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THE UNIVERSITY OF ALBERTA

FUNCTIONAL ADAPTATION TO VELOCITY - SPECIFIC RESISTANCE TRAINING

by

STEWART RICHARD PETERSEN

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled Functional Adaptation to Velocity-Specific Resistance Training submitted by Stewart Richard Petersen in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

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ABSTRACT

In order to investigate the effects of velocity-specific circuit resistance training on peak torque, muscle cross-sectional area, maximal aerobic power, recovery, and maximal cardiac output characteristics of previously well-trained subjects, four separate studies were undertaken. In all cases, subjects trained for either five or six weeks utilizing various circuit arrangements of hydraulic resistance equipment. It was found (Chapter II) that initial strength characteristics influenced the nature of the adaptation in peak torque output following training at 3.14 $rad \cdot s^{-1}$. Subjects with high initial peak torque output improved ($p < 0.05$) at 3.14 $rad \cdot s^{-1}$ while subjects in the low peak torque group improved ($p < 0.05$) when tested at 3.14 $rad \cdot s^{-1}$ and at 0.52 $rad \cdot s^{-1}$. It is suggested that the apparent controversy over velocity-specific strength training adaptation may be due in part to initial characteristics of subjects. It was also found (Chapter III) that training at either 3.14 $rad \cdot s^{-1}$ or 1.05 $rad \cdot s^{-1}$ resulted in increased ($p < 0.05$) cross-sectional area of the knee-extensors, however alterations in the in vivo torque-velocity relationship were different for each program. These data suggest that factors other than muscle size mediate the performance adaptation, and even in well-trained

individuals neural adaptation is probably an important factor. Circuit training at 3.14 rad·s^{-1} utilizing a 1:2 exercise:relief ratio increased ($p < 0.01$) the $\dot{V}O_2$ max and the training effect did not appear to be related to initial fitness (Chapter IV). It was also found (Chapter V) that adaptations for maximal cardiac output and recovery from heavy exercise may be achieved through circuit-resistance training independent of exercise velocity, while achieving an exercise intensity high enough to improve the $\dot{V}O_2$ max appears to be velocity-dependent. These results indicate that circuit resistance training may be an effective means of enhancing both strength and aerobic fitness characteristics of previously well-trained subjects and, therefore, may be an appropriate training mode for athletes during certain phases of a training calendar.

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CHAPTER I

Introduction

By way of introduction to the questions under investigation regarding the adaptive responses to velocity-specific circuit resistance training, this chapter will present brief discussions of salient topics which have bearing on velocity-specific strength training responses and aerobic system adaptation consequent to circuit resistance training. Hopefully, by this process, the rationale for the present investigations will become apparent. The development of research questions is summarized at the end of this chapter as a statement of purpose.

Force-Velocity Relationships in Exercise

The early work of Hill (1938) described what has become the classic force-velocity relationship in isolated muscle. This hyperbolic curve describes an inverse relationship between muscular tension and the velocity of shortening. The relationship may be partially replicated in vivo by plotting peak torque against angular velocity using an isokinetic dynamometer. The in vivo relationship differs from the in vitro relationship in several ways. First, the term velocity, when using an isokinetic dynamometer, is generally assumed to reflect the constant rotational velocity of a body segment rather than a constant rate of

muscle shortening. This distinction has been discussed in detail by Hinson et al. (1979). Secondly, peak torque is usually not measured with respect to a specific joint angle. Perrine and Edgerton (1978), and Caiozzo et al. (1981) have argued that unless torque is always measured at a specific angle, the measurement does not account for muscle length. Therefore, changes observed with training may reflect mechanical alterations rather than contractile alterations. This is probably only a concern when direct comparisons between the in vivo and in vitro curves are attempted. Functionally, changes in force output irrespective of joint angle may represent a realistic performance improvement for an athlete. Thirdly, the most common commercially available isokinetic dynamometer, the Cybex system, has an upper velocity limit of 300 $^{o}\cdot s^{-1}$, or 5.23 $rad\cdot s^{-1}$. This provides a restriction in consideration of the predicted unloaded knee extensor velocity of over 800 $o._s$-1 or approximately 14 $rad\cdot s^{-1}$ (Perrine 1986). Finally, as angular velocity approaches zero, the isokinetic muscle force curve tends to rise much less sharply than the in vitro curve. Perrine and Edgerton (1978) demonstrated an actual decline in peak torque below 96 $o._s$-1. A neural inhibition of force has been suggested as the main cause of this phenomenon (Barnes 1975; Perrine and Edgerton 1978). Caiozzo et al. (1981) have altered the shape of the in vivo

partially due to a reduction in this tension-limiting inhibitory effect. Several investigators (Lesmes et al. 1978; Coyle et al. 1981; Caiozzo et al. 1981; Kanehisa and Miyashita 1983) have speculated on the nature of training induced force and power output alterations which may be specific to exercise velocity. The nature of such a specificity, or the suggestion of greater transfer of strength from high-speed training to low-speed performance (Moffroid and Whipple 1970; Lesmes et al. 1978), or vice-versa (Caiozzo et al. 1981; Kanehisa and Miyashita 1983) is very uncertain.

Myogenic and Neurogenic Responses to Resistance Training

Because of the direct proportionality between muscle cross-sectional area and force production (McDonagh and Davies 1984), it might be assumed that resistance training to maximize muscle size might be most beneficial. However, the rate of strength adaptation is often not coincidental with the rate of muscle growth (Luthi et al. 1986) and several studies have reported data which indicates a poor correlation between changes in force and muscle size (Ikai and Fukunaga 1970; Costill et al. 1979; MacDougall et al. 1980). That is, strength tends to increase far more than muscle size.

Several findings would indicate that a change in nervous control of motor units may be more important than

muscle hypertrophy. Isometric training at one joint angle tends to increase muscle performance at that angle far more than at others (Lindh 1979). An increase in performance due to hypertrophy would presumably be evident at all joint angles. As well, improvements in force expression in an untrained limb after contralateral resistance training would suggest altered motor unit control patterns (Ikai and Fukunaga 1970). No direct evidence is available on neural adaptation which may be specific to exercise velocity, but Sale and MacDougall (1981) suggest that this may be the basis for velocity-specific strength training effects. This suggestion is underscored by the apparent link between hypertrophy and the amount of tension developed within the muscle during training (Goldberg et al. 1975; McDonagh and Davies 1984). At movement velocities of 3.14 $rad \cdot s^{-1}$, the peak torque drops to about 60-65% of the maximum value observed at very slow velocities, or in an isometric contraction (Lesmes et al. 1978; Caiozzo et al. 1981; personal observations). This may be a sufficient stimulus to induce fiber hypertrophy, as suggested by Costill et al. (1979), who reported increased Type II fiber area following training of the knee extensors at 3.14 $rad \cdot s^{-1}$ for seven weeks. Coyle et al. (1981) have also reported enlargement of Type II fibers following six weeks of training the knee extensors at 5.23 $rad \cdot s^{-1}$. Simple computations from their data would indicate that tension at 5.23 $rad \cdot s^{-1}$ was

approximately 30% of that output isometrically. Therefore, while maximal muscle tension may maximize hypertrophy, submaximal tensions at high movement velocities may initiate some hypertrophy as well. In that case, altered cross-sectional area of muscle may be an important consideration in the resolution of the specificity of velocity question. In many sport situations, athletes must perform at a variety of movement velocities. Therefore, in consideration of optimizing the effects of preparatory training, the influence of velocity-specific resistance training on the in vivo torque-velocity relationship may be a very important question.

Aerobic Power, Endurance, and Recovery

When a high rate of energy production is required over a time period exceeding approximately 2-3 minutes, the chief factor limiting performance is the ability to produce energy from the aerobic pathway (Gollnick and Hermansen 1973). Aerobic energy production is commonly considered to be dependent upon both the rate at which oxygen can be delivered to the tissue, and the rate at which the oxygen may be taken up and utilized by the mitochondria. Adaptation to physical training may be manifested in enhanced peripheral circulation (Brodal et al. 1977), increased mitochondrial enzyme activity (Holloszy 1975), and increased cardiac contractility (Bersohn and Scheuer 1976). The various

contributions and limitations of both central and peripheral phenomena to aerobic power and endurance have been recently reviewed in the literature (Saltin and Rowell 1980; Blomqvist and Saltin 1983; MacDougall and Sale 1981). The importance of a highly developed aerobic system in enhancing recovery from heavy exercise has been suggested (Wenger 1981; Brooks and Fahey 1984). Presumably, enhancement of both aerobic metabolic potential and circulatory function would facilitate more rapid lactate removal and utilization, thus speeding the recovery process. Therefore, the need for development of aerobic fitness may be rationalized for athletes involved in activities requiring repeated bouts of high-intensity exercise.

The production of lactate during rest and exercise is an ongoing process which should not be used as an indicator of oxygen availability (Brooks 1986; Gollnick et al. 1986). During very heavy exercise the rate of lactate production (entry into the blood) usually exceeds the rate of removal (from the blood). High intra-muscular lactate concentrations resulting from exhausting exercise have been associated with depressed intracellular pH which in turn has been associated with impairment of the contractile process and fatigue (Gollnick et al. 1986). While this should not be interpreted as a causal relationship (Mainwood and Renaud 1985), the rate of efflux of lactate into the blood, and the rate of transport to sites of oxidation clearly may influ-

ence the onset of fatigue and/or recovery. This may be dependent upon capillary density in the exercising muscle, or diffusion distances between capillary and muscle fiber membranes. The effect of resistance training on capillary density is unclear. Schantz et al. (1982) have reported that the mean number of capillaries in elite body-builders is double that in normal subjects. Luthi et al. (1986) reported no change in capillarity after eight weeks of heavy resistance training. Ozolin (1986) has stated that force-trained muscles have a lower capillary density than normal due to muscle fiber hypertrophy. Presumably this infers a reduction in the capillary/fiber area ratio rather than an absolute reduction. Saltin and Rowell (1980), and Klausen et al. (1983) have reported increased (20%) capillary density after short-term (8 weeks) endurance training. If hypoxia is a key factor in stimulation of enhanced peripheral circulation (Rhan 1966), then it would seem logical that resistance exercise which temporarily occludes local circulation may have a similar effect.

Interval or Sprint Training and Aerobic Function

Gollnick and Bayly (1986) have described the maximal power production accomplished in one minute or less as "ultra short-term" or sprint training. Due to the delay in the response of the aerobic pathway at the onset of exercise, oxygen consumption during such supramaximal exercise

may reflect only about 80% of the $\dot{V}O_2$ max (Saltin et al. 1971). Thus, in order to support the exercising muscle, energy production must come from all sources. Sprint training may increase the $\dot{V}O_2$ max (Saltin et al. 1976; Bell 1986). Aerobic energy pathway enzymes may show increased activity following sprint training (Davis et al. 1981; Saltin et al. 1976; but not Sharp et al. 1986). Glycolytic pathway enzymes may increase as well (Saltin and Gollnick 1983; Sharp et al. 1986) which has been linked to increased muscle lactate production, increased blood lactate concentration, and increased muscle buffer capacity (Sharp et al. 1986).

Resistance Training and Aerobic Function

Although many athletes use resistance training to enhance their muscular strength and power, this training mode has not generally been used to improve aerobic power. It has been suggested that the intermittent nature of resistance training with heavy weights, few repetitions, and frequent rest periods does not encourage an aerobic power training effect (Gettman et al. 1978). Byrd and Barton (1973) reported exercise and relief heart rates of 152 and 119 beats.min^{-1} for experienced lifters over an hour of exercise. They suggested that heart rate responses of this magnitude may elicit a change in maximum oxygen consumption ($\dot{V}O_2$ max) in running or swimming programs, but not in weight

lifting programs because of the short duration of the program, impaired local blood flow during heavy lifting, or assessment methods which do not reflect specificity of training effects. This is implied in the work of Hickson et al. (1980) who reported small increases in the absolute $\dot{V}O_2$ max after a 10 week program of heavy resistance training which was evident during cycling exercise, but not on the treadmill.

Circuit training usually involves many repetitions of light weights with short rest intervals between exercise stations. Wilmore et al. (1978) and Gettman et al. (1980; 1978) have reported enhanced $\dot{V}O_2$ max following 10, 12, and 20 week programs of circuit weight training, while Allen et al. (1976) reported no change in $\dot{V}O_2$ max over 12 weeks of training.

It is generally accepted that the training effects in muscle are most beneficial to the athlete when the training stimulus mirrors the event being trained for. The specificity principle may be extended to such parameters as motor unit recruitment, velocity of movement, and duration and intensity of exercise (MacDougall and Sale 1981). However, such sport-specific training is not always possible or even desirable, and therefore the question of transfer of enhanced function from non-specific training back to specific sport performance must be addressed when designing training programs for athletes. It has been suggested that

up to 50% of the increase in aerobic power following training may be due to enhanced circulatory function (Rowell 1974; Scheuer and Tipton 1977). Therefore, if non-specific activities such as circuit training may enhance oxygen transport, they may be of considerable benefit to the athlete. This is especially true if these adaptations are achieved simultaneously with the strength and power training effects which have been reported (Gettman et al. 1978, 1980). Gettman et al. (1982) have speculated that the enhanced $\dot{V}O_2$ max observed in previously untrained subjects consequent to circuit weight training programs may have been due to enhanced circulatory function. Haennel et al. (1985) have reported significant alterations in stroke volume and maximal cardiac output in previously sedentary males following circuit training programs of different intensities. Whether these adaptations may be achieved in previously trained individuals is a question for further research.

It seems likely that the nature of the circuit and the choice of resistance apparatus would influence the exercise intensity and therefore the effect on aerobic power. Recent developments in resistance training equipment provide accommodating resistance from variable hydraulic cylinders which allow safe, high-velocity movements. By demanding an "all-out" effort during exercise intervals, and by shortening the relief intervals between stations, a relatively high

exercise intensity may be achieved. Thus, circuit resistance training may be similar to interval or sprint training. Petersen et al. (1984) have reported oxygen consumption of up to 83% $\dot{V}O_2$ max and heart rates of up to 90% of maximum during 20 second exercise intervals. As well, post-exercise venous lactate concentrations of 17.1 $mmol \cdot l^{-1}$ were observed. Prolonged exercise of this type may then be an effective means of improving aerobic power.

Summary

Briefly, the nature of the in vivo force-velocity relationship has been discussed, as well as some of the adaptive responses of skeletal muscle to resistance training. Clearly, the ability to alter the force-velocity relationship may have important practical implications for muscular performance over a variety of sport-specific movement velocities, however, our understanding of this possibility is unclear.

The importance of the aerobic base to energy production and recovery has been discussed. The similarity between sprint (or interval) exercise and high-velocity circuit resistance exercise suggests that the latter may be an effective means of altering aerobic power.

Statement of Purpose

In consideration of the potential importance of velocity-specific resistance training to athletes who perform throughout a range of movement velocities, and that sprint or high-intensity circuit resistance training may enhance aerobic power, an initial training study was designed:

1. To investigate the question of velocity specific adaptation to high velocity resistance training and to consider the possible effects of initial strength levels and initial $\dot{V}O_2$max on strength adaptation; and secondly,
2. To determine if a high velocity circuit resistance training program could enhance aerobic power and to consider the effects of initial $\dot{V}O_2$ max on the aerobic training effect.

This study is summarized in Chapters II and IV respectively. Based on the findings of this preliminary work, another project was undertaken:

1. To investigate the effects of either high or low velocity resistance training on the in vivo torque-velocity relationship and the cross-sectional area of the knee extensor musculature; and
2. To investigate the effects of velocity-specific circuit resistance training on maximal aerobic power, maximal stroke volume and cardiac output,

and on the rate of lactate removal from the blood following exhausting exercise.

