.D., Associate Vice President for Graduate Studies and Research, at (408) 924-2480.
 7. No service of any kind, to which I am otherwise entitled, will be lost or jeopardized if I choose to "not participate" in the study.
 8. Consent is given voluntarily. I may refuse to participate in the study or in any part of the study. If I decide to participate in the study, I am free to withdraw at any time without prejudice to my relations with San Jose State University.
 9. I have received a signed and dated copy of the consent form.
- The signature of a participant on this document indicates agreement to participate in the study.
 - The signature of a researcher on this document indicates agreement to include the above named participant in the research and attestation that the subject has been fully informed of his or her rights.

Signature	Date
Investigator's Signature	Date

APPENDIX E

Raw Data

Participant	Age (yrs)	Body Fat (%)	Height (cm)	Weight (kg)	Activity Level Active: Somewhat Moderate Very	Resistance Training (sessions/ week)	Resistance Training (minutes/ session)	Aerobic Training (sessions/ week)	% Predicted HRR resistance exercise
7	27	9.0	172.2	75.0	Very	5	31-60+	5	19%
14	26	32.3	158.0	60.1	Somewhat	2.5	0-15	0	24%
10	20	36.0	170.2	80.9	Moderate	2.5	31-60+	0	25%
1	26	17.6	168.4	53.3	Very	2.5	31-60+	6	29%
4	19	28.2	164.3	63.2	Very	6	31-60+	5	30%
6	29	31.4	163.1	62.3	Moderate	2	16-30	3	37%
13	26	10.4	151.8	66.0	Somewhat	0.5	0-15	0	38%
20	31	14.8	165.1	78.5	Very	5	31-60+	1	39%
18	25	32.1	177.8	73.1	Somewhat	0.5	0-15	0	39%
8	23	11.0	181.6	84.6	Very	5.5	31-60+	2.5	40%
19	27	28.4	163.3	62.5	Very	3	31-60+	6	43%
3	20	29.8	176.0	104.2	Somewhat	2	31-60+	0.5	45%
9	24	21.5	184.2	100.6	Very	3	31-60+	3	48%
2	20	17.8	181.4	77.1	Moderate	2	31-60+	0	54%
12	23	22.1	189.0	106.6	Moderate	0.5	0-15	7	55%
17	27	13.1	175.0	70.8	Very	2.5	16-30	6	57%
15	27	20.6	168.7	91.7	Very	6	31-60+	5	58%
16	26	10.8	179.3	77.4	Very	3.5	31-60+	3.5	73%

Resistance VO2 (ml/min) Participant	Arm1	Rest A1	Leg1	Rest L1	Arm2	Rest A2	Leg2	Rest L2	Arm3	Rest A3	Leg3	RestL3
1	515.3	664.0	705.0	699.5	690.3	797.5	750.0	669.5	794.7	844.0	704.0	679.0
2	870.6	1144.0	1171.0	1406.0	1508.6	1529.6	1305.0	1406.0	1638.6	1566.0	1407.6	1477.5
3	1040.3	1561.5	1589.3	2148.0	2184.6	2065.0	1812.0	2394.5	2274.0	2152.0	1862.0	2280.5
4	589.0	923.5	790.0	871.0	930.6	967.5	999.3	953.0	924.0	1094.5	872.3	887.5
6	651.3	455.0	584.3	464.3	571.0	467.0	539.3	449.0	534.0	456.0	530.0	400.0
7	679.3	1070.0	1166.0	1326.0	1134.0	1220.5	1263.3	1357.5	1231.6	1450.0	1288.0	1303.5
8	865.6	1476.0	1607.3	1803.5	1919.0	2184.0	1930.3	1979.5	2004.6	2113.0	2044.3	1849.0
9	986.0	1618.5	1693.0	2097.5	2048.0	2219.5	1859.0	2362.5	2075.0	2341.5	2112.0	2012.5
10	782.3	810.5	813.6	1301.5	1145.3	1144.0	1162.0	1250.0	1216.3	1201.5	1234.3	1269.5
12	1130.6	1463.5	1418.3	1582.5	2010.3	1963.5	1709.3	1985.5	1994.6	2287.5	2036.3	2065.0
13	771.0	854.5	896.6	952.0	1123.3	1021.5	967.3	1005.5	1167.0	1030.5	969.0	948.5
14	440.3	749.0	677.0	874.5	814.0	738.0	674.6	747.5	849.6	788.0	674.0	748.5
15	769.0	1403.0	1439.3	1511.5	1786.6	1792.0	1678.0	1873.0	1846.0	1896.5	1904.3	2011.0
16	1179.3	1889.5	1616.6	1910.5	1550.0	1787.0	1738.6	2305.0	1973.0	1944.0	1895.3	2170.0
17	771.5	1252.0	1150.6	1312.5	1311.0	1436.0	1364.6	1436.0	1311.0	1566.0	1400.6	1423.0
18	670.6	615.0	912.0	772.0	935.6	837.0	931.3	886.0	1009.3	859.5	1043.3	929.0
19	681.6	821.0	905.6	942.5	950.3	945.0	972.0	1009.0	969.0	1064.5	1043.3	1006.5
20	878.3	1181.5	1136.6	1265.5	1501.3	1496.0	1341.6	1289.5	1646.6	1579.0	1364.6	1419.5
Mean	792.9	1108.4	1126.2	1291.2	1339.7	1367.3	1277.6	1408.8	1414.4	1457.4	1354.7	1382.2