This study is summarized in Chapters III and V respectively.

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CHAPTER II

The Acquisition of Strength: The Influence of Training Velocity and Initial Fitness[1]

Introduction

Many athletes engage in strength and power training to enhance their performance. In regard to the specificity of strength and power training effects, Sale and MacDougall (1981) and Sale (1986) have suggested that training responses are specific to movement patterns, the joint angle or angles at which the training occurs, and to the type of contraction (isometric, isotonic, or isokinetic) utilized during training. As well, Coyle et al. (1981) and Caiozzo et al. (1981) have suggested a velocity specific training effect where training at low velocity increased low velocity strength but had little effect on torque outputs at high velocity. Moffroid and Whipple (1970) and Lesmes et al. (1978), have also reported a greater strength transfer from high velocity training to low velocity performance than from low velocity training to higher velocity performance. This specificity of velocity principle was not entirely supported by the findings of Kanehisa and Miyashita (1983). In this study, the group which trained at slow speed improved performance at all test speeds while the high speed training

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[1] A version of this chapter has been published: Petersen, S.R., Miller, G.D., Wenger, H.A., and Quinney, H.A. Can. J. Appl. Sport Sci. 9:4, 176-180, 1984.

group improved only at fast test speeds. Therefore it would appear that such a specificity principle has not been clearly defined.

Much of the work regarding velocity specific training effects has utilized pools of untrained subjects (Moffroid and Whipple 1970; Coyle et al. 1981; Caiozzo et al. 1981) or has not reported information on the training state of the subjects (Kanehisa and Miyashita 1983). The effects of initial fitness on adaptation to aerobic training are well documented and have been recently reviewed (Wenger and Bell 1986). While such information is not available regarding initial fitness and strength adaptation, it would be reasonable to assume that the same principle would apply. That is, subjects with low initial strength would show greater improvement than subjects with high initial strength.

The importance of a well developed oxygen transport system for the acquisition of optimal power and strength has been suggested (Wenger 1981). Enhanced aerobic replacement of ATP during recovery intervals, the flushing of lactate from muscle and/or the aerobic oxidation of lactate should all assist in recovery and, therefore, permit greater quality of training.

The present study was undertaken to further investigate the question of velocity-specific strength training effects

and to consider the possible effects of initial strength levels and initial VO_2 max on strength adaptation.

Methods

Twenty apparently healthy and habitually physically active college-aged males provided informed consent to participate in the study. All subjects had some previous experience with resistance training. Subjects were assessed for VO_2 max and peak torque in knee extension and flexion and then assigned to either a training group (TG) or a control group (CG).

Peak torques for right and left knee extension and flexion at angular velocities of 0.52 and 3.14 $rad \cdot s^{-1}$ were assessed using a Cybex II isokinetic dynamometer system. Subjects were familiarized with the system and the required limb actions. Limb alignment and stabilization procedures were standardized for each subject. A minimum of three continuous maximal extensions and flexions were completed. Subjects were verbally encouraged to exert a maximal effort at all times. In all cases, the fast speed test was conducted first, with ample recovery time provided before the slow speed test. In order to minimize artifactual torques, the first extension and flexion torques from each series were omitted from the results, and an optimal damping setting of two on the Cybex II chart recorder was used. All

calculations of peak torque were performed by the same investigator.

Maximal oxygen consumption ($\dot{V}O_2$ max) was assessed on a motor-driven treadmill (Quinton 24-72). Subjects ran for one minute on a level treadmill at each of 3.1, 3.6, 4.0, and 4.4 $m \cdot s^{-1}$ and then the grade was increased 2 percent each minute until $\dot{V}O_2$ max was observed, or until volitional exhaustion. To confirm attainment of $\dot{V}O_2$ max, subjects met one or both of the following criteria: a levelling or decrease in $\dot{V}O_2$ with increasing exercise loads; and a respiratory exchange ratio greater than 1.15. The peak $\dot{V}O_2$ value obtained during the exercise was recorded as $\dot{V}O_2$ max. Expired gases were collected and analyzed each 30 s for volume and O_2 and CO_2 concentration with a Beckman Metabolic Measurement Cart calibrated before and after each test. Heart rate was recorded each minute from an electrocardiograph.

TG subjects trained four times weekly over a five week period for a total of 21 sessions. The first 15 sessions consisted of two circuits and the last six sessions of three circuits on variable hydraulic resistance stations (Hydrafitness Industries Ltd.). The program was designed to achieve a 1:2 work-relief ratio within each circuit. Each circuit consisted of two 20 second sets maximal exercise at six stations which emphasized the following movement patterns: knee flexion and extension; hip flexion and

extension; elbow flexion and extension; shoulder flexion and extension; and ankle plantar flexion.

Circuits were separated by four minutes of active (walking) recovery. Subjects worked in pairs and verbal encouragement was provided to ensure maximal effort and high quality contractions at all times. Subjects were required to complete at least 20 repetitions each 20-second exercise period. This approximated limb velocities of 3.1 $rad \cdot s^{-1}$. When subjects consistently exceeded 23 repetitions in 20 seconds, the resistance setting was increased by one increment. Each training session was monitored by a supervisor who adjusted and recorded resistance settings, numbers of repetitions, and precisely timed the work and rest intervals. Control subjects were permitted up to three moderate intensity aerobic exercise sessions and one resistance exercise session per week to facilitate maintenance of initial fitness levels.

For analysis, TG subjects were blocked on initial VO_2 max into two groups designated High Aerobic Power (HAP) and Low Aerobic Power (LAP). Similarly, for each peak torque variable, TG subjects were blocked into groups which represented high (HPT) and low (LPT) initial peak torque performance.

Data were analyzed with a two-way analysis of variance for repeated measures. Where post-hoc means comparisons

were warranted, such contrasts were made using two-tailed paired or independent t-tests.

Results

Characteristics of training and control subjects prior to the study are reported in Table II-1. Group means were similar for age, weight, height, and peak knee extension torques at both test speeds. Since no contralateral differences were observed on any strength variables in either group only values for the right leg have been reported in the interest of simplicity.

Peak torques for knee extension and flexion for both training and control groups before and after the study period are displayed in Table II-2. Increased ($p < 0.05$) peak torques in both extension and flexion at 3.14 $rad \cdot s^{-1}$ were observed for the training group. A decrease ($p < 0.05$) in peak flexion torque at 0.52 $rad \cdot s^{-1}$ was observed for the control group. No significant differences in performance on any peak torque variable were observed between training and control groups prior to the study.

Results of peak torque assessments for the TG with respect to initial aerobic power are displayed in Table II-4. No differences were observed between HAP and LAP group means prior to training. Improved ($p < 0.05$) peak torques in knee flexion at 0.52 $rad \cdot s^{-1}$ and both extension and flexion at 3.14 $rad \cdot s^{-1}$ were observed for the LAP group while the HAP group improved only in knee flexion torque at

3.14 rad·s^{-1}. A significant interaction effect was observed between the aerobic power groups in knee extension torque at 3.14 rad·s-1

Results of peak torque assessments for the TG with respect to initial strength on each torque variable are displayed in Table II-4. This analysis indicated a decline in peak torque in knee extension at 0.52 rad·s^{-1} and an improvement in knee flexion at 3.14 rad·s^{-1} for the HPT group. The LPT group improved peak torque output in knee flexion at 0.52 rad·s^{-1}, and in both extension and flexion at 3.14 rad·s^{-1}. Significant interaction effects were observed between groups on each torque variable except knee flexion at 0.52 rad·s^{-1}.

Table II-1

Characteristics of the Training (TG) and Control (CG) Groups Before the Training or Control Programs

Variable	TG (n=12)	CG (n=8)
Age (years)	18.8 (0.9)	21.4 (0.8)
Weight (kg)	72.2 (1.6)	76.6 (2.3)
Height (cm)	177.9 (2.5)	179.9 (2.4)
Knee extension torque @ 0.52 rad·s^{-1} (N·m)	239 (9)	243 (12)
Knee extension torque @ 3.14 rad·s^{-1} (N·m)	146 (4)	152 (9)

Values are Mean +/- (S.E.)

Table II-2

Peak Knee Extension and Flexion Torques for Training (TG) and Control (CG) Groups

Peak Torque (N·m)	TG (n=12) Pre	TG (n=12) Post	CG (n=8) Pre	CG (n=8) Post
Extension @ 0.52 rad·s^{-1}	239 (8)	230 (8)	243 (11)	233 (11)
Flexion @ 0.52 rad·s^{-1}	156 (7)	164^{d} (7)	169^{c} (9)	153^{cd} (7)
Extension @ 3.14 rad·s^{-1}	146^{a} (4)	156^{a} (4)	152 (8)	149 (6)
Flexion @ 3.14 rad·s^{-1}	109^{b} (5)	119^{b} (4)	120 (7)	120 (6)

Values are Mean +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups or significant ($p < 0.05$) interaction effects between groups over either training or control.

Table II-3

Pre-Training Characteristics of the High (HAP) and Low (LAP) Aerobic Power Groups

	HAP (n=6)	LAP (n=6)
Weight (kg)	71.6 (2.0)	72.3 (2.4)
Height (cm)	176.8 (2.1)	178.9 (2.8)
$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	60.5[a] (0.9)	53.9[a] (0.4)

Values are Mean +/- (S.E.).

Paired letters indicate significant ($p < 0.001$) differences between groups.

Table II-4

Peak Knee Extension and Flexion Torques for the High (HAP) and Low (LAP) Aerobic Power Groups Before and After Training

Peak Torque (N·m)	HAP (n=6) Pre	HAP (n=6) Post	LAP (n=6) Pre	LAP (n=6) Post
Extension @ 0.52 rad·s^{-1}	249 (15)	239 (9)	228 (8)	221 (13)
Flexion @ 0.52 rad·s^{-1}	154 (12)	157 (7)	158^{b} (11)	172^{b} (13)
Extension @ 3.14 rad·s^{-1}	146 (7)	156^{e} (5)	140^{c} (5)	156^{ce} (6)
Flexion @ 3.14 rad·s^{-1}	105^{a} (7)	118^{a} (5)	113^{d} (7)	121^{d} (6)

Values are Mean +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups and significant ($p < 0.05$) interaction between groups after training.

Table II-5

Peak Knee Extension and Flexion Torques for High (HPT) and Low (LPT) Initial Strength Groups Before and After Training

Peak Torque ($N \cdot m \cdot kg^{-1}$)	HPT (n=6) Pre	HPT (n=6) Post	LPT (n=6) Pre	LPT (n=6) Post
Extension @ 0.52 $rad \cdot s^{-1}$	3.60^{a} (0.14)	3.31^{ab} (0.15)	3.02 (0.05)	3.03^{b} (0.12)
Flexion @ 0.52 $rad \cdot s^{-1}$	2.43 (0.07)	2.47 (0.05)	1.86^{c} (0.06)	2.03^{c} (0.05)
Extension @ 3.14 $rad \cdot s^{-1}$	2.21 (0.05)	2.26^{e} (0.07)	1.84^{d} (0.02)	2.05^{de} (0.06)
Flexion @ 3.14 $rad \cdot s^{-1}$	1.68^{f} (0.05)	1.75^{fh} (0.04)	1.35^{g} (0.05)	1.55^{gh} (0.06)

Values are Mean +/- (S.E.)

HPT and LPT means are all significantly ($p < 0.05$) different prior to training.

Paired letters indicate significant ($p < 0.05$) differences within groups or significant interaction between groups after training.

Discussion

No changes in body weight were observed for either group over the training or control period. This result was not unexpected since all subjects were habitually physically active and judged to be at or near an optimal body weight prior to the study. It is worthy of mention, however, since the blocking TG subjects into sub-groups with respect to initial fitness levels was done on the basis of aerobic power or peak torques expressed relative to body weight.

A decline ($p < 0.05$) in peak torque in knee flexion at 0.52 $rad \cdot s^{-1}$ was observed in the control group. Performance on the other torque variables did not change over the control period. A significant interaction effect between training and control groups was observed on this variable, although the increased peak torque of the training group was not significant by post-hoc analysis. The training group did, however, improve peak torque output in both knee flexion and extension at 3.14 $rad \cdot s^{-1}$. This test speed was very similar to the angular velocities of the leg about the knee joint during training. These data would suggest a velocity-specific adaptation to the resistance training stimulus. The observed decline in peak torque in knee extension and the insignificant increase in knee flexion torque at the slow test speed for training subjects does not support the suggestion that high-speed training increases torque output at slower speeds (Kanehisa and Miyashita 1983;

Coyle et al. 1981; Lesmes et al. 1978). These data would support the finding of Caiozzo et al. (1981). In that investigation the high-speed training group demonstrated enhanced torque outputs at and near the training speed, but not at slow velocities. One major difficulty in comparing results from various studies is that categorization of training velocities as fast or slow is usually different. In the work of Caiozzo et al. (1981) for example, the "fast" training group worked at 4.19 $rad \cdot s^{-1}$ or approximately 33% faster than the subjects in the present study (3.14 $rad \cdot s^{-1}$). Lesmes et al. (1978), and Kanehisa and Miyashita (1983), however, did utilize training speeds of 3.14 $rad \cdot s^{-1}$. Another confounding issue may be that of "volume" of training. Intensity is relatively constant in that a maximal effort (usually under isokinetic conditions) is consistently reported, however, frequency and duration of exercise sessions are rarely similar, and training program duration has varied from four to eight weeks. A variety of mechanisms have been suggested to explain velocity-specific training responses including enhancement of motoneuron activation or reduction of neuromuscular inhibition (Caiozzo et al. 1981; Sale 1986), hypertrophy of selected fiber populations (Coyle et al. 1981), and enhanced myofibrillar ATPase activity (Belcastro et al. 1981). The extent to which any or all of these mechanisms may be affected by

frequency and duration of exercise and training program length is unclear.

When training responses are evaluated with respect to initial fitness levels, some clarification of this issue may occur. In direct contrast to the earlier suggestion that higher aerobic power might enhance the adaptation to high-intensity strength training are the results reported in Table 3. In fact, the HAP group improved peak torque only in knee flexion at 3.14 $rad \cdot s^{-1}$ while the LAP demonstrated enhanced torque output on three of four variables, including knee flexion torque at 0.52 $rad \cdot s^{-1}$. The same pattern is evident when TG subjects were blocked into high (HPT) and low (LPT) initial strength groups on each of the peak torque variables. Although separate groupings were formed on each of the initial fitness variables, group membership tended to remain quite similar. Therefore it would appear that initial fitness, even in a subject pool which was reasonably well-trained initially, may be a major factor in evaluating training responses. Present data would suggest that the majority of the training effects reported in Table II-2 for the entire training group were in fact due to relatively large amounts of adaptation from those training subjects whose initial fitness levels were relatively low. It is interesting to note (Tables II-4 and II-5) that TG subjects with low initial fitness improved torque outputs in knee flexion at the slow test speed which was not specific to the

training velocity while high-fit TG subjects did not. While such comparisons may be of little direct value, it is also interesting to note that even the low initial strength group ranked at the 60th and 45th percentile for knee extension and flexion respectively at 3.14 $rad \cdot s^{-1}$ when compared to elite amateur and professional ice-hockey players (Smith et al. 1981). Therefore, while in this study they were ranked as low strength, in contrast to the sedentary subjects used in other studies, it is likely that they would actually be relatively strong.

While the results of this investigation do not appreciably clarify the controversy over velocity-specific strength adaptation, several important points have been made. First, a five week high-intensity program did enhance peak torque outputs in a group of previously well-trained subjects when tested at an angular velocity which was specific to the training velocity. Secondly, the hypothesis that well-developed aerobic power would enhance adaptation from a high-intensity strength training program was not supported. It may be possible that the LAP group (mean VO_2 max = 53.9 $ml \cdot kg^{-1} \cdot min^{-1}$) exceeded a possible threshold or minimum level of aerobic power necessary to facilitate adequate recovery from the exercise stimulus. If this was the case, further study using subjects with yet lower aerobic power or, alternately, a more demanding work: recovery ratio during exercise sessions might test this hypothe-

sis more definitively. Finally, although the "low-fit" subjects in the present study may be well-trained in comparison to the average population, they still accounted for the majority of the TG adaptation to the training program. In light of the different responses relative to initial fitness levels reported above, it may be possible that some of the controversy over velocity-specific training effects is due to the characteristics of the subject pools in the various studies. It is therefore recommended that consideration of initial fitness levels be given when both designing strength training studies and when reporting results.

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CHAPTER III

The Influence of Velocity - Specific Resistance Training on the in Vivo Torque-Velocity Relationship and the Cross-sectional Area of the Knee Extensors[1]

Introduction

Strength training has been widely used to enhance athletic performance. The question of velocity-specific training effects has been addressed by a number of investigators (Caiozzo et al. 1981; Coyle et al. 1981; Kanehisa and Miyashita 1983; Petersen et al. 1984) and has been discussed in review articles (Sale and MacDougall 1981, Sale 1986). However, in spite of this attention, the nature of, or even the existence of such velocity-specific adaptations has not been sufficiently clarified. The categorization of various angular velocities as either "fast" or "slow", the nature of the training programs, the limitations of assessment equipment, and variable characteristics of the subject pools does not readily permit comparison of results between studies.

As well, the mechanisms of adaptation to resistance training at higher velocities are not well understood. In response to high resistance training (which necessitates slow contractions) increased whole muscle cross-sectional

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[1] A version of this chapter has been submitted for publication: Petersen, S.R., Bagnall, K.M., Wenger, H.A., Reid, D.C., Castor, W.R., Quinney, H.A. Int. J. Sports Med.

area has been consistently reported (Luthi et al. 1986; Young et al. 1983) to complement the observed increased in force (or torque) production. Other investigators have reported increased fiber cross-sectional area, especially in Type II fibers (MacDougall et al. 1980; Thorstensson 1976) and increased myofibrillar packing density (MacDougall et al. 1980). Interestingly, Luthi et al. (1986) have reported increased whole muscle area but insignificant changes with respect to individual fiber areas. This may be due to the fact that measurement of fiber cross-sectional area alone does not consider increased amounts of intramuscular connective tissue.

Increased areas of Type II muscle fibers have been reported (Coyle et al. 1981; Costill et al. 1979) following training of the knee extensors at 5.24 and 3.14 rad $\cdot s^{-1}$. However, these results are not confidently offered as explanation for the enhanced performance and there is evidence to suggest that much of the training effect may be mediated by neurological adaptation (Caiozzo et al. 1981; Coyle et al. 1981; Sale 1986). This would be reasonable considering the untrained state of the subject pools utilized in the majority of the investigations.

It would seem logical that high-velocity resistance training would be most appropriate for athletes who perform at relatively high speed. It should also follow that neurological adaptation might be minimized if the exercise

stimulus was in motor patterns which mirrored those utilized in competition. In many sports, athletes must work throughout a range of velocities and against a variety of resistances. This study was designed to investigate effects of either high or low velocity resistance training on the in vivo torque-velocity relationship and the cross-sectional area of the knee extensors in a group of previously well-trained subjects.

Method

Thirty apparently healthy college-aged males provided informed consent to participate in the study. All subjects were affiliated with either University football or rowing programs, however the study was designed to coincide with the off-season period for both sports. All subjects had past experience with resistance training.

Peak knee extension torque was assessed at angular velocities of 0.52, 1.05, 1.57, 2.08, 2.62, 3.14, 3.66, and 4.19 $rad \cdot s^{-1}$ using a Cybex II Isokinetic Dynamometer system (Lumex, Inc.). Stabilization and alignment procedures were standardized for each subject and calibration of the system was performed according to the manufacturer's recommendations. Subjects were familiarized with the dynamometer system and the test procedures on a separate occasion. A minimum of four continuous repetitions were performed at each velocity with 60 seconds rest provided between sets of

exercise. The first torque curve was omitted from the results and the highest value from subsequent trials was recorded as peak torque. All computations of peak torque were performed by the same investigator. An optimal damping setting of two was selected on the Cybex II chart recorder.

On a separate day a single image of the thigh was taken using computerized tomography (CT) scanning techniques (GE Model 8800). The scan location was determined by surface anatomical references to be at mid-femur. The scan location was confirmed by use of a scout scan which recorded precisely the distance from the site of the image to a predetermined landmark on the distal end of the femur. This procedure was repeated at the time of the second scan to ensure that the images were in fact overlapping. No exercise was permitted on the day of the CT scan assessments.

Black and white photographs were made from the CT images and from these a co-investigator determined the boundaries of the anterior thigh compartment musculature without knowledge of group membership or whether the scan was taken before or after training or control. Cross-sectional area (CSA) of the anterior compartment was then determined through the computer planimetry system associated with the GE 8800 scanner.

Subjects were assigned to either a high-velocity (HVR), low-velocity (LVR), or control (CG) group. Groups were

equated on initial peak torque at each of the two training velocities (1.05 and 3.14 $rad \cdot s^{-1}$) and body weight. Past experience has indicated the importance of initial fitness levels, and torque relative to body weight may be a more sensitive predictor than absolute torque.

Subjects trained on alternate days over a six week period for a total of 21 sessions. A thorough introduction and mock training session was held prior to actual commencement of the program. The resistance circuit consisted of two 20 second sets of all-out exercise separated by 20 seconds of relief at each of the following stations: unilateral seated knee extension and flexion [2]; bilateral seated leg press (hip extension and knee extension); unilateral supine hip extension and flexion; unilateral supine hip abduction and adduction; and bilateral inclined leg drive (hip and knee extension and ankle plantar flexion). Resistance at each station was supplied by variable hydraulic cylinders (Hydra-fitness Industries Ltd.) which were adjusted as necessary to maintain consistent angular velocities of the involved joints at approximately either 1.1 $rad \cdot s^{-1}$ (LVR) or 3.1 $rad \cdot s^{-1}$ (HVR). The cylinders do not provide an isokinetic loading system, however, calculation of the range of motion for each subject at each station, and careful monitoring of the number of repetitions in a fixed time period permits quite accurate estimation of average angular velocity. Subjects must be consistently

motivated to exercise at maximum intensity and to maintain a full range of motion. With training, subjects may exceed the number of repetitions necessary to maintain the desired velocity. At this time, the cylinder would be adjusted to provide increased resistance and thus decrease the velocity appropriately.

Initially two circuits were completed each session and during the second week of training this was increased to three circuits. Each session was monitored by a supervisor who recorded resistance settings and repetitions, and precisely timed the exercise and relief intervals.

Control group subjects were permitted up to three low-intensity aerobic and one resistance exercise session per week to maintain initial fitness levels. All controls complied with the request to refrain from any systematic application of high-intensity exercise stimuli over the control period.

Data were analyzed with 2-way analysis of variance for repeated measures (Winer, 1972). Post-hoc means comparisons within groups were made with two-tailed paired t-tests. Group means for results of all performance tests were the same for both legs, therefore in the interest of simplicity, only results from the right legs were reported in the results which follow.

Results

Characteristics of all study groups are reported in Table III-1. No differences were found on either age, height, or weight between groups. Body weight did not change for any of the groups over the study period.

Peak knee extension torques at the eight angular velocities tested are displayed in Table III-2. No differences were observed in group means on any variable prior to commencement of the study. Following the training program, the HVR group improved ($p < 0.05$) peak torque at 0.52, 2.62, 3.14, 3.66, and 4.19 $rad \cdot s^{-1}$. The LVR group improved ($p < 0.05$) peak knee extension torque at all eight angular velocities. Following the control period a decrease ($p < 0.05$) in peak torque was observed for the CG at 0.52 $rad \cdot s^{-1}$ only. These results are also displayed in Figures III-1, III-2, and III-3.

Cross-sectional areas of the quadriceps femoris muscle group before and after the training or control periods are reported in Table III-3. Increased ($p < 0.05$) cross-sectional area was observed for both training groups, and no change was observed for the controls.

In order to establish the relationship between muscle cross-sectional area and peak torque, and to detect changes in this relationship consequent to training the ratio of peak torque and cross-sectional area of the quadriceps femoris group at each angular velocity before and after

training was calculated. These data are displayed in Table III-4. Significant ($p < 0.05$) changes in this relationship were observed for the LVR group at angular velocities of 0.52 and 1.05 $rad \cdot s^{-1}$ only.

Table III-1

Characteristics of High (HVR) and Low (LVR) Velocity Training and Control (CG) Groups Prior to the Training Program

Variable	HVR (n=10)	LVR (n=10)	CG (n=10)
Age (years)	21.0 (0.5)	23.1 (1.0)	23.5 (1.0)
Height (cm)	181.9 (1.5)	184.4 (1.8)	183.4 (2.6)
Weight (kg)	84.1 (3.6)	80.6 (2.8)	81.4 (2.6)

Values are means +/- (S.E.)

Table III-2

Peak Knee Extension Torques for High Velocity (HVR) and Low Velocity (LVR) Training Groups and the Control Group (CG)

Angular Velocity ($rad \cdot s^{-1}$)	Peak Torque in Knee Extension ($N \cdot m$)					
	HVR (n=10)		LVR (n=10)		CG (n=10)	
	Pre	Post	Pre	Post	Pre	Post
0.52	257[a] (14)	282[a] (15)	251[f] (9)	291[f] (12)	261[n] (11)	247[n] (11)
1.05	244 (11)	255 (11)	237[g] (7)	271[g] (7)	235 (10)	228 (10)
1.57	221 (10)	226 (8)	212[h] (7)	234[h] (7)	210 (9)	208 (9)
2.09	200 (9)	201 (8)	196[i] (6)	209[i] (6)	190 (9)	192 (7)
2.62	177[b] (5)	185[b] (6)	173[j] (4)	190[j] (6)	171 (8)	167 (6)
3.14	161[c] (6)	172[c] (6)	163[k] (4)	175[k] (6)	158 (8)	155 (6)
3.66	149[d] (7)	159[d] (6)	150[l] (4)	162[l] (5)	147 (7)	145 (5)
4.19	140[e] (7)	149[e] (5)	138[m] (4)	153[m] (5)	141 (7)	136 (5)

Values are Means +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups following training or control

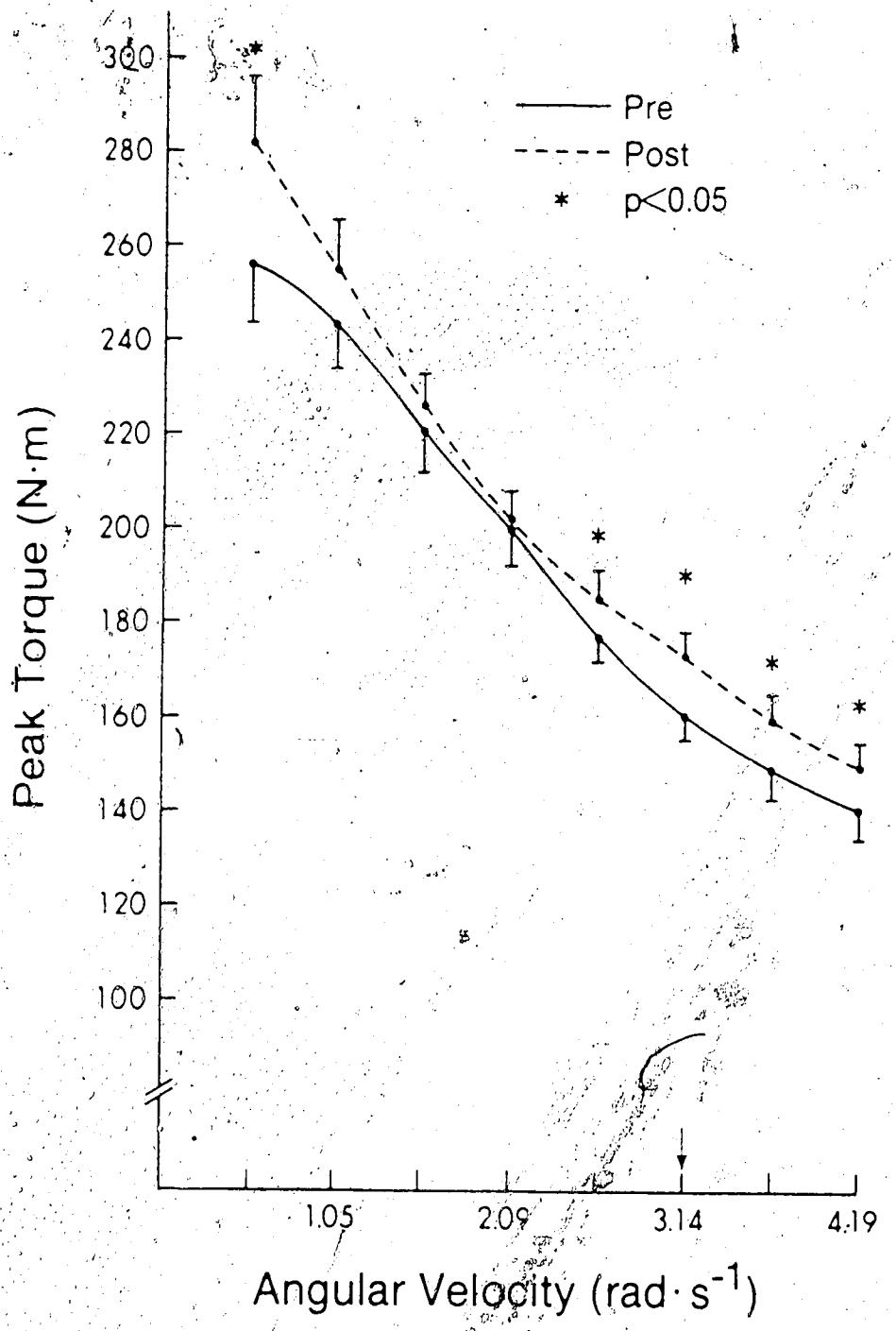

Fig. III-1. Peak knee extension torques (Mean ± S.E.) at selected angular velocities for the High-Velocity training group (n=10).
Arrow indicates training velocity.