Resistance HR (bpm) Participant	Arm1	Rest A1	Leg1	Rest L1	Arm2	Rest A2	Leg2	Rest L2	Arm3	Rest A3	Leg3	RestL3
1	95.7	100.5	94.3	107.0	94.7	101.0	97.0	111.5	96.7	96.5	93.0	110.0
2	146.6	123.0	138.3	142.5	150.6	124.0	145.0	154.5	157.0	136.5	151.3	149.0
3	113.0	116.5	138.0	138.5	145.0	130.5	147.0	150.5	161.3	146.5	159.0	157.5
4	108.6	109.0	118.3	121.0	128.3	112.0	121.0	124.0	129.3	108.5	112.3	115.5
6	97.6	97.5	93.3	105.5	100.6	109.0	98.6	112.0	101.3	103.0	98.3	101.3
7	78.0	71.5	86.3	86.0	82.6	75.5	86.0	87.0	91.6	88.0	90.6	87.5
8	113.0	90.0	124.0	124.5	146.6	133.5	147.3	148.0	150.0	150.5	158.0	142.5
9	121.6	110.0	125.0	125.5	136.6	118.5	133.3	140.5	149.0	138.0	145.0	148.5
10	108.3	96.0	109.6	114.0	136.0	109.5	123.3	126.0	144.3	119.0	131.6	123.0
12	119.3	94.5	128.6	126.0	151.6	121.5	148.0	147.0	169.0	147.0	161.6	106.5
13	99.0	89.0	110.0	110.5	122.3	103.5	110.6	128.5	134.0	119.0	116.0	120.5
14	94.3	98.0	108.6	125.5	121.3	103.5	108.6	115.0	121.6	111.5	108.6	111.0
15	115.3	119.5	141.3	149.5	147.6	126.0	140.3	144.0	168.6	172.0	175.6	171.0
16	146.3	131.5	159.3	152.0	149.0	150.5	168.0	169.0	161.3	169.0	175.3	176.0
17	113.6	107.5	127.6	122.5	143.6	140.0	139.0	146.0	147.3	152.0	147.3	140.5
18	113.3	116.0	100.0	116.0	124.0	134.0	105.3	126.0	130.6	144.0	110.6	126.0
19	117.6	108.0	113.0	124.0	120.3	104.5	113.6	128.5	126.3	113.5	115.6	115.5
20	89.6	87.5	116.6	116.0	97.5	125.0	118.0	118.0	120.6	94.0	100.6	134.0
Mean	111.8	104.6	118.6	123.0	129.5	117.5	125.4	132.8	137.6	130.3	132.3	129.5

Aerobic HR (bpm) Participant	Ex1	Rest1	Ex2	Rest2	Ex3	Rest3	Ex4	Rest4	Ex5	Rest5	Ex6	Rest6
1	103.7	96.5	99.3	87.5	98.0	97.0	101.3	97.0	97.3	97.0	100.3	96.0
2	146.6	139.0	133.0	135.5	129.0	136.0	130.6	131.0	136.0	140.0	137.0	140.5
3	156.3	141.0	145.3	135.5	139.0	138.0	141.0	144.0	147.3	143.0	145.3	146.0
4	128.3	126.0	112.0	117.5	111.3	120.0	113.6	123.0	114.6	118.0	114.6	121.0
6	96.3	106.0	109.0	115.5	115.6	113.5	113.0	123.5	127.0	114.0	116.0	118.5
7	89.3	73.0	87.3	72.5	82.0	76.5	83.0	77.5	87.6	80.0	84.3	80.5
8	131.6	121.0	122.0	117.0	124.0	120.5	129.3	130.0	132.3	135.5	132.0	125.5
9	128.6	120.0	120.3	124.5	122.6	127.0	126.3	127.5	128.0	130.0	128.3	128.5
10	121.3	117.5	112.6	124.0	111.3	118.0	109.6	118.0	109.0	116.5	108.0	115.5
12	129.3	135.0	131.0	144.0	131.0	138.0	130.0	144.5	135.3	146.0	132.3	143.0
13	122.6	114.0	108.0	101.5	102.0	102.5	103.3	106.0	106.0	112.0	108.3	111.5
14	104.3	103.0	98.6	102.5	105.0	110.5	107.0	112.5	101.6	109.0	106.6	119.0
15	147.3	147.0	139.6	143.5	139.3	144.5	140.0	147.0	138.3	147.5	146.0	150.5
16	156.3	152.0	149.6	147.5	143.0	150.5	147.0	150.5	152.6	158.5	148.0	151.0
17	137.3	122.5	106.0	122.5	121.6	133.0	136.0	133.0	128.6	131.5	136.3	131.0
18	138.6	142.0	132.0	132.0	124.0	136.0	126.6	136.0	126.6	134.0	126.6	132.0
19	116.3	108.5	104.0	112.0	110.3	113.5	109.6	125.5	110.3	118.0	107.3	123.0
20	115.6	118.5	114.0	119.0	109.6	121.0	109.6	123.5	112.6	118.5	114.6	122.5
Mean	126.1	121.3	118.0	119.7	117.7	122.0	119.8	125.0	121.7	124.9	121.8	125.3