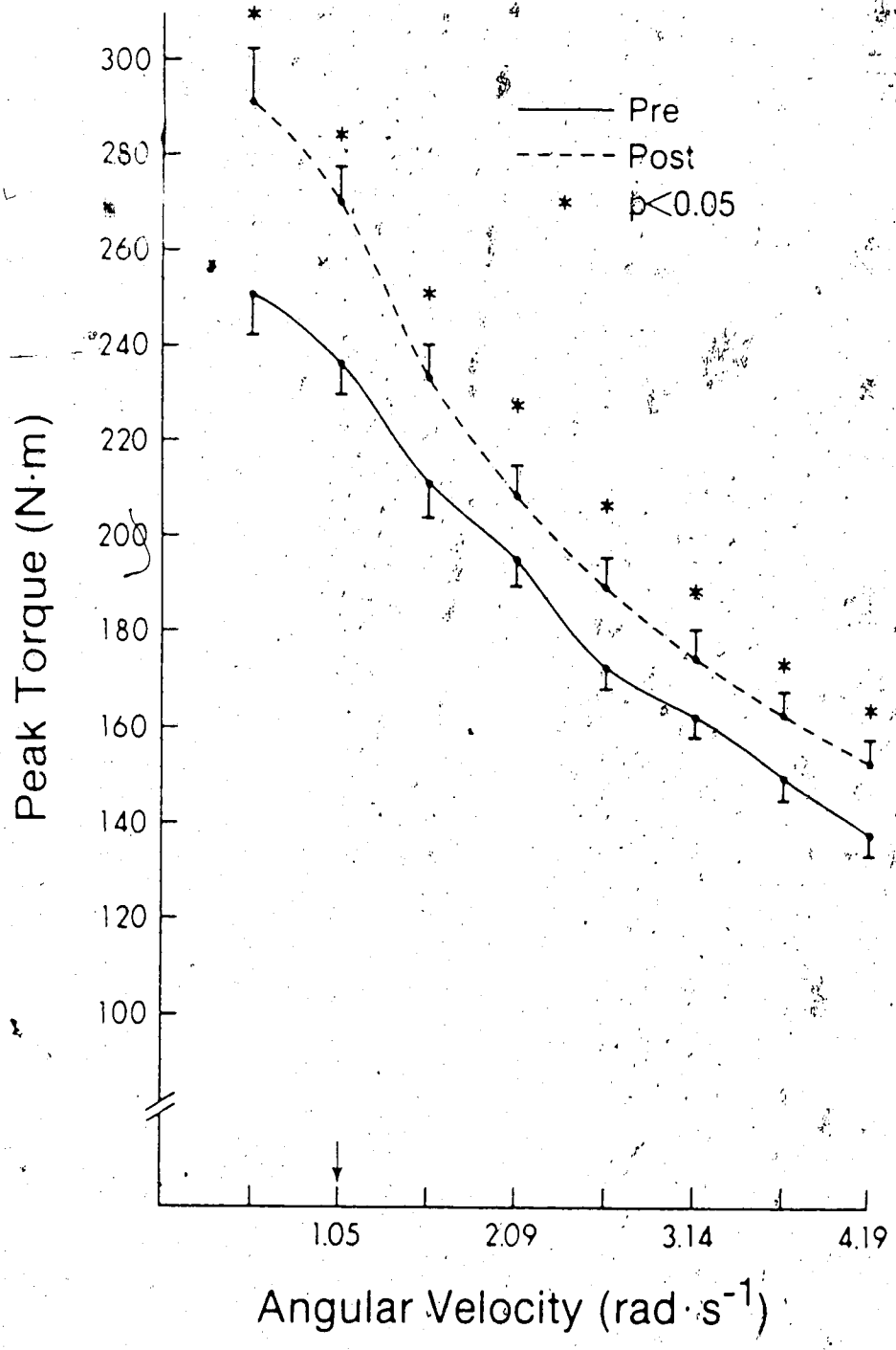

Fig. III-2. Peak knee extension torques (Mean ± S.E.) at selected angular velocities for the Low-Velocity training group (n=10).
Arrow indicates training velocity.

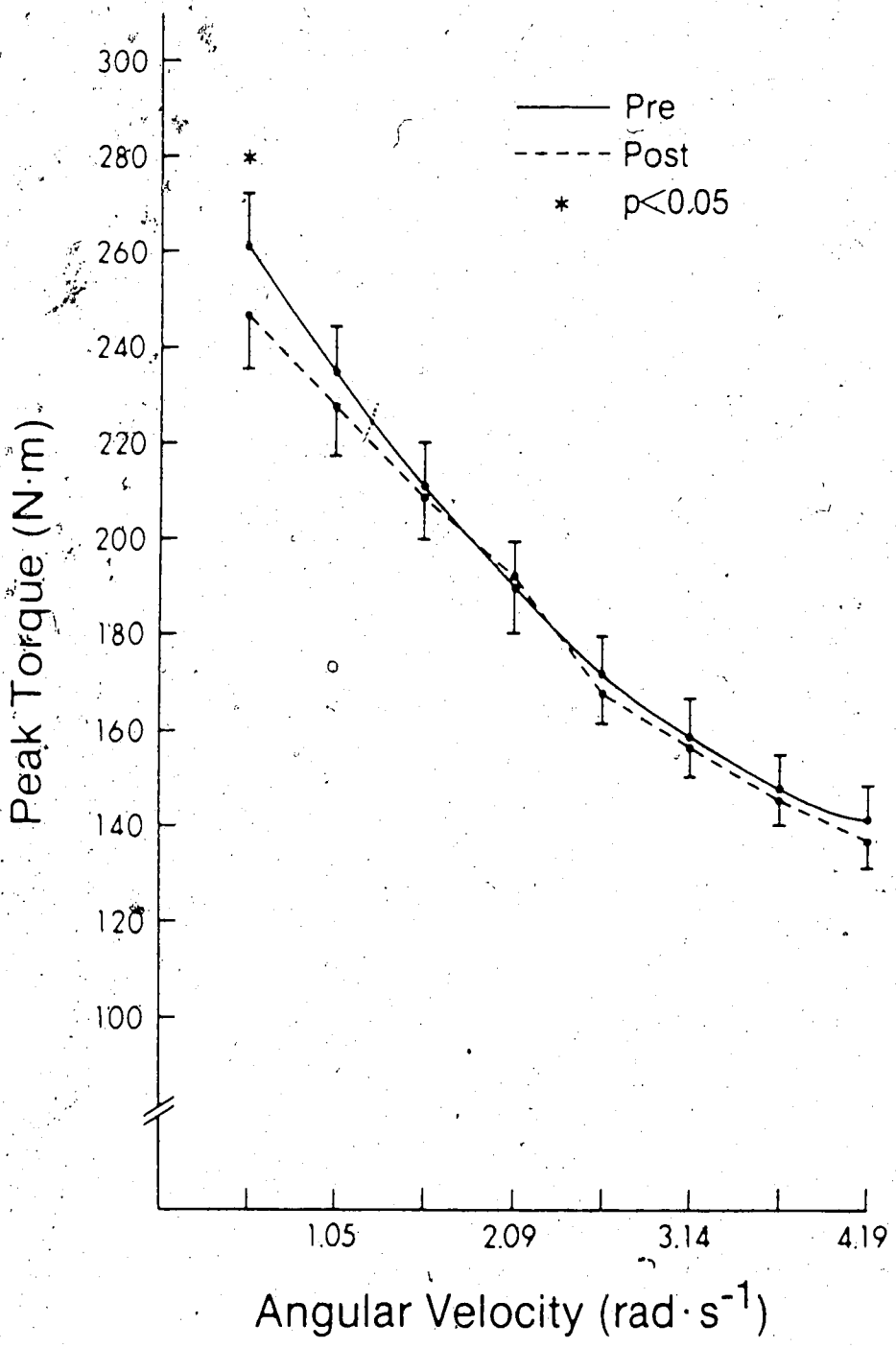

Fig. III-3. Peak knee extension torques (Mean ± S.E.) at selected angular velocities for the Control group (n=10).

Table III-3

Cross-sectional Area of the Quadriceps Femoris for High (HVR) and Low (LVR) Velocity Training and Control (CG) Groups

Anterior Thigh Compartment Area (cm^2)	HVR (n=9)	LVR (n=9)	CG (n=6)
Pre-training	89.97[a] (2.61)	88.42[b] (3.77)	85.29 (3.41)
Post-training	94.17[a] (3.13)	93.87[b] (3.85)	85.97 (3.50)

Values are Means +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups after training.

Table III-4

Peak Knee Extension Torque Expressed Relative to Quadriceps Femoris Cross-sectional Area ($N \cdot m/cm^2$) for High (HVR) and Low (LVR) Velocity Resistance Training and Control (CG) Groups

Angular Velocity ($rad \cdot s^{-1}$)	HVR (n=9)		LVR (n=9)		CG (n=6)	
	Pre	Post	Pre	Post	Pre	Post
0.52	2.92 (0.14)	3.07 (0.13)	2.79[a] (0.09)	3.14[a] (0.12)	3.04 (0.08)	3.01 (0.03)
1.05	2.79 (0.09)	2.76 (0.06)	2.66[b] (0.09)	2.92[b] (0.08)	2.85 (0.06)	2.76 (0.06)
1.57	2.53 (0.08)	2.47 (0.06)	2.38 (0.09)	2.53 (0.07)	2.56 (0.05)	2.55 (0.05)
2.09	2.28 (0.06)	2.18 (0.06)	2.22 (0.06)	2.25 (0.05)	2.28 (0.06)	2.31 (0.05)
2.62	2.13 (0.10)	2.02 (0.07)	1.95 (0.06)	2.05 (0.06)	2.05 (0.07)	1.99 (0.04)
3.14	1.84 (0.04)	1.88 (0.07)	1.85 (0.06)	1.88 (0.04)	1.89 (0.07)	1.86 (0.06)
3.66	1.71 (0.05)	1.74 (0.06)	1.70 (0.05)	1.75 (0.04)	1.75 (0.06)	1.76 (0.06)
4.19	1.60 (0.06)	1.63 (0.06)	1.61 (0.06)	1.65 (0.03)	1.67 (0.07)	1.65 (0.05)

Values are means +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups following training or control.

Discussion

It has been suggested (Ikai and Steinhaus 1961) that some type of neural inhibition prevents muscle from generating maximal tension, and that under special circumstances (eg. hypnosis) which might reduce this inhibition, performance is increased. It has also been suggested that this tension-limiting inhibition is greatest at slow contractile velocities (Perrine 1986) and consequently, the force-velocity relationship in the intact muscle does not follow the classic pattern established for isolated muscle (Hill 1938). However, there is some evidence that with training the inhibitory effect may be reduced and that the in vivo force-velocity relationship then begins to more closely resemble that of the in vitro muscle (Caiozzo et al. 1981). This hypothesis may be supported partially by the results of the present study, and may then offer an explanation for the improvement in peak torque observed in the HVR group at the slowest contractile velocity (0.52 $rad \cdot s^{-1}$). As well, it may offer a partial explanation of the decline in peak torque at that same velocity observed in the control group. That is, perhaps this inhibition effect is increased during a period of relative inactivity.

The increased torque outputs for the HVR group at higher velocities, at and around the training velocity are somewhat consistent with the findings of Caiozzo et al. (1981) although the "fast" training velocity in that study

was 4.19 rad·s^{-1} rather than approximately 3.14 rad·s^{-1} in the present study. They are not consistent with the results of other work (Lesmes et al. 1978; Kanehisa and Miyashita 1983) where training at 3.14 rad·s^{-1} improved peak knee extension torque at all speeds selected for assessment below the training velocity. The latter studies clearly suggest a pattern of transfer from fast training to slow performance, while, with the exception of peak torque increases at 0.52 rad·s^{-1}, present data do not.

The alterations in peak torque for the LVR group again coincide with those reported by Caiozzo et al. (1981). Although in that study the "slow" training velocity was 1.68 rad·s^{-1} (compared to approximately 1.05 rad·s^{-1} in the present study) the pattern of improvement at all test velocities above and below (0 - 4.19 rad·s^{-1}) the training velocity is consistent. Similar results were reported by Kanehisa and Miyashita where the group which trained at 1.05 rad·s^{-1} improved performance at all test velocities up to 5.24 rad·s^{-1}.

Interestingly, the absolute changes in peak torque for the LVR group appear to be greater at all test speeds than those observed for the HVR group. This is in contrast to the results reported by Kanehisa and Miyashita (1983) where the group who trained at 3.14 rad·s^{-1} improved far more at faster test speeds than the group who trained at 1.05 rad·s^{-1}. This may be partially due to differences in

initial fitness of their subjects at high movement velocities. Simple calculation of performance relative to body weight indicates a difference of 11% in initial levels at high test speeds which is not apparent at lower test speeds where improvement after training was relatively equal. The authors have not considered this possibility in their discussion. In the present study subjects were equated on peak torque by design at the two training velocities and coincidentally over the remainder of test velocities as well. Therefore, differences in magnitude of adaptation should not be due to initial fitness differences.

The increased performance may be partially explained by the observed changes in muscle cross-sectional area. The 5 - 6% increase in knee extensor cross-sectional area is consistent with other reports of change following five or six weeks of heavy resistance training (Young et al. 1983; Luthi et al. 1986). Coyle et al. (1981) and Costill et al. (1979) have reported increased cross-sectional area of Type II fibers following six and seven weeks of training the knee extensors at 5.24 and 3.14 $rad \cdot s^{-1}$ respectively. Certainly, an increase in cross-sectional area of muscle would help to explain the pattern of increased performance for the LVR group. However, if muscle hypertrophy alone were responsible for the change, then the pattern should be consistent across all test velocities, and this is clearly not so with the HVR group. As well, it would not explain

the decreased peak torque at 0.52 $rad \cdot s^{-1}$ for the CG considering that no change in cross-sectional area was evident. It would appear that other factors than muscle cross-sectional area are involved in the training adaptation.

Barany (1967) has reported a strong relationship between the speed of shortening in muscle and the activity of myofibrillar ATPase. Belcastro et al. (1981) have reported increased activities of this enzyme following six weeks of cycling at either high or low pedalling frequency, however, they also suggest that there may be a differential effect on the myofibrillar ATPase activities of different fiber types based on recruitment of fibers at different velocities. This may be supported by Faulkner et al. (1986) who reported that even at relatively slow velocities fast muscle contributes 2.5 times more than slow muscle to total power output. They go on to report that at velocities of shortening greater than those attainable by slow fibers, the power developed by mixed muscle is due exclusively to the action of fast fibers. Certainly the in vivo velocities discussed in the present study are relatively slow, but it would follow that in the mixed muscle of the knee extensors the different contribution of the fibers to total power output may result in differential contractile adaptations.

Another possibility for differential training effects with respect to training velocity may be in neural adapta-

tion. Electromyographic studies have attempted to monitor possible increases in motor unit activation following st n training. The integrated EMG has been shown to increase in strength-trained individuals (Komi et al. 1978; Hakkinen and Komi, 1983). As well, the responses to supramaximal nerve stimulation at rest and during voluntary contractions have been compared and changes in reflex potentiation with strength training have been observed (Sale et al. 1983a). Cross-sectional studies have indicated that reflex potentiation may be enhanced in weight-lifters (Sale et al. 1983b) and sprinters (Upton and Radford, 1975). Whether this response is affected by contractile velocity is left to speculation, however, since it is well known that motor units are composed of fibers of one type (Brooks and Fahey, 1984) and that motor unit activation is dependent upon the nature of the exercise (Burke 1986) it may follow that enhancement of motor unit recruitment and/or firing frequency may be mediated by training velocity. Interestingly, Appell (1984) recently reported increased proliferation of motor end-plates in mouse muscle which was myogenically induced. End-plate regions were enlarged in size after seven days and increased in number after 14 days. This phenomenon may help explain the increased performance early in strength training programs which is often attributed to neural adaptation. If the end-plate adaptation is caused by muscular activity, the possible effect of contractile

velocity on muscle recruitment may cause differential adaptation.

The ratio between peak torque and muscle cross-sectional area (Table III-4) was calculated to show the relationship between these two variables. Presumably, if cross-sectional area was the major factor responsible for changes in peak torque following training, then this relationship should remain consistent. If other factors were primarily responsible for enhanced performance, then the relationship should change (in this case, numerically increase). The ratio remained constant with the exception of significant ($p < 0.05$) increases observed for the LVR group at the two slowest test velocities. This would tend to support the contention that low-speed training reduces neural inhibition of tension development, especially at slow test velocities (Perrine 1986). This concept is supported by the reported increases in EMG activity at slow test speeds consequent to high resistance training (Komi 1986). These alterations in the torque-area ratio presumably reflect a significant influence on performance from neural adaptation to high resistance training. On the other hand, the general stability of the ratio would support existence of a strong relationship between peak tension and cross-sectional area of muscle.

Clearly, the nature of velocity-specific adaptation to resistance training is a complex issue and is very likely

mediated by morphological, biochemical, and neural factors which may or may not follow similar time courses over short-term training programs. In this study, it was hypothesized that subjects with previous weight-training experience and whose involvement with sports which demand regular high-intensity and high-velocity muscular contractions might show minimal neural adaptation. The inability to explain the performance changes based on increased muscle cross-sectional area alone has led to speculation that neural adaptation still remains a very important training adaptation. The potential differential effects of neural adaptation with respect to contractile velocity cannot be explained by the results of this study.

In summary, resistance training at high-velocity seems to be no more effective at increasing high-velocity peak torque than does training at low-velocity. The LVR program appears to have a greater and more uniform effect on the in vivo torque velocity relationship than the HVR program. The results of this study do not clarify the apparent controversy over velocity-specific training effects or transfer of enhanced function from high-speed training to low-speed performance (or vice versa). The existence of such phenomena remains an intriguing question for further study, and investigators may wish to consider a more mechanistic approach as well as considering the appropriateness of peak torque as a performance indicator. Within the limitations

of current instrumentation, such variables as the rate of peak torque generation or short-term power output may be better reflections of velocity-specific performance adaptations in skeletal muscle.

The findings of the present study do have some practical implications beyond the speculation that high-velocity training may improve high-velocity athletic performance. The potential for increased cross-sectional area of muscle and some increases in force expression with high-velocity resistance training may be most applicable in a rehabilitation setting where the high compressive forces on joint surfaces and high tractional loads on supporting ligaments which are associated with heavy resistance training are contraindicated.

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CHAPTER IV

The Influence of High-Velocity Resistance Training on $\dot{V}O_2$ max[1]

Introduction

Although many athletes use resistance training to enhance their muscular strength and power it has not generally been used to improve aerobic power. It has been suggested that the intermittent nature of resistance training with heavy weights, low repetitions, and frequent rest periods does not encourage an aerobic power training effect (Gettman et al., 1978). Byrd and Barton (1973) reported exercise and relief heart rates of 152 and 119 beats·min^{-1} for experienced lifters over an hour of exercise. They suggested that heart rate response of this magnitude may elicit a change in maximum oxygen consumption ($\dot{V}O_2$ max) in running or swimming programs, but not in weight lifting programs, because of the short duration of the program, impaired local blood flow during heavy lifting, or assessment methods which do not reflect specificity of training effects. This is implied in the work of Hickson et al. (1980) who reported small increases in the absolute $\dot{V}O_2$ max after a 10 week program of heavy resistance training

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[1] A version of this chapter has been submitted for publication:

Petersen, S.R., Miller, G.D., Quinney, H.A., and Wenger, H.A. J. Orthop. Sport Phys. Ther.

which was evident during cycling exercise, but not on the treadmill.

Circuit training usually involves many repetitions of light weights with short rest intervals between exercise stations. Wilmore et al. (1978) and Gettman et al. (1982, 1980) have reported enhanced $\dot{V}O_2$ max following 10, 12, and 20 week programs of circuit weight training, while Allen et al. (1976) reported no change in $\dot{V}O_2$ max over 12 weeks of training.

It seems likely that the nature of the circuit and the choice of resistance apparatus would influence the exercise intensity and therefore the effect on aerobic power. Recent developments in resistance training equipment provide accommodating resistance from variable hydraulic cylinders which allow safe, high-velocity movements. By demanding an "all-out" effort during exercise intervals, and by shortening the relief intervals between stations, a relatively high exercise intensity may be achieved.

Improvements in $\dot{V}O_2$ max have been shown to occur in 10 to 20 days of training (Hickson et al. 1981; Smith and Wenger, 1980). Therefore, the main purpose of this study was to determine if a high velocity resistance circuit could enhance aerobic power over 5 weeks. Since this mode of training may be most appropriate for athletes who may already be well-trained, the second purpose of the study was to determine whether initial aerobic power would have an

influence on adaptation from a relatively brief, high-intensity training program.

Methods

Twenty-three moderately to well-trained college-age males signed informed consent and were assigned to a high-velocity resistance training group (TG) or to a control group (CG). The groups were equated on initial absolute $\dot{V}O_2$ max, however in order to investigate the effects of initial fitness, subjects were selected for the TG to provide two sub-groups of significantly different $\dot{V}O_2$ max relative to body weight.

Maximal oxygen consumption ($\dot{V}O_2$ max) was assessed on a motor-driven treadmill (Quinton 24-72). Subjects ran for one minute on a level treadmill at each of 3.1, 3.6, 4, and 4.4 m s^{-1} and then the grade was increased by 2% each minute until $\dot{V}O_2$ max was observed, or until volitional exhaustion. Expired gases were collected and analyzed each 30s for volume, and O_2 and CO_2 concentration with a Beckman Metabolic Measurement Cart (Wilmore et al. 1976), calibrated before and after each test. The $\dot{V}O_2$ max was confirmed by a levelling or decrease in $\dot{V}O_2$ with increasing exercise loads and/or a respiratory exchange ratio in excess of 1.15. The peak $\dot{V}O_2$ value obtained during the exercise was recorded as $\dot{V}O_2$ max. During the exercise test, heart rates were recorded each minute from an electrocardiograph.

The high-velocity resistance (HVR) training program consisted of four sessions weekly over a five week period. The first 15 sessions consisted of two circuits and the last six sessions of three circuits on variable resistance equipment (Hydra-Fitness Industries Ltd.). The program was designed to achieve 1:2 work-relief ratio during each circuit. Each circuit consisted of two 20 second sets of maximal exercise at six stations which emphasized the following movement patterns: knee flexion and extension; hip flexion and extension; elbow flexion and extension; shoulder flexion and extension; and ankle plantar flexion.

Circuits were separated by four minutes of active (walking) recovery. Subjects worked in pairs and verbal encouragement was provided to ensure maximal effort and high quality contractions at all times. Subjects were required to complete at least 20 repetitions each 20 second exercise period. This approximated angular limb velocities of between 3.0 and 3.5 $rad \cdot s^{-1}$. When subjects exceeded 24 repetitions in 20 seconds, the resistance setting was increased. Each training session was monitored by a supervisor who adjusted and recorded resistance settings, numbers of contractions, and precisely timed the exercise and rest intervals.

Heart rates were monitored by cardio-tachometers (Exersentry) during the last training session of each week and were recorded at the end of each interval (24 times)

during each circuit to reflect exercise intensity and recovery levels.

Venous blood samples were drawn from the forearm prior to beginning exercise on the last day of training. Exactly five minutes following the third circuit, a second sample was drawn from the opposite arm. These were analyzed for lactate concentration (Sigma Chemical Company 1981).

Control group subjects were permitted up to three sessions weekly of low-intensity aerobic exercise to facilitate maintenance of initial VO_2 max. Some CG subjects engaged in heavy resistance exercise up to three times per week, but were restricted from any resistance exercise involving either high-velocity contractions or high numbers of repetitions.

Data were analyzed with a two-way repeated measures analysis of variance (Winer, 1972). As warranted, means comparisons were performed with two-tailed independent or paired t-tests.

Results

No significant differences were evident between TG and CG subjects prior to the training program in age (TG mean +/- SE = 19.4 +/- 2.5, CG mean +/- SE = 21.6 +/- 1.2), height (TG mean +/- SE = 179.3 +/- 1.9, CG mean +/- SE = 179.4 +/- 2.1), weight, or VO_2 max (Table IV-1).

The $\dot{V}O_2$ max for TG subjects was increased ($p < 0.001$) by 9% after the training program, while no change was observed for the controls. No change in body weight or maximum heart rate was observed for either group over the five weeks (Table IV-1).

When TG subjects were blocked on initial relative $\dot{V}O_2$ max into High Aerobic Power (HAP) and Low Aerobic Power (LAP) groups, no differences in the magnitude of training effects were observed. The $\dot{V}O_2$ max for the HAP group was significantly greater ($p < 0.001$) than for the LAP group when expressed either in absolute or relative fashion. The 2-way repeated measures ANOVA indicated no significant interaction term between groups. Both groups increased the $\dot{V}O_2$ max significantly ($p < 0.01$) over training (Table IV-2).

Mean (+/- SE) venous lactate concentrations from samples drawn prior to and following an exercise session were 1.2 (+/- 0.1) $mmol \cdot l^{-1}$. and 10.2 (+/- 0.1) $mmol \cdot l^{-1}$ respectively.

Heart rate responses recorded at the end of each of the 12 exercise and 12 relief intervals of each circuit are displayed in Figure 1 to indicate average exercise intensity and the degree of recovery within each circuit. To reflect exercise intensity during the entire exercise session, all heart rates were pooled and this value is also displayed in Figure 1 as "average" heart rate.

Table IV-1

Characteristics of Training (TG) and Control (CG) Subjects Before and After the Training Program

Variable	TG (n=12) Pre	TG (n=12) Post	CG (n=11) Pre	CG (n=11) Post
Weight (kg)	72.0 (2.3)	72.0 (2.3)	76.8 (2.0)	76.3 (1.9)
$\dot{V}O_2$ max ($l \cdot min^{-1}$)	4.06[a] (0.11)	4.43[a] (0.12)	3.96 (0.11)	4.00 (0.10)
$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	56.5[b] (1.3)	61.5[b] (1.3)	51.7 (0.9)	52.4 (0.8)
HR max (beats $\cdot min^{-1}$)	197 (2)	197 (2)	197 (2)	198 (1)

Values are Mean +/- (S.E.)

Paired letters indicate significant ($p < 0.001$) differences after training.

Table IV-2

Characteristics of the High Aerobic Power (HAP) and Low Aerobic Power (LAP) Groups Before and After the Training Program

Variable	HAP (n=6) Pre	HAP (n=6) Post	LAP (n=6) Pre	LAP (n=6) Post
Weight (kg)	71.6 (1.8)	71.9 (1.8)	72.3 (2.3)	72.0 (2.1)
$\dot{V}O_2$ max ($l \cdot min^{-1}$)	4.50^{ac} (0.09)	4.85^{c} (0.13)	3.72^{ae} (0.08)	4.13^{e} (0.10)
$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	60.6^{bd} (0.9)	65.2^{d} (1.1)	52.4^{bf} (0.4)	57.8^{f} (0.5)
HR max (beats$\cdot min^{-1}$)	194 (3)	196 (3)	201 (3)	198 (3)

Values are Mean +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences in group means between groups before training and within groups after training.

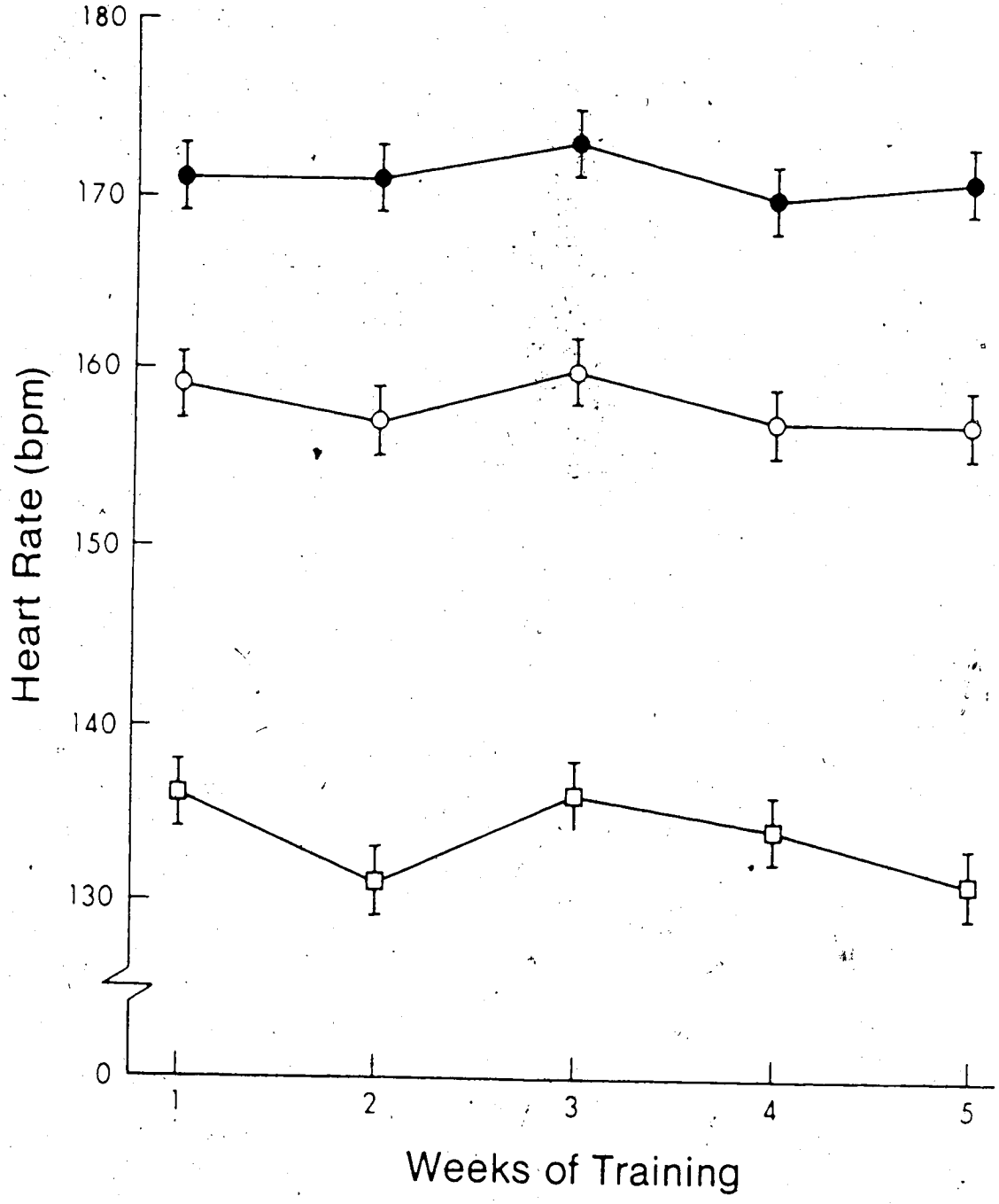

Fig. IV-1. Exercise (●), relief (□), and average (○) heart rates (Mean ± S.E.) during exercise sessions.

Discussion

The $\dot{V}O_2$ max increased ($p < 0.001$) for training subjects from 4.12 to 4.51 $l \cdot min^{-1}$, or by 9% over the five week program of HVR exercise. Aerobic power did not change significantly for control subjects. The small (1%) increase reported in Table IV-1 is well within the range of biological variability for $\dot{V}O_2$ max (Katch et al. [illegible]). Gettman et al. (1982) reported a 12% increase in $\dot{V}O_2$ [illegible]llowing 12 weeks of circuit weight training with [illegible]y untrained subjects (initial $\dot{V}O_2$ max = 2.22 $l \cdot min^{-1}$). Previous investigations (Gettman et al. 1978, 1979, 1980; Wilmore et al. 1978) have reported an average increase in $\dot{V}O_2$ max of about 5% following circuit weight training programs of up to 20 weeks duration. These studies represent the bulk of previous work in this area, and while direct comparisons are not possible, they are mentioned to indicate the significance of the results of the present study using young, previously well-trained subjects over a comparatively brief training period.