Aerobic VO ₂ (ml/min) Participant	Ex1	Rest1	Ex2	Rest2	Ex3	Rest3	Ex4	Rest4	Ex5	Rest5	Ex6	Rest6
1	710.3	752.5	847.3	690.5	810.0	684.5	843.3	671.5	815.6	751.5	827.3	748.0
2	1943.0	1669.0	1639.0	1460.5	1474.6	1386.5	1458.0	1246.0	1449.6	1351.0	1398.6	1257.5
3	2648.0	2214.0	2120.3	1931.5	1982.3	1784.0	2056.6	1873.0	2023.3	1828.5	1919.3	1888.5
4	1175.3	971.5	898.0	805.5	911.6	791.5	931.0	826.5	942.6	840.5	943.0	892.5
6	402.6	749.5	701.6	705.0	714.0	906.0	833.3	776.5	786.6	973.0	855.6	872.5
7	1686.3	1196.5	1363.3	941.0	1232.3	1104.0	1334.0	1078.5	1300.0	1061.0	1183.3	1158.5
8	2148.6	1594.5	1667.6	1395.0	1740.6	1671.0	1986.6	1665.5	1835.6	2094.0	2168.0	1667.0
9	2404.0	2144.0	2122.6	1999.5	2142.6	1959.5	2229.0	1932.0	2348.6	2068.0	1398.6	2014.5
10	839.3	558.5	740.0	512.0	754.3	505.5	678.3	519.0	661.6	603.5	602.0	698.5
12	2678.3	2118.0	2468.0	1680.5	2734.0	2069.0	2234.0	1742.0	2465.6	1913.5	2476.0	2049.0
13	1543.3	1320.5	1339.0	1067.0	1152.6	1015.0	1227.3	987.5	1144.6	1129.5	1138.3	985.0
14	666.6	494.0	576.6	564.5	667.3	595.0	722.0	613.5	587.3	555.0	706.6	625.0
15	2103.0	1497.5	1860.3	1492.0	1857.0	1280.5	1763.3	1236.0	1757.6	1309.0	1774.3	1467.0
16	2150.3	1575.5	1706.0	1490.0	1656.0	1531.0	1771.3	1586.0	1833.0	1693.5	1756.3	1622.0
17	1569.3	1036.5	1345.0	1111.5	1299.6	1277.5	1490.0	1265.0	1546.6	1256.5	1493.6	1379.5
18	1468.6	1303.5	1265.6	932.5	1067.3	830.5	1107.6	817.5	1049.6	962.0	1014.6	815.0
19	1316.6	945.5	937.3	844.5	1053.0	929.5	1003.0	935.0	1035.0	951.0	1008.3	1031.5
20	1472.6	979.0	1193.0	1043.5	1145.6	930.0	1285.3	1118.5	1326.3	1149.0	1277.0	1108.5
Mean	1607.0	1284.4	1377.3	1148.1	1355.3	1180.6	1386.3	1160.5	1383.8	1249.4	1330.0	1237.8

**O₂ Pulse
(VO₂/HR)**

Aerobic Means	Ex 1	Rest 1	Ex 2	Rest 2	Ex 3	Rest 3	Ex 4	Rest 4	Ex 5	Rest 5	Ex 6	Rest 6
	12.6	10.5	11.3	9.5	11.3	9.6	11.4	9.3	11.2	9.9	10.9	9.8
Resistance Means	Ex 1	Rest 1	Ex 2	Rest 2	Ex 3	Rest 3	Ex 4	Rest 4	Ex 5	Rest 5	Ex 6	Rest 6
	7.2	10.8	9.5	10.5	10.5	11.6	10.1	10.5	10.2	11.4	10.2	10.2