No changes in weight were noted over the five week training period. This result is not surprising as the subject pool was comprised of young, varsity athletes who were at or near optimal body weights at the commencement of the study.

Maximum heart rate values obtained during the aerobic power assessments did not change for either group (Table

IV-1). This result is reported to confirm the integrity of the initial $\dot{V}O_2$ max results, and that the reported changes were not due to test familiarization.

Average heart rates (Karvonen et al. 1957) recorded during the 20 second exercise intervals represented 83% of the maximum heart rate observed during the treadmill tests. At some exercise stations, heart rates often approached 100% of the maximum values. Mean relief interval heart rates were calculated to be 55% of maximum. Exercise and relief interval heart rates were pooled to indicate mean exercise intensity over the entire training session and represented 72% of maximum heart rate sustained over approximately 40 minutes.

The venous lactate concentrations after three circuits of HVR exercise were between the values suggested by Astrand and Rodahl (1977) and Brooks and Fahey (1984) as being indicative of exercise at $\dot{V}O_2$ max. In conjunction with the heart rate data, the lactate values suggest an exercise load approaching $\dot{V}O_2$ max.

A subsequent project designed to investigate acute metabolic and heart rate responses to the same exercise circuit used in the present study replicated the heart rate responses described above, and oxygen consumptions of up to 83% of $\dot{V}O_2$ max were observed at some exercise stations (Petersen et al. 1984). Katch et al. (1985) reported similar heart rate responses, but lower $\dot{V}O_2$ values during

exercise on some of the same apparatus using different exercise and relief intervals. It is likely that circulatory occlusion during the resistance exercises would contribute to the elevation of heart rate (MacDougall et al. 1985). The metabolic intensity is probably dependent upon the interaction between the duration of exercise and relief intervals as well as the motivation of the subjects to provide an "all-out" effort at each station.

The effect of initial aerobic fitness on training the $\dot{V}O_2$ max has been recently reviewed (Wenger and Bell 1986). Generally, it may be expected that individuals with high initial aerobic power have less potential for adaptation than those with lower initial fitness, provided other factors such as mode, intensity, frequency and duration of exercise are equated. In the present study, this principle was not observed as the High and Low Aerobic Power groups demonstrated equal change in $\dot{V}O_2$ max over the training program.

In summary, the 5-week program of high-velocity resistance training enhanced the $\dot{V}O_2$ max in a group of previously well-trained subjects, and in this study, initial aerobic fitness did not appear to influence adaptation from training.

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CHAPTER V

The Effects of Velocity-Specific Circuit Resistance Training on Maximal Aerobic Power, Maximal Cardiac Output, and Recovery[1]

Introduction

The effects of circuit resistance training on muscular strength, power, and endurance, as well as aerobic power have been relatively well documented (Gettman et al. 1979, 1980, 1982; Wilmore et al. 1978; Petersen et al. 1983, 1984a; Haennel et al. 1985). Therefore, in consideration of the potential for simultaneous enhancement of several fitness components, programs of circuit resistance training may be appropriate for certain athletes. However, the majority of previous research has utilized pools of relatively untrained subjects and, thus, the potential for enhancement of aerobic fitness in well-trained subjects has not been well established.

Recent investigations of acute responses to hydraulic resistance exercise (Katch et al. 1985; Petersen et al. 1984b) have reported that a relatively high exercise intensity, as reflected by heart rate, oxygen consumption, and venous lactate concentration, may be achieved. There-

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fore, a hydraulic resistance exercise circuit with relatively brief rest periods may provide sufficient stimulus for changing aerobic power in an athletic subject pool.

Haennel et al. (1985) have reported increased maximal stroke volume in previously sedentary males after hydraulic circuit training. [illegible]man et al. (1982) have speculated that such adaptation may occur based on their observations of alterations in oxygen pulse after circuit training. Kanakis and Hickson (1980) have reported impr[illegible]ft ventricular performance after 10 weeks of heavy resistance training. Stone et al. (1983) have also suggested that positive cardiovascular system adaptation may occur with heavy resistance training. MacDougall and Sale (1981) have suggested that enhancement of the oxygen transport system is readily transferable from one form of physical activity to another. Therefore, an improvement in stroke volume and consequently maximal cardiac output obtained from resistance training may assist in performance of other sports activities.

The importance of a highly developed aerobic system in enhancing recovery from heavy exercise has been suggested (Brooks and Fahey 1984; Wenger 1981). Presumably, improved aerobic power and circulatory function would facilitate more rapid removal and utilization of lactate, thus speeding the recovery process. Roberts et al. (1982) have reported significant increases in key glycolytic pathway enzymes

following high-intensity interval training. Sharp et al. (1986) have reported increased skeletal muscle buffer capacity with sprint training. These findings are supported by the comparison of buffer capacity between trained and untrained subjects reported by Sahlin and Henriksson (1984). Thus, high-intensity power training may affect anaerobic power output and recovery from resultant high lactate concentrations in several ways.

The purpose of the present study was to investigate the effects of six weeks of velocity-specific (either high or low) circuit resistance training on the maximal aerobic power, maximal stroke volume and cardiac output, and recovery from heavy exercise in a pool of previously well-trained individuals.

Methods

Twenty-four apparently healthy and habitually active males provided informed consent to participate in the study. All subjects were affiliated with either University football or rowing programs although the study coincided with the off-season for both sports. All subjects had previous experience with resistance training.

Maximal aerobic power was assessed during a progressive, continuous exercise test on a modified Monark cycle ergometer. Pedalling frequency was maintained at approximately 60 rpm with a metronome, and actual frequency was

counted electronically. The flywheel resistance was increased each minute to increase power output by either 60 watts (W) early in the test, or 30 W during the later stages. Expired gases were collected and analyzed for volume, and O_2 and CO_2 concentration each 30 seconds with a Beckman Metabolic Measurement Cart (MMC). The MMC was thoroughly calibrated according to the manufacturer's recommendations prior to each testing session, and calibration of the gas analyzers was performed before and after each test with gases of known concentration. The $\dot{V}O_2$ max was confirmed by a levelling or decrease in $\dot{V}O_2$ with increasing exercise loads and/or a respiratory exchange ratio in excess of 1.15. The peak $\dot{V}O_2$ value observed during the exercise was recorded as $\dot{V}O_2$ max. Heart rate was monitored continuously with a calibrated cardiotachometer and was recorded each minute.

Immediately following the exercise test, subjects were assisted to an adjacent chair where they sat quietly for 15 minutes. At exactly five minutes and 15 minutes post-exercise, a small sample of venous blood was drawn from the forearm. One-half ml of fresh blood was immediately added to 2 ml chilled perchloric acid. Following centrifugation the clear supernatant was frozen for subsequent analysis of lactic acid concentration (Sigma Chemical Company, 1981). All lactate assays were completed in triplicate.

On a separate day, maximal stroke volume was assessed using impedance cardiography (Surcom Inc. Model 304). Subjects exercised on an electrically-braked cycle ergometer at an initial power output of 30 W. At three minute intervals the load was adjusted in 20 W increments up to a terminal load of 190 W. This represented 60% of the average load which elicited VO_2 max for all three groups. Recordings of heart sounds, heart rate, and impedance waveforms were obtained immediately following each exercise load. These data were collected during breath-holding in order to minimize waveform distortion. Mean values obtained from five cardiac cycles were used to calculate stroke volume according to the method described by Teo et al. (1985). Maximal cardiac output was calculated as the product of maximal stroke volume and the maximal heart rate observed during the VO_2 max test.

The resistance training program consisted of 21 exercise sessions over 42 days. Subjects completed two circuits of 10 variable resistance exercise stations (Hydra-Fitness Industries Ltd.) for the first two weeks, and thereafter completed three circuits each exercise session. At each station, subjects completed two 20 second sets of maximal effort separated by 20 seconds of relief. A minute of rest was permitted between stations and four minutes was allowed between circuits. Within each circuit the exercise: relief ratio was 1:2. The order of exercise stations was

arranged to exercise upper and lower body musculature alternately wherever possible. The exercises included the following movements: unilateral (seated) elbow extension and flexion; bilateral (seated) shoulder horizontal abduction and adduction; unilateral (supine) shoulder horizontal abduction and adduction; bilateral (supine) shoulder extension and flexion; unilateral (seated) knee extension and flexion; bilateral (seated) hip and knee extension; bilateral (inclined) hip and knee extension; unilateral (reclined) hip abduction and adduction; and unilateral (supine) hip extension and flexion. Angular velocities of movement were maintained at approximately 1.1 (LVR) or 3.1 (HVR) rad $\cdot s^{-1}$. The hydraulic cylinders provided accommodating resistance but not in a true isokinetic fashion. However, knowledge of range of motion at each station permits computation of the average angular velocity based on the number of repetitions performed in a fixed time period. Subjects must be consistently motivated to work at maximal intensity through the full range of motion. The adjustments on the hydraulic cylinders permit quite accurate control of velocity as subjects' functional capacity increases with training. Each exercise session was monitored by a supervisor who adjusted resistance settings as necessary, and precisely timed the exercise and relief intervals.

Heart rates were monitored during an exercise session by cardio-tachometers (Exersentry). Heart rate responses to

each exercise and relief interval each circuit were recorded (120 times) to reflect exercise and recovery levels. On the same day venous blood samples were drawn from the forearm before exercise and at exactly five minutes following the third circuit. These were analyzed for lactate concentration according to the procedure described earlier.

Control subjects were permitted up to three low-intensity aerobic exercise sessions per week and one heavy resistance session per week in order to maintain initial fitness levels. They were restricted from any systematic application of high-intensity exercise stimulus over the five week period.

Data were analyzed with 2-way analysis of variance for repeated measures (Winer 1972). As warranted, post-hoc means comparisons within groups were performed using two-tailed paired t-tests. In each case the significance level was set at $p < 0.05$.

Results

Characteristics of the three study groups prior to either training or control periods are displayed in Table V-1. No significant differences were observed between group means on any variable. Body weight did not change significantly for any group over the study period. This was not surprising as all subjects were habitually active prior to the study and were probably close to an optimal body weight.

Aerobic power characteristics of the three groups before and after the study are displayed in Table V-2. Maximal aerobic power ($\dot{V}O_2$ max) was significantly ($p < 0.01$) increased for the HVR group when expressed in either absolute or relative fashion. No changes in either absolute or relative $\dot{V}O_2$ max were evident for either of the other groups. Maximal heart rate did not change for any group over the study.

Changes in venous lactate concentration during recovery from the aerobic power assessments are reported in Table V-3. Lactate removal from the blood, as indicated by the numerical difference between concentrations at five and 15 minutes post-exercise was increased ($p < 0.005$) for both training groups but not for the control group.

Maximal stroke volume as measured by impedance cardiography increased ($p < 0.05$) for both training groups, but not for the control group. Maximal cardiac output was predicted by multiplication of the maximal stroke volume by the maximal heart rate which was observed during the aerobic power test. Since maximal heart rate did not change over the study (Table V-2), the changes in maximal cardiac output bore the same significance as the alteration in maximal stroke volume. The increased stroke volumes of the training groups were complemented by a reduction ($p < 0.05$) in heart rate response to a fixed sub-maximal exercise load of 190 W

on the cycle ergometer. Again, no changes were observed for control subjects.

Average exercise and relief heart rate responses to the HVR and LVR exercise circuits were calculated by pooling all the exercise and relief heart rates recorded during three circuits. These means are reported for both groups in Table V-5.

Lactate concentrations from venous blood drawn before and at five minutes following three circuits of either HVR or LVR exercise are reported in Table V-5.

Table V-1

Characteristics of the High (HVR) and Low (LVR) Velocity Training Groups and the Control (CG) Group Prior to Training

Variable	HVR (n=8)	LVR (n=8)	CG (n=8)
Age (years)	21.6 (0.7)	23.3 (0.6)	21.1 (0.6)
Hei	184.0 (2.3)	177.9 (2.4)	182.9 (1.9)
	82.3 (3.5)	79.6 (3.1)	82.5 (2.8)
	4.32 (0.18)	4.25 (0.13)	4.32 (0.20)

Va[illegible]re Means +/- (S.E.)

Table V-2

Aerobic Power Characteristics of the High (HVR) and Low (LVR) Training and Control Groups (CG)

Variable	HVR (n=8) Pre	HVR (n=8) Post	LVR (n=8) Pre	LVR (n=8) Post	CG (n=8) Pre	CG (n=8) Post
$\dot{V}O_2$ max ($l \cdot min^{-1}$)	4.32[a] (0.18)	4.68[a] (0.24)	4.26 (0.13)	4.36 (0.12)	4.32 (0.20)	4.27 (0.19)
$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	52.6[b] (1.0)	56.7[b] (1.4)	53.7 (1.5)	54.6 (1.8)	52.4 (1.9)	51.4 (1.7)
Heart rate max (bpm)	193 (1)	192 (1)	194 (2)	193 (1)	194 (3)	193 (3)

Values are Means +/- (S.E.).

Paired letters indicate significant ($p < 0.01$) differences within groups after training

Table V-3

Changes in Venous Lactate Concentration During Recovery from Exhausting Cycle Ergometer Exercise for High (HVR) and Low (LVR) Velocity Training and Control Groups (CG)

Variable	HVR (n=8) Pre	HVR (n=8) Post	LVR (n=8) Pre	LVR (n=8) Post	CG (n=8) Pre	CG (n=8) Post
5 min ($mmol \cdot l^{-1}$)	15.5 (1.1)	17.4 (1.2)	14.7 (0.8)	15.8 (0.9)	14.1 (0.9)	14.2 (0.6)
15 min ($mmol \cdot l^{-1}$)	12.8 (1.2)	12.7 (1.0)	13.1 (0.6)	11.7 (0.7)	11.8 (0.8)	11.8 (0.7)
Change ($mmol \cdot l^{-1}$)	2.7[a] (0.9)	4.7[a] (0.7)	1.6[b] (0.5)	4.1[b] (0.8)	2.3 (0.7)	2.4 (0.9)

Values are Means +/- (S.E.)

Paired letters indicate significant ($p < 0.005$) differences within groups after training.

Table V-4

Maximal Stroke Volume, Maximal Cardiac Output, and Submaximal Heart Rate Responses of High (HVR) and Low (LVR) Velocity Training and Control (CG) Groups

Variable	HVR (n=8) Pre	HVR (n=8) Post	LVR (n=8) Pre	LVR (n=8) Post	CG (n=8) Pre	CG (n=8) Post
Stroke Volume (ml)	120[a] (6)	129[a] (5)	115[d] (6)	126[d] (6)	121 (5)	118 (6)
Cardiac Output ($l \cdot min^{-1}$)	23.11[b] (1.03)	24.65[b] (0.89)	22.27[e] (1.11)	24.32[e] (1.20)	23.41 (0.95)	22.61 (1.03)
Heart Rate at 190 Watts ($beats \cdot min^{-1}$)	153[c] (6)	146[c] (7)	159[f] (4)	148[f] (6)	155 (7)	158 (6)

Values are Means +/- (S.E.)

Paired letters indicate significant ($p < 0.05$) differences within groups after training.

Table V-5

Acute Responses to High (HVR) and Low (LVR) Velocity Circuit Resistance Exercise

Variable	HVR (n=8)	LVR (n=8)
Heart Rate		
Exercise ($beats \cdot min^{-1}$)	166 (2)	145 (2)
Relief ($beats \cdot min^{-1}$)	142 (4)	118 (4)
Venous Lactate		
Pre-exercise ($mmol \cdot l^{-1}$)	1.3 (0.1)	1.4 (0.2)
Post-exercise ($mmol \cdot l^{-1}$)	18.3 (1.2)	10.6 (1.2)

Values are Mean +/- (S.E.)

Discussion

The increased $\dot{V}O_2$ max for the HVR group is consistent with our previous findings (Chapter IV) regarding change in aerobic power following circuit resistance training. Since body weight did not change over the training period, the improvement represents an 8% increase when expressed in either absolute or relative terms. Maximal heart rate did not change for any group over the study period. This result indicates that the increased aerobic power for the HVR group was not due to test familiarization.

The maximal aerobic power did not change for either the LVR group or the controls. These test results are well within the reported range for biological variability in maximal aerobic power (Katch et al. 1982). The stable $\dot{V}O_2$ max for the LVR may be interpreted as a positive result since that program was essentially high-resistance training with no supplementary aerobic exercise permitted. It appears that performing heavy resistance exercise in circuit fashion with limited rest periods facilitates maintenance of initial aerobic power.

Blood lactate concentration during and following exercise is often used to indirectly indicate lactate concentration in the active muscle (Gollnick et al. 1986). At best this is a rough approximation of the effect that a particular exercise bout may have on muscle. The rate of lactate removal from the blood may be used as an indicator

of recovery from exercise. In heavy exercise, the turnover rate of lactate is decreased. That is, the rate of entry into the blood exceeds the rate of removal. During recovery, this relationship is reversed and may be influenced by the type of activity performed during recovery (Hermansen and Stensvold 1972; Belcastro and Bonen 1975). Venous lactate concentration is reported to peak at approximately five minutes following exhausting exercise (Gollnick et al. 1986) and the rate of removal remains relatively constant over about the next 20 minutes of recovery (Karlsson and Saltin 1971; Fox and Mathews 1981). In the present study, the difference in blood lactate concentration at five and 15 minutes into recovery following exhausting exercise was used to indicate the rate of lactate removal. These data are reported in Table V-3. The change in lactate concentration in venous blood was increased ($p < 0.005$) following training for both HVR and LVR groups. The concentration at five minutes was elevated (though not significantly) for both groups following training. However, this should not encourage speculation on peak venous lactate changes with training, since the single sample method employed here does not ensure a "peak" value.

Post exercise venous lactate concentration and removal rate are influenced by a number of factors including: a) oxidation within the muscle where the lactate was formed; b)

of lactate, by other tissues for oxidation or resynthesis to glucose; or c) some combination of the above (Gollnick et al. 1986). Presumably, all of these factors could be influenced by chronic high production of lactate which would normally be associated with sprint training (Roberts et al. 1982), and which has been observed in this study (Table V-5), and in previous investigation of HVR exercise (Chapter IV; Petersen et al. 1984b). More effective efflux of lactate from muscle could be associated with increased capillarity or decreased diffusion distances between fiber and capillary membranes (Saltin and Rowell 1980). Alternately, these data may reflect a trend towards increased lactate production. Roberts et al. (1982) have reported increased activities of key glycolytic enzymes following sprint training. Present results do not permit conclusions to be drawn regarding the nature of lactate production and/or turnover responses, however it seems clear that removal rate has been positively influenced by both training regimes.

Table V-4 summarizes the effects of training on the central circulation. Both HVR and LVR groups increased ($p < 0.05$) maximal stroke volume with training. The prediction of maximal cardiac output was increased by the same proportion since maximal heart rate did not change. The increases in stroke volume were complemented by a decreased ($p < 0.05$)

exercise load of 190 W. Calculation of cardiac output for this exercise load based on heart rate and maximal stroke volume indicated that no change occurred, and this would tend to substantiate the integrity of the reported alterations in stroke volume. These changes may be due to a variety of factors. Cross-sectional studies have reported different left-ventricle characteristics between strength trained and endurance trained athletes (Morganroth et al. 1975; Roeske et al. 1976). In brief, endurance training appears to increase ventricular volume while strength training appears to increase ventricular wall thickness. Several studies have reported enhanced left ventricular function following short-term endurance training (DeMaria et al. 1978; Ehansi et al. 1978; Perrault et al. 1982), while Kanakis and Hickson (1980) have reported increased left ventricular wall thickness with short-term strength training. These authors noted that left ventricular performance may be altered without change in left ventricular volume. As well, enhanced contractility following swim training of the rat has been associated with biochemical adaptation of the myocardium (Scheuer et al. 1974; Bahn and Scheuer 1975). Therefore, contractility may be altered by both qualitative and quantitative characteristics of the myocardium.

The reduced heart rate response to submaximal exercise

Alternately, the improved stroke volume may be the result of the decreased heart rate which permits greater diastolic filling and activation of the Frank-Starling mechanism. The role of skeletal muscle and cardiopulmonary afferents in regulation of the cardiovascular response to exercise has recently been reviewed (Stone et al. 1985). These receptors may be sensitive to muscle stretch, contraction, or biochemical disruption which occurs during exercise. Chronic stimulation of these receptors over training may influence their modulation role in the cardiovascular response to exercise. Such an adaptation could be manifest in either increased vagal or decreased sympathetic tone which would tend to reduce the heart rate. Since stroke volume and heart rate were assessed in a mode not specific to the training stimulus, it is not possible to speculate on the cardiovascular adaptations which may have been evident in that mode. However, the observation of enhanced function in a non-specific exercise mode indicates that the training effect in the oxygen transport system is at least partially transferable from one mode of training to another mode of performance.

MacDougall et al. (1985) and Lewis et al. (1985) have reported significant increases in systemic blood pressure with both heavy resistance and lighter resistance exercise to fatigue. Haennel et al. (personal communication) have

isometric exercise which were related to the active muscle mass but appeared to be independent of exercise velocity. The rapid increase in afterload which would be expected with resistance exercise and the reflex increase in heart rate necessary to maintain cardiac output would presumably place a relatively high load on the heart muscle. At the same time, sustained dynamic resistance exercise over an hour with restricted rest will elevate oxygen consumption and heart rate as well (Petersen et al. 1984b). The nature of the repetitive resistance exercise should also provide a powerful muscle pump to enhance venous return and activate the Frank-Starling mechanism. In light of these possibilities, the observed changes within the central circulation seem quite reasonable. As well, the observed transfer of enhanced function from circuit resistance training to cycle ergometer exercise has some very positive practical implications for athletes.

The acute heart rate responses to both exercise programs are summarized in Table V-5. Heart rate responses averaged over all exercise and relief intervals are clearly higher for the HVR exercise protocol. These results are in agreement with other observations of heart rate response to the HVR circuit (Chapter IV; Petersen et al. 1984b). The slightly lower exercise and slightly higher relief heart rates observed for the present HVR circuit may be due to

that the present circuit consisted of 10 stations compared to the previous six stations. The increased duration of exercise may have altered heart rate due to fatigue or subjects' "pacing" themselves.

Although exercise and relief heart rates for LVR exercise were considerably lower, they still represent a reasonable elevation over normal resting values and this may have contributed to the observed maintenance of aerobic power and to the enhanced recovery and circulatory responses. Again, the duration of the exercise sessions is probably a factor. External work performed in resistance exercise may be equated independent of velocity (Haennel 1987), however, since the heart rate and post-exercise lactate responses are quite different, it is suggested that metabolic intensity in resistance exercise is velocity-dependent. This concept is supported by the improved VO_2 max in the HVR group, while no change was observed in the LVR group.

In summary, maximal stroke volume and the rate of lactate removal from the blood during recovery were enhanced by the HVR and the LVR exercise programs. Maximal aerobic power was increased in the HVR group, but not the LVR group. It is therefore suggested that circulatory adaptations, and metabolic adaptations associated with recovery may be achieved with circuit resistance training,

appears that metabolic intensity and the consequent effects on the VO_2 max are velocity dependent. These implications should be considered when designing individual circuit training programs for athletes.

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CHAPTER VI

General Discussion

In Chapter I a number of concerns were expressed regarding the extent of current understanding of specificity of velocity in strength training and the potential for enhancement of aerobic fitness through velocity-specific circuit resistance training. Based on these concerns, four research projects were designed and the results have been summarized in the preceding chapters.

The results of the first project supported the general concept of velocity-specific training effects when the training group was considered as a whole. That is, peak torque expression was increased when measured at an angular velocity similar to the training velocity (3.14 $rad \cdot s^{-1}$). When the results were considered with respect to initial strength levels, a different pattern was evident. In brief, subjects with low initial strength improved far more than subjects with high initial strength. Thus it appears quite clearly that initial levels have considerable effect on strength changes consequent to training. This principle has been well documented with respect to aerobic training (Wenger and Bell 1986). The prediction that high initial aerobic power might facilitate the acquisition of strength was not supported by the findings of this study.

The influence of initial strength levels on the outcomes of the training program may provide some insight into the somewhat confusing and often conflicting results of investigations of velocity specificity. Some have suggested (Moffroid and Whipple 1970; Lesmes et al. 1978) that there is greater transfer of strength from high-speed training to low-speed performance. On the other hand, the findings of others (Caiozzo et al. 1981; Kanehisa and Miyashita 1983) would indicate the opposite. Apart from some obvious differences in the nature of the training programs, and the classification of either "fast" or "slow" velocities, it seems quite likely that initial strength levels of the subject pools may be an important factor in identifying this question. That is, subjects with low initial strength may adapt to the training stimulus far more than high-strength subjects. Similarly, any pattern of transfer may be as much a reflection of initial subject characteristics than a universal trend of specificity. In each case, the investigations cited have either utilized pools of untrained subjects or have not reported any information regarding previous training state. Therefore, it is suggested that study design and interpretation of results in strength training studies give due consideration to the potential influence of initial fitness levels. This is especially applicable in situations where investigators are tempted to

speculate on the transfer of enhanced function to other movement velocities.

Chapter III reported the study designed to further investigate the nature of strength training effects which might be specific to the movement velocity at which the training was performed. Of particular interest were alterations of the in vivo torque-velocity relationship and the cross-sectional area of the knee extensors which might be related to training at either slow (LVR, 1.05 $rad \cdot s^{-1}$) or fast (HVR, 3.14 $rad \cdot s^{-1}$) movement velocities. Subjects were assigned to either training or control groups in consideration of initial peak torque outputs relative to body weight at both training velocities. The improvement of the HVR group at 0.52 $rad \cdot s^{-1}$ was attributed in part to a reduction of the neural tension-limiting mechanism described by Perrine and Edgerton (1978), Caiozzo et al. (1981), and Perrine (1986). Improvement in performance at higher movement speeds at or near the training velocity indicate a velocity-specific adaptation, however, the nature of the adaptation or adaptations involved may not be resolved by the findings of the present work. Presumably there are neural and biochemical mechanisms which may be affected by movement pattern and speed of movement, and since the cross-sectional area of the anterior thigh compartment was significantly increased, the increased performance is very likely mediated by some combination of morphological,

biochemical, and neural factors. Further work is necessary to resolve these questions. To fully investigate the nature of specificity of velocity in strength training, several factors need to be evaluated simultaneously. Increased force expression is closely related to muscle cross-sectional area, however, the time course for strength improvement and hypertrophy are apparently not synchronized during the early stages of training (Luthi et al. 1986; Komi 1986). Changes in EMG activity and the ratio between force output and cross-sectional area might indicate the extent of neural adaptation. It might be reasonable to expect velocity-specific adaptation within specific muscle fiber populations as reported by Costill et al. (1979) and Caiozzo et al. (1981). Therefore, fibre area changes with respect to fast or slow classifications should be monitored in addition to whole muscle area. As well, the activity of myofibrillar ATPase could be used as a marker of adaptation within the contractile mechanism. Finally, in order to address the velocity-specificity issue, use of an isokinetic loading system (eg. Cybex II) would be suggested to provide accurate control of velocity.

The alterations observed for the LVR group would be relatively easily explained in the absence of the HVR results just discussed. It would seem reasonable that enhanced motoneuron activity or, alternately, reduced neural inhibition, in concert with the increased muscle mass would

explain the enhanced performance across the range of test velocities. The interaction of the neurogenic and myogenic components of performance is underscored by the lack of change or the reduction in peak torque expression which was observed for the controls with no concomitant change in the muscle cross-sectional area. The mechanisms responsible for reduced strength with inactivity or detraining are again intriguing questions for further study. It is possible that the tension-limiting inhibitory phenomenon described by Perrine (1986) is enhanced with detraining. This would be logical if in fact the effect is reduced with training. In a recent review, Komi (1986) has suggested that the enhanced maximal neural activation that apparently results from velocity-specific strength training must be maintained with regular exercise stimulus. Therefore, it would appear that the increased neural drive and the synchrony of motor unit recruitment which occurs with training may be reversed with detraining. In the present study, control subjects were encouraged to maintain initial strength levels, however, there was no structured training to facilitate this maintenance.

This paper has described some performance and gross morphological changes which seem to be related to the velocity of training, since as much as possible the subjects were grouped with respect to initial strength levels at both high and low speed, and the dimensions of both programs

(frequency, intensity, and duration) were equated as well. Exercise and relief intervals were identical, and all subjects were consistently motivated (externally) to provide a maximal effort. In that all subjects were athletes familiar with the concept of maximal effort, and volunteered to participate in the study, it was assumed that intrinsic motivation would not be a limiting factor. Clearly, the concept of specificity of velocity in strength and power training is potentially very important to athletes who perform at either high or low velocities (or both). However, it is also quite clearly a complex concept which will not be readily resolved.

The effect of high-velocity circuit resistance training on maximal aerobic power was investigated in Chapter III. The observed increase of 9% in an already well-trained group of subjects has indicated that this may be an effective means of aerobic training for athletes. In this investigation, initial aerobic power did not influence the magnitude of the $\dot{V}O_2$ max adaptation. This finding may be specific to the subject pool utilized rather than a challenge to the rather well established principle that trainability of the $\dot{V}O_2$ max is linked to initial levels. In a recent review, Wenger and Bell (1986) have indicated that over a wide variety of subjects and training programs, initial levels of between 50 and 60 $ml \cdot kg^{-1} \cdot min^{-1}$, show more potential for change than initial levels of greater than 60 $ml \cdot kg^{-1}$.

min^{-1}. The fact that the group with high initial aerobic power (> 60 $ml \cdot kg^{-1} \cdot min^{-1}$) in the present study showed equal improvement may be a reflection of an ultimately high potential for change.

Since aerobic power was assessed in an exercise mode which was not specific to the training mode (treadmill vs resistance training), and aerobic power was improved, it is tempting to speculate that the enhanced aerobic function was at least partially due to improved oxygen transport. MacDougall and Sale (1981) have suggested that alteration of the central component of aerobic power is more readily transferable to other activities than are peripheral muscle adaptations. Therefore, the effect of circuit resistance training on maximal stroke volume and cardiac output was identified as a question for further study. As well, the relatively high post-exercise blood lactate concentrations indicated a significant anaerobic contribution to total energy production. In light of the similarity of high-velocity resistance exercise with sprint exercise, it was reasoned that some of the aerobic benefits associated with interval training (Fox et al. 1977; Bell 1986) might be achieved if the exercise:relief ratio was fairly low. As well, the significant glycolytic contribution to total power output during sprint exercise creates high muscle and blood lactate levels (Roberts et al. 1982; Sharp et al. 1986). Chronic exposure to this condition might elicit adaptations

to facilitate recovery. The heart rate and oxygen consumption responses to cycling exercise have been reported to be a function of pedalling frequency when power outputs are equated (Lollgen et al. 1980). In isokinetic resistance exercise of the lower limb, external work appears to be relatively constant over a range of velocities (Haennel 1987). Therefore, it was hypothesized that high-velocity circuit training might be more effective at altering aerobic power than low velocity circuit training provided exercise time, total external work, and initial fitness of subjects were equated.

The design of the final study in this series considered the potential effects of exercise velocity on maximal aerobic power & maximal cardiac output predicted from maximal stroke volume, and the rate of removal of lactate from the blood during recovery from exhausting exercise. The exercise:relief ratio in the circuit was maintained at 1:2, however, the number of exercise stations was increased from six to 10. The work of Hickson et al. (1980) suggested that cycling may be a more appropriate mode of exercise for assessing aerobic power across resistance training programs in that leg strength is more limiting in cycling than in treadmill running. As well, the assessment of stroke volume with impedance cardiography can be performed more effectively during cycling exercise. For these reasons it was decided to use the cycling mode for the performance tests.

The aerobic power increase of eight percent with six weeks of HVR training was consistent with our previous findings. Considering the initial levels and previous training habits of the subjects, eight per cent represents a significant training response. Hickson et al. (1980) and Stone et al. (1983) have reported stable or slightly increased $\dot{V}O_2$ max over short-term heavy resistance training periods, and the results of the LVR program would support those findings. However, in those studies, subjects were previously untrained and initial $\dot{V}O_2$ max was considerably lower (3.14 - 3.40 $l \cdot min^{-1}$) than in the present subject pool (4.25 $l \cdot min^{-1}$). As well, the criteria for confirmation of $\dot{V}O_2$ max are somewhat sketchy and therefore it is tempting to speculate that the small improvements reported may have been due to some combination of the training programs, test familiarization, and the previously untrained state of the subjects. In the present study, it was evident that heavy resistance exercise performed in circuit fashion will effectively maintain aerobic power in previously trained subjects. However, the effectiveness of circuit resistance training with respect to improvements in $\dot{V}O_2$ max, appears to be a function of movement velocity.

Apparently independent of movement velocity, and probably more related to the exercise:relief ratio and the total work performed were improvements in maximal stroke volume and the rate of lactate removal during recovery.

Several suggestions have been offered as possible explanations for the observed changes, however, further investigation is necessary to resolve these questions. Use of echocardiography to assess ventricular wall thickness, and blood pressure and plasma catecholamine responses to fixed exercise loads may help to clarify the cardiovascular responses. Assessment of peripheral capillary density, muscle lactate concentration, and muscle buffering capacity might assist in understanding the relationship between lactate production and removal during exercise and recovery. As well, a better understanding of the acute cardiovascular and metabolic responses to velocity-specific resistance exercise would be most beneficial.

Although the results of this work do not appreciably clarify the underlying mechanisms of adaptation, the training programs described in each of the four studies have produced several interesting training effects and these have some practical applications. Hickson (1980b) has reported that simultaneous strength and endurance training compromises the strength training effect. While direct comparisons between the studies are not possible, the present results would indicate that strength and aerobic power may be simultaneously enhanced through high velocity circuit training. This may be particularly applicable in sports where a considerable portion of total training time must be devoted to the development of skills of tactics. Develop-

ment and/or maintenance of sport-specific fitness components could be accomplished simultaneously with circuit training by appropriate combinations of exercise stations, movement velocity, and frequency of exercise sessions. This, in theory, might reduce total training volume devoted to fitness development and permit more time allotment to skill or tactical practice. Alternately, the concept of performing heavy resistance training in circuit fashion without supplementary aerobic exercise to maintain VO_2 max seems warranted. Further study is necessary to determine if the strength training effect is compromised, however, the significantly improved peak torque outputs and the increased cross-sectional area of muscle are consistent with recent findings (Luthi et al. 1986) from more conventional heavy resistance programs. Based on that comparison, it seems likely that little strength training effect may be lost. In many sports, the enhanced cardiac output and recovery which were observed with the LVR program would be very important as well.

In ice hockey, for example, athletes must often develop maximal forces against high resistance and, correspondingly, relatively low forces, but at high movement velocities. The influence of resistance training on the in vivo force-velocity relationship is of great importance. In the same sport, brief high intensity bouts of exercise are interspersed with periods of lighter work or rest where recovery

may occur. Clearly, the ability to enhance recovery would be a desirable training adaptation. One of the limitations of the present work is the restriction of assessment of muscular performance to peak isokinetic torque. While it may be reasonable to expect a strong relationship between peak torque and ultra-short term power output (Smith 1987), the effect of velocity-specific resistance training on the latter may be ultimately more important.

In summary, responses of previously well-trained young males to programs of velocity-specific circuit resistance training have been documented and discussed. Circuit resistance training appears to have positive effects on muscle morphology and force-expression, as well as on parameters associated with aerobic fitness. With due consideration of initial fitness levels, it is suggested that programs of either high or low velocity circuit resistance training may be time efficient and effective modes by which athletes might enhance functional capacity.

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APPENDIX A

Methods

The intent of this section is to provide additional information on some of the data collection techniques used in the studies reported in the main text. This information is organized under separate headings as follows: Peak torque assessment; Computed tomography (CT) scanning; Impedance cardiography; Plasma lactate concentration; Hydraulic resistance training; and Data analysis.

Peak Torque Assessment

Peak torques in knee extension and flexion movements at selected angular velocities were measured using a Cybex isokinetic dynamometer. Until recently, this was the only commercially available system and has been widely used to assess isokinetic strength in athletes. Reliability coefficients of up to 0.99 have been reported (Clarkson et al. 1982) for repeated peak torque measures in young, athletic subjects. In pilot work, we have observed no significant variation across four repeated measures of knee extension or flexion torque over a range of angular velocities (0.52 - 4.19 $rad \cdot s^{-1}$). Subjects were male and female physical education students and all measures were taken on alternate days over a one week period. Reliability coefficients ranged from 0.92 to 0.94.

Two problems have been identified with regard to the validity of peak torque measures in isokinetic dynamometry. The first results from absorption of the momentum of the accelerating limb by the servomotor of the dynamometer. In effect, the dynamometer must apply a decelerating force to the limb to control angular velocity and this "extra" force may be recorded as a torque artifact. The implications of the "impact artifact" have been discussed in detail by Winter et al. (1981) and Sapega et al. (1982). Care must be taken to either consistently include or ignore the artifact depending upon whether the phenomemon is of interest to the investigator. In the present studies, a system of "curve-smoothing" was used to eliminate the obvious torque artifacts. All measures of peak torque were assessed by the same investigator using a consistent interpretation of the concept of curve-smoothing. Therefore, changes in performance reported in this work do not result from inconsistent measurement of torque artifacts.

The second problem results from performing the tests in the sagittal plane where knee extension occurs against gravity and knee flexion is accomplished with the assistance of gravity. This concern has again been discussed by Winter et al. (1981) who have suggested methods of correcting for gravitational influences. These problems would be of major importance if true measures of muscle force output were desired. However, in most training studies, change is the

most important consideration and, therefore, if the potential errors of torque artifacts and the constant force of gravity are dealt with consistently, a change in performance may be interpreted as a change in the functional capacity of the muscle.

The Cybex dynamometer may be calibrated for torque and velocity. According to the manufacturer's suggestions, torque calibration was performed by placing known weights at the end of a lever arm of known length and permitting the arm to rotate about the input axis at a velocity of 0.52 $rad \cdot s^{-1}$. Calibration of the speed control system was accomplished by determining the number of revolutions of the input shaft at velocities of 0.52 and 3.14 $rad \cdot s^{-1}$ over a period of 30 seconds. Calibration of the system was checked regularly, but no adjustments were made over the study period.

Positioning and stabilization of subjects relative to the input axis of the dynamometer was standardized and performed consistently over each test. The rotational axis of the knee joint was visually aligned with the input shaft of the dynamometer which in turn was maintained in a constant position relative to the testing chair and the floor. The lever arm was attached firmly to the leg just proximal to the malleoli. The length of the lever arm was kept constant for each subject over each test. Subjects were stabilized at the hips and the thigh with velcro

straps. They were encouraged to maintain a stable upper body position and to resist the tendency of the hips to displace forward during the tests. Subjects were always encouraged to exert a maximal effort, and were permitted adequate warm-up and rest (minimum of 60 seconds) between sets of exercise.

Computed Tomography (CT) Scanning

Recently, computerized x-ray tomography (CT) has been used as a precise, non-invasive technique for the determination of cross-sectional area (C.S.A.) of bone and soft tissue in humans (Maughan et al. 1983; Schantz et al. 1983; Luthi et al. 1986). CT scanning clearly defines the soft tissue borders, thereby permitting accurate distinction between fat and muscle tissue. Hudash et al. (1985) have investigated the validity and reliability of the technique for assessment of bone, fat, and muscle component C.S.A. in both cadaver and living human thighs. They reported no differences between cadaver tissue C.S.A. determined by the CT technique and by digitized planimetry of photographs. In live subjects they reported no difference between the C.S.A. determined from scans taken on separate occasions, and no difference between C.S.A. from the same scan measured on separate occasions. We have established reproducibility of C.S.A. from the same scan with less than 1% error.

Great care was taken to ensure that the first and second scans were overlapping. The pilot or scout scan was used to determine the precise distance of the scan site from the intercondylar notch on the femur on both occasions. Positioning of the thigh was standardized to ensure that the CT image was taken in the horizontal plane rather than an oblique plane. For the purposes of this study, the anterior compartment of the thigh was defined as that area including the muscles rectus femoris, vastus intermedius, vastus lateralis, and vastus medialis.

The use of the GE 8800 scanner was approved by the Cross Cancer Institute ethics committee who were fully aware of the nature of the study.

Impedance Cardiography

Impedance cardiography is a technique for non-invasive assessment of the cardiac output. An electrical field is created in the chest by passing a small (4 ma 100 KHz) across it. The movement of blood within the field, caused by ventricular ejection, alters the transthoracic impedance. The changes in impedance can be monitored electronically and are used to calculate stroke volume according to the following equation (Kubicek et al. 1966):

$$\text{Stroke Volume} = \frac{P \times L^2 \times (dZ/dt \text{ min.}) \times T}{Z_o^2}$$

where:

P = resistivity of blood which is a function of the hématocrit (H)

= $53.2e^{0.022H}$;

L = the average distance (cm) between the inner pair of electrodes measured at the anterior and posterior midlines;

dZ/dt min = minimum value for the rate of change of impedance (ohms) occurring during the cardiac cycle;

T = left ventricular ejection time (s), obtained from the impedance and phonocardiogram waveforms; and

Z_o = total mean impedance (ohms) between the inner electrodes.

Recordings were made before exercise and immediately following each of nine exercise loads which were initiated at 30 W and increased by 20 W each three minutes to a terminal load of 190 W. Following the method described by Teo et al. (1985) in validation of this technique applied to exercise testing, calculations of stroke volume were based on data obtained from five cardiac cycles with the breath held at normal end-expiration.

Stroke volumes were calculated for each exercise load and, generally, a plateau effect was observed at a heart

rate of approximately 125 beats per minute. Thereafter increases in cardiac output were achieved mainly through increased heart rate as stroke volume tended to remain relatively constant. For the purposes of this study only maximal stroke volume has been reported.

Plasma Lactate Concentration

Whole venous blood was collected at rest, and at various intervals following exercise as indicated in the main text. In each case, 0.5 ml of whole blood was immediately pipetted into 2.0 ml of chilled perchloric acid. This mixture was then shaken vigorously and placed on ice for at least five minutes to ensure complete protein precipitation. The tubes were then centrifuged for approximately 10 - 15 minutes, or until a clear, protein free supernatant could be obtained. This was then poured off into a sterile tube which was stoppered and frozen until analysis was performed.

The assay was based on the principle that lactate dehydrogenase (LDH) catalyzes the reversible reaction between pyruvic acid and NADH producing lactic acid and NAD. To measure lactate, the reaction is carried from right to left with excess NAD. To force the reaction to completion, it is necessary to trap the formed pyruvate with hydrazine. The increased absorbance at 340 nm due to

formation of NADH becomes a measure of the original lactate concentration.

All chemical reagents were obtained from the Sigma Chemical Company, and the analysis was performed according to the procedures detailed in the Sigma Technical Bulletin No. 825-UV (1981). All samples were assayed in triplicate and the mean of the three absorbance readings was used for calculation of lactate concentration.

A new standard curve was constructed for each reagent mixture based on six samples (in triplicate) of known concentration. In addition, several plasma samples were chosen at random and these were assayed with each batch of unknowns to ensure that results were consistent across different reagent cocktails, standard curves, and assay occasions. Spectrophotometric analysis was performed with a Phillips Pye Unicam PU 8800 instrument.

Hydraulic Resistance Exercise

One of the fundamental goals of the work summarized in this thesis was to investigate the nature of adaptation to circuit resistance training in previously well-trained subjects. Therefore, the training programs were designed to be both appropriate for and appealing to highly motivated athletes. The resistance apparatus was selected on the basis of ease of use (particularly in the adjustment of resistance), adaptability to circuit format, sport-specific

movement patterns, and the ability to control exercise velocity. The latter was a very important design consideration, however, ultimately some control over velocity was compromised to permit the circuit format for up to 12 subjects to train simultaneously. The selection of a system which was not strictly isokinetic was made on the premise of practicality in the hope the results would be readily reproducible in other settings.

The hydraulic cylinders (Hydra-fitness Industries Ltd.) permit reasonable control of movement velocity during exercise, provided that the range of motion is maintained as a constant and that the subject consistently provides an "all-out" effort. Katch et al. (1985) have reported reliability coefficients of up to 0.93 for numbers of repetitions achieved during 20 second sets of maximal effort exercise on separate occasions. Thus, if the range of motion and the number of repetitions during a fixed time interval are constant, average movement velocity should be constant as well. This assumption may be affected by the force of gravity in movements like knee flexion and extension which occur in the sagittal plane. In this case, flexion is "assisted" by gravity while extension is performed against gravity. The relatively greater force potential of the knee extensors may offset the effect (if any) of gravity. In any case, the average angular velocity of individual movements appears to be quite consistent.

While the cylinders are not isokinetic, accommodating resistance is provided, and consistent average angular velocities may be observed. With training, subjects may exceed the number of repetitions consistent with the desired velocity, or, alternately, with fatigue near the end of a training session, may fail to achieve the desired number. In either case, the resistance setting on the cylinder would be adjusted as necessary to provide more or less resistance and hence permit faster or slower velocities. Each training session was monitored by a supervisor who precisely timed each work and rest interval. Resistance settings and numbers of repetitions were recorded for each set of exercise at each station for all circuits. These records were reviewed following each session and adjustments made as necessary for subsequent sessions.

Initially, most subjects could not tolerate three circuits of exercise, particularly at high velocity. Prior to the commencement of the programs, subjects were thoroughly oriented to the circuit and then began the program with two circuits. As soon as possible, usually at the end of the second week, the third circuit was added. Therefore, the application of the training load was progressive in several ways. First, the accommodating resistance properties of the cylinder provided greater resistance within each resistance or flow setting. When the subject consistently exceeded the desired number of repetitions the flow setting

could be altered to provide more resistance. Finally, the volume of exercise increased to three from two circuits as permitted by the tolerance of the subjects.

Data Analysis

All statistical analysis of data was performed on the Amdahl computing system at the University of Alberta. Programs were selected from the Division of Educational Research Services (D.E.R.S.) Library, where complete documentation of each program is available. Two programs were used consistently for data analysis in each of the studies reported in the main text as follows:

ANOV 26 - performs a two-way Analysis of Variance, with repeated measures on one factor. Post hoc Scheffe comparisons are made between the main effects for each factor (Winer 1972); and

ANOV 12 - tests for the significance of the difference between means and between variances for correlated samples. Both the "t" values and the associated probabilities are calculated for each case (Ferguson 1966).

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